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A Study on Incidence of Coronary Heart Disease in Different Age and Sex Groups of Inhabitants of Katihar and Its Surroundings

Dr. Sanjay Singh[1], Dr. Sanjay Tiwari[2], Prof. (Dr.) A. K. Ray[3]

INTRODUCTION

Coronary heart disease is the most common form of heart diseases and the most important cause of premature death in almost throughout the world [1].

There are several risk factors for the coronary heart disease, which includes genetic factor, obesity, diabetes mellitus, hypertension, hyperlipidemia, cigarette smoking etc. Coronary heart disease is one of the complications of diabetes mellitus. Untreated long continued diabetes mellitus produces three times more Macrovascular coronary artery disorder than in normal individuals [2].

Long continued hypertension its also one of the important risk factors for coronary heart disease which is perhaps produced by the endothelial damage and atherosclerosis on the damaged tissue.

AIMS AND OBJECTIVES

An attempt has been made to study the relationship of different age and sex on CHD.

MATERIALS AND METHODS

Hundred cases of CHD of different age groups were selected for study from ICCU and indoor medical wards of Katihar Medical College & Hospital with the permission of the competent authorities.

Only those cases of CHD were selected for study who don't show any associated cardiac lesion on valvular heart disease or cardio-myopathy etc.

The diagnosis of CHD was established with W.H.O criteria. If any of the two changes present was considered to be suffering from CHD.

1. Chest pain.
2. ECG-changes.
3. Serum enzyme rise.
4. Standard 12 lead ECG was done and changes in ST segment, 't' wave and presence of pathological 'q' wave were noted in different leads.
5. Serum enzyme, (LDH) estimation by king's method SGOT estimation by Reitmen& Franke method,

OBSERVATION

Table 01 : Incidence of Coronary Heart Disease in Different Age Groups

Age group in Years	Number of Cases	%
Below 30	01	01
31 - 40	08	08
41 - 50	35	35
51 - 60	30	30
61 - 70	23	23
Above 70	03	03
Total	**100**	**100**

Table 02 : Sex Incidence of Coronary Heart Disease

Sex	Number of Cases	%
Male	90	90
Female	10	10
Total	**100**	**100**

DISCUSSION

The incidence of coronary Heart Disease has been observed (Vide Table I) to be maximum (35%) in the Age group of 41 to 50 Years and only slightly less (30%) in the Age group of 51 to 60 Years. The overall incidence was 65% in the Patients of 41 to60 Years of Age, Only 01 Case was recorded before the Age of 30 Years in this series.

Peel in (1955) 3 found a peak incidence at 55 and 59 Years of Age and a steep decline thereafter. The present observation seems to differ from the observation of Peel. The difference could partly be explained by the fact that his study consisted of 865 cases of CHD which is quite a large study than the present series of 100 cases.

Among 100 cases of CHD 90% were males and 10% were females giving a male female ratio of 9:1. This suggests a clearly high incidence of CHD in males compared to females, The observation is close to one of Headly's (1939) 4 who has observed a male female ratio of 8:1 and in Subramanyan's5 series 91.23% were male and 8.77% were female.

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Comparative Study of the Various Risk Factors in 70 Patients of Coronary Heart Disease in different age groups at Katihar and Surrounding Areas in Bihar

Dr. Sanjay Singh[1], Dr. Sanjay Tiwari[2], Prof. (Dr.) A. K. Ray[3]

INTRODUCTION

Coronary heart disease is the most common form of heart diseases and the most important causes of premature death in almost throughout the world [1].

There are several risk factors for the coronary heart disease, which includes genetic factor, obesity, diabetes mellitus, hypertension, hyperlipidemia, cigarette smoking etc. Coronary heart disease is one of the complications of diabetes mellitus. Untreated long continued diabetes mellitus produces three times more Macrovascular coronary artery disorder than in normal individuals [2].

Long continued hypertension its also one of the important risk factors for coronary heart disease which is perhaps produced by the endothelial damage and atherosclerosis on the damaged tissue.

MATERIALS AND METHODS

Seventy cases of CHD of different age groups were selected for study from ICCU and indoor medical wards of Katihar Medical College & Hospital with the permission of the competent authorities.

Only those cases of CHD were selected for study who don't show any associated cardiac lesion on valvular heart disease or cardio-myopathy etc.

The diagnosis of CHD was established with W.H.O criteria. If any of the two changes present was considered to be suffering from CHD.

1. Chest pain.
2. ECG-changes.
3. Serum enzyme rise.

1. Standard 12 lead ECG was done and changes in ST segment, 't' wave and presence of pathological 'q' wave were noted in different leads.
2. Serum enzyme, (LDH) estimation by king's method SGOT estimation by Reitmen& Franke method,
3. Fasting blood sugar level by Folin & Wu method (1920)
4. Serum lipid – serum cholesterol and tryglycerides, both by priodynamic digital system (Kits).

OBSERVATION

The patients were observed in the hospital for a period of 2-3 weeks.

Risk factors	Age Groups					Total	%
	31-40yr	41-50yr	51-60yr	61-70yr	70yr		
Genetic factor	–	3	–	1	1	5	7.14
Obesity	1	1	3	1	1	7	10
Sedentary habbits	4	16	14	9	4	47	67.14
Personality type A	2	4	3	–	–	9	12.85
Smoking	4	14	14	9	2	43	61.42
Hypertension	3	9	12	5	3	32	45.71
Diabetes	1	9	10	9	1	30	42.85
Hypercholesterolemia	–	8	3	–	2	13	18.57
Hypertriglycerdemia	–	3	2	10	0	6	8.57

DISCUSSION

Family history of CHD, HTN, DM was present on 14.28% cases out of which only 7.14% showed positive family history of CHD. Where as Chesebro et al [3] observed family history of CHD out a younger age in 41% patients and Uhl et al [4] observed family history of significant atherosclerosis in 32% of patients on older age group and 69% of younger age group. The present observation vary from the above, It can be possibly explained that in India, Medical facilities were very poor previously and even now it is not better, so most of the patients remained undiagnosed. Subramanyam[5] also observed family history of CHD in 12.5% cases only which is very close to present study.

Obesity was present only in 10% patients in the present study. Which matched with the study.Uhel et al observed that half of the younger myocardial infarction patients were obese

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while 4% of older patients were obese. Subramanyam observed obesity in only 11.11% patients. There are conflicting view regarding obesity as on independent risk factors, but it is usually associated with hypercholesterolemia and hypertension. So obesity is probably an additive factors in patients with other risk factors.

In the present series 47 patients (67.14%) showed minimum physical activity while 22 (31.43%) showed moderate activity both at work and leisure. These observations are in conformity with the observation of Morris JN et al [6] & Kamnel WD [7] and American Health association committee report (1980) on coronary risk factors that physical exercise may protect CHD.

Fully developed personality type A was found in 12.85% patients of CHD in present study. All the cases were below 60yrs of age. Subramanyam observed personality type A in 11.12% cases.This figture is close to present study and difference can be explained that the number of cases in his study was quite large in comparison with present study.

In the present study 61.42% patients of CHD were smokers. Among them 43% patients smoked 20 cigarettes per day & 4 were hidi smokers. The findings are in harmony with most of the studies. Doyel J.T & Gordon, T found that after cessation of smoking cardiovascular risk begins to decline.

In the present study 32 (45.71%) were diagnosed hypertensive. Previously Subramanyam[8] observed hypertension in 22.06% patients in his series of cases. This variation may be due to his large number of cases. Various studies also observed hypertension to be the strongest predictive risk factor for CHD. Hypertension is less important in younger age group than in older patients as studied by Uhel et al.

In the present study 30 persons (42.85%) were diabetic. Keen et al (1965 [10]) observed association between CHD with in increasing level of blood sugar in both the sexes. Similar conclusion was also reached by Epstein et al 65. He also observed that the effect of hyperglycemia on arterial disease is independent of blood pressure and serum cholesterol. American health Association committee report on coronary risk factors suggested that hyperglycemia is associated with other risk factors as obesity, hyperlipidemia and hypertension and all these are associated with increased risk of CHD.

In the present study13 patients (18.57%) had significantly raised cholesterol level. Framingham's study also found raised plasma cholesterol as major risk factor for CHD. His study supports the present study

In the present study 6 patients (8.57%) showed increased Triglyceride (TG) level. Gotto et al [11] and Schaefer et al [12] also observed that serum TG level in their study was higher in CHD cases.

CONLUSION

From our present study we may conclude that sedentary habits(67.14%) and smoking (61.42%) were the most common risk factors for CHD followed by hypertension (45.71%), diabetes (42.85%), hypercholesteremia(18.57%), personality type A(12.85%), obesity (10%), hypertriglyceredemia (8.57%).

Though 7.14% of patients showed no apparent risk factors apart from the genetic factor.

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Respiratory Symptoms and Pulmonary Function Tests in Smokers of Katihar

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Abstract

Objectives : This study, using a questionnaire and pulmonary function tests (PFTs) was aimed at assessing the prevalence of respiratory diseases and the impact of cigarette smoking on these diseases. **Materials & Methods :** 101 male smokers & 101 male non-smoker residing in an urban area of Katihar are subjected to pulmonary function test. The data obtained was statically analyzed. **Results :** 48.51% subjects had one or more chronic symptoms. Cough was present in 31.68%, sputum production in 26.73%, wheeze 22.77% and wheeze & shortness of breath in 21.78%. Pulmonary functions were lower in subjects with symptoms as compared to those who were asymptomatic.

Key words : *Respiratory symptoms, smokers, PFTs.*

Introduction

Respiratory symptoms are the most common cause of presentation to the general practitioner. Chronic diseases of the respiratory system are one of the commonest cause of morbidity & mortality in India.(1)

Shortness of breath and cough are the primary symptoms for patients with respiratory system disease. Less common symptoms include wheeze, coughing up of blood, fever, chest-pain. Smoking is an established risk factors for the development of various respiratory diseases. Passive smoking is increasingly recognized as an independent risk factor Smoking is considered to be the self inflicted major health hazard world wide.(2) Smoking continues to be the largest preventable cause of premature morbidity and mortality throughout the world including chronic respiratory diseases such as Bronchial Asthma and Chronic Obstructive Pulmonary Disease (COPD).(3)

The aim of our analysis was to investigate the association between respiratory impairment and smoking.

Material and Methods

The study was conducted on 101 male subject age group 20-60 years taking more than 20 cigarette per day. & 101 non-smokers of the same age group.

PFTs were assessed by spirometry using RMS Helios 701 Spiroexcel. The measuring instruments were calibrated prior to each session. At least two acceptable spirometric measurements were obtained from a minimum of four forced expirations. This procedure is on accordance with the ATS criteria. (4). In our analysis, we used FVC, FEV_1, FEV_1 / FVC, PEFR.Linear regression model were used to predict the lung function parameters FEV_1 & FVC based on age, height, race & sex. We used the equations which are recommended by American Thoracic society. (4).

We considered PFTs to be impaired if FEV_1< 80% or FVC < 80% of the predicted value of the respective parameter.

Result & Observation

The prevalence of various respiratory symptoms in male smokers & non smokers are shown in Table - I & II. There were a total of 49 (48.51%) symptomatic subjects in smokers whereas 11 (10.89%) symptomatic in non-smokers.

List of Abbreviations :

FVC – Forced vital capacity

FEV_1 – Forced Expiratory Volume in one second.

PEFR – Peak Expiratory Flow rate

In smokers, out of 49 (48.51%) symptomatic subjects 32 (31.68%) showed cough, 27 (26.73%) sputum production, 23 (22.77%) wheeze and 22 (21.78%) wheeze with shortness of breath.

Table I : Prevalence of chronic respiratory symptoms

Group	Cough	Sputum production	Wheeze	Wheeze & Shortness of breath
Smoker	32(31.68%)	27(26.78%)	23 (22.77%)	22 (21.78%)
Non-smoker	5 (4.95%)	3 (2.97%)	2 (1.98%)	1. (0.99%)

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Table II : Observed values of Pulmonary function tests in relation to symptoms

Pulmonary Function Tests	Cough (Mean + SD)		Sputum Productions (Mean+ SD)		Wheeze (Mean + SD)		Wheeze & Shortness of Breath (Mean +SD)	
	Yes	No	Yes	No	Yes	No	Yes	No.
FVC	2.74 ± 0.18	3.01 ± 0.18	2.60± 0.27	2.78 ± 0.09	2.47± 0.37	2.82± 0.13	2.57 ± 0.52	2.78± 0.15
	P<0.05		P<0.05		P<0.05		P<0.05	
FEV_1	2.14 ± 0.61	2.23± 0.24	1.97 ± 0.65	2.25 + 0.17	1.86± 0.80	2.28± 0.52	1.90± 0.54	2.25 ± 0.53
	P<0.05		P<0.05		P<0.05		P<0.05	
$\frac{FEV_1}{PVC}$	76.41± 19.54	80.10 ± 3.41	76.59 ± 52.03	80.08± 6.41	79.70± 54.23	80.31 ± 7.67	81.47 ± 25.93	79.65 ± 4.20
	P<0.05		P<0.05		P<0.05		P<0.05	
PEFR	347.71 ± 155.22	379.21 ± 98.52	374.98 ± 117.03	382.65 ± 268.45	325.72± 228.20	384.05± 109.44	320.87± 119.30	380.05 299.61
	P>0.05		P>0.05		P>0.05		P>0.05	

* **Note :** *P value < 0.05 show significant. P value > 0.05 shows not significant.*

In Non- smokers, out of 11 (10.89%) symptomatic subject 5 (4.95%) shows cough, 3 (2.97%) sputum production, 2 (1.98%) wheeze and 1 (0.99%) wheeze & shortness of breath.

A statically significant relation of cough, sputum production, wheeze and shortness of breath with smoking was demonstrated in our studies.

Discussion

Our study showed significantly reduced pulmonary functions in smokers as compared to non-smokers among the parameters studied.

FEV_1 was most significantly reduced in the group of respondents who reported having wheeze and shortness of breath which implies that wheeze might be a good indicator of the presence of obstructive disease.

FVC, FEV_1 ,FEV_1 / FVC were lower in those with cough, sputum production, wheeze & shortness of breath than in those without the symptoms, and all these parameters have P value < 0.05 i.e. significant. PEFR was also lower in those with cough, sputum production, wheeze & shortness of breath than in those without the symptoms and have P value > 0.05 i.e. not significant. Our findings are similar to those observed by P Vaidya et al (7) and Behera D et al.(8)

FVC, FEV_1 ,FEV_1/FVC are significantly decreased in smokers but PEFR values are not significantly decreased.

The association of smoking with chronic bronchitis is well established. Non-smokers have a lower prevalence of disease than smokers which was corroborated by present study.

Conclusion

A statistically significant co-relation of cough, increased sputum production, wheeze and shortness of breath with smoking was demonstrated in our studies.

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ORIGINAL & CLINICAL RESEARCH

A Study on Incidence of Kala-Azar in Different Age and Sex Group of Inhabitants of Katihar along with Haematological Alterations

Dr. Sanjay Singh[1], Dr.Sanjay Tiwari[2], Prof.(Dr.) A. K.Ray[3]

NTRODUCTION

Leishmaniasis (kala-azar) is a group of protozoal diseases caused by parasites of the genus leishmania, and transmitted to man by the bite of female phlebotamine sandfly. They are responsible for various syndromes in humans — kala-azar or visceral leishmaniasis(VL), cutaneus leishmaniasis(CL), post kala-azar dermal leishmaniasis(PKDL) etc[1] The visceral type of disease, kala-azar, is still important disease in India. The majority of the leishmaniasis are zoonoses involving wild or domestic mammals (rodents, canines). Some forms (e.g. Indian kala-azar) are considered to be nonzoonotic infections[2].Leishmaniasis is endemic in many countries in tropical and subtropical region, including Africa, central and south America, Asia and the mid terranean region. About 2 – 4 lacs cases of VL are reported annually worldwide. Kala- azar is endemic in 52 districts in India. In Bihar(31), Jharkhand(4), West-Bengal(11)and Uttar Pradesh(4) districts are affected.

About 130million, population is at risk of the disease [3] The increase in leishmaniasis worldwide incidence is mainly attributed to the increase of several risk factors that are clearly man made and include massive migration, deforestation, urbanization, immunosupression, malnutrition and treatment failure[4].Kala-azar has been known to occur endemically in the eastern part of the Indian subcontinent, northern and eastern China, Africa and Latin America[5]

VL is a chronic infectious diease. It is characterized by fever, hepatomegaly, spleenomegly, weight loss, pancytopenia and hypergammaglobulinemia. Anaemia is the most common manifestation of VL. It may also be associated with leucopenia,thrombocytopenia, pancytopenia, haemophagocytosis and disseminated intravascular coagulation.

Haematological improvement is noted within a week and complete haematological respoose occurs in 4-6 weeks of treatment. Relapses are rare and increased risk of being diagnosed with haematolymphoid malignancies on long term follow- up is not noted[6].

AIMS & OBJECTIVES

An attempt has been made to study the

1. Relationship of different age and sex on kala-azar.
2. Haemoglobin alterations in different age and sex groups suffering from kala-azar.

MATERIALS AND METHOD

The study was conducted at the out-patient department of katihar medical college hospital (KMCH) with the permission of competent authorities. Blood sample of 130 diagnosed kala-azar patients were collected and haemoglobin level was determined for data analysis.

Sampling study was conducted on patients of age group 10yr to 60yr for data analysis of haemoglobin level of

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Table 1 Showing the age group, number of cases and sex ratio of kala-azar patients.

Age group	No. of cases	No. of male	No. of female
10 – 20	20	15(75%)	5(25%)
21 – 30	40	32(80%)	8(20%)
31 – 40	50	30(60%)	20(40%)
41 – 50	10	10(100%)	0
51 – 60	10	8(80%)	2(20%)

Table 2 Showing the age group, number of cases and the level of haemoglobin in kala-azar patients.

Age group	No. of cases	Haemoglobin level(g/dl)
10 – 20	20	8
21 – 30	40	8.5
31 – 40	50	7.5
41 – 50	10	8.1
51 – 60	10	8.5

kala-azar patients and its correlation with age and sex of the patients.Blood samples were collected in EDTA vial and the level of haemoglobin was estimated by blood cell counter (Medonic M-series).

