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CARING FOR WOMEN'S HEALTH : EVIDENCE, ATTITUDE & PRACTICE

Dedicated Issue:
Critical Care Obstetrics (Part-2)

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From the President's Pen

Welcome all the AOGD members!

I bring greetings from AOGD. The weather is changing and the autumn is round the corner. The festivals will begin soon. As well as our academic festivities are also all set to begin in near future. All our AOGD members will get the flavor of well-crafted and designed virtual, unique academic activities spread over a span of fifteen days. We are innovating, enthusiastically towards newer horizons, making experiments and striding untouched arenas. We are sure all our members will get once a lifetime experience to remember and cherish. Our core team – Dr. Kanika Jain and Dr. Mamta Dagar are really working very hard and literally burning midnight oil for the success of this mega event. With perfection, both of them keep a sharp eye on each and every major and minor details pertaining to this virtual conference. Ofcourse, unity is strength, proved again and again by both of them.

We are very proud that our AOGD members have bagged so many prizes from FOGSI events. Of course, we are very lucky to have so many talented, enthusiastic senior as well as junior members in our society. It gives us an immense pride when our AOGD members are acknowledged in a Pan-India competition. We have variety of academicians and clinicians who excel in their respective fields. All their talents have shown in those competitions of FOGSI like all previous years.

We have this bulletin dedicated to Critical Care Obstetrics. Dr. Preeti and Dr. Niharika Dhiman have nicely shared their experiences as "Lessons learnt so far from Maternal Mortility due to COVID-19". Dr. Sunita Malik has dealt with "Peripartum Cardiomyopathy" in details. We have our neurologist Dr. Rajeev Ranjan taking care of "Eclampsia" and guiding us. "Thyroid Storm and Diabetic ketoacidosis in Pregnancy: An algorithm" is nicely tackled by our endocrinologist Dr. Surendra Kumar. Finally, Dr. Jyoti Kotwal has taken care of "Coagulation derangement in pregnancy affected with COVID-19".

Yes, this pandemic has challenged the entire world. Yet, we have to face the challenges head on and continue with endeavor of clinical work as well as academic activities. We continue in our goals very energetically and enthusiastically. We know these areas of innovation were never explored in the past. Because such a pandemic had not struck the world in near past. But "Necessity is the mother of invention". Hence, we are innovating, striving and exploring newer horizons and enjoying our journey of learning, sharing and teaching.

Long live AOGD!

Dr Mala Srivastava
President, AOGD

From the Vice President's Pen

Dear Colleagues,

"Coming together is a beginning, keeping together is progress and working together is success"

With a sense of great humility, privilege and responsibility, I would like to thank you all for the immense faith and trust shown in us as this year's organizing committee to lead AOGD's efforts to upliG women's health in our country.

Our editorial team has dedicated this month's bulletin to Critical Care Obstetrics in which learned faculty in Obs & Gynae, Haematology, Endocrinology and Neurology have shared their experiences in dealing with critical cases during this Corona pandemic.

As we approach the mega event of the year, just a fortnight away – AOGD's annual conference "Women Healthcare in the current challenging scenario", It gives me true pleasure, pride and honour to invite you all on behalf of the organizing committee.

This year's event will be a completely virtual experience. It will be live streamed online from our virtual conference platform in consideration of the participants' health during Covid times. The attendees from all over the world can participate in all events from the safety of their homes.

We hope all the participants will enjoy this 42nd AOGD Virtual Conference as it offers-

- High-quality program: As the first E-Conference of AOGD with 22 International speakers and Over 300 Eminent National Faculty enthralling the audience for 15 days
- Credit points - Gain up to 45 ICOG credit points
- Lots of prizes to be won in competition papers, posters, free papers, quiz and slogans
- Flexibility: You will be able to watch sessions live from location that suits you
- Interaction & networking: You will be able to ask questions, interact one to one with delegates and speakers, experience 3D virtual lobby, share your opinion, and instantly provide feedback on sessions throughout the events
- Innovations: Many of our speakers will showcase the latest innovations, equipment and technology in our field
- Lower registration fees which entitles one to be part of all 15 days events

I thank all the AOGD members and non-members for the overwhelming response that we have received from Pan-India.

I would like to end with an inspirational quote my parents always instilled in me to become a doctor - "Don't Stop. One day you will be someone's hope, Someone's Hero"

Regards,

Dr Kanika Jain
Vice President, AOGD

From the Secretary's Desk

Greetings to all ! Hope you all are in good health and safe.

On behalf of AOGD, I humbly request your participation to the **42nd Annual Virtual AOGD Conference** planned to be held on **30th-31st October & 1st November, 2020**.

With the Conference activities commencing from **23rd October** with **E-Quiz & Slogan Competition**, we are just a week away, looking forward to it with utmost eagerness and enthusiasm. The Conference highlights include **11 Pre & Post Conference Workshops, 3 Orations, 6 Keynote Addresses, 4 Panel Discussions, 6 Video Sessions** and **Expert talks** besides **E-Competition papers, free Communication papers & E-Posters.**

We are overwhelmed with responses to Competition papers, free Communication papers & E-Posters submission and appreciate the spirit of our zealous members to showcase their work on virtual platform.

Our editorial team has worked hard to bring out the AOGD E-Bulletin October version dedicated to **Critical Care Obstetrics, Part-2** which should be of great interest and of immense use to our readers.

Let's all together make this Conference a majestic success and enjoy the amazing virtual experience.

Looking forward to your continued support.

Ability is what you are capable of doing. Motivation determines what you do. Attitude determines how well you do it. – Lou Holtz

Warm Regards

Dr Mamta Dagar
Hon. Secretary

Maternal Mortality due to COVID-19: Lessons Learnt so far

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In these unprecedented circumstances of COVID pandemic, health system around the world is overstretched. During a pandemic health services for women and children are among the first to be affected. Early available data did not indicate that pregnant individuals were at an increased risk of infection or severe morbidity compared with non-pregnant individuals in the general population.

In early March 2020 when Mullins et al. published their article on *'Coronavirus in pregnancy and delivery'*, there had been no reported maternal deaths as a result of SARS-CoV-2 infection till then.[1]

Hantoushzadeh **et al.**[2] in April 2020 published their data from Iran which analyzed nine pregnant women with SARS-CoV-2 infection; seven of these patients died due to COVID-19. Out of the seven reported maternal fatalities, five had no underlying health issues, which suggests that pregnancy could put women at higher risk of more severe consequences from SARS-CoV-2 infection. Elshafeey et al.[3] highlighted in a systematic review of spectrum of disease in pregnant women with SARS-CoV-2 that most patients had mild illness, and 17 of 385 SARS-CoV-2-positive pregnant women required intensive care treatment and six out of these seventeen required mechanical ventilation, with one reported death.

Evidence from other pandemics: Experiences from the previous Influenza and SARSCov1 pandemics show that there is a trend toward increased disease severity among pregnant women. During the 1918 influenza pandemic, the proportion of deaths was reported to be 27% among the 1350 reported cases in pregnant women.[4] Similarly, regarding the SARS virus, Wong et al reported a mortality rate as high as 50% in those pregnant women who required ICU admission.[5] In the 2009 H1N1 influenza virus outbreak, pregnant women were 4 times more likely to be hospitalized and at increased risk of complications compared with the general population.[6]

For India and other South-Asian countries, which were affected later i.e. by mid- March, large surveillance cohort data of pregnancy and COVID is still awaited. Our knowledge of maternal and neonatal care with COVID comes form the already published evidence which is based upon the case series and isolated case reports from China, North America and Iran.

In this article, we would highlight the factors which may affect maternal mortality directly or indirectly. The course of disease and its severity during pregnancy cannot be predicted accurately but there are certain pointers that may anticipate an early intervention.

I. **Susceptibility to SARS CoVI 2 infection in pregnancy**[7]

In contrast to the earlier published data latest research considers the following changes in pregnancy which may raise concern a about the clinical course of COVID-19 in pregnant women.

a. Anatomical and physiological changes during pregnancy - Under the effect of progesterone and other relaxants in pregnancy causes relaxation of the ligaments of the ribs,with the progressive increases in size of uterus the diaphragm is pushed up and the transverse diameter of the chest increases which leads to eventually lead to a 20 to 30% reduction in functional residual capacity (FRC), which makes the mother prone to hypoxia, subsequently compensated by increased tidal volume and hyperventilation.[8] The changes of nasal mucosa mediated by progesterone during pregnancy may lead to the adhesion of the virus in the upper respiratory tract and make it difficult to be cleared.[7] The cardiovascular and metabolic changes that normally occur during pregnancy increase the metabolic rate and oxygen consumption, the decrease in functional residual capacity, and the mismatch between basic ventilation

and perfusion, all of these factors caused by pregnancy are easy to lead to the occurrence of hypoxic respiratory failure in women after infection with SARS-CoV-2.[9] On the other hand, if virus infection occurs, pulmonary vascular resistance will increase, which may lead to pulmonary hypertension and heart failure.[10]

b. Change in immune system- A study by Thomas et al[11] assessed the relationship between pregnancy and virus immune,the results showed that compared with postpartum, the late gestation was characterized by a decreased number and activity in NK cells and T cells, which may affect the viral clearance rate and lays a foundation for the onset and deterioration of infectious diseases in later half of the pregnancy as seen in the previous pandemics of SARS CoVi 1 and H1N1.

c. Increase in Expression of ACE2-It is speculated that level of ACE2 is doubled during pregnancy to regulate blood pressure. This adaptation may be a favorable condition for SARS-CoV-2 infection. ACE2 is not only a receptor, but also involved in post-infection regulation, including immune response, Cytokiene secretion, and viral genome replication.

These adaptive changes may make pregnant women less tolerant to hypoxia. Therefore, until more evidence is available, pregnancy itself may be a high risk for acquiring COVID infection and worsening of the disease in later half of the pregnancy.

II. **Risk Stratification and Triaging -** The CDC classifies Co-morbidities as established or possible risk factors for severe COVID-19. [Table1] A pregnancy which itself is a possible risk factor (CDC risk factors) when complicated with any of these conditions may guide us in anticipating management of progression to severe disease.

Triaging a pregnant women at the time of admission and then serially monitoring by using Q-SOFA warning score or National Early Warning Score (NEWS) 2 (Recommended by the NHS in UK for use in critically ill patients) helps in identifying women at risk of deterioration by the infection.

Table 1: Risk factor associated with COVID -19

Established risk factors	Possible risk factors
Chronic kidney disease	Asthma (moderate to severe)
Chronic obstructive pulmonary disease	Cerebrovascular disease
Immunocompromised state	Hypertension
Obesity	Liver disease
Serious cardiovascular disease	Pregnancy
Heart failure	Pulmonary fibrosis
Coronary artery disease	Smoking
Cardiomyopathies	Thalassemia
Sickle cell disease	Type 1 diabetes mellitus
Type 2 diabetes mellitus	

III. **Biochemical predictors of progression to severe disease-**The progression of disease cannot be predicted accurately during pregnancy. Effective bio-markers can be helpful in screening, clinical management, and prevention of serious complications. These bio-markers can also guide when to start immune-therapy/supportive measures, thus can prevent overall maternal morbidity and mortality.

a. Hematological Markers: Henry et al.[12] concluded in a meta-analysis on 21 studies including 3377 COVID-19 positive patients that patients with severe and fatal disease had significantly increased WBC, and decreased lymphocyte and platelet counts compared to non-severe disease. Sever disease is also associated with a higher leukocyte-counts and higher NLR, as well as lower percentages of monocytes, eosinophils, and basophils.

b. Biochemical biomarkers - Higher concentrations of ALT, AST, creatinine, CK, LDH, cardiac troponin I, N-terminal pro-brain natriuretic peptide, D-dimer, fibrinogen degradation products and prothrombin time. However this evidence has been based upon meta-analysis on non- pregnant population. The values of these parameters may change for a pregnant cohort.

c. Inflammatory Markers -IL-6, IL-2, IL-7, tumor necrosis factor (TNF)-α, interferon-γ inducible protein (IP)-10, CRP, pro-calcitonin (PCT), and ferritin are significantly linked to the 'Cytokiene Storm' and appropriate management can be started if these markers are found to rise serially. This also marks the onset of acute lung injury and further tissue damage.

IV. **Prompt initiation of medical and supportive care**

The uncertainty in the progression of COVID-19 during pregnancy still remains however early detection of rise in the bio-markers, deterioration in the general condition of the mother - deterioration in consciousness, sudden onset of dysponea, tachpnoea, fall in oxygen saturation (Spo2 ≤ 96%) and requirement of oxygen therapy will not only help in reducing the maternal morbidity but will also prevent prolonged exposure of the fetus to hypoxia.

Oxygen therapy should be prompted with the aim of maintaining oxygen saturation between 94-96%, care should be taken to prevent hyper-oxygenation which can itself lead to free radical injury.Non invasive ventilation can be initiated by using a Non Re-breather Mask (NRM) or nasal cannula, increasing the oxygen flow rate as per the requirement. Inability to maintain oxygen saturation on the above mode a High flow nasal cannula(HFNC) can be used a mode of maintaining the desired saturation levels. If there is no clinical improvement of the pregnant lady consider invasive mechanical ventilation.

Medical therapy in form of Remdesivir, a nucleotide analogue pro-drug that inhibits viral RNA polymerase, have been used on compassionate grounds large randomised control trials in pregnancy are still awaited. Other medical therapies that have been tried include antibiotics, corticosteroids, plasma therapy and anticoagulants.

The role and timing of termination of pregnancy in a moderate to severe disease remains debatable until supported by a strong evidence.

To conclude, in our scenario the final measures to prevent Maternal Mortality because of COVID-19 infection requires a definitive evidence-based guidance. However early risk stratification and biochemical predictors can be helpful in implementing a aggressively team-based care model to prevent maternal mortality caused by the direct effect of COVID-19 infection. In the near future we may also recognize the indirect effect of COVID on maternal mortality with its possible impact on three key sexual and reproductive health (SRH) services: births assisted by skilled health-care providers; births taking place in health facilities; access to contraception and the overall access to health care facilities.

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Peripartum Cardiomyopathy

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Introduction

Peripartum cardiomyopathy (PPCM) is a rare, idiopathic and a subset of dilated cardiomyopathy that is marked by systolic dysfunction that presents in last month of pregnancy or within 5 months following delivery in the absence of any other known cardiac disease. Pearson (2000) reported findings of a workshop of the National Heart, Lung and Blood Institute that established the following diagnostic criteria:[1]

1. Development of cardiac failure in the last month of pregnancy or within 5 months after delivery
2. Absence of an identifiable cause for the cardiac failure
3. Absence of recognizable heart disease prior to the last month of pregnancy, and
4. Left ventricular systolic dysfunction demonstrated by classic echocardiographic criteria, such as depressed ejection fraction (<45%) or fractional shortening (<30%) along with a dilated left ventricle.

Although PPCM occurs worldwide but more prevalent in Haiti and certain parts of Africa, according to recent literature incidence vary from 1:100[2] to 1:20000[3], depends upon geographical location, genetic and cultural differences with three to four times higher in black women than white women.

Associated risk factors include maternal age of 30 years or more, African ancestry, hypertension, anemia, substance misuse, asthma, autoimmune disease, pre-eclampsia or eclampsia, multiple gestation pregnancy, obesity, thyroid dysfunction, prolonged tocolysis, tobacco use, primipara status and zinc and selenium deficiency in some parts of world.

Recent data suggest that 50-80% of women with PPCM recover to normal range left ventricular systolic function (LVEF>=50%), with most of this recovery occurring within the first six months. Left ventricular size and ejection fraction at the time diagnosis most strongly predict left ventricular recovery. In the IPAC cohort, LVEF <30% and left ventricular end diastolic diameter >6 cm was indicative of decreased likelihood of left ventricular recovery and increased risk of mechanical support, transplant and death.[4] Maternal mortality reports vary widely and rates upto 15% has been seen in some studies.

Case Study

A 26 years old gravida 3, obese woman at 33 weeks 5 days of gestation presented with the chief complaints of shortness of breath (NYHA grade IV) for 3 weeks, swelling over lower limbs for 2 weeks and cough for 3-4 days. Her GC was fair, afebrile to touch, pulse- 114/min, regular, BP-114/80, respiratory rate of 28/min, maintaining 96% oxygen saturation at room air. On chest auscultation there was bilateral wheeze and crepitations. Her fundal height was corresponding to the period of gestation with fetal heart rate of 130bpm. She had one previous preterm LSCS at 8 months of gestation 3 years back and one missed abortion 1 year back. Following 4 months of her first delivery she had breathing difficulty, cough and edema in lower limbs for which she was admitted in cardiology department. Her 2D ECHO was suggestive of moderate MR, Global hypokinesia and LVEF of 25-30%. She was managed conservatively and discharged from there on treatment with Tab Spironolactone 50 mg BD, Tab ivabradine 5 BD, Tab Losartan 50 OD, Tab torsemide 40 OD, Tab Pantoprazole. She was asked to follow up in the cardiology department strictly but she was non-compliant with the treatment. She had second episode of dyspnea 1 year back for which she was admitted in ICU for 3 months.

In current pregnancy she herself left all the medicines except Tab torsemide 40. The patient was admitted and evaluated after informing her about the prognosis and consequences of the disease. Her routine investigations were within normal limits; NT-proBNP was 1466 pg/ml. Chest Xray showed cardiomegaly. Echo was suggestive

of generalized hypokinesia, LVEF 25%, mild MR, tachycardia present, LV dilated in size, grade II diastolic dysfunction present, no pericardial effusion noted. The patient was shifted to ICU and conservative management is started with propped up position, oxygenation to maintain saturation, antibiotics, Tab ivabradine 5 mg BD, Tab Spironolactone 50 mg BD, Inj Furosemide iv TDS, and injectable corticosteroids for fetal lung maturity under supervision of intensivist, cardiologist and senior obstetrician.

Discussion

Peripartum cardiomyopathy is a rare but life threatening disease. However, a large proportion of patients who otherwise meet the criteria for PPCM present before 36 weeks' gestation, raising concerns that the NHLBI definition may be overly restrictive and lead to the under diagnosis of PPCM. Given the concern in 2010 the European Society of Cardiology (ESC) defines peripartum cardiomyopathy as heart failure that occurs "towards the end of pregnancy or in the months following delivery, where no other cause of heart failure is found.[5]

Etiopathogenesis

Although the complete pathogenesis of PPCM remains unclear, several theories have been proposed regarding the same. Current thinking favors a "two hit" model of PPCM pathogenesis, whereby a vascular insult caused by antivascular or hormonal effects of late pregnancy and the early postpartum period induces cardiomyopathy in women with an underlying predisposition.

1. ***Genetic predisposition***: It has long been observed that some cases of PPCM cluster in families. However, a study that sequenced 43 genes with variants associated with dilated cardiomyopathy from 172 women with PPCM revealed 26 (15%) distinct, rare truncating variants, 65% of which occurred in TTN, the gene that encodes titin.[6] The presence of a TTN variant predicted lower LVEF at 12 months.
2. ***Prolactin:*** Increased reactive oxygen species lead to secretion of cathepsin D by an mechanism that is currently not well understood. Cathepsin cleaves prolactin into a 16kDa prolactin. 16kDa Prolactin induces endothelial cells to package miR-146a into exosomes which are then taken up by cardiomyocytes. 16kDa Prolactin is associated with endothelial and myocyte apoptosis. miR-146a blocks several pathways including Eebb4, Nras and Notch1 that lead to cardiomyocyte death. Thus miR-146a may serve as both a biomarker and therapeutic target in PPCM.[7]
3. ***sFLT1 (Placental Angiogenic factors):*** A murine model of PPCM has implicated the loss of vascular endothelial growth factor (VEGF) in the pathogenesis of PPCM. Soluble fms-like tyrosine kinase receptor 1 (sFLT1) is a molecule secreted by placenta in late pregnancy and in higher levels in pre-eclampsia and twin gestations. sFLT1 neutralizes VEGF and decreases the level of VEGF in circulation which is thought to contribute to PPCM.[8] Taken together the increased production of prolactin and placental secretion of sFLT1 could be toxic to both the vasculature and the cardiac myocytes.
4. ***Myocarditis:*** The equal prevalence of myocardial inflammation and viral genomes[9] that has been noted in subjects and controls who underwent myocardial biopsy challenges the pathogenic role of myocarditis in PPCM.
5. ***Nutritonal factor:*** deficiency of selenium, zinc and iron have been proposed as causative factors in Haiti and Nigeria.[10]
6. ***Microchimerism:*** with fetal derived cells in the maternal circulation has been proposed as potential contributing factor for PPCM.[11]
7. ***Autoimmune Mechanism***: Small series have shown that autoantibodies against adrenergic receptors and sarcomeric proteins are more common in patients with PPCM.

