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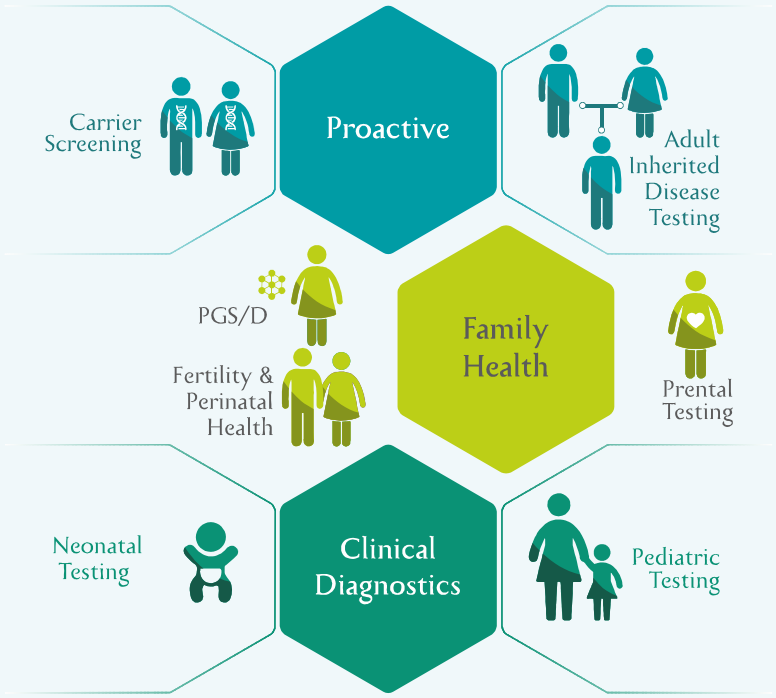

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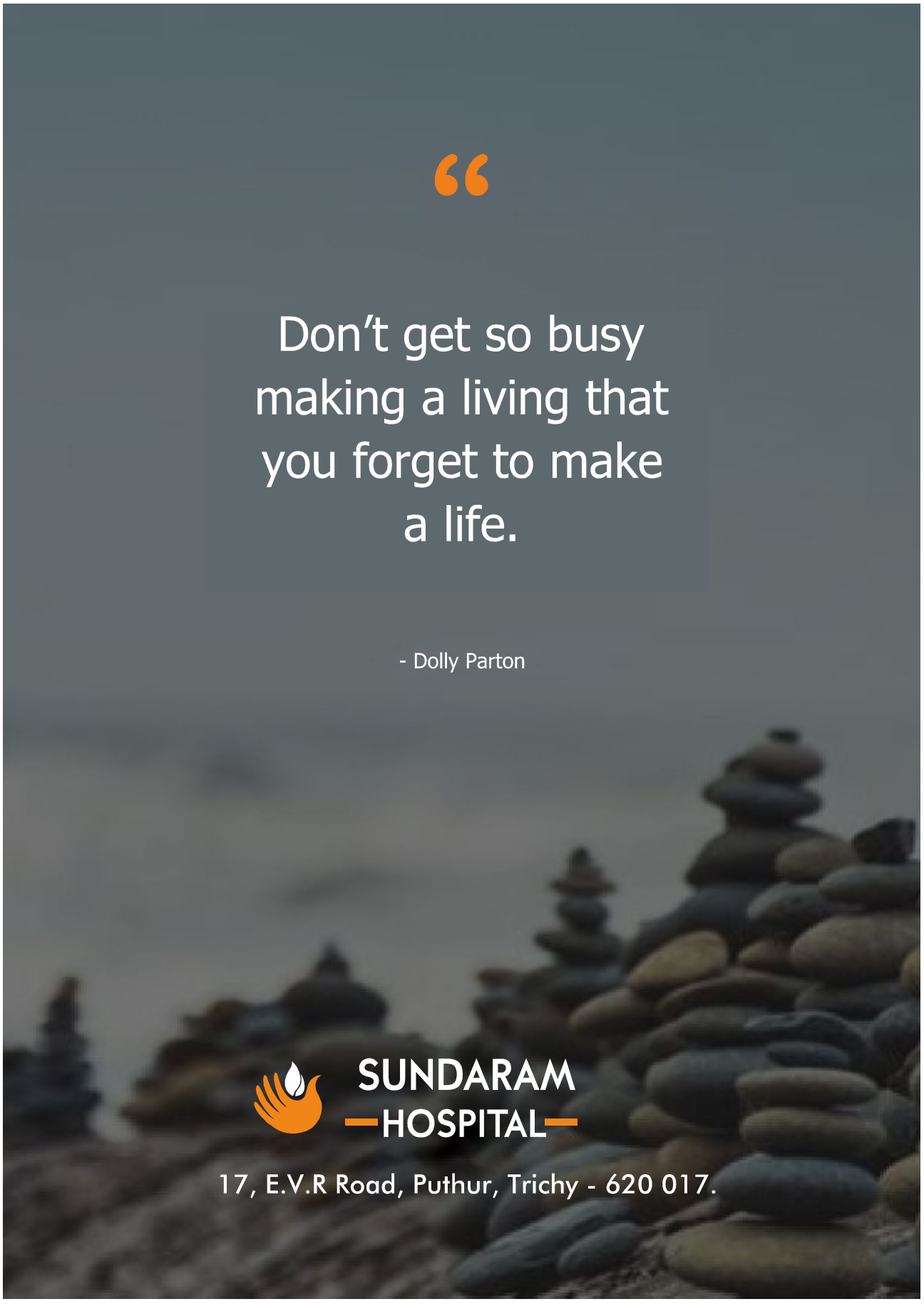
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Don’t get so busy making a living that you forget to make a life.
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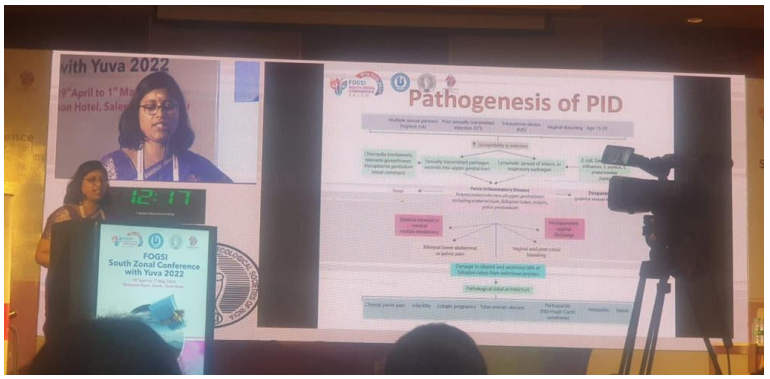

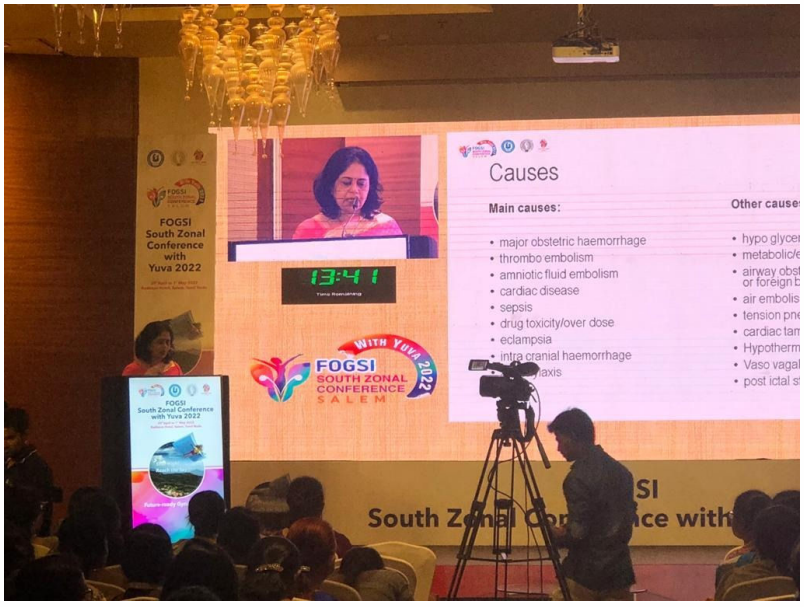

PRESIDENT'S MESSAGE

Dear friends and colleagues

It has been a difficult 2 years of COVID pandemic and all of us still sailing through it.

Our society is celebrating the 25 th year and we are a proud society of 250 members from the initial members of 50.

The journal is being released after 2 years with interesting topics and current updates in the field of obstetrits and gyneacology. I thank all of you for heartily contributing towards the journal.

The journal will be released on the day of the CME on 14 Aug 2022 and will be available as printed version.

My special thanks to the editorial board for the efforts taken to release the journal.

I extend my best wishes for a great academic program.

Dr MALATHI.G.PRASAD

SECRETARY'S MESSAGE

Aadi Peruku greetings to all. Aadi is always a joyful month where lots of celebrations take place. Lots of Batch reunions take place as most of us would have joined as medicos in this month. We also celebrate our 25th Annual CME on this auspicious month. I deem it a great honour and privilege to pen few words in our journal to be released on our 25 Annual CME. Amidst the covid pandemic we are trying our best to be back to normalacy. We also celebrate our Breast feeding week in this month. It is our duty to educate mothers on breast feeding. Theme of our Annual CME is

Update ourselves to make more women smile.
"Never stop Learning because life never stops Teaching."

I take this opportunity to congratulate the commitment of our journal team. Dr. J Prabha, Dr. Gayathri, Dr. Akila and Dr. Priyanka for all the hard work they have put in to release this journal within short notice. I thank all our senior members and authors for their contribution in releasing this Journal

Education is the most powerful weapon which you can use to change the world.
-Nelson Mandela

We as triogsians together we should share our knowledge and experience for the benefit of safe-motherhood.

Unity is strength we should be united as family and stand with each other at times of need.

A Journal's success is always its consistency and quality of articles published. I request all our dear members to actively involve and contribute articles for the upcoming issues.

Wishing all Members 3 p's
1) Physical fitness 2) Peace 3) Prosperity

"The value of your life is measured by the lives you touch with love, kindness respect and Hope."

TAKE CARE BE HAPPY

STAY SAFE ALWAYS WITH GODS BLESSINGS !!!!

Dr. LAKSHMI PRABHA

FROM THE EDITORS' DESK

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We, The Journal Committee of Trichy Obstetric and Gynecological Society-2022 are very happy and proud to publish and present "THE JOURNAL OF TRICHY OBSTETRIC AND GYNECOLOGICAL SOCIETY" for the year 2022.

"The only thing that does not change, is the need for change." As years pass by, the technique and treatments see great variations as researches make great forays in every field of medicine. The latest treatment of today becomes an obsolete one tomorrow. Hence there is a constant need for updating ourselves. Thus, publishing these journals are a great source to update ourselves. CMEs are conducted at regular intervals with the same motive. But those who are unable to attend them due to professional or personal commitments may lose to gain on them. But, these journals, when published at regular intervals, will bridge the gap as they can be read at our convenience.

This being the 25 years of our society, we have 25 articles to decorate the journal. The Almighty seems to have showered his benign benedictions upon us with such a sweet coincidence.

We thank all our members who have contributed topics for this Journal. We would like to thank the sponsors for supporting us.Special thanks to the President, Secretary, Office Bearers andbExecutive committee for giving us this opportunity.

JOURNAL COMMITTEE, 2022

TRIBUTE – Dr.VIDYA RAVI

Dr. Vidya Ravi was a renowned obstetrician and Gynecologist with a flair for teaching. She was a brilliant academician and was a fine teacher and popular among the undergraduate and postgraduate students. She was the secretary of our society in the year 2008 and became the president in the year 2020. She attained eternal peace in May 2021. While she was serving as president of our society, we wish strength and peace to her family members in this difficult situation.

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CLINICAL DIAGNOSIS AND TREATMENT OPTIONS OF A RARE ABNORMALITY – RANULAS

Dr. MALATHI.G.PRASAD MD, FRCOG [UK]
Dr. SREELAKSHMI DGO

ABSTRACT

Ranula is a type of extravasation mucocele, which can extend into the neck and the cranium which could easily cause severe intraoral swellings. Congenital Ranula may be diagnosed by prenatal ultrasound. When ultrasound was done, the report showed oropharyngeal mass possible Teratoma/ epignathus which was observed to be a ranula. Every single type lesions similar to ranulas requires different types of prognosis, as a result it is highly probable to carry out the wrong prognosis causing unsolicited problems for the patient. This case study shows the rare abnormality of a ranula and the basic methods which would be required for the purpose of treatment has been discussed.

INTRODUCTION

Ranulas is a type of an extravasation mucocele which arises from the sublingual gland, due to various possibilities like the rupturing of the main duct or rupturing of the acini after obstruction.[1] During the plunging ranula, the mucous collection can extend into the neck, causing severe intraoral swelling. Ranulas can be detected effortlessly using radiological specialised studies like Ultrasound, Computed Tomography (CT scan), or Magnetic Resonance Imaging (MRI).[2] Histologically Ranulas are very similar to epignathus.An epignathus, is a rare type of teratoma which is a tumour present at the base of the skull, either at the hard palate or the mandible.[3]These tumours usually arise due to the obstruction in the sub lingual or minor salivary glands which are present near the mandible and are usually associated as a congenital malformations.[2]

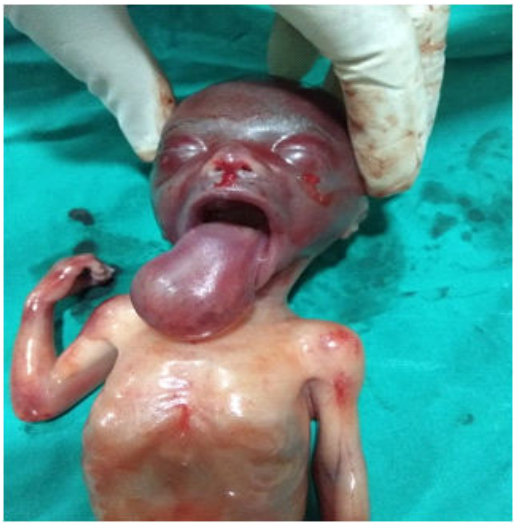


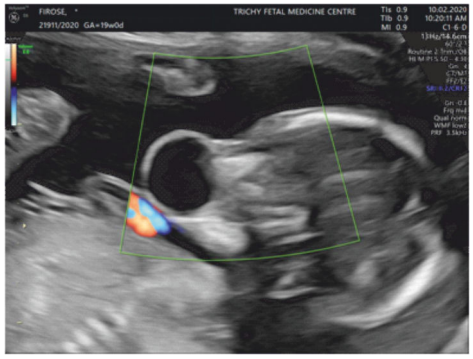
Figure 1: Photo of the foetus with a large Ranula.

Just like epignathus, ranulas are known to be benign however most of the babies with Ranulas have a poor prognosis due to late diagnosis and subsequently complicating the airway pathway and spreading to the nearby regions.[3] But with timely and early detection along with multidisciplinary healthcare options, an adequate treatment plan by surgically removing the tumour and securing the baby's airway can be planned.[4]Some major surgical procedures are marsupialisation, dissection, cryotherapy, sclerotherapy, hydro-dissection and LASER ablation.[3]

This case study describes the presence of a ranula in a fetus of a patient who has had

amenorrhea for the last 5 months.[4]The couple is married non-consanguineously and had a low risk screening report. The prenatal diagnosis of this condition has also been discussed in this case study report.

CASE REPORT



Figure; Ultrasound picture showing oropharyngeal mass present in the fetus

The name of the patient was Mrs. A, primigravida and she had been married for the past 3 year. The patient had a history of amenorrhea for the past 5 months due to this reason the patient was referred for fetal oropharyngeal mass examination using ultrasonography. Even though Mrs. A had a non-consanguineous marriage, the screening report of the patient suggested a low risk pregnancy, when the patient was subjected to ultrasound, it showed oropharyngeal mass present in the fetus which could be interpreted either as a Ranula or an epignathus teratoma. The differential diagnosis of the oropharyngeal mass included a teratoma or a tumour of neural origin. The significant structural observations were the appearance of the mandible and maxilla, the fetal breathing movements and vascularisation of the fetus,all appeared normal. However the oropharyngeal mass which was observed in the fetus measured approximately 20 x 21 mm dimensions. No other congenital malformations or polyhydramnios were observed.The patient was counselled regarding the risks and benefits of continuation and exit procedures and the option for terminating the pregnancy was also advised. The patient had opted for the termination of the pregnancy. The patient underwent spontaneous expulsion of the fetus, weighing just 500 grams with an oropharyngeal mass protruding from the foetus's posterior end of the palate, measuring at least 3cm in length. A tissue specimen of the mass was taken and its histopathological examination revealed a benign mucinous cyst, which was definitively suggestive of a ranula. The patient was given the option for fetal autopsy but she did not opt for it.

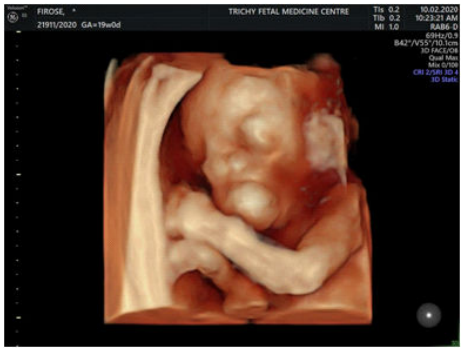


Figure 3: 3D ultrasound image of the foetus with a large ranula.

DISCUSSION

Congenital Ranula may be diagnosed by prenatal ultrasound. With Ultrasound, it is noted at the level of the floor of the mouth, a cystic, anechoic and avascular with colour Doppler mass, which sometimes moves with fetal swallowing movements, displacing the tongue upward; , in cases of large dimensions, can cause polyhydramnios.[1] Ranulas can actually be classified according to their localisation, for example simple ranulas are located on the floor of the mouth, whereas cervical ranulas are found in the paracervical area.[3] Plunging ranulas are found near the superior airway. Simple ranulas in the floor of the mouth usually occur secondary to a mucus leak following a severe disruption of the sublingual gland.[2],

histopathological evaluation could easily help in understanding the type of ranula.[4] It is essential for ranulas to be differentiated from lymphatic malformations which can be observed on ultrasound scan as solid-cystic masses with indistinct margins. These lymphatic malformations are generally present at birth itself and usually grow in proportion to the growth of the patient, even though it is benign in nature, it is possible for it to spread towards the thoracic cavity and towards the skull too. Another distinct feature of lymphatic cysts is that it can rarely drain spontaneously and are reported clinically as a permanent mass. Due to this it is highly essential to carry out differential diagnosis for epignathus.[4] There are other lymphatic teratomas which require differential diagnosis like epulis, oropharyngeal teratoma, gingival cysts (palatine), hamartomas, lymphangiomas, and thyroglossal duct cyst, congenital anomalies of the submandibular canal, heterotopic gastric cysts, and enterocystomas.[1] The teratoma appears as a solid–cystic tumour with mixed areas of hypo- and hyper echogenicity and is found in association with polyhydramnios, it had to be ruled out with the help of optimal management by ultrasound. Every single type lesions similar to ranulas requires different types of prognosis, as a result it is highly probable to carry out the wrong prognosis causing unsolicited problems for the patient. The confirmation of diagnosis for Ranula has to be performed with the help of posterior magnetic resonance, as it can help in the differentiation of types of lesions having similar hypo echogenic ultrasound patterns.

CONCLUSION

Ranulas are a type of rare type of extravasation mucocele, which is difficult to be diagnosed only with the help of ultrasound. It could also lead to a lot of complications if clinically wrong diagnosis is carried out. Thus magnetic imaging resonance is the best possible option which could help in differentiation of types of lesions having similar hypo-echogenic ultrasound patterns.

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CONQUERING THE ELUSIVE DISEASE

Dr. T. RAMANIDEVI, MD, DGO, FICS, FICOG

CONTENTS

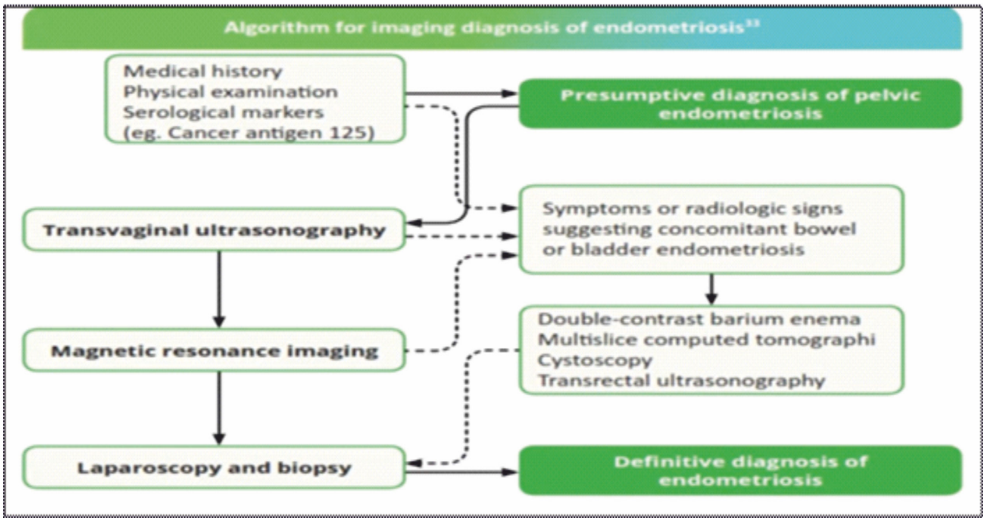
Introduction
Risk factors
Symptoms
Diagnosis
Prevention
Special situations
Creating awareness
Conclusion

Endometriosis is a mysterious disease as regards to its etiology, pathogenesis, diagnosis and treatment. It is the presence of normal endometrial tissue abnormally implanted in locations other than uterine cavity. Implants grow and invade tissue in their vicinity, causing inflammatory reaction.[1]1 in 10 women have endometriosis during their reproductive years.[2]As per recent estimates by WHO, about 196 million women suffer from Endometriosis globally. Of these, 50 million women belong to India alone!!

Endometriosis is a common disease-causing pain and infertility. Earlier occurrence leads to severe disease.25-40% of women with infertility, 75% of women with chronic pelvic pain and 40-60% of girls with severe dysmenorrhoea suffer from endometriosis.

As per American Society of Reproductive Medicine [ASRM],"Endometriosis should be viewed as a chronic inflammatory disease that requires a life-long management plan with the goal of maximizing the use of medical treatment and avoiding repeated surgical procedures"[3]. Treatment aims at alleviating the pain, preventing the recurrence and promoting the fertility.

Average diagnostic delay may vary from 6 to 8 years and the patients would have seen minimum of 7 consultants before the diagnosis is made. The natural course of the disease varies. 29% of the disease might progress or remain static and 42% might regress.[4]

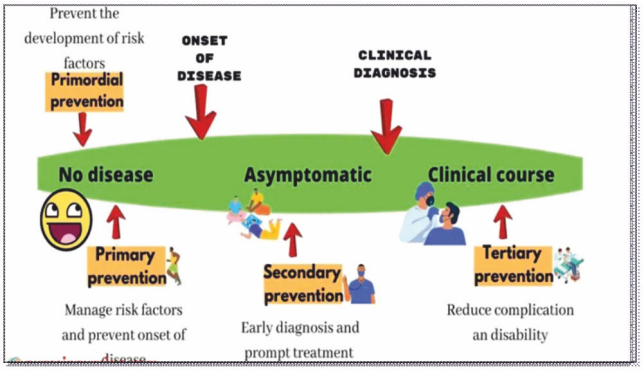


DIAGNOSIS OF ENDOMETRIOSIS

It is based on history, clinical examination, imaging modalities, biomarkers, trial of treatment and currently there is no role for laparoscopy to diagnose endometriosis. It should be only for therapeutic purpose. In 2019, there was NO PLACE for diagnostic laparoscopy when endometriosis was clinically suspected according to Prof.Chapron.[5]

IS PREVENTION POSSIBLE FOR ENDOMETRIOSIS?

