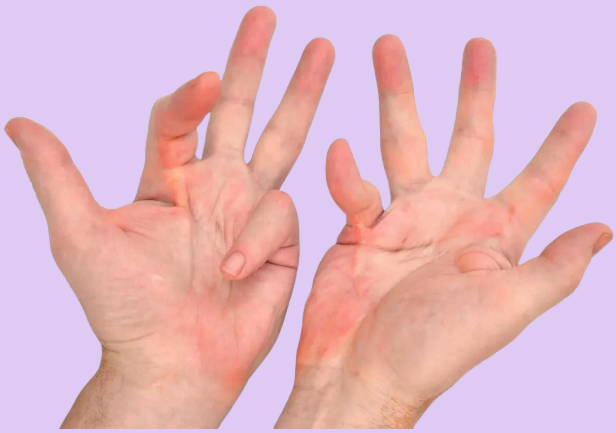
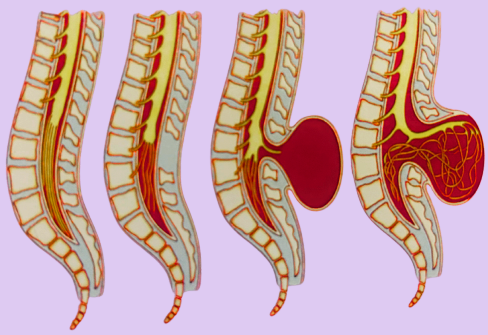
Volume 50, 2020

JOURNAL OF THE ANATOMICAL SOCIETY

KING GEORGE'S MEDICAL UNIVERSITY UP, LUCKNOW

CLINICAL ANATOMY





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Volume 50 ***2020***

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LT. GEN. (DR.) BIPIN PURI
PVSM, VSM,(RETD.)
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लेफ्टिनेंट जनरल (डॉ०) बिपिन पुरी
पी०वी०एस०एम०, वी०एस०एम० (सेवानिवृत्त)
कुलपति

KING GEORGE'S MEDICAL UNIVERSITY
U.P., LUCKNOW
किंग जार्ज चिकित्सा विश्वविद्यालय, उ०प्र०
लखनऊ

MESSAGE

Department of Anatomy KGMU has been one of the founding department of then King George's Medical College in 1911. During these 109 years of the Institute's history this department has made tremendous progress in teaching and training of young minds. Adapting to the new demand and change in approach from 'bench to bedside'; the department is making every attempt to deliver quality training in theoretical, practical, clinical and research fields.

The department has an Anatomical society which was established in 1956. The society organizes various academic activities annually viz. Paper writing and quiz competitions, debates etc. In this line the Anatomical Society, Department of Anatomy KGMU will be publishing this journal this year with theme - "Clinical Anatomy".

Through this opportunity the undergraduates will be exposed to the idea of evidence based medicine. This journal will help in introducing the students to the concept and idea of paper writing and will also spread the knowledge of various scientific data.

I congratulate all my dear students who have contributed in this journal and I hope this publication will also inspire other undergraduate students to contribute in future endeavors. I also compliment the faculty of Department of Anatomy KGMU for continuing this tradition and wish them good luck for the forthcoming issue of this journal.

Jai Hind

(Lt. Gen. (Dr.) Bipin Puri)
Vice Chancellor

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King George's Medical University
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Prof. Uma Singh
Dean, Faculty of Medicine

MESSAGE

It gives me immense pleasure to know that the Journal of the Anatomical Society, King George's Medical University, Lucknow is going to be published in the month of November 2020. It will provide a platform to students to enrich knowledge and develop skill by interacting and sharing their views with each other and faculty.

I extend my heartiest congratulations to Prof. Punita Manik, President, Anatomical Society and all the members of editorial board of the journal.

Wishing the journal a grand success.

13/11/20

(Prof. Uma Singh)
Dean, Faculty of Medicine
King George's Medical University, UP,
Lucknow.

MESSAGE

It is a pleasure to acknowledge that we have been successfully publishing the Journal "The Journal of Anatomical Society" from the Department of Anatomy, KGMU, Lucknow every year. I am happy to release the 50th volume of this journal this year.

Keeping in mind the changes in Medical Curriculum with stress on "Competency based teaching learning" the theme of the Journal was "Clinical Anatomy". We received an enthusiastic participation from students. The efforts put in by the students upgrades their knowledge in the field of Anatomy and the associated clinical application. The students have given their best.

I appreciate and express gratitude to Dr. Archana Rani, Chairman, Editoial Board and Dr. Garima Sehgal, Staff Editor and other members of the Editorial Board, Anatomical Society, KGMU, Lucknow in providing their valuable time and suggestions.

I wish Good Luck to all.

PManik

(Dr Punita Manik)
President
Anatomical Society
King George's Medical University
Lucknow

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The Journal of the Anatomical Society

2020

Volume L

Editorial Office : Department of Anatomy,
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Annual Report of Anatomical Society, KGMU, Lucknow

Human anatomy is an inextricable part of our medical education and learning anatomy not only from the books but also from cadaveric dissection is the prime focus of our anatomical society. While writing this report, I realise that we have come to the end of the wonderful academic session of 2019-2020. But as we enter another exhilarating year of our undergraduation, I Nikita Singh, Secretary of Anatomical Society KGMU takes up immense pride to give the annual report of the session in the new edition of the Journal of Anatomical Society (2019-2020).

Starting with the first event of the session i.e. The Debate Competition, held on 26th August, 2019. The topic of discussion was "India Requires Graduate Politicians". The panel of judges were:

1. Dr. Archana Rani
2. Dr. Siddhartha Koonwar
3. Dr. Anjani Pathak

The winners were:

1st – Sanya Dixit

2nd – Anshika Srivastava

3rd – Nikita Singh

Follwoing it, a ground breaking idea of organising a Rangoli Making Competition on 26th November 2019 on the occasion of the Constitution Day which was organised under the guidance of Dr. Anita Rani. The entire batch participated enthusiastically forming 10 groups and reflected their creativity in the form of colours on the same theme.

The Judges of this competition were:

1. Dr. Shally Awasthi

2. Dr Vinod Jain

The winners of this competition were the "Lucknawi Nawabs".

With all the academic work going on, soon the nation was hit by COVID-19, followed by a long period of lockdown due to which not only co-curricular but also curricular activities were affected but our dedicated teachers unquestionably adapted to the new mode of teaching via online live classes.

Following this, the next we had preliminary round of the prestigious Dr. Dharam Narayan Memorial Book Prize Competition which was held on 1st June, 2020 under the guidance of Dr. Jyoti Chopra. There were 20 participants in the first round who presented their topics via Google Meet which was judged by the professors of our department and eventually 10 students were selected for the final round which was again held via Google meet on 18th August, 2020.

The panel of judges for the final round were:

1. Dr. Anupam Mishra

2. Dr. Ashok Sahai

3. Dr. Ashish Kumar

The winners were:

1st- Nishant R. Subash

2nd- Shubhajeet Roy

3rd- Tanya Singh

The Anatomical society also issues the annual journal which publishes the articles submitted by the students. This year the topic for article writing competition was "Clinical Anatomy". Students presented their research works under the direction from Dr. Archana Rani and Dr. Garima Sehgal.

The best articles were of:

Winner- Bhavya Verma

Runner-up- Nikita Singh

The Annual Anatomy Quiz Competition was held on 25th November, 2020, where 5 teams participated.

The winners were:

Winners: Akanksha, Mahima Upadhyay, Fiza Akhtar, Shikhar S. Gupta

Runner-up: Bakshi Siddhant Vohra, Anuj Kumar, Abhishek Kumar, Kritika Jain

With all these activities the session comes to its end. In spite of many difficulties that students and teachers faced due to Covid lockdown, nevertheless this stirring session has taught us that undeterred determination of gaining and imparting knowledge is all that we need to keep growing.

At last I'd like to extend my sincere gratitude to our respected teachers for being a constant support throughout the year, placing their trust on me and giving me this golden opportunity.

Thank you.

Nikita Singh
(Secretary, Anatomical Society 2019)

EDITORIAL

The word 'Anatomy' finds its roots in the Greek language and literally means 'to cut apart' which is exactly one must do to observe and understand how something has been put together. Andreas Vesalius (Father of Anatomy), Charaka (FATHER OF INDIAN MEDICINE), and other great anatomists decoded the truth of the greatest work of evolution - the human body. Anatomy lays the foundation of all the clinical branches known to mankind.

'Cadaver' is the first teacher of a medical student. Cadaveric dissections give the opportunities to see anatomical variations as well as anomalies, which not only quenches the thirst of curiosity of students but also motivates them to study beyond anatomy. Though nothing can replace dissecting a human cadaver, but with the ongoing pandemic conditions of nationwide lockdown and COVID-19 raging across the globe, as the world became 'E', anatomy also switched to e-books and e-models. Even under difficult situation, Anatomy Department of King George's Medical University, Lucknow decided to go against all odds and preserve the tradition by publishing the 50th volume of Journal of the Anatomical Society.

Until and unless one is not well acquainted with the general perception of 'normal', the deviation cannot be identified. The journal presents systemic documentation of several anatomy topics and their applied. Thus, implementing the idea of early clinical exposure to first-year students by Medical Council of India.

We are eternally grateful to all the teachers for their efforts and commitments to the success of the journal. We would like to extend our heartfelt gratitude to Dr. Archana Rani, Chairman of the Editorial Board, and Dr. Garima Sehgal, Staff editor, for their never-ending patience and guidance, which was fundamental to the efficient completion of the journal. The journal is a sincere attempt to attract and inspire readers to take a deeper dive into the subject of Anatomy.

Poorvi Gupta
Ayush Yadav
(Student Editors)

J. Anat. Soc. KGMU, Lko
L, 1-4, 2020

ANORECTAL REGION: ANATOMY AND CLINICAL CORRELATION

Anuj Kumar
MBBS-2019 Batch

INTRODUCTION

The rectum and anal canal form the lower part of the gastrointestinal tract; rectum holds the stool until evacuation while the anal canal helps in the process of defecation through the internal sphincter muscle and the external anal sphincter. Rectum begins at the level of S3 and continues below as the anal canal that opens at the anal orifice and lies in the anal triangle of the perineum. The anorectal region is related to many maladies and the complications further increase with changing lifestyles. These anorectal disorders include anal fistula, anal fissure, anal abscess, fecal incontinence, and in several cases the malignant colorectal tumors.

The present article is the meta-analysis of the previous study on the rectum and anal canal general anatomy. It also reviews the common anorectal anomalies like hemorrhoids, anal abscess, anal fistula, anal fissure, fecal incontinence, and colorectal cancer.

RECTUM

The rectum begins at the sacral promontory, follows the sacrum and its concavity to reach the levator hiatus and then continues below as the anal canal. Typical characteristics of the large intestine i.e. appendices epiploicae, taenia coli and sacculations are absent in both rectum and the anal canal. It is covered by the peritoneum on the upper 2/3rd anteriorly and only upper 1/3rd laterally. The lower 1/3rd is devoid of any peritoneum. It is also covered by the endopelvic fascia called Denonvillers fascia where peritoneum is absent. It shows two anteroposterior curves and the three lateral curves. In males, the rectovesical pouch lies anterior to the rectum, and in females, the rectouterine pouch of Douglas lies anterior to the rectum. The interior of the rectum has both permanent and temporary mucosal folds. The temporary folds are longitudinal and disappear on distention. The permanent mucosal folds are called Houston's valve and are horizontal, semilunar, and are present along the concavity of lateral curves. The valves support the weight of the feces, prevent the passage of the instrument. The 3rd valve is most prone to injury as it is present on the anterior right wall along the cavity of the middle lateral curve.

The arterial supply of the rectum is mainly by the superior rectal artery which is the terminal branch of the inferior mesenteric artery after it crosses the pelvic brim to reach the pelvis. Its branches pierce the muscular coats to supply the internal of the rectum. The right and left middle rectal artery from the internal iliac artery also supplies the rectum distally and the anal canal proximally [1]. The middle rectal artery is inconstant in size and absent sometimes. The median sacral artery arises from the aorta and rums down in the anterior aspect of sacrum and terminates anteriorly in the rectal wall. The venous drainage mirrors the arterial supply. The superior rectal vein is the main venous drainage vein. It runs upwards crosses the pelvic brim to become inferior mesenteric vein which drains in splenic vein and thereby in the portal vein. Some of the veins are diverted bilaterally and drain in the internal iliac vein via the middle rectal vein. The venous blood also finds its way through the anal wall which then drains in the internal iliac vein via the internal pudendal vein. In the case of portal hypertension, these veins may enlarge and distend, and if ruptured may lead to heavy bleeding and can be life-threatening.

Lymphatic drainage follows the arterial supply, superior rectal lymphatics drain the upper part in the inferior mesenteric lymph nodes in the retroperitoneum, while they laterally drain into the

internal iliac nodes along the middle and inferior rectal veins through ischioanal fossa [2]. The lymphatics below the dentate line drain in the inguinal lymph nodes. Some of the rectal lymphatics drain into the lumboaortic lymph nodes that have no communication with internal iliac nodes [3]. The rectum receives both sympathetic and parasympathetic supply. The sympathetic nerves arise from the first 3 lumbar segments of the spinal cord and form the sub hypogastric plexus and are responsible for vasomotor supply. The parasympathetic supply is via pelvic splanchnic nerves and innervates the rectum to give secretomotor supply.

ANAL CANAL

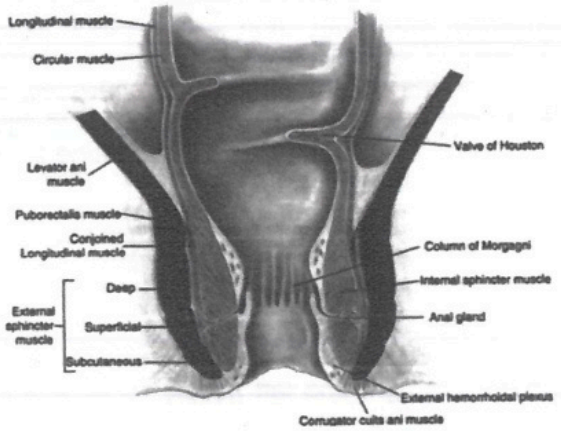

Fig. 1: Internal and external anal sphincters
(Adapted from Gordon PH, Nivatvongs S. Principles and practice of surgery for the colon, rectum, and anus. 3rd edition. Informaion Health-care; 2007)

Anal canal lies in the anal triangle region of the perineum and is 2.5 to 5 cm in length. It begins at the anorectal junction and runs downwards and backwards to open at anal orifice anterior to the coccyx. The internal anal sphincter is a ring of smooth muscle surrounding the anal canal and is involuntary. The external anal sphincter is a layer of voluntary muscle encircling the anal verge. It has three parts namely subcutaneous, superficial, and deep based on its location. The anorectal ring is formed by the puborectalis part of the levator ani and upper part of the internal and external anal sphincter. Injury to the anorectal ring may lead to incontinence. The mucus membrane of the anal canal shows many vertical mucosal ridges called the anal columns. In between the lower end of adjacent columns is present the curved mucosal folds called anal valves. Above each valve is a mucosal opening for the number of mucus-secreting anal glands. At the halfway in the anal canal present the dentate line [4]. The epithelium above the dentate line is the same as the rectum made of the columnar cells and goblet cells. The anal canal distal to the pectinate line is made up of non-keratinized stratified squamous epithelium.

The arterial supply of the anal canal is by the terminal branches of the superior rectal artery and the inferior rectal artery. The mucosa, proximal to the dentate line is supplied by the superior rectal artery while below the dentate line is supplied by the inferior rectal artery [5]. The internal and external anal sphincter also receives blood supply by the inferior rectal artery. Venous drainage from the upper part proximal to the dentate line is via superior rectal vein which eventually joins the portal venous system. Distal to dentate line venous blood drains via inferior rectal veins and the middle rectal vein. The dentate line forms the watershed between two different lymph node destinations. The distal half that is present below the dentate line is drained into superficial inguinal lymph nodes while proximal to the dentate line, lymphatics drain in the internal iliac lymph nodes. The nerve supply of the superior part of the anal canal is by the autonomic nervous system via inferior hypogastric plexus and the inferior part is via inferior rectal nerves.

