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

Dedicated Issue:
Critical Care Obstetrics (Part-1)

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From the President's Pen

I bring Greetings from AOGD!

It gives us an immense pleasure to announce the 42nd Annual Conference of AOGD which is totally planned virtually. We thought that this is an opportunity for us to work in the current challenging conditions and bring out something exclusive. We are innovating, evolving and experimenting on different segments of the annual conference. The real challenge stands out for us is the execution of e-quiz, e-slogan competition, free paper, and e-poster competition. These segments of the annual conference are extremely important segments. So, we are focusing highly on these areas for smooth execution. Yes, we are confident that our hard work and dedication will definitely give all the delegates and the entire faculty members a unique once in a life time experience to remember and cherish.

This bulletin is dedicated to critical care obstetrics. We have the inputs from Dr. Pratima Mittal on "Assessment of critically ill obstetric patient and importance of early warning scores". Our intensivist Dr. Prakash Shastri has given us vivid description of "Analysis of Arterial Blood Gases- A Primer for Obstetrician". The importance of "Fluid and Blood Therapy in Obstetric Shock" has been nicely highlighted by Dr. Divya Pandey. "The Management of septic shock in obstetrics- The Golden first hour" has been nicely depicted by Dr. Rekha Bharti. The star academician Dr. Jyotsna Suri has given guidelines for "Approach towards acutely dyspneic gravid women: Recognition and initial management". Our prized and highly esteemed Senior faculty member Dr. Achla Batra has written "Thromboprophylaxis in pregnancy".

Elections for the post of President and Vice President of AOGD has been declared. Dr. Kiran Guleria, Professor Director of UCMS and GTB hospital is the returning officer for the same.

I hope everyone stays safe during these corona challenging times. We are expecting second wave of pandemic, so it is a request to everyone to take care of your own health. We have to indulge in academics, do our clinical work but with care and precaution. These trying times will pass off as we await a new era. **"There was never a night or a problem that could defeat Sunrise or hope" by B. Williams.**

Long Live AOGD

Dr Mala Srivastava
President, AOGD

From the Vice President's Pen

Greetings to all members of the association!

Hope you and your families are safe and doing well!

Our Editorial team has brought out this bulletin dedicated to **'Critical Care Obstetrics'** with contributions from Excellent Clinicians & Academicians from SGRH and SJH.

I would like to use this month's column as an opportunity for 'brief heads up' to you all on upcoming **42nd AOGD National Conference and the 1st E- Conference** with the theme-**"Women's Health in the Current Challenging scenario".**

Through AOGD initiatives, we have always strived to ensure our best efforts to elevate the level of care in women's health and ensuring that all women have equal opportunities to a healthy life, a healthy pregnancy and a safe delivery.

Our **'Cutting-Edge E-Conference'** agenda is a well-balanced mix of Demonstrations, Videos and Lectures which will give you the clinical and scientific updates you need across all aspects of the Gynaecology and Obstetrics to provide excellent health to all women even in the Current Challenging Scenario.

It has been designed with **Multitude of Workshops** to choose from, **E-Quiz, E-Slogan, E-Poster, Competition and Free Paper Presentations** to participate and **Win Prizes** in.

Keynote presenters, Orators, Panelists, Experts, Specialists in their field from **all over the Globe** will be on hand to share their insights. You'll hear their success stories; and get the latest updates on Newer Technology and Medical Devices. You will have more presentations than ever before with **Conference events spread out from 23rd October till 6th November 2020.**

As the saying goes- **"Coming together is the Beginning, Keeping together is Progress, Working together is Success"**

Lets leverage the upcoming event to synergize and promote excellence in Women's Health !!!

Regards,

Dr Kanika Jain
Vice President, AOGD

From the Secretary's Desk

Greetings to all !

Hope you all are in good health and safe.

As we enter into the seventh month of this ongoing COVID Pandemic, I extend by sincere gratitude to all our members for being actively involved in various academic and non-academic activities with continuing positivity.

Our editorial team has brought the AOGD E-bulletin September version dedicated to "**Critical Care Obstetrics**" which should be of great interest and immense use to our readers.

I am pleased to share that, The Association of Obstetricians & Gynecologists of Delhi is holding **42nd Annual AOGD Conference, 1st E-Conference on 30th October, 31st October & 1st November 2020**. The Theme of the Conference is "**Women's Health Care in the Current Challenging Scenario**". Pre and Post Conference Events will extend from **23rd October till 6th November** including 11 workshops on important topics, E-Quiz & E-Slogan Competition, Competition Papers, Free Communication Papers and E-Posters.

Block your dates for amazing virtual experience and visit Conference website **www.aogdvirtual.com.**

Looking forward to connect with you at the Conference.

ARISE, AWAKE AND STOP NOT TILL THE GOAL IS REACHED – Swami Vivekananda

Warm Regards

Dr Mamta Dagar
Hon. Secretary

Monthly Clinical Meeting

AOGD Monthly Virtual Clinical Meet will be organised by DDU Hospital, New Delhi on **25th September, 2020 from 04:00pm to 05:00pm**.

From the Editor's Desk

Dr Geeta Mediratta
Chief Editor

Dr Chandra Mansukhani
Co-Editor

Dr Jyotsna Suri
Guest Editor

Dear Friends

It gives me immense happiness and satisfaction to write a guest editorial for this very special issue on 'Critical Care Obstetrics'.

Safe motherhood means that all women receive a certain level of care to remain healthy and safe throughout pregnancy and childbirth. According to WHO fact sheet 2019, in 2017, 810 women died each day due to pregnancy and childbirth complications. It is also estimated that 94% of these deaths occurred in low resource setting and around 1/5th of these maternal deaths were in Southern Asia. Women can die of either complications developed during pregnancy, childbirth and postpartum, or due to preexisting chronic diseases. Conditions that account for 3/4th of the maternal deaths include antepartum or postpartum haemorrhage, sepsis, hypertensive disorders, labour complications and septic abortions.[3] Most of these maternal complications can be treated and maternal mortality can be prevented.

Availability of critical care units in developed countries has played an important role in reducing their maternal mortality. Worldwide, there has been an increase in ICU admissions which has led to development of separate ICUs for various specialties like cardiac, neurosurgery, respiratory medicine, neonatology etc, but dedicated ICUs for obstetric women are not yet widely available in most developing countries. Further the pregnant women have an altered physiology best understood by the obstetrician and also poses the unique challenge of the second life within her. Herein comes the concept of 'Critical Care Obstetrics'. Setting up of dedicated Obstetric HDUs and ICUs all over India is now an issue of top priority for the Government of India.

I have no words to thank our most dynamic AOGD Editors Dr Geeta Mediratta and Dr Chandra Mansukhani for giving me this opportunity to bring forth for our colleagues this collection of articles written by stalwarts, who are adept in the management of critically ill pregnant women. I also take this opportunity to thank my mentor Dr Pratima Mittal and my colleague Dr Rekha Bharti for all their valuable inputs in planning this issue. I hope that our readers will find them useful and will be able to translate this knowledge into their daily practice.

Dr Jyotsna Suri
Professor and in Charge Critical Care Obstetrics
VMMC & Safdarjung Hospital
Chairperson Safe Motherhood Committee, AOGD 2019-21

Assessment of Critically Ill Obstetric Patients and Importance of Early Warning Scores

Pratima Mittal[1], Supriya Chaubey[2]
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Introduction

Pregnancy is a physiological state but any complication in pregnancy can lead to a life threatening condition. These conditions can impact both maternal and fetal wellbeing and the impact is reflected by a country's maternal and perinatal mortality rates. India in recent years has registered a significant decline in Maternal Mortality Ratio (MMR), recording a 22% reduction in such deaths since 2013, according to the Sample Registration System (SRS) bulletin 2017. The MMR has declined from 167 in 2011-2013 to 130 in 2014-2016, as per special bulletin of MOHFW[1]. Contributing factors for increased MMR are deficiencies in managing critically ill obstetric patients due to knowledge gap, communication gap & poor resuscitation skills[2]. Availability of facilities where these patients can be transported and managed effectively can definitely reduce MMR of our country.

Critical illness in pregnancy may be due to conditions unique to pregnancy, due to conditions exacerbated by pregnancy or due to coincidental conditions. Most of these patients are young and disease free initially, despite which the mortality still remains higher in pregnant/ post abortal/ post-partum patient, in comparison to non-obstetric patients. Obstetric patients are different due to anatomical and physiological changes in pregnancy, which modify presentation of the problem and response to treatment. Both mother & fetus are affected by the pathology & subsequent treatment. Mother's welfare always takes precedence over fetal concerns fetal survival is usually dependent on optimal maternal management.

Provision of optimal care for the pregnant woman requires an understanding of the causes of critical illnesses, physiological changes which make obstetric patients more susceptible to organ dysfunctions and the tools to identify these patients early.

Physiological Changes in Pregnancy

There are several important anatomical and physiological changes in pregnancy which influence management of critically ill obstetric patient[3].

There is an increase of 30%–50% in cardiac output during pregnancy due to increased stroke volume and increased maternal heart rate. The mean arterial pressure is reduced due to decreased systemic vascular resistance that may be mediated by an increase in progesterone, oestrogen, and nitric oxide, which are endogenous vasodilators and decreased BP in first and second trimesters. Shock may be missed till significant blood loss is there.

The enlarging uterus may compress the aorta to produce increased afterload and compress the inferior vena cava leading to decreased cardiac return. This can lead to hypotension in the supine position, which is the most favourable position for resuscitation. Left uterine displacement (LUD) is a very important manoeuvre during resuscitation to improve cardiac output

Functional residual capacity (FRC) decreases by 10% to 25% in pregnancy as the uterus enlarges and elevates the diaphragm. Pregnancy results in increased tidal volume and minute ventilation mediated by elevated serum progesterone levels that may result in mild alkalosis and compensatory renal excretion of bicarbonate. Reduced FRC reserves and increased oxygen consumption lead to rapid development of hypoxia in response to apnoea in pregnant women.

The oxyhaemoglobin dissociation curve shifts to the right in the woman (a higher partial pressure of oxygen is required to achieve maternal oxygen saturation). Pregnancy also leads to altered renal tubular functions, a narrowing of the oncotic pressure–wedge pressure gradient that increases risk for pulmonary oedema, progesterone-mediated gastroesophageal sphincter relaxation,

prolonged intestinal transit times, and altered drug metabolism. Upper airway oedema occurring as a result of hormonal changes may reduce visualisation during laryngoscopy and increase risk of bleeding. Creatinine clearance is increased in pregnancy to 120–160 mL/min and serum creatinine level decreases to 0.4–0.7 mg/dL.

The interpretation of arterial blood gas analyses in parturient is little different, because of the above physiological changes. The pH is 7.44, little alkalotic, and $PaCO_2$ is in the range of 28–32 mmHg. Base deficit up to −5 is considered normal and bicarbonates are in the range of 20–22 mmol/L. $PaCO_2$ >35 mmHg is considered as imminent respiratory failure and >40 mmHg is respiratory failure. Pregnancy is a hyperoxic state, and PaO_2 is in the range of 95–105 mmHg. Hence, during management of parturient with respiratory failure, it is aimed to maintain PaO_2 >70 mmHg. These factors and the gestational age of the foetus have to be considered during resuscitation.

Causes of Critical Illness in Pregnancy

The majority of cases that get admitted to critical care unit are primarily obstetric disorders. More than 80% of these admissions are because of pre-eclampsia and its complications, haemorrhage, and sepsis. Causes of critical illness in pregnancy can be attributed to[3]:

Conditions Specific to Pregnancy

Preeclampsia, acute Fatty liver, obstetric haemorrhage, Amniotic fluid embolism, Peripartum cardiomyopathy

Increased Susceptibility in pregnancy

Venous thromboembolism, aspiration syndromes

Underlying medical conditions that is exacerbated by pregnancy

Congenital heart disease, pulmonary hypertension and chronic renal failure

Unrelated to pregnancy and coincidently developed during pregnancy

Diabetic ketoacidosis, pneumonia, asthma

Non-obstetric disorders in pregnancy show large geographic variations. In South-East Asian countries including India, more often pregnancy is complicated by tropical and other infectious diseases such as malaria, leptospirosis, dengue, viral hepatitis, influenza, tuberculosis, rheumatic valvular heart diseases, cerebral sinus venous thrombosis, and endocrine disorders (diabetic keto-acidosis). In the developed nations, pneumonia, bronchial asthma, trauma, cancers, drug abuse, complicated urinary infections, pre-existing autoimmune disorders, chronic pulmonary disease, endocrine disorders, and pulmonary thromboembolism are common.

Initial Assessment

Initial management of critically ill obstetric patient consists of initial resuscitation and relieving of aortocaval compression from pregnant uterus by LUD (Left Uterine Displacement). This should be followed by quick history, systemic assessment with an individual organ-based approach with special consideration of the gestational age of the foetus.

Vital Signs to assess are- respiratory rate, oxygen saturation, pulse. blood pressure, level of consciousness, temperature and urine output.

Heart Rate Should be taken manually for one minute, noting the rate, volume and regularity. Normal rate: 60-100bpm. Abnormalities should be followed with an ECG.

Respiratory Rate This is the most sensitive indicator of potential deterioration. Normally should be between 12 and 20. Rising rate often is an early sign of deterioration. Using it in conjunction with other evidence i.e. use of accessory muscles, increased work of breathing, inability to speak, exhaustion, colour of patient, can help in identifying deteriorating condition

Blood Pressure is a LATE sign of deterioration. One should be aware of what is normal for the patient. Manual Blood pressure recording may be appropriate. Fall of systolic B.P>30mm. MAP<60mm. should be considered as shock.

Oxygen Saturation: All cells are dependent on an adequate constant supply of O2 as they are unable to store it and a reduction can lead to organ dysfunction and death. 'Target saturation' is *> 95% in pregnant patient. All acutely unwel*l patients should receive supplementary

Oxygen and then titrate to readings. ABG may be required for more in depth assessment.

Level of Consciousness: Assess for drowsiness, agitation, new changes and also assess pupils

whether reacting or not. Patients only responding to pain or unresponsive is at risk of aspiration.

Assessment of mental condition can be done by AVPU Scale. (A: - Alert, V: - Responsive to Voice, P: - Responsive only to painful stimuli, U: - Unresponsive)

Glasgow Coma Scale (GCS) Neurological scales can also be used for assessment. GCS uses Eye response (1-4) Eye opening in response to various levels of stimulus, Verbal response (1-5): Verbal communication in terms of comprehensibility Motor response (1-6): Movement in response to various stimuli. GCS less than or equal to 8 is consistent with severe brain injury when applied to head injured population. GCS 9 to 12 consistent with moderate brain injury. GCS greater than or equal to 13 is consistent with minor injury.

Temperature High or low can indicate sepsis; > 38 degrees one should consider blood cultures. Low temperature can be as important as high as low temp. (Denotes unresponsive thermoregulatory centre in hypothalamus)

Urine Output: It is a sensitive indicator of hydration status and should be 0.5ml/kg/hr. Generally this is a poorly recorded observation. Acute Kidney injury leads to ↓ urine output, ↑ toxic waste and needs urgent attention

Important Laboratory Parameters

Serum lactate levels can prognosticate mortality. Less than 2.5 mmol/L – 5% mortality and more than 4 mmol/L – 75% mortality is anticipated. Fall in serum lactate levels by 10% in 6 hours predicts reduction in mortality by 20%

Urine**:** Complete urine examination should be done. Protein Creatinine Ratio > 0.3, 24 hour proteinuria > 300 mg, Urine Albumin Creatinine Ratio > 30, are bad prognostic markers

Arterial Blood Gas (ABG) Analysis should be done to see if the patient is in metabolic acidosis. pH less than 7.35, Base deficit more than – 6, Bicarbonate < 18 (NP 22), PaO2 < 80, Lactate > 4, suggest metabolic acidosis.

Coagulation Studies One should assess if patient has Disseminated Intravascular Coagulation (DIC) Platelets < 100,000, PT / INR: INR > 1.5, aPTT : test > 5 sec + control. Fibrinogen levels and FDPs should also be assessed

History, Physical examination and Investigations should help the clinician to identify the etiopathology of the critical illness in the pregnant patient.

Early Recognition of Critically Ill Obstetric Patient

One of the proposed methods to reduce both maternal mortality and morbidity has been through the use of clinical tools, like "Early Warning Systems" that would allow early recognition of patients who would likely benefit from more aggressive interventions or transfer to a higher level of care. Physiological *Track* and *Trigger* systems are clinical prediction models that involve serial clinical observations (track) with criteria (trigger) to identify patients at risk of complications. Confidential Enquiry into Maternal and Child Health (CEMACH) report of UK first recommended the routine use of the modified early obstetric warning system (MEOWS)[4,5].

Shock Index

Shock Index (SI) has been proposed as a useful and reliable tool to predict hypovolaemic states and early haemodynamic compromise (e.g., major obstetric haemorrhage) in obstetric populations even when the individual vital signs are within the normal values. Shock Index calculated by dividing heart rate by systolic blood pressure. A normal Shock Index is considered to be between 0.5 and 0.7. Score less than 0.9 indicates that risk of massive resuscitation is low and >1.4 indicates urgent intervention or stabilisation and transfer to tertiary care facility.

Modified Shock Index

Diastolic blood pressure was incorporated in SI and the modified shock index (MSI) developed, which is a ratio of heart rate to mean blood pressure (MAP). MSI is a stronger predictor of emergency patient mortality compared to heart rate and blood pressure alone, whereas SI does not have a significant correlation with emergency patient mortality rate[6].

qSOFA Score

Obstetrically modified quick-SOFA score (omqSOFA) requires only clinical data for assessment and thus can be performed quickly without waiting for the results of biochemical or laboratory tests.

Obstetrically modified qSOFA score (obstetrically modified qSOFA)

Clinical parameter	Score
Systolic blood pressure ≤90 mmHg (≤100 mmHg in non-pregnant patient)	1
Respiratory rate ≥25/min (≥22/min in non-pregnant patient)	1
Altered mentation (any state other than alert) (Glasgow Coma Scale <15 in non-pregnant patient)	1
SOFA – Sequential organ failure assessment; Infection + omqSOFA ≥2 – maternal sepsis; omqSOFA – Obstetrically modified Qsofa	

PIERS Model

The mini PIERS (Pre-eclampsia Integrated Estimate of Risk) risk prediction model can also identify pregnant women at increased risk of death or major complications of pre-eclampsia. This model included the following:

Parity

Gestational age on admission,

Headache/visual disturbances,

Chest pain/dyspnoea,

Vaginal bleeding with abdominal pain,

Systolic blood pressure,

Dipstick proteinuria.

