



Endometriosis: imaging spectrum of unusual locations and atypical presentations

Alba Salgado-Parente^{1,2} · Elena Canales Lachén¹ · Vanessa Murad³ · Silvia Elizabeth Giménez⁴ · Carmen Sebastià Cerqueda⁵ · Blanca Paño Brufau⁵ · Isabel Fonseca Buelga¹ · Ana Villanueva Campos⁶

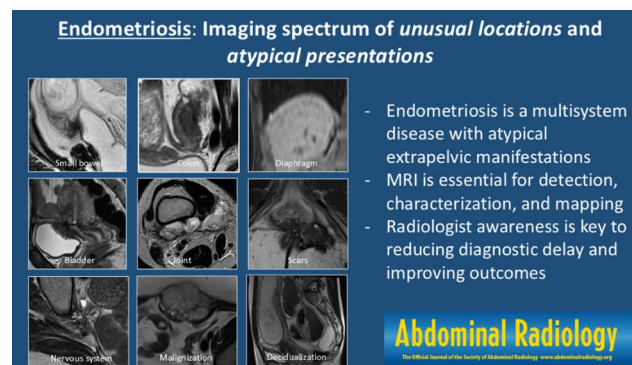
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Abstract

Endometriosis is a chronic, estrogen-dependent inflammatory disease defined by the presence of endometrial-like tissue outside the uterus, affecting approximately 10% of women of reproductive age as well as a notable proportion of adolescents and postmenopausal patients. Although traditionally considered a pelvic disorder, it is increasingly recognized as a multisystem condition with a broad spectrum of extrapelvic locations and atypical clinical and imaging presentations, which contribute to significant diagnostic delay. This review provides a comprehensive overview of the imaging spectrum of endometriosis beyond the pelvis, emphasizing the central role of magnetic resonance imaging (MRI) in lesion detection, characterization, and preoperative mapping. Particular attention is given to the diverse manifestations of extrapelvic disease, including gastrointestinal, genitourinary, thoracic, musculoskeletal, and neural involvement, each of which may present with nonspecific or misleading findings and mimic other pathological entities. Additionally, uncommon presentations such as infection, malignant transformation, and decidualization, as well as variations across different patient populations, further increase diagnostic complexity. Recognizing these patterns and understanding their imaging features are essential to avoid misdiagnosis and delays in care. A systematic approach combining clinical suspicion, tailored imaging protocols, and structured reporting is key to improving diagnostic accuracy and guiding appropriate management. Greater awareness of the multisystem nature of endometriosis is crucial to optimize patient outcomes and reduce the burden of this frequently underrecognized disease.

Graphical abstract



Keywords Endometriosis · Magnetic resonance imaging · Diagnostic imaging · Pelvic pain · Extrapelvic endometriosis · Deep infiltrating endometriosis

Abbreviations

ADC	Apparent diffusion coefficient
CT	Computed tomography
DIE	Deep infiltrating endometriosis
DWI	Diffusion-weighted imaging
EAOC	Endometriosis-associated ovarian cancer
ESUR	European Society of Urogenital Radiology
FS	Fat-suppressed
GU	Genitourinary
IBD	Inflammatory bowel disease
IVIM	intravoxel incoherent motion
MRI	Magnetic resonance imaging
O-RADS MRI	Ovarian-Adnexal Reporting and Data System magnetic resonance imaging
OHVIRA	Obstructed Hemivagina and Ipsilateral Renal Anomaly
SAR	Society of Abdominal Radiology
SE	Superficial peritoneal endometriosis
T1WI	T1-weighted images
T2WI	T2-weighted images
TES	Thoracic endometriosis syndrome
TVUS	Transvaginal ultrasound
US	Ultrasound

Background: think outside the box

Endometriosis is a chronic, multisystemic, estrogen-dependent inflammatory disease defined by endometrial-like tissue (glands and stroma) outside the uterus [1]. Increasingly recognized as a systemic disorder, it is associated with chronic inflammation, immune dysregulation, and multiple comorbidities [2, 3]. To date, its management remains challenging, with no definitive cure currently available for all patients [2].

Approximately 10% of reproductive-age women worldwide have endometriosis [3]. Notably, it is also prevalent in adolescents, with laparoscopic confirmation in 62% of girls presenting with dysmenorrhea or pelvic pain [4]. In postmenopausal women, the prevalence of active/symptomatic disease is 2–5%, primarily in those receiving hormone replacement therapy [5].

Despite its high prevalence, diagnosis is usually delayed, ranging from 4 to 11 years, due to multiple reasons such as nonspecific symptoms, normalization of menstrual pain, and limited awareness among both patients and healthcare providers [6]. Furthermore, prolonged use of oral contraceptives can mask endometriosis symptoms and lead to diagnosis at a more severe stage of disease and later in life [7].

A detailed patient history remains a cornerstone of endometriosis diagnosis. Symptoms may vary depending on the

location of the endometriotic implant. The condition should be suspected in women of reproductive age presenting with chronic pelvic pain, severe dysmenorrhea, deep dyspareunia, cyclic urinary or bowel symptoms, or unexplained infertility [8].

Classification, distribution, and MRI hallmarks

Endometriosis may present as pelvic and/or extrapelvic disease. Pelvic endometriosis is categorized into 3 subtypes: superficial peritoneal endometriosis (SE), endometriomas and deep infiltrating endometriosis (DIE) (Table 1) [9]. Subtypes may occur alone or in combination and are important to distinguish because they may affect the diagnostic and treatment approach [2]. A normal MRI examination does not exclude endometriosis, particularly in SE or very small endometriotic lesions.

Extrapelvic endometriosis is rare and underrecognized but reported in virtually every organ system. The gastrointestinal and urinary tracts are the most commonly affected extragenital sites, followed by the thoracic cavity and abdominal wall or surgical scar implants. Less frequently, endometriotic deposits have been described in neural tissue and musculoskeletal structures. Its pathogenesis remains multifactorial and incompletely understood. Proposed mechanisms include transperitoneal dissemination following retrograde menstruation, lymphatic and hematogenous spread to distant sites, coelomic metaplasia, and direct iatrogenic implantation during surgical procedures [2, 9, 10] (Fig. 1). Regardless of location, the unifying MRI hallmarks are T1-weighted images (T1WI) hyperintensity reflecting hemorrhagic content, often confirmed on fat-suppressed (FS) sequences, combined with variable T2-weighted images (T2WI) signal depending on the ratio of chronic fibrosis (T2WI hypointensity) to active hemorrhagic or glandular components (T2WI hyperintensity) [9] (Fig. 1). This signal behavior, together with cyclical symptom correlation, multiplicity of lesions, and the absence of features suggesting systemic malignancy constitutes the diagnostic framework that distinguishes endometriosis from its principal site-specific mimics, which are discussed in detail in the corresponding sections below.

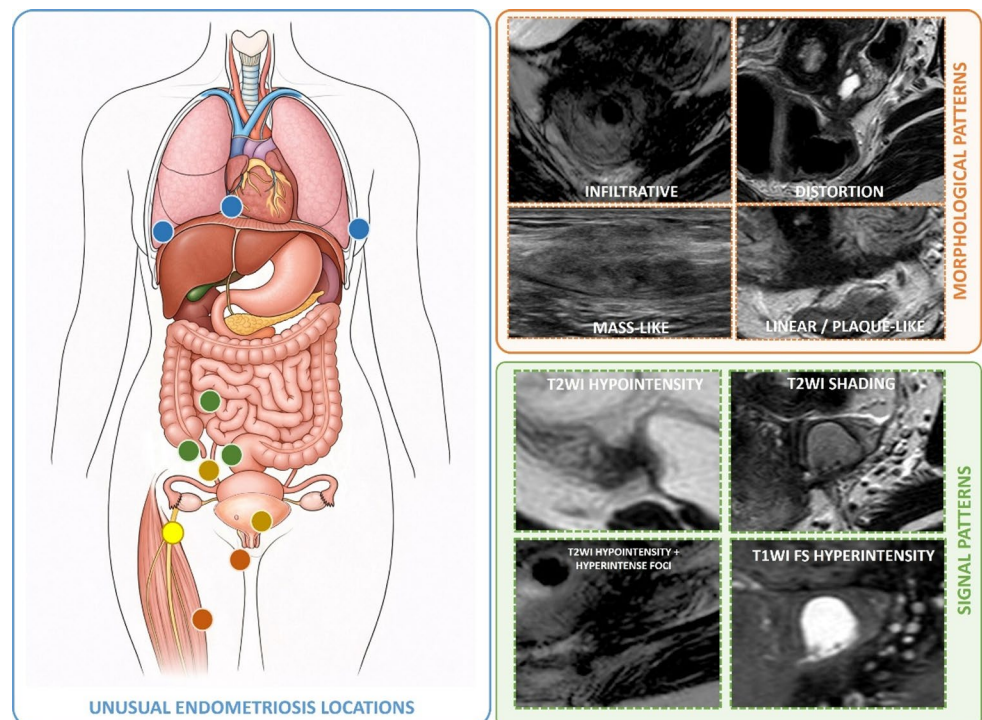
Imaging techniques

MRI is the modality of choice for the comprehensive evaluation of endometriosis, offering excellent soft-tissue contrast, multiplanar assessment, and accurate preoperative mapping. Table 2 summarizes the recommended MRI protocol based on guidelines from the European Society of Urogenital Radiology (ESUR), the Society of Abdominal Radiology (SAR) and current evidence, including patient

