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AOGD BULLETIN

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

THEME: OBESITY AND ESTROGEN DEPENDENT DISORDERS - CLINICAL IMPLICATIONS FOR FERTILITY AND REPRODUCTIVE HEALTH

AOGD SECRETARIAT
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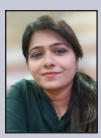

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AOGD Bulletin

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Dr. Pikee Saxena & Dr. Manisha Kumar on behalf of Association of Obstetricians & Gynaecologists of Delhi

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From the President's desk

Dear AOGD members,

Greetings from AOGD

As we know cancer cervix is the second most common cancer in women in India. The government of India has recently lauched a nationwide HPV Vaccination campaingn on 28th Feb. The programme provides free, voluntary single dose vaccination through schools and government health facilities. In the initial phase specific focus is on 14year old girls. AOGD has received a letter from MOHFW asking us to spread awareness regarding this campaign through our members. I urge all the members to make the public aware of this campaign so that more and more girls can be vaccinated.

The last online clinical meeting of our tenure is being organised by dept of obstetrics and Gynaecology, Lady Hardinge medical college. Three very interesting cases will be presented.

As we have come to end of our tenure, the Last GBM is scheduled on 30th March at MEU Hall at Lady hardinge, it will be followed by Handing over of AOGD Secretariat to AIIMS. The focus of this month bulletein is "Obesity and estrogen dependent disorders - clinical implications for fertility and reproductive health.

As Obesity is now recognised as a disease, it is important that we be aware of this and it's implication on women's health. Dr Pikee and her team has done a great job in bringing out this issue on this important topic.
Happy reading !
Hope to see you all at GBM.
Best wishes

Dr Reena Yadav
President AOGD

From the Secretarial Desk

Dr Ratna Biswas
Honorary Secretary

Dr Sharda Patra
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Dr Swati Agrawal
Joint Secretary

Dr Anuradha Singh
Joint Secretary

Season's greetings for a blossoming spring !

The month of February witnessed a spree of activities , foremost amongst which were the Public Awareness program on breast and cervical cancer on occasion of World Cancer Day organized by breast & cervical cancer subcommittee at GTB Hospital and AOGD Secretariat at Lady Hardinge Medical College on 1st & 4th February respectively. Both the activities had a sizeable attendance and was well received by the attendees.

CME on Vulval Disorders was organized at BLK hospital on 6th February by Oncology subcommittee of AOGD in association with DGF. The CME was focused on benign, premalignant and melanin disorders of the vulva.

A practical training course on Vulvo-Vaginal Module was conducted by Oncology Subcommittee of AOGD wherein hands on training was imparted on Vulvo-Vaginoscopy and Dermascopy.

Masterclass on Vulvar lesions was organized by AIIMS, Delhi under aegis of ISCCP, FOGSI and Oncology subcommittee of AOGD on 14th February 2026.

Adolescent health subcommittee organized a webinar on Abnormal uterine bleeding and ten rules of adolescent health care on 14th February. Their subcommittee members also participated in public awareness talk on primary prevention of cervical cancer through HPV vaccination.

Anemia detection and awareness camp was organized by Community Health & Public Awareness subcommittee on 16th February on occasion of World Anemia Day. Haemoglobin testing was conducted on 225 students and few teachers . Health talks were given on nutrition and prevention of anemia.

Webinar on PPH was conducted by the Safe motherhood subcommittee.

This month's Bulletin is structured on topics of "Obesity and estrogen dependent disorders - clinical implications for fertility and reproductive health" which are increasingly encountered in our clinical practice and requires a concentrated effort by the gynaecologists and allied specialities to manage the complexities of this condition. I congratulate the Editorial team headed by Dr Pikee for highlighting the intricacies and advances in this field and wish them all the success.

News in Focus : 28th February 2026: The Nationwide Single Dose HPV Vaccination Drive was Launched for 14-Year-Old Girls.

AOGD Secretariat

From the Editor's Desk

Dr Pikee Saxena

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Obesity and estrogen dependent disorders - clinical implications for fertility and reproductive health

Estrogen plays a central role in female reproductive physiology; however, dysregulated estrogen signaling also underlies several common gynecologic disorders that significantly impact women's quality of life and reproductive potential. Conditions such as endometriosis, adenomyosis, and uterine fibroids represent a spectrum of estrogen-dependent diseases characterized by chronic inflammation, aberrant tissue proliferation, and altered uterine function. In recent years, growing insights into metabolic influences, neurobiological mechanisms of pain, advances in imaging, and evolving therapeutic options have transformed our understanding and management of these disorders.

One of the most important systemic contributors to estrogen excess in contemporary clinical practice is obesity. Adipose tissue acts as an endocrine organ capable of converting androgens to estrogens through aromatization, thereby contributing to a hyperestrogenic milieu. This metabolic environment not only predisposes women to gynecologic disorders but also influences fertility, pregnancy outcomes, and long-term reproductive health. The expanding role of glucagon-like peptide-1 receptor agonists in obesity management offers new opportunities for gynecologists to address metabolic dysfunction while improving reproductive outcomes.

Alongside metabolic factors, the clinical burden of estrogen-dependent disorders is often dominated by chronic pelvic pain. Endometriosis and adenomyosis are increasingly recognized not merely as structural diseases but as complex conditions involving inflammatory pathways, neuroangiogenesis, and central pain sensitization. Understanding these mechanisms is essential for improving diagnosis and tailoring multidisciplinary management. Moreover, pelvic pain frequently coexists with other entities such as pelvic myofascial pain and bladder pain syndrome, highlighting the need for structured evaluation and integrated care strategies.

For women seeking fertility, clinical decision-making requires careful assessment of disease severity and its impact on reproductive outcomes. Tools such as the Endometriosis Fertility Index provide valuable prognostic information following surgical management and help guide individualized treatment strategies. At the same time, advances in imaging have enabled the development of radiology-based scoring systems that assist clinicians in determining whether medical or surgical intervention is warranted before proceeding to assisted reproductive technologies.

Adenomyosis, once considered primarily a condition of multiparous women with abnormal uterine bleeding, is now increasingly recognized in infertile populations. Its influence on implantation, pregnancy rates, and assisted reproductive technology outcomes continues to be an area of active investigation. Determining when and how to treat adenomyosis prior to in vitro fertilization remains an important clinical question requiring a nuanced, evidence-based approach.

Therapeutic innovations are also expanding the range of medical options available to clinicians. The introduction of oral gonadotropin-releasing hormone antagonists with add-back therapy represents a significant advance in the management of heavy menstrual bleeding associated with uterine fibroids. Such treatments offer effective symptom control while minimizing hypoestrogenic side effects, thereby improving patient acceptability and expanding non-surgical treatment pathways.

This issue brings together expert perspectives addressing the metabolic, pathophysiological, diagnostic, and therapeutic dimensions of estrogen-dependent gynecologic disorders. By integrating insights from gynecology, reproductive medicine, radiology, and pain management, the articles aim to provide clinicians with practical approaches to optimize care and fertility outcomes for affected women.

As the field continues to evolve, a holistic understanding of estrogen-driven disorders—spanning systemic risk factors, disease mechanisms, and fertility-focused management—will be essential in delivering personalized and effective reproductive healthcare.

The Editorial Team

Obesity Management in Women with GLP-1 Receptor Agonists: A Gynaecology-Focused Perspective

Pikee Saxena
Director Professor, Department of Obstetrics and Gynaecology, Lady Hardinge Medical College, New Delhi

Introduction

Obesity is a rapidly rising chronic disease globally and in India, with a disproportionate burden among women. Globally, approximately 43% of adults are overweight, with prevalence projected to increase further by 2035. In India, over 5% of adolescents are affected, while the ICMR–INDIAB study estimates that 254 million adults have generalized obesity and nearly 351 million have abdominal obesity.

In women, obesity extends beyond excess body weight and has important reproductive and metabolic consequences across the life course. It adversely affects women with polycystic ovary syndrome, fertility, pregnancy outcomes, cardiometabolic risk, metabolic dysfunction–associated steatotic liver disease, and overall quality of life from adolescence through postmenopause. Indian women develop these complications at lower body mass index thresholds due to a higher propensity for central adiposity and insulin resistance, emphasizing the need for early identification and timely intervention within gynecological practice.

Diagnosis and Risk Stratification in Indian Women

Body mass index is the most practical screening tool, with Asian cut-offs defining overweight as 23–24.9 kg/m^2 and obesity as ≥25 kg/m^2. Anthropometric assessment should include height, weight, waist circumference, and waist-to-hip ratio, with clinical screening for menstrual irregularities, polycystic ovary syndrome, and Cushingoid features.

Baseline evaluation should focus on identifying obesity-related comorbidities and includes assessment of glycemic status, lipid profile, liver enzymes, and fasting insulin or insulin resistance indices where indicated. Targeted endocrine evaluation and additional investigations are guided by clinical presentation, enabling individualized risk stratification and appropriate therapeutic escalation.

Lifestyle and Behavioural Therapy as the Foundation of Care

Lifestyle modification is the foundation of obesity management and should be offered to all women with overweight or obesity. It includes dietary caloric restriction, increased physical activity, and behavioural strategies tailored to cultural preferences, metabolic needs, and comorbidities. However, long-term success with lifestyle therapy alone is often limited due to compensatory biological adaptations, including increased appetite and reduced energy expenditure, which promote weight regain. In women, menopausal hormonal changes further worsen visceral adiposity and lean mass loss, explaining why many are unable to achieve or sustain the clinically meaningful 5–10% weight loss required for metabolic and reproductive benefit through lifestyle measures alone.

The Incretin Pathway: Why GLP-1 and Related Agents Work

Incretins are gut-derived hormones released in response to nutrient intake that play a central role in appetite regulation and glucose homeostasis. Glucagon-like peptide-1 (GLP-1) RA act through coordinated central and peripheral mechanisms to reduce hunger, increase satiety, lower energy intake, delay gastric emptying, and improve glucose-dependent insulin and glucagon regulation. These effects address both the homeostatic and hedonic drivers of eating behavior, making GLP-1RA particularly effective for obesity management.

Dual agonists that activate both GLP-1 and Glucose-dependent insulinotropic polypeptide (GIP) receptors further influence appetite control and metabolic pathways, resulting in enhanced weight loss in clinical trials. Emerging triple agonists that target GLP-1, GIP, and Glucagon Receptors are under promising and aim to combine satiety signalling with increased energy expenditure. However, these agents remain investigational and are not yet part of routine clinical guidance in practice.

Who Should Receive Anti-Obesity Medications

Anti-obesity medications (AOMs) are recommended as an adjunct to lifestyle intervention in adults with a BMI ≥30 kg/m^2, or a BMI of 27–30 kg/m^2 in the presence of at least one weight-related comorbidity such as type 2 diabetes mellitus, hypertension, dyslipidemia, MASLD, or PCOS. In reproductive-aged women, AOMs may also be considered at BMI ≥27 kg/m^2 after careful assessment of metabolic risk, menstrual function, fertility intentions, and contraception or pregnancy planning.

Obesity is increasingly recognized as a chronic, relapsing disease. Pharmacotherapy should therefore be framed as part of long-term disease management rather than a short-term intervention, with ongoing lifestyle support and

regular follow-up.

Incretin-Based Pharmacotherapy for Obesity: Evidence Across Molecules

Semaglutide

Semaglutide is one of the most extensively studied GLP-1RA for chronic obesity management. Across the STEP clinical trial program, once-weekly Semaglutide produced sustained weight loss of ~ 15–18 % over 68–104 weeks, along with consistent improvements in cardiometabolic parameters. Across the STEP trials, women comprised the majority of participants (≈50–80% across studies), making the evidence highly relevant to female obesity management. Real-world studies further confirm clinically meaningful reductions in body weight and BMI in routine practice, including among women with obesity and related comorbidities.

Greater Weight Loss in Women

Post hoc analyses of the STEP program demonstrate greater mean weight loss with Semaglutide in women than in men. Over 68 weeks, mean body weight reduction was ~16.1 % in females compared with 11.6% in males. Placebo-treated women showed slightly lower weight loss than placebo-treated men, supporting a treatment-specific benefit of Semaglutide in women.

Benefits Beyond Weight Loss

Greater weight loss with Semaglutide is associated with improvement in obesity-related complications relevant to gynaecology. Women achieving 10–15% or ≥15% weight loss reported the highest rates of improvement in urinary incontinence, with 44–45% experiencing a reduction in episodes, compared with approximately 26% among those achieving <5% weight loss.

Semaglutide also provides metabolic benefits independent of weight loss. In STEP 2, treatment was associated with a 0.35% reduction in HbA1c, a fasting plasma glucose reduction of 8.5 mg/dL, and a 28% improvement in insulin resistance as measured by HOMA-IR (ETR 0.72; 95% CI 0.66–0.78), favouring Semaglutide over placebo. These effects are particularly relevant in women with PCOS, prediabetes, and central obesity.

Semaglutide: Gynaecology-Relevant Evidence Across the Female Life Course

Real world evidence demonstrates that Semaglutide is associated with clinically meaningful reductions in body weight, body mass index, adiposity, and metabolic parameters in women with obesity across reproductive and postmenopausal life stages.

Polycystic Ovary Syndrome and Reproductive Outcomes

In women with polycystic ovary syndrome who were unresponsive to lifestyle intervention, Semaglutide (0.5 mg once weekly) was associated with ≥5% weight loss in approximately 80% of patients within three months, with menstrual cycle normalization reported in nearly 80% of responders.

Combination Therapy with Metformin

Comparative studies indicate that Semaglutide plus metformin resulted in greater reductions in body weight (−6.1 ± 3.3 kg vs −2.3 ± 4.3 kg), body mass index, waist-to-hip ratio, fasting insulin, fasting glucose, and insulin resistance (Homeostatic Model Assessment for Insulin Resistance) compared with metformin alone (all $p<0.01$). Combination therapy was also associated with higher menstrual normalization rates (62.5% vs 37.5%) and a more than two-fold increase in natural conception rates (35% vs 15%, $p<0.05$).

Durability of Weight Loss

Long-term follow-up data show that although partial weight regain occurs after Semaglutide withdrawal, over 80% of women remained below baseline body weight at two years when continued on background metformin, supporting sustained benefit in chronic weight management.

Menopausal Transition

Across menopausal stages, Semaglutide demonstrated consistent efficacy, with mean weight reductions of approximately 18.0% in premenopausal, 17.7% in perimenopausal, and 16.4% in postmenopausal women, accompanied by improvements in cardiometabolic risk markers. These findings support the relevance of anti-obesity pharmacotherapy throughout the female life course, including during the menopausal transition when cardiometabolic risk is heightened.

Cardiometabolic and Cardiovascular Outcomes

Pooled STEP analyses show that Semaglutide treatment resulted in reductions of up to 16.3 cm in waist circumference, 10.9 mmHg in systolic blood pressure, 5.3 mmHg in diastolic blood pressure, 22% in triglycerides, 1.9% in HbA1c, and 63.4% in C-reactive protein, indicating meaningful improvements in visceral adiposity, inflammation, blood pressure, and lipid metabolism.

In the SELECT trial, which enrolled 17,605 participants across 41 countries, including 492 from India, Semaglutide significantly reduced major adverse cardiovascular events in individuals with overweight or obesity without diabetes, establishing cardiovascular benefit beyond glycaemic control.

MASH (Metabolic Dysfunction–Associated Steatohepatitis)

Beyond cardiovascular risk reduction, emerging evidence suggests that Semaglutide confers broader multisystem metabolic benefits. In the phase 3 ESSENCE program, Semaglutide achieved significantly higher rates of steatohepatitis resolution without worsening of fibrosis (approximately 63% vs 34% with placebo), along with meaningful improvement in liver fibrosis. These findings extend the metabolic benefits of Semaglutide to obesity-related liver disease and are particularly relevant for women with central obesity and insulin resistance.

Safety and Clinical Experience

Across the STEP trials, Semaglutide was generally well tolerated. Gastrointestinal adverse events were the most common, typically mild to moderate and transient. No increased risk of malignancy or cardiovascular events was observed. Semaglutide is supported by data from over 60,000 participants, with follow-up of up to four years, demonstrating sustained efficacy and a well-established safety profile.

Dosing and Titration:

Initiation: 0.25 mg once weekly for 4 weeks

Dose escalation:

- 0.5 mg once weekly for 4 weeks
- 1.0 mg once weekly for 4 weeks
- 1.7 mg once weekly for 4 weeks

Maintenance dose: 2.4 mg once weekly (achieved by week 16)

Gradual titration strategy reduces gastrointestinal adverse effects and improves long-term treatment persistence. Dose escalation may be delayed in patients who experience significant gastrointestinal symptoms, supporting individualized therapy.

Tirzepatide (Dual GIP/GLP-1 Receptor Agonist)

Tirzepatide is a dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 receptor agonist that has demonstrated robust weight-loss efficacy in individuals with obesity, with and without type 2 diabetes. In the SURMOUNT clinical trial program, tirzepatide produced dose-dependent mean weight reductions of approximately 15–21% over 72 weeks, with the highest efficacy observed at the 15 mg dose.

A comprehensive systematic review and meta-analysis including SURMOUNT and SURPASS trials reported mean body weight reductions of approximately 8.2 kg, 10.7 kg, and 11.5 kg with the 5 mg, 10 mg, and 15 mg doses, respectively, compared with placebo. In individuals without diabetes, weight loss was more pronounced, reaching up to ~17.9 kg at the highest dose.

Tirzepatide has also demonstrated significant improvements in glycemic parameters, insulin resistance, waist circumference, and lipid profiles, supporting its role in addressing the metabolic drivers of obesity. Gastrointestinal adverse events remain the most common and are dose-dependent, with overall tolerability comparable to other incretin-based therapies.

While direct head-to-head randomized comparisons are limited, available data suggest that Tirzepatide achieves greater mean weight loss at higher doses compared with GLP-1 receptor agonists alone; however, differences in trial design, populations, duration, and background therapies warrant cautious interpretation. Long-term cardiovascular outcome data and gynaecology-specific studies (e.g., PCOS, fertility outcomes) are still emerging.

Standard Dosage Schedule for Diabetes and Weight Loss

- **Initiation:** 2.5 mg once weekly for the first four weeks
- **Maintenance (after 4 weeks):** 5 mg once weekly
- **Dose escalation:** Increase by 2.5 mg every four weeks
- **Maximum dose:** 15 mg once weekly

Evidence Landscape for GLP-1 and Dual GIP/GLP-1 Receptor Agonists

Semaglutide has more than 10 years of clinical exposure data and over 26,000 participants studied in obesity trials, whereas Tirzepatide has approximately four years of exposure data and around 5,800 participants studied in obesity trials. Semaglutide has 94 % structural homology to native human GLP-1, while Tirzepatide is a synthetic dual GIP and GLP-1RA. Mean weight loss with Semaglutide reaches up to 18 %, with higher investigational doses achieving approximately 21 %, compared with around 21 % mean weight loss reported with Tirzepatide in the SURMOUNT trials.

Liraglutide

Liraglutide, a once-daily glucagon-like peptide-1 receptor agonist administered at a dose of 3.0 mg, is an established pharmacological option for chronic weight management in adults with obesity, including individuals without diabetes. Randomized controlled trials and meta-analyses have demonstrated that liraglutide is associated with clinically meaningful reductions in body weight, body mass index, and waist circumference, along with improvements in cardiometabolic risk factors such as glycaemic parameters and lipid profiles.

