ICOG CAMPUS

UNDER AEGIS OF INDIAN COLLEGE OF OBSTETRICIAN & GYNAECOLOGISTS

Theme : Severe Acute Maternal Morbidity

MDG MOMENTUM

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Joint Editor
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message

Dear Colleagues,

I wish happy new year to all of you.

I congratulate Dr. Mandakini Megh and Dr. Sadhana Gupta, chief editors on the publication of this E ICOG FOCUS, dedicated to a very essential and relevant theme: Severe Acute Maternal Morbidity.

Obstetrics in India has grown by leaps and bounds in the last few decades. Given the sheer volume of patients our consultants see on a daily basis, our experiences alone are enough to educate us on how to deal with the most trying of situations. Along with this, we have access to the latest technologies and methods that were lacking in the past. However, the maternal mortality and morbidity we face here in India is still high compare to developed world and not reached desirable.

Maternal mortality is just the tip of an iceberg and there is a vast unseen base which is known as near missed cases or severe acute maternal morbidity. SAMM cases are on the brink- these patients, if managed correctly and on time, can prevent a patient from becoming another statistic on the maternal Mortality list. Educating and updating our clinicians about these cases, giving guidance for their prevention and management, providing guidelines about when to treat and when to refer, will all go a long way in bringing down maternal morbidity in our country.

I wish this E-ICOG FOCUS a great success

Dr. Alpesh Gandhi
President FOGSI

message

To Fellow & Members of ICOG,

It is our pleasure to release ICOG campus on theme of Severe Acute Maternal Morbidity along with our dedicated team of editorial board. While in last 10 years we have been quite successful in reducing maternal mortality, we as obstetrician and health programmer tend to face a diverse range of critical morbidity. The present ICOG focus has been planned to make our members acquainted and updated on key management issues in such situations.

Dr. Sadhana Gupta has done a tremendous work in the field of maternal and neonatal health, and I congratulate her for bringing up the idea and framing content of the ICOG Campus. I heartily thank all authors who are expert in their field and contributed excellent articles with carry home messages.

Indian College of Obs & Gynaecology (ICOG) aims for teaching and fellowship for quality health care to women and neonate. This campus is one of the steps in fulfilling aims and vision of ICOG. This year we had organized a series of certified course, Journal Club, national and international conferences and teaching course for fellow students of ICOG. I as a chairperson of ICOG urge you to involve in ICOG activities and join ICOG in large numbers.

Wishing you happy learning,

Dr. Madakini Megh
ICOG Chairperson

message

Dear Colleagues,

Managing acute life-threatening complications in our clinical practice is a huge challenge. It requires clinical acumen, quick responses and skill. Sound knowledge to manage and come out of these conditions is essential. This compilation will fulfill this need

Writing about these serious complications is also a huge challenge because it needs to discuss several issues, bringing out the importance and urgency of each step - in an unpredictable and ever changing clinical scenario.

I congratulate Dr Sadhana Gupta and her team for taking on this difficult task. It will be extremely helpful for every obstetrician who can get the latest know how on managing these high risk conditions in a concise booklet.

My best wishes with the team for this effort,

Dr. Uday Thanawala
MD, DGO, FCPS, DNB
Chairman Elect ICOG 2021
Vice President FOGSI 2015
Chairman,
Medical Disorders in Pregnancy Committee of
FOGSI (2006-2009)

message

Dear Colleague in FOGSI/ICOG

Allow me to wish you all the greetings of the Uttarayan, heralding the upcoming spring! I am very happy to note that one of our most active and energetic fellows of ICOG, Dr. Sadhana Gupta from Gorakhpur has painstakingly prepared the E-ICOG Focus on Severe Acute Maternal Morbidity. She has worked in collaboration with Dr. Mandakini Megh, (Chairperson ICOG), and brought out this important issue dealing with critical challenges in Obstetrics. I am very sure that we all shall benefit from this work, and it will make a positive change in our obstetric practice. The range of topics included is quite diverse, from rupture uterus and caesarean hysterectomy to severe sepsis, septic shock to amniotic fluid embolism. I commend Dr. Sadhana for her effort in difficult corona times, and wish this issue of FOGSI focus a great success!

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message

Greetings from ICOG!

I hope all our readers are staying safe during this difficult time of ongoing Covid pandemic. ICOG Team 20-21 brings in yet another publication on the important problem of "Severe acute maternal morbidity".

Maternal health is of utmost importance to us obstetricians. Most of labour and delivery have a favourable outcome with a heathy baby and safe mother. Some unfortunate women go through a stormy child birth and obstetricians need to prepare themselves to manage these patients carefully. Haemorrhage, sepsis & thromboembolism are situations seldom faced by Obstetricians but theses complicated scenarios need careful assessment, vigilant monitoring, judicial use of various life saving measures and timely referral to tertiary care centres with an aim to save lives of women and the unborn foetus. With increasing c section rates, lies a danger of problems of rupture uterus and abnormal adherence of placenta which at times requires caesarean hysterectomy.

I would like to congratulate editors, Dr. Madakini Megh and Dr. Sadhana Gupta for giving us a quality publication with senior teachers and practitioners throwing light on all situations endangering life of mothers. I thank all contributors for their practical deliberations on various clinical conditions.

Happy learning

Dr. Parag Biniwale
Secretary ICOG


Dr. Manadkini Megh


Dr. Sadhana Gupta


Dr. Mousumi Das Ghosh


Dr. Hema J Shobhane

from desk of editors

Dear ICOG Fellows, Members & FOGSIANS,

It is a moment of pride and pleasure while we release ICOG Campus on theme of Severe acute maternal morbidity as editors.

Severe acute maternal morbidity (SAMM) is a acronym for more popular term which we call Near Miss for women who became critically ill in course of pregnancy but/yet survived due to timely intervention, coordinated management added to luck & incidental factor. While in India there have been a sharp decline in maternal mortality in last 15 years, we as obstetrician & gynaecologist face many diverse, critical obstetrical situations of near miss due to changing obstetrical demography.

When a woman develops SAMM it is usually not a co incidence. It is a culmination of social economic cultural factors, missed opportunities in antenatal care, complacent action in emergency situations and sometimes due to procedure and intervention itself. Defining and categorising SAMM is difficult and complex and it varies in different categories of health facilities.

In present ICOG campus we have focused on wide range of subjects from common clinical situations like severe PIH, haemorrhage, sepsis, uterine rupture, anaesthetic mishaps to uncommon events like endocrine emergencies and amniotic fluid embolism. All authors are experts and experienced in the filed of high risk and critical obstetrics and have given concise knowledge and clear messages for benefits of all readers.

ICOG is academic wing of FOGSI and aimed to join hand in hand to spread education, knowledge and skill among its members and fellows. This ICOG Campus is aimed to create knowledge and good management skill to deal with critical obstetrical situations and saving lives.

Maternal criticality and maternal mortality both bear heavy collective consciousness of all who deal with it be it doctors, nurses or families. Yet near miss mothers who are saved after going through critical situations are the hard yet strong learning happy event not only for the individual doctors but the whole healthcare systems. We hope and believe that this issue of ICOG Campus will be taken as a key learning material by all of you.

I thank my co editor and ICOG chair person Dr. Mandakini Megh for her constant guidance and encouragement and my dear joint editors Dr. Mousumi Das Ghosh and Dr. Hema J. Shobhane for their hard work in editing the chapters. I am highly thankful to FOGSI President Dr. Alpesh Gandhi for his inspirational message, with all the high regards for his exemplary work in field of critical care in obstetrics. I also thank ICOG office bearers for their support and permission to release the ICOG campus.

We wish you happy learning, health and happiness to all of you. We will wait for your feedback remarks and suggestions which go a long way to improve such endeavours in future.

Chief Editor-
Dr. Manadkini Megh
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Severe Acute Maternal Morbidity (SAMM) - Concept, Causes and Way forward

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Severe acute maternal morbidity (SAMM) includes the unintended outcomes of the process of labour and delivery that result in significant short term or long term consequences to a womens health[1]. ''Near miss'' and ''SAMM'' are the two terms used interchangeably for a ''severe, life-threatening obstetric complication.'' The term ''near miss'' in health care is adapted from airline industry[2]. Maternal near-miss morbidity is defined to include women who experience a life-threatening event during pregnancy or postpartum (up to 42 days after the end of pregnancy) and survive because of good luck and the care that they receive[3]. In contrast, Severe Acute Maternal Morbidity refers to the morbidity a woman actually suffers. SAMM capture more accurately the serious and life-threatening conditions affecting pregnancy, labour, and the postpartum period that could potentially result in maternal death[4]. 'Nearmiss' now refers to avoidable risks whereas SAMM retains the concept of the harmed or damaged mother[5].

A woman's lifetime risk of dying during pregnancy and childbirth continues to be high in resource-limited countries like India and other Asian and African countries. In India, the policies promote institutional births, births by skilled birth attendants and provision of Emergency Obstetric Care. The Janani Suraksha Yojana (JSY) a cash incentive scheme promotes institutional deliveries. Despite a significant decline in the number of maternal deaths, the rate of decline is different in various parts of the country. SAMM is used along with maternal death review to identify the deficiencies in health care as they occur more frequently, share similar characteristics and is readily reported by health care providers[6].

Incidence of SAMM

The incidence of SAMM is difficult to predict as there is no international and uniform definition. Two methods have been described to address the relationship between severe morbidity and mortality. These are the mortality-to-morbidity ratio and the mortality index. The mortality-to-morbidity ratio describes the number of severe morbidity cases for each maternal death. The mortality index, on the other hand, is defined as the number of maternal deaths divided by the sum of women with SAMM and maternal deaths, and is expressed as a percentage[5].

It is estimated for every maternal death, between 20 and 70 women sustain a 'near miss' or severe maternal morbidity[7]. What we need is a multi-disciplinary approach aimed at cutting each thread of the web of causation of mortality and morbidity in pregnancy.

Concept of SAMM

Three approaches are used to identify the condition by WHO (2)

1. **Clinical criteria related to a specific disease entity**

   Specific diseases are used as the starting points and then for each disease, morbidity is defined. For example pre-eclampsia is the disease entity, and complications such as renal failure, eclampsia and pulmonary oedema are used to define severe morbidity

2. **Intervention based criteria**

In this system an intervention such as admission to an Intensive Care Unit (ICU), the need for an emergency hysterectomy, the need for blood transfusion and interventional radiology are used as markers. The data are easily available , both prospectively or retrospectively, even in low-resource settings.

3. **Organ system dysfunction based criteria**

Organ system dysfunction	Criteria
Cardiovascular dysfunction	Shock Cardiac arrest and cardiopulmonary resuscitation Use of continuous vasoactive drugs Severe hypoperfusion (lactate>5 mmol/l or >45 mg/dl Severe acidosis (pH < 7.1)
Respiratory dysfunction	Acute cyanosis Gasping Respiratory rate >40 or < 6/minIntubation and ventilation (not related to anaesthesia) Severe hypoxemia
Renal dysfunction	Oliguria non-responsive to fluids or diuretics Dialysis for acute renal failure Severe acute azotemia (creatinine e"3.5 mg/dl)
Coagulation / hematological dysfunction	Severe acute thrombocytopenia (< 50,000 platelets/ml) PT or aPTT > 1.5 times of normalTransfusion of e"5 units of blood/red cells
Hepatic dysfunction	Jaundice in the presence of pre-eclampsia severe acute hyperbilirubinemia (bilirubin > 6.0 mg/dl)
Neurological dysfunction	Prolonged unconsciousness (lasting e"12 hours) Coma (including metabolic coma) Stroke Uncontrollable fits/status epilepticus

The sequence from health to death includes diseases or injuries causing a systemic inflammatory response, organ dysfunction and death. The criteria for defining a maternal near miss are defined per organ system. The use of locally relevant potentially life-threatening conditions are encouraged and the WHO guidance provides a basic set of conditions together with operational definitions[8].

Few studies show that condition-based definitions are better than management-based ones as the former can be used in poorly resourced areas. In developed countries, 0.4% of women experience severe acute maternal morbidity (SAMM) when organ-system based criteria were used, however this rate increased to 1% when disease-specific criteria were used for monitoring practices. Similarly, in developing countries, the rate of women delivering in hospitals who experienced severe acute maternal morbidity was 1% when organ failure criteria were used and 4–8% using case-identification criteria[9].

The ACOG and the society for maternal foetal medicine recommend screening for SAMM with the following two criteria- a} transfusion of four or more units of blood and b} admission of pregnant or postpartum women to ICU[1].

Obstetric hemorrhage, hypertensive states associated with severe pregnancy, such as eclampsia and HELLP syndrome and sepsis, are events that occur in most countries, as the main cause of obstetric mortality and morbidity

The Risk Pyramid

Severe acute maternal morbidity (SAMM) includes events involved in the biological continuum that goes from the normal expected healthy situation of a pregnancy to maternal death. The pregnancy can be uncomplicated, severely complicated or life-threatening and a lady may recover, may be temporarily or permanently disabled or she may die[10]. Maternal deaths constitute the tip of the iceberg whose important part is the hidden one, which is formed by patients with acute severe obstetric morbidity, whose evolution could be towards recovery, or temporary disability or death[11].

Many factors can move a woman both up and down the risk pyramid for severity of morbidity

Factors responsible are categorised into –

a. Patient factors - which are potentially the hardest to rectify, especially in developing countries where access to education is limited[12].

b. System factors includes communication, policies/procedures, transport or administrative systems

c. Provider-related factors, mainly incomplete or inappropriate management. This is more likely due to non-availability of specialist staff, not adhering to protocols/standards. Non-adherence to guidelines has been identified as a risk factor for increased maternal morbidity, whereas dissemination of guidelines and skills

drills are associated with improved adherence to the agreed protocols and significant reduction in morbidity[5].

Any action or inaction on the part of the health care provider, system, patient, or a combination of these factors that may have caused progression to more severe morbidity can be a topic for audit and improvement.

Outcome of women who suffer SAMM

Complications as a result of SAMM and interventions to ensure survival impact the physical, mental and reproductive health of these women[3]. There is a growing body of evidence on the long-term adverse consequences for women's health and socio-economic conditions secondary to severe obstetric complications. The women continue to be a burden on health services with its personal, family and economic cost. Emotional support is needed after recovery from acute life threatening event or perinatal loss. There is a risk of abandonment of the woman who has had a hysterectomy and is therefore of no further reproductive use to the family and to huge indebtedness due to catastrophic out-of-pocket expenditure for treatment.

Death of a mother in itself is catastrophic and has a strong detrimental influence on the newborn as well as the entire family leading to a vast emotional, psychosocial and economic vacuum.

Audits- surveillance and response

Assessment of SAMM cases is increasingly used to complement maternal mortality review and a topic in quality of care issues in maternity care(13). The majority of SAMM cases are potentially preventable or required improvement in care. They have the advantage of not being as rare as maternal deaths for providing adequate information, as well as still being rare enough not to overload clinicians and data collection personnel within a facility[14].

As countries progress through the stages of obstetric transition and more and more women increasingly deliver in facilities, it is paramount to ensure that facilities and health systems have the tools to measure and improve quality of care. Every outcome as severe maternal morbidity may not have opportunity of improvement. Review and audit of such cases determine whether the morbidity could have been avoided and whether it should prompt changes in systems for care provision – which are important for quality obstetric care. They should be considered as free lessons and opportunities to improve the quality of service provision.

Overall, maternal death and near-miss audits generate valuable information responsible for the generation of actionable information that effectively guides immediate and long-term actions[8]. They are more likely to function as a positive entry point for critical assessment and subsequent behaviour change as health professionals are more open to discuss failures and successes during care. It also allows direct interviews with survivors providing an important and complementary perspective on how the care has been accessed, received and perceived including aspects of respectful care.

Implementing prospective surveillance also contributes to establishing an institutional culture of emergency preparedness, which is potentiated by training and emergency drills. Overall, the 'blame and shame' attitude should be avoided while conducting maternal death and maternal near-miss reviews because it erodes institutional cooperation and intrinsic motivation of health professionals[15]

The response component engages and promotes the interaction within multiple health service and health system building blocks, including the governance, information systems, finances, supplies, health services and human resources

Decreasing SAMM- moving forward

Analytic attempts to define the concept more strictly and descriptive efforts to measure and quantify new indicators of near-miss for different geographical regions should be made(16). Standardization and comparison can be made between maternal morbidity groups from different locations and over time. Identification of the obstacles and gaps in the health system and a coordinated approach to

resolve these can ultimately lead to an improved health system.

Based on the risk factors already identified and presently available treatments, interventions can be tested at different levels to reduce the rate of SAMM or to convert high-scoring cases to lower-scoring ones[17]. In the morbidity-to-mortality continuum, the key is to enable identification of the key factors responsible for moving women along the continuum and targeting interventions that shift women more towards morbidity rather than mortality. To effectively improve outcome, change has to be implemented at various levels, from primary-care providers, through secondary and tertiary centers to health-care systems.

