Volume 26 I August 2025 I Monthly Issue 4

AOGD BULLETIN

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

THEME: PLACENTAL ANOMALIES: DECODING PLACENTAL PUZZLES

AOGD SECRETARIAT
Department of Obstetrics and Gynaecology
Lady Hardinge Medical College & Associated Hospitals, New Delhi-110001
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AOGD 2025

47th Annual Conference of AOGD

Organised By: Department of Obstetrics and Gynaecology
Lady Hardinge Medical College & Associated Hospitals, New Delhi

Venue: India Habitat Centre, New Delhi

Theme - Tiny Heartbeats to Timeless Strength - Honouring the Journey of Women Through Birth & Beyond

Pre Conference Workshop- 11th & 12th Sep 2025
Main Conference - 13th to 14th Sep 2025

ABSTRACT IS LIVE NOW

Please visit the https://aogd2025conference.com/ website for more details.

Conference Secretariat:

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Dr Kiran Aggarwal (Organising Co- Chairperson)
Dr Ratna Biswas (Organising Secretary)
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President & Secretary 's Message

Dr Reena Yadav
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Joint Secretary

Dear Members,

Greetings from AOGD secretariat at Lady Hardinge Medical College!

The autumn breeze is setting in, beaconing pleasant days ahead as we prepare for the much awaited 47th Annual Conference of AOGD.

The conference is slated for 13th and 14th September at the India Habitat Centre and the preconference workshops are distributed over two days, that is 13th and 14th September in different venues.

Theme of the conference is- "From Tiny Heartbeats to Timeless Strength-Honouring the Journey of Women from Birth & Beyond".

The scientific program is in tune with our theme, encompassing all aspects of women's health. It will unravel the nuances on how to navigate through the tight spots in clinical practice, provide insights on emerging trends and recent advances, showcase videos on skills enhancing surgical techniques, and provide real time experience on managing patients through case -based discussions. The faculty are experienced and dedicated professionals with illustrious careers, possessing in depth knowledge in their core specialities and will be a treat to hear.

Each of the thirteen workshops are aimed at enhancing a particular skill set providing an opportunity to the delegates to choose a topic of their interest, from the wide range of subjects covered.

Have no doubts, the program will be worth your time spent towards achieving your personal goals and career development. Register now as the regular registration closes on 15th August.

In July we celebrated the population stabilization fortnight by organizing CME on Family Planning and conducted public awareness program on Contraception on occasion of World Population Day. The subcommittees were on a whirlwind, organizing a spectrum of activities from webinars on Rh isoimmunization, Ectopic pregnancy, Non Descent Vaginal Hysterectomy, PCOS to physical CMEs on Gynae oncology, ART, Vulval Disorders and Workshop on Colposcopy to name a few.

The topic of August issue of AOGD bulletin is "Placental anamolies: Decoding Placental Puzzles". It provide insights on placental disorders causing feto-maternal adverse outcome and calls for an interesting read. We appreciate the efforts of Dr Manisha and the editorial team for highlighting this important aspect of the placental origin of feto-maternal disorders.

Best Wishes

AOGD Secretariat

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,

We have come with yet another issue on "Placental anomalies: Decoding placental puzzles". Despite being the lifeline of pregnancy, the placenta remains one of the least understood organs in human biology. The placenta is, in essence, the first and most enduring partnership of human life. Its mysteries are not insurmountable, but unlocking them demand ingenuity, perseverance, and the convergence of multiple scientific disciplines.

Conceived anew with every gestation and discarded at birth, it performs functions of astonishing sophistication — respiratory, nutritional, endocrine, and immunological — with a precision that rivals any other organ, yet defies prolonged study because of its transient nature.

A uniquely collaborative creation between mother and child, the placenta is, paradoxically, both intimately connected to the newborn and routinely unseen by the mother. At delivery, it is severed and discarded, yet it holds within its structure a remarkable record — a biological diary of the health of both mother and baby. Proper placental development is essential: through the invasive remodelling of maternal blood vessels, it ensures the optimal exchange of nutrients, gases, and waste. When this finely tuned process falters — as it does in up to 20% of pregnancies — the consequences may be miscarriage, fetal demise, or growth restriction.

This issue is dedicated to the placenta, within these pages, experts explore the placenta, from the determination of **placental health** through its markers to the use of **aspirin for the prevention** of the adverse effects. The role of chronic hypoxia in **fetal growth restriction,** to the chronology of placental changes in **Diabetes Mellites**. Placental as a witness of tragedy called stillbirth is explored by its **examination and histopathology** postdelivery. We have also delved upon unique placental problems such as vessel sharing in **monochorionic twins** and placental stickiness in **placenta accreta spectrum** and c**aesarean scar pregnancy**. We have an exciting **quiz** on morphological **abnormality of placenta & cord**.

Hope this issue of AOGD bulletin gives us a new perspective as we look at diseases from the placental point of view.

Happy festivities

The Editorial Team

47th Annual Conference of AOGD 2025

Date: **13-14 September**

Venue: **India Habitat Centre, New Delhi**

Scientific Program 13.09.2025 Day 1 HALL A

Time	Hall A - Stein Auditorium
8:00-9:00 am	**Registration**
8.50am-9.00am	**AOGD Flag hoisting**
	Topic
9:00am-10:00 am	**Session 1: Controversies in Obstetrics** **Chairperson : Dr Renuka Malik, Dr Rekha Jain, Dr Uma Vaidyanathan, Dr Kiran Bala Dash**
9:00am-9:10am	Fetal intrapartum CTG Monitoring in Low-Risk Pregnancies – Overuse or Essential? **- Dr Rinku Sen Gupta**
9.10 am- 9.20 am	Cesarean on Demand – A Woman's Right or Medical Malpractice? - **Dr Manju Khemani**
9:20am -9:30 am	Role of Ultrasound – Too Much Screening or Essential for Fetal Health? - **Dr Sangeeta Gupta**
9.30am -9.40am	Pharmacotherapy in GDM- Metformin versus Insulin - **Dr Pikee Saxena**
9.40am -9.55am	Chairperson comments & Discussion
10:00-11:00am	**Session II Case based Panel discussion :** **When Infection Strikes – Obstetric Sepsis and Emerging Threats**
	Moderators : Dr Ratna Biswas , Dr Jyotsna Suri **Panelists : Dr Chandra Mansukhani, Dr Rachna Agarwal, Dr Rekha Bharti, , Dr Rashmi Malik , Dr Prasoon Gupta , Dr Vandana Goel , DrNalini Bala Pandey** **Experts: Dr Anjali Tempe , Dr Anjali Dabral,**
11:00am-12:00 noon	**Session :III Symposium** **Critical Crossroads in High-Risk Obstetrics – Navigating Dual Lives with Precision and Compassion** **Chairpersons: Dr Harsha Khullar, Dr Monica Madan, Dr Rajesh Kumari (SJH) , Dr Ritu Sharma**
11.00am-11.12am	Managing Cardiac Disease in Pregnancy – Walking the Tightrope Between Physiology and Pathology - **Dr YM Mala**
11.12am-11.24am	Severe Preeclampsia and HELLP Syndrome – Early Clues, Timely Action, Better Outcomes - **Dr Manisha Kumar**
11.24am-11.36am	Predicting and Preventing Preterm Birth – From Cervical Length to Progesterone Protocols - **Dr Kiran Aggarwal**
11.36am- 11.48 am	Autoimmune Thyroid Disease in Pregnancy: Silent Threats and Strategic Interventions **- Dr Muntaha Khan**
11.48am-12.00	Chairpersons comments & Discussion
12.00-12.30pm	**Brigadier Khanna Oration** **Dr Arun Prasad** **Topic: Robotic Surgery -Hype or Hope** **Chairperson :Dr Kamal Buckshee, Dr SB Khanna, Dr Neera Aggarwal, Dr SS Trivedi , Dr Reena Yadav**

12.30pm-12.50pm	**Key Note Address** **Speaker : Dr Neerja Bhatla** **Breast Cancer screening-Essential for Gynaecologists** **Chairpersons: Dr Chitra Raghunandan, Dr Sharda Jain, Dr Reva Tripathy, Dr Kawal Gujral, Dr Kiran Aggarwal**
1.00-1.30pm	**Inauguration**
1.30-2.15pm	**Lunch**
2.15pm-3.15pm	**Session IV Panel cum Symposium :** **Saving the Second Twin – Challenges in Multifetal Delivery** **Chairpersons: Dr Upma Saxena, Dr Indu khatri, Dr Richa Sharma, Dr Mamta Dagar**
2.15pm-2.25 pm	When to Deliver Twins – Timing It Right **- Dr Akshata Prabhu**
2.25pm-2.35 pm	Second Twin in Breech or Transverse – What's the Best Route - **Dr Sumitra Bachani**
2.35pm -2.45pm	Cord Prolapse and Fetal Distress – Real-time Decision Making **- Dr Deepika Meena**
2.45pm-3.25pm	**Panel discussion -Case scenarios with discussion:** **Saving the Second Twin – Challenges in Multifetal Delivery**
	Moderator : Dr Aparna Sharma , Dr Reema Bhatt **Panelists :Dr Neha Gupta, Dr Rachna Gupta, Dr Sumedha, Dr Richa Aggarwal ,Dr Neha Gulati , Dr Anju Kumari** **Expert: Dr Deepika Deka , Dr Anita Kaul**
3.30pm onwards	**Session V (A) : Surgical videos in Obstetrics: Difficult Cesarean Section** **Chairpersons: Dr Mrinalini Mani, Dr Suman Mediratta, Dr Poonam Kashyap, Dr Divya Pandey**
3.30pm-3.40 pm	Difficult Cesarean with Previous Scar : Techniques for Safe Delivery - **Dr Vinita Gupta**
3.40pm-3.50 pm	Cesarean Section in deeply impacted head - **Dr Shakun Tyagi**
3.50pm -4.00 pm	Cesarean hysterectomy in Placenta Accreta - **Dr Taru Gupta**
4.00pm-4.30pm	**Session V (B) : Cutting-Edge Obstetric Surgery – Saving Lives, Preserving Futures** **Chairpersons :Dr Nutan Aggarwal, Dr Alpana Singh, Dr Pooja Pathak, Dr Divya Chauhan**
4.00-4.10 pm	POCUS in Obstetric Emergency Protocols for Pulmonary Edema and Hypertensive Disorders - **Dr Nishant Kumar**
4.10-420 pm	Cesarean Myomectomy – Safer Technique - **Dr Aruna Nigam**
4.20-4.30 pm	Laparoscopic Cervico-isthmic Cerclage in Second Trimester - **Dr Garima Kachhawa**
	Free Papers in Maple and Magnolia halls

47th Annual Conference of AOGD 2025

Date: **13-14 September**
Venue: **India Habitat Centre, New Delhi**

Scientific Program 13.09.2025 Day 1 HALL B

Time	Hall B – Jacaranda
8:00-9:00 am	**Registration**
	Topic
9:00am-10:00 am	**Session :1 : Controversies in Gynecology** **Chairpersons Dr Deepti Nabh , Dr Surveen Ghumman, Dr Himsweta Srivastav , Dr Garima**
9:00am-9:10am	Vaginal Rejuvenation and Cosmetic Gynecology – Should It Be a Priority ? - **Dr JB Sharma**
9.10 am- 9.20 am	Mid-Urethral Slings: Still the Gold Standard or Facing a Global Recall? - **Dr Amita Jain**
9:20am-9:30 am	Fertility Preservation surgery – Should it be Standard Practice for Women with Cancer ? - **Dr Shalini Rajaram**
9.30am-9.40 am	PCOS Management in Adolescents: Lifestyle First or Medical Therapy Upfront? **- Dr Asmita Rathore**
9.40am-9.50am	Chairperson comments & Discussion
9.50am -10.20am	**Session II : Panel cum Symposium- Adenomyosis – The Overlooked Twin of Endometriosis"** **Chairpersons: Dr Sudha Salhan, Dr Kuldeep Jain, Dr Monika Bhatia, Dr Sheeba Marwah**
9.50am-10.00am	Emerging imaging criteria: transvaginal USG vs MRI **- Dr Bharti Jain**
10.00am-10.10am	Molecular and Genetic Insights into Adenomyosis: Pathogenesis and Future Therapeutic Targets **-Dr Nishtha Jaiswal**
10.10am-10.20am	Newer uterine-sparing interventions **- Dr Dinesh Kansal**
10.20am -11.10am	**Panel discussion -Case scenarios with discussion -Adenomyosis – The Overlooked Twin of Endometriosis"**
	Moderator ;Dr Reena Yadav, Dr Indu Chugh **Panelists : Dr Neeta Singh, Dr Meenakshi Singh , Dr Reeta Mahey, Dr Neema Sharma, Dr Vidhi Chaudhary, Dr Renu Tanwar, Dr Kavita Agarwal**
11:10-12:00 noon	**Session :III Surgical Innovation in Gynecology – Laparoscopy, Robotics and Beyond"** **Chairpersons : Dr Renu Mishra , Dr Rama Joshi, Dr Sabhyata Gupta, Dr Debashish Dutta ,**
11.10-11.25am	Next-Gen Laparoscopy – Smarter, Safer, Sharper **- Dr Alka Kriplani**
11.25-11.40am	Robotic Gynecology – Expanding Access, Redefining Precision - **Dr Rupinder Shekhon**
11.40am-11.55am	"Digital Surgery, AI, and the Operating Room of the Future"- **Dr Aruna kumari Yerra**

11.55-12.00 noon	Chairperson comments & Discussion
12.00-12.30pm	**Brigadier Khanna Oration** **Dr Arun Prasad** **Topic: Robotic Surgery –Hype or Hope** **Chairperson : Dr Kamal Buckshee, Dr SB Khanna, Dr Neera Aggarwal, Dr SS Trivedi , Dr Reena Yadav**
12.30pm -1.00pm	**Key Note Address** **Speaker : Dr Neerja Bhatla** **Breast Cancer screening-Essential for Gynaecologists** **Chairpersons: Dr Chitra Raghunandan, Dr Sharda Jain, Dr Reva Tripathy, Dr Kanwal Gujral, Dr Kiran Aggarwal**
1.00-1.30pm	**Inauguration**
1.30-2.15pm	**Lunch**
2.15-2.35 pm	**Session IV on Medical legal aspects** **Chairpersons**
2.35pm-2.45pm	**TBA**
2.45pm -3.30pm	**Session V : Panel Discussion on Pelvic masses demystified- Malignancy or Mimic**
	Moderator- Dr Sharda Patra , Dr Urvashi Miglani **Panelists: Dr Swasti , Dr Bindiya Gupta, Dr Jyoti Meena, , Vikas Chaudhary , Dr Amita Naithani , Dr Pakhee Aggarwal , Dr Nishat Amin** **Experts- Dr Vijay Zutshi , Dr Sunita Malik,**
3.30pm onwards	**Session VI A Surgical Videos in Gynaecology** **Chairperson: Dr Gouri Gandhi, Dr Neha Kumar, Dr Rahul Modi, Dr Vinita Jaggi , Dr Vaishali Palliwal**
	Topic: Precision and Progress in Gynecologic oncologic Surgery
3.30-3.40 pm	Cyto reductive surgery in ovarian malignancy **- Dr Bhagyalaxmi Nayak**
3.40-3.50 pm	Type C1 Radical Hysterectomy for Cervical Cancer - **Dr Seema Singhal**
3.50-4.00 pm	Radical Vulvectomy - **Dr Upasana Barooah**
4.00pm-4.40pm	**Session VI-B Laparoscopic & Hysteroscopic Video** **Chairpersons: Dr Latika Sahu, Dr Punita Bhardwaj, Dr Sumita Mehta , Dr Shivani Sabharwal**
4.00-4.10 pm	Total Laparoscopic Hysterectomy (TLH): Step-by-Step for a Difficult Uterus - **Dr Nikita Trehan**
4.10-420 pm	Laparoscopic vascular complication - **Dr Bijoy Nayak**
4.20-4.30 pm	Hysteroscopic myomectomy **- Dr Biswa Dash**
4.30-4.40 pm	Laparoscopic management of adenexal masses - **Dr Kanika Jain**
	Free Papers in Maple and Magnolia halls

AOGD 2025
Scientific Program 14.09.2025 Day 2 HALL A

Time	Hall A Stein Auditorium
8:00-9:00 am	**Registration**
	Topic
9:00 -10:00 am	**Session 1: Symposium: Hormonal Harmony: Redefining Care in Reproductive Endocrinology** **Chairpersons: Dr Rita Bakshi, , Dr Leena Wadhwa, Dr Poonam Laul, Dr Raka Guleria**
9.00am-9.10am	When to suspect pituitary or adrenal pathology in menstrual disorders - **Dr Aishwarya Kapur**
9.10am-9.20am	Modern diagnostic dilemmas – adolescent vs adult PCOS - **Dr Neena Malhotra**
9.20am-9.30am	Navigating Premature Ovarian Insufficiency – Restoring Hope, Not Just Hormones - **Dr Abha Majumdar**
9.30am-9.40am	Diagnosing perimenopause: Estrogen excess to estrogen exit –What's missed and misinterpreted - **Dr Mala Srivastava**
9.40am- 9.50 am	Chairperson comments & Discussion
10.00am-11:00am	**Session : II (J& J Sponsor)** **Chairperson : Dr Poonam Goyal , Dr Anita Sabharwal, Dr Poonam Sachdeva , Dr Astha Srivastava**
10.00am-10.20am	Barbed sutures – Cutting Edge of Technology for Gynaecological surgery - **Dr Jaishree Sundar**
10.20am-10.40am	Science of Energy **Dr Eham Arora/ Dr BB Das**
10.40-11.00am	Chairperson comments & Discussion
11.00am-12.00	**Session :III Debate -The Evidence Face-Off** **Chairpersons : Dr Deepa Gupta, , Dr Shivani Aggarwal, Dr Seema Prakash, Dr Archana Mishra**
11.00am-11.30am	**Routine HPV Vaccination in Adults Over 26: Beneficial or Unnecessary**
11.00am-11.10am	For -Beneficial: **Dr Nilanchali Singh**
11.10am-11.20am	Against -Unnecessary: **Dr Satinder Kaur**
11.20am-11.30am	Discussion -5mins
11.30am-12.00	**Should Opportunistic Salpingectomy Be Routine for Ovarian Cancer Prevention**
11.30am-11.40am	Yes - **Dr Niharika Dhiman**
11.40am-11.50am	No - **Dr Kanika B Modi**
11.50am-12.00	**Discussion**

12.00-1.00pm	**Session IV: Keynote Lectures** **Chairpersons: Dr Sunita Mittal., Dr Usha Gupta, Dr Sanjivini Khanna , Dr Bindu Bajaj, Dr Ratna Biswas**
12.00-12.15am	Navigating the Uterine Niche-Cesarean scar defects and reproductive implications -**Dr Ritu Rana (UK)**
12.15pm-12.30pm	Laparoscopic Vaginal reconstructive surgery - **Dr Hafeez Rehman (Dubai)**
12.30pm-12.45pm	Fatty liver is core to all NCD'S **- Dr Shiv Kumar Sarin (ILBS)**
12.45pm-1.00pm	Chairperson comments & Discussion
1.00-1.30pm	**AOGD Past President Oration** **Dr Ashok Kumar** **Topic : Oral health & Pregnancy** **Chairpersons: Dr Maya Sood, Dr Abha Singh, Dr NB Vaid , Dr Achala Batra, Dr Malvika Sabharwal**
1.30-2.15pm	**Lunch**
2.15-3.15pm	**Session V: Competition paper** **Judges**
3.15-4.15 pm	**Session VI-: Quiz-Final round** **Quiz Masters : Dr Meenakshi Singh , DrVidhi Chaudhary**
4.15 pm onwards	**Valedictory & Vote of Thanks**
	Free Papers in Maple and Magnolia halls

47th Annual Conference of AOGD 2025

Date: **13-14 September**
Venue: **India Habitat Centre, New Delhi**

Scientific Program 14.09.2025 Day 2 HALL B

Time	**Hall B - Jacaranda**
8:00-9:00 am	**Registration**
	Topic
9:00am -10:00 am	**Session I Game changer Guidelines in Obstetrics & Gynaecology** **Chairpersons: Dr Shanti Jaisheelan, Dr Gouri M Devi, Dr Vandana Gupta, Dr Manju Hotchandani**
9.00am-9.10am	Management of Intraamniotic Infection - **Dr Harsha Gaikwad**
9.10-9.20am	Rh iso immunisation - **Dr Vandana Chaddha**
9.20-9.30am	CIN2 Conservative management - **Dr Prabha Lal**
9.30-9.40am	AUB Classification- FIGO 2023 - **Dr Krishna Aggarwal**
9.40-9.55am	Chairperson comments

10.00am-10.45am	**Session : II Panel Discussion** **Labor That Stalls – Dystocia Dilemmas in Real Time**
	Moderator- Dr Manju Puri, Dr Shilpi Nain **Panelist: Dr Madhavi M Gupta, Dr Monica Gupta , Dr Priti Dhamija , Dr Meenakeshi Rana , Dr Madhu Goel, Dr Neha Pruthi, Dr Aruna Verma**
10.45am-11.00am	**Robotic Video Session** **Chairpersons; Dr Anuradha Kapur, Dr Bela Makhija, Dr Sonia Naik , Dr Anita Rajorhia**
10.45am-10.55am	Robotic assisted total omentectomy as part of Cytoreductive surgery in ovarian cancer - **Dr Rama Joshi**
11.00am-11.40am	**Session III- Obstetrics & Gynecology Conundrums – Decoding Diagnostic Dilemmas** **Chairpersons: Dr Kiran Guleria, Dr Uma Rai, Dr Ranjana Sharma, Dr (Brig) Sunil Takiar ,**
11.00-11.10am	Detrusor overactivity or just a nervous bladder – Decoding the unstable tracings - **Dr Geeta Mendiratta**
11.10am-11.20am	Postmenopausal Bleeding with Thin Endometrium - **Dr AG Radhika**
11.20-11.30am	Polyhydramnios with Unknown Etiology- **Dr Anjali Taneja**
11.30-11.40am	Eclampsia without Hypertension-Diagnostic miss or atypical variant - **Dr Wansalan K Shullai**
11.40-11.45pm	Chairperson comments & Discussion
11.45am-12.00pm	**Session IVA The Vaginal Route Reimagined – From Classical Mastery to VNOTES Innovation"(Sponsored)** **Chairpersons: , Dr Abha Sharma , , Dr Rajesh Kumari (AIIMS) , Dr Swati Sinha , Dr Vijayata Sangwan**
11.45-12.00pm	VNOTES – Basics to advanced **Dr Swati Agrawal**
12.05-12.35pm	**Session IV B Sponsor Session (Plus plus Life sciences , Makers of TRIMACARE)** **Chairperson: Dr JB Sharma, Dr R S.Sharma**
12.05-12.25pm	Improve pregnancy outcome : Redefining antenatal care with nutrition -first approach -**Dr Vidya Thobbi**
12.25-12.35pm	Chairperson comments
12.35-1.00PM	**Session V – Keynote Address** **Dr SN Basu, Dr Raksha Arora, Dr Reena Yadav Dr Nirmala Aggarwal ,Dr Sushma Sinha**
12.35-12.55pm	FGR – Prediction and Prevention **- Dr Srividhya Sankaran(UK)**
12.55-1.00pm	**Discussion**
1.00-1.30pm	**AOGD Past President Oration** **Dr Ashok Kumar** **Topic : Oral health & Pregnancy** **Chairpersons: Dr Maya Sood, Dr Abha Singh, Dr NB Vaid , Dr Achala Batra, Dr Malvika Sabharwal**

page no. 7

1.30-2.15pm	**Lunch**
2.15-3.15pm	**Session VI- Fertility, Contraception & Beyond – Clinical Priorities in 20s and 30s** **Chairpersons: Brig. Dr Prasad Lele, Dr Sohani Verma, Dr Jyoti Bali, Dr Lisa Sharma**
2.15 pm-2.25pm	Fertility preservation for late motherhood and career planning - **Dr Sonia Malik**
2.25-2.35pm	Contraceptive choices: tailoring to lifestyle and comorbidities - **Dr Shikha Chadda**
2.35- 2.45pm	Preconception health – optimizing before the bump - **Dr Anuradha Singh**
2.45pm-3.00 pm	Endometriosis and fibroids in young women: fertility-friendly management -**Dr Anjela Aneja**
3.00pm-3.15pm	Chairperson comments & Questions
3.15-4.15pm	**Session VII: Debate -The Evidence Face-Off"** **Chairpersons : Dr Deepika Loganey , Dr Vandana Bansal, Dr Shilpa Ghosh, Dr Rinku Lodha Negi**
3.15-3.40pm	**Non-invasive Prenatal Testing (NIPT) for All**
3.15-3.25pm	For- **Dr Jaya Chawla**
3.25-3.35pm	Against- **Dr Apoorva Kulshreshtha**
3.35--3.40 pm	**Discussion**
3.40-4.055pm	**Universal Aspirin Use in Pregnancy: Prevention or Overprescription**
3.40-3.50pm	Prevention - **Dr Sruthi Bhaskararan**
3.50pm-4.00pm	Over prescription - **Dr Kamna Dutta**
4..00pm-4.10pm	**Discussion**
4.15pm onwards	**Valedictory & Vote of Thanks**
	Free Papers in Maple and Magnolia halls

Markers of placental health

Manisha Kumar
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Introduction

Placenta is a mirror of fetal health and wellbeing. It is often called a black box, and has records of all adverse events culminating in fetal adverse outcome if it occurs. There has been tremendous interest in the ways to look at the placenta in recent years. The conditions like hypertension, diabetes, infection, genetic, inflammation have all been found to contribute to fetal and neonatal morbidity and mortality through placental pathology.[1] The markers of placental pathology range from maternal high-risk factors, biochemical, biophysical and morphological markers. [2]

Various research project undertaken in our institute is given below :

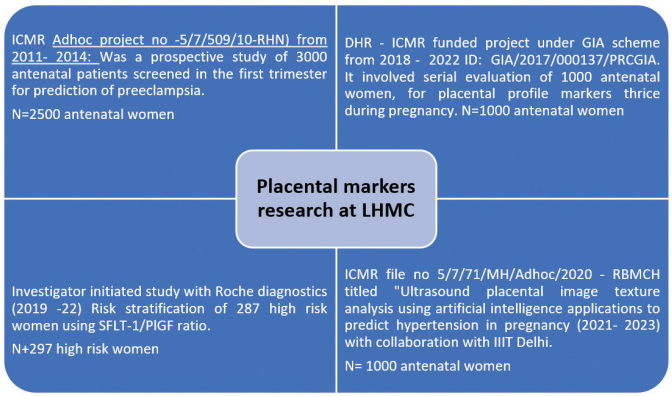


Figure 1: Research projects on Markers of adverse pregnancy outcome done in LHMC

Development of placenta

The first wave of trophoblastic invasion starts with the implantation of embryo. In it the cytotrophoblast invades the decidua. From 11 to 16 weeks the there is second wave of trophoblastic invasion in which the cytotrophoblast invade and replace the endothelium, of vascular smooth muscle, and elastic lamina of the spiral arteries with an amorphous fibrinoid material.

