Volume 26 I December 2025 I Monthly Issue 8

# AOGD BULLETIN

## "Women's wellness-From tiny heartbeats to timeless strength"


## THEME: BRIDGING THE GAP BETWEEN MATERNAL AND FETAL CARE

**AOGD SECRETARIAT**
Department of Obstetrics and Gynaecology
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# From the Secretarial Desk

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Greetings from AOGD Secretariat!

The winter cold is setting in with forecast of frosty morning and promises of hot steaming cups of coffee. Celebrations are round the corner – "Wishing you all a Merry Christmas and Happy New year"

There were a slew of activities in November beginning with a Cervical Cancer awareness camp on 4th November by Urogynaecology subcommittee and Webinar on Ultrasound in endometriosis on 17th November by Endometriosis subcommittee.

The AOGD secretariat at Lady Hardinge Medical College hosted a very successful CME on GDM and a very interactive FOGSI -JOGI PICSEP workshop on consecutive Saturdays i.e. 15th and 22nd November. The scientific content of both programs were outstanding with a rich discourse of ideas and invaluable exchange of knowledge.

A Public Awareness program on Vasectomy was organized by Lady Hardinge Medical College on 26th November. It was an interactive event with audience quiz which witnessed enthusiastic participation by male members who answered well and were awarded many prizes .

The 68th All India Congress of Obstetricians and Gynaecologists (AICOG 2026) is approaching. It is scheduled from 14th to 18th January 2026 at the Yashobhoomi Convention Centre, Dwarka, New Delhi. All are cordially invited to attend.

The December Bulletin is dedicated to Obstetric Medicine and Fetal Medicine . It explores on important fetal complications which is a must know for all . I congratulate Dr Manisha and her team for covering such useful topics which will certainly benefit the readers.

**AOGD Secretariat**

# President's Message

Greeting from AOGD,

December is usually a pleasant month, but because of pollution it is not so pleasant and cool. In November a number of Academic activities were conducted under AOGD, We at Lady Hardinge organized CME on hyperglycemia in pregnancy and conducted a workshop on research methodology in addition to other activities by various subcommittees

The last clinical meeting was by MAMC. Three very interesting cases were discussed. Thin attendance is an issue in these clinical meetings. I urge members to attend clinical meetings.

Theme for December bulletin is "Obstetric medicine and fetal Medicine at crossroads" Dr Manisha and her team has put efforts in bringing out this issue with very informative topics.

We pay homage to very senior members of our fraternity Dr S.N. Mukherjee and Dr Sarla Mukherjee who have left for their heavenly abode.

President AOGD

# From the Editor's Desk

**Dr Pikee Saxena**

**Dr Manisha Kumar**

**Dr Vidhi Chaudhary**

**Dr Shilpi Nain**

**Dr Apoorva Kulshreshtha**

**Dr Divya Gaur**
*Co-editor*

Dear readers,

As the year 2025 draws to a close, we are pleased to present our final issue for the year 2025, centered on the theme: "Bridging the Gap Between Maternal and Fetal Care."

Obstetricians are often the first to encounter patients following a fetal soft marker report. This issue addresses the critical need for informed guidance on fetal echocardiography and the nuances of interpreting TORCH serology—a task that can be particularly challenging when testing is performed without a clear clinical indication.

Our contributors also delve into essential topics including ventriculomegaly, Rh alloimmunization, and fetal growth restriction. We provide specialized insights into complex scenarios, such as twin pregnancies complicated by a single fetal demise.

Furthermore, we highlight the clinical utility of intrapartum ultrasound, a powerful yet underutilized modality often overlooked due to a lack of awareness. We conclude this issue with a cross-disciplinary quiz on obstetrics and fetal medicine. We wish our readers an insightful experience and a very joyous new year eve.

**The Editorial Team**

# Soft Markers: Subtle Signs, Substantial Perplexity

**Ayushi Sinha[1], Manisha Kumar[2]**
[1]Director Professor (Obstetrics and Gynecology), [2]Ayushi Sinha, Fellow National Board, Maternal Fetal Medicine
Lady Hardinge Medical College, New Delhi

## Introduction

Soft markers on ultrasound are minor, often transient anatomical variations that usually represent variants of normal but are statistically associated with an increased risk of aneuploidy. Soft Markers are

- Variants of Normal Anatomy
- Non- Specific
- Seen in some normal Fetuses
- Associated with increased risk of aneuploidy

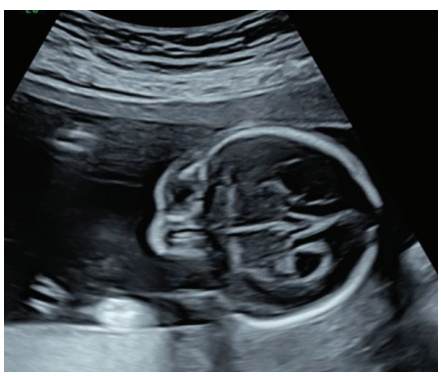
The presence or absence of these soft markers can be used to adjust a patient's priori risk for aneuploidy based on biochemical screening results or maternal age. This becomes particularly important in screening for Trisomy 21, as approximately 75% of fetuses affected by Trisomy 21 will not have major ultrasound detectable congenital anomalies at the time of second Trimester anatomic survey. This is in contrast to fetus affected by Trisomy 18 and 13, of which greater than 90 % will have major structural malformations detectable in second trimester of Pregnancy.

### 1. Choroid Plexus Cyst

A CPC is a small, fluid-filled structure within the choroid of the lateral ventricles of the fetal brain, may be Uni or Bilateral. CPCs, are seen in 1-2 percent of second trimester Scans, and are mostly isolated findings in Euploid fetuses, with resolution by 28 weeks of gestation. CPCs are seen in 30–50% of fetuses with trisomy 18.

Features of CPC such as size, complexity, laterality, or persistence do not modify risk for trisomy 18.

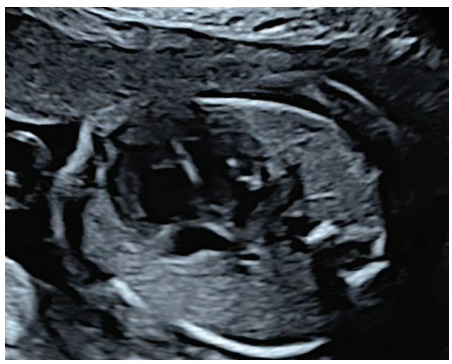
## How to counsel?



- Look carefully at any other high risk
- Age, DM, drug exposure
- Any other associated finding on ultrasound
- Whether combined test was done or not?
- If yes- what was the risk?
- If not
- Less than 20 week- Advise quadruple test
- If more than 20 week - Reassure –
- Trisomy 18 associated with other markers as well
- A CPC is not considered a structural or functional brain abnormality
- Nearly all CPCs resolve by 28 weeks
- No further evaluation needed (Gr I C)[7]

### 2. Echogenic intracardiac Foci (EICF)

An EIF refers to a tiny echogenic focus, usually less than 6 mm, located in one or both cardiac ventricles and with brightness comparable to adjacent fetal bone, which should be confirmed in at least two imaging planes. It is typically a benign papillarymuscle microcalcification that does not indicate structural heart disease.



- It is seen in 3 to 5% of normal fetuses and is present in 15-30 % of Down's syndrome fetuses.
- Single or multiple has same significance
- Positive LR is low (0.95 –1.8)

## How to counsel?

- Look carefully at any other associated finding on ultrasound

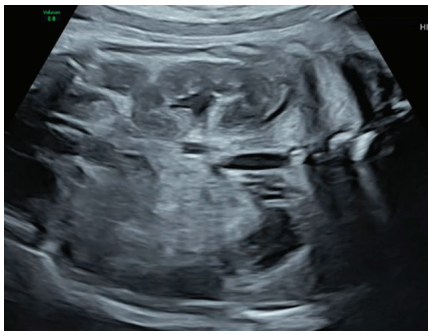
## Whether combined test done or not?

- If not
- Less than 20 wk- Advise quadruple test
- If more than 20 week - Reassure –
- EIFs do not represent a structural or functional cardiac

abnormality, No need to do fetal echocardiography

- No further evaluation needed (Gr I C)7

3. **Renal Pylectasis-** It is seen in 0.6%–4.5% of euploid fetuses. In 80% cases, it is transient variation, and reverts back to normal. Pylectasis is often seen as a normal variant or may be an early sign of genitourinary obstruction.



- Defined as Renal pelvis AP diameter, >4 mm 16 -27 weeks, >7 mm after 28 weeks
- Likelihood ratio of Down syndrome - 1.5–1.6

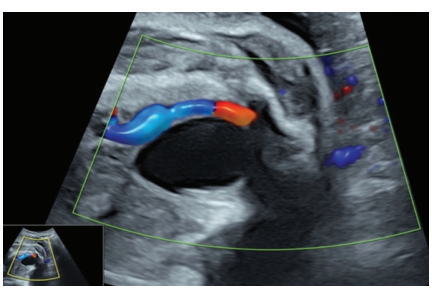
## How to counsel?

- Look carefully at any other high risk
  - Any associated finding on US
  - Combined test result
- Period of Gestation- If < 20 weeks- advise quadruple test
- Advise that it may be a transient physiologic state and reverts to normal in ~ 80%
- May progress to severe in 10-20 % cases
- Needs ultrasound at 32 weeks
- Follow up scan after one week of delivery

4. **Single Umbilical Artery**

- **Single umbilical artery** is a congenital absence of either the right or left umbilical artery, (70 % absence of left Umbilical Artery) .
- Results from - atrophy / agenesis of one of the arteries
- Incidence - 0.2% to 1% fetus

## How to counsel?



- Look for associated finding on ultrasound – cardiac & renal
- Anomaly may be present in 7%
- With multiple structural - associated aneuploidy - 4% to 50%
- If Isolated SUA-
  - No additional evaluation for aneuploidy is recommended (GRADE 1C).
  - Third-trimester ultrasound - to evaluate growth
  - Weekly antenatal fetal surveillance beginning at 36 weeks (GRADE 1C).

## If isolated no need to refer for fetal medicine opinion

5. **Aberrant Right Subclavian Artery (ARSA)**

- It is a congenital variation in the anatomy of the aortic arch, the Right subclavian artery originates from the aorta instead of the brachiocephalic artery. Prevalence is seen in 1-3.5% in euploid fetuses. It is associated with increased risk of Trisomy 21, 18, 22q11 microdeletion and heart defects in fetus.

## How to counsel?

- Look whether ARSA is Isolated / non isolated
- Fetal echo advised
- Isolated ARSA
  - No clinical significance
  - Invasive prenatal chromosomal test- not required
- Non isolated –
  - May be associated with other chromosomal abnormalities, such as 22q11 deletion
  - Karyotyping should be offered

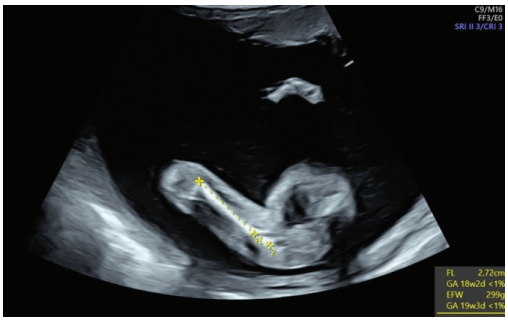
6. **Short Humerus And Femur**

Shortened Humerus: Defined when the ratio of observed humeral length to expected humeral length (based on biparietal diameter) is < 0.90.

Shortened Femur: Defined when the ratio of observed femoral length to expected femoral length (based on biparietal diameter) is < 0.92. They are nonspecific findings that can be associated with aneuploidy (especially trisomy 21), fetal growth restriction, constitutionally small fetuses, and, if disproportionate or with other bony changes, skeletal dysplasia.

Short femur and short humerus is seen in 24 – 45% and 24 – 54% fetuses with Down Syndrome respectively. It is seen in less than 5% euploid fetuses.

## How to counsel?



Any other associated finding on ultrasound?

**Whether combined test was done or not?**

If not & < 20 weeks, advise quadruple test / NIPS

**Follow up scan at 32 weeks**

Fetal growth abnormalities

Skeletal dysplasia

### 7. Echogenic Bowel

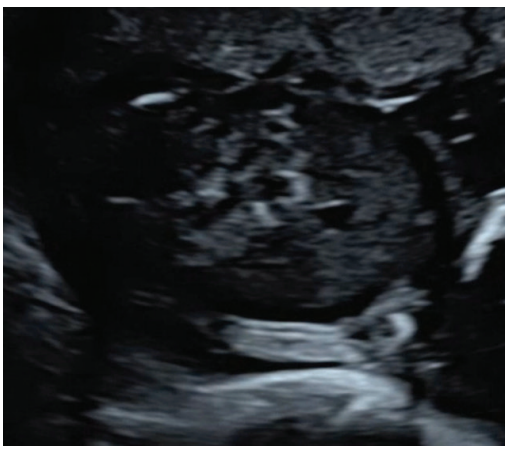
A fetal bowel segment is labeled echogenic when its ultrasound brightness matches or exceeds that of nearby osseous structures, typically using the iliac crest as the reference.

The proposed mechanism in aneuploid fetuses is thought to be decreased bowel motility & increased water absorption leading to subsequent dehydration of meconium. It can also be seen in patients experiencing bleeding in first trimester.

Echogenic bowel is also present in Cystic fibrosis, because disordered pancreatic exocrine function results in unusually viscous meconium within the fetal intestine.

Present in 0.2-1.4% of all pregnancies and in 3-5% of aneuploid fetuses, it is an isolated finding.

- How to counsel?
- Look carefully at any other high risk
- Bleeding in early pregnancy
- Any other associated renal & cardiac anomaly

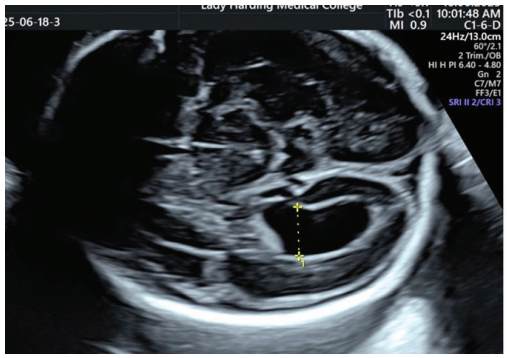


- Whether combined test was done or not?
  - Invasive testing not required for isolated EB
  - NIPS can be done
- Investigate for
  - FGR
  - Congenital infection (CMV)
  - Intra-amniotic bleeding (2-4%)
  - Cystic fibrosis (0-13%)
- Third-trimester ultrasound examination for reassessment and evaluation of fetal growth (Gr 1C)7

Refer for fetal medicine opinion

### 8. Ventriculomegaly-

- VM: Lateral ventricle >10mm but < 15 mm at any gestational age
- Seen in 1-2% normal population
- Incidence of severe VM - 2/1000
- How to counsel?
- Is there any other high risk?



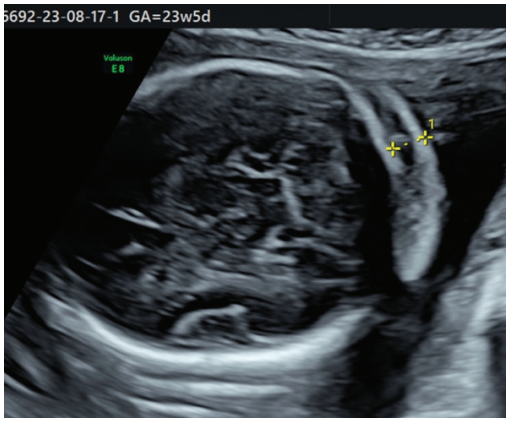
Any other associated intracranial or extra cranial abnormality on ultrasound

- May offer invasive testing
- Investigate for
- Congenital infection (CMV), toxoplasmosis
- 5% risk of later severe brain abnormality
- 15% risk of mild problems later
- Follow up ultrasound, MRI

### 9. Thickened Nuchal Fold

It is defined as ≥6 mm Nuchal fold thickness between 15–20 weeks of gestation. Has a high specificity for aneuploidy. LR - 11–18.6. 40% sensitivity, > 99% specificity for DS. Also associated with increased risk of Trisomy 18, 13 and Turners syndrome. In the absence of aneuploidy, it may be associated with structural

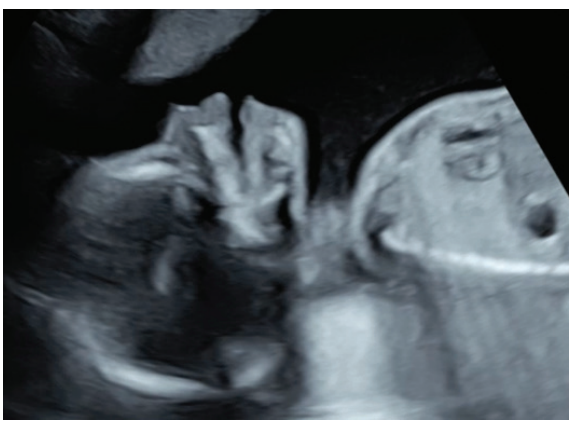
abnormalities like cardiac defects, Genetic syndromes or skeletal dysplasia.




- How to counsel?
- Refer to Fetal medicine counselling
- Any other high risk?
- Any other associated finding on ultrasound
- Fetal echocardiography
- Offer invasive testing - CMA/karyotyping

**10. Absent/Hypoplastic Nasal Bone**

A hypoplastic nasal bone in the second trimester of pregnancy is defined as a ratio against the biparietal diameter (biparietal diameter-to-nasal bone; BPD/NBL > 11), by length (≤ 2.5 mm) and by gestational age-based percentiles (<5$^{th}$ percentile). Seen in 0.1–1.2% of euploid pregnancies. Absent nasal bone is associated with increased risk of Trisomy 21.



