Volume 17; Issue No.2; June 2017

Price: ₹ 30 only

AOGD BULLETIN

AOGD Theme 2017-18

'Optimizing Women's Health Through Enhanced Skills and Best Practices'

Issue:

Red Alert
Maternal Near Miss

AOGD SECRETARIAT

Room No 712, 7th Floor, Private Ward, MCH Block
Department of Obstetrics and Gynecology
Guru Teg Bahadur Hospital & University College of Medical Sciences
Dilshad Garden, Delhi-110095, India
secretaryaogd2017@gmail.com, info@aogd.org

www.aogd.org

AOGD Secretariat
Room No 712, 7th Floor, Private Ward, MCH Block
Department of Obstetrics & Gynaecology
Guru Teg Bahadur Hospital & University College of Medical Sciences
Delhi-110 095, India
www.aogd.org

AOGD BULLETIN
Volume 17-2, June 2017

Contents

Publisher/Printer/Editor
Dr Rashmi on behalf of Association of Obstetricians & Gynecologists of Delhi.

Printed at
Process & Spot C-112/3, Naraina Industrial Area, Phase-1, New Delhi 110 028

Published from
AOGD Office, Room No 712, 7th Floor, Private Ward, MCH Block, Department of Obstetrics & Gynaecology, Guru Teg Bahadur Hospital & University College of Medical Sciences, Delhi-110 095, India

Editor
Dr Rashmi
Ph. No. 011-22692505; Email: info@aogd.org

Total number of pages = 68

AOGD Office-Bearers

Dr Shalini Rajaram
President

Dr Kiran Guleria
Vice President

Dr Gita Radhakrishnan

Dr Amita Suneja

Scientific Advisors

Dr Abha Sharma
Hon. Secretary

Dr A G Radhika
Chairperson
Skill Workshops

Dr Himshweta Srivastava

Dr Sandhya Jain

Joint Secretaries

Dr Alpana Singh
Treasurer

Dr Archana Chaudhary
Co Treasurer

Editorial Board

Dr Rashmi

Dr Bindiya Gupta

Editors

Dr Rachna

Dr Anshuja

Web Editors

Dr Richa Aggarwal

Dr Sruthi

Co-Editors

Committees

Dr Richa Sharma

Dr Sanjeeta Behera

Dr Bhanu Priya

Coordinators Skill Workshops

Dr Vishnu Bhartiya

Dr Shweta Prasad

Clinical Secretaries

Dr Rashmi Gupta

Dr Seema Prakash

Public Relations & Hospitality

President's Message

Dear Friends

At the outset I urge all AOGD members to support Dr. Sudha Prasad as Vice-President FOGSI, (North zone) 2019 and post your ballots in time. You have seen the support and camaraderie she enjoys with members, and her capacity to take on a leadership role nationally. We wish her success in this endeavour.

We are now in the second month of holding office of AOGD and so much has happened since then. Active AOGD members have invested their time and commitment to bring diverse issues related to women's health to the table and I applaud them. Our first 'Basic life Support Skill workshop' on 5th May was well attended by practitioners and residents who had hands on experience at the 'State of the Art Skill lab' at GTBH with a bonus of BLS certification. Dr. AG Radhika, AOGD Skills workshop Chairperson and Dr. Sujata, Director Professor Anesthesiology deserve accolades for the well conducted workshop. 'ABC' of CPR has now changed to 'CAB' with cardiac compressions of prime importance, done first, correctly and with adequate depth and number. I suggest all AOGD members get a BLS certification and this is non-negotiable!

Dr. Ranjana Sharma of Apollo hospital, Chairperson, Urogynecology Commitee (2015-17) Dr. Kuldeep Jain, Chairperson, FOGSI endometriosis committee and Dr. Geeta Mehdiratta, Secretary, IMS Delhi chapter, from Sir Ganga Ram Hospital all held academically and intellectually stimulating CMEs in May. GTB hospital under the leadership of Dr. Kiran and Dr. Abha updated more than 100 delegates on best practices on Antenatal care and culminating May's activities was Dr. Urvashi Jha and team from Fortis Hospital, Vasant Kunj who held the monthly AOGD clinical meeting.

Hope all of you enjoyed the April issue on 'Adolescent Health'. The feedback from AOGD members was very extremely encouraging and this issue on 'Maternal Near Miss' promises not to disappoint. Every day the obstetrician awakens to the possibility that catastrophes can occur without warning and she/he is always in a state of 'high alert'. A seemingly normal antenatal woman in the third trimester may go into labor, rupture her membranes, become dyspnoieic and suddenly there's a 'Near-Miss' on hand. Likewise, women with PPH, severe pre-eclampsia, sepsis, cardiac and other events need rational approach and management to avoid mortality. Setting up an obstetric high dependency unit in high volume centres keeping in mind staffing, equipment and training is a utopian dream waiting to happen.

Planning for the forthcoming FOGSI/AOGD 'BOH – The Triology' is underway and the organising team under the leadership of Dr. Ranjana Khanna are working hard for a successful conference on 19th and 20th August 2017. Please also save your dates for the 39th Annual Conference of AOGD to be held on the 18th & 19th November, 2017 at the India Habitat Centre and Pre-congress workshops on 17th November. Strategizing for the conference has begun and the first announcement with highlights will be made soon. This year Dr. Robert Leitao, Head, Robotic Surgery from Memorial Sloan Kettering Hospital will share his expertise and experiences and deliver the Brigadier Khanna oration.

Looking forward to exciting year-long interactions with all of you!

Shalini Rajaram
President, AOGD (2017-18)

Vice President's Message

Dear AOGD members

First of all I would like to congratulate our editorial team from newly elected office for doing a fabulous job with the inaugural issue of AOGD bulletin. I hope many members will respond enthusiastically to the monthly quiz. Their constructive criticism and valuable suggestions are also most welcome for quality improvement.

A critically ill pregnant woman who nearly died but survived to tell her tale is an important link in the chain of evidence to enhance best clinical practices, skills and policies governing maternal health. Nearly 118 such events occur for one maternal death. Thus MNM is an important tool to identify the contributory factors and delays in maternal death. GOI has recently released guidelines for MNM with a sharper focus on actions to reduce maternal mortality and morbidity in India. So this is just the right time for AOGD to bring out this issue with focus on Near Miss. I sincerely hope this stirs the obstetrician community to do their bit to save every mother's life because "She Matters".

I take this opportunity to invite you all for the forthcoming "FOGSI-BOH: The Triology" conference in partnership with AOGD, Delhi & NCR at The Leela Ambience, Gurugram on 19-20th August. Details are available on our website- www.fogsiboh2017.com Please participate in huge numbers and avail this opportunity to learn & discuss all aspects of BOH.

Cheers & Happy Reading!

Kiran Guleria
Vice President AOGD (2017-18)

From the Secretary's Desk.....

Dear Friends,

It was heartening to know of your enthusiastic response to our first bulletin "The Adolescent Issue". Your comments and suggestions are most welcome and help us in improving the bulletin.

The second edition of bulletin on "Maternal Near Miss" is in your hands and I am sure this will prove to be a valuable aid in managing critical patients. Over the years maternal deaths have reduced because of timely interventions and there are innumerable "near miss" events which have the potential to teach us important lessons.

April was an eventful month with several activities happening. We had an enlightening academic session on "Antenatal care – Best Practices", in which WHO recommendations for effective antenatal care was highlighted upon.

Our Skills Workshop team conducted a Workshop on fundamentals of Maternal Resuscitation at GTB Hospital with huge response from practitioners as well as residents.

We will have a similar skill enhancing session on Basics of Endoscopy in Gynecology, on 21st of July. Do attend , and enrich your skills!

I exhort you to vote for Dr Sudha Prasad For Vice President FOGSI (North Zone) 2019. Ballot papers will be delivered in first week of July. Lets keep the Delhi flag flying.

Cheerio & Happy Reading!

Abha Sharma
Secretary AOGD (2017-18)

Monthly Clinical Meet

Monthly Clinical Meet will be held at Army Hospital- Referral and Research on **30th June, 2017** from 4:00-5:00pm.

From the Editorial Board

Respected Seniors & Dear Friends,

Thanks for the over whelming response and appreciation for the first bulletin. With appreciation comes the responsibility and we hope that this issue too meets your expectations.

As well said by Hillary Clinton "If you want to know how strong a country's health system is, Look at the well-being of its mothers", both maternal mortality and maternal near miss cases reflect on the quality of obstetrical care available to the women. There is always a very thin line between the mortality and near miss. Obstetric patients who almost died but narrowly escaped give an opportunity to the clinicians as well as planners to be better equipped to avert the mortalities. Though our country has seen tremendous fall in the maternal mortality rates during last decade, it still remains a significant health problem. Near miss cases share same pathological and circumstantial factors as the mortality cases. Review of these cases help to identify the gaps in the existing care system and to take corrective measures. In this issue, we have tried to cover some important causes of maternal near misses. We start with understanding the maternal near miss approach. Important but complex topics like cardiac events in pregnancy, Sepsis, Amniotic fluid embolism are presented in simplified manner. Recent advances to tackle postpartum hemorrhage are as important as thromboprophylaxis when indicated. Also information is provided on setting up of HDU and maternal resuscitation as well as blood transfusions, which are essential for preventing maternal deaths.

In the section on Mind, Body and Soul, an interesting topic of Near Death Experiences in discussed, which is challenging for modern science. We continue with our quiz in the end as it was appreciated well in the first issue.

Hope this issue will have something for each and everyone of you in terms of information, knowledge. Feedbacks as well as the suggestions are always welcome.

With warm regards,

The Editorial Team
AOGD (2017-18)

Near Miss approach in Maternal Health

Kiran Guleria[1], Bhanupriya[2]

[1]Professor, [2]Assistant Professor, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

Introduction

A maternal near miss (MNM) is an event in which a woman nearly died, but survived a severe complication occurring during pregnancy, childbirth, or within 42 days of its termination. It represents the extreme degree of organ dysfunction/failure in the wide spectrum of morbidity and differs from death only by the outcome. Over 1 in 100 pregnant women suffer a life-threatening event and about 118 such events occur for each maternal death. The World Health Organization (WHO) used organ dysfunction criteria and parameters of extreme severity specific to obstetrics to define life-threatening conditions associated with pregnancy, standardizing the maternal near miss criteria.[1] Signs of organ dysfunction that follow life-threatening conditions are used to identify maternal near misses so that the same classification of underlying causes is used for both maternal deaths and near misses. Severe Acute Maternal Morbidity (SAMM) refers to a life-threatening disorder that can end up in near miss with or without residual morbidity. Women who develop SAMM during pregnancy share many pathological and circumstantial factors related to their condition. Although some of these women die, a proportion of them narrowly escape death. Near miss cases and maternal deaths together are referred to as severe maternal outcome (SMO).

Several initiatives for maternal and infant health have been implemented worldwide, aimed at achieving the millennium development goals.[2] Nevertheless, advances made over the years are far behind those required for effective morbidity and mortality reduction. As maternal deaths are relatively rare events, in order to overcome the difficulty in estimation and to track quality of service delivery, examining near-miss events has the potential to complement the maternal death reviews. This consistency and a set of near-miss indicators enables assessments of the quality of care provided to pregnant women. Structured health systems are identified as fundamental to obtain better results and accelerate progress for achieving these goals.

Implementation of this approach in health services will serve to:

- Determine the frequency of severe maternal complications, maternal near-miss cases and maternal deaths
- Evaluate a health-care facility or the health system's performance in reducing severe maternal outcomes
- Determine the frequency of use of key interventions for the prevention and management of severe complications related to pregnancy and childbirth; and
- Raise awareness about and promote reflection of quality-of-care issues and foster changes towards the improvement of maternal health care.

The ultimate purpose of the near-miss approach is to improve clinical practice and reduce preventable morbidity and mortality using best evidence-based practices.

Prevalence of Maternal Near Miss (MNM)

Due to the wide variation in identification of near miss cases, it has been difficult to make a summary estimate of the prevalence of near miss globally. In a recent review on articles between January 2004 and December 2010 the prevalence rates of maternal near miss varied between 0.6% -14.98% for disease-specific criteria, between 0.04% -4.54% for management-based criteria and between 0.14% - 0.92% for organ-based dysfunction. The rates are higher in low-income and middle-income countries of Asia and Africa. Based on meta-analysis, the estimate was 0.42% (95% confidence intervals CI 0.40-0.44%) for the organ dysfunction criteria.[3] There are not many studies available from India on maternal near miss. Prevalence was found to be 3.3 – 4.4% for disease specific & management –based criteria from three teaching hospitals including ours from India.[4,5]

Causes of Maternal Near Miss (MNM)

Severe morbidity data are vital for policy planners to know the requirements of essential and emergency obstetric care (EmOC) to manage these. It is also assumed to be a better indicator than maternal mortality alone for designing, monitoring, follow up and evaluation of safe motherhood programs.

Hemorrhage, hypertensive disorders, sepsis and obstructed labor are the most important causes in the developing countries. Causes of near miss are like causes of maternal deaths prevailing in the area. A systematic review to determine the causes of maternal deaths conducted by the WHO recorded wide regional variation. Hemorrhage was the leading cause of maternal deaths in Africa (33.9%) and in Asia (30.8%) while in Latin America and the Caribbean, hypertensive disorders were responsible for 25% deaths.[6,7]

Anemia was reported as an important cause in 12.8% deaths in Asia, 3.7% in Africa and none in the developed countries. Studies from our country have also reported anemia as an important cause and contributor to maternal mortality and severe maternal morbidity. [8,9]

Diagnosis of MNM

Inclusion criteria

Women who are pregnant, in labour, or who delivered or aborted up to 42 days ago arriving at the facility with any of the listed conditions or those who develop any of those conditions during their stay at the health-care facility are labelled as MNM. Women who develop those conditions unrelated to pregnancy (i.e. not during pregnancy or 42 days after termination of pregnancy) are not eligible. Women who are already dead or those who die on arrival at health-care facility should be included because they are likely to represent cases involving a major delay in accessing care.

Criteria for identifying and notifying the MNM case:

Whenever any pregnant woman comes to the health facility in a critical condition, she needs to be given urgent medical treatment. However, prior to the discharge of such cases, there is a need to identify whether the case falls under the category of Maternal Near Miss.

Three major criteria have been mentioned in a review conducted by the WHO, these are described in Table 1. The review has suggested the use of the organ system dysfunction based criteria supplemented with compatible clinical markers of organ system dysfunction that are feasible for collection in the absence of higher-level amenities based criteria for identifying all severe morbidity and investigating the cause as the most reproducible one across similar areas.[10]

For identification of an MNM case as per MOHFW guidelines, the following criteria (minimum three including one from each category) must be met with:[11]

1. Clinical findings (either symptoms or signs),
2. Investigations
3. Interventions

Or

Any single criteria, which signifies cardio respiratory collapse

The clinical criteria have been put under three broad categories:

1. Pregnancy specific obstetric and medical disorders,
2. Pre-existing disorders aggravated during pregnancy,
3. Accidental / Incidental disorders in pregnancy.

Above mentioned broader categories have further been segregated under different clinical situations like hemorrhage, sepsis, hypertension etc.

Inclusion criteria for baseline assessment of quality care:

A. ***Severe maternal complications***: Severe postpartum hemorrhage, severe pre-eclampsia/eclampsia, sepsis or severe systemic infection, rupture uterus and severe complications of abortion

B. ***Management specific criteria: based on critical interventions or intensive care unit use***: Admission to intensive care unit, interventional radiology, laparotomy (includes hysterectomy, excludes caesarean section), use of blood products

C. ***Organ function failure/ Dysfunction based criteria of severity:*** The various criteria for organ dysfunction are summarized in Table 2.

Table 1: Criteria for near miss cases

Criteria	Description	Advantages	Disadvantages
Clinical crteria related to a specific disease entitiy	Disease specific definitions used for common conditions and clinical criteria defined for severe morbity. e.g. Pre-eclampsia is a disease and complications such as eclampsia, renal failure and pulmonary edema identify severe cases	Easy to interpret cases can be identified retrospectively Quality-of-care of that disease can be identified	All problems may not be covered Difficult to define and quantify the condition
Management specific	Management or intervention to disease. e.g. hysterectomy, blood transfusion or admission to ICU	Simple to use in identification of cases	Depends on other variables such as availability of ICU beds, indications for hysterectomy
Organ system dysfunction or failure	Based on the concept that there is a sequence of events leading from good health to death. Death is preceded by organ dysfunction and organ failure. Markers for organ system dysfunction or failure are specified. e.g. Jaundice in the presence or pre-eclampsia	Allow for identification of critically ill women Keeps focus on severe diseases	Dependent on the existence of a minimum level of care including functioning laboratories and basic critical care monitoring

Table 2: Organ dysfunction criteria for MNM

System involved	Parameters
Cardiovascular dysfunction	Shock Lactate > 5, pH < 7.1 Use of continuous vasoactive drugs Cardiac arrest and Cardiopulmonary resuscitation (CPR)
Respiratory dysfunction	Acute cyanosis Respiratory rate >40 or <6/min Oxygen saturation <90% for ≥60 minutes Gasping PaO2/FiO2 < 200mmHg Intubation and ventilation not related to anesthesia
Renal dysfunction	Oliguria nonresponsive to fluids or diuretics Creatinine ≥300mmol/L or ≥3.5mg/dL Dialysis for acute renal failure
Coagulation/ hematological Dysfunction	Clotting failure Transfusion of ≥5 units of blood/red cells Acute thrombocytopenia (<50 000 platelets)
Hepatic dysfunction	Jaundice in the presence of preeclampsia Bilirubin >100mmol/L or >6.0mg/dL
Neurological dysfunction	Metabolic coma (loss of consciousness AND the presence of glucose and keto acids in urine) Stroke Status epilepticus/uncontrollable fits/ total paralysis Coma/loss of consciousness lasting 12 hours or more
Uterine dysfunction	Hysterectomy due to infection or hemorrhage

Simple organ dysfunction scores like Sequential Organ Failure Assessment (SOFA) and Modified Early Obstetric Warning System (MEOWS) have been used successfully and accurately in Indian obstetric population to predict severity of morbidity and mortality in ICU as well as general patients.[9,12]

D. Maternal vital status

- Maternal death

Maternal Near Miss Indicators

1. ***Severe maternal outcome (SMO) refers to*** a life-threatening condition (i.e. organ dysfunction), which includes all maternal deaths and maternal near-miss cases.
2. ***Women with life-threatening conditions (WLTC)*** refers to all women who either qualified as maternal near-miss cases or those who died (i.e. women presenting a severe maternal outcome). It is the sum of maternal near-miss and maternal deaths (WLTC = MNM + MD).
3. ***Severe maternal outcome ratio (SMOR)*** refers to the number of women with life-threatening conditions (MNM + MD) per 1000 live births (LB). This indicator gives an estimate of the amount of care and resources that would be needed in an area or facility [SMOR = (MNM +MD)/LB].
4. ***MNM ratio (MNMR)*** refers to the number of maternal near-miss cases per 1000 live births (MNMR = MNM/ LB). Like SMOR, this indicator gives an estimation of the amount of care and resources that would be needed in an area or facility.
5. ***Maternal near-miss mortality ratio (MNM: MD)*** refers to the ratio between maternal near miss cases and maternal deaths. Higher ratios indicate better care.
6. ***Mortality index*** refers to the number of maternal deaths divided by the number of women with life-threatening conditions expressed as a percentage [MI = MD/(MNM + MD)]. The higher the index the more women with life-threatening conditions die (low quality of care), whereas the lower the index the fewer women with life-threatening conditions die (better quality of care).
7. ***Perinatal outcome indicators*** (e.g. perinatal mortality, neonatal mortality or stillbirth rates) in the context of maternal near-miss could be useful to complement the quality-of-care evaluation.

Maternal Near Miss - Review (MNM-R)

The proportion of women arriving at a health-care facility with SMO provide information about the occurrence of the first delay (in deciding to seek care by the woman and/or her family) or second delay (in reaching an adequate health-care facility) and factors contributing to the delays. In developing countries, about 75% of women with severe obstetric morbidity are in a critical condition upon arrival, underscoring the significance of the first two delays. Availability, accessibility, cost of health-care and behavioral factors play an important role in the utilization of maternal health services. Understanding of these factors by the health personnel, authorities and policy makers and taking appropriate action to address them would improve utilization of maternal health-care services. There are two formats in which the data need to be entered –

1. Facility based Maternal Near Miss Review (FBMNM-R) form. (Available from WHO site)
2. MNM-R case register – details of columns to be made in the register (can be modified according to the facility)

Investigating severe maternal morbidity (near-miss) would aim to document the frequency and nature of maternal near-miss at hospital level and to evaluate the level of care at maternal life-saving emergency services.

This will also provide the gaps for corrective actions to be taken at various levels. Figure 1 summarizes the steps for recording MNM-R. A sample data collection form by WHO is shown in Figure 2.

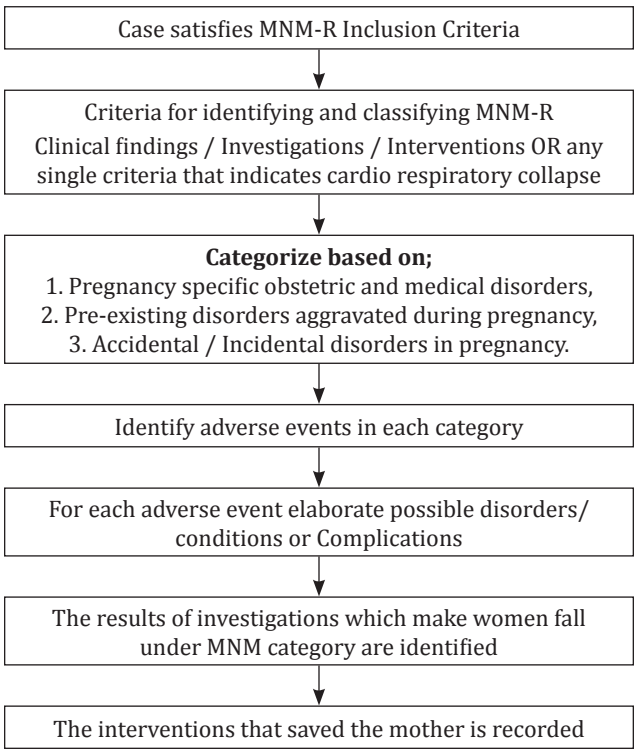


Figure 1: Diagnosing and notifying MNM[11]

Implementation Plan of MNM-R

In the initial phase the MNM-R is implemented in selected well performing medical colleges or tertiary centers. Once the implementation of Maternal Near Miss at Medical Colleges is successfully established, then states can decide to extend it to District hospitals/ other FRUs.

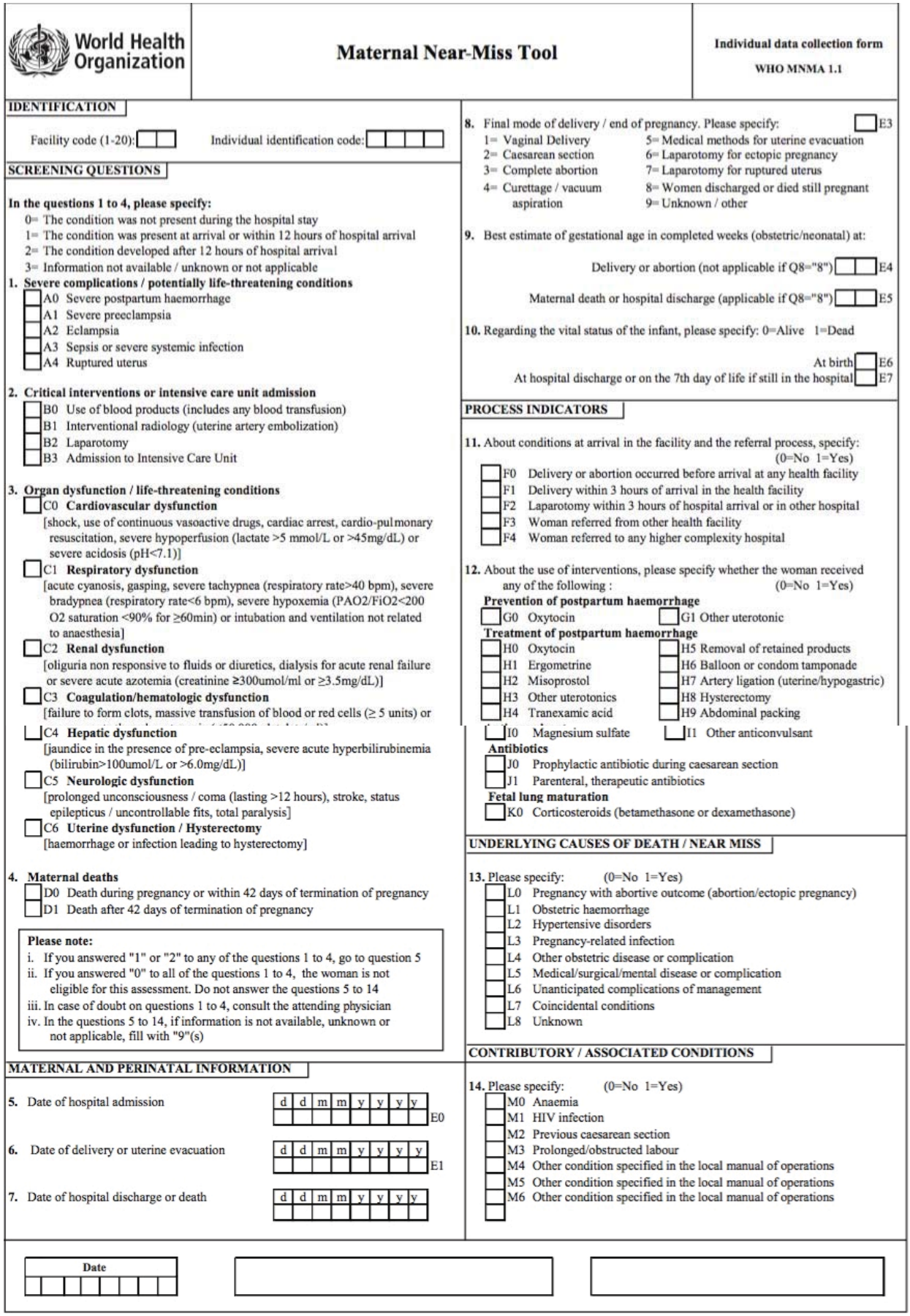
MNM-R is complementary to MDR and purpose of MNM-R is to identify the gaps in service delivery at the earliest which will ultimately help in preventing maternal morbidity and mortality.

