Chapter

Decoding Endometriosis: A Comprehensive Guide to Understanding Symptoms and Impacts

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Abstract

Up to 10% of all women suffer with endometriosis, a chronic inflammatory gynecological condition, that is dependent on estrogen. This prevalence rises to 30–50% among women who experience infertility and/or severe pelvic pain. Endometriosis is a disease that is remarkably underdiagnosed and undertreated due to a lack of exact knowledge about it. It takes an unreasonable amount of time (8–12 years) between the onset of symptoms and a conclusive diagnosis. This is due to the fact that the majority of the symptoms are non-specific and there are no non-invasive diagnostic procedures that can offer a conclusive diagnosis. These days, assessing all symptoms and indicators that may lead us to question the presence of endometriosis is crucial. We will investigate all symptoms of this disorder in this chapter.

Keywords: endometriosis, symptoms, signs, fatigue, chronic pelvic pain

1. Introduction

Endometriosis is a persistent inflammatory, estrogen-dependent disorder characterized by the growth of endometrial-like tissue outside the uterine cavity [1, 2]. This condition affects an estimated 175 million women of reproductive age worldwide [3].

Endometriosis is estimated to affect one in ten Australian women of reproductive age, incurring direct medical and surgical costs exceeding $6 billion annually for women over 18 years old [4]. The definitive diagnosis of endometriosis necessitates laparoscopy and histopathology [5]. For many women, the interval between the onset of symptoms and diagnosis can exceed 8 years. Consequently, there is significant interest in identifying clinical features that could predict the presence of endometriosis and reduce the delay in commencing active treatment [6].

Endometriosis is influenced by several known risk factors, including early menarche, late menopause, short menstrual cycles, low body mass index (BMI), and low parity [7, 8]. The etiopathogenesis of endometriosis remains not fully understood.

Potential contributing factors include uterine hyperperistalsis and hyperestrogenism, alongside genetic factors, the implantation theory, and cellular metaplasia [9, 10].

It is widely believed that the extent of anatomical distortion caused by adhesions and fibrosis from endometriosis correlates with higher incidences of infertility. Additionally, soluble factors such as inflammation, oxidative stress, hormonal abnormalities, and immune dysregulation play significant roles in infertility among endometriosis patients. Chronic wounds, including those from endometriosis, diabetic foot ulcers, and other non-healing conditions, undergo recurrent tissue damage and repair cycles [11, 12]. In endometriosis, fibrosis is induced by inflammatory responses, leading to processes like epithelial-mesenchymal transition (EMT), fibroblast-myofibroblast transdifferentiation (FMT), and smooth muscle metaplasia (SMM), perpetuating the cycle of wound healing and tissue remodeling [13, 14].

The classical clinical presentation of endometriosis includes dysmenorrhea, dyspareunia, infertility, and menstrual cycle-related lower abdominal pain. These symptoms can guide clinicians toward the correct diagnosis [15, 16]. However, in Germany, endometriosis is often diagnosed with a delay of up to 10 years, primarily due to misdiagnosis. This issue is particularly pronounced in cases of extragenital endometriosis (EE), which affects approximately 9% of women with endometriosis [17].

EE cases are frequently first presented to non-gynecological specialties, leading to delayed diagnosis and chronic pain, which can dysregulate the nervous system and cause abnormal pain patterns. This necessitates a more complex differential diagnosis process, having significant physical, psychological, and social impacts. Early recognition and proper treatment initiation are crucial [18, 19]. Recent research has concentrated on identifying reliable biomarkers for endometriosis, encompassing a wide range of indicators. These include immunologic markers such as immune cells, antibodies, and cytokines, as well as genetic and biochemical markers like microRNAs, long non-coding RNAs (lncRNAs), circulating and mitochondrial nucleic acids. Additionally, some hormones, glycoproteins, and signaling molecules have also been identified as potential biomarkers [20, 21].

