An update on adenomyosis


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Abstract

Adenomyosis is a common benign uterine pathology that is defined by the presence of islands of ectopic endometrial tissue within the myometrium. It is asymptomatic in one third of cases, but when there are clinical signs they remain non-specific. It can often be misdiagnosed on sonography as it may be taken to be multiple uterine leiomyomata or endometrial thickening, both of which have a different prognosis and treatment. Adenomyosis is often associated with hormone-dependent pelvic lesions (myoma, endometriosis, or endometrial hyperplasia). It is less commonly connected to infertility or obstetrical complications and indeed any direct relationship remains controversial. The purpose of imaging is to make the diagnosis, to determine the extent of spread (focal or diffuse, superficial or deep adenomyosis, adenomyoma), and to check whether there is any associated disease, in particular endometriosis. The aim of this article is to provide assistance in recognising adenomyosis on imaging and to identify the pathologies that are commonly associated with it in order to guide the therapeutic management of symptomatic patients. Pelvic ultrasonography is the first line investigation. Sonohysterography can assist with diagnosis in some cases (pseudothickening of the endometrium seen on sonography). MRI may be used in addition to sonography to back up the diagnosis and to look for any associated disease.

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Abbreviations: FS, Fat-Sat; DPE, deep pelvic endometriosis; MRI, magnetic resonance imaging; CT, computed tomography; US, ultrasound; JZ, junctional zone; IL, interleukin; MAP, medically assisted procreation; ETOP, elective termination of pregnancy; IUP, intrauterine pregnancy; HIFU, high-intensity focused ultrasound; GnRH, gonadotropin releasing hormone; NSAID, non-steroidal anti-inflammatory drug; HST, hysterectomy.

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Background

Adenomyosis is a common condition, usually affecting multiparous women over the age of forty. On histology, it is characterised by the presence of ectopic endometrial mucosa within the myometrium (invagination of endometrium in the myometrium at a depth of at least 2.5 mm below the basal layer of the endometrium) that leads to hypertrophy of the smooth muscle, which confirms the diagnosis [1,2]. It can be either focal (one or several foci in the myometrium; these are nodular in the case of adenomyoma) or diffuse (numerous foci spread throughout the myometrium) and it is often asymmetric, predominating in the posterior uterine corpus. Making the distinction between the superficial (simple thickening of the junctional zone seen on MRI or lesions that do not extend beyond one third of the depth of the myometrium) and deep forms (when lesions penetrate deeper then one third of the way into the myometrium) is more controversial. Adenomyosis is often associated with hormone-dependent pelvic lesions (myoma, deep pelvic endometriosis [DPE], polyps, or endometrial hyperplasia). These could be cases of "external" adenomyosis, connected to the lesions of deep pelvic endometriosis invading the myometrium from the outside inwards. This phenomenon has already been described for lesions found in the rectovaginal septum, and these are histologically similar to adenomyosis [3,4]. Recent MRI studies have shown that adenomyosis and endometriosis are probably two entities governed by a single pathophysiological and/or genetic process. In terms of the severity of DPE and adenomyosis lesions when they are possibly associated, a particular correlation has been found for lesions of the rectovaginal septum [5,6].

Its role in infertility is still debated: the association between adenomyosis and sterility is poorly understood (occurring in between 1 and 14% of the cases in the literature) but the frequency of infertility has not been assessed in large studies [7]. The theory of the pathophysiology is one of uterine hypermobility (irregular and uncontrolled) that inhibits the transfer of spermatosa from the uterus to the fallopian tubes, the mobility of the fertilised oocyte, normal implantation of the embryo, and the ability of trophoblasts to penetrate the myometrium (preventing the development of a functional placenta). Another hypothesis put forward has been junctional zone dysfunction. In parallel, while the relationship between deep pelvic endometriosis and fertility is recognised by the majority of authors, its mechanism remains poorly elucidated. Theories include irregular ovulation, transport abnormalities in the fallopian tubes, and abnormalities with fertilisation or implantation [7]. Adenomyosis is thought to be a significant factor for infertility in endometriosis, and its role in this is probably due to changes in uterine transport of spermatosa [6]. This was the conclusion of a study carried out on 34 patients after laparoscopic segmental colorectal resection for endometriosis [8,9]. Wang et al. [10] underlined the uncertain relationship between infertility and focal adenomyosis or adenomyoma, while noting that severe endometriosis does reduce the chances of successful treatment with medically assisted procreation (MAP) techniques. Today, there is no consensus on treatment for patients who wish to become pregnant, especially in terms of restoring their fertility. Various treatment options are used: hormone treatment, focal surgical resection, embolisation of uterine arteries, and other experimental techniques.