As a class, glucagon-like peptide-1 receptor agonists, including liraglutide, have been shown to improve appetite regulation, reduce caloric intake, and enhance

metabolic control. Liraglutide's daily dosing regimen may be suitable for selected patients based on clinical context, tolerability, and patient preference. Dosing schedules, titration protocols, contraindications, and management of missed doses vary across glucagon-like peptide-1 receptor agonists, and clinicians should adhere strictly to individual product labelling and approved prescribing information when initiating and maintaining therapy.

Standard Dosage Schedule for Adult Patients:

- **Starting dose:** 0.6 mg injected subcutaneously once daily for one week (for GI tolerability; not effective for glycaemic control)
- **Dose escalation:** Increase to 1.2 mg once daily after one week
- **Maximum recommended dose:** 1.8 mg once daily after at least one week at 1.2 mg (if additional glycaemic control is required)

Emerging Triple-Hormone Receptor Agonists

Triple-hormone receptor agonists that simultaneously target Glucagon-like peptide-1, Glucose-dependent insulinotropic polypeptide, and glucagon receptors represent a novel and evolving therapeutic approach in obesity management. These agents are designed to combine appetite suppression with potential increases in energy expenditure and enhanced metabolic effects.

In a phase 2 randomized controlled trial, Retatrutide demonstrated substantial and dose-dependent weight loss, with mean reductions exceeding 20 percent at higher doses over approximately 48 weeks, alongside improvements in cardiometabolic parameters. These findings suggest a potentially greater magnitude of weight reduction compared with currently available incretin-based therapies.

However, Triple-hormone receptor agonists remain investigational. Long-term safety data, durability of effect, and reproductive- and gynaecology-specific outcomes are still limited, and these agents are not currently included in national or international clinical practice guidelines. Their role in routine obesity management, including within gynaecologic practice, will depend on further phase 3 trials, long-term outcome data, and regulatory approvals.

Recommendations From Indian and International Guidelines on GLP-1 Receptor Agonists in Women

Federation of Obstetric and Gynaecological Societies of India (FOGSI)

At AICOG 2026, FOGSI released Good Clinical Practice Recommendations on the Management of Obesity in Women, recognizing obesity as a chronic disease and recommending escalation to pharmacotherapy, including GLP-1RA (Semaglutide, Liraglutide) and dual GIP/GLP-1 receptor agonists (Tirzepatide), when lifestyle measures are inadequate. Treatment selection is guided by metabolic profile, reproductive stage, and tolerability. Semaglutide is highlighted for its robust and sustained weight-loss efficacy, demonstrated cardiometabolic benefits, and breadth of evidence across the female life course, including PCOS, reproductive age, perimenopause, and post menopause, along with a longer safety profile

Indian Society for Assisted Reproduction (ISAR)

ISAR guidelines recognize obesity as a chronic disease in women and recommend early identification using body mass index and waist circumference. When lifestyle measures are insufficient, pharmacotherapy including orlistat and GLP-1 receptor agonists is recommended as part of comprehensive obesity management in gynaecological and fertility practice.

International Guidance

Guidance from the International Menopause Society (IMS) and the American Society for Reproductive Medicine (ASRM), supported by endocrine recommendations from the American Association of Clinical Endocrinology (AACE), endorses GLP-1 receptor agonists as adjuncts to lifestyle intervention for obesity management in menopausal women and in adults with polycystic ovary syndrome.

Where These Therapies Fit Best in Gynaecology Practice

PCOS with Obesity and Insulin Resistance

In women with PCOS, meaningful weight reduction of 5 to 15 % is associated with improvements in insulin resistance, hyperandrogenism, menstrual regularity, and ovulatory function. When lifestyle therapy is inadequate, incretin based therapies may be considered as adjuncts within a structured metabolic management plan.

Infertility and Assisted Reproductive Care

Obesity negatively affects ovulation induction, oocyte quality, implantation, and live birth rates. Early weight optimization using lifestyle measures, with pharmacotherapy when indicated, can improve metabolic readiness prior to fertility treatment. Treatment planning should align with reproductive intentions and preconception timelines.

Perimenopause and Post menopause

Menopause is characterized by increased visceral adiposity and cardiometabolic risk. In women with BMI ≥27 kg/m^2 with comorbidities or ≥30 kg/m^2, pharmacologic

therapy may be integrated with lifestyle interventions to support sustained weight reduction and metabolic health. Resistance training and adequate protein intake remain essential to preserve lean mass.

Cost and Access Considerations in the Indian Setting

In India, where most patients pay out of pocket, treatment selection is influenced not only by efficacy and safety but also by affordability. Depending on the incretin-based therapy selected, the approximate monthly cost during initiation (early dose-escalation phase) may range from around ₹8,500 for Semaglutide to approximately ₹12,500 for Tirzepatide (as of 31 January 2026).

Safety, Adverse Effects, and Special Precautions

The most commonly reported adverse effects of GLP-1RA are gastrointestinal and include nausea, vomiting, diarrhoea or constipation, and dyspepsia. These effects are typically dose-related and improve with slow and careful titration.

GLP-1RA are contraindicated in individuals with a personal or family history of medullary thyroid carcinoma or multiple endocrine neoplasia type 2, and in those with known hypersensitivity to these agents. Caution is advised in women with a history of pancreatitis, gallbladder disease, or significant gastrointestinal motility disorders such as gastroparesis.

In women with diabetes, baseline assessment of glycaemic status, renal function, and diabetic retinopathy is recommended. Rapid improvement in glycaemic control has been associated with transient worsening of diabetic retinopathy, necessitating appropriate monitoring in at-risk individuals.

Due to delayed gastric emptying and potential aspiration risk, guidance recommends withholding long-acting incretin-based agents prior to procedures requiring anaesthesia or sedation, in alignment with current institutional and anaesthesia society recommendations.

Pregnancy, Lactation, and Contraception: Key Considerations

Incretin-based anti-obesity medications are not recommended during pregnancy and should be discontinued prior to planned conception. A washout period of approximately two months is commonly recommended due to its long elimination half-life. If pregnancy occurs inadvertently during therapy, the medication should be discontinued and routine obstetric care provided with appropriate monitoring.

Use during lactation is not recommended due to limited human safety data. Gynaecologists should ensure effective contraception during treatment and provide clear counselling on reproductive planning when initiating therapy in women of childbearing potential.

Conclusion

Obesity is a chronic, progressive condition in women that adversely affects PCOS, fertility, pregnancy outcomes, menopause, cardiometabolic health, and quality of life, often at lower BMI thresholds in Asian Indians. A sustained 5–10% weight reduction is associated with meaningful gynaecologic benefits, including improved insulin resistance, menstrual regularity, ovulation, fertility outcomes, and reduced cardiometabolic risk. Lifestyle modification remains foundational but is frequently insufficient due to biological adaptations and menopause-related metabolic changes, supporting early use of adjunct pharmacotherapy. GLP-1RA effectively address appetite regulation and metabolic dysfunction. Semaglutide produces durable 15–18% weight loss with extensive evidence across PCOS, reproductive age, and menopause, while Tirzepatide demonstrates greater mean weight loss (up to ~21%) in obesity trials, with emerging long-term and gyne-specific data. Liraglutide remains an established daily GLP-1 option. Indian and international guidelines (FOGSI GCPR, ISAR, IMS, ASRM, AACE) recognize obesity as a chronic disease and recommends incretin-based therapies when lifestyle measures are inadequate, reinforcing their expanding role in routine gynaecologic and fertility practice.

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Obesity and Modern Obstetrics: Rethinking Risks and Care

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Introduction

Obesity in recent years has emerged as a Global Epidemic. It is one of the most important determinants of maternal and perinatal outcomes in modern obstetrics. The impact is seen from preconception to antenatal complications, intrapartum challenges, postpartum recovery, and longterm maternal and child health issues.

The prevalence of obesity is rising in reproductive age due to global trends in nutrition transition, urbanization, sedentary lifestyles, and metabolic disease.[1] In India, the coexistence of undernutrition and obesity creates a unique "double burden" scenario.

Up to 30% of pregnant women in high-income countries are classified as obese, and approximately 40% exceed the recommended gestational weight gain (GWG).[2] Maternal obesity and excessive GWG can lead to long-term effects on offspring adiposity, cardiovascular, and respiratory health.[3]

Maternal obesity affects hormonal balance, cardiovascular function, metabolism, and immune responses. It increases the risk of gestational diabetes mellitus (GDM), hypertensive disorders, preeclampsia, and non-alcoholic fatty liver disease (NAFLD), all of which contribute to adverse maternal and neonatal outcomes. It is linked to fetal macrosomia, preterm birth, congenital anomalies, and stillbirth.[3,4] It also impairs placental function, disrupts fetal growth, and predisposes children to long-term metabolic and cardiovascular diseases. It contributes to postpartum complications, difficulty in breastfeeding, and childhood obesity.

In addition, maternal obesity may alter breast milk composition, including macronutrient content and microbiota, influencing infant feeding behaviour and early metabolic regulation. Maternal obesity with placental inflammation, gut microbiome disruption, and oxidative stress, which may further compromise pregnancy outcomes.[5]

In pregnancy with maternal obesity, early intervention is crucial— timing, mode of delivery, and postpartum recovery along with clinical decisions and public health strategies to improve outcomes for mothers and their children.[6]

Epidemiology of Obesity in Pregnancy

Obesity among women of reproductive age has increased over the past three decades. WHO estimates indicate that more than 15% of women aged 20–49 years are obese, In the United States, over 30% of pregnant women in pregnancy have a BMI ≥30 kg/m^2, 8%Class III obesity (BMI ≥40 kg/m^2). India has 24% overweight and obese women aged 15–49 years with Urban prevalence: 33% and Rural prevalence: 19%. (NFHS V)

Pathophysiology of Obesity During Pregnancy

Maternal obesity triggers complex physiological and molecular alterations which include chronic low-grade inflammation, insulin resistance, oxidative stress, hormonal imbalances, placental dysfunction, and epigenetic modifications.[7]

Insulin Resistance and Glucose Dysregulation

Obesity is characterized by baseline insulin resistance, which leads to increased maternal glucose levels, higher fetal glucose exposure, enhanced fetal insulin secretion, and increased risk of macrosomia and neonatal hypoglycemia.

Chronic LowGrade Inflammation

Systemic inflammation due to excessive adipose tissue secrete pro-inflammatory cytokines such as TNF-α, IL-6, and CRP, which promote oxidative stress, and impair endothelial nitric oxide (NO) production—causing preeclampsia and gestational diabetes mellitus (GDM).[7,8,9]

Adipose tissue also modulates metabolic homeostasis contributing to excessive gestational weight gain.[10]

At the placental level, maternal obesity leads to increased expression of glucose (GLUT1) and lipid (FATP) transporters, enhancing nutrient transfer and predisposing to fetal overgrowth and macrosomia.[11] Elevated reactive oxygen species (ROS) impair trophoblast function and angiogenesis, increasing the risk of intrauterine growth restriction (IUGR) and stillbirth.[12]

Epigenetic modifications in the placenta and fetal tissues can program metabolic dysfunctions, increasing offspring risk for obesity and type 2 diabetes later in life.[13]

Obesity also impacts labor physiology. Reduced expression of oxytocin receptors and connexin 43 in myometrial cells weakens uterine contractility, increasing the likelihood of labor dystocia and cesarean delivery.[14]

Postpartum, obesity-related inflammation impairs wound healing via elevated matrix metalloproteinase (MMP) activity, while hormonal imbalances and insulin resistance hinder prolactin signalling and delay lactation.[15]

Cardiovascular and Respiratory Changes

Obesity increases cardiac output, blood volume, left ventricular mass, and risk of obstructive sleep apnea. These changes complicate anaesthesia, intrapartum monitoring, and postpartum recovery.

Placental Adaptations

Placental morphology and function are altered in obesity, including increased placental weight, altered villous architecture, dysregulated nutrient transporters, and increased inflammatory infiltrates. These changes contribute to both macrosomia and fetal growth restriction.[16]

Chronic Inflammation and Placental Dysfunction	Obesity-related adipose tissue secrets pro-inflammatory cytokines tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) → impair placental angiogenesis → preeclampsia → fetal growth restriction &preterm birth. Oxidative stress, in placenta → damages trophoblasts→ reduces vascularization → risk of fetal hypoxia and IUGR Glutathione peroxidase, superoxide dismutase, and catalase → oxidative stress and impair placental development and l angiogenesis→ adverse pregnancy outcomes.
Insulin resistance and GDM	Obesity heightens insulin resistance → gestational diabetes mellitus (GDM) and fetal macrosomia Dyslipidemia—elevated TGs, FFAs, and LDL/VLDL—disrupt glucose metabolism and promote inflammation. Placental lipid accumulation exacerbates oxidative stress and impairs fetal nutrient transfer → increases offspring risk of obesity, NAFLD, and metabolic syndrome
Renin-Angiotensin system dysregulation	Angiotensin II (Ang II),elevated in obese pregnancies → vasoconstriction, oxidative stress, and pro-inflammatory cytokine release. → impair placental perfusion → Preeclampsia Placental ischemia→ Release of bioactive factors including AT1 receptor agonistic autoantibodies→ sensitize the vasculature to Ang II, → vasoconstriction and inflammation
Cardiovascular and Hemdynamic effects	↑Cardiac output→ blood volume expansion and systemic vasodilation → hemodynamic overload and vascular dysfunction→Impaired myocardial efficiency→subclinical cardiac dysfunction ↓endothelial reactivity due to chronic low-grade inflammation and oxidative stress→ ↓nitric oxide bioavailability→Preeclampsia, ↓ placental perfusion. promote a prothrombotic vascular phenotype, ↑ von Willebrand factor→ thromboembolic events.
Microbiome Dysbiosis and Inflammation	Gut microbiota dysbiosis ↓microbial diversity↑ Firmicutes-to-Bacteroidetes ratio→ insulin resistance, lipid imbalance, and systemic inflammation→ GDM and preeclampsia. Maternal microbiota →fetal colonization→ programming obesity and immune dysregulation in offspring

Figure 1. Depicting - Pathophysiology Maternal Obesity and Pregnancy Complications

Prevalence and Trends

In India few states show a steady rise in maternal obesity- Kerala, Telangana, Tamil Nadu, Delhi, and Punjab. These states also report higher rates of GDM and hypertensive disorders.

Maternal–Fetal Outcomes in India

Indian data of Obesity during pregnancy :

- Higher GDM rates (18–25%)
- Increased preeclampsia risk (10–14%)
- Cesarean section rates 45–60%
- Macrosomia rates 8–12%
- Increased NICU admissions

Classification of Obesity - Healthcare providers classify obesity using the body mass index (BMI), a calculation based on a person's body weight in kilograms divided by their height in meters squared.

BMI scores are broken down into different categories:

Table 1. Classification of Obesity (WHO and Asian Cutoffs)

BMI	Category
less than 18.5	Underweight
between 18.5 and 24.9	Healthy or normal weight
between 25 and 29.9	Overweight
BMI of 30 or higher	Obesity

There are three classes of obesity:

- **Class 1** obesity is a BMI score between 30 and 34.9.
- **Class 2** is a score between 35 and 39.9.
- **Class 3** is a BMI of 40 or higher

Antenatal Care for Women With Obesity

There is need of customized care and targeted interventions in antenatal care to optimize fetal outcomes and reduce long term maternal and neonatal complications.

Booking Visit

At first visit accurate BMI measurement, early risk stratification, baseline investigations, early GDM screening, blood pressure monitoring, and counselling on diet and physical activity should be done.

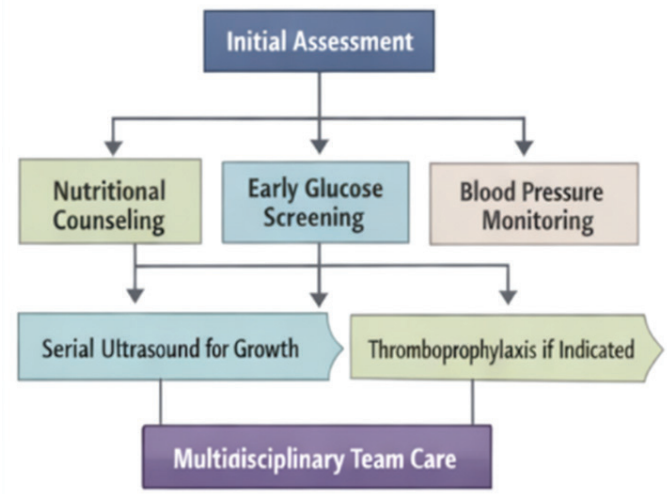

Figure 2. Care plan for OWO (Overweight & Obese) Pregnancy

Table 2. WeightGain Recommendations -Depending on pre pregnancy BMI

BMI 30–34.9	5–9 kg
BMI ≥35	Minimal weight gain or weight maintenance

Screening for GDM It is recommended to do early screening at booking and repeat screening at 24–28 weeks in OWO (Overweight Obese woman)

Blood Pressure and Preeclampsia Prevention -Lowdose aspirin Prophylaxis may be considered for obese women with multiple risk factors.

Fetal Surveillance -Depending on BMI and comorbidities: Fetal Growth scans at 28, 32, and 36 weeks as well as additional scans depending on other fetal risk conditions.

Lifestyle Modifications for Weight Management

Significant lifestyle counselling about proper diet, exercise, and weight control interventions are necessary to improve compliance and outcomes for both the mother and the fetus before and during pregnancy.

Nutritional Counselling - Focus on balanced diet, controlled carbohydrate intake, adequate protein, and culturally appropriate meal planning.

Physical Activity- Recommended: 150 minutes/week of moderate exercise or Walking, prenatal yoga, and swimming.

Multidisciplinary Care - Pregnancy-related obesity can be effectively managed with the help of multidisciplinary preventive care specially for women with BMI ≥40 kg/ m^2 that involve a dietician, obstetrician, anaesthetists, dietitians, and endocrinologists and physical activity specialist or physiotherapist.

Intrapartum Care - Timing and Mode of Delivery

It is important to consider the mother's BMI, her history of previous deliveries, and the well-being of the unborn child when determining the safest delivery method to improve outcomes and reduce risks associated with maternal obesity during childbirth; proactive planning, patient education, and individualized assessment are essential and aim to minimize challenges for both the mother and newborn while carefully balancing risks and benefits.

According to the FIGO recommendations maternal obesity represents a significant consideration when planning delivery, often necessitating individualized risk assessment for cesarean indications.

Place of Birth Obese women should deliver in facilities with 24/7 anaesthesia, advanced neonatal care, and operating theatre availability.

Labour Management considerations include early epidural placement, continuous fetal monitoring, preparedness for operative delivery, and active management of labour.

Cesarean Delivery Considerations - Women with a BMI ≥ 40 and other risk factors may be given consideration for a planned cesarean delivery. Important points include appropriate equipment, experienced anaesthetist, longer incision, deeper tissue planes, and higher risk of wound complications.

