Indian Journal of Anatomy

Indexing Information: Index Copernicus, Poland, Google Scholar, ProQuest, USA, Genamics JournalSeek.

Printed at Saujanya Printing Press, D-47, Okhla Industrial Area Phase-1, New Delhi - 20

Red Flower Publication Pvt. Ltd.
48/41-42 DSIDC, Pocket-II, Mayur Vihar Phase-I
Delhi - 110 091(India)
Phone: 91-11-22754205, 45796900, Fax: 91-11-22754205
E-mail: redflowerppl@vsnl.net
Web:www.rfppl.org

The Indian Journal of Anatomy is a quarterly print and online journal of the **Red Flower Publication Pvt. Ltd.** publishes original and peer-reviewed articles, for the dissemination of anatomical knowledge with clinical, surgical and imaging guidance. Includes articles of history, reviews and biographies, locomotors, splachnology, neuroanatomy, imaging anatomy, anatomical variations, anatomical techniques, education and pedagogy in anatomy, Human Anatomy, Veterinary Anatomy, Embryology, Gross Anatomy (Macroscopic), Microscopic Anatomy (Histology, Cytology), Plant Anatomy (Phytotomy), Comparative Anatomy, editorials, letters to the editor, and case reports. Articles of veterinary anatomy, comparative and other morphological sciences are accepted.

Subscription Information

India

Individual	
1 Year	Rs. 500
Life Subscription (Valid for 10 Years)	Rs. 3000
Institutional (1 Year)	Rs. 3200

Rest of the World

Individual (1 Year)	USD100
Institutional (1 Year)	USD260

PAYMENT METHOD

By Cheque/Demand Draft:

Cheque should be in the name of **Red Flower Publication Pvt. Ltd.** payable at Delhi.

By Bank Transfer/TT:

Bank name: Bank of India

Swift Code: BKIDINBBDOS

Account Name: Red Flower Publication Pvt. Ltd.

Account Number: 604320110000467

Branch: Mayur Vihar Phase-I

Delhi - 110 091 (India)

Send All Orders to: **Red Flower Publication Pvt. Ltd.,** 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091, India, Phone: 91-11-22754205, Fax: 91-11-22754205, E-mail: redflowerppl@vsnl.net, Website: www.rfppl.org

Indian Journal of Anatomy

April - June 2014
Volume 3 Number 2

Contents

Revised Rates for 2014 (Institutional)

Title	Freequency	Rate (Rs): India	Rate ($):ROW
Dermatology International	2	2500	280
Gastroenterology International	2	3500	360
Indian Journal of Agriculture Business	2	4500	300
Indian Journal of Anatomy	2	3200	260
Indian Journal of Ancient Medicine and Yoga	4	6600	330
Indian Journal of Anesthesia and Analgesia	2	4000	600
Indian Journal of Anthropology	2	8000	500
Indian Journal of Applied Physics	2	3500	400
Indian Journal of Biology	2	1500	170
Indian Journal of Cancer Education and Research	2	4500	500
Indian Journal of Communicable Diseases	2	1000	58
Indian Journal of Dental Education	4	3200	288
Indian Journal of Forensic Medicine and Pathology	4	12500	576
Indian Journal of Forensic Odontology	4	3200	288
Indian Journal of Genetics and Molecular Research	2	5000	262
Indian Journal of Law and Human Behavior	2	5000	500
Indian Journal of Library and Information Science	3	7500	600
Indian Journal of Maternal-Fetal & Neonatal Medicine	2	4500	400
Indian Journal of Mathematics and Statistics	2	3000	200
Indian Journal of Medical & Health Sciences	2	1800	120
Indian Journal of Obstetrics and Gynecology	2	2000	200
Indian Journal of Pathology: Research and Practice	2	3000	915
Indian Journal of Plant and Soil	2	5000	1700
Indian Journal of Preventive Medicine	2	3200	270
Indian Journal of Reproductive Science and Medicine	4	3000	180
Indian Journal of Scientific Computing and Engineering	2	3300	280
Indian Journal of Surgical Nursing	3	1800	70
Indian Journal of Trauma & Emergency Pediatrics	4	6500	302
International Journal of Agricultural & Forest Meteorology	2	8000	800
International Journal of Food, Nutrition & Dietetics	2	3200	900
International Journal of History	2	6000	500
International Journal of Neurology and Neurosurgery	2	7500	276
International Journal of Political Science	2	5000	400
International Journal of Practical Nursing	3	1500	70
International Physiology	2	4000	240
Journal of Animal Feed Science and Technology	2	3500	280
Journal of Cardiovascular Medicine and Surgery	2	5500	238
Journal of Orthopaedic Education	2	2500	190
Journal of Pharmaceutical and Medicinal Chemistry	2	3000	350
Journal of Psychiatric Nursing	3	1800	70
Journal of Social Welfare and Management	4	6600	276
Meat Science International	2	5000	500
Microbiology and Related Research	2	3800	150
New Indian Journal of Surgery	4	6500	360
Ophthalmology and Allied Sciences	2	3000	150
Otolaryngology International	2	2000	300
Pediatric Education and Research	4	3200	150
Physiotherapy and Occupational Therapy Journal	4	7000	360
Urology, Nephrology and Andrology International	2	2200	350

Terms of Supply:

1. Advance payment required by Demand Draft payable to Red Flower Publicaion Pvt. Ltd. payable at Delhi.
2. Cancellation not allowed except for duplicate payment.
3. Agents allowed 10% discount.
4. Claim must be made within six months from issue date.

Order from

Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India), Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205. E-mail: redflowerppl@vsnl.net, redflowerppl@gmail.com, Website: www.rfppl.org

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Perforated Olecranon Fossa in Humerus

Sayee Rajangam*, Vidhya R.**

Abstract

Context: Olecranon fossa in humerus may be perforated. The perforated olecranon fossa, in general is reported associated to the left humerus and humerus of the female sex. **Aims:** In the present study, it is aimed to find out the occurrence of the perforated olecranon fossa in the humerus. **Settings and Designs:** Department of Anatomy, International Medical School, Bangalore. **Methods and Materials:** 44 humerus were available in the institute; out of which, 22 each belonged to the right and left sides. The subjective sex determination could identify 24 as male and 20 as the female humerus. **Statistical Analysis:** The percentage analysis was done. **Results:** The perforated olecranon fossa was observed in 17 (38.6%) humerus. Their presence was found to be associated to the left (50%) and the female (50%) humerus. **Conclusion:** The finding could be of significance, academically for the anatomists and anthropologists and clinically for the radiologists and orthopedic surgeons for the transhumoral approach for the elbow arthroscopy.

Keywords: Perforation; Olecranon fossa; Humerus.

Introduction

Olecranon fossa, a deep hollow structure in the posterior surface of the distal end of the humerus lodges the tip of the olecranon process of the ulna during the extension of the elbow. The bone in the floor of the fossa is thin and may be partially deficient. The perforation is known as supratrochlear foramen (STF) or septal aperture. It is reported that the occurrence of the perforated olecranon fossa is more frequent in female and in left humerus (Godycki 1957)(cited in Krogman 1962). The reported incidence of STF in a South Indian study was 28% (Singhal and Rao 2007) and in a North Indian study was 24.1% (Diwan *et al* 2013). Further, Singhal and Rao (2007) have reported that the incidence of STF in different races ranged from 6 to 60%. The present study is aimed to report the occurrence of the perforation in the olecranon fossa i.e. STF in humerus.

Material & Method

The available humerii in the institute was 44; out of which right and left humerus were 22 each respectively. Subjective sexing based on the length, heaviness, girth and the roughness of the attachments of the muscles identified, that the male and female bones were 24 and 20. The percentage occurrence of the perforation in the olecranon fossa was calculated.

Result

The findings are tabulated. (Figure 1)

The olecranon fossa was perforated in 17 humerus and the rest (27) had a bony plate or septum.

Author's Affiliation: *Professor, **Lecturer, Department of Anatomy, International Medical School, New BEL Road, Bangalore 560054, Karnataka, India.

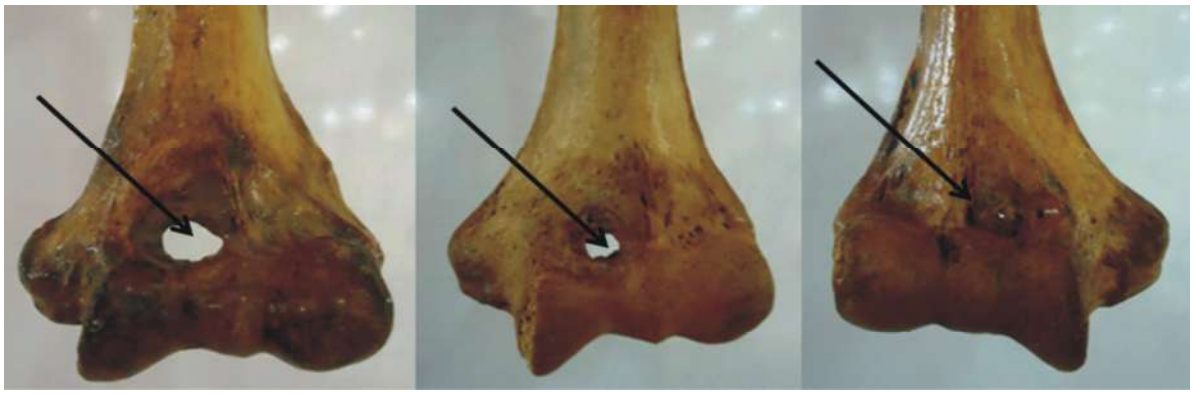
Reprint's Request: Dr. Sayee Rajanagm, Professor, Department of Anatomy, International Medical School, New BEL Road, Bangalore 560054, Karnataka, India.

E-mail: drsayee@gmail.com

Table 1: Perforated Olecranon Fossa

Total number of humerus: 44; Foramen present: 17 (38.6%); Foramen absent: 27 (61.4%)				
Total number of humerus: Right side: 22			Total number of humerus: Left side: 22	
Foramen present: 06 (27.3%)			Foramen present: 11(50%)	
Foramen absent: 16			Foramen absent: 11	
Total number of humerus: Male 24			Total number of humerus: Female: 20	
Foramen present: 07 (29%)			Foramen present: 10 (50%)	
Foramen absent: 17			Foramen absent: 10 (50%)	
	Right n22		Left n22	
Foramen	Male n11	Female n11	Male n13	Female n9
Present n17	03 (27.2%)	03 (27.2%)	04 (30.7%)	07 (77.7%)
Absent n 27	08	08	09	02

Figure 1: Arrows Indicate the Variations in the Size and Shape of the Perforations in Olecranon Fossa. (A): Large; (B): Medium; (C): Small.



The perforated olebranon fossa was found to be higher in left humerus (50%) and in female humerus (50%).

On associating the sides with that of the male and female humerus, the occurrence of the perforation was higher for the left side and especially for the female humerus (77.7%). The shape of the perforation in olecranon fossa was studied; it was either large (12/44, 27.3%) or medium (24/44,54.5%) or small (8/44,18.2%) (Figure 1).

Discussion

From the review of literature, it is seen, that in 1825, it was Meckel, who had first described STF in humerus. In 1832, Hardlicka observed that the perforation in the humerus seemed to be more frequent in higher primates other than man; hence, the presence of STF was considered to be an atavistic character. In 1927, Hirsh had stated that the thin plate of bone between the olecranon and coronoid fossa is always present until the age of 7 after which the bony septum occasionally becomes absorbed to form the STF (cited in Diwan *et al* 2013).

The article by Diwan *et al* (2013) indeed has reported in detail about STF: the history, its incidence in different populations in India and in different races, its occurrence as per the right/left sides and female/ male humerii, its association to the sides and female/ male humerii.

i) *Incidence:* In the present study, in South Indian Bones, the incidence of the perforated olecranon fossa was found to be 38.6%. It is within the reported range of 6% to 60%; but, more than the reported incidence from India: Central Indians 32%; South Indians 28%; North Indians 27.56%; Eastern Indians: 27.4% (Singhal *et al* 2007, Diwan *et al* 2013).

ii) *Incidence as per the Right and Left Sides:* The frequency of the STF was found to be more common on left side than on the right side (Godycki 1957(cited in Krogman 1962); Radi *et al* 2013) The reported range in the percentage occurrence of the STF in the right was 20.22% to 28% and in the left was 20% to 35%. (Singhal *et al* 2007, Diwan *et al*

2013) In the present study, the occurrence of the perforated olecranon fossa is found to be frequent on the left side as stated in literature. And the right (27.53%) side occurrence was within the reported range; but, on the left (50%) it was found to be very high than the range given in literature.

iii) *Incidence in the Male and Female Humerii:* Supratrochlear foramen was more often associated to female humerii. (Godycki 1957(cited in Krogman 1962) It was suggested that the association may be due to the 'inward curvature of the female elbow angle' (Mall 1905) (cited in Diwan *et al* 2013). In the present study too, the STF was found more common in the female humerii. But, the percentages for the male (29%) and female (50%) bones are found to be higher than what was reported in literature: Singh and Singh (1972): male (21%)/ female (38%)/ Diwan *et al* 2013: male 24.1%/ female 23.76%.

The male (n 6) to female (n 11) ratio of the occurrence of the perforated olecranon fossa in the present study is around 1:1.4; which is less than the reported male to female ratio as 1:3.7. (Godycki 1957) (cited in Krogman 962) Of course, opinions differed, whether the perforated olecranon fossa could be an indicator for the sex determination in humerus. In general, in long bones of the limbs, the sex is determined subjectively with their morphological features and objectively with morphometry. (Godycki 1957)(cited in Krogman 1962)

iv: *Incidence by Associating the Sides and the Sex Determined Humerii:* In a study on North Indian humeriir, the STF findings were: Female: right: 15.63%/ left: 31.89%; Male: right 26.76%/ left: 21.45%. (Diwan *et al* 2013) In the present study, the STF was associated to the left side and female humerii (77.7%) and it also was similar to the reports in literature.

v: *Shape and Size of the STF:* The shapes of the STF have been studied (Diwan *et al*; 2013). The authors have noted either oval or round or triangular shapes of STF and have observed that the oval shape was found to be the maximum (152/ 428,83.06%). In the present study, it is the size of the foramen which was studied and medium size foramen was found to be the maximum (54.5%).

The interesting information gathered from the literature on the perforated olecranon fossa were:

i) absent; in case, the humerus is small

ii) may be obliterated by a membrane

iii) communicates with the anterior compartment of the arm which is made possible through the enlargement of the foramen

iv) utilized in orthopedics in the transhumoral portal for the elbow arthroscopy and if found necessary by removing the membrane. (Chow 2000)

The differences observed reflect the sample size or ethnicity or race. It appears that in Indian population, the incidence of the STF seemed to be high. Hence the presence of STF should be kept in mind before any procedures (intramedullary nailing in the lower end of humerus/pseudo-lesion appearance in X-rays) are carried out in the elbow region especially on the left side and in female.

In the present study, the higher percentages of STF in the left female humerus may be because of the sample size. In spite of the sample size, the study was undertaken as an academic interest. Moreover, the observations of the present study was based on the subjective sexing of the humerus.

The surmised interpretations for the association of presence of the perforated olecarnon fossa to the left side and to the females are listed:

i) the ossification process at the lower end of humerus and or upper end of ulna especially the olecranon process at its tip?

ii) the biomechanics at the elbow and radio-

ulnar joints? (axis/planes/ movements/ work load) (flexion and extension/ supination and pronation/slight adduction and abduction

iii) the oblique axis of the shaft of the ulna from its upper end to the head of the ulna at its lower end?

iv) left cerebral dominance common in population leading to more work load and thereby to earlier the ossification, the less occurrence of STF in males and right side?

v) the left side and the females may be because of the more 'tasks' than the right side and the males?

Conclusion

The incidence of the STF or the perforated olecranon fossa was in 17 out of 44 humerii. It was found in 7 out of the 9 left female humerus. Its presence definitely may of clinical significance for the interpretation of X-rays and any arthroscopy procedures at elbow.

References

1. Krogman WM. The Human Skeleton in Forensic Medicine. Illinois, USA: Charles C Thomas Pub Ltd; 1962.
2. Chow JCY. Advanced Arthroscopy, 1st edition. Philadelphia, USA: Springer- US; 2000.
3. Diwan RK, Rani A, Rani A, Chopra J, Srivastava AK, Sharma PK, Verma RK, Pankaj AK. Incidence of Supratrochlear foramen of Humerus in North Indian Population. *Biomedical Research.* 2013;24(1):142-145.
4. Singhal S, Rao V. Supratrochlear foramen of the humerus. *Anat Sci Int.* 2007; 82: 105-107.
5. Singh S, Singh SP. A study of the supratrochlear foramen in the humerus of North Indians. *J Anat Soc India.* 1972; 21: 52-56.

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

A Study of Congenital Anomalies of Ureter

Suma Dnyanesh*, Daksha Dixit, Dnyanesh D.K.***, Amit Magadum******

Abstract

Background: The ureters are a pair of thick walled cylindrical tubes which convey urine from the corresponding kidney to the urinary bladder. They descend along the posterior abdominal wall to enter the bladder on its poster inferior surface. Premature division of urethral bud results in double ureter. Duplication of ureter can be incomplete or complete. The term 'bifid' ureter is used when it is incomplete. **Aims and Objectives:** To study the anomalous pattern of ureters and its clinical implications. **Materials and Methods:** We studied 150 cadavers over a period of 2 years for the presence of anomalies of ureters. **Results:** We found 2 cases of bifid ureter: in the first case, we found unilateral incomplete double ureter. The duplicated ureters joined with each other in lower part and finally opened in the urinary bladder by a common orifice. In the second case, we got bilateral double ureters. Both the ureters on both the sides opened independently in urinary bladder with 4 separate openings. **Conclusion:** Double ureter can go unnoticed many times and can give rise to a number of clinical manifestations. Variations are thus important for urological conditions, radiological interpretations and also for surgeries involving renal transplants.

Keywords: Double ureter; Urethral bud; Urinary bladder.

Introduction

The ureters are a pair of thick walled cylindrical tubes which convey urine from the corresponding kidney to the urinary bladder. They descend along the posterior abdominal wall to enter the bladder on its postero inferior surface. Premature division of ureteric bud results in double ureter. Duplication of ureter can be incomplete or complete. The term bifid ureter is used when it is incomplete. Incidence of bifid ureters is 0.8%, ranging from 0.5 to 3%.[2] Familial incidence of double ureters is reported, but is a rare incidence. Bifid uretes are seen twice more commonly in females and on right side compared to males.[3]

Double ureter can go unnoticed many times and can give rise to a number of clinical manifestations. It can lead to the formation of calculi, urinary tract infections, etc. A thorough knowledge of these clinical conditions and their correlation with the developmental basis will be of great help in their diagnosis and management.

Embryological Aspects[3]

Ureteric bud grows from the postero-medialside of the caudal part of the mesonephric duct. Its distal end dilates and invades the lower part of nephrogenic cord and divides into cranial and caudal parts, forming the future major calyces. These major calyces divide repeatedly to about 13 or more generations of tubules. The branches of 2nd, 3rdand 4th orders are absorbed to form the minor calyces. The stalk of the ureteric bud persists as the ureter and its dilated end forms

Author's Affiliation: *Assistant Professor, ** Professor & Head, ****Assistant Professor, Department of Anatomy, ***Associate Professor, Department of Pediatrics, KLE University's J. N. Medical College, Belgaum, Karnataka - 590010, India.

Reprint's Request: Dr. Suma Dnyanesh, Assistant Professor, Department of Anatomy, KLE University's J. N. Medical College, Nehru Nagar, Belgaum Karnataka, India Pin code: 590010.

E-mail: drdnyaneshk@gmail.com, drsuma410@gmail.com

the pelvis of the ureter.

Premature division of ureteric bud results in double ureter. The 2 ureters thus formed are connected to a single kidney and open in the bladder either by a common orifice or by 2 separate orifices, upper and lower. When 2 such separate orifices are present, the upper ureter drains urine from the upper part of kidney and the lower ureter drains from its lower part. In this condition, the mesonephric duct gives rise to two ureteric buds, cranial and caudal, which invade the metanephricblastema independently and induce the development of upper and lower poles of the kidney respectively. As the mesonephric duct undergoes loop formation in the posterior wall of the bladder, the lower ureter opens in the bladder in normal position, whereas the upper ureter migrates more caudally along with the caudal shift of the terminal part of mesonephric duct and opens in ectopic position. That is how the normal and ectopic ureters cross each other.

Materials and Methods

We studied 150 cadavers over a period of 4 years for the presence of anomalies of ureters. Of these, only 2 cadavers showed the presence of bifid uereters: 1 was unilateral and the other was bilateral. We then studied the course and termination of these ureters. The presence of these bifid ureters in adults and their effects were then correlated.

Figure 1: Showing Course of Bifid Ureter

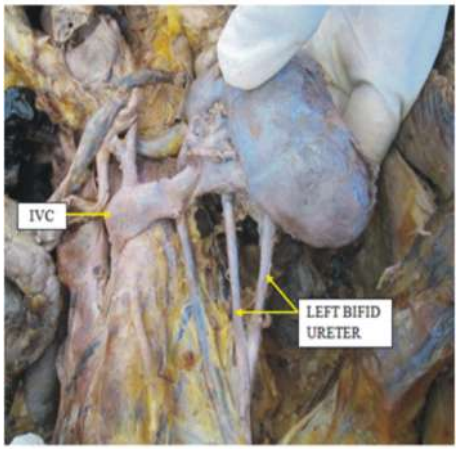
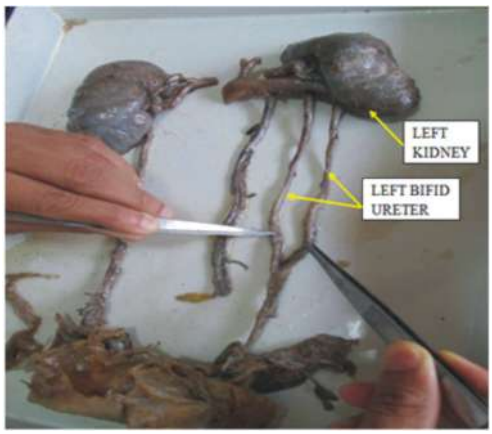


Figure 2: Showing the Bifid Ureter on Left Side



Case I

During dissection of a 60 year old male cadaver, a unique anatomical variation involving left ureter was found. Two ureters were found connected to the left kidney. They descended from separate renal pelvis from the upper and lower poles of the left kidney. The upper ureter was arising from the hilum of the kidney maintaining its normal position. The lower ureter was arising from the lower pole (Figure 1). The duplicated ureters joined with each other about 2.5cm proximal to vescicoureteraljunction and finally opened into the urinary bladder by a common orifice (Figure 2). Apart from this variation, the shape, size and position of both kidneys were normal. The right ureter was also normal

Fig 3: Showing the Course of Bilateral Bifid Ureters

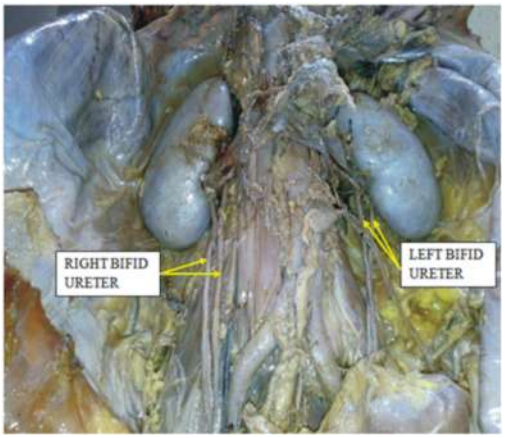
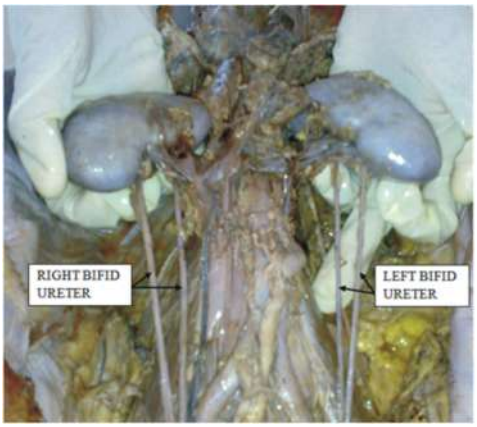


Fig 4: Showing Bilateral Bifid Ureters



(Figure 3).

Case II

We dissected a 55 year old male cadaver, in which we found that double ureters were connected to kidneys of both the sides and with further dissection the following was observed.

At their proximal ends, on both sides (right and left), the 2 ureters had separate renal pelves. The ureter which was laterally placed arose from lower pelvis and emerged from lower part of the hilum, the second ureter which was placed medially arose from upper pelvis and emerged from the upper part of the hilum.

On Right Side: Both ureterscame down parallel to each other up to the pelvic brim.

On Left Side: Both ureters descended down parallel to each other upto a level just above the bifurcation of aorta.

Then themedial ureters (right and left)which werearising from upper pelvis, crossed the lateral ureters superficially from medial to lateral side and remained lateral till they opened into the urinary bladder with separate openings placed at a lower level. The lateral ureters (right and left) which were arising from the lower pelvis opened into the bladder with separate openings at the normal position. Thus both the ureters on both the sides opened independently into the urinary bladder with 4 separate openings.

Discussion

The duplication of upper urinary tract is one of the commonest anomalies and occurs in 1 in 160 individuals.[5] In our study we got 2 cases out of 150 cadavers. Cases of familial bifid or double ureter which is an autosomal dominant condition have been reported. But such occurrence is rare.[6]

The incidence of duplex renal collecting system and ureter ranges from 0.5-3%. Unilateral ureteral duplication is more commonly seen than the bilateral duplication.[7,8] In the present study, out of 150 cadavers, we have got 1 case of unilateral bifid ureter and 1 case of bilateral complete ureteral duplication.

Duplex collecting system is seen in approximately 1.3% patients[9] and 0.7% of these have associated urinary tract anomalies.[10]

The incidence of bifid pelvis and bifid ureter is 4% in the population of North America.[11]

Table I: Morphology of Bifid Ureters

	Side	Feature of Ureter	Beginning	Termination	Openings in the bladder
Case – I	Right	Normal	Normal	Normal	2
	Left	Bifid	Separate pelvis	The duplicated ureters joined and opened into the bladder by a common orifice	
Case- II	Right	Bifid	Separate pelvis	Independent opening into the bladder	4
	Left	Bifid	Separate pelvis	Independent opening into the bladder	

A duplicated ureteral system is dangerous as it can give rise to an array of clinical manifestations. It can lead to the formation of urinary stones, vesicoureteral reflex, ureterocele, obstructive uropathy, etc.

Conclusion

Variations are important for urological conditions, radiological interpretations and also for surgeries involving renal transplants. Surgeons must be careful about the urinary tract anomalies before the surgical management of urinary stones or renal transplants, since co-existing ureteral duplication may increase the morbidity and mortality of the affected individual. The various manifestations and complications along with their developmental basis need to be kept in mind by clinicians and surgeons in order to bring about their early diagnosis and treatment.

References

1. Sadler TW. Urogenital system, Langman's medical embryology, 9thedition. LippincottWilliums and Wilkins; 2004: 321-6.
2. Setsuko Tohno, Cho A, Yoshiyki TJ. *NARA Med Assoc*. 2008; 59(1): 183-87.
3. Rege VM, Deshmukh SS, Borwankar SS, Gandhi RK. *Journal of Postgraduate Medicine*. 1986; 32: 233-35.
4. Dutta AK. Essentials of Human embryology 4th ed. Kolkatta: Current Books International; 2007, 215-22.
5. Campbell MF, Harrison JH. Urology, 3rd edition, vol. 2. Philadelphia: WB Saunders; 1970, 1488.
6. Atwell JD, Cook PL, Howell CJ, Hyde I, Parker BC. Familial incidence of bifid and double ureters. *Archives of Diseases in Childhood.* 1974; 49: 390.
7. Kawahara T, Ito H, Terao H. Ureteroscopy assisted retrograde nephrectomy (UARN) for an incomplete double ureter. *Urol Res*. 2012; 40: 781-2.
8. Inamoto K, Tanaka S, Takemura K. Duplication of renal pelvis and ureter: associated anomalies and pathological conditions. *Radiat Med*. 1983; 1: 55-64.
9. Stec AA, Baradaran N, Gearhart JP. *Urology*. 2012; 79(1): 207-209.
10. Amis ES, Cronan JJ, Pfister RC. *Urology*. 1985; 26: 82-88.
11. Adel K Afifi. Illustrated encyclopedia of human anatomic variation; organ systems: Urinary systems Kidney, ureters, bladder and urethra.

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Morphological Characteristic of Placenta in Sudanese

Abdelrahman M.A.

