12 - Precision in Practice
High-Risk Obstetrics

Gyan - Vahini

From

FOGSI, Food Drugs & Medicosurgical Equipment Committee December- 2025

Dr. Asha Jain

Editor & Chairperson, FOGSI FDMSE Committee

Message From Dr. Sunita Tandulwadkar

Dr. Sunita Tandulwadkar

President FOGSI-2025

High-risk obstetrics lies at the core of maternal and perinatal safety. This issue of the e-magazine focuses on conditions that most strongly influence outcomes in obstetric practice.

The chapter on **"Preeclampsia: Predicting the Storm"** highlights the importance of early risk identification and surveillance. **"Diabetes: CGM & Precision Care"** brings attention to evolving tools that improve glycaemic control and pregnancy outcomes. **"PAS Disorders (Placenta Accreta)"** addresses one of the most dangerous causes of obstetric haemorrhage, requiring anticipation and multidisciplinary planning. The article on **"FGR: Precision in Timing Delivery"** reminds us that correct diagnosis and timely intervention can change fetal outcomes dramatically.

Together, these topics reflect the need for vigilance, preparedness, and evidence-based decision-making. I congratulate the editorial team and authors for addressing these critical areas with clarity and clinical relevance.

FOGSI remains committed to strengthening capacity so that high-risk pregnancies are managed safely, confidently, and with compassion.

Warm regards,
Dr Sunita Tandulwadkar
President, FOGSI

Message from Dr Abha Singh

Dr. Abha Singh
Vice President FOGSI-2025

High-risk obstetric situations often demand swift judgement, coordinated teamwork, and readiness for emergencies. This issue addresses areas that frequently challenge clinicians.

"The PPH Toolkit: Beyond Oxytocin" reinforces structured, stepwise management of obstetric haemorrhage. **"Obstetric Sepsis & Critical Care"** highlights early recognition and timely escalation, which are crucial for maternal survival. **"Preterm Labor & Neuroprotection"** provides practical guidance for improving neonatal outcomes. The chapter on **"Monochorionic Twin Management"** explains the complexity of surveillance and intervention in these high-risk pregnancies.

These articles are particularly relevant for real-world practice, where preparedness often determines outcomes. I appreciate the practical orientation of this issue and encourage clinicians to use it as a reference for team discussions and protocol strengthening.

With best wishes,
Dr Abha Singh
Vice President In-Charge, FOGSI

Message from Dr Suvarna Khadilkar

Dr. Suvarna Khadilkar
Secretary General FOGSI-2025

High-risk obstetrics extends beyond delivery rooms into long-term planning, system readiness, and holistic care. This issue highlights important areas that require structured protocols and multidisciplinary involvement.

"Cardio-Obstetrics: Risk Stratification" emphasises planned care for women with cardiac disease. **"The Obesity Challenge in OBGYN"** addresses a growing contributor to obstetric risk across all stages of pregnancy. **"Thromboembolism (VTE) Safety"** focuses on prevention and timely management of a potentially fatal complication. **"Peripartum Mental Health Crises"** reminds us that psychological safety is as vital as physical health.

These topics underline the need for comprehensive, woman-centred care in high-risk pregnancies. I commend the editorial team for presenting these themes in a structured and clinically meaningful manner.

Best regards,
Dr Suvarna Khadilkar
Secretary General, FOGSI

Dr. Asha Jain
Chairperson
FOGSI FDMSE Committee

FOREWORD

As we bring out the ***12th issue of Gyan Vahini*, **this edition is both special and emotional. It marks the **completion of a full year of Gyan Vahini publications*** and is the ***final issue under the leadership of Dr Sunita Tandulwadkar as President and Dr Abha Singh as Vice President In-Charge***. This moment is one of gratitude, reflection, and quiet pride in what has been collectively achieved.

I begin by offering my heartfelt thanks to ***Dr Sunita Tandulwadkar, whose leadership throughout the year has been visionary yet grounded. Her emphasis on meaningful, practice-oriented academics gave Gyan Vahini its clear direction and purpose. I am deeply grateful to **Dr Abha Singh*** for her constant encouragement, guidance, and faith in this initiative, which helped us sustain momentum across all twelve issues. I also sincerely thank ***Dr Suvarna Khadilkar**, **Secretary General, FOGSI***, for her steady support, administrative clarity, and belief in structured academic communication.

The journey of ***twelve Gyan Vahini issues*** over this year has been intense, fulfilling, and deeply collaborative. Each issue was planned with a single intent—to address real clinical challenges faced by obstetricians and gynecologists across the country. The consistency of thought, relevance of topics, and readability of content are a reflection of the collective commitment of everyone involved.

This concluding issue on ***High-Risk Obstetrics*** is especially apt. High-risk care today is no longer limited to tertiary centres; it is encountered daily in OPDs, labour rooms, and emergency settings. The topics covered—ranging from hypertensive disorders, diabetes, placenta accreta spectrum, fetal growth restriction, postpartum haemorrhage, preterm labour, sepsis, multiple pregnancy, cardiac disease, obesity, thromboembolism, to peripartum mental health—represent the true spectrum of challenges that modern obstetric practice faces.

I place on record my sincere and profuse thanks to ***all the authors*** who contributed to this issue and supported Gyan Vahini through the year. I gratefully acknowledge ***Dr Vishnupriya KMN, Dr Sugandha Goel, Dr Sujayasri Sangita, Dr Jyothi G.S., Dr Neetha George, Dr Rimpi Singla, Dr Okram Sarda Devi, Dr Ginny Gupta, Dr Renu Jain, Dr Prabhdeep Kaur, and Dr Archana Singh****. Each of you brought depth, clarity, and practical wisdom to your chapters. Your willingness to share experience and write with honesty has been the backbone of this publication.

As we close this chapter, we do so with gratitude for the year gone by and confidence for the future. The foundation laid by these twelve Gyan Vahini issues will guide us forward—towards stronger academics, wider participation, and an unwavering focus on patient safety and ethical care.

To every reader who engaged with Gyan Vahini—read it, discussed it, shared it, or applied it in practice—thank you. This publication exists because of collective belief in shared learning. With gratitude for the journey so far, we now look ahead, committed to doing even better for the women we serve.

Dr Asha Jain
Chairperson, Food, Drugs & Medicosurgical Equipment Committee (2025–27), FOGSI

"Know Your Numbers" is an ambitious health initiative.

- This project seeks to gather vital health data- Weight, Blood pressure, Blood Sugar Level with HbA1C, and Hemoglobin level -from women across India.
- By focusing on these key health indicators, the project aims to foster a proactive health management culture among women.
- The data collected will be instrumental in identifying prevalent health issues early and promoting interventions that can significantly reduce the incidence of che diseases.
- This initiative not only emphasizes the importance of regular health monitoring but also strives to empower women with the knowledge and tools needed to take charge of their health, ensuring they lead longer, healthier lives.
- Collect key health data: weight, blood pressure, blood sugar, HbA1C, and hemoglobin from women across India.
- Encourage proactive health management for early identification of prevalent health issues.
- Promote timely interventions to reduce chronic disease incidence.
- Empower women with knowledge and tools for better health and longevity.
- Gather vital health data: weight, blood pressure, blood sugar (HbA1C), and haemoglobin levels from women across India.
- Foster proactive health management among women.
- Identify prevalent health issues early and promote timely interventions.
- Reduce the incidence of chronic diseases through regular monitoring.
- Empower women with knowledge and tools for healthier, longer lives.

SURVEY FOR KNOW YOUR NUMBER (KYN) PROJECT

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01 Preeclampsia: Predicting the Storm

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The burden of preeclampsia on maternal and neonatal morbidity and mortality is well known. Though there are multiple theories attempting to explain preeclampsia, its origins and consequences, a consistent finding is the imbalance of the circulating angiogenic factors.

The commonly described anti-angiogenic factor is soluble fms-like tyrosine kinase 1 (sFlt-1) and the commonly described pro-angiogenic factor is placental growth factor (PlGF).

These entities circulate in the plasma and can be measured using the automated platforms to get absolute values and also derived ratios.

Considering that they are mainly made in the placenta and are effective non-invasive markers of placental health, their importance has gradually increased. The ratio of these molecules is now definitely demonstrated to be associated with onset and severity of preeclampsia and eclampsia. [1]

The importance of the sFlt1/PlGF ratio is that there is a very high negative predictive value for ruling out the development of preeclampsia within 1 week duration and occurrence of maternal outcomes within 2 weeks duration and delivery within 2 weeks duration. These angiogenic markers aid in better decision making and care. Those at risk of severe maternal morbidity can also be identified. [2]

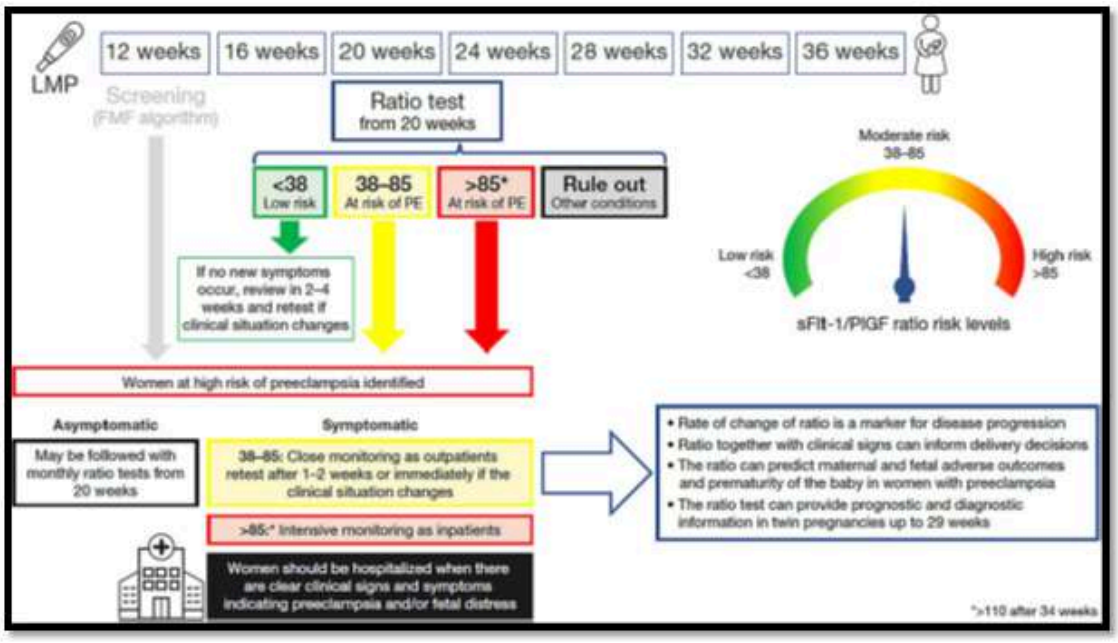

Figure 1. Clinical utility of sFlt-1/PlGF ratio.

Figure 1 described by Verlohren et al [3] summarizes the use of the ratio in the clinical setting after 20 weeks of gestation.
When the test ratio is

- Less than 38: Considered as low risk: If no new symptoms, review after 2 to 4 weeks. Retest if clinical situation changes.
- Between 38 and 85: Considered as "intermediate": Close monitoring is recommended but can be done as outpatient. Retest in 1-2 weeks. If clinical situation changes, retest can be considered earlier
- Above 85: Considered as "high risk": Intense monitoring is recommended.

If there is a change in ratio test, it indicates worsening and need for more intense follow-up. A summary of the most important practice points with respect to this are mentioned in table

Table 1. Consensus statements regarding sFlt-1/PlGF Ratio

General guidance	The sFlt-1/PlGF ratio test can be used from 20 weeks through to 36 + 6 weeks, as a tool for short-term prediction and aid for diagnosis in high-risk women or among women with a clinical suspicion of preeclampsia. The ratio test can also be used in women after 37 weeks, in all cases where preeclampsia is suspected or as a follow-up to evaluate uteroplacental dysfunction.
Management of women who present without clinical features of preeclampsia and have a test result < 38	Women who present without clinical features of preeclampsia should not generally be tested, except those who have a predicted high risk for preeclampsia; these asymptomatic women may be followed with monthly sFlt-1/PlGF ratio tests
Management of women who have clinical features suggestive of preeclampsia but have a test result < 38	For women who have clinical features suggestive of preeclampsia, an sFlt-1/PlGF ratio test result < 38 would rule out preeclampsia in the following 2–4 weeks
Management of women with a ratio test result of 38–85	Enhanced monitoring as outpatients Women with a sFlt1/PlGF ratio test result of 38–85 require enhanced monitoring, with a retest after 1–2 weeks or immediately if the clinical situation changes

Criteria for hospital admission (ratio test result of 38–85)	Women should be hospitalized when there are clear clinical features indicating preeclampsia and/or suspected fetal compromise based on local protocols
Management of women with a test result > 85	Women in this group most likely have, or will develop, preeclampsia and require intensive monitoring, probably as inpatients
Criteria for hospital admission (ratio test result of > 85)	Women should be hospitalized when there are clear clinical features indicating preeclampsia and/or suspected fetal compromise
Use of the ratio test for differential diagnosis of other conditions	The sFlt-1/PlGF ratio test can rule out the imminent occurrence of preeclampsia and may be especially useful in ruling out preeclampsia in conditions that mimic it
The rate of change (or delta, Δ) of ratio	The rate of change of the sFlt-1/PlGF ratio (or delta) indicates severity of disease and is an important marker for the progression of disease
The role of the ratio in estimation of time to delivery	The sFlt-1/PlGF ratio can be used to estimate the expected time to delivery only as an adjunct to other clinical features – delivery decisions should not be made using the ratio test alone
Maternal and fetal adverse outcomes	The sFlt-1/PlGF ratio test can be used to predict maternal adverse outcomes in women with preeclampsia
Twin pregnancies	The sFlt-1/PlGF ratio test can provide prognostic and diagnostic information in twin pregnancies, up to week 29 of gestation

Cost-effectiveness and turn-around time:

In the local Indian scenario, Roche diagnostics offers sFlt-1/PlGF ratio, which is processed centrally in few laboratories and hence, the diagnostic turn around time is longer than what is clinically useful. The cost is around Rs 6000 at present (with local variations).

Improvement of the cost-effectiveness and shorter turn-around times are much needed before it can be implemented [4]

For the prevention of preeclampsia, a variety of interventions have been suggested. However, the stand-out molecule is Aspirin. After the publication of the ASPRE trial in 2018, the utility of aspirin has been confirmed without any doubt.

Either a history-based approach or an investigation-based approach should be followed for identifying patients at risk of hypertensive disorders in pregnancy. Such a screening should be done in the late first trimester and the initiation of aspirin should be done before 16 weeks of gestation for maximum impact.
A summary of the recommendations regarding the timing and condition of the initiation of the aspirin in mentioned in table 2.

Table 2. Summary of clinical recommendations given by different organizations regarding prevention and prediction of preeclampsia

National Institute for Health and Clinical Excellence (NICE)	Advise pregnant women at high risk or with more than 1 moderate risk factor for pre-eclampsia to take 75–150 mg of aspirin daily from 12 weeks until the birth of the baby	High risk factors (any one): · Hypertensive disease during a previous pregnancy · Chronic kidney disease · Autoimmune disease such as systemic lupus erythematosus or antiphospholipid syndrome · Type 1 or type 2 diabetes · Chronic hypertension Moderate risk factors (>1 required): · First pregnancy · Age 40 years or older more at first visit · Family history of pre-eclampsia (mother, sister) · Multi-fetal pregnancy · Pregnancy interval of more than 10 years · BMI of 35 kg/m2
International Federation of Gynecology and American Stroke Association (ASA)	Daily administration of aspirin starting at≤ 16weeks and at a dose of ≥ 100 mg/d at night continuing until 36 weeks' gestation or presence of labor signs	Women with singleton pregnancy at gestational age 11-13 weeks based on risk assessment using a prediction algorithm using a combination of maternal factors with uterine artery pulsatility.