Prevention could be primordial, primary, secondary or tertiary.



PRIMORDIAL PREVENTION

This aims at prevention of risk factors for endometriosis. Life style modifications play a major role in primordial prevention. Moderate exercise, avoiding dairy products, refined sugars,soy products,red meat, trans-fat,caffeine,drugs, alcohol and smoking are beneficial. Omega 3 fatty acids and fruits should be promoted.[6]

PRIMARY PREVENTION

Identification of the risk factors and regular screening, non-invasive diagnosis, empirical treatment, regular follow up of the patients and intervention at the right time are the modalities for primary prevention.

RISK FACTORS FOR ENDOMETRIOSIS

Early age of menarche <12 years,frequent and prolonged cycles, girls with low BMI, Family history of endometriosis (6.9 times higher), severe dysmenorrhoea where OCP is used as analgesic and not as contraceptive, pain interfering with daily activities, dyschezia and deep dyspareunia in sexually active girls are the risk factors for endometriosis.[7] School absenteeism due to dysmenorrhoea is an indicator for endometriosis.[8]

What other health conditions are linked to endometriosis?

Allergies, asthma, and chemical sensitivities, autoimmune diseases like multiple sclerosis, lupus, hypothyroidism, exposure to dioxin, pesticides, chronic fatigue syndrome, fibromyalgia and certain cancers like ovarian and breast cancer are linked with endometriosis.

Presence of neonatal uterine bleeding may represent a warning sign for the future development of endometriosis and increase the awareness of devastating disease in the young woman.[9]

SECONDARY PREVENTION

Includes early diagnosis, empirical treatment and treatment after confirmation.It is always better to treat the symptoms and not the disease. Common symptoms are dysmenorrhoea, dysuria, dyschezia, dyspareunia, abnormal uterine bleeding, diffuse abdominal pain, and difficulty in conception. Women with these symptoms are likely to suffer from endometriosis. Ultimately all these symptoms lead to chronic fatigue and depression.Currently patients can be treated empirically without histological diagnosis. Management includes medical,surgical or combined.Follow up and prevention of complications are essential. Secondary prevention aims at treatment in early stages and prevention of complications.

EARLY DIAGNOSIS

Apart from clinical diagnosis, bio-markers can be used for early diagnosis. Imaging can pick up endometriosis only in a slightly advanced stages. Glycoproteins (CA 125,CA 19-9), growth factors, inflammatory cytokines like IL 6 and 8,TN alpha, angiogenic factors (VEGF), oxidative stress markers,Neutrophil / Lymphocyte ratio and miRNA are the common bio-markers used for early diagnosis.

PROTEOMICS

Specific plasma biomarkers obtained during menses identifies the protein finger prints

which are markers of the disease - these can be either up or down regulated. Proteomic technologies along with genetic profiling are newer modalities of non-invasive diagnosis.

GENETIC MARKERS

Saliva based diagnosis of genetic marker may replace surgical procedure for diagnosis.

Endometrial nerve fibers in the endometrium of endometriosis patients

Unmyelinated sensory nerve fibers (using the pan-neuronal marker PGP9.5) in the functional layer of endometrium in women with endometriosis and a significantly increased nerve fiberdensity in endometrium and myometrium in women with endometriosis compared with women without endometriosis have been reported. Sensory C nerve fibers were only detected in the functional layer of endometrium of women withendometriosis and never in women without endometriosis.There was a higher density of nerve fibers stained with PGP9.5 in the basal layer of endometrium and in myometrium in women with endometriosis (mean density ± SD, 18 ± 8/mm^2, 3.3 ± 1.2/mm^2,respectively) than in women without endometriosis (mean density ± SD, 0/mm^2, and 0.9 ± 0.8/mm^2.

ENDOMETRIOSIS AND VAGINAL MICROBIOME

Endometriosis appears to be associated with an increased presence of *Proteobacteria, Enterobacteriaceae, Streptococcus suppurata and Escherichia coli* across various microbiome sites. The phylum Firmicutes and the genus Gardnerella also appear to have an association; however, this remains unclear. Laboratory and clinical studies demonstrate that there are indeed differences in the microbiome composition of hosts with or without endometriosis.[10]

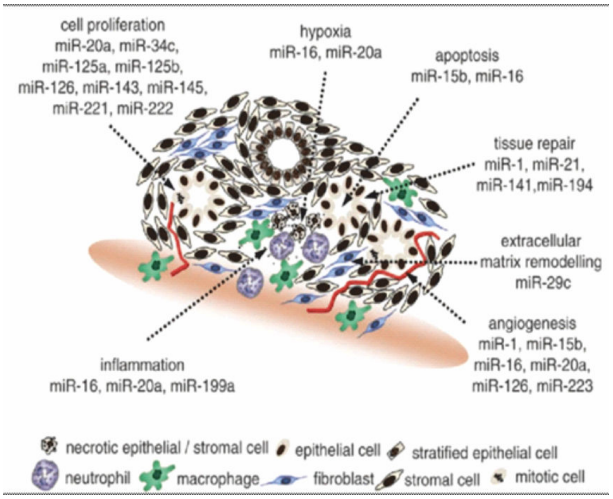
Samples from endometrium, DIE lesions and vaginal fluid were taken. DNA was extracted and the samples were analysed to identify the microbiome by DNA sequencing of the 16S rRNA marker gene which was done using next generation sequencing. Amplicon sequencing showed DIE lesions seems to have different bacterial composition, less predominant of Lactobacillus spp and with more abundant *Alishewanella*spp, *Enterococcus* spp *and Pseudomonas* sppthan the control group. There is significant increase in the presence of*Acinetobacter*spp, *Pseudomonas* spp, *Streptococcus* spp, and Enhydrobacterspp.

There is significant decrease in *Propionibacterium*spp ,*Actinomyces*spp, *and Rothia*spp in the endometriosis group compared to the control group ($p < 0.05$). These findings strongly suggest that microbiome composition is altered in the peritoneal environment in women with endometriosis.

SERUM MIRNAS

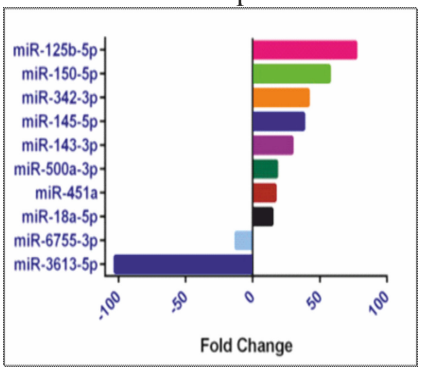
miRNA in endometriosis as a potential bio-marker

These are the numerous mi RNA involved in endometriosis



miRNAs are short nucleotide sequence of non-coding RNA involved in regulatory pathways. miRNA expression profiles are gaining appreciation as diagnostic measures in wide variety of diseases.[11]Many studies have shown differences in up-regulation and down-regulation of miRNAs in endometriosis Vs control group. There has been increased validity of panels of candidate miRNAs over a single miRNA. They can be used as a non- invasive bio-marker for diagnosis of endometriosis. Also, they can be used to find out the treatment

response to hormones. Moustafa et al., 2020 has recently shown that a set of 6 miRNAs are able to distinguish endometriosis from other gynaecological diseases, regardless of hormone treatment or phase of menstrual cycle.[12]miRNA panel used are miRNA 125b, miRNA150, miRNA342, miRNA451a, miRNA3613 and Let-7b.They resulted in 90% sensitivity and specificity. Hence patients presenting with symptoms of endometriosis if subjected to miRNA study, diagnosis can be established and treatment can be started earlier. More over various phenotypes of endometriosis are likely to have various miRNA expression.[13]



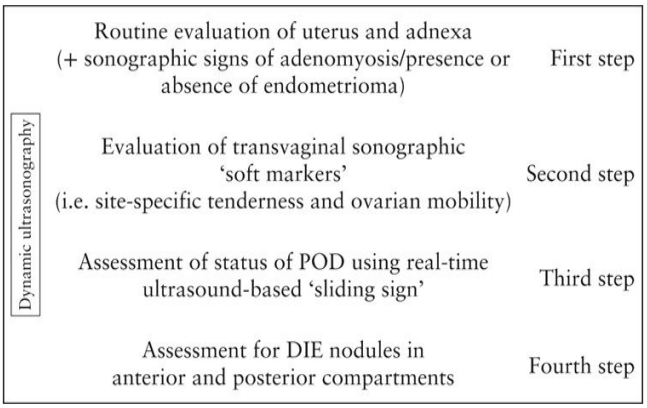
IMAGING MODALITIES

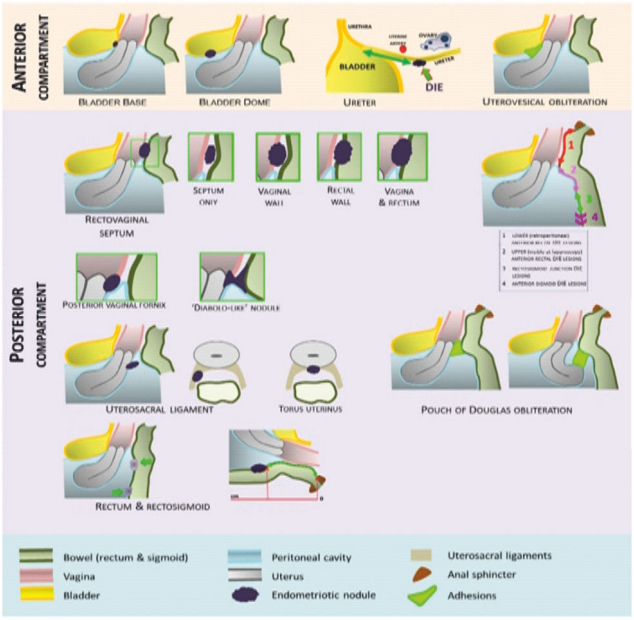
USG/CD/Elastography,CT,MRI are varying imaging modalities which can diagnose endometriosis of multiple phenotypes. Endometriomas are picked up by USG from minimum of 1 cm diameter. But superficial lesions and deep endometriosis can be diagnosed by varying signs. MRI helps in mapping deep endometriosis.

Brosens et al 2004[14] suggested in 1997 that "noninvasive techniques such as color Doppler USG and particularly MRI is more suitable for diagnosis and follow-up of endometriosis".

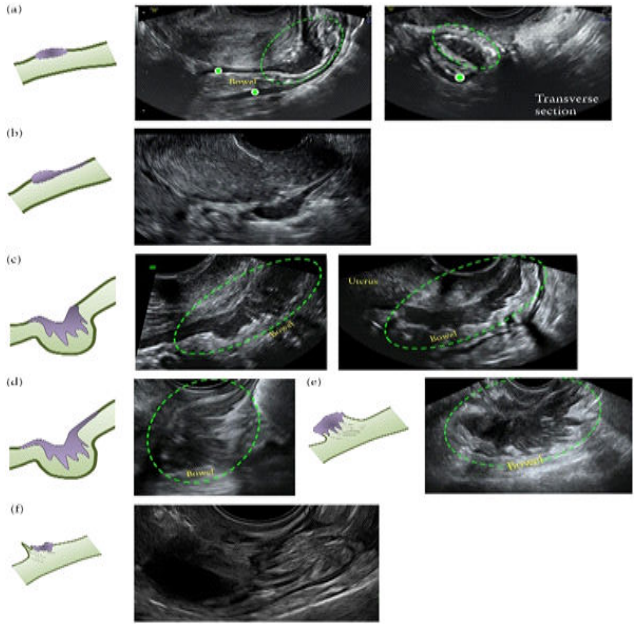
"The key paradigm shift in the management of women with endometriosis is that we now have specialists who can diagnose endometriosis on ultrasound," LukRombauts says[15]. USG is almost like a preoperative map of chocolate cyst or endometrioma but also deep endometriosis.

Four basic sonographic steps are described for examining women with clinical suspicion of deep infiltrating endometriosis (DIE) or known endometriosis





Schematic drawings and corresponding ultrasound images of bowel deep infiltrating endometriosis (DIE).[16]



(a) Absence of spikes

(b) Comet sign

(c) Moose antler sign

(d) Indian head-dress sign

(e) Pulling sleeve sign

SHORT ANO-GENITAL DISTANCE IN MRI IS A MARKER FOR ENDOMETRIOSIS

Endometriosis patient had shorter MRI-AGD especially MRI-AGD-AF. (Anterior anal verge to posterior fourchette).As, MRI-AGD was independent of r-ASRM and Enzianclassification; it can be used in diagnosing the early stage of disease.Optimal MRI-AGD-AF cut off is 20mm.

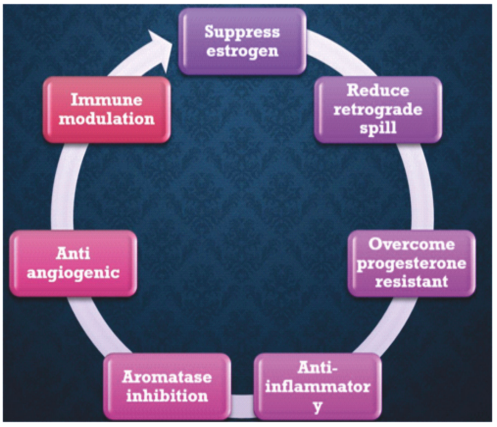
Since, MRI has sensitivity of only 42% in diagnosing stage I endometriosis – AGD measurement will be helpful for these patients.

TREATMENT WITH OR WITHOUT HISTOLOGY

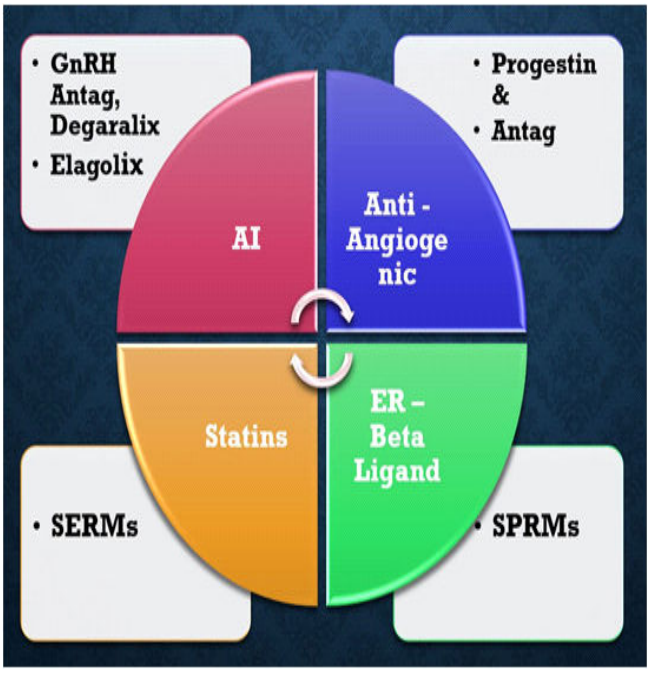
Numerous learned societies like ESHRE,SOGC,ASRM,WES and FOGSI have made recommendations that medical treatment can be prescribed for endometriosis without prior histological confirmation. Mere symptoms are enough for starting empirical treatment.

EMPIRICAL TREATMENT

Common drugs used are COCs, progestins and GnRH analogues and the concept of treatment is as follows



NEWER DRUG THERAPIES



MULLERIAN ANOMALY AND ENDOMETRIOSIS

Mullerian anomalies are associated with endometriosis.Incidence is more when there is outlet obstruction ($p<0.001$).[17]

In non-obstructive Mullerian anomaly also have endometriosis exist but with lower incidence and severity compared to women without Mullerian anomalies($p>0.05$)[18].

Girls with early onset dysmenorrhea have to be investigated for Mullerian anomaly and endometriosis. USG /MRI are the mode of diagnosis and treatment is by laparoscopy.

TERTIARY PREVENTION

It includes prevention and treatment of complications like recurrence and pain.

RISK FACTORS FOR RECURRENCE

Low Risk	High Risk
r AFS score < 70	r AFS score > 70
Pregnancy	Bilateral mass
OCP/progestins	Suboptimal surgery
Endometrial ablation	No post op medical management
Unilateral lesions	Young age
Complete surgery	Family history
Post op medication for a long period	H/O OI drugs for IUI and not IVF
BMI<23	

INCIDENCE OF RECURRENCE

Reappearance of pain after one year of surgery is 45%. Reappearance of the disease by USG or clinical examination is 9-15%.After conservative surgery and 6 months of medical treatment,1 year later 26% had pain recurrence and 8% had detectable disease. To prevent recurrence,one should aim at radical clearance of all endometriotic lesions. However smaller atypical lesions are overlooked, leading to persistence of the disease. It is ideal to do the surgery during follicular phase.Drainage of endometriomas (USG or laparoscopy guided) has 80-100% recurrence within 6 months.

Cyst drainage with cyst wall destruction had 3 times more recurrence than cystectomy (18.4 vs 6.4).Resection of recto vaginal nodule gives good pain relief in DIE. Radical surgery is mandatory to prevent recurrence, in patients who have completed family.The reported recurrence rate was high, estimated as 21.5% at 2 years and 40-50% at 5years.[19]

During endometriosis surgery, extensive procedure may reduce the ovarian reserve and incomplete surgery may lead to recurrence.

Post-operative medical suppression is needed.Medical treatment increases the apoptotic index, decreases the proliferative activity of the cells and estrogen biosynthesis by the ovary. Biosynthesis of estrogen in the peripheral tissue and endometriotic implants which is controlled by aromatase is not inhibited by routine drugs. But aromatase activity is high in endometrium of endometriotic patients, endometriotic lesions, fat, bone and adrenal tissue.Hence aromatase inhibitors are used.

Post-operative medical management should be instituted to minimize risk of recurrence. Hence it should be continued for a long time which extends beyond pain free period in severe disease after conservative surgery.

HOW LONG TO FOLLOW UP?

Data from recent large case series have documented cumulative rate of recurrences as high as 30-40% at the end of 2-3 years follow up. 2 years after surgery might be the minimum follow-up period. Many are lost to follow-up if the timing is prolonged beyond 2-3 years. Ideal follow-up is life-long or atleast upto menopause.[20]

RECURRENT ENDOMETRIOMA AND PAIN

Recommendation:

Surgery+ Long term suppression

- ✓ Only suppression is not effective
- ✓ Definitive surgery—based on patient's requirements
- ✓ LUNA—Not effective for pain
- ✓ PSN – Effective but creates new long-term problems
- ✓ If there is unilateral endometrioma, salpingo - oophorectomy followed by LNG-IUS gives excellent relief
- ✓ Hysterectomy with/without bilateral salpingo-oophorectomy if family completed or persistence of severe pain. If ovaries are left behind the incidence of recurrence and redo surgeries are high.[21]

AWARENESS ABOUT ENDOMETRIOSIS

At present, there is no known way to prevent endometriosis.Enhanced awareness, followed by yearly diagnosis and management may slow or halt the natural progression of the disease.Reduce the long-term burden of its symptoms, including possibly the risk of central nervous system pain sensitization. Endometriosis has significant social, public health and economic implications. It can decrease quality of life due to severe pain, fatigue, depression, anxiety, school absenteeism,work capacity and infertility. Dyspareunia affects sexual health and marital relationship.

Addressing endometriosis will improve the sexual and reproductive health, quality of life and overall well-being.But currently there is no cure; treatment is usually aimed at controlling symptoms.Access to early diagnosis and effective treatment of endometriosis is important, but it is limited in many settings, including in low- and middle-income countries.There is a need for more research and awareness around the world to ensure effective prevention, early diagnosis, and improved management of the disease.

CONCLUSION

Endometriosis is an elusive disease with variable etiopathogenesis. Its starts from womb and ends in tomb of a women's life. Major components of endometriosis are pain and infertility. As per the advice of ASRM classification endometriosis is a chronic inflammatory disease which needs long term medical management. Surgery should be minimized. The prevention of endometriosis starts from primordial, primary, secondary and tertiary prevention. Patients who are high risk for endometriosis should undergo primordial and primary prevention even before the disease manifest. Young women with symptoms of endometriosis associated pelvic pain should be started on empirical therapy with progestins which comes under secondary prevention. Non-invasive diagnosis of endometriosis should be planned for these women and treatment should be started. Whenever there is presence of visible lesions with pain, medical management should be the first line except in women who want pregnancy. Other patients needing treatment only for pain can be offered medical management, failing which definitive surgical management can be offered. Life style modifications including exercise, diet should be advised. Proper long term post-operative suppression with medical management should be aimed at. Awareness program regarding endometriosis and periodic follow upfor high risk patients should be advised. Ultimately, we cannot conquer endometriosis but control the symptoms, reduce recurrence and promote fertility.

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DENGUE FEVER IN PREGNANCY – A CASE REPORT

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ABSTRACT

Background : Dengue fever is prevalent in the world.40% of the world's population live in dengue prone zone.

Case Presentation : A 25 year old Primigravida presented with history of febrile illness associated with thrombocytopenia. The physical examination, laboratory investigations as well as serology confirmed dengue fever. The patient was under conservative management in spite of thrombocytopenia and elevated liver enzymes. Patient continued pregnancy till term without any complications and delivered a healthy baby by LSCS.

Conclusion: Dengue fever should be suspected in pregnant women when presented with fever, thrombocytopenia and elevated liver enzymes. Conservative management should be done in case of acute dengue fever unless there are any complications.

INTRODUCTION

Dengue is a mosquito-borne arbo virus infection and ranks as the most important, rapidly emerging disease in recent years and is endemic in all continents. WHO estimate indicates that 390 million dengue infections occur every year of which 96 million manifest clinically. [1] It is associated with maternal and fetal mortality. We reported a case of Dengue fever with thrombocytopenia during the third trimester of pregnancy with maternal and neonatal survival.

CASE REPORT

A 25 year old Primigravida with 35 weeks of gestation was admitted with history of fever and thrombocytopenia. She had a history of high grade fever for 2 days, 1 week before admission. She gave history of neighbours treated for dengue fever recently. She was referred to our hospital with platelet count 30,000 and no bleeding manifestations.

On admission, patient had Temperature-98.5 F with no signs of dehydration, not icteric, no pallor. Patient had no bleeding manifestations and on catheterization was found to have frank hematuria (figure 1, yellow arrow). Her Blood Pressure was 100/70mmhg, Pulse rate 82/min and SpO2 98% room air. She had normal breathing sound and heart sounds. The fundal height was corresponding to 34 weeks of gestation and FHR 138/min.