Conclusion

Maternal near miss has emerged as an adjunct to investigation of maternal deaths as the two represent similar pathological and circumstantial factors leading to severe maternal outcome. As the number of maternal near-miss cases is more than the maternal deaths and the cases are alive to directly inform on problems and obstacles that had to be overcome during the process of health-care, they provide useful information on quality of health-care at all levels. Thus, there is a need for application of the maternal near-miss concept for assessment of maternal health and quality of maternal care.

References

1. World Health Organization. Evaluating the Quality of Care for Severe Pregnancy Complications: the WHO Near-Miss Approach for Maternal Health. Geneva, Switzerland: WHO, 2011.
2. L. Cochet, R. C. Pattinson, and A. P. Macdonald, "Severe acute maternal morbidity and maternal death audit: a rapid diagnostic tool for evaluating maternal care," *South African Medical Journal*, vol. 93, no. 9, pp. 700–702, 2003.
3. Tunçalp O, Hindin MJ, Souza JP, Chou D, Say L. The prevalence of maternal near miss: A systematic review. BJOG. 2012;119:653–61.
4. Pragti Chhabra, Kiran Guleria, Narinder Kumar Saini, Kannan Tulip Anjur, Neelam Bala Vaid. Pattern of severe maternal morbidity in tertiary hospital of Delhi, India, a pilot study. Tropical doctor, 2008; 38(4): 201-204.
5. Jain S, Guleria K, Vaid NB, Suneja A, Ahuja S. Predictors and outcome of obstetric admissions to intensive care unit: A comparative study. Indian J Public Health 2016;60:159-63
6. Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Van Look PF. WHO analysis of causes of maternal death: A systematic review. Lancet. 2006;367:1066–74.
7. Walraven G, Telfer M, Rowley J, Ronsmans C. Maternal mortality in rural Gambia: Levels, causes and contributing factors. Bull World Health Organ. 2000;78:603–13.
8. Pragti Chhabra. Maternal Near Miss: An Indicator for Maternal Health and Maternal Care. Indian J Community Med. 2014; 39(3): 132–7.
9. Jain S, Guleria K, Suneja A, Vaid N. B. and Ahuja, S. Use of the Sequential Organ Failure Assessment score for evaluating outcome among obstetric patients admitted to the intensive care unit. IJGO 2016;132: 332–6.
10. Say L, Pattinson RC, Gülmezoglu AM. WHO systematic review of maternal morbidity and mortality: The prevalence of severe acute maternal morbidity (near miss). Reprod Health 2004;1:3.
11. Maternal near miss review operational guidelines. December 2014. Maternal health division. Ministry of health and family welfare. Government of India.
12. Singh A, Guleria K, Vaid NB, Jain S. Evaluation of maternal early obstetric warning system (MEOWS chart) as a predictor of obstetric morbidity: a prospective observational study. European J Obstet Gynecol Reprod Biol 2016;207: 11 – 17.



World Health Organization

Maternal Near-Miss Tool

Individual data collection form

WHO MNMA 1.1

IDENTIFICATION

Facility code (1-20): ☐☐ Individual identification code: ☐☐☐☐

SCREENING QUESTIONS

In the questions 1 to 4, please specify:

0= The condition was not present during the hospital stay
1= The condition was present at arrival or within 12 hours of hospital arrival
2= The condition developed after 12 hours of hospital arrival
3= Information not available / unknown or not applicable

1. Severe complications / potentially life-threatening conditions

☐ A0 Severe postpartum haemorrhage
☐ A1 Severe preeclampsia
☐ A2 Eclampsia
☐ A3 Sepsis or severe systemic infection
☐ A4 Ruptured uterus

2. Critical interventions or intensive care unit admission

☐ B0 Use of blood products (includes any blood transfusion)
☐ B1 Interventional radiology (uterine artery embolization)
☐ B2 Laparotomy
☐ B3 Admission to Intensive Care Unit

3. Organ dysfunction / life-threatening conditions

☐ C0 **Cardiovascular dysfunction**
[shock, use of continuous vasoactive drugs, cardiac arrest, cardio-pulmonary resuscitation, severe hypoperfusion (lactate >5 mmol/L or >45mg/dL) or severe acidosis (pH<7.1)]

☐ C1 **Respiratory dysfunction**
[acute cyanosis, gasping, severe tachypnea (respiratory rate>40 bpm), severe bradypnea (respiratory rate<6 bpm), severe hypoxemia (PAO2/FiO2<200 O2 saturation <90% for ≥60min) or intubation and ventilation not related to anaesthesia]

☐ C2 **Renal dysfunction**
[oliguria non responsive to fluids or diuretics, dialysis for acute renal failure or severe acute azotemia (creatinine ≥300umol/ml or ≥3.5mg/dL)]

☐ C3 **Coagulation/hematologic dysfunction**
[failure to form clots, massive transfusion of blood or red cells (≥ 5 units) or

☐ C4 **Hepatic dysfunction**
[jaundice in the presence of pre-eclampsia, severe acute hyperbilirubinemia (bilirubin>100umol/L or >6.0mg/dL)]

☐ C5 **Neurologic dysfunction**
[prolonged unconsciousness / coma (lasting >12 hours), stroke, status epilepticus / uncontrollable fits, total paralysis]

☐ C6 **Uterine dysfunction / Hysterectomy**
[haemorrhage or infection leading to hysterectomy]

4. Maternal deaths

☐ D0 Death during pregnancy or within 42 days of termination of pregnancy
☐ D1 Death after 42 days of termination of pregnancy

Please note:

i. If you answered "1" or "2" to any of the questions 1 to 4, go to question 5
ii. If you answered "0" to all of the questions 1 to 4, the woman is not eligible for this assessment. Do not answer the questions 5 to 14
iii. In case of doubt on questions 1 to 4, consult the attending physician
iv. In the questions 5 to 14, if information is not available, unknown or not applicable, fill with "9"(s)

MATERNAL AND PERINATAL INFORMATION

5. Date of hospital admission d d m m y y y y E0

6. Date of delivery or uterine evacuation d d m m y y y y E1

7. Date of hospital discharge or death d d m m y y y y

8. Final mode of delivery / end of pregnancy. Please specify: ☐ E3

1= Vaginal Delivery
2= Caesarean section
3= Complete abortion
4= Curettage / vacuum aspiration
5= Medical methods for uterine evacuation
6= Laparotomy for ectopic pregnancy
7= Laparotomy for ruptured uterus
8= Women discharged or died still pregnant
9= Unknown / other

9. Best estimate of gestational age in completed weeks (obstetric/neonatal) at:

Delivery or abortion (not applicable if Q8="8") ☐☐ E4

Maternal death or hospital discharge (applicable if Q8="8") ☐☐ E5

10. Regarding the vital status of the infant, please specify: 0=Alive 1=Dead

At birth ☐ E6
At hospital discharge or on the 7th day of life if still in the hospital ☐ E7

PROCESS INDICATORS

11. About conditions at arrival in the facility and the referral process, specify: (0=No 1=Yes)

☐ F0 Delivery or abortion occurred before arrival at any health facility
☐ F1 Delivery within 3 hours of arrival in the health facility
☐ F2 Laparotomy within 3 hours of hospital arrival or in other hospital
☐ F3 Woman referred from other health facility
☐ F4 Woman referred to any higher complexity hospital

12. About the use of interventions, please specify whether the woman received any of the following : (0=No 1=Yes)

Prevention of postpartum haemorrhage

☐ G0 Oxytocin ☐ G1 Other uterotonic

Treatment of postpartum haemorrhage

☐ H0 Oxytocin
☐ H1 Ergometrine
☐ H2 Misoprostol
☐ H3 Other uterotonics
☐ H4 Tranexamic acid
☐ H5 Removal of retained products
☐ H6 Balloon or condom tamponade
☐ H7 Artery ligation (uterine/hypogastric)
☐ H8 Hysterectomy
☐ H9 Abdominal packing

☐ I0 Magnesium sulfate ☐ I1 Other anticonvulsant

Antibiotics

☐ J0 Prophylactic antibiotic during caesarean section
☐ J1 Parenteral, therapeutic antibiotics

Fetal lung maturation

☐ K0 Corticosteroids (betamethasone or dexamethasone)

UNDERLYING CAUSES OF DEATH / NEAR MISS

13. Please specify: (0=No 1=Yes)

☐ L0 Pregnancy with abortive outcome (abortion/ectopic pregnancy)
☐ L1 Obstetric haemorrhage
☐ L2 Hypertensive disorders
☐ L3 Pregnancy-related infection
☐ L4 Other obstetric disease or complication
☐ L5 Medical/surgical/mental disease or complication
☐ L6 Unanticipated complications of management
☐ L7 Coincidental conditions
☐ L8 Unknown

CONTRIBUTORY / ASSOCIATED CONDITIONS

14. Please specify: (0=No 1=Yes)

☐ M0 Anaemia
☐ M1 HIV infection
☐ M2 Previous caesarean section
☐ M3 Prolonged/obstructed labour
☐ M4 Other condition specified in the local manual of operations
☐ M5 Other condition specified in the local manual of operations
☐ M6 Other condition specified in the local manual of operations
☐

Date ☐☐☐☐☐☐☐☐

Figure 2: Sample data collection form by WHO

Cardiac Events: Early recognition and treatment

Taru Gupta[1], Shweta Singh[2], Sangeeta Gupta[3]

[1]Professor and Academic Head, [2]Senior Resident, [3]Professor and head, ESIC-PGIMSR Basaidarapur, New Delhi

Introduction

Heart disease complicates around 0.3-3.5% pregnancies in India; out of which rheumatic heart disease and congenital heart disease are present in 69% and 21% respectively. Mitral stenosis (38.5%) and Atrial septal defect (25%) constitute majority of the congenital cases. In recent times risk of acute MI in pregnancy has also increased 3-4 times due to advanced maternal age, IVF pregnancies and life style factors (obesity, smoking, HT, DM)[1].

Diagnosing cardiac disease is a challenge due to similar complaints of normal pregnancy and initial stages of heart failure. Table 1 has illustrated the signs and symptoms differentiating heart disease from normal pregnancy.

Table 1: Sign and Symptoms of Normal vs. Heart Disease in Pregnancy

Normal pregnancy	*Heart Disease*
1. Fatigue 2. Exertional dyspnea 3. Palpitation 4. Elevated JVP 5. Sinus tachycardia 6. Third heart sound 7. Systolic flow murmur 8. Pedal edema	1. Chest pain 2. Severe breathlessness, orthopnea, Paroxysmal nocturnal dyspnea, cough 3. Atrial flutter or atrial fibrillation 4. Systemic hypotension 5. Fourth heart sound 6. Pulmonary edema 7. Pleural effusion

Acute on Chronic Heart Disease

Patients with congenital heart disease, prior cardiac surgery or cardiac related problems in previous pregnancy may show signs of clinical deterioration during pregnancy which include decreased exercise tolerance, increased palpitations, irregular pulse, change in previous heart murmur, increased blood pressure, decreased oxygen saturation, added sounds on auscultation of the lungs and increasing ankle edema.

Management[2,3]

Preconception Care

1. Baseline cardiac function status (Table no 2) and calculation of risk prediction according to CARPREG risk scoring (Table no 3).
2. Counseling the patient regarding pregnancy risk to female and fetus.
3. Opinion of cardiologist for optimization of their condition.
4. Review current medications to determine appropriateness of drugs.
5. Co-ordination between cardiologist, obstetrician, physician and anesthesiologist is necessary for successful outcome of pregnancy.
6. **Absolute contraindication for conception are as follows:**
   a. Primary pulmonary hypertension
   b. Eisenmenger's syndrome
   c. Coarctation of aorta with valvular involvement
   d. Marfan syndrome with aortic involvement
   e. Peripartum cardiomyopathy with persistent left ventricular dysfunction

Table 2: New York Heart Association functional classification of heart failure[4]

Class I	Patients with cardiac disease but without resulting limitations of physical activity.
Class II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest.
Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest.
Class IV	Patients with cardiac disease resulting in an inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency may even be present at rest.

Table 3: Risk prediction according to the CARPREG risk score[5]

Predictors of maternal cardiovascular events	
1. NYHA Class > II 2. Cyanosis 3. Prior cardiovascular event 4. Systemic ventricular ejection fraction <40% 5. Left heart obstruction	
For each CARPREG predictor a point is assigned	
No of predictors	**Risk of cardiac events in pregnancy (%)**
0	5
1	27
>1	75

Antepartum Care

- Regular evaluation of patients for any deterioration of symptoms
- Twice weekly follow-up till 28 weeks and weekly afterwards

- ANC checkup should be done by same clinician so that significant changes can be detected early, allowing timely intervention.
- ECG and ECHO to evaluate any clinical deterioration.
- Anomaly scan and fetal ECHO (in cases of congenital heart disease) between18-22 week.
- Antepartum fetal surveillance at 30-34 weeks in case of fetal growth restriction.
- Admission according to the functional status of patient.
- Multidisciplinary team approach involving obstetricians, cardiologist, pediatrician & anesthetist.

Labor and delivery

- Strict input output charting.
- Avoid supine position.
- Propped up position and O_2 supplementation.
- Frequent chest auscultation.
- Consultant delivery with high dependency care.
- Vaginal delivery preferable, LSCS only for obstetric indication.
- Endocarditis prophylaxis:
  a. In Indian scenario, prophylaxis given to patients with H/O infective endocarditis, congenital cyanotic heart disease, prosthetic valve and cardiac transplantation with valvulopathy. Drugs used are ampicillin, amoxicillin, gentamicin. Vancomycin is given in resistant cases
  b. Inj. Penicillin 1.2 MU 3 weekly – in rheumatic heart disease

Peripartum Cardiomyopathy (PPCM)[6]

It is defined as heart failure in last month of pregnancy or within 5 months postpartum in absence of prior heart disease. There is no determinable cause and Echo findings suggest left ventricular dysfunction. The latter includes ejection fraction (EF) <45%, functional shortening <30% and diastolic dimension (diameter/ Body surface area) >2.7 cm/m^2. The incidence is 1:1500 to 1:4000 with 90 % occurring in first 2 months postpartum. Fifty percent deaths occur in first 6 weeks postpartum. Mortality ranges from 18 to 56%.

Management

Once the diagnosis is suspected, other supportive investigations include:

1. ECG: Normal sinus rhythm or sinus tachycardia, T wave inversion, Q wave, Nonspecific ST segment
2. X ray Chest – Cardiomegaly
3. Blood samples – C- Reactive protein is ↑, Brain natriuretic peptide (BNP) ↑↑, LDL ↑, Interferon gamma ↑

Table 4: Drugs in management of Peripartum Cardiomyopathy

Principle of management		Drugs
Salt restriction		
Reduce preload	Diuretics	Furosemide 20-40 mg p/o every day
Reduce afterload	Vasodilators	1. Hydralazine 25-100 mg p/o every day and /or 2. Amlodipine 5-10 mg p/o every day 3. Postpartum – Enalapril 5 mg BD
Reduce myocardial oxygen requirement	Maintain HR b/w 80-100 bpm	1. Metoprolol 25-100 mg p/o every day or 2. Carvedilol 3.25-25 mg p/o every day
Reduce inflammation		Pentoxyphylline 400 mg p/o TDS
Inhibit prolactin secretion	Product of Prolactin act as antiangiogenic, proapoptotic and proinflammatory causing myocardial dysfunction, leading to PPCM	Bromocriptine
Anti-coagulation	If cardiomegaly and reduced EF	Heparin/ LMWH/ Oral Anticoagulants

Recent professional society guidelines recommend implantable cardioverter defibrillators for patients with nonischemic cardiomyopathy and LVEF of ≤40% for optimal medical therapy.

Patients presenting with or progressing to decompensated heart failure may exhibit hypoxemia, fulminant pulmonary edema, low cardiac output, and evidence of insufficient organ perfusion, and these individuals often require specialized care in an intensive care unit. Inotropic support, mechanical ventilation, as well as circulatory support in the form of intraaortic balloon pump counterpulsation, LV assist device, and cardiac transplantation all have been used in patients with PPCM

If the woman develops PPCM in antenatal period, delivery can reduce the hemodynamic stress on heart. Since it is more prevalent in last trimester, delivery is planned since the fetus usually attains lung maturity. Vaginal delivery is preferred and cesarean is reserved only for obstetric indications. Effective pain management like use of epidural analgesia is important to avoid increase in cardiac output from pain and anxiety. Central venous pressure monitoring is recommended for careful monitoring of fluid balance. General or regional anesthesia can be used during cesarean delivery. Drugs used in management are outlined in Table-4.

Outcome of PPCM depends on EF and left ventricular end-diastolic volume, response to medical management and normalization of left ventricular function within 6 months.

Acute Myocardial Infarction (MI)[5]

Incidence and etiology*:* 1:10,000 deliveries. More common in 3rd trimester or during perpeurium in first or second pregnancy. Besides high risk factors such as advanced maternal age, hyperlipidemia, diabetes, autoimmune factors and spontaneous coronary dissection, other causes specific to pregnancy include use of nifedipine in preterm labour and methyl ergometrine for PPH.

Presentation: Ischemic chest pain, abnormal ECG pattern, elevated cardiac enzymes – Troponin I > 0.15ng/ml (more sensitive than CPK-MB)

Management

Reperfusion therapy

1. Administer TPA 100 mg over 90 min to lyse intracoronary thrombus.
2. Early coronary angiography
3. Coronary stenting
4. Emergency coronary artery by-pass grafting

Medical management (MONA; should be completed in < 10 minutes)

- M : Morphine sulphate 2 to 4 mg IV
- O : oxygen nasal cannula or mask
- N : Nitroglycerine sublingual mg every 5 minutes × 3 doses
- A : Aspirin 160-325 mg chewed

1. Do a 12 lead ECG; If normal - repeat after 15 min, ST segment elevation or new left bundle branch block (LBBB) – treat as MI.
2. β blocker , IV nitroglycerin.
3. Anticoagulation – IV heparin, antiplatelet therapy (clopidogrel) and anti thrombin therapy
4. If fetus is viable- continuous fetal monitoring is recommended

Congenital Heart Disease

Due to advancement in cardiothoracic surgery, there has been increase in number of pregnant patients with repaired congenital heart disease. Such patients have good prognosis during pregnancy and postpartum. Patients of Eisenmenger's Syndrome have worst prognosis.

Atrial Septal Defect: It is the most common defect seen in pregnancy and is usually well tolerated. Closure is done in symptomatic patients or with pulmonary/ systemic shunt flow ratio >2:1.

Ventricular Septal Defect: Smaller lesions (<0.5 cm) have a lower risk of Eisenmenger's syndrome while larger unrepaired lesions are at high risk. Larger repaired lesions are well tolerated.

Persistent Ductus Arteriosus: This is usually uncommon in pregnancy. Large defects – have significant left to right shunts, may develop atrial fibrillation and congestive heart failure. Risk of paradoxical emboli is high.

Tetralogy of Fallot (TOF): There is an increased incidence of spontaneous fetal loss. Corrected TOF is well tolerated.

Management of septal defects

Echo should be done to evaluate the size of defect, shunt and measuring pulmonary pressure. Measures should be taken to avoid hypertension, arrhythmias and tachycardia. Medical therapy is recommended in ventricular dysfunction and anticoagulation is not indicated.

Others

Coarctation of aorta*:* It is rarely seen in pregnancy, mostly are corrected in childhood. Condition may be exacerbated by pregnancy.

Marfan Syndrome*:* It is an autosomal Dominant connective tissue disorder. Due to risk of rupture and dissection of aorta, pregnancy is contraindicated. Aortic root >4.5 cm should be corrected preconceptionally. Preimplantaton genetic diagnosis and selective replacement of unaffected IVF embryos can decrease incidence in offspring.

Pulmonary Hypertension[7]*:* It is clinically defined as persistently elevated pressure and mean pressure >25 mmHg at rest. Pulmonary HT is of 2 types. Primary is due to cardiac disease while secondary is due to pulmonary vascular disease. Pregnancy is contraindicated as mortality ranges from 30%-60% in primary and secondary hypertension respectively.

If patient conceives and continues pregnancy, treatment includes pulmonary vasodilators like Nifedepin, parenteral Prostacyclin and Nitric Oxide. Presently, Sildafenil and nebulized Iloprost is also recommended. Delivery is planned at 32-34 weeks. Vaginal delivery is planned under epidural analgesia and cesarean is done only in patients with poor cardiac function under GA.

Valvular Disease[5]

During pregnancy, Valvular incompetence is well tolerated than stenotic lesions. Complications include heart failure, dysrhythmias and pulmonary edema. Risk

of complication depends on specific lesion, number of valve involved and degree of obstruction.

Mitral Stenosis

It is the most common valvular lesion. Moderate and severe stenosis develop symptoms once cardiac load is increased due to pregnancy. Common complications include pulmonary edema, atrial fibrillation (AF), supraventricular tachycardia and thrombus formation. Tachycardia, fluid overload, hypotension and increase pulmonary vascular resistance should be avoided.

Management

1. ECHO –
   a. to see the severity of stenosis and size of left atrium and ejection fraction
   b. Moderate MS = 1-1.5 cm^2, Severe MS = <1 cm^2 of valve area
2. ECG – to exclude atrial fibrillation
3. Medical therapy:
   a. B blocker – to prevent tachycardia
   b. Pain management
   c. Diuretics – to treat pulmonary edema
   d. Digoxin- for AF
   e. Anticoagulation – dilated left atrium and chronic atrial fibrillation
4. **Labor and Delivery:** The woman should be kept propped up and oxygen saturation should be monitored. Tocolysis is contraindicated and concentrated oxytocin is used for augmentation. Vaginal delivery with epidural analgesia is preferred and LSCS is reserved for obstetric indication. Morphine can also be used for labour analgesia and it also reduces pulmonary edema. Second stage of labor should be cut short by using prophylactic forceps or vaccum. Injection frusemide must be given after delivery of the placenta and methyl ergometrine is contraindicated for active management. Endocarditis prophylaxis should be given in labor.

Aortic Stenosis (AS)

Isolated AS is due to congenital bicuspid aortic valve while multiple valve involvement is due to rheumatic heart disease (RHD). Mild disease (Valve area >1.5 cm^2, peak gradient <50 mmHg) is well tolerated while severe disease (Valve area <1 cm^2; peak gradient >75 mmHg) has significant risk and needs preconception correction. Stenosis leads to fixed output leading to complication of under perfusion. Complications include angina, syncope, arrhythmias and pulmonary edema. Hypervolemia, valsalva, hypotension and bradycardia must be avoided. Management is almost like mitral stenotic lesion except that medical therapy is for ventricular arrhythmias and anti-coagulation is not required.

Aortic and mitral valve insufficiency

It is well tolerated. Avoid arrhythmias, bradycardia, and increased systemic resistance.

Mitral valve prolapse

Most commonly encountered cardiac lesion during pregnancy and is well tolerated.

Mechanical Heart Valve

Mechanical valves require lifelong anticoagulation while bio-prosthetic valves do not require anti coagulation. Complications include valve failure, thrombosis and mortality (3%). Adequate anticoagulation recommended throughout pregnancy. Anticoagulation therapy is summarized in Table-5.

Table 5: Anti Coagulation Therapy (The 2012 ninth ACCP guidelines)[5]

	Drug	Dose	Goal
1	High dose LMWH therapy throughout gestation	Enoxaparin 1 mg/kg -every 12 hrs	Anti- Xa levels- 4 hr post injection ≈ 1 U/ml
2	High dose UFH throughout gestation	UFH 5000 - 7500 units S/C - every 12 hrs	• Anti- Xa levels 0.35 to 0.7 U/ml • APTT ≥2 times control
3	Either of 2 regimen till 12 weeks. Change to warfarin. At 36 weeks stop warfarin and change to UFH or LMWH until delivery. Switch back to warfarin post partum	-	INR = 2.5-3.5

Cardiac Transplantation[4]

Well tolerated if cardiac functions are stable prior to pregnancy. Complications include effects of immunosuppressive therapy, hypertension, pre-eclampsia, infections, acute rejection, low birth weight and prematurity. Vaginal delivery is preferred and LSCS is for obstetric indication.

Conclusion

Early detection, appropriate referral and multidisciplinary approach is the key to successful management of cardiac disease in pregnancy. Pre pregnancy counseling, workup and proper antenatal care is important. Low threshold should be kept for ECHO and pulmonary edema and arrhythmias are the most common complications. Intensive monitoring and elective planned vaginal delivery is recommended in tertiary care center and LSCS is reserved only for obstetric indication.