Abstract

The placenta is the most important and the only organ between mother and fetus, serving multiple functions endocrinal , respiratory , metabolic and main useful functions. Normal development of a placenta is the one of the important requirements for a healthy pregnancy, regulating fetal growth and fetal health. The objectives of this study were to compare the placental weight with the infant's weight, to measure the placental diameter and to count the numbers of cotyledons in each placenta. The study was carried out among 240 infant, 102 females and 138 males, delivered at Omdurman new (Alsaudi) and Alshaikh Mohammed Ali Fadol hospitals of obstetrics gynecology. All infants were looked healthy and their weight ranged from 1.85 to 4.18 kg with mean of 3.02. The weight of placenta ranged between 0.35 to 0.77 kg with mean of 0.535 kg. Placental weight in males ranged between 0.35 to 0.75 kg with mean of 0.537 kg. While the female placental weight ranged between 0.4 to 0.77 kg with mean of 0.531 kg. The placental diameter ranged between 15 to 23 cm with mean 18.64 cm. The diameter in males ranged between 16 to 23 cm with mean 18.52 cm. In female was ranged from 15 to 23 cm with mean of 18.81 cm. The mean of cotyledons number was 19 with std. Deviation 1.362 and ranged from 16 to 22. The mean of number in males was 19.17 and ranged from 16 to 22 and in females the mean was 18.88 and ranged from 16 to 22. According to Pearson, Kendall's Tau b and Spearman's rho correlations and found that there is a significant relationship between infant weight and placental weight.

Keywords: Placenta; Ccotyledon; Placental weight; Placental diameter; Umbilical cord.

Introduction

Early in gestation, the developing embryo is small and its nutritional and waste disposal needs are minor. At this point, the embryo absorbs nutrients from the mother's endometrial secretions and expels its waste into the uterus. As time passes, the needs of the embryo increase. As it progresses from embryonic stage to fetal stage, more nutrients are required and a much more sophisticated means of satisfying the nutritional and waste disposal needs must be established. This is accomplished only after the embryo develops a vascular system and can establish an effective and efficient interface (i.e., the placenta) between the mother's vascular system and its own. In addition to nourishing the fetus and providing a means for disposing of its wastes, the placenta secretes a number of hormones, including the steroid hormones estrogen and progesterone. It also secretes protein hormones and is the source of human chorionic gonadotrophin (hCG). A luteinizing hormone, hCG is secreted by the syncytiotrophoblast of the placenta in early pregnancy. It maintains the function of the corpus luteum and stimulates progesterone production in the placenta. Because hCG is found in the blood and urine of pregnant

Author's Affiliation: University of El-Imam El-Mahdi, Faculty of Medicine. Kosti, SUDAN.

Reprint's Request: Abdelrahman M.A., University of El-Imam El-Mahdi, Faculty of Medicine. Kosti, SUDAN.

E-mail: motaz82sd@yahoo.com

women, it is the basis for most common tests used to diagnose pregnancy.

The placenta secretes the hormone relaxin, as well, which is thought to relax the joints of the pelvis and assist in dilating the cervix during birth.[1]

The placenta is the most important and the only organ between mother and fetus, serving multiple functions. It acts as an endocrine organ producing several types of hormones e.g. lactogen, chorionic gonadotrophins etc. It allows the exchange of oxygen and CO_2, whereby transfer of oxygen takes place from maternal blood to the fetus, and carbon dioxide goes out from fetus to mother. It allows the transfer of carbohydrates, protein, amino acid, polypeptides, lipids, vitamins, water, electrolytes and pharmacological agents from the mother to the fetus.

A placenta has a maternal surface (basal plate) and a fetal surface (chorionic plate). The basal plate and chorionic plate meets at the placental margin and form the smooth fetal membranes. The space between chorionic and basal plate is filled with the intervillous lakes of maternal blood. The placenta develops from the same sperm and egg cells that form the fetus, and functions as a fetomaternal organ with two components, the fetal part (Chorion frondosum), and the maternal part (Decidua basalis).[2]

Besides secreting hormones, the placenta protects the fetus from immune attack by the mother and induces increased maternal blood flow to the placenta. Near the time of delivery, the placenta produces hormones that mature the fetal organs in preparation for life outside of the uterus. The placenta supports essential fetal respiratory functions before lung development, carrying oxygen and nutrients from the maternal blood across the membrane into the fetal circulation by diffusion and allowing carbon dioxide to pass in the opposite direction. The placenta provides the fetus with water, inorganic salts, carbohydrates, fats, proteins and vitamins and carries fetal waste into the mother's circulatory system to be secreted via her urinary system. The placenta also protects the fetus by prohibiting some harmful microorganisms from entering fetal circulation. A portion of the placental membrane called the placental barrier provides this protection. Storage is another function of the placenta. The placenta stores carbohydrates, calcium, iron and proteins for release into fetal circulation. Two portions make up the placenta: fetal and maternal. The fetal circulation enters the placenta via the two umbilical arteries that are embedded within the umbilical cord. Once the fetal arteries enter the placenta, they branch into units called cotyledons, which are structures similar to inverted trees. The tiniest branches of the fetal circulation are made up of capillary loops embedded within the chorionic villi. The fetal circulation continues to branch until it reaches capillaries of the villi. Once nutrients have been absorbed and waste products released, the fetal blood collects in the umbilical vein, where it returns to the fetus.The maternal portion of the placenta receives blood by way of the spiral arteries of the uterus. When the spiral arteries make contact with the placenta, they end in open channels that pour maternal blood into the intervillous space. The intervillous blood is returned to the maternal circulation through drain-like uterine veins. As much as 35% of the maternal blood will course through the intervillous space to support the fetus until the time of delivery.[2]

The fetal and maternal portions of the placenta connect via the umbilical cord. This sustaining connection between the fetus and the placenta is formed rudimentarily by the fifth week of gestation.

The fetal heart pumps fetal blood through the umbilical arteries into the placenta, where tiny branches are bathed in maternal blood. These vessels are drained by the tributaries of the umbilical vein, which take the blood back into the cord to the fetus for return to the heart. As a result, used blood is pumped through arteries to the mother and refreshed blood is returned to the fetal circulation by veins. After birth, this job is performed by the lungs. Shortly after birth the cord is clamped and cut and the remnant shrivels and separates from the

infant's navel 1 to 3 weeks following birth.(3)

Materials and Methods

Study Design

Descriptive prospective study in which samples was collected throughout the study.

Study Area

Study was carried out in Omdurman new (Alsaudi) and Alshaikh Mohammed Ali Fadol hospitals of obstetrics gynecology, which they located in Khartoum state, Omdurman city, which is located west to the Khartoum city the capital of Sudan.

Study Population

Include all newborn infants delivered at the time of the study with their placenta in Omdurman new (Alsaudi) and Alshaikh Mohammed Ali Fadol hospitals of obstetrics gynecology. It was done in period between October and November 2010.

Sample Size

A total of 240 infants (both sexes) with their umbilical cords according to the fallowing equation formula:

$$n = \frac{t^2 \times p(1-p)}{m^2}$$

n = required sample size.

t = confidence level at 95 % (standard value of 1.96).

p = estimated prevalence of umbilical cord measurement in target area.

m = margin of error at 5 % (standard value of 0.05).

Sample Collection

Samples was collected by immediately after delivery the weights of the newborn infants will estimated by sensitive balance for evaluate their weight. as the same time the placenta is prepared to evaluate their morphological parameters including weight, length and numbers of cotyledons.

Technique

The weight of the infants is measured by sensitive balance. Morphological parameters is measured as follows:

(A) Placental weight is measured by sensitive balance.

(B) Placental diameter is measured by plastic meter .

(C) Number of cotyledons is measured by inspection.

Data Analysis

Data will be analyzed by statistical package for social science (SPSS). Data will be displayed by tables and histogram.

Results

The study was carried out among 246 infant, 108 females and 138 males, All infants were looked healthy and their weight ranged from 2 to 4.18 kg with mean 3.01 and stander deviation 0.426, Table 1 and Figure 1.

Placental Weight

The weight of placenta ranged between 250 to 770 gm with mean 528.05 gm and Std. Deviation 110.916. There was deference in mean weight between males and females the former was the heavier. Placental weight in males ranged between 350 to 750 gm with mean 537.39 gm and Std. Deviation 104.730. While the female placental weight ranged between 250 to 770 gm with mean 516.11 gm and Std. Deviation 104.730, Table 2 and Figure 2. The maximum weight heavier in female than male that came from the infant weight which was heavier in that case of male infant.

Table 1: Weight Vs Sex

	Mean	Std. Deviation	Minimum	Maximum
Female	2.90	0.404	2	4
Males	3.09	0.424	2	4.18
Total	3.01	0.426	2	4.18

Figure 1: Weight Vs Sex

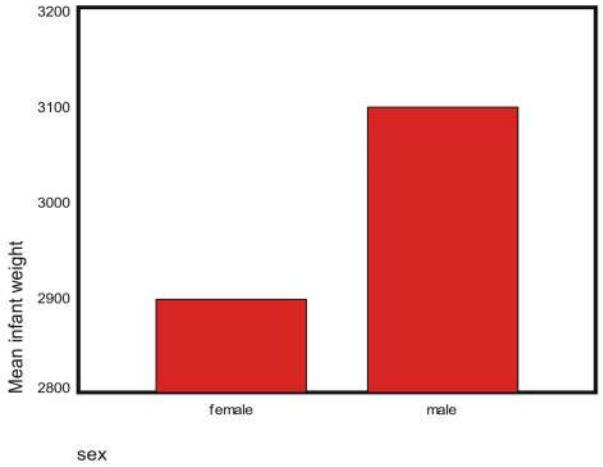
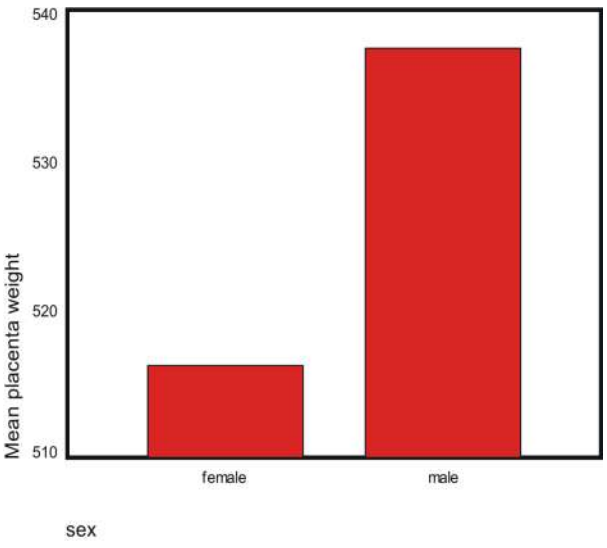


Table 2: Placental Weight

	Mean	Std. Deviation	Minimum	Maximum
Female	516.11	117.766	250	770
Male	537.39	104.730	350	750
Total	528.05	110.916	250	770

Table 2: Placental Weight



Placental Diameter

The placental diameter ranged between12 to 23 cm with mean 18.48 cm and Std. Deviation 2.094. The diameter in males ranged between 16 to 23 cm with mean 18.52 cm and

Table 3: Placental Diameter

	Mean	Std. Deviation	Minimum	Maximum
Female	18.43	2.348	12	23
Male	18.52	1.880	16	23
Total	18.48	2.094	12	23

Figure 3: Placental Diameter

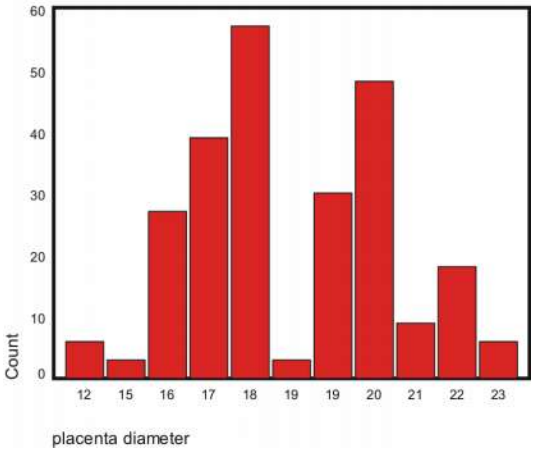


Table 4: Cotyledon Number

	Mean	Std. Deviation	Minimum	Maximum
Female	18.50	2.125	12	22
Male	19.17	1.278	16	22
Total	18.88	1.731	12	22

Std. Deviation 1.880, in female ranged between12 to 23 cm with mean 18.43 cm and Std. Deviation 2.348 (Table 3 and Figure 3).

Cotyledons Number

The mean of cotyledons number was 18.88 with std. Deviation 1.731 and ranged from 12 to 22. The mean of number in males was 19.17 with std. Deviation 1.278 and ranged from 16 to 22 and in females the mean was 18.50 with 2.125 std. deviation and ranged from 12 to 22 (Table 4).

Discussion

This research study the overall shape of the placenta without taking into account any diseases or ailments to the mother and the fetus developed, the study was only concerned with the external form of the placenta.

Most previous studies focused on studying

the changes occur to the placenta that result from external factors, but this research only interested in studying the overall shape of the placenta in Sudanese.

Weight and Sex: In this study the weight of babies at birth was 2.90 ± 0.4, 3.09 ±0.4 (mean ± standard deviations) in female and male babies respectively. Found that the male weight was great than female weight.

Placental Dimensions: The weight of placenta ranged between 250 to 770 gm with mean 528.05 gm and Std. Deviation 110.916. There was deference in mean weight between males and females the former was the heavier. Placental weight in males ranged between 350 to 750 gm with mean 537.39 gm and Std. Deviation 104.730. While the female placental weight ranged between 250 to 770 gm with mean 516.11 gm and Std. Deviation 104.730, table 2 and figure 2. The maximum weight heavier in female than male that came from the infant weight which was heavier in that case of male infant. While Jaya et al reported that the average weight of placenta of full term normal babies was 502.4 g (SD43.3) and studied the Relationship between placental weight and birth weight and reported that the average weight of placenta of full term normal babies was 502.4 g (SD 43.3).[4] The placental weight increased with the increase in birth weight and gestational age.[5] Park said that the average weight of the 378 placentas was 616 gm and for normal cases it was 621 gm.[6]

Ashfaq *et al* reported that the mean weight of full term normal placenta was 530+_10 and the mean diameter of full term placenta was 14, 26.[7] Majumdar *et al* found that The mean birth weight babies was 2, 8+_0, 32 kg The mean weight of the placenta was 485, 85+_47,31gm And the mean of cotyledon number of placenta was 17+_2.[8] Frisancho et al said that the mean of placenta was 551gm and the mean of new born infant was 3111.3 kg.[9]

The mean and standard deviation of neonates' weights at birth and placental weights were 3214.28 ± 529 and 529.72 ± 113 g, respectively was reported by Asgharnia.[10] I found that the diameters of placenta were symmetrical, in male babies 18.5 ± 1.9 cm and female babies 18.4 ± 2.3 cm.

Cotyledon Number: In the present study found that the cotyledon numbers was approximately equal in both sex of babies (18 .5 ± 2.2, 19.2 ± 1.3) in female and male respectively. There was a significant between infant's weight and placental weight. These studies agree with other studies reported by Manop *et al*.[11]

Conclusion

In this research, we find that the weight of males was larger than females weight in newborns Sudanese. While the weight of the placenta at birth 502.4 ± 43.3 g. The placental diameter 18.45 cm. But cotyledon numbers were equal in both sexes of children. This clearly shows the morphometric of the placenta is very important because it can determine the extent of the health of the newborn. it is crucial to link the microscopic characteristics of the placenta in future studies.

Conflict of Interest

NIL

Author's Contributions

1. Variations in origin of the Obturator artery in Sudanese cadavers with its clinical implications
   - A Thesis of Master Degree in Clinical Anatomy.
2. An article about : A Preliminary Study On The Morphological Variations in The Umbilical Cord Of Sudanese. http://www.timejournals.org/tjmsrr/archive/2013/August/pdf/NuggedAlla.pdf.

References

1. MacLennan AH. The role of relaxin in human reproduction. *Clin Reprod Fertil*. 1983; 2(2): 77-95.

2. Ellery PM, Cindrova-Davies T, Jauniaux E, Ferguson-Smith AC, Burton GJ. Evidence for transcriptional activity in the syncytiotrophoblast of the human placenta. *Placenta.* 2009; 30(4): 329–334.
3. Johnson P, Maxwell DJ, Tynan MJ, Allan LD. Intracardiac pressures in the human fetus. *Heart*. 2000; 84(1): 59 – 63.
4. Jaya DS, *et al*. Anthropometric indices, cord length and placental weight in newborns. *Indian Pediatrics*. 1995; 32(11): 1183-8.
5. Gupta SP, Bahl L and Dikshit SK. A study of placenta in relation to birth weight and gestational age. *Indian Journal of Paediatrics*. 1972; 39(296): 281-5.
6. Chung SO and Park KH. Clinical studies on Biometric of the Placenta. *Yonsei Medical Journal*. 1974; 15(2): 92-102.
7. Ashfaq M, Channa MA, Malik MA, Khan D. Morphological changes in human placenta of wet snuff users. *J Ayub Med Coll Abbottabad.* 2008; 20(2): 110-3.
8. Majumdar S, Dasgupta H, Bhattacharya K, Bhattacharya A. A study of placenta in normal and hypertensive pregnancies. *J Anat Soc India.* 2005; 54(2): 1-9.
9. Frisancho AR, Matos J, Bollettino LA. Influence of growth status and placental function on birth weight of infants born to young still-growing teenagers. *Am J Clin Nutr.* 1984; 40: 801–807.
10. Asgharnia M, Esmailpour N, Poorghorban M, Atrkar-Roshan Z. Placental weight and its association with maternal and neonatal characteristics. *Acta Med Iran.* 2008; 46: 467–72.
11. Manop Janthanaphan MD, Ounjai Kor-anantakul MP,Alan Geater Placental weight and its ratio to birth weight in normal pregnancy at Songkhlanagarind Hospital. *J Med Assoc Thai.* 2006; 89(2): 130-7.

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

The Effect of Green Tea Extract on Submandibular Salivary Gland of Methotrexate Treated Albino Rats: Immunohistochemical Study

Ali Sultan Al-Refai*, Ameera Kamal Khaleel, Shahen Ali*****

Abstract

Background and Objectives: Methotrexate had been used for many years and complications usually encountered during treatment especially in cancer patients. The aim of the present study was to determine the early-stage anti cytotoxic effects of green tea on the histology of the submandibular gland of rats treated by high single dose of methotrexate. **Materials and methods:** The study included 30 Albino rats. Twelve animals were used in the pilot study to found the maximum toxic dose, and the other eighteen were divided into three groups, control group, methotrexate treated group, and methotrexate and green tea extract treated group. Submandibular gland excision was then performed. Histopathological examination was performed with hematoxylin-eosin, Masson's trichrome, and PAS staining. Cell proliferation was examined using the Ki-67 antibody and anti apoptotic effect was determined based on Bcl-2 staining. **Results:** In the methotrexate and green tea extract treated group a non significant change in the Ki 67 expression and a significant increase in Bcl- 2 expression were seen in comparison with the methotrexate treated group. **Conclusion:** Green tea aqueous extract in ratio 1:10 produced protection against methotrexate induced cytotoxicity in rat submandibular gland by increasing the expression of Bcl-2.

Keywords: Methotrexate; Salivary gland; Chemotherapy.

Introduction

Chemotherapy is one of the most widely used interventions for treatment of cancer. The cytotoxic effect of cancer chemotherapy is not selective for cancer cells, it also affects the normal tissues, the amount of the damage and its severity is based on the type, amount and duration of drug used to treat the disease (Al-Moula *et al*, 2012). Methotrexate, a folic acid antagonist, is widely preferred as a cytotoxic chemotherapeutic agent in the treatment of malignancies and some autoimmune diseases, but the efficacy is limited due to its side effects (Klareskog *et al*, 2004). It was revealed that the systemic oxidative stress is an important factors background of the methotrexate induced toxicity. Methotrexate causes differential toxic effects on lipid peroxidation by significant reduction in glutathione levels leads to a reduction of effectiveness of the antioxidant enzyme defense system, sensitizing the cells to reactive oxygen species. Therefore, the antioxidants protect against oxidative stress and prevent damage to cells (Sener *et al*, 2006).

Some natural products, such as green tea is demonstrated to have a protective role against oxidative stress (Yapar *et al*, 2009; Braicu *et al*, 2013). Green tea from the plant Camellia Sinensis is rich in antioxidant polyphenolic flavonoids (Bajerska *et al*,2011; Chandra *et al*,2011). Epigallocatechin gallate (EGCG), a green tea polyphenol, possesses potent antioxidant, anti-apoptotic, anti-inflammatory, and autoantigen-inhibitory properties. It is therefore extensively studied for probable health benefits against oxidative

Author's Affiliation: *Asstt. Prof., **Asstt. Prof., ***Lecturer, College of Dentistry, Hawler Medical University, Erbil, Kurdistan , Iraq.

Reprint's Request: Dr. Ali Sultan Al-Refai, Assistant Professor, BDS, MSc, Oral Biology, Ph.D, Anatomy, College of Dentistry, Hawler Medical University, Erbil, Kurdistan, Iraq.

E-mail:

stress-related diseases, and could normalize abnormal cell proliferation of the salivary gland (Gillespie *et al*, 2008).

Methotrexate has been used for many years, but because of the side effects, there is a continuous search to lessen its toxicity, for this reason the current study was aimed to investigate the potential anticytotoxic effect of green tea on the submandibular glands of Albino rat treated with a high single dose of methotrexate. As variables to evaluate the grade of damage or protection, we used histological and immunohistochemical investigations to clarify its effect on cell proliferation and apoptosis.

Materials and Methods

Animals and Study Design

Thirty healthy Albino rats of 250± 25 mg weight obtained from the animal house, College of Medicine, Hawler Medical University, were used for the study and kept under laboratory conditions and housed in a temperature-controlled environment (21-24°C), maintained on a 12 hr light /12 hr dark cycle and given free access to food and water. The research project was approved by the Research Ethics Committee at College of Dentistry, Hawler Medical University under protocol.

In the pilot study, twelve animals were used to find the effect of different doses of methotrexate (Ebewe 50mg/5ml) on the rat submandibular salivary gland. The animals received intramuscular injection of methotrexate, 10 mg/kg, 20mg/kg, 40mg/kg, and 80mg/kg body weight respectively. In the second day of the experiment, all the animals were anesthetized by i.p. administration of 0.5 ml/kg b.w. ketamine (Murti and Kumar , 2012) and biopsies from the submandibular glands of the animals were taken, fixed and processed for H&E.

The Results of the Pilot Study

Results showed that when there was increase in the dose of methotrexate there was increase in the cytotoxic effect. After 48 hours, the methotrexate showed acute effect, the microscopical picture of rat's submandibular gland treated by 10mg/kg or 20mg/kg b.w. of methotrexate showed areas of hemorrhage and spaces surrounded the duct system. Acinar and duct cells swelling with involution and disfigured lobular structure of some acini, and the granules in acinus cell cytoplasm appear larger than normal (Figure-1 A, B). But in the submandibular gland of rats treated by 40mg/kg b.w. of methotrexate, some acini showing loss of their normal architecture, the lining cells of the acini were indistinct and showed numerous cytoplasmic vacuolations with scattered small and dark pyknotic nuclei of different size, and marked vasodilatation of the blood capillaries. The ducts become dilated with discontinuity of their epithelial lining in some areas with a morphological change in the striated ducts represented by shrinkage of the ductal cells that lead to change of these cells from columnar to cuboidal cells (Figure-1 C).

The result of pilot study also showed that the maximum toxic effects of the drug were seen in the submandibular glands of rats treated by 80mg/kg b.w. of methotrexate, as seen in Figure 3. For this reason the other eighteen animals were randomly divided into three groups (six each):

Group 1: Control group, they did not receive any things.

Group 2: Methotrexate (80mg/kg) -non green tea extract, treated group.

Group 3: Methotrexate (80mg/kg) and green tea extract, treated group.

Daily method of tea making (household preparation) from green tea (Alwazah Swan Brand 100% pure Nett 225g) was used to prepare aqueous extracts. The aqueous extract was daily prepared in ratio 1:10 with the consideration of the absorption coefficient of green tea leaves (which is 2). Five grams of dried green tea leaves were grinded to pieces of diameter lower than one mm were poured with 60 ml of boiling water, and time was

Figure 1: (A) A Photomicrograph of the Submandibular Gland of Rat Treated by 10mg/kg b.w. Methotrexate, Showing Areas of Hemorrhage Suround the Straiated Ducts Specially the Large One (A1,H&E x100; A2,H&E x400). (B) Submandibular Gland of Rat Treated by 20mg/kg b.w. Methotrexate, Showing Stasis of Acini Secretions within the Ducts (B1, H&E x 100). Some Times Seepage of the Duct Secretion Outside the Duct Causing Shrinkage of its Epithelium. Acinar Cells Swelling which Contain Large Granules in their Cytoplasms are also Seen (Large Arrow) with Enlargement of the Granular Convoluted Tubules (Small Arrow) (B2, H&E x 400).(C) Submandibular Gland of Rat Treated by 40mg/kg b.w. Methotrexate, the Acini Showing Loss of their Normal Architecture(C1,H&E x 100). Cytoplasmic Vacuolation of Some Cells (Arrow) which Showing Different Cell Sizes and Nuclear Sizes with Marked Congestion of the Blood Capillaries. The ducts Become Highly Dilated with Discontinuity of their Epithelial Lining in Some Areas (C2, H&E x 400)