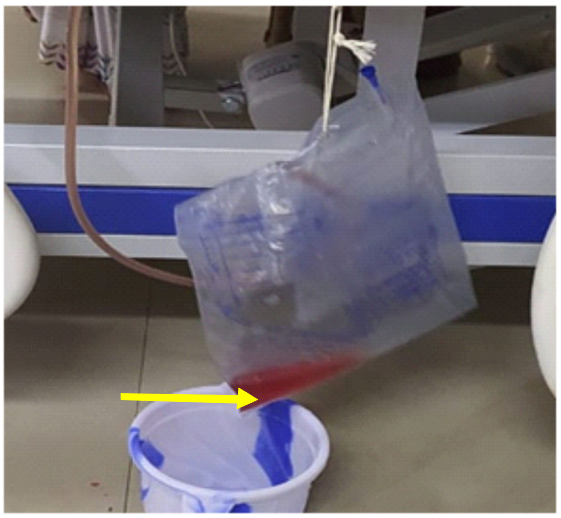

Figure 1

Figure 2

Laboratory analysis on admission showed: Haemoglobin 11.6g/dl, Hematocrit 35.3%, WBC 9700/uL, platelet 30,000/uL. SGOT 110IU/L, SGPT 52 IU/L . Expert USG bedside showed IVC diameter of 18mm and Gall bladder was normal. Dengue serology was sent. We had suspicion of dengue fever and HELLP. Patient had normal BP recording throughout the Antenatal checkup and during hospital stay so it was more likely to be Dengue fever. Since she had history of dengue positive patients in neighbourhood, her BP was normal , we treated her in favour of Dengue fever till serology reports came. As per physician opinion and haematologist opinion, She was treated 4 unit of platelet concentrate transfusions and packed cell transfusion along with intravenous fluid replacement. After about 6 hours, She was diagnosed to have Dengue fever with Dengue IgM positive. She had been under close observation for bleeding manifestations and vital signs monitoring.

Twenty four hours later patient had stable vitals and good urine output, With haemoglobin 10.1g/dl, Hematocrit 31.5%, WBC 8500/uL, platelet 56000/uL. SGOT 35IU/L, SGPT 20 IU/L, in between patient had hematuria. On third day patient was gradually recovering and had haemoglobin 11.6 , Hematocrit 35.2%, Platelet count of 103000/ul. She then had clear urine. (figure 2, black arrow).On sixth day , patient got recovered and her haemoglobin was 12.1g/dl., Hematocrit was 35.4% and platelet was 2.24lakhs. Patient was in Antenatal ward for fetal heart rate monitoring and vitals monitoring as she had risk of preterm labour due to the Dengue fever.

At 38 weeks of gestation, patient had complaints of decreased perception of fetal movement and her NST was non reactive. She was taken up for emergency LSCS in view of non reactive NST, she delivered an alive boy baby of birth weight 2.48kg with APGAR score of 8 and 9 at 1 and 5 mintues respectively. No abnormalities in the new born were detected. Intra operative and post operative period was uneventful. Patient was discharged on 5th post operative day.

DISCUSSION

Dengue is been regarded as the most important arthropod transmitted human viral disease. There are four dengue virus serotypes which are designated as DEN V1, DEN V2,DEN V3 and DEN V4. Most of the patients with Dengue remain asymptomatic. Others after an incubation period of 4-7 days develop a febrile illness like manifestations. They are grouped in 'Dengue Syndromes' that includes undifferentiated fever, Dengue fever (DF), Dengue hemorrhagic fever (DHF) and Expanded Dengue syndrome [1]. 40% of the world's population live in Dengue prone zone. WHO estimates atleast 100 million infections occur every year including 500,000 DHF cases and nearly 22000 deaths.[2] Dengue in pregnancy requires early diagnosis and treatment. Early diagnosis is difficult by the ambiguity of clinical findings and physiological changes in pregnancy [3]. Thrombocytopenia is observed in some patients with DF. A sudden drop in platelet count to below 1,00,000 usually occurs before the

onset of shock or subsidence of fever.[1]. The presentations of Dengue might be confused with other obstetric complications, such as HELLP syndrome (hemolysis, elevated liver enzymes and low platelet count) [3]. **In this patient we had diagnosed Dengue fever with thrombocytopenia before onset of shock and had treated her with Intravenous Fluid replacement and Platelet transfusion.** Patient had IVC diameter of 18mm and thrombocytopenia, hence she was treated with adequate intravenous fluid replacement. Most cases of dengue fever during pregnancy had favourable outcomes for the mother and neonate; thus patients could continue the pregnancy until late term or full term period with adequate fluid replacement.[4]. In our patient, patient gradually recovered and hydration was maintained, she did not go for any complications and hence she was allowed to continue the pregnancy till term. Patients with dengue fever with thrombocytopenia are at high risk of bleeding during delivery[5]. Dengue infection in pregnancy may increase the risk of preterm birth by 10.5% and low birth weight infants 13.4%[6] .Thus, Close monitoring required to prevent the risk of bleeding and preterm delivery.

CONCLUSION

Dengue is an arboviral infection transmitted by Aedes mosquito. The disease can affect anyone but pregnant patient are at risk. The gestation and the phase of dengue are important factors in determining the management. Dengue related thrombocytopenia can increase the risk of bleeding antenatally or postnatally and therefore leads to higher maternal mortality rate. Normal physiological changes in pregnancy make the diagnosis and assessment of plasma leakage difficult. Therefore, baseline parameters should be noted as early as possible and subsequent management should be planned according to that. The dengue fever could be prevented by Integrated vector management which involves larval source reduction, using mosquito net, coils, aerosols, repellents, etc at household level. Avoiding stagnation of water, cleaning and covering of water storages, promoting personal protection measures are some basic actions need to be taken to prevent the Dangerous Dengue fever.

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THE PREGNANCY' DISEASE

Dr. ABIRAMI KARPAGAM MD(OG) DRM

'Congratulations! You are pregnant ' The most desirable and delightful words any woman would love to hear . But are they really so much delightful for the mother ? A big NO. The most delightful moments are becoming the most disastrous moments for many of the pregnant women during their journey of carrying their child nowadays. In other words Pregnancy has become a disease!!!!

Gone are the days of grandmothers era. Welcome to the Google era !!! Gynaecologists may clear out the basic doubts of a pregnant woman. What if the whole of 40 weeks of pregnancy is loaded with doubts and non beliefs and intolerance? Not long ago just before Google era some 15 yrs ago, Pregnancy was a physiological process. Pregnant mothers took care of themselves, their in laws, and run their family without much difficulty. Pregnant women of 21st century is afraid of walking !!!!!! They are fond of eating only Avacadoes and strawberries than Indian seasonal fruits !!!!!!! They become angry often and demand more from their parents, husbands, doctors, by and large from the society just because they are pregnant. !!! Many at times pregnancy is a nightmare .!!

Where are we heading ? Where did it go wrong? Is it the belief system ? Grandparents words have become outdated and Parents words are not considered as they don't know much about the pregnancy disease. The only left out options are the obstetricians and The mastermind Google!!! No one in this world had become a doctor reading Google !!! So One must not ignore the doctor's whole lot of experience on medical education.

Pregnancy is a beautiful journey and a sense of completion of a womanhood. “What's the food that I must avoid” is the commonest question every obstetrician would confront everyday in their OPD. The doctor would love to say that the avoidable factors are Anxiety, pessimism, intolerance, ignorance. But rather she would say all home made food can be taken.

“Give the pupils something to do, not something to learn; and the doing is of such a nature as to demand thinking ; learning naturally results.” Those are the words by John Dewey,an American philosopher ,psychologist and educational reformer on education.

Education is learning the result on doing something not just only reading . It's also about spreading the knowledge what one have acquired by their experience . pregnancy is a bliss not a disease ! There's a science behind every practice in Indian culture.let's get educated and educate others and at the same time let's not neglect our ancestors words and their experience on not only pregnancy but also on life.

SOLID OVARIAN TUMOUR IN POST-MENOPAUSAL WOMEN

Dr. LAKSHMI PRABHA MBBS., DGO., DNB. (OG).,

71 year old unmarried women admitted with difficulty in passing urine and fullness of abdomen, attained Menopause 20 years. Uneventful Peri menopausal state. H/O Mass descending P/V 1 year. No H/O Difficulty in defecation. H/O Recurrent urine retention. Known case of HTN on treatment for 20years.

O/E Pt well, vitals stable, BP 140/90mm/hg, HR-84/mt, RR 20/mt. SpO2 :97% , Wt :66kg, CVS S1, S2, RS – BAE+.

P/A - soft, firm mass about 12 x12cm felt in right lower Abdomen

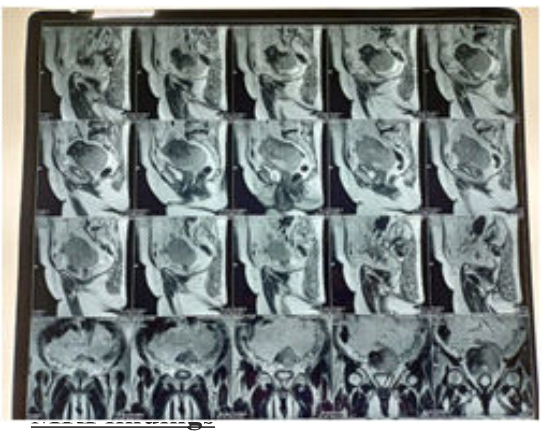
L/E II degree UV Prolapse

cervix healthy

P/V, UT. RV Atrophic

All pre op Investigation with urologist opinion obtained to rule out other causes of urinary retention. All Parameters were normal. CA 125 was 10.

MRI SCAN



MRI FINDINGS

A large well defined heterogeneous SOFT TISSUE MASS LESION seen in the pelvis.

- It measures about 11x9x7.5cms.

- Internal necrosis seen.

- No calcifications/fat components.

- Mass seen proximal to the uterus and abutting the fundus.

RIGHT OVARY: Not separately visualized.

- Possibly RIGHT OVARIAN MASS

SUGGESTED: HPE Correlation

LEFT OVARY: Atrophic.

Minimal free fluid in the pelvis.

LIVER & ADRENALS: No metastasis.

Bowel loops appears normal. No bowel wall thickening/bowel mass/bowel dilation.

No pelvic or para-aortic lymphadenopathy.

No ascites/pleural effusion.

Patient posted for TAH with BSO

PER OP FINDINGS:

Under SA abdomen opened by SPT incision

- Right side ovarian tumor about 12X12cm firm mass (+),

- Right adnexectomy done

- Left tube and ovaries Normal

- Uterus atropied, infra vaginal elongation of cervix (+)

- TAH with BSO done in the usual way by clamping, cutting and ligating the Bialteral infundibulo pelvic ligament. Round ligament, Uterine arteries and Mckendrots ligament. Vault closed with vicryl. Skin closed with 3-0 Monocryl. Hemostasis achieved.

HPE:

IMPRESSION

Uterine body: Proliferative endometrium; Cervix: Chronic hyperplastic cervicitis; No evidence of atypia. Right ovarian mass: Benign Fibro Thecoma; No evidence of malignancy; Left ovary & Tubes - Normal histology.

DISCUSSION

A fibroma is a benign stromal tumor composed of spindle fibroblastic cells. Ovarian fibroma is the most common benign solid ovarian tumor accounting for 4% of all ovarian neoplasms.Thecomas are neoplasm of theca cells.Thecomas account for about 1% of ovarian tumors. Ovarian fibrothecomas are composed of an admixture of fibrous and thecomatous elements. Histologically, these tumors are characterized by the presence of spindle, oval, or round cells forming various amount of collagen and also contain a smaller proportion of theca cells. However, there is no universal agreement on which neoplasms should be classified as a fibrothecoma rather than either a fibroma or thecoma.

Fibromas occur most frequently in women in their 50s during perimenopause and postmenopause. They are not hormonally active in most cases. Most of them are unilateral; however, bilateral cases may occur, especially in patients with Gorlin syndrome. In this syndrome, fibromas tend to occur at a younger age, often in children. Like fibromas, thecomas are usually unilateral and occur most commonly in postmenopausal women.

Fibromas/fibrothecomas most commonly present due to mass effect causing compression on different organs. In some cases, ovarian fibromas can be part of Meigs' syndrome, a triad of ovarian fibroma, ascites, and pleural effusion. Torsion occurs in 8% of the patients. Patients may also present with endocrine manifestations such as estrogenic or less commonly androgenic stimulation related to hormonally active thecoma elements. Serum CA-125 levels are usually found in normal range in ovarian fibroma/fibrothecoma. However, elevated levels have been reported in patients presented with Meigs' syndrome.

The modality of treatment could be tumor excision alone, uni - or bilateral salpingo oophorectomy with or without hysterectomy depending on the patient status and the aggressiveness of the tumor.

Differential diagnosis includes fibrosarcoma or recurrent STUMP. Smooth muscle tumours of uncertain malignant potential

A rare case of Solid Tumour in post-Menopausal lady Turning Benign.

DIAGNOSIS AND MANAGEMENT OF CERVICAL ECTOPIC PREGNANCY

Dr. VICTORIA JOHNSTON M.D. (OG)

INTRODUCTION

Cervical ectopic pregnancy (CEP) is a rare condition with an incidence of less than 0.1% of all ectopic pregnancies. It is associated with high maternal morbidity and mortality as patients present with unexpected life-threatening haemorrhage due to, erosion of cervical blood vessels. Timely intervention is required to preserve fertility and avoid the need for a hysterectomy.

Three cases of CEP are reported and the challenges in the diagnosis and management are discussed.

CASE REPORT I

A 25 year old primi gravida, presented with history of 7 weeks amenorrhea, mild pain abdomen and giddiness. Her urine pregnancy test was positive. She was married for 3 years.

Past relevant surgical history was a Diagnostic hystero laparoscopy done outside 2 yrs back elsewhere,followed by a hysteroscopy with septal resection done at our centre 1 ½ yrs back.

On examination, her vitals were stable. Per abdomen there was no guarding or tenderness. On per vaginal examination, uterus was anteverted and normal size, Cervix was bulky. Os was closed. There was no bleeding. On ultrasound, there was a viable cervical ectopic pregnancy with a CRL corresponding to 6-7wks pregnancy.

We gave her Inj. Methotrexate 50 mg IM on Day 1,4,7,10

Her βHCG was 4663, 4570, 7630, 5046, 5125m IU / ml on Day 1,3,5,6,9

On day 5, the TVUS showed a continuing cervical pregnancy.

On day 11, the FH was absent.

Hence, we proceeded with dilatation and evacuation of POC from the cervix with ovum forceps. Check USG showed empty uterine cavity & cervix. Bleeding was normal.

Hence, the patient was managed conservatively successfully & on follow up she had a normal vaginal delivery of a healthy infant 2yrs later.

CASE REPORT II

A 30yr old, G2 A1 presented with 44days amenorrhoea & a positive pregnancy test. She was married since 6yrs. She was being treated for secondary infertility. She underwent 3 IUIs in the last 6 months. Her vitals were stable. Per abdomen was soft, on PV the uterus was bulky, with a bulky cervix with minimal? blood clots protruding out through the cervix.

TVUS revealed a cervical pregnancy with an irregular gestational sac, MSD 15mm predominantly in the cervix and an empty uterus with thickened endometrium.

The sac contained a fetus with a CRL of 4mm corresponding to 6-7wks gestation with no cardiac activity.

Her serum βHCG was 23,296 m IU/ml.

A single dose of Inj: Methotrexate 50mg IM was given on the same day.

On day 3, we proceeded with evacuation of POC from the cervix. Blood clots were evacuated. Products sent for HPE. Bleeding was within normal limits.

Hence, by early diagnosis & conservative management her fertility was preserved.

CASE REPORT III

A 30 yrold G5 P2 L2 A2, presented with H/O 4months amenorrhea. She didnot get her normal periods after her last medical abortion in our centre as an outpatient 2 months back.She had irregular spotting and mild abdominal pain. She had not come for the follow up scan following a course of Tab. Mifegest& Misoprostol.

Her significant obstetric & surgical history was two C.Sections & a medical abortion 4yrs back. Her two children were 10yrs& 7 yrs old, alive & healthy.

On examination, vitals were stable. Per abdomen was soft, with 2 transverse scars.

On TVUS, there was a large irregular empty gestational sac with MSD 39mm low placed in the cervico isthmic region of the uterus. With a preliminary diagnosis of an inevitable abortion we posted her for dilatation & evacuation with laparoscopic sterilization.

At surgery, when evacuation of POC was being done there was torrential haemorrhage which did not decrease with tranexamic acid and other measures. Fresh bleeding continued. On TVUS, we found a spurting blood vessel in the dilated cervical canal.

Realizing, that it had been misdiagnosed as an IUP we were deciding whether to proceed with hysterectomy but, as a conservative alternative (as patient was only 30yrs old) we proceeded with Cervical tamponade using Foley's catheter with bulb inflated with 30ml normal saline and tight vaginal packing. Laparoscopic sterilization was done.The bleeding stopped and the patient was stable.

We transfused 1 unit of packed cells. βHCG next day was 114 m IU /ml.

On discharge, there was a 3.3 x 2.4cm organized blood clot in the cervical canal. 15days later the blood clot had resolved.

Hence, we managed the patient conservatively with cervical balloon tamponade successfully. We have to be wary of misdiagnosis of CEP as IUP.

DISCUSSION

The reported incidence of Cervical Ectopic pregnancy is 1 in 1,000–18,000 pregnancies [3]. The etiology of CEP is not fully understood but, reported risk factors for CEP include history of pelvic inflammatory disease, leiomyoma, smoking, previous pelvic surgery, previous ectopic pregnancy, intrauterine device use, congenital anatomic uterine anomalies, chronic endometritis, endometrial damage due to prior dilatation and curettage, previous cesarean delivery, previous uterine or cervical surgery, in vitro fertilization, and diethylstilbestrol exposure [2].

Paalman & McElin in 1959 proposed, 5 clinically practical signs of cervical ectopic pregnancy (1) uterine bleeding without cramping pain after a period of amenorrhea, (2) softened and disproportionately enlarged cervix equal to or larger than the corporal portion of the uterus (an hourglass-shaped uterus), (3) products of conception entirely confined within, and firmly attached to, the endocervix, (4) a snug internal os, and (5) a partially opened external os [2]. The diagnosis of CEP is established by transabdominal and/or transvaginal ultrasound. Sonographic diagnostic criteria are (1) empty

uterine cavity or thickened endometrium, (2) distended and/or enlarged cervix, (3) gestational sac or placental tissue below the level of the internal os, (4) negative "sliding organs sign", and (5) high peri trophoblastic vascularity on Doppler examination (peak velocity > 20 cm/s, pulsatility index < 1.0) [4].

CEP is traditionally considered as high risk for haemorrhage and has historically been treated with hysterectomy, leading to loss of fertility [5]. With improvements in ultrasound, early diagnosis of CEP is possible, allowing for the opportunity to use conservative medical management and interventional measures rather than surgical management [3]. Medical management options include systemic or local injection of methotrexate, KCl, local vasopressin injection, local or systemic prostaglandin, systemic mifepristone. [2]. The factors that favour conservative medical management are early diagnosis preferably before 12 weeks, low beta-hCG levels, and absence of cardiac activity. [2, 6].Arrest of bleeding is by cervical balloon tamponade with Foley balloon catheter, cervical circlage,hysteroscopic local endocervical resection, vaginal ligation of cervical arteries ,uterine artery ligation, internal iliac artery ligation and angiographic embolization of cervical, uterine or internal iliac arteries ,However, haemodynamically unstable patients with intractable haemorrhage or those who fail medical and conservative surgical management require hysterectomy.

In recent times, in vitro fertilization and other assisted reproductive technique have been reported to be associated with increased risk of cervical pregnancy and the etiology is attributed to the rapid transport of fertilized ovum into the endocervical canal because of, an unreceptive endometrium. According to one review, the incidence of cervical pregnancy is 0.1% among in vitro fertilization pregnancies. Conservative surgical methods can be employed, where future fertility is required.

CONCLUSION

Although cervical pregnancy is rare, increased number of cases are being reported because of risk factors like high caesarean section rate and increased use of assisted reproductive technique for management of infertility. The success of conservative treatment depends on the timely and prompt diagnosis by early ultrasound, which can reduce the chances of severe life threatening haemorrhage necessitating hysterectomy or blood transfusion.

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A RARE CASE REPORT – TYPHUS FEVER CAUSING INTRAOPERATIVE UNEXPLAINED TACHYCARDIA AND POSTOPERATIVE FEBRILE ILLNESS.

Dr. DEVAKHI CHINNARAJA III year MS OG post graduate.
Dr. PRABHA JANAKIRAMAN MD OG , MRCOG,
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ABSTRACT:

Scrub typhus is endemic and re-emerging in eastern and southern Asia. Illness varies from mild and self-limiting to fatal. We present a case of scrub typhus diagnosed in the postpartum period suspected with unexplained tachycardia during the intraoperative period. Early diagnosis of cases will help in less severe organ damage and easy recovery with antibiotics. Few evidences state that scrub typhus can spread through blood transfusion. Correlation between blood transfusion and scrub typhus has to be further evaluated. Serological diagnosis and prompt treatment led to favorable outcome.

INTRODUCTION:

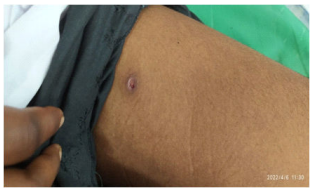
Scrub typhus is a mite born infectious disease causing acute febrile illness caused by Orientiatsutsugamushi. It is transmitted to humans through the bite of larval stage of trombiculid mites also known as chiggers (Ref harrisons textbook of medicine). The severity of infection can vary from mild symptoms to severe multiorgan failure. The most common clinical presentation of this disease is fever with chills, myalgia, eschar, headache, lymphadenopathy and other serious signs of organ damage. Hence diagnosis of scrub typhus should be included as a part of fever workup in endemic areas.

Key words : mite borne, febrile illness, postpartum infection.

CASE REPORT:

A 26 year old primigravida admitted in early labour with lower abdominal pain for safe confinement through our casualty. She was suspected to have a cephalopelvic disproportion and decided for emergency cesearean section . Intraoperatively patient had unexplained persistent tachycardia and hence patient was shifted to the HDU for postoperative monitoring. On the day following surgery she was transfused with one unit of packed cell as a part of anemia correction. Cardiology opinion and physician opinion obtained revealed normal echo findings with sinus tachycardia. On POD-2 patient developed fever on and off not relieved by antipyretics. Hence patient was started on higher antibiotics as per physician opinion and work up for fever was started. Since there was no improvement antimalarials also started .

Other than fever and unexplained tachycardia patient had no other symptom pointing to the exact diagnosis.Dengue and widal serology, peripheral smear for malarial and filarial parasite, sputum AFB found to be negative. All cultures including a high vaginal swab were sent to find out the source of infection. Expert ultrasound of abdomen and pelvis found to be normal and there was no pelvic collection or hematoma. Blood culture and high vaginal swab found to be negative for any infection.



CT CHEST taken found to have left sided minimal pleural effusion.Inspite of appropriate antibiotic patient had persistent fever with chills and rigor. Later a thorough physical examination in POD-4 revealed an eschar in the left upper arm of the patient and serology for scrub typhus taken and was found to be positive.

The patient was started on C.Doxycycline100 mg bd and T.Azithromycin500 mg od after shifting under imcu care and was continued for 7 days. Her fever settled within 24 hours of starting therapy. Her general condition improved and got discharged sucessfully on POD-14.

DISCUSSION:

Scrub typhus presents atypically in peripartum period due to altered immune response system of body. Previous analytical studies shows evidence that most women infected with scrub typhus never had eschar or any lymphadenopathy. Hence to conclude in post partum period scrub typhus presents in an atypical way and hence scrub typhus should be included as a part of fever work up in both endemic as well as non endemic areas.

CONCLUSION:

This case report emphasizes the need for primary care physicians to consider the most prevalent tropical illnesses in India while dealing with acute febrile illnesses. A high index of clinical suspicion, as well as rapid diagnosis, is required in managing tropical fevers, e.g., Scrub typhus, so that early treatment is initiated and end-organ damage be prevented. Even with the ongoing global public health crises due to COVID-19, simultaneous suspicion and evaluation of other common infections should not be delayed at primary healthcare, so that we achieve a timely diagnosis, early management, and favorable outcomes in our patients. No vaccine is available to prevent scrub typhus. Reduce your risk of getting typhus by avoiding contact with infected chiggers, avoid areas with lot of vegetations where chiggers may be found. Use protective insect nets and protective clothing for children to avoid bite by mites.(3)

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MALE INFERTILITY

Dr. GAYATHRI. N. M.D.,DNB(OG)

Due to changing life styles and priorities of the Modern World, the proportion of infertile couple approaching for medical help is in increasing many folds. When a couple have regular unprotected coitus for a year and have not conceived, we say they need evaluation. After evaluation if they are found to be normal (i.e) unexplained infertility – a quarter of them conceive in the next year with simple counselling and lifestyle and stress management.

