ORIGINAL ARTICLE

A POPULATION BASED STUDY OF PULMONARY FUNCTION BETWEEN URBAN AND RURAL SMOKERS OF KOSI REGION OF BIHAR, INDIA

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ABSTRACT: Smoking is well-known to cause respiratory disorders and pulmonary functions decline. In India, where majority of the population lives by agriculture and linked occupations in rural areas despite of rapid increase in urban population, the pulmonary function is expected to vary between rural and urban areas. Rural and urban variations in disease distribution are well known. Respiratory system evaluation and screening can easily be done by Pulmonary Function Tests. This study was carried out in the Kosi region of Katihar, Bihar, in 100 participants. For this study, computerized spirometer (RMS Helios 701) was used. In view of increasing behaviour of smoking among the rural and urban population of Kosi region of Bihar, this study was undertaken, for a better understanding of the correlation between smoking and its effects on pulmonary functions. It was observed that pulmonary function in mean ± standard deviation in urban smokers, FVC was 2.54 ± 0.86 litres, FEV_1 1.81 ± 0.88 litres, FEV_1 % was 74.83 ± 31.43 and PEFR was 5.98 ± 2.35 litres and $FEF_{25-75\%}$ was 2.95 ± 1.31 litres. The pulmonary function tests in rural smoker population in mean ± standard deviation, FVC was 2.56 ± 0.86 litres, FEV_1 2.21 ± 0.96 litres, $FEV_{1\%}$ was 86.00 ± 23.73 and PEFR was 5.65 ± 2.18 litres and $FEF_{25-75\%}$ was 3.34 ± 1.37 litres. The comparison of PFT in urban smokers and rural smoker population was significant with "p" value <0.05 only in FEV_1, other parameters showed insignificant results.

KEYWORDS: Smoking, Urban, Rural, Pulmonary function test, Bihar.

INTRODUCTION: Rural and urban variations in disease distribution are well known. Chronic bronchitis, accidents, lung cancer, cardiovascular diseases, mental illness and drug dependence are usually more frequent in urban than in rural areas. On the other hand, skin and zoonotic diseases and soil- transmitted helminthes may be more frequent in rural areas than in urban areas. Death rates, especially infant and maternal mortality rates are higher for rural than urban areas. These variations may be due to differences in population density, social class, deficiencies in medical care, and levels of sanitation, education and environmental factors.[1] Due to socioeconomic differences, the majority of rural population is migrating to the urban areas, seeking education, income & occupation. This trend greatly increased the urban population, including urban slums in recent years. Tobacco kills more than five million people worldwide. Tobacco uses both in the smoking and non-smoking form is quite common in India; about 15% to over 50% men use tobacco in this country. Thus tobacco smoke related respiratory diseases like COPD, lung cancer etc., are increasing rapidly. Furthermore, tobacco consumption has a

deleterious effect on the course of bronchial asthma, pulmonary tuberculosis, lung function and other lung diseases.[2]

Smoking produces inflammatory changes in small airways, especially in respiratory bronchioles. This leads to dilatation and destruction of small airways, characterized as emphysema.[3] The pulmonary damage induced by smoking acts slowly and may show no symptoms until pulmonary functions are lost.[4] In a study among urban and rural adult population Gaur et al., the prevalence of bronchial asthma and allergic rhinitis was found to be higher than reported earlier from India. Smoking was one of the major risk factors for higher prevalence of bronchial asthma and allergic rhinitis.[5] Pulmonary function test is a valuable tool for evaluation and assessment of the respiratory system. In India, where majority of the population lives by agriculture and linked occupations in rural areas despite of rapid increase in urban population, the pulmonary function is expected to vary between rural and urban areas. Spirometry test alone can identify substantial number of subjects with lung abnormalities, without exposing them to radiation or other expensive methods. It is useful for identifying both type of patients, patients with expiratory airflow limitations and patients with reduced lung volumes i.e. both obstructive and restrictive pattern can be identified.[6] Another commonly used method to assess pulmonary function is questionnaires, but the reliability of this method is limited.[7] Spirometry provides reproducible and quantifiable measures of pulmonary function.

MATERIALS AND METHODS: This study was carried out in the Kosi region of Katihar, Bihar, India. The sample size taken was 100. Prior consent was obtained from the Ethical committee. Informed consent was taken from the 100 participants before performing the pulmonary function tests.

For this study, the computerized spirometer (RMS Helios 701) was used. The spirometry test was done in the day time between 10.00 A.M. to 2.00 P.M to avoid any diurnal variations.

Daily calibration of spirometer was done with a 3 litre syringe, before starting the tests. The weighing machine was also calibrated daily with a standard 10 kg weight.

Written and informed consent was taken from the subjects before doing the pulmonary function test. A detailed history taking of the subject were asked. A complete general examination was done to rule out exclusion criteria.

SELECTION CRITERIA: Strict selection criteria were followed to select the sample size as mentioned below:

INCLUSION CRITERIA:

1. Informed consent from the subject.
2. Subjects in the age range between 20-70 years.
3. Smokers from rural and urban population of Kosi Region Katihar.
4. Smokers with present or past history of at least 5 years of smoking.

EXCLUSION CRITERIA:

1. Those subject who did not give consent.

2. Recent myocardial infarction less than one month old.
3. Asthma and COPD subjects.
4. Chronic infections such as tuberculosis or other infections of lungs.
5. Subjects with respiratory symptoms such as cough.
6. Hemoptysis of unknown origin (forced expiratory maneuver may aggravate the underlying condition).
7. Pneumothorax.
8. Thoracic, abdominal, or cerebral aneurysms.
9. Recent eye surgery (e.g., cataract).
10. Presence of an acute disease process that might interfere with test performance (e.g., nausea, vomiting).
11. Previous accidents or surgery involving thorax or abdomen.
12. Subjects who were not able to give desired co-operation for the test procedure.

In the start of study screening questionnaires were asked to confirm the exclusion criteria but later, it was omitted as history taking and general examination sufficed the purpose of selection criteria. A detailed history taking and general examination was done to rule out exclusion criteria.

Before performing pulmonary function test, following points were ascertained that the,

- Subject has not consumed alcohol within four hours.
- Has not smoked within one hour.
- Has worn comfortable clothing, not restricting chest and abdominal movements.
- Has not performed vigorous exercise within half an hour.

These subjects were advised to come on next day for test. The statistical analysis was done using the ' z ' test, assuming ' p ' <0.05 as significant

OBSERVATION: Observations are presented in tables and histograms.

I. Pulmonary Function Test (PFT) results in Kosi Region of Bihar. .

A. PFT results in urban smokers subjects.

PFT results in Urban Smokers (US) (n=50)		
Parameters	**Mean**	**Standard Deviation (SD)**
FVC (in litres)	2.54	0.86
FEV_1(in litres 1sec)	1.81	0.88
FEV_1% (percentage)	74.83	31.43
PEFR(in litres/min)	5.98	2.35
FEF_{25-75}% (in litres)	2.95	1.31

Table 1

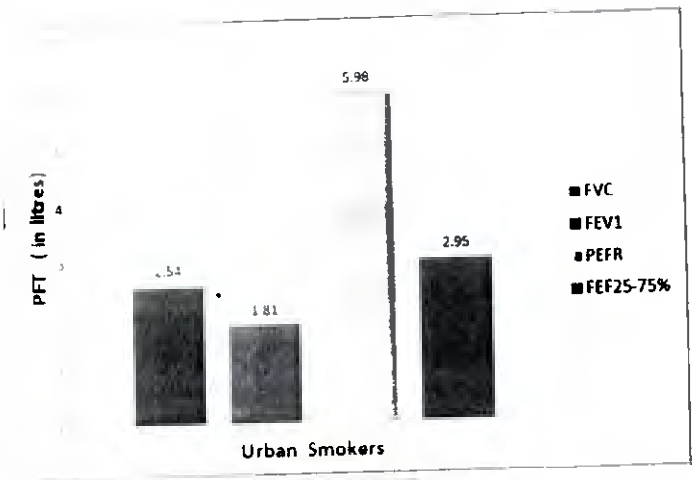

Chart 1

B. PFT results in rural smokers male.

PFT results in Rural Smokers (RS) (n=50)		
Parameters	**Mean**	**Standard Deviation (SD)**
FVC (in litres)	2.56	0.86
FEV_1(in litres 1sec)	2.21	0.96
FEV_1% (percentage)	86.00	23.73
PEFR(in litres/min)	5.65	2.18
FEF_{25-75}% (in litres)	3.34	1.37

Table 2

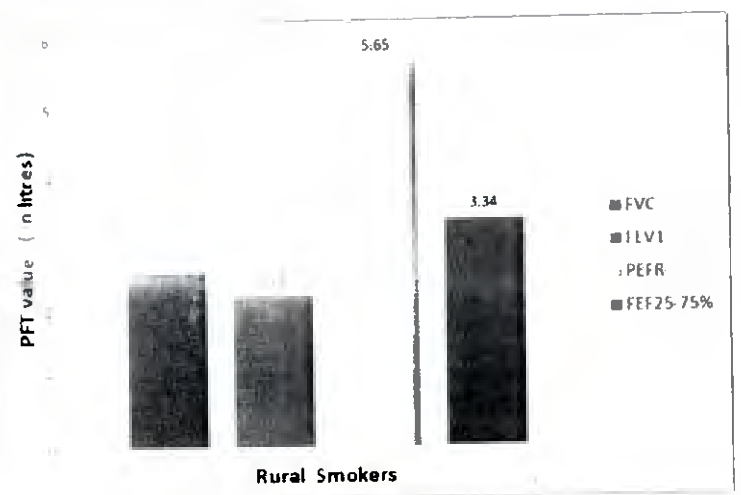

Chart 2

PFT	US MEAN±SD n=50	RS MEAN±SD n=50	S.E.D (Standard error of difference between two means)	Z value	P value
FVC	2.54±0.86	2.56±0.86	0.15	0.13	>0.05
FEV_1	1.81±0.88	2.21±0.96	0.16	2.5	<0.05 *
FEV_1%	74.83±31.43	86.00±23.73	5.56	2.00	>0.05
PEFR	5.98±2.35	5.65±2.18	0.44	0.75	>0.05
FEF_{25-75}%	2.95±1.31	3.34±1.37	0.24	1.62	>0.05

Table 3

* significant

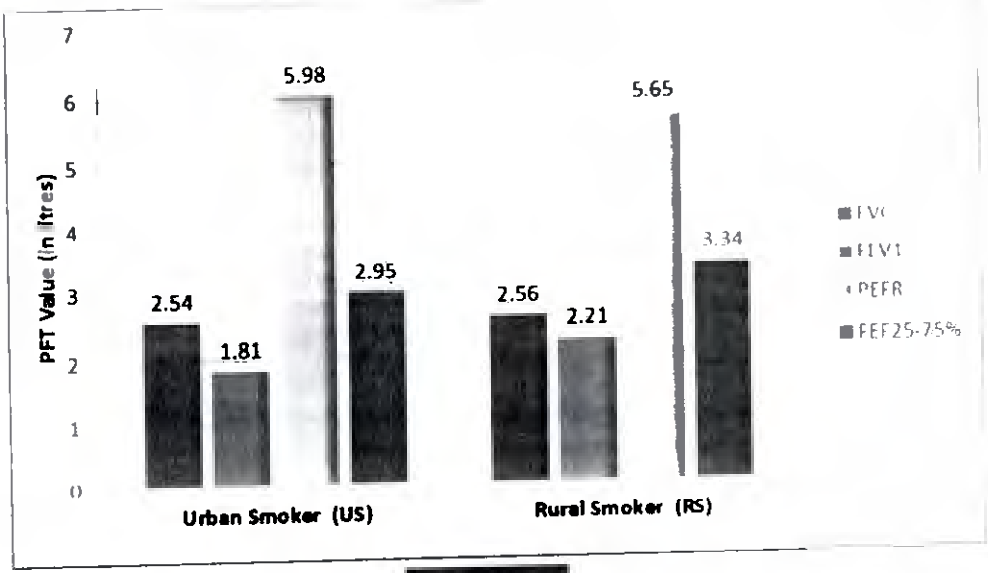

Chart 3

Table 1 and chart 1 shows pulmonary function tests in urban smoker population with different parameters, in mean ± standard deviation. FVC was 2.54± 0.86 litres, FEV1 1.81± 0.88 litres, FEV1 % was 74.83± 31.43 and PEFR was 5.98 ± 2.35 litres and FEF 25-75% was 2.95 ± 1.31 litres.

Table 2 and chart 2 shows pulmonary function tests in rural smoker population with different parameters, in mean ± standard deviation. FVC was 2.56 ± 0.86 litres, FEV_1 2.21± 0.96 litres, FEV_1 % was 86.00 ± 23.73 and PEFR was 5.65 ± 2.18 litres and FEF $_{25\text{-}75\%}$ was 3.34 ± 1.37 litres.

Table 3 and chart 3 shows the comparison of PFT in urban smokers and rural smoker population of Kosi region, Katihar, Bihar.

As shown in Table 3, chart 3 the mean FEV_1 in rural smoker population at a value of 2.21±0.96 litres was significantly higher in comparison to urban smoker males having a mean

FEV_1 of 1.81±0.88 litres. The mean FEV_1% also was higher in rural smoker population at a value of 86.00±23.73% in comparison to urban smoker population at a value of 74.83±31.43%. Data of comparative study was significant with "p" value <0.05. The other pulmonary function parameters were insignificant between smokers of urban and rural population ("p" value was >0.05).

DISCUSSION: This study included data on 100 subjects in the age group of 20 -70 years with 50 nonsmokers and 50 smokers. The study observed decreased pulmonary functions value in urban smoker population compared to the rural smoker population. It is showed in Table 3.that there were statistically significant changes in pulmonary function in terms of FEV_1 ("p" value < 0.05) between urban smokers and rural smoker population. The other pulmonary function parameters were insignificant between smokers of urban and rural population ("p" value was >0.05). Our study did not show any significant association between urban and rural population as shown by other workers. This may be explained by less sample size and lesser socioeconomic differences between urban and rural population of Katihar. Katihar is a small district of Bihar, where urban rural differences are not very wide. The air pollution and environmental factors are also not very much different between urban and rural population in Katihar as indicated by lesser number of vehicles, less construction works and industries in urban areas. The study done by previous workers were done mainly in major cities having wider variations between urban and rural areas. Smoking is well-known to cause respiratory disorders and pulmonary functions decline and when it co-exists with air pollution, the effects could be more harmful. Tobacco smoking is widely prevalent all over the world and it continues to rise in developing countries. By 2030 the developing world is expected to have 7 million deaths annually from tobacco use.[1] The study by Gaur, Gupta, et.al, was done in Delhi having wider difference between urban and rural population. Their study showed a higher FEV1 in rural population while FEV1% was higher in urban population.[5] The study by Mohan Rao, et.al, was done in Andhra Pradesh, showed increased vital capacity, reduced FEV1% and FEF25-75% in rural workers in comparison to urban workers.[9] The Lisa Iversen et.al, study done in Scotland, showed that living in rural areas was associated with lower prevalence of asthma and has better health status in compared to urban population.[10] Their study was carried out by collection of postal questionnaires but our study is done by actual performance of pulmonary function tests by computerized spirometer. In our study the rural population has better pulmonary function in comparison to urban population in smoker category. The study by Glew et.al in children of northern Nigeria also showed higher pulmonary function in rural population as indicated by higher FVC and PEFR values in rural males in comparison to urban population.[11] In the study by Kostas NP, Michael BA et al. in school childrens of Athens showed adverse effects on lung function in urban population as compared to those living in rural environment.[12] Their methodology consisted of both, questionnaires and spirometric evaluation of lung function. Physical activities also affect pulmonary functions and the higher FEV1 in the rural smokers in our study may also be attributed to physical activity. As most of the rural male smokers were agricultural or other type of workers and were physically more active then the urban smoker population. This is in accordance with the CCHS study by Judith Garcia et.al. The Copenhagen City Heart Study (CCHS) by Judith Garcia et.al showed that

moderate to high levels of regular physical activity were associated with a lower lung function decline in active smokers.[13] Our study was similar to the various studies done previously in Indian as well as Foreign studies and revealed urban population has decreased pulmonary function in FEV_1 parameter and a detailed pulmonary function assessment is required in Kosi Region of Bihar where prevalence of smoking is higher. The role of conditions like low socioeconomic status, malnutrition, pollution and other factors in affecting pulmonary function need to be evaluated.

CONCLUSION: There was significant decreased pulmonary function in the urban smoker population in comparison to the rural smoker population. Further study for the role of other factors affecting pulmonary function is required.

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ORIGINAL RESEARCH ARTICLE

Vitamin D3 Deficiency in Hypothyroidism Patients: A Hospital Based Prospective Study.

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Abstract:

Objectives: This present study was to find the various parameters like age, TSH levels, FT4, FT3, anti TPO levels and vitamin D3 levels in hypothyroidism patients. Methods: Detail history clinical examinations and relevant investigations were performed to all subjects. Sample was collected for the estimation of serum TSH, FT3, FT4, Anti-TPO and Vitamin D3 levels. Under complete aseptic conditions venous blood was withdrawn from antecubital vein. Levels of FT3, FT4, TSH, Anti-TPOAb and Vitamin D3 were estimated using fluorescence array. Results: Data was analyzed by using SPSS version 26 software. One sample statistical methods analysis was used. Mean ± standard deviation were calculated. P value was taken less than or equal to 0.05 for significant differences (p ≤ 0.05). Conclusions: Hypothyroidism patients had significantly decreased vitamin D3 levels and increased TSH levels. Therefore, regular screening of vitamin D3 levels should be performed in all hypothyroid patients. And vitamin D3 supplementation should be recommend for early prevention and management of vitamin D3 deficiency in hypothyroidism patients.

Key words: Vitamin D3, Hypothyroidism, TSH, FT4, FT3, anti TPO.

Introduction:

Vitamin D deficiency is a global health problem [1]. Over a billion people worldwide are vitamin D deficient or insufficient [1].

Vitamin D deficiency has been shown to be associated with autoimmune diseases, including rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), inflammatory bowel disease (IBD), multiple sclerosis (MS) and type 1 diabetes (T1DM), and that vitamin D supplementation prevents the onset and/or development of these autoimmune diseases [2]. Furthermore, it was reported that patients with Hashimoto's thyroiditis, an autoimmune thyroid disease had lower vitamin D levels [3].

The molecular mechanism by which vitamin D exerts its action seems to be mediated by its binding to VDR, an intracellular receptor belonging to the steroid/thyroid nuclear receptor family, expressed by human immune cells, such as macrophages, dendritic cells and T and B lymphocytes. Vitamin D central target are dendritic cells (DCs) [4]. In particular, it has been shown that 1, 25(OH)2D3 and calcifediol impair T-cell-stimulatory capacities of murine DC [5]. DCs isolated from VDR knockout mice were not impaired in their T-cell-activating potential, demonstrating that the inhibitory effect of these vitamin D metabolites was dependent on the presence of VDR [5].In particular, 1,25(OH)2D3 inhibited DC maturation as well as production of DC-derived cytokines, such as interleukin (IL)-12 and IL-23.

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Moreover, 1,25(OH)2D3 decreased the production of inflammatory Th1 cytokines such as IL-2 and interferon-γ, which promote cell-mediated cytotoxicity leading to thyroid destruction in HT [7]. In this regard, it is worth mentioning that the suppression of Th1 response by Vitamin D may counteract the onset of GD, as Th1 dominance seems to be involved in the induction of the disease [8]. Finally, 1,25(OH)2D3 inhibits Th17-derived cytokines IL-17 and IL-21 production, promoting the induction of regulatory rather than effect or T cells [7].

Vitamin D plays an essential role in calcium homeostasis and the development and maintenance of the skeleton [9]. It is recognized as the sunshine fat soluble vitamin. Exposure to ultraviolet B light (290–320 nm) are the main source of vitamin D [10]. Objectives of this present study was to found the levels of TSH, FT4, FT3, TPO ab and its correlation with vitamin D3 levels in hypothyroidisms patients.

Materials & Methods:

This present study was conducted in Department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar during a period from March 2019 to November 2019. Attendant of entire subjects /subjects signed an informed consent approved by institutional ethical committee of KMCH, Katihar, Bihar, India was sought.

In this present study, we were enrolled a total of 60 patients of hypothyroidism with age group 20 to 70 years. Data was collected with irrespective of sex by random sampling methods.

Procedures:

Detail history clinical examinations and relevant investigations were performed to all subjects. The patients who were known case of hypothyroidism were included in this study. And the patients were receiving any one Vitamin D3 supplement were excluded from this study.

Laboratory findings: Sample was collected for serum TSH, FT3, FT4, ANTI-TPO and Vitamin D3 level estimation. Under complete aseptic conditions venous blood was withdrawn from antecubital vein. Levels of FT3, FT4, TSH, Anti-TPOAb and Vitamin D3 were estimated using fluorescence array. Patients with TSH levels greater than 10 U/mL were taken as overt hypothyroids. Subclinical hypothyroid: >5-7 U/mL and Euthyroid: 0.25-5 U/mL.

Serum 25(OH)D3, the most abundant circulating precursor of active Vitamin D, is the most reliable and widely accepted indicator of Vitamin D status. Vitamin D deficiency is defined as a 25(OH)D3 below20 ng/ml and Vitamin Dinsufficiency as 25(OH)D3of 21–29 ng/ml. Levels of 25(OH)D3 >30 ng/ml are considered to be optimal [11].

Statistical Analysis:

Data was analysed by using SPSS version 26 software. One sample statistical methods was used. Mean ± standard deviation (S.D) and t- value were calculated. P-value was taken equal to or less than 0.05 for significant differences ($p \leq 0.05$).

Observations and Results:

In this present study, we were enrolled a total 60 patients of hypothyroidism. There were 20(33.33%) males and 40(66.66%) females. Male and female ratio was 1:2.

Table 1: various parameters of patients with hypothyroidism.

Parameters	Mean±S.D.	t-value	P-value
Age	53.266± 10.381	39.74	P<0.0001
TSH (uIU/ml)	20.266±5.329	29.45	P<0.0001
FT4(ng/dL)	3.892±1.560	19.32	P<0.0001
FT3(ng/dL)	2.583±1.093	18.30	P<0.0001
Anti TPO(IU/mL)	5.333±1.838	22.47	P<0.0001
Vit D3(ng/mL)	22.900±7.646	23.19	P<0.0001

In this present study, mean ± standard deviations of age of hypothyroid patients were 53.266± 10.381 years. And it was significantly differenced (p=0.000). Mean±S.D of TSH was 20.266±5.329 uIU/ml and it was significant differenced (P<0.0001). Mean±S.D of FT4, FT3 and anti TPO were 3.892±1.560 ng/mL, 2.583±1.093ng/Ml and 5.333±1.838 IU/mL respectively. All these parameters were also significantly differenced (p<0.0001). Similarly, Mean±S.D of vitamin D3 was 22.900±7.646 ng/mL, it was also significant differenced (p<0.0001).

Table.2. Status of vitamin D3 level in cases

Vitamin D3	No. of cases	Percentage
Sufficient	16	26.7%
Insufficient	24	40.0%
Deficiency	20	33.3%

In this present study, 16(26.67%) cases of hypothyroidism had sufficient vitamin D3 levels. And 44(73.33%) patients had decreased vitamin D3 levels. Among them 24(40%) had insufficient and 20(33.33%) patients had significant deficiency of vitamin D3 levels.

Discussions:

Vitamin D plays an important role in preventing the occurrence of many inflammatory diseases, infections, and autoimmune diseases [12]. In numerous studies, the relationship between vitamin D deficiency and a variety of diseases, including musculoskeletal [13], cardiovascular [14], kidney disease [15], diabetes [16] and infections [17] had been shown. The thyroid gland is also one of the organs that have a receptor for vitamin D. The vitamin D receptor in the thyroid is a member of a large group of receptors called nuclear receptors, which also belong to the thyroid hormones receptor [18]. Some studies indicated that vitamin D deficiency is associated with various autoimmune diseases [19].Today, Hashimoto is one of the mostcommon acquired hypothyroidism and autoimmune disease in children and adults [20]. The onset of autoimmune-thyroid disease with vitamin D deficiency is very common [21].

Few studies were conducted to find any significant association between the levels of Vitamin D and hypothyroidism and it's pathogenesis but yieldedconflicting results. Kivity et al. in 2011 documented significantly low levels of 25(OH)D3 with autoimmune thyroid disease, whereas a study by Goswami et al showed a weak association between 25(OH)D3 levels and thyroid peroxidase antibody (TPOAb) titers [22, 23].

In our present study, 60 hypothyroidism patients were enrolled. Male and female ratio was 1:2. Average age of patients was 53.266± 10.381 years. And it was significantly differenced (p=0.000).

Canaris GJ, et al (2000) conducted a study and found that 7-95% females and 1-2% males across the world that has variable thyroid conditions [24]. In our present study 66.67% females and 33.33% males hypothyroid patients were suffered with vitamin D3 deficiency.

Swati Sonawane et al. [25] observed that out of 90 subjects, there were 58.8% patients (n=53) who had Vitamin D deficiency i.e. the Vitamin levels were less than 20 ng/ml. There were 73 cases of euthyroid in which the TSH levels were between 0.25-5 U/U/ml. There were 10 cases of subclinical hypothyroid and 7 cases of overt hypothyroidism. The mean levels of Vitamin D in subclinical and overt hypothyroidism were 16.23+/-10.47 and 13.11+/-10.48 ng/ml respectively. There was a significant difference in the level of Vitamin D in all the cases.

In our present study, we were found that in out of 60 cases of hypothyroidism, mean level of TSH, FT4, FT3 and anti TPO were 20.266±5.329uIU/ml, 3.892±1.560 ng/mL, 2.583±1.093ng/Ml and 5.333±1.838 IU/mL respectively. All these parameters were also significantly differenced (p=0.000).

Vitamin D deficiency was considered virtually nonexistent In the Indian population as India lies in the tropical area [26].But now a days various studies have revealed that 50-90% of the Indian population is deficient in Vitamin D due to inadequate dietary intake of Calcium [27].

Several studies have reported low serum levels of vitamin D in hypothyroid patients which in turn may lead to some musculoskeletal complaints in these patients [28]. Other studies have demonstrated that the patients with Graves' disease also have low serum levels of vitamin D [29]. There are two mechanisms that may explain why serum levels of vitamin D is low in hypothyroid patients; one is that the low levels of vitamin D may be due to poor absorption of vitamin D from the intestine and the other is the body of these patients may not activate vitamin D properly [30].

In our present study, we were observed in atotal 50patients of hypothyroidism, 16 (26.67%) cases had sufficient vitamin D3 levels (>30 ng/mL). 24(40%) had insufficient vitamin D3 levels (20-30 ng/mL). And 20(33.3%) hypothyroidism patients had deficiency of vitamin D3 levels (<20g/mL). And among all 60 hypothyroid patients, average mean of vitamin D3 was 22.900±7.646 g/mL. And it shows significant deficiency of vitamin D3 (P=0.000).

In a study by Chaudhary et al [31] was seen that administration of 60,000 IU vitamin D weekly in autoimmune thyroid disorders (AITD) had a favourable effect on autoimmunity as evidenced by significant reductions in TPO Ab titers. In addition, vitamin D3 intake after 10 weeks in diabetic rats greatly corrected the alterations in thyroid profile and D2 (deiodinase 2) expression [32].

A study from Japan including 200 patients with Graves' disease demonstrated that 40% of women and 20% of men had vitamin D deficiency [33]. Some other studies have indicated that patients with Graves' disease also have low levels of vitamin D [34] According to these findings, Afsaneh Talaei, et al. [35] showed that the prevalence of vitamin D deficiency was high in hypothyroid patients. Vitamin Dsupplementation significantly decreased TSH levels but had no significant effect on T4 or T3 concentrations. They found significant relationship between vitamin D deficiency and hypothyroidism.

Mackawy et al. [36] concluded that the patients with hypothyroidism suffered from hypovitaminosis D and there was a positive significant correlation between serum level of vitamin D with thyroid hormones and a negative significant correlation with TSH levels and suggested that the deficiency of serum levels of vitamin D was significantly associated with the degree and severity of hypothyroidism.

Conclusions:

This present study concluded that the patients with hypothyroidism had significantly decreased vitamin D3 levels and increased TSH levels. Therefore, regular screening of vitamin D3 levels should be performed in all hypothyroid patients.

And vitamin-D3 supplementation should be recommended for early prevention and management of vitamin D3 deficiency in hypothyroidism patients.

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ORIGINAL RESEARCH ARTICLE

Study of Serum Mean Platelet Volume in Ischemic Stroke Patients: A Case - Control Study.

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Abstract:

Objectives: Platelets play a major role in pathogenesis of vascular disease. Platelet size and function is measured by Mean Platelet Volume (MPV). Platelet activity is accentuated in acute ischemic stroke due to blood vessel occlusion that leads to ischemia, endothelial damage and new platelet formation. Thus, the mean platelet volume is elevated in ischemic stroke. This present study was to correlate the serum mean platelet volume with modified rank in scale as functional outcome in ischemic stroke patients. Methods: This present study was to categorized into two groups (case group and control group). Case group had 35 ischemic stroke patients. And control group had 35 normal subjects. A complete assessment of all subjects (case-control) including detail history, clinical examinations and relevant investigations (platelet count, mean platelet volume) were performed. The diagnosis of ischaemic stroke was made clinically with the evidence of acute lesions (infarct) confirmed by brain CT or MRI within the first 24 h of presentation of symptoms. And modified Rankin Scale was used for assessment of functional outcome for each patient. Results: Data was analysed by using SPSS version 26 software. Paired samples statistics was used. Mean and standard deviation were observed. P value was taken less than or equal to 0.05 for significant differences ($p \leq 0.05$). Conclusions: Mean platelet volume was higher in ischemic stroke cases that had higher Modified Rankin scale. Mean platelet volume was very significantly elevated in ischemic stroke patients. Most of the ischemic stroke patients had moderate disability, required some help but able to walk without assistance. Hence, MPV can be used as laboratory marker for the diagnosis of ischemic stroke.

Key words: Mean platelet volume, Ischemic stroke, modified Rankin score.

Introduction:

Stroke is associated with increased long term mortality, residual physical, cognitive, and behavioural impairments, recurrence, and increased risk of other types of vascular events [1].Several factors are known to increase the liability to stroke, and it has been here that large-scale public health measures have had a substantial influence. The most important of these are hypertension, heart disease, atrial fibrillation, diabetes mellitus, cigarette smoking, and hyperlipidemia [2].

Acute ischemic stroke (AIS) has been clinically defined as a sudden-onset loss of focal cerebral function that persists for more than 24 hours. Worldwide, it is the second most prevalent reason for death and the most common reason for long-term incapability [3].

In 2013, stroke was the second most common cause of deaths (11.8% of all deaths) worldwide, after ischemic heart disease (14.8% of all deaths), and the third most common cause of disability (4.5% of DALYs from all cause) after ischemic heart disease (6.1% of DALYs from all cause) 2 in India.

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The overall age adjusted prevalence rate for stroke is estimated to lie between 84-262/100,000 in rural and between 334-424/100,000 in urban areas [4].

Platelets play a crucial role in the pathogenesis of atherosclerotic complications, contributing to thrombus formation [5]. Platelets are anucleate cells and are heterogeneous regarding their size, density and haemostatic potential. Platelet size (mean platelet volume, MPV) is a marker and possibly determinant of platelet function, large platelets being potentially more reactive.

In normal individuals, the platelet count is inversely proportional to MPV; platelet mass (the product of MPV and platelet count) is a near constant. Although platelets are incapable of de novo protein synthesis, they are very active metabolically and respond rapidly to vascular injury or trauma by undergoing a series of reactions (adhesion, release of granule contents, shape change and aggregation), which ultimately result in the formation of a platelet-fibrin plug. Thus, there is evidence that platelet function is accentuated in acute ischemic stroke [6].In this present study, we were evaluate the various factors like age, platelet count and mean platelet volume in ischemic stroke patients.

Materials & Methods:

This present study was conducted in Department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar, India during a period from September 2019 to February 2020. Attendant of entire subjects signed an informed consent approved by institutional ethical committee of KMCH, Bihar, India was sought.

This present study consists two groups. Group A had case group who were 35 subjects of ischemic stroke with age 35 to 70 years. Group Bhad control group who were 35 normal subjects with age 35 years to 65 years no clinical evidence of any active or old cerebrovascular accidents, malignant causes and not on any drugs (medications) affecting the function of the platelets.

Procedures:

A random sampling method was used for data collection with irrespective of sex.

Detail history clinical examinations and relevant investigations were performed to all subjects. Patients who had peripheral vascular disease, previous stroke patients, acute bacterial/viral infections, any inflammatory conditions, pregnancy, acute myocardial infarction, malignancies symptoms of cerebrovascular diseases were excluded from this study.

Lab parameters included as platelets counts, mean platelet volume and others were performed. The diagnosis of ischaemic stroke was made clinically with the evidence of acute lesions (infarct) confirmed by brain CT or MRI within the first 24 h of presentation of symptoms. Each patient condition was assessed by Rankin Scale. Severity of ischaemic stroke was Modified Rankin scale that scores on a scale of 0–6, with 0 as patients with no symptoms and 6 being dead. Patients with scores 1 are no significant disability and able to carry out all usual activities despite some symptoms. Patients with scores of 2 are considered slight disability and able to look after own affairs without assistance, but unable to carry out all previous activities. Patient with Scores of 3 or greater considered as more severe and need support to lead their daily life. Patients with scores 4 are considered as moderately severe disability and unable to attend to own body needs without assistance and unable to walk unassisted. And patients with scores 5 are considered as severe disability, requires constant nursing care and attention, bedridden, incontinent.

Statistical Analysis: Data was analyzed using SPSS version 26 software. Unpaired t-test was applied to check significance difference between two groups. Mean and standard deviation were calculated. 5% level of significance was considered.

Observations and Results:

This present study consists of two groups. Group A (case group) had 35 subjects with ischemic stroke. And group B (control group) had 35 normal subjects with no history of ischemic stroke.

Table.1. Gender wise distribution of patients

Gender wise distribution	Case (N=35)		Control (N=35)	
	Male	Female	Male	Female
No. of subjects	24(68.57%)	11(31.43%)	22(62.86%)	13(37.14%)

In this present study majorities of cases were male. 24(68.57%) and 22(62.86%) most of the males were seen in group A and group B respectively.

Table.2. Comparison of Mean Age, Platelet count & MCV

Parameters	Case (N=35) Mean ± S.D	Control (N=35) Mean ± S.D	t-value	p-value
Age	56.428 ± 9.245	50.828 ± 10.130	2.231	P=0.032
Platelet	215547.000 ± 66179.143	274607.657 ± 75244.315	3.368	P=0.002
MPV	9.600 ± 1.034	5.371 ± 1.086	15.361	P<0.0001

In this present study, mean age of group A (case) was 56.428±9.245 and group B was 50.828 ± 10.130 years. And it was significantly differenced (p=0.032). Mean platelet count of group A and group B was 215547.000 ± 66179.143 and 274607.657 ± 75244.315 respectively. And it was also significantly difference (p= 0.002). Mean platelet volume of group A and group B was 9.600 ± 1.034 and 5.371 ± 1.086 cells/mm^3 respectively. And it was significantly differenced (p=0.000).