A number of factors that encourage adenomyosis to develop have been found: the main factor is having had more than one pregnancy, but there is also spontaneous miscarriage, ETOP, curettage, hysteroscopic resection of the endometrium, uterine myomectomy, caesarean section, and taking Tamoxifen®. It is thought that there could be a genetic predisposition. Tobacco use is thought to be a protective factor (probably due to reduced plasma levels of oestrogens).

Aside from the hyperoestrogenaemia that has been demonstrated in ectopic endometrial tissue, a correlation has been made between adenomyosis and dysfunctional regulation or abnormally high quantities of some HLA2-type immune response proteins, without necessarily implying that there is a causative relationship: interleukin-18 (IL-18) [11] and leukaemia inhibitory factor (LIF) [12]. It has been suggested that abnormal secretion of LIF-6 [13] from endometrial stromal cells may play a role in forming the ectopic islands of endometrial tissue in adenomyosis. Overexpression of cyclooxygenase-2 [14] (COX-2) also seems to have a significant role in the pathogenesis and it could be a potential target for the treatment or prevention of adenomyosis.

Clinically, one third of patients are asymptomatic and when there are functional signs, they remain non-specific, consisting mainly of menometorrhagia, dysmenorrhoea, and pelvic pain. Superficial adenomyosis is thought to be more commonly expressed as minimally incapacitating abnormal uterine bleeding, while the deep form is thought to involve more severe pain, disabling menometorrhagia, or even dyspareunia. The gynaecological examination will look for a globular uterus that is painful on mobilisation. In practical terms, when there is clinical suspicion of adenomyosis, an ultrasound scan of the pelvis should be carried out first, and this may be enough to arrive at a positive diagnosis. If doubt persists over diagnosis (myoma? Endometrial thickening?) or an associated disease is also suspected (myoma, endometriosis), a pelvic MRI scan and sonohysterography may be required to complete investigations. Hysteroscopy is no longer considered to be indicated in this condition.

Imaging features of adenomyosis

Sonography

The direct signs are:

- anechoic subendometrial microcysts in the myometrium (around 2–4 mm in diameter) that can be distinguished from a vascular image on Doppler sonography because they are not vascularised (pathognomonic sign). This cystic space is the direct sign of the adenomyosis lesion and it corresponds to ectopic dilated endometrial glands positioned within the myometrium that may or may not be haemorrhagic (if they are, their contents show greater echogenicity) during and immediately post-menstruation;
- the myometrium has a non-homogenous appearance combining hyperechoic linear striations within the myometrium, small hyperechoic subendometrial nodules, pseudonodular hypoechoic zones with indistinct contours
Figure 1. Sonographic signs of adenomyosis: a, b: globular uterus and asymmetric myometrial walls; c: thickening of the interface between the myometrium and endometrium; d: subendometrial hyperechoic striations.

Figure 2. Pelvic sonogram, axial (a) and sagittal views (b) of the uterus. Significant thickening of the endometrial/myometrial junction and subendometrial microcysts pointing to diffuse adenomyosis. Thin endometrium (5 mm).
Figure 3. Differences in vascularisation between adenomyosis and leiomyoma demonstrated on power Doppler sonography: vascularisation crosses the hypertrophied myometrium in diffuse adenomyosis (a) as opposed to the peripheral vascular projections in leiomyoma (b).

Figure 4. Diffuse adenomyosis on sonography: comparison between transabdominal and transvaginal sonography in a 43-year old patient presenting menometrorrhagia: a, b: transabdominal: globular uterus (measuring 10 × 8 × 10 cm), uncertainty over the presence of leiomyoma; c, d: transvaginal approach in the same patient: at least one subendometrial microcyst, heterogeneous myometrium, hyperechoic linear striations within the myometrium, and asymmetric myometrial walls.
and no mass effect on the endometrium, and a poorly defined or thickened endometrial-myometrial junction.

