A Review of Contemporary Philosophy
ISSN: 1841-5261, e-ISSN: 2471-089X
Vol 23 (02) 2024
pp. 7612 - 7629

Endometritis: Clinical and Pathological Perspectives for Nursing Practice and Diagnostic Management

[1]-Razan Hamad Yahya Attaf, [2]-Metab Ahmed Mohammed Alsulami,[3]-Osama Alfadhel Yahya Zakri,[4]-Meshal Ali Al-Shehri,[5]-Nashmiah Ateeq Ghenmi Al-Johnani,[6]-Shatha Ali Albargi,[7]-Hind Mohsin Jordi Ibrahim,[8]-Hanin Eid Awad Alsehimy,[9]-Abrar Gazi Sadi,[10]-Asma Mohammed Alshehri,[11]-Jaber Ali Muhammad Moaidy,[12]-Salwa Khalif Al-Anazi,

1. Ksa, Ministry Of Health, Eradah Psychiatric Hospital
2. Ksa, Ministry Of Health, Althagr General Hospital
3. Ksa, Ministry Of Health, Prince Mohammed Bin Nasser Hospital
4. Ksa, Ministry Of Health, King Abdullah Hospital - Bisha
5. Ksa, Ministry Of Health, Al Eis Hospital
6. Ksa, Ministry Of Health, Al-Quwayiyah General Hospital
7. Ksa, Ministry Of Health
8. Ksa, Ministry Of Health, School Health Department
9. Ksa, Ministry Of Health, King Fahd Hospital
10. Ksa, Ministry Of Health, King Fahad Specialist Hospital In Tabuk
11. Ksa, Ministry Of Health, King Fahd Central Hospital Jazan
12. Ksa, Ministry Of Health, Al-Muzahmiyya

Abstract:

Background: Endometritis, an inflammatory condition of the endometrial lining, is classified into acute, chronic, and postpartum forms, each with distinct etiologies and clinical implications. Acute endometritis is often linked to sexually transmitted infections (STIs) or pelvic inflammatory disease (PID), while chronic endometritis is associated with persistent microbial colonization and reproductive complications such as infertility. Postpartum endometritis, a leading cause of postpartum fever, arises from polymicrobial infection following childbirth.

Aim: This article examines the pathophysiology, clinical presentation, diagnostic approaches, and evidence-based management strategies for endometritis, emphasizing the roles of nursing and healthcare professionals in early detection and treatment.

Methods: A comprehensive review of endometritis was conducted, focusing on histopathological findings, microbiological etiology, and clinical guidelines for diagnosis and treatment. The roles of imaging, laboratory testing, and interdisciplinary collaboration were also explored.

Results: Acute endometritis is managed with broad-spectrum antibiotics targeting STIs and anaerobes, while chronic endometritis requires prolonged antibiotic therapy, often guided by endometrial biopsy. Postpartum endometritis necessitates prompt intravenous antibiotics, particularly after cesarean delivery. Diagnostic challenges include overlapping symptoms with other pelvic pathologies and the asymptomatic nature of chronic cases.

Conclusion: Effective management of endometritis relies on timely diagnosis, appropriate antimicrobial therapy, and multidisciplinary care. Chronic endometritis, often undiagnosed, significantly impacts fertility and requires targeted treatment. Postpartum cases demand vigilant monitoring to prevent sepsis. Enhanced patient education and standardized diagnostic criteria are essential to improving outcomes.

Keywords: Endometritis, pelvic inflammatory disease, postpartum infection, chronic endometritis, infertility, antibiotic therapy.

Received: 29 Aug 2024 **Accepted:** 14 oct 2024 **Published:** 27 oct 2024

Introduction:

Endometritis refers to an inflammatory process affecting the endometrium, which is the innermost layer of the uterus. This condition is often caused by microbial infection [1]. When the infectious process extendns beyond the uterus to involve the fallopian tubes, ovaries, or the pelvic peritoneum, it is classified as pelvic inflammatory disease (PID) [2]. The clinical framework distinguishes endometritis into two primary types: acute and chronic. Among acute cases, a specific subtype is known as postpartum endometritis, which occurs in association with pregnancy and typically follows childbirth [3][4]. Acute endometritis that is not related to pregnancy is defined as an endometrial infection that persists for 30 days or less [3]. This form of infection commonly results from sexually transmitted infections or an imbalance in vaginal flora such as bacterial vaginosis. The symptoms often overlap with those of PID and may include fever, pelvic pain, and abnormal vaginal discharge. These similarities have led some clinicians to use the terms PID and acute endometritis interchangeably [2][3]. On histological examination, acute endometritis is marked by the presence of microabscesses and infiltration of neutrophils within the endometrial tissue [5]. Although acute salpingitis, which often coexists with acute endometritis in PID cases, is linked to infertility due to scarring of the fallopian tubes, acute endometritis on its own does not appear to affect fertility outcomes [5][6].

Chronic endometritis involves a prolonged, low-grade inflammatory response in the endometrium that persists for 30 days or more and is not typically associated with pregnancy [3]. This condition is usually a result of microbial colonization rather than acute infection. The histological hallmark of chronic endometritis is the presence of plasma cells within the endometrial stroma, alongside other signs of chronic inflammation. Many individuals with chronic endometritis are asymptomatic. However, when symptoms do occur, they often include abnormal uterine bleeding, painful intercourse, and pelvic discomfort. This condition has been increasingly recognized as a potential factor in recurrent pregnancy loss and unexplained infertility. The diagnostic process is complicated by the lack of specific criteria; most studies identify the detection of endometrial stromal plasma cells as the most consistent histological feature [5][7][8]. Postpartum endometritis remains the leading cause of postpartum infections. This condition commonly presents as a polymicrobial infection involving both aerobic and anaerobic organisms. The pathogenesis involves the upward migration of normal vaginal flora into the uterine cavity during labor and delivery [4]. The incidence of postpartum endometritis is significantly higher in cesarean deliveries, with rates reported to be between five and twenty times greater compared to those following vaginal births [4].

Etiology

Endometritis arises due to the upward migration of microorganisms from the lower reproductive tract, specifically from the cervix and vagina, into the endometrial cavity. The type of pathogens involved can vary depending on whether the case is acute, chronic, or postpartum, and in many cases, identifying the causative organisms may be difficult due to the polymicrobial nature of the infection and variability in clinical presentation [3][4].

Acute Endometritis

The majority of acute endometritis cases, over 85%, are attributed to sexually transmitted infections. Chlamydia trachomatis is considered the primary pathogen in this category, followed by Neisseria gonorrhoeae. Other contributing organisms include bacteria associated with bacterial vaginosis such as Gardnerella vaginalis and anaerobic species [3][6]. Unlike the chronic and postpartum types, acute endometritis tends to have a narrower infectious profile centered around STIs, making it more straightforward in terms of microbiological targeting for empirical therapy. Several patient-related and procedural risk factors increase the likelihood of developing acute endometritis. Women under the age of 25 are particularly vulnerable, as are individuals with a documented history of sexually transmitted

infections or those who engage in high-risk sexual activities, such as having multiple sexual partners. Medical interventions that involve intrauterine manipulation, such as insertion of intrauterine devices or performance of endometrial biopsies, also pose significant risk. These procedures may facilitate the direct entry of pathogens into the endometrial cavity, bypassing the natural barriers of the cervix and promoting microbial colonization and infection [9][5][6].

Chronic Endometritis

The underlying cause of chronic endometritis is often unidentified. However, in cases where an infectious agent is isolated, the condition typically involves a polymicrobial flora consisting of organisms commonly residing in the lower genital tract. This includes both aerobic and anaerobic bacteria. Genital tuberculosis, though less common in developed nations, remains an important cause of chronic granulomatous endometritis in developing countries [5]. In contrast to acute endometritis, Chlamydia trachomatis and Neisseria gonorrhoeae are not frequent causes of chronic endometrial inflammation [5]. The spectrum of microorganisms identified in chronic endometritis includes Streptococcus species, Enterococcus faecalis, Escherichia coli, Klebsiella pneumoniae, and Staphylococcus species. Additionally, atypical pathogens such as Mycoplasma, Ureaplasma, and Gardnerella vaginalis have been implicated. Opportunistic organisms like Pseudomonas aeruginosa and fungal agents such as Saccharomyces cerevisiae and various Candida species may also be involved, especially in immunocompromised individuals or those who have undergone repeated uterine instrumentation [11]. The risk factors contributing to the development of chronic endometritis include the use of intrauterine contraceptive devices, a history of multiple pregnancies (multiparity), previous abortions, and chronic abnormal uterine bleeding. These factors may alter the uterine environment, weaken local immune defenses, or provide a physical medium for microbial attachment and persistence, thereby predisposing the endometrial lining to chronic inflammation [5]. Noninfectious causes are also recognized, although they are less frequently documented. These may include retained intrauterine devices that serve as a nidus for chronic inflammation, endometrial polyps, or submucosal leiomyomas, all of which can trigger localized immune responses that result in chronic histopathological changes [10].

