HAPPY REPUBLIC Day

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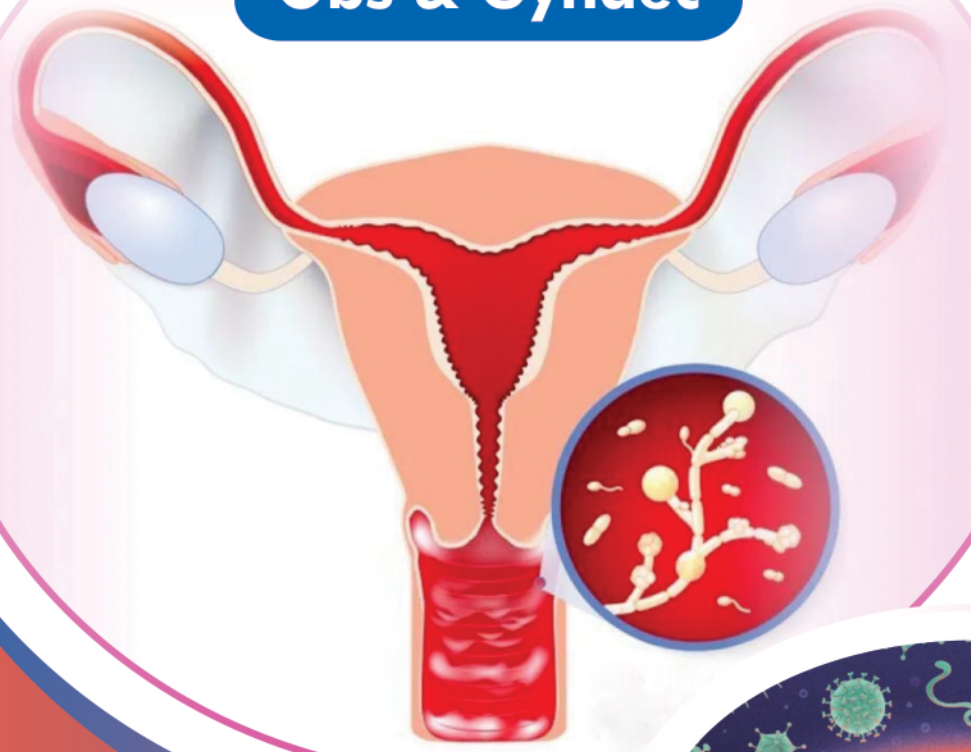
Theme :

Infection in Obs & Gynaec



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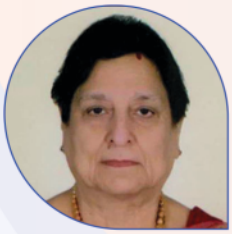

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PATRON MESSAGE

Infection in Obstetrics & Gynaecology : A Call for Action

Dear Esteemed Colleagues,

Infections in obstetrics and gynecology are largely iatrogenic, and the stark reality is that maternal sepsis accounts for 20-24% of maternal deaths. This is a number we simply cannot afford to lose. The time has come for us to rise to the occasion and take decisive action.

The power to change this lies in our hands. By adopting and practicing sepsis bundles rigorously, we can significantly reduce this burden. Let us make it our mission to spread awareness, share knowledge, and teach the importance of timely intervention to every healthcare provider.

I urge each one of you to commit to practicing prophylactic antibiotics wisely and no more. Overuse and misuse only add to the problem. The focus should be on prevention, early recognition, and prompt management.

Let this bulletin serve as a beacon of knowledge, guiding us towards a future free from preventable maternal deaths. Together, we can make a difference and ensure safer pregnancies and healthier mothers.

With warm regards,
Dr. Sharda Jain
Patron
North India Gynecologist Forum

PRESIDENTIAL MESSAGE

We wish you & family a very Happy, Peaceful & Prosperous New Year 2025.

It is great pleasure to release North India Gynaec Forum 5th Bulletin on eve of our National Republic Day 26th January. We convey our heartiest greetings & congratulations for Republic Day, which stands and speaks for equal rights and opportunities to all citizen of our country. May we all work together to make our country stronger and happier.

The theme of 5th Issue of NIGF Bulletin is Infection in Obstetrics & Gynecology. While 20th century has imparted knowledge and means for identifying, preventing and treating infectious disease, emerging infections and resistance to conventional antibiotics is one of the major challenges of current century.

Every New Issue of NIGF Bulletin has new sections. **Inclusion of Review Article and Research Abstract on the theme issue are innovative section in present bulletin.**

We are honored to have review articles by eminent Author Dr Mala Arora et al on emerging understanding of pre and pro biotic in infectious disease particularly in Ob & Gynecology. We express hearty gratitude for their valuable contribution.

Dr Arun Arora et al has systemically analyzed changing infectious challenges across the timeline in his review article. We believe that both Review article will be highly interesting & informative for readers.

In this NIGF bulletin editors have created new section on **research abstract on theme of Infections in Ob Gyn.** Best Abstract presenter will have special presence in our upcoming academic conferences. We look forward to increasing contribution from our teaching faculties as well research-oriented clinicians for this section.

In Main Section editorial board have tried to include all important infections in section of Obstetrics & Gynecology. We are highly thankful to our authors who have taken their precious time to contribute updated and high-quality articles on various subjects.

It is always pleasure to compile North India Gynaec Forum activities -virtual as well physical series, which gives us pause as well pleasure to look back as well thoughts for way forward.

At this moment I also thank every one for getting me elected for post of ICOG Vice Chair elect 2025 and my subsequent inning as ICOG Office bearer.

Once again, we wish a happy enjoyable learning and a great New Year Ahead.

Always Yours

Dr. Sadhana Gupta
President North India Gyanec Forum
ICOG Vice Chair Elect 2025

EDITOR'S NOTE

It is with great pride and enthusiasm that we present this 5th bulletin of NIGF dedicated to Infections in Obstetrics and Gynecology, a critical area of study that continues to challenge clinicians and researchers alike due to delayed diagnosis, lack of awareness and stigmas surrounding reproductive health. Infections in gynaecology and obstetrics are not just a clinical concern but a society issue that impacts the broader framework of women's health. Infections significantly impact maternal, fetal, and reproductive outcomes, demanding a thorough understanding of their pathophysiology, diagnosis, and management.

As advancements in medicine redefine clinical care, the understanding of infections has deepened, introducing new dimensions to their prevention and treatment. This bulletin addresses a wide spectrum of infections, ranging from bacterial vaginosis and urinary tract infections (UTIs) in pregnancy to puerperal sepsis and septic shock, conditions that require urgent recognition and intervention. The inclusion of special review article like prebiotics and probiotics in reproductive health reflects a shift towards harnessing the power of the microbiome in preventing infections. There has been a definite shift in the types of infection in recent years and this has been highlighted in the review article on changing trends in infection in Obstetrics and Gynaecology.

Pregnancy-specific infections such as TORCH infections, hepatitis B, and HIV demand special attention due to their profound implications for both the mother and foetus. This bulletin not only provides the latest guidelines for managing these infections but also highlights the importance of preventive measures like vaccination and early screening. Rare but critical conditions such as uncommon pneumonia complicating the peripartum period remind us of the need for multidisciplinary approaches in managing complex cases.

In gynecological practice, infections such as vaginal discharge, recurrent urinary tract infections, and female genital tuberculosis require meticulous evaluation and personalized management strategies. Furthermore, discussions on urogenital infections in postmenopausal women and vulvar pruritus address conditions that are often overlooked yet significantly impact quality of life.

This bulletin serves as a testament to the collaborative efforts of experts in the field, combining evidence-based recommendations with practical insights. It underscores the importance of staying updated with evolving research to improve patient care.

We hope this bulletin will become an invaluable resource for obstetricians, gynaecologists, and healthcare professionals striving to advance the standards of care in women's health. Together, let us continue our journey toward excellence, compassion, and innovation.

Dr. Meenakshi B Chauhan
Guest Editor

SECREATARY GENERAL MESSAGE

Wish everyone very happy and healthy new year.

It gives me a great pleasure that NIGF is publishing 5th bulletin on Infections in Obstetrics and Gyanecology. The Infections during pregnancy poses a great problem in diagnosis as well as the management. The most common infection during pregnancy are bacterial vaginosis, which may lead to increased incidence of preterm labour. The other infection for example - asymptomatic bacteruria may also affect pregnancy outcomes. The infection that can cause problems during pregnancy includes, chickenpox, CMV, group B streptococcus, hepatitis B, HCV, infection, TB, HIV, zika virus as well as TORCH infections.

Repeated UTI also has dilemma in diagnosis and management. Menopausal women have varied challenges in uro-genital infections.

We have eminent clinicians who have written about puerperal sepsis and septic shock. There are emphasis on how to deal with vaginal discharge STI s as well as vulvar pruritus. I am sure all the members of NIGF will be immensely benefitted from the elaborate documentation regarding the burning topics in obstetrics.

This bulletin will be adding to the already existing knowledge on the addressed subjects. Hope this bulletin has wide acceptance and will be highly appreciated by one and all. Such focused bulletins are really desirable and will always be in great demand.

Long live NIGF!

Dr. Mala Srivastava
Secretary General, NIGF

PRESIDENT ELECT MESSAGE

Dear Colleagues,

I congratulate Dr Sadhana Gupta to bring out this edition of our medical newsletter, focusing on a critical aspect of obstetrics and gynecology—**infection prevention, diagnosis, and management.**

Infections remain a significant concern in OB-GYN practice, impacting maternal and fetal outcomes, fertility, and overall reproductive health. As healthcare providers, we are responsible for staying informed about the latest advancements in infection control, antimicrobial stewardship, and preventive strategies to ensure the best possible care for our patients.

Hormonal changes during pregnancy, menopause, and contraceptive use can influence susceptibility to infections, making a thorough understanding of vaginal microbiota essential.

This issue will explore key topics, including:

- The role of early detection and treatment of **sexually transmitted infections (STIs)** in preventing complications such as infertility and pregnancy loss.
- Strategies for managing **perinatal infections** to safeguard maternal and neonatal health.
- Best practices for **infection prevention in surgical procedures** and postpartum care.
- The growing challenge of **antimicrobial resistance** and the importance of judicious antibiotic use in our field.
- 2 review article on very important topic

I encourage all members to engage with the insights shared by our esteemed contributors and to implement evidence-based practices in their daily work. Together, we can enhance patient safety, improve outcomes, and uphold the highest standards of care in women's health.

Thank you for your commitment to excellence in OB-GYN practice. I look forward to your feedback and continued collaboration in addressing this vital issue.

Sincerely yours

Dr. Ragini Agrawal
President Elect

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Dr. Isha Wadhawan
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Pre and Probiotic for Reproductive Health: The new therapy on the horizon

General Consideration

The upper female genital tract microbiome has recently been isolated and the endometrium harbours a lactobacilli dominated, low biomass, microbiome that maintains endometrial health. Endometrial health is essential for reproduction and beyond. Imbalances or dysfunctions in the endometrial microbiome can lead to various gynecological conditions, such as endometriosis, uterine polyps, Polycystic Ovary Syndrome (PCOS), chronic endometritis, Recurrent Implantation Failure (RIF), Recurrent Pregnancy Loss (RPL) and even endometrial hyperplasia and cancer. Increasing interest has emerged in maintaining a healthy lactobacillus dominated endometrial microbiome. The potential role of prebiotics and probiotics in maintaining or improving endometrial health, preventing vaginal dysbiosis is now well recognised. The gut plays a very important role in autoimmunity and in women with autoimmune disorders it is recommended to fix the leaky gut, more so due to its interplay with the reproductive system[1].

The human microbiome

The human microbiome refers to the trillions of microorganisms, including bacteria, viruses, fungi, and archaea, that inhabit various parts of the human body, such as the gut, skin, mouth, and reproductive organs. These microbes play a crucial role in maintaining health and homeostasis[2]. The gut microbiome, in particular, is integral to digestion, immune system regulation, and the synthesis of essential vitamins[2]. The balance of the microbiome is essential for preventing disease; dysbiosis, or microbial imbalance, has been linked to a wide range of conditions, including inflammatory bowel diseases (IBD), obesity, diabetes, implantation failure, and even neurodegenerative disorders[3].

The microbiome influences metabolic processes and protects against pathogenic microbes by outcompeting them for resources. Furthermore, emerging research suggests that the microbiome plays a role in reproductive health, influencing conditions like endometriosis and bacterial vaginosis[4]. The composition of the microbiome can be affected by factors such as diet, antibiotic use, sexual activity, personal hygiene and environmental exposures. Understanding the human microbiome offers new possibilities for therapeutic interventions, including probiotics, prebiotics, and microbiome-based treatments, to improve health outcomes across various medical fields.

Human microbiome project was launched in 2007 by Baylor College of Medicine in Houston USA. Their aim was to study the normal human commensals in healthy individuals first and later study them in specific diseases. It is an international project that aims to be able to manipulate and modulate human microbiome to improve health and make us understand that rapid and marked changes in lifestyle are not only affecting health of biosphere but causing changes in our microbiome ecology and contributing to our own normal physiology and diseases predisposition[5].

The reproductive microbiome

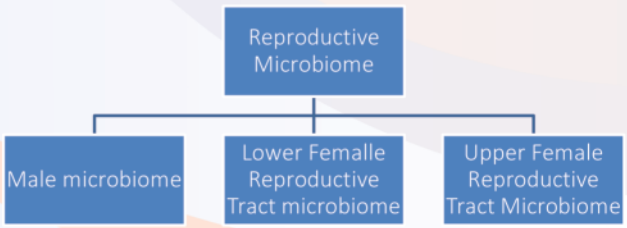
The reproductive microbiome encompasses the

microbial communities of the upper and lower female reproductive tract as well as the male reproductive tract. In females, the microbiome varies between the lower reproductive tract (vagina and cervix) and the upper reproductive tract (uterus, fallopian tubes, and ovaries). The vaginal microbiome is dominated by Lactobacillus species, which produce lactic acid to maintain an acidic pH, inhibiting the growth of pathogens and supporting reproductive health[4]. Dysbiosis in the vaginal microbiome, characterized by a reduction in Lactobacillus dominance, is associated with bacterial vaginosis, preterm birth, and increased susceptibility to sexually transmitted infections (STIs)[4]. The upper reproductive tract, once considered sterile, is now known to harbor a low-biomass microbiome with potential implications for conditions like endometriosis and infertility[6].

In males, the reproductive microbiome includes bacteria found in the seminal fluid, which is comprised of fluid from the prostate, seminal vesicles, testes, and epididymisn7. The male microbiome influences fertility, sperm quality, and immune defence mechanisms[7]. For instance, dysbiosis in the male reproductive tract has been linked to subfertility and prostatitis[7].

Both the male and female reproductive microbiomes are influenced by factors such as hormonal fluctuations, sexual activity, personal hygiene habits and antibiotic use[4,6,7]. Emerging research highlights the interplay between these microbiomes and their potential contributions to reproductive outcomes[6,7]. Understanding these microbial ecosystems may pave the way for novel interventions targeting fertility and reproductive health.

Gut microbiome interacts with hormones (estrogen, progesterone) and any imbalances may lead to pregnancy complications, as well as PCOD, diabetes, endometriosis, and cancer. However, evidence of this linkage is currently limited. Chronic endometritis, endometrial polyps & hyperplasia, endometriosis, and endometrial cancer may have its roots to a disrupted female genital tract microbiome[8].



The organisms of the lower female reproductive tract

The lower female reproductive tract, which includes the vagina and cervix, hosts a diverse and dynamic microbial community that plays a critical role in reproductive and overall health[4]. The dominant microorganisms in the vaginal microbiome are Lactobacillus species, particularly

- Lactobacillus crispatus,
- Lactobacillus iners,
- Lactobacillus gasseri,
- Lactobacillus jensenii[4].

These species are known for producing lactic acid, which maintains an acidic pH (typically below 4.5) that inhibits the growth of pathogenic bacteria and supports a healthy microbial balance. Lactobacilli also synthesize hydrogen peroxide (H2O2) that acts as a bacteriostatic agent. L jensenii and L vaginalis produce the highest levels of H2O2.

In addition to Lactobacillus, the vaginal microbiome also includes anaerobic bacteria such as Gardnerella vaginalis, Atopobium vaginae, and Prevotella species 4. While these are part of a normal microbiome, their overgrowth or dominance is associated with bacterial vaginosis, a common condition linked to reproductive health complications e.g. second trimester pregnancy loss, preterm prelabour rupture of membranes (PPROM) as well as Preterm labour (PTL)[4,9,10]. Other members of the vaginal microbiome include Mobiluncus species and Mycoplasma hominis, which can also become pathogenic under conditions of dysbiosis[9].

The vaginal microbiome composition varies due to factors such as age, hormonal changes, menstrual cycle, sexual activity, and hygiene practices[10]. A healthy microbiome, dominated by Lactobacillus spp., is essential for protecting against sexually transmitted infections (STIs) and supporting fertility and pregnancy outcomes[10]. It also acts as a protective barrier to maintain the sanctity of the uterine microbiome i.e. Upper Female Reproductive Tract Microbiome[4].

Diagnostic tests for dysbiosis

Vaginal culture is not a suitable test to diagnose dysbiosis as many of these organisms are anaerobic and will not grow in aerobic culture conditions. The following tests may be helpful 10.

- **Vaginal pH :** testing the pH of the vaginal secretions that accumulate on the blade of the Cusco or Sims speculum, with a strip of pH paper is the easiest way to diagnose dysbiosis. The normal pH of these secretions should be less than 4. A value of 6 and above suggests dysbiosis.

- **Microscopic examination of vaginal secretions:** demonstration of clue cells is diagnostic of bacterial vaginosis (BV).
- **Nugents Scoring:** Grams-stained smears of vaginal secretions are scored for presence of different kinds of bacteria. A score of 7-10 suggests bacterial vaginosis.
- **Amsell Criterea:** a set of four criteria of which 3 should be positive to diagnose BV. These are
  1. Thin white /yellow homogenous discharge
  2. Vaginal pH of >4.5
  3. Presence of clue cells
  4. Release of fishy odour on adding 10% potassium hydroxide (KOH) to a drop of vaginal secretion on a slide.
- Identification of bacterial composition by PCR testing. This is now the gold standard and will also direct appropriate treatment i.e. in presence of pathogenic bacteria e.g. E coli will need to be treated with antibiotics plus probiotics. In overgrowth of anaerobic species- metronidazole is indicated and in deficiency of lactobacilli – probiotics would be treatment of choice[10].

The organisms of the upper female reproductive tract

The upper female reproductive tract, which includes the uterus, fallopian tubes, and ovaries, was historically considered sterile. Recent advancements in next-generation sequencing have revealed a low-biomass microbiome in these regions, comprising of diverse microbial populations. This microbiome plays a significant role in reproductive health and disease.

1. **Dominant Microorganisms:** Studies indicate that the upper reproductive tract harbors species commonly found in the vaginal microbiome, such as Lactobacillus spp., albeit in lower abundance[11]. Among these, Lactobacillus iners and Lactobacillus crispatus are frequently detected and are thought to help maintain immune homeostasis and a protective microenvironment[12].
2. **Other Bacteria:** The upper reproductive tract also hosts other bacteria such as Gardnerella vaginalis, Prevotella spp., and Atopobium vaginae. While these species are often associated with dysbiosis in the vaginal microbiome, their presence in the upper tract is linked to conditions like pelvic inflammatory disease (PID) and endometritis[12].
3. **Endometrial Microbiota:** The endometrium (lining of the uterus) often harbors a unique microbiota dominated by Lactobacillus spp. or anaerobic bacteria like Bacteroides, Clostridium, and Streptococcus spp. These bacterial communities influence implantation success and pregnancy outcomes[11,13].
4. **Emerging Findings:** Fusobacterium nucleatum has been implicated in adverse pregnancy outcomes, including preterm birth[13]. Escherichia coli and Staphylococcus aureus have been detected in association with chronic endometritis and infertility[6,11].

Dysbiosis and Pathology

Dysbiosis in the upper reproductive tract microbiome has been linked to endometriosis, chronic endometritis, recurrent miscarriage, and unexplained infertility[6,11,13]. It may also play a role in gynecological cancers, though research is still emerging in this area.

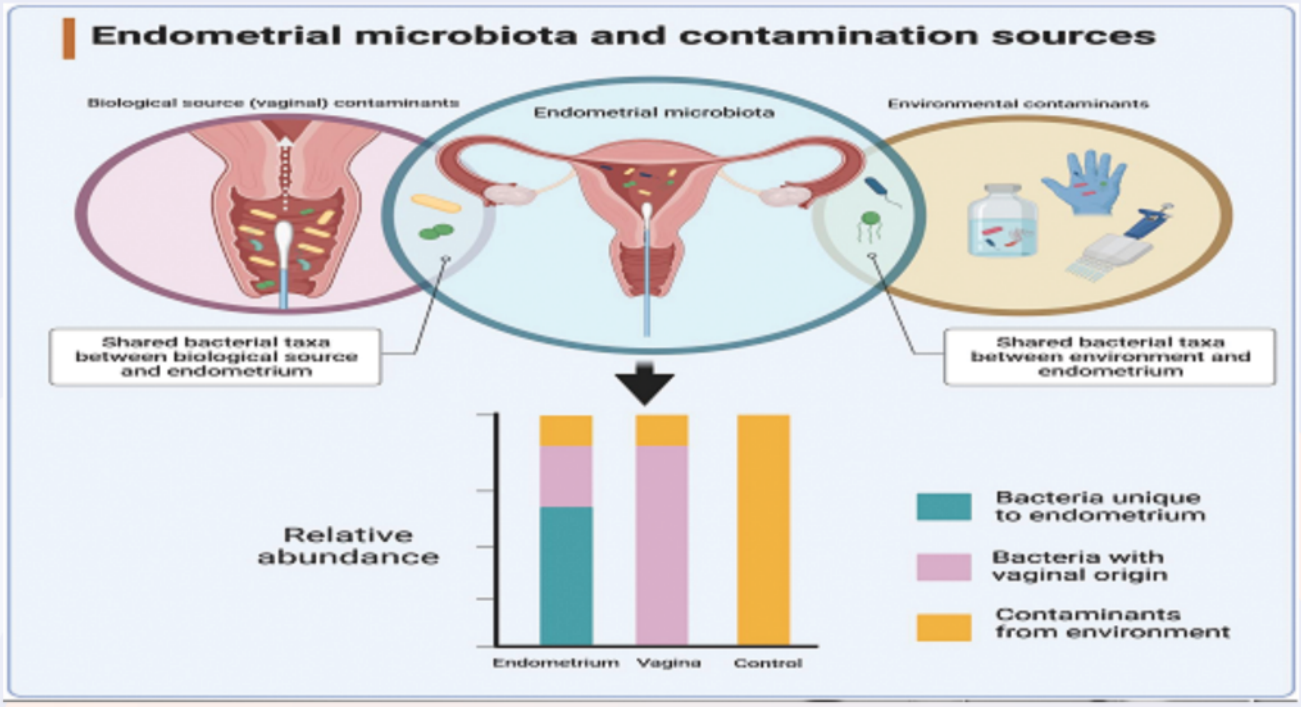
Significance in Reproductive Health

- **Fertility :** A balanced microbiome in the upper reproductive tract supports endometrial receptivity, implantation, and embryo development. Dysbiosis is associated with infertility and poor in vitro fertilization (IVF) outcomes[6,11].
- **Pregnancy Outcomes:** A healthy microbial composition reduces the risks of miscarriage, preterm birth, and other complications. There is five times higher prevalence of aerobic vaginitis in women with recurrent pregnancy loss. Studies have noted prevotella, gardenerella vaginalis, fannyhessea vaginae among most found in bacterial vaginosis in these women leading to adverse pregnancy outcomes. Presence of Streptococcus and urealpasma in the endometrium have been linked to recurrent pregnancy loss[14].
- Immune Regulation: The upper tract microbiome interacts with host immune responses, maintaining an anti-inflammatory environment critical for conception and pregnancy[13].

Diagnosis of Dysbiosis

Uterine microbiome is of very low biomass. Sampling methods are fraught with risk of contamination. Hence its study is only possible by meticulous collection and processing techniques and analysis by real time PCR or 16S rRNA screening. The following tests are of value

- EMMA Endometrial Microbiome Metagenomic analysis
- ALICE Analysis of Infectious Chronic endometritis



- Identification of lactobacilli colonies by real time PCR or 16S rRNA sequencing. Paucity of lactobacilli is associated with dysbiosis and poor implantation rates

The pregnancy microbiome

The pregnancy microbiome refers to the microbial communities within the maternal body that influence maternal and fetal health during pregnancy. These microbiomes are primarily located in the gut, oral cavity, vagina, placenta, and amniotic fluid. Pregnancy is characterized by dynamic shifts in these microbiomes to support the physiological changes required for fetal development and a healthy pregnancy outcome.

The gut microbiome undergoes significant changes during pregnancy. In the first trimester, microbial diversity is similar to that of non-pregnant women. By the third trimester, there is a notable increase in the abundance of Proteobacteria and Actinobacteria, which enhance energy storage and immune tolerance 15. The gut microbiome supports maternal metabolism, energy extraction, and immune modulation. Dysbiosis in the gut microbiome during pregnancy is linked to gestational diabetes, preeclampsia, and excessive gestational weight gain[15].

Studies have shown infection and inflammation of uterine cavity leads to very preterm birth (<32 week) which leads to increased neonatal mortality and morbidity[16]. Various evidences have indicated that changes in maternal microbiome during pregnancy play a critical but undefined role in development of gestation diabetes, low birth weight, preterm labour, and neonatal necrotising enterocolitis. The early neonatal microbiome is also linked to maternal microbiome[16].

The vaginal microbiome is dominated by Lactobacillus species during pregnancy, particularly L. crispatus, L. jensenii, and L. gasseri. These bacteria produce lactic acid, maintaining a low vaginal pH that protects against infections. A stable vaginal microbiome reduces risks of bacterial vaginosis, preterm birth, and sexually transmitted infections. Dysbiosis during pregnancy has been linked to preterm labour and miscarriage[4].

The placenta, once considered sterile, is now known to harbor a low-biomass microbiome. Microbes in the placenta are similar to those in the oral cavity, suggesting hematogenous transfer[17]. Dominant bacteria include Firmicutes (Lactobacilli) Proteobacteria, Bacteroidetes, and Fusobacteria. The placental microbiome influences fetal immune system development. Abnormalities in this microbiome are associated with preeclampsia, fetal growth restriction, and preterm birth[17].

Pregnancy-induced hormonal changes increase the prevalence of periodontal pathogens such as Porphyromonas gingivalis and Fusobacterium nucleatum 18. Periodontal disease during pregnancy is linked to adverse outcomes such as preterm birth, low birth weight, and stillbirth[18].

Evidence suggests that amniotic fluid and the

developing fetus also have microbiomes, likely influenced by the maternal microbiota[19]. Dominant organisms include Lactobacillus and Ureaplasma. These microbial communities may prime the fetal immune system and play a role in preterm labor when disrupted[19].

The pregnancy microbiome plays a pivotal role in maternal health, immune regulation, and fetal development. Alterations in these microbial ecosystems, known as dysbiosis, are linked to pregnancy complications such as gestational diabetes, preterm birth, and preeclampsia. Continued research into the pregnancy microbiome offers promising insights into improving maternal and neonatal outcomes through targeted interventions, such as probiotics, dietary modifications, and microbiota transplantation.

The Role of the Gut Microbiome in Reproductive Health

Recent research highlights the importance of the gut microbiome in overall health, including reproductive function[20]. The gut microbiota, made up of trillions of microorganisms, plays a critical role in regulating inflammation, immune responses, and metabolic functions, all of which can influence reproductive health[21]. Dysbiosis, or an imbalance in the microbiota, has been linked to several reproductive disorders, including endometriosis, PCOS and infertility[20,22].

Probiotics: Benefits for Endometrial Health

Probiotics have been defined by the International Scientific Association on Pre & Probiotics (ISAPP) as live microorganisms that provide health benefit to the host, when consumed in adequate amounts. Studies have shown that certain strains of probiotics can support immune function and reduce systemic inflammation, which is crucial in conditions like endometriosis, where chronic inflammation is a hallmark feature[21]. The anti-inflammatory effects of probiotics might help in reducing the excessive inflammatory responses in the endometrial tissue that contribute to the pain and dysfunction seen in conditions like endometriosis[21]. Lactobacilli help to fight infection without inducing inflammation, which is an advantage during pregnancy[20].

In addition to their anti-inflammatory effects, probiotics may also improve gut barrier function, reducing the risk of endotoxins and other harmful substances entering the bloodstream[22]. This is important because an altered gut barrier has been linked to increased systemic inflammation, which may affect endometrial health.

Specific probiotic strains, such as Lactobacillus and Bifidobacterium, have been studied for their beneficial effects on reproductive health. These strains have been found to help maintain a healthy balance of vaginal and gut flora, which may be especially beneficial for individuals with recurrent uterine infections or conditions like bacterial vaginosis[20].

While much of the research is still in its early stages, there is growing evidence supporting the use of prebiotics and probiotics in managing endometrial conditions. There is growing support of the incorporating prebiotics and probiotics into treatment plans to provide a holistic approach to managing reproductive health, particularly in conditions like endometriosis and related gynecological disorders. Clinical studies suggest that these therapies may play a role in improving symptoms of endometriosis, improving immune function, and reducing inflammation 21. However, more rigorous, large-scale studies are needed to establish their efficacy, dose and duration of treatment to provide relief. Current research on Probiotics is in a nascent stage and we await randomised control trials to provide clearer insight for treatment guidelines.

Prebiotics and Their Role

Prebiotics are defined as a substrates that are selectively utilised by the host micro-organisms to confer a health benefit. They comprise of non-digestible food components that promote the growth of beneficial gut bacteria. By enhancing the proliferation of healthy gut microbiota, prebiotics support the function of probiotics and help maintain a balanced gut environment. In the context of endometrial health, prebiotics may help reduce the systemic inflammation that often exacerbates endometrial conditions 23. Foods rich in prebiotics, such as fibre, oligosaccharides, inulin and fermented foods like pickles, Kimchhi and Sauerkraut are thought to have indirect benefits on the reproductive system by supporting the overall health of the gut microbiome.

Postbiotics and Live Biotherapeutic Agents

Postbiotics and live biotherapeutic agents (LBAs) are emerging as promising tools in managing reproductive health and endometrial disorders. Postbiotics are bioactive compounds produced by probiotic microorganisms during fermentation, including metabolites, enzymes, and cell wall components. LBAs are live microorganisms with therapeutic applications

and are regulated as biological drugs rather than dietary supplements.

Postbiotics exert anti-inflammatory, immunomodulatory, and antioxidant effects[24]. They modulate the endometrial microenvironment, potentially benefiting conditions such as endometriosis and recurrent implantation failure[11]. For instance, short-chain fatty acids (SCFAs), a key postbiotic, promote a balanced immune response in the endometrium by inducing regulatory T-cell activity and inhibiting inflammatory cytokines like IL-6 and TNF-α. Postbiotics also strengthen epithelial barriers, reducing the risk of microbial translocation and chronic inflammation associated with infertility and miscarriage[11,24].

LBAs, particularly Lactobacillus spp. and Bifidobacterium spp., restore dysbiotic microbial communities in the reproductive tract[25]. In the endometrium, LBAs enhance implantation rates by promoting a favorable microbiota dominated by Lactobacillus[11]. They also reduce pathogen-associated risks like chronic endometritis and pelvic inflammatory disease (PID)[25]. LBAs have shown potential in preventing preterm labor by maintaining vaginal and cervical microbial homeostasis[25].

Targeted therapies with postbiotics and LBAs can improve outcomes in assisted reproductive technologies (ART) such as in vitro fertilization (IVF). By modulating the reproductive microbiome and reducing systemic inflammation, these agents may enhance endometrial receptivity and fertility[24,25]. They have also been successfully utilised in patients with recurrent vaginal candidiasis and recurrent urinary tract infections.

Commercial Preparations, their utility and clinical evidence.

While probiotics, prebiotics, and postbiotics are widely available as dietary supplements, LBAs require regulatory approval due to their classification as drugs. Accurate dosing, strain-specific efficacy, and long-term safety profiles remain areas of active research.

Commercially available probiotic preparations vary widely in their composition and colony forming units of probiotic strains. Gut probiotic strains are predominantly Bifidobacterium, whereas probiotics for reproductive health are predominantly Lactobacilli. Commonly employed strains are Lactobacillus rhamnosus GR-1 and Lactobacillus reuteri RC-14 as found in products like Probiotic Vaginal Suppositories. Typically, a dosage of 1–2 billion CFUs daily orally or intravaginally for 7–14 days is advised. A study by Reid et al. demonstrated that these probiotics help maintain vaginal pH, reduce bacterial vaginosis, and prevent urinary tract infections[26].

Prebiotics are non-digestible fibres that promote the growth of beneficial bacteria in the reproductive tract. For instance, Inulin and Fructo-oligosaccharides (FOS) that are available in supplements like Prebiotin and Benefiber are given in a dose of 3–5 grams per day. A diet rich in fermented food and drinks is also advisable. They enhance Lactobacillus colonization in the vaginal and gut microbiomes, supporting reproductive health and reducing inflammation[27].