This process transforms the normally narrow, high-resistance arteries into wider, low-resistance vessels. A large volume of blood is pumped into maternal fetal interface every minute, where the chorionic villi containing arterial and venous capillaries are present, for exchange of oxygen and nutrients [3].

For some reasons if the remodeling of the spiral arterioles does not take place, there is hypoxia and release of inflammatory radicals which cascades the endothelial damage, and subsequent systemic pathology in the mother.

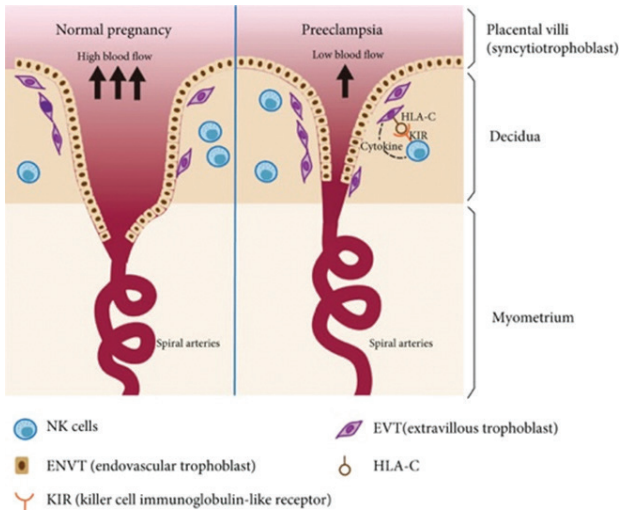


Figure 2: Spiral artery remodeling in health and disease

Hindawi BioMed Research International Volume 2020, Article ID 4808072, https://doi.org/10.1155/2020/480807. [4]

Maternal high-risk factors as placental markers

The maternal factors which point towards the possibility of placental pathology are mainly related to preexisting medical conditions, lifestyle factors and poor obstetric history[3]

- Stress and Lifestyle
- Maternal age > 40 years
- BMI > 35kg/m2
- Nulliparity
- Smoking
- Previous- FGR, stillbirth, abortion
- Maternal medical condition – hypertension, renal, APS, diabetes with vasculopathy

In our research study we found maternal factors to be present in 50% of the cases with stillbirth (16/33 cases of stillbirth).[5] Increased Mean arterial pressure and hypertension in previous pregnancy as significant factors in early onset FGR, stillbirth and PE. Increased BMI (> 27kg/m^2) as significant factor in adverse fetal outcome.[6]

Biophysical markers of placental function

Doppler ultrasound is a non-invasive tool used to assess fetal well-being during pregnancy, particularly in cases of suspected fetal hypoxia. potentially leading to serious complications such as fetal growth restriction and even

fetal demise.[6]

- Uterine artery Doppler was good in prediction of preeclampsia in all trimester of pregnancy, but most in the first trimester in our study.
- The UA Doppler reflects placental function. The UA Pulsatility Index (PI) is used to assess the progression of early onset fetal growth restriction
- MCA Doppler assessment indicates cerebral perfusion adequacy. Fetal hypoxia triggers the "brain-sparing effect," where blood flow is redistributed to prioritize the brain, leading to vasodilation in the MCA and a decreased MCA Pulsatility Index (MCA-PI).
- Ductus venosus Doppler has predictive capacity for perinatal mortality.
- A population study of all deliveries in Norway 1990–2014, showed that the use of dopplers lead to significant decline in preterm stillbirth.[6]

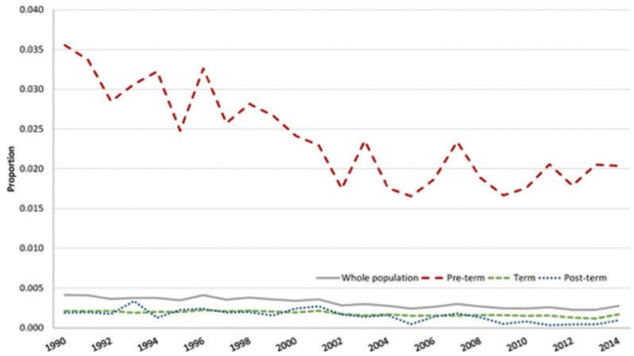


Figure 3: Stillbirth rate from 1990 – 2014 in Norway with the use of Dopplers[6]

J Gryttenet al. , *International Journal of Epidemiology*, Volume 50, Issue 6, December 2021, Pages 2038–2047[6]

Among low-risk women, uterine artery Doppler in the first trimester was abnormal in 21% cases who had stillbirth. CPR was found to be abnormal at 34-36 weeks in 12% of cases who had stillborn babies.[7]

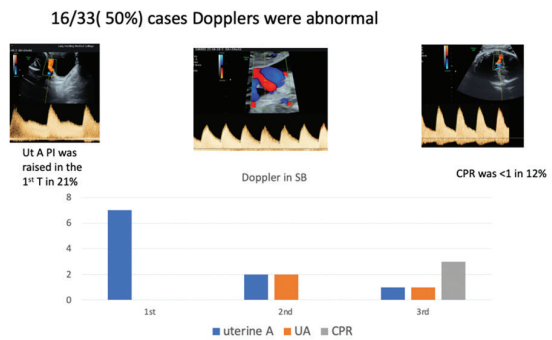


Figure 4: Doppler abnormality in antenatal women who had stillbirth later in pregnancy

In our study on high-risk women, we found that abnormal MAP and uterine artery PI at 20 – 24 weeks, predicted adverse materno fatal outcome in 100% cases[7].

Biomarkers and fetal adverse outcomes

1. Pregnancy associated plasma protein A

PAPP-A (Pregnancy-Associated Plasma Protein A) is a protein produced by the placenta which increases the bioavailability of insulin-like growth factor (IGF) by cleaving insulin-like growth factor binding proteins (IGFBPs), thus releasing active IGF to promote cell growth and proliferation. In the absence of PAPP-A, IGF fails to attach to the receptors and thus angiogenesis does not take place. In the study by Mastrodima et al, from Nicolaides group the Odds Ratio of predicting stillbirth in cases with PAPP-A values less than 0.4 MOM was 2.15. In subjects with stillbirth due to impaired placentation the PAPP-A was lower in comparison to cases with unexplained stillbirth [8]. With the advent of PlGF the importance of PAPP-A as a marker for adverse outcome, particularly preeclampsia, seems to have deceased but it is still relevant as it is a marker for first trimester combined screening for Down syndrome.

2. PlGF

Previous studies have estimated PlGF between a wide gestational age range of 20 weeks to 36 weeks and have taken 100 pg/ml as PlGF cut-off [9, 10], however, in the present study, the PlGF levels were performed thrice, successively and the gestation specific cut-offs were derived (224pg/ml, 211pg/ml and 176 pg/ml at 20-22, 28-30 & 34-36 weeks respectively).

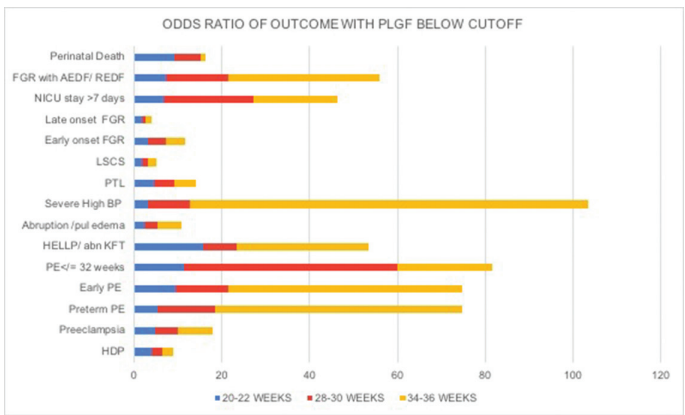


Figure 5: The odds ratio (OR) of PlGF in causing adverse outcome [11]

Nearly 30% high-risk women were found to have PlGF below the estimated cut-offs, which was similar to the study by Mclaughlin et al in which PlGF was below the cut-off (100pg/ml) in 29.5% case [10]. As the PlGF levels differ with the advancing gestation, using different cut-offs deemed more appropriate. In the study by Ormesher et al, a low PlGF (<12 pg/ml) was associated

with a shorter test-birth interval (100% PPV) [12], in our study, the women with values less than 12pg/ml constituted only 1.4% (4/287) cases and all had severe clinical features. Similarly, in the study by Mc Laughlin et al, low PlGF level was found to significantly increase the risk of preterm delivery and EO PE [adjusted odds ratio, 58.2 95% (CI, 32.1–105.4)][10]

PlGF at 28-30 weeks proved to be a useful adjunct to clinical parameters in determining risk of PE, it would predict early onset PE in 9 out of 10 cases. Low PlGF has a modest diagnostic value for predicting adverse outcome due to PE [11].

3. Soluble FMS like tyrosine kinase

Study on risk stratification by sFLT-1/PlGF in high-risk pregnant women: IIS with Roshe, we found that the sFlt-1/PlGF ratio of 38 or more at 20-22 weeks resulted in either PE or adverse fetal outcome in all cases. Whereas, the ratio of less than 38 ruled out PE in all cases up to 29^{+6} weeks. At 28-30 weeks, the ratio less than 38, predicted no PE up to 34 weeks and no complication up to 29^{+6} weeks. The sensitivity for the detection at later gestation further decreased as the gestation advanced however the specificity was above 98% at all gestations. The positive predictive value of the test increased with the advancing gestation; the negative predictive value was 93% or higher at all gestations.[7]

Previous studies have demonstrated the utility of the sFlt-1/PlGF ratio and MAP in diagnosis and prediction of PE and its adverse outcomes [9-10]. It has been found that the sFlt-1/PlGF ratio is a better predictor of EO PE compared to the late onset PE. It has also been confirmed through consensus statement that the sFlt-1/PlGF ratio is better in ruling out PE or its severe complications within one week of the test if the ratio is below the cut off of 38 [9]. In the previous studies the most appropriate time to perform the sFlt-1/PlGF ratio in a woman who was high-risk has not been specified. In the present study by doing serial testing in same women at different point of time, we found that the best time to do the test was between 28-30 weeks of gestation. Greater clinical significance of the findings of the study would be the high negative predictive value (>90%) in terms of better channelization of resources and restricting hospital admissions.

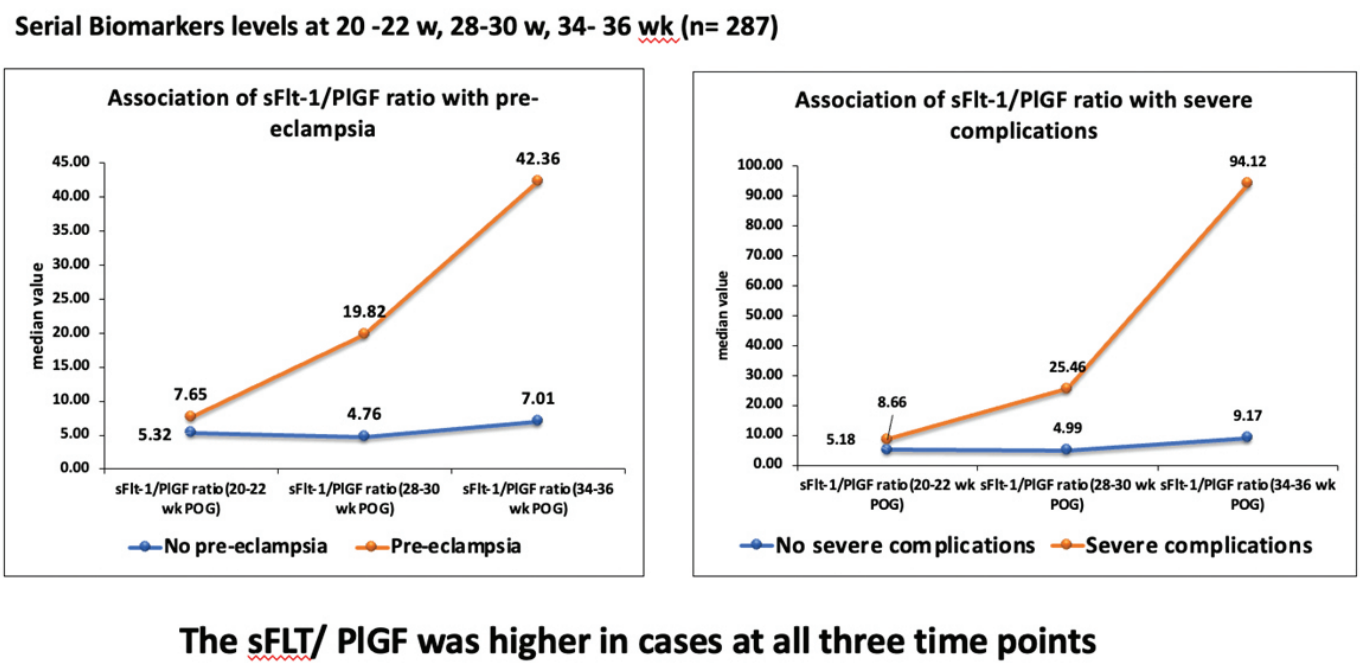


Figure 6: SFLT-1/ PlGF ratio in all three time points

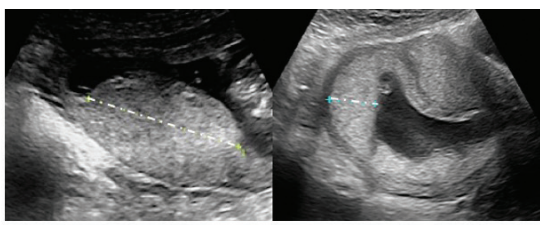
Variables	cut off	Sensitivity	Specificity
Prediction of complications till 30 weeks			
MAP at 20 w	95	100 %	65.6 %
Ut A PI at 20 w	> 95th c	71.4 %	96.4 %
sFLT/PlGF ratio 20 w	>38	100 %	98.6 %
MAP + Ut A PI at 20 w	–	100 %	67.4 %
Prediction of complications occurring till 34 weeks			
MAP at 20 w	95	50 %	94.8 %
Ut A PI at 28 w	>95th C	75 %%	83.0 %
sFLT/PlGF at 20 w	>38	50 %	98.9 %
sFLT/PlGF at 28 w	>38	62.5 %	94.8 %
MAP + Ut A PI	–	100 %	54 %0.1
Ut A PI + sFLT/PlGF at 28 w	–	100 %	79.3 %
MAP + sFLT/PlGF at 28 w	–	75 %	65.9 %
Prediction of complication occurring after 34 weeks till 36 + 6 weeks			
MAP at 20 w	95	59.1 %	68.6 %
Ut A PI at 28	>95th C	45.5 %	82.7 %
sFLT/PlGF at 20 w	>38	13.6 %	100.0 %
sFLT/PlGF at 28 w	>38	45.5 %	100 %
sFLT/PlGF at 34 w	>38	86.4 %	96.0 %
MAP + Ut PI	–	90.9 %	61.7 %
sFLT/PlGF ratio at 28 w + MAP at 20 w	–	90.9 %	71.3 %
MAP + sFLT/PlGF at 34 w	–	100 %	71.4 %
Ut A + sFLT/PlGF at 34	–	90.9 %	77.4 %
Prediction of complication occurring after 34 weeks till 40 weeks			
MAP	95	52.6 %	69.4
Ut A PI at 28w	>95th C	39.5 %	83.6 %
sFLT/PlGF at 20 w	>38	7.9 %	100 %
sFLT/PlGF at 28 w	>38	36.8 %	100 %
sFLT/PlGF at 34 w	>38	79.0 %	95.7 %
MAP + Ut PI	–	73.7 %	58.6 %
MAP + sFLT/PlGF at 34 w	–	81.3 %	68.5 %
Ut A + sFLT/PlGF at 34	–	81.3 %	81.5 %

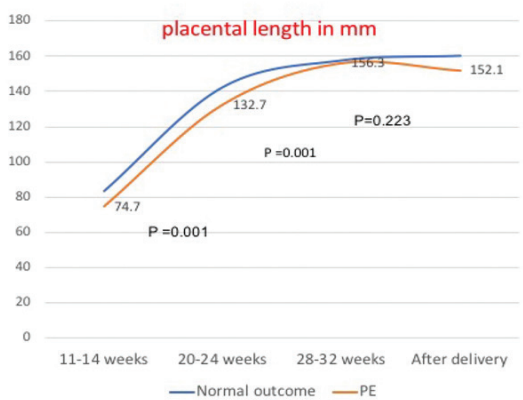
Figure 7: The cut off, sensitivity, specificity of maternal factors, doppler and sflt-1/ PlGF ratio among high-risk women for prediction of adverse pregnancy outcome

New research

1. Placental dimensions (length, breadth, thickness and volume)

In the study done to find the role of placental biometry and Doppler in predicting preeclampsia (PE) with DHR funding showed that the placental length (PL) and volume (PV) were significantly lesser in PE (p=0.005) compared to controls. The uterine artery PI (Ut A PI) at all trimesters were significantly higher in PE group (p<0.001). PL/Ut A PI and PV/Ut A PI ratio, were significantly lesser in cases compared to controls (p<0.001). In the first trimester, the area under curve (AUC), sensitivity and specificity of PV/Ut A PI for PE prediction was 0.801, 81.8% and 70.5%. At 20-24 weeks and 28-32 weeks of gestation, the sensitivity and specificity of PL/Ut PI ratio was 81.8%, 70.5% and 73.3%, 70.7% respectively.[13,14]





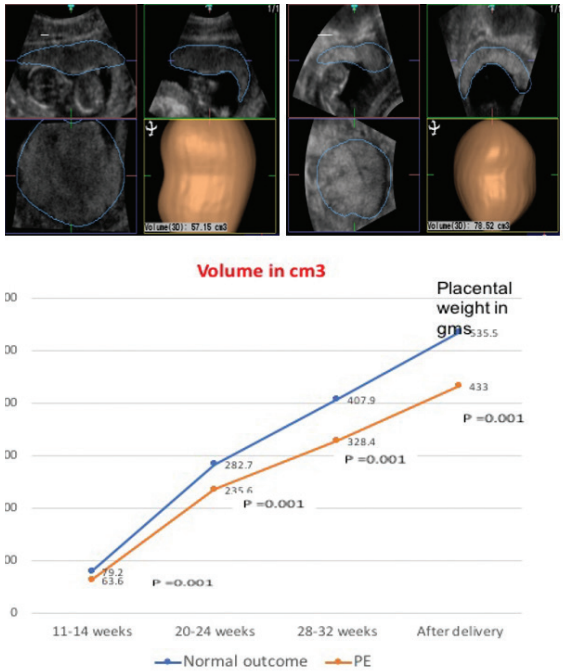


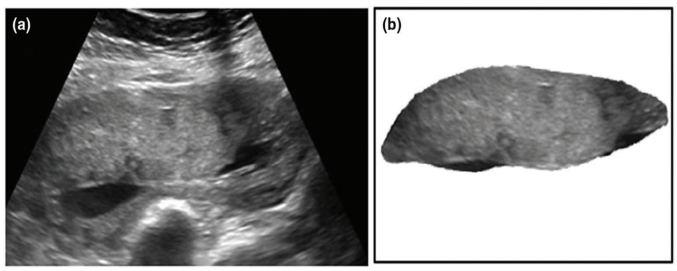
Figure 8: Placental length, breadth, thickness and volume of placenta in different trimesters in first, second and third trimesters in normal and adverse outcome.[13,14]

The predictive value of the placental profile markers in the first second and third trimesters in women with early onset FGR.