World Health Organization (WHO),2011	WHO recommends that women at high risk of preeclampsia take 75mg of aspirin daily, initiated before 20 weeks of pregnancy.	High risk: · Previous preeclampsia · Diabetes · Chronic hypertension · Renal disease · Autoimmune disease · Multiple pregnancy
FOGSI	Women should be initiated on low-dose aspirin if the GESTOSIS score >=3	Refer to figure 2 which contains the GESTOSIS scoring system. This is recommended by the Indian authorities and its utility has been proved by multiple recent studies

Figure 2. The gestosis scoring system

Risk factors	Score
Age >35 years	1
Age <19 years	1
Maternal anaemia	1
Obesity (BMI >30)	1
Primigravida	1
Short duration of sperm exposure (cohabitation)	1
Woman born as small for gestational age	1
Family history of cardiovascular disease	1
Polycystic ovary syndrome	1
Inter pregnancy interval more than 7 years	1
Conceived with assisted reproductive (IVF/ ICSI) treatment	1
MAP > 85 mm of Hg	1
Chronic vascular disease (dyslipidemia)	1
Excessive weight gain during pregnancy	1
Maternal hypothyroidism	2
Family history of preeclampsia	2
Gestational diabetes mellitus	2
Obesity (BMI > 35 kg/m^2)	2
Multifetal pregnancy	2
Hypertensive disease during previous pregnancy	2
Pregestational diabetes mellitus	3
Chronic hypertension	3
Mental disorders	3
Inherited/acquired thrombophilia	3
Maternal chronic kidney disease	3
Autoimmune disease (SLE/APLAS/RA)	3
Pregnancy with assisted reproductive (OD or surrogacy)	3

Novel methods for prevention of preeclampsia

- Statins – especially pravastatin has shown some progress, but has not yet reached recommendation stage.
- Metformin – Addition of metformin in the first trimester has been attempted for the prevention of preeclampsia.
- Addition of metformin in the mid trimester has been shown to prolong the duration of pregnancy in diagnosed preeclamptics.
- Proton- pump inhibitors. Very few studies have demonstrated that PPIs like esomeperazole have been found to prevent preeclampsia. There are ongoing trials on this topic.
- l -arginine: While l-arginine has been utilized to reduce the adverse effects of patients who have already developed preeclampsia, prevention of preeclampsia has been demonstrated in only one RCT. Results of ongoing studies are expected soon.
- Immunomodulators - Multiple drugs which act on the inflammatory cytokine cascade like tacrolimus, etanercept, eculizumab and sulfasalazine have undergone small animal trials but not yet reached human trial stage.
- Other novel molecules like angiotension-1 autoantibodies, rituximab and losartan are being considered for human trials.

Unanswered questions with respect to aspirin in pregnancy

- Is there a clinical benefit in terms of prevention of preeclampsia if aspirin initiation is done if much earlier in pregnancy – around 6 weeks of gestation – based on a clinical scoring system without waiting for uterine artery doppler at 11-14 weeks?
- What are the sequential ultrasonographic doppler parameters among those who are initiated with aspirin early in pregnancy?
- Is there a benefit of starting aspirin much later in pregnancy (after 24 weeks)?
- Among those who have been initiated on aspirin, is there a minimum duration for which it has to be continued to confer a clinical benefit?
- Among those who have been initiated on aspirin early enough, how early can aspirin be discontinued?

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02 The PPH Toolkit: Beyond Oxytocin

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1. Introduction

Postpartum haemorrhage (PPH) remains one of the most serious obstetric emergencies and is still the leading cause of maternal mortality worldwide, responsible for between one quarter and one third of obstetric deaths (1). Postpartum Haemorrhage is traditionally defined as blood loss of 500mL following vaginal delivery or 1,000 mL following cesarean birth. Despite the widespread use of active management of the third stage of labor (AMTSL) with oxytocin, the global incidence of PPH continues to rise. The "Beyond Oxytocin" paradigm acknowledges that uterine atony—responsible for 70-80% of cases—often requires a synergistic approach that addresses not just myometrial contraction, but also the coagulation cascade and mechanical uterine compression.

Pharmacological Advancements: The WOMAN Trial and TXA

The most significant pharmacological addition to the PPH toolkit in the last decade is Tranexamic Acid (TXA). While oxytocin addresses the contractility of the uterus, TXA addresses the coagulopathy that inevitably follows massive hemorrhage.

The WOMAN Trial Evidence

The World Maternal Antifibrinolytic (WOMAN) trial, a landmark study involving over 20,000 women, provided the definitive evidence for TXA in PPH. The trial demonstrated that:

- **Mortality Reduction**: Death due to bleeding was reduced by nearly one-third (31%) when TXA was administered within 3 hours of birth.(3)
- **The Time-to-Treatment Factor**: Every 15-minute delay in administration significantly diminished the survival benefit. After 3 hours, the benefit was negligible, likely because the coagulopathic process becomes irreversible or transitions into disseminated intravascular coagulation (DIC).(3)
- **Safety Profile**: Critically, the study found no increase in thromboembolic events such as pulmonary embolism or deep vein thrombosis in mothers, nor any adverse effects in breastfed infants.

Integration into Guidelines

As a result of the WOMAN trial, the **World Health Organization (WHO)** and **FIGO** updated their PPH bundles to include 1g of IV TXA as a mandatory first-line treatment for PPH, regardless of the cause of bleeding.

Mechanical Interventions: Bridging the Gap to Surgery

When pharmacological agents fail to achieve a "firm" uterus, the toolkit shifts toward mechanical compression. These interventions are vital "bridge therapies" that stabilize the patient for transfer or definitive surgery.

Uterine Balloon Tamponade (UBT)

ACOG Practice Bulletin 183 highlights UBT as a primary second-line intervention for refractory uterine atony. Devices such as the Bakri Balloon are inserted into the uterine cavity and inflated with 300–500 mL of sterile saline.(5)

- **Mechanism:** The balloon exerts hydrostatic pressure against the uterine wall, which exceeds the systemic venous pressure, thereby arresting capillary and venous bleeding.
- **The "Tamponade Test":** If the balloon successfully controls bleeding, it is left in place for up to 24 hours. If bleeding continues around the balloon, it serves as a clinical signal that the patient requires immediate surgical intervention (e.g., laparotomy or uterine artery embolization).

Surgical Compression Sutures

For cases where PPH occurs during or after a cesarean section, or when UBT fails, surgical sutures offer a conservative alternative to hysterectomy. WHO guidelines state that after the failure of conservative management, compression sutures should be attempted before vessel ligations.(6)

- **The B-Lynch Suture**: Described by RCOG as a "bracing" technique, this suture effectively wraps the uterus to maintain continuous compression.
- According to **RCOG Green-top Guideline 52**, these sutures have a high success rate (up to 90%) but require surgical expertise and carry a small risk of uterine necrosis or Asherman's syndrome. (7)

Systemic Resuscitation: Massive Transfusion Protocols (MTP)

The management of severe PPH has shifted away from aggressive crystalloid resuscitation. The Journal of Trauma and Acute Care Surgery has long advocated for "hemostatic resuscitation," a concept now central to PPH management.

The 1:1:1 Ratio (8)

Historical protocols favoured large volumes of saline or Ringer's Lactate, which often led to dilutional coagulopathy and the "lethal triad" (acidosis, hypothermia, and coagulopathy). Current MTP standards, supported by RCOG 52, recommend a balanced ratio of:

- **1 unit of Packed Red Blood Cells (PRBC)**
- **1 unit of Fresh Frozen Plasma (FFP)**
- **1 unit of Platelets**

The Role of Fibrinogen

Fibrinogen is the first coagulation factor to fall to critical levels during PPH. ACOG Bulletin 183 emphasizes that a fibrinogen level < 200 mg/dL is a strong predictor of severe PPH. The PPH toolkit must therefore include early administration of Cryoprecipitate or Fibrinogen Concentrate to maintain levels above 200mg/L.

The FIGO and E-MOTIVE Bundles: A Unified Strategy

The most modern evolution of the PPH toolkit is the concept of "Bundled Care." The FIGO PPH Bundles (9) and the WHO E-MOTIVE (10) study suggest that treating PPH sequentially (waiting for one drug to fail before trying the next) is too slow.

The **E-MOTIVE** strategy advocates for:

1. **Early Detection**: Using calibrated blood-collection drapes to avoid the 30-50% underestimation typical of visual assessment.
2. **Immediate Response**: Simultaneous administration of uterine massage, oxytocics, TXA, and IV fluids.
3. **Escalation**: Rapid transition to UBT or surgery if the bundle fails.

In clinical trials, this bundled approach reduced the rate of severe PPH (blood loss >1000 mL) by 60%.

MTP Activation & Ratio Strategy

Component	Protocol Detail	Clinical Rationale
Activation Trigger	Blood loss > 1,500 mL or hemodynamic instability (Shock Index > 1.1).	Early activation prevents the "lethal triad" (Acidosis, Hypothermia, Coagulopathy).
Fixed Ratio	1:1:1 Strategy (1 PRBC : 1 FFP : 1 Platelet).	Mimics the composition of whole blood and maintains oncotic pressure.
Fibrinogen	Administer Cryoprecipitate (10 units) if Fibrinogen < 2 g/L.	Fibrinogen is the first factor depleted in PPH; its levels predict severity.
Crystalloid Limit	Max 2 Liters of warmed Isotonic Crystalloid.	Prevents dilutional coagulopathy and pulmonary edema.
Permissive Hypotension	Maintain SBP ~90 mmHg until bleeding is controlled.	Avoids "popping the clot" by over-pressurizing the vascular system.

Note on Monitoring: ACOG 183 recommends "point-of-care" testing (like ROTEM or TEG) if available, to guide specific factor replacement (e.g., Prothrombin Complex Concentrate) rather than blind transfusion.

II. Step-by-Step Surgical Suture Guide (B-Lynch Technique)

When the FIGO "Refractory Bundle" (Uterotonics + UBT) fails, the B-Lynch Suture is the primary surgical intervention to avoid hysterectomy.

1. Preparation & The "Test"

- **Access:** Perform a midline or Pfannenstiel laparotomy (or utilize the existing C-section incision).
- **The Compression Test:** Exteriorize the uterus. Use both hands to manually compress the uterus. If this compression stops the bleeding from the vagina, the B-Lynch suture is likely to be successful.

2. Suture Selection

- Use a **large, curved needle** with **heavy**, **absorbable suture** (e.g., No. 1 or 2 Monocryl or Vicryl). Rapidly absorbable suture is preferred to prevent bowel entrapment as the uterus involutes.

3. The Stitch Path

1.**First Entry**: Enter the uterine cavity 3 cm below the right side of the lower segment incision.
2.**First Exit**: Exit the cavity 3 cm above the upper margin of the incision on the same side.
3.**The "Brace"**: Pass the suture over the fundus (vertical "suspenders") about 4 cm from the right cornua.
4.**Posterior Entry**: Enter the posterior wall of the lower segment at the same level as the anterior incision.
5.**Posterior Exit:** Cross the suture horizontally across the posterior wall and exit the other side.
6.**The Second "Brace"**: Bring the suture back over the fundus on the left side.
7.**Final Entry/Exit**: Repeat the anterior entry/exit on the left side.

4. Tension & Tying

- An assistant must manually compress the uterus before the surgeon ties the knot.
- The suture must be pulled taut to maintain the compression once the assistant releases their hands.
- **RCOG 52 Caution**: Ensure the suture does not slip off the fundus or into the broad ligament.

III. Integration into the FIGO Bundle

The FIGO PPH Bundles emphasize that these tools are not independent. The progression should look like this:

1.**Detection:** Calibrated Drape.
2.**First-Line (The First 15 Mins):** Massage + Oxytocin + TXA (WOMAN Trial protocol).
3.**Refractory (15–30 Mins):** Uterine Balloon Tamponade (UBT) + MTP Activation.
4.**Surgical (30+ Mins):** B-Lynch Suture - Uterine Artery Ligation - Hysterectomy.

Conclusion

The journey "Beyond Oxytocin" represents a transition from a drug-centric model to a systems-centric model. By combining the antifibrinolytic power of TXA, the mechanical efficiency of UBT and B-Lynch sutures, and the physiological support of 1:1:1 Massive Transfusion Protocols, clinicians can significantly reduce maternal mortality. The evidence is clear: speed, bundling, and a multimodal toolkit are the keys to surviving PPH in the 21st century.

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03 Diabetes CGM And Precision Care

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Introduction

Diabetes in pregnancy—including pre-gestational diabetes mellitus (type 1 and type 2) and gestational diabetes mellitus (GDM)—is associated with significant maternal and neonatal morbidity. Adverse outcomes include pre-eclampsia, large-for-gestational-age (LGA) infants, preterm birth, neonatal hypoglycemia, and increased neonatal intensive care unit (NICU) admissions[1]. Optimal glycemic control is therefore central to improving pregnancy outcomes.

Conventional glycemic assessment tools such as glycated hemoglobin (HbA1c) and self-monitoring of blood glucose (SMBG) provide limited insight into daily glucose variability and frequently fail to capture post-prandial excursions and nocturnal dysglycemia[2]. Continuous glucose monitoring (CGM) has emerged as an advanced modality that enables real-time or intermittently scanned assessment of glucose patterns, supporting a precision-based approach to diabetes management in pregnancy.

CGM in pregnancy:

Pregnancy is a state of rapidly changing insulin sensitivity. CGM helps as a precision care in

- Detecting **post-prandial spikes** and **nocturnal hypoglycemia**
- Reducing glycemic variability (not just HbA1c)
- Allowing **real-time treatment adjustments**
- Improving **maternal and fetal outcomes**

Principles of Continuous Glucose Monitoring

CGM systems measure interstitial glucose via a subcutaneous sensor at frequent intervals (typically every 5–15 minutes), generating a comprehensive 24-hour glycemic profile. Key CGM-derived metrics include:

- **Time in Range (TIR)**
- Time Above Range (TAR)
- Time Below Range (TBR)
- Glycemic variability

These metrics provide clinically actionable information beyond HbA1c[3], particularly in pregnancy where short-term glucose fluctuations directly influence fetal growth and metabolic programming.
It also enables **individualized management** by analyzing:

- **Time in range**
- **Glycemic variability**
- **Meal-specific glucose response**
- **Overnight glucose trends**

This allows:

- Tailored insulin timing/doses
- Diet personalization (carbohydrate quality & timing)
- Identification of placental insulin resistance patterns (especially after 28 weeks)

Evidence for CGM in Pregnancy
Pre-gestational Diabetes

Robust evidence supports CGM use in pregnant women with type 1 diabetes. The landmark **CONCEPTT trial** demonstrated that CGM use during pregnancy significantly improved TIR and was associated with reduced rates of LGA infants, neonatal hypoglycemia, and NICU admissions compared with SMBG alone[3]. Importantly, these benefits were achieved without an increase in severe maternal hypoglycemia[3].

Observational studies and cohort analyses in type 2 diabetes also suggest improved glycemic control and reduced neonatal morbidity with CGM-guided management.

Gestational Diabetes Mellitus (GDM)
Evidence for CGM in GDM is evolving. Randomized trials and systematic reviews indicate that CGM may[4]:

- Improve detection of post-prandial hyperglycemia
- Reduce maternal HbA1c and gestational weight gain
- Lower mean birth weight

However, large outcome-driven trials are still limited, and CGM is not universally recommended for all women with GDM.

Time in Range and Pregnancy-Specific Targets
International consensus recommends pregnancy-specific CGM targets:

- **TIR (63–140 mg/dL): >70%**
- TAR (>140 mg/dL): <25%
- TBR (<63 mg/dL): <4%

Studies demonstrate that even modest improvements in TIR (5–10%) are associated with clinically meaningful reductions in adverse neonatal outcomes, reinforcing TIR as a superior metric to HbA1c in pregnancy[3].

WHO Perspective on CGM in Pregnancy

The **World Health Organization** (WHO) currently does **not recommend routine CGM use** in pregnancy within its guidelines on hyperglycemia first detected in pregnancy and antenatal care.

WHO recommendations emphasize:

- Universal or selective screening for GDM
- Management using lifestyle modification, pharmacotherapy, and **self-monitoring of blood glucose (SMBG)**

CGM is acknowledged as an **emerging technology**, but WHO highlights limitations related to cost, accessibility, and insufficient population-level evidence to support universal implementation, particularly in low- and middle-income countries. Thus, CGM is considered **adjunctive rather than standard of care** from a public health perspective.