## How to counsel?

The size of the nasal bone varies with race and ethnicity

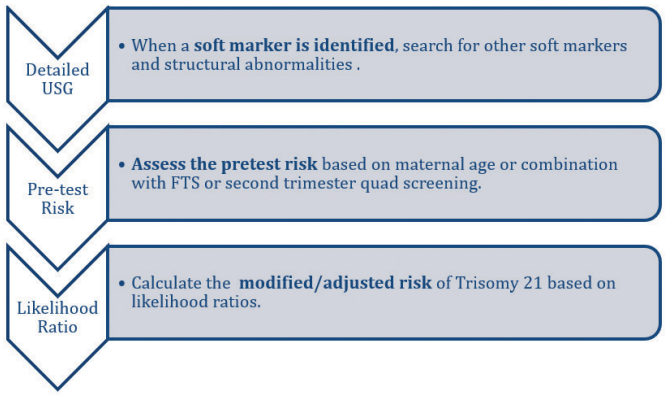
**Look carefully at any other high risk**

Any other associated finding on ultrasound

Diagnostic testing via amniocentesis – karyotyping

Non-invasive aneuploidy screening via cfDNA

## Approach to us soft markers in the second trimester




## Likelihood ratio

LR is the likelihood or probability that a given "marker" would be expected in a patient with the disease compared to the probability that the same "marker" would be expected in a patient without the disease

- A positive likelihood ratio (LR+) tells us how much to increase the probability of disease if the test is positive.
- A negative likelihood ratio (LR-) tells us how much to decrease the probability of disease if the test is negative.
- LRs from approximately 1.5 - 5 confer a small additional increase in the likelihood of the outcome;
- LRs between 5 - 10 confer a moderate additional increase in the likelihood of the outcome;

LRs of >10 confer a substantial additional increase in the likelihood of the outcome.

| Marker | LR+ | LR- | LR isolated* |
|---|---|---|---|
| Short Femur | 3.72 | 0.8 | 0.61 |
| Short Humerus | 4.81 | 0.74 | 0.78 |
| Echogenic intracardiac focus (ECF) | 5.83 | 0.8 | 0.95 |
| Mild pyelectasis | 7.63 | 0.92 | 1.08 |
| Echogenic Bowel (EB) | 11.44 | 0.9 | 1.65 |
| Increased (NFT) | 23.3 | 0.8 | 3.79 |
| Ventriculomegaly (VM) | 27.52 | 0.94 | 3.81 |
| ARSA | 21.48 | 0.71 | 3.94 |
| Absent/hypoplastic NB | 23.27 | 0.46 | 6.58 |

**EXAMPLE**

- A woman - 1: 2500, has mild hydronephrosis and intracardiac echogenic focus, all other markers absent.
- New risk = (1/2500)*7.63 (LR+ of mild hydronephrosis)*5.83 (LR+ of ICEF)*0.94*0.8*0.9*0.74*0.71*0.46 (LR – of all other markers not found) = 1/343

## Current status and clinical significance

- Over the last two decades, prenatal screening methods have evolved significantly. Ultrasound soft markers and maternal serum screening have historically formed the cornerstone of non-invasive assessment for aneuploidy risk. The introduction of cell-free DNA (cfDNA) testing has further revolutionized this field, providing the most accurate single screening approach for detecting common trisomies—21, 18, and 13
- With the widespread uptake of serum screening and cellfree DNA testing, the incremental value of individual soft markers for detecting trisomy 21 has diminished. Soft markers now function primarily as modifiers of prior risk and as clues to other pathology (e.g., infection, CNVs, structural anomalies) rather than primary indicators for karyotyping.

## Benefits in current practice

- Absence of markers in a detailed secondtrimester scan can substantially reduce background trisomy 21 risk when no cfDNA has been done.
- Multiple markers, or a marker plus structural anomalies, still significantly increase suspicion for chromosomal or other genetic conditions and justify offering diagnostic testing.
- Certain markers (echogenic bowel, urinary tract dilation, short long bones) also prompt evaluation for nonaneuploid etiologies such as IUGR, infection, CF, or postnatal urinary tract pathology.

## Suggested reading


1. Agathokleous M, Chaveeva P, Poon LCY et al (2013) Meta-analysis of second-trimester markers for trisomy 21. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 41:247–261. 10.1002/uog.12364
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3. Ultrasound of congenital fetal anomalies : differential diagnosis and prognostic indicators / Dario Paladini and Paolo Volpe. Description: Third edition
4. Callen's Ultrasonography in Obstetrics and Gynecology, 6e, Mary E. Norton, Leslie M. Scoutt, Vickie A. Feldstein
5. Creasy & resnik's maternal-fetal medicine, eighth edition ISBN: 978-0-323-47910-3
6. Raniga S, Desai PD, Parikh H. Ultrasonographic soft markers of aneuploidy in second trimester: are we lost? MedGenMed. 2006 Jan 11;8(1):9. PMID: 16915139; PMCID: PMC1681991.
7. Society for Maternal-Fetal Medicine (SMFM). Electronic address: pubs@smfm.org; Prabhu M, Kuller JA, Biggio JR. Society for Maternal-Fetal Medicine Consult Series #57: Evaluation and management of isolated soft ultrasound markers for aneuploidy in the second trimester: Am J Obstet Gynecol. 2021 Oct;225(4):B2-B15. doi: 10.1016/j.ajog.2021.06.079. Epub 2021 Jun 23. PMID: 34171388.


## Calendar for AOGD Monthly Clinical Meeting 2025-2026

| | |
|---|---|
| 26th December 2025 | Sir Ganga Ram Hospital |
| 30th January 2026 | Dr RML Hospital |
| 27th February 2026 | UCMS & GTB Hospital |
| 27th March 2026 | LHMC & SSK Hospital |
| 24th April 2026 | Hamdard Institute of Medical Sciences and Research |

# When to advise and not to advise fetal Echocardiography

**Krishna Agarwal[1], Iqra Riyaz[2]**
[1]Director professor, Obstetrics & Gynecology, [2]Maternal Fetal Medicine Fellow,
Maulana Azad Medical College, New Delhi

## Introduction

Fetal Echo, a common term for fetal echocardiography, is a specialized ultrasound technique used to assess the structure and function of the fetal heart during pregnancy.

Detailed assessment of fetal heart not only helps in the diagnosis of congenital heart disease but also identifies fetal arrhythmias, cardiomyopathies, intracardiac tumors, and hemodynamic disturbances associated with systemic fetal or placental disorders. With advancements in imaging technology and increasing awareness of the importance of prenatal cardiac screening, fetal Echo has become an indispensable component of modern obstetric and perinatal care.

However, it is essential to know when to advise a fetal echo and, more importantly, when not to recommend it, as obtaining one requires additional resources and time.

## Indications for Fetal Echocardiography

Fetal Echo is recommended when either maternal, fetal, or familial factors increase the probability of cardiac anomalies.

### 1. Maternal Indications

- Pre-gestational or insulin-requiring diabetes
- Autoimmune disease (anti-Ro/SSA or anti-La/SSB antibodies)
- Teratogenic drug exposure:
  - ACE inhibitors
  - Retinoic acid
  - Lithium
  - Antiepileptics
- Maternal infections such as rubella, parvovirus B19
- IVF and donor egg pregnancies
- Phenylketonuria

Maternal autoimmune antibodies are significant because they can cause fetal AV block and cardiomyopathy.[1,2]

### 2. Fetal Indications

- Abnormal cardiac findings on routine anomaly scan
  - Abnormal four-chamber view
  - Discrepant ventricular sizes
  - Suspicious outflow tract appearance
  - Increased nuchal translucency (NT > 3.0 mm)
  - Hydrops fetalis3
  - Fetal arrhythmias
  - Fetal significant non-cardiac anomalies suspected on routine ultrasound 4,5 (e.g., omphalocele, diaphragmatic hernia)
  - Chromosomal abnormalities (Trisomy 21, 18, 13)
  - Monochorionic twin pregnancies (risk of TTTS).

### 3. Family and Genetic Indications

- Previous child with congenital heart disease
- Parental congenital heart disease
- Known genetic syndromes associated with heart defects
  - 22q11 deletion
  - Noonan syndrome
  - Marfan syndrome

The recurrence risk of CHD rises significantly when first-degree relatives are affected.

## When not to advise Fetal Echocardiography

### 1. Maternal conditions

- Gestational diabetes mellitus diagnosed in the third trimester and well controlled
- Maternal obesity
- Preeclampsia
- Abnormal maternal serum analytes for aneuploidy
- TORCH test except in case of rubella infection

### 2. Fetal conditions

- Normal detailed anomaly scan
- Presence of an echogenic intracardiac focus or an isolated single umbilical artery
- Presence of other Soft markers of aneuploidy
- Fetal growth restriction
- Abnormal fetal Doppler study in cases with FGR
- DCDA twin with a routine, detailed anomaly scan

Fetal echocardiography requires specialized training and should be performed only by a trained person. When a fetal cardiac anomaly is diagnosed, the patient should be

referred to a multidisciplinary team including maternal-fetal medicine specialists, a geneticist, and a neonatologist.

## Principles of Fetal Echocardiography

Fetal echocardiography utilizes high-frequency ultrasound waves, combined with advanced Doppler modalities, to visualize fetal cardiac structures. A comprehensive fetal echo includes:

**1. Structural Assessment**

- Four-chamber view
- Left and right ventricular outflow tracts
- Three-vessel view and three-vessel–trachea view
- Aortic and ductal arches
- Systemic and pulmonary venous return
- Assessment of valves and septa

These standardized views ensure systematic evaluation of intracardiac anatomy and are recommended by both the International Society of Ultrasound in Obstetrics and Gynecology (ISUOG) and the American Heart Association (AHA).

**2. Functional Assessment**

- Ventricular contractility
- Ejection fraction (qualitative)
- Fractional shortening
- Myocardial performance index
- Cardiothoracic ratio

Functional evaluation is critical in fetuses with hydrops, severe anemia, twin-to-twin transfusion syndrome (TTTS), or arrhythmias.

**3. Doppler Studies**

- AV valves (mitral/tricuspid inflow)
- Aortic and pulmonary artery outflow
- Ductus venosus waveform
- Umbilical artery and vein
- Foramen ovale flow characteristics

Doppler analysis helps detect stenotic lesions, regurgitation, pressure gradients, and rhythm abnormalities.

**4. Rhythm Analysis**

- Heart rate and its variability
- Diagnosis of brady-arrhythmias and tachy-arrhythmias
- Assessment of atrioventricular (AV) conduction blocks
- Response to in-utero therapy (e.g., antiarrhythmic drugs)

Because fetal cardiac electrophysiology cannot be directly recorded, M-mode imaging and pulse Doppler are essential tools for differentiating sinus tachycardia, supraventricular tachycardia, atrial flutter, and complete heart block.

## Timing of Fetal Echocardiography

The optimal timing for fetal Echo is typically **18–24 weeks**. This period offers the best combination of fetal size, amniotic fluid volume, and acoustic windows.

However, fetal Echo may be performed earlier or later, depending on clinical needs:

## Early fetal Echo (12–14 weeks)

Indicated when:

- NT is elevated
- Family history is strong
- Genetic abnormalities are suspected

**Late fetal Echo (28–32 weeks)**

Indicated when:

- Lesions require monitoring (e.g., coarctation of the aorta)
- Rhythm disturbances need follow-up
- TTTS progression is suspected
- Fetal cardiac structures evolve dynamically; therefore, some lesions may appear or become more pronounced in late pregnancy.

## Conditions Detectable by Fetal Echocardiography

Fetal Echo is highly effective in identifying a broad spectrum of congenital cardiac abnormalities, including:

**Structural Heart Defects**

- Ventricular septal defects (VSD)
- Atrioventricular septal defects (AVSD)
- Tetralogy of Fallot
- Hypoplastic left heart syndrome (HLHS)
- Transposition of the great arteries (TGA)
- Common arterial trunk (truncus arteriosus)
- Ebstein anomaly
- Coarctation of the aorta
- Pulmonary atresia or stenosis

## Functional Disorders

- Cardiomyopathy
- Cardiac dysfunction due to anemia
- Fetal myocarditis

**Rhythm Abnormalities**

- Supraventricular tachycardia (SVT)

- Atrial flutter
- Premature atrial/ventricular contractions
- Complete heart block (especially in anti-Ro/La positive mothers)

## Clinical Benefits of Early Diagnosis

Early prenatal detection of CHD through fetal Echo provides multiple advantages:

### 1. Optimized Perinatal Planning

Prenatal diagnosis allows delivery in a tertiary center with pediatric cardiology, neonatology, and cardiac surgery support. This is crucial for duct-dependent lesions, such as HLHS or TGA, where early initiation of prostaglandin therapy is life-saving.

### 2. Immediate Neonatal Stabilization

Many severe cardiac lesions require:

- Continuous prostaglandin E1 infusion
- Avoidance of hypoxia and acidosis
- Specialized ventilation strategies

### 3. Counseling for Parents

Fetal Echo helps parents understand:

- The nature of the defect
- Expected course during the remainder of pregnancy
- Surgical options
- Survival probabilities
- Long-term functional outcomes

### 4. Prenatal Treatment Options

While limited, some fetal cardiac disorders can be treated in utero:

- Fetal arrhythmias: maternal digoxin, sotalol, flecainide
- Autoimmune complete heart block: steroids or IVIG (selected cases)
- Fetal aortic or pulmonary stenosis: interventional fetal cardiac catheterization in rare centers
- Such therapies can prevent progression to heart failure or hydrops in selected cases.

## Special Considerations in IVF Pregnancies

Accumulating evidence suggests IVF-conceived fetuses, especially from donor eggs, might have a marginally higher risk of CHD compared to natural conceptions. Causes may include:

- Epigenetic modifications
- Higher incidence of multiple pregnancies
- Underlying parental factors

Professional guidelines now encourage **routine fetal echocardiography in IVF pregnancies**, even when the anomaly scan is routine.

Routine fetal echocardiography is particularly relevant in practice, as IVF accounts for a large share of patient volume.

## Fetal Echo Views

Figure: Journal of the American Society of Echocardiography,2023

(I) Most caudal plane is Abdominal Situs plane showing fetal stomach , descending aorta and inferior vena cava, spine , and liver.

(II) Next is the Four-chamber view of fetal heart, showing right and left ventricles (RV, LV) and atria , foramen ovale , and pulmonary veins .

(III) Left ventricular outflow-tract view, showing proximal ascending aorta , LV, RV, LA, and cross-section of descending aort

(IV) Slightly more cephalad view (right ventricular outflow-tract view) showing the main pulmonary artery and bifurcation into right and left pulmonary arteries .

(V) Three-vessel-and-trachea view, showing superior vena cava (SVC), MPA, transverse aortic arch.

## Limitations of Fetal Echocardiography

While highly informative, fetal Echo does have limitations:

- Dependent on fetal position
- Breech or spine-up presentation can challenge acoustic windows.
- Limited predictive capacity
- Small defects such as tiny VSDs or minor valve regurgitations may close spontaneously before birth.
- Some lesions—especially coarctation—may only manifest postnatally.
- Operator expertise required
- Interpretation of fetal Echo demands extensive training.
- Amniotic fluid and maternal habitus influence quality
- Polyhydramnios helps; oligohydramnios hinder visibility.

## Conclusion

Fetal echocardiography has revolutionized prenatal diagnosis of congenital heart disease, enabling timely detection, multidisciplinary planning, and improved neonatal outcomes. As a safe, non-invasive, and highly informative imaging tool, it is indispensable for pregnancies with maternal medical disorders, abnormal ultrasound findings, high-risk genetic profiles, or IVF conception.

As the field of fetal cardiology continues to evolve—with advances in early first-trimester fetal Echo, fetal cardiac interventions, and three-dimensional imaging—its role

will further expand. In modern obstetric practice, a well-executed fetal echocardiogram is no longer optional but essential for comprehensive fetal assessment.

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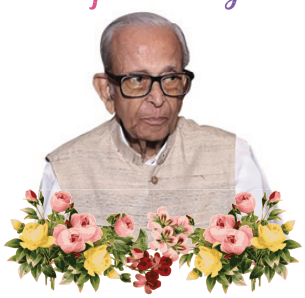
## Heartfelt Homage



**Dr SN Mukherjee 1928-2025**

**An Accomplished Obstetrician & Gynecologist/Inspirational Teacher / Compassionate Human Being & Revered Patron of AOGD**

**Dr SN Mukherjee was a deeply respected and admired father figure for the Association of Obstetricians & Gynecologist of Delhi. He was a highly accomplished, renowned and learned member who radiated warmth and positivity. He maintained a dignified persona and a benevolent attitude . He was man of high principles and values . He was a strong supporter of ethical medical practice and was deeply committed to women's health issues .**

**He was born in May 1928 in Burdwan, West Bengal.**

**He graduated from R G Kar Medical College Kolkata. His professional journey commenced as a lecturer at his alma mater. Thereafter he joined Central Health Services in 1961 as teaching faculty and served in Pondicherry Medical College (JIPMER), Shimla Medical College, Himachal Pradesh, UCMS & Safdarjung Hospital and MAMC.**

**His professional achievements were numerous and he inspired many lives . He was a WHO visiting Professor at renowned oncology centers of 3 countries, USA, UK and Japan. He was Deputy Commissioner of Family Planning to Government of India in 1978 . He also served as Additional DG, Health Services and Director CGHS .**

**He superannuated from government service in 1986. Thereafter he worked as WHO/UNFPA Consultant to Bangladesh and Bhutan for Family Planning activities and did Consultant practice post retirement .**

**He enjoyed academic activities under AOGD and NARCHI, gave orations, helped colleagues whenever requested, and spent time with friends and family. In an interview with AOGD he had expressed the high point of his life as the birth of his daughter.**

**His advice to all was-Have a definite aim in life and try to achieve it by following 3 P's. "Passion, Patience and Perseverance"**

**His life's journey was a setting example of kindness , generosity , humility and wisdom. He left for his heavenly abode on 27th November 2025 in peace and tranquility. Though he has left us, his presence is always felt and his memories will remain in our heart and minds forever .**

# Understanding TORCH Infection

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TORCH infections are important groups of organisms that are initially asymptomatic and can be difficult to diagnose clinically during pregnancy. Transplacental transmission of these infections to the fetus, especially early in pregnancy, has the potential to cause adverse obstetrical outcomes in the form of congenital anomalies, oligohydramnios, FGR (fetal growth restriction), IUFD (Intrauterine fetal death), and stillbirth. TORCH is an acronym that stands for (T) toxoplasmosis, (O) other agents, the most common being chickenpox, syphilis, parvovirus B19, and, more recently, Zikavirus, (R) rubella, (C) cytomegalovirus, and (H) herpes simplex virus. Pathogen transmission may occur prenatally via the transplacental route or perinatally through blood or vaginal secretions. Postnatal infections tend to have less impact. Other STIs, such as HIV, hepatitis B, and syphilis, can be transmitted to susceptible mothers via sexual contact.[1] Rubella and varicella can be prevented by properly immunising mothers. The majority of TORCH infections cause mild maternal illness, but the fetal consequences are serious. The key is prevention (vaccination before pregnancy for rubella/varicella); however, if infection occurs during pregnancy, monitoring (amniocentesis, scans) and treatment (antivirals/antibiotics) can help protect the fetus.

## Role of screening

Routine universal screening is not recommended for pregnant women at a low risk.

Indications for TORCH testing include a history of signs and symptoms suggestive of maternal or neonatal infection or any ultrasound findings suggestive of fetal infection.

**Diagnosis of maternal infection**- Enzyme – Enzyme-linked immunosorbent assay (ELISA)

## Interpretation

IgM and IgG both negative: patient is unexposed to infection or is unvaccinated

IgM-positive and IgG-negative: acute or primary infection. Repeat test advised after 4 weeks.

IgG positive with IgM negative: This suggests that the patient has immunity and a probable pre-gestational infection.

IgG positive with IgM positive: Indicates a recent subacute infection. It is advisable to wait for the results of the IgG avidity assay.[2]

Low IgG avidity (< 20%): cannot exclude a recent maternal infection (< 12 weeks of evolution)

Intermediate avidity (20-30%): probable infection > 12 weeks

High avidity (> 30%): confirms an infection > 20 weeks

Very high avidity (> 45%): probable infection > 40 weeks

If seroconversion is diagnosed in the context of serial gestational screening, it confirms maternal infection and treatment should be initiated immediately.

**Diagnosis of Fetal Infection**- Invasive testing (amniocentesis/cordocentesis) for pathogen-specific PCR at least 4 weeks after the start of maternal infection and not performed before 18 weeks of pregnancy. Ultrasound follow-up should be continued, as fetal infection does not necessarily mean fetal affection.[3]

## Toxoplasmosis

Toxoplasma gondii is a protozoan parasite. Infection is primarily acquired through the ingestion of raw or undercooked meat or meat products containing cysts or tachyzoites. Water, soil, and contaminated vegetables are the second sources of infection. Most women are asymptomatic, but some experience malaise, fever, headache, and lymphadenopathy, and only approximately 48% of affected individuals can recall symptoms.[4] The risk of transmission increases significantly as pregnancy progresses; however, fetal involvement decreases with gestational age. Toxoplasma IgG antibodies appear within 1–2 weeks, peak at 1–2 months, and persist at low titers lifelong. Since IgM antibodies can linger for up to 5 years post-infection, they should not be used alone for diagnosis; only ~40% of positive IgM tests indicate recent infection. Serological diagnosis relies on a rising IgG titer (repeated after 3 weeks). High IgG avidity, with no increase in titer, indicates a past infection, robust immunity, and no fetal risk.