The diagnostic process begins with a thorough clinical history, exploring whether symptoms correlate with menstrual cycle phases. Clinical examination includes speculum examination, palpation (including rectovaginal palpation), transvaginal ultrasound, and renal ultrasound. Diagnostic laparoscopy is the gold standard for histological confirmation [22].

Identifying superficial diseases, peritoneal lesions, or early/mild deep endometriosis through imaging techniques remains challenging, which suggests that a negative result does not exclude the presence of endometriosis. However, transvaginal sonography (TVS) and magnetic resonance imaging (MRI) are effective for detecting more advanced stages of the condition. Severe endometriosis is characterized by extensive adhesions to surrounding organs, such as significant inflammatory adhesions between ovarian endometrioma and the rectum. TVS is particularly valuable for diagnosing adhesions via dynamic manipulation of pelvic organs, where reduced ovarian mobility and limited sliding between the posterior uterine serosa and bowel indicate adhesion presence [23, 24].

Women displaying TVS signs of ovarian endometriomas exhibit higher levels of ovarian immobility than those without these features, with a sensitivity and specificity of 89% and 90%, respectively [25, 26]. The capacity of MRI to detect adhesions and obliteration of the pouch of Douglas is similar to that of dynamic TVS, which diminishes the necessity for routine MRI following TVS. Thus, the diagnostic

precision of dynamic TVS rivals may even surpass, that of routine MRI, although MRI offers greater objectivity and reproducibility. Both TVS and MRI serve as critical tools in assessing the severity of endometriosis, particularly in identifying adhesions, and may contribute to establishing a classification for endometriosis-associated pain. Conversely, a major challenge remains unresolved regarding endometriosis-related infertility, as imaging techniques focused on structural anomalies may not correlate with the progression of infertility severity [25, 27, 28].

Due to the chronic nature of endometriosis, a long-term, personalized treatment plan is essential, encompassing both conservative (symptomatic and hormonal) and surgical treatments, with the potential integration of complementary medicine. Surgical indications include organ destruction, differential diagnosis for sterility, and persistent pain, with a goal of complete laparoscopic resection where possible. Studies have not demonstrated a clear advantage of surgical treatment over pharmacotherapy for endometriosis-associated pain. Pharmacotherapy aims to achieve secondary amenorrhea, with dienogest being the first-line drug. Other options include combined oral contraceptives, gonadotropin-releasing hormone (GnRH) analogs, and local progestins. In order to lower the likelihood of recurrence, hormonal therapy is advised following surgery, unless pregnancy is urgently wanted [9].

2. Gastrointestinal symptoms

Bowel endometriosis is defined by the presence of endometriotic lesions that infiltrate at least the muscular layer of the intestinal wall [29]. Superficial endometriotic lesions, which only penetrate the intestinal serosa, should not be classified as bowel endometriosis and are generally asymptomatic. This condition is estimated to affect between 5% and 25% of patients diagnosed surgically with endometriosis [30]. The majority of bowel endometriotic nodules are located at the rectosigmoid junction and rectum (65.7%); however, lesions can also be noted in the sigmoid colon (17.4%), caecum and ileocecal junction (4.1%), appendix (6.4%), and omentum (1.7%) [31].

Patients with bowel endometriosis typically experience pain and intestinal symptoms. The pain can be attributed to the intestinal nodules as well as other deep endometriotic nodules, such as those found in the rectovaginal septum, uterosacral ligaments, and parametrium, which are often associated with intestinal lesions. In addition, the location, size, and degree of intestinal lumen stenosis of bowel nodules might result in a range of intestinal symptoms (**Figures 1** and **2**) [32].

Patients with rectosigmoid endometriosis may present with a range of intestinal symptoms, including dyschezia, cyclic bowel alterations, abdominal cramping, a sensation of incomplete evacuation, stool fragmentation, the passage of mucus with stools, and rectal bleeding [33].