References

1. Konar H, Chaudhury S. Pregnancy complicated by maternal heart disease: A review of 281 women. The Jr. Obstet. Gyn. India 2012; 62 (3): 301-6.
2. Ray P, Murphy GJ, Shutt LE. Recognition and management of maternal cardiac disease in pregnancy. Br J Anaesth 2004; 93: 428-39.
3. Gandhi M, Martin RS. Cardiac Disease in Pregnancy. Obstet Gynecol Clin N Am2015; 42:315-33.
4. The criteria committee of the New York Heart Association. Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis, 6th edn. Boston: Little, Brown, 1964.
5. Siu SC, Sermer M, Colman JM, Alvarez AN, Mercier LA, Morton BC, et al. Prospective multicentric study of pregnancy outcomes in women with heart disease. Circulation 2001; 104: 515-21.
6. Hibbard JU, Lindheimer M, Lang RM. A modified definition for Peripartum cardiomyopathy and prognosis based on echocardiomyopathy. Obstet Gynecol 1999;94:311-6
7. Gaine SP, Rubin LJ. Primary pulmonary hypertension. Lancet 1998; 352: 719-25.

Managing Septicaemic Shock and Assessment of Postpartum Sepsis

Nidhi Gupta[1], Aruna Nigam[2]

[1]Assistant Professor, [2]Professor, Department of Obstetrics & Gynaecology, Hamdard Institute of Medical Science and Research, Jamia Hamdard, New Delhi

"Sepsis is a life threatening condition that arises when the body's response to an infection injures its own tissues and organs. Sepsis leads to shock, multiple organ failure and death especially if not recognised early and treated promptly. Sepsis remains the primary cause of death from infection despite advances in modern medicine, including vaccines, antibiotics and acute care. Millions of people die of sepsis every year worldwide."

Merinoff Symposium 2010: Sepsis

Incidence

Sepsis may arise in pregnancy at any time: before delivery, during labour or postpartum. In addition, sepsis may arise from many sources and is not limited to infections arising from the genital tract. World wide up to 20 to 30 % of intensive care unit (ICU) admissions of obstetric patients result from sepsis in pregnancy which contributes to maternal mortality between 3 % in developed countries and 12 % in developing countries. Urinary tract infection and chorioamnionitis are common infections associated with septic shock in the pregnant women.[1]

Terminology

Systemic Inflammatory Response Syndrome (SIRS)*:* It is defined as more than one of the following clinical findings: Temperature > 38°C or < 36°C, heart rate > 90 per minute, hyperventilation (respiratory rate > 20 per minute or PCO2 < 32 mm Hg), WBC count >12,000 or < 3,000.

Sepsis: It is defined as presence of both infection (invasion of tissue, fluid or a body cavity by pathogenic micro-organisms) and systemic manifestations of inflammatory response syndrome (SIRS).

Severe Sepsis: It is defined as sepsis complicated by sepsis-induced organ dysfunction or developing tissue hypoperfusion.

Septic shock: It is defined as the persistence of hypoperfusion (hypotension) in a patient with sepsis, despite adequate volume resuscitation. Hypotension is defined as: Systolic blood pressure (SBP) < 90 mmHg or mean arterial blood pressure (MAP) < 60 mmHg, or reduction of SBP > 40 mmHg from baseline.

Sepsis-induced tissue hypoperfusion is defined as hypotension or blood lactate concentration ≥ 4 mmol/L persisting after initial isotonic crystalloid fluid challenge of 30mls/kg.

Puerperal sepsis: Infection of the genital tract occurring at any time between the rupture of membranes or onset of labor up to 42nd day postpartum, in which fever (oral temperature 38.5°C or higher on any occasion) and 1 or more of the following signs and symptoms are present which include pelvic pain, abnormal vaginal discharge, e.g. presence of pus, abnormal smell/foul odour of discharge and/or subinvolution, i.e. delay in the rate of reduction of the size of the uterus (<2cm/day during the first 8 days).

Risk Factors[2]

1) **Maternal:** Cesarean section, multiple vaginal exams (>5), prolonged rupture of membranes, prolonged labor, multiple obstetrical maneuvers, retained products of conception, anemia, poor nutrition, existing infection (HIV/AIDS, Malaria), primiparity, multiple pregnancy, obesity
2) **Community Based:** Low socioeconomic status, unhygienic conditions, lack of adequate healthcare, untrained birth attendant

Causes

The most common sites of infection in pregnancy are urinary tract infection (pyelonephritis), infection of pelvic structures (septic abortion, chorioamnionitis and endometritis), surgical wounds (caesarean section, perineal laceration) and breast (mastitis). Other causes can be infection of intravenous cannula sites, after urological procedures in the presence of urinary tract infection, related to regional anaesthesia e.g. spinal / epidural abscess (rare), pneumonia (viral and bacterial), acute appendicitis, acute cholecystitis, pancreatitis and necrotising fasciitis.[1,3]

Common Pathogens

The most prevalent bacterial organisms responsible for severe infection include Group A beta haemolytic streptococci (GAS) also known as Streptococcus pyogenes, Group B streptococcus, Escherichia coli, Klebsiella, Staphylococcus aureus and anaerobes like peptostreptococci, peptococci, bacteroides. Clostridium

species and listeria monocytogenes are less common pathogens involved in septic shock. There can be viral causes e.g. influenza, varicella, hepatitis and herpes simplex. Malaria and other tropical infections can also rarely cause septicaemia with superadded bacterial infection.

Diagnosis [1,4]

Severe sepsis or septic shock can be diagnosed on the basis of clinical as well as laboratory findings or investigations.

Signs and Symptoms

- Fever, temperature instability (higher than 38.0°C or lower than 36.0°C)
- Tachycardia (heart rate greater than 110 beats/min)
- Tachypnea (respiratory rate greater than 24 beats/min)
- Diaphoresis, clammy or mottled skin
- Nausea or vomiting
- Hypotension or shock
- Oliguria or anuria
- Pain (location based on site of infection)
- Altered mental state (confusion, decreased alertness)

Investigations

1) Complete blood picture	• White blood cell (WBC) count > 12 x 10^9 • Leucopenia - WBC count < 4 x 10^9 • Normal WBC count with > 10 % immature forms • Thrombocytopenia
2) Plasma C-reactive protein	• > 7 mg/L (usually significantly higher in bacterial sepsis)
3) Urea and electrolytes	• Creatinine rise of > 44.2 µmol/L; sepsis is severe if creatinine level > 176 µmol/L
4) Plasma glucose	• Hyperglycaemia in the absence of diabetes (plasma glucose > 7.7 mmol/L
5) Liver function tests (LFTs)	• Hyperbilirubinaemia (plasma total bilirubin > 70 µmol/L)
6) Coagulation profile	• Coagulation abnormalities (INR > 1.5 or APTT > 60 seconds • Disseminated intravascular coagulation
7) Blood gas	• Arterial hypoxaemia (PaO2 / FIO2 < 300 mmHg) • Sepsis is severe if < 250 mmHg in the absence of pneumonia or < 200 mmHg in the presence of pneumonia • Raised serum lactate ≥ 4 mmol/L • Low arterial pH • Increased base deficit • Metabolic acidosis
8) Positive culture from infection site or blood	

Scoring Systems[5]

There are several scoring systems like Modified Early Warning Score (MEWS), REMS score (Rapid Emergency Medicine Score) and Sepsis in Obstetrics Score (S.O.S.) that have been used to identify patients at risk for sepsis and septic shock, morbidity and mortality and need for ICU admission (Table 1). The sensitivity of these scores range from 80-100%, specificity is 80-99% and positive predictive value is 4.6-16%. All the scores have a high negative predictive value of 99-100%. The Modified Early Obstetric Warning Score (MEOWS) is a tool designed specifically for the obstetric population, and has an 89% sensitivity and 80% specificity in predicting morbidity. MEOWS cut off of >5 is critical and requires referral to an intensive care unit (Table 2).

Management

Septic Shock Management comprises initial resuscitation phase and later maintenance phase.

A) ***Initial Resuscitation Phase (first 6 h)***

Early goal-directed therapy (EGDT) is the mainstay of management of severe sepsis and septic shock that aims to restore perfusion and tissue oxygenation by achieving physiologic targets during the early phases of resuscitation. These include normal or near normal measurements of mean arterial pressure (MAP), central venous pressure (CVP), mixed venous oxygen saturation (SVO_2), and clearance of blood lactate. The steps include:

- Blood cultures should be obtained and empiric antibiotics should be initiated preferably within 1 hour
- Central line placement should be done and central venous pressure 8 mm Hg or higher should be achieved
- Norepinephrine infusion if indicated (mean arterial pressure lower than 65 mm Hg after resuscitation)
- Transfusion of packed red blood cells if indicated by hemoglobin less than 7 g/dL
  - *Hemodynamic Management:* Fluid resuscitation is started with the use warm normal saline or lactated Ringer's. Rapid infusion of 500 mL over 15 min, with a 1-h goal: total 20 mL/kg and 3-h goal: total 30 mL/kg

It is recommended that isotonic crystalloids are used as the initial fluid of choice in the resuscitation of severe sepsis and septic shock (Grade 1B, RCOG). The Guideline Development Group recommends AGAINST the use of hydroxyethyl starches for fluid resuscitation of severe sepsis and septic shock. (Grade 1B, RCOG) Albumin in the fluid resuscitation of severe sepsis and septic shock is suggested when patients require substantial amounts of crystalloids and a colloid is being considered. Physiologic perfusion end points: Central venous pressure 8–12 mm Hg, mean arterial pressure greater than 65 mm Hg, urine output greater than 25 mL/h

Table 1: Sepsis in Obstetrics Score (S.O.S.)

Variable	High abnormal range				Normal	Low abnormal range			
Score	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature (°C)	>40.9	39–40.9		38.5–38.9	36–38.4	34–35.9	32–33.9	30–31.9	<30
Systolic Blood Pressure (mmHg)					>90		70–90		<70
Heart Rate (beats per minute)	>179	150–179	130–149	120–129	≤119				
Respiratory Rate (beats per minute)	>49	35–49		25–34	12–24	10–11	6–9		≤5
SpO2 (%)					≥92%	90–91%		85–89%	<85%
White Blood Cell Count (/µL)	>39.9		25–39.9	17–24.9	5.7–16.9	3–5.6	1–2.9		<1
% Immature Beutrophils			≥10%		<10%				
Lactic Acid (mmol/L)			≥4		<4				

Table 2: The Modified Early Obstetric Warning Score (MEOWS)

Score	3	2	1	0	1	2	3
Temperature		<35°.C		35–37°.C		37.5–39°.C	>39°.C
Systolic* BP	≤70	71–79	81–89	90–139	140–149	150–159	≥160
Diastolic* BP			≤45	46–89	90–99	100–109	≥110
Pulse		≤40	40–50	51–100	101–110	111–129	≥130
Respiratory Rate		≤8		9–14	15–20	21–29	≥30
AVPU				Alert	Responds to Voice	Responds to Pain	Unconscious
Urine output mLs/hr	<10	≤30		Not Measured			

If the pulse rate is higher than the systolic blood pressure then score 2 for 'Pulse'

- Vasopressor therapy: Vasoactive agents are used if mean arterial pressure is lower than 65 mm Hg after fluid resuscitation. Inotropes are started if central venous oxygen saturation remains less than 70%. Vasopressin is added if vasopressor therapy is ineffective.
- Oxygen therapy with nasal cannula or face mask
- Intubate, mechanical ventilation, if respiratory failure
- Sedation, analgesia, neuromuscular blockade if required
- Antimicrobial Therapy[6] : Empirical antibiotic therapy should be started as early as possible. Therapy should not be delayed while awaiting cultures because survival differences are seen in delay of antibiotic therapy of only 1 h. If patient is in shock and blood culture reports are pending, then start Piperacillin-Tazobactam at 4.5 g intravenously every 6 h or Cefoperazone-sulbactam till the sensitivity report is available and modify as per the report. If patient has only fever, with no features of severe sepsis start amoxicillin clavulanate oral 625TDS/IV 1.2 gm TDS Or Ceftriaxone 2gm IV OD+ Metronidazole 500mg IV TDS +/-gentamicin 7mg/kg/day OD if admission needed. MRSA cover may be required if suspected or colonized (Vancomycin/ Teicoplanin)
- *Search and Eliminate Source of Sepsis:* This includes evacuation of retained products of conception, debridement of infected tissue (incision, episiotomy, fascia), drainage of abscess, pyuria with ureteral obstruction and appendicitis, cholecystitis should be dealt accordingly

B) ***Maintenance Phase***

The steps in maintenance phase include:

- Insulin protocol initiated, if indicated
- Corticosteroid therapy for refractory septic shock: Hydrocortisone is given at 50 mg intravenously every 6 h
- Thromboembolic prophylaxis: This includes sequential compression device and Enoxaparin at 40 mg subcutaneously once daily or 5,000 units heparin subcutaneously every 8 h if hepatic or renal impairment)
- Reassess antibiotic therapy and narrow spectrum if possible
- Stress ulcer prophylaxis: Famotidine at 20 mg every 12hourly

Conclusion

Sepsis remains a major cause of maternal morbidity and mortality. Scoring systems like MEOWS and SOS help in early identification of sepsis in pregnancy. Management includes initial resuscitation with early goal directed therapy followed by maintenance treatment.

References

1. South Australian Perinatal Practice Guidelines. Sepsis in pregnancy. Available at: http://www.sahealth.sa.gov.au/

wps/wcm/connect/public+content/sa+health+internet/clinical+resources/clinical+topics/perinatal/perinatal+practice+guidelines.

2. Van Dillen J, Zwart J, Schutte J, van Roosmalen J. Maternal sepsis: epidemiology, etiology and outcome. Curr Opin Infect Dis. 2010 ;23(3):249-54
3. Bacterial sepsis in pregnancy- RCOG. Available at: https://www.rcog.org.uk/globalassets/documents/guidelines/gtg_64a.pdf. Accessed 17.5.2017
4. Barton JR, Sibai BM. Management of severe sepsis and septic shock. In: Sibai BM, editor. Management of acute obstetric emergencies. 1st ed. Philadelphia (PA): Saunders, an imprint of Elsevier Inc; 2011. p. 93–100. Copyright © Elsevier, 2011.
5. Albright CM, Ali TN, Lopes V, et al. The Sepsis in Obstetrics Score: a model to identify risk of morbidity from sepsis in pregnancy. Am J Obstet Gynecol 2014;211:39.e1–8.
6. National Treatment Guidelines for Antimicrobial Use in Infectious Diseases. NATIONAL CENTRE FOR DISEASE CONTROL, Directorate General of Health Services, Ministry of Health & Family Welfare, Government of India. 2016

I Made a Promise

She came on a noisy trolley numb lifeless in a pool of blood
Her eyes unresponsive to worldly words and sank further
Accompanying women crying," please look what just happened?"
As a naive resident in the wee hours shook up a bit, looking at her

Was holding tight, she was on verge of death in full bloom of youth
Her pregnancy - a gift of life went totally uncared for!
She seemed to be slowly but steadily giving up
As I took few quick steps closer, I too wasn't very sure

With a dead baby inside and what they call as hemorrhagic shock
"Take the line sister, start it fast, I need blood, somebody run!"
We brisked as life was callously leaving her body, drop by drop
But we would do everything possible, "gear up every one!"

We become nocturnal, we get burned out, we work so hard
For little babies to safely land, promise women their motherhood
The first loud cry and her everlasting smile are our biggest reward
I prayed hard if we could make her alive, if we really could

I know she was utterly careless, and so were her caretakers
There's so much more, which these women of my country need
But nothing could deter us from making best of our efforts
Finally, we could save, and at dawn of hope,saw her breathe!

Cheers.

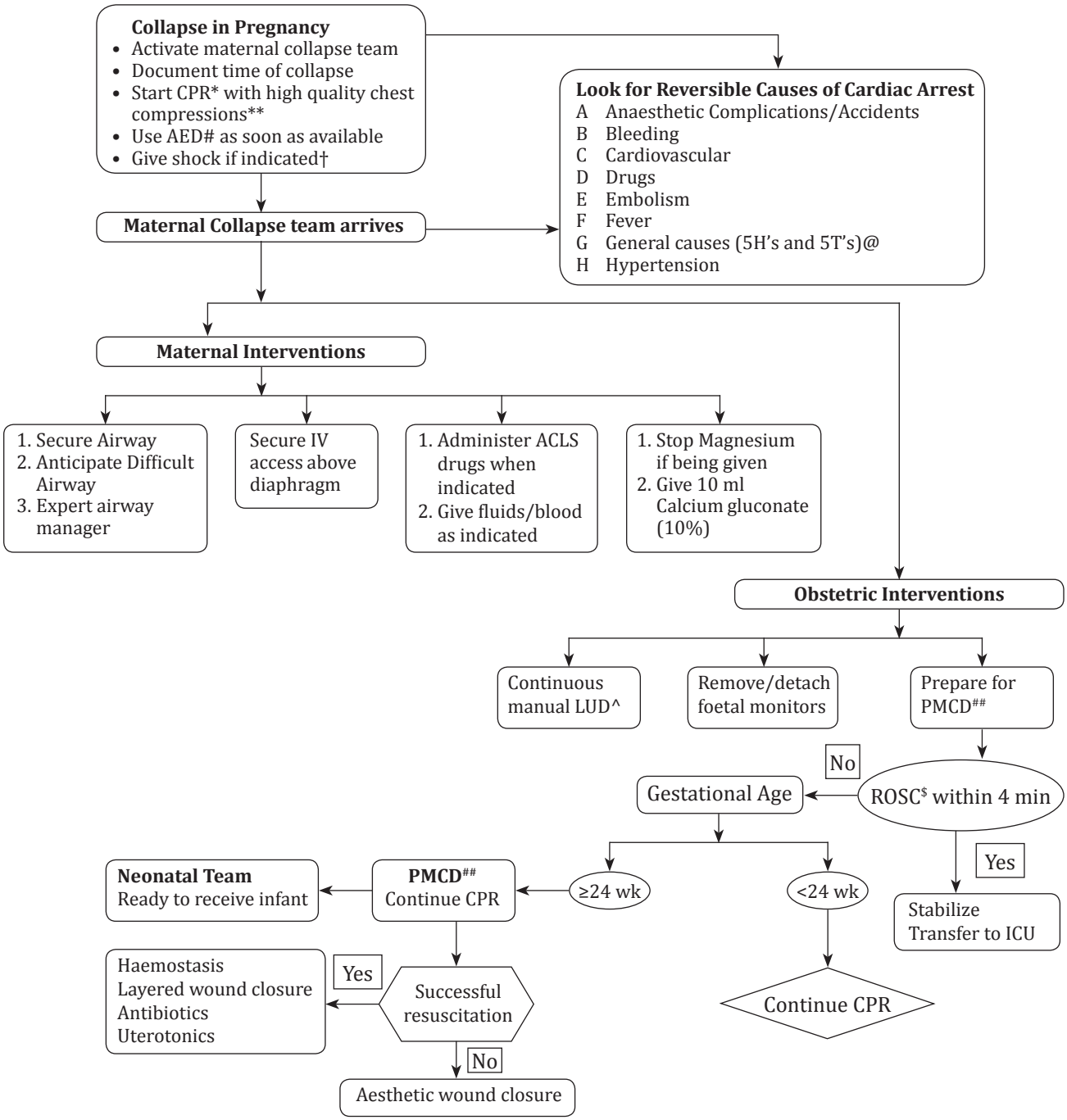
Dr Akanksha Tripathi
Assistant prof. (OBGY)
Pacific Medical College & Hospital,
Laparoscopic surgeon & infertilty specialist,
Pacific IVF centre & Mewar Hospital, Udaipur (Rajasthan)

Calendar of Monthly Clinical Meetings 2017-2018

Months	Name of the Institute
30th June 2017	Army Hospital- Referral and Research
28th July 2017	AIIMS
25th August 2017	VMMC & Safdarjung Hospital
29th September 2017	Hindu Rao Hospital
27th October 2017	ESI Hospital, Basaidarapur
24th November 2017	MAMC & LN Hospital
29th December 2017	Sir Ganga Ram Hospital
19th January 2018	Dr RML Hospital
23rd February 2018	Lady Hardinge Medical College
23rd March 2018	UCMS & GTB Hospital
27th April 2018	Apollo Hospital, Sarita Vihar

SOP: Basic Life Support and Maternal Resuscitation

Rashmi Salhotra
Associate Professor, Department of Anaesthesia, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi



*CPR: Cardiopulmonary resuscitation; #AED: Automated external defibrillator; ##PMCD: Perimortem Caesarean Delivery; ^LUD: Left uterine displacement; $ROSC: Return of spontaneous circulation

Causes of Cardiac Arrest

- All causes as enumerated above (A-H)
- General Causes: @ (5H's and 5T's)
  - Hypoxia
  - Hypovolemia
  - Hypo/hyperkalemia
  - Hypo/hyperthermia
  - H^+ ion (Acidosis)

- Toxins/tablets (poisoning/toxaemia of pregnancy)
- Tamponade (Cardiac)
- Tension pneumothorax
- Thrombosis (Cardiac)
- Thromboembolism (pulmonary)

**High Quality Chest Compressions in Pregnancy

- Place patient on a firm surface in supine position
- Site: Lower half of the sternum in the center of the chest
- Push hard and push fast
- Rate: 100-120/min
- Compression: Ventilation ratio=30:2
- Allow complete chest recoil
- Depth: 2-2.4 inch
- Minimize interruptions between compressions (<10s)
- Perform continuous manual Left Uterine Displacement (LUD)
- Breathing/ventilation: 2 breaths after every 30 compressions; each over a period of 1s, allowing 1s for exhalation

†Indications for Shock

- AED prompts to deliver shock
- Shockable rhythm on the monitor
  - Ventricular Fibrillation (VF)
  - Pulseless Ventricular tachycardia (VT)
- *No shock indicated for Asystole (flat line) or Pulseless Electrical Activity (PEA)*

Drugs for ACLS

Adrenaline

Indications:

1. Cardiac Arrest from VF
2. Pulseless VT unresponsive to first shock
3. Asystole
4. Pulseless Electrical Activity (PEA)
5. Symptomatic Bradycardia

Dose: 1 mg (diluted to 10 ml to make 1:10,000) IV stat followed by 20 ml flush and limb elevation for 10s, every 3 - 5 min

Amiodarone

Indications:

1. Persistent VT or VF after shock and adrenalin
2. Haemodynamically stable VT
3. Haemodynamically stable polymorphic VT
4. Haemodynamically stable wide-complex tachycardia of uncertain origin

Dose:

- VF / pulseless VT - 300 mg IV bolus followed by 150 mg IV
- In stable ventricular and supraventricular dysrhythmias - administer 150 mg IV over 10 - 15 min (not to exceed 30 mg/min), followed by an infusion of 1 mg/min over 6 hr, followed by 0.5 mg/min IV over next 18 hr

Adenosine

Indications:

1. Termination of paroxysmal supraventricular tachycardia (PSVT) (Re-entry type)
2. Supraventricular tachycardia (SVT)

Dose:

- 6 mg bolus IV over 1 - 3 sec, followed immediately by 20ml saline flush; preferably via antecubital or central vein
- If unsuccessful, give 12 mg bolus (maximum total dose of 30 mg)

Atropine

Indications:

1. Symptomatic bradycardia

Dose: 0.6 mg IV stat; may be repeated at 3 – 5 min intervals up to a max dose of 3.0 mg

Other Drugs

Lignocaine, Verapamil, Diltiazem, Magnesium: To be administered under the supervision/advice of physicia/ ACLS provider

Suggested Reading

1. Jeejeebhoy FM, Zelop CM, Lipman S, Carvalho B, Joglar J, Mhyre JM, et al. Cardiac Arrest in Pregnancy: A Scientific Statement From the American Heart Association. *Circulation*. 2015;132.
2. Lavonas EJ, Drennan IR, Gabrielli A, Heffner AC, Hoyte CO, Orkin AM, Sawyer KN, Donnino MW. Part 10: special circumstances of resuscitation: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(suppl 2):S501–S518.

Amniotic Fluid Embolism: An obstetrician's challenge

Rashmi[1], Anju Bala[2]

[1]Associate Professsor, [2]Senior Resident, Department of Obstetrics & Gynecology, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

Introduction

Amniotic fluid embolism (AFE) has been obstetrician nightmare since it was first described in 1941 by Steiner and Lushbaugh. It is described as one of the five major direct causes of maternal mortality in developed countries. But to this date the syndrome remains most enigmatic condition in obstetrics as it is still considered an unpredictable and unpreventable event with an unknown cause. To create awareness about AFE even a day i.e. 27th March has been designated as AFE Awareness Day.

Incidence

The reported incidence varies widely from 2-8 per 1,00,000 in different countries. Case fatality rates ranges from 11 to 43%. Between 5 and 15% of all maternal deaths in developed countries are caused by AFE. More than half of the patients (56%) die in the initial phase (0-23 h after initial clinical manifestations).

Etiopathogenesis

The pathophysiology is incompletely understood. The onset of AFE requires two necessary conditions:

1. An influx of fetal components into the maternal circulation
2. A significant pulmonary embolus or maternal immune/ anaphylactoid reaction against the amniotic fluid or fetal components.

Mechanical Obstruction Theory

Amniotic fluid (AF) can enter the maternal circulation via endocervical veins, lesions of the uterus, or the site of placental attachment and was once thought to cause a purely mechanical obstruction of the pulmonary vessels as hypothesized by Steiner and Luschbaugh. Introduction of pulmonary artery catheter into critical care obstetrics in 1980s refuted this hypothesis. Several reports documented fetal cells / adult squamous cells in pulmonary circulation in pregnant women with variety of conditions unrelated to amniotic fluid embolism.

Anaphylactoid reaction hypothesis

According to this hypothesis, fetal antigens entering maternal circulation activate pro-inflammatory mediators similar to seen in systemic inflammatory response syndrome (SIRS) leading to clinical presentation of AFE. As this is non IgE mediated response, AFE is also known as Anaphylactoid Syndrome of Pregnancy. AF contains vasoactive (bradykinin, histamine, and others) and procoagulant substances that can lead to endothelial activation and cause a massive inflammatory reaction mediated via Mast cell degranulation. The detection of a significantly higher number of mast cells and significantly higher levels of tryptase at pulmonary level in fatal AFE cases supports this mechanism. The other mechanism suggested is via activation of complement system. Abnormally low levels of C3 and C4 detected in AFE cases are suggestive of complement activation, either through the classical or alternative pathway.