FIGO Perspective on CGM in Pregnancy

The **International Federation of Gynecology and Obstetrics (FIGO)** has not issued a dedicated standalone guideline on CGM. However, FIGO's influential publication, "The FIGO Initiative on Gestational Diabetes Mellitus: A Pragmatic Guide for Diagnosis, Management, and Care" (2015), provides a framework that supports **individualized glucose monitoring strategies.**

FIGO acknowledges:

- Limitations of HbA1c in pregnancy
- The importance of post-prandial glycemia and glucose variability
- The need for context-specific, resource-sensitive care

Within this framework, CGM is recognized as a useful adjunct tool, particularly in:

- Pre-gestational diabetes
- Insulin-treated GDM
- Cases with discordance between SMBG values and fetal growth
- Suspected post-prandial hyperglycemia or recurrent hypoglycemia[6]

FIGO's Global Declaration on Hyperglycemia in Pregnancy (2018) and subsequent regional declarations further emphasize improving quality of glycemic monitoring, while allowing flexibility based on healthcare resources[7].

Advantages of CGM Over Conventional Monitoring

Compared with SMBG and HbA1c, CGM:

- Captures nocturnal and post-meal glucose excursions
- Identifies glycemic variability associated with fetal overgrowth
- Enables timely therapeutic adjustments
- Enhances patient engagement and self-management

These advantages align with the principles of **precision maternal–fetal medicine.**

Limitations and Challenges

Despite its promise, CGM use in pregnancy is limited by:

- Cost and device availability
- Limited high-quality outcome data in GDM

Need for confirmatory capillary glucose testing in symptomatic hypoglycemia or rapidly changing glucose levels

Conclusion

CGM represents a major advance in the msanagement of diabetes in pregnancy, offering continuous, dynamic insights into glycemic control that are not captured by traditional monitoring methods. While evidence strongly supports CGM use in type 1 diabetes and selected high-risk pregnancies, **WHO does not currently endorse routine CGM, and FIGO supports selective, individualized use** within a pragmatic care framework. Integration of CGM into pregnancy care should therefore be guided by clinical risk, resource availability, and patient-centered decision-making.

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04 Morbidly Adherent Placenta / Placenta Accreta Spectrum (PAS) Disorders

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Morbidly Adherent Placenta (Placenta Accreta Spectrum): From Early Detection to Modern Management

Morbidly adherent placenta — now more accurately termed **Placenta Accreta Spectrum (PAS)** — represents one of the most challenging obstetric complications in modern practice. Characterized by abnormal placental adherence or invasion into the uterine wall, PAS can transform a routine delivery into a life-threatening emergency. With rising cesarean section rates globally, PAS is no longer rare — and its recognition, planning, and multidisciplinary management have become essential components of safe obstetric care.

Understanding Placenta Accreta Spectrum

Placenta Accreta Spectrum encompasses three primary forms:

- **Placenta accreta**: placental villi attach directly to the myometrium without intervening decidua.
- **Placenta increta**: villi invade into the myometrium.
- **Placenta percreta**: invasion extends beyond the uterine serosa, potentially involving bladder, bowel, or pelvic vessels.

Classically, PAS becomes evident **at delivery**, when attempts to remove the placenta trigger catastrophic hemorrhage. Today, the emphasis has shifted toward **prenatal diagnosis**, which significantly improves outcomes through planned delivery, optimized blood resources, and coordinated surgical expertise.

Why PAS Is Increasing

The strongest determinant is prior cesarean delivery. Each subsequent cesarean increases the risk, especially when combined with placenta previa. Additional risk factors include:

- previous uterine surgery or curettage
- assisted reproductive technology
- multiparity
- advanced maternal age

Yet, the presence of risk factors alone is not enough — **skilled imaging remains the cornerstone** of diagnosis.

Early Ultrasound Markers: Seeing the Warning Signs Sooner

Ultrasound, especially when performed by trained clinicians, remains the first-line tool for PAS detection.

First-Trimester Clues

Increasingly, PAS can be suspected as early as 11–14 weeks when risk factors exist. Early markers include:

- Gestational sac implanted low over a previous cesarean scar
- Absence or thinning of the hypoechoic retroplacental zone
- Irregular uterine scar niche with placental tissue protruding
- Increased vascularity at the scar site on color Doppler

While first-trimester diagnosis is not definitive, it flags patients for close follow-up.

Second- and Third-Trimester Markers

By mid-pregnancy, features become clearer:

Loss of the retroplacental clear zone

- **Placental lacunae** ("swiss-cheese" vascular spaces)
- **Thinning or disruption of the uterine serosa–bladder interface**
- **Turbulent flow within lacunae on color Doppler**
- **Myometrial thickness <1 mm overlying placenta**

These findings, when interpreted in clinical context, offer high predictive value. However, ultrasound performance depends heavily on operator skill and maternal anatomy — which is where MRI may assist.

MRI Findings of Placenta Accreta Spectrum (PAS)

MRI is an adjunct tool when ultrasound findings are equivocal, in posterior placentation, obesity, or for surgical planning. Evaluation should be performed on T2-weighted sequences, with attention to placental–myometrial–serosal interfaces.

1. Placental Findings

- Heterogeneous placental signal intensity on T2-weighted images
- Dark intraplacental bands on T2 (thick, irregular, low-signal bands extending from the basal plate)
- Abnormal placental bulge: outward contour deformity of the uterus caused by the placenta
- Focal exophytic placental mass extending beyond the uterine contour (suggests increta/percreta)

2. Myometrial and Uterine Wall Findings

- Thinning or focal interruption of the myometrium beneath the placental bed
- Loss or irregularity of the hypointense myometrial–placental interface
- Uterine bulging with distorted uterine contour
- Disruption of the uterine serosa (highly suggestive of placenta percreta)

3. Extrauterine Extension (Key for Severity Assessment)

- Placental tissue breaching the uterine serosa
- Bladder tenting or focal bladder wall deformity
- Direct placental invasion of adjacent organs, most commonly the bladder
- Placental tissue extending into parametrial tissues, pelvic sidewalls, or broad ligament

4. Vascular Findings

- Marked placental and subplacental vascularity
- Dilated, tortuous flow voids within the placenta and myometrium
- Bridging vessels extending beyond the uterine serosa toward adjacent organs

5. Ancillary Findings

- Abnormal placental location, commonly over a prior cesarean scar
- Associated placenta previa
- Distortion of pelvic anatomy due to invasive placental tissue

MRI vs Ultrasound: Clarifying the Picture, Not Competing

MRI does **not replace ultrasound**, but complements it in selected cases. MRI is most useful when:

- placental location is posterior
- ultrasound findings are equivocal
- invasive disease, particularly percreta, is suspected

MRI can delineate **depth and lateral spread**, helping surgeons anticipate bladder or parametrial involvement. However, it is costlier, less widely available, and should be reserved for problem-solving — not routine screening.

The Multidisciplinary "Center of Excellence" Approach

One of the most transformative developments in PAS care is the move toward organized, team-based management. FIGO and ACOG emphasize that
outcomes dramatically improve when women with suspected PAS deliver at specialized centers.

What Makes a PAS Center of Excellence?

A designated center typically provides:

Experienced maternal–fetal medicine specialists

Gynecologic oncologists or pelvic surgeons skilled in complex dissections

- Readily available massive transfusion protocols and blood bank coordination
- Interventional radiology for balloon occlusion or embolization (where appropriate)
- Urologists and general surgeons on standby
- Advanced anesthesia and critical care support
- Structured checklists and simulation-based training

The cornerstone principle is **planned, elective delivery**, ideally between **34–36 weeks**, before labor or bleeding begins. Women are counseled comprehensively about risks, surgical strategies, and fertility implications enhancing both safety and autonomy.

Conservative vs. Radical Surgery: Choosing the Right Path

Historically, the standard treatment for PAS — especially accreta and increta — was **cesarean hysterectomy with the placenta left in situ**. This remains the safest option in many cases, particularly when bleeding is brisk or percreta threatens vital structures.

But evolving experience has reopened discussions about **uterus-preserving strategies**, especially for women desiring fertility or when hysterectomy carries exceptional risk.

Radical Management: Cesarean Hysterectomy

Advantages

- Definitive control of hemorrhage
- Lower risk of delayed complications such as infection or secondary hemorrhage
- Predictable surgical endpoint in experienced hands

Challenges

- Significant blood loss possible
- Longer operative time and recovery
- Loss of fertility
- Increased risk of adjacent organ injury, particularly bladder and ureter

Most guidelines still consider cesarean hysterectomy the **preferred approach** for extensive PAS, provided it occurs in a prepared center with expert surgeons.

Conservative Management: Preserving the Uterus

Conservative strategies include:

Leaving the placenta in situ with delayed resorption

- Limited resection with uterine reconstruction
- Use of uterine artery ligation, balloon occlusion, or embolization
- Adjunctive therapies such as methotrexate (used less commonly now)

Potential benefits

- Preserved fertility
- Reduced immediate surgical trauma

Risks

- Secondary hemorrhage weeks later
- Infection or sepsis
- Need for subsequent emergency hysterectomy
- Uncertain long-term uterine function

Evidence from BJOG and other journals shows that while conservative management can succeed in carefully selected patients, it requires meticulous monitoring, frequent imaging, and patient adherence — making it suitable only in specialized settings.

Counseling, Planning, and Emotional Support

Beyond technical choices, PAS carries profound emotional weight. Discussions must cover:

- maternal risks, including transfusion and potential ICU stay
- likelihood of hysterectomy even when conservative plans are attempted
- neonatal timing and outcomes
- future reproductive planning

Psychological support, including postpartum counseling, is essential. Many women grieve the unexpected loss of fertility or the fear associated with high-risk surgery — experiences that deserve validation, not minimization.

Future Directions: Prevention and Precision

As cesarean rates climb, preventing PAS begins with judicious decision-making avoiding non-medically indicated primary cesarean deliveries when safe alternatives exist. Research is also advancing toward:

- improved risk-stratification tools
- standardized imaging scoring systems
- registries capturing real-world outcomes
- refined surgical and interventional radiologic techniques

Collaboration across institutions — and adherence to consensus guidelines — will continue to shape safer care pathways.

Key Takeaways

- PAS is rising worldwide, driven largely by previous cesarean delivery.
- Early recognition using targeted ultrasound and selective MRI is critical.
- Outcomes improve markedly in multidisciplinary Centers of Excellence.

- Cesarean hysterectomy remains the gold standard for many cases, but conservative options exist for selected patients under expert supervision.

- Thoughtful counseling, coordinated planning, and long-term follow-up are central to patient-centered care.

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05 Cardio Obstetrics: Risk Stratification

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CARPREG II (Cardiac Disease in Pregnancy II)
is a clinical risk prediction index published in 2018. It is used to estimate the likelihood of maternal cardiac complications in pregnant women with pre-existing heart disease.

PREDICTOR	POINTS
Prior cardiac events or arrhythmias	3
Baseline NYHA III-IV or cyanosis	3
Mechanical valve	3
Ventricular dysfunction	2
High risk left-sided valve disease/ left ventricular outflow tract obstruction	2
Pulmonary hypertension	2
Coronary artery disease	2
High risk aortopathy	2
No prior cardiac intervention	1
Late pregnancy assessment	1

Predicted Risk of Primary Cardiac Event:

- 0 to 1 points (5%)
- 2 points (10%)
- 3 points (15%)
- 4 points (22%)
- >4 points (41%)

1. Risk Predictors (The 10 Factors)
The score is calculated by assigning weighted points to 10 specific predictors identified in a study of nearly 2,000 pregnancies:
General Predictors (Historical/Functional):

- **Prior cardiac events or arrhythmias** (e.g., heart failure, TIA, stroke) – **3 points**
- **Poor functional class (NYHA III/IV) or cyanosis – 3 points**
- **Left ventricular dysfunction** (LVEF <55%) – **2 points**
- **No prior cardiac interventions** (e.g., unrepaired defect) – **1 point**

Lesion-Specific Predictors:

- **Mechanical heart valves** – **3 points**
- **High-risk valve disease** or left ventricular outflow tract obstruction – **2 points**

Lesion-Specific Predictors:

- **Mechanical heart valves – 3 points**
- **High-risk valve disease** or left ventricular outflow tract obstruction – **2 points**
- **High-risk aortopathy** (e.g., Marfan syndrome with dilated aorta) – **2 points**
- **Pulmonary hypertension** (RVSP >49 mmHg) – **2 points**
- **Coronary artery disease** – **2 points**

Process of Care Predictor:

- **Late pregnancy assessment** (first visit after 20 weeks) – **1 point**

2. Scoring and Estimated Risk

The total sum of points determines the patient's risk category and the expected frequency of primary cardiac events:

Points	Risk of Cardiac Event
0–1	5%
2	10%
3	15%
4	22%
>4	41%

3. Clinical Utility and Comparisons

- **Improved Accuracy:** CARPREG II generally demonstrates better predictive performance (C-statistic/AUC ~0.77–0.78) than the original CARPREG score, the ZAHARA score, or the mWHO classification.
- **Validation:** It has been validated in various populations, though some studies (including a 2025 study) suggest its performance may vary based on whether the heart disease is congenital or acquired.

Application: It is intended for use during pre-conception or early pregnancy counseling to help multidisciplinary teams (cardio-obstetric teams) plan the level of

monitoring and delivery care needed. The World Health Organization (WHO) classification for heart disease in pregnancy, commonly referred to as the **modified**

WHO (mWHO) classification
is the gold standard for predicting maternal cardiovascular risk. It categorizes pregnant women into four risk classes based on their underlying cardiac condition and functional status.

mWHO Risk Classification

mWHO Class	Risk Level	Maternal Mortality & Morbidity Risk	Typical Conditions
Class I	Low Risk	No detectable increase in mortality; no or mild increase in morbidity.	Repaired simple lesions (ASD, VSD, PDA); small or mild pulmonary stenosis; mitral valve prolapse.
Class II	Mild Risk	Small increase in mortality; moderate increase in morbidity.	Unrepaired ASD or VSD; repaired Tetralogy of Fallot; most supraventricular arrhythmias.
Class III	High Risk	Significantly increased risk of mortality or severe morbidity.	Mechanical valves; Fontan circulation; systemic right ventricle; cyanotic heart disease; moderate mitral stenosis; severe asymptomatic aortic stenosis.
Class IV	Extremely high risk	Extremely high risk of mortality or severe morbidity.	Pulmonary arterial hypertension; severe systemic ventricular dysfunction (EF <30%); severe symptomatic aortic stenosis; severe mitral stenosis.

Clinical Management by Class

- **Class I**: Care can typically be managed at a regional or local hospital.
- **Class II**: Management usually involves a shared care model between regional and specialized centers.
- **Class III**: Requires intensive specialist monitoring by a multidisciplinary Pregnancy Heart Team in a tertiary center throughout pregnancy and the postpartum period.
- **Class IV**: Pregnancy is contraindicated. If pregnancy occurs, termination should be discussed due to the severe threat to the mother's life.