Facilities for blood transfusion, blood storage, quick referral should be made available at the primary level. The need to prioritize effective communications among nurses, obstetricians and other specialists to provide timely, responsive treatment should be emphasized. Prompt replacement of the lost blood volume is of vital importance in cases like post-partum and ante-partum haemorrhage[18]. Lack of manpower can be alleviated by appointing skilled health care providers at least at the district level and community level. Educating staff about the emergencies in obstetrics, conducting mock drills to handle emergency situations, conducting training programmes for improving obstetric skills can help. Lack of infrastructure can be solved by the joint effort of the health providers at the Primary health care level and the government by providing adequate funds and facilities. The findings can inform clinical educational programs and policies to improve maternal outcomes[13].

Conclusion

Tracking and evaluating the care provided to maternal near-miss cases has the potential to function as a catalyst for both improving women's delivery experiences and outcomes and strengthening the health systems. It is crucial to understand the processes of obstetric care in order to address any identified weakness or failure within the system The fear of blame and punishment is less in SAMM review, so, when performed effectively, it can more easily lead to implementation of changes that will improve the quality of services. Furthermore, accurate and routine measurement of the spectrum of maternal morbidity is necessary to inform policy and programme decisions to further reduce maternal mortality and morbidity.

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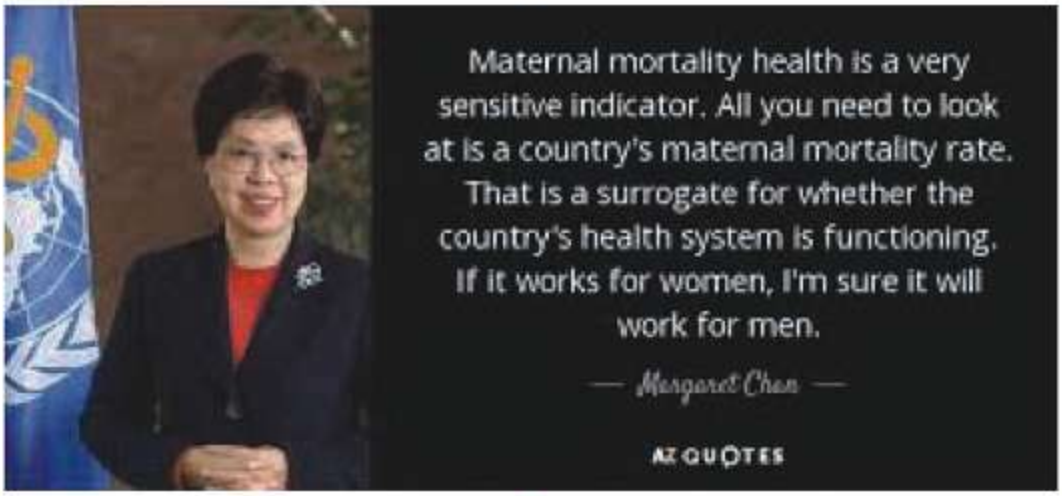

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Obstetrical Haemorrhage - Key issues to save lives

In most low-income, PPH affects about 5% of all birthing females and is the leading cause of maternal mortality. It is estimated that for every maternal death due to PPH, there are 10 'near misses'. The aim is to decrease the global ratio of maternal mortality to less than 70 per 100,000 live births by 2030. Reviews also shown that 93 percent of all deaths associated with hemorrhage are potentially considered.[1] Improving women's healthcare during childbirth for the prevention and treatment of PPH is an important step towards achieving sustainable health Development Goal 5 (SDG 3.1)[1]

Most of these fatalities occur within 4 hours of delivery, suggesting that they are a result of the third stage of labour. The frequency of PPH is 5.8 percent in the first pregnancy and the risk of first PPH is 4-5 percent in the second or third pregnancy. The chance of PPH recurrence is up to 15 percent in a subsequent pregnancy.[2]

Recognition of blood loss, lack of sufficient attention to clinical signs of hemorrhage and related hypovolemia, failure to respond decisively with lifesaving interventions, and failure to restore blood volume in a timely manner are common preventable errors.[3]

Etilogy :

The most prevalent and relevant cause of PPH is uterine atony. However, in women with no risk factors, 20% of postpartum hemorrhage occurs, so physicians must be trained for every delivery to treat this condition.

The causes of PPH, are classified in terms of the 4 Ts: (Table 1)

- Tone: uterine atony, distended bladder (incidence-70%)
- Tissue: retained placenta and clots (20%)
- Trauma: vaginal, cervical, or uterine injury (10%)
- Thrombin: coagulopathy (pre-existing or acquired) (1%)

Complications of postpartum hemorrhage are common, even in high-resource countries and well-staffed delivery suites. Based on an analysis of systems errors identified in The Joint Commission's 2010 Sentinel Event Alert, the commission recommended that hospitals establish protocols to enable an optimal response to changes in maternal vital signs and clinical condition. In response, The Council on Patient Safety in Women's Health Care outlined essential steps that delivery units should take to decrease the incidence and severity of postpartum hemorrhage[4]. The creation of a hemorrhage cart with supplies, and the use of huddles, rapid response teams, and massive transfusion protocols are among the recommendations. Advanced Life Support in Obstetrics (ALSO) training can be part of a systems approach to improving patient care. The use of interdisciplinary team training with in situ simulation, available through the ALSO program and from Team STEPPS (Team Strategies and Tools to Enhance Performance and Patient Safety), has been shown to improve perinatal safety.[5,6]

A patient safety kit is a series of simple, evidence-based guidelines proven to optimize results for practice and care processes. Such a kit is not a new guideline, but rather reflects a set of current guidelines and recommendations in a manner that facilitates the application and continuity of practice. 7 The Obstetric Hemorrhage Consensus Package is grouped into four areas of action: Readiness,

Recognition and Prevention, Response, and Reporting and Systems Learning.

There are 13 key elements within these four action domains.

Obstetric Hemorrhage Safety Bundle From the National Partnership for Maternal Safety, Council on Patient Safety in Women's Health Care.[7]

Readiness (Every Unit)

1. Hemorrhage cart with supplies, checklist, and instruction cards for intrauterine balloons and compression stitches
2. Immediate access to hemorrhage medications (kit or equivalent)
3. Establish a response team—who to call when help is needed (blood bank, advanced gynaecologic surgery, other support and tertiary services)
4. Establish massive and emergency-release transfusion protocols (type-O negative or un cross matched)
5. Unit education on protocols, unit-based drills (with post drill debriefs)

Recognition and Prevention (Every Patient)

6. Assessment of hemorrhage risk (prenatal, on admission,)
7. Measurement of cumulative blood loss (formal, as quantitative)
8. Active management of the 3rd stage of labor

Response (Every Hemorrhage)

9. Unit-standard, stage-based obstetric hemorrhage plan/with checklists
10. Support program for patients, families, and staff

Reporting and Systems Learning (Every Unit)

11. Establish a culture of huddles for high-risk patients and post-event briefs
12. Multidisciplinary review of serious hemorrhages for systems issues
13. Monitor outcomes and process metrics in perinatal quality improvement

Hemorrhage Cart

In order to deal with emergency hemorrhage cases, it should consist of a sterile tray with easy access to instruments. It is also useful for the cart to contain cognitive aids for theoretically seldom performed procedures, such as uterine tamponade balloon placement and uterine compression sutures.

Hemorrhage Medication Kit

Oxytocin (Pitocin)	10 units 2 vials
Oxytocin (Pitocin)	10-40 units per 500-1000mL solution 2 pre- mixed bags
15-methyl PGF2α	250 micrograms/milliliters 1 ampule
Misoprostol	200 microgram tablets 5 tab
Methyl ergonovine (Methergine)	0.2 milligrams/milliliters 1 ampule

Establish a Response Team

The team will likely engage physicians from anesthesiology, transfusion service (blood bank), pharmacy, advanced gynecological surgery, critical care medicine, the main operating room, interventional radiology, and other nursing services in addition to the primary maternity care provider and nurse. Also, use readily accessible phone or pager numbers or a "rapid response" or "code" system to alert appropriate team members.

Establish Massive and Emergency Release Transfusion Protocols

The operation of blood bank facilities should be ensured by any institution. A large transfusion protocol enables the rapid dispensing of a pre-defined ratio of RBCs, plasma, and platelets to prevent the creation of a dilutional coagulopathy that can occur if a substantial percentage of the blood volume of the patient is replaced.

Hemorrhage Drills

A drill refers to the simple and reasonable collection of patient care measures involved. It helps to explain how obstetric emergency drills can be created and implemented. Both members of the team are familiar with the whole security kit and the current management strategy. Participating members are assigned tasks and duties required

Assessment of Hemorrhage Risk

Identifying risk factors for postpartum hemorrhage may help improve preparation, encourage improved monitoring and early detection, increase the use of preventive measures, and prepare the team to initiate an early, aggressive bleeding response. Usually these methods consider 25 percent of women to be at higher risk who will then develop 60 percent of the serious hemorrhages (requiring transfusion).

Therefore, because approximately 40 percent of postpartum hemorrhage occur in women at low risk, it is important to recognize that and birth is at risk, reinforcing the need for universal vigilance. Risk evaluation should be considered during patient treatment at various points of time, including antepartum, on admission to labor and delivery, later in labor.

Risk Assessment[8]

- 1 Antenatal Risk Factors
- Previous retained placenta or PPH previous PPH or retained placenta
- Maternal anaemia. This should be investigated and treated appropriately as this may reduce the morbidity associated with PPH
- Maternal age (35 years or more)
- Body mass index greater than 35 kg/m2
- Grand multiparity (parity 4 or more)
- Antepartum haemorrhage
- Over distention of the uterus (i.e. multiple pregnancy, polyhydramnios or macrosomia)
- Pre-existing uterine abnormalities
- Low lying placenta
- Maternal age (35 years or more)
- Risk Factors in Labour
- Induction of labour
- Prolonged first, second or third stage of labour
- Secondary arrest of labour, especially in multi-gravid patients

Use of oxytocin

Precipitate labour Operative delivery birth or caesarean section

Measurement of Cumulative Blood Loss

A leading cause of delayed reaction that can result in morbidity or worse is the imprecise measurement of real blood loss during birth and the postpartum period. Visual blood loss estimation can result in a 33-50 percent underestimation of blood loss, especially when large volumes are lost.

Two complementary methods can achieve direct measurement of blood loss. Blood is collected in calibrated, under-buttock drapes for vaginal delivery or in calibrated cesarean birth canisters, the easiest to initiate. The second method is to measure blood-soaked products and clots, and to achieve an estimation of blood loss, the weight of dry pads is subtracted from the overall weight.

Methods / Materials Used	Estimated blood loss (ml)
Small 10x10 cm 32-ply swab (max saturated capacity)	60
Medium 30x30 cm 12-ply swab (max saturated capacity)	140
Large 45x45 cm 12-ply swab (max saturated capacity)	350
1 kg-soaked swabs	1000
Kidney dish full of clots	500
50 cm diameter floor spill	500
75 cm diameter floor spill	1000
100 cm diameter floor spill	1500
Vaginal PPH limited to bed only	< 1000
Vaginal PPH spilling over from bed to floor	> 1000

Active Management of Third Stage of Labor

It has been shown that successful management of the third stage of labor is the single most effective method to avoiding postpartum hemorrhage. Latest studies have shown that oxytocin is the main component of the three classic components: oxytocin, uterine massage, and cord traction. Oxytocin prophylactic application, 10 units intramuscular injection, remains the most powerful drug with the least side effects compared to ergot

alkaloids (nausea and vomiting) and misoprostoloids (hyperpyrexia).

Response (Every Hemorrhage)

Obstetric Hemorrhage Emergency Management Plan

Because obstetric hemorrhage represents a diverse group of diagnoses, a critical initial step is to determine the etiology. Stage-based management plans have been found to facilitate an organized, stepwise response to blood loss and maternal warning signs.[9,10] Each institution needs to adjust the plan to meet its individual capabilities.

CHECKLIST	DIAGNOSIS	INTERVENTION	ROLE OF BLOOD BANK
STAGE 0 Activate hemorrhage protocol	Asses risk factors for hemorrhage. Estimate blood loss	Active management of third stage	High risk-send type & cross matchLow risk-Type & screen
STAGE 1 Advance through medications and procedures-finding cause & treatment/ visualization	• Estimated Blood loss (EBL) > 500 mL vaginal > 1000 mL cesarean • Vitals/Lab values-NORMAL	• 16G/18G IV access • Increase IV fluid • Insert indwelling urinary catheter • Fundal massage • Increase oxytocin • Consider additional uterotonics	Type & cross match 2 units PRBCs
STAGE 2 Mobilizing help and Blood Bank support-Keeping check of volume and blood products.	Continued bleeding with EBL < 1500mL,or > 2 uterotonics Vitals/Lab values-NORMAL	• Second IV (16-18G) line • Draw labs tests (CBC, PT/ APTT, fibrinogen) • *Vaginal Birth:* Repair tears, rule out retained placenta • Place intrauterine balloon • Selective Embolization • *Cesarean*- Inspect broad ligament • B-Lynch Suture • Place intrauterine balloon • Uterine curettage • Placental bed suture • Uterine artery ligation • Uteroovarian ligation • Repair uterine rupture • Hysterectomy	• Consider 2Unit PRBC transfusion as per clinical signs – not wait for lab values • Use blood warmer • Consider thawing 2 FFP, if transfusing > 2Unit PRBCs • Check availability of additional RBCs and Coagulation products
STAGE 3 Activate massive transfusion protocol & Practice surgical Approach	• Bleeding with EBL over1500ml, OR • > 2 units PRBCs given • Vitals/Lab values-ABNORMAL • Suspicion of DIC	Mobilize team – Gynecologist – Anesthetist – Adult Intensivist • Repeat lab values • Central line insertion • Family support • Surgical management • Supportive compression stockings	Transfuse Aggressively-Massive Hemorrhage Pack (6:4:1). If not improving with 8-10 units PRBCs and full coagulation factor replacement: consider factor VIIa

Support Program for Patients, Families and Staff

Women and their families need timely information, reassurance, and opportunities to discuss the incident with the maternity care provider.

- Reporting and Systems Learning (Every Unit)
- Establish A Culture of Huddles and Debriefs

Briefs, huddles, and debriefs need to be routine. Briefs are planning meetings that are used to form the team, designate roles and responsibilities, establish goals, and engage the team in short-term and long-term planning. Huddles are brief ad hoc team meetings designed to regain situational awareness, discuss critical

issues and emerging events, anticipate outcomes and contingencies, assign resources, and express concerns. Debriefs are short, informal feedback sessions that occur after events and are designed to identify opportunities to improve teamwork, skills, and outcomes.[7]

Multidisciplinary Review of Serious Hemorrhages

These are formal meetings including staff involved in the incident, unit and facility leadership, and risk-management personnel. The purpose of these reviews is to identify systems issues or breakdowns that influenced the outcome of the event. Reviews should be sanctioned by the facility, protected from discovery in legal proceedings, and include a thorough record review, event timeline, and focused root-cause analysis.

Monitor Outcomes and Process Metrics Monitoring process and outcome measures is important for the successful introduction of quality- improvement projects. Project success is generally measured by improved outcomes. The overall goal is to reduce the number of obstetric hemorrhages that escalate into major blood loss resulting in severe maternal morbidity or mortality.[7]

Conclusion

Complications of postpartum hemorrhage are common, even in high-resource countries and well-staffed delivery suites. Globally PPH is the leading cause of maternal mortality and morbidity. Prevention plays a very important role by identifying high risk factors and active management of labour. A standardized approach to obstetric hemorrhage includes a clearly defined, staged checklist of appropriate actions to be taken in an emergency situation and can help to improve patient outcomes. Management is medical, mechanical, surgical and radiological. A multi-disciplinary approach is essential in severe haemorrhage Availability of blood and blood products is essential. It is very important to identify the aetiology, though uterine atony is common. Prediction and assessment of blood loss remains the cornerstone for prompt and effective management of PPH.

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Severe Pre-eclampsia & Eclampsia - Roadmap for Diagnosis & Management

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Introduction

Hypertensive disorders of pregnancy occur in about 10% of all pregnant women around the world. Preeclampsia affects 3–5% of pregnancies.[1] It contributes to 10% to 15% of direct maternal deaths globally. As per WHO 2 more than 35,000 mothers died due to hypertension related causes in 2017 in India.

Preecclmpsia is increasing in recent times due to obesity, increased maternal age & many mothers with comorbid conditions entering pregnancy. Preeclampsia-eclampsia not only put mothers and fetuses at increased risk of serious complications, during that index pregnancy but also in future.

Definition : Preeclampsia was previously defined as blood pressure records > 140/90 mm of Hg two readings measured 4 hours apart + significant proteinuria (>1+ on dipstick or >300mg/day in 24 hours urine sample) after 20 weeks of gestation.

Now proteinuria is no longer considered mandatory for diagnosis. Current definition of preeclampsia is hypertension with or without proteinuria with one or more system involvement and fetal growth restriction.