Some women may tolerate the transfer of amniotic fluid or its components with no problems if an anaphylactoid reaction is adequately prevented by biological inhibitors such as the C1 inhibitor. The balance between the inflow amount and quality of amniotic fluid and the potential of biological inhibitors may contribute to the occurrence of AFE with variable severities and conditions.

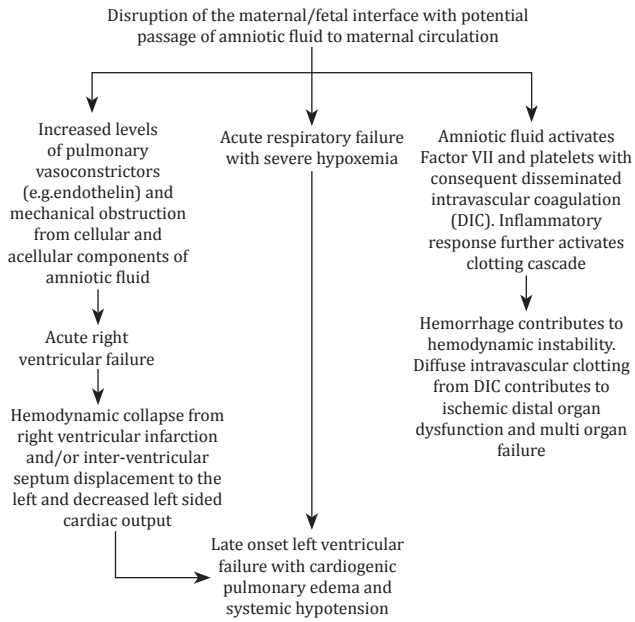


Figure 1: Proposed pathophysiology of AFE

The initial respiratory reaction possibly begins with a transient pulmonary vasospasm. The first phase of AFE develops within 30-60 min after the onset of clinical symptoms. Vasospasm may be caused by amniotic micro-emboli that trigger the release of arachidonic acid

metabolites and lead to pulmonary vasoconstriction and acute pulmonary hypertension leading to acute right heart failure with dilatation of the right ventricle and severe tricuspid insufficiency. Altered pulmonary perfusion and damage to the gas exchange surfaces caused by inflammation result in respiratory failure.

In the second phase of AFE, reactive hypovolemia, cardio-depressive humoral factors from the AF and myocardial ischemia may cause acute left heart failure with consequent pulmonary edema (51% to 100% of cases).

The third manifestation is a neurological response and multiorgan failure due to subsequent hypotension and hypoxia, which may include seizures, confusion, or coma.

In 30% to 45% of patients coagulopathy develops with severe bleeding resulting from disseminated intravascular coagulation (DIC). The procoagulant substances contained in the AF may activate the extrinsic coagulation cascade or urokinase-like plasminogen activator and plasminogen activator 1 contained in the AF may trigger massive hyperfibrinolysis.

Proposed mechanism is summarised in figure 1.

Pathophysiology of Uterine Type of AFE (Atypical AFE)

This is a condition presenting with PPH of unknown aetiology secondary to uterine atony with evidence of fetal components in the uterine vessels with no evidence of amniotic components in the lung. The local flow of amniotic fluid into uterine tissues may cause ananaphylactoid reaction in the uterus, resulting in an edematous uterus. These features of "Postpartum acute myometritis (PAM)" has been proposed as a histological characteristic in uterine-type AFE.

Predisposing Factors

Predisposing factors have been thought to be conditions where fetal tissue comes in contact with maternal circulation through one of the three routes – uterine trauma sites, endocervical veins or placental attachment sites. Table 1 lists the risk factors associated with amniotic fluid embolism. However, no demographic or clinical risk factor has been identified to justify alteration of standard obstetrics practice to reduce the risk of amniotic fluid embolism.

Table 1: Risk factors associated with amniotic fluid embolism

Age>35 years	Cervical trauma	Preeclampsia
Mutliparity	Uterine rupture	Eclampsia
Male fetus	Uterine hyperstimulation	Placenta previa
Instrumental delivery	Medical termination of pregnancy	Placental abruption
Caesarean section	Labour induction	multiple pregnancies

Clinical Presentation

The symptoms are often sudden. The presenting signs and symptoms of AFE involve many organ systems. The classical triad of symptoms of amniotic fluid embolism is hypoxia, circulatory collapse and coagulopathy with onset during labor or immediately after delivery.

AFE occurs during labor and delivery/Cesarean section (55% to 76%) or up to 48 h postpartum. It may rarely occur during pregnancy following intrauterine surgery (e.g. abortion), blunt abdominal trauma or amniocentesis. According to recent UKOSS data, AFE presented at or before delivery in 53% of women, at a median gestation of 39 weeks (range 28-42 weeks); 47% presented with AFE a median of 19 min after delivery (range 1 min to 6 h 27 min) having delivered at a median gestation of 39 weeks (range 28-42 weeks).

Recently, AFE has been categorized into two types:

Typical, (classic) with three phases: Phase 1-respiratory and circulatory disorders, Phase 2-coagulation disturbances of maternal hemostasis, Phase 3-acute renal failure and acute respiratory distress syndrome (ARDS), and leading to cardiopulmonary collapse.

Atypical: In contrast to typical embolism, cardiopulmonary collapse does not occur in atypical embolism but the first symptom is life threatening hemorrhage due to DIC. Atypical embolism was observed during caesarean section or immediately after it, in cases of profound rupture of uterine cervix, as well as in the course of placenta abruption and in association with induced midtrimester abortion.

The initial symptoms may be preceded by a non-specific prodromal phase or develop suddenly. Acute dyspnea or sudden agitation, sudden chills, shivering, sweating, coughing and anxiety are common premonitory symptoms.

The presenting symptoms may include acute dyspnoea (30-40%) and cyanosis (50% to 80%); sudden hypotension (56% to 100%), cardiac arrest (30% to 87%), or fetal distress (20% to 36%). Rarer clinical manifestations are: seizures, acute confusion and, in extreme cases, unconsciousness/coma (15% to 50%) or life-threatening haemorrhage resulting from coagulopathy (≤ 12%). Eventually DIC is present in more than 83% of patients with AFE. The onset can occur as quickly as 10-30 min from the onset of symptoms or may be delayed by as long as 4 h. The coagulopathy of amniotic fluid embolism may occur in conjunction with the cardiopulmonary manifestations, be manifest only after initial cardiopulmonary resuscitation has been completed, or in very rare cases may be the only finding in women without cardiorespiratory compromise.

Few researchers have proposed that two-thirds of AFE cases present with atonic bleeding, and only one-third with cardiopulmonary collapse.

Diagnosis

Amniotic fluid embolism is a clinical diagnosis and is primarily a diagnosis of exclusion. Other similar conditions need exclusion before making a diagnosis of amniotic fluid embolism Table 2. Detection of fetal cells in pulmonary circulation is no longer considered diagnostic of amniotic fluid embolism.

Laboratory Investigations

Hemodynamic parameters, ECG, blood gas analysis, chest X-ray and laboratory tests (including blood count, cardiac enzymes, and coagulation tests) and specific tests such as trans-esophageal echocardiography (TEE) and rotational thromboelastometry play a limited role in diagnosis and should be used instead for monitoring and treatment optimization.

1. ***Pulse oximetry and arterial blood gas (ABG) measurements*** to determine the degree of hypoxemia. ABG levels will show changes consistent with hypoxia/ hypoxemia (Decreased pH, decreased PO2, Increased PCO2 levels, Base excess increased).
2. ***Serial complete blood counts and coagulation studies*** should be sent to follow trends and detect early coagulopathy. Hemoglobin and haematocrit levels should be within reference ranges. Thrombocytopenia is rare. Among coagulation studies PT is prolonged and intervention is indicated when the PT is 1.5 times the control value. aPTT may be within reference range or shortened. If available, fibrinogen levels should be monitored.
3. ***Chest radiograph*** posteroanterior and lateral findings are usually nonspecific. The chief radiographic abnormalities in AFE are diffuse bilateral heterogeneous and homogeneous areas of increased opacity, which are indistinguishable from acute pulmonary edema.
4. A ***12-lead electrocardiogram*** may show tachycardia, ST-segment and T-wave changes, and findings consistent with right ventricle strain.

Diagnostic Markers

The search for specific laboratory markers for diagnosis continues. In fact, ***zinc coproporphyrin***, ***sialyl-Tn antigen***, ***tryptase*** or ***C3 and C4 complement*** and detection of insulin-like growth factor binding protein-1 appear promising diagnostic markers for AFE, but they have not been established in routine clinical diagnosis. Increased serum tryptase, urinary histamine concentrations and significantly lower complement concentrations suggest an anaphylactoid process. Tryptase has proven useful in the diagnosis of anaphylaxis. Decreased serum levels of C3 and C4 complement had sensitivities between 88% and 100% and a specificity of 100% for detection of inflammatory reaction. More studies are also needed to determine the utility of both monoclonal TKH-2 antibodies and zinc coproporphyrin as rapid diagnostic markers. Few authors reported a significant high serum level of STN antigen in the AFE cases.

Bedside transesophageal echocardiography may aid early diagnosis by showing acute pulmonary vasoconstriction, right ventricular dilation, and a collapsed left ventricle with leftward deviation of the intraventricular septum, but it is not easily available.

Though all these tests appear promising, the use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of amniotic fluid embolism is not recommended at present. Thus, the diagnosis of AFE is one of exclusion and should be considered in every case of sudden maternal cardiovascular collapse and/or maternal death in childbirth with unexplained aetiology (Grade 1C recommendation).

Table 2: Differential diagnosis of Amniotic Fluid Embolism

Non obstetric causes	Obstetric causes	Anesthesia related
Pulmonary embolism Pulmonary edema Tension pneumothorax Cardiac- myocardial infarction, heart failure, arrthymias, tamponade Anaphylaxis Septic shock Aspiration	Eclampsia Abrutio placenta Uterine rupture Postpartum hemorrahge Peripartum cardiomyopathy	local anesthesitic toxicity high spinal

Management

Prevention: To prevent AFE, trauma to the uterus must be avoided during manoeuvres such as insertion of a pressure catheter or rupture of membranes. Incision of the placenta during caesarean delivery should also be avoided if possible. Since, one of the most frequent predisposing factors is considered to be tumultuous labor that may occur naturally, excessively strong and frequent uterine contractions should be controlled by administration of intravenous β-adrenergic drugs or magnesium sulfate. Furthermore, oxytocic drugs, which can precipitate excessive tetanic uterine contractions must be used appropriately and judiciously.

The key factors in the management of AFE are early recognition, prompt resuscitation, and delivery of the fetus. It is not necessary to diagnose amniotic fluid embolism before starting treatment. Management is primarily supportive. Immediate resuscitation is instituted and managed in intensive settings. The mainstay of the treatment includes the following procedures:

- **Maintaining vital signs.** The initial goal is the rapid correction of maternal hemodynamic instability, which includes a correction of hypoxia and hypotension, for preventing the additional hypoxia and subsequent endorgan failure.
  1. ***Safeguard the airways***: endotracheal intubation and early sufficient oxygenation should be performed using an optimized Fi02: PEEP (positive end-expiratory pressure) ratio.
  2. ***Fluid based resuscitation*** is imperative to counteract hypotension and hemodynamic instability. Treatment of hypotension includes optimization of preload, with rapid volume infusion of isotonic crystalloid and colloids solutions.
  3. ***Pharmacological*** Vasopressors and inotropic support are generally needed to varying degrees in AFE. Central venous access should be established for vasopressor infusion and monitoring. Choice of drug depends on the clinical scenario. Epinephrine, Phenylephrine, Dopamine, Noradrenaline, Vasopressin may be used. Other specific interventions aimed at decreasing the pulmonary vascular resistance include sildenafil, inhaled or intravenous prostacyclin, and inhaled nitric oxide. Other drugs that may be used are Milrionone, Digoxin, Hydrocortisone.
- **Correction of Coagulopathy**—Blood and blood products, including fresh frozen plasma (FFP), platelets and cryoprecipitate, must be administered early in the resuscitation phase of AFE.
  a. If platelets are <20,000/μL, or if bleeding occurs and platelets are 20,000-50,000/μL, transfuse platelets at 1-3 U/10 kg/day.
  b. Administer FFP to normalize the PT if > 1.5.
  c. If fibrinogen level is <100 mg/dL, administer cryoprecipitate. Each unit of cryoprecipitate raises the fibrinogen level 10 mg/dL.
  d. In the setting of massive hemorrhage, blood product administration should not be delayed while awaiting the results of laboratory tests. Instead, early aggressive resuscitation with packed red blood cells, fresh-frozen plasma, and platelets at a ratio of 1:1:1 (hemostatic resuscitation) results in improved outcomes.
  e. PPH is managed with Oxytocin, Methergin & Prostaglandins. Other antifibrinolytic drugs, such as aminocaproic acid and tranexamic acid can also be used. Aprotinin & Recombinant Factor VIIa have also been used in AFE associated hemorrhage. Due to risk of diffuse thrombosis and multiorgan failure, Factor VIIa may be considered as a last resort option in cases of uncontrollable hemorrhage.
- **Urgent delivery**: If the patient is undelivered at the time of cardiac arrest, expeditious delivery is indicated if the fetus has reached an age of potential viability (≥23 weeks). Not only may this be life saving for the fetus but in theory may assist in maternal resuscitation by removing venacaval compression. Perimortem cesarean delivery should be performed if there is failure to obtain spontaneous circulation after 4 minutes of cardiopulmonary resuscitation.
- **Novel treatment strategies for AFE based on the pathophysiology:** Antithrombin concentrates may improve outcomes in patients with AFE that develop coagulopathy Invasive hemodynamic support may be considered when institutionally available in patients unresponsive to initial resuscitative interventions. Extracorporeal membrane oxygenation, cardiopulmonary bypass, intra-aortic balloon pump, pulmonary artery thromboembolectomy, hemofiltration and plasma exchange transfusions have been the object of several case reports, but their safety and efficacy needs to be further tested. Plasma exchange may remove chemical mediators and cytokines responsible for the anaphylactoid response. High-dose corticosteroid treatment is also supposed to counteract the inflammatory reaction. Administration of C1 esterase inhibitor (C1INH) has also been proposed as a therapeutic option. Use of Sodium bicarbonate is suggested in suspected cases of AFE with Right heart failure not responding to advanced life support measures.

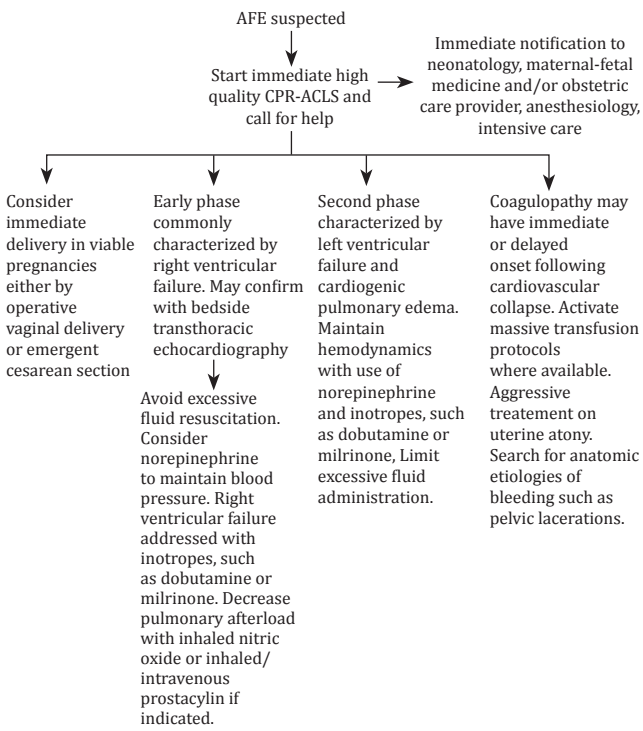


Figure 2: Immediate Supportive treatment in Cases of suspected AFE

Table 3: Summary of Recommendations

Number	Recommendation	Grade
1	Consideration of AFE in the differential diagnosis of sudden cardiorespiratory collapse in the laboring or recently delivered woman.	1C Strong recommendation Weak-quality evidence
2	Use of any specific diagnostic laboratory test to either confirm or refute the diagnosis of AFE is not recommended; at the present time, AFE remains a clinical diagnosis	1C Strong recommendation Weak-quality evidence
3	Immediate high-quality cardiopulmonary resuscitation with standard BCLS and ACLS protocols in patients who develop cardiac arrest associated with AFE.	1C Strong recommendation Weak-quality evidence
4	Multidisciplinary team including anesthesia, respiratory therapy, critical care, and maternal-fetal medicine should be involved in ongoing care of women with AFE.	Best practice
5	Following cardiac arrest with AFE immediate delivery in the presence of a fetus >23 weeks of gestation.	2C Weak recommendation Weak-quality evidence
6	Provision of adequate oxygenation and ventilation and, when indicated by hemodynamic status, the use of vasopressors and inotropic agents in the initial management of AFE. Excessive fluid administration should be avoided.	1 C Strong recommendation, Weak-quality evidence
7	Because coagulopathy may follow cardiovascular collapse with AFE, early assessment of clotting status and early aggressive management of clinical bleeding with standard massive transfusion protocols.	1C Strong recommendation, Weak-quality evidence

Conclusions

Amniotic fluid embolism is a rare but often lethal condition. Maternal and perinatal mortalities appear to have decreased during the last decades likely because of improvements in the delivery of critical care, recognition of atypical or milder cases with no cardiorespiratory collapse, and the likely inclusion of patients with conditions other than amniotic fluid embolism.

The diagnosis remains clinical and is often one of exclusion because no single specific diagnostic test is currently available. Treatment is mainly supportive and involves the delivery of the fetus when indicated, respiratory support (usually in the form of endotracheal intubation and mechanical ventilation), and hemodynamic support with the judicious use of fluids, vasopressors, inotropes, and pulmonary vasodilators. Rapid initiation of treatment, aided by a high index of clinical suspicion, is essential. Uniform diagnostic criteria for amniotic fluid embolism cases reported in research publications are badly needed and may accelerate our understanding of this condition.

Suggested Reading

1. Naoaki T, Mustari F, Tomoaki O, et al. Amniotic fluid embolism: Pathophysiology from the perspective of pathology. J. Obstet. Gynaecol. Res. 2017; 43(4): 627–632.
2. Society for Maternal-Fetal Medicine (SMFM) with the assistance of Pacheco LD, Saade G,et al. Amniotic fluid embolism: diagnosis and management. Am J Obstet Gynecol 2016; 215:B16-24.
3. Piva I, Scutiero G, Greco P. Amniotic Fluid Embolism: An Update of the Evidence. Med Toxicol Clin Forens Med. 2016, 2:2.
4. Kramer MS, Abenhaim H, Dahhou M, Rouleau J, Berg C. Incidence, risk factors, and consequences of amniotic fluid embolism. Paediatr Perinat Epidemiol 2013;27:436-41.
5. Clark SL. Amniotic fluid embolism. Obstet Gynecol 2014; 123:337-48.
6. Johnston TA, Grady K. Maternal Collapse in Pregnancy and the Puerperium. Green-top Guideline No. 56. London (UK): Royal College of Obstetricians and Gynaecologists; 2011. Available from: https://www.rcog.org.uk/globalassets/documents/guidelines/gtg_56.pdf.

Obstetrician Alert!!!

According to World Health Organization, India has reported three confirmed cases of Zika virus. from Gujarat. Cases were detected during testing in February and November last year, while one was detected in January this year.

WHO in its statement further added that "Zika virus is known to be circulating in South-East Asia Region and these findings do not change the global risk assessment. These findings suggest low level transmission of Zika virus and new cases may occur in the future and efforts should be made to strengthen surveillance."

Zika virus leads to birth defects like microcephaly and other neurological problems in newborns. It is of great worry in our country as India provides fertile climate for the aedes egypti mosquito to grow and multiply. With dengue and chikungunya viruses carried by same mosquito claiming 100s of lives every year, there is a potential of an outbreak situation of Zika in the country. Also, birth rate in India is high and special care needs to be taken.

Forthcoming Events

- "IUI Workshop" under the aegis of AOGD on 2nd June, 2017 by Arogya Hospital at Hotel Woodapple, Delhi.
- "AOGD Monthly Clinical Meeting", 30th June, 2017, Army Hospital (R&R) Delhi Cant, Sushruta Hall, 2nd Floor (above Accident & Emergency), contact Brig BK Goyal, 9831423985
- Skill Workshop on "Basics of Endoscopy in Gynecology" on 21st July, 2017, 11:00am-5:00pm, 7th Floor MCH Block, GTB Hospital.
- "Challenges in Management of Preterm Labour" on 23rd July, 2017 in Army Hospital by Dr BK Goyal and Team.
- "CME on Women's Reproductive Health", 12th August, 2017, 2.00pm-5.30pm, contact Dr. Nalini Mahajan, dr.nalinimahajan@gmail.com for further details
- "BOH- The Trilogy 2017" on 19th & 20th August, 2017 focused on current practices, breakthrough and current dilemmas on BOH patients by FOGSI - AOGD, Leela Ambience, Gurugram.
- DGES (Delhi Gynecological Endoscopists Society) Annual Conference & IAGE (Indian Association of Gynecological Endoscopists- NZ) in association with AOGD on 25th-27th August, 2017 at India Habitat Centre, Lodhi Road; PreCongress workshop on 25th August.
- "39th AOGD Annual Conference" on 18th and 19th November, 2017 at India Habitat Centre; Pre- conference workshops on 17th November 2017.

AOGD Sub-Committee Chairpersons

Congratulations to the newly elected chairpersons of AOGD sub- committees for the period 2017-19. All interested AOGD members working in the field may contact the concerned chairperson to become members of respective sub-committees.

Sub - Committee	Chairperson	Contact No.	E-mail
Urogynaecology Committee	Dr Amita Jain	9871136110	amita_jain75@yahoo.com
Adolescent Committee	Dr Shakuntala Kumar	9811445853	numesh_in@yahoo.com
Safe Motherhood Committee	Dr Ashok Kumar	9968604346	ash64kr@yahoo.com
Fetal Medicine & Genetics Committee	Dr Vatsla Dadhwal	9868397308	vatslad@hotmail.com
Oncology Committee	Dr Rupinder Sekhon	9810163076	rupysekhon@hotmail.com
Endoscopy Committee	Dr Anjali Tempe	9968604343	anjalitempe@hotmail.com
Endometriosis Committee	Dr Renu Misra	9811147217	drrenumisra@gmail.com
Reproductive Endocrinology Committee	Dr Nalini Mahajan	9810087666	nalinimahajan@hotmail.com

All AOGD members are directed to become members of only one sub-committee and not several sub-committees, as has been observed in the past, so that they can contribute meaningfully to the committee concerned.

Existing AOGD Subcommittee Chairpersons 2016 - 2018			
Sub - Committee	**Chairperson**	**Contact No.**	**E-mail**
Breast Cancer Prevention	Dr Sunita Malik	9818914579	svmalik@yahoo.com
Cervical Cancer Awareness and Prevention	Dr Mala Srivastava	9811228336	malasrivastava2001@yahoo.co.in
Infertility	Dr K D Nayar	9810398765	kdnayar@usa.net
Rural Health	Dr Achla Batra	9811105560	achla_batra@yahoo.com
Multidisciplinary Patient Sub-committee	Dr Jyotsna Suri	9810858358	jyotsnasuri@gmail.com

Events Held in May 2017

- AOGD 1st Skill workshop on Basic Life Support and Maternal Resuscitation was organized by the Department of Obstetrics and Gynaecology, GTB Hospital on 6th May 2017. Each lecture was followed by hands on training on mannequins. A total of 51 delegates attended the programme. It was well appreciated by the participants.

Workshop on Maternal Resuscitation at GTB Hospital

- CME on Interstitial cystitis and bladder pain syndrome organized by Global Interstitial cystitis/ Bladder pain syndrome society (GIBS) and endorsed by ICOG under the aegis of Urogynae subcommittee of AOGD on 6th May at Apollo Hospital, Sarita Vihar. It was attended by 85 delegates and was greatly appreciated.

CME on Interstitial cystitis and bladder pain syndrome at Apollo Hospital, Delhi

- CME on Menopause Management - An Executive Guide on 12th May 2017 (Friday), 12:00-5:00pm at Auditorium, Sir Ganga Ram Hospital, New Delhi.

CME on Menopause Management at Sir Ganga Ram Hospital

- 1st Endometriosis Summit Organized by Endometriosis Committee, FOGSI in association with AOGD and SIG-Endometriosis, IFS on 14th May 2017 at Silver Oak Hall IHC.

1st Endometriosis summit organized by Endometriosis Committee

- CME on 'Antenatal care: Best practices' by Dept. of Obs and Gynae UCMS and GTB Hospital on 19th May, 2017 attended by 101 delegates.

CME on 'Antenatal Care: Best Practices' at GTB Hospital

- AOGD Monthly Clinical Meeting at Fortis Vasant Kunj on 26th May, 2017. Very interesting cases including audit on thromboprophylaxis in gynecologic surgery presented.