Key Indicators for Assessment Risk stratification often involves objective measures to refine individual risk within these classes:

- **Ventricular Function**: Left ventricular ejection fraction (LVEF) <40% is a significant predictor of adverse outcomes.
- **Functional Class**: NYHA Class III or IV symptoms before or during pregnancy indicate high risk.
- **Valve Severity**: Specific area cut-offs (e.g., mitral valve area <1.0 cm2) or aortic valve area <1.0 cm2) define severe stenosis

PERIPARTUM CARDIOMYOPATHY

Idiopathic cardiomyopathy presenting with heart failure

a.Secondary to LV systolic dysfunction with an LVEF < 45% with or without LV dilation
b.Towards the end of pregnancy or upto 5 months postpartum
C.Absence of another identifiable cause of heart failure

RISK FACTORS

Age > 30 years
Parity > 4
Multiple pregnancy
Pre-eclampsia / Eclampsia/ Postpartum HT
Long term (> 4wks) tocolytic Rx.
Obesity
Maternal cocaine abuse

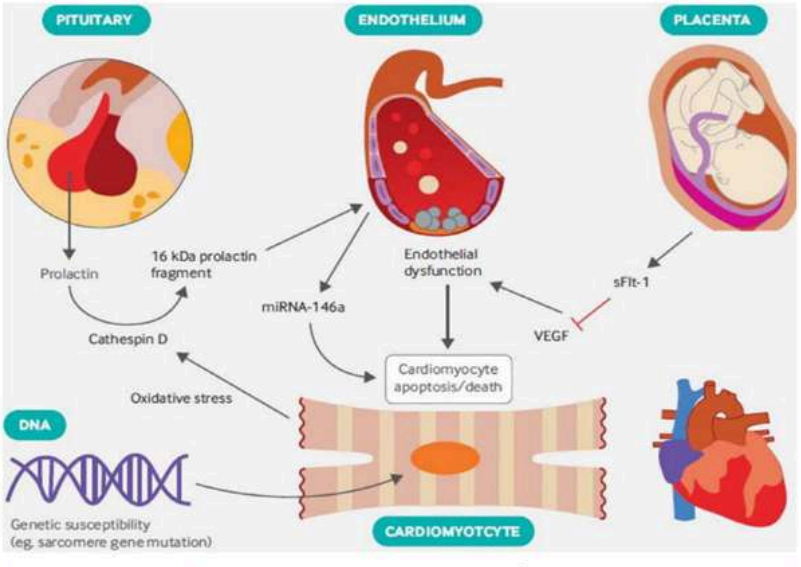


Figure 1. Two-hit hypothesis explanation for pathogenesis of peripartum cardiomyopathy.[9] Secretion of prolactin by the anterior pituitary enhanced production of endothelial microRNA-146a (miRNA-146a) and placental secretion of soluble fms-like tyrosine kinase receptor 1 (sFlt-1) or a background of genetic susceptibility ultimately leads to endothelial dysfunction and cardiomyocyte apoptosis. Abbreviations: VEGF = vascular endothelial growth factor. Figure reproduced with permission. [Colour figure can be viewed at wileyonlinelibrary.com]

78% develops PPCM – within 4months of delivery
9% - last month of pregnancy
13% - either prior to last month of pregnancy or 4 Months after delivery

Symptoms of PPCM
Dyspnoea,Orthopnoea,PND,PedalOedema,Unexplained cough,Dizziness,Palpitation,FatigueChest,Fatigue,Chest pain
SIGNS
Sinus tachycardia, ↑ JVP,Lung crepitations

INVESTIGATIONS
PPCM often a diagnosis of exclusion.
High index of suspicion and timely work up helps in early diagnosis

1. ECG – No specific pattern
2. Echo – LVEF <45%
3. Ventricles may or may not be dilated.

Most helpful tool for diagnosis and prognosis:
LV-end diastolic diameter (LVEDD) > 6cm
LVEF < 30%
Less prospect for spontaneous recovery,
↑ Need for mechanical support, transplant and death
Blood tests
BNP ↑ (N= < 100 pgm/ml)
NT - Pro BNP ↑ (N= < 300 pgm/ml)
CRP & TC – Non specific, often Increased
Hb - Decreased
Troponin T – Normal (marker of cardiac myocyte injury)
ABG – if pt. unstable
Chest X-ray – Non-specific
Cardiac MRI – Not done in pregnancy
Endomyocardial biopsy – Rarely done

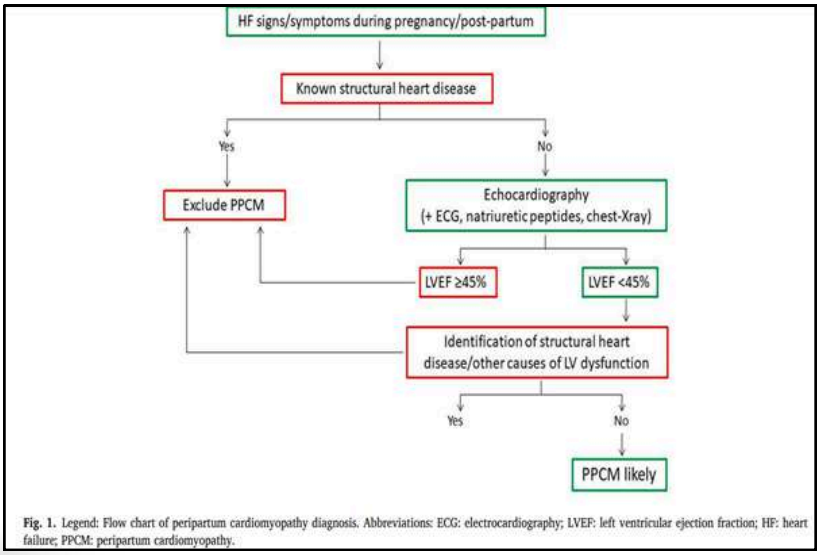


Fig. 1. Legend: Flow chart of peripartum cardiomyopathy diagnosis. Abbreviations: ECG: electrocardiography; LVEF: left ventricular ejection fraction; HF: heart failure; PPCM: peripartum cardiomyopathy.

MANAGEMENT OF PPCM

Broadly divided into 4 groups

Pregnant and hemodynamically stable

Postpartum breast feeding and haemodynamically stable

Postpartum and not breast feeding

Haemodynamically unstable

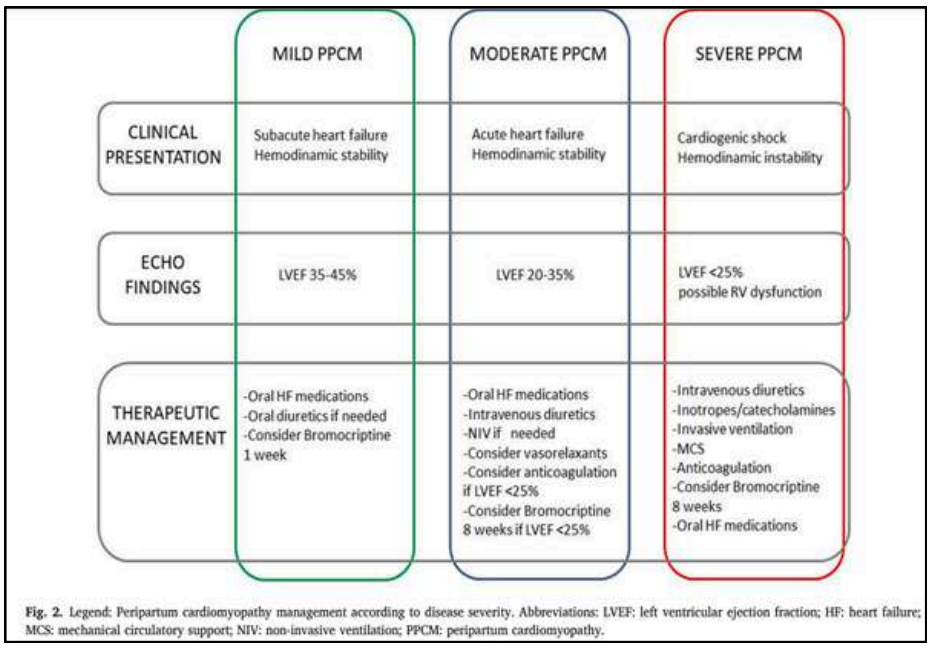


Fig. 2. Legend: Peripartum cardiomyopathy management according to disease severity. Abbreviations: LVEF: left ventricular ejection fraction; HF: heart failure; MCS: mechanical circulatory support; NIV: non-invasive ventilation; PPCM: peripartum cardiomyopathy.

BOARD REGIME

B – Bromocriptine (targeted drug)

O – Oral heart failure therapy

A - Anticoagulation

R – vaso Relaxing agents

D – Diuretics

A.Pregnant & haemodynamically stable

- Salt restriction
- Loop diuretics - furosemide / bumetanide

- Beta blockers- Selectiveβ_1, antagonists (preferred)
- - Metoprolol

-ACEi, ARB's, MRA are contraindicated

- Vasodilators – Hydralazine + Nitrates
- Anticoagulation - LMWH

Consider Bromocriptine

B.Postpartum breast feeding & Stable pt - Mx

Continue beta-blockers, hydralazine, Nitrate

ACEi & ARB's are safe (stop hydralazine & nitrates)

LMWH

Warfarin safe

C.Postpartum and not breast feeding-ALL DRUGS are safe
D.HEMPODYNAMICALLY UNSTABLE

Need rapid & aggressive therapy

Admission in ICU

Initial Mx includes:

1. Optimisation of preload – by IV diuretics

- IV vasodilators (Nitrates, hydralazine) if SBP ≥ 110 mm of Hg

2. Adequate oxygenation

- Maintain SPO2 > 95%
  - CPAP (Non-invasive ventilation)
  - Intubation & ventilation in refractory Hypoxia

3. Circulatory support

- Inotropes / Vasopressors
  - In cardiogenic shock

Noradrenaline - prefered

Adrenaline & Dobutamine – less preferred

Levosimendan (calcium sensitising agent) – preferred

4. Mechanical circulatory support when no improvement in circulatory stability despite optimal medical Mx.

5. Use of Bromocriptine (Dopamine D2 agonist)

(ESC 2018. IIb recommendation)

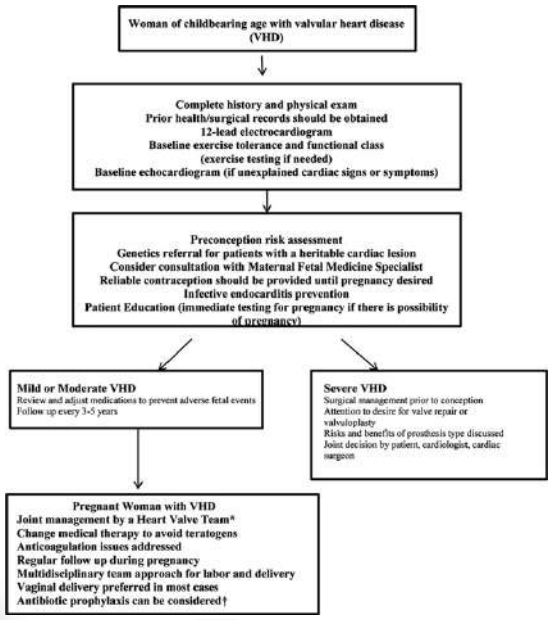
- Start LMWH when patient on Bromocriptine
- Dose = 2.5mg daily for 1 wk (in uncomplicated case)

- Dose = 2.5mg for 2 wks, then 2.5mg od for 6 wks

when LVEF < 25% with or without associated cardiogenic shock)

6. Anticoagulants
7. Oral HF medications
8. Urgent Delivery
   - When heart failure occurs in pregnancy

VALVULAR HEART DISEASE



An urgent or emergency structural intervention during pregnancy, could be required in the following clinical scenarios:

- Patients with high-risk VHD (mWHO Class IV) that was unknown at the time of conception (the incidence of this phenomenon is increasing due to migration flows) and when pregnancy interruption is refused.
- Patients with lower risk VHD, haemodynamic instability and refractory symptoms despite optimal medical therapy.
- Patients with acute onset of severe VHD during pregnancy (i.e. acute severe mitral regurgitation due to chordal rupture).
- Percutaneous Mitral Balloon Valvuloplasty for Mitral Stenosis

Treatment for mitral stenosis (MS) in pregnancy

focuses on managing symptoms with medications (beta-blockers, diuretics, anticoagulants), controlling heart rate, limiting activity, and potentially performing a balloon valvuloplasty if medical therapy fails, reserving surgery for severe, unresponsive cases. Pre-pregnancy counseling is crucial, and interventions like valve repair/replacement or balloon procedures are considered to improve maternal outcomes, with percutaneous balloon mitral valvuloplasty (PBMV) being a preferred intervention during pregnancy to minimize fetal risk.

Medical Management (First Line)

Heart Rate Control: Beta-blockers (like atenolol, propranolol, or esmolol) are used to slow the heart rate, reducing symptoms.

Fluid Management: Diuretics (e.g., furosemide) help manage fluid buildup (pulmonary congestion).

Anticoagulation: Low-molecular-weight heparin is used to prevent blood clots, especially with atrial fibrillation, due to pregnancy's increased clotting risk.

Activity Restriction: Limiting physical activity, sometimes requiring bed rest, helps manage symptoms.

Interventional & Surgical Options (If Medical Therapy Fails)

Percutaneous Balloon Mitral Valvuloplasty (PBMV): The preferred intervention during pregnancy for severe symptomatic MS, usually in the second trimester, to avoid fetal radiation, with high success rates and reduced risk compared to surgery.

Mitral Valve Surgery: Open commissurotomy or valve replacement is reserved for severe cases unresponsive to PBMV or with valve calcification/thrombi, with higher risks to the fetus.

Labor & Delivery Management

Vaginal Delivery: Generally preferred with good pain control (epidural anesthesia) to minimize tachycardia.

Cesarean Section: Performed only for obstetric reasons.

Monitoring: Invasive hemodynamic monitoring and careful management of fluid shifts are crucial during labor and postpartum.
Pre-Pregnancy Planning
Counseling to manage MS and potentially undergo valve intervention (repair/replacement) before conception is ideal for patients with severe symptoms.

Treatment for mitral regurgitation (MR) in pregnancy
focuses on managing symptoms with medication (beta-blockers, diuretics, nitrates/hydralazine) and activity restriction, aiming to avoid surgery if possible; interventions like valve repair (TEER) or replacement are reserved for severe, unresponsive cases, ideally done pre-pregnancy, while labor involves minimizing tachycardia with epidurals and prioritizing vaginal delivery. A multidisciplinary team approach is crucial for managing this complex condition.

Medical Management (During Pregnancy)
Activity Restriction & Rest: Limit physical activity, with bed rest in later stages for symptomatic patients.
Medications:
Beta-blockers: To control heart rate (e.g., <110 bpm).
Diuretics: Cautiously used to manage pulmonary edema, avoiding sudden preload drops.
Vasodilators: Hydralazine and nitrates for symptomatic relief if needed.
ACE Inhibitors/ARBs: Contraindicated in pregnancy.
Anticoagulation: If mechanical valves are present, warfarin is used (avoiding first trimester).

Interventional & Surgical Options (If Medical Therapy Fails)
Pre-Pregnancy: Surgery (repair preferred) or TEER (MitraClip) is ideal for severe MR before conception.
During Pregnancy:
TEER (MitraClip): A minimally invasive option for secondary MR, showing feasibility and safety.
Surgery: Reserved for life-threatening conditions (e.g., endocarditis, uncontrolled heart failure).
Delivery: Vaginal delivery is preferred; C-section only for obstetric reasons, with epidural anesthesia to control pain and tachycardia.

Postpartum Management
Treat anemia, optimize hemodynamics, and continue medical management.
Contraception counseling and anticoagulation management are important.
Multidisciplinary Team: Involves cardiologists, obstetricians, anesthesiologists, and heart valve specialists.
Fetal/Maternal Risk: High-risk patients need careful monitoring due to increased fluid, heart strain, and risk of heart failure.

Managing aortic stenosis (AS) in pregnancy

requires a multidisciplinary cardio-obstetrics team to balance maternal cardiac needs with fetal well-being, focusing on medical management like bed rest and beta-blockers for symptomatic cases, with intervention (balloon valvuloplasty, TAVR, surgery) reserved for refractory severe symptoms, and delivery often via C-section for severe disease to control hemodynamics, emphasizing close monitoring and avoiding medications that decrease afterload.

Management Principles

Multidisciplinary Care: Essential team includes cardiologists, obstetricians, anesthesiologists, and surgeons.

Preconception Counseling: Ideal for addressing severe AS before pregnancy, potentially with valve replacement.

Close Monitoring: Frequent echocardiograms to track valve gradients, which increase during pregnancy, and BNP levels to assess cardiac symptoms.

Medical Management:

Restriction: Strict bed rest.

Medications: Beta-blockers, careful use of diuretics; avoid drugs that reduce afterload (like nitrates).

Intervention During Pregnancy: Considered for refractory symptoms, ideally in the second trimester, using procedures like balloon aortic valvuloplasty (BAV) or TAVR, with transesophageal echo (TEE) to guide.

Delivery Management:

Cesarean Section (C-section): Often preferred for severe AS due to hemodynamic instability during vaginal delivery's second stage.

Spontaneous Vaginal Delivery: May be preferred if severe AS is absent (e.g., only moderate disease).

Anesthesia: Regional anesthesia (spinal/epidural) is often used and effective.

Postpartum: Continued monitoring is crucial as gradients can remain elevated.

Risk Factors & Considerations

Increased Risk: Severe AS significantly raises risks of maternal heart failure, arrhythmia, preterm birth, and fetal growth restriction.