## Rubella

Rubella is a single-stranded RNA virus that belongs to the Togavirus family. It spreads through respiratory droplets. The incubation period is 2-3 weeks, and the infectious stage lasts from 7 days before to 10 days after the onset of rash. Following the vaccination practices, the incidence of rubella has been reduced drastically, but the World Health Organisation (WHO) still estimates over 1,00,000 children worldwide are born with congenital rubella syndrome,especially in developing countries.[5] Maternal infection is diagnosed based on a fourfold increase in IgG titer or a single elevated IgM titer. If IgG antibodies are

negative and IgM antibodies are positive, repeat serology is advised after 2 weeks. If IgG antibodies are positive, a recent infection is confirmed. If infection is confirmed during pregnancy, abortion is often advised as there is no effective therapy. Immune globulin does not prevent viremia and is considered only if abortion is refused. For patients continuing pregnancy, PCR testing of amniotic fluid can confirm fetal infection and guide decisions on MTP. Note: A negative PCR does not rule out fetal infection.

## Cytomegalovirus

CMV is a DNA virus of the Herpesviridae family that establishes cellular latency after primary infection and canreactivate. Once infected, a latent infection exists for life and reactivates only in immunocompromised patients. It is the most common viral cause of congenital infection, affecting 0.2-2.2% of all live births.[6] Primary CMV infection during pregnancy has a 30-40% risk of congenital infection, as compared to 1-2% following a non-primary infection.[7,8] CMV is transmitted through close contact via saliva, urine, vaginal secretions, semen, placenta, and breast milk. In pregnant women, the main cause of infection is contact with children under 3 years of age, as, when they become infected, they shed the virus in saliva and urine for long periods of time. The incubation period is 3 to 4 weeks. The virus generally causes asymptomatic infection in immunocompetent adults but can occasionally cause flu-like illness with fever, asthenia, and arthralgia. Neonates are more symptomatic when maternal infection occurs earlier in pregnancy. The risk of SNHL and/or neurologic sequelae was 32.4% after maternal primary infection in the first trimester and zero after infection in both the second and third trimesters.[9] CMV is a leading cause of hearing loss in children.

## Herpes simplex virus

The herpes simplex virus is a double-stranded DNA virus belonging to the Alphaherpesvirinae family. HSV-1 is mainly responsible for herpes labialis and keratoconjunctivitis, whereas HSV-2 is the primary etiological agent of genital herpes. It primarily occurs through sexual contact and contact with mucosa or abraded skin. Vertical transmission: The risk of maternal-to-fetal transmission ranges from 20 to 25% if primary infection and seroconversion occur before 30-34 weeks. In recurrent infections, the transmission risk is <1%.

## Varicella

Varicella zoster is a DNA virus of the herpesvirus family. Herpes zoster infection during pregnancy does not cause fetal infection; however, varicella causes chickenpox. It is transmitted by respiratory droplets and by direct personal contact with vesicle fluid or indirectly via fomites. Varicella (chickenpox) is characterised by maculopapular rash that progresses to vesicular lesions, crusts, and eventually heals. It is accompanied by fever and malaise. The incubation period is 7–21 days, but patients are infectious from 48 h before the rash appears until the vesicles have crusted over. Fetal varicella can be indicated by the presence of ultrasonographic abnormalities after an acute maternal infection. Amniocentesis with varicella DNA PCR has a strong negative predictive value but a poor positive predictive value for detecting fetal damage that cannot be detected by non-invasive methods. Amniocentesis should not be performed before the skin lesions are completely healed. Neonatal VZV infection has a high mortality rate if maternal varicella occurs 5 days pre-delivery to 48 hours postpartum. After 36 weeks, up to 50% of babies are infected if delivery occurs within 4 weeks of maternal infection. If maternal varicella occurs after 36 weeks of pregnancy, delivery should be delayed by ≥7 days after rash onset (unless obstetric urgency exists). Neonatal prophylaxis VZIG should be administered within 72 h to infants born to women with varicella 5 days pre- to 2 days post-delivery. Breastfeeding may be continued.

## Parvovirus B19

Parvovirus B19 is a single-stranded DNA virus from the family Parvoviridae. It is cytotoxic to erythroid precursors. In childhood, it causes erythema infectiosum, also known as fifth disease, and in adults, it is generally asymptomatic, with transient aplastic crisis, which is more common in those with an underlying hemoglobinopathy, arthropathy, or myocarditis. Horizontal transmission occurs through respiratory secretions and hand-to-mouth contact. The infected person is generally infectious 5-10 days after exposure, before the onset of the rash or other symptoms. Vertical transmission: The risk of maternal-to-fetal transmission ranges from 25% to 32%.[10]

## Fetal affection

Most cases of fetal infection resolve spontaneously without adverse outcomes. Fetal parvovirus B19 has been associated with spontaneous abortion, anemia, myocarditis, non-immune hydrops fetalis (from anemia and myocarditis), and stillbirth. The overall incidence of hydrops in fetuses of mothers infected with parvovirus during pregnancy is 2.9%. The risk of fetal hydrops appears to be greater when the infection occurs earlier in pregnancy. The reasons for hydrops fetalis are aplastic anemia and myocarditis.

## Zika Virus

Zika is a Flavivirus transmitted by the mosquito Aedes aegypti. Symptoms include arthralgia, conjunctival congestion, rash, fever, and rarely, severe thrombocytopenia.[11] Diagnosis was performed using RT-PCR (blood: 7 days; urine: 14 days) or IgM ELISA (after

1 week). Prevention involves avoiding mosquito bites during pregnancy using bed nets, window screens, and diethyltoluamide (DEET)-containing topical repellents. Zika can also be sexually transmitted in semen for up to 6 months after infection, and hence, avoidance of sexual contact for 6 months after return from an endemic area is advisable if attempting conception. Zika infection causes brain abnormalities, such as microcephaly. Currently, there are no effective treatments or vaccines. Only preventive measures are effective.

## Remark

Good cleanliness, prenatal diagnosis, antiviral treatment, development and administration of booster vaccine may accomplish the goal of preventing TORCH infections in mother and infections related to congenital abnormalities in newborn

## Specific TORCH Agents: Infectivity, Diagnosis, Effect and Management

| Infection | Infectivity[12,13,14] | Diagnosis | Fetal/congenital effect | Managment |
|---|---|---|---|---|
| Rubella | Risk of fetal infection<br><12 weeks: 90%<br>12-16 weeks: 55%<br>>16 weeks: 45%<br><br>Fetal affection<br>12 weeks: 97%<br>12-16 weeks: 20%<br>16-20 weeks: only risk of deafness | Maternal: Serology<br>Fetal: Amniotic fluid PCR, fetal blood serology. | USG:<br>Cranial manifestation- Microcephaly<br>Extracranial- patent ductus arteriosus, pulmonary artery stenosis, hepatosplenomegaly, fetal hydrops, FGR, echogenic bowel, placental calcifications, placentomegaly, oligohydramnios, polyhydramnios<br>Congenital features in neonate: Gregg"s triad( microcephaly, PDA, and congenital cataract)<br>Sensorineural deafness, mental retardation, jaundice, thrombocytopenic purpura, blueberry muffin rash | MTP if seroconversion in the first trimester.(13)<br>2nd trimester: USG and fetal echo monitoring, prenatal diagnosis<br>3rd trimester: appropriate counselling<br>Pre-pregnancy screening is recommended.<br>Vaccination for those screened negative for both IgG and IgM. |
| Toxoplasmosis | Risk of fetal infection<br>13 weeks- 4-15%<br>26 weeks- 44%<br>36 weeks- >60%<br><br>Risk of fetal affection<br><br>13 weeks- 61%<br>26 weeks- 25%<br>36 weeks- 9% | Maternal infection detected by serological test should be followed by amniotic PCR for the detection of fetal infection, either 6 weeks after maternal infection or after 18 weeks of gestation.<br>Followed by a monthly serial USG.<br>A monthly ultrasound follow-up should be initiated even in cases of negative amniocentesis. | Fetal USG features:<br>Cranial manifestation- ventriculomegaly, hydrocephalus, microcephaly<br>Extracranial -<br>FGR, hyperechogenic bowel, placental calcifications,placentomegaly, olgohydroamnios, polyhydramnios, cataract formation, hepatosplenomegaly, and ascites.<br>Congenital features in a neonate:<br>Disseminated purpura<br>Hearing loss<br>Mental retardation, seizures, and hydrocephalus<br>Triad of congenital Toxoplasmosis: (chorioretinitis, intracranial calcifications, hydrocephalus) | Spiramycin 1gm orally three times daily until the end of pregnancy. Treatment of choice in the first trimester and in all cases of maternal infection suspected by serology. Follow up with 4 weekly USG.<br>If vertical transmission confirmed-<br>Pyrimethamine 50 mg/24 h orally + sulfadiazine 1 gm TDS+ folinic acid 50mg weekly<br>Pyrimethamine is contraindicated if < 14 weeks of gestation. |

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|---|---|---|---|---|
| Parvovirus B19 | Vertical transmission: 17 - 33%(14)<br>Rate of fetal loss among women with serologically proven parvovirus B19 infection<br>8-17% before 20 wks POG<br>2-6% after 20 wks POG | Maternal: Serology(seroconversion or presence of IgM antibodies)<br>Fetal: Targeted sonography +/-MCA PSV every 2 weeks for 12 weeks starting 4weeks after infection. Cordocentesis if the fetus affected<br>PCR in AF in fetuses with hydrops | Fetal USG- Ascites, pericardial effusion, skin edema, fetal anaemia (MCA-PSV > 1.5 MoM) , placentomegaly, polyhydramnios, fetal hydrops, organomegaly, | Intrauterine transfusion for fetal anaemia |
| Varicella | Risk of fetal infection<br>before 13 weeks- 0.4%<br>13-20 weeks - 2%<br>>20weeks- no risk | Maternal: Clinical<br>Fetal:<br>detailed USG 5 weeks after infection at 16 -20 weeks of gestation.<br>Amniocentesis for PCR | Fetal USG-<br>Cranial manifestation- Cortical atrophy, hydrocephalus, microcephaly<br>Extracranial- Soft tissue calcification, microphthalmia, polyhydramnios<br>Congenital features in neonate- Dermatomal scarring (76%), mental retardation, Horner syndrome<br>Eye: Chororetinitis, microphthalmia, cataract, optic atrophy (51%)<br>bone defects, limb hypoplasia (49% soft tissue calcifications<br>Hydronephrosis<br>Growth restriction, Low birth weight | Prepregnancy- Vaccination<br>Pregnancy -<br>Exposure to infection :<br>Give VZIG to the mother, best within 96 hours of exposure, can be given up to 10 days of exposure.<br>* A second dose of VZIG may be required if a further exposure is reported and 3 weeks have elapsed since the last dose.<br>Established infection :<br>* Do not give VZIG.<br>* Oral acyclovir 800mg five times daily for seven days should be started within 24 hours of developing the rash (15). IV in pneumonia or severe cases<br>Evaluate fetus by detailed USG, at 5 weeks after maternal infection (at 16-20 weeks) |
| CMV | Risk of Fetal infection<br>1st trimester- 36.8%<br>2nd trimester- 40.3%<br>3rd trimester- 66.2%<br>Reinfection: 1-2.2%<br>Risk of fetal affection-<br>1st trimester- 19.3%<br>2nd trimester- 0.9%<br>3rd trimester- 0.4% | Maternal: serology - seroconversion, avidity<br>Fetal : CMV detection in amniotic fluid by PCR, culture or immunofluorescence (Amniocentesis to be done 8 weeks after maternal infection or after 20 weeks gestation)<br>Fetal blood-serology. | Fetal USG findings<br>Cranial manifestation- Ventriculomegaly, Microcephaly, Intracranial calcification, periventricular pseudocyst, cerebellar abnormalities( vermian hypoplasia, haemorrhage)<br>Extracranial- hepatic calcifications, Echogenic bowel, Fetal hydrops, Meconium peritonitis, Growth restriction<br>Neonatal disease :<br>Cytomegalic inclusion disease<br>SNHL in 30%, Hepatosplenomegaly<br>Microcephaly with intracranial calcifications, Chorioretinitis, Optical atrophy, Mental and psychomotor delay<br>Dental abnormalities | USG monitoring. |

| | | | | |
|---|---|---|---|---|
| Herpes simplex | 20-25% with primary infection before 30-34 weeks.<br>41% risk of neonatal herpes when genital lesion present at the time of delivery(16)<br><1% in recurrent infections | Diagnosis by ELISA for HSV-1 and 2 antibody or by PCR and culture if genital ulcer, mucocutaneous lesion present | Fetal USG findings<br>Cranial manifestation- Microcephaly, ventriculomegaly<br>Extracranial- microphthalmia, FGR, placental calcifications,placentomegaly, olgohydroamnios, polyhydramnios<br>Neonatal disease – Meningoencephalitis, mental retardation, chorioretinitis,scarring or blisters on skin | Antenatal- Acyclovir orally 400mg three times a day for 7-10 days. Suppressive therapy from 36weeks until delivery, 400mg orally three times a day. (17)<br>Intrapartum- Active genital lesions over the perineum, vagina and cervix during labour are an indication for CS.<br>Allow vaginal delivery if no<br>genital lesions are seen.<br>In PPROM,<br>individualise patient management. |

Not all ultrasound signs are present in all infections; some tend to be more common in certain infections, depending on the pathogen.

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# Assessment Pathways and Parental Counselling in Fetal Ventriculomegaly

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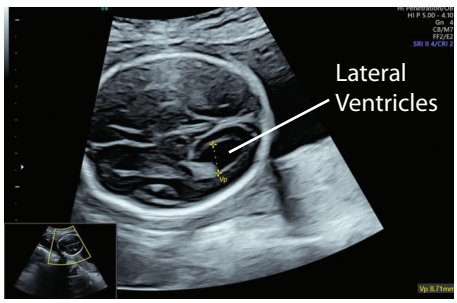
## Introduction

Fetal cerebral ventriculomegaly is among the most frequently encountered prenatal neurological findings, often first detected in approximately 1% of routine obstetric ultrasounds.[1] As our diagnostic capabilities advance, the central challenge lies not only in accurate identification but also in providing clear, compassionate, and evidence-based counselling regarding prognosis and postnatal care.

The cerebral ventricles form a connected system of CSF-filled cavities within the brain parenchyma. This system—comprising the paired lateral ventricles, third ventricle, cerebral aqueduct, and fourth ventricle—facilitates the production, flow, and resorption of cerebrospinal fluid. CSF is generated primarily by the choroid plexus, and any disruption in its circulation or clearance can lead to pathological dilatation.[2]




## Definition

Fetal ventriculomegaly is defined as:

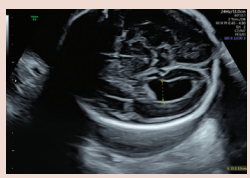
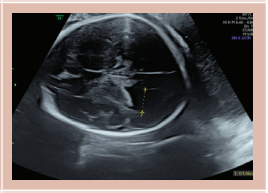
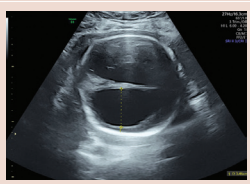
- Atrial diameter >10 mm at the level of the posterior (or anterior) horn of the lateral ventricle at any gestational age/ enlargement of the lateral ventricular atrium measuring ≥10 mm on the axial transcerebellar view.
- Alternatively, ventriculomegaly may be suggested by:
- Choroid plexus separation >3 mm from the medial wall of the lateral ventricle3.
- Beyond being a structural finding, it often serves as a sentinel marker for associated CNS anomalies, infections, or genetic disorders. Because the diagnosis evokes considerable parental anxiety, a systematic evaluation alongside sensitive, well-informed counselling is crucial.

## Prevalence

- Approximately 1 in 100 fetuses at 20 weeks' gestation
- Around 1 in 1,000 live births4

## Classification

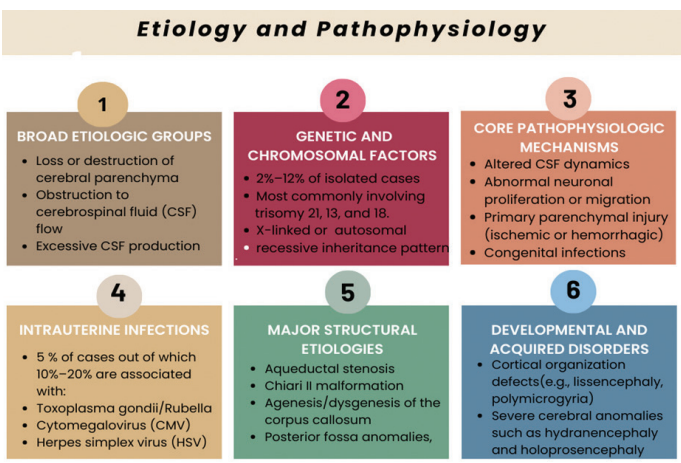
The degree of ventricular enlargement is commonly stratified as follows:

| Classification | | |
|---|---|---|
| Mild (borderline) ventriculomegaly | Atrial diameter 10–12 mm |  |
| Moderate ventriculomegaly | Atrial diameter 12.1–15 mm |  |
| Severe ventriculomegaly | Atrial diameter >15 mm, a category that may also be referred to clinically as fetal hydrocephalus |  |

- Isolated ventriculomegaly refers to ventricular dilatation not accompanied by structural anomalies or identifiable chromosomal or genetic abnormalities on detailed ultrasonography.[5]

## Etiology and Pathophysiology[6]

Fetal ventriculomegaly reflects a wide spectrum of underlying conditions ranging from benign variants to severe congenital or genetic disorders. Even when detected in isolation, it may signal chromosomal anomalies, intrauterine infection, or subtle cerebral maldevelopment.



## Evaluation

Once ventriculomegaly is detected, a **structured, multimodal evaluation** becomes essential. High-resolution ultrasonography forms the cornerstone of assessment, while fetal MRI offers complementary detail when greater anatomical clarity is required. Together, these modalities delineate the severity of ventricular dilatation, identify associated anomalies, and guide counselling, prognostication, and management.

## Imaging and Diagnostic Evaluation[8]

### 1. Fetal Ultrasonography

Ultrasonography remains the primary modality for diagnosis and surveillance of fetal ventriculomegaly. The lateral ventricles are reliably visualised by 13–14 weeks, with the choroid plexus serving as a consistent anatomical landmark. Although overall ventricular dimensions increase with gestation, the atrial diameter remains relatively constant from mid- to late pregnancy, making the second and third trimesters optimal for assessment.