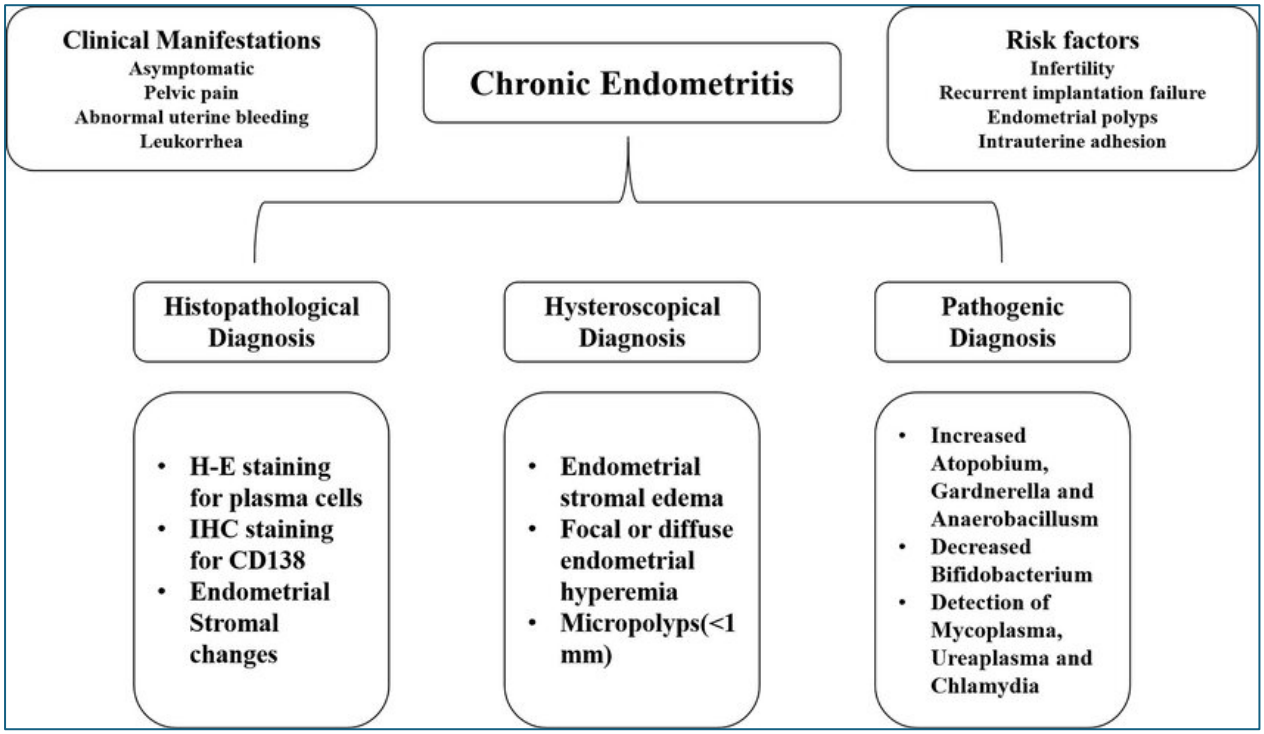

Figure 1: Chronic Endometritis.

Postpartum Endometritis

During pregnancy, the closed cervical canal and intact amniotic membranes provide a protective barrier that limits ascending infection. However, as labor progresses and the cervix dilates, followed by the rupture of membranes, the uterine environment becomes more susceptible to microbial invasion. This risk increases when labor is prolonged or when medical procedures such as repeated vaginal examinations or the use of intrauterine monitoring devices are conducted, which facilitate bacterial access to the upper genital tract [4][12]. Postpartum endometritis is typically a polymicrobial infection, encompassing a broad range of aerobic and anaerobic organisms. Common aerobic gram-positive cocci involved include Group A and B Streptococci, Staphylococcus, and Enterococcus. Among the gram-negative bacilli, Escherichia coli, Klebsiella pneumoniae, and Proteus species are often found. Anaerobic pathogens such as Bacteroides, Peptostreptococcus, Peptococcus, Prevotella, and Clostridium also play a prominent role in the infection process. Other notable organisms include Mycoplasma and, less commonly, Neisseria gonorrhoeae [3][13][14]. While Chlamydia trachomatis is a known cause of late-onset postpartum endometritis, it is considered a rare contributor in early postpartum cases [15]. Some severe and potentially life-threatening infections are caused by more virulent pathogens such as Streptococcus pyogenes, Staphylococcus aureus, Clostridium sordellii, or Clostridium perfringens. These organisms are associated with higher morbidity and mortality, often due to the production of toxins that induce rapid systemic illness, septic shock, or necrotizing infections [16].

The risk profile for postpartum endometritis is broad and includes both obstetric and maternal factors. Cesarean section delivery represents the single most significant risk, particularly when performed after the onset of labor or rupture of membranes. Other procedural risk factors include chorioamnionitis, defined as an intra-amniotic infection that arises during labor, which creates a favorable environment for microbial colonization and subsequent endometrial invasion. Prolonged labor or rupture of membranes increases the time during which bacteria can ascend into the uterus. Additional procedures such as multiple vaginal examinations, insertion of fetal scalp electrodes, or the use of intrauterine pressure catheters also elevate the risk due to mechanical disruption of the protective mucosal barriers [4][17]. Maternal comorbidities further contribute to susceptibility. Women with diabetes mellitus, HIV infection, or obesity are at higher risk of developing postpartum endometritis. These conditions may impair immune response or tissue healing, making the uterine lining more vulnerable to microbial attack. Manual extraction of the placenta and operative vaginal deliveries are also significant contributors, often resulting in trauma or introduction of pathogens into the uterine environment. Understanding the microbial landscape and associated risk factors for each type of endometritis is essential for guiding preventive, diagnostic, and therapeutic decisions. Acute endometritis requires consideration of STI-related organisms and behavioral risk factors. Chronic endometritis necessitates evaluation of polymicrobial involvement and structural uterine abnormalities. Postpartum endometritis, being multifactorial and heavily influenced by delivery practices, underscores the importance of aseptic technique and early recognition of obstetric complications. Accurate identification of causative agents and timely intervention remain central to reducing the burden of this condition in diverse clinical settings.

Epidemiology

Acute endometritis is often not reported as an isolated diagnosis, which complicates efforts to determine its precise incidence. Most cases appear within the broader context of pelvic inflammatory disease (PID), a condition that encompasses infections of the upper genital tract including the endometrium, fallopian tubes, and ovaries. In the United States, PID affects approximately 8% of women, while the incidence in developing countries can reach up to 32%, reflecting disparities in access to screening, treatment, and healthcare infrastructure [18]. Within the U.S., about half of PID cases are attributed to infections with *Chlamydia trachomatis* and *Neisseria gonorrhoeae*, both of which are major causative agents of acute endometritis [19]. These data suggest that the true burden of acute endometritis is likely underestimated, especially in clinical settings where PID is the more commonly recorded diagnosis. Furthermore, differences in sexual behavior, contraceptive use, and STI screening rates may explain regional variation in incidence and prevalence.

Chronic endometritis presents an even greater challenge in epidemiological research due to its often subclinical or nonspecific presentation. Most individuals with the condition are asymptomatic, or they present with mild symptoms that may not prompt evaluation. The condition is increasingly studied in populations with infertility or recurrent pregnancy loss, where its detection is more likely due to more thorough diagnostic workups, including endometrial biopsy and hysteroscopy. Studies have shown that chronic endometritis may be present in up to 30% of individuals experiencing recurrent pregnancy loss, indicating a possible link between persistent endometrial inflammation and implantation failure or miscarriage [7][10]. However, this rate is not consistent across studies, and it varies depending on when during the menstrual cycle endometrial sampling is conducted. Histological findings, including the presence of plasma cells, may fluctuate with hormonal status, contributing to diagnostic variability and inconsistent prevalence estimates. The absence of uniform diagnostic criteria also complicates comparative epidemiology across institutions and populations. Postpartum endometritis is widely recognized as the most common cause of fever in the postpartum period [20]. In women who deliver vaginally without risk factors, the incidence is estimated between 1% and 3%. However, this increases to 5% or 6% in the presence of risk factors such as prolonged labor, multiple vaginal examinations, or manual extraction of the placenta [4]. Cesarean section significantly raises the risk of postpartum endometritis, with an incidence up to 20 times higher than that associated with spontaneous vaginal delivery. The risk becomes even more pronounced when the cesarean is performed after rupture of membranes, suggesting that prolonged exposure of the uterine cavity to vaginal flora increases susceptibility to infection [21][22][13][14]. These observations underline the importance of infection control during labor and delivery, particularly in operative interventions.

Preventive strategies such as the administration of antibiotic prophylaxis before cesarean delivery are effective in reducing the incidence of postpartum endometritis. In the absence of such prophylaxis, approximately 20% of patients undergoing cesarean section develop the condition, indicating a substantial preventive benefit from routine antibiotic use [13]. Despite these measures, untreated or inadequately managed postpartum endometritis can result in severe complications. The condition has a reported mortality rate of up to 17% in cases that progress without effective intervention [14]. These outcomes highlight the need for timely recognition and treatment, particularly in settings with limited resources or barriers to postpartum care. Overall, the epidemiological profile of endometritis reflects significant variation by type, with acute cases commonly linked to STIs and PID, chronic cases often underdiagnosed due to subclinical presentation, and postpartum cases strongly associated with delivery mode and obstetric practices. Improved screening, clearer diagnostic criteria, and expanded access to preventive care are needed to better define the prevalence and reduce the burden of this condition.