Clinical Presentation

Patients with PPCM complain symptoms of weakness, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, edema of lower extremities, nocturia. Less commonly women present with arrhythmias, arterial thromboembolism and cardiogenic shock that requires inotropic and mechanical circulatory support. Physical examination finds lower extremities edema, raised respiratory rate and signs of left sided congestion e.g. pulmonary rales) and right sided congestion (e.g. Raised JVP, edema). On auscultation left or/ and right sided S3 gallop may be audible.

Diagnosis

Diagnosis of PPCM is often challenging specially with the slow developing form with nonspecific symptoms of cardiac congestion, abdominal discomfort, pleuritic chest pain and palpitations, paroxysmal nocturnal dyspnea, chest pain, cough, neck vein distention, new murmurs consistent with atrioventricular valve regurgitation and pulmonary crackles which might raise the suspicion of heart failure. The diagnosis requires excluding other causes of cardiomyopathy and is confirmed by standard echocardiographic assessment of left ventricular systolic dysfunction. Following investigations can be done:

A. ***Chest radiograph****:* depict signs of heart failure with pulmonary congestion, cardiac enlargement and pleural effusion.

B. ***Electrocardiography*****:** helps in excluding pulmonary embolism and myocardial infarction. Findings are often normal but can include sinus tachycardia, nonspecific ST and T wave abnormalities and voltage abnormalities. Complications like atrial fibrillation and flutter, Q wave in anteroseptal leads, prolonged PR nad QRS intervals and bundle branch block are also observed.

C. ***Echocardiography*****:** most essential form of imaging in PPCM, shows left ventricular dysfunction with LVEF <45% and often left ventricular dilatation, right ventricular dilatation and dysfunction, pulmonary hypertension, left atrial or bilateral enlargement, functional mitral and tricuspid regurgitation and intracardiac thrombus.

D. ***Cardiac magnetic resonance imaging***: enables the precise diagnosis of myocarditis, necrosis, LV thrombi, ventricular volumes, site of endomyocardial biopsy. Injection of contrast agent gadolinium required should be avoided during pregnancy as it crosses placenta.

E. ***Cardiac catheterisation endomyocardial biopsy*****:** highly specific, invasive, considered only when myocarditis is strongly suspected or no improvement is seen after 2 weeks of heart failure therapy.

Treatment

A multidisciplinary team comprising of obstetrics, anesthesia and cardiology should individualize patient management including decisions about the timing and mode of delivery after informing risk and benefits to the mother and fetus about the treatment. According to guidelines standard treatment for heart failure with reduced ejection fraction should be provided to the patient of PPCM. Extra care should be taken to avoid adverse fetal effects in pregnant women.

Patient should be transferred to intensive care unit in tertiary centre where she can be monitored and the facility of NICU can be provided. According to AHA and ESC guidelines sodium restriction is the mainstay of volume management and a loop diuretic may be added for symptomatic pulmonary and peripheral edema, over diuresis should be avoided to prevent maternal hypotension, uterine hypoperfusion and FGR.

Non Pharmacologic Treatment

Low sodium diet (2g/day). Physical activity should be limited according to patient's symptoms as bedrest may promote better uteroplacental perfusion but can cause venous thromboembolism.

Pharmacologic Management

The mainstays of medical therapy are digoxin, loop diuretics and beta-adrenergic blockade with carvedilol or metoprolol succinate as they have been shown to decrease all cause mortality and hospitalization in those with systolic dysfunction.

a. ***Diuretics*****:** are used to manage the maternal volume status with close monitoring of electrolytes. Loop diuretics should be the first line treatment in case of pulmonary edema, started with furosemide 10 mg as pregnant women have an increased glomerular filtration rate (GFR) that facilitates secretion of the drug into the loop of henle. Spironolactone should be given to patients with reduced left ventricular systolic function in the postpartum period, it is contraindicated during pregnancy due to its teratogenic effects. Bumetanide may be used when clinically indicated and a thiazide can be added cautiously to a loop diuretic for a synergistic effect in diuretic resistant patients. Diuretics should be used very cautiously as maternal volume depletion may lead to cause uteroplacental hypoperfusion specially in patients with preeclampsia because

intravascular volume depletion is a hallmark of preeclampsia.

b. ***Hydralazine and nitrates***: used to decrease maternal preload when indicated, safe for mother and fetus and compatible with breast feeding. Hydralazine in combination with nitrates is the first choice for afterload reduction and vasodilatation and supposed to be a preferred regimen during pregnancy; women should be started on angiotensin-converting enzyme inhibitor after delivery.

c. ***Beta blockers*****:** metoprolol tartrate has been most commonly used for PPCM during pregnancy. Carvedilol remains an alternative to metoprolol, given its potential antitocolytic activity. Atenolol should be avoided due to risk of fetal growth restriction. Fetal growth should be monitored.

d. ***Digoxin and inotropes***: Digoxin could be considered in women with an abnormal ejection fraction. I.V. inotropes should be considered in patients with hypotension and cardiogenic shock. Invasive hemodynamic monitoring should be done to gauge the response to therapy. Digoxin is safe with breastfeeding.

e. ***Anticoagulants*****:** Thromboembolism is a relatively common complication of PPCM and pregnancy itself is a hypercoagulable state. The risk is likely related to the degree of chamber enlargement, systolic dysfunction and the presence of atrial fibrillation. **ESC** guidelines advise anticoagulation in patients with PPCM and LVEF <35% and in those who have received bromocriptine. **AHA** guidelines advise considering anticoagulants in women with PPCM and LVEF <30%. Experts recommend use of anticoagulants until 8 weeks postpartum.

Warfarin, low molecular weight or unfractionated heparin are possible options for anticoagulation. Warfarin may be used safely in second and third trimester and then switch to heparin before delivery. Warfarin is proposed as drug of choice in postpartum period, compatible with breast feeding.

The ideal time to discontinue the medicines is not known but their use should be continued for at least 1 year. If medical treatment is not successful, heart transplantation is often the last resort. In recent years the rate of heart transplantation has decreased to about 4% from 7%. Transplantation success rates are good with favorable long term survival rates.

Emerging Role of Bromocriptine in PPCM

Bromocriptine and cabergoline are dopamine D_2 agonists and inhibit prolactin production thereby also suppressing lactation. Twenty PPCM patients in Africa were randomized to open label bromocriptine for 8 weeks or standard care; those receiving bromocriptine had more recovery of LV function and a reduction in clinical endpoints relative to standard care.[12] Further observational studies from Germany (N=115)[13] and Canada (N=76) have also observed an increased LV recovery in women who received bromocriptine compared to those who did not receive bromocriptine.

In a recent multicenter, blinded study of 63 German patients with PPCM and an LVEF below 35% who were randomized participants to receive either one week or eight weeks of Bromocriptine in addition to standard medical therapy for heart failure, LVEF as measured by MRI, increased in both groups at 6 months with no significant difference in the increase (29% to 49% in the 1 week group and 27% to 51% in the 8 week group) or the frequency of full recovery, defined as LVEF above 50% (52 % in 1 week group versus 68% in 8 group). Although no control group was included in this study, the authors compared a subset of their cohort with LVEF <30% (n= 37) to the IPAC cohort, in which patients with LVEF <30 % received standard therapy for heart failure without Bromocriptine. Persistent LV dysfunction was noted in 37% of the patients in the IPAC cohort and only 14% in the subset of patients with LVEF < 30% in the current trial.

Whether prolactin inhibition improves outcomes for all women with PPCM and thus should be part of standard treatment remains controversial. Currently, the use of bromocriptine in heart failure may be best justified in women with PPCM who have severe cardiomyopathy (LVEF<25%) or cardiogenic shock or both. Bromocriptine can be given as 2.5 mg daily for 1 week in uncomplicated cases. Higher doses (2.5mg bid for 2 weeks, followed by 2.5 mg daily for 6 weeks) are recommended in patients with complicated course (e.g. LVEF<25% or cardiogenic shock). Treatment with bromocriptine must always be accompanied by anticoagulation, given the increased risk of myocardial infarction and stroke with this drug.

Delivery

Vaginal delivery is preferred as it is associated with lesser complications like endometritis and pulmonary embolism. According to ESC and AHA guidelines cesarean delivery should be considered in cases of acute heart failure, otherwise reserved for obstetric indications. Hemodynamic shifts of labor may be managed by epidural anesthesia and an assisted second stage of labor (use of vacuum or forceps).

Lactation

Breast feeding provides considerable health benefits to the infant and is particularly beneficial in the developing world, where access to clean water and alternative nutrition sources may be limited. The use of pharmacologic prolactin inhibition and cessation of breast feeding is controversial. Currently it is considered that bromocriptine should be used in women with severe left ventricular dysfunction (LVEF<25%) or cardiogenic shock. Other women with less impaired left ventricular function should be allowed to breast feed if able.

Subsequent Pregnancy

The prognosis for women with PPCM depends on the normalization of left ventricular size and function within 6 months after delivery. In about 50% of patients, the ejection fraction normalizes.

In a review encompassing 191 subsequent pregnancies women with persistent left ventricular dysfunction (LVEF<50%) had a 50% risk of acute heart failure with worsening cardiomyopathy and in some subseries of South Africa, a 25-50% risk of mortality.[14] Regardless of recovery, currently there is no consensus regarding recommendations for future pregnancy because PPCM recurs in more than 30% of subsequent pregnancies, which puts both mother and baby at great risk.

ESC and AHA guidelines advise that repeat pregnancy is contraindicated in women with PPCM who have not recovered a normal LVEF.

The use of an intrauterine device is recommended for PPCM patients since hormonal contraceptives may interact with heart failure medication.

Conclusion

Peripartum cardiomyopathy is a rare but life threatening medical condition that affects women worldwide. The underlying pathophysiology is not known, vasculo-hormonal influences and genetic susceptibility probably play a role. According to guidelines second pregnancy is contraindicated in women with history of PPCM, If cannot be avoided the women should be counseled about the risks of subsequent pregnancy and should be followed closely throughout pregnancy and until six months postpartum with frequent clinical examinations and serial echocardiograms.

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Calendar of Virtual Monthly Clinical Meetings 2020-21

29th May, 2020	B L Kapoor Hospital
26th June, 2020	VMMC & Safdarjung Hospital
31st July, 2020	AIIMS
14th August,2020	Lady Hardinge Medical College
28th August, 2020	Army Hospital- Research & Referral
11th September,2020	Apollo Hospital
25th September, 2020	DDU Hospital
23rd October to 6th November, 2020	AOGD Annual Conference Activities
27th November, 2020	MAMC & LNJP Hospital
18th December, 2020	Sir Ganga Ram Hospital
1st January, 2020	ESI Hospital
29th January, 2021	Dr RML Hospital
26th February, 2021	UCMS & GTB Hospital
26th March, 2021	Lady Hardinge Medical College
23rd April, 2021	Apollo Hospital

Eclampsia, PRES, Status Epilepticus- Neurologist's Perspective

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Introduction and Definition

Pregnancy induced hypertension (pre-eclampsia / pregnancy toxemia) develops after 20 weeks of gestations in previously normotensive women and resolves by three months post partum: the possibility considered high if blood pressure > 140/90 mmHg or diastolic BP rises 15-25 mm Hg above pre pregnancy values[1], When such a patient has a convulsion they should be considered to have eclampsia unless proven otherwise.

It is most common in 3rd trimester and presents with proteinuria > 0.5gm/ 24 Hr, edema, headache, visual disturbance and gastrointestinal disturbances.

Clinical Features and Diagnosis

Convulsion, labile high BP, proteinuria (sometimes nephrotic range), visual disturbances in form of photopsia; cortical blindness, malaise, facial and peripheral edema, oligurea, restlessness and clonus.

Investigation may reveal hyperuricaemia, thrombocytopenia, raised liver enzymes and evidence of hemolysis.

Important differential diagnosis in pregnant women having the 1st seizure is subarachnoid hemorrhage, cerebral venous thrombosis, intra cranial SOL, intracranial infection, metabolic disorders, Autoimmune disorder- mostly SLE[2].

Pathophysiology

- It has been variously proposed that convulsions in eclampsia result from intra cranial hemorrhage, hypertensive encephalopathy, cerebral edema or cortical vasospasm[2,3].
- Typical postmortem findings reveal fibrinoid necrosis, thrombosed pre capillaries, micro hemorrhage and hypoxic ischemic changes[2,3,4,5].
- There is predilection for abnormalities in the occipital and parietal lobe in the watershed area between MCA and PCA[6,7]. There is growing evidence of vasoconstriction in such cases.

Management

- Eclampsia seizure should be managed initially with whatever anticonvulsant agents in hand and with which the practitioner is familiar with.
- Target should be to terminate seizure and prevent hypoxemia in mother and in the fetus.
- Magnesium sulphate has been the drug treatment of choice of eclampsia and pre eclampsia now[8].
- Proposed magnesium sulphate dose is to give 5 gm magnesium sulphate over 20-30 minutes (10 ml of 50% magnesium sulphate in 200 ml saline); Then infuse magnesium sulphate at 2 gm / hr. Magnesium level to be checked after 30-60 min and then every 6 hrs to maintain therapeutic range of 2-3 mmol/ litre.
- If the blood pressure falls below 110/70 mm hg: respiratory rate falls below 16, urine output is below 30 ml/ hr or areflexia occurs, we reduce the infusion rate to 1gm/ hr and treat accordingly.
- If seizure persists benzodiazepam and other antiepleptics (levetiracetam, phenytoin, valproate etc to be given according to the clinical situation.

Conclusion

Eclampsia is associated with disproportionate degree of maternal and fetal mortality. It is important to diagnosed early and treat adequately. Needs multi disciplinary approach for good outcome.

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PRES

- Disorder with acute neurological symptoms, for example seizure, headache, encephalopathy, visual disturbances, secondary to reversible subcortical vasogenic edema in the brain in the setting of hypertension, renal failure, pre eclampsia, eclampsia, cytotoxic drug or Autoimmune disorder.
- PRES is generally reversible both radio logically and clinically.
- It is caused by endothelial injury related to changes in the blood pressure in the background of above mentioned co-morbid states[1].
- Posterior brain regions can be particularly susceptible to hypo perfusion because little sympathetic intervention exists in posterior fossa[2].
- Can affect any age, predominantly middle aged female
- Co-morbid condition which can trigger PRES are Eclampsia, Hydatiform mole renal failure, alcohol withdrawal, sepsis, dyselectrolytemia, TTP, Henoch schonlein perpura, organ transplantation, autoimmune disorder, chemotherapeutic drugs etc.

Clinical presentation

- Encephalopathy- clinical severity ranging from confusion to coma
- Seizure in upto 92 % cases
- Visual abnormality
- Focal neurological defect
- Thrombotic Stroke

Diagnosis is based on clinical features[3]. CT / MRI reveal changes mainly in the parieto occipital area.

Differential diagnoses

- Infective encephalitis
- Autoimmune / para neoplastic Encephalitis
- Demyelinating / toxic leuko encephalopathy
- CNS vasculitis
- Malignancy and metastatic disease
- Progressive multifocal leuko encephalopathy
- Osmotic Demyelination syndrome

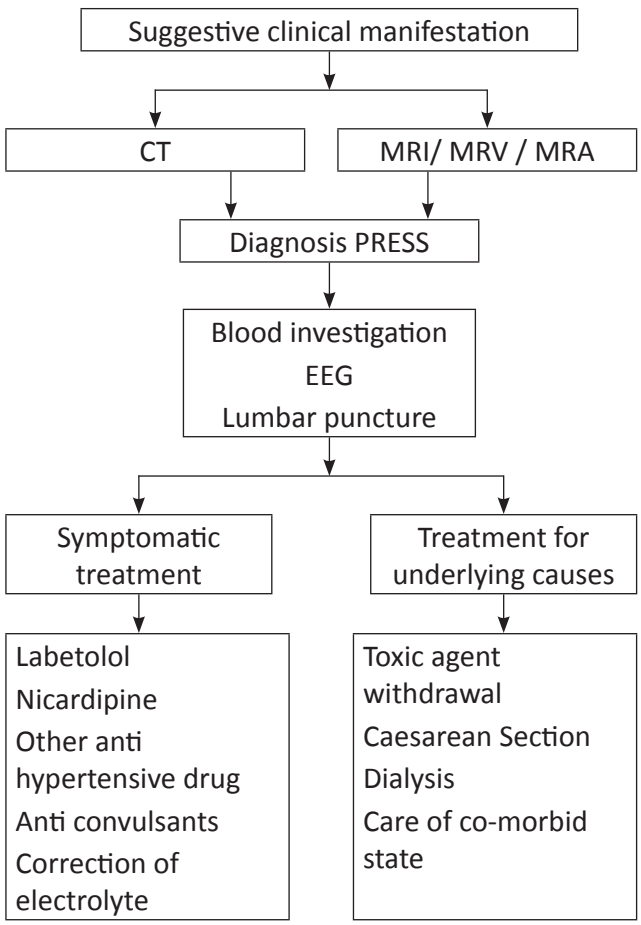
Treatment

General measures - to maintain airway patency, breathing and circulation of patient.

Symptomatic treatment

- Antihypertensive treatment to reduce mean blood pressure by 25% in first few hours, preferably Labetolol infusion to be started after 20 mg of bolus Dose and 20-60 mg/hour-BP should be monitored and infusion rate should be regulated. Later on to be shifted to oral Labetolol 100-400 mg/day or calcium channel blocker drugs
- Anticonvulsants-first line antiepileptic drugs are used. Levetiracetam 20 mg/kg bolus followed by

PRES: Flow chart



1-4 gm/day. Levetiracetam is the preferred drug as it is relatively safe in pregnancy and has the least drug interaction in co morbid state. In case of persistent seizures, they are treated according to the Status Epilepticus guidelines

- Treatment for co-morbid state
- Withdrawal of offending drug
- Termination of pregnancy

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Status Epilepticus

- Seizure is a paroxysmal event due to abnormal, excessive or synchronous neuronal electrical activity in the brain
- Epilepsy is a condition in which a person has recurrent seizures due to a chronic underlying process
- Status epilepticus-this is defined as 5 minutes or more of continuous seizure activity without recovery between seizure and continuous clinical or electrographic seizure activity
- Refractory Status epilepticus –when seizures do not respond to Antiepileptic drugs
- Super Refractory Status epilepticus- when seizures do not respond to Anesthetic drugs
- Most common cause of Status epilepticus -Anticonvulsant drug withdrawal or non-compliance

  CVA

  CNS infections

  Metabolic derangement

  Alcohol/drug toxicity

  Hypoxia

  Intracranial space occupying lesion

  Trauma

  Fever

Acute Management of seizures

0-5 minutes

Secure airway, breathing, circulation-stabilize patients

5-15 minutes

Give Dextrose unless patient is hyperglycemic/ Normoglycemic

Give Thiamine 100mg IV if Alcoholic/Malnutritioned/ Bed bound send blood investigations-CBC, RFT, LFT, Antiepileptic drug level (if on medication), Toxic screening, culture/sensitivity

Detailed physical Examination

15-35 minutes

Anticonvulsants

Lorazepam 0.1to 0.15 mg/kg IV (upto 4-6 mg) over 1-2 minutes. If seizure persists can be repeated after 5-10 minutes

Another longer acting AED is needed if underlying etiology is not rapidly reversible

Phenytoin 20 mg/kg or Fosphenytoin, 3 equivalent bolus dose to be given

Levetiracetam 20 mg/kg bolus or Valparin 25 mg/ kg can be given if seizure are not being controlled