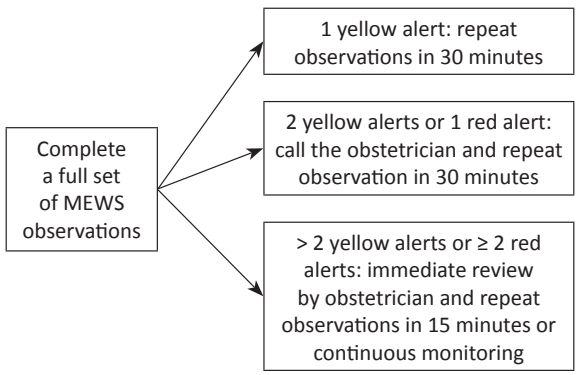
The full PIERS model also requires laboratory testing of platelet count, serum creatinine, lactate dehydrogenase, and aspartate transaminase and alanine aminotransaminase levels.

Modified Early Obstetric Warning System (MEOWS)

Confidential Enquiry into Maternal and Child Health (CEMACH) report of UK first recommended the routine use of the modified early obstetric warning system (MEOWS)[4]. Measurement of temperature (axillary), systolic blood pressure, diastolic blood pressure, heart rate, respiratory rate, oxygen saturation, conscious level (AVPU: Alert, responds to Voice or Pain, and Unresponsive), pain scores (0=no pain, 1=slight pain on movement, 2= intermediate pain at rest/moderate pain on movement), proteinuria, amniotic fluid (clear/ green) were documented at least every 12hours. A trigger was defined as a single markedly abnormal observation (red trigger), or a combination of two simultaneous mildly abnormal observations (two yellow triggers). A trigger prompted urgent medical intervention.

Table 1: Maternal early warning scores

Physiological parameters	Normal values	Yellow alert	Red alert
Respiratory rate	10-20 breaths pre minute	21-30 breaths per minute	< 10 or > 30 breathe per minute
Oxygen saturation	96-100%		< 95%
Temperature	36.0-37.4˚C	35-36 or 37.5-38˚C	< 35 or > 38˚C
Systolic blood pressure	100-139 mmHg	150-180 or 90-100 mmHg	> 180 or < 90 mmHg
Diastolic blood pressure	50-89 mmHg	90-100 mmHg	> 100 mmHg
Heart rate	50-99 beats per minute	100-120 or 40-50 beats per minute	> 120 or < 40 beats per minute
Neurological response	Alert	Voice	Unresponsive, pain



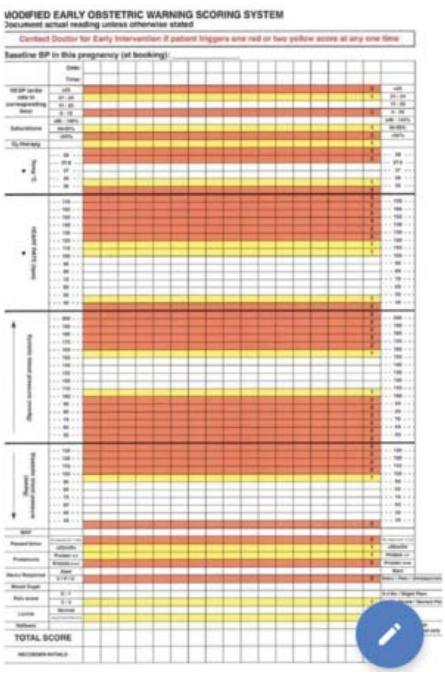


Fig 1: Obstetric early warning scores escalation protocol

However the limitation of this system was that it was developed on the basis of inputs from highly informed experts without formal prediction model, resulting in assignment of arbitrary cut offs values to continuous clinical variables. Carle et al produced a scientifically derived universal obstetric early warning score (EWS)[7]. In 2013, they came up with a statistically designed and clinically modified early warning score for the obstetric population.

The investigators utilised data from all primary obstetric admissions from the Intensive Care National Audit and Research Centre (ICNARC) over a 13 year period (December 1995 to September 2008). These data recorded the most extreme physiological measurement in the first day after admission to the intensive care unit (ICU). Significant variables were analysed with respect to mortality using a multiple logistic regression model, and then regression coefficients were used to weight each covariate. The impact of each variable was then assessed using the area under receiver operating characteristic curves. These data were then formulated to create a new, statistically-based, obstetrics EWS. There was excellent discrimination between survivors and non-survivors for this new score (area under the receiver-operating characteristic curve, 0.995).

In our institution a study has been carried out to determine validity of Carle's score in Indian setting in labour wards. 1000 patients have been assessed and the sensitivity of the score in predicting ICU and HDU admissions has been found to be significant.

The response recommended for Carle EWS is as follows

0- Routine Care

1-3- Aggregate Score for low grade response.

4-5- Aggregate Score or "3" in the vital sign for medium grade response

≥ 6- High Response

For Indian Setting 1-3 score should be referred to secondary healthcare systems, 4-5 scores to be referred to tertiary care & ≥ can be scaled upto HDU/IC

Components of Care Pathways for the Critically Ill Pregnant Woman

1. Recognition is the detection of clinical deterioration in pregnant woman that is life-threatening but potentially reversible and transferred to appropriate level.
2. Response is the provision of a multidisciplinary care plan with obstetric interventions/specialty interventions (e.g., radiological intervention as needed.
3. Level 2 critical care is the care provided in a delivery suite of a maternity unit or in a high dependency unit, justifying the recent terminology of mobile maternal critical care unit.
4. Levels 3 and 4 critical care is the care provided in a critical care unit.

Scoring Systems Used in ICU Settings

Predictive scoring systems are measures of disease severity that are used to predict outcomes, typically mortality, of patients in the intensive care unit (ICU). Such measurements are helpful for standardizing research and comparing the quality of patient care across ICU[8-10]. Disease severity scores are not the

	3	2	1	NORMAL	1	2	3
Systolic Blood Pressure, mm Hg	<80	80-89		90-139	140-149	150-159	≥160
Diastolic Blood Pressure, mm Hg				<90	90-99	100-109	≥110
Respiratory Rate/ min	<10			10-17	18-24	25-29	≥30
Heart Rate/ min	<60			60-110		111-140	≥150
$\%O_2$ required to maintain $SpO_2 \geq 96\%$				Room air	24-39%		≥40%
Temperature, ◦C	<34.0		34.0-35.0	35.1-37.9	38.0-38.9		≥39
Conscious level				Alert			Non alert

Fig 2: Carle's model of EWS

key elements of treatment, but they are an essential part of improvement in clinical decisions and in identifying patients with unexpected outcomes. They also allow an increased understanding of the effectiveness of treatment and optimizing the use of hospital resource and hence aid in the development of treatment standards.

In most of the scoring systems, scores are calculated from data collected on the first ICU day acute physiology and chronic health evaluation (APACHE), simplified acute physiology score (SAPS) and mortality prediction model (MPM). Others are repetitive and collect data every day throughout the ICU stay or for the first 3 days organ dysfunction and infection system (ODIN),sequential organ failure assessment (SOFA) multiple organs dysfunction score (MODS), logistic organ dysfunction (LOD) model and three-day recalibrating ICU outcomes (TRIOS)

Conclusion

Reducing MMR has been a part of India's Millennium Development Goals, as well as Sustainable Development Goals. The Government of India has taken various steps in this direction like the Janani Shishu Suraksha Karyakram, Pradhan Mantri Surakshit Matritva Abhiyan and many more. An early warning score if implemented at the national level could be another important step in this direction. Also it would help address the issue of skewed doctor patient ratio, especially in the rural areas and would strengthen the referral mechanisms.

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Analysis of Arterial Blood Gases — A Primer for Obstetrician

Prakash Shastri
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Abstract

The arterial blood gases (ABG) are utilised for assessment of clinical oxygenation and acid-base status in critically ill patients. Ventilation, oxygenation and acid-base status, are the three closely interrelated physiological parameters, which maintain the pH homeostasis. The correct interpretation of ABG requires the knowledge of basic physiology in relation to these parameters. Through this review article an attempt has been made to formulate a comprehensive approach by first describing the basic physiology in relation to these parameters followed by stepwise approach to analyse arterial blood gases.

Introduction

Arterial blood gases (ABG) plays a key supplementary role in deciding management strategies in critically ill patients. This role can be exemplified by considering the concept of respiratory failure. This term remains nonspecific and does not help much in deciding whether the respiratory failure is due to primary oxygenation failure or ventilatory failure. ABG is the only investigation, which can specify and quantify the respiratory failure.

High PaCO2, moderately low PaO2 and acidic pH (<7.3) indicate ventilatory failure. Also called Type II respiratory failure.

Low PaCO2, low PaO2 and alkaline pH (>7.45) indicate primary oxygenation failure or Type I respiratory failure.

The management plan will be ventilatory support in the former case and oxygen therapy in the latter case.

Basic Physiology in Relation to ABG

The ABG provides us with rapid information on following three physiologic processes, which maintain the pH

1. Alveolar ventilation,
2. Oxygenation,
3. Acid-base balance

These three processes are closely interrelated with each other and alteration in one process affects other process. For the sake of simplicity, the basic physiology in relation to each will be discussed separately avoiding the more complex details.

1. *Alveolar ventilation*: Alveolar ventilation is minute ventilation minus the dead space ventilation. The maintenance of CO2 level reflected by arterial CO2 tension (PaCO2) at any given moment depends on the quantity of CO2 produced in body and its excretion through alveolar ventilation (V̇A) and can be expressed by the equation,

   PaCO2 = CO2 production (V̇CO2)/V̇A (alveolar ventilation).

   Equation for the calculation of alveolar PO2:

   PAO2 = 713 X FiO2 – (PCO2 /0.8)

   (A – a O2 gradient = PAO2 – PaO2 (Normal: 5-25 mmHg on room air)

   PAO2 alveolar oxygen tension, Atmospheric pressure – saturated vapour pressure 760-47= 713 mm Hg

   Respiratory Quotient: Oxygen consumption/ CO2 produced (normal 0.8)

   The alveolar ventilation is that portion of total ventilation that participates in gas exchange with pulmonary blood. It is presumed that CO2 production is constant, then PaCO2 is the best index for assessment of alveolar ventilation. High PaCO2 (> 45 mmHg) indicates alveolar hypoventilation and low PaCO2 (< 35 mmHg) implies alveolar hyperventilation.
2. *Oxygenation*: The ultimate aim of oxygenation is to provide adequate delivery of oxygen to tissues. This is a function of cardiopulmonary system and various factors like arterial oxygen tension (PaO2), oxygen concentration of inspired air (FiO2) and haemoglobin content, and saturation

with oxygen contribute towards normal tissue oxygenation. The PaO2 and Pulse oximetry (SpO2) is primarily used for assessment of oxygenation status. A SpO2 >90% reliably indicates that the PaO2 is more than 60 mm of Hg.

Relation between PaO2 and FiO2: PaO2 alone provides little information regarding efficiency of lungs to transfer oxygen into the pulmonary capillary blood. That means that it does not quantify the physiological shunt (that part of pulmonary blood flow which bypasses oxygenation). This information is necessary in assessment of the severity of underlying disease in lungs (the so called ventilatory – perfusion mismatch) and in guiding oxygen therapy. There are various indices for calculation of physiological shunt, for example, classic shunt equation, which is the gold standard but which needs a bit of calculation. The simplified approach is to multiply the inhaled oxygen concentration (FIO2) by 5 (Since the normal PaO2 in an adult breathing room air with FiO2 of 20% is approximately 100 mmHg) the resultant value gives you the expected PaO2 and the difference between the observed and the measured value gives you an approximation of the percentage of venous blood that has bypassed oxygenation (the shunt). The other index of oxygenation is the PaO2/FiO2 ratio or oxygenation ratio (normal values 400-500 mmHg). PaO2/FiO2 ratio of less than 200 most often indicates a shunt greater than 20%.

Acid-base Balance: The aim of regulating acid base balance is to maintain pH within a narrow range. The pH homeostasis is accomplished through the interaction of lungs,

kidneys and blood buffers. This interaction is best represented by Henderson-Hasselbalch equation which describes the fixed inter-relationship between PaCO2, pH and $HCO3^-$ and is described as

pH = pKa + (log $HCO3^-$)/pCO2

The $HCO3^-$ is controlled mainly by kidney and blood buffers. The lungs control the level of PaCO2 by regulating the level of carbonic acid (dissolved CO2), in the blood. The major blood buffer system in body is carbonic acid/ bicarbonate base (H2CO3/$HCO3^-$). Buffer system can act within a fraction of a second to prevent excessive change in pH. Respiratory system takes about 1-15 minutes and kidneys hours to days to readjust the pH.

a. Indices for acid-base status: PaCO2 is clear marker of respiratory acid-base disturbance. A rise in PaCO2 always indicates increased carbonic acid and vice versa. The comparable metabolic index for metabolic acid base disturbance does not exist and following indices are commonly used.
   i. Plasma Bicarbonate (Actual bicarbonate): The plasma bicarbonate is a commonly used indicator and is easily calculated from PaCO2 and pH using Henderson-Hasselbalch equation. However, CO2 is also carried in blood as bicarbonate and so it is not a pure indicator of nonrespiratory acid-base problem. The normal value of $HCO3^-$ is 24 + 2 meq/l.
   ii. Standard Bicarbonate: This is plasma bicarbonate obtained after the blood has been equilibrated at 37°C with a PaCO2 of 40 mmHg. This should eliminate the hydrolysis effect.
   iii. Buffer Base (BB): Blood buffer base (BB) is the sum of all the buffer bases in a litre of blood. Thich can be sued as a metabolic index in exactly the same way as $HCO3^-$ is used. It is calculated from the measurement of pH, PaCO2 and haematocrit and reported as meq/l of base above or below the normal buffer base range. The negative base excess is often referred to as a base deficit. The normal value of BE is 0 ± 2 meq/L.

Plasma pH and K^+: The distribution of potassium ion (K^+) within intracellular and extracellular spaces affects acid base balance. In acidaemia, the excess of H^+ ions are transported to the intracellular space and to maintain electroneutrality, K^+ move out into the plasma leading to hyperkalaemia. The reverse reaction takes place in alkalemia.

In the distal renal tubule, the potassium or hydrogen ion can be exchanged for sodium ion (Na^+), depending on the body's requirement. Hence in the presence of alkalosis, H^+ions are retained and K^+ ions are exchanged for Na^+ leading to development of hypokalaemia.

Rules of Compensation

i. The compensatory response depends upon the proper functioning of organ system involved in the response (lungs or kidneys) and on the severity of acid-base disturbance. For example, the likelihood of complete compensation in chronic respiratory acidosis is less than 15% when PaCO2 exceeds 60 mmHg.

ii. Acute compensation occurs within 6-24 hours and chronic within 1-4 days. Respiratory compensation occurs faster than metabolic compensation.

iii. In clinical practice it is uncommon to see complete compensation. The maximum compensatory response in more cases is associated with only 50% to 75% return of pH to normal. However, in chronic respiratory alkalosis the pH may actually return completely to normal in some cases.

Simple Acid Base Disorders

- Metabolic Acidosis: primary decrease in HCO3
- Metabolic Alkalosis: primary increase in HCO3
- Respiratory Acidosis: primary increase in CO2
- Respiratory Alkalosis: primary decrease in CO2

Step Wise Approach to ABG Analysis

The following stepwise approach will help in correctly analysing simple acid-base disorders.

pH	$[H^+]$ nmol/L
7.7	20
7.6	25
7.5	32
7.4	40
7.3	50
7.2	63
7.1	79
7.0	100
6.9	126

Step 1: Check for internal consistency of parameters- that is the H^+ ion calculated by the following equation matches the pH.

$[H^+] = 24 \times pCO2/HCO3$

$= 24 \times 40/24 = 40$,

So [H+] is 40 at a pH of 7.4 which is consistent and the ABG is valid.

Step 2: Identify the primary problem by looking at pH and classifying it as acidotic (< 7.35) and alkalotic (> 7.45).

Step 3: What is the primary disturbance. Identify the cause of acidosis/alkalosis by looking at respiratory (PaCO2) and metabolic ($HCO3^-$) indices.

Assess the paCO2 level.

If pH decreases below 7.35, the paCO2 should rise.

If pH rises above 7.45 paCO2 should fall.

If pH and paCO2 moves in opposite direction – primary respiratory problem.

Assess $HCO3^-$ value: If pH increases the HCO3 should also increase

If pH decreases HCO3 should also decrease

They are moving in the same direction primary problem is metabolic

When the pH is in the normal range and the primary problem is not obvious, one should assess which side of 7.4 the pH is on. If pH is < 7.4, i.e. on the lower side of normal range (7.35-7.40), then the primary problem is acidosis. If pH is > 7.4, i.e. on the upper side of normal range (7.40 - 7.45), then the primary problem is alkalosis.

This reasoning is based on the concept that most of the time the compensatory response is never complete. If acidosis is a primary disturbance then the pH has been brought down to below 7.35 and the compensatory process will bring back the pH near the normal range but not in the normal range. The same logic stands true for alkalosis. However, when the pH is in the normal range this could also be because of mixed acid-base disturbance (two primary acid-base disorders). This will be elaborated later.

By classifying the pH as acidotic (< 7.35), alkalotic (> 7.45), lower normal range (7.35-7.40) and higher normal range (7.40-7.45) and considering the ventilatory parameter (PaCO2) and metabolic index ($HCO3^-$ or BE), the simple acid base disorders can be classified into four primary acid-base disorders.

a. pH < 7.35 (Acidosis)
   i. Acute respiratory acidosis (Acute ventilatory failure): PaCO2 > 45 mm Hg, $HCO3^-$ and BE - Normal.
   ii. Acute metabolic acidosis (Uncompensated PaCO2 = 35-45 mm Hg, $HCO3^-$/BE - decreased.

b. pH < 7.45 Alkalemia)

i. Metabolic alkalosis (Uncompensated): PaCO2 = 35-45 mmHg, $HCO3^-$/BE - increased.
ii. Acute respiratory alkalosis (Acute alveolar hyperventilation): PaCO2 < 35 mm Hg, $HCO3^-$/ BE -Normal.

Step 4a: If the primary disturbance is respiratory (that is it involves PaCO2), determine whether it is acute or chronic?

Respiratory acidosis

Acute: for every 10 mm Hg increase in pCO2, $HCO3^-$ increases by 1 and there is a decrease of 0.08 in pH

Chronic: for every 10 mm Hg increase in pCO2, $HCO3^-$ increases by 3 and there is a decrease of 0.03 in pH (Rule of 1 and 3)

Respiratory Alkalosis

Acute: for every 10 mm Hg decrease in pCO2 > HCO3 decreases by 2 and there is an increase of 0.08 in PH

Chronic: for every 10 decrease in pCO2, HCO3 decreases by 4 and there is an increase of 0.03 in PH (Rule of 2 and 4)

Step 4b: For a metabolic disturbance, is the respiratory system compensating OK?

