pISSN-2349-2562, eISSN-2349-2570

JEBMH

Journal of Evidence Based Medicine and Healthcare

Volume 1 / Issue 07 / September / 2014

Indexed in-

1. Index Copernicus (ICV 2013-5.64)
2. Google Scholar
3. WHO-HINARI
4. Infobase Index
5. J-Gate

ORIGINAL RESEARCH ARTICLE

OBSERVATION ON ANATOMICAL VARIATIONS OF VERMIFORM APPENDIX IN KOSI REGION OF BIHAR – A CADAVERIC STUDY

Sanjay Kumar Sinha[1], Vipin Kumar[2]

HOW TO CITE THIS ARTICLE:
Sanjay Kumar Sinha, Vipin Kumar. "Observation on Anatomical Variations of Vermiform Appendix in Kosi Region of Bihar – A Cadaveric Study". Journal of Evidence Based Medicine and Healthcare; Volume 1, Issue 7, September 2014; Page: 564-568.

ABSTRACT: Anatomical and topographical variations in positions of the vermiform appendix are known to occur. Appendicitis is one of the most common clinical conditions that requires immediate surgical intervention and is globally prevalent. Variations in anatomical position may cause different clinical presentations. The operating surgeons require a sound knowledge of positions of the appendix to minimize complications encountered during appendicectomy.

KEYWORDS: Variations, Positions, Vermiform Appendix.

INTRODUCTION: The caecum and its diverticulum the vermiform appendix appear in the sixth week of intrauterine life as an elevation on the antimesenteric border of the caudal limb of the midgut loop. The caecal apex does not grow as rapidly as the remaining portion hence the vermiform appendix initially appears to be a small diverticulum of the caecum. The appendix increases in length so that at birth it is a relatively long tube arising from the distal end of the caecum.[1] After birth due to differential growth of the caecal wall the appendix now comes to lie on the medial aspect. Developmentally the appendix varies considerably in position. The vermiform appendix is the most variable organ in terms of position, extent, peritoneal and organ relations.[2,3,4&5] As the ascending colon elongates the appendix may pass posterior to the caecum or colon and may also descend over the brim of the pelvis. In approximately 64% of the people the appendix is located retrocaecally.[1] The appendix is located in the right lower quadrant of the abdomen.[6] It usually arises as a diverticulum from the posteromedial wall of the caecum. Its opening is occasionally guarded by a semicircular fold of mucous membrane known as the valve of Gerlach.[7] The appendix is usually located at the junction of the taeniae found on the surface of the caecum.[8 & 9] Length of the appendix varies from 2-20 cms with and average length of 9 cms.[10] Unique 28 cms long vermiform appendix has been reported.[11] Base of the appendix is attached to the caecum and this attachment is fairly constant whereas the tip can be found in retrocaecal, pelvic, subcaecal, pre-ileal and post-ileal positions. A short triangular mesoappendix extends along the whole length almost up to the appendicular tip. The mesoappendix has a free border which carries the blood supply to the organ by the appendicular artery a branch from the ileocolic artery.[12] Appendicitis is the most common cause of acute abdomen in young people. Identification of the normal position of appendix is important because in appendicitis variant positions may produce symptoms and signs related to their position and hence can mimic other diseases.[14] This study has been conducted on cadavers in Kosi region of Bihar to observe the length and positions of the vermiform appendix along with the extent of mesoappendix.

MATERIALS AND METHODS: During routine cadaveric dissections performed in the Department of Anatomy, Katihar Medical College, Katihar, fifty specimens of composite viscera of caecum, appendix, last two inches of ileum and first two inches of ascending colon were dissected and removed from adult cadavers with intact mesoappendix. The specimens were washed under running tap water and were fixed in 10% formalin overnight. The specimens were measured using measuring tape and observations on length and positions were recorded. Extent of mesoappendix was also observed.

OBSERVATIONS: The findings are presented in tabular form.

Position	No. of Males (38)	Percentage (%)	No. of Females (12)	Percentage (%)
Retrocaecal	24/38	63	07/12	58
Pelvic	08/38	21	03/12	25
Paracolic	03/38	07	01/12	08
Promontoric	02/38	05	01/12	08
Splenic	00/38	00	00/12	00
Subcaecal	01/38	02	00/12	00

Table 1: No. of cases and their percentage in each position

Position	Avg. Length (cm) Males	Avg. Length (cm) Females
Retrocaecal	7.4	6.9
Pelvic	6.9	6.1
Paracolic	6.6	5.7
Promontoric	6.3	5.4
Splenic	0.0	0.0
Subcaecal	5.6	0.0

Table 2: Average length of appendix in each position

Males	Females
A = 36 (95%)	A = 11 (92%)
B = 02 (05%)	B = 01 (08%)

Table 3: Extent of meso-appendix

A = Mesoappendix extending up to the tip
B = Mesoappendix failing to reach the tip

Group	Males	Percentage (%)	Females	Percentage (%)
1	32/38	84	09/12	75
2	05/38	14	01/12	08
3	01/38	02	02/12	17

Table 4: Arterial supply of appendix

1. Single AA branching from inferior division of ileocolic artery
2. Single AA branching directly from ileocolic artery
3. Accessory AA present

AA = Appendicular Artery

DISCUSSION: In this study we observed the following. With reference to variations in position the findings in males were as follows: retrocaecal (63%), pelvic (21%), paracolic (07%), promontoric (05%), splenic (00%) and subcaecal (02%) while in females the findings were retrocaecal (58%), pelvic (25%), paracolic (08%), promontoric (08%), splenic (00%) and subcaecal (00%). In this study retrocaecal position was commonest and has been previously reported by Ajmani & Ajmani (1983).[15] The average length of the appendix was calculated to be 5.46 cm in males and 4.02 cm in females and was within normal limits.[10] Mesoappendix in males extended up to the appendicular tip in 95% while in females the extent was recorded to be 92%. Failure of the mesoappendix to reach the tip can make the appendix vulnerable to gangrenous changes as there is reduced vascularity.[17] In males 84% of cases showed a single appendicular artery branching from inferior division of ileocolic artery while in females it was 75%. Appendicular artery branching directly from the ileocolic artery was observed to be 14% in males and 08% in females. Accessory appendicular arteries were found in 2% of males and 17% in females. With reference to positions of appendix our results are not similar to another study in Zambia (Katzurskj et al, 1979) in which pelvic position was commonest (43%). But in other studies retrocaecal position was commonest position (Bahkeit and Warille, 1999; Collins, 1932; Ajmani & Ajmani, 1983). Common incidence of retrocaecal position have been reported in England 65% (Wakely, 1932), Nijeria 38% (Solanki, 1970), India 68% (Ajmani & Ajmani, 1983). The average length of appendix was 5.46 cm in males and 4.02 cm in females. This was less than that seen in other studies (Williams etal, 1995, Schwartz et al, 1999; Bakheit and Warille, 1999; Katzurskj et al, 1979; Collins, 1932; Ajmani & Ajmani, 1983). In 5% of males and 8% in females the mesoappendix failed to reach the appendicular tip which was lower, compared with 46.7% in Sudan (Bakheit and Warille, 1999). Due to failure of mesoappendix to reach the tip of the organ makes it more susceptible to perforation during inflammation (Anderson) et al, 1992). The classical teaching in medical colleges of India emphasizes on the fact the appendix lies deep at the junction between lateral 1/3 and medial 2/3 of the right spino umbilical line, the so called Mc Burney's point. It is not mandatory for the appendicular base to lie at this definite site. In India where open appendicectomies form the significant majority surgeons need to be aware of the variations for preoperative planning and surgical outcomes. Trainee surgeons should not be surprised if the appendix is not visualized when a transverse incision is made at the Mc Burney's point.

CONCLUSION: The vermiform appendix is the most variable organ in the abdomen and appendicitis should always be considered as a differential diagnosis in acute abdomen even when pain and tenderness do not originate from right iliac fossa. The location of the appendix is . . . . The area of tenderness in appendicitis will depend upon the length, position, part, inflammation and direction of appendix.