Once upon a time, the society has always been very sexist and in all cases of infertility, the blame was pinned on the lady. But in reality, 40-50% of infertility can be pinned on the man – either alone or as a combination with his partner. It is believed that about 7% of all men are infertile and they account for 40-50 % of cases of infertility. It is a pure male factor abnormality or a combination of both partners contributing to infertility.

But now a days, thoughts to the changing attitudes – more men come forward to test themselves. They are open to discuss their sexual problems and sexual dysfunction. Thus we are able to understand and manage male infertility better.

It is said that

40-50% is idiopathic

30-40% Testicular causes

10-20% Post testicular

1-2% Hypothalamo –pituitary

IDIOPATHIC 40-50%	TESTICULAR 30-40%
POST TESTICULAR 10-20%	HYPOTHALAMOPITUITARY 1-2%

And the single most investigation in male factor is “SEMEN ANALYSIS”

Common abnormalities encountered in SA include

Oligozoospermia / Azoospermia

Isolated Asthenozoospermia

Isolated Teratozoospermia not very common.

OligoAsthenotexatozoospermia (OATS)

Obstructive azoospermia.

Now, let us see how to go on further, in diagnosing these common abnormalities.

Table A1.3 Nomenclature related to semen quality

aspermia	no semen (no or retrograde ejaculation)
asthenozoospermia	percentage of progressively motile (PR) spermatozoa below the lower reference limit
asthenoteratozoospermia	percentages of both progressively motile (PR) and morphologically normal spermatozoa below the lower reference limits
azoospermia	no spermatozoa in the ejaculate (given as the limit of quantification for the assessment method employed)
cryptozoospermia	spermatozoa absent from fresh preparations but observed in a centrifuged pellet
haemospermia (haematospermia)	presence of erythrocytes in the ejaculate
leukospermia (leukocytospermia, pyospermia)	presence of leukocytes in the ejaculate above the threshold value
necrozoospermia	low percentage of live, and high percentage of immotile, spermatozoa in the ejaculate
normozoospermia	total number (or concentration, depending on outcome reported)* of spermatozoa, and percentages of progressively motile (PR) and morphologically normal spermatozoa, equal to or above the lower reference limits
oligoasthenozoospermia	total number (or concentration, depending on outcome reported)* of spermatozoa, and percentage of progressively motile (PR) spermatozoa, below the lower reference limits
oligoasthenoteratozoospermia	total number (or concentration, depending on outcome reported)* of spermatozoa, and percentages of both progressively motile (PR) and morphologically normal spermatozoa, below the lower reference limits
oligoteratozoospermia	total number (or concentration, depending on outcome reported)* of spermatozoa, and percentage of morphologically normal spermatozoa, below the lower reference limits
oligozoospermia	total number (or concentration, depending on outcome reported)* of spermatozoa below the lower reference limit
teratozoospermia	percentage of morphologically normal spermatozoa below the lower reference limit

*Preference should always be given to total number, as this parameter takes precedence over concentration.

Table 2
WHO 2010 (5th Edition) and WHO 2021 (6th Edition) lower fifth percentile (with 95% confidence interval) of semen parameters from men in couples starting a pregnancy within one year of unprotected sexual intercourse leading to a natural conception.

	WHO 2010	WHO 2021
Semen volume (mL)	1.5 (1.4–1.7)	1.4 (1.3–1.5)
Total sperm number (10^6 per ejaculate)	39 (33–46)	39 (35–40)
Total motility (%)	40 (38–42)	42 (40–43)
Progressive motility (%)	32 (31–34)	30 (29–31)
Non progressive motility (%)	1	1 (1–1)
Immotile sperm (%)	22	20 (19–20)
Vitality (%)	58 (55–63)	54 (50–56)
Normal forms (%)	4 (3–4)	4 (3.9–4)

The reference ranges in the reports...
5th centiles are not significantly different from the 2010 WHO 5th Edition

OLIGOZOOSPERMIA/ AZOOSPERMIA:

According to WHO, the sperm count or concentration below the minimum standards is oligozoospermia (according to WHO 2021 6 th ed – 39 million sperms per ejaculate.). Azoospermia is the absence of sperm in the ejaculate.

ASTHENOZOOSPERMIA

Total sperm motility less than 42% or progressive motility less than 30% (according to WHO 6 th ED 2021).

TERATOZOOSPERMIA

This denotes problems in morphology of the sperm. According to WHO manual 2021, using strict tigerberg criteria, less than 4% normal morphology is teratozoospermia.

OATS (OLIGO-ASTHENO-TERATOZOOSPERMIA SYNDROME)

Most severe form of semen abnormality, where all parameters are subnormal, i.e., count, motility, and morphology.

CAUSES:

1. OLIGO/ AZOOSPERMIA

a. Pre-testicular

b. Testicular

c. Post-testicular

a. Pre-testicular- Mostly ENDOCRINOPATHIES

i. Congenital – Kallmans syndrome/ immotile Celia syndrome – associated with oligozoospermia

ii. Acquired

1. Pituitary adenomas – Prolactinomas being the most common

2. Other pituitary tumors lcausing hypogonadotropism

3. Congenital adrenal hyperplasia- adult onset type

4. Thyroid dysfunction- both hyperthyroidism and hypothyroidism

5. Functional causes- stress induced, anorexia nervosa.

b. Testicular causes: usually defects in spermtogenesis.

i. Genetic – 47 XXY, Microdeletion of Y chromosome – most common testicular cause of male infertility.

ii. Viral – most common is mumps orchitis in childhood or adolescence

iii. Environmental – exposure to high environmental temperature as in factories, Velders, exposure in environmental toxins, using very tight undergarments

iv. Undescended testes – if detected, early correction (Orchidopexy) can avoid irreversible damage.

v. Varicocele – Grade II or Grade III - can locally cause a increase in temperature and hamper spermatogenesis

vi. Iatrogenic – Chemotherapy and Radiotherapy are known to cause damage to the testes.

c. Post-testicular causes: block in the outflow tract.

i. Congenital – congenital absence of vas, cystic fibrosis

ii. Infective – Tubercular, Gonococcal, Chlamydia

iii. Retrograde ejaculation – suspected when volume of ejaculate is low on repeated samples. Diagnosis is by examining post ejaculatory urine sample for the presence of sperms.

1. Psychological

2. Neurological – Diabetes

3. Post surgical or trauma. (damage to the sympathetic plexus.)

2. ASTHENOZOOSPERMIA:

a. Infections – infections of the glands of the reproductive system may cause a local rise in concentration of reactive free radicles, thereby increasing the oxidative stress and causing decrease in the motility profile.

b. Antisperm antibodies - breakage of blood testes barrier by trauma, infection, local rise in temperature - can cause anti sperm antibody - in turn causes decreased motility.

c. Defects in sperm - Axoneme defects, Mitchondrial defects, Centriolar dysfunction - all these can cause asthenozoospermia.

d. Epididymal pathology - "Carnitine" in the epididymis is involved in maintaining sperm viability and motility - Infection or any other epididymis pathology can affect these sperm functions.

e. Genetic - Kartageners syndrome is a well known genetic cause of asthenozoospermia

f. Iatrogenic - For asthenozoospermia, Iatrogenic causes are rare.

3. OATS:

Extreme degree of semen abnormality.

Involves count, motility and morphology.

Sperms of sample with OATS are usually not considered ideal even for ICSI.

4. TERATOZOOSPRMIA:

Plasma membrane defects, chromatin aberration, increased cytoplasmic vacuoles - all these can cause failure of fertilisation.

EVALUATION OF THE MALE PARTNER:

As has always been the teaching from our under graduation, evaluation starts with detailed history, examination and investigations.

Complete history and physical examination

Semen Analysis

Endocrine evaluation

Any additional special workup.

HISTORY

A detailed history of any penile/ scrotal trauma, Cryptorchidism, child hood infections – mumps, any other orchitis, accessory gland infections.

Environmental and occupational history to rule out any environmental causes of poor semen parameters.

Treatment history, history of drug abuse, alcoholism, smoking etc.

History of loss of libido, headache and visual symptoms may indicate central cause such as pituitary tumour.

Past medical and surgical history, history of metabolic disorders, thyroid and liver diseases, long standing diabetes with neuropathy, pelvic surgeries, hernia repairs.

Family history of genetic diseases or if the male is a known case of any genetic issues.

EXAMINATION

A systematic and detailed examination of the male partners reproductive organs should be done and should include examination of the penis, scrotum, testes, vas deference, spermatic cord, ejaculatory ducts, urethra, urinary bladder, rectum and the anus.

INVESTIGATIONS:

Semen analysis with leucoscreen when indicated is the first and foremost investigation needed to evaluate the male partner. All other investigations are done only when indicated.

If the leucoscreen is positive, semen culture may be needed.

In suspected case of retrograde ejaculation, a post ejaculatory urine analysis is done to look for sperms.

Sperm DNA fragmentation and Sperm FISH are other special investigations which are required in special conditions.

USG abdomen and scrotum are usually needed to look for testicular volume and any other pathologies.

Blood investigations generally called for in evaluation of the male factor include, FSH/LH, Testosterone, thyroid profile, prolactin. When genetic disorders are suspected, genetic counselling, karyotying and genetic mapping may be indicated.

The other tests available, but not commonly used are – ASA testing, test for reactive oxygen species, sperm penetration tests.

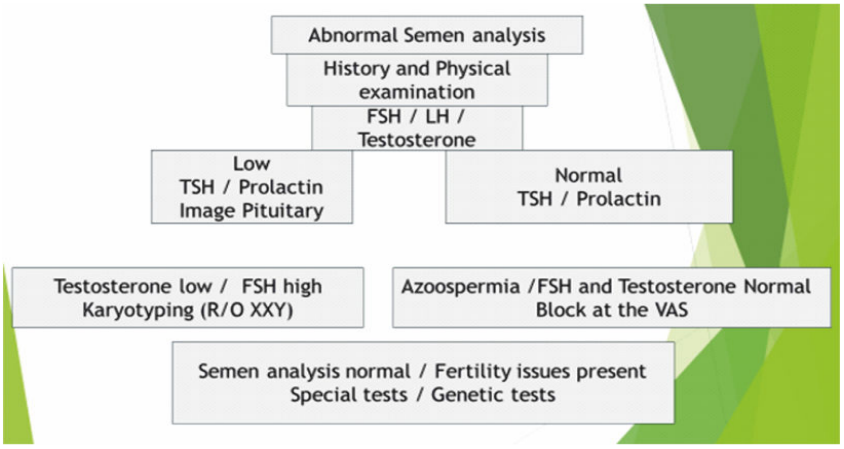
SPERM DNA FRAGMENTATION

Abnormal DNA fragmentation occurs in defective packing of DNA at spermatogenesis, during the process of cell death, oxidative stress.

More than 30% DNA fragmentation is considered abnormal.

It helps us select candidates for varicocelectomy in men with normal or borderline parameters, provides an explanation for unexplained infertility, recurrent pregnancy loss, repeated IUI failure, provides prognostic information in couple with failed IVF/ICSI.

TO SUM UP THE DIAGNOSTIC ALGORITHIM



MANAGEMENT:

Pre testicular causes will need management of the endocrine or other underlying condition and lifestyle modification.

Testicular causes- correction or treatment is seldom likely to succeed. The management will usually be assisted reproductive technology – IUI/ IVF / ICSI

Post testicular causes (Obstructive) - usually treatment is testicular aspiration procedures combined with ICSI.

Isolated asthenozoospermia and other isolated mild abnormalities have shown good improvement with the use of antioxidants is some cases. It is worth trying a course of 3 months of medical management with antioxidants in such cases.

MEDICAL MANAGEMENT:

There are a number of drugs for male infertility in the market with different combinations. But those antioxidants with promising benefits in a lot of trials include-

VIT C – 1 g / day

VIT E – 1 g / day

L-Carnitine – 3-4 g / day

Lycopene – 8 mg / day

CoQ 10 – 100 mg / day

Astaxanthine – 16 mg / day

We can choose a combination with all or most of these for better results in the repeat sample. The other drugs used in male infertility are...

FSH, LH in hypogonadotropic hypogonadism,
Clomiphene citrate 25 mg
Cabergolin (antiprolactin)
Correction of Thyroid disorder
Sildinafil citrate (Sexual / Erectile dysfunction)
Antibiotics

After a thorough evaluation we may still need an andrologist opinion to complete the management. There are a few conditions when referral to the andrologist has to be early -

Advanced age

Years of infertility

Medical management tried outside

Anxiety level of couple.

Response to treatment is not as expected

When patient gives history of sexual dysfunction, even with normal semen parameters.

Thus, when as gynaecologist we have exhausted all we have in our basket, we refer to our andrology colleagues.

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THE CHANGE

Dr. K. PARIMALARANI M.D.,OG

Aren't you perplexed most of the time when you hear as to why people prefer sons over daughters?

As an obstetrician, I often get intrusive, when an antenatal mother confronts me, if her baby is a boy or a girl. Most of the time, I might have sounded rude to them and my answer would be, what does it matter, if it were a boy or a girl?

My life is a sheer privilege, because my parents did not love me less because I was born as a daughter. I owe my life to my parents, I owe my education to my father, I owe my carrier to my husband.

Isn't it ridiculous, that we often land up thanking many, for the basic rights of a woman. The right to live, the right to education, the right to have a carrier of her choice, the right to make decisions about her own body, the right to procreate as well as to abstain from procreating and her liberty over personal choices.

The common wisdom in the Indian scenario, for the preference of sons over daughters, is motivated by socioeconomic, religious and emotional desires and norms, that favor males and make females less desirable. The sons continue the family lineage, they add to family wealth and property and are expected to provide financial and emotional support for their old parents, while daughters are perceived as a liability and to be creating a burden on the household.

Emma Waston said, "Don't you think it is right that she should be paid as her male counterpart for the same job? Don't you think that women be involved in policies and decisions that will affect a woman's life on her behalf? Don't you think that she is afforded the same respect as men? But sadly there is no country in the world, where all women can expect to receive these rights.

If each family raises their boy and girl with equal opportunities, we will leave behind generations of inequalities and exclusions shortly. Let's teach our children that there is no sustainable development without equal participations of women. Educating women is not a privilege or a prize, it is the weapon to bring down boundaries and to see what good can they bring to the rest of the world. If girls are educated, they will not be married of early, If girls are educated, they reason and they question, If girls are educated, they will have lesser children, If girls are educated they boost up the economy.

It's time to build an equal future, whether running a home, a business, a country or a popular movement. Let's bring change in our mind, our thoughts our ideas and our perspective and believe and work on it for creating a future, that hold more good times for women. Her problem shouldn't be solved by any form of coercion or making her welfare conditional, is a hallmark of coercion. When women embody the values of resistance and courage, then there is no limit what she can accomplish.

I am imagining a time where women's voices are heard, in a no longer patriarchal world , I am imagining a time when women no longer belong only to the kitchen, fighting greasy pots and pans, I am imagining a time when women has liberty and decisional autonomy over those governing reproduction, I am imagining a time when there is no invasion upon her most personal and ethical choices.

As Christine Legard said,

"I would love to see a world where women let their confidence roar from the rooftops, their cups run over with self assurance and their voices resound across the pinnacles of power".

THE WINDS OF CHANGE HAS

STARTED TO BLOW

THE SOUND OF MUSIC IS JUST

ROUND THE CORNER

THE SMELL OF FRUITS IS JUST

A FEW STEPS AWAY

THE GOOD TIMES ARE HERE

TO STAY

THROMBOCYTOPENIA IN PREGNANCY

Dr. L.LATHA DGO.,DNB(OG),

Thrombocytopenia is defined as blood platelet count below 1,50,000 / ml during pregnancy. It complicates 7-10% of pregnancies.

CAUSES:

- Gestational thrombocytopenia- 70-80%
- Hypertensive disorder- 20%
- Immune thrombocytopenic purpura - 3-4%

Physiology : This is a physiological decrease in platelet count during normal pregnancy due to haemodilution, increased consumption in pheripheral tissue and increased aggregation due higher thromboxane A2.The physiological thrombocytopenia of pregnancy is mild and do not affect the fetus and mother. On contrast significant thrombocytopenia can cause serious maternal - fetal consequences and requires specific monitoring and appropriate management. We will focus on gestational thrombocytopenia and ITP.

GESTATIONAL THROMBOCYTOPENIA:

It is a benign condition with moderate thrombocytopenia 1,30,000 to 1,50,5000/ml in most cases, platelets below 50,000/ml excludes GT and requires search for another etiology.GT is diagnosis by exclusion.

- Usually asymptomatic
- Occurs in second half of pregnancy
- No H/O thrombocytopenia prior to pregnancy. Platelet count resolve within first 2 months postpartum.

MANAGEMENT:

1. GT is not associate with maternal and fetal risks. So periodic monitoring of blood count is done
2. If platelet count falls between 50,000 to 80,000/ ml, ITP cannot be excluded. In such case methylprednisolone in daily dose for 10 days before birth should be administered in order to increase platelet count and avoid obstetrical and anaesthesia risk during delivery.

IMMUNE THROMBOCYTOPENIC PURPURA:

- Rare cause of thrombocytopenia in pregnancy.
- Chronic form is common in women.
- Symptoms are related to platelet count.
- Presence of splenomegaly rules out GT.
- Usally asymptomatic.
- Some present with ecchymosis, Petechiae ,purpura, gum bleeding.
- For safe vaginal delivery platelet count more than 30,000 /ml is sufficient.
- For operative vaginal delivery and caesarian section platelet count at least 50,000/ml is ideal.
- Safe platelet level for epidural / spinal is around 75,000 to 80,000/ml.

- Spontaneous bleeding occurs if platelet count less than 20,000/ml.
- Prednisone 1mg/ kg or IVIG.1 mg/kg is the first line of treatment.
- Therapeutic response occurs within 2-14 D
- If unresponsive to above treatment, splenectomy is usually done .Usually done in 2nd trimester.
- For emergency LSCS ,if platelet count is less than 50,000/ml, Platelet transfusions along with IVIG is recommended.

NEONATAL RISK:

Neonatal thrombocytopenia occurs maximum at day 2-5. Hence daily platelet monitoring should be done. In newborn, platelet transfusion done if platelet count less than 50,000/ml .USG Head should be done to rule out intracranial haemorrhage.

If platelet count between 30,000 to 50,000/ml .the newborn may require IVIG.

NON-CIRRHOTICPORTAL HYPERTENSION IN PREGNANCY

Dr. PRIYANKA VELCHAMY DGO.,

Portal hypertension is defined as a pathologic increase in the pressure of the portal venous system. Cirrhosis is the most common cause of portal hypertension, but it can also be present in the absence of cirrhosis, a condition referred to as "noncirrhotic portal hypertension."

Noncirrhotic portal fibrosis or idiopathic portal hypertension is a disease of uncertain etiology characterized by periportal fibrosis and involvement of small and medium branches of the portal vein, resulting in the development of portal hypertension with preserved liver function and structure.

HEMODYNAMIC CHANGES IN PREGNANCY

Pregnancy is associated with number of changes in maternal physiology as an adaptive response to the physiologic needs of the developing fetus. In patients with portal hypertension, these changes can worsen the portal hypertension and markedly increase the risks of variceal hemorrhage.

Pregnancy is associated with vasodilation of the systemic vasculature and the maternal kidneys. The systemic vasodilation of pregnancy occurs as early as at 5 weeks and therefore precedes full placentation and the complete development of the uteroplacental circulation.

In the first trimester, there is a substantial decrease in **peripheral vascular resistance,** which decreases to a nadir during the middle of the second trimester with a subsequent plateau or slight increase for the remainder of the pregnancy. The decrease is ≈ 35% to 40% of baseline. Systemic vascular resistance increases to near-pre pregnancy levels postpartum,and by 2 weeks after delivery, maternal hemodynamics have largely returned to nonpregnant. Systemic vascular resistance increases to near-pre pregnancy levels postpartum.**Vasodilation of the kidneys** results in a 50% increase in renal plasma flow and glomerular filtration rates by the end of the first trimester. This results in decreases in serum creatinine, urea, and uric acid values.

Cardiac output increases throughout pregnancy. The sharpest rise in cardiac output occurs by the beginning of the first trimester, and there is a continued increase into the second trimester. After the second trimester, there is debate as to whether cardiac output increases, decreases, or plateaus. By 24 weeks, the increase in cardiac output can be up to 45% in a normal, singleton pregnancy.

There is a decrease in **arterial pressures**, including systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure, and central SBP during pregnancy. DBP and mean arterial pressure decrease more than SBP during the pregnancy. Arterial pressures decrease to a nadir during the second trimester (dropping 5–10 mm Hg below baseline), but the majority of the decrease occurs early in pregnancy (6- to 8-week gestational age) compared with preconception values.

Arterial pressures begin to increase during the third trimester and return close to preconception levels postpartum

Heart rate increases during normal gestation. Heart rate increases progressively throughout the pregnancy by 10 to 20 bpm, reaching a maximum heart rate in the third trimester. The overall change in heart rate represents a 20% to 25% increase over baseline.

PATHOPHYSIOLOGICAL EFFECTS OF PORTAL HYPERTENSION

HPVG (mm Hg)	Clinical features	Stage of cirrhosis
1-5	Normal, non-cirrhotic	-
6-10	Compensated cirrhosis	1
>10	Compensated cirrhosis with development of varices	2
>12	Decompensated cirrhosis with ascites,variceal bleed, hepatic encephalopathy	3-4

Portal hypertension most commonly results from cirrhosis. Due to irreversible progressive damage in cirrhosis, these women usually have amenorrhea and infertility. Noncirrhotic portal hypertension can be encountered without evidence of liver disease. The first known mechanism of portal hypertension is an increase in intrahepatic resistance to blood flow. Hepatic damage thus caused results in shunting of hepatic blood, development of extrahepatic collaterals and elevated pressure in the portal venous system.

MATERNAL COMPLICATIONS

In pregnancies with portal hypertension 30%–50% of pregnancy suffer from portal hypertension associated complications, resulting mainly because of variceal bleed and hepatic failure. The severity of complications depends on the cause of portal hypertension and disease severity.

Anemia of microcytic, hypochromic (due to GI blood loss) or normocytic, normochromic, leukopenia (< 50,000 mm−3) may be present and are due to hypersplenism but usually asymptomatic. Jaundice, ascites, signs of chronic liver disease are uncommon in non-cirrhotic portal HTN

ESOPHAGEAL VARICES

Gastro-intestinal hemorrhage remains the most catastrophic complication of portal hypertension during pregnancy. Variceal bleed has been reported in 18–32% of pregnant patients with cirrhosis and in 50% with a known portal hypertension. About 75% of patients with varices bleed during pregnancy which is one of the most serious consequences. This is due to increased flow and pressure transmitted to collaterals due to hyperdynamic circulation during pregnancy. The dreaded complication of active variceal bleeding may occur at all stages of the pregnancy though second and third trimester and second stage of labor are the time of greatest risks of variceal bleed. Predictors of variceal bleed during pregnancy associated with portal hypertension are large varices, presence of endoscopic red signs and history of pre conceptional variceal bleed and untackled or undiagnosed varices. Approximately 7–9% of patients with portal hypertension suffer from symptomatic anemia irrespective of the cirrhotic state. The upper gastrointestinal endoscopy is safe in pregnancy with a small risk of fetal hypoxia from sedation and patient positioning. The mainstay of treatment remains endoscopic variceal ligation (EVL). Pregnant patients at risk for variceal bleed should receive primary prophylaxis, with either endoscopic variceal ligation or b blockers. b blockers are generally considered safe in pregnancy however propranolol and nadolol both carry category C risk and have risk of causing fetal bradycardia, growth retardation and neonatal hypoglycemia.