Preeclampsia was previously classified as mild & severe. Now it is divided as preeclampsia without severe features & with severe features (ACOG 2019)[3]. Severe features are as follows :

Blood pressure >160mm Hg systolic or >110 mm Hg diastolic

Thrombocytopenia (platelet count <100,000 x 109/L)

Impaired liver function as indicated by abnormally elevated blood concentrations of liver enzymes > twice and severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses

Renal insufficiency (serum creatinine concentration more than 1.1 mg/dL)

Pulmonary edema

New-onset headache unresponsive to medication and not accounted for by alternative diagnoses

Visual disturbances like scotoma, blurring of visison

Also fetal growth restriction due to uteroplacental insufficiency is considered feature of severe disease.

It is also divided in early onset preeclampsia (onset <34 wks) & late onset preeclampsia (>34 wks). Early onset PE is less common but more severe as compared to late onset PE.

Diagnosis of severe preeclampsia

It is a disease of signs rather than symptoms & confirmed by lab investigations. Preeclampsia is usually initially symptomless & becomes symptomatic quite late.

Blood pressure measurement (>160/110 mm of Hg), significant proteinuria, symptoms of systemic involvement likeheadache, visual symptoms, nausea, vomiting, epigastric pain, oliguria, facial & hand edema & laboratory tests as mentioned above confirms the diagnosis.

For proteinuria 24 hours collection is sometimes difficult & delays the diagnosis & treatment, so protein/creatinine ratio of 0.3 (both measured in mg/dl) or more in a spot urine sample is diagnostic.

Currently there is no ideal screening test for prediction of PE. FIGO4 has recently suggested that all pregnant women should be screened for preterm preeclampsia by first trimester combined test with maternal risk factors, mean arterial pressure, uterine artery Pusatality index & placental growth factor. In high risk women defined by this first trimester combined test low dose aspiri (150 mg) & calcium (1.5 to 2 gm) should be given from 12 wks of pregnancy.

Management of severe PE

Delivery is the cure.

Management depends upon the gestational age :-

< 24 wks → Stabilize & terminate. There is very high perinatal mortality (>80 %) & high maternal morbidity & mortality in continuing the pregnancy for fetal maturity. One should share the decision with patient & her family.

> 34 wks → Stabilize & deliver. > 34 wks : Fetal lung maturity is achieved, so terminating pregnancy decreases maternal risks. However if maternal condition is stable and lab parameters are normal one can prolong pregnancy till 37 wks& then terminate.

24 - 34 wks → Stabilize & continue the pregnancy. Terminate when indicated

Hospitalization

Imminent symptoms of eclampsia are looked for daily

BP monitoring 4 hourly except at night.

Daily weight measurement, input output charting.

Antihypertensive are started.

Lab investigations: Baseline investigations include CBC, LFT, RFT, Coagulation profile, grouping & crossmatching on admission & repeated twice a week or more frequently as necessary.

Fundoscopy is done.

Fetal evaluation; DFMC is advised, NST done twice weekly, USG with AFI and umblical doppler to be done weekly.

Antihypertensive therapy

Aim is to bring down systolic BP at 140-150 mm Hg & diastolic BP at 90-100 mm of Hg. Excessive & abrupt reduction of BP is not advisable as it may affect uteroplacental circulation & harm the fetus.

- Labetalol: 20mg bolus IV in 10 ml dilution if not responding in 10 minutes repeat dose of 40mg IV, followed by 80mg in 10 min again if bp still high. Not more than 300mg can be given.
- Nifedipine- 10mg of oral nifedipine can be given every 30 min. Dose should not increase 50mg.
- Hydralazine- 5mg IV repeat every 20 min, maximum upto 50mg.
- Nitroglycerine – 5 ug /min infusion double every 5 min.

  Oral Labetolol (200-1200 mg/day) or Nifedipine (30-120 mg/day) may be started once BP decreases to desirable level.

  The choice & route of administration of an antihypertensive drug should be based primarily on the clinician's experience with that particular drug & local availability.

Steroids for fetal lung maturity

- All women before 34 weeks. As per RCOG guideline it is given till 38 wks 6 days if elective CS is planned.

  Dexamethasone 6 mg I/M 4 doses every 12 hours or Betamethasone 12mg I/M 2 doses 24 hours apart are given.

Indications for delivery in severe preeclampsia

- 34 wks reached
- HELLP syndrome
- Eclampsia/ Imminent eclampsia
- Abruption
- Progressive Deterioration of hepatic or renal function
- IUGR/ Oligohydramnios

- Non-reassuring fetal status (on NST or BPP or doppler)
- Uncontrolled BP (SBP > 160 mm Hg or DBP > 110 mm Hg) despite treatment by 3 drugs.
- Pulmonary oedema
- Oliguria 0.5 ml/kg/hr
- Thrombocytopenia

*The mathematical model called preeclampsia integrated & estimated risks (full PIERS) was developed with the aim to reduce uncertainty in these decisions. It helps in predicting adverse ourcomes within 48 hours of admission of the patient.

Intrapartum management

- Induction of labour can be done with prostaglandins or foley's catheter. Oxytocin should be used judiciously as it can cause water retention and hyponatremia.
- CS is favored in preterm fetus remote from term & for obstetric indications.
- BP is measured every 15 min.
- CVP line is indicated if there is pulmonary edema or renal disease.
- Electronic fetal monitoring is must.
- $MgSO_4$ full dose is given if hypertension remains uncontrolled or there are features of impending eclampsia. Famous MAGPIE trial confirmed the role of prophylactic $MgSO_4$[6]
- Second stage of labour is cut short only if hypertension is uncontrolled.
- Ergometrine is avoided in third stage. It may be required if PPH occurs.
- For CS regional anesthesia is favoured except when there is Coagulation disorder or thrombocytopenia.
- Routine preload is not necessary for regional anesthesia.

Postpartum

- BP should be monitored & antihypertensive should not be stopped suddenly even if BP becomes normal. On 3rd to 5th day there can be rebound increase in BP.
- ACE inhibitors (Enalapril, Captopril) can now be used

HELLP Syndrome

It is complication of severe PE. It is hemolysis (H) with elevated Liver Enzymes (EL) & Low Platelet Count (LP). The acronym, HELLP syndrome, was coined by Dr. Louis Weinstein in 1982.7 Complete HELLP should have all the 3 components, however in partial or incomplete HELLP syndrome, there or only 1 or 2 components.

Incidence of HELLP is 0.2-0.6%. About 10- 20% of woman with severe pre-eclampsia develop HELLP; in 70% it is developed antepartum and in 30% it develops after delivery.

Criteria for diagnosis of HELLP Syndrome:

1. Hemolysis (at least two of the following)
   a. Peripheral smear(schistocytes,burr cells)
   b. Serum bilirubin (>1.2mg/dL)
   c. Low serum haptoglobin
   d. Severe anemia unrelated to blood loss
2. Elevated Liver enzymes
   a. ALT or AST >/= twice upper level of normal
   b. LDH >/= twice upper level of normal
3. Low platelets < 100.000/mm3

Clinically the patients present with increased BP, significant proteinuria, oedema, right hypochondriac pain, vomiting, headache, visual change, bleeding from mucosal surfaces, hematuria and petechiae and non-specific viral syndrome (bodyache, low grade fever, rash etc).

The presence of HELLP syndrome is associated with an increased risk of maternal death and increased rate of maternal morbidities.

Management :

- Admission in high dependency unit
- Investigations
- Start antihypertensive therapy
- Blood component transfusions

- Vitamin K, prevention of hepatic encephalopathy
- Termination

Mode of Delivery :

Caesarean is restricted for obstetric indications.

Platelets >20,000 are needed for vaginal Delivery and 40,000 for caesarean section.

Atypical Preeclampsia :

- Atypical cases are those that develop at < 20 weeks of gestation and > 48 hours after delivery and that have some of the signs and symptoms of preeclampsia without the usual hypertension or proteinuria.
- Criteria for Atypical Preeclampsia : Gestational hypertension **OR** Gestational proteinuria plus ≥ 1 of the following items:
  - Symptoms of preeclampsia
  - Hemolysis
  - Thrombocytopenia (<100,000/mm3)
  - Elevated liver enzymes.
- Of total cases of preeclampsia incidence of atypical preeclampsia is 8%
- They should be treated as severe preeclampsia.

Eclampsia

It is defined as preeclampsia complicated by tonic clonic convulsions. Any convulsion during pregnancy, labour and postpartum period should be considered as eclampsia until proved otherwise.

Incidence of eclampsia in India is about 1.5%. Maternal mortality varies from 2.2 to 9.[8]

Differential diagnosis:

- Epilepsy
- Hysteria
- Encephalitis
- Meningitis
- Cerebral tumour
- Rupture of cerebral aneurysm
- Cerebrovascular accidents
- Hypertensive encephalopathy
- Hypoglycemia
- Strychnine posisoning

Stages in Eclampsia

Premonitory stage - Eyes rolled up, twitching on face and limbs, loss of consciousness

Tonic stage - Generalized tonic contraction of entire body. Opisthotonos and cessation of respiration. This lasts for half minute.

Clonic stage - There is alternative contraction and relaxation of the muscles. Tongue may be bitten; breathing resumes and become stertorous. Blood stained frothy secretions come out of the mouth & nose. It lasts for about 1-3 minutes.

Coma or post convulsion stage - It may last for few minutes or persist till another convulsion.

Management

If convulsion occurs outside the hospital, patient's face is turned to one side, padded spoon (or any such object) is inserted in mouth (to prevent tongue bite & tongue fall). Injection Magnesium sulfate loading dose is given & patient is shifted to tertiary care hospital.

In the hospital :

- Patient is kept in High Dependency Unit (HDU), with one to one nursing care.
- Eclampsia cot should have guard rails to prevent fall.
- Suction is done during & after convulsions as required.
- Oxygen is given by nasal catheter, 5 litre/min.
- Mouth gag is inserted in unconscious patient or kept handy.
- Monitoring of pulse, BP, respiration & oxygen saturation is done.
- Intra-cath No. 18 is inserted, blood is taken for investigations & cross matching & 5% glucose drip is started.
- Anticonvulsants & anti-hypertensives are started
- Antibiotics are given.
- Foley's catheter is inserted for drainage of

bladder & for measuring urine output.

– Fetal monitoring.

Obstetric Management

Termination of pregnancy after initial stabilization of the mother should occur in 12 hours. CS is usually indicated in < 34 wks gestation as induction of labour takes longer time for delivery. After 34 wks IOL can be done if cervix is favourable & there are no other obstetric indication for CS. However If delivery does not take in 12 hours or maternal or fetal condition deteriorates CS is readily done. Transient fetal bradycardia immediately after convulsion is common & is not an indication for CS. On other side sometimes even if the fetus is dead or nonviable CS is preferred for maternal viewpoint.

Anticonvulsant Therapy

Magnesium Sulphate ($MgSO_4$) Regimens

Pritchard regime:

Intravenous : 4 gm of 20% $MgSO_4$ is given IV over more than 4 min.

Intramuscular : 10 gm of 50%w/v $MgSO_4$ is given IM with 5 gm in each buttock using long bore needle with 1 ml of 2 % lignocaine.

If convulsions persists after 15 minutes 2g (10 ml of 20%) is given over 2 minutes.

If convulsions still persists IV Phenytoin (1gm slowly in 20 min) or Diazepam (10 mg in 2 min) can be given OR patient is given GA & Emergency CS is carried out.

Maintenance dose : 5g (10 ml of 50%)is given every 4 hours in alternate buttocks after assuring :- 1) presence of knee reflex, 2) urine output > 100 ml & 3) respiratory rate >14/min

If creatinine is more than 1.2mg/dl, then half the dose is to be given.

NB: Intramuscular route is contraindicated in patients with HELLP syndrome or thrombocytopenia due to risk of hematoma formation at injection site.

Full IM & IV dose is given when $MgSO_4$ is used for prophylactic purpose e.g. in imminent eclampsia.

Zuspan regime :

Loading dose : 4g of $MgSO_4$ is diluted in 100ml of IV fluid (5% Dextrose) & is given over 15-20 min.

Maintenance dose : 1g/hr of $MgSO_4$ is infused in 100 ml of IV fluid.

Sibai modification :

Here the loading dose is 6g of $MgSO_4$ & Maintenance dose of 2g/hr required in large woman.

$MgSO_4$ is continued for 24 hrs of delivery or of last convulsions, whichever is later. Recently it is suggested that 12 hours time is sufficient to prevent recurrence of convulsion instead of 24 hours.[9]

Therapeutic serum levels of magnesium is 5-8 mg/dl

Loss of tendon reflexes occurs 9-12 mg/dl

Respiratory depression at 15-20 mg/dl

Calcium gluconate is to be given in case of $MgSO_4$ toxicity (repiratory toxicity) at a dose of 1gm (10ml of 10% Soln) over 10 min IV.

$MgSO_4$ is very safe drug. Even in case when any sign of toxicity is seen generally withholding the next dose is sufficient to address it. Failing to administer $MgSO_4$ is much more dangerous than any risk from using it.

Modifications of standard regimens

In the last decade researchers in the developing countries are constantly striving to steadily decrease the doses of $MgSO_4$ regimens.

1. **Sardesai Suman et al** suggested 4 g of $MgSO_4$ as loading dose IV and subsequently 2 g of $MgSO_4$ was given every 3rd hourly IV/IM.
2. **Begum et al** used low dose 'Dhaka regimen' comprising 10g of loading dose, following this 2.5g was given intramuscularly 4th hourly.
3. Joshi et al suggested single dose $MgSO_4$ **'VIMS regimen'** (4g diluted intravenously plus 4g intramuscularly) at Primary Health care level itself before transferring the patient to tertiary care centre for definitive, disciplined management.

PRES (Posterior Reversible Encephalopathy Syndrome)

Posterior reversible encephalopathy syndrome (PRES) is commonly found in patients of preeclampsia. It is a clinico-radiological syndrome characterized by headache, seizures, altered mental status and visual loss and characterized by vasogenic edema of white matter affecting the posterior occipital and parietal lobes of the brain predominantly.

PRES should be managed in ICU with BP control measures along with Neuroprotective agents.

Key Messages

- Severe preeclampsia and eclampsia are dreaded life-threatening diseases of pregnancy specially in country like ours. Increased vigilance should be implemented in patients with high risk of developing Hypertension. Many women will develop preeclampsia with no clinical symptoms. Prediction for early onset preeclampsia by multiparametric predictive models are promising. Low dose aspirin & Calcium in high dose from 12 wks of pregnancy helps in prevention. Delivery is the only cure. Conservative management can be considered in view of reducing risk of prematurity in fetus with high end monitoring and ICU facilities.

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> Prenatal care is one of the most effective ways to reduce maternal mortality because it identifies complications or high risks before emergency situations.
> ~ Liya Kebede

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Obstetrical Sepsis

Introduction

Sepsis during pregnancy and the puerperium remains a leading cause of maternal morbidity and mortality.[1] It is the third cause of maternal mortality worldwide.[2] WHO estimated that the global prevalence of maternal sepsis is 4.4% among live births, with an incidence of 9–49 per 100 000 deliveries in high-income countries.[3,4]

Efforts to implement early warning systems, revise the definition of sepsis, and develop maternal sepsis care bundles, diagnosis and management of maternal sepsis lead to better maternal and fetal outcomes.[5]

Definitions of Sepsis and Septic Shock

The original definition of sepsis dates back to 20 years. In 1991, American College of Chest Physicians defined sepsis as Systemic inflammatory Response Syndrome (SIRS) with infection. SIRS is an inflammatory response to physiological insult which is characterized by the presence of:

1. Temperature : Hyperthermia (>38°C) or Hypothermia(< 36°C)
2. Tachycardia (> 90 beats/ min)
3. Tachypnea (> 20 /min) or PaCO2 <32mm Hg
4. Leucophilia (>12,000/ mm3) or leucopenia (<4000/mm3)

Severe sepsis is defined as Sepsis associated with organ dysfunction, hypotension or hypoperfusion.[6]

Septic shock is defined as severe sepsis with persistent hypotension despite adequate volume resuscitation or need for inotropic or vasopressor agents.

Revised Definitions

In 2016, international consensus of sepsis and septic shock (Sepsis 3) revised the definition of sepsis as "a life-threatening organ dysfunction caused by a dysregulated host response to infection (Infection + Organ Dysfunction)"

The term severe sepsis has been removed and septic shock has been defined as Sepsis with persistent hypotension requiring vasopressors to maintain mean arterial pressure (MAP) 65 mmHg and a serum lactate level >2 mmol/L (18 mg/dL).**7**

According to WHO, Maternal sepsis is a life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion, or the postpartum period.[3]

Risk factors for maternal sepsis

The largest independent obstetric risk factor for postpartum maternal sepsis is operative intervention, and caesarean section is associated with a 5 to 20% increase in infectious morbidity compared with vaginal birth. Cesarean section after the onset of labour poses the greatest risk, followed by elective Cesarean section and then operative vaginal delivery.