Symptom Mimicry: Pregnancy symptoms (shortness of breath, fatigue) can mask cardiac decompensation, requiring thorough evaluation.

Fetal Impact: Critical AS can lead to hypoplastic left heart syndrome, but fetal intervention can sometimes prevent this.

Goal

To optimize maternal hemodynamics and fetal outcomes through careful, individualized management, often by avoiding high-risk scenarios and intervening when necessary.

Managing aortic regurgitation (AR) in pregnancy

involves a multidisciplinary team, focusing on close monitoring, lifestyle adjustments (salt restriction), medications like vasodilators

(hydralazine/nitrates), and potentially delaying interventions until postpartum due to high fetal risks, though surgery or TAVR (Transcatheter Aortic Valve Replacement) may be needed for severe symptoms, ideally in the second trimester. Mild-moderate AR is often well-tolerated due to reduced systemic vascular resistance, but severe cases need careful planning for delivery and anticoagulation if a prosthetic valve is present.

Pre-Pregnancy Counseling

Risk Assessment: Evaluate valve function and left ventricular (LV) status; severe symptomatic AR or LV dysfunction may warrant intervention before conception.

Aortopathy Screening: Screen for ascending aorta dilation, especially with bicuspid valves, as this increases dissection risk.

During Pregnancy Management (Multidisciplinary Team)

Monitoring: Regular check-ups with cardiology and maternal-fetal medicine are crucial.

Medical Therapy:

Salt restriction, diuretics, and digoxin for symptomatic patients with LV dysfunction.

Hydralazine and nitrates are preferred vasodilators over ACE inhibitors (contraindicated).

Activity: Avoid strenuous activity, manage heart rate, and ensure adequate rest.

Delivery Management

Vaginal Delivery: Often preferred for stable patients to avoid large blood volume shifts of C-sections.

Caesarean Section: May be better for high-risk cases as it provides a controlled environment.

Anesthesia: Careful selection is needed (e.g., spinal-epidural) to maintain hemodynamics, avoiding agents that drastically lower vascular resistance.

Interventions (If Necessary)

Timing: Surgery or valve interventions (like TAVR) are ideally done in the second trimester, when fetal development is more complete and risks lower.

Transcatheter Balloon Aortic Valvuloplasty (BAV): May be considered in specific valve anatomies, but cautiously, as it can worsen regurgitation.

TAVR/Surgery: Reserved for severe, refractory symptoms, with careful consideration of risks.

Anticoagulation (For Mechanical Valves)

Challenge: Warfarin crosses the placenta (teratogenic) but is effective; Heparin/LMWH are safer for the fetus but less effective and require careful dosing.

Strategy: Often involves bridging therapy with UFH/LMWH and transitioning to warfarin, with decisions tailored by the team.

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06 Obstetric Sepsis & Critical Care

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Background: Pregnancy-related infections are responsible for 10.7% of maternal deaths according to the WHO's recent estimates. Results from the multi-country Global Maternal Sepsis Study (GLOSS) suggested that 70.4 pregnant or recently pregnant women per 1000 livebirths admitted or already hospitalized present with maternal infection, with large variations between regions. According to GLOSS, the most common sources were those of the urinary tract (27.9%), genital tract (endometritis (15.1%), chorioamnionitis (14.9%), and abortion-related uterine infection (8·5%), skin or soft tissues (14.8%), and respiratory tract (9%).

Definitions:
Maternal sepsis: A life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion, or postpartum period.

Septic shock: A clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain an adequate blood pressure (MAP 65 mm Hg or more), alongside a persistent serum lactate level more than 2 mmol/L despite adequate volume resuscitation.

Common organisms implicated in maternal sepsis include Escherichia coli, Group B beta-haemolytic streptococcus (GBS), Anaerobes, Staphylococcus aureus, Group A beta-haemolytic streptococcus, Coliforms other than Escherichia coli, Haemophilus influenzae, and Listeria monocytogenes.

Risk factors for maternal sepsis:

Maternal	Obstetric
Obesity	Prolonged rupture of membranes
Diabetes in pregnancy	Caesarean birth

Iron deficiency anaemia	Vaginal trauma
Maternal age > 35 years	Retained pregnancy tissue
Impaired immunity/Immunosuppressant medication	Amniocentesis and other invasive procedures
Women of ethnic minority	Multiple gestation
Renal/Cardiac/Liver disease	Cervical cerclage
History of pelvic infection	
Contact with iGAS	
Intravenous drug use	

Think Sepsis to treat sepsis:

Key to reduction in maternal deaths from sepsis is high level of vigilance and to "Think Sepsis" at an early stage with any unwell, pregnant or recently pregnant woman. Infection should be suspected in pregnant or postpartum patient with significant abdominal pain not relieved by usual analgesia or persistent vaginal bleeding. Early recognition is vital because disease progression may be much more rapid than in the non-pregnant state and deterioration may be abrupt following a period of physiological compensation. Moreover, signs of infection, such as fever, may not always be present. Early escalation of care is recommended.

Clinical presentation may suggest actual pathogen and helps in appropriate treatment:

- Severe pain, out of proportion to clinical signs, suggests necrotising fasciitis.
- Blisters developing on a background of inflammation or a watery vaginal discharge suggests haemolytic streptococcal infection.
- A 'sunburn' rash and conjunctival suffusion suggests early TSS (i.e. due to S. aureus)

Red Flags that indicate high risk of sepsis (recommend immediate initiation of sepsis bundle)

Objective evidence of altered mental state	GCS < 15 or 'not alert' in AVPU: Alert, vocal, pain, unresponsive (AVPU) classification
Respiratory rate	≥ 25 breaths/min
Oxygen saturation	< 94% on room air
Heart rate	> 130 bpm
Blood pressure Systolic	< 90 mmHg
Urine output	Not passed urine in > 12 hours or if catheterised < 0.5 ml/kg urine per hour

Amber flags or features that indicate moderate risk of sepsis (review within 1 hour)
Behavioural/mental status change
Acute deterioration in functional ability
Respiratory rate: 21–24 breaths/minute
Heart rate: 100–130 beats/minute or new dysrhythmia
Systolic BP: 91–100 mmHg
Urine output: Not passed urine in last 12 hours or if catheterised 0.5–1 ml/kg urine per hour
Has had invasive procedure in last 6 weeks
Impaired immune system (illness or medication, including oral steroids)
Temperature: < 36C or > 38C
Current diabetes or gestational diabetes
Close contact with GAS during the seven days before the onset of illness
Prolonged rupture of membranes 18–24 hours
Prolonged vaginal bleeding and abdominal pain post birth
Offensive vaginal discharge

Screening tests to recognise sepsis:
Non-obstetric and non-sepsis scores, such as qSOFA and SIRs, performed poorly as predictor of adverse outcomes in pregnant patients with sepsis. Monitoring of a woman with suspected sepsis should be performed using an early warning system modified for obstetrics, managed through a multidisciplinary approach with early escalation and senior input. OBSTETRIC-SPECIFIC early warning signs (EWS) coupled with clinical investigations can improve the recognition, assessment, and prompt treatment of women at risk of sepsis and severe maternal outcomes.

Components of Modified Maternal Early Warning Criteria (Positive if any 1 criterion is met (sustained for 20 min):
_Systolic BP (mm Hg) lower than 90 or higher than 160
_Diastolic BP (mm Hg) higher than 100
_Heart rate lower than 50 bpm or higher than 120 bpm
_Respiratory rate lower than 10/min or higher than 24/min
_O2 saturation on room air less than 95%
_Oliguria less than 35 mL/h32 h
_Temperature lower than 36°C or higher than 38°C
_WBC count higher than 15,000/mm3 or less than 4,000/mm3
_Maternal agitation, confusion, or unresponsiveness

Management
Immediate actions within an hour of recognition of sepsis:
1.Check Lactate level:
Serum lactate of 2 mmol/l or more should initiate immediate senior review, intravenous fluid administration and repeat lactate measurement thereafter to gauge response to treatment.
Serum lactate of 4 mmol/l or more should prompt immediate escalation of care,

2.Obtain Blood cultures before administering antibiotics and empirical treatment started without waiting for any microbiology results; but do not delay giving antibiotics
Two sets of blood cultures (each set including an aerobic and anerobic bottle) should be taken sequentially, within in minutes of each other.

3.Administer broad-spectrum antibiotics: Administration of intravenous broad-spectrum antibiotics is recommended within one hour in women at high risk of sepsis, with or without septic shock. Choice of empirical antibiotic therapy should be guided by local epidemiology.

4.Early fluid resuscitation of crystalloid should be administered with an immediate 500ml fluid bolus in women with hypotension or elevated lactate above 4mmol/L. This may need repeating. Urine output should be measured with precision using an hourly urometer.

5.Apply vasopressors if hypotensive during or after fluid resuscitation to maintain mean arterial pressure (MAP) ≥ 65 mm Hg

Investigations:
Imaging: Relevant studies like Pelvic ultrasound scan or computer tomography scan if pelvic abscess is suspected, or a chest x-ray for possible chest pathology.

Other microbiological samples taken should be guided by the clinical suspicion of focus of infection as appropriate: Swabs taken from throat, vagina, caesarean or other wounds, stool samples

Appropriate virological testing, including, influenza or SARS-Cov-2 testing if indicated. Women suspected of having influenza should be tested immediately using a viral nasal/throat swab for influenza PCR, barrier nursed and treated with antivirals while awaiting results.

Routine blood tests should include full blood count, coagulation screen, urea, electrolytes, creatinine, liver function tests (LFT), C-reactive protein (CRP), venous blood gas (or other near patient testing) for glucose and lactate.

Choice of Antimicrobials: For life threatening sepsis, where the sensitivity of the organism is unknown, a combination of either piperacillin/tazobactam or meropenem (for Gram negative cover) plus clindamycin (for Gram positive and anaerobic organisms) provides very broad cover, but local guidelines should be considered.

With any history of MRSA in a woman, the addition of vancomycin is advisable.

Empirical antimicrobials should be reviewed with culture results and targeted oral agents used as soon as clinically appropriate.

If a change in antibiotic regimen is considered because of deterioration despite first-line treatment, consider the addition of IV aciclovir 500mg 8 hourly as part of the second-line regimen.

Source control:
Remove the source of infection: Timely decision for Surgery to drain pus, Expedited birth where necessary, or remove the retained products of conception

Intravenous immunoglobulin G: should be considered as part of treatment for Gram positive necrotising infections and toxic shock when other measures are failing. Use should be limited to the sickest women, and administered in a critical care setting, with a blood warming device.

Recognise and treat further complications:
Necrotising fasciitis (NF) is serious abdominal or remote sequelae of wound infection. NF refers to infection causing necrosis of the superficial and/or deep fascia and subcutaneous tissue Should be suspected when very severe pain out of proportion to what can be seen, typically, requiring increasingly stronger analgesia.

Acute respiratory distress syndrome: A nonspecific response of the lung, characterized by diffuse inflammation, increased fluid level in the lung due to increased vascular permeability, and loss of aerated lung units. These lead to poor compliance, greatly increasing the breathing efforts, and profound hypoxemia. Pregnant women are at increased risk of developing ARDS and needing mechanical ventilation compared with nonpregnant women.

Mechanical ventilation is life-saving, but high concentrations of oxygen and the physical effects of positive pressure ventilation can damage the lungs. Low-tidal-volume ventilation, which aims to limit inflation pressures is preferable rather than trying to normalize arterial blood gases.

Thromboembolism: Systemic infection is an important risk factor for venous thromboembolism and should prompt reassessment for the correct thromboprophylaxis, if there is no coagulopathy or risk of haemorrhage

Indications for ICU care

Cardiovascular: Hypotension (< 90 mmHg systolic) or raised serum lactate (> 4 mmol/l) persisting despite fluid resuscitation, suggesting the need for vasopressor and/or inotrope support

Respiratory: Pulmonary oedema, Need for mechanical ventilation, or need for airway protection

Renal: Need for Renal replacement therapy

Neurological: Decreased conscious level

Miscellaneous: Multi-organ failure, Uncorrected acidosis, Hypothermia

Fetal concerns:

Decisions about fetal monitoring should be made based on the gestational age of the fetus, desires of the patient and her family, and feasibility of intervention based on maternal status. The care plan must be reevaluated regularly as the risks and benefits tend to change with the course of illness.

Neither necessary medications nor diagnostic imaging should be withheld from a pregnant woman because of fetal concerns related to decreased placental perfusion or increased risk of malformations.

Non-reassuring electronic fetal heart rate monitoring reflects uteroplacental perfusion, and may reflect worsening maternal end-organ function. Therefore, even in a situation in which delivery may not be possible, fetal heart rate monitoring can be useful to prompt reassessment of maternal BP, oxygenation, ventilation, acid-base balance, or cardiac output. However, if the patient is not stable for operative delivery, a clear plan must be made with the understanding that delivery is not safe regardless of deterioration in the fetal condition.

Transfer of patient to higher center for ICU care:
Pretransport evaluation of the woman and her fetus, as well as stabilization of maternal status, must be ensured before transport. Continuous monitoring is recommended during transit. Venous access must be established before transport, and all existing lines should be secured. Left uterine displacement should be routine during transport. If there is a high probability that intubation and mechanical ventilation will be needed during transport, it should be accomplished before departure.

Cardiac arrest:
Cardiac arrest in pregnancy is treated with the same ratio of chest compressions to breaths, respiratory support, drugs, and defibrillation as for any adult in cardiac arrest. It is important to achieve left uterine displacement during cardiopulmonary resuscitation in order to alleviate aortocaval compression. The American Heart Association recommends manual uterine displacement, rather than tilting the patient for more effective chest compressions.

Resuscitative Hysterotomy: If efforts to resuscitate a pregnant woman in cardiac arrest have been unsuccessful, resuscitative hysterotomy (eg, perimortem cesarean delivery) is recommended in women with a uterine size at or above the umbilicus (20 weeks of gestation or more). Resuscitative hysterotomy should be considered as soon as there is a maternal cardiac arrest and be performed if the return to spontaneous circulation does not occur within the first few minutes of maternal resuscitation. Once the decision is made to perform a resuscitative hysterotomy, there is no need to move a patient to an operating room or undertake extensive preparations. The only essential instrument in this setting is a scalpel.

Conclusions:
Sepsis remains a clinical condition without a diagnostic test. Treatment for sepsis is predicated on timely suspicion, fluid resuscitation, and antibiotic therapy within the first hour. Clinical judgment can always supersede scoring systems and published vital sign parameters when determining who requires ICU admission. The clinician should be aware that fever may be absent, cultures may be negative, and a source is not always identifiable.

07

The Obesity Challenge in OBGYN

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Obesity is becoming increasingly prevalent in India and its becoming a common risk factor in OBGYN practice also. In pregnancy obesity is linked with increased morbidity both in normal and cesarean deliveries, adverse perinatal outcomes, anesthetic challenges, post operative complications like wound gaping and VTE (Venous thromboembolism). With the rising trends in obesity, it has become utmost urgent for all the OBGYN practitioners to bring together a multidisciplinary team of general medicine physicians, neonatologists and anesthesiologists to deliver good care to this particular group of patients.

The article focuses on 3 critical aspects of obesity in OBGYN practice namely.
1)Anesthetic and Surgical Considerations in obesity
2)Thromboprophylaxis and Dose Adjustment
3)Fetal Programming Effects of Maternal Obesity

1. Anesthetic and Surgical Considerations in obesity:
WHO classified Obesity using the Body Mass Index (BMI), which provides a simple estimate of body fat based on height and weight (Table No 1). Although obesity is traditionally associated with increased perioperative morbidity and mortality, emerging evidence over the past decade has challenged this assumption, particularly for individuals with class I and class II obesity. Several studies have demonstrated that overweight and mildly obese patients may experience comparable or even lower postoperative complication and mortality rates compared to individuals with normal BMI, a phenomenon often referred to as the “obesity paradox.” In contrast, class III obesity is consistently associated with significantly increased postoperative morbidity and mortality. WHO has also classified central obesity as in (Table No. 2).

Beyond BMI, fat distribution plays a critical role in determining clinical risk. Central (abdominal) obesity has been shown to predict metabolic and perioperative risk more accurately than BMI alone. Central adiposity is strongly associated with metabolic syndrome, cardiovascular disease, increased difficulty in airway management and ventilation, and higher overall perioperative risk.

