Abdominal Radiology
https://doi.org/10.1007/s00261-019-02309-4

SPECIAL SECTION: ENDOMETRIOSIS

Endometriosis in the postmenopausal female: clinical presentation, imaging features, and management

Adela G. Cope[1] · **Wendaline M. VanBuren**[2] · **Shannon P. Sheedy**[2]

Abstract
Postmenopausal endometriosis is an important clinical entity which is likely under-recognized and in which the Radiologist can play a valuable role. In this review, we describe the clinical presentation and management of postmenopausal endometriosis, appraising the literature and providing case examples. Persons with postmenopausal endometriosis may present with symptoms including pelvic pain or dyschezia, but endometriosis may also be an asymptomatic, incidental finding. Women may or may not have a prior history of endometriosis or a history of symptoms consistent with it. Therapies and conditions which increase exogenous or endogenous estrogen, respectively, increase the risk. Endometriosis can be found in different locations throughout the body, and the possibility of malignancy should be assessed, especially in the postmenopausal population, where age increases cancer risk. Treatment may involve surgery or medical interventions. Guidelines describing appropriate imaging surveillance in these patients are lacking. In the postmenopausal population, Radiologists need to consider endometriosis as a diagnosis, recommend appropriate exams such as MRI and US, and suggest endometriosis-associated malignancies when appropriate, based on classic morphologic features.

Keywords Endometriosis · Postmenopause · Magnetic resonance imaging · Ultrasonography

Introduction

Five to ten percent of women of reproductive age have endometrial glands and stroma present outside of the uterus, resulting in the condition known as endometriosis. Symptoms of the disease vary and may include dysmenorrhea, chronic pelvic pain, dyspareunia, dyschezia, and infertility. The pathogenesis of this chronic gynecologic condition is complex and likely multifactorial with genetic, epigenetic, environmental, and immunologic factors contributing to disease development [1, 2]. Lesion types include superficial peritoneal deposits, deep infiltrating endometriosis (implant or nodule with at least 5 mm depth), and endometriomas (chocolate cysts). A key factor in the development of endometriosis lesions and their associated symptoms appears to be estrogen exposure. Estrogen has a stimulatory effect on endometrial tissue both inside and outside of the uterus and may play a role in the associated inflammatory response. Additionally, inhibition of estrogen production with combined hormonal contraceptives, progestins, gonadotropin-releasing hormone agonists and antagonists, and aromatase inhibitors can result in improved symptoms in patients with endometriosis [1]. As systemic estrogen levels decrease significantly following menopause, endometriosis lesions regress in the majority of cases, resulting in symptom resolution. However, it is estimated that 2–4% of postmenopausal women may continue to be affected by endometriosis [3–5]. Similar to premenopausal patients, this condition can present in a variety of different ways and may be initially unrecognized or misdiagnosed.

Risk factors

Postmenopausal endometriosis should be considered when a patient has a history of symptoms before menopause that could potentially be related to previously unrecognized endometriosis, including dysmenorrhea, dyspareunia, dyschezia, infertility, and chronic pelvic pain. In patients with

✉ Adela G. Cope
cope.adela@mayo.edu

[1] Department of Obstetrics and Gynecology, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA

[2] Department of Radiology, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA

Published online: 07 November 2019

a history of prior hysterectomy or pelvic surgery, reports of adhesions, difficulty performing the procedure related to adhesions, or distorted anatomy with or without mention of lesions consistent with endometriosis could be evidence of prior endometriosis. Absence of these historical factors does not preclude its possibility, as demonstrated in case 1 (Fig. 1).

Hormone replacement therapy has been associated with postmenopausal endometriosis secondary to stimulation of endometrial deposits by exogenous estrogen [3, 6]. This effect may be more significant in unopposed estrogen therapy in which there is no progestin component [6]. Case 2 demonstrates a patient on hormone replacement therapy who was affected by postmenopausal endometriosis (Fig. 2). Other conditions that potentially increase serum estrogen levels, such as obesity, may also result in stimulation of endometriosis lesions, subsequent symptoms, or findings on imaging [3]. Tamoxifen is a selective estrogen receptor modulator known to have an agonist effect on endometrial tissue, and it is associated with endometrial abnormalities, including polyps, hyperplasia, and malignancy [7, 8]. Similar agonist activity on endometriosis lesions may occur in the setting of tamoxifen use, resulting in the development or progression of the disease [9–13]. In those with no prior diagnosis of endometriosis, it is unclear if this agonist activity results in formation of new lesions or unmasking of previously subclinical disease.

There are also reported cases of postmenopausal endometriosis in the absence of an identifiable source of systemic estrogen exposure or endogenous agonist activity [14]. In some patients, investigators have posited that such instances may be due to local estrogen production in the presence of aromatase within the endometriosis lesions themselves [3, 15]. Case 3 shows classic features of endometriosis in a postmenopausal patient on pelvic ultrasonography (US) and magnetic resonance imaging (MRI); however, there was no identifiable stimulation of exogenous estrogen activity (Fig. 3).

Signs, symptoms, and locations

Postmenopausal endometriosis may present as an asymptomatic incidental finding during imaging or surgery performed for other indications, as illustrated in case 4 (Fig. 4). When present, symptoms may be similar to those in premenopausal women, specifically pelvic pain, dyspareunia, and dyschezia, as was the instance for the patient in case 5 (Fig. 5). Patients may also present with abnormal vaginal bleeding [16, 17]. In all women, the most common locations where endometriosis is found are the ovaries, uterosacral ligaments, and pelvic peritoneal surfaces including the broad ligament, anterior cul-de-sac, posterior cul-de-sac, retro-ovarian fossa, and pelvic side walls [2]. While postmenopausal endometriosis is most commonly located in the ovary [4, 16, 17], it has also been reportedly found in many other locations, such as the urinary tract [18–20], large and small bowel [21–25], stomach [26], vagina [27, 28], skin [29, 30], diaphragm [20], and inferior vena cava [31]. Presenting symptoms can be related to the organ system where