Variables	Cut - off	Sensitivity	Specificity
11–14 weeks gestation			
EFW	64.5 gms	48.8 %	67.0 %
Placental length	74.5 mm	53.5 %	70 0.3 %
Placental Volume	53.2 cm^3	51.2 %	74.0 %
PAPP-A	4000.0	46.6 %	70.4 %
PlGF	90 pg/ml	53.5 %	70.0 %
Ut A PI	1.73	50.0 %	70.2 %
PL/Ut A	44.01	51.2 %	70.1 %
PV/Ut A	36.05	60.5 %	70.1 %
EFW/Ut PI	39.95	46.5	70.3
MAP + PL/Ut A	90 mmHg + 44.01	70 %	49.3 %
20–24 weeks gestation			
EFW	503 gms	61.9 %	70.1 %
Placental Length	132.5 mm	40.5 %	70.6 %
Placental Volume in 3D	217.3 cm^3	50.0 %	70.1 %
PlGF	268.8 pg/ml	47.6 %	70.1 %
sFLT -1	If rises at 20–24 weeks	59.5 %	58.1 %
UA PI	1.2	48.6 %	69.6 %
Ut PI	1.08	42.9 %	71.4 %
PL/Ut A	130.4	64.3 %	70.1 %
PV/Ut A	225.5	66.7 %	70.1 %

2. AI model - Application of deep learning on placental Image texture[15,16]

A total of 1008 cases were fully followed, and 46.7% (471/1008) of them had a normal outcome. A total of 600 images from each trimester among the controls were used for analysis. This analysis included the image classification for first-second, second-third, and first-third trimester pairs. Out of all the models, the Inception v3 model achieved an accuracy of 83.3%, a Cohen-kappa score of 0.662, a sensitivity of 78.7%, and a specificity of 91.1% for distinguishing between first and second trimester images. For classifying second and third trimester images, the Efficient Net B0 model achieved an accuracy of 69.2%, a Cohen-kappa score of 0.383, a sensitivity of 80.0%, and a specificity of 65.0%. Finally, for first and third trimester images, the EfficientNetB0 model achieved an accuracy of 87.5%, a Cohen-kappa score of 0.749, a sensitivity of 83.4%, and a specificity of 88.9%. Study presented a novel technique to classify placental ultrasound image texture. Images of each trimester were classified using artificial intelligence (AI). Images of 1st and 3rd were best classified using transfer learning.



Sensitivity and Specificity for best model of trimester classification and for prediction of adverse pregnancy outcome.

Prediction	Trimester classification among controls with normal outcome			Predictive value for adverse maternofetal outcome		
	T1 & T2	T2 & T3	T1 & T3	T1	T2	T3
Sensitivity (%)	78.67	80.00	86.36	77.42	75.00	81.03
Specificity (%)	91.11	65.00	88.89	80.21	85.07	93.94
PLR	4.27	3.25	6.52	3.91	5.03	13.37
NLR	0.11	0.44	0.13	0.28	0.29	0.20
Positive Predictive Value (%)	71.93	86.67	84.21	71.64	81.82	92.16
Negative Predictive Value (%)	93.65	53.33	90.48	84.62	79.17	84.93

This technique proved good in differentiating normal outcome and adverse pregnancy outcome

Key Points

- PAPP-A a metalloproteinase insulin-like growth factor (IGF) plays an important role in placental growth and development. A low level of circulating PAPP-A has been shown to be associated with PE.
- Placental growth factor (PlGF) is the most validated and studied biomarker of PE screening. PlGF levels decrease substantially 5 weeks before the onset of PE.
- All pregnant women should be screened at 11–14 weeks for preterm preeclampsia by the first-trimester combined test with maternal risk factors, mean arterial pressure, uterine artery pulsatility index, and placental growth factor as available, even if they have been already been identified as having clinical 'high-risk' factors (ISSHP 2021)
- PlGF along with MAP and uterine artery Doppler is known as triple test for PE. It has got highest sensitivity for detecting preeclampsia before 32 weeks.
- Alternative screening method i.e. contingent screening method includes assessment of PlGF levels only in

women with a significant positive history or raised MAP or raised uterine artery pulsatility index.

- Given the resource constraints in low/middle-income countries, variations of the first trimester combined test should be considered but the baseline test should be maternal risk factors combined with mean arterial pressure.
- In high-risk women, defined by the first-trimester combined test, aspirin in the dose of 150 mg/night should be commenced at 11–14+6 weeks of gestation until either 36 weeks of gestation when delivery occurs, or when preeclampsia is diagnosed.
- Extremely elevated sFlt-1/PlGF values have been shown to be closely related to the need for immediate delivery. The sFlt-1/PlGF ratio allows the identification of women at high risk for imminent delivery.
- The knowledge of high-risk of PE would help in increasing surveillance in those who would be high risk, and delayed delivery in women who were low risk
- It has been found that the sFlt-1/PlGF ratio is a better predictor of EO PE compared to the late onset PE

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Aspirin for Hypertension in Pregnancy: A Comprehensive Review

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Introduction

Hypertensive disorders of pregnancy are a leading cause of maternal and perinatal morbidity and mortality worldwide. Preeclampsia, in particular, remains a formidable obstetric challenge. Over the past two decades, low-dose aspirin has emerged as an effective prophylactic intervention to reduce the risk of preeclampsia and associated complications. This chapter provides a detailed review of the rationale, evidence, recommendations, and practical aspects of aspirin use for hypertension in pregnancy.

Hypertensive Disorders in Pregnancy

Hypertensive disorders affect approximately 5–10% of all pregnancies. They encompass:

- **Chronic hypertension**: pre-existing or diagnosed before 20 weeks.
- **Gestational hypertension**: new-onset hypertension after 20 weeks without proteinuria.
- **Preeclampsia**: hypertension after 20 weeks with proteinuria or evidence of maternal organ dysfunction or uteroplacental insufficiency.
- **Eclampsia**: seizures in a woman with preeclampsia.
- **Superimposed preeclampsia**: preeclampsia in a woman with chronic hypertension.

Preeclampsia remains the most important hypertensive complication, accounting for significant perinatal morbidity (intrauterine growth restriction, prematurity) and maternal morbidity (eclampsia, HELLP syndrome, stroke).

Pathophysiology of Preeclampsia and Rationale for Aspirin Use

Preeclampsia is a multisystem disorder originating in early placental development. The underlying pathophysiology includes:

1. **Abnormal placentation**: Failure of trophoblastic invasion leads to incomplete remodeling of spiral arteries. This results in high-resistance uteroplacental circulation and placental ischemia.
2. **Endothelial dysfunction**: Hypoxic placenta releases antiangiogenic factors (sFlt-1) and pro-inflammatory mediators, causing widespread maternal endothelial injury.
3. **Prothrombotic state**: Increased platelet aggregation and thromboxane A2 (TxA2) production contribute to microthrombi in the placental vasculature.

Prostaglandin imbalance is a key component:

- **Thromboxane A2** promotes vasoconstriction and platelet aggregation.
- **Prostacyclin (PGI2)** promotes vasodilation and inhibits platelet aggregation.

In preeclampsia, **TxA2 increases while PGI2 decreases**—creating a vasoconstricted, prothrombotic environment.

Aspirin, at low doses, selectively inhibits platelet cyclooxygenase-1 (COX-1), reducing TxA2 synthesis and restoring the TxA2–PGI2 balance. This mechanistic rationale underpins its prophylactic use.

Pathophysiology of Aspirin

Aspirin (acetylsalicylic acid) is a nonsteroidal antiinflammatory drug (NSAID) that works primarily through its inhibition of two cyclooxygenase isoenzymes (**COX-1 and COX-2**), which are necessary for prostaglandin biosynthesis. The COX-1 isoform is present in the vascular endothelium and regulates the production of prostacyclin and thromboxane A_2, prostaglandins with opposing regulatory effects on vascular homeostasis and platelet function. Prostacyclin is a potent vasodilator and inhibitor of platelet aggregation, whereas thromboxane A_2 (TXA2) is a potent vasoconstrictor and promotes platelet aggregation. The COX-2 isoform is inducible and expressed almost exclusively following exposure to cytokines or other inflammatory mediators. The effect of aspirin on COX-dependent prostaglandin synthesis is **dose dependent**. At lower dosages (60–150 mg/day) aspirin irreversibly acetylates COX-1, resulting in decreased platelet synthesis of TXA2 without affecting vascular wall production of prostacyclin. At higher doses, aspirin inhibits both COX-1 and COX-2, effectively blocking all prostaglandin production.

Evidence suggesting that an imbalance in prostacyclin and **TXA2 metabolism was involved in the development** of preeclampsia prompted the initial studies of aspirin for preeclampsia prevention because of its preferential inhibition of TXA2 at lower doses. However, it is likely that preeclampsia is a result of poor placentation from a variety of causes, including ischemia, reperfusion, or dysfunctional maternal inflammatory response towards the trophoblast. Whether low-dose aspirin improves early placental

perfusion is unknown, and likewise, the precise mechanism by which low-dose aspirin prevents preeclampsia in some women is also uncertain

Recommendations

The American College of Obstetricians and Gynaecologists (ACOG) and the Society for Maternal–Fetal Medicine make the following recommendations:

- Low-dose aspirin (81 mg/day) prophylaxis is recommended in women at high risk of preeclampsia and should be initiated between 12 weeks and 28 weeks of gestation (optimally before 16 weeks) and continued daily until delivery.
- Low-dose aspirin prophylaxis should be considered for women with more than one of several moderate risk factors for preeclampsia.
- Low-dose aspirin prophylaxis is not recommended solely for the indication of prior unexplained stillbirth, in the absence of risk factors for preeclampsia.
- Low-dose aspirin prophylaxis is not recommended for prevention of fetal growth restriction, in the absence of risk factors for preeclampsia.
- Low-dose aspirin prophylaxis is not recommended for the prevention of spontaneous preterm birth, in the absence of risk factors for preeclampsia.
- Low-dose aspirin prophylaxis is not recommended for the prevention of early pregnancy loss.

Historical Context and Evolution of Evidence

The idea of aspirin prophylaxis for preeclampsia emerged in the late 1970s when researchers observed that prostaglandin imbalance was a hallmark of the disease. Early small trials produced conflicting results.

Over time, larger randomized controlled trials and meta-analyses consistently demonstrated a modest but meaningful reduction in preeclampsia risk.

Some important milestones:

- **CLASP Trial (1994):** One of the largest early trials of low-dose aspirin in pregnancy. It showed a 12% relative reduction in preeclampsia risk, though results were not statistically significant across all subgroups.
- **Subsequent meta-analyses:** Pooled data indicated a 10–15% reduction in preeclampsia and better outcomes in high-risk women.
- **ASPRE Trial (2017):** A landmark multicenter randomized trial. In 1776 high-risk women identified by first-trimester screening, aspirin 150 mg daily started before 16 weeks reduced preterm preeclampsia by 62%.

This accumulating evidence has led to strong recommendations in guidelines globally.

Guidelines and Recommendations

Multiple authoritative bodies now endorse low-dose aspirin prophylaxis in selected pregnant women at increased risk of preeclampsia.

American College of Obstetricians and Gynecologists (ACOG):

- Recommends **81 mg daily aspirin** for women with ≥1 high-risk factor or ≥2 moderate-risk factors.
- Initiate between 12–28 weeks, optimally before 16 weeks, continue until delivery.

National Institute for Health and Care Excellence (NICE, UK):

- Recommends **150 mg daily aspirin** for high-risk women from **12 weeks until birth**.

Royal College of Obstetricians and Gynaecologists (RCOG):

- Advises **150 mg daily aspirin**, starting from 12 weeks.

WHO:

- Recommends **75 mg aspirin daily** starting before 20 weeks in women at high risk.

FIGO:

- Endorses universal first-trimester screening and aspirin prophylaxis in high-risk women.

Indications for Aspirin Prophylaxis

Risk stratification is essential. ACOG criteria are widely referenced:

High-risk factors (any one):

- History of preeclampsia (especially severe or preterm).
- Multifetal gestation.
- Chronic hypertension.
- Type 1 or 2 diabetes.
- Chronic kidney disease.
- Autoimmune diseases (e.g., antiphospholipid antibody syndrome, SLE).

Moderate-risk factors (≥2):

- Nulliparity.
- Obesity (BMI ≥30).
- Family history of preeclampsia.
- Maternal age ≥35 years.
- Socioeconomic disadvantage.

- Personal history (e.g., >10-year interval between pregnancies).

Women meeting these criteria should be offered prophylactic aspirin.

Dosage and Timing

Dose:

- Most guidelines recommend 100–150 mg daily.
- ASPRE used 150 mg night time.

Timing of initiation:

- Before 16 weeks gestation is critical, as placentation completes by this time.
- The greatest benefit is seen when started between 12–16 weeks.

Duration:

- Continued until 36 weeks gestation or delivery.

Administration:

- Taken preferably at night to optimize pharmacodynamics.

Efficacy and Impact

Meta-analyses, including the Cochrane review, show that low-dose aspirin:

- Reduces the risk of preeclampsia by ~10–15%.
- Reduces preterm preeclampsia by ~50% if started early.
- Lowers the risk of intrauterine growth restriction and preterm birth.
- Modestly reduces perinatal mortality.

ASPRE results:

- Preterm preeclampsia incidence: 1.6% (aspirin) vs. 4.3% (placebo).
- No significant increase in major bleeding.

Safety Considerations

Low-dose aspirin is generally considered safe in pregnancy when used appropriately:

Maternal safety:

- No increase in placental abruption.
- No significant postpartum hemorrhage increase in most studies.
- Minimal gastrointestinal side effects.

Fetal safety:

- No teratogenicity.
- No increased risk of ductus arteriosus constriction at low doses.
- No effect on neurodevelopment.

Important caution:

- **High-dose aspirin (>300 mg/day) is contraindicated**, especially in the third trimester, due to:
  - Ductus arteriosus constriction.
  - Oligohydramnios.
  - Platelet dysfunction in the fetus.

Contraindications:

- Aspirin allergy.
- Active peptic ulcer disease.
- Bleeding disorders.
- Severe thrombocytopenia.

Practical Implementation

To integrate aspirin prophylaxis effectively:

1. Identify at-risk women early—ideally at the booking visit.
2. Counsel about benefits, safety, and importance of adherence.
3. Prescribe appropriate dose (150 mg daily).
4. Document initiation and review compliance.
5. Monitor for any bleeding complications.

Special Populations

Women with chronic hypertension:

- Aspirin significantly reduces the risk of superimposed preeclampsia.
- Should be commenced early and combined with tight BP control.

Women with antiphospholipid syndrome:

- Aspirin is used in combination with prophylactic LMWH to improve pregnancy outcomes.
  - **LDA alone**: may be considered for asymptomatic aPL-positive women.
  - **LDA + prophylactic LMWH**: recommended for women with obstetric APS.
  - **LDA + therapeutic LMWH**: used for women with prior thrombosis or severe APS.
- Hydroxychloroquine (HCQ) is increasingly considered, especially in refractory cases.

Multiple gestation:

- Higher baseline risk of preeclampsia warrants prophylactic aspirin.

Controversies and Debates

- **Optimal dose:** While 81 mg is standard in the U.S., ASPRE used 150 mg. Some experts advocate higher doses for greater efficacy.
- **Universal vs. targeted prophylaxis:** Some propose offering aspirin to all pregnant women, but current evidence supports selective prophylaxis.
- **Role of biomarkers:** First-trimester screening combining uterine artery Doppler, maternal characteristics, and biochemical markers is increasingly advocated but is not yet universally implemented.

Future Directions

Ongoing research is exploring:

- Personalized dosing strategies.
- Combining aspirin with other interventions (e.g., calcium supplementation).
- Improved risk prediction algorithms incorporating angiogenic factors.
- Longer-term follow-up of offspring to assess subtle neurodevelopmental effects.

Newly added (from Biomarker Scoping Review – Al-Khulaifi et al., 2024):

- **Aspirin responsiveness biomarkers** are an emerging area of interest.
- The 2024 scoping review identified that **no reliable biomarkers** currently exist to assess how well aspirin works in pregnancy.
- Future studies are needed to validate tests like **thromboxane B2 levels or platelet aggregation assays** to guide **dose adjustments** or detect non-responders.
- This points toward a future of **individualized aspirin prophylaxis** based on objective response data.

Conclusion

Low-dose aspirin prophylaxis is a cornerstone strategy to prevent preeclampsia and related hypertensive complications in pregnancy. Its efficacy is greatest when initiated early in high-risk women, and safety is well established at low doses. Obstetric care providers should proactively identify eligible women, counsel them effectively, and ensure adherence to this simple, evidence-based intervention.

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Adaptations of the Human Placenta to Hypoxia: Opportunities for Interventions in FGR

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Introduction

The placenta is a unique, transient organ essential for the survival and development of the fetus. Acting as the vital interface between mother and fetus, it facilitates the exchange of oxygen, nutrients, and waste products while also producing hormones that adjust maternal physiology to meet the demands of pregnancy.[1] The evolution of the placenta marked a defining milestone in mammalian biology, enabling prolonged in-utero development and ensuring offspring are born at a more advanced stage of maturity.[2]

The growth of the placenta and fetus are intimately connected, and the impact of this partnership extends well beyond the antenatal period. A successful placental-fetal relationship is crucial in shaping long-term health outcomes, including cardiovascular development during childhood and beyond.[3] Far from being a passive conduit, the placenta is a highly dynamic organ — capable of sensing the fetus's needs and adapting to changes within the intrauterine environment to support healthy growth and development.[2]

Placental Development

Early placental development begins with the invasion of trophoblast cells and the remodelling of maternal spiral arteries, a process essential for establishing a low-resistance uteroplacental circulation. This critical phase, occurring within the first trimester, anchors the placenta to the uterine wall and initiates the exchange of nutrients and gases between mother and fetus.[2]

In early pregnancy, the developing embryo exists within a naturally low-oxygen environment (physiological hypoxia) due to the absence of substantial maternal blood flow, both at the site of fertilization in the fallopian tube and later at the implantation site within the endometrial cavity (Figure 1). Rather than being harmful, this state serves protective functions — shielding the embryo from reactive oxygen species (ROS) during the vulnerable period of organogenesis and potentially preserving the pluripotency of embryonic stem cells.[4]

As gestation progresses, the metabolic and oxygen requirements of the fetus increase substantially to support rapid somatic growth, organ development, and functional maturation. To accommodate these rising demands, the mechanism of nutrient and oxygen delivery transitions from histotrophic — reliant on uterine gland secretions — to hemotrophic, involving direct perfusion of maternal blood through the intervillous space of the placenta.[2] This shift marks a crucial phase in placental function, ensuring that the fetus receives an adequate and continuous supply of oxygen and nutrients necessary for healthy growth.

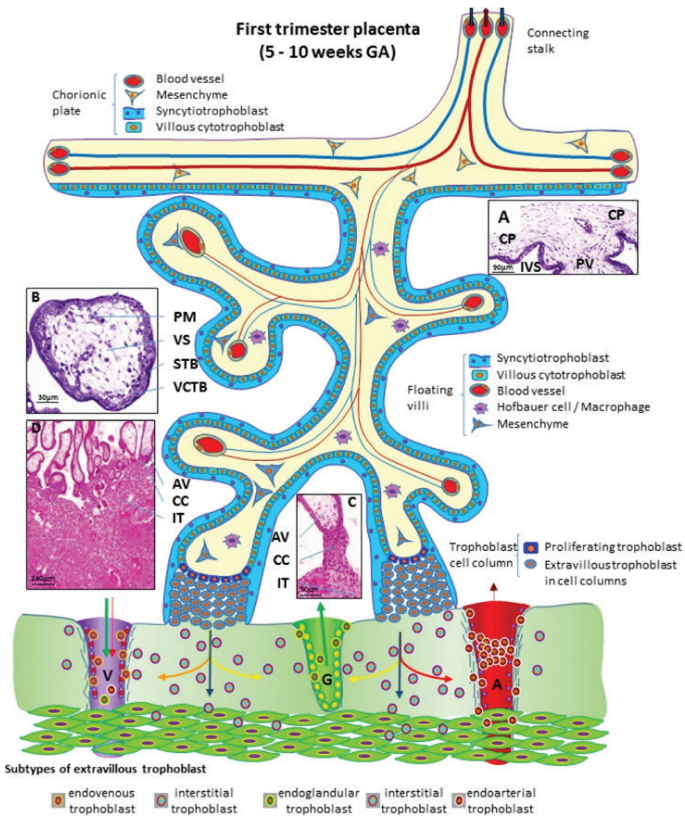

Figure 1. Schematic representation of a human placenta during the first trimester of pregnancy. Histological images of (A) first trimester chorionic plate with a placental villus extending into the intervillous space, (B) first trimester mesenchymal villus with the cover of villous trophoblast and the mesenchymal villous stroma, (C) anchoring villus that is attached to the uterine wall by a trophoblast cell column, (D) first trimester placenta showing a number of anchoring villi attached to the uterine wall by trophoblast cell columns. A, uterine spiral artery; AV, anchoring villus; CC, trophoblast cell column; CP, chorionic plate; G, uterine gland; GA, gestational age; IT, interstitial trophoblast; IVS, intervillous space; PM, placental macrophage (Hofbauer cell); PV, placental villus; STB, syncytiotrophoblast; V, uterine vein; VCTB, villous cytotrophoblast; VS, villous stroma.

Source: Wikimedia Commons

On the maternal side, this transition is facilitated by progressive and extensive remodelling of the uterine vasculature. The arcuate and radial arteries undergo

significant dilation throughout pregnancy, a process regulated by a complex interplay of hormonal influences, including estrogen, progesterone, human chorionic gonadotropin (hCG), and several locally acting placental-derived factors. These hormonal signals promote vascular smooth muscle relaxation and extracellular matrix remodelling, contributing to increased vessel diameter and reduced vascular resistance.[5]

By approximately 20 weeks of gestation, this remodelling is so advanced that the diameter of the arcuate arteries approaches that of the main uterine artery, effectively creating a low-resistance, high-capacity uteroplacental circulation capable of sustaining the growing demands of the fetoplacental unit.[6]

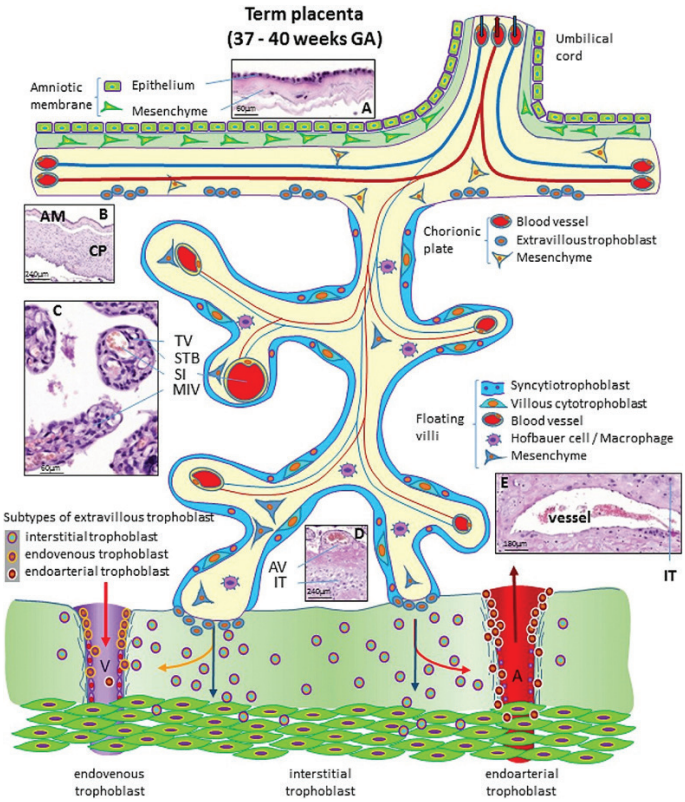

Figure 2. Schematic representation of a human placenta at term. Histological images of (A) term amniotic membrane with epithelium and avascular mesenchyme, (B) term chorionic plate covered by the amniotic membrane, (C) placental villi of a term placenta with a sinusoid in a terminal villus and a neighbouring mature intermediate villus, (D) anchoring villus that is attached to the uterine wall where interstitial trophoblast can be found, (E) vessel in the basal plate of a term placenta. The vessel is surrounded by interstitial trophoblast. A, uterine spiral artery; AM, amniotic membrane; AV, anchoring villus; CP, chorionic plate; GA, gestational age; IT, interstitial trophoblast; MIV, mature intermediate villus; SI, sinusoid; STB, syncytiotrophoblast; TV, terminal villus; V, uterine vein.