Table.3. Modified Ranking scores of stroke patients

Modified Ranking Scores	Stroke patients	Percentage
2	3	8.57%
3	16	45.71%
4	10	28.57%
5	6	17.14%
Total	35	100%

In this present study, among all ischemic stroke cases, Modified Rankin scores of majorities of cases 16(45.71%) had 3 score. 10(28.57%), 6(17.14%) and 3(8.57%) cases had 4, 5 and 2 scores respectively.

Discussions:

Stroke is the most common neurological disorder worldwide and it is the most frequent of all the neurological disorders. Stroke is also known Cerebrovascular Accident (CVA), derived from Greek word in the year 1599 which means 'Struck Down' [7]. It is thought that MPV is an indicator of platelet activation that is accepted to be associated with systemic inflammatory responses. The relationship between ischemic stroke and MPV has been thoroughly examined in numerous publications. In earlier studies, it was generally accepted that increased platelet activation is related to cerebral infarction and coronary heart disease, while recent reports have shown controversial results regarding the association between MPV and stroke [8,9].

In present study, 35 ischemic stroke patients were included in case group A and 35 normal subjects were included in control group B. Mean age of ischemic stroke patients had 56.428 ± 9.245 years. And control group had 50.828 ± 10.130 years. Mean age group of case and control were significantly differenced (p=0.032).Most of the ischemic stroke patients were male 24(68.57%).

These findings were in concordance with the study done by Javed Akhter Rathore et al [10] and R P Eapen et al [11] where maximum frequency of stroke was seen between ages 55-74 years and 51-60 years. Age is the single most important risk factor for stroke. For each successive 10 years after age 55, the stroke rate doubles in both men and women [12,13]. Study done by V.R Bhatt el al [14] and R P Eapen [15] also suggest that incidence of stroke events are more in males than in females. The most common gender difference in stroke is due to lifestyle factors such as cigarette smoking and alcohol consumption in males.

O'Malley et al [16] reported a higher elevated MPV in patients with acute and chronic ischemic stroke than in controls, but they did not find any significant distinction in the MPV values between the acute and chronic phases of stroke. They also speculated that changes precede the vascular event. Finally, they concluded that MPV is the most important significant variable of the factors associated with stroke; an elevation in MPV level is independently regarded with stroke. Domac et al [17 reported that a severe stroke had significantly increased MPV levels at admission, reflecting higher platelet reactivity. Muscari et al [18] observed higher MPV values in arterial stroke with the greatest neurological impairment, while D'Erasmo et al [19] and Butterworth et al [20] reported higher platelet volume in patients with AIS than in control individuals. Greisenegger et al [21] concluded that patients who had suffered an intense stroke attack already had an elevated MPV level, reflecting increased platelet reactivity just before the stroke occurred.

In our present study, a significant (p=0.002) decrease of Mean platelet count of ischemic stroke patients (215547.000 ± 66179.143) was seen with respect to normal subjects (274607.657 ± 75244.315).

But mean platelet volume of ischemic stroke patients (9.600 ± 1.034) was very significantly higher (p=0.000) with respect to normal subjects (5.371 ± 1.086).

Bath et al [22] concluded that MPV is increased with AIS, but that the physiological mechanisms that regulate MPV within the megakaryocyte require explanation. Some studies have argued that high MPV is associated with acute cerebral strokes in patients with atrial fibrillation and sinus rhythm [23]. Li et al [24] demonstrated that individuals with a high MPV have a higher prevalence of silent cerebral infarction. Ha et al [25] stated that the patients with MPV higher than 9.4 fL had an up to four-fold elevated risk for cerebral stroke attack. Similar findings was obtained in present study.

Functional outcomes, which are measured by means of disability and an individual's loss of

independence in activities of daily living, are considered to be among the most meaningful patient outcomes [26]. In this study modified Rankin scale (MRS) was used as a measure to assess the functional outcome in neurologic patients. It is a clinician reported measure of global disability and has been widely applied for evaluating stroke patient outcomes, degree of disability or dependence in daily activities [27].

In our present study, majorities of ischemic stroke cases 16(45.71%) had 3 modified Rankin score. They had moderate disability, required some help but able to walk without assistance. 10(28.57%) ischemic stroke cases had 4 modified Rankin scores. They had moderately severe disability, unable to walk without assistance and unable to attend to own bodily needs without assistance.

Elsayed AM et al [28] were supported the findings of our study. They stated that MPV value was higher and more significant (p= 0.011) in patients' group with high Rankin scale (MRS) in comparison with those with lower scores.

Our findings showed no correlation between MPV and MRS scores. We could not show any correlation between worst outcome and elevated MPV levels. In accordance with our data, Ntaios et al [29] concluded that MPV is not related to stroke severity or functional outcome, and does not differ between stroke subtypes. Similarly, O'Malley et al [16] found no association between platelet volume and prognosis.

Conclusion:

This present study concluded that the mean platelet volume was higher in ischemic stroke cases that had higher Modified Rankin scale. Mean platelet volume was very significantly elevated in ischemic stroke patients. Most of the ischemic stroke patients had moderate disability, required some help but able to walk without assistance. Hence, MPV can be used as laboratory marker for the diagnosis of ischemic stroke.

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Hospital Based Study of Metabolic Syndrome in Female Patients of Hypothyroidism.

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ABSTRACT

Background: Hypothyroidism was found to be associated with metabolic syndrome, with females being more at risk than males. The potential contributory role of the metabolic syndrome to cardiovascular risk and its scope in subjects with hypothyroidism is the focus of this study.**Methods:** Forty untreated hypothyroid women and forty normal, healthy subjects were recruited for the study. Fasting blood samples were collected for lipid profile, glucose and insulin level estimation.**Results:** Systolic and diastolic blood pressures in hypothyroidism patients were significantly higher than values in controls. Similarly, BMI, waist circumference was higher among hypothyroidism subjects. Again, fasting plasma glucose, total cholesterol, triglycerides and LDL-cholesterol levels were higher in patients with hypothyroidism in comparison to controls, while HDL- cholesterol, insulin, HOMA-IR were higher among controls. **Conclusion:** Hypothyroidism is significantly associated with metabolic syndrome and its components like dysglycemia, hypertension and dyslipidemia and obesity. Increased cardiovascular and other risk factor among hypothyroidism patients need to be addressed by further studies.

Keywords: Metabolic syndrome, Hypothyroidism, Cardiovascular risk.

INTRODUCTION

Metabolic syndrome is a cluster of risk factors characterized by hypertension, atherogenic dyslipidemia, hyperglycemia, prothrombotic and proinflammatory conditions.[1]

Thyroid function affects the parameters causing the metabolic syndrome, including low density lipoproteins, triglycerides, blood pressure and plasma glucose.[2]

The association between diabetes, obesity and hyperlipidemia is long known and has been termed "insulin resistance syndrome", syndrome" and metabolic syndrome by various researchers.[3]

The prevalence of thyroid dysfunction was reportedly more among women with metabolic syndrome.[4,5] In an Indian report, hypothyroidism was found to be associated with metabolic syndrome, with females being more at risk than males.[6]

Overt hypothyroidism is reported to be a recognized risk factor for atherosclerotic cardiovascular disease, hyperlipidemia, low-grade inflammation and hypercoagulability.[7,8] Therefore, the aim of study was to investigate the prevalence of metabolic syndrome among hypothyroid subjects and to assess cardiovascular risk factors in these patients.

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MATERIALS AND METHODS

The observational hospital based study was carried out in the department of medicine Katihar Medical College and Hospital, Katihar for a period of more than one and half year I, e. from November 2013 to July 2015 which was preapproved by the Ethical Committee of this institution review board. The patients attending the thyroid clinic as well as outdoor patients who fulfilled the diagnostic criteria as per WHO as well as ATP-III terms were included in the study. Based on thyroid profile, forty subjects and forty age-matched controls were included in the study. Any patient having significant renal disease, hepatic disease, or is immobile, or having a myocardial condition, or pregnancy was excluded from the study. After taking the history (especially of smoking, alcohol, physical activities, diabetes and hypertension), physical examination was conducted which include height, weight, BP, and abdominal girth. Then these patients were subjected to a complete haemogram with ESR, fasting blood sugar, complete lipid profile, ECG, fibrinogen level, T3, T4, TSH estimation and insulin level estimation. Metabolic syndrome was diagnosed when three or more of the following were present: Waist circumference more than 35 inch (women), Blood pressure > 130/85 mm Hg, plasma glucose > 100 mg/dL, triglycerides > 150 mg/dL, HDL-C < 50 mg/dl. Lipid profile of all participants was estimated by enzymatic methods[9,10] at day 0. Other routine investigation was also done. Data obtained were statistically analyzed using Student t test, assuming $P<0.05$ as significant.

RESULTS

Table 1: Clinical and biochemical characteristics of the hypothyroid and control groups.

Variables	Hypothyroidism Group(n=40)	Control Group(n=40)	" p" Value
Age	36.36±12.8	32.74±7.89	NS
BMI	27.64±3.6	26.12±2.8	0.03
Serum TSH(µIU/ml)	16.78±7.89	2.5±2.8	<0.0001
Serum T_3(nmol/ml)	1.1±0.6	2.9±2.68	<0.0001
Serum T_4(nmol/dl)	82.6±26.34	86.24±26.6	NS
Waist Circumference(cm)	84.1±11.3	79.65±10.6	NS
Systolic blood pressure(mmHg)	128.1±14.4	110.8±9.65	<0.0001
Diastolic blood pressure(mmHg)	86.1±10.6	78.1±8.6	<0.0004
Total Cholesterol(mg/dl)	179.8±32.89	156.83±24.66	<0.0007
Triglyceride(mg/dl)	167.6±48.62	102.8±28.7	<0.0001
LDL-C(mg/dl)	104.8±22.7	92.8±21.1	0.0166
HDL-C(mg/dl)	40.1±8.4	44.8±8.2	0.0133
Fasting glucose(mg/dl)	116.8±12.64	84.2±6.82	<0.0001
Insulin(µIU/ml)	9.06±6.52	11.12±6.54	NS
HOMA-IR	2.28±1.12	2.36±1.58	NS

"P" value<0.05 are considered significant. NS= Not significant

[Table 1] Shows Systolic and diastolic blood pressures in hypothyroidism patients were significantly higher than values in controls. Similarly, BMI, waist circumference was higher among hypothyroidism subjects. Again, fasting plasma glucose, total cholesterol, triglycerides and LDL-cholesterol levels were higher in patients of hypothyroidism in comparison to controls, while HDL- cholesterol ,insulin, HOMA-IR were higher among controls.

DISCUSSION

In our study, forty female subjects and age matched forty controls were included and study shows higher prevalence of hypertension, dyslipidemia and dysglycemia in hypothyroidism patients. The potential contributory role of the metabolic syndrome to cardiovascular risk and its scope in subjects with hypothyroidism was the focus of this study.

Abnormal lipid profile is an often-documented abnormality in thyroid disorders, and some reports[11] demonstrated that thyroid hormones influence LDL-C by various mechanisms, which include catabolism of LDL-C–independent alterations in metabolism, stimulation of the synthesis of cholesterol as well as the influence on biliary lipid metabolism. Well-documented lipid abnormalities in hypothyroidism include hypercholesterolemia and elevated LDL levels, but HDL-C levels may be normal or elevated in severe hypothyroidism.[12] Our study shows the prevalence of hypertension in hypothyroidism subjects which is similar to Saito *et al*.[13] Hypothyroidism is a potentially important but overlooked cause of hypertension, and possible pathophysiological mechanisms responsible for the occurrence of hypertension in hypothyroidism include changes in circulating catecholamines, their receptors and renin–angiotensin–aldosterone.[14] Higher BMI and central obesity were more common among hypothyroid subjects; central obesity has a role in the development of the metabolic syndrome and is reported to sometimes precede the appearance of other metabolic syndrome components.[15] Dysglycemia with higher fasting glucose, reduced concentration of insulin and HOMA-IR seen in hypothyroid subjects, which is similar to findings of Ohisen PM *et al*.[16] In subjects with hypothyroidism, insulin resistance is suggested as the possible underlying pathophysiological basis for glucose intolerance when present.[17] The metabolic syndrome, which is a set of lipid and non-lipid risk factors of metabolic origin linked to insulin resistance, is believed to be associated with an elevated risk for cardiovascular diseases.[18-20]

CONCLUSION

Our study revealed that hypothyroidism is significantly associated with metabolic syndrome and its components like dysglycemia, hypertension and dyslipidemia and obesity. Increased cardiovascular and other risk factor among hypothyroidism patients need to be addressed by further studies. Hypothyroidism and higher prevalence of metabolic syndrome among them needs careful assessment of risk factors by clinicians.

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Research Article

Study of etiological and clinical profile of pericardial effusion in a tertiary care hospital in Kosi region of Bihar, India

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ABSTRACT

Background: Pericardial effusion is one of the common diseases presenting in emergency and outpatient departments of a tertiary care center. Pericardial effusion can cause significant symptoms and diminished quality of life, but more importantly, is associated with increased risk of cardio respiratory failure, mortality and death. The etiology of pericardial effusion varies in different parts of the world and is related to the relative prevalence of different diseases.
Methods: This is a retrospective where data from all the cases diagnosed with pericardial effusion in the medicine department of KMCH from July 2014 to July 2015 were included. Altogether 66 cases diagnosed with pericardial effusion were established by Echocardiograpy. Evaluation for the cause of pericardial effusion included complete blood count with ESR, Blood urea, serum creatinine, tuberculin skin test, Chest X-ray, ECG, Thyroid profile, ANA, Rheumatoid factor, CT chest / MRI and pericardiocentesis. Pericardial fluid was analysed for cells, proteins, LDH, malignant cells, ADA, PCR (for mycobacterium tuberculosis), gram staining, AFB staining and cultures. Iatrogenic (cardiac surgery, catherterization) and post-traumatic cases and age <15 years were excluded.
Results: Majority of patients ware aged between 56-75 years. Thirty-five patients (53.03%) were male and 31 patients (46.96%) were female. Most common etiology of pericardial effusion was tuberculosis 27.27% followed by Idiopathic 19.69% then Uremia 16.66%, and Malignant 13.63%. The least common etiology of pericardial effusion was HIV infection 1.51%.The most common clinical feature was Tachycardia 69.69%, followed by Breathlessness 60.60% and fever was 54% of patients.
Conclusions: By this study, we have observed various presenting feature for pericardial effusion are tachycardia, shortness of breath, fever, heaviness of chest, cough, chest pain etc. The important disease factor for the occurrence of pericardial effusion such as tuberculosis, idiopathic/viral, uremic, neoplastic, CCF, hypothyroidism, post MI, etc.

Keywords: Pericardial effusion, Clinical features, Tachycardia, Etiology, Tuberculosis

INTRODUCTION

The pericardium is a fibrous sac surrounding the heart, the thicker, outer parietal pericardium and an inner, thinner visceral layer. Pericardium normally contains up to 50 ml of serous fluid within its two layers.[1] Pericardial fluid is an ultrafiltrate of the plasma, from the fibrous parietal pericardium and is resorbed by the lymphatics. The normal pericardium, by exerting a restraining force, prevent sudden dilation of cardiac chamber, especially the right atrium and ventricle, during the exercise and with hypervolemia.[2] The most common forms of pericardial diseases include acute and recurrent

pericarditis, isolated pericardial effusion with or without cardiac tamponade, and constrictive pericarditis.

Pericardial effusion is a relatively common finding in every day clinical practice. Pericardial effusion (PE) is the presence of an abnormal amount of fluid in the pericardial space. It is caused by a variety of local and systemic disorders, or may be idiopathic. Pericardial effusions can be acute or chronic. The cause of abnormal fluid production depends on the underlying etiology, transudative fluids result from obstruction to fluid drainage, which occurs through lymphatic channels. Exudative effusion occurs secondary to inflammatory, infectious, malignant or autoimmune processes within the pericardium. Clinical manifestations of pericardial effusion are highly dependent on the rate of accumulation of fluid in the pericardial sac. Rapid accumulation of pericardial fluid may cause elevated intrapericardial pressures with as little as 80 ml of fluid, while as slowly progressing effusions can accumulate upto 2 liters without symptoms.[3,4]

Commonly encountered causes of pericardial effusion are infectious/idiopathic pericarditis, malignancy, renal failure and collagen vascular disease. Pericardial effusion resulting from acute pericarditis of no more than 1 to 2 weeks duration is considered idiopathic. Most idiopathic cases are presumed to be of viral etiology, but testing for specific viruses is not routinely done because of the cost involved, low yield, and negligible impact on management.[5-7] In any case, the finding of cardiomegaly with clear lungs should raise the suspicion of a pericardial effusion. The echocardiogram is the most available and reliable technique in order to verify the presence and the amount of a pericardial effusion; in addition, the echocardiogram offers valuable data for evaluation of hemodynamic repercussion. In these cases, computed tomography (CT) is a reliable method to precisely identify the nature of this echocardiographic finding[8]. Mild pericardial effusion consider when echo free space is > 10 mm by M-mode echocardiography while the moderate effusions were defined as an echo-free space of anterior plus posterior pericardial spaces of 10-20 mm during diastole, and severe effusions as a sum of echo-free spaces > 20 mm.[9,10] In developed countries the different study show the following results such as commonest cause of pericardial effusion is neoplastc, idiopathic and uremic, but less common cause is infectious, collegen vascular disease and post MI.[10,11]

METHODS

The observational hospital based study was carried out in the department of medicine Katihar Medical College and Hospital, Katihar. This is a retrospective where data from all the cases diagnosed with pericardial effusion in the medicine department of KMCH from July 2014 to July 2015 were included. Data was taken from medical record section. Altogether 66 cases diagnosed with pericardial effusion was established by echocardiography, seen as echo free space of pericardial fluid more than 10 mm deep in front of the right ventricle and beyond the left ventricle.

Evaluation for the cause of PE included complete blood count with ESR, Blood urea, serum creatinine, tuberculin skin test, chest X-ray, ECG, thyroid profile, ANA, rheumatoid factor, CT chest/MRI and pericardiocentesis. Pericardial fluid was analysed for cells, proteins, LDH, malignant cells, ADA, PCR (for mycobacterium tuberculosis), gram staining, AFB staining and cultures. Final diagnosis was based on clinical history, examination, and specific laboratory investigations for tuberculosis, uraemia, malignancy, collagen vascular disease hypothyroidism etc. The diagnosis of acute idiopathic/viral etiology was presumptive and was based on the clinical picture, and negative screening tests for other etiologies. Therapeutic Echo-guided percutenaous pericardiocentesis was performed by placing pigtail catheter in pericardial space through subxiphoid approach. Iatrogenic (cardiac surgery, catherterization) and post-traumatic cases and age <15 years were excluded.

RESULTS

Table 1: Age wise distribution of patients.

Age in years	No of patients	Percentage
16-25	9	13.63
26-35	4	6.06
36-45	5	7.57
46-55	10	15.15
56-65	13	19.69
66-75	16	24.24
76-85	9	13.63

Table 2: Gender wise distribution of patients.

Sex	No of patients	Percentage
Male	35	53.03
Female	31	46.96

This study included 66 patients with age ranging from 16 to 85 years, Table-1 show majority of patients ware aged between 56-75 years (n=29, 44%) only 4 patients 6.06% admitted with pericardial effusion of the age group between 26-35 years. Thirty-five patients (53.03%) were male and 31 patients (46.96%) were female Table 2.

Table 3 show the most common etiology of pericardial effusion was tuberculosis (n=18; 27.27%), followed by Idiopathic (N=13; 19.69%) then Uremia (n=11; 16.66%), and Malignant (n=09; 13.63%). The least common etiology of pericardial effusion was HIV infection (n=01; 1.51%).

The most common clinical feature was Tachycardia (n=46; 69.69%), followed by Breathlessness (no=40;

60.60%) and fever was (no=36; 54%) of patients. The least common clinical feature was Hypotention (no=20; 30.30%) (Table 4).

Table 3: Distribution of pericardial effusion patients based on diagnosis.

Diagnosis	No of patients	Percentage
Tubercular effusion	18	27.27
Idiopathic/viral	13	19.69
Ureamia	11	16.66
Malingnant	09	13.63
CCF	05	7.57
Hypothyroidism	03	4.54
Collegen vascular disease	02	3.03
Post MI	02	3.03
Pyogenic	02	3.03
HIV infection	01	1.51

Table 4: Clinical presentation of patients of pericardial effusion.

Sign and symptoms	No of patients	Percentage
Tachycardia	46	69.69
Breathlessness	40	60.60
Fever	36	54.54
Heaviness of chest	31	46.96
Cough	30	45.45
Chest pain	23	34.84
Pulsesparadoxes	22	33.33
Hypotension	20	30.30

Table 5: Pericardial effusion patients presented with shortness of breath with different etiology.

Diagnosis	No of patients	Percentage
Tubercular effusion	13	72.22
Idiopathic/viral	06	46.15
Ureamia	09	81.81
Malignant	03	33.33
CCF	05	100.00
Hypothyroidism	01	33.33
Collegen vascular disease	00	00.00
Post MI	02	100.00
Pyogenic	01	50.00
HIV infection	00	00.00

Table 5 show, 13 patients (72.22%) of tubercular pericardial effusion out of 18 patients of pericardial effusion presented with shortness of breath, 6 patients (46.15%) of pericardial effusion due to idiopathy out of 13 patients presented with shortness of breath, all Five patients (100%) of pericardial effusion due to CCF and 2 patients of Post MI admitted with shortness of breath. No any patients of pericardial effusion due to collegen vascular disease and HIV presented with shortness of breath.

Table 6: Pericardial effusion patients presented with fever with different etiology.

Diagnosis	No of patients	Percentage
Tubercular effusion	16	88.88
Idiopathic/viral	07	53.84
Ureamia	03	27.27
Malingnant	06	66.66
CCF	01	20.00
Hypothyroidism	00	00.00
Collegen vascular disease	00	00.00
Post MI	00	00.00
Pyogenic	02	100.00
HIV infection	01	100.00

Table 6 shows that sixteen patients (88.88%) of Tubercular pericardial effusion out of 18 patients admitted with fever. Only one patients (20%) out of 5 patients of CCF presented with fever. All patients (100%) of pericardial effusion due to pyogenic and HIV admitted with fever. No any patients of collegen vascular disease, post MI and Hypothyroidism admitted with fever.

Table 7 shows, twelve patients (66.66%) of tubercular pericardial effusion admitted with cough. Only 2 patients (18.18%) of Uremic pericardial effusion out of 11 patients presented with cough. About 60% of CCF patients presented with cough, no any patients of collegen vascular and post MI patients presented with cough.

Table 7: Pericardial effusion patients presented with cough with different etiology.

Diagnosis	No of patients	Percentage
Tubercular effusion	12	66.66
Idiopathic/Viral	06	46.15
Ureamia	02	18.18
Malingnant	04	44.44
CCF	03	60.00
Hypothyroidism	01	33.33
Collegen vascular disease	00	00.00
Post MI	00	00.00
Pyogenic	01	50.00
HIV infection	01	100.00

DISCUSSION

Pericardial effusion can occur at any age but age specific etiologies may differ. In our study, Out of 66 patients of pericardial effusion, majority of patients ware age group of 56-75 years 43.99% (Table 1). This finding is consistent with poor and developing countries studies, but differ from some western studies. Due to low prevalence

of infecious disease and high prevalence of neoplastic disease. In their population there is no interpretation of sex with pericardial effusion in this population.

In develop countries, The finding of a pericardial effusion in patients with underlying malignancy creates a more complex dilemma, as not infrequently pericardial effusion is due to alternative causes and not to direct neoplastic pericardial involvement. In Posner's series[12] malignant pericardial disease was diagnosed in 18 (58%) of 31 patients with underlying cancer and pericarditis, while 32% of the patients had idiopathic pericarditis and 10% had radiation induced pericarditis. Porte et al[13] studied 114 patients with recent or remote history of cancer and a pericardial effusion of unknown origin requiring drainage for diagnostic or therapeutic purposes. Pericardioscopy was performed in 112 patients with pericardial fluid analysis and biopsy of abnormal structures or deposits under direct visual control. Malignant pericardial disease was found in 44 (38%) patients, while 70 (61%) patients had non-malignant pericardial effusions, Idiopathic in 33 patients, radiation-induced in 20 patients, infectious effusion in 10 patients, and hemopericardium as a result of coagulation disorders in 8 patients.

Corey et al[14] investigated the etiology of pericardial effusion in 57 patients.

An etiologic diagnosis was made in 53 patients (93%). The most common diagnoses were malignancy (23%), viral infection (14%), radiation–induced inflammation (14%), collagen-vascular disease (12%) and uremia (12%).

The study by Sagristà- Sauleda et al[10] included 322 patients, 132 with moderate and 190 with severe pericardial effusion. In this series, the most common diagnosis was Idiopathic-20%, Neoplastic-13%, Post MI-8%, uremia-6%, collegen vascular disease-5%, Tubercular-2%.

But in our study, the commonest cause of pericardial effusion was infectious, Tubercular 18 patients (27.27%), idiopathic/viral 13 patients (19.69%), but Neoplastic cause is only 13.63%. There is no any case of pericardial effusion due to radiation.

The clinical features that led referring physicians to order the echocardiographic study included dyspnea in 44 patients (83%), pleuritic chest pain in 22 (42%), cough in 5 (9%) and hypotension in 2 patients. At physical examination systolic blood pressure was higher than 100 mmHg in 94% of patients, elevation of the jugular venous pressure was suspected in only 74%, hepatomegaly was present in 28%, and pulsus paradoxus was appreciated in only 36% of patients.[15]

In our study, the most common clinical feature was tachycardia (n=46; 69.69%), followed by breathlessness (no=40; 60.60%) and fever was (no=36; 54%) of patients. The least common clinical feature was hypotension (no=20; 30.30%).

CONCLUSION

By this study, we have observed various presenting feature for pericardial effusion are tachycardia, shortness of breath, fever, heaviness of chest, cough, chest pain etc. The important disease factor for the occurrence of pericardial effusion such as tuberculosis, idiopathic/viral, uremic, neoplastic, CCF, hypothyroidism, post MI, etc. This study would help in early diagnosis and prompt treatment of patients with pericardial effusion especially in remote areas which remains a challenging problem for diagnosis and treatment. More detailed epidemiologic studies are required to improve understanding of the burden of pericardial effusion.

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Research Article

Clinical and etiological profile of acute kidney injury in cases attending a tertiary care center

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ABSTRACT

Background: Acute kidney injury or acute renal failure is a common problem in hospitalized patients. A large number of conditions can cause AKI which can present with different clinical features. The aim of present study was to analyze etiological and clinical features of AKI.
Methods: This study involved 68 patients of AKI admitted in a teaching hospital. The clinical and laboratory data were collected at admission and during follow up till the final outcome and these data were analyzed.
Results: 68 patients were studied. Largest no of patient was in the age group of 41-50 years and 55.88% of patients were male. The causes of AKI were medical (77.94%), surgical (11.76%) and obstetrical (10.29%). Sepsis was the most common medical cause (41.50%). Patients were treated either by conservative methods or by haemodialysis. Overall mortality was 20.58%.
Conclusions: Non-oliguric AKI was more common but oliguric AKI has high mortality. The mortality in dialysis treated patients is high than patients who are treated conservatively. The high mortality in dialysis treated patients is due to multi organ failure.

Keywords: Acute kidney injury, Clinical, Etiological, Oliguric, Profile

INTRODUCTION

Acute Renal failure is characterized by sudden reduction in renal function resulting in retention of nitrogenous and other waste products in the body.

Due to usage of more than 30 definitions of ARF in literature, there is wide variations in reported incidence and outcome, the term ARF was recently replaced by acute kidney injury with a view to provide uniform definition and to standardise patient care.[1]

AKI is not a single disease but rather a heterogenous group of conditions that share common diagnostic features specifically an increase in blood urea nitrogen (BUN) concentration and/or increase in serum creatinine concentration often associated with a reduction in urine volume. Reduction of urine volume to less than 400 ml in 24 hours is called oliguria. Oliguria is a frequent but not invariable feature.[2]

AKI is defined as any of the following,[3]

1. Increase in serum creatinine by 0.3 mg/dl within 48 hours or
2. Increase in serum creatinine to 1.5 times baseline value which is known or presumed to have occurred within prior 7 days or

3. Urine volume to less than 0.5 ml/kg/hour for 6 hours

Kidney is unique among the organ of the body in its ability to recover from almost complete loss of function.[4] AKI is reversible when recognized and managed early.[5] Delay in diagnosis may lead to increased mortality and morbidity.[6]

Acute kidney injury is sub classified into three categories

Pre renal, intrinsic renal disease and post renal. Pre renal failure is the most common form and is characterized by renal hypoperfusion without compromising the integrity of renal parenchyma. Intrinsic renal failure is produced by disorders that directly involve renal parenchyma i.e due to ischemia, various nephrotoxins and diseases of glomeruli or interestitium. Post renal failure is due to urinary tract obstruction.[7]

Advanced age, liver disease, underlying renal insufficiency and diabetes have been implicated as risk factors for the development of AKI.[8]

The incidence of AKI in hospitalised patients is 2-5%.[8] The high mortality is due to sepsis, respiratory failure and multi organ dysfunction.

AKI tells the gravity of underlying disease and not the cause of death.[9] The present study was done to describe various etiological and clinical features of AKI in a teaching hospital of north Bihar.

METHODS

Material and methods-this was hospital based study of patients with AKI admitted to Katihar medical college hospital, Katihar from October 2013 to September 2014. Patients of more than 15 years of age who presented with oliguria or anuria for more than 24 hours with rise in blood urea and serum creatinine concentration or rise in serum creatinine concentration more than 50 percent over normal values were selected for the study. The exclusion criterion was patients of less than 15 years of age, with underlying CKD or bilaterally small kidneys.

All patients were enquired about nausea and/or vomiting, breathlessness, loose stools, fever, any bleeding, yellowish discolouration of sclera, change in colour of urine or any difficulty in passing urine during micturition.

History regarding current or past use of medications, any underlying chronic disease or hepatits/jaundice in past, was taken.

After history taking all patients were clinically evaluated. Routine and microscopic examination of urine, complete blood count, blood urea, serum creatinine, serum electrolytes and ultrasonography of whole abdomen was done in all patients. Arterial blood gas analysis was done in selected patients when required.

Table 1: Age distribution of patients of AKI.

Age	No of PTS	Percentage (%)
16-20	3	4.41
21-30	9	13.23
31-40	13	19.11
41-50	16	23.52
51-60	11	16.17
61-70	12	17.64
>70	4	5.88

Table 2: Sex distribution of patients of AKI.

Sex	No of patients	Percentage
Male	38	55.88
Female	30	44.12

Out of 68 patients of AKI studied male to female ratio was 1.26:1.

Table 3: Etiology of AKI.

Etiology	No of patients	Percentage
Medical	53	77.94
Surgical	8	11.76
Obstetrical	7	10.29

Table 4: Medical causes of AKI.

Causes	No of cases	Percentage
Sepsis	22	41.50
Acute gastroenteritis	13	24.53
Malaria	8	15.09
CCF	4	7.54
Acute glomerulonephritis	2	3.77
Chronic liver disease	2	3.77
Drug toxicity	1	1.88
Organophosphorus poisoning	1	1.88

All patients were followed up with daily input/output charting and regular blood urea and serum creatinine concentration.

Patients were treated either by conservative method or haemodialysis. The indications for haemodialysis were encephalopathy, metabolicacidosis, hyperkalemia, oliguria/oedema not responding to diuretics rising blood urea and/or serum cretinine concentration and uremic pericarditis.

The outcome of treatment was noted for all patients.

Table 5: Surgical causes of AKI.

Cause	No of patients	Percentage
Post-operative	4	50.00
orthopaedic surgery	3	37.50
Urinary tract obstruction	1	12.50

Table 6: Obstetrical causes of AKI.

Cause	No of patients	Percentage
Puerperal sepsis	5	71.42
Pre eclampsia	2	28.57

Table 7: Relation of type of AKI to mortality.

Type of AKI	Total No of patients	Survival	Death
Oliguric	31 (45.58)	20 (64.51)	11 (35.49)
Non oliguric	37 (54.42)	34 (91.89)	3 (08.11)

Figure in ( ) indicates percentage, Chi sqare=7.73 p<0.05(Significant), oliguric AKI has high mortality.

Table 8: Relation of Treatment modality to mortality.

Mode of treatment	No of patient	Survival	Death
Conservative	46(67.64)	38(82.60)	8(17.40)
Dialysis	22(32.36)	16(72.72)	6(27.28)

Figure in ( ) indicates percentage, Chi sqare=3.20 , p <0.05 (Significant) , Hgh mortality present in patients treated with dialysis.

Table 9: Clinical features of AKI patients.

Symptoms and sign	No of patients	percentage
Vomiting	47	69.11
Dyspnoea	42	61.76
Oliguria	31	45.58
Loose stools	16	23.52
fever	33	48.52
Jaundice	14	20.58

Table 10: Complications of AKI.

Complications		Percentage
Metabolic acidosis	13	19.11
Hyperkalemia	29	42.64
Encephalopathy	12	17.64
Hypotension	19	27.94
Multi organ dysfunction	17	25.00
Pulmonary oedema	26	38.23
Bleeding tendency	3	4.41

RESULTS

The study included 68 patients of AKI between 15-75 years of age group.

Maximum numbers of patients were in the age group of 41-50 years. The male to female ratio was 1.26:1. Causes leading to AKI were medical (77.94%), surgical (11.76%), and obstetrical (10.29%). Among medical causes most common cause was sepsis (41.50%) followed by hypovolemia due to gastroenteritis (24.53%). Postoperative lack of care was the most important cause of AKI in the surgical group. Puerperal sepsis and pre eclampsia were the two conditions associated with AKI in obstetrical cases. Patients were treated either conservatively or by haemodialysis. 46 patients (67.64%) were treated conservatively and 22 patients (32.36%) were treated by haemodialysis. Out of 46 patients who were treated conservatively 38 (82.6%) survived and 8 (17.40%) expired. Out of 22 patients who were treated by haemodialysis 16 (72.7%) survived and 8 (27.28%) expired. Out of 68 patients 31 (45.58%) had oliguria 78.57% of death was due to olguric AKI. Among the 54 patients who survived 38 (70.3%) were treated conservatively and 16 (29.7%) were treated by haemodialysis. Out of 14 patients who expired 8 (57.14%) were treated conservatively and 6 (42.86%) were treated by haemodialysis.

Vomiting was the most common symptom reported in 69.11% of patients followed by breathlessness in 67.76%. Fever was present in 48.52% of patients. Hyperkalemia was the most common complication in 42.64% of patients. Multi organ dysfunction was present in 23.52% of patients.

DISCUSSION

Acute kidney injury is a very common condition in hospitalised patients. This study included 68 patients of AKI studied over one year period. Barret et al reported 200 patients of ARF in three year period.[10]

In this study cause of AKI was medical (77.94%), surgical (11.76%) and obstetrical (10.29%). Liano et al has grouped ARF into medical (34%), ICU (27%), surgical (23%), obstetrical (1%), nephrological (13%) and traumatic(2%).[11] Saxena et al in his review classified etiology of ARF into medical (75%), obstetrical (15%), obstructive and surgical (10%).[12] Naqvi et al reported medical cause (57%) followed by obstetrical (24%), obstructive (7%), surgical (5%) and undetermined cause (7%).[13] These differences in etiology is due to the fact that infectious diseases are less prevalent in developed countries but are still a significant problem in developing countries. In developed countries sepsis and multi organ failure is the commonest cause and most cases of AKI occur in older age group due to presence of comorbid illnesses. In developing world due to poor sanitation and unhygienic practices infectious diseases like gastroenteritis and malaria is more prevalent. AKI is more common in younger age group in healthy individuals in developing countries.