RESULTS & DISCUSSION

Study of incidence of kala-azar patients and its correlation with sex ratio revealed that among all kala-azar patients the male patients were more in number than female patients in every selected age group (Table-1).

Data analysis of haemoglobin level of kala-azar patients indicated that there were more number of cases from the age groups of 31yr – 40yr having severely depleted level of haemoglobin (7.5g/dl) in comparison to the normal level of haemoglobin(12g/dl – 15g/dl) found in healthy patients of the age group of 10yr – 60yr (Table-2).

In a study it has been also observed that anaemia in all the cases of kala-azar is very common which is followed by neutropenia(43%)lymphocytosis(86%) with thrombocytopenia(79%), bone marrow in most of the cases showed myeloid hyperplasia with increased megakaryocytes[7] .The cellular infiltrate and parasitization of the RES is accompanied by typical cellular and biochemical changes in the blood. Erythrocytes are sequestrated in the spleen and have shortened to half(1/2) due to haemolysis[8]. In present study similar findings anaemia has been reported.

As early as in 1906, Rogers found certain characteristic haematological changes in Indian kala-azar such as anaemia, Leucopenia & occasionally thrombocytopenia, The life spans of RBCs were reduced to half as compared to normal, It was also confirmed that there is marked sequestration in the spleen as demonstrated by Cr 51 tagged erythrocyte study, leading to the conclusion that autoimmune mechanism is responsible for anaemia in kala-azar or visceral leishmeniasis misdiagnosed as connective tissue disorder is well reported in literature. [9 10] It has been observed by other worker that the haematological changes are result of direct bone marrow damage by the organisms.[11] The bone marrow becomes hyperplastic, and parasitized macrophages replace the normal haemopoitic tissues. In a laboratory investigation, 7.89g/dl, haemoglobin has been recorded in kala-azar patients[6].

Study of incidence of kala-azar patients and its correlation with sex ratio revealed that males are more affected than females with the disease. Data analysis of haemoglobin level of kala-azar patients and its correlation with age of the patients indicated that age group of 21yr – 30yr, were more susceptible to the dreaded diseases, kala-azar. However for that, further investigations would be very much helpful in drawing conclusive inferences.

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A Study on Nutritional Anemia with Special Relation to Hemoglobin, MCV And MCHC Among Defferent Age and Sex Group in Inhabitants of Katihar

Dr. Sanjay Tiwari[1], Dr. Sanjay Singh[2], Prof.(Dr.) A.K. Ray[3]

INTRODUCTION

The term Anaemia or Anemia literally means "without blood". Anemia comes from the greek word anaemia meaning "lack of blood". The anemia is defined as decrease in total number of red blood cells or less than the normal quantity of hemoglobin in the blood. This results in reduced ability of blood to transfer oxygen to tissues.

Historically (1500BC), IN Ayurvedic literature, charak samhita described fatigue and pallor caused by 'bloodlessness" which can be cured by Lauha bhasm calcified iron. In greek literature (1554-1700) 'chlorosis or green sickness" was described as curable by drinking iron rust dissolved in water or wine. Hemoglobin is the iron containing oxygen transport metallo-protein in the red blood cells of venebrates[2] and the tissue of some invertebrates. Hemoglobin has an oxygen binding capacity between 1.36 and 1.37 ml of oxygen per gram of hemoglobin[3] which increases the total blood oxygen capacity to seventy fold[4].

The world Health organization defined anemia as hemoglobin or hematocrit level below normal for the age ,sex, attitude and physical state of an individual[5].In children aged six months to five years anemia is defined as hemoglobin less than 11 g/dl while those above five years to fourteen years it is less than 12 g/dl. Anemia is widespread public health problem.

The world Health organization estimates that over 2 billion people are anemic worldwide. It primarily affects women [6]

Anemia is a common condition worldwide although the incidence is highest in the developing countries where the nutrient deficiencies and chronic infection are prevalent. A Significant percentage of adolescents in the developing world are anemic causing consitlerable health consequences for this age group. In India, in 1968, Dr Gopalan constituted an expert committee of the nutrition society of India, to suggest measures to control anemia in country. About 44% populations are estimated to be anemic in developing countries compared to 13% in developed countries. (As in project "Anemia free India 2005").

Dietics and nutrition survey in India reveal that 87% of pregnant women suffer from anemia. According to the nutrition Foundation of India, 90% of adolescent girls, women and children suffer from iron deficiency[7, 8]. In most developing countries, anemia in pregnancy makes an important contribution to maternal mortality and morbidity [9]. A hemoglobin concentration of less than 11g/dl is commonly taken as indicative of anemia in pregnancy[10].

Nutritional anemia is a disease syndrome caused by malnutrition in its widest sense. By far the most frequent cause of nutritional anemia is iron deficiency and less frequently folate or vit B12. Nutritional anemia is worldwide problem with highest prevalence in developing countries. It is found especially among women of child bearing age, young children and during pregnancy and lactation. It is estimated to affect nearly two-third of pregnant and one-half of non-pregnant women in developing countries[11].

Nutritional anemia is diagnosed on the basis of MCV and MCHC. MCV Less than 76 fl and MCHC Less than 31g/dl is diagnosed as iron deficiency (microcytic hypochromic) anemia and MCV more than 96 fl and MCHC less than 31g/dl is megaloblastic (macrocytic hypochromic) anemia.

MATERIAL AND METHODS

The study was carried out in the out patient department and patients admitted in wards of Katihar Medical College and Hospital, Katihar, with the permission of competent authorities. The cases include male and female above the age of 11years and less than 50years. 200 Patients including male and females of newly diagnosed anemia of different age & sex group were selected. 3ml venous blood samples from 200 patients were collected in E.D.T.A containing vials.

A complete blood count was done on electronic cell counter, (ACCUREX INSTRUMGNT-CBC-360-AUTOMATIC HEMATOLOGY ANALYSER).

RESULT AND DISCUSSION

Table 1 : Showing the incidence of anemia in different age & sex groups:-

Age (in years)	Male	Female	Total No of cases
11-20	15	24	39
21-30	30	42	72
31-40	16	44	60
41-50	9	20	29

Incidence of anemia was maximum in male between the age group (21 – 30)

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Out of 200 cases of anemia nutritional anemia is 140. Of these 80 cases (40%) are microcytic hypochromic iron deficiency anemia and 60 cases (30%) are macrocytic hypochromic (megaloblastic) anemia.

Table 2 : Showing the incidence of microcytic hypochromic anemia in different age groups:-

Age (in years)	No. of cases	Percentage
11-20	15	18.75
21-30	30	37.5
31-40	21	26.25
41-50	14	17.5

Maximum cases of microcytic hypochromic anemia were found in age group (21 – 30) followed by age group (31 – 40).

Table 3 : Showing the incidence of macrocytic hypochromic anemia in different age groups:-

Age (in years)	No. of	Percentage cases
11-20	18	30
21-30	24	40
31-40	10	16.6
41-50	8	13.3

Maximum number of macrocytic hypochromic anemia were found in age group (21 – 30) followed by age group (11 – 20).

Table 1 : Showing the incidence of types of anemia in different sexes:-

Type of anemia	Males		Females	
	No. of cases		No. of cases	%
Microcytic hypochromic	30	37.5	50	62.5
Microcytic hypochromic	18	30	42	70

According to sex wise incidence, microcytic hypochromic anemia was maximum in both male and female.

A Study was done among 280 health workers ranging 19-56years and found that microcytic hypochromic anemia was most common in both sexes[12]. In our study too, microcytic hypochromic anemia was most common 40% followed by macrocytic hypochromic 30% which is very much close to the above result. Highest incidence of nutritional anemia was found in age group 21-30 in our study in both sexes. Similar findings were observed by Napier and Das Gupta.

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where as in females it was maximum in age group (31 - 40).

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ORIGINAL & CLINICAL RESEARCH

An Evaluation of Prescribing Pattern of the Private Practitioners

Dr. Sudha Kumari[1], Dr. Sanjay Singh[2], Dr. A.K. Ray[3]

INTRODUCTION

Medically inappropriate and economically inefficient use of medicines is observed throughout the world. These features are more marked in the developing countries like India. Rational use of medicines is one essential element to be achieved to improve quality of health and medical care for the patients and the community, Laing, 1990:.

Medical science in general and therapeutics in particular is developing very quickly under going fast transition. Therefore it has become imperative to train the physicians for self-directed learning (Joshi, 1996). Prescribing appropriate medicines for a disease condition and proving related information in a meaningful way to the patients should be regarded as the key 'transferable skills' to be achieved through pharmacology courses, Shankar et al; 2003). Generalized Presence of irrationalities in Prescribing indicates that traditional teaching in medial schools does not adequately prepare students for rational therapeutics. Prescribing behavior of the medical graduates depends upon how and what they have been taught and trained about drugs during their undergraduate course (Schwartz and Griffin, 1986). A survey had revealed that medical students felt the need for more teaching of therapeutics (Ward and Miolzweski,2002). The current study was and attempt to evaluate prescribing, whether appropriate or rational.

MATERIALS AND METHODS

Prescriptions of the registered Physicians and specialists of different sectors of town at random within a period of two months from june 10 to august.2016 A total of 500 prescriptions were collected during this tow month long prospective study from the private clinics They were randomly approached either outside at chemist shop with a request to have their Prescriptions Photocopied. The clinic.

Collect4ed Prescriptions of Private Practitioners were analysed on the basis of following parameters:

(I) To estimate the total number of drugs prescribed.
(II) Generic Vs brand products.
(III) Commonly Prescribes drugs.
(IV) Total injectable Presparation.
(V) Prescription in format or not.

No attempt has been made to categorize the Prescriptions according to patients age, sex or disease profile. Results After compiling the results it was observed that there were average 4 drugs per prescriptions(Table). Only in 10 prescriptions the drugs were prescribed in generic name; only 30% of Prescriptions were complete in regard

Table 1 : Results of prescription audit (n=500)

Prescribing indicators	Number
Average drug per prescription	20,(4%)
Prescribed in generic name(%)	10(2%)
Antibiotics prescribed (%)	350(70%)
Injections Prescribed (%)	70(14%)
Percentage of drugs prescribed from essential drug list	250(50%)
Whether Prescription is complete with respect to Format	150(30%)
Dosage and duration	375(75%)
Patient medical information	100(20%)

Standard prescription format; only 50% drugs were prescribed from the essential drug list; only 20% of prescriptions were complete in respect to patient medical information. Antibiotics were prescribed in 70.33% of the prescriptions; injections were prescribed in about 4.3% of the prescriptions.

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DISCUSSION

Researchers have collected, analyzed and audited500 prescriptions of the private practitioners using INRUD indicators. Through the exercise it was revealed the most of the private prescribers dit not follow the criteria of rational prescribing. On an average, 3.78 drugs were prescribed per prescription, which was 3.41 in a study conducted in 1996 (Baqui and Choudhury, 1996) and 3.31 (chukwuani et al, 2002). In the current study, the prescribers prescribed the drugs in generic name only in 10 prescriptions (2.55%), which was much lower than (4.10%) the finding of the previous study (Baqui and Choudhury,1996). In the current study revealed that prescribers frequently prescribed antibiotics (70.3%) in their prescriptions. This finding is in agreement with the study done by Baqui and Choudhury (1996) where the percentage of patients receiving antibiotics was 75.33%. In an Iranian study (Ansari, 2001) percentage of patients receiving antibiotics was found high (86.2%). However, all these finding of a study conducted In 1998, which reported only 40.7% prescriptions contain antimicrobials (Rahman et al; 1998). In the present study, about 50% of the drugs were prescribed from the Essential Drug List which was almost similar, i.e; 49% to the findings of Baqui and Choudhury (1996) and 8.2% (Rahman et al; 1998). About 60% patients were provided with proper instructions regarding drug dosing and duration (Baqui and Choudhury, 1996). Which has increased to 70% nevertheless, only 12.4% prescriptions contained proper instructions about side effects of the prescribed drugs, other relevant advice and follow up of the patients.

From these observations it was evident that the prescribing pattern of the private practitioners is not improving regarding some particular parameters like generic perescribing, polypharmacy, use of antibiotics and provision fo information. The reason of this irrational prescribing is perhaps dur to the lack of knowledge of the private practioners on how to prescribe a drug' and 'what information they should provide to their patients' (deVries, 1994; Rahman et al; 1998). The present exercise was an attempt to evaluate the prescribing pattern.

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A Preliminary Study on Factors Affecting Drug Abuse in Medical Students in Medical College and Hospital in Kolkata & Jhanshi

Dr. Sudha Kumari[1], Dr. Sanjay Singh[2], Dr. A.K.Ray[3]

ABSTRACT

Drug abuse is a very important social problem. The current study was carried out to ascertain the pattern of drug abuse among the medical students in a tertiary care medical college.

Materials and methods:- A prospective, cross-sectional questionnaire based study was conducted in a tertiarycare medical college in Jhanshi from January 2016 to March 2016.

Results : A total of 150 students successfully completed the questionaire. Among them 90(60%) students reported substance abuse. The source of introduction to drugs was friends in majority of the cases. The major reasons of drug abuse are curiosity i8n maximum students followed by a measure to combat stress.

Conclusion : It is quite evident from the study that the parents of medical students in the young age group should be more vigilant in their day to day activities.

INTRODUCTION

Drug abuse is a very important social problem.It is particularly common in younger age group, specially the student population. Studies in India have indicated that almost 25% of student and non-student use alcohol and 20% use other dtugs. 1Substance abuse assumes a special significance among the medical students as they are the future medical practioners and have a potential role in treating and counseling the patients of substance abuse disorder. In this prospective the current study was carried out to ascertain the pattern of drug abuse among the medical students.

Table 1 : Source of Introduction to first drug

Source of Introduction to first drug use	Number
Friend	65 (72.23%)
Advertisement / Promo	20 (22.23%)
Others	5 (5.5%)

Table 2 : Reason for drug use

Reason	Number
Curiosity	60 (66.67%)
To be considered smart and social	20 (22.23%)
Relief of psychological stress	6 (6.66%)
'Others	4(4.44%)

MATERIALS AND METHODS

A prospective cross-sectional study was conducted in a tertiary care medical college in Kolkata from January 2016 to March 2016. A predesigned questionnaire was given to the 2nd year medical students during their routine pharmacology classes. Informed consent was taken from all the students before supplying the questionnaire. They were requested to fill the questionnaire assuring confidentiality about their identity. The questionnaire was designed to elicit history about drug use, factors provoking drug use. The returned questionnaires were checked for completeness and incompletely filled questionnaires were excluded.

RESULTS AND DISCUSSION

A total of 150 students successfully compared the questionnaire. Among them 90 students reported substance abuse. The source of introduction to drugs was friends in majority of the cases followed by advertisement and promos. The major reasons of drug abuse are curiosity in maximum students followed by a measure to combat stress. Details of the table are shown in Table 2. Previous studies conducted with this objective also gave similar results. In studies conducted by Ganguly, Curiosity was the major cause for 58.8% of drug use followed by peer pressure (13.61%) and relief of psychological stress (13%). Curiosity is also the laeding cause in study conducted by Jagnany et al.

CONCLUSION

It is quite evident from the study that the parents of medical students in the uoung age group should be more vigilant on their day to day activities. The students should learn to cope with stress. IEC activities should raise voice against drug abuse.

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Association of ABO Blood Group with Breast Cancer: An Observational Study

Neelima Kumari[1], Ashutosh Kumar[2], Manish Kumar[3]

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Abstract

Introduction: The present study was conducted to analyse the relation of ABO blood groups with breast carcinoma.

Materials and Method: The study was conducted on 100 clinically diagnosed breast cancer patients. The standard agglutination test was used to determine the blood groups. Association of ABO blood groups and risk of breast cancers was found out with Odd Ratios (ORs) with 95% Confidence Interval (CI).

Results: Breast cancer was found minimum in blood group 'AB' and maximum in blood group 'A'. It may be due to influence of blood group antigens on systemic inflammatory response which has been associated with the malignancies. The ABO antigen expressed on the surface of malignant cells appears to be different from the antigen expressed on normal tissue.

Conclusions: High frequency of breast cancer was found in blood group A followed by B and O strong relationship between blood group and breast cancer. The different expression of antigens on the surface of cancer cells might alter motility, apoptosis and immune escape. These mechanisms might influence the initiation and spread of malignancies.

Keywords: *ABO blood group, Breast cancer.*

Introduction

About one million new cases of breast cancer are diagnosed every year. [1] In some tumors, alteration of ABO/Lewis-related antigens is associated with malignant transformation.[2] Blood group carbohydrate antigens on the surface of cancer cells can be regarded as an end product of tumor progression that can be used as useful prognostic and diagnostic markers. [3] ABO blood group genes are mapped at 9q34.2 region in which genetic alteration is common in many cancers. The loss or presence of blood group antigens can increase cellular motility or facilitate the interaction between tumor cells and endothelial cells of distant organs. [4] In many cancers, the deficiency of A or B epitope has been reported which is associated with accumulation of their precursor, which causes enhanced malignancy.

Material and Method: This observational study was conducted in the Department of Physiology, Surgery and Obstetrics & Gynaecology of Katihar Medical College, Katihar for a period of 12 months from May 2017 to April 2018.

A total of 100 newly and confirmed diagnosed breast cancer patients were taken for this study as cases. A written informed consent was obtained from all subjects before their participation. The data of age, sex, ABO blood group and pathological status of cancer were collected from the outdoor department.

Inclusion criteria:

1. Female patients of any age group.
2. Pathologically confirmed diagnosis of breast cancer attending OPD.

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Exclusion criteria:

1. Familial cancer history,
2. Patients on oral contraceptive pills,
3. Patients having menopause.

History taking, detailed physical examinations performed, routine radiological and laboratory investigations including complete blood count (CBC), tumor markers for breast cancer was done.