Source: Wikimedia Commons

On the fetal side, the placenta responds adaptively to the escalating demands of late gestation through structural and vascular modifications. The villous tree undergoes progressive on the branching and elongation, markedly increasing the surface area available for maternal-fetal exchange. Concurrently, extensive angiogenesis occurs within the terminal villi, enhancing the density and complexity of the fetoplacental capillary network. These adaptations are essential to maintain efficient nutrient and gas exchange as fetal metabolic needs rise exponentially during the third trimester (Figure 2).[7]

Together, these dynamic and highly regulated changes on both the maternal and fetal sides of the placenta ensure optimal support for the developing fetus. A thorough understanding of these processes is fundamental for recognizing pregnancies at risk of placental insufficiency and developing strategies for early detection and intervention.[6]

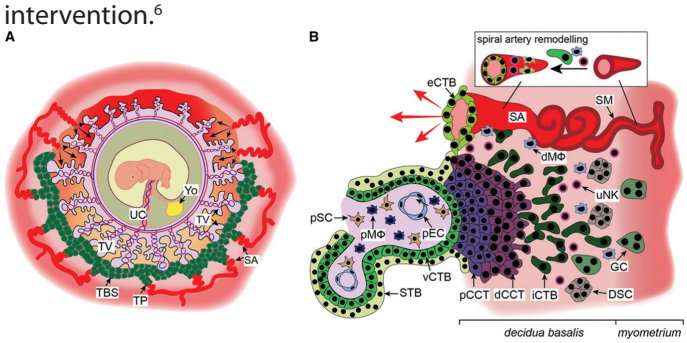

Figure 3. Development of the trophoblastic shell and formation of placental anchoring villi. A, Structure of the human trophoblastic shell and its surrounding arterial vessels. B, Depiction of a placental anchoring villus, spiral artery (SA) remodelling and interaction of extravillous trophoblasts (EVTs) with different decidual cell types. dCCT distal cell column trophoblast, dM decidual macrophage, DSC decidual stromal cell, eCTB endovascular cytotrophoblast, GC giant cell, iCTB interstitial cytotrophoblast, pCCT proximal cell column trophoblast, pEC placental endothelial cell, pM placental macrophage, pSC placental stromal/mesenchymal cell, SM smooth muscle layer, STB syncytiotrophoblast, TBS trophoblastic shell, TP trophoblast plug, TV tertiary villi, UC umbilical cord, uNK uterine NK cell, vCTB villous cytotrophoblast, YO yolk sac.

Source: Wikimedia Commons

Placenta and Fetal Growth Restriction

Fetal growth restriction (FGR) affects approximately 5–10% of pregnancies and is linked with higher rates of perinatal mortality and morbidity, including neurodevelopmental impairment, respiratory distress, and neonatal infections. From a long-term perspective, the implications of FGR extend beyond the immediate perinatal period. Epidemiological and experimental studies have consistently demonstrated associations between FGR and an increased risk of cardiovascular, metabolic, and neurodevelopmental disorders in later life.[7]

Though FGR is commonly attributed to placental insufficiency, the association between abnormal placental development and fetal growth restriction (FGR) is complex

and not entirely well understood. Impaired trophoblastic invasion, inadequate spiral artery remodelling, and suboptimal villous angiogenesis are central features of placental insufficiency, which underlies not only FGR but several other significant obstetric complications such as preeclampsia, preterm birth, and stillbirth (Figure 3). (2)

Placental Adaptations to Hypoxia

A central feature of placental dysfunction is reduced oxygen availability to the fetus. Since oxygen is essential for fetal growth, organ development, and survival, the placenta has evolved multiple adaptive mechanisms to maintain fetal viability under hypoxic conditions. In response to decreased oxygen supply, the placenta initiates structural, vascular, metabolic, and molecular modifications aimed at preserving critical nutrient and gas exchange, often prioritizing fetal survival at the expense of optimal growth and long-term health.

However, in pathological states such as fetal growth restriction (FGR), these adaptations may become maladaptive. Instead of restoring functional balance, they can further compromise placental efficiency, exacerbate hypoxia, and restrict fetal growth, perpetuating a cycle of dysfunction. (6)

1. Structural Adaptations

In hypoxic conditions, the placenta exhibits reduced villous branching, which diminishes the available surface area for maternal-fetal exchange (Mayhew, 2002). Additionally, there is an increase in syncytial knots — aggregates of syncytiotrophoblast nuclei — which serve as histological markers of oxidative stress and accelerated placental aging. (8). Thickening of the trophoblastic basement membrane has also been observed, further limiting oxygen diffusion and contributing to fetal hypoxemia. (6)

2. Vascular Remodelling

Hypoxia disrupts the balance of pro- and anti-angiogenic factors. While there is an upregulation of vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) to promote angiogenesis, this is counteracted by an increase in soluble fms-like tyrosine kinase-1 (sFlt-1), an anti-angiogenic molecule that binds and neutralizes VEGF and PlGF. (9) Elevated levels of sFlt-1, a hallmark of both preeclampsia and FGR, contribute to endothelial dysfunction and placental hypoperfusion. Concurrently, defective remodelling of the maternal spiral arteries — a structural hallmark of these conditions — further exacerbates the hypoxic intrauterine environment. (10)

Under hypoxic stress, the placenta shifts its metabolism from oxidative phosphorylation to anaerobic glycolysis to maintain ATP production, although this is a less efficient energy source. Mitochondrial dysfunction in this setting leads to excessive generation of reactive oxygen species (ROS), contributing to oxidative stress, DNA damage, and further impairment of placental function. (11)

4. Gene Expression and Epigenetic Modifications

A key regulator of the placental response to hypoxia is Hypoxia-Inducible Factor-1α (HIF-1α). Stabilized under low-oxygen conditions, HIF-1α activates transcriptional programs that support cell survival, modulate angiogenesis, and regulate nutrient transport. (12) In addition, chronic hypoxia induces epigenetic modifications, including alterations in DNA methylation and histone modifications. These changes can reprogram placental and fetal gene expression, with potential long-term effects on fetal development and susceptibility to diseases in later life. (13)

Opportunities for Intervention In Fgr

An improved understanding of the mechanisms underlying placental hypoxia and maladaptation has paved the way for exploring targeted interventions to improve outcomes in pregnancies complicated by fetal growth restriction (FGR). While most strategies remain investigational, several promising therapeutic avenues are emerging:

1. Antioxidant Therapy

Oxidative stress plays a central role in the pathogenesis of FGR, contributing to placental dysfunction and impaired fetal growth. Various antioxidants, including melatonin, vitamins C and E, and mitochondrial-targeted agents like MitoQ, have been investigated for their potential to mitigate oxidative damage. Among these, melatonin has demonstrated antioxidant, anti-inflammatory, and anti-apoptotic properties, with some evidence suggesting improved placental efficiency in experimental models. (14) However, clinical trials in humans have produced inconsistent outcomes, largely due to challenges with optimal dosing, timing of administration, and bioavailability. Recent research suggests that combination antioxidant therapies or targeted delivery systems may enhance therapeutic efficacy.

2. Enhancing Uteroplacental Perfusion

Improving uteroplacental blood flow represents a rational approach to alleviating hypoxic stress in FGR. Sildenafil citrate, a phosphodiesterase-5 inhibitor, has shown promise in increasing uterine artery blood flow in animal studies. However, results from the large multicentre STRIDER trials in humans failed to demonstrate significant fetal benefits and raised safety concerns, leading to early termination. (15) Other agents, such as nitric oxide (NO) donors like glyceryl trinitrate patches, are under ongoing investigation for their vasodilatory effects on the uterine circulation, though definitive clinical evidence remains lacking.

3. Modulating Angiogenic Balance

Disruption in angiogenic regulation—characterized by

elevated soluble fms-like tyrosine kinase-1 (sFlt-1) and reduced placental growth factor (PlGF)—is a hallmark of placental insufficiency. Therapeutic apheresis to reduce circulating sFlt-1 levels has shown potential in the management of preeclampsia and may offer future applications in severe FGR.[16] Additionally, statins, particularly pravastatin, have demonstrated the capacity to restore angiogenic balance and reduce systemic inflammation. Early-phase clinical studies report promising results, though larger randomized trials are needed to confirm safety and efficacy.[17]

4. Gene and Epigenetic Therapies

Emerging research is exploring the manipulation of epigenetic and gene regulatory mechanisms involved in placental adaptation to hypoxia. Modulating the expression of Hypoxia-Inducible Factor-1α (HIF-1α), a key regulator of the hypoxic response, has shown promise in promoting angiogenesis and improving placental function. Preclinical studies using epigenetic editing tools (e.g., CRISPR-dCas9 systems) and small molecules that target DNA methylation or histone modifications offer potential for disease-modifying interventions in FGR.[18] These approaches, while still in experimental stages, hold considerable promise for future translation into clinical practice.

5. Nutritional Interventions

Maternal nutrition exerts a direct influence on placental function and fetal growth. Supplements such as L-arginine, a nitric oxide precursor, have been associated with improved uteroplacental circulation and fetal growth in certain cases.[19] Omega-3 fatty acids may enhance endothelial function and possess anti-inflammatory properties beneficial to the placental vasculature. Additionally, micronutrients like iron, folate, and vitamin D play supportive roles in maintaining placental health. Importantly, nutritional strategies should be individualized, taking into account the underlying etiology of FGR—whether maternal undernutrition, placental insufficiency, or genetic factors.

Future Perspectives

Despite advances in understanding the pathophysiology of FGR, effective therapeutic options remain limited, with current management largely focused on surveillance and timing of delivery to optimize perinatal outcomes. The increasing recognition of the placenta as both a diagnostic and therapeutic target offers exciting opportunities for future research and clinical innovation.

Emerging strategies such as gene editing, targeted drug delivery systems, and epigenetic therapies hold potential for directly modifying maladaptive placental responses to hypoxia, potentially restoring normal function rather than merely prolonging pregnancy.[20]

Preclinical studies using CRISPR-based technologies and small molecule epigenetic modifiers are already providing proof of concept for such interventions, though translation to human pregnancies will require careful assessment of safety, feasibility, and ethical considerations.

Advancements in placental imaging and circulating biomarkers, including cell-free RNA, exosomes, and novel angiogenic profiles, could enable earlier and more precise identification of at-risk pregnancies, facilitating timely, individualized therapeutic strategies. Integration of these tools into routine prenatal care would represent a significant shift from reactive to proactive management of FGR.[21]

Furthermore, combination therapies that simultaneously target oxidative stress, angiogenic imbalance, and metabolic dysfunction may offer synergistic benefits, particularly if delivered through nanocarrier systems capable of selectively targeting the placenta while minimizing fetal exposure.[22]

Conclusion

The placenta exhibits a remarkable capacity to adapt to hypoxic stress through a complex network of structural, vascular, metabolic, and molecular responses. While many of these adaptations are initially protective, they can contribute to poor fetal growth, preterm birth, and adverse long-term health outcomes when they become maladaptive. A deeper understanding of these mechanisms offers valuable opportunities for the development of diagnostic tools and targeted therapies for pregnancies at risk of fetal growth restriction (FGR).

However, the clinical translation of these insights remains challenging, particularly in balancing the timing, safety, and efficacy of emerging interventions. Although no single, definitive therapy for FGR currently exists, ongoing research into antioxidant, vascular, angiogenic, gene-based, and nutritional interventions presents a multi-faceted therapeutic horizon.

In summary, meaningful progress in the prevention and management of FGR will depend on a multidisciplinary, translational approach that bridges basic science, clinical research, and technological innovation. Continued investment in collaborative, large-scale studies and the development of precision, placenta-targeted therapies hold promise for improving both short- and long-term outcomes for affected pregnancies.

Key Takeaways

- The placenta adapts to hypoxia through structural, vascular, metabolic, and molecular responses — initially protective but potentially maladaptive.
- These changes can contribute to fetal growth restriction (FGR), preterm birth, and long-term health risks.
- Understanding placental adaptations offers

opportunities for new diagnostic tools and targeted therapies.

- Clinical translation faces challenges of timing, safety, and efficacy.
- No single therapy exists yet, but antioxidant, vascular, angiogenic, gene-based, and nutritional strategies show promise.
- A multidisciplinary, translational approach integrating basic science, clinical research, and technology is essential for future progress.

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Placenta in gestational Diabetes Mellites

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Introduction

Gestational diabetes mellitus (GDM) is generally defined as "any degree of glucose tolerance with onset or first recognition during pregnancy". It currently is one of the diseases with the highest morbidity among pregnant women. Globally, its prevalence ranges from 1% to 14%. About 50% of women diagnosed with GDM during pregnancy will develop type 2 diabetes mellitus (T2DM) in the future. Furthermore, fetuses from mothers with GDM can present with short-term complications, such as macrosomia, shoulder dystocia and neonatal hypoglycaemia. These children also have a greater risk of developing obesity and T2DM in adulthood.

The Placenta: Structure and Function

The placenta is a unique organ that forms the interface between mother and fetus. The placenta has a fetal side (chorionic plate) and a maternal side (basal plate). The villous trees, covered by the cytotrophoblast and syncytiotrophoblast, are the main sites for exchange. The endocrine function of the placenta is to produce a variety of hormones (e.g., hPL, PGH, leptin) and cytokines that regulate maternal metabolism, immune tolerance, and fetal growth.

Hormonal and Molecular Changes in GDM

Physiological Insulin Resistance vs. GDM

- **Normal Pregnancy:** Early pregnancy is characterized by increased insulin sensitivity, which shifts to insulin resistance (up to 60% decrease) in the second and third trimesters. This ensures an adequate glucose supply to the fetus. The maternal pancreas compensates by increasing insulin secretion through beta-cell hyperplasia and hypertrophy.
- **In GDM:** The maternal pancreas fails to compensate for increased insulin resistance, resulting in hyperglycemia.

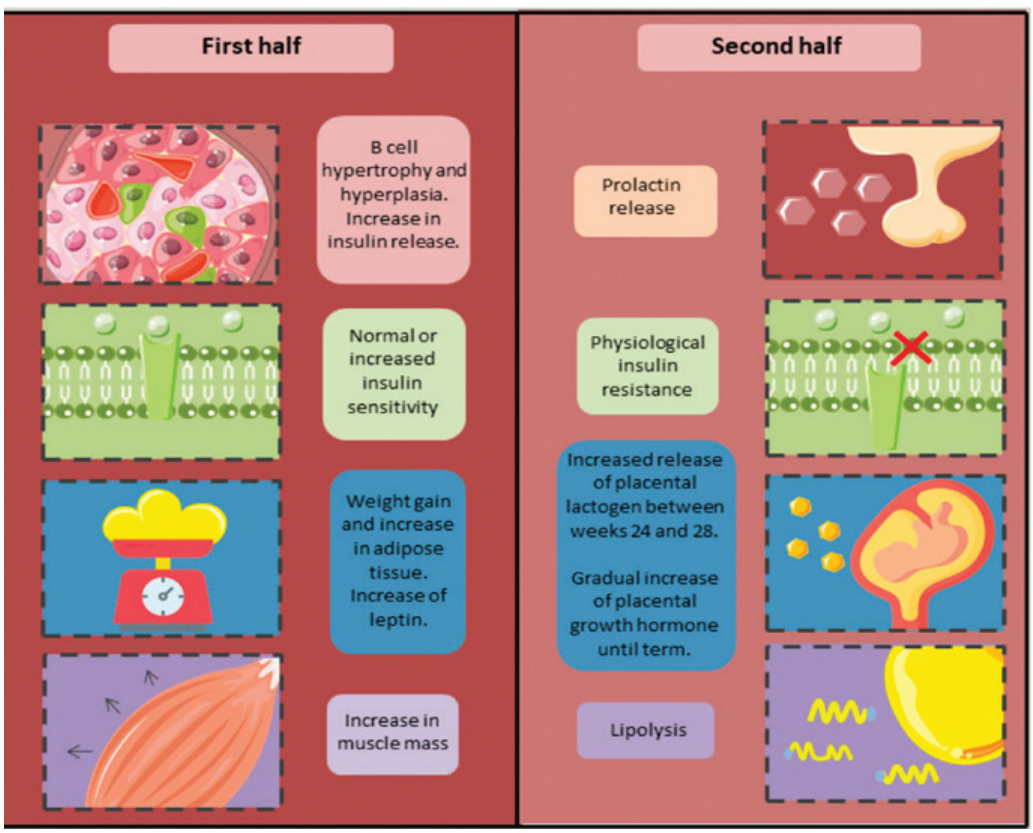


Figure 1: Physiological changes in macromolecule metabolism during first and second half of pregnancy6
Adapted from https://touchendocrinology.com/diabetes/journal-articles/the-placental-role-in-gestational-diabetes-mellitus-a-molecular-perspective/

Key Placental Hormones and Their Roles

- **Placental Growth Hormone (PGH)**

  PGH is made by the placenta starting around week 13 of pregnancy and takes over from the mother's own growth hormone. It helps the placenta grow and increases blood flow, but also causes the mother's body to become more resistant to insulin (insulin resistance or IR). This means the mother uses less glucose, leaving more for the baby. PGH does this by lowering the hormone adiponectin (which normally helps insulin work better) and by blocking the insulin signaling pathway, making it harder for cells to take in glucose. Too much PGH can contribute to gestational diabetes mellitus (GDM).

- **Human Placental Lactogen (hPL)**

  hPL is another hormone from the placenta, rising in the second trimester. It helps the mother's pancreas make more insulin by encouraging the growth and survival of pancreatic beta cells. Some genetic differences in the receptor for hPL (PRLR) can increase the risk of GDM. If these pathways are disrupted, the mother's pancreas may not produce enough insulin, leading to GDM.

- **Leptin**

  Leptin is mainly made by fat tissue but is also produced by the placenta during pregnancy, leading to higher levels. It helps with pregnancy processes like implantation and fetal growth. In GDM, leptin levels are even higher, especially in women who are obese or gain a lot of weight during pregnancy. High leptin can worsen insulin resistance by increasing inflammatory signals and fat breakdown, making it harder for the mother's body to use glucose.

- **Resistin**

  Resistin is a hormone from fat cells and the placenta, peaking in late pregnancy. It is linked to insulin resistance and is often higher in women with GDM, especially if they are obese. Resistin can reduce insulin release from the pancreas and make muscle, fat, and liver cells less sensitive to insulin, leading to higher blood sugar. However, its exact role in GDM is still unclear.

Hormone	Source	Role in Pregnancy	Alteration in GDM
hPL	Syncytiotrophoblast	Promotes beta-cell proliferation, enhances insulin secretion	May be downregulated in obesity; PRLR gene polymorphisms linked to GDM
PGH	Syncytiotrophoblast	Induces insulin resistance, regulates IGF-1	Overproduction may worsen insulin resistance
Leptin	SCTB, adipose tissue	Regulates placental growth, nutrient transport, maternal appetite	Hyperleptinemia increases IR, inflammation
Resistin	Placenta, adipose	Modulates insulin resistance, lipid metabolism	Elevated in GDM, may impair insulin action

Key Point: In GDM, the balance of these hormones is disrupted, contributing to the metabolic disturbances characteristic of the disease.

Inflammatory and Immune Pathways in GDM

- **Low-Grade Inflammation:** GDM is associated with a chronic, low-grade inflammatory state in the placenta. Pro-inflammatory cytokines such as TNF-α, IL-6, and chemokines like CXCL10 are upregulated.
- **Signaling Pathways:** These cytokines activate inflammatory pathways such as NF-κB, JAK2/STAT5, MAPK, and PI3K/AKT, which further impair insulin signaling and beta-cell function.
- **Macrophage Polarization:** GDM placentas show an increased ratio of pro-inflammatory M1 macrophages and a decreased ratio of anti-inflammatory M2 macrophages, leading to a pro-inflammatory environment.
- **Novel Immune Genes:** Recent transcriptomic studies have identified immune-related genes (FABP4, DKK1, CXCL10, IL1RL1) as potential biomarkers for GDM.

Placental Anatomical and Functional Alterations

Macroscopic Changes

- Size & Weight: Placentas from GDM pregnancies are often larger, thicker, and heavier (up to 22% heavier than normal).
- Vascularity: Increased diameter and central thickness, reflecting trophoblastic hyperplasia and altered vascular development.

Microscopic Changes

- Lesions: Fibrinoid necrosis, vascular lesions, increased villous blood vessels, edema, calcifications, and Hofbauer cell hyperplasia are common.
- Cell Cycle: Reduced apoptosis and altered expression of cell cycle regulators (e.g., p27, p57) lead to excessive trophoblast proliferation.

Immune Genes and Hofbauer Cells

- Immune Genes: FABP4, DKK1, CXCL10, and IL1RL1 are highly predictive for GDM when measured in placental tissue.
- Hofbauer Cells: These fetal macrophages show increased soluble epoxide hydrolase (sEH) expression in GDM, contributing to abnormal placental vascularization and delayed villous maturation.

Macrophage Polarization and Cytokine Pathways

- M1/M2 Shift: The shift towards pro-inflammatory M1 macrophages in GDM placentas is associated with increased inflammation and impaired placental function.
- IL-1β and TLR Pathways: Elevated IL-1β and Toll-like receptor signaling create an autocrine inflammatory loop in GDM placentas. Inhibiting these pathways reduces inflammation in experimental models.

Placental Structural Abnormalities: New Observations

- Villous Immaturity & Hypovascularization: GDM placentas are more likely to display immature villi and vascular lesions, which can impair nutrient and oxygen delivery to the fetus.
- Delayed Villous Maturation: More pronounced in GDM than in type 1 diabetes, suggesting unique mechanisms.

Marker	Description	Clinical Relevance
IGFBP1 and IGF-1 as early biomarkers	Low levels predict GDM risk, independent of obesity	Early screening, prevention
Immune gene signatures (FABP4, etc.)	Key genes identified as GDM biomarkers in placenta	Diagnostic model development
Macrophage polarization shift	Increased M1/M2 ratio in GDM placentas	Target for anti-inflammatory Rx
Hofbauer cell sEH expression	Increased in GDM, linked to villous hypovascularization	Insight into placental pathology
Leptin-regulated nutrient transport	Direct upregulation of placental transporter genes in GDM	Fetal macrosomia risk
Delayed villous maturation	More pronounced in GDM than T1DM	Placental function assessment
Epigenetic regulation	DNA methylation and non-coding RNAs modulate placental gene expression in GDM	Personalized medicine potential

Impact on Fetal Development

Fetal Macrosomia

- **Definition:** Birth weight ≥4000 g, often with increased abdominal and shoulder circumference.
- **Mechanism:** Maternal hyperglycemia leads to increased fetal insulin secretion (fetal hyperinsulinemia), promoting excess fat and protein deposition.
- **Placental Genes:** Overexpression of PPARα/γ and IGF-1, and activation of the mTOR pathway, drive overgrowth.
- **Anthropometric Indices:** Maternal Body Roundness Index and Body Shape Index can help predict macrosomia risk.

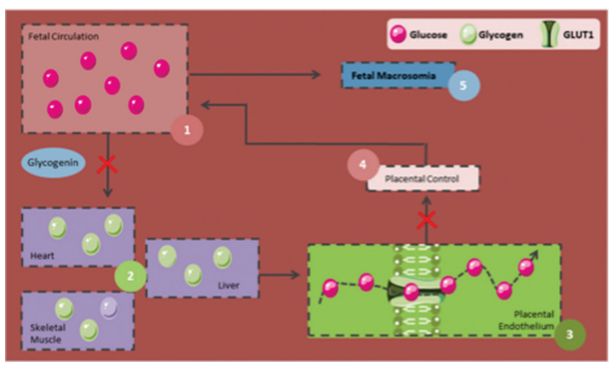


Figure 2: Physiopathology of foetal macrosomia in GDM
Adapted from https://touchendocrinology.com/diabetes/journal-articles/the-placental-role-in-gestational-diabetes-mellitus-a-molecular-perspective/

Decreased Fetal Muscle Mass

- Inflammation: Chronic placental inflammation inhibits myogenesis and promotes adipogenesis in fetal muscle.
- PGH Overexpression: Impairs insulin signaling in fetal skeletal muscle, reducing GLUT4 expression and glucose uptake.