## Measurement Technique

- Atrial width should be assessed in a true axial plane, visualizing the atrium of the lateral ventricle at the level of the parieto occipital fissure with magnification involving 90% screen.
- The measurement is taken inner-to-inner, from the medial ventricular wall to the lateral wall

**Key parameters include:**

a) Normal atrial width: 4.5–7.6 mm between 15–40 weeks

b) Ventriculomegaly: atrial diameter ≥10 mm (approximately 2.5 SD above the mean)

c) Technical considerations: Breech presentation may necessitate a fundal approach or upright scanning to obtain accurate multiplanar views

### 2. Fetal MRI

Fetal MRI provides substantial incremental value, identifying additional structural abnormalities in nearly half of fetuses with ventriculomegaly. It is especially useful for diagnosing:

a) Agenesis of the corpus callosum

b) Absence of the septum pellucidum

c) Cortical malformations

d) Cerebrovascular or parenchymal injury

e) Abnormalities of neuronal migration such as lissencephaly

## Salient Differentiating Features

| Feature | Neurosonogram (Detailed Ultrasound) | Fetal MRI |
|---|---|---|
| Primary role | First-line test | Second-line confirmatory test |
| Key strengths | • Real-time views<br>• Accurate ventricle measurement<br>• Good midline and posterior fossa assessment | • Excellent tissue detail<br>• Better for cortex, white matter, hemorrhage |
| Best for | Ventricular size, shape, midline structures | • Cortical malformations<br>• Corpus callosum anomalies<br>• Parenchymal injury |
| Limitations | Affected by maternal obesity, oligohydramnios, fetal position; operator-dependent | • Motion artefacts<br>• Higher cost<br>• Limited availability |
| Measurement notes | Standard for atrial measurement | Axial values may be ~1–2 mm larger than US |
| Repeat use | Easy and frequent follow-up | Not ideal for repeated scans |
| Effect on counselling | Essential for baseline assessment and monitoring progression | Improves prognosis by detecting subtle lesions |

## Follow-Up Imaging

A repeat scan in 2–4 weeks is recommended to determine whether ventricular size is:

- Stable
- Improving
- Progressive
  - Progressive ventriculomegaly is consistently associated with less favourable neurodevelopmental outcomes.

### 3. Genetic Evaluation[9]

Genetic assessment constitutes an essential component of the diagnostic work-up.

a) Karyotype identifies aneuploidy in ~5% of mild–moderate cases, most frequently trisomy 21.

b) Chromosomal microarray (CMA) offers a substantially higher diagnostic yield, detecting clinically significant copy number variants in up to 10% of fetuses.
   - Importantly, ≈6% of cases with a normal karyotype still demonstrate pathogenic findings on CMA.

- Given its superior sensitivity, CMA is recommended as the first-tier genetic investigation for fetuses presenting with ventriculomegaly.
- Chromosomal microarray (CMA) enhances detection of pathogenic CNVs, providing an additional 8–12% yield over karyotype and achieving overall detection rates of ~9–16% even in mild isolated ventriculomegaly.
- Prenatal exome sequencing (WES/ES) offers a further diagnostic yield of ~31% after normal karyotype and CMA, with higher yields (~45%) in non-isolated or severe cases.
- Offer CMA for all ventriculomegaly; consider WES/ES in moderate or severe cases.

#### 4. Evaluation for Congenital Infection[8]

Assessment for intrauterine infection is crucial, as congenital infections account for approximately 1–2% of ventriculomegaly cases. The most frequently implicated pathogens include:

- Cytomegalovirus (CMV)
- Toxoplasma gondii

## Diagnostic Approach

a) Other infections—rubella, parvovirus B19, Zika virus, and HSV—should be considered based on maternal exposure risks.

b) Infection-related ventriculomegaly often occurs with accompanying CNS or multisystem abnormalities.

c) Amniotic fluid PCR remains the most accurate confirmatory test.

d) Maternal serology is used as an initial screen.

e) Negative IgG and IgM titres effectively exclude infection.

f) Equivocal serology may require IgG avidity testing or review of stored first-trimester samples.

g) Test maternal blood for antiplatelet antibodies when fetal intracranial hemorrhage is suspected.

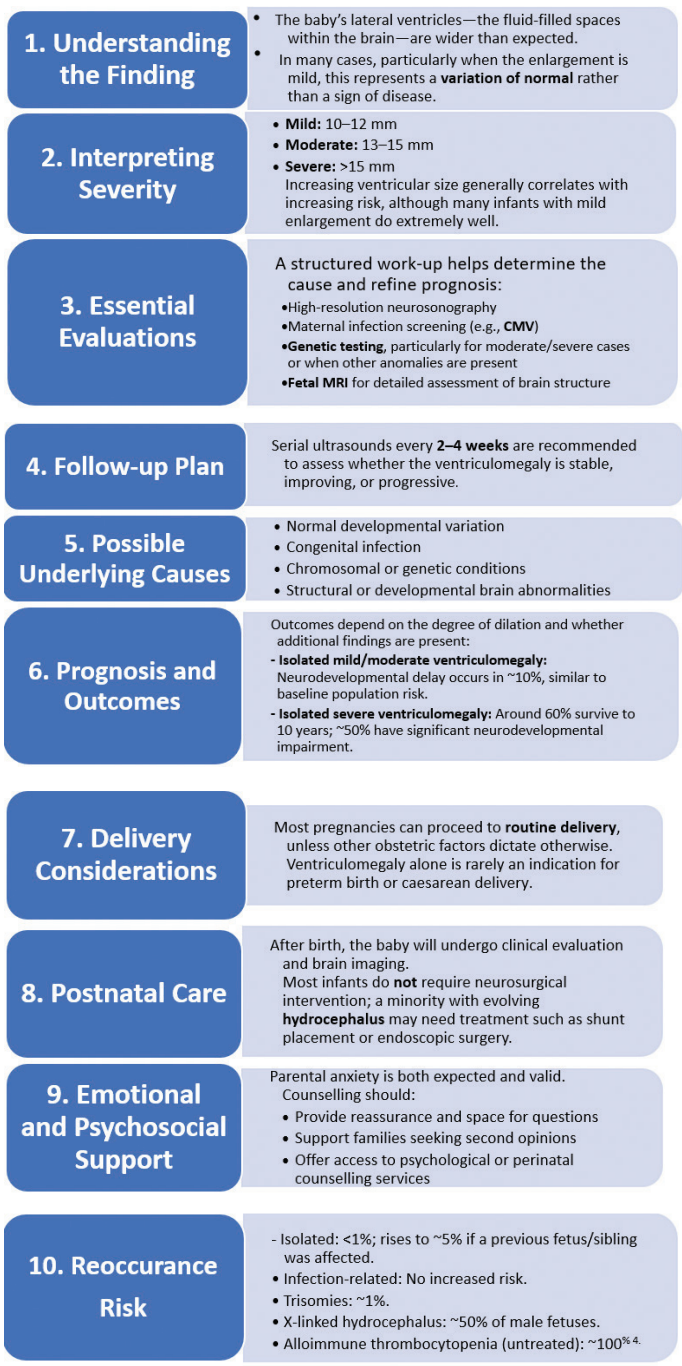
## Key Prognostic Indicators

Prognosis is influenced by several interrelated factors:

1. **Degree of Ventricular Dilatation** – Larger atrial measurements correlate with poorer outcomes.
2. **Trend Over Time** – Progressive enlargement is associated with unfavourable prognosis.
3. **Head Circumference** – Reduced head size for gestational age suggests impaired brain growth.
4. **Fetal Growth Restriction** – Coexisting FGR further worsens prognosis.
5. **Genetic or Infection Findings** – Abnormal results significantly influence outcome.
6. **Associated Structural Anomalies** – Presence of additional CNS or extracranial anomalies increases risk of neurodevelopmental impairment.
7. **Gestational Timing** – Early-onset ventriculomegaly, especially marked dilation in the first or early second trimester, carries higher risk.

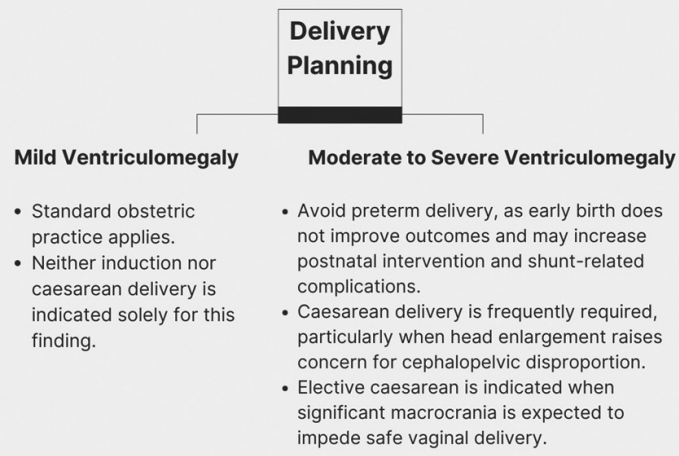
## Counselling: Principles and Best Practice[10]

Effective counselling for fetal ventriculomegaly balances scientific clarity with empathy. Parents need information that is accurate, comprehensible, and framed with hope wherever appropriate.



## Delivery Planning

Delivery decisions in pregnancies complicated by ventriculomegaly should prioritize obstetric safety while recognizing that timing and mode of birth rarely influence neurological outcomes.




- Cephalocentesis has an extremely limited role and is reserved only for pregnancies with a lethal fetal prognosis, performed solely to protect maternal health.

## General Recommendation

Delivery planning should be **individualized**, balancing maternal safety with anticipated neonatal needs.

- Isolated mild or moderate ventriculomegaly: routine timing and mode of birth
- Severe ventriculomegaly or head circumference ≥40 cm: consider caesarean delivery
- Ensure neonatal team availability, particularly when additional anomalies or postnatal compromise are anticipated

## Postnatal Evaluation

A structured postnatal assessment is essential to define the underlying cause, evaluate neurological status, and plan long-term follow-up.

- Cranial ultrasound or MRI to reassess ventricular size, parenchymal integrity, and associated malformations
- Ophthalmic evaluation when congenital infection is suspected
- Neurodevelopmental monitoring through infancy and early childhood to identify delays early and initiate intervention
- Genetic evaluation, particularly when prenatal testing was declined, incomplete, or yielded uncertain results

## Conclusion

The evaluation of fetal ventriculomegaly demands a structured, evidence-based, and family-centred approach. High-quality neurosonography remains the cornerstone of diagnosis, while adjunctive fetal MRI, chromosomal microarray, and targeted infection testing significantly enhance diagnostic precision and reveal subtle yet clinically important abnormalities. Integrating imaging findings with etiological evaluations and longitudinal trends allows accurate characterization of the condition and meaningful prognostication. Because outcomes depend on the degree and progression of ventricular dilation, associated anomalies, and underlying genetic or infectious causes, counselling must be individualized, balanced, and delivered with clarity and empathy. Ultimately, rigorous assessment paired with compassionate, informed communication supports families in navigating complex decisions and optimizes clinical care for pregnancies affected by ventriculomegaly.

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# Management of Rhesus Negative Pregnancy: Prevention is the key

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## Introduction

Isoimmunization is the development of antibodies against the antigens of another individual . Antigens which are present on the human RBCs are:-

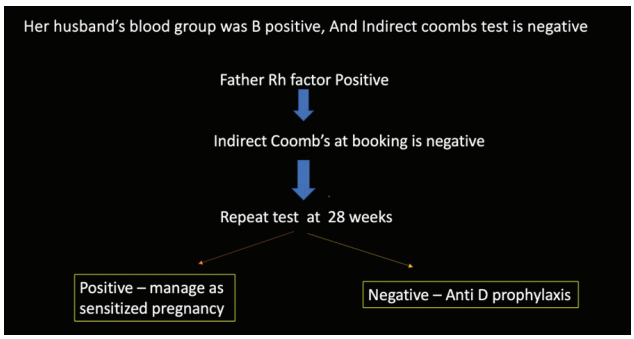
- ABO antigens (A, B, AB),
- Rhesus D antigen (Rh-D)
- Other atypical antigens - Cc, Ee, Kell (K), Duffy (Fya), Kidd

## Management of Rh-negative pregnancy – 3 case scenarios

### Case 1

A 25 year old, primigravida, came for second visit at 20 weeks with the report of investigations advised in the first visit which shows the blood group as B negative. What is the next step?

- Indirect Coomb's test (ICT)




Anti D Immunoglobulin: A prophylactic dose of 300 micrograms of anti-D immune globulin can prevent Rh D alloimmunization after exposure to up to 30 mL of Rh D-positive fetal whole blood or 15 mL of fetal red blood cells. Most of the pregnancy – FMH – 4ml

## Conditions which increase chances of FMH

### Antepartum

- Abortion, Ectopic pregnancy
- Abdominal trauma
- Obstetric procedures- amniocentesis, chorionic villus sampling
- External cephalic version

### Intrapartum

- Forceps delivery
- Caesarean sections
- Stillbirths
- Multiple pregnancies
- Placental abruption
- Manual removal of placenta

### Birth planning

- Wait for spontaneous labor or till 40 weeks
- Precautions to be taken at delivery are:-
  - Clamp cord immediately
  - Keep the cord long for possible catheterization
  - Do not use prophylactic ergometrine to prevent PPH.
  - At caesarean section, blood spilling should be avoided.

## How can we quantify the feto-maternal hemorrhage?

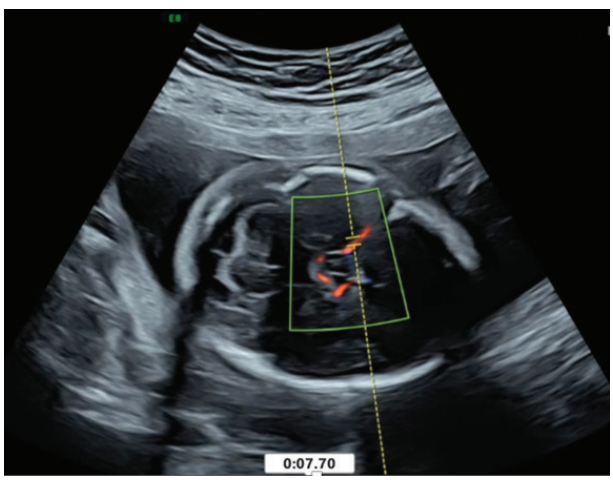
- By Kleihauer- Betke test

### Is there some way by which we can know the status of fetal blood group during pregnancy?

- Cell free fetal DNA – can be tested for Rh status

### Case 2

- A 24 yr old , G2P0L0E1 came at 20 weeks , her ABO Rh typing was A -ve, the ICT – was found to be positive, the titer was 1:2
- Management in current pregnancy
- Middle cerebral artery Peak systolic velocity



Axial section of the base of brain

- Insinuate MCA close to its origin
- Angle of insinuation <300
- Flow of blood -towards the probe
- Avoid pressure from probe
- Fetal Head should not be moving
- Repeated every 1- 2 weeks
- Additional signs of severe anemia on ultrasound
  - Polyhydramnios
  - Increased Cardiothoracic ratio
  - Thick placenta
  - Hydrops
- When should we plan delivery if there is no fetal anemia on MCA PSV ?
- At 37-38 weeks

## Investigations to be done from cord blood

- Hemoglobin, Hematocrit
- Reticulocyte count
- Blood group (ABO Rh)
- Serum bilirubin level
- Direct Coombs Test

**Summary of management of case with first isoimmunized pregnancy**

**Summary of management of case with first isoimmunized pregnancy**

| Rh antibody titre- | MCA peak systolic | Mari et al ref chart | Delivery-Timing |
|---|---|---|---|
| • If titre is below the critical value, titre is repeated every 4 weeks until 28 weeks and then every 2 weeks. | • Velocity- serial measurements done every 1-2 weeks and compared with the reference centile charts | • Above threshold of 1.5 MoM for that gestational age | • Depends on gestational age, severity of fetal anemia and fetal maturity. Delivery is planned between 37-38 weeks if fetal surveillance is reassuring. |

**Case 3**

- 29 year old, G3P2L1, 30 weeks, non-consanguineous marriage referred due to previous baby with hydrops and high MCA PSV values – indicating fetal anemia
- Her blood group is B -ve and husband is O +ve
- Obstetric history:
- First pregnancy – 5 yrs ago
- Spontaneous conception, unsupervised pregnancy, received Inj Anti D postnatally
- Second pregnancy – 2 yrs ago
- History of Intrauterine fetal demise at term, Baby was hydropic at birth
- At 30 weeks the fetus is found to be severely anemic …….

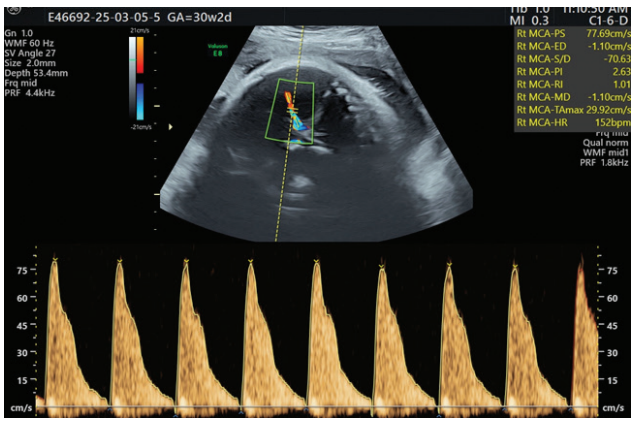



**Table 3.** Expected peak velocity of systolic blood flow in the middle cerebral artery as a function of gestational age.

| Week of Gestation | Multiples of the Median | | | |
|---|---|---|---|---|
| | 1.00 (Median) | 1.29 | 1.50 | 1.55 |
| | cm/sec | | | |
| 18 | 23.2 | 29.9 | 34.8 | 36.0 |
| 20 | 25.5 | 32.8 | 38.2 | 39.5 |
| 22 | 27.9 | 36.0 | 41.9 | 43.3 |
| 24 | 30.7 | 39.5 | 46.0 | 47.5 |
| 26 | 33.6 | 43.3 | 50.4 | 52.1 |
| 28 | 36.9 | 47.6 | 55.4 | 57.2 |
| 30 | 40.5 | 52.2 | 60.7 | 62.8 |
| 32 | 44.4 | 57.3 | 66.6 | 68.9 |
| 34 | 48.7 | 62.9 | 73.1 | 75.6 |
| 36 | 53.5 | 69.0 | 80.2 | 82.9 |
| 38 | 58.7 | 75.7 | 88.0 | 91.0 |
| 40 | 64.4 | 83.0 | 96.6 | 99.8 |

MCA PSV chart by Mari et al

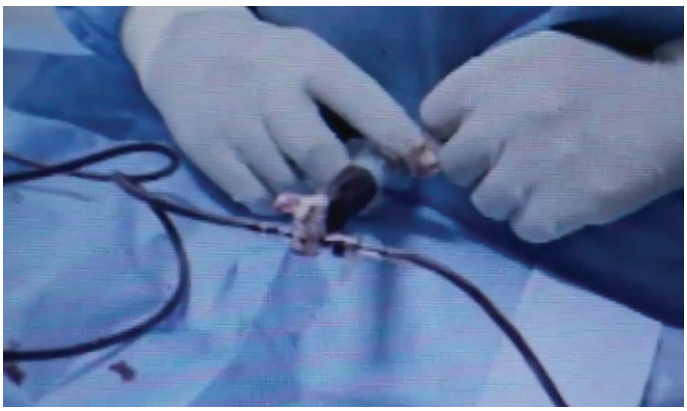
## Prerequisites blood to be transfused

- O negative
- Packed cell – Hct – 75-85%
- Leuco-reduced
- Fresh- < 5days
- Irradiated
- Crossed matched with mother's blood

## Intrauterine transfusion procedure

**The Check list before blood transfusion**

- Consents/forms
- Steroid cover
- Blood, NPO
- Inj Vecuronium
- Spinal needle 20 G
- CBC vials, 3 way cannula, tubing
- Inj H progesterone
- Antibiotic
- Pretest Hct
- Transfusion – 10ml/min
- Post BT hct
- NST after 2-3 hrs
- MCA c/m



**Complications of IUT**

- Potential fetal complications
  - Transient Fetal bradycardia
  - Cord accident – cord hematoma
  - Umbilical artery spasm
  - Hemorrhage from cannulation site
  - Overloading of fetal circulations
- Potential maternal complications
  - Chorioamnionitis
  - PROM
  - Preterm labor
- Timing of delivery
- No high-quality data regarding the optimal timing of delivery in anemic fetus
- The continuation of intrauterine transfusion therapy until the end of the 35th week of pregnancy; and
- Prolonging gestational age to between 37 weeks 0 days and 38 weeks 6 days before proceeding to delivery.
- Balance the Risk of stillbirth, risk of another procedure Vs Risk of prematurity
- The goal is to deliver a fetus with no or only mild to moderate anemia.