Table 1 Summary of the main morphological subtypes of pelvic endometriosis

Morphologic subtypes	Definition	Location	MRI features
Superficial/peritoneal endometriosis (SE)	Tiny foci on the peritoneal surface or visceral serosa Variable on laparoscopy: blue, brown, red, white	Ovarian fossa, pouch of Douglas, parietal peritoneum, uterine serosa	Difficult to identify at MRI unless they are hemorrhagic or cystic If visible: small T1WI FS/T2WI-hyperintense linear or nodular foci
Endometriomas	Cysts lined by endometrial epithelium containing blood-stained brown fluid Sometimes referred to as a chocolate cyst	Ovarian Para-ovarian	Unilocular or multilocular T1WI hyperintense: higher signal intensity than fatty tissue on non FS T2WI hypointense: T2WI shading sign Other: fluid-fluid level, T2WI-dark rim, T2WI-dark spot sign (clots may mimic solid tissue; may restrict DWI but should not enhance)
Deep infiltrating endometriosis (DIE)	Lesions penetrate the peritoneal surface. They can show invasive patterns when infiltrating the muscularis propria of pelvic visceral organs	Most frequently located in the torus, uterosacral ligaments and rectum. First sites to check	Direct signs: 1. Implants: nodular, spiculated, plaque-like 2. Adhesions: linear or reticular Predominantly T2WI hypointense (compared to muscle) due to their fibrotic nature Variable foci of T1WI and T2WI hyperintensity if hemorrhagic or cystic components are present Indirect signs: 1. Retroverted/retroflexed uterus 2. Elevation of the posterior vaginal fornix 3. Kissing ovaries 4. Tethering of the bowel loops 5. Loss of fat planes

Fig. 1 Schematic anatomical illustration of the female body showing unusual sites of endometriosis. Thoracic involvement includes the pleura, pericardium, and diaphragm (blue); GI involvement includes the colon, small bowel, and appendix (green); GU involvement includes the urinary bladder and ureter (dark yellow); musculo-skeletal involvement includes episiotomy scars, cesarean section scars, and muscle (orange); and nervous system involvement includes the peripheral nerves (yellow). The panels on the right illustrate the main morphological patterns and MRI signal characteristics of endometriotic implants



preparation, core sequences, and protocol adaptations for specific disease locations [9, 11–16].

Alongside MRI, transvaginal ultrasound (TVUS) plays a central role in the diagnosis and preoperative assessment

of endometriosis [15]. TVUS is the first-line technique for pelvic disease, with excellent diagnostic performance for ovarian endometriomas and pelvic DIE [2, 17, 18]. However, its limited field of view restricts assessment of lesions

Table 2 Summary of current recommendations for MRI evaluation of endometriosis, including patient preparation, essential imaging sequences, advanced techniques, and protocol modifications for specific disease locations

	RECOMMENDATION	RATIONALE/KEY DETAILS
Patient preparation		
Fasting	3–6 h prior to examination	Reduces bowel content and peristaltic artifacts
Antiperistaltic agent	Glucagon 1 mg IV/IM or hyoscine butylbromide (Buscopan®) 20–40 mg IV	Reduces bowel motion artifacts; hyoscine preferred in Europe, glucagon in the US (FDA-approved)
Bladder filling	Moderate (patient voids 1–2 h before scan)	Displaces small bowel loops cranially; reduces uterine anteversion; improves delineation of anterior compartment
Rectal gel	Optional: ultrasonographic gel via catheter-tip syringe	Distends rectal walls; improves evaluation of rectovaginal septum and bowel wall infiltration; SAR considers optional, ESUR does not mandate
Cleansing enema	Bowel preparation with low residue diet and enema is preferred Note: bisacodyl enemas may cause rectal wall edema that can mimic pathology	Reduces fecal content and rectal distension artifacts; improves rectosigmoid wall delineation.
Vaginal gel	Optional: ultrasonographic gel	Improves sensitivity for posterior vaginal fornix and rectovaginal septum nodules; recommended by SAR, optional per ESUR
Menstrual cycle timing	First half preferred but not mandatory	May increase sensitivity for small hemorrhagic foci
Core sequences		
T2WI TSE multiplanar	Sagittal, axial oblique, coronal; 3–5 mm slices	Backbone of the protocol; depicts fibrotic DIE, uterosacral ligaments, vaginal fornix and bowel wall layers
T1WI without FS	Axial; 5–6 mm	Detects hemorrhagic content as high signal; essential to differentiate blood from fat
T1WI with FS	Axial±sagittal; 3–5 mm	Most sensitive sequence for hemorrhagic implants and SE
Advanced/tailored sequences		
Small FOV T2WI	FOV ≤ 20 cm; 2–3 mm slices	Improves assessment of the rectovaginal septum, rectosigmoid wall, and uterosacral ligaments; evaluates depth of bowel invasion
Renal coverage	Coronal T2W or axial extending to renal hila	Screens for hydronephrosis secondary to ureteral involvement
Contrast-enhanced T1W FS+subtraction	Dynamic contrast-enhanced MRI (≥ 4–6 phases over 3–5 min) + subtraction	Recommended when malignancy is suspected; subtraction is essential to identify true enhancement within hemorrhagic content
DWI/ADC	Axial; b = 0 and 800–1000 s/mm ²	Limited specificity; in cases of suspected malignancy, DWI findings should be interpreted in conjunction with conventional morphological sequences
Special indications		
MR neurography	Contrast-enhanced 3D STIR T2W SPACE (or equivalent). Diffusion tensor imaging with tractography is an emerging technique	Indicated for cyclic sciatica, weakness, or incontinence
MR enterography	Oral negative contrast (PEG, mannitol, or water) 45–60 min pre-scan to distend small bowel, combined with T2WI and T1WI FS±gadolinium	Suspicion of multicentric bowel disease or supra-rectosigmoid involvement
Thoracic/diaphragmatic coverage	Sagittal and coronal fat-suppressed T1WI and T2WI through diaphragm and lower thorax	T1WI FS for identifying blood products; T2WI FS increase sensitivity for cystic/vesicular tissue. Breath-hold and respiratory-triggered or navigated techniques for mitigating motion artifacts

proximal to the rectosigmoid junction and most extrapelvic sites, for which MRI is generally preferred [17]. High-frequency linear abdominal ultrasound (US) is the initial imaging technique for abdominal wall and scar endometriosis [19]. US may also be used to guide biopsy for histological confirmation and percutaneous cryoablation of extraperitoneal lesions [20].

CT is the first-line imaging modality for thoracic endometriosis syndrome (TES), the most frequent extraabdominal form, where it helps exclude other pulmonary diseases [21]. CT is often the initial study in emergency presentations as bowel obstruction, hydronephrosis or sepsis of suspected pelvic origin [9, 11, 22].

Endometriosis: unusual locations

Gastrointestinal (GI) endometriosis

GI endometriosis is the most common form of extragenital endometriosis, affecting 3–37% of all women with endometriosis [18, 23]. The rectosigmoid colon is by far the most frequently involved segment ($\approx 90\%$) followed by the distal small bowel (ileum, $\approx 7\%$), cecum ($\approx 3.6\%$) and appendix ($\approx 3\%$) [23]. One-third of patients with rectosigmoid disease also show right-sided extrapelvic bowel involvement [18].