AOGD Monthly Clinical Meeting at Fortis Vasant Kunj

DGES (Annual Conference) & IAGE (NZ) 2017

Delhi Gynaecological Endoscopists Society (DGES)

Indian Association of Gynaecological Endoscopists (IAGE)

In association with

Association of Obstetrician & Gynaecologists of Delhi (AOGD)

25th -27th August, 2017

25th August, 2017 : Pre Congress Workshops

- **Laparoscopic Endosuturing Training** -Apollo Spectra Hospital, New Delhi
- **Basic Hysteroscopy Training** -Sita Ram Bhartiya Hospital, New Delhi

26th & 27th August: Indian Habitat Centre, Lodhi Road, New Delhi

26th August: Live Surgical Workshop, Relay from Apollo Spectra Hospital, New Delhi

27th August: Scientific Session

Operating Faculty

Dr. Alka Kriplani
Dr. B. Ramesh
Dr. Dinesh Kansal
Dr. Hafeez Rahman
Dr. Krishan Kumar
Dr. Malvika Sabharwal
Dr. Prakash Trivedi
Dr. Rajendra Sankpal
Dr. Sanjay Patel
Dr. Shailesh Putambekar
Dr. Sunita Tandulwadkar
Dr. Vivek Marwah

Planned Surgeries*

- **TLH - with different vessel sealers**
  Large Uterus/ In Scarred abdomen/ Endometriosis/
  Pelvic Anatomy Demonstration
- **Laparoscopic Myomectomy**
- **Laparoscopic Adenomyoma Resection**
- **Laparoscopic Radical surgery with Pelvic node dissection**
- **Laparoscopic Burschcolposuspension**
- **Laparoscopic Sacrocolpopexy / Hysteropexy**
- **Laparoscopic Dermoid cystectomy**
- **Laparoscopic Endometriotic clearance**
- **Hysteroscopic Myoma Resection**
- **Hysteroscopic Adhesiolysis**
- **Hysteroscopic Septal Resection**
- **Hysteroscopic Tubal Cannulation**

*Cases Subject to Availability

Registration Fee (26th, 27th August - Surgical Workshop and Scientific Session)

Dates	DGES Members	Non Members	PG
Upto 15th July, 2017	4000	4500	2500
16th July - 05th August, 2017	4500	5000	3000
Late & Spot	5500	6500	3000

Registration Fees for 25th August, 2017 - Pre Congress Workshop - Rs.1500 (Limited Registration)

Conference Secretariat:

Jeewan Mala Hospital
67/1 New Rohtak Road, New Delhi-110005
(L): 011-40043355 (M): 9212150571, 9212526855, 9811557511
E-mail: dgesjmh2017@gmail.com • Website: www.dges.in

Recent Advances in Management of Post Partum Haemorrhage

Esha Gupta[1], Richa Aggarwal[2]

[1]Senior Resident, [2]Assistant Professor, Department of Obstetrics and Gynaecology, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi

Introduction

Postpartum hemorrhage (PPH) is the leading cause of maternal mortality worldwide, with an estimated mortality rate of 140,000 per year or one maternal death every four minutes. It is also a major contributor to maternal morbidity, such as anemia. Its reported incidence in India is 2% - 4% after vaginal delivery and 6% after caesarean section with uterine atony being the most common cause (50%). It contributes to 19.9% of maternal mortality in India.

Etiology of PPH is traditionally referred to as the '4 Ts': tone, trauma, tissue and thrombin. 'Tone' describes uterine atony, responsible for approximately 70% of cases. Genital tract 'trauma' is responsible for about 20% of PPH. 'Tissue' etiologies including retained placenta and abnormal placentation are responsible for 10% of cases. 'Thrombin' refers to inherited or acquired coagulation disorders including dysfunctions of the clotting cascade or platelets, and disseminated intravascular coagulopathy (DIC), which cause approximately 1% of PPH.

Diagnosis

Early diagnosis is the key to proper management of PPH. Majority of PPH occurs without warning. Frequent monitoring of vital signs and palpation of the uterine fundus after delivery is recommended to identify PPH development. The gold standard for blood loss estimation is photospectrometry or colorimetric measurement of alkaline hematin but is impractical in clinical settings. Visual estimation is the most common method of quantifying blood loss, however, it underestimates blood loss to an extent of 30 to50%. A calibrated, plastic, closed-ended under buttock blood-collection drape improves valid estimation Figure 1 .

Figure 1: Blood Collection Drape

Trigger systems have been designed for early diagnosis of PPH. The California Maternity Quality Care Collaborative (CMQCC) has proposed designated values for alert and action lines (e.g., heart rate±110 bpm, blood pressure (BP) 85/45 mmHg and oxygen saturation <95%), and the UK Confidential Enquiry into Maternal and Child Health (CEMACH) developed an 'Obstetric Early Warning Chart' to alert providers when either one markedly abnormal observation or a combination of two mildly abnormal observations for the vital signs are being found (e.g., respiratory rate, O2 saturation, temperature, heart rate, BP). The shock index, a combined measure of pulse and systolic blood pressure (pulse/systolic BP) has been found to have clinical utility for early diagnosis of hemorrhage in a recent systematic review. A low cost application for mobile phones is being developed to estimate blood loss using the camera of the phone and a built in algorithm to provide real-time blood loss monitoring.

Prevention

PPH is preventable entity. Active management of third stage of labour (AMTSL) is the cornerstone in PPH prevention. WHO recommends prophylactic uterotonic administration during the third stage of labor, with oxytocin (IM/IV, 10 IU) being the preferred drug. Where oxytocin is unavailable ergometrine/methlergometrine or oxytocin/ergometrine or oral misoprostol (600 mcg) can be used. In low resource settings in the absence of skilled workers, oxytocin in a Uniject system, an easy-to-use, prefilled, single-dose injection with a fixed needle has been found to be safe and feasible for AMTSL. Pharmaceutical development of powdered, heat-stable oxytocin that can be inhaled is also being developed for an aerosol delivery system to remove the need for cold supply chain, sterile conditions and trained health workers. Recent literature suggests that carbetocin, a long acting oxytocin analogue is equally efficacious as oxytocin with decreased need for subsequent uterotonic administration, less blood loss, fewer adverse effects and greater cost effectiveness than syntometrine.

Medical Management

Pharmacologic management of atonic PPH includes the use of oxytocin, ergometrine and prostaglandins. If

bleeding is unresponsive to uterotonics, consideration may be given to tranexamic acid (TXA), a synthetic derivative of lysine with antifibrinolytic properties, or recombinant activated factor VII (rvFIIa).

A 2010 Cochrane Review of TXA reported decreased blood loss after vaginal and cesarean birth but called for further investigation around efficacy and safety. The recently concluded WOMAN (World Maternal Antifibrinolytic) Trial has supported the use of TXA for PPH treatment.The administration of tranexamic acid to women with PPH reduces deaths due to bleeding and laparotomy to control bleeding with no evidence of any adverse effects. When given soon after delivery, mortality reduction is by 30%.

Recombinant Activated Factor VII (rFVIIa) is an effective, yet expensive, synthetic agent used to control bleeding among patients with hemophilia and factor VII deficiency but is now used in refractory PPH also. The effectiveness of rFVIIa is dependent upon adequate fibrinogen and platelets. rFVIIa assists hemostasis in PPH patients with bleeding refractory to pharmacologic management and uterus sparing surgical techniques (e.g., uterine and hypogastric artery ligation). It should be given only when hematocrit is adequate, platelet count is >50x109/l, fibrinogen >1 gm/l, pH>7.2 and temperature >34°C. Dose is 90 µg/Kg IV over 3-5 minutes, repeated only if necessary. Its use may lead to thrombotic complications.

Mechanical Procedures

These include uterine massage, uterine packing and tamponade. The use of uterine massage for treatment of PPH is strongly recommended by WHO and FIGO. Uterine packing is no longer recommended. However, WHO recommends the use of intrauterine balloon tamponade (IUB) for atonic PPH unresponsive to uterotonics or when uterotonics are unavailable. Uterine balloons such as the Sengstaken tube, Bakri and Rusch balloons are available but are expensive for use in low-resource areas. The commercially available uterine-specific devices are designed with an intrauterine drainage port but have a prohibitively high cost. Low resource settings have to rely on lower cost adaptations like condom balloon tamponade which is the most cost-effective option.

The condom balloon tamponade has two main disadvantages. First, it does not having a drainage port and therefore the clinician cannot assess the actual blood loss and secondly, the thread or suture is used to tie the condom to the catheter which often causes leakage of saline. An innovative variation of condom balloon catheter developed in India is **"CG Balloon" (CG is our state of Chhattisgarh in India).**

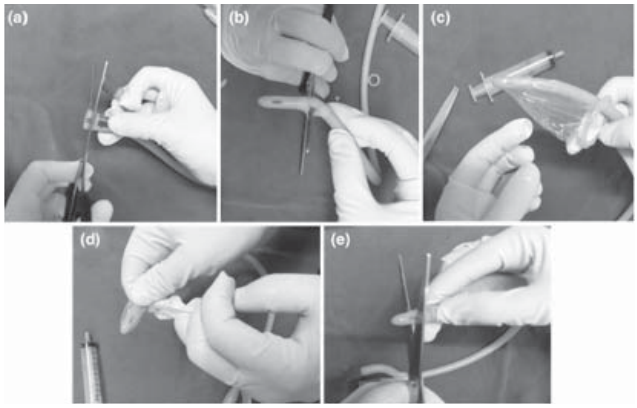


Figure 2: Preparation of CG balloon

It is prepared manually with all aseptic precautions as follows (Fig. 2):

1. Collect a Foley's catheter of size 20–22, a packed condom, scissors, two 20-ml syringes and 500-ml bottle of saline in a tray.
2. From the drainage tube of the catheter, cut two rings of approximately 1–2 mm width (Fig. 2a).
3. Excise (not merely incise) the bulb of the catheter after inflating it with air (Fig. 2b).
4. Unfold the condom over distal one-third of the catheter (Fig. 2c).
5. Use these rings encircling twice only (like a rubber band in a ponytail) to secure the condom over catheter leaving 1.5–2 cm from both the ends of condom (Fig. 2d).
6. Excise the tip of the Foley's catheter and condom together to facilitate drainage of blood (Fig. 2e). Wash the device with antiseptic solution.

It overcomes the major disadvantages of conventional condom balloon tamponade by having a drainage channel for assessing the uterine bleeding. It uses rings cut out of catheter only and not the thread to tie the condom to catheter which avoids loose/too tight knots, saves time and is simple to use. CG Balloon is successful in 92.3 % cases. Larger trials are needed to confirm the findings.

The newly developed Belfort-Dildy Obstetrical Tamponade System Figure-3 has an upper uterine balloon approved for filling to 750 ml and a lower vaginal balloon for filling to 300 ml. It prevents displacement of the uterine balloon and obviates the need for vaginal packing.

Figure 3: Belfort-Dildy System

Various other modifications of condom catheter have been used including the use of surgical gloves in place of condom.

Surgical Management of PPH

It includes uterine compression sutures, vascular ligations and Peripartum Hysterectomy.

Uterine compression sutures: These compression sutures cause mechanical compression of the uterine vascular sinuses without occluding uterine arteries or uterine cavity. Several techniques like B-lynch, Cho-square sutures, Hayman sutures, Pereira suture, Cervico Isthmic Compression sutures etc. have been used. Complications like pyometra, endometritis, sepsis, ischemic uterine necrosis, uterine suture erosion, uterine synechiae have been reported. Uterine synechia has been reported with 18-24% frequency, which surely compromises fertility. The risk of potential complication appears to be higher when non absorbable sutures are used.

Recently, a removable uterine brace suture has been used which is supposed to be free from above said complications. It compresses uterus against the pubis. After uterine exteriorization and pelvic exploration, tamponade test is carried out to assess the effectiveness. The bladder peritoneum is reflected inferiorly. Using a number 2 sliding non-reabsorbable suture wire with 70mm round-bodied hand needle or with wire guide, the first stitch is applied from outside, running through the full thickness of anterior abdominal wall immediately above the pubis and 2cm laterally from the median line. The needle is then passed through the inferior uterine segment from the anterior to posterior wall as low as possible, under sutured hysterotomy, and 2cm inside from uterine artery cross. The wire is then passed over as abrace to compress the uterine fundus by approximately 3 or 4cm inside the cornua. Finally, the last stitch is applied from inside to outside through the abdominal wall 2cm above the first parietal stitch but 4cm laterally from the median line. The same procedure is performed on the other side. Finally, the right and the left lower suture extremities are tied anteriorly, followed by the upper extremities with added curves and compression of the uterus against the pubis Figure 4. Twenty four to forty eight hours later, the throws are cut, and sutures are removed by simple wire traction without any anaesthesia.

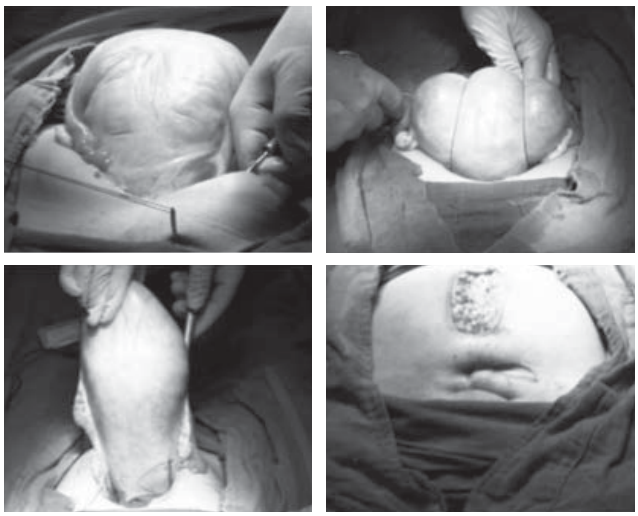


Figure 4: Removable Uterine Brace Suture

Vascular Ligations

Systematic devascularisation starting from bilateral uterine artery ligation followed by ovarian artery ligation followed by internal iliac artery ligation is performed in refractory PPH and prevents the need for hysterectomy. Peripartum hysterectomy is done as a last resort when all other methods to control PPH fail.

Pelvic packing Severe haemorrhage and emergency hysterectomy are often accompanied by secondary coagulopathy when bleeding may continue despite securing surgical pedicles. Abdominal and pelvic postsurgical packing is an old concept where a number of gauze bandages are tied end-to-end to pack the pelvis tightly and tamponade the hemorrhage. The free end of the gauze is extracorporealized through one end of the main incision and the abdomen is closed in the usual fashion. This procedure however, requires relaparotomy after initial stabilization to remove the packing materials.

A variation to this is to fill a sterile plastic bag (eg, drawstring bag used to cover x-ray film) or cloth container with gauze and place it against the pelvic bleeders. The drawstrings are pulled through the vagina and attached to a weight, which provides traction so that the pack exerts pressure against the pelvic floor Figure 5. This is known as umbrella, parachute, mushroom, pelvic pressure, or logothetopoulos pack. Re-laparotomy is usually not required and the pack can be removed transvaginally without anaesthesia. The pelvic pressure pack controls hemorrhage from large raw surfaces, venous plexuses and inaccessible areas by exerting well distributed pressure, compressing bleeding areas against the bony and fascial resistance of the pelvis.

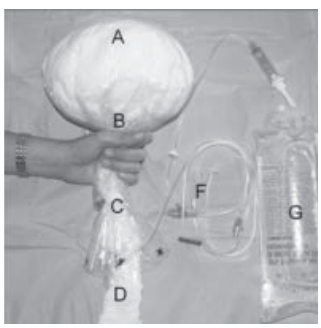


Figure 5: Umbrella Pack

Temporizing Measures for PPH Management

Temporizing measures recommended for intractable atonic and non-atonic PPH include external aortic compression, bimanual uterine compression and the non-pneumatic anti-shock garment (NASG).

External aortic compression significantly reduces blood flow to the pelvic organs. It has traditionally been accomplished manually by applying pressure with a closed fist on the abdominal aorta slightly to the patient's left and immediately above the umbilicus. Recently, the external aortic compression device (EACD), a hand-made spring device held in place by a leather belt, is being used as a first aid temporizing intervention.

Non Pneumatic anti shock garment (NASG) is a lightweight, re-usable lower-body compression garment made of neoprene and Velcro. It increases blood pressure by decreasing the vascular volume and increasing vascular resistance within the compressed region of the body, but does not exert pressure sufficient for tissue ischemia. It reverses shock by returning blood to heart, lungs and brain. This restores the woman's consciousness, pulse and blood pressure. Additionally, NASG decreases bleeding from the parts of the body compressed under it. It has been designed to allow perineal access so that examinations and vaginal procedures can be performed without it being removed Figure 6.

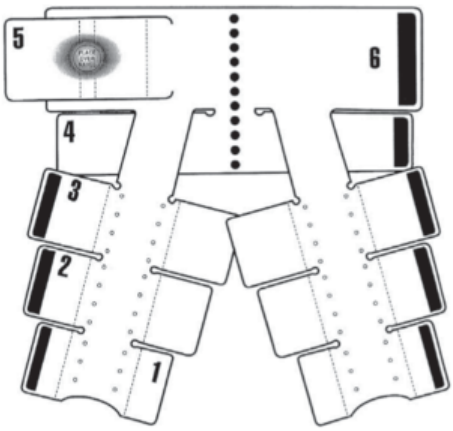


Figure 6: Non Pneumatic Anti Shock Garment

Application: Place NASG under woman; Close segments 1 tightly around the ankles; Close segments 2 tightly around each calf and segments 3 tightly around each thigh, leaving knees free. Segment 4 is closed around pelvis and segment 5 with pressure ball over the umbilicus. Finish closing the NASG using segment 6. Segments 1,2,3 can be applied by two persons, however, segments 4,5,6 should be applied by only one person. Removal of NASG occurs only when the source of bleeding is treated, the woman has been hemodynamically stable for at least 2 hours, and blood loss is less than 50 ml/hour. Removal begins at the ankles and proceeds slowly, waiting 15 minutes between opening each segment.

NASG seems a promising device to be used in developing countries for preventing maternal deaths as it allows additional time for referral and transfer to higher level of facility and also gives time to arrange blood. It is easy to use and does not require trained personnel. The relatively low cost together with the possibility of reuse makes this device an attractive option. However, it is not a definitive treatment and the need for substantive PPH treatment remains.

Radiological Management

Arterial balloon occlusion & embolization can prevent major blood loss, obviating the need for blood transfusion & hysterectomy. Arteries targeted for embolization are anterior division of internal iliac artery, uterine artery and ovarian artery and materials used for embolization are gel foam particles (larger >700 um) as they are temporary embolic agents. As an elective procedure, USG or fluroscopy guided arterial puncture of anterior division of internal iliac artery is done. Once the baby is delivered by CS, occlusion balloons should be inflated. Once haemostasis is achieved, balloons should be deflated and observed for bleeding. If bleeding does occur, the balloon should be reinflated and embolisation performed.

Intravascular Aortic Balloon Occlusion (IABO) has emerged as a prophylactic, simple, safe and minimally invasive method in the management of life threatening PPH and in the conservative management of abnormal placentation. An infrarenal intraaortic ballon is placed in aorta through femoral artery which is inflated after delivery of baby. To prevent reperfusion injury, it is deflated every 20-30 minutes for 2 minutes. It has similar results in terms of blood loss and absence of need of blood products as internal lilac artery occlusion. but requires further research before regarding this method as an ancillary procedure of choice during scheduled caesarean hysterectomy in known or suspected cases of abnormal placentation.

Transfusion Protocols for PPH

Conventional resuscitation follows a stepwise approach starting with intravenous fluids, followed by red blood cells (RBCs) and clotting factors or platelets. While this approach corrects hypovolemia, it worsens existing dilutional coagulopathy, enhances fibrinolysis and contributes to acidemia and hypothermia. Recent advances from trauma medicine suggest that increasing the ratio of fresh frozen plasma (FFP) to RBCs from 1:3 and 1:4 to 1:1 or 1:2 improves survival. The fibrinogen decrease seen in severe PPH is of great concern and is considered an early predictor of hemorrhage severity. Treatment of hypofibrinogenemia involves

cryoprecipitate transfusion to maintain fibrinogen levels (100–200 mg/dl). Fibrinogen concentrate contains a greater concentration of fibrinogen and is stable at room temperature and can be rapidly administered, unlike cryoprecipitate, which must be kept frozen and then thawed prior to administration. Rapid blood product selection may benefit from the use of a thromboelastograph, a point-of-care device that examines clot formation and dissolution in whole blood, and provides faster results than laboratory testing and its availability in labour rooms can significantly improve PPH management.

Conclusion

Timely diagnosis and treatment and prevention are the cornerstone in PPH management. Better training in the form of PPH teaching and drills can improve team preparedness for managing PPH. Greater use of obstetric warning systems and more precise identification of warning thresholds such as the shock index to trigger focused medical attention should expand across facilities.

Suggested Readings

1. Shakur H, Elbourne D, Gulmezoglu M, Alfirevic Z, Ronsmans C, Allen E, et al. [33] The Woma Trial (World Maternal Antifibrinolytic Trial): Tranexamic acid for the treatment of postpartum haemorrhage: An international randomized double blind placebo controlled trial. Trials 2010; 11: 40.
2. World Health Organization. WHO recommendations for the prevention and treatment of postpartum hemorrhage. (2012).
3. Usman N, Nobelet J, Low D, Thangaratinam S. Intra aortic balloon occlusion [12] without fluoroscopy for severe postpartum haemorrhage secondary to placenta percreta. Int J Obstet Anesth. 2014;23:91-93.
4. Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial. Lancet 2017;389(10084):2105-16.

Thromboprophylaxis in Pregnancy and Puerperium

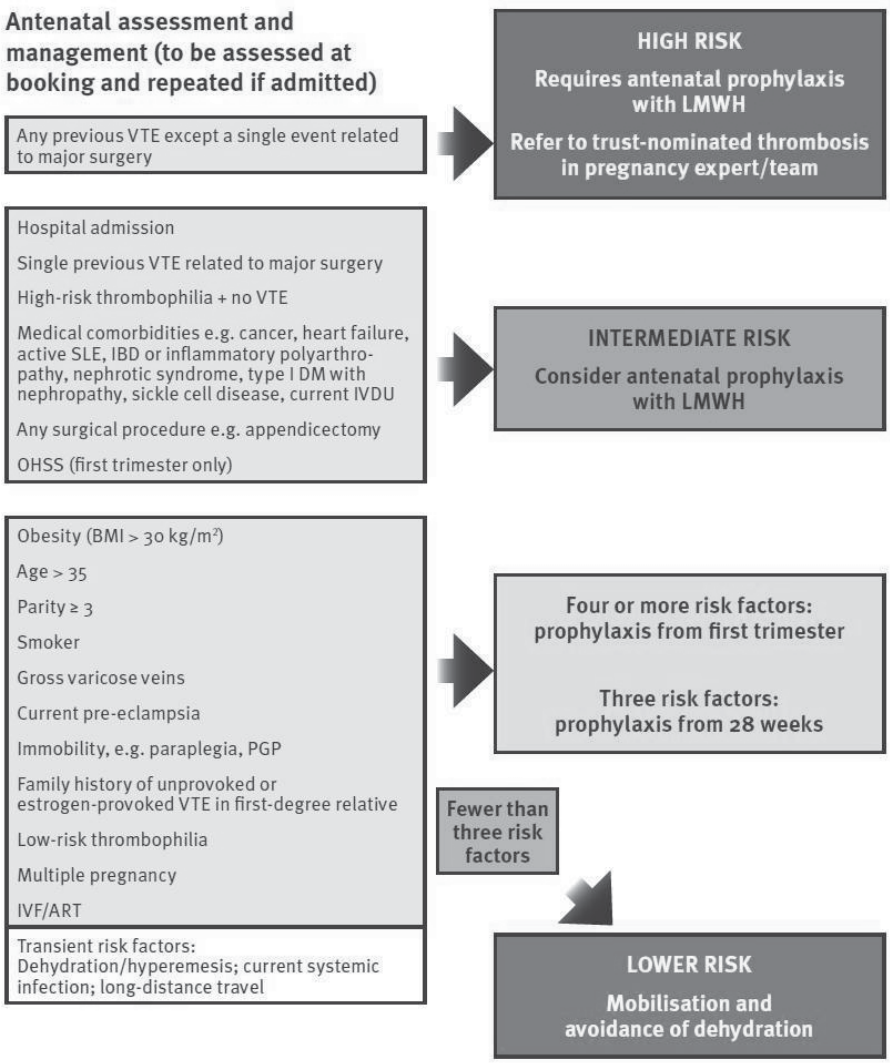
Sandhya Jain[1], Vasudha Gupta[2]

[1]Associate Professor, [2]Senior Resident, Department of Obstetrics & Gynecology, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

Venous thromboembolism (VTE) is a major cause of maternal morbidity and mortality. The incidence has been reported to range from 0.5-3/1,000 pregnancies occurring with approximately equal frequency in all three trimesters and postpartum[1,2]. Physiological changes in pregnancy increase the risk of VTE due to all the three factors described by Rudolf Virchow as Virchow's triad, i.e. hypercoagulability, stasis and endothelial damage

Obstetric Risk Assessment and Management (Figure 1&2)

All women should undergo a documented assessment of risk factors for VTE in early pregnancy. Risk assessment should be repeated when a woman is admitted to hospital for any reason or develops other intercurrent problems.



APL = antiphospholipid antibodies (lupus anticoagulant, anticardiolipin antibodies, β_2-glycoprotein 1 antibodies); ART = assisted reproductive technology; BMI based on booking weight; DM = diabetes mellitus; FHx = family history; gross varicose veins = symptomatic, above knee or associated with phlebitis/oedema/skin changes; high-risk thrombophilia = antithrombin deficiency, protein C or S deficiency, compound or homozygous for low-risk thrombophilias; IBD = inflammatory bowel disease; immobility = ≥ 3 days; IVDU = intravenous drug user; IVF = in vitro fertilisation; LMWH = low-molecular-weight heparin; long-distance travel = > 4 hours; low-risk thrombophilia = heterozygous for factor V Leiden or prothrombin G20210A mutations; OHSS = ovarian hyperstimulation syndrome; PGP = pelvic girdle pain with reduced mobility; PPH = postpartum haemorrhage; thrombophilia = inherited or acquired; VTE = venous thromboembolism.