ANORECTAL DISORDERS

(A) HEMORRHOIDS

Hemorrhoids are the most common disease affecting the anorectal region. It refers to the dilated or enlarged arteriovenous plexus in the lower region of rectum and anus. In a study, it is estimated that 50% of the population would have hemorrhoids probably by the age of 50 [6]. The main causes which lead to hemorrhoids are mainly constipation and prolonged straining; obesity, and heavy weight lifting further complicate the situation. Hemorrhoids are more common in pregnant females. This is due to constipation and increasing pressure. The common symptoms include anorectal bleeding, bright red stools, rectal prolapse, anal itching, extreme pain, etc. based on the location these are classified as the internal hemorrhoids if present above the dentate line and external hemorrhoids if present below the dentate line. Internal hemorrhoids are further graded on the degree of prolapse. Grade

1: the hemorrhoids bleed but not prolapse. Grade 2: the hemorrhoids bleed, prolapse but reduce spontaneously. Grade 3: the hemorrhoids prolapse and require manual replacement. Grade 4: The prolapse is irreducible [7]. The injection sclerotherapy, rubber band ligations, infrared photocoagulation or hemorrhoidectomy are the common non-surgical and surgical procedures performed to treat hemorrhoids.

1. ANAL FISSURE

Anal fissure is a tear in the mucosa or skin of the anal canal distal to the dentate line. Approximately 90% of anal fissures in both men and women lie posteriorly in midline due to maximum stretching on this site and less tissue perfusion. The main cause is the anal trauma due to the strained evacuation of hard stool or repeated passage of stool as in diarrhea. It typically causes pain and bleeding during defecation. Patients may also experience spasms in the sphincter muscles. Management generally includes increased fibers in diet and warm baths, resulting in healing of approximately half of the anal fissures [7].

2. ANORECTAL ABSCESS

Anorectal abscess refers to the abscess in the region of the anal canal and rectum. It is the infection originates most often from an obstructed anal crypt gland, with the resultant pus collecting in the subcutaneous tissue. This is mainly due to a blocked anal gland, sexually transmitted infection, or anal fissure. These are classified according to spaces they occupy as submucosal, perianal, ischiorectal, perirectal abscess [8]. The symptoms may be painful, hardened tissue around the anus, discharge pus from rectum, fever, constipation. The pain is usually constant, throbbing, and worse when sitting down and fatigue.

3. ANORECTAL FISTULA

An anal fistula is an abnormal connection between the epithelialized surface of the anal canal and the perianal skin. Most anal fistulas develop after an anal abscess does not heal properly. The risk factors which lead to a perianal abscess are Crohn's disease, diabetes mellitus, tuberculosis, trauma, radiation proctitis, foreign-body, infectionssuch as HIV, tuberculosis, or actinomycosis, and malignancy [8]. The patient generally complains of intermittent discharge of mucus or pus in the perianal region, often faecally stained. The bleeding, painful bowel movements, fever further complicate the symptoms. To minimize the risk of fistula, any abscess in the anal region should be drained early and thoroughly.

4. FECAL INCONTINENCE

Fecal incontinence is the inability to control the bowel movements, causing feces to leak unexpectedly. Aging has been consistently identified as a major risk factor for the development of fecal incontinence. According to a study after the age of 65, 20% of community-dwelling women and 60% in home residents, it is a common problem [9]. The decreased strength of internal anal sphincter, weak anal squeeze, anal sensation are other common causes leading to it. Dietary modification is the initial step in the treatment of incontinence. Anal sphincter and pelvic floor exercises are some of the nonsurgical methods. Surgical methods for its treatment include sphincteroplasty, muscle transposition, and artificial sphincter [10].

CONCLUSION

The anorectal region consists of a small but complex region. Anorectal disorders may be benign and can be treated easily by nonsurgical methods and changing the daily lifestyle. The clinician should take a proper history and physical examination of the patient for the correct diagnosis, severity, and treatment. Clinicians should maintain a high index of suspicion for anorectal sepsis and anorectal neoplasms.

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L, 5-8, 2020

ARCHES OF THE FOOT: ANATOMY AND ANOMALIES

Nikita Singh
MBBS- 2019 Batch

INTRODUCTION

The foot is made up of tarsus (includes five tarsal bones) and five metatarsal bones which do not lie in the horizontal plane instead they are arranged in the form of arches which are both longitudinally and transversely arranged [1]. These arches are just like curved stone bridges that are supported by slings, tie beams and intersegmental ties. This underlying engineering method is replaced in the body by the ligaments and the various muscles of the leg and the foot which provides flexible platform for bearing the body weight during standing, act like spring boards for shock absorption during jumping and are also important for the propulsion of the body during walking and running [2].

ANATOMY OF THE ARCHES OF FOOT

The arches which are present in the foot since birth include; the medial longitudinal arch, the lateral longitudinal arch and the anterior and posterior transverse arches.

Arch Bones

Visual inspection of an articulated foot or the lateral radiograph of the foot helps to observe the bones that form the arches. The bones mainly involved are: the calcaneus, the navicular, the cuboid, the talus, the cuneiform as well as the metatarsals. The phalanges are not involved in the formation of the arches.

Medial Longitudinal Arch

Lies along the medial border of the foot and is the largest, the highest and most resilient arch of the foot.

Bones: Formed by comprises the calcaneum (the posterior strong pillar), talus, navicular, medial, intermediate and lateral cuneiform bones and the first three metatarsal bones (anterior long/weak pillar). The summit of the arch is formed by superior articular surface of the body of talus (Fig.1).

Arch Support Mechanism

(i) **Shape of the bones:** The tarsals and the metatarsals involved in supporting this arch are wedge-shaped where the broader part lies on the dorsal side and the smaller thin part lies inferiorly.

(ii) **Intersegmental ties:** This is formed by the plantar calcaneonavicular ligament (also known as the spring ligament) and the interosseous ligament which connects the adjacent bones. This counteracts the tendency of the lower edges of the stones to separate when the arch is bearing weight.

(iii) **Tie beams:** These are structures which connect the anterior and the posterior ends together thus preventing the separation of the pillars. It is formed by the medial part of the plantar aponeurosis, abductor hallucis and flexor hallucis brevis.

(iv) **Suspension slings:** Tibialis anterior and peroneus longus act as slings which support thebridge from above and prevent it from sagging.

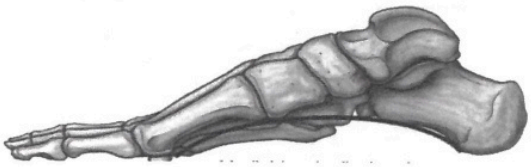

Fig.1: Formation of Medial longitudinal Arch [1]

Lateral Longitudinal Arch

This arch lies longitudinally along the lateral border of the foot. It is characteristically lower and its main function is the weight transmission to the ground.

Bones: This consists of the calcaneum, cuboid, and 4th and 5th metatarsal bones (Fig. 2).

Arch Support Mechanism

(i) **Shape of the bones:** Minimal shaping of the distal ends of the calcaneum and the proximal end of the cuboid is present. The cuboid is the keystone.

(ii) **Intersegmental ties:** The adjacent bones are held in position posteriorly by the short and long plantar ligaments.

(iii) **Tie beams:** The anterior and posterior ends of the lateral longitudinal arch are connected by the plantar aponeurosis, the abductor digitiminimi and the lateral part of flexor digitorum longus and flexor digitorum brevis tendons.

(iv) **Suspension slings:** The tendons of peroneus longus and peroneus brevis serve thisfunction.

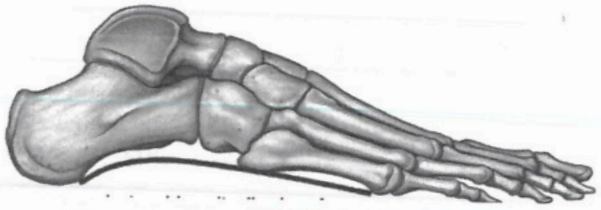

Fig. 2: Formation of Lateral longitudinal Arch [1]

Transverse Arch

The transverse arches include (Fig. 3):

1. Anterior transverse arch: is a complete arch formed by the heads of five metatarsal bones.
2. Posterior transverse arch: is an incomplete arch composed of the base of metatarsals and the greater portion of the tarsus.

Arch Support Mechanism

(i) **Shape of the bones**: There is a marked wedge shaping of the cuneiform bone and bones of the metatarsals.

(ii) **Intersegmental ties:** The inferior edges of the bones are tied together by the deep transverse ligament, the strong plantar ligament, the dorsal interossei and the transverse head of adductor hallucis.

(iii) **Suspension slings:** The transverse arches are supported by tibialis posterior along with peroneus longus and peroneus brevis tendon.

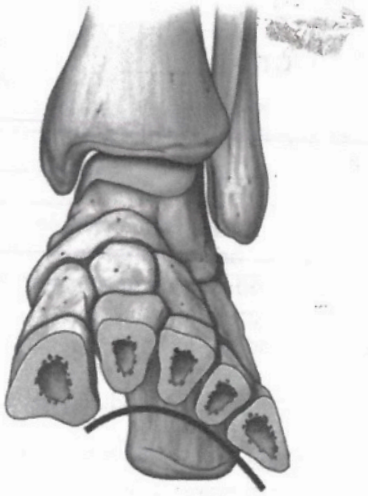

Fig. 3: Formation of Transverse Arch [1]

FUNCTIONS OF THE ARCHES OF FOOT

Weight distribution: On examination of foot imprint the medial margin of the foot is not seen, implying that it arches above the ground. Hence the weight is distributed on the heels and from the lateral border of the foot to anterior six points including two sesamoid bones under the head of first metatarsal and the heads of remaining four metatarsals.

Adaptation to uneven surfaces: The arches of the foot are segmented i.e., they are not formed by a single long bone but instead they are formed by many small bones with multiple joints between them making the foot capable of adapting to uneven surfaces.

Shock absorption: The medial longitudinal arch is mainly involved in shock absorption due to its high resiliency.

These arches also protect the underlying soft tissues that lie on the plantar surface of the foot.

ANOMALIES

Deviation from the normal structure produces unbalanced and functionally unstable condition of the foot; such as pes cavus and pes planus [3]. This variation in the foot posture is also associated with development of some lower limb injuries [4].

PES PLANUS

This condition is also referred as the flat foot, the arches of the foot especially the medial longitudinal arch loses its convexity and collapses (Fig. 4). Pes planus is fairly common in infants [5].

Etiology:

This may be congenital or acquired [6] and the cause may also be physiological or pathological. Acquired flat foot is likely to be secondary to the dysfunction of the tibialis posterior due to trauma, degeneration with age or denervation. In the absence of the normal passive or dynamic support, the planter calcaneonavicular ligament fails to support the head of the talus and the head of the talus displaces inferomedially, resulting in flattening of the medial longitudinal arch which occurs along with the lateral deviation of the foot [7].

It is commonly seen in older people, particularly if they undertake prolonged standing or gain rapid weight adding stress on the muscle and increasing strain on the ligament supporting the arches [7]. The common point in all deformities causing adult flat foot is failure of foot-locking during gait [8].

Treatment:

Children do not require much treatment for pes planus [9], foot orthodontics are indicated for foot pain secondary to pes planus and surgery is only indicated for rigid pes planus.

The pain and disability caused by flat foot can be treated by using shoe inserts, stretching, footwear selection and modification, activity manipulation, applying series casts, losing weight (if appropriate) and medication for pain and inflammation. Surgery is resolved for cases resistant to the therapy.

PES CAVUS

Pes cavus also known as claw foot, hollow foot or covovarus foot is a foot deformity which is characterized by elevated longitudinal medial plantar arch of the foot (Fig. 4) that does not flatten with weight bearing [10]. High-arched foot is the most common foot defect among children 3–13 years old regardless of gender [11]. It is a common finding in general population.

Etiology:

Pes cavus deformities may be associated with acquired, hereditary neurological disorders or various musculoskeletal conditions (e.g. peroneal tendonitis). Pes cavus may be a sign of an underlying neurological disorder including spinal cord and peripheral nerve pathologies like Charcot Marie Tooth (CMT) disease. It can also be associated with posterior compartment syndrome of leg or idiopathic subtle cavus feet without neurological disorders.

Treatment:

Treatment differ depending upon etiologies and severity of pes cavus.In cases of acute pes cavus, NSAIDs are given, biomechanical deficit are identified and addressed with orthosis. In chronic pes cavus, inspection of the footwear is done. While for individuals with idiopathic subtle pes feet without neurological conditions, addressing the presenting symptoms and conditions is the first step.

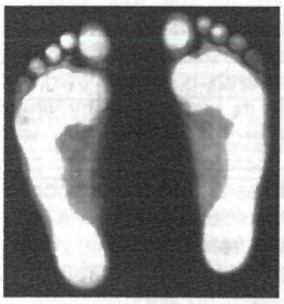

Fig.4.1: Scan of normal feet[11]

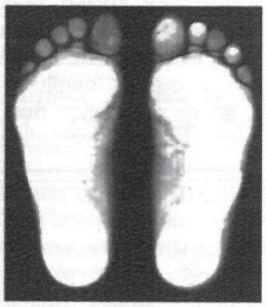

Fig.4.2: Scan of flat feet [11]

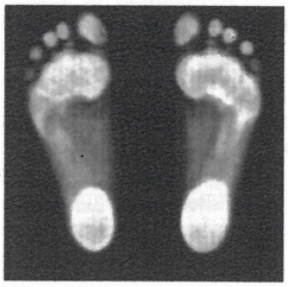

Fig.4.3: Scan of claw feet [11]

TALIPES / CLUBFOOT

This deformity is secondary to the defects in arches of the foot, talipes may be congenital or acquired and subdivided into [12,13]:-

(i) Talipes Calcaneus: the patient walks on the heels with the forefoot raised.

(ii) Talipes Equinus: the person walks on toes with the heels raised.

(iii) Talipes Valgus: foot is everted and abducted and the person walks on the inner border of his foot.

(iv) Talipes Varus: foot is inverted and adducted and the person walks on the outer border of foot.

(v) Talipes Equinovarus: excessively turned-in foot (equinovarus) and high medial longitudinal arch (cavus).

(vi) Talipes Calcaneovalgus: foot is hyper dorsiflexed, abducted and everted.

SUMMARY

The arches of foot are distinctive feature of the human race which are present right from birth. They are very known features of the foot which play important role in standing, walking and running .They also sustains stress of thrust and body weight. The medial longitudinal arch is mainly involved in shock absorption due to its high resiliency whereas the lateral longitudinal arch is involved in weight distribution as it makes more contact with the ground than the medial one. These arches are supported by various ligaments and muscle tendon of the leg and foot which prevents them from collapsing. However, if the support mechanism fails to maintain the shape of the arches then it leads to anomalies like pes cavus and pes planus.

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BLOOD SUPPLY OF FEMORAL HEAD AND OSTEONECROSIS

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MBBS 2019 Batch

INTRODUCTION

The femoral head gets its blood supply from the profunda femoris artery, the obturator artery, and occasionally from the superior and inferior gluteal arteries. They anastomose around the femoral neck and pass superomedially to supply the head. Osteonecrosis is a condition in which the arterial supply of the femoral head is compromised either due to traumatic or atraumatic conditions. The bone tissue undergoes necrosis and femoral head collapses causing impairment of hip joint mechanism. It requires managements like hip arthroplasty, restricted steroid, alcohol use, etc.

BLOOD SUPPLY OF FEMORAL HEAD

The femoral head gets its blood supply from four major arterial groups; medial circumflex femoral artery (MCFA), lateral circumflex femoral artery (LCFA), obturator artery and occasionally from superior and inferior gluteal arteries [1].

The profunda femoris artery (deep femoral artery) is a large branch that arises from the femoral artery and gives two branches i.e. medial circumflex femoral and lateral circumflex femoral arteries which are the major supplying vessels of the femoral head. It also gives three perforating arteries and itself gets terminated as the fourth perforating artery.

LCFA and MCFA via their ascending branches contribute to the anterior and posterior part of the extracapsular arterial ring, respectively. This ring is formed at the junction between the neck and shaft of the femoral neck and in turn, gives rise to ascending cervical vessels.

These vessels run along the extracapsular femoral neck and at the attachment of capsule, they enter intracapsular neck to continue as retinacular vessels. Retinacular vessels are in three groups; anterior group contributed by LCFA, posterosuperior and posteroinferior groups that receive contributions from MCFA. Out of these three, the posterosuperior group is most important and supplies a major part of the femoral head (Fig. 1).

The retinacular branches anastomose to form subsynovial arterial ring over the base of femoral head at the margin of articular cartilage which further gives rise to epiphyseal arteries that enter the head. The artery of ligamentum teres is more often a branch of the obturator artery but can also arise from MCFA. It traverses through the ligamentum teres attached at the margin of the fovea (a rough slit present on the posterior side of the center of the head) and supplies only a small portion of the femoral head at its entry.