Lacteal Peptides found in products like Hylak Forte are postbiotics, are administered in a dosage of 1 mL three times a day. They have shown anti-inflammatory and barrier-enhancing effects in the endometrium, improving implantation rates in IVF settings[24].

LBAs are live microorganisms with therapeutic potential and therefore are strongly regulated. Live Lactobacillus crispatus (LACTIN-V) is a vaginal gel specifically designed for preventing recurrent bacterial vaginosis 28. It is to be applied intravaginally every 3 days for up to 12 weeks. Clinical trials demonstrate a 50% reduction in bacterial vaginosis recurrence rates and improved reproductive outcomes.

Additionally, the route of administration for probiotics and prebiotics—vaginal or oral—plays a significant role in their efficacy, particularly in the context of reproductive health. Both methods have advantages, but their effectiveness depends on the target condition, desired microbial colonization, and the physiological barriers involved.

1. Vaginal Administration results in direct delivery to the reproductive tract and ensures high local concentrations of beneficial microbes. This results in rapid re-establishment of Lactobacillus-dominated vaginal microbiota, critical for maintaining vaginal pH and reducing bacterial vaginosis (BV) and yeast infections 29. Bacterial vaginosis is one of the most common infectious vaginal diseases in reproductive age females implicated as a cause of premature birth, PID, STDs. Bacterial vaginosis is cured better when antibiotics are combined with probiotics than antibiotics alone 30. And there are fewer systemic barriers (e.g., stomach acid, bile) that may reduce the survival of probiotics when they are administered vaginally. A study by Homayouni et al. (2014) demonstrated that vaginal administration of Lactobacillus rhamnosus and Lactobacillus reuteri significantly improved clinical symptoms of BV and maintained vaginal pH better than oral administration 29.

Vaginal probiotic gel formulations, such as LACTIN-V (Lactobacillus crispatus), showed a 50% reduction in BV recurrence rates compared to placebo in clinical trials 28. However vaginal absorption can be hampered by changes in pH as during menstruation, sexual activity and douching.

2. Oral Administration can influence the gut-vaginal axis by improving overall microbial balance, immune modulation, and systemic inflammation. It is definitely more convenient and non-invasive, enhancing compliance for long-term use. Probiotic bacteria are often administered as spores which are more stable, have a longer shelf life and can survive the harsh stomach pH. It has the potential to modulate the gut microbiome, which can indirectly benefit vaginal and endometrial microbiota[31]. Reid et al. (2001) showed that oral administration of L. rhamnosus GR-1 and L. reuteri RC-14 was effective in reducing BV and urinary tract infections (UTIs) 26. Other oral agents like lactobacillus acidophillus, lactoferrin RCX7M have proved to be useful in treatment of bacterial vaginosis[31]. Oral probiotics reach the vagina by both the haematogenous route as well as by transperineal migration from the anus. However, oral administration required longer durations to achieve effects compared to vaginal delivery. Oral probiotics, particularly those containing Lactobacillus spp., have shown promise in improving pregnancy outcomes by modulating systemic immune responses.

A meta-analysis by Huang et al. (2014) found that both routes of administration improved BV outcomes, but vaginal delivery showed faster symptomatic relief and a more pronounced effect on vaginal microbiota restoration[31].

In summary, while vaginal route may be preferred for localized infections like BV, yeast infections, and for restoring vaginal microbiota rapidly; oral probiotics are more suitable for systemic effects, such as improving gut health and its indirect benefits on reproductive health, especially in conditions like endometriosis or recurrent implantation failure.

Conclusion

Lactobacilli are a prominent part of the reproductive tract microbiome, as well as the placental and pregnancy microbiome. They are already major players in gut health. Treatment with probiotics may hold the key to avert many pregnancy complications like miscarriage, preterm birth, PPROM, and fetal growth restriction. Besides many gynecological pathologies like recurrent bacterial vaginosis, recurrent candidiasis, recurrent UTI and recurrent miscarriages are more successfully treated with Probiotics as compared to antibiotics.

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If you check the health of a woman, you check the health of society.

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Changing Trends in Infections in Obstetrics & Gynaecology

General Consideration

All kinds of living species have been interacting with each other in an open, yet close manner ever since the first evidence of life on earth approximately 3.7 billion years ago. Presence of 9% of viral DNA in our genome, which had been vectored by retroviruses about 70–90 million years ago, illustrates the great power of invisible intruders on the one hand, and vulnerability of humans on the other.

In 1980s, small pox was declared to be eradicated, polio was tamed by an aggressive vaccine programme, syphilis disappeared, swine flu had just been avoided, and Legionnaire's disease deciphered. New more powerful antibiotics were being introduced, and no infectious disease appeared to be immune from the eventual development of effective vaccines or antibiotics. The medical field felt invincible when it came to infectious diseases.

We were wrong. We subsequently entered a new era of infection-related public health crises. First the HIV epidemic came, followed by an international recurrence of congenital syphilis. Then came Severe Acquired Respiratory Syndrome (SARS) outbreak in 2003, H1N1 influenza in 2008–2009, recurrent episodes of Ebola Virus infection, the appearance of Zika virus in 2018 with its devastating fetal and neonatal sequela, and, beginning in 2019, the SARS-CoV-2 viral pandemic. Concurrent research advances in the identification, isolation and genetic sequencing of causative agents of new infectious diseases led to the sequencing of the SARS-CoV-2 virus within weeks of its initial appearance and creation of safe and effective vaccines in less than a year.

Despite these impressive accomplishments, clearly infectious diseases cannot be conquered. A new pandemic, potentially more deadly than prior ones, seems to now appear every 3–5 years. While mortality from infectious diseases had declined precipitously from 1900 to 1980, this trend has been reversed over the past 40 years. **At the time of writing, new HMPV (Human Meta Pneumo Virus) is in the news.**

In obstetrics and gynecology, the landscape of infections has been evolving. Newer infections that have emerged or gained increased attention in recent years reflect both shifts in medical practices, bacterial resistance, and shifts in patient demographics. Some key trends in infections related to this field include:

1. **Increased Antimicrobial Resistance (AMR):** Infections caused by resistant bacteria, such as multidrug-resistant (MDR) Escherichia coli, Staphylococcus aureus, and Group B Streptococcus, are becoming more prevalent in obstetric and gynecological settings. These strains complicate the treatment of common infections like urinary tract infections (UTIs) and post-surgical infections. Conditions like sepsis, tetanus, and malaria still impact obstetric care, particularly in rural and underserved regions. In many developing countries, maternal and neonatal infections remain leading causes of death. Group B Streptococcus (GBS) is a bacterium that can cause infections in pregnant women, newborns, and adults with chronic medical conditions. Although not new, there is increasing concern about antibiotic resistance in GBS strains. Screening for GBS during pregnancy (usually between 35-37 weeks) and the use of intrapartum antibiotics for carriers have been the main preventive strategy to reduce neonatal infection

rates. Vaccination efforts against rubella, hepatitis B, and other infections continue to be important for improving maternal and fetal health globally.

2. **Infections in Pregnancy:** Conditions like urinary tract infections, chorioamnionitis, and sexually transmitted infections (STIs) have gained more attention for their potential impact on maternal and fetal health. Untreated infections during pregnancy can lead to preterm labor, stillbirth, low birth weight, and other adverse outcomes.

- **Zika virus** outbreaks have raised awareness about infections during pregnancy and their impact on fetal development. Zika infection during pregnancy has been linked to microcephaly, a serious birth defect in infants, and other neuro-developmental issues. Pregnant women are at the greatest risk for severe outcomes. Preventive measures focus on mosquito control, using insect repellents, and avoiding travel to areas with active transmission.
- **COVID-19** pandemic has highlighted the impact of viral infections on pregnant women, who appear to be at a higher risk for severe illness from COVID-19. Pregnant women infected with SARS-CoV-2 have shown increased risks for preterm labor, and adverse pregnancy outcomes. There are also concerns about vertical transmission of the virus during pregnancy. This has led to changes in care practices, with more precautions in place to prevent the spread of the virus in hospitals and clinics. Vaccination of pregnant women, management of COVID-19 infections during pregnancy, and the provision of personal protective equipment (PPE) in healthcare settings are essential measures to control the spread.
- **Hepatitis E Virus (HEV):** While traditionally linked to developing countries, recent cases of HEV infection in pregnant women in developed countries have raised alarms. Hepatitis E can lead to severe liver failure in pregnant women, with high mortality rates. It is particularly dangerous in the third trimester. Preventive measures include avoiding contaminated water sources, and management may involve antiviral therapies and supportive care during pregnancy.
- **Human Herpes Virus 6 (HHV-6):** HHV-6 has been increasingly associated with various pregnancy complications and fetal abnormalities. Reactivation of HHV-6 during pregnancy may be linked to miscarriage and birth defects, though more research is needed. No specific treatment is available, but it is important to monitor pregnancies with known infections for complications.
- **Monkeypox,** a viral infection in the same family as smallpox, has become more prominent with a 2022 outbreak outside endemic areas. While rare, cases of monkeypox during pregnancy could result in adverse outcomes like miscarriage or stillbirth. Strict infection control and quarantine measures for infected individuals and vaccination strategies are being considered in certain regions.
- **Listeriosis (Listeria monocytogenes):** Listeria is a bacterial infection that can be contracted from contaminated food, especially unpasteurized dairy products, deli meats, and ready-to-eat meals. In pregnancy, listeriosis can lead to **miscarriage, stillbirth, preterm delivery,** and serious neonatal infections. Avoiding high-risk foods and following food safety guidelines during pregnancy.
- **Cytomegalovirus (CMV):** A common virus in the herpesvirus family, often transmitted via bodily fluids such as saliva, urine, and breast milk. CMV can cause birth defects including hearing loss, vision problems, and neurological impairments. CMV infection can be difficult to prevent, but good hygiene practices (e.g., handwashing after handling diapers) can reduce the risk.
- Varicella-Zoster Virus (Chickenpox): Chickenpox during pregnancy can lead to **miscarriage, preterm birth,** and congenital varicella syndrome, which can cause birth defects like **limb abnormalities** and **eye issues.** Pregnant individuals who have not had chickenpox should be vaccinated before pregnancy or, if exposed during pregnancy, may need antiviral treatment.
- **Toxoplasmosis:** Toxoplasma gondii, a parasite often contracted through contaminated food, soil, or cat feces. Toxoplasmosis during pregnancy can cause **miscarriage, stillbirth,** and severe birth defects like **brain damage** and **eye issues** in the baby. Avoiding raw or undercooked meat and practicing good hygiene with pet care can reduce the risk.
- **Parvovirus B19 (Fifth Disease):** A virus commonly seen in children, causing a rash illness called "fifth disease." In pregnancy, parvovirus can cause **miscarriage, fetalanemia,** and **hydrops fetalis.** There is no vaccine for

parvovirus B19, but avoiding exposure to infected individuals can reduce the risk.

3. **Sexually Transmitted Infections (STIs):** STIs, particularly chlamydia, gonorrhea, and syphilis, are increasing globally. Newer strains or types of sexually transmitted infections continue to emerge, and they pose a significant challenge in gynecological health. These infections are often asymptomatic but can lead to pelvic inflammatory disease (PID), infertility, and complications in pregnancy, including preterm birth and miscarriage and neonatal infections if untreated. These bacteria are increasingly showing signs of antibiotic resistance, particularly gonorrhea, which has raised concerns about untreatable strains. Regular screening for STIs in at-risk populations, timely antibiotic treatment, and surveillance for resistant strains are key.

   Some key infections include:

   - **Mycoplasma genitalium** is a sexually transmitted bacterium that has recently gained attention due to its role in causing cervicitis, pelvic inflammatory disease, and infertility. Mycoplasma genitalium infections can often be asymptomatic but are increasingly recognized as a cause of chronic pelvic pain, infertility, and miscarriage. The detection of Mycoplasma genitalium and appropriate antibiotic treatment is important. However, increasing resistance to antibiotics poses a challenge.
   - **Human Papillomavirus (HPV) Variants:** Newer, high-risk strains of HPV can lead to more aggressive cervical cancers or pre-cancerous changes in the cervix. While vaccines exist, the emergence of resistant strains complicates the management and prevention of these cancers.
   - **Chlamydia and Gonorrhea Resistance:** The rise of antibiotic-resistant strains of Chlamydia and Gonorrhea has made these infections harder to treat. Left untreated, they can cause pelvic inflammatory disease (PID), infertility, and ectopic pregnancies.
   - **Syphilis:** Though an older infection, syphilis is experiencing resurgence in some regions, leading to increased rates of maternal and fetal complications, such as congenital syphilis.

4. **Postpartum and Post-Surgical Infections:** With an increasing number of cesarean deliveries, there is a rising concern regarding infections related to surgical wounds and hospital-acquired infections (HAIs), including endometritis and surgical site infections (SSIs). The use of pre-operative antibiotics is now standard to prevent infection, but complications still occur. Postpartum infections, particularly sepsis due to retained products of conception, are rare but serious. Enhanced surveillance for these conditions is important to reduce maternal morbidity and mortality.

5. **Infections in Menopausal and Older Women:** As women age, hormonal changes related to menopause can lead to alterations in vaginal flora, increasing susceptibility to infections like bacterial vaginosis, yeast infections, and urinary tract infections. Older women, particularly those in postmenopausal years, may experience a higher frequency of recurrent UTIs due to changes in vaginal and urinary tract pH, reduced estrogen, and other age-related factors.

6. **Infection Control in Fertility Clinics:** With the rise in assisted reproductive technologies, there are increasing concerns about infections that may arise from procedures like in vitro fertilization (IVF). Infection prevention measures in ART clinics are critical to minimizing risks such as pelvic infections following egg retrieval or embryo transfer.

8. **Vaccine Preventable Infections:** While HPV has been known for decades, new high-risk strains are emerging, potentially increasing the incidence of cervical cancer and other anogenital malignancies. The recent variants of HPV, especially those that evade immune detection, may contribute to the rise in cervical dysplasia and carcinoma. Routine screening (Pap smear and HPV DNA testing) and vaccination programs are essential for prevention. Vaccination against HPV has significantly reduced the incidence of cervical cancer and related infections, though challenges remain in ensuring widespread vaccine coverage, especially in low-income regions. Vaccination against influenza and COVID-19 is becoming a standard part of prenatal care to protect both mother and baby, given the risks associated with these infections during pregnancy.

9. **Human Microbiome and Infections:** Research into the human microbiome, especially vaginal and gut microbiomes, is shedding light on how disruptions in normal flora may predispose women to infections like bacterial vaginosis, candidiasis, and even more complex conditions such as pelvic inflammatory disease.

   Candida auris (C. auris) is a multidrug-resistant fungus that has emerged as a global health threat. It is resistant to multiple antifungal drugs, making it

difficult to treat. While C. auris primarily affects immunocompromised patients in hospital settings, there have been concerns about its potential role in gynecologic infections, especially in hospitalized pregnant women or women with comorbidities. There's growing interest in using probiotics or other microbiome-modulating strategies to prevent infections, particularly in recurrent conditions like vaginal infections or UTIs.

All these infections can have significant implications for pregnancy. Their impact on pregnancy can range from mild to severe, affecting both maternal and fetal health. There can be increased risk of miscarriage or still birth (Zika virus, Listeria, and Cytomegalovirus (CMV)), preterm birth (Group B Streptococcus, Urinary Tract Infections (UTIs), Chlamydia and Covid 19), intrauterine growth retardation (CMV, syphilis, and toxoplasmosis), placental dysfunction, complicated delivery and psychosocial and economic impacts like stress, anxiety, and fear of poor outcomes, impacting the mental health of the mother. The need for special treatments, prolonged hospital stays, or intensive care can also result in financial strain.

Conclusion

Newer infections in obstetrics and gynecology continue to evolve, with some of them presenting unique challenges in terms of diagnosis, treatment, and prevention. Emerging pathogens, such as Zika and COVID-19, have had significant implications for pregnancy, while other infections, like C. auris and multidrug-resistant gonorrhea, emphasize the growing concern of antimicrobial resistance. These changing trends reflect a complex interplay of emerging infectious agents, antimicrobial resistance, new medical technologies, and changing patient demographics. Enhanced screening, vaccination, public health awareness, and infection control measures are crucial to mitigate the risks associated with these emerging threats. Addressing these trends requires a multi-pronged approach involving prevention, early diagnosis, appropriate use of antibiotics, and patient education.

As long as there is life on earth, there will be this interplay between bacteria, viruses and living beings. Given this, we cannot fore see how newly emerging or re-emerging infections will threaten us in the future.

> Love yourself enough to live a healthy lifestyle.
>
> Jules Robson

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Clinical approach to Vaginal Discharge

General Consideration

Vaginal discharge is a frequently presenting complaint in outdoor, especially in the women of reproductive age. But women of all age from prepubertal to postmenopausal and rarely newborn may present with this.The distress caused by discharge is subjective. Some patients are annoyed by the slightest amount of discharge, while others make no complaints in spite of marked discharge.

Symptomatic vaginal discharge may be due to physiological changes or infections or may be presentation of genital neoplasm. A step-by-step approach for diagnosis and treatment will be discussed in this article.

Physiological vaginal discharge

The slight discharge which is normally seen at the vulva and vagina is a mixture of vulval secretion, vaginal discharge, utero-cervical secretion and fallopian tube secretion. The amount of vaginal discharge ordinarily present in the adult is such that the introitus feels comfortably moist but that is not enough to stain the undergarment.

It is clear or white, mucus-like and does not smell bad and its amount and character changes with the menstrual cycle. The character of the discharge tends to be clearer with a stretchable consistency around ovulation, then may be thicker and slightly yellow during luteal phase. Normal discharge should not be associated with symptoms such as itching, redness, swelling, dysuria, malodour or adherent to the vaginal wall.

It normally increases to the extent of becoming noticeable during oestrogen states, such as :at the time of ovulation when there is the "ovulation cascade" from the cervix, during a few days premenstrually when there is increased secretion from all parts of genital tract, during pregnancy when there is increase in vaginal and cervical discharges, and during sexual excitement when there is an outpouring of Bartholin's secretion onto the vulva.

Physiological discharge is found in 10% of those who present with complain of vaginal discharge.(1)

Abnormal vaginal discharge

Abnormal vaginal discharge is characterised by a change in colour, consistency, volume or odour, and may be associated with symptoms such as itch, soreness, burning, dysuria, dyspareunia, pelvic pain, intermenstrual bleeding or blood mixed discharge.

Table 1- Enumerating causes of vaginal discharge

Infectitious 70%	Noninfectious 30%
Vulvovaginitis- Bacterial vaginosis, candidiasis, trichomoniasis, Gonococcal (prepubertal), Atrophic vaginitis	**Leuckorrhea**
	Traumatic vaginitis and cervicitis (tampons, pessaries, Foreign body)Contact or allergic vaginitis (Use of local soaps, powders, deodorants, antiseptics, spermicide, nylon underwear)
Cervicitis- Gonococci, Chlamydia, Herpes, Puerperal	**Neoplasm-** Benign (cervical polyp, sloughing submucous leiomyoma) Malignant-Cervical, endometrium
Endometritis- Gonococcal, Puerperal or senile, Pyometra	Urinary and faeculent discharge
Secondary infection of cervical erosion, abrasion, polyp, foreign body or chemical injuries and neoplasm sited in any part of genital tract	Intermittent emptying of hydrosalpinx (rare)

Infectotious cause

Vaginal discharge is most commonly caused by the infection of vagina and cervix. Most common cause of Vaginal infections causing vaginal discharge are bacterial vaginosis (BV), vulvovaginal candidiasis (VVC) or trichomoniasis (TV). Prepubertal and postmenopausal vulvovagitis may be due to nonspecific organism including candida albicans, streptococcus, staphylococcus and E coli.

Cervicitis is another important cause of vaginal discharge most commonly due to infection by Chlamydia trachomatis, Neisseria gonorrhoeae and herpes. Cervicitis should be suspected in sexually active women found to have mucopurulent discharge.

Monthly shedding of endometrium prevents colonization of pathogens to endometrium. That's why endometritis associated discharge is common in postmenopausal women and sometimes it is associated with pyometra. In premenopausal women it present with discharge coming from within the uterus and menorrhagia.

Secondary infection of cervical ectopy, cervical polyp, submucus fibroid polyp or atrophic vagina and endometrium also present with dischrge.

Non Infectious Causes of Vaginal Discharge

Leucorrhea

Leucorrhea mean an excessive amount of normal discharge. It dries to leave a brownish yellow stain on clothing.

Leuckorrhea is a nuisance in that it stains clothing and ,if the woman fails to bathe and change, frequently causes excoriation and soreness of the vulva. But it never causes pruritis.

It may be present during peripubertal period, cervical ectopy, due to active and passive congestion of pelvic organs, prolong standing in hot atmosphere, sedantary occupation, anxeity neurosis, prolong ill health, vaginal adenosis and women taking OCPs. Rarely mother of newborn babies may complain about discharge between day 1-10 neonatal life due to stimulation by placental oestrogen.

Use of local products

Vaginitis due to use of local soaps, powders, deodorants, antiseptics, spermicide, nylon underwear are now increasing. Some woman uses strong antiseptics for wash.

Neoplasm

Any growth which is exposed to lumen of genital tract either in vagina or uterus can cause continuous discharge which initially white or cream and nonoffensive but if ulcerated and infected discharge become purulent, offensive and blood stained. Such symptoms are characteristic of benign cervical polyp, sloughing submucous fibroid or malignant growth.

Urinary and faeculent discharge

Woman with genitourinary fistula usually present with complain of watery discharge from vagina. Now a days postsurgical ureteric fistula presenting weeks after surgery with vaginal discharge are increasing. Sometime woman with small faecal fistula may confuse for vaginal discharge. They can be recongnised easily because of peculiar smell.

A diagnostic approach to vaginal discharge

History, clinical examination is most important key for diagnosis. Microscopy and culture if required and available are better guide for treatment.

A good history should include characteristics of the vaginal discharge, relation to menstrual cycle and coitus, associated symptoms, local hygiene habits and sexual, medical and drug history (OCP, HRT, Immunosuppressive drugs, steroids, Prolong antibiotics).

Consider the age of pateints. In peripubetral girls possibility of sexual abuse or inappropiate hygeine habits should be ruled out. History of sexual contact in unmarried adolescent girls are not uncommon now a days. In a recently married woman possibility of trichomonal, gonococcal and chlamydia infection is high.

Ask if the discharge is acute, chronic, or frequently recurrent. Discharges that patients claim "never go away" are likely to be bacterial vaginosis, if pathologic, or simply physiologic discharges. Leuckorrhea has a gradual onset. History of high risk sexual behaviour in case of recurrent infection should be taken.

An important question to ask is whether the vaginal discharge is significantly altered from the woman's usual pattern. History of use of local hygeine or toilet preparations or douch should be taken.

History of symptoms in male partner like dysuria, local inflammation and tenderness is suggestive of trichomonal or gonococcal infection. As with all sexually transmitted diseases, it is important to know

the sex of the patient's sexual partner(s), the number of partners and any recent change in partner. Vulvovaginal candidiasis, trichomonas, bacterial vaginosis and herpes can all be transmitted between lesbian partners; gonorrhea, very rarely.

The intense inflammatory reaction of yeast or trichomonas can cause dyspareunia. Simultaneous dysuria and frequecy indicate infective causes like trichomoniasis, gonorrhea, chlamydia. Presence of postcoital bleeding may due to chlamydial infection or cervical malignancy. Presence of blood mixed offensive dischrage is more proximal to neoplasm.

Pruritis is associated with candida or trichomonal infection. Trichomonas vaginitis tend to occure or relapse during menstruation or postcoital. A change in sexual partner or a foul-smelling discharge suggest trichomoniasis. Pregnancy, diabetes, or recent antibiotic treatment suggest candidiasis. Discharge associated with gonococci and chlamydia cause soarness but never pruritis.

Clinical examination

Valval soreness, scratch marks may be present in candidasis and trichomaniasis. Vulval soarness, Periurethritis, bartholinitis but not pruritis is present in gonococcal infection.

On per speculum examination watch for charactristic of discharge, cervix and vagina. In cervicitis and endometritis, it may be seen coming through cervical os.

Vagina is coated with adherent discharge but not inflammed in bacterial vaginosis. Vagina may look inflamed in trichomoniasis. Cervical erosion or ectropin are cause of leuckorrhea. Hypertrophic cervicitis which bleeds on touch may be present in chlamydia.

Forgotten pads or swabs may found in a women who recently had a obstetric or gynaecological procedure.

If a woman has lower abdominal pain or if examination reveals lower abdominal tenderness, mucopurulent cervicitis or cervical excitation, one should consider upper genital tract pathology.

Look for mucopus in the endocervix by cleaning the ectocervix with a swab, inserting a small sterile swab in the endocervix and noting the presence of yellow pus on the swab. A bimanual examination should be done. Appropriate laboratory specimens should be obtained.

Woman with history of recent pelvic surgery and presenting with watery discharge should be tested by 3 swab test as a screening test in OPD.

Woman may have coinfections. So mixed clinical presentations are not uncommon.

Table 2: Characteristics of different type of vaginal discharge (2)

Features	Bacterial vaginosis (BV)	Vulvovaginal candidiasis (VVC)	Trichomoniasis (TV)	Gonococci	Chlamydia
Symptoms	Homogeneous, thin discharge adherent to vaginal walls Fishy odour, No itch or discomfort	Thick white discharge with non Offensive odour Valvul itch, dyspareunia	Scanty to profuse frothy yellow-green malodorous discharge Valvul itch, dyspareunia, dysuria, discomfort	Purulent discharge Associated urethritis, cystitis, Bartholinitis may present	Purulent discharge
Signs	No inflammation on vulva and vagina	Vulval erythema, excoriation	Vulvitis and vaginitis, Strawberry vagina and cervix	Vulval erythema, Cervicitis	Cervicitis
Need to treat sexual Partner	No	No	Yes	Yes	Yes
Vaginal pH	>4.5	<4.5	>4.5	>4.5	
Need for testing for other STI	No	No	Yes	Yes	Yes
				Co-treatment of chlamydial infection is recommended	

Treatment

Coinfection is very common due to common predisposing factors. Infections should be treated with appropriate antibiotics. There are 4 Cardinal rules for effective treatment.

1. Use of appropriate antibiotic in appropriate dose for treatment of infection

Table 3: Recommended treatment of vaginal/Cervical infection (2)

	Oral	Vaginal gel	Vaginal ovules
BV	Metronidazole 500 mg BD for 7 days OR Clindamycin 300 mg BD for 7 days OR Secnidazole 2 g single dose OR Tinidazole 2 g once daily for 2 days OR Tinidazole 1 g once daily for 5 days	Metronidazole gel 0.75% one full applicator (5 g) intravaginally, once a day for 5 days OR Clindamycin cream 2% one full applicator (5 g) intravaginally at bedtime for 7 days	Clindamycin ovules 100 mg once at bedtime for 3 days
Trichomoniasis	Metronidazole 500 mg BD for 7 days OR Tinidazole 2 g single dose		
VVC	Fluconazole 150 mg single dose OR Tab Itraconazole 200 mg BD for 3 days	Clotrimazole 1% cream 5g intravaginally daily for 7–14 days OR Clotrimazole 2% cream 5g intravaginally daily for 3 days OR Miconazole 2% cream 5g intravaginally daily for 7 days	Miconazole 100 mg vaginal suppository one suppositor daily for 7 days OR Clotrimazole 100mg vaginal tablet daily for 6 days
Gonococci	Single 1 g oral dose of azithromycin OR Single 500 mg IM dose of ceftriaxone PLUS doxycycline 100 mg orally BD for 7 days OR Cefixime 800mg single dose PLUS Azithromycin or Doxycyclin for 7 days		
Chlamydia	Doxycycline 100 mg orally BD for 7 days ORAzithromycin 1g orally in a single dose OR Levofloxacin 500 mg orally OD for 7 days		

2. Treatment of partner
3. Screening for STIs
4. Steps to prevent recurrence

Treatment of partner

For TV, gonorrhea and chlamydia all sexual partners should be concurrently treated and sexual intercourse avoided for at least one week until they and their partners have completed treatment and follow-up.

Screening for sexually transmitted infections

It is recommended to screen for STI in women who are diagnosed with TV, Gonorrhea, chlamydia and in women who are at high risk of STI. They should be offered testing for HIV, syphilis and hepatitis B virus.

Prevention of recurrent pathological vaginal discharge

Local hygeine practices has great impact on normal vaginal flora. Vaginal dysbiosis predisposes women to recurrent vaginal infections. Simultaneous treatment of vaginal infection and healthy local hygeine practices shift vaginal dysbiosis to eubiosis and prevent recurrence specially bacterial vaginosis.

Woman is advised to keep vagina dry. Change out wet clothes promptly after swimming or working out. Wash pubic area gently with warm water. Soap is not needed on vulva and should never be used in vagina. Wear cotton underwear. Cottonlets vaginal area breathe more than synthetic fabrics. Clean reusable products like menstrual cups, diaphragms, cervical caps and spermicide applicators after every use.Do not douche. Douching wash away normal vaginal flora. Avoid using scented soap gels or deodorants. Wipe from front to back after using the toilet or changing a pad. Change pads every 2–4 hours. Minimize the use of tampons. Practise safe sex.(3)

There is no clear and consistent evidence across currently published studies regarding the role of

probiotics for vaginal health.

Chemical cautry or Cryotherapy or electrocoagulation treatment of cervical ectopy can be done in case of persistant leuckorrhea. In a diabetic woman emphasis on good glycemic control should be given.

Key points

1. Vaginal discharge can be either a normal physiologic occurrence or a pathological manifestation. It is important to differentiate by history and clinical examination. A history of a change from the usual pattern of vaginal discharge and presence of associated symptoms like dysuria and pruritis is an important differentiating factor.
2. Characteristic of vaginal discharge and local features like erythema and oedema of vulva, vagina and cervix during the physical examination may be helpful in narrowing the causes of the vaginal discharge.
3. Vaginal discharge in postmenopausal women should always considered pathological.
4. STI testing is recommended in high-risk sexually active woman or those with TV, Gonorrhea or chlamydia.
5. Partner should be treated simultaneously in trichomoniasis, gonorrhea and chlamydia.
6. Local hygeine practices, safe sex practices and vaginal eubiosis has great impact on vaginal discharge.
7. Benefit of probiotics in vaginal discharge is still not proven by any RCT. Any present guidelines don't recommend it for treatment of abnormal vaginal discharge. It is sold under banner of health supplements only.

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A strong woman stands up for herself. A stronger woman stands up for others.

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Female Genital Tract Tuberculosis- Diagnosis & Management Update

Background

Genital tuberculosis is responsible for menstrual symptoms, infertility, abdominal pain, tubo-ovarian masses, ectopic pregnancy, PID refractory to treatment, cervical growth, ulcers and vulval lesions. According to the World Health Organization Global Tuberculosis Report from 2024, a total of 8.2 million people were reported as newly diagnosed with TB in 2023, up from 7.5 million in 2022[1] and India alone contributed 26% to this global scenario. Female Genital TB (FGTB) is one of the forms of extrapulmonary TB (EPTB) and has been reported to contribute up to 9% cases of EPTB[2].

The exact incidence of Female Genital Tuberculosis (FGTB) is not known due to under-reporting as it frequently presents without symptoms, requiring high index of suspicion for diagnosis. Up to 10% patients lack symptoms.[2] FGTB produces devastating effects by causing irreversible damage to fallopian tubes which is difficult to cure both by medical and surgical methods. It also poses a diagnostic dilemma because of its varied clinical presentation and lack of sensitive and specific diagnostic methods.

Incidence/Prevalence:

The incidence of GTB varies and can be as low as 0.69% in Australia or as high as 19% in India.3 The fallopian tubes and endometrium are the prime sites of involvement in FGTB. Around 19% of infertile women are reported to have genital tuberculosis. Tubercular endometritis accounts for 1% of postmenopausal bleeding4. Around 10% women are asymptomatic or may present with varied symptomatology.

When to suspect?

- History of infertility
- Chronic lower abdominal or pelvic pain
- Amenorrhoea or other menstrual disturbance
- Abnormal vaginal discharge
- Constitutional symptoms of TB like low grade fever, night sweats, loss of weight or appetite

Diagnostic tests for FGTB

In absence of gold standard, diagnosis of genital TB is a dilemma. For suspected FGTB, appropriate specimens from the suspected sites (endometrial aspirate) should be obtained for microscopy, culture, and histopathological examination in a quality-assured laboratory. Endometrial aspirate is preferably taken in luteal phase of cycle from day 21-day 23 of cycle.