The indirect signs are those that result from reactive hypertrophy of the smooth muscle fibres of the myometrium around the ectopic endometrial glands and they suggest that adenomyosis is likely. The sonographer should look for:
- the classic sign of a uterus that is enlarged, soft, and rounded but has regular contours (as opposed to the uterus with multiple leiomyomata, which has relatively irregular contours that are deformed by the fibroids);
- asymmetrical myometrial walls (a sign of muscular hypertrophy);
- linear pattern of vascularisation on Doppler sonography, crossing the myometrium within the adenomyosis lesions (as opposed to the peripheral and central vascularisation seen in leiomyoma) (Figs. 1—3).

The limitations of both transabdominal and transvaginal (Fig. 4) for positively diagnosing adenomyosis have been studied [15]. Taking as transabdominal sonography sign: a large uterus regular, possibly asymmetric, more or less heterogeneous, without individualized myoma, with possible intra myometrial cystic images, the transabdominal sonography had a poor sensitivity of 30% but a good specificity of 97%. The combination of both approaches increased the accuracy diagnosis. The presence of subendometrial cysts was the most specific sonographic sign for diagnosis. Myometrial linear striations were correlated to hypertrophy of the myometrium. The transvaginal sonography may also be limited in the case of large uterus, heterogeneous and hypoechoic (uterine leiomyomata and/or adenomyosis) default in depth-study; the use of MRI appears to be useful.

Differential diagnosis:
- multiple uterine leiomyomata is the main differential diagnosis for diffuse adenomyosis on sonography (Table 1);
- the use of Doppler sonography means that external myometrial vessels are not mistaken for the cystic spaces seen in adenomyosis;
- cystic glandular endometrial hypertrophy is more difficult to differentiate from the subendometrial cysts seen in adenomyosis using sonography: in these cases MRI may prove to be useful. It is worth noting that Tamoxifen® encourages three types of endometrial change: thickening due to cancer, adenomyosis and cystic glandular hypertrophy (Fig. 9);
- endometrial thickening can also mimic diffuse adenomyosis. If this is suspected, sonohysterography may be useful for diagnosis (Fig. 6).

### Sonohysterography

This technique uses a saline-infusion to opacify the subendometrial cysts and it can be used to differentiate focal or superficial adenomyosis from a pseudothickening of the endometrium seen on sonography (this is suspicious, for example, for carcinoma in a postmenopausal patient) (Fig. 6). If adenomyosis is suspected on sonohysterography, the diagnosis is confirmed on MRI in 96% of cases [16]. This examination performs by demonstrating that there is continuity between the subendometrial cystic spaces characteristic of adenomyosis and the endometrial cavity [17]. The superficial sites remain in continuity with the cavity while the deep sites have usually lost all continuity with the uterine cavity.

### Hysteroscopy

This is no longer used for positive diagnosis because it is neither sensitive nor specific. When used for primary or secondary investigations into fertility in addition to a pelvic ultrasound scan, findings can nonetheless be made of spicules measuring 1—4 mm in length, extending from the endometrium towards the myometrium, or a rigid or dilated uterine horn with the "tuba erecta" image (Fig. 7).

### CT scanning

CT is not suitable for diagnosing adenomyosis. It is possible that CT could suggest this diagnosis based on finding a globular uterus. In one study, 16 patients with adenomyosis demonstrated on MRI had needed a CT scan for another reason, and on a second reading it was found that out of eight patients, the diagnosis could have been made on the CT scan but it had not been put forward on the initial assessment [18].

### MRI [19]

Pelvic MRI is superior to transvaginal sonography in terms of specificity and sensitivity for both focal and diffuse adenomyosis [20—22]. There is some overlap between the signs found on MRI and on sonography [23,24] (Table 2):
- globular uterus with regular contours
- asymmetrical thickening of the myometrial walls (more common at the posterior wall)
- thickening of the JZ ≥ 12 mm [25] (Fig. 8). Thresholds for measurement have been proposed (≤ 8 mm or ≥ 12 mm with a zone of uncertainty between 8 and 12 mm) but none is able to confirm or exclude adenomyosis (studies show varying sensitivity and specificity) [25]
- greatest JZ thickness to total myometrium ratio > 40—50% (connected to muscular hypertrophy)
- foci of high signal intensity running alongside the endometrium on T2 and sometimes also T1-weighted images that persist on Fat-Sat (F5). These correspond