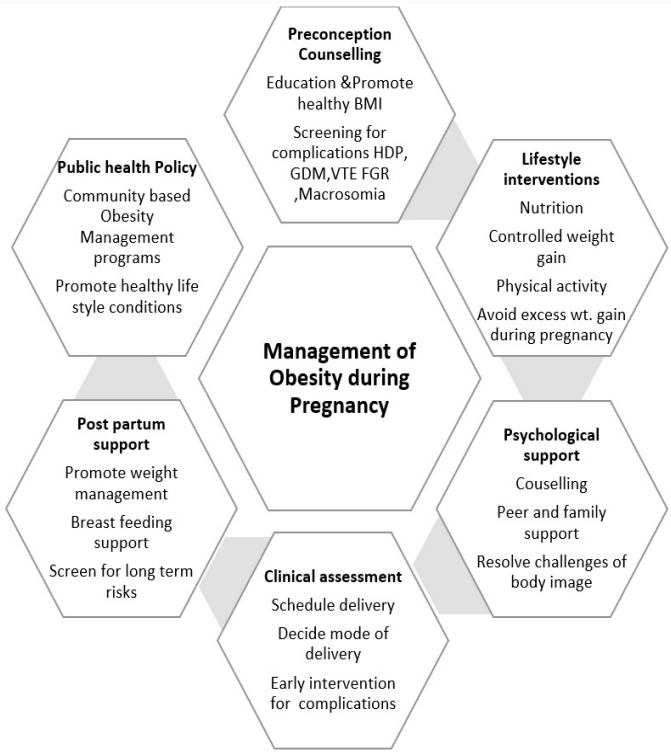

Figure 3. Comprehensive approach to maternal obesity management. This figure shows main strategies for managing maternal obesity at preconception, pregnancy, delivery, and postpartum care stages

Mode of Delivery in Obese Women

Maternal obesity is often associated with elevated cesarean delivery rates, 50–60% in obese women compared to 20–30% in those with a normal BMI. Cesarean sections are indicated due to fetal macrosomia, labor dystocia, comorbidities, and failed inductions.

Intraoperative challenges like surgical exposure difficulties, extended operative times, and anesthesia-related complications - hypoxia and difficult intubation are higher in obese patients. While evaluating the patients for vaginal birth after cesarean (VBAC) decision should be individualized as repeat cesareans in obese women carry a greater risk of uterine rupture due to increased intra-abdominal pressure and inflammatory responses.

The mechanical and metabolic effects of obesity impair uterine contractility and may lead to labor complications. Labor dystocia because of increased fetal size, decreased uterine contractility, and excess soft tissue in the birth canal fetal macrosomia, Shoulder dystocia, birth trauma, and a higher risk of emergency cesarean section or an operative vaginal delivery are major delivery issues. Hence careful fetal monitoring during labor and early intervention should be done.

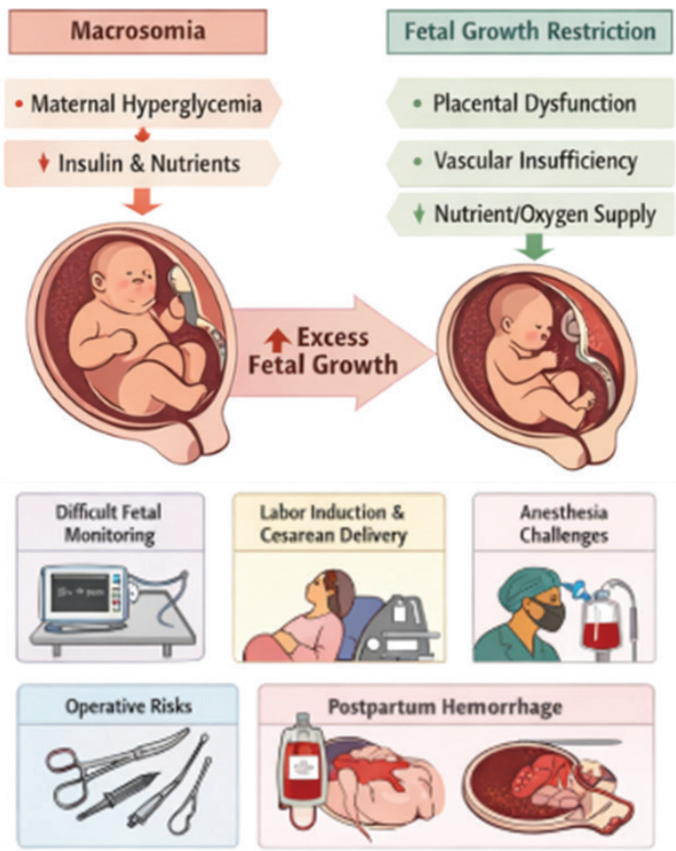

Figure 4 & 5. Fetal Growth Outcomes in OWO pregnancy and Intrapartum and postpartum risks

Anesthesia and Surgical Considerations

Anesthesia challenges include mainly perioperative risks. Neuraxial anesthesia is preferred but technically more difficult due to fat deposition and distortion.General anesthesia carries risks of failed intubation, aspiration, and respiratory compromise. Multimodal analgesia is recommended for pain control while minimizing respiratory depression.

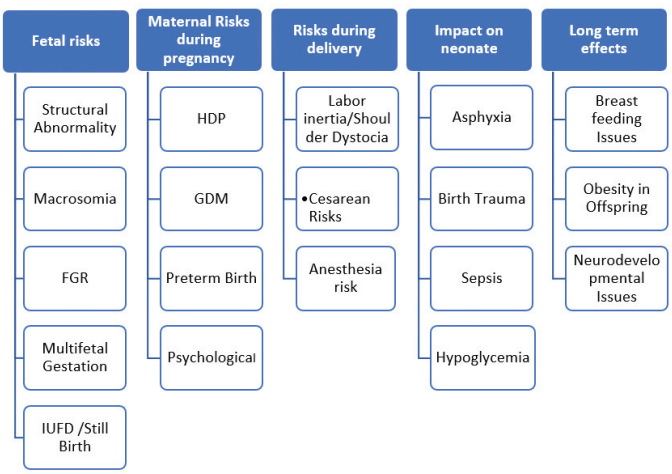

Figure 6. Maternal, Fetal ,Neonatal and long term risks of Obesity in pregnancy In

Surgical risks - Prolonged operative time, greater blood loss, and elevated infection rates, impaired healing. Obese women are more susceptible to postpartum hemorrhage and thromboembolic events. Standard preventive strategies such as early mobilization and thromboprophylaxis are essential.

Prophylactic strategies like Preoperative weight-based antibiotic dosing, negative-pressure wound therapy, and subcutaneous drain placement should be considered.

Fetal Outcomes

Obesity in mothers can lead to stillbirth, abnormal growth(FGR) and fetal structural anomalies/congenital defects and macrosomia which increases the risk of birth trauma and shoulder dystocia. Obesity is a risk factor for both spontaneous and medically indicated preterm birth more so in pregnancies complicated by gestational diabetes, preeclampsia, and chronic inflammation.

Neonatal Health at Birth and Long-Term Implications

Maternal obesity is highly associated with several short and long-term neonatal issues. The concept of "fetal programming" suggests that intrauterine exposure to maternal obesity may predispose offspring to obesity, metabolic syndrome, and cardiovascular disease later in life [3] Metabolic programming also increases the risk of perinatal ischemic stroke (PIS), a severe neurological complication resulting from reduced cerebral blood flow or vascular occlusion in the perinatal period. Maternal obesity contributes to fetal vascular dysfunction through multiple mechanisms, including endothelial inflammation, hypercoagulability, and placental hypoxia.[17] Obese pregnancies are associated with increased fibrin deposition and thrombotic activity, predisposing the fetus to cerebrovascular events. Maternal hyperlipidemia and insulin resistance result in altering placental perfusion, increasing the risk of fetal hypoxic–ischemic injury.

Neonatal Complications include shoulder dystocia, birth trauma, neonatal hypoglycemia, respiratory distress, and NICU admission.

Postpartum Recovery Issues

The postpartum period is a critical time for maternal health, and recovery from obesity is especially challenging for obese mothers.

Postpartum Support for Weight Management and Mental Health

Postpartum issues like delayed wound healing, infections, postoperative complications, and breastfeeding difficulties, are associated with maternal obesity that may cause psychological distress, anxiety and postpartum depression and chronic physical discomfort, negative body image and

social stigma, Postpartum lifestyle interventions, such as physical activity and nutritional counselling, can reduce these risks. New treatment approaches like omega-3 fatty acids, prebiotics, and probiotic supplements benefits in postpartum mental health care by lowering systemic inflammation and elevating mood. Peer support groups, cognitive behavioral therapy, and mindfulness-based stress reduction are effective interventions.

Recurrent spontaneous abortion (RSA)

Obese women are more likely to experience pregnancy loss due to dysregulated inflammatory responses, impaired endometrial receptivity, and metabolic disturbances ,insulin resistance and altered glucose metabolism, polycystic ovary syndrome (PCOS) and GDM. Insufficient progesterone production in obese women has been associated with defective decidualization and an increased risk of early pregnancy loss.[16]

Breastfeeding Challenges

Obese women may face physical, physiological, and psychological Breastfeeding difficulty leading to delayed lactation initiation, difficulties with positioning, difficulties with latching, and a lack of confidence in one's ability to breastfeed.

Public Health Implications and Clinical Recommendations

Medical professionals should emphasize the role of prevention, lifestyle modification, personalized prenatal care to decide the optimal timing and method of delivery. Clinical guidelines and effective public health strategies can improve the health of mothers and their offspring by reducing the negative effects of maternal obesity.[18]

Preventive Strategies and Recommendations

Single most important strategy to lessen unfavorable outcomes is the prevention of obesity. To implement healthy lifestyle interventions both before and during pregnancy.[19]

Diet, exercise, and behavioural support for weight management should be the focus of public health interventions for expectant mothers.

Conclusion

- Obesity in pregnancy represents a complex metabolic state characterized by chronic lowgrade inflammation, insulin resistance, endothelial dysfunction, and altered placental signalling. These pathophysiological changes lead to the increased risk of gestational diabetes, hypertensive disorders, thromboembolism, fetal growth abnormalities, and operative delivery.
- Maternal obesity significantly impacts pregnancy outcomes, including birth timing and method, maternal recovery, and neonatal health.
- Maternal obesity raises the risk of neonatal problems like macrosomia, increased neonatal intensive care unit admissions, and long-term health issues like metabolic syndrome and juvenile obesity.
- Obesity also affects uterine contractility and the progression of labor. increasing the risk of cesarean delivery and extending labor.
- Significantly higher cesarean delivery rate (50–60% vs. 20–30% in normal BMI pregnancies.
- Fetal macrosomia affects 15–25% of obese pregnancies along with labor dystocia, decreased uterine contractility makes vaginal delivery risky.[20]
- Obese women are at a two-fold increased risk of postpartum hemorrhage and a three-fold increased risk of surgical site infections.
- Protocolized care bundles that include negative-pressure wound therapy and preoperative chlorhexidine bathing may lower infection rates by 40%.[21]
- Obese women have lower breastfeeding continuation rates at 6 months (32% vs. 54%) and delayed lactogenesis onset by 72 h in only 68% vs. 90% in women with normal BMI.[22]
- A multidisciplinary approach involving obstetricians, anesthesiologists, and maternal–fetal medicine specialists is vital to optimizing outcomes.
- Preoperative planning and postoperative surveillance can reduce maternal morbidity and improve neonatal health.
- The concept of "fetal programming" suggests that intrauterine exposure to maternal obesity may predispose offspring to obesity, metabolic syndrome, and cardiovascular disease later in life.[4]
- Early counselling for obese pregnant women with aim to optimize outcomes for both mother and child and contribute to combating the global obesity crisis.
- Single most important strategy to lessen unfavorable outcomes is the prevention of obesity.

Table 3. Summary of Overweight/Obese Women (OWO) Pregnancy

Physiologic consequences	Emerging Risks	Challenges in Prenatal Care	Intervention strategies
Metabolic and inflammatory pathways Altered laboratory tests "fetal programming" (intrauterine exposure to maternal obesity may predispose offspring to obesity, metabolic syndrome, and cardiovascular disease later in life)	GDM Preeclampsia HDP NAFLD Fetal complications Macrosomia FGR Malformations Preterm birth Still birth Prolonged NICU Caesarean risk Delayed Post partum recovery Risk of infections Breast feeding challenges	Early counselling Optimize maternal weight gain during pregnancy Challenges in Ultrasound measurements	Prioritize strategies for early counselling Optimize maternal fetal outcomes Reduce long term risks of OWO pregnancy Combating global obesity crisis

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Chronic Pelvic Pain in Endometriosis and Adenomyosis: Bridging Pathology and Neurobiology

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Introduction

Chronic pelvic pain (CPP) represents one of the most complex and challenging conditions in gynecological practice. It is defined as intermittent or continuous pelvic pain lasting at least six months, of sufficient severity to cause functional impairment or require medical intervention.[1] CPP significantly affects physical health, emotional wellbeing, sexual function, and socioeconomic participation, resulting in substantial individual and societal burden.[2] Among reproductive-aged women, endometriosis and adenomyosis are the leading gynecological causes and frequently coexist, sharing overlapping pathological and neurobiological mechanisms that complicate diagnosis and management.[3]

Endometriosis affects approximately 10% of women of reproductive age globally[4] and up to 30–50% of women with chronic pelvic pain or infertility.[5] It is characterized by the presence of endometrial-like glands and stroma outside the uterine cavity, commonly involving the peritoneum, ovaries, uterosacral ligaments, and bowel.[6] Adenomyosis, defined by ectopic endometrial tissue within the myometrium, is increasingly recognized as a significant contributor to dysmenorrhea, abnormal uterine bleeding, and CPP.[7] Advances in imaging have demonstrated that adenomyosis coexists with endometriosis in up to one-third of affected patients, suggesting shared pathogenetic pathways.[8]

A defining clinical paradox in both conditions is the poor correlation between lesion burden and pain severity. Some women with minimal disease experience severe pain, whereas others with extensive disease remain relatively asymptomatic. This discrepancy underscores the importance of integrating peripheral pathology with contemporary understanding of neurobiological pain mechanisms, including sensitization and central nervous system modulation.

Pathophysiology: Peripheral Mechanisms

Endometriosis

Endometriosis is a chronic inflammatory condition driven by hormonal, immunological, and neurovascular processes. Although retrograde menstruation is widely accepted as a contributing factor, additional mechanisms—including immune dysfunction, altered apoptosis, stem cell activity, and genetic susceptibility—are essential for lesion establishment and persistence.[9]

Peripheral nociceptive activation in endometriosis is mediated by several key mechanisms:

Inflammatory activation

Endometriotic lesions secrete pro-inflammatory mediators including prostaglandin E2 (PGE2), interleukin-1β, interleukin-6, tumor necrosis factor-alpha, and chemokines. These mediators sensitize peripheral nociceptors, lowering pain thresholds and amplifying nociceptive signaling.[9]

Neuroangiogenesis

Increased expression of nerve growth factor (NGF) and vascular endothelial growth factor (VEGF) promotes simultaneous nerve fiber proliferation and angiogenesis. This process increases nociceptive innervation density within lesions, enhancing pain transmission.[9]

Cyclic bleeding and fibrosis

Repeated cyclic hemorrhage within ectopic tissue leads to oxidative stress, inflammation, and fibrotic adhesion formation. Adhesions restrict organ mobility and generate mechanical and inflammatory pain stimuli.[4]

These mechanisms create a self-sustaining inflammatory microenvironment that perpetuates peripheral nociception.

Adenomyosis

Adenomyosis involves invasion of endometrial tissue into the myometrium, disrupting normal uterine architecture and function. Several pathogenetic theories exist, including junctional zone disruption, tissue injury and repair, and metaplastic transformation.,[8]

Peripheral pain mechanisms in adenomyosis include:

Myometrial inflammation and hypercontractility

Elevated prostaglandin production and increased oxytocin receptor expression contribute to uterine hypercontractility, resulting in dysmenorrhea and pelvic pain.[5]

Local estrogen overproduction

Aromatase overexpression within adenomyotic tissue increases local estrogen synthesis, promoting lesion survival and inflammatory activity.[7]

Neurovascular proliferation

Increased nerve fiber density and neurotrophic signaling

within the myometrium contribute directly to pain generation and sensitization.[8]

These processes contribute to persistent nociceptive input and symptom chronicity.

Neurobiological Pain Mechanisms

Pain in endometriosis and adenomyosis extends beyond peripheral pathology and involves complex neurobiological alterations.

Peripheral Sensitization

Persistent inflammatory stimulation reduces nociceptor activation thresholds through upregulation of ion channels such as TRPV1 and voltage-gated sodium channels. This enhances afferent nerve excitability and amplifies pain transmission. Clinically, peripheral sensitization manifests as localized tenderness and reproducible pain on examination.

Central Sensitization

Ongoing peripheral input may induce neuroplastic changes in the spinal cord and brain, resulting in central sensitization. This phenomenon is characterized by hyperalgesia, allodynia, and pain persistence independent of peripheral disease activity.[9]

Central sensitization explains persistent pain despite adequate surgical treatment and the poor correlation between lesion severity and symptom intensity.

Neuroimaging of Pain Networks

Advanced neuroimaging techniques, including functional MRI and diffusion tensor imaging, have demonstrated altered connectivity and activation in key brain regions involved in pain processing, including the anterior cingulate cortex, insula, thalamus, and prefrontal cortex.[10] These changes reflect maladaptive neuroplasticity and impaired descending pain inhibition.

Neuroimaging findings support the concept that chronic pelvic pain represents a disorder of both peripheral pathology and central pain amplification. These insights may facilitate identification of patients at risk of persistent pain and guide multimodal treatment strategies.

Clinical Evaluation

Diagnosis requires integration of clinical history, physical examination, and imaging.

Clinical History

Pain characterization remains the cornerstone of evaluation.

Important features include:

- Dysmenorrhea, particularly progressive or severe
- Non-cyclical pelvic pain
- Deep dyspareunia
- Dyschezia or dysuria
- Infertility
- Heavy menstrual bleeding (common in adenomyosis)

Pain quality may include deep, cramping, stabbing, or neuropathic sensations. Radiation to the lower back or rectum may suggest deep infiltrating disease.[11]

Assessment of functional impact, psychological wellbeing, and previous treatment response is essential.

Physical Examination

Examination findings may include:

- Enlarged, globular uterus suggestive of adenomyosis
- Reduced uterine mobility due to adhesions
- Tender nodularity of uterosacral ligaments
- Adnexal tenderness or masses
- Pelvic floor muscle hypertonicity

Pain mapping can help differentiate visceral, neuropathic, and myofascial pain sources.

Imaging Evaluation

Transvaginal Ultrasound

Transvaginal ultrasound is the first-line imaging modality. It reliably detects ovarian endometriomas and characteristic features of adenomyosis, including myometrial heterogeneity, cysts, and junctional zone irregularity.[12]

Specialized ultrasound techniques can identify deep infiltrating endometriosis involving bowel, bladder, and uterosacral ligaments.

Magnetic Resonance Imaging

MRI provides superior soft tissue characterization and is particularly useful for deep infiltrating disease and adenomyosis mapping.[13]

Characteristic features include:

- T1 hyperintensity and T2 shading in endometriomas
- Thickened junctional zone in adenomyosis
- Fibrotic nodules in deep infiltrating disease

Imaging complements clinical evaluation but does not exclude disease when normal.