Criteria for a definitive diagnosis includes[5]

1. **Demonstration of Mycobacterium tuberculosis on microscopy** has poor pick up rate as FGTB is a paucibacillary disease and AFB Microscopy requires ≥10 000 bacilli/mL specimen.
2. **Culture (on solid and liquid media)** is the most widely used gold standard for validating results in diagnosing EPTB specimens, although pick up rates are low as culture requires ≥100 bacilli/mL
3. **Histopathology** confirming the presence of epitheloid granulomatous lesions.
4. **Composite reference standard(CRS).** Whenever practical, every effort should be made to obtain clinical samples for both microbacteriology (AFB

smear and culture) and histopathologic tests.[5]

5. Laparoscopy offers the dual advantage of pelvic organ visualization and sample collection from inaccessible sites
6. The inconsistency among various laboratory tests signifies the need for multisampling for multiple tests which may increase the diagnostic yield.
7. **TST** (Tuberculin skin testing- Mantoux test) in diagnosis of GTB in suspected patients: Mantoux test cannot distinguish between infection and disease. Reported sensitivity is 55%, specificity is 80%, with 45% false negativity in women with laparoscopically diagnosed tuberculosis. A positive skin test supports a diagnosis, but a negative test does not necessarily exclude EPTB.[6]
8. **Interferon gamma radio immunoassay (IGRA)** is more specific than TST particularly in people who have received BCG, however like TST, it cannot distinguish between infection and disease. Currently there is no evidence to support the use of IGRA in routine practice.
9. **Endometrial Aspirate (EA)** for AFB microscopy, culture and histopathology in patients with suspected FGTB: EA for AFB microscopy has a pickup rate of 1.1% to 8.3%. EA culture for AFB by LJ (solid) medium has a pickup rate of 2% to 18.3% and by Bactec (liquid medium) method, the pickup rate is 2% to 8.8%. EA histopathology pick-up rate is 3.4% to 18.5%[7].
10. **Endometrial Aspirate DNA PCRin patients with suspected GTB:** The role of PCR is controversial because of the high rate of false positives. Most studies advocate the commencement of ATT in women with a positive PCR result only if there is evidence of GTB on clinical examination also or if the hysteroscopy or laparoscopy is suggestive of GTB.[9]
11. **Newer molecular tests RT PCR in the diagnosis of GTB:** There is evidence that mRNA PCR was only positive if the culture was also positive- hence offered no extra benefit.[10]
12. **Gene Xpert in the diagnosis of GTB.** Gene Xpert had sensitivity of 35.63%, specificity of 100%, positive predictive value of 100% and negative predictive value of 58.82% and diagnostic accuracy of 66.47% in this study[11]
13. **Adenosine deaminase** (ADA) testing on ascitic fluid in patients presenting with adnexal mass with ascites: They are non-specific chemical marker, yet helpful in pelvic-peritoneal tuberculosis presenting as an adnexal mass and mimicking ovarian cancer. Ascitic fluid ADA activity has good accuracy but poor sensitivity and imperfect specificity. There is no consensus in exact cut off, however values between 21-40IU/L are considered as cut offs
14. **Diagnostic laparoscopy:** Laparoscopy is increasingly being used for the early detection of FGTB because it offers the dual advantage of pelvic organ visualization and sample collection from inaccessible sites. Laparoscopic findings including presence of tubercles, caseation, or beaded tubes confirm the presence of tuberculosis5. Presence of straw-colored fluid in the pouch of Douglas, extensive, dense pelvic and/or peri-tubal/periovarian adhesions, hydrosalpinx, tubo-ovarian mass, thick fibrosed tubes, mid-tubal block, perihepatic adhesions, hyperemic tubes/blue uterus on chromo-tubation also may point towards tuberculosis but need additional evidence for confirmation. Hysteroscopy may detect uterine adhesions, presence of tubercles

Summary of all Diagnostic tests

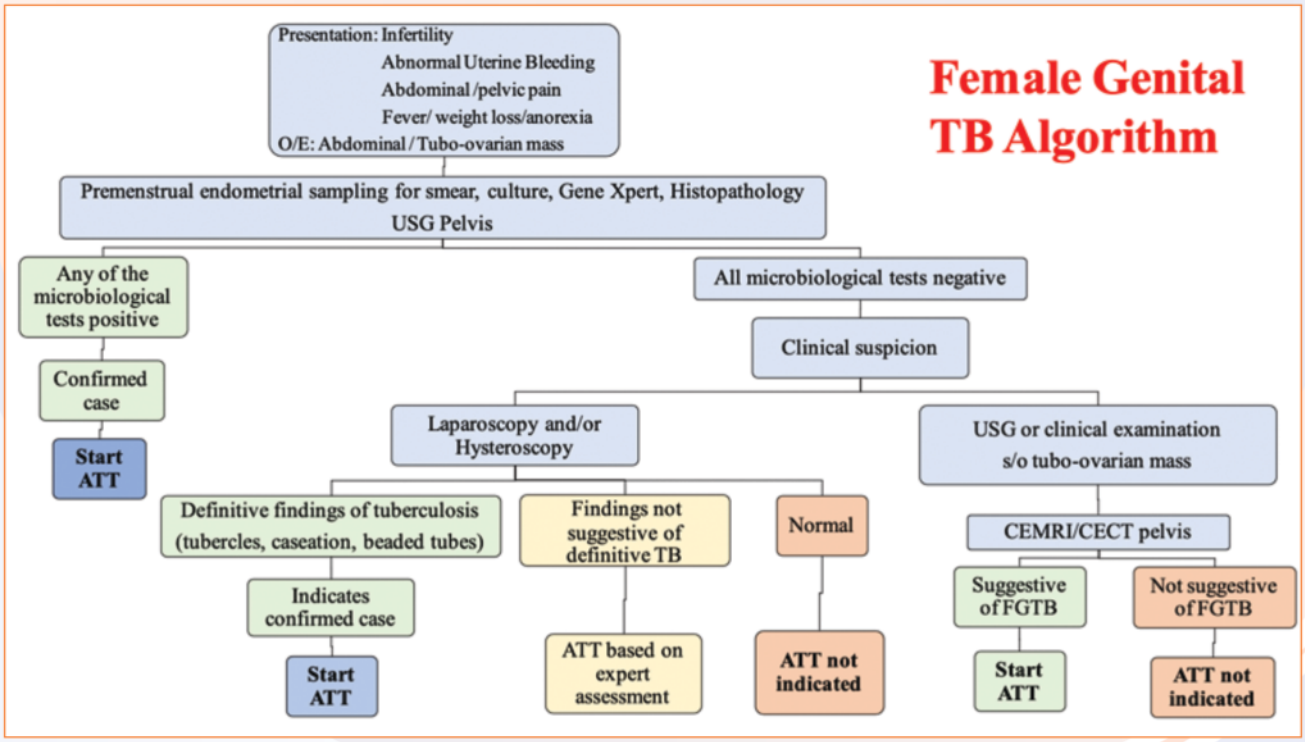
Test	Which patients?	Comments
X-ray of chest	All	All patients presenting with symptoms consistent with TB should have a chest X-ray to look for evidence of previous or active pulmonary TB.
HIV test	All	EPTB is associated with HIV infection. All patients should be offered HIV testing after counselling.
Pregnancy test	All	To rule out pregnancy as possible cause of symptoms, and to ensure further testing is safe and appropriate.
Pelvic ultrasound	All	Part of the initial assessment of most patients presenting with gynaecological symptoms.
Hysterosalpingogram	Selected	May be done as part of the investigation of infertility. To be avoided in presence of acute symptoms or vaginal discharge. Findings suggestive of FGTB include cornual block, tobacco pouch appearance of tubes, beaded tubes, filling defect in the uterine cavity but many women with FGTB will have a normal HSG.
CT pelvis or MRI pelvis	Selected	To further characterise lesions and plan surgical intervention in selected patients. Disadvantage of CT is exposure to ionising radiation, particularly a concern in women of childbearing age.

Test	Which patients?	Comments
FDG-PET CT	Selected	Although not widely available, PET scans may give more information about the presence and activity of tubercular tubulo-ovarian mass lesions. Further evidence about the diagnostic accuracy of PET CT for detecting and monitoring the progression of FGTB is needed.
Endometrial aspirate	Selected	Where facilities exist, endometrial aspirate can be obtained and sent for a) staining and microscopy for AFBs; b) culture and drug susceptibility testing; c) Histopathology. Sensitivity is low, and negative results cannot rule out FGTB.
Laparoscopy	Selected	Laparoscopy with biopsy of lesions is required when - Other less invasive tests are inconclusive - Laparoscopy is needed for evaluation of infertility Laparoscopy offers the dual advantage of pelvic organ visualization and specimen collection from otherwise inaccessible sites. Specimens should be subject to a) staining and microscopy for AFBs; b) culture and drug susceptibility testing; c) histopathology.

As per Index TB guidelines, 20165 (Index-TB Guidelines: Guidelines on extrapulmonary tuberculosis), the diagnosis of FGTB should be made based on any one of the following

- Laparoscopic appearance typical for FGTB (tubercles, caseation or beaded tubes).
- Any gynaecological specimen positive for AFB on microscopy, GeneXpert or positive for M.tuberculosis on culture.
- Any gynaecological specimen with findings consistent with FGTB on histopathological examination.

Many times, none of the tests are positive, however ATT can still be started based on expert clinical judgement. The decision in such cases should be taken by an expert clinician.



Reference[12]

Management:

Aim of treatment is to achieve tuberculosis cure in genital tract and to prevent the long term sequalae. If possible effort is made to restore normal anatomy of female genital tract.

All new patients should receive treatment as per INDEX TB guidelines.[5,13] The initial phase should consist of two months of Isoniazid (H), Rifampicin(R), Pyrazinamide (Z), and Ethambutol (E). The continuation phase consists of three drugs (Isoniazid, Rifampicin, Ethambutol) for four months

In Genitourinary disease, 6 months of treatment may be

Treatment plan	(2)HRZE	(4)HRE

Drug dosage for adult TB- Fixed dose combinations[12]

Weight Band (Kg)	No. of FDC tablets#IP – 4FDC (HRZE) 75/150/ 400/275 (mg)	No. of FDC tablets CP – 3FDC (HRE) 75/150/275 (mg)
25-34	2	2
35-49	3	3
50-64	4	4
65-75	5	5
>75	6	6

*can be given 5 tablets/day if they do not tolerate the revised doses.

#FDC: Fixed Dose Combination tablets containing 3(3FDC) or 4(4FDC) drug combination

adequate. No difference in efficacy of 6 months vs 9 months ATT. There were equal number of treatment failures(2 in each group)after the end of treatment)5

Monitoring of treatment is done as other EPTB

Follow up depends on initial presenting symptoms. In case of badly damaged tubes, In vitro fertilisation/ surrogacy/adoption is to be offered.

In case of persistent TO mass, PET CT can be done to determine the activity.

Surgery is not primary treatment in genital tuberculosis, it should be avoided, however it is needed for large, residual TO abscess. Surgery in FGTB is associated with higher complication rates as there are a lot of adhesions as well as possibility of infection flare up. Tubal anatomy should be restored surgically as far as possible in infertile women.

Outcomes (successful treatment, treatment failure)

Cured Cases (successful treatment)

- If initially diagnosed with microbiology/ HPE, patients with negative respective tests at the end of ATT
- If initially diagnosed with laparoscopic findings suggestive of active TB (tubercles, caseation, beaded tubes), negative relook laparoscopy
- Resolution of clinical symptoms

Confirmed Treatment failure

Persistent microbiology/HPE or persistent laparoscopic findings are suggestive of active TB (tubercles, caseation, beaded tubes) at the end of ATT. Appropriate tissue sample should be obtained for drug sensitivity testing for MDR.

Management of treatment failure

Drug resistant EPTB should be managed by obtaining tissue specimen and subjecting to drug sensitivity testing and ATT should be started accordingly. The duration of the treatment is to be decided as per the guidelines released by Government of India14

Treatment in special groups

- **In pregnancy :** Pregnant women with TB should start or continue ATT in the same way as other patients.

  These first-line drugs cross the placenta but do not appear to be teratogenic. Streptomycin can cause congenital deafness and prothionamide is teratogenic, so both should be avoided. Ethionamide causes birth defects at high doses in animals. Pyridoxine 10 mg/day is recommended for pregnant women taking isoniazid.

- **In lactating mothers :** Women who are breast-feeding should be given standard TB treatment regimens. Nursing mothers should continue breast feeding as its discontinuation poses a serious risk to the infant's health.

- **Contraception :** Since rifampicin reduces the effectiveness of oral contraceptives, for effective contraception, women should be advised to choose between one of two options for contraception:

  An oral contraceptive pill containing estrogen (50 ìg) following consultation with a clinician, a nonhormonal method of contraception throughout rifampicin treatment and for at least one month subsequently.

Key Points

- FGTB causes gynecological symptoms such as infertility, menstrual dysfunction and chronic pelvic pain
- Diagnosis is made by history, clinical examination and Composite Reference Standard which includes endometrial aspirate for AFB, culture, GeneXpert PCR and histopathology aided by endoscopy
- Treatment is as per Standard TB guidelines i.e. 2 months of HRZE followed by 4 months of HRE.
- Fertility outcome is poor in FGTB, but In-Vitro Fertilization (IVF) can be performed for tubal blockage with normal endometrium with a good outcome.

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A woman's health
is her capital

Harriet Beecher Stowe

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Recurrent UTI - Current Recommendations & update

General Consideration

Urinary tract infections (UTI) are the most common infections in women worldwide. Approximately 60% of women will experience acute UTI in their lifetime & 30-40% will experience recurrent UTIs (rUTI). The diagnosis of UTI is made by the presence of clinical symptoms (dysuria, suprapubic tenderness, urinary urgency, & frequency) & the presence of 10^5 cfu/ml.

A patient can be asymptomatic despite the presence of bacteria in the urine, known as Asymptomatic bacteriuria. The most common bacterium associated with UTI is Escherichia coli (E. coli), which is most often looked for in culture. Escherichia coli (E. coli), which invades the urothelium and multiplies rapidly. Other organisms, such as Klebsiella, Proteus, and Enterococcus species, are less commonly implicated but can pose significant challenges, especially in complicated UTIs.

Risk Factors for UTIs

UTIs are predominantly seen in women due to shorter urethras and proximity to the anus, facilitating bacterial access. Key risk factors include:

1. **Sexual Activity:** Sexual intercourse, particularly with a frequency greater than twice per week, increases the risk due to mechanical introduction of bacteria.
2. **Spermicides and Contraceptive Devices:** The use of spermicidal agents can disrupt the normal vaginal flora, increasing susceptibility to infection.
3. **Post-Menopausal Changes:** Oestrogen deficiency leads to atrophic vaginitis, reduced lactobacilli, and higher vaginal pH, all of which predispose to infections.
4. **Urinary Stasis and Obstruction:** Conditions like renal calculi, cystocele, or incomplete bladder emptying contribute to recurrent infections.

Recurrent UTIs (rUTIs)

rUTIs are recurrences of uncomplicated &/or complicated UTIs with a frequency of atleast 3 UTIs/year or 2 UTI's in the last 6 months. rUTIs are common. Risk factors are outlined below in table1. The diagnosis should be confirmed by urine culture. If renal calculi, outflow obstruction, interstitial cystitis or urothelial cancer suspected, cystoscopy & imaging to be performed.

Mechanisms of Recurrent UTIs

Recurrent UTIs can result from either reinfection by different bacterial strains or relapse of the same strain. Differentiating between these mechanisms is critical:

- **Relapse:** Occurs within two weeks of completing therapy with the same organism and often indicates incomplete eradication.
- **Reinfection:** New infections diagnosed more than two weeks after treatment are usually caused by different organisms.

Bacterial biofilms within the bladder lining represent a significant challenge, as they protect pathogens from antibiotics and host immune responses.

Treatment Options

Antibiotics

First-line treatments include:

- **Nitrofurantoin** (100 mg twice daily for 5-7 days)
- **Fosfomycin** (3 g as a single dose)

- **Trimethoprim-sulfamethoxazole** (160/800 mg twice daily for 3 days)

For severe or complicated cases, hospitalization and intravenous antibiotics may be required. The goal of treatment is to prevent progression to pyelonephritis and its complications, such as abscess formation or sepsis.

Non-Antibiotic Interventions

1. **Hydration:** Increasing fluid intake can reduce bacterial adherence to the bladder wall.
2. **Probiotics:** Specific strains like Lactobacillus rhamnosus GR-1 and L. reuteri RC-14 help restore vaginal flora and reduce UTI episodes.
3. **Cranberry Products:** While evidence is mixed, cranberry has been suggested to prevent bacterial adhesion to the urothelium.
4. **D-Mannose:** This sugar may prevent bacterial attachment, though evidence remains inconclusive.

Special Considerations in Post-Menopausal Women

Hormonal changes in post-menopausal women require tailored strategies:

1. **Vaginal Oestrogen Replacement:** Restores the vaginal flora and reduces the recurrence of UTIs.
2. **Management of Co-Morbidities:** Address conditions like urinary incontinence and increased post-void residual volume.

Recurrent UTIs are recurrences of uncomplicated &/or complicated UTIs with a frequency of atleast 3 UTIs/year or 2 UTI's in the last 6 months. rUTIs are common. Risk factors are outlined below in table 1.

Table 1: Age-related association of rUTI in women

Young & pre menopausal women	Post-menopausal & elderly women
Sexual intercourse (higher frequency >twice/week, tripples the risk of UTI)	History of UTI before menopause
Use of spermicide	Urinary incontinence
A new sexual partner	Atrophic vaginitis due to oestrogen deficiency
A mother with a history of UTI	Cystocoele
History of UTI during childhood	Increased post-void urine volume
Blood group antigen secretory status	Blood group antigen secretory status
	Urine catheterisation & functional status
	Deterioration in elderly institutionalised women

The diagnosis should be confirmed by urine culture. If renal calculi, outflow obstruction, interstitial cystitis or urothelial cancer suspected, cystoscopy & imaging to be performed.

GUIDELINE STATEMENTS (AUA -American Urological Association)

Evaluation

- Clinicians should obtain a complete patient history & perform a pelvic examination in women presenting with rUTIs. (Clinical Principle)
- To make a diagnosis of rUTI, clinicians must document positive urine cultures utilizing a midstream clean catch sample, associated with prior symptomatic episodes. (Clinical Principle)
- Clinicians should obtain repeat urine studies when an initial urine sample is suspect for contamination, with consideration for obtaining a catheterized sample. (Clinical Principle)
- Cystoscopy & upper urinary tract imaging should not be routinely obtained in the index patient presenting with a rUTI. (Expert opinion)
- Clinicians should obtain urinalysis, urine culture & sensitivity with each symptomatic acute cystitis episode prior to initiating treatment in patients with rUTIs. (Moderate Recommendation: Evidence Level: Grade C)
- Clinicians may offer patient-initiated treatment (self-start treatment) to select Ruti patients with acute eisodes while awaiting urine cultures. (Moderate Recommendation: Evidence Level: Grade C)

Asymptomatic Bacteriuria

- Clinicians should omit surveillance urine testing. Including urine culture, in asymptomatic patients with rUTIs. (Moderate Recommendation: Evidence Level: Grade C)
- Clinicians should not treat ASB in patients. (Strong Recommendation:

  Evidence Level: Grade B)

Antibiotic Treatment

- Clinicians should use first-line therapy (i.e., nitrofurantoin, TMP-SMX, Fosfomycin) dependent on the local antibiogram for the treatment of symptomatic UTIs in women. (Strong Recommendation: Evidence Level: Grade B)
- Clinicians should treat rUTI patients experiencing acute cystitis episodes with as short a duration of

antibiotics as reasonable, generally no longer than seven days. . (Moderate Recommendation: Evidence Level: Grade B)

- In patients with rUTIs experiencing acute cystitis episodes associated with urine cultures resistant to oral antibiotics, clinicians may treat with culture-directed parenteral antibiotics for as short a course as reasonable, generally no longer than seven days. (Expert Opinion)

Antibiotic Prophylaxis

- Following discussion of the risks, benefits & alternatives, clinicians may prescribe antibiotic prophylaxis to decrease the risk of future UTIs in women of all ages previously diagnosed with UTIs. (Conditional Recommendation: Evidence Level: Grade B)

Non-Antibiotic Prophylaxis

- Clinicians may offer cranberry prophylaxis for women with rUTIs. (Conditional Recommendation: Evidence Level: Grade C)

Follow-up Evaluation

- Clinicians should not perform a post-treatment test of cure urinalysis or urine culture in asymptomatic patients. (Expert Opinion)
- Clinicians should repeat urine cultures to guide further management when UTI symptoms persist following antimicrobial theapy. (Expert Opinion)

Estrogen

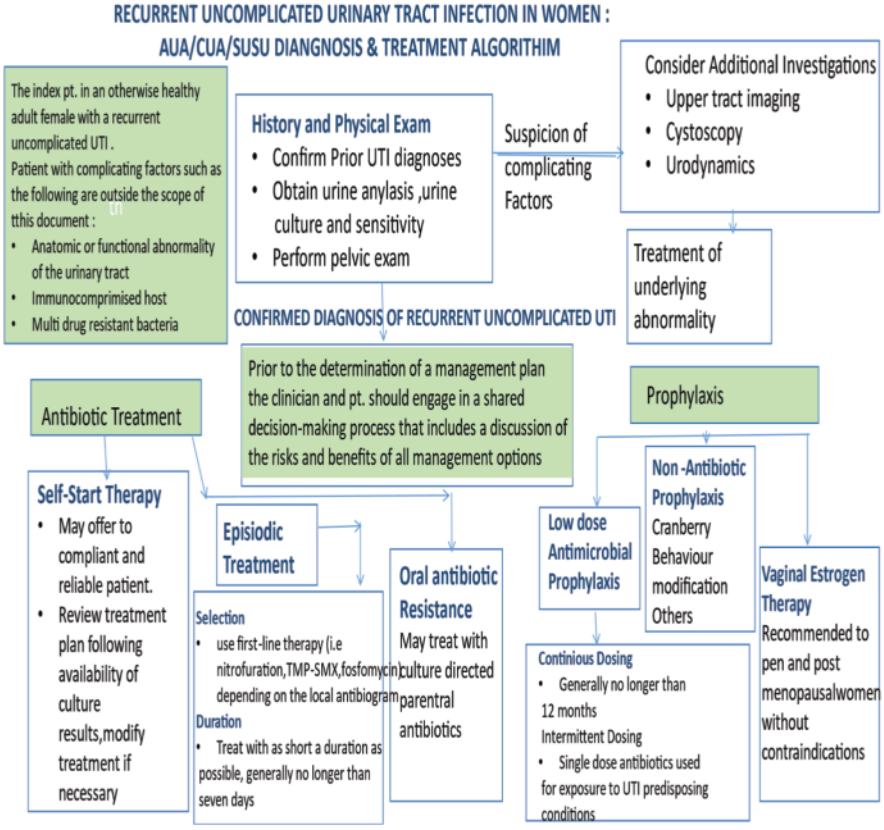
- In peri & post-menopausal women with rUTIs, clinicians should recommend vaginal estrogen therapy to reduce the risk of future UTIs if there is no contraindication to estrogen therapy. (Moderate Recommendation: Evidence Level: Grade B)

Summary of evidence and recommendations for the diagnostic evaluation and treatment of rUTIs (EAU guidelines)

Evidence	Level of Evidence (LE)
Extensive routine workup including cystoscopy, imaging, etc., has a low diagnostic yield for the diagnosis of rUTI.	3
Increased water intake is an effective antimicrobial-sparing strategy to prevent rUTI in premenopausal women who drink low volumes (<1.5 L) of fluid daily.	3
Vaginal oestrogen replacement has shown a trend towards preventing rUTI in post-menopausal women.	1b
Immunoactive prophylaxis (Escherichia coli extract OM-89) has been shown to be more effective than placebo in female patients with rUTIs in several randomized trials with a good safety profile.	1a
Probiotics containing L. rhamnosus GR-1, L. reuteri B-54 and RC-14, L. casei shirota, or L. crispatus CTV-05 are effective for vaginal flora restoration and have shown a trend towards prevention of rUTIs.	1b
Current scientific evidence regarding the efficacy of cranberry products in the prevention of rUTIs is inconclusive.	1a
There is contradictory evidence on the efficacy of D-mannose to reduce the number of UTI episodes.	2
Based on limited evidence, intravesical GAG therapy can reduce the number of UTIs per patient per year and prolong the time interval between rUTI episodes.	2
A randomized controlled trial demonstrated the non-inferiority of twice-daily methenamine hippurate to daily antibiotic prophylaxis.	1b
Both continuous low-dose antimicrobial prophylaxis and post-coital antimicrobial prophylaxis have been shown to reduce the rate of rUTI.	1b
A prospective cohort study showed that intermittent self-start therapy is effective, safe, and economical in women with rUTIs.	2b

Recommendations	Strength Rating
Diagnose recurrent UTI by urine culture.	Strong
Do not perform an extensive routine workup (e.g., cystoscopy, full abdominal ultrasound) in women younger than 40 years of age with recurrent UTI and no risk factors.	Weak
Advise pre-menopausal women regarding increased fluid intake as it might reduce the risk of recurrent UTI.	Weak
Use vaginal oestrogen replacement in post-menopausal women to prevent recurrent UTI.	Strong
Use immunoactive prophylaxis to reduce recurrent UTI in all age groups.	Strong
Advise patients on the use of local or oral probiotic-containing strains of proven efficacy for vaginal flora regeneration to prevent rUTIs.	Weak

Recommendations	Strength Rating
Advise patients on the use of cranberry products to reduce recurrent UTI episodes; however, patients should be informed that the quality of evidence underpinning this is low with contradictory findings.	Weak
Use D-mannose to reduce recurrent UTI episodes, but patients should be informed of the overall weak and contradictory evidence of its effectiveness.	Weak
Use methenamine hippurate to reduce recurrent UTI episodes in women without abnormalities of the urinary tract.	Strong
Use endovesical instillations of hyaluronic acid or a combination of hyaluronic acid and chondroitin sulfate to prevent recurrent UTIs in patients where less invasive preventive approaches have been unsuccessful. Patients should be informed that further studies are needed to confirm the results of initial trials	Weak
Use continuous or post coital antimicrobial prophylaxis to prevent recurrent UTI when non antimicrobial interventions have failed. Counsel patients regarding possible side effects	Strong
For patients with good compliance self-administered short term antimicrobial therapy should be considered	Strong



AUA **(American Urological Association), CUA (Canadian Urological Association)**

SUFU (Society of Urodynamics, Female Pelvic Medicine & Urogenital Reconstruction)

Summary of Preventing Infections

- Wipe in the correct direction, from front to back.
- Wash hands before wiping or washing
- Use a clean, gentle liquid soap because it is cleaner than bar soap
- When washing, clean the urethral area first to prevent bacterial contamination from other body parts.

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- When washing, clean the urethral area first to prevent bacterial contamination from other body parts.
- Drink extra water (recommend with 1 extra glass with each meal) & take vitamin C, cranberry.
- Instruct the patient to empty the bladder after intercourse. Clinicians may advise patients to take urinary antibiotic after intercourse.
- Menopausal patients to use estrogen cream

Key points

- The definition (3 per year or 2 in 6 months) & management of rUTIs & A/B prophylaxis strategies, were mostly consistent across guidelines & emphasised the importance of doing urine cultures & not treating asymptomatic bacteriuria (ASB).
- Clinicians should use first-line therapy (i.e. nitrofurantoin/TMP-SMX, Fosfomycin) for as short a duration as reasonable, no longer than 7 days & should not perform a post-treatment test of cure in asymptomatic patients.
- Clinicians may offer cranberry prophylaxis for women with rUTIs & should recommend vaginal estrogen therapy in peri & post-menopausal women.

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Self care is
not a luxury.
It's a necessity.

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Pruritis Vulvae : An In-depth Review

Introduction

Vulval pruritus, or itching of the external female genitalia, is a common clinical condition that can significantly impact a woman's quality of life. It is often a symptom of various underlying conditions rather than a diagnosis in itself. The severity of vulval pruritus can range from mild discomfort to intense, persistent itching that interferes with daily activities and sexual function. It is a condition that requires a thorough diagnostic approach to determine the underlying etiology and to implement effective management strategies.

This article will discuss the multifactorial etiology of vulval pruritus, the diagnostic work-up, treatment options, follow-up strategies, and the newer techniques that have emerged for its management.

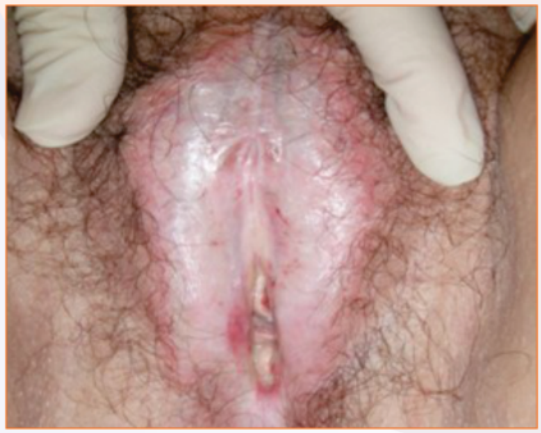


FIG. 1 Clinical image in a postmenopausal women

Etiology

Vulval pruritus can result from a wide variety of conditions, categorized into primary and secondary causes1. Primary causes refer to localized vulval conditions, while secondary causes involve systemic or dermatologic diseases that affect the vulva. Understanding the root cause of pruritus is crucial for appropriate treatment.

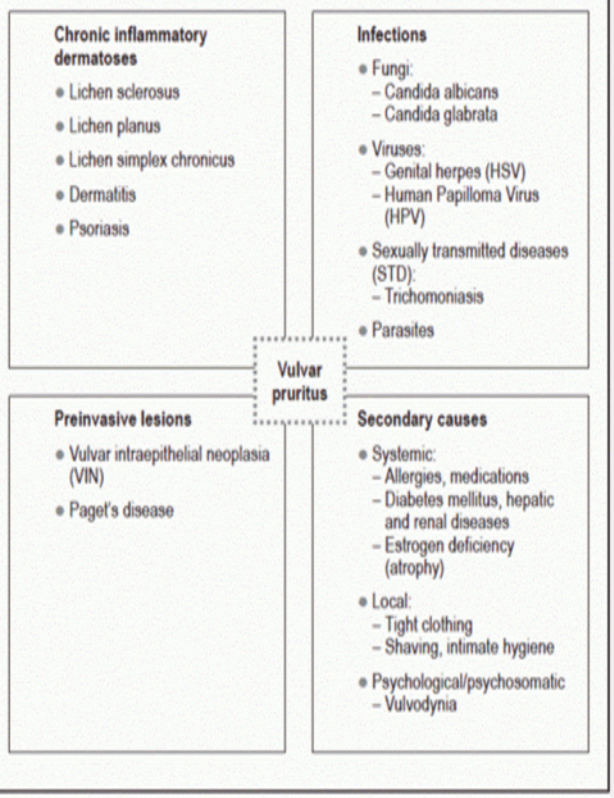
Primary Causes:

1. **Infections**
   - **Vulvovaginal Candidiasis:** One of the most common causes, caused by overgrowth of Candida albicans. It is often accompanied by discharge, erythema, and swelling.
   - **Bacterial Vaginosis (BV):** A shift in the normal vaginal flora can lead to itching, foul-smelling discharge, and discomfort.
   - **Trichomoniasis:** A sexually transmitted infection caused by Trichomonas vaginalis.
   - **Herpes Simplex Virus (HSV):** Recurrent outbreaks may cause pruritus along with lesions.
2. **Dermatological Conditions**
   - **Contact Dermatitis:** Allergic or irritant reactions to products like soaps, perfumes, or sanitary pads can cause localized itching and erythema.
   - **Lichen Sclerosus:** A chronic inflammatory skin condition leading to atrophic, white patches on the vulva, causing intense itching.
   - **Lichen Planus:** An autoimmune disease causing purple, flat, itchy papules that may affect the vulva.
   - **Psoriasis:** A chronic skin condition that may involve the vulva, characterized by scaly plaques.
3. **Vulvar Cancer:** Though rare, cancer of the vulva, particularly squamous cell carcinoma, may present with pruritus, ulceration, or bleeding.

Secondary Causes:

1. **Systemic Conditions:**
   - **Diabetes Mellitus:** High glucose levels predispose to fungal infections like candidiasis.
   - **Hypothyroidism:** Can lead to dry, itchy skin, including the vulva.
   - **Hepatic or Renal Disease:** Both conditions can cause generalized pruritus due to accumulated toxins.
2. **Hormonal Changes:**
   - **Menopause:** Reduced estrogen levels can lead to vaginal atrophy and subsequent itching.
   - **Pregnancy:** Hormonal fluctuations during pregnancy may lead to pruritus, including pruritic urticarial papules and plaques of pregnancy (PUPPP).

FIGURE 2



Common differential diagnoses of vulvar pruritus

Investigations

When investigating vulval pruritus, a systematic and step-by-step approach is required to identify the underlying cause. The investigation should start with a detailed history, including the onset, duration, and associated symptoms, followed by a physical examination.

Step 1: Clinical History and Examination

- **Duration of symptoms:** Acute or chronic?
- **Associated symptoms:** Pain, burning, discharge, swelling, or changes in appearance.
- **Medical history:** Previous infections, systemic diseases, or skin conditions.
- **Use of medications:** Antibiotics, topical agents, or hormonal therapies.
- **Sexual history:** Unprotected sex or potential exposure to sexually transmitted infections (STIs).