The most common complaints among patients included constipation (40%), a feeling of incomplete evacuation (36%), and stool fragmentation (52%). The severity of dyschezia, as measured on a 10-point visual analog scale, averaged 7.1. Patients with deep endometriosis infiltrating the rectum were more likely to experience cyclic defecation pain (67.9%) and cyclic constipation (54.7%), and they also exhibited a significantly longer time to evacuate stools. However, these symptoms were also prevalent in other groups studied, with 38.1% and 33.3% for the superficial endometriosis group, and 42.9% and 26.2% for the group with deep endometriosis sparing the rectum, respectively. Women with rectal endometriosis were also more prone to appetite disorders [34].

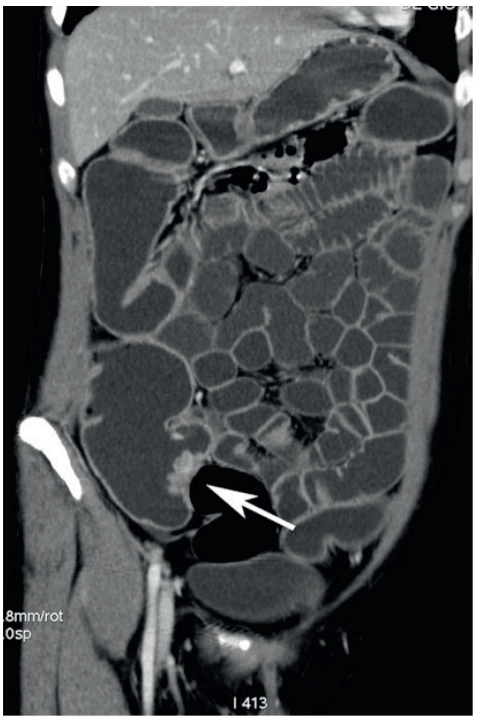

Figure 1.
Cecal endometriotic nodule (arrow) [32].

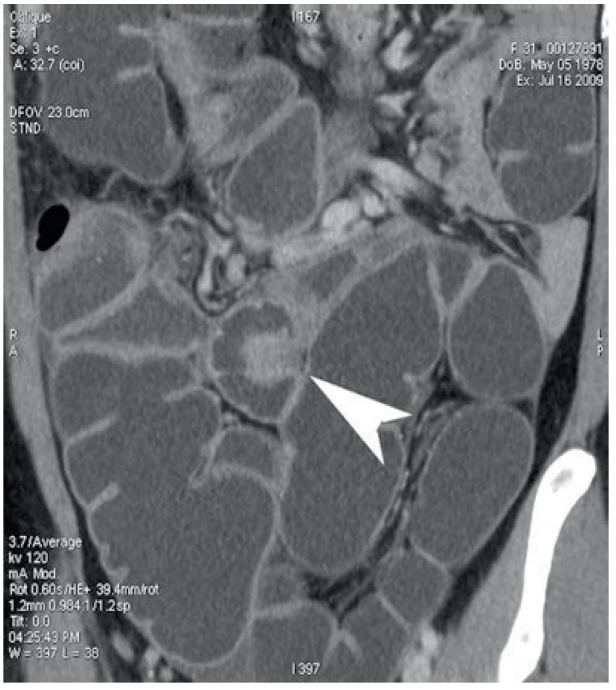

Figure 2.
Ileal endometriotic nodule (arrowhead) [32].

The pain and intestinal symptoms associated with rectosigmoid endometriosis are nonspecific, often leading to diagnostic challenges. Prior to receiving a definitive diagnosis, patients with endometriosis are frequently misdiagnosed with conditions such as irritable bowel syndrome (IBS). An Australian study examined the intestinal symptoms of patients with endometriosis, highlighting these diagnostic complexities [35].

Ileocecal endometriosis may manifest as intestinal obstruction, intussusception, or ileocecal perforation, leading to symptoms such as intestinal cramps, vomiting, abdominal distention, and catamenial subocclusion [36–39]. In some cases, ileocecal endometriosis can cause nonspecific symptoms that resemble those of intestinal malignancies or Crohn's disease. While magnetic resonance imaging and computed tomography may detect an ileocecal mass, they do not always conclusively indicate endometriosis [32].