Many comorbidities associated with obesity contribute to increased perioperative risk. Sleep-disordered breathing, particularly obstructive sleep apnoea (OSA) and obesity hypoventilation syndrome (OHS), is linked with difficult airway management, unplanned tracheal reintubation, and postoperative cardiopulmonary complications.

The perioperative mortality risk score Obesity Surgery–Mortality Risk Score (OS-MRS) (Table 3) developed for bariatricsurgery can also be applied to non-bariatric patients. A score of 4–5 identifies high-risk patients who may require postoperative critical care admission.

For any women with a BMI ≥ 40 admitted to the labor ward duty anesthesia should be promptly notification early if operative delivery or intervention is anticipated. Operating theatre teams should be alerted for women weighing > 120 kg to ensure appropriate tables and transfer equipment are available before transfer.

It is very necessary for all maternity to have environmental risk assessment for women with a booking BMI ≥ 30 kg/m². This should include evaluation of circulation space, accessibility, doorway widths, safe working loads of floors and equipment (up to 250 kg), appropriate theatre gowns, staffing levels, transportation logistics, and the availability of OT-specific equipment such as large blood pressure cuffs, sit-on weighing scales, reinforced beds, operating tables, theatre trolleys, and lifting or lateral transfer devices.

For any women with a BMI ≥ 40 admitted to the labor ward duty anesthesia should be promptly notification early if operative delivery or intervention is anticipated. Operating theatre teams should be alerted for women weighing > 120 kg to ensure appropriate tables and transfer equipment are available before transfer.

Table 1. Classification of Obesity According to Body Mass Index (BMI):

Category	BMI (kg/m²)
Normal range	18.50–24.99
Overweight	≥25.00
Pre-obese	25.00–29.99
Obese Class I	30.00–34.99
Obese Class II	35.00–39.99
Obese Class III (Morbid obesity)	≥40.00

Table 2. Definition of Central Obesity (WHO Criteria):

Population	Waist Circumference (Men)	Waist Circumference (Women)
Western population	>102 cm	>88 cm
Asian population	>90 cm	>80 cm

Table 3. Obesity Surgery–Mortality Risk Score (OS-MRS)

Risk Factor	Points
BMI >50 kg/m²	1
Male sex	1
Hypertension	1
Age >45 years	1
Risk factors for PE (previous VTE, OSA/OHS, right heart failure, pulmonary hypertension, vena cava filter)	1

Risk interpretation:

- Score 0–1: Low risk
- Score 2–3: Moderate risk
- Score 4–5: High risk → consider postoperative critical care admission

Surgical and Wound Care Considerations:

Women with a BMI ≥30 undergoing caesarean section have an increased risk of wound infection. Prophylactic antibiotics should be administered. In addition, women with more than 2 cm of subcutaneous fat should have closure of the subcutaneous tissue space to reduce the risk of wound infection and wound separation. Meticulous hemostasis, layer closure, early mobilization is the key to prevent postoperative wound infections, seroma and dehiscence.

2. Thromboprophylaxis and Dose Adjustment:

Maternal obesity is a significant independent risk factor for venous thromboembolism (VTE) during both antenatal and postnatal periods. Women with a booking BMI ≥30 should undergo VTE risk assessment at the first antenatal visit and throughout pregnancy, with thromboprophylaxis . Pharmacological thromboprophylaxis with low molecular weight heparin should be weight-adjusted. (Table 4) The RCOG recommends higher prophylactic doses for women weighing over 90 kg, with further escalation for those above 130 kg and individualized dosing for weights exceeding 170 kg. Regardingpostnatal thromboprophylaxis all women with BME ≥ 40 should have thromboprophylaxis along with early mobilization.

Table 4. RCOG-Recommended Weight-Adjusted LMWH Dosing for Thromboprophylaxis:

Maternal Weight (kg)	Enoxaparin	Dalteparin	Tinzaparin
91–130	60 mg daily	7,500 units daily	7,000 units daily
131–170	80 mg daily	10,000 units daily	9,000 units daily
>170	0.6 mg/kg/day	75 units/kg/day	75 units/kg/day

3. Fetal Programming and Long-Term Consequences of Maternal Obesity:

Maternal obesity exerts profound effects on fetal development, influencing both immediate perinatal outcomes and long-term health in adulthood. The concept of "fetal programming," introduced by British epidemiologist David Barker, describes how adverse intrauterine environments predispose individuals to chronic diseases like ischemic heart disease, hypertension.

The mechanisms underlying fetal programming in obese pregnancies are primarily epigenetic, involving DNA methylation and chromatin remodeling within fetal progenitor cells. Aberrant methylation patterns have been identified in genes regulating insulin signaling, lipid metabolism, and appetite control in both placental and fetal tissues.

These epigenetic alterations are stable and persist through successive cell divisions, limiting the capacity for postnatal modification and creating a transgenerational, non-genetic propagation of metabolic disease. Historical examples such as the Dutch Hunger Winter of 1944–1945 demonstrate how early gestational exposure to adverse nutritional environments results in increased rates of adult obesity and metabolic disease decades later.

Thus, maternal obesity not only affects pregnancy outcomes but also establishes a biological foundation for chronic disease in the next generation, underscoring the importance of preconception counseling, weight optimization, and targeted antenatal interventions.

08 FGR: Precision in Timing Delivery

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Introduction: The Evolution of Growth Restriction

Fetal Growth Restriction (FGR) remains one of the most significant challenges in modern obstetrics, contributing to nearly 30% of stillbirths worldwide. In the past, our management was purely reactive—delivering babies simply because they were "small." Today, in 2025, we practice **Precision Obstetrics**. We recognize that a small fetus is not always a sick fetus, and a "normal-sized" fetus can still be growth-restricted.

The goal of this chapter is to provide a roadmap for the clinician to navigate the delicate balance between the risks of intrauterine hypoxia and the morbidities of iatrogenic prematurity. Using the ISUOG guidelines and landmark trial data like TRUFFLE, we move toward a protocol-driven approach that prioritizes fetal neurodevelopment and maternal safety.

1. Distinguishing SGA vs. FGR: The Diagnostic Divide

The Biological Potential vs. Statistical Percentiles

The first step in precision management is the correct classification.

- **Small for Gestational Age (SGA)**: This is a purely biometric description. A fetus is SGA if its Estimated Fetal Weight (EFW) or Abdominal Circumference (AC) is <10th percentile. Approximately 50–70% of SGA fetuses are "constitutionally small." They follow their growth curve, have normal liquor, and normal Doppler studies. These babies are healthy and do not require early intervention.

- **Fetal Growth Restriction (FGR)**: This is a functional and pathological diagnosis. It occurs when a fetus fails to reach its biological growth potential due to an underlying factor—most commonly placental insufficiency.

The ISUOG/Delphi Consensus Classification

To prevent confusion, we now categorize FGR based on the timing of presentation:

A. Early-Onset FGR (<32 Weeks)

- **Prevalence**: 1 in 100 pregnancies.
- **Challenge**: High prematurity risk.
- **Pathophysiology**: Severe placental insufficiency, often associated with Preeclampsia.
- **Doppler Pattern**: Typically follows a predictable sequence: UA PI increases $\rightarrow$ AEDV $\rightarrow$ REDV $\rightarrow$ DV changes.

B. Late-Onset FGR (≥32 Weeks)

- **Prevalence:** 1 in 20 pregnancies (much more common).
- **Challenge:** Difficult to detect. These fetuses are often >10th percentile but have "fallen off" their own growth curve.
- **Pathophysiology:** Mild placental insufficiency that becomes apparent only as fetal oxygen demands increase in the third trimester.
- **Doppler Pattern:** UA Doppler is often normal. The key marker is the Cerebroplacental Ratio (CPR) and MCA vasodilation.

2. Prevention and Optimization: Can we change the outcome?

One of the most common questions in clinical practice is: “Doctor, what can I eat or take to make the baby grow?” It is vital for practitioners to separate evidence from myth.

Low-Dose Aspirin (The ASPRE Trial)

The only proven intervention to prevent FGR (specifically early-onset) is the use of **Low-Dose Aspirin (150 mg)**.

- **Timing:** It must be started before 16 weeks of gestation.
- **Target**: Women identified as "High Risk" via first-trimester screening (combining maternal history, MAP, PlGF, and Uterine Artery Dopplers).
- **Efficacy**: It can reduce the incidence of early-onset FGR by up to 60%. Starting aspirin after 20 weeks has negligible benefit for placental remodelling.

Nutritional Myths and Realities

There is currently **no evidence** that maternal bed rest, "L-Arginine" granules, or protein powders can reverse true placental FGR. While adequate nutrition is essential for maternal health, FGR is a problem of **transport (the placenta)**, not a lack of **supply (the food)**. Over-feeding a mother with a failing placenta does not increase fetal weight; it only increases the risk of maternal gestational diabetes.

3. Doppler Indices: The Physiological Roadmap

Doppler indices are not just numbers; they are a window into the fetal compensatory response to hypoxia.

The Umbilical Artery (UA): The Placental Gatekeeper

The UA Doppler measures the resistance within the placenta. As the "functional" placental area decreases, resistance rises.

- **High PI (>95th centile)**: Indicates early placental damage.
- **Absent End-Diastolic Velocity (AEDV)**: Suggests that 60–70% of the placental vascular bed is non-functional.
- **Reversed End-Diastolic Velocity (REDV)**: A critical sign of extreme resistance, where blood flows back toward the fetal heart during diastole. This is a pre-terminal sign.

The Middle Cerebral Artery (MCA): The "Brain-Sparing" Effect

When oxygen supply is limited, the fetus redistributes blood flow to vital organs (brain, heart, adrenals). The MCA dilates to increase blood flow to the brain, leading to a **low Pulsatility Index**.

- **Clinical Pearl**: While brain-sparing is a protective mechanism, prolonged brain-sparing is associated with lower IQ scores and neurodevelopmental delays in childhood. It is a sign of "compensated" hypoxia.

The Cerebroplacental Ratio (CPR)

The CPR (MCA PI / UA PI) is the most sensitive marker for late-onset FGR. It captures the relationship between increased placental resistance and the fetal brain-sparing response. A CPR <1.0 (or <10th percentile for GA) is a strong predictor of:

- Emergent Cesarean for fetal distress.
- Low Apgar scores.
- Admission to the NICU.

The Ductus Venosus (DV): The Heart's Warning

The DV is the most important longitudinal marker in early-onset FGR. It reflects the pressure in the right atrium.

- **Increased PI**: Suggests the heart is starting to struggle.
- **Absent/Reversed a-wave**: This indicates that the fetal heart can no longer compensate. This is the "trigger" for delivery in the TRUFFLE protocol to avoid stillbirth and minimize brain damage.

4. The TRUFFLE Trial: Shifting the Paradigm

The **TRUFFLE Trial (Trial of Randomized Umbilical and Fetal Flow in Europe)** provided the highest level of evidence for managing the preterm growth-restricted fetus.

Key Findings:

1. Timing Delivery: In babies between 26 and 32 weeks, waiting for changes in the Ductus Venosus (late changes) resulted in better neurodevelopmental outcomes at age 2 compared to delivering based on UA Doppler or CTG changes alone.

2. The "Safety Net": While waiting for DV changes, practitioners must use **Computerized CTG (cCTG)** to monitor Short-Term Variability (STV). If the STV drops below 3ms, delivery is indicated regardless of Doppler.

5. Management Protocol and Precision Timing

The decision to deliver involves four stages of deterioration, as outlined by the Barcelona Fetal Medicine group and adapted by ISUOG.

Stage 1: Mild Placental Insufficiency (The "Monitor" Phase)

- **Findings:** UA PI >95th centile OR CPR <10th centile.
- **Action:** Weekly Doppler.
- **Delivery:** 37–38 weeks. Vaginal delivery is acceptable with continuous monitoring.

Stage 2: Severe Placental Insufficiency (The "Prepare" Phase)

- **Findings**: AEDV in Umbilical Artery.
- **Action**: Admit patient. Administer Corticosteroids. Doppler every 24–48 hours.
- **Delivery**: 32–34 weeks. Cesarean is usually preferred as these fetuses have poor reserve for labor.

Stage 3: High Risk of Acidosis (The "Act" Phase)

- **Findings**: REDV in Umbilical Artery OR MCA PI <5th centile.
- **Action**: Admit to high-dependency unit. Complete steroids. Start Magnesium Sulfate for neuroprotection.
- **Delivery:** 30–32 weeks Stage 4: Imminent Fetal Death (The "Emergency" Phase)
- **Findings**: Absent/Reversed DV a-wave OR cCTG STV <3ms.
- **Action**: Immediate delivery.
- **Delivery**: >26 weeks. Survival depends heavily on NICU capabilities.

6. Long-term Implications: The Barker Hypothesis

Precision in FGR is not just about the first week of life; it is about the next fifty years. According to the Barker Hypothesis, a fetus that is growth-restricted undergoes "epigenetic programming" to survive in a nutrient-poor environment.

When this child is born into a nutrient-rich world, they are at a significantly higher risk for:

- Metabolic Syndrome and Type 2 Diabetes.
- Hypertension and Coronary Artery Disease.
- Obesity in adolescence.

Clinical Recommendation: Parents of FGR babies should be counseled to avoid "catch-up growth" that is too rapid. Slow, steady weight gain is healthier for the FGR neonate than rapid fattening, which triggers metabolic dysfunction.

7. Case-Based Scenarios for the Practitioner

Case A: The 36-weeker with "Normal" Dopplers

A patient presents at 36 weeks with a fundal height lagging by 3 weeks. USG shows EFW at the 12th percentile (Not SGA). However, the UA PI is at the 90th percentile and the MCA PI is low, resulting in a CPR <1.0.

- **Verdict**: This is Late-Onset FGR. Despite the weight being "normal," the brain-sparing indicates placental stress.
- **Action**: Monitor closely and plan for induction at 37 weeks. Do not wait until 40 weeks.

Case B: The 28-weeker with AEDV

A patient at 28 weeks has a fetus at the 2nd percentile with AEDV in the Umbilical Artery. The DV a-wave is present and positive.

- **Verdict**: Early-onset FGR, currently compensated.
- **Action**: Administer steroids and MgSO4. Do not deliver immediately. Every day in utero at this gestation increases survival by 2%. Wait for DV changes or CTG deterioration as per TRUFFLE.

8. Conclusion: The Future of FGR Management

As we conclude 2025, our understanding of FGR has moved from "size-based" to "physiology-based." The integration of CPR into routine third-trimester screening will likely reduce the incidence of late-term stillbirths. For the early-onset cases, the TRUFFLE trial remains our gold standard for protecting the fetal brain. By applying these precision timings, we ensure that we are not just delivering babies, but delivering healthy, thriving children.

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09

Thromboembolism Safety in Pregnancy

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Thromboembolism in pregnancy—including **Deep Vein Thrombosis (DVT)** and **Pulmonary Embolism (PE)**—is a leading cause of maternal mortality and morbidity. The risk of venous thromboembolism (VTE) is 4 to 6 times higher during pregnancy and rises to many times higher during the postpartum period.

Pathophysiology: Virchow's Triad

Pregnancy naturally expresses all three components of Virchow's Triad, creating a highly thrombogenic environment:

- **Hypercoagulability**: Increased levels of procoagulant factors (VII, VIII, X, von Willebrand factor, and fibrinogen) and decreased activity of natural anticoagulants like Protein S.
- **Venous Stasis**: Caused by hormonally induced vasodilation and mechanical compression of the pelvic veins (iliac veins and inferior vena cava) by the growing uterus.
- **Endothelial Injury**: Often occurs during delivery (vaginal or cesarean), specifically affecting pelvic veins.

Clinical Features

Diagnosis is challenging because many common symptoms overlap with normal physiological changes of pregnancy.

- **Deep Vein Thrombosis (DVT)**: Typically presents as unilateral leg pain, swelling, and tenderness. In pregnancy, **80–90% of DVTs occur in the left leg** due to May-Thurner anatomy (right iliac artery compressing the left iliac vein).
- **Pulmonary Embolism (PE)**: Most common symptoms include sudden shortness of breath, pleuritic chest pain, cough, and tachycardia.