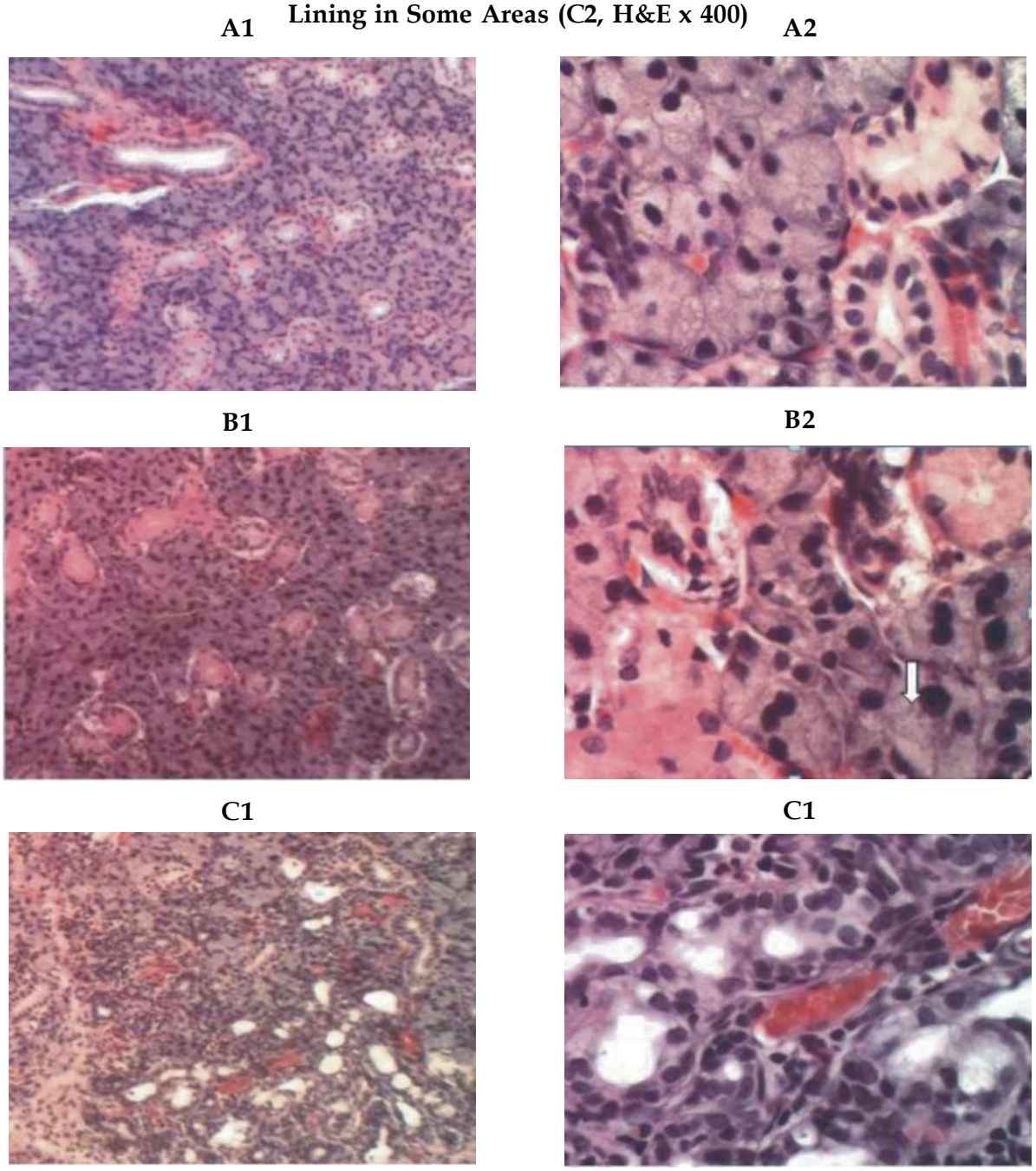
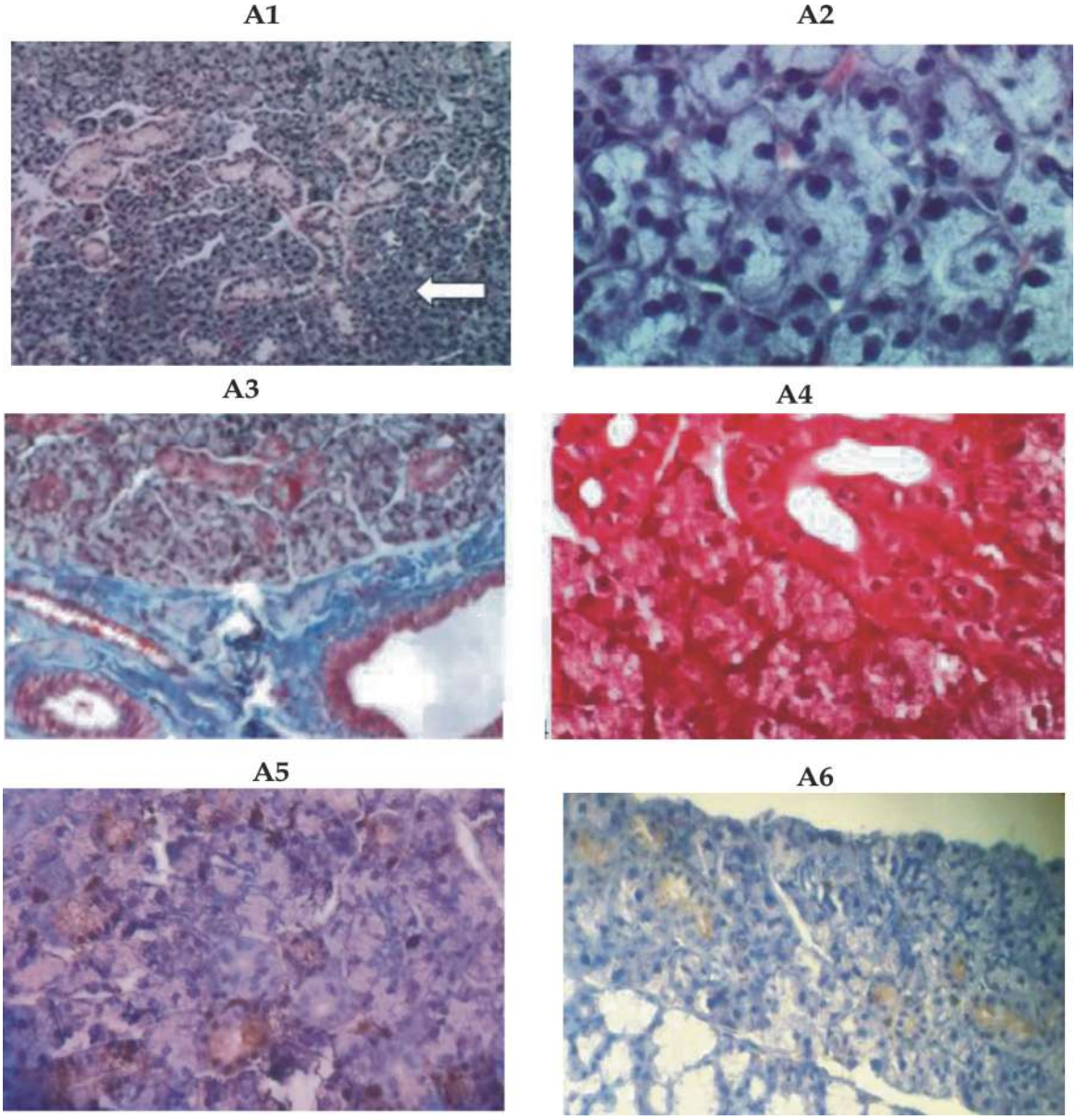


Figure 2: Photomicrographs of Rat Submandibular Gland of Control Group showing Regular Structured Acini (Arrows) (A1, H&Ex100; A2, H&Ex400). The Collagen Fibers are Distributed in the Stroma between the Acini and Duct (A3, Trichrome x100). Strong Positive PAS Reaction in Striated Duct (Upper Arrow) and Acini (Lower Arrow) which is Observed more at their Basement Membrane (A4, PASx400). (A5) Moderate Ki 67 Immuno Reactivity in Nuclei of Cells of Ducts and Some Acini. (A6) Moderate Cytoplasmic Reaction to Bcl-2 in Duct's Cells (Immunohistochemistryx400).



given for the extraction to cool down and then used (Armoskaite *et al*, 2011). The extract of green tea (1ml/250g b.w) was administered orally two times daily by intragastric gavage needle, starting three days before methotrexate injection and continues until the last day of the experiment. While the methotrexate - non green tea extract treated group, they received distilled water orally two times daily. In the second day of the experiment, the animals were anesthetized and biopsies from the submandibular glands of the animals were taken. Samples were then fixed in neutral buffered 10% formalin, processed for H&E, Masson's trichrome(Bancroft and Gamble,2002),

PAS(Bancroft and Gamble,2002) and for immunohistochemical analysis using Bcl-2 and Ki-67 immunolabeling.

Immuohistochemical Staining

Immunostaining for Ki-67 and Bcl-2 was performed using monoclonal Mouse Anti-Human Ki-67 Antigen, Clone MIB-1, Code No. M 7240 staining system, and a monoclonal Mouse Anti-Human BcL-2 Oncoprotien Clone 124 Code No 1587 ready to use N-series primary antibody, for use with Dako EnVision ™, EnVision ™ double staining and LASAB ™ 2 systems. The staining procedure sections of the instructions included with each detection system were followed. Positive and negative controls were run simultaneously with biopsy specimen.

Positive cells expressing Ki67 were identified by a brown precipitate in the nucleus except in mitotic cells, where the chromosomes and the cytoplasm are labeled, while Bcl-2 was demonstrated brown cytoplasmic staining. To ensure the objectivity of the analysis, the evaluation was carried out by 2 independent observers. Five sections were randomly chosen for each animal. Approximately 1000 cells from cell population were counted by two observers at a magnification of 400x (Olympus, Japan) and the percentages of ki 67 and Bcl- 2 positive cells were calculated. The level of Ki-67 and Bcl-2 expression was evaluated according to the scoring system of Seleit *et al* (2010). The application of this system gives a score ranging from 0 to 3 for both degree of positivity: percentage of positively stained cells [(absent: < 1%), (mild: 1 - 10%), (moderate: 10 - 50%), (strong: > 50%)]. The Kruskal-Wallis test was used to compare the results of Ki 67 and Bcl 2 staining. Level of significance was set as p d" 0.05.

Results

Anatomical and Microscopical Features of Rat's Submandibular Salivary Gland in the Control Group: Careful removal of the skin of the neck and the face reveal the presence of the salivary glands. Rat sublingual glands are located together with the submandibular glands on the sides of the neck between the submandibular lymph nodes and the sternum. Both glands were included within a common connective tissue capsule. They were separated from each other by fine connective tissue septa. The submandibular gland in the control group was composed of crowded serous acini only and not more types that in most mammals (Figure 2), there are no mucous or serous demilunes, the serous acini had narrow lumen and lined by pyramidal cells with pale basophilic granular cytoplasm and basal rounded nuclei. The duct system consists of intercalated duct, granular convoluted tubules, striated and excretory ducts.

The Submandibular Salivary Gland in the Methotrexate (80mg/kg) -Non Green Tea Extract, Treated Group: Microscopically, severe focal areas of acini destruction can be seen especially near the large ducts, swelling and increase in the sizes of some acini were also seen. The serous cells showed deeply stained atrophied nuclei, different cell sizes (anisocytosis), and different nuclear sizes(anisonucleosis). Giant nuclei and marked congestion of the blood capillaries were also seen. The cytoplasmic vacuolation in acinar and ductal cells were increased with destruction in some duct walls(Figure 3).

The Submandibular Salivary Gland in the Methotrexate (80mg/kg) and Green Tea Extract, Treated Group: Microscopical picture revealed marked improvement in cells of acini as well as cells of ducts lining, and the acini relatively preserved their shape. The numbers of vacuoles decreased and well formed striated ducts were also detected. They restored their basal striations and had intact epithelial lining. The intercalated ducts were noticed in between the acini. The granular convoluted tubules were lined by simple columnar epithelium with eosinophilic cytoplasm and basal rounded nuclei. Blood vessels were seen around these ducts, no congestion or areas of hemorrhage were seen (Figure 4).

Figure 3: A Photomicrograph of a Section in Submandibular Gland of Rat Treated by 80mg/kg b.w. Methotrexate, Showing Swelling (Upper Arrow) and Disarrangement of Some Acini (Lower Arrow) (A1,H&Ex100). Severe Cellular Changes with Irregular Configuration of the Acini and Detached Acinar Cells, Some Cells Showed Increased Nuclear Cytoplasmic Ratio, Hyperchromatic Nuclei and Extreme Anisonucleosis, Interacinar Oedema and Signs of Intracellular Vaculation (A2,H&Ex400). The Collagen Fibers Seen Distributed in the Stroma in between the Acini and Duct, with Destruction in Duct Wall, Arrow (A3, Trichrome x400). PAS - Stained Tissue Showing Duct Cells with Increase in Nuclear Cytoplasmic Ratio, Dense Nuclei and Lost Cytoplasm (Arrow), Acinic Cells Shows Vaculation with Loss of Some Nuclei (A4, PASx400).(A5) Mild Ki 67 Immuno Reactivity (Arrow) in Nuclei of Few Cells (Immunohistochemistryx400).(A6) Negative Cytoplasmic Reaction to Bcl-2 (Immunohistochemistryx400).

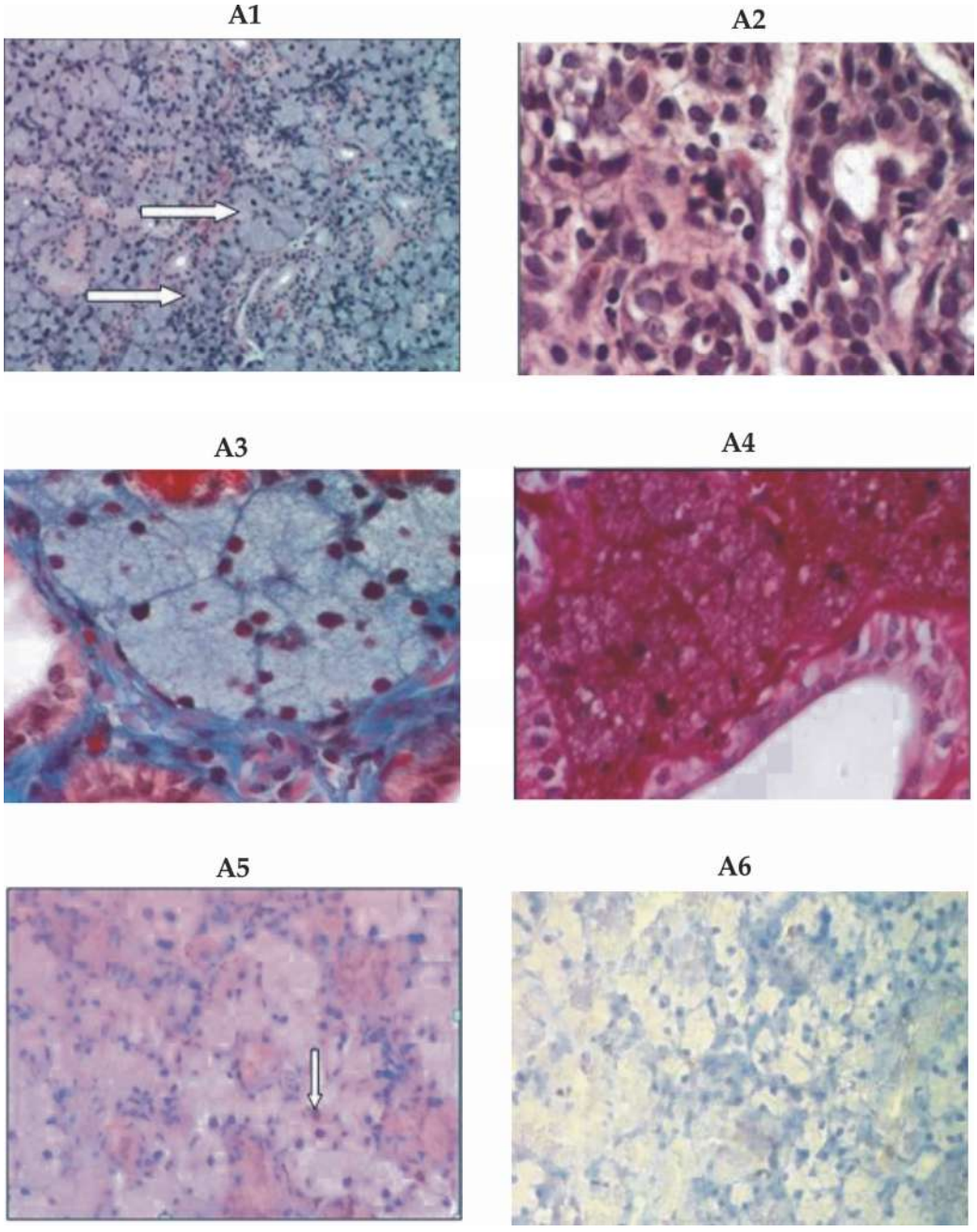
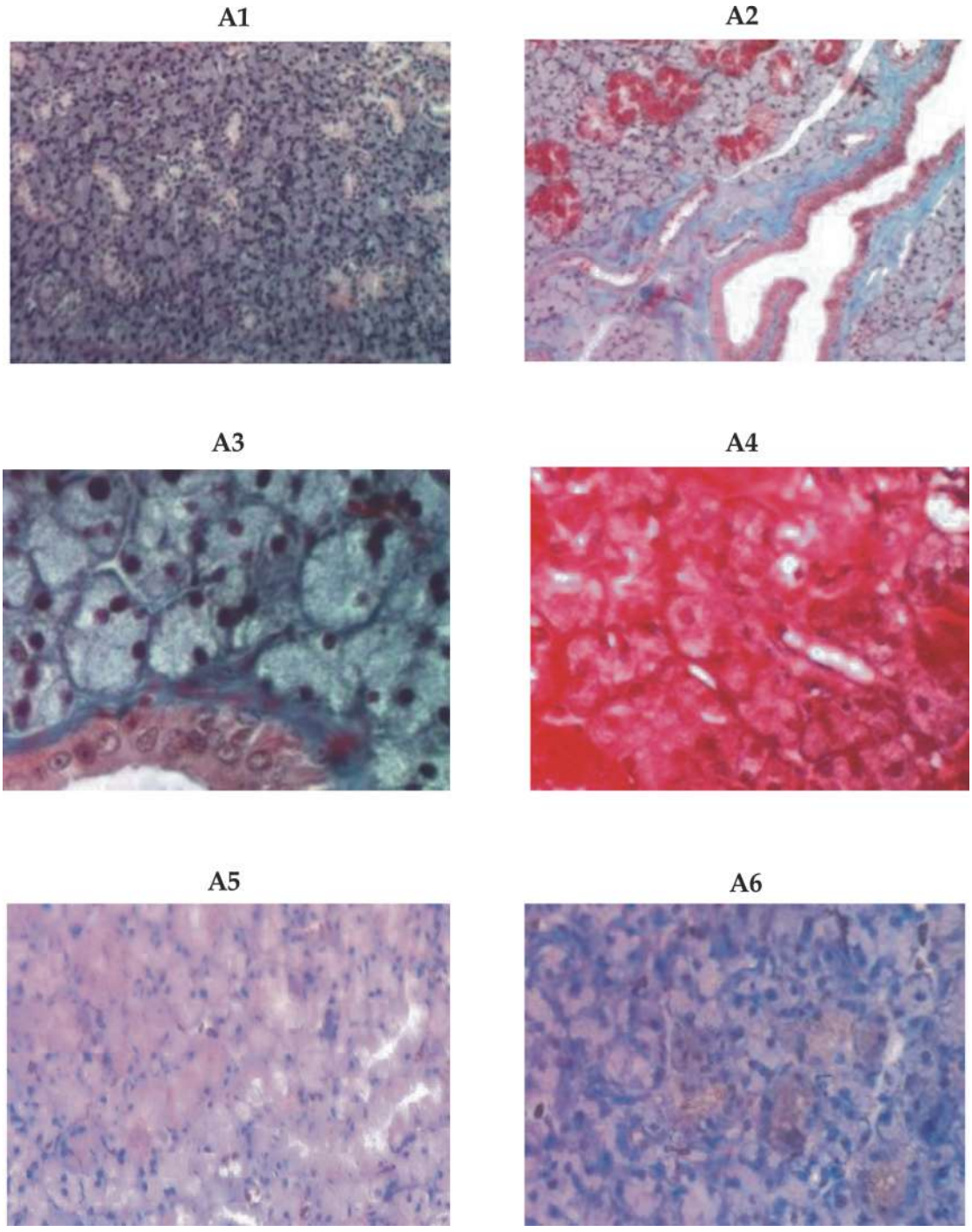


Figure 4: A Photomicrograph of a Section in Submandibular Gland of the Methotrexate and Green Tea Extract Treated Group Showing Well Formed Acini and Duct Lining (A1,H&Ex100), the Collagen Fibers are Distributed in the Stroma between the Acini and Ducts, Intact Multiple Granular Convoluted Tubules and Intact Duct Lining are Seen, no Congested Blood Vessels were Found (A2 Trichrome x100, A3, Trichrome x400). Strong Positive PAS Reaction in the Ducts and Acini with more Concentration at their Basement Membrane, Arrow (A4, PASx400). (A5) Mild Ki 67 Immuno Reactivity in Nuclei of Some Acinar Cells (Immunohistochemistryx400).(A6)Moderate Positive Cytoplasmic Reaction in Duct's Cells to Bcl-2 (Immunehistochemistry x400).



Histopathological examination of the submandibular glands that were excised from the rats in all three groups showed no evidence of collagen fiber breakdown, fibrosis, or an increase in inflammatory cells. A PAS positive reaction produces an intense magenta color, mainly indicating the presence of glycoprotein and glycogen. Only sections in the submandibular salivary gland of control albino rat's and in the methotrexate and green tea extract treated group showed strong positive PAS reaction, appear in both ducts and acini which is observed more at their basement membrane (Figure 2,Figure 4).

Immunohistochemical Results: The Ki-67 immunostaining results in the methotrexate and green tea extract treated group showed mild positive Ki-67 immunoreaction in the nuclei of acinar and ductal cells. This may suggest both the undamaged parenchymal and ductal cells attempts for regeneration and proliferation(Figure 4). There was a statistically significant difference ($P< 0.05$) present between group 1 (22.166±2.483) and the other two groups in terms of the rate of proliferation, but a statistically no significant difference ($P>0.05$) was observed between group 2 (1.246±0.624) and 3 (1.966±0.628).

The Bcl-2 immunostaining results in the methotrexate and green tea extract treated group showed moderate positive Bcl-2 immunoreaction in the cytoplasm of most ductal cells. There was a statistically significant difference present ($P< 0.05$) between group 2 (0.466±0.320) and the other two groups in terms of the rate of anti apoptosis, but a statistically no significant difference($P > 0.05$) was observed between groups 1 (17.31 ±3.538) and 3 (19.75 ±4.239).

Discussion

The result showed that the rat's submandibular gland contain serous acini only, this results disagree with that of Ahmad (2007) in which they found that it was mixed salivary gland, but agree with the result of Miclaus *et al* (2009). They reported that the acini of submandibular glands of rats had the feature of serous acini, similar but not identical with those from parotid gland. In addition of excretory channels present in mammalian salivary glands, in submandibular gland of the rats there's one more type of channels, named granular channels. Cells from walls of granular channels contain numerous polymorphous intracytoplasmatic granulations which suggest that they have an intense secretory activity. Authors added that granulations from cytoplasm of cells from granular channels are different from those of the acini structure and their secretion is muco-proteic. By mixing serous secretions of acini with that muco-proteic of the granular channels cells, resulted in a mixed final secretion which make us to consider submandibular gland of the rat a mixed particular gland.

The salivary glands have become a useful investigative tool for the study of basic problems in pharmacology (Rafah *et al*, 2006). Despite the low mitotic rate, the salivary gland tissue loses its function regularly with significant reduction of saliva production after exposure to chemotherapeutic doses like methotrexate, a folic acid antagonist (Jensen *et al*, 2003). In the present study, one dose of injected methotrexate had adversely affected the histological structure of the rat submandibular glands, this could be due to suppression and/or disruption of protein synthesis through depletion of folate co-factors, this can lead to formation of cytolysosome, may be an evidence of the distinctive process of apoptosis, the apoptotic bodies which are found in small numbers in normal tissues are greatly increased in tissues which have been subjected to chemotherapy (Al-Moula *et al*, 2012). Damaging of the salivary gland (acinar and ductal cell vacuolization, apoptosis in the acinar cells with pyknosis in the nuclei) following methotrexate treatment might be also related to the free radical damaging effect. These free radicals released during the intracellular metabolism of methotrexate, which interacts with the cell membrane, causing membrane lysis and release of major scavenger enzymes

(glutathione based enzymes) such as (glucose 6-phosphate dehydrognase and glutathione reductase) into the serum. This affects the physiological level of this antioxidant enzyme in serum and gives an indication of an adverse effect of methotrexate on cellular integrity (Hsu *et al*, 2006). But Wolff *et al* (1989) found that 18 hour after (15 mg/kg, i.p.) methotrexate administration no salivary effects could be detected consistently and this dose does not exert an acute cytotoxic effect on tissue with a slow turnover rate like the salivary glands.

The result also showed that the severity of pathological changes increased as chemotherapeutic doses increased. This comes in agreement with the result of Ozel *et al*, 2010).The enlargement of the acini might be due to dysfunction of the gland and disturbed salivary secretion leading to accumulation of the salivary secretion in the acini following by its swelling. Some ducts showed dilation with retaining secretion in their lumen, this dilation of ducts suggested the pathological effect of methotrexate on myoepithelial cells embracing them with failure of expelling the secretion into the oral cavity as a result of glandular dysfunction leading to xerostomia. Mahmoud *et al* (2012) found that the rabbits when injected with (15mg/kg, i.p.) methotrexate for two weeks, the same result appear.

The extent of damage in the salivary gland tissue of rats treated by high dose of methotrexate depend on the size of the affected duct, this comes in agreement with the results of (Al-Moula *et al*, 2012). The accumulation of secretory material within the cytoplasm of acinar cells led to degenerative changes in the acinar cells that frequently resulted in cell death and replacement of secretory cells by connective tissue elements. Granular changes were observed in the cytoplasm of acinar cells in the submandibular gland of the rats in methotrexate treated group. The secretory granules in the serous cells include many proteolytic enzymes and heavy metals. This condensation in the granules indicates non-specific cytoplasmic degeneration and shows reversible cellular damage (Ozel *et al*, 2010).

To our knowledge no investigation using green extract to fight the cytotoxic effect of a high toxic dose of methotrexate on rat submandibular salivary gland has been performed. In the present study, the combined treatment of methotrexate and green tea extract ameliorated the histological changes in salivary gland tissue induced by methotrexate alone. The antioxidant properties, reactive oxygen species scavenging, and cell function modulation of flavonoids could account for the large part of pharmacological activity of green tea (Hafez, 2006).

A recognized indicator of cell mitotic activity is Ki67, an increase in Ki67 expression is indicative of increased cell mitotic activity and proliferation. Ki-67 monoclonal antibodies detect a nuclear antigen expressed exclusively at the level of cells in the proliferation phase (phases G1, S, G2 and mitoses), but not in the G0 phase. Therefore, Ki-67 antibodies allow for the imunohistochemical determination of the tissue growth fraction (Lazari *et al*,2010). Bcl-2 is important effecter gene during the apoptosis process and has been reported to prolong the survival of cells by specifically inhibiting apoptosis. The balance between mitotic activity and apoptosis is thought to regulate normal development (Ribeiro *et al*,2005).

The data presented in our study showed that green tea cause non significant increase the expression of Ki 67, and significant increase in the antiapoptotic activity in the glands in relation with the methotrexate group. Previous report found that EGCG significantly inhibited TNF-á induced apoptosis in human salivary gland acinar cells in vitro. This inhibitory effect on apoptosis could be due to modulation of MAPK signal to interrupt an apoptotic signal (Hsu *et al*, 2007). So with the administration of green tea extract that protects salivary gland from apoptosis by increasing the anti apoptotic activity, it can be used as a criterion and can be used as, a novel approach to decrease the cytotoxicity

of methotrexate on submandibular gland.

Conclusion

The beneficial effect of green tea aqueous extract in ratio 1:10 against methotrexate induced cytotoxicity in the submandibular salivary glands of rats stem from its antiapoptotic effect and therefore can be used as a protective natural product to the salivary glands in individuals undergoing cancer therapy.

References

1. Al-Moula AD, Al-Mashhadane F, Mammdoh JK. Effects of 6-mercaptopurine on salivary glands in rabbit. *Al-Rafidain Dent J.* 2012; 12(2): 266-273.
2. Klareskog L, Vander Heijde D, de Jager JP, Gough A, Kalden J,Sanda M. Therapeutic effects of the combination of etanerceept and methotrexate compared with each treatment alone in patients with rheumatoid arthritis: Double blind randomized controlled trial. *Lancet*. 2004; 363: 675-681.
3. Sener G, Eksioglu-Demiralp DE, Cetiner M. L-carnitine ameliorates methotrexate-induce oxidative organ injury and inhibit leukocyte death. *Cell Biol Toxicol*. 2006; 22: 47-60.
4. Yapar K, Avus KC, Glo O, Oruc E, Yalc E. Protective effect of royal jelly and green tea extracts effect against cisplatin-induced nephrotoxicity in mice: a comparative study. *Journal of Medicinal Food*. 2009;12(5):1136-1142.
5. Braicu C, Ladomery MR,. Chedead VS, Irimie A, Berindan-Neagoe I. The relationship between the structure and biological actions of green tea catechins. *Food Chemistry*. 2013; 141(3): 3282–3289.
6. Bajerska J, Wozniewicz M, Jeszka J, Drzymala-Czyz S, Walkowiak J. Green tea aqueous extract reduces visceral fat and decreases protein availability in rats fed with a high-fat diet. *Nutrition Research.* 2011; 31: 157–164.
7. Chandra A, Choudhury SR, De N, Sarkar M. Effect of green tea (Csmellia sinensis L) extract on morphological and functional changes in adult male gonads of albino rats. *Indian Journal of Experimental Biology.* 2011; 49: 689-697.
8. Gillespie K, Kodani I, Dickinson DP, Ogbureke KU, Camba AM, Wu M, Looney S, Chu TC, Qin H, Bisch F, Sharawy M, Schuster GS, Hsu SD. Effects of oral consumption of the green tea polyphenol EGCG in a murine model for human Sjogren's syndrome, an autoimmune disease. *Life Sci.* 2008; 83: 581–588.
9. Murti K, Kumar U. Enhancement of wound healing with roots of Ficus racemosa L. in albino rats. *Asian Pac J Trop Biomed.* 2012; 2(4): 276–280.
10. Armoskaite V, Ramanauskiene K, Maruska A *et al*. The analysis of quality and antioxidant activity of green tea extracts. *Journal of Medicinal Plants Research.* 2011; 5(5): 811-816.
11. Bancrof J, Gamble A. Theory and Practice of Histological Techniques, 5th ed. New York and London: Churchil, Livingstone; 2002, 165-180.
12. Seleit IA, Asaad N, Maree A, Abdel Wahed M. Immunohistochemical expression of p53 and Ki-67 in cutaneous lupus erythematosus. *J Egypt Women Dermatol Soc.* 2010; 7(1): 5-15.
13. Ahmad SM. Histological and immunohistochemical study of the submandibular salivary gland in experimentally induced diabetes mellitus in adult male albino rats. MSc Thesis, Zagazig University; 2007.
14. Miclaus V, Oana L, Ober C, Rus V, Pestean C. Observations concerning features of submandibular gland secretion in rats. *Lucrari Stiinlifice Medicina Veterinara.* 2009; 9(2): 382-386.
15. Rafah A, Alham DH, Tahani A. Effect of natural apple cider vinegar on morphometric changes of salivary glands in hydroxyl urea treated mice. *J Edu Sci.* 2006; 18(3): 62-77.
16. Jensen SB, Pedersen AM, Reibel J, Nauntofte B. Xerostomia and hypofunction of salivary glands in cancer therapy. *Support Care Cancer.* 2003; 11: 207-225.
17. Hus PC, Hour TC, Liao YF, Hun YC, Chang WH, Kao MC,Tsay GJ, Hung HC, Liu GY. Increasing ornithine decarboxylase activity is another way of prolactin preventing methotrexate induced apoptosis; Crosstalk between ODC and Bcl-2. *Apoptosis.* 2006; 11(3): 389-399.

