



Understanding malignant transformation of endometriosis: imaging features with pathologic correlation

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Abstract

Purpose Transformation of benign endometriosis to endometriosis-associated ovarian carcinoma (EAOC) is rare; however, women with endometriosis are four times more likely to develop EAOC which can present 20 years earlier than de novo ovarian cancer. Presenting symptoms are often vague and the radiologist's role in recognizing EAOC is critical for early detection and treatment. Histopathologic evaluation remains the mainstay for definitive diagnosis.

Methods Using a case-based approach, this article will review the sonographic, CT, and MRI features of EAOC with an emphasis on MRI. Histopathologic correlation of benign and malignant endometriosis will be reviewed.

Results Multiple factors contribute to the malignant transformation of endometriosis including genetic alterations, hormonal influences, oxidative stress, and inflammation. Malignancy most often occurs in ovarian endometriomas with less common sites involving the rectovaginal septum, rectosigmoid colon, and abdominal wall scars. The most common pathologic subtypes are endometrioid adenocarcinoma and clear cell carcinoma. MRI is the most specific imaging modality for evaluating EAOC. Key MR features include solid enhancing nodules (accentuated by subtraction imaging), nodular septations, loss of T2 shading within the endometrioma, and diffusion restriction.

Conclusions EAOC is a distinct disease that affects women with benign endometriosis at younger ages than classic ovarian cancer. Understanding the imaging features of malignant transformation of endometriosis is essential for early diagnosis and timely definitive treatment.

Keywords Endometriosis-associated ovarian carcinoma · Malignant endometriosis · Endometriosis · Pathology · Clear cell carcinoma · Endometrioid adenocarcinoma

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Introduction

Ovarian carcinoma is the seventh most common cancer in women worldwide and accounts for 4.3% of all female cancer-related deaths each year [1]. Endometriosis-associated ovarian carcinoma (EAOC), the malignant transformation of endometriosis, is a unique pathologic subset of ovarian malignancies with distinct histologic and imaging features. Although transformation from benign to malignant endometriosis is rare, occurring in approximately 1%, the relative risk of developing ovarian cancer is up to 4.2 times greater in women with long-standing endometriosis than the general population [2, 3]. Additionally, women with EAOC typically present 10 to 20 years younger than de novo ovarian cancer [4–6].

Endometriosis-associated ovarian carcinoma has been clinically reported to have better outcomes and prognosis than non-EAOC. It remains unclear if this is due to the distinct biological differences between the two cancer types or because EAOCs typically present with a higher rate of well-differentiated, early-stage tumors. Unfortunately, EAOCs are less sensitive to conventional platinum-based chemotherapy regimens and therefore early detection, diagnosis, and treatment are imperative [7, 8].

Imaging plays a crucial role in identifying suspicious features that distinguish benign endometriomas and endometriosis from their malignant counterparts. This enables clinicians to appropriately recommend surgical consultation and intervention, as histologic evaluation remains the mainstay for definitive diagnosis.

This article discusses the role of imaging in diagnosis of EAOC, with emphasis on the MR imaging features. Histologic correlation will exemplify the common pathologic findings encountered with EAOC.

Pathogenesis of malignant transformation of endometriosis

Historically, endometriosis-associated ovarian carcinoma (EAOC) has been defined as having ovarian cancer plus at least one of the following: (1) endometriosis in the ipsilateral ovary, (2) endometriosis in the contralateral ovary, (3) pelvic endometriosis, or (4) histopathology demonstrating the transition from benign to malignant endometriosis [9]. The most common location of origin is the ovary (75%) with less common sites involving those affected by benign deep infiltrating endometriosis such as the rectovaginal septum, the rectum and colon, pelvic peritoneum, and abdominal wall scars (Figs. 1, 2) [8]. The most common histologic subtypes of endometriosis-associated

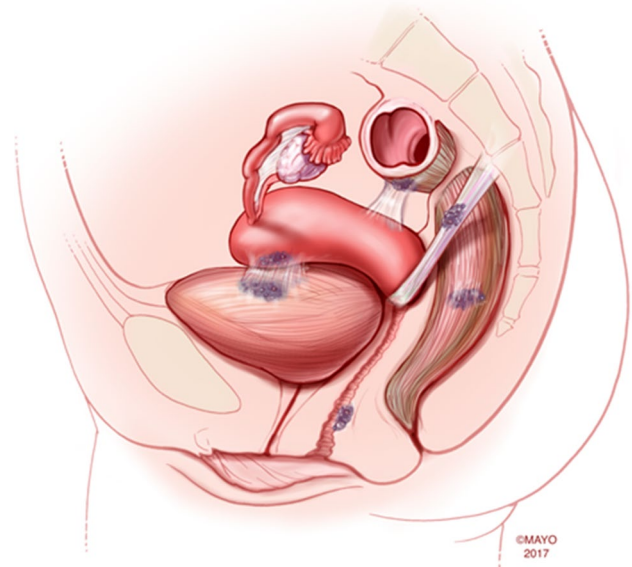


Fig. 1 Common extra-ovarian sites of benign deep infiltrating endometriosis. Extra-ovarian sites of deep infiltrating endometriosis commonly include the uterosacral ligaments, rectouterine pouch, vagina, rectosigmoid colon, and bladder

malignancy are endometrioid adenocarcinoma and clear cell carcinoma. Less common tumors include endocervical type mucinous borderline tumors, endometrial stromal sarcoma, and Müllerian adenosarcomas [8].

The proposed transformation from benign to atypical (borderline) to EAOC involves a combination of molecular genomic alterations, oxidative stress, inflammation, and hormonal influences such as hyperestrogenism [9]. Both endogenous and exogenous hyperestrogenic states such as obesity and therapy with unopposed estrogens after hysterectomy have been shown to be risk factors for developing EAOC [10]. Modern genome sequencing studies report several frequently encountered molecular abnormalities in EAOC including the activation of oncogenic KRAS and PI3 K pathways and inactivation of tumor suppressor genes PTEN and ARID1A (Figs. 3, 4) [9, 11]. The molecular alterations of EAOC are becoming more understood, and currently these mutations are regarded as somatic mutations of the tumor and not part of a genetic syndrome [11, 12]. However, clear cell carcinoma specifically has been associated with Lynch syndrome [13, 14].

Although the precise mechanism is not completely understood, multiple studies have shown that the risk for developing EAOC is significantly reduced by inhibiting ovulation and/or reducing retrograde menstruation from the use of hormonal contraceptives, multiple pregnancies, tubal ligation, and hysterectomy [9].