Step 2: Laboratory Tests

1. **Microscopy and Culture:**
   - **Vaginal Swab:** Used to identify infections such as candidiasis (KOH preparation for yeast), bacterial vaginosis (gram stain), or trichomoniasis.
   - **Polymerase Chain Reaction (PCR):** For herpes simplex virus or human papillomavirus (HPV) DNA testing.
2. **Patch Testing:**
   - Performed in suspected cases of contact dermatitis or allergic reactions to personal hygiene products, including soaps, lotions, or sanitary pads.
3. **Biopsy:**
   - In cases where there is a suspicion of dermatologic conditions like lichen sclerosus, lichen planus, or vulvar cancer, a biopsy is required to confirm the diagnosis.
4. **Blood Tests:**
   - **Complete Blood Count (CBC):** To check for any signs of infection or anemia.
   - **Thyroid Function Tests:** To rule out hypothyroidism.
   - **Blood Glucose Levels:** To check for diabetes.

Step 3: Imaging

- **Colposcopy:** In cases where there is an abnormal lesion, a colposcopy is used for detailed visualization of the vulva.
- **Ultrasound:** If there is suspicion of a deep pelvic pathology, such as ovarian cysts, an ultrasound may be used.

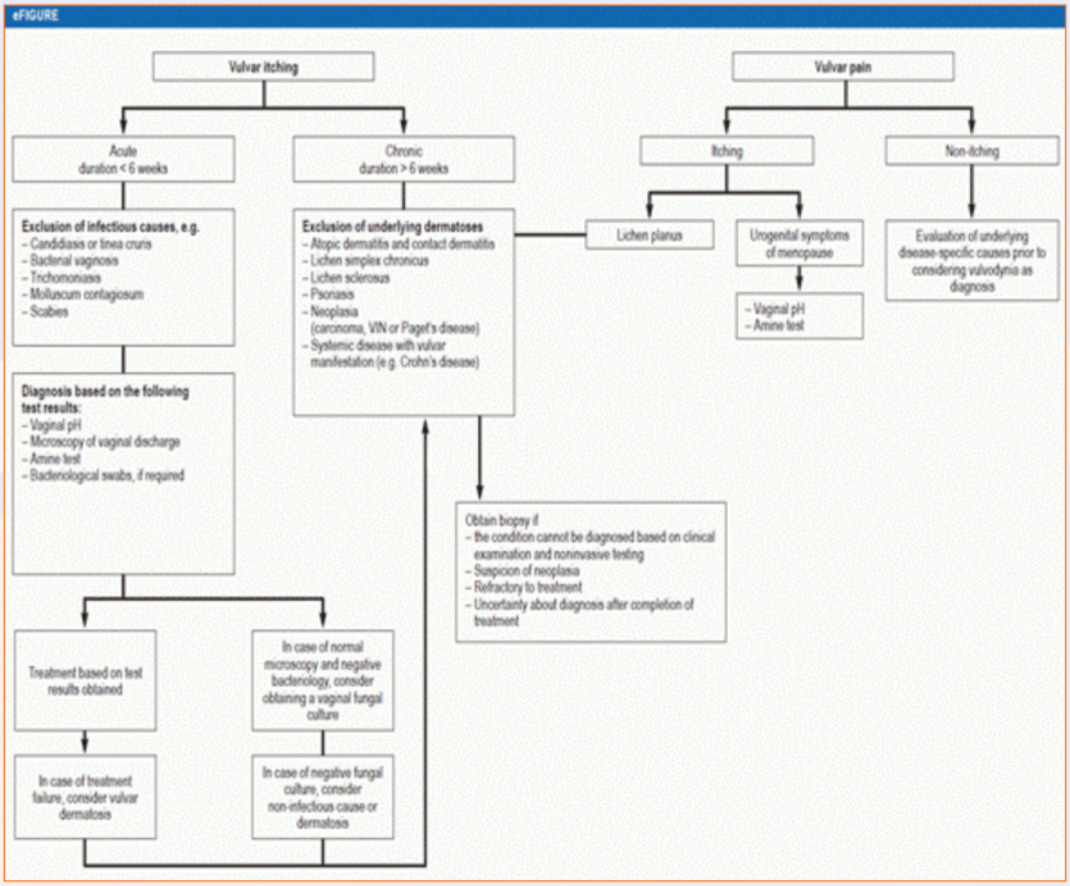


FIG. 3 Workup and Diagnosis of pruritis vulvae

Management

Management of vulval pruritus is based on the underlying cause. A step-wise approach is essential to alleviate symptoms, treat the causative factor, and prevent recurrence.

General Measures[2]:

- **Hygiene:** Advise patients to avoid douching or using scented products, as they may worsen irritation.
- **Moisturization:** Regular use of emollients can reduce dryness and soothe itching, especially in cases of vaginal atrophy or dermatitis.
- **Cotton Underwear:** Wearing loose, cotton underwear can help reduce irritation.
- **Avoidance of Irritants:** If allergies or irritants are identified, avoiding the offending products is crucial.

Specific Treatments[3]:

1. **Infections:**
   - **Vulvovaginal Candidiasis:** Antifungal creams (e.g., clotrimazole, miconazole) or oral antifungals like fluconazole.
   - **Bacterial Vaginosis:** Metronidazole or clindamycin topical treatments or oral antibiotics.
   - Trichomoniasis: Metronidazole or tinidazole, typically as a single-dose oral treatment.
   - Herpes Simplex Virus: Acyclovir or valacyclovir can be used to reduce the frequency and duration of outbreaks.
2. **Dermatological Conditions:**
   - **Lichen Sclerosus:** Potent topical steroids like clobetasol can help reduce inflammation and itching.
   - **Lichen Planus:** Steroids (topical or oral), tacrolimus, or phototherapy may be used.
   - **Psoriasis:** Topical corticosteroids, vitamin D analogues (e.g., calcipotriene), or phototherapy.
3. **Vulvar Cancer:** Early-stage vulvar cancer may require excisional surgery, while advanced cases may need radiation therapy or chemotherapy.
4. **Hormonal Therapy:**
   - For menopausal women, local estrogen therapy (vaginal creams, rings, or tablets) can alleviate vulvar dryness and pruritus.

- Oral contraceptives: May help in cases related to hormonal fluctuations.

Adjunctive Therapies:

- **Antihistamines:** Used for symptomatic relief of itching, especially if there is a suspected allergic component.
- **Topical Anaesthetics:** Such as lidocaine, for localized pain or pruritus relief.

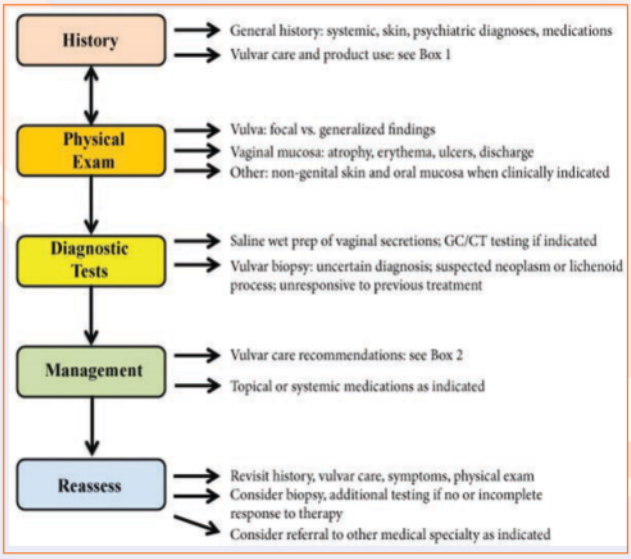
Newer Techniques of Treatment[4]

In recent years, there have been advancements in the management of vulval pruritus, especially in chronic or refractory cases.

1. **Laser Therapy:** Fractional CO2 laser treatment has shown promise in treating conditions like lichen sclerosus and vaginal atrophy by stimulating collagen production and improving skin elasticity and moisture.
2. **Phototherapy:** Narrowband UVB phototherapy is being used to treat chronic inflammatory conditions like lichen planus and psoriasis.
3. **Immunomodulators:** The use of topical tacrolimus, an immunosuppressant, is gaining traction for conditions like lichen planus and lichen sclerosus.
4. **Stem Cell Therapy:** Though still in the experimental phase, stem cell injections are being investigated for their potential in regenerating the vulvar skin in conditions like lichen sclerosus.

Follow-Up

Follow-up care is essential, especially in chronic cases or conditions like lichen sclerosus or vulvar cancer. Patients should be scheduled for regular visits to assess treatment efficacy, monitor for side effects, and adjust the treatment plan as needed. The frequency of follow-up depends on the underlying cause:



- **For infections:** Follow-up is usually not required unless symptoms persist.
- **For dermatological conditions:** Every 3–6 months for monitoring treatment response, especially for lichen sclerosus or lichen planus.
- **For cancer:** Every 3–6 months for the first 2 years, then annually thereafter.

Key Points

- **Treat the Cause:** Use antifungal, antibiotic, or antiviral medications for infections; corticosteroids or immunomodulators for inflammatory conditions.
- **Symptom Relief:** Apply topical emollients or antihistamines to soothe itching.
- **Hormonal Therapy:** Manage atrophic vaginitis with estrogen creams in postmenopausal women.
- **Lifestyle Changes:** Avoid irritants like scented products, wear breathable cotton underwear, and maintain proper hygiene.
- **Referral:** Seek specialist care for persistent symptoms or suspected malignancy.

Red Flags

- Persistent symptoms despite treatment
- Unexplained lesions, ulcers, or nodules
- Symptoms associated with systemic signs (e.g., weight loss, fatigue)
- Postmenopausal women with new-onset pruritus
- Early diagnosis and tailored treatment are crucial for effective management and to prevent complications such as chronic irritation or malignancy.

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Urogenital Infections in Postmenopausal Women

Introduction

Urogenital infections are a significant health concern in postmenopausal women, affecting their quality of life and overall well-being. The levels of estrogen fluctuate as menopause approaches, leading to dynamic hormonal changes that differ from woman to woman. Clinically, this leads to hypoestrogenic state and changes in the vulva, labia, vestibule, vagina, urethra, and/or bladder in up to 60% of postmenopausal women[1], making them more susceptible to infections. The urinary tract and vaginal microbiome play major roles in health and disease. Diagnosing genitourinary infections can be challenging as it is important to distinguish infection versus asymptomatic presence of bacteria to spare patients unnecessary and potentially harmful courses of antibiotics[2]

Causes and Risk Factors

Several changes in the urogenital tract during menopause make women susceptible to catch infections. Apart from general risk factors of elderly age group like diabetes, functional disability, prolapse etc., postmenopausal women can have following specific causes and risk factors.

1. **Urogenital atrophy:** The major cause of urogenital atrophy in menopausal women is estrogen loss. The decrease in estrogen levels causes the substantial reduction in epithelial proliferation leading to vaginal walls to become thinner, drier, and less elastic and making it more susceptible to infections[3].
2. **Change in microbiota:** The physiological changes associated with menopause can perturb the vaginal microbiota in ways that can result in unfavourable shifts in Lactobacillus-dominance. Lactobacilli ferment glycogen and create lactic acid, which is inhibitory to other bacteria, therefore maintaining a protective vaginal microbiome and preventing dysbiosis and infection. Lactobacilli also maintain vaginal health by preventing adherence of uropathogens to the vaginal epithelium. The hypoestrogenic state of menopause can directly impact the vaginal microbiota and have been associated with decreased glycogen deposition. These changes can result in lower levels of vaginal Lactobacillus spp. and higher levels of facultative and strict anaerobic bacteria (i.e., Anaerococus, Peptoniphilus, and Prevotella spp, etc.)[4].
3. **pH imbalance:** The normal vaginal pH is acidic, ranging from 3.8 to 4.5. However, the resultant Lactobacillus-deficient state during menopause can manifest with elevated vaginal pH levels >4.5 and a decreased ability of the vaginal microenvironment to protect against colonization by potential pathogenic micro-organisms (i.e., F. vaginae); thus creating an environment conducive to bacterial growth[5].
4. **Decreased immune function:** The decline in estrogen levels also affects the immune system, making it less effective in fighting off infections.
5. **Incontinence and poor hygiene:** Irreversible structural and functional changes occur in bladder, bowel, urethral, and neural control with aging. Atrophic changes and deterioration of tissue strength due to lack of hormones led to further worsening in postmenopausal ladies. Urodynamic

evaluation reveals a decrease in maximal cystometric bladder capacity and maximal urine flow rate, an increase in voiding time and post-void residual urine volume, and a decrease in muscle contraction rate owing to increased fibrosis in postmenopausal women[6].

Overall, the incidence of urgency, frequency, intermittency, urgency urinary incontinence, frequency of incontinence episodes, stress urinary incontinence, and fecal incontinence was found to be higher in elderly population and some unhealthy toileting behaviours (premature voiding, straining during voiding, etc.) are more prevalent in older women [7]. These ailment again make them prone to catch urogenital infection due to poor health and hygiene of urogenital region.

6. **Sexual activity:** While coitally-associated UTIs are less common in postmenopausal women, but those who are receiving estrogen replacement therapy[8] and are sexually active, continue to be at risk for Bacterial vaginosis (BV)[9].

Additional specific risk factors for UTI in postmenopausal women include history of premenopausal UTI, cystocele, and blood group Ag secretory status[8].

Various Infections with differentiating features and Diagnostic Tests

1. **Bacterial Vaginosis (BV):** BV is a vaginal dysbiosis. It manifests due to reduction in number of lactic acid and hydrogen peroxide-producing lactobacilli and increase in number of facultative and strict anaerobic bacteria (i.e., Gardenerella spp, Prevotella spp, Fannyhessea vaginae (previously Atopobium vaginae), etc.) in the vaginal microbiome. Common presentation include a thin, homogeneous white-greyish vaginal discharge, vaginal odour often described as "fishy", and less frequently, vaginal irritation.

   A recent systematic review of the literature and meta-analysis reported that the prevalence among postmenopausal women ranges between 2%-57% (overall

   16.9%, in comparison to 23%-29% among premenopausal women.

   Traditional diagnostic methods Amsel criteria and Nugent score validated to diagnose BV, were originally developed using data from a college-age and pregnant women respectively and therefore do not have the account for the effect of hypoestrogenism on the vaginal pH and the composition of the vaginal microbiota in the setting of menopause. Thus, the clinicians must be cautious while using these methods and the whiff test may not be of great value to make a diagnosis of BV in post-menopausal women.

   Recently, highly sensitive and specific BV molecular diagnostics have ben introduced. Food and Drug Administration (FDA) has approved nucleic acid amplification tests (NAATs) for diagnosing BV. Although, diagnosing BV in terms of the composition of the vaginal microbiota by methods such as 16s rRNA gene sequencing or shotgun metagenomic sequencing could provide more clarity in symptomatic postmenopausal women on estrogen replacement therapy. But, these methods can be costly, time-consuming, and are not currently approved for use in clinical practice. Thus, due to lack of the optimal diagnostic approach and absence of the true epidemiologic impact, treatment of this infection in postmenopausal women must be driven by symptoms[9].

2. **Vulvoaginal Candidiasis (VVC):** The odds of developing VVC have found to decrease by 7% with each year of age after 57, as predicted by the low glycogen levels occurring in postmenopausal women. However, certain medications and comorbidities such as tamoxifen, antibiotics, sodium-glucose cotransporter-2 (SLGT-2) inhibitors, diabetes mellitus, hormone replacement therapy (HRT), and immunosuppressants have been found to increase the prevalence of VVC in the postmenopausal population[11].

3. **Urinary Tract Infection (UTI):** UTIs are bacterial infections that occur in the urinary tract. The classic symptoms of "cystitis" (a.k.a. lower tract UTI) include dysuria, frequency, urgency, and suprapubic pain. Older women may report other symptoms as well, including foul odor, incomplete emptying, constipation, hematuria, generally feeling "ill", and altered mental status. Alarm symptoms that should prompt clinicians to consider further diagnostic workup include gross hematuria, passage of tissue or feculent material in the urine, history of urogenital mesh-based procedure, or history of urologic malignancy. Fever, tachycardia, or costovertebral tenderness should raise concern for an upper tract UTI, such as pyelonephritis.

   Pelvic examination is not routinely required for postmenopausal women for infrequent, sporadic UTI. However, in case of uncertain diagnosis or frequent/recurrent UTI, the pelvic examination should be done to assess urogenital atrophic

changes and to rule out other findings such as suburethral masses, pelvic organ prolapse, foreign bodies, or fistulous tracts. Since gradual deterioration in detrusor muscle function is common with age, a post void residual volume should be obtained to rule out incomplete emptying as it can be a cause for UTI.

Urine dipstick tests, Urinalysis and standard urine culture are usual investigations done to diagnose UTI. Despite common use, the standard culture has limitations that it does not detect all relevant uropathogens. Therefore, new urine culture technique Expanded Quantitative Urinary Culture (EQUC) is available. It inoculates 100x more urine (100ìl) on diverse types of media, anaerobic conditions, varying temperatures, and time periods up to 5 days, with a lower threshold of detection than standard urine culture at 10 CFU/ml. This should be used only in rUTI patients with multiple negative standard urine cultures or symptoms that do not improve with standard urine culture-directed treatment.

Women with persistent relapses or reinfections despite preventative measures, continued infection with urea-splitting organisms, elevated serum creatinine, neurogenic bladder dysfunction, hematuria, concern for urinary tract malignancy, or a history childhood UTIs, renal calculi, or genitourinary surgery may benefit from cystoscopy and/or upper tract imaging.

In complex patients of rUTI with a history of a mesh-based surgery, urogenital tract abnormalities, a significant smoking history etc; a multi-disciplinary approach with the involvement of Female Pelvic Medicine and Reconstructive Surgery, Infectious Disease and Urology is necessary [8].

Treatment

The treatment of urogenital infections in postmenopausal women depends on the type of infection. The following treatment options may be used:

1. **Antibiotics:** Antibiotics are used to treat bacterial infections such as BV and UTIs are not different than women for reproductive age group. For BV; Metronidazole, Clindamycin, Tinidazole or Secnidazole are traditionally used and these therapies are aimed at restoring a lactobacillus-dominant vaginal microbiota. However, in postmenopausal women, concurrent estrogen replacement therapy will be required to support this restoration.

   For treatment of UTI, The antibiotic should be chosen based on prior culture results and community resistance levels when treatment is started without culture results; the antibiotic choice can be modified, if necessary, when the culture results become available. The benefits of immediate relief with empiric antibiotics must be carefully weighed against the possibility of unnecessary or inappropriate antibiotic use, potentially contributing to growing antibiotic resistance.

2. **Vaginal Estrogen therapy:** A recent meta-analysis concluded that vaginal estrogen could reduce the episodes of rUTIs in postmenopausal women in comparison to placebo but not the oral estrogen. It also lowers vaginal pH significantly without adverse events, including vaginal discomfort, irritation, burning, or itching.

   One study also reported that vaginal estrogen could reduce levels of urine interleukin 6 and reduce urine inflammatory scores, indicating the genitourinary inflammatory response and suppression of associated symptoms with local estrogen treatment.

   As its absorption is dose dependent and may be influenced by the delivery system, various formulations have been proposed. The preparations of vaginal estrogen therapy can be classified as low-, intermediate-, and high-dosage preparations.

   Low- dose vaginal estrogen is around 7.5 µg for vaginal rings and 10 µg for tablets. Long-term administration has been reported to possibly increase plasma estradiol levels not above the normal range of i.e. ≤ 20 pg/ml, suggesting that it is safe.

   Intermediate dose is 25 µg estradiol or 0.3 mg conjugated equine estrogen.

   High dose is 50–2000 ìg estradiol or 0.625–2.5 mg conjugated equine estrogen.

   Intermediate and high doses may result in plasma estradiol levels > 20 pg/ml. Usually, all vaginal estrogen preparations uses higher doses; however, no serious adverse events were reported, if used <1 year. It is also noteworthy that vaginal estradiol absorption is acute with peaks at about 8 h that return to baseline at 12 h[12]. Within the first 12 weeks of use, Lactobacillus is restored and there is an associated recovery of the host defences[13].

3. Vaginal or oral probiotics are currently not recommended for the treatment or prevention of vaginal dysbiosis and/or BV in any age group[9],

although recent studies indicates that continued usage for a longer period of time (>1 year) has more promising results and preferably a probiotic containing L. crispatus with concentration of 1×108 colony forming units should be recommended[13].

4. D-Mannose an over-the-counter monosaccharide sugar, acts in decreasing bacterial adherence to the bladder mucosa.
5. Methenamine salts prescribed as methenamine, enact their antibacterial properties via conversion to formaldehyde in acidified urine. A Cochrane review showed that methenamine prevent UTI in those with a normal urinary tract and a nonneuropathic bladder, with a low rate of adverse events.
6. Cranberry products which contain proanthocyanodin, have long been associated with bladder health; however a Cochrane review found that cranberry products had no significance.

Other treatments like antifungal etc. can follow usual recommendation and are not specific for postmenopausal women.

Prevention Strategies

Antibiotic resistance is an ongoing concern and repeated use of antibiotics can have untoward side effects, including gut dysbiosis and other conditions. Therefore, there is growing interest in "natural" treatments and prevention strategies. Following may be used to prevent urogenital infections in postmenopausal women:

1. **Practice of good hygiene and general urogenital care:** following instructions should be given
   - Advise the woman to quit smoking as smoking reduces oestrogen levels and decreases vaginal lactobacilli; which further aggravates irritative LUTs.
   - Avoid wearing tight-fitting underwear, pantyhose/tights, jeans or trousers as this may lead to sweating and skin irritation and preferably underwear made of natural fibres should be used.
   - Limit time in damp or wet swimming costumes or exercise clothing.
   - Wash clothing with non-perfumed or low-allergenic washing products.
   - Avoid using fabric softeners and consider double-rinsing underwear in clear water if symptoms persist.
   - Avoid the use of feminine hygiene sprays, perfumed wipes and douching.
   - Avoid scented panty-liners and toilet paper.
   - Avoid shaving or waxing the genital area, particularly if active infection is present.
   - Gently wash the skin of the genital area with plain water only. Soap alternatives are gentler on older skin and soap, liquid soap, bubble baths and shower gels are best avoided. Always pat dry as opposed to rubbing.
   - Adequate drinking (~ 2 to 3 litres a day) in distributed form and regular voiding (every 3-4 hourly) in daytime.
2. **Use of lubricants:** Regular use of vaginal moisturisers during intercourse, such as polycarbophil gel or hyaluronic vaginal gel 0.2% may help relieve general vulvovaginal dryness and thus, reduce the risk of vaginal trauma and infection. But, fact is that vaginal moisturizers, personal lubricants, douches, and spermicides suppress the growth of Lactobacillus in vitro.
3. **Maintaining a healthy diet:** Maintaining a healthy diet rich in fruits, vegetables, and whole grains can help boost the immune system and prevent infections.

Key Points

- Urogenital infections affects the quality of life and overall well-being of postmenopausal women.
- Main etiological factors include urogenital atrophy, change in microbiota and decreased immune function etc
- Treatment includes antibiotics, estrogen therapy, probiotics,D-Mannose .
- Risk can be reduced by practicing good hygiene, using lubricants, getting tested regularly, and maintaining a healthy diet.

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Each individual woman's body demands to be accepted on its own terms.

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Navigating Bacterial Vaginosis : From Diagnosis To Treatment

General Consideration

Bacterial vaginosis (BV) is the most common cause of abnormal vaginal discharge in women of reproductive age group. It is characterized by an imbalance in the vaginal microbiota, where the dominance of lactobacilli is disrupted and replaced by an overgrowth of anaerobic bacteria, including Gardnerella vaginalis, Atopobium vaginae, and Prevotella species[1]. This shift leads to an increase in vaginal pH and the production of volatile amines, which are responsible for the characteristic malodorous discharge associated with BV[2]. Previously, bacterial vaginosis was known as Gardnerella vaginitis, attributing the condition solely to Gardnerella bacteria.

The prevalence of bacterial vaginosis ranged from 4%, as reported among asymptomatic college-going females, to 61% of females when attended a sexually transmitted disease clinic[3]. Around the globe its prevalence varies from 20% to 60% among females[4]. Gardnerella vaginalis is detected in the vaginal tracts of upto 50% asymptomatic women, indicating that it is likely a natural part of the vaginal microbiota.

Pathophysiology

The pathophysiology of Bacterial Vaginosis (BV) is multifaceted, involving a shift in the normal vaginal microbiota from a healthy state dominated by Lactobacillus species to an overgrowth of diverse anaerobic and facultative bacteria. This imbalance, known as dysbiosis, increases vaginal pH and promotes the growth of pathogenic bacteria, creating an environment conducive to infection and inflammation.

1. Microbial Shift and Dysbiosis with altered vaginal pH

In a healthy vaginal microbiome, Lactobacillus species, such as Lactobacillus crispatus, Lactobacillus jensenii, and Lactobacillus iners, predominate. These bacteria maintain an acidic vaginal pH (around 3.8–4.5) through the production of lactic acid, which is toxic to many potential pathogens. Additionally, lactobacilli produce hydrogen peroxide and other antimicrobial peptides that further protect against harmful bacteria.

In BV, this Lactobacillus-dominant environment is disrupted leading to elevation of vaginal pH, often above 4.5. The number of Lactobacillus species declines, and anaerobic bacteria, including Gardnerella vaginalis, Prevotella spp., Atopobium vaginae, and Mobiluncus spp., become overrepresented. These bacteria produce volatile amines, such as putrescine and cadaverine, which contribute to the characteristic fishy odor of BV[5].

2. Biofilm Formation

Many of the bacteria involved in BV, such as Gardnerella vaginalis, have the ability to form biofilms on the vaginal epithelial surface. Biofilms are clusters of bacteria embedded in a self-made matrix of extracellular polymeric substances which protects the bacteria from both the host immune system and antimicrobial treatment, making BV particularly difficult to treat and contributing to its high recurrence rate[6].

3. Host Immune Response and Inflammation

The shift in microbiota in BV is accompanied by an altered immune response. The pathogenic bacteria induce an inflammatory response characterized by the recruitment of immune cells, such as neutrophils and macrophages, to the vaginal mucosa. This inflammatory response leads to the production of pro-inflammatory cytokines, such as

IL-1β, IL-6, TNF-α, and IL-8, which contribute to the symptoms of BV, such as discharge and odor. Chronic inflammation causes epithelial cell damage and disrupt the integrity of the vaginal epithelium, making it easier for pathogens to invade and persist[7]. Hormonal fluctuations, particularly during menstruation, pregnancy, or menopause, can affect the vaginal microbiome by altering the vaginal pH or immune environment[1].

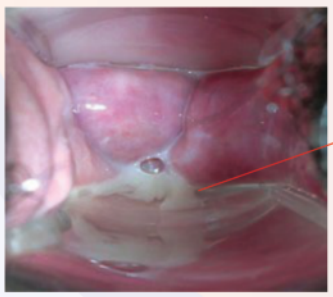
Recent studies suggest that the host's genetic predisposition may influence the immune response to BV. Variations in immune-related genes, such as those involved in the production of cytokines, can either promote or inhibit the inflammatory response and may influence the severity and recurrence of BV. Genetic variants in stress-related genes such as corticotropin-releasing hormone (CRH), receptor 1, receptor 2 and binding protein (CRH-BP) are associated with BV[8].

Clinical Features of Bacterial Vaginosis (BV)

Bacterial Vaginosis (BV) is characterized by:

1. Vaginal Discharge

The most common clinical symptom of BV is a homogenous thin, grayish-white vaginal discharge having a fishy odor which becomes more pronounced after sexual intercourse. This distinctive smell is attributed to the production of amines (such as putrescine and cadaverine).



2. Vaginal Irritation and Itching

Some women with BV may experience mild vaginal itching or irritation, although it is not as common as the discharge or odor. In more severe cases, there may be some discomfort or pain during urination or intercourse (dyspareunia).

3. Asymptomatic Cases

Upto 50% women are asymptomatic. However, the condition may be detected during routine gynecological exams or when testing for sexually transmitted infections (STIs).

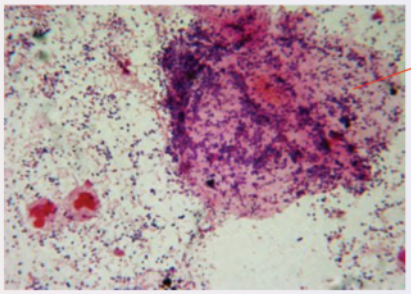
4. Increased Risk of Complications

Females with BV have increased risk of STIs such as HIV, chlamydia, gonorrhea, and trichomoniasis due to the altered vaginal environment. It has been associated with preterm labor, low birth weight, spontaneous abortion, postpartum infections. Increased risk of infertility and endometritis[9].

Diagnosis of Bacterial Vaginosis

1. Amsel Criteria (Clinical Criteria)

1.	Homogenous, thin, grayish-white vaginal discharge that coats the vaginal walls evenly
2.	Vaginal pH >4.5
3.	On a wet mount presence of e"20% clue cells (epithelial cells that are coated with bacteria, making them appear granular or stippled when observed under a microscope). **Clue cells are a hallmark of BV.**
4.	Positive "whiff test": A fishy odor is released when a drop of 10% potassium hydroxide (KOH) is added to the vaginal discharge (due to the production of amines).
*	**3 out of 4 criteria must be present to diagnose bacterial vaginosis** In Modified Amsel criteria, the presence of just 2 of the above factors, and it is equally diagnostic (4).



Microscopy showing clue cells on wet mount preparation

2. Nugent Scoring System (Microscopic Criteria)

The Nugent score is based on the examination of a Gram-stained vaginal smear under a microscope. This scoring system evaluates the relative abundance of Lactobacillus species, gram-positive rods, gram-negative rods, and cocci. This system has a higher level of specificity and sensitivity than the Amsel criteria.

Scoring system	Vaginal flora
Score 0–3	Normal vaginal flora with predominant Lactobacillus species
Score 4–6	Intermediate flora, suggestive of a mild disturbance

Score 7–10	Strongly indicative of BV, with an overgrowth of **anaerobic bacteria** (e.g., Gardnerella vaginalis, Atopobium vaginae).

3. **Hay/Ison's criteria :** Grading system for diagnosing BV by examining a gram stained vaginal smear.

Category	Vaginal flora
1	Normal flora (Lactobacillus only)
2	Intermediate (Lactobacillus = Gardnerella)
3	Bacterial vaginosis (Lactobacillus < Gardnerella)

4. **Culture**

While not commonly used for routine diagnosis, culture methods can sometimes be employed to identify specific anaerobic bacteria that may be responsible for BV. However, culturing Gardnerella vaginalis is not always definitive, as this bacterium is found in the vaginal flora of both healthy and infected women.

5. **Molecular Tests**

i) Real-time polymerase chain reaction (RT-PCR) and multiplex next-generation sequencing (NGS) are used in clinical practice as they allow quantitative detection and accurate identification of bacteria, associated with BV.

ii) A multiplex PCR test allows an established sequencing algorithm for BV detection and identifies Gardnerella spp. and Fannyhessea vaginae (formerly known as Atopobium vaginae), as well as lactobacilli species and other BV-associated microorganisms.

iii) Fluorescence in situ hybridization (FISH) simultaneously assess bacterial taxa composition and their spatial arrangement[10].

It is not typically employed as a first-line diagnostic tool in clinical practice.

5. **Additional Considerations**

STI Testing : Since BV increases the risk of STIs, healthcare providers may recommend testing for other STIs, such as chlamydia, gonorrhea, and HIV, in women diagnosed with BV.

Pregnancy Testing : If a woman is pregnant and presents with symptoms of BV, pregnancy-related complications (e.g., preterm labor) should be considered, and additional prenatal care may be required.

Treatment and Prevention of Bacterial Vaginosis (BV)

It focus on restoring the balance of the vaginal microbiota, alleviating symptoms, and preventing recurrence. Therapeutic approaches typically target the overgrowth of pathogenic bacteria and promote the restoration of Lactobacillus species, which are beneficial for maintaining vaginal health.

1. Treatment of Bacterial Vaginosis

A. Antibiotic Therapy

The cornerstone of BV treatment is antibiotic therapy, which aims to eliminate the overgrowth of pathogenic bacteria. The most commonly used antibiotics include:

Metronidazole (oral/topical/Suppositories/ Cream)

Oral Metronidazole: The most common first-line treatment for BV. Dose : 500 mg twice a day for 7 days.

Topical Metronidazole: Applied as a vaginal gel (0.75%) once daily for 5 days. It is effective in treating BV but may have a higher relapse rate compared to oral treatment.

Vaginal metronidazole suppositories are particularly beneficial for localized infection in the vaginal tract,administered as a gel or suppository once a day for 5–7 days.

Metronidazole disrupts bacterial DNA, leading to the death of anaerobic bacteria such as Gardnerella vaginalis and Atopobium vaginae.

Side effects : nausea, metallic taste, and, rarely, an allergic reaction.

Clindamycin (oral or topical)

Oral Clindamycin: Dose: 300 mg twice daily for 7 days.

Topical Clindamycin: Available as a vaginal cream (2%) to be applied once nightly for 7 days.

Clindamycin is effective against many anaerobic bacteria that cause BV but can sometimes lead to yeast infections (candida) as it disrupts the normal microbial balance.

Tinidazole (oral)

Dose: 2 grams once daily for 2 days

It is effective against the same range of anaerobic bacteria as metronidazole but is more expensive and may have fewer side effects.