REFERENCES:

1. Moore K.L. and Persaud T.V.N: The Developing Human Clinically Oriented Embryology. 8th ed; Saunders Elsevier. 2008 .p228.
2. Delic J, Savkovic A and Isakovic E: Variations in the position and point of origin of the vermiform appendix. Medicinski Archiv, 2002; 56(1): 5-8.
3. Ahmed I, Asgeirrson K.S, Beckingham I.J. and Lobo D.N: The position of vermiform appendix at laparoscopy. Surgical and Radiologic Anatomy, 2007; 29(2):165-168.
4. Paul U.K, Naushaba H, Alam M.J, Begum T, Rahman A and Akhter J: Length of vermiform appendix: A post-mortem study. Bangladesh Journal of Anatomy. 2011; 9(1):10-12.
5. Banerjee A, Kumar I.A, Tapadar A and Pranay M: Morphological variations in the anatomy of caecum and appendix – A cadaveric study. National Journal of Clinical Anatomy, 2012; 1(1): 30-35.
6. Sabiston D.C: Sabiston's Textbook of Surgery, The Biological Basis of Modern Surgical Practice. Appendix. 16th ed. Saunders.2001; 2: 918.
7. Singh I.B: Chaurasia's Human Anatomy Regional and Applied. 3rd ed; CBS Publishers.1999, 223-225.
8. Williams P.L, Bannister L.H, Berry M.M, Collins P, Dyson M, Dussek J.E and Fergusson M.W.J: Gray's Anatomy. Alimentary System. 38th ed; Churchill Livingstone. 1995, 1775-76.
9. Schwarts S.J, Shires G.T, Spencer F.C, Dally J.M, Fisher J.E and Galloway A.C: Schwarts Principles of Surgery. The Appendix. 7th ed; McGraw-Hill. 1999; 3:1383-85.
10. Buschard, K and Kjaelguard A: Investigation and analysis of the position, length and embryology of the vermiform appendix. Acta Chirugica Scandinavica. 1973; 139 (3):293-98.
11. Bodetti R.K, Kulkarni R and Murudhar P.K.H: Unique 28 cms long vermiform appendix. Int. J. Anat Res 2013, 1 (2): 111-114.
12. Zinner M.J, Schwartz S.I and Ellis H: Maingot's Abdominal Operations. Appendix and Appendicectomy. 10th ed. Vol.2; Appleton & Lange. 1997; 1190-1193.
13. Addiss D.G, Shaffer N, Fowler B.S.F and Tauxe R.V: The epidemiology of appendicitis and appendicectomy in the United States. American Journal of Epidemiology 1990, 132; 910-925.
14. Bakheit M.A and Warille A.A: Anomalies of the vermiform appendix and prevalence of acute appendicitis in Khartom. East African Medical Journal. (1999), 16(6): 338-340.
15. Ajmani M.L and Ajmani K: The position, length and arterial supply of vermiform appendix. Anatomischer anzeiger. 1983; 153 (4): 369-374.
16. Garg K: B. D. Chaurasia's Human Anatomy Regional and Applied Dissection and Clinical. 4th ed. CBS Publishers. 2004, 2; p. 257.

ORIGINAL RESEARCH ARTICLE

17. Anderson R.E, Houganer A and Thalin A.J.D: Diagnostic accuracy and perforation rate in appendicitis: Association with age and sex of the patients and with appendicectomy rate. European Journal of Surgery. 1992; 158: p37-41.

AUTHORS:

1. Sanjay Kumar Sinha
2. Vipin Kumar

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Anatomy, Katihar Medical College, Katihar, Bihar.
2. Assistant Professor, Department of Anatomy, Lord Buddha Koshi Medical College, Saharsa, Bihar.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sanjay Kumar Sinha,
Associate Professor,
Department of Anatomy,
Katihar Medical College,
Katihar – 854105, Bihar.
E-mail: dr.s.k.sinha.2709@gmail.com

Date of Submission: 22/07/2014.
Date of Peer Review: 23/07/2014.
Date of Acceptance: 22/08/2014.
Date of Publishing: 02/09/2014.

ORIGINAL ARTICLE

VARIATIONS IN DISTRIBUTION PATTERNS OF CORONARY ARTERIES IN CADAVERIC HEART

Sanjay Kumar Sinha[1], Vipin Kumar[2], Madhu Sinha[3], Pramod Kumar Agrawal[4]

HOW TO CITE THIS ARTICLE:
Sanjay Kumar Sinha, Vipin Kumar, Madhu Sinha, Pramod Kumar Agrawal. "Variations in Distribution Patterns of Coronary Arteries in Cadaveric Heart". Journal of Evidence based Medicine and Healthcare; Volume 1, Issue 12, November 24, 2014; Page: 1511-1517.

ABSTRACT: The heart is supplied by two coronary arteries and their branches. Variations occur occasionally in their origin and patterns of distribution. The coronary circulation and its collateral circulation has long been a topic of interest. The branches of the coronary arteries are generally considered to be functionally end arteries. However anastomoses do exist between branches of coronary arteries on the surface of the heart and between these arteries and extra cardiac vessels. These collaterals may or may not be well developed in individuals with normal cardiac anatomy. Vulnerability of an individual to develop coronary collaterals may characterize the myocardial susceptibility hence knowledge of branching pattern and mechanisms involved in formation of collaterals may aid in the treatment of coronary heart diseases.

KEYWORDS: Heart, Coronary arteries, Variations, Anastomosis, Distribution.

INTRODUCTION: Heart is a pair of valved muscular pumps combined together in a single organ.(1) The heart needs an uninterrupted supply of nutrition and oxygen throughout life due to increased susceptibility of the myocardium to ischaemia. The heart is supplied by the two coronary arteries and their multiple branches. The veins that drain the myocardium do not have names that corresponds to arteries in this respect the heart resembles the brain.(2) The arterial supply of heart is provided by the right and left coronary arteries which arise from the ascending aorta immediately above the aortic valve. The coronary arteries and their major branches are distributed over the surface of heart lying within subepicardial connective tissue.(3) The importance of knowledge of the course and branches of both coronary arteries has increased considerably in recent years as these arteries are often visualized both diagnostically and therapeutically in living during coronary angiography.

The pattern of branching of coronary arteries shows considerable variations.(4) The right coronary artery (RCA) arises from the anterior aortic sinus, passes anteriorly and to the right between the right auricle and the pulmonary trunk and descends vertically in the coronary sulcus between right atrium and right ventricle. Near its origin, the RCA gives off sinoatrial nodal branch. The RCA then descends in the atrioventricular groove and gives off the right marginal branch. Then the RCA on reaching the inferior margin of the heart turns to left and continues in the coronary sulcus on to diaphragmatic surface and base of heart. Here it gives off the atrioventricular nodal branch at the crux of heart and a final major branch the posterior interventricular branch which lies in the posterior interventricular sulcus.

Typically the RCA supplies the right atrium, most of right ventricle, part of left ventricle (diaphragmatic surface), part of interventricular septum, sinoatrial node and the atrioventricular node. The left coronary artery (LCA) originates from left posterior aortic sinus and passes between the pulmonary trunk and left auricle and runs in the coronary sulcus. While still posterior to the pulmonary trunk, the artery divides into its two terminal branches, the anterior interventricular branch (left anterior descending artery) and the circumflex branch.

The anterior interventricular branch continues and descends obliquely towards the apex of heart in the anterior interventricular groove and turns around the inferior border of heart and commonly anastomoses with the posterior interventricular branch of the right coronary artery. Along its course it gives off one or two diagonal branches. The smaller circumflex branch of the LCA runs in the coronary sulcus around the left border of heart to reach the posterior surface of the heart. A large branch the left marginal artery usually arises from it and continues across the rounded obtuse margin of heart.

Typically the LCA supplies to the left atrium, most of the left ventricle, part of right ventricle, and most of interventricular septum including the atrioventricular bundle of His and its branches. Variations in branching patterns and distribution of coronary arteries are common. The most common variant is right dominant pattern, present in approximately 67% of people where the posterior interventricular branch arises from the RCA. In about 15% of people the LCA is dominant, where posterior interventricular branch arises from the left circumflex branch of LCA. Apart from right and left coronary dominance, there is balanced coronary circulation.[5]

Anastomosis between right and the left coronary arteries are abundant during foetal life, but are much reduced by the end of first year of foetal life. Anastomoses providing collateral circulation may become prominent in conditions of hypoxia and in coronary artery diseases.[6] The potential for development of collateral circulation probably exists in most of heart if not all hearts. The anterior interventricular branch ends in terminal anastomosis with posterior interventricular branch of right coronary artery.

MATERIALS & METHODS: The present study was conducted in the Department of Anatomy, Katihar Medical College, Katihar, Bihar over a period of 5 years during which 30 specimens from both sexes of intact heart with great vessels were dissected and removed from the adult cadavers during routine dissection. The specimens were washed in running water to remove coagulated blood clots and were fixed in 10% formalin. Photograph of each specimen was taken prior to injection of Eosin solution (Fig. 1). Eosin solution was injected into each coronary artery by polythene cannulae under constant pressure. The pressure was maintained in order to prevent rupture of small vessels and for optimum penetration of eosin solution.

Fig. No 1: Intact Cadaveric Heart Prior to Injection of Eosin Solution.

Fig. 1

OBSERVATION: Both coronary arteries were observed to arise from the root of the aorta and were seen to course subepicardially in the atrioventricular and interventricular grooves and the RCA was seen to be arising from the anterior aortic sinus and its branches originated at about right angles (Fig. 2). The RCA terminated on the diaphragmatic surface of the heart with two or more branches descending in or parallel to the interventricular sulcus towards the apex cordis.

Fig. No 2: AO=Aortic Orifice, LCA= Left Coronary Artery & RCA= Right Coronary Artery.

Fig. 2

Almost in all cases the LCA was short and normally had no branches proximal to its bifurcation. The left descending branch is a continuation of the left main coronary trunk which was seen to course in the anterior interventricular sulcus up to the apex cordis. The left circumflex coronary artery was seen to originate from the LCA at right angles (Fig. 3). 2 out of 30 examined hearts (6.6%) had a third coronary artery (TCA) (Fig.4). They were seen to arise from the right aortic sinus and their orifices in all cases were in front and to the left of the orifice of the right ventricle.

Fig. No. 3: Left Anterior Descending Branch of Left Coronary Artery.