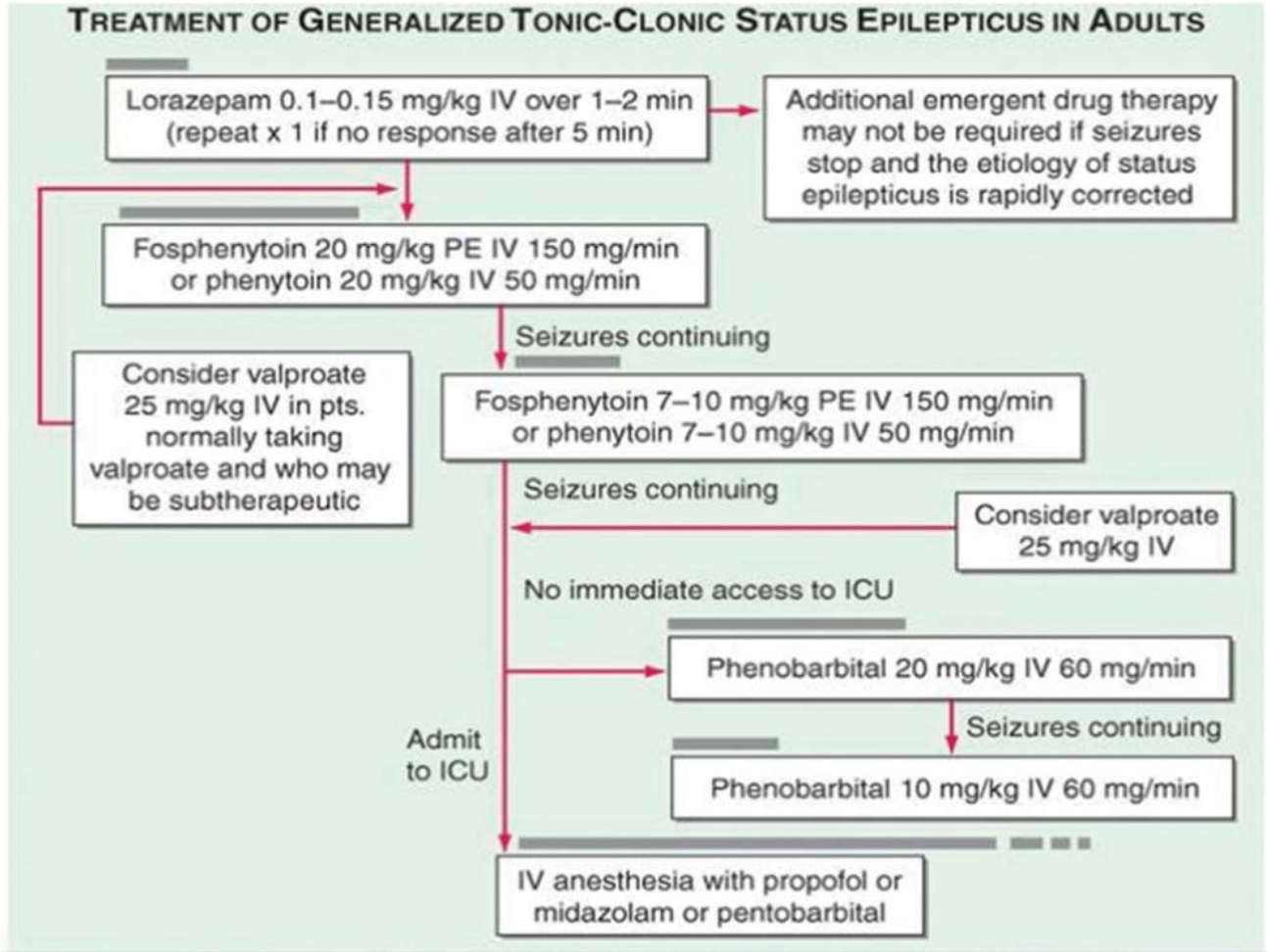
45-60 minutes

if seizure continues beyond 60 minutes, it is Refractory Status epilepticus-General Anesthesia should be induced

Propofol-2mg/kg IV bolus followed by 2-10 mg /kg/ hour

Thiopental 100-250 mg IV bolus over 20 seconds followed by infusion 3-5 mg/kg/minute

Midazolam 0.3 mg/kg IV bolus at the rate of 4 mg/ minute-repeat every 5 minutes-3 doses, followed by infusion 2 mg/kg/hour. If seizure is controlled dose should be reduced after 12 hours


TREATMENT OF GENERALIZED TONIC-CLONIC STATUS EPILEPTICUS IN ADULTS
Lorazepam 0.1–0.15 mg/kg IV over 1–2 min (repeat x 1 if no response after 5 min)
Additional emergent drug therapy may not be required if seizures stop and the etiology of status epilepticus is rapidly corrected
Fosphenytoin 20 mg/kg PE IV 150 mg/min or phenytoin 20 mg/kg IV 50 mg/min
Seizures continuing
Consider valproate 25 mg/kg IV in pts. normally taking valproate and who may be subtherapeutic
Fosphenytoin 7–10 mg/kg PE IV 150 mg/min or phenytoin 7–10 mg/kg IV 50 mg/min
Seizures continuing
Consider valproate 25 mg/kg IV
No immediate access to ICU
Phenobarbital 20 mg/kg IV 60 mg/min
Seizures continuing
Admit to ICU
Phenobarbital 10 mg/kg IV 60 mg/min
IV anesthesia with propofol or midazolam or pentobarbital

Thyroid Storm and Diabetic Ketoacidosis in Pregnancy: An algorithmic approach

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Thyroid Storm in Pregnancy

1. Introduction

Thyroid storm is an acute complication of thyrotoxicosis. It manifests as severe life-threatening symptoms of thyrotoxicosis. Thyroid storm carries high mortality of 10-30%.[1]

Hyperthyroidism complicates 0.2% of pregnancy and thyroid storm is known to occur in less than 1% of patients with hyperthyroidism.[2] Hence Thyroid storm in pregnancy is a very rare entity with the knowledge restricted to mainly case reports and case series.

2. Precipitating factors for thyroid storm

Thyroid storm is often precipitated in pregnant women with uncontrolled hyperthyroidism during parturition. Sudden discontinuation of anti-thyroid medications during pregnancy can also potentially precipitate a thyroid storm. The likelihood of a pregnant woman developing thyroid storm is 10 times more than for a non-pregnant individual.[3]

The pathophysiology behind the precipitation of a thyroid storm as compared to routine hyperthyroidism is unclear. A sudden increase of thyroid hormone can be one factor that leads to thyroid storm. Apart from this increased tissue response to thyroid hormone and increased catecholamine action are other factors that may precipitate an acute thyroid event like a thyroid storm.[1]

3. Clinical features of thyroid storm

Patients with thyroid storm often present with typical symptoms of hyperthyroidism but in an exaggerated form.[1]

New onset of Atrial fibrillation or cardiac tachyarrhythmia are some common presentations. The patient can also have heart failure. Hyperpyrexia is often present. Gastrointestinal symptoms like nausea and vomiting may be exaggerated especially in pregnancy. The patient may also have jaundice and other signs of liver failure. Neurological symptoms ranging from anxiety to comatose state may be associated with the other clinical symptoms.[1]

On clinical examination, one must look for the presence of goiter and the presence of thyroid-associated orbitopathy (also called Graves' ophthalmopathy). [1]

4. Diagnosis of Thyroid storm

In a patient with Thyroid storm, the lab evaluation may show thyrotoxicosis with elevated T3 and T4 and normal TSH. The degree of thyroid hormone elevation does not correlate with the severity of symptoms. Hyperglycemia and hypercalcemia may be often associated with thyroid storm.[4]

A high tire of the TSH Receptor antibody (TRAb) is useful for establishing the diagnosis of Graves' disease as an etiology for the thyroid storm. However, in an emergency situation, the treatment should not be delayed because of non-availability or delay in the results TRAb.[4]

An ECG with a rhythm strip must be done in all patients and cardiac monitoring in ICU is advisable for all patients with thyroid storm. A liver function test is mandatory to rule out liver involvement which is not uncommon in thyroid storm.[3]

The diagnosis of Thyroid storm is established based on a scoring system called the Burch and Wartofsky score (**Figure 1**). A score of more than 45 is the sine-qua-non of thyroid storm. A score of less than 25 makes thyroid storm unlikely. A score of 25-45 is suggestive of an impending thyroid storm.[4]

5. Management of Thyroid Storm

A patient of thyroid storm is typically managed in the ICU. The basics of any critical illness disease management like securing the airway, breathing and circulation should be followed.[2] The initial assessment and triage of the patient with potential thyroid storm is summarized in **Figure 2**

Criteria	*Points*	*Criteria*	*Points*
Thermoregulatory dysfunction		Gastrointestinal–hepatic dysfunction	
Temperature (°F)[b]		Manifestation	
99.0–99.9	5	Absent	0
100.0–100.9	10	Moderate (diarrhea, abdominal pain, nausea/vomiting)	10
101.0–101.9	15	Severe (jaundice)	20
102.0–102.9	20		
103.0–103.9	25		
≥104.0	30		
Cardiovascular		Central nervous system disturbance	
Tachycardia (beats per minute)		Manifestation	
100–109	5	Absent	0
110–119	10	Mild (agitation)	10
120–129	15	Moderate (delirium, psychosis, extreme lethargy)	20
130–139	20	Severe (seizure, coma)	30
≥140	25		
Atrial fibrillation			
Absent	0		
Present	10		
Congestive heart failure		Precipitant history	
Absent	0	Status	
Mild	5	Positive	0
Moderate	10	Negative	10
Severe	20		
Scores totaled			
>45	Thyroid storm		
	Impending storm		
<25	Storm unlikely		

Fig 1: The Burch and Wartofsky score for the diagnosis of Thyroid storm. [4]

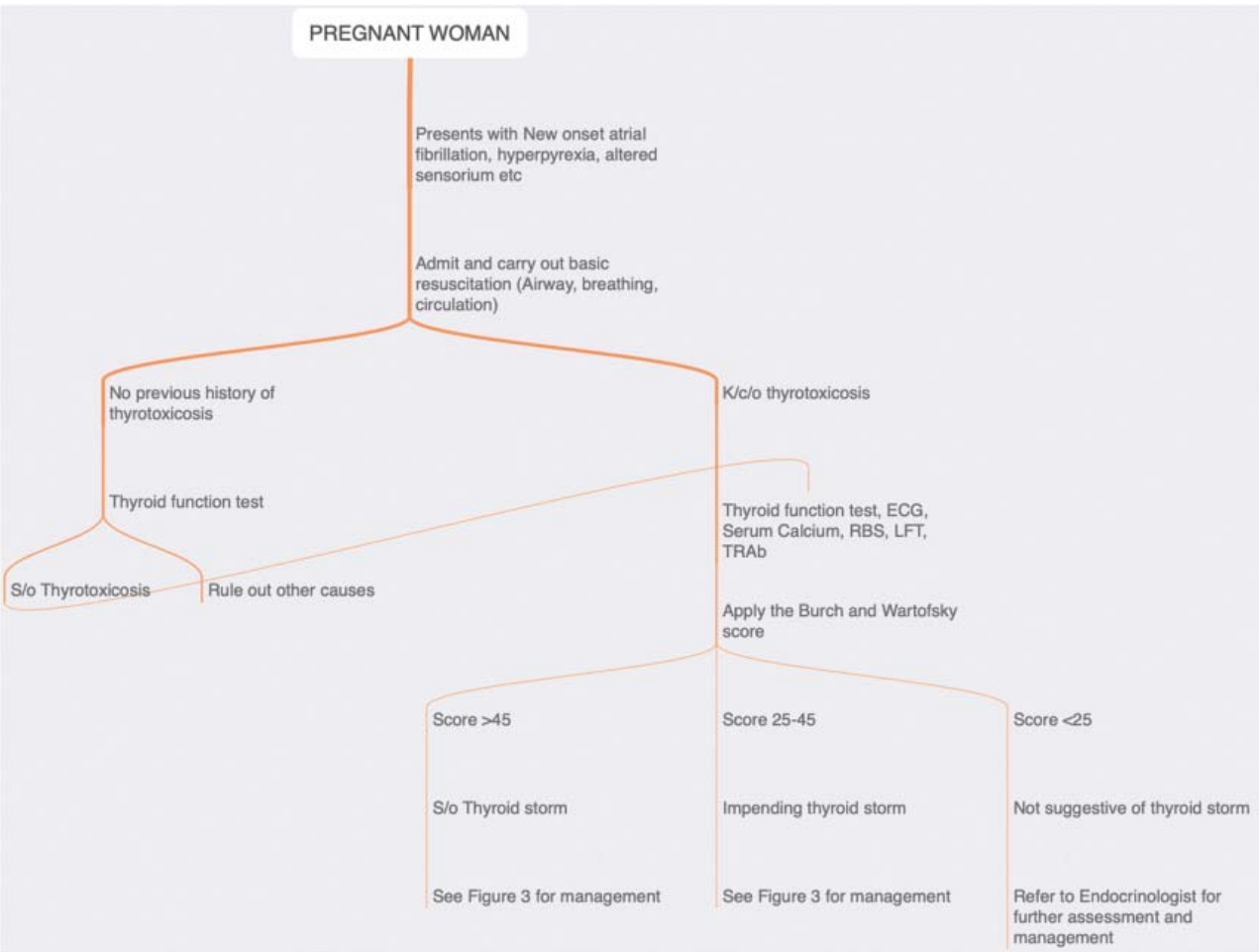

Fig 2: Initial assessment and triage of a pregnant woman suspected to have thyroid storm. (k/c/o is known case of; s/o suggestive of; ECG- Electrocardiogram; RBS- Random blood sugar; TRAb- TSH receptor antibody; LFT- liver function test)

Beta-blockers are started in these patients as early as possible. Beta-blockers block the over-activation of the sympathetic system that occurs in thyrotoxicosis. Propranolol is used as the beta-blocker of choice. An intravenous bolus of Esmolol followed by infusion is another alternative.[4]

Propranolol is typically given in the dose of 20 mg every 4-6 hourly either orally or as a crushed suspension via the Ryle's tube (RT). Propranolol also blocks the T4 to T3 conversion.[4] It must be remembered that propranolol is a pregnancy category C drug. It can lead to fetal bradycardia, hypoglycemia, and respiratory depression. Hence fetal monitoring is vital.[5]

Amongst the anti-thyroid drugs, Propylthiouracil (PTU) is proffered over carbimazole or methimazole in thyroid storm. The onset of the action of PTU is faster than carbimazole/ methimazole and additionally, it is not known to produce any significant teratogenicity. PTU can also be used in the first trimester of pregnancy. PTU is typically given in a higher dose of 200 mg every 4 hourly. PTU can be delivered by RT if the patient cannot take the same orally. PTU can lead to idiosyncratic hepatotoxicity.[1,2,4]

Lugol's iodine or Saturated solution of Pottasium iodide (SSKI) may be used one hour after the administration of PTU. Glucocorticoids like hydrocortisone as given along with the other medications. Glucocorticoids also block T4 to T3 conversion and help correct an undiagnosed adrenal insufficiency which may be unmasked in the period of severe physiological stress. Hydrocortisone in the dose of 100 mg IV every 8 hourly or dexamethasone, 1 to 2 mg every six hours can be used.[4]

Other agents that can be used in case of non-availability of contraindications to any of the above agents include Lithium or Cholestyramine.[6] In patients with pre-existing hepatotoxicity, the use of PTU may be deemed inappropriate. Hence in such cases, these other agents may prove to be effective. Plasmapheresis has also be used in thyroid storm in severe cases.[7]

There is little or no data on the management of the obstetric aspect of thyroid storm. The question of continuing the pregnancy should be decided based on individual cases. It must be kept in mind that thyroid storm is a potentially life-threatening condition of the mother.[2]

Diabetic Ketoacidosis in Pregnancy

1. Introduction

Diabetic ketoacidosis (DKA) in pregnancy complicates about 1-2% of pregnancies and leads to fetal loss in 10-30% of cases. [8] DKA can occur in both type 1 diabetes, Latent-autoimmune diabetes of adulthood (LADA) as well as in selected cases of type 2 diabetes. DKA can be the first presentation of type 1 diabetes.[9]

The standard diagnostic criteria for DKA is described in table 1.

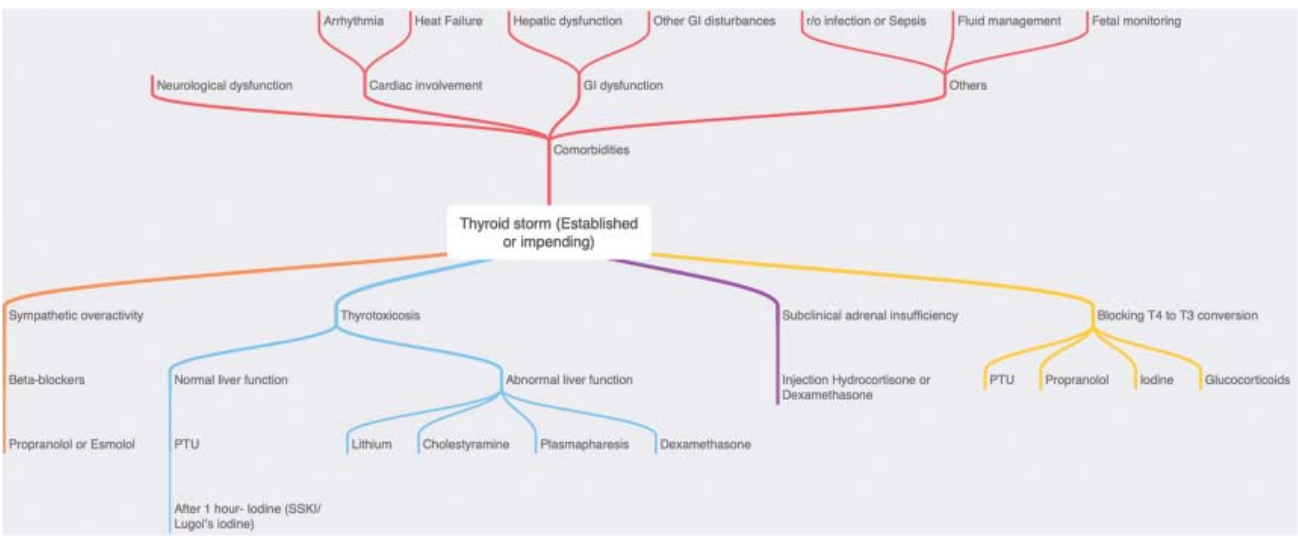

Fig 3: Overview of the management of thyroid storm in pregnancy (PTU- Propylthiouracil; GI- Gastrointestinal; SSKI - Saturated solution of pottasium iodide)

Table 1: Diagnostic criteria for Diabetic ketoacidosis in pregnancy

1. Hyperglycemia- glucose value >250 mg/dl
2. Ketonemia
   a. Serum beta-hydroxybutrate or point-of-care value >3 mmol/l
   b. High serum acetone
   c. Presence or ketonuria- more than 2+ in urine ketone-sticks
3. Acidosis
   a. pH <7.3
   b. Bicarbonate <15 meq/
4. Anion-gap >12 meq/

2. Pathophysiology of Diabetic ketoacidosis

DKA is a result of an imbalance between insulin production and counter-regulatory hormones like glucagon. Low insulin and high glucagon in background of diabetes mellitus leads to DKA.[10] Ketone bodies are reserve fuel for the human body. In the absence availability of intracellular glucose as a fuel, beta-oxidation of fatty acid occurs which kicks off the ketogenesis pathway. This pathway is suppressed by insulin and enhanced by glucagon, growth hormone, and epinephrine. In the situation of an imbalance between insulin and these counter-regulatory hormones, these pathway's come into play and lead to ketogenesis.[10]

Ketone body formation in excess leads to high anion gap metabolic acidosis. DKA is also a state of total body potassium deficit. Most importantly, high glucose levels in the blood lead to high glucose filtration in the urine which leads to dehydration.[10]

3. Precipitating factors for DKA in pregnancy

The most common cause of precipitation of DKA in pregnancy is the discontinuation of insulin in patients with type 1 diabetes or ketosis-prone type 2 diabetes. Miscommunication and misinformation are often the causes of such a situation.[8]

In some cases, the non-availability of insulin, incorrect insulin delivery, and loss of efficacy of insulin due to poor storage and transport can lead to precipitation of DKA. For those patients with type 1 diabetes on an insulin pump, the disconnection of the pump can lead to DKA.[10]

Apart from this infection is the second most common cause of DKA in pregnancy. Urinary tract infection (UTI) is often a culprit and a urine routine examination must be performed in all such patients. In some cases, hyperemesis graivdarum, new-onset, or poor controlled thyrotoxicosis in pregnancy and antenatal glucocorticoid injection in patients with poorly controlled diabetes can precipitate DKA.[11]

4. Clinical features and diagnosis of DKA in pregnancy

Symptoms of DKA in pregnancy can often mimic other symptoms of pregnancy. The patient may present with incessant vomiting, abdominal pain, and signs of dehydration. It is essential to have a high index of suspicion for recognizing DKA, especially in patients having pre-existing diabetes.[8]

Measurement of serum beta-hydroxybutyrate (BOHB) is sin-quo-non for the diagnosis of DKA.[12] In absence of the same, serum acetone levels of urinary ketone levels may act as a substitute.[13] Currently, we have a point of care devices available in India which help in the assessment of beta-hydroxybutyrate in a matter of seconds.[14]

A blood gas analysis should follow the initial testing for ketone bodies. It is not necessary to perform an arterial blood gas analysis, a venous blood gas analysis may also suffice. Patients typically have metabolic acidosis with a high anion gap.[10]

The patient may have low serum potassium and also hyponatremia. The presence of hyponatremia may be spurious and the sodium levels need to be corrected for glucose levels. [10]

Renal function needs to be assessed and many patients may have pre-renal acute kidney injury because of associated dehydration. A urine function test, not only reveals the presence of glycosuria and ketonuria but also the presence or absence of pus cells in urine suggestive of UTI needs to be assessed. As described earlier, UTI is an important precipitating factor for DKA in pregnancy.[11]

5. Management of DKA in pregnancy

Management of DKA in pregnancy has three main principles: Correction of dehydration, management of potassium, and insulin infusion. [13] Table 2 summarizes the management of DKA in pregnant women.