Mortality in ARF in hospitalized patients is reported from 14-70% in different studies.[10] Mortality in this study was 20.58%.

Non olguric AKI was more common in this study. Oliguric cases had high mortality in this study. Non olguric AKI is recognized more frequently and causes less mortality and morbidity and dialysis is required less often.[10] Dialysis is required in oliguric cases more frequently. Obialio and associates reported oliguria as a factor causing high mortality and morbidity.[10] In this study patients who were treated with dialysis had high mortality. This high mortality was due to association of multi organ failure in those cases who required dialysis.

CONCLUSION

AKI is a common problem in hospitalized patients. Early diagnosis and management is essential. Early and aggressive management of sepsis, restoration of intravascular volume and avoidance of nephrotoxic medications reduces mortality due to AKI.

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A correlation study for detection of Left Atrial Enlargement of patients with cardiac and non cardiac disease: A hospital based study

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Abstract
Objective: *Our study was to detect the prevalence and correlation of echocardiography and ECG finding of left atrial enlargement of patients with cardiac and non cardiac disease.*
Methodology: *A total of 75 patients were included on the basis of inclusion criteria. A detail assessment was taken to all patients. A standard 12 lead ECG was recorded and Echocardiography was performed in all the cases. Left atrial size was measured by 2D echo guided M-mode echocardiography.*
Results: *Data was analyzed by using MS-Office software.*
Conclusions: *Patients with RHD with mitral and aortic valve disease, hypertension, atrial fibrillation and IHD were more associated to left atrial enlargement (LAE). 2 D echocardiography was able to categorize LAE into mild, moderate and severe. ECG was only able to predict LAE. Hence echocardiography is superior than electrocardiography for detection of LAE.*
Keywords: *Left atrial enlargement, echocardiography, electrocardiography.*

Introduction

Detection of left atrial enlargement or its progression is frequently important in clinical medicine [1]. Left atrium is affected directly by increased ventricular filling pressure, increased resistance across mitral valve or volume overload. [2] Left atrial enlargement is an important pathologic change in many forms of heart disease [3].

Left atrial enlargement is an important pathologic change in many forms of heart disease.[3] detection of left atrial enlargement or its progression is frequently important in clinical medicine.[1] There is growing recognition of the importance of left atrial enlargement and its association with increased morbidity and mortality in patients with cardiovascular disease [4]. The left atrium is affected directly by increased ventricular filling pressure, increased resistance across the mitral valve, or volume overload [2]. Left atrial enlargement occurs in various conditions like mitral valve disease, aortic valve disease, combined valvular lesions. Hypertension, ischemic heart disease, mitral valve prolapsed, cardiomyopathies, congenital heart disease, and pericardial effusion [2]. Left atrial enlargement

can be mild, moderate or severe depending on the extent of the underlying condition. Although other factors may contribute, left atrium size has been found to be a predictor of mortality due to both cardiovascular issues as well as all-cause mortality. Current research suggests that left atrium size as measured by an echo-cardiograph may have prognostic implications for preclinical cardiovascular disease. However, studies that have found LAE to be a predictor for mortality recognize the need for more standardized left atrium measurements than those found in an echo-cardiogram. [17]

Many numbers of studies has shown that a chronic hemodynamic burden initially produces atrial dilatation and structural damage to atrial wall; this in turn increases likelihood of the development of atrial fibrillation. Once atrial fibrillation is present, atrial dilatation could progress a consequence of continued hemodynamic burden, the less of atrial systole or both. [6] Left atrial enlargement has been found to be a significant predictor of recurrent and chronic atrial fibrillation[7]. ECG assessment of left atrial enlargement is a noninvasive and universally available method[8]. Echocardiography has proven to be a valuable non-invasive tool for quantitatively assessing left atrial size [6]. Aim of this study was to detect the left atrial enlargement by echocardiography and ECG correlation in cardiac and noncardiac diseases patients.

Materials and Methods

Data was collected using random sampling method on the basis of inclusion and exclusion criteria, with irrespective of sex in OPD or the ward, of department of Medicine, Katihar Medical College, Katihar, Bihar during period of January 2017 to June 2017.

This prospective correlation study was conducted on 75 patients aged between 14-80yrs with clinically suspected left atrial enlargement. The attendant of entire subject/patients signed an informed consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar, India was sought. LAE has close relationship with atrial fibrillation, systemic thromboembolism events and heart failure. [3] ECG assessment of left atrial enlargement is a noninvasive and universally available method. [8] ECG analysis for left atrial enlargement includes configuration, amplitude and duration P wave [9]. Echocardiography LA dimension is the best noninvasive test of left atrial size [10].

Inclusion criteria of this study was Rheumatic mitral and aortic valvular diseases, Isolated aortic valve diseases, Hypertension, Coronary artery diseases (IHD), Mitral valve prolapsed, Cardio-myopathy, Thyrotoxicosis, Atrial fibrillation. Exclusion criteria was patients with Age < 14 years, pericardial effusion, Chronic obstructive pulmonary diseases (COPD), Chest trauma

Methods

A detail assessment was taken to all patients. It includes detailed history, thorough general physical examination, systemic examination and investigations like 12 lead ECG and echocardiography. Patients having P wave ECG changes in lead II and V_1 submitted for echocardiography evaluation over a period for 6 months.

Electrocardiogram

A standard 12 lead ECG was recorded in all patients and was analyzed for evidence of left atrial enlargement and/or AF.

Echocardiography

Echocardiography was performed in all the cases. Left atrial size was measured by 2D echo guided M-mode echocardiography. Measurements were obtained as per the recommendations of American society of echocardiography. Left atrium was measured at end systole in parasternal long axis as a maximum distance between the anterior margin of posterior aortic root echo and the anterior margin of a posterior wall of left atrial echo at the aortic valve levels.

Statistical analysis

Simple analysis method was taken to analyzed the data with the help of MS-Office soft ware.

Observations

This study was carried out on 75 patients with left atrial enlargement OPD and ward of department of Medicine, Katihar Medical College, Katihar, Bihar, India.

In the present study, age of the patients were taken from 17 years to 80 years. There were 45 males and 30 females. In age group of 14-20 years, 2(4.44%) were male and 2(6.66%) females. In age group of 21-30 years, 4(8.89%) were males and 2(6.66%) females. In age group of 31-40 years, 5(11.11%) were males and 5(16.66%) females. In age group of 41-50 years, 11(24.44%) were males and 11(36.66%) females. In age group of 51-60 years 11(24.44%) were males and 02(6.66%) females. Above of age 60 years, 12(26.67%) were males and 08(26.66%) females.

In this study, 21(28%) cases were with rheumatic mitral and valvular disease. 14(18.66%) cases were with hypertension. 10(13.33%) cases were with ischemic heart disease (IHD). 8(10.66%) cases were with isolated aortic valve disease. 2(2.66%) cases were with mitral valve prolapsed. 4(5.33%) cases were with cardiomyopathies. 2(2.66%) cases were with thyrotoxicosis. And 14(18.66%) cases were with atrial fibrillation with or without rheumatic heart disease (RHD).

In this study, 41(54.66%) patients were symptoms with breathlessness. 24(32%) patients were with symptoms of chest pain. 13(17.33%) patients were with symptoms of cough. 23(30.66%) patients were with palpitation. 18(24%) patients were with symptoms of swelling of lower limbs. 16(21.33%) patients were with symptoms of easy fatigability. 5(6.66%) patients were history of neurological deficits. 5(6.66%) patients were with symptoms of hemoptysis. And 10(13.33%) patients were with symptoms of syncope.

In the present study, 40 (53.33%) patients had left atrial size in the range 39 to 45 mm followed by 17(22.66%) patients in the range of 46 to 50 mm, 08 (10.66%) patients in the range 51-55mm, 06(8%) patients in 56-60 mm and 4(5.33%) in the range ≥61mm. In the study, LA size varied from 40mm to 76mm.

In this study, least number of patients was in the age group of 14-20 years (6 patients). Among them 02(33.33%) had mild, 04(66.66%) had moderate LAE. 7 patients were in age group of 21-30 years. Among them 04(57.14%) had mild, 1(14.28%) had moderate and 2(28.57%) had severe LAE. 9 patients were in age group of 31-40 years. Among them 05(55.55%) had mild, 3(33.33%) had moderate and 1(11.11%) had severe LAE. 24 patients were of 41-50 years age group. Among them 08(33.33%) had mild, 8(33.33%) had moderate and also 8(33.33%) had severe LAE. 13 patients were of 51-60 years age group. Among them 06(46.15%) had mild, 2(15.38%) had moderate and 5(38.46%) had severe LAE. 16 patients were of Age above than 60 years. Among them 8(50%) had mild, 6(37.5%) had moderate and 2(12.5%) had severe LAE.

In this study 45 patients were male. Among them 24(53.33%) had mild, 10(22.22%) had moderate and 11(24.48%) had severe left atrial enlargement. 30 patients were females. Among them 08(26.66%) had mild 14(46.66%) had moderate and 08(26.66%) had severe left atrial enlargement.

In this study of total 75 patients. LAE was seen in 22 patients of RHD with mitral and aortic valve disease. Among them 15(68.18%) had mild, 05(22.72%) had moderate and 02(9.1%) had severe LAE. 15 patients were seen with hypertension. Among them 7(46.66%) patients had mild, 5(33.33%) had moderate and 3(20%) had severe LAE. 11 patients were seen with IHD. Among them 4(36.36%) patients had mild, 7(63.63%) had moderate LAE. 6 patients were seen with isolated aortic valve disease. Among them 3(50%) patients had mild, 2(33.33%) had moderate and 1(16.67%) had severe LAE. 14 patients were seen with AF. Among them 1(7.14%) patients had mild, 3(21.43.33%) had moderate and 10(71.43%) had severe LAE. 3(100%) patients with cardiomyopathies were seen with LAE. 2 patients were seen with thyrotoxicosis. Among them 1(50%) patients had

mild, 1(50%) had moderate LAE. 2(100) patients with mitral valve prolapsed were seen mild LAE.

In this present study, out of 13 patients of AF all 13 (100%) had shown ECG characteristics of AF like absent P wave and varying RR interval. P duration in lead II > 0.11 seconds was seen in 45(82.46%) patients. Morris and Macruz index characteristics were seen in 39(77.12%) of patients each. P wave notch duration > 0.04 seconds was seen also in 39(77.12%) of patients.

In this study ECG predicted overall 76 % positivity in LAE. But when AF alone was considered, 100 % ECG features of AF were seen. ECG positivity were seen in 100% in AF and cardiomyopathy followed by 72.72% in RHD with mitral and aortic valve disease, followed by hypertension(69.23%), isolated aortic valve disease (71.42%). IHD, MVP and thyrotoxicosis had shown 50% positive prediction. Sensitivity of ECG for detecting LAE-100%. But specificity - 75%.

Positive correlation between ECG and Echo in relation to disease findings: In this study, Echo was able to identify all 75(100%) patients of LAE, where as ECG detected only 55(76%) patients of LAE.

ECG wise prediction of LAE in Echo graded LAE: In this study, out of 75 patients, ECG detected LAE in 18(90.71%) of severe, 1(78.26%) of moderate and18(56.25%) of mild LAE patients. This is significant difference of ECG +ve and –ve prediction of patients with LAE.

Relation between LA size and congestive cardiac failure: Out of 75 patients, 30(40%) of patients shown features of CCF. Among them 14(46.67%) were severe LAE, 9(30%) were moderate LAE and 7(23.33%) were mild LAE.

In this study of 75, 10 patients with neurological deficits were LAE. Among them 3(30%) patients had moderate and 7(70%) had severe LAE.

In this present study, out of 75 patients, mortality was seen in 9(12%) patients. Among them 07(77.77%) were with severe LAE and 2(22%) were with moderate LAE.

Table.1. ECG and Echo correlation of patient's with different conditions

Conditions	ECG			ECHO			Total	
	+ve prediction	-ve prediction	% of positivity	+ve prediction	-ve prediction	% of positivity	No	%
RHD with mitral & aortic valve disease	16	6	72.72	22	0	100	22	100
Hypertension	9	4	69.23	13	0	100	13	100
IHD	5	4	55.59	9	0	100	9	100
Isolated aortic valve disease	5	2	71.42	7	0	100	7	100
Atrial fibrillation	16	0	100	16	0	100	16	100
Cardiomyopathy	4	0	100	4	0	100	4	100
Thyrotoxicosis	1	1	50	2	0	100	2	100
Mitral valve prolapsed	1	1	50	2	0	100	2	100
Total	57	18	76	75	0	100	75	100

Discussion

Enlargement of the left atrium is well known as one of the earliest manifestations of rheumatic mitral valvular dysfunction. On rare occasions this chamber may become enormous, expanding to the right and posteriorly to form a huge sac that encroaches upon adjacent structures and may eventually rest against the right chest wall. The clinical course of patients who develop such extreme left atrial enlargement is remarkably uniform, and the diagnosis can be suspected from the presence of a number of characteristic symptoms and signs. [5]

In our study age of patients ranged from 17 years to 90 years. There were 45(60)% males and 30(40%) females with a ration 1.5:1 showing male

preponderance. Wagner Ad et al. [16] in their study involving 339 patients, 58.99% were females and 41.01% were males. In a study by Levy et al [33] F: M was 1.3:1. So this study is comparable to Levy et al study.[12]

Rheumatic mitral and aortic valve disease was the most common cause of left atrial enlargement accounting for 28% in this study. Next in this order were atrial fibrillation and hypertension 18.66%, IHD 13.33%, isolated aortic valve diseases 10.66%, cardiomyopathies 05.33%, MVP and thyrotoxicosis each accounting for 2.66%. Hamid Ikram et al [1] found rheumatic mitral and aortic disease as the most common cause of left atrial enlargement (48.64%), next in this order was hypertension (7.08%).

In this study, left atrial size was varied from 40 mm to 76 mm. AF was more common (53.33%) when left atrial enlargement was 39-45 mm. Kulkarni AG et al [11] has shown in their study 97.14% of population had LA size > 40mm. In a study by Levy et al [12] patients with AF had left atrial size of 43.8±8.6mm. The findings of present study are comparable to the above mentioned studies.

In this study, out of 22(29.33%) rheumatic mitral and aortic valve disease patients, 68.18% of them were with mild LAE with mean left atrial size 44.75mm. 18.66% patients were of atrial fibrillation, among them 71.43% had severe LAE with mean left atrial size 58mm. 14.66% patients were of IHD, among them 63.63% had moderate LAE with mean left atrial size of 44.62mm. There were 20% patients of hypertension, of them 46.66% were mild LAE with mean LA size of 40.72mm. 4% were of cardiomyopathy, All of them were of severe left atrial enlargement with a mean LA size of 56.33mm. 2.66% cases were of thyrotoxicosis, of the 50% each of mild and moderate LAE with a mean LA size 42mm. 8% were of isolated aortic valve disease of which 50% were mild LAE with mean LA size 44.83mm. 2.66% patients were of mitral valve prolapsed, all of them were of mild left atrial enlargement with mean LA size of 43mm. In study of Levy et al. [12] Cardiomyopathy accounted for 5.06% of cases with a mean LA size of 43.8±8.6 mm. Coronary artery disease accounted for 16.3% of patients with mean left atrial size of 43.8±8.6mm. Hypertensive heart disease accounted for 21.44% patients with a mean LA size of 43.8±8.6mm.

Howad DC et al [1] IHD patients accounted for 23 % cases of atrial fibrillation with mean left atrial size of 47±8mm. In a study Papazolgloue NM et al [13] hypertension accounted for 22% of patients. Cardiomyopathy accounted for 11% of cases. In a study by Iwasaki T et al [14] of thyrotoxicosis patients, mean age was 54.1±8.2 years with mean LA size of 42.8±3.6mm. In a study by Owen R Brown et al [15] isolated aortic valve disease formed 23.52% of cases. In a study by Raul Chirife et al [2] mitral valve prolapsed formed 10.41% of cases of left atrial enlargement.

In this present study, out of 13 patients of AF all 13 (100%) had shown ECG characteristics of AF like absent P wave and varying RR interval. P duration in lead II > 0.11 seconds was seen in 45(82.46%) patients. Morris and Macruz index characteristics were seen in 39(77.12%) of patients each. P wave notch duration > 0.04 seconds was seen also in 39(77.12%) of patients.

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patients. This is significant difference of ECG +ve and –ve prediction of patients with LAE.

In a study by Raul Chirife et al[2] shown ECG positivity of 89%, when p wave duration was considered alone, 83% when Morris index considered alone and 89% when Marcuz index alone was considered. Alan D Waggoner et al [16] over all ECG predictability of left atrial enlargement was 80%. And showed 100% positivity when LA size>50 mm, 70.83% positivity when LA size was 46 to 50mm and 56.41% when LA size was 41to45mm. So our study is comparable to these studies.

In this study, out of 75 patients, 10 patients with neurological deficits were LAE. Among them 7(70%) had severe LAE. Mortality was seen in 9(12%) patients. Among them mortality 07(77.77%) were with severe LAE.

So that echo was found to be more specific investigation to detect LAE compared to ECG. Echo was non invasive simple technique to detect positive LAE than ECG. As LA enlarged from mild to severe grade, ECG predictability of LAE was also increased (90.71% in severe LAE group). In the study it was found that complications of LAE like CCF, AF, Embolic stroke and mortality were increased as size of LA increased. Thus early detection of LAE using ECHO and ECG will help in reducing further complications.

Future Research

Science is dynamic and there is always a scope of improvement and change in time to come ahead. With progressive aim to move ahead we aspire to achieve highly accurate and reliable results. Thus every study leaves back scopes for other researcher to do something more advanced and varied in order to touch the height of perfection. This study examined only 75 subjects (45 males and 30 females), future researchers can expand the study by including more number of subjects so as to make generalization of the results and practice, further studies with a larger sample size and in multiple centers are required. Thus it could be applied to real life situation.

Limitation

There were several limitations like, the sample size was small, and it was a hospital-based study, the prevalence of exposure and outcome variables may be different from a community setting.

Conclusion

This study concluded that patients with rheumatic heart disease with mitral and aortic valve disease, hypertension, atrial fibrillation and ischemic heart disease were more prevalence to left atrial enlargement (LAE). 2 D echocardiography was able to categorize LAE into mild, moderate and severe. ECG was only able to predict LAE, it could not categorize LAE. So that 2 D echo is superior to ECG. Congestive cardiac failure, atrial fibrillation, neurological deficits (stroke) and mortality were associated with severity of LAE. So that, early detection of LAE is need for appropriate management and prevention of complications.

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ORIGINAL RESEARCH ARTICLE

An Observational Study on the Cardiovascular Abnormalities of Patients with Chronic Renal Failure

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Abstract:

Chronic renal failure is defined as either kidney damage or glomerular filtration rate less than 60 ml/min for three months or more. This is invariably a progressive process that results in end stage renal disease. Today, cardiovascular complications are a major clinical problem in ureamic patients accounting for 44% of all death in this population.

Objective: Present study was to observe the risk factors and cardiovascular abnormalities of patients with chronic renal failure. Methodology: A total of 110 patients with chronic renal failure were included on the basis of inclusion criteria. A details assessment and investigation were performed. Creatinine clearance was calculated by Cockcroft Gault equation. Data was analyzed by using MS-Office software.

Conclusions: Diabetes with hypertension was major risk factors of chronic renal failure. And chronic renal failure patients had higher prevalence of left ventricular hypertrophy.

Key words: chronic renal failure, cardiovascular abnormalities

Introduction:

Chronic renal failure is defined as either kidney damage or glomerular filtration rate less than 60 ml/min for three months or more. This is invariably a progressive process that results in end stage renal disease [1].

End stage renal disease and cardiac disease seem to be inextricably linked. Of various causes, infection and cardiovascular events contribute towards large proportion of increased morbidity and mortality [2].

As early as 1827 Richard Bright drew attention to the common presence of left ventricular hypertrophy and thickening of the aortic wall in patients with end stage renal disease [3]. Today, cardiovascular complications are a major clinical problem in ureamic patients accounting for 44% of all death in this population [3,4].

Death from cardiac causes is 10-20 times more common in patients with renal failure than in matched segements of the general population.

Left Ventricular Hypertrophy (LVF) is a major echocardiographic finding in Chronic Renal Failure (CRF) [5]. LVH is an independent predictor of survival, present in approximately 70% of patients at the initiation of dialysis [6]. Aims of our study were to observe the prevalence of cardiovascular disease in chronic renal failure patients and to aid in prompt diagnosis and effective management of cardiovascular complications in chronic renal failure cases.

Method and Materials:

Data was collected on the basis of inclusion and exclusion criteria, with irrespective of sex in OPD or the ward, of department of Medicine, Katihar Medical College, Katihar, Bihar during period of July 2016 to December 2016.

This prospective correlation study was conducted on 110 patients aged between 31-80 years with clinically suspected chronic renal failure.

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Subject: Medical Sciences

The attendant of entire subject/patients signed an informed consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar, India was sought

Inclusion criteria:

The study population consists of patients with mild, moderate and severe chronic renal failure attending the Hospital. Mild chronic renal failure: includes patients with serum creatinine 1.5-3 mg/dl. Moderate chronic renal failure: includes patients with serum creatinine values 3.0-6.0 mg/dl. Severe chronic renal failure: includes patients serum creatinine values > 6.0 mg/dl.

Exclusion criteria: Patients with other cardiac disorder such as valvular heart disease, congenital heart disease. All pediatric cases of chronic renal failure.

There were several investigations: 1. complete haemogram 2. Renal function test 3. Liver function test 4. Urine analysis and culture (if required) 5. Renal ultrasound 6. Lipid profile 7. Serum electrolytes, serum calcium, serum phosphorous 8. Chest radiography 9. Electrocardiography -12 lead 10. 2 D echocardiography.

Normal value of creatinine clearance for men- 90-140 ml/minute and for woman 80-135 ml/minute.

The creatinine clearance was calculated by the Cockcroft Gault formula [7]:

[140 – age (years)] × body weight (kg) / [0.815 × pla sma creatinine (μmol/l)].

For women, the correction factor of 0.85 was used.

All clearances were expressed as ml/min/1.73 m^2 after correction for body surface area (BSA) according to the DuBois–DuBois formula [8]:

BSA (m2) = 0.007184 × height (cm)

0.725 × weight (kg)0.425.

The creatinine concentration in plasma was measured with an enzymatic PAP+ (phenol/4-aminoantipyrine) assay on a Hitachi 747 analyser (Roche Diagnostics, Mannheim, Germany). The upper limit of the creatinine reference range was 110 μmol/l for males and 95 μmol/l for females [9].

Statistical analysis:

Simple analysis method was taken to analyzed the data with the help of MS-Office soft ware.

Results & Observations:

The present study comprised 110 patients (37 females and 73 males) of chronic renal failure admitted patients visiting OPD and patient undergoing dialysis in dialysis unit of Katihar medical college, Katihar, Bihar, India during the period from July 2016 to December 2016.

In this study, age variation was from 31 to 80 years. 6(5.45%) patients were belong from age group 31-40 years. 18(16.36%) patients were belong from age group of 41-50 years. 34(30.91%) patients were belong from age group of 51-60 years. 41(37.27%) patients were belong from age group of 61-70 years. And 11 (10%) patients were belong from age group of 71-80 years. Majority of patients 41(37.27%) were belong from age group of 61-70 years.

This study was included 73(66.36%) male and 37(33.63%) female.

In this study, 48(43.63%) patients were diabetes with hypertension. This was the leading cause of chronic renal failure. 42(38.18%) patients were diabetes. Hypertension was in 16(14.54%) patients (13%). Adult polycystic kidney disease (APKD) was in 2 patients (1.82%), chronic glomerulonephritis in 1(0.91%) patient, and 1(0.91%) patient was suffered from obstructive pathology.

In this study, the range of blood urea level was between 50-280 mg/dl. 34(30.91%) patients were belonged with range of blood urea 50-100 mg/dl. 43(39.09%) patients were belonged with range of 101-150 mg/dl blood urea. 28(25.45%) patients were blood urea range 151-200 mg/dl. And 5(4.54%) patients were with range of blood urea greater than 200mg/dl. Maximum number of patients 43(39.09%) were belonged with blood urea range 101-150 mg/dl.

In this study, the range of serum creatinine level was between 1.5-20.8 mg/dl. 23(20.91%) patients were suffered from mild chronic renal failure and level of serum creatinine 1.5-3 mg/dl. 45(40.91%) patients were suffered from moderate chronic renal failure and level of serum creatinine 3-6 mg/dl. And 42(38.18%) patients were suffered from severe stage of chronic renal failure and of serum creatinine level greater than 6 mg/dl. Maximum numbers of patients 45(40.91%) patients were suffered from moderate chronic renal failure.

Table.1. distribution of serum creatinine levels of patients with CRF.

Level of Serum Creatinine (mg/dl)	Frequency	Percentage
1.5-3 (mild CRF)	23	20.91
3-6 (moderate CRF)	45	40.91
>6 (severe CRF)	42	38.18
Total	110	100

On the basis of creatinine clearance, 8(7.27%) patients were stage 3: moderate chronic renal insufficiency (glomerular filtration rate: 30-59%). 36(32.72%) patients were stage 4: severe chronic renal insufficiency (GFR: 15-29%). And 66(60%) patients were compromised stage 5: end stage renal diseases (GFR <15%). This study shown, majority of patients 66(60%) were in stage 5: end stage of renal disease.

In this study, 22(20%) patients were 5.1-7 gm% hemoglobin level. 54(49.09%) patients were 7.1-9 gm% hemoglobin. 32(29.09%) patients were 9.1-11 gm% hemoglobin level and 2(1.82%) were greater than 11 gm% hemoglobin. That is, majority of patients 54(49.09%) were 7.1-9 gm% hemoglobin level.

In our present study, 1(0.91%) patients with chronic renal failure was less than 3 mEq/L serum potassium level. 22(20%) patients were serum potassium level in between 3.1-4 mEq/L. 45(40.91%) patients were 4.1-5 mEq/L serum potassium level. 42(38.18%) patients were 5.1-7 mEq/l serum potassium level. Majority of patients 45(40.91%) were serum potassium level in between 4.1-5mEq/L.

In this study, 9(8.18%) patients were level of serum calcium in between 6-7 mg/dl. 23(20.91%) patients were 7.1-8 mg/dl serum calcium level. 42(38.18%) patients were 8.1-9 mg/dl serum calcium. 22(20%) patients were 9.1-10 mg/dl serum calcium level. And 12(10.91%) patients were 10.1-11 mg/dl serum calcium level. Majority of patients 42(38.18%) were 8.1-9 mg/dl serum calcium level.

In this study, 22(20%) patients with chronic renal failure were 2-4 mg/dl serum phosphorus level. 49(44.54%) patients were serum phosphorus in between 4.1-5 mg/dl. 29(26.36%) patients were 5.1-6 mg/dl serum phosphorus level. 7(6.36%) patients were serum phosphorus level 6.1-7 mg/dl. And 3(2.73%) patients were serum phosphorus level in between less than 7 mg/dl. Majority of patients 49(44.54%) in this study were serum phosphorus level in between 4.1-5 mg/dl.

6(5.45%) patients were less than 150 mg/dl total serum cholesterol level. 70(63.63%) patients were 151—200 mg/dl total serum cholesterol level. And 34(30.91%) patients were greater than 200 mg/dl total serum cholesterol level. In this study, maximum number of patients 70(63.63%) were serum cholesterol level in between 151-200 mg/dl.

In this present study, 16(14.54%) were less than 150 mg/dl level of triglyceride of patients with chronic renal failure. 64(58.18%) patients were 151-170 mg/dl triglyceride level. And 30(27.27%) patients with chronic renal failure were greater than 170 mg/dl triglyceride level.

In this study, 15(13.63%) patients with chronic renal failure were less than 100 mg/dl LDL cholesterol level. 74(67.27%) patients were 101-130 mg/dl LDL cholesterol level. And 21(19.09%) patients with chronic renal failure were greater than 130 mg/dl LDL cholesterol level. In this study, maximum number of patients 74(67.27%) were LDL cholesterol level in between 101—130 mg/dl.

57(51.81%) patients were less than 30 mg/dl HDL cholesterol level. 37(33.63%) patients were 31-40 mg/dl HDL cholesterol level. And 16(14.54%) patients were greater than 40 mg/dl HDL cholesterol level. In this study, maximum number of patients 57(51.81%) were HDL cholesterol level less than 30 mg/dl.

Electrocardiographic changes of patients with chronic renal failure: 68(61.82%) patients were left ventricular hypertrophy, 32(29.09%) patients were ST-T changes on electrocardiogram. 33(30%) patients with chronic renal failure were no LVH on electrocardiogram. 7(6.36%) patients were showed low voltage complexes on electrocardiogram.

In this study, echocardiographic findings of patients with chronic renal failure: 52(47.27%) patients were concentric LVH. 22(20%) patients were eccentric LVH. 44(40%) patients were diastolic dysfunction. 21(19.1%) patients were systolic dysfunction. 21(19.1%) patients were pericardial effusion on echocardiogram. And 32(29.1%) patients were normal echocradiographic changes. Majority of patients 52(47.27%) with chronic renal failure were concentric left ventricular hypertrophy findings on echocardiograph. This echocardiographic features of patient were followed by each others.

In this study, comparison of severity of chronic renal failure with presence of left ventricular hypertrophy on 2 D Echo: out of 76 patients (mild, moderate and severe CRF) with left ventricular hypertrophy, 13(17.10%) patients with mild chronic renal failure were feature of left ventricular hypertrophy, and 10(29.41%) patients were no left ventricular hypertrophy. 20(26.31%) patients with moderate chronic renal failure were features of left ventricular hypertrophy), and 19(55.88%) patients were not the left ventricular hypertrophy. 43(56.58%) patients with severe chronic renal failure were features of left ventricular hyper trophy and 5(17.70%) patients were not the features of left ventricular hypertrophy.

Table.2. Severity of CRF with presence of LVH or not on echocardiography:

Severity of CRF	LVH		NO LVH	
	No	%	No	%
Mild CRF	13	17.1%	10	29.41%
Moderate CRF	20	26.31%	19	55.88%
Severe CRF	43	56.58%	5	17.7%
Total	76	100%	34	100%

Discussion:

A total of 110 patients with chronic renal failure were taken in this study. This study was conducted in OPD/ IPD of department of Medicine, Katihar Medical College, Katihar, Bihar.

Chronic Renal Failure (CRF) is one of the common conditions which a physician comes across in day to

day practice. Chronic renal failure affects every aspect of the lives of the patients and involved all systems of body and results in various abnormalities. Diabetes and hypertension are the two most common causes of end stage renal disease and are associated with a high risk of death from cardiovascular disease. Mortality in patients with end stage renal disease remains 10-20 times higher than that in the general population. The focus in recent years has thus shifted to optimising the care of these patients during the phase of chronic kidney disease, before the onset of end stage renal disease [10].

Serum creatinine is commonly used to estimate creatinine clearance but is a poor predictor of glomerular filtration rate, as it may be influenced in unpredictable ways by assay techniques, endogenous and exogenous substances, renal tubular handling of creatinine, and other factors (age, sex, body weight, muscle mass, diet, drugs) [11]. Glomerular filtration rate is the "gold standard" for determining kidney function, but its measurement remains cumbersome. For practical purposes, calculated creatinine clearance is used as a correlate of glomerular filtration rate and is commonly estimated by using the Cockcroft-Gault formula or the recently described modification of diet in renal disease equation.

Premature cardiovascular disease is a significant cause of morbidity and mortality among patients with CRF. Four main structural abnormalities of the heart have been described in patients with CRF :

1. LV hypertrophy,
2. Expansion of the nonvascular cardiac interstitium leading to inter-myocardiocytic fibrosis,
3. Changes in vascular architecture, and
4. Myocardial calcification.

All these abnormalities promote systolic as well as diastolic LV dysfunction which predisposes to symptomatic heart failure, which is a risk factor for premature death.

Various diagnostic modalities, non-invasive such as electrocardiography, echocardiography are utilized for diagnosing cardiovascular function. Echocardiography provides an excellent non-invasive method to delineate details of the anatomy of cardiac cavity, wall dimensions and wall movements. It is now increasingly used in the assessment of cardiac performance and is also invaluable in the demonstration of structural abnormalities such as LVH and pericardial effusion.

Left ventricular hypertrophy is the single strongest independent predictor of adverse cardiovascular events.

LVH is a major echocardiographic finding in uremic patients. In the present study, 13 (17.10%) patients of mild, 20(26.31%) patients of moderate and 43 (56.58%) patients of severe chronic Renal Failure group had Left Ventricular Hypertrophy.

S A Kale, N S Kulkarni, S Gang, A Ganju, L Shah, MM Rajapurkar [12]conducted prospective study includes 161 patients of end stage renal disease and they concluded Left ventricular disease was common & encountered in 105 (65.2%) patients. Only 56(34.8%) had normal echocardiogram.

Our study was conducted on 110 patients and observed the 52(47.27%) patients had concentric left ventricular hypertrophy, 22(20%) patients had eccentric LVH, 44(40%) patients had diastolic dysfunction, 21(19.1%) patients had systolic dysfunction, and 21(19.1%) patients had pericardial effusion. Diabetes with hypertension(43.63%), diabetes(38.18%) ,Hypertension(14.54%), older age, male sex, anemia, hyperalbuminemia and hypocalcaemia were found to be significantly associated with manifestations of left ventricular disorders. Patients of end stage renal disease with combine of diabetes and hypertension(43.63%) had higher frequency of systolic dysfunction and 29.1% patients had normal echocardiogram.

N A Tomilina, G. V. Volgina, et al conducted a study on 150 patients with CRF and 160 patients with ESRD, and conluded that risk factors for LVH were the decline in Ccr, age, increase of blood pressure, anaemia [13].

Parfrey PS, Foley RN, Harnett JD, Kent GM, Murray DC, Barrey PE conducted study on 432 end-stage renal disease patients who survived at least 6 months had an echocardiogram on initiation of dialysis therapy. On initiation of ESRD therapy 16% of patients had systolic dysfunction, 41% concentric LV hypertrophy, 28% LV dilatation, and only 16 % had normal echocardiograms [4].

Alattin Yildiz, East Memisoglu, Husyin Oflaz, Halil Yazicil, et al. conducted a measured biochemical parameters, including BUN, creatinine, albumin, haemoglobin, C-reactive protein and fibrinogen levels and performed echocardiography, high resolution B-mode carotid ultrasonography and EBCT. LVH was detected in 75% of patients. Plaque-positive patients had higher left ventricular mass index (LVMI) than plaque-negative patients [14].

In the present study, the most common cause of CRF was diabetes with hypertension in 43.63 % followed by diabetes alone in 38.18%.