Fig. 3

Fig. No. 4: 1 = Left Coronary Artery, 2= Right Coronary Artery, 3 = Third Coronary Artery

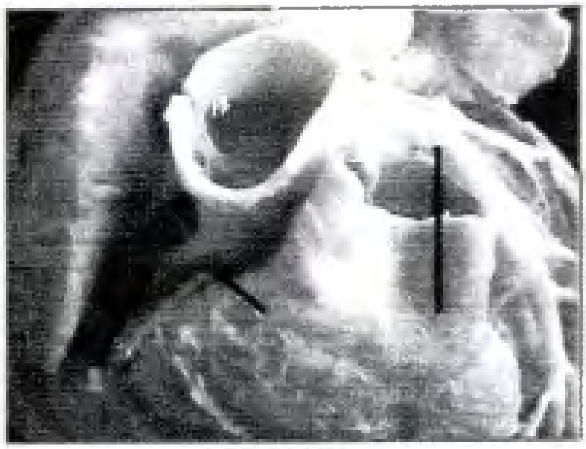

Fig. 4

Based on the distribution of coronary arteries and their branching patterns, 3 types of coronary circulations were considered. [A] Right dominant; [B] Balanced and [C] Left dominant. The RCA dominance was observed in 21/30 cases (70%) and in these cases, right coronary extended beyond the crux cordis. Balanced coronary artery circulation was observed in 6/30 cases (20%). LCA dominance was found in 3/30 cases (10%). In 20/30 cases (67%) ramus osticavaesuperioris was observed. It arose from the RCA in 12/20 cases (60%) and from LCA in 8/20 cases (40%). This vessel was not found to arise from both coronary arteries in same heart. The interventricular septum was observed to receive most of its blood supply from the left anterior descending branch.

In 22/30 cases (73%) the septal blood supply was provided by branches of left descending artery and 8/30 cases (27%) by branches from the posterior descending artery.

DISCUSSION: In regard to the classification of type of coronary circulation into right or left dominance and balanced circulation, in this study percentages were similar to those reported by Pino et al.[7] Freuqency of right dominance was lower than those reported by Blunk[8] and di Dio, Lima Junior et al[9] and Falci Junior et al.[10] In none of the hearts studied did the RCA and the circumfex branch of the LCA ended in parallel in posterior interventricular branches as reported before. The anterior interventricular branch ended in termino-terminal anastomosis with posterior interventricular branch of the RCA and in a frequency between those reported by Hadziselimovic[11] and Secerov and Cavalcanti et al.[12]

Data about the frequency of the third coronary artery is rather diverse, from 1.5% and 3% as stated by Lo and Kurija.[13, 14] In this study the incidence of TCA was found to be 6.6% which was consistent with the result received by Villanoga and Yamagishi.[15, 16] Hadziselimovic[17] points out that even three coronary arteries may arise independently from the right aortic sinus. In 15% cases trifurcation of coronary artery was present where TCA was known as median artery reported by Sinha P et al.[18] Literature describes cases with anastomosis of TCA with the anterior interventricular branch, diagonal branch, circumflex branch as well as with the branches of RCA. This study ascertained solely anastomosis of the TCA with anterior interventricular branch with formation of Vieussens arterial ring.

This artrial ring may be found by conal branch of RCA but it is less significant as it is more often enfolded with the atherosclerotic process. According to Miyazaki and Kato[19] the TCA may develop after birth. Observations of the present study has revealed that TCA gives 3 branches in 50% of the cases. In this study we also observed that the TCA extend epicardially to supply apex of the heart.

The right and left coronary ostia were present at the anterior and left posterior aortic sinuses respectively in all cases and there were no variations in the locations of the ostia. Sinha[20] et al has reported that in 5% cases two separate ostia were found for anterior interventricular and circumflex artery. As per the documented literature the S.A. node received special arterial supply. The present findings are in accordance of studies of James and Burch.[21]

These authors have pointed out that S.A. nodal artery is the largest atrial coronary branch which originates from the RCA or LCA. This study has confirmed the presence of main septal branches of the anterior and posterior descending arteries as described by such workers as Gross (1921),[22] James (1961),[23] Fulton (1965),[24] and Mitchell & Schwartz (1965).[25] The present findings are in agreement with their descriptions that the major part of the interventricular septum of all hearts is supplied by branches of anterior descending artery. In this study RCA dominance was 70% and LCA dominance was 10% and balanced dominance was 20% which were similar to those reported by Pino at al.[26]

CONCLUSION: In all the cadaveric hearts the coronary arteries arises from the aortic sinus. The incidence of third coronary artery holds importance in clinical practice as sometimes atherosclerotic lesions are found in this vessel. The results shows that pattern of coronary supply along with anastomosis are present in the heart with some variations. The distribution was more in favour of right coronary dominance. The coronary collateral blood flow may be an important protective response to acute and chronic Ischaemia. The distribution of S.A. nodal arteries allows understanding the possible aetiology of the Sinusal Nodal Syndrome and permits the surgeon a safe approach to cardiac disease. Caution should therefore be taken during surgical procedure along the anterior wall of the right ventricle and infundibulum since such a long TCA may present a surgical hazard.

REFERENCES:

1. Standring, S. Gray's Anatomy. The Anatomical Basis of Clinical Practice. Churchill Living Stone Elsevier, Spain, 40th ed.2008; 960-961.
2. Sinnatamby C.S. Last's Anatomy. Regional and Applied, Churchill Living Stone Elsevier, China, 12th ed. 2011; 204-205.
3. Snell, R.S. Clinical Anatomy by Regions. Lippincott Williams & Wilkins, Wolters Kluwer, South Asian Edition, India, 9th Edition, Second Indian Reprint, 2013; 86-87.
4. Singh, I. Textbook of Anatomy with Colour Atlas, Jaypee Brothers Medical Publishers, New Delhi, 4th ed. 2008; 978-979.
5. Pino, J.H. Disposicion de lasramasarterialesventriculares en corazones de individuoschilenos. An AnatNor 1987; (5):67-72.
6. Standring, S. Gray's Anatomy. The Anatomical Basis of Clinical Practice. ChurchillLivingstone Elsevier, Spain, 40th Edition, 2008; 978 -979.
7. Pino J.H. Disposicion de lasramasarterialesventriculares en corazones de individuoschilenos. An AnatNor 1987; (5):67-72.
8. Blumk, J.N. Types of coronary circulation in human hearts. Ohio State Med J, 1971 ;(67):596-607.
9. Lima, J. Tipos de circulacao e predominancia das arteriascoronariasemcoracoes de brasileiros. Rev Bras Cir Cardiovasc 1993; (8):9-19.
10. Junior, F. Estudo comparative do padrao de circulacocoronariana entre pecasanatomicas e pacientescirurgicos. Rev HospClinFac Med S Paulo 1996; (51):224-7.
11. Hadziselimovic, H, Seceriv, D: Superficial anastomosis of blood vessels in human heart. Acta Anat. 1979; 104: 268-78.
12. Cavalcanti, JS, Oliveira, ML. Melo JR, AVP, Balaban, G, Oliveira, CLA, Oliveiea, RL Contribuicaoaoestudo das variacoesanatomicas das arteriascoronarias. Arq Bras Cardiol 1995; 65:489-92.
13. Lo E.A., dia A., Ndiaye A., sow M.L.: Anatomy of coronary arteries. Dakar med. 1994.39(1):23-29.
14. Kurjia H.Z., Chaudhaey M.S., Olson T.R: coronary artery variation in a native Ieag population. Cathet. Caediovasc. Diagn 1986:12(6):386-390.
15. Vilallonga J.R: Anatomical variations of the coronary arteries: The most frequent variations. Eur.J.Anat. 2003;7(1): 29-41.
16. Yamagishi M., haze K., Tamai J., Fukami K., Beppu S., akiyama T., Miyatake K: Visualization of isolated conus artery as a major collateral pathway in patients with total left anterior descending artery occlusion. Cathet. Cardiovasc. Diagn. 1988; 15(2):95-98.
17. Hadziselimovic, H, Seceriv, D: Superficial anastomosis of blood vessels in human heart. Acta Anat. 1979; 104: 268-78.
18. Sinha P., Mehrotra N., Jethani S.L., Bindal U., Histo- Morphological study of coronary artery. J.Anat. Soc. India 58(2) 166-168 (2009).

19. Miyazaki, M. & Kato, M: Third coronary artery: its development and function. Actacardiol, 43(4): 449-57; 1988.
20. Sinha P., Mehrotra N., Jethani S.L., Bindal U., Histo- Morphological srudy of coronary artery. J.Anat. Soc. India 58(2) 166-168 (2009).
21. James, T.N.(1961): Anatomy of the human Sinus node. Ant.Rec., 141, 109.
22. Gross, L. (1921). The blood supply of heart. Oxford university press, London.
23. James, T.N.(1961): Anatomy of the coronary arteries. Hober. New York.
24. Fulton, W.F.M. (1965). The coronary Arteries. Thomas Springfeild, Illinois.
25. Mitchrll, J.R.A., and Schwartz, C.J. (1965). Arterial Disease. Blackwell, Oxford.
26. Pino, Jh, Riffo, EO, Vargas, JE. Disposicion de lasramasarterialesventriculares en corazones de individuoschilenos. An AnatNor 1987; 5:67-72.

AUTHORS:

1. Sanjay Kumar Sinha
2. Vipin Kumar
3. Madhu Sinha
4. Pramod Kumar Agrawal

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Anatomy, Katihar Medical College, Katihar, Bihar.
2. Assistant Professor, Department of Anatomy, Lord Buddha Koshi Medical College, Saharsa, Bihar.
3. Medical Officer, Department of Health, Madhubani, Bihar.
4. Professor and HOD, Department of Medicine, Katihar Medical College, Katihar, Bihar.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sanjay Kumar Sinha,
Associate Professor,
Department of Anatomy,
Katihar Medical College,
Katihar-854105, Bihar.
E-mail:dr.s.k.sinha.2709@gmail.com

Date of Submission: 18/10/2014.
Date of Peer Review: 20/10/2014.
Date of Acceptance: 11/11/2014.
Date of Publishing: 20/11/2014.