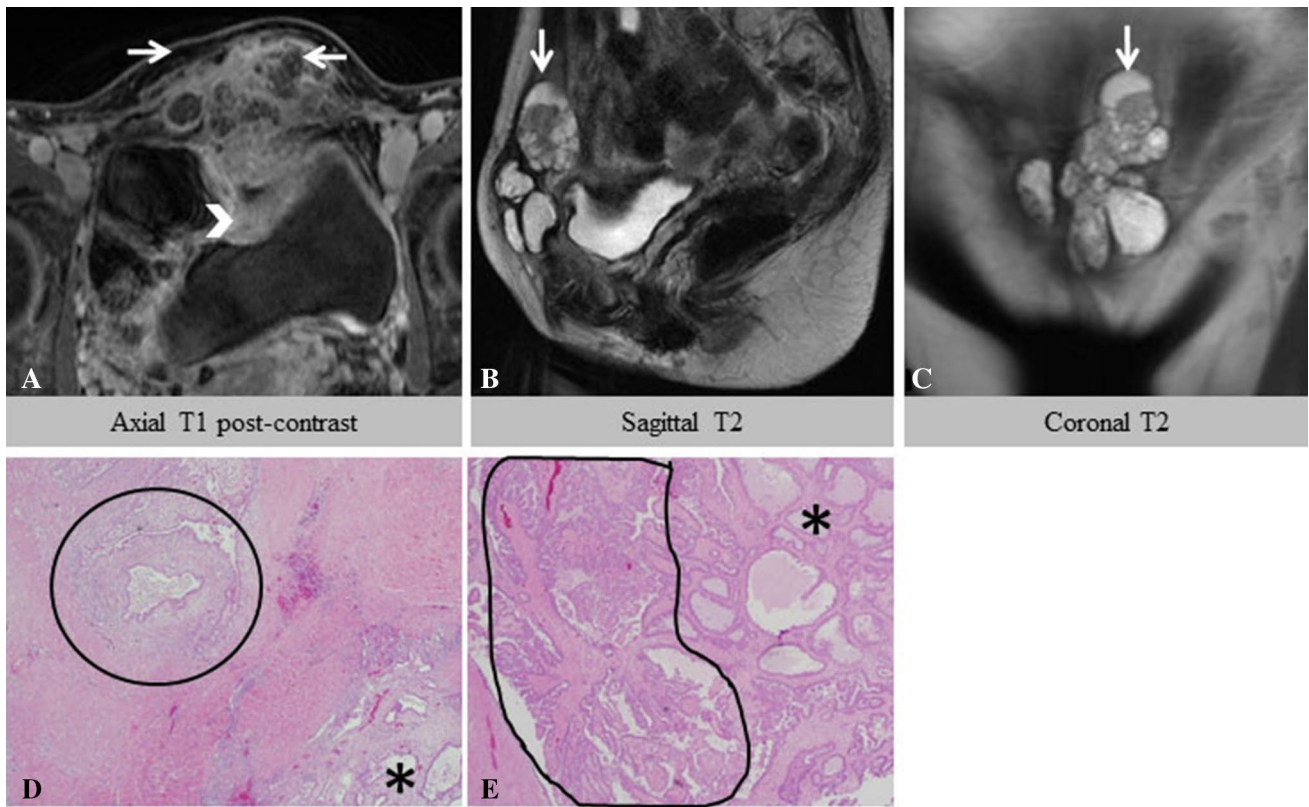


Fig. 2 MRI showing EAOC of the abdominal wall arising in a cesarean section scar **a** Axial post-contrast image of the pelvis shows a large mass with multiple enhancing solid nodules in the anterior abdominal wall musculature (arrows) invading into the bladder wall (arrowhead). **b** and **c** Sagittal and coronal T2 images show the exten-

sive lobulated cystic components of the mass (arrows). **C**. Surgical pathology revealed clear cell carcinoma (*) arising in a background of endometriosis (circle). **d** Low-power magnification of the tumor shows both the papillary (outline) and tubulocystic (*) patterns of clear cell carcinoma

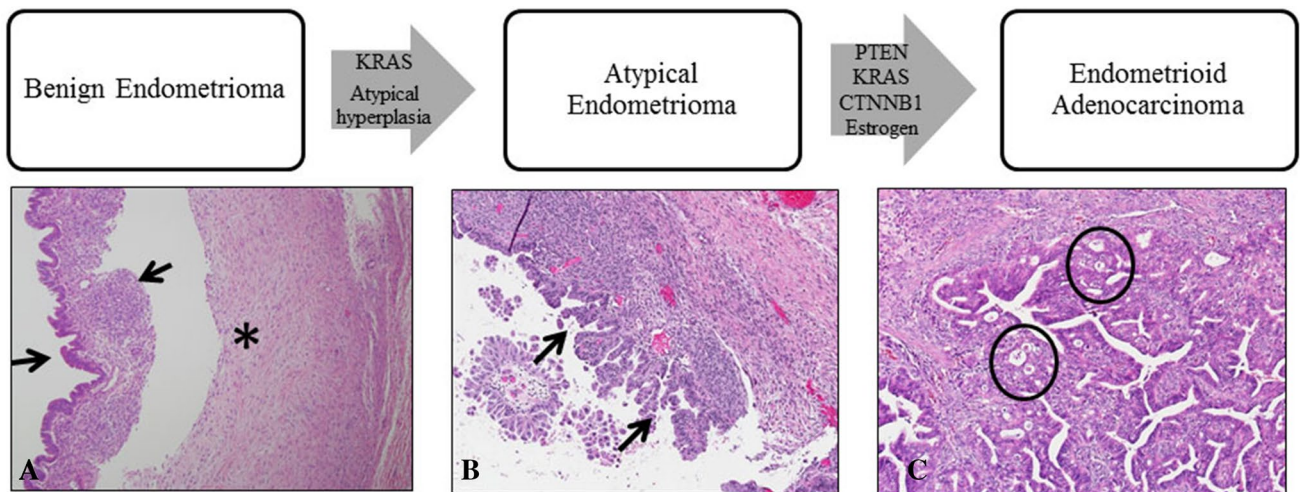


Fig. 3 Common genomic alterations encountered in malignant transformation of a benign endometrioma to endometrioid adenocarcinoma. **a** Benign endometrioma with endometrial epithelium lining and endometrial stroma (arrows) adjacent to normal ovarian stroma

(star). **b** Endometrioma with papillary proliferation and hyperplasia (arrows). **c** Endometrioid adenocarcinoma with back-to-back glands and cribriform formation (circles)

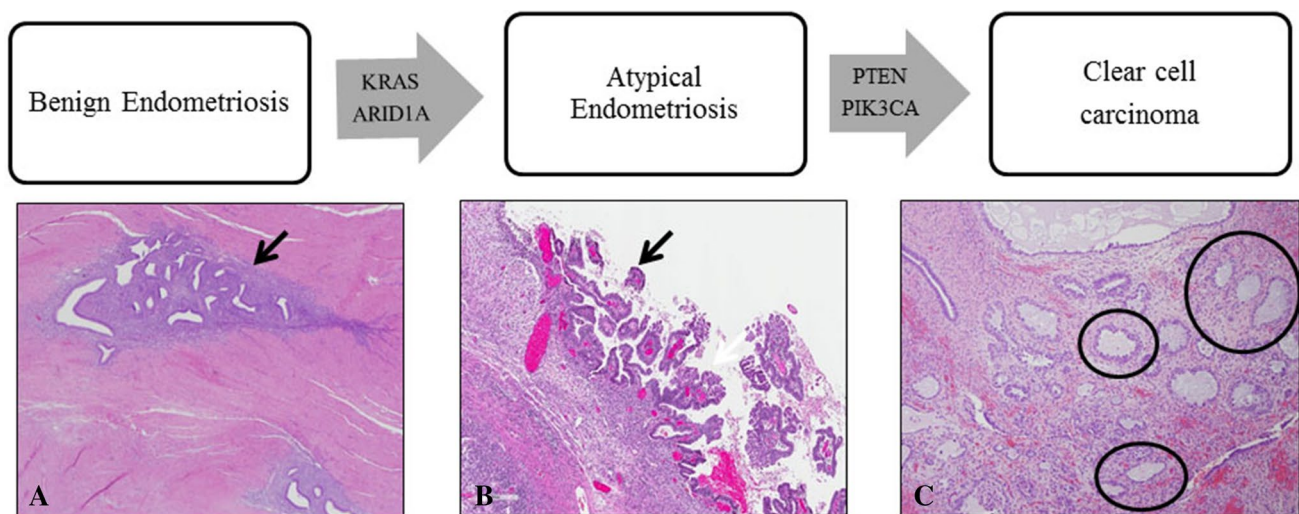


Fig. 4 Common genomic alterations encountered in malignant transformation of benign endometriosis to clear cell carcinoma. **a** Benign endometriosis (arrow) surrounded by normal stromal tissue. **b** Atypical

cal endometriosis with hyperplasia and papillary proliferation (arrow) **c** Classic tubulocystic pattern of clear cell carcinoma with numerous various-sized tubules and cysts (circles)

Histopathology of malignant transformation of endometriosis

Benign endometriosis

Endometriomas (endometriotic cysts) are cystic forms of endometriosis which may or may not be associated with endometriosis elsewhere in the pelvis. Endometriomas vary in size and can measure up to 15 cm. The cyst is lined with endometrial epithelium with underlying endometrial stroma and often shows focal areas of hemorrhage [15]. The internal contents are dark brown, old blood products giving rise to the term “chocolate cyst.” Other histologic hallmarks of an endometrioma include hemosiderin-laden macrophages and fibrosis. The epithelial lining of the cyst can show nuclear atypia and mitotic figures [15].