Jose Luis Rodriguez, Roberto Arteaga Crespo concluded that the hypertensive nephropathies (24%) was the commonest cause of CRF. Diabetic nephropathy (20%), obstructive nephropathy (16%), chronic pyelonephritis, glomerulonephritis, polycystic kidney, schistosomiais (Bilharzias) were less common. Males (63%) were more suffered with CRF than females (37%) [15].

Present study was showed 73(66.36%) men and 37(33.63%) women were suffered from CRF. This present study supported the findings of above study.

Rizvi SA, Manzoor K concluded that the two most common causes of CRF were diabetes mellitus in 113 (22.3%) and hypertension in 92 (18.1%) patients respectively [16].

Findings of our study was shown the more common causes of CRF were diabetes and hypertension, diabetes with hypertension (43.63%), diabetes (38.18%) and hypertension (14.54%). Our study correlated with the above study.

Frommer JP et al has reported an incidence of pericardial effusion in 18(36%) patients [17].

Our study was showed that pericardial effusion was found in 21 (19.1%) patients with chronic renal failure.

Future Research:

Science is dynamic and there is always a scope of improvement and change in time to come ahead. With progressive aim to move ahead we aspire to achieve highly accurate and reliable results. Thus every study leaves back scopes for other researcher to do something more advanced and varied in order to touch the height of perfection. This study examined only 110 subjects, future researchers can expand the study by including more number of subjects so as to make generalization of the results and practice, further studies with a larger sample size and in multiple centers are required. Thus it could be applied to real life situation.

Limitation:

There were several limitations like, the sample size was small, and it was a hospital-based study, the prevalence of exposure and outcome variables may be different from a community setting.

Conclusion:

Present study concluded that the patients of diabetes with hypertension have higher prevalence of chronic renal failure. Majority of patients with chronic renal failure have left ventricular hypertrophy, it leads to severe renal failure. So that, Echocardiographic findings of patients with chronic renal failure implies that detail cardiovascular evaluation are needed despite of absence of symptoms in renal insufficiency.

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ORIGINAL RESEARCH ARTICLE

A Study on the Clinical Profile and Hepatitis B and C Viral Markers in Acute and Chronic Liver Disease.

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Abstract:

Objective: our study was to evaluate the viral markers presented in acute and chronic liver diseases, the clinical profile of patients and to examine the biochemical changes in acute and chronic liver diseases. Methodology: A total of 110 patients with the case of liver disease were considered on the basis of detail clinical history and laboratory findings and sonography evidence. Results: Data was analyzed by using MS-Office software. Conclusions: Maximum numbers of patients with liver disease were involved with age group of 41-60 years. Majority of patients were presented with abdominal distension, anorexia and jaundice. All the patients were abnormal LFT. Few patients were abnormal prothrombin time and abnormal viral markers in the form of HBsAg and antiHCV.

Key words: liver disease, viral markers, clinical profile.

Introduction:

Liver is one of the most vital organs of body which happens to be second largest organ in the body next to skin. Liver has the critical job of maintaining body's metabolic homeostasis which includes processing of dietary aminoacids, carbohydrates, lipids and vitamins, removal of microbes and toxins from splachnic blood, enroute to systemic circulation, detoxification and excretion imto bile of endogenous waste products [1,2,3].

The diversity and complexity of hepatic function is such that, it exceeds brain in terms of biologic sophistication and no doubt liver is held in high esteem since ancient times.

Enormous functional reserve of liver masks the clinical impact of early liver damage. However, with progression of diffuse disease the consequences of deranged liver function becomes life threatening [1,2].

Liver is vulnerable to wide variety of metabolic, toxic, microbial, circulatory and neoplastic insults [1,2,3].

The dominant primary disease of liver are viral hepatitis, alcoholic liver disease and hepatocellular carcinoma.

Infectious disorders of liver dominant the clinical practice of hepatology. Hepatology experienced an extraordinary boost when major viruses that affect the liver were identified.

Hepatitis virus A-G have been identified and studied as etiological agents for various liver disorders [1,2,3].

Hepatitis B and C virus are the major cause of chronic liver disease in India [1,2,4].

Hepatitis B is a common disease worldwide with an estimated global prevalence of over 350 million or approximately 5 % of world's population [1,2,3,4].

Also 2/3rd of all cases of hepatocellular carcinoma is caused by hepatitis B virus [1,2,3,5,6].

Total hepatitis B virus occurrence in India is around 3-4 % [7,8].

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The WHO places HBV in the top 10 causes of death worldwide [9]. Chronic hepatitis B constitutes more than 50% of chronic hepatitis cases.

In this context of large population and absence of a national immunization programme would mean a projected increasing burden of infection and liver disease due to HBV in India in the years to come. In this perspective, HBV epidemiology in India becomes relevant not only nationally but also internationally, because of the possibility of India becoming the largest HBV infection pool in the world [7,10,11].

WHO estimates that 3 % of world population is infected with HCV and around 170 million individuals are chronic carriers at risk of developing liver cirrhosis and hepatocellular carcinoma [12,13,14].

Patients with dual HBV and HCV infection may also have higher rate of hepatocellular carcinoma than patients infected with either virus alone.

It is a paradox that such an important organ has little capacity to repair itself once it is damage beyond a critical level. Damaged liver not only affects normal functioning of liver but also, will lead to derangement of function of other organs.

To cap it all there are no satisfactory specific treatment for liver disease. Hence prevention is more important and more relevant. Early diagnosis may contribute to prevention of complications. Objective of our study was to evluate the viral markers presented in acute and chronic liver diseases, the clinical profile of patients with acute and chronic liver disease and to examine the biochemical changes in acute and chronic liver diseases.

Material and Methods:

Study design:

A total of 110 subjects of liver disease with age of 22 years to greater than 60 years were included. All Patients fulfilled the inclusion criteria attending either outpatient department or inpatients department of Katihar Medical College, Katihar, Bihar during period of January 2015 to august 2015 were selected for this study.

The attendant of entire subject/patients signed an informed consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar, India was sought.

Simple random sampling was done for selection of data.

Inclusion criteria of Present study was, the Clinical features suggestive of hepatitis, liver function test (LFT) for detection of acute and chronic liver disease. And clinically suspected hepatocellular carcinoma patients.

Exclusion criteria of our study was Malaria, Leptospirosis, Dengue, and Drug induced hepatitis, Auto immune hepatitis, Toxins, Haemolytic anemia and Infiltrative disorders.

Detailed history, physical examination and necessary investigation of all patients were recorded.

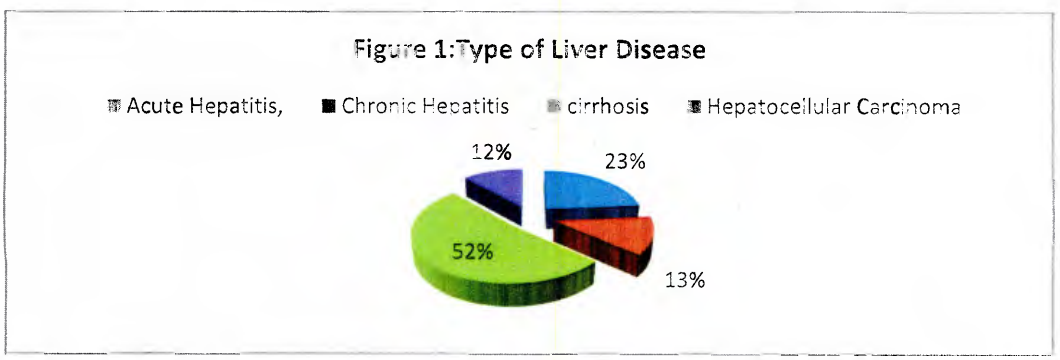
Investigation: Haemoglobin, Total count, Differentrial count, ESR, Random blood sugar, Blood urea, Serum creatinine, Urine analysis, USG abdomen, Liver function test (LFT), HBsAG, Anti HCV, Liver biopsy when indicated, Reticulocyte count and Prothrombin time was done.

Statistical analysis:

Simple analysis method was taken to analyzed the data with the help of MS-Office soft ware.

Observation and Results:

One hundred ten patients with acute and chronic liver disease during period of January 2015 to August 2015, who attended outpatient clinic and admitted in ward of department of medicine of Katihar Medical College, katihar, Bihar, India were enrolled in study.



Out of 110 patients , 26(23%) patients were acute Hepatitis, 14(13%) patients were Chronic Hepatitis, 57(52%) and 13(12%) patients were diagnosed as cirrhosis & Hepatocellular Carcinoma respectively.

Table 1 : Distribution of patients according to Gender and Type of Liver Diseases

	Male	Female	Total
Acute Hepatitis,	20(76.9%)	06(23.1%)	26(100%)
Chronic Hepatitis	09(64.3%)	05(35.7%)	14(100%)
cirrhosis	41(71.9%)	16(28.1%)	57((100%)
Hepatocellular Carcinoma	09(69.2%)	04(30.8%)	13(100%)
Total	79(71.82%	31(28.19%)	110(100%)

In this study 26 patients (male: 20 & female: 6) with acute hepatitis, 14 patients (male: 9 & female: 5) with chronic hepatitis, 57 patients (male: 41 & female: 16) with cirrhosis, and 13 patients (male:9 & female: 4) with hepatocellular carcinoma were selected.

Table 2 : Presenting symptoms:

Diagnosis	anorexia	nausea	vomiting	Jaundice	fever	Abdominal Pain	abdominal distension	abnormal behavior	Gi bleed	
									haemet	malena
Acute hepatitis	13 (50%)	18 (69.23%)	14 (53.85%)	19 (73.07%)	13 (50%)	10 (38.46%)	-	-	-	-
Chronic hepatitis	4 (28.57%)	-	2 (14.29%)	3 (21.42%)	-	2 (14.29%)	-	-	-	-
cirrhosis	51 (89.48%)	30 (52.63%)	6 (10.53%)	36 (63.16%)	3 (5.26%)	14 (24.56%)	55 (96.50 %)	5 (8.78%)	10 (17.54%)	29 (50.88%)
hepatocellular carcinoma,	5 (38.45%)	6 (46.15%)	7 (53.85%)	4 (30.77%)	1 (7.70%)	5 (38.50%)	3 (23.08 %)	-	-	-

Patients with acute hepatitis, 13(50%) were anorexia, 18(69.23%) were nausea, 14(53.85%) were symptoms of vomiting, 19(73.07%) were jaundice, 13 (50%) were fever. And 10 (38.46%) patients were abdominal pain.

In patients with chronic hepatitis, 4(28.57%) were anorexia, 2 (14.29%) patients were vomiting, 3(21.42%) were jaundice. And 2 (14.29%) patients were complained pain in abdomen.

In cases of cirrhosis, 51 (89.48%) were anorexia, 30 (52.63%) were complained nausea, 6 (10.53%) were vomiting, 36 (63.16%) were sign of jaundice, 3 (5.26%) were fever, 14 (24.56%) were complained pain in abdomen, 55 (96.50%) were abdominal distension, 5 (8.78%) patients were showed abnormal behavior. And 39 (68.42%) patients were gastrointestinal bleeding, in which 10 (17.54%) were haemetemesis, 29 (50.88%) patients were malena. These all symptoms were followed to each other.

Patients with hepatocellular carcinoma, 5 (38.45%) were anorexia, 6 (46.15%) were nausea, 7 (53.85%) were vomiting, 4 (30.77%) were jaundice, 1 (7.70%) was fever, 5 (38.50%) were abdominal pain. And 3 (23.08%) patients were abdominal distension. Above all these symptoms were followed to each other.

Past history:

Past history of patients with acute hepatitis, 6 (23%) were the case of HIV. In chronic hepatitis 4(28.56%) were the past history of chronic renal disease. And past history of patient with cirrhosis, 18 (31.56%) patients were the past history of diabetes mellitus, 7 (12.29%) were history of HIV infection, 9 (15.79%) were chronic renal disease, 10 (17.54%) patients were the past history of tattooing. 4 (30.76%) patients with hepatocellular carcinoma were the past history of alcoholism.

Table 3 : Physical examination of patients with liver disease.

	Acute hepatitis		Chronic hepatitis		Cirrhosis		HCC	
	NO	%	NO	%	NO	%	NO	%
pallor	-	-	2	14.29%	22	38.60%	2	15.39%
icterus	26	100%	3	21.43%	31	54.39%	4	30.77%
edema	-	-	-	-	44	77.20%	4	30.77%
Parotid enlargement	-	-	-	-	28	49.12%	-	-
Loss of SSC	-	-	-	-	34	59.65%	-	-
ascitis	-	-	-	-	54	94.74%	8	61.54%
hepatomegaly	10	38.46%	5	35.71%	3	5.23%	13	100%
splenomegaly	-	-	-	-	52	91.23%	-	-
Dilated veins	-	-	-	-	52	91.23%	12	92.31%

Pallor was present in 2 (14.29%) of patients with chronic hepatitis, 22 (38.60%) patients with cirrhosis and 2 (15.39%) patients with hepatocellular carcinoma. Icterus was present in 26(100%) patients with acute hepatitis, 3(21.43%) patients with chronic hepatitis, 31(54.39%) patients with cirrhosis and 3(30.77%) patients with hepatocellular carcinoma. Edema was present in 44 (77.20%) patients with cirrhosis and 4 (30.77%) patients with hepatoccellular carcinoma. Parotid enlargement was present in 28 (49.12%) patients with citrrhosis. Loss of secondary sexual character was seen in 34 (59.65%) patients with cirrhosis. Ascitis was seen in 54 (94.74%) patients with cirrhosis and 8 (61.54%) patients with hepaticcellular carcinoma. Hepatomegaly was present in 10(38.46%) patients with acute hepatitis, 5 (35.71%) patients with chronic hepatitis, 3(5.23%) patients with cirrhosis and 13 (100%) patients with hepatoccellular carcinoma. Splenomegaly was present in 52 (91.23%) patients with cirrhosis. Dilated vein was seen in 52 (91.23%) patients with cirrhosis and 12 (92.31%) patients with hepatoccellular carcinoma.

Hemoglobin: All the patients with acute hepatitis, hemoglobin was greater than10 gm%. 31 % Patients with cirrhosis, hemoglobin was less than 10gm% and remaining patients with cirrhosis, hemoglobin was varied between 10-12 gm %. 77% patients with hepatocellular carcinoma, hemoglobin was varied between 10-12 gm%.

Bilirubin: Patients with acute hepatitis, bilirubin was varied between 1.3 to greater than 20. And patients with acute hepatitis B, bilirubin was varied between range of 5to 20. Patients with chronic hepatitis except chronic hepatitis C, bilirubin was varied between 1.4 to 5. And in patients with chronic hepatitis C was normal serum biulirubin.

15.4% patients with cirrhosis was normal serum bilirubin. And 42.6 % patients were bilirubin varied between 1.4 to 5. Patients with hepatocellulr carcinoma (HCC), bilirubin was varied between 1.4 to 5.

AST and ALT: Patients with acute hepatitis, mean AST was 331.5. Patients with chronic hepatitis, mean AST was 195.6, patients with cirrhosis was 116.6 and patients with hepatocellular carcinoma, mean AST was 101.7.

Mean ALT in patients with acute hepatitis was 392.1, in chronic hepatitis was 190.5, in cirrhosis was 130.6. And mean ALT patients with hepatocellular carcinoma was 103.8.

Prothrombin time: Elongation of prothrombin time was present in 52.31% patients with acute hepatitis, 18 % patients with cirrhosis and 28 % patients with HCC.

HBsAg: HBsAg positive was positive in 37.1 % patients with acute hepatitis, 26 % patient with chronic hepatitis, 28 % patients with cirrhosis and 52 % patients with HCC.

HCV: 27 % patients with acute hepatitis was positive anti HCV and HCV RNA. 28.01% patients with cirrhosis was positive anti HCV and HCV RNA. And 28 % patients with HCC was positive for anti HCV.

Table 4: Imaging (Ultrasonography) of patients with liver disease.

Diagnosis	SL+SM+AS	Normal	AE+AS	AE+AS+SM	SL+SM
Acute hepatitis	-	**12(10.91%)**	-	-	-
Acute hepatitis B	-	**6(5.45%)**	-	-	-
Chronic hepatitis	-	-	-	-	-
Chronic hepatitis B	-	-	-	-	-
Chronic hepatitis C	-	3(2.73%)	-	-	-
Cirrhosis with portal hypertension	16(14.55%)	-	**1(0.91%)**	23(20.91%)	-
Cirrhosis with portal hypertension with hepatitis B	8(7.27%)	-	-	2(1.82%)	-
Cirrhosis with hepatitis C	-	-	-	**1**(0.91%)	-
HCC	-	-	-	-	-

SL+SM+AS: Shrunken liver with splenomegaly with ascetic, AE+AS: altered echo texture with ascitis, AE+AS+SM: altered echo texture with ascitis with splenomegaly, SL+SM: Shrunken liver with splenomegaly.

18 patients with acute hepatitis was normal sonographic findings. And 11 patients were shown hepatomegaly on sonography in acute hepatitis. 7 Patients with chronic hepatits and 4 patients with chronic hepatitis B were shown hepatomegaly on sonography. 3 patients with chronic hepatitis C were normal sonography finding. 16 patients with cirrohosis with portal hypertension were shown shrunken liver with splenomegaly with ascitis (SL+SM+AS). 1 patients with cirrohosis with portal hypertension was shown altered echo texture with ascitis (AE+AS).

23 patients with cirrohosis with portal hypertension were shown altered echo texture with ascitis with splenomegaly(AE+AS+SM). 8 patients Cirrhosis with portal hypertension with hepatitis B were shown sonography evidence of Shrunken liver with splenomegaly with ascetic (SL+SM+AS).

2 Patients cirrhosis with portal hypertension With hepatitis B were shown altered echo texture with ascitis with splenomegaly (AE+AS+SM). 1patient with Cirrhosis with hepatitis C was shown on sonography evidence of altered echo texture with ascitis with splenomegaly(AE+AS+SM). All 13 patients with HCC were shown sonography evidence of hepatomegaly.

AG reversal: All patients with cirrhosis were AG ratio reversal.

Discussion:

Our study was taken 110 patients with age group of 22 years to greater than 60 years. In this study, maximum number of patients with acute hepatitis was 9 (34.61%) out of 26, belonged from age group of 31 to 50 years. Maximum number of patients with chronic hepatitis was 5 (35.71%) which was belonged from age 51 to 60 years.In cirrhosis, maximum number of patients 21(36.84%) out of 57, were with age group of 51 to 60 years. And in case of hepatocellular carcinoma, maximum number of patients 5(38.50%) were belonged with age group of 51 to 60 years.Similar study was done by Ravinder Kaul, et al [15]. They were taken the total of 306 patients with acute hepatitis with age group of 1to 68 years. Estrad J Y et al [16] also done the similar study and they included the total of 203 patients.

Results of present study was shown that patients with chronic hepatitis were without any clustering.

In present study, patients with cirrhosis were 51.89%. Tarun Kumar, et al. [17] was studied on 46.83 %with cirrohosis.

Sashi Bala Paul et al. [18] Study on 301 patients with hepatocellular carcinoma. We were studied on 13 patients (11.82%) patients with hepatocellular carcinoma.

In current study, 79(71.88%) patients was male and 31(28.18%) patients was female in out of total 110 patients. In acute hepatitis male and female ratio was 10:3, in chronic hepatitis, male:female was 9:5, in cirrhosis male and female ratio was 41:16 and in hepatocellular carcinoma male and female ratio was 9:4. Similar studiy was done by Kaur H, et al. [19] and found that male and female ratio in acute hepatitis was 1.65:1. Golnaz Bahramedi, et al [20] was studied on Iranian patients and they were found that male and female ratio was 4:1 in chronic hepatitis patients. Paul SB, et al. studied on cirrhosis patients and found that male and female ratio was 6:1. Khan et al [21] studied on hepatocellular carcinoma patients and found male and female ratio was 4.5:1.

Among all those study, we were found that male was more suffered from liver disease than female.

.In the present study, jaundice was the commonest clinical symptom and sign, and it was found in 73.07% patients with acute hepatitis. Holgado GM, et al. [22] were found in 92.7% patients with acute hepatitis.

We were found that jaundice followed by anorexia in 50 % patients with acute hepatitis, 28.57 % patients with chronic hepatitis, 89.48 % patients with cirrhosis and 38.45 % patients with HHC. Kaur H, et al. [19] was found in 72.6 % patients and Holgado GM, et al. [22] was found in 61.1 % patients.

Pain abdomen was found in 38.46 % patients with acute hepatitis, 14.29 % patients with chronic hepatitis, 89.48% patints with cirrhosis and 38.50 % patients with hepatocellular carcinoma.

In our study pain abdomen was found in 50% patients with acute hepatitis, 0% patients with chronic hepatits, 5.26 % patients with cirrhosis and 38.50% patients with hepatocellular carcinoma.

Nausea was found in 69.23% patients with acute hepatitis, 52.63 % patients with cirrhosis, 46.15% patients with HHC. Vomiting was found in 53.85% patients with acute hepatitis, 14.29% patients with chronic hepatitis, 10.53% patients with cirrhosis and 53.85% patients with HHC. Abdominal distension was found in 96.50 % patients with cirrhosis, 23.08% patients with HHC followed by anorexia. Abnormal behavior was found in 8.78% patients with cirrhosis. Haemetemesis was found in 17.54 % patents with cirrhosis. And malena was found in 5.88% patients with cirrhosis.Usha Arora, et al [23] was found that 35.71% patients was sign of jaundice.

Our study found that, pallor was present in 14.29 % patients with chronic hepatitis, 38.60% patients with cirrhosis and 15.39% patients with HHC. Edema was present in 77.20% patients with cirrhosis and 30.77% patients with hepatocellular carcinoma. Parotid enlargement was seen in 49.12% patients with cirrhosis, icterus was seen in 100 % patients with acute hepatitis, 21.43 % patients with chronic hepatitis, 54.39 % patients with cirrhosis and 30.77 % patients with HHC. Ascitis was seen in 94.74 % patients with cirrhosis and 61.54 % patients with HHC. Hepatomegaly was seen in 38.46% patients with acute hepatitis, 35.71 % patients with chronic hepatitis, 5.23 % patients with cirrhosis and 100 % patients with HHC. Dilated vein was seen in 91.23 % patients with cirrhosis and 92.31% patient with hepatocellular carcinoma.

42.11% patients with cirrhosis with portal hypertension were Shrunken liver with splenomegaly with ascetic, 1.75% patients were altered echo texture with ascitis. 45.61% patients were altered echo texture with ascitis with splenomegaly.

All the patients were hemoglobin ranges from 10 to 12 gram percent. 1.3 to greater than 20 bilirubin was found in acute hepatitis patients and 1.4 to 5 in chronic hepatitis patients. 15.4 % patients with cirrhosis was normal serum bilirubin. And 42.6 % patients were bilirubin varied between 1.4 to 5. Patients with hepatocellulr carcinoma (HCC), bilirubin was varied between 1.4 to 5.

Patients with acute hepatitis, mean AST was 331.5. Patients with chronic hepatitis, mean AST was 195.6, patients with cirrhosis were 116.6 and patients with hepatocellular carcinoma, mean AST was 101.7.

Mean ALT in patients with acute hepatitis was 392.1, in chronic hepatitis were 190.5, and in cirrhosis were 130.6. And mean ALT patients with hepatocellular carcinoma (HCC) were 103.8.

In acute hepatitis there was high AST, ALT and bilirubin compared to other groups probable because of active hepatitis, comparable to study group. In other groups values matches with comparable studies.

Elongation of prothrombin time was present in 52.31% patients with acute hepatitis, 18 % patients with cirrhosis and 28 % patients with HCC.

HBsAg positive was positive in 37.1 % patients with acute hepatitis, 26 % patient with chronic hepatitis, 28 % patients with cirrhosis and 52 % patients with HCC.

27 % patients with acute hepatitis was positive anti HCV and HCV RNA. 28.01% patients with cirrhosis was positive anti HCV and HCV RNA. And 28 % patients with HCC were positive for anti HCV.

Future Research:

Science is dynamic and there is always a scope of improvement and change in time to come ahead. With progressive aim to move ahead we aspire to achieve highly accurate and reliable results. Thus every study leaves back scopes for other researcher to do something more advanced and varied in order to touch the height of perfection. This study examined only 110 subjects (79 male and 31 female), future researchers can expand the study by including more number of subjects so as to make generalization of the results and

practice, further studies with a larger sample size and in multiple centers are required. Thus it could be applied to real life situation.

Relevance to clinical practice:

This study is relevant to the high incidence liver disease. It opens up new possibilities of prevention of liver disease and makes maintain the good health of population. Such knowledge in future would not only reduce this disease but also have significant medical benefits on the health care systems

Limitation:

There were several limitations like, the sample size was small, and it was a hospital-based study, the prevalence of exposure and outcome variables may be different from a community setting. Hepatocellular carcinoma was identified as a mass lesion on the investigation of ultrasound and CT abdomen. Histopathology examination was not done.

Conclusion:

Maximum numbers of patients with liver disease were involved with age group of 41-60 years. Majority of patients were presented with abdominal distension, anorexia and jaundice. All the patients were abnormal LFT. Few patients were abnormal prothrombin time and abnormal viral markers in the form of HBsAg and antiHCV.

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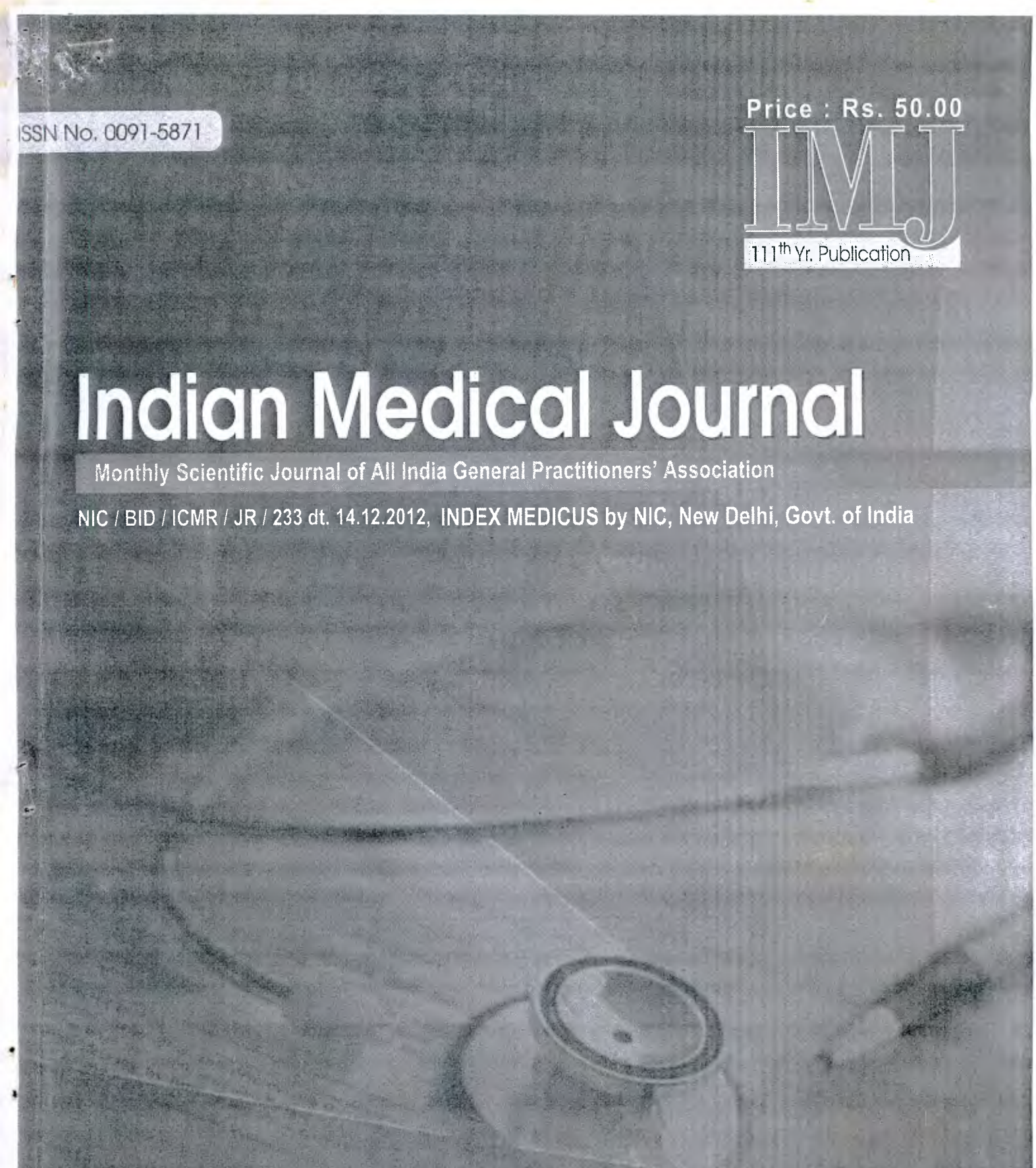

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CME Notice —CME Notice 108(A)

Association News —*Dr. Pinaki Ghosh* 128(A)

Original & Clinical Research

A Comparative Evaluation of Efficacy of Methotrexate versus Methotrexate and Sulphasalazine Combination Therapy in Early Rheumatoid Arthritis with Low and Moderate Disease Activity and in Absence of Poor Prognostic Factors; First Study From Eastern India
—Dr. Sk Minhajuddin Siraj, Dr. Sudipta Pal, Dr. Kaushik Manna, Dr. Swayang Prakas Chowdhury, Dr. Syamal Kundu, Dr. Subhrajyoti Mitra 7(A)

A Comparative Study of Vaginal and Oral Misoprostol for Induction of Labour
—Dr. Punam Singh, Dr. Sunita Kumari, Dr. Chitra Sinha, Dr. Chander Kiran 10(A)

A Study of Ormeloxifene in Case of Dysfunctional Uterine Bleeding
—Dr. Anupam 11(A)

Clinico-Epidemiological Study of HIV Positive Female Patients
—Dr. Anupam 14(A)

Congenital Talipes Equino Varus
—Dr. Rajnish Kumar, Dr. Vibhash Chandra 15(A)

Maternal and Perinatal Outcome following Premature Rupture of Membrane
—Dr. Minkoo Singh 21(A)

Observation of Neonatal Apnea in Relation to Aetiopathogenesis and their Outcome
—Dr. Shilpi Golwara 23(A)

Perianal Dermatitis Treatment with Tacrolimus
—Dr. Rajeev Ranjan Jha 24(A)

Salicylic Acid Gel in Molluscum Contagiosum
—Dr. Rajeev Ranjan Jha 24(A)

Prevalence of Lupus Anticoagulant in Pregnancy Hypertension and Its Significance on Pregnancy Outcome
—Dr. Minkoo Singh 25(A)

Gynaecological Problem in Adolescent Girl
—Dr. K. Manju, Dr. Rashmi Kishore 30(A)

Pruritus in Late Pregnancy and Its Impact on Fetal Well-Being
—Dr. Anupam 31(A)

Study of Risk Factors for Ectopic Pregnancy
—Dr. Anupam 32(A)

Study on Hepatitis 'B' Surface Antigen (HbsAg) by ELISA Method in Hepatitis 'B' Virus Infected Persons
—Dr. Spriha Smriti 34(A)

Sustained Cervical Traction as a Method to Reduce 3rd Stage Bleeding
—Dr. Sunita Kumari, Dr. Punam Singh, Dr. Chitra Sinha, Dr. Chander Kiran 35(A)

Original & Clinical Research

Universal Screening for Gestational Diabetes Mellitus in India
—Dr. Anupam 37(A)

Urticaria –Classification
—Dr. Rajeev Ranjan Jha 38(A)

A Comparative Study of Trochanteric Fracture Femur Treated with Dynamic Hip Screw and Proximal Femoral Nailing
—Dr. Pawan Kumar, Dr. Vikash Ranjan 39(A)

Antibiotics: The Most Misused Drugs
—Dr. Indra Deo Singh, Dr. Abhijeet Alok 42(A)

Assessment of Results of Open Arthrolysis in Post Traumatic Stiff Elbow- A Retrospective & Prospective Study
—Dr. Vikash Ranjan, Dr. Pawan Kumar 43(A)

Spectrum of Bacterial Pathogens Associated with Community Acquired Pneumonia in Under 5 Children
—Dr. Amit kumar, Dr. Sushil Kumar Pathak, Dr. Ram Prakash Saha 47(A)

Clinical and Biochemical Study on Changes of Non Alcoholic Fatty Liver Disease
—Dr. Varsha Sinha 48(A)

Clinical Evaluation and Prognostic Factors in Cases of Acute Respiratory Distress and Chest Wheezing in Infants
—Dr. Shilpi Golwara 50(A)

Comparative Study of Intramedullary Supracondylar Nailing with Conservative Treatment in Cases of Distal Femoral Fracture
—Dr. Pawan Kumar, Dr. Vikash Ranjan 53(A)

Compasion of Amphotericin B Vs. Miltefosine Regarding Efficacy in Pediatric Cases of Visceral Leishmaniasis
—Dr. Arvind Kumar 58(A)

Development and Progression of Diabetic Retinopathy after Cataract Surgery
—Dr. Rajiv Kumar Singh, Dr. Kamlesh Sharma, Dr. Rakesh Kumar, Dr. Satyendu Sagar 61(A)

Epidemiology of Hepatitis B in Children Less than 12 Year
—Dr. Vimlesh Kumar 64(A)

Evaluation of Functional Outcome of Pre-Contoured Olecranon Locking Plate in Fractures of the Olecranon
—Dr. Pawan Kumar, Dr. Vikash Ranjan 66(A)

Evaluation of Management of Fractures of Long Bones of Lower Limb in Children by Titanium Elastic Nails
—Dr. Vikash Ranjan, Dr. Pawan Kumar 74(A)

Evaluation of Plasma Rich in Growth Factors as A Therapeutic Agent for Dry Eye
—Dr. Rajiv Kumar Singh, Dr. Kamlesh Sharma, Dr. Rakesh Kumar, Dr. Satyendu Sagar 76(A)

Fine Needle Aspiration Cytology (FNAC) is Excellent First Line Diagnostic methods for Head and Neck Swelling
—Dr. Prahalad Sharma, Dr. Satyendu Sagar, Dr. Anil Kumar[3] Dr. Manoj Kumar 79(A)

Microbial Spectrum and their Antibiogram in Paediatric and Adult Chronic Suppurative Otitis Media (CSOM)
—Dr. Rajesh Kumar, Dr. Satyendu Sagar, Dr. Prabhat Kumar 83(A)

Myocardial Dysfunction Predictors in Children with Scorpion Sting Envenomation In Bihar
—Dr. Amit kumar, Dr. Sushil Kumar Pathak 86(A)

Observation on Levels of Glycosylated Haemoglobin (HbA_{1c}) in Non-Insulin Dependent Diabetes mellitus (NIDDM) and Healthy people of North Bihar
—Dr. Prahalad Sharma, Dr. Satyendu Sagar, Dr. Anil Kumar[3], Dr. Manoj Kumar 87(A)

Prospective Randomised Comparision of One-Day Versus One-Hour Application of Topical Moxifloxacin in Eliminating Preoperative Conjun tival Bacterial Flora
—Dr. Rajiv Kumar Singh, Dr. Kamlesh Sharma, Dr. Rakesh Kumar, Dr. Satyendu Sagar 90(A)