## Conclusion

Rh iso immunization is the most common cause of fetal anemia. It has been found that 5% of Indian population is Rh negative, hence screening is essential. If anti D is not given at all 17% cases will be isoimmunized, If given just after delivery, the risk of isoimmunization is reduced to 2%, if given in antenatal period also the risk is reduced to 0.14% only, therefore, Anti D advised in antenatal period is the best form of prevention. MCA – PSV has good accuracy for diagnosing fetal anemia. Intrauterine transfusion is the most successful in utero therapeutic procedure.

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# Fetal growth restriction: Saving babies' lives care bundle

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## Introduction

**Maternal–fetal medicine expertise is essential** for accurate diagnosis, risk stratification, and timely intervention to improve outcome in fetal growth restriction. This article highlights the diagnostic and management challenges of fetal growth restriction, affecting ~10% of pregnancies and a major cause of perinatal morbidity and mortality. The key challenge is differentiating constitutional smallness from pathological FGR due to placental dysfunction.

## Definitions and Classification of FGR

An appropriate-for-gestational-age (AGA) fetus has a size between the 10th and 90th percentiles for its gestational age.

Small for Gestational Age (SGA) at birth is diagnosed when birthweight is below the 10th centile for gestational age.[1]

Fetal Growth Restriction (FGR): A Delphi consensus–based definition of FGR (Table 1) has been proposed for use in both clinical practice and research, encompassing early-onset FGR (before 32 + 0 weeks) and late-onset FGR.[2]

**Table 1:** Delphi Consensus [2016] based definitions for early and late fetal growth restriction (FGR) in absence of congenital anomalies.[2]

| Early FGR: Gestational age < 32 weeks, in absence of congenital anomalies | Late FGR: Gestational age ≥ 32 weeks, in absence of congenital anomalies |
|---|---|
| AC/EFW < 3rd centile or UA-AEDF | AC/EFW < 3rd centile |
| OR | OR at least two out of three of the following: |
| AC/EFW < 10th centile combined with either: | 1. AC/EFW < 10th centile |
| 1. UtA-PI > 95th centile and/ or | 2. AC/EFW crossing centiles > two quartiles on growth centiles* |
| 2. UA-PI > 95th centile | 3. CPR < 5th centile or UA-PI > 95th centile |

*Growth centiles are non-customised centiles. AC – Fetal abdominal circumference; EFW – Estimated fetal weight; CPR – Cerebroplacental ratio PI – Pulsatility index; UA – Umbilical artery; UtA – Uterine artery.

- Differentiating FGR from constitutional smallness is essential for management. SGA includes many healthy, constitutionally small fetuses (≈50–70%) with low risk of adverse outcomes. In contrast, FGR represents pathological failure to reach growth potential; some affected fetuses may measure above the 10th centile yet still carry increased perinatal risk.
- The Saving Babies' Lives Care Bundle version 3 (SBLCBv3) provides practical definitions for FGR, identifying absent or reversed end-diastolic flow in the umbilical artery as a feature of early-onset FGR. Importantly, it emphasizes that a normal umbilical artery Doppler from 32 + 0 weeks' gestation does not exclude fetal growth restriction or the risk of fetal compromise.[3]
- Suboptimal growth is difficult to define. SBLCBv3 describes it as slowing growth velocity with a downward centile trend, while the Delphi consensus defines it as AC or EFW crossing more than two quartiles on non-customised charts at ≥32 + 0 weeks (≈50-centile drop). Static growth denotes no increase in AC or EFW over at least 14 days.[1]

## Risk Assessment and Stratification

The risk of FGR and FGR-related stillbirth is determined by the interaction of multiple factors. A priori risk reflects the baseline probability at the start of pregnancy based on maternal and obstetric history. While the absence of individual risk factors may lower overall risk, it does not eliminate it. Importantly, the coexistence of multiple risk factors can have a cumulative effect, substantially increasing the likelihood of FGR or stillbirth.[1]

## Booking History

As per RCOG 2024, booking history should assess previous FGR/SGA, stillbirth, preterm birth or other placenta-mediated complications; maternal conditions such as chronic hypertension, vascular diabetes, renal or autoimmune disease; and current antenatal risks including hypertensive disorders, abnormal Doppler, low PAPP-A, smoking, and poor weight gain.

## Current pregnancy risk factors for FGR

In the current pregnancy, biochemical markers such as low PAPP-A (<5th centile), raised AFP or inhibin A (>2 MoM) indicate placental dysfunction and increased risk of SGA/FGR, particularly later in gestation, and should prompt uterine artery Doppler and serial growth surveillance, though they are not recommended as primary screening tools. Uterine artery Doppler at 18–23+6 weeks is indicated in high-risk women and when anomaly scan findings such as echogenic bowel, isolated single umbilical artery, or EFW <10th centile are present. Risk increases with hypertensive disorders, significant first-trimester bleeding, or antepartum haemorrhage, warranting reclassification to high-risk, consultant-led care with serial growth assessment.[1]

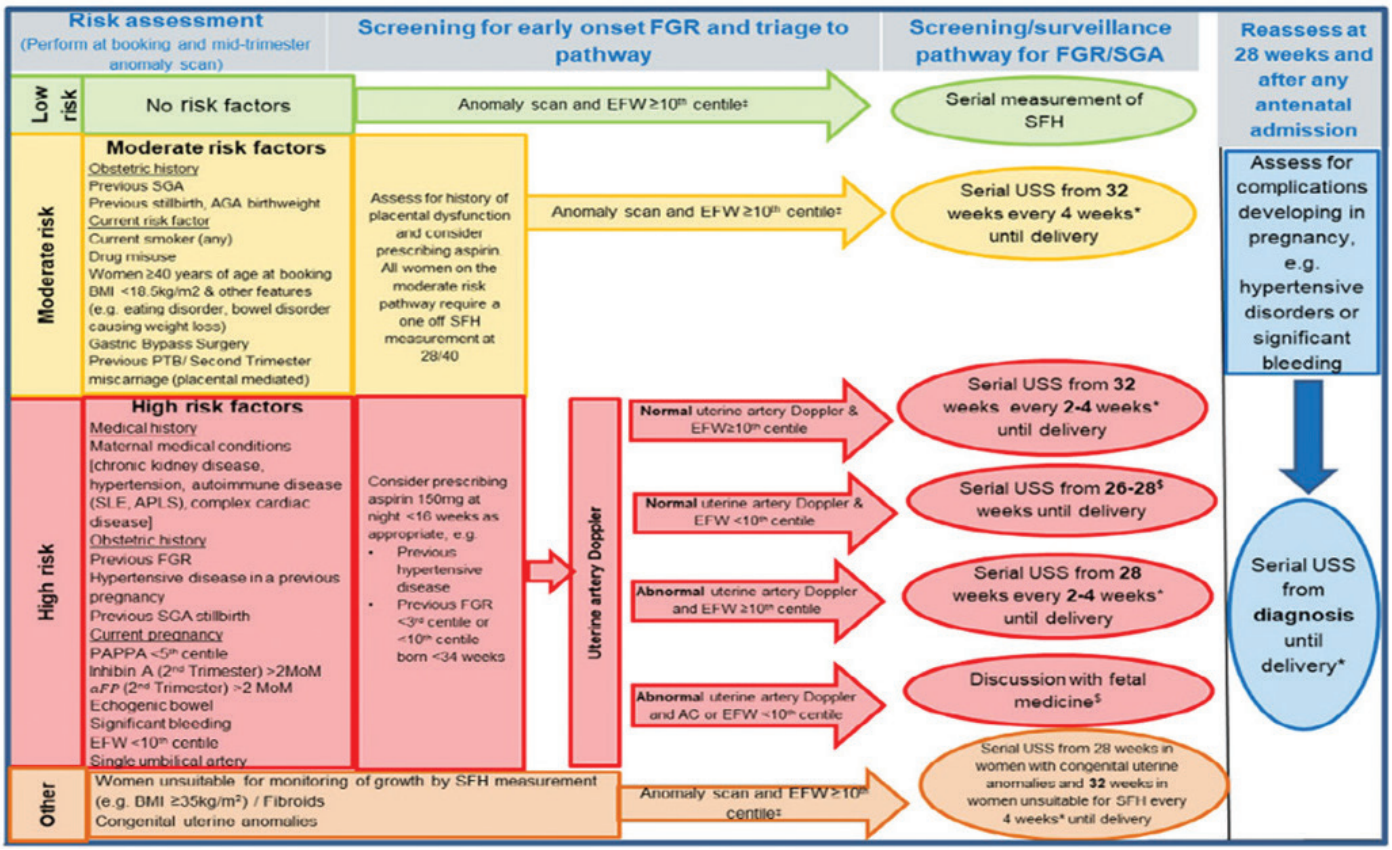



**Figure 1:** Risk Stratification of Fetal Growth Restriction (Adapted from RCOG Green-top Guideline No. 31, 2024) [1]

## Role of Fetal Medicine Assessment

### When to refer?

- Referral to fetal medicine is recommended when EFW is <3rd centile, or <10th centile with abnormal uterine artery Doppler at the anomaly scan, to assess aneuploidy and uteroplacental insufficiency. Severe SGA (<3rd–5th centile) carries an aneuploidy risk up to 20%.
- Offer invasive testing with microarray in anomalous or early (<23 weeks) SGA.
- Exome sequencing is reserved for multisystem anomalies or isolated short long bones.
- Screen for CMV and toxoplasmosis, as infections cause ~5% of SGA; consider malaria or Zika in high-risk populations.[1]

## Surveillance

Surveillance frequency is individualized based on gestation, severity of FGR, and Doppler findings.

### Ultrasound parameters

- Surveillance in fetal growth restriction requires a multimodal approach. Serial fetal biometry (EFW and AC) should be performed every 2 weeks to assess growth velocity.
- Umbilical artery Doppler is the cornerstone of surveillance, evaluating placental resistance through PI and the presence of absent or reversed end-diastolic flow.
- Middle cerebral artery Doppler, including PI and CPR/UCR, aids risk stratification, particularly in late-onset FGR.
- Ductus venosus Doppler is essential in early-onset FGR to detect fetal cardiovascular compromise and guide timing of delivery.
- Amniotic fluid volume should be assessed using AFI or deepest vertical pocket, as reduced liquor reflects chronic placental insufficiency.

## Cardiotocography

- Cardiotocography is a key surveillance tool in FGR. Computerized CTG is preferred in early-onset FGR, with short-term variation (STV) guiding detection of hypoxia and timing of delivery. In late or term FGR, conventional CTG evaluates variability and decelerations, with closer monitoring when Doppler abnormalities or reduced fetal movements occur.

## Maternal factors

- Key maternal contributors include hypertensive disorders, renal disease, diabetes with vascular

disease, autoimmune conditions (APS/SLE), anemia, malnutrition, infections, smoking, and substance use. Extremes of maternal age also increase risk. Worsening maternal disease dictates surveillance intensity and timing of delivery, independent of fetal findings.

## Growth charts

- Fetal biometry charts should be prospectively derived, truly population-based, and methodologically robust.[8] INTERGROWTH-21st was a multicentre, multi-ethnic population-based study (2009–2014) across eight sites, including India. WHO charts were developed from a multinational longitudinal study of low-risk singleton pregnancies in 10 countries.[9]

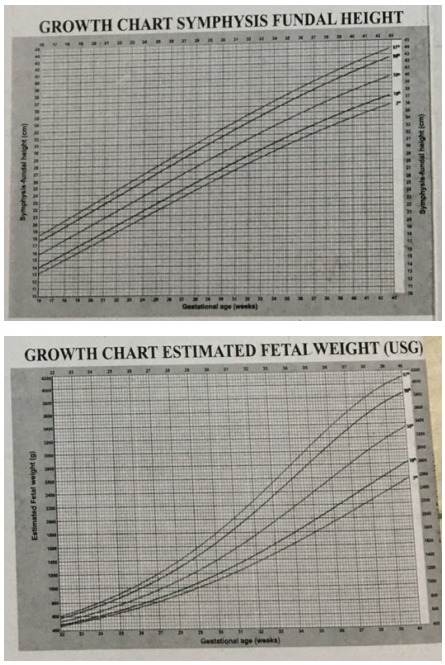



**Figure 2:** Intergrowth 21 SFH & EFW charts

- GROW 2.0 is a customized fetal growth assessment tool developed by Gardosi (Perinatal Institute, UK) that adjusts fetal and birthweight centiles for maternal height, weight/BMI, parity, ethnicity, and fetal sex, aiming to differentiate constitutional smallness from pathological FGR and improve detection of at-risk fetuses.[6]

## Symphysis–fundal height

- Symphysis–fundal height is a simple screening tool for fetal growth after 24 weeks. Serial measurements plotted on a chart help detect suspected FGR; a ≥3 cm lag or static/falling SFH prompts ultrasound evaluation. It is not diagnostic and is less reliable with obesity, fibroids, multiple pregnancy, or abnormal liquor.

## Risk prediction models for FGR

These models combine maternal factors, obstetric history, biomarkers, blood pressure, uterine artery Doppler, and ultrasound findings to estimate FGR risk. They improve early risk stratification, especially for early-onset FGR, but have limited accuracy for late-onset disease and are used to guide surveillance, not diagnosis.

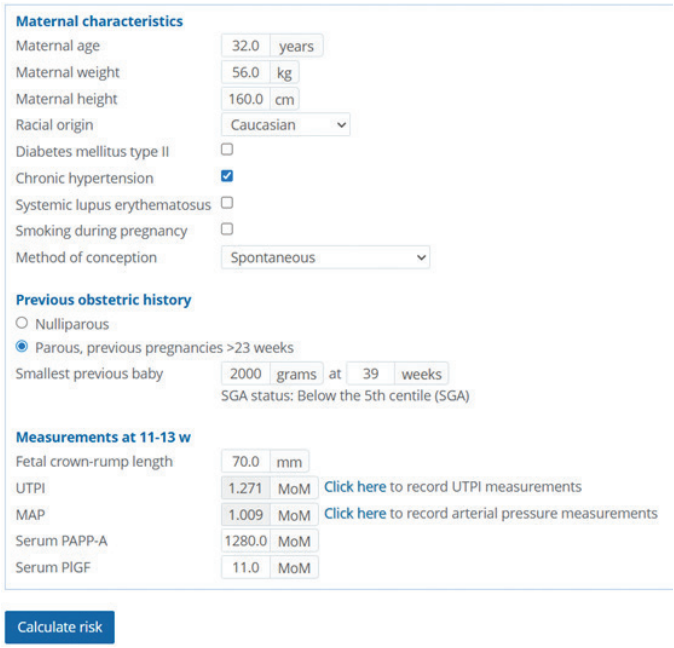



**Figure 3:** Fetal Medicine Foundation (FMF) First-Trimester Risk Prediction Model for Fetal Growth Restriction (SGA)

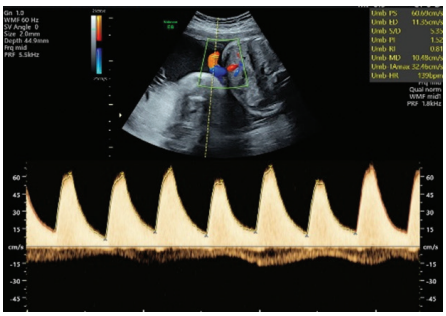
## Management

### Early ONSET FGR

- Early FGR should be managed in tertiary centres with highest-level neonatal care; care must be multidisciplinary (fetal medicine obstetricians + neonatologists), especially <28 weeks .
- For very preterm/severe FGR, counselling by experienced obstetrician and neonatologist; decisions based on gestation and fetal weight.
- Biometry: Repeat every 2 weeks; more frequent scans increase false-positive FGR diagnosis (1– B).
- **Fetal surveillance:**
  - At least weekly if stable.
  - 2–3 times/week if abnormal umbilical artery (UA) Doppler .
  - Must include cCTG (Dawes–Redman; STV key) and/or ductus venosus (DV) Doppler.
- Key predictors of deterioration: Raised DV PIV and low cCTG STV—most discriminatory for timing of birth (TRUFFLE data).
- Role of MCA: CPR/UCR may guide monitoring frequency, but should not determine delivery before 37 weeks (GPP).

- Reduced fetal movements: Maternal concern or reduced/absent movements on scan → prompt cCTG (GPP).
- Timing of birth (based on fetal wellbeing or maternal indication):
    - o <26 weeks: Individualised care (no RCT evidence).
    - o 26+0–28+6 weeks: Deliver if DV a-wave at/below baseline or STV <2.6 ms.
    - o 29+0–31+6 weeks: Deliver if DV a-wave at/below baseline or STV <3.0 ms.
    - o 32+0–33+6 weeks (consider ≥30 weeks): Deliver if UA reversed EDF or STV <3.5 ms.
    - o ≥34 weeks (consider ≥32 weeks): Deliver if UA absent EDF or STV <4.5 ms.
- Evidence base: TRUFFLE showed best 2-year neurodevelopmental outcomes (≈95%) when delivery was timed using late DV changes/cCTG safety net; overall perinatal mortality ≈8%, cerebral palsy ≈1%.[4]

A)



B)

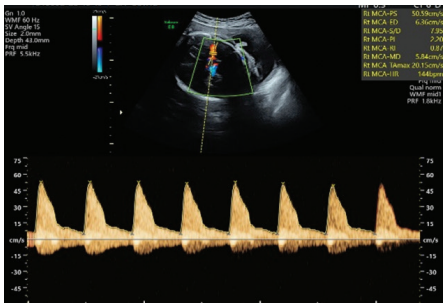
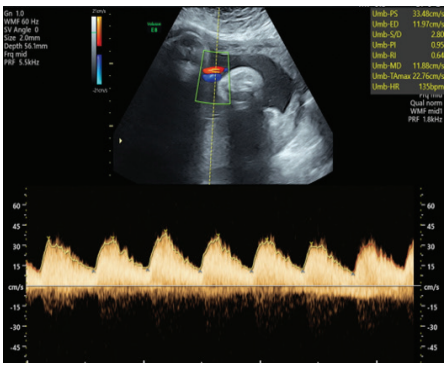


**Figure 4:** Case of patient with early onset doppler A) umblical artery doppler ( 1.52 - >95TH centile) and B) middle cerebral artery doppler (2.20 - 85TH centile). The patient was followed up with weekly dopplers till term.