As discussed in the previous section, identical mutations of ARID1A, PI3KCA, and loss of heterozygosity of PTEN are histologically detected in the normal epithelium of an endometrioma as well as EAO, indicating these molecular alterations play an important role in tumor development [15].

Endometrioid carcinoma

Endometrioid carcinoma is an ovarian epithelial tumor that resembles endometrioid carcinoma of the uterus and accounts for 10–15% of ovarian carcinomas (the second most common form of ovarian epithelial malignancy and the most common in the 5th to 6th decades of life). Up to 42% of these tumors are associated with endometriosis

in the same ovary or elsewhere in the pelvis [16, 17]. Endometrioid carcinomas on average measure 15 cm. Macroscopically, the tumors are friable soft masses with solid components partially filling cystic spaces containing blood-stained fluid. Typical histological features are small to large back-to-back glands with labyrinthine branching or cribriform architecture (Fig. 3) [15]. The glands are typically lined by stratified columnar epithelium with pseudostratified nuclei and moderate cytological atypia; destructive growth with stromal invasion and desmoplastic/inflammatory stromal reaction can be seen [18]. Grading of ovarian endometrioid carcinoma is the same as uterine endometrioid adenocarcinoma and most are grade 1 or 2 [15].

Clear cell carcinoma

Clear cell carcinoma is the ovarian tumor most frequently associated with ovarian or pelvic endometriosis; arising from endometriosis in 50–70% of cases [19, 20]. These tumors are typically unilateral; measuring on average 15 cm. Grossly, they range from solid, to solid and cystic, to mainly cystic with fleshy nodules lining an endometriotic cyst [21]. Histopathologically, the tumors display tubulocystic, papillary, and solid patterns which can be admixed to varying degrees (Fig. 4) [15, 21]. They are composed of clear, eosinophilic, flattened, or hobnail cells with moderate to marked cytological atypia. Psammoma bodies and eosinophilic hyaline bodies can be present. Clear cell carcinomas are considered high grade [15].

MR imaging features of benign endometriosis

The distinctive MR imaging features of benign endometriosis have been well described in the literature. Endometriomas are ovarian cysts with thick fibrous walls lined by endometrial tissue which lead to high concentrations of hemorrhagic breakdown products within the cyst over repeated menstrual cycles [22]. These blood products appear hyperintense on T1-weighted fat-suppressed sequences and exhibit classic “T2 shading” on T2-weighted sequences, with a T2 dark hemosiderin rim (Fig. 5). Associated T2 dark focal spots have also been described [5, 22, 23]. Constellation of these features increases the specificity of MR to 98% for characterizing a lesion as a benign endometrioma and helps in its differentiation from other hemorrhagic lesions [5]. Bilaterality and multiplicity of adnexal lesions also help establish the diagnosis of endometriomas [5, 24]. Post-gadolinium

sequences show enhancement of the fibrous cyst wall which appears thin and without distinct nodules (Fig. 5). Diffusion-weighted imaging has become a common sequence in pelvic imaging protocols but the presence of restricted diffusion can be seen in both benign and malignant processes. Benign etiologies exhibiting restricted diffusion include hemorrhagic cysts, endometriomas, infiltrating endometrial implants, and mature cystic teratomas [24]. Characteristics of abnormal restricted diffusion favoring a malignant process will be discussed in a subsequent section.

Deep pelvic endometriosis, or infiltrating endometriosis, is defined as endometriotic implants with subperitoneal invasion penetrating greater than 5 mm in depth [25]. Repeated menstrual cycles cause bleeding in these implants which incite an inflammatory response and fibrous reaction over time [25]. These fibrotic endometrial implants appear as T2-hypointense solid foci on MRI that can progressively enhance after gadolinium contrast administration depending on the proportion of inflammatory reaction, glandular

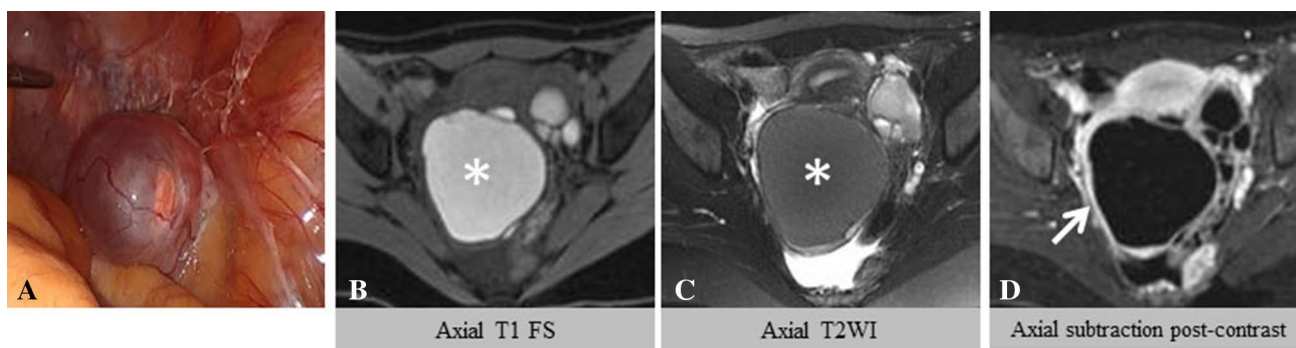


Fig. 5 Surgical and MRI appearance of a benign endometrioma. **a** Intraoperative photograph of a benign endometrioma. **b** MRI axial T1-weighted fat-suppressed image shows hyperintense blood products within the endometrioma (*). **c** The T2-weighted image demon-

strates classic T2 shading of the blood products (*). The T1-weighted post-contrast sequence shows enhancement of the thick, fibrous wall (arrow)

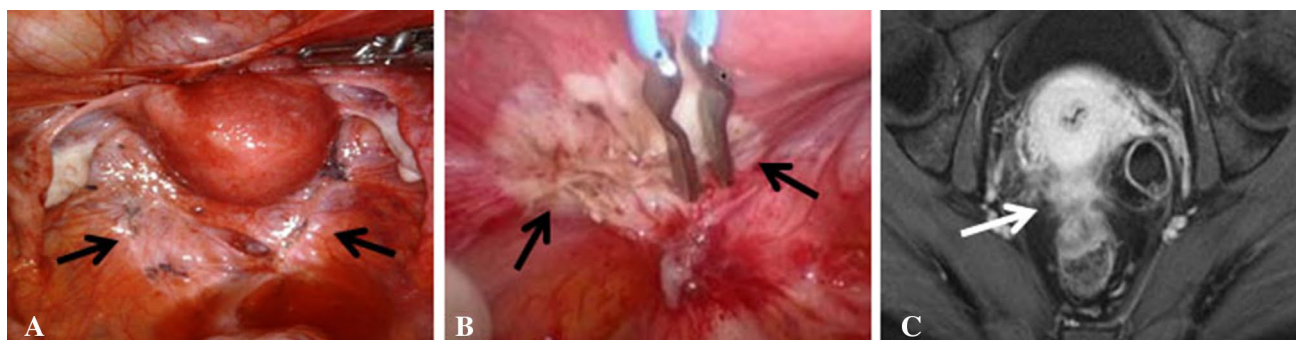


Fig. 6 Surgical and MRI appearance of benign deep infiltrating endometriosis. **a** and **b** Intraoperative photographs of deep infiltrating endometriosis involving the uterosacral ligaments and the pelvic

peritoneum (arrows). **c** MRI axial T1-weighted, delayed post-gadolinium image of the pelvis shows enhancing fibrotic, spiculated bands of endometriotic implants at the rectouterine pouch

tissue, and fibrosis (Fig. 6) [25]. Tethering of involved pelvic structures and organs is a common feature of infiltrating endometriosis which can contribute to a tubal abnormality such as hydrosalpinx in almost one-third of women [22, 26].