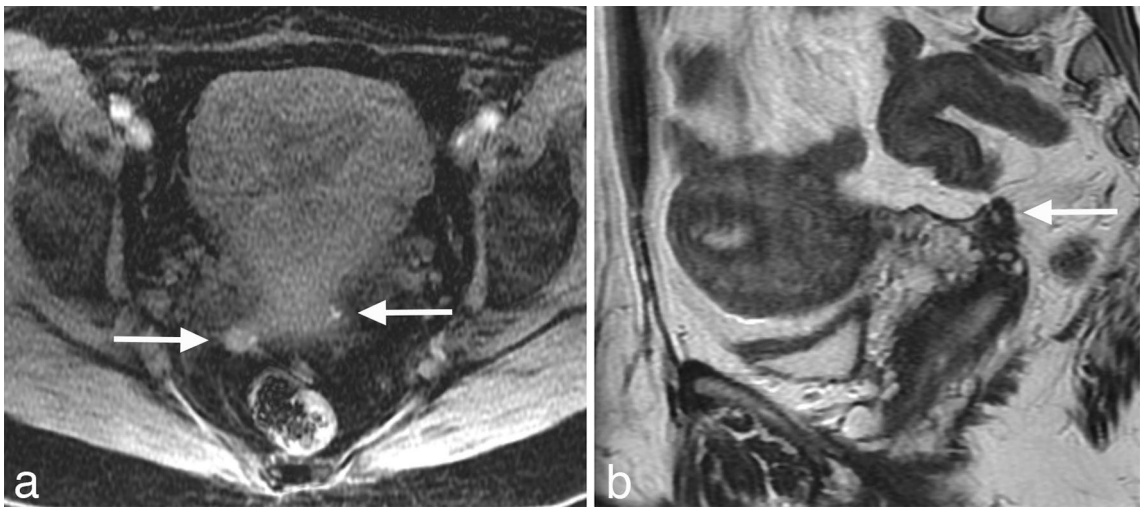

Fig. 1 Post-menopausal 59-year-old female with 2 years of bleeding was diagnosed with complex hyperplasia without atypia. Bleeding persisted despite several months of medroxyprogesterone. She underwent an abdominal hysterectomy, at which time unsuspected pelvic endometriosis was discovered. In retrospect, typical changes of endometriosis were present on pelvic MRI. (**a**) T1-weighted, fat-saturated axial image without contrast demonstrates small nodular hyperintense endometriotic deposits along the uterosacral ligaments bilaterally, right greater than left (arrows). (**b**) T2-weighted sagittal image demonstrates hypointense fibrotic nodularity and thickening from endometriosis involving the right uterosacral ligament (arrow)

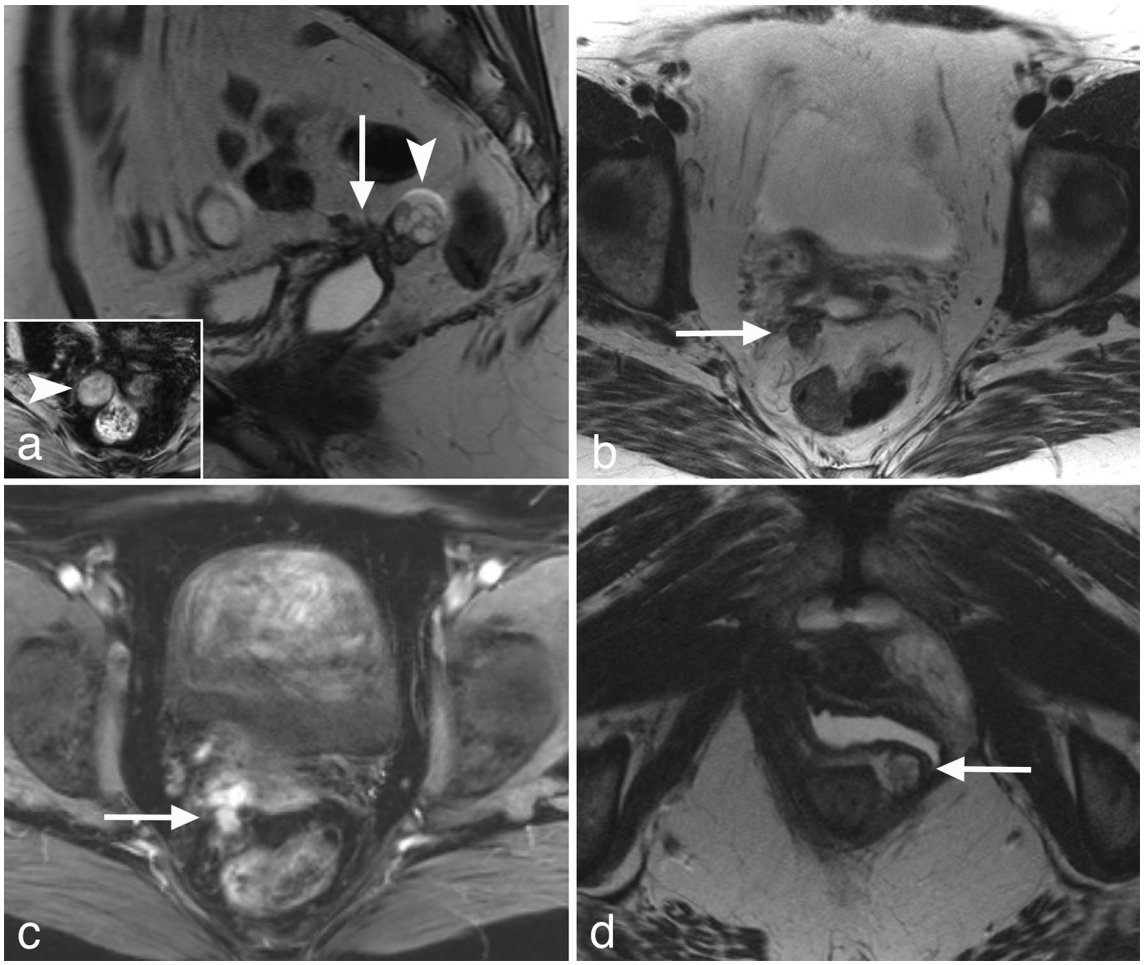

Fig. 2 Post-menopausal 54-year-old female with no known history of endometriosis, now with new diagnosis 1.5 years after starting hormonal replacement therapy for menopausal symptoms. **a** T2-weighted sagittal MR image demonstrates irregular, stellate T2 hypointense soft tissue thickening (arrow) and associated cystic change (arrowhead) at the right side of the vaginal cuff with adhesions and tethering to the posterior dome of the urinary bladder. The cystic portion of this process was hyperintense on T1 fat-saturated images (arrowhead, inset). Note distension of the vagina, achieved with administration of sterile sonographic gel. **b**, **c** Axial T2-weighted (**b**) and axial postcontrast T1-weighted (**c**) MRI images demonstrate intermediate T2 signal enhancing nodular soft tissue at the right side of the vaginal cuff (arrows). **d** Axial T2-weighted image demonstrates a rounded T2 hyperintense structure (arrow) in the left side of the anorectal vaginal septum. These surgical findings were found on pathology to represent endometriosis

lesions are located. For example, lesions involving the bladder can cause voiding dysfunction or hematuria, whereas lesions involving the bowel can result in rectal bleeding or bowel obstruction.

Typical MRI imaging appearance of pelvic endometriosis

Laparoscopy is the gold standard for the diagnosis of pelvic endometriosis; however, MRI can help the surgeon preoperatively identify the extent and severity of disease and can in particular identify disease that may be obscured at laparoscopy by adhesions.