Figure 1: Obstetric thromboprophylaxis (Antenatal)

Single previous VTE, previous VTE associated with antithrombin deficiency, VTE associated with APLA

Women with previous thromboembolism whether single previous VTE (except those with a single previous VTE related to major surgery and no other risk factors), previous VTE associated with antithrombin deficiency, VTE associated with the antiphospholipid antibody syndrome should be given LMWH antenatally and for at least 6 weeks postpartum or until returned to oral anticoagulant therapy after delivery.

The management of these patients should have a multidisciplinary approach involving obstetrician, hematologist and rheumatologist. Proper counseling of patient and the family members should be done regarding the risks and benefits of anticoagulant therapy to the patient and the baby.

Recurrent VTE

Women with recurrent VTE and on long-term warfarin or other oral anticoagulants should be

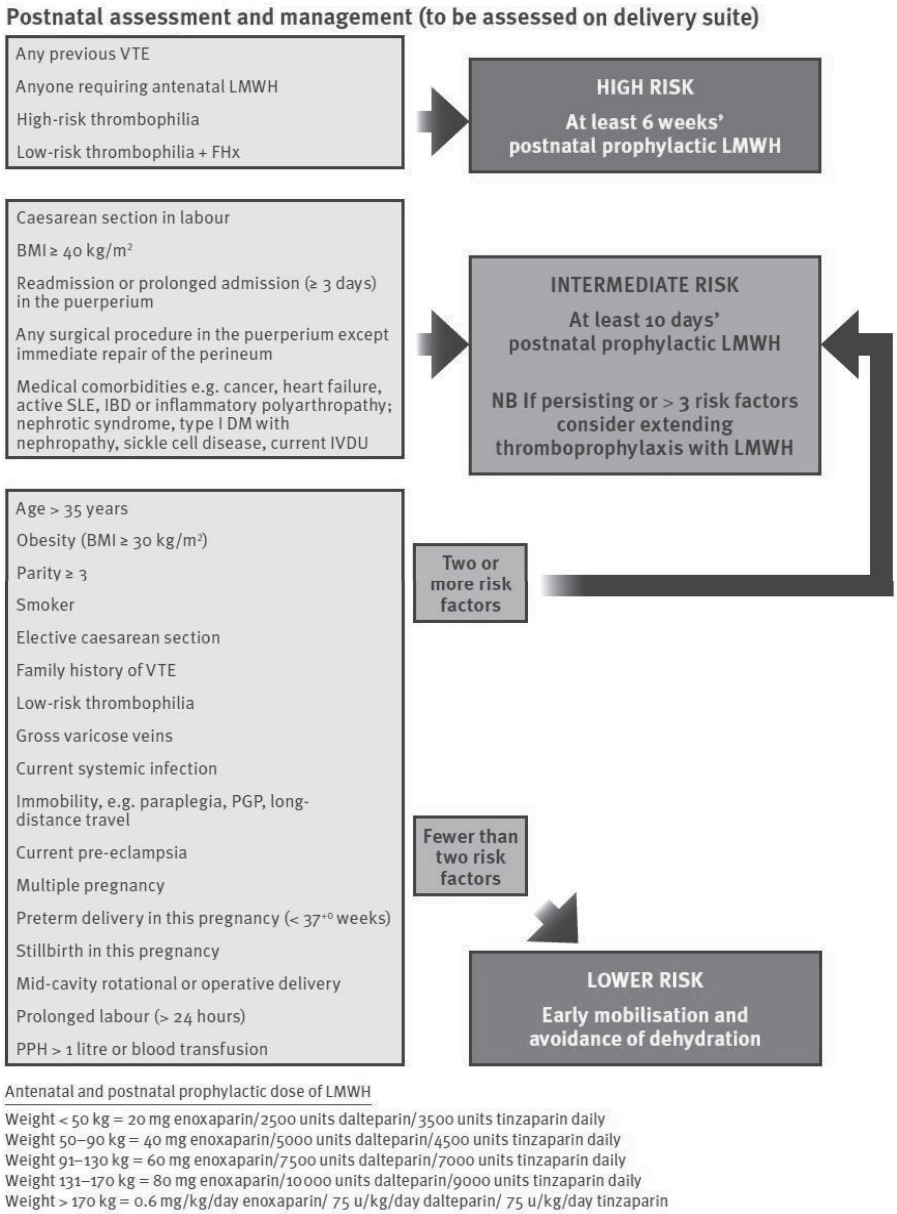


Figure 2: Obstetric thromboprophylaxis (Postnatal)

counseled about the risks of these agents to the fetus. Fetal warfarin syndrome is a condition associated with administration of warfarin during pregnancy. The symptoms are nasal hypoplasia, stippling of uncalcified epiphyses during first year, shortened fingers, low birth weight etc. Patients are advised to stop their oral anticoagulant therapy and change to LMWH as soon as pregnancy is confirmed, ideally within 2 weeks of the missed period and before the sixth week of pregnancy. Women not on warfarin or other oral anticoagulants should be advised to start LMWH as soon as they have a positive pregnancy test.[3]

Other Risk Factors

According to RCOG 2015 guidelines women with hyperemesis gravidarum who are admitted to the hospital for this condition should be given LMWH till the condition resolves. LMWH should also be considered for women with ovarian hyperstimulation syndrome.

Screening for Thrombophilias

Thrombophilia screening is **not recommended** for women who have experienced recurrent fetal loss, placental abruption, FGR, preeclampsia because it is unclear if anticoagulation therapy reduces recurrence. Management decisions regarding thromboprophylaxis or antepartum surveillance should be made through an individual risk assessment including prior history of VTE, inherited thrombophilia, cesarean delivery, immobility, obesity, and family history of VTE as mentioned earlier.

Recommendations for screening include:

- Women with a personal history of VTE.
- Women with a first-degree relative (parent or sibling) with a history of thrombophilia.

Ideally, screening tests for thrombophilias should be done at least 6 weeks after a thrombotic event and while the patient is not pregnant or on anticoagulation therapy.

If a patient has these clinical indications, recommended screening tests include[4]:

- Factor V Leiden mutation
- ProthrombinG20210A mutation
- Protein C deficiency
- Protein S deficiency
- Antithrombin deficiency

Thromboprophylaxis during labour and delivery, including the use of regional analgesia

LMWH, UFH

Regional anaesthesia should be avoided if possible until at least 12 hours after the previous prophylactic dose of LMWH and 24 hours after last therapeutic dose of

LMWH. LMWH should not be given for 4 hours after use of spinal anesthesia or after the epidural catheter has been removed and the catheter should not be removed within 12 hours of the most recent injection.

Women receiving antenatal LMWH having an elective caesarean section should receive a thromboprophylactic dose of LMWH on the day prior to delivery and, on the day of delivery, any morning dose should be omitted. Warfarin, LMWH, and UFH do not accumulate in breast milk thus they can be used in women who breastfeed.

The first thromboprophylactic dose of LMWH should be given as soon as possible after delivery provided there is no postpartum haemorrhage and regional analgesia has not been used. The risk of postpartum VTE after an emergency caesarean section is twice that after an elective caesarean section and four times that after a vaginal delivery. Therefore women who have had emergency caesarean section in labour should be considered for thromboprophylaxis with LMWH for 10 days after delivery apart from those having an elective caesarean section who should be considered for thromboprophylaxis with LMWH for 10 days after delivery if they have any additional risk factors.[3]

Women at high risk of haemorrhage including major antepartum haemorrhage, coagulopathy, progressive wound haematoma, suspected intra-abdominal bleeding and postpartum haemorrhage may be managed with anti embolism stockings (AES), foot impulse devices or intermittent pneumatic compression devices. Unfractionated heparin (UFT) may also be considered.

Agents Used for Thromboprophylaxis

LMWHs are the agents of choice for antenatal and postnatal thromboprophylaxis and the dose is weight based.[3]

Table 1: Suggested thromboprophylactic doses for antenatal and postnatal LMWH

Weight	Enoxaparin	Dalteparin	Tinzaparin (75 u/kg/day)
<50 kg	20 mg daily	2500 units daily	3500 units daily
50-90 kg	40 mg daily	5000 units daily	4500 units daily
91-130 kg	60 mg daily*	7500 units daily	7000 units daily*
131-170 kg	80 mg daily*	10 000 units daily	9000 units daily*
>170 kg	0.6 mg/kg/day*	75 u/kg/day	75 u/kg/day*
High prophylactic dose for women weighing 50–90 kg	40 mg 12 hourly	5000 units 12 hourly	4500 units 12 hourly

*may be given in 2 divided doses

Monitoring of anti-Xa levels is not required when LMWH is used for thromboprophylaxis. In women at very high risk of thrombosis UFH may be used peripartum period in preference to LMWH where there is an increased risk of haemorrhage or where regional anaesthetic techniques may be required. The precautions for LMWH use are summarised in Table 2.

If **UFH** is used after caesarean section (or other surgery), the platelet count should be monitored every 2–3 days from days 4–14 or until heparin is stopped. The risk of heparin-induced thrombocytopenia (HIT) is substantially lower with LMWH.

Warfarin use in pregnancy is restricted to the few situations where heparin is considered unsuitable, e.g. women with mechanical heart valves. Women receiving long-term anticoagulation with warfarin can be converted from LMWH to warfarin postpartum when the risk of haemorrhage is reduced, usually 5–7 days after delivery. Warfarin is safe in breastfeeding.

The use of properly applied **anti-embolism stockings (AES)** of appropriate size and providing graduated compression with a calf pressure of 14–15 mmHg is recommended in pregnancy and the puerperium for women who are hospitalized and have a contraindication to LMWH. These include women who are hospitalized post-caesarean section (combined with LMWH) and considered to be at particularly high risk of VTE (e.g. previous VTE, more than four risk factors antenatally or more than two risk factors postnatally) and women travelling long distance for more than 4 hours.[3] Aspirin is not recommended for thromboprophylaxis in obstetric patients.

Table 2: Contraindications/cautions to LMWH use

Known bleeding disorder (e.g. haemophilia, von Willebrand's disease or acquired coagulopathy)
Active antenatal or postpartum bleeding
Women considered at increased risk of major haemorrhage (e.g. placenta praevia)
Thrombocytopenia (platelet count < 75 × 109/l)
Acute stroke in previous 4 weeks (haemorrhagic or ischaemic)
Severe renal disease (glomerular filtration rate [GFR] < 30 ml/minute/1.73m2)
Severe liver disease (prothrombin time above normal range or known varices)
Uncontrolled hypertension (blood pressure > 200 mmHg systolic or > 120 mmHg diastolic)

References

1. Walker MC, Garner PR, Keely EJ. Thrombosis in pregnancy: A review. J Soc Obstet Gynaecol Can 1998;20(10):943-52.
2. Toglia MR, Nolan TE. Venous thromboembolism during pregnancy: A current review of diagnosis and management. Obstet Gynecol Survey 1996;52(1):60-72.
3. Reducing the Risk of Venous Thromboembolism during Pregnancy and the Puerperium Green-top Guideline No. 37a April 2015. Available from: https://www.rcog.org.uk/globalassets/documents/guidelines/gtg-37a.pdf. Accessed on 20th May 2017.
4. ACOG. Inherited Thrombophilias in Pregnancy Practice Bulletin #138. The American College of Obstetricians and Gynecologists, 2013.

Setting up of an Obstetric Intensive Care Unit and High Dependency Unit

Pikee Saxena[1], Supriya Goyal[2]

[1]Professor, [2]Senior Resident, Department of Obstetrics & Gynecology, Lady Hardinge Medical College & Smt. Sucheta Kriplani Hospital, New Delhi

India has made tremendous strides in healthcare and the maternal mortality ratio (MMR) has reduced from 600 in 1990 to 167 per 100,000 live births in 2011-2013. Though maternal mortality since 1990 has reduced by 69 % in India, as compared to a global reduction of 44%, India still accounted for 15% of global maternal deaths in 2015. While India's MMR is reducing at an average of 4.5% annually, this rate has been increased to 5.5% to meet the Millennium Development Goal of 140 deaths per 100,000 live births.

National Family Health Survey 2015-16 (NFHS-4) data shows significant improvement in several maternal and child health indicators due to initiatives started by the government under National Rural Health Mission. The proportion of institutional deliveries has increased from 38.7% in 2005-06 to 78.9% in 2015-16. Birth assisted by trained personnel has improved from 46.6% in 2005-06 to 81.4% in 2015-16. Although these interventions have helped to bring down MMR, for further reduction of MMR, there is a need to understand the relation between morbidity and mortality. Any pregnant woman can develop life threatening complications with little or no warning. A continuum of adverse pregnancy events may be recognized as follows: Normal healthy pregnancy→ high risk pregnancy→ morbidity→ severe morbidity→ near miss → mortality. Therefore, there is a need to target high risk mothers who constitute approximately 7-8%, pick up early warning signs and step up their management to provide high quality, dedicated critical maternal care to alter the future outcome/prevent progression to death.

Critical Care: Grading

Critical care has been graded into 4 levels

Grade 0- Patients who can be treated in the general ward

Grade 1- Patients at risk of deterioration of clinical condition or those who have been shifted out from higher level of care, who need to be cared for in the general ward with additional advice and support of critical care specialists/ multidisciplinary team.

Grade 2- Patients who require detailed monitoring and intervention including support for a single failing organ system or post operative care or those who have been shifted out of higher level of care.

Grade 3- Patients who require advanced respiratory support or respiratory support along with support of at least 2 organ systems including patients with multi organ failure.

Definition and Indications

Intensive care unit (ICU) is a specialized area of the hospital which is specifically designed, staffed, located, furnished and equipped, dedicated to management of critically sick patient, injuries or complications. ICU provides highest grade or grade 3 critical care. **Obstetric ICU** is an ICU which is dedicated to pregnant women before, during and after delivery. **High Dependency Unit (HDU) or intermediate care unit or critical care unit** is an area in a hospital where patients can be cared

Table 1: The Scope of Obstetric HDU: Following conditions may require admission in Obstetric HDU

Obstetric complications • Pregnancy/ labor pain with sever anaemia (< 7 gm%) and its complications • Accidential hemorrhage • Multiple gestation with complications. • Post Partum Hemorrhage • Placenta previa, morbidly adherent placenta • Severe pre-eclampsia/hypertensive crisis, ecclampsia, HELLP syndrome • Obstetric hysterectomy, broad ligament hematoma • Perforation during abortion • Sepsis & Systemic inflammatory Syndrome(SIRS).
Pregnancy with Medical complications • Diabetes and diabetic ketoacidosis • Cardiac Disease • Jaundice • Thyroid storm, thyrotoxicosis • Pheocromocytoma, other endocrinal crisis like Addison's disease • Renal disease like ARF • Leukemia and other hemolytic disorders • Infections: Dengue, complicated malaria, tuberculosis • Asthma and other respiratory problems • Thrombophillia, DIC • Pulmonary Edema due to perioperative fluid overload, CCF, complication of severe pre-eclampsia or tocolytic therapy with beta-agonists etc
Others: Ruptured ectopic, Hydatidiform mole, Complications due to uterine anomaly and pathologies, Postoperative patients requirig hemodynamic monitoring or intensive nursing care, Appendectomy or any other surgical emergency, pregnancy with trauma, poisoning, burns, cancer, OHSS and pregnancy

more comprehensively than in a normal ward, but not to the point of intensive care. Patients in HDU may require ICU admission later (step up) or may be shifted from ICU to HDU (step down), before shifting them to the general ward. HDU provides grade 2 critical care.

Obstetric HDU is a part of the obstetrics unit located near the labor room and operation theatre, for easy and prompt shifting of the patient whenever required. It is recommended that all pregnancies with complications be managed in obstetric HDU. The HDU should be headed by an experienced obstetrician with significant input from anesthetists and other multispecialty physicians. HDU is more patient friendly where breastfeeding can be continued and has lower rates of infection than in the ICU. The indications are summarized in Table 1.

Process of Shifting Patient from Ward/ Triage Room to HDU or ICU/ Obstetric ICU

The key steps to be taken are:

1. Inform the family/companion of pregnant woman of the decision, and take a written informed consent.
2. Case sheets containing history, examination, investigations and management should be maintained.
3. Obstetric HDU staff should be appropriately informed.
4. The patient should be escorted by doctor/staff with all existing treatment including continuation of patent IV line.
5. Keep monitoring the vitals of the patient, temperature should be maintained.
6. Adequate follow-up by the treating doctor.
7. Supine hypotension prevention (by performing lateral tilt to 15-20 degree), if required.
8. Ensure patent airway.
9. Baby should be shifted along with the mother, if delivered already.

Obstetric ICU/ HDU: The set up

Where to set up Obstetric HDU & Obstetric ICU?

It has been suggested that all District Hospitals should have an Obstetric HDU and all the Medical Colleges should have both- an obstetric HDU and an obstetric ICU (or ICU with dedicated obstetric beds). It should have an isolation room for mothers with infections requiring isolation (H1N1, Chicken pox etc.).

Table 2: Number of beds in obstetric HDU

Number of deliveries per month	Number of beds required
Up to 250	4
250-500	8
>500	Can be increased

Location

It should be located near the labor room and operation theatre and should have proximity to other areas and essential support services such as the main wards, ICU, radiology, laboratory, blood bank etc. There should be single entry/ exit point to obstetric HDU and obstetric ICU. The isolation room should have a separate entry.

There should be provision for emergency exit points for ease of evacuation in cases of disasters.

Space

The space should be 120-130 sq. ft. per bed in obstetric HDU and 130-150 sq. ft. per bed in obstetric ICU. Apart from the patient care area, there should be an ancillary area (of 100% to 120% extra space) for accommodating the nursing station/ storage/ patient movement area/ equipment area, patient toilet and to maneuver equipment, beds and trolleys etc. The beds should be at least 2 ft. from the back wall to give caregivers an easy access to the head in case of any emergency. There should be space for bed-side movable lockers with facility of trolleys/ drawers and for keeping medicines, consumables and personal belongings of the patient.

Privacy

There should be a partition between the beds for ensuring privacy of the patients.

If the space is more, each bed can be separated by fixed partitions or curtains. The curtain fabric should be fire and water proof, washable, clean, light colored, inherent stain resistance and non- allergic. The curtain height is determined by the floor-to-ceiling height, and curtains usually should finish approximately 8-10" above the finished floor (Joint Commission's Patient Privacy Standards). The curtains should have mesh at the top which will allow both light and ventilation in the patient room. The curtains should be hanging from the overhanging rails.

Flooring

The floor should be made of large vitrified, antiskid, stain proof and easy to clean tiles with seamless joints. The tiles should be of light color (preferably white or off-white).

Walls

The walls should be of durable glazed tiles which are easy to clean, stain resistant, flame resistant and have a visual appeal. It will be preferable to have a finishing of wall height of up to 6-7 ft. with the tiles like floor tiles. Colors should be chosen carefully to avoid an adverse impact on the skin color of patients and neonates, light color (white or off-white) being the preferable choice.

Ceiling

The ceiling should be leak proof. It is suggested that no lines or wires be kept or run over the ceiling or underground. It should be easy to explore in case repair

is required, as damages are common and may occur any time.

Nursing Station

The nursing station should be with the following facilities and seating capacity:

Table 3: Staff requirement for Obstetric HDU/ ICU

Staff Requirements (per shift)	
8 bedded HDU	4 dedicated nurses and 2 EmOC/ MOs
4 bedded HDU	2 dedicated nurses and 1 EmOC/ MO
For 4 bedded obstetric ICU (no separate guidance is being given for existing ICU in medical colleges)	4 dedicated nurses and 1 intensivist

Space requirements for nursing station:

a. Adequate space for central monitoring and 2 computers (for 8 bedded HDU/ ICU)
b. Adequate space for central monitoring and 1 computer (for 4 bedded HDU).
c. Scrub area and wall clock behind the nursing station.
d. Facility for keeping records and emergency medicines.

Health Management and Information System (HMIS): Facility of HMIS entry should be provided for data collection, data transmission, data storage, data processing, data analysis and presentation of the data. This will help in decision making by the hospital management for improved health care services.

Store Room: A separate room should be made to keep the equipment such as the sonography machine, portable X-ray machine, transport ventilator, nebulizer, radiant warmer, blood warmer, crash cart(s), BIPAP/ CPAP machine, etc. A general store room should be made separately to keep bed linen, disposables and consumables, personal protective attire like caps and masks, slippers, etc.

Buffer Zone: A protective clean zone should be made available located before the entry in obstetric HDU/ ICU. This zone would include area for changing shoes, patient's changing room with lockers, janitor's closet, two changing/ overnight stay rooms for doctors (1 each for male and female with lockers, beds, book shelves etc.), dining area, space to keep wheel chairs and trolleys, changing room for staff, toilets (1 each for male and female with urinal in the male toilet with flush facility).

Autoclaving and Sterilized Supply: From the Central Sterilized Supply Department (CSSD).

Waiting Area: Waiting area for the patients' attendants should be provided, with facility for seating capacity of at least 2 relatives per patient, facility of drinking water, a large TV with LCD display, toilets, newspapers and educational material etc. The waiting area can be shared with waiting area for the LR and/or OT.

Facilities:

a) All cots in the obstetric HDU/ ICU must be birthing cum patient bed with the following features: Safe working load of 180 kg (392 lbs). Size 1800x620x800 mm. Electrically/manually operated with battery back-up in the event of power failure. Dual-sided manual CPR levers. Easy-to-operate Trendelenburg, reverse Trendelenburg, with both side tilts with compact size to fit into the small delivery room. Retractable platform should be present, optimizing access to perineal area, mother and baby during the delivery procedure. Mattress should be in 3-parts of minimum 6 inch, with perineal cut-out seat section. Mattress should be washable with seamless molding. Adjustable safety rails should be present on both the sides. Good quality castors with brake system for optimum mobility/stability. Comprehensive range of easy-to-fit accessories, including adjustable leg rests, IV rod, handgrips, high and low foot supports, a removable step and a fluid collection bowl. Removable, moulded head and foot panels should be present.
b) Each bed will require at least 2 oxygen, 1 air and 2 suction outlets, and at least 10 central voltage stabilized power points (6 power-points of 5 amps and 4 points of 15amp) preferably 5 on each side of the bed. Adapters should be discouraged as they tend to become loose.
c) Heating, Ventilation and Air-conditioning (HVAC) system along-with ceiling fans & power backup exclusive for HDU for uninterrupted power supply should be installed. Voltage stabilization is mandatory. Suitable and safe air quality must be maintained always. Temperature should be adjustable within each cubicle/ room as per the patient comfort and choice.
d) General Lighting: Access to outside natural light is recommended. The colorless concealed LED Lights with sufficient high illumination should be provided. It should be bright enough to ensure adequate vision without eye strain.
e) Hand Hygiene and Prevention of Infection: Sink should be of the operation room style with water supply through elbow operated taps. Every bed should have alcohol based anti-microbial instant hand wash solution source. All entrants should don a mask and cap, and ideally an apron which should be replaced daily.
f) Waste Disposal and Pollution Control: This is mandatory and a huge safety issue both for the patient and the staff/doctors of the hospital, and society at large. It is important that all government regulations (State Pollution Control Board) should strictly be complied with.
g) Laboratory backup facility for 24 hours.
h) In-house or nearby blood bank.
i) In- house or nearby neonatal intensive care unit.
j) Lactation Support: Facilities for breast feeding (or

use of breast pump) should be available to all post-natal mothers in obstetric HDU. Babies are usually not allowed in obstetric ICU, but breast pump facility should be made available.

k) Provision for fire safety, preferably through automatic water sprinkling system or normal fire extinguishers.

l) For uninterrupted power supply, noiseless generator or inverter with instant switchover, search protector and voltage stabilizer. An earthing pit for proper earthing should be ensured, with monitoring once in 2 years.

m) Nurse calling system with a central display and an audio-visual alarm.

n) Facility for keeping records and registers at the nursing station.

o) Ambulance: One hi-tech ambulance with all advanced life support measures for critically ill mother, and with a transport incubator and ventilator support for critical new born.

Equipment required in HDU

It includes vital monitors depicting Pulse, BP, RR, ECG, SPO_2 and with transducer facility for invasive monitoring, equipment for insertion and management of invasive monitoring (arterial line and central venous pressure (CVP)), piped oxygen and suction, anesthesia trolley, IV fluid warmer, forced air warming device, electric blanket, blood gas analyzer, infusion pumps, syringe pumps, uroflowmeters, delivery tray, emergency hemorrhage/eclampsia trolley, intubation tray, neonatal resuscitation equipment, mobile X ray, USG machine, CTG machine, transfer equipment – monitor and ventilator, computer terminal to facilitate access to blood results, a crash cart fully loaded with BCLS (basic cardiac life support) medications, copy of hospital obstetric guidelines, resuscitation trolley with defibrillator and airway management equipments, CNS tray with torch and hammer. Availability of all essential and emergency drugs should be ensured after checking the date of expiry on a regular basis. Intermittent compression device for DVT prophylaxis, color coded bins for biomedical waste, wall clock should be available.

Human Resources

Obstetrician is the head of the obstetric HDU. He/she will decide when and whom to call from the list of multidisciplinary teams (wherever available). For management of the obstetric patients

Emergency obstetric care/ Medical Officers should be posted round the clock. Obstetric nursing staff (24x7) with nursing staff to patient ratio should be 1:2; there should be 1 extra for lay off or covering leave/day off. Support Staff includes pharmacist, dietician, counsellor, housekeeping and cleaning, security, data entry operator and electrical technician.

Training of Staff of HDU

Training should be given to staff, medical officer, residents and consultants by posting them in the ICU and labor room for 3 months for basic resuscitation, intubation, IV fluid management, blood component transfusion, ICU procedures, obstetric drills, record keeping and management of equipment used in the HDU. Refresher training should also be arranged at regular intervals.