Other obstetric related risk factors include cervical cerclage, prolonged rupture of the membranes, a history of pelvic infection, a history of group B streptococcal infection or group A streptococcus in close contacts or family members, vaginal discharge, multiple pregnancy, retained products of conception, preterm prelabour rupture of membranes (PPROM) and amniocentesis or other invasive procedures.[8]

Patient related risk factors include primiparity, preexisting medical conditions, impaired glucose tolerance, obesity, anemia, use of antibiotics within

two weeks of delivery and on immunosuppressants.[9]

Causes of Maternal sepsis[10]

(i) Genital tract causes: chorioamnionitis, endometritis, septic abortion, wound infection after vaginal tear, episiotomy, or Caesarean section

(ii) Renal causes: lower urinary tract infection, pyelonephritis

(iii) Respiratory causes: pneumonia—bacterial, viral; tuberculosis

(iv) Intraperitoneal causes: ruptured appendix, acute appendicitis, acute cholecystitis, bowel infarction

(v) Other causes: breast infection, septic pelvic thrombophlebitis, necrotizing fasciitis, malaria, miliary tuberculosis.

Physiological changes during pregnancy and their impact[11]

System	Physiologic change	Impact
Cardiovascular	Decreased arterial pressure Increased heart rate and cardiac output	Increased risk of hypoperfusion in sepsis Abnormal baseline may mask signs of sepsis
Gastrointestinal	Decreased esophageal tone and delayed gastric emptying	Aspiration pneumonia risk Increased aspiration risk with airway interventions
Genitourinary	Decreased vaginal pH	Increased risk of chorioamnionitis
Hematology	Increased WBC, plasma volume without proportional increase in red cell mass, hemoglobin Increased production of factors VII, VIII, IX, X, XII and von Willebrand factor	Physiologic anemia, decreased O2 supply to tissues Increased risk of disseminated intravascular coagulation and venous thromboembolic disease
Respiratory	Increased tidal volume and minute ventilation with typically unchanged respiratory rate	Decreased PaCO2 levels
Renal	Ureteral dilation and increased vesicoureteral reflux Increased renal plasma flow and glomerular filtration rate	Increased risk of pyelonephritis Abnormal baseline may mask renal injury in sepsis

Etiopathogenesis

The pathophysiology of sepsis is complex and involves the interaction of multiple biological pathways via positive and negative feedback loops.

Pelvic infections are usually polymicrobial, bacteria that cause severe sepsis syndrome are frequently endotoxin-producing Enterobacteriaceae, most commonly *E coli*. Others are aerobic and anaerobic streptococci, *Bacteroides* and *Clostridium* species. Some strains of group A β-hemolytic streptococci and *Staphylococcus aureus*—including community-acquired methicillin-resistant strains (CA-MRSA)—produce a superantigen that activates T cells to rapidly cause all features of the sepsis-*toxic shock syndrome*. Exotoxins leads to rapid and extensive tissue necrosis and gangrene, especially of the postpartum uterus, and may cause profound cardiovascular collapse and maternal death.

Sepsis is usually inflammatory response to endo and exo toxins, which stimulate T cells and leukocytes, increasing the production of proinflammatory compounds, resulting in cytokine storm. This causes selective vasodilation with maldistribution of blood flow. Leukocyte and platelet aggregation cause capillary plugging. Worsening endothelial injury causes profound permeability, capillary leakage, and interstitial fluid accumulation.

In its early stages, clinical shock results primarily from decreased systemic vascular resistance that is not compensated fully by increased cardiac output. Hypoperfusion results in lactic acidosis, decreased tissue oxygen extraction, and end-organ dysfunction that includes acute lung and kidney injury.

Clinical Features

1. Central nervous system: confusion, delirium, somnolence, coma, combativeness, fever
2. Cardiovascular: tachycardia, hypotension
3. Pulmonary: tachypnea, arteriovenous shunting

with dysoxia and hypoxemia, exudative infiltrates from endothelial-alveolar damage, pulmonary hypertension

4. Gastrointestinal: gastroenteritis—nausea, vomiting, and diarrhea; ileus; hepatocellular necrosis—jaundice, transaminitis
5. Renal: prerenal oliguria, azotemia, acute kidney injury, proteinuria
6. Hematological: leukocytosis or leukopenia, thrombocytopenia, activation of coagulation with disseminated intravascular coagulopathy
7. Endocrine: hyperglycemia, adrenal insufficiency
8. Cutaneous: acrocyanosis, erythroderma, bullae, digital gangrene.

Diagnosis

The anatomic and physiologic changes of pregnancy pose a challenge in the early recognition and management of sepsis as they overlap with hemodynamic changes associated with the initial presentation of sepsis. Maternal sepsis can associated with early pregnancy loss, intrauterine death, fetal tachycardia or fetal bradycardia. While fever is often the first vital sign change that raises the index of suspicion of maternal sepsis for clinicians, temperature alone may not be a reliable indicator of sepsis. There are some scoring systems that have been used to aid diagnosis and prognostication of sepsis. However, these may assist, but do not replace clinical judgement.

1. *Obstetrically modified SOFA* [Sequential (sepsis-related) organ failure assessment] score which was given by the Society of Obstetric Medicine of Australia and New Zealand and uses pregnancy-specific physiological variables to identify a critically ill obstetric patient.[12]

System	Parameter	Score		
		0	1	2
Respiratory	PaO2/FiO2	≥400	300-<400	<300
Cardiovascular	Mean arterial pressure (mmHg)	≥70	<70	Vasopressors required
CNS	Consciousness	Alert	Arousable by voice	Arousable by pain
Liver	Bilirubin (µmol/l)	≥20	20-32	>32
Coagulation	Platelets (X 10^9/l)	≥150	100-150	<100
Renal	Creatinine (µmol/l)	≥90	91-120	>120

2. *Quick SOFA (qSOFA)* score is a rapid clinical assessment prior to the investigation reports and is as follows. An abnormal score should prompt initiation or escalation of therapy and a score of e"2 is associated with an increased risk of mortality.[12]

Parameter	Score	
	0	1
Mentation	Alert	Not alert
Systolic BP (mmHg)	≥90	<90
Respirator rate (breaths/min)	<25	≥25

3. The RCOG recommends the use of *Modified early obstetric warning system* (MEOWS) which has 89% sensitivity and 79% specificity in identifying maternal morbidity.[13] It includes parameters like temperature, systolic and diastolic BP, heart rate, respiratory rate, oxygen saturation, pain score, neurological response, proteinuria, colour of amniotic fluid, lochia (normal/ heavy/ offensive) and whether patient looks unwell.[14]
4. *'Maternal Early Warning Trigger (MEWT)'* tool identifies four of the major causes of maternal morbidity resulting in ICU admission (i.e. sepsis, cardiopulmonary dysfunction, preeclampsia-hypertension and haemorrhage).[15]
5. Sepsis in obstetrics score (SOS) was developed by Albright et al16 and used a composite of physiological variables of pregnancy and biomarkers to identify pregnant women with an increased risk of morbidity in association with suspected sepsis. Patients with an SOS score ≥6 are more likely to be admitted to the ICU.[12]

Variables	Score								
	+4	+3	+2	+1	0 (normal)	+1	+2	+3	+4
Temp (°C)	>40.9	39-40.9		38.5-38.9	36-38.4	34-35.9	32-33.9	30-31.9	<30
SBP (mHg)					>90		70-90		<70
HR (/min)	>179	150-179	130-149	120-129	≥119				
RR (/min)	>49	35-49		25-34	12-24	10-11	6-9		≥5
SpO2 (%)					≥92	90-91		85-89	<85
Leukocytes (/µl)	>39.9		25-39.9	17-24.9	5.7-16.9	3-5.6	1-2.9		<1
Immature neutrophils (%)			≥10%		<10%				
Lactic acid (mmol/l)			≥4		<4				

Biomarkers[12]

These help in identifying patients at the early stages of sepsis, differentiating sepsis from other non-infectious inflammatory pathologies, predicting clinical severity or outcome and guiding escalation/de-escalation of treatment.

Total leukocyte count (TLC) and C- reactive protein (CRP): Nonspecific for infection versus inflammation but are widely used.

Procalcitonin : It more specific for bacterial infection. NICE and Surviving sepsis campaign (SSC) guidelines do not recommend its routine use to guide the acute treatment during suspected bacterial infection and should be used with caution.

Lactate : A level of >2 mmol/l should prompt critical care input. Hyperlactataemia is a marker for anaerobic metabolism subsequent to tissue hypoperfusion. Every 1mmol/L increase in lactate is associated with a 2.34-fold increased risk in the need for ICU admission.[17]

Management

The aim is to maintain oxygenation and perfusion of vital organs and placenta while identifying and treating infection.

RCOG recommends the use of sepsis bundles which are a group of interventions designed to allow the team to follow the timing, sequence, and goals of individual elements of care to improve the outcome.

Updated sepsis bundle from the Surviving Sepsis Campaign (SCC)[17]

Within 1 h, the 'Sepsis Six' has to be followed:

(i) Administer high-flow oxygen

(ii) Take blood cultures both aerobic and anaerobic without delay. At least two sets of blood culture should be obtained, one drawn percutaneously and one from a vascular site, unless the device was recently inserted

(iii) Administer broad-spectrum antibiotics

(iv) Fluid resuscitation—administer 20 ml kg21 crystalloid for hypotension or if lactate 4 mmol/litre21

(v) Measure serum lactate

(vi) Catheterize and measure accurate hourly urine output

Within 6 h:

(i) Administer vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain MAP65 mm Hg

(ii) In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate 4 mmol/l, maintain CVP 8–12 mm Hg.

(iii) Serial lactate measurement if initial lactate was elevated.

Treatment of organ dysfunction:[4]

(i) Fluid resuscitation: The SSC recommends initial intravenous fluids, crystalloids at a rate of 30 ml/kg. RCOG modified it to 20 ml/kg due to an increased risk of pulmonary oedema in pregnancy caused by decreased colloid oncotic

pressure, increased plasma volume, pre-eclampsia or due to the uterotonic drugs causing retention. Thus, non-invasive cardiac output monitoring should be considered to guide fluid resuscitation.[9,18,19]

(ii) Vasopressors and ionotropes: norepinephrine is the first choice as a vasopressor to maintain mean arterial pressure (MAP) of 65 mm Hg. Epinephrine and vasopressin can be added if necessary. In the presence of myocardial dysfunction or ongoing signs of hypoperfusion, dobutamine should be administered.

(iii) Current RCOG guidelines do not consider steroid use for sepsis, but recommend cautious use in the context of promoting fetal lung maturity. They may be considered if haemodynamic instability continues despite resuscitation.

(iv) Glucose levels are maintained less than 180 mg/dl (10 mmol/l).[12]

(v) In the absence of ischaemic heart disease or signs of hypoperfusion, it is suggested to maintain haemoglobin at 70–90 g/l.

(vi) Sepsis is associated with coagulopathy; monitoring and correction of coagulopathy is required.10

(vii) Extracorporeal membranous oxygenation (ECMO) has been used for either cardiac or respiratory failure. It leads to good recovery of cardiac function with survival rate of 70% and 80% in the fetus and mother respectively.[12]

2. Identification and control of source of infection:[4]

In septic shock, there is a 7.6% decrease in survival for every hour of delay in antibiotic administration after the onset of hypotension. Initial antibiotics administered in sepsis should be broad spectrum, administered within one hour of suspected sepsis, after blood for culture has been taken.

Proposed broad-spectrum empiric antibiotic coverage in sepsis complicating pregnancy[20]

Community-acquired pneumonia: Cefotaxime, ceftriaxone, ertapenem, or ampicillin plus azithromycin, clarithromycin, or erythromycin

Hospital-acquired pneumonia: Low-risk patients may be treated with piperacillin-tazobactam, meropenem,imipenem, or cefepime. High risk of mortality need Pseudomonas (beta lactam plus an aminoglycoside or a quinolone) and MRSA coverage with vancomycin or linezolid.

Chorioamnionitis: Ampicillin plus gentamicin. Add anaerobic coverage with clindamycin or metronidazole if cesarean delivery required.

Endomyometritis: Ampicillin, gentamicin, and metronidazole (or clindamycin)/ cefotaxime or ceftriaxone plus metronidazole.

Urinary tract infections: Gentamicin with ampicillin/ monotherapy with a carbapenem or piperacillin-tazobactam.

Abdominal infections: Ceftriaxone, cefotaxime, ceftazidime, or cefepime plus metronidazole. Complicated cases may require monotherapy with a carbapenem or piperacillin-tazobactam.

Skin and soft tissues (necrotizing): Vancomycin plus piperacillin-tazobactam. If Streptococcus Group A or Clostridium perfringens are present, use penicillin G plus clindamycin.

Intravenous immunoglobulins (IVIgs) can be considered as an adjunct to antibiotics, particularly during severe invasive staphylococcal and streptococcal sepsis.[12]

Timing of delivery: Attempting early delivery in patients with severe cardiovascular compromise due to sepsis may increase maternal and fetal mortality,unless chorioamnionitis is suspected as the source of sepsis or a septic abortion has occurred.If the risks of continuing the pregnancy outweigh those of early delivery, administration of antenatal steroids and neuroprophylaxis should be considered to improve the fetal outcome.[12]

Surgical management in maternal sepsis:[10]

(i) Evacuation of retained products of conception

(ii) Debridement of wound infection or fasciitis

(iii) Percutaneous drainage of abscesses

(iv) Stent or percutaneous nephrostomy for

obstructive pyelonephritis

(v) Delivery of fetus if chorioamnionitis is suspected

(vi) Hysterectomy for myometrial necrosis

Prevention[10,12]

Antenatal screening for asymptomatic bacteriuria.

NICE guidelines antibiotic prophylaxis use during caesarean section and also following operative vaginal delivery.

Hand hygiene and frequent use of alcohol gel by hospital staff and relatives.

Use of personal protective equipment—gloves, disposable aprons, gowns, face mask and eye protection.

Use of modified early warning scoring systems and education to enable early identification of septic patients Involvement of infection control surveillance teams to monitor progress.

WHO and Jhpiego launched the Global Maternal and Neonatal Sepsis Initiative with the goal to accelerate the reduction of preventable maternal and neonatal deaths related to sepsis. This aims to foster collaboration, research, innovation and advocacy to eradicate sepsis. The STOP SEPSIS campaign helped build awareness among healthcare providers for the prevention, detection and treatment of maternal and neonatal sepsis. The Global Maternal Sepsis Study (GLOSS) is an ongoing research in 52 countries to estimate the burden of maternal infections around the world.[21,22]

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"Maternal mortality is not statistics. It is not numbers. It is not rates or ratios. Maternal mortality is people. It is women, women who have names, women who have faces and we have seen these faces in the throes of agony, distress and despair. They are faces that continue to live in our memory and haunt our dreams. This is not simply because these are women who die in the prime of their lives, at a time of great expectation and joy. And it is not simply because a maternal death is one of the most terrible ways to die, be it bleeding to death, the convulsions of toxemia of pregnancy, the unbearable pangs of obstructed labor, or the agony of puerperal sepsis. It is because in almost each and every case, in retrospect, it is an event that could have been prevented. It is an event that should never have been allowed to happen. It is an event that bears and should bear so heavily on our collective conscience."

Dr Mahmoud Fathalla
World Health Day, April 7, 1998.

Amniotic Fluid Embolism & Thromboembolism - Readiness Matters

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Introduction

It is one of the rarest but unpredictable, catastrophic and devastating complications unique to pregnancy associated with high maternal and neonatal mortality and morbidity resulting from dissemination of amniotic fluid, fetal cells and other debris into the maternal pulmonary circulation. Historically, it dates back to 1926 when it was first reported by Meyer and later described by Steiner and Luschbaugh in 1941.1 Lack of international consensus over diagnostic criteria and under reporting of non fatal cases results in variation of incidence rates as well as mortality rates throughout the world. Incidence rate is estimated to be 2 to 8 per 100,000 births in different countries and the case related maternal mortality ranges between 0.5 to 1.7 deaths per 100,000 deliveries in the developed world and 1.9 to 5.9 deaths per 100,000 deliveries in the developing world.[2,3,4]

Risk Factors

AFE is so occasional that there are no well established risk factors. 70% of cases occur intrapartum, 11% after vaginal delivery and 19% during cesarean section.[5] Rarely it may occur during termination of pregnancy or during procedures like amniocentesis or chorionic villous sampling in first and second trimester.

Table 1 : Risk Factors of AFE[6,7,8]

Advanced age (> 35 years)	Eclampsia
Multiparity	Multiple pregnancy
Caesarean section	Genital tract lacerations
Male fetus	Induction of labour
Placental abruption/previa	Meconium stained liquor
Intrauterine fetal death	

Pathophysiology

It is a complex sequence of events triggered in a subset of women due to dispersion of materials from fetal into maternal compartment resulting in exaggerated inflammatory response. Attwood and Benson suggested anaphylaxis as a mechanism and supported this hypothesis by testing women for serum tryptase released by mast cells in patients with AFE rather than mechanical obstruction.[7] Therefore, terminology of anaphylactic syndrome of pregnancy has been recommended as fetal tissue or amniotic fluid components are not always found on autopsy in females with signs and symptoms attributable to AFE. There are two theories that have been postulated which is depicted in flowcharts below.