On MRI, ovarian endometriomas present as solitary or multiple masses, typically with relatively homogeneous hyperintense signal on T1-weighted images. T1-weighted, fat-saturated images are critical to differentiate endometriomas from fat-containing mature cystic teratomas. On T2-weighted images, endometriomas have a variable appearance, ranging from homogeneous, heterogeneous, or gradated hypointensity (also known as "T2 shading") to intermediate signal intensity to high signal intensity, depending on the degree of hemoconcentration within the cyst [32]. Bilaterality and/or multiplicity of T1 hyperintense ovarian cysts and lack of resolution over time favor endometriomas over hemorrhagic ovarian cysts. A thick, T2 hypointense fibrous capsule (due to the presence of hemosiderin-laden macrophages from repeated hemorrhage) and the presence

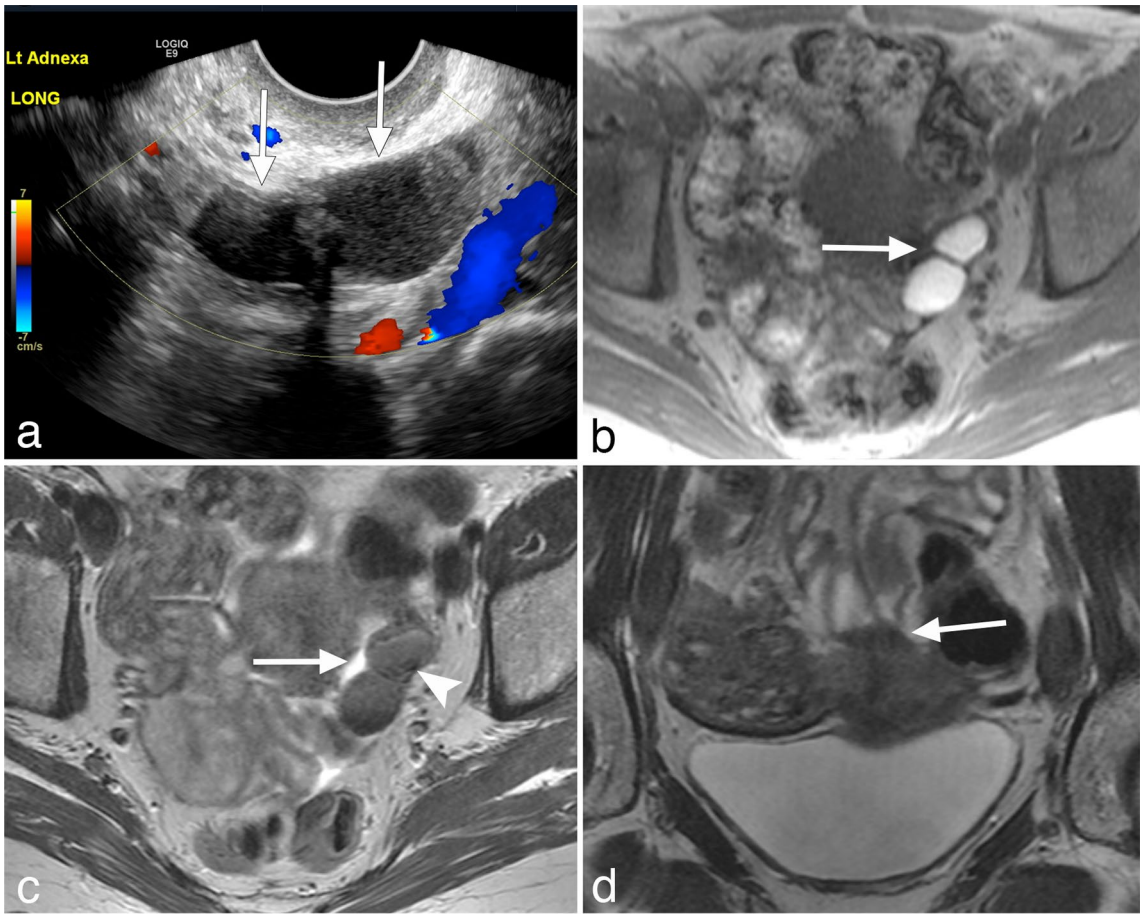

Fig. 3 Post-menopausal 49-year-old female with chronic but worsening pelvic pain newly diagnosed with endometriosis. **a** Transvaginal ultrasound demonstrates two adjacent cystic structures in the left adnexa with diffuse low-level internal echogenicity and no internal blood flow on color Doppler (arrows). Axial T1-weighted, fat-saturated (**b**) and T2-weighted (**c**) MR images demonstrate T1 hyperintensity and corresponding T2 shading in the cystic lesions, consistent with endometriomas (arrows). A "T2 dark spot" characteristic of endometrioma is also present in the more anterior lesion (**c**, arrowhead). **d** Coronal T2-weighted image demonstrates T2 hypointense fibrotic endometriosis along the posterior aspect of the uterus (arrow)

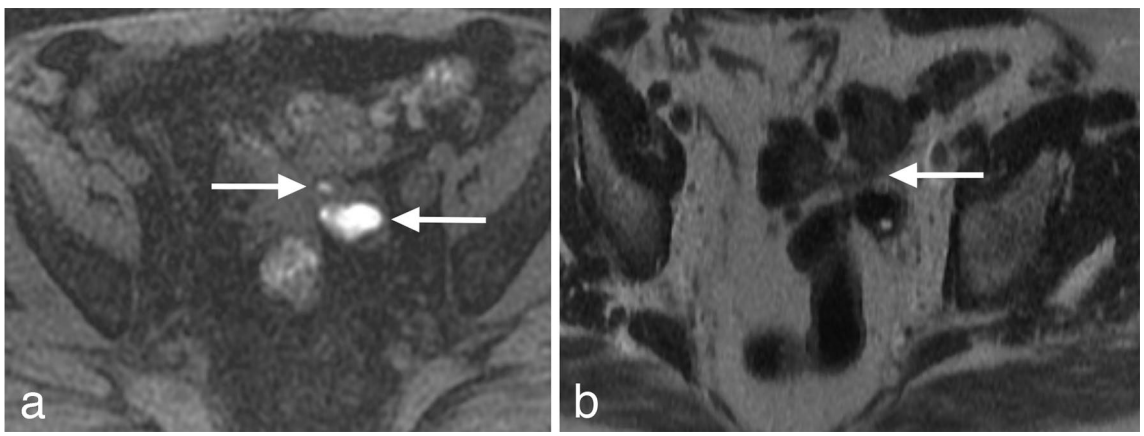

Fig. 4 Postmenopausal 64-year-old female with left ovarian remnant endometriomas incidentally discovered on an abdominopelvic MRI obtained for staging a clear cell carcinoma of the kidney. The patient had a hysterectomy and bilateral salpingo-oophorectomy at age 35 for endometriosis. **a** Axial T1-weighted, fat-saturated image demonstrates two T1 hyperintense ovarian endometriomas within the left ovarian remnant (arrows). **b** T2-weighted axial image demonstrates hypointense fibrotic bands between the remnant and the sigmoid colon (arrow)