Secnidazole (oral)

Secnidazole is an alternative oral treatment for BV and is given as a single 2-gram dose. It is effective with fewer side effects than oral antibiotics like metronidazole or clindamycin.

B. Probiotics

The use of probiotics (specifically Lactobacillus strains) as adjunct therapy has gained attention as a means to restore the vaginal microbiome and reduce BV recurrence. Some studies suggest that oral probiotics or vaginal probiotics (e.g., Lactobacillus rhamnosus, Lactobacillus reuteri) may help increase lactobacilli in the vagina and prevent relapse, although more evidence is needed on their long-term effectiveness[11].

C. Treatment of Sexual Partners

Although BV is not classified as a sexually transmitted infection (STI), some studies suggest that treating sexual partners may help reduce the recurrence of BV in women with frequent relapses. Treatment is generally recommended in women with recurrent BV (more than 3 episodes per year) or in cases where a male partner is diagnosed with a urethral infection related to BV-associated bacteria.

D. Treatment for Pregnant Women

Pregnant women with BV are treated with oral or topical metronidazole as the first-line treatment. BV in pregnancy has been associated with an increased risk of preterm birth, low birth weight, and other complications, so it is important to manage and treat the condition during pregnancy.

Clindamycin can be considered if metronidazole is contraindicated (e.g., due to intolerance). Both antibiotics are considered safe for use during pregnancy, although the benefits and risks should be assessed by the healthcare provider.

E. Recurrent BV Treatment

BV has a high recurrence rate, even after successful treatment. Studies suggest that prolonged metronidazole therapy (either oral or topical) or maintenance therapy with probiotics may help reduce recurrence. In cases of recurrent BV (more than 3 episodes in 12 months), continuous or intermittent treatment with antibiotics or probiotics may be considered.

2. Prevention of Bacterial Vaginosis

It involves avoiding risk factors that contribute to the imbalance of the vaginal microbiota by lifestyle changes and strategies which may reduce the risk of developing BV.

A. Good Vaginal Hygiene

Avoid douching:The vaginal environment is self-regulating, and douching may disrupt this balance, making it easier for harmful bacteria to overgrow.

Use of mild, unscented products: Avoiding scented soaps, sprays, or vaginal products is recommended as they irritate the vaginal lining leading to disruption of natural flora which increases the risk of infections like BV.

Proper wiping techniques: Wiping from front to back of the perineal area to avoid transferring bacteria from the rectal area to the vagina.

B. Safe Sexual Practices

Although BV is not directly classified as an STI, unprotected sex with a new or multiple partners is a risk factor for BV. Barrier methods, limiting sexual partners and practicing monogamy reduces the recurrence of BV, especially in women who have frequent episodes.

C. Hormonal Contraception

In women at risk for BV, the use of hormonal contraceptives that stabilize estrogen levels may help maintain a healthy vaginal environment. Recommendations should be tailored to each individual's health status and risk factors.

D. Probiotic Supplementation

Probiotics, particularly Lactobacillus species, helps to restore the vaginal flora after antibiotic treatment. Oral or vaginal administration of probiotics has shown some benefit in maintaining the dominance of lactobacilli and preventing recurrence of BV[11].

E. Avoid Smoking

Smoking is a risk factor for BV, and women who smoke are more likely to develop BV and have recurrent infections. Smoking cessation can reduce the risk of BV.

F. Diet and Lifestyle

High-fiber diet and lower intake of refined sugars helps to promote healthy vaginal microbiota. Regular physical activity can support immune function and decrease susceptibility to infections.

3. Follow-Up and Monitoring

Women, treated for BV, should be followed-up to ensure that the infection has resolved. If symptoms persist or recur within a few weeks, additional treatment may be necessary.Recurrent infections may require long-term or maintenance treatment.

Key Points

1. Bacterial vaginosis (BV) is the most common cause of abnormal vaginal discharge in women of reproductive age group occurring due to a disruption of lactobacilli and an overgrowth of anaerobic bacteria, including Gardnerella vaginalis.
2. The treatment of BV focuses on eradicating the overgrowth of pathogenic bacteria and restoring the natural Lactobacillus-dominated vaginal microbiome, typically through the use of antibiotics like metronidazole and clindamycin.
3. Probiotics may be considered as adjunct therapy to restore the vaginal flora and prevent recurrence.
4. Prevention strategies include maintaining good vaginal hygiene, practicing safe sex, using hormonal contraceptives appropriately, and avoiding risk factors such as smoking and douching.

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UTI in Pregnancy

Introduction

Urinary tract infection (UTI) is one of the most common perinatal complications, affecting approximately 8% of pregnancies[1,2]. According to the World Health Organization (WHO) estimates, various infections during pregnancy account for 10.7% of pregnancy-related maternal deaths worldwide. Recent studies estimate that most of these infections, almost 28%, are located in the urinary tract[2].

These infections represent a spectrum, from asymptomatic bacteriuria to symptomatic acute cystitis, to the most serious, pyelonephritis.

Known risk factors for UTI in pregnancy include diabetes, polycystic kidney disease, congenital abnormalities of the urinary tract, sickle cell disease, and recurrent UTI.

The presence of UTIs has been associated with adverse pregnancy outcomes, including increased rates of preterm delivery and low birth weight.

Predisposing Factors

The anatomy of the urinary tract undergoes significant changes during pregnancy, with hormonal and mechanical factors such as ureteral dilation, dilation of the renal calyces, and urinary stasis, all of which predispose pregnant patients to urinary tract infections (UTIs). Progesterone relaxes smooth muscles, and the gravid uterus compresses the bladder, decreasing bladder capacity. Vesicoureteral reflux, increased residual urine in the bladder, and urinary stasis are all contributing factors. The patterns of spread of UTIs in pregnancy often involve ascending infection from the lower urinary tract, facilitated by reduced urethral tone and altered bladder dynamics due to pregnancy changes. Hormonal changes also affect the vaginal flora, potentially increasing the colonization of uropathogens. Moreover, a decrease in cell-mediated immunity may impair the body's ability to combat bacterial infections effectively. Together, these factors create an environment conducive to the development of UTIs in pregnancy.

Bacteriology

UTIs are commonly caused by ascending movement of bacteria that colonize the lower gastrointestinal and genitourinary tract, particularly E. coli or other gram-negative bacteria[9].

The organisms that cause UTIs during pregnancy are the same as those found in nonpregnant patients. Escherichia coli accounts for 80 to 90 percent of infections. Others, such as Proteus mirabilis and Klebsiella pneumoniae, are also common. Gram-positive organisms, such as group B streptococcus and Staphylococcus saprophyticus, are less common causes of UTI. Other less common organisms include enterococci, Gardnerella vaginalis, and Ureaplasma ureolyticum (9).

Classification

Lower Urinary Tract Infections

- Asymptomatic bacteriuria (2-10% prevalence) (7)
- Acute cystitis (1-4%) (7)

Upper Urinary Tract Infections

- Pyelonephritis (1-2% prevalence) (7)

Asymptomatic Bacteriuria (ASB)

ASB is defined as >100,000 organisms/mL on a clean catch urinalysis obtained from an asymptomatic patient. A midstream urine culture is recommended for

ASB screening[3,4].

If ASB is untreated in pregnancy, the rate of subsequent UTI has been quoted at approximately 25%[7].

ASB is seen more frequently in parous women and women of low socioeconomic status. Women who are carriers of sickle cell trait also have a higher incidence of ASB[7].

Due to both the high rate and potential seriousness of pyelonephritis, it is recommended by most prenatal guidelines that every pregnant person be screened for ASB once early in prenatal care, either in the first or second trimester.

Treatment

A 5–7-day course of targeted antibiotics is prescribed to treat ASB with colony counts of 100,000 CFU/mL or higher[3,8]. There is insufficient evidence to recommend for or against repeat screening after appropriate treatment of an initial episode of ASB[3,7].

Acute Cystitis or Urethritis

Acute cystitis is distinguished from asymptomatic bacteriuria by the presence of symptoms such as dysuria, urgency, and frequency in afebrile patients with no evidence of systemic illness[2]. Up to 30 percent of patients with untreated asymptomatic bacteriuria later develop symptomatic cystitis[1,3,8].

Diagnosis

Patients with symptoms of acute cystitis should be evaluated with a urine culture. UTI should be suspected based on the presence of symptoms like burning dysuria, urgency, frequency, suprapubic pain, and may be supported by a positive urinalysis result. The diagnosis is confirmed by urine culture showing 100,000 CFU/mL or more[1,3,7].

Treatment

Acute cystitis in pregnant individuals is treated with a 5–7-day course of a targeted antibiotic (6, 8). If empiric therapy is started before culture and sensitivity results are available, amoxicillin or ampicillin regimens should be avoided due to high rates of resistance in Escherichia coli to these antibiotics in most areas.

Choice of antibiotic treatment is dependent not only on culture results but also susceptibility and safety profiles, and includes nitrofurantoin, â-lactams, sulfonamides, and fosfomycin.

Antibiotic choices for ASB or cystitis in pregnancy may include the following:

- **First-line therapy**[5,7]

  1. Nitrofurantoin 50 mg orally BID for 5 to 7 days
     - Low bacterial resistance rates
     - Therapeutic tissue levels obtained in the bladder
     - Use in the first trimester of pregnancy is reasonable if no alternatives are available
     - Not used for treatment of pyelonephritis due to inadequate tissue levels in the kidney
     - Avoid in patients with G6PD deficiency
  2. β-lactams
     - E. coli shows a high degree of resistance
     - Amoxicillin clavulanate 500/125 mg orally BID for 5 days
     - Fosfomycin 3 grams orally once
     - Not used for treatment of pyelonephritis due to inadequate tissue levels in the kidney
  3. Cephalexin 250 mg to 500 mg orally BID for 5 to 7 days

Nitrofurantoin can be given in the first trimester if no appropriate alternatives are available. Use of nitrofurantoin in the second and third trimesters can continue as first-line treatment for UTI[6,7,8]. Cephalosporins are the most common antibiotics used for pregnant patients with UTIs; however, over a quarter of E. coli isolates have known resistance to third-generation cephalosporins.

There is insufficient evidence to guide management after acute cystitis treatment in pregnancy. A urine culture can be repeated 1–2 weeks after completing treatment for acute cystitis or evaluated only if symptoms recur.

Pyelonephritis

Pyelonephritis is one of the most common reasons for hospitalization in pregnancy. Untreated pyelonephritis can lead to severe maternal and obstetric complications, including preterm labor and delivery, sepsis, septic shock, and ARDS[1,3,4].

Diagnosis

Pyelonephritis should be suspected in the presence of fever of 38.0°C or higher and urine studies suggesting UTI, with additional symptoms of upper genitourinary tract infection (such as flank pain or costovertebral angle tenderness) supporting the diagnosis.

When pyelonephritis is suspected in pregnancy, laboratory analysis should include a complete blood count (CBC), electrolytes, and serum creatinine. Tailored

studies should be included as appropriate to exclude other causes of the patient's symptoms, such as amylase and lipase if pancreatitis is considered a diagnosis[5,7]. If sepsis is suspected, lactic acid and blood cultures should be obtained. All cultures should be obtained as soon as possible and before starting antibiotic therapy.

Treatment

Pyelonephritis in pregnancy should always be managed in the inpatient setting. Empiric antibiotic therapy should have adequate renal tissue penetration and be targeted against the most likely pathogens. Antibiotic therapy should be adjusted as needed based on urine culture and sensitivity. Parenteral antibiotics should be continued until the patient is clinically improving. Patients should complete a total of 14 days of antibiotic therapy.

Antibiotic choices for pyelonephritis in pregnancy may include the following:

1. Ceftriaxone or cefepime 1g IV every 24 hours[6,7,8]
2. Ampicillin 2 g IV every 6 hours PLUS gentamycin 1.5 mg/kg IV every 8 hours (or 5 mg/kg IV every 24 hours)[7]
3. Aztreonam (for use in patients with β-lactam allergy) 1g IV every 8 hours to 12 hours[7]

There is insufficient evidence to guide management after treatment of pyelonephritis in pregnancy, and suppressive therapy can be considered for the remainder of the pregnancy, as for recurrent UTI.

Complications

The maternal and neonatal complications of a UTI during pregnancy can be devastating. Thirty percent of patients with untreated asymptomatic bacteriuria develop symptomatic cystitis, and up to 50 percent develop pyelonephritis. Asymptomatic bacteriuria is also associated with intrauterine growth retardation and low-birth-weight infants. Intensive care unit (ICU) admission may be required. Preterm premature rupture of membranes (PPROM) is not uncommon (6.3%), while preterm delivery (11%) and birth weight <2800 g (8.2%) are additional complications that may be seen due to UTI in pregnancy. Delivery within 72 hours has been noted in 7% of patients.

Pulmonary complications occur in up to 10% of pregnant patients undergoing treatment for pyelonephritis. This is due to endotoxin-mediated alveolar damage and may manifest as pulmonary edema or ARDS. Endotoxin release may lead to anemia, which typically resolves spontaneously following treatment. This is the most common complication seen with pyelonephritis, occurring in up to 25% of patients. Endotoxin release may also cause uterine contractions, and patients should be monitored for preterm labor, preterm delivery, and PPROM.

Recurrence and Prophylaxis

Recurrent UTI is defined as having two or more UTIs diagnosed during pregnancy and occurs in 4–5% of pregnancies.

The majority of UTIs are caused by gastrointestinal organisms. Even with appropriate treatment, the patient may experience a reinfection of the urinary tract from the rectal reservoir. The risk of developing pyelonephritis is the same as the risk with primary UTIs.

A single, postcoital dose or daily suppression with cephalexin or nitrofurantoin in patients with recurrent UTIs is effective preventive therapy.

A postpartum urologic evaluation may be necessary in patients with recurrent infections because they are more likely to have structural abnormalities of the renal system. Patients who are found to have urinary stones, who have more than one recurrent UTI, or who have a recurrent UTI while on suppressive antibiotic therapy should undergo a postpartum evaluation.

Prevention and Patient Education

Prevention of UTIs in pregnancy is essential for the health of both the mother and the fetus. Strategies for deterrence include promoting optimal hygiene practices, such as frequent handwashing and proper perineal care, to minimize the risk of bacterial colonization and ascending infection. Additionally, encouraging pregnant individuals to maintain adequate hydration and empty their bladder regularly can help prevent urinary stasis, which will decrease the likelihood of UTIs.

Preventive measures also involve screening for asymptomatic bacteriuria during prenatal visits and promptly treating positive cases to prevent progression to symptomatic infection. Education plays a key role in empowering pregnant individuals to recognize UTI symptoms and seek timely medical evaluation and treatment.

Key Points

- Urinary tract infections are a common yet preventable cause of maternal and fetal morbidity.
- Early screening, accurate diagnosis, and timely treatment are essential to mitigate risks.

- A UTI may manifest as asymptomatic bacteriuria, acute cystitis, or pyelonephritis.
- All pregnant women should be screened for bacteriuria and subsequently treated with appropriate antibiotic therapy.
- Acute cystitis and pyelonephritis should be aggressively treated during pregnancy.
- The prognosis of patients with UTIs in pregnancy largely depends on the promptness and effectiveness of diagnosis and treatment.
- Oral nitrofurantoin and cephalexin are good antibiotic choices for treatment in pregnant women with asymptomatic bacteriuria and acute cystitis, but parenteral antibiotic therapy may be required in women with pyelonephritis.
- Health care providers must be vigilant in managing UTIs, promoting preventive measures, and handling emerging challenges like antibiotic resistance.

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To achieve beauty,
A woman must first
achieve health.

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HIV in Pregnancy

Introduction

HIV, a retrovirus that weakens the immune system, affects 39.9 million people globally, with 53% being women and girls. 84% of pregnant women have access to antiretroviral therapy to prevent HIV transmission. Early HIV screening is crucial for preventing transmission and ensuring maternal and fetal health. HIV increases maternal susceptibility to infections and complications, including anaemia, tuberculosis, and opportunistic infections. Untreated HIV transmission during pregnancy, labour, and breastfeeding can lead to early childhood mortality and developmental delays. Early diagnosis, treatment, and prevention strategies are essential for safeguarding both mother and neonate's health and improving survival rates.

Basic Understanding of HIV

HIV enters the body through various means. It infects CD4 cells, binding to T-cells and immune cells, and replicating itself. The virus then replicates, producing new particles that weaken the immune system over time. HIV can progress through three stages: acute retroviral syndrome, clinical latency, and AIDS. In acute retroviral syndrome, the immune system tries to control the virus's replication. Clinical latency, or chronic HIV, is active but reproduces at low levels. AIDS is diagnosed when CD4 count drops below 200 cells per microliter of blood.

HIV-1 is the most prevalent global strain, causing most infections and rapidly progressing to AIDS without treatment, while HIV-2, primarily found in West Africa, is less transmissible and slows progression.

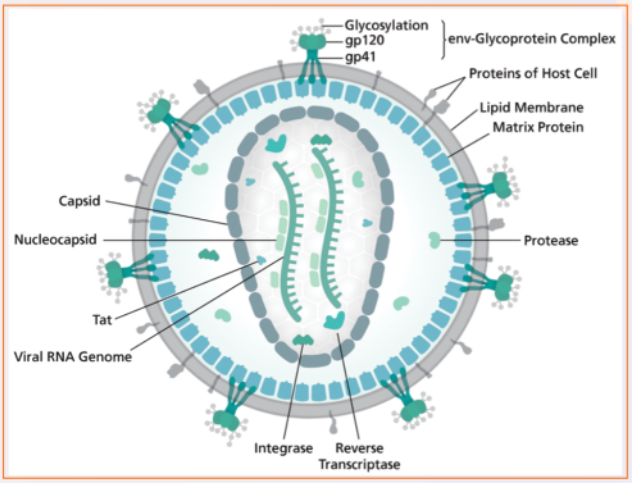

Fig 1: HIV Retrovirus: an enveloped virus, contains GP 120, 41facilitates entry into host cells, reverse transcriptase changes RNA into DNA, Integrase helps in viral DNA integration into host DNA. (Image courtesy: https://en.wikipedia.org/wiki/HIV)

HIV Transmission Routes

Sexual Contact: Unprotected sex, vaginal, anal, oral.

Blood : Sharing contaminated needles, transfusions, occupational exposure.

Vertical Transmission: Infected mother to child during pregnancy, childbirth, breastfeeding.

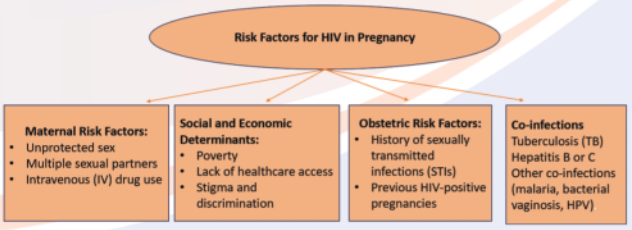

Fig 2: Risk factors for HIV in pregnancy

Table 1: Factors Influencing Vertical Transmission:

Maternal Viral Load	Higher viral load increases the risk of transmission; undetectable viral load (with effective ART) lowers the risk.
ART Adherence	Proper adherence to ART during pregnancy significantly reduces transmission risk.
Timing of ART Initiation	Starting ART before conception and continuing throughout pregnancy is most effective.
Type of Delivery	Caesarean section (C-section) may reduce transmission risk, especially with high viral loads. Vaginal delivery has a higher risk, it is wise to do caesarean only for obstetric indications.
CD4 Count	A low CD4 count (<200 cells/mm^3) is linked to higher transmission risk. Higher CD4 counts are associated with lower risk
Infections During Pregnancy	Co-infections (e.g., STIs, bacterial vaginosis) increase the risk of transmission.
Breastfeeding Practices	HIV-positive mothers with high viral loads may transmit the virus through breast milk; exclusive breastfeeding with ART reduces transmission risk.

Diagnosis of HIV in Pregnancy

Routine Screening Recommendations (Antenatal Care Protocols)

Universal HIV Testing: In many countries, including India, universal screening for HIV is recommended for all pregnant women during the first antenatal visit. In India, the National AIDS Control Organisation (NACO) follows a well-established protocol for HIV screening during antenatal care.

Method for HIV Testing During Antenatal Care in India

1. **Initial Screening:**

   The ELISA test is used in India for HIV testing during antenatal care, detecting antibodies. Blood samples are collected and performed at various facilities, with rapid tests providing results in 20 minutes. Positive ELISA test results indicate HIV antibodies in blood, indicating infection. However, false positives require confirmatory testing to rule out.

2. **Confirmatory Test:**

   The Western Blot test is the standard confirmatory test used in India. This test detects specific HIV proteins (p24, gp41, and gp120) in the blood, confirming the presence of the virus. The test is more specific than ELISA and can confirm whether a woman is indeed HIV-positive.

3. **Additional Testing (if needed):**

   PCR (Polymerase Chain Reaction) Test: In cases where further confirmation or monitoring of the virus is needed, the HIV RNA PCR test may be used. PCR directly detects the virus itself, not just the antibodies. This is especially useful in detecting HIV in infants born to HIV-positive mothers (within the first few months of life) or in women with very recent infections.

Timings for HIV testing in Pregnancy:

The National AIDS Control Organization (NACO) of India emphasizes routine and timely HIV testing during pregnancy to prevent mother-to-child transmission (PMTCT) of HIV.

Table 2: Timing of HIV Testing in pregnancy

Timing	when	Target group	rationale
First HIV test -	First trimester or as soon as pregnancy is confirmed	All pregnant women	Early diagnosis for timely initiation of ART Reduces maternal viral load and vertical transmission risk
Second HIV Test	Third trimester (28–36 weeks)	Women initially testing HIV-negative High-risk individuals (e.g., HIV-positive partner, history of STIs, substance use)	Detects seroconversion during pregnancy Allows preventive measures closer to delivery
Intrapartum Testing	During labour	Women with unknown HIV status- Women who missed earlier testing	Immediate detection enables intrapartum ART prophylaxis- Ensures neonatal prophylaxis
Postpartum HIV Testing	After delivery	Women who tested negative during pregnancy but remain at high risk (e.g., ongoing exposure, HIV-positive partner)	Identifies seroconversion postpartum Protects breastfeeding infant through maternal ART and monitoring

Management of HIV in pregnancy

Intervention to decrease MTCT:

1. Primary Prevention
2. Secondary prevention
3. Obstetric intervention
4. Immunological intervention
5. ART

1. **Primary Prevention:** The goal is to prevent HIV acquisition in women before or during pregnancy by using Pre-Exposure Prophylaxis (PrEP) for HIV-negative women at high risk, HIV counselling and education about transmission, safer sex practices, and risk reduction strategies, and promoting HIV testing and ART for HIV-positive partners to achieve viral suppression and reduce transmission risk.
2. **Secondary Prevention:** The goal is to prevent HIV progression and minimize transmission to the foetus in HIV-positive pregnant women by routinely testing them during antenatal care visits.

   Antiretroviral Therapy (ART): Immediately start antiretroviral therapy (ART) for HIV-positive pregnant women, regardless of CD4 count, to suppress viral load and reduce MTCT risk. Regular viral load and CD4 count monitoring ensures efficacy.
3. **Obstetric Intervention:** Advise mothers to quit alcohol, smoke, or illicit drugs during pregnancy and beyond, avoid artificial rupture of membranes during labour, and Efforts should be made to prevent chorioamnionitis.
4. **Immunological Intervention:** These are still under trial and no proven vaccine is available till now.
5. **ART:** Initially monotherapy was tried in year 1994 which was zidovudine, then Nevirapine was tried by NACO in India. But monotherapy are always in danger of developing Drug-resistant strains. These days multidrug regimens are recommended to avoid resistance.

Table 3: Various ART's:

Drug Class	Commonly Used Drugs	
Nucleoside Reverse Transcriptase Inhibitors (NRTIs)	Tenofovir Disoproxil Fumarate (TDF) - Lamivudine (3TC) - Abacavir (ABC)	Backbone of most ART regimens. Safe in pregnancy with regular renal and liver
Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)	Efavirenz (EFV)Nevirapine (NVP)	Efavirenz is safe after the first trimester. Nevirapine requires caution due to hepatotoxicity.
Integrase Strand Transfer Inhibitors (INSTIs)	Dolutegravir (DTG)Raltegravir (RAL)	Dolutegravir is preferred due to high efficacy and tolerability.
Protease Inhibitors (PIs)	Lopinavir/Ritonavir (LPV/r)Atazanavir/ Ritonavir (ATV/r)	Often used when INSTIs are contraindicated. Require careful monitoring for metabolic effects
Entry Inhibitors	Maraviroc (MVC)	Used less commonly; requires CCR5-tropism testing
Fusion Inhibitors	- Enfuvirtide (T-20)	Rarely used; requires subcutaneous administration.
Nucleotide Reverse Transcriptase Inhibitors (NtRTIs)	Tenofovir Alafenamide (TAF)	A newer version of Tenofovir with fewer renal and bone side effects.

Certain considerations may necessitate adjustments to ART during pregnancy to ensure maternal and fetal safety. ART should be initiated early in HIV-positive pregnant women, regardless of CD4 count or clinical stage. Women with renal impairment may need dose adjustments. Review concomitant medications, avoid drug interactions, and undergo resistance testing for regimen selection. Adherence counselling is crucial for viral suppression.

Antenatal management of HIV in Pregnancy

Antenatal care for HIV-positive pregnant women focuses on maternal health, prevention of mother-to-child transmission (MTCT), and minimizing risks to the foetus.

Management should be multidisciplinary including counsellor, obstetrician, physician, mental health support including psychologist and psychiatrist, and paediatrician.

Work up at first antenatal visit

Routine history taking, risk identification, general examination, blood investigations, screening for other STIs, counselling patients for pros and cons of pregnancy with existing HIV infection, screening for endocrine disorders, and baseline LFTs if patient is on protease inhibitors. If the patient chooses MTP, respectful abortion care and contraception should be provided, and imaging should be offered at 11-13 weeks gestation.

Second and third trimester

In the second trimester, HIV-positive women should receive regular visits to ART and ANC clinics, genetic sonograms, and routine tests. Nutritional and lifestyle

guidance includes dietary advice, iron supplementation, and safe sexual practices. Regular antenatal check-ups, monitoring for ART side effects, and during third trimester fetal growth ultrasounds are essential for monitoring high-risk pregnancies.

Table 4: ART in Antenatal Management:

Target population	Drugs	Remark
regnant or breastfeeding women with HIV.	Tenofovir + Lamivudine + Dolutegravir (TDF + 3TC + DTG) Fixed-dose combination (FDC) of TDF (300 mg) + 3TC (300 mg) + DTG (50 mg).	- Administered once daily. - Effective against HIV-1, HIV-2, and dual HIV-1 & 2 infections. - Suitable for women previously exposed to single-dose Nevirapine (NVP) or co-infected with TB or Hepatitis.- - Pregnant women should be informed about the benefits and risks of DTG to facilitate an informed choice

CDC recommend ARV medication to all pregnant women with HIV irrespective of their viral load and CD4 counts.

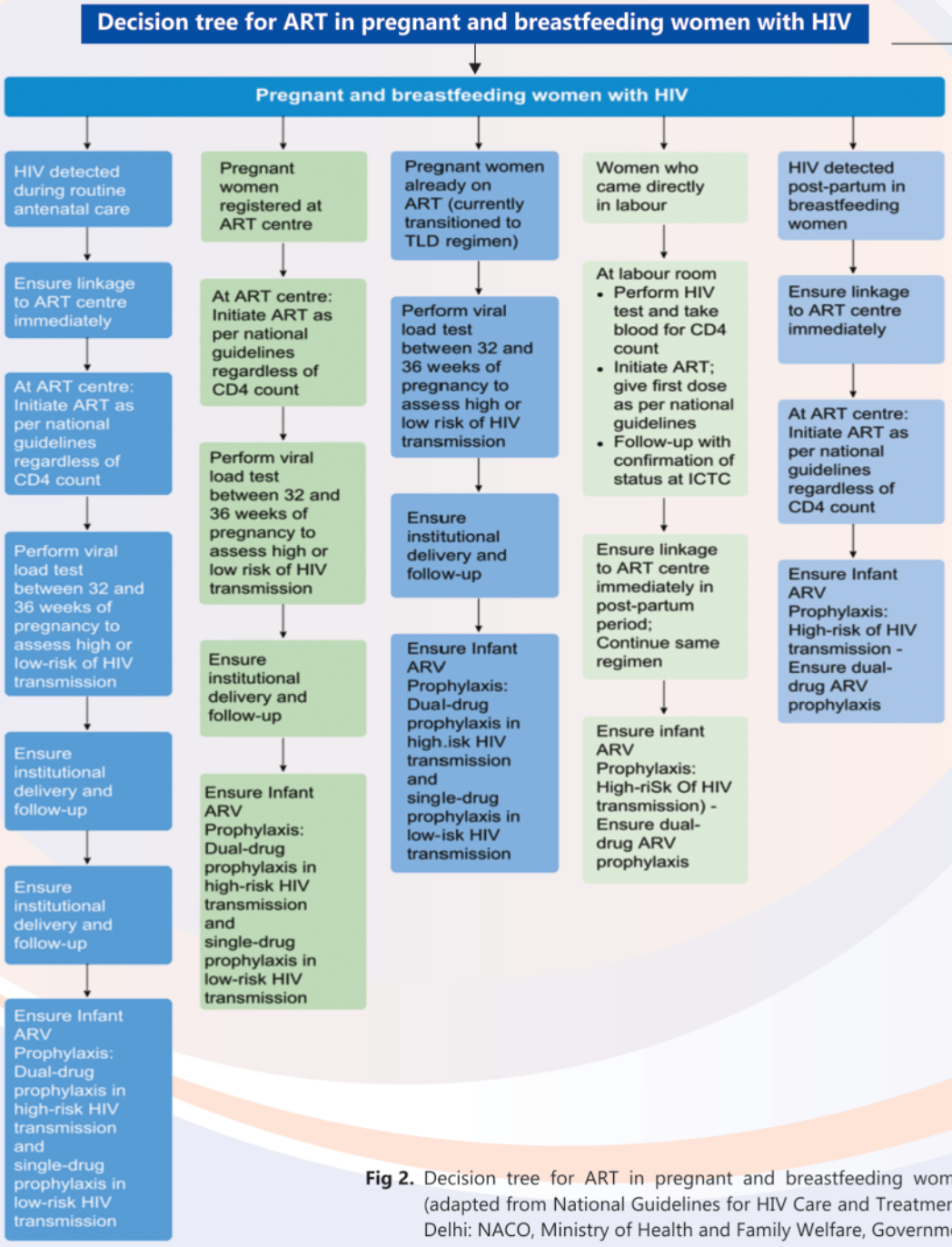

Fig 2. Decision tree for ART in pregnant and breastfeeding women with HIV (adapted from National Guidelines for HIV Care and Treatment, 2021. New Delhi: NACO, Ministry of Health and Family Welfare, Government of India

Intrapartum Management: Mode of delivery

Intrapartum management for HIV-positive women includes choosing the mode of delivery based on viral load. If the viral load is <1,000 copies/mL, vaginal delivery is safe. For viral loads e1,000 copies/mL or unknown, a planned caesarean section at 38 weeks is recommended to minimize the risk of mother-to-child transmission (MTCT).

Table 5: Intrapartum Management

Intrapartum Management	Remarks
General Principles	Reduce fetal exposure to maternal blood.Multidisciplinary team for care coordination.
Mode of Delivery	Viral Load < 1,000 copies/mL: Vaginal delivery recommended.Viral Load e 1,000 copies/mL: Elective caesarean at 38 to 39th weeks. Although it is now suggested that caesarean should be done only for obstetric reasons.
ART During Labor	Continue maternal ART.
Obstetric Practices	• Avoid ARM, • Minimize vaginal examination and use aseptic techniques • Avoid routine episiotomy as far as possible. • Avoid fetal scalp sampling, • unnecessary instrumental delivery. • Prevent infections like chorioamnionitis.
Neonatal Care	• It is advised to avoid suctioning a newborn with a nasogastric tube unless there is meconium staining of the liquor. • Start antiretroviral prophylaxis immediately after birth. • Minimize invasive procedures.

Postpartum care and follow up

Postpartum care for HIV-positive women is critical in managing their health and preventing mother-to-child transmission (MTCT) of HIV.

Table 6: Postpartum Management

1. Maternal care	• Continue ART as prescribed to suppress the virus and prevent resistance. • Check viral load at 6 weeks to assess ART effectiveness. • Contraception Counselling and Advise on safe contraception methods (e.g., IUDs, hormonal options). • Mental Health Support Offer support to address HIV-related stigma and anxiety. • Screening for Co-Infections Screen and treat for infections like TB, hepatitis, and STIs if not done prior during Antenatal care
2. Neonatal Care	• Neonatal Antiretroviral Prophylaxis Give HIV prophylaxis (e.g., Zidovudine) to the baby for 4-6 weeks. • test the baby at 6 weeks and 12 months for HIV. • Schedule visits to monitor the infant's health and HIV status • Ensure the baby receives all necessary vaccinations.
3. HIV Testing and Counselling	• Postpartum HIV Testing for Mother If not tested, offer HIV testing postpartum for diagnosis and care.
4. Breastfeeding Counselling	• Advise on breastfeeding based on viral load and ART status.
5. Long-Term Monitoring and Care	• Ongoing ART and Health Monitoring Monitor ART adherence, side effects, and any HIV-related issues. • Mental Health Follow-up Provide ongoing support for emotional challenges related to HIV. • Support for Future Pregnancies Offer counselling on ART use and reducing MTCT in future pregnancies.