18. Wolff A, Moreira JE, Marmary Y, Fox PC. Lack of acute effects of methotrexate on rat parotid salivary gland function. *Arch Oral Biol.* 1989; 34(2): 109-15.
19. Ozel O, Aycicek A, Kenar F, Aktepe F, Sargin R, Yillmaz MD, Fevzi Sefa Derekoy FS. Histopathologic changes in the rabbit submandibular gland after 5-fluorouracil chemotherapy. *Turk J Med Sci.* 2010; 40(2): 213-220.
20. Mahmoud EF, Mahmoud MF, Abd Al Haleem MA. Royal jelly ameliorates oxidative stress and tissue injury in submandibular salivary gland of methotrexate treated rabbits: Immunohistochemical study. *Journal of American Science.* 2012; 8(11): 501-508.
21. Hafez SE. Using green tea in controlling structural disorders of radiation in some organs in male albino rats. *J Drug Res.* 2006; 27: 90-97.
22. Lazari D, Taban S, Sporea I, Dema A, Cornianu M, Lazar E, Goldisi A, Vernic C. Ki-67 expression in gastric cancer. Results from a prospective study with long-term follow-up. *Romanian Journal of Morphology and Embryology.* 2010; 51(4): 655–661.
23. Ribeiro DA, Salvadori DMF, Marques MEA. Abnormal expression of bcl-2 and bax in rat tongue mucosa during the development of squamous cell carcinoma induced by 4-nitroquinoline 1-oxide. *Int J Exp Pathol.* 2005; 86(6): 375-381.
24. Hus S, Dickinson DP, Qin H, Broke J, Ogbureke K, Winger JN, Walsh DS, Bollag WB, Stoppler H, Sharawy M, Schuster G. Green tea polyphenols reduce autoimmune symptoms in a murine model for human Sjogren's syndrome and protect human salivary acinar cells from TNF-alpha induced cytotoxicity. *Autoimmunity.* 2007; 40: 138-147.

Indian Journal of Anatomy

Library Recommendation Form

If you would like to recommend this journal to your library, simply complete the form below and return it to us. Please type or print the information clearly. We will forward a sample copy to your library, along with this recommendation card.

Please send a sample copy to:

Name of Librarian

Library

Address of Library

Recommended by:

Your Name/ Title

Department

Address

Dear Librarian,

I would like to recommend that your library subscribe to the **Indian Journal of Anatomy**. I believe the major future uses of the journal for your library would be:

1. As useful information for members of my specialty.
2. As an excellent research aid.
3. As an invaluable student resource.
4. **I have a personal subscription and understand and appreciate the value an institutional subscription would mean to our staff.**
5. Other

Should the journal you're reading right now be a part of your University or institution's library? To have a free sample sent to your librarian, simply fill out and mail this today!

Stock Manager

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II, Mayur Vihar, Phase-I

Delhi - 110 091 (India)

Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205

E-mail: redflowerppl@gmail.com, redflowerppl@vsnl.net

Website: www.rfppl.org

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

A Study of Anatomical Variations in the Arteries Supplying Gut Derivatives

Deepa G.*, Shivakumar G.L.**

Abstract

Anatomical variations in the branching pattern and distribution of the arteries supplying gut derivatives is very important especially for surgeons undertaking surgeries in the abdominal region. Anatomical variations contribute to the misinterpretation and leads to major postoperative complications. The present study was carried out in 32 adult cadavers (5 females and 27 male cadavers) which were used during routine dissection for undergraduate medical students. The course and branches of all the ventral branches of aorta was traced. Any arterial variation was observed and recorded. Anatomical variations related to the trifurcation of coeliac trunk, origin of the inferior phrenic artery, origin of the left gastric artery, origin of the accessory hepatic artery and the origin of the accessory right colic artery were noted and documented. In two cases, left colic artery was absent and inferior mesenteric artery gave rise to 3-4 sigmoid branches. The present study highlights on the importance of arterial variations in the abdomen which should not be ignored. Hence, the accurate knowledge of such variations is important in carrying out surgical procedures in the abdomen safely and also in the interpretation of angiographic reports.

Keywords: Arteries; Anatomic variation; Abdomen; Aorta; Cadaver.

Introduction

Abdominal aorta begins at the median, aortic hiatus of the diaphragm, anterior to inferior border of the 12th thoracic vertebra and the thoracolumbar intervertebral disc.[1] The major branches of abdominal aorta supply nearly all the organs in the abdominal cavity. The three ventral branches supply all the gut derivatives. The coeliac trunk which is the first ventral branch of abdominal aorta supplies the foregut derivatives. The trunk divides into the left gastric artery, the common hepatic artery and the splenic arteries. This trifurcation was first described by Haller in 1756. This 'Tripus Halleri' was and is still being considered to be the normal appearance of the coeliac trunk.[2] Superior mesenteric artery supplies the mid gut[3] which include the portion of digestive tract extending from the duodenum at the opening of thee bile duct to the junction between the right two third and left one third of the transverse colon.[4] Inferior mesenteric artery is usually smaller in caliber than the superior mesenteric artery which arises at about the level of L3, 3 to 4 cm above the aortic bifurcation and supplies the left one third of transverse colon, the entire descending and sigmoid colon, the rectum and the upper part of the anal canal up to the pectinate line.[1] Anatomical variations in the branching pattern and distribution of these arteries is very important especially for surgeons undertaking surgeries in the abdominal region.

Arterial variations in the ventral branches of abdominal aorta have been described by many authors. Anatomical variations in the coeliac trunk were first classified by Adachi in 1928, based on 252 dissections of Japanese cadavers, where 6 types of divisions of the coeliac trunk and superior mesenteric artery

Author's Affiliation: *Assistant Professor, **Professor, Department of Anatomy, Navodaya Medical College, Raichur Karnataka-584103, India.

Reprint's Request: Dr. Deepa G., Assistant Professor, Department of Anatomy, Navodaya Medical College, Raichur, Karnataka-584103, India.

E-mail: drdeepagadwal@gmail.com

Figure 1: Showing Coeliac Trunk Dividing into Left Hepatic Artery, Splenic Artery and Gastroduodenal Artery. Right Hepatic Artery was Observed to Be Arising from Superior Mesenteric Artery. Lha: Left Hepatic Artery. Ct: Coeliac Trunk. Sa: Splenic Artery. Gda: Gastroduodenal Artery. Sma: Superior Mesenteric Artery. Rha: Right Hepatic Artery.

were described.[5] Variations in the branching pattern of superior mesenteric artery is also been observed. In about 50% of cases, the marginal artery which is the result of anastomosis of the branches of superior mesenteric artery and inferior mesenteric artery may be discontinuous because of the failure in the anastomosis between the left and right colic arteries.[6] There have been reports of cases where the right and middle colic

Figure 2: Showing Inferior Phrenic Artery Arising from Left Gastric Artery. LGA: Left Gastric Artery. IPA: Inferior Phrenic Artery.

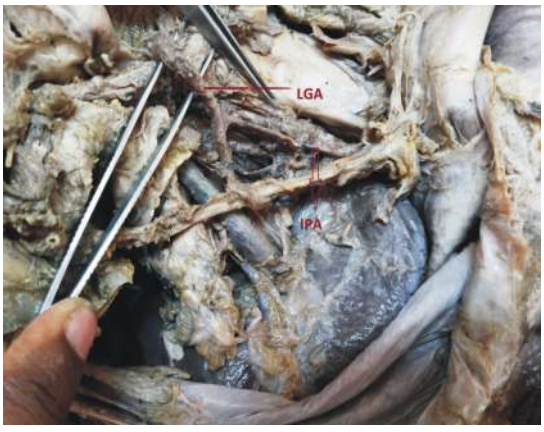
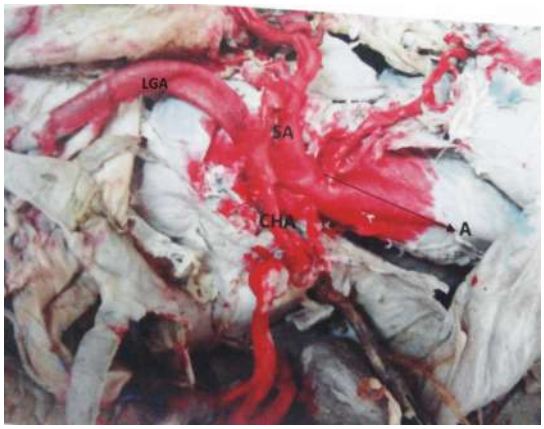


Figure 3: Showing Left Gastric Artery Arising from Abdominal aorta. Coeliac Trunk is Observed to Be Bifurcating into Splenic Artery and Common Hepatic Artery. A: Aorta. SA: Splenic Artery. CHA: Common Hepatic Artery. LGA: Left Gastric Artery.



arteries were absent leaving the entire supply of the colon to the inferior mesenteric artery.[7] Anatomical variations contribute to the misinterpretation and leads to major postoperative complications.[8]

Hence, the proper knowledge on these variations is necessary in order to avoid surgical injury and help the surgeons to minimize the aforementioned complications related to abdominal surgery and also to avoid improper imaging during surgery.

Figure 4: Showing Accessory Hepatic Artery Arising from Superior Mesentric Artery. SMA: Superior Mesenteric Artery. Ac. HA: Accessory Hepatic Artery.

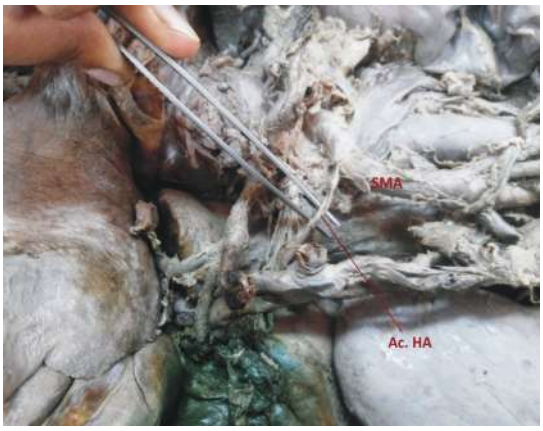
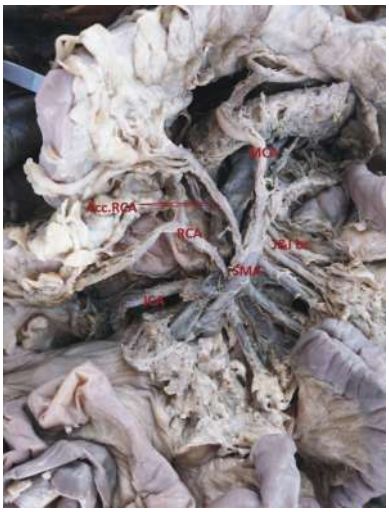


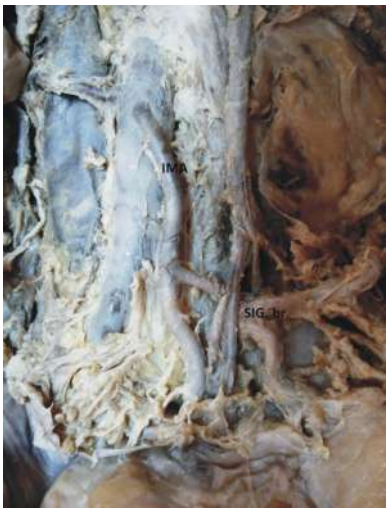
Figure 5: Showing Accessory Right Colic Artery Arising from Superior Mesenteric Artery. SMA: Superior Mesenteric Artery. MCA: Middle Colic Artery. RCA: Right Colic Artery. ICA: Iliocolic Artery. Acc.RCA: Accessory Right Colic Artery. J&I br.: Jejunal & Iliac branches.



Materials and Methods

The present study was carried out in 32 adult cadavers (5 females and 27 male cadavers) which were used during routine dissection for undergraduate medical students in Department of Anatomy, Navodaya Medical College, Raichur. Anterior abdominal wall was opened, and peritoneum and viscera were carefully separated. The course and branches of all the ventral branches of aorta was traced. Any arterial variation was observed and recorded.

Figure 6: Showing Absence of Left Colic Artery and Inferior Mesenteric Artery Giving Arise to only Sigmoidal Branches



Results

The following arterial variations were observed:

1. In one case, the normal trifurcation of coeliac trunk was not seen. Instead coeliac trunk divided into splenic artery, left hepatic artery and gastroduodenal artery. The left gastric artery was seen to be arising from left hepatic artery and right gastric artery originated from gastroduodenal artery. Right hepatic artery was observed to be arising from superior mesenteric artery. (Fig 1)
2. In two cases, the inferior phrenic artery was observed to be arising from left gastric artery instead of abdominal aorta.(Fig 2)
3. In one case, left gastric artery originated from directly from abdominal aorta. The coeliac trunk bifurcated into splenic artery and common hepatic artery. (Fig 3)
4. In three cases, accessory hepatic artery was noticed to be arising from superior mesenteric artery. (Fig 4)
5. In one case, superior mesenteric artery gave origin to accessory right colic artery. (Fig 5)
6. In two cases, left colic artery was absent and inferior mesenteric artery gave rise to 3-4 sigmoid branches. (Fig 6)

Discussion

The careful identification of anatomical vascular variations is very important and is of great importance for surgeons. Angiography is not routinely recommended, but it should

be mandatory when complex surgeries are planned.[9] Reported variations in the branching pattern of the coeliac trunk include absence of the trunk[10], presence of collateral vessels[11] and bifurcation of the coeliac trunk.[12] Such variations in the pattern of branching of the coeliac trunk may predispose to iatrogenic injury during surgical procedures such as total pancreatectomy[13] and resection of tumours of head of pancreas.[14] Knowledge of this variable anatomy may be useful in planning and executing radiological interventions such as celiacography[15], chemoembolization of hepatic tumours.[16]

The embryological basis related to these variations was made by Tandler and Morita. Tandler provided an embryological explanation for the variations in the coeliac trunk and the superior mesenteric artery in 1904. The ventral branches develop initially from the abdominal aorta as paired vessels, which then coalesce in the median line to form the four roots for the gut. The four roots are connected by a ventral longitudinal anastomosis.[17,18] Normally, the first root forms the left gastric artery, the second root forms the splenic artery and third root forms the common hepatic artery. The first three roots coalesce by the longitudinal anastomotic trunk to form the coeliac trunk. Superior mesenteric artery develops from the forth root, which migrates caudally with the ventral migration of the gut.[19,20] According to Morita, the anomalous ramification of the coeliac trunk and superior mesenteric artery are due to the primitive ventral splanchnic arteries and their longitudinal anastomosis.[18,21]

Various studies were carried out regarding arterial variations. An angiographic study by Kostelic *et al* reports the prevalence of an accessory hepatic artery as 33%.[22]. Lippert and Pabst reported that they determined accessory left hepatic artery in 11% of cases, with varying origins, more often from the left gastric artery or the coeliac trunk.[23] Molmenti *et al* reported that the presence of replaced right hepatic artery in 15-20% and accessory left hepatic artery in 35% of cases.[24] Malnar *et al* reports that the coeliac trunk divides into the common hepatic artery and splenic artery, where left gastric artery originates separately, proximal to the bifurcation of the coeliac trunk in 72% of cases in their study on Croatian cadavers.[25] A similar case was observed in the present study. Judy J Moon *et al* reported right hepatic artery branching from superior mesenteric artery.[26] Similar case was also noticed in the present study. This anatomical variant must be identified prior to procedures such as laparoscopic cholecystectomy to prevent vascular or biliary damage.[27] Preoperative detection of an aberrant right hepatic artery in prospective transplant donors and recipients is essential for the proper management of living donor liver transplantation, as transplantation of the right lobe is heavily favored over the left, and the aberration affects the safety of both donor and recipient.[28]

Cavdar *et al* reported a case, in which the left inferior phrenic artery and left gastric artery arose from the long coeliac trunk (4.3 cm) via a common trunk[29] Piano *et al* stated that the right and left inferior phrenic artery occasionally originated as a common trunk from the aorta, coeliaco-mesenteric system or adrenorenal system.[30] He observed that inferior phrenic arteries were usually paired(left and right) and their origin were summarized as follows – a) the abdominal aorta itself (61.6%), b) ventro-visceral arteries (coeliaco-mesenteric system of aorta) including coeliac trunk (28.2%) and left gastric artery (2.9%), c) the latero-visceral arteries (adreno-renal system of the aorta) including the middle adrenal artery (2.9%), and renal artery(4.3%).

Vascular variations of superior mesenteric artery have been studied by many authors. Right gastroepiploic artery has been reported to arise from superior mesenteric artery.[31] In the present study superior mesenteric artery was observed to give rise to accessory right colic artery. Middle colic arteries have been found to originate from the coeliac trunk at a rate of 0.5 – 1%.[32,33] An anomalous middle

colic artery originating from the common hepatic artery has been reported by Wadwa *et al*.[34] Gracia-Ruiz *et al* reported the presence of double middle colic artery in their cadaveric study.[35]

The arterial variations in the abdomen are usually asymptomatic. But the complete knowledge about vascular variations is very important during planning major abdominal surgeries and imaging procedures. Variations in the coeliac trunk may become important in patients who undergo coeliacography for gastrointestinal bleeding and the coeliac trunk compression syndrome, prior to an operative procedure or trans catheter therapy and for chemoembolization of pancreatic and liver tumours.[36] During minimally invasive or complicated hepatobiliary surgery an understanding of arterial variants in the lesser omentum is necessary if serious problems are to be avoided.[9] The knowledge about the hepatic arterial variation is very important for surgical gastroenterologists and interventional radiologists for preoperative planning and intraoperative imaging during procedures like liver transplantation, cholecystectomy, gastrectomy, hiatal hernial repair, trans arterial chemotherapy and hepatic arteriography.[37]

Conclusion

The present study highlights on the importance of arterial variations in the abdomen which should not be ignored. Hence, the accurate knowledge of such variations is important in carrying out surgical procedures in the abdomen safely and also in the interpretation of angiographic reports.

References

1. Susan Standring. Posterior abdominal wall and retroperitonium. Gray's Anatomy-The anatomical basis of clinical practice, 39th ed. Philadelphia: Elsevier Churchill Livingstone; 2005, 1118.
2. Hemanth K, Garg S, Yadav TD, Sahni D. The hepato-gastro-phrenic trunk and the hepato-spleno-mesenteric trunk: A rare anatomical variation. *Tropical Gastroenterology*. 2011; 32(1): 56 – 59.
3. Moore KL and Persuad TVN. Clinically Oriented Embryology. 4th Ed. New Delhi: Saunders–Elsevier; 2004.
4. Moore KL and Dalley AF. Clinically oriented Anatomy. 5th Ed. Baltimore: Williams and Wilkin; 2006.
5. Adachi B (Das Arterien system der Japaner). Vol 2. Verlag der Kaiserlich-Japanischen Universitat Zu Kyoto, 1928. Japanese.
6. Basmajian JV. Grant's Method of Anatomy, 10th Ed. Baltimore: William and Wilkins; 1980.
7. Ano zeng Oyono Igiri, *et al*. The pattern of Arrangements and Distributions of the Superior Mesenteric Artery in a Nigerian Population. *Int J Morphol*. 2010; 28(1): 33–36.
8. Hiatt J R, Gabby J, Busutil R W. Surgical Anatomy of the hepatic arteries in 1000 cases. *Ann Surg*. 1994; 220: 50-2.
9. Lippert H, Pasbst R. Arterial variations in man. Classification and frequency. JF Bergmann Verlag. *Munchen*. 1985; 34-35: 71-3.
10. Van Damme JP and Bonte J. The branches of the celiac trunk. *Acta Anat*. 1985; 122(2): 110-4.
11. Cavdar S, Gurbuz J, Zeybek A, Sehirli U, Abik I and Ozdogmus O. A variation of coeliac trunk. *Kaibogaku Zasshi*. 1998; 3(5): 505-8.
12. Ucerler H and Asli A. Multiplicity of the variations in the ventral branches of abdominal aorta. *Ital J Anat Embryol*. 2001; 111(1): 15-22.
13. Van Damme JP and Bonte J. Vascular anatomy in abdominal surgery. Stuttgart: Thieme; 1990, 27-86.
14. Lin J. Celiomesentric trunk demonstrated by 3 – dimensional contrast – enhanced magnetic resonance angiography. *Hepatobiliarypancreas Dis Int*. 2005; 4(3): 472-4.
15. Gluck E, Gerhardt P and Schroder J. Value of vascular morphology for the selection of catheters in selective celiacography and mesentericography. *Rofo*. 1983; 138(6): 664-9.
16. Aigner KR and Gailhofer S. Celiac axis infusion and microembolization for advanced stage III/ IV pancreatic cancer – a phase II study on 265 cases. *Anticancer Res*. 2005; 25(6c): 4407-12.

17. Tandler J. Uberdie varietaren der arteriacoelia caunderen Entwiklung. *Anat Hefte*. 1904; 25: 473-500 (In German).
18. Morita M. Reports and conception of 3 anomalous cases of the superior mesenteric arteries. *Igaku Kenkyu*. 1935; 9: 1993-2006.
19. Adachi B. Das Arterien system Der Japaner, Band II. Kyoto: verlag der keiserlich Japanischen Universitatzu Kyoto, Maruzen publishing co; 1928; 28, 38, 54. (In Japanese).
20. Shomura S, Emua S, *et al*. Anatomical study on the branches of coeliac trunk (IV) comparison of the findings with Aachi's classification. *Acta Anat Nippon*. 1991; 66: 452-61.
21. Limura A, Oguchi T, Shibata M, Takahashi T. An anomalous case of the hepatic artery arising from the superior mesenteric artery. *Okajimas Folia Anat Jpn*. 2007; 84(2): 61–66.
22. Lurie AS. The significance of the variant left accessory hepatic artery in surgery for proximal gastric cancer. *Arch Surg*. 1987; 122: 725-8.
23. Couinaud C. L artere hepatique In: foie: Etudes Anatomiques et chirurgicales. Paris: Masson et cie; 1957; 146-86.
24. Molmenti EP, Pinto PA, Kiein J, Klein AS. Normal and variant arterial supply of the liver and gall bladder. *Pediatr Transplant*. 2003; 7: 80-82.
25. Malnar D, Klasan GS, Miletic D, *et al*. Properties of the CT – anatomical study. *Coll Anthropol*. 2010; 34: 917 -21.
26. Judy J Moon *et al*. Right hepatic artery branching off the superior mesenteric artery and its potential implications. *International J of Anatomical Variations*. 2009; 2: 143–145.
27. Nicholson T, Travis S, Ettles D, Dyst J, Sadman P, Wedgewood K, Roysten C. Hepatic artery angiography and embolization for hemobilia following laparoscopic cholecystectomy. *Cardiovascular Intervent Radiol*. 1999; 22: 20-24.
28. Orguc S, Tercan M, Bozoklar A, Akyildiz M ET AL. Variations in hepatic veins: helical computerized tomography experience in 100 consecutive living donors with emphasis on right lobe. *Transplant Proc*. 2004; 38: 2727-2732.
29. Hollinshed WH. Anatomy for surgeons. Vol. 2 (The Thorax, Abdomen and Pelvis. The stomach, duodenum, pancreas and spleen), 2nd ed. New York: Harper and Row Publisher; 1961, 590.
30. Piano Dx, Ohtsuka A, Murakmi T. Typology of abdominal arteries, with special references to inferior phrenic arteries and their oesophageal branches. *Acta Med Okayama*. 1988; 52: 189–96.
31. Sakamoto H, *et al*. An anomalous right gastroepiploic artery arising from the superior mesenteric artery. *Surg Radiol Anat*. 1999; 21(4): 283-6.
32. Michels NA, *et al*. Variant blood supply to the descending colon, rectosigmoid and rectum based on 400 dissections. Its importance in regional dissections: a review of medical literature. *Dis Colon Rectum*. 1965; 8: 51- 278.
33. Amonoo kuofi HS, *et al*. Anomalous origin of colic arteries. *Clin Anat*. 1995; 8: 288-93.
34. Wadhwa S, Barua MP. Anomalous middle colic artery originating from common hepatic artery: a case report. *Clin Anat.* 2008; 21: 798–9.
35. Garcia–Ruiz A, Milsom JW, Ludwig KA, Marchesa P. Right colonic arterial anatomy. Implications for laparoscopic surgery. *Dis Colon Rectum*. 1996; 39: 906 – 11.
36. Antony Sylvan D'souza, *et al*. Anatomical variations in the branches of the coeliac trunk. *Journal of Clinical and Diagnostic Research*. 2012; 6(3): 333–335.
37. Narayanaperumal Mugunthan *et al*. Anatomical variations in the arterial supply of liver. *International J of Anatomical Variations.* 2012; 5: 107–109.

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Sexual Dimorphism in Foot Length and its Comparison with Height and Weight

Syed Sadiqali Abbasali*, Herekar N.G., Phad V.V.*****

Abstract

The study was done to find sexual dimorphism in foot length and to correlate foot length with height and weight. The present study was done on 150 medical college students, 68 males and 42 females of 19-23 year of age. Foot length was determined from foot sketches taken in anatomical position. Height and weight was measured with standard height and weight measuring instrument. It was found that male foot length was proportionately larger than female foot length for given height. There was statistically significant correlation between height and foot length in males and females. Results of present study are helpful for estimation of stature from lower extremity parts and establishing personal identity in forensic sciences.

Keywords: Foot length; Forensic science; Height; Stature; Weight.

Introduction

Human foot has a very complex structure formed by union of tarsals, metatarsals and phalanges. Studies have been done on foot size for improvements in footwear design. Also some studies have been done in forensic science to determine stature from foot length. Many studies documented that in proportion to stature male have larger foot than female. Their exist lots of variation in foot size and it shows sexual dimorphism, which changes with region of study.

Ossification of bones in the foot occurs earlier than long bones so height could be more accurately predicted from foot measurement as compared to that from long bones inadolescence age. In medico-legal cases, estimation of stature from extremities and their parts plays an important role in establishing personal identity. So present study was undertaken to find out the correlation between foot length with height and weight of an individual and to derive regression formulae to estimate the height from the foot length.

Materials and Methods

Present study was performed on 150 medical college students, 68 male 82 females of age between 19 years and 23 years. Students having no any physical deformity were selected for the study. By using standard instruments height in centimetre and weight in kilograms, of students were recorded. Foot outline of right and left foot were drawn in normal anatomical position. To find foot length, on sketches, first long axis of foot was determined by joining two points, first most distal point on second toe and second most distal point at the heel end. Line passing through these two points was long axis of foot. Perpendicular was drawn to this axis passing by touching most distal point at front, which may be 1st toe or 2nd toe, whichever was longer. Another perpendicular was drawn at most distal point at heel end. The distance between these two perpendiculars was foot

Author's Affiliation: *Assistant Professor, **Professor and Head, ***Assistant Professor, Dept. of Anatomy, Government Medical College, Miraj, Maharashtra, India.

Reprint's Request: Dr. Syed Sadiqali Abbasali, Mamta Nursing Home, 39/B, Subhashnagar, Miraj, Maharashtra, India.