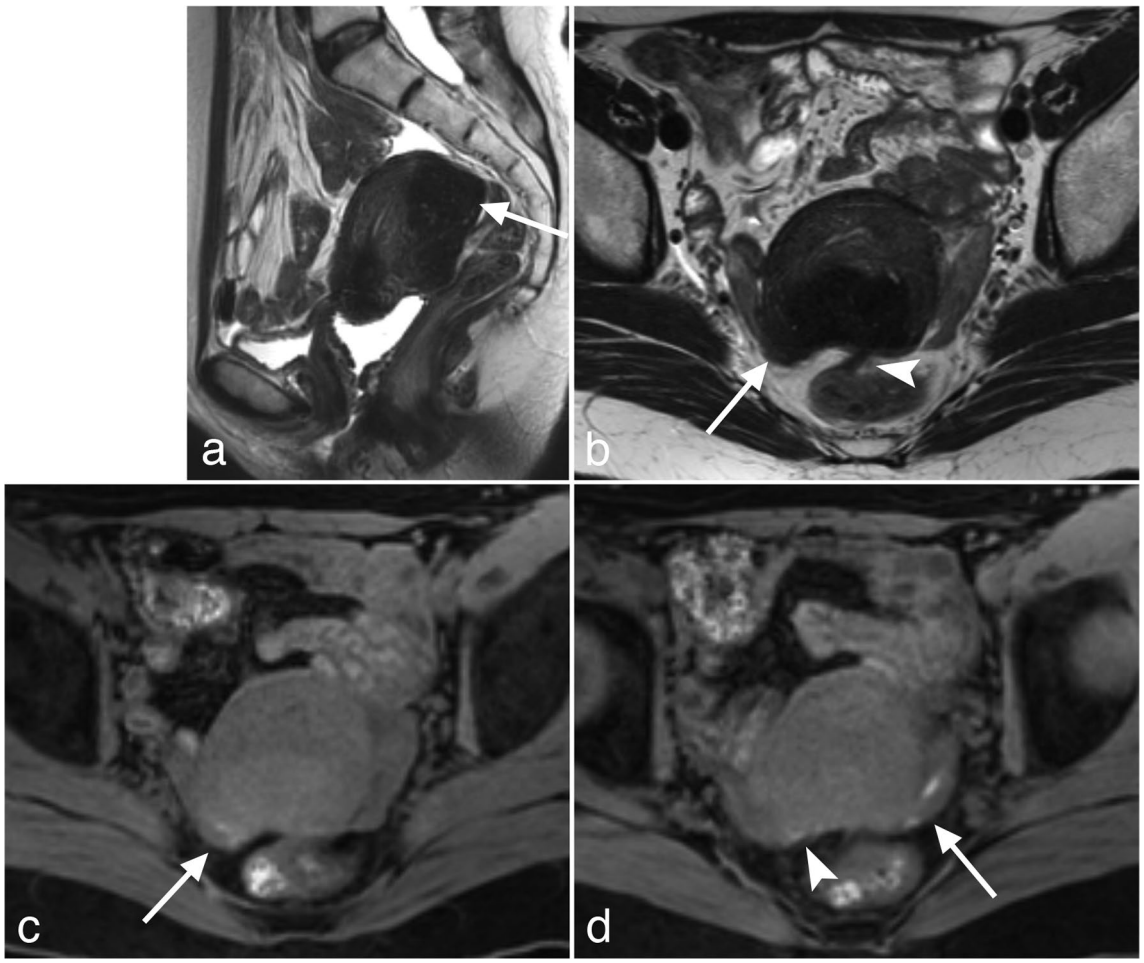

Fig. 5 Postmenopausal 56-year-old female diagnosed with invasive endometriosis in the posterior cul-de-sac. She had amenorrhea for 3 years, was not on hormone replacement therapy, and presented with a long history of pelvic pain and dysmenorrhea. Physical exam revealed a globular, slightly enlarged, retroverted uterus. Sagittal **a** and axial **b** T2-weighted MR images demonstrate a large T2 hypointense mass-like area involving the posterior aspect of the uterus (arrows). Note the distension of the vagina, achieved with sterile sonographic gel. This process was initially mistaken for a uterine fibroid, but given the punctate T2 hyperintense glandular foci, the lack of involvement of the junctional zone, and the non-rounded, more contour-conforming shape, it is consistent with invasive endometriosis. Note the small band of fibrosis extending posteriorly from the surface of the invasive endometriosis to the anterior rectal wall (**b**, arrowhead). **c**, **d** Axial T1-weighted, fat-saturated images demonstrate tiny T1 hyperintense endometriomas in the ovaries (arrows), both of which are drawn posteromedially toward the invasive endometriosis. Several additional tiny T1 hyperintense hemorrhagic foci can be seen along the posterior serosa of the uterus (**d**, arrowhead)

of a "T2 dark spot" can also help differentiate an endometrioma from a hemorrhagic ovarian cyst [33].

Superficial endometriosis may be visualized laparoscopically as red, clear, vesicular, black, or blue lesions along the peritoneum and serosal surfaces of the pelvic organs. These tiny plaque-like lesions are not identifiable on MRI unless they have associated T1 hyperintensity from hemorrhage; thus, MRI cannot adequately screen for superficial pelvic endometriosis. Deeply infiltrating endometriosis (DIE) describes subperitoneal invasion by endometriotic lesions exceeding 5 mm in depth. These endometriotic deposits respond to hormonal stimulation, resulting in a variable amount of cyclic bleeding and a variable inflammatory response and fibrous reaction [34]. Patients with DIE may have more severe symptoms which can vary with location, depth, and extent of disease. DIE typically appears as solid nodules, plaque-like thickening, or stellate soft tissue with intermediate T1 signal intensity and low T2 signal intensity. These lesions will variably demonstrate T2 hyperintense foci which correspond to the dilated ectopic endometrial glands and will have variable T1 signal intensity, enhancement, and signal on diffusion weighted images. Small hyperintense foci on fat-saturated T1-weighted images correspond with sites of hemorrhage, and occasionally fluid–fluid levels may be present if bleeding was recent. The presence of these T1/T2 hyperintense

foci within a solid mass can help increase the specificity of the diagnosis of endometriosis.

The most common site of DIE is the posterior cul-de-sac. MRI plays a key role in identifying an obliterated cul-de-sac which develops as a result of adhesion formation between the posterior aspect of the uterus/cervix and surrounding structures, including the anterior rectum [35]. The presence of an obliterated cul-de-sac may shroud additional underlying disease at the time of laparoscopy; thus, MRI is critical to define extent of disease in these patients. MRI also helps alert the surgeon to potential bowel involvement, most commonly in the anterior rectosigmoid wall, and Radiologists can help with surgical planning by determining the depth of bowel wall infiltration, the length of the bowel involved, and by determining the location of the bowel involvement relative to the anus. The classic "mushroom cap" on T2-weighted images is a specific sign of deep invasion of the bowel wall [36]. This MRI sign describes the appearance of a hypointense fibrotic implant along the bowel wall with associated hypertrophic muscularis propria and hyperintense, edematous, and curvilinear overlying mucosal/submucosal layers on the luminal side. In the case of circumferential or near circumferential involvement, or increasing depth of invasion, the bowel may become obstructed. Identifying the involvement of rectum or other pelvic organs on MRI can aid in preoperatively assembling an appropriate multidisciplinary team if other surgical subspecialties, such as colorectal surgery or urology, will be required for optimal treatment. Spiculated and band-like hypointense adhesions from endometriosis can also fixate pelvic organs, resulting in angulation of bowel and displacement of pelvic organs. In advanced posterior cul-de-sac disease, endometriosis and adhesions may result in the "kissing ovaries" configuration, characterized by ovaries which are displaced posteriorly and medially toward one another and toward the diseased uterosacral ligaments and torus uterine [37]. Additionally, the presence of hematosalpinx, even if an isolated finding, should raise the suspicion for endometriosis [38].