ORIGINAL ARTICLE

OBSERVATION ON ARRANGEMENT OF HILAR STRUCTURES IN CADAVERIC KIDNEYS AND THEIR CLINICAL SIGNIFICANCE

Sanjay Kumar Sinha[1], Nafees Fatima[2], Ghazala Perween[3], Madhu Sinha[4]

HOW TO CITE THIS ARTICLE:
Sanjay Kumar Sinha, Nafees Fatima, Ghazala Perween, Madhu Sinha. "Observation on Arrangement of Hilar Structures in Cadaveric Kidneys and their Clinical Significance". Journal of Evidence based Medicine and Healthcare; Volume 1, Issue 17, December 29, 2014; Page: 2153-2155.

ABSTRACT: Hilum of an organ is a depression, pit or slit like opening through which vital structures enter or leave the organ. In addition to the kidney, hilum is also observed in the cerebellum, lung, ovary, spleen and suprarenal gland. Laparoscopic nephron-sparing surgery for solid renal masses can be achieved successfully both transperitoneally and retroperitoneally if a comprehensive knowledge of both normal and variant hilar anatomy of the kidneys is in the mind of the operating surgeon. Documented text is available on various aspects of the kidneys but an observation on variations in hilar arrangement is infrequently cited. In standard text from anterior to posterior the structures at the renal hilum are renal vein, renal artery and the renal pelvis.
KEYWORDS: Hilum, Kidneys, Arrangement, Variant, Anatomy.

INTRODUCTION: The kidneys are a pair of reddish brown retroperitoneal organs which secrete the end products of metabolism and excess water. They perform a dual role by maintaining water and electrolyte balance and releasing erythropoietin which affects red blood cell formation and renin which influences blood pressure. In the foetus and newborn, the kidney normally has 12 lobules. These are fused in adults to present a smooth surface although traces of lobulation may remain.[1] The normal kidney measures about 12x6x3 cm and weighs 130-150 g. The hilum of the right kidney lies just below, and of the left just above, the transpyloric plane 5 cm from the midline.[2] The hilum of each kidney lies over psoas. The hilum of the kidney leads into a central sinus, lined by the renal capsule and almost filled by the renal pelvis and vessels, the remaining space being filled by fat. Dissection into this plane can be challenging but is important in surgery on the renal pelvis, particularly open stone surgery.[3] For many operations on the kidney including nephrectomy and nephrolithotomy a lumbar approach is used. The renal vessels can be exposed, ligated and divided to mobilize the organ further and transect the ureter.[4] Clamping of individual structures at the hilum is preferred rather than en-bloc clamping.[5] Knowledge of structures at the renal hilum is thus necessary prior to any surgical intervention of the kidney.[6] The arrangement of structures at the renal hilum, anteroposteriorly as described in standard anatomical texts is: renal vein, renal artery and renal pelvis. A systematic study with this specific focus has seldom been reported in literature.

MATERIAL & METHOD: The present study was undertaken to observe the arrangement of structures in hilar regions. 40 (20 Right [R] and 20 Left [L]) embalmed cadaveric kidneys from the Department of Anatomy were observed in this study. Hilar dissection was carried out to observe the arrangement of structures entering or leaving the hilum of the kidney. Anteroposterior relations of the structures at the hilum were also recorded. The arrangement of renal artery, renal vein and the renal pelvis, anteroposteriorly exhibited noticeable variations in their hilar relations. In majority of the cases renal artery divided into anterior and posterior divisions. In few cases anterior trunk of renal artery is the most anteriorly placed structure at the renal hilum. In some cases retropelvic structures are observed at the hilum.

OBSERVATIONS: The following types of structural arrangements at the renal hilum were observed. They are A, B, C & D respectively.

ANTERIOR			
Type A	Type B	Type C	Type D
RV RA RP	RV AD of RA PD of RA RP	AD of RA RV PD of RA RP	AD of RA RV RP PD of RA
N=24/40 R=14/40 & L=10/40	N=08/40 R=05/08 & L=03/08	N=05/40 R=03/05 & L=02/05	N=03/40 R=02/03 & L=01/03
POSTERIOR			

Table 1: Arrangement of structures at the renal hilum from anterior to posterior

Legends: A=Artery, AD=Anterior Division, PD=Posterior Division, P=Pelvis, R=Renal, V=Vein.

DISCUSSION: In our study we observed the standard textbook arrangement of hilar structures in 60% specimens. The remaining 40% specimens showed variations. In 20% specimens we observed anterior and posterior divisions of the renal artery occupying the bulk of the hilum whereas in 12.5% specimens the renal vein was sandwiched between the two divisions of the renal artery. Only in 7.5% specimens we observed the retro pelvic occurrence of posterior division of the renal artery. It may be noted that none of the variants have been mentioned in textbooks of Anatomy but have only been encountered during routine cadaveric dissections. Embryo logically the right renal vein develops from a single anastomotic channel whereas the left renal vein develops from multiple anastomotic channels. Hence the predominant left sided occurrence may have an embryological explanation. Developmental malformations may change the interrelationship of the hilar structures. Surgical interventions that require hilar dissections are technically more challenging in laparoscopic approach as compared to open surgeries.

CONCLUSION: Knowledge of anatomical variations at the renal hilum assumes clinical importance for operating surgeons as laparoscopic surgeries have become the treatment of choice in recent advancement. Bulk of the hilum is occupied by branches of renal arteries and tributaries of renal veins. Variations are predominant on the left side and presence of retropelvic structures should be kept in mind during renal manipulation.

ORIGINAL ARTICLE

REFERENCES:

1. Standring S. Gray's Anatomy, The Anatomical Basis of Clinical Practice. 40th Ed. London: Churchill Livingstone Elsevier; 2008. Chapter 74, Kidney and Ureter; p1225.
2. Sinnatamby CS. Last's Anatomy Regional and Applied. 12th Ed. China: Churchill Livingstone Elsevier; 2011. Chapter 5, Abdomen; p283.
3. Standring S. Gray's Anatomy, The Anatomical Basis of Clinical Practice. 40th Ed. London: Churchill Livingstone Elsevier; 2008. Chapter 74, Kidney and Ureter; p1230.
4. Sinnatamby C. S. Last's Anatomy Regional and Applied. 12th Ed. China: Churchill Livingstone Elsevier; 2011. Chapter 5, Abdomen; p286.
5. Rapp, DE. En bloc stapling of renal hilum during laparoscopic nephrectomy and nephroureterectomy. Urology 2004; 64(4):655-659.
6. Gill, IS. Laparoscopic partial nephrectomy for hilar tumours. J Urol 2005; 174(3):850- 854.
7. Lattouf, JB. Laparoscopic partial nephrectomy for hilar tumours: technique and results. Eur. Urol. (2008); 54(2):409-416.
8. Rogers, CG. Robotic partial nephrectomy for renal hilar tumours: a multi-institutional Analysis. J Urol 2008; 180(6):2353-2356.

AUTHORS:

1. Sanjay Kumar Sinha
2. Nafees Fatima
3. Ghazala Perween
4. Madhu Sinha

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Anatomy, Katihar Medical College, Katihar, Bihar.
2. Assistant Professor, Department of Anatomy, G.C.R.G. Institute of Medical Sciences, Lucknow, Uttar Pradesh.
3. Junior Resident, Department of Anatomy, Katihar Medical College, Katihar, Bihar.
4. Medical Officer, Department of State Health Services, Madhubani, Government of Bihar.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sanjay Kumar Sinha,
Associate Professor,
Department of Anatomy,
Katihar Medical College,
Katihar-854105, India.
E-mail: dr.s.k.sinha.2709@gmail.com

Date of Submission: 18/12/2014.
Date of Peer Review: 19/12/2014.
Date of Acceptance: 20/12/2014.
Date of Publishing: 24/12/2014.

ORIGINAL RESEARCH ARTICLE

CADAVERIC STUDY ON ARTERIAL PATTERNS OF VERMIFORM APPENDIX

Sanjay Kumar Sinha[1], Vishal Parmar[2], Jay Prakash Bharti[3]

HOW TO CITE THIS ARTICLE:
Sanjay Kumar Sinha, Vishal Parmar, Jay Prakash Bharti. "Cadaveric Study on Arterial Patterns of Vermiform Appendix". Journal of Evidence Based Medicine and Healthcare; Volume 1, Issue 5, July 2014; Page: 288-290.

ABSTRACT: Appendicectomy is a common surgical procedure worldwide and it refers to the surgical removal of the vermiform appendix for therapeutic reasons. This surgical procedure when performed classically or laparoscopically requires thorough knowledge of surface, gross and vascular anatomy of the vestigial organ. Deficiency in knowledge of variations in blood supply of the appendix can prove to be a problem for the operating surgeon.
KEYWORDS: Vermiform appendix, Appendicectomy, Vestigial organ.