According to the American College of Radiology, dynamic TVUS, including assessment of the sliding sign and soft markers such as kissing ovaries and bowel tethering, is the first-line imaging modality for suspected rectosigmoid endometriosis, with diagnostic performance

comparable to MRI. When TVUS findings are negative, equivocal, or incomplete, MRI is recommended for comprehensive disease assessment and preoperative planning, offering accurate evaluation of lesion extent and depth of bowel wall infiltration together with a wider field of view [11, 17, 18]. For suspected small bowel endometriosis, particularly in cases of multifocal or multicentric intestinal involvement, MR enterography should be considered as an adjunct technique [9]. Optimized bowel evaluation benefits from dedicated patient preparation, included in Table 2 [12].

Deep infiltrating rectosigmoid endometriosis may show a “mushroom cap” appearance, a T2WI-hypointense crescentic-shape nodule caused by muscularis propria hypertrophy and retraction of the serosa. The stalk of the mushroom corresponds to the commonly associated retrocervical or posterior uterine DIE, which may demonstrate intralesional T1WI hyperintense or T2WI hyperintense hemorrhage or glandular foci (Fig. 2A) [9, 18]. Small bowel lesions present as multifocal T2WI-hypointense fan-shaped lesions on ileal loops and may exhibit a characteristic “fortune-cookie sign” (Fig. 2B) [9].

MRI reporting should specify the involved bowel segment, including the distance from the ileocecal valve and the presence of appendiceal involvement when the small bowel is affected. In colonic disease, reports should additionally detail lesion location, distance to the anal verge, longitudinal extent, mural thickness, degree of circumferential involvement, and depth of infiltration, with clear differentiation between serosal implants and true bowel wall invasion (muscularis propria or submucosa), given its implications for surgical planning [11, 18]. Differential diagnoses mainly

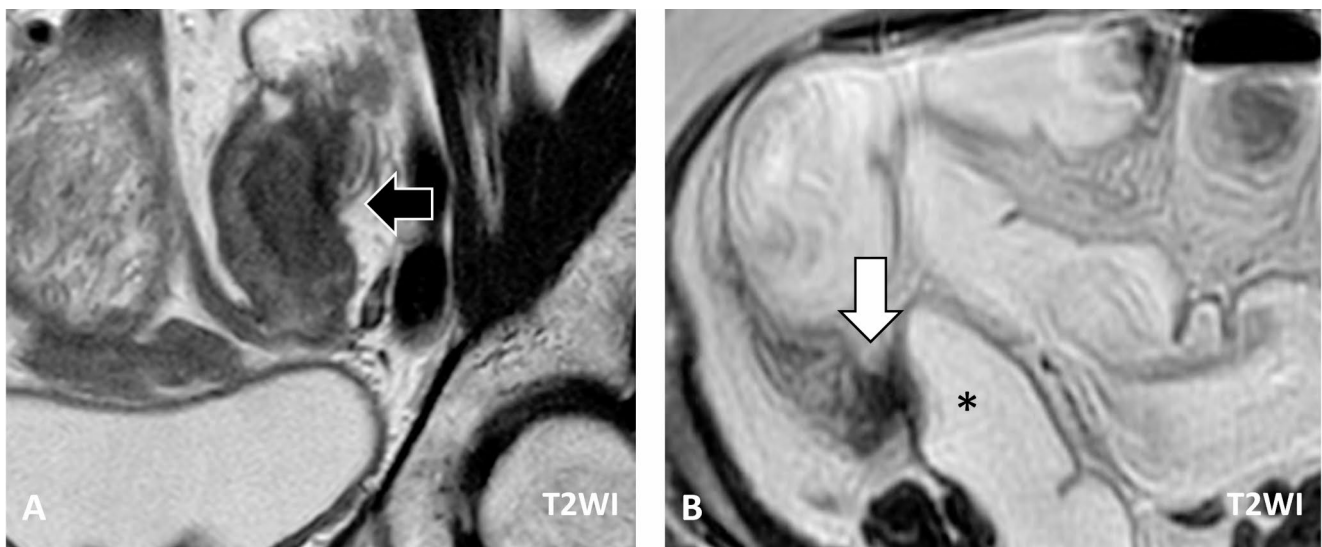


Fig. 2 **A** 40-year-old woman with abdominal pain. Pelvic MRI: axial T2WI demonstrates a low T2WI mural thickening of the sigmoid colon with a characteristic “mushroom cap” appearance, consistent with bowel endometriosis **B** 38-year-old woman with intermittent severe

abdominal pain. Pelvic MRI: T2WI demonstrates focal, circumferential, stenotic wall thickening in the terminal ileum (arrows), markedly hypointense on T2WI, consistent with ileal endometriosis. These findings result in retrograde bowel dilatation (asterisk)

Table 3 Comparative features of bowel involvement by endometriosis, carcinoma, and IBD

Feature	Endometriosis	Carcinoma	IBD
Origin	Serosal or subserosal	Mucosal (epithelial)	Mucosal and submucosal
Morphology	Crescent-shaped nodule extending from the outside inward	Irregular circumferential wall thickening	Segmental or diffuse mural thickening with mucosal enhancement
T2WI signal	Hypointense	Intermediate	Variable. Hyperintense in the acute phase (edema), hypointensity in chronic fibrosis
MRI pattern	Focal lesion, sometimes with “mushroom cap sign”	Infiltrating, stenotic lesion, may invade perirectal fat	Segmental symmetric transmural thickening. Skip lesions
Rectal bleeding	Uncommon, only if mucosa is involved	Common	If rectal involvement: common during active inflammation
Associated findings	Adnexal endometriosis and other sites of DIE	Lymphadenopathy, metastases	Mesenteric fat stranding, “comb sign”, areas of bowel dilatation and stenosis, possible fistulas or abscesses

include colorectal carcinoma and inflammatory bowel disease (IBD) (Table 3).

Genitourinary (GU) endometriosis

GU involvement in endometriosis is uncommon but clinically relevant due to the risk of silent obstruction and long-term renal impairment. This has been reported in approximately 1–3% of all patients with endometriosis, with higher prevalence (up to 50%) in patients with DIE [24]. The bladder is the most frequently affected organ (70–85%), followed by the ureter (9–23%). Renal and urethral involvement are rare, with only isolated case reports described [9, 24, 25]. Patients may present with nonspecific pelvic pain, dysuria (20–70%), urinary frequency (up to 40%), and cyclical hematuria (10–20%) [9, 25].

A moderately filled bladder is recommended by ESUR and SAR because it not only prevents effacement of the bladder walls but also increases the angle of the anteverted uterus allowing for better visualization of the anterior compartment [12, 26]. Bladder endometriosis is defined by detrusor muscle infiltration with partial or full-thickness involvement, which translates on MRI as nodules



Fig. 3 38-year-old woman with chronic post-pregnancy abdominal pain and a history of endometriosis. Sagittal T2WI MRI shows a spiculated, mass-like lesion at the cesarean section scar extending to the vesicouterine space and transmurally invading the urinary bladder dome (white arrows), consistent with cesarean section scar and vesical endometriosis

obliterating the hypointense wall signal in T2WI, with possible T1WI-hyperintense foci (Fig. 3). Superficial vesical implants should not be classified as bladder disease. Reporting should describe the lesion location, size, and distance to the ureteral orifices, as these features directly influence management [9, 24, 25].

Pelvic ureteral involvement typically manifests as partial or complete circumferential encasement of the ureter by T2WI-hypointense soft tissue, frequently extending contiguously from adjacent deep infiltrating lesions of the posterior compartment. As with bladder involvement, ureteral endometriosis seldom occurs in isolation; therefore, the presence of ovarian endometriomas and DIE should prompt careful evaluation for concomitant ureteral involvement [26]. When hydronephrosis is identified or only partially visualized on pelvic MRI, dedicated upper urinary tract imaging is recommended [9, 16].

Thoracic endometriosis

TES is the most frequent extrapelvic manifestation of endometriosis and coexists with pelvic disease in approximately 51% of cases, supporting its role as a marker of advanced disease [9, 21]. The most widely accepted pathophysiological mechanism is the transdiaphragmatic migration of endometrial tissue through diaphragmatic defects, driven by peritoneal fluid dynamics — specifically, the clockwise peritoneal circulation that carries endometrial cells along the right paracolic gutter, where the falciform ligament traps them against the right hemidiaphragm — which explains the predominant right-sided involvement (>90%) [21]. Lesions are most commonly identified on the diaphragm (79%),

followed by the pleura (14%) and, less frequently, the lung parenchyma (4%) [9, 21].