Utility of imaging in the evaluation of EAO

The clinical presentation of ovarian cancer is often non-specific as symptoms can be vague [27]. Endometriosis-associated ovarian carcinoma can present with overlapping symptoms of benign endometriosis such as dysmenorrhea and dyspareunia [28, 29]. Pelvic pain, urinary frequency, gastrointestinal symptoms like bloating and abdominal distention, a palpable mass, and newly developed or exacerbated dysmenorrhea and dyspareunia have been shown to be more common presenting symptoms unique to women with EAO [28, 29]. In addition to non-specific symptoms, the clinical diagnosis of EAO is also confounded by non-reliable tumor bio-markers, as normal CA-125 levels can be seen with EAO and elevated CA-125 can be seen in benign endometriosis [28, 30–32].

Evaluation preceding a diagnosis of ovarian cancer often includes abdominal and pelvic imaging. One study showed that in the 1 to 3 months preceding a diagnosis of ovarian cancer, 70% of patients with suspicious symptoms (defined as gastrointestinal symptoms, abdominal pain, pelvic pain, and abdominal swelling) had abdominal imaging and 54% of such patients had pelvic imaging (including X-rays, ultrasound, CT, and/or MRI) [33]. This significant proportion of women undergoing imaging for the workup of sometimes vague clinical symptoms exemplifies the importance for radiologists to be able to recognize the imaging findings of ovarian cancer.

After taking a history and physical examination, ultrasound of the pelvis is usually the first imaging modality

recommended by primary care physicians to evaluate symptoms of pelvic pain or abnormalities discovered on physical exam [34–36]. Several sonographic features have been reported to distinguish benign from malignant endometriomas/endometriosis.

Sonographic features

Size, echogenicity, and loculations

Malignant lesions are often larger and contain solid tissue and/or papillary excrescences that demonstrate internal vascularity with Doppler flow compared to benign endometriotic lesions [30]. In one study, the median maximum diameter of malignant endometriotic tumors was 10.7 cm compared to 5.8 cm for benign endometriomas ($p < 0.0001$) [30]. Benign endometriomas often show internal contents of homogeneous, low-level echoes, whereas EAOs exhibit more variable internal contents including anechoic fluid, low-level echoes, and mixed internal echogenicity (Fig. 7). Malignant endometriomas are more often multilocular (47%) compared to benign endometriomas (9.7%) [30].

Solid mural nodules, papillary projections, and doppler flow

Solid mural nodules with Doppler flow indicating vascularity are more common in malignant tumors and should be considered a highly suspicious and concerning feature (Fig. 8). Papillary projections are also more frequently found in malignant lesions compared to benign endometrioid cysts [30].

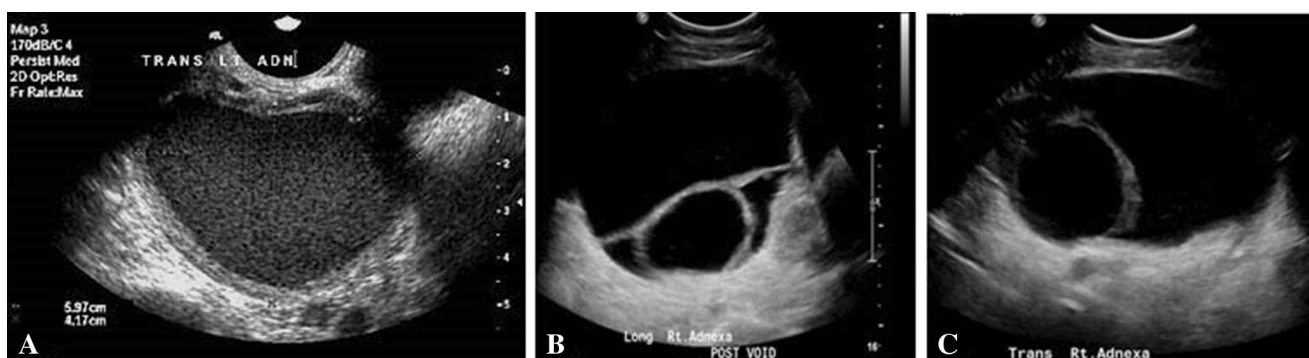


Fig. 7 Sonographic appearance of a benign unilocular endometrioma versus a malignant multilocular endometrioma. **a** Classic sonographic appearance of a benign, unilocular endometrioma with homogenous, low-level internal echoes. **b** and **c** Transverse and long views of a

13-cm multilocular anechoic right adnexal mass with thick internal septations in a patient with endometrioid cystadenocarcinoma arising from an endometrioma

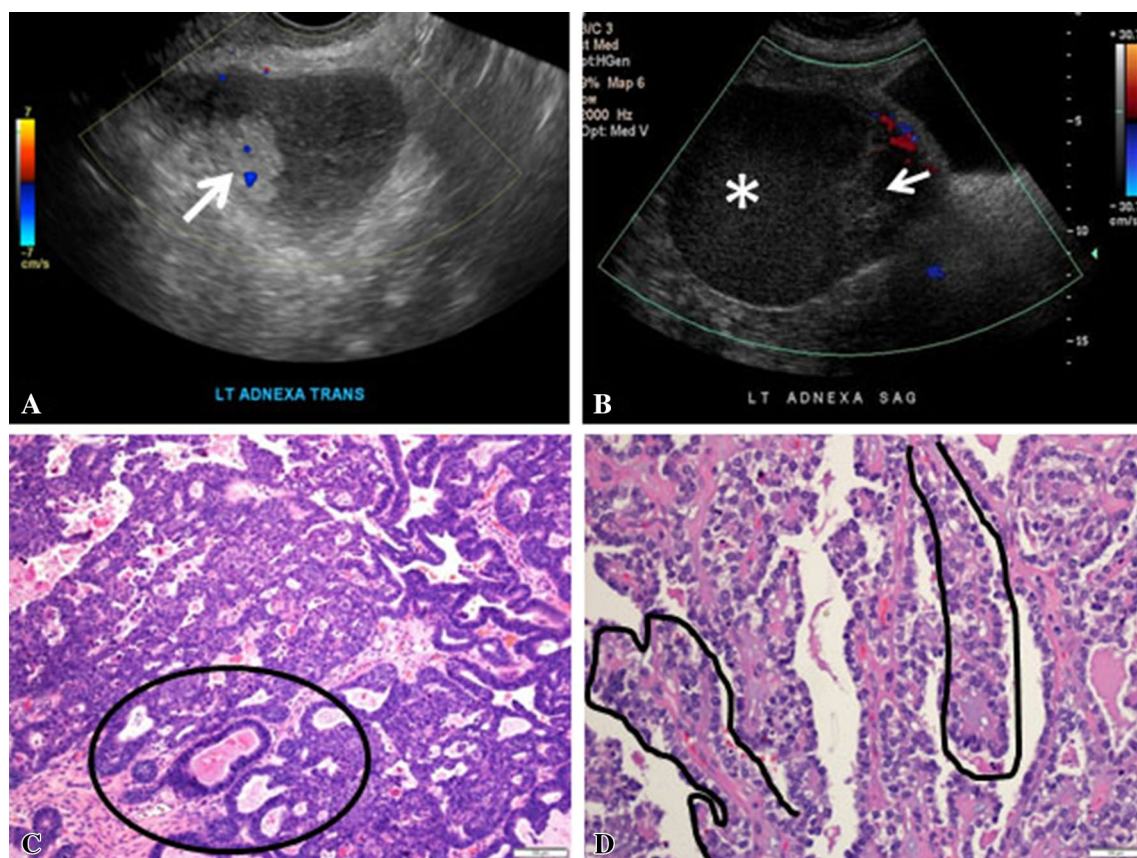


Fig. 8 Malignant ultrasound feature of solid mural nodules with Doppler flow. **a** A 61-year-old female with a 4.5-cm complex left adnexal mass showing a vascular, solid mural nodule (arrow). Surgical pathology revealed ovarian endometrioid adenocarcinoma. **b** A 60-year-old

female with abdominal bloating. Ultrasound shows a 13-cm complex left adnexal mass (*) with a solid, vascular mural nodule (arrow). **c** and **d** Surgical pathology revealed a small focus of clear cell carcinoma (circle) and endometrioid adenocarcinoma (outline)

Role of CT

The role of CT in EAO is primarily for staging and not for lesion characterization although the hallmark of ovarian carcinoma staging remains surgical. CT is more sensitive than ultrasound in detecting pelvic lymphadenopathy and peritoneal implants and can more easily assess for distant thoracic metastases than MRI [38]. Additionally, patients undergoing neoadjuvant chemotherapy for unresectable stage IV disease can be followed with CT to assess treatment response, and those who have undergone surgical resection can be followed with CT surveillance.