Age-Related Changes in Blood supply:

1. < 4 yrs.: Lateral epiphyseal vessels at the posterosuperior aspect and metaphyseal vessels perforating the physis.
2. 4-8 yrs.: The epiphyseal plate develops between epiphysis and metaphysis and acts as a barrier to the supply of the metaphyseal branch. This time the only source of blood supply is the lateral epiphyseal branch of MCFA.
3. >8 yrs.: Artery of ligamentum teres become prominent and in anastomosis with the lateral epiphyseal branch, supplies the femoral head. Only in the pediatric population, it is a major supplying vessel [2].
4. Adolescence: Physis closes and the blood supply reaches the adult pattern. Via formation of sub synovial arterial ring, the blood supply from metaphyseal and lateral epiphyseal branch continues with the foveolar branch of the obturator artery.

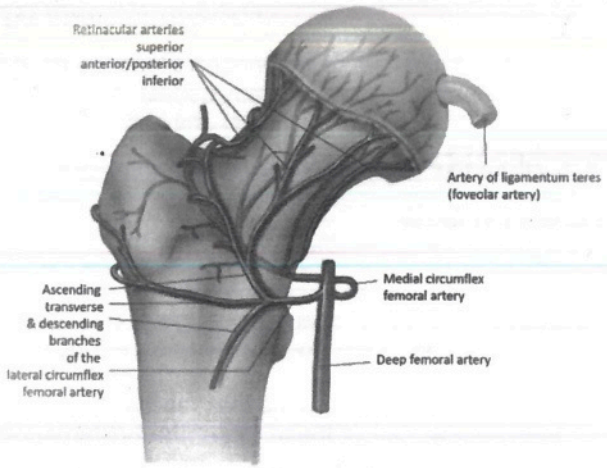

Fig.1: Arterial Blood Supply of the Femoral Head (Source: ALPF Medical Research)

CLINICAL ANATOMY

OSTEONECROSIS

Overview: Osteonecrosis (ON) is a condition in which the blood supply of the bone is compromised and leads to the death of bone marrow and osteocytes. It occurs mostly at the hip, knee, shoulder joints. The blood supply of the femoral head is fragile, vulnerable to injuries, and diseases; hence the hip joint is mostly affected by osteonecrosis. In the hip joint, the absence of adequate blood supply to the femoral head causes shape deformation of the femoral head and articular surface collapse leading to joint space reduction (degenerative arthritis). Damage to acetabular arterial blood supply might also take place besides disruption of femoral blood supply resulting in joint dysfunction [3].

Epidemiology: A study was conducted for the risk factors of ON of femoral head in the North Indian population. It included 249 patients diagnosed with ON that were evaluated between Jan 2005 - Jun 2013. It concluded that this disease affects people usually in the age group of 20-40 years [4]. Earlier this starts as a unilateral lesion but proceeds bilaterally within 2 years in more than 50% cases and requires total hip arthroplasty (THA). It is more frequent in males than in females with a mean ratio of 5:1 respectively [5]. The evaluated data of people affected by ON by different causes is given in Fig. 2.

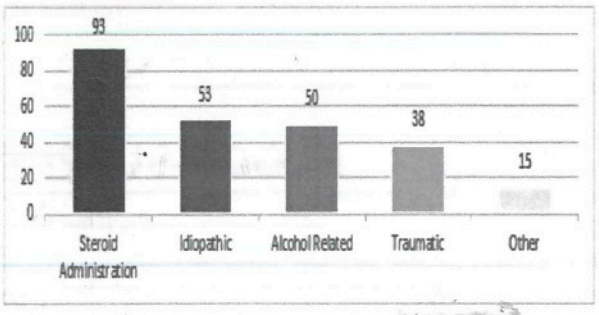

Fig. 2: Graph showing common etiologies of 249 affected patients (Source: Indian Journal of Orthopedics)

Etiology: The various causes can be broadly classified into:

1. **Traumatic**: Common types of traumatic injuries include hip dislocations and femoral neck fractures, of which, neck fractures are the most common cause (15-50%) than hip dislocations (10-25%) [6]. Hip dislocation usually results from a high energy trauma in a motor vehicle accident or fall from a height on legs. Hip dislocation is divided into two types: anterior and posterior dislocation of which posterior dislocations are most common. Femoral neck fractures are having common etiologies as hip displacement, the difference being fracture of the femoral neck instead of the femoral head like in hip dislocation. Both conditions include disruption of anastomosing blood vessels around femoral head and neck limiting/ blocking blood supply of femoral head and causing ON.

2. **Atraumatic**: Including alcohol abuse, chronic steroid use, sickle cell disease, idiopathic, etc. out of which steroid-related ON is the most common cause, followed by idiopathic and alcohol-related ON. Since the exact cause behind steroid-related ON isn't clear, several factors such as fat cell embolism and hypertrophy may cause increased intraosseous pressure, hyperlipidemia, or defect in stem cells of bone marrow resulting in ischemia and ON. Also, the pathophysiology of alcohol-induced ON isn't clear but some studies indicate fat cell hypertrophy and proliferation in bone marrow might be a possible cause. Other factors like hyperlipidemia, blood vessel occlusion, lack of perfusion in capillary bed also lead to the buildup of intraosseous pressure worsening the condition[7].In the case of sickle cell disease, the deformed and rigid structure of RBCs occlude the perforating arteries and cause

ischemia, while the femoral head being a common site affected in this disease[8].

Early diagnosis of disease must be done to preserve the hip joint. Once the joint collapse more than 2mm from the acetabulum and if it proceeds to secondary degeneration, hip arthroplasty is the only option left to cure disease.

History: Earlier, the condition is asymptomatic and pain is felt in the hip region radiating towards the groin. The pain usually worsens as one does exercises like walking, lifting weights, climbing stairs, but disappears on rest. One can easily evaluate whether having a condition of ON by doing a simple examination of hip abduction and medial rotation, as the range of movements in these exercises gets limited [9].X-ray scans take several months for the diagnosis of disease while MRI with the highest sensitivity and specificity is the best technique to identify early asymptomatic stages of ON.

Imaging Techniques: Various imaging methods are used for diagnosis of ON:

1. **Radiograph**: Plane anteroposterior and frog-leg lateral radiographs are used for diagnosis. Although they may be cost-effective, easily available but may not be effective enough to detect early stages and provides only a sensitivity of 40% [10]. Usually, radiography provides with a subchondral fracture that is present in later stages of ON [11]. As seen in Fig. 3, the superior aspect of the femoral head has been collapsed and became flatten due to ON.

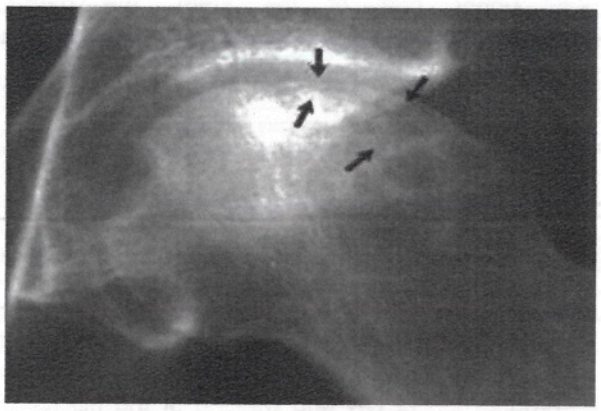

Fig. 3. Anteroposterior pelvic radiography showing flattening of the superior aspect of the femoral head of the left side (Source: Radiopaedia)

2. **CT Scans**: Usually, this technique isn't used for patients suspected of ON over MRI. But it gives an idea of subchondral fractures and a clearer image of bone changes than the MRI scan. Hence CT scans are better in a case to visualize subchondral lesions of the femoral head (Fig. 4) [11].

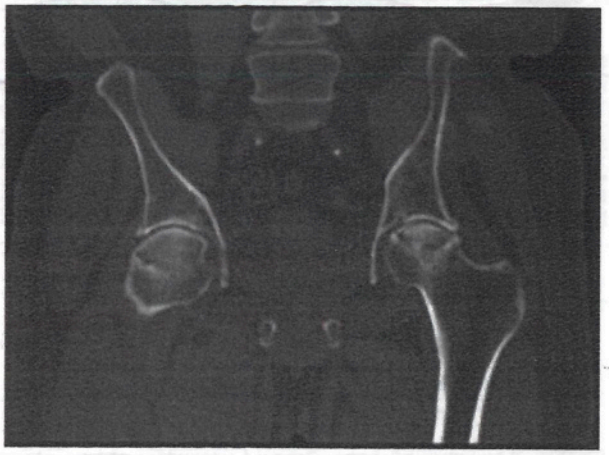

Fig. 4: Coronal bone CT findings showing bilateral ON of hip (Source: Radiopaedia)

3. **MRI**: Most appropriate imaging method with a sensitivity and specificity both being 99% when compared to plain radiographs, CT scans. Instead, MRI is used to detect alterations in the bone marrow which a radiograph is unable to identify. It is used for early detection of disease and evaluates the progression of necrosis in the femoral head. It shows a characteristic single dense band-like lesion in T1 weighted images showing regions of healthy and necrotic bone and bone marrow edema, indicating ischemia. Double band signs found in T2weighted images show the formation of hypervascular granulation tissue on the outer surface of the necrotic head during the healing process [12]. Fig. 5 shows a MRI scan of a patient diagnosed with bilateral ON in which the region of increased signal in the superior aspect of the femoral head represents fat, while the surrounding area of the decreased signal represents sclerotic reactive margin.

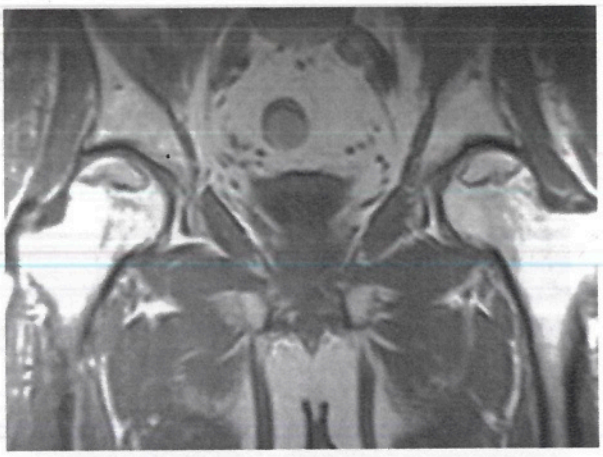

Fig. 5. Coronal T1 Weighted MRI Scan of a patient with bilateral ON of the femoral head (Source: Medical University Publishing House, Craiova)

4. Management: Before finalizing any operative methods, certain things must be evaluated in a patients' case like age, level of pain, the extent of ON, and femoral head collapse. As soon as the diagnosis is made, treatment must start including operative and non-operative techniques. Any delay in the condition might cause a subchondral fracture within 2-3 years [13].

Management Strategies:

1. Non-Operative Methods: Used to treat small to medium-sized pre-collapsed lesions either symptomatic or asymptomatic. These include restricted weight-bearing, alcohol use, abolish steroid therapy, etc.[14]. Even these treatments might give a sudden relief to the person, most of the time, the disease will progress and require surgical treatment.

Use of vasodilators and anticoagulants might release intraosseous pressure allowing blood to continue and revascularization of the femoral head [15]. Anticoagulants prevent embolism and coagulation preventing the progression of ON [16].

2. **Operative/Surgical Methods**: They are of two types:

(a) **Joint Reconstruction**: It includes bone grafting, core decompression, osteotomy, and cellular therapies.

Bone Grafting: Existing necrotic bone replaced by another bone tissue either taken from patients' own body (autograft), might be having its blood supply (vascularized bone graft), or from a bone bank (allograft).

Core Decompression: Three to four drills made through bone near the site of necrosis so that the intraosseous pressure developed is released. Caution is taken while deciding the point of drilling to be above lesser trochanter to avoid post-operative problems.

Cellular Therapies: Bone marrow is collected from the iliac crest portion and mesenchymal stem cells are isolated, cultured, and concentrated. This is mixed with platelet-rich plasma and injected via core decompression into the zone of the necrotic lesion. Then the tract is packed with a cancellous bone to prevent the backflow of products [17].

Osteotomy: It aims to shift the necrotic tissue from weight-bearing to non-weight bearing region hence allowing bone to heal up and healthy bone to bear the weight of joint. It includes trans-trochanteric or angular intertrochanteric (valgus or varus) rotational procedures. Trans-trochanteric rotational procedure is less suggested since it requires a greater area of healthy bone and repair of the joint by THA might be difficult [18,19].

(b) **Total Hip Arthroplasty**: Removal of the complete hip joint and replacement with a prosthetic joint made of metal/plastic is inserted which acts as a normal joint and allows similar functioning [20]. It contains a metal stem that is inserted into the femoral stem pressed or cemented, a ball either made of ceramics or metal replaces the femoral head is placed in the upper part of the stem, the damaged acetabulum of the hip joint is replaced by a metal socket and finally, a plastic liner is inserted in between the head and metal socket which provides smooth gliding surface.

CONCLUSION

Patients with symptoms of osteonecrosis must seek medical assistance immediately and get themselves diagnosed in the orthopedics radiology department. As earlier the diagnosis is made and preventive measures are taken, less will be the chances of progression of ON and wasting of the hip joint. As soon the person is diagnosed, respective measures should be taken to save the joint or if it's too late, THA is the only option left to go with.

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CONGENITAL VERTEBRAL DEFECTS: A CHRONICLE

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INTRODUCTION

Vertebral column is the central axis of the skeleton in vertebrates that protects the spinal cord and stabilizes the body by providing attachments to various muscles. It anatomically represents musculoskeletal and neural elements. In humans, it consists of 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 4 coccygeal vertebrae. Development begins during the gastrulation phase of embryonic development mesoderm that surrounds notochord separates into paraxial, lateral, and intermediate areas. Somites form in the paraxial mesoderm and develop in a craniocaudal direction; later each somite differentiates into a dermomyotome that forms muscle and dermis of the skin and a sclerotome which becomes the skeleton of the vertebral column.

Development of vertebral bodies is brought about by a complex process involving the Wnt, FGF, and Notch signaling pathways. Hox gene plays a role in orientation of the vertebrae in the craniocaudal direction [1] and the SHH gene plays an essential role in anterior-posterior development and ventral neural tube and somite patterning [2]. Disruption of this process and errors in genes and cell signaling processes can result in congenital vertebral deformities.

Congenital anomalies of spine can be classified on the basis of the type of error during the embryonic developmental process, and the region of the spine involved [2]:

1. **Neural Tube Defect/Spinal Dysraphism:** failure in closure of the neural tube during embryonic development, common example is spina bifida (defective fusion of the posterior spinal bony elements).
2. **Failure of segmentation:** failure of complete separation and division of vertebrae with accompanying partial/complete loss of a growth plate. Eg. congenital scoliosis and congenital thoracic hyperkyphosis.
3. **Failure of formation:** absence of the structural element of a vertebra. e.g. congenital scoliosis, kyphosis, and lordosis (least common) are examples of failure of formation. Failure of formation occurs in Klippel-Feil syndrome (typical observable defects are hemivertebrae or wedge vertebrae) [3,4].

1. NEURAL TUBE DEFECTS (NTDs)

Neural tube defects (NTDs) originate during embryonic development when the neural tube fails to close completely during the fourth week of embryogenesis, also referred to as spinal dysraphism (coined by Lichtenstein, 1940). They involve incomplete fusion or malformations of structures in the dorsal midline of the back. NTDs are common congenital anomalies, incidence ranges from <1 to 7 per 1000 live births; myelomeningocele (spina bifida) is the most common NTD [5–9].

Spinal dysraphism includes both open and closed defects based on the clinical presentation (Fig. 1).

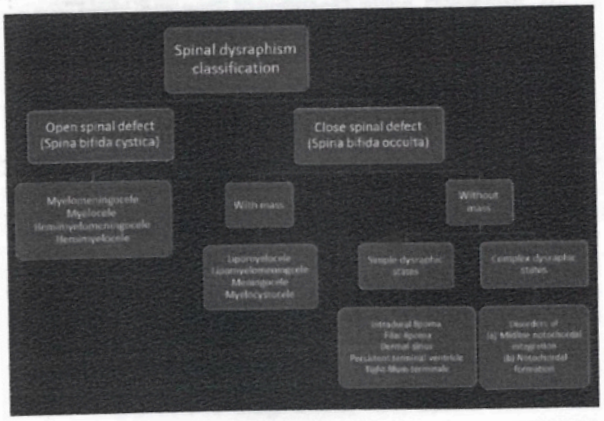

Fig. 1: Spinal Dysraphism (Reference: Columbia Asia Referral Hospital Yeshwanthpur- Bangalore/IN) [10]

Open spinal dysraphism or "spina bifida aperta/cystica":

Presence of a cleft in the vertebral column, and an associated defect in the skin, thus, the meninges and spinal cord are exposed/open to the external environment. Common manifestations are meningocele, myelomeningocele, lipomeningocele.