While diagnostic laparoscopy is still considered the gold standard for diagnosing TES, radiological imaging has an emerging role as a non-invasive tool. Chest radiography and CT are typically used in the acute setting to evaluate pneumothorax or hemothorax and to exclude alternative thoracic pathology. CT may also demonstrate small pulmonary nodules, consolidations, or cavitary lesions with surrounding ground-glass opacities, which can fluctuate with the menstrual cycle and occasionally mimic malignancy [13]. Nevertheless, MRI is the preferred modality for lesion characterization and preoperative mapping, with coronal and sagittal planes being particularly useful. Tiny T1WI FS hyperintense foci should be actively sought, as they may represent small hemorrhagic implants that, due to their location, can be easily overlooked [9, 12] (Fig. 4). Thoracic endometriotic implants, unlike deep pelvic endometriosis, more often manifest as T2WI-hyperintense vesicles on

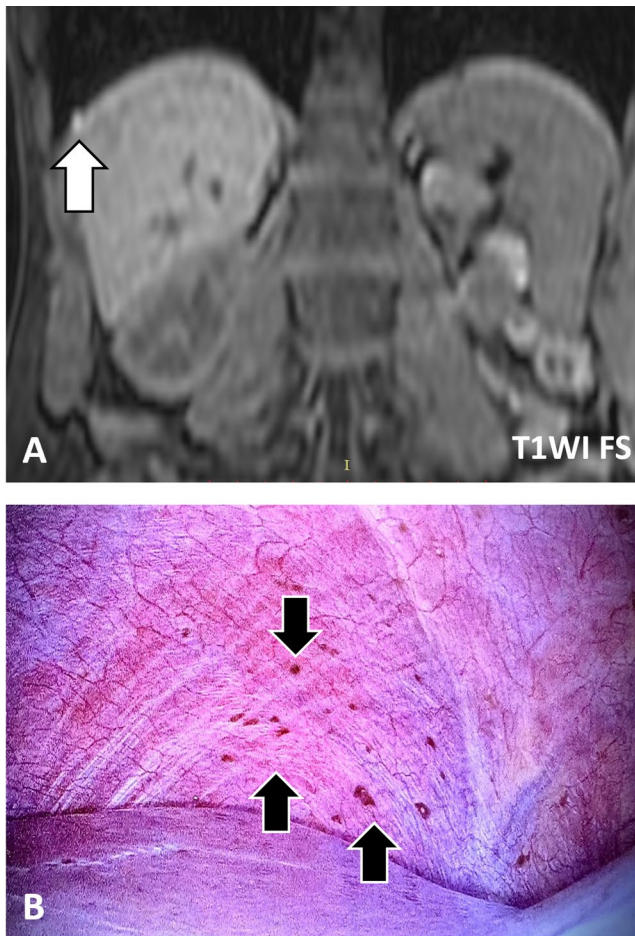


Fig. 4 18-year-old woman with right upper quadrant pain. **A** MRI: axial T1WI FS image demonstrates a single right subdiaphragmatic endometriotic implant. **B** Laparoscopy reveals multiple reddish and bluish subphrenic lesions (black arrows) corresponding to hemorrhagic endometriotic implants at different stages of evolution

diaphragmatic and pleural surfaces, with or without hemorrhagic content [27]. These imaging findings, together with the absence of associated lymphadenopathy, metastatic disease, or an identifiable primary malignancy, may help differentiate pleural endometriosis from pleural metastases or malignant pleural mesothelioma [13].

Musculoskeletal and soft tissue endometriosis

Musculoskeletal and soft-tissue endometriosis is a rare but increasingly recognized extrapelvic manifestation, often resulting in diagnostic delay due to nonspecific symptoms and frequent mimicry of neoplastic or inflammatory processes. Musculoskeletal involvement most commonly affects the abdominal wall ($\approx 50\%$), pelvic floor, and paraspinal muscles [28]. History of prior pelvic or obstetric surgery is present in over 60% of cases, supporting iatrogenic implantation as a key pathogenic mechanism [28, 29].

Abdominal wall scar endometriosis, particularly following cesarean section, is the most frequent manifestation of soft-tissue endometriosis. Prevalence ranges from 0.03% to 1%, typically presenting as a painful, palpable subcutaneous or intramuscular mass with cyclical symptom exacerbation [30]. US usually demonstrates a solid hypoechoic and ill-defined mass. MRI is particularly valuable for defining depth of invasion, including involvement of the rectus sheath or muscle, which directly influences surgical planning. The main imaging differential is desmoid tumor (aggressive fibromatosis), which can be distinguished by the presence of T2WI dark strands, a fascial tail sign, higher ADC values, and the absence of hemorrhagic T1WI-hyperintense foci [31].

Episiotomy scar endometriosis is rare ($\approx 0.01\%$), but clinically significant due to its proximity to the anal sphincter complex [32]. It typically presents as a firm, tender perineal nodule that may be occult on inspection. MRI helps accurately define lesion extent and exclude sphincter or rectal involvement (Fig. 5).

Beyond surgical scars, endometriosis can rarely involve the umbilicus (Villar's nodule), deep musculature, as well as synovial tissue (Figs. 6 and 7). The differential diagnosis in these locations is often challenging, and histopathological confirmation is frequently required to establish a definitive diagnosis [33]. Cyclical symptoms and hemorrhagic MRI features help prevent misdiagnosis and unnecessary intervention [34].

Nervous system: endometriosis Involving peripheral nerves

Pelvic nerves may be entrapped or infiltrated by endometriotic lesions, either in isolation or through direct extension

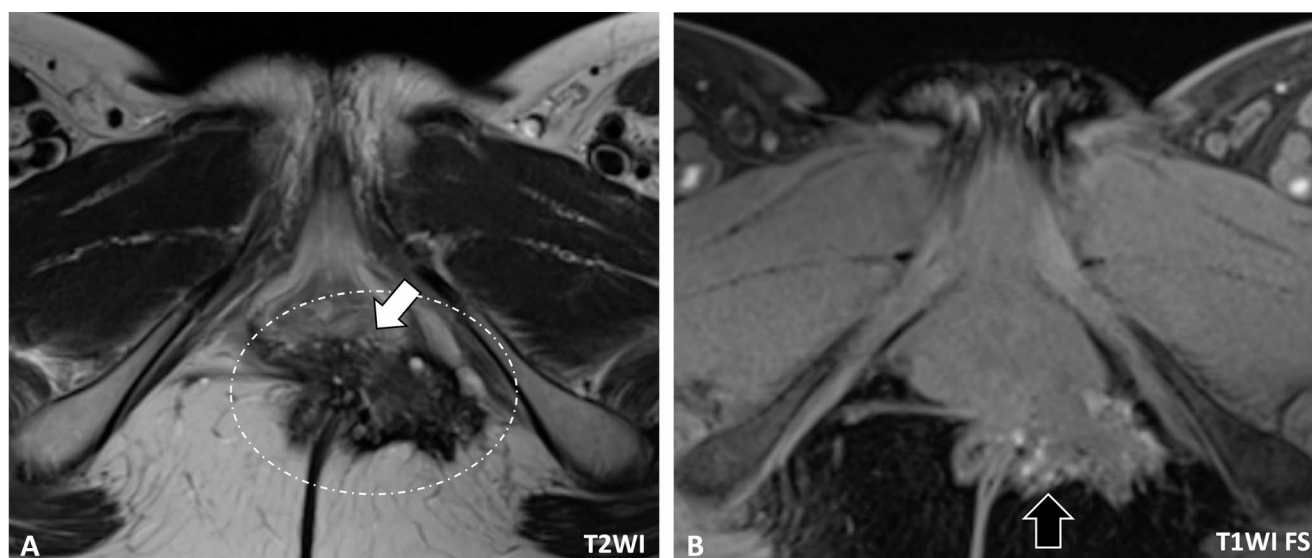


Fig. 5 44-year-old woman presenting with perianal pain and intermittent bleeding. Pelvic MRI: axial T2WI (**A**) and pre-contrast axial T1WI FS (**B**). Images demonstrate a spiculated, predominantly T2WI-hypointense lesion within the left ischioanal fossa, at the site of a prior

episiotomy scar. The lesion shows broad-based contact with the left internal anal sphincter (arrow in **A**) and contains small hemorrhagic T1WI-hyperintense foci (black arrow in **B**), consistent with endometriotic implant