Original & Clinical Research

Role of C-reactive protein & HbA1c in Type2 Diabetes mellitus in patients attending Jawahar Lal Nehru Medical College, Bhagalpur, Bihar
—Dr. Varsha Sinha 93(A)

Seroprevalence of Hepatitis B and Hepatitis C Virus Coinfection, among patients with chronic Liver Disease (CLD)
—Dr. Rajesh Kumar, Dr. Satyendu Sagar[2], Dr. Prabhat Kumar 97(A)

Study on Iron Deficiency Anemia and Its Relation to Neuro Developmental Status of Children 0 to 2 Years
—Dr. Vimlesh Kumar 99(A)

Zinc Status in Febrile Seizure
—Dr.Kaushalendra Kumar Singh, Dr.Binod Kumar Singh, Dr. Ram Prakash Saha 102(A)

Biochemical and Radiological Parameters of Pleural Fluid in Predicting the Treatment Outcome in Children with Empyema Thoracis
—Dr. Md. Nasim Ahmed, Dr. Anuradha Singh, Dr. Rishika Verma, Dr. Ram Prkash Saha 103(A)

A Comparative Study between Laparoscopic Hernia Repair and Open Lichtenstein Mesh Repair
—Dr. Manoj Kumar 109(A)

A Comparative Study of Intraoperative Retinoscopy and Biometry for Calculation of Power of Intraocular Lens
—Dr. Sunita Kumari, Dr. Umesh Kumar Sinha 112(A)

A Prospective Study on Risk Factors in Acute Respiratory Distress in Infants {2 Months to 1 year} Admitted in Pediatrics Department S.K.M.C.H., Muzaffarpur
—Dr. Vijay Kumar, Dr. Braj Mohan 115(A)

A Study on Clinical Profile of Patients of Hepatic Abscess
—Dr. Md. Aftab Alam 117(A)

Clinico-Demographic Study of Lichen Planus and It's Association with Other Dermatological & Non-Dermatological Diseases Including Hepatitis C Virus Infection
—Dr. Loknath Ghoshal, Dr. Aritra Sarkar, Dr. Alok Kumar Roy, Dr. Susmita Bhattacharya 119(A)

Clinico Pathological Profile of Significant Cervical Lymphadenopathy in Pediatric Age Group (1 Month to 12 Years)
—Dr. Krishna Murari, Dr. K. N. Mishra, Dr. Zoha Ansari, Dr. Rizwan Haider, Dr. Nitish Kumar, Dr. Chandan Kumar Mishra 123(A)

Comparative Study between the Efficacy of Conservative Treatment and Enoxaparin in Management of Unstable Angina
—Dr. Md. Aftab Alam 125(A)

Comparative Study of Nebulized Salbutamol versus Adrenaline in Wheeze Associated Respiratory Tract Infection in Infants
—Dr. Vijay Kumar, Dr. Braj Mohan 129(A)

Expandable Proximal Femoral Nails versus 95^0 Dynamic Condylar Screw-plates for the Treatment of Reverse Oblique Intertrochanteric Fractures
—Dr. Narendra Kumar Sinha 131(A)

Fracture of Talar Body Treated by Open Reduct and C.C. Screw Fixation
—Dr. Narendra Kumar Sinha 134(A)

Incidence and Outcome of Poisoning Patients in a Tertiary Care Hospital in New Delhi
—Dr. Sanjay Kumar, Dr. M. Walli 138(A)

Incidence of Gall Bladder Malignancy in Cholecystectomy Cases Found benign Clinically and Radiologically at Presentation
—Dr. Manoj Kumar 140(A)

Incidence of Tuberculosis among Patient of Bronchial Asthma Receiving Treatment with Metered Dose Corticosteroid by Inhalation
—Dr. Md. Aftab Alam 143(A)

Metabolic Changes in Psoriatic Skin under Topical Corticosteroid Treatment
—Dr. Pritam Pankaj 146(A)

Original & Clinical Research

Observation on Australia Antigen Positivity and Risk Factors of HBV Infection in a Sample of Apparently Healthy Mothers and their Infants
—Dr. Sanjay Kumar, Dr. Ashok Kumar Singh 149(A)

Observation on Fetomaternal Outcome in Cases of Obstructed Labour
—Dr. Reena Kumari, Dr. Ram Ratan Thakur 151(A)

Relation between Insulin Resistance and Lung Function: A Cross Sectional Study
—Dr. Shri Mohan Mishra, Dr. Kamlesh Tewary 153(A)

Study of Efficacy of Iron Sucrose in Severe Nutritional Iron Deficiency Anemia
—Dr. Shri Mohan Mishra, Dr. Kamlesh Tewary 156(A)

Study of Glycosylated Hb in Cases of Diabetes Mellitus with Microangiopathy
—Dr. Md. Aftab Alam 158(A)

Study of Maternal and Perinatal Outcome and Mode of Delivery following Induction of Labour in Eclampsia-Preeclampsia
—Dr. Reena Kumari, Dr. Ram Ratan Thakur 161(A)

Study of Modifiable Risk Factors for Acute Lower Respiratory Tract Infection in Under Five Children
—Dr. Zoha Ansari, Dr. K. N. Mishra, Dr. Krishna Murari, Dr. Rizwan Haider, Dr. Nitish Kumar, Dr. Chandan Kumar Mishra 164(A)

Surgical Management of Midclavicular Fracture
—Dr. Kanhaiya Lal Gupta, Dr. Pradeep Kumar Nayak 166(A)

To Evaluate the Combined Spinal Epidural Technique with Spinal and Epidural Anaesthesia in Patients Undergoing Abdominal Surgery
—Dr. Rajeev Babu, Dr. Sanjeev Kumar Sinha 170(A)

Topical Treatment with Fresh Human Milk Versus Emollient on Atopic Eczema Spots in Young Children
—Dr. Pritam Pankaj 173(A)

A Comparison of Intramedullary and Extramedullary Fixation Devices in Unstable Trochanteric Fractures
—Dr. Kanhaiya Lal Gupta, Dr. Pradeep Kumar Nayak 176(A)

Case Report

Case Report : Depression in Diabetes Mellitus
—Dr. Abhay Kumar Sinha 180(A)

Case Report: Diabetic Muscle Infarction
—Dr. Abhay Kumar Sinha 183(A)

Case 1: Intractable Hiccups in a Patient with Type 2 Diabetes Mellitus
—Dr. Abhay Kumar Sinha 185(A)

Case Report : Oculomotor Nerve Palsy in a Patient with Type 1 Diabetes Mellitus
—Dr. Abhay Kumar Sinha 187(A)

Study of Glycated Hemoglobin Levels in Diabetic Retinopathy Patients in a Rural Area of Bihar
—Dr. Abhay Kumar Sinha 191(A)

Isolated Splenic Hydatid Cyst-a Rare Entity
—Dr. Mukesh Bihari, Dr. Anand Kr. Murarka 192(A)

A Case of Celiac Disease with Cutaneous Lichen Planus – A Rare Co-existence of Two Common Entities
—Dr. Abhinav Kumar, Dr. Pravesh Yadav, Dr. Sunita Kumari 194(A)

CONCLUSION

Rural setting, low socioeconomic status ,improper breast feeding upto 6 months, improper immunization and low birth wt. for age comes to be major burden of Acute respiratory distress. It can be removed by education and general public awareness by which the burden of disease and cost of treatment can be minimized.

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ORIGINAL & CLINICAL RESEARCH

A Study on Clinical Profile of Patients of Hepatic Abscess

Dr. Md. Aftab Alam[1]

INTRODUCTION

The liver is the organ most subject to the development of abscess. Hepatic abscesses made up 13% of the total number of abscesses or 48% of the visceral abscesses [Harrison 17th ed, pg 811]. It may be bacterial, parasitic, fungal. It is present in all age groups but are more common in adult of 50-60 yrs in pyogenic liver abscesses and in adult of 20-40 yrs in amoebic liver abscesses [Cecil medicine 23rd ed, pg 1129]. Males are predominantly involved.

A pyogenic liver abscess is the most common form ,accounting for 40 to 60 % of cases, while amebic and fungal infection collectively comprise about 20 to 30 % of cases [Sherlock S et al, 1993]. Amoebic liver abscess which is caused by invasive strain of Entamoeba histolytica, is common in those people who are residing or travel to endemic area. It is prevalent in 3 to 10 percent of people with amoebiasis [Maltz G and Knauer CM,1991].

The patient usually presents with fever, right upper quadrant abdominal pain accompanied with nausea, vomiting, anorexia and weight loss. A physical examination discloses fever, hepatomegaly and liver tenderness which is accentuated by movement or percussion.

The present study has been taken up to observe clinical profile of patients with liver abscess reported in the JLNMCH, the change in trends of the site of involvement in liver and the incidence of amoebic liver abscess in toddy abusers.

MATERIAL AND METHODS

The present study on "Clinical profile of patients with hepatic abscess" was carried out in the Department of Medicine, Katihar Medical College and Hospital (KMCH), Katihar, Bihar. This study included ninety patients, out of 104 patients who were reported in Outdoor or admitted in Emergency Department. Remaining 14 patients were dropped out. The selection of the patients was based on clinical history, clinical examination and ultrasound findings. Those patients who were serologically positive for viral hepatitis, malaria (optimal test) and kala-azar (rK 39) were excluded from this study.

Each patient was subjected for detailed clinical history and clinical examination. In detailed clinical history, patients' name, age, gender, registration number and clinical symptoms with duration from onset and signs on thorough clinical examination were noted down.

The following investigations were done in all cases like CBC,ESR,USG whole abdomen,chest x-ray, LFT, RFT, Serological test for Entamoeba histolytica, Culture and sensitivity of the drained pus (in selected cases), routine examination of stool for Entamoeba histolytica, HIV I & II.

RESULTS

A total of 90 adult cases of hepatic abscess were selected from amongst the patients reported or admitted in the department of Medicine at Katihar Medical College and Hospital, Katihar, Bihar. They were evaluated for detailed clinical history, thorough clinical examination, radiological and laboratory findings and clinical response after treatment.

Study shows that pain abdomen and fever were most common presentation seen in 97.8% and 90% of patients respectively.

This study revealed that raised temperature (>98.6F) and local tenderness were most common physical signs seen in

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90% and 73.3% of patients respectively where hepatomegaly (50%) , icterus (15.6%), rales (8.9%), shifting dullness (4.4%) were less common among them.

Data shows that majority of cases [64/90] had leukocytosis (median -13,400) but 26 cases had white blood cells count within normal limit. Polymorphnuclear cells (median-83%) were predominantely present. Abnormally high alkaline phosphatase were seen in 74% of cases. The SGPT level [median-130] were mildly elevated. ESR was raised in 81 cases (90%). Routine examinations of stool for E. histolytica were positive in six cases. All cases were HIV-negative. Serum creatinin levels were within normal range in all cases.

It shows that among the 90 cases under study, 93.3% of patients had positive serological tests for Entamoeba histolytica. Qin SL et al (2000) studied in 36 cases with amebic liver abscess and noted that 92.6% of cases had positive serologies against Entamoeba histolytica.

In this study, all patients (100%) were toddy abusers. Seven cases of them were also alcoholic. There is no documented evidence which may show an association of toddy with amebic liver abscess. The growth of E. histolytica requires in cultures the presence of starch or rice floor and some metabolic associates, such as enteric bacteria, organisms (non pathogenic bacterium) or the parasitic flagellate T.cruzi, living or dead. It supposed that toddy might be the good culture media for Entamoeba histolytica.

Data revealed that about 95.6 % of patients had upto 10 cm size diameter of abscess while 4.4% of patients had larger size (>10 cm) abscess measured by ultrasonogram. The data showed that the most common site involved was the right lobe of the liver (90%). 6.7% of cases presented involvement of both right and left lobe. 3.3% patients had isolated left lobe abscess.75.6% of patients had single abscess where 24.4% had multiple lesions.

It showed that among 46 patients in which USG guided percutaneous needle aspiration were done for diagnostic and therapeutic purpose, 15 cases were culture-positive. Escherichia coli in 3 cases, Staphylococcus aureus in 6 cases, Bacteroides spp. in 2 cases, Peptostreptococcus spp in 1 case, Proteus spp. in 1 case, Enterobacter spp. in 1 case and Clostridium spp. in 1 case were present. Culture of remaining cases was sterile but recovery was fast after drainage.

The study showed two complications, pleuropulmonary rupture in one case, peritoneal rupture in one case and Budd-chiari syndrome in three cases. All survived after percutaneous drainage and antibiotic therapy. There was no mortality. These findings were consistent with other reported cases.

CONCLUSION

This study comprised of 90 patients of hepatic abscess from indoor and outdoor of department of medicine, KMCH, Bhagalpur. Each patient was evaluated for clinical history, thorough clinical examination, ultrasonographic study, biochemical paraemeter and serological markers.

The present study reveals the following observation :

1. There are 86.7 percent male and 13.3 percent female in this study. Age range varied from 19 to 65 years. The highest incidence was found in 31-40 year age group.
2. Maximum number of cases were from rural areas (92.2 percent) and remaining (7.8%) from urban.
3. 51.1 percent of patients presented their symptoms within one to two weeks of onset and 36.7 percent of them within two to three weeks.
4. Pain abdomen in right upper quadrant (97.8%) and fever (90%) were most common clinical symptoms whereas loss of appetite, nausea/vomiting, cough, and dyspnea were less common symptoms and usually seen in patients with larger size abscess.
5. Raised temperature (90%) and local tenderness (73.3%) were most common clinical sign and enlarged liver on palpation were seen in half of the cases. Icterus (15.6%) and rales (8.9%) were present in few cases. Shifting dullness were also seen in 4 cases.
6. Leuokocytosis (64/90) with neutrophilia (median - 83%) were present in most of them. Erythrocyte sedimentation rate (81/90) were also raised in maximum cases. Raised alkaline phosphatase (74%), serum AST (median -130) and bilirubin level (median – 1.1) were less common non specific findings.
7. There were six cases in which E. histolytica was detected in routine examination of stool. No single case was seropositive to HIV I & II serum creatinine level found to be within normal range in all cases.
8. 93.3% of cases was positive to serological test for E. histolytica. It means that out of 90, 84 cases were of amebic liver abscess and remaining six with negative serological marker and positive pus culture belonged to pyogenic liver abscess. Few cases with positive serological marker had positive pus culture suggestive of superinfecion.
9. All the patients were toddy abusers. It suggests an association with amoebic liver abscess. Six of them were alcoholic too.
10. Involvement of right lobe of liver and single abscess were most common ultrasonographic findings. There was also increasing incidence of multiple liver abscess. Isolated left lobe abscess were seen in three cases.
11. Out of 90 patient, 46 patient were undergone USG guided percutaneous needle aspiration. Recovery was fast in non-responder.
12. In liver abscess, E.coli, Bacteroides, Peptostreptococcus, Proteus etc, were common pathogens isolated from drained pus.
13. Budd-chiari syndrome was complication seen in 3 cases. It subsided after drainage of pus. Pleuropulmonary rupture in one case and peritoneal rupture in one case were also seen.
14. There was no mortality. Prognosis was better. It was due to early diagnosis of the diseases and early intervention.

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ORIGINAL & CLINICAL RESEARCH

Clinico-Demographic Study of Lichen Planus and It's Association with Other Dermatological & Non-Dermatological Diseases Including Hepatitis C Virus Infection

Dr. Loknath Ghoshal[1], Dr. Aritra Sarkar[2], Dr. Alok Kumar Roy[3], Dr. Susmita Bhattacharya[4]

ABSTRACT

INTRODUCTION : Lichen planus (LP) is a common, pruritic, inflammatory disease of the skin, mucous membranes, nails and hair follicles. Though lichen planus is primarily known as a dermatological disorder, possibility of existence of systemic connotations is unknown.

OBJECTIVES OF THE STUDY : To study the clinico-demographic profile of patients with LP, to identify non-dermatological (including metabolic disorders) and dermatological diseases that may be associated with LP and to identify association with Hepatitis C seropositivity, if any.

MATERIALS AND METHODS : The study was conducted at the departments of Dermatology and Biochemistry of Nilratan Sircar Medical College over 12 months from March 2013- February 2014.

All new consecutive patients, attending the Dermatology OPD and subsequently diagnosed as LP were recruited in the study after obtaining informed consent. Patients who did not express consent were excluded.

RESULTS AND ANALYSIS : The age of presentation ranged from 7 to 75 years. The mean age of presentation was 34.6± 15.4 years. Of the 100 patients of our study, 65 were females and 35 males; the male to female ratio was thus 1: 1.85.

DISCUSSION : Body mass index (BMI) distribution in the population (21.44) was comparable to the general Indian standards. Diabetes mellitus was found to be no commoner in LP patients than in the general population (P=0.12). The prevalence of dyslipidemia in LP patients was comparable to general population Hepatitis C serology was not associated with occurrence of LP (P=0.32).

Keywords: lichen planus, dermatological, hepatitis C

INTRODUCTION

Lichen planus (LP) is a pruritic, inflammatory disease of the skin, mucous membranes, nails and hair follicles. LP is characterized by typically polygonal, flat (hence, *planus*), and pruritic papules which occur commonly on the wrists, shins, lower back and genitalia.

Involvement of the mucosa, scalp and nails may lead to scarring. LP has a worldwide distribution and affects 0.22-1% of world population.[1]

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ORIGINAL & CLINICAL RESEARCH

Comparative Study between the Efficacy of Conservative Treatment and Enoxaparin in Management of Unstable Angina

Dr. Md. Aftab Alam[1]

ABSTRACT

The main concern of the medical fraternity in the treatment of unstable angina has been prevention of progress to the thrombosis over the ruptured atheromatous plaque, so that rupture of the plaque may heal without any sequelae. For this, use of oral anticoagulants became part of the treatment of unstable angina. But due to late onset of action of oral anticoagulants, results were not so rewarding. The real breakthrough came with use of heparin.

Heparin is a naturally occurring anticoagulant in the human body, produced by mast cells located in the pericapillary connective tissue throughout the body, especially in the lung, liver and skin.

Heparin was unexpectedly identified in 1916 by J. McLean, a young medical student at Johns Hopkins University, during the course of an investigation attempting identify the procoagulant substances. Howel named it heparin from the Greek word "Heper" meaning "Liver". It plays an important role in human physiology serving as a powerful anticoagulant. Though its concentration in blood is very little yet some of its anticoagulant effect are of physiological importances in many situation.

INTRODUCTION

Unstable angina is a clinical syndrome characterized by angina of new onset (within 2 month), angina at rest or with minimal exertion, or a crescendo pattern of angina with episodes of increasing frequency, severity or duration. Unstable angina may develop in a patient with a history of stable exertional angina or may occur in postmyocardial infarction setting. It may present in either of three ways e.g., angina of new onset or angina at rest or with minimal exertion or crescendopattern of angina with increasing frequency, severity or duration.

The pathophysiology of unstable angina is heterogenous though in most cases the transition from stable to unstable ischaemia appears to be due to rupture or fissuring of an atherosclerosis plaque, resulting in a thrombus formation, increasing platelets reactivity and increased coronary vasomotor tone.

Anginal pain was relieved in Heberden's time by Landanum, a preparation of opium or by alcoholic stimulant like brandy.

Modern era of therapy of angina began with the introduction of Amyl Nitrite. Laudern Brunton first referred to it in his article "On the Use of Nitrite of Amyl in Angina Pectoris" (1867). But the real revolution in therapy came with introduction of Nitroglycerine by William Murrel in his paper "Nitroglycerine as Remedy for Angina Pectoris" (1989).

Traditionally, unstable angina is treated by bed rest, aspirin, beta blocker, intravenous nitrate and sedatives. For the past few years either heparinization or angioplasty is getting more and more acceptance in most of the hospitals for treatment of unstable angina.

AIMS AND OBJECTIVES

The present study has been designed keeping in view the following aims and objectives –

(i) To determine the role of low molecular weight heparin in unstable angina in an Indian setting.

(ii) To determine the safety of low molecular weight heparin in such patient.

MATERIAL AND METHODS

The present study was carried out in Katihar Medical College and Hospital, Katihar, Bihar. The study include a total of 65 patients with unstable angina. Patients with acute chest pain due to unstable angina were selected from the ICCU and medical wards of Katihar Medical College

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and Hospital, Katihar, and admitted to the coronary care unit for the evaluation between March 2014 to February 2016.

Inclusion Criteria :

Patients were included in the present study on the basis of following criteria.

1. Age : The cases were selected in between the ages from 40 years to 65 years.
2. Sex : Both males and females were selected for the study.
3. Diagnostic criteria : Patients presenting with acute chest pain, anginal in nature, persisting more than 10 minutes or of recent onset (within 2 month) or occurring more frequently recently were selected. The ECG showing significant ST-T changes ranging from ST-T depression to ST elevation and T wave inversion or symmetrical tall T wave with changing pattern were taken up should have been cardiac enzymes were negative for acute myocardial infarction. Thus only patients with chest pain as well as ECG changes and negative for cardiac enzyme were included in the study.
4. Patients with hypertension and diabetes were taken for the study provided blood pressure and blood sugars under good control.
5. Patients with past history of myocardial infarction (> 1 year before) were also taken for the study, if they presented with typical anginal chest pain, but negative for cardiac enzymes.
6. Informed consent was taken.

Exclusion Criteria :

Patients were excluded on the basis of following criteria. -

1. Age : if > 65 years.
2. Uncontrolled hypertension (> 180/110 mm Hg).
3. Severe left ventricular failure with pulmonary oedema.
4. History of bleeding disorder or active peptic ulcer disease or haemorrhagic stroke.
5. Severe hepatic or renal disease.
6. Pregnancy.

Design of the Study :

All patients with unstable angina were admitted in the Coronary Care Unit, put on cardiac monitor and given standard antianginal treatment which included bed rest, Aspirin (325 mg initially then 150 daily), Nitroglycerin infusion, Beta-adrenergic blocker, Calcium channel blocker as and when required.

Patients were randomized to one of the two groups, Group A of 40 patients were kept on standard antianginal treatment plus low molecular weight heparin (Enoxaparin 1mg/kg) subcutaneously twice daily for 6 ± 1 days from the 1st day of admission. Group B of 25 patients were given only standard antianginal treatment and kept as the control group.

The patients were monitored daily during the period of hospitalization and after discharge, on the 14th, 28th and on 42nd day. All the patients were evaluated clinically, electocardiographycally and biochemical for and cardiac enzymes as per the detailed performa given below :

The results of the two group were compared. For statistical purpose only the 1st, 2nd, 3rd, 7th, 14th and 42nd day findings were analysed by standard statistical methods.

PROFORMA OF OBSERVATION

Case No. -

Registration No. -

I. Particulars of Patients -

1. Name –
2. Age –
3. Sex –
4. Marital Status : Married/Unmarried -
5. Occupation –
6. Address –

II. History -

1. Duration of chest pain – Hours or days
2. Mode of onset –Acute/Subacute/Chronic
3. Progress –
4. Symptoms with duration –
   i. Breathlessness Grade - Duration
   I -
   II -
   III -
   IV -
   ii. Palpitation – On rest/ On exertion
   iii. Cough - With expectoration/without expectoration
   iv. Swelling of feet.
   v. Pain abdomen.
5. History related to aetiology –
   i. Dyslipidemia - Duration
   ii. Cigarette smoking – Duration/ Amount
   iii. Alcohol – Duration/Amount
   iv. Hypertension – Controlled/Duration, Uncontrolled
   v. Diabetes – Controlled/Duration, Uncontrolled
   vi. Myocardial infarction.
   vii. Others.

On examination :

i. Vital signs
- Pulse
- B.P.
- Temperature.
- Res.

ii. Examination of cardiovascular symptoms

1. Apex beat
2. JVP
3. Swelling of feet
4. Liver
   - Size
   - Tender/Non-tender
   - Pulsatile/Non-pulsatile.
5. Heart Sound
   - S1
   - S2
   - S3/S4
6. Murmur
7. Bilateral crepts - Yes/No
8. Other

III. Investigations –

1. Hb gm%
2. TLC/cmm
   Poly Lymph. Eiosino. Mono. Baso.-
   ESR mm. 1st Hr.
   mm. 2nd Hr.

3. Blood : Urea mg% Sugar mg%
4. Serum
   Sodium meq/L
   Potassium meq/L
   Chloride meq/L
   Calcium mg%
   Phosphate mg%
   Creatinine mg%
   Total protein g% A : G Ratio
   SGOT
   Alk. Phosphatase
   Lipid profile

IV. **Cardiac Enzymes -**

a. CK-MB
b. Troponin T
c. Troponin I

V. **X-ray chest** – PA view

VI. **ECG -**

VII. **Treatment given –**

VIII. **Follow up –**

- Pulse
- B.P.
- JVP
- Oedema of feet
- Liver size
- S3 gallop
- Murmur
- Bilateral crepts – Yes/No
- X-ray chest
- ECG
- Troponin T
- CK-MB
- Troponin I.

RESULTS

The age range of the patients in the present study varied between 42 to 65 years of age. Maximum number of patients were over 50 years of age. As shown in the table and corresponding graph, the mean age of the study group (Group A) was 62.5 years, while that of the control group (Group B) was 60.38 years. This difference is not statistically significant.

There were 17 cases (42.5%) of smokers in the study group, while there were 10 (40%) smokers in the control group. Thus, the number of non-smokers in the study group was 23 (57.5%) while the number of non-smokers in the control group was 15 (60%). This indicates that the percentage of smokers and non-smokers in the study and control group was almost equal. The minor difference observed between these two groups in the present series is statistically not significant.

The total number of diabetic patients in the study group and the control group was 7 (17.5%) and 4 (16%) respectively. While the number of nondiabetics in the study and control group was 33 (82.5%) and 21 (84%) respectively. The difference is statistically insignificant.

The corresponding graph that only 21 (52.5%) cases of the study group and 13 (52%) of the control group were hypertensive. While 19 (47.5%) cases of the study group and 12 (48%) cases of the control group were non hypertensive. This difference is statistically not significant.

The lipid profile of 25 (62.5%) patients in study group was within normal limit, while 15 (37.5%) patients of the same group were dislipidemic whereas lipid profile of 15 (60%) cases of the control group was within normal limit and 10 (40%) cases were dyslipidemic. This can be clearly appreciated.

As far as past history of the angina is concerned 26 (56%) cases of the study group and 16 (64%) cases of the control group were having past history of angina. While there was no history of angina in 14 (35%) cases in the study group and 9 (36%) cases in the control group.

It can easily be observed that the 7 cases (17.5%) and 4 cases (16%) from the study group and the control group respectively were having a definite history of myocardial infarction now presenting as cases of unstable angina. Thus in 82.5% of cases (33 cases) of study group and 84% of cases (24 cases) of control group there was no past history of myocardial infarction.

Twelve lead ECG was done for all the patients after admission. Baseline ECG changes were observed in the form of ST depression in 29 (72.5%) cases, ST depression with T inversion in 5 (12.5%) cases, T inversion with near normal ST segment in 4 (10%) cases and other changes such as tall T wave in 2 (5%) cases in group A versus 29 (72.5%) cases, 3 (12%) cases and 1 (4%) cases in group B respectively. These difference were not significant statistically.

After discharge and during the course of follow up cardiac enzymes were assessed on 42nd day. CK-MB was negative in all the patients while tropinin-T was positive in 1 (2.5%) and 5 (20%) patients in group A and group B respectively. As tropinin T is a definite and specific marker of myocardial infarction, it can easily be concluded that at least these patients (1 of group A and 5 of group B) had an episode of myocardial infarction at least 2 weeks after discharge or after the 28th visit. Among the 5 patients of Group B with elevated Troponin T on day 42, there had already taken admission to the ICCU in the 4th to 5th week and were available to follow up on the 42nd day. Their ECG's showed definite MI changes and their course of treatment altered accordingly. As for the other 2 from Group B and the only patient from Group A, they all had silent information with no increase in the intensity of chest pain and all were diabetics. They all had persistent ECG changes.

Clinical response was assessed as relief of chest pain among the two groups. 31 (77.5%) cases in group A had chest pain on 2nd day while 23 (92%) in group B continued to have chest pain on the same day. This difference however was not significant statistically significant (P value > 0.05). From the 3rd day onwards more patients had good clinical response in group A as compared to group B. On 3rd day only 47.5% of cases (19 cases) in group A had pain, while 80% cases (20 cases) of group B had chest pain. This difference was statistically significant (T value 2.60, P value < 0.01). On the day 7th and 14th, 11 (27.5%) cases and 6 (15%) cases of group A had pain respectively while 16 (64% cases and 13 (52%) cases of group B had chest pain on day 7th and 14 respectively This difference was statistically significan (On the 7th day t value 2.90, p value 0.01 and on the day 14, t value 3.19, P value < 0.001).

This difference persisted till th conclusion of the study on 42nd day whe only 4 (10%) cases of group A still ha chest pain as compared to 9 (36%) case

of group B, (t value 2.55, P value < 0.05).

At the beginning of the study all the patients in both the group had ECG abnormalities. These ECG abnormalities gradually improved or returned to normal in both the groups in course of time with treatment. However the change reversed more rapidly in the group treated with enoxaparin. On the 2nd day 33 (82.5%) cases in group A still had ECG changes as compared to 24 (96%) in group B. The ECG abnormalities became statistically significant 48 hrs after the treatment with low molecular weight heparin. On the 3rd, 7th, 14th and 42nd day of treatment 21 (52.5%), 15 (37.5%), 7 (17.5%) and 6 (15%) patients respectively in group A still had ECG abnormalities as compared to 21 (84%), 18 (72%), 15 (60%) and 11 (44%) in group B. These differences were statistically significant. (P value < 0.01 on 3rd, 7th and 42nd day and P< 0.001, 14 day of treatment).

As a consequence of treatment 35 (87.5%) patients in group A had became asymptomatic by the 42nd day of assessment as compared to only 11 (44%) patients in group (P value < 0.001) In Timi IIB, Enoxaparin was use as 30 mg IV bolus initially followed by 1mg/kg sc BD on sample size of 390 patients. The end results on 14th day was 14.2% vs 17.5% in the present study but on the 43rd day end result was 17.3% vs 12.5% on 42nd days.

In the ESSENCE trial conducted by Cohen et al (1997) in which Enoxaparin was used in the same dose (1mg/kg SCBD) as in this study, showed end result (MI death and recurrent angina) 16 6% on 14th day vs 17.5% in present study (P<0.05). This is approximately equal. But at the end of study on 30 day (in ESSENCE trial) end result was 19.8% vs 12.5% on 42nd day which appears better.

This difference may be due to the larger number of patients (3171) in ESSENCE trial, as compare to only 40 patients in the present study.

The result of present study is in conformity with the observations of Cohel et al (1997).

Out of 40 cases observed in the study group and 25 cases for the control group, there was not a single major bleeding episode in either group. although epistaxis was observed in a single patient in the study group that consistutes an incidence of 2.5% of the total. Since this patient had received enoxaparin for 5 days he was including in the study. Epistaxis improved on stopping enoxaparin the following day.

In the ESSENCE study, conducted by Cohen et al (1997) also there was no serious bleeding but there was about 4% minor bleeding including ecchymosis at the site of injection.

CONCLUSION

It is therefore concluded that Enoxaparin an LMWH is a highly effective, safe and potent therapeutic tool in the management of unstable angina, and should be recommended as a routine therapy in this condition.

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Association News

- 248th CME programme will be held at NRS Medical College & Hospital at 2 PM held on 23rd April 2016. All hon'ble life members are requested to attend the meeting.

(Pinaki Kr. Ghosh)
Secretary, General, AIGPA

Incidence of Tuberculosis among Patient of Bronchial Asthma Receiving Treatment with Metered Dose Corticosteroid by Inhalation

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ABSTRACT

Objectives : To study the incidence and pattern of tuberculosis in Bronchial asthma patients receiving inhalational corticosteroid therapy by MDI.

Method : Eighty four patients taking MDI steroid for bronchial asthma and seventy eight patients suffering from the similar disease but not receiving MDI steroids were followed up for twenty months to study the incidence and pattern of tuberculosis.

Results : Five patients (5.5%) receiving MDI steroid developed tuberculosis as against non among the control ($p<0.05$). out of five patients, who developed tuberculosis, two developed sputum smear positive pulmonary disease, one had sputum smear negative disease and two had extra pulmonary tuberculosis in form of pleural effusion and gland tuberculosis one each. All patients were treated with standard antituberculos therapy using RNTCP guide line and all patients recovered from the disease.

Conclusion : Inhalational corticosteroid in the form of MDI causes a significant increase in incidence of tuberculosis.

INTRODUCTION

Tuberculosis is a serious public health problem in India causing immense morbidity, mortality and distress to individuals families and community. About 40% of patients is infected with tuberculous bacilli in India as judged by positive tuberculin skin test. One fifth of global tuberculosis incidence is in India, with 1.9 million new cases occurring every year and 0.87 million of these being infectious smear positive cases. In India an estimated 2.76 lakhs deaths occur from tuberculosis every year.

The predisposing factors for tuberculosis are corticosteroid therapy, immunosuppressive and anticancer therapy, co-infection with HIV, alcoholic, presence of diabetes mellitus, renal transplant, gastrojejinostomy patients and not the list is alcoholic persons.

Bronchial asthma supposed to be an allergic disease in which different allergense by a set of immunological reactions superimposed by neurological actions cause release of mediators of inflammation causing bronchial hyperresposiveness, mucus hypersecretion and paroxysmal attacks of bronchoconstriction manifesting as wheeze, cough and dyspnoea. Anti inflammatory drugs like oral and inhalational corticosteroids in form of MDI play a major role in abuting an acute attack of asthma as well as they are useful in management of chronic asthma. Corticosteroid used in MDI are beclomethasone diapropinate (100 µg, 200 µg), Budesonide (100 µg, 200 µg), fluticasone propionate (250 µg) and Formaytarole (200 µg, and 400 µg) etc. have local anti-inflammatory effects and are useful in management of bronchial asthma.

Corticosteroids cause systemic disease by impairing antibody production and cell mediated immunity, and there by blunting the patients response to infections. So treatment with MDI in bronchial asthma might facilited infection with micobacterium tuberculosis in the form of either reinfection or reactivation.

This study evaluates the possible role of corticosteroid in causing tuberculosis in patients with bronchial asthma neccesating use of MDI therapy.

MATERIAL AND METHODS

Study was done in Katihar Medical College and Hospital, Katihar, Bihar. One hundred patients suffering from bronchial asthma and taking MDI corticosteroid therapy were inrolled in the study group. After taking detailed history, clinical examinations including relevant examination and investigations were done, to diagnose tuberculosis. Anti-tuberculous therapy was started after diagnosis of tuberculosis. An equal number of age and sex matched patients suffering from asthma but not taking MDI corticosteroids constituted control group. Patients of both groups were followed for twenty months. Patients with preexisting tuberculosis or giving past history of in take of antituberculos therapy (ATT) were excluded from study.