Closed spinal dysraphism or "occult spinal dysraphism" or "spina bifida occulta:

There is failure of fusion of the vertebral bodies, skin overlying the defect is intact, and hence neural tissue is not exposed. Isolated vertebral bony defects are more common and least severe forms. The lumbosacral region is the most common location of these defects [11].

Embryological mechanisms underlying NTD

Primary neurulation and occlusion: The central nervous system (CNS) is represented as a plate of thickened ectoderm, at the beginning of the third week of embryonic life, called the neural plate [12]. Around the 18th day, this develops into neural folds which fuse to form the neural tube, which subsequently expands cranially and caudally. Cranial neuropore closes on the 25th day followed by closure of caudal neuropore approximately two days later. This process is primary neurulation and is responsible for formation all of the functional CNS [13].

Myelomeningocele is caused by a failure of primary neurulation and poses the risks of CSF leakage and exposure of neural placode [14]. Extent and severity of neurological deficit depends on the degree of malformation and the level of defect, higher level is associated with worse prognosis [14].

Secondary neurulation: The mesenchymal cells fuse to form a solid neural rod, which later on forms a neural tube. [15]. It occurs below the surface ectoderm, hence abnormalities of secondary neurulation produce lesions that are covered by skin (occult spinal dysraphism) but the spinal cord is anchored to various tissues starting from the skin, subcutaneous tissue, adipose tissue or cartilage.

Anatomical Considerations of the Lesion

Myelomeningocele occurs due to failure of closure of spinal neural tube leading to malformation of the vertebral column and spinal cord. A split cord malformation may also occur in which case the dorsal portion of the spinal cord is only a hemicord, and the other portion of the split cord is ventral to the lesion, having a small midline opening at the superior margin which in opens into the central canal of the closed spinal cord. If sac ruptures, CSF from the central canal of the spinal cord can be seen coming out of this opening.

Etiology

The majority of myelomeningoceles are isolated malformations of multifactorial origin [15]. Most common causes and associated conditions are:

- In utero exposure to certain medications like valproate (antiepileptic) or folic acid antagonists [16].
- Gestational diabetes
- Maternal obesity
- Heritability (the genetic component of risk)
- Certain regional factors and consanguinity [14].

Clinically observed anatomical features

- Open dysraphism presents with a projecting membranous sac over the back recognized at birth, sac appears as a raw, red, fleshy plaque covered by granulation tissue or vascular malformations and contains meninges, cerebrospinal fluid (CSF), and nerve roots under the dysplastic spinal cord [Fig.2].
- Skin over the swelling is poorly developed and may rupture during labor, or after birth resulting in CSF leakage leading to neural infection.
- Neurological deficits may be variable depending upon severity and level of involvement, ranging from no deficits (in spina bi-fida occulta) to severe motor, sensory, and sphincter dysfunction. Severe cases may be associated with hypotonic limbs, atonic sphincters and with rectal prolapse.
- 80% of newborns with spina bifida (aperta) present with hydrocephalus causing cognitive deficits, attention deficits, poor executive skills, stridor, and apnea displayed by many patients with myelomeningocele, and are also responsible for most of the increased mortality.

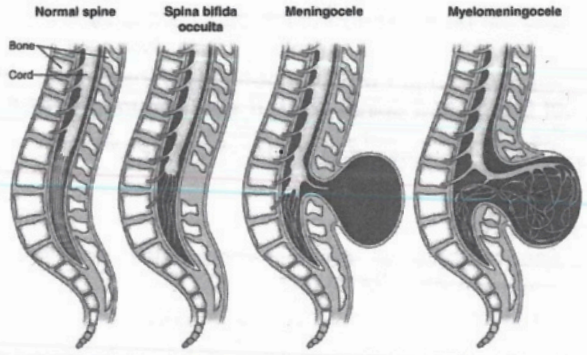

Fig. 2: Illustration showing characteristics of spina bifida occulta, meningocele, and myelomeningocele [17]

- Other associated congenital anomalies such as congenital heart disease, dislocation of hip, vertebral anomalies, kyphoscoliosis, cleft lip palate, ectopic development of renal and intestinal tissue, and umbilical inguinal hernias may be found.
- In spina bifida occulta, there is no protrusion of the cord and the meninges, the overlying skin is either normal or may show hypertrichosis, pigmentation, haemangioma, dimples, sinuses, and pedunculated tail.

MANAGEMENT

Pre-Natal

Preventive measures:

- Certain medications like antiepileptics to be avoided during pregnancy.
- Folic acid supplementation to be given during pregnancy (400 mcg daily).

Investigations:

- Prenatal ultrasound screening for anomalies performed routinely, up to 90% of cases of myelomeningoceles may be identified [14].
- Maternal Alpha Feto Protein (AFP) screening at 12, 22, and 32 weeks in high risk cases [14].
- Maternal serum screening for chromosomal abnormalities [18,19].
- MRI to study the neural tissue abnormalities and to assess the severity of hydrocephalus (investigation of choice).

Post-Natal

- Assessment of the newborn by a pediatrician and a neurosurgeon.
- CT scan for visualizing associated hydrocephalus and bony abnormalities of the spine.
- MRI to assess neural tissue deformities of the spine.

Surgical Management: Associated anomalies like hydrocephalus, Chiari malformation etc. should be identified and managed before considering surgical intervention. A pure meningocele involves excision of the meningeal sac and repair, a myelocele or myelomeningocele needs dissection, separation, and repair of the sac while preserving neural tissue.

2. FAILURE OF SEGMENTATION/FORMATION

Somites form and undergo a process of segmentation and recombination, errors during this process may lead to either failure of formation, failure of segmentation, or a combination of both (Fig. 3).

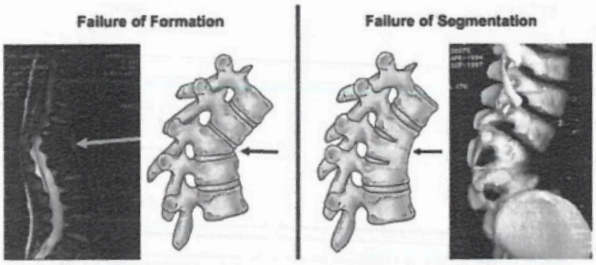

Fig. 3: MRI showing multiple wedged vertebrae (left image), failure of segmentation (right image). CT 3D reconstruction shows an example of combined vertebrae (right) [20].

Failure of segmentation results in:

- Congenital Thoracic Hyperkyphosis
- Congenital Scoliosis.
- Congenital Spinal Canal Stenosis

Failure of formation results in:

- Klippel-Feil syndrome
- Congenital kyphosis
- Congenital Scoliosis

CONGENITAL KYPHOSIS

Kyphosis is the forward curvature of the spine (30◦ to 60◦deformities) in a lateral view. Failure of the spine to segment or failure of separation of segments in utero, can give rise to a sharp angulation called congenital kyphosis, can occur in any part of the spinal column.

CLASSIFICATION

Winter et al. classified congenital kyphosis, it also helps to identify possible neurological dificits [20, 21, 22]:

- Failure of formation of the vertebral body (Type1),
- Failure of segmentation of the vertebral body (Type2)
- Formation failure with segmentation failure of the vertebral body (Mixed Type-Type3).

Pathogenesis

Developmental anomalies of the vertebra occur during the cartilage formation and ossification stages [22]. Longitudinal growth of the spine extends from superior and inferior endplate epiphysis of vertebral body [23]. The vertebral rotation insufficiency of the growth plate in front of the transverse axis on the sagittal plane leads to congenital kyphosis [22]. Hemivertebrae, butterfly vertebrae, and wedge vertebrae are formed due to the failure of segmentation of the vertebral body.

Symptomatology

The signs and symptoms in congenital kyphosis vary according to the severity of the curve, most symptoms remain constant and do not worsen with time, but, in more severe cases may lead to worsening if left untreated.

Symptoms include:

- **Physical Deformities:** in the form of rounded shoulders and a visible hump on the back giving appearance of poor posture
- **Stiffness:** spinal stiffness is common tight hamstrings are reported in many (the muscles in the back of thigh).
- **Pain:** constant pain in the back is also common and can worsen if not treated.
- **Fatigue:** day-to-day activities feel more taxing, muscle fatigue is also a common symptom.

Management of Kyphosis

Left untreated the curve continues to progress at about 7 degrees every year. The curve reaches its maximum orientation during the adolescent growth spurt and brace treatment during this period is ineffective [21].

The surgical choice for progressive disease is solid fusion of the involved vertebrae.

In Situ Fusion: involves fusion "where it is" with little or no correction of the spine

Instrumented Fusion Osteotomy: involves instrumentation by rods, hooks, and screws, to correct the deformity with progressive kyphotic curves in older children. Removal of distorted vertebrae may be considered by the surgeon to realign and straighten the spine.

Posterior fusion: without instrumentation (stopping the growth of the convex side) is considered for patients with Type 1 deformity, younger than 5 years of age, with a kyphotic angle lower than 50 degrees [24].

Posterior fusion without correction: is considered for Type 2 deformities (Winter and Moe).

Modified wheelchairs and orthoses are used to manage congenital kyphosis when operative treatment does not take place.

CONGENITAL SCOLIOSIS

Congenital Scoliosis (CS) is a lateral deviation of the vertebral column characterized by a longitudinal and rotational imbalance that develops during the intrauterine life, incidence lies between 0,5-1 per 1000 births.

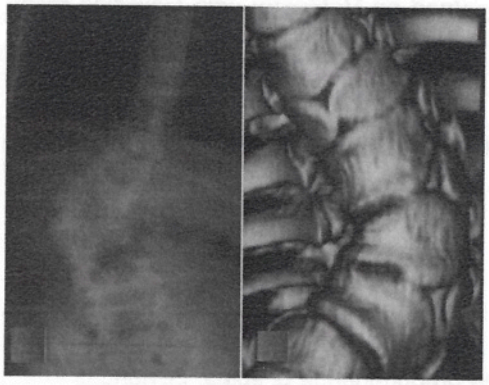

Fig. 4: Double curved congenital scoliosis due to 2 hemivertebrae disposed bilaterally, with a segment of 5 normal vertebrae in between [25]

This involves

- Three-dimensional deformity of lateral curvature and rotation
- Benign prognosis
- Worst curves with unilateral defects
- Curve site dependent on location of apical vertebra [26].

Congenital scoliosis represents 10% of pediatric spine deformity and is characterized by a longitudinal and rotational imbalance. Mortality rate increases in untreated cases, due to pulmonary problems.

The risk factors for progression are type of defect, size of the defect, and the patient's age.

ETIOLOGY

No specific etiology of has been identified.

The exposure to carbon monoxide during the formation period, fetal hypoxia of maternal, fetal or placental origin may be predisposing [27] Other inducing factors include gestational diabetes, intake of antiepileptics, prolonged febrile states of the pregnant women, or exposure of the fetus to temperatures higher than normal.

CLASSIFICATION

Winter studied 234 patients for the natural history and progression of congenital scoliosis. Winter's classification is widely accepted and followed classification [8, 9]. It was further modified and enlarged by McMaster who classified these defects into 4 types [10]:

- Type I — failure of formation
- Type II — failure of segmentation
- Type III — combination of an anterolateral unsegmented bar and one or more contralateral posterolateral quadrant vertebrae.
- Type IV — unclassified.

G Brunei, et al, in their study proposed a classification containing 2 large categories related to the predominance of spinal deviances in the coronal and transversal planes [25].

Congenital scoliosis with longitudinal imbalance:

- Hemivertebrae may be fully segmented, hemisegmented (partially segmented), or unsegmented.
- Malformations characterized by the presence of more than one hemivertebra may be:
- Adjacent (successive) - 2-3 hemivertebrae disposed unilaterally inducing short arch scoliosis, being noticed at birth and having a high rate of evolution
- Unilateral alternant (intermittent) - 2-3 hemivertebrae placed unilaterally leading to long arch scoliosis and a unique curve
- Bilateral alternant which maybe:

(a) Compensated: 2 symmetric hemivertebrae in a 4- 5 vertebral segment inducing an equilibrated spinal deformity not requiring surgery (Fig 4).

(b) Uncompensated: If the hemivertebrae are disposed on a distance of more than 6 vertebrae leading to a double congenital scoliosis.

Congenital scoliosis with rotational imbalance

- Spinal traction - osseous bridges with congenital transverse-sacralsynostosis.
- Spinal pushing-mega-apophysis of the L5 transverseprocess.
- Mixed (traction and pushing)-sacral agenesis with pelvic malposition.

ASSOCIATED ANOMALIES

Congenital scoliosis can occur in association with other anomalies /malformations

- **Neurologic malformations**

  In 35% of the patients it is associated with other neurologic malformations; the most frequently encountered are diastematomyelia, Chiari's malformation, intradural lipoma, and tethered cord.

- **Congenital heart malformations**

  In 25% of the patients congenital heart malformations are present, like Fallot tetralogy or the transposition of the great vessels that require surgery before a spinal surgical

approach [28].

- **Urological malformations**

  In 20% of the cases urologic anomalies are encountered, like horseshoe kidney, vesicoureteral reflux, or hypospadias.

Rates vary from 5 percent in patients with sacral level deficits, to 70 percent in patients with L3 level deficits, to over 90 percent in patients with deficits at L1 or above (Figure 5) [29]. The clinician should be particularly vigilant for the development of scoliosis in patients with a high level of neurologic involvement.

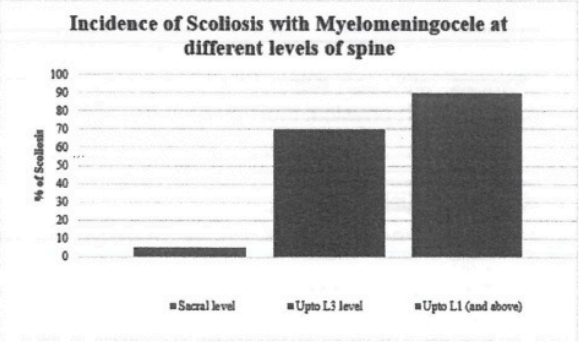

Fig. 5: Association of Scoliosis with severity of Myelomeningocele

Natural history

The progression depends on anomaly type, patient's age, and the location of the curve. Deformities are more with anomalies situated in the cervicothoracic or lumbosacral area. Type III-kyphosis/ kyphoscoliosis has highest progression rate followed by type I (about 10 degrees per year).

Management of Scoliosis

Conservative management (braces) is considered before surgery, to preserve the shape of the spine and delay early surgery. Spinal instrumentation for congenital spine deformity is safe and effective. Resection and monosegmental fusion are effective for treating congenital scoliosis caused by hemivertebra. Early surgery is indicated for children with congenital scoliosis. Growing rod surgery is an option for the correction of scoliosis, a modern alternative treatment for young children with early-onset scoliosis.

CONGENITAL SPINAL CANAL STENOSIS

The sagittal diameter must be <13 mm to be considered congenital spinal canal stenosis.

These patients have a greater risk of developing the spinal canal compression syndrome due to congenitally narrowed spinal canal. Clinical manifestations develop on the basis of the level of compression.

Congenital spinal canal stenosis is common in achondroplasia and may progress to paraplegia in some cases.

3. FAILURE OF FORMATION

Failure of formation may result in formation of:

- A Hemivertebra, which is a complete unilateral failure of the vertebral formation that can be: fullysegmented, partiallysegmented, non-segmented
- A hemivertebra can be further described as incarnated or non-incarnated.
- A wedge vertebra is a partial unilateral failure [30].

Congenital scoliosis can occur in association with other anomalies and syndromes like Klippel-Feil syndrome.

KLIPPEL-FEIL SYNDROME

The Klippel-Feil syndrome (KFS) is a congenital anatomical defect in the neck, which includes the fusion of two or more cervical vertebrae [31], also described as congenital brevi collis syndrome [32].

Feil classified this syndrome into 3 categories:

1. Type I = A massive fusion of the cervical spine
2. Type II = Fusion of 1 or 2 cervical vertebrae
3. Type III = Type I or II Klippel-Feil syndrome with thoracic and lumbar spine anomalies [33].

Etiology

It is caused due to formation and segmentation failure of cervical vertebrae during early weeks of development [32]. Proposed hypotheses include primary vascular disruption, global fetal insult, primary neural tube anomaly, genetic predisposition, and facet joint segmentation failure or may be the

result of maternal alcoholism, due to fetal alcohol syndrome [34]. Complications occur due to injury of the spinal cord which occurs by a minor fall. As there is absence of population screening studies on Klippel-Feil syndrome an approximate incidence between 0.5 - 0.7% of births has been described [34,35].