Screening for esophageal varices is recommended by most experts during the early second trimester or before pregnancy.

POSTPARTUM HEMORRHAGE

These patients are at a high risk of post-partum hemorrhage which occurs in 7%–10% of cases and is commoner in patients with cirrhosis. Post-partum hemorrhage may be due to associated coagulopathy as a result of liver dysfunction and thrombocytopenia due to hypersplenism associated with portal hypertension or cirrhosis per se. The treatment remains the same as those in patients without cirrhosis of liver. These patients require blood and coagulation factors along with uterine contractile agents such as oxytocin. Prevention by active management of third stage of labor is the mainstay of management.

PERINATAL COMPLICATIONS

The rates of spontaneous abortion, premature birth, still births and perinatal death are increased in women with portal hypertension. There is 10%–66% fetal wastage in patients of liver cirrhosis and spontaneous abortion rate of about 20% first trimester abortion.

MANAGEMENT IN PREGNANCY

Pregnancy in a patient with portal hypertension requires a multispecialty team approach including expert obstetrician, hepatologist, neonatologist and anesthesiologist in a tertiary care center.

PRE-CONCEPTION

A complete medical history, detailed examination, lab investigations and imaging studies need to be performed to assess the cause of the disease and its status. Various poor predictors of a successful pregnancy with portal hypertension include history of variceal bleed, large varices, presence of other co-morbidities like jaundice, thrombocytopenia, ascites, hypersplenism, etc. A complete medical history, detailed examination, lab investigations and imaging studies need to be performed to assess the cause of the disease and its status. Various poor predictors of a successful pregnancy with portal hypertension include history of variceal bleed, large varices, presence of other co-morbidities like jaundice, thrombocytopenia, ascites, hypersplenism, etc.

Surveillance endoscopy should be done in the pre-conceptional period. Varices should be tackled prior to planning a pregnancy, endoscopic variceal ligation is the preferred therapy and non-responders should be offered surgery in the form of shunt procedure or splenectomy.

Drugs should be reviewed for adverse effects on the fetus and alternative safe drugs to be changed, and also dose needs to be tailored. Prednisolone and azathioprine, if needed, can be continued in the minimum effective doses. Spironolactone should preferably be discontinued. Selective b blockers can be continued as their benefits outweigh risks.

ANTENATAL MANAGEMENT

Maternal and fetal prognosis is dependent on the cause of underlying liver disease and its status at the time of conception. Pregnancy is not a contra-indication if the disease is well compensated. Antenatal management requires strict maternal and fetal monitoring by a multidisciplinary team. The routine antenatal management should be given with special watch out for the potential complications like variceal bleed and liver failure. Anemia should be avoided and if present treated emphaticallyas anemia itself also leads to cardiac compromise in addition to being a risk factor for pre-term labor, low birth weight. Liver function and

hematological assessment should be done 4 weekly, fetal growth needs to be monitored and effects of the drugs need to be watched. Close maternal and fetal monitoring by the joint team is recommended two weekly. The principles of management include anticipation, early recognition and management of the antenatal complications associated with portal hypertension.

Variceal bleeding being one of the most common complication should be tackled pre pregnancy. Upper GI endoscopy if needed, even prophylactically in the 2nd trimester as the maximum increase in portal pressure occurs at that time. Esophageal variceal ligation can be done when needed. Though non-selective beta blockers used to reduce portal pressure also reduce the risk of first bleed by half but the principal risk of using them in pregnancy is fetal growth restriction and fetal bradycardia. EVL of the large varices can also be done during pregnancy to prevent variceal bleeding. Current literature recommends EVL for acute esophageal variceal bleed, although, endoscopic sclerotherapy may be used if banding is technically difficult. Aspirin or nonsteroidal anti-inflammatory drugs should be avoided. There are no controlled trials for efficacy and safety of medical versus surgical treatment during pregnancy, most of the reports are from cirrhosis patients. Pregnancy can be allowed to go to term if the disease is well compensated. Early termination of pregnancy may be warranted in case of any obstetrical indication or progressive liver failure.There are no recommendations as to the preferred mode of delivery- vaginal vs caesarean section in patients with portal hypertension. Cesarean can be usually be done for obstetric indications.

PERIPARTUM MANAGEMENT

Labour management should be individualized according to the patient.Adequate amount of blood and plasma should be arranged and measures for balloon tamponade for the variceal hemorrhage must be handy. Second stage of labor may be shortened prophylactically to avoid overstraining by the mother.Postpartum hemorrhage should be anticipated and managed vigilantly. Antibiotics use needs to be individualized. Caesarean delivery is usually carried out in case of obstetric indications.

POSTPARTUM MANAGEMENT

Strict vigilance for postpartum hemorrhage should be done. Antibiotics should be given in the postpartum period. Spontaneous bacterial peritonitis is a specific complication which may develop in the puerperium especially in the presence of ascites. Puerperal fever should be investigated and treated with appropriate antibiotics. Breast feeding is not contraindicated as such. Reliable contraception must be advised in the form of barrier methods, intra uterine devices or permanent sterilization.Hormonal contraception is usually avoided as they can cause cholestasis.

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MULLERIAN CYST OF VAGINA- A CASE REPORT

Dr. ARTHI. G

INTRODUCTION:

Benign cystic lesions of vagina are uncommon and mostly asymptomatic.Prevalence of vaginal cyst is < 1%. They typically present in the third or fourth decade of life.Most common vaginal cysts are Mullerian cyst (30%), Epidermal inclusion cyst(25%), Gartner's duct cyst, Mucous inclusion cyst, Embryonic cyst and Urothelial cysts. Less common vaginal cysts are endometrial cysts and vaginal emphysematosa.

CASE SUMMARY:

31yrs old, P3L3, Sterilised, All Normal Vaginal Delivery, came with the C/O mass descending through vagina since 5yrs, non progressive, not associated with pain.H/o dyspareunia present. No other significant complaints.

Vitals : HR- 78beats /min , normal volume, regular rhythm, BP-110/70 mmHg, Spo2-99% at room air , RR-16 cycles /min

Examination :

CVS - S1, S2 heard, No murmurs
RS - B/L air entry present
CNS- No focal neurological deficit
P/A - soft , Non tender

Local examination :

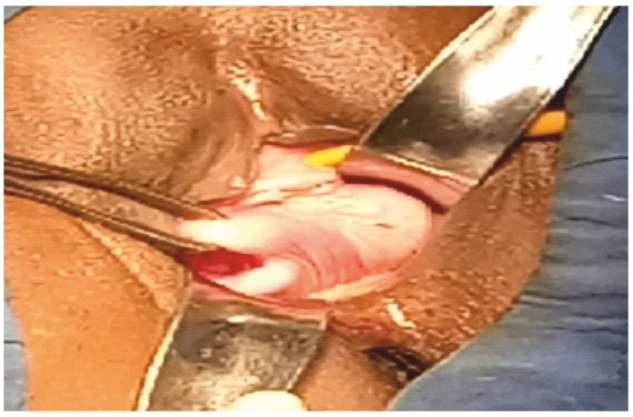
Cystic mass of size 4x3 cms seen protruding from vagina

Per speculum examination :

Cystic swelling arising from anterior vaginal wall. Vaginal rugosities over the swelling was absent. No cough impulse on swelling. Cervix healthy.

Per vaginal examination :

Cervix downwards, Uterus Anteverted, Normal size, Fornices Free, No Forniceal tenderness.



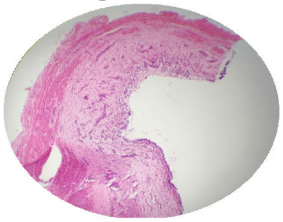
Investigations:

Complete haemogram, Renal function test, Liver function test, Thyroid function test - normal, USG Abdomen and Pelvis shows normal study, Pap smear report – Inflammatory smear, Urine C/S – No growth after 48 hrs of inoculation.

Complete excision of the cyst via a vaginal approach, under spinal anaesthesia.

HISTOPATHOLOGY:

Cyst measuring 3x2.2x0.5cms. Cross section shows unilocular cyst. Cyst wall lined by columnar to low cuboidal mucinous/tubal epithelium with underlying congested fibrous layer and muscle tissue. Cyst reported as **Mullerian cyst of Vagina.**



DIFFERENTIAL DIAGNOSIS:

The differential diagnosis of cyst in lower female genital tract include

Mullerian cyst	Mesonephric cyst (Gartner's duct)
Inclusion cyst	Bartholin gland cyst
Urethrocele	Urethral diverticulum
Skene's duct cyst	Pelvic organ prolapse
Hematocolpos	Myxomatous tumour

DISCUSSION:

Reported in approximately 1 in 200 females.Among all vaginal cysts, Mullerian cyst is commonest.Mullerian cysts are usually small, ranging from 0.1 to 2 cm in diameter. Rarely, they may be enlarged and mistaken for other structures, such as a cystocele.Usually it involves Anterolateral vaginal wall.Usually asymptomatic, but it can also present as mass descending per vaginum, pain, dyspareunia and abnormal vaginal discharge.They are usually single but occasionally may be multifocal.They are usually benign but malignant transformation has been noted once.Imaging modalities like USG and MRI are useful in exact localisation and to know the number and communication with surrounding structures.This cyst can be lined with epithelium from the cervix, uterus and fallopian tubes. Most commonly by endocervical (mucinous).Vaginal cysts are treated via excision. The entire cyst wall must be removed to prevent recurrence.

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AN INTERESTING CASE OF VESTIBULAR ANUS WITH ACCESSORY OVARY -AN INCIDENTAL FINDING DURING LAVH

Dr. R. GOWRI MANOHARI

Guided By :

Dr. PILLAI ARTHI KARUNANITHI

ABSTRACT

41 years old P2L2 with h/o previous 2 LSCS presented with complaints of heavy menstrual bleeding and anaemia. On examination uterus was 14 weeks size with USG showing fibroid uterus with left side adnexal mass (?Tubo-Ovarian mass).

In local examination anal opening was absent and it was found to be opening above the fourchette. Patient was aware about the condition and didn't seek treatment as she was not having any inconvenience.

After correcting anaemia patient was taken up for laparoscopic assisted vaginal hysterectomy in which accessory ovary was found in the left side adnexa.

This is a case report of two rare entities incidentally found in a fibroid case during LAVH surgery.

CASE REPORT

Vestibular anus is a type of anogenital tract abnormality in which anus is located in a location other than the usual location and it is usually diagnosed early in Infancy and corrected. But in this patient it was unusual, she is 41 years old P2L2 and was examined for complaints of abnormal uterine bleeding and in local examination anal opening was not found and anal opening was seen located above the fourchette and separated from the vagina by a thin septum and perineal body was seen located behind the fourchette. There was no dimple or depression in the perineum. Anal orifice was completely within the vulva. When asked the patient about the deformity she said she is aware of it and since birth she has no difficulty in passing stools or no history of faecal or flatus incontinence except on days when she has loose stools hence she didn't seek any medical advice.

And when asked about the labour details, she said her first baby was Breech hence she was taken up for Elective LSCS and 2nd delivery was elective repeat LSCS.

Patient was anaemic and was corrected with blood transfusion and taken for LAVH intra operatively accessory ovary was found in the left adnexa separated from the normal ovary by a band of tissue and laterally attached to Infundibulo pelvic ligament so as per Wharton's rule it was classified to be Accessory Ovary.

Coexistence of Vestibular Anus and Accessory ovary is a very rare finding which was incidentally found in a case admitted for LAVH was found interesting hence Embryology of Accessory ovary and Vestibular anus was discussed in this paper.

DISCUSSION

1) VESTIBULAR ANUS:

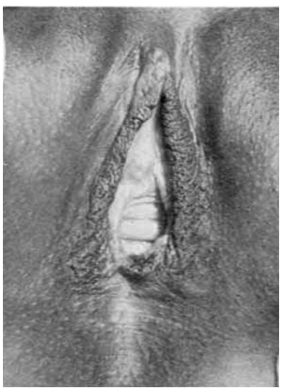
It occurs due to failure in the development of the Urogenital septum, so primitive cloaca is not divided into sinus urogenitalis and the rectum. Cloacal duct persists, so rectum opens

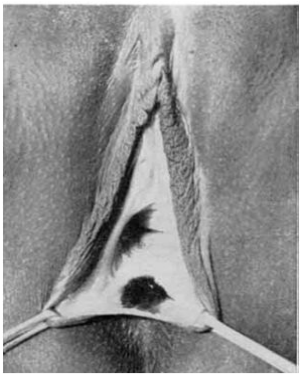
into the posterior vaginal wall, vestibule, perineum, bladder or urethra.

It may be associated with other malformations like pelvic bone deformities, VACTERAL group of anomalies. Surgical evacuation in infancy is not mandatory if satisfactory bowel evacuation is possible.

The corrective operation usually to be performed for this condition is isolating the anal canal from the vaginal opening and is directed towards the new position posteriorly. Perineal body will then be build up using chromic catgut sutures and the anal orifice will be stitched to the overlying skin. The skin around the new anal opening will form cogwheel rugae like a typical anal margin.

In our case LAVH was done for this patient. She was prepared for surgery by Colon wash instead of enema. Intraoperatively there was no difficulty in applying the clamps from down as the vaginal orifice and pelvic space was good and no injury happened to the septum or anal mucosa and post operatively bowel motility was good and passed stools on POD #3 with good continence as before





2) ACCESSORY OVARY:-

Accessory ovary is a type of ectopic ovary and the other being Supernumerary ovary. It is a rare encounter with an incidence of 1 in 29,000 – 93,000 cases.

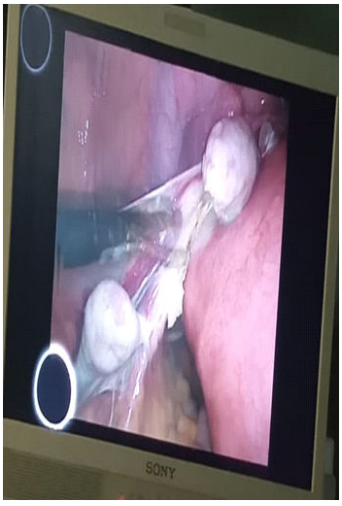
Ectopic ovaries are usually associated with Mullerian anomalies, renal agenesis, Duplicated ureters, bladder diverticulum, accessory adrenal glands and a lobulated liver

In this patient in her pre operative evaluation for AUB in the Ultrasound in the left adnexa a homogenous mass of size 2.1* 3.4 cm was visualized separately away from the left ovary which was suspected to be a? TO mass /Adnexal mass

Hence tumor markers-Ca-125 and CEA was done and was found to be normal During LAVH procedure, Two ovaries were visualized in the left adnexa, an accessory ovary was visualized attached to the Infundibulo pelvic ligament laterally and medially to another ovary through a ligamentous band of tissue, which was suggestive of Accessory ovary as it was attached to Gynecological ligament and also attached to the other ovary by a band of tissue.

Since the risk of malignant transformation of the accessory ovary is high all three ovaries were removed and sent for Histopathological examination

In the histopathological evaluation, Section from the labeled accessory ovary showed-Ovarian stroma with a single corpus luteal cyst.



SUPER NUMERARY OVARY	ACCESSORY OVARY
They have no connection with any Gynecological ligaments There is an aberration in the migration of the gonadocytes form the yolk sac endoderm through the dorsal mysentry before reaching the genital ridge during embryogenesis So this results in development of ovaries in a location which was unexpected and coincides with the gonadocytes location at the time of disruption.	These are extra ovaries which are found attached to any of the gynecological ligaments along the direction of the Genital ridge. After arriving at the genital ridge primordial gonodocyte groups split and mature into separate ovaries.

The clinical implication of the ectopic ovary is to identify it and remove it when ovaries are being prophylactically removed in cases with BRCA mutation positive cases, if the ectopic ovary is not identified and left without removing chances of Malignancy will be high.

CONCLUSION:-

1. Two different rare entities of anomalies one in the Genito urinary tract and one in the anogenital tract seen in the same patient and was managed efficiently such that no complications happened. Patient was discharged on POD # 4 and was reviewed after 1 week and was found to have no complaints and was stable and healthy 1. A case of Vestibular anus and Accessory ovary in the same patient has been described and its embryology over viewed
2. This patient didn't needed corrective surgery as her Anal continence was good so in those with the same type of malformation it could be managed without corrective surgeries provided they are able to maintain their bowel evacuation control effectively.
3. The risk of malignant transformation of accessory ovary and its correct identification in Prophylactic Salphingo oopherectomy procedures has been described.

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AN "ODE" TO THE OBSTETRICIAN

Dr. VIJAYAPRABHA CHEZIAN

The delivery board is like a cricketing field.

The obstetrician is more like a person waiting for the catch surrounded on all sides by fielders.

And **Lo** and **bestow!!**.

When the new comer arrives She is quick and **agile.**

And pulls the little one so **fragile**

From the canal so **narrow**

like a rabbit from its **burrow**.

Everybody's heart forgets to **tick**.

Till the baby gives a **Kick** and there's a shout and **cry**

The crowd disappears for the next **try**.

At times so quite and pensive.

The baby's cry so **Shrill** sends down our spine a **thrill**.

A testimony to prove that God is **STILL** Working wonders, through you all Obstetricians.

Gokulnathan

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TEST CODE: WM15S

Swasth Fit Super - 3
NO. OF PARAMETERS - 29 | ~~₹5510~~ ₹1550
Sugar Fasting, Thyroid Profile, Lipid Profile, LFT & KFT, Vitamin-D, Vitamin B-12.
TEST CODE: WM16S

Swasth Fit Super - 4
NO. OF PARAMETERS - 49 | ~~₹5950~~ ₹1750
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NAUSEA AND VOMITING OF PREGNANCY

Dr UMA VELMURUGAN MD(OG); FRCOG; CCT UK

Nausea and vomiting is a common condition that affects the health of a pregnant women and her fetus. NVP affects up to 80% of pregnant women and is one of the most common indications forhospitalisation.

The women's perception of the severity of her symptoms, her desire for treatment and potential effect of treatmenton her fetus all influence clinical decision making .There is variation in the management of women who had NVP or hyperemesis gravidarum(HG). Early treatment of nausea and vomiting may be beneficial to prevent progression to hyperemesis gravidarum. HG is clinical diagnosis of exclusion of otherdiseases causing nausea and vomiting.

Aetiologyof NVP in pregnancy is thought to be associate with rising levels of beta human chorionic gonadotropin (HCG) hormone .Conditions with higher HCG levels such as trophoblastic disease and multiple pregnancy have been associated with increased severity of NVP.

DIAGNOSIS

NVP should only be diagnosed when onset is in the first trimester of pregnancy and other cause of nausea and vomiting have been excluded. HG is diagnosed when there is protracted NVP with triad of more than 5% prepregnancy weight loss, dehydration and electrolyte imbalance An objection validated index of nausea and vomiting such as Pregnancy- Unique quantification of Emesis (PUQE) score can be used to classify the severity NVP.

Pregnancy-Unique Quantification of Emesis (PUQE) index 20

Total score is sum of replies to each of the three questions. PUQE-24 score: Mild ≤ 6; Moderate = 7–12; Severe = 13–15.

Motherisk PUQE-24 scoring system

In the last 24 hours, for how long have you felt nauseated or sick to your stomach?	Not at all (1)	1 hour or less (2)	2–3 hours (3)	4–6 hours (4)	More than 6 hours (5)
In the last 24 hours have you vomited thrown up?	7 or more times (5)	5–6 times (4)	3–4 times (3)	1–2 times (2)	I did not or throw up (1)
In the last 24 hours how many times have you had retching or dry heaves	No time (1)	1–2 times (2)	3–4 times (3)	5–6 times (4)	7 or more times (5)

PUQE-24 score: Mild ≤ 6; Moderate = 7–12; Severe = 13–15.

Features in the history, examination and investigations to monitor severity and other causes

History _previous history of NVP/HG

Quantify severity using PUQE score: nausea, vomiting, hypersalivation, spitting, and loss of weight, inability to tolerate food and fluids, effect on quality of life

History to exclude other causes:

Abdominal pain

Urinary symptoms

Infection

Drug history

Chronic *Helicobacter pylori* infection

Examination Temperature

Pulse

Blood pressure

Oxygen saturations

Respiratory rate

Abdominal examination

Weight

Signs of dehydration

Signs of muscle wasting

Other examination as guided by history

Investigation _Urine dipstick:

Quantify ketonuria as 1+ ketones or more

MSU

Urea and electrolytes:

Hypokalaemia/hyperkalaemia

hyponatraemia

Dehydration– renal disease

Full blood count:

Infection

Anaemia

Haematocrit

Blood glucose monitoring:

Exclude diabetic ketoacidosis if diabetic

Ultrasound scans:

Confirm viable intrauterine pregnancy

Exclude multiple pregnancy and trophoblastic disease

In refractory cases or history of previous admissions, check:

TFTs: hypothyroid/hyperthyroid

LFTs: exclude other liver disease such as hepatitis or gallstones, monitor malnutrition

Calcium and phosphate

Amylase: exclude pancreatitis

ABG: exclude metabolic disturbances to monitor severity

Differential diagnosis

Symptoms of nausea and vomiting of pregnancy manifest before 9 weeks of gestation .When a patient experiences nausea and vomiting for the first time after 9 weeks of gestation other conditions should be considered in differential diagnosis

Differential Diagnosis of Nausea and Vomiting of Pregnancy

Gastrointestinal conditions

Gastroenteritis	intestinal obstruction
Gastroparesis	peptic ulcer disease
Achalasia	pancreatitis
Biliary tract disease	appendicitis
Hepatitis	

Conditions of the genitourinary tract	*Metabolic conditions*
Pyelonephritis	Diabetic ketoacidosis
Uraemia	Porphyria
Ovarian torsion	Addison's disease
Kidney stones	Hyperthyroidism
Degenerating uterine leiomyoma	Hyperparathyroidism

Neurologic disorders	*Miscellaneous conditions*
Pseudotumor cerebri	Lymphocytic hypophysitis
Vestibular lesions	Drug toxicity or intolerance
Migraine headaches	Psychologic conditions
Tumors of the central nervous system	

Pregnancy-related conditions

Acute fatty liver of pregnancy

Preeclampsia

Management

Patients can be managed as outpatients when PUQE score is less than 13.

In patient management should be considered

1. Continuous nausea and vomiting and inability to keep down anti emetics
2. Continuous nausea and vomiting associated with ketonuria and or weight loss (greater than 5%of body weight) despite oral antiemetics.
3. Confirmed or suspected comorbidity such as urinary tract infection and inability to tolerate oral antibiotics

Treatment

First line antiemetics such as antihistamines (H1 receptor antagonists) and phenothiazines should be prescribed for NVP & HG.

Combination of different drugs should be used in women who do not respond to single antiemetic. For severe or persistent vomiting and HG parenteral or rectal route may be necessary.

Treatment of nausea and vomiting of pregnancy with vitamin B6 (pyridoxine) alone or vitamin B6 (pyridoxine) plus doxylamine in combination is safe and effective and should be considered first-line

Metoclopramide is safe and effective but because of extra pyramidal effects it should be used as second line therapy.

Ondansetron is safe and effective, but as we have limited data ,it should be used as second line therapy.