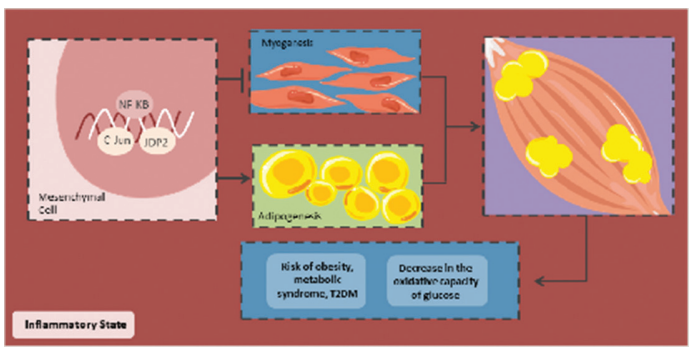
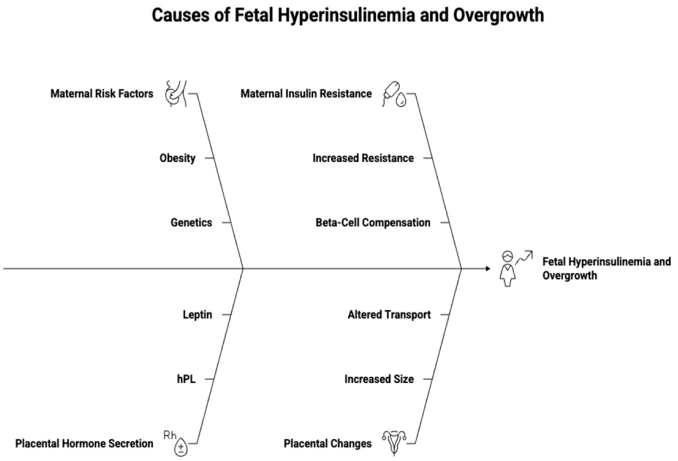


Figure 3: Mechanism of Decrease in the oxidative capacity of skeletal muscle in GDM
Adapted from https://touchendocrinology.com/diabetes/journal-articles/the-placental-role-in-gestational-diabetes-mellitus-a-molecular-perspective/

- Long-Term Risk: Offspring are at increased risk of insulin resistance, obesity, and type 2 diabetes in later life.

Diagnostic Criteria for GDM

- Diagnosis is typically made after 24 weeks gestation using oral glucose tolerance tests (OGTT).
- Various criteria exist internationally, including those by the American Diabetes Association (ADA), World Health Organization (WHO), and International Association of Diabetes and Pregnancy Study Groups (IADPSG).
- Screening involves fasting glucose, 1-hour, and 2-hour glucose measurements after glucose load.



Conclusion

Understanding the placental role in GDM is essential for medical students and clinicians. The placenta is not just a passive conduit but an active participant in the metabolic interplay between mother and fetus. Its hormonal, inflammatory, and structural adaptations—or maladaptations—are key to both the development of GDM and its consequences. As research advances, new insights into placental biology are transforming our approach to screening, prevention, and treatment, with the ultimate goal of safeguarding the health of both mothers and their children.

Suggested reading

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Silent Witness: Histopathology of Placenta in Stillbirth

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Background

Nearly 5,000 women around the world experience a stillbirth every day- tragically, most of these losses could be prevented with timely, high-quality care throughout pregnancy and childbirth. In 2023, almost 2 million babies were stillborn during the last trimester of pregnancy (at or after 28 weeks of gestation). To understand the exact cause of fetal death and to prevent similar complications in subsequent pregnancies, it is crucial to perform the complete workup, including the placental histopathological examination. Abnormalities of the placenta, umbilical cord, or membranes are potentially preventable causes of stillbirth. Placental disorders are the cause of fetal death, ranging from 11% to 65% of stillbirths in various studies, depending on the different classification systems. This chapter aims to understand the histopathological findings, their correlation with the clinical condition, and implications for subsequent pregnancies to optimize maternal and perinatal outcomes.

Placenta Normal Anatomy & Physiology

During embryogenesis, on day 4 after fertilization, the blastocyst, which is a 16- to 32-cell stage with a blastocoele, enters the uterus, where it divides into trophoblast and embryoblast. On day 6 or 7, blastocysts implant into the uterine decidua. The trophoblast penetrates deeper into the endometrium until the blastocyst completely lies within the decidua, a process known as interstitial implantation, which is characteristic of humans. The section of the decidua that extends into the blastocyst is called the decidua basalis or decidual plate and is fused with the chorion. The functional unit of the placenta consists of small, finger-like processes called chorionic villi, which are surrounded by maternal blood. These villi contain small capillaries through which fetal blood circulates. They are formed from the trophoblast and, along with the surrounding extraembryonic mesoderm (chorion), also grow; therefore, these structures are called chorionic villi. The villi that grow into the decidua basalis undergo further development and form a disc-shaped placenta. The trophoblast and syncytiotrophoblast further develop and form primary, secondary, and tertiary villi.

The Amsterdam classification system defines the following major patterns of placental injury:

I. Maternal vascular malperfusion
II. Fetal vascular malperfusion
III. Delayed villous maturation
IV. Acute chorioamnionitis
V. Villitis of unknown etiology

I. Maternal Vascular Malperfusion:

MVM, or maternal vascular malperfusion, is an abnormality of the maternal blood vessels supplying the placenta and is linked to fetal growth restriction, stillbirth, and other adverse outcomes. Maternal spiral arteries undergo characteristic changes, such as the loss of smooth muscles and elastic lamina, up to the inner third of the myometrium. These arteries adapt to deliver large amounts of blood to the placental intervillous space at an appropriate rate and pressure. However, due to abnormal implantation with insufficient spiral artery remodeling, blood flow becomes erratic and heterogeneous, resulting in areas of underperfusion and zones of high-velocity flow.

MVM can be:

1. Global MVM- Starts early in the pregnancy and has a high recurrence rate in subsequent pregnancies. The severity of MVM determines the disease spectrum of fetal growth restriction of pre-eclampsia.
2. Segmental MVM- It is usually seen in acute or intermittent events and associated with cases of thrombophilia and placental abruption.

As per the Amsterdam consensus, MVM can be diagnosed based on both gross and or microscopical findings, which are as:

1. Gross findings

a. Placental Hypoplasia: Placental hypoplasia is diagnosed when the weight of the placenta is less than the 10th centile for that gestation or when there is a thin cord. (10th centile or 8-mm diameter at term).

b. Infarction: Any infarction of more than 5% of the non-peripheral area is significant.

c. Retroplacental hemorrhage

2. Microscopic findings (Figure 1): abnormalities of villous development

a. Distal villous hypoplasia: it occurs when there is paucity of villi surrounding the stem villi,

or the villi are thin and relatively elongated in appearance, and syncytial knots are increased in numbers. It is more commonly seen in early-onset FGR.

b. Accelerated villous maturation: It is characterised by the presence of small or short hyper-mature villi for the gestational period, with an increase in syncytial knots. It is also seen in placental insufficiency, which includes FGR, preeclampsia, and preterm labour.

c. Decidual arteriopathy: It includes acute atherosis, fibrinoid necrosis with or without foam cells, mural hypertrophy, chronic perivasculitis, and absence of spiral artery remodelling, arterial thrombosis, or persistence of intramural endovascular trophoblast in the 3rd trimester.

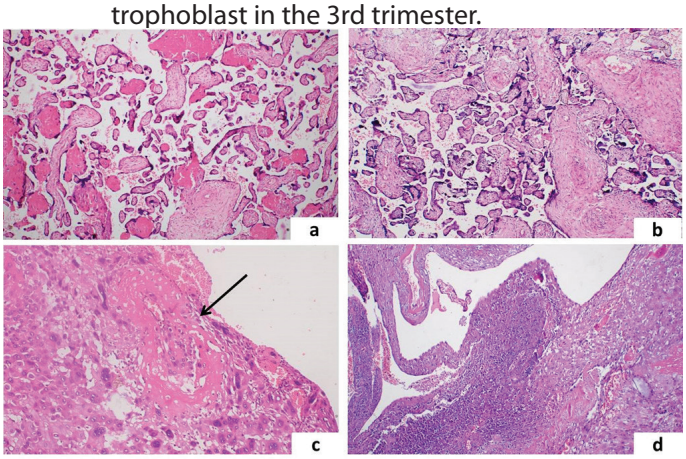

Figure 1: A panel of microphotographs showing Maternal Vascular Malperfusion (MVM); a: Distal villous hypoplasia with paucity of villi which are thin and elongated (H&E x100X); b: Accelerated villous maturation small villi having increase in syncytial knots and micro-calcification (H&E x100X); c: Fibrinoid necrosis in the maternal artery (H&E 200X); d: Dense necrotizing inflammation in the decidua (H&E x100X)

II. Fetal Vascular Malformation (FVM)

FVM is caused by obstruction in fetal blood flow that can result from various conditions, such as umbilical cord lesions, hypercoagulability, or hypoxia due to fetal cardiac dysfunction. The pathophysiology responsible for fetal vascular malformation includes thrombosis, segmental avascular villi, villous stromal vascular karyorrhexis, intramural fibrin deposition, stem vessel obliteration, and vascular ectasia etc. The findings of FVM were seen in both live births and stillbirths; therefore, it is difficult to interpret whether FVM is caused by a blood-clotting disorder or blockage, or if it happened after fetal demise.

Fetal vascular malformation can be:

a. Low grade/segmental- It is due to the thrombotic occlusion of chorionic or stem villous vessels, or stem vessel obliteration, resulting in complete obstruction to the villi downstream.

b. High grade- It is due to the partial obstruction of large vessels with venous ectasia, intramural fibrin deposition, and/ or small foci of avascular or karyorrhectic villi. The obstruction is partial or intermittent, but the lesions can be distributed over a large part of the placenta.

Findings of FVM

a. Thrombosis: It is important to see that thrombosis is arterial or venous, and the location, i.e, umbilical, chorionic plate, or stem vessel vascular level, or any combination.

b. Avascular Villi: It is categorized as small foci, i.e., 3 or more foci of 2 to 4 terminal villi showing total loss of villous capillaries and bland hyaline fibrosis of the villous stroma (Figure 2). Intermediate foci are 5 to 10 villi, and large foci are more than 10 villi.

c. Intramural Fibrin Deposition

d. Villous Stromal-Vascular Karyorrhexis

e. Stem Vessel Obliteration

f. Vascular Ectasia

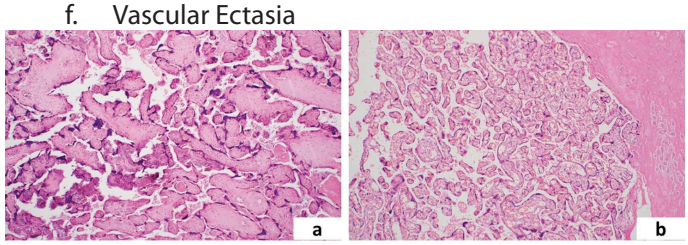

Figure 2: a: Avascular villi with terminal villi showing loss of villous capillaries and bland hyaline fibrosis of the villous stroma (H&E x100X); b: Congested terminal villi (H&E x40X)

III. Delayed Villous Maturation

It is characterised by monotonous villous population with reduced vasculo-syncytial membranes for that period of gestation age. It is defined as at least 10 such villi, in at least 30% of 1 full-thickness parenchymal slide. It is a villous maturation defect or villous dysmaturity and is usually seen after 36 weeks of pregnancy.

IV. Ascending uterine infection – Acute chorioamnionitis

The ascending intrauterine infection is clinically important; however, histologic chorioamnionitis may not be equivalent to clinical chorioamnionitis. Histologic chorioamnionitis refers to the acute inflammation of the fetal membranes and chorion of the placenta, which includes both clinically unapparent (subclinical) chorioamnionitis as well as clinical chorioamnionitis. Therefore, it is suggested to grade the maternal and fetal inflammatory response (Table 1). Poor fetal outcomes are more often associated with a severe fetal inflammatory response. As per the Amsterdam Placental Workshop Group Consensus, histologic acute

inflammation in the placenta, extraplacental membranes, and umbilical cord should be reported clearly as acute chorioamnionitis or acute chorionitis with/without fetal inflammatory response in chorionic vessels, umbilical vein, and/or umbilical artery (or arteries) as each has different clinical implications. (Figures 3 and 4).

Table 1: Staging and Grading of the Maternal and Fetal Inflammatory Responses in Ascending Intrauterine Infection (source: Amsterdam Placental Workshop Group Consensus Statement)

Staging and Grading of the Maternal and Fetal Inflammatory Responses in Ascending Intrauterine Infection

Maternal Inflammatory Response	
Stage 1-acute subchorionitis or chorionitis	Grade 1-not severe as defined
Stage 2 acute chorioamnionitis: polymorphonuclear leukocytes extend into fibrous chorion and/or amnion	Grade 2-severe: confluent polymorphonuclear leukocytes or with subchorionic microabscesses
Stage 3-necrotizing chorioamnionitis: karyorrhexis of polymorphonuclear leukocytes, amniocyte necrosis, and/or amnion basement membrane hypereosinophilia	
Fetal Inflammatory Response	
Stage 1-chorionic vasculitis or umbilical phlebitis	Grade 1-not severe as defined
Stage 2-involvement of the umbilical vein and one or more umbilical arteries	Grade 2-severe: near-confluent intramural polymorphonuclear leukocytes with attenuation of vascular smooth muscle
Stage 3-necrotizing funisitis	

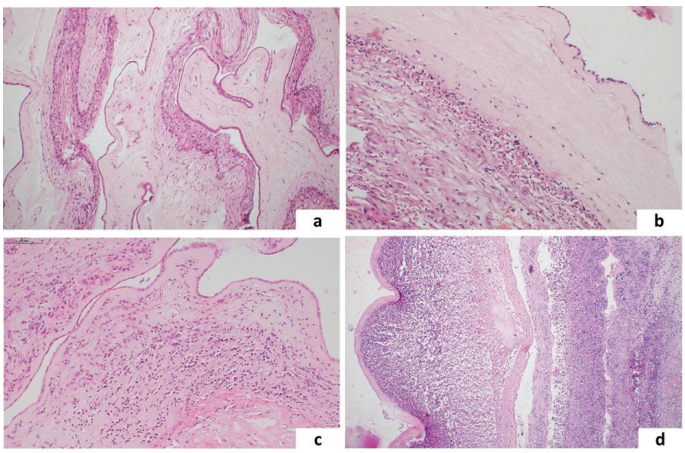

Figure 3: A panel of microphotographs showing fetal membranes; a: Section showing normal fetal membranes (H&E x200X); b: Acute subchorionitis [Stage 1, Grade 1] (H&E x200X); c: Acute chorioamnionitis [Stage 2, grade 2] (H&E x200X); d: Necrotizing chorioamnionitis [Stage 3, Grade 2] (H&E x200X)

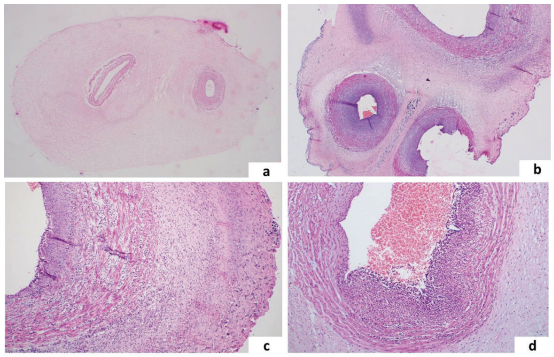

Figure 4: A panel of microphotographs showing Umbilical cords; a: Section showing single umbilical artery (H&E x40X); b: Acute funisitis showing involvement of the umbilical vein and both the umbilical arteries [Stage 2, Grade 2] (H&E x40X); c: Acute funisitis showing transmural polymorphonuclear infiltrate with nuclear debris [Stage 2, grade 2] (H&E x200X); d: Necrotizing funisitis [Stage 3, Grade 2] (H&E x200X)

Villitis of Unknown Etiology (VUE)

Villitis of unknown etiology and chronic histiocytic intervillositis are the two inflammatory conditions in the absence of infection. It is the diagnosis of exclusion and is associated with poor outcome i.e. fetal growth restriction and stillbirths. VUE is attributed to maternal immune rejection of a semi-allogeneic placenta, characterised by the presence of elevated numbers of fetal macrophages (Hofbauer cells) and an infiltrate of maternal T lymphocytes in the villous stroma.

VUE is graded as:

Low grade: Presence of inflammation affecting fewer than 10 contiguous villi in any one focus.

High grade: Presence of multiple foci, on more than one section, at least one of which shows inflammation affecting more than 10 contiguous villi. High-grade VUE has significant associations with FGR, neurodevelopmental impairment, and recurrence in subsequent pregnancies as compared to low-grade VUE (Figure 5).

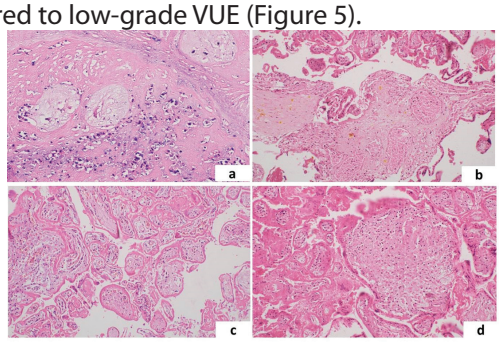

Figure 5: A panel of microphotographs showing Umbilical cords; a: Section showing single umbilical artery (H&E x40X); b: Acute funisitis showing involvement of the umbilical vein and both the umbilical arteries [Stage 2, Grade 2] (H&E x40X); c: Acute funisitis showing transmural polymorphonuclear infiltrate with nuclear debris [Stage 2, grade 2] (H&E x200X); d: Necrotizing funisitis [Stage 3, Grade 2] (H&E x200X)

Placental infarction and retroplacental clot:

Placental infarction occurs due to impaired maternal blood flow resulting from preeclampsia or maternal hypertension. These can be recognised on gross specimens as firm, pale areas. Retroplacental clot is blood clot between the placenta and myometrium mainly due to placental abruption. On gross examination, it is dark red to brownish area which may be seen compressing the placental tissue (Figure 6).

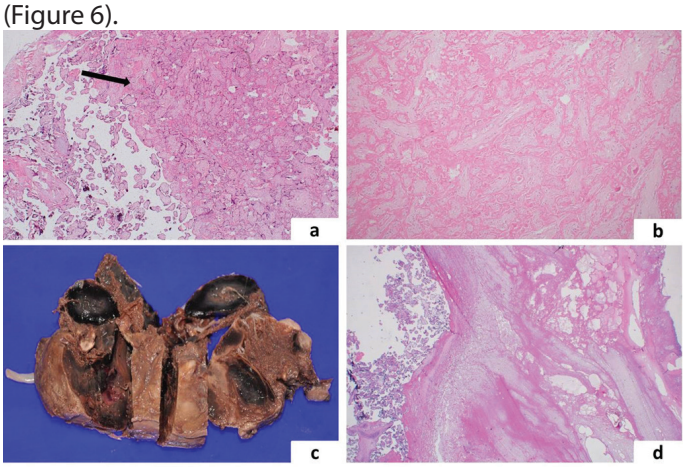

Figure 6: A panel of microphotographs; a: A normal area with area of infarction {arrow} (H&E x40X); b: Whole area showing infarction (H&E x100X); c: Gross specimen showing retro-placental blood clot; d: Retro-placental blood clot on microscopy (H&E x40X)

Conclusion

Placental histopathological lesions associated with stillbirth are highly variable and often poorly defined due to inconsistencies in the classification and reporting formats. Collaboration among obstetricians, neonatologists, and pathologists is essential to improve understanding of the underlying pathophysiology, thereby aiding in the prevention of adverse neonatal outcomes and recurrence in future pregnancies.

Key points

- Global maternal vascular malformation (MVM) and high-grade villitis of unknown etiology (VUE) carry the highest risk of recurrence in subsequent pregnancies.
- Fetal vascular malformations (FVM) are challenging to interpret, as it is often unclear whether they result from a blood-clotting disorder, vascular obstruction, or if they developed after fetal demise.
- Histologic chorioamnionitis does not always correlate with clinical chorioamnionitis; therefore, grading maternal and fetal inflammatory responses is important for interpretation.
- Severe fetal inflammation is more strongly associated with adverse fetal outcomes.
- Villitis of unknown etiology (VUE) and chronic histiocytic intervillositis (CHI) are two inflammatory placental lesions believed to result from maternal immune rejection of the fetus.

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Monochorionic placenta: Types of vascular anastomoses and their effects

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1. Introduction

Monochorionic placenta: Types of vascular anastomoses and their effects

Monochorionic placentas, shared by twins, feature three types of vascular anastomoses: arterioarterial (AA), venovenous (VV), and arteriovenous (AV). AA and VV anastomoses are superficial and bidirectional, located on the chorionic plate surface. AA anastomoses connect arteries between twins and help balance blood flow, compensating for imbalances from AV connections. Most monochorionic placentas have only one AA anastomosis. VV anastomoses, seen in about 25% of cases, link veins and may be associated with sudden changes in venous return and reduced survival, though evidence is inconsistent.[1,2] In placentas without VV connections, each twin retains distinct venous drainage territories.

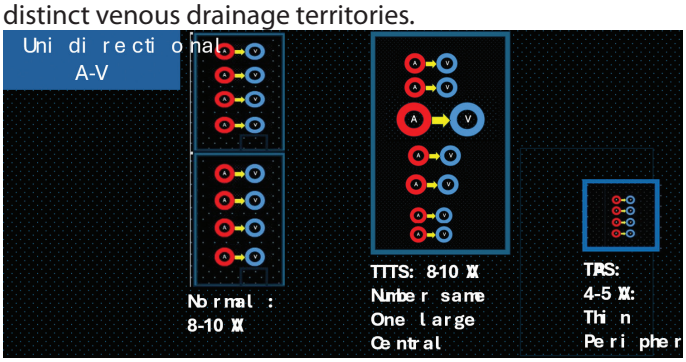

Figure 1: Number, Position and Caliber of AV anastomoses

AV anastomoses are deep, occurring within shared placental lobules, and are unidirectional, allowing blood to flow from one twin's artery to the other's vein. This can result in transfusion imbalance, particularly if no compensating bidirectional connections are present. Surface clues of AV anastomoses include an unpaired artery and vein entering the placenta close together. These anastomoses divide the placenta into three functional zones: two individual and one shared. Around 90% of monochorionic placentas have multiple AV (and venoarterial) connections, with AA and/or VV also present, while about 5% have only AV anastomoses, and another 5% lack any detectable anastomoses.[1,2] The result of these anastomoses are the complications encountered by us as obstetricians including TTTS, TAPS, TRAP and discordant growth.