Monitoring and Record keeping in a HDU is of utmost importance and should include history, thorough examination, details of investigations and management, probable or final diagnosis, details of any operative management, nursing charts including one hourly temperature, pulse, respiratory rate, blood pressure, pulse oxymetry, strict input and output, fluid balance and details of fluid administered, abdominal girth, CVP charting whenever required, anesthesia charts and detailed drug charts. Antenatal patients should be nursed in left lateral position and fetal heart sound should be monitored by CTG. Adequate pain management should be done. All teams looking after the patient should have appropriate coordination with each other and should be informed about any change in treatment. Timing, date and signatures should be put on case records.

The patient and her relatives should be treated with kindness, respect and dignity always. The relatives need to be updated regularly regarding the patient's progress and a written informed consent should be obtained for any procedure. Ideally protocols and standard operating procedures should be developed to improve care for patients at a local, national and international level.

Suggested discharge/transfer criteria for the HDU is that the patient is conscious and alert, has stable and normal respiratory status and hemodynamic parameters with no evidence of hemorrhage, intensive/invasive monitoring is no longer required and 4-hourly recording of vital signs is considered appropriate. In case the patient develops pulmonary edema / ARDS/ systemic inflammatory response syndrome/ DIC / multiorgan failure with poor cardiac output despite fluid resuscitation/septic shock, patient should be shifted to Obstetric ICU.

Suggested Readings

1. Guidelines for Obstetric HDU & ICU. Maternal Health Division, Ministry of Health and Family Welfare, Government of India, March 2016
2. rchiips.org/NFHS/factsheet_NFHS-4.shtml. Accessed on 20/5/17.
3. Handbook of Obstetric High Dependency Care, 1st edition. By © D. Vaughan, N. Robinson, N. Lucas and S. Arulkumaran. Published 2010 by Blackwell Publishing Ltd
4. ICU planning and designing in India-guidelines 2010, Guidelines Committee (ISCCM).

Transfusion Guidelines in Obstetric Practice

Priyanka Gogoi
Associate Professor, Department of Pathology and Blood Bank, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

Obstetric hemorrhage and anemia remain a major cause of maternal morbidity and mortality worldwide. Thrombotic and vascular complications of several maternal diseases like diabetes, renal failure, hemoglobinopathies, systemic lupus, and antiphospholipid syndrome do not cause primary bleeding per se but increase maternal and fetal morbidity by inducing preeclampsia, thrombo-embolism, and placental insufficiency. These maternal diseases associated with maternal thrombocytopenia and coagulopathies can pose primary bleeding problems requiring transfusion.

Indications for Maternal Transfusions

Major obstetric bleeding

The goals are to establish rapid control of bleeding and restore systemic oxygen delivery. Recent complex trauma protocols advocate that when massive bleeding is anticipated, the patient should be rapidly transfused with an optimal ratio of RBC, plasma, cryoprecipitate and platelets, without waiting for consumption and dilutional coagulopathies. Restoration of the components of the blood is also essential to ensure adequate tissue perfusion and to prevent acidosis and hypothermia.

The therapeutic components are sequentially administered, beginning with crystalloid- colloid solutions infused to replace lost intravascular volume. Second, red blood cells (RBCs) are transfused to restore oxygen carrying capacity. Most protocols recommend six units of packed red cells (PRBCs) be prepared and available and hematocrit be maintained minimally at 21–24%. WHO recommends transfusing Group O negative antibody-screened blood, and/or uncrossmatched group specific blood until fully cross-matched blood is available.

Component therapy

Fresh frozen plasma (FFP) is plasma that has been separated from a unit of whole blood within 6–8 hours of donation and is rich in all intrinsic clotting factors. Cryoprecipitate is the cold-insoluble portion of plasma remaining after FFP has been thawed. It contains approximately 50% of the Factor VIII and von Willebrand Factor, 20–40% of the fibrinogen and some of the Factor XIII originally present in the fresh plasma. Coagulation screens should be performed after 500ml blood loss, in case of active ongoing bleeding. The tests include prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, D-dimer, and a complete blood count. Clotting factors and platelets (PLTs) are delivered to restore physiologic hemostasis.

Fresh frozen plasma (12-15 ml/kg), 1 unit for every 4–6 units of blood is given to prevent coagulation defects resulting from use of stored red cell concentrates. At least 10 packs of cryoprecipitate, prepared from single donor units, containing 3–4 gm fibrinogen in total should be transfused in massive hemorrhage. Some studies have shown that using a high ratio of PRBCs to FFP (1.5:1 or 1:1) has significantly improve survival from hemorrhage after trauma and similar recommendations have been established for massive obstetric hemorrhage protocols with the goal of maintaining the INR at <1.5–1.7, fibrinogen level above 1.5 gm/L and platelet count at >50×10^9/L. Blood products should not be continued on basis of clinical suspicion alone and their use should be guided by the results of coagulation tests only**.** The FFP and cryoprecipitate should ideally be of the same group as the recipient. If unavailable, FFP of a different ABO group is acceptable, if it does not have a high titer anti-A or anti-B activity. If these blood components are not available, the freshest whole blood available (ideally no more than 36 hours old) is transfused.

Platelet transfusions during pregnancy and for delivery

Whatever be the cause of thrombocytopenia, most guidelines recommend a platelet count above 50×10^9/L for vaginal delivery. Guidelines differ in their threshold for giving local anesthesia, most guidelines advise 50 or 80×10^9/L. According to RCOG, platelet counts should not fall below 50×10^9/L in a woman with ongoing hemorrhage, and 75×10^9/L should be the threshold for platelet transfusion in ongoing bleeding to keep the safety margin. The recommendation of Indian Society of Hematology and Transfusion (ISHBT) for platelet threshold for vaginal delivery is 30×10^9/L, for cesarean section is 50×10^9/L and for epidural anesthesia is 80×10^9/L. Because of different pathophysiology and bleeding history, an individual treatment plan for every pregnant patient with thrombocytopenia is required. Except in case of TTP which is a relative contraindication for platelet transfusions, platelet transfusions are not contraindicated in ITP or DIC. The platelets should ideally

also be group compatible and if RhD-negative woman receives RhD-positive platelets, anti-D immunoglobulin (dose of 250 IU) should be administered.

If a patient continues to bleed or future hemorrhage is anticipated, additional components either in the original ratio or customized as per the patient's need, can be requested from the transfusion service. Additional samples for coagulation profile are sent with each request and abnormal laboratory values such as prolonged PT and/or aPTT, low PLT count, and low fibrinogen levels can be addressed individually.

Intraoperative cell salvage (IOCS)

It may have a role in obstetric practice in reducing the exposure of obstetric patients to the risks of allogenic blood transfusion. It is the process by which blood shed within the surgical field is retrieved by an anticoagulated suction apparatus and collected within a reservoir from where it is centrifuged, washed and pumped into an infusion bag. This salvaged blood can then be returned to the patient. The process has been widely used in adult orthopedic and cardiac surgery without complication. However, its use in obstetric practice has been limited, owing to concerns about contamination by amniotic fluid, specifically the risks of amniotic fluid embolism, and by fetal blood cells. In the present scenario, the use of cell salvage in obstetrics remains controversial and the experience of its use remains limited.

In a similar vein, the efficacy and safety of the use of recombinant factor VIIa (rFVIIa) for cases of obstetric hemorrhage remains to be established.

Chronic anemia in pregnancy

Transfusion protocols should not be based on the patient's hemoglobin concentration alone, but also on her clinical needs, including stage of pregnancy and existing clinical conditions like established or incipient cardiac failure or clinical evidence of hypoxia, co-existing infections and morbidities including pre-existing heart disease. The need for transfusion is also determined by the mode of delivery and history of hemorrhage in the present or previous pregnancies. The specific indications for transfusion for chronic anemia in pregnancy should be based on national guidelines, modified as appropriate to the local situation. Usually hemoglobin levels of < 7gm% require transfusion.

Postpartum anemia

Although guidelines suggest a transfusion threshold at a hemoglobin concentration of 7.0-8.0gm/l, results of various studies underscore that in case where there is no continuing or threat of bleeding, transfusion is only required for symptomatic anemia.

Storage of Blood and Transfusion Practices

Whole blood and red cells must be stored at a temperature of +2°C to +6°C and red cells must never be allowed to freeze. This is important to maintain ATP and prevent clotting, hemolysis and infection. The maximum storage time is 35 days. Fresh frozen plasma (FFP) and cryoprecipitate are rapidly frozen, and maintained at, a temperature of –20°C or colder. Once thawed they must be kept at +2°C to +8°C and transfused within 6 hours. To maintain platelet function, platelet concentrates should be stored at a temperature of +20°C to +24°C with continuous agitation on a special platelet agitator. They should never be placed in a refrigerator or freezer. They can be stored for up to 72 hours and upto five days in specialized platelet packs. If no platelet agitator is available, platelet concentrates must be transfused within 30 minutes of completion of testing, else within 4 hours.

Special Precautions and Complications

Ensure blood group cross matched transfusion of packed RBC and platelets to minimize risks of isoimmunization. A written informed consent with complete identity check is advisable. Always match the details on the bag with patient details and check compatibility report.

Besides enhanced induction of alloantibodies, blood products can potentially transmit infections {including HIV, Hepatitis B, Hepatitis C, malaria, syphilis, cytomegalovirus (CMV)} which can be reduced by proper selection and screening of donors. Voluntary, non-remunerated donors have been proven to be safer donors than relatives. Improvement of a system of repeat replacement donors is feasible and is worth giving more attention to avoid first-time relatives who have high rates of hepatitis and HIV infections.

Transfusion reactions (haemolytic and nonhaemolytic) causing signs of fever, rash, urticaria, tachycardia and hypotension are still common, occurring in up to 13% of blood recipient. Most of these are minor reactions but immune hemolysis which can be fatal is estimated to occur in about 1 in 6000 units transfused. Rapid infusion of cold blood and stored red cell may give rise to hypothermia, hyperkalemia and hypocalcemia.

Conclusion

Strategies aimed at diagnosing and treating chronic anemia during pregnancy and active management of the third stage of labor to minimize blood loss may reduce the need for blood transfusion. This is important as unnecessary blood transfusion is not only wastage of a precious resource but also increase exposure to transfusion-related risks and infections. Protocol to

manage major obstetric hemorrhage should be in place with involvement of a consultant obstetrician, anesthetist and hematologist and the blood bank.

Suggested Reading

1. Carrolli G, Cuesta C, Abalos E, Gulmezoglu AM. Epidemiology of post partum haemorrhage: a systematic review. *Best Pract Res Clin Obstet Gynaecol* 2008;22:999–1012.
2. Royal Australian and New Zealand College of Obstetricians and Gynaecologists. Management of Postpartum Haemorrhage (C-Obs 43). Australia, 2011.
3. University of Washington. Obstetric Emergency Hemorrhage Protocol. CMQCC Obstetric, Hemorrhage Toolkit, Obstetric Hemorrage Care Guidelines and Compendium of Best Practices. Reviewed by CADPH-MCAH: 11/24/09.
4. Burtelow M, Riley E, Druzin M, Fontaine M, Viele M, Goodnough LT. How we treat: management of life-threatening primary postpartum hemorrhage with a standardized massive transfusion protocol. Transfusion. 2007; 47(9):1564-72.
5. Shields LE, Wiesner S, Fulton J, Pelletreau B. Comprehensive maternal hemorrhage protocols reduce the use of blood products and improve patient safety. *AmJ Obstet Gynecol* 2015;212(3):272–80.

Day of the Month: World Blood Donor Day

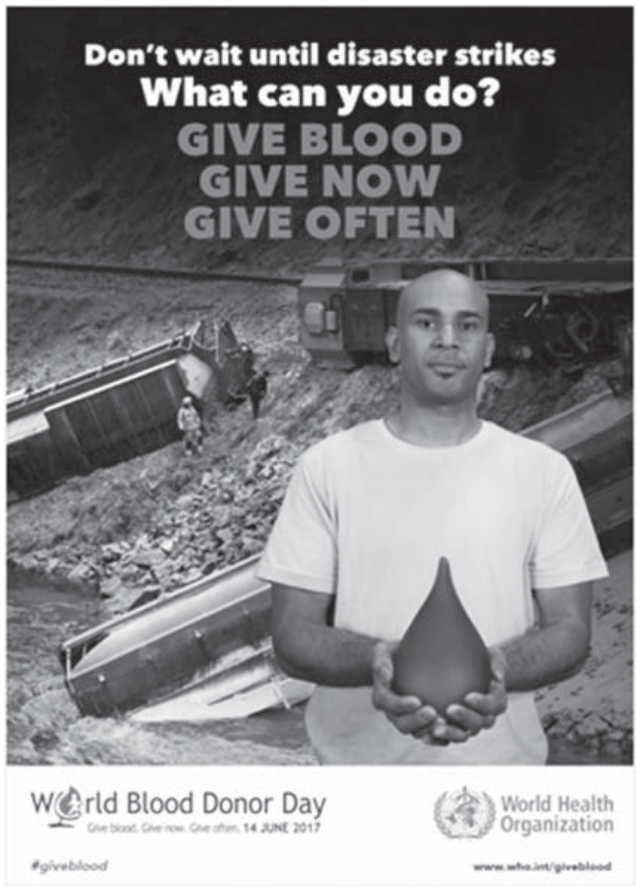
Bindiya Gupta

Assistant Professor, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

World Blood Donor Day 2017 will be celebrated by people all around the world on 14th of June 2017 to mark the birth anniversary of Karl Landsteiner on 14th of June in 1868. This event celebration was first started in the year 2004 by "the World Health Organization, the International Federation of Red Cross and Red Crescent Societies" to raise public awareness about the need for safe blood donation (including its products) voluntarily and unpaid by a healthy person. The slogan of the year 2017 celebration is "Give blood, Give now, Give often" . World Blood Donor Day is celebrated to fulfill the need of blood transfusion, save more than millions of lives annually, help patients suffering from variety of life-threatening health conditions and caring for the women during pre-and post pregnancy. Some of the objectives of the World Blood Donor Day celebration are mentioned below:

- World Health Organization has aimed to obtain the sufficient blood supplies from the voluntary and unpaid blood donors all over the world by 2020.
- To motivate more voluntary blood donors through educational programs and campaigns worldwide and acknowledge the people who are already donors.
- WHO runs this campaign by organizing many activities in all countries highlighting people's stories who need immediate blood donation to continue their heart beat.
- It is celebrated to reduce the mortality rate because of insufficient blood supply.

WHO Theme poster 2017



"Mind, Body & Soul"

Mystery Surrounding Near Death Experiences

Rashmi

Assistant Professor, Deptt of Obstetrics & Gynecology, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

"It is worth dying to find out what life is." - T.S. Eliot

Nothing fascinates humans more than the mystery of death or especially what lies beyond death. Death is defined as the cessation of all vital functions of the body. Death is basically the moment at which life ends and life may be defined in terms of consciousness. A **near-death experience** (**NDE**) is a personal experience associated with death or impending death. They have also been described as Out of Body Experiences. When someone almost died only to return back and live further, their experiences arouse curiosity and perhaps an insight into the moment of death. The question one wants to know is as to what happens at that moment, Do we just die or part of us, the Soul or Consciousness, (as believed in many religions and spiritual systems) moves to another realm.

The first recorded NDE was in the time of Plato. NDEs have been recorded over the centuries from all around the world and from countries with widely diverse cultures. Reported incidence of NDEs varies from 4-15%. NDEs are not dreams, hallucinations, or the random memories of a dying brain. In 1968 Celia Green published an analysis of 400 first-hand accounts of out-of-body experiences. These experiences were popularized by the work of psychiatrist Raymond Moody in 1975 as the near-death experience (NDE).

ND Experiences

About 10-20% of people experiencing a life-threatening event will have a NDE. Although the features of NDEs vary from one case to the next, a striking feature is their remarkable similarity of content like:

- A sense of peace, well-being and painlessness. A sense of removal from the world.
- An out-of-body experience. A perception of one's body from an outside position. Sometimes observing medical professionals performing resuscitation efforts.
- A "tunnel experience" or entering a darkness. A sense of moving up, or through, a passageway or staircase.
- A rapid movement toward and/or sudden immersion in a powerful light (or "Being of Light") which communicates with the person.
- An intense feeling of unconditional love and acceptance.
- Encountering "Beings of Light", "Beings dressed in white", or similar. Also, the possibility of being reunited with deceased loved ones.
- Receiving a life review, commonly referred to as "seeing one's life flash before one's eyes".
- Receiving knowledge about one's life and the nature of the universe.
- Approaching a border, or a decision by oneself or others to return to one's body, often accompanied by a reluctance to return.
- Suddenly finding oneself back inside one's body.
- Connection to the cultural beliefs held by the individual, which seem to dictate some of the phenomena experienced in the NDE and particularly the later interpretation thereof.[13]

Kenneth Ring (1980) subdivided the NDE on a five-stage continuum of Peace, Body separation, Entering darkness, Seeing the light & Entering the light. He stated that 60% experienced stage 1 but only 10% experienced stage 5.

After-effects

NDEs are associated with changes in personality and outlook on life like greater appreciation for life, higher self-esteem, greater compassion for others, less concern for acquiring material wealth, a heightened sense of purpose and self-understanding, desire to learn, elevated spirituality, greater ecological sensitivity and planetary concern, and a feeling of being more intuitive.

The NDE literature contains other accounts of remarkable and even apparently medically inexplicable healing following a NDE. Recently lot of interest & curiosity about NDEs has been generated on social media by videos and talks by Anita Moorjani. She was diagnosed with terminal cancer. It was at this point that she had Near Death Experience which was described as crossing over to other dimension and then returning again into this world with a clearer understanding of her life and purpose on earth. This understanding subsequently led to a total miraculous recovery of her health.

Near-death Studies

Raymond Moody's book *Life After Life*, which was released in 1975, brought public attention to the topic of NDEs. This was soon to be followed by the establishment

of the International Association for Near-Death Studies (IANDS) in 1981 which encourages scientific research and education on the physical, psychological, social, and spiritual nature and ramifications of near-death experiences.

In 1998 Dr. Long (a radiation oncologist) and his wife, Jody, began the Near Death Experience Research Foundation with the goal of creating a forum for near death "experiencers" to share their stories. **Awareness during Resuscitation (AWARE) study** was a multicenteric study in UK & USA which tested consciousness, memories and awareness during cardiac arrest. 9% could be described as real NDEs. Results from AWARE II study which was completed in March, 2017, are still awaited.

Approaches to explain NDEs fall into three broad groups i.e. Spiritual theories (also called transcendental), Psychological theories and Physiological theories that provide a physical explanation for NDEs.

Psychological Explanations

These explain NDEs as psychological responses resulting in hallucinations or withdrawal from reality and events being constructed in the mind, to extreme stressful situations like near death events. But the main shortcoming of this theory is that people with NDE never lose the feeling of their identity.

Physiological Explanations

Physiological theories explain NDEs to be due to cerebral hypoxia, anoxia, and hypercarbia; endorphins and other neurotransmitters; and abnormal activity in the temporal lobes. Further studies are underway trying to find the functional neuroanatomy of near-death experiences. Some theories hypothesize that drugs used during resuscitation may be responsible for NDEs. But these theories fail to explain all features of NDEs and the after effects.

Spiritual Explanations - After life claims and skeptical responses

Many individuals who experience an NDE see it as a verification of the existence of an afterlife, and as evidence that human consciousness may continue to exist after death. According to this interpretation, consciousness can become separated from the brain under certain conditions and glimpse the spiritual realm to which souls travel after death. The transcendental model is in some friction with the dominant view from mainstream neuroscience; that consciousness is a product of, and dependent on, the brain. According to the mainstream neuroscientific view, once the brain stops functioning at brain death, consciousness fails to survive and ceases to exist.

So the Near Death Experiences remain challenging to the modern sciences. Perhaps this is the proof that human understanding is still limited and there is a whole dimension about consciousness that is beyond understanding of scientific methodology. These experiences are bridging the gap between science and spirituality and perfect example where the boundaries between Mind, Body and Soul are getting blurred.

Maternal Near Miss: Case snippets

Case 1

Repeated Maternal Near Miss in a case of Recurrent Rupture Uterus: A report

Taru Gupta[1], Shweta Singh[2], Sangeeta Gupta[3]
[1]Professor, [2]Senior Resident, [3]Senior Consultant & HOD, Department of Obstetrics & Gynecology, ESI PGIMSR, Basaidarapur, Delhi

A 25 years female, G3P1+0+1+0, married for 10 years at 25 weeks 5 days of gestation presented to Gynae casualty of ESI- PGIMSR, Basaidarapur New Delhi, with complaints of pain abdomen which was initially localised in the pelvis but later on became generalized.

She had a history of infertility and D&C done two years ago. Her first pregnancy was unsupervised and she had gone to a dai for delivery. After trial of labour at home for two days, she was referred to a private hospital where she underwent emergency laparotomy for Rupture uterus and a star shaped fundal rupture was found. Rent was repaired and 6 units of PCV and 4 FFP were transfused. After 2 year she conceived spontaneously. Her pregnancy was uneventful till 26 week of gestation when she had sudden pain abdomen. On USG spontaneous rupture uterus was found and on laparotomy again a fundal rupture was found and repair of rent was done in Pvt. Hospital. She received 7 units of RCC and was kept in ICU for 2 days.

When she came to our casualty on examination, her general condition was poor, she was in shock. She was pale (clinically 4-5 gm%), with PR of 110/min , low volume, and B.P of 77/54 mg hg. Abdomen was mildly distended; tense, and tender, uterus was 26 weeks palpable and tender and dullness was present in bilateral flanks. On per vaginal examination the cervix was closed. USG showed a posterior uterine rupture and fetus was lying in Pouch of Douglas, and cardiac activity was absent, moderate amount free fluid was present in POD and abdominal cavity. On laparotomy an intact sac of amniotic fluid containing a fetus of 600 gms and the placenta in the abdominal cavity was found and there was rent of 6×7 cm on fundo-posterior part of uterus and 1100cc of hemoperitoneum was drained. The uterus was sutured in 3 layers with Vicryl® n°1.Bilateral tubal ligation was done using the Modified Pomeroy technique. She was resuscitated with 6 unit of PRBC + 4 FFP. Post-op period was uneventful. Her post op Hb was 9.6 gm. Pt. was discharged after counseling as she has no live issue.

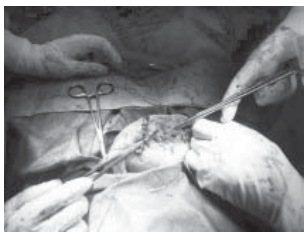


Figure 1: Fundal rupture of the rent from previous rupture site

Discussion

Incidence of rupture of an upper segment scar and spontaneously healed or repaired iatrogenic uterine perforation are respectively 4% to 19% and 0.16% to 0.9% (3-4).The patient survived thrice due to recurrent rupture of uterus at the same fundal site. We propose our patient had 3 predisposing factors. Firstly, a suspected uterine perforation during D&C 8 years ago, leading to inadequate healing of scar by fibrosis. Secondly, prolonged trial of labor during first pregnancy that lead to rupture at the potential weak area. Lastly, in successive pregnancies she had scarred uterus. The repeat rupture occurred spontaneously at 26 weeks and 25 weeks respectively. And every time she needed multiple transfusions for resuscitation, which led here to fall in near miss criteria.

Conclusion

Uterine rupture must be viewed now a days as a potentially preventable complication. Great caution during trial of labor in patient with scarred uterus and detailed postnatal discussions with uterine rupture patients about subsequent fertility and contraceptive advice is also a good practice, in view of its possible recurrence and prevent subsequent near miss.

References

1. Hofmeyr GJ, Say L , Gulmezoglu AM. WHO systematic review of maternal mortality and morbidity: the prevalence of uterine rupture. BJOG. 2005; 112: 1221–8
2. Amarin ZO, Badria LF. A survey of uterine perforation following dilatation and curettage or evacuation of retained products of conception. Arch Gynecol Obstet 2005;271(3):203-6.

Case 2

A Case of Sudden Cardio Respiratory Collapse

Vishnu Bhartiya
Assistant Professor, Department of Obstetrics & Gynecology, UCMS & GTB Hospital, Delhi

A 35- yr Old woman, G5P4L4 at 26 weeks gestation was admitted to gynae casuality with complaints of leaking per vaginum from 2 days and bleeding per vaginum and pain abdomen for 2 hrs. There was no h/o fever, foul smelling discharge and trauma. On examination, her GC -fine, temperature normal, no pallor, icterus and pedal edema. The vitals were stable (pulse 86/min, BP-110/60 mmHg) and chest and CVS examination were normal. On per abdomen examination uterus was 26 wk soft, relax, non tense, non tender, and fetal heart was not localised. Perspeculum examination revealed blood mixed discharge and or was closed. Her obstetric scan revealed a single live intrauterine

fetus, anterior low lying placenta 3cm from OS with severe oligohydramnios. With a provisional diagnosis of preterm PROM she was put on conservative management including temperature and pulse charting and antibiotics. Her Hb-10.5 gm%, TLC -12,000 /cumm, P/C-255 lacs, BU-18 mg/dl, s.cret-1.1 with normal LFT and coagulation profile.

On third day of admission patient had high grade fever, tachycardia, raised TLC (26,000 /cumm), and deranged coagulation profile (PT-17 sec, PTTK-72 sec, INR1.9). Abdominal tone was raised, cardiac activity was absent and there was dark altered blood per vaginum. After transfusing 4unit FFP she was induced in view of chorioamnionitis and abruption with septicaemia and DIC. Immediately after induction the patient rapidly deteriorated with peripheral oxygen saturation - 80%, BP- 80/60 mmHg, PR-126 beats/min, RR-30/min, urine output-20ml/hr. She was put on ventilatory support, inotropes and metabolic acidosis was corrected. She delivered uneventfully within four hours of induction and one packed cell and four FFP were further transfused to correct anaemia and DIC.