Metabolic Acidosis

Winter's formula: pCO2 = 1.5[HCO3] + 8 ± 2

pCO2= last two digits of pH

Metabolic Alkalosis

pCO2 = 0.7[HCO3] + 21 ± 2

pCO2= last two digits of pH

Step 5: If there is metabolic acidosis, then what is the anion gap (AG)?

AG = $(Na^+ + K^+) - (Cl^- + HCO3^-) = 12$

In case of metabolic acidosis find out the anion gap (normal -12 ± 12 meq/L) to classify it into high anion gap acidosis or normal anion gap acidosis. Anion gap is used to differentiate between metabolic acidosis caused by increased acid production and loss of alkali.

Step 6: When high AG metabolic acidosis is present, calculate the gap-gap (Delta gap)

Delta gap is used to diagnose mixed METABOLIC acid base disorders

Delta gap is necessary to look for co-existence of two metabolic acid-base disturbances.

Delta gap = delta AG – delta HCO3-

Δ gap = (patient's AG gap – 12) – (24 – patient's $HCO3^-$)

> 6 mEq/L = metabolic alkalosis co exists with high AG metabolic acidosis

< 6 mEq/L = non-AG metabolic acidosis co exists with high AG metabolic acidosis

Mixed acid-base disorders are characterized by presence of two or more primary acid-base disorders and are not uncommon in the hospital setting. It is essential to identify these mixed disturbances by observing certain clues so that appropriate treatment can be initiated.

Common settings of mixed acid - base disorders: Following are common clinical settings of mixed acid-base disorders:

i. Metabolic acidosis/Respiratory acidosis: Severe pulmonary oedema, cardiopulmonary arrest.
ii. Metabolic acidosis/Respiratory alkalosis: Renal failure with vomiting, severe liver disease.
iii. Metabolic acidosis/Metabolic alkalosis: Renal failure with vomiting, alcoholic ketoacidosis with vomiting.
iv. Metabolic alkalosis/Respiratory acidosis: COPD with vomiting or diuretics use.

Step 7: Assess hypoxemic state: As mentioned earlier PaO2 must be assessed in conjunction with FiO2 and age and oxygenation ratio should be calculated for assessment of severity of shunting.

To summarise

Step 1: Check for internal consistency of parameters

Step 2: Acidemic, alkalemic, or normal?

Step 3: Is the primary disturbance respiratory or metabolic?

Step 4: For a primary respiratory disturbance, is it acute or chronic?

Step 5: For a metabolic disturbance, is the respiratory system compensating OK?

Step 6: For a metabolic acidosis, is there an increased anion gap?

Step 7: For an increased anion gap metabolic acidosis, are there other derangements?

Clues to Mixed Acid-Base Disorders

A 42 yearold man was brought to the ER after he was found lying in an alley with an empty liquor

bottle nearby. O/E BP=120/80, HR=110, RR=28, Temp=37°C. He was unresponsive, pupils minimally reactive to light, fundus-normal. Bibasilar crackles, DTR-brisk and symmetric, plantar-normal

ABG: pH=7.71; pCO2=35; pO2=90

Labs: Na=145; K=5; Cl=97; HCO3=12;

BUN=3; creat=1.5, glucose=110, lactate=1;

ketones-negative; salicylates=negative;

urine microscopy - calcium oxalate crystals

Step 1

(Validity) $H^{=}$ = 24 X 35/12 = 70 (which is consistent with our pH of 7.71)

So the ABG is valid

Step 2

pH is 7.71, so it is acidemia

Step 3

pH and HCO3 both are moving in same direction, so the primary disorder is metabolic

Step 4

Not required as primary disorder is metabolic

Step 5

Is Respiratory compensation adequate

$pCO2 = 1.5 \times (12 + 8 \pm 2) = 26 \pm 2$

Actual pCO2 is 35, which is more than expected so there is associated Respiratory Acidosis

Step 6

Since the primary problem is metabolic calculate the anion gap

AG = 145 – (12 + 97) = 36 which is in excess of 12 so we are dealing with HIGH AG METABOLIC ACIDOSIS (increased acid production)

Step 7

Δ gap = AG excess – HCO3 deficit

= 24-12 = 12 (more than 6) which indicates concomitant metabolic alkalosis

So now the patient has

HIGH AG METABOLIC ACIDOSIS

+

RESPIRATORY ACIDOSIS

+

METABOLIC ALKALOSIS

To conclude, knowledge of basic physiology in relation to oxygenation, ventilation and acid-base status helps us in formulating a comprehensive approach to analyse ABG. ABG report is to be interpreted in relation to its internal validity and compatibility with the patient's clinical condition before making changes in therapeutic plan.

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Fluid and Blood Therapy in Obstetric Shock

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Introduction

Obstetrics shock management requires a detailed knowledge of physiological changes during pregnancy. These vital changes are not only important for optimal fetal growth but also for protecting the mother from the pregnancy complications like hemorrhage. The key physiological cardiovascular **changes during pregnancy** include increased cardiac output, expanded blood volume, and reduced systemic vascular resistance and blood pressure.

The plasma volume rises by 50% while red cell mass increases by 20-30% and this discrepancy leads to physiologic fall in hemoglobin concentration, leading to 'Physiological Anemia'.[1] Maternal cardiac output starts rises by 30% to 50% over the non-pregnant values, near the end of the second trimester. This is due to increase in both stroke volume and heart rate. In the third trimester, heart rate rises by 10-15 beats/min above the base-line. Both systolic and diastolic blood pressures first decrease during the first two trimesters; reaching a nadir at 24-28 weeks and later at term, increase to non-pregnant levels. The systolic pressure falls by about 5-10 mmHg, while diastolic pressure falls by 10-15 mmHg. Moreover, the changes in cardiac output and blood pressure lead to an initial fall in systemic vascular resistance (SVR), followed by an increase toward non pregnant values near term. Both plasma and interstitial Colloid oncotic pressure (COP) reduce throughout pregnancy, the latter decreasing to a greater degree. There is a rise in hydrostatic pressure along with fall in plasma COP leading to oedema in late gestation.

These physiologic changes influence both the assessment of a pregnant patient's volume status as well as subsequent treatment. In this article, we will discuss pathophysiology behind shock along with fluid and blood therapy in Obstetric shock after appropriate assessment of the fluid responsiveness and amount of blood loss.

Pathophysiology of Shock

"**Shock**" refers to an acute clinical emergency characterised by circulatory failure leading to impaired tissue oxygenation and subsequently cellular dysfunction or death.

Oxygen delivery (DO_2) is calculated using the following equation

DO_2 (ml O_2/min) = Cardiac output (l/min) x Hb (gm/l) x 1.34(ml O_2/gm Hb) x %Oxygen saturation

So Oxygen delivery (DO_2) is directly proportional to cardiac output (product of stroke volume and heart rate), hemoglobin concentration (Hb) and oxygen saturation. This forms the basis of understanding pathophysiology and management of various types of shock which is actually impaired tissue oxygenation. Different types of Shock in Obstetrics are **Hypovolemic (**due to reduced intravascular volume leading to decreased preload e.g. Antepartum hemorrhage/Postpartum hemorrhage), **Cardiogenic** (due to decreased myometrial contractility e.g. Cardiomyopathy, CHF), D**istributiv**e(due to decreased systemic vascular resistance e.g. septic shock, anaphylactic shock, transfusion reaction or neurogenic shock; inversion uterus) and O**bstructive** (decreased preload due to obstruction to venous return e.g. Venous thromboembolism, trauma-cardiac tamponade or pneumothorax)

Fluid Resuscitation

This refers to administration of fluid rapidly and in boluses to patient with impaired hemodynamic status. Conventionally it has been indicated where Mean Arterial Pressure(MAP) is less than 60 mmHg or Central Venous Pressure(CVP) less than 8 mm Hg in absence of cardiogenic pulmonary edema. The **therapeutic goal of fluid resuscitation** is to improve preload to increase stroke volume and hence cardiac output. In addition to static parameters like CVP and MAP, dynamic measures like changes in stroke volume to fluid load can accurately predict fluid responsiveness.[2] Passive Leg Raise (PLR)

manoeuvre is one such test to assess volume/ fluid responsiveness. By transferring a volume of about 300 ml of venous blood from lower limbs to the right heart, PLR test mimics a fluid challenge and is thus extremely helpful in predicting whether Cardiac output will increase with volume expansion. Moreover as the hemodynamic effects are rapidly reversible and it obviates the risks of fluid overload.[3,4]

How is PLR Test Done?

It is a simple, dynamic bedside clinical technique with high sensitivity and specificity. This is used as a pseudo-fluid challenge of 150-300 ml by placing patient head end flat and feet up at a 45 degree angle from horizontal. If the patient is responsive, there will be increase in cardiac output and stroke volume. The BP is first checked with patient at 45degree head up, followed by changing the position to 45 degrees legs up. BP is rechecked after about 5 minutes in this positon. If the difference in Pulse Pressure (Diastolic BP-Systolic BP) is more than 9%, the patient is considered to be fluid responsive and is likely to get benefit from fluid resuscitation. The test can be repeated after each volume infusion to see if additional volume would be beneficial without risk of pulmonary edema.[4] Another dynamic bedside non-invasive test of knowing fluid responsiveness is to determine Inferior Vena Cava collapsibility during inspiration on ultrasound.[3]

Assessment of Blood Loss in Hypovolemic Shock

Obstetric haemorrhage is the deadliest and commonest form of hypovolemic shock which is dealt by the Obstetricians. The estimation of blood loss so as to ensure adequacy of fluid and blood products, can be done by judging clinical signs and symptoms. Owing to physiological cardiovascular adaptation in pregnancy, a pregnant woman can withstand blood loss to a larger extent than her non-pregnant counterpart. This is due to about 40% rise in blood volume which keeps her asymptomatic and compensated till blood loss as much as 1 litre. The clinical signs of blood loss are (1) increase in heart rate (2) increase in respiratory rate (3) decrease in pulse pressure (4) increase in shock index(Heart rate/SBP) (5) fall in blood pressure (6) increase capillary refill time (>2 seconds) (7) urine output<30 ml/hour (Table 1).

Table 1: Clinical Assessment of blood loss

Parameters	Compensation	Mild	Moderate	Severe
Blood loss	15%	15-30%	30-40%	>40%
Non-pregnant(ml)	750	1000	1500	2000
Pregnant(ml)	<1000	1000-2000	2000-2700	>2700
Respiratory rate (per min)	14-20	20-30	30-40	>40
Heart Rate(per min)	<100	>100	>120	>140
BP (systolic)	No change	Minor (postural) fall (80-100 mm Hg)	Marked fall (70-80 mm Hg)	Profound fall (<70 mm Hg)
DBP (diastolic)	No change	Increased	Decreased	Decreased
Mental status	Anxious	Anxious	Restless / confused / agitate	Lethargic
Urine (ml/hr)	>30	20-30	5-15	Negligible
Fluid of choice	Crystalloid	Crystalloid/Blood products	Blood products	Blood products

Table 2: Main differences between the Crystalloids and Colloids

Crystalloids	Colloids
Small solute molecules with low oncotic pressure	Large solute molecules with considerable oncotic pressure
Short intravascular stay.(30 minutes.)	Long Intravascular stay.(for 24 hours)
Administered in the ratio of 1:3	Administered in the ratio of 1:1
Used in immediate fluid resuscitation of lost volume.	Can be used in situation of massive blood loss till blood products are available or in severe hypovolemia.
e.g. Ringer Lactate, Normal Saline	e.g. Natural Colloids- Human Albumin, Synthetic- Dextran, Hydroxyethyl starch

Choice of Fluid for Resuscitation

The types of fluids available are **crystalloids and colloids**. The major differences are mentioned in Table 2. In hypovolemia due to haemorrhagic shock, Packed Cells remain the fluid of choice especially in moderate and severe shock. But for other types of shock or even in haemorrhagic shock till the blood component is available, the fluid resuscitation can be done by crystalloids.

Crystalloids versus Colloids for Resuscitation

In systematic review, there had not been any benefit of using colloids over crystalloids. Infact it may exacerbate dilutional coagulopathy due to fibrin polymerisation and platelet aggregation. The use of colloids is not recommended as part of initial management of haemorrhagic shock.[5] Moreover colloids have been associated with mortality by causing anaphylaxis, coagulopathy, Acute kidney injury (especially by HES). Infact HES is contraindicated in septic shock as it leads to acute kidney injury and even mortality.[6] Eventually the colloidal solutes tend to enter the interstitial space and lead to third space loss thereby again creating a state of low intravascular volume.

Among crystalloids, Normal Saline (NS) and Ringer Lactate (RL) are the fluids of choice. Normal saline is considered safe with small concern of normal anion gap metabolic acidosis of uncertain significance. RL has most physiologic resemblance to plasma. It is relatively contraindicated in hyperkalemia. Moreover it should not be given along with blood transfusion due to tendency of calcium to bind with citrate in blood. Dextrose 5% is isotonic but after administration as the glucose gets metabolised and gets converted to free water and thus hypotonic. Thus should never be used as resuscitative fluid in women who are prone to develop cerebral edema like preeclampsia.

Initial Fluid Therapy: Optimum Dosing

Resuscitative fluids are given in boluses. This can be done by peripheral line of 14 or 16 gauge cannula or through central venous line. One to two litres fluid is given at a time in patient in shock but without Congestive Heart Failure (CHF) or End Stage Renal Disease (ESRD). The initial fluid is given at 30 ml/kg body weight in patients with septic shock. However the dose of fluid bolus is adjusted according to patients' status. In women with mild CHF or ESRD it is reduced to 500 to 1 litre while in those with severe CHF, the bolus is reduced to 250-500 ml. The clinical parameters should be reassessed after each bolus. Even the fluid responsiveness by passive leg raising test can be judged after each bolus to effectively manage shock without causing overload.

Monitoring of Response and Effect of Resuscitation

It can be assessed by the following parameters (1) **Mean arterial pressure≥** 65 mm Hg (2) urine output ≥ 0.5 ml/kg/hr (3) Central Venous Pressure between 8-12 mm Hg (4) Lactate levels normalizes (5) mental state improves or normalises.

Blood and Component Therapy

World Health Organisation strategy emphasizes the need to reduce unnecessary transfusions. Thus blood component therapy should be given judiciously. **The goal of blood component therapy** is (1) to restore intravascular volume (2) to restore the oxygen capacity of blood by replacing red blood cells. (3) to replace clotting factor and correction of anemia.

Significant blood loss resulting in symptomatic decreased oxygen-carrying capacity requires blood transfusion. The indications of blood transfusion in acute blood loss are (1) Moderate to severe shock (2) Diastolic blood pressure <60 mmHg (3) Fall in Systolic blood pressure >30 mm Hg (4) Oliguria/Anuria suggestive of acute renal failure (5) Tachycardia (>100beats/min) (6) Mental status changes (7) Shortness of breath, light-headedness or dizziness with mild exertion.

Blood Component Therapy

In modern practice, blood component therapy is preferred and has practically eliminated the use of whole blood in hypovolemic shock. Reasons for preferring blood components over whole blood are decreased availability due to short shelf life (less than 7 days old) and inactivity of platelets, factor V and factor VIII after 24 hours. Volume overload is also avoided. Broadly plasma expanders are needed for hypovolemia correction, packed cells

are needed for increasing oxygen carrying capacity, FFP and cryoprecipitate for clotting factors and platelets for thrombocytopenia correction.

Massive Transfusion Protocol

For blood component therapy in massive obstetric haemorrhage, there should be two I/V lines. Through first line packed cells transfusions, and through second line cryoprecipitate, platelets and FFP should be given. Massive transfusion protocol is also known as **rule of 4**. In patients with who need massive transfusion, resuscitation should be started as soon as possible to prevent dilutional coagulopathy. The blood products are transfused in a ratio of 4 units Packed celld:4 units of FFP:4 units of platelets:4 units of cryoprecipitate.

Various Blood Products Transfused in Clinical Practice

1. **Whole Blood**

   A unit of whole blood has a volume of 350 ml. One unit increases hemoglobin by 1 g/dl and hematocrit by 3%. It is used only in case of non-availability of Packed Red Blood Concentrates (PRBCs). There is no justification for whole blood transfusion to stop bleeding due to coagulopathies. Care must be taken and it should be used within 30 minutes of removal from refrigerator. It should be transfused at the rate of 150-200 ml/hr and must be completed within 4 hours (discard unit if this period has exceeded). ABO and Rh compatibility and Cross matching are a must. However, in emergency, till availability of compatible blood, O negative blood can be used.

2. **Packed Red Blood Cell Concentrates (PRBCs) / Packed Cell Volume (PCV)/Red Cell Concentrates (RCC)/Plasma Reduced Blood (PRB)**[7]

   A unit of PRBC has a volume of 200 ml and one unit increases hemoglobin by 1 g/dl and hematocrit by 3%. It is indicated in cases with severe anemia during pregnancy associated with maternal decompensation, severe acute blood loss following spontaneous delivery or cesarean section, intrapartum Hb < 7 gm%, replacement of acute blood loss in obstetric hemorrhage along with other components and post-partum anemia with signs of shock. It must be used within 30 minutes of removal from refrigerator and should be completed within 4 hours (discard unit if this period has exceeded). The rate of transfusion should be 150-200 ml/hr. The target Hb to be achieved is 7-9 gm/dl. ABO and Rh compatibility and cross matching is a must. In emergency, till availability of compatible blood, O negative blood can be used.

3. **Platelet Rich Plasma (PRP)**

   One PRP unit has 50-70 ml volume and increases platelet count by 5-8x10^9 /l. One Platelet pharesis unit increases platelet by 40-50x10^9 /l. It is indicated in bleeding due to thrombocytopenia, with count less than 50X10^9 /l where surgery or delivery is anticipated and cases where platelet count is less than 20X10^9 /l. The rate of transfusion should be 50-150 ml/hr. A unit of PRPs should be transfused immediately and should be completed within 30 minutes of removal from optimal storage condition. The target platelet count to be achieved is 50X10^9 /l . ABO and Rh compatibility is needed.[7]

4. **Fresh Frozen Plasma (FFP)** [7]

   One unit of FFP has 50-70ml volume and 1 ml of FFP contains 1 unit of coagulation factor activity. It is indicated for replacement of multiple coagulation factors, Disseminated Intravascular Coagulation (DIC), depletion of coagulation factors with patients with large amount of transfusion and warfarin overdose. The dose of FFP is 12-15ml/kg and rate of transfusion is 150-300 ml/hr. It should be transfused as soon as possible preferably, but can be done within 6 hours of thawing and must be completed within 30 minutes. The aim of transfusion is to achieve target PT and APTT INR less than 1.5. ABO compatibility is needed; however cross matching is not required. Anti D prophylaxis is not needed, if an Rh negative woman has received Rh positive FFP.