A)



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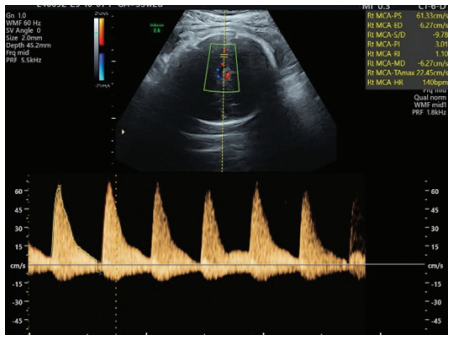

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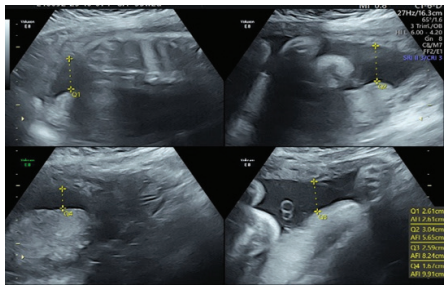

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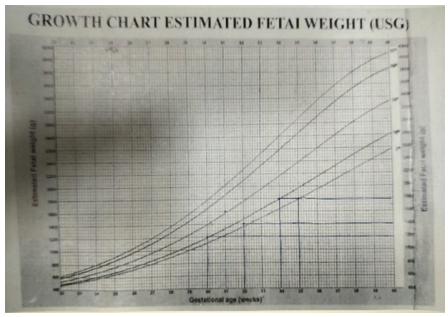



**Figure 5:** Case of patient with early onset doppler A) umblical artery doppler B) middle cerebral artery doppler C) Amniotic fluid index. D) Showing growth velocity of the fetus on subsequent scans.

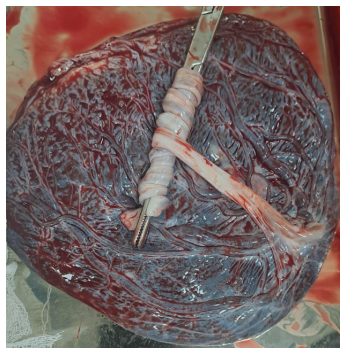


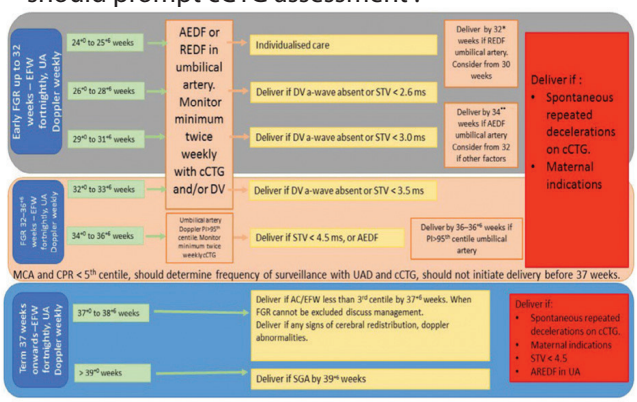
**Figure 6:** The patient was induced at 37 weeks due to bleeding per vaginum. At delivery, the placenta showed marginal cord insertion, which likely explains the early-onset fetal growth restriction.

## Late and Term FGR

- Role of ductus venosus: DV assessment is not informative in late or term FGR and is recommended only for early-onset FGR (GPP).
- In late FGR, timing of birth should be guided by fetal well being or maternal indications (e.g. severe pre-

eclampsia).

- Indications for delivery (fetal):
  - Recurrent, spontaneous, persistent unprovoked FHR decelerations.
  - cCTG STV <3.5 ms at 32+0–33+6 weeks, or <4.5 ms at ≥34+0 weeks.
  - Umbilical artery Doppler:
    - Absent EDF (consider at 32 weeks; mandatory by 34 weeks).
    - UA PI >95th centile at 36+0–36+6 weeks (GPP).
- Normal UA Doppler Does not exclude placental dysfunction, hence additional surveillance is required.
- All FGR pregnancies should be delivered from 37+0 weeks and completed by 37+6 weeks (1+ A). Fetuses <3rd centile have the highest stillbirth risk beyond term.
- Cerebral Doppler: Abnormal MCA/CPR/UCR may guide monitoring frequency, but should not determine delivery before 37 weeks. After 37+0 weeks, abnormal values can inform timing of birth; normal values do not provide reassurance (2++ D).
- Maternal perception of reduced or absent movements should prompt cCTG assessment .




*Consider after $30^{+0}$ weeks; **Consider after $32^{+0}$ weeks; EFW, estimated fetal weight; UA, umbilical artery; DV, ductus venosus; cCTG, computerised cardiotocograph; STV, short-term variation; ms, milliseconds; AC, abdominal circumference; PI, pulsatility index; AREDF, absent reversed end-diastolic flow.

**Figure 7:** Algorithm for Investigation and Management of Fetal Growth Restriction (Adapted from RCOG Green-top Guideline No. 31, 2024)[1]

- Antenatal corticosteroids should be offered to women at risk of preterm birth between 24+0 and 34+6 weeks' gestation, ideally administered at least 48 hours before anticipated delivery, to reduce neonatal morbidity and mortality.
- Magnesium sulphate for neuroprotection should be offered at 24+0–29+6 weeks and considered up to 33+6 weeks in women at risk of preterm birth, as it reduces the risk of cerebral palsy in preterm infants.

## Outcome

Prior to 27 weeks, **gestational age is the strongest determinant of neonatal outcome** in growth-restricted infants delivered before 33 weeks, with increased risks of hypoxia and meconium aspiration. Severely FGR neonates are highly susceptible to hypothermia, hypoglycaemia, polycythaemia and hyperviscosity, particularly at extreme low birthweights. SGA infants have higher mortality (≈1% vs 0.2%), doubled stillbirth risk, increased neurodevelopmental impairment, and markedly higher risks of stillbirth, neonatal death, and major morbidities—especially when FGR is diagnosed before 32 weeks.

### Postnatal counselling after FGR:

Postnatal counselling should review the FGR diagnosis, neonatal outcome, and **placental histology** to clarify cause and recurrence risk. Evaluation for **underlying conditions**, including acquired thrombophilias where appropriate, should be considered. Counselling should focus on **modifiable risk factors** (smoking, BMI, comorbidities) and include a clear **plan for future pregnancies**, such as preconception optimisation, consideration of low-dose aspirin, and tailored surveillance.

## Prevention

Prevention begins before conception. Currently, no pharmacologic therapies prevent growth restriction.

- Optimize control of chronic conditions (hypertension, diabetes, renal disease, SLE/APS).
- Encourage smoking cessation (CO testing with opt-out referral to cessation services).
- Start low-dose aspirin 150 mg at bed time from 12+0 to 36+6 weeks in women at risk of placental disease.
- Ensure folic acid (400 µg) and vitamin D at recommended doses (higher doses show no added benefit).[1]

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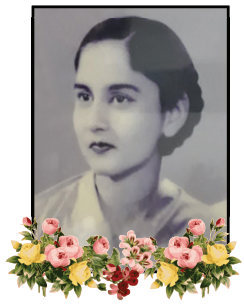
## Heartfelt Homage



### Dr Sarla Mukherjee 1934-2025

**An Accomplished Obstetrician & Gynecologist &**
**Senior Member of AOGD**

**Dr. Sarla Mukherjee (1934–2025), an accomplished Obstetrician and Gynecologist, Senior Member of AOGD, and former Head of the Department, MAMC.**
**Dr. Sarla Mukherjee was a distinguished clinician, teacher, and mentor whose lifelong dedication to women's health and medical education touched countless lives. Her professionalism, integrity, and compassion will always be remembered by colleagues, students, and patients alike.**

**She left for her heavenly abode on 23rd November 2025 in peace and tranquility. Though she has left us, her presence is always felt and her memories will remain in our heart and minds forever .**

# Management Dilemma in Spontaneous Intrauterine Demise in Twins

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## Introduction

Twin pregnancies are associated with increased complications. One of the most challenging scenarios is the spontaneous intrauterine demise (IUD) of one twin—a situation that raises difficult clinical, ethical, and legal issues. Reported in up to 2.6%–6.8% of twin gestations[1], the outcome after fetal loss depends largely on chorionicity and gestational age. Single IUFD in the first trimester may occur in up to 50% and in the second or third trimesters in 0.5% to 6.8% of twin pregnancies. It is estimated that there is a threefold to fourfold increase in intrauterine death with monochorionic twins as compared with dichorionic twins. In addition, a single fetal death is also more common among twins with a structural abnormality.

In India, where clinical resources and legal guidelines vary widely, hence managing such cases is a challenge and requires careful attention to communication and documentation. A multidisciplinary approach may be required particularly in cases of monochorionicity.

## Clinical Background

### Causes

- The etiology of intrauterine fetal demise (IUFD) in a multiple pregnancy may be similar to singletons or unique to the twinning process.
- IUFD may be due to genetic or anatomical anomalies, abruption, placental insufficiency, cord abnormalities such as a velamentous cord insertion, infection, and maternal disease such as diabetes and hypertension (as seen in singleton pregnancies). If the fetal death is due to maternal medical disease like diabetes, placental disorders (Preeclampsia, abruption or placental insufficiency there is high likelihood of surviving co-twin for adverse outcomes.
- In monochorionic pregnancies, IUFD may result from complications of the twin-to-twin transfusion syndrome (TTTS), selective fetal growth restriction (sFGR), twin anemia polycythemia syndrome (TAPS) and twin reversed arterial perfusion sequence (TRAPS)
- In monoamniotic twins there is increased risk of cord entanglement and subsequent IUFD.

### Chorionicity

- Dichorionic Twins: Each fetus has a separate placenta. Here, the demise of one twin may pose fewer risks to the surviving twin, though complications like infection or coagulopathy in the mother remain a concern, though rarely seen.
- **Monochorionic Twins:** Sharing a common placenta via vascular anastomoses, these pregnancies are at higher risk. The sudden loss of one twin can lead to rapid hemodynamic changes that may cause cerebral injury (25%) or even death (10%) of the surviving co-twin.[2] There is sudden shunting of blood from surviving twin to demised twin, leading to profound hypotension in the survivor. It is believed that blood from the surviving twin may rapidly "back-bleed" into the demised twin through placental anastomoses, a form of acute feto-fetal transfusion. The demised twin may become congested while the surviving twin may become anemic. Multicystic encephalomalacia is the cystic lesion in the cerebral white matter distributed in areas supplied by the anterior and middle cerebral arteries which is associated with profound neurologic handicap. This risk following single IUFD in a monochorionic pregnancy may be as high as 20-25% for the surviving co-twin. If the hypotension is significant, the surviving twin is at risk for ischemic damage to vital organs. Since the damage has already occurred at the time of demise. There is no benefit of immediate delivery once demise of co-twin has occurred but rather adds to the risk of prematurity. Incidence of neurological damage is very low in dichorionic twins.

**Gestational Age:** The timing of loss is another critical determinant of outcome

- First Trimester: Most multiple gestations diagnosed in the first trimester undergo spontaneous reduction of one sac in the first trimester, referred to as the "vanishing twin." Demise in first trimester has limited impact on the surviving fetus. Biochemical screening in vanishing twin pregnancies is often misleading due to residual trophoblastic activity from the demised twin. Serum biochemistry and NIPS can yield false-positive aneuploidy risks, prompting undue interventions. Nuchal translucency and other ultrasound markers are more reliable.[3] (Discussed in other section in detail)
- Second and Third Trimesters: Associated with increased risks such as preterm labor, fetal growth restriction, neurological damage or demise of surviving co-twin and theoretical risk of maternal complications including disseminated intravascular coagulation

(2%).[4] IUFD of one twin can result in preterm delivery in both monochorionic and dichorionic pregnancies with double the background incidence of reported preterm delivery in twins.

**Table 1.** illustrates the Risks of Single Fetal Demise in Twin Pregnancy

| Risk | Dichorionic Twin | Monochorionic Twin |
|---|---|---|
| Co-Twin Death | 3% | 15% |
| Preterm Birth | 54% | 68% |
| Abnormal postnatal cranial imaging | 16% | 34% |
| Neurodevelopmental impairment | 2% | 26% |

Table 1- Risks of Single Fetal Demise in Twin Pregnancy.[5]

## Diagnostic Approach

1. Ultrasound Imaging: The sonographic findings in single IUFD in twin pregnancy depends on the interval between the death and performance of the ultrasound examination. Sonographic assessment should include complete biometric and anatomic assessments of the dead and surviving twins, an assessment of amniotic fluid volume, evaluation of the cord insertion sites and doppler assessment of umbilical artery, middle cerebral artery and ductus venosus. Determination of chorionicity is attempted if not assigned previously, however, it may be a challenge to determine it after 16 weeks. If the fetal death is due to maternal medical disease like diabetes, placental disorders (Preeclampsia, abruption or placental insufficiency there is high likelihood of surviving co-twin for adverse outcomes. Sonographic examination may reveal maternal causes or placental abnormalities like abruption as a causative factor.
2. MRI Fetal Brain Imaging: In monochorionic twin pregnancies, single fetal demise can lead to severe neurological morbidity in the surviving twin due to acute hemodynamic shifts through shared placental vessels. This can result in cerebral palsy, periventricular leukomalacia, or multicystic encephalomalacia, with reported risks ranging from 18–34%. Fetal MRI performed 2–3 weeks after the event is the preferred modality for detecting early cerebral injury. Dichorionic twins are less affected due to independent placental circulations.[5]
3. Laboratory Evaluation:
    - Maternal coagulation profile
    - Kleihauer-Betke test in Rh-negative mothers[6]

## Preventive Strategies

- Early detection of chorionicity
- Maternal Fetal Medicine referral particularly in monochorionic twins
- 2 weekly antenatal visits and proper ultrasound evaluation
- Optimising maternal medical disease
- Aspirin to prevent Hypertensive Disorders of pregnancy
- Screening and management of maternal diabetes
- Screening of TTTS, TAPS, TRAPS, sFGR. Twin-twin transfusion syndrome has significant mortality (>70%) if left untreated, particularly if diagnosed in the second trimester. Laser coagulation is the treatment of choice for stages II–IV between 16 and 24 weeks of gestation.
- There is no role of intrauterine transfusion of surviving twin in single fetal demise after TTTS in monochorionic twins

## Management Strategies

- Referral to a tertiary care perinatal unit is advised when single IUFD in multiple gestation is diagnosed.
- Clinical management depends on chorionicity, gestational age, fetal lung maturity, detection of in utero compromise of the surviving twin and maternal disease.
- The goal is to optimize outcome for the surviving twin while avoiding prematurity and its potential adverse sequelae.

## Maternal Monitoring

- Baseline maternal hematological laboratory results are obtained including a prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen level, and platelet count due to theoretical risk of DIC.
- IUFD in singleton pregnancies may cause psychological distress. The psychological impact of fetal loss in multiple pregnancies has not been well studied. As a result, patients with a multiple pregnancy complicated by IUFD must be offered psychological and/or bereavement counseling.

## Dichorionic twins with single fetal demise

- The patient is managed expectantly until 37 weeks of gestation if pregnancy is complicated by IUFD prior to viability
- Fetal surveillance and a single course of antenatal steroids may be considered once viability is achieved in cases where preterm labor commences or preterm termination of pregnancy is indicated

## Monochorionic twins with single fetal demise

- Monochorionic pregnancies complicated by IUFD are challenging. Patients are counselled regarding the risk of multiorgan injury including multicystic encephalomalacia, but no interventions are

available to impact on this risk. It is difficult to predict in utero which cases of surviving monochorionic twin will have cerebral injury. MRI is scheduled 2 to 3 weeks after the diagnosis of IUFD in a monochorionic pregnancy. There is no role of intrauterine transfusion of packed cells to surviving twin for prevention of this complication.

- A single course of antenatal corticosteroids is administered if delivery is anticipated within 7 days and the gestational age is between 24 and 34 completed weeks.
- In pregnancies complicated by IUFD once viability has been achieved
    - Nonstress test and the biophysical profile give insight into the physiological status of the fetus as reflected in the autonomic nervous system.
    - Elective delivery is scheduled at approximately 37weeks of gestation
    - Vaginal delivery is not contraindicated, and cesarean delivery is reserved for routine obstetric indications
- At delivery, umbilical cord blood gas and hematocrit measurements are performed.
- Fetal autopsy and placental examination is offered.
- Postnatal follow up of neonates of monochorionic twins for signs or symptoms of cerebral impairment or any other vital organ damage.
- Postnatal assessment of the surviving twin includes neuroimaging and close developmental monitoring. A multidisciplinary team approach involving maternal-fetal medicine, neonatology, and radiology is essential for optimal outcomes.[7]

## Fetal Intervention to prevent SIUF demise in monochorionic twins

- In known monochorionic pregnancies with impending death of one twin, preterm delivery may be indicated in order to prevent neurologic injury to the surviving co-twin.
- The risk of neurologic handicap to the surviving twin may be higher than the risk of prematurity.
- If chorionicity is unclear and the death of one twin seems imminent, it is necessary to weigh the risks and benefits to each twin of expectant management versus early delivery.
- If dichorionicity is clearly demonstrated, there is no benefit to the healthy, appropriately grown co-twin of elective preterm delivery in cases in which the smaller twin is likely to die.
- Indications for considering a fetoscopic cord ligation procedure to prevent neurologic handicap in monochorionic twins include anomalies incompatible with life or significant growth discordance at a previable gestational age.

## Counseling, Informed Consent and Psychological Support

- Collaborative Approach: Multidisciplinary counseling is essential, involving obstetricians, neonatologists, legal advisors, and mental health professionals ensures comprehensive support.
- Transparent Communication: Clear information about risks and outcomes helps parents make informed decisions
    - Risk Disclosure: Parents should be informed about potential outcomes such as cerebral palsy, intrauterine growth restriction (IUGR), or stillbirth of the surviving twin [8]
    - Limits of Prediction: While ultrasound and MRI provide insight, they do not guarantee long-term outcomes.
- Emotional Support:
    - Counseling should be culturally sensitive and, if possible, provided in the family's native language. Additional psychological support should be made available.
    - Support Networks: Establishing access to counseling services and support groups is important for emotional well-being.

## Balancing Maternal Autonomy and Fetal Rights

Shared decision-making is crucial in situations where continuing the pregnancy might harm the surviving twin, even if maternal health is stable as decisions are ethically complex and familial opinions often influence outcomes.

## Judicial Rulings

Recent Indian judgments stress:

- Expert Opinions: Courts often require multidisciplinary medical board reviews. Documentation: Detailed records of decision-making help protect against legal actions [9].

### Legal Case Scenarios in India

**Case 1:** Bombay High Court – 2023

**Scenario:**

A 29-year-old woman with a monochorionic twin pregnancy at 27 weeks experienced the demise of one fetus, while the surviving twin exhibited early neurological distress. The parents requested immediate delivery.

**Outcome**

After a writ petition, the Bombay High Court upheld

expectant management based on expert multidisciplinary review and mandated rigorous documentation and surveillance.[9]

### Implication

Adhering to institutional protocols and thorough documentation helps defend clinical decisions in court.

**Case 2:** Composite Analysis from Supreme Court Judgments

### Scenario

A rural hospital managed a twin pregnancy where one fetus demised at 30 weeks. Due to insufficient counseling and delayed referral, the surviving twin was delivered at 34 weeks and later diagnosed with severe cerebral palsy. A negligence suit ensued.

### Outcome

The Supreme Court partially favored the family, citing inadequate communication and documentation. This ruling highlights the importance of clear counseling and timely referrals.[10]

### Implication

Comprehensive communication and timely referrals are essential to prevent legal repercussions, even when clinical management aligns with standard protocols.