Blood samples were obtained into vacuum glass tubes containing EDTA. ABO blood typing was carried out with standard agglutination method. ABO blood groups were determined by using antiserum A and Antiserum B. [5]

Standard Agglutination Method: In agglutination test firstly, we prepare red cell suspension in a test tube and then in under aseptic precautions add a drop of blood. Then a drop of each antiserum (antiserum A, antiserum B) on is placed on glass slide with the help of dropper and a drop of isotonic saline (used as control) also placed on the slide. The slide is accordingly labelled as anti-A, anti-B and control. After 10 minutes, examined for the presence of agglutination (clumping of RBC) under low power microscope, if there is no agglutination (RBC remain separated and evenly distributed), and if agglutination occurs the RBC are massed together in clumps.

Statistical analysis: For each factor, we calculated the adjusted Odds Ratios (OR) and 95% confidence Interval (CI) using maximum likelihood estimation.

Results

Table I: Association of risk of breast cancer in relation to ABO blood group

Blood Group A		Blood Group B		Blood Group O		Blood Group AB	
No. of cases	OR's with 95% CI	No. of cases	OR's with 95% CI	No. of cases	OR's with 95% CI	No. of cases	OR's with 95% CI
n= 37	8.54	n=33	7.28	n=23	4.88	n=7	2
(0.476-2.103)		(4.098-13.522)		(3.365-11.195)		(2.087-7.169)	

In this study we found that there was an association exists between blood groups A with breast cancer in sample population. Above table described a total of 100 breast cancer cases. Maximum cancer cases were found in blood group A.

Discussion

Blood group A person, who cannot make anti-A antibodies will be more likely to tolerate cancer, and blood group A person's immune system will less likely to attack the body's own tissues.[6]

A study of rapidly progressive breast cancer in Tunisian women found a slightly increased risk of a positive diagnosis in blood type A was reported by Mourali. [7] There are also some contradictory reports available about the association of blood group with breast cancer.

Jayant K [8] reported no relation among breast cancer to blood groups whereas Surekha et al [9] have reported a high incidence exist between breast cancer and blood group B individuals. In the last 25 years, there has been a tremendous amount of work published on the chemistry of blood group antigens and tumor immunology.

As cells (e. g. in tissue) become malignant, they tend to lose normal antigens and acquire new antigens; these are so called tumor antigens. It has been proven that ABO antigens diminish on malignant cells as the malignancy progresses the loss of A, B and H antigen is proportional to the metastatic potential of the tumors.[8, 10] The reason that deletion or reduction of the A or AB antigens in tumors of A or B individuals correlate with malignancy a metastatic potential may be due to lack of adhesiveness that a cancer cell achieves when its losses blood group antigens. The loss of blood antigen results in the tumor cells gaining the ability to move and circulate through the body, because blood type antigens loss the ability to express many cell adhesion proteins, such as integrins, which normally express an A like antigen on their receptor and control cell movement. [11]

Blood group A cancer patients had the greatest and most uniform suppression of the level of Tn antigens, irrespective of age, cancer stage, or tumor morphology and lower level of anti-B isohemagglutinnins. This is probably at least a part of the explanation for the poorer outcomes in many cancers among blood group A individuals. [12]

Hakomori suggested that if the immune surveillance theory is correct and we recognize tumor antigens as foreign, leading to attack of the tumor, then the "A-like" properties of tumor antigens may not be recognized by group A patients. [13]

Tumor Immune Surveillance in the immune system can specifically identify and eliminate tumor cells on the basis of their expression of tumor specific antigens or molecules induced by cellular stress whereby immune system identifies the cancerous or precancerous cells and eliminates them before they can cause harm. [14] It would be interesting to know that the percentage of patients in this particular study were of Blood Group "A". [15] It appears that a more integrated treatment protocol should be considered using conventional modalities as well as dietary modifications.

Blood Group "A" individuals have a very low immunologic response to T and Tn antigens because they share the same sugar (N-acetygalactosamine). This allows the cancer cells to bypass the immune system and replicate with little interference from the type A antibodies will have an effect on cancer survivorship. [15]

Conclusion

Some studies on blood groups showed positive association and others were negative. It appears that different blood groups are associated with breast cancer; Blood group A apparently increases the risk for cancer. This study concludes that, in case of breast cancer, high frequency of breast cancer was found in blood group A followed by B and O strong relationship between blood group and breast cancer.

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Conflict of Interest: None

Ethical Clearance: Taken

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Study of Minute Ventilation, Maximum Voluntary Ventilation and Dyspneic Index During Pregnancy: An Observational, Prospective and Comparative Study

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Abstract

Introduction: This study was designed to evaluate the Minute ventilation (MV). Maximum Voluntary Ventilation (MVV) & Dyspneic Index (DI) in different trimesters of pregnancy and compare the results with non pregnant control group.

Materials and Method: This study was carried out in 80 healthy women in the age range of 20-40 years with 20 subjects each in 1st, 2nd, 3rd trimesters of pregnancy and non-pregnant control group. The respiratory parameters were recorded in study and control groups. Statistical analysis was done by SPSS Software Package.

Results: It was observed that there was a significant decrease in MVV and dyspneic index (DI) in all trimesters of pregnancy and an insignificant variation in MV when compared to the control group. These changes are due to pressure of enlarging gravid uterus, elevating the diaphragm and restricting the movements of lungs thus hampering forceful expiration. The decrease seen in MVV in 1st trimester might be due to the effect of bronchoconstriction due to decreased alveolar Pco2.

Conclusions: Decrease in respiratory parameters was seen particularly in first trimester of pregnancy compared to 2nd & 3rd. The normal Minute Ventilation tries to maintain the respiratory need of pregnancy at rest. At increased physiological needs of respiration or during exercise the decreased Maximum Voluntary Ventilation makes pregnant female dyspneic.

Keywords: *Pregnancy, Minute Ventilation, Maximum Voluntary Ventilation and Dyspneic Index.*

Introduction

The changes that occur in thoracic cage are rise in the diaphragm by four centimetres, widening of sub-costal angle increasing the transverse diameter by two centimetres & thoracic circumferences by six centimetres. These changes begin before the size of uterus can have an effect. [1] In Pregnant women there is increases in minute ventilation (VE), tidal volume, alveolar ventilation and a reduction in arterial PCO2. [2, 3] There is renal excretion of bicarbonate, resulting in a state of partly compensated respiratory alkalosis (arterial pH 7.43-7.47). [4] These effects appear in the first trimester and may promote placental gas exchange before development of an effective fetal circulatory system. [4]

The aim of the study was to evaluate the effect of pregnancy on Static & dynamic lung volumes and capacities in the subjects of Indian pregnant women in the age range of 20-40 years in different trimesters of pregnancy and compare them with healthy age matched non-pregnant control group.

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Material and Method: This observational, prospective and comparative study was conducted in the Department of Physiology and Obstetrics & Gynaecology of Katihar Medical College, Katihar to determine the pulmonary function changes in 1st, 2nd & 3rd trimesters of pregnancy and results were compared with age matched healthy non pregnant women. This study was conducted for 6 months from October 2017 to April 2018.

The study group comprised of 80 pregnant women in the age group of 20-40 years. This study group was further subdivided into 3 subgroups. Each sub group comprised of 20 women in 1st, 2nd and 3rd trimesters of pregnancy. The Control Group comprised of 20 healthy age matched (20-40 years) non pregnant women. The study was explained to the subjects. An informed written consent was obtained. A thorough physical & systemic examination (cardiovascular and respiratory system) of each subject was done. Recordings were taken between 8 am to 11 am.

Inclusion criteria:

1. All apparently healthy female subjects (80 pregnant and 20 non pregnant) between 20-40 years of age group were included in this study.
2. The health status of the subject was determined by history taking and thorough clinical examination.

Exclusion criteria:

1. Asthma,
2. Acute respiratory infection in the previous three months,
3. History or clinical signs of cardiovascular diseases, diabetes mellitus, hypertension, tobacco consumption, alcohol intake,
4. Endocrine disorders,
5. Obesity,
6. Moderate to severe anaemia.

The following parameters were recorded in each subject:

A. **Anthropometric parameters** like Height (in centimetres), Weight (in kilograms). Body Mass Index

B. **Respiratory parameters**: The subjects were informed about the procedure. For each test, three readings were taken. The highest of the three was considered for calculation. All tests were recorded in a sitting posture at room temperature, in morning hours.

I. Respiratory Rate (RR) (cycles/minute) was recorded.

II. The following pulmonary parameters were recorded by Computerized Spirometer

1. MV (Minute Ventilation =TV x RR in L/min). Minute Ventilation (MV) or Pulmonary Ventilation (PV) is the volume of air expired or inspired by the lungs in one minute. Normal value: 6 L/minute. [7]
2. MVV (Maximum Voluntary ventilation in L/min). It is the largest volume of air that can be moved in and out of the lungs in one minute by maximum voluntary efforts. Normal: 120-170 litres/minute. [8]
3. DI (Dyspneic index = MVV-MV)/MVV) x 100. Refers to breathing reserve percentage of MVV. Breathing reserve is the difference between MVV & MV. Normal value-70-95% and DI <60% is dyspnea. [9]

BMI: Body mass index, RR: Respiratory rate, MVV: Maximum voluntary ventilation, MV: Minute ventilation, DI: Dyspneic index of pregnancy.

Statistical analysis: The results were expressed as Mean±SD. Comparison done between the study (1st, 2nd and 3rd trimesters of pregnancy) and control groups and data were statistically analysed using SPSS software. p value ≤0.05 was considered statistically significant.

Results

Table 1: Age, anthropometric and respiratory parameters of different study group subjects

Parameters	Group 1	Group 2	Group 3	Group 4	P Values
Age (yrs)	26.08±5.76	27.02±4.41	26.76±3.57	27.84±3.39	0.200
weight (kG)	56.68±8.61	50.24±6.09	52.48±6.08	57.46±8.23	0.000
bmi (kg/m^2)	22.17±3.4	20.91±3.76	21.37±3.69	23.71±2.98	0.001
rr (PM)	16.72±3.00	23.26±3.00	24.38±4.00	27.26±3.00	0.000
mvv (l/min)	70.28±18.63	39.82±11.78	40.61±14.16	40.35±13.72	0.000
mv (l//min)	14.34±7.54	14.68±8.09	14.28±7.22	15.42±5.84	0.752
DI (%)	78.35±11.80	60.84±25.87	48.98±53.01	55.66±29.55	0.000

Anthropometric parameters: The Mean±SD of age, weight, BMI have been shown in Table 1. All groups are similar by age. There was a decrease in weight in 1st & increase in 3rd trimester compared to control. BMI increased significantly in 3rd trimester compared to control

Respiratory parameters: The Mean±SD of RR. MVV, MV and DI have been presented in Table I. There was a gradual increase in RR from 1st to 3rd trimesters compared to control. There was no significant difference in the minute ventilation between the study and control groups. A highly significant decrease in MVV was observed in all trimesters with a maximum decrease in 1st trimester. DI was significantly reduced in all trimesters compared to control group with maximum decrease in 2nd trimester.

Discussion

The present study showed a significant increase in weight & BMI in 3rd trimester. [10] A significant increase in RR from 1st to 3rd trimester of pregnancy as compared to control group which is in agreement with Bernhard Heidemann, who stated that PaCO2 falls and then levels off at 4.1kPa (31 mmHg) by the end of the first trimester. This is caused by a 10% increase in the respiratory rate, secondary to progesterone mediated hypersensitivity to CO2, and an increase in alveolar and minute ventilation, secondary to increased respiratory rate and tidal volume.[11]

Present study showed insignificant increase in MV in all trimesters as compared to control group. A study by Emilia Kolarzyk showed increase in MV during pregnancy. The increase in MV was caused by a significant increase in tidal volume. [12] The study by Aaron P also showed increase in MV which is due to changes in osmolality, (SID) strong ion differences & angiotensin II levels, which have been implicated in the control of ventilation.[13]

There was a significant decrease in MVV in all trimesters compared to control group with maximum decrease in 1st trimester. The decline in the MVV in first trimester is due to morning sickness (lack of nutrition) and also due to lodging of trophoblast cell in the alveoli from the maternal uterine sinuses. In the 2nd and 3rd trimester, it may be due to mechanical pressure of enlarging gravid uterus, elevating the diaphragm and restricting the movements of lungs and thus hampering the forceful expiration and may also be due to bronchoconstriction effect of decreased alveolar Pco2. [14] Present study also demonstrates a significant decrease in DI in all trimesters as compared to control group with maximum decrease in 2nd trimester. The decrease in the DI shows that pregnant women in all trimesters are dyspneic on exertion, [5] but some individuals showed negative DI indicating dyspnea at rest in all trimesters.

Conclusion

The normal Minute Ventilation tries to maintain the respiratory need of pregnancy at rest. At increased physiological needs of respiration or during exercise the decreased Maximum Voluntary Ventilation makes her dyspneic. Further studies are needed to establish the cause for decrease in respiratory parameters particularly in first trimester of pregnancy compared to 2nd & 3rd.

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Conflict of Interest: None

Ethical Clearance: Taken

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An Observational and Comparative Study of Diurnal Variation of Spirometry Test Parameters among First and Second Year Normal and Healthy Medical Undergraduate Students

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Abstract

Introduction: This study was designed to assess and compare diurnal variability of FEF25, FEF50, FEF75, FEF25-75, PEF and FEV1 by measuring these parameters during morning and evening hours in normal healthy subjects.

Materials and Method: 190 students were enrolled and divided intogroupsof 8-10 students. Each group were directed to appear at different dates in Pulmonary Function Test (PFT) Laboratory at 7:30 AM and again at 5:00 PM for spirometry testing. Spirometry was performed with Spiro Excel 1.1 as per the ATS guidelines and by trained technician. Finally, data from 169 subjects was found to be complete and appropriate and was taken for the analysis.Diurnal variability in FEF25, FEF50, FEF75, FEF25-75, PEF and FEV1 were determined and compared.

Results: Allparameters were more in male than female. All the parameters were significantly high in evening tests as compared to morning tests except FVC. Diurnal variability among different spirometry parameters was significantly different (ANOVA, $p<0.05$) in morning and evening tests. The diurnal variability was highest in large airways as reflected by FEF75 and lowest in smaller airways as reflected by FEF25. The diurnal variability was lowest for FEV1%. It revealed that all parameters exhibit significant diurnal variability.

Conclusion: FEV1, FEF and PEF had shown diurnal variability which was directly related to the airway calibre. Greater variability was seen in PEF as compared to FEV1 i.e. proximal airways showed greater diurnal variation than distal airways.

Keywords: *Spirometry test, Pulmonary Function Test, Diurnal variability, FEV1, PEF.*

Introduction

Variability in calibre of airways is a normal physiological process in normal personsand this variability may become exaggerated in patients of asthmatic and chronic obstructive pulmonary disease (COPD).Measurement of bronchial hyper-reactivity and airways variability has always posed challenge performing experiments on pulmonary function. Variability in peak expiratory flow (PEF) has been suggested as indicator for bronchial hyper-reactivity.[1-8]

The phenomenon of nocturnal asthma has always perplexed clinician's and researcher's mind. Peak expiratory flow rate (PEFR) variability has been

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suggested as a marker for bronchial hyper-reactivity in asthmatic individuals.[9,10] PEFR variation has been widely advocated and used in clinical practice and asthma research. The National Heart Lung and Blood Institute (NHLBI) and others have recommended, a diurnal variation of 20% or more, as a diagnostic benchmark for asthma.[11,12]

Airway function exhibit variability over 24-h periods. This variability has a base that lung function gets worse at night in nocturnal asthma patients and to a lesser extent with COPD.[13-16] As nocturnal asthma is common and troublesome,[13,14,17] circadian variation in airway function has been of considerable interest in respiratory medicine. It has been recognized that diurnal variation in airway calibre occurs in healthy subjects as well.[18-20]

It has been suggested that diurnal variation of PEFR in excess of 20% can be used for diagnosis of bronchial asthma in remission where routine spirometry may not show any significant obstructive defect.[19,21] Previous studies mentioned that PEFR shows time to time variation with respect to day and night cycle with specific pattern of lowest at early morning and highest at evening in normal as well as in asthmatics.[6,7,22] PEFR variation has been widely advocated and used in clinical practice and asthma research.

Several evidences suggest that airway variability exhibits a definite circadian pattern in which morning PEF levels are lower than daytime values, with a minimum in early morning and peak in evening. [6,7,22,23] The pattern of variability is exaggerated in smokers and in COPD and in asthmatic patients.[6]

The various spirometry indices reflect airflow characteristics of different airways. Forced expiratory flow (FEF), at 25% FVC, i.e. FEF25 reflects small airways, at 75% FVC (FEF75) reflects large airways and at 50% FVC (FEF50) reflects mid/small airways. FEF from 25% to 75% FVC (FEF25-75), reflects mid/small airways and is also known as mid expiratory flow. Forced expiratory volume in one second (FEV1) reflects the calibre of both large and small airways, whereas PEF is more a reflection of the calibre of large airways.[24,25] In general FEV1 is a more reliable indicator of airflow limitation than PEF. [26]

Unfortunately, most studies that describe diurnal variability in airways calibre in asthmatics have used PEF rather than FEV1. Moreover, the diurnal variability of small, mid and large airways has not been studied systematically. This study was designed to assess and compare diurnal variability of FEF25, FEF50, FEF75, FEF25-75, PEF and FEV1 by measuring these parameters during morning and evening hours in normal healthy subjects.

Materials and Method

Study Site/Place: This study was conducted in the Department of Physiology of Katihar Medical College, Katihar.

Study Duration: September 2018 to February 2019 (Six months).

Inclusion Criteria:

1. First and second year MBBS students
2. 17 to 30 years of age and all the gender
3. Healthy students having almost similar daily routine

Exclusion Criteria:

1. Students have history of smoking.
2. History of severe chest trauma, with chest and spinal deformity.
3. Personal/family history of asthma, chronic obstructive pulmonary diseases and
4. Personal/family history of other cardiovascular and/or respiratory diseases.

Study Design: An observational and prospective study.

190 students,100 from first and 90 from second year MBBS batch students were selected for this study after considering inclusion and exclusion criteria. The study protocol was duly approved by Head of the Department of Physiology and Pharmacology. After enrolment students were explained about the study.A thorough clinical history was taken and anthropometric measurements (height and weight) were recorded. Brief clinical examination was done to rule out any obvious cardio-pulmonary compromise.