E-mail: sadiqalisyed3@gmail.com

Table 1: Showing Height Groups and Average Foot Length in Males and Females

Height (Group) (cm)	Mean Height	Average Foot Length Male (cm)	Average Foot Length Female (cm)
140-144	142		20.6
145-149	147		21.8
150-154	152		23.1
155-159	157	23.7	23.1
160-164	162	24.7	23.9
165-169	167	25.6	24.6
170-174	172	26	24.8
175-179	177	26.2	
180-184	182	27.8	

Graph 1: Comparasion of Male and Femele Foot Length with Height

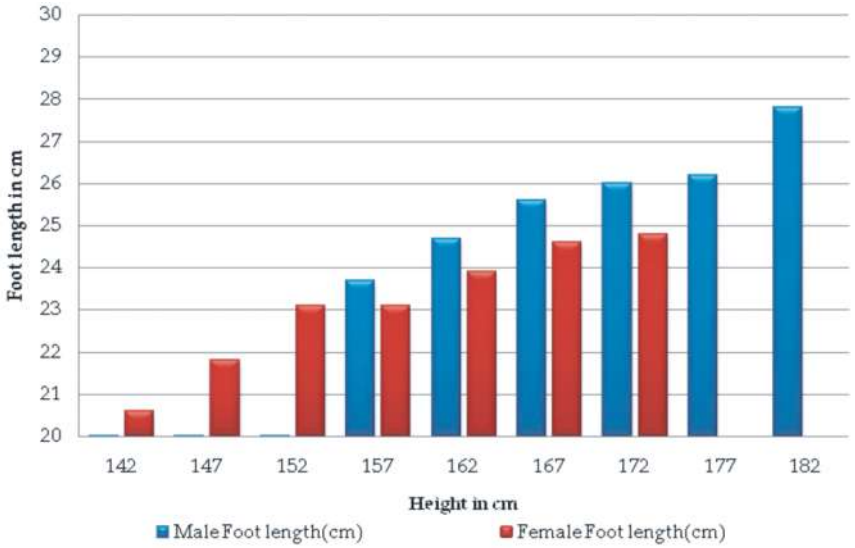


Table 2: Showing Weight Groups and Average Foot Length in Males and Females

Weight (Group)	Mean Weight	Average Foot Length Female	Average Foot Length Female
35-39	37		22.5
40-44	42	23.65	23.4
45-49	47	30.3	23.2
50-54	52	25.7	23.4
55-59	57	25.5	23.7
60-64	62	26.2	23.7
65-69	67	25.1	25
70-74	72	26.8	22.9
75-79	77	25.3	
80-84	82	25.6	
85-89	87	26.4	
90-94	92		24

length. Foot length was measured in right and left foot and average was drawn which was correlated with height and weight.

Observations and Results

The observations were done on 68 males and 82 females, total 150 students. The data obtained for height and average foot length was divided into small groups of height for comparison as shown in the Table 1.

The data in table 1 is plotted graphically as seen in Graph 1.

From above graph it was noted that both in

Graph 2: Comparasion of Male and Femele Foot Length with Weight

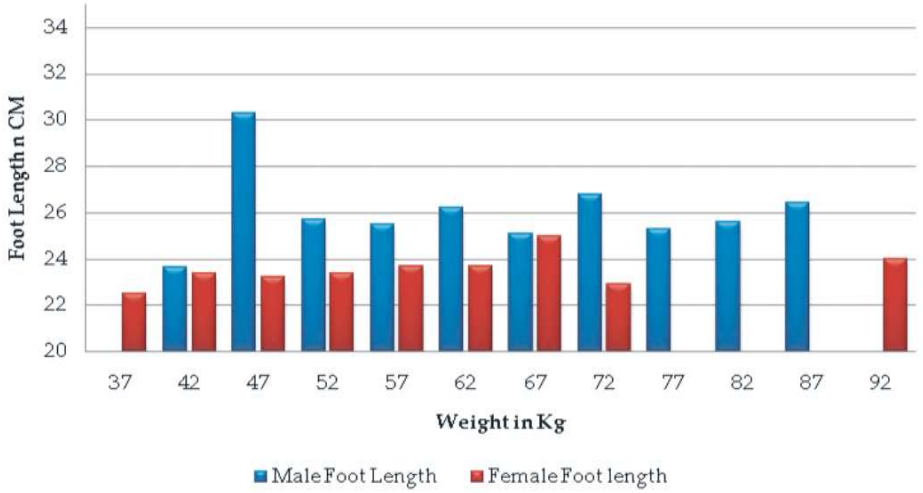
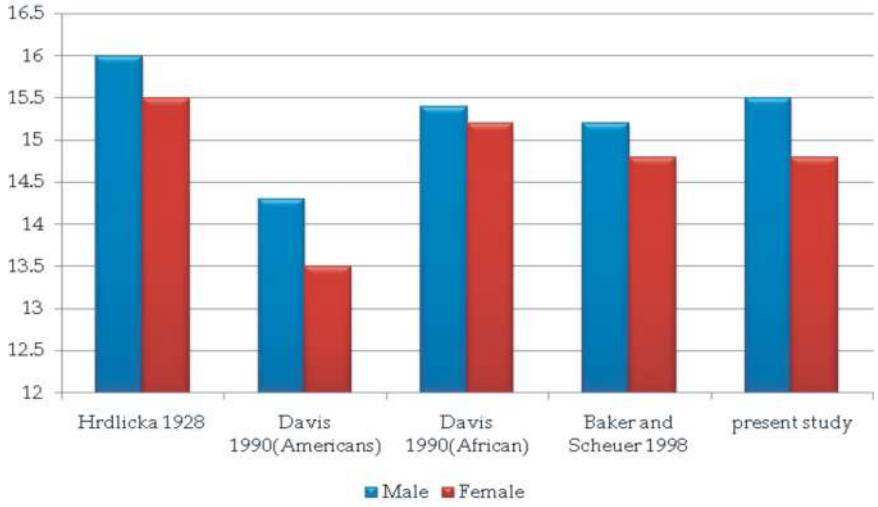


Table 3: Showing Correlation Coefficient(r) and Regression Equation in Different Studies

	Correlation coefficient(r)		Regression equation	
Studies	Male	Female	Male	Female
Mansur DI et al.	0.688	0.578	Y = 100.1+2.74x (Right Foot) Y= 100.2+2.738x (Left Foot)	Y = 96.31+ 2.66x (Right foot) Y = 96.40+2.66x (Left Foot)
Dr. Sonali Khanapurkar et al.	0.702	0.645	Y=72.8+3.7x	Y =90.0+ 3.2x
Patel S.M. et al.	0.65	0.80	Y=75.45+3.64x	Y=75.41+3.43x
Jakhar J.K. *et al*	0.725	0.719	Y=82.597+3.572 x	Y= 65.406+4.057 x
Present study	0.691	0.694	Y=71.095+3.834x	Y=62.054+4.112x

Graph 3: Foot Length as a Proportion of Stature in Male and Female



males and females, as height increases average foot length also goes on increasing. It was also observed that for any given height, average foot length of male was always larger than average foot length of female.

Foot length as a proportion of stature for men and women was also calculated. It was

15.2 in males and 14.8 in females.

The data obtained for weight and average foot length was also divided into small groups of weight as shown in the Table 2.

The data in table 2 is plotted graphically as seen in Graph 2.

From above graph it was observed that, there was no increase in foot length with increase in weight. So there was no any correlation between weight and foot length. But it was observed that for any given weight, average foot length of male was always greater than average foot length of female.

Discussion

Various studies had done earlier on comparison of foot length in male and female.Ross and Ward (1982)[1], Robbins (1986)[2], Giles and Vallandigham (1991)[3], Barker and Scheuer (1998)[4], Wunderlich and Cavanagh (2001)[5] all documented that male foot length was proportionately larger than female foot length for given height. The findings of present study correlate with all above studies.

Foot length as a proportion of stature for men and women was 15.2 in males and 14.8 in females. This proportion was compared with previous study as shown in graph 3. It is observed that findings of present study correlateswith findings of with Baker and Scheuer[4].The proportion cited by Hrdlicka[6] was on higher side.Daniel M. T *et al*[7], in their study, quoted findings of Davis and Bake. According to Davis and Bake,foot length as a proportion of stature for men and women in American population was 14.3 and 13.5 and in African population was 15.4 and 15.2. Findings of present study were correlating with findings of African population.

Correlation coefficient (r) was calculated from entire data. It was 0.691 (p=0.00) for males and 0.694 (p=0.00) for females. So it means that there is strong positive correlation between height and foot length in males and females. These findings are also correlating with previous studies.

The regression equation for height and foot length was calculated. It was Y=71.095+3.834X in males and Y=62.054+4.112X in females, where Y is the height and X is the mean foot length. Comparison of correlation coefficient and regression equation drawn in different studies was compared with Mansur DI *et al*[8], Dr. Sonali Khanapurkar *et al*[9], Patel S.M. *et al*[10], Jakhar J.K. *et al*[11] seen in Table 3.

Correlation between weight and foot length was not observed in present study, but average foot length of male was always greater than average foot length of female for given weight group.

Conclusion

1. Male foot length is proportionately larger than female foot length for given height.
2. There was statically significant correlation between height and foot length in males and females.
3. The regression equation for height and foot length was Y=71.095+3.834x in males and Y=62.054+4.112x in females, where Y is the height and x is the mean foot length.

References

1. Ross WD, Ward R. Human proportionality and sexual dimorphism. In: Hall RL, editor, Sexual dimorphismin Homo sapiens: A question of size. New York: Praeger; 1982, 317–361.
2. Robbins LM. Estimating height and weight from size of footprints. *J Forensic Sci*. 1986; 31: 143–152.
3. Giles E, Vallandigham PH. Height estimation from foot and shoeprint length. *J Forensic Sci*. 1991; 36: 1134–1151.
4. Barker SL, Scheuer JL. Predictive value of human footprints in a forensic context. *Med Sci Law*. 1998; 38: 341–346.
5. Wunderlich RE, Cavanagh PR. Gender

differences in adult foot shape: Implications for shoe design. *Med Sci Sports Exerc*. 2001; 33: 605–611.

6. Hrdlicka A. The full-blood American negro. *Am J Phys Anthropol*. 1928; 12: 15–33.
7. Daniel MT *et al*. Sexual dimorphism in foot length proportionate to stature. *Annals of Human Biology*. 2005; 32(1): 44–59
8. Mansur DI, Haque MK, Sharma K, Karki RK, Khanal K, Karna R. Estimation of Stature from Foot Length in Adult Nepalese Population and its Clinical Relevance. *Kathmandu Univ Med J*. 2012; 37(1): 16-9.
9. Khanapurkar S *et al*. Estimation of stature from the measurement of foot length, hand length and head length in Maharashtra region. *Indian Journal of Basic & Applied Medical Research*. 2012; 1(2): 77-85.
10. Patel SM *et al*. Estimation of height from measurement of foot length in Gujarat region. *J Anat Soc India*. 2007; 56(1): 25-27.
11. Jakhar JK *et al*. Estimation of Height from Measurements of Foot Length in Haryana Region. *J Indian Acad Forensic Med*. 32(3).

Original Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

An Anatomic Study of Branching Pattern of Right Coronary Artery (RCA)

Charanjeet Kaur*, Navtej Singh, Jyotsna Singh***, Prithpal S. Matreja******

Abstract

Introduction: Malformations of position of ostia and origin of coronary arteries leads to potentially fatal significance unless diagnosed and treated surgically, because the coronary artery, and the myocardium it supplies, is fed with pulmonary rather than systemic blood. The present study aims to establish morphometry and pattern of right coronary artery with a special emphasis on its branching pattern, anastomoses and area of distribution. **Material and Methods:** The hearts of 25 adult human cadavers comprised the material for the study. The branching pattern of the Right Coronary Artery along with any variation were observed and noted down. Drawing of each artery was made and each specimen was photographed. The data was collected, finalized, analyzed & compared with the available data. **Results:** The observations from present study show that there is lot of variation in number, site of origin, and area supplied by the RCA. In 10 specimens a third coronary artery was seen arising from anterior aortic sinus just in front of the proper right coronary artery, 21 cases (84%) the posterior interventricular artery was a continuation of right coronary artery. In 21 specimens single conus artery was found out of which in 15 specimen conus artery was seen arising from RCA. In 18 specimens single marginal artery was found. **Conclusion:** The number of variations seen in branching pattern of RCA it becomes very difficult to assign the normal pattern.

Keywords: Coronary artery; Marginal artery; Conus artery; Variations; Branching pattern.

Introduction

Coronary arteries are the vasa vasorum of ascending aorta, right coronary artery arises from right coronary sinus (anterior aortic sinus) and left coronary artery arises from left posterior aortic sinus of ascending aorta.[1] Ostia of the coronary arteries are located in center of corresponding aortic sinuses. Malformations of position of ostia and origin of coronary arteries lead to high risk of sudden death.[2]

Clinically, the anomalies are arbitrarily divided into benign and malignant, based on their potential to cause myocardial ischaemia. The three most common benign anomalies are separate origins of left anterior descending and circumflex in left sinus of valsalva; origin of circumflex from right sinus of Valsalva or from right coronary artery; and ectopic origin of right coronary artery from aorta, a high origin being particularly prone to accidental cross clamping or side clamping, or transection during aortotomy. The most common malignant anomaly is the ectopic origin of right coronary artery from the left sinus of Valsalva.[3]

The right coronary artery, which in nine-tenths of individuals supplies most of diaphragmatic surface of ventricular mass, emerges from right coronary aortic sinus in upper part of right anterior surface of aortic

Author's Affiliation: *Demonstrator, Department of Anatomy, Gian Sagar Medical College, Ram Nagar, Rajpura, Patiala 140601, **Associate Professor, Department of Medicine, BPS Government Medical College for Women, Khanpur Kalan, Sonepat 131305, Haryana, ***Assistant Professor, Dept. of Anatomy, Govt. Medical College & Hospital, Sector-32, Chandigarh, ****Associate Professor, Department of Pharmacology, Gian Sagar Medical College, Ram Nagar, Rajpura, Patiala 140601, Punjab, India.

Reprint's Request: Prithpal Singh Matreja, Associate Professor, Department of Pharmacology, Gian Sagar Medical College and Hospital, Ramnagar, Patiala 140601, Punjab, India.

E-mail: drpsmatreja@yahoo.co.in

root. In many instances, two arterial orifices are found in this sinus, with the second orifice most often giving rise to infundibular, or conal, artery, but sometimes giving rise to artery of sinus node.[4,5]

Having emerged from its aortic sinus, right coronary artery occupies right atrioventricular groove. Its first part extends to right, or acute, margin of ventricular mass, where it gives rise to acute marginal artery, with several atrial branches taking origin from its cranial surface. It also gives rise in this part of its course to infundibular artery, if this vessel has not taken origin directly from the aorta, and in just over half the population, to artery supplying sinus node. The right artery itself then continues to encircle the vestibule of tricuspid valve, extending to cardiac crux. Throughout this course, artery gives rise to right inferior ventricular branches, which supply diaphragmatic wall of right ventricle.[6]

In nine-tenths of population, having reached crux, right coronary artery gives rise to inferior interventricular artery and artery to atrioventricular node, then continuing to supply a variable portion of diaphragmatic wall of left ventricle. This arrangement is called right ventricular coronary arterial dominance.[4]

Of the named branches of right coronary artery, infundibular or conal branch is present in approximately half the population. Patients with well-developed infundibular arteries have more extensive distribution to the anterior wall of right ventricle through preventricular and ventricular branches of this artery. In some individuals, the artery anastomoses with an infundibular branch of anterior interventricular artery, forming so-called arterial circle of Vieussens. [7]

The anterior ventricular branches, usually two or three, ramify towards cardiac apex, which they rarely reach unless right marginal artery is included in this group of branches. Anastomoses are found at apex between this artery and anterior interventricular artery.[8]

When right coronary artery is dominant, it gives rise to artery supplying atrioventricular node, typically from a U-loop that extends in tissue plane forming floor of triangle of Koch. This, artery in conjunction with septal perforating branches of anterior interventricular artery, supplies proximal right and left branches of atrioventricular conduction axis in nine-tenths of individuals, with sole supply by the nodal artery reported in one-tenth of cases. The inferior interventricular artery, also known as posterior descending artery or posterior interventricular artery, arises from right coronary artery in all of those nine-tenths of individuals with right coronary arterial dominance, and from circumflex artery in remaining one-tenth, latter feature allegedly being more common in males. Branches of this artery can meet parallel branches of right marginal artery, and perpendicular branches of anterior interventricular artery, in inferior atrioventricular groove and at apex.[4]

Perforating branches from the artery supply myocardium of inferior part of muscular ventricular septum, adjacent ventricular walls, and infero-septal papillary muscle of the mitral valve in those individuals with right coronary arterial dominance.[9] In rare cases, anterior interventricular artery can extend into inferior interventricular groove, taking over territory usually supplied by inferior interventricular artery.[10]

In most individuals it is right coronary artery that supplies inferior wall of right ventricle and inferior diaphragmatic portion of muscular ventricular septum. The branches of left coronary artery supply majority of the sternocostal walls of heart and obtuse margin of left ventricle.[5] In up to half population, ominant right coronary artery, in addition to supplying inferior interventricular artery, also supplies a significant part of diaphragmatic wall of left ventricle.[6] In these individuals, it is branches of right coronary artery that typically supply infero-septal papillary muscle of mitral valve, and sometimes supero-lateral muscle.[9] In case of an extremely dominant right coronary artery, with hypoplasia of circumflex artery, the branches of right coronary artery can supply all inferior wall of

left ventricle.[11]

A solitary right coronary artery can take two forms. The right coronary artery itself can continue beyond crux, run through left atrioventricular groove, and terminate as anterior interventricular artery. Alternatively, right coronary artery can give rise to main stem of left coronary artery, which can then take a retroaortic, interarterial, or prepulmonary course before branching into anterior interventricular or circumflex arteries.[12]

Unlike the situation in which one of coronary arteries takes an anomalous origin from aortic root, ectopic origin is generally considered a congenital malformation in its own right. Very rarely a coronary artery can take an ectopic course from a brachiocephalic artery, or from a branch of subclavian artery. The most frequent ectopic origin, nonetheless, is from the pulmonary trunk, or one of its branches.[13]

This has potentially fatal significance unless diagnosed and treated surgically, because the coronary artery, and the myocardium it supplies, is fed with pulmonary rather than systemic blood. The condition is generally know as the Bland-White-Garland Syndrome, and is estimated to involve one in every 300,000 liveborn infants.[14]

This is a group of very infrequent anomalies, generally found during angiographic exploration. The right coronary artery originating from left coronary artery, a few millimeters from its beginning, has been described. This location is in fact similar to that of right coronary artery originating in left aortic sinus, and strictly speaking, is a case of a single coronary artery with a left-sided origin.[15]

Anomalous origin of coronary arteries from opposite sinus is potentially serious especially among young subjects and when a vessel runs between aorta and pulmonary artery.[16] Circumflex branch of coronary artery originating in right side, is most common "benign" coronary anomaly and is not considered cause of ischemia or myocardial infarction.[17]

Anomalous aortic origin of a coronary artery from an opposite sinus of Valsalva is a rare and sometimes lethal congenital anomaly. Anomalous coronary ostia are a recognized cause of sudden death, especially associated with high-intensity exercise in young adults. Traditional diagnostic techniques, such as coronary angiography and, to a lesser extent, transesophageal echocardiography, are invasive and ultimately underused. Improvements in noninvasive diagnostic techniques, such as transthoracic echocardiography and CT angiography, have increased ability to easily and safely screen for condition, leading to increased rates of diagnosis. Sudden death is thought to be associated with restriction of flow down anomalous artery, causing myocardial ischemia and ventricular arrhythmias, especially when anomalous coronary artery courses between great vessels (aorta and pulmonary artery). At present, mechanisms that lead to myocardial ischemia are unclear, but several potential mechanisms have been proposed.[18]

The present study aims to establish morphometry and pattern of right coronary artery with a special emphasis on its branching pattern, anastomoses and area of distribution. This information is definitely useful for clinicians as anomalous patterns of artery are related with ischaemia, myocardial infarction, ventricular arrhythmias and sudden death.

Material and Methods

The study was conducted in the department of Anatomy Government Medical College, Patiala. The hearts of 25 adult human cadavers comprised the material for the study. The hearts were labeled from 1-25. Mediastinum was dissected. Hearts were taken out from the cadaver & Right Coronary Artery was located in the Coronary sulcus. The fat from the coronary sulcus was carefully removed to avoid damage to small branches. Right Coronary Artery was followed from its origin to termination by careful dissection. The branching pattern of the artery along with any

variation were observed and noted down. Drawing of each artery was made and each specimen was photographed. The data was collected, finalized, analyzed & compared with the available data. The diameter of artery was taken at its origin & at the right border of the heart and at the point where it enters in posterior interventricular sulcus and after giving right marginal branch.

Observations

The observations from present study show that there is lot of variation in number, site of origin, and area supplied by the RCA. In no case particular text book description was found. Therefore it is very difficult to assign the normal course, site of origin, girth, branches and area supplied

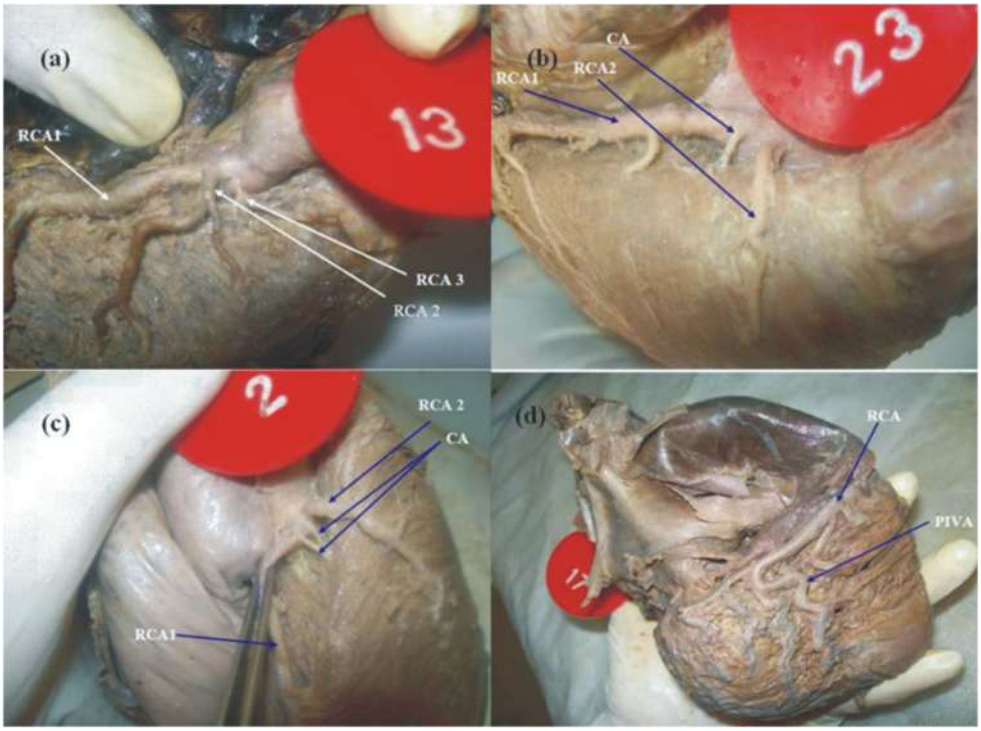
The branching pattern of the artery along with any variation was observed in all twenty five specimens. In all the specimens right coronary artery was seen arising from anterior aortic sinus.

Variations

There are two coronary arteries i.e. right and left in 15 specimens and in the other 10 specimens a third coronary artery was seen arising from anterior aortic sinus just in front of the proper right coronary artery. In these 10 cases 2 ostia were clearly defined in right aortic sinus. Out of 25 hearts studied RCA was seen arising from right anterior aortic sinus. In one case there were 3 independent arteries arising from aorta and in 9 cases there were two independent RCA arising from anterior aortic sinus where as in rest of cases there was single artery arising from anterior aortic sinus (Figure 1).

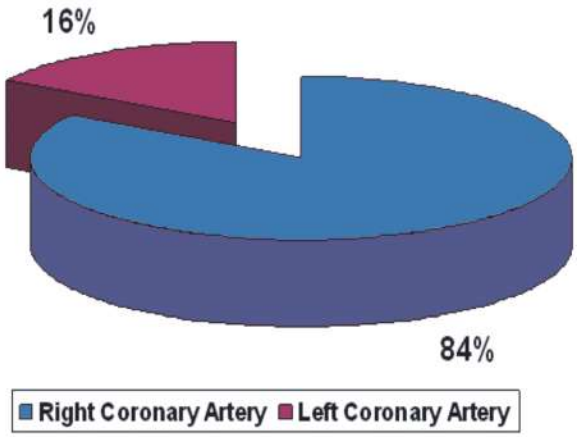
From the total 25 specimens studied SA nodal artery was arising from RCA in all 24 specimen and in specimen no. 1 this artery was found absent and could not be traced from

Figure 1: Variations in Heart (a): Heart showing 3 right coronary arteries (b): Heart showing 2 right coronary arteries with conus artery arising from right coronary artery (c): Heart showing 2 right coronary arteries and 2 conus arteries arising from anterior aortic sinus (d): Heart showing right coronary artery and posterior interventricular artery



Abbreviations used: RCA-Right coronary artery, CA-Conus artery, PIVA –Posterior interventricular artery

Figure 2: Dominant Circulation



LCA.

From the total 25 specimen in 21 cases (84%) the posterior interventricular artery was a continuation of right coronary artery and in 4 specimens (16%) it is a branch of left coronary artery (Figure 2). In 4 cases the RCA did not terminate as PIV artery. Out of these cases in one case the artery was seen running in posterior sulcus but finished before the crux where as in another case artery was seen running in the right half of right posterior sulcus. In other 2 cases major branch was not seen in coronary sulcus at all. In rest of 21 cases main branch crossed the crux to variable extents. As the artery occupied whole of the posterior coronary sulcus and was seen giving branches to whole of the sternocoastal surface. In 21 cases right dominance was present (Figure 1).

Mean diameter (Mean ±SD) (in mm) of right coronary artery at origin is 4.49±0.94, just before origin of right marginal artery is 4.11±0.93, just after giving right marginal artery is 3.55±1.06 and right marginal is 2.87±0.53.

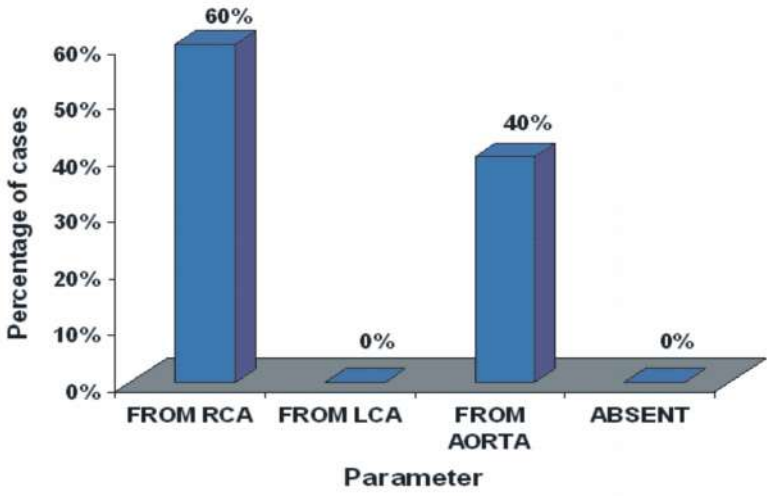
Conus Artery

In 21 specimens single conus artery was found out of which in 15 specimen conus artery was seen arising from RCA and in 6 specimen conus artery was seen arising from anterior aortic sinus (referred as third coronary artery). Conus branches varied from 1-3 in numbers (Figure 1 and 3).

In 3 specimens, 2 conus arteries were seen and out of these one specimen two conus arteries were seen arising directly from anterior aortic sinus. In the other two specimen's one conus branch was seen arising from anterior aortic sinus and second conus branch was seen arising from RCA (Figure 1).

In 1 specimen there were 3 conus arteries. One conus artery was seen arising from anterior aortic sinus (referred as third coronary artery) and 2 more conus arteries were seen arising from RCA.

Figure 3: Origin of Conus Arteries



Right Marginal Artery

Right marginal artery was present in all the specimens arising from the right coronary artery and the number varied from 1-3. In 18 specimens single marginal artery was found. Morphologically in one specimen marginal artery did not reached the proper right margin. In 6 specimens 2 marginal arteries were found. In 1 specimen 3 marginal arteries were present

Discussion

Knowledge of the normal and variant anatomy and anomalies of coronary circulation is an increasingly vital component in management of congenital and acquired heart disease. Several studies have been conducted by various authors on origin of coronary artery and found many variations.