Clinical Presentation

- Short neck with a low hairline.
- Decreased mobility in the neck (sideways and rotational movements are more affected)
- Neck and extremity pain, weakness, ataxia, headaches, vision or hearing problems, and vertigo
- Torticollis or facial asymmetry in 21-50% of patients with KFS.
- Instability of the cervical spine can also develop between two sets of fusion anomalies separated by a normal segment.
- Between 30% and 60% have genito-urinary problems.
- Neurologic problems may develop in 20% of patients.

Several studies showed that the syndrome can present with other syndromes like Goldenhar syndrome, anomalies of the extremities.

Diagnostic Procedures

Radiographic evaluation is necessary to determine the diagnosis of Klippel-Feil syndrome.

- **X-Rays** for showing structural deformities.
- **MRI** for imaging of the spine. MRI and echocardiography should be an essential part of any evaluation of patients with congenital spinal deformity.
- **CT Scans** preferred for the assessment of localized bony abnormalities, or a calcified component, of the spinal canal, foramina, neural arches, and articular structures [36].

Management of Klippel-Feil syndrome

Degenerative changes may develop due to the formation of osteophytes due to osteoarthritic changes in the immobile joint, causing radiculopathy and/or myelomathy. Physical therapy is done to prevent or to delay this damage.

Surgical intervention is indicated in:

Cases with instability of the cervical spine and progression of the deformity, with neurologic deficits and persistent pain, with development of a compensatory curve in the thoracic spine.

Symptomatic spinal stenosis may require decompression and fusion.

Non-surgical approach:

Physical therapy along with non-steroidal medications could be useful to prevent degenerative changes, although Klippel-Feil syndrome cannot be resolved with physical therapy.

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DEVELOPMENT OF TONGUE AND CONGENITAL MALFORMATIONS

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MBBS-2019 Batch

INTRODUCTION

The tongue is a muscular organ that plays a role in of deglutition, taste and speech. It has oral and pharyngeal parts, and gains attachment to the hyoid bone, mandible, styloid processes, soft palate and the pharyngeal wall through muscles. It is subdivided into a root, an apex, dorsum and an inferior surface [1]. The embryological development of the tongue is complex and embryological development is represented by their adult anatomy. The substance of tongue includes mucous membrane, muscles and fibroareolar stroma; lingual mucosa is derived from endoderm of foregut, muscles are derived from the occipital myotomes and fibroareolar stroma is derived from the pharyngeal arch mesoderm. Epithelium is at first layered which later becomes stratified and papillae arise. Taste buds are formed near the terminal branches of the innervating neurons. The congenital malformations of the tongue are common, are of a wide variety and may be associated with a wide range of congenital syndromes, knowing which can help to predict possible deformities in other parts of the body. Deformities of the tongue not only have effects on the ingestion of food, taste sensation or speech, but also is a prime determinant of one's facial look and personality, thus having mechanical and social impacts as well [2].

Pax3, Dlx, TGF beta, CNCC molecules, Myf5, MRF4, FGF and SHH are important genes that have a major role in the entire developmental process, and mutation in any one of them might have serious consequences. Common congenital malformations of the tongue like ankyloglossia, cleft tongue and fissured tongue, not only affect deglutition, taste and speech, but have mechanical and social implications as well.

Hence, its early detection and correction is of prime importance.

STRUCTURAL DEVELOPMENT

Mucous membrane is derived from foregut, fibroareolar stroma is derived from the mesoderm of cranial 4 pharyngeal arches. The first sign of development of tongue is in the 4thweek, and by the 5th week, it is present as 2 lateral swellings and 1 medial swelling (tuberculum impar) (Fig. 1A) from the 1st pharyngeal arch. The lingual swellings enlarge to overgrow the tuberculum impar and merge to form the anterior 2/3rd of tongue (body) (Fig. 1B). Sensory innervation of this area is derived from the lingual branch of mandibular nerve (post-trematic branch of the 1st pharyngeal nerve). Their line of fusion is marked by the median sulcus. A sulcus forms along the ventral and lateral margins of the elevation and deepens to form the linguogingival sulcus. A midline swelling is formed in the floor of 2nd pharyngeal arch, known as copula, in the 4th week. The hypobranchial eminence forms in the floor of the 3rd arch, with contribution from the 4th arch as well. During 5th and 6th weeks, it grows over the 2nd arch and fuses with the anterior tongue rudiment along a V-shaped line called as sulcus terminalis thus forming the posterior 1/3rd. The sensory supply is from the glossopharyngeal nerve i.e. nerve of the 3rd pharyngeal arch (Fig. 1D). Also a small median diverticulum known as foramen caecum (thyroid diverticulum), is formed immediately caudal to the median tongue bud, present at the tip of sulcus terminalis. A final swelling originates from the posterior part of the 4th arch, which finally develops into the epiglottis. This part is innervated by superior laryngeal branch of vagus. Immediately behind this swelling is the laryngeal orifice, flanked by arytenoid swellings.

Most of the tongue muscles are derived from the myoblasts originating from the occipital somites. Tongue muscles precede the development of masticatory muscles and development is complete

by birth, all the muscles except palatoglossus are innervated by the hypoglossal nerve.

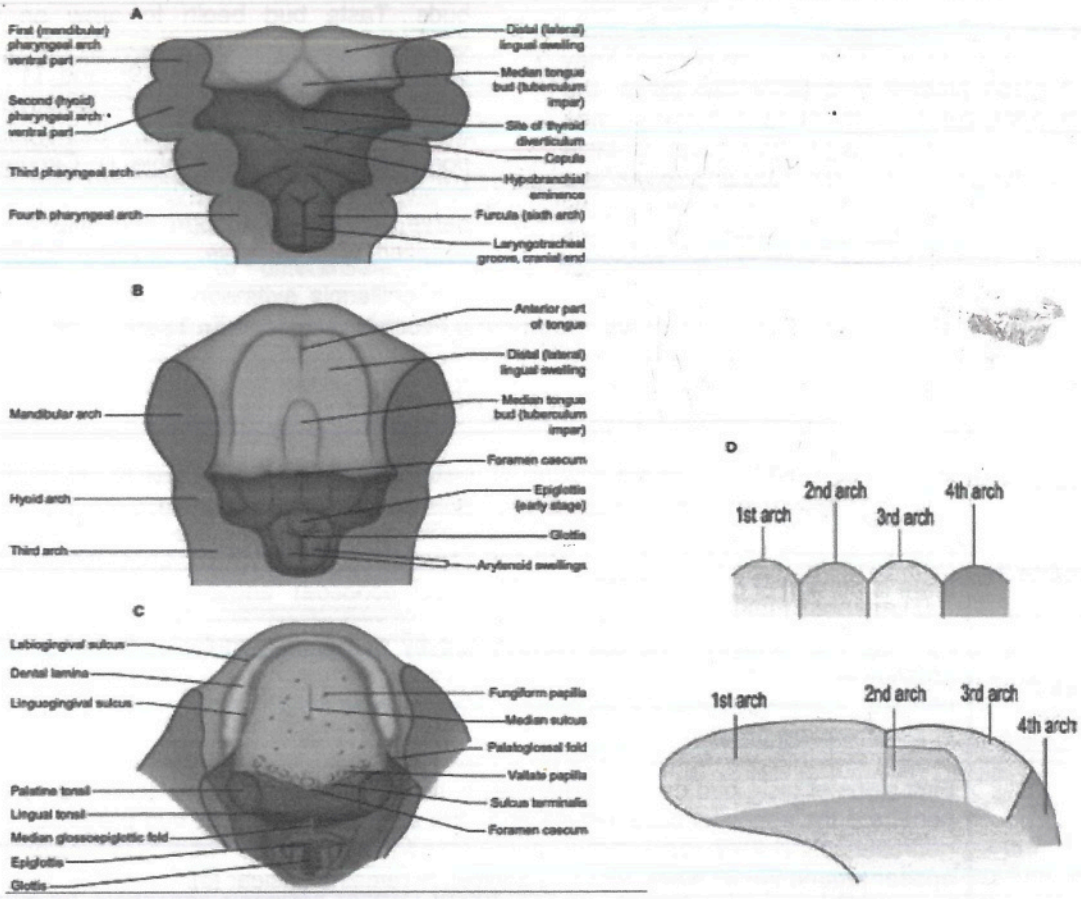

Fig. 1: (A) 4th week (development of tongue) (B) 5th week development (fusion of lateral swellings) (C) At birth[1] (D) Schematic diagram showing the burial of 2nd arch by overgrowth of 3rd arch [3]

The following graph shows the variation of foetal tongue circumference with gestational age (linear relationship) from 12th to 24th week (Fig. 2) [4].

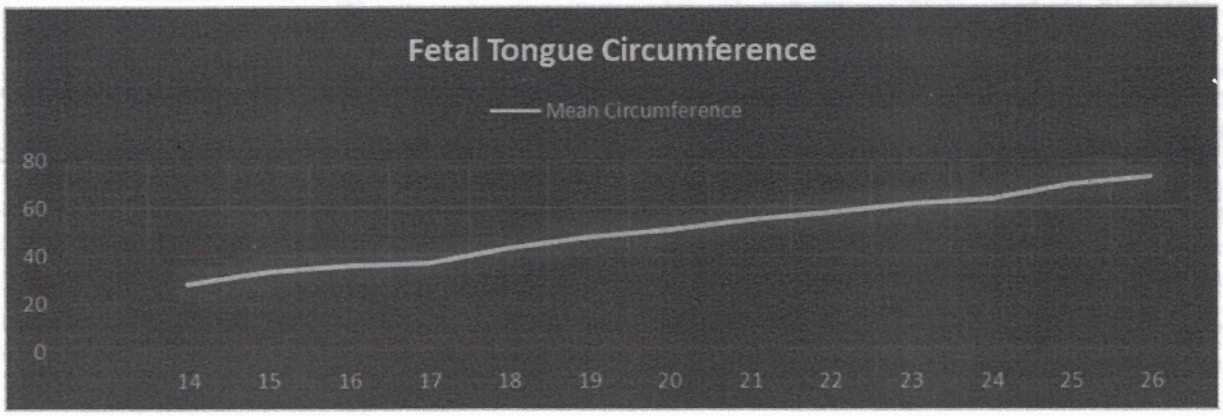

Fig. 2: Foetal tongue circumference variation with time. X axis is gestational age (in weeks) and Y axis is mean circumference (mm). Measurements are based upon multiple (2-10) separate ultrasound measurements

DEVELOPMENT OF PAPILLAE

Papillae are small nipple-like structures on the upper surface of the tongue giving it a rough texture. They are of 4 types present in anterior two thirds; the filiform papillae are the most numerous and provide a surface to assist in chewing but are devoid of taste buds. The fungiform and the foliate papillae are larger, less numerous and contain taste buds. Lastly the circumvallate papillae are located at the sulcus terminalis and also contain taste buds. Posterior third of the tongue is devoid of papillae and taste buds. Taste bud begin to grow on the lingual epithelium at about 8th weeks, many taste bud primordia develop between 9th and 11th week, and around the 11th through the 13th postovulatory week they differentiate into different cell types and taste pores develop as well (Table 1). Circumvallate and foliate papillae are innervated by glossopharyngeal nerve, whereas fungiform and filiform papillae are supplied by facial nerve.

Table 1: Detailed timeline of Developmental Processes of Human Taste Buds

Postovulatory Week	Description
Week 6	Surface covered by nearly flat epithelium, first gustatory papillae appear in the cin mid-line near foramen caecum and first circumvallate papilla develop on the dorsal mid-line [5].
Weeks 6 to 7	Epithelium consists of 2-3 cell layers and nerve fibers approach the basal lamina of epithelium there is no sign of cell specializations [6].
Week 7	Formation of fungiform papillae represented by series of irregular epithelial swellings in the anterior and marginal parts and also a V-like lane anterior to sulcus terminalis (circumvallate papillae). Papillae (fungiform) increase in size over the 8-15th weeks of gestation, whilst the size of circumvallate papillae tend to remain constant [5].
Week 8	First signs of taste bud development seen in lingual epithelium, nerve fibers from dermal papilla penetrate the epithelial basal lamina and form synapses with taste bud progenitor cells. These neuronal connections reach a maximum around the 12th to 13th week. Some ciliated cells also appear around the 8th week, whose significance remains unclear [6].
Week 9	Surface of circumvallate papillae contains a taste pit partly filled with microvillus-like processes from the underlying taste bud cells, taste pore not seen[5].
Weeks 10-11	Epithelium comprises of about four cell layers, papillae containing taste bud primordia are seen also visible are the first signs of a primitive pore formation [6].
Week 12	Clearly differentiation of taste bud cells into epithelial cell types II and III [6].
Weeks 12-13	The taste bud primordia are all located on the top of dermal papillae. Maximum synapses between cells and afferent nerve fibers, which intermingle with each other to form a plexus-like structure [6].
Weeks 14 -15	Shape and size of taste buds primordia of adult taste buds, by the 14th week taste pores develop. Taste buds acquire fully developed function in the 15th week, with the development of type I cells to produce the mucous material in the taste pit [6].

GENETICAL AND BIOCHEMICAL ASPECTS

Development involves complex interactions between Pax3, Pax7 and Dlx genes, for expansion and patterning of muscle. Signals from TGF beta controls proliferation of myogenic cells during morphogenesis. Interactions between CNCC molecules and myogenic regulatory factors- Myf5, MRF4, MyoD and myogenin influences development [7]. Taste buds develop from the mesenchyme but require local signalling to differentiate, proper development requires cooperative signalling of Six genes (Six1 and Six4) [8]. Transcription factors play an important role in gustatory neuronal development, i.e. placode formation and neuron differentiation: Differentiation of epibranchial placodes into petrosal and geniculate ganglion is brought about by transcription factors Six1/2, Six1/5 and Eya. BMP7 induces epibranchial placode formation. NGN2 causes the neuroblasts to migrate and fuse to form neurons, Phox2b maintains structural integrity of neurons (absence causes atrophy of the petrosal and geniculate ganglia). Neurons are overproduced and undergo programmed apoptosis, the total amount of neurons in the geniculate ganglion are quite stable. Neurotrophins BDNF, NT4/5 and NT3 regulate neuronal survival but also assist in axon growth from the sensory ganglion.

Sonic hedgehog signalling is essential for neural crest-dependent patterning of the intrinsic tongue musculature [9]. Hedgehog signalling controls SOX2 regulation, inhibition of the HH pathway causes taste bud loss. SHH over-expression induces ectopic taste buds with locally increased SOX2 expression hence taste bud differentiation depends on SOX2 [10].

A Wnt/Notch/PAX7 signalling network supports tissue integrity during development. Disruption of epithelial Wnt production by Wls deletion in epithelial cells causes failure in lingual epidermal stratification, loss of the lamina propria and underlying superior longitudinal muscle. Epithelial WNT production is required for activation of the Notch signalling pathway, which promotes proliferation of myogenic progenitor cells. Notch signalling in turn negatively regulates Wnt signalling during tongue morphogenesis. Pax7 is a direct Notch target gene in the embryonic tongue [11].

P2X receptors P2X2 and P2X3 are essential for taste transduction, knocking out P2X receptors reduces transmitter secretion of ATP in taste buds, but if only one receptor from the P2X family is knocked out, taste response may still be present [12].

FGF signalling and Spry genes regulate the development of circumvallate papillae (CVP). By knocking out both Spry1 and Spry 2 genes, number of CVP doubled hence Spry1/2 antagonize Fgf10 to limit the size of the CVP progenitor placode.

CONGENITAL MALFORMATIONS

Aglossia Congenita: is a rare defect involving a complete absence of tongue, commonly associated with Adactylia syndrome, belongs to a family of oromandibular limb hypogenesis syndrome (OLHS). Heat induced vascular disruption near 4th week of embryonic development may be causative [13].

Microglossia / Hypoglossia: is an uncommon developmental malformation, characteristic feature is a rudimentary abnormally small tongue with limitations in muscular movement and associated with the Hanhart syndrome [7].

Macroglossia: is an infrequent condition, characterized by tongue enlargement, commonly associated with Down's syndrome and Beckwith-Wiedemann syndrome. It is manifested by noisy breathing, drooling, difficulty while eating, lisping speech, mandibular prognathism and open bite [7].