Diagnostic Strategy

Current guidelines recommend a stepwise approach:[14]

1. Comprehensive clinical history and examination
2. Exclusion of alternative diagnoses
3. Targeted imaging
4. Empirical medical therapy

5. Selective use of diagnostic laparoscopy

Modern consensus no longer requires surgical confirmation before initiating treatment in patients with typical symptoms.

Management

Medical Management

Hormonal suppression forms the cornerstone of treatment.

Combined oral contraceptives

Suppress ovulation and reduce inflammation and pain.

Progestogens (dienogest, LNG-IUS)

Induce lesion atrophy and reduce symptom severity.

GnRH agonists and antagonists

Suppress estrogen production and reduce lesion activity. Antagonists provide rapid and reversible suppression but require bone density monitoring.[13]

NSAIDs provide adjunctive symptom relief but do not modify disease progression.

Surgical Management

Surgical excision improves pain in selected patients, particularly those with deep infiltrating disease or infertility. However, recurrence remains common, and surgery does not address central sensitization.[13]

Hysterectomy remains definitive for adenomyosis in women who have completed childbearing.[15]

Multidisciplinary Management

Central sensitization requires a broader therapeutic approach, including:

- Pelvic floor physiotherapy
- Cognitive behavioral therapy
- Neuropathic pain medications
- Psychological support

Multidisciplinary care improves functional outcomes and quality of life.

Emerging Directions

Advances in biomarker discovery, neuroimaging, and artificial intelligence are transforming diagnosis and management.

Biomarkers including inflammatory cytokines, microRNAs, and molecular signatures may enable earlier non-invasive diagnosis and treatment monitoring.[16]

Artificial intelligence enhances imaging accuracy, improves lesion detection, and supports individualized treatment planning.Precision hormonal therapies and mechanism-based pain classification enable personalized treatment strategies.These advances reflect a paradigm shift from lesion-centric diagnosis to integrated, patient-centered care.

Prognosis

Pain outcomes vary widely. Some patients achieve sustained remission with medical therapy, while others experience persistent or recurrent symptoms. Central sensitization, comorbid pain syndromes, and psychological factors predict poorer outcomes.

Early diagnosis and multidisciplinary treatment improve long-term prognosis.

Conclusion

Chronic pelvic pain in endometriosis and adenomyosis represents a complex interaction between inflammatory pathology and neurobiological pain processing. Peripheral inflammation, neuroangiogenesis, and hormonal dysregulation initiate nociceptive signalling, while central sensitization perpetuates pain independent of lesion burden.

Effective management requires recognition that pain is not solely determined by anatomical disease but reflects dynamic interactions between peripheral pathology and central nervous system modulation.

Future advances in neuroimaging, biomarker discovery, and precision therapies promise to improve diagnostic accuracy, enable individualized treatment, and enhance long-term outcomes for affected women.

Chronic Pelvic Pain in Endometriosis & Adenomyosis- Protocol

STEP 1 — INITIAL ASSESSMENT

Clinical

- CPP ≥ 6 months (cyclic/non-cyclic)
- Dysmenorrhea, dyspareunia, dyschezia
- AUB (adenomyosis suspicion)
- Fertility desire documented

Investigations

- TVUS (first-line)
- MRI pelvis (deep infiltrating disease/adenomyosis)
- CBC ± CA-125 (adjunct only)

STEP 2 — FIRST-LINE MEDICAL THERAPY (3–6 MONTHS)

- NSAIDs
- Continuous COCs
- Progestins (dienogest / norethisterone)
- LNG-IUS (especially adenomyosis)

➞ Reassess (VAS score, QoL)

Improved → Continue & monitor
Persistent pain → Step 3

STEP 3 — ADVANCED HORMONAL SUPPRESSION

GnRH Antagonist Options

- **Elagolix** (150 mg OD / 200 mg BD) ± add-back
- **Relugolix combination therapy**
- **Linzagolix**

✓ Consider add-back therapy

✓ Monitor BMD if >6 months

✓ Limit duration per institutional protocol

➞ Reassess at 3–6 months

STEP 4 — MULTIDISCIPLINARY PAIN PATHWAY

- Pelvic floor physiotherapy
- Pain specialist (neuromodulators)
- CBT / psychological support
- Sexual health counseling
- Nutrition & lifestyle therapy

STEP 5 — SURGICAL MANAGEMENT

Indications

- Failed optimal medical therapy
- Endometrioma ≥3–4 cm
- Deep infiltrating disease
- Bowel/bladder involvement
- Infertility with anatomic distortion

Options

- Advanced laparoscopic excision
- Robotic deep infiltrating resection
- Adenomyomectomy (fertility desired)
- Hysterectomy (completed family, severe adenomyosis)
- Uterine artery embolization (selected adenomyosis)

STEP 6 — POST-SURGERY STRATEGY

- Hormonal suppression to reduce recurrence
- Pain rehabilitation
- Fertility planning
- Follow-up every 6–12 months

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The Endometriosis Fertility Index (EFI): Clinical Utility and Applications in Practice

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What is Endometriosis Fertility Index?

Endometriosis is a common disease that occurs in 6 to 10% of reproductive-age women. Approximately 25 to 50% of infertile women have endometriosis, and 30 to 50% of women with endometriosis are infertile. Basic pathology in endometriosis is extra uterine presence of endometrial tissue leading to chronic inflammation. Women with endometriosis may be asymptomatic. The extrinsic localization of endometrial tissue is responsible for chronic inflammation generating in anatomical pelvic modifications

The most widely used staging system of endometriosis is the revised American Fertility Society classification (r-AFS classification)[1]. The r-AFS classification is used to predict the recurrence potential of endometriosis after surgery. However, it has limited predictive ability for pregnancy after surgery.

The Endometriosis Fertility Index was described by the World Endometriosis Society (WES) in 2017 as being a robust and clinically valid score to predict fertility after surgery in patients with endometriosis[2]

Adamson[3] and Pasta proposed Endometriosis fertility index (EFI) scoring system in 2010 to predict the fecundity rate in women who underwent laparoscopic surgery for endometriosis that includes patient characteristics-age, duration of infertility, prior pregnancy, intra operative lesions description (American Society for Reproductive Medicine, American fertility society), functional postoperative score (least functional score). Adamson and Pasta[3,5] suggested that the r-AFS classification depends mainly on morphological descriptions, whereas Vercellini et al[6]. observed no association between the endometriosis stage or lesion type and lesion site and the cumulative probability of pregnancy.

The EFI score was calculated according to the EFI developed by Adamson and Pasta. It includes the following clinical and surgical factors : age, duration of infertility (years), pregnancy history, least-function (LF) score (including fallopian tubes, tubal fimbriae, and ovaries; LF score = the least score of the left side + the least score of the right side; if any ovary was absent, the LF score was obtained by doubling the LF score of the contralateral side), r-AFS score of the lesion, and total r-AFS score.

ENDOQUAL study–a prospective observational[4] bi-center cohort study conducted between 2012 and 2018–who underwent surgery for infertility were asked to complete a questionnaire collecting time and mode of conception. Of the 234 patients analyzed, 104 (44.4%) conceived postoperatively including 58 (55.8%) spontaneous pregnancies. An EFI of 0–4 for spontaneous pregnancies was associated with a lower cumulative pregnancy incidence compared to an EFI of 5–10 (52 versus 34 pregnancies respectively). An EFI of 0–4 was associated with a higher cumulative pregnancy rate for pregnancies obtained by artificial reproduction technology (ART), compared to an EFI of 5–10 (12 versus 6 pregnancies respectively). Fecundability decreased from 12 months for EFI 0–4 and from 24 months for EFI 5–10. This analysis suggests that patients with an unfavourable EFI (≤4) have more ART pregnancies than patients with a favourable EFI (≥5) and should be referred for ART shortly after surgery. Patients with a favourable EFI may attempt spontaneous pregnancy for 24 months before referral.

How is EFI score calculated?

EFI: The score is divided into historical and surgical factors:

- **History Factors (0–4 points):**

  Age (younger = higher score).

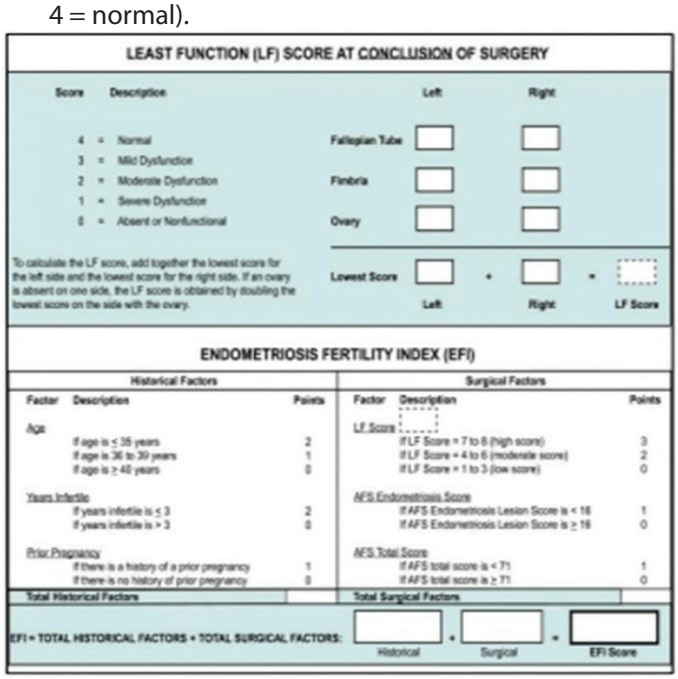
  Duration of infertility.

  Obstetric history (prior pregnancy = higher score).

- **Surgical Factors (0–6 points):**

  rASRM Score: Stage of disease.

  Least Function Score (LF): Assesses the functionality of the tubes, fimbriae, and ovaries (0 = nonfunctional, 4 = normal).



LEAST FUNCTION (LF) SCORE AT CONCLUSION OF SURGERY

Score	Description		Left	Right
4	= Normal	Fallopian Tube		
3	= Mild Dysfunction			
2	= Moderate Dysfunction	Fimbria		
1	= Severe Dysfunction			
0	= Absent or Nonfunctional	Ovary		

To calculate the LF score, add together the lowest score for the left side and the lowest score for the right side. If an ovary is absent on one side, the LF score is obtained by doubling the lowest score on the side with the ovary.

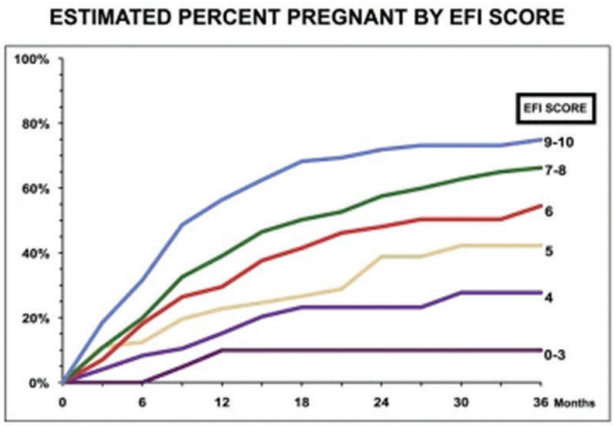
Lowest Score: Left + Right = LF Score

ENDOMETRIOSIS FERTILITY INDEX (EFI)

Historical Factors			Surgical Factors		
Factor	Description	Points	Factor	Description	Points
Age			LF Score		
	If age is ≤ 35 years	2		If LF Score = 7 to 8 (high score)	3
	If age is 36 to 39 years	1		If LF Score = 4 to 6 (moderate score)	2
	If age is ≥ 40 years	0		If LF Score = 1 to 3 (low score)	0
Years Infertile			AFS Endometriosis Score		
	If years infertile is ≤ 3	2		If AFS Endometriosis Lesion Score is < 16	1
	If years infertile is > 3	0		If AFS Endometriosis Lesion Score is ≥ 16	0
Prior Pregnancy			AFS Total Score		
	If there is a history of a prior pregnancy	1		If AFS total score is < 71	1
	If there is no history of prior pregnancy	0		If AFS total score is ≥ 71	0
Total Historical Factors			Total Surgical Factors		

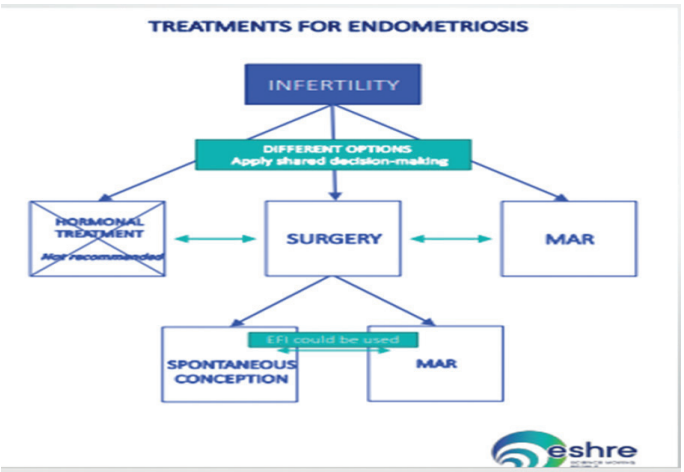
EFI = TOTAL HISTORICAL FACTORS + TOTAL SURGICAL FACTORS: Historical + Surgical = EFI Score

Endometriosis fertility index (EFI) surgery form.



EFI clinical use and applications

- A robust tool for predicting the pregnancy rate after surgery in women with endometriosis-related infertility, the EFI is also an important element to be considered in the therapeutic decision-making process.
- Women should be counselled of their chances of becoming pregnant after surgery.
- To identify patients that may benefit from ART after surgery, the Endometriosis Fertility Index (EFI) should be used as it is validated, reproducible and cost-effective. The results of other fertility investigations such as their partner's sperm analysis should be taken into account.



EFI could be used for treatment of endometriosis decision.

EFI Score Interpretation for predicting prognosis for 3-Year: The EFI is considered superior to the rASRM stage alone in predicting pregnancy.

0–3 (Low): ~10–13% chance of natural pregnancy.

4–5 (Low-Moderate): ~30% chance.

6–7 (Moderate-High): ~45% chance.

8–10 (High): ~69–75% chance.

Clinical Utility

Post-Surgery: Validated to predict 36-month pregnancy rates.

Decision Tool: Helps decide between immediate IVF or attempting natural conception/IUI post-surgery.

Wang et al[1] in in 2013 analysed 199 women with endometriosis receiving IVF treatment after surgery. The EFI score and r-AFS classification in their ability to predict these IVF outcomes were compared in the same population. ROC curves were used to analyse the predictive values of the EFI and r-AFS indices for clinical pregnancy, and their accuracies were evaluated by sensitivity, specificity, and the Youden's index. The Area Under the Curve (AUC) of the EFI score (AUC = 0.641, Standard Error (SE) = 0.039, P = 0.001, 95% CI = 0.564-0.717, cut-off score = 6) was significantly larger than that of the r-AFS classification (AUC = 0.445, SE = 0.041, P = 0.184, and 95% CI = 0.364-0.526). The antral follicle count, oestradiol level on day of hCG, number of oocytes retrieved, number of oocytes fertilised, and number of cleaved embryos in the greater than or equal to 6 EFI score group was greater than that of the lower than or equal to 5 EFI score group, and the dose of gonadotropin of the greater than or equal to 6 EFI score group were less than that in the lower than or equal to 5 EFI score group. Implantation rate, clinical pregnancy rate, and cumulative pregnancy rate in the greater than or equal to 6 EFI score group were higher than in the lower than or equal to 5 EFI score group. They suggested that the EFI has more predictive power for IVF outcomes in endometriosis patients than the r-AFS classification. The clinical pregnancy rate was higher in patients with EFI greater than or equal to 6 score than with EFI lower than or equal to 5 score.

Observational cross-sectional study was conducted by Kavya et al[8] in 2022 on 76 patients who desire to conceive with suspected endometriosis related infertility treated by laparoscopic surgery. EFI score was calculated and information on mode of conception (spontaneous or assisted reproductive technology-ART) was collected by contacting the patients. Results: In women who conceived spontaneously, mean age was 31.33+3.29 years. Factors found to be significant were age (pvalue-0.0001), time to achieve spontaneous pregnancy (median-9 months). Patients with higher EFI score (5-10) has good spontaneous conception rate (96.2%) compared to those with lower EFI score (0-4) that conceived better with ART (60%) women with lower least function score has better outcome for spontaneous pregnancy (p<0.001).

Limitations

Using the EFI in routine clinical practice to triage patients remains a challenge. Patients with an EFI ≤4 may have residual endometriosis (incomplete surgery, a poor LF Score). Zhang et al[7] explained that visible endometriotic

lesions are not sufficient to describe disease severity and that surgery cannot correct the associated molecular and immune phenomena.

Gynaecologists may find it difficult to persuade women to opt for an expectant management, but clinical experience shows that patients prefer to attempt a natural conception. Surgery for endometriosis infertility was always to improve natural conception.

The EFI is useful only for infertility patients who have had surgical staging of their disease. It is not intended to predict any aspect of endometriosis-associated pain

In ART, limitation of the study was that, the stimulation protocols used in assisting conception, were not included as a part of the studies.

Conclusion

Endometriosis fertility (EFI) score is clinically useful in patients with surgically and histologically confirmed endometriosis who wish to be pregnant. The higher the EFI score, higher the chances of spontaneous pregnancy. EFI scoring system is effective in predicting postoperative successful spontaneous pregnancy rates in the patients with endometriosis. Patients with higher EFI score achieved successful spontaneous pregnancy. EFI scoring system is highly effective in predicting postoperative spontaneous pregnancy rate in patients with endometriosis and can be implemented in the clinical practice effectively.

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Calendar for AOGD Monthly Clinical Meeting 2025-2026

27th March 2026	LHMC & SSK Hospital
24th April 2026	Hamdard Institute of Medical Sciences and Research

Leiomyomas, Imaging and Assisted Reproductive Technology (ART): Why, What and How

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Introduction

Leiomyomas, commonly known as uterine fibroids, represent the most frequent benign tumors of the female reproductive tract and are detected in up to 40% of women of reproductive age.[1] Their prevalence is even higher among women undergoing infertility assessment. Although many fibroids remain asymptomatic, they may negatively influence fertility and pregnancy outcomes.

Several studies have reported that fibroids—particularly those involving the endometrial cavity—are associated with decreased clinical pregnancy rate (CPR), implantation rate (IR), and live birth rate (LBR), along with increased miscarriage rates.[2,3] Conversely, other studies have shown satisfactory ART outcomes in the presence of fibroids that do not distort the uterine cavity.[4] These conflicting findings have generated uncertainty among clinicians regarding whether fibroids should be treated prior to ART.

In this context, accurate imaging evaluation is fundamental. Imaging allows identification of clinically significant fibroids, assessment of their relationship to the uterine cavity, and appropriate planning of uterine-preserving interventions when needed. A structured imaging approach therefore plays a crucial role in optimizing fertility management.