The Pulmonary Function Test was done using Digital Spirometer Machine (Spiro Excel 1.1 by Medicaid Systems). Parameters which had been interpreted like Forced Expiratory Volume in 1 second (FEV1), Peak Expiratory Flow(PEF), Forced Vital Capacity (FVC), Forced Expiratory Flow (FEF), Forced Expiratory Time (FET) and Flow/Volume Curves.

Enrolled students were divided into different groups with 8-10 students in a group. Each group were directed to appear at different dates in Pulmonary Function Test Laboratory at 7:30 AM and again at 5:00 PM for spirometry testing. Spirometry was performed with Spiro Excel 1.1 by trained technician between 7:30 to 8.00 AM in morning and 5:00-5:30 PM in evening. PFT was done as per the ATS guidelines[27] The test curve with the highest sum of the FVC and FEV1 were taken for further analysis.

Recorded data was scrutinized and any incomplete or inadequate test record was rejected. Finally, data from 169 subjects was found to be complete and appropriate and was taken for the analysis.

Statistical Analysis: Paired t-test was used to analyse and compare FEV1, FEF25, FEF50, FEF25-75, FEF75 and FVC values obtained from morning and evening tests of each student. Diurnal variation (dv) i.e. difference between morning and evening values of all parameters for each student were calculated as mean ± SD. The Diurnal variabilities of different parameters were compared using one-way analysis of variance. The statistical analysis was performed by Instat GraphPad Software. A p-value ≤0.05 was considered as significant.

Results

Out of enrolled 190 students,data of 169 students were analysed. Male (n=96) and female (n=73) ratio was 1.32:1. Mean age of all students was 24.48±3.12. Mean height and mean weight of students was 168.22±8.68 and 60.37±10.42 respectively.

Table 1: Anthropometric and Spirometrydata (Mean±SD) between male and female students measured at 7:30 AM

Basal Parameters	Males (n=96)	Females (n=73)
Age	24.68±3.20	24.10±3.28
Height	171.85±8.06	157.04±5.12
Weight	63.88±11.14	54.07±9.23
FEF25	7.08±1.12	6.46±1.31
FEF25-75	4.06±0.81	3.48±0.87
FEF50	4.47±0.92	3.75±0.80
FEF75	1.97±0.49	1.68±0.58
FEV1	3.84±0.41	3.11±0.33
FVC	4.49±0.48	3.56±0.29
FEV1%	85.06±4.97	86.57±5.37
PEF	9.15±1.11	8.21±0.96

Table 2: Spirometry parameters (Mean±SD) recorded in all students (n=169) in morning (7:30 AM) and in evening (5:00 PM) and their diurnal variability

Parameter	Morning Values	Evening Values	p value	Diurnal Variability	p value
FEF25	6.90±1.21	7.09±1.24	HS	7.83±6.23	S
FEF25-75	3.88±0.86	4.07±0.91	HS	9.57±9.64	S
FEF50	4.28±1.04	4.45±1.15	HS	10.75±11.31	S
FEF75	1.89±0.54	2.01±0.55	HS	13.15±11.92	S
FEV1	3.64±0.52	3.68±0.51	S	3.91±3.63	S
FEV1%	85.42±4.97	86.63±4.86	HS	3.25±2.90	S
FVC	4.29±0.77	4.32±0.76	NS	4.27±4.90	S
PEF	8.89±1.15	9.11±1.10	HS	6.42±5.78	S

HS- highly significant ($p<0.001$) S- significant- ($p<0.05$), NS- not significant ($p>0.05$)

All the spirometry parameters were significantly high in evening tests as compared to morning tests except FVC. Diurnal variability among different spirometry parameters was significantly different (ANOVA, $p<0.05$) in morning and evening tests. The diurnal variability was highest in large airways as reflected by FEF75 and Lowest in smaller airways as reflected by FEF25. The diurnal variability was lowest for FEV1%.

Discussion

Spirometry parameters had shown gender variation. All parameters were more in male than female. Showed a clear evidence that sex is a factor that affects PEF.[28]

Spirometry parameters exhibits circadian pattern andthey were less in morning compared to evening time. Diurnal variability may be seen due to variability in airway calibre during morning and evening time.[6,7,22,23]

Various studies have shown the diurnal variability of different spirometry parameters like Kondo S, Erban J et al. and Troyanov S et al. had demonstrated that spirometry parameters had significant difference during morning and evening time especially FEV1% and PEF and consistent with the results obtained from present study.[29-31]

Present study had made an attempt to differentiate the diurnal variability in spirometry test due to change in calibre of proximal and distal airway using PEF and FEV1. Present study results were consistent withthe study done by Hegewald MJ et al., who had exhibited that intrinsic variability in a single session (both morning and evening) spirometry test was higher for PEF than FEV1 also diurnal variability of PEF was higher than FEV1 in healthy subjects.[32]

Changes in proximal airway calibre results in changes in PEF while changes in FEV1 is related to calibre of proximal and peripheral airway.[24]

Studies have interpreted that the variability in proximal airways is largely due to changes in airway geometry. Fractional reduction in large airway calibre leads to greater decrease in flow compared to smaller airways. And it occurs due to a theory according to that flow rate or resistance is inversely proportional to the fourth power of radius. Also, it is a fact that the proximal and distal airways differ in smooth muscle content and nerve supply. The density of nerve supply and smooth muscle mass decreases as we proceed from proximal to distal airways.[33] This is why the diurnal variability in smaller airways is lower than larger airways.

Correlation between PEF and FEV1 and their diurnal variability was significant. This feature was representation of changes in proximal airways calibre corresponding to changes in distal airways calibre. Morning and evening mean of both PEF and FEV1 were significantly different and showed diurnal variability. Previous study also supported results of this study.[2,6,23]

FEV1 was clinically more suitable to know the diurnal variability because total variability was lowest and maximum variability seen was less than 10%. Clinical use of Mid Expiratory Flow was not justified because it showed high variability. FEV1 and PEF showed variability according to the previous study.

Conclusion

FEV1, FEF and PEF has shown diurnal variability which was directly related to the airway calibre. Proximal airways showed greater diurnal variation than distal airways due to in their calibre, reflected by greater variability in PEF as compared to FEV1. In this study only two readings were taken to investigate the diurnal variability. Further study with multiple recordings in 24-hour duration should be tried to better characterize the circadian pattern of spirometry parameters and exploring their physiological basis.

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Original Research Article

Comparison of cardiovascular, cognitive and stress parameters in presence and in absence of examination among medical students: An observational and prospective study

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ABSTRACT

Introduction: Medical student during undergraduate course of 4 $\frac{1}{2}$ years including 1-year internship study hard, tirelessly for longer periods of day or night and often work beyond their mental threshold and physical strength resulting in stress. The present study was designed to evaluate and compare the stress status among first and second year MBBS students.

Materials and Methods: 160 (62 males and 98 females) first (n=90) and second (n=70) year healthy MBBS students were enrolled for this study. Cardiovascular parameters like pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), Cognitive function tests like auditory reaction time (ART) and visual reaction time (VRT) and Stress score (by stress questionnaire) was evaluated and compared in presence of examination (pre-examination) and during absence examination (post-examination, 10-15 days after pre-examination). Data obtained from this study was analysed by Instat Graph Pad using paired t-test.

Results: All parameters studied in this study were increased in almost all students during pre-examination. In females compared to males all parameters were significantly less in pre-examination except PR (i.e. less SBP, DBP, less cognition function that means high ART & VRT and less stress score). During post-examination study comparison between males and females, difference of means of parameters were not significant except VRT (Visual reaction time was high in females). Cognition function was less in females as compared to males in both pre as well post examination.

Conclusion: Students were in stress with increased all cardiovascular parameters, cognitive parameters and stress score. This may affect the performance and can produce anxiety and/or depression subsequently. Students who are at risk of excessive stress should be identified and faculties should help them to deal the examination stress, anxiety or depression effectively and the earliest.

1. Introduction

Medical curriculum is a vast and complex study course with training of four and half year with one year of internship. This long period of course includes heavy load of text books and study materials, different ward or clinical postings and numerous semester and university examinations. To achieve good grades student study hard, tirelessly for longer periods of day or night and often work beyond their mental threshold and physical strength resulting in stress. Stress refers to conditions that arouse anxiety or fear. The transient rise in systolic blood pressure during stress is a common observation.[1–5]

Several studies have shown correlation between chronic life stress and cardiovascular disease.[6] Psychological stress is a risk factor for hypertension[7] and coronary artery disease (CAD).[6] Different physiological studies have proved that stress is linked with excessive sympathetic nervous system activation[6] and thus influence the endocrine, haemopoietic

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and immune systems.[8] Cytokines and cortisol seem to play an important role in the communication between these systems.[9] The well documented changes that occur are increase in erythrocytes, neutrophils and platelets, whereas lymphocytes, eosinophils and monocytes decrease in number. Lymphocytes and monocytes express receptors for several stress hormones, including norepinephrine and epinephrine,[10] thus stressful events could alter immune function.

It has also been observed that female students respond to examination situation with stronger anxiety and more intense stress related behavioural, metabolic and psychological changes. Menstrual cycles of females also seem to get affected during the pre-examination period owing to hormonal changes as observed in previous studies.[11]

Cardiac parameters like pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), Cognitive function tests like auditory reaction time (ART) and visual reaction time (VRT), Stress score (by stress questionnaire), anxiety scale and cortisol level were evaluated and compared before or at the time of examination among medical students in various studies.[12–15]

Some studies have compared the difference of reaction time in male and females and thus evaluated stress (e.g., environmental).[16,17]

Different factors may influence severity of stress on academic performance like age, gender, ethnicity and marital status.[18]

Anxiety may be potentiated by increase in glucocorticoid which directly effects on corticotrophin releasing hormone in limbic system.[19,20]

Distraction model (attentional control theory) is one of the models, developed to show effect of stress on cognitive function. In this model performance of movement execution (e.g., about the location of a target) become less accurate and more attempts or more time may be required to successfully perform a certain task and this occurs when person is under anxiety.[21,22] However, "execution focus model" argues that limited attentional resources cannot explain the negative effects of anxiety upon performance.[23]

The alteration of reaction time occurs due to both physiological and pharmacological factors like stress, gender, and arousal. This alteration indicates the impairment of sensory-motor association.[24]

In this study Cardiovascular parameters like pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), Cognitive function tests like auditory reaction time (ART) and visual reaction time (VRT) and Stress Score through questionnaire was evaluated and compared in presence and during absence of examination among first and second year MBBS students.

2. Materials and Methods

2.1. Study site/place

This study was conducted in the Department of Physiology and Pharmacology of Katihar Medical College, Katihar.

2.2. Study duration

January to July 2018 (Seven months)

2.3. Study design

An observational and prospective study

2.4. Inclusion criteria

1) First and second year MBBS students
2) $\geq$ 17 years of age and all the gender
3) Healthy students

2.5. Exclusion criteria

Students have history of neurological or psychiatric disorders, taking of medicines affecting emotional status and endocrinological disorder, any visual and auditory disorder, addiction to tobacco or alcohol

160 students were selected from first and second year MBBS batch before internal assessment theory examination considering after inclusion and exclusion criteria. 80 students were from first year and 70 students were from second year MBBS batch. Following tests were done.

2.5.1. Cardiovascular parameters

Tests like PR (Pulse Rate - beats/min) and BP (Blood Pressure- mm of Hg) were recorded in supine position by palpating radial artery and sphygmomanometer respectively.

2.5.2. Cognitive parameters

Test like ART (Auditory Reaction Time, in milliseconds) and VRT (Visual Reaction Time, in milliseconds) were recorded by using Audio Visual Reaction Time Machine, in a well illuminated and quiet surrounding in Physiology research laboratory. This instrument had two modes one for Auditory and another for Visual reaction time. It had three frequencies i.e. 250Hz, 500Hz and 750Hz which were randomly used for auditory stimulus. Red, Yellow and Green flashing lights were used randomly for visual stimulus. Students were directed to press the response switch by the index finger of the dominant hand as soon as the response would be perceived. The reaction time was displayed on the Reaction Time Machine and was recorded.

2.5.3. Stress status

Stress status was assessed by a questionnaire. Which contained 20 questions with 0-4 points given to each

(i.e. no stress (Score=0) to extremely stressful (Score=4). Questionnaire was given to the students and collected after 10min to assess stress score. The total score obtained from this questionnaire was analysed.[14,25,26] According to scores given by student, stress status was interpreted like

a. Score between 0-20: - Good control over stress,
b. Score between 21- 40: - Low level of stress,
c. Score between 41- 60: - Medium level of stress,
d. Score between 60-80: - High level of stress.

On following areas of stress producing scenario, questions were framed like

1. Academic demands,
2. Peer pressure,
3. Lack of time for personal needs,
4. Interpersonal relationships including those with teaching and administrative staff.
5. Inability to sleep well,
6. Worrying,
7. Feeling tense and
8. Unhappy.

Anthropometric measurements like weight in kilograms and height in centimetres were assessed using standardised weighing machine and height measurement scale.

Studies were done for twice in following manner in all enrolled students.

2.6. Pre-examination study

All enrolled students were instructed to appear in Physiology Research Laboratory 1.15 hours prior starting the final internal examination without consuming any kind of caffeinated drinks like coffee or tea. Before starting the experimental session, students were given rest of 15 minutes. Experimental sessions were completed 10 mins before starting the theory examination.

2.7. Post-examination study

10 to 15 days after completion of final internal assessment theory examination i.e. when students were practically free of examinations, once again all students were instructed to appear in Physiology Research Laboratory on different day. They were strictly instructed to appear without consuming any kind of caffeinated drinks like coffee or tea. Before starting the experimental session, students were given rest of 15 minutes.

In 1^{st} year students, pre-examination study was performed on 3 0 students each on 1^{st}, 2^{nd} and 3^{rd} day (According to the three-subject examination in first year). In 2^{nd} year students, pre-examination study was performed on 14 students each on 1^{st}, 2^{nd}, 3^{rd}, 4^{th} and 5^{th} day (According to the five-subject examination in second year)

2.8. Statistical analysis

Data obtained from this study was analysed by Instat Graph Pad. The pre and post -examination data was analysed using paired t-test. Results were tabulated and presented as Mean+SD.

3. Results

All parameters like PR, SBP, DBP, VRT and Stress score were increased (extremely significant) in pre-examination as compared to post- examination study.

Mean PR in female students was significantly more than male students. Mean SBP and DBP in females were significantly less than males but the mean difference SBP was significant and DBP was not significant. Mean ART and VRT, both were significantly high (reaction time high) in females as compared to males. Mean stress score was also significantly less in females compared to males.

During post-examination study, mean difference of PR, SBP, DBP and stress score was not significant in males and females. Compared to males, mean ART (difference not significant) and VRT (very significant) were more in females.

Delta PR (calculated by subtracting pre and post-examination study value) was increased in females as compared to males irrespective of the study setting and the difference was extremely significant. Mean difference of delta SBP, DBP, ART, VRT and Stress score were not significant between males and females.

4. Discussion

In pre-examination compared to post-examination study period irrespective of gender all parameters like PR, SBP, DBP ART, VRT and stress scores were increased significantly. Most common cause may be due to increase in sympathetic stimulation that increases PR and BP (both systolic and diastolic blood pressure). ART and VRT may be increased due to release of epinephrine and glucocorticoid.[26,27] Under stressful conditions, the cognitive system becomes overloaded thus reduces a person's attentional resources.[28]

Due to increased sympathetic nervous system and brain-pituitary-adrenocortical axis during stress acting either directly or indirectly can alter decision making and attention. In this study pre-examination compared to post-examination, stress score was increased significantly. This has similar result from previous study in which stress was common among first year medical students due to academic demands.[2,3,29]

In this study PR, ART and VRT we re increased in females as compared to males in pre-examination, but difference in PR was more significant. These findings were similar with other study.[12,30]

Table 1: Comparison of cardiovascular parameters, cognitive parameters and stress score in pre-examination and post-examination study

Parameters	Pre-examination (n=160) (mean±SD)	Post-examination (n=160) (mean±SD)	p-value
PR (beats/min)	88.21±12.26	78.17±12.47	0.0001***
SBP (mmHg)	129.20±11.35	122.70±10.55	0.0001***
DBP (mmHg)	86.70±7.36	80.86±5.26	0.0001***
ART (ms)	180.89±27.69	167.74±29.60	0.0001***
VRT (ms)	211.49±25.67	190.56±31.08	0.0001***
Stress score	28.34±9.28	21.38±5.64	0.0001***

*p<0.05-Significant; *p<0.01-Significant, **p<0.001-Very Significant, ***p<0.0001- Extremely Significant, p>0.05- Not Significant (NS)

Table 2: Comparison of cardiovascular parameters, cognitive parameters and stress score in pre-examination study on the basis of gender

Parameters	Male (n=62) (mean±SD)	Female (n =98) (mean±SD)	p-value
PR (beats/min)	84.09±10.28	92.38±13.16	0.0001***
SBP (mmHg)	130.48±10.56	125.56±12.88	0.013*
DBP (mmHg)	85.62±7.06	84.5 6±6.8 8	0.349, NS
ART (ms)	174.8 9±24.72	185.96±29.27	0.015*
VRT (ms)	205.9 2±19.58	217.66±29.49	0.006*
Stress score	30.1 5±8.84	26.7 9±9.25	0.024*

Table 3: Comparisons of cardiovascular parameters, cognitive parameters and stress scores in post- examination study on the basis of gender

Parameters	Male (n =62) (mean±SD)	Female(n=98) (mean±SD)	p-value
PR (beats/min)	82.26±13.98	78.5 2± 9.86	0.98, NS
SBP (mmHg)	120.36±10.76	120.20±10.35	0.93, NS
DBP (mmHg)	77.42±5.63	77.94±6.08	0.59, NS
ART (ms)	163.2 5±28.76	171.68±29.55	0.08, NS
VRT (ms)	183.96±27.86	199.52±32.60	0.002**
Stress score	21.56±5.57	20.62±5.72	0.31, NS

Table 4: Changes of cardiovascular parameters, cognitive parameters and stress scores based on gender

Parameters	Male (n =62) (mean±SD)	Female (n =98) (mean±SD)	p-value
Delta PR (beats/min)	-1.83±16.97	-13.86±17.46	0.0001***
Delta SBP (mmHg)	-10.1 2±1 4.88	-5.36±15.09	0.052, NS
Delta DBP (mmHg)	-8.2±14.98	-6.62±9.01	0.406, NS
Delta ART (ms)	-11.6 4±3 7.56	-14.28±41.33	0.684, NS
Delta VRT (ms)	-21.96±34.78	-18.14±44.05	0.564, NS
Delta Stress score	-8.59±8.37	-6.17±8.16	0.072, NS

Similar to a previous study, Stress score through stress questionnaire was also significantly more in females compared to males.[31] But in pre-examination of this study, stress scores were significantly increased in males also. Difference of stress levels between males and females were not significant reported by a study.[32] PR, SBP, DBP and stress scores in males and females in post-examination study was not significantly different.