Corticosteroids should be offered for cases when standard therapies have failed.

Treatment of nausea and vomiting of pregnancy with ginger has shown some beneficial effects in reducing nausea symptoms and can be considered as a nonpharmacologic option.

Hydration

Normal saline with additional potassium chloride, guided by daily monitoring of electrolytes is most appropriate hydration.

Correction of ketosis and vitamin deficiency should be strongly considered. Dextrose and vitamins should be included in the therapy when prolonged vomiting is present, and thiamine should be administered before dextrose infusion to prevent Wernicke encephalopathy.

All therapeutic measures should have been tried before offering termination of a wanted pregnancy

Women with NVP and HG should have an individualised management plan in place when they are discharged from hospital.

Women with severe NVP or HG who have continued symptoms into the late second or the third trimester should be offered serial scans to monitor fetal growth

References

RCOG Green top guidelines on management of Nausea and Vomiting of pregnancy and Hyperemesis Gravidarum

ACUTE PULMONARY EDEMA IN PREGNANCY AND ITS MANAGEMENT – CASE REPORT

Dr. L. LOHA PUSHPANJALI, MD OG

INRODUCTION

Pulmonary edema complicates both low risk as well as high risk pregnancies, but the incidence stands higher in pregnancies complicated by preeclampsia. It is most seen when there is associated medical, surgical, or obstetric complications. Acute pulmonary edema is a medical emergency. This is because of the physiological changes in pregnancy, presence of fetus associated with poorly understood pathophysiology of pregnancy related diseases like pre-eclampsia which has significant morbidity and mortality. Intensive care is required since there may be persistent hypoxemia. This may ultimately result in mortality in around 10% of the cases. Prompt delivery irrespective of the gestational age is the best method of management.

CASE SUMMARY:

A case of 30 years old Primi gravida with gestational age 30 weeks and 5 days with history of neurosensory signs such as headache and blurring of vision, was referred for Preeclampsia complicating pregnancy. She had history of severe anemia corrected with 3 units of blood transfusion. While receiving in the casualty patient had acute breathlessness altered consciousness responding only to painful stimuli. She had generalized anasarca, significant tachycardia, tachypnoea, and desaturation. Her blood pressure was 160/100mmHg with albuminuria. On auscultation bilateral lung crepitations present. Ultrasonography revealed Intrauterine fetal demise corresponding to 25weeks.Echocardiography revealed coronary artery disease with Ejection fraction of 40% and left ventricular dysfunction. Blood investigations revealed elevated inflammatory markers. Diagnosis of Acute pulmonary edema was made.

Patient was put on Noninvasive ventilation and given Diuretics, Steroids, Morphine, Antihypertensives and Antibiotics. After stabilizing the patient,termination of pregnancy was initiated with medical method and dead fetus was expelled. Patient was weaned off Noninvasive ventilation and antibiotics and antihypertensives were continued. Heparin was given. Patient's condition improved and was weaned off oxygen support completely and was discharged on post abortal day 7.

DISCUSSION:

Pulmonary edema is a life-threatening condition that can be associated with preeclampsia. It accounts for only 0.08%. It is more often caused by severe preeclampsia which is associated in 2.9% with pulmonaryedema. Pre-eclampsia is due to abnormal placentation caused by failure of trophoblastic invasion into the myometrial segment of spiral arteries. The resultant local hypoperfusion leads to release of antiangiogenic factors and pro inflammatory cytokines such as soluble form fms like tyrosine kinase 1 (sFlt 1), soluble endoglin (s-Eng), vascular endothelial growthfactor (VEGF) and

placental growth factor (PLGF). These factors stimulate vascular endothelial cells and produce vasoconstriction resulting in preeclampsia and intrauterine growth restriction.

The etiology of ischemic heart disease in pregnant women is like that of non-pregnant women. Risk factors that expose these individuals to ischemic heart disease include hypertension, hyperlipidemia and hypertriglyceridemia, diabetes mellitus, obesity, smoking, and immobility. Many mechanisms have been proposed to explain the pathogenesis of pulmonary oedema in severe pre-eclampsia including hypervolaemia, left ventricular failure and pulmonary capillary leakage. Pulmonary oedema could be due to a combination of these factors. However, it is thought that increased systemic vascular resistance induces significant changes in loading conditions of the ventricular myocardium contributing to diastolic filling abnormalities and to the development of an ischemic substrate with the potential for development of heart failure, pulmonary oedema and/or sudden death.

Immediate management: The occurrence of acute pulmonary oedema in a hypertensive pregnant or recently pregnant woman is a medical emergency and should trigger an emergency response. Treatment aimed at rapidly assembling an experienced team of staff (level 3 evidence). Further deterioration may occur, leading to cardiac arrest, and staff should be prepared to institute advanced life support and consider peri-mortem caesarean section. Transthoracic echocardiography can assist in differentiating a low cardiac output from a high cardiac output state, as well as exclude other important causes of acute pulmonary oedema. Despite the risks of aspiration, non-invasive ventilation should be tried as the initial technique before tracheal intubation, as it provides increased inspired oxygen concentration, displaces fluid from the alveoli into the pulmonary and subsequently systemic circulation, decreases the work of breathing, and decreases the need for tracheal intubation (level 1++ evidence). The use of non-invasive ventilation also avoids the complications associated with tracheal intubation in pregnant or recently pregnant women who are hypertensive, such as intracerebral haemorrhage. Mechanical ventilation strategies incorporating the known cardiorespiratory and metabolic changes of pregnancy need to be considered when ventilating the lungs of a pregnant or recently pregnant woman, as well as the lung protective strategies of low tidal volumes and low peak pressures. Avoidance of aortocaval compression is essential. Urgent reduction of critically high blood pressure with an intravenous antihypertensive agent is necessary. Nitro-glycerine (glyceryl trinitrate) is recommended as the drug of choice in pre-eclampsia associated with pulmonary oedema. Reduction in systolic and diastolic blood pressure should occur at a rate of approximately 30 mmHg over 3–5 min followed by slower reductions to blood pressures of approximately 140/90 mmHg. Intravenous furosemide (bolus 20–40 mg over 2 min) is used to promote venodilation and diuresis, with repeated doses of 40–60 mg after approximately 30 min if there is an inadequate diuretic response (maximum dose 120 mg).

Long-term management: Women who suffer from severe pre-eclampsia and experience acute pulmonary oedema are at increased risk of cardiovascular complications in later life, including hypertension, ischaemic heart disease, stroke and renal disease. They should be closely monitored with control of blood pressure until resolution of the initial disease process and then followed up regularly, with observation for the

long-term complications of the disease. Angiotensin-converting enzymes, whilst contraindicated in pregnancy, are safe to use in the postpartum period. Risk reduction strategies should be offered, such as weight reduction and smoking cessation programs, dietary modification, encouragement of regular exercise and control of hypertension.

CONCLUSION:

Acute pulmonary oedemais a medical emergency leading to morbidity and mortality in pregnant mothers. Therefore, it is important to identify the high-risk patients and keep them under monitoring so that the signs of critical illness can be identified and managed by multidisciplinary team. Risk reduction strategies should focus more onmaintenance of fluid balance and regular clinical observation. Necessary long-term follow up is done to reduce chances of further complications later. Transthoracic echocardiography should be made mandatory in the antenatal period which aids in early diagnosis and management.

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A PICTORIAL ESSAY OF THE UTERINE ANOMALIES ACCORDING TO ESHRE-ESGE CLASSIFICATION

Dr. RAJA ILAVARASI MBBS, MS, DNB OG, FRM

INTRODUCTION

The female reproductive tract develops from the Mullerian duct, which later differentiates into the fallopian tubes, uterus, cervix and the upper part of the vagina. Any deviation from this embryological development of Mullerian ducts can lead to an array of congenital malformations, termed as the "MULLERIAN DUCT ANOMALIES". Mullerian anomalies are prevalent in 6.7% of the general population, 7.3% of the infertile couples and 16.7% of the recurrent miscarriage population[1]. Among the various uterine anomalies, the most common abnormality leading to infertility and recurrent first trimester miscarriages is the septate uterus. There are various methods of classification to classify these anomalies:

- American Society of Reproductive Medicine system (ASRM)
- The embryological-clinical classification system of genito-urinary malformations
- The Vagina, Cervix, Uterus, Adnexae and associated Malformations system based on the tumour nodes metastases (TNM) principle in oncology
- European Society of Human Reproduction and Embryology (ESHRE) and the European Society for Gynaecological Endoscopy (ESGE)

Among these, we will be discussing about the ESHRE – ESGE classification (Fig: 1&2) by the CONUTA (CONgenitalUTerine Anomalies) working group as it is simple, more accurate and very much applicable for the clinical management of these cases as it is based on the uterine anatomy. It also discusses the cervical and vaginal anomalies in independent subclasses. Unlike the ASRM classification by the American fertility society, which uses absolute numbers (ex: indentation of 5mm) as cut-offs to classify an anomaly, ESHRE – ESGE classification uses the uterine wall thickness of the person as the reference standard, and all anomalies are classified with regards to it. The less severe anomalies are placed at the beginning of the classification, while the more severe ones are placed towards the end[2].

As transvaginal ultrasound is the first line of investigation for assessing the uterus and its anomalies, the usefulness of 2D, 3D and 4D (volume) ultrasonography (USG) in diagnosing these anomalies will be discussed in detail in this article[3].

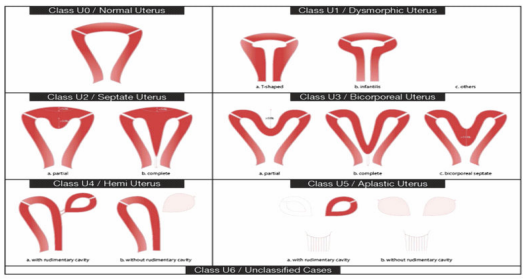

Fig 1: ESHRE/ESGE classification of congenital uterine anomalies: schematic representation (Class U2: internal indentation >50 % of the uterine wall thickness & external contour straight or with indentation 50 % of the uterine wall thickness, Class U3b: width of the fundal indentation at the midline >150 % of the uterine wall thickness). Uterine wall thickness is measured as the distance between the inter-ostial line and the outer margin of fundal myometrium.

	Uterine anomaly		Cervical / Vaginal anomaly	
	Main class	*Sub-class*	*Co-existent class*	
U0	Normal uterus		C0	Normal cervix
U1	Dysmorphic uterus	a. T-shaped b. Infantilis c. Others	C1	Septate cervix
U2	Septate uterus	a. Partial b. Complete	C2	Double "normal" cervix
U3	Bicorporeal uterus	a. Partial b. Complete c. Bicorporeal septate	C3	Unilateral cervical aplasia
U4	Hemi-uterus	a. With rudimentary cavity (communicating or not horn) b. Without rudimentary cavity (horn without cavity / no horn)	C4	Cervical Aplasia
U5	Aplastic	a. With rudimentary cavity (bi- or unilateral horn) b. Without rudimentary cavity (bi- or unilateral uterine remnants / Aplasia)	V0	Normal vagina
			V1	Longitudinal non-obstructing vaginal septum
			V2	Longitudinal obstructing vaginal septum
			V3	Transverse vaginal septum and/or imperforate hymen
			V4	Vaginal aplasia
U6	Unclassified Malformations			
U			*C*	*V*

Fig 2: ESHRE/ESGE classification of female genital tract anomalies

The classification is described as follows:

CLASS U0- NORMAL UTERUS:

- Any uterus with either a straight or curved inter-ostial line.
- With an internal indentation at the fundal midline not more than 50 % of the uterine wall thickness (Fig3).

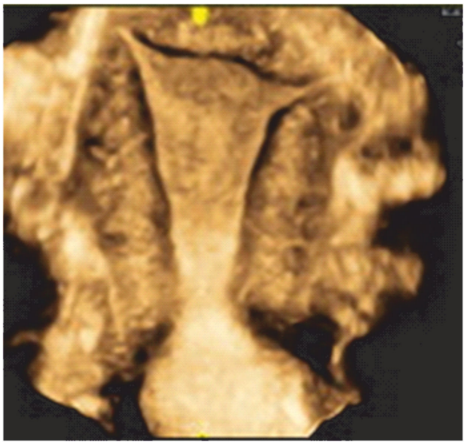

Fig 3: 3D ULTRASONOGRAPHY (USG) OF NORMAL UTERUS

CLASS U1 / DYSMORPHIC UTERUS:

- Normal uterine outline
- Abnormal shape of the uterine cavity excluding septa.

It has three subclasses:

- **Class U1a / T-shaped uterus**: Narrow uterine cavity; thick lateral walls with a 2:1 ratio of the uterine corpus and cervix being maintained (Fig4).
- **Class U1b / uterus infantilis**: Narrow uterine cavity without lateral wall thickening; inverse correlation of 1/3 uterine body and 2/3 cervix (Fig5).
- **Class U1c or others**: All minor deformities of the uterine cavity including those with an inner indentation at the fundal midline level of less than 50 % of the uterine wall thickness (ARCUATE) (Fig6).

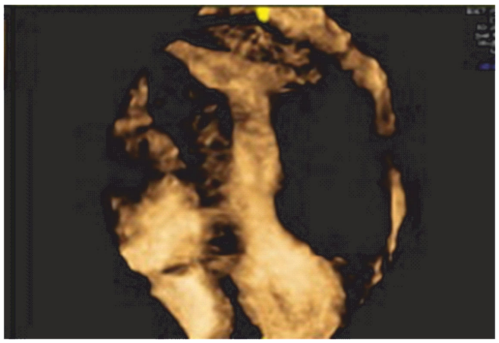

Fig 4:
3D USG OF T-SHAPED UTERUS

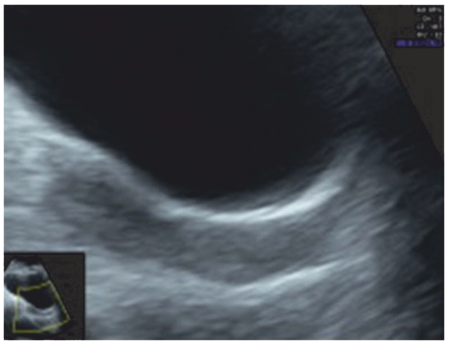

Fig 5:
2D USG OF UTERUS INFANTILIS

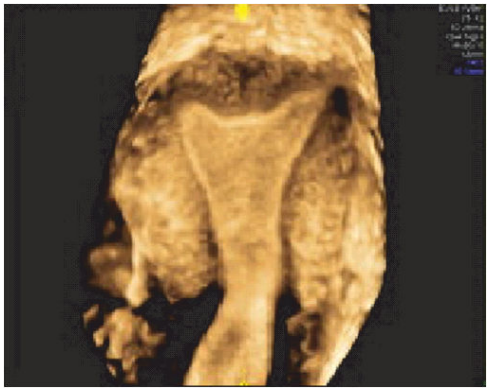

Fig 6: 3D USG OF DYSMORPHIC UTERUS - U1c

CLASS U2 / SEPTATE UTERUS:

- Uterus with a normal fusion of the mullerian ducts but an abnormal absorption of the midline septum.
- Shows a normal fundal outline and an internal indentation at the fundal midline which is more than 50% of the uterine wall thickness.
- This indentation (septum) could divide partially or completely the uterine cavity including in some cases the cervix and/or vagina.

It has two subclasses:

- **Class U2a / partial septate**: Septum divides the uterine cavity (partially) above the level of the internal cervical os (Fig7).
- **Class U2b / complete septate**: Septum divides the uterine cavity completely up to the level of the internal os. May or may not have cervical and/or vaginal septum (Fig8)

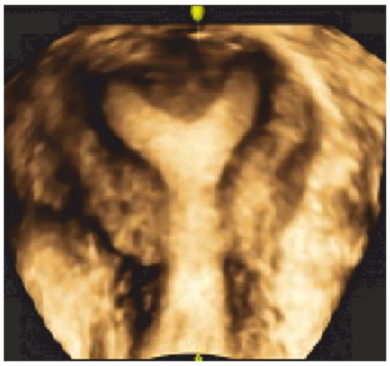

Fig7:
3D USG OF PARTIAL SEPTATE UTERUS

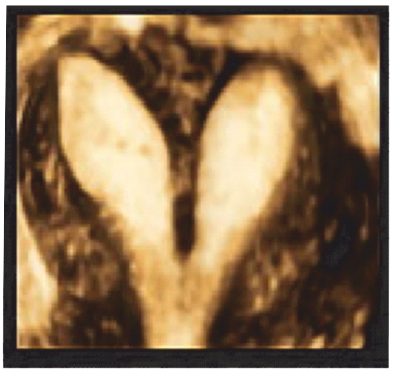

Fig 8:
3D USG OF COMPLETE SEPTATE UTERUS

CLASS U3 / BICORPOREAL UTERUS:

- Fusion defect of the mullerian ducts.
- Uterus has an abnormal fundal outline - an external indentation at the fundal midline which is more than 50% of the uterine wall thickness. This indentation could partially or completely divide the uterine corpus including in some cases the cervix and/or vagina.
- It also has an inner indentation at the midline level that divides the cavity just like the septate uterus.

Class U3 is divided into three sub-classes:

- **Class U3a / partial bicorporeal uterus**: Uterus with an external fundal indentation that partially divides the uterine corpus above the level of the cervix (Fig9).
- **Class U3b / complete bicorporeal uterus**: The external fundal indentation divides the uterine corpus completely up to the level of the cervix. There can be co-existing cervical (e.g. double cervix) and/or vaginal defects (Fig10).
- **Class U3c /bicorporeal septate**: There is presence of an absorption defect in addition to the main fusion defect. The width of the midline fundal indentation is more than150% of the uterine wall thickness (Fig11).

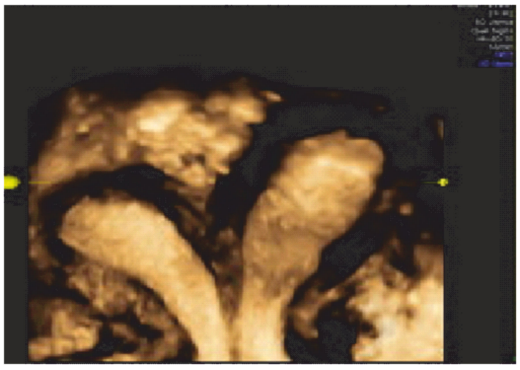

Fig9 :3D USG OF
PARTIAL BICORPOREAL UTERUS

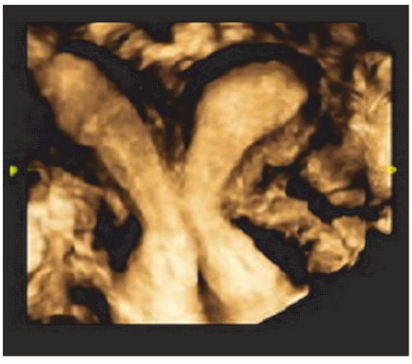

Fig10: 3D USG OF
COMPLETE BICORPOREAL UTERUS

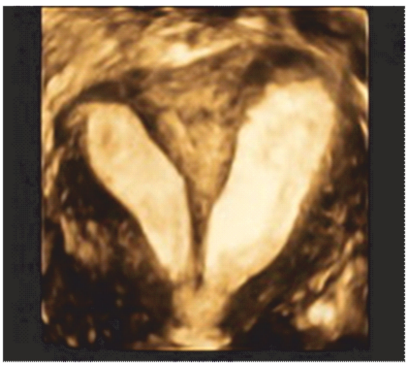

Fig11:
3D USG OFBICORPOREAL SEPTATE UTERUS

CLASS U4 / HEMI-UTERUS:

- Formation defect with unilateral development of functional uterus.
- The contralateral part of the uterus could be either incompletely formed or absent.
  Class U4 is further divided into two sub-classes:
- **Class U4a / hemi-uterus with a rudimentary (functional) cavity**: Uterus with the presence of a communicating or non-communicating functional contralateral horn (Fig 12).
- **Class U4b / hemi-uterus without a rudimentary (functional) cavity**: Uterus with the presence of a non-functional contralateral uterine horn or aplasia of the entire contralateral part of the cavity (Fig13).

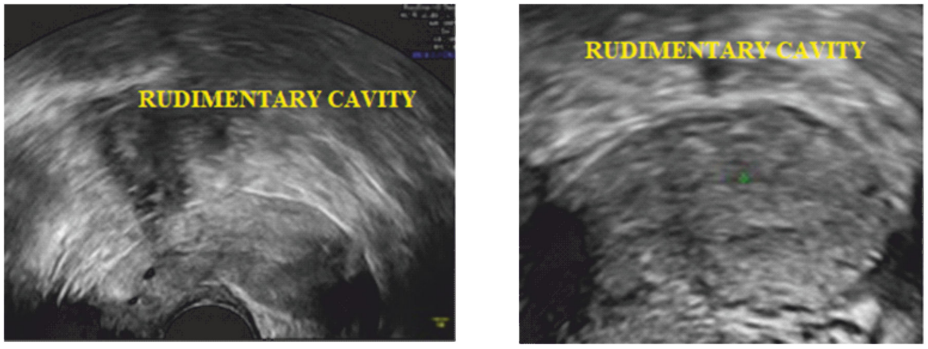

Fig12: 2D USG OF HEMI-UTERUS WITH A RUDIMENTARY CAVITY

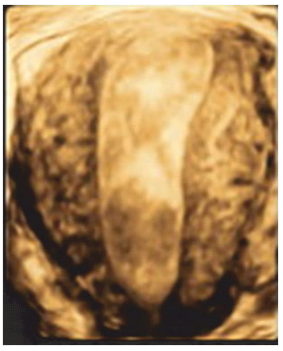

Fig13: 3D USG OF HEMI-UTERUS WITHOUT A RUDIMENTARY CAVITY

CLASS U5 / APLASTIC UTERUS:

- It is a formation defect - absence of fully or unilaterally developed uterine cavity.
- Unilateral or bilateral rudimentary horns with cavity may be present in some cases; while others may have only uterine remnants without cavity.
- Co-existent defects (e.g. vaginal aplasia / Mayer-Rokitansky-Kuster-Hauser syndrome) are common in this group.
  Class U5 is divided into two sub-classes:
- **Class U5a / aplastic uterus with rudimentary (functional) cavity:** Characterized by the presence of uni- or bilateral functional horn.
- **Class U5b / aplastic uterus without a rudimentary (functional) cavity:** Has either uterine remnants or a complete uterine aplasia.

CLASS U6 / UNCLASSIFIED CASES:

- Includes infrequent anomalies, subtle changes or combined pathologies.
- Duplication defects and ectopic mullerian anomalies are included here.

CERVICAL ANOMALIES:

- **SUB-CLASS C0 / NORMAL CERVIX:** Includes all cases of normal cervical development (Fig 14).
- **SUB-CLASS C1 / SEPTATE CERVIX:** Includes cervical absorption defects. It is a normal externally rounded cervix with the presence of a septum (Fig 15).
- **SUB-CLASS C2 / DOUBLE CERVIX:** Includes cervical fusion defects. It is characterized by the presence of two distinct externally rounded cervices (Fig 16); these two cervices can be either fully divided or partially fused. It can be combined with a complete bicorporeal uterus as a Class U3b/C2.
- **SUB-CLASS C3 / UNILATERAL CERVICAL APLASIA:** It includes unilateral cervical development; the contralateral part could be either incompletely formed or absent. Rare anomalies like the complete bicorporeal uterus with unilateral cervical aplasia can be classified as Class U3b/C3.
- **SUB-CLASS C4 / CERVICAL APLASIA:** It is categorized as the complete absence of any cervical tissue or by the presence of severely defective cervical tissue such as the cervical cord, cervical obstruction and cervical fragmentation.