Pathophysiology

Acute endometritis develops when an infection ascends from the lower genital tract, primarily involving the cervix and vaginal vault, into the endometrial cavity. The most common causative agent is *Chlamydia trachomatis*. This organism typically initiates infection at the endocervix, compromising the protective epithelial barrier and mucosal integrity of the cervical canal. Once this barrier is disrupted, pathogens gain access to the sterile endometrial lining, where they proliferate and induce inflammation [5]. The host immune system responds by recruiting neutrophils and other inflammatory cells to the endometrial tissue. This immune infiltration can result in histological features such as microabscesses and necrotic debris, indicating acute infection. In many cases, the ascending infection does not remain confined to the endometrium and may extend to involve the fallopian tubes, leading to pelvic inflammatory disease (PID). The pathophysiology of acute endometritis is closely linked to sexual behavior, exposure to STIs, and procedures that breach the endocervical barrier, such as uterine instrumentation. Chronic endometritis, in contrast, is not necessarily caused by organisms that originate in the cervix or vaginal tract. Instead, it often results from long-standing low-grade microbial colonization of the endometrium, which may occur in the absence of overt symptoms or detectable infection in the lower reproductive tract. The condition triggers a persistent immune response characterized by a chronic inflammatory infiltrate. The hallmark of chronic

endometritis is the presence of plasma cells within the endometrial stroma, which are not typically found in healthy endometrial tissue [5][23]. Other pathological findings include micropolyps and stromal edema.

In addition to histological changes, chronic endometritis is associated with alterations in the local immune and hormonal environment. Pro-inflammatory cytokines, including interleukin-1β (IL-1β) and tumor necrosis factor-alpha (TNF-α), are elevated in the affected endometrium. These cytokines stimulate local estrogen production by endometrial glandular cells. The resulting increase in estrogen levels may contribute to the formation of micropolyps and disrupt normal endometrial receptivity, which is a key concern in women experiencing recurrent pregnancy loss or infertility. Hysteroscopic evaluation in these patients frequently reveals micropolyps, which serve as visual markers for chronic endometrial inflammation [5]. Postpartum endometritis has a distinct pathophysiological profile. Under normal conditions, the amniotic sac and intact cervical canal provide an effective barrier that protects the uterine cavity from infection during pregnancy. However, once the amniotic membranes rupture, whether spontaneously or during labor, the pathway becomes open for ascending microorganisms from the cervix and vagina to enter the uterus [4]. The risk of infection increases significantly when the protective environment of the uterus is disrupted by prolonged labor, invasive obstetric procedures, or tissue trauma.

Devitalized or damaged uterine tissue—such as that caused by cesarean section, placental removal, or uterine manipulation—provides an ideal environment for bacterial adherence and colonization. In such cases, the bacterial pathogens not only affect the endometrial lining but can also infiltrate deeper layers of the uterus, including the myometrium. This can result in more extensive inflammation, localized abscess formation, and, in severe cases, systemic infection. The polymicrobial nature of postpartum endometritis means that multiple organisms, including both aerobic and anaerobic bacteria, may be involved simultaneously, each contributing to the inflammatory response and tissue injury [4]. Overall, the pathophysiology of endometritis varies by type, but all forms share common features of microbial invasion, immune response activation, and disruption of normal endometrial function. Acute cases involve rapid neutrophilic infiltration and are often part of broader infections like PID. Chronic endometritis is associated with sustained immune activation and hormonal imbalances, contributing to reproductive complications. Postpartum endometritis arises from loss of the protective intrauterine environment, leading to widespread infection that can affect both the endometrium and underlying myometrium. Understanding these mechanisms is essential for accurate diagnosis and effective treatment.

Histopathology

The histological features of endometritis provide important diagnostic distinctions between its acute and chronic forms. In cases of acute endometritis, the primary histopathological hallmark is the infiltration of neutrophils. These immune cells invade the superficial endometrial epithelium, the glandular lumens, and the endometrial cavity. In some cases, microabscesses form, indicating focal accumulation of neutrophils and tissue necrosis. These findings are consistent with an active inflammatory process that reflects the rapid immune response to acute infection, often caused by ascending sexually transmitted pathogens or other invasive bacteria [5]. Chronic endometritis, by contrast, presents with a different pattern of inflammation. The most consistent and specific histological marker is the presence of endometrial stromal plasma cells (ESPCs). These are not normally found in the healthy endometrium and are identified using immunohistochemical staining, particularly for CD138, a marker for plasma cells. Chronic endometritis is also associated with the presence of small endometrial polyps, termed micropolyps, which are less than 1 mm in diameter. These are frequently seen during hysteroscopic evaluation in affected individuals. In addition to micropolyps, histologic sections often reveal stromal edema, particularly during the proliferative phase of the menstrual cycle, suggesting a prolonged or dysregulated immune response [5][23][7]. Another feature of chronic endometritis is out-of-phase endometrial morphology, where the maturation of the stroma and epithelium is not synchronized. This mismatch may interfere with normal implantation and has been associated with reproductive failure. Furthermore, although less well understood, the accumulation of B lymphocytes in both the endometrial stroma and glands has been observed in chronic endometritis. The pathological role of these B cells remains unclear, but their presence supports the notion of sustained immune activation within the endometrium [5][23][7]. These histological

patterns are essential for differentiating between acute and chronic forms, guiding appropriate treatment strategies, particularly in women presenting with infertility or recurrent pregnancy loss.

History and Physical

Clinical History

The diagnosis of endometritis relies heavily on clinical history and physical examination. Acute and postpartum forms are typically diagnosed based on symptoms and exam findings, whereas chronic endometritis often lacks clear clinical indicators and is usually confirmed histologically. Symptoms can overlap among different types of endometritis, as well as with other gynecological or obstetric conditions. A detailed history is necessary to identify specific clinical features and assess individual risk. This should include a thorough review of obstetric events, contraceptive use, menstrual patterns, sexual history, and exposure to sexually transmitted infections (STIs) [6]. In acute endometritis, the history usually includes the sudden onset of pelvic symptoms in sexually active individuals. The most frequently reported complaints are pelvic pain, abnormal vaginal discharge, dyspareunia, and menstrual irregularities such as postcoital bleeding, intermenstrual spotting, or menorrhagia [6][9]. Some patients may present with systemic features such as low-grade fever, malaise, or general fatigue, especially in moderate to severe cases. Dysuria may also be reported. In advanced or complicated cases of pelvic inflammatory disease, patients might experience right upper quadrant abdominal pain, which may suggest perihepatitis (Fitz-Hugh-Curtis syndrome). Symptoms of tubo-ovarian abscess or salpingitis may be reported as generalized lower abdominal or adnexal pain [6]. Although these symptoms support the diagnosis, some patients may remain asymptomatic, making diagnosis based on history alone unreliable in early or mild cases.

Chronic endometritis is often presented in a more insidious and less defined manner. Many individuals are asymptomatic and are only evaluated in the context of infertility, recurrent pregnancy loss, or repeated embryo implantation failure [5][23]. When symptoms are present, they are typically mild and nonspecific. The most commonly reported complaints include abnormal uterine bleeding, pelvic discomfort, and leukorrhea. These symptoms are subtle and easily misattributed to more common conditions such as dysfunctional uterine bleeding, uterine fibroids, or hormonal imbalance. Consequently, a detailed reproductive history, including assisted reproductive techniques and outcomes, is often key to raising suspicion of chronic endometritis. Postpartum endometritis typically presents in women who have recently delivered or undergone miscarriage. A hallmark symptom is fever, often accompanied by chills, malaise, and fatigue. The timing of symptom onset is important: early-onset postpartum endometritis occurs within 48 hours after delivery, while late-onset cases can appear up to six weeks postpartum [4]. Additional symptoms include uterine tenderness, persistent lower abdominal pain, malodorous or purulent lochia, and delayed uterine involution. A history of cesarean section, manual removal of the placenta, prolonged labor, or numerous vaginal examinations during labor should heighten suspicion [22]. In some cases, symptoms may escalate quickly and can be early signs of sepsis, requiring urgent evaluation.

Clinical Examination

Physical examination remains a key step in the evaluation of suspected endometritis. A focused abdominal and pelvic examination should be performed in all cases. The examination should start with general vital signs to assess fever, tachycardia, hypotension, or other signs of systemic infection. A speculum examination should follow, with attention to the characteristics of the vaginal discharge. In both acute and chronic endometritis, abnormal vaginal secretions may be present and should be sampled for laboratory evaluation, including Gram stain, culture, and nucleic acid amplification testing (NAAT) for STIs. In acute endometritis, cervical motion tenderness is a consistent finding, often accompanied by cervical friability and mucopurulent discharge [9][24]. These signs reflect cervical involvement and inflammation, especially in infections caused by *Chlamydia trachomatis* or *Neisseria gonorrhoeae*. Bimanual examination findings help assess uterine and adnexal tenderness. In acute endometritis, palpation often elicits significant tenderness over the uterus or adnexa, sometimes mimicking appendicitis or ovarian torsion. In contrast,

chronic endometritis may only cause mild uterine sensitivity or no pain at all. Despite this, a tender or irregular uterine contour, especially in patients with abnormal bleeding or reproductive failure, may prompt further investigation [3].