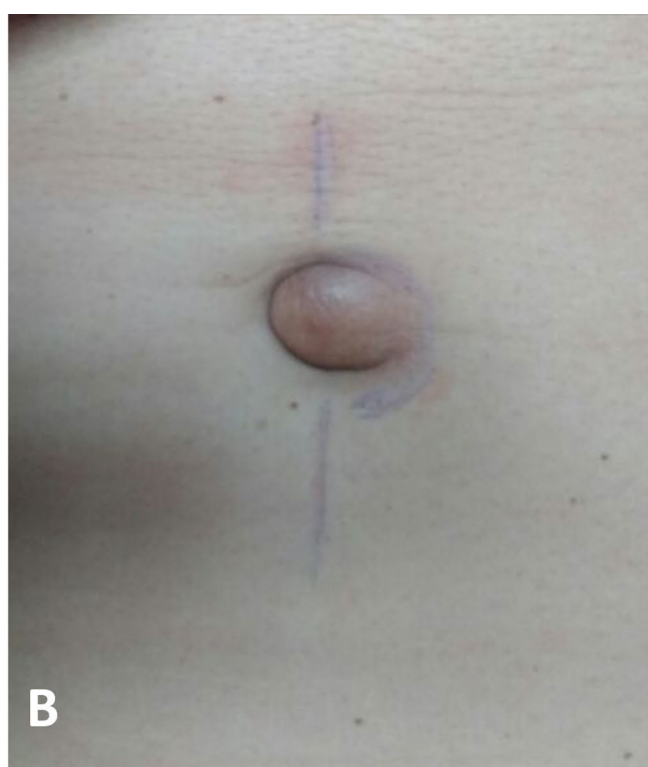
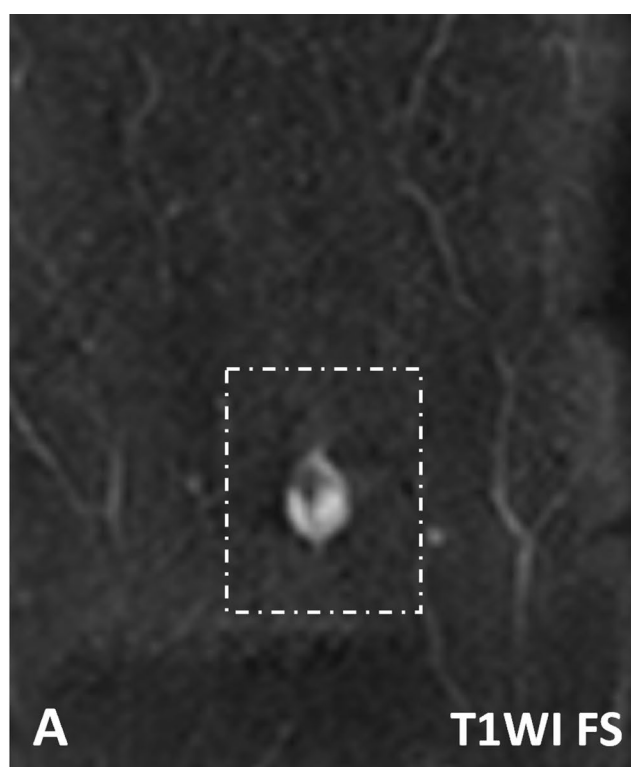


Fig. 6 46-year-old woman presenting with a painful umbilical mass that increases in size during menstruation. **A** MRI: coronal T1WI FS demonstrates a well-defined nodular lesion in the umbilical region,

showing heterogeneous hyperintensity on T1WI FS, consistent with umbilical endometriosis (Villar's nodule). **B** Physical examination image shows the corresponding lesion

Fig. 7 16-year-old girl with intermittent knee pain. Knee MRI: Axial T2WI (A) and T1WI FS (B) demonstrate an irregular mass adjacent to the posterior capsule and synovium showing T2WI hyperintensity with areas of T2WI shading, fluid-fluid level (white arrow), and mild T1WI hyperintensity. Associated locoregional inflammatory changes are present (black arrow). Histopathology confirmed an endometriotic implant

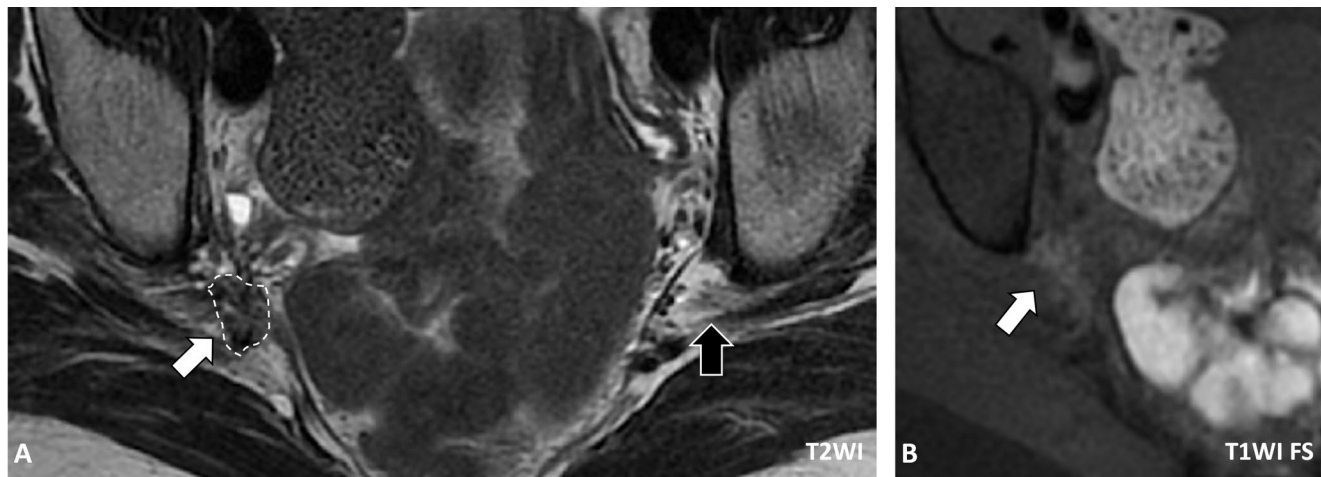
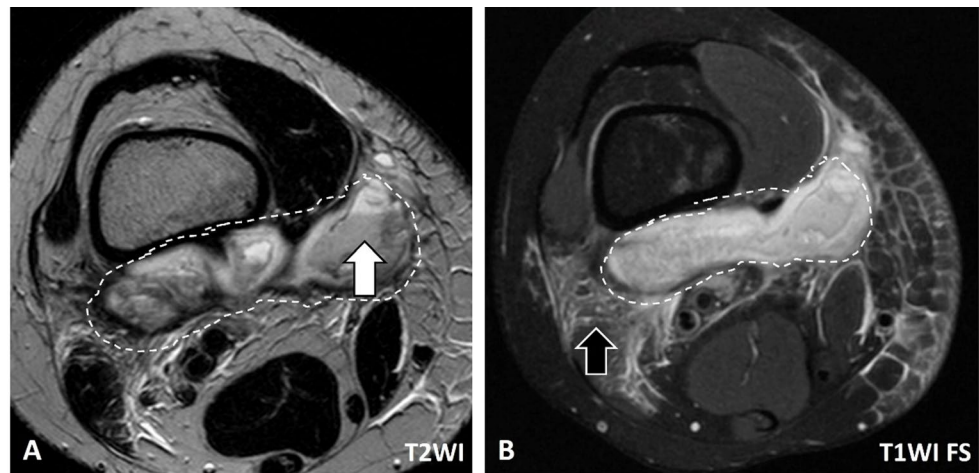


Fig. 8 40-year-old woman with right-sided sciatica, presenting as pain radiating from the gluteal region to the posterior thigh. Pelvic MRI: axial T2WI (A), axial T1WI FS (B). Images demonstrate a spiculated implant at the right sciatic notch, appearing hypointense on T2WI

(arrow, A) and slightly hyperintense on T1WI (arrow, B). The lesion distorts the normal fascicular architecture of the sciatic nerve, with asymmetry compared to the contralateral side (black arrow, A)

of DIE. The true incidence of neural endometriosis remains unknown and is likely underrecognized. Most affected are the inferior hypogastric plexus (57%) (bladder/rectal dysfunction, dysuria, dyschezia), sacral plexus (pelvic organ dysfunction), sciatic nerve (39%) (catamenial sciatica, foot drop), and pudendal nerve (perineal burning worsened by sitting). Involvement of the obturator and femoral nerves is uncommon (1%) [14, 15, 35].