Her condition kept on worsening with persisting tachycardia and tachypnea, and inotrope support was increased. Chest x ray revealed reticulonodular opacity in B/L lung,CP angles clear and Cardiomegaly. ECG also showed some non specific changes in ST segment andT waves. USG doppler of B/L leg veins and abdomen and pelvis were normal.

2D ECHO revealed mild MR with trivial TR with normal ejection fraction excluding postpartum cardiomyopathy. D- dimer levels and fibrin degradation products levels were raised (7256 ng/ml and 40ug/ml respectively). Provisional diagnosis of ARDS, sepsis and DIC secondary to amniotic fluid embolism was made. Low molecular weight heparin (clexane 0.6 ml S/C 12 hourly) was started on 3rd postpartum day after correction of coagulation profile. She responded to the treatment dramatically. The vitals became stable, (BP norm100/60 mmHg, pulse: 82-90/min), saturation improved and ventilatory support was discontinued on day 3 and inotrope support was tapered and stopped on day 6. Injection clexane was continued for 1 week and patient was discharged on day postpartum in a stable condition.

Discussion

Amniotic fluid embolism (AFE), a rare but potentially catastrophic obstetric emergency presents with variable presentation, ranging from cardiac arrest and death through to mild degrees of organ system dysfunction with or without coagulopathy. As there is no diagnostic test for AFE, diagnosis is purely clinical based on high suspicion and combination of left ventricular dysfunction and acute lung injury occur, with activation of several clotting factors. In the present case,the patient deteoriated rapidly, immediately after induction. The risk of amniotic fluid embolism is further increased in abruption. However, this case was further complicated due to chorioamnionitis and sepsis. The role of Heparin in AFE is controversial.

Journal Scan

Sruthi Bhaskaran
Assistant Professor, Department of Obstetrics and Gynecology, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

1. Lancet. 2017 Apr 26. pii: S0140-6736(17)30638-4. doi: 10.1016/S0140-6736(17)30638-4

Effect of early tranexamic acid administration on mortality, hysterectomy, and other morbidities in women with post-partum haemorrhage (WOMAN): an international, randomised, double-blind, placebo-controlled trial

WOMAN Trial Collaborators

Background
Post-partum haemorrhage is the leading cause of maternal death worldwide. Early administration of tranexamic acid reduces deaths due to bleeding in trauma patients. We aimed to assess the effects of early administration of tranexamic acid on death, hysterectomy, and other relevant outcomes in women with post-partum haemorrhage.

Methods
In this randomised, double-blind, placebo-controlled trial, we recruited women aged 16 years and older with a clinical diagnosis of post-partum haemorrhage after a vaginal birth or caesarean section from 193 hospitals in 21 countries. We randomly assigned women to receive either 1 g intravenous tranexamic acid or matching placebo in addition to usual care. If bleeding continued after 30 min, or stopped and restarted within 24 h of the first dose, a second dose of 1 g of tranexamic acid or placebo could be given. Patients were assigned by selection of a numbered treatment pack from a box containing eight numbered packs that were identical apart from the pack number. Participants, care givers, and those assessing outcomes were masked to allocation. We originally planned to enrol 15 000 women with a composite primary endpoint of death from all-causes or hysterectomy within 42 days of giving birth. However, during the trial it became apparent that the decision to conduct a hysterectomy was often made at the same time as randomisation. Although tranexamic acid could influence the risk of death in these cases, it could not affect the risk of hysterectomy. We therefore increased the sample size from 15 000 to 20 000 women in order to estimate the effect of tranexamic acid on the risk of death from post-partum haemorrhage. All analyses were done on an intention-to-treat basis.

Findings
Between March, 2010, and April, 2016, 20 060 women were enrolled and randomly assigned to receive tranexamic acid (n=10 051) or placebo (n=10 009), of whom 10 036 and 9985, respectively, were included in the analysis. Death due to bleeding was significantly reduced in women given tranexamic acid (155 [1·5%] of 10 036 patients *vs* 191 [1·9%] of 9985 in the placebo group, risk ratio [RR] 0·81, 95% CI 0·65–1·00; p=0·045), especially in women given treatment within 3 h of giving birth (89 [1·2%] in the tranexamic acid group *vs* 127 [1·7%] in the placebo group, RR 0·69, 95% CI 0·52–0·91; p=0·008). All other causes of death did not differ significantly by group. Hysterectomy was not reduced with tranexamic acid (358 [3·6%] patients in the tranexamic acid group *vs* 351 [3·5%] in the placebo group, RR 1·02, 95% CI 0·88–1·07; p=0·84). The composite primary endpoint of death from all causes or hysterectomy was not reduced with tranexamic acid (534 [5·3%] deaths or hysterectomies in the tranexamic acid group *vs* 546 [5·5%] in the placebo group, RR 0·97, 95% CI 0·87-1·09; p=0·65). Adverse events (including thromboembolic events) did not differ significantly in the tranexamic acid versus placebo group.

Interpretation
Tranexamic acid reduces death due to bleeding in women with post-partum haemorrhage with no adverse effects. When used as a treatment for postpartum haemorrhage, tranexamic acid should be given as soon as possible after bleeding onset.

Editor's Comments
WHO statistics suggest that 25% of maternal deaths are due to PPH. Tranexamic acid reduces bleeding by inhibiting the enzymatic breakdown of fibrinogen and fibrin by plasmin. Based on results of clinical trials in surgery and trauma, tranexamic acid is recommended by WHO (2012) for the treatment of primary post-partum haemorrhage if uterotonics fail to control the bleeding or if the bleeding is thought to be due to trauma. This study suggests that if tranexamic acid is used in the treatment of post-partum hemorrhage it should be given soon after the onset of post-partum hemorrhage alongside uterotonics preferably within 3 h of birth. In contrast, a recent systematic review of 26 studies concluded that existing trials are unreliable, with serious flaws and there is no evidence that TXA prevents postpartum hemorrhage.

Suggested Reading
1. *Ker K, Shakur H, Roberts I. Does tranexamic acid prevent postpartum haemorrhage? A systematic review of randomized controlled trials. BJOG. 2016;123(11):1745-52.*
2. *Novikova N, Hofmeyr GJ, Cluver C. Tranexamic acid for preventing postpartum haemorrhage. Cochrane Database Syst Rev. 2015;16(6):CD007872. doi: 10.1002/14651858.CD007872.*

2. EBioMedicine 2016;6:253-7.

Maternal collapse: Challenging the four-minute rule

Benson MD, Padovano A, Bourjeily G, Zhou Y

Introduction

The current approach to, cardiopulmonary resuscitation of pregnant women in the third trimester has been to adhere to the "four-minute rule": If pulses have not returned within 4 min of the start of resuscitation, perform a cesarean birth so that birth occurs in the next minute. This investigation sought to re-examine the evidence for the four-minute rule.

Methods

A literature review focused on perimortem cesarean birth was performed using the same key words that were used in formulating the "four-minute rule." Maternal and neonatal injury free survival rates as a function of arrest to birth intervals were determined, as well as actual incision to birth intervals.

Results

Both maternal and neonatal injury free survival rates diminished steadily as the time interval from maternal arrest to birth increased. There was no evidence for any specific survival threshold at 4min. Skin incision to birth intervals of 1min occurred in only 10% of women.

Conclusion

Once a decision to deliver is made, care providers should proceed directly to Cesarean birth during maternal cardiac arrest in the third trimester rather than waiting for 4min for restoration of the maternal pulse. Birth within 1min from the start of the incision is uncommon in these circumstances.

Editor's Comments

If resuscitative efforts following maternal circulatory arrest are unsuccessful, cesarean delivery should be commenced at 4 minutes and completed by 5 minutes to optimize fetal outcome- A landmark 1986 review of all case reports led to the adoption of this 4-5 rule for viable pregnancies. An article published in AJOG 2015 suggested modifications based primarily on maternal status. They suggested to change its terminology from peri mortem cesarean delivery (PMCD) to resuscitative hysterotomy and immediate delivery of the baby for Maternal cardio pulmonary arrest (MCPA) with non-shockable rhythm as this will enhance success of other interventions and procedures while optimizing fetal outcome.

Suggested Reading

1. *Rose C, Faksh A, Traynor K etal. Challenging the 4- to 5-minute rule: from perimortem cesarean to resuscitative hysterotomy. Am J Obs & Gyn, Nov 2015; 653.e1*

3) **PLoS One. 2016 Dec 20;11(12):e0168535. doi: 10.1371/journal.pone.0168535.**

Determination of normal ranges of shock index and other haemodynamic variables in the immediate postpartum period: A Cohort study

Nathan HL, Cottam K, Hezelgrave NL, Seed PT, Briley A, Bewley S, Chappell LC, Shennan AH

Objective

To determine the normal ranges of vital signs, including blood pressure (BP), mean arterial pressure (MAP), heart rate (HR) and shock index (SI) (HR/systolic BP), in the immediate postpartum period to inform the development of robust obstetric early warning scores.

Study Design

We conducted a secondary analysis of a prospective observational cohort study evaluating vital signs collected within one hour following delivery in women with estimated blood loss (EBL) <500ml (316 women) delivering at a UK tertiary centre over a one-year period. Simple and multiple linear regression were used to explore associations of demographic and obstetric factors with SI.

Results

Median (90% reference range) was 120 (100-145) for systolic BP, 75 (58-90) for diastolic BP, 90 (73-108) for MAP, 81 (61-102) for HR, and 0.66 (0.52-0.89) for SI. Third stage Syntometrine® ***administration was associated with a 0.03 decrease in SI (p = 0.035) and epidural use with a 0.05 increase (p = 0.003). No other demographic or obstetric factors were associated with a change in*** shock index in this cohort.

Conclusion

This is the first study to determine normal ranges of maternal BP, MAP, HR and SI within one hour of birth, a time of considerable haemodynamic adjustment, with minimal effect of demographic and obstetric factors demonstrated. The lower 90% reference point for systolic BP and upper 90% reference point for HR correspond to triggers used to recognise shock in obstetric practice, as do the upper 90% reference points for systolic and diastolic BP for obstetric hypertensive triggers. The SI upper limit of 0.89 in well postpartum women supports current literature suggesting a threshold of 0.9 as indicating increased risk of adverse outcomes.

Editor's Comments

Prompt identification and treatment are crucial to reduce obstetric haemorrhage related maternal mortality and morbidity. However, blood loss is often underestimated and hence, the importance of routine measurement of vital signs and the use of modified early obstetric warning score (MEOWS) charts in all pregnant and postpartum women, to aid more timely recognition of compromise, cannot be over emphasized. Impending shock may be masked by the hemodynamic changes of pregnancy, making conventional vital signs less useful and signs taken in isolation may miss impending deterioration. Shock index (SI), the ratio of pulse to SBP, is proposed as an earlier marker of hemodynamic compromise in few studies. Its incorporation in early warning system needs exploration.

Suggested Reading

- Nathan H, Cottam K, Natasha L etal. Vital Sign Prediction of Adverse Maternal Outcomes in Women with Hypovolemic Shock: The Role of Shock Index. PLoS One. 2016 Feb 22;11(2):e0148729. doi: 10.1371/journal.pone.0148729.

Calendar of AOGD Skill Workshops (2017-2018)

AOGD & The Department of Obstetrics and Gynecology, UCMS & GTBH plan to hold skill workshop series in the year April 2017 to March 2018.

Basics of Endoscopy in Gynaecology	21st July	**Venue** 7th Floor Seminar Room Department of Obstetrics & Gynaecology MCH Block, GTB Hospital, Delhi **Contacts** Mr Ashish -9136708721 Dr A G Radhika -9868399726 Dr Richa Sharma -9868399747
Interpreting the CTG: practical aspects	1st Week of October	
Techniques for control of PPH (including Bakri Balloon demonstration & internal iliac artery ligation)	1st Week of December	
Basics of Evidence Based Health Care	1st Week of February	

AOGD Skill Workshop

Basics of Endoscopy in Gynaecology

21st July, 2017, 11:00am - 05:00pm

Registration Free

Only 30 spots available. Confirm attendance through Registration Form

Content overview

Lectures followed by skills training

- Laparoscopy: Instruments & safe entry into pelvis
- Hysteroscopy: What every Gynecologist should know
- Principles of energy sources in endoscopic surgery
- Knot tying & suturing techniques
- Sterilization and maintenance of instruments
- Troubleshooting
- Basic Endotrainer session

Registration Form

Name: ..... AOGD Membership No.: .....

Mailing Address: .....

City: ..... State: ..... Pin Code: .....

Affiliated Institution: ..... Designation: .....

Email id: ..... Mobile no.: ..... Tel no.: .....

Filled Form to be sent to:

AOGD Office,7th Floor MCH Block, Department of Obstetrics & Gynecology
UCMS & & Guru Teg Bahadur Hospital, Shahdara, Delhi-110095
info@aogd.org

Proceedings of AOGD Monthly Clinical Meet

AOGD Monthly Clinical Meet was held at Fortis Hospital, Vasant Kunj on 26th May, 2017, 4:00pm-5:00pm. Three interesting presentations were made including a review, a case report and a clinical audit.

Hysteroscopy -2yrs experience in a private urban Hospital in India

Neema Sharma, UP Jha, Ramandeep

Introduction

Hysteroscopy represents standard diagnostic and therapeutic method in the treatment of endometrial pathology. In majority, patient selection for this procedure depends on the preoperative uterine ultrasound scan. Hysteroscopy can be used for removal of polyps or myomas, endometrial tumor resection, synechiolysis, sterilisation or removal of remnants from pregnancy. Hysteroscopic surgery can be also an option for patients who wish to preserve the uterus for the treatment of recurrent bleeding.

Aim

In this study, we present a review of hysteroscopic procedures performed over a 2-year period analyzing the indications,diagnosis and complications associated with it.

Material & Methods

363 hysteroscopic procedures performed over a period of 2 years were reviewed retrospectively. Indications, intraoperative diagnoses, and complications were particularly highlighted.

Results

During study period a total of 363 hysteroscopies were performed. 190 cases were diagnostic, in 170 cases some operative intervention was required, in 2 cases procedure had to be abandoned and in 1 case procedure could not be done.

The most frequent indication for hysteroscopy was menorrhagia (230cases),followed by postmenopausal bleeding (70 cases) , Infertility (40cases) and recurrent miscarriage (10cases).

There were normal findings in 190 cases, endometrial polyp in 70cases, Uterine fibroid in 40 cases, Endometrial hyperplasia in 20 cases, Uterine malignancy in 12 cases, 10 cases each of uterine tuberculosis and Asherman's syndrome, 5 cases of uterine septum, 3 cases of cornual block and 2 cases of retained bone were there. Complication rate was 4.1%. The most frequent surgical complication was intraoperative bleeding in 4 cases, 3 cases each of false passage and post operative infection, 2 cases each of fluid overload and air emboloism, 1case of uterine perforation.

Conclusion

Hysteroscopy is a safe, minimally invasive procedure with a very low rate of complications.

Reproductive outcome in a woman with bilateral ovarian agenesis

Mamta Mishra

Case history

A 25 year old,married woman presented with vague menstrual history. She complained of irregular bleeding only on medication. On examination she was 158cm tall, thin built, breast –tanner stage 2, pubic and axillary hair were absent. External genitalia, vagina and were well developed but uterus was small. Ultrasound suggested infantile uterus and ?hypoplastic or absent ovaries .S FSH was 60.4mIu/ml, LH 28.6mIu/ml and oestradiol was 6pg/ml . S.TSH and prolactin was normal. Karyotype was XX. Diagnosis of hypergonadotropic hypogonadism due to ovarian agenesis was made.

She was started on cyclical progesterone and oestrogen and after one and half year ET increased from 2mm to 7.5mm.IVF with donor ovum and husband sperm was done. She conceived and had dichorionic diamniotic twin pregnancy. She developed morbid oedema at 33 weeks and preeclampsia at 35 weeks.

LSCS was done at 35 weeks and two baby of weight 1.9 kg and 2.3 kg were delivered. She had moderate PPH which responded to conservative management.

Discussion

Incidence of XX gonadal agenesis is 0.0089%. It is a heterogeneous condition. In some forms, the defect is restricted to the gonads, whereas other affected females show neurosensory hearing loss (Perrault syndrome). In another form, brothers may have germ cell aplasia.It is an an autosomal recessive condition. Only two cases of pregnancy in woman with bilateral ovarian agenesis have been reported.

A clinical audit on adequacy of venous thromboembolism prophylaxis in Patients undergoing hysterectomy in Fortis Flt. Lt. Rajan Dhall Hospital, Vasant Kunj.

Maria, UP Jha

Background and Aim

Surveillance studies have found the absolute risk of DVT ranges up to 15-40% in patients hospitalized for gynecologic surgery in U.S. Our aim was to study current status of the following in hysterectomy patients:

a. VTE risk assessment; b. Use of perioperative VTE prophylaxis; c. Adequacy of VTE prophylaxis given.

To compare the above with the standard recommendations by NICE guidelines and make appropriate recommendations regarding VTE prophylaxis protocol in hysterectomy patients in hospital.

Materials and Methods

Retrospective review of 86 case files of women who have undergone hysterectomy(by any route) between 01 Jan 2016 to 31st Dec 2016.

Factors assessed:

1. If patients were assessed on admission and after 24hrs to identify those who are at increased risk of VTE and bleeding.
2. If procedure took more than 60 minutes (surgery+ anaesthesia)
3. Any associated inflammatory condition.
4. Any expected significant reduction in mobility.
5. Other Risk factors (cancer, >60 yrs, dehydration, BMI>30, one or medical co morbidities, personal or family history of VTE, HRT, OC use, Varicose veins)
6. Mechanical prophylaxis (midthigh stockings and ICD pump) given and any contraindications.
7. Pharmacological prophylaxis given and any contraindications.

   Inj clexane 40 mg SC starting 12 hours before major surgery and continued postop and extended for one week postop in all major surgeries and 4 weeks in cancer patients as per NICE guidelines.
8. Was thromboprophylaxis adequate to the level of risk assessed in accordance with NICE guidelines?

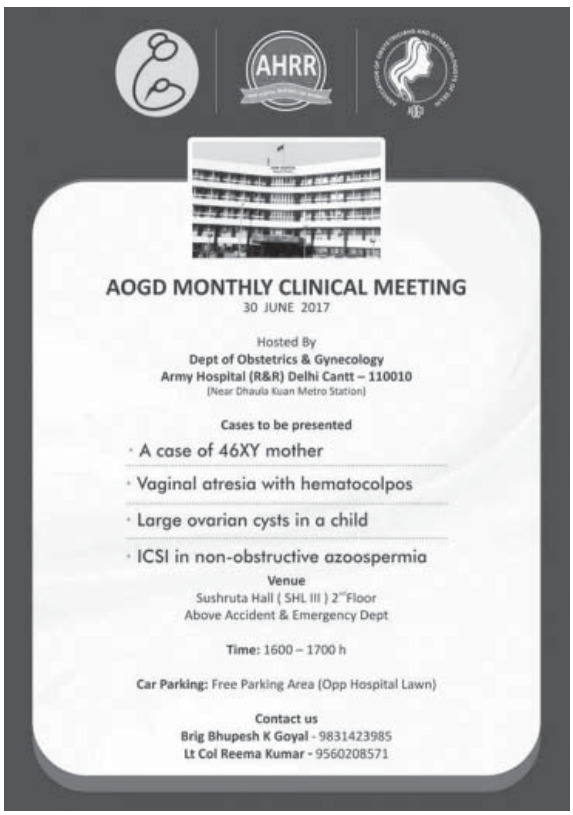
Exclusion criteria-
Patients under the age of 18 and who are admitted to hospital with suspected or diagnosis of DVT or pulmonary embolism.

Results

- None of the patient were assessed for risk factors for VTE.
- 72.61% of the total patient under evaluation did not receive adequate prophylaxis for VTE .
- 20 % of the total patient under evaluation received only preop LMWH.
- 15 .11% of the total patient received only mechanical prophylaxis.
- DVT/PE at the time of admission,as result of surgical complication during same admission : none
- Patient readmitted for DVT/PE within 30 days of surgical procedure: none

Recommendations

Every patient should be assessed preoperatively / at admission for individual risk factor for developing VTE. Preoperative explanation of VTE, importance of stockings, hydration, early ambulation to patient and relatives. Risk factor stratification CAPRINI SCORE needed to start standardized regimen. Postoperatively early ambulation, hydration, chest and leg physiotherapy is recommended.



Quiz Time: *Tick it, Fill it, Click it, Whatsapp/ Email it*

Rashmi, Bindiya Gupta

Asstt Professor, Department of Obstetrics & Gynecology, University College of Medical Sciences & Guru Teg Bahadur Hospital, Delhi

1. All are the components of CAPREG score except:
   a. NYHA Class > II
   b. Cyanosis
   c. Atrial fibrillation
   d. Left heart obstruction
   e. Systemic ventricular ejection fraction <40%

2. All is true for peripartum cardiomyopathy except:
   a. Heart failure in last month of pregnancy or within 12 weeks postpartum
   b. Digitalization is not the treatment of choice
   c. Ejection fraction (EF) <45%, functional shortening <30%
   d. Bromocriptine has a role in management

3. ***Sepsis-induced tissue hypoperfusion*** is defined as ……

4. Which statement is true regarding fluid management in Septic shock:
   a. Use of hydroxyethyl starch is recommended for initial fluid resuscitation
   b. Physiologic perfusion end points: Central venous pressure 8–12 mm Hg, mean arterial pressure greater than 80 mm Hg
   c. Fluid resuscitation is started with the use of warm normal saline or lactated Ringer's
   d. Albumin should not be used for fluid management

5. Name any two scoring systems used in management of septic shock: ……

6. HDU provides grade …….. critical care

7. Indications of shock therapy in maternal resuscitation includes all except:
   a. Ventricular Fibrillation (VF)
   b. Pulseless Ventricular tachycardia (VT)
   c. Asystole (flat line)

8. In typical Amniotic fluid embolism the sequence of events is as follows:
   a. Left heart failure, Respiratory Failure, Seizure, Right heart failure
   b. Respiratory failure, right heart failure, left heart failure, Seizure
   c. Right heart failure, Left heart failure, Respiratory failure, Seizure
   d. Right heart failure, Respiratory failure, Left heart failure, Seizure

9. All these bio markers may be increased in Amniotic Fluid Embolism except
   a. Serum Tryptase
   b. Urinary Histamine
   c. Serum C2 & C3 complements
   d. Serum STN Antigen

10. No of HDU beds required if number of deliveries per month are 400-500
    a. 2
    b. 4
    c. 8
    d. 12

11. Near Miss cases and maternal mortality are together referred to as

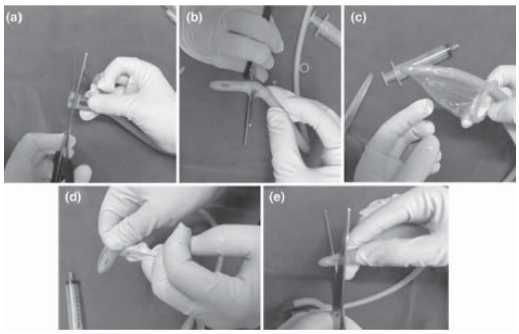
    ……

12. As per MOHFW, for diagnosis of a case of MNM how many criteria must be met
    a. Three including one each from clinical findings, investigations and interventions
    b. Any three from clinical findings, investigations and intervention
    c. Three criteria including one signifying cardiovascular collapse

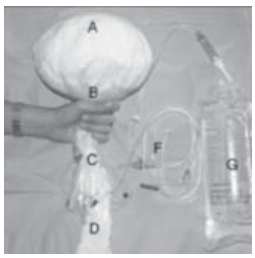
13. Name this system:

……

14. The following sequence shows preparation of ……..



15. This is …….. bag, used for ……. bleeding



16. When is Amniotic Fluid Embolism Day?

……..

17. Near Death Experience consists of all these except:
    a. Sense of peace and well being
    b. Review of life events
    c. Profound desire to be alive
    d. Moving towards a light

18. The goals of transfusions in massive obstetric hemorrhage are all except:
    a. Fibrinogen level above 1.5 gm/L
    b. INR at <1
    c. platelet count at >50×10^9/L

19. Following are not clearly established for management of massive hemorrhage in obstetrics except:
    a. Intraoperative Cell salvage
    b. Recombinant factor VIIa
    c. Tranexamic acid
    d. Ethamsylate

20. All are true regarding anticoagulation and regional anaesthesia except
    a. Regional anaesthesia should be avoided if until 12 hours after the previous prophylactic dose of LMWH
    b. Regional anaesthesia should be avoided if until 24 hours after last therapeutic dose of LMWH.
    c. LMWH should not be given for 12 hours after use of spinal anesthesia
    d. Epidural catheter should not be removed within 12 hours of the most recent injection

Tick the MCQs and fill in the blanks.
Click a pic and whatsapp or email to us
Whatsapp Nos.: 9810645212, 9810719002
Email: info@aogd.org

Winners of Quiz - May Issue

1. Dr Anita Rajorhia **2. Dr Rachna Agarwal** **3. Dr Anshul Grover**

Answers Key to Quiz - May Issue

1. B, 2. C, 3. B, 4. C, 5. C, 6. A, 7. D, 8. C, 9. D,
10. Youth Unite for Victory on AIDS,
11. Peritoneal Alan-Masters windows; Endometriosis,
12. U2bC0V0, Robert's Uterus,
13. Cervico vaginal agenesis with hematometra,
14. Mucinous Ovarian neoplasm…Malignant/ Borderline,
15. Mature Teratoma Ovary,
16. Laparoscopic Ovarian transposition

Postal Registration No. DL(E)-20/5525/2017-19
June 14-15, Date of Publication, June 7-8
Registered with Registrar of Newspapers for India
DELENG/2001/04547/8.25" x 11.25"