Double-contrast barium enema is ineffective at detecting small extraluminal lesions. Occasionally, isolated ileocecal endometriosis may be asymptomatic and can present as a submucosal polyp during screening colonoscopy [40]. There have been documented cases of ileocecal perforation related to endometriosis occurring during pregnancy and postpartum. Due to the high vascularization of ectopic endometriotic tissue, ileocolic perforation during pregnancy can lead to significant intraperitoneal hemorrhage [41, 42].

Appendiceal endometriosis occurs in approximately 2.6% of patients undergoing surgery for endometriosis [43]. The diagnosis of appendiceal endometriosis is often made incidentally during surgery for endometriosis-related pain, without preoperative suspicion of its presence on the appendix. However, in some patients, gross alterations of the appendix may necessitate a selective appendectomy [44].

Appendiceal endometriosis can mimic acute appendicitis, presenting with symptoms such as fever, right lower quadrant pain, nausea, and vomiting, and signs such as pain at McBurney's point [45]. There have been reports of appendiceal perforation due to endometriosis [46]. The acute inflammation is often a result of endometriosis causing partial or complete occlusion of the appendiceal lumen. Rarely, endometriosis can result in appendiceal intussusception as well [47].

3. Urogenital symptoms

Urogenital tract endometriosis (UGE) is the second most common form of EE, primarily affecting the bladder (over 85% of cases) and, less frequently, the ureters (10%), kidneys (4%), and urethra (2%) [48].

It typically occurs in women aged 30 to 45 years, with prior pelvic surgery considered a risk factor. Familial aggregation has also been reported [49]. UGE can be asymptomatic in up to 50% of cases, though it can lead to significant complications such as complete loss of kidney function in severe cases of ureteral endometriosis [50].

Bladder endometriosis may present with dysuria, recurrent urinary tract infections, hematuria, irritable bladder symptoms, vesical tenesmus, and incontinence. About 40% of women with bladder endometriosis experience perimenstrual symptoms. Ureteral endometriosis, which affects about 15% of patients, may present with costovertebral angle pain or hematuria [48, 51, 52].

Surgery is advised for bladder endometriosis lesions, and hydronephrosis is a clear sign that surgery is necessary. Re-implantation and ureteral excision are further treatment options, with ureterolysis being successful in 86.7% of cases [51].

4. Thoracic symptoms

Thoracic endometriosis (TE) is a rare form of endometriosis affecting the diaphragm (**Figure 3**) (44.5%), pleura (12.7%), and lungs (4.5%), often involving multiple structures simultaneously. Genital endometriosis coexists in 53–84% of TE cases. TE typically presents around the ages of 30 to 34, about 5 years later than genital endometriosis [18, 53].

Symptoms include menstrual cycle-related, usually right-sided pain in the thoracic, scapular, or shoulder region, and catamenial pneumothorax [18]. Diagnosis involves correlating symptoms with menstruation and diagnostic radiology, with MRI being the preferred modality [54].

Bronchoscopy is useful in cases of hemoptysis to rule out other conditions. Histological confirmation is necessary for a definitive diagnosis. Surgical management often involves a two-stage approach followed by medical treatment, with video-assisted thoracoscopic surgery (VATS) and, in some cases, laparoscopy [54–56].