In the present study the right coronary ostium was present in all the specimens in the right anterior aortic sinus in other study by Vlodaver *et al* (1972) the author observed that in most of cases (56%) the orifice of coronary arteries were situated in aortic sinus below supravalvular ridge and in 8% of cases the origin of right coronary artery occurred above supravalular ridge.[19]

In the present study, third coronary artery was present in 10 cases (40%) of the specimens studied where as in rest of cases conus artery was almost first branch. The results are quite similar to a study were the first and highest branch of the proximal segment of right coronary artery, the conus artery arose in 36% of the cases from a separate ostium (third coronary artery), in right aortic sinus of valsalva.[20] In another study by Kalpana R. the author dissected 100 hearts and observed the third coronary artery presents in 24% of the specimens.[21]

In the present study, 96% of specimens the sinuatrial node artery is a branch of right coronary artery is in contrast to a study were second branch of first segment of right coronary artery was sinuatrial node artery. The sinuatrial node artery arose from right coronary artery in more than 60% and from left coronary artery in less than 44% of the specimens.[22]

In other study the author observed in 51% of the specimen sinuatrial node artery arose from right coronary artery and 41% from left coronary artery. In 8% of the specimens the vessel arose from both right and left coronary artery.[23] Similarly, another study observed that in 54% of the cases the sinuatrial artery was a branch of right coronary artery and in 42% of the specimens the artery arose from left coronary artery and in 4% of the cases the sinuatrial node artery was seen arousing directly from aorta.[24]

The dominance of artery is determined by the posterior interventricular artery. It is termed right dominance if the posterior interventricular artery is a branch of right coronary artery and if the posterior interventricular artery is a branch of left coronary artery it is termed left dominance. In present study in 21 cases (84%) the posterior interventricular artery is a branch of right coronary artery hence it was right dominance. The results are similar to a study were the author observed posterior interventricular artery as a terminal branch of right coronary artery in 90% of the cases.[24] Similarly another study observed that 80% of the specimens showed right dominance and 11% of the cases showed left dominance.[25]

In the present study in 6 cases posterior descending artery was found to arise before the crux and contributed to the supply of left ventricle. Similar results have been found by James and he observed a vessel arising before the crux contributed to the supply of the left ventricle in 7 cases.[26]

It is concluded with the remark that from the number of variations seen in branching pattern of RCA it becomes very difficult to assign the normal pattern. The variations are very important for cardiologists and radiologists.

References

1. Datta AK. Essentials of human anatomy. Thorax and abdomen. 3rd Ed. Calcutta: Current Books International; 1994, 80–86.
2. Frescura C, Basso C, Thiene G, Corrado D, Pennelli T, Angelini A, *et al*. Anomalous origin of coronary arteries and risk of sudden death: a study based on an autopsy population of congenital heart disease. *Hum Pathol*. 1998; 29: 689–95.
3. Yamanaka O, Hobbs RE. Coronary artery anomalies in 126,565 patients undergoing coronary arteriography. *Cathet Cardiovasc Diag*.1990; 21: 28-40.
4. James TN. Anatomy of the coronary arteries in health and disease. *Circulation*.1965; 32: 1020–33.
5. Schlesinger MJ, Zoll PM, Wessler S. The conus artery; a third coronary artery. *Am Heart J*. 1949; 38: 823–36.
6. Williams PL, Warwick R. Dyson M, Bannister L. Gray's Anatomy. 37th Ed. London: Churchill Livingston; 1989, 727-32.
7. Loukas M, Clarke P, Tubbs RS, Kapos T. Raymond de Vieussens. *Anat Sci Int*. 2007; 82: 233-6.
8. Shah P. Heart & great vessels. In: Standring S. Gray's Anatomy: The Anatomical Basis of Clinical Practice. Elsevier Churchill Livingstone; 2004: 39, 1014-7.
9. Estes EH, Entman ML, Dixon HB II, Hackel DB. The vascular supply of the left ventricular wall. Anatomic observations, plus a hypothesis regarding acute events in coronary artery disease. *Am Heart J*. 1966b; 71: 58–67.
10. Levin DC, Baltaxe HA. Angiographic demonstration of important anatomic variations of the posterior descending coronary artery. *Am J Roentgenol Radium Ther Nucl Med*. 1972; 116: 41-9.
11. Hadziselomovic HA. Blood Vessels of the Human Heart. *Thieme Leipzig*.1982; 14–100.
12. Dollar AL, Roberts WC. Retroaortic epicardial course of the left circumflex coronary artery and anteroaortic intramyocardial (ventricular septum) course of the left anterior descending coronary artery: An unusual coronary anomaly and a proposed classification based on the number of coronary ostia in the aorta. *Am J Cardiol*. 1989; 64: 828–9.
13. Gonzalez-Angulo A, Reyes HA, Wallace SA. Anomalies of the origin of coronary arteries. (Special reference to single coronary artery). *Angiology*. 1966; 17: 96–103.
14. Greenberg MA, Fish BG, Spindola-Franco H. Congenital anomalies of the coronary arteries. Classification and significance. *Radiol Clin North Am*. 1989; 27: 1127–46.
15. Barbour DJ & Roberts WC. Origin of the right from the left main coronary artery (single coronary orifice in aorta). *Am J Cardiol*. 1985; 55: 609.
16. Hemery Y, Richard P, Belaouchi F, Heloire F, Monsegu J, Varenne O *et al*. Anomalous origin of coronary arteries from three separate ostia in the right sinus of valsalva: a case report. *Arch Mal Coeur Vaiss*. 2000; 93(12): 1565-69.
17. Samarendra, Kumari P, Hafeez S, Vasavada M, & Sacchi. Anomalous circumflex coronary artery: benign or predisposed to selective atherosclerosis. *Angiology*. 2001; 52(8): 521-26.
18. Fedoruk LM, Kern JA, Peelar BB, Korn IL. Anomolous origin Right Coronary Artery: Right Internal Thoracic Artery to Right Coronary Artery bypass is not the answer. *J Thorac Cardiovas Surg*. 2007; 133: 456-60.
19. Vlodaver Z, Newfeld HN, Edwards JE. Pathology of coronary disease. *Seminars Roentgenol*. 1972; 7: 376-94.
20. Williams PL, Bannister LH, Berry MM, Collins P, Dyson M, Dussek JE, Ferguson MWJ. Grays anatomy in: circulatory system 38th Ed. New York: Churchill Livingstone; 1995, 1505-1510.
21. Kalpana R. A study on principal branches of coronary arteries in Humans. *J Anat Soc India*. 2003; 52(2): 137-40.
22. Uemura, H. Ventricular Morphology and coronary arterial anatomy in hearts with isometric atrial appendages. *American Thoracic Surgery*. 1999; 67(5): 1403-1411.
23. Laurie W, Woods JD. Anastomoses of the coronary circulation. *Lancet*. 1958; 2: 812.
24. Thomas NJ. Anatomy of Coronary arteries. New York: Harper and Rao Publications Inc;

1961.

25. Cavalcanti JS, de Lucena Oliveira M, Pais e Melo AV Jr, Balaban G, de Andrade Oliveira CL, de Lucena Oliveira E. Anatomic variations of the coronary arteries. *Arq Bras Cardiol*. 1995; 65(6): 489-92.
26. Adams J, Treasure T. Variable anatomy of the right coronary artery supply to the left ventricle. *Thorax*. 1985; 40(8): 618-20.

Instructions to Authors

Submission to the journal must comply with the Guidelines for Authors.

Non-compliant submission will be returned to the author for correction.

To access the online submission system and for the most up-to-date version of the Guide for Authors please visit:

http://www.rfppl.org

Technical problems or general questions on publishing with IJA are supported by Red Flower Publication Pvt. Ltd's Author Support team (http://ww.rfppl.org)

Alternatively, please contact the Journal's Editorial Office for further assistance.

A Lal

Publication -in-Charge

Indian Journal of Anatomy

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi – 110 091

India

Phone: 91-11-22754205, 45796900, Fax: 91-11-22754205

E-mail: redflowerppl@gmail.com, redflowerppl@vsnl.net

Website: www.rfppl.org

Review Article

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Morphometric Study of the Pituitary Gland on MRI

Pandhare S.R.*, Gaikwad A.P., Shinde R.B.***, Bahetee B.H.****, Vahane M.*****, Pawar S.******

Abstract

Aims: 1. To study the shape and size of normal pituitary gland with respect to both the lobes in all age groups of both genders by studying MRI images of brain. 2. To determine and evaluate mean normal size and shape of normal pituitary gland with relation to age and gender. **Setting and Design:** For the study MR images all ages were used to characterize the effect of age and sex on pituitary size and shape. All the three dimensions of both the lobes of pituitary gland and stalk were measured using mid sagital and axial MR images. **Methods and Material:** Images of 160 living subjects (76 females and 84 males) of all ages were used. By using electronic caliper all above mentioned parameters were measured. **Statistical Analysis Used:** The data was grouped in age group 0-10, 11-20, 21-50, >51 years of both the genders, analyzed by One way ANOVA test, unpaired t-test. For shape of gland percentage and Chi-square test used. **Results:** The dimensions were steadily increased up to 21-50 years group later decreased. The changes were noted statistically significant. Also significant change in shape. More convex up to 50 yrs then become concave. **Conclusion:** This study demonstrated the database all age groups, which was its mean size and shape of both lobes of pituitary gland, can apply for clinical evaluation.

Keywords: Pituitary gland; MRI, anterior and posterior lobe; Shape.

Introduction

Previous studies describing the growth patterns of midline brain by MR Imaging with the measurement of four midline brain structure, one of that was a pituitary gland.[1] High-resolution computed tomography (CT) with intravenous contrast material is recognized as the method of choice for imaging the pituitary gland. However, there are limitations to the ability of CT to identify micro-adenomas, and magnetic resonance imaging (MRI) is increasingly being recognized as an excellent imaging method for the central nervous system with morphological details of normal pituitary gland.[2] MRI allows multiplanar scan without artifact of bone shadows. In addition, T1-weighted sequences permit evaluation of the hyper intense signal of the posterior pituitary (HSPP), although the origin of this signal remains controversial.[3,4] Various studies has been published about shape and size of pituitary gland of age group from new born child to adult individually or compiled of all age groups.[5,6,7,8,9] All these studies were retrospective and very few studies are available from Indian population. As we have not found a report with regards to separate dimensions specifically of the anterior and posterior lobes in Indians; hence we report this research for the purpose of:

1. To study the shape and size of normal pituitary gland with respect to the both lobes in all age groups of both genders by studying MRI images of brain.
2. To determine and evaluate mean normal size and shape of normal pituitary gland with relation to age and gender.

Author's Affiliation: *Assistant Professor, **Associate Professor, ***Assistant Professor, ****Professor, Dept. of Anatomy, *****Professor, ******Associate Professor, Dept. of Radiology, B.J.G.M.C., Pune, Maharashtra, India.

Reprint's Request: Dr. Pandhare S.R., Assistant Professor, Dept. of Anatomy, B.J.G.M.C., Pune, Maharashtra, India.

E-mail: swatipandhare84@gmail.com

Fig 1: Methods: For Measurements by Using Electronic Caliper: Sagital View

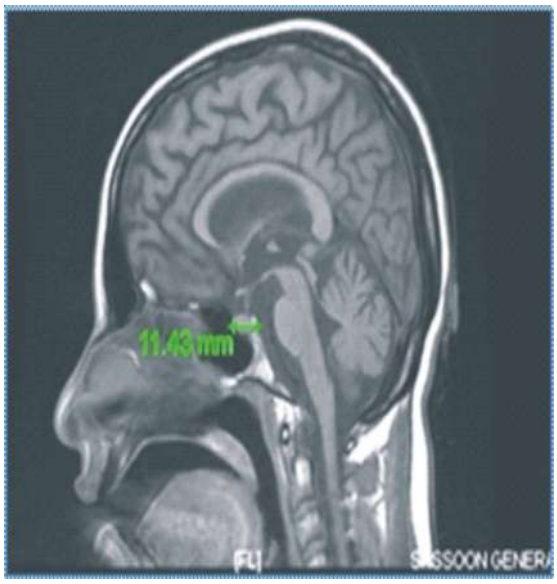
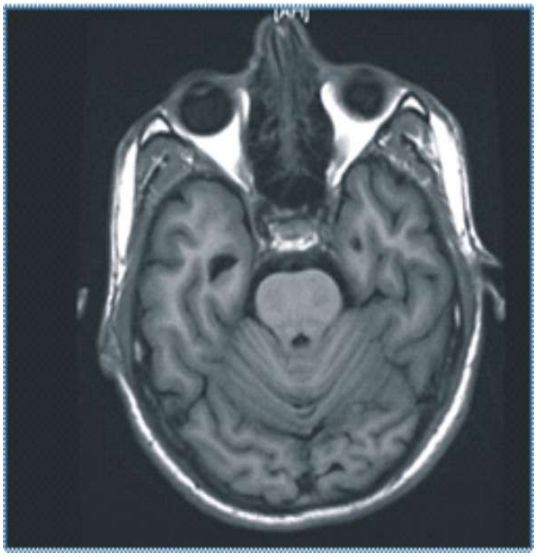


Fig 2: Axial View: for Transverse Dimensions



Material and Methods

The sample size was 160 patients (84 male and 76 female), at age 1year to maximum of 83 years old. All were from Maharashtra from in-patient and outside-patient departments of general hospital of medical college. For the study, routine high field MR images at 1.5 T of brain since January 2013 were studied. Only those showing normal anatomy, with no pathology, with no history related to the pituitary gland or hormonal disorders, surgery or history of treatment by hormone therapy were included in this study. In case of unclear MRI images or those showed pathology of the pituitary and abnormality in sellar and parasellar region were excluded. The subjects were grouped gender wise into eight different groups of four age groups (1-10, 11-20, 21-50, >51 years).

In the sagital and axial views of MR scan, the measurements of the antero-posterior, vertical and transverse dimensions of both lobes of pituitary gland and stalk were taken by using the electronic caliper of the display. (Fig 1 & Fig 2) Also the shape of superior surface of pituitary gland was studied as shown in Fig 3. The T1-weighted sequences

Fig 3: Shape of Superior Surface

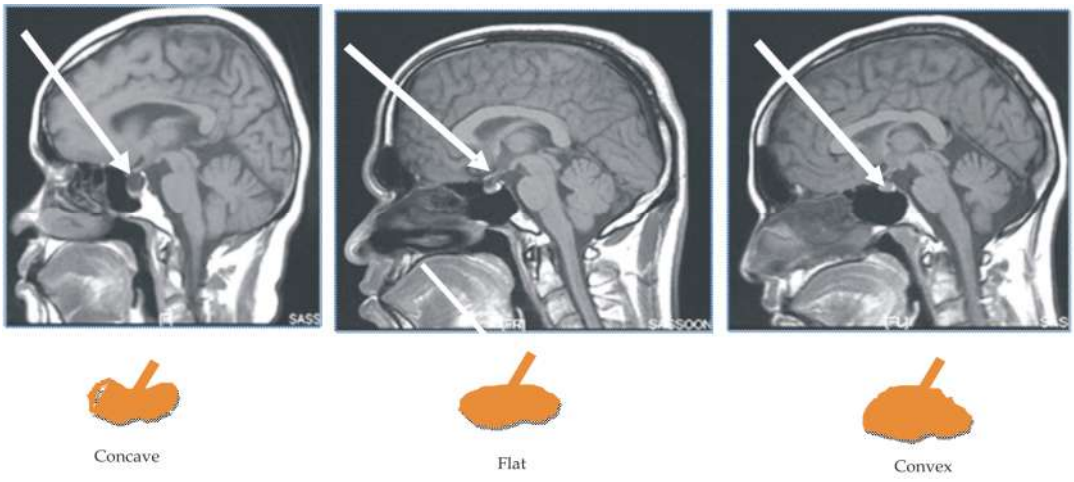
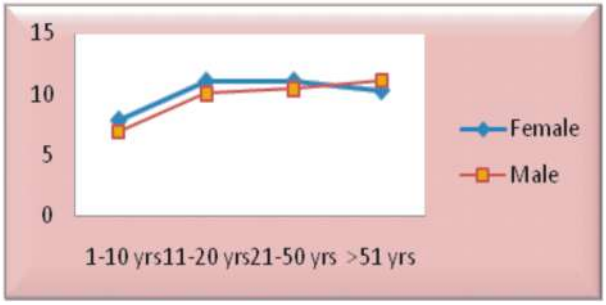


Table 1: Total Antero-Posterior Diameter of Whole Gland

Patient age groups (years)	Mean measurement: mm ±SD	
	Total Antero-posterior diameter of whole gland	
	Female	Male
1-10 n= 18	7.88± 1.06	6.97±0.67
11-20 n= 20	11.05 ± 1.55	10.04 ±1.60
21-50 n= 68	11.07 ±1.10	10.41 ± 2.30
>51 n= 54	10.33 ± 1.4	11.08 ± 3.05
p Value * ANOVA	0.0186	0.0190

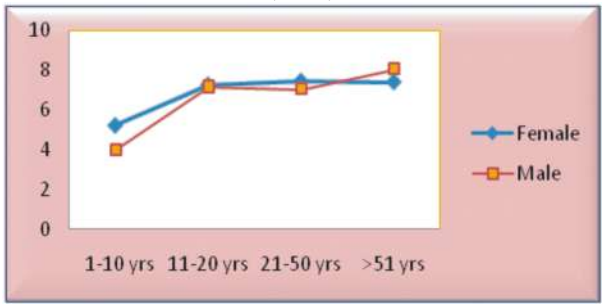
Graph 1: Total Antero-Posterior Diameter (mm) of Whole Gland



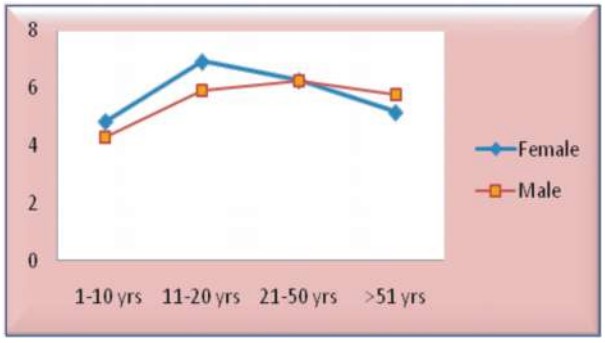
permits evaluation of the hyper intense signal of the posterior pituitary (HSPP) that was adventitious for measuring the dimensions of posterior lobe. For visualization of the stalk of the pituitary gland, Sylvia's aqueduct and posterior gland bright spot were considered to be the criterion of true midline views, in sagital view but in axial posterior gland bright spot could not be seen. The dimensions were measured.

The evaluations were carried out by the authors independently, with subsequent discussion, as necessary, to agree on the hyper intense signal of the posterior pituitary to minimize observation variation. The measurements were analyzed in 8 different age groups of both genders separately for mean ± SD and One way ANOVA test. Further data

Graph 2.1: Anterior Lobe Mean Measurement: Antero-Posterior Diameter (mm)



Graph 2.2: Anterior Lobe Mean Measurement: Height (mm)



was subjected by unpaired t-test using open EPI software for analyzing the sexual dimorphism. The shape of gland evaluated to percentage and Chi-square test. P-value less than 0.05 considered as significant.

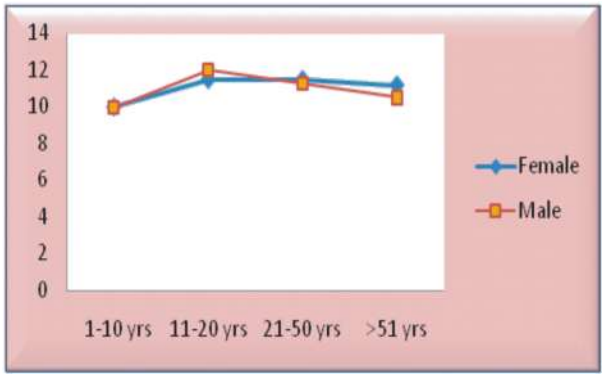
Results

A total of 200 cases were selected for study, out of that 40 were excluded according to the selection criteria. In this 76 were female and 84 were male. The data was arranged into eight groups on the basis of age and gender (1-10, 11-20, 21-50, >51 years).

Table 2: Anterior Lobe Mean Measurement

Patient's age groups (years)	Anterior lobe Mean measurement: mm ±SD					
	Antero-posterior diameter		Height		Transverse	
	Female	Male	Female	Male	Female	Male
1-10 n= 18	5.25±1.00	4.00±0.32	4.83±0.91	4.30±1.15	9.97±3.24	9.95±2.16
11-20 n= 20	7.30 ± 1.18	7.15±1.50	6.93±1.47	5.92±1.27	11.46±3.64	12.02±2.92
21-50 n= 68	7.49±1.17	7.07±1.66	6.27±1.37	6.24±1.26	11.46±0.78	11.26±2.58
>51 n= 54	7.45±1.45	8.05±2.13	5.15±0.62	5.78±1.24	11.17±1.98	10.49±2.96
p Value * ANOVA	0.0159	0.0140	0.0088	0.0117	0.0310	0.0228

Graph 2.3: Anterior Lobe Mean Measurement: Transverse Diameter (mm)



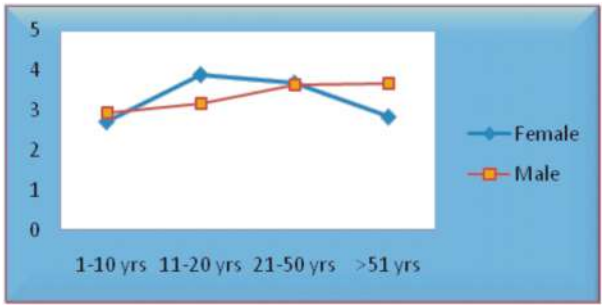
Size

As seen in Table 1 and corresponding graph 1, the mean value of total antero-posterior diameter of whole gland in female (1-10 yrs, n = 10) was 7.877mm ±1.06 (SD) and that of male (n = 8) was 6.973 mm ± 0.6683 (SD). Group 2, (11-20 yrs) was 11.05 ± 1.54mm in female (n=8) and 10.04 ±1.59 mm in males (n=12). In group 3 (21-51yrs) was 11.07 ±1.102 mm in females (n=33) and 10.41± 2.29 mm in males (n=35). While in group 4 (>51 yrs) that was 10.33 ± 1.396 mm in females (n=25) and 11.08 ± 3.051 mm in males (n=29). These were significantly different (p< 0.01) among the both male and female groups, as shown in Table 1.

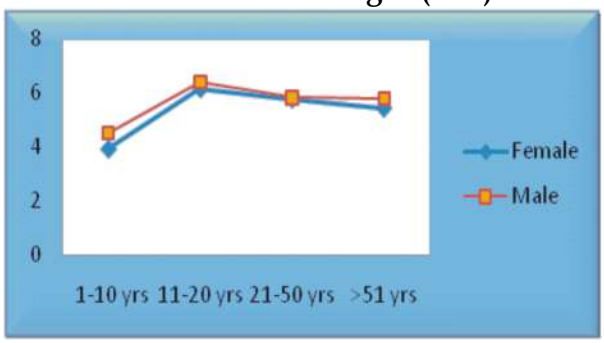
Similarly, the mean and standard deviation of different dimensions of anterior lobe, posterior lobe and that of stalk were given in Table 2, 3, 4 and their corresponding graphs, respectively. The means of different dimensions of both the genders among the all age groups were significantly related with each other as tested by One way ANOVA (p<0.01- p <0.03).

Further, the sexual dimorphism was analyzed among the all age groups by using unpaired t-test. In all age groups, majority of male mean dimensions were nearly similar

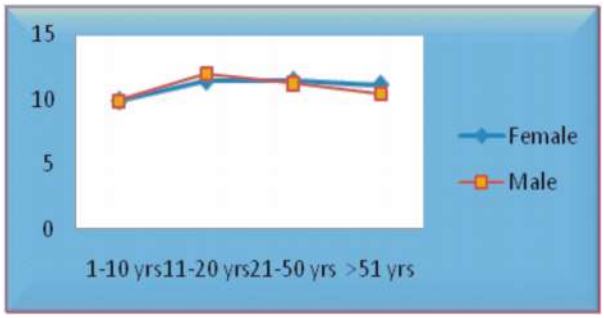
Graph 3.1: Posterior Lobe Mean Measurement: Antero-Posterior Diameter (mm)



Graph 3.2: Posterior Lobe Mean Measurement: Height (mm)



Graph 3.3: Posterior Lobe Mean Measurement: Transverse Diameter (mm)



with that of females except the few like antero-posterior diameter of anterior lobe, at age group I (1-10yrs), (p=0.004) antero-posterior diameter of posterior lobe, at age group IV (>51

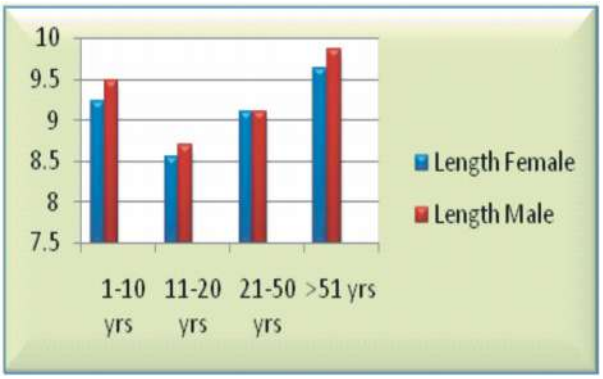
Table 3: Posterior Lobe: Mean Measurement

Patient age groups (years)	Posterior lobe Mean measurement: mm ±SD					
	Antero-posterior diameter		Height		Transverse	
	Female	Male	Female	Male	Female	Male
1-10 n= 18	2.72±0.46	2.94±0.54	3.96±1.15	4.55±1.17	6.39±1.07	6.81±1.86
11-20 n= 20	3.89±0.61	3.16±0.85	6.17±0.8	6.40±2.04	9.17±4.35	10.34±4.16
21-50 n= 68	3.68±0.93	3.63±1.01	5.79±1.43	5.84±1.52	9.60±4.42	9.58±4.14
>51 n= 54	2.84±0.39	3.67±0.84	5.46±1.98	5.79±1.79	7.89 ±1.78	9.58±4.25
p Value * ANOVA	0.0095	0.0084	0.0144	0.0129	0.029	0.0280

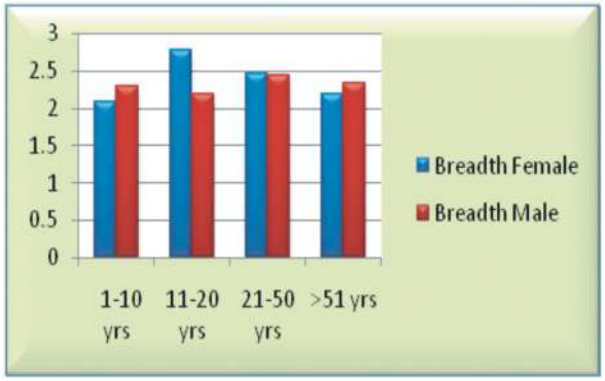
Table 4: Stalk Mean Measurement: Length and Breadth.

Patient age groups (years)	Stalk Mean measurement: mm ±SD			
	Length		Breadth	
	Female	Male	Female	Male
1-10 n= 18	9.227±2.07	9.49±1.55	2.08±3.95	2.29±0.50
11-20 n= 20	8.544±2.01	8.69±1.68	2.77±0.46	2.20±0.43
21-50 n= 68	9.10±1.92	9.10±1.51	2.47±0.51	2.44±1.06
>51 n= 54	9.63±2.03	9.86±1.82	2.19±0.25	2.33±0.46
p Value * ANOVA	0.0218	0.0165	0.0083	0.0071

Graph 4.1: Stalk Mean Measurement: Length (mm)



Graph 4.2: Stalk Mean Measurement: Breadth (mm)



yrs) (p=0.0033) and breadth of stalk, at age group II (11-20 yrs) (p=0.011), showed no statistically significant difference. By considering these results, there was no sexual dimorphism in different dimensions of the both lobes of Pituitary.

Shape

The shape of the superior surface of the gland (SS) was observed in all 160 patients. The total frequency of convexity seen for 70% females and 50% males, and it was found to be significant. (F, p=0.03; M, p=0.029) (Table 5). This convex upper border was more common in group III (21-50 yrs) of both males and females with frequency (28%) and (35%) respectively. In males, the frequency of flat upper surface was seen in 4% cases while only 3% female patients showed the flat surface, which was not significant in both (p> 0.5). And remaining all cases were having concave upper surface (27% in females and 46% in males). There was no significant gender difference in convex shape of the upper border (p=0.3) and hence no sexual dimorphism seen.