The first step for the management of DKA is fluid resuscitation. Fluid management precedes insulin delivery and should be aggressive. The initial fluid of choice is isotonic normal saline (0.9% NS). Once the glucose levels fall below 250 mg/dl, the use of Dextrose continuing fluids is encouraged so as to prevent hypoglycemia.[8]

The second step is to assess serum potassium. Since potassium is predominantly an intracellular ion, the serum potassium levels do not adequately represent the total body potassium stores. Patients with DKA is invariability potassium deficient irrespective of the serum potassium levels. It is also important to note that the use of insulin infusion leads to a further reduction of serum potassium levels due to the intracellular shift of potassium. Hence it is important to first assess and correct potassium deficient before initiating insulin infusion.[10]

The third step in the management of DKA is insulin infusion. In most patients, we give an initial bolus of insulin followed by a steady insulin infusion. The infusion is continued till the DKA is resolved and the patient is able to take orally. The use of sliding scale subcutaneous insulin is discouraged. If the patient had been taking long-acting insulin previously, the same may be given in parallel to the insulin infusion.[10]

Bicarbonate infusion generally has little or no role in DKA management. Correction of ketogenesis leads to correction or the metabolic acidosis without requiring bicarbonate. In some cases, bicarbonate infusion may be detrimental.[15]

Obstetric management includes fetal monitoring. The decision to continue the pregnancy should depend on the clinical circumstances and no general guidance can be suggested for the same. As described earlier, DKA can lead to fetal loss in 10-30% of the cases.[8]

The precipitating factor needs to be identified and corrected, especially if it involves any infection. DKA itself leads to leucocytosis and hence an underlying infection may often be missed out in the presence of diabetic ketoacidosis.[8]

Table 2: Summary of Management of Diabetic ketoacidosis in pregnancy

Fluids

- Initial correction with NaCl 1-1.5 litre/hr during first hour
- Then Calculate corrected sodium (Hyperglycemia causes hyponatremia)
  - Measured sodium + 0.024 * (Serum glucose - 100)
  - If Hypernatremia / normal - 0.45% normal saline- 500 ml/hr
  - If hyponatremia- 0.9% normal saline- 500 ml/hr
  - Continue till glucose reaches <250mg/dl - then start 5% dextrose

Insulin

- 10 units IV bolus- check potassium levels first before starting insulin drip
- With INFUSION PUMP
  - Insulin drip – 50 units in 50 ml of normal saline
  - Start is 3-5 ml/hr and titrate based on glucose values
  - The insulin should fall by 50 mg/dl per hour
  - If no fall than double the rate to 10ml/hr – recheck the fall after 1 hr – keep doubling the rate till glucose in target range
  - Continue till glucose <250 mg/dl

Glucose <250 mg/dl

- Start 5% dextrose @ 200 ml/hr
- Continue insulin drip ½ of above rate
- Keep glucose between 150-200 mg/dl

Potassium

- If K <3.3 meq/l
  - Do not give insulin.
  - 2 ampules of 11.2% KCl in 500ml of NS @200ml/hr
- If K - 3.3- 5 meq/l
  - Can start insulin
  - 1 ampule of 11.2% KCl in 500ml of NS@ 200ml/hr
- If K >5.0 – can give insulin without starting potassium
- For maintenance 20-30 meq of K in 1 litre of fluid is generally adequate

Monitoring therapy

- Glucose monitoring every hourly using Point-of-care device
- Every 2 -4 hrs measure the following
  - Serum electrolytes

6. Resolution of DKA

The anion gap is a useful parameter to assess for resolution of DKA. The measurement of ketone bodies (Serum BOHB, Serum acetone, and/or urine ketone bodies) may be misleading. Normalization of the anion gap is a sign of a good

prognosis and suggests a possible resolution of DKA. The criteria for the resolution of DKA are described in table 3.[10]

Table 3: Resolution of DKA

1. Blood glucose <200 mg/dl
2. Serum bicarbonate- >18 meq/l
3. pH > 7.3
4. Anion gap <10 meq/l
Patient is able to take orally without emesis

It is important to note that apart from the high anion-gap metabolic acidosis, the patients with DKA may also have a hidden normal anion-gap metabolic acidosis due to aggressive fluid infusion of sodium chloride. Hence patients may continue to demonstrate metabolic acidosis even after normalization of the anion gap.[8]

Once the patient has a good oral intake and does not have emesis, the insulin infusion may be gradually discontinued while overlapping with subcutaneous insulin. The switch-over though, seemingly innocuous is often the critical part of the process and endocrinologists should be involved in handling the switch of the insulin regimen. If not done right, it can both lead to the recurrence of DKA on one hand and severe hypoglycemia on the other.[13]

Table 4: shows the process of switching from insulin infusion to subcutaneous insulin

Table 4: Switching from Insulin resolution to subcutenous insulin once DKA is resolved

1. Calculate the insulin requirement in the last 6 hours and multiply that by 4 which gives the insulin requirement over the last 24 hours
2. 80% of the total insulin requirement as described above is split into 50% basal and 50% of bolus insulin.
3. The basal insulin is given 3-4 hours before discontinuing the insulin infusion. Insulin detemir and insulin NPH are generally the basal insulin that are safe to use in pregnancy.
4. The bolus dose is divided into three equal doses given before the three major meals. Regular human insulin, insulin lispro and insulin aspart are the various short acting insulin which are safe to use in pregnancy.

7. Preventing recurrence of DKA

The patients with type 1 diabetes and/or ketosis-prone type 2 diabetes should be kept on a close follow-up of an endocrinologist. The use of multi-dose insulin (MDI) an/or continuous subcutaneous insulin infusion (CSII, also called insulin pump) is the standard of care in these patients.[10]

Older generation insulin-like insulin NPH and regular human insulin are cost-effective and are available as essential medications in most government supplies and government drug repositories.[16] Hence all patients with type 1 must be placed on standard MDI therapy with regular glycemic monitor using SMBG (self-monitoring of blood glucose). These patients must have regular access to the medical staff person who has expertise in insulin dose adjustment. Self-adjustment of insulin dose may be taught to selected patients based on their status of education and knowledge.

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COVID-19 in Pregnancy- A hematologist's perspective

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The world is struck by the most dreaded pandemic, i.e., Coronavirus disease (COVID-19) this year. The severe acute respiratory syndrome coronavirus (SARS-CoV2), initially called novel coronavirus, has not spared anyone. Coronavirus is an enveloped single-stranded RNA virus belonging to the family *Coronaviridae* of the order *Nidovirales*[1]. Among them, alpha- and beta-coronaviruses infect mammals. The diagnosis is by detecting viral nucleic acid using real-time reverse-transcription polymerase chain reaction (RT-PCR)[2]. The viral envelope has three structural proteins; out of them, the Spike protein helps coronavirus bind to angiotensin-converting enzyme 2 (ACE2) receptors, thus gaining entry through the respiratory epithelium[3,4]. The pulmonary damage results from immunothrombosis[5]. Pathogenesis involves inflammation, hypoxia, vascular stasis, endotheliopathy, and thrombosis. It predisposes the patient to venous, arterial as well as microvascular thrombosis[5,6]. At cellular level, coagulopathy of COVID-19 involves endothelial injury, activation of platelets, an increase in prothrombotic factors such as tissue factor, suppression of antithrombin, and injury via neutrophil extracellular traps (NETosis)[6]. The immunocompromised patients due to chemotherapy, cancers, cardiovascular diseases, HIV/AIDS, diabetes, chronic renal diseases are particularly susceptible[7]. Such comorbidities increase the risk of severity. Pregnancy is a state of physiological alteration of the immune system. This unique state places pregnant women at an increased risk of coronavirus infection. Morbidity and mortality weekly report (MMWR) study in the US suggests that pregnant women with COVID 19 are more likely to be hospitalized and are at increased risk for ICU admission and mechanical ventilation than non-pregnant women[8].

COVID-19 in pregnancy has different implications than non-pregnant women. Apart from physiological differences, there is a risk of vertical transmission, and management of labor is an added problem for obstetricians.

Immunological Alterations

The first- and third-trimesters of pregnancy are pro-inflammatory states that help implantation, placental development, and initiation of parturition, respectively. The T cell subsets like Tregs, naive T cells, and memory T cells gain the function of downstream signaling proteins like STAT5.[9] There is a delicate balance between tolerance for the fetal antigens and protective immunity against viral antigens. Therefore, the innate immune players like monocytes and NK cells are activated during pregnancy, while adoptive immune responses are suppressed. Moreover, high levels of estrogen and progesterone cause edema of the upper respiratory tract, niche for viral pathogen.[9]

Pregnancy complications can be caused by direct effects of the virus and antibody dependant enhancement (ADE).[10] This means there is an enhanced immune response to SARS-CoV 2 due to antigenic similarities with previous viral infections. Severe infection can lead to cytokine storm with increased production of inflammatory cytokines like IL-1, IL-2, IL-6, TNFα, MIP, MCP.[11] These could be associated with complications such as spontaneous abortion, preterm delivery, intrauterine growth retardation. Induction of IL17 due to viral infection can lead to neuronal dysfunction and behavioral changes in the child. All these lead us to suggest that maternal morbidity and mortality may be higher in pregnant than non-pregnant COVID-19 patients. It has been postulated that there is hardly any evidence of hematogenous spread in SARS CoV2; hence the risk of vertical transmission infection is minimal.[12]

Hematological changes

Coagulopathy- Coagulopathy in COVID-19 neither befits disseminated intravascular coagulation (DIC) nor sepsis-induced coagulopathy (SIC). There is an interplay of thrombosis and inflammation in COVID-19.[13] Role of thrombosis in pregnant

COVID-19 patients is highlighted by a study where placentas from patients showed increased placental injury and decidual arteriopathy. This reflects abnormalities in oxygenation within the intervillous space and is associated with adverse perinatal outcomes.[14] Pregnancy is itself a hypercoagulable state. There is an increase in procoagulant factors and up to 3-fold rise in D-dimer levels. Plasma fibrinogen increases, and mild thrombocytopenia can occur in a healthy pregnancy. There is an increase in factor VIII, von Willebrand factor levels (VWF), and a fall in protein S in normal pregnancy. These findings can confound coagulopathy related to COVID-19 in pregnancy. Laboratory parameters such as Prothrombin time (PT), Activated partial thromboplastin (APTT) time will be shortened in a normal pregnancy due to a rise in procoagulant factors.

In general, ISTH recommends admission for COVID patients with a 3- to 4-fold rise in D-dimer even if asymptomatic[15]. As D-dimer values are already high in the third trimester of pregnancy, more studies are needed to establish cut-offs for D-dimer levels in COVID-19 pregnancy. Initial prolongation of PT, APTT values may be masked in COVID-19 in pregnancy. The first reported acutely progressive coagulopathy in severe COVID-19 was described in April 2020[16]. The authors suggested APTT and fibrinogen testing, in addition to D-dimers, PT, and platelet count in all patients as they may add diagnostic and risk stratifying value.[16]

Blood Counts and Inflammatory Markers

Lymphopenia predicts disease severity.[17] Pathogenesis of lymphopenia involves their direct infection due to the presence of ACE receptors, destruction of the lymphatic organs by the virus, lymphocyte apoptosis due to altered cytokine balance and metabolic alteration like hyperlactic acidemia that can inhibit lymphocytic proliferation.[17]

In a study of 116 pregnancies, Yan et al. analyzed hematological parameters on admission. They found lymphopenia in 44%, leukopenia in 24.1%, and increased C-reactive protein (CRP) in 44%. Eight (6.9%) cases had severe disease. Severity correlated with leukopenia and lymphopenia. Severe cases had lymphopenia in the range of 300-1420/µl. Mild thrombocytopenia was observed in one case. Elevation of PT, APTT was associated with elevation of transaminases.[18] Chen et al. observed that patients had decreased lymphocyte count and increased hypersensitive CRP.[19] In his study, 55% had lymphopenia, 66% had elevated concentrations of CRP, and 33% had increased levels of ALT and AST amongst nine pregnant women with COVID-19 pneumonia.[19] Also, 77% of patients had a normal WBC count. The leukocytosis and elevated neutrophil ratio were reported to be more common in the COVID-19 infected pregnant women in Liu H, et al. study[20]. However, they did not find significant differences regarding lymphopenia among pregnant and non-pregnant groups. They also found CRP elevation in most of the cases[20]. In the study of Wang X, et al., laboratory findings included high leukocyte count, elevated neutrophil ratio, lymphopenia, and elevated CRP, D-dimer, and LDH. However, aminotransferase levels and creatinine were reported in a normal range[21]. Moreover, Liu W et al. reported leukocytosis and elevated neutrophil ratio, elevated CRP and interleukin 6, and low albumin, while the levels of ALT, AST, ferritin, and ESR were the normal[22]. So high CRP, leukocytosis, and elevated neutrophil ratio were found in most studies on COVID-19 pregnant women, and other laboratory tests are conflicting[23]. However, physiological changes in pregnancy involve leukocytosis and high neutrophil ratio and this complicates the interpretation. Though platelet count remains normal in most cases, severe thrombocytopenia has been reported[24]. Comprehensive data on a larger population of pregnant women with COVID-19 are needed to better understand the hematological parameters and their role in maternal and birth outcomes.

Role of Hematological Parameters in Management

Lymphopenia and raised CRP are more common findings in COVID-19 infected pregnancy. D-dimer levels, raised fibrinogen levels, leucocyte count and neutrophil ratio and their cut offs are debatable. Also, if there is elevation of leucocyte count with neutrophilia, then sepsis should be evaluated[28].

Though PT, APTT may be prolonged, this should not deter neuraxial block as coagulopathy in COVID-19 does not increase the risk of bleeding[25]. It is recommended to administer LMWH to

COVID-19 infected symptomatic pregnant women. Obstetricians should be mindful of the interaction of LMWH and management of labor/ neuraxial block to minimize the risk of bleeding[25]. Though there may be a prolongation of PT, APTT, sole prolongation without major bleeding is not an indication of fresh frozen plasma transfusion[26]. COVID-19 may be associated with transaminitis, elevated creatinine, and thrombocytopenia. It is crucial to distinguish preeclampsia, hemolysis, elevated liver enzyme levels, and low platelet count (HELLP) syndrome from the manifestation of COVID-19 in hypertensive patients[27].

There is a lack of data on coagulopathy in pregnancy and COVID-19. Therefore, International society of thrombosis and hemostasis (ISTH) has commenced an international registry including confirmed cases of COVID-19 in pregnancy in all trimesters and study their hematological and coagulation parameters and their effect on pregnancy outcome.

The practicing guidelines based on multicentric studies or clinical trials on pregnancy with COVID-19 are largely deficient in India. We have one of the largest cohorts of pregnant females who are susceptible to COVID-19. Thus, the need of the hour is for all obstetricians of our country to come up with recommendations suitable in our setting.

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A Case of Recurrent Postpartum Pulmonary Oedema

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Introduction

Acute pulmonary edema in pregnant woman in postpartum period is a rare but life-threatening event & causes significant mortality and morbidity. Preeclampsia remains an important cause of acute pulmonary edema with an incidence of 3 % overall & out of which 70 % cases occur in postpartum period. It occurs most frequently 48-72 hours postpartum, probably due to mobilization of extravascular fluid in that period. Increased hydrostatic pressure, decreased colloid oncotic pressure & increased capillary permeability are the predisposing factor in preeclampsia for the development of pulmonary oedema. We report a case of postpartum pulmonary oedema in PIH patient on day 9 with similar event in pervious pregnancy.

Case Presentation

A 39-year female G2P1L1 booked case came to OPD (Indraprastha Apollo Hospitals) at 34 weeks and 3 days of gestation came with complains of breathlessness Grade 3 and headache of 2 days duration. She also complained of sudden increase in bilateral Pedal edema, facial puffiness for the past 3 weeks and the gain of 6 kg of weight within last 2 weeks.

Previous Pregnancy 7 Years Back

In previous pregnancy at 24 weeks, patient was diagnosed with gestational hypertension and GDM in 3rd trimester she developed Intrahepatic Cholestasis. At 37 weeks, she developed mild breathlessness and got admitted, delivered by LSCS in view of Short stature with borderline CPD with good size baby with PIH and GDM on insulin. Procedure went uneventful. (Baby weight 3.1 kg) Total 20 units Oxytocin was given. Fluid intake approx. 2880ml @120ml/hr, urine output approx. 2100ml in 1st 24 hr. Pre-Pregnancy weight was 65kgs and on admission weight 85kg. Net gain of 20kgs.

On her 3rd post-operative day, the patient suddenly started complaining of breathlessness and her SpO2 dropped to 75% in room air, on chest examination bilateral crepts were present with reduced air entry. O2 started at 4L/hr and shifted to ICU as she could not maintain her oxygen saturation for further evaluation and management. Chest X Ray showed prominent right para hilar and paracardiac bronchovascular marking and mild pleural effusion. Computed tomography (CT) suggested pulmonary interstitial edema. Her ECG, Echocardiogram, Venous Doppler lower limb, Coagulation profile (D- Dimer – 0.3) were normal. She was managed in ICU with O2 supplement, fluid restriction, Antihypertensive, Injection Furosemide and DVT prophylaxis given. Patient was discharged on 7th postoperative day in stable condition and with normal SpO2 level at room air. On a follow-up visit, 3 weeks after discharge, the patient had no complaints. Her blood pressure remained normal without blood pressure medication and her, Echocardiogram & Chest x ray were normal.

In this Pregnancy

After confirming single viable pregnancy at 6 weeks, she was put on tab Aspirin 150mg OD. At 9 weeks of gestation her BP found to be 140/90mmhg hence, tab Labetalol 100 mg BD was started, by 30 weeks of gestation she was on labetalol 200mg TDS and Tab Amlodipine 5 mg OD to control her BP. At 24 weeks of gestation diagnosed with GDM and was on diet control. She also developed Intrahepatic Cholestasis by 30 weeks of gestation. At 34 weeks and 3 days she came with breathlessness, headache, pedal oedema and sudden weight gain. On examination her BP- 140/94 mmhg, Net weight gain of 13kgs noted. She had 2+ proteinuria, her ultrasound suggested fetal growth restriction with

fetal weight 1.9kg with normal liquor and doppler.

Patient was admitted, corticosteroid cover given., All her routine test were normal except S.Uric Acid-7.8. Her BP remain fluctuating, with persistence breathlessness, headache and with off and on blurring of vision hence, decision of delivery was taken. A 1.9 kg baby delivered by LSCS + bilateral Tubal Ligation done. In view of atonic PPH 30 units Oxytocin along with Injection Prostodin was used.

On 1st postoperative day, patient was on Oxygen 3-4L/hr since SpO2 84%, she could not maintain SPO2 at room air. IVF restricted to 60-80ml/hr, overall approx. 1800ml of fluid was given. Urine Output was 1390ml. Tab Labetalol continued since her BP was high. Injection Furosemide and DVT prophylaxis was also given. Due to her previous history of pulmonary oedema, 2Decho and Chest X ray done and found normal. She was on O2 supplement for next 3 days since her SPO2 was not maintained at room air. On day 5, patient was comfortable and Maintained SPO2 95% at room air hence, she was discharged in stable condition on day 6.

After discharge, at home on 9th postoperative day patient developed severe breathlessness and SpO2 drop till 70% Room Air. She was on Oxygen, fluid, salt restriction and Injection furosemide was given. Chest x-ray showed Pulmonary oedema. Later she recovered in 3 days. On 12th day she weighed 82 kgs. (loss of 11 kgs noted)

Discussion

Postpartum pulmonary oedema in pre-eclampsia itself is a rare phenomenon & more so a recurrent one. But its occurrence on 9th postoperative day is not only rare but enigmatic too. Iatrogenic fluid overload in preeclampsia is one of the most important causes. Fluid redistribution from the systemic to pulmonary circulation due to vasoconstriction (due to sympathetic system activation) even in euvolemic patient is now recognised as one of the factors too. Higher incidence of pulmonary edema is observed in women with increased maternal age, obesity, caesarean delivery, tocolytic therapy (F. Afridi_et al 2020) and chronic hypertension with superimposed preeclampsia 7.1% as compared to l. 7% in those with pure pre-eclampsia

Restricted fluid administration of 2100ml on peripartum period has no report of adverse outcome. While relative risk of pulmonary oedema with 5000ml of peripartum fluid administration was 1.9. (Thornton CE et al 2011) So pulmonary oedema happening in our patient in her first pregnancy postpartum with normal IV Fluid intake on postoperative Day 3 can be easily explained but with fluid & salt restriction, Furosemide therapy, close observation its occurrence on 9th postoperative day is rare one.