| Table 1 Making the differential diagnosis between adenomyosis and leiomyoma on sonography. |
|----------------------------------|---------------------------------|
| **Leiomyoma**                    | ± Associated with adenomyosis   |
| **Defined margins**              | Poorly defined margins         |
| Round                            | Variable shape                 |
| Mass effect                      | No mass effect                 |
| Calcifications ±                 | No calcification               |
| Attenuation with edge shadowing  | Multiple foci of attenuation   |
| Peripheral vascularisation       | Rectilinear vascularisation    |
|                                  | pattern crossing the           |
|                                  | hypertrophied                  |
|                                  | myometrium                    |

Figure 5. Glandular and cystic hypertrophy of the endometrium (a pitfall encountered on sonography): a–c: features on transabdominal and then transvaginal sonography: diffuse adenomyosis? d, e: magnetic resonance imaging of the same patient, sagittal and axial T2-weighted sequences; f: plus gadolinium-enhanced sagittal T1-weighted sequence.

to the dilated fluid-filled and possibly haemorrhagic endometrial glands (Figs. 9 and 10).

There are pitfalls on MRI that it is important to be aware of because JZ thickness varies [25,26]:

- depending on the menstrual cycle (thickest between day 8 and day 16, variable during menstruation);
- during the menopause: thinner or possibly absent;
- during pregnancy: thins until it disappears;
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Figure 6. Sonohysterography carried out after sonography in a 54-year old patient presenting postmenopausal metrorrhagia: a–c: globular uterus, subendometrial cysts, and suspected endometrial thickening (15 mm) on sonography: endometrial cancer? d–f: sonohysterography, catheter balloon at the cervix and intracavitary injection of saline solution: opacification of the subendometrial cysts due to superficial adenomyosis, thin atrophic endometrium (< 5 mm).

- with age: thickens up to the age of 50 and then thins;
- with use of the contraceptive pill and GnRH agonists: thins (Fig. 11);
- with myometrial contractions: pseudothickening of the JZ (Figs. 12 and 13).

The MRI must therefore be carried out by experienced radiologists when the patient is not menstruating. Rapid T2 sequences can be repeated and should be correlated with other views in case of uterine contractions. The standard MRI protocol combines sagittal and axial views on T2-weighted and spin echo sequences, and T1-weighted axial sequences with and without fat-saturation. In the rare cases when embolisation is planned, the examination can be completed by dynamic gadolinium-enhanced sagittal sequences every 5 to 20 seconds for 2 to 4 minutes and later views taken after the gadolinium injection.
It is important to also look for the commonly associated conditions on MRI, making use of clinical information:
• adenomyosis and leiomyomata (Fig. 14);
• adenomyosis and deep pelvic endometriosis (Figs. 15–17);
• differential diagnoses:
  ○ diffuse thickening of the JZ during menstruation or of hormonal origin,
  ○ myoma: round, well-circumscribed, low T2 signal intensity. MRI is more sensitive than sonography for identifying adenomyosis with concomitant myomata [20],
  ○ myometrial contractions: transient, wedge-shaped areas of low T2 signal intensity,
  ○ metastases (rare): low T2 signal intensity, with a globular uterus, primary breast or gastrointestinal cancer,
  ○ cystic endometrial hyperplasia,
  ○ endometrial carcinoma: comparison with biopsies is required because it can be difficult to judge the depth of the myometrial invasion on MRI when there are associated adenomyosis lesions (Fig. 18). An endometrioid adenocarcinoma that extends into an underlying adenomyosis is thought to be more invasive, and the probable mechanism of this is an increase in the surface of the interface with the adjacent myometrium. These tumours are not only thought to be likely to invade the myometrium to a greater extent, but they also encourage invasion into the deepest half of the myometrium (>50%), and beyond this threshold there is an increased risk of lymph node metastases [27,28],