Ankyloglossia (tied tongue): occurs due to failure in cellular degeneration, the frenulum linguae is extremely short and thick, restricting movement of tongue, oral hygiene, speech and feeding is also affected (Fig. 3) [14]. There is also difficulty in range and rate of articulation and compensation leading to Cupid's bow of the tongue [1]. It may also cause difficulties in breast feeding, leading to untimely weaning [15]. Mechanical and social effects like discomfort underneath tongue, difficulties with kissing, keeping one's tongue clean and performing tongue tricks may also be seen. Due to abnormal resting position of the tongue, nasal breathing is compromised, leading to chronic mouth breathing, it is correlated with tonsillitis, chronic ear infections; bruxism, gingival recession, open bite and TMJ pain may also occur. Due to an over tight frenulum linguae, tightness travels to neck, causing muscle tightness and poor posture [16]. Severity ranges from mild to complete, it is associated with Rainbow syndrome, Oral-facial-digital syndrome (Type 1), Opitz syndrome and Van der Woude syndrome.

Cleft tongue (bifid tongue): due to failure of lingual swellings to merge (Fig. 3), may be partial or complete; partial is more common and visible as a deep groove on the middle of dorsum of tongue. It is associated with Opitz G BBB syndrome, Oral-facial-digital syndrome Type 1, Klippel-Feil Anomaly and Larsen syndrome [7].

Fissured tongue / scrotal tongue / lingua fissurata: manifested as anteroposterior grooves (2-6mm in depth), on dorsal aspect of tongue with multiple branches extending towards the lateral aspect (Fig.3). In extreme conditions tongue appears to be lobulated. It is associated with Down's syndrome, Melkerson-Rosenthal syndrome (triad of fissured tongue, granulomatous cheilitis and VIIth nerve paralysis) and Cowden's syndrome [17].

Geographic tongue / benign migratory glossitis: inflammatory disorder, by loss of filiform papillae.

Black Hairy tongue: accumulation of excess keratin on filiform papillae, on the mid dorsal surface (Fig. 3), formation of elongated strands resembling hair. Gagging sensation, bad taste and intraoral halitosis are common [18].

Median Rhomboid glossitis: presents as a well-demarcated, symmetric and depapillated area in midline of dorsum of tongue just in front of the circumvallate papilla.

Leiomyomatous hamartoma: appears as a painless, soft polypoid mass, found in the tongue (Fig. 3). The lesion is composed of a proliferation of fusiform and spindle smooth muscle cells [19].

Glossoptosis: downward displacement or retraction of the tongue, may further lead to cleft palate. It is associated with Pierre Robin Sequence and Down's syndrome [20].

Choristomata: rare, nodule on the dorsum containing mature lamellar bone without osteoblastic or osteoclastic activity [21]. Cartilaginous and glial choristomas may occur very rarely [22,23].

Lingual thyroid: ectopic thyroid tissue is located at the base of the tongue, just posterior to the foramen caecum. It is 4-7 times more common in females, with symptoms during puberty, pregnancy or menopause. Symptoms include dysphagia, dysphonia and dyspnea [18].

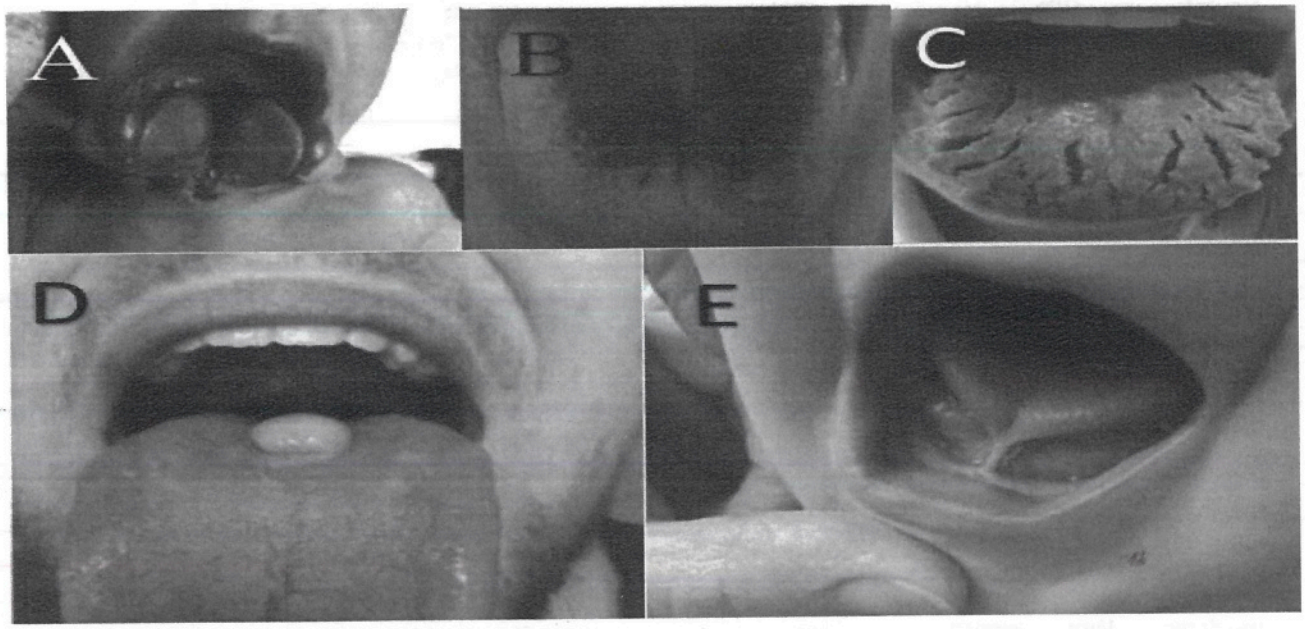

Fig 3: (A) Cleft Tongue (B) Black Hairy Tongue (C) Fissured Tongue (D) Leiomyomatous hamartoma (E) Ankyloglossia(Source: Google)

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DUPUYTREN'S CONTRACTURE: CURRENT CONCEPT AND LITERATURE

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INTRODUCTION

Dupuytren's contracture is a deformity of the hand involving fixed flexion contracture of the fingers mostly affecting the ring and little finger. There is difficulty in extension of fingers; fingers remain in a flexed state due to nodule formation and pathological cord contracture. Hypoxia theory suggests the involvement of Xanthine oxidase pathway in the pathophysiology of this disease. Many factors like alcohol, trauma, gender, rheumatoid arthritis, gout, diabetes mellitus, epilepsy, HIV, etc. are believed to be associated with Dupuytren's contracture but the true cause is still unknown. The Hueston tabletop test is performed to diagnose. Treatment options available include surgical and nonsurgical; some non-surgical options are corticosteroid injections, enzyme injections, needle aponeurotomy. Surgery is done for restoring function and correcting deformity; surgery usually remains successful, but recurrence is common.

Dupuytren's Contracture or Palmar fibromatosis, is a hand deformity in which 4th and 5th digits (ring and little finger) are pulled inward, towards the palm due to digital contracture caused by slowly progressing thickening, nodule formation or/and pathological cords formation in the Palmar and digital fascia of the hand. It is named after Baron Guillaume Dupuytren as he correctly identified the condition as fibrotic contracture of palmar fascia and described its surgical release in 1831. He published his work in The Lancet in 1834. The disease process has been described as early as 1777 and many more have contributed in the history of understanding and explaining this disease [1].

ANATOMY & PATHOANATOMY

To understand pathoanatomy of DD, it is important to know the normal anatomy [2]. It is a triangular layer of fascia with its apex towards palmaris longus muscle; it divides distally into 4 pretendinous bands which split at the distal edge of metacarpals into three layers. The 3 layers superficial layer, central layer and deepest layer attach to the skin at the palmar digital crease; give rise to two spiral bands which form web space coalescence (transverse ligaments named natatory ligaments join spiral bands in web space coalescence) and dive dorsally around metacarpophalangeal joint to insert on interosseous muscle fascia and deep transverse metacarpal ligament respectively. By this arrangement, seven short channels are formed in front of the head of metacarpal bones; through these flexor tendons and neuromuscular bundles pass [3].

The pathological changes seen in DD usually commence with nodule formation, generally between palmar distal crease and metacarpal crease. As the disease progresses, involvement of bands occurs. Normal bands form pathological cords which cause tissue or joint contracture. Firstly superficial layer of pretendinous band gets involved and then as Dupuytren'sdisease progresses, pretendinous bands, spiral bands and natatory ligaments get affected in this order. Dermal cords, pretendinous cords, spiral cords and natatory cords are the pathological palmar cords; mainly responsible for the clinical signs represented in disease. Other palmar cords; named according to their origin and location; Vertical cords, Digital cords, Ulnar cords, Radial cords, Thenar cords are also involved in DD at different grades [4, 5].(Fig. 1)

CLINICAL CORRELATION

Early sign- Skin on the palm of hands begins to thicken. Skin might appear puckered, pitted as nodules of hard tissue begin to form.

Intermediary sign- It is difficult to straighten out the fingers as nodules developed form thin bands of collagen, which pull fingers in towards the palm.

Later sign- Fingers remain bent towards the palm due to flexion contracture.

Commonly, both hands get involved, but one hand is more severely involved than the other, 4th and 5th digits are most affected. Nodules formed are generally painless [6, 7].

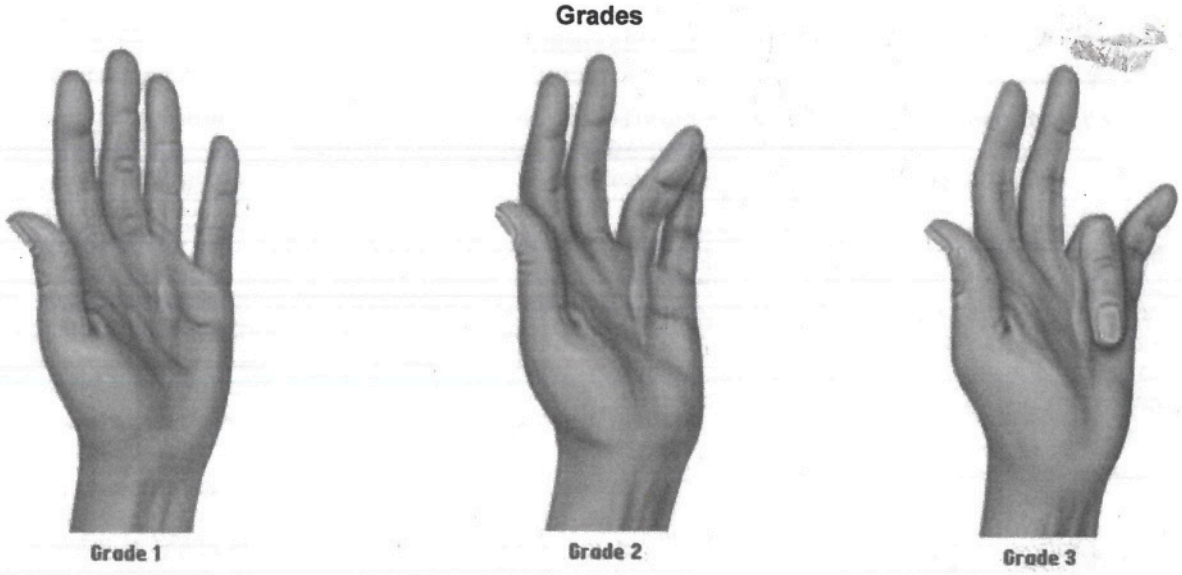

Figure 3: Grades of Dupuytren's Contracture [11]

Grade I (Fig.3a) - Thickened nodules and bands in the palmar aponeurosis with associated skin puckering. It shows early signs. Pathoanatomical changeoccurred till this stage is 1 (Fig. 1)

Grade II (Fig.3b) - Thickened nodules and bands, associated skin puckering and difficult to straighten out the fingers, with limitation of extension. Pathoanatomical change occurred till this stage is 1 + 2 (Fig. 1)

Grade III (Fig.3c) - Thickened nodules and bands, associated skin puckering and difficult to straighten out the fingers with flexion contracture. Pathoanatomical changes have happened till this stage is 1 + 2 + 3 + 4 (Fig. 1)

Etiology

Heredity: There is genetic susceptibility to the disease that follows an autosomal dominant pattern of inheritance.

Alcohol: Increased prevalence is seen in alcoholics (due to effect of alcohol on local circulation of palm. But the presence of DD in a person does not necessarily point to a high alcohol intake.

Trauma: Injuries and heavy work have been mentioned as predisposing factors due to immobility and swelling associated with injury [8].

Gender: DD is less prevalent in women than men, but its symptomatic presentation and surgical outcomes is similar to that in men. Recently, a difference in androgen receptor expression in DD's myofibroblasts has been noted that may explain the predominance of DD in men.

Rheumatoid Arthritis: Prevalence is reduced in patients of Rheumatoid, because of anti-inflammatory drugs taken by the patient as medication of Rheumatoid arthritis.

Gout: Lower risk in patients of gout because they are treated with allopurinol which inhibits Xanthine oxidase pathway.

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ECTOPIC PREGNANCY: A NARRATIVE

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INTRODUCTION

Ectopic pregnancy is also referred to as the extrauterine pregnancy, in this type of pregnancy the zygote is implanted outside the uterine cavity and the final outcome of which is usually an abortion or rupture. Clinical features of ectopic pregnancy may vary, the woman may be asymptomatic or may suffer from severe pelvic pain that is worse on one side or there may be rupture of the fallopian tube with hemorrhagic shock. The incidence of ectopic pregnancies has been on the rise, mainly due to an increase in the incidence of pelvic inflammatory diseases. Other contributing factors include advanced maternal age, assisted reproductive techniques, tubal surgery, congenital anomalies, and intra-uterine devices. It must be excluded in a female who has a positive pregnancy test, abdominal pain, and vaginal bleeding, diagnosis is confirmed by ultrasonography and laparoscopy. Treatment may medical management or surgical (laparoscopic salpingostomy) depending upon case requirements.

Ectopic pregnancy or extrauterine pregnancy involves implantation and development of the zygote outside the uterine cavity [1]. Implantation at ectopic sites affects the growth of zygote and thus results in abortion or rupture; very rarely does the fetus continue to grow in peritoneal cavity (abdominal pregnancy) even after tubal rupture.

ANATOMY

The most common site of ectopic pregnancy is the fallopian tube (97%); 75–80 % in the ampulla, 10–15 % in the isthmus, about 5 % in the fimbria of the fallopian tube, [2] 2.5% in the uterine cornu and 2% in other locations including the cervix, abdomen, and ovary [3] . Ovarian pregnancy is the most common non tubal ectopic type of pregnancy [4].

CLINICAL CORRELATION

Tubal pregnancy is the most clinically significant extra- uterine pregnancy [5] and is due to the inability of the fertilized egg to make its way through the Fallopian tube into the uterus. Any alteration in the normal tubal transport mechanism may lead to ectopic pregnancy. Alteration in tubal transport such as delay may be caused by episodes of previous pelvic inflammation that damage the tubal epithelium. Sometimes, the condition can occur without any apparent predisposing factor. The most frequent site for abnormal implantation is the wider ampullary portion of the uterine tube, but, it may also occur in the narrow intramural portion or in the ovary itself [6].

Sites of ectopic implantation are variable [7] (Fig.1)

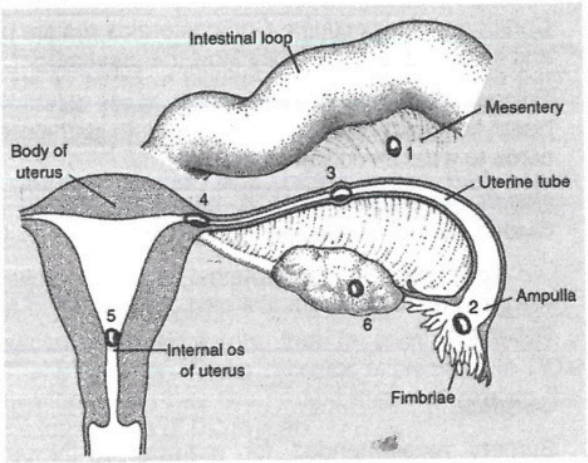

Fig.1: Sites of ectopic implantation

Most ectopic pregnancies are an embryonic; due to the continuing growth of trophoblast the pregnancy test may be positive. Ectopic pregnancy with live embryo is more dangerous because of rapid growth; it may usually be detected by 8 weeks of gestation when it has eroded the uterine tube wall

and surrounding blood vessels. Ectopic pregnancy in the uterine cornua may present with catastrophic haemorrhage because of rich blood supply in the surrounding muscularis. If an early diagnosis is missed ectopic pregnancy can be potentially fatal [8, 9]. The risk of tubal rupture is reduced and more conservative medical treatments can be employed if an early diagnosis is made [10].

Ectopic pregnancy occurs in 1% to 2% of all pregnancies [11]. Although its incidence has been increasing, mortality has declined as many cases are diagnosed early before rupture. It accounts for up to 6% of pregnancy-associated mortality [5].

The highest incidence is in the age group 35 to 45 years due to the cumulative effect of multiple risk factors [12].