2. TTTS

Twin-Twin Transfusion Syndrome (TTTS) is a serious complication affecting approximately 10% of monochorionic diamniotic (MCDA) twin pregnancies, typically occurring between 16 and 26 weeks' gestation.[3] It results from unbalanced blood flow through vascular anastomoses in a shared placenta. These connections include arterioarterial (AA), venovenous (VV), and arteriovenous (AV) anastomoses. AV anastomoses are deep, unidirectional, and the main drivers of TTTS, enabling blood to flow from the donor twin to the recipient twin. AA and VV anastomoses are superficial and bidirectional; the presence of an AA anastomosis is considered protective, helping compensate for the AV flow imbalance.[4]

TTTS is diagnosed antenatally by ultrasound, characterized by a polyhydramnios-oligohydramnios sequence. The recipient twin shows a deepest vertical pocket (DVP) of ≥8 cm (≥10 cm after 20 weeks in Europe) with a distended bladder, while the donor twin has a DVP ≤2 cm with a small or absent bladder. Unlike the name suggests, there is usually no significant difference in hemoglobin between the twins, meaning the problem is more related to fluid imbalance than actual red cell transfer.[5]

The underlying mechanism involves unidirectional AV shunting, where blood chronically flows from the donor to the recipient. The donor becomes hypovolemic, leading to reduced renal perfusion and oliguria, while the recipient becomes hypervolemic, developing cardiac overload and increased natriuretic peptides, resulting in polyuria and polyhydramnios. The imbalance can result in growth restriction, cardiac dysfunction, or even fetal demise.[6] Renal tubular dysgenesis, and oliguria in the donor and visceromegaly and polyuria in the recipient. A better understanding of its pathophysiology could contribute to improving the management of TTS, which still carries a high perinatal mortality in both twins. As well as several other candidates, the renin-angiotensin system might be involved in TTS. To evaluate its role in the pathogenesis of the syndrome, we studied the kidneys of 21 twin pairs who died from TTS at 19 to 30 weeks, compared with 39 individuals in a control group, using light microscopy, immunohistochemistry, and in situ hybridization. The overexpression of the renin protein and transcript with frequent evidence of renin synthesis by mesangial cells was observed in the donor kidneys, presumably as a consequence of chronic renal hypoperfusion. This upregulation of renin synthesis might be beneficial to restore euvolemia. In severe cases of TTS, however, angiotensin-II-induced vasoconstriction acts as an additional deleterious factor by further reducing the renal blood flow in donors. In recipients, renin expression was virtually absent, possibly because it was down-regulated by hypervolemia. However,

in addition to congestion and hemorrhagic infarction, there were severe glomerular and arterial lesions resembling those observed in polycythemia- or hypertension-induced microangiopathy. We speculate that fetal hypertension in the recipient might be partly mediated by the transfer of circulating renin produced by the donor, through the placental vascular shunts.","container-title":"The American Journal of Pathology","DOI":"10.1016/S0002-9440[10,7]occurs due to antidiuretic and vasoconstrictive activity of vasopressin (AVP)

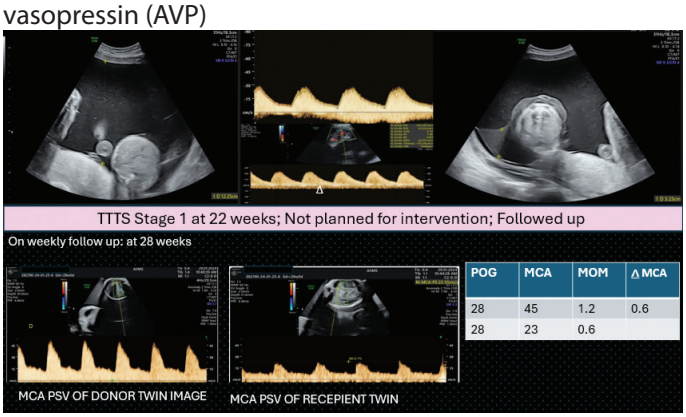

Figure 2: TTTS with TAPS

Fetoscopic laser photocoagulation is the gold standard treatment, aiming to interrupt all placental vascular anastomoses, thereby functionally separating the circulations.(8) The Solomon technique, which involves lasering a line across the entire vascular equator, has been shown to reduce recurrence and complications like twin anemia-polycythemia sequence (TAPS).[9] The optimal management of stage I is still debated. The \"Solomon\" technique showed a significant reduction in recurrent TTTS and post laser twin anemia-polycythemia sequence (TAPS After laser therapy, there is a 50–60% chance of both twins surviving, and an 80% chance of at least one surviving. (10) Neurological complications occur in about 10% of survivors, with cerebral palsy in 5%.

TTTS has a five-stage classification (Quintero Staging System), ranging from fluid discordance (Stage I) to fetal demise (Stage V). Stage I is often managed expectantly, while Stages II–IV require laser intervention. Without treatment, TTTS frequently leads to miscarriage, preterm delivery, or neonatal morbidity. Early detection is vital. Therefore, fortnightly ultrasound screening is recommended for all MCDA pregnancies starting at 16 weeks, focusing on amniotic fluid levels, fetal bladders, and Doppler studies.

One major limitation of laser therapy is the risk of missed small AV anastomoses, which may not be visible due to intrauterine pressure. This can lead to recurrent TTTS or TAPS, which is typically detected only through middle cerebral artery Doppler showing anemia/polycythemia patterns.[11] Laser improves outcomes but does not eliminate all risks with only nearly half of all couples seeking this treatment getting the joy of carrying two healthy babies home after delivery.

3. TAPS

Twin Anemia-Polycythemia Sequence (TAPS) is a serious complication that occurs in monochorionic twin pregnancies due to chronic, unbalanced blood transfusion between twins through tiny (<1 mm), unidirectional placental anastomoses.[12] Allowing the transfer of blood from one fetus to the other and vice versa. These anastomoses are the essential anatomical substrate for the development of several complications, including twin-twin transfusion syndrome (TTTS Unlike Twin-Twin Transfusion Syndrome (TTTS), which is defined by amniotic fluid discordance, TAPS is characterized by a severe hemoglobin difference without significant differences in amniotic fluid volumes.[13]

TAPS can develop spontaneously in about 5% of otherwise uncomplicated monochorionic pregnancies, usually after 26 weeks, or as an iatrogenic complication in up to 13% of pregnancies treated with fetoscopic laser coagulation for TTTS.[3,14] Iatrogenic TAPS often develops within 1–5 weeks after laser treatment, typically when small anastomoses are missed during the procedure.[14]

Antenatal diagnosis of TAPS relies on discordant middle cerebral artery peak systolic velocities (MCA-PSV). The anemic twin shows an MCA-PSV >1.5 MoM, while the polycythemic twin shows MCA-PSV <1.0 MoM. There may be mild amniotic fluid differences, but not to the extent required to diagnose TTTS. Postnatal diagnostic criteria include a hemoglobin difference ≥8 g/dL, a reticulocyte count ratio >1.7, and confirmation of small vascular connections on placental injection studies.(12)allowing the transfer of blood from one fetus to the other and vice versa. These anastomoses are the essential anatomical substrate for the development of several complications, including twin-twin transfusion syndrome (TTTS

Placental studies show that TAPS is associated with fewer, smaller anastomoses compared to TTTS, supporting the theory of chronic red blood cell transfer without plasma exchange. Arterioarterial anastomoses, which are protective, are rarely present or are too small in TAPS cases to compensate for the imbalance.[15] Not complicated by twin-to-twin transfusion syndrome and resulting in double survival. The study was conducted at two European Fetal Therapy Centers between 2002 and 2008. Placental angioarchitecture was evaluated using colored dye injection. Diagnosis of twin anemia-polycythemia sequence was based on the presence of large intertwin hemoglobin difference without the degree of amniotic fluid discordance that is required for the diagnosis of twin transfusion syndrome.

RESULTS: Three-hundred thirteen monochorionic twin

pregnancies were eligible for the study but placental data could not be completed for 62 placentas (20%

Apart from the obvious USG findings, following depicted below could also act as indirect pointers towards the same.

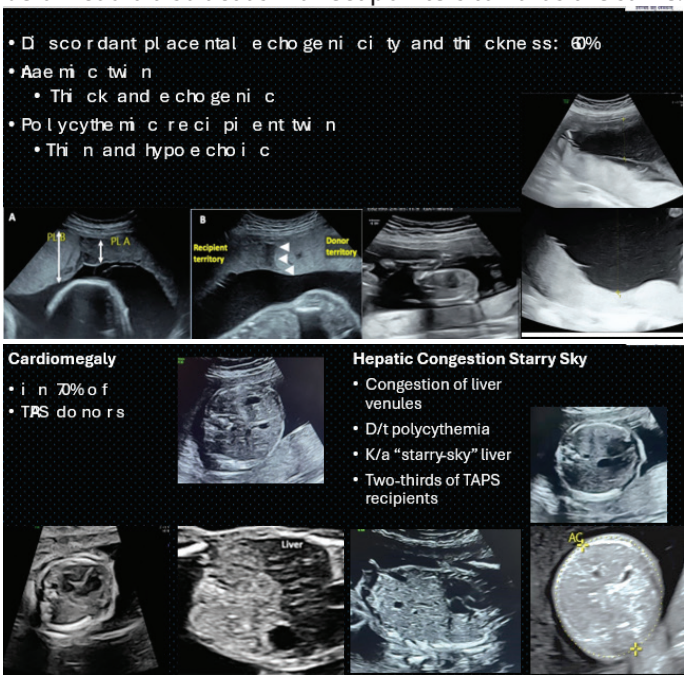

Figure 3: USG finding is TAPS

Complications of TAPS include neurological injury, such as cerebral infarction or cerebellar hemorrhage, especially in cases of severe anemia or delayed treatment. Neonates may require transfusions or partial exchange transfusions, and early delivery is often needed. Because of the high risk of missed diagnosis, fortnightly MCA Doppler surveillance is recommended from 20 weeks onward, especially in twins with growth discordance.[16]

Management depends on gestational age and disease severity. Prior to 30 weeks, options include intrauterine transfusion (IUT) for the anemic twin and close monitoring.[13] If anemia recurs or complications arise, repeat laser therapy, selective reduction, or early delivery may be considered. After 30 weeks, elective delivery following corticosteroid administration is often the preferred approach.

4. TRAP

Twin Reversed Arterial Perfusion (TRAP) sequence is a rare and severe complication that occurs in approximately 1% of monochorionic twin pregnancies. It involves abnormal blood flow between twins, where the pump twin supplies deoxygenated blood in reverse direction via an arterioarterial anastomosis to the perfused (acardiac) twin, whose blood then returns to the pump twin through a venovenous anastomosis[17] especially heart defect. In spite of the aggressive treatment (serial amnioreduction, digoxin treatment The perfused twin lacks a functional heart and is essentially a parasitic twin, relying entirely on the pump twin's circulation.

Because the blood the acardiac twin receives is poorly oxygenated, development is severely impaired. The upper body structures, including the head, heart, and upper limbs, are typically underdeveloped or absent. Development of TRAP sequence requires two key elements: [1] an arterioarterial anastomosis, and [2] discordant development or early intrauterine demise of one twin. In contrast to dichorionic twins, where early demise leads to a vanishing twin, monochorionic placental connections can sustain some continued growth in the demised fetus.(17) especially heart defect. In spite of the aggressive treatment (serial amnioreduction, digoxin treatment,[13]

TRAP sequence can be diagnosed early, often in the first trimester, using color Doppler imaging to detect reversed blood flow in the umbilical artery of the acardiac twin. However, intervention is typically delayed until after 16 weeks, once the amnion and chorion have fused, to allow for safer access and treatment.[18]

The pump twin faces a high risk of perinatal death, primarily due to high-output cardiac failure from the increased circulatory demand, and polyhydramnios, which can lead to preterm labor.[19] The prognosis for the pump twin can be improved with prophylactic intrauterine intervention, such as cord occlusion, coagulation of placental anastomoses, or intrafetal techniques like laser or radiofrequency ablation (RFA) to stop blood flow to the acardiac twin.[20] percutaneous fetoscopic laser coagulation of placental anastomoses (n = 18)

Although early diagnosis is possible, timing of intervention is critical. One study of 24 TRAP cases found that 33% of pump twins died before scheduled surgery at 16–18 weeks. In contrast, 90% of those who underwent intervention survived. While some first-trimester interventions have shown promise, more evidence is needed to confirm their safety and efficacy.[21]and two pregnancies were scheduled for later treatment. One pregnancy was treated with fetoscopic laser ablation and excluded from analysis. The delivery reports of all pregnancies were collected, the neonatal health status recorded and the median time of delivery and the treatment to delivery interval calculated.\ nRESULTS: Six of seven pump fetuses in TRAP pregnancies treated with interstitial laser therapy at a median of 16+2 (range 13+1 to 20+3,(22)twin-to-twin transfusion.[6]

5. Demise of 1 twin:

Unequal placental sharing, placental insufficiency, or hemodynamic imbalances such as TTTS or TAPS can cause the intrauterine death of one twin in monochorionic pregnancies. Due to their shared circulation, the death of one twin may lead to the death of the surviving cotwin or cause antenatal brain damage from acute blood loss into the deceased twin's circulation. A systematic review found that single fetal demise results in double demise in about 15% of cases, neurodevelopmental impairment in 25%,

and a 68% risk of preterm birth.[23]

Brain damage may be detectable weeks after the insult via ultrasound, while MRI can identify lesions earlier with better detail. Initially thought to result from thromboembolism, evidence now supports acute exsanguination as the main cause, with surviving twins showing decreased hematocrit but normal coagulation.[24]

The risk to the surviving twin depends on the type and size of vascular anastomoses and the placental mass of the demised twin. Arterioarterial anastomoses may increase death and neurological damage risk, but exsanguination can also occur without them through other vascular connections.(25) Laser coagulation treatment for TTTS leads to fewer double demises and less anemia in survivors compared to amniodrainage, supporting the role of anastomoses in adverse outcomes.[26]

Monitoring fetal anemia using MCA-PSV is effective, and if anemia is absent, prognosis is better.(27) Rescue interventions like intrauterine transfusion lack strong evidence for improving neurological outcomes, highlighting the need for careful prenatal monitoring and imaging.

6. Discordant Growth

Discordant growth, defined as a weight difference of 25% or more between twins, occurs in about 10-15% of twin pregnancies and is equally common in monochorionic (MC) and dichorionic twins.[28,3] In MC twins, who share identical genetics, growth differences mainly result from unequal placental sharing and vascular anastomoses that affect blood flow and nutrient delivery.

Vascular anastomoses further influence growth by creating imbalanced transfusion between twins, as seen in conditions like Twin-to-Twin Transfusion Syndrome (TTTS) and Twin Anemia Polycythemia Sequence (TAPS). These imbalances may restrict growth in the donor twin and enhance it in the recipient. Laser coagulation of anastomoses can reduce growth discordance by improving donor growth or limiting recipient overgrowth but carries risks, especially when placental sharing is unequal. In such cases, laser treatment may cause demise of the smaller twin who might have survived otherwise.

Discordant growth presents differently depending on its onset. Early-onset discordance (before or at 20 weeks) is associated with unequally shared placentas, large anastomoses, and abnormal umbilical artery Doppler flow in the smaller twin, with a roughly 20% risk of intrauterine death. Late-onset discordance (after 26 weeks) usually occurs with more equally shared placentas, smaller anastomoses, and normal Doppler readings, showing nearly 100% survival but raising suspicion for TAPS, which requires careful monitoring via middle cerebral artery peak systolic velocity (MCA-PSV) measurements.[16]

Umbilical artery Doppler patterns in the smaller twin classify discordance into three types: Type I (normal flow, favourable outcome), Type II (persistent absent/reversed end-diastolic flow, poor prognosis), and Type III (intermittent absent/reversed flow, unpredictable course). Type II carries the worst prognosis.[30]

Management focuses on close surveillance, with selective reduction or preterm delivery considered in severe early-onset cases. Laser coagulation is generally not recommended solely for discordant growth due to limited benefit and technical challenges, especially in the absence of polyhydramnios and with unequal placental sharing.

7. Summary

The monochorionic twin pregnancy needs a protocolised approach to management with timely USG to detect complications. The DCDA twins should be followed up 4 weekly while MCDA twins can be followed up 2 weekly 16 weeks onwards to detect complications and provide timely intervention.

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Placenta Accreta Spectrum

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Introduction

Placenta accreta was first described in 1937 by Irving et al. as failure of separation of the placenta from the uterine wall following delivery of the human fetus leading to the often used term morbid placental adherence.[1] This abnormal placentation results in catastrophic haemorrhage causing maternal morbidity as well as mortality. PAS refers to a spectrum of abnormal placental adherence that could be ranging from the subclinical (often microscopic) finding of adherent myometrial fibres within the basal plate to a dramatic presentation of placenta percreta, where invasion through the serosa into the peritoneal cavity or bladder.

Prenatal Diagnosis

RISK FACTORS

At the initial obstetric visit, a detailed history is the most important tool for an obstetrician. The identification of the risk factors for placenta accreta will raise a suspicion and hence result in adequate antenatal testing and investigations to rule out any evidence of accreta.[2]

- Previous cesarean section - The scarred tissue causes a defect at the endometrial-myometrial interface allowing for abnormally deep placental anchoring villi and trophoblastic infiltration.
- Advanced maternal Age
- Multiparity
- Assisted Reproductive Technology (ART)
- Placenta previa
- Multiple Abortions
- History of uterine surgeries like Myomectomy, curettage, hysteroscopic surgery, prior endometrial ablation, Uterine embolization
- Pelvic irradiation
- Smoking

IMAGING

- Ultrasound: USG findings such as placental lacunae, placenta invading the uterine serosa, loss of the retroplacental hypoechoic clear zone, turbulent lacunar blood flow, increased sub-placental vascularity, gaps in myometrial blood flow, and vessels bridging the placenta to the uterine margin on colour flow Doppler imaging can aid in antenatal diagnosis of placenta accreta.[3]

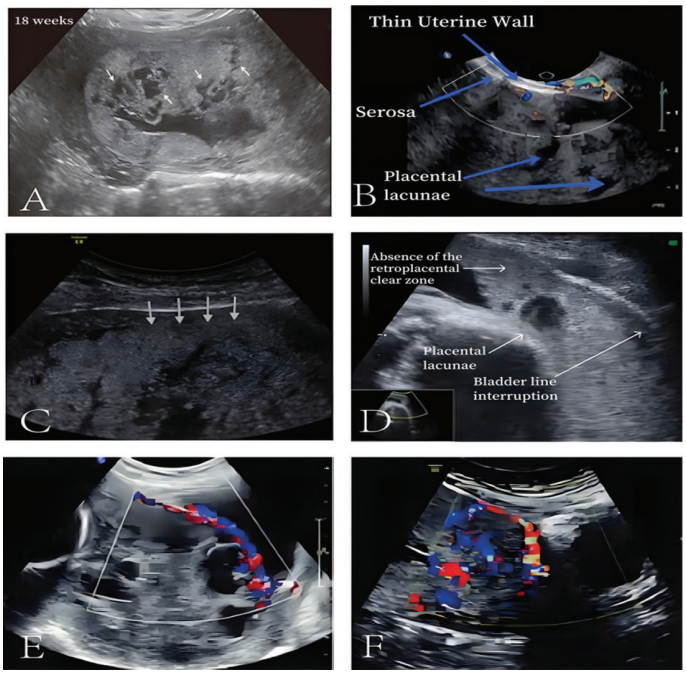

Figure: Ultrasonic images of placenta accreta spectrum. (A) White arrows represent different sizes of placental lacunae at 18 weeks of pregnancy. (B) Blue arrows represent large placental lacunae images with placenta invading the uterine serosa. (C) White arrows represent partial loss of the retroplacental hypoechoic clear zone. (D) White arrows represent complete loss of the retroplacental clear zone accompanied by large placental lacunae and uterine-bladder boundary line thinning and irregularity. (E and F) Increased placental blood flow on 2D ultrasound. Red color represents increased placental blood flow moving to the transducer; Blue color represents placental blood flow leaving away from the transducer.[3]

- Magnetic Resonance Imaging: This has become an effective auxiliary examination method especially in the case of the posterior placenta or placenta percreta. MRI findings suggestive of PAS include abnormal placental bed vascularization, bladder wall interruption, dark intra-placental bands, heterogenous placenta, myometrial thinning, loss of retroplacental dark zone and loss of placental bulge. As antepartum diagnosis is crucial to minimize maternal morbidity and mortality, the use of MRI is mandatory in cases with nonconclusive ultrasonography findings especially in women with posterior placenta previa where the accuracy of ultrasound is low.[4]

The absence of ultrasound/ MRI findings does not preclude a diagnosis of placenta accreta spectrum; clinical risk factors remain important predictors of placenta accreta spectrum.

CLASSIFICATION

FIGO's Expert Consensus Panel[5] has developed a classification of placenta accreta spectrum:

- **Grade 1:** abnormally adherent placenta (placenta adherent or accreta) - attached directly to the surface of the middle layer of the uterine wall (myometrium) without invading it
- **Grade 2:** abnormally invasive placenta (increta) with invasion into the myometrium
- **Grade 3:** abnormally invasive placenta (percreta) with invasion reaching surrounding pelvic tissues, vessels and organs.
    - 3a- Limited to the uterine serosa
    - 3b- urinary bladder invasion
    - 3c- Invasion of other pelvic tissue/organs

Management

Encountering an adherent placenta at the time of delivery is fraught with dangers, more so if undiagnosed earlier. Problems include extensive haemorrhage, requirement of massive blood

transfusion, decision-making on uterus preservation, future fertility and injury to adjacent organs. As a result, PAS becomes an important contributing factor to maternal mortality.

Pre-operative

- Maximization of pre-operative haemoglobin
- Notifying the team (experienced obstetricians and maternal–fetal medicine subspecialists, pelvic surgeons, urologists, interventional radiologists, obstetric anesthesiologists, critical care experts, general surgeons, neonatologists, nursing staff and transfusion team)
- Discussions with patient and family

Timing of delivery

Decisions need to balance maternal risks and benefits with those of the fetus or neonate. It appears that performing a cesarean delivery followed immediately by cesarean hysterectomy before the onset of labor improves maternal outcomes, yet the optimal timing remains unclear. Although 34 weeks of gestation appears optimal for centres comfortably handling neonatal complications at that gestational age and the increased risk of bleeding after 36 weeks, a window of 34 0/7–35 6/7 weeks of gestation is suggested as the preferred gestational age for scheduled cesarean delivery or hysterectomy.[6]

Use of antenatal corticosteroids for lung maturation is appropriate in women with antenatally diagnosed accreta and anticipated delivery before 37 0/7 weeks of gestation.

Opening abdomen

- Choice of skin incision is left to operator judgment, although many employ vertical incisions for better access and visualization. Reasonable alternatives are wide transverse incisions such as a Maylard or Cherney incision.
- Inspection of the uterus after peritoneal entry is obtained is highly recommended to discern the level of placental invasion and specific placental location, which allows for optimizing the approach to the uterine incision for delivery and likely hysterectomy.
- Whenever possible, the incision in the uterus should avoid the placenta, which sometimes makes a non-traditional incision necessary.
- Careful dissection in the retroperitoneal space with attention to devascularization of the uterine corpus in proximity to the placenta often is required given the overwhelming vascularity and friability of involved tissues.

Intra-operative

- Close monitoring of volume status, urine output, ongoing blood loss, and overall hemodynamics is critically important. Ongoing dialogue between surgical, anesthesia, and nursing staff are recommended.
- Use of hemorrhage checklists is strongly encouraged. Use of a 1:1:1 strategy of packed red blood cells: fresh frozen plasma: platelets is recommended.6 The use of autologous cell-saver technology is an option.
- Patients should be kept warm because many clotting factors function poorly if the body temperature is less than 36°C. Acidosis should be avoided. If blood loss is excessive (1,500 mL or greater), prophylactic antibiotics should be re-dosed.
- Baseline assessment should include platelet count, prothrombin time, partial thromboplastin time, and fibrinogen levels. Rapid and accurate results can guide transfusion management. Hypofibrinogenemia is the biomarker most predictive of severe postpartum hemorrhage. Functional assays using viscoelastic coagulation testing such as thromboelastography or rotational thromboelastometry are helpful.