Diagnosis

- **Imaging:**
  - **DVT**: Compression duplex ultrasound is the first-line investigation.
  - **PE**: Choice of **V/Q scan** or **CT Pulmonary Angiography (CTPA)**. CTPA is preferred if the chest X-ray is abnormal. Radiation exposure to the fetus is considered safe in both.

- **Laboratory**: Routine **D-dimer testing is not recommended** for diagnosis as levels naturally increase throughout pregnancy, leading to low specificity.

Management and Treatment of DVT and PE occurring in pregnancy

- **Mainstay: Low-Molecular-Weight Heparin (LMWH)** is the preferred treatment as it does not cross the placenta or enter breast milk.
- **Duration:** Therapeutic anticoagulation is usually continued for the remainder of the pregnancy and for at least **6 weeks** postpartum, with a total minimum duration of **3 to 6 months.**

- **Avoided Anticoagulants: Warfarin** is generally avoided due to teratogenicity (especially in the first trimester), except in rare cases like mechanical heart valves.
- **DOACs** (Direct Oral Anticoagulants like apixaban or rivaroxaban) are currently contraindicated.
- **Emergencies:** Massive life-threatening PE may require intravenous **Unfractionated Heparin (UFH)**, thrombolysis, or surgical thrombectomy.
- **Inferior Vena Cava filter placement may be needed before delivery reinforcing the need for management of antenatal or peripartum DVT /PE in cardioobstetric centers with CTVS teams on standby.**

Mainstay in bringing down morbidity and mortality from DVT / PE entails risk stratification of all ante/post partum patients

RISKS MAY BE

Pre-existing: Previous VTE, heritable thrombophilias (e.g., Factor V Leiden), obesity (BMI >30), and advanced maternal age (>35) chronic.smokers.

Obstetric: Multiple pregnancy, preeclampsia, assisted reproduction (IVF), and cesarean delivery (especially emergency).

Major guidelines (ASH 2018, RCOG 2015, ACOG) use clinical history to determine the need for prophylaxis.

The High Risk (Antepartum & Postpartum Prophylaxis):

- Previous unprovoked or estrogen-associated VTE.
- Recurrent VTE (two or more prior events).
- High-risk thrombophilias: Antithrombin deficiency, homozygous Factor V Leiden, or compound
- heterozygosity (Factor V Leiden + Prothrombin mutation).
- Antiphospholipid Syndrome (APS) with prior VTE.
- Intermediate/Low Risk (Usually Postpartum Prophylaxis Only):
- Prior VTE provoked by a major non-hormonal risk factor (e.g., surgery).
- Asymptomatic thrombophilias

TREATMENT INVOLVES PREVENTIVE MEASURES AND PHARMACOLOGICAL AGENTS

IMPORTANCE OF THESE SEEMINGLY SIMPLE POINTS IS PARAMOUNT

PATIENT AWARENESS AND EDUCATION ABOUT MOBILTY

SPECIFIC LOWER LIMB MOVEMENTS TO BE PERFORMED DAILY FOR CALF PUMPING DAILY

USE OF COMPRESSION STOCKINGS
EDUCATION OF OBSTETRIC UNIT MEMBERS FOR AMBULATION POST OP AND ELECTRICAL COMPRESSION OF CALVES WHEN PATIENTS ARE UNDER ANESTHESIA NOT FULLY MOBILE

ROUTINE USE OF SIMPLE CLINICAL SIGNS LIKE HOMANS IN ANTENATAL, POST ,PERIPARTUM PERIOD

PHARMACOLOGICAL AGENTS
LMWH is the safest in pregnancy,low fetal passage, lower risk of osteoporosis and heparin induced thrombopenia, easily available ,long half life thus once a day dosing.Safe in post partum.

UFH : RESERVED for those with renal impairment or those near delivery where rapid reversal may be needed

FONDAPARINUX: used if there is severe allergy to to LMWH BUT AVOIDED IN FIRST TRIMESTER

WARFARIN is teratogenic esp between 6 to 12 weeks , used rearely eg in prosthetic heart valves
DOAC avoided due to lack of safety data and placental transfer.

WHEN TO START THE ANTICOAGULANT ?
LMWH is usable in first trimester for patients identified as above , can be used as soon as situation develops in pregnancy waranting its use though some may prefr unfractionated Heparin to get rapid onset of action then switch over to LMWH.

Monitoring done with your APTTK level serially

MANAGEMENT AT DELIVERY

To allow for safe neuraxial anesthesia (epidural/spinal), timing of the last dose is critical:

Prophylactic Dose: Hold LMWH for at least 12 hours before anesthesia.

Therapeutic Dose: Hold LMWH for at least 24 hours before anesthesia.

Postpartum Resumption: Typically restarted 6–12 hours after vaginal delivery or 12–24 hours after cesarean section, provided there is no active bleeding.

POSTPARTUM DURATION

Postpartum prophylaxis is generally continued for 6 weeks for those at high risk (prior VTE or high-risk thrombophilia) or 10 days for those with lower-level transient risk factors like cesarean delivery with additional risk markers.

TO SUMMARIZE : WE NEED TO IDENTIFY RISKS ,ADOPT PREVENTIVE MEASURES AND DRUGS WHERE INDICATED FOR PREVENTION OF THROMBOEMBOLISM IN PREGNANCY .

ALL OBSTETRIC UNITS SHOULD BE TRAINED TO SUSPECT DVT AND PE AND ACT SWIFTLY TO BRING DOWN MORBIDITY AND MORTALITY ASSOCIATED WITH THIS .

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10 Preterm Labor and Neuroprotection

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INTRODUCTION

Preterm birth (PTB), defined as delivery that occurs between 20 and 37 weeks of gestation, is a major obstetric and global health concern. The American College of Obstetricians and Gynecologists (ACOG) states that Preterm labor(PTL) can be diagnosed in patients with regular uterine contractions and cervical dilation of at least 2 cm before 37 weeks of gestation, while the international guidelines from the World Association of Perinatal Medicine and the Perinatal Medicine Foundation (WAPM-PMF) recommend using a cutoff of at least 3 cm to diagnose spontaneous preterm labor (sPTL).

The worldwide incidence of PTB is approximately 10%. Preterm birth may be spontaneous (following PTL, PPROM, or cervical insufficiency) or it may be indicated by a specific maternal or fetal complication. Spontaneous PTL with intact membranes is responsible for 40% to 45% of all PTBs.

Preterm birth is the leading cause of death in children under 5 years, and an estimated 35% of neonatal deaths in the first 28 days of life are caused by preterm birth complications. Preterm newborns are at increased risk of short-term morbidities, including respiratory distress syndrome, intraventricular haemorrhage, necrotizing enterocolitis and sepsis, as well as long term morbidities, such as chronic lung disease and neurological disabilities. For this reason, accurate diagnosis and management of sPTL is imperative for improving neonatal outcomes.

CERVICAL ASSESSMENT FOR PREDICTING PRETERM BIRTH

A short cervical length as measured with endovaginal ultrasonography has been associated with increased risk of preterm birth. Assessment of cervical length in the second trimester has been shown to identify women at increased risk of PTB. A short cervical length is defined as less than 25mm, typically measured between 16 and 24 weeks of gestation.

- **In patients with a singleton pregnancy with no prior spontaneous preterm birth** - cervix should be visualized as part of the 18 0/7 -22 6/7 weeks of gestation anatomy assessment. If on transabdominal ultrasonography a short cervix is found or suspected , endovaginal ultrasonography is recommended to more accurately assess cervical length. Screening of cervical length with serial endovaginal ultrasonography is not indicated.

- **In patients with a singleton pregnancy and a prior spontaneous preterm birth**- serial (every 1-4weeks) endovaginal ultrasound measurement of cervical length beginning at 16 0/7 weeks of gestation and repeated until 24 0/7 weeks of gestation is recommended.

- **In patients with multiple gestation**- as with singleton pregnancy cervix should be visualized as part of the 18 0/7 -22 6/7 weeks of gestation anatomy assessment. If on transabdominal ultrasonography a short cervix is found or suspected , endovaginal ultrasonography is recommended to more accurately assess cervical length.

PROGESTERONE OR CERVICAL CERCLAGE TO PREVENT PRETERM BIRTH

Endogenous progesterone is essential for the maintenance of pregnancy, and local decline in progesterone activity is thought to have a role in labor induction. Therefore, progestogens have been increasingly used in women at high risk of preterm birth as they are believed to counter this functional decline and provide anti- inflammatory effects. Several randomized controlled trials (RCTs) and meta- analyses have been undertaken to help provide an evidence- based approach to prevent preterm birth and determine the optimal regimes and populations to target.

- **Patients without a history of preterm birth**- Most guidelines recommend progesterone treatment for cervical shortening, for patients without a history of preterm birth. Most studies used 200mg progesterone daily by vaginal route from the time of identification of a short cervix less than 25mm at 18 0/7 – 25 6/7 weeks of gestation until 36-37 weeks of gestation. Intramuscular 17 -alpha hydroxy progesterone caproate is not recommended. Cervical cerclage is of uncertain effectiveness in patients with a short cervix and no history of preterm birth. However there is evidence of potential benefit in patients with a very short cervical length(<10mm).
- **Patients with a singleton pregnancy and a prior spontaneous preterm birth** -ACOG practice advisory on Updated Clinical Guidance for the Use of Progestogen Supplementation for the Prevention of Recurrent Preterm Birth does not recommend vaginal progesterone or Intramuscular 17 -alpha hydroxy progesterone caproate (insufficient data) for the primary prevention of preterm birth. FIGO recommends daily vaginal progesterone or weekly 17- OHPC treatment to prevent preterm birth.
- **Patients with a singleton gestation , a prior spontaneous preterm birth, and a short cervix in the second trimester** consider cerclage versus vaginal progesterone if not already on progesterone supplementation(ACOG). A cerclage has been found to be beneficial when inserted at gestations less than 24 weeks. Patients who are on progesterone supplementation should be informed of their increased risk of preterm birth, and cerclage may be offered in addition to continuation of progesterone
- **Multiple gestation** - . Routine prophylactic use of vaginal progesterone or Intramuscular 17 -alpha hydroxy progesterone caproate is not recommended for prevention of preterm birth based solely on the indication of multiple gestation.. Cervical cerclage is not recommended for prevention of preterm birth based solely on the indication of multiple gestation.

- **Physical examination indicated cerclage -**All guidelines recommend a physical examination indicated cerclage for cervical opening under the condition that the cervix is not dilated more than 4 cm, the patient has no contractions and there is no sign of infection.
- **A history- indicated cerclage** should be offered in women who have had three or more preterm deliveries and/or mid- trimester losses.

CORTICOSTEROID USE IN PRETERM LABOR

- Corticosteroid use in women with established preterm labor reduces the risks of respiratory distress syndrome, intraventricular hemorrhage, neonatal necrotizing enterocolitis, NICU admissions, systemic infections in the first 48 hours of life, and neonatal death.
- Many guidelines recommend that antenatal corticosteroids are offered to all women who are in established preterm labor between 24+0 and 33+6 weeks. The Antenatal Late Preterm Steroids (ALPS) trial, a systematic review and meta- analysis of six trials (including the ALPS trial) demonstrated benefits of steroids in reducing respiratory distress syndrome in late preterm infants (34+0 to 36+6 weeks). Based on this evidence, antenatal corticosteroids should also be considered for women between 34+0 and 36+6 weeks who are in confirmed preterm labor. Currently, both NICE and RCOG recommend that antenatal corticosteroids should be considered in women between 22 and 35+6 weeks. Start corticosteroids even with risk of incomplete course completion, it may be of benefit in reducing respiratory distress syndrome for women who are in established preterm labor.
- Antenatal corticosteroids repeat after 7 days with proven preterm labor if preterm birth does not occur and there is a high risk of preterm birth in the next 7 days, because the optimal benefits of antenatal corticosteroids last for 7 days. More than two courses is not recommended owing to the deleterious effects of multiple corticosteroid doses on fetal growth and neurodevelopment.
- Either two doses of 12 mg intramuscular betamethasone are given 24 hours apart or four doses of 6 mg intramuscular dexamethasone are given 12 hours apart.

TOCOLYSIS

- Tocolytics are generally recommended for patients with threatened PTL between viability and 34 weeks EGA.
- Tocolytic agents decrease the strength and frequency of uterine contractions, which may briefly prolong pregnancy. However, they do not treat the underlying causes of sPTL, have not been shown to delay birth until term, have not been shown to improve neonatal outcomes on their own, and are associated with a range of mild to severe adverse events. For these reasons, **their use should be limited to a 48-hour course of treatment** and reserved for patients who would benefit from a 48-hour delay in birth, such as patients who need the time for a course of corticosteroids to achieve maximal effect. A limited course of tocolytics may also be appropriate during patient transfers and in the case of self-limited or treatable conditions known to cause PTL, such as a urinary tract infection or intraabdominal procedures.

- The use of tocolytics in women with PPROM is controversial. WHO, RCOG, and ACOG do not recommend tocolysis for women with PPROM.
- **Contraindications to tocolysis** include preeclampsia with severe features, intraamniotic infection, antepartum hemorrhage, intrauterine fetal demise, lethal fetal anomaly, and significant maternal cardiac disease.
- The drug classes with the best evidence to support their use for tocolysis include calcium channel blockers (nifedipine), cyclooxygenase (COX) inhibitors (indomethacin), and beta-agonists (terbutaline). Other tocolytic agents include oxytocin receptor antagonists (eg, atosiban), magnesium sulfate, and nitric oxide donors (eg, nitroglycerin). Combinations of tocolytics are generally not recommended.
- When tocolysis is indicated, nifedipine and atosiban (if nifedipine is contraindicated) are recommended as first- line tocolytic agents. Betamimetics are not recommended for tocolysis owing to their frequent cardiovascular adverse effects, which could be life threatening.

Calcium channel blockers: Nifedipine has a better safety profile than other tocolytic medications and is often used for tocolysis, especially after 30 to 32 weeks of EGA. Nifedipine is ACOG's recommended first-line agent for patients between 32 and 33 6/7 weeks and is the agent that the WHO and NICE recommend for tocolysis from 24 to 33 6/7 weeks EGA. The commonly used regimen for nifedipine (immediate release) is an initial oral dose of 20 mg followed by 10 mg every 6 hours for 3–7 days or until transfer is completed, whichever comes first. CCBs do not have significant known adverse effects on fetal or newborn babies. As a peripheral vasodilator, CCB therapy is associated with dizziness, palpitations, tachycardia, nausea, and flushing. This therapy is contraindicated in patients with hypotension and preload-dependent cardiac conditions.

Cyclooxygenase inhibitors: Many experts prefer indomethacin for tocolysis before 32 weeks EGA based on the results of a large meta-analysis of 58 randomized clinical trials that showed it to be the superior first-line tocolytic agent due to its efficacy and tolerability. They are contraindicated, however, in the third trimester due to an association with increased risk of premature closure of the ductus arteriosus and the potential for renal dysfunction leading to oligohydramnios.

Beta-2 receptor agonists: These agents lead to smooth muscle relaxation, reducing myometrial contractions. However, myometrial cells become desensitized to these drugs, limiting their effectiveness over time. Their use is associated with a risk of serious maternal adverse effects, which may sometimes be life-threatening. Although still used in some circumstances, beta-2 agonists are generally considered second-line tocolytics behind COX inhibitors and CCBs due to relatively lower efficacy and safety. These drugs are relatively contraindicated in patients with significant tachycardia, cardiac disease sensitive to tachycardia, and poorly controlled diabetes mellitus.

Oxytocin receptor antagonists. Atosiban is the most frequently used drug in this class. Atosiban has relatively few maternal adverse effects beyond injection site reactions, and there are no absolute contraindications to its use beyond allergy to the medication.

Magnesium sulfate: Magnesium sulfate appears to be as effective as other tocolytic agents at delaying sPTB. However, it is generally considered a second-line tocolytic due to its higher potential for serious adverse maternal events. It is often given to patients in early sPTL for neuroprotection and can simultaneously function as the primary tocolytic. Despite generally recommending against combination tocolytic therapy, ACOG notes that a second tocolytic agent can be given to patients with persistent PTL despite being on magnesium sulfate for neuroprotection. In these circumstances, indomethacin is often preferred. CCBs and beta-2 agonists should not be used concurrently with magnesium sulfate, as they can work synergistically, leading to profound smooth muscle relaxation and serious respiratory depression.