CT features

Enhancing solid mural nodules, peritoneal metastases, and ascites

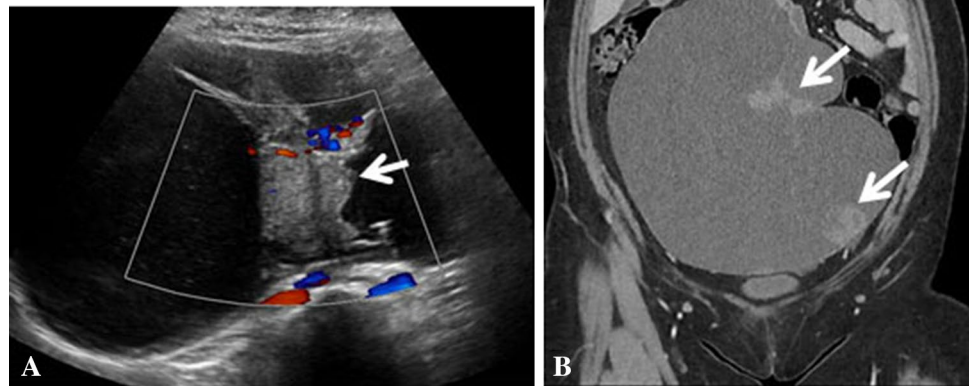
CT findings of ovarian lesions reported to be predictive of malignancy include papillary projections in a cystic lesion,

enhancing solid mural nodules, peritoneal metastases, and ascites [37]. The solid, papillary components show heterogeneous enhancement after intravenous contrast administration (Fig. 9). Large-volume ascites tends to more often be malignant compared to minimal or small volume ascites [39]. Lack of ascites should not, however, exclude malignancy, as large-volume ascites is much more common in patients with serous carcinomas and mucinous cystadenocarcinomas than patients with EAO [22].

Role of MRI

The emerging role of MRI in the workup of adnexal masses is most recognized for sonographically indeterminate lesions. Recent studies show that in routine clinical practice, 5 to 25% of adnexal masses will be sonographically indeterminate [40]. Although many of these will be pathologically benign, the ability to make a definitive imaging diagnosis using MRI can reduce unnecessary surgery, imaging follow-up, and patient anxiety. Additionally, MRI has been shown

Fig. 9 Enhancing solid mural nodules in a malignant endometrioma. **a** and **b** Sonographic and coronal CT images of a large cystic mass with solid mural nodules (arrows) in a patient with mixed ovarian clear cell and endometrioid adenocarcinoma arising from an endometriotic cyst. Note the lack of ascites despite the large tumor size



to be superior to ultrasound in determining organ of origin for large pelvic masses [40].

MRI features

Solid enhancing mural nodules

Many studies have shown that a solid, enhancing mural nodule within an otherwise cystic pelvic lesion is a morphologic MRI feature suggestive of malignancy [40–42]. These enhancing mural nodules correlate to the vascular solid nodules and papillary projections on ultrasound. The nodules are commonly hypo or isointense on pre-contrast T1-weighted sequences and homogeneously or heterogeneously enhance on T1-weighted post-gadolinium sequences (Fig. 10). The nodules can show variable signal intensity on T2-weighted sequences. The shape and size of the nodule do not correlate to a specific histologic subtype of EAO [41].

Subtraction imaging after gadolinium administration is essential to evaluate small nodules, the enhancement of which can be masked by hyperintense hemorrhagic fluid on T1-weighted sequences (Fig. 11).

Septations with nodules

Septations within benign adnexal masses such as endometriomas typically relate to retracting blood clot [43, 44]. Differentiating benign versus malignant appearing septations can be challenging; however, benign septations should remain smooth even if thick. Irregular and/or nodular septations should be considered highly suspicious [5, 45]. Malignant, nodular septations enhance after the administration

of intravenous contrast and can show restricted diffusion (Fig. 12) [47].

Loss of T2 shading

Benign endometriomas classically exhibit the MR feature of T2 shading. This refers to their higher signal intensity on T1-weighted imaging caused by the proteinaceous and viscous contents of the endometrioma which expectedly exhibits T2 signal intensity lower than that of simple fluid. Malignant endometriomas can exhibit loss or absence of this T2 shading, thought possibly due to dilution of proteinaceous contents by serous fluid secreted by the tumor (Fig. 13) [22, 46]. Although helpful when present in conjunction with other features of malignancy, loss of T2-shading alone is non-specific and can also be seen in benign endometriomas.

Diffusion restriction

In many organ systems, malignant tumors will show restricted diffusion on high *b* value diffusion-weighted imaging (DWI) with corresponding low ADC values. Abnormal restricted diffusion does occur in EAO; however, it is not specific for malignancy and can also be seen in benign hemorrhagic lesions such as physiologic hemorrhagic cysts, endometriomas, and benign mature cystic teratomas [24, 47]. Benign endometriomas can also display low ADC values therefore mimicking malignancy [24].

Currently, differing ADC values of benign and malignant ovarian lesions have not been shown and diffusion-weighted imaging cannot be used in isolation [24, 48]. However, in EAO, the areas of restricted diffusion and low ADC values should overlap with a lesion's solid mural nodules and enhancing nodular septations (Fig. 14) [47]. This is in