Conventional approach[6]

- Having the most experienced pelvic surgeons involved from the outset is recommended.
- Delivering the baby via Caesarean section, closing the incision after tying the cord, removing the uterus with the placenta still attached is recommended.
- Cystoscopy may be done if bladder involvement is suspected and ureteral stents may be placed pre-

operatively.

- Hysterectomy may be total or subtotal, depending on the situation. Lower uterine segment or cervical bleeding frequently precludes a supracervical hysterectomy.

Uterine-sparing approach

- **Expectant management:** It involves leaving the placenta in situ after delivery of the baby. It is important to counsel the patient extensively on potential risks like hemorrhage, sepsis, need for hysterectomy, and accreta recurrence (15%-30%) in future pregnancies.
- Methotrexate use is advocated by some authors who contend that it will hasten placental involution and resorption. Due to its potential for maternal hematologic and nephrologic toxicities and neonatal morbidity, methotrexate is not recommended.
- **Partial Resection and Reconstruction of Uterus:** Focal resection can be considered when the PAS area involves less than 50% of the anterior wall of the uterus. However, a clear recommendation on who should be offered these techniques is still lacking.[7]
- **Triple-P procedure:** This involves three main steps: placental upper edge localisation, pelvic devascularisation by Uterine artery embolization and placental non-separation. The placenta with adherent myometrium is excised, followed by repair of the defect.8
- A modified triple-P approach suggests ligation of the internal iliac vessels, which may be more suitable when facility for UAE is not available.[9]
- **Modified One-Step Conservative Uterine Surgery (MOSCUS):** In addition to uterine artery ligation, the surgeons have used cervical tourniquets and transverse B lynch to address haemorrhage.[10]
- **PABO:** Prophylactic balloon occlusion is the occlusion of the feeding vessels by a balloon device during surgery to reduce bleeding. This device is placed preoperatively and then inflated after the delivery of the baby. Occlusion of the infrarenal abdominal aorta[11] or internal iliac vessels[12] has also been advocated by few authors. Intra-operative POIIA combined with post-operative UAE may be an effective strategy in conservative management.[13]

Delayed interval hysterectomy

Delayed interval hysterectomy is a derivative of an expectant approach to placenta accreta spectrum, except that future fertility is not a consideration, and minimizing blood loss and tissue damage are the primary goals. Patients with placenta percreta are optimal candidates for this procedure because they have an increased risk of blood loss and tissue damage if hysterectomy is performed at the time of cesarean delivery. Although these preliminary data are encouraging, use of this method warrants caution and presently not recommended by ACOG.

Post-operative

- Vigilance for complications such as renal or liver failure, infection, unrecognized ureteral, bladder, or bowel injury, pulmonary edema and diverse intravascular coagulation is warranted.
- Attention to the small but possibility of Sheehan syndrome (also known as postpartum pituitary necrosis) is warranted given the clinical scenario and the potential for hypoperfusion.

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Calendar for AOGD Monthly Clinical Meeting 2025-2026

29th August 2025	AIIMS
26th September 2025	VMMC &Safdarjung Hospital
31st October 2025	DDU Hospital
28th November 2025	MAMC & LNJP Hospital
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	To be decided

Caesarean SCAR Pregnancy

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Introduction

Caesarean scar pregnancy (CSP) is a rare but increasingly recognized form of ectopic pregnancy in which the embryo implants within the myometrial defect of a previous caesarean section scar. First reported by Larsen and Solomon in 1978[1], its incidence is estimated at approximately 1 in 1,800 to 2,216 pregnancies, accounting for nearly 6% of all ectopic gestations[2]. The rising global caesarean delivery rate has led to a proportional increase in CSP cases, with significant implications for maternal morbidity. If unrecognized, CSP can result in life-threatening complications such as uterine rupture, massive haemorrhage, and loss of fertility.

Advancements in high-resolution transvaginal ultrasonography, particularly with Doppler imaging, now permit early and accurate diagnosis, while magnetic resonance imaging (MRI) serves as an adjunct in ambiguous cases. Despite improvements in diagnostic modalities, management remains complex and must be tailored to individual patient characteristics—including clinical presentation, fertility desires, β-hCG levels, and expertise available.

CSP is not a homogenous condition; it encompasses various clinical phenotypes depending on the location and depth of gestational sac implantation, the integrity and thickness of the residual myometrium, and the configuration of the caesarean scar niche. These factors significantly influence treatment decisions and outcomes[3].

This review aims to critically evaluate the most recent clinical guidelines and emerging evidence on the diagnosis and management of CSP—including recommendations from the Society for Maternal-Foetal Medicine (SMFM)[4] and recent systematic reviews—to support evidence-based decision-making in clinical practice

Classification and Types of Caesarean Scar Pregnancy

CSP classification is based on imaging characteristics—including gestational sac(Gsac) orientation, depth of implantation, residual myometrial thickness (RMT), and proximity to the uterine cavity, bladder, and vascular structures[5]. These features influence prognosis and guide individualized management.

Multiple classification systems have evolved to stratify risk and inform treatment, with the **Delphi consensus (Types I–III)**, **Jordans' sonographic grading**, and the **Timor-Tritsch system** being the most widely adopted. Each incorporates ultrasound and Doppler criteria to predict outcomes such as rupture, haemorrhage, and treatment failure.

This section summarizes and compares key CSP classification systems, highlighting their diagnostic and clinical relevance.

Table 1: KEY CSP classification systems

Key recommendations from prominent societies and publications are summarized below:

System	Type/Grade	Key Features	Clinical Implication
VIAL Classification, 20006	Endogenic CSP (Type I)	Gestational sac grows towards the cervico-isthmic or the uterine cavity; may continue as intrauterine pregnancy but with risk of placenta accreta spectrum (PAS).	Allows conservative/medical treatment but requires close follow-up due to PAS risk.
	Exogenic CSP (Type II)	Sac deeply invades myometrium and grows toward the bladder or abdominal cavity; high risk of early rupture and severe haemorrhage.	Often requires early surgical or interventional management.
TimorTritsch grading, 20107	Grade I:	Gestational sac embedded in the niche with ≥3 mm myometrial thickness.	-Low risk of rupture - Often hemodynamically stable - Amenable to conservative or medical therapy
	Grade II:	Myometrial thickness <3 mm with or without detectable trophoblastic blood flow.	Intermediate risk of rupture - Greater likelihood of persistent trophoblastic activity - Increased risk of incomplete response to MTX alone
	Grade III:	Complete absence of myometrium between the sac and the serosa, often associated with high risk of uterine rupture.	High risk of uterine rupture, severe haemorrhage, and bladder invasion - Typically requires urgent, definitive intervention

Lin et al., 2018 (Ultrasound Grading)[8]	Grade I	Sac embedded in ≤50% of myometrial thickness.		May respond to methotrexate or minimally invasive therapy.
	Grade II	Sac extends into >50% of myometrium.		Higher risk; surgical or combined approaches preferred.
	Grade III	Sac bulges outward, thinning myometrium.		High risk of rupture; early surgical management advised.
	Grade IV	Irregular mass with rich vascularity replacing scar area.		Often requires complex surgical resection and uterine repair.
BAN et al, 20239		Anterior Myometrium Thickness(mm)	Average Diameter of Gsac (mm)	
	Type I	Greater than 3		- Lower risk of rupture or haemorrhage - May resemble low intrauterine pregnancy or cervical ectopic - Often asymptomatic
	Type II	1-3	IIa: 30 mm or less IIb: greater than 30 mm	Moderate risk of rupture and bleeding, with the potential to progress to Type III if untreated with a greater likelihood of incomplete resolution with medical management.
	Type III	1 or less	IIIa: 50mm or less IIIb: > 50 mm or with uterine arterio venous fistula	High to catastrophic risk of uterine rupture and severe haemorrhage, often presenting with pain or spotting, and may be associated with bladder invasion or uterine arteriovenous malformations.
Jordans et al., 2024 (Type I–III)/ Delphi Consensus Classification10	Type 1	Similar to endogenic CSP with minimal invasion.		Medical treatment often effective.
	Type 2	Moderate exogenic invasion.		Mixed medical-surgical approaches.
	Type 3	Deep implantation with severe scar niche invasion.		Surgical removal or niche excision is usually required.
Three-Type with Subtypes Classification (Ultrasound-based CSP Types I–III), 2024, Fu et al.6	Type I	Closer type: sac adjacent to scar (not implanted) Ia: Gsac located in the lower uterine segment Ib: Gsac located in the cervical canal		Lower risk of complications (rupture, bleeding). Often represents early implantation near the scar niche without true invasion.
	Type II	Implantation type: Implanted into scar IIa: myometrial thickness ≥0.2 cm IIb: 0.1–<0.2 cm		Moderate risk of uterine rupture or heavy bleeding depending on myometrial thickness; increased vascularity often noted
	Type III	Infiltration type: • IIIa: myometrium <0.1 cm, sac near serosa • IIIb: sac protrudes beyond serosa		High risk of catastrophic rupture, massive haemorrhage, bladder invasion; often life threatening

Table 2: Key Recommendations

Guideline/Body	Classification Approach	Recommendation Focus
SMFM (2020)4	Vial (Endogenic/Exogenic); individualized	Clinical decision-making, early diagnosis
RCOG11	Descriptive imaging; supports Timor-Tritsch	Surgical planning and risk prediction
ESHRE (2022)12	Imaging-based staging (Timor-Tritsch, Delphi)	Fertility-preserving interventions
ISUOG (2021)13	Delphi consensus (3-tier system)	Standardization in diagnosis and reporting
FIGO14	Endorses Delphi-based classification	Global consistency and data harmonization

Recommendations on Diagnosis

Early first-trimester ultrasound (<9 weeks) significantly improves detection of caesarean scar pregnancy (CSP), as the gestational sac (GS) is still localized low in the uterus. With advancing gestation, the GS may appear to migrate toward the fundus, making CSP harder to identify. In one review of published CSEP case series, the average gestational age at diagnosis was 7.5_2.5 weeks.Despite this, the placenta often remains implanted within the scar "niche," serving as a critical marker for placenta accreta spectrum (PAS) in later scans. A low-lying, anteriorly positioned gestational sac on initial ultrasound should prompt careful evaluation for possible CSP.

RISK FACTORS	Clinical Presentation	IMAGING
- Multiple prior cesarean deliveries - Maternal age >35 years - High gravidity (>3 pregnancies) - >2 induced abortions - Short interpregnancy interval (<5 years) after cesarean -Retroverted uterus -Low or cervical extension of uterine incision -Inadequate scar suturing or poor healing -Surgical factors favoring adhesion formation -Patient-related factors: impaired wound healing, chronic inflammation - More often seen after cesarean for breech (a less-developed lower uterine segment and thicker hysterotomy scar, predisposing to poor healing and microscopic dehiscence.)[15]	-Vaginal bleeding (spotting to heavy bleeding). -Pelvic pain or discomfort. -Early pregnancy symptoms -Asymptomatic cases detected incidentally during early pregnancy scans.	1. Transvaginal Ultrasonography (TVUS) with Color Doppler – Primary Modality (Sensitivity ~86%) -Empty uterine cavity and endocervical canal - A Gsac occupying the cesarean scar niche,appearing triangular before 8 weeks and becoming rounded or oval after 8 weeks. -Gsac located in the anterior isthmic wall above the internal os -Discontinuity of anterior uterine wall on sagittal view through sac -Gsac/placenta embedded in hysterotomy scar -Thin (1-3 mm) or absent myometrium between sac and bladder -Negative sliding sac sign: sac remains fixed with probe pressure -Doppler: high-velocity, low-impedance peritrophoblastic flow at or in the area of a cesarean scar. -Crossover sign (COS)- This sign depicts the gestational sac's superior–inferior diameter, drawn perpendicular to the endometrial line from internal os to fundus, to assess its relation to the cesarean scar and anterior uterine wall. 2. Magnetic Resonance Imaging (MRI) – Adjunctive Tool -Confirms TVUS findings -Provides soft tissue characterization and precise myometrial thickness -Maps sac depth and invasion for surgical or fertility-preserving planning[10,16]

Figure 1: Recommendations for Diagnosis

Management

Various treatment modalities have been reported in the medical literature but with predominance of case series and limited randomized controlled trials. Recent guidelines emphasize early diagnosis through transvaginal ultrasound and individualized management based on clinical presentation, gestational age, and future fertility desires.

Overview of Treatment Options

Management of caesarean scar pregnancy (CSP) encompasses medical therapy, surgical intervention, adjunctive interventional techniques, and, in select cases, expectant management. The choice of approach is influenced by gestational size and location, myometrial thickness, presenting symptoms, and hemodynamic stability. Across all strategies, maternal safety remains the primary objective, with fertility preservation as a secondary goal whenever feasible.

Reported Treatment Modalities for CSEP [7,8,17,18,19]

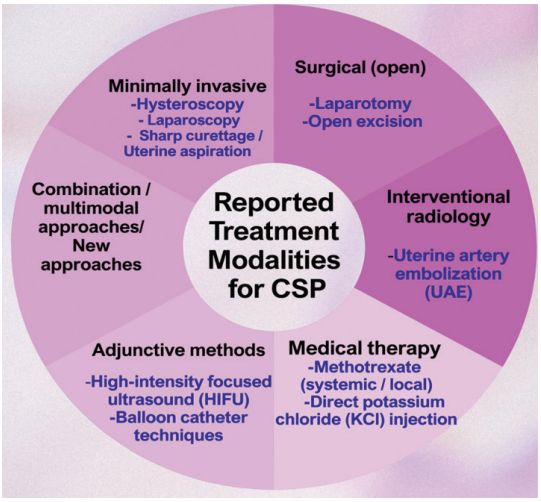

Figure 2 : Treatment Modalities

Medical Management[7,8,17,18,19]

Indications: Hemodynamically stable, early gestations, absent fetal cardiac activity, fertility-sparing preference. A beta-hCG level > 100,000 IU/L is associated with higher treatment failure.

- **Methotrexate (MTX):**
  - Local or intra-sac ± systemic administration preferred.
  - Dose: Intra-gestational injection with a dose of 1mg/kg up to 50 mg, using a 20 G needle under ultrasound guidance.
  - Intramuscular MTX alone is less effective.
- **Potassium chloride (KCl):**
  - Ultrasound guided intra-gestational KCl is preferred in heterotopic pregnancies to avoid systemic MTX embryotoxicity.

Efficacy & Monitoring:

- Efficacy varies with gestational age and vascularity of the lesion.
- Success rate: 65–90%, higher with combined local + systemic MTX.
- Serial β-hCG (days 4 & 7); ≥15% between days 4 and 7 decline indicates response.
- Levels normalize in ~3 months; monitor for haemorrhage or AVM.
- Interval ultrasonographic surveillance plays a vital role in observing for CSEP resolution.

Surgical Management

- **USG-guided suction evacuation:**
  - First-line for early gestations with myometrium >4.5 mm.
  - Safer than blind curettage; risk rises with gestational age ≥7 weeks or deep implantation.
  - Curettage alone carries a higher risk of perforation and haemorrhage than ultrasoundguided suction evacuation due to incomplete tissue removal and poor scar contractility
- **Hysteroscopic resection:**
  - Direct sac visualization, allows scar excision + myometrial repair.
- **Laparoscopic resection:**
  - Preferred in thin myometrium or high vascularity.
- **Hysteroscopic/Laparoscopic resection:**
  - Both approaches carry the advantage of scar tissue excision followed by myometrial re-approximation.
- **Open surgery (laparotomy/hysterectomy):**
  - Reserved for unstable patients or completed family; feasible in low-resource settings.

Adjunctive & Interventional Techniques

- **Uterine artery embolization (UAE):**
  - UAE can be performed pre- or post-operatively to control haemorrhage
  - Reduces intraoperative blood loss; fertility-sparing.
- **Balloon tamponade:**
  - Ultrasound guided insertion of Foley's catheter for compression in cases of hemorrhage.
  - Can be used as a standalone treatment or in conjunction with surgical evacuation to control bleeding.

Expectant Management

- **Reserved for:** Asymptomatic, non-viable CSP with declining β-hCG.
- Weekly ultrasound is advised to confirm non-progression, as some non-viable CSPs resolve spontaneously.
- **Risks:** Delayed rupture, massive hemorrhage.
- **SMFM advises against routine use.**
- If pregnancy continues: **Elective cesarean 34⁰⁄₇–35⁶⁄₇ weeks (GRADE 1C)**

Factors Influencing Choice of Management

Management decisions are influenced by:

- Hemodynamic stability good candidates for

conservative or fertility-

- Gestational age and sac size sparing interventions
- Desire for future fertility
- Myometrial thickness
- Presence of fetal cardiac activity
- Serum β-h

Follow-Up and Monitoring

- **β-hCG:** Weekly until undetectable; plateau/rise warrants reassessment.
- **Imaging:** Serial TVUS to track resolution.
- **MRI** for complex/persistent cases.
- Decrease in volume of gestational sac and degree of vascularization may be employed for monitoring.

Future Pregnancy Counselling

- **Risks:** Recurrence, PAS; require early first-trimester TVUS (<8 weeks).
- **Contraception:** Long-acting reversible or permanent methods.
- **Interpregnancy interval:** 12–24 months suggested; evidence limited.
- **Preconception imaging:** Saline sonohysterography may detect scar defects; routine surgical revision not supported.
- In a subsequent pregnancy, early transvaginal ultrasonography (<8 weeks) is recommended to confirm intrauterine implantation and exclude recurrence or PAS.
- **Delivery planning:** Elective cesarean at 34⁰⁄₇–35⁶⁄₇ weeks with corticosteroids; anticipate major hemorrhage ± hysterectomy.

Summary of International Guidelines

ISUOG, FIGO, SMFM

- **ISUOG (2022)[7]:** Recommends early transvaginal ultrasound for diagnosis; individualized management; highlights UAE and hysteroscopic resection.
- **FIGO (2021)[19]:** Emphasizes prevention through careful caesarean technique; supports both medical and surgical approaches.
- **SMFM (2020)[19]:** Suggests that operative resection (with transvaginal or laparoscopic approaches when possible) or ultrasound-guided uterine aspiration be considered for the surgical management of CSEP, and that sharp curettage alone be avoided.

Supports ultrasound-guided suction and local MTX for stable patients.

Comparison Table of Recommendations

Organization	Diagnosis	Medical Management	Surgical Options	Adjunctive Measures
ISUOG	Early TVUS	Local/systemic MTX	Hysteroscopy, suction	UAE, tamponade
FIGO	Early TVUS	Systemic MTX	Laparoscopy, laparotomy	UAE
SMFM	Early TVUS	Local MTX	Suction evacuation/ Operative resection	Not specified

Discussion

Evolving trends favor uterus-sparing, minimally invasive interventions, including UAE, Uterine

Artery Chemoembolization (UAC), hysteroscopy and HIFU[20,21,22].

a) Uterine Artery Chemoembolization (UAC) in CSP

- **Efficacy:** Reported success rate 83–99%.
- **Outcome determinants:** Gestational sac size, fetal cardiac activity, and CSP type.
- **β-hCG kinetics:** Levels usually normalize within 1 month posttreatment.
- **Meta-analysis findings (Qiao et al.):**
  - Faster β-hCG resolution
  - Shorter hospital stay
  - Reduced blood loss
  - Fewer adverse events than systemic MTX
- **Technique:** Minimally invasive intraarterial MTX injection, often combined with suction curettage for optimal results.

b) High-intensity focused ultrasound (HIFU) + USG-D&C:

- Safe and minimally invasive for CSP-I and CSP-II;
- Prospective QoL (quality of life) data lacking.

Evidence gaps include:

1. Lack of randomized controlled trials.
2. Limited long-term fertility and recurrence data.
3. Unclear optimal interpregnancy interval.

Need for Multidisciplinary Approach

Management of CSP requires collaboration between

obstetricians, radiologists, interventional radiologists, and fertility specialists for optimal outcomes.

Conclusion

Scar ectopic pregnancy is an emerging clinical challenge driven by rising caesarean delivery rates and the complexity of both diagnosis and management. Management strategies remain individualized, guided by hemodynamic stability, gestational age, imaging findings, and fertility preferences, as no universal protocol currently exists. Early detection using highresolution transvaginal ultrasonography and coordinated interdisciplinary care are pivotal to optimizing outcomes.

Emerging evidence links postcaesarean 'niche' formation—caused by poor myometrial approximation or inadvertent endometrial inclusion—to impaired scar healing and abnormal implantation in subsequent pregnancies, often predisposing to placenta accreta spectrum. Surgical techniques that restore normal uterine wall anatomy and endometrial continuity may reduce defect prevalence and improve reproductive safety. Standardized, evidencebased protocols are needed to balance maternal safety with fertility preservation.

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Placental & cord abnormality : Quiz

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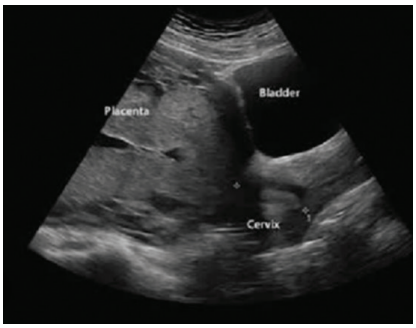
Assistant of Professor, Department of Obstetrics & Gynaecology, Lady Hardinge Medical College, New Delhi

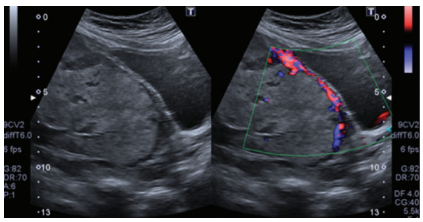
Q1: Identify the type of placenta previa shown:



A) Lowlying placenta
B) Marginal previa
C) Partial previa
D) Complete previa

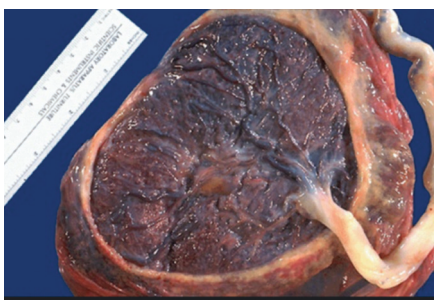
Answer D
Radswiki T, Campos A, Alhusseiny K, et al. Placenta previa. Radiopaedia.org. 2024.

Q2: What ultrasound finding is characteristic of placenta accreta?



A) Multiple placental lacunae with turbulent flow
B) Hypoechoic placental lake
C) Retroplacental hematoma
D) Thickened placental edge
Answer: A
Ref: Radswiki T, Knipe H, Salehzadeh H, et al. Placenta accreta. Radiopaedia.org 2025

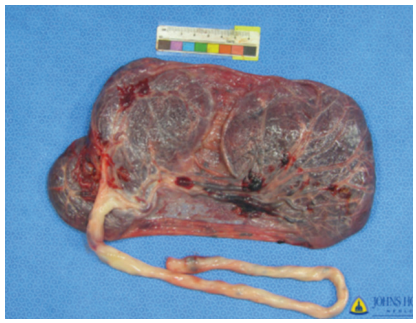
Q3: Which feature indicates circumvallate placenta?



A) Bilobed lobes
B) Thickened annular fetal surface edge
C) Vasa previa vessels
D) Hematoma beneath placenta

Answer: B

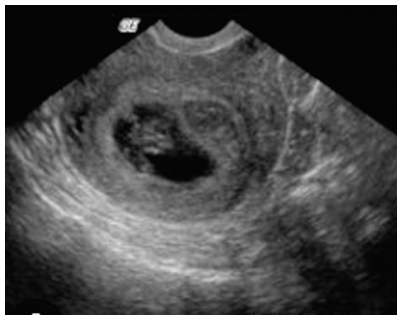
Q4: Which is a complication of bilobed placenta?