Cognitive function was decreased (i.e. both audio and visual reaction time were increased) in females as compared to males in both pre and post-examination. Increase in VRT in females may be explained by change in steroid hormone during menstrual cycle. Cognitive function of female brain is under control of ovarian steroid and this ovarian steroid has widespread effects throughout the brain regions.[33,34]

Difference of studied parameters were not significantly different between both the genders except delta PR, which was significantly higher in females. Hypothalamic-pituitary-axis and autonomic nervous system activity may be increased in females due to examination stress. This could be the reason behind the increase in PR. In this study females were more distressed compared to male and this result was supported by other studies too.[35]

Limitations of this study was that we measured stress by questionnaire and not studied psychological factors that may influence the stress response, stress scores were obtained at only one point of time, other sources of stress such as familial or interpersonal pfroblems were not examined

and internal assessment scores of the students were not correlated in this study with pre-examination stress level.

5. Conclusion

Cardiovascular parameters, cognitive parameters and stress scores were increased in almost all of the students irrespective of gender in pre-examination study. This may negatively affect the performance of students and can produce anxiety and/or depression subsequently. Students who are at risk of excessive stress should be identified and faculties should help them to deal the examination stress, anxiety or depression effectively and the earliest.

6. Source of funding

None.

7. Conflict of interest

None.

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Original Research Article

Study on Relationship between Foetus, Neonatal & Maternal Haemoglobin Level

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Abstract
Objectives: *Our study was to find out the relationship between foetus, neonatal and maternal haemoglobin level of non-anaemic and anaemic mothers and their new borns.*
Methodology: *A total of 100 full term pregnant women and their full term newborn were enrolled in this study. A detail history, dietary pattern, clinical examination and relevant investigation were performed. Maternal blood and cord blood of newborns were examined. Haemoglobin level was estimated by Sahli's acid haematin method.*
Results: *Data was analyzed by using SPSS software. Mean, standard deviation and t value were observed. P value was taken ≤ 0.05 for significant differences.*
Conclusions: *Our study concluded that if the mother suffers from anemia, i.e. low hemoglobin level, the baby born to will also have low cord hemoglobin. Maternal anemia has a definite bearing on neonatal hemoglobin level.*
Keywords: *Anaemia, Pregnancy, Maternal blood, cord blood, Haemoglobin.*

Introduction

Anaemia is a common medical problem in pregnancy. The extent up to which maternal anaemia affects maternal and neonatal health is still uncertain[1]. India has reported high prevalence of anaemia in pregnancy. In one of the studies conducted on a large population, it was estimated that 87% of the Indian population of the Indian women are anemia[2].

Anemia is defined as the most common hematological disorder during pregnancy having decreased hemoglobin level or circulating red blood cells[3]. The World Health Organization (WHO) has estimated that prevalence of anemia in pregnant women was found 14% in developed, 51% in developing countries and 65-75% in India. Prevalence of anemia in all the groups is higher in India as compared to other developing countries. WHO recommends that hemoglobin ideally should be maintained at or above 11.0 g/dl in the second trimester[4].

Hematology of newborn recently represented as area of study that focusing in study of umbilical cord blood and its elements in general.[5] Umbilical cord blood count at birth shows that there is an increased in hemoglobin, hematocrit, mean corpuscular volume, leukocyte count, reticulocyte count and nucleated red blood cells with presence of occasional immature white blood cells or left-shifted in peripheral blood of healthy infants, with variable degree in immature sick newborns.[6] The mean cord hemoglobin value varies approximately between 16.6 and 17.1 gm/dl of blood.[7] The average hematocrit level approximately 0.55 L/L (55%) at birth.[8] The total white blood cell count at birth generally high in ranges between 9 and 30 x 109 / liter.[9]

Reticulocyte number at birth about 4% to 6% and reflected the activity of the red cell formation in fetal life.[10] Variable number of platelets during neonatal period was reported; figure reported at time of birth ranges from 150 x 109/liter to 350 x 109/liter.[11] Intrauterine fetus is maternal dependent from embryonic stage, fetal hood up to birth; hence anemia during pregnancy play a major role in causes of fatal life threatening to the mother and her fetus, and considered to make serious complications resulting from lower oxygen delivery, elevation of erythropoietin level, reticulocyte counts, and nucleated red blood cells of valuable inspections of neonatal healthy status.[12] Hence Increased erythropoietin level of cord blood at time of birth used as indicator markers for fetal hypoxia.[13]

Maternal and child health is an important problem of public health, influencing the development of the family and the community. Mother and infant protection is a priority in the health field because these population groups are the most exposed to the sickness and death, consequently to their low reactivity to the environmental factors and to their high responsiveness to the disorders[14].

Objectives of our study was to assess and determine the maternal haemoglobin (Hb) level on pregnancy outcome and to find out the association between maternal Hb level and its effect on neonates.

Materials and Methods

The present study was carried out on 100 full term pregnant women and their full term newborn admitted in the department of Obstetrics and Gynaecology, Katihar Medical College & Hospital, Katihar, Bihar, India during a period from January 2016 to December 2017. The attendants of entire subjects signed an inform consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar was sought. The relevant investigations were carried out in the department of Physiology and upgraded department of Obstetrics and Gynaecology, Katihar Medical College, Katihar, Bihar.

Pregnant mothers without complications and their single born term normal neonates delivered spontaneously by vaginal route were considered for this study. Detail history of the mothers was noted and complete physical examination of them and their newborns was carried out.

Determination of hemoglobin level, P.C.V and total W.B.C count of mothers at the time of onset of labour was carried out. Same investigations were carried out on the cord blood of the newborns of these mothers.

Pregnant women with hemoglobin level of more than 12.0 gm/dl constituted the normal non-anaemic control group or group I. Pregnant women with hemoglobin level of less than 12.0 gm/dl were further divided into three anemic groups or case group (group II, III & IV). Group II: included pregnant women were with hemoglobin level of 9.1 to 12.0 gm/dl. Group III: included pregnant women with hemoglobin level of 6.1 to 9.0 gm/dl. Group IV: included pregnant women with hemoglobin level of less than 6.0 gm/dl. On the same pattern babies were assigned to their respective mother groups. A total of 100 pregnant women were enrolled in this study. Among 100 pregnant women, 25 pregnant non-anaemic women were in group I, 30 pregnant

anaemic women were in group II, 30 pregnant anaemic women were in group III and 15 pregnant anaemic women were in group IV.

Methods

Detail assessment and clinical examination were performed to all case like identification: name, address, age, religion, occupation, gravida, parity, Presenting complaints, menstrual history, obstetrical history, history of past illness, family history, personal history, socioeconomic status.

General examination: Appearance, built, state of nutrition, height and weight, pallor, cyanosis, jaundice, pulse, respiration, temperature, blood pressure, tongue, gums, clubbing, koilonychias, thyroid, lymphodenopathy, any other specific nutrition deficiency sign.

Systemic examination: Abdominal and pelvic examination: size of uterus interms of weeks, uterine contraction, lie and presentation of foetus, foetal heart sound. mothers cardiovascular system, respiratory system, digestive system and central nervous system.

Examination of newborn: Apgar scoring, weight, length, skin, heart, chest, abdomen, cord, rectum and genitilia, reflexes, circumference of head and circumference of chest.

Blood samples: Mothers venous blood and the cord blood of respective newborns were collected. Mother's blood was collected from antecubital vein 2-48 hours before delivery. Newborns blood was collected from umbilical cord just after delivery, when cord pulsation has ceased but before the placental separation.

Investigation done on maternal and cord blood: Estimation of Haemoglobin level, total R.B.C count, P.C.V. estimation, calculation of M.C.H and M.C.H.C.

Estimation of Haemoglobin

Haemoglobin estimation of both maternal and cord blood was done by Sahli's acid haematin method.

Principle: Anticoagulated blood is added to the 0.1 N HCl and kept for 5-7 minutes to form acid haematin. The color of this acid haematin should be matched with the solution, present in the calibration tube. Distilled water is added to the acid haematin until the color matches and the final reading is directly noted from the graduation in the calibration tube. [Please note that 100 percent on the scale corresponds to 14.5gm % to 15gm %].

Requirements: Sahli's haemoglobinometer, Hydrochloric acid, distilled water.

Procedure: Place N/10 HCL in diluting tube up to the mark 20. Take blood in the haemoglobin pipette up to 20-cubic-mm-mark and blow it into diluting tube and rinse well. After 10 minutes add distilled water in drops and mix the tube until it has exactly the same color as the comparison standards. Note the reading, which indicates the percentage of haemoglobin.

Precautions: i. Pipetting of blood should be done cautiously. ii. Mix the blood properly with HCl by using stirrer. iii. Match the color cautiously.

Determination of total R.B.C count:

Principles: A known volume of blood is diluted (200 times) with an isotonic solution containing anticoagulant. R.B.C. in a known volume of blood are counted in special counting chamber and number of R.B.C. per cu mm of blood is calculated there from.

Materials and Instruments

a) Whole blood, using EDTA or heparin as an anticoagulant. Using capillary blood.
b) Hayem's solution:Hgcl2 0.05g, NaSO4 2.5g, NaCl 0.5g and distilled water 100ml.
c) RBC pipette
d) Hemocytometer (Neubauer's counting chamber) with coverslip.
e) Microscope.
f) Lancet.
g) Alcohol 70%.
h) Pipette rotator
i. Aspirator connected to a faucet with running water.

Procedure

a) Wipe finger with cotton soaked with alcohol, with a sterile lancet do small prick

on the finger tip. Use pipette. Aspirate blood to 0.5.

b) Aspirate diluting Hayem's solution to the 101 mark.
c) Hold the pipette horizontally and role it with both hands between finger and thumb.
d) Touch the tip of the pipette on the surface of the counting chamber 45 degree.
e) Place the chamber on the stage of the microscope and allow 2 minutes for the cell to settle.
f) Scan the counting area with 10x objective lens.
g) Use 45x objective, include all cells lying on the upper and left lines of any sequare, omit the cells on the lower and right- hand lines.
h) Count the cells in five groups of 16 small sequares i.e 80 small sequares.

Calculation: The total number of red cells/c.mm=NX 10 000, where N is the number of red cells found in 80 squares.

Determination of packed cell volume

Principle: packed cell volume is determined by centrifuging a sample of blood made uncoagulable by a suitable anticoagulant.

Apparatus and reagent:

1. Wintrobe's haematocrit tube.
2. Anticoagulant mixture (potassium oxalate + ammonium oxalate)

Procedure: 2 ml of blood was added in a test tube containing anticoagulant mixture. This was thoroughly mixed by gently shaping and rotating the test tube. This blood was drawn in the Wintrobe's haematocrit tube up to zero mark. It was centrifused at the speed of 3000 r.p.m for a period of one hour. Then direct reading was taken of the upper limit of packed cells. This used the packed cell volume as percentage, of the original blood.

Calculation of Mean Corpuscular Haemoglobin

Mean Corpuscular Haemoglobin (M.C.H) is the average amount of haemoglobin contained in a single R.B.C. and is expressed in micro-microgram.

M.C.H = Hb in gm/100 ml of blood ÷R.B.C. in million/cu. mm of blood x 10

Normal range of M.C.H IS 24-33 micro-microgram.

Calculation of Mean Corpuscular Haemoglobin Concentration

Mean corpuscular haemoglobin concentration (M.C.H.C) is the percentage saturation of red cell with haemoglobin.

M.C.H.C. = Hb in gm/100 ml of blood ÷ P.C.V. 100/ml of blood x 100.

Normal range of M.C.H.C is 30-36 %.

Statistical Analysis

Date was analyzed by using the SPPSS software. Mean, standard deviation, t value were calculated. P value was taken less than 0.05 for significant differences.

Observation

A total of 100 full term pregnant women and their full term newborn (25: non-anaemic, 30: mild anaemic, 30: moderate anaemic and 15: severe anaemic) were enrolled in this study.

Table.1. Distribution of cases according to maternal haemoglobin level in gm percent

Group		Maternal Hb in gm percent	No. of cases
Non-anaemic	Group I (Control)	12.1 – 14.8	25
Anaemic	Group II	9.1 – 12.0	30
	Group III	6.1 – 9.0	30
	Group IV	3.4 – 6.0	15

Table.2 Showing mean value and standard deviation of maternal Hb in control group and case group mothers.

Mothers	Hb in gm percentage	
Group I (Control)	Mean	Standard deviation
	13.28	0.74
Group II (Anaemic)	9.95	0.91
Group III	7.35	0.85
Group IV	4.85	0.81

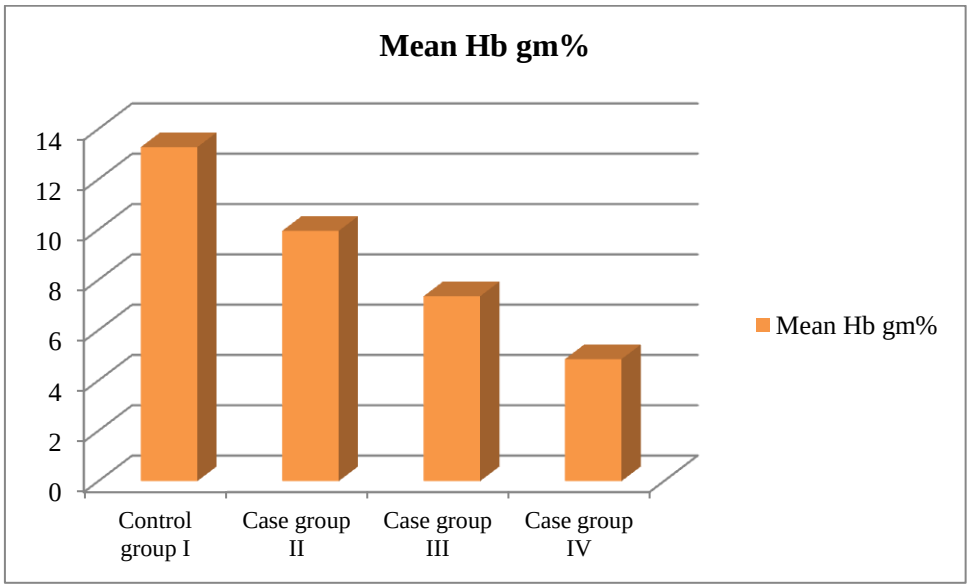

Figure.1. Mean value of maternal Hb of non-anaemic (control group) and anaemic (case group) mothers.

Table.3 Comparison of mothers Hb level of control group (non-anaemic) with case group(anaemic).

Group	t-value	Df	P value	Conclusion
Group I with Group II	11.17	53	< 0.001	Significant
Group I with Group III	21.81	47	< 0.001	Significant
Group I with Group IV	24.97	27	< 0.001	Significant

When group I was compared with group II, group III and group IV, p value was found to be less than 0.05. That was highly significant.

Table.4. Showing maternal R.B.C. count in millions/cu.mm, mean value and standard deviation of control and case group.

Group	R.B.C. in million/cu.mm.		
	Range	Mean	S.D
Group I	3.4 - 4.5	3.98	0.27
Group II	3.1 - 4.2	3.75	0.29
Group III	2.8 – 4.2	3.51	0.35
Group IV	2.0 – 3.4	2.72	0.34

Table.5 Comparison of R.B.C. count of group I with group II, group III and group IV

Group	t-value	df	P-value	Conclusion
R.B.C. count of group I with group II	2.47	56	< 0.01	Significant
R.B.C. count of group I with group III	4.31	47	< 0.001	Highly significant
R.B.C. count of group I with group IV	7.17	23	< 0.001	Highly significant

Above table shows statistically significant fall in R.B.C. count in different groups of anemic mothers as compared with those of control group.

Table.6. Mean value and standard deviation of maternal P.C.V of mothers of control group and case group.

Mothers	Packed cell volume in ml. percent		
	Range	Mean	S.D
Group I	38-48	42.76	2.47
Group II	30-43	35.40	3.71
Group III	24-42	33.26	4.43
Group IV	17-29	23.92	3.67

Table.7 Comparison of P.C.V. of normal mothers of group I with anemic mothers of case group II, III and IV

Mothers	t-value	df	p-value	Conclusion
Group I with Group II	7.59	61	< 0.001	Highly significant
Group I with Group III	23.76	49	< 0.001	Highly significant
Group I with Group IV	32.91	31	< 0.001	Highly significant

Above table shows that there was statistically significant fall in P.C.V. values in different groups of anemic mothers as compared with those of control group.

Table. 8. Mean value and standard deviation of maternal M.C.H in micro micrograms of mothers of control and case group.

Mothers	Mean Corpuscular Hb in micro micrograms		
	Range	Mean	S.D
Control group I	31.70 – 35.52	33.46	1.05
Case group II	23.33 – 29.37	26.66	0.17
Case group III	18.00 – 25.31	21.60	1.62
Case group IV	15.65 – 22.30	17.69	1.00

Above table shows significant fall in M.C.H. values in different group of anemic mothers as compared with those of control group non-anemic mother.

Table. 9. Ranges, mean values and standard deviation of maternal M.C.H.C. values of mothers of control group and case group.