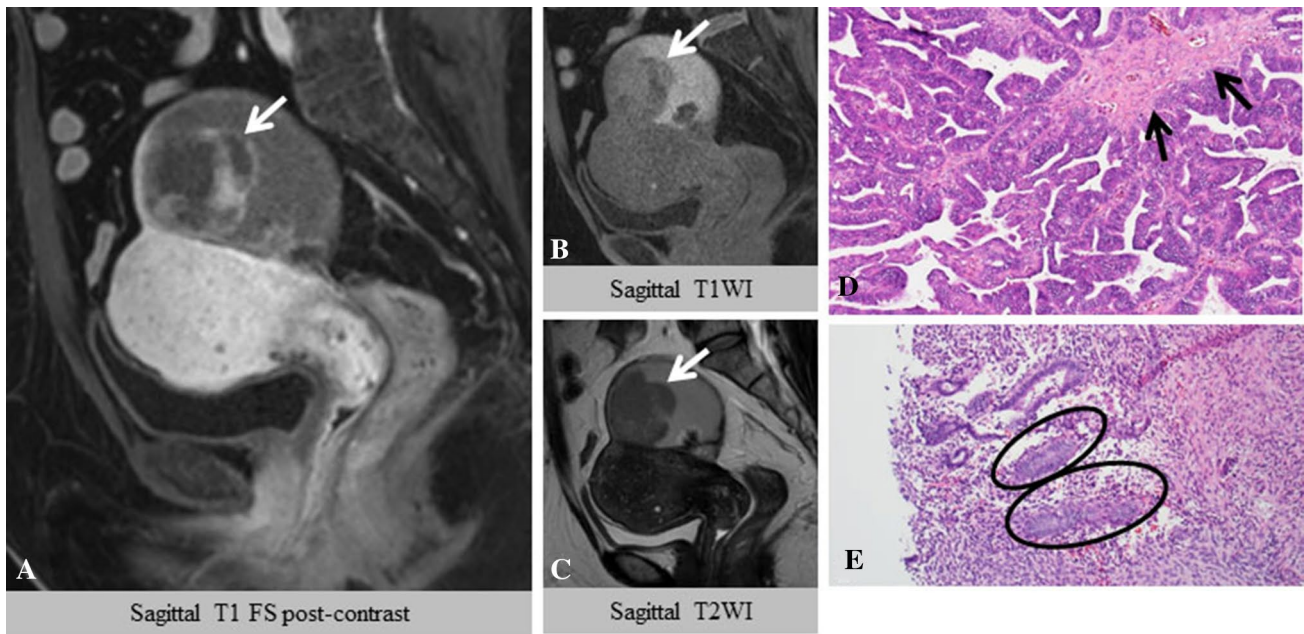


Fig. 10 Malignant MRI feature of solid enhancing nodules. **a** Sagittal images of the pelvis show a heterogeneously enhancing, centrally necrotic solid mass arising from the left ovary (arrow). **b** and **c** The solid nodule (arrow) is primarily T1 and T2 hypointense. **d** and **e**

Surgical pathology confirmed well-differentiated endometrioid carcinoma (arrows) with areas of benign endometriosis in the contralateral right ovary and fallopian tube (circles)

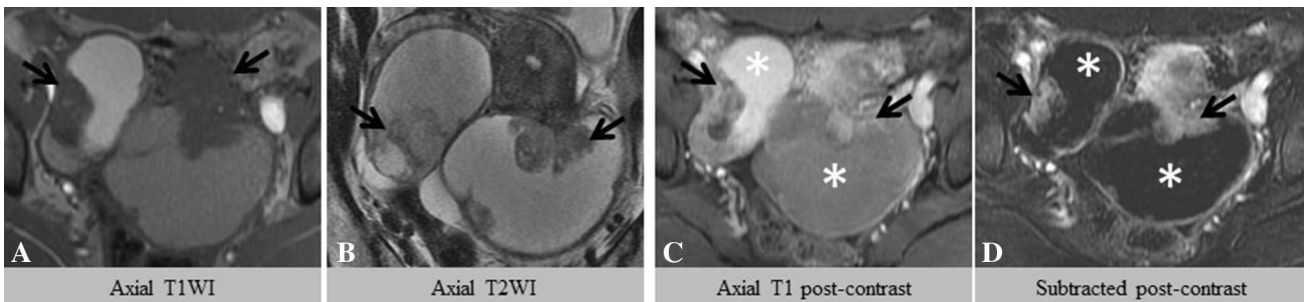


Fig. 11 MRI Subtraction imaging for distinguishing solid mural nodules. **a** and **b** T1 hypointense, T2 isointense solid enhancing nodules in bilateral adnexal masses (arrows). **c** and **d** Subtraction imaging bet-

ter differentiates the solid mural nodules (arrows) from the surrounding T1 bright hemorrhagic fluid (*). Surgical pathology showed bilateral mixed clear cell and endometrioid adenocarcinomas

contradistinction to the typical pattern of diffusion restriction in benign endometriomas which occurs more diffusely in the intracystic portion of the lesion [22, 47].

Pitfalls

There are several mimickers of endometriosis-associated ovarian cancer of which the radiologist must be aware. The normal ovarian parenchyma adjacent to an endometriotic cyst can be mistaken for an enhancing solid malignant nodule. The irregular or papillary like solid enhancing components of EAOc should be distinguished from the

crescent-shaped solid ovarian parenchyma which can characteristically contain T2-bright, non-enhancing follicles [41].

Deep invasive endometriosis of the rectosigmoid colon can mimic malignancy because the endometriotic implants grow through the rectal serosa into the muscularis propria causing a stricturing mass leading to the imaging and gross pathologic “mushroom cap” sign. The mushroom cap sign is a specific finding of solid invasive endometriosis of the rectosigmoid colon. T2-weighted MR images show low-signal-intensity mushroom base from fibrosis of the muscularis propria and a high-signal-intensity mushroom cap from the mucosa and submucosa displaced into the bowel lumen [49]. Post-contrast images exhibit heterogeneous enhancement

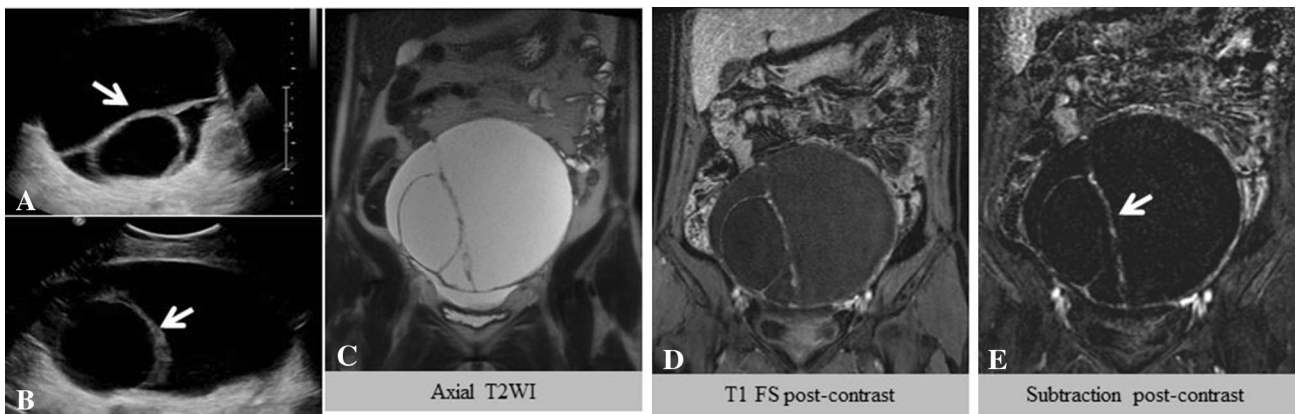


Fig. 12 Malignant MRI feature of enhancing nodular septations. **a** and **b** Transverse and longitudinal pelvic ultrasound shows a multilocular, cystic right adnexal mass with thick internal septations (arrows). **c–e** Pelvic MRI coronal images confirm an 18-cm cystic

pelvic mass containing nodular, enhancing septations accentuated on the subtraction sequence (arrow). Surgical pathology was endometrioid cystadenocarcinoma arising from an endometriotic cyst