Disruption in the barrier between placental and amniotic fluid interface.
↓
Entry of amniotic fluid and its debris into the systemic circulation causing mechanical obstruction of the pulmonary circulation.
↓
Intense pulmonary vasoconstriction associated with bronchoconstriction.
↓
Acute pulmonary arterial occlusion. Both right atria and ventricles gets dilated along with Tricuspid regurgitation. Enlargement of the right ventricle deviates the interventricular septum towards the left side leading to the systolic dysfunction and further aggravating the pulmonary arterial pressure → Decreased cardiac output → Hypoxemia and hypotension Sudden cardiovascular collapse.[10,11]

Amniotic debris enter into the maternal circulation.
↓
Activation of severe inflammatory response
↓
Humoral or immunological response[12,13]

- Pregnancy is a hypercoagulable state and introduction of amniotic fluid trigger inflammatory mediators leads to activation of

coagulation cascade and fibrinolytic system resulting in severe and unrelenting DIC which many a times is refractory to treatment.[14]

Table 2: Clinical types of AFE

	Initial symptom	Time from symptom onset to cardiac arrest	Histology	Initial management
Cardiopul-monary collapse type (classic type)	• Sudden dyspnea • Severe hypotension (including cardiac arrest) • seizure	Very short (0-60 minutes in typical cases)	Amniotic components in pulmonary vessels	CPR including inotropes
DIC type	• Massive bleeding without clotting • Uterine atony	Several hours	Amniotic components in uterus &/ or uterine vessels Thrombus in uterine vessels Interstitial edema in uterus	Volume resuscitation including supplement of platelets and clotting factors

Signs and Symptoms

Clinical presentation is abrupt with sudden cardio respiratory collapse followed by severe coagulopathy and remains a diagnosis of exclusion. Anxiety and agitation with altered mental status may precede the event rapidly progressing to cardiac arrest with pulse less electrical activity, arrhythmia or ventricular fibrillation. Diagnosis is suspected when there is classical triad of hypoxia, hypotension and coagulopathy developing during labor or immediately after delivery, caesarean section, dilation and evacuation or within 30 min postpartum with no other explanation of findings. There is still lack of uniform and international consensus in the diagnosis of AFE but Benson and UKOSS criteria have been proposed to overcome this problem.[16,17]

Recently Society of Fetal Medicine and AFE foundation have defined objective criteria of AFE

Uniform diagnostic criteria for research reporting of AFE[18]

- Sudden onset of cardio respiratory arrest, or both hypotension (systolic BP <90 mm Hg) and respiratory compromise (dyspnea, cyanosis, or peripheral capillary oxygen saturation [SpO2] <90%)
- Documentation of overt disseminated intravascular coagulation (DIC) following appearance of these initial signs or symptoms, using scoring system of Scientific and Standardization Committee on DIC of the International Society on Thrombosis and Homeostasis (ISTH), modified for pregnancy.

Table 3 : ISTH Criteria for DIC in pregnancy

	0	1	2
Platelet count	> 100,000/mL	< 100,000/mL	< 50,000/ mL
Prolonged PT/INR	< 2 5% increase	25-50% increase	> 50% increase
Fibrinogen level	> 2 g/L	< 2 g/L	

- Clinical onset during labor or within 30 min of delivery of placenta
- No fever (≤38.0°C) during labor

Table 4 : Differential Diagnosis[7,19]

Non obstetric causes :	Obstetric causes :
• Pulmonary embolism	• Eclampsia
• Pulmonary edema	• Abruption placenta
• Tension pneumothorax	• Uterine rupture
• Anaphylaxis	• Genital tract lacerations
• Aspiration	• Post partum hemorrhage
• Anesthesia related	• Peri partum cardiomyopathy
• Cardiac – Myocardial infarction, heart failure, Arrhythmias, tamponade	

Investigations

Till date, there are still no diagnostic assays, imaging studies or pathological markers that have been approved to confirm the diagnosis of AFE; these are used instead for monitoring of patient and optimization of treatment. Evaluation is tailored to two main systems failure: hemodynamic and hematological.

- Serial complete blood counts to detect the degree of anemia as well as for appropriate

quantity of blood transfusion required.

- Comprehensive metabolic panel is also advised as electrolyte disturbances are common.
- Coagulation profile (PT, aPTT, D-dimer, FDP) should also be done serially for evaluation of early coagulopathy so that correction of all parameters should be done urgently.
- Pulmonary and metabolic acidosis is common and therefore intra-arterial lines are useful to facilitate minute to minute pressure measurement and frequent arterial blood gas sampling .Bedside measurement of pulmonary wedge pressure, cardiac output, central venous pressure, pulse oximetry, arterial waveform should be started.
- Arterial blood gas analysis should be done for detection of degree of hypoxia/hypoxemia. The expected change would be decreased pH, decreased pO2, increased pCO2 level with increase in base excess.
- Chest radiograph: Findings are suggestive of acute pulmonary edema.12 Leads ECG detect changes in ST segment and T wave suggestive of right ventricle strain.
- Echocardiography (transthoracic/ transesophageal) aid early diagnosis by showing acute pulmonary vasoconstriction, right ventricle dilation, hypo kinesis, tricuspid regurgitation and right atrial enlargement and collapsed left ventricle due to left deviation of inter ventricular septum. Use of Transthoracic esophageal echocardiography allows identification of specific pathophysiological processes that are available to targeted hemodynamic intervention.[21,22]
- Increased serum tryptase, urinary histamine concentration and significantly lower complement concentrations suggest an anaphylactic process.[23]
- Few studies have evaluated the diagnostic accuracy of serum sialyl Tn (STN), a fetal antigen present in meconium and amniotic fluid, detected through the use of TKH-2 monoclonal antibody. TKH-2 reacts with meconium and mucin and stains the lung tissue in those with AFE. For serum levels >50U/ml, the sensitivities varied between 78% and 100% and the specificities between 97% and 99%.[24,25]

Complications[26,27]

• Left ventricular systolic dysfunction
• Prolonged coagulopathy
• Liver failure
• Seizures
• Anoxic encephalopathy
• Renal failure

Management

Goals of treatment[28,29,30]

- Maintenance of vital signs along with rapid correction of maternal hemodynamic instability,correction of hypoxia and hypotension, for preventing subsequent end-organ failure.
- Oxygenation and control of the airway with tracheal intubation and administration of 100% O2 with positive pressure ventilation should be performed as soon as possible.
- Fluid resuscitation is imperative to counteract hypotension and hemodynamic instability. A central venous pressure line may assist in assessing right sided preload. Crystalloids must be given judiciously as copious fluid administration may dilute the clotting factors, worsening the bleeding and therefore decision to use vasopressors should be considered earlier as compared to significant bleeding from other etiologies.
- DIC management includes administration of units of packed RBC, fresh frozen plasma and platelets in a ratio 1:1:1 until bleeding is controlled. Maintain fibrinogen levels >200 mg/dl. Administer FFP to normalize the PT. Tranexemic acid should be given to prevent fibrinolysis.

If fibrinogen level is <100mg/dl, administer cryoprecipitate and each unit raises fibrinogen level by 10 mg/dl.

- Use of vasopressors, antiarrhythmic agents and defibrillating doses is not different from those utilized in non pregnant individuals. Ideal management is to maintain mean arterial pressure(MAP) >65 mmHg, a cardiac index of more than 2L per meter square, an adequate urine output of 40-50 ml/hr, and a PaO2/FiO2 ratio more than 250.
- Use of veno arterial extra corporeal membrane oxygenation (ECMO) has been described refractory to conventional resuscitation maneuvers. However, use of anticoagulants during extra corporeal membrane oxygenation may worsen bleeding in the profoundly coagulopathic patient with active hemorrhage. Because of these as well as lack of adequate evidence of benefit of extra corporeal membrane oxygenation, its role is controversial and not routinely recommended in the management of AFE.
- Obstetrical management include delivery of the foetus by emergency cesaerean section especially if gestational age is >23 weeks to improve outcome.

Various agents are recommended in cases of acute right ventricular failure[31] like sildenafil, inhaled nitric oxide, inhaled prostacyclin, intravenous prostacyclin acting as Pulmonary vasodilator; dobutamine, milrinone (inotropes); norepinephrine (vasopressors).

Prognosis

More than half of the patients succumbed to AFE within 1½ - 2 hrs of initial clinical presentations due to sudden cardiac arrest, haemorrhage, ARDS or multiple organ failure. Among the survivors, persistent neurological impairment occurs in 6-61% of females[23]

Recommendation[32]

AFE should be considered in the differential diagnosis of sudden cardiorespiratory compromise in any pregnant or recently postpartum patient (GRADE 1C).

Use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE is not recommended at present; remains a clinical diagnosis (GRADE 1C).

Provision of immediate high-quality cardiopulmonary resuscitation with standard basic cardiac life support and advanced cardiac life support protocols in patients who develop cardiac arrest associated with AFE is recommended (GRADE 1C).

A multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in the ongoing care of women with AFE (Best Practice).

Following cardiac arrest with AFE, immediate delivery in the presence of a fetus ≤23 weeks of gestation (GRADE 2C).

Because coagulopathy may follow cardiovascular collapse with AFE, the early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols (GRADE 1C).

Conclusion

AFE is a diagnosis of exclusion with no proven risk factor to predict its occurrence.

- AFE typically occurs intrapartum or immediately postpartum and diagnosed solely on clinical criteria consisting of the classical triad hypotension, coagulopathy and hypoxia.
- AFE is not a mechanical embolic phenomenon but an overwhelming inflammatory response due to exposure of fetal antigen.
- Prognosis is dismal associated with high maternal and neonatal mortality and morbidity.
- Management is only supportive and immediate aggressive respiratory and hemodynamic support along with replacement of clotting factors in a well equipped ICU with multi-

disciplinary approach. Sometimes immediate delivery is necessary especially if period of gestation is more than 23 weeks to improve the fetal and maternal outcome.

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Published:July 31, 2019DOI:https://doi.org/10.1016/j.ajog.2019.07.036

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Life-threatening Endocrinological Emergencies in Obstetrics

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Introduction

Endocrinal emergencies in obstetrics are not so common but can be life threatening for both mother and foetus. All endocrinological emergencies require a high clinical suspicion, rapid treatment and multi-disciplinary team approach with endocrinologist, maternal-foetal specialist and intensivist to ensure best outcome. Delivery is generally avoided until maternal status improves.

The following are the potentially fatal common endocrinological emergencies in pregnancy:-

a) Thyroid storm,

b) Diabetic ketoacidosis,

c) Primary hyperparathyroidism

d) Acute pituitary complications in pregnancy
   i. Prolactinoma
   ii. Sheehan's syndrome
   iii. Pituitary apoplexy

Thyroid storm /Thyrotoxiosis

Uncontrolled or untreated hyperthyroidism can potentially develop into thyroid storm or thyrotoxicosis with high maternal and foetal mortality. Graves' disease is the commonest cause of hyperthyroidism during pregnancy.

Thyroid storm is an acute hypermetabolic state precipitated by an excess of endogenous thyroid hormone and may lead to non-obstetric maternal death. The incidence of hyperthyroidism in pregnancy is about 0.2% and mostly subclinical but thyrotoxicosis is a fatal complication in hyperthyroidism patient (1-2% of cases of hyperthyroidism). Total mortality rate in the thyroid storm is 10 to 20% due to late diagnosis and treatment[1-3]. It is defined by a markedly depressed (<0.1mU/L) or undetectable (<0.01 mU/L) serum thyroid stimulating hormone (TSH), along with elevated serum free thyroxine (T4) and/or tri-iodothyronine (T3) levels[4].

Thyroid storm has an acute onset and is diagnosed based on symptoms like fever, tachycardia, and central nervous system dysfunction (altered mental status, restlessness, and seizures). Most cases are due to poorly controlled hormone levels. Preceding events such as pre-eclampsia, infection, stress, trauma, surgery, and ketoacidosis has been associated with storm[5,6]. Presenting features may be non-specific, so can be confused with other conditions[7]. Elevated blood pressure, headaches, abdominal pain and pulmonary edema or heart failure are features compatible with preeclampsia can delay the diagnosis[8]. But overt sign of hyperthyroidism such as goitor, exophthalmos or thyroid bruit help in diagnosis. Burch and Wartofsky[9] have outlined a clinical scoring system for the probability of early diagnosis of thyroid storm (Table 1).

Cautious interpretation of thyroid hormone profile is needed in the first trimester due to human chorionic gonadotropin effect on the thyroid gland. An associated leucocytosis, hyperglycaemia, hypercalcemia, elevated liver enzymes, and electrolyte disturbances on metabolic panel screening are characteristics of thyroid storm. Few patients can have thyrotoxic heart failure due to myocardial effects of excess free T4. Respiratory failure is generally not associated with thyroid storm.

The treatment is rapid inhibition of thyroid hormone synthesis and its peripheral conversion, aggressive management of the systemic disturbances and identification and treatment of the precipitating cause[10]. Aggressive treatment of the cardiac failure is essential. Dexamethasone, 8 mg /day can be administered as it blocks the deiodination T4 to T3. Alternatively, hydrocortisone or prednisone can also be given. Propylthiouracil (PTU) is the preferred anti-thyroid medication. It can also block the peripheral conversion of T4 to T3. PTU can be administered orally/ by nasogastric tube/and rectally. PTU is given at a dose of 200-250mg every 6 hours. Iodine can be administered 1 hour after PTU to inhibit the release of preformed thyroid hormone. Iodine can be given orally (Lugol's solution or saturated solution of potassium iodide, 4-8 drops every 6-8 hours; radiographic contrast media: iopanoic acid or sodium ipodate-2 gm loading dose followed by 1gm daily) or intravenously (Sodium iodide 1gm in 250 - 500 ml normal saline, infused 12 hrly).

Management of hyperpyrexia, treatment of precipitating factors and control of hyperadrenergic activity should be instituted simultaneously. Propranolol should be administered orally (40 -80 mg every 6-8 hours) or intravenously (0.5 -1 mg over 10 minutes followed by 1 -3 mg every 4 hours). The short acting beta 1-selective antagonist, esmolol can also be used intravenously (0.25 -0.5 µg/kg loading dose, followed by an infusion of 0.05 -0.1 µg/kg/ minute). Emergency thyroidectomy with or without plasmapheresis has been described successfully in thyroid storm but must be considered high risk and last line of treatment[11,12]. (Figure 1)

All these metabolic changes can lead to foetal heart rate tracing abnormalities[13]. It is important to correct the underlying maternal abnormalities before intervening for the foetus as usually correction of the underlying metabolic abnormality will improve foetal status[14]. The presence of persistent foetal bradycardia or the development of category iii heart rate tracing that is unresponsive to treatment may require expedited delivery.

Acute diabetic complications during pregnancy

Diabetic ketoacidosis (DKA) is usually observed in patients with type 1 diabetes, but it can also complicate pregnancies with type 2 DM and gestational diabetes[15]. With prompt recognition and aggressive multidisciplinary management, the overall incidence has decreased from 10% to 20% in the late 1970s to 1 to 2% in most recent reports [3,16,17]. DKA occurs due to the lack of insulin resulting in a perceived hypoglycaemia at target cells such as those in liver, adipose, and muscle tissue. So, stores of glucagon are released, worsening the hyperglycaemia, and causing osmotic diuresis, hypovolemia, and electrolyte depletion. Adipose tissue secrets some counter regulatory hormones which causes the release of free fatty acids into the circulation. These free fatty acids are oxidized to ketone bodies and a metabolic acidosis ensues which manifests the anion gap. ketoacids bind sodium and potassium, and are excreted in urine, further worsening the electrolyte balance. In untreated cases, it can lead to cardiac dysfunction decreased tissue perfusion and worsened renal function leading to shock, and death[3,18].

Pregnancy increases the susceptibility to DKA. Insulin resistance primarily due to human placental lactogen causes insulin requirements to increase with advancing gestation. Respiratory adaptation during pregnancy end in a compensated maternal alkalosis. The compensatory decrease in serum bicarbonate reduces the body's normal buffering capacity, thus predisposing the patient to DKA[3,16,18]. Precipitating factors and signs/symptoms are given in tables 2 and 3.

DKA is a medical emergency and a multidisciplinary team, including maternal foetal medicine, endocrinology, pneumatology, an intensive care should be assembled. Treatment includes correction of significant fluid deficits and electrolyte abnormalities and insulin. Basic laboratory analyses, including a complete account, metabolic panel with magnesium and phosphorus, urine analysis, fingerstick blood glucose, arterial blood gas and

serum ketones should be done. Investigation criteria for diagnosis of DKA are given in table 4. Additional testing (urine culture, blood culture, chest x-ray etc.) should be performed based on clinical suspicion and any potential underlying processes. Serum ketones, electrolytes, and maternal acid/base status should be monitored every 2 hours until ketosis and acidosis are resolved. Blood sugar should be corrected hourly during this time to titrate insulin[5,16,18,19,20].