Mothers	M.C.H.C, in percent		
	Range	Mean	S.D.
Control group I	29.54 – 33.09	31.11	0.80
Case group II	25.55 – 32.33	28.32	1.88
Case group III	19.41 – 27.34	22.98	2.06
Case group IV	17.30 – 22.50	20.35	1.49

Above table shows that there was significant fall in M.C.H.C. values in different groups anemic mothers (case group) as compared with those of mothers of control group.

Table. 10. Range, mean and standard deviation of cord blood haemoglobin level of newborns of mother of control group (non-anemic mothers) and case group (anemic mothers).

Mothers			
	Range	Mean	S.D.
Control group I	13.5 – 19.3	15.96	1.79
Case group II	11.5 – 17.3	13.9	1.63
Case group III	11.5 – 16.1	13.44	1.25
Case group IV	13.4 – 18.3	14.28	0.57

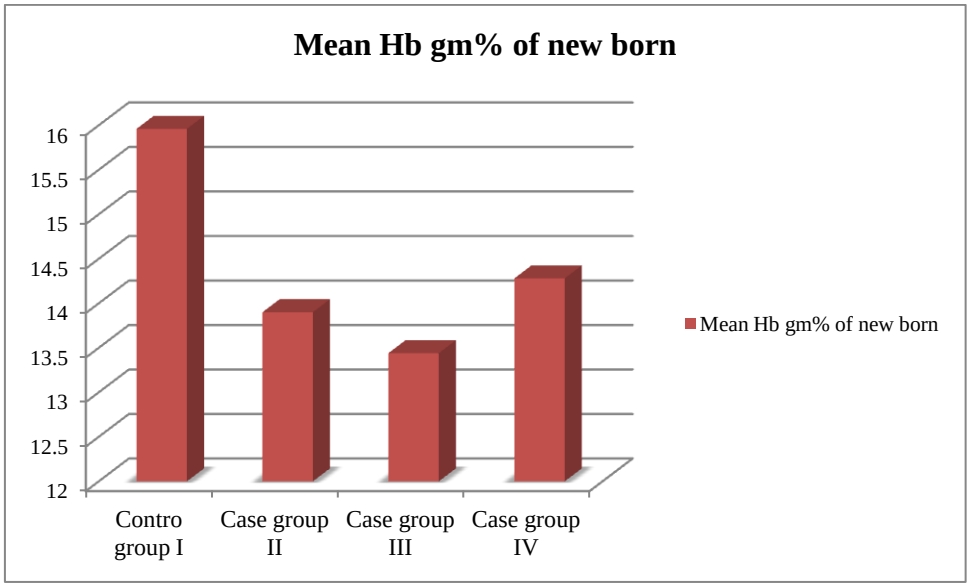

Figure.2. Cord Hb level of newborn of mothers of control group and case group

Table.11. Comparison of cord blood Hb of newborns of control group with case group.

Group	t-value	df	P value	Conclusion
Group I with Group II	4.08	59	< 0.001	Highly significant
Group I with Group III	4.13	43	< 0.001	Highly significant
Group I with Group IV	3.94	27	< 0.001	Highly significant
Group II with Group III	0.47	71	> 0.05	No significant
Group II with Group IV	0.91	49	> 0.05	No significant
Group III with Group IV	0.73	39	> 0.05	No significant

Above table shows that when cord blood Hb of control group was compared with case group, it was highly significant. But, when intra group analysis was done in case group (group II), it was not significant differences.

Table.12. Range, mean and standard deviation of cord blood R.B.C. count in millions/cu.mm of newborns of mother of control group and case group.

Newborns	R.B.C. count in million/cu.mm.		
	Range	Mean	S.D
Control group I	4.0 – 6.2	5.1	0.63
Case group II	3.6 – 5.3	4.52	0.48
Case group III	3.4 – 4.6	4.03	0.29
Case group IV	3.8 – 4.7	4.15	0.29

Table.13 Comparison of cord blood R.B.C. count of newborn of control group with case group.

Group	t- value	df	p- value	Conclusion
Group I with Group II	2.71	59	< 0.01	Significant
Group I with Group III	3.57	47	< 0.001	Highly significant
Group I with Group IV	3.93	26	< 0.001	Highly significant
Group II with Group III	1.02	71	> 0.05	Not significant
Group II with Group IV	2.29	49	> 0.05	Not significant
Group III with Group IV	0.51	39	> 0.05	Not significant

Above table shows that when cord blood R.B.C. of newborn of control group was compared with case group, it was found to be highly significant. But when intra group comparison of cord blood R.B.C. of newborn of mothers of case group, it was not significant differences.

Table. 14. Range, mean and standard deviation of cord blood P.C.V. value of newborn of mothers of control group and case group.

Group	Packed cell volume in ml. percent		
	Range	Mean	S.D.
Control group I	43 - 61	51.28	4.99
Case group II	38 - 56	46.76	4.98
Case group III	39 - 48	43.33	2.67
Case group IV	39 - 49	43.66	3.33

Table.15. Comparison of P.C.V. of newborn of mothers of Control group with Case group.

Group	t-value	df	p-value	Conclusion
Group I with Group II	6.317	59	< 0.001	Highly significant
Group I with Group III	9.326	50	< 0.001	Highly significant
Group I with Group IV	8.416	31	< 0.001	Highly significant
Group II with Group III	3.81	69	< 0.01	Significant
Group II with Group IV	4.03	47	< 0.01	Significant
Group III with Group IV	1.61	29	> 0.801	Not significant

Above table shows that when cord blood P.C.V. value in newborn of control group was compared with case group, differences was highly significant. When intra group comparison of case group II with case group III and case group IV, difference was significant. But intra group comparison of case group III with case group IV, there was found to be not significant differences.

Table.16. Range, mean and standard deviation of M.C.H. value of cord blood of newborn of mothers of control group and case group

Group	M.C.H. in micro micrograms		
	Range	Mean	S.D.
Control group I	27.73 – 34.28	31.73	1.53
Case group II	28.77 – 33.80	30.71	1.36
Case group III	27.04 – 37.56	34.07	2.14
Case group IV	31.86 – 39.23	34.31	2.13

Above table shows that M.C.H. values of cord blood of newborns of control group and different group of case group do not fall in spite of fall in maternal hemoglobin level.

Table.17. Range, mean and standard deviation of M.C.H.C. values of cord blood of newborns of control group and case group

Group	M.C.H.C. in percent		
	Range	Mean	S.D.
Control group I	28.43 – 32.79	30.98	1.005
Case group II	26.72 – 33.00	29.73	1.09
Case group III	29.31 – 35.34	31.58	1.4
Case group IV	30.68 – 36.41	32.72	1.09

Above table shows that M.C.H.C. level in cord blood of newborns of case group was not fall in spite of fall in maternal hemoglobin level.

Discussion

There is a significant association between maternal Hb level and pregnancy outcome like type of delivery, birth weight. Study conducted on risk for preterm delivery and low birth weights are independently increased severity of maternal anaemia.

This study compromised of 100 pregnant women from different socioeconomic strata with varying nutrition status. Out of 100 pregnant women 25 served as normal non-anemic control group. This group having hemoglobin level above 12.0 gm/dl with a mean value of 14.03 gm/dl was labeled as non-anemic control group I.

Remaining 75 pregnant women with hemoglobin level bellow 12.0 gm/dl were designated as anemic case group. This case group was further divided into three groups. Group II: Included 30 pregnant women with hemoglobin ranges from 9.1 gm/dl to 12.0 gm/dl. Group III: included 30 pregnant women with hemoglobin ranges from 6.1 gm/dl to 9.0 gm/dl. Group IV: included 15 pregnant women with hemoglobin level of 6.0 gm/dl and bellow (table 1).

This arbitrary grouping of the degree of maternal anemic on the basis of their hemoglobin content was done in accordance with the line adopted Goswami et al (2014).[15]

The anemic pregnant mothers (case group) with hemoglobin bellow 12 gm/dl were suffering from iron deficiency anemia by the presence of low hemoglobin level, low R.B.C. count, decreased P.C.V., M.C.H. and M.C.H.C. values.

The mean hemoglobin level of Group I mothers serving as control was 13.28 gm/dl, with a range of 12.1-14.0 gm/dl, standard deviation ± 0.74. The mean hemoglobin level of group II mothers was 9.96 gm/dl with a range of 9.1-12.0 gm/dl, standard deviation being ± 0.91. This value for group III and group IV mothers was 7.35 gm/dl (ranges 6.1-9.0 gm/dl, S.D. ± 0.85) and 4.85 gm/dl (range 3.4-6.0 gm/dl, S.D. ± 0.81) respectively.

The R.B.C. count of control group I mothers ranges from 3.4 - 4.5 millions/cu.mm. with a mean value of 3.98 millions/cu.mm., S.D.± 0.27. in case group II mothers R.B.C. count was ranging from 3.1 - 4.2 million/cu.mm with a mean value of 3.75 million/cu.mm., S.D. ± 0.29. In group III mothers the R.B.C. count ranges from 2.8 – 4.2 million/cu.mm with a mean value of 3.5 million/cu.mm., S.D. ± 0.35. The R.B.C. count of case group IV mothers ranged from 2.1 – 3.4 million/cu.mm with a mean value of 2.72 millions/cu.mm., S.D. ± 0.34.

The P.C.V. of control group I ranged from 38 – 48% with a mean value of 42.76, S.D. ± 2.47. While that of group II mothers ranged from 30-43 % with a mean value of 35.40 %, S.D. ± 3.71. In group III mothers the ranged of P.C.V. values was 24 – 42 % with a mean value of 33.26%, S.D. ± 4.43 and in group IV mothers this values was ranging 17 – 29% with a mean value of 23.92%, S.D. ± 3.67.

The maternal M.C.H. value for control group I was 31.7 – 35.52 micro microgram with a mean of 33.46 micro microgram, S.D. ±1.05. The M.C.H. level of group II mothers ranged from 23.33 – 29.37 micro microgram with a mean value of 26.66 micro microgram, S.D. ± 0.71. In group III mothers the M.C.H. value was 18.00 – 25.31 micro microgram with a mean value of 21.60 micro microgram, S.D. ± 1.62. In group IV mothers the M.C.H. values was 15.65 – 22.30 micro microgram with a mean value of 17.69 micro microgram, S.D. ± 1.80.

The M.C.H.C. of control group I mothers was in the range of 29.54 – 33.09 % with a mean value of 31.11 %, S.D. ± 0.8. In group II mothers M.C.H.C ranged from 25.55 – 32.33% with a mean value of 28.32%, S.D. ± 1.88. The M.C.H.C. of group III mothers ranged from 19.41 – 27.24 % with a mean value of 22.98%, S.D. ± 2.86. In group IV mothers the M.C.H.C. value ranged from 17.30 – 22.50 % with a mean value 0f 20.35%, S.D. ± 1.49.

On subjecting the above data to statistical analysis in differences in the mean hemoglobin, R.B.C. count and P.C.V. of control group I and anemic group II, III and IV were found to statistically significant, p value < 0.001. Similarly, M.C.H. and M.C.H.C. values also showed statistically significant.

The cord hemoglobin level of newborns to control group I was in the range of 13.5 – 19.3 gm/dl with a mean value of 15.96 gm/dl, S.D. ± 1.79. The newborns to group II mothers had cord hemoglobin level ranging from 11.5 – 17.3 gm/dl, with a mean value of 13.9 gm/dl, S.D. ± 1.63. the babies born to group III mothers had cord hemoglobin level ranging from 11.5 – 16.1 gm/dl with a mean value of 13.44 gm/dl, S.D. ± 1.25. The cord hemoglobin level of babies born to group IV mothers ranged from 13.4 – 15.3 gm/dl with a mean value of 14.28 gm/dl, S.D. ± 0.57. On

subjecting these data to statistical analysis the cord hemoglobin of newborn to non-anemic control group I was found to be significantly higher than those belonging to anemic case group II, III and IV. On the other hand it was also observed that the difference between cord hemoglobin of neonates delivered to group II, III and IV mothers were not statistically significant with the p value being more than 0.05.

A drop in maternal hemoglobin level from a mean value of 13.28 gm/dl to 9.95 gm/dl resulted in a significant fall of cord blood hemoglobin level with p value being less than 0.001. However, further fall in maternal hemoglobin to 7.35 gm/dl and 4.85 gm/dl did not result in any significant decline in hemoglobin level of the cord blood of babies born to those mothers.

The mean value of total R.B.C. count in control group I mothers was 5.1 millions/cu.mm, S.D. ± 0.63 with a range of 4.0 – 6.2 millions/cu.mm. In case group II mothers the means total R.B.C. count was 4.52 millions/cu.mm, S.D. ± 0.48 with a range of 3.6 – 5.3 millions/cu.mm. The mean total R.B.C. count of the cord blood of newborns of group III was 4.03 millions/cu.mm, S.D. ± 0.29 with a range of 3.4 – 4.6 millions/cu.mm. In group IV newborns the total R.B.C. count in cord blood ranged from 3.8 – 4.7 millions/cu.mm with a mean value of 4.13 millions/cu.mm, S.D. ± 0.29. On statistical analysis a significant differences was observed between total R.B.C. count of cord blood of babies born to case group(anemic) and control group (non-anemic) mothers. It was further observed that a fall in maternal hemoglobin level from 13.20 to 9.95 gm/dl resulted in a significant fall in cord blood total R.B.C. count of the babies. However, further lowering of maternal hemoglobin level failed to produce any significant lowering of R.B.C. count in respective cord blood.

The P.C.V. value of babies born to non-anemic control group I mothers was in the range of 43 – 61% with a mean value of 51.28%, S.D. ± 4.99. In the cord blood of babies born to group II, III and IV mothers, the P.C.V. values ranged from 38 to 51%, 39 to 48% and 39 to 49% with the mean value of 46.76 ± 4.98, 43.33 ± 2.67 and 43.66 ± 3.33 respectively. The P.C.V. values also showed similar pattern on statistical analysis, i.e. significant difference was noted in P.C.V, values of babies born to anemic and non-anemic mothers.

The M.C.H. value of babies born to non-anemic control group I ranged from 27.73 – 34.28 micro microgram with a mean value of 31.73, S.D. ± 1.53. This value in cord blood of babies born to case group II, III and IV mothers were in the range of 28.77 – 33.80 micro micrograms, 27.04 - 37.56 micro micrograms and 31.86 – 39.23 micro micrograms with the mean value of 30.71 ± 1.36, 34.07 ± 2.14 and 34.31 ± 2.13 respectively. On statistical analysis it was seen that fall in maternal hemoglobin level does not produce any significant decline in M.C.H. value of cord blood of babies born to such mothers.

Elgari and Waggiallah et al (2013) was studied on anaemic and anaemic mothers and their neonates, their findings supported the findings of our study.[16]

Summary & Conclusion

Investigations were conducted on one hundred full term pregnant women with uneventful gestation and their single born full term normal babies to elucidate the effect of maternal low hemoglobin level on the hemoglobin level of the newborns. All the babies were born spontaneously through vaginal route.

A series of haematological investigations namely haemoglobin estimation, R.B.C. count, P.C.V. estimation and calculation of M.C.H. and M.C.H.C. were done 2 – 48 hours before delivery on maternal blood with a purpose of establishing the presence or otherwise of iron deficiency anemia and to determine its severity.

The normal non-anemic controls designated as group I compromised of 25 pregnant women having a hemoglobin level of more than 12.0 gm/dl. The remaining 75 pregnant women having hemoglobin level below 12.0 gm/dl were regarded as anemic case group and were further devided

into three groups (group II: Hb range 9.1 – 12.0 gm/dl, group III: Hb range 6.1 – 9.0 gm/dl and group IV: Hb range 6.0 gm/dl or less).

After delivery, the newborns were assigned to their mothers group. These newborns were subjected to the same set of investigations which were carried out on their mothers prior to delivery. The results of these investigations were analyzed statistically and the level of their significance was determined.

For group I, II, III and IV newborns, the mean hemoglobin value was 15.96 gm/dl, 13.9 gm/dl, 13.44 gm/dl and 14.26 gm/dl respectively.

When maternal hemoglobin falls from the level above 12.0 gm/dl to a range between 9.1 to 12.0 gm/dl, there occurs a significant fall ($p<0.001$) in cord blood hemoglobin level. However, a further fall in maternal hemoglobin does not result in any significant fall ($p<0.05$) in cord blood hemoglobin level of the respective newborn.

Values of R.B.C. count, P.C.V., M.C.H., and M.C.H.C., of mothers belonging to various groups and their newborns cord blood, more or less similar results were obtained.

Thus, it could be propounded that interrelationship between maternal and neonatal hemoglobin level is more complex than ordinarily conceived. However, it can be concluded from this study that maternal anemia has a definite bearing on neonatal hemoglobin level.

Hence we concluded that, if the mother suffers from anemia, i.e. low hemoglobin level, the baby born to will also have low cord hemoglobin level and it may also be the cause of anemia in further stages of his growth as an individual.

Relevance to Cinical Practice

In our country, anemia associated with malnutrition is very common, especially among poor socio-economic groups. Furthermore, in our country, anemia antedates pregnancy and gets aggravated during pregnancy and labour. It is further propounded by successive pregnancies and lactation. Therefore, it is essential to improve the nutritional status of women by combating faulty dietary habits arising from poverty, ignorance of food values, illiteracy and superstitions.

We can be safely recommended that public health programs should give top priorities to program to ensuring adequate hemoglobin level by proper screening program and nourishment to expectant mothers.