## Recommendations for Practice in India

1. Early Determination of Chorionicity:
    - Establish chorionicity early (preferably in the first trimester) through ultrasound to stratify risks.
2. Develop Clear Protocols and Referral Pathways:
    - Create guidelines for timely referral to tertiary centers with advanced imaging and NICU facilities.
3. Form Multidisciplinary Medical Boards:
    - Include obstetricians, neonatologists, radiologists, and legal experts in complex decision-making. Detailed documentation of discussions is critical.
4. Ensure Prompt Legal and Ethical Reviews:
    - Expedite review processes in cases involving selective termination to avoid delays that could worsen outcomes.
5. Enhance Documentation and Communication:
    - Maintain meticulous records of all clinical discussions and decisions to support continuity of care and legal defense.
6. Prioritize Parental Counseling and Support:
    - Provide culturally sensitive and comprehensive counseling along with continuous psychological support.

## Conclusion

Spontaneous intrauterine demise in twin pregnancies poses significant challenges in India due to resource limitations and evolving legal frameworks. Clinicians must balance the benefits of continued gestation against potential risks to the surviving twin and maternal health. Equally important is the need for clear communication and thorough documentation to support clinical decisions and safeguard against legal repercussions.

The legal case scenarios illustrate the crucial role of multidisciplinary consultation, prompt decision-making, and comprehensive documentation. National guidelines from bodies such as the Federation of Obstetric and Gynaecological Societies of India (FOGSI) or the Indian Council of Medical Research (ICMR) would help standardize care and improve both maternal and fetal outcomes.

By adopting structured protocols, ensuring robust counseling, and facilitating timely referrals, healthcare providers can effectively navigate this complex landscape, ensuring compassionate care and legal defensibility.

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# Intrapartum Ultrasound: Redefing Labour Ward Decision

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## Introduction

Intrapartum ultrasound provides objective, real-time assessment of fetal position, descent, and labor progress, overcoming limitations of digital vaginal examination , which is subjective and less reliable with caput, molding, asynclitism, or malpositions. With rising cesarean and instrumental delivery rates, accurate intrapartum evaluation is increasingly important.[1] Ultrasound improves detection of malpositions, refines assessment of station, and enhances decision-making during second-stage and operative deliveries, reducing failed instrumental attempts and unnecessary cesareans. .International bodies such as ISUOG recommend its use when digital vaginal examination findings are uncertain or operative delivery is planned. However, ultrasound remains complementary; a clinically indicated digital vaginal examination must still be performed when required to assess cervical dilation and membrane status.[1]

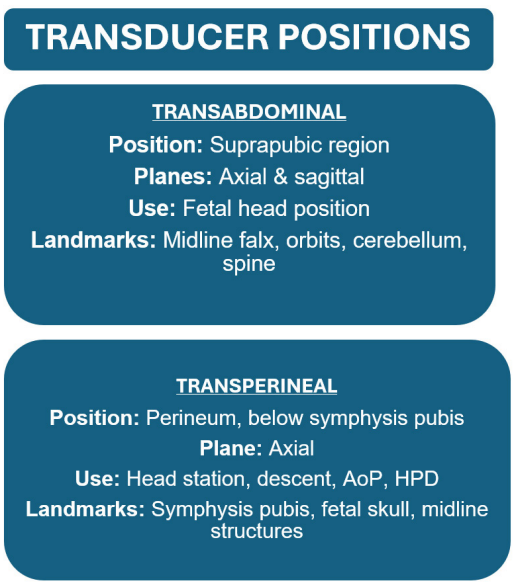


**Figure 1:** Overview of transducer position during intrapartum ultrasound scanning and identifiable landmarks

## Assessment of Labour Parameters

## 1. Assessment of Fetal Head Position

Fetal head position—defined by occiput orientation—critically influences labor mechanics. Persistent OP or transverse positions prolong labor and increase operative delivery, maternal morbidity, and neonatal complications. Misidentifying position before operative vaginal delivery raises the risk of failed instrumentation, perineal trauma, and conversion to cesarean section.[1]

Digital palpation of sutures and fontanelles remains the traditional method but is limited by low accuracy. Studies demonstrate error rates ranging from 20% to 70%, with poor reproducibility even among experienced clinicians, particularly in OP, OT, and deeply engaged heads affected by caput or molding.[1,2]

## Ultrasound approach

### Transabdominal ultrasound

Transabdominal ultrasound is considered the gold standard for intrapartum determination of occiput position.[2] The maternal position for transabdominal scanning is supine with a left lateral tilt.

### Technique

- The fetal spine is identified in an axial abdominal view.
- The probe is moved caudally to obtain an intracranial axial image.
- Recognition of structures:
  - OP (Occipito posterior) positions: the fetal orbits appear first.
  - OT (Occipito transverse) positions: the midline echo lies horizontally.
  - OA (Occipito anterior) positions: the occiput and spine appear in the anterior field.

## Occiput classification

Positions are defined clockwise as: OA position between 10 and 2 o'clock; OP between 4 and 8 o'clock; LOT (left occiput transverse)between 2 and 4 o'clock and ROT (right occiput transverse) between 8 and 10 o'clock.as shown in figure 2.[2]

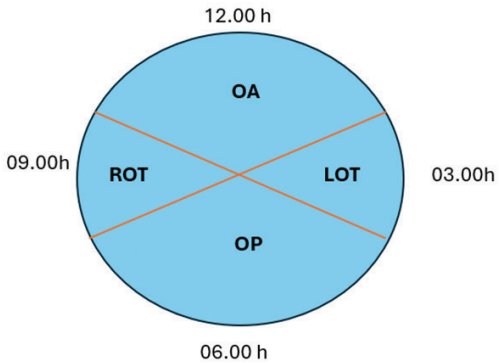


**Figure 2:** Classification of fetal occiput position based on positions of hour hand on a clock face

## Transperineal ultrasound

Transperineal imaging offers similar accuracy and is particularly advantageous at low head stations, avoiding pelvic shadowing.[2] It provides fetal head images to calculate a proposed fetal head station.It is done with mother in semi-recumbent position with legs flexed at the hips and empty bladder.The transducer is placed between the labia majora or at the level of the posterior fourchette.

## Key markers

- Axial transperineal view: the choroid plexus consistently points toward the occiput.
- The occipital bone produces a broader contour than the frontal bone.
- Cranial molding patterns provide positional clues[3]:
  - Occipitoparietal molding → OA
  - Frontoparietal molding → OP

## 2. Assessment of Fetal Head Station

Fetal station, representing the relationship between the presenting part and ischial spines, is traditionally assessed per vaginally. However, digital vaginal examination is highly subjective, influenced by caput, molding, intact membranes, high stations, asynclitism, and fetal malposition.[1]

## Ultrasound-based assessment

Transperineal ultrasound provides objective and reproducible measurements. In a midsagittal view, the pubic symphysis and fetal skull are visualized, and fetal descent is inferred relative to the pubic bone axis. Although ischial spines are not directly imaged, CT correlations show that the interischial plane is approximately 3 cm caudal to the infrapubic line.[2]

### 2.1 Angle of Progression (AoP)

The AoP is the most widely validated sonographic measure of head station.

It is the angle between the long axis of the pubic symphysis, and a line from the lowest border of the pubis tangentially touching the deepest bony part of the fetal skull as shown in figure 3.[2]

## Key thresholds

- **120°** ≈ station **0**
- **130°** ≈ station **+1**
- **140°** ≈ station **+2**

The ΔAoP, the difference between AoP during maternal rest and pushing, predicts the likelihood of spontaneous delivery and progression.[3]

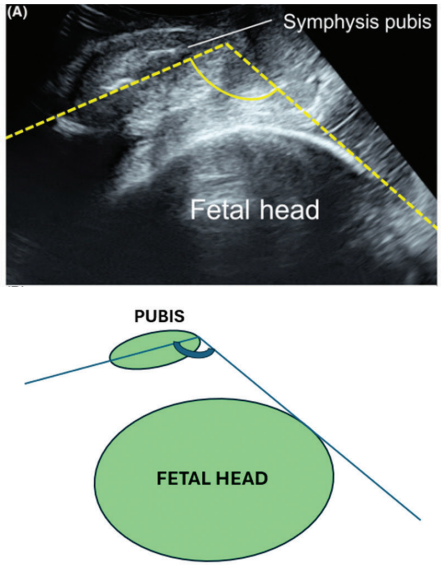


**Figure 3:** Angle of progression: (A) ultrasound image (B) schematic diagram

Adapted from: Tang H et al. Int J Gynecol Obstet. 2021;156:1–7.[16]

### 2.2 Head–Perineum Distance (HPD)

Measured in a frontal transperineal plane from the outer fetal skull to perineum.(Figure 4)

The compressed HPD involves full compression of the soft tissues of the perineum against the pubic bone. The transducer should be angled until the fetal skull contour is as clear as possible.The advantage of HPD for obstetrician is that it is quick and easy measurement particularly useful in emergency situation such as before an operative vaginal delivery.The major limitation being difficulty of standardize the operator pressure on the maternal soft tissue.

A compressed HPD of 36 mm, 31 mm and 25 mm correspond to head stations of 0, +1, and +2, respectively.

The uncompressed HPD does not involve any type of compression of the perineum

An uncompressed HPD of 60 mm, 50 mm, and 40 mm correspond to head stations of 0, +1, and +2, respectively.[3,4]

The ΔHPD may be also assessed as the difference in HPD during rest and maternal pushing efforts.[5]

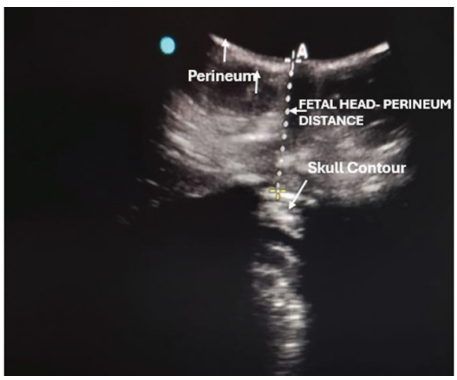


**Figure 4:** USG image showing head to perineum distance

### 2.3 Fetal Head Direction

Based on the angle between the axis of the fetal head and the pubic symphysis:

- **Head down (<0°)** → pelvic inlet
- **Horizontal (0–30°)** → mid-pelvis
- **Head up (>30°)** → beyond pubic symphysis (outlet). An upwards head direction ("head up sign") indicates a favorable station before operative vaginal delivery[2]

### 1.4 Midline Angle (MLA)

Measured in the axial transperineal plane between the fetal midline cerebral echo, and the maternal anteroposterior axis (Figure 5). It reflects **rotational progress**, not descent.

In one study, an MLA ≥45° corresponded to a head station of ≤+2 in 98.6% of cases, whereas an MLA <45° corresponded to a station of ≥+3 in 83.7% of cases.[6]

### 2.5 Progression Distance (PD)

Shortest distance between infrapubic line and the presenting part. While reproducible, it does not account for the curved birth canal therefore AoP is preferred.

## Limitation

Most normative data for sonographic station are based on OA fetuses. OP fetuses follow a different descent pathway—deeper before extension—limiting applicability of OA-derived thresholds.[6]

## 3. Assessment of Fetal Head Attitude

Fetal attitude describes the degree of flexion or deflexion of the head, influencing both descent and engagement.

### 3.1 Occiput–Spine Angle (OSA)

Used for visualizing the degree of flexion in OA and OT fetuses. An OSA ≥ 109° indicates good flexion. (Figure 5) Deflexion predicts prolonged labor and increased operative delivery.[7]

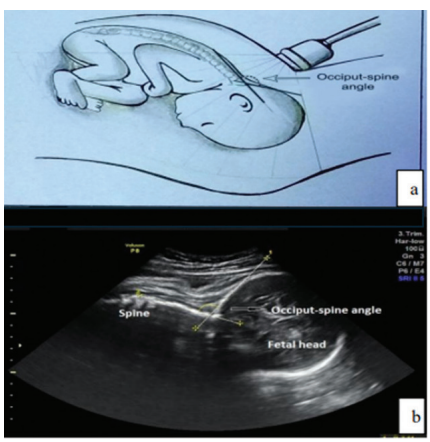


**Figure 5:** (A) Schematic representation and (B) Transabdominal ultrasound image of measurement of Occiput-spine angle.

**Adapted from:** Somu K, Sujatha S, Hebbar S, Guruvare S, Pai M. Int J Reprod Contracept Obstet Gynecol. 2019.[17]

### 3.2 Chin-to-Chest Angle (CCA)

- It is used to assess flexion in OP fetus in the sagittal plane.
- This angle is formed by lines tangential to the longest axis of the fetal sternum and the skin covering the inferior part of the fetal chin (Figure 6)
- CCA <35° indicates good flexion. Larger angles indicate deflexion, increasing rates of dystocia and cesarean section.[15]

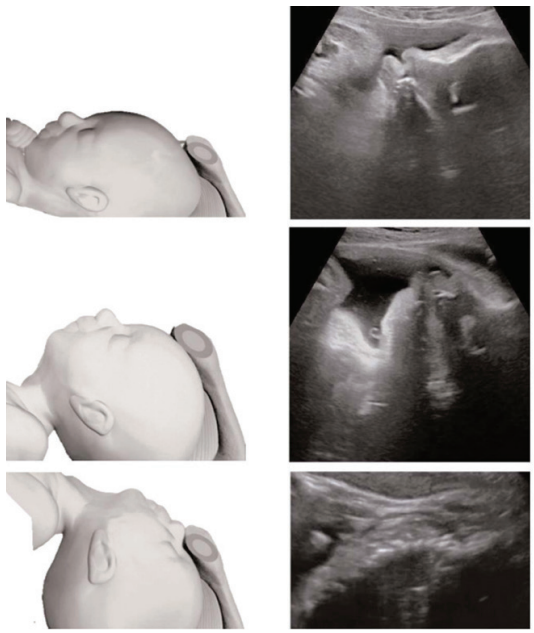

**Figure 6:** Visual assessment of the "chin-to-chest" angle (CCA) in fetuses with occiput posterior position.

**Adapted from:** Rizzo G, Ghi T, Lees C, Henrich W, Mappa I, Tutschek B, et al. Perinatal Journal. 2022;30(2):103–127.[15]

## 4. Asynclitism

Asynclitism refers to lateral deviation of the sagittal suture from the midline of the maternal pelvis, causing one parietal bone to present first

## Ultrasound markers

- On axial transperineal imaging: inability to view the fetal midline echo perpendicularly.
- **Probe adjustments**:
  - Tilting posteriorly → identifies anterior asynclitism.
  - Tilting anteriorly → identifies posterior asynclitism.[8]
- **Lateral asynclitism**: Simultaneous visualization of the four-chamber heart view and facial profile in a single transabdominal plane.

Asynclitism is often underdiagnosed although it could be as common as 15% in second stage of labor amongst nulliparous women.A simple rule for diagnosis is that the midline echo will not be seen easily at the head-perineum

distance plane.Obstetricians must be aware of this condition, especially when encountering fetuses in non OA position before attempting instrumental deliveries.[9]

## Indications for Intrapartum Ultrasound

### 1. Protracted or Arrested First Stage

In OA/OT positions, fetal head flexion should be assessed using the OSA; OSA <109° indicates malpresentation and increases risks of prolonged labor and cesarean delivery, whereas OSA ≥109° favors vaginal birth. Persistent high station—HPD >50 mm or AoP <100°, also predicts prolonged labor and cesarean section. OP position further raises cesarean risk, especially when head deflexion is present (CCA >35°), which ultrasound identifies reliably.[10,11]

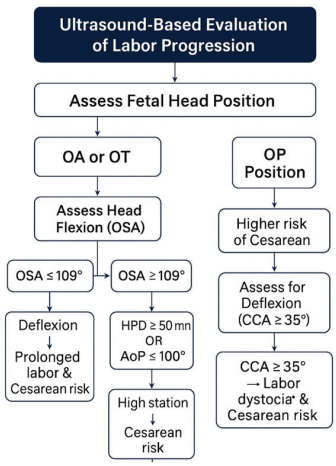


**Figure 7:** Flow chart showing USG based evaluation of fetal head in first stage of labour.

## 2. Arrest of Second Stage

- Ultrasound can help identify fetuses likely to achieve spontaneous delivery, making expectant management a reasonable option.
- Favorable indicators include OA position and good fetal head descent – AoP ≥120°, ΔAoP ≥15°, "head up" direction, and a midline angle <35° – all of which correlate with higher rates of spontaneous vaginal delivery and shorter second stage duration.[12]

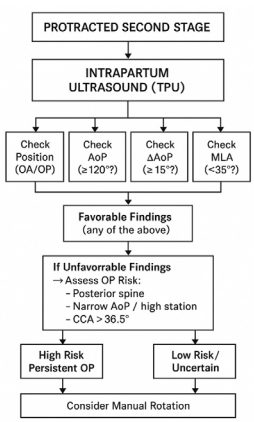


**Figure 8:** Flow chart showing USG based evaluation of fetal head in second stage of labour

## 3. Planning Instrumental Delivery

Decision-making between operative vaginal delivery and cesarean section can be guided by intrapartum ultrasound assessment of fetal head station and related parameters.

In low-cavity situations (station ≥ +2, angle of progression >140°, compressed head–perineum distance <25 mm, and uncompressed head–perineum distance <40 mm), operative vaginal delivery is appropriate.

In mid-cavity positions (station 0 to +1, angle of progression 120–140°, compressed head–perineum distance 25–36 mm, and uncompressed head–perineum distance 40–60 mm), fetal head position and dynamic changes should be assessed. When the head is in an occiput anterior position, operative vaginal delivery is favored if there is adequate descent, defined by an increase in angle of progression of at least 15° and a reduction in head–perineum distance of at least 2 mm; inadequate change supports cesarean section. In occiput posterior positions, the presence of risk factors—such as estimated fetal weight above 4500 g, suspicion of cephalopelvic disproportion, or significant asynclitism or deflexion indicates cesarean section.

High-cavity findings (station ≤ 0, angle of progression <120°, compressed head–perineum distance >36 mm, and uncompressed head–perineum distance >60 mm) favor cesarean section.[2]

## Future Perspectives of Intrapartum Ultrasound

Intrapartum ultrasound is expected to evolve into an essential component of labor monitoring.

### 1. Standardized Protocols

Development of uniform assessment criteria for position, descent, rotation, and asynclitism will:

- Improve reproducibility
- Facilitate training
- Enable cross-center comparisons[1]

### 2. Sonopartogram

The sonopartogram represents a digital, ultrasound-based labor chart replacing subjective clinical assessments. It provides:

- Real-time tracking of descent, rotation, and flexion
- Objective documentation of labor progression[13]

### 3. Artificial Intelligence (AI) Integration

AI and machine learning may:

- Automatically recognize fetal landmarks
- Predict labor outcomes
- Assist in timing of operative delivery[2]

**4. Emerging Imaging Technologies**

- 3D/4D ultrasound will improve visualization of molding, malpositions, and soft-tissue dynamics, enhancing operative planning .
- Portable wireless probes will increase accessibility, particularly in low-resource settings.[14]

## Conclusion

Intrapartum ultrasound provides objective, reproducible assessment of fetal position, descent, rotation, and attitude, enhancing accuracy beyond digital examination. Its integration improves diagnosis of labor abnormalities, guides operative delivery, and reduces maternal–neonatal morbidity. With standardization, AI integration, and training, ultrasound will become an essential component of modern labor management.

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## Forthcoming Events

- 03.01.2026 - Awareness Session on Cyber Security and Public Health will be conducted by Community Health and public awareness SubCommitte in association with Delhi Paramedical & Management Institute (DPMI) is organizing at DPMI Campus, New Ashok Nagar.
- Nominations for the Chairperson Medico-legal subcommittee of AOGD is extended till 15 January 2026 as no nominations have been received so far. Nominations for other subcommittees are closed .