Leiomyomas, Imaging and ART

Guideline Perspective

Professional societies including the American Society for Reproductive Medicine, American College of Obstetricians and Gynecologists, Royal Australian and New Zealand College of Obstetricians and Gynaecologists, Society of Obstetricians and Gynaecologists of Canada, and Collège National des Gynécologues et Obstétriciens Français broadly agree that the adverse reproductive impact of fibroids is primarily related to distortion of the endometrial cavity.[5]

Current guideline-based recommendations indicate:

- Submucosal fibroids should be treated prior to ART because of their consistent negative effect on implantation.[5,6]
- Evidence regarding intramural fibroids without cavity distortion remains inconclusive, and management decisions should be individualized.[7]

These recommendations highlight the importance of detailed imaging assessment before initiating ART.

Role of Imaging in ART Planning

Imaging helps identify fibroids that may interfere with implantation and determine whether surgical intervention is required.

Fibroids commonly requiring treatment before ART

- Submucosal fibroids (FIGO types 0–2)
- Intramural fibroids larger than 4–5 cm
- Fibroids causing endometrial cavity distortion[6,7]

Fibroids usually not requiring treatment

- Small subserosal fibroids
- Intramural fibroids without cavity distortion[7]

Imaging Modalities

Ultrasound

Ultrasound is the preferred first-line imaging modality for assessing uterine fibroids in infertility workup.[8]

Transvaginal ultrasound provides:

- High sensitivity for detecting fibroids
- Clear visualization of the endometrial cavity
- Cost-effective and widely available evaluation

The introduction of three-dimensional ultrasound and multislice reconstruction has improved the ability to assess the spatial relationship between fibroids and the uterine cavity.[9]

Despite these advantages, ultrasound may have limitations in women with a markedly enlarged uterus, numerous fibroids, or suspected adenomyosis.[10]

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) offers superior soft-tissue contrast and multiplanar imaging capability. It is particularly valuable when:

- Multiple or large fibroids are present
- Precise preoperative mapping is required
- Differentiation between fibroids and adenomyosis is necessary
- Degenerative changes within fibroids need

evaluation[10,11]

MRI is also considered the reference imaging modality prior to uterine artery embolization and high-intensity focused ultrasound therapies.[11]

Complementary Role of Ultrasound and MRI

Rather than competing modalities, ultrasound and MRI serve complementary roles in clinical practice.

Transvaginal ultrasound is ideal for

- Initial infertility evaluation
- Detection of small fibroids
- Assessment of submucosal lesions
- Evaluation of endometrial cavity distortion

Transabdominal ultrasound may be added for

- Large uterine size
- Multiple fibroids
- Limited visualization with transvaginal imaging

MRI is particularly useful for evaluating

- Junctional zone abnormalities
- Exact fibroid location relative to the uterine cavity
- Coexisting adenomyosis
- Degenerative changes within fibroids[10,11]

Fibroid Classification Systems for Surgical Planning

Lasmar / STEP-W Classification

The STEP-W classification evaluates five parameters: size, topography, extension of the base, penetration into the myometrium, and lateral wall involvement.

Each parameter receives a score, and the cumulative score predicts the complexity of hysteroscopic resection.[12] Three-dimensional ultrasound can improve the accuracy of this classification.

Parameters	Escore	0	1	2
S		≤ 2	2 a 5	> 5
T		low	mid	upper
E		≤ 1/3	1/3-2/3	> 2/3
P		0	≤ 50%	> 50%
W		a / p	lateral	

Figure 1. STEP-W (Lasmar) Classification for Submucosal Fibroids

The STEP-W classification system proposed by Lasmar et al. evaluates the complexity of hysteroscopic resection of submucosal fibroids using five parameters: Size (S), Topography (T), Extension of the base (E), Penetration into the myometrium (P), and Wall involvement (W). Each variable is assigned a numerical score, and the cumulative score predicts the technical difficulty of hysteroscopic removal. Lower scores indicate lesions that are easier to resect hysteroscopically, whereas higher scores suggest more complex procedures that may require staged surgery or alternative approaches. This classification assists surgeons in preoperative planning and counseling of patients undergoing hysteroscopic myomectomy. (Adapted from Lasmar et al.[12]

FIGO Classification

The International Federation of Gynecology and Obstetrics classification categorizes fibroids according to their relationship with the endometrium and serosal surface.[13]

Type	Description
0	Pedunculated intracavitary
1	Submucosal (<50% intramural)
2	Submucosal (≥50% intramural)
3	Intramural contacting endometrium
4	Intramural
5	Subserosal ≥50% intramural
6	Subserosal <50% intramural
7	Pedunculated subserosal
8	Other locations (e.g., cervical)

This system standardizes communication between radiologists and surgeons, although it does not incorporate fibroid number or size.

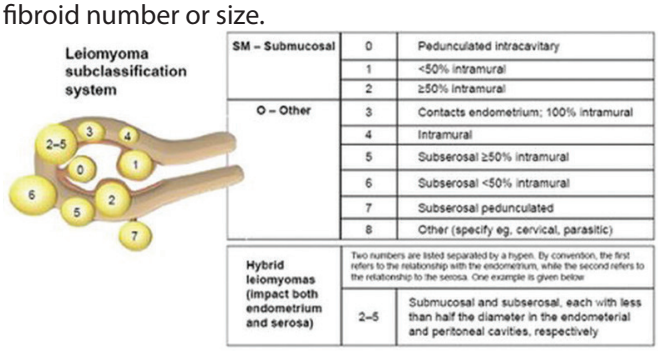

Figure 2. FIGO Classification of Uterine Leiomyomas

The fibroid classification system proposed by the International Federation of Gynecology and Obstetrics categorizes leiomyomas according to their relationship with the endometrial cavity and the uterine serosal surface. Types 0–2 represent submucosal fibroids with varying degrees of myometrial involvement; Type 3 and

Type 4 represent intramural fibroids; Types 5–7 represent subserosal fibroids; and Type 8 includes fibroids in atypical locations such as cervical or parasitic leiomyomas. The FIGO system provides a standardized method for describing fibroid location and is widely used in both imaging and surgical planning. (Adapted from Munro et al.[13]

Morphological Uterus Sonographic Assessment (MUSA)

The MUSA consensus, developed by experts from the International Ovarian Tumor Analysis Group and the European Society of Gynaecological Endoscopy, standardizes ultrasound terminology used for describing uterine pathology.[14]

Recommended Reporting Parameters

Key elements include:

- Uterine size and orientation
- Serosal contour
- Myometrial symmetry
- Junctional zone appearance
- Number and size of fibroids
- FIGO classification and localization

When numerous fibroids are present, all submucosal lesions and at least four representative non-submucosal fibroids should be documented.

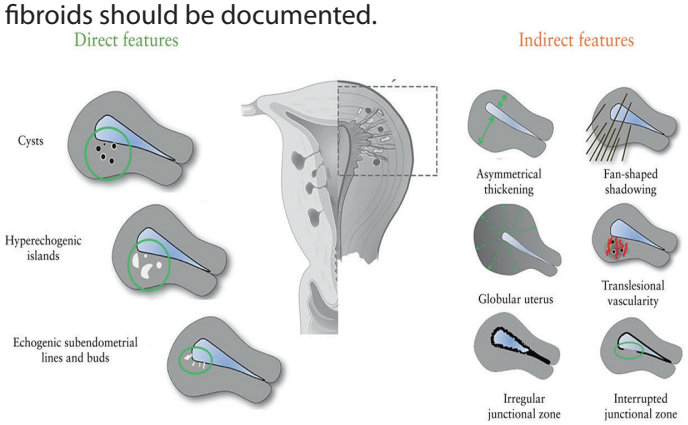

Figure 3. MUSA (Morphological Uterus Sonographic Assessment) Reporting Framework

The MUSA consensus provides a standardized approach to describing uterine morphology on ultrasound. The system recommends structured reporting of uterine characteristics including uterine size and orientation, serosal contour, myometrial echotexture, junctional zone appearance, and characteristics of fibroids such as number, size, location, and vascularity. Adoption of this standardized terminology improves communication between imaging specialists and clinicians and enhances reproducibility of ultrasound findings in clinical practice and research. (Adapted from Van den Bosch et al.[14]

Myometrial Mantle Assessment

Assessment of myometrial mantle thickness is important prior to hysteroscopic surgery.

- **Inner mantle:** distance between fibroid margin and endometrial surface
- **Outer mantle:** distance between fibroid margin and serosal surface

In FIGO type 2 fibroids, an outer myometrial mantle thickness less than 5 mm may increase the risk of uterine perforation during hysteroscopic resection.[12]

MRI Evaluation of Fibroids

Key MRI Sequences

MRI Sequence	Clinical Role
Axial T2-weighted	Fibroid localization
Coronal T2-weighted	Relationship to uterine cavity
T1-weighted	Detection of hemorrhage
T1 fat-suppressed	Identification of red degeneration
Diffusion-weighted imaging	Evaluation of atypical lesions
Dynamic contrast imaging	Assessment of vascularity
MR angiography	Planning uterine artery embolization

MRI Appearance of Fibroid Degeneration

Degeneration Type	MRI Appearance
Typical fibroid	Low T2 signal
Hyaline degeneration	Very low T2 signal
Cystic degeneration	High T2 signal
Red degeneration	High T1 signal
Calcification	Signal void

Key Imaging Reporting Parameters

Parameter	What to Report	Clinical Relevance
FIGO classification	Types 0–8	Standardized surgical classification
Relationship to cavity	Distortion / indentation / none	Determines ART impact
MRI signal characteristics	T1/T2 intensity	Detects degeneration
Enhancement pattern	Homogeneous / heterogeneous	Helps identify atypical lesions
Vascularity	Doppler flow	Important for embolization planning
Associated pathology	Adenomyosis, polyps	Comprehensive pelvic assessment

Conclusion

- Uterine fibroids are common in women undergoing

infertility evaluation and may influence ART outcomes.

- The effect of fibroids on fertility largely depends on their relationship to the endometrial cavity.
- Submucosal fibroids consistently impair implantation and should be removed before ART.
- The impact of intramural fibroids without cavity distortion remains debated, and management should be individualized.
- Transvaginal ultrasound is the first-line imaging modality for fibroid evaluation in infertility workup.
- MRI serves as a complementary technique, particularly for complex cases, large fibroids, or when adenomyosis is suspected.
- Standardized classification systems such as FIGO, STEP-W, and MUSA improve reporting and surgical planning.
- Imaging assessment of cavity distortion, fibroid size, and myometrial mantle thickness is critical before hysteroscopic resection.
- Accurate imaging helps guide decisions regarding myomectomy versus uterus-sparing therapies in women planning ART.

Imaging is a critical component of infertility evaluation in women with uterine fibroids. A structured imaging approach enables precise characterization of fibroids and their relationship to the endometrial cavity. This information is essential for determining whether intervention is necessary prior to ART.

By providing detailed information regarding **fibroid number, size, location, and cavity distortion**, imaging guides clinical decisions between myomectomy and uterus-sparing treatments such as uterine artery embolization, radiofrequency ablation, or high-intensity focused ultrasound. Integrating ultrasound and MRI findings therefore allows individualized management aimed at improving reproductive outcomes in women undergoing ART.

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Adenomyosis and Infertility: Do We Treat Before IVF?

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1. Introduction

Adenomyosis is a benign gynecological disorder characterized by the presence of ectopic endometrial glands and stroma within the myometrium, accompanied by hypertrophy and hyperplasia of surrounding smooth muscle fibers. Traditionally, adenomyosis was considered a disease affecting multiparous women in their fourth or fifth decade of life and was usually diagnosed after hysterectomy. However, advances in imaging technologies such as high-resolution transvaginal ultrasonography and magnetic resonance imaging have led to increased detection in younger women, including those presenting with infertility.[1]

Recent evidence suggests that adenomyosis may significantly impair reproductive outcomes. The disorder alters uterine architecture, affects endometrial receptivity, and disrupts normal uterine peristalsis, thereby interfering with embryo implantation. As a result, adenomyosis has been associated with reduced implantation rates, lower clinical pregnancy rates, and increased miscarriage rates in women undergoing assisted reproductive technologies.[2]

The management of adenomyosis in infertile patients remains challenging. A major clinical question is whether adenomyosis should be treated before IVF to improve reproductive outcomes. Current evidence regarding the relationship between adenomyosis and infertility and contemporary strategies for managing adenomyosis before IVF need discussion.

2. Epidemiology

The reported prevalence of adenomyosis varies widely due to differences in diagnostic criteria and study populations. Earlier studies relying on histological diagnosis reported prevalence rates ranging from 5–20%. With modern imaging techniques, the prevalence among infertile women is estimated to range from 7% to 28%.[3]

Adenomyosis frequently coexists with other gynaecological conditions (Table 1), particularly endometriosis and uterine fibroids, which further complicates fertility outcomes.[4]

Table 1. Prevalence of Adenomyosis in Infertile Women

Clinical Condition	Estimated Prevalence
Isolated adenomyosis	10–15%
Adenomyosis with endometriosis	6–20%
Adenomyosis with fibroids	5–10%
Adenomyosis with both endometriosis and fibroids	5–7%

The increased use of ultrasound-based diagnostic criteria, particularly those proposed by the Morphological Uterus Sonographic Assessment (MUSA) group, has improved diagnostic accuracy in clinical practice.[5]

3. Pathophysiology of Infertility in Adenomyosis

The mechanisms by which adenomyosis affects fertility are complex and multifactorial. Structural changes within the uterus and molecular alterations within the endometrium contribute to impaired reproductive outcomes.

3.1 Disruption of the Junctional Zone

The junctional zone (JZ) is the inner layer of the myometrium responsible for uterine peristalsis and sperm transport. Adenomyosis leads to thickening and distortion of the junctional zone, which may interfere with embryo implantation.[6]

3.2 Abnormal Uterine Peristalsis

Adenomyosis disrupts the junctional zone (JZ) between endometrium and myometrium. This leads to excessive uterine peristalsis and dysperistalsis

Consequences:

- Impaired sperm transport
- Embryo displacement after transfer
- Reduced implantation stability

3.3 Chronic Inflammation

Adenomyotic tissue produces inflammatory cytokines and prostaglandins that create a hostile uterine environment. Elevated levels of inflammatory mediators such as interleukin-6 and tumour necrosis factor-alpha have been demonstrated in adenomyotic uteri . Adenomyotic tissue produces high levels of :

- **Pro-inflammatory cytokines**
  - IL-6
  - TNF-α
  - IL-1β
- **Prostaglandins**

It impacts fertility by

- Hostile endometrial environment
- Reduced embryo–endometrium signalling
- Increased oxidative stress

2.4 Progesterone Resistance

Adenomyotic tissue shows **reduced progesterone receptor expression. This** leads to:

- Poor decidualization
- Luteal phase dysfunction
- Implantation failure

2.5 Altered Implantation Markers

Key implantation markers are dysregulated (7) (Table 2):

Table 2: Implantation Markers

Marker	Effect in Adenomyosis
HOXA10 / HOXA11	Downregulated → impaired receptivity
Integrins (αvβ3)	Reduced expression
LIF (Leukemia inhibitory factor)	Decreased
Pinopodes	Reduced formation

These changes impair the **window of implantation contributing to implantation failure**.

2.6 Enlarged Uterine Volume

Large adenomyotic uteri has a reduced or distorted uterine cavity and implantation surface. Hence it is ssociated with lower IVF live-birth rates

3.7 Altered Immune Environment

Immune dysregulation includes:

- Increased **macrophages and NK cells**
- Elevated **Th17 cytokines**
- Reduced **Treg cells**
- This leads to increased embryo rejection risk.

3.8 Increased Angiogenesis and Fibrosis

Adenomyosis increases:

- VEGF
- TGF-β

This results in

- Abnormal vascularization
- Fibrotic myometrium
- Poor endometrial–embryo interaction

4. Diagnosis of Adenomyosis

4.1 Transvaginal Ultrasound

Transvaginal ultrasound is the first-line imaging modality for diagnosing adenomyosis. Characteristic ultrasound features include:

- Globular enlargement of the uterus
- Asymmetric myometrial thickening
- Myometrial cysts
- Hyperechoic linear striations
- Fan-shaped shadowing

Typical Ultrasound Signs of Adenomyosis in MUSA[5]

1. Globular Uterus

- The uterus becomes rounded or enlarged instead of its normal pear shape.
- This occurs due to diffuse thickening of the myometrium.

2. Asymmetrical Myometrial Thickening

- One uterine wall (usually posterior wall) is thicker than the other.
- This asymmetry is a common sign of adenomyosis.

3. Myometrial Cysts

- Small anechoic (black) cystic spaces within the myometrium.
- Usually 1–7 mm in size.

4. Hyperechoic Islands

- Bright echogenic spots inside the myometrium.
- Represent ectopic endometrial tissue.

5. Fan-Shaped Shadowing

- Radiating acoustic shadows from the myometrium.
- Unlike fibroids, these shadows do not come from a single mass.

6. Irregular or Interrupted Junctional Zone

The junctional zone is the boundary between endometrium and myometrium

In adenomyosis it appears:

- Irregular
- Thickened
- Poorly defined

7. Linear Striations (Sub endometrial Lines/Buds)

- Thin echogenic lines extending from endometrium into the myometrium.
- These lines represent endometrial invasion into muscle tissue.

4.2 Magnetic Resonance Imaging

MRI is particularly useful in complex cases or when ultrasound findings are inconclusive. Diagnostic criteria include:

- Junctional zone thickness greater than 12 mm
- Myometrial cysts and myometrium with scattered high-signal intensity foci
- Diffuse or focal adenomyosis

5. Classification of Adenomyosis

Several classification systems have been proposed based on lesion location and severity. These are based on histopathology, ultrasound and MRI findings. Usually classifications used are based on imaging for clinical decisions. They are based on junctional zone thickness, focal or diffuse lesions, cystic appearance, single or two myometrial walls involved, size and number of lesions and volume of uterus.[8]

Table 3. Imaging-Based Prognostic Categorisation of adenomyosis

Grade	Description	Clinical Relevance
Mild	Small focal lesions	Minimal impact on fertility
Moderate	Multiple lesions	Reduced implantation
Severe	Diffuse disease	Poor IVF outcomes

Disease severity is strongly associated with poorer reproductive outcomes.(Table 3)

6. Impact of Adenomyosis on IVF Outcomes

A growing body of literature has evaluated the effect of adenomyosis on ART outcomes. A large meta-analysis demonstrated that adenomyosis significantly reduces live birth rates and increases miscarriage risk in women undergoing IVF (2). Women with adenomyosis had lower live birth rate (OR 0.59) and clinical pregnancy rate (OR 0.66), with higher miscarriage risk (OR 2.11).(9) Another metanalysis of 9 studies by Paolo Vercellini showed that adenomyosis reduced clinical pregnancy rates by 28% and increased miscarriage risk compared with controls. Screening before ART for adenomyosis was recommended in these studies.[10]

Assessing adenomyosis's impact on reproductive outcomes is challenging due to the limited differentiation between endometriosis and adenomyosis in many studies.