In postpartum endometritis, clinical examination typically reveals more overt signs. The uterus may be enlarged, soft, and tender on palpation, reflecting ongoing infection and subinvolution. The abdominal exam often shows suprapubic pain and guarding. Fever and tachycardia are commonly observed, and the lochia may be foul-smelling or purulent [1][9]. It is critical to evaluate for signs of sepsis, particularly in severe infections caused by virulent organisms such as Group A *Streptococcus* or *Clostridium* species. These infections may present high-grade fever, hypotension, altered mental status, and rapid clinical deterioration. Necrotizing infections or toxic shock syndrome should be suspected in any postpartum patient with disproportionate pain, hemodynamic instability, or signs of end-organ dysfunction [25]. Because clinical signs can overlap with other pelvic and intra-abdominal infections, clinicians must remain vigilant. Symptoms of urinary tract infection retained products of conception, pelvic abscess, or even non-gynecologic conditions like appendicitis can mimic endometritis. Integration of clinical history, examination findings, laboratory testing, and imaging when necessary is vital for accurate diagnosis and early management. In summary, obtaining a complete clinical history and conducting a targeted physical examination are critical steps in the assessment of endometritis. While acute and postpartum endometritis often present with distinct and recognizable symptoms, chronic endometritis requires a high index of suspicion, especially in the context of infertility or pregnancy complications. Proper evaluation of risk factors, timing, and associated signs helps differentiate endometritis from other pelvic pathologies and informs the need for further diagnostic testing or empirical treatment.

Evaluation

The approach to evaluating endometritis depends on its clinical context and type. Acute and postpartum endometritis are primarily diagnosed through clinical findings, including patient history, physical examination, and risk factor assessment. Imaging and laboratory studies are used selectively, often when there is diagnostic uncertainty, failure to respond to empirical treatment, or suspicion of alternative diagnoses. Chronic endometritis differs substantially in that it is typically asymptomatic and requires histologic or hysteroscopic confirmation for diagnosis [1][8].

Acute Endometritis

Acute endometritis shares many clinical features with pelvic inflammatory disease (PID), making differentiation difficult. Because acute endometritis often occurs as part of the spectrum of PID, patients presenting with relevant symptoms are frequently diagnosed and treated for PID rather than isolated endometritis. To minimize the risk of delayed treatment and complications, the Centers for Disease Control and Prevention (CDC) recommend empiric treatment of PID when patients present with any of three minimum clinical criteria: cervical motion tenderness, uterine tenderness, or adnexal tenderness [6][9][3]. As a result, true cases of acute endometritis may be underdiagnosed or misclassified. Diagnostic laboratory tests for acute endometritis begin with screening for common sexually transmitted infections. Nucleic acid amplification tests (NAATs) from endocervical or vaginal swabs are the preferred method to detect *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and *Mycoplasma genitalium*. These organisms are frequently associated with acute endometrial infection. In addition, *Trichomonas vaginalis* and bacterial vaginosis (BV) can be diagnosed through microscopic examination of vaginal secretions using wet mount preparations. In facilities lacking access to microscopy, molecular diagnostic kits are increasingly used to identify these organisms [9][3].

Patients evaluated for PID or endometritis should also undergo testing for syphilis and HIV, as co-infections are common in at-risk populations and may influence treatment decisions and follow-up care [9]. Inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) may be elevated in acute cases but are not diagnostic due to low specificity. Leukocytosis may be present, though a normal white blood cell count does not exclude the diagnosis in milder cases [9][6]. Endometrial biopsy is not routinely indicated in acute endometritis, especially if PID is suspected and the diagnosis is based on

clinical criteria. However, biopsy may be considered in refractory or atypical presentations. Imaging studies, particularly transvaginal ultrasound, are not mandatory in all cases but are recommended if symptoms persist despite treatment, if pelvic abscess is suspected, or if an alternative diagnosis must be excluded. In more complex presentations, pelvic computed tomography (CT) or magnetic resonance imaging (MRI) may be used to evaluate for other conditions such as appendicitis, tubo-ovarian abscess, or adnexal torsion [9][6].

Chronic Endometritis

The diagnosis of chronic endometritis is more challenging because the condition is frequently asymptomatic and lacks specific laboratory or imaging findings. In most cases, diagnosis requires histopathologic confirmation. An endometrial biopsy is obtained to detect the presence of endometrial stromal plasmacytes (ESPCs), which are the key histological marker of chronic inflammation. These cells can be identified using hematoxylin and eosin staining or, more reliably, with immunohistochemistry using the CD138 marker [5][23][7]. Despite the importance of ESPC detection, there is no universally agreed threshold for what constitutes a definitive diagnosis. To address this, the International Working Group for Standardization of Chronic Endometritis Diagnosis proposed specific histological and hysteroscopic criteria [8]. Histologic confirmation includes identifying either 1 to 5 ESPCs per high-power field or small clusters of fewer than 20 ESPCs when stained with CD138. Hysteroscopy can also aid diagnosis and is particularly useful in identifying visual features suggestive of chronic inflammation. Diagnostic findings include endometrial micropolyposis (small protrusions 1–2 mm in size), stromal edema with a thickened or pale endometrial appearance (usually observed during the follicular phase), and focal areas of hyperemia with sharp and irregular margins. Other findings may include large areas of hyperemic endometrium interspersed with white central spots and diffuse focal reddening. These visual patterns, when combined with histology, offer a reliable basis for diagnosis, especially in patients with unexplained infertility or recurrent implantation failure [8][7].

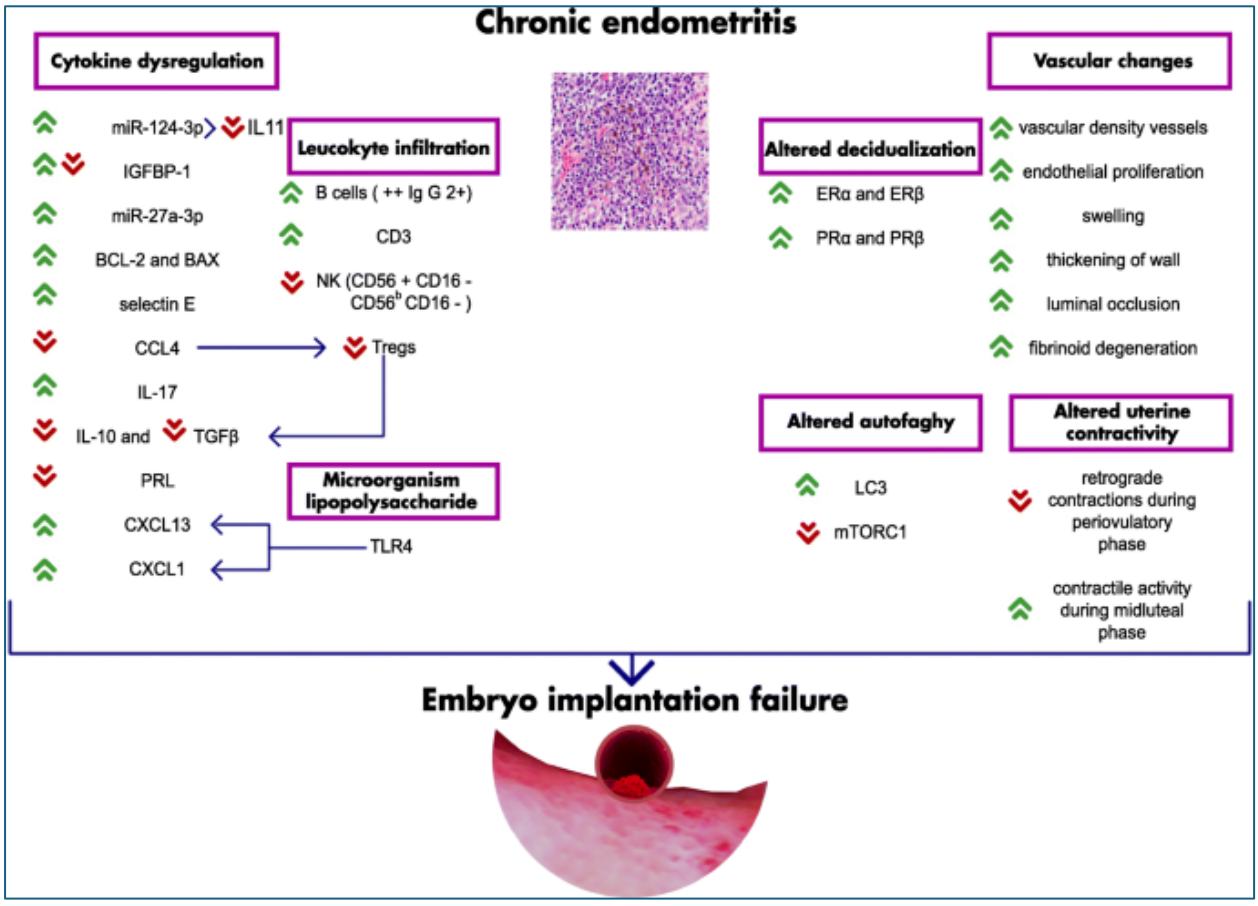

Figure 2: Picture of Altered Markers in Endometritis.