# Obstetrics and Fetal Medicine Cross Quiz

**Inderpreet Kaur**
Senior resident (Department of Obstetrics and Gynecology), Lady Hardinge Medical College

1. **The most sensitive marker for first-trimester detection of Trisomy 21 is:**
   A. Nasal bone B. Nuchal translucency
   C. Ductus venosus PI D. Tricuspid regurgitation
   **Explanation:** NT remains the single most sensitive sonographic marker; others add incremental value.

2. **A fetus with non-immune hydrops at 24 weeks shows MCA-PSV 1.8 MoM. The next step is:**
   A. Await repeat Doppler in 1 week
   B. Maternal corticosteroids
   C. Intrauterine transfusion
   D. TORCH PCR
   **Explanation:** MCA-PSV >1.5 MoM with hydrops strongly indicates severe anemia needing urgent IUT.

3. **The most common lethal skeletal dysplasia detected on antenatal scan is:**
   A. Thanatophoric dysplasia
   B. Achondroplasia
   C. Osteogenesis imperfecta type I
   D. Campomelic dysplasia
   **Explanation:** Thanatophoric dysplasia is the most common lethal skeletal dysplasia with characteristic cloverleaf skull and severe micromelia.

4. **Which congenital heart defect is most commonly associated with increased nuchal translucency and normal karyotype?**
   A. Tetralogy of Fallot
   B. Transposition of great arteries
   C. Coarctation of aorta
   D. Atrioventricular septal defect
   **Explanation:** AVSD is strongly associated with increased NT even in euploid fetuses.

5. **The most common congenital diaphragmatic hernia (CDH) subtype is:**
   A. Morgagni hernia
   B. Right-sided posterolateral hernia
   C. Left-sided posterolateral hernia
   D. Bilateral hernia
   **Explanation:** Left-sided Bochdalek hernia is the most common type (>80%).

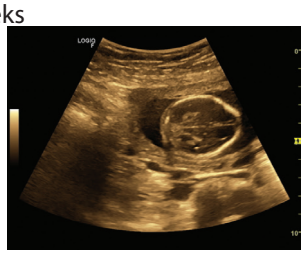
6. **Select the correct management strategy for Stage II TTTS at 22 weeks:**
   A. Expectant management
   B. Amnioreduction
   C. Fetoscopic laser photocoagulation
   D. Selective feticide
   **Explanation:** Laser ablation of placental anastomoses is standard for Quintero Stage II–IV TTTS (16–26 weeks).

7. **A low PAPP-A (<0.4 MoM) is associated with all, EXCEPT:**
   A. Pre-eclampsia B. Fetal growth restriction
   C. Placental abruption D. Neural tube defects
   **Explanation:** PAPP-A reflects placental function; NTDs relate to AFP elevation, not PAPP-A reduction.

8. **A cystic structure seen in midline posterior fossa with enlarged cisterna magna suggests:**
   A. Chiari II malformation
   B. Dandy–Walker malformation
   C. Encephalocele
   D. Vein of Galen malformation
   **Explanation:** DW malformation shows enlarged posterior fossa + elevated tentorium + cystic dilation of 4th ventricle.

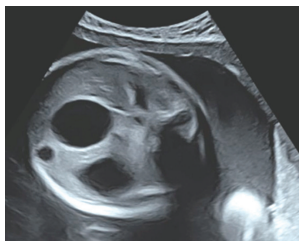
9. **The biggest risk of fetoscopic surgery (any indication) is:**
   A. Maternal bleeding
   B. Chorioamniotic membrane separation
   C. Infection
   D. Fetal bradycardia
   **Explanation:** Membrane separation → PPROM → preterm birth is the major complication.

10. **Which of the following is TRUE regarding the banana sign?**
    A. It appears only after 30 weeks
    B. It is specific for Dandy–Walker malformation
    C. It is due to downward displacement of cerebellum
    D. It indicates mild ventriculomegaly only



    **Explanation:** Caudal herniation of cerebellum through the foramen magnum → banana-shaped cerebellum.

11. **The anomaly depicted in the given picture is MOST strongly associated with which chromosomal anomaly?**
    A. Trisomy 13
    B. Trisomy 18
    C. Trisomy 21
    D. Triploidy
    **Explanation:** 25–40% of fetuses with duodenal atresia have Trisomy 21.



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**Answers**
1. B, 2. C, 3. A, 4. D, 5. C, 6. C, 7. D, 8. B, 9. B, 10 C, 11 C

# AOGD Clinical Meet from MAMC & LNJP Hospital held on 28th November 2025

## Not just rash : Varicella induced complete heart block.

**Poonam Kashyap**
Professor department of OBG

Patient is a case of G2P1L1 with 39wks of pregnancy with previous LSCS referred for ECG changes.

The patient has a history of chickenpox 3 months back at 29 weeks of pregnancy. During her illness, there is history of syncope and palpitations.

She was managed at home. She followed in antenatal period in a district hospital where she was plan for cesarean section. During her work up, ECG changes were suggestive of complete heart block and she was referred. Patient had two episodes of syncope in Lok Nayak Hospital.Her pulse rate was 46 beats per minute. Cardiology evaluation was done.

ECHO showed complete heart block with no other abnormality. Temporary pacemaker was installed. On postoperative day 2 ,LSCS was done. She delivered a baby of 1.8 kg ,Boy with normal apgar score .On 6th post operative day, permanent pacemaker was installed and she was discharged on day 14 in good condition.She is doing fine in follow up.

**Key message**

Acquired causes of complete heart block are rare. A strog index of suspicion with a multidisciplinary team approach is required for the successful outcome.

## Navigating the challenges of a dual disease

**Niharika Dhiman**
Professor Department Of Obstetrics and Gynaecology

A 75-year-old multiparous woman presented with a one-year history of prolapse and foul-smelling discharge for one month. Examination revealed 3rd-degree prolapse with a 10×6 cm ulcerative, fungating lesion on the lateral vaginal wall, bleeding on touch, and 1–2 decubitus ulcers. The unusual lateral site, rather than a dependent area, raised suspicion of malignancy. Kidney function tests were deranged, possibly secondary to chronic prolapse or malignancy. USG KUB showed bilateral gross hydronephrosis with ureteric dilatation. MRI demonstrated a heterogeneous right anterolateral vaginal mass with diffusion restriction, muscularis loss, parametrium invasion, loss of bladder fat plane, Grade 1 cystocele, Grade 2 rectal prolapse, and bilateral hydronephrosis. Imaging was technically difficult due to the prolapsed organ, raising consideration of reduction before scanning. Cystoscopy and proctoscopy were normal. Histopathology revealed pleomorphic cells with mitoses, necrosis, and prominent nucleoli. Immunohistochemistry (pan-CK, EMA, vimentin positive) confirmed sarcomatoid squamous cell carcinoma. Management was challenging, with dilemmas regarding primary surgery versus neoadjuvant or adjuvant therapy versus palliative treatment. Prognosis was assessed with ECOG score. As radiotherapy was not feasible in the prolapsed organ and renal dysfunction limited options, low-dose paclitaxel-based chemotherapy was initiated.

This case highlights a rare malignant transformation in longstanding prolapse, complicated by atypical ulcer location, renal dysfunction, technical imaging limitations, and restricted treatment options, requiring individualized multidisciplinary management.

## When hormones fail: A rare case of 46XY DSD

**Deepti Goswami**
Director Professor and HOD Maulana Azad Medical College Delhi

A 15yr old girl presented in Gynaecology opd with complaints of primary amenorrhea and non development of breast. On examination her height was 154cm with BMI of 19 kg/m2 . Breast and pubic hair tanner 1. Two cm blind vagina present. Her karyotype depicted XY with testosterone deficiency. Further investigation showed increased progesterone with low cortisol, low estradiol. MRI revealed absent uterus, ovaries and presence of gonads with seminal vesicles like structure present deep in inguinal canal. Liquid chromatography - mass spectrometry suggestive of 17 hydroxylase and 17,20 lyase deficiency. Molecular genetics analysis revealed CYP17A1 gene missense mutation on chromosome 10. Laparoscopic gonadectomy was done to prevent malignancy in future after multidisciplinary team meeting. She was started on spironolactone for hypertension management and estrogen for breast development.

## AOGD Subcommittees Chairperson Election ( 2026-28)
## Call for nominations

Nominations for the **Chairperson Medico-legal subcommittee of AOGD** is extended till 15 January 2026 as no nominations have been received so far. Nominations for other subcommittees are closed

Last date for submission of nominations is **15/01/2026**

- ✓ Applications by desirous candidates should be submitted on the prescribed form available on AOGD website (www.aogd.org) / bulletin / office, with due entry in the office register in a sealed envelope & through email aogdlhmc2025@gmail.com
- ✓ Nominations as per the eligibility criteria should reach AOGD secretariat: Department of Obst. & Gynae LHMC & SSK Hospital, New Delhi- 110001 (Phone no. 9717392924 ) by **15/01/2026.**

Dr. Ratna Biswas (Secretary AOGD , 9971372695)

Important announcement : The chairpersons after being nominated have the responsibility to call for application for members of their respective subcommittee for up to a maximum of 10 members.

**Eligibility Criteria for AOGD Sub-committee chairperson**

1. The chairperson of a sub-committee should have been a member of the sub-committee in question for at least one term, with one term being equivalent to two years, prior to his/her appointment as chairperson of that sub-committee.
2. He/she should have been a member of the AOGD for fifteen years.
3. He/she should have experience in the field related to the subcommittee.
4. He/she should have completed at least fifteen years from the date of his/her registration as a medical practitioner. Further, he/she should have held a senior / faculty position for not less than that of associate professor, senior consultant or an equivalent there of in his/her respective organization, for a period of at least five years .
5. No person should hold chairperson ship of the same subcommittee for two consecutive terms with each term comprising of two years. Further, a person who has been chairperson of one subcommittee cannot be nominated as chairperson of another subcommittee unless separated by a duration equivalent to two terms of the subcommittee.
6. The Executive Committee may lay down additional criteria for the eligibility and pre-requisites for appointment as chairperson of each sub-committee from time to time.
7. An eligible member must send an application for nomination as chairperson of a sub-committee stating therein his/her previous experience in the field related to the sub-committee and future vision for furthering the goals of the AOGD through such sub-committee. One person shall not apply for chairpersonship of more than one sub- committee at a time. The application shall be scrutinized by the Executive Committee of AOGD for nomination as chairperson.
8. In the event of more than one application being received for appointment as chairperson of a subcommittee, and in the absence of unanimous decision of the Executive committee in this regard, the Executive Committee shall decide the nomination by cast of secret ballot.
9. The tenure of the chairperson of subcommittee shall be for a period of two years.

# The Association of Obstetricians & Gynaecologists of Delhi

## Nomination Form

Name:_____

Designation/Affilation_____

AOGD Membership no:_____

Official Address:_____

Residential Address:_____

Phone:_____Email:_____

Bio Sketch (Relevant to the Eligibility Criteria in 250words)

| |
|---|
| |

Post Applied for

Sub-committee Chairperson
2026-28

| Subcommittee Name |
|---|
| |

Proposed by – Name AOGD Membership no. Signature

1.

Seconded by

1.

2.

Nominations should reach at AOGD Office
For any Query please call Mrs. Sarita : 9211656757, 9717392924

# Prize Winners

**Competition Paper/ Free Paper/Poster/Quiz**
**47th Annual Conference AOGD**
**13th - 14st September, 2025**

| Category | Award | Name | Institute | Title |
|---|---|---|---|---|
| Dr Neera Agarwal's Medal for Best paper on theme topic: **Maternal Health** | **Gold Medal** | Dr Sampada Kundal | AIIMS | Muscle Fatigue and Motherhood: Myasthenia Gravis in Pregnancy |
| Dr Suneeta Mittal's Medal for Best paper on theme topic: **Population Stabilization** | **Gold Medal** | Dr Ayushi Hada | LHMC & SSK Hospital | Empowering Choices : Implants Reshaping the future of LARC |
| Dr U.P Jha & Raj Soni's Medal for Best paper on theme topic: **Endoscopy** | **Gold Medal** | Dr Ayushi Negi | AIIMS | Healing the Scar: Fertility restoration post -isthmocoele repair |
| Dr U.P Jha & Dewan Balakram's Medal for Best paper on theme topic: **Gynae - Oncology** | **Gold Medal** | Dr Jagriti Bajaj | MAMC | Effectiveness of Antepartum Health Education on Awareness and Acceptance of Human Papilloma Virus (HPV) Vaccine in Postpartum Period |
| Mr S. Bhattacharya & Dr Ganguli's Medal for Best paper on theme -**Miscellaneous Category** | **Gold Medal** | Dr Srishti | VMMC & SJH | A Prospective Study on Predictors and Outcomes of Surgical Site Infections Following Elective Caesarean Section |
| Best paper on theme topic: **Reproductive Endocrinology** | **Gold Medal** | Dr Sowmiya Rajendran | LHMC | Beyond Insulin-TyG Index as a Cost-Effective Marker of Insulin Resistance in PCOS |
| **Poster Presentation** | **Gold Medal (tie)** | Dr Garima Wadhwa | AIIMS | A Benign Masquerade of Malignancy: Diffuse Peritoneal Leiomyomatosis – A rare Case Report |
| | **Silver Medal (tie)** | Dr Monika Jain | MAMC | Recurrent Vulvar Aggressive Angiomyxoma with Hormonal Receptor Shift following Treatment Interruption- A Rare Case Report |
| | | Dr Parul Kargwal | VMMC & SJH | Zoomed Zoned verified: The diagnostic leap from conventional to three ring vulvoscopy . |
| **Slogan** | **1st Prize** | Dr Kanika Chopra | LHMC & SSKH | |
| Research Paper- **Best Competition Paper** | **Gold Medal** | Dr Divya Khurana | SRHC NARELA | Rapid cycle improvement model as an effective quality tool for rationalizing oxytocin usage in third stage of labour |
| | **Silver Medal** | Dr Nisha Chopra | VMMC & SAFDARJUNG HOSPITAL | Grobman Score for Predicting Successful Trial of Labor After Cesarean in a North Indian Population |
| | **Bronze Medal** | Dr Megha | LHMC & SSK Hospital | Accuracy of Modified Cardiovascular Sequential Organ Failure Assessment (M-Cv Sofa) Score For Predicting The Duration of Critical Care Unit Stay in Maternal Sepsis |
| Dr Batra's Medal winner of **AOGD Quiz** | **Gold Medal** | Dr Saipriya & Dr Shivangi Singh | | |
| | **1st Runner Up** | Dr Rahul & Dr Shagun | | |
| | **2nd Runner Up** | Dr Nilufer & Dr Akanksha | | |
| Dr S N Mukherjee **Rotating Trophy** | **Best AOGD Monthly Clinical Meeting** | VMMC & Safdarjung Hospital | | |

# Events Held 2025

**A cervical cancer vaccination camp conducted by Urogynecology Subcommittee at Mandi, Himachal Pradesh on 4th November 2025.**



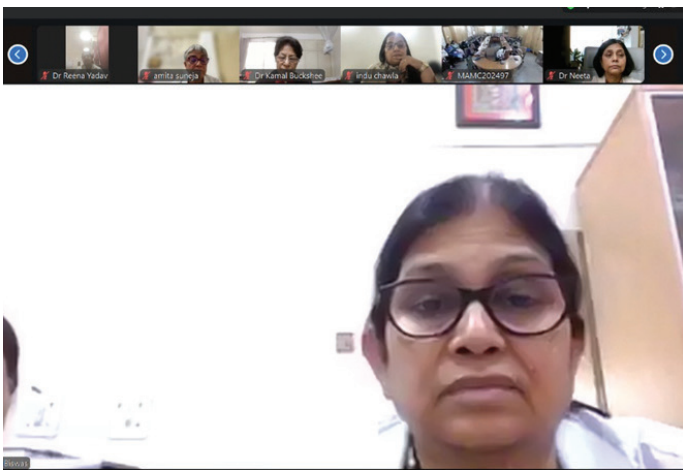
**CME on "Gestational Diabetes Mellitus ( GDM ): Updates, Challenges and Clinical Pearls" conducted by Infertility & Reproductive Endocrinology Subcommittee on 15th November, 2025 at LHMC & SSK Hospital**

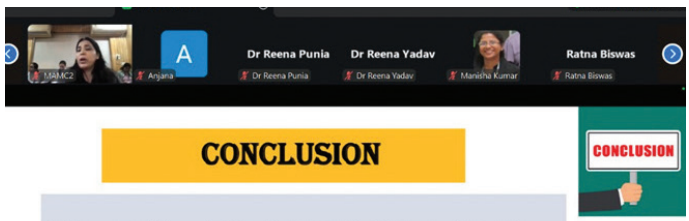
**Webinar on Ultrasound in Endometriosis conducted by FOGSI, Infertility committee in association with AOGD Endometriosis committee on 17th November, 2025**

**FOGSI-JOGI-PICSEP workshop on "Research Methodology" conducted by AOGD on 22nd November,2025 at LHMC**


**Public awareness program for Vasectomy Fortnight conducted by Dept. of Obst. & Gynae , LHMC in associated with AOGD on 26th November 2025 at LHMC & SSK hospital.**


**The AOGD Monthly Clinical Meeting (virtual) conducted by the Department of Obst & Gynae, MAMC & LNJP Hospital on 28th November, 2025**





# Association of Obstetricians & Gynaecologists of Delhi

## MEMBERSHIP FORM

**Name:**.....

**Surname:** .....

**Qualification (year):** .....

**Postal Address:** .....

**City:**..... **State:** ..... **Pin code:** .....

PHOTO

**Place of Working:** .....

**Residence Ph. No.** ..... **Clinical / Hospital Ph. No.** .....

**Mobile No:**..... **Email:** .....

**Gender: Male:**..... **Female:**.....

**Date of Birth: Date**.....**Month** ..... **Year**.....

**Member of Any Society:**.....

**Proposed by** .....

**Cheque/DD / No:** .....

Cheque/Demand Draft should be drawn in favour of: **Association of Obstetricians and Gynaecologists of Delhi**

FOR ONLINE TRANSFER THROUGH NEFT/RTGS

**Name of Account: Association of Obstetricians and Gynaecologists of Delhi**
**Account no: 5786412323**
**Name of Bank: Central Bank of India**
**Branch: LHMC & SSK Hospital**
**IFSC code: CBIN0283462**
**MICR code: 110016067**

ASSOCIATION OF OBSTETR

12418708@cbin

BHIM UPI

| | |
|---|---|
| For Life Membership | : Rs. 11,000 + Rs. 1,980 (18% GST applicable) = Rs. 12,980 |
| For New Annual Membership* | : Rs. 2,000 + Rs. 360 (18% GST applicable) = Rs. 2,360 |
| For Old Renewal Membership+ | : Rs. 1,200 + Rs. 216 (18% GST applicable) = Rs. 1,416 |

**Encl.: Attach Two Photocopies of All Degrees, DMC Certificate and Two Photographs (Self attested)**

*** Annual Membership is for the calendar year January to December.**

*** In case of renewal, mention old membership number.**

**Note: 18% GST will be applicable as FOGSI requires it.**

Send Complete Membership Form Along With Cheque / DD and Photocopy of required documents to the secretariat.
For online transaction send scan copy of all documents with payment slip on given mail id

**Secretariat**
Department of Obstetrics and Gynaecology
Lady Hardinge Medical College & SSK Hospital, New Delhi-110001
**Tel.: 011-23408297, (M): 9717392924 | Email Id: aogdlhmc2025@gmail.com**

**AOGD SECRETARIAT**
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