Contraception in PLHIV

Estrogen-based contraceptives should be avoided, particularly in those taking Rifampicin for tuberculosis treatment.

Table 7 : Postpartum Contraception

Postpartum Contraception options	Interval contraception options
• Progestin-only pills (POP) • Depot Medroxyprogesterone Acetate (DMPA) • Implants • Copper IUD • Hormonal IUD • Male and Female condoms • Condom use with other methods (e.g., DMPA, implants) • Tubal ligation (permanent)	All these methods can be used as interval contraception as well.

Key Points

- HIV is a retrovirus that weakens the immune system by targeting CD4 cells.
- It is transmitted through sexual contact, blood, and mother-to-child transmission (MTCT).
- Pregnant women with HIV face risks like preterm birth, low birth weight, and increased maternal morbidity.
- Early screening, antiretroviral therapy (ART), and proper care are crucial to prevent MTCT and protect maternal and neonatal health.
- Universal HIV screening is recommended during pregnancy, with initial ELISA tests and confirmatory testing.
- High maternal viral load, low CD4 count, lack of ART adherence, co-infections, and certain obstetric factors increase the risk of MTCT.
- ART should be initiated immediately for all HIV-positive pregnant women to reduce viral load and prevent MTCT.
- Mode of delivery depends on viral load; vaginal delivery is safe for viral loads below 1,000 copies/mL, while caesarean section is recommended for higher loads.
- Effective contraception is crucial to prevent unintended pregnancies.

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TORCH Infections in Pregnancy

Introduction

TORCH infections represent a group of congenital infections with significant potential to impact fetal and neonatal health adversely. These infections include:

T : Toxoplasmosis

O : Others (Hepatitis B, Syphilis, Varicella Zoster, Zika, Malaria)

R : Rubella

C : Cytomegalovirus (CMV)

H : Herpes simplex virus (HSV)

Each pathogen's clinical impact varies depending on the timing of maternal infection during gestation and the pathogen's virulence. Common fetal abnormalities include central nervous system damage, microcephaly, hearing loss, and ophthalmic impairments. Early recognition and management are critical to mitigating adverse outcomes.

Significance of TORCH Infections

The severity of TORCH infections depends on the gestational age at exposure. Early detection is crucial, as fetal and neonatal complications include:

- Central nervous system abnormalities, such as ventriculomegaly and calcifications.
- Sensorineural hearing loss, which may present in late childhood.
- Ophthalmic impairments, including cataracts and chorioretinitis.

These complications can significantly impact the quality of life, necessitating a multidisciplinary approach for prevention, diagnosis, and treatment.

Diagnosis and Management of TORCH Infections

General Diagnostic Approach

1. **Clinical Suspicion:**

   TORCH infections should be suspected in pregnancies where specific markers or abnormalities are identified:

   - Fetal abnormalities on ultrasound, such as hydrops fetalis, brain lesions, or intrauterine growth restriction (IUGR).
   - Maternal systemic infections presenting with symptoms like rash or fever.
   - Pregnant woman with significant Contact with a Person of suggestive viral illness Should be investigated for Rubella and Parvo Virus B19 infection irrespective of whether they develop rash or not

   Routine Universal Screening: Routine Full Torch panel is not recommended in low risk asymptomatic pregnant women. It is not recommended to investigate TORCH infections as they are not responsible for recurrent miscarriage.

2. **Diagnostic Testing**

   - **Serological Testing:** Enzyme-linked immunosorbent assay (ELISA) for detecting IgM and IgG antibodies against TORCH pathogens.
   - **Avidity Testing:** Determines the timing of infection by measuring antibody strength, aiding in distinguishing recent from past infections.
   - **Molecular Diagnostics:** PCR testing of amniotic fluid provides a definitive diagnosis of fetal

infection.

- Elisa most cost effective test should be done in paired serum 4 week apart when first sample has been drawn during clinical illness.

IgM	Ig G	Significance
Negative	Negative	Patient is unexposed to infection. Not vaccinated for Rubella
Positive	Negative	Acute/Primary infection
Repeat serological test after 4 weeks IgG becomes positive with diminishing or absent IgM antibodies. It will suggest recent maternal infection.		
Negative	Positive	Past Infection.
• Do Avidity Test: o Low Avidity - Infection Within 3 months o High Avidity - Infection occurring Before 3 months		
Positive	Positive	Remote infection

Pathogen-Specific Considerations

Toxoplasmosis

Etiology and Transmission: Caused by Toxoplasma gondii, this protozoal infection is transmitted through the ingestion of undercooked meat or contaminated food.

Clinical Impact:

- Severe ocular and intracranial lesions are common with early gestational infections.
- Later infections are less severe but may still cause ocular involvement.
- Congenital Toxoplasmosis :

Gestational age	Transmission	Fetal infection	Clinical Manifestation
< 14 Week	< 10%	60%	Serious ocular and intracranial lesions
14 to 28	15-55%	25%	Ocular lesions (generally non serious)
>28 Week	55-80%	15%	Ocular lesions (rarely intracranial involvement)

Ultrasound Findings:

- Ventriculomegaly
- Intracranial calcifications
- Hepatosplenomegaly
- **Other finding** - Cases of Porencephaly, Macrocephaly, Ascites, Hydrops, Hepto-megaly, Splenomegaly, Intra-hepatic Calcification, Thickened Placenta have been described

Management

- Spiramycin (3 g/day): Used during the first trimester to reduce vertical transmission.
- Pyrimethamine-Sulfadiazine with folinic acid: Recommended for confirmed fetal infections (Positive PCR-Toxo in amniotic fluid) after 14 weeks.

Breastfeeding not contraindicated

Rubella

Etiology and Transmission: A viral infection (RNA Virus) spread via respiratory droplets. Vaccination has significantly reduced its prevalence.

Screening during pregnancy:

IgM	IgG	Significance
Negative	Negative	Vaccination of Woman
Positive	Negative	Recent Infection, delay pregnancy by 3 Month
Negative	Positive	Suggestive of immunity against Rubella

Amniocentesis for Viral RNA: Indications

- Maternal Primary Infection between 12 and 20 weeks.
- Uncertain Maternal infection before 20 weeks (in conclusive Maternal serological study)
- Documented maternal Re-infection 20 week (Low risk of transmission and congenital impairment)
- Ultrasound marker of Rubella Infection.

An interval between maternal infection and amniocentesis > 6 week is recommended with negative result < 21 weeks and high suspicion of maternal infection it is recommended to repeat the sample

Cordocentesis: If there is serological and clinical suspicion of maternal infection and RNA-PCR is negative in amniotic fluid or the result is not available cordocentesis can be performed to check fetal IgM. It should not be performed before 22 weeks as fetus rarely produce IgM below this gestational age

Congenital Rubella Syndrome:

Gestational Age	Vertical Transmission	CRS	Risk with Unknown transmission	Tipo de disorder
< 12 weeks	90%	80-90%	85%	Cardiovascular defects (mainly < 8 weeks) Ocular defects, deafness, psychomotor retardation Miscarriage 20%.
12-16 weeks	55%	30-35%	15%	Unilateral or bilateral deafness Retinopathy/microcephaly (occasionally)
16-20 weeks	25%	0%		Deafness (minimum risk)

Ultrasound Features:

- Cardiac anomalies
- Microphthalmia
- Hepatomegaly

Management:

- Termination of pregnancy may be considered for confirmed maternal infections in the first trimester due to high risk of serious Fetal Impairment and Sequalae.
- Prevention remains key, with pre-pregnancy vaccination offering lifelong immunity.

Breastfeeding not contraindicated

Cytomegalovirus (CMV)

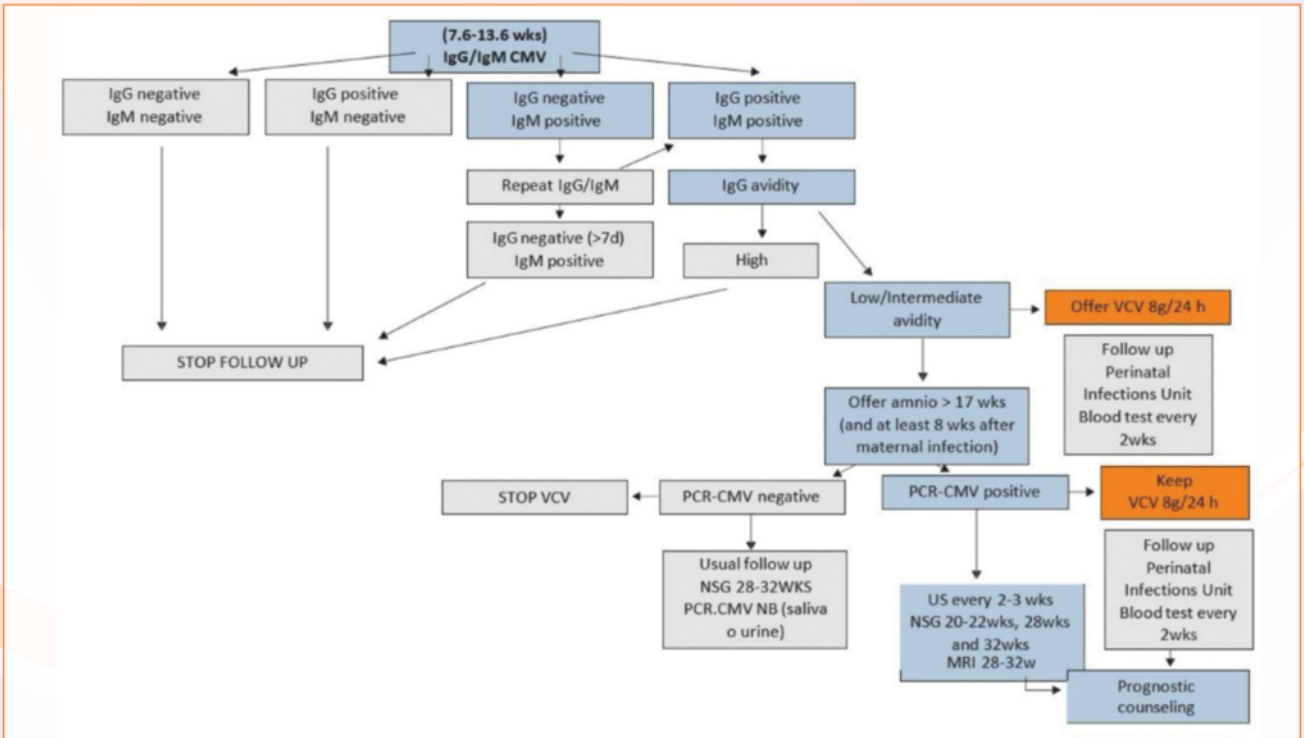
Epidemiology: CMV is the most common congenital infection globally.

Timing of infection	Vertical Transmission (VT)	Risk of fetal/ neonatal symptoms if VT	Risk of long-term severe sequelae (neurologic & hearing)	Risk of fetal/ neonatal symptoms with unknown VT
Pregestational (up to 10-12 weeks pre LMP)	5-6%	No data	No data	No data
Perigestational (4 weeks pre - 6 weeks post LMP)	21%	29%	No data but estimated > 1st trimester	6%
1st trimester	37%	19%	23%	7%
2nd trimester	40%	<1%	<1%	<1%
3rd trimester	66%	<1%	0%	<1%

Table: Risk of Vertical Transmission (VT) and central nervous system injury in the foetus and newborn and risk of severe neurological and hearing sequelae after primary maternal CMV infection, Source: chatzakis et al., AJOG 2020

Clinical Features:

- Sensorineural hearing loss (30-60%)
- Chorioretinitis and psychomotor retardation



Algorithm: CMV infection screening in the first trimester. Management and follow-up

Diagnosis:

- Maternal infection is confirmed by seroconversion or IgM/IgG testing.
- Fetal infection is confirmed by PCR testing of amniotic fluid.

Management:

- **Valaciclovir (2 g every 6 hours):** Reduces vertical transmission.
- Prognosis of the infection, legal assumptions about termination of pregnancy and the limited evidence about treatment with Valaciclovir should be explained to the couple.
- **Preconceptional Advice:** in case of previous CMV Infection : Avoid pregnancy for next 6 months.
- Multidisciplinary counseling to discuss prognosis and management.

Herpes Simplex Virus (HSV)

Etiology and Transmission: HSV-1 and HSV-2 are DNA viruses. HSV-2 causes genital infections, which are transmitted in most cases from an asymptomatic carrier. Vertical transmission of the virus at the time of delivery can cause severe neonatal infection. Intrauterine transmission from maternal viremia or ascending infection with intact membranes is extremely rare and occurs in less than 5% of primary herpes infections.

If transmission occurs, there is a higher risk of abortion and premature delivery.

HSV has little teratogenic capacity, but there are isolated cases of defects following maternal infection in the first and second trimesters.

Clinical Manifestations:

- Disseminated disease or localized central nervous system involvement in neonates.

Management:

Pre-Pregnancy

- Avoid pregnancy until clinically cured.

Pregnancy – During Active Infection

- Hospitalization may be required for monitoring and supportive care.
- Watch for uterine activity to assess potential complications.
- Administer acyclovir 200 mg five times a day for 10 days to reduce the severity of the infection.

Labor

- **Cesarean Section (LSCS):** Recommended if active genital lesions are present over the perineum, vagina, or cervix to prevent neonatal transmission.
- **Vaginal Delivery:** Can proceed if no genital lesions are present, ensuring a lower risk of transmission to the newborn.
- In cases of premature rupture of membranes, the approach should be individualized based on the severity of the infection and maternal health status.

Neonate

- Evaluate the newborn for signs of neonatal herpes, particularly if active maternal lesions are present at the time of delivery.
- **Neonatal Prophylaxis:** Acyclovir prophylaxis is advised for neonates if the mother has active lesions or is known to have genital herpes.
- **Breastfeeding:** It is not contraindicated in mothers with HSV, as long as there are no active lesions on the breast or nipple.

Parvovirus B19

Etiology and Transmission: This DNA virus is transmitted via respiratory droplets, causing erythema infectiosum (fifth disease).

Weeks at Maternal infection	Fetal Hydrops	Intrauterine fetal demise without hydrops
<9	<1%	4%
9-12	7%	11%
13-16	12%	9%
17-20	12%	2%
>20	<5%	<1%

Table: Probability of foetal hydrops and foetal death (in the absence of hydrops) according to gestational age at maternal infection by PVB19

Diagnosis:

1. **Seroconversion:** Diagnosis is confirmed through seroconversion, which indicates a recent infection.
2. **Identification of IgM Antibodies:** The presence of IgM antibodies is used to identify recent or acute infection.

Treatment:

1. **Symptomatic Treatment:** The mother is treated symptomatically to manage any discomfort or complications arising from the infection.

Fetal Screening:

1. Fetal Anemia and Hydrops Evaluation: The fetus should be screened for anemia and hydrops through serial ultrasound (USG) every two weeks.
2. Intrauterine Transfusion: If fetal anemia is detected, intrauterine transfusion is performed to treat the anemia.

Monitoring Fetal Anemia:

1. **Doppler Assessment of Middle Cerebral Artery (MCA):** The Doppler of the systolic velocity of the middle cerebral artery (PSV-MCA) is the primary tool for diagnosing and monitoring fetal anemia.
2. **Criteria for Intervention:** An increase in PSV-MCA > 1.5 MoM (multiples of the median) or the presence of fetal hydrops indicates the need for cordocentesis in pregnancies over 18-20 weeks to assess the degree of fetal anemia.

Hydrops Evaluation:

1. **Hydrops Definition:** Hydrops is defined as the presence of excess fluid in two or more cavities and/or subcutaneous edema.
2. **Ultrasound Evaluation:** Hydrops should be evaluated by ultrasound to assess the severity and potential complications.

Conclusion

TORCH infections pose a significant challenge due to their potential for severe fetal morbidity and mortality. Early identification, tailored diagnostic approaches, and informed management strategies are essential. Continued research and public health efforts are crucial to reducing the burden of these infections.

Key Points

TORCH infections represent a group of congenital infections with significant potential to impact fetal and neonatal health adversely

The accurate diagnosis of TORCH infections requires a combination of clinical history, imaging, and laboratory testing.

Recent advancements in molecular diagnostics have enhanced detection and management of these infections effectively.

Preventive measures, such as rubella vaccination and proper hygiene to prevent toxoplasmosis, remain vital.

Early detection is important for treatment

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Hepatitis B in Pregnancy

Introduction

Hepatitis B virus (HBV) infection is a global problem with nearly 350 million chronic carriers, who are at risk of liver cirrhosis & hepatocellular carcinoma[1]. Over 50% of these carriers are believed to have acquired their infection vertically from their mothers, i.e. through mother - to - child transmission (MTCT). Vertically - acquired HBV infections frequently (>90%) become chronic[2]. The proportion of babies that became HBV chronic carriers is about 10% to 30% for mothers who are HBsAg positive but HBeAg negative. However, the incidence of perinatal infections is higher, i.e. 70% to 90%, when the mother is also HBeAg positive[3].

Review of Literature

Chronic HBV infection during pregnancy is an important opportunity to interrupt perinatal transmission of HBV. HBV infection does not appear to influence fertility or conception per se, beyond the effects of cirrhosis or liver failure[5]. If cirrhosis has set in, then pregnancy may be a rare event. Women with advanced chronic liver disease, regardless of cause, have decreased fertility as a result of frequent occurrence of anovulatory cycles and amenorrhoea6. The rate of spontaneous abortion is also significantly higher in women with cirrhosis, reaching 30% to 40% vs. 15% to 20% in the general population[5]. Cirrhotic women are at risk of developing significant perinatal complications and poor pregnancy outcomes, including intrauterine growth restriction, intrauterine infection, premature delivery, and intrauterine foetal demise. In recent years, the availability of reproductive technologies and advanced support measures has allowed a larger proportion of women with cirrhosis to carry pregnancies successfully to term[6]. HBV Infection during pregnancy does not appear to increase maternal or fetal mortality and morbidity. A large study that compared 824 HBeAg positive mothers to 6,281 HBsAg negative control mothers found no difference in rates of preterm delivery, birth weight, neonatal jaundice, congenital anomalies, or perinatal mortality[7]. However, a recent study showed that HBsAg carrier mothers had an increased risk of gestational diabetes mellitus, antepartum haemorrhage, and threatened preterm labour[8]. The American Association for the Study of Liver Disease (AASLD) recommends that all pregnant women be screened for HBsAg during the first trimester, even if previously vaccinated or tested[9]. Screening allows for identification of infants requiring immunoprophylaxis with HBV vaccine and hepatitis B immune globulin (HBIG), anti-viral treatment of pregnant carriers if indicated, and counselling of sexual and household contacts[2]. The HBsAg-positive pregnant woman should be counselled to inform their obstetricians so that immunoprophylaxis can be administered to the newborn immediately after delivery[9]. Women who test negative for HBsAg and are at risk of acquiring HBV infection should be immunized during pregnancy. The American Congress of Obstetricians and Gynecologists (ACOG) and AASLD guidelines suggest that HBsAg-positive mothers be referred for further medical evaluation so that those with liver disease can be identified and monitored frequently by a team of specialists. This should not be deferred to the postpartum period[9].Vertical transmission is the transmission of pathogen from mother to child during pregnancy or childbirth, or by breastfeeding and it is the main reason for the continued endemic infection of HBV in Asia. Approximately 90% of children who get HBV infection vertically from their mothers fail to clear the infection and develop chronic infection[6]. The risk of

vertical transmission of HBV predominantly depends on the maternal HBV viral load and HBeAg status. In the absence of prophylaxis, the risk of vertical transmission of HBV infection is as high as 70% to 90% for infants born to HBeAg-positive mothers, and 10% to 40% for infants born to HbeAg-negative mothers6.Vertical transmission of HBV is defined as positivity at 6-12 months of life of the hepatitis B surface antigen or of HBV DNA in an infant born to an infected mother. The presence of both HBsAg and HBV DNA at birth are transitory events and do not imply transmission of the infection. Similarly the presence of antibodies against hepatitis B e antigen or antibodies against Hepatitis b core antigen at birth or upto two years of age is simply due to crossing the placenta from mother to the fetus and therefore is unrelated to infection.

Modes of vertical transmission

Vertical transmission can occur in-utero, during delivery, or after delivery.

In-utero Transmission

HBV can cross the placental barrier and reach the fetus, however the impact of this mode is not clear. In a study from the United States, of 72 pregnancies, 13 (18%) cord blood samples were positive for HBsAg10; however, HBV DNA was detected in only three (23%) of these. In a Chinese study, only 3.7% of babies tested HBsAg-positive at birth from in-utero infection11. Hence it is suggested that in-utero transmission may not be the predominant mode of transmission of HBV. In-utero or trans placental HBV infection cannot be blocked by HBV vaccine or HBIG given at birth, and is an important reason for immunoprophylaxis failure. The mechanism of intrauterine transmission of Hepatitis B was studied by Zhan et al and they concluded that although the predominant rout of transmission was transplacental, other routes of infection may exist4. The main risk factors for intrauterine HBV infection are maternal serum HBeAg positivity, high maternal viral load, and a history of threatened preterm labor or threatened abortion6. It has been found that maternal pre-delivery HBV DNA level > 6 log copies/ ml are associated with reduced immunoprophylaxis effectiveness.

Transmission during delivery

This is widely believed to be the most frequent mode of MTCT. This is the reason why the neonatal administration of HBIG with vaccination is able to prevent newborn HBV infection in more than 85% of cases. In one study, duration of labor showed a positive correlation with HBV antigenemia of the cord blood especially when the labor exceeded nine hours[12]. An elective cesarean section performed before the onset of labor and rupture of membranes may effectively reduce the risk of vertical transmission as compared with vaginal delivery or cesarean section performed after the onset of labor or after rupture of membrane[13]. However, there is lack of agreement on this issue.

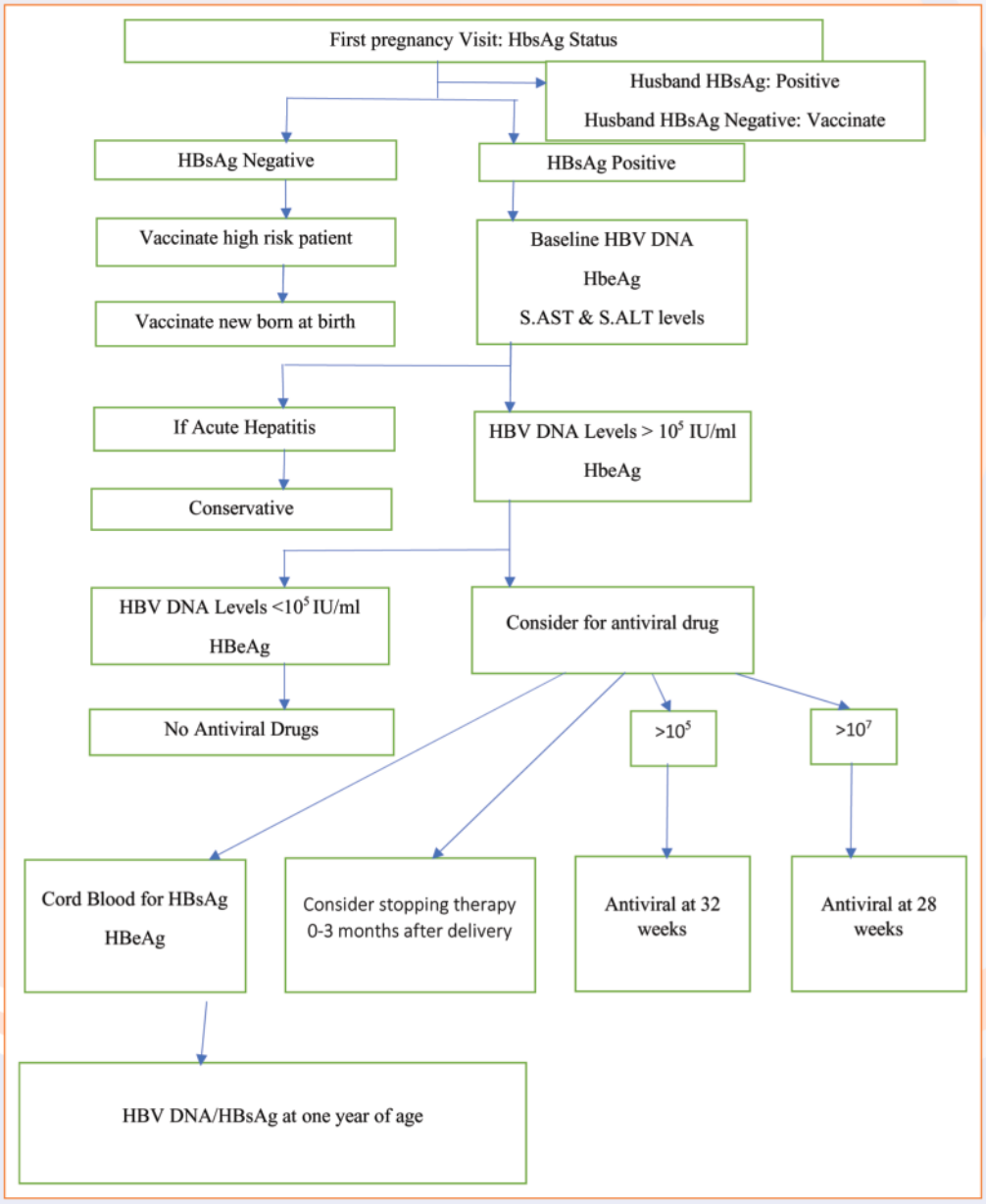
Postpartum Transmission

In the immediate postpartum period, transmission results from close contact between mother and baby. Transmission of HBV by breastfeeding, either through ingestion of the virus or by contact with skin lesions on the mother's breast, is another potential mechanism. Early studies reported HBsAg, HBeAg and HBV DNA detection in colostrum, with higher levels in mothers with high serum HBV DNA, suggesting that breast milk may be an important vehicle for transmission of HBV[14]. However, several studies have reported that breastfeeding carries no additional risk of transmission[15]. It has also been suggested that breast milk may have antiviral properties since it contains immunoglobulins and other proteins such as lactoferrin. In view of several benefits of breast feeding, WHO recommends breastfeeding for infants of HBsAg-positive mothers even in endemic areas where HBV vaccination may not be readily available[14]. High maternal HBV DNA titer is probably one of the most important risk factors for vertical transmission of HBV. HBV infection was found to occur in upto 10% of babies despite immunoprophylaxis and high maternal HBV DNA level was one of the most important risk factor for this. Traditionally HBeAg-positive mothers were considered to be at a higher risk of transmitting HBV infection to newborns than HBeAg-negative mothers, with the risks of chronic HBV infection by age of 6 months of 70% to 90% and 10% to 40%, respectively, in the absence of post-exposure immunoprophylaxis. The mechanisms for high rate of infection in infants born to HBeAg-positive mothers remain unclear. Maternal HBeAg positivity is strongly correlated with high levels of maternal viremia[16]. The American College of Gastroenterology (ACG) and AASLD guidelines both strongly recommend initiation of antiviral drugs in highly viremic patients at 28-32 weeks of gestation in order to reduce MTCT. Anti-viral therapy during pregnancy provides potent anti-viral suppression, is relatively safe and well tolerated, and reduces perinatal HBV transmission. Problems associated with treatment include the risk of viral drug resistance in the mother depending on the antiviral agent used and the risk of hepatitis flares upon discontinuation[14]. Current recommendations by the AASLD cite HBV DNA levels > 2 X 105 IU/ml as an

indication for initiation of therapy as risk of HBV transmission increases with level of viremia. Tenofovir, a nucleotide analogue with activity against HV polymerase, is currently a preferred oral agent for HBV therapy. It has been used by pregnant women for HIV infection with no increase in congenital malformations. Preliminary data show no evidence of renal impairment, abnormal bone metabolism or impaired growth in children exposed to tenofovir in utero[17].

There is conflicting evidence surrounding the effect of the mode of delievery on the risk of MTCT. A more recent meta-analysis revealed a 17.5% absolute risk reduction with cesarean section compared to immunoprophylaxis alone, suggesting a benefit of elective cesarean section to reduce MTCT. Lee et al investigated 1409 infants over a four-year period who had received appropriate immunoprophylaxis at birth and who had been born to HBsAg-positive mothers. They reported MTCT rates of 1.4% with elective cesarean section compared to 3.4% with vaginal delievery and 4.2% with urgent cesarean section. However the society for Maternal Fetal Medicine states that cesarean section should not be performed for sole indication of reducing vertical transmission.

Most guidelines now recommend that infants born to HBsAg-positive women should receive both HBIG and hepatitis B vaccine within 12 h of birth, preferably in the delivery room. This should be followed by at least two more doses of hepatitis B vaccine within the first 6 months of life. Passive immunoprophylaxis with HBIG at birth followed by at least 3 doses of the vaccine provides 90% to 95% protection from perinatal infection, and is superior in reducing MTCT than HBIG or vaccine alone (RR 0.08, 95% CI 0.03–0.17)18. After completion of the vaccine series, HBsAg and anti-HBs should be tested by 9 months of age. HBsAg-negative infants with anti-HBs levels >10 mIU/mL are protected and no further medical management is required. Those with anti-HBs levels <10 mIU/mL are not protected and should be revaccinated with another three-dose series followed by retesting 1 to 2 months after the final dose. With appropriate immunoprophylaxis, including HBIG and hepatitis B vaccine, breastfeeding of infants of chronic HBV carriers poses no additional risk of transmission of HBV18. In view of several benefits of breastfeeding, WHO recommends breastfeeding even for infants of HBsAg-positive mothers in endemic areas where HBV vaccination may not be readily available[14].



Key Points

- Every Pregnant patient should be mandatory screened for Hepatitis B.
- All hepatitis B pregnant should get HBV DNA quantitative and HbeAg test, in addition to liver function test at 28 weeks of pregnancy and if HBV viral load or HbeAg is found to be significantly high, then antiviral treatment tenofovir 300 mg should be started.
- Tenofovir is safe for both mother and newborn.
- Caesarean section has to be done for obstetric indications only and not for mere just prescence of HBV infection.
- Every new born of hepatitis B mother should be mandatory given 0.5 ml hepatitis B immunoglobulin, along with zero dose Hepatitis B vaccination within twelve hours of birth and later on full course of HBV should be completed.
- Breast feeding is allowed for the new born.
- The newborn should be tested for HBV at 18 months of age for determining vertical transmission.
- Husband of HBV pregnant patient should be screened for HBV and if found negative should be vaccinated against the same.

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Peurperal Pyrexia

General Consideration

Puerperal pyrexia is defined as a maternal fever of 38°C (100.4°F) or higher, occurring on any two of the first ten postpartum days, excluding the first 24 hours[1]. It is a common postpartum complication, significantly contributing to maternal morbidity and mortality, particularly in low-resource settings. The condition arises from various etiologies, with infections being the most prevalent. Timely diagnosis, effective management, and preventive measures are vital in reducing its impact.

Etiology

The causes of puerperal pyrexia can be classified into infectious and non-infectious categories.

1. **Infectious Causes:**

   Puerperal Endometritis: This is the leading cause of puerperal pyrexia, often resulting from ascending infections of the endometrium following vaginal or cesarean delivery. Risk factors include prolonged labor, premature rupture of membranes, and retained placental tissue[2]. Common pathogens include Escherichia coli, Group B Streptococcus, and anaerobic bacteria.

   Urinary Tract Infections (UTIs): Postpartum UTIs are common due to catheterization during labor, trauma to the urinary tract, and residual urine post-delivery.

   Surgical Site Infections (SSIs): Cesarean deliveries, which account for a significant proportion of births globally, pose a risk for wound infections, typically presenting with localized erythema, purulent discharge, and fever[3].

   Mastitis: Infection of the breast tissue, often caused by Staphylococcus aureus, is common in breastfeeding women. It manifests as localized pain, redness, and systemic symptoms such as fever and chills.

   Septic Pelvic Thrombophlebitis: This rare condition involves infected blood clots in pelvic veins, leading to fever unresponsive to antibiotics[4].

2. **Non-Infectious Causes:**

   Drug Reactions: Certain medications, such as prostaglandins used to manage postpartum hemorrhage, can cause transient fever.

   Deep Vein Thrombosis (DVT): Immobility and hypercoagulable states during the postpartum period increase the risk of DVT, presenting with fever and limb swelling.

   Autoimmune Conditions: Flare-ups of conditions like systemic lupus erythematosus may occur postpartum, leading to fever.

Pathophysiology

The pathophysiology of puerperal pyrexia is often rooted in the disruption of natural barriers during childbirth. For example, the endometrium becomes vulnerable to microbial invasion following placental detachment, especially if aseptic precautions are inadequate. Similarly, surgical interventions like cesarean sections compromise tissue integrity, creating an entry point for pathogens (5). Infections trigger a systemic inflammatory response, resulting in fever and other clinical manifestations.