INTRODUCTION: The appendicular artery is normally a branch of the inferior division of the ileocolic artery, which runs behind the ileum to enter the mesoappendix.[1] In its course it gives off a recurrent branch which anastomoses with a branch of the posterior caecal artery. The artery enters the mesoappendix a short distance from the appendicular base.[2] The appendicular artery traverses the free margin of the mesoappendix and tends to approach the tip of the organ. As the mesoappendix does not continue upto the tip of the appendix, the artery now lies in direct contact with the appendicular tip. It must be remembered that the appendicular artery is an end artery.[3] Accessory arteries are common and many individuals possess two or more arteries of supply to the appendix. Accessory appendicular artery which is usually a branch of posterior caecal artery supplies the appendicular base. Damage to this artery can lead to significant intra-operative and postoperative hemorrhage and should be searched for carefully and ligated once the main appendicular artery is controlled.[4]

MATERIALS AND METHODS: During routine cadaveric dissections performed in the Department of Anatomy, Katihar Medical College, Katihar, fifty specimens of composite viscera of caecum, appendix, last two inches of ileum and first two inches of ascending colon were dissected and removed with intact blood vessels. The specimens were washed under running tap water and were fixed in 10% formalin. The ileocolic artery along with its branches to the appendix was traced and variations in arterial patterns of the appendix were recorded.

OBSERVATIONS: Observations are presented in tabular form.

Group	Feature	No.	Percentage
A	Single AA branching from inferior division of ileocolic artery	28/50	56%
B	Single AA branching directly from ileocolic artery	12/50	24%
C	Accessory AA present	10/50	20%

Table 1

AA = Appendicular Artery

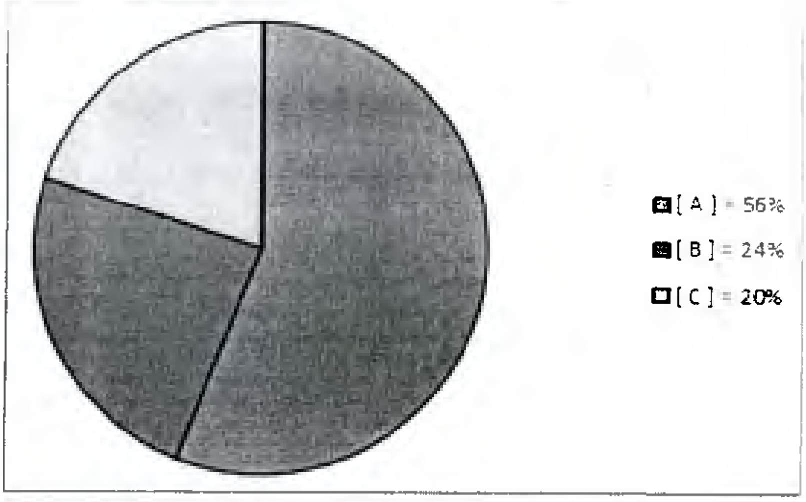

Fig. 1: Pie chart depicting the findings in percentage

DISCUSSION: Out of the 50 specimens observed, 28 specimens (56%) revealed the presence of a single appendicular artery which branched from the inferior division of ileocolic artery. This is a normal finding and has been documented previously by Cunningham[5] and Standring.[2] In 12 specimens (24%) a single appendicular artery which branched directly from the ileocolic artery was observed. This type of origin has also been described by Haller.[6] 10 specimens (20%) showed the presence of accessory appendicular arteries. Accessory appendicular arteries have been previously mentioned by Anson[7]and Das.[3] In this study, out of the 10 accessory appendicular arteries 8 had originated from the posterior caecal artery and 2 had branched from the common caecal artery. Accessory appendicular arteries branching from the posterior caecal artery has been reported by Bergmann.[8]

CONCLUSION: Normally the appendicular artery originates from the inferior division of the ileocolic artery but it may also originate directly from the ileocolic artery. Variations in its origins and in particular knowledge of accessory appendicular arteries should be kept in mind by the operating surgeons to avoid complications during appendicectomy.

REFERENCES:

1. Sinnatamby C.S, Last's Anatomy Regional and Applied; Churchill Livingstone; 11th Edition; Chapter 5; Part 6, pp266.
2. Standring S. Gray's Anatomy The Anatomical Basis of Clinical Practice. Churchill Livingstone; 40th Edition; Section 8; Chapter 67; 2008. pp 1144-1145.
3. Das S. A Concise Textbook of Surgery. Das Publishers. 6th Edition; 2010; pp 904.
4. Liu C.D, McFadden D.W, Acute Abdomen and Appendix. Surgery: Scientific Principles and Practice; Williams & Wilkins; 2nd Edition; 1997; pp 1246-1261.

5. Romanes G.J, Cunningham's Textbook of Anatomy. 12th Edition; Oxford University Press; 1981; pp 926-927.
6. Haller V. Icones of Haller: 1756.
7. Barry JA. Morri's Anatomy. McGraw-Hill Book Company; 12th Edition; 1966; pp 744.
8. Volker S. Appendix and Caecum Embryology, Anatomy and Surgical Applications. The Surgical Clinics of North America. Feb. 2000; 80(1): pp 301-302.

AUTHORS:

1. Sanjay Kumar Sinha
2. Vishal Parmar
3. Jay Prakash Bharti

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Anatomy, Katihar Medical College.
2. Assistant Professor, Department of General Medicine, Katihar Medical College.
3. Tutor, Department of Anatomy, Patna Medical College.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sanjay Kumar Sinha,
Associate Professor,
Department of Anatomy,
Katihar Medical College,
Katihar - 854105, Bihar.
E-mail: dr.s.k.sinha.2709@gmail.com

Date of Submission: 12/07/2014.
Date of Peer Review: 14/07/2014.
Date of Acceptance: 16/07/2014.
Date of Publishing: 18/07/2014.

Current Neurobiology 2010, 1 (1): 25-29

Comparative Light Microscopic Study of Trigeminal Ganglion Neurons in Mammals

M. Naushad A Dilkash, Syed Sayeed Ahmed* and Aijaz Ahmed Khan

Dept. of Anatomy, JN Medical College, Aligarh Muslim University, Aligarh, India
*Dept. of Oral and Maxillofacial Surgery, Dr. Z A Dental College, AMU, Aligarh, India.

Abstract

Trigeminal ganglion (TRG) consists of collection of primary sensory neurons. The different subsets of neurons have been identified on the basis of morphological and neurochemical characteristics. It remains to be resolved as to whether the various neuronal subsets remain alike across the mammalian species and if there exists some species specific characteristic neurons. The present study was conducted on adult mammals (rat, rabbit, and goat) of either sex. TRG of both sides were procured and fixed in 10% buffered formalin and processed for paraffin embedding. 10 µm thick sections stained with Haematoxylin and Eosin were examined under light microscope and relevant findings were recorded in photomicrographs. It was noticed that the main cellular constituents (neuron and glia) of TRG could be easily identified and features of most of the neurons matched with earlier light microscopic descriptions [1, 2]. However, few neurons in the present study revealed certain additional features. For example – in the medium size neuron, large Nissl granules formed single peripheral ring; in the medium and large sized neurons, coarse Nissl granules formed two concentric (perinuclear and peripheral) rings; and a couple of neurons appeared to share common sheath – a kin to binucleate neurons. In addition, the neuronal somatic size appeared to have direct relationship with the body size of the animal. The number of nucleoli and Cajal bodies per neuron and the number of cells involved in the formation of satellite glial cell (SGC)-sheath could be correlated with the size of neuronal somata. It was concluded that the neuronal subgroups of mammalian TRG remain fairly similar across the species. However some less common neurons with single and double rings of coarse Nissl granules need suitable categorization with respect to their neurochemical and functional characteristics.

Key words: Trigeminal Ganglion, sensory neuron, Nissl body, Cajal body, satellite glial cells.

Accepted July 01 2009

Introduction:

Like many other sensory ganglia associated with the cranial nerves, TRG also consists of collection of primary sensory neurons but unlike others, part of its primary sensory neurons in addition are located in the central nervous system as mesencephalic nucleus of trigeminal nerve [3]. It has similarity with the dorsal root ganglia (DRG) in being commonly affected by herpes virus [4, 5] resulting into postherpetic neuralgic pain syndromes and therefore, becoming the focus of large number of studies. Like DRG [6], TRG is also known to consist of different subsets of neurons e.g., two types of neurons based on staining intensity (dark and light) [2]; three types of neurons based on somatic size [7,3], and four types of neurons based on characteristics of Nissl granules [1] have been described. Type I-large neurons have diffusely distributed fine granules, type II-neurons contain coarse sparsely distributed Nissl granules, type III- neurons possess dense Nissl granules of varying size, and type IV-small neurons depict granules concentrated peripherally [1]. In cat the cell innervating meninges are said to be smaller than average [8]. Immunocytochemical studies have shown that to some extent certain morphological subsets of TRG neurons are positive for specific neurotransmitter or its receptors e.g., large TRG neurons are positive to NPY and Peptide-9 [3] and small to medium sized neurons are positive to substance-P, NKA, CGRP [3], NADPH diphorase [9], BDNF ([10]. However, while substance-P positive neurons may be small, medium or large [11] and GABA positive neurons are primarily large but may be medium or small [7] and Glutamate positive neurons are primarily small to medium size but may also be large [7]. In case of Galanin, changing pattern of intensity and somatic sizes of neurons was reported during the course of healing [12]. Four types of neurons based on Nissl granule characteristics [1] have further been defined electron microscopically by length and arrangement of flattened cisterns of granular endoplasmic reticulum and number of neurofilaments Type I- largest and type IV smallest. Type III and type IV lack neurofilaments. Seven types of neurons based on size, shape and electron density of the neuronal somata have been described [13]. It still remains to be resolved as to what extent the light microscopic features of TRG neuronal heterogeneity (existence of subsets) are maintained across the mammalian species and if there exists some species specific characteristic features of neurons in TRG.