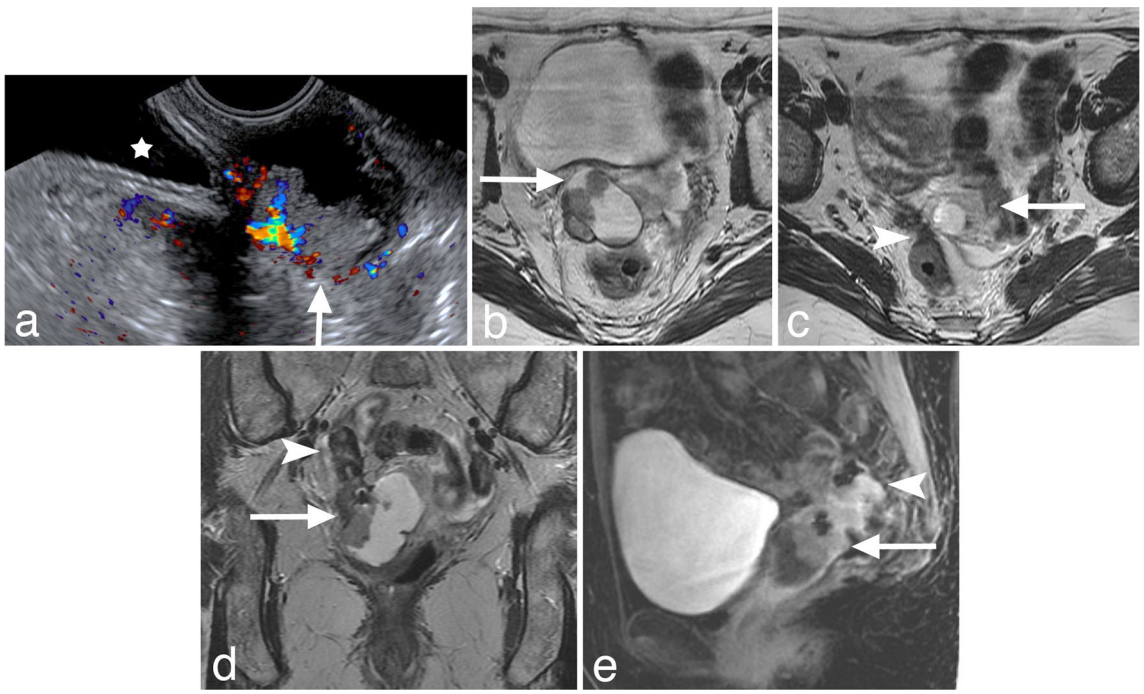

Fig. 6 Postmenopausal 55-year-old diagnosed with endometriosis-associated malignancy. At 32, she had a hysterectomy for menorrhagia and dysmenorrhea secondary to transmural adenomyosis. Superficial endometriosis was found at that time. She remained asymptomatic for many years but subsequently presented with dyspareunia and 2 weeks of spontaneous vaginal bleeding, pelvic cramping, and urinary urgency. On physical exam, the right upper vagina was attenuated by a firm, fixed mass. **a** Longitudinal transvaginal ultrasound image to the right of midline demonstrates a mixed solid and cystic mass with internal vascularity at the vaginal cuff (arrow). Neither ovary was visualized at ultrasound. Note the close proximity with the urinary bladder (star). **b**, **c** Axial T2-weighted MR images demonstrate the mixed cystic and solid lesion (arrows) at and above the vaginal cuff. **c** also shows nodular tethering to the sigmoid colon (arrowhead). **d** Coronal T2-weighted MR image demonstrates the soft tissue component (arrow) extending to involve the right ovary (arrowhead). **e** Sagittal postcontrast T1-weighted MR image demonstrates enhancement of the soft tissue component of the mass (arrow) with extension posteriorly into the presacral space involving the bowel (arrowhead). At surgery, the patient was found to have a 2.3-cm tumor involving the vaginal apex, the surface of the right ovary, and invading through the muscularis propria of the upper rectum. Pathology revealed moderately differentiated endometrioid adenocarcinoma originating in endometriosis

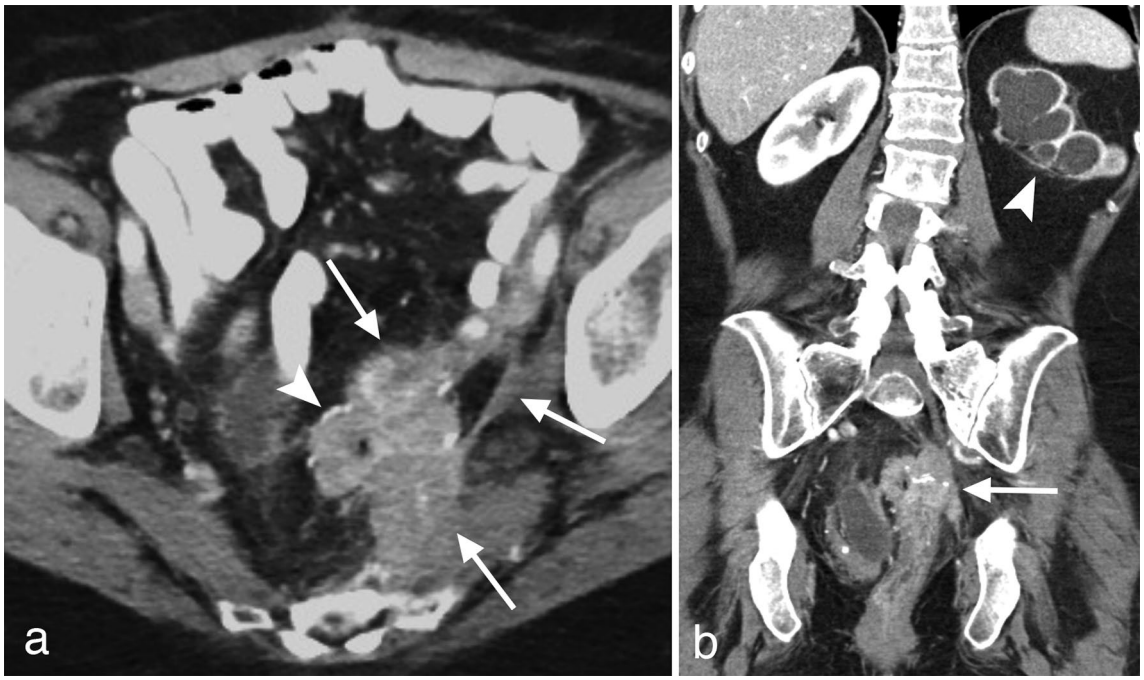

Fig. 7 64-year-old woman diagnosed with recurrent endometriosis-associated endometrioid and clear cell carcinoma. Many years prior, she had a hysterectomy due to an abnormal PAP smear. There was no mention of pelvic endometriosis at that time. At age 54, she presented with abdominal pain and left ureteral obstruction secondary to a left pelvic mass, which was found to be endometriosis-associated malignancy. She was treated with surgical resection and chemoradiation. Now, 10 years later, she presented with pelvic pain secondary to recurrent malignancy. **a** Axial postcontrast CT image demonstrates a large, enhancing recurrent malignancy (arrows) in the left pelvis involving the left presacral space, left piriformis muscle, left pelvic sidewall, and the sigmoid colon. Note the surgical anastomosis in the sigmoid colon from a prior segmental resection (arrowhead). **b** Coronal postcontrast CT image shows the mass invading the sigmoid colon at the anastomosis and invading the left pelvic sidewall (arrow). The mass encases the left ureter, resulting in obstruction and high-grade hydronephrosis (arrowhead)