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Antenatal Severe Peripartum Cardiomyopathy (PPCM) with Prophylactic Use of Intra-aortic Balloon Pump During Caesarean Delivery

Sohani Verma, Vivek Gupta (Invasive Cardiologist)

Introduction

PPCM is a potentially life threatening condition and defined as- *"an idiopathic condition presenting with left ventricular (LV) systolic dysfunction (ejection fraction- EF <45%. There may or may not be ventricular dilatation) towards the end of pregnancy or in the months following delivery, when no other cause of heart failure (HF) is found"* (Heart Failure Association of the European Society of Cardiology Working Group –ESC 2010).

The intra-aortic balloon pump (IABP) is a mechanical device used by invasive cardiologists for mechanical cardiac support in heart failure patients

for a short duration usually as a bridge to recovery or alternative procedure. Although it is used not so infrequently in non-pregnant female and male patients, there are only limited case reports of the use of IABP counterpulsation for circulatory support in pregnant women with cardiac failure.

Case Summary

We present a rare case of antenatal severe peripartum cardiomyopathy in a 25 years old woman P0G2A1 at 31 weeks gestation. The patient presented at the emergency department of Indraprastha Apollo Hospitals (IAH) in February 2020. As per the history provided, patient was alright till one week back and then developed shortness of breath and palpation. Her dyspnea progressed quite rapidly and on admission patient had severe dyspnea of Grade III-IV NYHA. She also had complaints of anxiety, weakness, fatigue and reduced urine output since last 2-3 days. There was no history of any pre-existing cardiac or respiratory disease, hypertension etc. She had uneventful antenatal progress, was perceiving good foetal movements and had no history of abdominal pain or any abnormal vaginal discharge. A 2D Echocardiography done outside Apollo had showed - Global hypokinesia, Left Ventricular Ejection Fraction (LVEF) -28%, moderate to severe MR and, left atrium dilated, hence presented at IAH.

She was admitted in CCU under cardiology and high risk obstetric team and managed by a multidisciplinary (MD) team which also included Internal Medicine, Nephrology, and Psychiatry specialists. Despite standard medical management for heart failure and high doses of ionotropic support over next four days, patient's cardio-respiratory condition did not improve. In view of worsening signs (LVEF-25%) and hemodynamic instability, case was discussed in the MD team meeting and also with the patient and family. A shared decision was taken for immediate LSCS delivery. Patient had already received a course of corticosteroids before admission. As caesarean delivery was highly likely to cause sudden cardiac decompensation and significant risk to mother's life (estimated mortality risk 20%-30%), various options to reduce the risk were explored and discussed among the MD team and family. It was decided to use intra-aortic balloon pump (IABP) counter-pulsation for the mechanical cardiac support and same was inserted just before the LSCS surgery by Dr Vivek Gupta. A planned high risk LSCS under GA was performed on D5 after admission. Indication for LSCS - severe PPCM with congestive heart failure and hemodynamic instability refractory to the medical management. Surgery was uneventful. A live female baby of 1.6 Kg weight was born and had Apgar Scores of 8/9. With the help of IABP counter pulsation, patient remained hemodynamically stable during and after surgery. Baby was shifted to NICU due to prematurity. Baby had no major problems. Expressed breast milk was started and gradually increased to full feeds by D5 of life. Baby was discharged home in good condition on D14.

Intra-aortic balloon pump was removed 2 days after delivery. Standard management of heart failure along with LMW Heparin was continued. Patient and family were counselled regarding Bromocriptine therapy, but it was refused as patient was extremely keen on breastfeeding. Patient had no surgical complications, but her cardio-respiratory status showed very little improvement. She was discharged home 14 days after LSCS on medications for chronic cardiac failure including Inj Clexane. A 2-D Echocardiography and Color Doppler on the day of discharge showed- normal mitral and aortic valve, dilated LV and normal LA. There was moderate to severe global LV hypokinesia with moderate to severely reduced global LV systolic function LVEF=30%. Good RV systolic function. No LV thrombus.

At the post-natal visit, contraception was emphasized and safe options (Mirena /Inj DMPA / Barrier method) discussed (Estrogen contraindicated). Couple were also counselled about serious risks involved in any future pregnancy. Patient remains under close follow up by the cardiologist (mostly through tele-consulation due to current COVID situation) and cardiac medications are being continued. The patient's cardiac recovery remains poor till date. Her latest LVEF, six months after the onset of PPCM is still only 25-30%.

Discussion

PPCM is a global disease with an estimated incidence of 1 in 1000 to 1 in 10,000 live births. About one third cases occur before delivery and two third cases post-delivery. 44% cases occur within first month after delivery. PPCM can be associated with

severe adverse outcomes including brain injury, cardio-pulmonary arrest, pulmonary oedema, arrhythmia, thromboembolic complications and death with reported mortality rates between 6%-32% (Azibani F et al 2018, Davis MB et al 2020). Although some women have relatively mild disease and complete recovery, others experience significant morbidity and mortality. Stable patients are delivered vaginally unless there are obstetric reasons for a caesarean section. However, early delivery or termination of pregnancy should be considered in case of hemodynamic instability. (Davis MB et al 2020). Thromboembolism is the most common severe complication of PPCM affecting around 5% to 9% of all cases. LV thrombus has been identified in as much as 10% to 17% of initial echocardiograms. Ionotropic support can be used for short duration only. Studies suggest that both nor-adrenaline and dobutamine (β agonist) may be detrimental to myocardial recovery with poor long term results (Honigberg MC et al 2019).

In the case presented, despite high doses of ionotropic support, patient's hemodynamic condition did not improve, therefore decision was taken for LSCS at 31weeks gestation. It has been suggested that unstable patients may benefit from invasive hemodynamic optimization prior to delivery and monitoring during delivery and the early postpartum period (Davis et al 2020). IABP counterpulsation was used in this case as a prophylactic measure to reduce the anticipated risk of decompensation and optimize haemo-dynamic state during critical period of delivery and immediate post-partum. The intra-aortic balloon pump is a mechanical device. Once it is placed in the aorta, the balloon inflates and deflates via counterpulsation, meaning it actively deflates in systole and inflates in diastole. These actions combine to decrease myocardial oxygen demand and increase myocardial oxygen supply. Very few cases of the use of IABP in pregnant women are reported in literature (Samalavicius R et al 2018). A multi-disciplinary approach involving obstetrician, invasive cardiologist and a cardiac anesthetist, is essential to achieve optimum outcome.

Based on the role of prolactin in pathogenesis of PPCM, several studies have advocated Bromocriptine in the management of PPCM and reported better myocardial recovery and reduced mortality rates (Davies et al 2020). However, the role of bromocriptine as a therapeutic agent in PPCM is currently experimental. If used, therapeutic anticoagulation is recommended, as it is prothrombotic and it suppresses lactation. In the case presented, post LSCS patient and family were counselled regarding bromocriptine therapy, however, patient was extremely keen to breast feed her baby and refused. Patient remains in chronic cardiac failure (LVEF 25%-30%) even six months after delivery. Left Ventricular Assist Device (LVAD) or cardiac transplant or both, may be further options in this case.

Further research about use of bromocriptine, mechanical device therapy, continued drug therapy and long term outcomes are needed.

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Fetal Tuberous Sclerosis and in Utero Therapy

Akshatha Sharma, Anita Kaul, Madhu Roy

Introduction

The incidence of tuberous sclerosis is 1/6000 births. A constellation of findings such as cardiac rhabdomyomas, cortical, renal, dermatological manifestations are diagnostic of Tuberous sclerosis. In utero, Rhabdomyomas can be isolated or multiple and associated with a host of other extracardiac findings that suggest a diagnosis of Tuberous Sclerosis in utero. We report a case of fetal tuberous sclerosis and a novel in-utero therapy that was initiated due to the rapid growth of the rhabdomyoma.

Case Report

A 35 year old pregnant lady presented to us at 28 weeks of gestation. This was her third pregnancy. The first pregnancy was a normal uneventful

delivery with a male child who was 7 years old ,alive and healthy. The second pregnancy was a caserean for fetal distress, this male child was diagnosed with leukaemia and succumbed to it at 4 years of age. In this pregnancy, her first trimester screening and an anomaly scan done elsewhere was reported to be normal. At her 28 weeks scan, her fetal growth was on the 10th centile. Amniotic fluid and fetal Dopplers were normal. A prominent, echogenic, papillary muscle was noted in the left ventricle. A review was suggested for growth and heart in 4 weeks. At 32 weeks, the growth was maintained on the 8th centile however the echogenic structures had now evolved into multiple rhabdomyomas across the interventricular septum, left ventricular and right ventricular wall, the largest measuring 6x 5x 6mm along the left ventricular outflow tract. A detailed neurosonogram at our neuro clinic revealed multiple cortical tubers and subependymal nodules. Bilateral kidneys showed cysts. An MRI confirmed the findings alongwith the renal lesions suspected to be angiomyolipomas.

The couple was counselled that the multiorgan involvement was suggestive of tuberous sclerosis and would require multidisciplinary management. An amniocentesis for TSC mutations(causative for tuberous sclerosis) was suggested but the couple declined. Paediatric cardiologist, paediatric neurologist, neonatologist and the obstetrician were involved in discussion and management of the pregnancy. They were informed that the neurological ultrasound findings could lead to seizures/neurodevelopmental delays in upto 60% of the cases. The cardiac rhabdomyomas usually regress after birth however the rapid increase in the rhabdomyomas especially in the left outflow tract was of concern and could lead to obstruction or rarely arrhythmias. If the renal angiomyolipomas increased beyond 3 cm, postnatal sirolimus would be recommended.

We discussed the options of a novel in-utero medical therapy with Sirolimus to contain the growth of these rhabdomyomas. After a detailed counseling, the couple was agreeable to the plan and under the renal transplant specialist's guidance, to minimize the side effects ,l owest dose of 3mg /day of Sirolimus was initiated. Maternal blood counts, LFTs, urine proteins and serum levels of sirolimus were checked in a week. The dose of sirolimus was doubled to achieve the therapeutic level. At the end of 4 weeks of therapy, the cardiac rhabdomyomas showed regression thus allaying the fear of obstruction of the left outflow tract.

The therapy was stopped at 36+5 weeks and delivery planned at 37 +5 weeks in view of fetal growth restriction and rising umbilical artery resistance. A 2.6 kg girl was delivered by LSCS with APGARS of 9,9. Postnatal imaging confirmed the findings. The baby was discharged with a plan for follow up at 3,6months and 1 year for review of the lesions. A genetic consultation was advised.

Discussion

Tuberous sclerosis is caused by mutations in the TSC1 and TSC2 gene. These genes inhibit the mTOR protein that is responsible for cell growth. Mutations in these genes therefore lead to abnormal cell proliferation leading to overgrowth of cardiac myocytes(rhabdomyomas), abnormal differentiation of neuronal cells(tubers),renal angiomyolipomas and other angiofibromas (dermatological manifestations). Sirolimus is a drug that inhibits the mTOR protein and thus prevents cell proliferation. Postnatal use of sirolimus is well established in refractory cardiac rhabdomyomas, giant cell astrocytomas causing ventricular obstruction and large renal angiomyolipomas. This therapy was described in four different antenatal case reports by Barnes et al(2018,NEJM); Vachon, Marceau et al (2019,UOG); Park et al (2019, Obstet Gynecol Sci); Pluym (2019, Prenatal Diagnosis) where its use shrunk the cardiac rhabdomyomas thus permitting prolongation of pregnancy. The side effect profile should be kept in mind and the pregnant woman should be monitored for these effects. In our case, other than mild mucositis that was treated with oral lignocaine gels, the drug was well tolerated. The regression of the cardiac rhabdomyomas in our case supports the review of literature that this novel therapy can be considered in cases at high risk of outflow obstruction that could cause hydrops or demise. Finally, genetic testing is always recommended as 1/3 of these cases could be familial (or germline mosaicism) with need of parental testing (Autosomal dominant inheritance) so that recurrence be predicted and future pregnancies be screened early. Importance of postnatal follow up (such as EEG, Imaging,

sensitization of the parents to infant seizure signs and symptoms)at above mentioned intervals needs to be emphasized in these cases such that postnatal therapy(Antiepileptics, sirolimus, etc) can be timely initiated.

Conclusion

1. Detailed scan/follow up of any suspicious lesions in the third trimester.
2. In utero Sirolimus therapy for Cardiac Rhabdomyomas can be considered if rapidly growing in size or deteriorating cardiac function.
3. Insufficient evidence for regression of in-utero brain lesions.
4. Genetic testing of the family is recommended
5. Multidisciplinary approach balancing the risks vs benefits for in utero treatment must be initiated.

World's Largest Ovarian Tumour!!

Arun Prasad, Abhishek Tiwari
Geeta Chadha, Nilima G

Mrs X, a 52 years old postmenopausal lady, was referred to our hospital with complaints of progressive abdominal distension which had worsened over the past 4-5 months. She attained menopause one and half years back and had regular cycles prior to that. Her last childbirth was 18 years back. She does not have any other significant medical or surgical history.

CT abdomen revealed a huge cyst extending from the pelvis till the xiphisternum, measuring 41 x 38 x 35 cm. It was a multiseptate cyst which was compressing and displacing the bowel loops. Ca 125 was 35 U/ml. Decision was taken for doing exploratory laparotomy followed by frozen section. Preoperatively 2 units of PRBC were transfused as patient's hemoglobin was 6.8 g/dl.

Intraoperatively dense adhesions were seen between the cyst wall and the adjacent structures with complete distortion of the anatomy. Upon opening the cyst wall which was fused with the peritoneum, up to 80 litres of chocolate brown fluid was evacuated. Further dissection was carried out after the decompression of the cyst. The cyst wall had to be removed piecemeal as it was adherent to several vital structures and then sent for frozen section. Total abdominal hysterectomy and bilateral salpingo-oophorectomy was done. Frozen section was reported as a benign cyst. Prophylactic diversion ileostomy was done after abdominoplasty. Intra operatively 4 units PRBC'S were transfused. Patient recovered well in the postoperative period. Final histopathology was reported as endometriotic cyst.

Review of literature revealed that while a few such cases of huge ovarian endometriomas have been reported in the past, this is the largest ovarian cyst reported till date. Another interesting feature is that such huge endometriomas are rarely seen in the postmenopausal age. Hence in such cases the gynecologist should always consider the possibility of a malignant ovarian tumor but endometriosis too should be included in the list of differential diagnosis.

Proceedings of AOGD Monthly Clinical Meeting held at Deen Dayal Upadhyay Hospital, New Delhi on 25th September, 2020

An Intriguing Case of a Rare Abdominal Tumour

Urvashi Miglani, Gargi Chowdhary, Poonam Laul
Neeta Bindal, Pinkee Saxena, Pratibha Nanda
Shashi Kabra, Usha Yadav

ESS is a malignant mesenchymal tumour arising from the uterus. It rarely may occur in the extrauterine location where it is called endometrioid stromal sarcoma [also known as extrauterine endometrial stromal sarcoma (EESS)]. Patient Ms. X 20-year-old unmarried female presented to the outpatient department of a tertiary hospital in July 2018 with the complaint of pain in lower abdomen for 1 week. MRI pelvis showed a large complex space occupying lesion with mixed cystic and soft tissue component measuring 7.2 by 8.3 by 6.8 cm showing non-homogeneous contrast enhancement. Per-operatively a highly vascular 10 by 20 cm cystic mass with necrotic and haemorrhagic areas was present which was densely adherent to omentum and mesentery. Uterus with bilateral tubes and ovaries was normal, and mass was arising from left round ligament. The final pathological diagnosis was given as stromal sarcoma of the round ligament with associated endometriosis.

Extrauterine endometrial stromal sarcoma (EESS) is an uncommon tumour that occurs in women over a wide age range. The extrauterine location, non-gynaecologic symptoms and signs at presentation, and confounding histologic features can pose a diagnostic challenge. In view of the rarity of these tumours, they need to be differentiated from spindle cell tumours of the abdominal peritoneum such as GIST (gastrointestinal stromal tumour) and leiomyosarcoma. Immunohistochemical stains are useful to distinguish these two entities. The typical immunohistochemical profile for endometrial stromal sarcoma is positivity for vimentin, CD10, and estrogen receptor, and negativity for c-kit, CD34, cytokeratin, and smooth muscle markers. GIST is known to stain diffusely for CD117 and CD34. Surgery is the mainstay of treatment. The effective duration of preventive hormonal therapy is still undetermined. The survival benefit of radiation therapy and chemotherapy in adjuvant setting is not clear. This case highlights the presence of such rare tumours masquerading as ovarian masses. Also, in light of limited evidence available for adjuvant therapy in this scenario, a multidisciplinary approach in concurrence with medical and radiology oncologist may go a long way in improving the survival. An intriguing case of a rare abdominal tumour

An Unusual Presentation of Vaginal Fibroid

Sunita Seth, Soma Mitra, Rita Ranjan, Ritu Goyal
Harvinder Kaur, Usha Yadav

Vaginal mass can present with a varied symptom. It is always difficult to diagnose and management is challenging. Vaginal leiomyomas are rare smooth muscle tumours. It is histologically diagnosed as interlacing fascicles of spindle cells with whorled pattern. It is morphologically similar to uterine leiomyoma. We report a case of vaginal leiomyoma arising from anterolateral wall of vagina presenting as a necrotic mass coming out per vagina, pain abdomen with vaginal bleeding. Preoperative evaluation done with routine investigations, USG and MRI. Enucleation of mass done through perineal route along with laparotomy with dissection of course of ureter.

Placenta Accreta Spectrum Disorder-Case Series

Shashi L Kabra Maheshwari, Pinkee Saxena
Harvinder Kaur, Neeta Bindal

PAS (placenta accrete spectrum disorder), refers to a spectrum of abnormal placental adherence ranging from microscopic finding of adherent myometrial fibres within the basal plate to a dramatic presentation of placenta percreta, where there is placental invasion through the uterus and

the serosa into the peritoneal cavity or bladder.

A Prospective Observational study was carried out to to observe the fetomaternal outcome in Placenta Previa and PAS and to find the predictive value of Tovbin et al score in diagnosis of PAS.

Material & Methods: 35 Antenatal female with > 28 weeks of gestation with bleeding PV and or diagnosed with placenta previa and PAS on USG were recruited. Scoring done by Tovbin et al score (based on clinical and USG parameter) to determine their probability of PAS. Fetomaternal outcome noted.

Results: Tovin et al score had high predictive value in diagnosing PAS. In PAS group 9 patients required hysterectomy and ICU admission and there were 3 mortality. In placenta praevia group 7 patients required ICU admissions and there was no mortality. In PAS group there were 6 preterm babies with no neonatal death. In placenta praevia group there were 18 preterm with 2 neonatal deaths.

Case 1: A 21-year-old, G3 P2, previous 2 lscs, 28 weeks pregnancy presented to emergency with abdominal pain, vaginal bleeding and h/o expulsion of fetus at home. Oxytocin started. Patient had persistent, active bleeding for which decision of laparotomy taken. Per op –Placenta was found to be protruding through the lower uterine segment involving the bladder. Total abdominal hysterectomy performed with bilateral internal iliac artery ligation. But generalized oozing was still present for which tight packing of abdomen was done with full drape sheet. Patient received transfusions. Relaparotomy was done later for removal of drape sheet. HPE suggestive of placenta percreta. Highlights- Importance of packing when all measures fail.

Case 2: 26 yrs old, P1, previous LSCS, 38 +3 weeks pregnancy was admitted with USG report of low lying placenta accreta. LSCS was planned. Per op - Placenta was invading and extending up to the bladder. Baby delivered. Bilateral internal iliac artery ligation done, followed by hysterectomy with placenta in situ. Patient received transfusions and had uneventful recovery. Highlights- Good outcome in planned cases.

Case 3: 28 yrs old, G2 P1 L1, previous LSCS with 37 weeks pregnancy referred from peripheral hospital, with bleeding PV. USG showed anterior low lying placenta covering the OS. Emergency LSCS was done. Placenta found invading into serosal layer of bladder. Obstetric hysterectomy performed during which bladder was injured. Bladder repaired, drain put and transfusions given. Next day drain had 700 ml fresh blood. Reopened in view of hemoperitoneum. Generalised oozing from bladder base and pedicles sutured again. Internal iliac artery ligation performed. Patient shifted to ICU and received massive transfusion. However patient later developed AKI & MODS and died. Highlights- Any case with previous LSCS can turn out to be PAS.

Conclusion: Prenatal diagnosis of PAS is of paramount importance. Multidisciplinary team and planned approach can help to decrease surgical complications, maternal blood loss and prolonged intensive care unit admissions. Treat all cases of previous LSCS with anterior low lying placenta as PAS until proved otherwise. One should always aim to reduce the incidence of primary caesarean section.

The election for the post of President & Vice President of AOGD has been declared.
Dr. Kiran Guleria Dir. Prof. UCMS & GTB Hospital has been declared the returning officer.
AOGD will take help from FOGSI for the purpose of election.