5. **Cryoprecipitate**[7]

   It is indicated in correction of microvascular bleeding in massively transfused patients with hypofibrinogenemia. It has no advantage over FFP and is especially useful in patients who need fluid restriction. The rate of transfusion should be 150-300 ml/hr. It should be transfused as soon as possible preferably but can be done within 6 hours of thawing. The transfusion should be

completed within 30 minutes. The target is to achieve Fibrinogen level more than 150 mg/dl. Although ABO compatibility is preferred, yet it's not essential. 1 unit/10 kg body weight raises plasma fibrinogen by 50mg/dl. Anti D prophylaxis is not needed, if Rh negative women receive Rh positive cryopecipitate.

7. **Newer Alternative: Recombinant Factor VIIa (rFVIIa)**

   It is a promising new alternative to blood component therapy. The mechanism of action is to augment the intrinsic clotting pathway by binding with tissue factor and directly activating factors IX and X. Its use may be considered as a treatment for life-threatening postpartum hemorrhage (PPH), but should not delay or be considered a substitute for a live-saving procedure such as embolization or surgery, or transfer to a referral centre. The most commonly reported effective dose is 50-100 ug/kg intravenously every 2 hours until haemostasis is achieved, with vast majority of patients requiring only one dose.[8]

   There is no evidence to support the prophylactic use of rFVIIa to reduce blood loss for caesarean section. It is important to ensure adequate levels of platelets and clotting factors because rFVIIa increases clotting by acting on these subtrates.[9] There is no risk of viral transmission as the drug is derived from recombinant technology.

Conclusion

Careful assessment of volume status and fluid responsiveness are important determinants of outcome in shock. Dynamic tests like passive leg raising tests are preferred over static tests like MAP and CVP to determine fluid responsiveness. Crystalloids are the fluid of choice for resuscitation. Normal Saline and Ringer Lactate are equivalent though Ringer lactate may lead to lesser acute kidney injury. A wide bore cannula should be used for fluid resuscitation and fluids should be given in boluses. Adequacy of resuscitation should be assessed before giving another bolus. Subsequently patient can be shifted to maintenance therapy. However for haemorrhagic shock, packed cell remains the fluid of choice. Blood and its components are life-saving but with inherent risks. So a blood transfusion should be judiciously ordered. The collected blood should be separated into its components and used in conditions with specific requirements for optimal utilization. The aim should be to minimize unnecessary blood transfusions, thereby promoting proper use. Recombinant factor VIIa is a new adjunct for treatment of massive hemorrhage and should be considered, if available.

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Management of Septic Shock in Obstetrics- The Golden First Hour

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Introduction

Sepsis is derived from Greek sēpein (make rotten) in the sense of putrefaction. It is a common and fatal problem which should be recognised as medical emergency. Prompt recognition and rapid resuscitative measures are of utmost importance to prevent mortality. When it comes to obstetrics, it causes 11% of maternal deaths worldwide and is the 3rd most common direct cause of maternal death.[1] The prevalence found to be 16.5/10,000 live births in a recent study conducted in Delhi.[2]

Definition of Sepsis and Septic Shock

The term sepsis and septic shock has been defined with help of clinical and laboratory criteria. It is linked with infection and inflammatory response, but the defining parameters have changed since times.

The Sepsis-3 task force in 2016 has given the new definition of sepsis and septic shock.[3]

Sepsis is now defined as 'life threatening organ dysfunction caused by dysregulated host response to infection'. It is diagnosed as presence of infection and a raised Sequential Organ Failure Score (SOFA), with a change of score of 2 or greater. (Table 1)

Septic shock is defined as a 'subset of sepsis with profound circulatory, cellular and metabolic abnormalities associated with a greater risk of mortality than sepsis alone'. It is diagnosed as sepsis with

- Vasopressor requirement to maintain a MAP of >65 mmHg
- Serum lactate level >2 mmol/L

WHO (2018) has defined maternal sepsis as 'a life threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post abortion or postpartum period'.[4]

Role of Quick SOFA

SOFA is well known within the intensive care community, but not applied widely. It is complex, cumbersome and also the information required to calculate the score may not be available at the first point of contact when the patient is acutely ill. The task force developed a simple bedside tool to identify those with infection who are more likely to have poor outcomes, quick SOFA. (Table 2)

Table 2: q SOFA Parameters

Parameter	Point
Respiratory Rate	≥22/ min
Systolic Blood Pressure	≤ 100 mm Hg
Mentation	GCS< 15

Those who fulfil two of these criteria have similar outcomes to those having an increase in 2 points on full SOFA. Therefore, task force recommends use of qSOFA to prompt clinicians to:

- Further investigate for organ dysfunction
- Initiate or escalate therapy as appropriate, and

Table 1: Sequential Organ Failure Assessment Score (SOFA) Score

SOFA Score	**0**	**1**	**2**	**3**	**4**
Respiration PaO_2FIO_2 or SaO_2/FIO_2 mmHg	> 400	< 400 221-301	< 300 142-220	< 200 67-141	< 100 < 67
Coagulation	> 150	< 150	< 100	< 50	< 20
Liver Bilirubin (mg/dL)	< 1.2	1.2-1.9	2.0-5.9	6.0-11.9	> 12.0
Cardiovascular Hypertension	No hypertension	MAP < 70	Dopamine ≤ 5 or any	Dopamine > 5 or norepinephrine ≤ 0.1	Dopamine > 15 or norepinephrine > 0.1
CNS (GCS)	15	13-14	10-12	6-9	<6
Renal Crestinine (mg/dL) or urine output (ml/dL)	< 1.2	1.2 - 1.9	2.0 - 3.4	3.5 - 4.9 or < 5.00	> 5.0 or <200

- Consider referral to critical care or increase the frequency of monitoring, if such actions have not already been undertaken.

Identifying infection as the cause of organ dysfunction is solely dependent on the clinician. And when there is presence of pre-existing chronic illness or other cause causing organ dysfunction, they may interfere with accuracy of qSOFA or SOFA. It can lead to misdiagnosis and unnecessary treatment with antimicrobials. The SIRS criteria for diagnosing sepsis has a higher sensitivity, and studies have suggested to use it to detect sepsis, while qSOFA should be used only as a triaging tool.[5]

Pathogenesis

The pathogenesis is not completely understood till date. The host response to infection plays an important role, initial pro inflammatory pathways are activated, anti-inflammatory pathways are also activated and can down regulate corrective responses later in the course of sepsis. The four main features are endothelial dysfunction, coagulopathy, cellular dysfunction and cardiovascular dysfunction. (Figure 1)

Risk Factors for Sepsis in Obstetrics

- Low socio economic status
- Poor nutrition
- Primipara
- Anaemia
- Obesity
- Prolonged labour with multiple (>5) vaginal examination
- Diabetes
- Not maintaining asepsis
- Vaginal trauma
- C-section
- Retained products of conception
- Prolonged rupture of membranes

Management: "Golden Hour of Sepsis"

The optimum management of sepsis lies in early recognition and prompt initiation of resuscitation. The window between the onset and identification of sepsis is often where significant delays in management occur.[6] The initial resuscitation and the investigations goes hand in hand. The investigations are aimed at identifying the infective organism and to assess the organ functions as shown in Table 3

Table 3: Investigations for sepsis

Assessment for end organ hypoperfusion	Assessment of infective organism
Complete blood counts	Blood culture
Kidney function tests	High vaginal swabs
Liver function tests	Urine culture
Serum electrolytes	Wound swab culture
Arterial blood gases with lactates	Products of conception for culture
Coagulation profile	Any other- peritoneal fluid/ pleural fluid culture if relevant
Blood sugar	

The **"golden hour of sepsis"** stresses the relationship between timely initiation of antibiotic treatment and outcome. Studies have found that each hour delay in treatment reduces sepsis survival by 7.6%.[7] The surviving sepsis campaign (2016) has also given key recommendations about management of sepsis

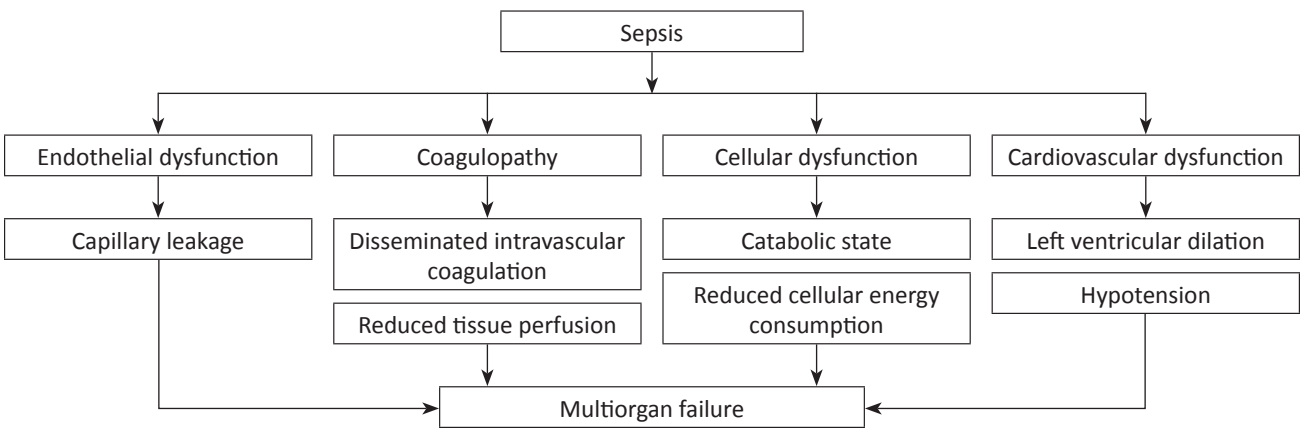

Fig 1: Pathophysiology of sepsis leading to multi organ failure

and septic shock. Data from a prospective cohort study from the SSC showed that compliance with SSC bundles led to a 25% relative risk reduction in mortality.[8]

Early Management Comprises Six Management Bundle to be Completed within 1 hour

- Administer oxygen to maintain SpO_2 at >94%
- Take blood cultures and consider infective source
- Administer intravenous antibiotics. Intravenous antibiotics should be started within 1 hour of sepsis recognition and should include combination therapy (at least two classes of antibiotics to cover a known or suspected pathogen) for patients with septic shock. Combination therapy should not routinely be used for patients without shock.
- Check serial lactates. Results from SSC database has found that patients with lactate >4 mmol/L had significantly increased mortality and there is a linear relationship between lactate and mortality. A baseline lactate helps in monitoring the resuscitation by targeting its clearance. Another entity is 'cryptic septic shock' that is those who have end organ hypoperfusion without hypotension due to compensation. Difficulty in recognition causes delay and poorer outcomes. Lactate is used to identify this condition.
- Fluid resuscitation with crystalloids guided by lactates. 30 mL/ kg of intravenous crystalloid within 3 hours and should be re-assessed frequently. Commence hourly urine output measurement. Colloids may be associated with increased risk of acute kidney injury.[9,10]
- Early administration of vasopressors to maintain MAP of > 65 mm Hg. Norepinephrine is the first choice for patients who need vasopressors. Vasopressin or epinephrine can be added.

Antimicrobial Therapy

According to the SSC guidelines, administration of IV antimicrobials should be there within one hour after recognition of both sepsis and septic shock. Appropriate culture should be taken to guide the future antimicrobial therapy before beginning the same, however, if there is delay in obtaining culture samples antimicrobial therapy ***should be initiated and not delayed***. Empirical broad spectrum antibiotics should be started, combination therapy is always preferred than monotherapy. Antimicrobial therapy should be tailored adequately or changed after identifying the causing organism/organisms. Routine administration of antifungal therapy is not warranted in non-neutropenic patients.

National Treatment Guidelines for Antimicrobial Use in Infectious Diseases, GOI recommends antimicrobial therapy as follows[11]

Sepsis in Pregnancy/after pregnancy- Piperacillin-Tazobactam 4.5 g IV 6 hourly or Cefoperazone+Sulbactam 3 g IV 12 hourly and MRSA cover with Vancomycin/ Teicoplanin may be required.

Chorioamnionitis- Clindamycin/ vancomycin/ teicoplanin and cefoperazone- sulbactum, if patient is not in sepsis then IV Ampicillin.

Septic abortion/Endomyometritis/Septic Pelvic Vein Phlebitis- Empirical therapy with Ampicillin 500 mg 6 hourly + Metronidazole 500 mg IV 8 hourly. In case of previous partial treatment with antibiotics, send blood cultures and start Piperacillin-Tazobactam or Cefoperazone-sulbactam till the sensitivity report is available. Alternative regimen- Ceftriaxone 2g IV OD.

Uncomplicated Pyelonephritis- Empirical therapy with Amikacin 1 g OD IM/IV or Gentamicin 7 mg/kg/day OD, Alternative regimen- Piperacillin-Tazobactam 4.5g IV 6 hourly or Cefoperazone+Sulbactam 3 g IV 12 hourly or Ertapenem 1 g IV OD.

Complicated Pyelonephritis- Empirical therapy with Piperacillin-Tazobactam 4.5 gm IV 6 hourly or Amikacin 1 g OD IV or Cefoperazone-Sulbactam 3gm IV 12 hourly. Alternative regimen- Imipenem 1g IV 8 hourly or Meropenem 1gm IV 8 hourly. De-escalate to Ertapenem 1 gm IV OD, if Imipenem/ meropenem is initiated. Monitor renal function if aminoglycoside is used.

Necrotizing fasciitis- Empirical therapy with Piperacillin-Tazobactam 4.5 gm IV 6 hourly or Cefoperazone-Sulbactam 3 gm IV 12 hourly + Clindamycin 600-900 mg IV 8 hourly. Alternative regimen- Imipenem 1g IV 8 hourly or Meropenem 1gm IV 8 hourly + Clindamycin 600-900 mg IV TDS/ linezolid 600 mg IV BD/daptomycin 6mg/kg/day.

Secondary peritonitis, Intra-abdominal abscess/ GI perforation- Empirical therapy with Piperacillin-Tazobactam 4.5 gm IV 8 hourly or Cefoperazone-Sulbactam 3 gm IV 12 hourly, In sick patients-fluconazole iv 800 mg loading dose day 1, followed by 400 mg OD. Alternate regimen- Imipenem 1g IV 8 hourly or Meropenem 1gm IV 8 hourly or Doripenem 500 mg 8 hourly or Ertapenem 1 gm IV OD. Source control is important to reduce bacterial load, if excellent source control is achieved antimicrobials for 5-7 days; other wise 2-3 weeks.

Community acquired Pneumonia- Empirical therapy- Amoxycillin-clavulanate 1.2 g IV TDS or Ceftriaxone 2g IV OD Duration 5-8 days. Alternative regimen- Piperacillin-Tazobactam 4.5 gm IV 6 hourly or Imipenem 1g IV 6 hourly or Cefoperazone-Sulbactam 3 gm IV 12 hourly. If MRSA is a concern-add Linezolid 600mg IV/Oral 12 hourly. If atypical pneumonia suspected- Doxycycline 100mg 12 hourly or Azithromycin 500 mg oral/IV OD.

Mastitis with abscess- Drainage with antibiotic cover for MRSA, Clindamycin 300 QID or Vancomycin 15 mg/kg IV 12 hourly (maximum 1gm 12 hourly) Or Teicoplanin 12 mg/kg IV 12 hourly x 3 doses followed by 6 mg once daily IV

Other Important Recommendations of Surviving Sepsis Campaign

Definitive treatment- Source control

The SSC guidelines recommended that this should take place as soon as possible after stabilising the patient and within first 12 hours after diagnosis; the least invasive procedure should be used. This may include drainage of infected fluid collections, evacuation of infected products of conception, delivery of fetus and placenta in chorioamnionitis, debridement of infected solid tissue and removal of devices and foreign bodies.

Role of sodium bicarbonate

Sodium Bicarbonate is not usually indicated if pH on arterial blood gases is > 7.15. Instead the initial interventions including fluid resuscitation, oxygenation and vasopressors should be used to normalise the arterial pH.

Transfusion of blood and blood products

Those who have haemoglobin level <7 g/dl must receive red cell transfusion (packed red cell transfusion is better than whole blood transfusion); however the threshold is higher in patients with active bleeding, severe hypoxia or myocardial ischaemia. Indications for platelet transfusion are < 10,000/mm^3 without bleeding, <20,000/mm^3 if there is bleeding or the patient receiving chemotherapy and <50,000/mm^3 if invasive procedure are planned. FFP is not required unless there is intercurrent bleeding or invasive procedure is planned in presence of coagulopathy.

Role of corticosteroids

They help in combating the relative adrenal insufficiency caused by sepsis and in septic shock; they have a vasopressor sparing role. However they are not recommended if fluid resuscitation and vasopressor restore the hemodynamic stability, rather they can be used as adjuvant therapy where higher dose of vasopressor fail to achieve adequate MAP.

If corticosteroids are used in septic shock, current guidelines recommend intravenous hydrocortisone 200 mg per day as a continuous drip or 50 mg bolus in 4 divided doses for at least 3 days. There is no clear consensus about duration of therapy, whether to tapper the dose or not, but in practice mostly steroids are stopped after cessation of vasopressors.

Biomarkers

They facilitate early diagnosis, monitor the progress of disease, and direct the management and also the prognosis. These are CRP, ESR, procalcitonin, galactomannan, beta D glucan and cytokines (IL 6,8,10). Among all procalcitonin can guide de-escalation of antibiotics and reduces duration of antibiotic exposure. It helps in decision making with clinical assessment. Individual marker has limited sensitivity and specificity.

Recent Advances

In 2012, the sepsis guidelines recommended protocolized resuscitation with quantitative end points on the basis of Early Goal Directed Therapy (EGDT)[12]. But recently three randomized trials have shown no outcome benefit from EGDT compared with usual care in septic shock. These are, the Protocolised Care for Early Septic Shock (ProCESS), the Australian Resuscitation in Sepsis Evaluation (ARISE) and the Protocolised Management in Sepsis (ProMISe) trial.[13-15] The new SSC guidelines de-emphasise the protocolisation and recommend that patients should be re-evaluated frequently and patient-specific tailoring of management.[8]

Conclusion

To conclude, it is very crucial and critical to act fast in a case of sepsis. The one hour sepsis bundle emphasis that all the following interventions within the first hour of recognition of sepsis- namely oxygenation, fluid resuscitation, serial monitoring of lactates, early administration of vasopressors, administration of broad spectrum antibiotics ideally after taking cultures (but antibiotics not to be withheld if that is not feasible) are associated with lower morbidity and mortality. This should be followed as early as possible by the definitive management, which is source control wherever applicable.