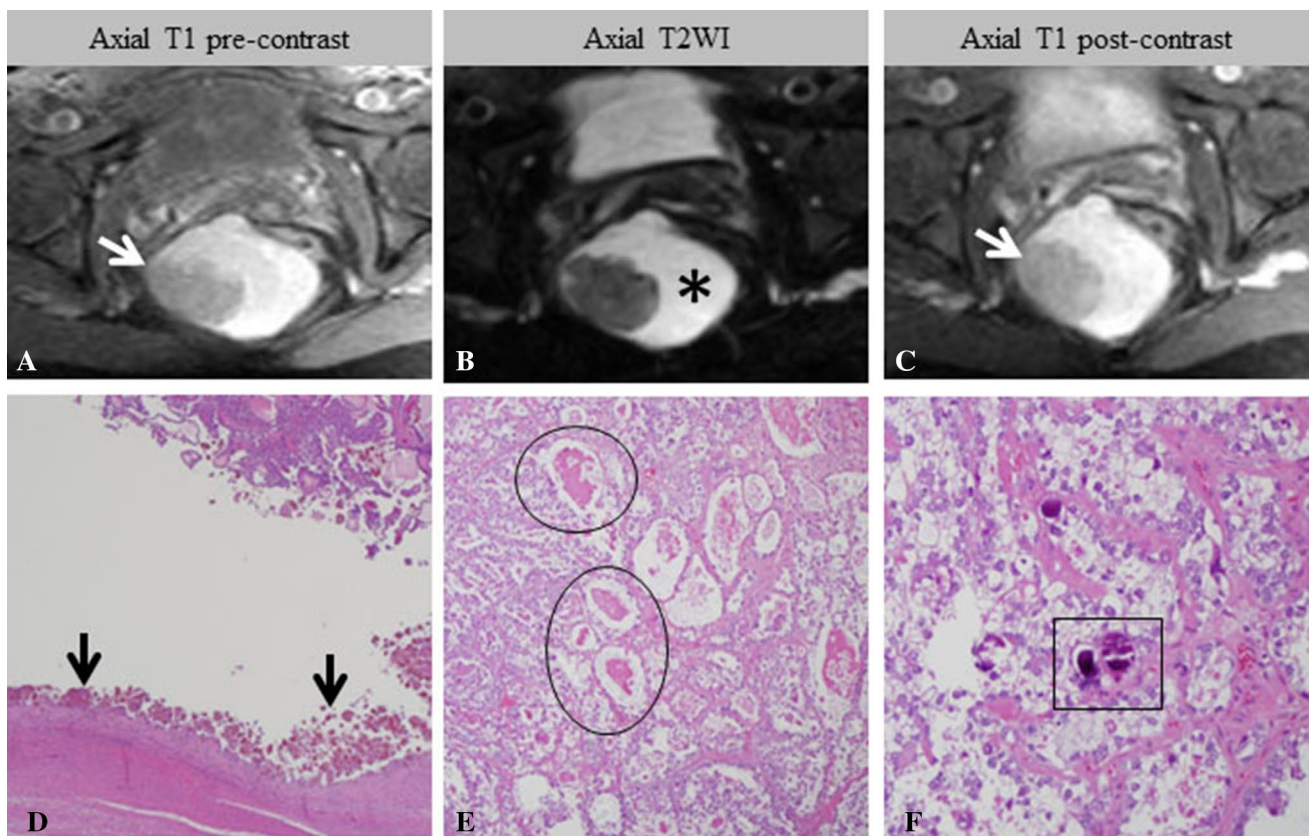


Fig. 13 Malignant MRI feature of loss of T2 shading in a malignant endometrioma. **a–c** Axial pre and post-contrast T1-weighted images show an enhancing mass arising in a right endometrioma (arrows) and loss of shading in the endometrioma which remains T2 hyperintense (*). **d–f** Surgical pathology is clear cell carcinoma.

d Low-power magnification shows hemorrhage in an endometrioma (arrows). **e** and **f** High-power magnification shows the tubulocystic pattern of clear cell carcinoma with classic eosinophilic secretions (circles), abundant clear cytoplasm, and psammoma bodies (square)

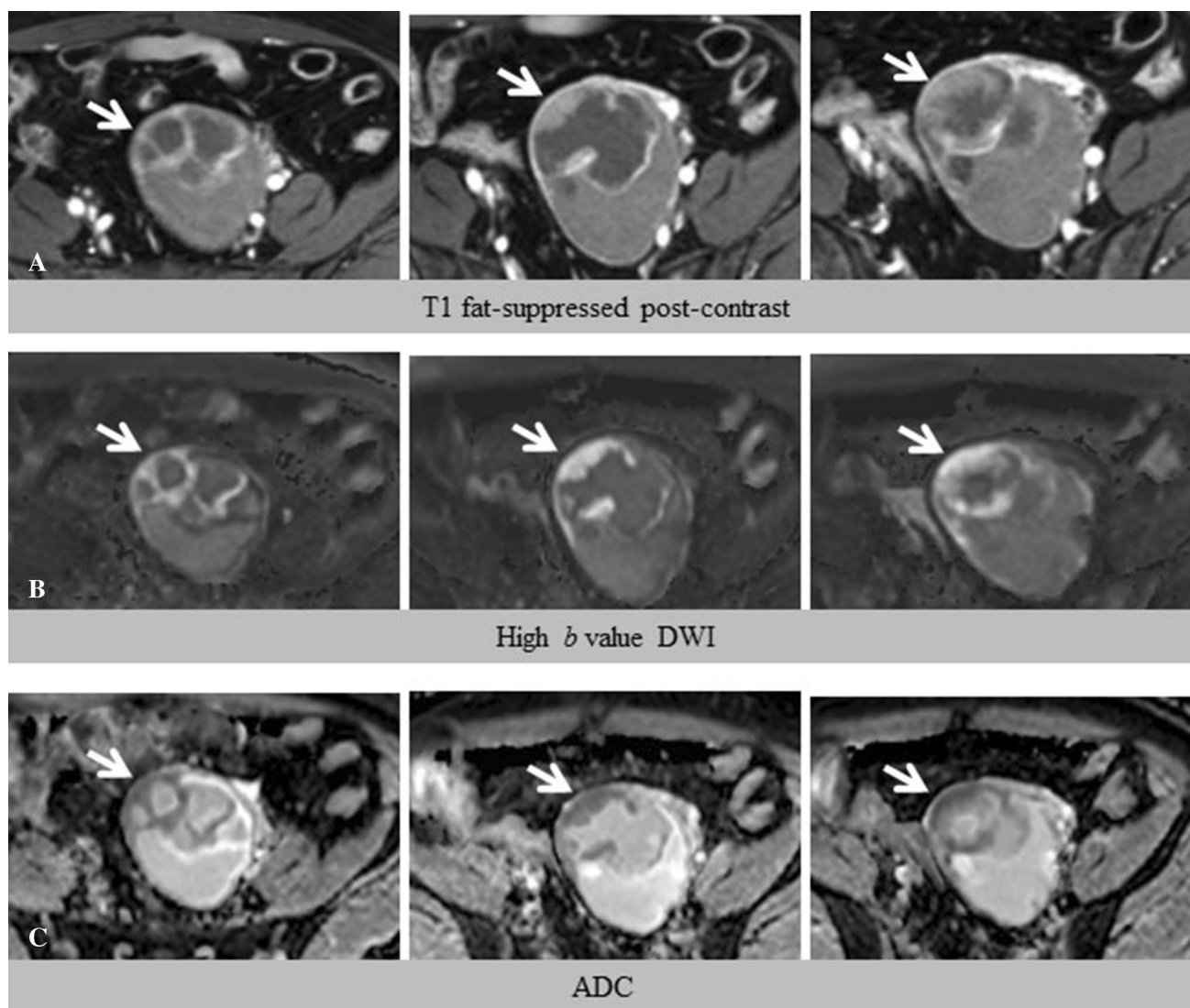


Fig. 14 Malignant MRI feature of restricted diffusion. Endometrioid adenocarcinoma shown on multiple images of the pelvis. **a** Axial T1-weighted fat-suppressed post-contrast images demonstrate

a peripherally enhancing, centrally necrotic nodule in a cystic left adnexal mass. **b** and **c** The enhancing portions of the nodule exhibit abnormal restricted diffusion with corresponding low ADC values

of the displaced mucosa, further adding to the malignant appearance (Fig. 15). Definitive diagnosis is often made with surgical pathology because these patients typically require resection of the affected rectosigmoid colon to alleviate symptoms of pain, rectal bleeding, and/or obstruction.

Decidualization of endometriomas during pregnancy is a well-known, yet rare entity that could potentially mimic malignancy (Fig. 16). Hypertrophy of both the endometrial stromal cells that line the gravid uterus and endometriomas occurs during pregnancy. Decidualized endometriomas mimic malignancy by exhibiting rapid growth, solid mural nodules or papillary excrescences, and vascularity on Doppler imaging. As gadolinium is contraindicated in pregnancy, MRI features that can help aid in the correct diagnosis include maintenance of T2 shading in the

cystic portions of the lesion and recognition that the solid portions should follow the signal intensity of the decidualized uterine endometrium (characteristically showing T2-hyperintensity) [50]. Additionally, diffusion imaging can help distinguish benign from malignant solid mural nodules in this setting. The solid mural nodules of decidualized endometriomas have been shown to have high signal intensity on low b value DWI, low signal intensity on high b value DWI, and high ADC values from T2 shine through. This differs from malignant solid mural nodules which exhibit high signal intensity on both low and high b value DWI with corresponding low ADC values [51]. Close postpartum follow-up imaging is required to ensure the lesions decrease in size and to exclude malignancy.