Endometriosis-associated malignancy

Endometriosis is associated with an increased risk of ovarian cancer, specifically clear cell, endometrioid, and low-grade serous types [5, 39]. Increasing age, such as in the postmenopausal population, is also a risk factor for malignancy; thus, it is important to assess for the possibility of cancer in patients who present with possible postmenopausal endometriosis. In endometriosis-related ovarian cancer, symptoms may include early satiety, abdominal bloating or pressure, and increasing abdominal girth. Persons with advanced disease may present with bowel obstruction or pleural effusion. Endometriosis-related malignancy has also been reported arising from deeply infiltrative lesions in the bowel [21, 24] and may present with obstruction or bleeding.

Imaging features of endometriosis-associated malignancy are similar to those with non-endometriosis-related malignancy (Figs. 6 and 7). On MRI, it often has an intermediate T2 signal, with early avid enhancement and restricted diffusion regardless of where it occurs. Specific considerations in the evaluation of endometriosis-associated ovarian carcinoma include postcontrast T1-weighted images with fat suppression and subtraction to assess for enhancing mural nodules or septations in the setting of T1 hyperintense endometriomas. These nodules may show color Doppler flow on ultrasound. Restricted diffusion can be helpful when it corresponds to concerning nodules or septations; however, it can occur in benign endometriomas secondary to blood products [40]. Also helpful, but non-specific, is the loss of T2 shading that can occur in malignant endometriomas which is thought to be the result of increasing fluid produced by the tumor [41]. Extra-ovarian endometriosis-associated malignancy may have an infiltrative appearance, and benign variants, such as polypoid endometriosis, may mimic features of malignancy, as shown in case 8 (Fig. 8). The T2 hyperintense "cap" of the mushroom in bowel invasive endometriosis is often lost when there is mucosal invasion and the T2 hypointense fibromuscular stroma may appear more intermediate in signal. Cystic or glandular element can remain in the setting of malignant transformation, and often benign and malignant endometriosis co-exists whether it is in the abdominal wall, pelvis, abdomen, or involving nerves [42].

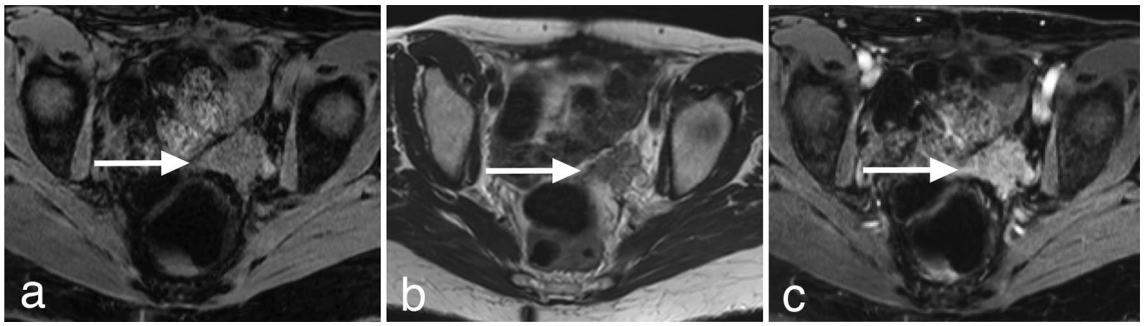

Fig. 8 57-year-old female diagnosed with polypoid endometriosis. She had a remote history of multiple surgical interventions for chronic pelvic pain and dyspareunia secondary to endometriosis, including hysterectomy and bilateral oophorectomy, multiple bowel resections, and ureteral reimplantation. She presented with recurrent pelvic pain and dyspareunia after many symptom-free years. Axial MR images demonstrate an intermediate T1 (**a**) and T2 (**b**) signal intensity mass (arrows) with avid contrast enhancement (**c,** arrow) in the left side of the pelvis and involving the left side of the vaginal cuff. Because of the concern for malignancy, the patient underwent exploratory laparotomy with partial vaginectomy and resection of the pelvic mass. Pathology revealed polypoid endometriosis

Management

Given the potential risk of malignancy in this population, treatment of postmenopausal endometriosis is often surgical. If initially detected on US, consideration should be given to obtaining a pelvic MRI to further assess disease severity, to evaluate for the presence of deep infiltrative endometriosis, and to help guide surgical planning. If there are obvious features concerning for malignancy on imaging, surgery should be performed by a surgeon with training to perform appropriate staging, if indicated. In cases where imaging features are reassuring and most consistent with benign pathology, medical management could be considered, particularly in patients who are poor surgical candidates. If on hormone replacement therapy, increasing the ratio of progestin to estrogen, or discontinuation of the estrogen component, are possible options for management in symptomatic postmenopausal endometriosis. In those receiving tamoxifen, transition to an aromatase inhibitor could be considered. Even in persons not taking tamoxifen, an aromatase inhibitor may be an effective treatment option for suppression of extra-ovarian estrogen production, a main source of endogenous estrogen in postmenopausal women [43].

In patients who do not elect to undergo surgical intervention for their endometriosis, there are limited guidelines available to direct providers on how to appropriately provide ongoing surveillance, specifically regarding imaging. In patients with previously identified endometrioma on imaging who do not undergo surgical excision, the Society of Radiologists in Ultrasound recommends a minimum of one pelvic ultrasound per year to monitor the increases in lesion size or evidence of other worrisome changes over time since an estimated 1% of endometriomas undergoes malignant degeneration [44]. In patients with previously identified deeply infiltrative endometriosis, there is thought to be a similar elevated risk of malignancy; however, no guidelines are currently available for follow-up imaging, and practice varies among providers. Given the risk for malignant degeneration, one could consider an initial follow-up MRI in 6 months, followed by individualized intervals depending on the results. If minimal change is noted, one could consider repeat imaging in 2–3 years for follow-up. If there is evidence of change in size or features of the lesions present at 6 months, continued close surveillance with repeat MRI in 6–12 months could be performed. Discontinuation of imaging surveillance of endometriomas and deeply infiltrative endometriosis should be individualized to the patient based on their goals and overall health status.