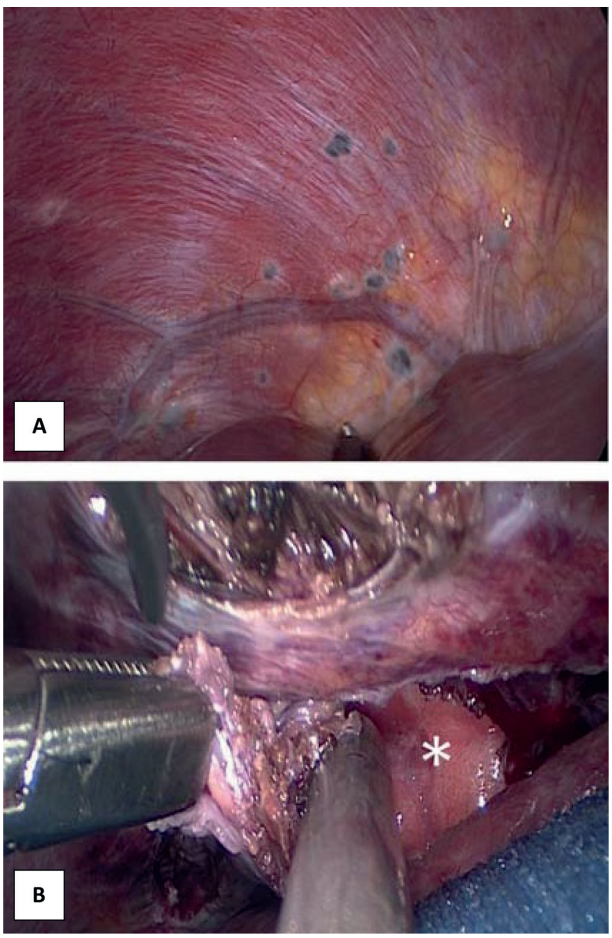

Figure 3.
Multiple diaphragmatic endometriosis (the star in picture B is the lung tissue) [22].

5. Skin symptoms

Cutaneous scar endometriosis can occur following cesarean delivery, hysterectomy, or laparoscopy, presenting as nodules in the epifascial tissue. This pathology affects less than 1% of women with endometriosis and can be easily excised. Overall, endometriosis requires a comprehensive diagnostic and treatment approach, tailored to the individual patient's needs and clinical presentation [22, 57].

6. Neurology symptoms

Nerve involvement, particularly of the sacral plexus, including the sciatic nerve, is a rare manifestation of EE. Approximately 34% of patients exhibit nerve involvement without peritoneal lesions. The etiology may involve the development of endometriosis lesions from undifferentiated cells within the nerve [58].

Symptoms include cyclic (perimenstrual) sciatica, and prolonged untreated conditions may lead to constant pain and neurological deficits. MRI is the diagnostic tool of choice, with ultrasonography as an alternative. Successful drug treatments are rare, and surgical excision of parametrial and peritoneal lesions significantly improves quality of life and pain symptoms [58, 59].

7. Conclusions

This comprehensive review of endometriosis symptoms highlights the multifaceted nature of the disease, which presents with a wide range of symptoms affecting various systems including gastrointestinal, urogenital, thoracic, cutaneous, and neurological. Despite its prevalence, endometriosis remains underdiagnosed and undertreated, with significant delays in diagnosis that can exacerbate patient suffering and complicate treatment.

Key findings from this review include the recognition of bowel endometriosis as a significant source of gastrointestinal symptoms, often misdiagnosed as irritable bowel syndrome (IBS). Similarly, urogenital and thoracic endometriosis present with symptoms that are frequently mistaken for other conditions, further complicating timely diagnosis. The review also emphasizes the importance of considering less common manifestations of the disease, such as nerve involvement and cutaneous scar endometriosis, which, though rare, can significantly impact the quality of life.

The challenges in diagnosing endometriosis underscore the need for greater awareness among healthcare providers and the development of more accurate and less invasive diagnostic tools. Additionally, given the chronic nature of endometriosis, long-term management strategies that integrate both medical and surgical approaches are essential.

Future research should focus on improving diagnostic methodologies, including the development of non-invasive tests, and exploring the pathophysiological mechanisms underlying the diverse presentations of the disease. Furthermore, clinical practice would benefit from a multidisciplinary approach to treatment, tailored to the individual symptoms and needs of patients, to optimize outcomes and improve the quality of life for those affected by endometriosis.

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Conflict of interest

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