7. Medical Management Before IVF

Medical management before IVF for adenomyosis is recommended to surpress the adverse impact of the disease on implantation. Various medical interventions are used for suppression like GnRH agonists and antagonist, progesterones and Letrozole

7.1 GnRH Agonists

GnRH agonists are the most commonly used medical therapy before IVF. (Fig 1)

They induce hypoestrogenism and suppress adenomyotic lesions, leading to:

- Reduced uterine volume
- Reduced inflammatory activity
- Improved endometrial receptivity.

Pre-treatment with GnRH agonists for **2–3 months prior to embryo transfer** has been associated with improved implantation and pregnancy rates in several studies.(11) It has been seen that uterus larger than 8 weeks of gestation had a higher miscarriage rate and a lower live birth rate in all ET cycles. These are the cases requiring volume reduction.[12]

However a recent metanalysis has shown that GnRH agonist pre-treatment before ART carries a potential benefit in improving outcomes in terms of higher CPR, but there is no significant impact on LBR and MR. Pre-treatment with GnRH agonists could be adopted as a possible alternative ART protocol in selected patients with adenomyosis and infertility.[13]

6.2 Oral GnRH Antagonists

Oral GnRH antagonists have been used for supressing endometriosis and adenomyosis. They reduce the lesion and decrease inflammation helping in implantation. The ones in use are Elgolix, Relugolix and Linzagolix. The advantage is immediate effect and unlike the GnRH agonist immediately reversible. Add back therapy with oestrogen and progesterone is recommended when used for long periods.

7.3 Progestins

7.3.1.Oral progestins

Progestins such as **dienogest** suppress adenomyotic activity and may reduce uterine inflammation. However, their role before IVF remains under investigation.

7.3.2 LNG – IUD

Pre-treatment with the LNG-IUD for 3 months before embryo transfer has been proposed to improve the reproductive outcomes of patients undergoing in vitro fertilization with a significantly increased ongoing pregnancy rate (41.8% versus 29.5%)[14]

7.4 Aromatase Inhibitors

Aromatase inhibitors reduce local oestrogen production in adenomyotic tissue and may be used in combination with GnRH agonists.

GnRH agonist therapy
↓
Estrogen suppression
↓
Reduction in adenomyotic lesions
↓
Improved uterine environment
↓
Better implantation potential

Figure1: Mechanism of action of GnRH agonists in improving uterine receptivity before IVF.

8. Surgical Management

Surgical treatment may be considered in selected cases before IVF.[15]

Indications

- Focal adenomyoma
- Severe symptomatic disease
- Recurrent implantation failure
- Large uterine size affecting implantation.

Surgical options.

- Adenomyomectomy[16]
- Cytoreductive surgery
- Laparoscopic excision

However, surgery carries potential risks like intrauterine adhesions and uterine rupture in pregnancy due to reduced uterine integrity. Therefore, surgery is usually reserved for carefully selected patients. The reproductive outcomes following fertility- sparing interventions are promising for women with adenomyosis who desire fertility.[17]

9. Non-Surgical Techniques

Non-surgical thermal techniques, including high-intensity focused ultrasound ablation, percutaneous microwave ablation, and radiofrequency ablation, are much less invasive techniques that have shown effectiveness in improving fertility. Although evidence remains limited, all these procedures have demonstrated a favourable safety profile. (Table 4)

Table 4: Interventions for adenomyosis before ART

Medical Treatment	Surgical Treatment	Non-Surgical Ablative Techniques
GnRH analogues	Reduction Surgery	High-intensity focused ultrasound
Progesterone & LNG IUD	Excision of localized lesion	Percutaneous microwave Ablation
Letrozole		Radiofrequency Ablation

10. IVF Strategies for Patients With Adenomyosis

Several ART strategies may improve reproductive outcomes in adenomyosis.(Fig 2)

10.1 Long GnRH Down-Regulation Protocol

Prolonged suppression before ovarian stimulation may improve implantation rates.

9.2 Freeze-All Strategy & Frozen Embryo Transfer

Embryos are cryopreserved and transferred in a later cycle after uterine suppression. Frozen embryo transfer may improve outcomes by allowing time for uterine preparation.[18]

However a recent metanalysis has shown that in IVF/ICSI, the FET strategy has been associated with more favourable reproductive outcomes compared to the fresh ET strategy in women with endometriosis. Whereas in women with adenomyosis, pregnancy outcomes were comparable between the FET and fresh ET groups.[19]

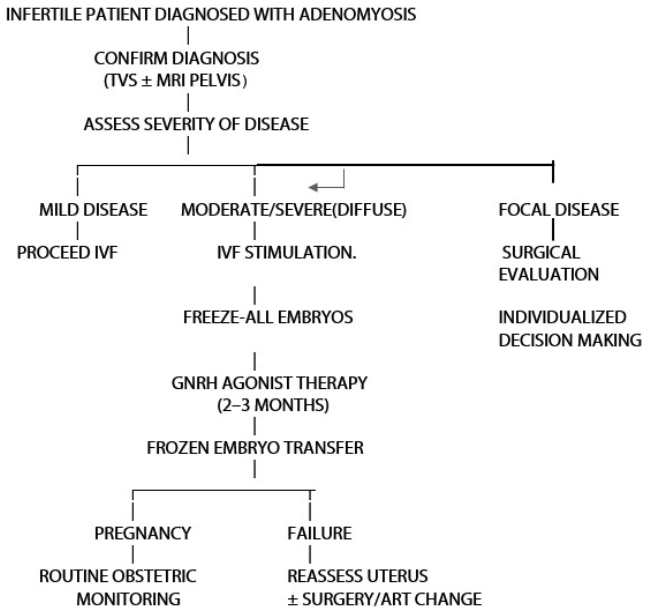

Figure 2. Management Algorithm for Adenomyosis Before IVF

10. Obstetric and Perinatal Outcomes in Adenomyosis

Beyond implantation, adenomyosis impacts obstetric outcomes.[20]

1. Preterm Birth

Meta-analyses demonstrate increased risk of preterm delivery in women with adenomyosis. Chronic inflammation and impaired placentation likely contribute.

2. Hypertensive Disorders of Pregnancy

Impaired spiral artery remodeling may increase risk of preeclampsia. Horton et al. identified higher rates of hypertensive disorders in affected women.

3. Placenta Previa and Placental Abruption

Abnormal uterine architecture may increase placental implantation abnormalities.

4. Small for Gestational Age (SGA)

Defective placentation may impair fetal growth.

5. Cesarean Delivery

Higher cesarean section rates are reported, potentially

related to uterine dysfunction.

6. Uterine Rupture (Post-Surgical)

Following adenomyomectomy, uterine rupture risk must be considered in subsequent pregnancies.

Therefore, these pregnancies require careful antenatal monitoring.

11. Current Evidence and Guideline Recommendations

Although universal guidelines are lacking, most experts recommend an individualized management approach.[21]

Key recommendations include:

1. Accurate diagnosis using standardized imaging criteria
2. Assessment of disease severity
3. Medical suppression with GnRH agonists in moderate-to-severe disease
4. Consideration of frozen embryo transfer
5. Surgical treatment only in selected cases .

13. Conclusion

Adenomyosis is increasingly recognized as a significant contributor to infertility and poor outcomes in assisted reproductive technology. The condition alters uterine architecture, disrupts uterine contractility, and impairs endometrial receptivity. Evidence indicates that adenomyosis reduces implantation and live birth rates while increasing miscarriage risk in IVF cycles.

Treatment prior to IVF remains controversial. Women with mild disease may proceed directly to IVF, whereas those with moderate or severe adenomyosis may benefit from medical suppression with GnRH agonists before embryo transfer. Surgical management should be reserved for selected cases such as focal adenomyoma or recurrent implantation failure. Individualized management strategies based on disease severity, patient age, and prior ART outcomes are essential to optimize reproductive success.

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Forthcoming Events

- 27.03.2026 - The AOGD Monthly Clinical Meeting (virtual) conducted by the Department of Obst. & Gynae, LHMC & SSK Hospital on 27th March, 2026
- 30.03.2026 – General Body Meeting of AOGD & Handing Over to AIIMS at MEU hall, Swarn Jayanti Auditorium, LHMC & SSK Hospital

Pelvic Myofascial Pain and Bladder Pain Syndrome: Structured Clinical Evaluation and Integrated Management Approach

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Introduction

Chronic pelvic pain (CPP) is defined by the American College of Obstetricians and Gynecologists, The International Association for the Study of Pain (IASP), and the ReVITALize Data Definitions Initiative as pain perceived to arise from pelvic organs or structures that persists for more than six months.[1] This condition is frequently accompanied by significant cognitive, behavioral, emotional, and sexual consequences and may be associated with symptoms involving the lower urinary tract, bowel, pelvic floor, myofascial structures, or gynecological disorders. Cyclical pelvic pain may also be categorized under CPP when it results in substantial cognitive, behavioral, sexual, or emotional impact.

Chronic pelvic pain differs from acute pelvic pain in several ways. Acute pain usually results from inflammatory, infectious, ischemic, or traumatic causes and resolves with treatment and healing. In contrast, persistent pain may lead to a chronic stress phenotype characterized by a cycle of physical and psychological effects, often resulting in functional impairment and reduced quality of life.[1]

Prevalence

The prevalence of chronic pelvic pain varies depending on the definition used and whether chronic gynecological conditions such as endometriosis, adenomyosis, and pelvic inflammatory diseases are included. It affects approximately **15% of women of reproductive age**.[2] A community-based study reported a three month prevalence of 24%, with nearly one-third of affected women experiencing pain for more than five years.[3]

Causes of CPP

The causes of chronic pelvic pain (CPP) are multifactorial and include gynecological conditions such as endometriosis, adenomyosis, pelvic adhesions, chronic pelvic inflammatory disease, remnant ovary syndrome, trapped ovary syndrome, and pelvic congestion syndrome. Non-gynecological causes include gastrointestinal disorders (irritable bowel syndrome, inflammatory bowel disease, coeliac disease), urinary conditions (interstitial cystitis, urethral syndrome), neurological disorders (pudendal neuralgia, nerve entrapment, trigger points), and musculoskeletal abnormalities (Fig 1).

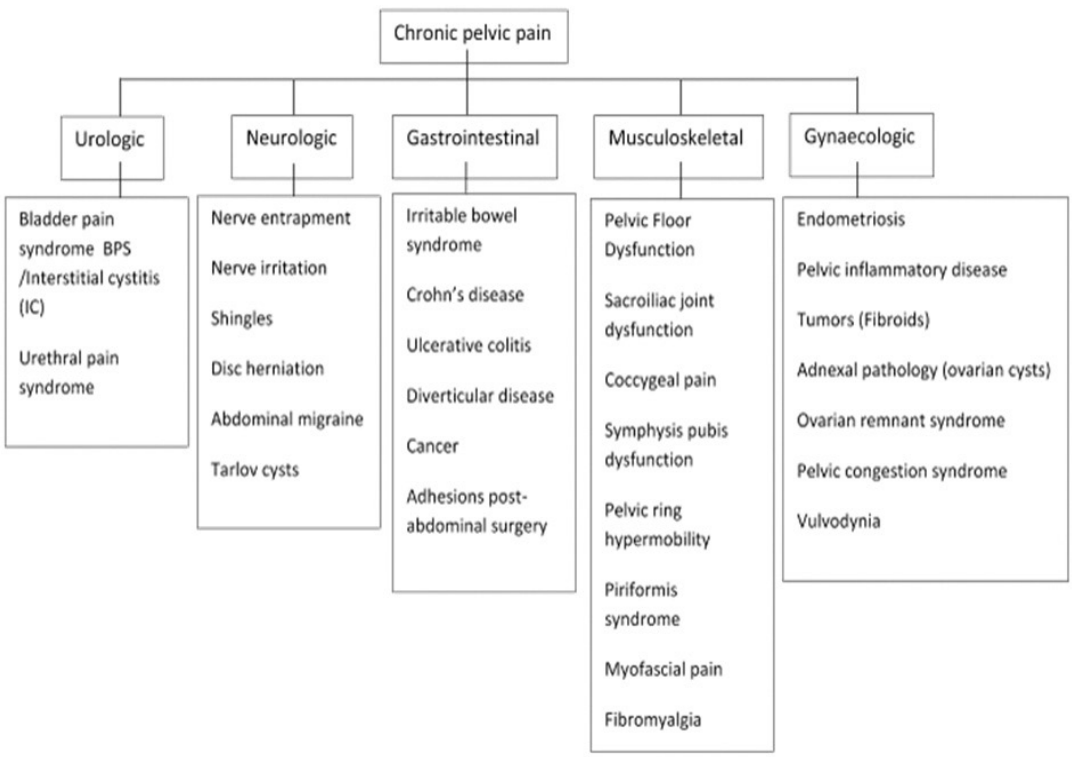

Figure 1: Causes of Chronic Pelvic Pain

This article primarily focuses on the evaluation and management of myofacial/**musculoskeletal pain** arising from pelvic muscles and fascia and **interstitial cystitis/ Bladder pain syndrome** as cause of chronic pelvic pain.

Evaluation of patient with CPP

For detailed evaluation of women with chronic pelvic pain (CPP), history taking is guided by questionnaire outlined in the article by Fred M. Howard and in Chapter 12 of Williams Gynecology (Fig 2).[2,4] Assessment is ideally conducted in a specialized clinic, with careful attention to psychosocial factors and any history of abuse. Because of the sensitive nature of these issues, patients may require multiple consultations to share such information with the treating gynecologist. Clinical findings are documented using the "Pelvic Pain Assessment Form" developed by the International Association for the Study of Pain, available through the reference link.[4,5]

1. Describe the location, quality, severity, and timing of your pain.
2. When and how did your pain start and how has it changed?
3. What makes your pain better or worse?
4. What other symptoms or health problems do you have?
5. Do you have frequency, urgency, or bloody urine?
6. Do you have nausea or vomiting, diarrhea, constipation, or rectal bleeding?
7. Do you have pain with your periods?
8. Did your pain start initially as menstrual cramps?
9. Have you had surgery? What was the reason?
10. How many pregnancies have you had?
11. How did you deliver? Was there an episiotomy?
12. What form of birth control do you use and have you used in the past?
13. Have you ever been treated for a sexually transmitted disease or pelvic infection?
14. Do you have pain with deep penetration during intercourse?
15. Are you depressed or anxious?
16. Have you been treated for mental illness in the past?
17. Have you been or are you now being abused physically or sexually?
18. What prior evaluations or treatments have you had for your pain?
19. Have any of the previous treatments helped?
20. What medications are you taking now?
21. How has the pain affected your quality of life?
22. What do you believe or fear is causing your pain?

Figure 2: Key Questions for CPP (Williams Gynecology. 4th ed.)[2]

A detailed abdominal examination is performed to localize tenderness. The **Carnett test** helps differentiate pain arising from the abdominal wall from intra-abdominal visceral pain. Examination of the external genitalia is conducted using a cotton-tipped swab to identify local and vestibular tenderness. The area beneath the urethra and bladder is palpated with a single digit to detect localized tenderness. Tenderness behind the urethra may indicate a urethral diverticulum, whereas pain posterior to the bladder is more commonly associated with interstitial cystitis or bladder pain syndrome.

Pelvic musculoskeletal assessment includes sweeping finger along the **pelvic floor muscles**, pubococcygeus, iliococcygeus, and **obturator internus**, to identify trigger points with gentle pressure (Fig 3). The **piriformis muscle** is then palpated posterolaterally above the ischial spine; it is made taut by asking the patient to abduct the thigh against resistance. Reproduction of the patient's typical pain suggests myofascial trigger points. Finally, the coccyx is palpated and mobilized. Pain reproduced with coccygeal movement of less than 30° suggests coccydynia. Single digit examination is followed by per speculum and bimanual per vaginal examination.

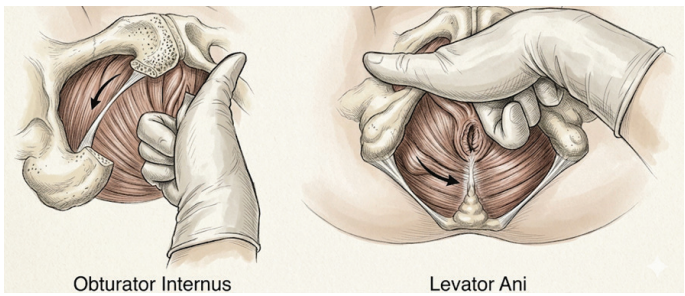

Figure 3: Single digit palpation of Levator Ani and Obturator Internus muscles

It is important to note that speculum and bimanual examination should not be done before performing single digit examination.

Treatment of Pelvic Myofascial Pain

The **non pharmacological therapies** include tissue mobilization for **myofascial release**, manipulative therapies, electrical stimulation, retraining of pelvic muscles and stretching of pelvic floor muscles etc.[6]

In our practice the local massage of the trigger points in the levator and obturator muscles were poorly tolerated by patient as it caused pain flare. The pelvic muscle stretch exercises advised by the physiotherapist were not regularly practiced as there was no instant relief of symptoms.

Cognitive Behavior Therapy: The patients of CPP suffer from depression and anxiety which may be a primary disorder associated with fibromyalgia or secondary to the chronicity of debilitating pain. The studies have shown mild to moderate improvement in symptoms with cognitive behavior therapy. It helps patients in coping up with pain

and learning to modulate thoughts. At most of the centers, for CBT patient needs to be referred to psychiatrist which is not acceptable to many patients in the beginning of treatment.

Neuropathic Medications: serotonin–norepinephrine reuptake inhibitors (duloxitene, venlafaxine), tricyclic antidepressants (amitriptyline nortriptyline, and desipramine)), and Calcium channel alpha 2-delta ligand medications (gabapentin or pregabalin) have been used for treatment of neuropathic pain. These medications can be prescribed by gynecologist and are being used in the management of chronic pelvic pain after ruling out visceral pathology.[1,2]

Role of analgesics: For immediate pain relief non opioid analgesics like NSAIDS are preferred over opioid analgesics.

Our experience

Profile of patients referred to chronic pelvic pain clinic at our center- Most patients referred to our chronic pelvic pain clinic had previously received multiple courses of antibiotics for presumed pelvic inflammatory disease. Some had also been referred to medical or surgical outpatient departments because no significant tenderness was identified on bimanual pelvic examination.

For pelvic myofascial pain, treatment is initiated with **duloxetine**, a serotonin–norepinephrine reuptake inhibitor. **Gabapentin**, an α2-δ calcium channel ligand, is added if symptoms persist. In the initial phase, a short course of nonsteroidal anti-inflammatory drugs (NSAIDs) is prescribed, followed by intermittent use as required. For patients with persistent pain, trigger point injections using a local anesthetic combined with a long-acting steroid are offered. These injections may provide temporary relief lasting from one to ten days. In some patients the pain score decreases, while others experience little change in pain intensity.

Our experience suggests that emotional support and identification of precipitating factors play an important role in symptom control. One patient with tenderness in the piriformis muscle did not respond to neuropathic medications and had only partial relief with NSAIDs. After several pain clinic visits, it was noted that she worked as a tailor and sat on the floor for prolonged periods, which triggered her symptoms. She was advised to modify her working posture by using a table and chair. Following this change, her pain score decreased from 8 to 3 out of 10.

Another patient, a 25-year-old unmarried woman with past history of consensual intercourse, presented with severe burning pain in the vestibular region. After three trigger point injections with a local anesthetic and long-acting steroid, her pain score decreased from 9 to 6 out of 10. Although relief was incomplete, ongoing supportive and empathetic care helped her cope better with the symptoms.