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Approach Towards Acutely Dyspneic Gravid Woman: Recognition and initial management

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Introduction

Dyspnea is defined as a "subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity"[1]. Acute dyspnea is the development of symptoms over hours to days whereas chronic dyspnea is presence of symptoms for last 4-8 weeks.

There can be several causes of dyspnea in pregnancy which can range from physiological dyspnea of pregnancy to serious causes involving the lungs or cardiovascular system. In this article we will discuss the characteristics of physiological dyspnea and recognition of acute respiratory failure along with its initial management. A typical scenario presenting in emergency with acute dyspnea will also be illustrate.

Etiology of Dyspnea in Pregnancy

There are several causes of dyspnea in pregnancy. They can be divided into pulmonary, cardiac, metabolic, miscellaneous and causes specific to pregnancy. The causes are shown in Table 1.

Physiological Changes in Pregnancy Involving the Respiratory System and 'Physiological Dyspnea'

Physiological dyspnea is a commonly seen condition in pregnancy and is due to the following reasons

- Under effect of progesterone, depth of respiration increases; rate however remains constant[2,3].
- Tidal volume increases by 40%
- The minute volume (respiratory rate * tidal volume) *increases* by 40%, from 7.5 L/min to 10.5 L/min
- The increase in ventilation is greater than the corresponding elevation in oxygen consumption (approximately 20 percent)

Physiological Dyspnea can be Differentiated from Pathological Dyspnea by the following:

- Physiological dyspnea begins from the end of 1st trimester/beginning of 2nd trimester and is gradual in onset.
- Is not associated with symptoms like cough, chest pain or fever
- Is not associated with abnormal findings on respiratory and cardiovascular examination such as crackles, rhonchi, whispering pectoriloquy, broncophony or murmurs on CVS examination.

Table 1: Causes of dyspnea in pregnancy

Pulmonary	Cardiac	Metabolic	Miscellaneous	Specific to pregnancy
COPD	Rheumatic heart disease	Sepsis	Anxiety	Preeclampsia
Asthma	cardiomyopathy	Diabetic ketoacidosis	Hyperventilation	Eclampsia
Pneumonia Bacterial Viral- H1 N1; COVID 19	High output failure	Anaemia	Anaphylaxis	Peripartum cardiomyopathy
ARDS	Arrhythmias	Salicylate, CO and organophosphorus poisoning	Ascites/effusion	Tocolytic induced pulm edema
Pulmonary embolism	Cardiac tamponade		Stroke	Amniotic fluid embolism
Pneumothorax	Acute coronary syndrome		Massive obesity	Pregnancy induced physiological dyspnea
Pulmonary contusion	Decompensated heart failure			

Acute Respiratory Failure in Pregnancy

Acute respiratory failure is a rare but life threatening cause of dyspnea in pregnancy. Acute respiratory failure requiring mechanical ventilation may occur in 0.1-0.2 % of all pregnancies[4].

Common Causes of acute respiratory failure in pregnancy

1. Pulmonary edema- non cardiogenic-most commonly due to preeclampsia/eclampsia
2. Pulmonary edema –cardiogenic- CCF due to peripartum cardiomyopathy, rheumatic heart disease, dilated cardiomyopathy
3. Community-acquired pneumonia
4. Viral pneumonia- H1N1, COVID 19
5. Acute Respiratory Distress Syndrome (ARDS)- pulmonary or extra pulmonary causes
6. Aspiration pneumonitis especially in eclampsia
7. Pulmonary embolism
8. Asthma exacerbation
9. Amniotic fluid embolism

Recognition of Acute Respiratory Failure

1. Rapid shallow breathing- RR > 25/min; as discussed above, the physiological changes of pregnancy results in increased tidal volume which increases the depth of respiration. However the rate remains same. An increase in respiratory rate is an important sign of maternal deterioration.
2. Use of accessory muscles of respiration and flaring alae nasi
3. Confusion, agitation, somnolence which may be due to brain hypoxia
4. On auscultation of chest- Crackles, wheeze, whispering- pectoriloquy, bronchophony
5. On pulse oximetry a low SpO2 < 90% is a sign of respiratory failure
6. On ABG partial pressure of oxygen (PaO2) < 60 mm Hg

Initial Management of Acute Respiratory Failure

Initial management of all cases with respiratory failure is same with the aim to stabilize the patient. Only after the patient is stabilized is the definite management instituted according to the diagnosis[5,6]. The steps of initial management are

1. Oxygenate the patient – **The goal is to achieve a saturation of >95% which translates to a PO2 > 70 mm Hg.** The preferred method of administering the oxygen depends upon the severity of the hypoxemia. For patients with mild hypoxemia, administration via nasal cannula may be sufficient. More severe hypoxemia generally requires administration via a facemask, high flow nasal cannula, or a non rebreather mask. The important features of different devices which are used are to deliver oxygen are:
   - Prongs- nasal prongs are used when the requirement of oxygen is from 1L/min to 6 L/min (Fig 1). They are used to deliver a FiO2 (Fraction of inspired oxygen) of 0.24 to 0.44. If requirement is more than 6 L/min they will not be useful and the above goal is not met a face mask should be used.
   - Face mask- A simple face mask can deliver a FiO2 of upto 0.6 and is used with oxygen flow rate of 5L/min to 8L/min (Fig 2). So if requirement is more than 8 L/min, we should use the next device
   - Oxygen mask with reservoir bag (Fig 3)- If requirement of oxygen is more than 8 L/min, the mask with reservoir bag should be used. These are of 2 types- partial re-breathing mask (8-12 L/min, delivers FiO2 of 0.5-0.7) and non-rebreathing mask (10-15 L/min, delivers FiO2 of 0.7-0.9). These masks are very useful for short term oxygenation when high FiO2 is required. Any patient who needs a high FiO2 of more than 0.6 for many hours should receive positive pressure ventilation
4. If the patient is not oxygenated with the above measures she will need positive pressure ventilation, which may be either non-invasive ventilation or invasive ventilation after endotracheal intubation. Noninvasive ventilation has the advantage that it avoids intubation. However due to the raised intragastric pressure during pregnancy and decreased stomach emptying time, these patients are more prone to aspiration. On the other hand intubation in pregnancy is also more difficult and challenging because of difficult airway.

   The main indications for intubation are as follows:
   - Not achieving oxygenation goals with prongs/face mask

- Not getting ventilated- as indicated by high PCO2 on ABG
- Circulatory collapse
- Altered mentation with GCS <8

5. Secure good IV access for any fluid or injectable drugs
6. Urgent investigations which are required for therapeutic and diagnostic decision are:
   - ABG- identifies and quantitates the severity of any ventilatory abnormalities. It also guides ventilator adjustments in mechanically ventilated patients. Ventilatory goals are different in pregnant patients. The target $PaCO_2$ is 30 to 32 mmHg, since this is the normal level during pregnancy. Marked respiratory alkalosis should be avoided because it may decrease uterine blood flow[7].
   - X-Ray Chest to narrow the differential diagnosis and confirm placement of ET tube
   - ECG to assess cardiac status
   - ECHO is a very important investigation in acute respiratory failure. It can rule out peripartum cardiomyopathy and congestive heart failure due to rheumatic heart disease and congenital heart disease.
   - POCUS (Point of Care USG)- Lung ultrasound can help in diagnosisng pneumothorax, pleural effusions, pulmonary edema and pulmonary consolidation. There are specific ultrasound patterns seen for a normal lung, a pneumothorax, interstitial edema, and pneumonia
   - Baseline KFT, LFT, serum electrolytes to evaluate the organ functions
   - A complete blood count- Leuckocytosis can point towards sepsis/ARDS and leucopenia can be a result of viraemia and also in severe sepsis. Thrombocytosis may be an indication of purulent collection in closed spaces (empyema, pyoperitoneum) whereas thrombocytopenia may be seen in sepsis and DIC.
   - A urine routine and microscopic and culture is essential as this is pyelonephritis is one of the common cause of sepsis and ARDS in pregnancy.
   - A viral culture panel including swabs for H1N1 and COVID 19
7. Other focused investigations according to suspected pathology which has been zeroed down by initial investigations, for example radionuclide scan (V/Q perfusion) or CT pulmonary angiography for suspected pulmonary embolism, brain natriuretic peptide(BNP) for heart failure
8. Specific treatment of patient according to the suspected or diagnosed pathology. A case of pulmonary edema which is one of the commonest presenting cases is described in Box 1[8-10].
9. After initial stabilization of mother, the fetal gestational age and fetal wellbeing are assessed
10. Expedite delivery after stabilization of the patient especially if patient is near term.

Conclusion

Hence it can be concluded that though dyspnea in pregnancy is a common complaint because of the physiological changes of pregnancy, it should be differentiated from the serious and life threatening causes of acute respiratory failure. The most common cause of acute respiratory failure in pregnancy is pulmonary edema. The initial approach consists of oxygenating and ventilating the patient. Once stabilized the diagnosis is established and definitive management started.

Fig 1: Nasal Prongs

Fig 2: Simple face mask

Fig 3: Face mask with reservoir bag

BOX 1: Case Study of acute respiratory failure

Mrs X, 32 years G2P1 at 34 weeks gestation, came to the emergency with history sudden onset breathlessness for the past 4 hours. She also gave a history of swelling of feet for the past 3 weeks. Patient was being followed up in a dispensary; last visit was 2 weeks ago when BP was documented as 130/90mm Hg.

On Examination her respiratory rate was 38/min, Pulse110/min, BP-160/110mmHg, JVP-not raised, Pallor +, edema feet ++, Bilateral chest auscultation showed basal crepitations, Per abdomen her uterine fundus corresponded to 32 weeks and was relaxed. Fetal heart tones were present. Her SpO2 was 80 % and urine albumin+++.

Management: Oxygen was started with face mask@ 8 L/min; Inj Lasix 40 mg was given I/V. Her SPO2- improved to 92%. Magnesium sulphate loading dose was given 4 g I/V and 5 g I/M on each buttock. Inj Labetalol 20 mg was also given I/V. Arterial blood sample was taken for ABG analysis. Blood was also drawn for hemogram with platelets, kidney and liver function tests, coagulation profile, serum electrolytes, blood grouping and cross matching. Indwelling Foleys catheter was inserted. In the mean time the reports were available which revealed

- ABG- PH-7.5, PO2-52,PCO2-28,HCO3-18.8 Respiratory alkalemia with compensatory metabolic acidosis with hypoxaemic respiratory failure
- Haemoglobin-10 gm/dl, hemogram-WNL, platelet count 80,000; INR 1.3, KFT & LFT-WNL
- Chest X ray- bilateral alveolar infiltrates
- Fetal evaluation showed Type 3 decelerations On CTG

A diagnosis of acute pulmonary edema with preeclampsia with FGR with fetal distress was made

Patient shifted to the Operating room for LSCS with 2 PCV and 4 FFP, 4 PRP ready in blood bank. LSCS was performed under GA. Live infant weighing 2.2 kg was delivered and transferred to NICU. Post operatively, patient was shifted to obstetric ICU. She was maintained on positive pressure invasive ventilation on VCV mode with PEEP of 8 cm water. Post op fluids were administered @ 60ml/hr; Inj Lasix 20 mg was given I/V 12 hourly. Cardiac Echo with Doppler done on day 2 post surgery, it showed Grade II-III diastolic dysfunction. Hence, she was started on ACE inhibitors. She was extubated on day 2 and was discharged on day 10 with her baby with advice to follow in cardiology OPD.

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Thromboprophylaxis in Pregnancy

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Introduction

Venous thromboembolism (VTE) includes deep vein thrombosis (DVT) and pulmonary embolism (PE) as well as other more rare forms such as mesenteric vein thrombosis and intracranial venous thrombosis. The incidence of VTE ranges from 1 to 2 per 1000 and is 2 to 4 fold higher in pregnant women compared to non pregnant population[1]. The risk is increased as pregnancy advances and is greatest in the postpartum period. The prevailing belief that VTE in the Asian population is less than in the Western population has essentially been disproved Moreover the changing profile of Indian pregnant women being older, heavier, smokers, higher rate of pregnancy with previous caesarean as well as increase in number of IVF pregnancies has increased the risk of VTE in pregnant Indian women.

Venous thromboembolism is one of important cause of maternal morbidity and mortality worldwide. A systematic review performed by the World Health Organization implicated embolism in 14.9% of maternal deaths in high-resource countries. Indian studies of maternal mortality have also shown that pulmonary embolism is emerging as one of leading cause of maternal mortality.

Rationale of Thromboprophylaxis

Thromboprophylaxis has been identified as the most readily implementable means of reducing maternal mortality from thromboembolism and adoption of comprehensive thromboembolism prevention strategies have reduced death from this cause in western countries. All pregnant women do not require VTE prophylaxis, therefore identification of women at risk of pregnancy associated VTE and instituting appropriate prophylaxis in them is important[2].

Various societies all over the world have formulated guidelines based on risk factors for initiation of thromboprophylaxis but there is no uniformity in prophylaxis recommendations from them. Further there is inadequate evidence from randomized clinical trials on which to base management. This had led to confusion regarding administration of thrombo prophylaxis. A workgroup of the National Partnership for Maternal Safety (NPMS) of the Council on Patient Safety in Women's Health Care, representing all major women's health care professional organizations, has developed a thromboembolism safety bundle that both critically reviews current guidelines and research evidence and has made recommendations for prophylaxis in pregnancy[3].

Risk Assessment

All guidelines recommend that a comprehensive assessment of the pregnant women for risk of VTE should be done at four time points in pregnancy[4]:

1. First prenatal visit
2. At time of all antepartum admissions
3. Immediately postpartum during a hospitalization for childbirth
4. On discharge home after a birth

This risk assessment is done on the basis of woman's past and family history of VTE, congenital or acquired thrombophilia, pre-existing or current medical disorders, presence of obstetrical complications during pregnancy, transient risk factors in current pregnancy and personal history of woman[5].

High risk factors

- Recurrent unprovoked VTE
- VTE plus high risk thrombophilia
- VTE in current pregnancy
- Personal history of unprovoked VTE
- Personal history of VTE with hormonal risk factor
- Antithrombin deficiency and family history of VTE in a first-degree relative
- Homozygous Factor V Leiden
- more than one thrombophilia
- Ovarian hyperstimulation syndrome requiring admission (given up to 13 wks)

Intermediate Risk Factors

- Personal history of provoked VTE with non-hormonal risk factor
- Protein C defeciency
- Protein S deficiency
- Homozygous Prothrombin G20210A (PT) defeciency
- Acquired thrombophilia
- Non-obstetric major surgery during pregnancy
- Obesity (BMI ≥ 40kg/m2
- Current medical condition eg. heart or lung disease, SLE, cancer, nephrotic syndrome, sickle cell disease, pre-existing diabetes with vascular complication, systemic inflammatory condition
- Current sepsis (requiring IV antibiotics)

Cumulative Risk Factors

- Family history of unprovoked or estrogen-related VTE in a first-degree relative
- Known low-risk thrombophilia
- Age >35 years
- Obesity ≤40kg/m2
- Parity 3
- Smoker
- Gross varicose veins
- Preeclampsia in current pregnancy
- ART/IVF
- Multiple pregnancy
- Cesarean section, Midcavity or rotational operative delivery
- Prolonged labor (>24 h)
- PPH (>1 L or transfusion)
- Preterm birth
- Stillbirth in current pregnancy
- Current systemic infection
- Immobility and dehydration

Agents for Thromboprophylaxis

General measures- Maintaining hydration and avoiding prolonged immobilisation at any time in antenatal period, intrapartum and postpartum period

Mechanical- Mechanical methods are aimed at decreasing venous stasis. And have been found to reduce the risk of deep vein thrombosis by two-thirds in general surgical patients. There is little evidence for the efficacy of these methods in pregnancy because there are no large-scale studies Mechanical strategies to prevent VTE include-

- Graduated venous compression stockings
- Sequential compression devices (SCD)

Indications for mechanical methods are as follows:

- Contraindication to pharmacological thromboprophylaxis.
- SCD should be used after all emergency caesarean section or after elective caesarean with at least one risk factor.
- Graduated elastic compression stockings are used in previous VTE or thrombophilia throughout pregnancy & for 6–12 weeks after delivery In patients at increased risk of VTE.
- For pregnant women traveling by air.

Pharmacological thromboprophylaxis

Choice of pharmacological anticoagulant during pregnancy needs to take into account fetal safety and maternal peripartum issues (eg, unpredictable onset of labor, use of neuraxial anesthesia for management of labor pain). The goal of thromboprophylaxis is to administer a dose of medication that reduces VTE risk while minimizing the risk of bleeding. The options for pharmacological thrombo prophylaxis include-

A. Heparins- Low Molecular Weight Heparin (LMWH) and Unfractionated Heparin (UFH)
B. Warfarin
C. Others- Hepranoids, Fondaparinux, Argatroban, Danaparoid, Dabigatran, Rivaroxaban

A. **Heparins**

Heparins act indirectly by binding to antithrombin, which then inhibits thrombin, and inactivates factor Xa. Heparins are used for most pregnant women because they do not cross the placenta and do not anticoagulate the fetus and are safe in breast feeding. Both Unfractionated Heparin and Low Molecular Weight Heparin can be used.

1. **Unfractionated Heparin (UFH)**

   UFH is cheaper, has shorter half-life, can be reversed rapidly but it causes heparin induced thrombocytopenia(HIT)and bone loss, needs monitoring baseline platelet count, after 2-3

days, weekly x 2 weeks then monthly. UFH is sometimes preferred peripartum when increased risk of haemorrhage is there or where regional anesthesia may be required. In severe renal insufficiency (eg, creatinine clearance <30 mL/min) also UFH is preferred because LMWH metabolism is exclusively renal, while metabolism of UFH is both renal and hepatic. It can be given S/C or I/V.

2. **Low Molecular Weight Heparin (LMWH)**

   LMWH is effective, easy to administer S/C, has more predictable response and do not require routine monitoring Low molecular weight heparin is preferred for routine thromboprophylaxis except in renal disease. No monitoring is required but it's a good practice to repeat platelet count once after 3 to 4 weeks.

   Dosage- Heparins can be administered during pregnancy at different doses depending upon the risk of thromboembolism and desired degree of anticoagulation.[1]

   Prophylactic dose anticoagulation refers to the use of low doses of anticoagulants which aims to reduce the risk of thromboembolism while minimizing bleeding complications as shown in Table 1.