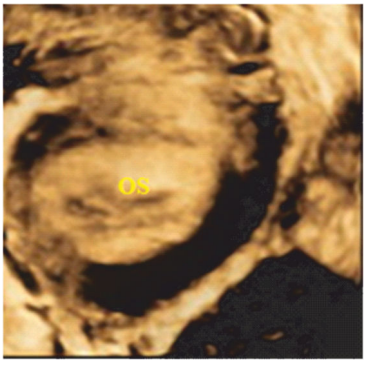

Fig 14: NORMAL CERVIX

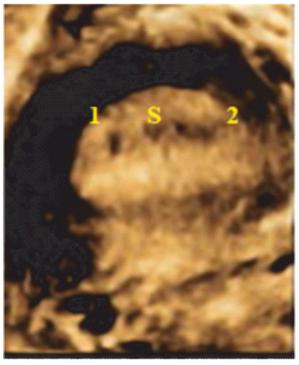

Fig 15: SEPTATE CERVIX; S – septum; 1&2- os

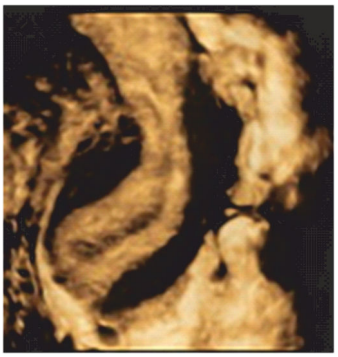

Fig 16: DOUBLE CERVIX

VAGINAL ANOMALIES

- **SUB-CLASS V0:** Normal vagina.
- **SUB-CLASS V1 / LONGITUDINAL NON-OBSTRUCTING VAGINAL SEPTUM**: Variants of septate or bicorporeal uteri together with septate or double cervices & vagina are classified here.
- **SUB-CLASS V2 / LONGITUDINAL OBSTRUCTING VAGINAL SEPTUM:** Obstructing anomalies due to vaginal defects are classified here.
- **SUB-CLASS V3 / TRANSVERSE VAGINAL SEPTUM AND/OR IMPERFORATE HYMEN:** It includes transverse vaginal defects.
- **SUB-CLASS V4 / VAGINAL APLASIA:** Includes complete or partial vaginal aplasia.

CONCLUSION:

This classification system of uterine anomalies is more clinically applicable with increased precision in diagnosing the anomalies, as the cervical and vaginal anomalies are classified separately into sub-classes. The uterine anomalies are more clearly classified here, which will pave the way for further research in this field regarding the diagnosis of these cases with the help of advanced USG technologies and treatment of them.

Image credits: From MS hospital data base.

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GENITAL WARTS IN PREGNANCY -A CASE REPORT

Dr. DHIVYA SETHURAMAN M.D. (OG), **&**
Dr. RAJALAKSHMI RAMALINGAM M.D. (Derm)

INTRODUCTION

Genital warts are the most prevalent form of viral genitalmucosal lesions. They are caused by infection with severaltypes of HPV[1]. Approximately 90% of allgenital warts are related to HPV types 6 and 11.

We present a case report of a24-year-old primigravida with florid Condyloma Accuminata presenting early in the second trimester that was managed in conjunction with theDermatologist.This case report also highlights the importance of multidisciplinary management when treating such lesions.

CASE HISTORY

A 24 year old primigravida presented at 14 weeks gestation to the Obstetrics out patient department with complaints of extensive cauliflower like growths with itching and pain over the genitalia of 2 months duration.She noticed a very small lesion 2 months backwhich increased suddenly in size and extent over the past 2 weeks. She did not have any other urinary symptomsor genital discharge.Patient was married one year back.Her husband also had similar lesions.Both denied pre-marital and extra-marital sexual contact.She was not a diabetic.Obstetric examination was corresponding to the period of gestation.Genital examination revealed multiple genital warts involving fourchette and vaginal mucosa(Figure1), of firm consistency. Vulva and anal region was otherwise normal.VDRL, HIV and HBV serology were non-reactive. Cryotherapy was done once in 15 days for two sittings. Majority of the warts resolved after the second sitting(Figure 2). She isunder regular follow up. Her husband was treated with topical podophyllin.

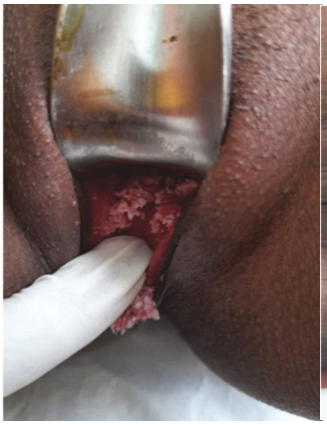

FIGURE 1

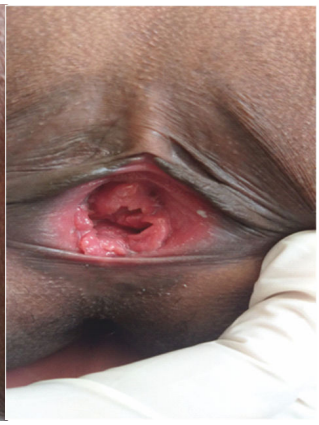

FIGURE 2
(Following Treatment)

DISCUSSION

The infection manifests as verrucous fleshy whitish tored papules that may coalesce into plaques, ranging insize from a few millimetres to several centimetres[2].The warts may be located anywhere in the ano-genitalarea, including the mucosal surfaces[3]. Lesions typically appear within weeks to months after exposure, and they are generally asymptomatic, but may be painful,friable or pruritic[4].

It commonly manifests in the sexually active group. The risk factors for acquiring HPV infection include early age of intercourse[5], high of number of sexual partners and high number of partners' sexual partners[6]. Spontaneous

resolution of HPV infectionis less likely in immunocompromised individuals[7].

The diagnosis is usually made based on the clinical presentation of lesions located on the anogenital area or adjacent areas, such as the mons pubis [6]. Biopsy is generally not performed for the diagnosis of genital wartshowever it may be indicated if the warts appear fixed to underlying structures, refractory to standard therapy, ulcerated or if an individual wart is larger than 1 cm[4].

In pregnancy, genital warts may proliferate and become easily irritated due to the increased vascularity and altered immunity, therefore prophylactic removal might be indicated.

The genital warts may be removed with destructive methods including cryotherapy, surgery or laser[4]. Cryotherapy is one of the methods that is safe, highly successful and can be used during pregnancy. Freezing of each genital wart separately is recommended and the treatment isrepeated every 7 days. A total of seven treatments are allowed. Usually, healing takes 3-4 treatments[8].

Nearly 94% of treated show complete resolution and recurrence occurs in 10% of patients, 1-3 months after treatment. Frequent side effects of cryotherapy are : exudation, swelling, local destruction of the tissue, painful therapy treatments, ulceration and loss of pigment in skin [9].

Trichloroacetic acid can be also used but this is usually more effective for the treatment of moist warts. Podophyllum resin and podophyllotoxin must be avoided, as they areteratogenic. Imiquimod has not been adequately studied in pregnant patients thus should also be avoided [4].

HPV types 6 and 11 have been associated with laryngeal papillomatosis in infants. However, the presence of genital warts is not an indication for caesarean delivery[10]. A caesarean section is indicated only in the rare circumstance of obstruction or bleeding.

There is no cure for genital warts. Administration of Quadrivalent and Nanovalent HPV Vaccine can help preventing genital warts. The goal of treatmentis to eliminate visible lesions. There is no evidence to show that treatment affects the natural course of HPVinfection [11].

CONCLUSION

Early diagnosis and prompt treatment is required for genital warts in pregnancy particularly if it happens in the early stages and shows a tendency for rapid growth.The multidisciplinary approachensures a good outcome.

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MY SHORT STORY

Dr. RAVI STEPHEN DGO.,

A senior medical student in Stanley Medical College fell in love with her classmate. On completing MBBS, they got married and moved to Dalmiapuram, to serve as Factory Doctors in a Cement Factory. They were not happy working there.

In 1953, the lady was offered the post of Medical Superintendent in a dilapidated hospital on the verge of closing down. This was the C.S.I Mission Hospital for Women and Children in Woraiyur. The Managing Committee decided to give the hospital one last chance and this lady doctor decided to take up that challenge.

She was told, “If you can revive and run the hospital, do so, but if you cannot, then close the hospital and hand over the keys “. One of the members of the management committee was Dr. Joseph, the founder of Joseph Eye Hospital, Trichy.

The couple came to Trichy with their one year old son. The second son, 'in utero' at that time, was precious me! My father, Dr. S. S. S. Stephen started his private practice in Lalgudi and later shifted to Woraiyur, Trichy.

I was born in the C.S.I Mission Hospital and grew up in the premises as the Medical Superintendent quarters was within the compound. I used to admire my mother taking care of maternity patients.

I used to see my mother being called to the hospital at odd hours. Throughout my life and till the day she left this earth, I was a witness to her commitment and care for patients.

Those days obstetricians never practised 'Active Management of Labour'. Patients used to be in labour for a few days. Some died due to Atonic PPH.

My mother would be upset and share her anguish with all of us, saying that the patient was talking till she passed away.

Sensing my interest in her work, my mother wanted me to become a doctor. I could not be sponsored for CMC even though she was the Diocesan Medical Secretary.Every year I used to apply to five medical colleges. In the fourth year, I got admission in Thanjavur Medical College through the 10% quota for BSC graduates. My mother was very happy.

As a medical student, I used to come to Trichy during weekends and holidays. Sometimes I assisted my mother in pulling the forceps. She was not strong enough when she was well into her fifties.She was wonderful in applying forceps and I learnt it from her.

And do you know what anaesthesia we used to give? It was 'Open Ether'.One of us would do it!!

In 1981 my wife and I left India to work in Zambia where I learnt my OBG. Based on my training in OG for close to four years. I was selected for DGO in Dublin University, Ireland. I got to know about this DGO through my mother who did a Certificate Course in the same hospital “Rotunda” , when I was in my eleventh standard. My mother had kept the prospectus safe with her and pushed it into my suitcase

when I left for Zambia. But then she died before I could perform my first LSCS in Zambiain 1982.

After completing my DGO, I returned to India and took my mother's place in 1985.

Since then, we have continued handling not only OG patients, but medical cases also.We had so many good things happening to us but I wish to share a few unfortunate experiences that happened in our practice.

CASE 1:

Early in my practice, after a Vaginal delivery, a village women ended up with a Vesico-Vaginal Fistula. Her home started smelling of urine. Being young and bold then, I read up on the surgery and did it myself. It was a success. She conceived again and had her second delivery by LSCS.

CASE 2:

After a Spontaneous Vaginal Delivery, this patient went on to an Atonic PPH. We had to quickly decide and take her on for emergency hysterectomy. In the hurry, one ureter got caught in the ligature. Subsequently the patient went on to Uretro Vaginal Fistula and we were preparing her for surgery. But in three weeks, when the catgut got absorbed – the Ureter opened up and the fistula healed – that was purely God's grace.

CASE 3:

When we started doing Laproscopy, we did an LAVH. Post-operation, there was a big swelling in the vault. I pushed my finger through it and it ended up as a Recto-Vaginal Fistula.

I was upset and anxious and scared as well. This also got healed completely without any intervention. The patient saw my anxiety and used to send me Christmas greetings every year for a few years! Again GOD was merciful to me.

Last but not least… my wife Dr.Rohini, stood by me like a rock supporting me all through, right from the beginning.

Thanks be to God for all His Mercies which are new every morning!

ROBOTIC SURGERY IN GYNAECOLOGY - HAS TIME HAS COME TO REPLACE CONVENTIONAL SURGERY?

Dr. RUBINA SHANAWAZ M.S.(OG)

ABSTRACT

Having been allotted this topic, I would first like to clarify that our definition of "conventional" surgery as we know it is changing day by day, thanks to the rapid technological advances world over. That being said, the basics of anatomy, physiology and surgical principles of open surgery are the foundation on which every innovation in technique of surgery rely on. It is only our approach to alleviate disease via minimally invasive surgery which is evolving with the times!

Laparoscopy created a huge revolution from open surgery ,where with time, even the most complex of cases are being managed completely via laparoscopy, very rarely needing conversion to open surgery.But with greater widespread acceptance of laparoscopic approach, comes the challenge of the steeper learning curve, where a disparity arises in the quality of outcome.Another disadvantage to the laparoscopic surgeon is the strain on the spine and shoulder with increased workload which lead to decreased quality of life.

Here is where Robotics comes in .In this era of rapidly advancing technology at the speed of lightning in every aspect of our daily living, why not make use of technological advances to enhance surgical outcome?Yes, robotic platform does offer superior ergonomics to the surgeon making her/him comfortable during the entire length of the surgical procedure.But that is not all that there is to it.

The immersive 3D along with stereoscopic vision gives the operating surgeon such depth of anatomic visualisation which has never before been experienced.The 10X magnification enhances and delineates tissue planes with greater clarity.

The 360 degree articulation at the instrument tip along with added extra length conferred by the ability of the instruments to slide in and out of the robotic arms makes the surgeon fall in love with the Art of surgery all over again, thanks to the dexterity enhancement.

Filtration of tremors is also an added advantage especially when operating back to back.

In an operation theatre setting where assistance is frequently changing, Robotics confers the huge advantage of constant camera stability ,which is an essential cornerstone of endoscopic surgery, as anyone who has performed laparoscopic surgery will vouch for.

Thanks to all these advances, the learning curve also becomes significantly shorter .

What advantage does it confer to the patient?

The same advantage of having had a highly complex surgery being performed by a minimally invasive approach is transferred to the patient as well. Especially the advantage of a

highly precise surgery being performed with minimal collateral damage .By this I refer to complications due to injury to the other organs, which we all know is our primary fear when operating on complex cases.

The post operative pain is also significantly reduced, thanks to the fulcrum effect which is at the abdominal wall level with laparoscopic surgery moving near the target organ with robotic surgery.

All these factors contribute to a very short hospital stay with very few readmission rates.

Where is Robotic Surgery indicated in gynaecology ?

Worldover, after oncology and urology, gynaecology is the speciality where most number of Robotic surgeries are being performed .

In benign gynecology, Robotics comes as a boon in patients who have had multiple previous surgeries where we expect severe adhesions.In patients with endometriosis ,where the spread to the adjacent pelvic organs is increasingly becoming common,Robotics helps a great degree in giving maximal clearance with minimal collateral damage .

In gynaec oncology where extensive dissection especially in the lateral pelvic wall is needed, the enhanced depth of vision and magnification conferred by Robotics confers a great advantage in achieving maximal oncological clearance.

In Urogynaecology ,where dissection deep into pelvis is performed as with Sacrocolpopexy and where there is extensive adhesions post hysterectomies with vault prolapse and fistula repairs,Robotics confers significant advantage in performing a satisfactory repair .

The barriers to widespread use of Robotics are the cost and length of surgery.With the advent of newer and more adaptable models coming in,it is only a matter of time before we cross these hurdles as well.

Thus,the time has definitely come for us to stop looking at Robotics as a luxurious toy and incorporate Robotics as a necessary tool in the armamentarium of every surgeon.

UTERINE MYOMA AND THROMBOEMBOLISM - A RARE SCENARIO

Dr. SUDHA SHANTHI MASILAMANI

BACKGROUND

Venous thromboembolism (VTE) is a challenging complication in patients with fibroids due to the increased risk of bleeding with anticoagulation, especially in the setting of associated menorrhagia. The incidence of deep vein thrombosis (DVT) is increased with higher uterine weight which may be related to the extrinsic venous compression of the iliac veins or the inferior vena cava. However, there is reported discordance between the site of uterine fibroid and the site of the DVT along with pulmonary embolism (PE) without evidence of lower extremity DVT suggesting an underlying hypercoagulable state that is more complex than mere hormonal and mechanical factors[1]. Incidence of pulmonary embolism and deep vein thrombosis with uterine myoma is rare[2]. There have been few reports on this association. These concomitant finding occurred in a woman with no other risk factors for development of deep vein thrombosis/pulmonary embolism. The risk factor for deep vein thrombosis are those patient with heart failure, malignant, pregnancy, prolonged bed rest, obesity, long haul air travel, use of OCP and so on[3] . Here we present our lady whose large myoma was the sole identified risk factor for the development of thromboembolism. Our patient was successfully treated with anti coagulants and total hysterectomy.

CLINICAL CASE:

39 year old nulli gravida had syncope and progressive dyspnoea. She was hospitalised and evaluated. Her D dimer was elevated. CTPA showed embolism in right distal pulmonary artery. Doppler of lower limb showed acute thrombus in the left common femoral vein. She had no other risk factor for thromboembolism.

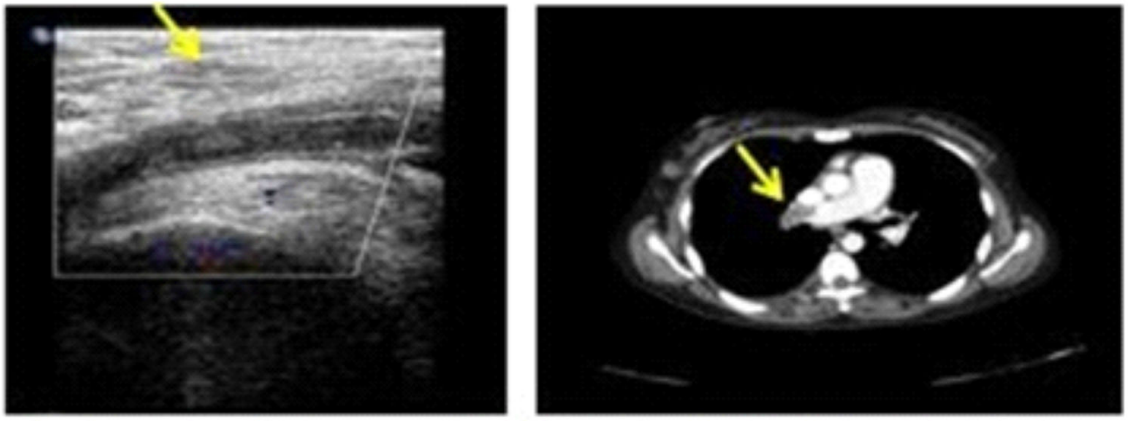

Fig 1a. Venous Doppler of left common femoral vein demonstrating thrombus(hyperchoic) (yellow arrow) Fig 1b. CT pulmonary angiography demonstrating thrombus in right pulmonary artery.(yellow arrow)

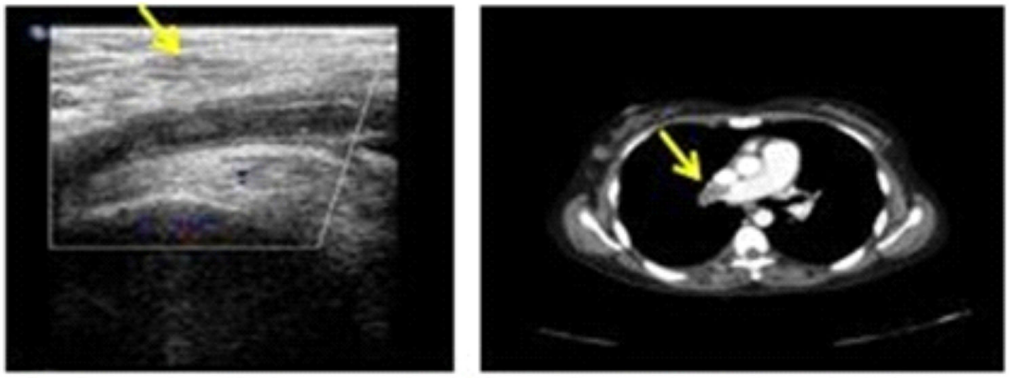

Fig 1a. Venous Doppler of left common femoral vein demonstrating thrombus(hyperchoic) (yellow arrow) Fig 1b. CT pulmonary angiography demonstrating thrombus in right pulmonary artery.(yellow arrow)

She was thrombolysed and treated with parentral and oral anticoagulants. Examination revealed a large pelvic mass confirmed to be uterine myoma by ultrasound examination. Only positive symptoms were dysmenorrhoea for past 3 years and menorrhagia for past 6 months. Her haemoglobin level dropped and treated with 3 units of packed cell transfusion Total abdominal hysterectomy with left salphingo oophorectomy done. Per op findings- uniformly enlarged to 24 weeks size and left ovary was cystic. Hysterectomy and post op period went uneventful patient on follow up.

DISCUSSION :

In classic triad of Virchow[4] which includes stasis of blood flow, disruption of vascular endothelium, and hyper coagulability have all been recognized as a risk for developing for deep vein thrombosis. Many cases of intra peritoneal organ compression by large myoma noted. It depends on the size, location of myoma. large tumors can cause pelvic vein congestion. Fibroid uteri may be an independent risk factor for developing a DVT in women ages 18 to 65 years old. These patients should be risk-stratified and appropriate chemoprophylaxis should be considered in an attempt to prevent thrombotic events.[5]

CONCLUSION :

Though pulmonary embolism and deep vein thrombosis in myoma is a rare event, it can be managed successfully with anticoagulants and hysterectomy.

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URINARY TRACT INFECTIONS IN PREGNANCY

Dr. AKILA V M.D.(OG).,

UTI is frequently encountered in pregnant women. Changes in the urinary tract and immunological changes of pregnancy predispose women to UTI. The physiological dilatation of ureters and renal calyces are due to compression of the gravid uterus and smooth muscle relaxation caused by the progesterone hormone. Decreased bladder capacity and increased vesico urethral reflex are also noticed. Pregnancy by itself is a state of relative immune-compromise. All those put together increases the incidence of UTI in pregnancy.

This smooth muscle tone reduction causes slowing of urethral peristalsis and urethral sphincter relaxation. Pregnancy-specific biochemical changes in urine –with high art of glucose amino acids and hormone degradation produces increases the urinary PH

EPIDIMEOLOGY:

Prevalence of Asymptomatic bacteria (ASB) varies between 2% to 18.5% . Acute cystitis is observed in 1-4%. Acute pyelonephritis ranges from 0.5-2%.

Risk factors for UTI during pregnancy are lower socio-economic status, sexual activity, older age, multiparity, anatomical urinary tract abnormalities sickle cell anaemia and diabetes are some of them. History of pre-pregnancy UTI is a strong predictor of pyelonephritis after 20 weeks of gestation.

CLASSIFICATION OF UTI	Asymptomatic bacteriuria
	Acute Cystitis
	Acute Pyelonephritis

Asymptomatic Bacteriuria :

6% of Pregnant women, suffer from ASB Patient does not have symptoms, hence to screening is important.The serious consequence of ASB when not treated risk of pyelonephritis is high (30-40% vs 3-4 % in treated population) Rrisk of premature birth and lower birth weight infants are also higher in ASB.

A clean catch midstream urine for urine analysis and culture in reliable test to identify bacteriuria. Routine midstream urine for culture and sensitivity in early pregnancy to screen for ASB is Grade A recommendation by RCOG. Treatment for Asymmetrical Bacteriuria is short for 5 -7 days – oral antibiotics.

Diagnosis and treatment of asymptomatic bacteriuria (ASB) and acute cystitis/urethritis (doses for normal renal function)

	Asymptomatic bacteriuria	Acute cystitis/urethritis
Screening (obligatory)	1st prenatal visit or 12–16 HBD	
First line treatment	Amoxicillin 500 mg every 8–12 h for 3–7 days	For 7 days
	Cephalexin 500 mg every 12/6 h for 3–7 days	For 7 days
FDA cat. B	Amoxicillin/clavulanic acid 500 mg every 12 h – for 3–7 days	For 7 days
	Nitrofurantoin 100 mg every 12 h for 5–7 days	For 7 days
	Cefuroxime 250 mg every 12 h – for 3–7 days	For 7 days
	Cefpodoxime 100 mg every 12 h	
FDA cat. C	Trimethoprim with sulfamethoxazole 960 mg every 12 h for 5 days	For 7 days

*Treatment limited to the 2nd and 3rd trimester (except last 2 weeks); should not be used in the 1st trimester if other first line agents may be administered.