Neural involvement in extrapelvic endometriosis is exceptionally rare and typically occurs through perineural spread along pelvic autonomic nerves or hematogenous/lymphatic dissemination, affecting extremity nerves beyond the sciatic notch, or central nervous system (conus medullaris/cauda equina with only 13 reported cases) [36, 37]. Notably, the absence of additional pelvic endometriotic implants does not exclude neural involvement [14, 38, 39].

T1WI non-FS MRI images are critical for identifying nerves and tracing their expected course, facilitated

by the high signal intensity of the surrounding perineural fat. Three-dimensional MRI neurography, functioning as heavily T2WI, maximizes nerve signal intensity against the background of adjacent structures such as muscles [14].

On MRI, neural endometriosis typically presents as T2WI-hypointense solid nodules or fibrotic thickening along the course of the affected nerve, often associated with nerve enlargement, distortion, or encasement. Small cystic or hemorrhagic foci may occasionally be present [14, 38] (Fig. 8). In chronic stages, fatty muscle atrophy due to denervation may represent an indirect imaging sign [15]. Isolated involvement underscores diagnostic challenges, mimicking nerve sheath tumors or melanin-containing neoplasms, representing a potential diagnostic pitfall (Table 4) [39].

Endometriosis: unusual manifestations

Beyond its typical chronic pelvic presentation, endometriosis may present with a range of uncommon manifestations including infectious complications, malignant transformation, hormonally driven changes such as decidualization, as well as age-specific variations.

Infection

Endometriosis may rarely present with acute symptoms due to complications such as rupture or superinfection, mimicking pelvic inflammatory disease, ovarian torsion or appendicitis, requiring early recognition to prevent sepsis [40, 41].

Infected endometrioma is uncommon (2.2–2.3%) [42] and frequently underdiagnosed in the absence of strong clinical suspicion. Most cases are iatrogenic following aspiration or surgical drainage. Other potential routes include ascending genital tract infection, hematogenous or lymphatic spread, and extension from adjacent bowel inflammation [42, 43]. The endometrioma's iron-rich, hypoxic content creates an ideal bacterial growth medium, explaining their infection susceptibility [41].

Table 4 Imaging features differentiating neural endometriosis from peripheral nerve sheath tumors (schwannoma and neurofibroma)

Feature	Neural endometriosis	Peripheral nerve sheath tumors (Schwannoma/Neurofibroma)
Growth pattern on nerve	Predominantly extraneural/perineural, encasing the nerve; may extend intraneurally	Typically intraneural (eccentric in schwannomas, fusiform in neurofibromas)
MRI signal	T1WI hyperintensity may be present due to hemorrhage; T2WI signal variable depending on fibrosis, hemorrhage, and cystic change	T2WI hyperintense. Neurofibromas may demonstrate a target sign (peripheral hyperintensity and central hypointensity)
MRI/US morphology	Irregular lesion	Well-defined and encapsulated
Enhancement pattern	Variable; fibrotic components enhance, whereas hemorrhagic or cystic areas do not	Avid mainly homogeneous enhancement
Cyclic imaging changes	Present. Nerve enlargement and cystic/hemorrhagic components may fluctuate with the menstrual cycle	Absent

Imaging findings are nonspecific, and differentiation from uncomplicated endometrioma can be challenging [43]. On US, wall thickening, septations, fluid–fluid levels, mural nodularity, and echogenic wall foci may be seen in both infected and non-infected lesions. Peripheral hypervascularity and adjacent inflammatory changes may suggest superinfection. CT is typically reserved for acute settings and may demonstrate a complex cystic mass with thick enhancing walls, internal debris, perilesional fat stranding, and surrounding inflammatory changes. The presence of intral-lesional gas in the absence of prior intervention strongly suggests superinfection [41].

Malignant transformation

Malignant transformation of endometriosis, specifically leading to endometriosis-associated ovarian cancer (EAOC), is a rare but clinically relevant complication (≈ 0.5 –1%) [44]. Epidemiologically, EAOC typically occurs in middle-aged women, about 10–20 years earlier than de novo ovarian cancer [45, 46]. According to classic criteria established by Sampson [47] and Scott [48], diagnosis requires coexistence of carcinoma and endometriosis in the same ovary with histological transition [44, 49]. The most common histological subtypes identified are clear cell carcinoma and endometrioid adenocarcinoma [45, 50, 51]. Risk is higher in DIE and ovarian endometriomas than in SE [50]. Additional risk factors include infertility, postmenopausal status, large endometriomas (> 9 cm), and prolonged estrogen exposure [50, 52].

Imaging findings on MRI are essential for early detection. The most reliable sign of malignancy in endometriomas is the presence of enhancing mural nodules. Subtraction imaging should be routinely performed whenever mural nodules are suspected, as high T1WI signal from hemorrhagic contents may obscure true enhancement [45, 46, 53]. Additional suspicious features include loss of T2WI shading, interval increase in cyst size, thick or nodular enhancing septations, and solid components that show diffusion restriction with low ADC values [46, 53, 54]. However, diffusion restriction should be interpreted with caution, as blood products within endometriomas may also demonstrate restricted diffusion and low ADC values. Dynamic contrast-enhanced MRI is recommended in cases of suspected malignancy. O-RADS MRI scoring provides standardized risk stratification for indeterminate adnexal lesions, helping guide subsequent surveillance and clinical management [9, 55]. More recently, advanced MRI techniques such as intravoxel incoherent motion (IVIM) and T2 mapping have shown promising diagnostic performance for the noninvasive detection of malignant transformation in endometriosis, demonstrating

lower true-diffusion coefficient, microcirculation perfusion fraction, and T2 values in malignant lesions [56].

Extraovarian malignant transformation is rare but can also occur. It predominantly affects the rectovaginal septum, colorectum [57], and post-cesarean section abdominal wall scars (≈ 20 -year latency) [58] (Fig. 9).

Differential diagnosis is essential to avoid unnecessary surgical intervention. In particular, decidualized endometriomas during pregnancy may closely mimic malignancy due to rapid growth and the development of vascular mural projections (Table 5) [45, 53].

Endometriosis decidualization

Decidualization of endometriosis is a hormone-driven phenomenon occurring primarily during pregnancy due to elevated levels of progesterone and estradiol [59, 60]. This process involves the hypertrophy of ectopic endometrial stromal cells, transforming them into large, vascularized decidual tissue [60, 61]. This process is more frequent in ovarian endometriomas than in DIE, with 4–5% of early pregnancies showing ovarian endometriomas and up to 16% undergoing decidualization in the first trimester, typically regressing postpartum [62, 63].

Imaging presents a significant diagnostic challenge, as decidualized lesions often mimic malignancy. On US, they