Postpartum Endometritis

Postpartum endometritis is diagnosed clinically and should be suspected in any patient presenting with unexplained fever following delivery or miscarriage. Early-onset cases occur within 48 hours of delivery, while late-onset presentations may occur up to six weeks postpartum [4]. Clinical features such as uterine tenderness, foul-smelling or purulent lochia, and delayed uterine involution strongly support the diagnosis. Laboratory tests assist in ruling out differential diagnoses or evaluating disease progression. White blood cell counts are frequently elevated in postpartum patients due to physiological changes, particularly following cesarean delivery. Nonetheless, leukocytosis ranging between 15,000 to 30,000 cells/μL may indicate infection when interpreted in context. A complete blood count with differential should be obtained, with particular attention to the presence of a left shift (bandemia >10%), which may signal a bacterial infection [27]. Urinalysis and urine culture should be performed to rule out urinary tract infections, which can mimic symptoms of endometritis. Endocervical cultures are rarely helpful, as contamination from vaginal flora is common. They may be useful in cases where group A *Streptococcus* or a sexually transmitted infection is suspected [22][26]. Blood cultures, serum lactate levels, and other sepsis-related tests—including comprehensive metabolic panel, coagulation profile, and surgical site cultures—should be ordered if there are signs of systemic infection or hemodynamic instability (e.g., fever above 38.9 °C, tachycardia, hypotension, altered mental status) [25]. Imaging in postpartum endometritis is not routine but may be warranted in patients with persistent fever, abdominal pain, or when the clinical course is atypical. Transvaginal ultrasound is typically the first-line imaging modality and may help identify retained products of conception or intrauterine debris. However, many postpartum changes—such as uterine enlargement or small intrauterine gas pockets—can appear on ultrasound and are not necessarily pathological. Therefore, findings must be correlated with clinical signs and symptoms. CT imaging may be used to detect complications such as pelvic abscess or septic pelvic thrombophlebitis, especially when ultrasound is inconclusive or when systemic deterioration is evident despite treatment [1]. In conclusion, evaluation of endometritis varies significantly by type. Acute and postpartum forms are often diagnosed through clinical judgment, guided by physical findings, laboratory results, and risk factors. Imaging and specialized testing are reserved for atypical, persistent, or complicated cases. Chronic endometritis, on the other hand, requires endometrial biopsy and sometimes hysteroscopy for accurate diagnosis. A structured approach combining symptom assessment, diagnostic testing, and imaging ensures effective identification and treatment of this condition, ultimately reducing the risk of reproductive complications and systemic infections.

Treatment and Management

Management of endometritis varies based on the type, severity, and clinical setting. Treatment aims to eliminate infection, relieve symptoms, prevent complications such as infertility or sepsis, and reduce recurrence. Acute and postpartum endometritis typically require empiric broad-spectrum antibiotic therapy based on clinical diagnosis, while chronic endometritis is managed after histologic confirmation.

Acute Endometritis

The Centers for Disease Control and Prevention (CDC) provides standardized recommendations for managing acute endometritis, which is often treated similarly to pelvic inflammatory disease (PID) due to overlapping etiology and clinical presentation [28][29]. Outpatient management is appropriate for mild to moderate cases with reliable follow-up. Recommended outpatient regimens combine antibiotics that cover common pathogens, including *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and anaerobes. One preferred regimen includes a single intramuscular dose of ceftriaxone 500 mg, followed by doxycycline 100 mg twice daily and metronidazole 500 mg twice daily, each for 14 days. An alternative is a single dose of cefoxitin 2 g intramuscularly with 1 g oral probenecid, followed by the same doxycycline and metronidazole regimen. Another option is the use of parenteral third-generation cephalosporins such as ceftizoxime or cefotaxime, followed by oral doxycycline and metronidazole [28]. For patients with severe allergies to cephalosporins, fluoroquinolone-based regimens may be used. Levofloxacin 500 mg daily or moxifloxacin 400 mg daily for 14 days, combined with metronidazole 500 mg every 8 hours, is one alternative.

Azithromycin 500 mg intravenously daily for one to two doses, followed by 250 mg orally daily, combined with metronidazole 500 mg orally twice daily for 12 to 14 days, is another option, particularly when *Mycoplasma genitalium* is suspected [28]. Hospitalization is indicated for patients with tubo-ovarian abscess, those who cannot tolerate oral therapy, fail outpatient treatment, have severe illness or systemic symptoms (e.g., vomiting, high fever), or when surgical emergencies cannot be ruled out [28]. Inpatient treatment involves initial parenteral therapy until clinical improvement is observed, typically within 24 to 48 hours, followed by an oral step-down regimen to complete a 14-day course. Recommended inpatient regimens include cefoxitin 2 g intravenously every 6 hours or cefotetan 2 g intravenously every 12 hours, combined with doxycycline 100 mg orally or intravenously every 12 hours. Alternative regimens include ampicillin-sulbactam 3 g intravenously every 6 hours with doxycycline 100 mg, or a combination of clindamycin 900 mg intravenously every 8 hours with gentamicin 3–5 mg/kg intravenously or intramuscularly every 24 hours [28].

Chronic Endometritis

Chronic endometritis treatment is typically initiated after histologic or hysteroscopic diagnosis. The most widely accepted first-line treatment is doxycycline 100 mg orally twice daily for 14 days. This approach targets common organisms involved in chronic endometrial inflammation, including *Streptococcus*, *Enterococcus*, *Gardnerella vaginalis*, *Escherichia coli*, *Mycoplasma*, and *Ureaplasma* [5]. For patients who do not respond to doxycycline, combination regimens may be considered. A common alternative includes metronidazole 500 mg orally daily combined with ciprofloxacin 400 mg orally daily for 14 days. This broadens coverage to include anaerobic and Gram-negative organisms. In patients diagnosed with chronic granulomatous endometritis caused by genital tuberculosis, anti-tubercular therapy is necessary. The standard four-drug regimen includes isoniazid 300 mg daily, rifampicin 450–600 mg daily, ethambutol 800–1200 mg daily, and pyrazinamide 1200–1500 mg daily [5]. Treatment duration typically ranges from 6 to 9 months, depending on the clinical response and local guidelines for tuberculosis management.

Postpartum Endometritis

Postpartum endometritis often requires inpatient care, especially in patients with moderate to severe symptoms, those who underwent cesarean delivery, or patients with signs of systemic infection or sepsis. Empiric intravenous antibiotic therapy should be started promptly and should cover aerobic and anaerobic bacteria, Gram-positive cocci, and Gram-negative bacilli. A Cochrane review identified the combination of clindamycin and gentamicin as the most effective empiric therapy for postpartum endometritis [4]. Gentamicin is dosed at 5 mg/kg intravenously every 24 hours or 1.5 mg/kg every 8 hours, while clindamycin is given as 900 mg intravenously every 8 hours. If patients are group B *Streptococcus* positive or show no improvement after 48 hours, ampicillin may be added to the regimen. Ampicillin can be administered as 2 g intravenously every 6 hours, or as a loading dose of 2 g followed by maintenance doses of 1 g every 4 to 8 hours. Alternatively, ampicillin-sulbactam 3 g every 6 hours can be used [25]. Failure to improve after 72 hours necessitates reconsideration of the diagnosis. Conditions such as retained products of conception, pelvic abscess, pyelonephritis, pneumonia, or septic pelvic thrombophlebitis should be evaluated. At this stage, imaging studies and laboratory workup for systemic infection are warranted. Blood cultures, urinalysis, chest imaging, and pelvic ultrasound or CT can guide further management. Intravenous antibiotics should be continued until the patient has been afebrile for at least 24 hours, with clear improvement in pain and normalization of inflammatory markers such as white blood cell count. There is no strong evidence supporting routine continuation of oral antibiotics after resolution of symptoms in patients who have completed adequate intravenous therapy. Therefore, transition to oral antibiotics should be individualized and generally reserved for mild late-onset cases identified post-discharge [30][4][15].