Discussion

Different textbooks of anatomy mentioned the measurements of Pituitary gland. According to Gartnermasurements were 10×6×5 mm in new borns; 15×10×6 mm at 20 years; 16×13×7 mm at 40 yrs, 17×12×6 mm at

Table 5: Shape of Superior Surface

Total Frequency of	Female	Male
Convex surface	70%	50%
Concave surface	27%	46%
Flat surface	3%	4%
P value by Chie- square test among convex and non convex frequency	0.03	0.029

70 yrs and 13×11×6 mm at 80 years; this showed a decrease in measurements as a whole after the age 40 yrs.[10] According to Harrisonthe diameters were; 14 mm-transverse, 9 mm-anteroposterior and 6 mm-vertical.[11] Williams *et al* reported the measurements of the gland as 12 mm transverse and 8mm anteroposteriorly.[12] Using MRI Gonzalez *et al* found the average measurements, in 20 normal Mexican living women to be 12.4 mm (transverse) 7.9 mm (anteroposterior) and 6 mm (vertical).[13] Also a useful guide to the gland's height in relation to age is "Elster's rule"of 6,8,10,12: 6 mm for infants and children, 8 mm in men and postmenopausal women, 10 mm in women of childbearing age and 12 mm for women in late pregnancy or postpartum women. Andthe pituitary stalk has a normal thickness of 2 mm, and it should not exceed a maximum of 4 mm or the width of the basilar artery.[14]

In our study also, the measurements were 9.97; 9.95 (F;M) – transverse, 7.88; 6.97 (F;M) – antero-posterior, 4.83; 4.30 (F;M) – height at 1-10 yrs, 11.46; 11.26 (F;M) – Transverse, 11.07; 10.41(F;M) – anteroposterior and 6.27; 6.24 (F;M) – vertical i.e height at 21-50 yrs, while at >51 yrs measurements were 11.17; 10.49 (F;M) – transverse, 10.33; 11.8 (F;M) – anteroposterior and 5.15; 5.78 (F; M) – height. This also showed a decrease in measurements as a whole after 50 years as like previous authors.[8,9]

Sexual Diamorphism

Lurie *et al* measured length, breadth and height of the pituitary gland in 35 (16 male and 19 females) adult volunteers between the age 26-79 yrs by MRI in sagittal and coronal sections of the head.[15] They observed that, statistically there was no sexual difference in the size of pituitary.

Similarly, in our study except for few parameters, remaining all suggested that there was no statistically significant sexual dimorphism in the size of pituitary gland. The parameters like antero-posterior diameter of anterior lobe, at 1-10 yrs and that of posterior lobe, at >51yrs and also the breadth of stalk, at 11-20 yrs showed statistically significant difference among the both sexes.

Along with these dynamic changes in size of pituitary, this study also showed the range of appearances i.e. the shape of superior surface of the normal whole Pituitary gland during different stages of life. Pronouncely, at age of 21-50 yrs, the superior surface showed convexity in 35% females & 28% of males out of total frequency of convex surface which was in 70% females and 50% males.

These dynamic changes occur in the size, shape Pituitary gland during life were similar with the previous authors.[5,8,9,10] It was suggested that, there was a complex hormonal environment of the pituitary gland reflect into the variability in size and shape of gland at different stages of life and they were most pronounced at times of hormonal flux such as menarche[16,17] and pregnancy.[18]

Observations of the pituitary gland during puberty have shown a definite but transient increase in size, attributed to the increased hormonal activity at this time.[16,17] The hormonal levels of pituitary at puberty, even though higher than adult levels, but are much lower in the neonatal period. This may account for the change in size of the gland.

It has been documented that the pituitary gland also enlarges and may increase in signal intensity during pregnancy.[18] The changes in appearance of the gland during different phases of the menstrual cycle have been also shown (R. Shankwiler, M. E. Mawad, C. Valdes,K. Elkind-Hirsch, "Biphasic Morphological Changes of the Normal Menstrual Cycle demonstrated on Gadolinium MRI," presented at the29th Annual Meeting of the American Society of Neuroradiology, Washington, DC, June 13,1991).[8] The children with precocious puberty showed changes in the gland paralleling to those seen in healthy adolescents irrespective of their chronologic age.[19] It therefore appears that, there is a strong link between the fluctuation in hormone levels and the changing appearances of the pituitary gland during life.

Hence we conclude that, the changes in shape and size of Pituitary gland observed in our study also reflect the same reason of hormonal level fluctuation.

The Age related changes in the shape of upper border have been documented.[20,21] The frequency of convex upper border of the pituitary gland is reported to be higher in young age group. It becomes very important to differentiate the normal convex upper border of the pituitary from an abnormal convex upper border due to microadenoma, lymphocytic adenohypophysitis and pituitary cyst etc.[8] For this reason, observations of chronological changes in the shape of the pituitary upper border in young age group, was done in this study which were similar to that of the previous authors.

The studies of Pituitary gland in childhood also have clinical application in the evaluation of children with possible pediatric endocrine abnormalities, who may demonstrate an abnormal appearance of the gland for their age. The demonstration of a small, low-signal-intensity gland in an infant younger than 6 weeks of age may raise the possibility of panhypopituitism. By contrast, in an older child with precocious puberty, an enlarged gland may be seen at a time when the gland should have a flat or concave appearance.[20]

Conclusion

This study demonstrates the database of Indian people in age groups from children to puberty to young adults to old age whose mean size and shape of pituitary gland from each age groups, was acquired by MR Imaging, can apply to clinical evaluation particularly of clinical symptoms of patients in pubertal period and young adults in which physiologic pituitary hyperplasia can mimic pituitary tumor.

Acknowledgement

My sincere thanks to head of department, senior teachers, colleagues of Department of Anatomy, and Department of Radiology Govt. Medical College and Hospital for their valuable co-operation and support. Also my sincere thanks to; the Statistician, Dept PSM, Govt. Medical College.

Key Message

The database of Indian people, with respect to morphometry of both lobes of pituitary gland.

References

1. Hayakawa K, Konishi Y, Matsuda T, Kuriyama M, Konishi K, Yamashita K. Development and Aging of Brain Midline structure: Assessment with MR Imaging. *Radiology.* 1989; 172: 171-7.
2. Davis PC, Hoffman JC, Tindall GT, Braun IF. Prolactin secreting pituitary microadenomas: inaccuracy of high resolution cr. *AJNR.* 1984; 5: 721-726.
3. Fujisawa I, Nishimura K, Asato R, *et al*. Posterior lobe of the pituitary in diabetes insipidus: MR findings. *J Comput Assist Tomogr*. 1987; 11: 221–225.
4. Kucharczyk J, Kucharczyk W, Berry I, *et al*. Histochemical characterization and functional significance of the hyperintense signal on MR images of the posterior pituitary. *AJR Am J Roentgenol*. 1989; 152: 153–157.
5. Sahni D, Jit I, Harjeet, Neelam, Bhansali A. Weight and dimensions of the pituitary in northwestern Indians. *Pituitary*. 2006; 9(1): 19-26.
6. Kato K, Saeki N, Yamaura A. Morphological changes on MR imaging of the normal pituitary gland related to age and sex: main emphasis on pubescent females. *J Clin Neurosci*. 2002; 9(1): 53-6.
7. Rosalind B Dietrich, Leon E Lis, Fred S Greensite, and Duane Pitt. Normal MR Appearance of the Pituitary Gland in the First 2 Years of Life. *AJNR*. 1995; 16: 1413–1419.
8. Muhammad Faisal Ikram, Zafar Sajjad, Ishrat Shokh, Amir Omair. Pituitary Height on Magnetic Resonance Imaging Observation of Age and Sex Related Changes. *J Pak Med Assoc*. 2008; 58(5): 261-265.

9. C Keanninsiri1, P Cheiwvit, S Tritrakarn1, K Thepamongkhol and J Santiprabhop. Size and Shape of the Pituitary Gland with MR Imaging from Newborn to 30 Years: A Study at Siriraj Hospital Available from: http://www.tmps.or.th/meeting2012/FullPaper/Chonticha.pdf
10. Gartner WU. The endocrine glands and unclassified organs In: Anson BJ. (ed), Morris' Human Anatomy 12th edn. London: McGraw Hill Book Company; 1966, 1540-42.
11. Harrison RG. The ductless glands. In: Romanes GJ. (ed.) Cunninghams's Text book of Anatomy, 12th edn. Oxford: Oxford University Press; 1995, 602-607.
12. Williams PL, Bannister LH, Berry MM, Collins P, Dyson M, Dussek JE, Ferguson MWJ (eds) Gray's Anatomy, 38th ed. Edinburgh: Churchill Livingston; 1995, 1983.
13. Gonzalez JG, Elizondo G, Saldivar D, Nanez H, Todd LE, Villarreal JZ. Pituitary Gland growth during normal pregnancy: An in vivo study using magnetic resonance imaging: the effect of age. *Am J Med*. 1988; 85: 217-220.
14. Vikas Chaudhary and Shahina Bano. Imaging of the pituitary: Recent advances. *Indian J Endocrinol Metab*. 2011; 15(3): 216–223.
15. Luriec, Doraiswamy PM, Husain MM, Boyko OB, Ellinwood EH, Figiel GS, Krishnan KRR. In vivo Assessment of Pituitary gland volume with magnetic resonance imaging: the effect of age. *J Clin Endocrinol Metabo.* 1990; 505-508.
16. Elster AD, Chen MYM, Williams DW, Key LL. Pituitary gland: MR imaging of physiologic hypertrophy in adolescence. *Radiology*. 1990; 174: 681–685.
17. Peyster RG, Hoover ED, Adler LP. CT of the normal pituitary stalk. *AJNR Am J Neuroradiol*. 1984; 5: 45–47.
18. Miki Y, Asato R, Okumura R, *et al*. Anterior pituitary gland in pregnancy: hyperintensity at MR. *Radiology*. 1993; 187: 229–231.
19. Kao SCS, Cook JS, Hansen JR, *et al*. MR imaging of the pituitary gland in central precocious puberty. *Pediatr Radiol*. 1992; 22: 481
20. Dietrich RB, Lis LE, Greensite FS, Pitt D. Normal MR appearance of the pituitary gland in the first 2 years of life. *AJNR Am J Neuroradiol*. 1995; 16: 1413-9.
21. Tien R D, Kucharczyk J, Bessette J, Middleton M. MR imaging of the pituitary gland in infants and children: changes in size, shape, and MR signal with growth and development. *AJR Am J Roentgenol*. 1992; 158: 1151-4.

Case Report

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Possible Developmental Origin and Clinical Implications of a Brachial Plexus Variation

Mona Sharma* Rani Kumar, Renu Dhingra*****

Abstract

The highly organized network of human nervous system is well appropriate for the perfect coordination and functioning of the body. Thus, a well studied and thoroughly researched knowledge regarding the anatomy of nerves is highly vital. The brachial plexus formation has been associated with lots of anatomical variations. The present case report describes another variation where musculocutaneous nerve after giving its motor component in the arm is not continuous with its sensory part in the forearm. The motor component of musculocutaneous nerve ends by supplying coracobrachialis muscle whereas the other forearm muscles are innervated directly from the lateral cord and one of these branches is continuous as the sensory component of the normal musculocutaneous nerve named as lateral cutaneous nerve of forearm. In our case report, we also aim to explain the possible developmental mechanisms behind the nerve variations along with their clinical implications.

Keywords: Brachial plexus; Musculocutaneous nerve; Lateral cutaneous nerve of forearm; Median nerve.

Introduction

The brachial plexus is the network of nerves supplying brachium, formed by the ventral rami of C5-T1 roots of spinal nerves. The roots join and form trunks (upper trunk C5,6; middle trunk C7; lower trunk C8,T1), each of which further separate into anterior and posterior divisions. The divisions integrate and organize into three cords- lateral, medial and posterior. The terminal branches of cords supply arm and forearm muscles. It is the lateral cord which gives rise to musculocutaneous nerve opposite the lower border of pectoralis minor which after supplying coracobrachialis muscle, courses through this muscle and runs laterally between biceps and brachialis muscle while innervating two of these. On further course, it comes out lateral to elbow joint and continue down as its sensory component- the lateral cutaneous nerve of forearm.[1] The hand to hand knowledge regarding brachial plexus variations is greatly useful for the surgeons to assess the functional loss due to trauma, to plan constructive surgeries, during reduction of fractures and dislocations, repair of wounds and nerve entrapement syndromes.

Case Report

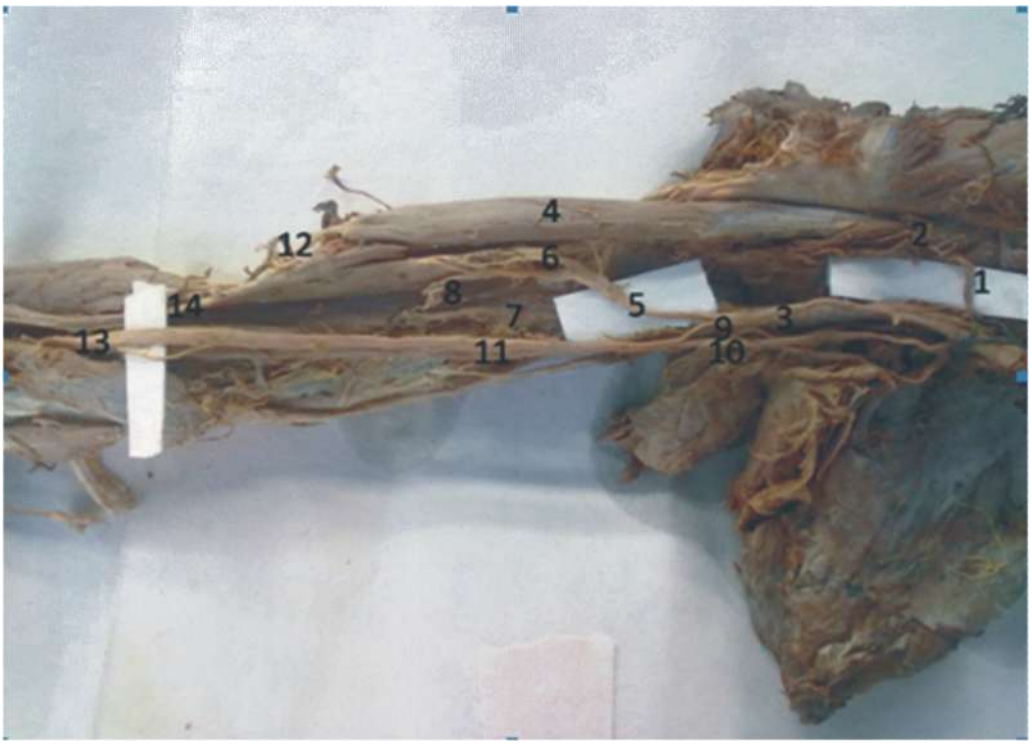
A unilateral variation in brachial plexus was noticed during routine educational dissection of right arm of 55 yr old male cadaver in the department of anatomy, All India Institute of Medical Sciences, New Delhi, India. The brachial plexus dissection was started by exposing first the coracobrachialis and short head of biceps muscles arising from the tip of coracoid process. A branch from the lateral cord was noticed distributing to the deep surface of coracobrachialis muscle which was

Author's Affiliation: *Professor & Head, Dept of Paediatrics, **Assistant Professor, ***Residents, Padmashree Dr. Vithalrao Vikhe Patil Medical College & Hospital, Near Govt. Milk Dairy, Vilad Ghat, Ahmednagar – 414111, Maharashtra, India.

Reprint's Request: Dr. Sunil Mhaske, Professor & Head Dept of Paediatrics; Padmashree Dr. Vithalrao Vikhe Patil Medical College & Hospital, Near Govt. Milk Dairy, Vilad Ghat, Ahmednagar – 414111, Maharashtra, India.

E-mail: sunilmhaske@rocketmail.com

Fig 1: 1. Nerve to coracobrachialis; 2.Coracobrachialis; 3.Lateral cord; 4.Biceps; 5.Branch from lateral cord; 6. Branch to biceps; 7.Brachialis; 8.Nerve to brachialis; 9.Lateral root of median nerve; 10.Medial root of median nerve; 11.Median nerve; 12. Lateral cutaneous nerve of forearm; 13. Median nerve in cubital fossa; 14.Tendon of biceps



initially thought to be the musculocutaneous nerve. On further dissection, it was found to end in the muscle itself. On searching the other branches of lateral cord, we found one branch given distally soon dividing into multiple small twigs supplying biceps and brachialis muscle. The branch given to the biceps passed further distally and came out laterally through it in a more superficial plane above elbow joint which continuous down as lateral cutaneous nerve of forearm. At the same level from the lateral cord, fibers of lateral root of median nerve joined with the medial root from medial cord to form median nerve. The course of median nerve was found normal. There were no other variations seen in medial or posterior cord branches. (Fig 1)

Discussion

Variations in Literature

The complexity of brachial plexus anatomy makes it vulnerable to present with various alterations in its formation and branches. The variations have been mentioned in literature since 19th century. The nerve distribution and variations in the upper limb have been given in detail in the past.[2] Le Minor classified these variations in five types.[3]

Type 1: No communication between Median and Musculocutaneous nerve

Type 2: Medial root of median nerve passes through musculocutaneous nerve and later join the median nerve

Type 3: Lateral root of median nerve passes through musculocutaneous nerve and later form the lateral root of median nerve

Type 4: The musculocutaneous nerve join the lateral root of median nerve and later musculocutaneous nerve arise from median nerve

Type 5: The musculocutaneous nerve is absent, all fibers pass through lateral root of median nerve and fibers to the muscles

supplied by musculocutaneous branch out directly from median nerve.

Our case report presents a different variation not coinciding with any of the types stated above. Musculocutaneous nerve not piercing coracobrachialis and communicating with median nerve was also noticed.[4] Median nerve with three roots and its communication with musculocutaneous nerve has also been mentioned.[5] Absence of musculocutaneous nerve, median nerve with three roots, accessory head of biceps was found.[6] A case of four headed biceps brachii, three headed coracobrachialis muscle with communicating branch between median and musculocutaneous nerve has also been reported.[7] The brachial plexus consisting of a single common cord with absence of musculocutaneous nerve have also been mentioned.[8]

Developmental Origin

The understanding of these nerve variations is highly complex as it starts during embryonic development. The mesenchymal cells of lateral plate mesoderm initiates the limb development. The patterning in this process is guided through homeobox genes HOX D1-D5.[9] The limb buds are formed deep to a thick band of ectoderm (apical ectoderm ridge). Therefore, each limb bud consists of a mass of mesenchyme derived from the somatic layer of mesoderm covered by ectoderm. Simultaneous to this phenomenon of limb development, nerve axons from the spinal cord segment also assemble opposite to limb buds and start growing into the limb. The axons are distributed to the muscles getting differentiated from the myogenic cells originating from the somites. The ventral primary rami of these nerves are joined and form plexuses that grow into the developing limb. The growth of motor axons into the limb buds occur during 5th wk and later continued with entry of sensory axons. Once the axonal transport occurs, neural crest cells, the precursor of schwann cells, surround the motor and sensory nerve fibers and form myelin sheaths.[10] The brachial plexus cone after entering into the upper limb bud divide into dorsal and ventral segments.[11] The ventral segment give rise to median and ulnar nerve. The musculocutaneous nerve further arises from median nerve and so the lateral cutaneous nerve of forearm also.[12] During the axonal course from the spinal cord to the developing limb bud, the failure of differentiation and coordination between nerve fibers and axonal growth cone migration leads to communications and hence, variations. A nerve growth cone is the region of tip of the axon which leads ahead during nerve outgrowth and is considered to be the locomotor organelle of the neuron. During migration of this growth cone, the complete neuronal pool gets arranged in to three longitudinal columns.[13] Column of Terni projects ventrally into the sympathetic ganglion. Lateral motor column extends into the developing limbs and medial motor column projects into the axial muscles. Each group of neurons is specified by a particular group of Lim proteins. These gets induced during neuronal migration and guide the neurons towards their target cells.[14] This guiding process occur in three steps of selection process.[15] Firstly, axons travel along a path to a particular region in embryo (pathway selection). Once the axons reach a particular region, they recognize and bind to the specific cells by making connections (target selection). Each axon binds to a subset of target cells due to interneuronal activity (address selection). This process further refine the overlapping projections into a fine pattern of connections. The pattern of axon transfer is decided by various factors guiding the growth cone of different neurons in different directions so that even the adjacent neurons are given different instructions. The guiding factors can be the matrix substrates or diffusible molecules. Extracellular matrix substrates facilitate the axons to travel on these substrate which can be either chemotactic like laminins.[16] causing adherence and axonal migration by a process known as "haptotaxis" or the chemorepulsive like ephrins, semaphorins leading to retraction of growth cone.[17] The diffusible molecules can also be chemotactic like netrin 1,2[18] or

chemorepulsive like slit proteins.[19] Once axons reaches the target cells, its response is guided by various chemical substances termed as neurotrophins produced by target cells (nerve growth factor, brain derived growth factor, neurotropic factors 3,4,5).[20] Throughout the course of axonal migration, the growth cone sense a wide range of chemotactic and chemorepulsive substances. The growth cone integrates simultaneously with these molecules and the response is based on the combined input of these molecules. Any alteration between the mesenchyme target cells and these signaling molecules can lead to variation in nerve patterns.[21]

Clinical Implications

The knowledge regarding the anatomical variations is essential for perfect interpretation of clinical neurophysiology as these can cause unusual clinical signs. Musculocutaneous nerve is involved in traumatic nerve compressions during weight lifting, neuralgic amyotropy, during anterior dislocation of shoulder joint. It leads to flexor paralysis and anaethesia in nerve distribution. According to our case report, nerve damage at axilla may leads to paralysis of coracobrachialis muscle, the other stronger flexor muscles like biceps and brachialis and sensory function of lateral cutaneous nerve of forearm are spared. Therefore, the present case report should be considered during assessment of nerve entrapement injuries. This becomes also important while doing surgeries like constructive arthroplasty on shoulder and nerve grafting using musculocutaneous nerve.[22] The more superficial lateral cutaneous nerve of forearm can get injured in elbow injuries and compression by anomalous head of biceps.[23] Cutaneous nerves running in more superficial plane in the region of elbow increases the probability of a needle injury during venipuncture. This leads to pain and internal bleeding. This condition is often referred as complex regional pain syndrome. Therefore, location of superficial vein should be considered along with location of superficial nerves by selecting safer site for venipuncture.[24] During surgeries on forearm, wrist and hand, the axillary approach is most commonly used for producing brachial plexus block. The radial side of forearm is anesthesised in addition by targeting musculocutaneous nerve separately in the belly of coracobrachialis just superior to pulsation of axillary artery at the lateral border of pectoralis major muscle.[25] The present case definitely alter the result of anesthesia procedure as the nerve to coracobrachialis is localised at the muscle, therefore, to bring about anesthesia in its distribution, lateral cutaneous nerve of forearm should be blocked separately between brachioradialis and lateral side of biceps tendon. This variation can increase chances of nerve block failure in surgical procedures.

It is beyond doubt that the present case report along with the previously reported ones should be thoroughly remembered by the neurophysicians and surgeons to avoid confusions in clinical settings. More important step to be taken by the researchers is to find out the factors causing imbalance between chemical molecules leading to nerve variations as well as to search for the ways by which the ongoing or soon occurring imbalances can be estimated during intrauterine life. This is to ensure that preventive and therapeutic measures can be taken during fetal growth and development.

References

1. Standring S, Borley NR, Collins P, Crossman AR, Gatzoulis MA, Healy JC. Gray's Anatomy, Pectoral region, shoulder region and axilla, 40th ed. London: Churchill Livingstone; 2008, 791-822.
2. Linell EA. The distribution of nerves in the upper limb, with reference to variabilities and their clinical significance. *J Anat*. 1921; 55: 79-112.
3. Le Minor JM. A rare variation of the median and musculocutaneous nerves in man. *Arch Anat Histol Embryol*. 1990; 73: 33-42.
4. Sud M, Sharma A. Absence of

musculocutaneous nerve and the innervations of coracobrachialis, biceps brachi, brachialis from median nerve. *J Anat*. 2000; 49(2): 176-177.

5. Chauhan R, Roy TS. Communication between the Median and musculocutaneous nerve: A case report. *JASI*. 2002; 51: 1-6.
6. Arora L, Dhingra R. Absence of musculocutaneous nerve and accessory head of biceps brachii: a case report. *Indian Journal of Plastic Surgery*. 2005; 38(2): 144-146.
7. Mehmet MC, Umut O, Yasemin K, Arzu H, Fatos BY, Levent S. Four headed biceps brachii, three headed coracobrachialis muscle associated with arterial and nervous anomalies in the upper limb. *Anat Cell Boil*. 2012; 45: 136-139.
8. Jamuna M. A single common cord in the infraclavicular part of brachial plexus. *Int J Health Sci Res*. 2012; 2: 108-111.
9. Morgan BA, Tabin C. Hox genes and growth: early and late roles in limb bud morphogenesis. *Dev Suppl*. 1994; 181-186.
10. Moore KL, Persaud TVN. The Developing Human. Cutaneous innervation of limbs, 8th ed. Philadelphia: Saunders Elsevier; 2008, 368-371.
11. Iwata H. Studies on development of brachial plexus in Japanese embryo. Republic department anatomy mie perfect university school of medicine. 1960; 13: 129-144.
12. Horiguchi M. The recurrent branch of lateral cutaneous nerve of forearm. *J Anat*. 1981; 132: 243-247.
13. Hollyday. Organization of motor pools in the chick lumbar lateral motor column. *J Comp Neurol*. 1980; 194: 143–170.
14. Tsushida T, Ensini M, Morton SB, Baldassare M, Edulund T. Topographic organization of embryonic motor neurons defined by expression of LIM homeobox genes. *Cell*. 1994; 79: 957–970.
15. Goodman CS, Shatz CJ. Developmental mechanisms that generate precise patterns of Mona Sharma *et al* / Possible Developmental Origin and Clinical Implications of a Brachial Plexus Variation neuronal connectivity. *Cell*. 1993; 72: 77–98.
16. Letourneau P, Madsen AM, Palm SM, Furcht LT. Immunoreactivity for laminin in the developing ventral longitudinal pathway of the brain. *Dev Bio.* 1988; 125: 135–144.
17. Wang HU, Anderson DJ. Eph family transmembrane ligands can mediate repulsive guidance of trunk neural crest migration and motor axon outgrowth. *Neuron*. 1997; 18: 383–396.
18. Kennedy TE, Serfini T, Dela Torre JR, Tessier-Lavigne M. Netrins are diffusible chemotropic factors for commissural axons in the embryonic spinal cord. *Cell*. 1994; 78: 425–435.
19. Brose K. Slit proteins bind robo receptors and have an evolutionary conserved role in axon guidance. *Cell*. 1999; 96: 795–806.
20. Paves H, Saarma M. Neurotrophins as *in vitro* growth cone guidance molecules for embryonic sensory neurons. *Cell Tissue Res*. 1997; 290: 285–297.
21. Sannes HD, Reh TA, Harris WA. Development of the nervous system In: Axon growth and guidance. New York: Academic Press; 2000, 189-197 .
22. Wen-Chieh L, Jeng-Rung C, Yueh JW, Guo FT. The efficacy of end to end and end to side nerve repair (neurorrhaphy) in the rat brachial plexus. *J Anat*. 2009; 215: 506–521.
23. Gilingham BL, Mark GR. Compression of lateral antebrachial cutaneous nerve by biceps tendon. *J Shoulder Elbow Surg*. 1998; 5: 330-332.
24. Yamada K, Yamada K, Katsuda I, Hida T. Cubital fossa venipuncture sites based on anatomical variations and relationships of cutaneous veins and nerves. *Clinical Anat*. 2008; 21: 307-313.
25. Brown DL. Atlas of regional anesthesia 2nd Ed. Philadelphia: 1999.

Case Report

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Pentalogy of Fallot

Sunil Mhaske*, Ramesh B. Kothari, Gaurav Machale**, Sandeep Deokate***, Pavan Suryawanshi***, Nishad Patil***, Rahul Maski*****

Abstract

The pentalogy of Fallot is a variant of the more common tetralogy of Fallot, comprising the classical four features with the addition of an atrial septal defect or patent ductus arteriosus.

The five features therefore are:

1. Ventricular septal defect (VSD)
2. Right ventricular outflow tract narrowing or complete obstruction
3. Right ventricular hypertrophy
4. Over riding aorta
5. Atrial septal defect (ASD).

Keywords: Pentalogy of Fallot; VSD; Right ventricular outflow tract narrowing; Right ventricular hypertrophy; Over riding aorta; ASD.

Introduction

Tetralogy of Fallot, also known as Fallot's syndrome or Fallots tetrad, has four key features. A ventricular septal defect (a hole between the ventricles) and many levels of obstruction from the right ventricle to the lungs (pulmonary stenosis) are the most important. Also, the aorta (major artery from the heart to the body) lies directly over the ventricular septal defect, and the right ventricle develops thickened muscle. Because the aorta overrides the ventricular defect and there is pulmonary stenosis, blood from both ventricles (oxygen-rich and oxygen-poor) is pumped into the body. Sometimes the pulmonary valve is completely obstructed (pulmonary atresia). Infants and young children with unrepaired tetralogy of Fallot are often blue (cyanotic) as in the present case. The reason is that some oxygen-poor blood is pumped to the body. When the above condition is associated with atrial septal defect, it is called Pentalogy of Fallot.