Figure 1. Rat TRG: Cluster of neurons along with SGCs among fascicles of nerve fibres (Nf). Note typical neuron with single nucleolus and three Cajal bodies (1); single nucleolus, three Cajal bodies and Nissl granules in two concentric rings (2); medium sized neuron with double nucleoli (3). H & E; X 400.

Material and Methods

In the present study TRG from both sides of 5 adult healthy animals of either sex (rat, rabbit, and goat) were included. Those from rat and rabbits were procured by euthanising the animals with over dose of general anaesthesia, followed by fixation with intracardiac perfusion of 10% buffered formalin. TRG from goat were collected from slaughter house within 15 to 20 minutes of sacrifice of the animals and was immersion fixed in 10% buffered formalin. TRG from each animal were processed separately for paraffin embedding. 10 µm-thick sections cut with the rotary microtome and stained with Haematoxylin and Eosin were observed under light microscope. Salient findings were recorded in photomicrographs taken at final magnification of X400 and X1000 (Olympus BX40).

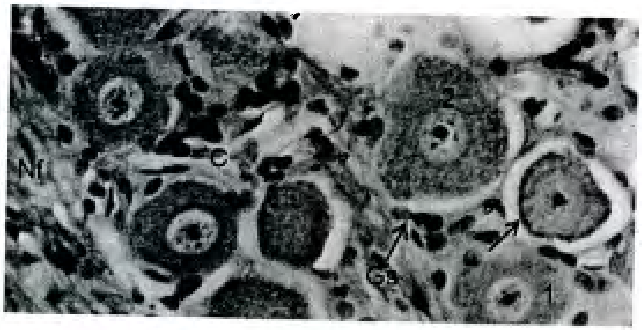

Figure 2. Rabbit TRG: Neurons of different size interspersed among nerve fascicles (Nf) which is permeated by capillaries (C). Large Nissl granules forming prominent peripheral ring (↑), neurons 1, 2, and 3 have Cajal bodies. Also note the marked perinucleolar halo in neuron -2; and satellite glial cells (Gs) forming sheath. H & E X400.

Observations

The TRG contained two main types of cells namely neurons and glia. Neurons were larger and more prominent and less numerous while glia cells were smaller in size, more numerous and surround each neuron. Clusters of neurons were interspersed among nerve fascicles (Fig. 1 and Fig. 2). In cross sections, the nerve cell clusters appeared to assume different size and shapes. Almost all nerve cell bodies were circular to oval in outline and revealed a wide range of size variation both in an individual and across the species. The neuronal somatic sizes in rat, rabbit and goat ranged from 12 to 30 µm, 15 to 75 µm and 25 to 120 µm respectively. Each nest of neurons possessed cells of different sizes and there appeared to be no specific pattern in their manner of arrangement. In all animals, most of the neurons were lightly stained with some small and medium sized neuron took dark staining. Neuronal cell was characterized by large centrally placed euchromatic, vesicular nucleus and prominent nucleolus. The number of nucleoli per neuron ranged from 1-3 (Fig. 1). In addition some neurons depicted variable number (1-5) of Cajal bodies of varied size, staining intensity and arrangement with respect to the nucleolus (Figs. 1, 2, 4) and are believed to reflect a distinct transcription-dependent organization of nucleolus splicing machinery of TRG neurons [17]. Occasionally, a perinucleolar halo could also be visualized (Fig. 2, neuron-2). The perikaryon was filled with Nissl substance which assumed different appearance in terms of its overall amount, distribution pattern, size of granules, and intergranular space. Features of most of the TRG neurons matched with light microscopic features mentioned in the literature [1, 2]. However, few neurons in the present study revealed certain additional interesting features. For example – in the medium sized neuron, large Nissl granules formed single peripheral ring while fine granules occupied rest of the soma (Fig. 2), and medium and large sized neurons, coarse Nissl granules were arranged in the form of two concentric (perinuclear and peripheral) rings (Fig. 1). Normally, each neuron was surrounded by SGCs which actually formed a sheath and thus each individual neuron along with its SGCs could be identified as isolated units. But sometimes two medium sized or small neurons were placed so closely that part of SGC-sheath between adjacent neurons remained invisible even at higher magnification and it appeared that both neurons shared a common sheath (Fig. 3) or else represented a case of binucleated sensory neuron. As seen in the cross section, the number of SGCs involved in the formation of perineuronal sheath varied with the size of neuronal cell body ranging from just 3 around small neuron to 25 around large neurons. In other words, number of glial cells taking part in the formation of SGC-sheath seems lower in rats as compared to rabbit and goat. The cytoplasm of SGC was revealed only occasionally(Fig. 4).

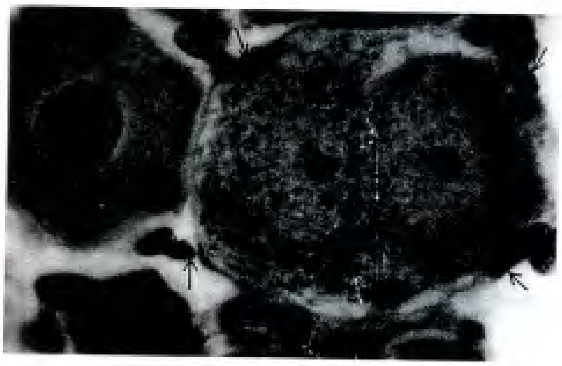

Fig. 3. *Rabbit TRG: Three neurons with their SGC-sheaths. Two neurons within common sheath (small arrows). No trace of connective tissue between two neurons (vertical long and thick arrow) thus giving appearance of a binucleate neuron. H & E; X 1000.*

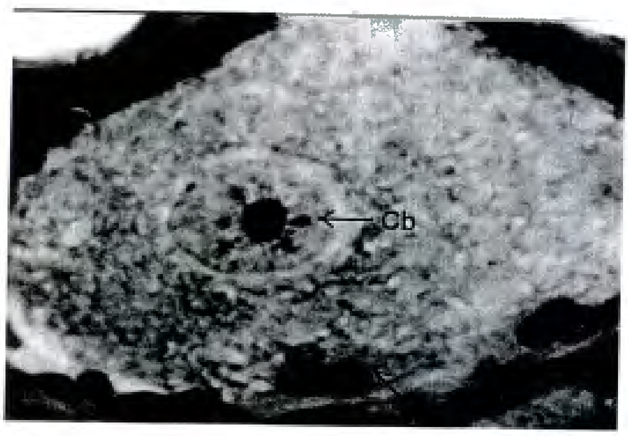

Fig. 4. *Goat TRG: Large oval neuron. Uniformly distributed Nissl granules, Single prominent nucleolus and five Cajal bodies (Cb). Perineuronal glial cell sheath (Gs) can easily be identified. H & E; X 400.*

Discussion

Sensory neurons in TRG are structurally, functionally and neurochemically heterogeneous [1,2,3,7,9,10,11,12,13]. In the present study only light microscopic morphological criteria have been taken for identification of different subsets of TRG neurons. The features of almost all TRG neurons fell within one or the other e.g., dark and light [2]; type I, II, III and IV [1]. However, few neurons revealed features which did not match with those described previously (Fig 1, 2 and 3). Neuron -2 in figure 1 is large and possesses double rings of coarse Nissl granules-and hence different from those describe earlier. The neuron in fig. 2 with single ring of coarse Nissl granule is akin to type IV neuron [1] but here it is of medium size neuron rather than small size. Thus these neurons present certain atypical features analogous to those described recently in the cervical DRG neurons of rabbit [15]. How these minor features can be related with some known characteristics of certain subsets of neurons in health and disease remains to be resolved.

In one study [7] it was noticed that around 20-24 % of TRG neurons were GABA positive while 64-66% were glutamate positive and was speculatively concluded that these amino acids act not only as neurotransmitter but also act as neuromodulators of sensory information. In other study [3] it was noticed that neurons showing immunoreactivity with glutamate, substance P, neurokinin, CGRP, CCK and SOM had small and medium sized somata, while those with NPY, and Peptide-19 had large sized somata, but nitric oxide synthase and paralbumin immunoreactive cells fell within all somata sizes. Thus, because of only partial correlation between somatic size and neurochemical characteristics,. in the present study it will not be appropriate to comment on the neurochemical nature of these atypical neurons. Differential susceptibility of TRG neurons to herpes virus [16] and their subsequent atrophy or apoptosis [17 and demyelination of nerve fibres may be the morphological basis for the development of trigeminal neuralgia [18].

Santiago Ramón y Cajal described a new organelle in the nuclei of vertebrate neurons. When viewed with the electron microscope, these subnuclear 'organelles' appear to consist of a tangle of coiled threads and were hence also referred to as coiled bodies. Small Cajal body-specific RNAs (scaRNAs) are a class of small nucleolar RNAs (snoRNAs) which specifically localise to the Cajal body, a nuclear organelle involved in the biogenesis of small nuclear ribonucleoproteins (snRNPs) [19, 20]. In the present study these bodies are commonly noticed in large neurons of all species. Their number appears to be directly related to the size of neuronal somata e.g., their number is commonly 2-3 in rat and rabbit and may be 5-6 in case of goat (Fig. 4). Recent studies indicate that the Cajal body has numerous roles in the assembly and/or modification of the nuclear-transcription and RNA-processing machinery [21].