Clinical Presentation

Patients with puerperal pyrexia typically present with fever, accompanied by other symptoms depending on the underlying cause. Endometritis manifests with lower abdominal pain, foul-smelling lochia, and uterine

tenderness. UTIs may present with dysuria and suprapubic pain, while mastitis is characterized by painful breast engorgement and systemic signs of infection[6]. A detailed clinical history and physical examination are crucial in identifying the source of infection.

Diagnostic Approach

The diagnosis of puerperal pyrexia involves a combination of clinical, laboratory, and imaging studies:

1. **Laboratory Tests:**

   Complete blood count (CBC) often reveals leukocytosis with a left shift.

   Blood cultures may identify bacteremia, especially in severe cases.

   Endometrial or lochial cultures help isolate causative organisms in suspected endometritis.

   Urine analysis and culture are essential for diagnosing UTIs[7].

2. **Imaging:**

   Ultrasound is useful for detecting retained products of conception or uterine abscesses.

   Contrast-enhanced CT or MRI may be required for diagnosing septic pelvic thrombophlebitis or deepseated infections.

Management

The treatment of puerperal pyrexia involves addressing the underlying cause and providing supportive care:

1. **Antibiotic Therapy:**

   Broad-spectrum antibiotics targeting common pathogens are initiated empirically. Common regimens include clindamycin and gentamicin for endometritis and cefazolin or amoxicillin-clavulanate for UTIs[8].

   Therapy is adjusted based on culture and sensitivity results.

2. **Surgical Interventions:**

   Retained placental fragments or uterine abscesses may require surgical evacuation or drainage.

   Incision and drainage are indicated for wound infections and breast abscesses.

3. **Supportive Care:**

   Antipyretics, hydration, and adequate pain management are essential.

   Encouraging ambulation reduces the risk of thromboembolic complications.

4. **Management of Complications:**

   Severe sepsis or septic shock necessitates intensive care management, including intravenous fluids, vasopressors, and monitoring[9].

Prevention

Preventive strategies are vital to reducing the burden of puerperal pyrexia, particularly in low-resource settings:

1. **Aseptic Techniques:**

   Maintaining strict asepsis during delivery and postpartum care reduces infection risks.

   Hand hygiene and sterilization of instruments are critical components.

2. **Antenatal Care:**

   Screening and treating maternal infections, such as asymptomatic bacteriuria, during pregnancy can prevent postpartum complications[10].

   Administering prophylactic antibiotics before cesarean delivery significantly lowers the incidence of SSIs.

3. **Breastfeeding Support:**

   Proper breastfeeding techniques prevent nipple trauma, reducing the risk of mastitis.

   Early intervention for engorgement or blocked ducts can prevent progression to abscess formation.

4. **Patient Education:**

   Educating mothers about signs of infection and the importance of follow-up care ensures timely medical attention.

5. **Systemic Interventions:**

   Strengthening health systems to improve access to skilled birth attendants and emergency obstetric care is crucial.

Global Perspective

The burden of puerperal pyrexia varies globally, with higher rates in low-resource settings due to limited access to healthcare, poor hygiene, and delayed interventions. For example, in sub-Saharan Africa, puerperal infections account for approximately 10% of maternal deaths[11]. In contrast, high-resource settings have significantly reduced the incidence through advancements in obstetric care and infection control practices.

Efforts by international organizations, such as the World Health Organization (WHO), aim to address these disparities by promoting clean delivery practices and enhancing maternal healthcare infrastructure[12].

Key Points

- **Prompt intervention:** Puerperal pyrexia requires immediate attention.
- **Antibiotics:** Antibiotics are the primary treatment for puerperal pyrexia. The type of antibiotic depends on the location of the infection and the bacteria causing it.
- **Pain relievers:** Acetaminophen and ibuprofen can help treat fevers.
- **Intravenous fluids:** Hospitalized patients are often given intravenous fluids.
- **Rest:** Patients should get plenty of rest.
- **Laboratory tests:** If the fever persists while taking antibiotics, or if there is suspected disease spread, laboratory tests and imaging studies may be performed.
- **Multidisciplinary team:** A multidisciplinary team should be involved in the management of puerperal pyrexia.
- **Transfer to specialist unit**: If severe sepsis is suspected or diagnosed, the patient should be transferred to a specialist unit immediately.
- **Prophylactic antibiotics:** Prophylactic antibiotics may be used for invasive procedures, manual removal of placentae, internal version, and third degree perineal tears.
- Preventive measures, such as aseptic delivery practices and patient education, play a critical role in reducing its incidence

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Septic Shock in Obstetrics

General Consideration

Sepsis in obstetrics is an emergency which should be recognized without any delay and managed promptly. It accounts for 11% of maternal deaths worldwide and is the 3rd most common direct cause of maternal death.[1] The prevalence was found to be 16.5/10,000 live births in a study done in VVMC & SJH in 2017.[2]

Sepsis is defined as "life threatening organ dysfunction caused by dysregulated host response to infection".[3] WHO (2018) has defined maternal sepsis as 'a life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post abortion or postpartum period'.[4] Organ dysfunction is defined as an increase in the SOFA Score of 2 or more (Fig 1). The SOFA Score assesses six organ systems and has been used in the critical care units for prognostication of the patients on admission and thereafter every day. However the sepsis-3 guideline has now incorporated the SOFA Score as an integral part of the sepsis definition.

Septic shock is defined as a 'subset of sepsis with profound circulatory, cellular and metabolic abnormalities associated with a greater risk of mortality than sepsis alone'. It is diagnosed when sepsis is associated with a vasopressor requirement to maintain a MAP of >65 mmHg and a serum lactate level >2 mmol/L. Septic shock is a continuum of sepsis and hence early recognition and management of sepsis is crucial, as sepsis is associated with a mortality of around 10% whereas septic shock has a mortality of more than 40%.

It has been strongly established that early identification and appropriate management in the initial hours improves the outcome in cases of sepsis.[5]

The pathogenesis is not completely understood till date. The host response to infection plays an important role, initial pro inflammatory pathways are activated, anti-inflammatory pathways are also activated and can down regulate corrective responses later in the course of sepsis. The four main features are endothelial dysfunction, coagulopathy, cellular dysfunction and cardiovascular dysfunction.

Risk factors for sepsis in obstetrics

There are various risk factors for sepsis in obstetrics. Some of them are unique to pregnancy as pregnancy is a state of immune compromise. These consist of prolonged rupture of membranes, prolonged labour, vaginal trauma, caesarean birth, multiple vaginal examinations and any intrauterine procedure. The general risk factors include, low socio-economic status, poor nutrition, anemia, diabetes mellitus, obesity.

Predictor tools: There have been various predictive scores for early recognition of sepsis. The evolution of newer scores has improved the sensitivity and positive predictive value. In 2016, the Sepsis-3 task force recommended to use a much simpler score, the quick SOFA (qSOFA) score as it has to consider only three parameters. The Surviving Sepsis Campaign (SSC) in their recent guideline in 2021 has recommended against use of qSOFA as a single screening tool in sepsis or septic shock.[5] Studies have shown that qSOFA is more specific but less sensitive than SIRS criteria for early identification of infection induced organ dysfunction.[6-9] Another study showed 24% of infected patients had a qSOFA score 2 or 3, but these patients accounted for 70% of poor outcomes.[10] However, a raised qSOFA should always alert the clinicians about the possibility of sepsis but does not necessarily imply it's presence or absence.

The Modified Early Obstetric Warning Score (MEOWS) is implemented for early recognition of deterioration of the critically ill obstetric patients.[11] (Fig 2) It uses physiological variables and the score is calculated. If the score is beyond the threshold, medical review and intervention is required. If the MEOWS score is 4, any single parameter score is 3 or the patient deteriorates further or fails to respond to treatment then one should "THINK SEPSIS".

Score points	1	2	3	4
Respiration				
PaO_2/FiO_2	<400	<300	<200 with respiratory support	<100 with respiratory support
Cardiovascular				
Hypotension*	MAP <70 mmHg	Dopamine ≤5 or dobutamine in any dose	Dopamine >5 or epinephrine ≤0.1 or norepinephrine ≤0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
Liver				
Bilirubin mg/dl	1.2–1.9	2.0–5.9	6.0–11.9	>12.0
Renal				
Creatinine mg/dl or urine output	1.2–1.9	2.0–3.4	3.5–4.9 or <500ml/2 4h	5.0 or <200ml/24 h
Coagulation				
Platelets $\times 10^3/mm^3$	< 150	< 100	< 50	< 25
Central nervous system				
Glasgow Coma Scale	13–14	10–12	6–9	< 6

* Adrenergic agents administered for at least 1 h (doses are given in µg/kg/min)

FIG 1: SOFA(sequential organ failure assessment) Score

Carle et al designed another modified obstetric early warning score (Carle's OEWS)[12] In a study conducted in VMMC and SJH during 2017-2019 used this score in labour wards in predicting admission to obstetric critical care unit, maternal near miss and mortality. They found it as a useful screening tool and it can be routinely used to ensure timely intervention[13]

Management: The optimum management of sepsis lies in early recognition and prompt initiation of resuscitation. The window between the onset and identification of sepsis is often where significant delays in management occur.[14] The initial resuscitation and the investigations goes hand in hand.

The **"golden hour of sepsis"** stresses the

Score	3	2	1	0	1	2	3
Temperature		<35 °c	35-35.9 °c	36-37.4 °c	37.5-37.9 °c	38.0-38.9 °c	≥39 °c
Systolic BP	≤70	71-79	80-89	90-139	140-149	150-159	≥160
Diastolic BP			≤49	50-89	90-99	100-109	≥110
Pulse		<40	40-49	50-99	100-109	110-129	≥130
Respiratory Rate	≤10			11-20	21-24	25-29	≥30
Oxygen Saturations	≤94%			≥95%			
AVPU				Alert	Responds to Voice	Responds to Pain	Unconscious
Urine output mLs/hr	<10	<30		Not Measured			

Figure 2: Modified Early Obstetric Warning Score (MEOWS) in detecting the seriously ill and deteriorating woman

relationship between timely initiation of antibiotic treatment and outcome. Studies have found that each hour delay in treatment reduces sepsis survival by 7.6%.[15] Here lies the rationale of obtaining sample for culture to diagnose the infection and initiating appropriate antimicrobial later on.

Investigations

To asses for end organ hypoperfusion	To asses infective organism
• Full blood count • LFT • KFT • Serum lactate • ABG • Coagulation profile	Blood culture, wound swabs, vaginal swabs

Components of the one hour bundle

1. Measure lactate level, remeasure lactate if initial lactate elevated (> 2 mmol/L)- in 2-4 hours (GRADE 1B)
2. Obtain blood cultures prior to administering antibiotics. Do not delay antibiotics in the situation where culture is not feasible
3. In pregnant or postpartum patients with septic shock or a high likelihood of sepsis, administer empiric broad-spectrum antimicrobial therapy, ideally within 1 hour of recognition - (strong recommendation)
4. Begin rapid administration of 30mL/kg crystalloid (balanced solution) in first 3 hours- for hypotension or lactate ≥4 mmol/L (strong recommendation)
5. Apply vasopressors if hypotensive during or after fluid resuscitation to maintain a mean arterial pressure ≥65 mm Hg- in first hour itself (strong recommendation)

The surviving sepsis campaign (2016) has also given key recommendations about management of sepsis and septic shock. Data from a prospective cohort study from the SSC showed that compliance with SSC bundles led to a 25% relative risk reduction in mortality.[16]

In the recent guideline, SSC (2021) has upgraded and changed few of their recommendations regarding management of sepsis and septic shock. Salient points regarding the management is as follows-

Initial resuscitation:

- Management of sepsis and septic shock should be initiated immediately after recognition
- Those who require ICU admission should be admitted within 6 hours
- For sepsis induced hypoperfusion, intravenous crystalloids at least 30ml/kg should be given within first three hours of resuscitation
- Rather than physical examination or static parameters, dynamic tests like passive leg raising test should guide fluid resuscitation
- Serum lactate level should guide the resuscitation, aim should be lowering the elevated lactate level
- Capillary refilling time is now considered as a guiding parameter for resuscitation in adjunct to other measures of perfusion
- The target mean arterial pressure (MAP) in septic shock (on vasopressor) is 65 mmHg

Antimicrobial therapy:

- Initiation of IV antimicrobials immediately or within one hour of recognition of sepsis with or without shock is strongly recommended (Fig. 2)
- Continuous re-evaluation and search for alternate diagnosis should be done in cases with suspected sepsis without confirmed infection. Empirical antibiotics should be discontinued if alternate diagnosis is established or strongly suspected
- Serum procalcitonin level is not to be used along with clinical evaluation regarding decision when to start antimicrobials
- Empiric broad-spectrum therapy with one or more antimicrobials to cover all likely pathogens (bacterial, fungal, viral) is recommended in sepsis and septic shock
- Antimicrobials with methicillin resistant staphylococcus aureus (MRSA) coverage is recommended only in those patients who are at high risk
- Source control should be done as soon as possible
- Daily assessment and de-escalation of antimicrobials is strongly recommended
- Using a shorter course of antimicrobials in cases with adequate source control is recommended

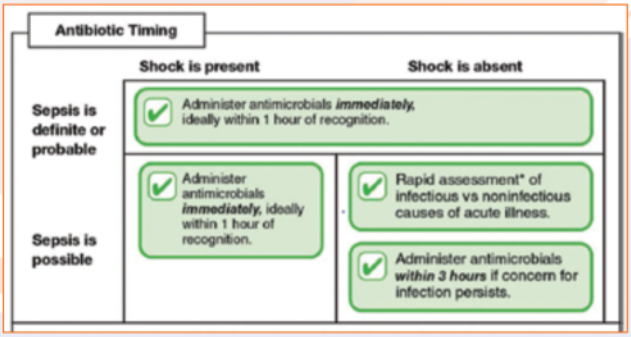

Figure 3: Antimicrobial protocol in sepsis and septic shock (SSC, 2021)[5]

Haemodynamic management

- Crystalloids are the fluid of choice in sepsis and septic shock, starch or gelatine should not be used
- Vasopressor should be started by peripheral access to restore MAP rather than delaying for establishing a central access
- Nor-epinephrine is the first line vasopressor (starting dose of norepinephrine is in the range of 0.25"0.5 ìg/kg/ min.
- When target MAP is not achieved with nor-epinephrine adding vasopressin is recommended rather than increasing the dose of it. The next agent which has to be added is epinephrine

Ventilation:

- In sepsis induced hypoxemic respiratory failure, high flow nasal oxygen is to be used rather than non-invasive ventilation
- In sepsis-induced severe ARDS, wherever feasible Veno-Venous (VV) ECMO is to be used when conventional mechanical ventilation fails

Additional therapy:

- If fluid resuscitation and vasopressor therapy are able to maintain the hemodynamic status of patients with septic shock, steroid are not to be used. It is started when ongoing requirement of norepinephrine or epinephrine e" 0.25 mcg/kg/min for at least 4 hours after initiation to maintain the target MAP. The dose is 200 mg/day given as 50 mg intravenously every 6 hours or as a continuous infusion
- Restrictive transfusion strategy is followed, the trigger is Hb < 7gm/dl along with the overall clinical condition of the patient
- IV immunoglobulins, IV vitamin C are not recommended
- Stress ulcer prophylaxis is to be considered in patients with high risk for GI bleed
- Venous thromboprophylaxis is to be considered and low molecular heparin is of choice rather than unfractionated heparin
- Insulin therapy to be started if the blood sugar level is ≥ 180mg/dL
- Sodium bicarbonate is to be considered if there is severe metabolic acidaemia (pH< 7.2) or acute kidney injury
- Early initiation (within 72 hours) of enteral feeding is to be considered

Termination of pregnancy

Sepsis is not an indication for pregnancy termination unless the source is the feto placental unit, like in chorioamnionitis. In other situations the decision needs to be taken on case to case basis by a senior obstetrician. Maternal stabilisation is critical before planning delivery.

Care for the sepsis survivors:

- Shared decision-making regarding discharge planning, educating patient and family members about maintaining asepsis, referral to peer support groups is encouraged

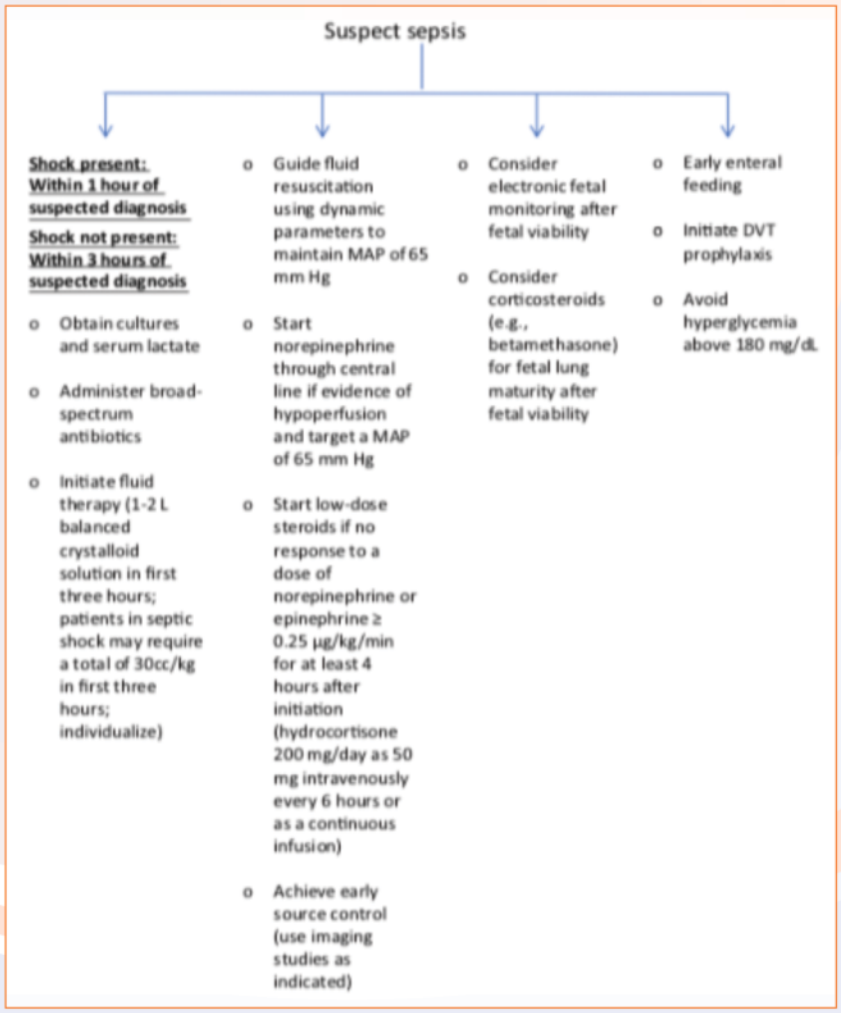

Fig 4: Summary of management of sepsis and septic shock

- Post discharge follow up should be scheduled
- Survivors who received mechanical ventilation for > 48 hours or an ICU stay of > 72 hours, a post-hospital rehabilitation program is suggested

Key Points

- Sepsis and septic shock are medical emergency. Prompt recognition and immediate management are keys to improve outcome.
- The screening tools are effective and should be used judiciously.
- Continuous re-evaluation and search for alternate diagnosis when suspected and discontinuation of empiric antibiotics is recommended.
- Adequate source control reduces morbidity and mortality.
- Post discharge care is also of utmost importance, educating the patient and family members how to prevent sepsis, timely follow up and referral to peer support group should be encouraged.

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Time to expand our testing of infections among pregnant women: Lessons from case of acute & chronic severe pneumonia complicating the partum period

Abstract

Aims & Objective: Pregnancy is highest risk population for influenza infection and vaccination is a good preventive strategy. A patient with cute pneumonitis similar presentation to influenza that adversely affects foetus is seen respiratory syncytium virus (RSV) infection and almost never diagnosed due to non-testing. Similarly in a chronic pneumonia either bacterial or tuberculosis is considered and fungal pneumonia is almost never diagnosed during pregnancy. We explore diagnose of these infection with our 2 cases.

Methodology: Cases of fungal pneumonia and RSV during pregnancy was selected for reporting.

Results & Discussion: The first case of pneumonitis had progressive respiratory failure in mother and later the delivered foetus had developed similar ARDS and eventually died of respiratory failure. The mother eventually recovered with the help of Ribavirin and was discharged in stable condition. Second case showed a peri-partum non resolving pneumonia not responding to antibiotics & ATT. Broncho-alveolar lavage (BAL) eventually grew candida species which is usually a coloniser and not considered for therapy. As it was refractory in case we decided to treat it with antifungal agents and the patient recovered completely along with the baby. A 6 month follow up showed no recurrence of the disease. RSV infection is important as diagnosis because it shows response to Ribavirin which is not used in treating other viral pneumonia. It adversely affects the foetus and thus results in foetal demise. There is vaccination of mothers available for prevention of disease in new-born. Similarly, fungal pneumonia adversely affects the pregnancy as well as the mother. It is non-responder to antibiotics and anti-tubercular therapy.

Conclusion: RSV is an underestimated cause of ARDS in pregnant female and actual burden of disease is needed in Indian population in order to plan vaccination stategy. It is extremely important to thoroughly test all non-responding pneumonias in pregnancy for fungal infection as it can be successfully treated with antifungal.

Introduction

Infections are common morbidity affecting pregnancy. Pregnancy is highest risk population for influenza infection and vaccination is a good preventive strategy. A patient with cute pneumonitis similar presentation to influenza that adversely affects foetus is seen respiratory syncytium virus (RSV) infection and almost never diagnosed due to non-testing. Similarly in a chronic pneumonia either bacterial or tuberculosis is considered and fungal pneumonia is almost never diagnosed during pregnancy. We explore diagnose of these infection with our 2 cases.

Case 1

34 year old pregnant lady at term presented to us with fever. Dry cough and progressive shortness of breath developed over 2 days. Her examination showed normal vitals, bilateral diffuse rhonchi & crepitation. X ray chest showed pneumonitis, Total leukocyte count (TLC) was 4500 cells/mm3; 50% lymphocytic and liver & renal function test (LFT, KFT) was normal. Her C - reactive protein (CRP) was 12 IU/L and sputum showed no significant findings. Her oxygen requirement

increased over next day to 8 litres/min and intermittent use of non-invasive ventilation (NIV) was started. Her throat swab was negative for H1N1, H3N2 & bacterial organism. She delivered the baby on the 4th day of illness and clinical condition of baby was stable. On day 2 the baby developed progressive hypoxia and was shifted to NIV. Over next 2 days baby got intubated and developed progressive respiratory failure. ON 4th day baby died of respiratory failure. As the mother as well as the baby both had develops similar illness in form of ARDS we considered re-testing for throat swab. A repeat test came negative for COVID-19 but came positive for Respiratory Syncytium Virus (RSV) by RT-PCR in both mother & child. Hence this is a case of RSV pneumonitis. Meanwhile the patient showed sustained improvement in all clinical parameters the use of antiviral Ribavirin was not considered. The mother was discharged in stable condition on 10th day of illness.

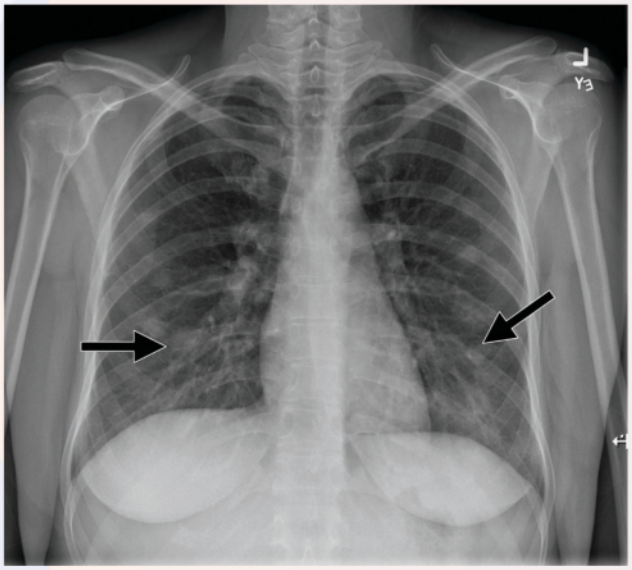

Image 1: diffuse pneumonitis in young lady

Case 2

A 32 year 7 month pregnant lady presented to OPD with complaints of intermittent fever with progressive cough for 2 weeks. She had received 7 days course of 2 antibiotics but found no relief. An ultrasound thorax was done and there was left lobe pneumonia. Her TLC was 16700 cells/mm3 87% neutrophil with CRP of 78. Her sputum was induced and sends for investigation. She was started on piperacillin-tazobactem and supportive medications. There was no clinical response in 3 days. Later her sputum grew Pseudomonas aerigenosa sensitive to carbapenem, tobramycin, colistin. She was started on meropenem infusion and there was mild clinical improvement in cough but fever was persistent along with new onset dyspnoea. Her oxygen saturation (SpO2) had dropped to 92% on room air. In this view she underwent bronchoscopy and brocheo-alveolar lavage (BAL) was done and sample was send for investigation. It showed growth of Pseudomonas with similar susceptibility and candida species which was considered as coloniser. Her galactomanon & Beta D Glucan were negative. She was shifted to meropenem with ploymyxin B for next 7 days. She had partial improvement in cough and fever but intermittent spikes were evident. Now duration of pneumonia was more than 40 days and she was started on anti-tubercular drugs (ATD). She underwent LSCS and delivered a healthy baby. After 10 days her BAL culture grew candida species but it was considered as coloniser and she was continued on same therapy. After 2 weeks there was no clinical improvement and her SpO2 remained below 94%. Her ATT was stopped and she was started on fluconazole. At this juncture of time she visited my clinic and ordered for antifungal susceptibility of fungal growth and it showed fluconazole & itraconazole resistance. She was started on caspofungin 70 mg stat and 50 mg once daily. On 5 th day of her therapy her dyspnea had subsided and SpO2 came above 95% on room air. By 10th day of therapy there was significant decrease in cough and improvement in general condition. She was discharged on voriconazole and breast feeding was discontinued. After 2 weeks of oral therapy her CT scan of thorax showed almost resolution of pneumonia with minimal remnant scarring. Hence her diagnosis was candida pneumonia and not pseudomonal pneumonia.

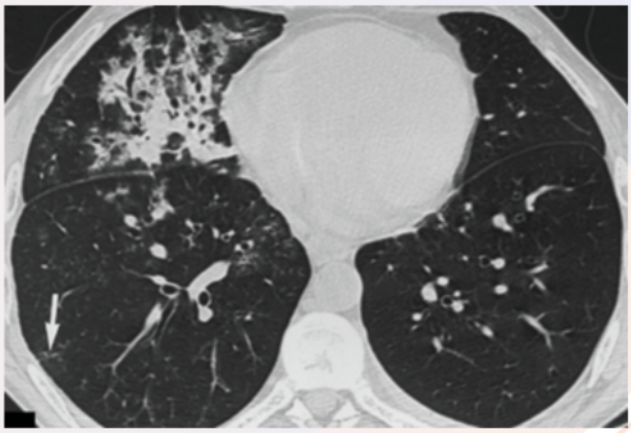

Image 2: Right sided consolidation with tree in bud appearance

Discussion

Viral pneumonitis among pregnant female and special reference to RSV infection

Important viral infections associated lung disease in pregnancy includes- Influenza (H1N1, H3N2), COVID-19, & RSV. RSV infection is associated with a similar disease burden to influenza in high-risk and elderly

adults, and may be associated with adverse outcomes in pregnancy. The clinical presentation of RSV illness during pregnancy and the postpartum period and the effect of RSV infection on maternal, foetal, and infant outcomes are not well-described in large prospective studies. Although in a large, geographically diverse, population-based surveillance system that contained data from >11 000 adults with laboratory-confirmed RSV-associated hospitalizations, a small proportion (n = 279, 2.5%) of all adult hospitalizations occurred in pregnant women, and pregnant women hospitalized with RSV rarely experienced severe outcomes. These findings are consistent with smaller studies with limited numbers of RSV infections and hospitalizations among pregnant women. Other studies have not identified an association of preterm birth among women who were positive for RSV during their pregnancy, and Trinh et al found that prenatal infection with RSV was associated with a lower birth weight and postnatal growth.

RSV was also detected in a substantial proportion of infants of mothers with postpartum RSV illness, with evidence of the same subtype in mother-infant pairs. This agrees with studies showing mothers as a potential source of RSV introduction to infants in households in rural Kenya. RSV had an attack rate of 10–13% among ambulatory pregnant women receiving routine prenatal care during the respiratory virus season. Maternal RSV antibodies are efficiently transferred transplacentally to the fetus during gestation, and have been associated with decreased risk of RSV infection in young infants. Assessment of risks of RSV infection in women becomes more important as maternal immunization with RSV subunit vaccines is considered as an approach to prevent symptomatic lower respiratory tract disease in both mothers and infants. The RSV maternal vaccine are approved for administration during pregnancy in a specific gestational period to optimize maternal antibody transfer to the infant

Unfortunately Indian data in this aspect is very thin and need more reporting & structured studies.

Systemic & Deep fungal pneumonia among pregnant patients

Common deep fungal infections include- Aspergilus, Candida, Mucor, Histoplasma, Cryptococcus, etc. Infections of these organisms can be life or organ threatening. It is sometimes associated with preterm labour, abortion or foetal infection. Candida is the commonest of them in pregnancy and associated with neonatal candidiasis which is associated with mortality. Although vulvovaginal candidiasis predominates the picture but disseminated disease is also observed. It is only Coccidioidomycosis where pregnancy is a risk factor. A thorough literature search showed absence of systemic or deep fungal infection studies and only presence of case reports. This highlights the negligence of diagnosis towards fungal infections. A review showed -Fungal infections commonly present with myriad symptoms that mimic other clinical entities, notable amongst which is tuberculosis. Besides histoplasmosis and chronic pulmonary aspergillosis, which can mimic TB, this review has identified several other fungal infections which also do. A total of 80 individual cases misdiagnosed as TB are highlighted: aspergillosis (n=18, 22.5%), histoplasmosis (n=16, 20%), blastomycosis (n=14, 17.5%), cryptococcosis (n=11, 13.8%), talaromycosis (n=7, 8.8%), coccidioidomycosis (n=5, 6.3%), mucormycosis (n=4, 5%), sporotrichosis (n=3, 3.8%), phaeohyphomycosis (n=1, 1.3%) and chromoblastomycosis (n=1, 1.3%). Case series from India and Pakistan reported over 100 cases of chronic and allergic bronchopulmonary aspergillosis had received anti-TB therapy before the correct diagnosis was made. Forty-five cases (56.3%) had favorable outcomes, and 25 (33.8%) died, outcome was unclear in the remainder. Seventeen (21.3%) cases were infected with human immunodeficiency virus (HIV). Diagnostic modalities were histopathology (n=46, 57.5%), culture (n=42, 52.5%), serology (n=18, 22.5%), cytology (n=2, 2.5%), gene sequencing (n=5, 6.3%) and microscopy (n=10, 12.5%) including Gram stain, India ink preparation, bone marrow smear and KOH mount.

Conclusion

RSV is an underestimated cause of ARDS in pregnant female and actual burden of disease is needed in Indian population in order to plan vaccination stategy. It is extremely important to thoroughly test all non-responding pneumonias in pregnancy for fungal infection as it can be successfully treated with antifungal.

Key points (Carry home messages)

1. Respiratory syncytium virus (RSV) is primarily a disease of childhood although there is growing burden of disease among adults including pregnant women.
2. It should be looked into as cause of severe acute respiratory infections (SARI) who are negative for influenza test.
3. RSV database for India should be made by testing at different centres across India.
4. Maternal vaccination against RSV prevents the disease in foetus and should be advocated when prevalence increases.

5. While investigating for a pneumonia in pregnant ladies which is not responding to common antibiotics fungal infection should be investigated.
6. A bronchoscopy should be prioritising in such cases and microscopy, culture or PCR based test should be done.
7. The closest differential of Pulmonary Tuberculosis is fungal infection esp. Aspergilosis. Candida in lung should be treated when no other diagnosis is possible.
8. Treatment outcome of both RSV & fungal infection is excellent & thus should be tested.

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> Communities and Countries and Ultimately the World are only as Strong as the Health of their Women.
>
> Michelle Obama

Dr. Taru Chhaya
NIGF
Quiz Coordinator

Infections in Pregnancy

1. Which of the following is the most common bacterial infection in pregnancy in India?

A. Group B Streptococcus (GBS)
B. Urinary Tract Infections (UTI)
C. Chlamydia trachomatis
D. Gonorrhea

2. The incidence of hepatitis B virus (HBV) infection among pregnant women in India is estimated to be:

A. <1% B. 2–7%
C. 10–15% D. >20%

3. Regarding rubella infection in pregnancy in India, which of the following is correct?

A. Congenital rubella syndrome (CRS) is rare in India.
B. The majority of women of childbearing age are seronegative for rubella.
C. Vaccination coverage for rubella is high across all Indian states.
D. CRS occurs in 10–20% of susceptible pregnancies if infected during the first trimester.