All patients in study and control groups were followed for twenty months at four monthly intervals. On each follow up visit response of MDI corticosteroids therapy and development of fresh symptoms such as fever, cough, expectoration, hemoptasis, loss of weight, loss of appetite and breathlessness were recorded. Chest roentgenogram were done at each visit for assessment of emergence of fresh pulmonary infections and for evaluation of previous shadows. All suspects developing fresh respiratory symptoms and infiltrates in X-rays were subjected to sputum examination for acid fast vaccilise (AFB) using Ziehl-Nelson stain on two consecutive days. Additional tests such as plueural fluid analysis, pleural biopsy, fine needle aspiration cytology and/or biopsy of enlarged nodes, bronchoalveolar lavas (BAL), transbronchial lung biopsy and CT

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scan of chest were also done for confirmation of tuberculosis whenever required. All subjects confirmed or strongly suspected to have developed tuberculosis were started on antituberculous chemotherapy using RNTCP regimen as per WHO guidelines.

Comparison between two groups were carried out using student t-test for continuous variables and X^2 test for categorical variables. Statistical significance were tested at a level of $p<0.05$. In addition, multivariate logistic regration analysis were carried out to identify risk factor(s) responsible for development of tuberculosis.

RESULTS

Out of the two hundred subjects (100 cases and 100 controls), the age and sex distribution are summarized in table 1. During study 16 cases and 22 controls did not come for regular follow up and were subsequently excluded from analysis.

Table 1 : Patients characteristic in the study population

Patients characteristic	Cases	Controls
Mean Age	46.2±14.0	36.8±16.6
Male	38 (45.25%)	46 (58.97%)
Female	46 (54.76%)	32 (45.02%)

The dose of MDI ranged from 200 µg to 800 µg/day and the duration of treatment varied from two months to twenty months.

Five (5.9%) out of the 84 cases developed tuberculosis as against non of the controls ($p<0.05$). Of these 5 patients three developed pulmonary tuberculosis (two were sputum smear positive and one had clinicoradiological evidence), one developed pleural effusion and another one developed cervical lymphadenopathy confirmed positive for tuberculosis by FNAC. All patients who developed tuberculosis were treated successfully with RNTCP regimen according to WHO guideline.

Multiple logistic analysis were done to evaluate risk factors like age, sex, underline disease, maximum dose of inhalational corticosteroid, duration of treatment and additional supportive therapy. The incidence of tuberculosis did not show significant association with any of these variable.

Table 2 : Details of Five patients who developed tuberculosis during the study period

	Case 1	Case 2	Case 3	Case 4	Case 5
Age (yrs.)	50	44	48	28	35
Sex	Female	Male	Male	Female	Male
Max. daily dose	400mg	200mg	400mg	800mg	600mg
Duration in months	8	20	12	16	8
Site of Tuberculosis	Pulmonary	Pulmonary	Pleural effusion	Cervical gland	Pulmonary
Sputum smear for acid fast bacilli	Positive	Negative	--	--	Positive
Outcome	Improved	Improved	Improved	Improved	Improved

DISCUSSION

Over the years, several studies have been carried out to determine the influence of corticosteroid therapy in development of tuberculosis but non is done with inhalational corticosteroid therapy.

In 1971, a joint statement of American Thoracic Society, National Tuberculosis and Respiratory Disease Association and Communicable Disease Centre, commented that there is danger of reactivation of latent tuberculosis or developing re-infection with micobacterium tuberculosis after therapy with corticosteroid and recommended that patients with healed pulmonary tuberculosis receiving systemic corticosteroid should receive isoniazid profylaxis.

Am J Respir. Crit. Care Med. 2011, also published the study of effects of inhaled corticosteroid and risk of pulmonary tuberculosis and found no positive correlation. American College of Chest Physician in October 15, 2012, published in chest journal that inhaled corticosteroid is associated with an increased risk of tuberculosis in patients with chronic obstructive pulmonary disease (COPD).

Corticosteroids, through their immunosuppressive and anti-inflammatory effects on many organ systems, impair antibody formation and cell mediated immunity. They transiently sequester T-cells, decreased monocyte, lymphocyte, basophil, eosinophil count in peripheral blood, and reduce polymorphonuclear inflammatory response. They also inhibit cytocine production through the effects on lymphocyte and monocyte and adrlitionally block the effects of cytocine on some target cells. Through these actions corticosteroid predisposes patients to a variety of secondary infections, reactivation of latent tuberculus infection and reinfection with micobacteria tuberculosis. These effects on cells are more evident if inhaled corticosteroid doses exceeds 1000 mg/day. The therapy is given continuously for longer period. The prolonged therapy has more profound immunosuppressive activity as compare with intermittent therapy.

In our study, five cases out of 84 cases (5.55%) on MDI corticosteroid therapy developed tuberculosis. Out of 5 cases 2 were sputum smear positive (40%). The zero incidence of tuberculosis in control group (88 cases) is not surprisingly in light of estimated risk of tuberculosis of <1%/ year in general population.

The duration of MDI corticosteroid therapy before development of tuberculosis in the present study varied from 4 months to 20 months. This suggest that reactivation can occur either shortly after therapy is started, or several months or year later. In all three cases that developed pulmonary tuberculosis in this study, the appearance of fresh symptoms led to the suspicion of tuberculosis. A high index of suspicion is necessary for early detection of tuberculosis in these patients.

The region for sputum negativity in one patient with pulmonary tuberculosis in this study could be antibiotics therapy (Ceprofloxacine and Amikacin) received by the patient before the diagnosis of tuberculosis. These drugs are second line anti tuberculosis drugs.

All the patients 100% in this study responded well to standard short course anti tuberculus chemotherapy under guidelines of RNTCP.

We conclude that there is the statistically significant increase in incidence of tuberculosis due to inhalational corticosteroid therapy, specially in areas with high prevalence of tuberculosis such

as Bihar. The disease, however, response well to standard anti tuberculosis chemotherapy. Duration and dose of inhalational corticosteroid also influences the development of tuberculosis. A longer study, involving a large study population, would be desirable for further verification of these results.

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Study of Glycosylated Hb in Cases of Diabetes Mellitus with Microangiopathy

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INTRODUCTION

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia (Polyuria, Polydipsia, Polyphagia) and glycosuria resulting from defective insulin secretion or insulin action or both leading to disturbance in carbohydrate, protein and fat metabolism. Vascular disorders are the major long term complications of diabetes that result in increased morbidity and mortality. These vascular disorders can be broadly categorized into microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (coronary, peripheral and cerebrovascular) disease.

Three hypothesis are being investigated that potentially explain the mechanism by which high glucose level can result in vascular damage.

One hypothesis is that increased intracellular glucose leads to the formation of advanced glycosylation end products (AGP) via the non-enzymatic glycosylation of cellular proteins. The serum level of AGEs correlates with the level of glycemia and these product accumulate as glomerular filtration rate declines. Second hypothesis is sorbitol theory. Increased sorbitol concentration affects several aspect of cellular physiology and may lead to cellular dysfunction.

Third hypothesis proposes that hyperglycemia increases the formation of diacylglycerol leading to activation of certain isoforms of protein kinase. Which in turn affect a variety of cellular events that lead to diabetes mellitus related complication.

There are no excellent biochemical markers with high predictive value for future microangiopathy. The best available markers today are glycosylated haemoglobin (Hb A_1 C) and urinary albumin. Hb A_1 C concentration reflects mean blood glucose levels during last 3 months before examination.

The worsening of complications in this study is defined as increase of one or more steps in four stages of modifies ETDRS (Early treatment diabetes retinopathy study) interim scale for retinopathy and an increase of one or more steps in three stages. (normoalbuminuria < 30 mg/24 hr.; microalbuminuria 30-300 mg/day, macroalbuminuria > 300 mg/day) for nepropathy. For neuropathy loss of tendon reflexes in the lower limbs, deminished perception of vibration sensation distally, glove and stocking impairment of all other modaleties of sensation.

AIM AND OBJECTIVE

The aim of this is to evaluate the clinical significance of measurement of glycated haemoglobin (HbA_1C) in the assessment of metabolic control in diabetes mellitus with microangiopathy.

MATERIAL AND METHODS

The present work is carried out in the department of Medicine and Pathology, Katihar Medical College & Hospital, Katihar, Bihar and in some private clinics on newly diagnosed cases of diabetes. (Fasting plasma glucose >126mg /dl two hour plasma glucose >200 mg/dl. during an oral glucose tolerance test, HbA1C > 6%).

SELECTION OF THE PATIENTS :

In the present study, 80 cases of well known diabetes were selected at random and comprised both sexes in different age groups, age ranging from 20 years to 80 years . First of all these diabetic patients were screened for micro angiopathy i.e. (retinopathy, nephropathy or neuropathy.) The duration of diabetics in the patients varied from 1 month to 21 years before the time of detection.

Twenty cases of non –diabetic, formed control group varied from 20 years to 80 years of age. They were also screened for diseases like haemolytic anaemia, iron deficiency anaemia , jaundice , chronic renal, failure. Which defect glycosylated haemoglobin level, such were not included in the control group.

In the group of patients of diabetes without evidence of microangiopathy, 30 cases were studied. In the group of patients of diabetes with microangiopathy (Retinopathy, Nephropathy, Neuropathy) 50 cases were studied.

The examination of all these patients was done as follows :

Name of Patient, Age of patient, Sex, weight, Religion, Address, Occupation, Chief complains and clinical history in detail, Past Medical History, Family History, Drug History.

Examination :-

1. General
2. Systemic.

In general examination, more stress was given on pulse rate and change after the valsalva manoever, B.P. in lying down and standing position.

Retinopathy was detected by direct opthalmoscopic examination of fundus with pupil dilated. The worsening of complications in this study was defined as an increase of one or more steps in the 4 stages of the modified ETDRS interim scale for retinopathy.

Classification of Diabetic Retinopathy :- (ETDRS Classification)

ETDRS –Early treatment Diabetic Retinopathy Study :-

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1. **Non- proliferative Diabetic Retinopathy (NPDR) :-**

A. **Mild NPDR** : Atleast 1 microaneurysm (MA).

B. **Moderate NPDR** : Hard exudates, Venous beading and intra retinal microvascular abnormalities(IRMA) definitely present.

C. **Severe NPDR**

- Haemorrhages / microaneurysms in all four quadrants of retina.
- Venous beading in 2 or more quadrants.
- IRMA in atleast 1 quadrant.

D. **Very severe NPDR** : Any 2 or more of C.

METHODS

There are different methods used to measure glycosylated haemoglobin can be divided into three broad groups.

I. METHODS BASED ON CHARGE

II. METHODS BASED ON STRUCTURAL CHANGES

III. METHOD BASED ON CHEMICAL REACTION

IV. ION EXCHANGE SEPARATION METHOD

The glycosylated haemoglobin (HbA) test was done by HUMAN KIT : supplied by Human Gesellschaft fur biochemical and Diagnostica mbH Max Planck Ring 21-D65205 Wiesbaden Germany. The corresponding blood glucose level was estimated by glucose oxidase /peroxidase (GOD/POD). Kit method.

RESULTS

The present study was carried out in 100 cases. Out of this, 20 healthy non-diabetics, 30 cases of diabetes mellitus without microangiopathy and 50 cases of diabetes mellitus with microangiopathy.

The patients group made for this study are as follows.

(1) Normal healthy control : This group was having total patients. Out of that 12 (60 %) male and 8 (40 %) females.

(2) Patients of diabetes without microangiopathy : This group was consisting 30 patients with equal sex distribution i.e. 15 (50 %) male and 15 (50 %) females.

(3) Diabetic with microangiopathy.

(a) Retinopathy : This group was having 15 cases with 10 (67 %) males and 5 (33 %) females.

(b) Retinopathy with Nephropathy and Neuropathy : In this group also there were male predominance i.e. out of 19 cases 12 (63 %) were males and 7 (37 %) were females.

(c) Nephropathy group was having 16 cases in which sex distribution was 10 (63 %) males and 6 (7 %) females.

The incidence of microangiopathy is more 32 (64 %) in the age group above 60 years, than other groups like 13 (26 %) in the age group 41-60 years and 5 (10 %) in the age group less than 40 years.

The incidence of Retinopathy is 8 (53 %) in the age group more than 60 years, 4 (27 %) in the age group 41-60 years and 3 (20 %) in age group less than 40 years.

The incidence of combined microangiopathy i.e. Retinopathy with nephropathy and neuropathy is 14 (73 %) in age group more than 60 years, 3 (16 %) in age group 41-60 years and 2 (11 %) in the age group less than 40 years.

The incidence of Nephropathy is 10 (62 %) in the age group more than 60 years, 6 (38 %) in the age group 41-60 years.

The above data shows that incidence of combined microangiopathy i.e. Retinopathy with Nephropathy and Neuropathy is more than isolated microangiopathy.

In control group, the mean glycosylated Hb level at beginning is 5.12 and after 3 month, 4.8 %. There is no marked difference between this two value.

In diabetes mellitus without microangiopathy, the mean glycosylated Hb is 8.5 % at beginning and 7.57 % after 3 months of treatment.

In Retinopathy group, the mean glycosylated Hb is 10.89 % at beginning and 8.86 % after 3 months. In diabetics with combined microangiopathy i.e. Retinopathy with Nephropathy and Neuropathy the mean glycosylated Hb is 12.73 % at beginning and 10.52 % after 3 months.

In Nephropathy group, the mean glycosylated Hb is 11.76 % at beginning and 8.39 % after 3 month. In this study we found that HbA_1C levels in all three groups of diabetes with microangiopathy category were on higher side than those of diabetes without microangiopathy. It is always more than normal healthy individuals. This reflects the view that HbA_1C level may be better indicator than blood sugar levels is assessing the severity of the disease.

The blood sugar levels are indicative of one time values. While HbA_1C reflects a time bound value of approximately 8-12 weeks. A large study may be of proven HbA_1C value in two category i.e. diabetes with microangiopathy and without microangiopathy.

HbA_1C level reflects the average blood glucose level during the previous 8-12 weeks, hence may fail in giving an accurate evaluation of blood glucose control throughout the entire duration of the disease.

The argument for the non-significant increase in HbA_1C in diabetes with microangiopathy is that, once complication develops in patients, he is more aware of treatment and patient attempt to control hyperglycemia.

The mean fasting blood glucose level in normal healthy individual varies from 70 mg % to 120 mg % among 20 persons. The corresponding glycosylated Hb varies from 4 % to 6.5 %. The mean fasting blood glucose level is 92.35 mg % and glycosylated Hb level is 5.12.

The fasting blood glucose level in diabetes mellitus without microangiopathy varies from 135 mg % and 255 mg %. The corresponding glycosylated Hb level varies from 6.5 to 10.4 %. Mean fasting blood glucose value is 197.2 % and glycosylated Hb is 8.5 %.

The fasting blood glucose level of Retinopathy cases varies from 236 mg % to 302 mg % mean is 266.93 mg %. The corresponding glycosylated Hb level is 9.6 % to 21.1 %. The mean is 10.89 %.

The fasting blood glucose level in diabetes with combined microangiopathy varies from 270 mg % to 378 mg %. The mean is 322.1 mg %. The corresponding

Hb level is 11 % to 14.6 %. The mean is 12.73 %.

The fasting blood glucose in nephropathy cases varies from 240 mg % to 315 mg %. The mean is 289.81 mg %. The corresponding glycosylated Hb level is 10.1 % to 12.5 %. The mean is 11.76 %.

These table show that fasting blood glucose and glycosylated Hb level is more in diabetes with microangiopathy cases than normal individuals.

The blood glucose levels (fasting and post-prandial) at the beginning of study and after 3 months treatment. It also shows the corresponding HbA_1C level at the beginning of study and after three months of treatment. This observation shows that there is excellent relation in fasting blood glucose levels and HbA_1C level in both category i.e. diabetes with microangiopathy or without microangiopathy. In this study we find that blood glucose level declines sharply after treatment but blycosylated Hb does not full such rapidly.

The duration of diabetes and incidence of microangiopathy. This study show, that duration of diabetes was more prolonged in cases of microangiopathy as compared to the duration in group of diabetes without microangiopathy. Same table also shows the mean HbA_1C level which indicates that there is no significant correlation between duration of diabetes and HbA_1C concentration.

The comparison of mean fasting blood sugar in beginning of study of normal, diabetes mellitus without microangiopathy and diabetes mellitus with microangiopathy. This study shows that mean fasting blood sugar of diabetes mellitus without microangiopathy and diabetes mellitus with micropathy is higher than normal people. 'p' value is < 0.001 which is highly significant.

The comparison of mean glycosylated haemoglobin of normal, diabetes mellitus without microangiopathy and diabetes mellitus with microangiopathy. 'p' value is < 0.001 which is highly significant.

CONCLUSION

1. The three months follow up study showed a significant fall in HbA_1C level in all groups of diabetes with and without microangiopathy. But it does not touch to normal level 4-7 % of total Hb.

2. HbA_1C level reflects average blood glucose concentrations of previous two to three months in diabetics with or without microangiopathy.

3. Microangiopathy does not hamper to decline of HbA_1C levels and attainment of good metabolic control.

4. Glycosylated Hb level does not flutuate in relation to diet, exercise and anti diabetic treatment on the day of testing.

5. Assessing Glycaemic control in diabetes with high glycosylated haemoglobin levels, concurrent fasting blood glucose level estimations are essential.

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27, Dixon Lane, Kolkata 700014, Phone : +91-033-22270102

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Ref. No. 2513/V/2016.

Dated : 07/03/16.

To,
DR. (Major) Durga Shankar,
Associate Professor, Dept. of Medicine,
KMCH, Katihar, Bihar.

SUB: "Evaluation of renal function in hepatic failure."

Respected Sir/Madam,

We would like to inform you that the above mentioned article has been accepted suitable for publication in IMJ (NIC/BID/ICMR/JR/ 233 Dt. 14.12.12, INDEX MEDICUS by NIC, NEW DELHI, GOVT. OF INDIA).
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SUB: "Observation of renal functions in HIV positive patients and its relationship with CD 4 count."

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To,
DR. (Major) Durga Shankar,
Associate Professor, Dept. of Medicine,
KMCH, Katihar, Bihar.

SUB: "A clinical study of hepatorenal and haematological profile in falciparum malaria in KMCH, Katihar, Bihar."

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INDIAN MEDICAL JOURNAL

A Clinical Study of Hepatorenal and Haematological Profile in Falciparum Malaria in K.M.C.H., Katihar, Bihar

Dr. (Major) Durga Shankar[1], Dr. Vishal Parmar[2]

ABSTRACT

BACKGROUND AND OBJECTIVE: Malaria is a major health problem in India. This study was conducted in Tumkur district of Karnataka, which is endemic for malaria. Several factors have been attributed to increased morbidity and mortality in malaria of which hepatorenal and haematological parameters take an important role.

MATERIAL AND METHODS: 50 patients of malaria underwent detailed clinical history, thorough physical examination and were investigated with haematological, hepatic and renal parameters. This was followed by monitoring the outcome of patient with respect to morbidity and mortality.

RESULTS: Anaemia was seen in 52% of patients. Normocytic normochromic blood picture was the most common type seen in our study. Thrombocytopenia was seen in 38% of patients. Mild thrombocytopenia was common and was seen in 76.31% of patients. The total leucocyte count was normal in majority of the patients. Out of the 11 patients with jaundice, majority had conjugated hyberbilirubinaemia. Renal failure was seen in 2.77% of falciparum malaria patients due to acute renal failure. One patient in our study expired, as he had multi organ dysfunction.

CONCLUSION: Severe anaemia is a poor prognostic factor and has adverse outcome. Thrombocytopenia and hepatic dysfunction alone does not have correlation with mortality. Hepatic dysfunction as a part of multi organ dysfunction especially with renal failure has poor outcome.

KEY WORDS: *Malaria; Plasmodium Falciparum; Plasmodium Vivax; Anaemia; Thrombocytopenia; Conjugated hyperbilirubinemia; acute renal failure.*

INTRODUCTION

Malaria is one of the major public health problems of the country.

In manifestation of severe Plasmodium Falciparum Malaria the signs may include severe normocytic, normochromic anemia, renal failure, cerebral malaria, acidosis, acute respiratory distress syndrome, hypoglycaemia, jaundice, hyperparasitemia, disseminated intravascular coagulation(DIC), haemoglobinuria convulsions and shock.

The hepatorenal parameters indicating poor prognosis in severe malaria are elevated serum creatinine >3 mg/dl, total bilirubin > 3mg/dl, elevated liver enzymes (aspartate transaminase/ alanine transaminase >3 times upper limit of normal).

Malaria affects kidneys leading to both tubulointerstitial damage as well as glomerulonephritis. Acute renal failure due to acute tubular necrosis occurs in falciparum malaria. Glomerulonephritis in malaria is due to Plasmodium malariae. Though nephrotic syndrome is commonly associated with Plasmodium malaria it can also be seen with other malarial species. Renal impairment is common with adults with severe Plasmodium falciparum malaria, most commonly presents as acute renal failure.

Hepatic involvement commonly presents as jaundice which can be due to intravascular haemolysis of the RBC, DIC, microangiopathic haemolysis and hepatitis.

The haematogical parameters indicating poor prognosis are leucocyte count > 12,000/microlt, severe anemia (Haemoglobin <5gm/dl) and coagulopathy.Common hematological abnormalities seen are anemia, thrombocytopenia with coagulopathy. Pancytopenia reflects hypersplenic state in malaria due to increased peripheral destruction of all cell lineages.

Since malaria is quiet common in Darbhanga which often leads to complications,this study was conceived to know the hepatorenal and hematological profile, and hence the degree of complications that can arise from these deranged parameters in this part of the country.

AIMS AND OBJECTIVE

1. To determine the haematological parameters in patients with malaria.
2. To determine the hepatic parameters in patients with malaria.
3. To determine the renal parameters in patients with malaria.
4. To correlate the clinical and biochemical profile of hepatic, renal and haematological dysfunction with final outcome.

MATERIAL AND METHODS

SOURCE OF DATA

Confirmed cases of Falciparum Malaria admitted in wards at Katihar Medical College and Hospital, Katihar. 50 clinically and Microscopically proven cases of Falciparum Malaria would be studied over a period of one year

METHOD OF COLLECTION OF DATA

A case study of patients with falciparum malaria, to delineate the clinical and biochemical characters of hepatic, renal

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and haematological involvement which involves- detailed history followed by clinical examination.

Malaria patients will be investigated by doing complete haemogram, blood urea, serum creatinine, liver function tests and ultrasound abdomen.

Patients in this study were proved to be cases of malaria either by peripheral smear examination (both thick and thin smear) or MPQBC.

The following investigations for haematological and hepatorenal parameters were carried out :

Complete haemogram: Haemoglobin estimation, total count and differential leucocyte count, total platelet count, erythrocyte sedimentation rate estimation and peripheral smear.

Renal function test: Blood urea, serum creatinine and input/ output chart.

Liver function tests (total bilirubin, direct biriribin, indirect bilirubin, aspartate aminotransferase, alanine aminotransferase, total protein, serum albumin and serum globulin estimation)

Additional investigations: Ultra sound abdomen, lumbar puncture for cerebrospinal fluid analysis and random blood sugar.

INCLUSION CRITERIA

Clinically and Microscopically proven cases of Falciparum malaria admitted to medicine ward.

EXCLUSION CRITERIA

1. Cases with abnormal liver function tests with positive viral markers, cirrhosis and hepatotoxic drug use
2. Known case of acute/chronic renal failure due to any other cause.
3. Bleeding diasthesis

Study period : One year. (December 2014 to November 2015)

Statistics used : Non-parametric tests will be used to analyze the data.

RESULTS

The present clinical study on hepatorenal and haematological profile in falciparum malaria was conducted in the department of medicine, at Katihar Medical College and Hospital, Katihar. 50 consecutive malaria cases were studied.

Majority of the patients in our study belong to age group of 20-50 years and male to female ratio was 1.50: 1. Fever was the most common presenting symptom (96%) followed by chills and rigor (70%). Nausea and vomiting (36%), Headache (48%), Altered sensorium (8%) and cough was present in 4% of patients.

The most common clinical sign seen were pallor in 68% followed by splenomegaly 42%, then hepatomegaly in 26% and CNS involvement in form of seizures and altered sensorium in 26%, Icterus in 22% and pedal oedema in 8% of the patients.

Anaemia was seen in 70% of patients of falciparum malaria, severe malaria was seen in 10% of patients. Normocytic normochromic blood picture was the most common type seen (62.85%)

Thrombocytopenia was seen in 48% of patients. Mild thrombocytopenia is very common and is seen in 75% of patients.

Total leucocyte count is normal in 94% of patients and leucopenia is seen in 2% of patients and leucocytosis in 4% of patients.

Of the 11 patients with jaundice majority had conjugated hyberbilirubinaemia (90.9%) with increased transaminase is seen in 81% especially mild raise of ALT (>3 times) and normal serum proteins, albumin and globulin.

Renal failure was seen in 2% of falciparum malaria patients.

Death associated with falciparum malaria as a consequence of multi organ dysfunction.

CONCLUSIONS

The incidence is higher in males than in females with peak incidence in 2nd and 3rd decade.

- Fever is the presenting complaint in almost all the cases.
- Pallor and splenomegaly are the important signs in malaria.
- Anaemia is the most common haematological abnormality
- Thrombocytopenia is very common in malaria, Even though malaria is commonly associated with thrombocytopenia, rash and petechial hemorrhages in the skin or mucous membranes are not the common presentation features.
- Jaundice can be either conjugated or unconjugated hyberbilirubinaemia. Conjugated hyberbilirubinaemia with raised transaminase especially ALT is the most common pattern seen.
- In patients with falciparum malaria renal involvement is seen in form of acute renal failure.
- Severe anaemia, liver failure and acute renal failure as a part of multi organ system failure are poor prognostic factors and it increases the risk of mortality.

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Comparative Study on the Incidence of Proteinuria in Cases of Pulmonary Tuberculosis before and After Anti-Tubercular Therapy

Dr. (Major) Durga Shankar[1], Dr. Vishal Parmar[2]

INTRODUCTION

Pulmonary tuberculosis is still a giant killer in underdeveloped countries. It accounts for 12% of mortality and 25% of morbidity in Afro-Asian countries (WHO).

Apart from clinical, radiological and sputum evidence of pulmonary tuberculosis other attempts have been made to find out the correlative evidences of tuberculosis. Positive tuberculin test and recent Immunological tests have been found to be helpful.

Increase in urinary proteins in pulmonary tuberculosis is one of these correlative findings. Proteinuria is seen in numerous infectious diseases, metabolic and renal diseases. Proteinuria was first noted in cases of pulmonary tuberculosis in 1924 by Holten et al. They made a large observation and followed the patients from a fortnight to three years in their observation. Proteinuria was a common finding in cases of pulmonary tuberculosis and it disappeared with anti tubercular treatment. The disappearance of urinary protein was almost parallel with the response of anti tuberculous treatment.

Similar observations by Kennedy et al (1974), R.P. Somvanshi et al (1989) and Gracia et al (1990) have been published in the corresponding years. All of them had observed significant proteinuria in cases of pulmonary tuberculosis and its disappearance with anti tubercular treatment. The degree of fail in urinary protein was proportional to the duration of anti tubercular treatment.

AIMS

The present series of work is aimed to study the followings :

(A) The incidence of proteinuria in cases of pulmonary tuberculosis.

(B) Whether urinary proteinuria is relative to the degree and duration of anti tubercular treatment.

(C) Whether urinary proteinuria can be used as a prognostic index in cases of pulmonary tuberculosis.

(D) Whether proteinuria is related with the success of anti tuberculous treatment.

(E) To show that even kidney is not spared in pulmonary tuberculosis, and,

(F) Whether renal involvement is reversible or not.

MATERIAL AND METHODS

The present study as conducted to observed the comparative incidence of proteinuria in cases of pulmonary tuberculosis before and after antitubercular therapy.

MATERIAL :

SCREENING AND CASE SELECTION : Cases for this study were selected from patients admitted into male and female Medical wards as well as patient admitted in C.C.W. and those attending out-patient Department of Medicine, K.M.C.H., Katihar, Bihar.

CRITERIA FOR SELECTING A CASE OF PULMONARY TUBERCULOSIS :

1. Sputum smear positivity for Acid Fast Bacilli.
2. Radiological examination (X-ray chest P.A. view).
3. Clinical parameters
4. Haematological examination :
5. Quantitative estimation of 24 hours urinary protein (by TCA/Biuret method).

EVALUATION OF PATIENTS : All those patients who had been selected for the study group were evaluated before treatment in the following manner :

1. Name : Address : Age : Sex : Registration No. :
2. History :
3. History of present illness.
4. History of past illness.
5. Treatment history.
6. Socio-economic history.
7. Family history.

CLINICAL EXAMINATION :

(A) General Examination :

(B) Systemic Examination :

(I) RESPIRATORY SYSTEM :

Inspection :

Palpation :

Percussion :

Auscultation :

(II) CARDIO-VASCULAR SYSTEM :

(III) GASTROINTESTINAL SYSTEM :

(IV) CENTRAL NERVOUS SYSTEM :

INVESTIGATIONS :

1. Sputum examination for AFB.
2. X-ray chest P.A. view.
3. Routine examination of urine for

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proteinuria.

4. Quantitative examination of 24 hours urinary protein.
5. TLC, DLC of WBC, Hb%, ESR.

Other Investigation – Mantoux test.

SPUTUM EXAMINATION FOR AFB :

METHOD : Ziehl Neelsen.

Carbol fuschin, 20% sulphuric acid, distilled water, Loeffler's methylene blue.

Procedure : The sputum of suspected patient was collected for 24 hours for 3 consecutive days. Sputum was collected in a wide mouth container free antiseptics. Smear were made on a clean and fresh glass slide from the thick purulent part of the sputum rather than from thin watery part. Then the smear were dried, fixed and stained by Ziehl Neelsen technique.

- Smear was covered with carbol fuschin gently heated to steaming and dried (without boiling).
- Slide washed with water.
- Decolourised with 20% sulphuric acid till no more stain comes off.
- Washed with 95% ethanol for 2 minutes.
- Counterstained with Loeffler's methylene blue for 1 minute.
- Dried and examined under oil immersion objective.

AFB were seen as bright red rods against the blue blackground, NB-At least 50,000 to 1,00,000 AFB should be present per ml of sputum for them to be readily demonstrable in direct smears.

A negative report should not be given till at least 100 fields have been examined.

A positive report can be given only if 2 or 3 typical bacilli have been seen, smears have been graded depending on the number of bacilli seen :

1+ : When 3-9 bacilli are seen in entire smear.

2+ : 10 or >10 bacilli seen in the whole smear.

3+ : When 10 or >10 bacilli in most oil immersion fields.

X-RAY EXAMINATION OF CHEST POSTERO-ANTERIOR VIEW :

EXAMINATION OF URINE :

QUALITATIVE TEST FOR PROTEINURIA :

BOILINGT AND ACETIC ACID TEST :

Other methods are :

(a) Sulfosalicylic acid test.

(b) Commercial reagent strip test.

QUANTITATIVE ESTIMATION OF 24 HOURS URINARY PROTEIN:

TCA/BIURET METHOD :

CALCULATION :

Mg per colorimeter of Tube determined From standard Graph ×

$$\frac{\text{Total volume specimen}}{\text{ml urine used In step 3}} \times \frac{\text{lg}}{1000 \text{ mg}} = \text{g/24 hours.}$$

CORRECTION : Protein excretion rate may be corrected for the patients deviation from average adult body surface area (1.73 m^2) by –

G/24 hours ×

$$\frac{1.73 \text{ m}^2}{\text{Patient's body surface area in m}^2}$$

The surface are can be determined from height and weight of the patient either from the formula of Du Bois and Du Bois or, from a table prepared from this formula.

Normal value : 100 to 150 mg/24 hours of this about one third of the protein is plasma albumine, while one forth represents mucoprotein (Tamm. Horsfall) and the remainders plasma globulins.

METHOD

Thus, 50 freshly diagnosed cases of pulmonary tuberculosis were subjected to the estimation of 24 hours urinary protein before initiation of specific antibuberculous therapy and was repeated monthly for 3 months after starting antituberculous drugs. All 50 cases were put on following treatment regimens :

1st 2 months :

- Rofampicin (10mg/kg upto maximum 600 mg daily)
- INH (5 mg/kg upto maximum 300 mg/daily).
- Pyrazinamide (25 mg/kg upto maximum 1500 mg/daily).
- Ethambutol (15 mg/kg upto maximum 1200 mg/daily).

Next 4 months : Rifampicin INH

After conducting the study, the were analysed statistically to find o followings :

I. To determine the inciden significant proteinuria in pulm tuberculosis without eviden prior renal involvement.

II. Judging the response of antitube therapy on proteinuria.

III. To show whether proteinu pulmonary tuberculosis due to involvement is reversible or n

RESULTS

The present study was conduct diagnosed patients of pulmo tuberculosis both in O.P.D. and admit Medical Ward of K.M.C.H.

The study included 50 case pulmonary tuberculosis, 40 males ar females ranging between 15 to 60 ye age. All the 50 cases were pu antitubercular treatment and subject the estimation of 24 hour urinary pr before initiation of antitubercular treat and was repeated monthly for 3 m during antituberculous therapy.

Before treatment urinary protein estimated in each case of the study g of 50 cases; 19 patients (38%) had incre urinary protein. 62% of the cases normal 24 hour urinary protein. B starting treatment, the mean urinary pr of the study group was 0.85±0.19 g hour. The mean urinary protein 1 m after treatment was 0.73±0.16 gm/24 and it was further low as 0.43±0.09 g hour 2 months after treatment. And af months of treatment it was 0.27±0.06 24 hour.

When compared statistically there a significant fall of (P<0.001) urinary pr before and after 1 month of treatm There was further significant fall (P<0. in urinary protein after 2nd and 3rd m of antitubercular treatment. With t statistical comparison it is evident that t was a statistically significant fal proteinuria after antitubercular treatm The degree of fall of proteinuria statistically significant (P<0.001) duration of antitubercular treatment. T was minimum fall after 1st month

treatment and the fall was maximum after 3 months of treatment meaning thereby that the fall was directly proportional to the duration of antitubercular therapy.

Thus, it could be inferred that urinary proteinuria due to renal involvement was an important finding and it was related with antitubercular treatment and its duration and it may be used as a prognostic index as well as it can reflect the response of antitubercular treatment.

CONCLUSION

Lastly, it was concluded that renal involvement in pulmonary tuberculosis is reversible after completion of antitubercular treatment.

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ORIGINAL & CLINICAL RESEARCH

Effect of Yogic Training on Physiological Variables in Working Women of Bhagalpur

Dr. Arohi Abhinav Jayaswal[1]

ABSTRACT

In today's world most of the persons are in high level of stress. Working women have high level of stress than non working women. Increasing amount of work stress at home and work place and its impact on family and home environment can be seen, which affect their emotional, psychological and physical health. The concept of yoga is helpful for reducing anxiety and improving cardiorespiratory parameters has created a great interest in the medical research field. The present study was conducted to assessing the effect of yogic exercises and meditation in working women. Yogic session was carried out for 16 weeks. Cardiorespiratory parameters (pulse rate, respiratory rate, blood pressure and breath holding time) were measured before and after yoga training. Stress was measured by anxiety score as an indicator of stress, also Visual reaction time (VRT) as an indicator of cognitive function and finger dexterity score as an indicator of motor skills were measured before and after yoga training. Statistical analysis was done by paired't' test. It was found that statistically significant improvement in cardiorespiratory parameters, anxiety score and visual reaction time after yogic training. Thus, a combined practice of asana, breathing exercises, and meditation & relaxation technique in a sequence is the best available resource to meet the present day needs of society.