Neurons of TRG are anatomically isolated from one another and are not synaptically interconnected. And as such they are classically thought to function as independent sensory communication elements. In the present study a couple of neurons appear to share a common sheath formed by SGCs (Fig. 3). And the interneuronal sheath element though not visible cannot be ruled out with certainty and significance of such intimate association between certain neurons remains unclear. On one hand absence of interneuronal sheath element makes its appearance akin to a binucleate neuron, similar to one shown recently in DRG [15]. In DRG most neurons are transiently depolarized when axons of neighboring neurons of the same ganglion are stimulated repeatedly [22]. Such cross-depolarization contributes to mutual cross-excitation. Thus it appears that such type of intimate association (Fig. 3) may provide a suitable morphological substrate for the intraganglionic communication [22,23]. Although much less is known about SGCs in sensory ganglia, it appears that these cells share many characteristics with their central counterparts. Like Schwann cells, SGCs cytoplasm contains peroxisomes which may influence oxygen levels in the vicinity of perikarya [24], and in adult TRG responds by proliferation to the mitogenic protein from explanted sensory neuron [25]. In the present study the SGCs number seems to positively correlate with the size of neuronal cell somata and this is in agreement with the finding in other species showing the volume of SGC-sheath to be directly proportional to both the volume and surface area of the related neuronal cell body [26].

Conclusion

Basic neuronal subtypes in TRG are similar across the species. Neurons with single or double rings of coarse Nissl granules require suitable categorization with respect to their neurochemical and functional characteristics.

Acknowledgements

We acknowledge the co-operation of technical and other supporting staff, Department of Anatomy, JN Medical College, Aligarh Muslim University, Aligarh.

References

1. Sugiura Y, Kitoh J, and Sakai H: A comparative ultrastructural study of the trigeminal ganglion in the rat and chicken. J Morphol.1983; 175: 101-113.
2. Gaik GC, and Farbman AI: The chicken trigeminal ganglion I. An anatomical analysis of neuron. J Morphol. 2005; 141: 43-45.
3. Lazarov NE: Comparative analysis of the chemical neuroanatomy of the mammalian trigeminal ganglion and mesencephalic trigeminal nucleus. J Morphol. Prog. Neurobiol. 2002; 66: 19-59.
4. Bastian FO, Rabson AS, Yee CL, and Tralka TS: Herpes hominis: Isolation from human trigeminal ganglion. J Neurosci.1972; 6: 2215-2225.
5. LaGuardia JJ, Cohrs RJ, and Gilden DH: Prevalence of varicell-zoster virus DNA in dissociated human trigeminal ganglion neurons and nonneuronal cells. J Virol. 1999; 73: 8571-8577.
6. Rambourg A, Clermont Y, Beaudet A: Ultrastructural features of 6 types of neurons in rat dorsal root ganglia. J Neurocytol 1983; 12: 47-66.
7. Stoyanova I, Dandov A, Lazarov N, and Chouchkov C: GABA- and Glutamate-Immunoreactivity in sensory ganglia of cat: a quantitative analysis. Arch Physiol and Biochem. 1998;106: 362-369.
8. Steiger HJ and Meakin CJ: Meningeal representation in the trigeminal ganglion and experimental study in the cat. Headache: J of Head and Face Pain.2005; 24: 305-309.
9. Kolesar D, Kolsarova M, Schreiberova A, Lackova M and Marsala J (2006): Distribution of NADPH Diphorase-exhibiting primary afferent neurons in the trigeminal ganglion and mesencephalic trigeminal nucleus of the rabbit. Cellular and Mol. Neurobiol. 26: 1263-1277.
10. Quartu M, Serra MP, Bachis A, Lai ML, Ambu R, and Fiacco MD: Glial cell line-derived neurotrophic factor-like immunoreactivity in human trigeminal ganglion and nucleus. Brain Res. 1999; 847: 196-202.
11. Prosba-Mackiewicz M, Zoltowaska A and Dziewiatkowski J: Substance P of neural cells in human trigeminal ganglion. Folia Morphol. 2000; 59: 327-331.
12. Deguchi T, Yabuuchi T, Ando R, Ichikawa H, Sugimoto T and Takano-Yamamoto T: Increase of galanin in trigeminal ganglion during tooth movement. J. Dental Res. 2006; 85: 658-663.
13. Krastev D, Paloff A, Krastev N, Apostolov A, and Ovtscharoff W: Ultrastructure of trigeminal ganglion in human. J of IMAB- Annual Proceed (Sci papers) Book 1. 2008.
14. Pena E, Berciano MT, Fernandez R, Ojeda JL, Lafarga M: Neuronal body size correlates with the number of nucleoli and Cajal bodies, and with the organization of splicing machinery in rat trigeminal ganglion neurons. J Comp Neurol. 2001; 430: 250-263.
15. Khan AA, Dilkash MN, Khan MAand Faruqi NA: Morphologically atypical cervical dorsal root ganglion neurons in adult rabbit. Biomed Res 2009; 45-49.
16. Geenen K, Gavoreel HW, and Nauwynck HJ: Cell type-specific resistance of trigeminal ganglion neurons towards apoptotic stimuli. Vet. Microbiol. 2006; 113: 223-229.
17. Hufner K, Horn A, Derfuss T, Glon C, Sinicina I, Arbusow V, Strupp M, Brandt T, and Theil D: Fewer latent herpes simplex virus type 1 and cytotoxic T cells occur in the ophthalmic division than in the maxillary and mandibular divisions of the human trigeminal ganglion and nerve. J Virol, 2009; 83: 3696-3703.

18. Burchiel KJ, and Baumann TK: Pathophysiology of trigeminal neuralgia: new evidence from a trigeminal ganglion intraoperative recording: case report. J Neurosurg. 2004; 101: 872-873..
19. Carmo-Fonseca M. New clues to the function of the Cajal body: EMO report. 2002; 3: 726-727.
20. Darzacq X, Jady BE, Verheggen C, Kiss AM, Bertrand E, Kiss T: "Cajal body-specific small nuclear RNAs: a novel class of 2'-O-methylation and pseudouridylation guide RNAs". EMBO J. 2002; 21: 2746–2756.
21. Gall JG. Timeline: The centennial of the Cajal body. Nature Reviews Mol. Cell Biol. 2003; 4: 975-980.
22. Amir R and Devor M: Chemically mediated cross-excitation in rat dorsal root ganglia. J Neurosci. 1996; 16: 4733-4741.
23. Hanani M: Satellite glial cells in sensory ganglia: from form to function. Brain Res. Rev. 2005; 48: 457-476.
24. Citkowitz E and Holtzman E: Peroxisomes in dorsal root ganglia. Histochem. Soci Inc USA. 1973; 21: 34-41.
25. Wen JY, Morshead CM, and van der Kooy D: Satellite cell proliferation in the adult rat trigeminal ganglion results from the release of a mitogenic protein from explanted sensory neurons. J Cell Biol. 1994; 125: 1429-1435.
26. Lee KH, Chung K, Chung JM and Coggeshall RE.: Correlation of cell body size, axon size, and signal conduction velocity for individually labeled dorsal root ganglion cells in the cat. J Comp. Neurol 1986; 243: 335-346.

Correspondence:

Aijaz Ahmed Khan
Department of Anatomy
J.N. Medical College,
Aligarh Mulslim University
Aligarh-202002
India
e-mail: aijazahmedkhan7@live

Current Neurobiology 2010; 1 (1): 55-57

Effects of Lead on the Olfactory Bulb of the Adult Albino Rats

Naushad A Dilkash, Syed MD Husain, and Aijaz A Khan

Department of Anatomy, JN Medical College, Aligarh Muslim University, Aligarh, India

Abstract

Lead is virtually toxic to every organ of body including central nervous system where it may manifest as encephalopathy and hyposmia yet the exact mechanism of these clinical manifestations remains inconclusive. The present study was aimed to see the microscopic changes in the olfactory bulb of rat induced by oral administration of a lead compound in adult albino rats. A total number of 12 adult albino rats of either sex were included in the present study consisting of equal numbers in both control and experimental groups. Experimental group received 4% aquous lead acetate orally for a period of 3 weeks then animals of both groups were euthanized with overdose of general anaesthesia and perfused with 10% formalin. Olfactory bulbs were dissected out and processed for paraffin embedding. 10 µ-thick sections were stained with H&E and observed under light microscope. On gross examination brains from the experimental group revealed generalized edema and petechial haemorrhages. Histopathology of the olfactory bulbs revealed edema and congestion with vacuoles of variable sizes almost throughout. Distortion of glomeruli, clumping of periglomerular cells and increasing number of pyknotic cells were also noticed. It was concluded that lead has toxic effects on the central nervous system including olfactory bulb in the form of edema, microscopic hemorrhages and neuronal loss which may explain the clinical manifestations of lead toxicity.