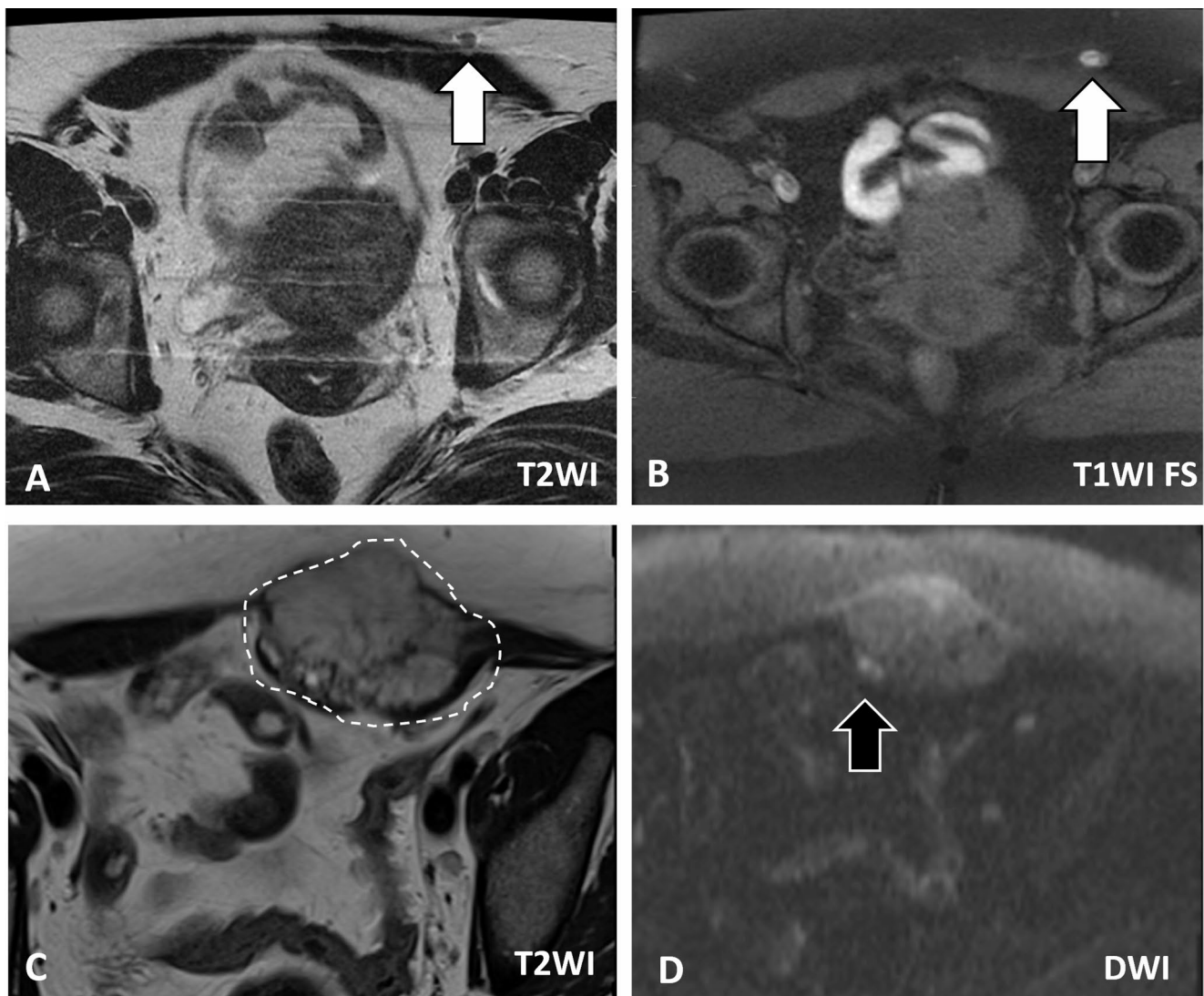


Fig. 9 44-year-old woman with prior Pfannenstiel scar endometriosis resected in 2012. (A, B) Pelvic MRI (2014): axial T2WI (A) and axial T1WI FS (B). Images demonstrate a small endometriotic implant in the anterior abdominal wall. (C, D) Follow-up MRI (2022): axial T2WI (C) and DWI (b value=800 s/mm²) (D). Marked growth of the

lesion, with a heterogeneous appearance, loss of T2WI shading, internal septations, and focal areas of restricted diffusion. Postsurgical histopathology confirmed clear cell carcinoma (malignant extraovarian endometriosis)

Table 5 Key MRI features for differentiating decidualized endometriomas from EAOC

Feature	Decidualized endometrioma	EAOC
Clinical context	Pregnancy or progestosterone stimulation; regresses postpartum	Older age (~50 years), postmenopausal status, prolonged history of endometriosis
Cyst morphology	Stable size; thin regular septations; preserved T2WI shading and T1WI-hyperintense fluid	Progressive enlargement; thick or nodular septations; loss of T2WI shading; decreased T1WI signal due to dilution of hemorrhagic content
Mural nodule morphology	Multiple (mean~11), small, smooth, sessile nodules; low height (~11 mm)	Fewer (mean~4), larger, lobulated or pedunculated nodules; greater height (>11 mm)
Mural nodule signal	T1WI hyperintense; markedly T2WI hyperintense; signal intensity similar to the placenta	T1WI low-to-intermediate signal; variable T2WI signal intensity
DWI/ADC	Intermediate-to-high DWI signal due to T2WI shine-through; high ADC values	True restricted diffusion; low ADC values
Contrast enhancement	Mild enhancement of mural nodules	Avoid enhancement; best seen on subtraction imaging
Advanced MRI techniques (IVIM, T2 mapping)		Lower true-diffusion coefficient, microcirculation perfusion fraction, and shorter T2 relaxation time compared with benign endometriosis
Associated findings	No lymphadenopathy or peritoneal disease	Possible peritoneal carcinomatosis, lymphadenopathy, and elevated CA-125 levels

typically appear as unilocular cysts with “ground-glass” or low-level echogenicity, containing smooth, vascularized papillary projections or mural nodules [62]. MRI features include T1WI hyperintensity and T2WI shading, reflecting hemorrhagic content, while mural nodules demonstrate T2WI hyperintensity (often isointense to the placenta) and higher ADC values compared to malignant lesions [60, 63]. Mural nodules in decidualization are generally smaller (<11 mm), more numerous (mean 11 nodules), and have smooth, non-lobulated margins [64] (Fig. 10).

The primary differential diagnosis is EAOC (Table 5) with other differentials including polypoid endometriosis, colorectal neoplasms in DIE, tubo-ovarian abscess, and ovarian torsion [59, 62, 64]. Current clinical guidelines favor expectant management with serial imaging for asymptomatic patients [62].

Adolescent endometriosis and Müllerian duct anomalies

Endometriosis is the most common cause of secondary dysmenorrhea in adolescents [65]. Younger patients often report more noncyclic pelvic pain and dyspareunia, although the latter may be underestimated in sexually inactive individuals [66, 67].

Imaging can be subtle, and lesions are easily overlooked on TVUS and MRI, making the decision to perform pelvic laparoscopy particularly delicate [68, 69]. Lesions are predominantly white or small hemorrhagic implants, unlike the typical dark brown or black “powder burn” aspect in adults [68, 70]. Often, there is clinical-radiological dissociation, as superficial lesions may cause severe symptoms [67].

SE is more common than DIE in adolescents. Small peritoneal defects, or Allen-Master windows, are frequently associated with these lesions [71]. These ‘windows’ arise from chronic peritoneal remodeling and scarring, forming pockets that may harbor occult endometriotic implants. They are challenging to detect on MRI and may only be seen at laparoscopy [71]. Suspicion should be raised when free fluid is located abnormally in extraperitoneal spaces or when subtle signs of SE (such as peritoneal thickening or hemorrhagic spots along the uterine or ovarian serosa) are present [12, 71].

When present, DIE in adolescents tends to be multifocal, commonly affecting the retrocervical region and uterosacral ligaments, and rarely involving the bowel or bladder [68, 72] (Fig. 11). In most cases, progression is slow.

Obstructive Müllerian anomalies, including imperforate hymen, cervical or vaginal aplasia, unicornuate uterus with a non-communicating functional horn, and OHVIRA (Obstructed Hemivagina and Ipsilateral Renal Anomaly) syndrome, are associated with higher risk of endometriosis [73]; whereas non-obstructive anomalies such as septate or bicornuate uterus do not significantly increase risk compared to the general population [73–76].

Endometriosis in postmenopausal women

Postmenopausal endometriosis is morphologically similar to premenopausal disease but generally less active and extensive, with fewer hemorrhagic components and glands [77]. There are some aspects that may differ from premenopausal women:

Endometrioma: Less common than in premenopausal women, they often show reduced T1WI hyperintensity and T2WI shading, reflecting decreased cyclical bleeding. Importantly, postmenopausal endometriomas have a higher risk of malignant transformation [78]. Any new or enlarging adnexal mass in a postmenopausal woman with a history of