All Pregnant women with Assymptomatic Bacteriuria, should be screened after therapy. 1/3 of them will experience recurrent infection. Follow up culture should be done 1-2 weeks after treatment and then after 1 month.

Acute cystitis – occurs in 1%. Presents with symptoms like dysuria pregnancy, urgency and suprapubic pain but no systemic illness, 30% of women with ASB develop cystitis during their pregnancy – Treatment is as same as Asymmetrical Bacteriuria.

In these women, its important to do a clinical examination to rule out vulvitis, vaginitis and cervicitis Pyelo nephritis –incidence is nearly 2% associated with perinatal complications and occurs in II and IIIrd trimester.

When symptoms of cystitis is associated with systemic illness- such as pyrexia, rigor, nausea, vomiting and tenderness in the renal angle diagnosis of pyelonephritis should be made.

Diagnosis and treatment of acute pyelonephritis (doses for normal renal function)

Diagnosis	Symptoms+urine culture: Fever > 38°C, lumbar pain, skeletal and joint pains, nausea/vomiting with or without accompanying dysuria, polyuria $\geq 10^5$ CFU/ml in mid-stream urine specimen
Mild or moderate acute pyelonephritis	Ceftriaxone 1 g every 24 h Cefepime 1 g every 24 h Amoxicillin with clavulanic acid 1.2 g every 12 h Aztreonam 1 g every 8–12 h
Severe acute pyelonephritis/ immunosuppression/ urinary stasis	Ticarcillin with clavulanic acid 3.1 g every 6 h Piperacillin with tazobactam 3.375 g every 6 h Meropenem 0.5 g every 8 h Ertapenem 1 g every 24 h Doripenem 1 g every 8 h

Recurrent infections: 4-5% of pregnancies risk of pyelo nephritis should be kept in mind. Long term, low dose antimicrobial therapy is advocated for the reminder of pregnancy.

Antimicrobials in pregnancy

Increased glomerular filtration rate results in quick elimination of renally excreted medications. Increased plasma volume, reduces the drug concentration hence the bioavailability is low. It is essential to remember potential maternal side effects and possibility of teratogenicity before choosing he effective drug. Nearly all antibiotics cross placenta only a few exert teratogene effect. US FDA-category B drug –cephalosporins are the commonly accepted drugs in all trimesters.

UTI in pregnancy pose a clinical problem and a great challenge for physicians.The antibiotic chosen should have a good maternal and fetal safety profile.

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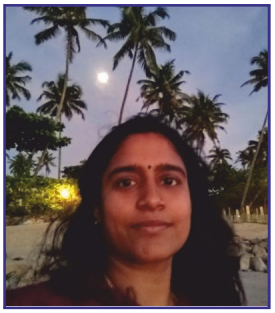
SPAGHETTI IN PINK SAUCE



Dr. THAMIZHSELVI NAVEEN, MD(OG), DNB (OG),

Pasta is one dish that is liked by the kids universally. This is an Indianized version of the classic Italian spaghetti in marinara sauce. This is to make the kids eat all veggies without any whining. It really is yummilicious and loaded with calories, that's a warning for all diet conscious people. But once in a while they too can dig in, to have a wholesome heartful meal. I have given the vegetarian recipe for a family. Non vegetarian version call for less veggies and more minced meat to the recipe.I have added the ingredients required and cooking time for the non-vegetarian recipe in the post script. Thanks in advance.

INGREDIENTS

For 6 servings

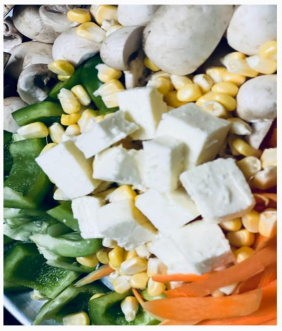
- ❖ Spaghetti 450 g (durum wheat)
- ❖1 cup Cheese (parmesan/cheddar/mozzarella)

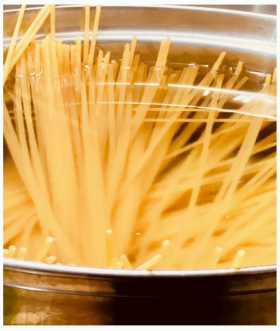
PINK SAUCE

- ❖ 1/3 cup Olive oil (100 ml)
- ❖ 3 large onions, finely chopped
- ❖ 12 cloves garlic, minced
- ❖ salt, to taste
- ❖ pepper, to taste
- ❖ 10 dried red chillies
- ❖12 medium tomatoes, finely chopped
- ❖ Fresh cream (50 ml)
- ❖ 1 tablespoon dried basil
- ❖ 1 tablespoon dried oregano
- ❖1 tablespoon dried parsley

VEGGIES

- ❖ 1 cup mushroom (cut into halves)
- ❖ 1 cup carrot (julienned)
- ❖ 1 cup sweet corn (boiled)
- ❖ ½ cup cabbage (julienned)
- ❖1 cup bell pepper (red,green and yellow,cut into squares)
- ❖ ½ cup paneer (diced)
- ❖ Black and green olives 10 nos., sliced.





PREPARATION

1. Bring a large pot of water to boil and add a generous pinch of salt. (Whole durum wheat pasta already has salt in it. Hence add a little less salt to the water when it's used). Cook the pasta according to the package instructions, usually 8-10 minutes, until soft and completely cooked. Stir occasionally to keep the spaghetti from sticking together.

Make the pink sauce:

2. Heat the olive oil in a large saucepan over medium heat. Add the onions and garlic and season with salt and pepper. Cook until soft and caramelized, about 15 minutes.
3. Grind the dried red chillies in a mixer with 2 tomatoes cut into larger pieces to a coarse paste. Other tomatoes can be finely chopped. Alternatively, all the tomatoes can be ground coarsely and added to the sauce.
4. Stir the chopped tomatoes and ground tomatoes with red chillies into the onions and sauté for 2-3 minutes.Reduce the heat to low, cover, and simmer for at least 15 minutes.Wait for the oil to be released over the sides of the pan.
5. Add the dried basil, dried oregano, and dried parsley.
6. Add all the veggies and allow the sauce to cook for another 5 minutes.
7. The sauce should be of pouring consistency. Adjust the salt according to the taste.
8. Add 50 ml of cream to the sauce. It gives the nice pink colour to the sauce.
9. Drain the pasta and add it directly to the simmering pink sauce. Reserve some pasta water, in case you need it to loosen the sauce. Stir the pasta into the sauce until fully coated.
10. Add cheese to the pasta and give a nice coating to the spaghetti before serving it. Parmesan cheese is ideal, if not available, cheddar can be used. Mozzarella gives the pasta a nice gooey consistency which would be loved by the kids.
11. Add the spaghetti to the serving bowls, top it with extra cheese if required and garnish it with fresh basil if available, and also somecracked pepper for an extra punch of spice.
12. Enjoy!

P.S: For the Non-vegetarian version, 1 cup of minced meat (poultry or lamb) can be added to the onions before adding the tomatoes. It has to be sautéed for 5 minutes and to be covered and cooked for 15 minutes before adding the tomatoes.

A RARE CASE OF RED CELL APLASIA

Dr. PUNITHA RAJESH M.D (OG).,

A 32 yr old Primi gravida with DCDA twin gestation, known case of Red cell Aplasia (diagnosed in1990 during her early infancy), conceived with ART(DO, ET done on 23.09.2021) at Chennai.

Had regular ANC at Chennai and got booked herself for ANC at 28 weeks at SreeDevi Hospital & Fertility Centre, Trichy on 21.03.2022

H/o several blood transfusions, IV iron and and Inj.B12 at Chennai since the confirmation of pregnancy.

H/o PC transfusions during infancy and no history of any transfusion after infancy till pregnancy.

Regular menstrual cycles(5/25) since menarche at the age of 14.

Married since 5years.

No consanguinity.

H/o ART once with self oocytes and was adviced DO at Chennai.

Hb% was 5.6gm/dl and peripheral smear showed Dimorphicanaemia and relative neutrophilia on the day of booking. Haematologist's opinion was sought. AN corticosteroids given. PC transfusions given till 36weeks whenever the Hb % was <9gm/dl.

Elective LSCS was done on15.05.2022.

3 units PC transfused peri operatively.

Patient developed postpartum PIH.

Discharged on 19.05.2022 with Hb% of 10.5gm/dl and was on 2anti hypertensive drugs with the advice of regular follow up.

Pure red cell aplasia (PRCA) is a rare disorder that designates anemia secondary to failure of erythropoiesis. It is characterized by normocytic, normochromic anemia, which is associated with reticulocytopenia in the peripheral blood and absent or infrequent erythroblasts in the bone marrow.

ETIOLOGY:

CONGENITAL PRCA

Diamond-Blackfan or Blackfan-Diamond syndrome

Acquired PRC

1. Autoimmune/ Collagen disorders - systemic lupus erythematosus, rheumatoid arthritis, inflammatory bowel disease.

   Leukemias

2. Lymphoproliferative disorders

   Chronic lymphocytic leukemia (CLL) (common)

   Large granular lymphocytic leukemia (LGL)(common)

   Hodgkin disease

   Non-Hodgkin lymphoma

   Multiple myeloma

   Castleman disease

   Waldenstrom macroglobulinemia

   ABO-incompatible stem cell transplant

SOLID TUMORS

Thymoma

Breast

Biliary

Gastric

Lung

Thyroid

Renal cell

Carcinoma of unknown origin

Virus

Parvovirus B19 (most common) - can lead to transient aplastic crises

Human immunodeficiency virus (HIV)

T-cell leukemia-lymphoma virus, Epstein-Barr virus (EBV)

Hepatitis A, B, C, and E

Cytomegalovirus

Bacterial infections - group C

streptococcus, tuberculosis, bacterial sepsis

Drugs - recombinant erythropoietin (rhEPO) is most common.

Pregnancy

The peripheral smear demonstrates normocytic normochromic anemia with reticulocytopenia. The white cell count and platelet count are normal in number and morphology. The histological picture seen on bone marrow examination depends on the cause of PRCA. Complete absence or near absence of erythroblasts (less than 1% erythroblasts on marrow differential count) from an otherwise normal marrow is a characteristic of autoimmune PRCA. Rarely, a few erythroblasts or basophilic erythroblasts are present, but their number never exceeds 5% of the differential count.

In patients with B19 parvovirus infection, large proerythroblasts with vacuolated cytoplasm and pseudopodia (giant pronormoblasts) appear on the marrow exam; however, this is not a diagnostic finding.

Lymphoid aggregates along with plasmacytosis and lymphocytes point to an inflammatory reaction. Any signs of hypercellularity, presence of ringed sideroblasts, or dysplastic features that extend beyond one cell line are suggestive of a myelodysplastic variant of PRCA or MDS itself.

There are no specific signs or symptoms associated with PRCA. The most common presentation is the same as that of anemia. Generalized fatigue, decreased exercise tolerance, palpitations, and in extreme cases, presyncope or syncope (when associated with cardiac stress due to increased work of function). The physical exam is also non-specific. Pallor is a feature in all patients. A thorough skin exam is necessary to look for erythema infectiosum, which correlates with parvovirus B19. A prodromal rash is sometimes present in children where a reticular and lacy body eruption is visible. The systemic examination should look for swollen lymph nodes, hepatomegaly, and splenomegaly. None of these signs or symptoms are definitively diagnostic of PRCA by themselves but will provide vital clues in establishing etiology.

Diamond-Blackfan syndrome is associated with physical anomalies in up to a third of all patients.

Craniofacial dysmorphism and thumb abnormalities are classic in DBA.

A classic description is that of Cathie: “tow-colored hair, snub nose, wide-set eyes, thick upper lips, and an intelligent expression.”

Aase and Smith described the triphalangeal thumb abnormality along with anemia seen in DBA.

TREATMENT / MANAGEMENT
INHERITED PRCA

Untreated inherited PRCA results in severe anemia, which leads to congestive heart failure and death. Glucocorticoid use, blood transfusion, and allogeneic stem cell transplant (ASCT) are the mainstay of treatment in children.

Any patient presenting with anemia and reticulocytopenia requires evaluation for PRCA. The low reticulocyte count helps differentiate PRCA from hemolytic anemia (which can also have isolated anemia, albeit with reticulocytosis).

The prognosis depends on the etiology of PRCA.

Review of literature shows cases of red cell aplasia caused by pregnancy. There is no documented evidence of pregnancy in a patient with known pure red cell aplasia so far.

JUST FOR FUN !!

At the Hotel Bar ...

A man was sitting at a hotel bar, when a group of men sat down next to him and ordered a round of drinks.

"You guys with a convention?"

"Yes, we're with the Gynaecology conference"

"Really? I was this close" he holds up his finger and thumb about an inch apart "to becoming a Gynaecologist."

"So what did you end up doing?"

"I'm a proctologist."

What do you call a gynaecologist who really loves her job?

Ovary Enthusiastic

Lady: My husband just swallowed an Aspirin by mistake, what shall I do?

Dr: "Give him a headache now, why waste medicine!"

How is a hospital gown like insurance?

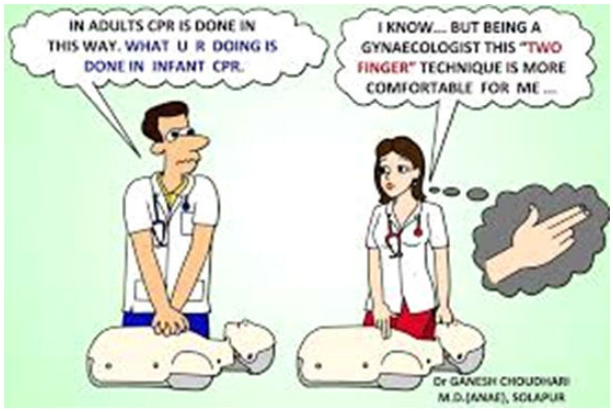
You're never covered as much as you think you are.

I've got a disease where I can't stop telling airport jokes. The doctor says its terminal.

A doctor accidentally prescribes his patient a laxative instead of cough syrup.
Three days later the patient comes for a check-up and the doctor asks,
"Well? Are you still coughing?"
The patient replies, "No, I'm afraid to."

Patient: "Doctor, are the test results ready yet? I'm dying of curiosity!"
Doctor: "Heh… not only from curiosity."

Patient: "Someone decided to graffiti my house last night!"
Doctor: "So why are you telling me?"
Patient: "I can't understand the writing. Was it you?"



Doctor: "Quick, he's losing a lot of blood. He needs an infusion — what's his blood type?!"
Nurse: "B positive."
Doctor: "I'm trying, but he's lost a lot of blood."

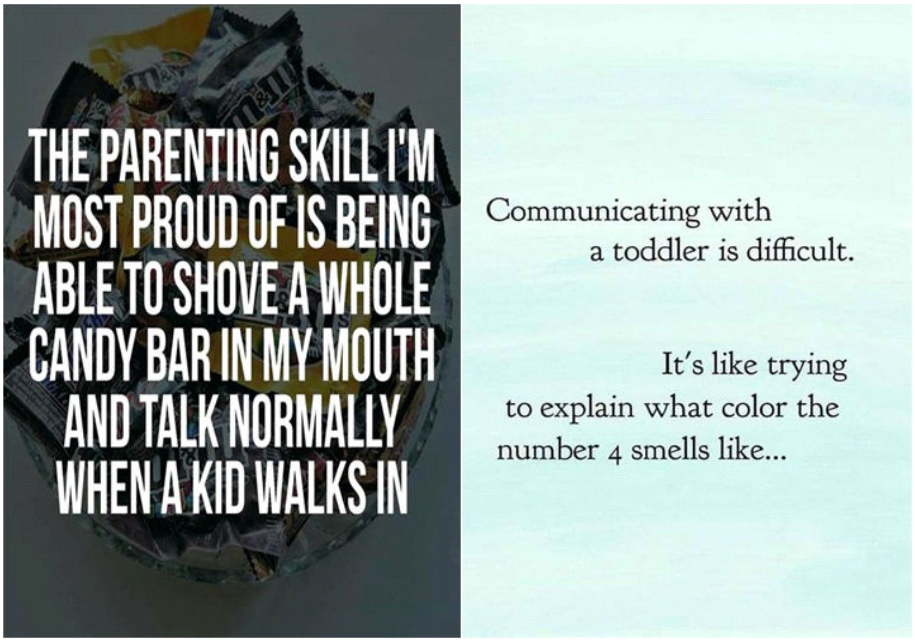
At the Birth
"Will the father be present during the birth?" asked the obstetrician.
"Nah," replied the mother-to-be.
"He and my husband don't get along."

Why did the stand-up comic quit comedy to become a obstetrician?
He needed to work on his delivery.

How does the receptionist at a urology department answer the phone?
“Urology office— can you hold?”

I've never vaccinated any of my kids.
I just pay the paediatrician to do it.

Inside Parents' Mind



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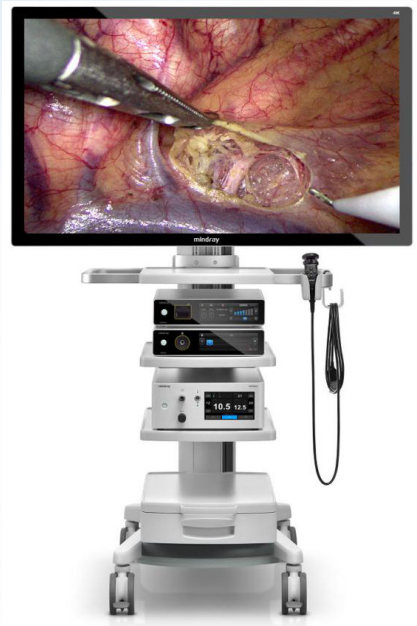

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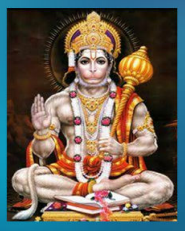

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Social Security Scheme

of

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To,
All Members of
FOGSI

Sub : FOGSI – Social Security Scheme.

Dear Members,

We are glad to inform you that FOGSI has launched a Social Security Scheme (SSS) for its members.

The important feature of this scheme is that each member contributes Rs.100/- for every unfortunate death of the fellow member. On submission of Death Certificate by the nominee to the FOGSI Office, the office will dispatch the cheque to the nominee within 15 days without asking the cause of death. This money will be of great use to the nominee to complete the formalities / rituals after the unfortunate's demise of their beloved.

The fraternity amount will be directly proportional to the strength of SSS Membership. More the members, larger the amount will be available to the nominee.

Thus we request all the FOGSI members to join the scheme, so that a handsome amount is gifted to the nominee upon their sad demise. If you are already a member than motivate others to join the scheme.

With regards,

Thanking you,

Yours sincerely,

Dr.Jaydeep Tank
Deputy Secretary General
FOGSI

Encl : Application form + Details.

FOGSI – Social Security Scheme : Salient Features

Membership Criteria

1. Life / Ordinary member of a constituent society of FOGSI for not less than three consecutive years at the time of joining this scheme.
2. Membership of this scheme shall be subject to continuously being an active member of FOGSI, throughout the duration of membership of the FOGSI Social Security Scheme.
3. This must be accompanied by an endorsement by the President / Secretary of the member society to which the applicant belongs.

Age Criteria

1. The age will be considered as age in completed years on the date of encashment of the draft / cheque received.
2. Proof of age must be attached with application.

Admission Fee

Age	Rate
At or below 30 years	Rs.1,000/-
Between 31 and 40 years	Rs.2,000/-
Between 41 and 50 years	Rs.3,000/-
Between 51 and 60 years	Rs.5,000/-
Above 60 years	Rs.10,000/-

Membership Fee

Every member of this scheme shall pay Rs.100/- as membership fees. The membership fee is non-refundable.

Advance Fraternity Contribution

Every member has to pay initially Rs.1,500/- as A.F.C. along with the admission fee and membership fee, which will be adjusted as Rs.100/- (Death Fraternity Contribution) per death of member during the year.

Fraternity Benefit

1. Benefit of fraternity contribution of the scheme is available to nominees of scheme members after completion of two years of membership of FOGSI S.S.S. An exception is made to this clause in the first two years of this scheme.
2. If the death of a member occurs in an accident after joining the scheme, the nominee has to present the police file number and post mortem report.
3. On receipt of information from a nominee about the death of the member, his/her nominee shall be paid the fraternity contribution as per entitlement under this scheme and the balance if any lying in A.F.C. account of member.

Death Fraternity Contribution (D.F.C.)

1. Every member of the scheme shall contribute D.F.C. of Rs.100/- in the event of death of a member.
2. This amount shall be adjusted against the A.F.C. during the year.
3. Out of the above amount, Rs. 90/- shall be paid to the nominee of deceased member, with Rs. 10/- being retained by this scheme, for utilization for administrative expenses and to bridge any problem shortfall in overall D.F.C. collection.

Mode of Payment

The payment is to be made by cheque / draft payable in Mumbai, drawn in favour of **"FOGSI–S.S.S."**.

Details about the Payment

Age	Admission Fee	Membership Fee	Advance Fraternity Contribution	Total Amount
At or below 30 years	Rs.1,000/-	Rs.100/-	Rs.1,500/-	Rs.2,600/-
31 and 40 years	Rs.2,000/-	Rs.100/-	Rs.1,500/-	Rs.3,600/-
41 and 50 years	Rs.3,000/-	Rs.100/-	Rs.1,500/-	Rs.4,600/-
51 and 60 years	Rs.5,000/-	Rs.100/-	Rs.1,500/-	Rs.6,600/-
Above 60 years	Rs.10,000/-	Rs.100/-	Rs.1,500/-	Rs.11,600/-

Every member of the scheme shall contribute D.F.C. of Rs.100/- in the event of death of a member.

** If a member furnishes any wrongful information in the application form or at any time during the membership term, the Managing Committee of this scheme shall have the right to terminate the membership of the member concerned without any benefit.*

The Federation of Obstetric & Gynaecological Societies of India
C-5,6,7,12,13, 1st Floor, D-Wing Entrance, Trade World, Kamala City,
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*Tel : +91-022-24951648, 24951654
*Email : fogsischemes@gmail.com *Website : www.fogsi.org

Membership Form
FOGSI – Social Security Scheme

Personal Details :-

Name : _____

Age : _____ Date of Birth : _____ Sex : _____

Address: _____

_____

City : _____ Pincode : _____ State : _____

Contact Numbers : _____ Email : _____

Name of the Parent Society : _____

Life Member : Yes / No Duration of Membership : _____ years

Nomination Details : -

Name of the Nominee : _____ Relationship : _____

(Full name)

Alternate Nominee : _____ Relationship : _____

(Full name)

Membership Contribution :-

Admission Fees(As recommended): Rs._____ *(Please attached age proof).*
Membership Fee : Rs.100/-
Advance Fraternity Contribution : Rs.1,500/-

Total Amount Paid : Rs. _____ by Cheque / Demand Draft No._____

dated _____ Bank : _____

_____
Signature of Applicant

To be filled by the Member Society (Certificate by the Member Society)

This is to Certify that Dr._____ is a continuous active Member of the Society for the last _____ years.

Seal of the Society

_____
Signature of the President / Secretary

For Office Use Only

FOGSI Membership No. : _____ Application No._____

Recipt No. _____ dated _____

Office Superintendent **Treasurer**

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