Nitric oxide donors: Nitric oxide is a potent smooth muscle relaxer, and the nitric oxide donor, nitroglycerin, has been studied for tocolysis. A 2014 Cochrane review of randomized clinical trials comparing nitroglycerin to placebo, nifedipine, or beta-2 agonists concluded that there is "insufficient evidence to support the routine administration of nitric oxide donors in the treatment of threatened preterm labor."

MAGNESIUM SULFATE FOR NEUROPROTECTION

- Magnesium sulfate provides neuroprotection for the neonate when given within 24 hours of early PTB.
- Results from a 2024 Cochrane review confirmed that predelivery magnesium sulfate reduces the risk of cerebral palsy and probably reduces rates of severe IVH when given to patients at imminent risk of PTB before 34 weeks EGA.
- Similar to recommendations regarding antenatal corticosteroids, recommendations regarding when to offer magnesium sulfate for neuroprotection vary slightly across the globe. The WHO and ACOG strongly recommends magnesium sulfate for women at risk of imminent PTB before 32 weeks. NICE guidelines recommend offering magnesium sulfate between 24 and 29 6/7 weeks EGA and considering it between 30 and 33 6/7 weeks EGA.
- Dose- Most guidelines recommend an intravenous loading dose of 4 g administered slowly over 20– 30 minutes, followed by a maintenance infusion of 1 g/hour continued until delivery or up to a maximum duration of 24 hours if delivery does not occur within 24 hours. There is not enough evidence to support a recommendation for a repeat course of magnesium sulfate for fetal neuroprotection after an initial course has been administered.
- Where MgSO4 is administered, monitor women for clinical signs of magnesium toxicity at least every 4 h by recording pulse, blood pressure, respiratory rate, and deep tendon (for example, patellar) reflexes. With appropriate monitoring and care in high-resource settings, the 2024 Cochrane review found that magnesium sulfate did not increase rates of maternal death or cardiac or respiratory arrest when compared with placebo.
- Contraindications-Magnesium sulfate is contraindicated in patients with myasthenia gravis and should be used with caution in patients with neuromuscular disease and heart block.

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11 Management of Monochorionic Twin Pregnancy

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Abstract

Monochorionic twin pregnancies account for approximately 20% of all twin gestations and are associated with significantly higher perinatal morbidity and mortality compared with dichorionic twins. The shared placental circulation predisposes these pregnancies to unique complications, including twin-to-twin transfusion syndrome (TTTS), selective fetal growth restriction (sFGR), twin anemia–polycythemia sequence (TAPS), and acute intertwin transfusion events. Optimal management relies on early determination of chorionicity, intensive ultrasound surveillance, timely diagnosis of complications, and evidence-based interventions such as fetoscopic laser ablation. This review summarizes current best practices for the antenatal monitoring, diagnosis, management of complications, and delivery planning in monochorionic twin pregnancies, drawing on international guidelines and key clinical trials.

Introduction

The incidence of twin pregnancies has increased globally over recent decades, largely driven by assisted reproductive technologies and advanced maternal age. Among twin gestations, chorionicity is the most important determinant of perinatal outcome. Monochorionic twins arise from division of a single fertilized ovum and share a single placenta, resulting in placental vascular anastomoses that connect the fetal circulations.

Monochorionic diamniotic (MCDA) twins represent the most common form of monochorionic gestation and are associated with a perinatal mortality rate two to three times higher than that of dichorionic twins. Monochorionic monoamniotic (MCMA) twins carry even higher risks but are less common. This review focuses primarily on MCDA pregnancies, which account for the majority of clinical management challenges.

Determination of Chorionicity and Early Pregnancy Assessment

Importance of Early Diagnosis

Accurate determination of chorionicity is essential and should be performed as early as possible, ideally in the first trimester (11–14 weeks' gestation). Chorionicity dictates the intensity of antenatal surveillance, counseling regarding risks, and timing of intervention.

Ultrasound Features

Key sonographic features include:

- **Lambda (twin peak) sign,** suggestive of dichorionicity
- **T-sign,** indicating monochorionicity
- Number of placentas and membrane thickness

International Society of Ultrasound in Obstetrics and Gynecology (ISUOG) and Royal college of obst n gynae (RCOG) guidelines emphasize that chorionicity assignment is most accurate in the first trimester and should be clearly documented in the medical record.

2. Surveillance Protocols in Monochorionic Twin Pregnancy

First-Trimester Assessment

- **Chorionicity determination is crucial** at 11–14 weeks:
  - T-sign → monochorionic
  - Lambda sign → dichorionic

Accurate early identification allows **tailored surveillance** and counseling

Routine Surveillance

- **Frequency**: Every **2 weeks from 16 weeks** until delivery.
- **Assessment includes**:
  - Amniotic fluid (deepest vertical pocket)
  - Fetal growth parameters (EFW, head/abdomen ratios)
  - Umbilical artery Doppler
  - Bladder filling in both twins

- **Additional tools:**
  - Middle cerebral artery (MCA) Doppler: Detect TAPS(twin anemia polycythemia sequence)or anemia.
  - Fetal echocardiography: Recommended in TTTS(twin twin transfusion syndrome)for recipient twin cardiac function.

Special Considerations

- **Selective fetal growth restriction (sFGR)**: Defined by EFW < 10th percentile in one twin or ≥25% intertwin discordance.
- **TAPS monitoring**: Small-caliber vascular connections → chronic slow transfusion. Diagnosed by abnormal MCA PSV.
- **Single fetal demise**: Surviving twin at risk of cerebral injury; intensive surveillance and delivery planning required.

Delivery Planning Considerations

- Surveillance intensity increases after **26 weeks**, when complications like TTTS or sFGR may progress rapidly.
- Management ideally in **tertiary care or fetal medicine centers.**

1. Complications of Monochorionicity: Twin-to-Twin Transfusion Syndrome (TTTS)

Epidemiology and Risk

Monochorionic twins have a **shared placenta**, leading to vascular anastomoses that connect the two fetal circulations. TTTS occurs in **10–15% of MCDA pregnancies**, typically between **16–26 weeks**. Without intervention, **perinatal mortality is 50–90%,** and survivors have increased risk of **neurological morbidity**.

Pathophysiology

- **Donor twin**: chronic hypovolemia → oliguria → oligohydramnios → growth restriction.
- **Recipient twin**: chronic hypervolemia → polyuria → polyhydramnios → cardiac overload → risk of hydrops fetalis.
- **Placental anastomoses**:
  - Arteriovenous (AV) → main contributor to TTTS
  - Arterio-arterial (AA) → may be protective, sometimes prevents progression
  - Veno-venous (VV) → rarely significant

Diagnosis

- **Ultrasound criteria:**
  - Donor DVP < 2 cm
  - Recipient DVP > 8 cm (<20 weeks) or >10 cm (>20 weeks)
- **Quintero staging:**
  a. Stage I: Oligohydramnios/polyhydramnios present; donor bladder visible
  b. Stage II: Donor bladder not visible; polyhydramnios persists
  c. Stage III: Abnormal Doppler in either twin
  d. Stage IV: Hydrops fetalis
  e. Stage V: Demise of one or both twins

Management

- **Stage I**: Expectant management with close surveillance; 30–40% may progress.
- **Stage II–IV**: Fetoscopic laser photocoagulation of placental anastomoses is **first-line therapy**.
  - AJOG studies show dual survival rates of 60–70% post-laser, with improved long-term neurodevelopment.
- **Amnioreduction**: Reduces polyhydramnios-related maternal symptoms; temporizing measure if laser unavailable.

Selective reduction or early delivery: Considered in severe, pre-viable cases

Selective Fetal Growth Restriction (sFGR)

Definition and Classification

sFGR occurs when one twin demonstrates significant growth restriction due to unequal placental sharing. Diagnostic criteria include:

- Estimated fetal weight <10th percentile in one twin
- Intertwin weight discordance ≥25%

sFGR is classified into three types based on umbilical artery Doppler patterns:

- **Type I:** positive end-diastolic flow (best prognosis)
- **Type II:** persistently absent/reversed end-diastolic flow
- **Type III:** intermittent absent/reversed flow

Management

- **Type I**: expectant management with close surveillance
- **Type II and III**: individualized management, including consideration of fetoscopic laser ablation or early delivery depending on gestational age and fetal condition

Twin Anemia–Polycythemia Sequence (TAPS)

TAPS is caused by small-caliber placental anastomoses leading to chronic slow transfusion without amniotic fluid discordance. Diagnosis relies on MCA Doppler velocimetry demonstrating:

- Anemia in one twin
- Polycythemia in the co-twin

Management options include:

- Expectant management
- Fetoscopic laser ablation
- Intrauterine transfusion
- Early delivery, depending on gestational age and severity

Other Complications

Single Fetal Demise

Single intrauterine fetal death in monochorionic twins carries a substantial risk of neurological injury or death in the surviving twin due to acute intertwin transfusion. Management includes:

- Immediate assessment of the surviving twin
- Neuroimaging
- Expectant management if stable, with delivery timing individualized

Preterm Birth

Monochorionic twins have a high risk of spontaneous and iatrogenic preterm birth. Antenatal corticosteroids should be administered when preterm delivery is anticipated.

3. Timing and Mode of Twin Delivery

Timing of Delivery

- **Uncomplicated MCDA twins:** Deliver at **36–37 weeks.**
- **Complicated pregnancies:** Earlier delivery may be indicated for:
  - TTTS progression
  - sFGR with abnormal Dopplers
  - TAPS
  - Maternal or fetal compromise
- Aim**: Balance risk of prematurity vs. intrauterine demise.**

Mode of Delivery

- **Cephalic first twin ≥32 weeks:**
  - Vaginal delivery is safe (Twin Birth Study, NEJM)
- **Non-cephalic first twin:**
  - Cesarean section recommended.

Management of Second Twin

- **Cephalic presentation:** Vaginal delivery straightforward.
- **Non-cephalic presentation:**
  - **Internal podalic version + breech extraction** if skilled operator
  - Cesarean delivery if fetal compromise, non-cephalic first twin, or lack of expertise

- **Monitoring:**
  - Continuous fetal heart rate monitoring for the second twin after first twin delivery
  - Minimize inter-twin interval (<30 min) to reduce perinatal risk

Cesarean Indications Summary

- First twin non-cephalic
- Extreme prematurity (<32 weeks) with non-vertex twin
- Fetal compromise in either twin
- Operator inexperience with vaginal twin delivery

Key Takeaways

- Monochorionic twins are high-risk; TTTS is the most critical complication.
- Intensive 2-weekly ultrasound surveillance from 16 weeks is standard.
- Fetoscopic laser ablation is preferred for Stage II–IV TTTS.
- Delivery planning should consider fetal presentation, gestational age, and operator expertise.
- Vaginal delivery is safe for cephalic first twins ≥32 weeks, but careful management of the second twin is essential

Long-Term Outcomes and Counseling

Parents should be counseled regarding:

- Increased risk of perinatal mortality and morbidity
- Potential neurodevelopmental sequelae, particularly following TTTS or single fetal demise
- Importance of adherence to surveillance schedules and delivery planning in specialized centers

Conclusion

Management of monochorionic twin pregnancy requires meticulous antenatal surveillance, early detection of complications, and timely intervention guided by evidence-based protocols. Advances in fetal therapy, particularly fetoscopic laser ablation, have significantly improved outcomes for conditions such as TTTS. Multidisciplinary care in specialized centers, combined with individualized delivery planning, remains essential to optimizing maternal and neonatal outcomes in these high-risk pregnancies.

References (as requested)

- ISUOG Practice Guidelines: Role of ultrasound in twin pregnancy
- ACOG Practice Bulletin No. 231: Multifetal Gestations
- RCOG Green-top Guideline No. 51: Management of Monochorionic Twin Pregnancy
- Twin Birth Study, New England Journal of Medicine
- AJOG: Fetoscopic laser therapy for TTTS
- Fetal Diagnosis and Therapy

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12 Peripartum Mental Health Crises

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Introduction

Peripartum mental health crises encompass the **acute onset or exacerbation of psychiatric symptoms** during pregnancy and up to **12 months postpartum** that pose **immediate risk** to maternal and/or infant safety or result in **severe functional impairment**. These include severe mood disorders (major depression with suicidality), acute psychosis (including postpartum psychosis), bipolar episodes, severe anxiety, and other psychiatric emergencies precipitated or unmasked by the peripartum period [1,2].

Timely identification and management are essential due to the **high maternal morbidity and mortality** associated with untreated peripartum psychiatric crises, particularly with suicidal and infanticidal ideation [2].

Epidemiology and Risk Profile

Peripartum mood and anxiety disorders are common; depression affects up to one in seven women, and psychotic disorders — while less frequent — represent **obstetric emergencies** due to rapid decompensation and risk of harm [1,3].

Risk factors include:

- Personal history of mood disorders (especially bipolar disorder)
- Prior peripartum psychiatric episodes
- Abrupt discontinuation of psychotropic medications
- Severe **sleep deprivation** and psychosocial stressors
- Limited support systems or history of trauma
- These factors heighten the likelihood of crisis presentations including **postpartum psychosis** and **suicidality** [1,3].

Clinical Spectrum

1. Severe Depression with Suicidality

Presents with profound low mood, loss of interest, hopelessness, and suicidal thoughts or intent. Immediate risk assessment and urgent psychiatric intervention are required [1,3].

2.Acute Psychosis / Postpartum Psychosis

Characterised by **delusions, hallucinations, confusion, and disorganised thought** with rapid onset in the early postpartum period. This is a psychiatric emergency requiring hospitalisation and collaborative multidisciplinary care [1,4].

3. Manic or Mixed Episodes

In women with bipolar disorder, the peripartum period can precipitate **mania or mixed affective states**, frequently leading to erratic behaviour and loss of insight, which increases risk for harm [1].

4. Severe Anxiety and Trauma-Related Crises

Excessive panic attacks, phobic avoidance, and intrusive trauma symptoms can significantly impair functioning and warrant urgent intervention.

Screening and Identification

Routine screening for peripartum mental health conditions should occur at:

- Initial prenatal visit
- Later pregnancy
- Postnatal visits
- using validated instruments (e.g., EPDS, PHQ-9, GAD-7) as part of a **structured clinical evaluation** [1].

When screening reveals **self-harm or suicidal ideation**, clinicians must **immediately assess acuity and risk** and initiate **risk-tailored management without delay** [1].

Immediate Clinical Actions

- **Emergency evaluation**: Any woman presenting with psychosis, severe depression with suicidal ideation, or inability to care for self/infant must be treated as a **psychiatric emergency** [1].
- **Multidisciplinary involvement**: Immediate collaboration with perinatal psychiatry, neonatology, and social work safeguards mother and infant.
- **Safety planning**: Ensure continuous supervision if safety concerns exist; restrict unsupervised contact with infants when necessary.
- **Inpatient care**: Most acute presentations necessitate **urgent hospitalisation**, preferably in a **mother–baby unit (MBU)** when available.

Diagnostic Considerations

Differentiating peripartum mental health crises from:

- **Medical mimics** (thyroid dysfunction, infection, metabolic derangements)
- **Medication** or **substance-induced states**
- **Delirium** with fluctuating consciousness

This requires thorough clinical and laboratory assessment to identify or exclude organic contributors.

Management Principles

Management is led by psychiatric specialists but requires obstetric integration:

- **Psychopharmacotherapy**: Antidepressants, mood stabilisers, and antipsychotics may be indicated depending on severity, with careful risk–benefit considerations in pregnancy and lactation [2,5].
- Cognitive and behavioural therapies for moderate depression and anxiety states where appropriate.
- **Electroconvulsive therapy (ECT)**: Recommended in life-threatening, treatment-resistant, or severe peripartum depression or psychosis scenarios per guideline-based recommendations [3,5].

All treatment decisions should include **shared decision-making** and consideration of maternal comorbidity, obstetric context, and breastfeeding status.

Follow-Up and Long-Term Care

- Structured follow-up for recovery monitoring
- Risk assessment for future pregnancies
- Tailored pre-conception counselling for women with prior crises or severe peripartum psychiatric history

Integration of community mental health resources and ongoing psychosocial support improves outcomes and reduces recurrence.

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