Conclusion

Postmenopausal endometriosis is a relatively uncommon clinical entity that can be incidentally found or present with a variety of different symptoms and in different locations in the body. Risk factors include prior history suggestive of endometriosis, hormone replacement therapy, tamoxifen use, and other conditions resulting in increased estrogen exposure, such as obesity. Assessment for possible malignancy should be performed, as this population is at elevated risk. Treatment is generally surgical; however, it is reasonable to consider medical management in some patients. Guidelines for surveillance in patients undergoing conservative therapy are lacking. Future research is needed to determine the optimal follow-up imaging modality and interval. Studies should also be directed at defining the prevalence of endometriosis-associated malignancies associated with deep infiltrative disease, which is likely under-recognized at this time, particularly with reference to bowel endometriosis. The role of Radiologists going forward is to consider endometriosis

as a diagnosis in the postmenopausal population, recommend appropriate exams such as MRI and US, and suggest endometriosis-associated malignancies when appropriate, based on classic morphologic features.

Acknowledgements The authors wish to thank Sonia Watson, PhD, and Desiree J. Lanzino, PT, PhD, for their help in editing the manuscript.

Funding No sources of funding were utilized to conduct this review.

Compliance with ethical standards

Conflict of interest No conflicts of interest to disclose.

References

1. Bulun SE (2009) Endometriosis. The New England journal of medicine 360 (3):268-279. doi:https://doi.org/10.1056/NEJMra0804690
2. Falcone T, Flyckt R (2018) Clinical Management of Endometriosis. Obstetrics and gynecology 131 (3):557-571. doi:https://doi.org/10.1097/aog.0000000000002469
3. Bendon CL, Becker CM (2012) Potential mechanisms of postmenopausal endometriosis. Maturitas 72 (3):214-219. doi:https://doi.org/10.1016/j.maturitas.2012.04.010
4. Brosens I, Puttemans P, Benagiano G (2013) Endometriosis: a life cycle approach? American journal of obstetrics and gynecology 209 (4):307-316. doi:https://doi.org/10.1016/j.ajog.2013.03.009
5. Streuli I, Gaitzsch H, Wenger JM, Petignat P (2017) Endometriosis after menopause: physiopathology and management of an uncommon condition. Climacteric : the journal of the International Menopause Society 20 (2):138-143. doi:https://doi.org/10.1080/13697137.2017.1284781
6. Gemmell LC, Webster KE, Kirtley S, Vincent K, Zondervan KT, Becker CM (2017) The management of menopause in women with a history of endometriosis: a systematic review. Human reproduction update 23 (4):481-500. doi:https://doi.org/10.1093/humupd/dmx011
7. Cohen I (2004) Endometrial pathologies associated with postmenopausal tamoxifen treatment. Gynecologic oncology 94 (2):256-266. doi:https://doi.org/10.1016/j.ygyno.2004.03.048
8. Mirkin S, Archer DF, Taylor HS, Pickar JH, Komm BS (2014) Differential effects of menopausal therapies on the endometrium. Menopause (New York, NY) 21 (8):899-908. doi:https://doi.org/10.1097/gme.0000000000000186
9. Bese T, Simsek Y, Bese N, Ilvan S, Arvas M (2003) Extensive pelvic endometriosis with malignant change in tamoxifen-treated postmenopausal women. International journal of gynecological cancer : official journal of the International Gynecological Cancer Society 13 (3):376-380
10. Buckley CH (1990) Tamoxifen and endometriosis. Case report. British journal of obstetrics and gynaecology 97 (7):645-646. doi:https://doi.org/10.1111/j.1471-0528.1990.tb02557.x
11. Cohen I, Altaras MM, Lew S, Tepper R, Beyth Y, Ben-Baruch G (1994) Ovarian endometrioid carcinoma and endometriosis developing in a postmenopausal breast cancer patient during tamoxifen therapy: a case report and review of the literature. Gynecologic oncology 55 (3 Pt 1):443-447. doi:https://doi.org/10.1006/gyno.1994.1319
12. Cohen I, Beyth Y, Shapira J, Tepper R, Fishman A, Cordoba M, Bernheim J, Yigael D, Altaras MM (1997) High frequency of adenomyosis in postmenopausal breast cancer patients treated with tamoxifen. Gynecologic and obstetric investigation 44 (3):200-205. doi:https://doi.org/10.1159/000291520
13. Ford MR, Turner MJ, Wood C, Soutter WP (1988) Endometriosis developing during tamoxifen therapy. American journal of obstetrics and gynecology 158 (5):1119. doi:https://doi.org/10.1016/0002-9378(88)90233-5
14. de Almeida Asencio F, Ribeiro HA, Ayrosa Ribeiro P, Malzoni M, Adamyan L, Ussia A, Gomel V, Martin DC, Koninckx PR (2019) Symptomatic endometriosis developing several years after menopause in the absence of increased circulating estrogen concentrations: a systematic review and seven case reports. Gynecol Surg 16 (1):3. doi:https://doi.org/10.1186/s10397-019-1056-x
15. Bulun SE, Yang S, Fang Z, Gurates B, Tamura M, Sebastian S (2002) Estrogen production and metabolism in endometriosis. Annals of the New York Academy of Sciences 955:75-85; discussion 86-78, 396-406. doi:https://doi.org/10.1111/j.1749-6632.2002.tb02767.x
16. Morotti M, Remorgida V, Venturini PL, Ferrero S (2012) Endometriosis in menopause: a single institution experience. Archives of gynecology and obstetrics 286 (6):1571-1575. doi:https://doi.org/10.1007/s00404-012-2473-5
17. Sun PR, Leng JH, Jia SZ, Lang JH (2013) Postmenopausal endometriosis: a retrospective analysis of 69 patients during a 20-year period. Chinese medical journal 126 (23):4588-4589
18. Dick AL, Lang DW, Bergman RT, Bhatnagar BN, Selvaggi FP (1973) Postmenopausal endometriosis with ureteral obstruction. British journal of urology 45 (2):153-155. doi:https://doi.org/10.1111/j.1464-410x.1973.tb12132.x
19. Maeda T, Uchida Y, Nakajima F (2009) Vesical endometriosis following the menopause. International urogynecology journal and pelvic floor dysfunction 20 (12):1515-1517. doi:https://doi.org/10.1007/s00192-009-0925-7
20. Thylan S (1995) Re: Renal and diaphragmatic endometriosis de novo associated with hormone replacement therapy. The Journal of urology 154 (3):1143. doi:https://doi.org/10.1016/s0022-5347(01)67004-6
21. Andriola V, Battaglia M, Ditonno P, Fiore MG, De Fazio M, Memeo R, Altomare DF (2016) The unexpected conundrum of endometrioid carcinoma in deep rectal endometriosis arising 11 years after total hysterectomy bilateral salpingo-oophorectomy. International journal of colorectal disease 31 (2):475-477. doi:https://doi.org/10.1007/s00384-015-2188-8
22. Deval B, Rafii A, Felce Dachez M, Kermanash R, Levardon M (2002) Sigmoid endometriosis in a postmenopausal woman. American journal of obstetrics and gynecology 187 (6):1723-1725. doi:https://doi.org/10.1067/mob.2002.128394
23. Izuishi K, Sano T, Shiota A, Mori H, Ebara K (2015) Small bowel obstruction caused by endometriosis in a postmenopausal woman. Asian journal of endoscopic surgery 8 (2):205-208. doi:https://doi.org/10.1111/ases.12154
24. Magtibay PM, Heppell J, Leslie KO (2001) Endometriosis-associated invasive adenocarcinoma involving the rectum in a postmenopausal female: report of a case. Diseases of the colon and rectum 44 (10):1530-1533. doi:https://doi.org/10.1007/bf02234612
25. Popoutchi P, dos Reis Lemos CR, Silva JC, Nogueira AA, Feres O, Ribeiro da Rocha JJ (2008) Postmenopausal intestinal obstructive endometriosis: case report and review of the literature. Sao Paulo medical journal = Revista paulista de medicina 126 (3):190-193
26. Mohamed AAA, Selim YARM, Arif MA, Albroumi SA (2016) Gastric wall endometriosis in a postmenopausal woman. Egypt J Radiol Nucl Med 47 (4):1783-1786. doi:https://doi.org/10.1016/j.ejrnm.2016.08.005