DKA presents a big risk to overall foetal wellbeing. The likely mechanism is related to maternal ketoacids which cross the placenta leading to decreased foetal tissue perfusion and oxygenation[18]. The foetus has a limited ability to buffer significant acidemia and therefore is quite sensitive to maternal acidosis. Once viability is confirmed, foetal monitoring should be done by foetal heart tracing. Maternal oxygen supplementation and left lateral positioning should be done to increase blood flow to foetus and improve oxygenation. Adequate hydration and correction of acid\base derangements must be started. Delivery is generally postponed until maternal metabolic condition is stabilized as this will usually correct the foetal heart tracing abnormalities. Table 5 illustrates a general algorithm for treatment of DKA in pregnancy.

Hyperparathyroidism in pregnancy

Primary hyperparathyroidism (PHP) is characterised by hypercalcemia with a raised or high normal parathormone (PTH). It is estimated that PHP occurs with an incidence of 1.4% of the overall general population but quite 80% of PHP cases remain asymptomatic in both the pregnant and non-pregnant patients. This disease is present in 0.5 to 1.4% pregnant women, 85% among them are due to parathyroid adenoma[21].

The increased calcium demand in pregnancy is fulfilled by increasing absorption in the bowels, kidneys, and a higher intake of this microelement[22]. Physiological changes of pregnancy can mask the diagnosis of PHP as hypoalbuminemia, increase glomerular filtration rate, calcium transport across the placenta, and oestrogen inhibition of PTH-mediated bone resorption can result in appearance of lower calcium levels[23]. From the 12th week the urinary excretion of the calcium rises and exceeds normal values. PTH levels are in low normal range in the first trimester and slowly increases to normal at the time of Labour[24]. The foetal calcium and phosphorus concentrations are higher than mothers[25]. Maternal PTH does not cross the placenta but foetal parathyroids start synthesizing it since 10th week of gestation. So, secretion of the foetus PTH can be regulated by mothers and its own calcium levels. So, suppression of foetal parathyroid hormone occur in case of hypercalcemia in mother and maternal hypocalcaemia leads to secondary hyperparathyroidism in neonates[24]. Thus, the foetal skeleton participates in the calcium-phosphorus balance.

Typical presentation of hyperparathyroidism is "bones, stones, abdominal moans and psychic groans", which explained by bony fractures, nephrolithiasis, gastritis, and mental status changes[26]. The symptoms include nausea, vomiting, anorexia, constipation, depression, and mental confusion. kidney stones, pancreatitis, abdominal pain as well as EKG changes including short QT interval and arrhythmia may be found. PHP is often associated with pancreatitis in the pregnant population due to elevated serum calcium levels resulting in damaged pancreatic ducts[27].

Increasing hypercalcemia was significantly associated with intra uterine fetal demise[28]. Nausea, dehydration, and mental status changes indicates the most life-threatening consequence of PHP which is hypercalcemia crises. It may occur during pregnancy but also after delivery when transport of calcium via placenta abruptly stops[29]. Foetal complications are seen in upto 80% of cases and fetal demise was reported in 27 to 31% of cases. Other adverse effects are intrauterine growth restriction and low birth weight[30]. After delivery when calcium supply is cut off, the neonate may experience tetany.

An increase in corrected or ionised calcium levels together with PTH above or high normal range in pregnant women is due to PHP but other possible reasons of hypercalcemia are need to be excluded[31]. Radiological localization of parathyroid disease limited in pregnancy as computed tomography and the gold standard 99 mTc Sestamibi scan are contraindicated, so ultrasound remains the only suitable imaging modality[32]. To increase the accuracy of the diagnosis an ultrasound guided fine needle aspiration biopsy of the lesion may be done.

Treatment includes conservative therapy like increase fluids and decrease calcium intake along with vitamin D supplement. Calcitonin does not cross the placenta but not generally effective. Bisphosphonates should be avoided unless absolutely necessary due to its side effects on the foetal bones. Calcimimetics has not been widely used during pregnancy and there are only 3 cases described in literature [33-35]. In two of them, cinacalcet was given for 2 weeks before delivery and in 3rd case the women suffered from parathyroid carcinoma and received cinacalcet throughout the pregnancy with no adverse effects. But the safety of cinacalcet is not documented .

Surgical removal of parathyroid gland is generally reserved for symptomatic hypercalcemia and preferred in the second trimester[36]. A review which compares surgery to other treatment methods had shown that the incidence of neonatal complications are less with surgical treatment[37]. Recently minimally invasive parathyroidectomy, having the same effectiveness as bilateral neck dissections, can be performed.

Majority of primary hyperparathyroidism cases (90%) occur sporadically due to parathyroid adenoma and remaining 10% cases as hereditary disorders[38]. Genetic testing in pregnant women with primary hyperparathyroidism should be considered as familial hyperparathyroidism are related to an earlier age of onset compared to sporadic disease[38].

Acute pituitary complications in pregnancy

Prolactinoma

Prolactinoma is characterised by autonomous production of prolactin due to a primary micro or macroadenoma. Estrogen can potentially increase the size of prolactinoma during pregnancy leading to pituitary mass effect, irreversible visual loss, and potential apoplexy. The risk of tumor enlargement during pregnancy has been reported to be as high as 35% in women with macro prolactinoma[39,40]. Periodic prolactin monitoring is of limited value during pregnancy due to wide variation in levels during pregnancy. Visual field testing is mandatory and pituitary imaging with MRI is needed in patients who develop symptoms of increased intracranial pressure and visual abnormalities.

If there is evidence of tumour enlargement, dopamine agonist therapy (i.e., bromocriptine or cabergoline) should be resumed immediately and continued throughout pregnancy. The evidences do not suggest any increased rates of abortion or congenital malformations with bromocriptine[41], while experience with cabergoline during pregnancy is more limited[42]. Pre-pregnancy trans-sphenoidal debulking or radiation therapy of a macroprolactinoma reduces the risk of significant tumour enlargement during pregnancy[39]. Surgery during first or second trimester is associated with an increased risk of foetal loss Surgery or delivery (in advanced pregnancy) should be attempted if there is lack of response to dopamine agonist or progression of visual deficits.

Sheehan's Syndrome

During pregnancy, the normal pituitary gland enlarges by about one-third, primarily from an increase in lactotrophs size, and number in response to elevated plasma estrogen. The blood supply to pituitary gland increases during pregnancy, so any interruption of blood flow to the anterior pituitary lobe will result in infarction.[43]. Hypotension from severe haemorrhage is the major causal factor [44,45].

The acute phase is associated with almost 90% of infarction of the pituitary gland. It is potentially lethal if not recognised and managed with glucocorticoid replacement immediately[46]. In case of obstetric haemorrhage, patient may complain of nausea, vomiting, vision impairment and sudden onset headache associated with an increase in intracranial pressure. Haemodynamic instability which is not responsive to fluid challenge may be there [39]. MRI usually reveals an enlarged low density seller mass with rim enhancement following contrast administration. Treatment with stress doses of glucocorticoids should be given as soon as the clinical diagnosis is made, and should not delay for the results of diagnostic testing[39]. Aggressive fluid resuscitations and blood replacement may minimize the pituitary necrosis.

Pituitary apoplexy

Pituitary apoplexy during pregnancy is an endocrine emergency with significant morbidity and mortality to mother and foetus. It occurs most commonly in patients with pituitary macroadenoma when infarction happens in tumor due to haemorrhage or ischemia or both. Pituitary apoplexy is defined as acute bleeding into pituitary gland which is a very rare entity although several cases have been reported in literature[47]. Pre-disposing factors include radiation therapy, anticoagulant therapy, diabetes mellitus, hypertension, use of bromocriptine and disseminated intravascular coagulopathy[47]. It presents as sudden onset of severe headache, vomiting, meningism and altered mental status due to increased intracranial pressure[48]. Ophthalmoplegia and cranial nerve palsy can occur if the enlarged pituitary gland extends laterally and compresses on the optic chiasm. A clinical diagnosis needs a high index of suspicious to ensure timely management. Treatment should be started immediately before imaging aiming at measures to maintain hemodynamic stability. Hydrocortisone should be given intramuscularly or intravenously (100mg at every 6 hrs) or as a continuous infusion (5-10 mg / hr after stabilization).

MRI with dynamic contrast of the pituitary shows high signal on T1 d T2-weighted images and provide information of the extent of tumor enlargement.

Expected medical management may be adequate in cases with minimal visual impairment. Transsphenoidal decompression surgery is needed if there is deterioration in conscious level or there are visual deficits. All patients should have an endocrinological evaluation for pituitary function at 4 to 8 weeks following the event. Long term followup is required to monitor the tumor growth and replace the missing hormones.

Table 1 : Burch and Wartofsky's clinical scoring for thyroid storm[9]

Criteria	Points
Thermoregulatory dysfunction	
Temperature (°C)	
37.2–37.7	5
37.8–38.3	10
38.4–38.8	15
38.9–39.4	20
39.4–39.9	25
≥ 40.0	30
Cardiovascular	
Tachycardia (beats per minute)	
100–109	5
110–119	10
120–129	15
13–139	20
≥ 140	25
Atrial fibrillation	
Absent	0
Present	10
Congestive heart failure	
Absent	0
Mild	5
Moderate	10
Severe	20
Gastrointestinal-hepatic dysfunction	
Manifestation	
Absent	0
Moderate (diarrhea, abdominal pain, nausea/vomiting)	10
Severe (jaundice)	15
Central nervous system disturbance	
Manifestation	
Absent	0
Mild (agitation)	10
Moderate (delirium, psychosis, extreme lethargy)	20
Severe (seizure, come)	30
Precipitating event	
Status	
Absent	0
Present	10
Total score	
> 45	Thyroid storm
25–45	Impending storm
< 25	Storm unlikely

Table 2 : Precipitating factors for DKA in pregnancy

Precipitating factors for diabetic ketoacidosis in pregnancy	
• Protracted vomiting	• Hyperemesis gravidarum
• Infections	• Insulin non-compliance
• Insulin pump failure	• β-sympathomimetic tocolytic agents
• Conditions such as gastroparesis	
• Medications precipitating diabetic ketoacidosis in pregnancy.	

Table 3: Signs/symptoms suggesting DKA in pregnancy

Signs and symptoms	
• Nausea or vomiting	• Abdominal pain
• Polyuria or polydipsia	• Blurred vision
• Muscle weakness	• Drowsiness Lethargy
• Changes in mental status	• Tachypnoea
• Hypotension	• Tachycardia
• Hyperventilation (Kussmaul breathing)/pear drop odour	
• Coma	• Shock
• Abnormal fetal heart tracing	

Table 4 : Investigation criteria for diagnosis of DKA

Positive serum/urine ketones
Lab glucose hyperglycaemia (e" 11.0 mmol), but DKP can occur at lower glucose levels.
Low serum bicarbonate (<15mEq/l)
Elevated base deficit ≥ 4 mEq/l
Potassium level may be falsely normal/elevated.
Arterial pH ≤ 7.30 Anion gap >12

Table 5 : Illustrates a general algorithum for treatment of DKA in pregnancy

	Treatment Modality	PLAN
MATERNAL	Identify cause	H&P, rule out infection, place foley catheter, serial vital signs, I/0's Consider ICU admission Consult critical care, endocrinology, maternal fetal medicine
	Fluid replacement (estimated ~100 mL/kg)	Correct 75% total deficit in first 24 h Begin with 0.9% normal saline Convert to D5-0.45% normal saline when FSBG < 250
	Insulin administration Goal FSBG 150-200 in DKA	Regular insulin via IV IV bolus 0.1units/kg followed by 0.1 u/kg/h continuous infusion Goal reduction 20%-25% over 2 h (if not increase IV infusion 1.5-2×) Continue IV insulin until acidosis and ketosis resolves Start SQ insulin therapy 1-2 h before stopping IV insulin
	Laboratory evaluation	CMP/Mg and Phos, pH, serum ketones every 2-4 h initially Replete K+ once 7.0
FOETAL	Monitoring Fetal optimization	Consult maternal fetal medicine Initiate fetal monitoring if viability achieved Maternal left lateral decubitus, maternal O2 supplementation, stabilize maternal condition before delivery

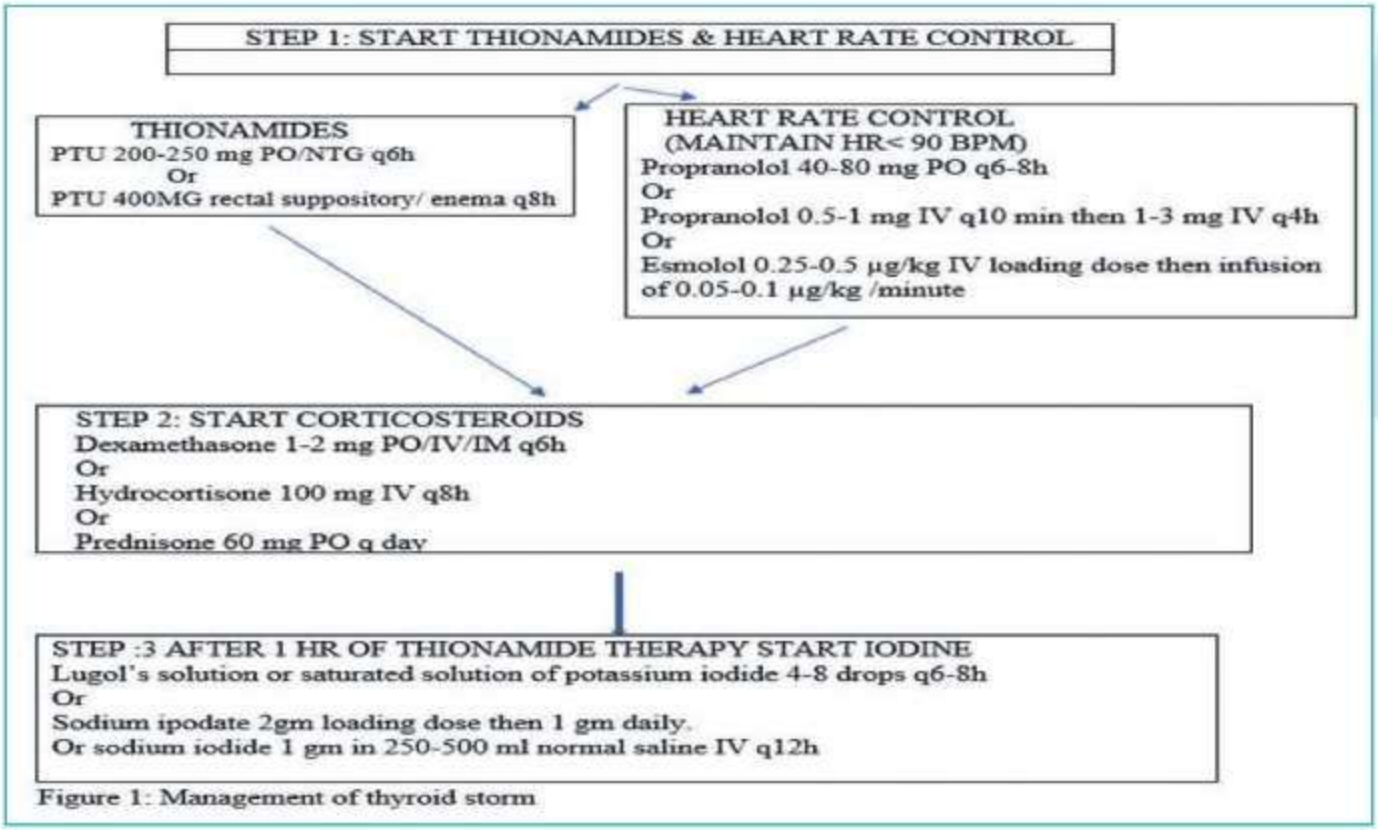

Figure 1: Management of thyroid storm

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Anesthetic Near Miss Situation in Obstetrics

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The maternal health is always a priority for the practicing obstetricians. But the shortcomings in managing the preventable errors in obstetric patients goes a long way in affecting the maternal outcomes. Each obstetric patient receiving anesthesia should have risk stratification before induction so that maternal outcomes can be improved.[1]

Anesthetic near miss situation is defined as any preventable event or error associated with the administration of either general or regional anesthesia and which may have led to an undesirable patient outcome.[2]

The anesthesia near miss situation is influenced by the type of surgery, patient profile or the anesthesia itself.3 Besides, the anesthesia related errors there may be due to human shortcomings, equipment failure or due to drug interactions.