Congratulaitons to Winners of FOGSI Awards

S.No.	Name of Awards	Winners
1.	FOGSI - Dr. Mehroo Dara Hansotia Prize for the best work done by Committee of FOGSI 2020	Chairprerson – FOGSI Food Drugs and Medicosurgical Equipment – Dr. Vidya Thobbi, Bijapur
2.	FOGSI - Dr. Vasantben Shah Scholarship for Overseas Study 2020	Dr. Ritu Hinduja, Mumbai
3.	FOGSI - Travelling fellowship 2020 (2 Fellowships)	Dr. Purnima Tiwari (Gupta), Bhopal Dr. Deepthi Nayak, Pondicherry
4.	FOGSI - Late Dr. Pravin Mehta training fellowship in Laparoscopy 2020	Dr. Rohan Palshetkar, Mumbai
5.	FOGSI - IAEC Sun International Travelling Fellowship 2020	Dr. Priyankur Roy, Siliguri
6.	FOGSI - Dr. Duru Shah Distinguished Community Service award 2020	**Dr. Sonal Bhatla, Delhi**
7.	FOGSI - Dr. R. D. Pandit Research Prize 2020	**Dr. Ankita Jain, Delhi & Dr. Chingbiaklun Shoute, Delhi (TIE)**
8.	FOGSI - Dr. Kumud P. Tamaskar Prize 2020	Dr. Niharika Malhotra, Agra & Dr. Rohan Palshetkar, Mumbai (TIE)
9.	FOGSI - Dr. D C Dutta prize 2020 for best publication	1. Text Book (A): Dr. Mala Arora & Dr. Monika Gupta 2. Hand Book (B): Dr. Poonam Yadav, Agra 3. FOGSI FOCUS (C): Dr. Nandita Paishetkar, Dr. Hrishikesh Pai, Dr. Rishma Dhillon Pai and Dr. Paratik Tambe, Mumbai
10.	FOGSI - Dr. Kamini A. Rao orator for the year 2020	**East Zone:** Non application received **West Zone:** Dr. Munjal Pandya, Ahmedabad Society **North Zone:** **Dr. Avantika Gupta, Delhi Society** **South Zone:** Dr. Jeevitha K.J., Mangalore Society
11	FOGSI - The Padmabhushan Kamlabai Hospet Award 2020	No suitable Application

12	FOGSI - Late R. B. Dr. S. N. Malhotra appreciation award 2020	No suitable Application
13	FOGSI Movicol (Corion) Awards 2020	**Senior Category** **Winner:** Dr. Sweta Singh, Odisha **1st Runner up:** No suitable Application **2nd Runner up:** No suitablel Application **Junior Categroy** **Winner:** Dr. Namrata Bhattacharya, Bengal **1st Runner up:** Dr. Neha Agarwal, Agra **2nd Runner up:** Sneha Sathe, Mumbai
14		
15	The FOGSI, IPAS, Young Talent Promotion Committee and MTP Committee Award 2020	Dr. Rana Choudhary, Mumbai
16	FOGSI - Dr. Nimish R. Shelat Research Award 2020	Dr. Priyankur Roy, Siliguri
18	FOGSI - Late Prof. D. Kutty Life Time Achievement Award 2020	Dr. Jayantibhai I. Patel, Baroda
19	FOGSI - Mr. N. A. Pandit & Mrs. Shailaja N. Pandit Women's Empowerment Award 2020 (2 awards)	Dr. Sadhana Gupta, Gorakhpur Dr. Shyjus P., Cannanore
20	FOGSI - Dr. Shanti Yadav Award in Infertility (3 awards)	**Winner:** Dr. Neharika Malhotra, Agra **1st Runner Up:** **Dr. Divya Pandey, Delhi** **2nd Runner Up:** Dr. Priyankur Roy, Siliguri
21	FOGSI - Dr. Rajat Ray Award in Fetal Medicine (3 awards)	**Winner:** **Dr. Kiran Guleria, Delhi** **1st Runner Up:** Dr. Gaana Sreenivas, Bangalore & **Dr. Nutan Agarwal, Delhi (TIE)** **2nd Runner UP:** **Dr. Chanchal Singh, Delhi**
22	Winner of the best paper publishedin FOGSI Journal during the year 2018 in Senior Category. (3awards)	**1st Prize:** Dr. Vasundhara Kamineni, Nalgonda **2nd Prize:** Dr. Hemant G. Deshpanda, Pune **3rd Prize:** **Dr. Taru Gupta, Delhi**
23	Winner of the best paper published in FOGSI Journal during the year 2018 in Junior Category. (3 awards)	**1st Prize:** Dr. Reshu Agarwal, Kanpur **2nd Prize:** Dr. Shobha A. Alluvala, Hyderabad **3rd Prize:** Dr. Kavitha Yogini Duraisamy Covai

AOGD Events Held

On 16th September 2020 - Webinar on **Medical Disorders in Pregnancy** by SFM Delhi Chapter.

On 16th September 2020 - Webinar on **Recurrent Pregnancy Loss/ Recurrent Implantation Failure** by Dr. Kavita Agarwal.

On 19th September 2020 - International Webinar on **Public Awareness Programme COVID-19** organized by Rural Health Committee AOGD 2020-2022 & Women Doctors Wing, IMA West Ghaziabad.

On 20th September 2020 - International Webinar on **Public Awareness Programme COVID-19** organized by Rural Health Committee AOGD 2020-2022 & Women Doctors Wing, IMA West Ghaziabad.

On 23th September 2020 - Virtual meeting on **Orientation on FOGSI Manyata Initiative: Improving Quality of Care in Maternity Units of Delhi.**

On 25th September 2020 - **AOGD Virtual Monthly Clinical Meeting** organised by DDU Hospital, New Delhi, 04:00-05:00 pm.

On 26th September 2020 - International webinar on **High Risk Pregnancy Management-Challenges in Pregnancies with Heart Disease** by Dept. of Ob-Gyn, ABVIMS & Dr. RML Hospital, New Delhi under the Aegis of NARCHI and AOGD.

On 26th September 2020 - International webinar on **High Risk Pregnancy Management-Challenges in Pregnancies with Heart Disease** by Dept. of Ob-Gyn, ABVIMS & Dr. RML Hospital, New Delhi under the Aegis of NARCHI and AOGD.

On 28th September 2020 - Webinar on **Scope and Challenge of Hysteroscopy in very Elderly** and **Laparoscopic Myomectomy** by IAGE Delhi Chapter & AOGD Endoscopy Committee.

On 4th October 2020 - Webinar on **Infertility** by AOGD Infertility Committee.

On 5th October 2020 - Webinar on **Cervical Stenosis: Overpassing Safely** and panel discussion on **Intrauterine Adhesions: Diagnostic & Therapeutic Challenges, Experience and Evidences** in Endoscopy Fiesta by Endoscopy Committee AOGD & IAGE Delhi Chapter.

On 5th October 2020 - Webinar on **Transcending Hormones** by Reproductive Endocrinology Committee, AOGD DGF South-West Delhi and Sonoschool India.

On 8th October 2020 - Public awareness webinar on **Menstrual Disorders and PCOD** by Rural Health Committee of AOGD in association with LIONESS CLUB.

On 10th October 2020 - Webinar on **Onco-fertility** by IMA Dwarka Gynae Forum and Infertility Committee of AOGD.

On 10th October 2020 - Webinar on **Oncology Classes Part I** by DF South-West and Multidisciplinary Patient Sub Committee of AOGD.

Forthcoming Events

On 23rd October 2020 - **E-Quiz -Slogan Competition**

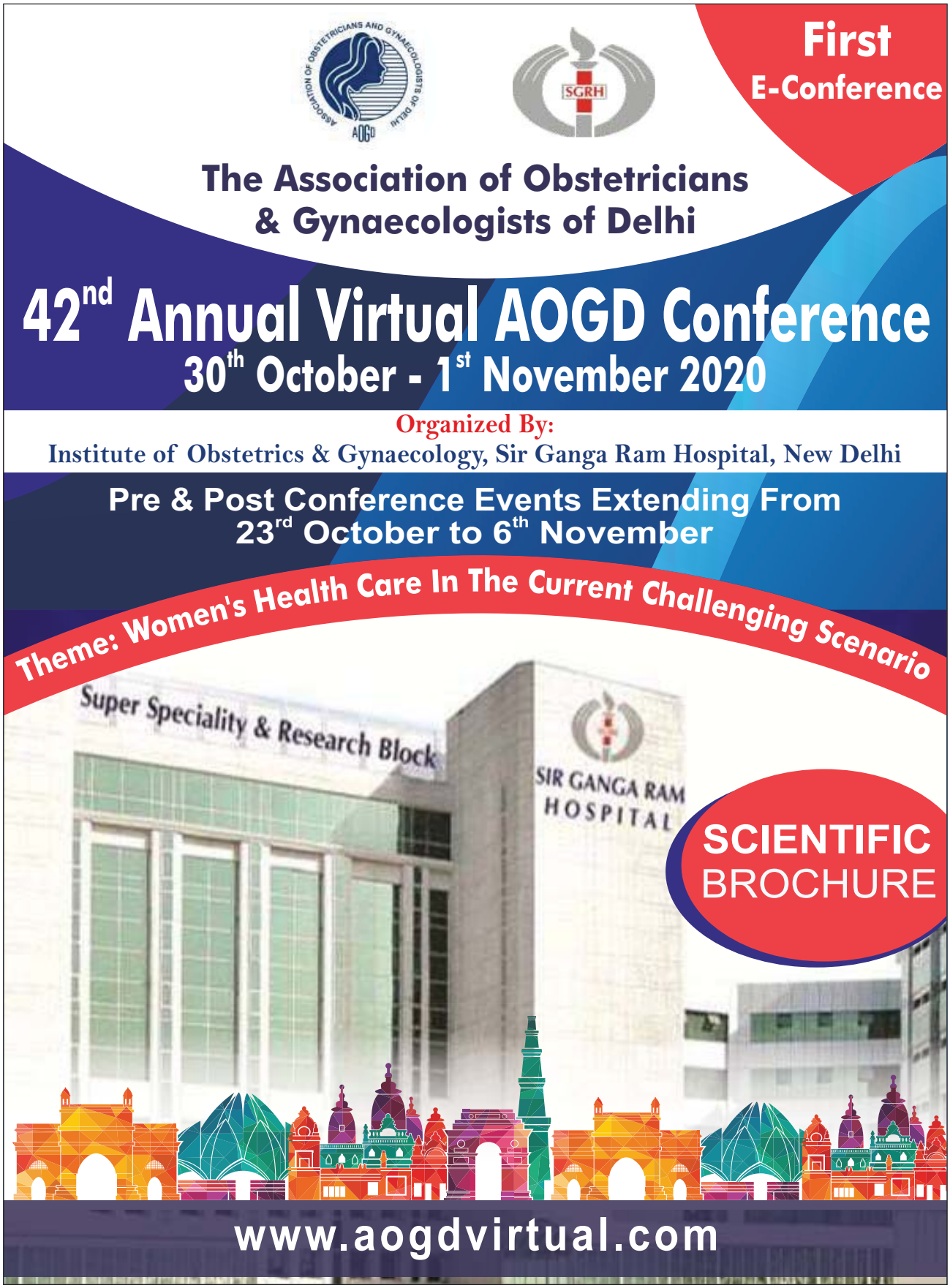
On 24th October 2020 - **E-Poster & Free Papers**

On 26th -29th October 2020 - **Pre Conference Workshops**

30th-31st October & 1st November 2020 - **42nd Annual Virtual AOGD Conference**

2nd -6th November 2020 - **Post Conference Workshops**

Conference Details



Invitation

Dear AOGD Members,

It gives us immense pleasure to invite and connect you to the **42nd Annual Conference of AOGD on 30th, 31st October and 1st November 2020.** The Theme of the Conference has been aptly chosen as **"Women's Health Care In The Current Challenging Scenario".** We intend to offer a basket of approaches and recent advances to safeguard Women's Health.

As this Corona Pandemic has challenged us with unique and unknown situations, we have devised strategies to overcome them and strive for a safer and better future. It's important that we adhere to the safety norms, as well as continue with the academic activities. Keeping this in mind, we have organized the 42nd Annual Conference of AOGD as the **1st Annual E-Conference** of our members.

Through this Conference, we wish to provide an academic feast to all our viewers. It would include Pre and Post Conference Workshops on various sub-specialties, Orations, Keynote Addresses, Panel Discussions, E-Posters and Paper Presentations, E-Quiz, Slogan Competition, and Video sessions spread out from **23rd October till 6th November 2020.**

Please block your dates and participate in this scientific extravaganza. Looking forward to an interactive conference.

Warm Regards
Organizing Team, AOGD

Dr. Mala Srivastava
Organizing Chairperson

Dr. Kanika Jain
Organizing Co-Chairperson

Dr. Mamta Dagar
Organizing Secretary

Conference Highlights

- 5 Pre-Conference Workshops
- 6 Post-Conference Workshops
- 3 Orations
- 6 Keynote Addresses
- 4 Panel Discussions
- 6 Video Sessions
- E-Quiz
- E-Competition Papers
- E -Posters & Free Papers
- E-Slogan Competition

Scientific Committee Advisors

Dr. Kanwal Gujral

Dr. Harsha Khullar

Dr. Abha Majumdar

Scientific Committee Chairpersons

Dr. Mala Srivastava

Dr. Geeta Mediratta

Dr. Chandra Mansukhani

Scientific Committee Co-Chairpersons

Dr. Debasis Dutta

Dr. Punita Bhardwaj

Joint Secretaries

Dr. Neeti Tiwari

Dr. Ruma Satwik

Treasurer

Dr. Shweta Mittal Gupta

Co Treasurer

Dr. Tarun Kumar Das

Workshop Committee

Dr. Debasis Dutta
Dr. Kanika Jain
Dr. Shweta M Gupta

Quiz Committee

Dr. Mamta Dagar
Dr. Richa Sharma
Dr. Sharmistha Garg
Dr. Ila Sharma

Competition Papers

Dr. Kanwal Gujral
Dr. Harsha Khullar
Dr. Sunita Kumar

Free Papers / Posters

Dr. Abha Majumdar
Dr. Sumita Mehta
Dr. Ruma Satwik
Dr. Sakshi Nayar

E- Slogans

Dr. Kanika Jain
Dr. Neeti Tiwari
Dr. Ankita Srivastava

E - Souvenir

Dr. Geeta Mediratta
Dr. Chandra Mansukhani
Dr. Sharmistha Garg

Registration

Dr. Shweta Mittal Gupta
Dr. Sunita Kumar
Dr. Tarun Das

Agenda at a Glance

23th October, 2020
E-Quiz & E-Slogan Competition

24th October, 2020
E-Poster & Free Papers

26th October - 29 th October, 2020
Pre Conference Workshops

30th October - 01st November, 2020
Scientific Programme

02nd November - 06 th November, 2020
Post Conference Workshops

"Past President's Oration"

Friday: 30th October 2020

Topic: Speciality of Obstetrics & Gynecology Then and Now

ORATOR

Dr. Sunesh Kumar

Past President AOGD
Professor and HOD
Department of Obstetrics and Gynaecology
All India Institute of Medical Sciences,
Delhi

"FOGSI President's Oration"

Saturday: 31st October 2020

Topic: Women's Health Crisis in Covid 19 Pandemic

ORATOR

Dr. Alpesh Gandhi

President FOGSI
Critical Care in Obstetric Specialist
ICOG Governing Council Member
Chairperson, Practical Obstetric Committee FOGSI (2008-2011)
Past President Ahmedabad Ob-Gyn Society

"Brigadier S. D. Khanna Oration"

Sunday: 01st November 2020

Topic: Redefining Intrapartum Care Based on Recent Evidence

ORATOR

Dr. S. Arulkumaran

Professor Emeritus of Obstetrics & Gynecology
St George's, University of London
Visiting Professor- Institute of Global Health Innovation
Foundation Professor of O&G,
University of Nicosia


5 Pre-Conference Workshops
(26th-29th October 2020)
Updating Surgical Skills In Gynae Oncology
26th October 2020
02:00PM - 06:15PM
Convenor:
Dr. Amita Suneja
GTB
Enhancing Surgical Skills In Gynae Endoscopy
27th October 2020
12:00PM - 06:30PM
Convenor:
Dr. Kanika Jain
SGRH
Medico-Legal Concerns in Obstetrics and Gynaecology
28th October 2020
9:30AM - 01:30PM
Convenor:
Dr. Asmita Rathore
MAMC
Revisiting IUI In The Era of IVF
28th October 2020
03:00PM - 07:00PM
Convenor:
Dr. Shweta Mittal Gupta
SGRH
CTG - Basics To Advanced
29th October 2020
02:00PM - 05:00PM
Convenor:
Dr. Reva Tripathi
HIMSR

6 Post-Conference Workshops (2nd-6th November 2020)

Fetal Medicine - Care of Fetus Across All Trimesters
2nd November 2020
09:30AM - 01:30PM
Convenor:
Dr. Sunesh Kumar
AIIMS

Management of PPH
2nd November 2020
03:00PM - 06:15PM
Convenor:
Dr. Shashi Lata Kabra
DDU

Tackling Unmet Need For FP Services In Times of COVID-19
3rd November 2020
09:00AM - 01:00PM
Convenor:
Dr. Mrinalini Mani
GGSGH

Critical Care Obstetrics
4th November 2020
09:15AM - 01:30PM
Convenor:
Dr. Jyotsna Suri
SJH

Care Bundle For Multiple Pregnancies
5th November 2020
10:00AM - 02:00PM
Convenor:
Dr. Manju Puri
LHMC

Urogynaecology
6th November 2020
03:00PM - 06:00PM
Convenor:
Dr. Amita Jain
Medanta Medicity

Scientific Program

Day 1, Friday, 30th October, 2020 | 01:00 PM-07:00 PM

Time	Topic	Speaker
01:00PM-02:00PM	**INAUGURATION**	
	Master of Ceremony: Dr Neeti Tiwari	
	CHIEF GUEST- Dr Alpesh Gandhi	
	GUEST OF HONOUR- Dr D S Rana & Dr S P Byotra	
02:00PM-03:00PM	**Session 1: ORATION** **Master of Ceremony: Dr Kanika Jain** **Chairpersons : Dr S N Mukherjee, Dr Kamal Buckshee, Dr Abha Singh, Dr Mala Srivastava**	
	SPECIALITY OF OBSTETRICS & GYNECOLOGY THEN AND NOW	**Dr Sunesh Kumar**
03:00PM-04:00PM	**Session 2: VIDEO SESSIONS** **Master of Ceremony: Dr Punita Bhardwaj** **Chairpersons: Dr L Mettler, Dr P Mangeshikar**	
03:00PM-03:10PM	Laparoscopic Sling Surgery - Variety and Perspective	**Dr P Palaskar**
03:10PM-03:20PM	Novel Fluid Management System	**Dr A Kumar**
03:20PM-03:30PM	Laparoscopic Assisted Radical Trachelectomy	**Dr G Mehra**
03:30PM-03:40PM	VVF - Robotic Approach	**Dr M Sundaraman**
03:40PM-03:50PM	Laparoscopic Intricacies of Ureteric Dissection in DIE	**Dr S Pandey**
03:50PM-04:00PM	Enbloc Paraaortic Dissection - Laparoscopic/Laparotomy	**Dr J Mehta**
04:00PM-06:00PM	**Session 3: COMPETITION PAPERS** Master of Ceremony: Dr. Sunita Kumar **JUDGES: Dr N B Vaid, Dr S S Trivedi, Dr Suneeta Mittal, Dr Reva Tripathi**	
06:00PM - 06:30PM	**Session 4 | EXPERTS SPEAK** **Chairpersons: Dr Mamta Dagar, Dr Savita Singhal, Dr Vatsla Dadhwal, Dr Richa Sharma**	
06:00PM-06:10PM	Protocol for Medico-legal Examination of Sexual Assault	**Dr Sushma Sinha**
06:10PM-06:20PM	Vaccination in women	**Dr Seema Prakash**
06:20PM-06:30PM	Understanding PCOS	**Dr Anita Rajorhia**
06:30PM - 07:00PM	**Session 5 | EXPERTS SPEAK** **Chairpersons: Dr Sunita Malik, Dr Col. Reema Bhat, Dr Neeti Tiwari, Dr Ruma Satwik**	
06:30PM-06:40PM	Luteal Phase Support - Are Long Prescriptions Required ?	**Dr Kavita Agarwal**
06:40PM-06:50PM	Conserative Management of Prolapse	**Dr Achla Batra**
06:50PM-07:00PM	Mullerian Anomalies - Fertility Outcome	**Dr Kuldeep Jain**