4. What is the approximate prevalence of HIV infection in pregnant women in India?

A. 0.05% B. 0.2%
C. 1% D. 5%

5. Which of the following infections is a significant cause of maternal mortality in India?

A. Hepatitis E B. Hepatitis B
C. Dengue D. HIV

6. Which of the following parasitic infections has a significant impact on pregnancy outcomes in India?

A. Giardia lamblia
B. Toxoplasma gondii
C. Taenia solium
D. Plasmodium falciparum

7. Syphilis in Pregnancy - Which statement regarding syphilis in pregnancy is correct?

A. Treponema pallidum cannot cross the placenta.
B. The risk of congenital syphilis is highest in tertiary syphilis.
C. Penicillin is the treatment of choice at all stages of pregnancy.
D. Jarisch-Herxheimer reaction is rarely seen in pregnant women.

8. Which of the following is false regarding congenital CMV infection?

A. Vertical transmission is higher in primary infection compared to reactivation.
B. Universal screening for CMV in pregnancy is recommended.
C. CMV is the most common congenital infection worldwide.
D. Antiviral therapy may reduce viral load in primary maternal infection.

9. Listeriosis in Pregnancy - Which of the following is true regarding listeriosis in pregnancy?

A. It primarily affects the fetus in the first trimester.
B. Ampicillin is the treatment of choice.

C. Vertical transmission is extremely rare.

D. Neonatal listeriosis is associated with low mortality.

10. Group B Streptococcus (GBS) - Routine intrapartum antibiotic prophylaxis for GBS is indicated in:

A. All pregnant women.

B. Women with a previous baby affected by GBS disease.

C. Women who test positive for GBS at any point in pregnancy.

D. Preterm labor regardless of GBS status.

11. The most severe fetal complication of maternal Zika virus infection is:

A. Microcephaly.

B. Cardiac anomalies.

C. Limb hypoplasia.

D. Cleft palate.

12. A pregnant woman at 18 weeks gestation presents with a primary VZV infection. The best management includes:

A. Immediate delivery.

B. Administration of varicella-zoster immune globulin (VZIG).

C. Antiviral therapy with acyclovir.

D. Both B and C.

13. Which antifungal is safest for treating vaginal Candidiasis in pregnancy?

A. Fluconazole

B. Clotrimazole

C. Ketoconazole

D. Itraconazole

14. Which of the following is false regarding dengue fever in pregnancy?

A. Dengue infection increases the risk of preterm labor.

B. Maternal platelet transfusion is indicated for platelet counts <20,000/mm^3.

C. Vertical transmission of dengue is uncommon.

D. Nonsteroidal anti-inflammatory drugs (NSAIDs) are safe for pain management.

15. Which of the following is correct regarding COVID-19 management in pregnancy?

A. Remdesivir is contraindicated.

B. Low-dose aspirin is indicated for preeclampsia prevention.

C. Corticosteroids should not be used for maternal respiratory distress.

D. Cesarean section is mandatory for COVID-19-positive women.

Answers

1. B	2. B	3. D	4. B	5. A
6. D	7. C	8. B	9. B	10. B
11. A	12. D	13. B	14. D	15. B

Time to expand our testing of infections among pregnant women: Lessons from case of acute & chronic severe pneumonia complicating the partum period.

Dr. Saurabh Pandey
Gorakhpur

Aims & Objective :

Pregnancy is highest risk population for influenza infection and vaccination is a good preventive strategy. A patient with cute pneumonitis similar presentation to influenza that adversely affects foetus is seen respiratory syncytium virus (RSV) infection and almost never diagnosed due to non-testing. Similarly in a chronic pneumonia either bacterial or tuberculosis is considered and fungal pneumonia is almost never diagnosed during pregnancy. We explore diagnose of these infection with our 2 cases.

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Cases of fungal pneumonia and RSV during pregnancy was selected for reporting.

Results & Discussion :

The first case of pneumonitis had progressive respiratory failure in mother and later the delivered foetus had developed similar ARDS and eventually died of respiratory failure. The mother eventually recovered with the help of Ribavirin and was discharged in stable condition. Second case showed a peri-partum non resolving pneumonia not responding to antibiotics & ATT. Broncho-alveolar lavage (BAL) eventually grew candida species which is usually a coloniser and not considered for therapy. As it was refractory in case we decided to treat it with antifungal agents and the patient recovered completely along with the baby. A 6 month follow up showed no recurrence of the disease. RSV infection is important as diagnosis because it shows response to Ribavirin which is not used in treating other viral pneumonia. It adversely affects the foetus and thus results in foetal demise. There is vaccination of mothers available for prevention of disease in new-born. Similarly, fungal pneumonia adversely affects the pregnancy as well as the mother. It is non-responder to antibiotics and anti-tubercular therapy.

Conclusion :

RSV is an underestimated cause of ARDS in pregnant female and actual burden of disease is needed in Indian population in order to plan vaccination stategy. It is extremely important to thoroughly test all non-responding pneumonias in pregnancy for fungal infection as it can be successfully treated with antifungal.

laVisual, clinical and microbiological diagnosis of abnormal vaginal discharge : A comparative study

Dr. Aradhna Gupta, Dr. Jaya Patel

Aims & Objective :

To compare the visual and clinician presumptive diagnosis with Microbiological diagnosis and also to compare the various etiological agents on basis of Microbiological diagnosis.

Methodology :

This was a descriptive type of observational study, conducted in sexually active 100 non pregnant women of reproductive age group (18-45 years) attending the OPD/IPD of Aaradhna Hospital Bhopal(MP) over a period of 9 months from January 2024 to September 2024. Pregnant, post menopausal & menstruating women and those who had received antimicrobial /antifungals in the previous 1 month and patient who had delivered or aborted six weeks prior were excluded from the study. After taking consent general physical

examination along with pelvic examination was performed. Two high vaginal swabs and blood sample were collected for various tests. Hanging drop preparation was made immediately.This was followed by gram staining and culture. Chlamydia trachomatis IgM antibody was detected by ELISA method.

Result & Discussion: Among 100 women, specific diagnosis was obtained in 91% of cases. Mean age was 30.60 years. Bacterial vaginosis was found in 58%, candidiasis was found in 18% cases, 12% had Chlamydia trachomatis infection while Trichomonas vaginalis infection was detected in 3% cases. Homogenous discharge was most prevalent (56%), followed by mucopurulant discharge in 20% of women.

Conclusion: Vaginal infections are the common gynecological infection. Judicious management may be helpful in prevention of HIV, HPV, CIN and post infection sequelae.

Assessment of Factors for Surgical Site Infection following Cesarean Section

Dr. Vani Malhotra, Dr. Aditi Sangwan
Department of Obstetrics & Gynaecology.
Pt. BDS PGIMS, Rohtak

Background :

Surgical Site Infection (SSI) is defined as infection developing at the site of surgical site within 30 days of surgery. Assessment of SSI is an important factor to determine the functioning of the health care system. Objectives of this study was to estimate the incidence of surgical site infection among caesarean section cases and to determine the risk factors associated with surgical site infection and comparison with patients having healthy wounds.

Methods :

A prospective observational study was carried out on one thousand pregnant women who underwent caesarean section in the department of Obsterics and Gynaecology, Pt BDS PGIMS ROHTAK. A total of 1000 women were enrolled in the study and were further divided into two groups: Group 1 (cases): Those who had SSI within 30 days of caesarean section and Group 2 (controls): Those who didn't have SSI. All the preoperative risk factors related to SSI were noted. Preoperative skin preparation, vaginal cleaning with betadine solution and antimicrobial prophylaxis was provided. Details of operative procedure were also noted. Patients were assessed for SSI in their postoperative period and classified as superficial or deep on the basis of CDC guidelines. Pus was sent for culture and sensivity followed by appropriate treatment.

Results :

Mean age of group I was 25.35±4.40 and 21.12±3.60 years in group II ($p > 0.05$). Mean gestational age of group I cases was 38.07±1.88 weeks and in group II, it was 38.17±2.06 weeks ($p > 0.05$). A total of 37(82.5%) women in group I and 931(96.98%) women in group II underwent emergency caesarean section ($p < 0.05$). In group I, mean duration of surgery was 1.0±0.13 hours and 1.02±0.21 hours in group II ($p < 0.05$). Maximum number of patients i.e. 22(55%) had wound discharge between 4-7 days followed by 11(27.5%) between 8-10 days. Mean wound discharge was 7.32±3.45 days in group I. Majority of women, i.e. 27(67%) found to be sterile in the present study followed by 7(17.5%) women were found to have staphylococcus aureus. Mean duration of resuturing was 17.42±6.98 days. Mean baby weight in group I was 2.72±0.53 kg and in group II it was 2.95±0.53 kg ($p < 0.001$).

Conclusions :

Risk of developing SSI after caesarean section is multi-factorial and found to be influenced by emergency surgery, PROM, pre-operative anaemia, multiple vaginal examinations, interrupted skin suturing, raised BMI, nulliparity, emergency caesarean, duration of surgery.

Keywords :

Assessment, Caesarean section, Infection, Risk factors, Surgical site, Surgical site infection

Surgical Site Infections in Obstetrics and Gynaecologic Surgeries: A Prospective Study in a Tertiary Care Centre

Prof. Minakshi Rohilla, Dr. Tanvi Katoch, Prof. Vanita Jain

Department of Obstetrics & Gynecology.
PGIMER Chandigarh.

Aims & Objectives :

Infection of the surgical site which can be incisional, or organ space and develops within 30 days of surgery is labelled as Surgical site infection (SSI). Opening of the female genital tract during surgery exposes the operative field to the vaginal flora thus, the surgical site in obstetric and gynecologic surgeries has the inherent tendency of getting contaminated. An in-depth analysis of factors which can have an influence on SSI is necessary before effective steps could be taken to reduce SSI. This prospective study was conceptualized, aiming to determine the associated risk factors of SSI in surgeries conducted in the Department of Obstetrics & Gynaecology of Post Graduate Institute of Medical Education and Research Chandigarh over a period of 3 years.

Materials and Methods :

Data were analysed for association of risk factors, calculate SSI rates, type of SSIs and cumulative incidence of SSI by category.

Results :

The overall rate of SSIs in this study was 2.5%, approximately 78 % of SSI were superficial incisional type, 12.68% were deep incisional and 8.9% was organ space. Most common micro-organism in wound Swabs was E. coli (51.51%). Wound debridement and Re-suturing was done in 22.76% cases. Cumulative incidence of SSI was relatively higher in patients undergoing Radical Vulvectomy, Wertheim's Radical Hysterectomy and Peri-partum hysterectomy.

Discussion :

Strategies like pre-operative optimisation of patient's health, optimal antimicrobial prophylaxis, ideal skin aseptic techniques, appropriate intraoperative measures can decrease the SSI rate.

Conclusion:

This study emphasizes upon the need of active surveillance for SSI along with preventive strategies to reduce SSIs related morbidity and mortality.

Histopathological Changes in Placenta of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-Cov-2) Infection and its Corelation with Maternal and Perinatal Outcome

Dr. Itishree Jena

Introduction : Histopathological findings in placentas of patients with SARS-CoV-2 infection show a higher prevalence of maternal vascular Malperfusion (MVM) and fetal vascular malperfusion (FVM) such as

- decidual arteriopathy
- atherosclerosis
- fibrinoid necrosis
- mural hypertrophy of membrane arterioles

Fibrinoid necrosis and mural hypertrophy of membrane arterioles are suggestive of placental hypoxia Placental calcification may be the result of exposures to infection, hypoxia or systemic stress.

Aim :

To Study Histopathological Changes In Placenta Of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-Cov-2) Infection And Its Corelation with Maternal and Perinatal Outcome

Objectives :

1. To determine histopathological changes in placentas of pregnant women who are positive for RTPCR of SARSCoV-2.
2. To compare changes in SARSCov-2 positive pregnant women placenta with placenta of pregnant women with no infection.
3. To determine correlation of placental changes with maternal and perinatal outcome of pregnancy.

Materials and Methods :

Place of Study- Department of obstetrics and Gynecology S. N. Medical College, AgraProspective Cohort Study

Sample Size- 45 cases of pregnant women who are positive on RTPCR for SARSCoV-2 and 45 controls who are pregnant with no evidence of infection

Results :

Both groups were comparable with respect to parity pregnancy induced hypertension, gestational diabetes and hypothyroidism . All the women included in study were in third trimester.

Conclusion :

Relative to controls, COVID-19 placentas had increased prevalence of decidual arteriopathy and placental injury reflecting hypoxia and uteroplacental insufficiency within the intervillous space and evidence of maternal and fetal malperfusion even after matching for comorbidities like preeclampsia. Thus a systemic inflammatory or hypercoagulable state can be postulated. Future studies should target infections in different stage of gestation, including in first and second trimesters.

Efficacy of Prophylactic Autologous Platelet Rich Plasma in Caesarean Wound Healing

Dr. Mitali, Dr. Richa Sharma

Introduction :

Surgical site infection complicates 38% deliveries nationwide. PRP has factors like VEGF, PDGF, IGF contributing to wound healing. Thus, administration of prophylactic autologous PRP has shown to improve wound quality.

Aims and Objectives :

Aim : To study the efficacy of Prophylactic Autologous PRP in Caesarean wound healing

Objective : To compare wound healing by REEDA score in both groups on day 3,7or 10(for primary and repeat caesareans respectively) and finally at day 42.

Materials and Methods :

This was a RCT which incorporated 44 women who were divided into two groups after following the inclusion and exclusion criteria. The patients in the case group were administered subcutaneous injection of autologous PRP during caesarean which was prepared in our lab. Whereas, the patients in the control group were managed by the existing hospital protocol. The patients were then assessed on day 3,day7/10 and on day 42 using the REEDA scale.

Results :

The proportion of patients with SSI was significantly lower in the case group (4.55%) compared to control group (31.82%) with a p value of 0.046.The mean REEDA on day 7/10 was 1.77 ± 0.61 for case group which was significantly lower than the control group(3.27 ± 2.1) with a p value of 0.009.Similarly mean REEDA on day 42 was found to be 0.55 ± 0.51 and 1.41 ± 1.01 for case and control respectively, the difference of which was statistically significant.

Conclusion :

Prophylactic administration of Autologous PRP to caesarean wound significantly improved wound quality and reduced the development of SSI .Our study recommends the use of PRP for a seamless postoperative experience.

Kanoon Ki Pathshala

Convenor
Dr. Sadhana Gupta
Dr. Sangeeta Gupta

Advisor
Dr. Sharda Jain

- NIGF Kanoon Ki Pathshala is regular in imparting lessons on important medicolegal areas in obstetric & Gynecology practice

Subjects covered

- Postpartum Haemorrhage
- Medicolegal aspect In Assisted Reproductive Technology
- Medicolegal issues in Missed Fetal Congenital Anomalies & Birth Defects
- Crisp and clear deliberation by our Key Note Speaker, Moderator and Faculties
- Recording link is available on NIGF face book page

Expert Speaks Series

NIGF expert Speaks Series on 19th November on Endocrine Emergencies in Obstetrics by Prof Meenakshi Rohilla and case based discussion by Dr. Nidhi Khera.

Recording link available for those who have missed.

Quiz

NIGF quiz is going on every 2nd and 4th Sunday on diverse subject.

Quiz coordinator Dr Taru Chhaya is doing great job with fine sets of questions.

We heartily congratulate NIGF members for participation in great numbers.

All central as well state chapter winners get certification regularly.

Physical CMEs

- At Alwar Physical CME was organized by Dr. Monika Gupta & Dr. Taru Chhaya on 9th November as key resource persons on subject of overactive bladder. UTI in Pregnancy & Pain in Pregnancy.
- Speaker were Dr. Avadh Agrawal, renowned urologist, Dr. Taru Chaya & Dr. Neelam Jain.
- CME was attended by 40 Participants with good audience interaction. Hearty Congratulations.

- At Gorakhpur on 14th December Physical CME was organized by NIGF in collaboration with Gorakhpur Menopause Society on subject of wrinkle management, nutritional deficiencies and unique program of understanding menopause through Bhagvad Gita.

- Speaker were Dr. Shweta Mishra, Dr. Sunil Gupta & Dr. Amit Ranjan. Key organizer was Dr. Rita Singh, Dr. Madhu Gulati with guidance of Dr. Sadhana Gupta.

At Sri Ganganagar Physical NIGF CME was organized on 12 Dec, on subject of PCOD. Speaker was Dr. Rashmi Vohra. Meeeting was attended by 25-30 Participants.

State Chapter Activities

Rajasthan Chapter organized regular virtual academic program on diverse subjects of Menopause, Osteoporosis with involvement of senior as well young Ob Gyn doctors. Hearty Congratulations to Dr. Veena Acharya, Dr. Monika Gupta and Dr. Taru Chhaya.

North India Gynaecologist Forum
Rajasthan Chapter
In Association with
JMS

Organized a Webinar on

Menopause

December 16th 2024, Monday
3:30 PM to 5 PM


Dr. Sharda Jain
Patron, NIGF


Dr. Sadhana Gupta
President, NIGF


Dr. Veena Acharya
President, NIGF Rajasthan


Dr. Monika Gupta
Secretary, NIGF Rajasthan


Dr. Taru Chhaya
Coordinator, NIGF Rajasthan

Keynote Address on Non hormonal Management of Manopause


Dr. Archana Verma
Past Vice President, FOGSI

Panel Discussion on HRT in Menopause

Moderators


Dr. Taru Chhaya


Dr. Madhulika Agarwal

Expert


Dr. Neelam Jain

Panelists


Dr. Richa Gupta


Dr. Indu Verma Sood


Dr. Bajrang Lal Rar


Dr. Savita Lalgariya


Dr. Mamta Gupta


Dr. Sangeeta Agarwal


Dr. Indra Lamba


Dr. Sarika


Dr. Harshika Bhagat


Dr. Surekha Jangir

MOC


Dr. Monika Gupta

Digital Partner
CLINET

Click Here To Register

North India Gynaecologist Forum
Rajasthan Chapter

Osteoporosis

October 25th 2024, Friday
04:00 PM to 05:00 PM


Dr. Sharda Jain
President, NIGF


Dr. Sadhana Gupta
President, NIGF


Dr. Veena Acharya
President, NIGF Rajasthan


Dr. Monika Gupta
Secretary, NIGF Rajasthan


Dr. Taru Chhaya
Coordinator, NIGF Rajasthan

Keynote Address on Menopause


Dr. Preeti Sharma

Panel Discussion on Osteoporosis

Moderators


Dr. Taru Chhaya


Dr. Monika Gupta

Panelists


Dr. Vinita Bansal


Dr. Charu Johari


Dr. Anu Meena


Dr. Aditi Mourya


Dr. Priyanka Maheshwari


Dr. Shrutee Birla


Dr. Suchitra Narayan

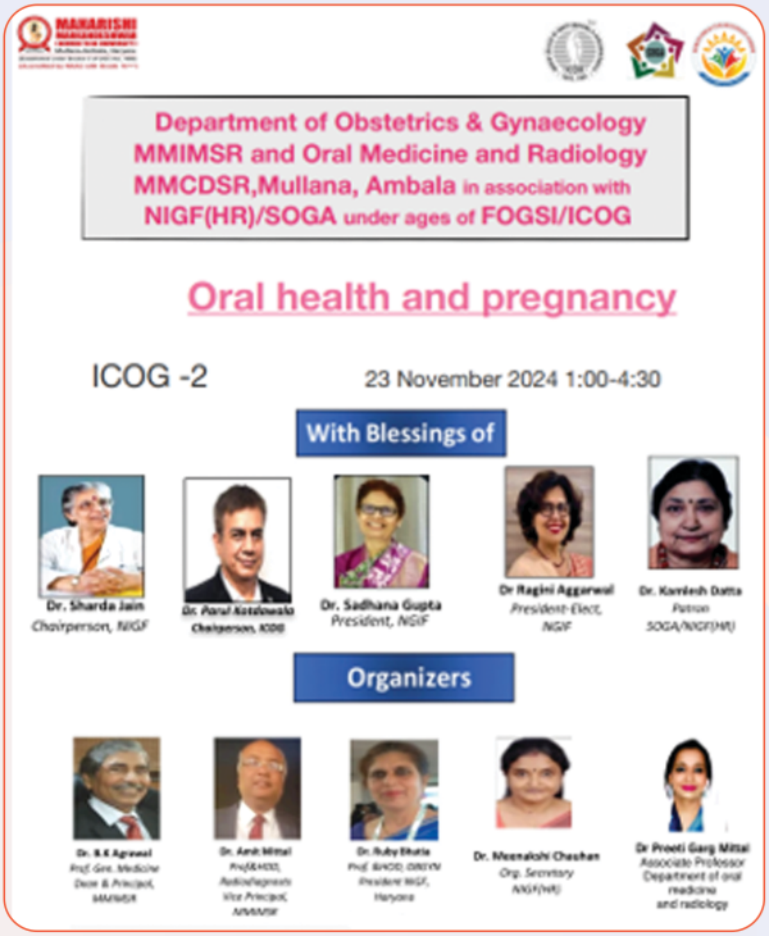

Dr. Vinita Gupta


Dr. Deepika Verma


Dr. Renuka Mundliya

Digital Partner
CLINET

Haryana Chapter NIGF organized virtual CME on 23rd November on oral health in Pregnancy along with ICOG. Speakers were Dr. Meenakshi B Chauhan, Dr. Ruby Bhatia and Dr. Vidushi Tiwari, Dr. Amandeep Kaur and Dr. Preeti Garg. Unique program with deep insights. Hearty Congratulations to organizers Dr. Ruby Bhatia and Dr. Meenakshi Chauhan.



On 10th October Dr. Deepti Chaturvedi organized UP State NIGF Virtual CME on Choriocarcinoma with eminent speaker Dr. Suhana Punneshetty. Hearty Congratulations.

NIGF Celebrates Navratri & International Adolescent Health Week

- On 9th October NIGF celebrated Nav Ratri Festival & International Adolescent week
- Key note address on comprehensive adolescent health by Dr Chandan Kachru.
- Release of 4th issue of NIGF Bulletin on comprehensive Adolescent health
- Poetry competition on subject of Happy & Healthy adolescent
- All winners as well participants awarded certification
- First 7 entries of poetry competition
- Innovative thoughts and beautiful expression
- Hearty congratulations to all winners and participants
- Let us make our youth happy & healthy, dedicated & dynamic.

NIGF Poem Writing Result (9 Oct. 2024)

Rank	Name	Final Scoring
1	Taru Chhaya	185
2	Saroj Aggarwal	180
3	Preeti Pathak	179
4	Ruchi Singhal	175.5
5	Praveen Tomar	174
6	Chetna Bansal	172.5
7	Mridula Sharma	172
7	Amrita Sarkari Jaipuriar	172
8	NIVEDITA PRASHAR	171
9	Yukti Bhardwaj	169.5
10	SUDHA LAVANIA	169.5

1st Winner
Dr. Taru Chhaya

2nd Winner
Dr. Saroj Aggarwal

3rd Winner
Dr Preeti Pathak

4th Winner
Dr Ruchi Singhal

5th Winner
Dr Praveen Tomar

6th Winner
Dr Chetna Bansal

7th Winner
Dr Mridula Sharma

"स्वस्थ एवं सुखद किशोरावस्था – हमारा योगदान"

आओ, किशोरावस्था के सपनों में सुंदर रंग भरें,
नई उमंग, नये सफर के लिए उन्हें तैयार करें।
थामें अपने हाथों में उनकी खुशियों की डोर,
स्वास्थ्य और सुख की कामना हो सब ओर।

तन को दें सबलता, मन को शांति का उपहार,
संतुलित आहार, सही आवरण पर भी हो विचार।
हम हैं स्वास्थ्य के प्रहरी, नवांकुरों को दें सहारा,
उनके जीवन में रोशनी भरने में हो साथ हमारा।

शारीरिक व मानसिक स्वास्थ्य पर हो पूरी पहरेदारी,
सकारात्मकता सोच देने में भी हो हमारी हिस्सेदारी।
क्रीड़ा, विज्ञान व जीवन- शैली का संतुलन सिखाएं,
हर क्षण उनकी मस्तिष्क की ऊर्जा व शक्ति बढ़ाएं।

तो आओ आज हम करते हैं एक वादा .....,
हर किशोरावस्था को स्वस्थ बनाने का हो अपना इरादा।
यही है हमारा सच्चा व वास्तविक योगदान,
यही है हमारी सेवा का अमूल्य व अद्भुत प्रमाण।

7th Winner
Dr Amrita Sarkari Jaipuriar

Adolescents are like budding flowers,
Ready to bloom in their growing hours.

They need care, love and affection,
Along with all the guidance and attention.

We need to be wise, while criticizing!
Choosing our words correctly before commenting.

Allowing them to grow independently,
Paving their way very intelligently.

Holistic development needs discipline.
It can't be forced. It grows within.

Inculcating good habits needs perseverance.
We need to go slow and have patience.

Reading and exercising both have their own role.
Children will grow well and, achieve their goals.

Life is just not about glamour, glitter & being mod.
Teach them humanity and to have faith in God

Dr. Amrita Sarkari Jaipuriar
Gorakhpur

8th Winner
Dr NIVEDITA PRASHAR

HAPPY AND HEALTHY ADOLESCENT:OUR CONTRIBUTION

O mom,I am no more your little princess and prince now,
So many changes I feel are happening but how??
Praises,love,admiration of all were my safe heavens,
All that has turned into remarks full of questions..
My body is changing inside out,
Fear is engulfing my mind and thought..
Little babies,you are not changing but it's a blossom,
Your individuality from this would be awesome..
Periods of five days not to stop you but to prepare for better,
Open yourself to parents and all knowledge you can cater..
Lets shift from cute looks to healthy eating,
lets start maintaining hygiene and vitality.
Talk to doctor if you have any problem,
changes are there to make you gem.
Lets foster healthy relationship
Remember u are important than all friendships
Dear teenager,you are prone to fear
Donot worry our helpers aew always there,
Doesnot matter what happens inside or out
You are growing better,beautiful,...no doubt

DR NIVEDITA PRASHAR
JAMMU .

9th Winner
Dr Yukti Bhardwaj

A vibrant youth, with smile so bright,
A healthy body, a heart full of light.
Doctors' care, with skill and art,
Helped shape this adolescent's happy start.

With vaccinations, they protected their fate,
From diseases that could have sealed their state.
Regular check-ups, with expert eyes,
Caught potential issues, before they arise.

The doctors' guidance, on nutrition and rest,
Helped fuel their growth, and put their best.
Mental health support, when needed most,
Ensured their well-being, like a precious boast.

From pediatricians to Gynaecologists too,
The medical team, saw them through.
Adolescence's challenges, navigated with care,
Thanks to doctors, who showed they truly care.

Let's appreciate these healers true,
Whose work ensures our youth shine through!
For a healthy tomorrow, they pave the way,
Doctors' contributions, every single day...!!!

Dr. Yukti Bhardwaj

10th Winner
Dr SUDHA LAVANIA

In the hush of night, a whisper starts,
A journey blooms within their hearts.
Menarche arrives, a sign of change,
A cycle turning, both wild and strange.

Thalarch whispers secrets of the flesh,
A budding strength, the world to refresh.
Puberche ignites a flame so bright,
Awareness rising, casting off night.

Let's speak of bodies, of power and grace,
In safety's embrace, every heart finds place.
Menstrual hygiene, a right to uphold,
In knowledge, a treasure more precious than gold.

Stand firm against shadows that threaten their peace,
With voices united, let silence cease.
Empower the young with wisdom and care,
To navigate storms, to breathe open air.

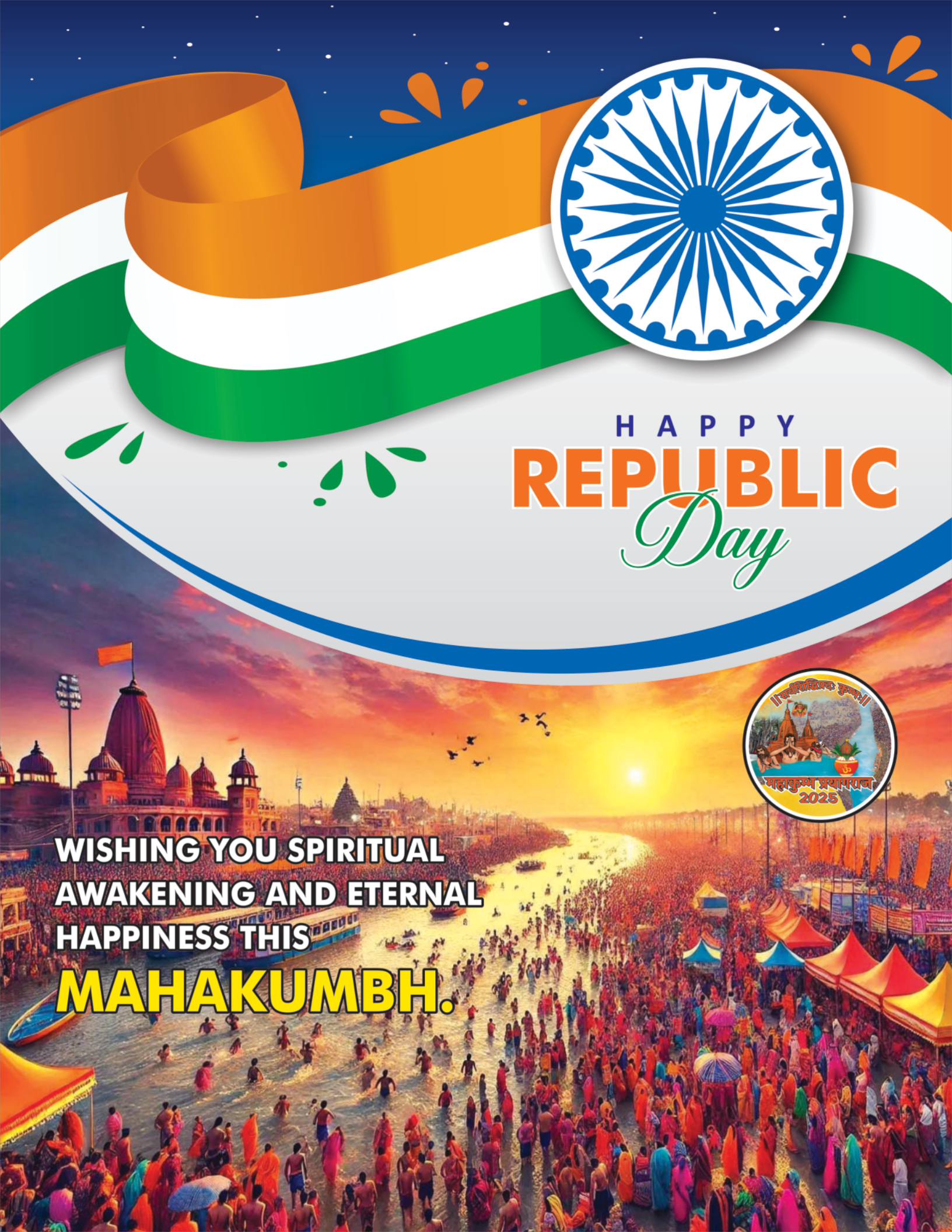
Together we rise, with courage we stand,
Supporting each other, hand in hand.
In every transition, let love be the guide,
With open hearts, let no one hide.

NIGF Diamond Oration

On 24th December NIGF organized Diamond Oration by eminent geneticist & Fetal Medicine Specialist Prof Mandakini Pradhan from Sanjay Gandhi Postgraduate Institute of Medical Sciences Lucknow on topic of Hemoglobinopathies & Obstetrician Perspective.

A highly academic oration with imparting of clear clinical messages on the subject with pearls of wisdom from guest and chairpersons.

Let us join hands to make Our country and Northern India Thalassemia Free Country.


HAPPY
REPUBLIC
Day
WISHING YOU SPIRITUAL
AWAKENING AND ETERNAL
HAPPINESS THIS
MAHAKUMBH.
महाकुम्भ प्रयागराज
2025

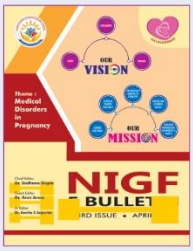
Click on the below Links to read and download previous NIGF Bulletin & NIGF Conference Souvenir

1st Issue of NIGF Bulletin- Maternal Care Bundle

https://drive.google.com/file/d/1FMXQosjREARhA8_AJ3-93oDwC5dg4MiW/view?usp=drive_link

2nd Issue of NIGF Bulletin- Anemia Free India

https://drive.google.com/file/d/1qp3kbPDIkhoW3EjUNqFAABt-heYfLfm3/view?usp=drive_link



3rd Issue of NIGF Bulletin- Medical Disorders in Pregnancy

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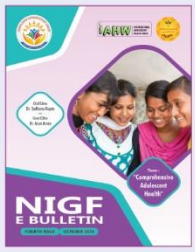

4th Issue of NIGF Bulletin – Comprehensive Adolescent Health

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BLOCK APRIL 27

YOUR DATE

Midterm North India Gynaec Forum (NIGF) Conference

Date :
27th April, 2025

Venue :
Sarovar Portico, Jhansi

Theme :
High Risk Pregnancy - Health for Youth

Please write to nigfoffice@gmail.com for your area of interest

Join **NIGF** for active participation in **All NIGF** academic activities

Show case & share your talent

Involve Innovate implement

Become **Member** of **NIGF**
Rs. 1000/- for 5 years

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