During 1970s and 1980s, the anesthesia had been the major risk factor for maternal mortality and morbidity during caesarean section.4&5 In 1990s, the patients who had LSCS under general anesthesia had higher risk of maternal morbidity and mortality than those patients who had regional anesthesia. The major causes of compromise because of anesthesia includes:

- Maternal hypoxia secondary to failed or difficult intubation,
- Bradycardia or tachycardia i.e. more than 30% deviation from the baseline
- Hypothermia
- Aspiration and hypoventilation leading to respiratory problems and desaturation.[6]

WHO "Near Miss" Criteria[7&8]	
Organ system dysfunction	**Marker for organ dysfunction**
Cardiovascular dysfunction	1. shock and cardiac arrest. 2. use of continuous inotrophs, 3. severe hypoperfusion and severe acidosis
Respiratory dysfunction	1. acute cyanosis, and gasping, 2. severe tachypnea and severe bradypnea 3. intubation and ventilation not related to anesthesia, 4. severe hypoxemia
Renal dysfunction	1. un -responsive oliguria. 2. dialysis for acute renal failure 3. severe acute azotemia
Coagulation / hematological dysfunction	1. failure to form clots 2. massive transfusion of blood or red cells (e"5 units) 3. severe acute thrombocytopenia (<50 000 platelets/ml)
Hepatic dysfunction	1. Jaundice in the presence of pre eclampsia
	2. severe acute hyperbilirubinemia (bilirubin >100 ìmol/l or >6.0 mg/dl)
Neurological dysfunction	1. prolonged unconsciousness, 2. stroke and total paralysis 3. uncontrollable fits/status epilepticus

Nowadays there has been a great improvement in the monitoring and anesthesia devices, as a result there has been a marked reduction in the airways complications during general anesthesia in pregnant women.[9&10]

Case 1

A 31 years old, G2P1A0 with previous LSCS, term pregnancy admitted for elective caesarean section. This patient was a diagnosed case of bronchial asthma for last 1 month on medication. She had LSCS under spinal anesthesia. As soon as the baby was delivered, patient developed severe hypotension not responding to IV fluids, blood volume expander, and mephentine. Patient was intubated, put on intermittent positive pressure respiration and steroids. Since, she was not maintaining her BP and oxygen saturation, she was shifted to ICU. She stabilized in 8-10 hours, but her tachycardia persisted. She was put on venti mask from BIPAP after 12 hours. Patient was put back on BIPAP support as her oxygen saturation fell to 85%, she was dyspneic and her CVP was 22cms.

ECG was done and showed nonspecific ST changes. Echo showed generalized hypokinesia. LVEF 30% severe MR, TR with PAH. She was diagnosed as a case of peripartum cardiomyopathy. She responded to ACE inhibitors, digoxin, antibiotics, low molecular weight heparin and Lasix and was discharged on 10th post op day in stable condition. She still has LVEF of 30-35% even after eleven years, and continues to be on treatment for cardiomyopathy.

The lesson was, if an antenatal patient complaint of breathlessness; better to rule out cardiomyopathy rather than thinking her as a case of bronchial asthma which had developed during pregnancy. With this in mind, we subsequently diagnosed a series of eight cases of peripartum cardiomyopathy antenatally and managed successfully. The high index of suspicion in all cases helped us in peripartum managed of these patients and successful results.

The outcome due to anesthesia is definitely influenced by the patient profile to begin with. So risk stratification before giving anesthesia is important. The leading cause of anesthetic near miss in obstetrics is:

1. Patient profile
2. Surgical factor (which surgery being done – cesarean hysterectomy)
3. Human error
4. Equipment failure
5. Drug-interaction

The patient profile includes:

- LSCS done due to obstetric hemorrhage- APH and abnormal placentation (placenta previa, placenta accrete or placenta percreta) uterine rupture, uterine leiomyoma, thrombocytopenia or coagulopathy.
- Hypertensive disorders of pregnancy.
- Sepsis
- Obesity
- Conversion of regional to general anesthesia
- Age - especially more than 40 years, nowadays due to ART techniques, more patients above the age of 40 years are getting pregnant, and their deliveries fall into high-risk category.
- Baseline medical co-morbidities like anemia, cardiac problems, renal problems, diabetes or jaundice.
- Delays in seeking, reaching and receiving quality care.
- The general anesthesia may itself pre-dispose to increased incidence of hemorrhage, impaired platelet function and uterine atony.

The main responsibility of an anesthetist is to take care of the anesthetized patients during surgery. There should be presence of a responsible anesthetist throughout the surgery. During the entire period of anesthesia, the patients' oxygenation, ventilation, circulation and temperature should be continuously monitored.[11]

The operation theatre is a critical place where vulnerable incidents do occur. During induction of anesthesia and during the maintenance many

untoward incidents can occur.12 In general anesthesia; both intubation as well as extubation are crucial events.

The critical incidents that may occur are due to:

- Airway management.
- Cardiovascular causes due to hypotension and bradycardia.
- Desaturation due to respiratory problems like endotracheal tube into one side of trachea into bronchus etc.
- Hypotension due to uncorrected dehydration. The patients are sometimes into prolonged dehydration as in prolonged labor. These patients should be adequately hydrated before inducing any kind of anesthesia.
- The drugs used for induction of anesthesia like sodium thiopentone and propofol are known to cause hypotension worsened in the dehydrated state.
- Drug errors are also a frequent cause of untoward incidents especially when diluting the drug is required.
- Sometimes blood transfusion errors occur, when massive blood transfusion is necessary during surgery.
- Un-interrupted power supply is essential to ensure gadgets and equipments to function during power failure. So the facility should arrange for an un-interrupted power supply.

There may be error of judgments, lack of experience and skill, in- attention to details, failure to check as well fatigue that may contribute in human errors in management of these high-risk patients.[13]

Therefore, consultants' inputs and supervision of senior professionals goes a long way in preventing these human errors. Where-ever necessary two or more senior consultants should be involved to tide over a high-risk situation and give a safe landing to the patients.

Case 2

A young booked Primigravida 29 year goes for an emergency LSCS for fetal distress. The spinal anesthesia was tried four to five times yet failed, so she was given general anesthesia. On third post-operative day she developed weakness of right leg. The obstetrician on rounds did SLR test and found positive and gave reference to anesthetist (thinking it to be due to failed spinal anesthesia) and orthopedic surgeon. MRI spine was done and it showed a huge spinal tumor. Neurosurgeons evaluated her and operated her (decompression) on sixth post-operative day. So, wisdom prevailed in this case together with multidisciplinary approach with right judgment saved the limbs of this young mother. Otherwise, it would have passed off as a case of sequalae of the spinal anesthesia and the patients would have permanent damage of the power of her lower limbs.

Drug interactions: Sometimes the patients and the relatives forget to tell the treating obstetricians and the anesthetist about the drug allergies. As a result, the patient may suffer severe anaphylaxis.

Case 3

An incident relating to a couple - both anesthetists, is worth documenting. The wife was to undergo LSCS. Both the husband and the wife forgot to tell the obstetricians and the anesthetist about the allergy to paracetamol. During LSCS, she was given paracetamol (perfalgun) infusion. When the entire infusion was gone she became red all over the body. Since, she was in Operating Room quick action was taken and she was saved.

Similarly, sometimes the patients and their relatives forget to tell about the drugs that the patient is taking e.g. steroids, anti-platelets, pain-killers or anti-epileptics. All these drugs may produce drug interactions.

In CVS dysfunction, methergin is to be avoided, and direct acting inotropes like phenyl-ephrin are used and other inotropes are avoided if possible.

In respiratory dysfunction e.g. asthma- drugs like carboprost, thiopentone, succinylcholine and NSAIDS are to be avoided. Only paracetamol may be used as a pain-killer.

In liver and renal dysfunction- short acting muscle relaxants and auto-degrading muscle relaxants are used .e.g. atracurine, cis-atracurine. Even short acting opioids like fentanyl or rami-fentanyl are used.

In neurological disorders- preferably regional anesthesia is avoided. Better to use general anesthesia. MRI in third trimester is a preferred protocol in case patient is a known case of epilepsy or other neurological disorder. Again, short acting drugs are preferred in these cases.

For difficult intubations, in an emergency there is a role of laryngeal mask airway (LMA) or better still Pro-seal LMA. The other options in difficult intubations are Bougies. The gold standard is to use fiber-optic Bronchoscope, especially in planned or an elective LSCS where we are anticipating problems. The use of Video-laryngoscope is also available in some centers to assist in difficult intubations.

Post-operative period

It is important to be vigilant in post-operative period, the common causes of morbidity in antenatal period also continues in the post-operative period as well.

- The errors of monitoring are the commonest cause of failure.
- Equipment failure can be an important cause.
- Massive hemorrhage due to uterine atony and ruptured uterus or abnormal placentation.
- Hypertension was another leading cause of near miss and mortality e.g. pre eclampsia with HELLP syndrome.
- Sepsis is also a preventable cause of maternal near miss in post operative period.
- Anemia and other medical co-morbid conditions have to be addressed as well.

Conclusions

The health of the mother and child are always a priority. The operation theatres are areas where most vigilance is required. The preventable errors in operating room goes a long way in patient outcomes. The patient profile, the type of surgery, underlying medical co-morbities are to be judged and managed accordingly.

Key points

- Before any kind of anesthesia, the risk stratification of the patients should always be done.
- Preparedness, prevention and performance are important.
- Senior inputs and presence of senior anesthetist in high-risk cases is a useful decision.
- Timely action, alertness during anesthesia and anticipation of problems do give better results.

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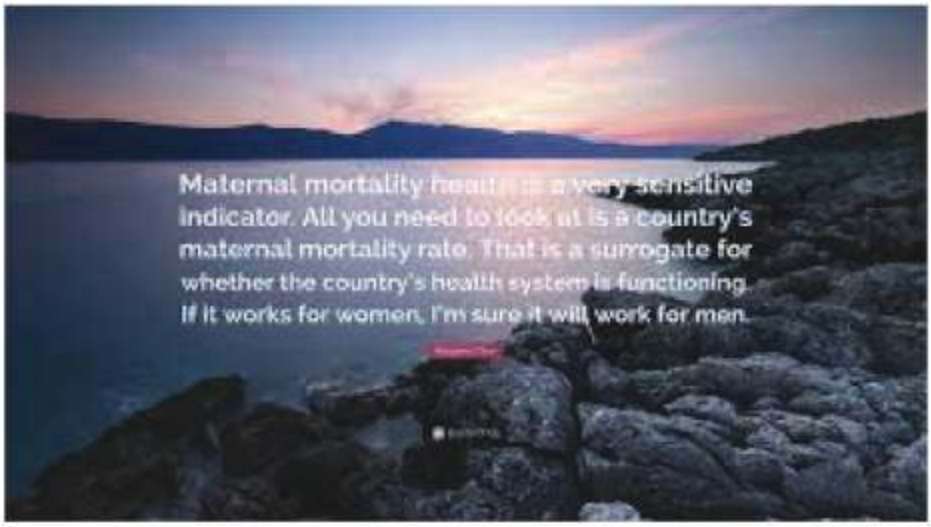

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Caesarean Hysterectomy

Introduction

Peripartum hysterectomy is a life-saving procedure performed in the setting of acute, life threatening haemorrhage when all conservative measures to achieve haemostasis have failed. The major challenges of the procedure are :

1. Unplanned nature of the surgery and the need to perform it with expedite.
2. Owing to the already acute loss of blood, the patient is already not in an ideal condition to undergo a major surgical intervention.

Definition

Peripartum hysterectomy can be defined as a hysterectomy performed at the time, or within 24 hours, of delivery.

Another definition is: A hysterectomy performed any time from delivery to discharge from the same hospitalization.

History and Background

The first hysterectomy documented in the history of obstetrics was on a patient at caesarean section, performed in 1869 in the United States by Horatio Storer. But although the uterus was removed completely, the patient died of complications after 68 hours of surgery.

Eduardo Porro, Head of midwifery school in Milan, in 1876, described the first caesarean hysterectomy in which both the mother and baby survived. The Porro Procedure: He documented his case in detail in a publication as Julia Cavallani, who presented to his clinic with extreme malformed pelvis in relation to a past history of rickets in childhood. After caesarean delivery, in view of intractable bleeding, the decision for hysterectomy was taken. A wire snare of an instrument called the cintrat was passed around the neck of uterus at the level of internal os and sufficiently tightened to control haemorrhage. The uterus was then excised above this ligature. The uterine stump was then brought out through the abdominal wound and closed with sutures of silver wire.

Various modifications to the original Porro's procedure were made by Richardson in the United States, Godson and Tait in Great Britain in 1890, who recommended exteriorization of uterus and suturing the cervical stump to the abdominal wall.

Indications and Risk factors

1) Atonic PPH (last resort after all other methods of management fail)
2) Rupture uterus
3) Placenta accrete, increta, percreta
4) Extension of caesarean section incision into uterine vessels
5) Chorioamnionitis with sepsis

Based on population-level data, the peripartum hysterectomy rate is nearly 1 per 1000 deliveries [1].

Significant risk factors include: [2-7]

a) Abnormal placentation
b) Advanced maternal age
c) Multiparity
d) Multiple gestations
e) Antepartum bleeding
f) Preeclampsia
g) Bleeding disorders
h) Use of assisted reproductive technologies[2-7].

Secondary analysis of a large, multi-centre

postpartum haemorrhage trial reported that abnormal placentation was the most common cause of hysterectomy and that advanced maternal age and delivery by caesarean section were significant risk factors [8].

Emergency vs Elective peripartum hysterectomy

The procedure may be planned or performed in an emergency situation. Severe uterine haemorrhage that cannot be brought under control by conservative methods is the most common indication for emergency procedures. Such haemorrhage is most commonly due to abnormal placentation or uterine atony and each account for 30 to 50 percent of peripartum hysterectomies[9-12]. Other potential causes include uterine rupture, leiomyomas, and laceration of uterine vessels[4,13]. Elective peripartum hysterectomy is performed as a planned procedure in patients with an antepartum diagnosis of placenta accreta, or more rarely for stage IA2 and IB1 cervical cancer or very large fibroids[14,15]. Infection appears to be an important contributor to peripartum hysterectomy. Not only is severe postpartum pelvic infection a potential indication for the procedure, but uteri removed for atony also show a relatively high rate of infection and inflammation on pathologic analysis[14].

Surgical anticipation and preparation

Sometimes the obstetrician can anticipate the possible need for peripartum hysterectomy based on the patient's risk factors. This enables patient preparation and counselling in the antenatal period, detailed surgical planning, and possibly avoidance of an emergency procedure. This is true primarily for women with abnormal placentation. Most patients with placenta accreta, increta, or percreta will undergo hysterectomy at delivery (79 of 133 patients [60 percent] in one study[16]). Placenta previa is associated with an approximately 5 percent risk of hysterectomy, usually in cases with placenta accreta[17]. The frequency of abnormal placentation rises substantially as the number of prior caesarean deliveries increases, as well as with maternal age [18].

Even in the absence of abnormal placentation, caesarean delivery, as well as prior uterine surgery, appears to be a risk factor for peripartum hysterectomy[2,3,19]. In a population-based, case-control study, the risk of peripartum hysterectomy was lowest in women undergoing a first delivery that was vaginal (1 in 30,000) and highest in women with a history of two or more prior caesarean deliveries (1 in 220) [20]. In a study of 30,000 women undergoing caesarean delivery, the risk of peripartum hysterectomy was <1 percent for the first, second, or third caesareans; 2 to 4 percent for the fourth and fifth procedures; and 9 percent after six or more caesareans[19]. While abnormal placentation contributes to hysterectomy risk with repeat caesareans, it is unclear whether the increased risk with primary caesarean delivery relates to the surgery itself, or reflects the indication for caesarean. Indications such as abruption, infection, macrosomia, or multiple gestations are risk factors themselves for haemorrhage and/or uterine atony.

Surgical procedure:

The surgical pitfalls of the procedure are:

1. Enlarged uterus
2. Thick, vascular pedicles

   The physiologic changes of pregnancy in the maternal pelvis are responsible for the major surgical challenges associated with the procedure. The uterus is five times enlarged and veins are extremely engorged and tortuous. Collateral vessels have emerged.
3. Pelvic tissues are oedematous, fragile and friable- risk of improper knot placement, cut through and knot slippage. Meticulous care is required in the manipulation of clamps, cutting of pedicles and placement of sutures to prevent severe bleeding.
4. Due to fully dilated cervix after normal delivery, the margins of the cervix and vagina are difficult to identify
5. Separation of bladder maybe difficult due to oedema

6. In the setting of advanced stages of labor, the vaginal tissues are extremely necrotic and devitalised and get cut through when sutured.
7. Oedema of the structures surrounding the uterus allows easy dissection of surgical planes but produces large pedicles from which blood vessels may escape and get separate from uterus.
8. In the setting of uterine trauma or rupture, haematomas of the broad ligament and neighbouring structures make visualization difficult and distort anatomic relations. Traumatised tissues at the base of pelvis may continue to bleed even after surgery despite proper ligation of obvious bleeding pedicles.

Total vs Subtotal Peripartum Hysterectomy

Peripartum hysterectomy can either be performed in the more traditional way of total abdominal hysterectomy (TAH) or as subtotal abdominal hysterectomy, depending on the patient's condition, the indication for peripartum hysterectomy, the surgeon's experience and the initial risk assessment for the probability of complications during and after the operation. The latter requires a less complex surgical technique, reduces operating time, diminishes blood loss/need for blood transfusion and lowers intra- and postoperative complication rates especially injury to the bladder and ureter, with the added fact that the cervix may not be easily differentiated especially if the patient has been in labour for a long time and whose cervix is soft, friable, fully dilated and effaced. But total abdominal hysterectomy is required for haemostasis in cases of placenta previa, increta, accrete, percreta, where the entire placenta and placental bed has to be removed, and if the lower segment, cervix is involved in the trauma and haemorrhage.