Table 1: Prophylactic doses of LMWH and UFH

Prophylactic dose S/C	Dalteparim	Enoxiparin	Heparin Sodium
	5000 units OD	40mg OD	5000 units BD

Intermediate dose anticoagulation refers to the adjustment of prophylactic dose anticoagulation according to body weight (Table 2).

Table 2: Dose of heparins according to body weight

Current weight (kg)	Daleparin	Enoxaparin	Heparin Sodium
Less than 50	2500 units OD	20 mg OD	-
50-90	5000 units OD	40 mg OD	5000 units BD
91-130	7500 units OD	60 mg OD	7500 units BD
131-170	10000 units OD	80 mg OD	
171ormore	75 units /kg / day	0.5 mg /kg / day	

Therapeutic dose anticoagulation refers to the use of anticoagulants at doses typically reserved for treatment of thromboembolic disease. Despite the nomenclature, therapeutic dosing may be used prophylactically in some patients at very high risk of thromboembolism.

-Pre-pregnancy therapeutic anticoagulation, recurrent unprovoked VTE (2 or more), VTE in current pregnancy, any previous VTE plus high risk thrombophilia, any previous VTE and acquired thrombophilia.

Table 3: Therapeutic dose of Heparins

Current weight (kg)	Daleparin	Enoxaparin	Heparin Sodium
Less than 50	2500 units BD	4omg OD	5000 units BD
50-130	5000 units BD	8omg OD	7500 units BD
91-130	7500 units BD	6omg BD	7500 units TDS

Contraindications to heparin

- Known hypersensitivity
- History of or current HIT
- Creatinine clearance less than 15 mL/minute associated with significant platelet dysfunction—seek expert advice before use Cautions
- Renal impairment (creatinine clearance less than 30 mL/minute) for LMWH
- Hepatic impairment
- Thrombocytopaenia (platelets less than 100 x 109/L or trending down)

Caution for using heparin if risk factors for bleeding present-

- Active antenatal or postpartum bleeding (requiring at least two units of blood or blood products to be transfused in 24 hours, or primary postpartum haemorrhage (PPH) greater than 1 L)
- Chronic, clinically significant and measurable bleeding over 48 hours
- Women at risk of major haemorrhage (e.g. placenta praevia)
- Acquired or inherited bleeding disorders (e.g. acute liver failure, Von Willebrand's disease)
- Recent central nervous system bleeding
- Intracranial or spinal lesion
- Abnormal blood coagulation
- Thrombocytopenia
- Severe platelet dysfunction (e.g. Bernard Soulier, Glanzmann's thrombasthenia)
- Antiplatelet drug use
- Active peptic ulcer or active ulcerative gastrointestinal disease
- Obstructive jaundice or cholestasis

- Recent major surgical procedure of high bleeding risk
- Concomitant use of medications that may affect the clotting process
- Neuraxial analgesia (epidural in labour ward) or anaesthesia (spinal or epidural for operative procedure) or diagnostic lumbar puncture

B. **Warfarin**

Warfarin is a Vit. K antagonist, it reduces the synthesis of active clotting factors by depleting the levels of functional vitamin K. Warfarin crosses the placenta and has been associated with fetal anomalies, such as midface hypoplasia, stippled chondral calcification, scoliosis, and short proximal limbs when exposure occurs in the first trimester Therefore it cannot be used in early pregnancy and because of its long half-life it cannot be used in late pregnancy. Its use is limited to mid pregnancy and requires close monitoring of APTT. As it crosses placenta, there is risk of bleeding in the fetus also. Use of warfarin later in gestation is associated with fetal intracranial hemorrhage and schizencephaly. Nowadays warfarin is not used for thromboprophylaxis except in women with mechanical heart valves.

C. **Other Anticoagulants**

Danaparoid- Danaparoid is a heparinoid that is mostly used in patients intolerant of heparin, either because of HIT or a skin allergy to heparins. The half-life of danaparoid is long (24 hours) and regional anaesthesia should be avoided in women receiving it for thromboprophylaxis. Although direct evidence is limited, breastfeeding should be safe while on danaparoid since little if any appears in breast milk and oral absorption is unlikely.

Rivaroxaban is a synthetic selective inhibitor of factor Xa, and has been used in patients who develop adverse skin reactions with the use of heparins It has to be withheld for 5 days prior to birth due to long half-life. A small observational study of 12 pregnancies in 10 women showed that fondaparinux did not cause hypersensitivity skin reactions and was not associated with bleeding complications to mother or fetus.

Apixaban, edoxaban, dabigatran have not been extensively studied in or approved for patients who are pregnant.

Antenatal Thromboprophylaxis: Indications and Duration

Extended– Started as soon as pregnancy diagnosed and continued throughout pregnancy

Any high risk factor present

- Two or more intermediate risk factors
- IVF pregnancy and three other risk factors
- Four or more cumulative risk factors

Extended- starting at 28 weeks.

- Patients with 3 cumulative risk factors

Short term

- Hyperemesis till it resolves.
- Ovarian hyperstimulation syndrome till first trimester
- Major surgery till patient is ambulatory
- Admitted and bed ridden for 72 hours, thromboprophylaxis till patient becomes mobile

Thromboprophylaxis Around Delivery

The half-life of UFH is about 1.5 hours, whereas the half-life of LMWH is approximately 4 hours. The risk of bleeding around the time of delivery and limitations in predicting the onset of labor makes management challenging there are 2 common approaches to managing the use of thromboprophylaxis in this time period.

- Transition from LMWH to UFH before delivery, with the patient being instructed to stop UFH when signs of labor occur.
- Stopping LWMH 24 hours before the scheduled induction of labor.

Postpartum Thromboprophylaxis

The threshold for anticoagulation is lowered in the postpartum setting largely because the risk of VTE is increased, and the potential for the more serious adverse effects of anticoagulation, including placental haemorrhage, spinal hematoma and fetal haemorrhage are also not there. Compared with the antepartum period, VTE, especially pulmonary embolism, is two to five times more common in the puerperium with persistent risk for six weeks beyond delivery.

Additional risk factors develop during and after intrapartum period and every woman should have

a repeat VTE risk assessment after delivery and the need and duration of thromboprophylaxis decided.

Prolonged postpartum thromboprophylaxis

Pospartum thromboprophylaxis for 6 weeks should be given to:

- All women who received antenatal LMWH thromboprophylaxis
- Women with positive family history of VTE along with documented thrombophilia, or presence of other risk factors
- Women with diagnosed high risk thrombophilia
- Complete immobilisation for ≥3 days

Short term postpartum thromboprophylaxis upto 10 days

Short term thromboprophylaxis should be given till mobilization to women:

- Delivered by emergency caesarean
- Elective caesarean and presence of another risk factor
- Prolonged admission for ≥3 days or readmission after delivery for≥3 days
- If ≥ 2 risk factors (Antenatal and postnatal combined)are present

Neuroaxial anesthesia and thromboprophylaxis

The time between starting neuroaxial blockade and restarting thromboprophylaxis depends on the type and dosage regimen as shown in Table 4.

Table 4: Neuroaxial block and thromboprophylaxis

Regime	Hours for inserting catheter or giving spinal	Postpartum
Prophylactic LMWH	12 hours	12 hour after block and 4 hours after removal
Therapeutic LMWH	24 hours	24 hour after block and 4 hours after removal
Prophylactic UFH S/C	24hours	1 hour after block and 1 hour after removal
Prophylactic UFH infusion	4 hour	1 hour after block and 1 hour after removal

Note: For UFH it is advisable to do aPTT after 3-4 hours

Thromboprophylaxis for Pregnancy with COVID 19

Guidelines for thromboprophylaxis in pregnancy with COVID 19 infection issued by North America Anticoagulation forum and RCOG in 2020 have given following recommendations[6]:

- All pregnant women admitted with confirmed or suspected COVID-19 should receive prophylactic LMWH, unless birth is expected within 12 hours
- During a period of self-isolation, a clinical VTE risk assessment (in person or remotely) should be performed, and thromboprophylaxis considered and prescribed on a case-by-case basis
- Thromboprophylaxis commenced for pregnant women who are self-isolating should continue until they have recovered from the acute illness (between 7 and 14 days).
- Women already prescribed thromboprophylaxis should continue administering it.
- All hospitalised pregnant women with confirmed COVID-19 should receive thromboprophylaxis for 10 days following hospital discharge. For women with persistent morbidity, longer duration of thromboprophylaxis is considered.
- Confirmed or suspected COVID-19 in women within 6 weeks postpartum should receive thromboprophylaxis for the duration of their admission and for at least 10 days post discharge. Consider extending this until 6 weeks postpartum for women with significant ongoing morbidity.

Conclusion

VTE is a significant contributor to maternal morbidity and mortality. Strategies to decrease the incidence of VTE in pregnancy include using mechanical and pharmacologic thromboprophylaxis for women at highest risk. Identifying patients who may benefit from thromboprophylaxis involves thinking critically about each patient's risk factors in the antepartum period, during hospital admission, at the time of caesarean delivery, and in the postpartum period. The indications of thromboprohylaxis and duration to be given are summarized in Table 5.

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Table 5: Summary of Thromboprophylaxis in Pregnancy

Condition	Method	Time of starting	Postpartum
Pre-pregnancy therapeutic anticoagulation	Heparin therapeutic dose	Antenatal	6 weeks
Any previous VTE plus high risk thrombophilia			
Any previous VTE and acquired thrombophelia			
Recurrent unprovoked VTE (2 or more)			
VTE in current pregnancy			
Any single previous VTE not provoked by surgery	Heparin Standard Prophylaxis	Surveillance & Start at 28 weeks	6 weeks
Recurrent provoked VTE			
Active autoimmune or inflammatory disorder			
Medical co-morbidity: (e.g. cancer, nephrotic syndrome, heart failure, sickle cell, type I diabetes with nephropathy			
Presence of 3 cumulative risk factors			
Any single previous VTE with low risk thrombophelia	Heparin Standard Prophylaxis	Surveillance	6 weeks
VTE unprovoked			6 weeks
Antenatal hospital admission>3days	Heparin standard prophylaxis	Till patient is not mobile	As per risk factor postpartum
Any surgery (pregnancy or postpartum)			
Severe hyperemesis or dehydration requiring IV fluid			
Ovarian hyperstimulation syndrome (first trimester only	Heparin standard prophylaxis	First Trimester	

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Answer: September 2020 Issue

Crossword

1. Nsaid
2. Sepsis
3. Physiological
4. Ventilatory
5. Cardiogenic
6. Four
7. Blynch
8. Preeclampsia
9. Six
10. Fondaparinux

Pictorial Quiz Answers

Q1. What is the most likely clinicla diagnosis?
Ans. Clinical Diagnosis Diabetic Ketoacidosis blood sugar 632 mg/dl

Q2. What acid-base disorder is present?
Ans. Acid Base Disorder Severe High Anion Gap Metabolic Acidosis

Q3. What other test would you perform on this patient?
Ans. Additional tests Urine ketones, HbA1C

Journal Scan

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This paper is still in the pre-print stage but has undergone a review and been accepted by the American Journal of Obstetrics and Gynaecology. The reason why this has been brought out here even before it could be indexed is to be able to update ourselves on the continuously evolving pandemic situation with respect to pregnancy and its outcomes with COVID-19. It also employs a propensity matched case-control methodology to overcome the effect of known confounders, is multicentric and involves a relatively large cohort as compared to the papers published thus far on this subject. Its findings that COVID-19 positive PREGNANT women are likely to have worse outcomes than COVID-19 positive NON-PREGNANT women is in contrast to the prevailing knowledge and hence worth a read. The entire paper has been brought out for your consumption.

Are Clinical Outcomes Worse for Pregnant Women ≥ 20 Weeks' Gestation Infected with COVID-19? A Multicenter Case-control Study with Propensity Score Matching

Badr DA, Mattern J, Carlin A, Cordier A-G, Maillart E, El Hachem L, El Kenz H, Andronikof M, De Bels D, Damoisel C, Preseau T, Vignes D, Cannie MM, Vauloup-Fellous C, Fils J-F, Benachi A, Jani JC, Vivanti AJ

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Introduction

The first cases of a novel coronavirus (SARS-CoV-2/COVID-19) were reported in Wuhan in December 2019[1]. Over 12.1 million people have been infected with over 550,000 deaths. These cases include an increasing number of pregnant women, however we are still relatively early in our understanding of the severity of the disease on pregnancy and vice versa. Early reports focused solely on the fetal risks, however, the emphasis has correctly shifted towards maternal health[2-6]. A recent study reported a hospitalization rate of 52% including 10% in intensive care unit (ICU)[5]. Nevertheless, the available literature is somewhat conflicting with some studies suggesting that pregnancy is not associated with markers of disease severity and others reporting worse outcomes. This contradiction implies the need for larger and more methodologically robust matched case-control studies to clarify the association between pregnancy and COVID-19. The objective of our study was to compare clinical outcomes and laboratory findings in infected pregnant women ≥ 20 weeks with a cohort of non-pregnant COVID-19 positive women after closely matching the two groups using a propensity score.

Methods

Study design: This was a retrospective study conducted in 4 large university hospitals in France and Belgium between 1st January 2020 and 13th May 2020. Inclusion criteria were: female patients of reproductive age with positive SARS-CoV2 RT-PCR tests in nasopharyngeal swabs samples. Included patients were then divided into two groups: Group 1, non-pregnant controls and Group 2, pregnant cases. The primary outcome was admission to ICU. The secondary outcomes included: hospitalization for clinical deterioration, need for supplemental oxygen therapy (OT) and endotracheal intubation (ETI).

Data collection: The following variables were analyzed: patient age, ethnicity, weight, height, body mass index (BMI), pre-existing medical conditions (diabetes mellitus type I and II, hypertension and asthma), symptoms, physical examination, pregnancy status and gestational age at the initial presentation. Laboratory tests analyzed included: hemoglobin, white blood cell count (WBC), platelet count, absolute neutrophil and lymphocyte counts, liver function tests (alanine transaminase (ALT), aspartate transaminase (AST)), lactate dehydrogenase, fibrinogen, D-dimers coagulation tests. All data were anonymized.

Outcomes and variables definitions: Hospitalization for clinical deterioration was defined as an admission to a regular care facility, a dedicated COVID-19 ward or an ICU due to complications directly related to a confirmed COVID-19 infection.

Statistical analysis: Data were analyzed with the statistical software packages SPSS 25 statistical software (IBM 91 SPSS statistics), R version 3.6.2 (R Core Team, 2019), and Excel version 15.0 (Microsoft, Redmond, WA, USA). We used the Fisher's exact test to compare the proportions of binomial categorical variables. After checking the normal distribution of continuous variables, we used the Student's T-test or the Mann-Whitney U test to compare their means in the 2 groups of the study. We undertook a propensity score analysis to match women between both groups. The CBPS R package and survey R packages were used to determine the propensity score as previously described. A two-sided $p< 0.05$ was considered significant.

Results

A total of 201 patients met the inclusion criteria. Eleven patients were excluded from the study: six non-pregnant patients (four receiving hemodialysis, one patient affected by trisomy 21, one patient with complex congenital heart disease) and five pregnant patients (all < 20 weeks gestation). This left 190 eligible patients for the final analysis who were 106 divided into two groups: a non-pregnant control group 1 (N=107) and the pregnant case 107 group 2 (N=83). Table 1 demonstrates the propensity score matching for a variety of pre-defined variables. The first part of the table (before matching) indicates that in almost all cases, the two groups had different means/proportions for the different variables before 20 weeks gestation) before matching was applied. The mean age in control group was significantly higher than that in case group (36.46 ± 6.89 years versus 31.97 ± 6.24 years; $p<0.0001$) but no statistically significant differences were observed for BMI or comorbidities between the two groups. The second part of the table presents the results after matching where we observe that the means, standard deviations and the proportions are now much closer between the two groups. The absolute standardized difference values (ASD), are equal to 0, indicating that the two groups now had similar means/proportions for the different variables after matching was applied. Based on this matching table, we consider the non-pregnant and pregnant groups similar on covariates chosen for the propensity score.

Table 1: Propensity score matching for age, body mass index, and comorbidities in case and control groups.

	Before Matching				After Matching		
	Control group 1 N = 107	Case group 2 N = 83	ASD	p-value	Control group 1 N = 107	Case group 2 N = 83	ASD
Age (years)	36.46 ± 6.89	31.97 ± 6.24	68.26%	0.001	34.17 ± 7.37	34.17 ± 6.49	0.00%
DM (type I or II)	4.67%	4.82%	0.69%	1.000	4.24%	4.24%	0.00%
Hypertension	7.48%	4.82%	11.08%	0.556	5.6%	5.6%	0.00%
Asthma	10.28%	8.43%	6.34%	0.804	8.34%	8.34%	0.00%
BMI (kg/m2)	28.25 ± 6.3	27.97 ± 6.41	4.4%	0.752	28.02 ± 6.25	28.02 ± 6.63	0.00%

Abbreviations: ASD: Absolute Standardized DIfference; BMI: body mass index; DM: diabetes mellitus.

Symptoms and laboratory tests at presentation: Table 2 displays the differences between the control and case groups in relation to symptoms and laboratory tests at presentation. The incidences of fever and cough did not differ significantly between the groups (57.8% versus 60.6%; p=0.765, and 78.3% versus 73.1%; p=0.495, respectively). Nevertheless, dyspnea, anosmia/ageusia, fatigue/myalgia, upper respiratory tract symptoms, gastrointestinal symptoms, and other symptoms, such as headache, chest discomfort, and cutaneous rash were all significantly lower in pregnant women. Moreover, there was significant difference of hemoglobin level, AST, ALT, CRP, creatinine and D-Dimers between the 2 groups. Other laboratory tests were similar in both groups.

COVID-19 severity among pregnant and non-pregnant women: Table 3 demonstrates the comparison of primary and secondary outcomes between both groups of the study after applying the propensity

score matching and performing a series of logistic regressions. Pregnant women were at higher risk for ICU admission than non pregnant women (11.08% versus 2.38%; p= 0.024). In addition, they were also at higher risk for hospital admission because of COVID-19 respiratory decompensation such as dyspnea and hypoxemia (58.21% versus 17.4%; p<0.001), for the need for OT (36.04% versus 17.24%; p=0.006), and for ETI (10.16% versus 1.67%; p=0.022). However, there were no cases of mortality in either of the 2 groups.

Table 2: Comparison of symptoms and laboratory tests at presentation between the 2 groups.