Key words- *Systolic Blood Pressure, Diastolic blood pressure, Visual ReactionTime, Breath HoldingTtime, Pulse Rate, Respiratory Rate*

INTRODUCTION

Stress is considered to be a crucial trigger for physical and mental illness (1). Mind influences the body in profound manner: this forms the basis of psychosomatic origin of disease. Since 1968, women lifestyle have changed in many ways, many more women now work outside the home (2). Women mental health is different from men in so many ways. A female has to go through different psycho physiological changes resulting in hormonal issues. And to add to this she is also expected to give birth to children, nurture them, educate them and make them good citizens while also doing her office work, house chores and taking care of husband and of course in laws, especially in our society (3). Women are more sensitive than their spouse to the equality of their family relationship and tend to devote enormous amount of emotional energy to maintain intimate relationship. These women are finding it increasingly difficult to balance home and work, so more attention should be invested in working women (4).

Stress causes an imbalance of the parasympathetic and sympathetic nervous system due to psychic stimuli which lead to disturbances of homeostasis in the body (5). Here comes the role of yoga which not only improves the physical but the mental stress as well as establishing equilibrium between the sympathetic and parasympathetic components. Fewer studies worked out on women health problems due to stress, Hence the present study is attempted to see the effect of yogic exercises and meditation in working women. This of

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Evaluation of Renal Function in Hepatic Failure

Dr. (Major) Durga Shankar[1], Dr. Vishal Parmar[2]

INTRODUCTION

Austin Flint (1863) first recognized an association between decompensated cirrhosis and oliguric (volume 300-500 cc) renal failure. Merklin (1913) first used the term hepatorenal syndrome (Syndrome : hepatourologic syndrome, hepatic death syndrome, urohepatic syndrome, hepatonephritis serosa acuta, cholemic nephrosis, Bile nehrosis, Flint's syndrome, Heyd's syndrome, Functional renal failure of cirrhosis). Papper (1983) defined hepatorenal syndrome as unexplained renal failure in patients with advanced liver diseases.

In spite of large functional reserve and regenerative capacity acute hepatic failure is caused by viral hepatitis, hepatotoxic drugs and chemicals, massive liver necrosis of unknown cause. Chronic hepatic failure occurs most commonly in cirrhosis of liver. Besides causing general symptoms like anorexia, nausea, vomiting and fever, hepatic failure causes jaundice, hepatic coma, ascites, fetor hepaticus, endocrine imbalance etc.

Arroyo V, Gines P, Gerbes Al, et al (1996) gave definition and diagnostic criteria of hepatorenal syndrome in ascites.

5th Pan American Congress of Gastroenterology (1956) gave the criteria of hepatic failure, which is being followed in this work is as follows :

1. Jaundice
2. Ascites
3. Precoma and coma
4. Low serum albumin level
5. Prothrombin deficiency not corrected by vit. K.

Richard D. Morre et al (1986) defined liver disease by the presence of at least three of the following six criteria.

1. An aspartate aminotransferase level greater than twice Normal (Normal range 0-35 IU/L).
2. A total serum bilirubin level greater than 2.5 mg/dl.
3. A serum albumin level less than 30 gm/L.
4. An elevated alkaline phosphatase level (Normal range 0-90 IU/L).
5. A prothrombin time more than 15 seconds (Normal, 11.5 seconds).
6. Ascites.

He further defined renal dysfunction as an increase in the serum creatinine level of more than 0.5 mg/dl if initial value was less than 3.0 mg/dl or an increase of more than 1.0 mg/dl if initial value was more than 3.0 mg/dl.

Hepatorenal syndrome occurs when there is a decrease in kidney function in person with a liver disorder. This is often exhibited by decreased urine production. Nitrogen containing waste products accumulate in the blood stream (azotemia).

The exact cause of hepatorenal syndrome is unknown. There is an unclear relationship between the liver and the kidney, but in hepatorenal syndrome there is a drastic reduction in blood flow to the kidneys (Kew MC, Brunt PW, Varma RR et al 1971).

The kidney structure remains essentially normal (1863, Austin Flint) and the kidneys often will instantly function well if the liver disease is corrected for example by liver transplantation (Arroyo V, 2000). Hepatorenal syndrome develops in approximately 15% patients within 6 months of first hospitalization with ascites and 40% within 5 years (Gines A, Escorsell A, Gines P, 1993). It may be a sign of impending death caused by the accumulated effect of liver damage and kidney failure in people with acute liver failure, cirrhosis or alcoholic hepatitis. It is diagnosed when other causes of kidney failure are ruled out.

Risk factors include cirrhosis, alcoholic hepatitis, acute liver failure, recent abdominal paracentesis, gastrointestinal bleeding, use of diuretics and the presence of orthostatic hypotension.

AIMS AND OBJECTIVE

The aim of the present study is to evaluate Renal function in hepatic failure patients and also, if possible, to ascertain the cause of renal failure. This will help in better understanding of disease and better management of the patient, hence improving prognosis.

MATERIAL AND METHODS

The present study, "Evaluation of Renal Function in hepatic failure" was carried out in Department of Medicine, Katihar Medical College and Hospital, Katihar, Bihar.

Twenty controls without suffering from any disease were selected from the staffs of Katihar Medical College and Hospital and the attendants of the patients. They were investigated for serum sodium, serum potassium, serum chloride, serum calcium, blood urea, serum creatinine and GFR (by endogenous creatinine clearance rate) in Department of Pathology, Biochemistry of this hospital.

Forty patients of hepatic failure were studied just after admission to the hospital. Criteria as laid down by Pan-American Cogress of Gastroenterology (1956) for hepatic failure was followed. This was by the presence of 1. Jaundice 2. Ascites 3. Precoma and coma 4. Low serum albumin level. 5. Prothrombin deficiency not corrected by vitamin K administration.

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Patients were divided into 3 groups –

Group I – Hepatic failure due to viral hepatitis – twenty two cases.

Group II – Hepatic failure due to cirrhosis of liver – fourteen cases.

Group III – Hepatic failure due to miscellaneous causes eg.

Malignancy of liver – 1 case

Porto caval anastomotic – 1 case.

Drug induced hepatitis – 2 case.

The patients were clinically examined according to the following schedule : Name, Age, Sex, Marital status, Social status, Occupation, Date, Address, Presenting complains, History of present illness, Family history : history of jaundice in the family, Social history : Domestic hygiene etc., Past history : Particularly jaundice etc.

Personal history : Alcohol, drug etc.

1. General Examination :
2. Abdomen :
3. Cardiovascular system : Apex beat, Heart sounds, Gallop rhythm, Murmurs.
4. Respiratory system : Respiration rate, Breath sounds, Added/Adventitious sounds
5. Central Nervous System :

Following investigations were done :

Haemogram :

1. Total R.B.C. count.
2. Total W.B.C. count.
3. Differential W.B.C. count.
4. Total platelet count.
5. Hemoglobin estimation.

Liver Function Tests :

1. Serum bilirubin
2. S.G.P.T.
3. Alkaline Phosphatase
4. Total protein
5. Paper electrophoresis of serum protein.
6. Prothrombin time.

Renal Function Tests :

1. Blood urea
2. Serum cretinine
3. G.F.R. (Glomerular filtration rate by endogenous creatinine clearance).
4. Special – 24 hour urinary collection.

Serum electrolytes :

1. Serum sodium
2. Serum chloride
3. Serum calcium
4. Serum potassium

METHODS :

1. **Blood urea :** When urea is heated with compounds containing two adjacent carbonyl groups, such as diacetyl CH_3 $CO.CO.CH_3$; coloured products are formed Diacetyl monoxime has been used because of its greater stability.

Urea reacts with diacetyl monoxime in strongly acid medium to form yellow coloured complex. The reaction is intensified by ferric ions and thiosemicarbazide and the red colour produced is more linear with the concentration of urea. The method covers the range of 0-300 mg/dl, within this range the optical density is linear with the concentration.

II. **Estimation of creatinine in blood :** After precipitating protein, the creatinine is absorbed on to Lloyd's reagent, a hydrated aluminium silicate and the colour then developed with alkaline picrate.

III. **Estimation of creatinine in urine :** Creatinine when heated with picric acid in an alkaline medium was converted into creatine picrate which is an orange red compound. The red colour produced is proportional to the amount of creatinine present.

IV. **Glomerular filtration rate (G.F.R)** was calculated by the endogenous creatinine clearance, by the following formula.

$$\frac{U \times V}{P} \text{ ml per minute.}$$

Where,

U = Creatinine concentration in urine (mg/100 ml).

V = Urine volume per minute (ml/min).

P = Plasma concentration of creatinine (mg/100 ml).

V. **Determination of sodium and potassium by Flame Photometer :**

VI. **Estimation of chloride (Schales and Schales) :**

VII. **Estimation of serum calcium**

Principle – The calcium of seru precipitated as calcium oxalate w ammonium oxalate is added. Calc oxalate when treated with sulfuric a liberates equal amount of free oxalic a which is then titrated against a stan postassium permanganate solution presence of sulfuric acid. The oxalic ac oxidized to CO_2 and water.

RESULTS

The mean blood urea level in hep failure due to viral hepatitis (group I), hep failure due to cirrhosis of liver (group hepatic failure due to miscellaneous c (group III), and controls were 38.77 mg 38.7 mg/dL, 27.5 mg/dL and 24.55 m respectively. The mean blood urea lev hepatic failure due to viral hepatitis 0.01) and hepatic failure due to cirrhos liver ($p < 0.05$) was significantly highe compared to other study groups.

The mean serum creatinine leve hepatic failure due to viral hepatitis (g I), hepatic failure due to cirrhosis of (group II), hepatic failure due miscellaneous cause (group III) and con were 1.96 mg/dl, 1.6 mg/dl, 1.25 m and 0.929 mg/dl respectively. The m serum creatinine in hepatic failure du viral hepatitis group I ($p < 0.0001$) hepatic failure due to cirrhosis of group II ($p < 0.01$) was significantly hi as compared to other study groups.

The mean glomerular filtration rat hepatic failure due to viral hepatitis g I (Mean 90.86 ml/min, $p > 0.5$), hep failure due to cirrhosis of liver, grou (mean 84.81 ml/min, $p > 0.1$), hep failure due to miscellaneous cause, g III (mean 90.29 ml/min, $p > 0.5$) controls (mean 92.825 ml/min) v statistically insignificant in all the s groups.

The mean serum sodium level in hep failure due to viral hepatitis group I (m 130.8 mEq/l, $p < 0.1$), hepatic failure to cirrhosis of liver, group II (mean 1 mEq/l, $p > 0.1$), hepatic failure du miscellaneous cause, group III (mean mEq/l, $p > 0.05$) and control (mean 1 mEq/l) was statistically significant in hepatic failure due to viral hepatitis compared to other study groups.

The mean serum potassium level in hepatic failure due to viral hepatitis, group I (4.23 mEq/L, $p > 0.1$), hepatic failure due to cirrhosis of liver, group II (4.41 mEq/L, $p > 0.05$), hepatic failure due to miscellaneous cause group III, (4.25 mEq/L, $p > 0.05$) and controls (mean 4.0 mEq/L) was statistically insignificant in all the study groups.

The mean serum calcium level in hepatic failure due to viral hepatitis, group I (9.15 mg/dl, $p > 0.1$), hepatic failure due to cirrhosis of liver, group II (9.65 mg/dl, $p > 0.1$), hepatic failure due to miscellaneous cause, group III (9.28 mg/dl, $p > 0.5$) and controls (9.37 mg/dl) was statistically insignificant in all the study groups.

The mean serum chloride level in hepatic failure due to viral hepatitis, group I (100.63 mEq/L, $p > 0.1$), hepatic failure due to cirrhosis of liver, group II (104.14 mEq/L, $p > 0.1$), hepatic failure due to miscellaneous causes, group III (107.5 mEq/L, $p < 0.1$) and control (102.95 mEq/L) was statistically significant in hepatic failure due to miscellaneous causes, group III as compared to other study groups.

CONCLUSION

On the basis of above mentioned observations it can be concluded that renal failure can occur in patients of hepatic failure due to viral hepatitis and hepatic failure due to cirrhosis of liver. Electrolyte changes can also occur in patients of hepatic failure due to viral hepatitis and hepatic failure due to miscellaneous causes and cirrhosis of liver.

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ORIGINAL & CLINICAL RESEARCH

Evaluation of Serum Prolactin Assay in Infertile Women of North Bihar

Dr. Prachi Singh[1]

INTRODUCTION

Infertility is a global issue in reproductive health. A WHO scientific group has suggested that following definition of infertility– "failure to achieve a pregnancy after 12 months or more of regular unprotected sexual cutercourse".

Prolactin is a 198-amino acid protein (23 kD) produced in lactotroph cells and anterior pituitary gland. Although identified about half a century ago in vertebrates, it's existence as unique human prolactin remained controversial until it was finally purified away from contaminated pituitary growth hormone (Hwang et al, 1971).

AIMS AND OBJECTIVES

In this study an attempt has been made to identify and characterize the hyperprolactinemic state in patients with amenorrhea, oligomenorrhea, luteal phase defect and regular cycle with infertility whether or not associated with galactorrhea, patient with regular cycles were also selected; as it is well known that hyperprolactinemia in milder degrees may not affect the menstrual rhythm. But patient with even mild hyperprolactinemia and regular cycle may harbor a pituitary microadenoma or may have been associated with hypothyroidism. Hence it is essential to screen such patients too. In addition women with normal menstrual cycle and reproductive performance were also selected to define the normal range of prolactin in our population.

MATERIALS AND METHODS

160 women, presenting with infertility either primary and secondary, with or without menstrual dysfunction, attending the outpatient of Obstetrics and Gynaecology ward of Aayushman Hospital, Muzaffarpur, Bihar, were included in the study group. Serum prolactin estimation was done by Elisa method. Patients with regular cycles were also selected as it is well known that hyperprolactinemia in milder degree may not affect regular cycles may harbour a pituitary microadenoma, hence it is essential to screen such patients too. 50 women with normal menstrual cycle and fertility were selected to form the control group to define the normal range and mean serum prolactin in our population. The radioimmuno assay of prolactin was carried out in the department of Pathology of Darbhanga Medical College and Hospital.

Clinical Study :

History –

Menstrual History –

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Observation of Renal Functions in HIV Positive Patients and its Relationship with CD_4 Count

Dr. (Major) Durga Shankar[1], Dr. Vishal Parm

INTRODUCTION

Much has been learned about the pathogenesis and treatment of HIV-associated renal diseases because of the development of animal models and the molecular evaluation of clinical samples. Although the pathogenesis of HIV-associated nephropathy is clearly linked to the viral illness, over the next decade we must determine how infection results in the development of disease. HIV peptides rather than infection may be more important in nephropathogenesis. We must determine why some patients are susceptible to disease development and others are not. Genetic factors, the host response, and effects of HIV peptides on podocytes, on renal cellular apoptosis, and on the ability to present antigen may be critical to pathogenesis. Although highly active antiretroviral therapy will play an important role in preventing and treating HIV-associated nephropathy, well-designed and – controlled clinical trials are necessary to determine the roles of therapy with glucocorticoids and ACE inhibitors. Knowledge about the treatment of HIV-infected patients with renal transplantation and proper treatment of HIV-associated renal diseases is rudimentary.

The etiologic agent of AIDS in Human Immunodeficiency virus, which belongs to the family of human retroviruses (Retroviridae) and subfamily of Lentiviruses.

Human immunodeficiency viruses are two types :

1. HIV – 1
2. HIV – 2

The most common cause of HIV disease throughout the world is HIV-1.

First case of acquired immunodeficiency syndrome (AIDS) was identified nearly 29 years back when healthy individuals were developing unusual and dramatic opportunistic infections and cancers known to occur in immunosuppressive states.

This clinical consequences of HIV infections encompass a spectrum ranging from an acute syndrome associated with primary infection to a prolonged asymptomatic state to an advanced disease. It is best to regard HIV disease as beginning at the time of primary infection and throughout progressing through various stages.

Apart from a variety of opportunistic infections and cancers, HIV involves almost each and every organ system in one or another way, leading to derangement of function related with that particular organ system.

AIMS AND OBJECTIVES

The present study was undertaken to evaluate the derangement of renal function due to HIV in patients having different level of CD4 count.

MATERIAL AND METHODS

Present study was conducted on 90 patients who were diagnosed of having HIV-1 by Elisa and admitted in different units or undergoing treatment on OPD basis at ART centre, and Katihar Medical College & Hospital, Katihar, Bihar, for the various complications of HIV infections.

Distribution of Cases :

90 patients were viewfully allocated into 3 groups, each containing 30 patients on the basis of their CD4 count.

- Group A (n=30) - CD4 count more than 350.
- Group B (n=30) - CD4 count between 350-200.
- Group C (n=30) - CD count below 200.

Now each group has been subdivided into 2 subgroups, that is x and y on basis of receiving HAART or not.

- Subgorup X – Receiving HAART
- Subgroup Y – Not Receiving HAA

Hence in each group A, B, and subgroup.

- X – Taking HAART
- Y – (30-X) – Not taking HAART

SELECTION OF THE CASES

Inclusion Criteria : The cases selected only after they are proved t infected by HIV-1 on the basis of H Elisa test.

Relevant history, thorough phy check-up and biochemical tests are to rule out any pre-existing acute or ch renal problem in the selected patient

Exclusion Criteria : The study exc the patients having DM, HTN, preg or previous known renal disease that c acute or chronic renal failure, as to the possibility of obscuring the findi well as results of the study.

Parameters of Study : Parameters are studied to make assumptions o functions in HIV patients are –

1. Blood urea level
2. Serum creatinine level
3. 24 hr urinary protein/micral
4. Glomerular filteration rate (G

1. Associate Professor, Department of Medicine, Katihar Medical College & H Katihar, Bihar.
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Using Cockcroft Gault Formula

For males GFR =

$$\frac{(140\text{-age}) \times \text{body weight (kg)}}{\text{Sr. cr.} \times 72}$$

For female GFR =

$$\frac{0.85 \times (140\text{-age}) \times \text{body weight(kg)}}{\text{S.cr.} \times 72}$$

5. CD4 count

This was necessary for the distribution of cases among 3 groups for the purpose of study.

FACTS :

In brief present study observed the renal functions in HIV positive patients who, are free from any acute or chronic pre-existing kidney disease at different level of CD4 counts. I had to find out the relationship in level of CD4 count and renal function in patients of HIV.

The results were evaluated after seeing the above number of patients by collecting all the relevant datas and interpreting them. The correlation observed will e presented in "percentage form i.e. % comparing with available literature and studies".

CASE PROFORMA

Patient's Name - Sex - Reg. No.

Age - Wt.

Date of Admission

Presenting Complaint –

Past illness –

General Examination :

General Health

Pulse - B.P. - Cyanosis - Pallor – Oedema - Icterus - Lymphadenopathy

Systemic Examination :

Chest –

CVS –

Abdomen –

Investigations :

Elisa for HIV

CD4 count

Bl Urea

Sr. Creatinine

Bl Sugar

24-hr Urinary protein (Albumin) Micral Test.

RESULTS

HIV infection is a very common and rapidly spreading health problem prevailing in the society leading to morbidity and mortality. In patients infected with HIV, involvement of kidney further complicates the issue and further increases the morbidity and mortality. As already described a broad spectrum of renal diseases has been reported in patients of AIDS.

Hence this study was conducted to observe the derangement of renal function in HIV patients in relation with different level of CD4 count.

In this study, certain parameters were selected to study the renal function in HIV patients. They were†–

- Micral test
- S. creatinine 1.4 mg/dl.
- GFR 60 ml/min/1.73 m^2 BSA.
- Blood urea.

A total of 90 patients having HIV positive were enrolled. 3 Groups of 30 patients each were made on the basis of CD4 count, as†–

Group A - CD4 count > 350/µL

Group B - CD4 count 200-350/µL

Group C - CD4 count < 200/µL

The detailed history and investigations were recorded. The data were collected and analysed :

Incidence in male was found to be more in comparison to females in each of the groups. Mean age ± SD were almost comparable in both males/females in each of the group allocated. All within sexually active period of life. Average weight ±SD was minimum in Group C patients both male and females and it relatively increases with increasing CD4 counts. Out of total 90 cases, 30 from each group micral test was +ve in (28%), but it was more +ve (53.33%) in Group C having CD4 count <200/ m L (P=0.0018). Out of total 90 cases, 30 from each group S. creatinine level 1.4 mg/dl was found in (42%). But it was found in (60%) of patients from Group C (P=0.0354). After studying GFR by Cockcroft Goult Formula GFR level 60 ml/min/1.73 m^2 BSA was found in 47 (52%) of patients. It was found in (80.33%) of patients from group C (P=0.0006).

CONCLUSION

Involvement of Renal system is now established and common complication of HIV infection or full blown AIDS. All the patients infected with HIV at the time of evaluation, even in the absence of clinical symptoms, should undergo diagnostic tests for CD4 count, micral test/24 hr urinary protein, Serum creatinine level, blood urea level and by taking age (yr) and weight (kg) GFR should be calculated by Cock Croft formula.

If micral test, serum creatinine, GFR or blood urea are found to be abnormal in the absence of any pre-existing renal disease, patients should be further tested with USG (Abdomen) and those having microalbuminuria with renal biopsy, to establish the diagnosis of different renal syndromes in HIV.

Because the incidence of renal syndrome is high in patients having lower CD4 count early diagnosis and treatment destined to aim the renal disease as well as institution of HAART improves the prognosis.

Therefore early recognition and prompt treatment of renal disease in HIV patients reduce the morbidity and mortality of patients specially having low CD4 count level.

This cross-sectional study was conducted on small group of patients (90) to know about the details of relationship in level of CD4 count and renal function in HIV positive patients. It requires larger study and follow up of the same patient on HAART vis a vis improvement/deterioration of renal function.

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Hence in each group A, B, and C subgroup.

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Relevant history, thorough physical check-up and biochemical tests are done to rule out any pre-existing acute or chronic renal problem in the selected patients.

Exclusion Criteria : The study excluded the patients having DM, HTN, pregnancy or previous known renal disease that causes acute or chronic renal failure, as to avoid the possibility of obscuring the findings as well as results of the study.

Parameters of Study : Parameters which are studied to make assumptions of renal functions in HIV patients are –

1. Blood urea level
2. Serum creatinine level
3. 24 hr urinary protein/micral test
4. Glomerular filteration rate (GFR)

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Research Article

Prevalence and pattern of Hypothyroidism in patients attending the Outpatient department of a tertiary care teaching hospital.

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Abstract

Introduction: Among the endocrine disorders, Hypothyroidism is now believed to be the worldwide problem next to Diabetes.

Aims: This study was carried out to ascertain the prevalence and pattern of Hypothyroidism.

Methods: This prospective, epidemiological study was conducted in patients aged ≥20 years suspicion of thyroid disorders at a tertiary care teaching hospital from Jan 2012 to Dec 2013 in Bhuj, Gujrat. Thyroid abnormalities were diagnosed on the basis of laboratory results (serum FT3, FT4 and Thyroid Stimulating Hormone [TSH]).

Results: Out of 425 patients enrolled in our study, only 345 patients completed left. Out of the 345 analysable subjects, Female [n=217 (62.89%)] predominance is seen. Around n=72 cases were confirmed having thyroid abnormalities, Out of n=72 cases, only n=32 (9.27%) participants were found to have Hypothyroidism, Subclinical hypothyroidism was detected in n=28(8.11%) cases. Only n=12 (3.47%) cases had signs and symptoms of Clinical hyperthyroidism.

Conclusion: The prevalence of hypothyroidism was high, affecting approximately 9.27% study population. Hypothyroidism is found to have close association with Female gender and older age. Iodine intake ceases to be the sole etiological contender for thyroid disorders in urban areas.

Key Words

Hypothyroidism, free T3, free T4, prevalence, subclinical hypothyroidism, Clinical Hyperthyroidism

Introduction

Most of the patients nowadays are suffering from Thyroid disorders worldwide next to Diabetes Mellitus. Similarly In India too, there is a significant burden of thyroid diseases. To confirm the statement, a recent report shows that 300 million people in the world are suffering from thyroid disorders and among them about 42 million people resides in India. [1] The prevalence of hypothyroidism in the developed world is about 4-5%. [2,3] Hypothyroidism is one of the most common thyroid abnormality characterized by a broad clinical spectrum ranging from an overt state of myxoedema, end-organ effects and multisystem failure to an asymptomatic or subclinical condition with normal levels of thyroxine and triiodothyronine and mildly elevated levels of serum thyrotropin. [4-7] To add more, low-functioning thyroid gland or hypothyroidism is predominant in females leading to a significant percentage of infertility, recurrent miscarriages, irregular menstrual cycles, unexplained weight loss or gain.

In India, previously hypothyroidism was usually categorized under iodine deficient disorders (IDDs), [8, 9] but ever since India adopted the universal salt iodization program in 1983, [10] India is supposedly undergoing a transition from iodine deficiency to sufficiency state. Previous studies depicted the prevalence and pattern of thyroid disorders depends on sex, age, ethnic and geographical factors and especially on iodine intake. [11]

Therefore, this epidemiological study was carried out to estimate the Prevalence and pattern of Hypothyroidism in patients attending the Outpatient department of a tertiary care teaching hospital and to confirm that Iodine deficiency solely is no more the reason for higher prevalence of hypothyroidism in other parts of India as shown by previous studies.

Method

A cross-sectional, prospective, epidemiological study was

conducted to study the prevalence of Hypothyroidism in patients aged ≥20 years suspicion of thyroid disorders at a tertiary care teaching hospital from Jan 2012 to Dec 2013 in Bhuj, Gujrat. Thyroid abnormalities were diagnosed on the basis of laboratory results (serum FT3, FT4 and Thyroid Stimulating Hormone [TSH]). The serum sample of all the individual with suspicion of thyroid dysfunction were subjected to thyroid profile (Total T4, Total T3, Free T4, Free T3 and TSH). Patients with history of hypothyroidism and receiving levothyroxine therapy or those with serum free T4 <0.89 ng/dl and TSH >5.50 µU/ml, were categorized as hypothyroid. Initially n=425 OPD patients were enrolled in our study but out of 425, only n=345 patients were finally included on the basis of inclusion and exclusion criteria and referred to Biochemistry department during the period January 2012 to December 2013 for the assessment of thyroid hormone profile.

Primary outcome measure of the study was the prevalence of hypothyroidism assessed by measurement of thyroid hormones. Secondary outcome measures were the prevalence of: i) Sub-clinical hypothyroidism, ii) Hyperthyroidism. Written informed consent was taken prior to the study. The study was commenced after being approved by an Institutional Ethical Committee.

A central certified hospital laboratory performed the haematological and biochemical investigations. Assays for thyroid hormone (FT3, FT4 and TSH) were performed. Analytical sensitivity of the kit used to measure TSH, FT3, FT4 was 0.010 µIU/mL, 0.1 ng/mL, and 0.3 µg/dL, respectively.

Based on previous thyroid history and current thyroid function test results, participants were classified using following definitions:

Hypothyroid: Serum-free thyroxine (FT4) <0.89 ng/dL and thyroid stimulation hormone (TSH) >5.50 µU/mL,

Hyperthyroid: Serum FT4 >1.76 ng/dL and TSH <0.35 µIU/mL,

Subclinical hypothyroidism: Normal serum FT4 and TSH >5.50 µIU/mL.

Results

Out of 425 suspected thyroid disorder patients enrolled in our study, only 345 fulfilled our inclusion and exclusion criteria and were enrolled. Out of the 345 analysable subjects, n=217 (62.89%) were females and n=128 (37.10%) were males (Table 1).

Among 345 cases, n=72 cases were confirmed having thyroid abnormalities on the basis of Laboratory tests. Out of n=72 cases, only n=32 (9.27%) participants were found to have Hypothyroidism (elevated level of TSH and low levels of total T3 and total T4 in blood serum) (Table 2).

Table 2: Patients developing thyroid abnormalities at follow up

Thyroid disorders	No. of Patients
Hypothyroid	32(9.27%)
Subclinical hypothyroidism	28(8.11%)
Hyperthyroid	12(3.47%)

Among those clinical hypothyroid patients n=10 (31.25%) were males and n=22 (68.75%) were females. Subclinical hypothyroidism was detected in n=28(8.11%) cases who had no significant clinical signs and symptoms of hypothyroidism but the serum TSH level was elevated but exhibited normal range of T3 and T4 levels. Among 28 cases of subclinical hypothyroidism n=9 (32.14%) were male and n=19 (67.85%) were females. N=12 (3.47%) cases had signs and symptoms of hyperthyroidism and the laboratory findings of thyroid profile showed significant elevation of total T3, total T4 and free T4 levels in blood serum and low levels of TSH. Out of 12(3.47%) hyperthyroid cases n=3 (25.0%) were male and n=9 (75. 0%) were females.

Discussion

This prospective study was carried out to know the prevalence of thyroid disorders in a tertiary care teaching hospital. According to WHO, India has been classified having optimal iodine nutrition and hence iodine deficiency does not seem to play important role for thyroid disorders presently for India. [12,13] Therefore this study was conducted in adults residing in urban city with the aim to ascertain whether Iodine is the only cause of Hypothyroidism or there are other causes also.

In our study, most of the patients suffering from thyroid disorders were female (n=50, 69.44%) than male (n=22, 30.55%) (Table 1). This predominance of thyroid dysfunction in women is consistent with worldwide reports, especially those in midlife (46-54 years). [14] Also frequency of patients were found to be more in 41-60 age group (n=40, 55.55%) followed by 20-40 age group (n=24, 33.33%) and least in 61-80 age group (n=8, 11.11%). These findings are parallel to the studies conducted by Jatwa J and Ismail B

Table. 1 Demographic details of patients enrolled (n=345).

Age Group	Males n=128 (37.10%)		Females n=217 (62.89%)		Total n=345	
	Euthyroid (n=106)	Abnormal (n=22)	Euthyroid (n=167)	Abnormal (n=50)	Euthyroid (n=273)	Abnormal (n=72)
20-40	38	8	49	16	87	24
41-60	56	12	92	28	148	40
61-80	12	2	26	6	38	8

2012. [15] Hypothyroidism was found to be a common form of thyroid dysfunction affecting 10.9% of the study

population. In our study also, Hypothyroidism was found to be a common form of thyroid dysfunction [Clinical, n =32(9.27%) & Subclinical hypothyroidism, n= 28(8.11%)] and least thyroid disorder found was Hyperthyroidism [n=12(3.47%)] (Table 2) This is in accordance with the study conducted by Yadav NK et al., 2013 according to him, incidence of subclinical hypothyroidism was 7.9% and is much similar to our data. [16] Similar results were seen in study conducted by Unnikrishnan AG et al., 2013 who showed that Hypothyroidism was found to be a common form of thyroid dysfunction affecting 10.9% of the study population. [14] Abraham R et al., 2009 revealed that Hyperthyroidism had the lowest incidence among thyroid disorders and is comparable to our study. [17] Similar findings were revealed by Abbot India, a leading health care and pharmaceutical company in a Thyroid Epidemiological Study that Over 11% of the study population from Delhi reported hypothyroidism and one-third of them were not even aware of their disease. [18]

Thus the present study reflects the high prevalence of thyroid disorders in our hospital. This type of studies should be carried out in other parts of India as well to know the more precise incidence of Thyroid disorders in India. Since iodine deficiency is not now the only factor responsible for this issue, further studies need to be carried out to know the exact aetiology of Hypothyroidism.

Since the study was conducted in urban city and the patients enrolled were having good demographic characteristics, subjects were presumed iodine sufficient, without testing for reliable markers such as iodine content in salt samples or urinary iodine excretion. Thus, with regard to the cause of hypothyroidism, there may be etiological factors other than the iodization status.

Conclusion

Hypothyroidism is a commonly prevailing disorder in adult Indian population with predominance of female. Furthermore, female with advance age are more prone to thyroid abnormalities. Autoimmune mechanisms appear to play an etiological role in a significant proportion of patients. Iodine intake ceases to be the sole etiological contender for thyroid disorders in urban areas. Therefore physicians are required to conduct such type of studies more in different parts of our country to ascertain other causes of thyroid disorders. Identification of multiple risk factors and plausible underlying mechanisms is warranted.

What this study adds:

1. What is known about this subject?

Thyroid disorders are related to iodine deficiency.

2. What new information is offered in this study?

Besides, iodine deficiency there are other causes of Hypothyroidism.

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ETHICS COMMITTEE APPROVAL Approved

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Orthopaedics

Vitamin D Status in Post-Menopausal Female Including Post-Menopausal Osteoporosis and Prevalence of Hypovitaminosis-D

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Abstract **Original Research Article**

Context: Vitamin D status plays an important role in mineralisation of the skeleton at all ages. An alteration in vitamin D status and/or a reduced synthesis of 1, 25-dihydroxy vitamin D predispose to secondary hyperparathyroidism, which enhances bone remodelling and causes cortical bone loss. Aims: The present study was designed to evaluate: the prevalence of hypovitaminosis D in post-menopausal females and Relationship between vitamin D status in postmenopausal females with osteoporosis and without osteoporosis. Materials and methods: One hundred and forty-six post-menopausal women between 45 to 75 years attending the hospital OPD were studied. To be eligible for the study they had to have been post-menopausal for at least one year. The diagnosis of osteoporosis was made based on T-scores (BMD) at the lumbar spine (L1 to L4) and femoral neck by DEXA (GE Lunar Densitometer). Patients with chronic conditions affecting skeletal health and patients on drugs affecting the skeleton were excluded from the study. Serum 25(OH) vitamin D was estimated using LIAISON 25 OH Vitamin-D chemiluminescent immunoassay. Results: Out of 146 post-menopausal females, 100 subjects had vitamin D deficiency (≤ 20 ng/ml) and 29 subjects had vitamin D insufficiency (21 - 29 ng/ml). Thus, prevalence of hypovitaminosis D in post-menopausal females was 88.35%. Serum vitamin D was found to be significantly lower in post-menopausal women with osteoporosis as compared to post-menopausal women without osteoporosis ($p< 0.05$). On correlation analysis a positive correlation was noted between BMD and vitamin D, it was statistically significant. Conclusion: Serum vitamin D is a promising marker of bone turnover in post-menopausal women with osteoporosis, as it was found to be decreased in osteoporosis; therefore, it provides a dynamic measure of bone remodelling and it can be potentially useful in diagnosis and monitoring of response to therapy in patients of osteoporosis.

Keywords: Vitamin D, mineralisation, hypovitaminosis, osteoporosis.

INTRODUCTION

A lack of estrogen in postmenopausal women prevents the absorption and utilization of calcium and is the single most important factor in the development of osteoporosis [1].

Osteoporosis has a tremendous impact on the lives of many postmenopausal women. Fractures are potentially devastating complications of osteoporosis. Also, the number of osteoporotic fractures is increasing as the population ages, and assessment of skeletal health is becoming an important component of a woman's routine care [2]. Worldwide the life-time risk for women to have osteoporotic fracture is 30-40% [3]. Occurrence of osteoporosis is 10 years earlier in Indian people than in the West. It currently affects approximately one in three women and one in five men over age 50[2]. Nutrition is a critical component in the pathogenesis, prevention, and treatment of osteoporosis [4]. Among nutrients, calcium and vitamin D play an important role in the mineralisation of the skeleton at all ages, an alteration in vitamin D status and/or a reduced synthesis of 1, 25-dihydroxy vitamin D predispose to secondary hyperparathyroidism, which enhances bone remodelling and causes cortical bone loss [5]. Although present in food, the major source of vitamin D is synthesized in skin after exposure to sunlight. Variety of factors influence cutaneous production of Vitamin D. such as aging, melanin content of skin ,geographical location, seasons, and level of clothing and use of sunscreens[6,7].