Day 2, Saturday, 31st October, 2020 | 01:00 PM-07:00 PM

Time	Topic	Speaker
01:00PM-01:30PM	**Session 1: PRESIDENTIAL SESSION Masters of Ceremony: Dr Mala Srivastava & Dr Mamta Dagar Chairpersons: Dr Archana Verma, Dr Rekha Mehra, Dr Neera Agarwal**	
01:00PM-01:10PM	When to shift from IUI to IVF	**Dr Sudha Prasad**
01:10PM-01:20PM	How to make an effective Power Point Presentation	**Dr Sharda Jain**
01:20PM-01:30PM	Women's intimate health - Let's Talk	**Dr Ragini Aggarwal**
01:30PM-02:00PM	**Break**	
02:00PM-03:00PM	**Session 2: FOGSI ORATION Chairpersons: Dr Harsha Khullar, Dr Shalini Rajaram, Dr Ashok Kumar, Dr Renu Arora**	
	WOMEN'S HEALTH CRISIS IN COVID-19 PANDEMIC	**Dr Alpesh Gandhi**
03:00PM-04:00PM	**Session 3: KEYNOTE ADDRESSES Chairpersons: Dr Chandra Mansukhani, Dr Achla Batra, Dr Sanjeevani Khanna, Dr Indu Chawla**	
03:00PM-03:20PM	Laparoscopic Management of Cesarean Complications	**Dr Alka Kriplani**
03:20PM-03:40PM	PPH- New Thoughts on Management	**Dr V P Paily**
03:40PM-04:00PM	Controversies in Management of Tubal Ectopic Pregnancy	**Dr Bhaskar Pal**
04:00PM-06:00PM	**Session 4: PANEL DISCUSSIONS**	
04:00PM-05:00PM	**TOPIC: MENOPAUSAL HORMONE THERAPY- MADE TO ORDER (Case Based Discussion) PANELISTS: Dr Meeta, Dr Parag Biniwale, Dr Neelam Aggarwal, Dr Hephzibha, Dr Srikanthan, Dr Anupama Mane**	**MODERATOR: Dr Jyothi Unni**
05:00PM-06:00PM	**TOPIC: COVID-19 IN PREGNANCY PANELISTS: Dr Sushma Malik, Dr Pradnya Changede, Dr Neelam Redkar, Dr Chand Wattal, Dr Pratima Mittal, Dr Narayan Jana, Dr Chinmayee Ratha**	**MODERATOR: Dr Reena Wani CO-MODERATOR: Dr Naina Dalvi**
06:00PM-06:30PM	**Session 5 | EXPERTS SPEAK Chairpersons: Dr Arbinder Dang, Dr Asmita Rathore, Dr A G Radhika, Dr Renuka Malik**	
06:00PM-06:10PM	Abnormal Placentation and Adverse Obstertic Outcome: Screening, Diagnosis and Treatment	**Dr Manisha Kumar**
06:10PM-06:20PM	Preterm Labour	**Dr Manju Khemani**
06:20PM-06:30PM	Jaundice in Pregnancy - Differential Diagnosis and Management Principles	**Dr Jyotsna Suri**
06:30PM - 07:00PM	**Session 6 | EXPERTS SPEAK Chairpersons: Dr Aparna Sharma, Dr Abha Sharma, Dr Kanika Jain, Dr Anjali Dabral**	
06:30PM-06:40PM	Restructuring Obstetric Practices in the COVID Pandemic : A Journey of Quality Improvement	**Dr Manju Puri**
06:40PM-06:50PM	Vulvar Intraepithelial Neoplasia - Recent Concepts	**Dr Amita Suneja**
06:50PM-07:00PM	Genitourinary Syndrome of Menopause	**Dr Shashi Lata Kabra**

Day 3, Sunday,1st November, 2020 | 01:00 PM-06:30 PM

Time	Topic	Speaker
01:00PM-01:30PM	**Session 1: PRESIDENTIAL SESSION** **Masters of Ceremony: Dr Geeta Mediratta & Dr Chandra Mansukhani** **Chairpersons: Dr Anita Sabharwal Kapoor, Dr Kiran Guleria, Dr Ratna Biswas**	
01:00PM-01:10PM	Adolescent PCOS: Resolving Dilemmas	**Dr Kiran Agarwal**
01:10PM-01:20PM	One Stop Treatment for CIN	**Dr Sarita Shamsunder**
01:20PM-01:30PM	Safety Issues in Geriatric Population	**Dr Harsha Khullar**
01:30PM-02:00PM	**Break**	
02:00PM-03:00PM	**Session 2: BRIGADIER S D KHANNA ORATION** **Chairpersons: Dr S B Khanna, Dr Ranjana Sharma, Dr Kanwal Gujral, Dr Sadhna Gupta**	
	REDEFINING INTRAPARTUM CARE BASED ON RECENT EVIDENCE	**Dr S Arul Kumaran**
03:00PM-04:00PM	**Session 3: KEYNOTE ADDRESSES** **Chairpersons: Dr J B Sharma, Dr Geeta Mediratta, Dr Pratima Mittal**	
03:00PM-03:20PM	Basics of Urogynecology	**Dr Ajay Rane**
03:20PM-03:40PM	Impact of a Stillbirth - A Preventable Tragedy	**Dr Nuzhat Aziz**
03:40PM-04:00PM	Changing the Care of Multiple Pregnancies	**Dr Soma Mukherjee**
04:00PM-06:00PM	**Session 4: PANEL DISCUSSIONS**	
04:00PM-05:00PM	**TOPIC: ART- MEDICOLEGAL ASPECTS** **PANELISTS: Dr Manish Banker, Dr Kamini Rao,** **Dr Leena Patankar, Dr Deepak Goenka, Dr Sohani Verma,** **Dr Neena Malhotra, Dr Sujata Agarwal**	**MODERATOR:** **Dr Geetendra Sharma**
05:00PM-06:00PM	**TOPIC: MANAGEMENT OF PREINVASIVE LESIONS OF THE CERVIX (CASE BASED DISCUSSION)** **PANELISTS:Dr Vijay Zutshi, Dr Gauri Gandhi, Dr Sweta Balani,** **Dr Seema Singhal, Dr Partha Basu, Dr Theresa Freeman Wang**	**MODERATOR:** **Dr Neerja Bhatla**
06:00PM-06:30PM	**Session 5: VALEDICTORY FUNCTION**	

REGISTER ONLINE @ www.aogdvirtual.com

Contact Us

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First E-Conference

The Association of Obstetricians & Gynaecologists of Delhi

42nd Annual Virtual AOGD Conference

30th October - 1st November 2020

ORGANISED BY
Institute of Obstetrics & Gynaecology, Sir Ganga Ram Hospital, New Delhi

Pre & Post Conference Events Extending From 23rd October to 6th November

Women's Health Care In The Current Challenging Scenario

Registration Form

DELEGATE DETAILS

AOGD Membership _____ MCI/State Medical Council Reg. No _____

Title: Prof. ○ Dr. ○ Mr. ○ Ms. ○ Mrs. ○ **Gender:** Male ○ Female ○

First Name **Last Name**

Address:

Country: **City:** **State:** **Pin:**

Telephone: **Mobile No. with Country Code:**

Email:

Registration Fees (Conference & Workshop)

Category	Early Bird (Till 30th September, 2020)	1st October, 2020 Onwards
AOGD Member	INR 3000 ☐	INR 3500 ☐
Non AOGD Member	INR 3500 ☐	INR 4000 ☐
AOGD Annual Membership + Conference (Delegates) Suitable for Presenting E Poster/Free Paper	INR 5360 ☐	INR 6360 ☐
PG Students	INR 2000 ☐	INR 2000 ☐
AOGD Annual Membership + Conference (PG Only) Suitable for Presenting E Poster/Free Paper/Competition Paper	INR 4360 ☐	
AOGDIANS (Above >70) Please Submit A Copy Of Your Aadhar Card As Age Proof By Email	Free	

Note: *Post Graduates To Attach A Certificate From HOD
Also Should Be An Annual Member Of The AOGD In Order To Attend

Inclusive of 18% GST

Note: Conference Registration Is Mandatory to Attend Both Pre & Post Conference Workshops

Pre-Conference Workshops (26th - 29th October, 2020)

Workshop Name		Time	Date
Updating Surgical Skills In Gynae Oncology	☐	02:00 PM-06:00 PM	26th October 2020
Enhancing Surgical Skills In Gynae Endoscopy	☐	12:00 PM-06:15 PM	27th October 2020
Medico Legal Concerns in Obstetrics & Gynaecology	☐	09:30 AM-01:30 PM	28th October 2020
Revisiting IUI In The Era of IVF	☐	03:00 PM-07:00 PM	28th October 2020
CTG - Basic To Advanced	☐	02:00 PM-05:00 PM	29th October 2020

Post-Conference Workshops (2nd - 6th November, 2020)

Workshop Name		Time	Date
Fetal Medicine - Care Of Fetus Across All Trimesters	☐	09:30 AM-01:30 PM	2nd November 2020
Management of PPH	☐	03:00 PM-06:00 PM	2nd November 2020
Tackling Unmet Need For FP Services In Times of COVID-19	☐	10:00 AM-02:00 PM	3rd November 2020
Critical Care Obstetrics	☐	10:00 AM-02:00 PM	4th November 2020
Care Bundle For Multiple Pregnancies	☐	10:00 AM-02:00 PM	5th November 2020
Urogynaecology	☐	03:00 PM-06:00 PM	6th November 2020

Mode of Payment

1. Bank Draft/Cheque - To be made in favor of "**Association of Obstetricians and Gynaecologists of Delhi**" payable at New Delhi

DD/Cheque No. /Cash ☐ Total Amount (INR) ☐

For Online Registration, Visit - www.aogdvirtual.com

Bank Transfer Details

Account Name: Association of Obstetricians and Gynaecologists of Delhi

Account No.: 3674596638

Bank: Central Bank of India

Branch: Lady Hardinge Medical College Branch

Connaught Place, New Delhi | Delhi 110001

IFSC Code: CBIN0283462

MICR Code: 110016067

Cancellation & Refund Policy

- Cancellation till 31st August, 2020 - 75% Refund
- Cancellation till 15th September, 2020 - 25% Refund
- Cancellation after 15th September, 2020 Onwards - No Refund
- All Refunds Will be Made after the Conference

Note: Whats app your Registration Form with payment details to Mr. Vikas Sharma on +91-9999216837. Payment confirmation will take 3-4 working days

Virtual Congress's Manager

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WORKSHOP
BROCHURE

Organized By:
Institute of Obstetrics & Gynaecology, Sir Ganga Ram Hospital, New Delhi

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Pre-Conference Workshop

Monday 26th October 2020 | 02:00 PM - 06:15 PM

Updating Surgical Skills in Gynaecologic Oncology

Convenor: Dr. Amita Suneja & Dr. Shalini Rajaram
Co-Convenors: Dr. Rashmi Malik & Dr. Bindiya Gupta

Time	Topic	Speaker
02:00PM-02:15PM	**INAUGURATION CEREMONY** **GUEST OF HONOUR -** Dr. Neera Aggarwal (HOD, MAX SSH, Patparganj)	
President's Address - Dr. Mala Srivastava (President AOGD)		
Overview of Workshop - Dr. Amita Suneja (UCMS & GTBH)		
Surgical Training : Quality Indicators - Dr. Shalini Rajaram (AIIMS Rishikesh)		
02:15PM - 03:15PM	**Session 1: FINE TUNING SURGICAL MANAGEMENT OF CERVICAL CANCER** **Chairpersons : Dr. Swaraj Batra (Ex HOD, MAMC & LNH) & Dr. Gauri Gandhi (MAMC & LNH)**	
02:15PM - 02:30PM	Surgical Anatomy of Pelvis	**Dr. Neerja Bhatla (AIIMS, Delhi)**
02:30PM - 02:45PM	Open Radical Hysterectomy	**Dr. Amita Maheshwari (TMH, Mumbai)**
02:45PM - 03:00PM	Radical Trachelectomy	**Dr. Sunesh Kumar (AIIMS, Delhi)**
03:00PM - 03:15PM	**Discussion**	
03:15PM - 04:15PM	**Session 2: MASTERING NODAL DISSECTION** **Chairpersons : Dr. Kiran Guleria (UCMS & GTBH) & Dr. Reena Yadav (LHMC & SKH)**	
03:15PM - 03:30PM	Robotic Pelvic Sentinel Lymph Node Dissection For Gynecologic Cancers	**Dr. Rama Joshi (FMRI, Gurugram)**
03:30PM - 03:45PM	Perfecting Pelvic Lymphadenectomy	**Dr. Bindiya Gupta (UCMS & GTBH)**
03:45PM - 04:00PM	Open Paraaortic Lymphadenectomy	**Dr. SP Somashekhar (MCCC, Bangalore)**
04:00PM - 04:15PM	**Discussion**	
04:15PM - 05:15PM	**Session 3: OPTIMAL CYTOREDUCTION IN ADVANCED OVARIAN CANCER** **Chairpersons : Dr. Neelam B Vaid (Consultant Fortis, Shalimar Bagh) &** **Dr. Sabhyata Gupta (Medanta, Gurugram)**	
04:15PM - 04:30PM	Surgical Approach To A Complex Pelvis	**Dr. Kavita Singh (PBGCC, UK)**
04:30PM - 04:45PM	Total Omentectomy& "Hand-Sewn" Side To Side Small Bowel Anastomosis	**Dr. Shylasree T Surappa (TMH, Mumbai)**
04:45PM - 05:00PM	Peritonectomy: Pelvic and Abdominal	**Dr. Pankaj Garg (AIIMS, Rishikesh)**
05:00PM - 05:15PM	**Discussion**	
05:15PM - 06:15PM	**Session 4: TAILORING SURGERY IN VULVAR CANCER** **Chairpersons : Dr. Saritha Shamsunder (VMMC & SJH) & Dr. Vijay Zutshi (VMMC & SJH)**	
05:15PM - 05:30PM	Groin Sentinel Lymph Node Detection	**Dr. Jason Yap (PBGCC, UK)**
05:30PM - 05:45PM	Reconstructive Surgery For Vulvar Cancer	**Dr. S K Giri (Ex HOD, AHRCC, Cuttack)**
05:45PM - 06:00PM	Robotic VEIL	**Dr. Rupinder Sekhon (RGCIRC, Delhi)**
06:00PM - 06:10PM	**Discussion**	
06:10PM - 06:15PM	**Vote of Thanks : Dr Rashmi Malik**	

Pre-Conference Workshop

Tuesday 27th October 2020 | 12:00 PM - 06:30 PM

Enhancing Surgical Skills In Gynae Endoscopy

Convenor: Dr. Kanika Jain & Dr. Debasis Dutta
Co-Convenors: Dr. Richa Sharma, Dr. Madhu Goel

Time	Topic	Speaker
12:00PM - 12:05PM	Welcome Address	**Dr. Mala Srivastava (SGRH)**
12:05PM - 12:15PM	Introduction to Workshop	**Dr. Mamta Dagar (SGRH)**
Master of Ceremony - Dr Kanika Jain, Dr Mamta Dagar		
Session I - Chairpersons : Dr. Geeta Mediratta, Dr. A Jyotsna, Dr Chandan Dubey(UAE) & Dr. Swati Agarwal		
12:15PM - 12:30PM	Pelvic Anatomy- Avascular Pelvic Spaces	**Dr. Sandesh Kade (Solapur)**
12:30PM - 12:45PM	Energy Sources in Gynae Endoscopy- Then and Now	**Dr. Malvika Sabharwal (JMH)**
12:45PM - 01:00PM	Tips and Tricks - How to Make Laparoscopy Safer	**Dr. Arvind Kumar (New Delhi)**
Session II - Chairpersons: Dr. Kanwal Gujral, Dr. Sharda Jain, Dr. S. S. Trivedi, Dr. Mamta Dagar		
01:00PM - 01:20PM	TLH- A Day Care Surgery	**Dr. Rajesh Modi (Akola)**
01:20PM - 01:40PM	Difficult TLH- How to Handle	**Dr. Vivek Marwah (Max, Saket)**
01:40PM - 02:00PM	Laparoscopic Wertheim's/Radical Hysterectomy - A Step by Step Guide	**Dr. Hafiz Rehman (Kochi)**
Session III - Chairpersons: Dr. Tripti Saran, Dr. Ritu Rana (UK), Dr Jyoti Bhaskar & Dr Nandini Bhattacharya		
02:00PM - 02:20PM	Laparoscopic Myomectomy	**Dr. Tom Kieran Holland (Guys & Thomas Hospital, London)**
HYSTEROSCOPY – BASICS Master of Ceremony : Dr. Richa Sharma, Dr. Madhu Goel **Session IV - Chairpersons: Dr. Chandra Mansukhani, Dr. Himani Agarwal, Dr. Anita S. Anand, Dr. Sharmistha**		
02:20PM - 02:30PM	Principles of Hysteroscopy	**Dr. Manjit Sidhu (Bhatinda)**
02:30PM - 02:40PM	Hysteroscopy Vs 3D USG For Identification of Uterine Pathologies	**Dr. Narendra Malhotra (Agra)**
HYSTEROSCOPY - SURGICAL TECHNIQUES **Session V - Chairpersons: Dr. Renu Mishra, Dr. Ruma Satwik, Dr. Surveen Ghumman, Dr Seema Gupta**		
02:40PM - 02:50PM	Septal Resection; When & How Far	**Dr. Richa Sharma (GTB)**
02:50PM - 03:00PM	Asherman's Syndrome: Current Treatment Plan & Follow Up	**Dr. Pragnesh Shah (Ahmedabad)**
03:00PM - 03:20PM	Hysteroscopic Myomectomy & Morcellation: Dealing Safely	**Dr. Sergio Haimovich (Spain)**
COMPLICATIONS OF HYSTEROSCOPY - TIPS & TRICKS **Session VI - Chairpersons: Dr. Nidhi Khera, Dr. Sabuhi Qureshi,Dr Neeti Tiwari, Dr. Anupama Bahadur**		
03:20PM - 03:30PM	How to Avoid	**Dr. K .K Roy (New Delhi)**
03:30PM - 03:45PM	How to Tackle	**Dr. Sandip Datta Roy (Thrissur)**
Master of Ceremony - Dr Kanika Jain, Dr Mamta Dagar **Session VII - Chairpersons : Dr. Harsha Khullar, Dr. Sanjeevani Khanna, Dr. Laxmi Mantri, Dr Tarun Das**		
03:45PM - 04:05PM	Laparoscopic V- Note Surgery- New Minimally Invasive Scarless Surgery	**Dr. Suyash Naval (Maharashtra)**
04:05PM - 04:25PM	Various Techniques of Laparoscopic Vaginoplasty	**Dr. A Chatterjee (Calcutta)**
04:25PM - 04:45PM	Laparoscopic Sentinel Lymph Node Mapping	**Dr. Dinesh Kansal (BLK Hospital)**
04:45PM - 05:15PM	Complications in Laparoscopic Surgeries- Learn from the Expert	**Dr. Shailesh Puntambekar (Pune)**
Session VIII - Chairpersons: Dr. Subhash Mallaya, Dr. Sabhyata Gupta, Dr Shweta M Gupta		
05:15PM - 05:55PM	**PANEL: SURGICAL MANAGEMENT OF ENDOMETRIOSIS** **PANELISTS: Dr. Nutan Jain (Muzaffar Nagar) , Dr. Sanjay Patil (Ahmedabad), Dr. Punita Bhardwaj (SGRH), Dr. D Dutta (SGRHI)**	**MODERATORS:** **Dr. Manju Khemani (Max, Saket), Dr. Madhu Goel (La-Femme)**
Session IX - Chairpersons: Dr Kanika Jain, Dr Jyoti Bali, Dr Lalita Budhwar,Dr. Panchampreet Kaur		
05:55PM - 06:25PM	New Developments and Strategies In The Management of Severe Endometriosis-"The Intelligent Light"	**Dr. Ceana Nezhat (Atlanta, USA)**
06:25PM - 06:30PM	**Vote of Thanks : Dr. Kanika Jain (Vice President AOGD)**	