In case of therapy refractory severe postpartum haemorrhage any delay performing emergency peripartum hysterectomy significantly boosts the probability of DIC and as a consequence, the mortality-risk in the patient. EPH is to be performed before full-blown coagulopathy is established. When PH is inevitably required, clear decision making, involvement of an experienced obstetrician and prompt surgery goes along with a lower blood loss/transfusion requirement and an overall better patient outcome[21].

Surgical principles of Peripartum Hysterectomy

Basic surgical principles and tips of peripartum hysterectomy have been developed that help to proceed with the surgery safely and reduce maternal morbidity and mortality associated with the procedure.

1. A midline vertical abdominal incision is made: Provides best exposure
2. Uterus is brought out of the abdominal incision: Exteriorization of uterus is important to clamp and ligate under direct vision.
3. Clamp, cut and drop technique of Mickel and Plauche
4. All pedicles are ligated as close to the uterus and cervix as possible- Sliding off technique
5. Adequate size of the stump should be kept.
6. Round ligament with the Sampson artery should be ligated separately
7. Cornual and vascular pedicles: Extreme meticulous care is to be taken and double ligation is done- proximal simple ligation and distal transfixation. Clamps on the vascular pedicles should be manipulated as little as possible due to the danger of trauma and excessive bleeding. If the uterine clamp slips and suture is blindly applied in the bleeding area, the ureters can be accidently clamped and sutured where they run under the uterine vessels at the lateral aspects of the lower segment.
8. Bladder dissection: Remain in the midline while dissecting the bladder as laterally, the dilated plexuses of Sanorini may bleed. Adhesions of bladder with the lower uterine segment require sharp dissection. It is important to keep bladder safety in mind as bladder wall is oedematous

and needs to be protected with a mop kept between the bladder and doyens retractor.

9. Define the lower limit of cervix by making an incision in the lower segment or check through the LSCS incision.
10. The uterosacrals should always be excised and sutured as separate pedicles.
11. When tying the last cardinal ligament, it is important to take the angle of friable and oedematous vagina in it to prevent angle bleeding.
12. Before closure: Complete haemostasis should be checked for by inspecting all pedicles carefully. No peritonization of pedicles should be done. Drains maybe placed: intra- abdominal 14 no. drain and superficial drain. Mops, instruments, needles count should be noted.

Complications of peripartum hysterectomy

The principal complications after peripartum hysterectomy are febrile morbidity, haemorrhage, urinary tract injury, coagulopathy, paralytic ileus or bowel obstruction, and reoperation. Emergency procedures are associated with a higher rate of complications than planned procedures[22]. Mortality should be <1 percent, though a global summary of emergency cases reported mortality rates ranging from 0 to 59 percent, with a composite rate of 5.2 percent[4,23]. The findings from several large series of peripartum hysterectomy are illustrated below:

A prospective series by the Maternal-Fetal Medicine Units (MFMU) Network included 186 caesarean hysterectomies performed in 1999 and 2000 and reported the following complications and their frequencies: red blood cell transfusion (84 percent), transfusion of other blood products (34 percent), fever (11 percent), ileus (5 percent), exploratory laparotomy (4 percent), hospital readmission (4 percent), urinary tract infection (3 percent), cuff abscess (2.7 percent), maternal death (1.6 percent), bowel injury (1 percent), wound dehiscence (1 percent), pelvic or deep vein thrombosis (1 percent)[24].

Data from the Nationwide Inpatient Sample (a random sample of 20 percent of hospital discharges in the United States) from 1998 to 2007 showed the following types and rates of complications in 4967 peripartum hysterectomies: transfusion (46 percent), infection (12 percent), wound complication (10 percent), bladder injury (9 percent), reoperation (4 percent), venous thromboembolism (1 percent); and intestinal, ureteral, or vascular injury (≤1 percent)[25]. Transfusion, infection, urinary tract injury, and reoperation were much more frequent than in non-obstetric hysterectomy.

A review of six studies of peripartum hysterectomy reported the following ranges of complications: febrile morbidity (11 to 34 percent), cystotomy (6 to 29 percent), ureteral injury (2 to 7 percent), oophorectomy (6 percent), reoperation (4 to 33 percent), thromboembolism (1 to 4 percent), death (0 to 4.2 percent)[26].

Post-operative care

Routine post-hysterectomy and postpartum care is appropriate if the patient is stable. Patient-controlled analgesia (either intravenous or epidural) is preferable until she can take oral medicine. Wound care is conducted according to the surgeon's preference. If the operation was clean contaminated, no postoperative antibiotics are indicated. Active infection should be treated appropriately. Women undergoing peripartum hysterectomy are at moderate risk or high risk of postoperative thromboembolic disease, depending on individual risk factors; therefore, both mechanical and pharmacologic prophylaxis for deep venous thrombosis are suggested. Pharmacologic prophylaxis is initiated at least four hours postoperatively; timing depends on patient-specific factors in the balance between risk of bleeding and risk of venous thrombosis, and whether a neuraxial catheter is left in place after surgery is completed. Prophylaxis is discontinued when the patient is discharged, but may be prolonged in selected women at highest risk of venous thromboembolism, such as those who have had a previous thromboembolic event.

Bladder care : If urinary stents were placed, they can be removed immediately postoperatively. The bladder catheter can also be removed within 24 hours postoperatively in stable patients who do not require monitoring hourly urinary output. However, if a cystotomy was repaired, then drainage is generally continued for 5 to 10 days. A large repair (>2 cm) should be evaluated with a cystourethrogram prior to discontinuing the catheter.

Breastfeeding : Haemorrhage and hysterectomy are not contraindications to breastfeeding. Patients admitted to the intensive care unit may use a breast pump once they are extubated and stabilized. Early pumping is encouraged when the new born is admitted to the neonatal intensive care unit. Breast milk output should be considered in the patient's fluid management, although this is usually not a concern by the time her milk supply develops. In the setting of a physically or psychologically difficult recovery, patients who are unwilling or unable to breastfeed should be supported.

Conclusion

Peripartum hysterectomy is associated with high mortality and morbidity despite advances in medicine and surgery. It is most effective in cases of persistent uterine atony, morbidly adherent placenta and rupture uterus. Time is of utmost importance as any delay in taking a decision of carrying out the procedure and performing the procedure itself can be dangerous to the patient. Prompt restoration of circulating volume in the setting of good ICU backup, perfect counselling of the relatives by a senior, experienced obstetrician can be of great help in carrying out this majorly morbid procedure smoothly. This said, antenatal anticipation, diagnosis of abnormal placentation and elective peripartum hysterectomy as a planned procedure is associated with much less complications.

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Rupture Uterus - An Obstetrician's Nightmare

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Background

Uterine rupture is an acute obstetric emergency, which accompanies severe maternal and fetal risk of morbidity and mortality. **Rupture uterus can be defined as a** complete separation of the wall of the pregnant uterus, with or without expulsion of the fetus, involving rupture of membranes at the site of the uterine rupture or extension into uterine muscle separate from any previous scar, and endangering the life of the mother or fetus.[1]

Uterine Rupture can spontaneously in an unscarred uterus in multiparas but is more likely to occur in women with previous uterine surgery like caesarean section, hysterotomy or myomectomy. Commonly occurs in labour but can occur in late pregnancy.

Labour in women attempting VBAC is increasingly common and a single cesarean scar increases the overall rupture rate to 0.5%, with the rate for women with 2 or more cesarean scars increasing to 2%, dependent upon whether labour was spontaneous or induced.[2]

1) Traditional classification	
• Complete/ true rupture	The visceral peritoneum overlying the uterus is disrupted along with uterine myometrial layers
• Incomplete / dehiscence / uterine window	Overlying peritoneum is intact, only uterine myometrial layer is disrupted
• Not clinical relevant	
2) Etiological classification	
Spontaneous rupture	
Scar rupture	
Traumatic rupture	
Surgical intervention Internal	Version Forceps delivery
	Manual removal of placenta
	Destructive operations
Medical intervention	Uterine stimulation

Women with normal, intact uteri are at the lowest risk for uterine rupture (1 in 8,434 -22,000 pregnancies [0.012%])[3,4]. Rupture in unscarred uterus has higher major maternal and neonatal morbidity and occurs in resource-limited countries like India where prompt management of obstructed labor may not be available.

Risk factors identified for uterine rupture for women with a previous caesarean section or scar due to myomectomy is the number of previous caesarean deliveries, shorter interpregnancy interval and labour induction and/or augmentation. Uterine perforation at the time of hysteroscopy, coil insertion or evacuation of retained products also increases the risk to a similar extent.

The associated risk factors for a Spontaneous Rupture occurring in an unscarred Uterus are grand multiparity, injudicious oxytocin administration, undiagnosed cephalopelvic disproportion or malpresentation, macrosomic baby, placenta percreta (morbidly adherent placenta), during version or uterine abnormalities. Traumatic ruptures occur due to internal obstetric manipulations and forceps delivery.[5-7]

Uterine rupture prediction can be done antenatally. Those with a high predicted caesarean section risk also had a higher risk of uterine rupture. A scoring system based on factors identified at the first antenatal visit was developed incorporating number of caesarean sections, interpregnancy interval and maternal age and showed low risk of rupture for those with low scores.[8]

Prediction of rupture during labour can be by use of a simple tool such as the partograph, zone 2–3 hours after the alert line in women undergoing trial of labour following caesarean section represents a time

of high risk of rupture.

Ultrasound measurement of both the thickness of the residual myometrium in the lower uterine segment and the width, depth, and length of the hypoechoic uterine defect at the site of the previous cesarean is the method most studied to predict risk of rupture. Antepartum imaging of the uterine scar in the third trimester is a useful tool and a value of 3.5 mm has been found to carry a significant negative predictive value (99.3%)[9]

Although sonographic lower uterine segment thickness near term is inversely correlated with the risk of uterine scar dehiscence or rupture, no myometrial thickness threshold value performed well enough to use in clinical practice to predict rupture.

Inter pregnancy imaging of the hysterotomy scar, showing U or V wedge-shaped hypoechoic uterine defects (also called niches) have been observed several months after delivery. No guidelines are available for management of these defects, as available data are limited by small numbers but hysteroscopic management is most commonly used. Management of these women should be decided on a case-by-case basis depending on symptoms.

Clinical manifestations of uterine rupture will depend upon the time of rupture and type of rupture. Uterine rupture can occur in antepartum, intrapartum or postpartum period. It can also be recognised in the Intraoperative time.

In a labouring women, the signs of rupture are an abnormal fetal heart rate (FHR) in (55–87%) or sudden development of category II or III FHR patterns. The most common FHR abnormality in rupture is fetal bradycardia. Category III FHR tracings may occur in the hour preceding diagnosis of rupture.[10]

Abdominal pain is of sudden onset, can come after after previously effective neuraxial anesthesia may be a sign of uterine rupture. Vaginal bleeding may occur but can be mild or even absent despite a major intra-abdominal hemorrhage after complete rupture. Hematuria is a rare sign and extension to the bladder dome causes it.

In most cases diagnosis is clinical but where ultrasonography is available, it is probably the safest and most useful imaging technique during pregnancy. The sonographic findings in rupture include extra peritoneal hematoma, intrauterine blood, free peritoneal blood, an empty uterus with whole gestational sac with fetus above the uterus or a large uterus mass with gas bubbles. Myometrial thickness of greater than 4.5 mm has negative predictive value of 100% as reported by a study, but the positive predictive value of thickness less than 3.5 mm is poor at only 11.8%.[11]

The symptoms of rupture in postpartum period are pain and persistent vaginal bleeding despite the use of uterotonic agents, hematuria may occur if the rupture extends into the bladder.

The intraoperative findings in suspected case of rupture are quite evident with presence of hemoperitoneum, fetal parts or membranes visible.

Management of Ruptured Uterus is outlined in a stepwise manner.[12]

a. Dependent on the degree of maternal shock and fetal condition

b. Should be supportive following the ABC approach:
   - AIRWAY: check whether airway is open, give 100% oxygen, consider intubation if patient is unconscious
   - BREATHING: check breathing and oxygen, if intubated ventilate with 100% oxygen
   - CIRCULATION: check for a pulse, insert 2 large bore IV cannulas (16G or above) take blood for cross match (4 to 6 units), FBC, U&E, LFTs & clotting screen. Administer rapid IV fluids such as hartmans or volplex in an initial bolus of 20ml/kg. Administer further fluids, blood and blood products as required.

c. Preparation for immediate delivery or laparotomy if already delivered.

d. Surgical repair of damage (including bladder trauma) – ultimately hysterectomy may be necessary – decision made by Consultant Obstetrician and will be dependent on site and severity of the rupture, the extent of the

bleeding and the ease of control. If the surgeon thinks that the patient is heading towards peripartum hysterectomy to request a colleague to come in to be actively involved in theatre in the management of peripartum hysterectomy earlier rather than later.

e. Intraoperative and postoperative antibiotic therapy should be given as per protocol.

f. Foley's catheter to remain in situ as indicated by surgeon/ hourly measurement with urometer initially.

g. Replacement of fluid loss as per haematological requirements.

h. Monitor vital signs as maternal condition dictates to include: blood pressure, pulse (attach pulse oximeter) respirations and temperature; to be recorded on the MEOWS chart. These observations should be undertaken every 5 minutes initially. Observe PV blood loss.

i. Assess the airway and apply high flow oxygen 15 litres per minute via a reservoir face mask.

j. Consider CVP – discussion with anaesthetic team to decide if needs transfer to ITU / HDU.

k. Check coagulation status and renal function.

l. Commence an intravenous infusion of 1 litre of Hartmanns solution, titrate as necessary depending on cause for collapse (i.e. give rapidly for hypovolaemic / hypotensive patient but with caution in cases with raised blood pressure and suspected heart failure)

m. Assess neurological status using CAVPU score or Glasgow coma scale if able

n. Treat peri-arrest arrythmias

o. If the baby is alive, the head fully engaged and the cervix fully dilated, instrumental delivery may be carried out.

p. Document events on maternal collapse proforma and in the maternal notes. Counsel the patient, family and staff.

q. The risk event form and a 24 hour serious incident (SIRI) report should be completed.

CESDI made recommendations for the management of patients with a previous uterine scar. They reiterated that all staff involved in intrapartum care must be aware of the factors that can lead to uterine rupture.

Apart from above outlined management, the coexistent complications of uterine rupture like concomitant uterine atony, bladder or a ureteral injury, injuries to blood vessels and other pelvic organs, placenta accreta spectrum needs careful evaluation and management

The severe life-threatening complications of uterine rupture are Massive Obstetric haemorrhage/ DIC, Hysterectomy, Injury to baby/HIE, Damage to bladder/ ureter. In the later post-operative time Ileus, infection, VTE can also complicate.[13] In outcome, death from uterine rupture is not uncommon. Mortality appears to be higher in women who have an unscarred uterus and when the rupture occurs outside the hospital and time to reach hospital is delayed. Although rare, death for the mother reported (1 death per 500 uterine ruptures) whereas developing countries maternal mortality reported is as high as 4.2%. perinatal death (5 to 26%) or neurologic morbidity for the fetus/neonate has been reported.

During the trial of a cesarean scar, obstetricians need to watch out for any fetal heart abnormalities, maternal tachycardia, vague abdominal pain in between contractions, suprapubic tenderness, vaginal bleeding and bladder tenesmus which would give warning for impending uterine rupture.

The risk of recurrence of uterine rupture varies (range 0 to 40 percent). The risk of rupture is highest when the previous rupture was in the fundus or longitudinal. A previous rupture that occurred during labor, at term, and in the lower uterine segment, a scheduled cesarean delivery at 36+0 to 37+6 weeks is the preferred approach.

Uterine dehiscence is a clinically occult and incomplete disruption of uterine myometrium. There are no serious maternal or neonatal consequences associated with it. Most are incidentally identified at repeat cesarean delivery, some detected during prenatal ultrasound examination[14]. It is managed at cesarean delivery with repair of the defect. The timing of delivery depends on the gestational age when the

dehiscence is detected. Delivery is recommended at or near term prior to the onset of labor.

The prevention involves good obstetric practice, like avoiding fundal pressure in the second stage of labor. Uterine manipulation, such as external cephalic version for malpresentation in a woman with a significant uterine anomaly, should be performed gently or not at all[15]. Uterotonic agents for induction and augmentation of labor is a potentially modifiable risk factor and should be used judiciously. Appropriate use of cesarean delivery for management of protracted or arrest of labor prevents rupture associated with obstructed labour.

Conclusion: Obstetricians and labour room teams should learn to identify the risk of rupture. Take help to manage cases or shift the patient to higher facility along with medical personell if emergency management services are not available. Adequate training is required for early detection of warning signs and symptoms of uterine rupture, as they are non-specific.Caution should be exercised during oxytocin augmentation, especially in poorly progressing multiparous women and those with a history of prior caesarean section. Proper supportive and resuscitation methods may aid in preventing maternal morbidity and maternal death. Surgical intervention after uterine rupture in less than 10-37 minutes is essential to minimize the risk of permanent perinatal injury to the fetus. Senior obstetrician involvement in TOLAC/VBAC is recommended.

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