	Control group 1 N = 107	**Case group 2 N = 83**	**p-value**
Symptoms at presentation			
Fever	63 (60.6%)	48 (57.8%)	0.765
Cough	76 (73.1%)	65 (78.3%)	0.495
Dyspnea	46 (44.7%)	25 (30.1%)	0.049
Anosmia/ageusia	36 (34.6%)	15 (18.1%)	0.013
Fatigue/myalgia	70 (67.3%)	26 (31.3%)	<0.001
URT symptoms (runny nose, blocked nose, sore throat)	41 (39.4%)	9 (10.8%)	<0.001
Gastrointestinal symtoms (diarrhea, abdominal pain, nausea, vomiting	22 (21.2%)	8 (9.6%)	0.044
Others (headache, chest discomfort, cutaneous rash)	44 (42.3%)	10 (12%)	<0.001
Laboratory tests			
hemoglobin g/dL	12.98 ± 1.69	11.23 ± 1.32	<0.001
Platelets count, x10*9/L	236.91 ± 123.39	228.97 ± 92.55	0.896
White blood count, x10*9/L	6.93 ± 4.55	7.49 ± 3.38	0.066
Lymphocytes count, 10*9/L	1.45 ± 0.81	1.17 ± 0.51	0.116
Lymphocytopenia	13 (29.5%)	31 (45.6%)	0.114
Neurophils count, 10*9/L	4.74 ± 3.97	3.84 ± 3.26	0.876
Prothrombin time activity, %	97.46 ± 13.55	102.4 ± 11.28	0.160
aPTT ratio	105 ± 0.18	1.08 ± 0.22	0.131
Abnormal aPTT	5 (13.5%)	19 (13.1%)	0.066
Fibrinogen, mg/dL	513.25 ± 135.07	488.56 ± 133.43	0.339
AST, IU/L	47.97 ±36.6	35.49 ± 23.85	0.004
ALT, IU/L	45.5 ± 40.44	27.84 ± 30.51	<0.001
CRP, mg/dL	73.5 ± 78.23	34.17 ± 37.1	0.014
Creatinine, mg/L	0.69 ± 0.16	0.61 ± 0.41	<0.001
LDH, IU/L	320.08 ± 119.48	246 ± 4.58	0.396
D-dimer, ng/mL	781.5 ± 508.58	1112 ± 388.69	0.046

Abbreviations: ALT: alanine transminase; aPTT: activated partial thromboplastin time; AST: aspartate transminase; CRP: C-reactive protein; dL: deciliter; g: gram; IU: International unit; L: liter; LDH: lactate dehydrogenase; mg: milligram, ng: nanogram; URT: upper respiratory tract.

Table 3: Comparison of primary and secondary outcomes between the 2 groups after applying the propensity score matching.

Variable	**Control group 1 N = 107**	**Case group 2 N = 83**	**Adjustable p-value**
Primary outcome			
ICU admission	2.38%	11.08%	0.024
Secondary outcomes			
Hospital admission for COVID-19	17.4%	58.21%	<0.001
Need for oxygen therapy	17.24%	36.04%	0.006
Endotracheal intubation	1.67%	10.16%	0.022

Abbreviations: COVIDD-19: coronavirus disease 2019; ICU: intensive care unit.

Discussion

Main finding: Our propensity score-matched case control study has demonstrated that pregnant women infected with COVID-19 ≥ 20 week's gestation, have more severe outcomes than their non pregnant counterparts.

Comparison with literature: A small number of case-control studies have been published but few of those have attempted to match cases against the controls for a variety of parameters and/or demographic features. Liu et al (2) observed that the pregnant women had less fever at presentation, higher WBC counts and more consolidation on CT-chest scans. Blitz et al (3) described that among hospitalized women who are infected with COVID-19, pregnant women are not at increased risk for ICU admission. Qiancheng et al (4) showed that pregnancy was not associated with increased severity of the disease, shorter virus clearance time, or longer hospital stay after comparing 28 cases to 54 controls. On the contrary, significant maternal mortality has also been documented in a cohort of patients from Iran (6). These studies demonstrate not only the difficulties in determining the absolute risk of clinical deterioration specifically related to pregnancy but also the importance of correct case and control group matching. In our study, we showed that pregnant women had higher rates of ICU admission, need for supplemental OT and ETI compared to non-pregnant 160 women.

Strengths and limitations: This is the first multicenter case-control study of COVID-19 in pregnancy using a propensity score. We have included a relatively high number of pregnant women in the study almost matching the number of available controls, lending more validity to the strength of our findings. However, as with all retrospective designs there are certain limitations. These include missing data for laboratory examinations, making it difficult to evaluate more deeply the differences between the pregnant and non-pregnant populations. One relevant criticism could be that the threshold for diagnostic evaluation, hospitalization and certain treatments, may in fact be lower for pregnant women than for others, which may bias our finding of increased disease severity in this group. However, the participating centers involved did not drastically alter their management of COVID-19 patients on the basis of pregnancy, except in cases of deterioration during the third trimester, where emergency delivery was sometimes needed to alleviate the additional physiological demands of pregnancy (data not shown in this study).

Conclusion

On the basis of this study and that of some other groups (1-6), we advise clinicians to exercise prudence when planning the management of pregnant women with COVID-19 infections, particularly in the latter half of the pregnancy, when maternal risk of clinical decompensation and complications may be higher.

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Proceedings of AOGD Monthly Clinical Meeting held at LHMC, New Delhi on 14th August, 2020

Dynamic Triaging to Prevent The Admission of COVID Positive Obstetric Patients in Green Zones: A Quality Improvement(QI) initiative

Manju Puri
(on behalf of the Department of Obstetrics & Gynaecology, LHMC)

Background

A single undiagnosed COVID-19 positive patient admitted in the green zone has the potential to infect many HCWs(health care workers) and other patients at any given time with resultant spread of infection and reduction in the available workforce.

Problem

Despite the existing triaging strategy at the Department of Obstetrics & Gynecology of a tertiary hospital in New Delhi, where all COVID suspects obstetric patients were tested and admitted in Orange zone and Non-suspects in Green zone, asymptomatic COVID positive patients were found admitted in the green zone. Universal screening for all was not possible with given resources and holding all patients in orange areas pending reports was not ethical. This was the trigger to undertake a quality improvement (QI) initiative to prevent the admission of asymptomatic COVID positive patients in green zones.

Aim

The QI project aimed at reducing the admission of COVID-19 positive patients in the green zone of the hospital from 20% to 10% in 4 weeks' time starting 13/6/2020 by means of dynamic triaging.

Method

A COVID-19 action team was made and after an initial analysis of the problem multiple Plan-Do-Study-Act (PDSA) cycles were run to test the change ideas. The main change ideas included revised testing strategies like decreasing threshold for admission in orange zones, re screening with temperature at green zone entry, hold non urgent admissions till test report, test patients admitted in green zones were tested with TruNaat with shorter turnaround time and creating Grey Zones near green zones for green patients awaiting COVID-19 screening test results.

Result

The admission of unsuspected COVID-19 positive cases in the green zone of the hospital reduced from 20% to 0% during the stipulated period. There was a significant reduction in the number of HCWs, posted in the green zone, being quarantined or test positive for COVID-19 infection as well.

Conclusion

Quality Improvement methods have the potential to develop effective strategies to prevent spread of the deadly Corona virus.

Dull Aching Postpartum Pain: Just an ache or a red flag?

Aishwarya Kapur, Kiran Aggarwal, Khushbu Kumari, Aprajita Gupta

Abdominal pain is a complaint in postpartum period which may be due to afterpains, endometritis, urinary tract infection etc. We discuss a case of persistent abdominal pain in a postpartum woman due to a rather potentially life-threatening condition. The patient was a 35years old G7P3L3A3 at 27 weeks gestation with PTPROM with fever with chorioamniotis. Broad spectrum antibiotics were given and labour was induced, she delivered uneventfully but continued to run fever in postpartum period. Antibiotics were continued and she became afebrile. After 2 days of being afebrile she developed dull aching left flank pain. She was being managed symptomatically and was evaluated for renal pathology in view of left hydroureteronephrosis on USG. A CT urography done to rule out renal pathology due to persistent pain revealed left ovarian vein thrombosis.

She was treated with therapeutic anticoagulation and was discharged in stable condition with advice to continue Tab Warfarin for 6 months.

Postpartum Ovarian Vein Thrombosis (POVT) is a rare condition, incidence varying from 1 in 2000 to 1 in 5800 vaginal deliveries. It is more common on right side due to longer vein and more susceptibility to compression by dextro rotated gravid uterus. It has high incidence of serious complications like pulmonary thromboembolism and sepsis. High index of suspicion is needed to diagnose POVT in a postpartum patient with mild lower abdominal pain. These patients are at high risk and thromboprophylaxis is recommended in antenatal period and six weeks postpartum.

This case was presented so that unexplained fever, persistent unexplained abdominal pain should merit a differential diagnosis of ovarian vein thrombosis. This condition if diagnosed in time may save a life.

Managing Severe Ovarian Hyperstimulation Syndrome at a Non-IVF Facility.

Reena Yadav, Kanika Chopra

Introduction

OHSS is a serious complication of artificial reproductive technology. Incidence is on its rise because of increase in the number of IVF cycles. We present here a case of severe OHSS which was successfully managed at our hospital.

Case

A, 28-year-old, P4L5, professional oocyte donor, presented in the causality of department of Obstetrics and Gynaecology of our hospital accompanied by an agent from neighbouring state after oocyte donation 3 days back. She came with complaints of abdominal distension and pain for 3 days. She also had 8-10 episodes of vomiting and was unable to tolerate orally. On enquiring telephonically from the doctor regarding the ovulation induction regimen used, it was found that total dose of recombinant FSH used was around 2000 IU from day 3 for 5days, injection HMG used was 2500 IU with fixed dose of injection Cetrorelix of 0.25mg from 6th day to 12th day. Injection HCG, 10,000IU was used to trigger ovulation. On examination, her vitals were pulse rate 104 per minute, blood pressure 100/60 mmHg, respiratory rate 37/ minute. Chest examination revealed decreased air entry on right basal lung. Abdomen was distended, with shifting dullness and tenderness present in lower abdomen. Her abdominal girth was 35 inches. All her investigations were done, including hemogram, haematocrit, coagulation profile, ABG, liver, kidney function tests and serum estradiol levels along with chest XRAY and transabdominal ultrasound. Chest XRAY showed right sided pleural effusion. Ultrasound pelvis showed bilateral adnexa with complex cystic lesions of size 33cc and 75.8cc. Moderate ascites was seen with no internal echoes. Patient was given cabergoline 1 mg/day and thromboprophylaxis with injection enoxaparin 40mg subcutaneously. Patient's condition deteriorated on day 4 of presentation. She had multiple episodes of vomiting, her urine output decreased, her abdomen became distended, abdominal girth increased to 38.5 inches and she developed respiratory distress. In blood investigations, her hematocrit and serum potassium increased. Serum sodium and serum albumin decreased. Ultrasound pelvis revealed increased size of bilateral adnexal masses. Ultrasound guided paracentesis was decided and 800cc of ascitic fluid was drained and injection Cetrorelix 0.25 mg subcutaneously was administered along with intravenous albumin infusion. On sixth day, patient again had breathlessness due to abdominal distension, ultrasound guided paracentesis was repeated and 1.5 litres of ascitic fluid was drained. Injection cetrorelix 0.25mg with cabergoline was continued. Size of ovaries gradually decreased after 13 days of oocyte pick-up i.e. 10th day of admission and patient started menstruating and was discharged in a stable condition on 11th day with markedly improved biochemical parameters.

Discussion

Risk factors leading to increase in the possibility of developing OHSS in our case were young age, multiple follicles more than 20, high serum estradiol levels, previous history of OHSS and high doses of exogenous gonadotropins. Dopamine agonist, cabergoline decreases VEGF mediated capillary permeability by inhibiting phosphorylation of VEGF-2R. Initially dose of 0.5mg/day was found to be effective starting from the day of ovulation trigger. Later, Baris Ata et al, reported that the dose of 0.5 mg/ day was not effective enough to prevent the development of OHSS or resolve it and thus they used 1mg/day. GnRH antagonist work by causing sudden pituitary

dysfunction leading to ovarian regression and thus symptom relief. Lainas et al were the first to report use of GnRH antagonist for 7 days in 3 patients, that successfully recovered from severe OHSS. We used both cabergoline in high dose and GnRH antagonist in our case. Our patient responded to supportive management along with GnRH antagonist.

Conclusion

High suspicion, early diagnosis, supportive therapy and intensive monitoring are the key to successful management of patients presenting with severe OHSS. Available guidelines on management of OHSS do not mention use of Dopamine agonist and GnRH antagonist for the management of OHSS. A number of case reports published has shown effectiveness of these drugs. These agents should be considered in severe OHSS where patient is not responding to supportive management. There is an urgent need for rules and regulations for ART services. The availability of pre-structured counselling for oocyte donors with plan of management in case of complications is must.

Fetal hydrops: Light at the end of the tunnel

Manisha Kumar, Kanika Chopra, Reena, Manju Puri

Introduction

Non immune hydrops has high mortality but chylothorax has a better prognosis[1]. We present the case of hydrops fetalis in which there was a high index of suspicion of chylothorax and in spite of associated poor prognostic factors, the outcome was successful.

Case

A 26-year-old, primigravida was referred at 33 weeks 3 days with antenatal ultrasound showing polyhydramnios with bilateral fetal pleural effusion which was more on right side, there was associated mild ascites and subcutaneous edema. Her antenatal ultrasound at 18 weeks was normal. In the investigations, the indirect Coomb's test was negative, suggesting non immune hydrops. The TORCH serology, HbA1c, fetal echocardiography, middle cerebral artery peak systolic velocity were all normal. There was a suspicion of chylothorax as pleural effusion was more than ascites and right pleural effusion was more than left, therefore thoracocentesis was performed, 130 cc of fluid was drained. Pleural fluid cytology report showed 95% lymphocyte, hence suggested presence of chylothorax, the karyotype was normal.

Patient was kept under observation, within the next week the fetal ascites and subcutaneous edema decreased but there was re-accumulation of fluid in the pleural cavity. At 34 weeks 2 days thoracocentesis was done for the second time, 80 cc of fluid was drained . Within the next week aspiration was done for third time. At 37 weeks, labour was induced after pre- delivery tapping the pleural fluid for the fourth time. A 2.5 Kg, boy baby delivered vaginally, the baby showed signs of respiratory distress. After resuscitation and insertion of intercostal drain the condition improved and became stable. The baby's repeat chest X ray was normal the intercoastal drain was removed on the tenth day. There was no re-accumulation of pleural fluid , baby was discharged in a stable condition on day 19 of birth.

Discussion

Chylothorax is defined as accumulation of lymph in the pleural cavity, it is usually due to anomaly in lymphatic development, the incidence is 1 in 15,000 pregnancies[2]. Right side is more frequently involved as thoracic duct lies largely in right mediastinum. Perinatal mortality is 30-70%[2]. Thoraco-amniotic shunting is the best option. Pleurodesis with OK 432 has been tried. The factors associated with poor outcome after intervention are early gestational age of diagnosis, bilateral pleural effusion, presence of hydrops associated polyhydramnios[3]. The prenatal factors associated with poor neonatal survival are re-appearance of effusion, premature delivery and the persistence of hydrops at birth and the absence of thoracocentesis procedure.[3]

In spite of the bilateral and recurring pleural effusion and associated hydrops, the outcome in the present case was successful, and there was light at the end of the tunnel.

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Proceedings of AOGD Monthly Clinical Meeting held at Army Hospital, New Delhi on 28th August, 2020

Pregnancy after Medical Management of Carcinoma Endometrium - A journey from despair to hope

Col MK Tangri, Surg Cdr Hrishikesh Magdum

A 36 yrs old nulligravida, married for 7yrs, cohabitated for 04 yrs, presented with complaints of inability to conceive since 4 years and AUB since 6 months duration. The patient had taken 2 cycles of ovulation induction and timed intercourse (OI with TI) with Clomiphene Citrate for infertility. Her GPE, systemic and gynae examination was unequivocal.

On USG, Endometrial thickness was 14 mm. Endometrial biopsy revealed well differentiated endometroid adenocarcinoma, ER & PR positive. No myometrial invasion/ lymph node Involvement on MRI. Male factor was normal. Patient wanted to conceive so was started on fertility preserving medical management with oral progestins (megestrol acetate), histopathology (HPE) negative result achieved after one year of medial management.

After two consecutive negative HPE reports first cycle of IVF-ET was performed with long protocol. Cycle was unsuccessful due to infection in all culture plates. Patient was revaluated & diagnosed to have developed recurrence before the second cycle of IVF- ET. She was restarted on fertility preserving medical management. After 9 months of treatment the histopathology report was negative again and second cycle of IVF-ICSI and frozen embryo transfer was done resulting in successful pregnancy. Antenatally pregnancy was uneventful. Patient was induced at term for vaginal delivery. However an emergency LSCS had to be done in view of foetal distress and also found to have placenta accrete per-operatively for which a peripartum hysterectomy with B/L salphingo- oophorectomy was performed.

Discussion

Total abdominal hysterectomy with B/L salphingo-oophorectomy is the management for stage 1A endometroid adeno carcinoma. Medical management can also be considered with oral progesterone therapy for fertility preservation. MRI is the preferred modality to assess for invasive disease. Follow up done by EB at 3-6 monthly interval. Complete response is seen in 70-80%, with recurrence risk in 24-41% of patients on medial management.

Pregnancy with Pulmonary Atresia with VSD with Eisenmengerized Collateral with Major Aortopulmonary Collateral Arteries System Moral Vs Medical Dilemma

Gargi Vikas Sharma, Ravi Ramamurthy, Shakti Prasad Panda

Survival till adulthood is rare in cases of Pulmonary Atresia with VSD with Major Aortopulmonary Collateral Arteries (MAPCA) and rarer still is successful pregnancy outcome. 28 year old Primigravida with Pulmonary Atresia with VSD with MAPCAs was admitted at 15 weeks gestation in NYHA class II. Patient was diagnosed during Preanesthetic evaluation for Diagnostic Hysterolaproscopy. MAPCAs were hypertensive, so, no corrective surgery could be carried out and only option for the patient was Heart Lung Transplant. On admission, oxygen saturation was only **74%** on room air with maximum of 86% with oxygen supplementation, Grade 2 clubbing and a continuous murmur heard over the back. 2 D echo suggested PA, VSD, absent native branch pulmonary arteries with MAPCAs supplying both lungs with no signs of RVF. Hb was 13.5g/dl and all antenatal investigations within normal limits.