Pre-Conference Workshop

Wednesday 28th October 2020 | 09:30 AM - 01:30 PM

Medico-Legal Aspects in Obstetrics and Gynaecology

Convenor: Dr. Asmita M Rathore, Dr. Deepti Goswami, Dr. Niharika Dhiman

Time	Topic	Speaker
9:30AM - 09:35AM	Welcome Note	**Dr. Asmita M Rathore**
Chairpersons : Dr. Gauri Gandhi, Dr. Pikee Saxena & Dr. Nalini B Pandey		
09:35AM - 10:00AM	Consent and Counseling - Role Play	**Dr. Devender Kumar & Dr. Niharika Dhiman**
Chairpersons : Dr. Vijay Zutshi, Dr. Poonam Sachdeva, & Dr. Rachna Sharma		
10:00AM - 10:15AM	Family Planning Services - Provision and Penalties	**Dr. Sumita Mehta**
Chairpersons : Dr. Kanwal Gujral, Dr. Krishna Agarwal & Dr. Shakun Tyagi		
10:15AM - 10:30AM	Medicolegal Concerns in Fetal medicine	**Dr. Sangeeta Gupta**
10:30AM - 10:45AM	**Discussion**	
10:45AM - 11:30 AM	**PANEL DISCUSSION: MEDICOLEGAL ASPECTS IN OBSTETRICS AND GYNAECOLOGY PANELISTS: Dr. Indu Chawla, Dr. Jyotsna Suri, Dr. Poonam Kashyap, Dr. Rachna Agarwal, Dr. Garima Kachhawa, Dr. Tapas Koley**	**MODERATOR : Dr. Deepti Goswami**
Chairpersons : Dr. Madhavi M Gupta, Dr. Preeti Singh & Dr. Reena Rani		
11:30AM- 11:45AM	Examination of Survivor	**Dr. Chetna A Sethi**
Chairpersons : Dr. Y M Mala, Dr. Latika Sahu & Dr. Nidhi Garg		
11:45AM - 12:00PM	POCSO Act - Case Based Scenarios	**Dr. Bidhisha Singha**
Chairpersons : Dr. Reena Yadav, Dr. Sangeeta Bhasin & Dr. Shalini Shakarwal		
12:00PM - 12:15PM	Recording of MLC	**Dr. Monisha Pradhan**
12:15PM - 12:30 PM	**Discussion**	
12:30PM - 1:15 PM	**PANEL DISCUSSION: ART AND SURROGACY PANELISTS: Dr. Surveen Ghumman, Dr. K D Nayar, Dr. Shikha Sharma, Dr. Leena Wadhwa, Dr. Pushpa Mishra, Mrs Rucha Mayee**	**MODERATORS : Dr. Anjali Tempe & Dr. Renu Tanwar**
1:15PM - 1:30PM	**Vote of Thanks**	

Pre-Conference Workshop

Wednesday 28th October 2020 | 03:00 PM - 07:00 PM

Revisiting IUI in the Era of IVF

Convenor: Dr. Shweta Mittal Gupta
Co-Convener: Dr. Neeti Tiwari, Dr Kavita Agarwal

Time	Topic	Speaker
03:00PM - 03:05PM	Welcome & Introduction	**Dr. Shweta Mittal**
Session 1 **Chairpersons: Dr. Sonia Malik, Dr Mala Srivastava, Dr Kavita Agarwal, Dr. Surveen Ghumman**		
03:05PM - 03:25PM	Revisiting IUI In The Era Of IVF	**Dr. Abha Majumdar**
03:25PM - 03:45PM	The Art and Science of Ovarian Stimulation for IUI	**Dr. Gulam Bahadur (UK)**
03:45PM - 04:05PM	Fine-Tuning of IUI to Optimize its Success	**Dr. Mohan Kamath**
04:05PM - 04:15PM	**Audience Interaction**	
Session 2 **Chairpersons : Dr Kanika Jain, Dr. Jyoti Bali, Dr. Shalini Chawla**		
04:15PM - 04:30PM	Medicolegal Aspects of IUI	**Dr. Tanya Buckshee**
04:30PM - 04:45PM	Luteal Phase Support After IUI Practical Approach	**Dr. Rashmi Sharma**
04:45PM - 04:50PM	**Audience Interaction**	
04:50PM - 06:00PM	**Session 3 - PANEL DISCUSSION** : CASE BASED SCENARIOS IN OVARIAN STIMULATION AND IUI **PANELISTS:** Dr. C.M. Nagori, Dr. Manish Banker, Dr. Madhuri Patil, Dr. Priya Bhave, Dr. Leena Wadhwa, Dr. Pikee Saxena	**MODERATORS:** **Dr. Shweta Mittal Gupta &** **Dr. Neeti Tiwari**
Session 4 - VIDEO SESSIONS **Chairpersons: Dr. Asha Baxi, Dr Mamta Dagar, Dr. Rupali Bassi**		
06:00PM - 06:20PM	Steps to Optimize IUI Outcomes in the Andrology Lab – (Demonstration of Semen Preparation)	**Dr. Gaurav Majumdar**
06:20PM - 06:40PM	Demonstration of IUI Procedure and Difficult IUI	**Dr. Ruma Satwik**
06:40PM - 06:50PM	**Audience Interaction**	
06:50PM - 07:00PM	Take Home Message from the Workshop	**Dr. Shweta Mittal**
	Vote of Thanks	**Dr. Neeti Tiwari**

Pre-Conference Workshop

Thursday 29th October 2020 | 02:00 PM - 05:00 PM

CTG: Basics to Advanced

Convenor: Dr. Reva Tripathi
Co- Convenor : Dr Aruna Nigam

Organizing Committee Members : Dr Neha Gupta, Dr Arifa Anwar Elahi, Dr Sumedha Sharma, Dr Arpita De, Dr Nidhi Gupta

Time	Topic	Speaker
2:00PM - 2:10PM	**OVERVIEW OF CTG**	**Dr. Reva Tripathi**
Session 1: BASICS OF CTG		
2:10PM-2:20PM	Physiology of Acid Base Balance	**Dr. Shelly Arora**
2:20PM -2:30PM	Obstetrician and CTG	**Dr. Nadia Khursheed**
2:30 PM -2:40PM	**Discussion**	
Session 2: INTERPRETATION		
2:40PM -2:55PM	DR C BRAVADO Approach to CTG	**Dr. Ayesha Ahmad**
2:55PM -3:10PM	How to Classify CTG	**Dr. Shalini Mehrotra**
3:10PM -3:25PM	CTG in Special Situations	**Dr. Chanchal**
3:25PM -3:40PM	**Discussion**	
Session 3: ADVANCES IN CTG		
3:40-3:55PM	cCTG/STAn	**Dr. Smriti Agarwal**
3:55-4:10PM	Cord Sampling and Correlations	**Dr. Nidhi Bedi**
4:10-4:20PM	**Discussion**	
Session 4		
4:20PM-5:00PM	**CASE BASED PANEL DISCUSSION** **PANELISTS: Dr. Rinku Sen Gupta, Dr. Shakun Tyagi , Dr. Jayasree Sunder, Dr. Neha Gupta & Dr. Bindiya Jhamb**	**MODERATORS :** **Dr Aruna Nigam &** **Dr Arpita De**

Post-Conference Workshop

Monday 02nd November 2020 | 09:30 AM - 01:30 PM

Fetal Medicine- Care of Fetus Across All Trimesters

Convenor: Dr. Sunesh Kumar (AIIMS), Dr Vatsla Dadhwal
Co-Convenors: Dr Aparna Sharma, Dr Anubhuti Rana

Time	Topic	Speaker
PANEL DISCUSSION : THE SCREENING CONUNDRUM		
9:30 AM – 10:30 AM	Sonographic Clues To Aneuploidy And Beyond In The First Trimester	**MODERATORS : Dr. Chinmayee Ratha & Dr Akshtha Sharma**
	Unravelling Biochemical Screening and NIPT	
	PANELISTS: Dr. Madhulika Kabra, Dr. Nandita Dimri, Dr. Krishna Gopal, Dr. Sumitra Bachani, Dr. Vandana Chaddha, & Dr. Ritika Bhandari	
10:30 AM- 10.45 AM	**INAUGURATION**	**Dr. Sunesh Kumar, Dr. Mala Srivastava & Dr. Neerja Bhatla**
10.45 AM -11.25 AM	CONSENSUS TO CONTROVERSIES Chairpersons **:** Dr. KK Roy & Dr. Jyoti Meena	**Dr. Sangeeta Gupta & Dr. Reema Bhatt**
11:25 AM – 12:30 PM	**PANEL CASE BASED DISCUSSION ON FETAL GROWTH** **PANELISTS : Dr. Anita Kaul, Dr. Kiran Guleria, Dr. Kanwal Gujral, Dr. Anubhuti Rana, Dr. Poonam Tara**	**MODERATORS : Dr. Vatsla Dadhwal & Dr. Chanchal Singh**
12:30 PM - 1:30 PM	Quiz : Approach to Common Congenital Anomalies	**Dr. Aparna Sharma, Dr. Jaya Chawla, Dr. Latika Chawla & Dr. Rinchen**

Post-Conference Workshop

Monday 02nd November 2020 | 03:00 PM - 06:15 PM

Management of PPH

Convenor: Dr. Shashi Lata Kabra Maheshwari; Dr. Leena N Sreedhar
Co-Convenor: Dr. Yogita Parashar

Experts : Dr. Sharda Jain (Secretary General DGF),
Dr. Sanjeevani Khanna (Chairperson DGF NORTH) &
Dr. Mitra Saxena (Chairperson Elect Practical Obst. Committee FOGSI)

Time	Topic	Speaker
INAUGURATION BY GUEST OF HONOUR : Dr. Alpesh Gandhi (President, FOGSI)		
Chief Guest : Dr. Ragini Aggarwal (VP, FOGSI) & Dr. Mala Srivastava (President, AOGD)		
03:00PM - 04:00PM	**Session 1**	
03:00PM - 03:30PM	PPH - Nightmare to Expertise	**Dr. Shashilata Kabra**
03:30PM - 03:40PM	Blood Component Therapy in PPH	**Dr. Pinkee Saxena**
03:40PM - 03:50PM	Uterine Balloon Therapy	**Dr. Leena Sreedhar**
03:50PM - 04:00PM	Non Pneumatic Antishock Garment (NASG)	**Dr. Sushma Sinha**
04:00PM - 06:00PM	**Session 2 - PPH Drill**	
04:00PM - 04:10PM	**Station 1** NASG	**Dr. Sushma Sinha**
04:10PM - 04:20PM	**Station 2** Assessment of Blood Loss in PPH	**Dr. Yogita Parashar**
04:20PM - 04:40PM	**Station 3** Medical Management of PPH Demonstration of PPH Kit	**Dr. Soma Mitra &** **Dr. Harvinder**
04:40PM - 05:10PM	**Station 4** Uterine Balloon Tamponade	**Dr. Leena Sreedhar**
05:10PM - 06:00 PM	**Station 5** Uterine Compression Sutures IIAL ligation	**Dr. Shashi L Kabra**
06:00PM - 06:15PM	**INTERACTION WITH FACULTY**	

Post-Conference Workshop

Tuesday 03rd November 2020 | 09:00 AM - 01:00 PM

Tackling Unmet Need For FP Services In Times of COVID-19

Convenor : Dr. Mrinalini Mani
Co-ordinator : Dr. Shobha N Gudi, Chairperson, Family Welfare Committee, FOGSI

Time	Topic	Speaker
GUEST OF HONOUR : Dr. Alpesh Gandhi, Dr Ragini Aggarwal & Dr Mala Srivastava		
Comperes : Dr. Riju Chimote & Dr. Kavita Agarwal		
09:00AM - 10:00AM	**Session 1 : EXPANDING THE BASKET OF CHOICE Chairpersons : Dr. Poonam Shivkumar & Dr. Meena Agnihotri**	
09:00 AM - 09:15 AM	Medical Eligibility Criteria : Optimizing the Implementation	**Dr. Radhika A G**
09:15 AM- 09:30 AM	Ensuring Reach to the Unreached in Public Sector	**Dr. Jyoti Sachdeva**
09:30 AM - 09:45 AM	Updates in Contraception	**Dr. Basab Mukherjee**
09:45 AM - 10:00 AM	Fine Tuning Counseling : The Critical Factor	**Dr. Mrinalini Mani**
10:00AM - 11:00AM	**Session 2 - KEY NOTE ADDRESSES - MOVING FORWARD : LEVERAGING PARTNERSHIPS FOR BETTER IMPLEMENTATION OF FP SERVICES Chairpersons : Dr. Chandrawati, Dr. Ragini Aggarwal, & Dr. Atul Ganatra**	
10:00 AM - 10:15 AM	FOGSI Vision	**Dr. Alpesh Gandhi**
10:15 AM - 10:30 AM	Global Perspective	**Dr. Ben Bellows**
10:30 AM - 10:45 AM	NGO Perspective	**Mr. Vijay Paulraj**
10:45 AM- 11:00 AM	**Interaction**	
11:00AM - 12:00PM	**Session 3 : VITAL AREAS OF PRACTICE Chairpersons : Dr. Usha Sharma & Dr. Puneeta Mahajan**	
11:00 AM - 11:15 AM	Safe Abortion : The Ideal Method	**Dr. Bharti Maheshwari**
11:15 AM - 11:30 AM	MTP Act and its PITFALLS	**Dr. M C Patel**
11:30 AM - 11:45 AM	Adolescent Contraception : Protecting the Future	**Dr. Chandan Kachru**
11:45 AM - 12:00 PM	Benefits Beyond Contraception	**Dr. Charmila Ayyawoo**
	Session 4	
12:00 PM - 01:00PM	**PANEL DISCUSSION : INCREASING MET NEEDS FOR FP - INVOLVING COMMUNITY / HEALTHCARE PROVIDERS / DIGITAL PLATFORM AND FOGSI I CARE CLINICS PANELISTS : Dr. Neelam, Dr. Poornima J, Dr. Alka Kuthe, Dr. Sushma Sinha, Dr. Kalpana Apte, Dr. Saurabh Chawla & Dr. Anita Rajorhia**	**MODERATOR : Dr. Shobha N Gudi**

Post-Conference Workshop

Wednesday 04th November 2020 | 09:15 AM - 01:30 PM

Critical Care Obstetrics

Advisor : Dr Pratima Mittal
Convenor: Dr. Jyotsna Suri
Co- Convenor : Dr Rekha Bharti
Organizing Secretary : Dr Sheeba Marwah

Time	Topic	Speaker
09:15AM - 09:30AM	Introduction to Workshop and Welcome Address	**Dr. Pratima Mittal, Dr. Anjali Dabral**
	Session 1 : INAUGURAL SESSION Chairpersons : Dr. Mala Srivastava, Dr. Rupali Dewan & Dr. Renu Arora	
09:30AM - 10:00AM	Why Women Should Become Critically Ill- Pre-Conceptional Care As Relevant to High Risk Pregnancy	**Dr. Alpesh Gandhi, FOGSI President**
	Session 2: OBSTETRICS CCU ROUND- CASE BY CASE DISCUSSION	
10:00AM - 10:25AM	Oxygen Therapy including NIV	**Dr. Archana Mishra EXPERT: Dr. Pratima Mittal**
10:25AM - 10:50AM	ABG & Bicarbonate Therapy	**Dr. Niharika Dhiman EXPERT: Dr. Jyotsna Suri**
10:50AM - 11:15AM	Fluid Management in Shock & Vasopressors	**Dr. Monika Gupta EXPERT: Dr. Rekha Bharti**
	Session 3 : KEY NOTE ADDRESS Chairpersons : Dr. Vijay Zutshi, Dr. Anjali Dabral, Dr. Bindu Bajaj	
11:15AM - 11:45AM	Acute Respiratory Failure in Pregnancy- What the Obstetrician Should Know	**Dr. J C Suri**
	Session 4: PANEL DISCUSSION	
11:45AM - 12:30PM	**PANEL DISCUSSION : DIABETIC KETOACIDOSIS, PERIPARTUM CARDIOMYOPATHY PANELISTS: Dr. Sunita Malik, Dr. Manju Puri; Dr. Asmita Rathore, Dr. Kiran Guleria, Dr. Upma Saxena, Dr Anita Rajorhia**	**MODERATORS: Dr. Jyotsna & Dr. Sheeba**
	Session 5: VIDEO SESSIONS	
12:30PM - 12:55PM	Management of PPH	**EXPERTS: Dr Divya Pandey (Medical Management) & Dr. Achla Batra (Surgical Management)**
12:55PM - 01:20PM	Resuscitation of Pregnant Woman (Dr. Dipti, Dr. Ankita Jain, & Dr. Megha)	**EXPERTS: Dr. Shipra Aggarwal (Resuscitation) & Dr. Rekha Bharti (Perimortem cesarean Section)**
01:20PM - 01:30PM	**AUDIENCE INTERACTION**	**Dr. Sheeba Marwah**

Post-Conference Workshop

Thursday 05th November 2020 | 10:00 AM - 02:00 PM

Care Bundle for Multiple Pregnancies

Convenor: Dr.Manju Puri, LHMC
Co-Convener: Dr Manisha Kumar

Organizing Secretaries: Dr Shilpi Nain/Dr Deepika Meena
Joint Secretary: Dr Kanika Chopra

Time	Topic	Speaker
10:00 AM - 10:10 AM	Introduction to the Workshop	**Dr. Manju Puri**
Session 1 : ANTEPARTUM CARE Chairpersons : Dr. SS Trivedi, Dr. Geeta Mediratta		
10:10 AM - 10:30 AM	First Trimester Care: More to it than Chorionicity	**Dr. Anita Kaul**
10:30 AM - 10:50 AM	Screening for Aneuploidy in Multiple Gestations: Challenges and Options	**Dr. Chanchal Singh**
10:50 AM - 11:10 AM	Antenatal Care: Singleton Vs Multiple Pregnancy	**Dr. Kiran Aggarwal**
11:10 AM - 11:30 AM	Situations Requiring Foetal Medicine Interventions: Red Flags	**Dr. K Aparna Sharma**
11:30 AM - 11:40 AM	Picture Quiz	**Dr. Ratna Biswas**
Session 2 : INTRAPARTUM CARE Chairpersons : Dr. Abha Singh, Dr. Pikee Saxena		
11:40 AM - 12:00 PM	Delivery Preparedness: When, How & by Whom?	**Dr. Reena**
12:00 PM - 12:20 PM	Delivery in Twins: Honing the Art	**Dr. Shilpi Nain & Dr. Kanika Chopra**
12:00 PM - 12:30 PM	Picture Quiz	**Dr. Deepika Meena**
Session 3 : POSTPARTUM CARE Chairperson : Dr Usha Gupta, Dr Prabha Lal		
12:30 PM - 12:50 PM	Postpartum Challenges	**Dr. Jyoti Bhaskar**
12:50 PM - 01:00 PM	Picture Quiz	**Dr. Deepika Meena**
1:00 PM - 02:00 PM	**PANEL DISCUSSION - MANAGEMENT TIGHT SPOTS: CASE BASED DISCUSSION MANAGEMENT DILEMMAS PANELISTS: Dr. Sangeeta Gupta (MAMC), Dr. Vatsala Dadhwal,Dr. Nandita Dimri, Dr. Reema Bhatt, Dr. Sumitra Bacchani**	**MODERATOR: Dr. Manisha Kumar**
	Vote of Thanks	**Dr. Shilpi Nain**

Post-Conference Workshop

Friday 06th November 2020 | 03:00 PM - 06:00 PM

Urogynaecology

Convenor: Dr. Amita Jain
Co-Convenor: Dr. Tanudeep Kaur

Time	Topic	Speaker
03:00PM - 03:10PM	Welcome Address	**Dr. Amita Jain New Delhi, India**
03:10PM - 03:20PM	Introduction to Faculty	**Dr. Tanudeep New Delhi, India**
03:20PM- 03:40PM	Lessons learnt in COVID times- Defining "new" Normal in Urogynecological Practise	**Dr. Ranee Thakar London, UK**
Chairpersons : Dr. Nirmala Agarwal, Dr. J B Sharma		
03:40PM - 04:00PM	Clinical approach to a case of incontinence to choose right Management	**Dr. Jagdish Gandhi Hull, UK**
Chairpersons : Dr. Ranjana Sharma, Dr. Ragini Agarwal		
04:00PM - 04:20PM	Undesired consequences of Pelvic Floor Surgeries: Prevention & Treatment	**Dr. G. Willy Davila, Cleveland Clinic, USA**
AOGD- GIBS JOINT SESSION Chairpersons : Dr. Aparna Hegde, Dr. Uma Rani Swain		
04:20PM - 04:40PM	Confounded by Pelvic Pain- What Should be My Approach to Deal	**Dr. Mauro Cervigni, Rome, Italy**
Chairpersons : Dr. Rajesh Taneja, Dr. Vidya Bandookwala		
04:40PM - 05:20PM	**PANEL DISCUSSION - PRACTICAL APPLICATION OF URODYNAMICS IN UROGYNECOLOGY AND ITS IMPACT ON MANAGEMENT PANELISTS: Dr. Aparna Hegde, Dr. Mohan Regmi, Dr. Geeta Mediratta, Dr. Sandhya Jain**	**MODERATOR: Dr. Amita Jain, New Delhi, India**
05:20PM - 05:40PM	Basics of "Biofeedback"- When & How!!	**Dr. Bary Berghmans, Maastricht Neatherland**
Chairpersons: Dr. Achla Batra, Dr. Meera Raghwan		
05:40PM - 06:00PM	**Vote of thanks**	**Dr. Achla Batra, New Delhi, India**