The present study was designed to evaluate: the prevalence of hypovitaminosis D in post-menopausal females, Relationship between vitamin D

status in postmenopausal females with osteoporosis and without osteoporosis.

MATERIALS AND METHODS

The present study was conducted at the Department of Medicine and Department of orthopaedics, Rohilkhand Medical College and Hospital, Rohilkhand University, Bareilly, UP. One hundred and forty-six post-menopausal women between 45 to 75 years attending the hospital OPD were studied. To be eligible for the study they had to have been postmenopausal for at least one year.

Out of 146 post-menopausal women 72 had osteoporosis (according to the above mentioned definition). These 72 females also included 16 patients presenting to the emergency/orthopaedic department with fragility fracture. A fragility fracture was defined as one that occurred as a result of minimal trauma, such as a fall from a standing height or less, or occurred without identifiable trauma. Rest of 74 post-menopausal women were without osteoporosis.

Exclusion criteria for the study were oestrogen replacement therapy within 1 year, deranged renal function (serum creatinine >1.5 mg %) or renal calculi, abnormal thyroid function, significant liver disease, history of cancer, peptic ulcer, or oesophageal disease requiring prescription. Regular therapy with phosphate binding antacid, therapy with any other drug that affects the skeleton, e.g. steroids, anti-resorptive therapy, anticonvulsant, anticoagulants, etc. Informed consent was obtained from all the subjects participating in the study; and the study was approved by the local ethical committee. A detailed history and physical examination was carried out for every subject who entered in the study as per a pre-designed Performa. Examination comprised of a thorough physical examination, assessment of vital parameters, anthropometry and systemic examination. Bone mineral density (BMD) was measured using DEXA by GE Lunar Densitometer. Serum 25 (OH) vitamin D was estimated using LIAISON 25 OH vitamin D chemiluminescent immunoassay. vitamin D sufficiency was defined as serum 25 (OH) vitamin D in the range of 30 to 100 ng/ml, vitamin D insufficiency as values between 21 - 29 ng/ml, and values $\leq$ 20 ng/ml were defined as vitamin D deficiency. Statistical analysis was performed using SPSS version 16 statistical package for windows (SPSS, Chicago, IL).

OBSERVATIONS

The baseline characteristics of 146 post-menopausal women are shown in Table I. The mean age of post-menopausal females without osteoporosis was 51 ± 3.88 years and post-menopausal with osteoporosis was 56.57 ± 8.15 yrs. Similarly, mean duration of menopause in post-menopausal females without osteoporosis group was 3.85 ± 1.82 and in postmenopausal females with osteoporosis group was 9.56 ± 5.80 suggesting significant difference in the two groups. There was a significant difference in the BMD lumbar spine in the two groups ($p < 0.05$). BMD lumbar spine was 1.20 ± 0.18 in the post-menopausal women without osteoporosis group as compared to 0.81 ± 0.13 in the osteoporosis group. Similarly there was significant difference in the BMD hip in the two groups ($p < 0.05$).

Mean T- score at lumbar spine was 1.41 ± 0.29 and -3.14 ± 1.0 in post-menopausal women without osteoporosis and with osteoporosis respectively, suggesting a significant difference in the two groups ($p < 0.05$). Similarly, there was significant difference in the mean T-score at hip ($p < 0.05$).

Out of 146 post-menopausal females, 100 subjects had vitamin D deficiency ($\leq$ 20 ng/ml) and 29 subjects had vitamin D insufficiency (21-29 ng/ml). Thus, prevalence of hypovitaminosis D in post-menopausal females was (88.35%). Out of seventy--two post-menopausal female with osteoporosis, fifty-five (77.33%) had vitamin D deficiency ($\leq$ 20 ng/ml), eleven (16.0%) had vitamin D insufficiency (21-30 ng/ml), and only six females (5.40%) had vitamin D in the normal range (30 - 100 ng/ml). Out of seventy-four post-menopausal females without osteoporosis group, forty-five (58.90%) had vitamin D deficiency ($\leq$ 20 ng/ml), eighteen (24.65%) had vitamin D insufficiency (21 - 30 ng/ml) and eleven females (15.27%) had vitamin D in the normal range (30-100 ng/ml). So, prevalence of vitamin D deficiency and insufficiency was 68.49% and 19.86% respectively in post-menopausal females.

Serum vitamin D was found to be significantly lower in post-menopausal women with osteoporosis as compared to post-menopausal women without osteoporosis ($p < 0.05$), mean serum vitamin D was 15.22 ± 6.23 ng/ml in osteoporosis group as compared to 19.51 ±8.58 ng/ml in the other group. Whole blood ionised calcium was 1.16 ±0.10 mmol/l and 1.07 ± 0.09 mmol/l in post-menopausal women without osteoporosis and with osteoporosis respectively, suggesting a significant difference between the two groups. On correlation analysis a positive correlation was noted between BMD and vitamin D, it was statistically significant ($r 2 = 0.201$, $p < 0.05$).

On comparing impact of duration of menopause on BMD and serum vitamin D levels, patients with < 10 years of menopause had mean BMD at spine 0.86 ± 0.13 g/cm^2, mean BMD at hip 0.86 ± 0.15 g/cm^2 and mean serum vitamin D 15.42 ± 6.30 ng/ml, while patients with > 10 yrs of menopause had mean BMD at spine 0.75 ± 0.11 g/ cm^2, mean BMD at hip 0.75 ± 0.13 g/cm^{2}and mean serum vitamin D 15.02 ±6.16 ng/ml. On applying paired t-test there was significant difference in the two groups in terms of

mean BMD, as well as mean serum vitamin D levels (Table II).

Table-I: Showing baseline characteristics of study subjects

S. No.	Parameter	Post-menopausal without osteoporosis		Post-menopausal with osteoporosis		P value
		N = 74			N = 72	
		Mean	S.D	Mean	S.D	
1	Age (years)	51	3.88	56.57	8.15	< 0.05
2	Time since menopause (years)	3.85	1.82	9.56	5.80	< 0.05
3	BMI (kg/m²)	26.61	4.12	25.73	5.72	NS
4	BMD - lumbar spine (g/cm²)	1.20	0.18	0.81	0.13	< 0.05
5	T. score - lumbar spine	1.41	0.29	-3.14	1.10	< 0.05
6	BMD - hip (g/cm²)	1.22	0.15	0.81	0.15	< 0.05
7	T. score – Hip	1.34	0.26	-1.87	1.03	< 0.05
8	Whole blood ionised calcium (mmol/l)	1.16	0.10	1.07	0.09	< 0.05
9	Serum 25 (OH) vit D (ng/ml)	19.51	8.58	15.22	6.23	< 0.05
	Deficiency (≤ 20 ng/ml)	13.64	3.87	12.49	3.12	<0.06
	Insufficiency (21 - 29 ng/ml)	23.56	2.77	21.78	1.71	< 0.05
	Normal (30 - 100 ng/ml)	34.49	2.63	31.34	1.66	< 0.05
10	S. TSH (mIU/l)	2.90	1.23	2.65	2.05	NS
11	S. creatinine (mg/dl)	1.14	0.19	0.95	0.16	< 0.05

Table-II: Impact of duration of menopause on BMD and Serum 25 (OH) vit D (ng/ml)

S. No.	Parameter	Patients with < 10 years of menopause			Patients with ≥ 10 years of menopause			P value
			N = 38			N = 35		
		Mean		S.D	Mean		S.D	
1	BMD (spine) (g/cm²)	0.86		0.13	0.75		0.11	< 0.05
2	T. score (spine)	-2.73		1.08	-3.59		0.96	< 0.05
3	BMD (hip) (g/cm²)	0.86		0.15	0.75		0.13	< 0.05
4	T. score hip	-1.52		0.85	-2.25		1.09	< 0.05
5	Serum 25 (OH) vit D (ng/ml)	15.42		6.30	15.02		6.16	< 0.05

DISCUSSION

The present study was carried out with the aims to determine the prevalence of hypovitaminosis D in postmenopausal women, significance of serum vitamin D in evaluation of osteoporosis, Relationship between vitamin D status in postmenopausal females with osteoporosis and without osteoporosis.

Normal bone metabolism depends on the presence of appropriate repletion of vitamin D. Although only few patients with osteoporosis exhibit obvious biochemical signs of hypovitaminosis D, vitamin D insufficiency has been shown to have adverse effects on calcium metabolism, osteoblastic activity, matrix ossification, bone mineral density (BMD), and bone remodelling[8]. Low serum 25 (OH) vitamin D concentration is associated with secondary hyperparathyroidism, increased bone turnover, reduced BMD, and increased risk of osteoporotic fractures [9]. In our study, we found that majority of the subjects had hypovitaminosis D. Out of seventy--two post-menopausal female with osteoporosis, fifty-five (77.33%) had vitamin D deficiency (≤ 20 ng/ml), eleven (16.0%) had vitamin D insufficiency (21 - 30 ng/ml), and only six females (5.40%) had vitamin D in the normal range (30 - 100 ng/ml). Out of seventy-four post-menopausal females without osteoporosis group, forty-five (58.90%) had vitamin D deficiency (≤ 20 ng/ml), eighteen (24.65%) had vitamin D insufficiency (21 - 30 ng/ml) and eleven females (15.27%) had vitamin D in the normal range (30 - 100 ng/ml). So, prevalence of vitamin D deficiency and insufficiency was 68.14% and 20.21% respectively in post-menopausal females.

So, prevalence of vitamin D deficiency and insufficiency was 68.49% and 19.86% respectively in post-menopausal females. The overall prevalence of hypovitaminosis D was 88.35%. Prevalence of hypovitaminosis D in post-menopausal women was found to be 47% in Thailand, 49% in Malaysia, 90% in Japan, and 92% in South Korea [10]. Harinarayan *et al.* reported vitamin D deficiency in 70% females and insufficiency in 23% females in their study from South India in 2011[11]. Goswami *et al* found hypovitaminosis D was present in up to 90 per cent of apparently healthy subjects in Delhi [12]. Skin complexion, poor sun exposure, vegetarian food habits, low milk intake, high phytates in food, and lack of vitamin D food fortification programme explain the

high prevalence of vitamin D deficiency in India despite its sunny climate.

In the present study, Serum vitamin D was found to be significantly lower in post-menopausal women with osteoporosis as compared to post-menopausal women without osteoporosis ($p < 0.05$), mean serum vitamin D was 15.22 ± 6.23 ng/ml in osteoporosis group as compared to 19.51 ±8.58 ng/ml in the other group. Our study showed a positive correlation between vitamin D and BMD, and the relation was statistically significant ($r2 = 0.201$, $p < 0.05$). Thus, in osteoporosis, low level of vitamin D is seen. Kuchuk *et al.* studied on 7,441 post-menopausal

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Original Research Article

Osteocalcin, a promising marker of osteoporosis: evaluation in post-menopausal females with osteoporosis

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ABSTRACT

Background: Osteocalcin, has high affinity for calcium. In osteoporotic women, deficiency of calcium may lead to lowering of the formation of hydroxyapatite crystals. Thus, in the state of hypo mineralization, free osteocalcin available in the circulation. Therefore, present study was designed to evaluate significance of serum osteocalcin in diagnosis of osteoporosis, and relationship between Serum Osteocalcin and BMD (Bone mineral Density) in post-menopausal females with osteoporosis and without osteoporosis.
Methods: One hundred and forty seven post-menopausal women between age 45 to 80 years attending the hospital OPD were studied. To be eligible for the study they had to have been postmenopausal for at least one year. The diagnosis of osteoporosis was made based on T-Scores (BMD) at the lumber spine (L1 to L4 and femaral neck) by DEXA (GE lunar Densitometer). Serum osteocalcin level was estimated by LIAISON osteocalcin assay. Patients with chronic conditions affecting skeletal health and patients on drugs affecting the skeleton were excluded from the study.
Results: Serum osteocalcin level in post-menopausal female without osteoporosis was 9.87±1.04ng/ml, while post-menopausal female with osteoporosis had 22.62±2.25ng/ml suggesting significant increase in bone marker level in osteoporotic females ($p<0.05$.) Correlation study between BMD and osteocalcin showed strong Negative Correlation ($r=-0.77$, $p<0.05$).
Conclusions: Serum osteocalcin can be considered as a specific marker of osteoblast function as its levels have been shown to correlate with bone formation rates. Thus, serum osteocalcin can be used for diagnosis and monitoring of response to therapy and this may be the better predictor than BMD.

Keywords: Bone mineral density, Mineralization, Osteocalcin, Postmenopausal osteoporosis

INTRODUCTION

Osteoporosis is a progressive systemic skeletal disorder characterized by consequent increase in bone fragility and susceptibility to fracture, according to the world Health Organization worldwide the lifetime risk for women to have osteoporotic Fracture is 30-40%.[1,2] Occurrence of osteoporosis is 10 years earlier in Indian women and one in five men over age 50. Because of related morbidity, disability, diminished Quality of life and mortality, osteoporosis and fractures associated with it are Major public health concern.[3] Osteoporotic patients are characterized by significant Lower body weight higher level of bone turnover marker.[4] The levels of osteocalcin, Bone alkaline Phosphatase, cross linked telopeptide-C parathyroid hormones and 1, 25 dihydroxy vitamin D have been shown to be significantly higher in osteoporotic females Bone Turnover markers can prove beneficial when bone mass density (BMD) changed are too small to be utilized clinically particularly within the first 6 months.[5]After antiresorptive therapy initiation.[6] In women receiving antiresorptive therapy, Short term

changes in bone turnover markers are related to long term changes in BMD and may also predict long term increases therein Serum Osteocalcin can be Considered as a specific marker of osteoblast Function as its levels have been shown to correlate with bone formation rates the change in serum osteocalcin have been shown to correlate with changes in BMD.[7-9] The Present study was designed to evaluate significance of serum osteocalcin in evaluation of osteoporosis, relationship between Serum Osteocalcin and BMD (Bone mineral Density) in post-menopausal females with osteoporosis.

METHODS

The present study was conducted at the Department of medicine and Department of Orthopaedics Katihar Medical College, Katihar, Bihar from April 2018 to April 2019, one hundred and forty seven post-menopausal women between age 45 to 80 years attending the hospital OPD were studied. Informed consent was obtained from all the subjects participating in the study and study was approved by the Institutional ethics committee.

Inclusion criteria

To be eligible for the study subjects had to have been postmenopausal for at least one year and in good health with no vertebral abnormalities in the L1-L4 region and the decreased bone mineral density (lumber spine on right or left femoral meck or both T-Scare-1.0 or less). Out of one hundred forty seven post-menopausal women seventy three subject aged between 45 to 80 years had osteoporosis (T-Scare - 2.5 or less). Rest 74 postmenopausal women were without osteoporosis. The diagnosis of osteoporosis was made based on T-Scores (BMD) at the lumber spine (L1 to L4 and femaral neck) by DEXA (GE lunar Densitometer).

Exclusion criteria

Subjects who had a history of HRT intake within 1 year, Hypothyroidism, Hyperthyroidism, Renal failure, liver disease. H/O Cancer, Peptic ulcer or esophageal Disease requiring prescription, Regular therapy with phosphate binding antacid therapy, any other drug that affects the skeleton e.g. steroids, antiresorptive therapy, anticonvulsant, anticoagulants, etc.

A detailed history and physical examination was carried out for every subject who entered in the study as per a pre-designed performa. Examination comprised of a through physical examination assessment of vital parameters, anthropometry and systemic examination. Serum osteocalcin level was estimated by LIAISON osteocalcin assay. Statistical analysis was performed using SPSS version 16, statistical package for windows (SPSS, chicago, IL)

RESULTS

The baseline characteristics of 147 Postmenopausal Women are shown in Table-1. The mean age in post-menopausal female without osteoporosis was 51±3.88 years and post-menopausal with osteoporosis was 56.57±8.15 years. Similarly, mean duration of menopause in post-menopausal female without osteoporosis group was 3.85±1.8 and with osteoporosis was 9.56±5.8 suggesting significant difference between the two group. In both the group, the patients were above 45 years. The mean body mass index (BMI) was 26.61±4.12kg/m^2 and 25.73±5.72 kg/m^2 respectively in post-menopausal female without osteoporosis and post-menopausal female with osteoporosis groups.

Table 1: Baseline characteristics of study subject.

S.no.	Parameter	Postmenopausal without osteoporosis (n=74)		Postmenopausal with osteoporosis (n=73)		p value
		Mean	SD	Mean	SD	
1.	Age (years)	51	3.88	56.57	8.15	<0.05
2.	Time since menopause (years)	3.85	1.82	9.56	5.80	<0.05
3.	BMI (kg/$\mathbf{m^2}$)	26.61	4.12	25.73	5.72	NS
4.	BMD-lumbar spine (g/$\mathbf{cm^2}$)	1.20	0.18	0.81	0.13	<0.05
5.	T-score lumbar spine	1.41	0.29	-3.14	1.10	<0.05
6.	BMD hip (g/$\mathbf{cm^2}$)	1.22	0.15	0.81	0.15	<0.05
7.	T-score hip	1.34	0.26	-1.87	1.03	<0.05
8.	Ionised calcium (MIu/l)	1.16	0.10	1.07	0.09	<0.05
9.	S. Tsh (MIu/L)	2.90	1.23	2.65	2.05	NS
10.	S. Creatinine (mg/dl)	1.14	0.19	0.95	0.16	<0.05
11.	Serum osteocalcin (ng/ml)	9.87	1.04	22.62	2.25	<0.05

The mean BMD at Lumbar spine was 1.20±0.18g/cm^{2}and 0.81±0.13g/cm^2 respectively and means BMD at hip was 1.22±0.15g/cm^2 and 0.8±0.15g/cm^2 respectively in post-menopausal female

without osteoporosis and post-menopausal female with osteoporosis groups while mean T-score at lumbar spine was 1.41±0.29 and -3.14±1.0 respectively and T-score at hip was 1.34±0.26 and -1.87±1.03 respectively in post-menopausal female without osteoporosis and post-menopausal female with osteoporosis group which was suggestive of significant difference in both groups.

Serum osteocalcin was 9.87±1.04ng/ml and 22.62±2.25ng/ml respectively in both groups. Serum osteocalcin level was found to be significantly higher in post-menopausal women with osteoporosis as compared to post-menopausal women without osteoporosis ($p<0.05$).

On comparing impact of duration of menopause in BMD and serum osteocalcin level at baseline patients with less than 10 years of menopause had mean BMD at spine 0.86±0.13g/cm^2, Mean BMD at Hip 0.86±0.159/cm^2 and Mean Serum osteocalcin 22.35±2.10ng/mi while Patients with >10yrs of Menopause had Mean BMD at spine 0.75±0.119/cm^2, Mean BMD at hip 0.75±0.139/cm^2 and Mean Serum osteocalcin 22.92±2.41ng/ml on applying paired t-test showed significant difference in two groups in terms of mean BMD but there was not much difference in two groups in terms of mean serum osteocalcin as shown in Table 2.

Table 2: Impact of duration of menopause in BMD and Serum Osteocalcin.

S no	Parameters	Patients with <10yrs of menopause n=38		Patients with >10yrs of menopause (n=35)		p value
		Mean	SD	Mean	SD	
1	BMD spine (9/**cm^2**)	0.86	0.13	0.75	0.11	<0.05
2	T-score spine	-2.73	1.08	-3.59	0.96	<0.05
3	BMD hip (9/**cm^2**)	0.86	0.15	0.75	0.13	<0.05
4	T-score hip	-1.52	0.85	-2.25	1.09	<0.05
5	Serum osteocalcin (ng/ml)	22.35	2.12	22.92	2.41	<0.27

DISCUSSION

The Present study was carried out with the aims to determine the significance of Serum Osteocalcin in diagnosis of osteoporosis and relationship between S. osteocalcin and bone mineral density in post-menopausal females with osteoporosis and without osteoporosis women. Osteocalcin has high affinity for calcium in osteoporotic women, deficiency of calcium may lead to lowering of the formation of Hydroxyapatite crystals. Thus in the state of Hypo mineralization free osteocalcin available in the circulation this may explain the increased concentration of serum osteocalcin in osteoporotic female. Osteocalcin is a promising marker of bone turnover useful in the diagnosis and follow up of high turnover osteoporosis.[10-13] Serum osteocalcin has also been reported as predictive of the rate of bone loss after menopause and as a tool to selecting appropriate therapy.[14,15] In the present study serum osteocalcin level in post-menopausal female without osteoporosis was 9.87±1.04ng/ml, while post-menopausal female with osteoporosis had 22.62±2.25ng/ml suggesting significant increase in bone marker level in osteoporotic females ($p<0.05$.) Correlation study between BMD and osteocalcin showed strong Negative Correlation ($r=-0.77$, $p<0.05$) A case control study of 90 Postmenopausal women showed results that were consistent with the results of the present study conducted by Pirro Metal, 2010.[16] Hari Kumar KV et al, 2008 studied on 82 post-menopausal osteoporotic female in Hyderabad and found similar results.[17]

CONCLUSION

Thus serum osteocalcin can be used for diagnosis and monitoring of response to therapy and this may be the better predictor than BMD.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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ORIGINAL RESEARCH

Comparative evaluation of complications and mortality in ST-segment elevation acute myocardial infarction (STEMI) in diabetic and non-diabetic patients

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ABSTRACT

Background: Diabetes is a universal problem and is becoming a major concern at old age especially in obese people and in people with sedentary life style. The present study was conducted to assess complications and mortality in ST-segment elevation acute myocardial infarction in diabetic and non-diabetic patients.

Materials & Methods:160 consecutive patients of diabetic (group I) and non-diabetic (group II) having acute ST-segment elevation myocardial infarction (STEMI) of both genders were included. Treatment was given to all patients.

Results: Age group <44 years had 15 diabetic and 10 non- diabetic, 45-54 years had 25 diabetic and 12 non- diabetic, 55-64 years had 10 diabetic and 20 non- diabetic and >65 years had 40 diabetic and 28 non- diabetics. The site was anterior in 26 and 20, inferior in 38 and 40, inferior+ right ventricular in 20 and 10 and lateral in 6. Mortality was seen in those in which streptokinase was given in 7 and 3 and streptokinase not given in 15 and 10 in group I and group II respectively (P< 0.05).

Conclusion: With streptokinase administration there was reduction in mortality in diabetic as well as in non- diabetic. However, diabetics not on streptokinase had higher mortality as compared to non- diabetics.

Key words: Diabetes, STEMI, streptokinase

INTRODUCTION

Acute myocardial infarction (AMI) is one of the leading causes of all acute emergencies and is becoming an important public health problem in the developing countries. Diabetes is a universal problem and is becoming a major concern at old age especially in obese people and in people with sedentary life style.[1]

Worldwide the number of people diagnosed with diabetes mellitus is increasing rapidly. Diabetes is associated with a two- to four-fold increase in the risk of developing CHD, and cardiovascular diseases are the major cause of death among diabetic persons.[2] It remains controversial whether diabetic persons have a similar risk of developing acute CHD events as non-diabetic patients who have suffered a prior myocardial infarction (MI).[3] Diabetes mellitus is a majorrisk factor for cardiovascular disease in general and for coronary heart disease in particular. Furthermore, the recent National Cholesterol Education Programme III guidelines have elevated diabetes to a coronary disease risk equivalent. Among patients with diabetes who survived myocardial infarction (MI), less is known about subsequent morbidity and mortality.[4]

In patients with AMI, heart failure is characterized by diastolic dysfunction alone or systolic and diastolic dysfunctions together. About 3% of the adult patients develop systolic dysfunction which recognized by echocardiography and is asymptomatic in about of them. Re-infarction is diagnosed by persistent and typical severe chest pain along with re-elevation

of ST-segment and increased concentrations of cardiac markers in the blood.[5] The present study was conducted to assess complications and mortality in ST-segment elevation acute myocardial infarction in diabetic and non-diabetic patients.

MATERIALS & METHODS

The present study comprised of 160 consecutive patients of diabetic (group I) and non-diabetic (group II) having acute ST-segment elevation myocardial infarction (STEMI) of both genders.

Demographic data was recorded. A 12-lead ECG of each patient was recorded. The patients were divided into four groups on the basis of ST-segment elevation in different leads. ST-segment elevation in leads V1-V6 (anterior AMI), in II, III, aVF (Inferior AMI), in II, III, aVF+ V4R (Inferior + Right ventricular AMI) and in I, aVL, V5, V6 (Lateral AMI). 5 ml blood sample was collected and analysed for serum CK and CK-MB and Trop-T level. Treatment was given to all patients. Results were compiled and assessed statistically. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Age group (Years)	Diabetic (90)	Non- diabetic (70)	P value
<44	15	10	0.01
45-54	25	12	
55-64	10	20	
>65	40	28	

Table I shows that age group <44 years had 15 diabetic and 10 non- diabetic, 45-54 years had 25 diabetic and 12 non- diabetic, 55-64 years had 10 diabetic and 20 non- diabetic and >65 years had 40 diabetic and 28 non- diabetics. The difference was non- significant (P< 0.05).

Table II Comparison of parameters

Parameters	Variables	Group I	Group II	P value
Site	Anterior	26	20	0.05
	Inferior	38	40	
	Inferior+ right ventricular	20	10	
	Lateral	6	0	
Mortality	Streptokinase given	7	3	0.01
	Streptokinase not given	15	10	

Table II, graph I shows that site was anterior in 26 and 20, inferior in 38 and 40, inferior+ right ventricular in 20 and 10 and lateral in 6. Mortality was seen in those in which streptokinase was given in 7 and 3 and streptokinase not given in 15 and 10 in group I and group II respectively (P< 0.05).

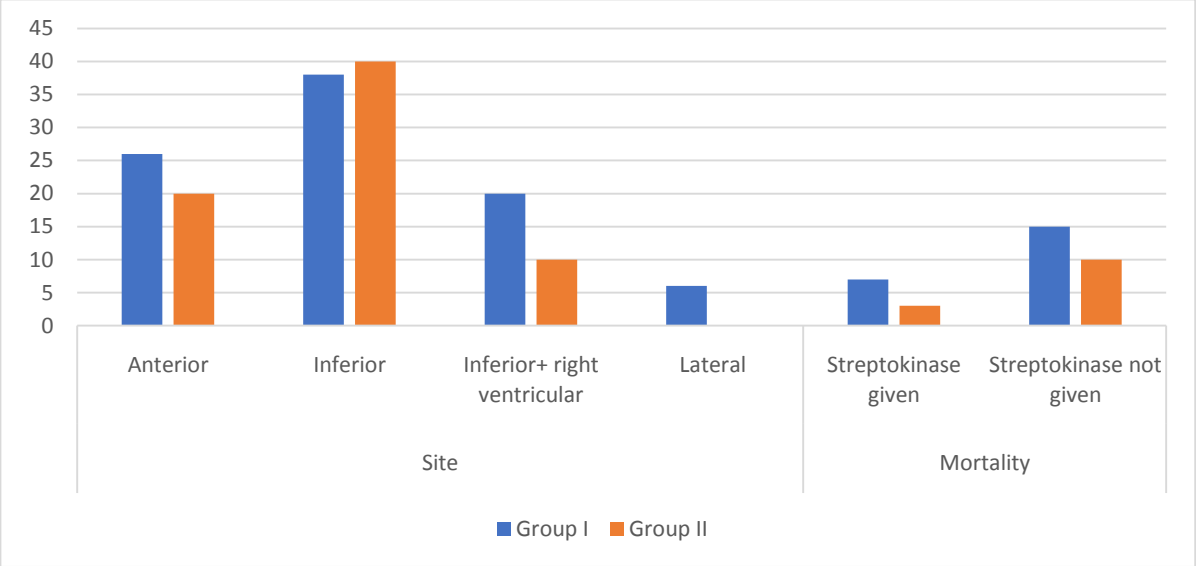
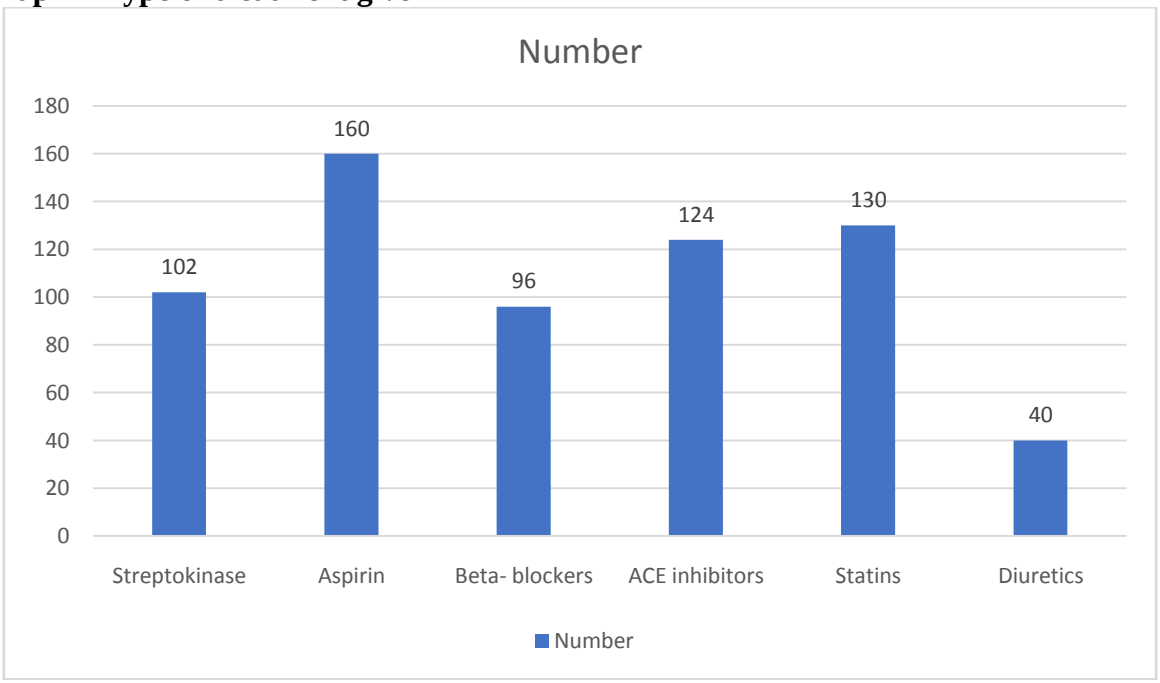


Table III Type of treatment given

Treatment given	Number	P value
Streptokinase	102	0.01
Aspirin	160	
Beta- blockers	96	
ACE inhibitors	124	
Statins	130	
Diuretics	40	

Table III, graph II shows that type of treatment given was streptokinase in 102, aspirin in 160, beta- blockers in 96, ACE inhibitors in 124, statins in 130 and diuretics in 40 patients. The difference was significant (P< 0.05).

Graph IIType of treatment given



DISCUSSION

Diabetes is associated with a marked increase (by a factor of two to four) in the risk ofcoronary heart disease. Clinically established coronary heart disease itself is associated with an increase in mortality from coronary heart disease by a factor of three to seven, depending on the mode of presentation.[6]The plasma cholesterol level is a strong predictor of the risk of cardiovascular events both in patients with diabetes and in patients with coronary heart disease.[7]The high-risk status of these groups of patients and their need for more aggressive lipid-lowering therapy have been recognized by both the National Cholesterol Education Programmeand the American Diabetes Association.[8]The reduction in plasma lipids recommended by the National Cholesterol Education Programme is greater for patients with coronary heart disease than for patients with diabetes.[9] However, there were differing opinions among members of the National Cholesterol Education Programme panel, with some suggesting that diabetic patients should have the same intensity of cholesterol-lowering therapy as patients with coronary heart disease. Thus, there is controversy about how aggressively to treat cardiovascular risk factors in patients with diabetes. It has been suggested that such patients should be treated as if they had established coronary heart disease.[10] The present study was conducted to assess complications and mortality in ST-segment elevation acute myocardial infarction in diabetic and non-diabetic patients.

In present study, age group <44 years had 15 diabetic and 10 non- diabetic, 45-54 years had 25 diabetic and 12 non- diabetic, 55-64 years had 10 diabetic and 20 non- diabetic and >65 years had 40 diabetic and 28 non- diabetics. Iqbal et al[11] in their study complications of acute myocardial infarction (AMI) and the outcome were compared between diabetics and non-diabetic patients. Different complications studied varied significantly within diabetics, non-diabetics and in overall after controlling for diabetes. Complications showed similar pattern (heterogeneity test P >0.5) in diabetic and non-diabetic patients. The abnormalities including Cardiogenic shock, left ventricular failure (OR = 2.5), re-infarction (OR= 2.2), arrhythmia (OR= 2.04) and ventricular septal defect (OR= 2.17) were 4.2, 4.7, 21.3, 4.2 and 85.24 times higher in diabetics, respectively. However, occurrence of post myocardial angina was low in diabetics than non-diabetics. Odds of having diastolic dysfunction were 1.8 times higher in diabetic patients. The moderate and severe LV-dysfunction was 3.3 and 2.5 times higher diabetics, while mild LV-dysfunction in was 2.1 times higher in non-diabetics. Mortality due to STEMI in diabetics was 2.3 times higher than in non-diabetics. Mortality varied significantly between different age groups in non-diabetics and in overall after controlling for diabetes. In non-diabetic group, mortality was 8.4 times higher in patients those were not given streptokinase than those were given streptokinase, while in diabetic group it was 2.5 times higher in patients were not given streptokinase than those were given streptokinase. The results indicate that the diabetics have higher risk of mortality.

We found that site was anterior in 26 and 20, inferior in 38 and 40, inferior+ right ventricular in 20 and 10 and lateral in 6. Mortality was seen in those in which streptokinase was given in 7 and 3 and streptokinase not given in 15 and 10 in group I and group II respectively. We found that type of treatment given was streptokinase in 102, aspirin in 160, beta- blockers in 96, ACE inhibitors in 124, statins in 130 and diuretics in 40 patients. Haffner et al[12] compared the seven-year incidence of myocardial infarction (fatal and nonfatal) among 1373 nondiabetic subjects with the incidence among 1059 diabetic subjects. The seven-year incidence rates of myocardial infarction in nondiabetic subjects with and without prior myocardial infarction at base line were 18.8 percent and 3.5 percent, respectively (P<0.001). The seven-year incidence rates of myocardial infarction in diabetic subjects with and without prior myocardial infarction at base line were 45.0 percent and 20.2 percent, respectively (P<0.001). The hazard ratio for death from coronary heart disease for diabetic subjects without prior myocardial infarction as compared with nondiabetic subjects with prior

myocardial infarction was not significantly different from 1.0 (hazard ratio, 1.4; 95 percent confidence interval, 0.7 to 2.6) after adjustment for age and sex, suggesting similar risks of infarction in the two groups.

CONCLUSION

Authors found that with streptokinase administration there was reduction in mortality in diabetic as well as in non- diabetic. However, diabetics not on streptokinase had higher mortality as compared to non- diabetics.

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