A multidisciplinary team counseled the couple about the risks and complications to the mother and the fetus and the risk to the patient's life in continuing the pregnancy, in spite of repeated counseling sessions, the couple decided to continue the pregnancy. She was hospitalized under constant close surveillance in the Obstetric HDU till delivery, placed on continuous oxygen supplementation, thromboprophylaxis and transfusion of packed

red blood cells with a target Hb of 16g/dl. She required to be transfused PRBCs 6 times antenatally. An intense and strict fetal surveillance was performed. Steroid induction was done at 28 weeks anticipating preterm delivery. At 28 weeks 05 days gestation, she developed Gestational Hypertension with tachycardia and was started on labetalol to prevent MAPCA rupture. In view of persistent maternal tachycardia, hypoxaemia with imminent risk of MAPCA Rupture and right ventricular failure, a multidisciplinary medical board was held at 31 weeks 5 days and the decision was taken to deliver the patient. She underwent an Elective Caesarean Section under Combined low dose spinal anesthesia and epidural analgesia to deliver a live male neonate weighing 1.380 kg at 32 weeks 01 day. Intra-op patient was transfused 4 PRBCs along with Fresh frozen plasma.

Postoperatively, she was managed in ICU with labetalol and NTG infusions to maintain her blood pressure with continuous oxygen supplementation and six packed RBCs transfused to maintain her oxygen saturation and good biventricular function. 9th post-operative day she was shifted to HDU and was kept on oxygen supplementation and six more packed RBCs transfused. On 18th post-operative day she was weaned off oxygen with resultant SpO2 of 74% at room air. Patient was discharged after 05 months of hospitalization with intense monitoring and interventions with a baby weighing 1.935 kg with no neurological deficit. The patient is currently planned for Heart Lung Transplant in near future.

Navigating a Precious Twin Pregnancy through The Doldrums of Alloantibodies

Col (Prof) Reema Kumar Bhatt, Maj Pranjali Trainee

A primigravida post IVF with O-positive blood group and spouse being A positive had presented first time at 12 weeks POG. During evaluation she was found to have severe anaemia. Peripheral blood smear (PBS) picture showed microcytic hypochromic anaemia & iron studies reflected iron deficient anaemia (IDA) favoured by low serm iron and ferritin, raised TIBC, RDW & Mentzer's Index with normal HPLC. Evaluation of other causes of anaemia like Paroxsymal Nocturnal Hemoglobinuria, G6PD deficiency and immunological causes were ruled out. Ultrasound confirmed mild hepatosplenomegaly and short cervix. 2 Θ PBCs (packed red blood cells) were transfused to correct anaemia, however this was followed by worsening of clinical picture in terms of increasing easy fatiguability, pallor(Hb - 5.1 g/dl) with new onset of icterus, high LDH, increased polychromasia on PBS, unconjugated hyperbilirubinemia, reticulocytosis, spherocytosis and positive Coomb's test, with all pointers to hemolysis. Antibody typing with antigram revealed that the patient lacked antigens c, E, Kell, Duffy, Kidd and MNS system and had antibodies against them. More than 400 donors had to be matched to get compatible blood. She was managed with pulse steroid therapy, intravenous immunoglobulins and weekly erythropoietin. Genome analysis revealed homozygous deletion of beta globin gene. Fetuses were monitored for fetal anaemia by MCA-PSV weekly. At 32 weeks, Emergency Caesarean was done for preterm labour with first twin in breech where both twins were DCT positive and required phototherapy postnatally. In the postpartum period patients Hb fell to 6.1 g/dl, oral steroids were restarted and repeat Hb after 6 weeks was 12.9 g/dl. Thereafter, steroids were stopped. Final diagnosis was Thalassemia Intermedia with Alloimmune Haemolytic anaemia.

Discussion

HbA2 levels may be masked by coexisting iron deficiency and may be falsely negative. Mutation analysis clinched the diagnosis of thalassemia intermedia which explained her clinical, haematological and biochemical picture. There were multiple alloantibodies due to her previous blood transfusion which triggered an anamnestic reaction after blood transfusion in current pregnancy. She was a high responder in the background of thalassemia intermedia. Struggle was of finding the right match and tools in armamentarium which were IVIG and Steroids. Plasmapheresis could not be offered due to low Hb. Both fetuses underwent strict surveillance with MCA PSV weekly.

Conclusion

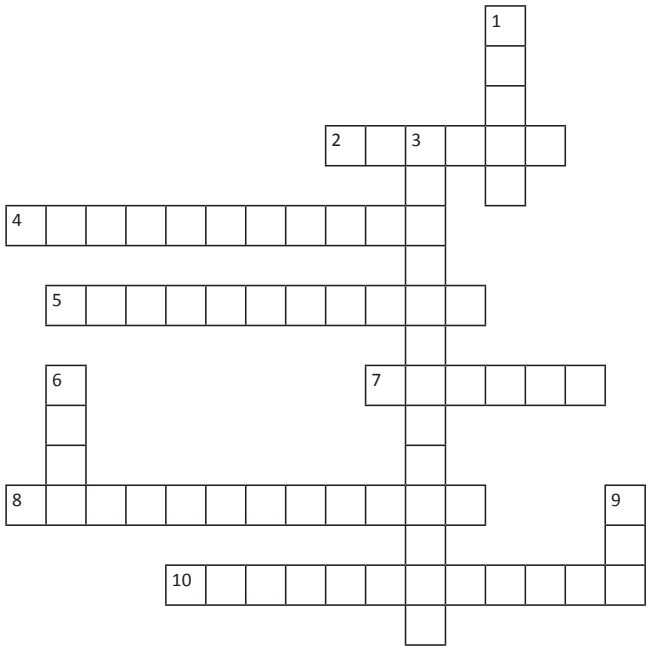
No transfusion is a safe transfusion. We should do ICT before each transfusion and blood crossmatched for minor antibodies should be administered.

Cross Word Puzzle

Ruma Satwik
Consultant, Centre of IVF and Human Reproduction, Sir Gangaram Hospital, New Delhi

CROSSWORD

Test your knowledge of Reproductive Anatomy and Physiology



Across

2. Life threatening organ dysfunction caused by dysregulated body's response to infection
4. A type of respiratory failure characterized by high PaCO2 and acidic pH
5. A type of shock caused by poor myocardial contractility
7. Uterine compresssion stitch for control of post partum haemorrhage
8. A major obstetric cause for acute pulmonary edema
10. Alternate anticoagulant in women with intolerance to LMWH

Down

1. Drug group to be avoided in women with OHSS
3. A cause of dyspnoea in pregnancy
6. Loading dose of Magnesium sulphate in grams in eclampsia as per the Collaborative Eclampsia trial regimen
9. Duration of Thromboprophylaxis in weeks in women with high risk thrombophilia and previous VTE episode

PICTORIAL QUIZ

Prakash Shastri
Senior Consultant, Institute of Critical Care Medicine, Sir Ganga Ram Hospital, New Delhi

A 22 year old female is brought to Emergency department complaining of nausea and abdominal pain.

He looks extremely unwell and appears confused.

The following blood tests are obtained:

Glucose 1mmol/L = 18 mg/dl

Arterial blood gas			Serum blochemistry		
Sample	**Ref range**		**Sample**	**Ref range**	
pH	7.13	(7.34 -7.45)	Sodium	136 mmol/L	(137-145)
pO2	85 mmHg	(80-95)	Potassium	6.4 mmol/L	(3.5-5.0)
pCO2	16 mmHg	(35-45)	Chloride	92 mmol/L	(98-106)
HCO3-	5.2 mmol/L	(22-26)	Glucose	39 mmol/L	(3.5-6.0)

Q1. What is the most likely clinicla diagnosis?

Q2. What acid-base disorder is present?

Q3. What other test would you perform on this patient?

Answer to August Crossword and Pictorial Quiz given on Page No. 38

AOGD Events Held

- On 15th August 2020 - A webinar on **Public Awareness Campaign** moderated by Dr. Richa Sharma and Dr. Seema Prakash.
- On 16th August 2020 - A webinar on **Gynae Surgical Skills Enhancement Series-3** moderated by Dr. Sudesh Agarwal and Dr. Richa Sharma.
- On 16th August 2020 - A Virtual CME on **Current Updates on Contraception by** Dr. Sumita Mehta.
- On 17th August 2020 - A webinar on **Medico-legal Aspects of Obstetrics and Gynecology** by FOGsd.
- On 17th August 2020 - A Webinar on **Tutorials on Laparoscopic Cystectomy by** Dr. Richa Sharma.
- On 18th August 2020 - A webinar on **Paradigm Shift in Management of Endometriosis** and panel discussion on **Evidence based Solutions to Endometriosis** by Faridabad Obstetrics and Gynecology Society and AOGD.
- On 21st August 2020 - Panel discussion on **Pregnancy and Working Women: The Changing Dynamics, the Prejudices & the Bias** Organised by BLK Hospital and AOGD.
- On 22nd July 2020 - A webinar on **Re-energies, Re-imagine and Reaffirm in Challenging Times** by Dr. Malvika under the aegis of Delhi Gynae forum central, under the aegis of AOGD.
- On 23rd August 2020 - A webinar on **Recurrent Pregnancy Loss-How I Treat** by Dr. Kuldeep Jain.
- On 24th August 2020 - A webinar on **Preterm Birth Management Caesarian Myomectomy** by Dr. Anita Sabharwal.
- On 24th August 2020 - A webinar on **Endometriosis – Ending the Endless Enigma** by Infertility Committee AOGD, DGF Outer Delhi and Haryana Association of Obs. and Gynae.
- On 25th August 2020 - A webinar on **Menopause & Bone Health** moderated by Dr. Chitra Setya.
- On 26th August 2020 - A webinar on **HIV & AIDS A way forward for HIV free generation** by FOGSI and AOGD.
- On 26th August 2020 - A webinar on **Alloimmunization-Anticipate and Act** by AMOGS and Jharkhand Societies.
- On 27th August 2020 - A webinar on **Ovarian Rejuvenation & ART Pregnancies** moderated by Dr. Asha Baxi.
- On 28th August 2020 - **AOGD Virtual Monthly Clinical Meeting** organised by Army Hospital Research And Referral New Delhi, 04:00-05:00 pm.
- On 29th August 2020 - A webinar on **Pregnancy Management with Medical Condition** by AOGD**.**
- On 29th August 2020 - A webinar on **Challenges of Pre-Eclampsia** by Department of Obs. and Gynae UCMS and GTBH Delhi with Medical Education Committee, FOGSI and AOGD.
- On 30th August 2020 - A webinar on **Endometriosis, an Elusive Challenge** by Endometriosis Committee FOGSI and AOGD.
- On 5th September 2020 - A E-CME webinar on **Evidence Based Management of Maternal Sepsis** by Dr. (Lt. Col) Leena N Sreedhar and Dr. Shashi Lata Kabra.
- On 5th September 2020 - A webinar on **Luteal Phase Defect** and **Teacher's Day Celebration** by FOGsd.
- On 6th September 2020 - A webinar on **Masterclass on Fibroids** by Dr. Kavita Agarwal.

AOGD Forthcoming Events

- On 12th September 2020 - A webinar on **Management Approach in AUB (O & M)** by Department of Obs. and Gynae UCMS and GTBH Delhi with Medical Education Committee, FOGSI and AOGD.
- On 16th September 2020 - A webinar on **"Medical Disorders in Pregnancy"** by SFM Delhi Chapter.
- On 25th September 2020 - **AOGD Virtual Monthly Clinical Meeting** to be hosted by DDU Hospital, New Delhi, 04:00-05:00 pm.
- On 23rd October to 6th November 2020 - **AOGD Pre and Post Conference Event**
- On 30th October to 1st November 2020 - **42nd Annual Virtual AOGD Coference**

Abstract Submission Guidelines for Competition Papers

1. Last Date for Competition Paper & Abstract Submission is 15/09/2020
2. Candidates should be less than 30 years of age.
3. Place of study should not be mentioned anywhere in the main body of the manuscript.
4. Conference registration is mandatory.
5. Only members of AOGD are entitled for paper presentation (Proof of membership should be enclosed)
6. If not a life member or annual member of AOGD, they need to be a student to present. All abstracts entered by Students should be accompanied by a student certificate forwarded by their Head of the Department.
7. Competition papers may be submitted online through the conference website http://conference.aogd.org/
8. All further correspondence will be sent to the contact email entered for the designated abstract Presenter.
9. All the papers should be original manuscripts and not already published anywhere else.
10. Please follow the Submission guidelines given below.

Guidelines enclosed

1. Use Microsoft Word to create your manuscript
2. Title- To be entered first.
   a. Title will be in BOLD UPPERCASE CHARACTERS,
   b. Title should be concise and short.
3. Authors and Disclosures-
   a. The names of authors should follow immediately under the title in one line. Type initials and family name of authors in BLOCK letters and underline the presenter's name. DO NOT include degrees or professional designations.
   b. UP TO 12 Authors and corresponding contact details may be entered into the submission system
4. The names of institution, city and country should be in lower case, following immediately after the authors, on a different line.
5. Leave one line between the title/ authors/ institution block and the body of the abstract.
6. Body of abstract: approximately 500 words: Text should be in lower case, black only, Font: Times New Roman, Font size: 12.
7. Please use the required headings listed below to construct your abstract:
   a. Introduction: Describe the background supporting the relevance of the research question.
   b. Objective: State the purpose of the study or investigation.
   c. Methods: State details on study subjects, techniques, and/or observational/analytical methods.
   d. Results: Include your main findings, noting statistical data.
   e. Conclusions: Summarize principal conclusions, emphasizing new and important aspects.
8. Full text should follow the above structured abstract in 3000 – 4000 words.
9. All tables and graphs in the full text should be appropriately labelled & numbered.
10. References: As per the Vancouver style.
11. Use of standard abbreviations is desirable. Please write special or unusual abbreviation in brackets after the full word, the first time it appears. Use numerals to indicate numbers, except to begin sentences.
12. Do not include graphs and references in the abstract.
13. Use single-line vertical spacing and leave one line between paragraphs.
14. Submission should include all the details of your competition papers.
15. The entire paper will be reviewed and rated by scientific committee prior to final decision on acceptance.
16. Please use the online abstract submission portal to upload this word document. http://conference.aogd.org/
17. All the information required on the online abstract/paper submission form must be entered in various fields before uploading your word document.

Abstract Submission Guidelines for Free Papers

1. Abstract Submission Deadline is September 30, 2020.
2. Only registered delegates are entitled to present the selected posters/papers.
3. Abstracts may be submitted online through the conference website http://conference.aogd.org/
4. All further correspondence will be sent to the contact email entered for the designated abstract Presenter.
5. One must be life/annual member to present oral/poster in the conference.
6. If not a LIFE member of the above bodies, they need to be a student to present. All abstracts entered by Students should be accompanied by a student certificate forwarded by their Head of the Department.
7. Abstracts may be submitted on the following theme:
   a. High risk pregnancy
   b. Benign Gynaecological Conditions
   c. ART- Recent Advances
   d. Gynae-Oncology
   e. Miscellaneous
8. All Case Presentations to be kept for Poster Presentation.
9. Title- To be entered first.
   a. Title will be formatted in BOLD UPPERCASE CHARACTERS, should be concise and short.
10. Authors and Disclosures
    a. The names of authors should follow immediately under the title in one line. Type initials and family name of authors in BLOCK letters and underline the presenter's name. DO NOT include degrees or professional designations.
    b. UP TO 12 Authors and corresponding contact details may be entered into the submission system
11. The names of institution, city and country should be in lower case, following immediately after the authors, on a different line.
12. Leave one line between the title/ authors/ institution block and the body of the abstract.
13. Body of abstract: approximately 500 words: Text should be in lower case, black only, Font: Times New Roman, Font size: 12.
14. Please use the required headings listed below to construct your abstract:
    a. Introduction: Describe the background supporting the relevance of the research question
    b. Objective: State the purpose of the study or investigation.
    c. Methods: State details on study subjects, techniques, and/or observational/analytical methods.
    d. Results: Include your main findings, noting statistical data.
    e. Conclusions: Summarize principal conclusions, emphasizing new and important aspects.
15. Use of standard abbreviations is desirable. Please write special or unusual abbreviation in brackets after the full word, the first time it appears. Use numerals to indicate numbers, except in the beginning of sentences.
16. Do not include graphs and references in the abstract.
17. Use single-line vertical spacing and leave one line between paragraphs.

Please use the online abstract submission portal to upload this word document. http://conference.aogd.org/

All the information required on the online abstract submission form must be entered in various fields before uploading your word document.

Abstracts will be reviewed and rated by scientific committee prior to final decision on acceptance.

Decision for acceptance as oral presentation or poster presentation rests with the Scientific Committee.

This would be communicated to you approximately a week before the conference proceedings initiate.

List of Prizes

Competition Paper/ Free Paper/Poster/Slogan/Quiz
42nd Annual Conference of AOGD

Category	Award
Dr Neera Agarwal's Medal for Best Paper on **Theme Topic of High Risk Pregnancy**	Gold Medal
Dr Suneeta Mittal's Medal for Best Paper on **Theme Topic of Benign Gynaecological Conditions**	Gold Medal
Dr U P Jha & Raj Soni's Medal for Best Paper on **Theme Topic of ART- Recent Advances**	Gold Medal
Dr U P Jha & Dewan Balakram's Medal for Best Paper on **Theme Topic of Gynae - Oncology**	Gold Medal
Mr S Bhattacharya & Dr Ganguli's Medal for Best Paper on **Theme - Miscellaneous Category**	Gold Medal Silver Medal
Poster Presentation	Gold Medal Silver Medal
Slogan	First Prize Second Prize Third Prize
Research Paper- **Best Competition Paper**	Gold Medal Silver medal Bronze Medal
Dr Batra's Medal Winning Team of **AOGD Quiz**	Gold Medal
Dr S N Mukherjee **Rotating Trophy**	Best AOGD Monthly Clinical Meeting

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


First
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Theme: Women's Health Care In The Current Challenging Scenario
Super Speciality & Research Block
SIR GANGA RAM
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www.aogdvirtual.com

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 - 29th October, 2020
Pre Conference Workshops

30th October - 01st November, 2020
Scientific Programme

02nd November - 06th 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

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 - 06:00PM
Convenor:
Dr. Amita Suneja
GTB

Enhancing Surgical Skills In Gynae Endoscopy
27th October 2020
12:00PM - 06:15PM
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 - Basic 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 - 06:00PM
Convenor:
Dr. Shashilata Kabra
DDU
Tackling Unmet Need For FP Services In Times of COVID-19
3rd November 2020
10:00AM - 02:00PM
Convenor:
Dr. Mrinalini Mani
GGSGH
Critical Care Obstetrics
4th November 2020
10:00AM - 02:00PM
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 | 01:30 PM-06:00 PM

Time	Topic	Speaker
01:30PM-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 - 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 Chitra Raghunandan**	

Day 2, Saturday, 31st October, 2020 | 01:00 PM-06:00 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	**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**

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 Kuldeep Jain, 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 Ranjana Sharma, Dr S B Khanna, 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**	**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**	

Contact Us

AOGD Secretariat

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First E-Conference

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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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