Key words: Albino rats, Olfactory bulb, Lead acetate, Neurotoxicity, Edema, Haemorrhage

Accepted December 15 2009

Introduction

Exposure of lead can take place either through inhalation of dust, fumes, vapours, or ingestion of contaminated foods or drinks. Because of its cumulative property it is capable of exerting toxic effects at any level of exposure. Toxic effect of lead on the body is known as Plumbism which on the central nervous system manifests as encephalopathy that is accompanied by areas of focal cortical necrosis. Its clinical manifestation includes headache, incoordination, tremor, twitching, convulsion, paralysis, coma and death [1]. In the brain, cerebellum was found to be most severely affected [2]. Significant decrease in spine density [3] and reduction in the maximum width of the hippocampus [4] have also been reported. Bilaterally symmetrical spongiform changes in the roof nuclei of cerebellum [5] was also reported in dogs exposed to orally fed lead while bilaterally symmetrical areas of vacuole formation were observed at the tips of cortical gyri [6]. Other heavy metals like Cadmium dust induced anosmia [7] and in another study it was reported that inhalation of cadmium affected olfactory bulb and contributed to olfactory dysfunction [8]. Zinc gluconate trihydrate induced cellular and tissue damages to olfactory neuroepithelium and to mitral cells in rat olfactory bulb [9]. Exposure to high levels of mercury (a heavy metal) has also been thought to cause olfactory loss [10]. The present study was aimed to see the effect of lead on the histology of the olfactory bulb which may explain the olfactory dysfunction in the individuals exposed to lead.

Material and Method

A total number of 12 adult albino rats (6 male & 6 female) weighing 120g (±10g) were used in the present study. 6 rats with equal number of either sex were treated with 4% lead acetate, while the other 6 (3 male and 3 female) served as control did not receive any active compound. The concentration of lead acetate was ascertained after a careful trial in order to find maximum survival of 15 to 20 days. After this period, rats were anaesthetized with ether and perfused with buffered 10% formalin. Both olfactory bulbs were dissected out from superior aspect and separated from the brain. Olfactory bulbs were cut transversely into two parts and processed for paraffin embedding. From each blocks 10 μ thick sections were cut with rotary microtome. Haematoxylin and eosin stained sections were used for light microscopic observations.

Observations

On histological examination of olfactory bulb of treated group, it was observed that as compared to control (Fig. 1A, B) there was generalized edema and congestion in almost all layers of olfactory bulb. Capillaries appeared dilated and congested. Distortion of glomerular contour was obvious. Periglomerular cells were hyperchromatic and showed clumping. Multiple vacuoles of variable sizes were noticed in the outer plexiform layer (Fig. 1C and 1D). Granule cell layer showed loss of cells. Dark and pyknotic nuclcei were also present. No such types of abnormalities were found in control group of rats.

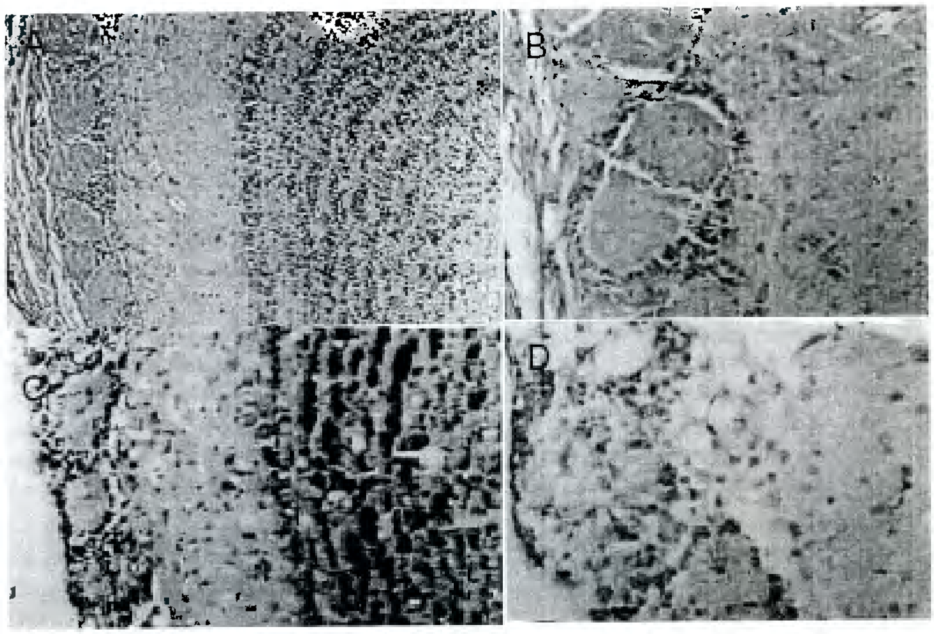

Figure 1. Photomicrographs from olfactory bulb of control rat (A and B) showing typical laminar pattern without edema, congestion or vacuolation while those from experimental group (C and D) show edema, vacuolation, congestion, loss of glomerular contour and clumping of periglmerular cells.
H &E stain, X100 (A &C). X400 (B&D).

Discussion

The layers of the olfactory bulb which show damage mainly include the lamina glomerulosa, outer plexiform layer and the granule cell layer. Gross damage in the region of olfactory bulb was also seen in present study in the form of petechial haemorrhage which might have been due to capillary dilatation. These findings are in partial agreement with those reported by certain workers [11] who exposed adult guinea pig to Lead carbonate and reported vascular changes in addition to encephalopathic effects of lead mediated directly at the neuronal level. Some other workers [12] have demonstrated hypertrophy of vascular pericytes. Lead pellets implantation in the rat forebrain produced vascular changes in addition to parenchymal necrosis and spongiosis in the hypothalamas [13]. Histological study of many parts of brain e.g. cerebral cortex , corpus striatum, choroid plexus and cerebellum after lead exposure revealed cerebellum to be most severely damaged [2]. In addition in this study [2] hemorrhages noticed along with damage to molecular and Purkinje cell layers and edema in the granule cell layer which correlated very well with the findings of the present study.

Histopathological findings of olfactory bulb on neuron and neuropil in the present study are to a great extent in agreement with those reporting degeneration of cells in the cerebral cortex [4] and reduced number of Purkinje and granule cells [14] of cerebellum and of hippocampal neurons on lead exposure [15] as well as vacuolations after incubation of guinea pig hippocampus in a lead containing medium [16] which was more pronounced in outer plexiform layer of olfactory bulb. The vascular changes observed in the present study are in agreement with those reported after exposure of lead in dogs [5] which indicates that irrespective of animal species, olfactory bulb is vulnerable to lead acetate toxicity.

Conclusion

From the above study it was concluded that olfactory bulb is vulnerable to toxicity of lead similar to the other parts of brain and that histopathological changes mainly included edema, vacuolation and congestion, glomerular distortion and pyknotic periglomerular cells.

Acknowledgement

We acknowledge the co-operation of technical and other supporting staff, Department of Anatomy, J.N. Medical College, AMU, Aligarh.

References

1. Balbus-Kornfeld JM, Stewart W, Bolla KI. Schwartz BS; Cumulative exposure to inorganic lead and neurobehavioral test performance in adults: an epidemiological review Journal: Occup Environ Med 52: 2-12; 1995.
2. Press, MF a) Lead encephalopathy in neonatal Long-Evans rats: morphologic studies. J Neuropathol Exp Neurol. 36: 169-93; Jan 1977
3. Patrick GW, Anderson WJ; Dendritic alterations of cerebellar Purkinje neurons in postnatally lead-exposed kittens. Dev Neurosci. 2000; 22: 320-8; 2000.
4. Bansal MR, Kausiial N. Banerjee UC; Effect of oral lead acetate administration on the mouse brain, J Trace Elem Exp Med, 3 : 235-246; 1990.
5. Hamir AN, Sullivan ND, Handson PD; Neuropathological lesions in experimental lead toxicosis of dogs. Neurobiol Aging 1984 Winter; 5: 297-307; 1984.
6. Stowe HD, Vandevelde M; Load-induced encephalopathy in dogs. J Neuropathol Exp Neurol; 38: 463-74; 1979.
7. Adam and Crabtree. Anosmia in alkaline battery workers. Br J Ind Med. 18: 216-221. 1961.
8. Jean Robert; Harmful effects of cadmium on olfactory system in Mice. Inhalation toxicology. 20: 1169-1177; 2008.
9. Carboni AA; Oral administration of zinc gluconate trihydrate. Am J Rhinology. 20: 262-268; 2006.
10. Upadhya U; Olfactory loss as a result of toxic exposure.Otolaryngo Clinics of North America; 37;1185-1207. 2004.
11. Bouldin and krigman; Acute lead encephalopathy in the guinea pig. Acta Neuropathologica; 33:185-190; August 1975.
12. Markov DV and Dimova RN; Ultrastructurai alterations of rat brain microglial cells and pericytes after chronic lead poisoning. Acta Neuropathol; 25-35; 1974.
13. Hirano A and Kochen JA; Some effects of intracerebral lead implantation in the rat; Acta Neuropathol (Berl) 30:: 307-15; 1975.
14. McConnell P, Berry M; The effects of postnatal lead exposure on Purkinje cell dendritic development in the rat. Am. J. Dis. Child; 133: 786-90; 1979.
15. Zook BC, London WT, Wilpizeski CR, Sever JL: Experimental lead paint poisoning in nonhuman primates. Pathologic findings; Brain Res ; 189: 369-76; 1980.
16. Brink U, Wechsler W; Microscopic examination of hippocampal slices alters short-term lead exposure in vitro. Neurotoxicol Teratol; 11: 539-43, 1985.

Correspondence:

Aijaz A Khan
Department of Anatomy
JN Medical College
Aligarh Muslim University
Aligarh 202 002
India

E-mail: aijazahmedkhan7@live.com