Case Summary

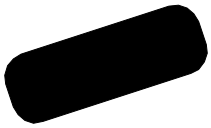
5 month female baby was admitted in our hospital with complaints of breathlessness, cough since last 7days. She was having history of recurrent respiratory tract infections since she was 1month of age. She was having history of 1 episode of bluish discoloration of tongue, nail beds in last 1month. She had been treated for her previous illnesses by local practitioner by symptomatic treatment. Her parents had family history of 3rd degree consanguineous marriage. She was 6 months 3 weeks preterm vaginal hospital delivery with poor cry and birth weight of 750gms. Our case is 4th issue of her parents. First two baby were still births (1st male and 2nd female) We admitted the girl to our hospital in paediatric

Author's Affiliation: *Professor & Head, Dept of Paediatrics, **Assistant Professor, ***Residents, Padmashree Dr. Vithalrao Vikhe Patil Medical College & Hospital, Near Govt. Milk Dairy, Vilad Ghat, Ahmednagar – 414111, Maharashtra, India.

Reprint's Request: Dr. Sunil Mhaske, Professor & Head Dept of Paediatrics; Padmashree Dr. Vithalrao Vikhe Patil Medical College & Hospital, Near Govt. Milk Dairy, Vilad Ghat, Ahmednagar – 414111, Maharashtra, India.

E-mail: sunilmhaske@rocketmail.com

Cyanosis of Lips & Cyanosis of Tongue



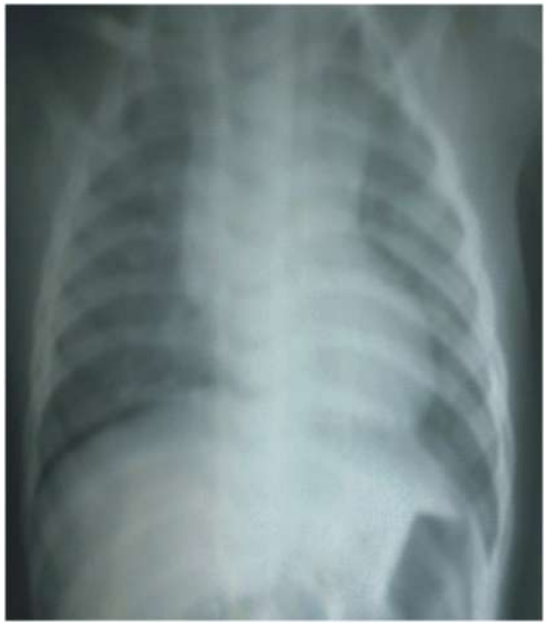
ward for further investigations & management.

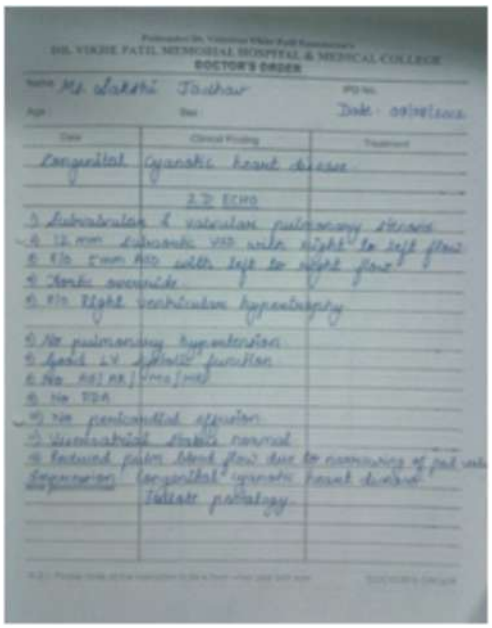
On examination, her pulse was 118beats/ minute & blood pressure was 85/50 mmHg. Central cyanosis was present. On examination of cardiovascular system, she was having pansystolic murmur. The murmur was prominent over left parasternal, mitral & aortic areas. No parasternal thrill was present.All other systemic examinations were within normal limits.

Her investigations were as follows:

- Hb: 10.9 gm%
- TLC: 8400/cmm

X-ray Chest Showing Boot 2D-ECHO Report Shaped Heart with Cardiomegaly



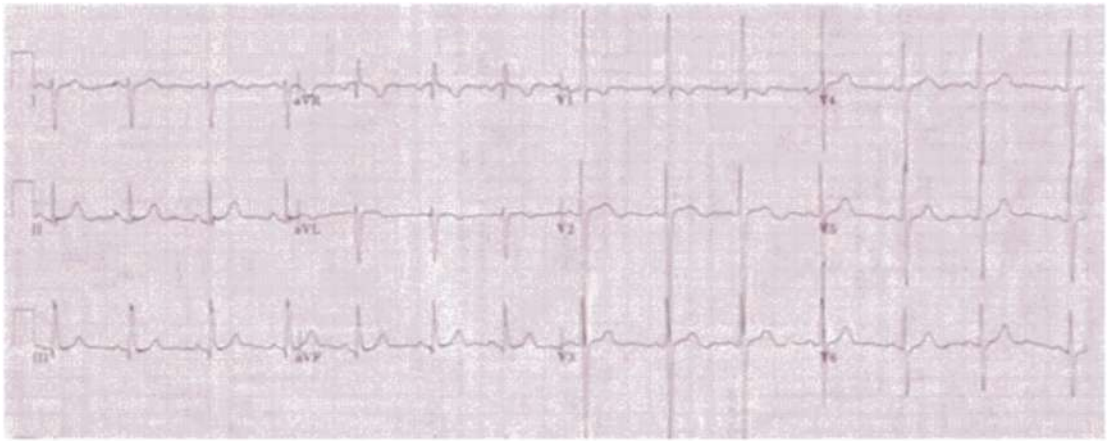


- P – 25, L – 70, E – 03, M – 02, B – 00.
- Platelet count: 2.28 lacs/cmm.
- ESR: 12 mm at end of 1st hr.
- ECG revealed right ventricular hypertrophy.
- Chest x-ray showed boot shaped heart with cardiomegaly.
- 2D ECHO & Color Doppler of heart states presence of a ventricular septal defect of 12 mm diameter with bidirectional shunt, atrial septal defect(septum secondum defect) of 5 mm diameter with left to right shunt, presence of overriding of aorta & reduced pulmonary blood flow due to narrowing of pulmonary valve(valvular and subvavular stenosis). There was evidence of right ventricular hypertrophy (RV wall thickness -18 mm)

Discussion

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart disease in children but occurs rarely in adults. Its etiology is still not clear but its embryogenesis involves anterior deviation of the septal insertion of the infundibular ventricular septum resulting in

ECG Showing R Wave in Lead V1 with RS in V2 (Sudden Transition), Right Axis Deviation, No Q Waves in Lateral Leads Suggesting Decreased Pulmonary Blood Flow



mal-alignment of the outlet septum, ventricular septal deviation (VSD), pulmonary outflow tract stenosis and aortic over-ride. In addition, right ventricular hypertrophy is noted secondary to pulmonary stenosis.

Clinical presentation consists of cyanosis, clubbing of the fingers, polycythemia and exertional dyspnea. Cyanosis and polycythemia may be noted in the newborn. The extent of cyanosis depends on the balance of systemic and pulmonary vascular resistance, which depends on the severity of right ventricular outlet obstruction. The more severe the obstruction, the more blood flows into the left side. Therefore, the more severe the pulmonary stenosis, the more protection from lung disease is noted. Mild pulmonary stenosis may present with mild cyanosis or even acyanosis, termed pink TOF or acyanotic TOF. Patients with this condition may have lung disease and may expire in early childhood if no repair or palliative surgery is performed.

Signs and Symptoms

Tetralogy of Fallot results in low oxygenation of blood due to the mixing of oxygenated and deoxygenated blood in the left ventricle via the VSD and preferential flow of the mixed blood from both ventricles through the aorta because of the obstruction to flow through the pulmonary valve. This is known as a right-to-left shunt. The primary symptom is low blood oxygen saturation with or without cyanosis from birth or developing in the first year of life. If the baby is not cyanotic then it is sometimes referred to as a "pink tet." Other symptoms include a heart murmur which may range from almost imperceptible to very loud, difficulty in feeding, failure to gain weight, retarded growth and physical development, dyspnea on exertion, clubbing of the fingers and toes, and polycythemia.

Children with tetralogy of Fallot may develop "tet spells." The precise mechanism of these episodes is in doubt, but presumably results from a transient increase in resistance to blood flow to the lungs with increased preferential flow of desaturated blood to the body. Tet spells are characterized by a sudden, marked increase in cyanosis followed by syncope, and may result in hypoxic brain injury and death. Older children will often squat during a tet spell, which increases systemic vascular resistance and allows for a temporary reversal of the shunt.

Treatment

Complete surgical correction is now the most important and standard treatment of TOF. Several factors, such as old age, high hemoglobin level, pulmonary artery hypoplasia and a diminutive left ventricle, have been identified as risk factors for operative mortality in many previously published series. Palliative surgery includes the B-T shunt or Potts shunt, which constructs a

communicating shunt between the systemic and pulmonary circulation. However, the outcome is poor and it is no longer standard treatment for TOF except when the patient's condition means they are not suitable for repair. It may be a bridge from symptom relief to total correction. Medication is used for symptom relief only.

Prognosis

Mortality is about 3% in children and 2.5% to 8.5% in adults. The survival rate of patients who receive repair surgery is about 86% at 32 years follow-up and 85% at 36 years follow up; survival rates of un-operated TOF patients older than 10 years is about 30%, older than 20 years 11%, older than 30 years 6% and older than 40 years only about 3%.

References

1. Topol Eric J and Robert M Califf. Textbook of cardiovascular medicine. Lippincott Williams & Wilkins; 2007.
2. Murphy JG, Gersh BJ, Mair DD, *et al*. Long-term outcome in patients undergoing surgical repair of tetralogy nof Fallot. *N Engl J Med*. 1993; 329: 593-599.
3. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults. Second of two parts. *N Engl J Med*. 2000; 342: 334-342.
4. Presbitero P, Prever SB, Contrafatto I, Morea M. As originally published in 1988: Results of total correction of tetralogy of Fallot performed in adults. *Ann Thorac Surg*. 1996; 61: 1870-1873.
5. Lien WP, Chen JJ, Chen JH, *et al*. Frequency of various congenital heart diseases in Chinese adults: Analysis of 926 consecutive patients over 13 years of age. *Am J Cardiol*. 1986; 57: 840-844.
6. Michael A Gatzoulis, Gary D Webb. Diagnosis and Management of Adult Congenital Heart Disease. 2003.
7. Fernando A Atik, Edmar Atik, Claudio R da Cunha. Longterm results of correction of tetralogy of Fallot in adulthood. *European Journal of Cardio-thoracic Surgery*. 2004; 250-255.
8. Wu Q. Indication and technique of total correction of tetralogy of Fallot in 228 patients. *Ann Thorac Surg*. 1996; 61: 1769-1774.
9. Nollert G, Fischlein T, Bouterwek S, Bohmer C, Klinner W, Reichart B. Long-term survival in patients with repair of tetralogy of Fallot: 36-year follow-up of 490 survivors of the first year after surgical repair. *J Am Coll Cardiol*. 1997; 30: 1374-1383.

Short Communication

Indian Journal of Anatomy
Volume 3 Number 2, April - June 2014

Nerve Blood Flow in Diabetic Peripheral Neuropathy: Revisiting the 'Vasa nervorum' Hypothesis

Kumar Senthil P.*, Adhikari Prabha, Jeganathan P.S.*****

Abstract

The aim of this short communication article was to highlight the role of altered nerve blood flow as a patho-anatomical manifestation in diabetic peripheral neuropathy (DPN), a common microvascular complication of globally prevalent metabolic disorder, diabetes mellitus. Limited evidence suggested that higher epineurial blood flow was present in DPN, which indirectly influenced impairments in exercise-induced nerve conduction increments. Newer methods like Nerve photography and fluorescein angiography provide useful objective information on nerve blood flow in terms of epineurial vessel pathology score, epineurialarteriovenous shunting, nerve fluorescein appearance time and intensity of fluorescence. Treatments using Adenosine and adenosine A2A receptor agonist, and alpha-lipoic acid administration were shown o be beneficial, with dose-dependent effects on nerve blood flow.

Keywords: Neuroanatomy; Vascular neurology; Epineurial circulation; Neural hemodynamics.

The aim of this short communication article was to highlight the role of altered nerve blood flow as a patho-anatomical manifestation in diabetic peripheral neuropathy (DPN), a common microvascular complication of globally prevalent metabolic disorder, diabetes mellitus.

Eaton *et al*[1] compared epineurial haemodynamics (epineurial intravascular oxygen saturation and blood flow)in patients with 11 chronic painful and eight painless neuropathy subjects. Intravascular oxygen saturation was found to be higher in the painful neuropathy group compared to those without pain. Faster Fluorescein rise time in those with painful symptoms also indicated higher epineurial blood flow in those subjects.

Author's Affiliation: *Professor, Maharishi Markandeshwar Institute of Physiotherapy and Rehabilitation (Maharishi Markandeshwar University), Mullana-Amabala, Haryana, **Professor, Dept. of Medicine, ***Professor, Dept. of Physiology, Kasturba Medical College (Manipal University), Mangalore, Karnataka, India.

Reprint's Request: Senthil P. Kumar, Professor, Maharishi Markandeshwar Institute of Physiotherapy and Rehabilitation (Maharishi Markandeshwar University), Mullana- Ambala, Haryana, India.

E-mail: senthilparamasivamkumar@gmail.com

Nerve photography and fluorescein angiography:Tesfaye *et al*[2] developed newer techniques of sural nerve photography and fluorescein angiography as an index of nerve blood flow and studied 13 subjects with chronic sensory motor neuropathy, five non-neuropathic diabetic and nine normal control subjects. The study had following findings; "The mean epineurial vessel pathology score of the neuropathic group was significantly higher than the combined normal control and non-neuropathic diabetic groups. Direct epineurialarteriovenous shunting was observed in six neuropathic and one non-neuropathic diabetic patients and not in any of the normal control subjects. The nerve fluorescein appearance time was significantly delayed in subjects with chronic sensory motor neuropathy compared to both normal and non-neuropathic diabetic subjects. The mean intensity of fluorescence at 96, 252 and 576s, was significantly lower in subjects with chronic sensory motor neuropathy compared with other two groups."

Exercise-induced Responses

Tesfaye *et al*[3] recorded sural sensory conduction velocity in 12 non-neuropathic

diabetic subjects, 15 diabetic subjects with established neuropathy and 16 age-matched normal control subjects, before and after exercise to 80% age/sex predicted maximum heart rate, and concluded that the impairment of exercise-induced nerve conduction increment in diabetic neuropathy indirectly implicated impaired nerve blood flow in diabetic neuropathy.

Adenosine and Adenosine A2A Receptor Agonist

Kumar *et al*[4] examined the effects of chronic administration of adenosine and CGS 21680 hydrochloride (adenosine A (2A) receptor agonist) on motor nerve conduction velocity (MNCV), nerve blood flow (NBF) and histology of sciatic nerve in DPN rats. Adenosine (10 mg/kg, i.p.) Showed improvements insciatic MNCV and NBF in diabetic ratswhile CGS 21680 (0.1 mg/kg, i.p.) significantly improved NBF; but not MNCV.

Lipoic Acid

Nagamatsu *et al*[5] studied the effects of lipoic acid (LA) on oxidative stress in diabetic peripheral nerves of rats by measuring nerve blood flow (NBF), electrophysiology, and indexes of oxidative stress. LA was shown to have dose-dependent influence on NBF in diabetic nerves, but not on normal nerves.

Stevens *et al*[6] reported therapeutic effects of administration of the antioxidant DL-alpha-lipoic acid (LA) to streptozotocin-injected diabetic rats, as follows;"LA improved digital sensory but not sciatic-tibial motor NCV, corrected endoneurial nutritive but not composite NBF, increased the mitochondrial oxidative state without correcting nerve energy depletion, and enhanced the accumulation of polyol pathway intermediates without worsening myo-inositol or taurine depletion."

Limited evidence suggested that higher epineurial blood flow was present in DPN, which indirectly influenced impairments in exercise-induced nerve conduction increments. Newer methods like Nerve photography and fluorescein angiography provide useful objective information on nerve blood flow in terms of epineurial vessel pathology score, epineurialarteriovenous shunting, nerve fluorescein appearance time and intensity of fluorescence. Treatments using Adenosine and adenosine A2A receptor agonist, and alpha-lipoic acidadministration were shown o be beneficial, with dose-dependent effects on nerve blood flow.

References

1. Eaton SE, Harris ND, Ibrahim S, Patel KA, Selmi F, Radatz M, *et al*. Increased sural nerve epineurial blood flow in human subjects with painful diabetic neuropathy. *Diabetologia*. 2003; 46(7): 934-9.
2. Tesfaye S, Harris N, Jakubowski JJ, Mody C, Wilson RM, Rennie IG, *et al*. Impaired blood flow and arterio-venous shunting in human diabetic neuropathy: a novel technique of nerve photography and fluorescein angiography. *Diabetologia*. 1993; 36(12): 1266-74.
3. Tesfaye S, Harris ND, Wilson RM, Ward JD. Exercise-induced conduction velocity increment: a marker of impaired peripheral nerve blood flow in diabetic neuropathy. *Diabetologia*. 1992; 35(2): 155-9.
4. Kumar S, Arun KH, Kaul CL, Sharma SS. Effects of adenosine and adenosine A2A receptor agonist on motor nerve conduction velocity and nerve blood flow in experimental diabetic neuropathy. *Neurol Res*. 2005; 27(1): 60-6.
5. Nagamatsu M, Nickander KK, Schmelzer JD, Raya A, Wittrock DA, Tritschler H, *et al*. Lipoic acid improves nerve blood flow, reduces oxidative stress, and improves distal nerve conduction in experimental diabetic neuropathy. *Diabetes Care*. 1995; 18(8): 1160-7.
6. Stevens MJ, Obrosova I, Cao X, Van Huysen C, Greene DA. Effects of DL-alpha-lipoic acid on peripheral nerve conduction, blood flow, energy metabolism, and oxidative stress in experimental diabetic neuropathy. *Diabetes*. 2000; 49(6): 1006-15.

Note: In this research no any own findings of authors study & results , there is only focus on previous studies.

Guidelines for Authors

Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journal" developed by international committee of medical Journal Editors.

Types of Manuscripts and Limits

Original articles: Up to 3000 words excluding references and abstract and up to 10 references.

Original articles: Up to 2500 words excluding references and abstract and up to 10 references.

Case reports: Up to 1000 words excluding references and abstract and up to 10 references.

Online Submission of the Manuscripts

Articles can also be submitted online from http://www.rfppl.com (currently send your articles through e-mail attachments)

I) First Page File: Prepare the title page, covering letter, acknowledgement, etc. using a word processor program. All information which can reveal your identity should be here. use text/rtf/doc/PDF files. Do not zip the files.

2) Article file: The main text of the article, beginning from Abstract till References (including tables) should be in this file. Do not include any information (such as acknowledgement, your name in page headers, etc.) in this file. Use text/rtf/doc/PDF files. Do not zip the files. Limit the file size to 400 kb. Do not incorporate images in the file. If file size is large, graphs can be submitted as images separately without incorporating them in the article file to reduce the size of the file.

3) Images: Submit good quality color images. Each image should be less than 100 kb in size. Size of the image can be reduced by decreasing the actual height and width of the images (keep up to 400 pixels or 3 inches). All image formats (jpeg, tiff, gif, bmp, png, eps etc.) are acceptable; jpeg is most suitable.

Legends: Legends for the figures/images should be included at the end of the article file.

If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks from submission. Hard copies of the images (3 sets), for articles submitted online, should be sent to the journal office at the time of submission of a revised manuscript. Editorial office: **Red Flower Publication Pvt. Ltd., 48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi – 110 091, India, Phone: 91-11-22754205, 45796900, Fax: 91-11-22754205, E-mail: redflowerppl@vsnl.net.**

Preparation of the Manuscript

The text of observational and experimental articles should be divided into sections with the headings: Introduction, Methods, Results, Discussion, References, Tables, Figures, Figure legends, and Acknowledgment. Do not make subheadings in these sections.

Title Page

The title page should carry

1) Type of manuscript (e.g. Original article, Review article, Case Report)
2) The title of the article, which should be concise, but informative;
3) Running title or short title not more than 50 characters;
4) The name by which each contributor is known (Last name, First name and initials of middle name), with his or her highest academic degree(s) and institutional affiliation;
5) The name of the department(s) and institution(s) to which the work should be attributed;
6) The name, address, phone numbers, facsimile numbers and e-mail address of the contributor responsible for correspondence about the manuscript;
7) The total number of pages, total number of photographs and word counts separately for abstract and for the text (excluding the references and abstract);
8) Source(s) of support in the form of grants, equipment, drugs, or all of these;
9) Acknowledgement, if any; and
l0) If the manuscript was presented as part at a meeting, the organization, place, and exact date on which it was read.

Abstract Page

The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Material, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

Introduction

State the background of the study and purpose of thestudy and summarize the rationale for the study or observation.

Methods

The methods section should include only information that was available at the time the plan or protocol for the study was written such as study approach, design, type of sample, sample size, sampling technique, setting of the study, description of data collection tools and

methods; all information obtained during the conduct of the study belongs in the Results section.

Reports of randomized clinical trials should be based on the CONSORT Statement (http://www. consort-statement. org). When reporting experiments on human subjects, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000 (available at http://www.wma.net/e/policy/l 7-c_e.html).

Results

Present your results in logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra or supplementary materials and technical details can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal.

Discussion

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, what this study adds to the available evidence, effects on patient care and health policy, possible mechanisms); Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

References

List references in alphabetical order. Each listed reference should be cited in text (not in alphabetic order), and each text citation should be listed in the References section. Identify references in text, tables, and legends by Arabic numerals in square bracket (e.g. [10]). Please refer to ICMJE Guidelines (http://www.nlm.nih.gov/bsd/uniform_requirements.html) for more examples.

Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. J Oral Pathol Med 2006;35:540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003;61:347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997;195 Suppl 2:3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000;71:1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM, editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ 20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

More information about other reference types is available at www.nlm.nih.gov/bsd/uniform_requirements.html, but observes some minor deviations (no full stop after journal title, no issue or date after volume, etc).

Tables

Tables should be self-explanatory and should not duplicate textual material.

Tables with more than 10 columns and 25 rows are not acceptable.

Number tables, in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each.

Explain in footnotes all non-standard abbreviations that are used in each table.

For footnotes use the following symbols, in this sequence: *, ¶, †, ‡‡,

Illustrations (Figures)

Graphics files are welcome if supplied as Tiff, EPS, or PowerPoint files of minimum 1200x1600 pixel size. The minimum line weight for line art is 0.5 point for optimal printing.

When possible, please place symbol legends below the figure instead of to the side.

Original color figures can be printed in color at the editor's and publisher's discretion provided the author agrees to pay

Type or print out legends (maximum 40 words, excluding the credit line) for illustrations using double spacing, with Arabic numerals corresponding to the illustrations.

Sending a revised manuscript

While submitting a revised manuscript, contributors are requested to include, along with single copy of the final revised manuscript, a photocopy of the revised manuscript with the changes underlined in red and copy of the comments with the point to point clarification to each comment. The manuscript number should be written on each of these documents. If the manuscript is submitted online, the contributors' form and copyright transfer form has to be submitted in original with the signatures of all the contributors within two weeks of submission. Hard copies of images should be sent to the office of the journal. There is no need to send printed manuscript for articles submitted online.

Reprints

Journal provides no free printed reprints, however a author copy is sent to the main author and additional copies are available on payment (ask to the journal office).

Copyrights

The whole of the literary matter in the journal is copyright and cannot be reproduced without the written permission.

Declaration

A declaration should be submitted stating that the manuscript represents valid work and that neither this manuscript nor one with substantially similar content under the present authorship has been published or is being considered for publication elsewhere and the authorship of this article will not be contested by any one whose name (s) is/are not listed here, and that the order of authorship as placed in the manuscript is final and accepted by the co-authors. Declarations should be signed by all the authors in the order in which they are mentioned in the original manuscript. Matters appearing in the Journal are covered by copyright but no objection will be made to their reproduction provided permission is obtained from the Editor prior to publication and due acknowledgment of the source is made.

Abbreviations

Standard abbreviations should be used and be spelt out when first used in the text. Abbreviations should not be used in the title or abstract.

Checklist

- Manuscript Title
- Covering letter: Signed by all contributors
- Previous publication/ presentations mentioned Source of funding mentioned
- Conflicts of interest disclosed

Authors

- Middle name initials provided.
- Author for correspondence, with e-mail address provided.
- Number of contributors restricted as per the instructions
- Identity not revealed in paper except title page (e.g. name of the institute in Methods, citing previous study as 'our study')

Presentation and Format

- Double spacing
- Margins 2.5 cm from all four sides
- Title page contains all the desired information. Running title provided (not more than 50 characters)
- Abstract page contains the full title of the manuscript
- Abstract provided: Structured abstract provided for an original article.
- Key words provided (three or more)
- Introduction of 75-100 words

- Headings in title case (not ALL CAPITALS). References cited in square brackets
- References according to the journal's instructions

Language and grammar

- Uniformly American English
- Abbreviations spelt out in full for the first time. Numerals from 1 to l0 spelt out
- Numerals at the beginning of the sentence spelt out

Tables and figures

- No repetition of data in tables and graphs and in text.
- Actual numbers from which graphs drawn, provided.
- Figures necessary and of good quality (color)
- Table and figure numbers in Arabic letters (not Roman).
- Labels pasted on back of the photographs (no names written)
- Figure legends provided (not more than 40 words)
- Patients' privacy maintained, (if not permission taken)
- Credit note for borrowed figures/tables provided
- Manuscript provided on a CDROM (with double spacing)

Submitting the Manuscript

- Is the journal editor's contact information current?
- Is a cover letter included with the manuscript? Does the letter

1. Include the author's postal address, e-mail address, telephone number, and fax number for future correspondence?
2. State that the manuscript is original, not previously published, and not under concurrent consideration elsewhere?
3. Inform the journal editor of the existence of any similar published manuscripts written by the author?
4. Mention any supplemental material you are submitting for the online version of your article?

Contributors' Form (to be modified as applicable and one signed copy attached with the manuscript)

Call for Reviewers

The Indian Journal of Anatomy (ISSN 2320–0022) is a tri-annual print and online journal of the **Red Flower Publication Pvt. Ltd.** publishes original and peer-reviewed articles, for the dissemination of anatomical knowledge with clinical, surgical and imaging guidance. Includes articles of history, reviews and biographies, locomotors, splachnology, neuroanatomy, imaging anatomy, anatomical variations, anatomical techniques, education and pedagogy in anatomy, Human Anatomy, Veterinary Anatomy, Embryology, Gross Anatomy (Macroscopic), Microscopic Anatomy (Histology, Cytology), Plant Anatomy (Phytotomy), Comparative Anatomy, editorials, letters to the editor, and case reports. Articles of veterinary anatomy, comparative and other morphological sciences are accepted.

Readership: Anatomical specialties, veterinarian, embryologists.

Indexing Information: Index Copernicus, Poland, Google Scholar, ProQuest, USA, Genamics JournalSeek.

One must have at least five years of experience in the field after completion of the education in that field and at least five original research papers in journal(s).

Please note that the acceptance of your application will be at the sole discretion of the editors.

Please provide your complete information and affiliation in brief through e-mail or you can register your self on our website www.rfppl.org.

For more information, please contact:

Publication–in-charge

Red Flower Publication Pvt. Ltd.

48/41-42, DSIDC, Pocket-II

Mayur Vihar Phase-I

Delhi – 110 091

India

Phone: 91-11-22754205, 45796900, Fax: 91-11-22754205

E-mail: redflowerppl@vsnl.net, redflowerppl@gmail.com

Website: www.rfppl.org

Subscription Form

I want to renew/subscribe to international class journal **"Indian Journal of Anatomy"** of Red Flower Publication Pvt. Ltd.

Subscription Rates:
• India: Institutional: Rs.3200, Individual: Rs.1000, Life membership (10 years only for individulas) Rs.5000.
• All other countries: $260

Name and complete address (in capitals):

Payment detail:
Demand Draft No.
Date of DD
Amount paid Rs./USD

1. Advance payment required by Demand Draft payable to Red Flower Publicaion Pvt. Ltd. payable at Delhi.
2. Cancellation not allowed except for duplicate payment.
3. Agents allowed 10% discount.
4. Claim must be made within six months from issue date.

Mail all orders to
Red Flower Publication Pvt. Ltd.
48/41-42, DSIDC, Pocket-II, Mayur Vihar Phase-I, Delhi - 110 091 (India)
Tel: 91-11-22754205, 45796900, Fax: 91-11-22754205
E-mail: redflowerppl@vsnl.net, redflowerppl@gmail.com
Website: www.rfppl.org