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Original Research Article

A Study on the Correlation of Serum uric acid and Dyslipidemia with Glycaemic Status in Type 2 Diabetes Mellitus

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Abstract
Objectives: *Our study was to detect the correlation of serum uric acid level with glycaemic status and with lipid profile. And also evaluate the various biochemical parameters, anthropometric measurements, blood pressure, serum uric acid level and associated factors.*
Methodology: *A 100 subjects with type 2 diabetes mellitus as a case and 100 subjects with non diabetics as control with age group greater than 40 years were enrolled in this study. A detail history, dietary pattern, clinical examination and relevant investigation were performed. Anthropometric examination like as measurement of BMI, measurement of waist-hip ratio and biochemical investigations like as blood glucose, Serum HbA1C estimation, Serum uric acid and Serum lipid profile were performed to all subjects.*
Results: *Data was analyzed by using SPSS software (Version 17). Mean ± SD was observed. One way analysis of variance (ANOVA) with post hock analysis using Tukey's multiple comparison test and Pearson correlation coefficient (r) was applied. P value was taken ≤ 0.05 for significant differences.*
Conclusions: *Type 2 diabetes mellitus patients is a strong negative correlation between blood glucose level and serum uric acid level. So that serum uric acid can be used as an important parameter to assess future cardiovascular risk in a type2 diabetes mellitus patient.*
Keywords: *Type 2 diabetes mellitus, anthropometric examination, Serum uric acid, serum lipid profile*

Introduction

Diabetes mellitus (DM) is a hereditary, chronic and endocrine metabolic disorder.[1] It may be associated with a number of complications including microangiopathies e.g. nephropathy, neuropathy, retinopathy, dermopathy and macroangiopathies e.g. coronary artery disease (CAD), cerebrovascular disease, peripheral vascular disease.

India, a developing Asian country with fast industrialization and a modern lifestyle is facing a grave problem in having the largest number of people with diabetes[2,3] which is estimated to reach 80 million by the year 2030.[4,5] It is close to becoming the diabetic capital of the world. The age of the diabetic patients play a significant role in the risk of developing type 2 DM especially after 40yrs.[6] Type 2 DM is caused by relatively

impaired insulin secretion and peripheral insulin resistance.[7,8] Lack of insulin or relatively low insulin levels affects the metabolism of carbohydrate, protein, fat, water and electrolyte balance resulting in diabetes.[9]

Several distinct types of diabetes mellitus exists and are caused by a complex interaction of genetics, environmental factors and most importantly on lifestyles. There is a very important role of diet on both causation and treatment of diabetes mellitus. Depending on the etiology of the diabetes mellitus factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose utilization and increased glucose production. The metabolic dysregulation associated with diabetes mellitus causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes as well as on the health care system. The incidence of cardiovascular disease is increased in individuals with type 2 diabetes mellitus. The Framingham Heart study revealed a marked increase in coronary artery disease, myocardial infarction and sudden cardiac death in diabetes mellitus patients. The absence of chest pain (silent ischaemia) is also very common in patients of diabetes mellitus. So, to avoid such catastrophies various biochemical blood parameters, cardiological investigations should be done as a part of follow up in a diabetes mellitus patients.

Plasma uric acid, an end product of purine metabolism, is related to the purine bases of the nucleic acids. Its levels are genetically determined, but are influenced by multiple environmental factors. Previously it had been thought to be a metabolically inert end product without any physiological significance. Recently, it has been shown that there is a definite relationship between hyperglycemia and uric acid levels. Studies done so far have shown that, in the early stages of diabetes, the levels were high and as the diabetic status progresses, there is a gradual decline of uric acid levels in many patients. Studies showed that uric acid can act as an important water soluble antioxidant.[10,11] Urate, the soluble form of uric acid, can scavenge the superoxide and the hydroxyl radical and it also can chelate the transition metals.[12] In a study by J. Fang, M.H. Alderman on serum uric acid and cardiovascular mortality it has been shown that serum uric acid level has a continuous, independent, specific and significant negative relationship with cardiovascular mortality.[13] Studies have shown that serum uric acid level is negatively correlated with serum total cholesterol, LDL-cholesterol and triglyceride level, and positively correlated with serum HDL-cholesterol level. This dyslipidemia is also a cause of cardiovascular mortality.[14]

There are evidences to suggest that low serum uric acid levels may precede the onset of diabetic retinopathy. It has been reported that hypouricemia may also predict the future progression and hence be an indicator of incipient nephropathy in Type 2 DM.[15] Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high-density lipoprotein-Cholesterol (HDL-C) level that contributes to the development of atherosclerosis, which may be primary (genetic) or secondary and diagnosed by measuring plasma levels of total cholesterol (TC), TGs, and individual lipoproteins. It is traditionally classified by patterns of elevation in lipids and lipoproteins.[16] Patients with type 2 DM are at greater risk of developing vascular diseases because of lipid changes. Aims of our study was to detect the correlation of serum uric acid level with glycaemic status as well as with lipid profile, and to find out various biochemical parameters, anthropometric measurements, blood pressure, measure the serum uric acid level and evaluate the association, if any, between the serum uric acid level and the factors measured of subjects.

Materials & Methods

A descriptive case –control study was conducted on the basis of inclusion and exclusion criteria, in department of Physiology, with the help of department of Medicine, Katihar Medical College,

Katihar, Bihar during period of June 2016 to July 2017.

A 100 diagnosed type 2 diabetes mellitus patients were enrolled in this study and 100 nondiabetes healthy individuals age and sex matched were taken as control. Male and female ratio was same. Entire subjects signed an informed consent approved by institutional ethical committee of Katihar Medical College, Katihar, Bihar, India was sought. Data was collected using random sampling.

Inclusion criteria of this study were subjects with age more than 40 years, diagnosed type II diabetes mellitus with no previous history of diabetic keto acidosis or pancreatitis. Exclusion criteria were patients suffering from Kidney disease, hepatic disorder, patients on diuretic therapy (mainly thiazides), history of alcoholism, suffering from myeloproliferative disorders, lymphoproliferative disorders and patients suffering from Psoriasis.

Methods

A detailed history was taken about their dietary pattern. Subjects were demonstrated steps of investigations properly. Next they were undergone investigations. All the reports of investigations and any altered status were explained to the patients.

History

A case data sheet was used to assess the clinical history, including past and present diseases both acute and chronic from all the subjects. The subjects were asked about their diet pattern. Emphasis was given on occupation, family history of diabetes mellitus, coronary artery diseases, musculoskeletal disorders, arthropathy. Their family income, history of addiction and level of physical activity was assessed.

Regarding present illness of the cases a meticulous history was taken about the chief complaints, symptoms of diabetes mellitus, duration of diabetes, symptoms of diabetes related complications e.g. decreased vision, pedal edema, chest pain, calf muscle pain, respiratory distress, increased frequency of micturation. Treatment history, both for diabetes and its complications, in the form of drugs, dietary modification, lifestyle modification were also carefully noted.

Clinical assessment: It was started with general survey followed by systemic examinations.

General survey: Built, decubitus, anaemia, jaundice, cyanosis, clubbing, oedema, pulse, blood pressure, neck veins, neck glands, skin changes, height, weight.

Systemic examination: Emphasis was given upon the examination of cardiovascular system and endocrinal system. Other systems were also examined in brief.

Anthropometric measurements: Body mass index and waist- hip ratio.

Study tools was used in our study had stadiometer, measuring tape, weighing machine, Mercury sphygmomanometer.

Measurement of BMI: Height of subjects was measured with the help of a stadiometer. Weight was measured by the help of a weighing machine. BMI was calculated by dividing the weight of subjects in kilograms by the square of the height in meters. [17]

Measurement of waist-hip ratio: Waist circumference was measured (in centimetres) around the narrowest point between the lowest rib and hip when viewed from the front after exhaling. Hip circumference was measured (in centimetres) at the point where buttock is maximally extended, when viewed from the side. The ratio was calculated.[18]

Biochemical Investigations

Plasma Glucose was estimated by GOD-POD Method, End Point Assay and Kinetic Assay. [19] Glycosylated Haemoglobin was estimated by ion exchanged resin method for quantitative determination of glycohaemoglobin in blood. [20] Serum uric acid was estimated by Uricase / PAP method.[21] LDL Cholesterol was estimated by direct determination of LDL Cholesterol.[22] HDL Cholesterol was found by direct enzymatic method.[23] Triglycerides estimation was done by GPO / PAP method.[24]

Investigations proper

Blood glucose: Subjects had blood drawn after an overnight fast for fasting blood sugar. Analysis of post prandial blood sugar was done 2 hours after having a meal.

Plasma glucose was estimated by GOD/POD method by using spectrophotometer in the department of physiology, Katihar Medical College and Hospital. The kit for estimation of glucose was supplied by Crest Biosystems.

Serum HbA1C estimation: Serum glycosylated haemoglobin concentration of the patients was measured in the department of Biochemistry Katihar Medical College and Hospital. It was measured by ion –exchange HPLC with a glycosylated haemoglobin analysing system (DIAMAT, Bio-Rad Laboratories, Hercules,CA, USA).

Serum uric acid: Venous blood samples were taken in the morning with the subjects fasting for 12 hours. The uric acid was measured by the uricase method. [25]

Serum lipid profile: Serum lipid profile was measured of each subject in the department of Biochemistry by using different kits and spectrophotometer. Total cholesterol was estimated by CHOD-POD (cholesterol oxidase peroxidise) method. [26] A kit manufactured by LOGOTECH INDIA Pvt. Ltd was used.

Statistical Analysis

Data was analyzed by using SPSS software (Version 17). Mean ± SD was observed. One way analysis of variance (ANOVA) with post hock analysis using Tukey's multiple comparison test was used for parametric data. Pearson correlation coefficient (r) was used for correlation of data. P value was taken ≤ 0.05 for significant differences.

Observations & Results

A comparative cross sectional study was conducted on randomly selected 100((50: females and 50: males) diagnosed type2 diabetes mellitus patients. 100 subjects age and sex matched nondiabetic persons were taken as controls.

Table.1. Basic characteristics of the study subjects.

Parameters	Cases (N=100) MEAN±2SD	Controls (N=100) MEAN±2SD	P-value
Age(years)	56.70±14.10	56±13.24	0.869
BMI(kg/m2)	27.35±5.15	26.49±4.25	0.011
WHR	0.92±0.22	1.77±0.12	0.336
SBP	135.96±32	127±28.4	<0.05*
DBP	83.72±24	78.6±21.54	0.002*

Intergroup comparison shown that samples was age matched (P>0.05).There was no significant difference of BMI, WHR among the groups. There was significant difference (i.e.P<0.05) of systolic and diastolic blood pressure among the case and control groups.

Table.2. Distribution of subjects according to age.

Age distribution(Years)	Cases Percentage (%)	Control Percentage (%)
<50	52.6 (n=30)	47.4 (n=27)
51-60	47.5 (n=38)	52.5 (n=42)
>60	50.8 (n=32)	49.2 (n=31)

The above table shows that among the age distribution <50, cases were 52.6%, controls 47.4%. In the age group 51-60, cases were 47.5% and controls 52.5%.Among the age group>60, cases were 50.8% ,controls 49.2%.

Table.3. Distribution of BMI among subjects.

BMI distribution	Case Percentage (%)	Control Percentage (%)
18.5-22.9	83.3 (n=10)	16.7 (n=2)
23-29.9	42.7 (n=64)	57.3 (n=86)
≥30	68.4 (n=26)	31.6 (n=12)

This table shows that in the obese BMI group (23-29.9) cases were 42.7% and controls 57.3%. Among the morbid obesity group (BMI>30) cases were 68.4% and controls 31.6%.

Table 4 Distribution of WHR among cases and controls.

WHR	Cases(%)	Controls(%)
>1	80.95(n=17)	19.04(n=4)
<1	46.37(n=83)	53.63(n=96)

Patients who had waist-hip ratio >1, cases were 80.95% and controls 19.04%. Of those who had waist –hip ratio <1, cases was 46.37% and controls 53.63%.

Table 5: Intergroup comparison of serum glucose levels between cases and controls.

Parameters	Cases MEAN±2SD	Controls MEAN±2SD	P value
FBS	210.51±106.14	75.62±20.66	<0.05*
PPBS	282.24±119.89	123.57±20.23	<0.05*
HbA1c	11.54±4.8	4.95±11.64	<0.05*

Above table shows significant differences (P<0.05) of fasting, post-prandial blood glucose levels and glycated haemoglobin level between cases and controls. All the values was significantly higher in cases (type2 diabetics) than control (normoglycaemics) group.

Table.6. Intergroup comparison of lipid profiles between cases and controls.

Parameters	Cases MEAN±2SD	Controls MEAN±2SD	P values
TOTAL CHOLESTEROL	264.43±89.52	176.13±52.04	<0.05*
LDL-C	180.28±89.78	106.20±51.78	<0.05*
HDL-C	39.70±23.08	56.71±16.14	<0.05*
TRIGLYCERIDE	204.78±97.42	148.40±67.38	<0.05*

Above table shows significant difference exists between cases and controls in total cholesterol, LDL-cholesterol, HDL-Cholesterol and triglyceride levels. The total cholesterol, LDL-cholesterol and triglyceride in cases (Diabetics) was significantly higher than controls (Normoglycaemics) and HDL-Cholesterol level in cases is significantly lower than control group.

Table.7. Distribution of dyslipidemia among subjects.

Lipid Profile Status	Cases (%)	Control (%)
Dyslipidemic	67.5 (n=85)	32.5 (n=41)
Non-dyslipidemic	20.3 (n=15)	79.7 (n=59)

Table shows that among the dyslipidaemics 67.5% were cases and 32.5% controls. Among non-dyslipidaemics 20.3% were cases, 79.7% controls. So it can be inferred that distribution of dyslipidaemia was more in cases (diabetics) than controls (non-diabetics).

Table.8. Intergroup comparison of serum uric acid levels between cases and controls.

Parameter	Cases MEAN±2SD	Controls MEAN±2SD	P-Value
Serum uric acid	3.55±2.436	7.38±2.142	<0.05*

When compared the serum uric acid, p value was found to be ≤ 0.05. Significant difference of serum uric acid exists between cases (diabetics) and controls (nondiabetics). It also shows that serum uric acid level was significantly lower in diabetic group than normoglycaemics.

Table 9. Correlation of serum uric acid level with blood glucose parameters.

Blood glucose parameters	Correlation of serum uric acid	
	Pearson correlation coefficient(r)	Significance (2-tailed)
Glycated haemoglobin	-0.918**	P<0.05*
Fasting blood sugar	-0.829**	P<0.05*
Post prandial blood sugar	-0.879**	P<0.05*

The above table shows a significant strong negative correlation exist between serum uric acid level and glycated haemoglobin, fasting blood sugar, post prandial blood sugar levels. This correlations was also demonstrated by scatter diagrams.

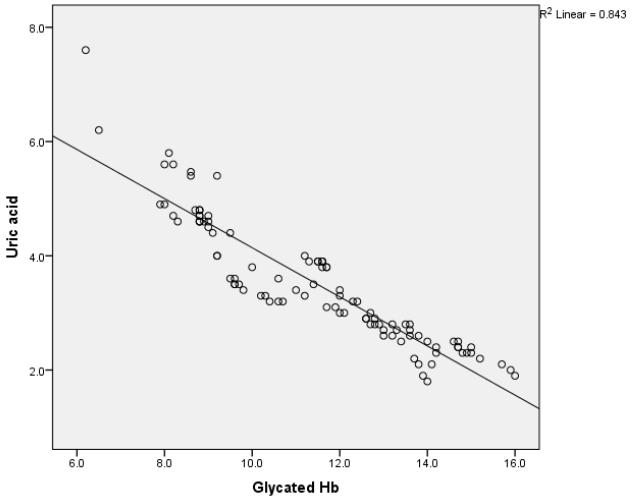

Figure.1. Correlation of uric acid with glycated Hb.

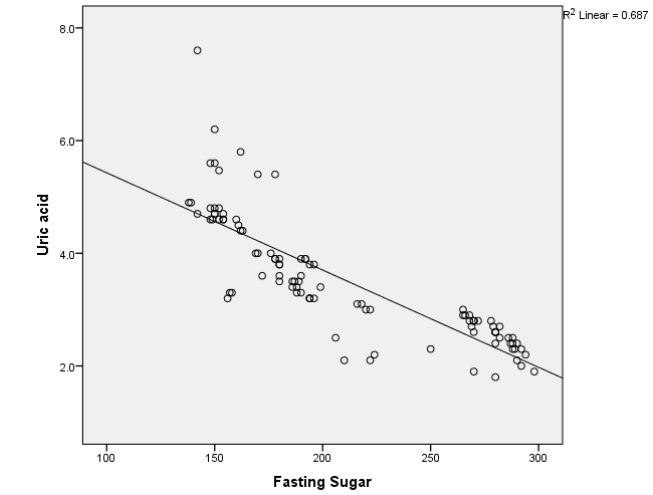

Figure.2. Correlation between uric acid and fasting sugar.

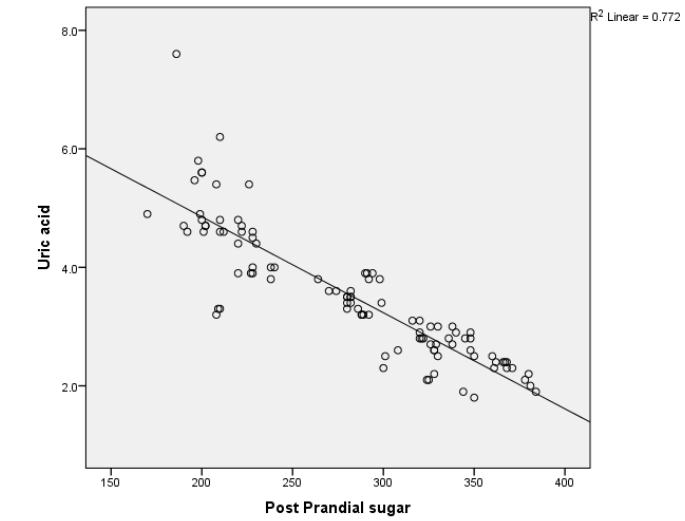

Figure.3. Correlation between uric acid and post prandial sugar

Table.10. Correlation of serum uric acid level with serum lipid profile.

LIPID PROFILE	Correlation of serum uric acid	
	Pearson correlation coefficient(r)	Significance (2 – tailed)
Total cholesterol	-0.642**	0.000 *
LDL-cholesterol	-0.616**	P<0.05 *
HDL-cholesterol	0.651**	P< 0.05 *
Triglyceride	-0.721**	P<0.05 *

Table6. shows a significant strong negative correlation was exist between serum uric acid level and total-cholesterol, LDL-cholesterol and triglyceride levels. A significant positive correlation was exist between serum uric acid level and HDL-cholesterol level. This correlations was also demonstrated by scattered diagrams.