Monitoring and Follow-up

In all types of endometritis, clinical improvement is expected within 48 to 72 hours after starting appropriate therapy. This includes reduction in fever, pain, abnormal discharge, and normalization of

leukocytosis. If there is no improvement, reconsideration of the diagnosis, need for surgical intervention, or adjustment in antibiotics is necessary. For example, tubo-ovarian abscess may require drainage or surgical management. In chronic endometritis, follow-up often involves repeat endometrial biopsy to confirm histologic resolution, especially in the context of infertility or prior implantation failure. Hysteroscopic reevaluation may also be used to assess for resolution of micro polyps or stromal abnormalities. In postpartum cases, follow-up focuses on monitoring for sepsis resolution and evaluation of reproductive tract healing. Counseling on hygiene, sexual activity, contraception, and recognition of warning signs for recurrence or complications should be provided before discharge. Effective treatment of endometritis depends on timely diagnosis and tailored antibiotic regimens. Acute endometritis is treated with multidrug regimens targeting sexually transmitted and anaerobic pathogens, with hospitalization reserved for severe cases. Chronic endometritis requires histologic diagnosis and longer antibiotic courses, with anti-tubercular therapy used for granulomatous forms. Postpartum endometritis warrants prompt intravenous antibiotic therapy, especially after cesarean delivery, and careful monitoring to prevent severe complications. Adherence to evidence-based protocols and prompt clinical reassessment are essential to achieving favorable outcomes.

Differential Diagnosis

When evaluating a patient for suspected endometritis, a broad differential diagnosis must be considered to avoid misdiagnosis and to ensure timely treatment of other potentially serious conditions. Acute endometritis commonly presents with pelvic or lower abdominal pain, fever, and abnormal vaginal discharge—features that overlap significantly with several gynecologic, urologic, and gastrointestinal disorders. Ectopic pregnancy remains a high-priority exclusion diagnosis in all reproductive-age women with pelvic pain and possible pregnancy. Hemorrhagic or ruptured ovarian cysts and ovarian torsion also present with acute pelvic pain and may mimic endometritis. Ovarian torsion typically presents with sudden-onset unilateral pain and may be accompanied by nausea or vomiting. Endometriosis is another important differential, presenting chronic pelvic pain and dysmenorrhea that may be mistaken for recurrent endometritis. Tubo-ovarian abscess, although often a complication of PID and endometritis, should be considered separately due to its higher severity and need for potential surgical intervention. Acute cystitis or urinary tract infection may present pelvic discomfort and urinary symptoms. Renal stones can cause flank pain radiating to the groin and hematuria, which may mimic lower abdominal or pelvic pathology. Gastrointestinal causes such as appendicitis, diverticulitis, and irritable bowel syndrome also need to be considered, particularly in patients with nonspecific abdominal symptoms or those who do not respond to initial gynecologic treatment strategies [26]. For chronic endometritis, abnormal uterine bleeding (AUB) and infertility are frequent presentations. The American College of Obstetricians and Gynecologists (ACOG) recommends the PALM-COEIN classification system for AUB. This framework divides causes into structural and non-structural categories: polyps, adenomyosis, leiomyomas, malignancy and hyperplasia (PALM); coagulopathy, ovulatory dysfunction, endometrial causes such as chronic endometritis, iatrogenic factors like medications, and not-yet-classified causes (COEIN) [31]. Infertility workups must also explore tubal obstruction, uterine anomalies, ovulatory or hormonal dysfunction, genetic abnormalities, and male infertility factors, as chronic endometritis alone rarely explains the full clinical picture [32]. In the postpartum setting, fever must prompt evaluation beyond endometritis. Differential diagnoses include surgical site infections, urinary tract infections, pyelonephritis, mastitis, postpartum pneumonia, and sepsis. Less commonly, septic pelvic thrombophlebitis and peritonitis may also occur and mimic signs of uterine infection [15][1]. Comprehensive evaluation is necessary to accurately differentiate these conditions.

Prognosis

The overall prognosis of endometritis depends on timely diagnosis, appropriate treatment, and the presence of complications. Postpartum endometritis, when left untreated, carries a high mortality risk, with fatality rates reaching approximately 17% [14]. This elevated risk highlights the importance of early recognition and antibiotic administration. In high-resource settings, where prompt medical intervention is accessible, outcomes are generally favorable, and most patients recover fully without long-term sequelae.

Acute endometritis, particularly when isolated and promptly treated, has an excellent prognosis. However, it is often part of a broader pelvic inflammatory disease (PID) spectrum. When acute endometritis occurs alongside salpingitis, the risk of long-term reproductive complications, especially tubal factor infertility, increases significantly [5][6]. This makes early diagnosis and treatment of PID crucial to preserving fertility in affected individuals. Chronic endometritis presents a more complex picture. Although frequently asymptomatic, it can be a contributing factor in infertility and repeated implantation failure. Studies suggest that treatment of chronic endometritis leads to marked improvements in reproductive outcomes. In one study evaluating patients undergoing fresh day 3 embryo transfer, those who received treatment for chronic endometritis had live birth rates of 60% to 65%, compared to just 6% to 15% among untreated individuals [11]. Another study involving patients with recurrent pregnancy loss showed that live birth rates improved from 7% before treatment to 56% after appropriate therapy [33]. These findings support the view that identifying and managing chronic endometritis can have a substantial positive impact on fertility and pregnancy outcomes.

Complications

Endometritis, if not promptly diagnosed and effectively treated, may lead to serious and sometimes irreversible complications. Acute endometritis, particularly when it occurs as part of pelvic inflammatory disease (PID), has the potential to cause long-term reproductive health issues. The infection can result in tubal scarring and adhesions, leading to tubal factor infertility. Additionally, patients may develop chronic pelvic pain, which can persist even after the resolution of the infection. Ectopic pregnancy is another recognized complication, as damage to the fallopian tubes may prevent normal embryo transport [26]. In severe or inadequately managed cases, the infection may extend beyond the uterus to form a tubo-ovarian abscess, a serious condition that often requires hospitalization and may necessitate surgical drainage. Chronic endometritis is also associated with significant reproductive consequences. It has been linked to repeated implantation failure in assisted reproductive technology procedures and recurrent pregnancy loss. Abnormal uterine bleeding (AUB) is another common complication, leading to diagnostic uncertainty and potential delays in identifying the underlying cause [7]. In the postpartum setting, endometritis can become life-threatening if complications develop. Between 1% and 4% of patients with postpartum endometritis may experience progression to sepsis, intra-abdominal abscesses, pelvic hematomas, septic pelvic thrombophlebitis, or necrotizing fasciitis [22]. These complications often require escalation of care, including intravenous broad-spectrum antibiotics, imaging-guided drainage, or surgical intervention. Cases involving group A *Streptococcus* or *Clostridium* species are especially dangerous due to their association with toxic shock syndrome and rapid clinical deterioration. Early identification and intervention are essential to reduce morbidity and prevent mortality.

Consultations

Management of endometritis often requires coordinated care involving multiple specialties, depending on the clinical presentation and setting. Patients presenting signs of postpartum endometritis should be evaluated and managed by clinicians with obstetric expertise. This is particularly important for patients seen in emergency departments, urgent care centers, or primary care clinics where obstetric services are not routinely provided. Prompt consultation with an obstetrician ensures accurate diagnosis, appropriate treatment, and monitoring for potential complications such as sepsis or retained products of conception [26][34]. Patients with abnormal uterine bleeding (AUB), persistent pelvic pain, or suspected complications from endometritis may require evaluation by a gynecologist. These specialists are equipped to perform diagnostic procedures such as transvaginal ultrasound, hysteroscopy, or endometrial biopsy, which are often necessary for cases of chronic endometritis or when alternative diagnoses are being considered. Gynecologists also provide surgical management when needed, including drainage of abscesses, removal of retained tissue, or in rare cases, hysterectomy for uncontrolled infection. In cases involving infertility or repeated implantation failure linked to chronic endometritis, referral to a reproductive endocrinologist or infertility specialist is appropriate. These professionals can coordinate treatment of the infection while managing assisted reproductive interventions. Their role is especially important when endometritis is identified during fertility evaluations or after failed embryo transfers. Early

involvement of the appropriate specialist—obstetrician, gynecologist, or fertility expert—improves outcomes, reduces delays in care, and ensures patients receive targeted, effective management.

Patient Education

Education plays a critical role in the prevention and management of endometritis, particularly in settings involving surgical delivery or risk of sexually transmitted infections. Cesarean delivery significantly increases the risk of developing postpartum endometritis compared to vaginal delivery. For this reason, the American College of Obstetricians and Gynecologists (ACOG) recommends administering prophylactic antibiotics prior to all cesarean deliveries. This recommendation is supported by a Cochrane review, which demonstrated a marked reduction in postpartum infections, including endometritis, when prophylactic antibiotics were used appropriately. The ACOG Practice Bulletin advises that patients undergoing cesarean delivery should receive a single dose of a first-generation cephalosporin, such as cefazolin 1 g administered intravenously within one hour before the surgical skin incision. This regimen helps prevent bacterial contamination during the procedure and has been proven to lower infection rates significantly. Furthermore, evidence supports that administering azithromycin 500 mg IV, infused over one hour, in addition to cefazolin, provides added protection for patients undergoing cesarean delivery after the onset of labor. This combination reduces maternal infection risk and improves clinical outcomes [35]. Informed consent discussions before cesarean delivery should include education about the elevated risk of postpartum infections and the preventive strategies in place, including antibiotic prophylaxis. This conversation ensures that patients understand the risks and benefits of the procedure and are better prepared for postoperative care. Outside of pregnancy, preventive strategies for endometritis focus on reducing the risk of pelvic inflammatory disease (PID) and sexually transmitted infections (STIs). This includes the timely treatment of symptomatic bacterial vaginosis (BV) and routine STI screening. All sexually active women under 25 years of age and women aged 25 and older with risk factors such as new or multiple sexual partners, a partner known to have an STI, or a partner with concurrent partners should be screened for STIs. Behavioral counseling is also recommended for adolescents and adults at increased risk for STIs. These interventions are aimed at early identification and treatment of infections that could ascend and result in endometrial inflammation [3][26]. Overall, patient education should emphasize the importance of preventive measures, adherence to treatment, and follow-up care to avoid complications and preserve reproductive health.