27. Threadcraft M, Fouad L, Bruce A, Nakhleh R, Dinh T (2017) Endometriosis in a Postmenopausal Patient Presenting as an Erythematous Vaginal Plaque. Journal of minimally invasive gynecology 24 (4):516-517. doi:https://doi.org/10.1016/j.jmig.2016.07.017
28. Turkyilmaz E, Cinkaya A, Secen EI, Kayacetin S, Yavuz Avsar AF (2016) Postmenopausal Vaginal Endometriotic Cyst: A Case Report. J Clin Anal Med 7 (4):563-566. doi: https://doi.org/10.4328/jcam.4401
29. Cameron M, Westwell S, Subramanian A, Ramesar K, Howlett D (2017) Postmenopausal Cutaneous Endometriosis: Mimicking Breast Metastasis. The breast journal 23 (3):356-358. doi:https://doi.org/10.1111/tbj.12742
30. Choi SW, Lee HN, Kang SJ, Kim HO (1999) A case of cutaneous endometriosis developed in postmenopausal woman receiving hormonal replacement. Journal of the American Academy of Dermatology 41 (2 Pt 2):327-329. doi:https://doi.org/10.1016/s0190-9622(99)70377-4
31. Flyckt R, Lyden S, Roma A, Falcone T (2011) Post-menopausal endometriosis with inferior vena cava invasion requiring surgical management. Human reproduction (Oxford, England) 26 (10):2709-2712. doi:https://doi.org/10.1093/humrep/der260
32. Glastonbury CM (2002) The shading sign. Radiology 224 (1):199-201. doi:https://doi.org/10.1148/radiol.2241010361
33. Corwin MG, EO; Lamba R; Wilson, M; McGahan JP (2014) Differentiation of Ovarian Endometriomas from Hemorrhagic Cysts at MR Imaging: Utility of the T2 Dark Spot Sign. Radiology 271 (1):126-132. doi:https://doi.org/10.1148/radiol.13131394
34. Coutinho AB, LK; Pires, CE; Junqueira, F; Lima, CM; Coutinho, E; Domingues, MA; Domingues, RC; Marchiori, E (2011) MR imaging in deep pelvic endometriosis: a pictoral essay. Radiographics 31 (2):549-567. doi:https://doi.org/10.1148/rg.312105144
35. 35. Macario S, Chassang M, Novellas S, Baudin G, Delotte J, Toullalan O, Chevallier P (2012) The value of pelvic MRI in the diagnosis of posterior cul-de-sac obliteration in cases of deep pelvic endometriosis. AJR Am J Roentgenol 199 (6):1410-1415. doi:https://doi.org/10.2214/AJR.11.7898
36. Yoon JH, Choi D, Jang KT, Kim CK, Kim H, Lee SJ, Chun HK, Lee WY, Yun SH (2010) Deep rectosigmoid endometriosis: "mushroom cap" sign on T2-weighted MR imaging. Abdom Imaging 35 (6):726-731. doi:https://doi.org/10.1007/s00261-010-9643-3
37. Ghezzi F, Raio L, Cromi A, Duwe DG, Beretta P, Buttarelli M, Mueller MD (2005) "Kissing ovaries": a sonographic sign of moderate to severe endometriosis. Fertil Steril 83 (1):143-147. doi:https://doi.org/10.1016/j.fertnstert.2004.05.094
38. Outwater ES, ES; Chiowanich, P; Kilger, AM; Dunton, CJ; Talerman, A (1998) Dilated Fallopian Tubes: MR Imaging Characteristics. Radiology 208:463-469. doi:https://doi.org/10.1148/radiology.208.2.9680577
39. Li J, Liu R, Tang S, Feng F, Liu C, Wang L, Zhao W, Zhang T, Yao Y, Wang X, Sun C (2019) Impact of endometriosis on risk of ovarian, endometrial and cervical cancers: a meta-analysis. Archives of gynecology and obstetrics 299 (1):35-46. doi:https://doi.org/10.1007/s00404-018-4968-1
40. Dhanda ST, M; Kerkar, R; Jagmohan, P (2014) Diffusion-weighted imaging of gynecologic tumors: diagnostic pearls and potential pitfalls. Radiographics 34 (5):1393-1416. doi:https://doi.org/10.1148/rg.345130131
41. Tanaka YO, Okada S, Yagi T, Satoh T, Oki A, Tsunoda H, Yoshikawa H (2010) MRI of endometriotic cysts in association with ovarian carcinoma. AJR Am J Roentgenol 194 (2):355-361. doi:https://doi.org/10.2214/AJR.09.2985
42. Robinson KA, Menias CO, Chen L, Schiappacasse G, Shaaban AM, Caserta MP, Elsayes KM, VanBuren WM, Bolan CW (2019) Understanding malignant transformation of endometriosis: imaging features with pathologic correlation. Abdom Radiol (NY). doi:https://doi.org/10.1007/s00261-019-01914-7
43. Polyzos NP, Fatemi HM, Zavos A, Grimbizis G, Kyrou D, Velasco JG, Devroey P, Tarlatzis B, Papanikolaou EG (2011) Aromatase inhibitors in post-menopausal endometriosis. Reproductive biology and endocrinology : RB&E 9:90. doi:https://doi.org/10.1186/1477-7827-9-90
44. Levine D, Brown DL, Andreotti RF, Benacerraf B, Benson CB, Brewster WR, Coleman B, Depriest P, Doubilet PM, Goldstein SR, Hamper UM, Hecht JL, Horrow M, Hur HC, Marnach M, Patel MD, Platt LD, Puscheck E, Smith-Bindman R (2010) Management of asymptomatic ovarian and other adnexal cysts imaged at US: Society of Radiologists in Ultrasound Consensus Conference Statement. Radiology 256 (3):943-954. doi:https://doi.org/10.1148/radiol.10100213