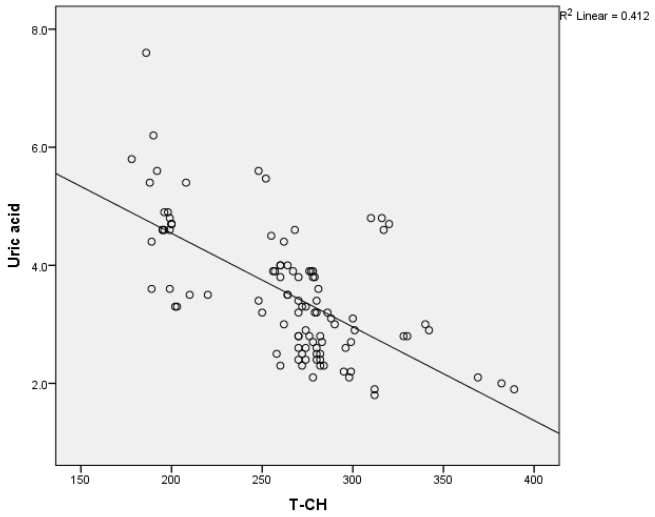

Figure.4 Correlation between uric acid and T-CH.

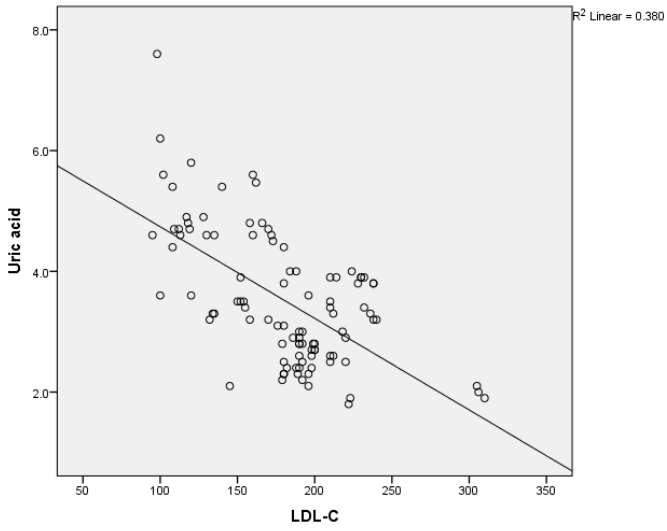

Figure.5. Correlation between serum uric acid and LDL-C.

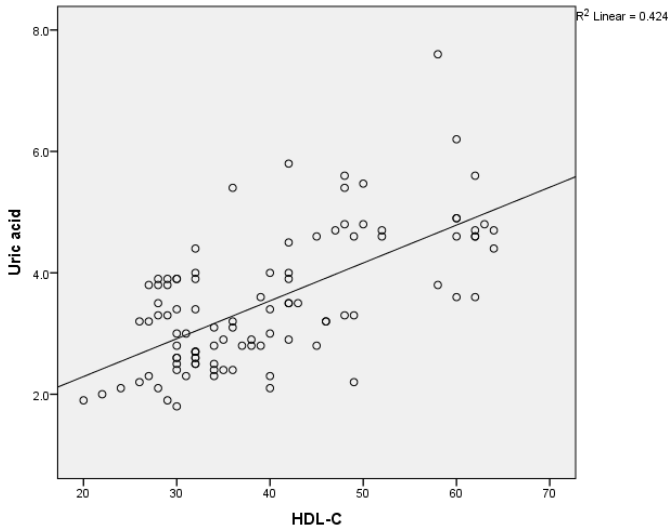

Figure.6. Correlation between uric acid and HDL-C.

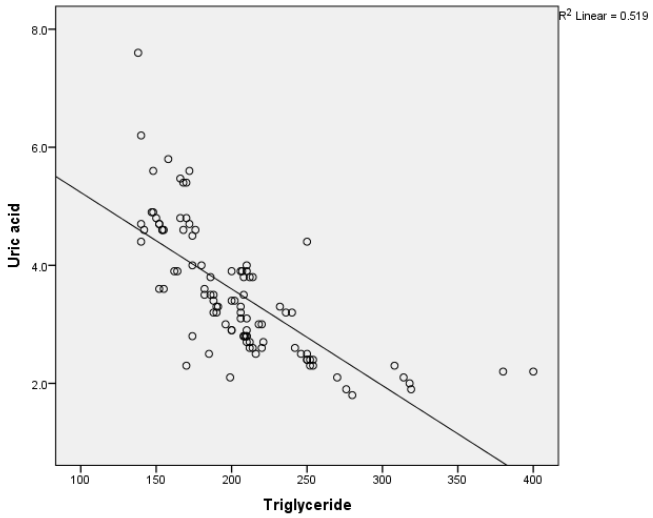

Figure.7. Correlation between uric acid and triglyceride.

Discussion

India, a developing Asian country with fast industrialization and a modern lifestyle is facing a grave problem in having the largest number of people with diabetes [2,3] which is estimated to reach 80 million by the year 2030.[4,5] It is close to becoming the diabetic capital of the world.

The incidence of cardiovascular disease is increased in individuals with type2 diabetes mellitus. The Framingham Heart study revealed a marked increase in coronary artery disease, myocardial infarction and sudden cardiac death in diabetes mellitus patients. The absence of chest pain (silent ischaemia) is also very common in patients of diabetes mellitus. So, to avoid such catastrophies various biochemical blood parameters, cardiological investigations should be done as a part of routine follow up.

Plasma uric acid, an end product of purine metabolism, is related to the purine bases of the nucleic acids. Its levels are genetically determined, but are influenced by multiple environmental factors. Previously it had been thought to be a metabolically inert end product without any physiological significance.

Recently, it has been shown that there is a definite relationship between hyperglycemia and uric acid levels. Studies done so far have shown that, in the early stages of diabetes, the levels were high and as the diabetic status progresses, there is a gradual decline of uric acid levels in many patients. Studies showed that uric acid can act as an important water soluble antioxidant[27,28]. Urate, the soluble form of uric acid, can scavenge the superoxide and the hydroxyl radical and it also can chelate the transition metals.[29] There are evidences to suggest that low serum uric acid levels may precede the onset of diabetic retinopathy. It has been reported that hypouricemia may also predict the future progression and hence be an indicator of incipient nephropathy in Type 2 diabetes mellitus patients. In diabetes mellitus the uric acid excretion is increased due to osmotic dieresis caused by high plasma glucose level. As a result the plasma uric acid level is also decreased. However the exact relationship between the uric acid level and blood glucose parameters are still unknown. To further investigate these observations, we have conducted a case – control study on 100 type2 diabetes mellitus patients and 100 normoglycaemic healthy subjects to assess the blood glucose parameters, serum uric acid levels, lipid profiles and some anthropometric parameters.

In this study, we were found that there was no significant difference of Age, BMI, WHR among the groups. There was significant difference (i.e.P<0.05) of systolic and diastolic blood pressure among the case and control groups. It was found that the cases had a mean age of 56.70 whereas the mean age among the controls was 56.54. It also shown that all the cases and controls were age matched (P>0.05).

Among the age distribution <50, cases was 52.6%, controls 47.4%. In the age group 51-60, Cases were 47.5% and controls 52.5%. Among the age group>60, cases were 50.8%, controls 49.2%.

Regarding BMI it was suggest that in the obese BMI group (23-29.9) cases were 42.7% and controls 57.3%. Among the morbid obesity group (BMI>30) cases were 68.4% and controls 31.6%. Mean ± S.D. BMI of the cases (i.e. in Type2 diabetics) was 27.35 ± 5.15 and among the normoglycaemic control groups it was found to be 26.49 ± 4.25.

In this study, who had waist-hip ratio >1, cases were 80.95% and controls 19.04%. Of those who had waist –hip ratio <1, cases were 46.37% and controls 53.63%. It was found that mean ± S.D, waist-hip ratio of the cases were 0.92 ± 0.22 and among the control group it was 1.77 ± 0.12.

Intergroup comparison of various blood glucose parameters between cases and controls: It was seen that fasting, post prandial blood sugar and glycated haemoglobin levels in cases were 210.51±106.14, 282 ± 119.89 and 11.54 ±4.8, and in controls were 75.62 ± 20.66, 123.57 ± 20.23 and 4.95 ±1.64 respectively. It was seen that all the blood glucose parameters were significantly higher in cases than control groups ($p<0.05$). All these data confirms the selection of diabetics as cases.

Inter comparison of total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride levels between cases and controls: It was shown significant difference exists between cases and controls in total cholesterol, LDL-cholesterol, HDL-Cholesterol and triglyceride levels. The total cholesterol, LDL-cholesterol and triglyceride in cases (Diabetics) was significantly higher than controls (Normoglycaemics) and HDL-Cholesterol level in cases was significantly lower than control group($p<0.05$).

Distribution of dyslipidaemia among cases and controls: It was shown that among the dyslipidaemics 67.5% and 32.5% cases were in controls. Among non-dyslipidaemics 20.3% and 79.7% cases were in controls. So it could be inferred that distribution of dyslipidaemia was more in cases (diabetics) than controls (non-diabetics). There were various studies shown that high blood glucose level is associated with dyslipidaemia. One of the recent studies published in Indian Journal of Clin Biochem done by Mullugeta Y, Chawla R,KebedeT, in the year 2012 on 165 type2 diabetics were classified as good glycaemic control (group1) and poor glycaemic control (group2) on the basis of their blood HbA1C values.[30] The group2 was characterized with serum triglyceride (190.46± 15.20mg/dl), total cholesterol (175.3±6.31mg/dl), as well as high LDL-cholesterol levels (109.0±5.88mg/dl). Significant correlations was exists between HbA1c and dyslipidaemia, particularly serum TG ($r=0.28, p<0.05$), and between HbA1C and total cholesterol ($r=0.310$, $p<0.05$). So it can be said that our finding corroborates with the previous study results.

Intergroup comparison of serum uric acid level between cases and control groups: It was found that significant difference of serum uric acid level exists between cases (diabetics) and controls (nondiabetics). It was also shown that serum uric acid level was significantly lower in diabetic group than normoglycaemics. The mean and standard deviation of serum uric acid level in cases was 3.55 ±2.436 and 7.38(±2.142).The p value was <0.05.

In previous studies, Godfredsen et al [31] showed that diabetics had a 42% increase in renal uric acid excretion rate compared with normal. Diabetics had significantly lower mean serum uric acid concentrations. 17% of the diabetic patients had serum concentrations below the normal mean ± 2 standard deviations.

In a study in Israel by Herman and goldbourt in the year 1982 showed that prediabetic subjects had higher uric acid level s than non diabetics and that overt (clinically diagnosed) diabetics had lower uric acid levels than non diabetics.[25]

Their finding of a negative association at the highest extreme of the glucose distribution was further supported by another study done by Derek G.Cook, A.G.Shaper, D.S.Thelle and T.P. Whitehead on 7735 British men aged 40-59 in British regional heart centre.[26] The findings of our present study also agree with the findings of the previous studies.

In our study significant strong negative correlation exist between serum uric acid level and glycated haemoglobin, fasting blood sugar, post prandial blood sugar levels ($p<0.05$). The pearson's correlation coefficient (r) were -0.918,-0.829,-0.879 respectively.

In a study by Eiji Odda, Ryu Kawai et al on 2449 Japanese men and 1448 Japanese women the prevalence of metabolic syndrome and diabetes was calculated by the quartiles of serum level of uric acid levels. The results showed that prevalence of diabetes in third quartile was significantly lower than that in first quartile (lowest quartile) and the prevalence of diabetes in forth quartile was significantly lower than that in first and second quartile in men. The prevalence of diabetes was not significantly different among the quartiles of uric acid in women. They concluded that serum uric acid level is negatively associated with diabetes in Japanese men.[32]

In a study conducted by Quingdao Diabetes epidemiology study group, Quingdao, China over a total of 1288 men and 2344 women showed that serum uric acid levels declined with increasing fasting plasma glucose levels in individuals with diabetes mellitus, with standardized coefficient of – 0.26 in men and – 0.20 in women. [33]

A recent study published in international journal of endocrinology in the year 2011, done by Pavani Bandaru and Anoop Shankar, showed the association between serum uric acid levels and diabetes mellitus in participants from the NHANES (n=18,825,52.5% women). Serum uric acid was divided into quartilers . In multivariate logistic regression models, they found that higher serum acid levels were inversely associated with diabetes mellitus after adjusting for age, sex, race, smoking, alcohol intake, body mass intake, hypertension and serum cholesterol level. Compared to quartile 1 of serum uric acid ,the odd's ratio (95% confidence interval)of diabetes mellitus was 0.48 (0.35-0.66;P trend<0.0001). They concluded that higher serum uric acid levels were inversely associated with diabetes mellitus in a representative sample of US adults.[34] Our findings agree with the results of the previous studies.

In our study, the correlation of serum uric acid levels with the total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride levels were performed. It shown a significant strong negative correlation exists between serum uric acid level and total-cholesterol, LDL-cholesterol and triglyceride levels. A significant positive correlation was existed between serum uric acid level and HDL-cholesterol level. The pearson's correlation coefficients were -0.642,-0.616,0.651,-0.721 respectively.

In a study by J. Fang, M.H. Alderman on serum uric acid and cardiovascular mortality it shown that serum uric acid level had a continuous, independent, specific and significant negative relationship with cardiovascular mortality.[13] Serum uric acid level was negatively correlated with serum total cholesterol, LDL-cholesterol and triglyceride level, and positively correlated with serum HDL-cholesterol level. Finding of our study results was simillar with the findings of previous study.

In hyperglycemic state, the increasing glucose reabsorption may impair the tubular reabsorption of uric acid, as both glucose as well as filtered uric acid are reabsorbed at the same site, the proximal convoluted tubule. Continued hyperexcretion of uric acid due to hyperglycemia could deplete the uric acid pool and gradually reduce serum uric acid levels. Geoffrey Boner and Rieselbach[35,36] concluded that the presence of glucose in the renal tubule lumen at a site distal to that of normal glucose reabsorption inhibits the tubular reabsorption of uric acid. Later in 1987, Schichiri et al emphasized that there is a possible mechanism of glomerular hyperfiltr-ation, which brought about the increased renal clearance of urate and ultimately results in low serum uric acid level.

Limitation

1) The study population was small.
2) This was a cross sectional study with no follow up.
3) Study period was only one year.
4) The other parameters of nephrological and cardiological complications should be assessed (i.e. eGFR, ECG changes e.t.c.). That was not done in our study.

Summary & Conclusion

We were conducted a case –control study on 100 type 2 diabetes mellitus patients (taken as cases) with 100 non diabetic subjects(taken as controls) to assess whether there are any correlation between the serum uric acid level and the blood glucose parameters and the lipid profile, the serum uric acid level between the cases and controls are also compared. After inclusion of cases and controls, the whole procedure was explained to every subject and consent forms were duly signed. After that they underwent history taking, proper clinical examination, and special investigations (serum uric acid, blood glucose parameters, serum lipid profile). Then the data were collected and epilated on SPSS (Version-17) software and statistical analysis were done. Following results were obtained:

- Among the age distribution <50 years, 52.6% cases were in case group, and 47.4% in control group. In the age group 51-60 years, cases were 47.5% and controls were 52.5%. Among the age group>60 years, cases were 50.8%, controls were 49.2%.
- Regarding BMI, it was suggested that in the obese BMI group (23-29.9) case were 42.7% and controls were 57.3%. Among the morbid obesity group (BMI>30) cases were 68.4% and controls were 31.6%. BMI of the cases (i.e. in type2 diabetics) was 27.35 ±5.15 and among the normoglycaemic control groups was 26.49±4.25.
- Those who have waist-hip ratio >1, cases was 80.95% and controls was 19.04%.Of those who had waist –hip ratio <1, cases was 46.37% and controls was 53.63%. Mean ± S.D) waist-hip ratio of the cases was 0.92±0.22 and among the control group it was 1.77±0.12.
- The inter group comparison of total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride levels between cases and controls shown significant difference between cases and controls in total cholesterol, LDL-cholesterol, HDL-Cholesterol and triglyceride levels. The total cholesterol, LDL-cholesterol and triglyceride in cases (diabetics) were significantly higher than controls (Normoglycaemics) and HDL-Cholesterol level in cases was significantly lower than control group ($p<0.05$).It also shown that among the dyslipidaemics 67.5% subjects was cases and 32.5% subjects were controls. Among non-dyslipidaemics 20.3% were cases and 79.7% were controls. So it can be inferred that distribution of dyslipidaemia was more in cases (diabetics) than controls (non-diabetics).
- A significant difference of serum uric acid level was exists between cases (diabetics) and controls (nondiabetics). It shown that serum uric acid level was significantly lower in diabetic group than in normoglycaemics. The mean ± S.D. Serum uric acid level in cases was 3.55±2.436 and in cases was 7.38±2.142.The p value was <0.05.
- There was a significant strong negative correlation exist between serum uric acid level and glycated haemoglobin, fasting blood sugar, post prandial blood sugar levels ($p<0.05$). The pearson's correlation coefficient (r) were -0.918,-0.829,-0.879 respectively.
- A significant strong negative correlation was existed between serum uric acid level and total-cholesterol, LDL-cholesterol and triglyceride levels. A significant positive correlation was existed between serum uric acid level and HDL-cholesterol level. The pearson's correlation coefficients were -0.642,-0.616,0.651,-0.721 respectively.

Conclusion

Our study concluded that type 2 diabetes mellitus patients is a strong negative correlation between

blood glucose level and serum uric acid level. So it can be said that as the blood glucose level increases the serum uric acid level decreases. As the serum uric acid is an important water soluble antioxidant, low serum uric acid may give rise to much further oxidative damage to mainly small and large blood vessels. Serum uric acid level decreases the lipid profile worsens, which can give rise to cardiovascular complications in future. So, that serum uric acid level can be used as an important parameter to assess future cardiovascular risk in a type2 diabetes mellitus patient.

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