Other Issues

Endometritis is defined as inflammation of the endometrial lining of the uterus and presents in different clinical contexts, each requiring specific attention. It may occur acutely, often as part of pelvic inflammatory disease (PID), or chronically, often undetected until complications such as infertility or recurrent pregnancy loss arise. While it may develop outside of pregnancy, postpartum endometritis is the most common infection following delivery. Clinicians should maintain a high index of suspicion in any postpartum patient presenting with unexplained fever, especially within six weeks of delivery. Supporting clinical signs such as purulent or foul-smelling lochia and uterine tenderness strongly suggest postpartum endometritis. Early recognition and intervention are critical. Prompt obstetric consultation and appropriate antibiotic therapy help reduce morbidity and prevent escalation to sepsis or other severe complications. In non-obstetric populations, acute endometritis is commonly considered a manifestation of PID and is rarely seen as an isolated diagnosis. It often coexists with salpingitis and cervicitis. Because acute endometritis and PID share overlapping symptoms and clinical criteria, distinguishing between them is often not necessary. Clinical guidelines recommend managing both conditions similarly, with empiric broad-spectrum antibiotics targeting likely pathogens. Delay in treatment may increase the risk of reproductive complications. Chronic endometritis poses different challenges. It is typically asymptomatic or presents with vague complaints such as abnormal uterine bleeding or pelvic discomfort. Its strong association with repeated implantation failure and recurrent miscarriage makes its detection crucial in infertility workups. Histologic confirmation via endometrial biopsy is often needed for diagnosis, and treatment with antibiotics can significantly improve reproductive outcomes. Regardless of the type, the cornerstone of endometritis treatment remains targeted antimicrobial therapy. The severity of disease presentation

should guide the urgency of intervention. In patients with signs of systemic infection or hemodynamic instability, immediate resuscitative measures including fluid administration, early initiation of intravenous antibiotics, and hospitalization should be prioritized. The aim is to prevent progression to severe complications such as abscess formation, pelvic thrombophlebitis, or septic shock.

Enhancing Healthcare Team Outcomes

Postpartum endometritis remains the leading cause of postpartum fever and requires early recognition and coordinated response from the healthcare team. Nurses play a frontline role in identifying early signs of infection, such as unexplained fever, uterine tenderness, purulent lochia, and general malaise. Their continuous bedside presence allows them to observe subtle clinical changes that may indicate the onset of endometritis. Prompt recognition by nursing staff and immediate notification of the obstetrician can significantly shorten the time to diagnosis and treatment initiation, reducing the risk of severe complications including sepsis. Nurses are also essential in executing initial treatment protocols. Once the diagnosis is suspected, intravenous access, fluid administration, laboratory sampling, and early antibiotic delivery often fall under nursing responsibilities. Monitoring the patient's response to treatment—vital signs, urine output, and laboratory trends—requires close attention and frequent reassessment by nurses. This monitoring ensures that any deterioration is promptly communicated to the medical team for escalation of care. Education of the patient and family regarding signs of worsening infection or the importance of follow-up also falls under the nursing scope. Effective treatment of endometritis demands strong interprofessional collaboration. Physicians rely on the accuracy of nursing assessments and depend on timely communication to make clinical decisions. Pharmacists contribute by optimizing antimicrobial regimens, adjusting dosages, and preventing medication errors. Radiologists and imaging technicians support diagnosis when further clarification is needed, particularly in cases involving retained products of conception or pelvic abscesses. In emergency settings or outpatient centers, where patients with acute or chronic endometritis may first present, nurses play an important role in triaging symptoms, documenting obstetric and sexual histories, and ensuring the patient receives rapid medical attention. Early obstetric or gynecologic consultation should be facilitated, and nurses must coordinate logistics to expedite specialist evaluation. If surgical intervention becomes necessary, for example in cases involving abscess drainage or retained placental tissue, the role of the perioperative nursing team expands to preoperative preparation, intraoperative support, and postoperative recovery monitoring. Anesthesiologists and surgical teams depend on accurate preoperative assessments and stabilization efforts led by nursing staff. Improved outcomes in patients with endometritis depend on seamless coordination among all healthcare team members. Nursing professionals serve as the foundation of this effort, ensuring rapid response, continuous care, and effective communication throughout the patient's clinical course.

Conclusion:

Endometritis, whether acute, chronic, or postpartum, poses significant clinical challenges due to its varied presentations and potential complications. Acute endometritis, frequently associated with STIs and PID, demands prompt antibiotic therapy to prevent long-term sequelae such as tubal infertility and chronic pelvic pain. The CDC's treatment guidelines, emphasizing broad-spectrum coverage for *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, and anaerobes, remain foundational in managing acute cases. However, the overlap with PID often leads to underdiagnosis of isolated endometrial inflammation, highlighting the need for heightened clinical suspicion in high-risk populations. Chronic endometritis, though often asymptomatic, has emerged as a critical factor in reproductive dysfunction, particularly recurrent pregnancy loss and implantation failure. Histologic diagnosis via endometrial biopsy (e.g., CD138 staining for plasma cells) is essential, yet the lack of standardized diagnostic criteria complicates consistent identification. Treatment with doxycycline or combination antibiotics improves reproductive outcomes, underscoring the importance of screening in infertility workups. Future research should refine diagnostic thresholds and evaluate the role of microbial resistance in refractory cases. Postpartum endometritis, the most common postpartum infection, carries substantial morbidity without timely intervention. Cesarean delivery, prolonged labor, and invasive obstetric procedures elevate risk, necessitating prophylactic antibiotics per ACOG guidelines. Empiric therapy with clindamycin and gentamicin is highly effective,

though worsening symptoms should prompt evaluation for abscess or septic thrombophlebitis. Maternal mortality, though rare in high-resource settings, remains a concern in cases of necrotizing infections or delayed treatment. The management of endometritis hinges on interdisciplinary collaboration. Nurses play a pivotal role in early symptom recognition, while obstetricians, gynecologists, and infectious disease specialists guide targeted therapy. Pharmacists ensure appropriate antibiotic selection, particularly in penicillin-allergic or polymicrobial cases. For chronic endometritis, fertility specialists contribute to long-term care, addressing underlying inflammation that may impede conception. Patient education is equally critical. Prenatal counseling on infection prevention, postpartum hygiene, and STI screening reduces incidence, while clear discharge instructions mitigate readmission risks. In chronic cases, explaining the link between endometritis and infertility fosters adherence to treatment. In conclusion, endometritis requires a tailored approach based on its subtype and clinical context. Acute and postpartum cases benefit from rapid, guideline-driven therapy, while chronic endometritis necessitates a nuanced diagnostic and therapeutic strategy to preserve fertility. Strengthening interdisciplinary protocols, advancing diagnostic precision, and prioritizing patient education will optimize outcomes across all forms of this consequential condition.

References:

1. Gonzalo-Carballes M, Ríos-Vives MÁ, Fierro EC, Azogue XG, Herrero SG, Rodríguez AE, Rus MN, Planes-Conangla M, Escudero-Fernandez JM, Coscojuela P. A Pictorial Review of Postpartum Complications. Radiographics. 2020 Nov-Dec;40(7):2117-2141.
2. Soper DE, Wiesenfeld HC. The Continued Challenges in the Diagnosis of Acute Pelvic Inflammatory Disease: Focus on Clinically Mild Disease. J Infect Dis. 2021 Aug 16;224(12 Suppl 2):S75-S79.
3. Ravel J, Moreno I, Simón C. Bacterial vaginosis and its association with infertility, endometritis, and pelvic inflammatory disease. Am J Obstet Gynecol. 2021 Mar;224(3):251-257.
4. Mackeen AD, Packard RE, Ota E, Speer L. Antibiotic regimens for postpartum endometritis. Cochrane Database Syst Rev. 2015 Feb 02;2015(2):CD001067.
5. Kitaya K, Takeuchi T, Mizuta S, Matsubayashi H, Ishikawa T. Endometritis: new time, new concepts. Fertil Steril. 2018 Aug;110(3):344-350.
6. Gradison M. Pelvic inflammatory disease. Am Fam Physician. 2012 Apr 15;85(8):791-6.
7. Pirtea P, Cicinelli E, De Nola R, de Ziegler D, Ayoubi JM. Endometrial causes of recurrent pregnancy losses: endometriosis, adenomyosis, and chronic endometritis. Fertil Steril. 2021 Mar;115(3):546-560.
8. Cicinelli E, Vitagliano A, Kumar A, Lasmar RB, Bettocchi S, Haimovich S., International Working Group for Standardization of Chronic Endometritis Diagnosis. Unified diagnostic criteria for chronic endometritis at fluid hysteroscopy: proposal and reliability evaluation through an international randomized-controlled observer study. Fertil Steril. 2019 Jul;112(1):162-173.e2.
9. Ross J, Guaschino S, Cusini M, Jensen J. 2017 European guideline for the management of pelvic inflammatory disease. Int J STD AIDS. 2018 Feb;29(2):108-114.
10. Song D, Feng X, Zhang Q, Xia E, Xiao Y, Xie W, Li TC. Prevalence and confounders of chronic endometritis in premenopausal women with abnormal bleeding or reproductive failure. Reprod Biomed Online. 2018 Jan;36(1):78-83.
11. Cicinelli E, Matteo M, Trojano G, Mitola PC, Tinelli R, Vitagliano A, Crupano FM, Lepera A, Miragliotta G, Resta L. Chronic endometritis in patients with unexplained infertility: Prevalence and effects of antibiotic treatment on spontaneous conception. Am J Reprod Immunol. 2018 Jan;79(1)
12. Committee Opinion No. 712: Intrapartum Management of Intraamniotic Infection. Obstet Gynecol. 2017 Aug;130(2):e95-e101.
13. Smaill FM, Grivell RM. Antibiotic prophylaxis versus no prophylaxis for preventing infection after cesarean section. Cochrane Database Syst Rev. 2014 Oct 28;2014(10):CD007482.
14. Meaney-Delman D, Bartlett LA, Gravett MG, Jamieson DJ. Oral and intramuscular treatment options for early postpartum endometritis in low-resource settings: a systematic review. Obstet Gynecol. 2015 Apr;125(4):789-800.
15. Dalton E, Castillo E. Post partum infections: A review for the non-OBGYN. Obstet Med. 2014 Sep;7(3):98-102.

16. Donders G, Greenhouse P, Donders F, Engel U, Paavonen J, Mendling W. Genital Tract GAS Infection ISIDOG Guidelines. J Clin Med. 2021 May 10;10(9)
17. Prevention of Group B Streptococcal Early-Onset Disease in Newborns: ACOG Committee Opinion, Number 782. Obstet Gynecol. 2019 Jul;134(1):1.
18. Haggerty CL, Ness RB. Diagnosis and treatment of pelvic inflammatory disease. Womens Health (Lond). 2008 Jul;4(4):383-97.
19. Kreisel KM, Llata E, Haderxhanaj L, Pearson WS, Tao G, Wiesenfeld HC, Torrone EA. The Burden of and Trends in Pelvic Inflammatory Disease in the United States, 2006-2016. J Infect Dis. 2021 Aug 16;224(12 Suppl 2):S103-S112.
20. Chaim W, Bashiri A, Bar-David J, Shoham-Vardi I, Mazor M. Prevalence and clinical significance of postpartum endometritis and wound infection. Infect Dis Obstet Gynecol. 2000;8(2):77-82.
21. Boggess KA, Tita A, Jauk V, Saade G, Longo S, Clark EAS, Esplin S, Cleary K, Wapner R, Letson K, Owens M, Blackwell S, Beamon C, Szychowski JM, Andrews W., Cesarean Section Optimal Antibiotic Prophylaxis Trial Consortium. Risk Factors for Postcesarean Maternal Infection in a Trial of Extended-Spectrum Antibiotic Prophylaxis. Obstet Gynecol. 2017 Mar;129(3):481-485.
22. Karsnitz DB. Puerperal infections of the genital tract: a clinical review. J Midwifery Womens Health. 2013 Nov-Dec;58(6):632-42.
23. Kitaya K, Ishikawa T. Chronic endometritis: simple can be harder than complex? Fertil Steril. 2021 Jun;115(6):1443-1444.
24. Soper DE. Pelvic inflammatory disease. Obstet Gynecol. 2010 Aug;116(2 Pt 1):419-428.
25. Shields A, de Assis V, Halscott T. Top 10 Pearls for the Recognition, Evaluation, and Management of Maternal Sepsis. Obstet Gynecol. 2021 Aug 01;138(2):289-304.
26. Curry A, Williams T, Penny ML. Pelvic Inflammatory Disease: Diagnosis, Management, and Prevention. Am Fam Physician. 2019 Sep 15;100(6):357-364.
27. Conde-Agudelo A, Romero R, Jung EJ, Garcia Sánchez ÁJ. Management of clinical chorioamnionitis: an evidence-based approach. Am J Obstet Gynecol. 2020 Dec;223(6):848-869.
28. Hazra A, Collison MW, Davis AM. CDC Sexually Transmitted Infections Treatment Guidelines, 2021. JAMA. 2022 Mar 01;327(9):870-871.
29. Dalby J, Stoner BP. Sexually Transmitted Infections: Updates From the 2021 CDC Guidelines. Am Fam Physician. 2022 May 01;105(5):514-520.
30. DeNoble AE, Kuller JA, Heine RP, Dotters-Katz S. Antibiotics for the Prevention and Treatment of Postsurgical Obstetric Infections. Obstet Gynecol Surv. 2018 Aug;73(8):475-485.
31. Committee on Practice Bulletins—Gynecology. Practice bulletin no. 128: diagnosis of abnormal uterine bleeding in reproductive-aged women. Obstet Gynecol. 2012 Jul;120(1):197-206.
32. Infertility Workup for the Women's Health Specialist: ACOG Committee Opinion, Number 781. Obstet Gynecol. 2019 Jun;133(6):e377-e384.
33. McQueen DB, Perfetto CO, Hazard FK, Lathi RB. Pregnancy outcomes in women with chronic endometritis and recurrent pregnancy loss. Fertil Steril. 2015 Oct;104(4):927-931.
34. Wouk N, Helton M. Abnormal Uterine Bleeding in Premenopausal Women. Am Fam Physician. 2019 Apr 01;99(7):435-443.
35. Committee on Practice Bulletins-Obstetrics. ACOG Practice Bulletin No. 199: Use of Prophylactic Antibiotics in Labor and Delivery. Obstet Gynecol. 2018 Sep;132(3):e103-e119.

ملخص:

الخلفية : التهاب بطانة الرحم، وهو حالة التهابية في البطانة الداخلية للرحم، يصنف إلى ثلاثة أشكال: الحاد، المزمن، وما بعد الولادة، لكل منها مسببات وتداعيات سريرية مميزة. يرتبط الالتهاب الحاد عادةً بالعدوى المنقولة جنسياً (STIs) أو مرض التهاب الحوض(PID) ، بينما يرتبط الالتهاب المزمن بالاستعمار الميكروبي المستمر ومضاعفات تناسلية مثل العقم. أما التهاب بطانة الرحم بعد الولادة، وهو أحد الأسباب الرئيسية للحمى بعد الولادة، فينتج عن عدوى متعددة الميكروبات بعد الولادة.

الهدف :تبحث هذه المقالة في الفيزيولوجيا المرضية، والمظاهر السريرية، والمناهج التشخيصية، واستراتيجيات الإدارة القائمة على الأدلة لالتهاب بطانة الرحم، مع التركيز على أدوار الممرضات والمتخصصين الصحيين في المعامل في الكشف المبكر والعلاج.

المنهجية :تم إجراء مراجعة شاملة لالتهاب بطانة الرحم، مع التركيز على النتائج النسيجية المرضية، والمسببات الميكروبيولوجية، والمبادئ التوجيهية السريرية للتشخيص والعلاج. كما تم استكشاف أدوار التصوير، الاختبارات المعملية، والتعاون متعدد التخصصات.

النتائج :يتم التعامل مع الالتهاب الحاد بالمضادات الحيوية واسعة الطيف التي تستهدف العدوى المنقولة جنسياً واللاهوائيات، بينما يتطلب الالتهاب المزمن علاجاً بالمضادات الحيوية لفترة طويلة، غالباً ما يتم توجيهه عن طريق خزعة بطانة الرحم. ويستلزم التهاب ما بعد الولادة استخدام المضادات الحيوية الوريدية الفورية، خاصة بعد الولادة القيصرية. تشمل التحديات التشخيصية تداخل الأعراض مع أمراض الحوض الأخرى والطبيعة غير العرضية للحالات المزمنة.

الاستنتاج :تعتمد الإدارة الفعالة لالتهاب بطانة الرحم على التشخيص في الوقت المناسب، والعلاج بالمضادات الحيوية المناسبة، والرعاية متعددة التخصصات. يؤثر الالتهاب المزمن، الذي غالباً ما يكون غير مشخص، بشكل كبير على الخصوبة ويتطلب علاجاً مستهدفاً. بينما تتطلب حالات ما بعد الولادة مراقبة دقيقة لمنع تسمم الدم. إن تعزيز تثقيف المرضى والمعايير التشخيصية الموحدة أمران ضروريان لتحسين النتائج.

الكلمات المفتاحية :التهاب بطانة الرحم، مرض التهاب الحوض، عدوى ما بعد الولادة، التهاب بطانة الرحم المزمن، العقم، العلاج بالمضادات الحيوية.