The exact causative mechanisms are unknown; four main possibilities include: anatomic obstruction to passage of the zygote, an abnormal conceptus, abnormalities in the mechanisms for tubal motility, and transperitoneal migration of the zygote. Migration of the conceptus may be arrested at any point in the uterine tube where it may implant in its wall [6]. Spontaneous multiple ovulations or ovulation induction increases the risk of ectopic pregnancy [13].

Contributing factors include advanced maternal age, pelvic inflammatory diseases, assisted reproductive techniques, tubal surgery, congenital anomalies, intrauterine device [14]. Endosalpingitis leads to mucosa damage and may cause entrapment of migrating embryo, exosalpingitis may lead to formation of peritubal adhesions impairing peristaltic movements [9]. Risk factors for ectopic also include; infection with Chlamydia trachomatis or Neisseria gonorrhoeae, appendicitis [15], prior tubal surgery/ prior tubal pregnancy, or postpartum pregnancy where incomplete tubal occlusion due to edematous, congested and friable tube increases the chance of ectopic implantation [5]. No recognizable factor may be identified in half of all women with an extrauterine pregnancy.

Abdominal pregnancies have a prevalence of about 1%, the gestational sac is implanted outside the uterus, ovaries, and fallopian tubes, placenta can be attached to the uterine wall, bowel, mesentery, liver, spleen, bladder and ligaments, may undergo a partial or complete placental separation, causing massive hemorrhage and consequently increased risk of maternal mortality (20%) [16, 17].

The rise in incidence can be attributed to early diagnosis. Diagnosis of extrauterine is mostly done in the 6th to 9th week [18]. Indicators of ectopic pregnancy include mild vaginal spotting in first trimester, aching pelvic pain, and secondary amenorrhea. Other symptoms may be; abdominal pain radiating to the shoulders, abdominal guarding, acute abdomen, pain on displacement of the vaginal portion of the cervix, hemorrhagic shock/ hemodynamic instability (dyspnea, hypotension, tachycardia), and syncope, adnexa on the affected side is often enlarged and tender [5].

The pregnancy test, serum β-hCG (Beta human chorionic gonadotropin) concentration and ultrasound are used for making a diagnosis. Ultrasonography should be the initial investigation for symptomatic women in their first trimester; and serial measurement of β-hCG and progesterone concentrations may be useful if the diagnosis remains unclear [8].

A rise in β-hCG levels by < 66%, or a fall by< 13% from the baseline level, in 48 hours, along with an absolute β-hCG levels above 1500 IU/L in the absence of any visualizable intrauterine pregnancy, can be taken as evidence for a probable ectopic pregnancy. This combined criterion is 92% sensitive and 84% specific [5].

Treatment depends on clinical presentation, size and β-hCG levels. While performing surgery close inspection of the abdomen and pelvis especially the contralateral fallopian tube must be done. In several cases, the contralateral pregnancy can be found days to weeks after the initial surgery [12]. Surgical management is done in acute ruptured ectopic pregnancy, in haemodynamically unstable patients or in those who have failed medical treatment or where medical treatment is contraindicated. An early diagnosis reduces the risk of tubal rupture and the patient can be managed by medical or conservative surgical procedures [18]. The improved diagnostic methods have made the treatment elective rather than being an emergency condition. Laparoscopy is the gold standard, surgery recommended is salpingectomy however salpingostomy may be considered in a women with one tube who wishes to preserve fertility. Laparoscopy is preferred due to lower cost, less operating time, less blood loss, less extensive postoperative adhesions, faster recovery, and lower costs of hospitalization and rehabilitation.

[19, 20, 21]. Laparotomy is performed only if it is not possible to do laparoscopy due to some technical, logistic, or medical reasons. The level of serum β-hCG level falls markedly on the first postoperative day to less than half of its initial value [22].

Early diagnosis, identification of risk factors and conservative or surgical treatment at the right time helps in reducing the morbidity and mortality associated with ectopic pregnancies.

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SCIATIC NERVE: ANATOMY AND VARIATIONS

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INTRODUCTION

Knowledge about sciatic nerve anatomy in relation to piriformis muscle is especially important during the surgical procedures of gluteal region and back of thigh. Though normal anatomy is seen in most of the cases, variation is also not unusual. Variations in anatomy have been reported by many authors in previous studies. Apart from normal anatomy, anatomy seen in rare cases should also be known.

Sciatic nerve is the thickest nerve in the body originating from roots of lumbosacral plexus; it descends inferiorly, deep to piriformis muscle, through the gluteal region towards the lower limb. It consists of two parts, tibial part and common peroneal. These two parts are normally separate from each other, but within the same connective tissue sheath. At the superior angle of popliteal fossa they separate out as terminal branches of sciatic nerve; but it is not always true. It may divide at any position from the pelvic region to the popliteal fossa. Trifurcation of this nerve may also occur but it is rare.

ANATOMY

Sciatic nerve takes origin ventral rami of L4-S3 spinal nerves. The fibers converge to form a single nerve that passes out of the pelvis through the greater sciatic foramen below piriformis (infrapiriform area) along with pudendal nerve and vessels, inferior gluteal nerve and vessels, nerve to obturator internus, and posterior cutaneous nerve. The nerve then descends in the posterior compartment of thigh deep to the long head of the biceps femoris and terminates by dividing into its 2 terminal branches (tibial nerve (TN) & common peroneal nerve (CPN)) at the superior angle of popliteal fossa. Tibial part originates from ventral divisions of the anterior primary rami of L4-S3 and descends in the posterior compartment of leg and foot. Common Peroneal part originates from dorsal divisions of anterior primary rami of L4-S2, and reaches the lateral and anterior compartment of leg and foot. Although, this is the typical anatomical pattern, variations have been observed in relation to the location of termination, its course and relation to the piriformis.

CLINICAL CORRELATION Knowledge of sciatic nerve anatomy is required to perform surgical procedures in the region of its vicinity. Variations in its anatomy also play a crucial role during surgical procedures. The best way to learn the normal anatomy and variations is the cadaveric dissection as had been observed previously by many scientists at different times over the years.

Beaton and Anson observed the sciatic nerve in relation to Piriformis in 120 specimens during 1937 and 240 specimens during 1948 and classified them into variuos types [1]:

- Type 1: Undivided nerve below undivided muscle.
- Type 2: Divisions of nerve between and below undivided muscle.
- Type 3: Divisions above and below undivided muscle.
- Type 4: Undivided nerve between heads.
- Type 5: Divisions between and above heads.
- Type 6: Undivided nerve above undivided muscle.

Variations were also observed by many scientists during their studies. Ugrenović S et.al (2005) [2] observed normal division of sciatic nerve in 72.5% foetuses while high division in the gluteal region in 27.5% foetuses. Type 1 variation was found in 192 (96%) cases, Type 2 in 5 lower extremities (2.5% cases) and Type 3 in 3(1.5%) cases. Pokorny et.al (2006) [3] observed 91 cadavers and found Type 2 variations in 14.3%

cases, Type 3 variations in 4.4% cases and Type 4 variations in 2.2% cases while the rest followed the normal Type 1 anatomy. Natsis K et.al(2014) [4] also observed sciatic nerve variations in their study on 147 cadavers.Type 1 anatomy was observed in 275 limbs (93.6 %); in 12 limbs (4.1%) double piriformis muscle was present and the common peroneal nerve passed through it and the tibial nerve below; in 1 limb (0.3 %) CPN passed above and TN below piriformis; in 1 limb (0.3 %) both nerves passed through piriformis; and in 1 limb (0.3 %) both nerves passed superior to piriformis. They also observed non-classified anatomical variations in 4limbs (1.4%).

Gomes BA et.al (2014) [5] observed variable relationship between the sciatic nerve and piriformis muscle. Jacomo AL et.al (2014) [6] found an accessory piriform muscle in the depth of piriform muscle in a 51 year old Caucasian male cadaver. The common peroneal nerve was seen on the superficial surface of deep accessory piriformis muscle and the inferior gluteal nerve originated from the peroneal branch, both emerged from the top edge of the muscle. The tibial branch and posterior femoral cutaneous nerve emerged from the bottom of the deep accessory piriform muscle. Sulak O et.al (2014) [7] observed a variant anatomy in 2% cases in a study of 200 foetuses. Berihu BA and Debeb YG (2015) [1] in a study of 28 cadavers found variations in 14(25%) lower limbs. Out of 6 lower limbs (11 %); CPN and TN component were seen separate below the piriformis in 5 lower limbs (9 %), rejoined posterior to quadratus femoris muscle and separated again at the superior angle of popliteal fossa, whereas in 1 lower limb (2 %), the CPN component emerged above the piriformis, the TN component emerged below and they descended separately. In 1 male subject right sciatic nerve trifurcated into tibial nerve, common peroneal nerve and an unusual trunk, in the middle of the popliteal fossa.The unusual trunk branched into lateral cutaneous nerve of the calf and peroneal communicating nerve. In 1 female subject left sciatic nerve trifurcated into tibial, superficial and deep peroneal nerves at the superior angle of the popliteal fossa. In 5 lower limbs (9 %) sciatic nerve terminated into three branches: tibial, common peroneal and sural nerves. Kabacki AD et.al(2016) [8] dissected 60 aborted foetuses and observed that sciatic nerve divided atthe popliteal fossa in 99 (82.5%) lower extremities, at a level above the popliteal fossa in 19 (15.83%), and high division was observed in 2 (1.67%). Sciatic nerve relation to piriformis muscle was also assessed in 120 lower extremities, in 118 (98.3%) it coursed below the piriformis muscle undivided and in the remaining 2 (1.67%) it divided at higher levels. In one right lower extremity, tibial nerve branch followed a path under piriformis muscle and common peroneal nerve was observed passing through the piriformis nerve. Lewis S et.al (2016) [9] in a cadaveric study found normal anatomy in 90 out of 102 lower limbs (89%) and identified two distinct variations: in one the common fibular branch of the sciatic nerve passed through the piriformis (in 8.8% lower limbs examined); and second involved the common fibular branch passing over the piriformis (in 2.9%). Carnevalli FU et.al (2017) [10] observed Type 1 anatomy in 87.5% cases, Type 2 or Type 5 in 11.54% cases while Type 6 in 0.96% cases. Varenika V et.al (2017) [11] reviewed 755 consecutive scans; conventional anatomy (type I) was observed in 87%, in remaining 13% a type II pattern was seen and two type III variants were identified. These variations have been observed by scientists previously but other variations may also be present which may not have been observed till now but we have to be careful for those cases.

CLINICAL SIGNIFICANCE

It is important to have a prior knowledge about anatomical variations of sciatic nerve before a surgical intervention in the gluteal region in order to reduce the risk of injury to the nerve. A detailed anatomical study of such variations will be helpful for evaluating the pain in various test positions.

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STERNOCLEIDOMASTOID MUSCLE AND ASSOCIATED ANOMALIES: AN INSIGHT

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INTRODUCTION

Human neck is supported by 10 pairs of muscles out of which one is the sternocleidomastoid muscle. No doubt, the beauty of this muscle lies in the various complex movements of our neck performed by it and thus has always fascinated researchers to explore and investigate more. It is the major muscle responsible to maintain our neck posture and performs some major actions which have been explained in this article in a very simplified manner. It is also associated with some complex anomalies which can affect day to day lives; the symptoms, diagnosis and treatment options for conditions involving the muscle have also been discussed.

ANATOMY

The muscle appears like an oblique band crossing on the side of neck from sternoclavicular joint to the mastoid process of the skull. It divides the neck region into anterior and posterior triangles. It has 2 heads of origin; a sternal/tendinous end from the upper part of anterior surface of manubrium sterni and a clavicular/fleshy head from superior surface of medial one-third of the clavicle. The muscle is inserted on the lateral surface of mastoid process and lateral half of superior nuchal line [1, 2].

Superficially it is covered with skin, superficial fascia, deep fascia and the External Jugular Vein passes over it. Its deep relations include the Thyroid gland, Internal Jugular Vein, Carotid arteries and deep cervical lymph nodes. Motor supply to the muscle is derived from spinal part of the accessory nerve and proprioception is conveyed by ventral primary rami of C2, C3 spinal nerve. When acting unilaterally the muscle turns the chin in opposite direction (contralaterally) and ear of the same side towards the shoulder of the same side; when acting bilaterally along with longus colli it helps to flex the neck against any resistance, for example- "Lifting your head from a pillow (in this case resistance is offered by the gravity)".It also acts as an accessory muscle for forceful inspiration. It protects the underlying neurovascular bundle, branches from cervical plexus, deep cervical lymph nodes and other soft tissues present beneath it. Also, as it is a strong and thick muscle, thus suicidal or murderous attempts to cut-throat generally fail.[1][2]It receives blood supply from variable sources; upper 1/3rdfromtwo sternocleidomastoid branches of the occipital artery; middle 1/3rd from a sternocleidomastoid branch of superior thyroid artery and lower 1/3rd from the sternocleidomastoid branch of suprascapular artery [3].

CLINICAL CORRELATION

Morphological Anatomical Variations of Sternocleidomastoid Muscle:

During dissection of cadavers, sometimes an additional head of origin of the sternocleidomastoid muscle has been identified as a rare case.

The additional head has been identified to be originating from the middle part of the clavicle (Fig 1) thus forming 2 supraclavicular fossae in total [4].

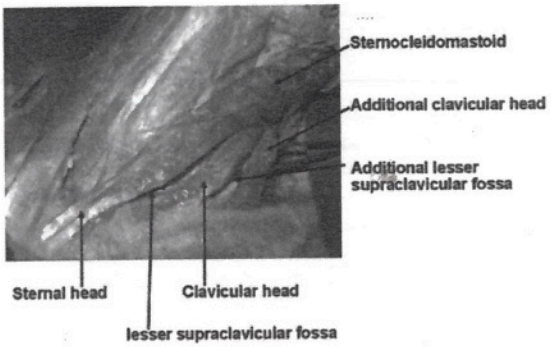

Fig. 1: Figure shows an extra head sternocleidomastoid muscle originating from the upper-middle 1/3rd of the clavicle [5]

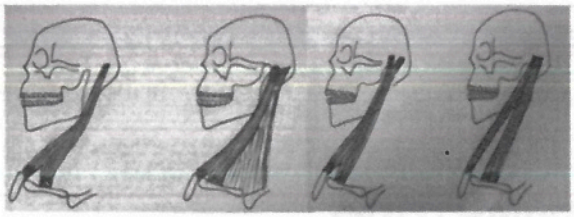

fig 2a fig 2b fig 2c fig2d

Fig 2a- sternocleidooccipital belly with absent mastoid insertion
Fig 2b- fusion of sternocleidomastoid with trapezius
Fig 2c- lateral and medial slips of sternocleidomastoid insertion on mastoid
Fig 2d- separate bellies of sternomastoid and cleidomastoid with extended lesser supraclaavicular fossa

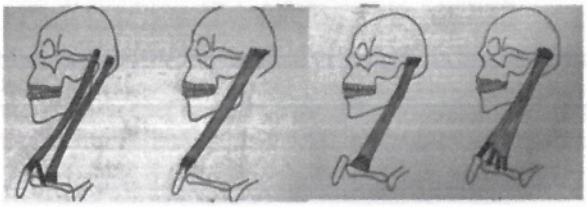

fig 2e fig.2f fig.2g fig.2h

Fig 2e- cleido occipital belly separate from sternocleidomastoid belly
Fig 2f- sternomastoid with absent clavicular head
Fig 2g- cleidomastoid with absent sternal head
Fig 2h- supernumerary clavicular heads

Fig. 2: Figure depicting various morphologic variations of sternocleidomastoid muscle [4]

CLINICAL MANIFESTATIONS

1. **Torticollis** [6]:

"Torticollis" or "Wry Neck" is a deformity involving the sternocleidomastoid muscle.

The patients present with a tilted head, chin pointing towards one shoulder and ear of the opposite side pointing towards the shoulder of the same side as of the pointing ear

It is grossly classified into 3 categories-

- Permanent (E.g. Congenital Torticollis)
- Temporary
- Spasmodic (A most common acquired form of torticollis)

Most often, spasmodic torticollis follows the pain and a reflex muscle spasm, but the patient recovers once the inflammatory process subsides.

- Congenital Torticollis: It is the most common cause of permanent torticollis.

It occurs due to the fibrosis of the sternocleidomastoid muscle of one side. Thus, the muscle fails to elongate as the child grows. Cause of fibrosis is generally a response to the ischaemic necrosis which may occur during delivery of a child having a breech presentation in the mother's womb. The cause is confirmed by a lump (Sterno-Mastoid Tumour) in the neck, probably a swollen ischaemic muscle. This lump probably disappears in a few months after its appearance leaving the fibrosed muscle behind.