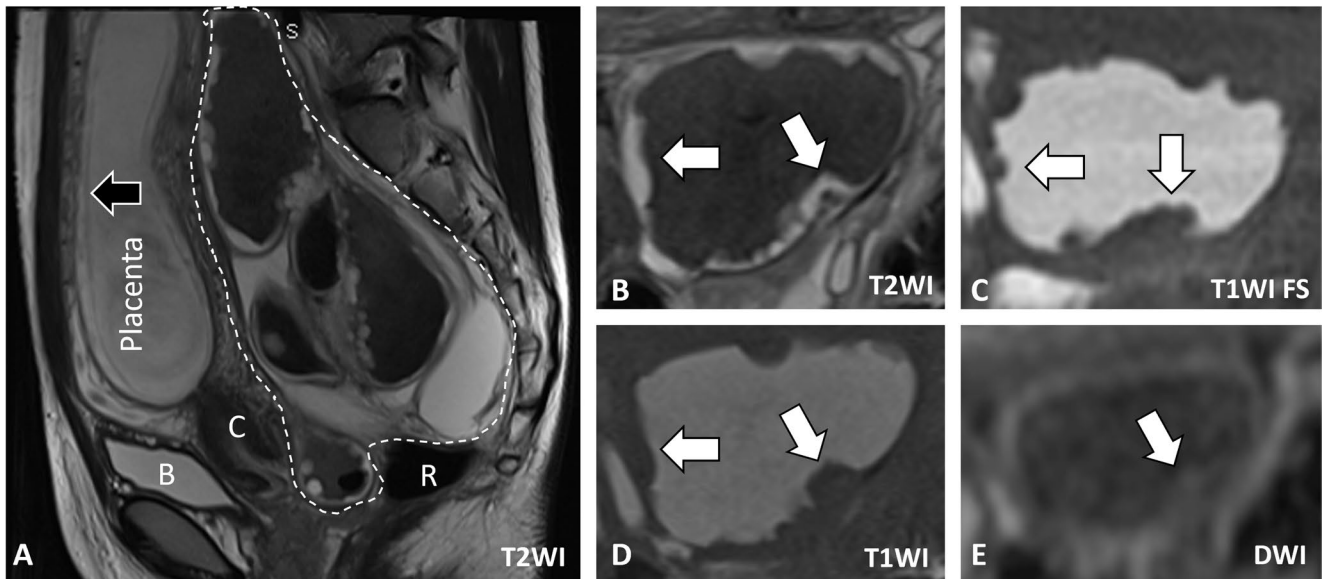


Fig. 10 23-year-old primigravid woman at 16 weeks' gestation. Pelvic MRI: axial T2WI (A), high-resolution T2WI (B), T1WI FS (C), T1WI (D), and DWI (b value = 800 s/mm²) (E). Large retrouterine multicystic mass (dashed line in A). The lesion contains multiple locules with variable signal intensity. Multiple intracystic small broad-based pap-

illary projections (arrows, B-E), show marked T2WI hyperintensity similar to placental signal (black arrow in A), low signal on T1WI and T1WI FS, and no true diffusion restriction (E). Findings are consistent with decidualized endometriosis, which regressed after pregnancy. B = bladder. C = Cervix. R = Rectum

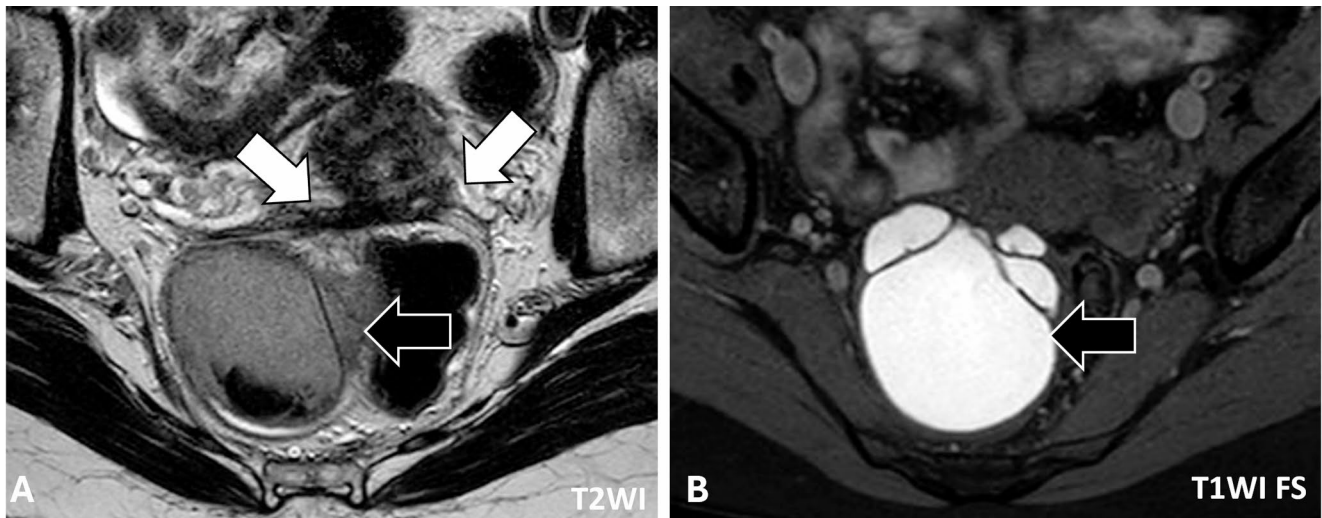


Fig. 11 21-year-old woman with chronic pelvic pain. Pelvic MRI: axial T2WI (A) and axial T1WI FS (B). Images demonstrate findings consistent with DIE, including a fibrotic plaque at the torus uterinus,

thickening of both uterosacral ligaments (white arrows) and abnormally located right adnexa containing an endometrioma (black arrows)

endometriosis warrants careful evaluation for malignancy and the use of IV contrast is recommended [11].

DIE: Lesions are often more fibrotic and less likely to demonstrate hemorrhagic foci, making them less conspicuous on T1WI (Fig. 11). Chronic fibrosis and adhesions predominate [77] and DIE can occur without endometriomas [9].

SE: Small lesions are often undetectable by MRI and even less visible in postmenopausal women due to absence of cyclical hemorrhage [78].

Polypoid endometriosis: A rare, mass-like variant occurring in peri- or postmenopausal women, frequently under hormonal therapy (~50% of cases) [79]. MRI shows signal and enhancement patterns similar to normal endometrium, a T2WI hypointense peripheral rim, and no diffusion restriction [79]. However, no imaging features confidently exclude malignancy and even tissue sampling may not be representative [9].

Conclusion

Endometriosis is a complex disease with manifestations that extend far beyond the pelvis, often posing significant diagnostic challenges. MRI plays a pivotal role in the detection, characterization, and preoperative assessment of both typical and atypical disease sites. Awareness of uncommon locations and presentations is essential to avoid misdiagnosis and optimize patient management. Ultimately, early recognition, standardized imaging assessment, and multidisciplinary collaboration are critical to improving outcomes in this frequently underdiagnosed condition.

Key imaging pearls

- A normal MRI does not exclude endometriosis, particularly superficial peritoneal disease.
- MRI is the modality of choice for mapping unusual and extrapelvic endometriosis, allowing comprehensive assessment of GI, GU, thoracic, musculoskeletal, and neural involvement. TVUS and CT serve as valuable complementary techniques in selected clinical settings.
- Cyclical symptoms combined with hemorrhagic MRI features (T1WI FS-hyperintense foci and T2WI hypointensity or shading) are key clues for recognizing atypical endometriosis and avoiding misdiagnosis as neoplastic or inflammatory disease.

Author contributions All authors contributed substantially to the conception and design of this educational review. ASP led the conceptualization of the manuscript and defined its structure and scope. Literature search, selection of relevant references, and synthesis of evidence were performed by ASP, ECL, VM, SEG, IFB and AVC. Imaging contributions were provided by ASP, ECL, VM, SEG, CSC, BPB, and AVC. Imaging case selection, curation, and figure preparation were carried out by ASP and AVC. ASP, ECL, and AVC performed the final internal review prior to submission to all co-authors. All authors approved the final version of the manuscript.

Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

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Authors and Affiliations

Alba Salgado-Parente^{1,2}  · Elena Canales Lachén¹  · Vanessa Murad³ · Silvia Elizabeth Giménez⁴ · Carmen Sebastià Cerqueda⁵ · Blanca Paño Brufau⁵ · Isabel Fonseca Buelga¹ · Ana Villanueva Campos⁶

✉ Alba Salgado-Parente
albasalgadoparente@gmail.com

Elena Canales Lachén
canaleslachen@gmail.com

Vanessa Murad
Vanessa.Murad@uhn.ca

Silvia Elizabeth Giménez
gimenez.silvia@gmail.com

Carmen Sebastià Cerqueda
MSEBASTI@clinic.cat

Blanca Paño Brufau
BPANO@clinic.cat

Isabel Fonseca Buelga
isaceceda@gmail.com

Ana Villanueva Campos
avillanuevacampos10@gmail.com

¹ Department of Radiology, Hospital Universitario Ramón y Cajal, Madrid, Spain

² Department of Medical Imaging, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, Canada

³ Department of Medical Imaging, University Health Network, University of Toronto, Toronto, Canada

⁴ Department of Radiology, Hospital Británico de Buenos Aires, Buenos Aires, Argentina

⁵ Department of Radiology, Hospital Clínic de Barcelona, Barcelona, Spain

⁶ Department of Radiology, HT Medica San Rafael University Hospital, Madrid, Spain