Patients with chronic pelvic pain should ideally be managed in specialized clinics, where ongoing evaluation helps identify precipitating or trigger factors. Emotional support and cognitive behavioral therapy play an important role in symptom relief and improving quality of life.

Interstitial Cystitis and Bladder Pain Syndrome

The terms interstitial cystitis (IC) and bladder pain syndrome (BPS) are used interchangeably. They describe an unpleasant sensation—such as pain, pressure, or discomfort—perceived to originate from the urinary bladder and associated with lower urinary tract symptoms lasting more than six weeks, in the absence of infection or other identifiable causes.[2]

Etiology

The exact etiology of IC/BPS remains unclear. Proposed mechanisms include increased bladder mucosal permeability due to defects in the glycosaminoglycan layer, mast cell activation, altered expression of HLA class I and II antigens, abnormalities in Tamm–Horsfall protein, increased expression of inflammatory mediators such as interleukin-6 and P2X3 ATP receptors, and decreased expression of uroplakin and chondroitin sulfate.

Evaluation

Patients typically present with pelvic pain or pressure related to bladder filling, accompanied by urinary frequency and urgency. Symptoms may be aggravated by dietary triggers such as citrus fruits, caffeine, alcohol, and carbonated beverages, although patients may not always recognize these associations. On examination, single-digit vaginal palpation may elicit tenderness behind the urethra or bladder.

Investigations include urine analysis, cystoscopy, and urodynamic studies. IC/BPS is largely a diagnosis of exclusion, and investigations are aimed at ruling out other causes of similar symptoms. Cystoscopic findings such as Hunner lesions and glomerulations were previously considered characteristic of IC but may also occur in individuals without bladder pain syndrome. Urodynamic testing may demonstrate reduced bladder compliance and sensory urgency.

Potassium sensitivity test (PST) is not recommended due to high false negative results and for fear of exacerbation of symptoms. Instillation of lidocaine for confirmation of BPS is also not recommended.

Management of Interstitial Cystitis / Bladder Pain Syndrome

Management of interstitial cystitis/bladder pain syndrome (IC/BPS) is individualized and primarily directed toward

symptom relief and improvement in quality of life. Treatment generally follows a stepwise approach, beginning with conservative measures.[6]

Lifestyle and behavioral modifications constitute the initial management strategy. Patients are advised to avoid foods and beverages known to exacerbate symptoms, such as caffeine, citrus products, alcohol, and carbonated drinks. Bladder training techniques, adequate hydration, and stress management may help reduce symptom severity.

Pharmacologic treatment may be required in patients with persistent symptoms. Analgesics such as nonsteroidal anti-inflammatory drugs can be used for pain relief. Neuropathic pain medications including amitriptyline or gabapentin are often prescribed to modulate pain perception. Pentosan polysulfate sodium has been used to help restore the protective glycosaminoglycan layer of the bladder epithelium. Antihistamines such as hydroxyzine may also provide benefit due to their effect on mast cell activity.

Intravesical therapy is considered when oral medications fail to provide adequate relief. Bladder instillations using agents such as local anesthetics, heparin, or dimethyl sulfoxide may reduce inflammation and improve bladder symptoms.

Procedural treatments may be helpful in selected patients. Cystoscopic hydrodistension can provide temporary symptom relief in some cases. In patients with Hunner lesions, cystoscopic fulguration or steroid injection into the lesion may improve symptoms.

Patients with refractory symptoms often benefit from a **multidisciplinary approach**, involving gynecologists, urologists, pain specialists, and pelvic floor physiotherapists. Psychological support and cognitive behavioral therapy can further assist patients in coping with chronic symptoms and improving overall well-being.

Conclusion

Chronic pelvic pain is a multifaceted disorder with multiple possible causes, and musculoskeletal as well as urinary sources are frequently overlooked during routine gynecological evaluation. Pelvic myofascial pain and bladder pain syndrome should therefore be considered in women who present with persistent pelvic pain when standard gynecological investigations do not identify a clear etiology. Detailed history taking and focused clinical examination, particularly assessment of pelvic floor muscles and bladder-related tenderness, are important for establishing the diagnosis.

Management should be individualized and often requires a multidisciplinary approach. Treatment strategies such as pharmacologic therapy, trigger point injections, lifestyle adjustments, and identification of precipitating factors may help reduce symptom burden. Continuous patient education, empathetic clinical support, and regular follow-up are equally important, as they help patients understand and cope with chronic symptoms. Early recognition and appropriate management can reduce unnecessary investigations and treatments while improving overall patient well-being.

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Breaking New Ground in OB-GYN: GnRH Antagonist with add back therapy in Heavy Menstrual Bleeding Associated with Uterine Fibroids: Expanding Medical Options in Indian Gynaecological Practice

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Introduction

Uterine fibroids (leiomyomas) are the most common benign tumors of the female reproductive tract and represent a significant cause of gynaecological morbidity worldwide. Epidemiological studies suggest that up to 70% of women develop fibroids during their reproductive years, although only 20–30% become symptomatic.[1,2] The true prevalence may be even higher when assessed using imaging modalities such as transvaginal ultrasonography or magnetic resonance imaging. India, with its large reproductive-age population, contributes substantially to the global burden of fibroid disease.

Heavy menstrual bleeding (HMB) is the most common presenting symptom of uterine fibroids and a major contributor to iron deficiency anemia. According to the National Family Health Survey (NFHS-5), more than half of Indian women of reproductive age are anemic.[3] Fibroid-associated HMB further aggravates this public health concern, leading to fatigue, reduced work productivity, impaired quality of life, and increased healthcare utilization. Beyond physical morbidity, symptomatic fibroids significantly impact psychological well-being, sexual health, and social functioning.[4] Despite advances in surgical techniques and hormonal therapies, long-term medical options have historically been limited by safety concerns, tolerability issues, and restrictions on duration of use.

Pathophysiology of Uterine Fibroids and Heavy Menstrual Bleeding

Uterine fibroids are monoclonal smooth muscle tumors arising from the myometrium. Molecular studies have identified MED12 mutations in a substantial proportion of fibroids, implicating dysregulated transcriptional control in tumorigenesis.[5] Additional mechanisms include aberrant growth factor signalling, extracellular matrix deposition, angiogenesis, and altered inflammatory pathways.

Fibroid growth is hormonally dependent, driven primarily by estrogen and progesterone.[6] Estrogen upregulates progesterone receptor expression in fibroid tissue, thereby enhancing progesterone-mediated cellular proliferation and extracellular matrix accumulation. Progesterone, in turn, stimulates mitotic activity and inhibits apoptosis within fibroid cells. This hormonal interplay explains the regression of fibroids after menopause and their responsiveness to endocrine manipulation.

Heavy menstrual bleeding in women with fibroids results from multiple mechanisms. Submucosal and intramural fibroids increase endometrial surface area and disrupt uterine contractility. Altered vascular architecture and abnormal angiogenesis contribute to increased blood flow. Additionally, local overexpression of cytokines and growth factors may impair endometrial hemostasis.[6] These pathophysiological insights provide the rationale for targeting ovarian steroid production as a therapeutic strategy.

Limitations of Current Therapies

Management of fibroid-associated HMB includes non-hormonal agents, hormonal therapies, interventional procedures, and surgery. However, each modality has limitations.

Non-hormonal agents such as tranexamic acid reduce menstrual blood loss by inhibiting fibrinolysis but do not affect fibroid size or underlying pathology.[7] They are useful for episodic symptom control but do not provide long-term disease modification.

Hormonal therapies including combined oral contraceptives and oral progestins can reduce bleeding but have limited impact on fibroid volume.[7,8] The levonorgestrel-releasing intrauterine system (LNG-IUS) is effective for reducing menstrual blood loss; however, expulsion rates are higher in women with distorted uterine cavities due to large or submucosal fibroids, and its efficacy may be reduced in this subgroup.[8]

Selective progesterone receptor modulators (SPRMs) demonstrated promising efficacy in reducing bleeding and fibroid size. However, concerns regarding rare but serious hepatotoxicity led to regulatory restrictions and withdrawal of certain agents in multiple countries.[9] This development significantly narrowed medical options for long-term management.

GnRH agonists effectively suppress ovarian steroidogenesis and reduce fibroid volume. However, their use is limited by an initial flare phenomenon, hypoestrogenic adverse effects such as vasomotor symptoms and vaginal dryness, and significant bone mineral density (BMD) loss with prolonged therapy.[10] Consequently, treatment duration is generally restricted to 3–6 months, primarily for preoperative optimization.

Surgical management, including myomectomy and hysterectomy, remains definitive. While hysterectomy

eliminates recurrence, it permanently removes fertility. Myomectomy preserves the uterus but carries risk of recurrence, estimated at 15–30% over time.[11] Surgical interventions are also associated with perioperative morbidity and may not be accessible to all patients in resource-variable settings.

These limitations underscore the unmet need for an effective, well-tolerated, long-term oral therapy that preserves bone health and fertility potential.

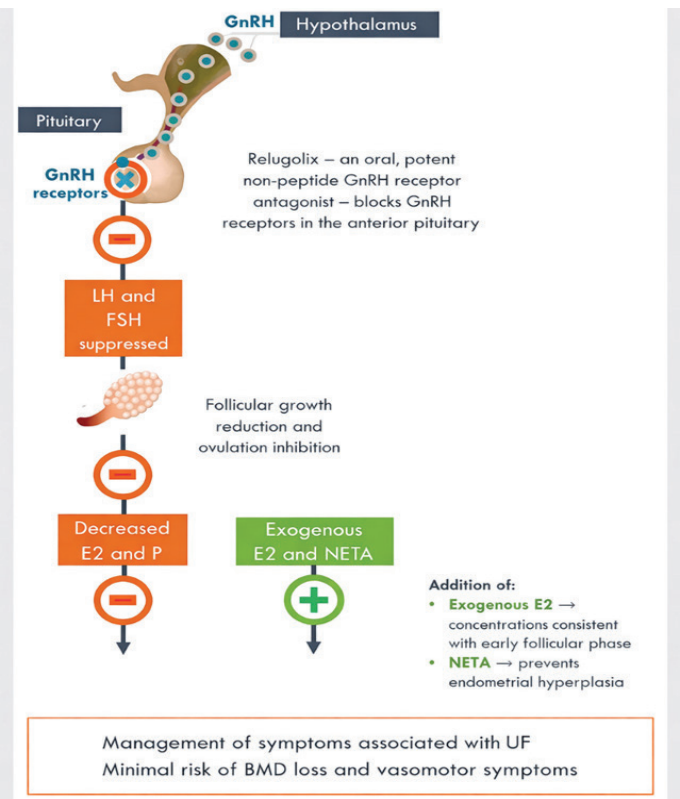
Role of GnRH Antagonists in Uterine Fibroids

GnRH antagonists directly block pituitary GnRH receptors, resulting in rapid suppression of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) secretion without the initial flare seen with GnRH agonists.[15] This mechanism leads to immediate reduction in ovarian estradiol and progesterone production.

Relugolix is a non-peptide, orally active GnRH receptor antagonist. It produces dose-dependent suppression of estradiol levels and has demonstrated efficacy in reducing fibroid-associated heavy menstrual bleeding.[15] Unlike injectable GnRH analogues, relugolix offers the convenience of oral administration and rapid reversibility upon discontinuation.

However, profound suppression of estrogen is associated with vasomotor symptoms and accelerated bone loss. Therefore, monotherapy with GnRH antagonists may not be suitable for extended use without protective strategies.

Role of Add back therapy



The estrogen threshold hypothesis proposes that maintaining circulating estradiol levels within a specific range (approximately 20–50 pg/mL) is sufficient to suppress fibroid growth while minimizing bone resorption and hypoestrogenic symptoms.[12] This concept forms the foundation of add-back therapy.

Relugolix combination therapy is a fixed-dose combination containing relugolix 40 mg, estradiol (as estradiol hemihydrate) 1 mg, and norethindrone acetate (NETA) 0.5 mg. Low-dose estradiol (1 mg) in the fixed-dose combination (FDC) maintains systemic estradiol levels within a therapeutic window (approximately 20-50 pg/mL). This mitigates hypoestrogenic side effects of GnRH antagonist-induced suppression, such as hot flushes, vasomotor symptoms, and clinically significant BMD loss. NETA (0.5 mg), a synthetic progestin, opposes the proliferative effects of estradiol on the endometrium, preventing unopposed estrogen-induced hyperplasia or endometrial thickening.[13,14] This balanced approach allows long-term therapy by combining efficacy with safety.

Regulatory Approval and Indian Context

Relugolix combination therapy received approval from the United States Food and Drug Administration and the European Medicines Agency in 2021 for management of heavy menstrual bleeding associated with uterine leiomyomas.[13] It has been incorporated into international treatment algorithms for symptomatic fibroids.[7,8]

In India, the fixed-dose combination of relugolix 40 mg with estradiol 1 mg and norethindrone acetate 0.5 mg received regulatory approval in 2025 for management of heavy menstrual bleeding associated with uterine leiomyomas in premenopausal women, with an approved duration of therapy up to 24 months.[14]

Given India's high anemia prevalence, delayed surgical access in certain regions, and cultural preference for uterine preservation, this therapy provides a valuable non-surgical alternative.

Dosage, Administration, and Duration

Relugolix combination therapy is administered as one oral tablet once daily, with or without food. Treatment should ideally begin as early as possible after the onset of menses but no later than seven days after menstruation has started. Pregnancy should be excluded prior to initiation, and hormonal contraceptives should be discontinued before starting therapy.

The recommended total duration of treatment is up to 24 months. If a dose is missed, it should be taken on the same day as soon as remembered, and the regular dosing schedule resumed the following day.

Contraindications and Precautions

Relugolix combination therapy is contraindicated in women

at high risk of arterial or venous thromboembolic disorders, during pregnancy, in those with known or suspected hormone-dependent malignancy, in patients with osteoporosis, significant hepatic impairment, undiagnosed abnormal uterine bleeding, or hypersensitivity to any component of the formulation.[13,14]

Caution is advised in women with risk factors for thromboembolism or bone loss. Baseline clinical assessment and periodic monitoring should be individualized based on patient risk profile.

Clinical Evidence

The efficacy and safety of relugolix combination therapy were established in the phase 3 LIBERTY 1 and LIBERTY 2 trials, randomized, double-blind, placebo-controlled studies evaluating women with fibroid-associated heavy menstrual bleeding.[15] Approximately 71–73% of women receiving relugolix combination therapy achieved the primary endpoint of menstrual blood loss reduction to less than 80 mL and at least 50% reduction from baseline, compared with 15–19% in the placebo group (p<0.0001).

Bleeding reduction occurred early and was sustained throughout treatment. Improvements were observed in hemoglobin levels, pain scores, and quality-of-life measures. The most common adverse events included mild vasomotor symptoms and headache. Importantly, add-back therapy preserved bone mineral density.

Extension studies demonstrated maintenance of efficacy and bone safety up to 24 months.[16,17] In the randomized withdrawal study within the LIBERTY program, sustained bleeding control was observed in a majority of women over extended follow-up, supporting long-term medical management.

Indian data from the REEMEMBER trial, a phase III randomized active-controlled study, further confirmed high rates of bleeding control and significant fibroid volume reduction in Indian women, with a favorable tolerability profile. Improvements in quality of life and anemia were consistent with global findings.[18]

Sr. No	Study Details	Primary Outcome (% Achievement)	Key Findings	Conclusion
1	LIBERTY 1 (N = 387) & 2 (N = 381)Phase III, randomized, double-blind, placebo-controlled 24 weeks Post-hoc Analysis (L 1 & 2) Fibroid location (N=509) & adenomyosis (N=111)	71–73% achieved target MBL reduction vs 15–19% placebo (p<0.0001)	Rapid and significant control of UF-associated HMB with improved pain and QoL Most common AE: VMS: 10.6% & AUB: 6.3% Mostly mild AEs; add-back therapy preserved BMD	Relugolix FDC demonstrated robust short-term efficacy in UF-HMB management Relugolix FDC is well tolerated with favourable long-term safety
			83.8% responders with adenomyosis subgroup Consistent bleeding reduction irrespective of fibroid location or co-existing adenomyosis	Efficacy of Relugolix FDC is independent of fibroid type and uterine pathology
2	LIBERTY Extension Study Open-label continuation 52 weeks (N=476)	87.7% sustained improvement in bleeding control;	Sustained symptom relief with stable bleeding patterns and preserved BMD	Relugolix FDC provides durable efficacy with bone safety up to 52 weeks
3	Randomised withdrawal Study (LIBERTY Program) Up to 104 weeks (N= 229)	Sustained achievement in 69.8–78.4%	Long-term maintenance of bleeding control with mostly mild AEs; add-back therapy preserved BMD	Supports long-term, uterus-preserving medical therapy with favourable long-term safety
4	REEMEMBER Trial – Indian Phase III, randomized, Assessor-blind, active-controlled study, 12 weeks (N=338)	95% achieved PBAC <75; 48.8% fibroid volume reduction (p<0.0001)	Most common AE: Pyrexia 8.3%, VMS: 5,9%, Mostly mild AEs; add-back therapy preserved BMD, Improved QoL	Confirms high efficacy and tolerability of Relugolix FDC in Indian women

Safety Profile

Bone mineral density is largely preserved during treatment with relugolix combination therapy due to the inclusion of estradiol and norethindrone acetate (15-18). The magnitude of BMD change over 24 months is modest and substantially lower than that observed with GnRH agonist monotherapy.

Common adverse events include mild hot flushes, headache, mood changes, and occasional nausea (15-18). The incidence of vasomotor symptoms is lower than with GnRH agonists. Because of the estrogen-progestin component, there is a theoretical risk of thromboembolic events similar to other combined hormonal therapies, and appropriate patient selection is essential.

Clinical Positioning in India

Relugolix combination therapy can be considered in women with moderate-to-severe heavy menstrual bleeding associated with uterine fibroid who desire uterine preservation, in those awaiting surgery, in perimenopausal women seeking symptom control, and in patients unfit or unwilling for surgery. It may also be valuable for preoperative optimization of anemia.

In the Indian setting, where anemia prevalence is high and access to minimally invasive surgery may vary geographically, an effective long-term oral therapy offers significant public health and individual patient benefits.

Conclusion

Relugolix combination therapy represents a significant advancement in the management of fibroid-associated heavy menstrual bleeding. By combining effective GnRH antagonism with physiologic add-back therapy, it achieves sustained bleeding control, preserves bone mineral density, and improves quality of life. In Indian clinical practice, it addresses an important unmet need by providing a long-term, uterus-preserving alternative between short-term hormonal suppression and definitive surgical intervention.