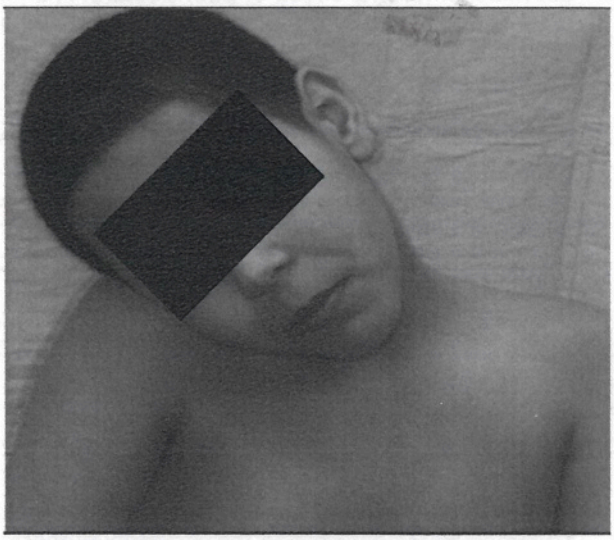

Fig. 3: Image of a 9-year-old child suffering from torticollis [7]

The child generally presents at 3-4 years of age, many times as late as puberty (may be due to late onset of prominent symptoms or less seriousness posed from the side of a guardian during the early age of the child).The head is tilted towards one side, compelling the chin topoint in the opposite side. When tried to straighten the head passively, muscle of the side where the head is tilted becomes more prominent. A radiological examination can be performed, to rule out the case of cervical scoliosis or any other underlying bone defect.

Prevention in a child with progressive torticollis can be done by passive stretching and splinting. Release of the contracted sternocleidomastoid muscle is required in older children with severe deformities. Usually, it is released from the lower end, but in severe cases, a release may be required from both attachments.

Post-surgery, the neck needs to be supported in the corrected position in a *Callot's Cast*.

2. Sternocleidomastoid Pain Syndrome

Is a chronic pain disorder involving the sternocleidomastoid muscle [8] It is characterized by presence of hyperirritable spots located in skeletal muscle (trigger points) that can be felt as a band or a nodule of muscle of harder than normal consistency. Trigger points develop as a result of injury to the muscle; caused due to sports related trauma or accident, or may be a chronic muscle overuse brought about by repetitive occupational activities, emotional stress or poor posture. A trigger point is composed of many contraction knots where individual muscle fibres contract and cannot relax. The sustained contraction compresses local blood supply, resulting in energy shortage of the area, and this metabolic crisis activates pain receptors, generating a regional pain pattern that follows a specific nerve passage. The pain patterns are therefore consistent and are well documented for various muscles, but sometimes palpation of trigger points may elicit referred pain in a different area of the body making diagnosis difficult [9,10]. Trigger points are associated with persistent pain in the region associated with restricted range of motion of the affected muscle, pain is most frequently found in the head, neck, shoulders, extremities, and lower back.

Treatment is directed at releasing trigger points. Common treatment options include manual therapy, such as massage, to release trigger points by pressure, the outcome of manual therapy is strongly dependent on the skill of the therapist. The spray and stretch technique makes use of a vapour coolant that decreases skin temperature (for pain relief) while simultaneously passively stretching the affected muscle. Other effective treatments include injecting trigger point injections with saline, local anaesthetics or steroids or a dry needling technique involving insertion of a needle without injecting any solution [11,12].

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SUPPORTS OF THE UTERUS AND UTERINE PROLAPSE: AN ANATOMICAL PERSPECTIVE

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INTRODUCTION

Uterine Prolapse is a condition experienced by a large proportion of women. Older, postmenopausal, multiparous women are highly susceptible to prolapse, but, it is not limited to the said group and might affect women of all ages. It is also important to mention that cases of congenital uterine prolapse in newborns and prolapse in nulliparous women are extremely rare. Different types of childbirth techniques present different percentages of cases of uterine prolapse.

This article studies the impact of different types of birthing methods on uterine prolapse. After reviewing multiple articles it was found out that a large proportion of the parous women experience incidences of uterine prolapse during post-menopausal stages. Several factors may result in uterine prolapse and childbirth is one of them. Women undergoing caesarian section are least susceptible to uterine prolapse and other associated disorders like pelvic floor weakness, urinary, and fecal incontinence, whereas those undergoing vaginal delivery are highly susceptible. Childbirth at home poses high risks as compared to a hospitalized setting with simple childbirth but vaginal deliveries requiring forceps and vacuum suctions are most injurious to pelvic floor strength.

ANATOMY

Location and structure of the uterus: The uterus is a component of the female reproductive system lodged between the urinary bladder anteriorly and the rectum posteriorly within the pelvic cavity [1]. It is pear-shaped and highly muscular, measures about 9cm in length, 6.5cm in width, and 3.5cm in thickness and weighs about 90g in an adult woman [2, 3]. It is divided anatomically and functionally into body and cervix. The fundus is present superior to the fallopian tubes and is the site of implantation of the embryo. The cervix is separated from the body by the internal os and opens into the vagina via the external os. It is subdivided into: the vaginal and supravaginal part. The uterus presents an angle of anteversion (tipped anteriorly concerning the axis of the vagina) and anteflexion (flexed or bent anteriorly concerning the cervix) [2]. It is a 3-layered structure; outermost is perimetrium: composed of connective tissues, middle is thick myometrium composed of muscles, and the innermost is mucous layer.

Ligaments attached to the uterus: Various ligaments are attached to the uterus, the ligament of the ovary and the round ligament are attached to posteroinferiorly and anteroinferiorly respectively. They are the remnants of ovarian gubernaculum and are the false ligaments as they don't provide support. The broad ligament is the fold of peritoneum, it keeps the uterus in its position. All the organs in the pelvis are surrounded by layers of condensed pelvic cellular tissue and these layers are called endopelvic fascia. It provides the uterus with necessary support and at the same time helps it to expand. Another important support to the uterus is the Mackenrodt's ligament (also called as cardinal ligament or transverse cervical ligament); it lies below the level of the uterine vessels and passes out in a fan-shaped manner. There are 3 parts of the Mackenrodt's ligament [2]:

i. Lateral, transverse cervical part, the (textbook Mackenrodt's ligament)
ii. Posterior uterosacral ligament
iii. Anterior pubocervical ligament (contributing towards supporting the bladder)

These 3 together form a triradiate ligament they are essentially a single unit (Fig.1)

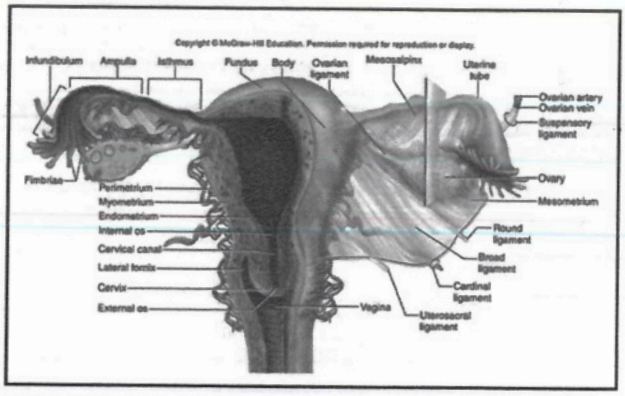

Fig. 1: Uterus and its Supports
(Source: ©McGraw Hill Education)

CLINICAL CORRELATION

Pregnancy and uterus: During pregnancy the size increases to about 35×25×20 cm, and weight increases to almost 1000g at term due to hyperplasia and hypertrophy of myometrium as well as due to an increase in the mass of elastic connective tissue. Its volume increases to about 4000mL at term. Cervix also hypertrophies and its vascularity increases. Due to this excessive increase in size and weight and due to the weight of fetus and surrounding fluids, the pressure and stretch on uterine ligaments increases making the ligaments weak and the uterus susceptible to prolapse. During the period of pregnancy, there is an increase in the size of the urogenital hiatus this also increases the risk of uterine prolapse [4].

Uterine prolapse: Prolapse is a condition in which straining causes protrusion of the vaginal wall at the vaginal orifice or in severe cases, the cervix of the uterus or the entire uterus may be extruded from the vagina. Weakness of the pelvic floor, enlarged urogenital hiatus and weakened uterine ligaments are the cause of uterine prolapse.

Classification of prolapse [2] (Fig. 2):

Anterior vaginal wall:

- Upper 2/3rd: Cystocele
- Lower 1/3rd: Urethrocele

Posterior vaginal wall:

- Upper 1/3rd: Enterocele
- Lower 2/3rd: Rectocele

Uterine Descent:

- 1°- descent of cervix in the vagina
- 2°- descent of the cervix to the introitus
- 3°- descent of the cervix outside the introitus
- Procidentia- all of the uterus outside the introitus

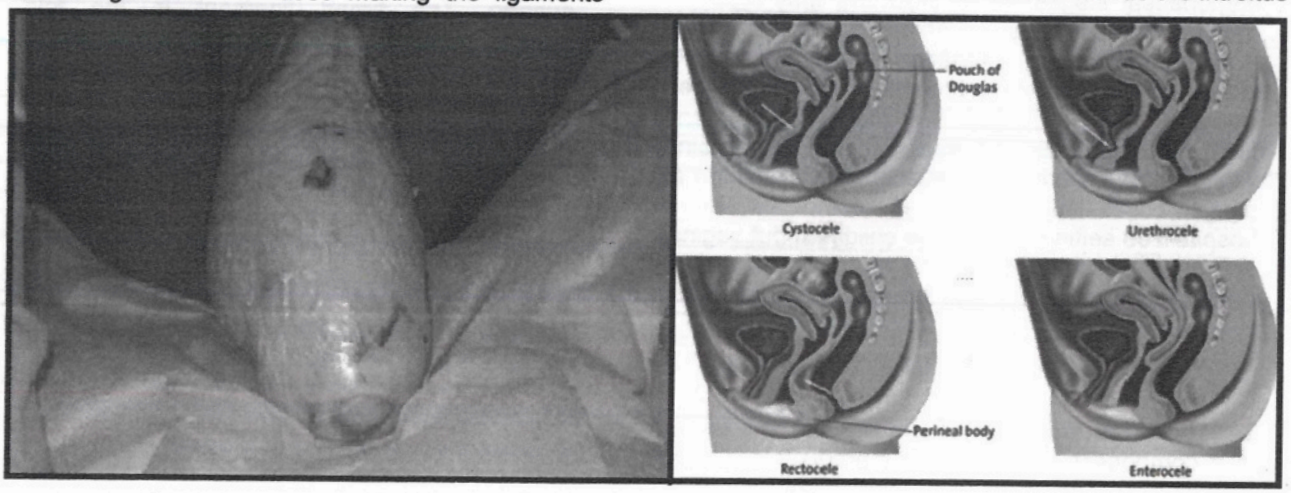

Fig. 2: Procidentia (left,) and Vaginal wall prolapse (right)

Source: a) Left (procidentia): Source: Journal of Clinical & Diagnostic Research, Year: 2009, Month: Sep Volume: 9 Issue: 9 Page: QD01-QD02 b) Right (vaginal wall prolapse): Source: Shaw's Textbook of Gynaecology ©2011, Elsevier, India

a) Left (procidentia): Source: Journal of Clinical & Diagnostic Research, Year: 2009, Month: Sep Volume: 9 Issue: 9 Page: QD01-QD02 b) Right (vaginal wall prolapse): Source: Shaw's Textbook of Gynaecology ©2011, Elsevier, India

Childbirth methods and incidences of prolapse: Childbirth is one of the most important cause of prolapse. There are 2 major methods of childbirth: the vaginal delivery and the caesarean section (C-section). Vaginal births may take place either at home or in hospitals whereas the caesarean section always takes place in hospital settings. The difference in setting whether home or hospital; influences the techniques utilized during the process and affects the causation and predisposition to prolapse.

Childbirth at home: Natural childbirth or vaginal birth without any medical interventions at home is still favoured by many cultures across the globe. In developing countries greater percentage of deliveries taking place at homes (large population; economically weak; lack of education) [5]. Majority of home deliveries are carried out by midwives who have not received a formal training but have joined the profession as they have witnessed and assisted in many births. An excessively long second stage labor in women undergoing home delivery by midwife is detrimental to the strength of the pelvic floor and these women are highly prone to prolapse as well as associated disorders [6] (Table 1).

Table 1: Comparison between women with prolonged stage 2 labor and women who underwent C-section [6]

	Women with prolonged stage 2 labor	Women who underwent a C-section
Urinary incontinence	55%	46%
Fecal incontinence	8%	13%
Positive PTT[1]	17%	6%
Stage ≥2 prolapse	22%	15%

Also, there is no provision of episiotomy to broaden the passage (prevents muscle stretch and atonicity) and reduce the duration of stage 2 of labor [2]. Lack of episiotomy may result in tearing of perineal body, women with a history of more than one spontaneous perineal laceration were significantly more likely to have prolapse [7]. According to research conducted by V.L. Handa and coworkers on 449 patients, it was found out that episiotomy significantly reduces the risk of prolapse as compared to delivery without episiotomy but with perineal lacerations. Increase in incidence also relates to the fact that many women hail from poor economic backgrounds and join work (manual labor and fieldwork) soon after giving birth which increases pressure on the uterus which already has weakened supports post-partum. A study carried out between 2009 and 2014 in south- India showed that 78.6% of all women who participated in the study and showed prolapse were manual laborers [8].

Institutionalized vaginal birth: Number of people approaching the hospitals and consequently institutionalized births is increasing due to improvement in socio-economic status and growing awareness about its benefits. A study was conducted by Zhu YC et al on 578 women; the women were divided into 4 groups: women with normal vaginal delivery, women with forceps delivery, women with cesarean section, and nullipara women. Women with forceps delivery had the highest probability of uterine prolapse (50.8% using POP-Q and 52.6% using ultrasound exam) against 11.1% using POP-Q and 8.9% using ultrasound exam in case of nullipara women [9]. Compared with spontaneous vaginal delivery, cesarean delivery was associated with significantly lower hazard for stress urinary incontinence, overactive bladder, and pelvic organ prolapse, while operative vaginal delivery was associated with a significantly higher hazard of anal incontinence and pelvic organ prolapse [10,11] (Table 2).

Table 2: Comparison between cases of Caesarean birth, spontaneous vaginal birth and operative vaginal birth [10]

	Caesarean Birth	Spontaneous Vaginal Birth	Operative Vaginal Birth
Overreactive bladder	10.4%	15.8%	24.3%
Anal incontinence	19%	22.8%	31.4%
Pelvic Organ Prolapse	5%	16.7%	30.3%

Cesarean section: C-section is a major surgery involved in giving birth, it is suggested for many reasons like prolonged labor, abnormal positioning of fetus, fetal distress, or birth defects which pose risk to either mother or child or both. Even though the cesarean section reduces the risk of prolapse considerably, yet it is not 100% assuring regarding uterine prolapse. Research by Larsson C et al who investigated 1.4 million women reported a strong and statistically significant association between cesarean section and pelvic organ prolapse [12]. C section poses risks like blood loss, organ damage, and infections to the woman undergoing the surgery. Studies have been conducted to see the effects of C-section on urinary tract injuries and gastrointestinal tract injuries where it was found that incidences of the bladder and ureteral injury were 0.3% and 0.1%, respectively whereas injury to the bowel at the time of cesarean section was exceedingly rare (0.1%) [13,14].

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7. ABHISHEK SINGH-II-S/0 S.K.S.
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9. ACHINTYA GOYAL
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11. ADITI CHAUHAN (Km)
12. ADITYA KESHARI
13. ADITYA KUMAR
14. AJAY KUMAR VERMA
15. AJAY PRATAP SINGH
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17. AKSHAR KANT
18. AKSHAY KUMAR
19. AKSHITA V1SHWANADHA (Km)
20. ALINA KHAN (Km)
21. ALOK AGRAHARI
22. ALOK RANJAN SINGH
23. ALQUAMA ISRAIL (Km)
24. AMAN GUPTA
25. AMAN KUMAR
26. AMIT KUMAR
27. AMIT YADAV
28. AMLAN JAIN
29. ANANYA GUPTA (Km)
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31. ANIL KUMAR
32. ANIRUDHA PRASAD

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8. ANUBHAV MUKHERJEE
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14. ARNAVI SINGH (Km)
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20. ASHISH VARSHNEY
21. ASHUTOSH JAISWAL
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23. ASHUTOSH MEHROTRA
24. ATIQUE ANWAR
25. AYUSH GUPTA
26. AYUSH MALIK
27. AYUSH PAUL
28. AYUSH SINGH
29. AYUSH SIROHI
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