A) PAS
B) Vasa previa
C) Abruption
D) Chorangiosis

Answer: B

Q5: Crescent-shaped hypoechoic area between chorion and uterus on first-trimester scan shows which of the following?



A) Subchorionic hematoma
B) Placental Lake
C) PAS
D) Vasa previa
Answer: A
Ref: Chhabra A, et al. Subchorionic hemorrhage overview. Medscape. 400971. 2025

Q6. Which of the following placental abnormalities is most strongly associated with vasa previa?

A. Placenta percreta

B. Succenturiate lobe

C. Circumvallate placenta

D. Placenta membranacea

Answer: B. Succenturiate lobe

Cunningham FG, Leveno KJ, Bloom SL, et al. Williams Obstetrics, 26th Edition. McGraw-Hill Education, 2022.

Q7. Which of the following is most commonly associated with a chorioangioma >5 cm in size?

A. Preeclampsia

B. Polyhydramnios

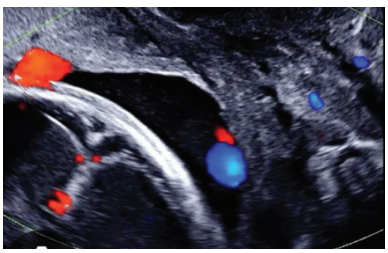
C. Intrauterine fetal demise

D. Placenta previa

Answer: B. Polyhydramnios

Zalel Y, Gamzu R, Weiss Y, et al. Chorioangiomas of the placenta: sonographic and Doppler flow characteristics. J Ultrasound Med. 2002.

Q8: Identify the condition.



Which is the most appropriate next step in management after identifying this finding on ultrasound?

A) Immediate delivery

B) Expectant management till term

C) Corticosteroids and planned cesarean at 34–36 weeks

D) Amniocentesis for fetal lung maturity

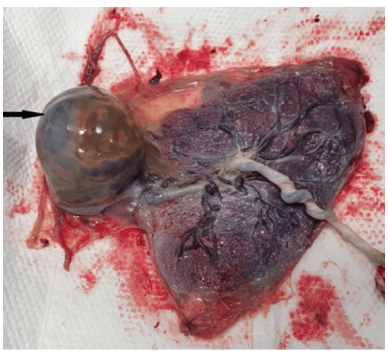
Answer: C) Corticosteroids and planned cesarean at 34–36 weeks

Explanation: Vasa previa carries a high fetal mortality risk if undiagnosed; cesarean before rupture is critical.

Reference: ACOG Practice Bulletin No. 183 (2024): Postpartum Hemorrhage; UpToDate – Vasa Previa

Q9. A large reddish, well-circumscribed mass protruding from the fetal surface of the placenta

This placental mass is associated with all of the following except:



A) Polyhydramnios

B) Fetal hydrops

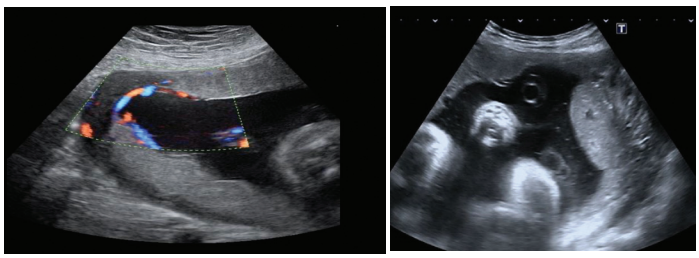
C) Intrauterine growth restriction

D) Umbilical artery thrombosis

Answer: D) Umbilical artery thrombosis

Explanation: Chorioangioma is a benign vascular tumor that can cause polyhydramnios, fetal hydrops, and IUGR, but not directly vessel thrombosis.

Q10.The following ultrasound image shows a smaller accessory placental lobe separate from the main disc, with connecting vessels bridging between them on color Doppler. No vessels cross the cervical os.



Which diagnosis and key complication is most consistent with these features?

A. Bilobed placenta with marginal insertion, risk of placenta previa

B. Succenturiate lobe of placenta, risk of retained placental tissue postpartum

C. Placenta membranacea, risk of placenta accreta spectrum

D. Circumvallate placenta, risk of fetal growth restriction

Answer: B. Succenturiate lobe of placenta, risk of retained placental tissue postpartum
Explanation: A succenturiate placenta consists of one or more accessory lobes connected by vessels, increasing the chance of retained lobe postpartum and postpartum hemorrhage

AOGD Clinical meeting 25th July 2025, Army Hospital (Research & Referral)

Case 1 : Random Start Protocol : Medicine that adapts is medicine that empowers

Presented by : Lt Col Ipsita Sahoo

Discussed by : Col Nikita Naredi

Random start stimulation is a novel approach in controlled ovarian stimulation where treatment is initiated at any point during the menstrual cycle, irrespective of the conventional day 2 or 3 start. This strategy has revolutionized fertility treatment in oncology patients. We carried out a prospective observational study to see the outcome of random start stimulation in 56 patients with PCOS and 6 patients with malignancy undergoing IVF cycle. Oocytes were retrieved in all cases. Embryo formation rate was 87.5 %. Random start stimulation appears to be a promising tool in PCOS IVF cycles too.

Case 2 : A case of Frasier syndrome

Presented by : Maj Kapila Saxena

Discussed by : Wg Cdr Abha Khurana

Frasier syndrome is a rare genetic disorder resulting from mutation in WT1 gene. Individuals have 46XY karyotype, female external genitalia, progressive steroid resistant nephrotic syndrome and are at high risk for gonadal tumours like gonadoblastoma and dysgerminoma. Our case is a 15 year old girl who presented with primary amenorrhoea, end stage renal disease and on evaluation was diagnosed as Frasier syndrome. Laparoscopic bilateral gonadectomy was done followed by renal transplantation in her case. Early genetic evaluation, appropriate surgical intervention and multidisciplinary approach is essential in such cases.

Case 3 : Laparoscopic management of Accessory Cavitated Uterine Malformation

Presented by : Maj Gunjan Bhutani

Discussed by : Col Bikram Bharadwaj

Accessory cavitated uterine malformation (ACUM) is a rare mullerian anomaly, characterized by non communicating accessory uterine cavity. Patients present with increasing pelvic pain and dysmenorrhea. We report two cases of ACUM ; first patient presented with primary infertility and 5.6 × 4.6 × 4.2 cm ACUM was excised laparoscopically. Second patient had chronic pelvic pain and on laparoscopy 1.5 × 2.3 × 1.7 cm ACUM was removed. Awareness of this entity is required for timely detection and correct management.

Events Held 2025

From Suspicion to Survival: Red flags and real-world management conducted by Max Saket, New Delhi in collaboration with AOGD Oncology committee on 5th July, 2025

Webinar on "Silent Threat: Unmasking Rh Isoimmunisation in Antenatal Care" conducted by Fetal medicine & Genetics Subcommittee AOGD on 8th July 2025

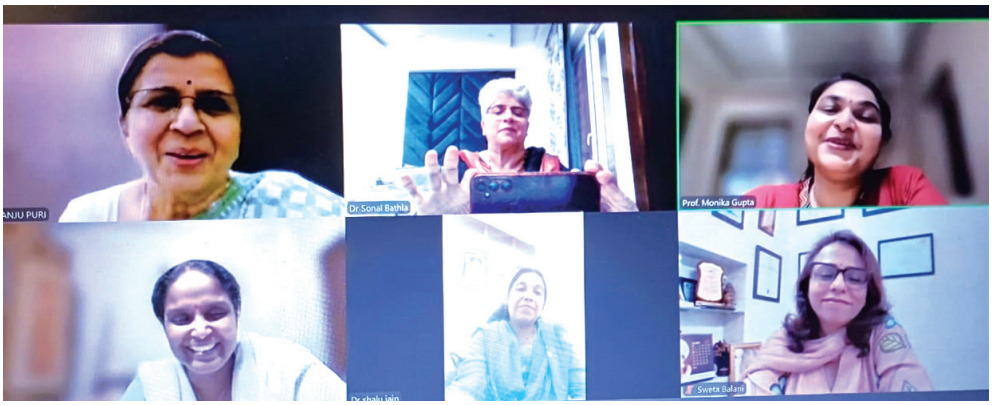
Public Forum on "Family Planning" on the occasion for World Population Day, conducted by Dept. of Obst & Gynae, LHMC & SSK Hospital in association with AOGD on 11th July 2025

Public Awareness Session on Contraception On occasion of World Population day conducted by Community Health and public awareness Sub Committee AOGD on 11th July, 2025

Skill Enhancing Workshop in Colposcopy and Treatment of Pre Invasive lesion of Cervix conducted by Oncology Committee, AOGD in collaboration with ISCCP, DGF& Oncology Committee, FOGSI on 13th July 2025

Webinar on "Scar ectopic and cesarean complications" conducted by Fetal Medicine & Genetics Subcommittee AOGD on 15th July 2025

Webinar on "Non-Descent Vaginal Hysterectomy (NDVH)" conducted by Urogynae Committee AOGD in association with Society of Vaginal Surgeons India (SOVSI), and DGF Civil Lines on 15th July 2025



Aprajita Sampoorna CME conducted by QI Obst gynae practice subcommittee of AOGD on 16th July 2025

FOGSI Presidential Initiative Aparajita Sampoorna CME conducted by Community Health and public awareness Subcommittee AOGD on 17th July 2025

Aparajita Sampoorna CME conducted by Infertility & Reproductive Endocrinology Committee of AOGD 18th July 2025

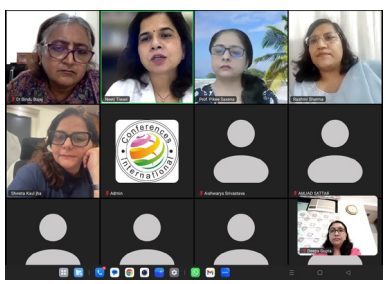

CME on "Family Planning: Empower Women, Empower Future: Build Families With Choices"conducted By Dept. of Obst & Gynae, LHMC & SSK Hospital in association with AOGD on 19th July 2025

CME on "Demystifying the Success of Assisted Reproductive Technology " conducted by VMMC & SJH Hospital in association with AOGD & IFS on 19th July 2025.

The AOGD Monthly Clinical Meeting (virtual) conducted by the Department of Obst & Gynae, Army Hospital – Research & Referral, New Delhi on 25.07.2025

ISCCP in collaboration with AOGD Oncology Committee, NARCHI Delhi and SOVSD organised a conference V Insight 2025 at Hotel Lalit , New Delhi on 26.7.25.


Webinar on "PCOS" conducted by Infertility and Reproductive Endocrinology in association with SIG Early Pregnancy IFS and ISAR on 31st July 2025



Guidelines for submission of Free Communication (Oral & E- Poster)

1. Last date for abstract submission is 25th August, 2025.
2. Only registered delegates are entitled to submit posters/papers.
3. One must be a life / annual member to submit oral/ poster in the conference.
4. Presenting author details – phone no. & email Id should be entered into the submission system. All further correspondence will be sent only to the contact email entered.
5. Students should submit a certificate forwarded by their Head of the Department.
6. Abstracts are to be submitted on the following themes:
   - High Risk Obstetrics
   - Gynaecological Oncology
   - Endoscopy
   - Reproductive Endocrinology
   - Miscellaneous
7. Theme to be selected at the time of submission.
8. All Case reports will be admitted as Poster Presentation.

Instruction for the abstract

a) Title should be concise and short.

b) The names of authors should follow immediately under the title (Maximum 6 authors). Underline the presenter's name. Do not include degrees or professional designations.

c) The names of institution, city and country should follow after the authors names, on a different line.

9. Abstract should be upto 250 words.

a) Text should be in lower case, black only, Font: Times New Roman, Font size: 11

b) Headings listed below are to be used to construct the abstract:
   - Introduction: Describe the background supporting the relevance of the research question
   - Objective: State the purpose of the study or investigation.
   - Methods: State details on study subjects, techniques, and/or observational/analytical methods.
   - Results: Include the main findings, and statistical data.
   - Conclusions: Summarize principal conclusions, emphasizing new and important aspects.

   Poster should be divided into 3 sections

   Background, Case Report , Discussion.

c) Use of standard abbreviations is desirable. The first time it appears, the abbreviations are to be written in brackets after the full word.

d) Use numerals to indicate numbers, except in the beginning of sentences.

e) Do not include graphs and references in the abstract.

f) Use single-line vertical spacing and leave one line between paragraphs.

11. Decision of scientific committee / judges will be final.

AOGD 2025

47th Annual Conference of AOGD

Organized By:
Department of Obstetrics and Gynaecology
Lady Hardinge Medical College
New Delhi

13th & 14th September 2025 | Venue: India Habitat Centre, New Delhi

Tiny heartbeats to timeless strength - Honouring the journey of women through birth & beyond

REGISTRATION FORM

AOGD Member: ☐ Yes ☐ No **AOGD Membership No:** _____ **DMC No:** _____

Title: Prof. ☐ Dr. ☐ Mr. ☐ Ms. ☐ Mrs. ☐ **Gender :** Male ☐ Female ☐

First Name: _____ **Middle Name:** _____ **Last Name :** _____

Address: _____

Country: _____ **City:** _____ **State:** _____ **Pin:** _____

Telephone: _____ **Mobile No. With Country Code :** _____

Email: _____

(Please use block letter only)

(All the above fields are mandatory)

CONFERENCE REGISTRATION FEES

CATEGORY	Early Bird (Till 30th June 2025)			Regular (1st July to 15th Aug 2025)			From 16th August 2025 Onwards/On-spot		
	Amount	GST 18%	Total	Amount	GST 18%	Total	Amount	GST 18%	Total
☐ AOGD Member	6000	1080	7080	6500	1170	7670	7000	1260	8260
☐ Non-Member	7000	1260	8260	7500	1350	8850	8000	1440	9440
☐ PG Students	5000	900	5900	5500	990	6490	6000	1080	7080
☐ AOGD Member (above 75yrs)	Complimentary (Kindly email duly filled Registration Form along with age proof on our offcial email id mentioned below)								

Pre-Conference Workshop - 11th - 12th September 2025

Early Bird (Till 30th June 2025)			Regular (1st July to 15th Aug 2025)			From 16th August 2025 Onwards/On-spot		
Amount	GST 18%	Total	Amount	GST 18%	Total	Amount	GST 18%	Total
1500	270	1770	1800	324	2124	2000	360	2360

Opting For: 11th ☐ 12th ☐ Both Days ☐

Pre-Conference - 11th September 2025 (Tick your choice of workshop)

Name of Workshop	Time	Venue
☐ Mastering POP Surgery: Techniques, Complications, and Comprehensive Management	9:00 AM - 2:00 PM	Auditorium, Sant Parmanand Hospital, Civil lines, Delhi
☐ Laparoscopy and beyond: A hands on workshop	9:00 AM - 5:00 PM	Skill centre, Sir Gangaram Hospital
☐ From Imaging to Incision: Advancing Precision in Gynae-Oncologic Surgery	9:00 AM - 2:00 PM	AIIMS, New Delhi
☐ Preventive Oncology	10:00 AM - 4:00 PM	Library Hall UCMS & GTB Hospital Delhi
☐ Bringing quality control into managing PCOS	09:00 AM - 5:00 PM	Hotel Eros Nehru Place
☐ Maternal Hope: Ending Preventable Losses, Saving Lives	10:00 AM - 4:00 PM	Northern Railway hospital auditorium, Connaught Place.
☐ Menopause prescription: Hormones and more, Master the art	9:00 AM - 2:00 PM	Mini Auditorium, LHMC

Pre-Conference - 12th September 2025 (Tick your choice of workshop)

Name of Workshop	Time	Venue
☐ Teens Timelines & Trust : Demystifying Amenorrhea and Contraception	9:30 AM - 1:00 PM	Kailash Deepak Hospital , Vikas Marg Delhi -110091
☐ ENDOMETRIOSIS DECODED What the text books don't tell.	1:00 PM- 5:00 PM	AIIMS, New Delhi
☐ VAX TALK .... Adults Too Need Vaccines	1:00 PM – 5:00 PM	Sir Gangaram Hospital Auditorium
☐ Bump to Birth: Foundations of Fetal Health &Genetics	10:00 AM - 5:00PM	Old LT, Behind OPD Block, VMMC & Safdarjung Hospital, New Delhi – 110029
☐ Controversies in Reproductive Medicine: Case-Based Challenges in Infertility and IVF	9:00 AM - 4:00 PM	Mini Auditorium,LHMC
☐ Postpartum Haemorrhage: Prevention & Cure- Learn The Art	2:00 PM - 5:00 PM	Auditorium, ABVIMS and Dr RML hospital

Note:

- The above-mentioned fees are applicable per workshop. If a participant wishes to attend 2 workshops, the fee will be charged separately for each.
- Post graduates to attach a certificate from HOD and also should be a member of the AOGD in order to attend and present a paper.
- Membership number is mandatory for registration in membership category. For any queries related to membership, you may contact **Ms. Sarita** (+91 92116 56757).
- For spot registration: payment will be accepted only by mode of Cash/Card/UPI.
- The disbursal of Delegate kit for the same will be subject to availability Delegate kit would be handed over only to registered delegate.
- Registration is non transferable. Post conference, no kit or any workshop material will be disbursed to the Delegate/associate Delegate/PG student.

Offline Payment Details

Account Name: SEM Events and Meetings OPC Pvt Ltd **Account No:** 143611010000011 **Bank Name:** Union Bank of India

Branch: Indirapuram Branch (NCR) **IFSC Code:** UBIN0814369 **MICR Code:** UBININBBGHZ

Mode of Payment: NEFT/RTGS ☐

TR/Reference Number/Transaction Id : _____

Disclaimer: Before making the payment, we request you to fill the online registration form, for meeting the criteria of the conference & send the transaction details to the conference email ID for registration confirmation.

For Cheque/DD Payments

For Cheque/DD Payment: Please issue the cheque in favor of "ASSOCIATION OF OBSTETRICIANS AND GYNAECOLOGISTS OF DELHI"
Submit to: **Ms. Sarita** (+91 9211656757)
Dept. of OBGYN
Lady Hardinge Medical College & Hospitals
New Delhi – 110001

Payment Details

DD/Cheque No: _____ **Dated:** _____

Drawn on (Name of the Bank): _____ **Branch:** _____ **Amount:** _____

Cancellation & Refund Policy

1. All cancellation should be made in writing and sent to AOGD secretariat.
2. All cancellation received on or before 15th July 2025 will be entitled for 75% refund of the amount paid.
3. All cancellation received between 16th July 2025 to 14th August 2025 will be entitled for only 25% of the amount paid.
4. No refund for cancellation made on or after 15th August 2025.
5. The refund process will begin only 30 days after the completion of the conference

NOTE: The organizing committee shall not be held liable for any delay or cancellation of the AOGD 2025 conference due to events beyond its control, including natural disasters, terrorism, war, or labor disputes.

AOGD Office

Secretariat Address
AOGD, Department of Obstetric and Gynaecology, Lady Hardinge Medical College & Associated Hospitals, NEW DELHI- 110001
Email: aogdlhmc2025@gmail.com
Telephone: 011-23408297
Mobile: 9717392924

Conference Manager

Sem Events & Meetings OPC Pvt. Ltd.
59-60, A2, Shiv Arcade, Acharya Niketan, Mayur Vihar Phase 1 New Delhi, India
M: +91 81714 92255 | 93544 81701
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Association of Obstetricians & Gynaecologists of Delhi

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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

68th All India Congress of Obstetrics & Gynaecology

aicog Delhi 2026

Advocate Innovate Challenge Optimise Globalise

Ek Rashtra - Ek Mission
Swastha Naari - Samruddha Vatan
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Conference Theme

68th AICOG 2026 DELHI
All India Congress of Obstetrics & Gynaecology

Inspire, Include, Invent: Improving Women's Health in India

All India Congress of Obstetrics & Gynaecology

14-18 January, 2026
Yashobhoomi, Dwarka | New Delhi
(India International Convention & Expo Centre)

Abstract Submission is Now Open

Abstract Themes

1. Maternal & Child Health
2. Minimal Invasive Gynaecological Surgery
3. Population Stabilization
4. Sexual & Reproductive Health
5. Gynaecologic Oncology
6. Midlife & Geriatric Gynaecology
7. Innovation in OBGYN
8. Miscellaneous

Last Date of Submission
15th October

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CHIEF ORGANISER - LEGENDS GO LIVE
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- RECORD FOR THE LARGEST FIBROID REMOVE LAPAROSCOPICALLY OF 6.5 KG
- RECORD FOR THE OLDEST PATIENT OPERATED IN THE WORLD OF 107 YEAR OLD
- RECORD FOR THE LARGEST UTERUS REMOVED LAPAROSCOPICALLY OF 9.5 KG

OPERATING FACULTY


Dr. Mario Maizoni


Dr. Nikita Trehan


Dr. Jay Mehta


Dr. Sandesh Kade


Dr. Shailesh Puntambekar


Dr. Dipak Limbachiya


Dr. Rajesh Modi


Dr. Sanjay Patel


Dr. Osama Shwaki

OT Co-ordinator


Dr. Pooja Garg

PROGRAM DETAILS

DAY 1 - Saturday, Aug. 30, 2025

We have planned a "Surgical Bonanza" where more than 25 surgeries will be relayed LIVE from Sunrise Hospital to Hotel Hyatt Regency Bhikaji Cama Place New Delhi from 08:00 AM to 08:00 PM.

DAY 2 - Sunday, Aug. 31, 2025

8:30 AM to 4:30 PM - "Conference CME and Socratic Seminar" at Hotel Hyatt Regency New Delhi (Oval Banquet)

4:30 PM to 5:00 PM - VALIDECTORY

PLANNED SURGERIES

ENDOMETRIOSIS OT

- Demonstration of CO2 Laser (Boston Scientific) for Endometrioma Ablation
- Laparoscopic Shaving / Discoid Resection of Rectovaginal (RV) Endometriosis
- Laparoscopic Excision of Bladder Nodule
- Laparoscopic Excision of Diaphragmatic Nodule
- Laparoscopic Excision of Sciatic Nerve Endometriosis

BENIGN SURGERY & HYSTEROSCOPY OT

- Laparoscopic Myomectomy
- Laparoscopic Hysterectomy"Sunrise Method"
- Laparoscopic Recanalization
- Hysteroscopic Septal Resection
- Hysteroscopic Subendometrial Stem Cell Injection
- Hysteroscopic Myomectomy

ONCO & ADVANCED OT

- Laparoscopic Extra-Fascial Hysterectomy & Pelvic + Para-Aortic Lymphadenectomy
- Laparoscopic VVF Repair
- Laparoscopic Pregnant Cerclage
- Laparoscopic Ureteric Reimplantation
- Laparoscopic Ileal Vaginoplasty
- Laparoscopic Adenomyomectomy "Sunrise Method"

REGISTRATION FEES DETAILS

REGISTRATION FEES :- Rs 9,500/-
SPOT REGISTRATION :- Rs 11,000/-
ACCOMPANYING PERSON :- SAME AS ABOVE
FOR PG STUDENTS :- Rs 6,000/-
(LETTER FROM HOD IS COMPULSORY)

SPECIAL DISCOUNT (FOR PG STUDENTS)

FOR REGISTRATION DETAILS PLEASE CONTACT ON:

MR. SANJEEV KHURANA: +91-9213179913
MR. RAVI PRAKASH: +91-9711437535

AOGD SECRETARIAT
Department of Obstetrics and Gynaecology
Lady Hardinge Medical College & Associated Hospitals, New Delhi-110001
Tel.: 011-23408297, (M) : 9717392924 | Email Id: aogdlhmc2025@gmail.com