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Quiz Time- Estrogen Dependent Gynaecological Disorders

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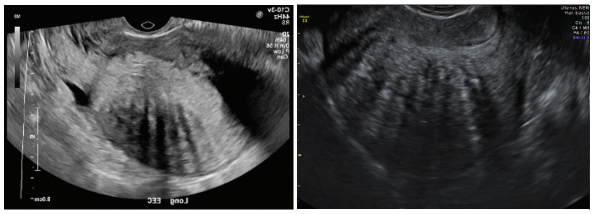
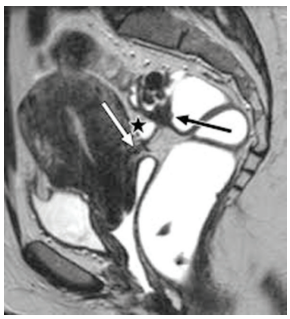
Q1. A 35-year-old woman presents with:

- Severe dysmenorrhea
- Heavy menstrual bleeding
- Secondary infertility



1. What is the most likely diagnosis?
2. Mention two ultrasound features supporting this diagnosis.
3. What imaging modality confirms the diagnosis?

Q2. MRI pelvis shows

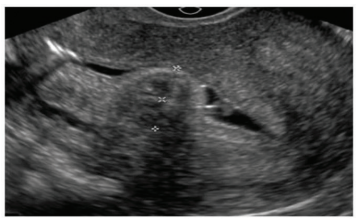


Clinical features include:

- Severe dyspareunia
- Dyschezia

1. What is the most likely diagnosis?
2. Which symptom is strongly associated with this lesion?
3. What is the definitive treatment?

Q3. Ultrasound shows



Patient history:

- Infertility for 3 years

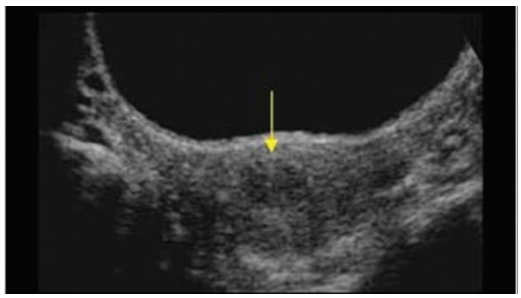
1. What type of fibroid is present?
2. Why does this lesion impair fertility?
3. What is the best surgical management?

Q4. MRI pelvis demonstrates:

1. What is the diagnosis?
2. Which MRI feature is most specific?
3. Name one medical treatment option

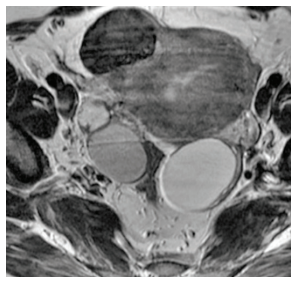
Q5. A 34-year-old woman planning IVF has on TVS has

- 3 cm intramural fibroid
- No distortion of the endometrial cavity



1. Does this fibroid significantly affect fertility?
2. What factors determine need for surgery?
3. What management is recommended?

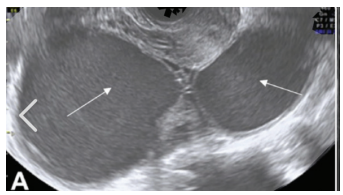
Q6. MRI shows



1. What is the diagnosis?
2. What causes the T2 shading sign?
3. What fertility-preserving treatment can be offered?

Q7. Ultrasound demonstrates

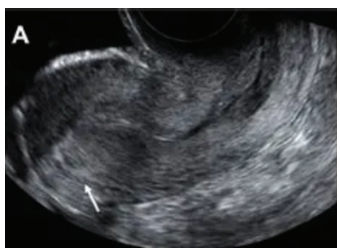
Patient symptoms:



- Chronic pelvic pain
- Infertility

1. Which disease is most likely responsible?
2. What stage of disease does this usually indicate?
3. Which imaging modality best maps disease extent?

Q8. Transvaginal ultrasound shows:



1. What is the diagnosis?
2. What symptom commonly occurs?
3. Name one fertility-preserving treatment.

Q9. MRI pelvis demonstrates

1. What gastrointestinal symptom is common?
2. Which specialist team should manage such cases?
3. Name one surgical technique used.

Q10. A 37-year-old woman with infertility has:

1. How can this fibroid affect fertility?
2. What is recommended before ART?
3. What surgical approach is preferred?

Answer Key

Q1. Diagnosis: Adenomyosis

Ultrasound features:

- Heterogeneous myometrium
- Fan-shaped shadowing
- Myometrial cysts
- Globular uterus

Confirmation: MRI pelvis

Q2. Diagnosis: Deep infiltrating endometriosis

Associated symptom: Dyschezia

Management: Laparoscopic excision surgery

Q3. Diagnosis: Submucosal fibroid

Effect on fertility:

- Distorts uterine cavity
- Impairs implantation

Management: Hysteroscopic myomectomy

Q4. Diagnosis: Adenomyosis

Specific MRI sign: Junctional zone thickness >12 mm

Treatment options:

- LNG-IUS
- GnRH agonists
- Dienogest

Q5. Small intramural fibroids may not significantly affect fertility.

Management: Expectant unless cavity distortion or size >4–5 cm.

Q6. Diagnosis: Ovarian endometrioma

Cause of T2 shading:

- Chronic hemorrhage with high iron concentration

Management: Laparoscopic cystectomy

Q7. Diagnosis: Severe endometriosis

Stage: Stage III–IV

Best imaging: MRI pelvis

Q8. Diagnosis: Adenomyosis

Common symptom: Dysmenorrhea

Treatment options:

- LNG-IUS
- Hormonal therapy

Q9. Common symptom: Painful defecation during menstruation

Management: Multidisciplinary endometriosis surgery

Possible procedures:

- Bowel shaving
- Disc resection
- Segmental bowel resection

Q10. Effect on fertility:

- Impaired implantation
- Distortion of uterine cavity

Management: Myomectomy before ART

Preferred approach: Laparoscopic myomectomy

AOGD Clinical Meet from UCMS & GTB Hospital held on 27th February 2026

"Breaking the bullae in unresponsive Pemphigoid Gestationis – A rare case"

Rajeshwari kumari, Sandhya Jain, Rachna Agarwal, Anshuja Singla, Bhanupriya, Upasana, Priya

Background

Pemphigoid gestationis (PG) is a rare autoimmune vesiculobullous disorder unique to pregnancy (1 in 50000-60000 pregnancies), most commonly managed with systemic corticosteroids. However, a subset of patients may exhibit steroid resistance, pose therapeutic challenges and increase the risk of adverse maternal and fetal outcomes.

Case Presentation

We report a case of a 28 years old primigravida who presented at 30 weeks of gestation with severe pruritus and widespread erythematous plaques progressing to tense bullae, predominantly involving the periumbilical region, trunk extremities and face. Despite adequate doses of systemic corticosteroids, the patient showed minimal clinical improvement, with persistent new lesions formation. Skin histopathological examination revealed a subepidermal blister with eosinophilic infiltrate, and direct immunofluorescence demonstrated linear C3 deposition along the basement membrane zone, confirming the diagnosis of pemphigoid gestationis. Owing to steroid resistance, oral cyclosporine was initiated under close maternal and fetal monitoring, resulting in rapid disease control and significant symptomatic relief. Strict feto-maternal surveillance was done but patient developed oligohydramnios with FGR. Emergency LSCS was done for fetal distress at 36 weeks, with no evidence of cutaneous involvement in neonate. On follow up patient was improving with no postpartum flareup and discharged on tapering doses of steroids and cyclosporin and was advised to follow up regularly in dermatology OPD.

Discussion

Steroid-resistant pemphigoid gestationis is a rare, but challenging variant of this autoimmune blistering disorder of pregnancy. Failure to respond to adequate systemic corticosteroid therapy necessitates alternative immunosuppressive strategies while carefully balancing maternal disease control and fetal safety. Literature search for steroid resistant PG on Goggle engine showed only 8 case reports. Most of them had presented in 2nd trimester and late trimester. Most of them treated with various immunosuppressants, only one treated with cyclosporin. All the cases including ours had favourable outcomes. So, early identification of resistance and timely initiation of second-line agents, such as cyclosporine or other steroid-sparing therapies, can improve maternal outcomes and potentially reduce obstetric complications. Multidisciplinary management is essential to optimize both maternal and fetal prognosis.

Conclusion

Steroid-resistant pemphigoid gestationis is rare but can be effectively managed with immunosuppressants (cyclosporine) when conventional therapy fails. This case highlights cyclosporine as a viable second-line treatment option in steroid resistant cases, with favorable maternal and fetal outcomes when used judiciously under multidisciplinary supervision.

Keywords

Pemphigoid gestationis, steroid resistance, cyclosporine, autoimmune blistering disease, pregnancy dermatoses

Key message

Pemphigoid Gestationis is a rare but must know differential for dermatosis in pregnancy. It's first line treatment is steroids & if resistant immunosuppressants are added to bring dramatic relief in symptomolgy. Additional materno-fetal surveillance is needed for good fetal outcomes.

Uterine PEComa: Sinister pathology masquerading as uterine polyp

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Department of Obstetrics & Gynaecology

Senior resident[1], Associate professor[2], Professor[3], Dir Professor[4], Senior Consultant[5]

Background

Perivascular epithelioid cell tumors (PEComas) are rare mesenchymal neoplasms characterized by distinctive perivascular epithelioid cells exhibiting dual melanocytic and smooth muscle differentiation. The uterus represents the most common gynecologic site; however, uterine PEComas remain exceptionally uncommon and often mimic more prevalent uterine malignancies clinically and radiologically. Their biological behavior ranges from benign to overtly malignant, necessitating definitive histopathological and immunohistochemical evaluation for diagnosis.

Case

A 51-year-old multiparous, postmenopausal woman presented with intermittent vaginal bleeding for two months, passage of clots, and lower abdominal pain with a sensation of heaviness. On speculum examination, a large, vascular, friable mass was seen protruding through the cervical canal, with intact cervical lips.

Ultrasonography revealed a hypoechoic lesion in the lower endometrial cavity extending into the cervix and vagina with mild vascularity. MRI pelvis demonstrated a large lesion occupying the endometrial cavity with >50% myometrial invasion and thinning of the residual myometrium, along with grade 3 left hydroureteronephrosis. PET-CT showed an FDG-avid heterogeneous lesion involving the endometrium and myometrium, extending toward the left adnexa, with a small FDG-avid right-sided deposit.

Biopsy revealed an aggressive non-epithelial tumor with strong vimentin positivity. On extended immunohistochemical panel, tumor cells were positive for HMB-45, Melan-A, and vimentin, and negative for cytokeratin—confirming the diagnosis of PEComa.

Patient underwent staging laparotomy with total abdominal hysterectomy, bilateral salpingo-oophorectomy, right pelvic lymphadenectomy, excision of left parametrial deposit, and infracolic omentectomy. The postoperative course was uneventful. Final histopathology confirmed malignant uterine PEComa, pathological stage pT2aN0M0. She was advised mTOR inhibitors adjuvant chemotherapy but due to non-availability she was given gemcitabine and docetaxel based 6 cycles chemotherapy. Patient is on regular 3 monthly follow ups and doing well.

Discussion

Uterine PEComa is an extremely rare mesenchymal tumor with variable malignant potential and significant diagnostic challenges. A review of the literature reveals fewer than 100 reported cases, with the uterus being the most common gynecological site of involvement. The typical age group affected is between 40 and 55 years, and the most common presenting symptom is postmenopausal bleeding, often leading to an initial suspicion of endometrial carcinoma. Radiological and histopathological findings frequently overlap with leiomyoma, leiomyosarcoma, and endometrial malignancy, making diagnosis difficult. Approximately 60% of uterine PEComas are benign, while 15–20% demonstrate malignant behavior, as seen in our case. Definitive diagnosis requires extended immunohistochemistry showing dual melanocytic and smooth muscle differentiation. Risk stratification using Folpe and modified Folpe criteria is essential for prognostication and management. Complete surgical excision remains the cornerstone of treatment, and targeted therapy with mTOR inhibitors has shown promising outcomes in malignant cases. This case highlights the importance of maintaining a high index of suspicion and a multidisciplinary approach to ensure timely diagnosis and optimal patient care.

Conclusion

Uterine PEComa is a rare diagnostic entity that can masquerade as more common uterine malignancies in postmenopausal women. Accurate diagnosis relies on a high index of suspicion and comprehensive immunohistochemical profiling. Early recognition and appropriate surgical management are crucial in optimizing outcomes in these rare but potentially aggressive tumors.

"High index of suspicion and comprehensive immunohistochemistry are essential for the accurate diagnosis of rare uterine tumors. A multidisciplinary approach, along with consideration of uncommon pathologies in atypical presentations, enables timely targeted therapy and ultimately improves patient survival."

Placenta to Platelets: Regenerative Biologics in Preventing Cesarean Surgical Site Infections

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Background:

Cesarean section (CS) rates have increased globally; reaching approximately 21%, making it one of the most commonly performed surgical procedures worldwide. Surgical site infection (SSI) remains a significant postoperative complication, occurring in 3–15% of cases and contributing to maternal morbidity, prolonged hospitalization, increased healthcare costs, and psychological stress. SSIs are classified as superficial incisional (skin and subcutaneous tissue), deep incisional (muscle and fascia), and organ/space infections. Despite improvements in surgical techniques and antibiotic prophylaxis, there is no consensus regarding an optimal wound dressing method. We conducted 2 randomized clinical trials utilizing biological therapies such as autologous human amniotic membrane (HAM) patch dressing and platelet-rich plasma (PRP) injection at the Cesarean surgical sites. These approaches have emerged as the promising modalities due to their regenerative, anti-inflammatory, antimicrobial, and angiogenic properties that enhance wound healing.

Aim

To evaluate the effectiveness of autologous HAM patch dressing and PRP injection in reducing surgical site infections and improving wound healing following cesarean delivery.

Methods

Two prospective randomized controlled trials were conducted, including a total of 144 women undergoing cesarean delivery at the Department of Obstetrics and Gynaecology, University College of Medical Sciences, GTB Hospital.In the HAM study, 100 women were randomized into intervention (n=50) and control (n=50) groups. The HAM patch dressing (10 × 2 cm) was harvested from the patient's placenta during cesarean section, washed with normal saline, and applied over the suture line before skin closure in the intervention group. The control group received conventional dressing.In the PRP study, 44 women were randomized into case (n=22) and control (n=22) groups. In the intervention group, 2.5 mL of autologous PRP, prepared by centrifugation of 10 mL of the patient's blood, was injected subcutaneously along the incision line during wound closure.Inclusion criteria included women ≥28 weeks gestation undergoing cesarean delivery. Exclusion criteria were thrombocytopenia, prolonged rupture of membranes (>18 hours), viral infections (Hepatitis B, Hepatitis C, HIV, syphilis), local skin infection, and febrile illness.

Wound healing was assessed using the REEDA score (Redness, Edema, Ecchymosis, Discharge, Approximation) on postoperative days 3, 8 (or 10), and 42. Primary outcomes included SSI rates and REEDA score comparison between intervention and control groups.

Results

In the HAM study, SSI occurred in 6% of the intervention group compared to 24% in controls, demonstrating a significant reduction. In the PRP study, SSI rates were 4.55% in the PRP group compared to 31.82% in controls, also statistically significant.REEDA scores were significantly lower in both HAM and PRP groups at all evaluated time points (days 3, 8/10, and 42), indicating enhanced wound healing.

Common comorbidities among patients who developed SSI included insulin-treated gestational diabetes mellitus (n=7), corrected severe anemia (n=7), previous ≥2 cesarean sections (n=2), PROM >12 hours (n=2), non-progress of labor (n=3), and obesity (n=2).

Conclusion

Autologous HAM patch dressing and PRP injection, significantly reduce SSI rates and accelerate wound healing after cesarean delivery. These biologically active, patient-derived, and cost-effective interventions show strong potential as adjunctive strategies in routine obstetric surgical practice.

Govt. of India has launched a vaccination campaign against Human Papilloma Virus (HPV) on 28th February 2026. This HPV vaccination for the girls of age 14-15 years provides significant preventive protection against this Cervical Cancer. The campaign aims to provide one dose of HPV Vaccine to all eligible girls during the following period of 3 months. The vaccination will be conducted at Government Health Facilities by a trained vaccination team.

Events Held February 2026

Public Awareness Camp conducted by Breast and Cervical Cancer Awareness & Prevention subcommittee on 1st February, 2026, at GTB Hospital

Public Awareness Program on the occasion of World Cancer Day conducted by Dept. of Obst. & Gynae, LHMC & SSK Hospital Under the aegis of AOGD on 4th February 2026

CME on Vulval Disorders conducted by Oncology committee AOGD and DGF- West on 6th February at Radisson Blue Hotel

A Practical training course on Vulvo vaginal module- V insight 2026 conducted by the Dept. of Obst. & Gynaecology, UCMS and GTB hospital under the aegis of AOGD and FOGSI Oncology Committee on 13.2.2026

Masterclass on Vulvar lesions conducted by Dept. of Obst. & Gynae, AIIMS Delhi under the aegis of ISCCP, FOGSI & Oncology subcommittee of AOGD on 14th February, 2026.

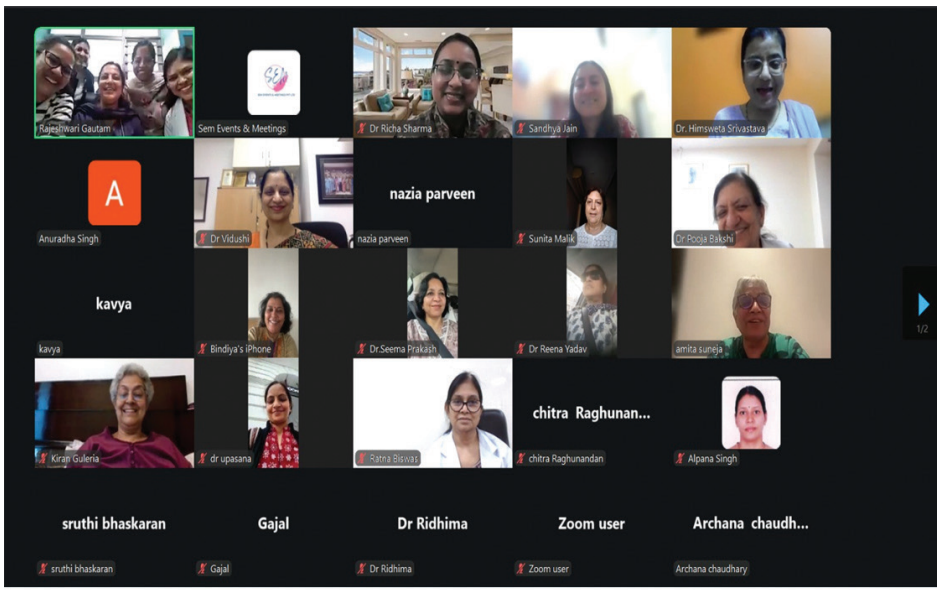
Webinar on Abnormal uterine bleeding and ten rules of adolescent health care conducted by Adolescent Health Subcommittee AOGD and DGF on 14th February, 2026 at Maya Muni Ram Charitable Hospital

Anaemia Detection & Awareness camp on the occasion of World Anemia Awareness Day conducted by Community Health and Public Awareness Sub Committee AOGD in association with IAP Delhi on 16th February at Janki Devi women's college

Webinar on PPH conducted by

Safe Motherhood Committee, on 20th February 2026

The AOGD Monthly Clinical Meeting (virtual) conducted by the
Department of Obst & Gynae, Dr UCMS & GTB Hospital on 27th February, 2026



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