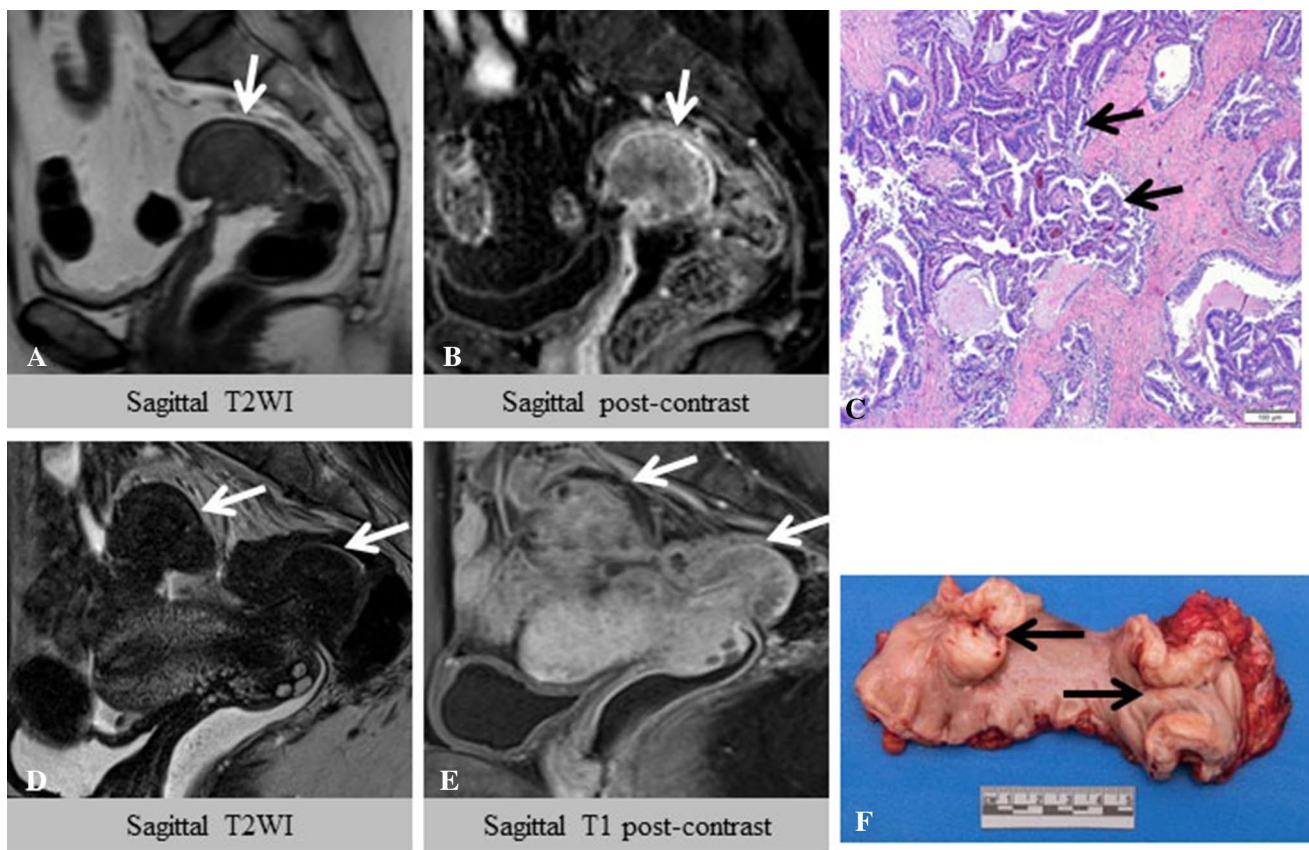


Fig. 15 Malignant and benign solid invasive endometriosis of the sigmoid colon. Top row: A 55-year-old female with endometrioid adenocarcinoma surgically resected 10 years ago presents with bright red blood per rectum. **a** and **b** Sagittal T2-weighted and post-contrast images of the pelvis show a mass within the sigmoid colon (arrows) demonstrating the “mushroom cap sign.” **c** Surgical pathology confirmed recurrent endometrioid adenocarcinoma in a background of

endometriosis invading the colon (arrows). Bottom row: A 31-year-old female with endometriosis, bloody stools, and cyclical abdominal pain. **d** and **e** Sagittal T2-weighted and post-contrast images of the pelvis show two endometriotic masses with full thickness intestinal wall involvement also exhibiting the “mushroom cap” sign (arrows). **f** Surgical pathology here revealed two strictures from benign deep infiltrating endometriosis (arrows)

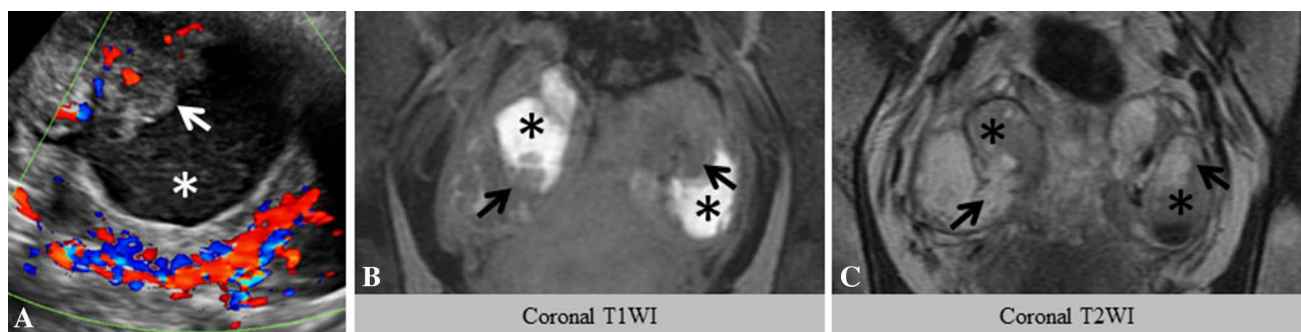


Fig. 16 Malignancy mimic of decidualized endometriomas during pregnancy. A 26-year-old pregnant female presents for 1st trimester OB scan. **a** Ultrasound of the right ovary shows a mass with mixed internal echogenicity including homogeneous low-level echoes (*) and intramural nodules (arrow). **b** The unenhanced coronal T1-weighted sequence demonstrates bilateral heterogeneous, multiloculated cystic ovarian masses with marked T1 hyperintense blood

products (*) and T1 isointense solid components (arrows). **c** The T2-weighted sequence shows shading of the T1 hyperintense material (*) and mild T2 hyperintensity of the solid portions (arrows). The findings are most consistent with decidualized endometriomas. On postpartum imaging, the masses all decreased in size confirming the suspected diagnosis

Summary

Endometriosis-associated ovarian carcinoma is unique from other ovarian tumors in its clinical presentation, prognostic implications, imaging appearance, and pathologic features. The radiologist's ability to recognize concerning features of EAOC is important to triage these patients quickly and appropriately. Ultrasound is often the first imaging exam ordered for women presenting with abdominopelvic symptoms. Suspicious features of an endometrioma, or any pelvic mass, include solid nodules and papillary excrescences with Doppler flow. CT plays a role in staging EAOC, but is not optimal for lesion characterization. MRI is the modality of choice for evaluating a suspicious or indeterminate mass in patients with endometriosis. MRI features of EAOC include enhancing solid mural nodules, nodular septations, restricted diffusion of the solid components, and loss of the classic endometriotic T2-shading. A combination of these features should prompt surgical evaluation and histologic diagnosis.

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