<table>
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<tr>
<th>Table 2</th>
<th>Standardised criteria for the positive diagnosis of adenomyosis on magnetic resonance imaging.</th>
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<tr>
<td>JZ ≥ 12 mm</td>
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<td>Max ratio &gt; 40–50%</td>
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<td>Foci of high T2 signal intensity ± high T1 FS signal intensity</td>
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<td>JZ: junctional zone; FS: Fat-Sat.</td>
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Figure 9. Correlations between sonography and magnetic resonance imaging: globular uterus due to diffuse adenomyosis. Subendometrial cysts presenting high signal intensity on T2-weighted sequences, some of which are hemorrhagic exhibiting high signal intensity on T1-weighted FS sequences: a, b: pelvic sonogram; c: sagittal T2-weighted magnetic resonance imaging sequence; d: axial T2-weighted sequence; e: Axial T1-weighted FS sequence.
Figure 10. Deep diffuse adenomyosis on magnetic resonance imaging: subendometrial fluid-filled cysts exhibiting high signal intensity on T2-weighted images, non-haemorrhagic: a: sagittal T2-weighted sequences; b, c: axial T2-weighted sequences; d: sagittal T1-weighted sequence; e: gadolinium-enhanced axial T1-weighted FS sequence.
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○ abscess secondary to adenomyosis mimicking a malignant tumour [29].

Specific cases [30]

Simple adenomyoma

This is a localised nodular lesion that is often poorly defined with extensive muscle response that may be intramyometrial, subserosal, and possibly even intracavitary. It consists of a local coalescence of adenomyotic glands and can lead to a mass effect on the endometrium. On MRI, it produces relatively non-homogenous low signal intensity on T2-weighted images. It remains difficult to differentiate from leiomyoma but if foci of high signal intensity are seen on T2-weighted images, then this is suggestive of adenomyoma (Fig. 19). Both adenomyoma and leiomyoma can cause a significant mass effect on the uterine cavity and can even produce intracavitary growth (Figs. 20 and 21). This lesion is not encapsulated, meaning that there is no straightforward surgical cleavage plane.

Figure 11. On gonadotropin releasing hormone, the endometrium appears atrophied and the uterus showing menopausal features exhibits overall low signal intensity on T2-weighted sequences: junctional zone cannot be measured.

Figure 12. Uterine contractions (can be mistaken for junctional zone thickening on magnetic resonance imaging): a, b: striped appearance of the myometrium with low T2 signal intensity on both sagittal and axial views; c: areas of low T2 signal intensity disappear on the ultra rapid sagittal sequence: uterine contractions.
Haemorrhagic cystic adenomyosis ("adenomyotic cyst")

Cystic adenomyosis occurs when there is bleeding into an ectopic island of endometrial glandular tissue that is totally or partially surrounded by myometrium. Images of intramyometrial cysts present slightly echogenic contents with or without a nodular appearance when there is adenomyoma. They vary in size. They are normally situated within the wall, although they may sometimes be submucosal or subserosal. On MRI, the fluid content is haemorrhagic exhibiting high signal intensity on T1-weighted images that persists on FS, while showing variable signal intensity on T2-weighted images (Figs. 22 and 23). The finding of low signal intensity on T2-weighted images from the wall combined with haemorrhagic contents is suggestive of cystic adenomyosis. A fluid-fluid level is possible.

Vascularisation

In the absence of objective published data, it seems that adenomyosis presents variable vascularisation (Figs. 19 and 20).

Decidualisation of adenomyosis lesions

This process entails an increase in pre-existing adenomyosis lesions due to pregnancy, which may be explained by the hormonal effects of pregnancy on the ectopic endometrium (Fig. 24).

Treatment

If the patient is asymptomatic no treatment is indicated. If symptoms are present, there are several potential treatment options [31,32]: medical (hormonal) or surgical treatment (conservative or radical). A treatment will be chosen based on the patient’s symptomatology and whether she is interested in future child bearing [31].

First line medical treatment

Oral progestogens such as dydrogesterone are often used to treat premenopausal menometrorrhagia that is probably caused by adenomyosis. They cause endometrial atrophy due to their anti-oestrogen action but that do not have any effect on cells involved in adenomyosis that are hormone resistant. They are an effective treatment for pain, dysmenorrhoea, and other symptoms in endometriosis but one study [33] emphasised the lack of reliable data to validate their use in adenomyosis. Functional signs reappear after six months in 30 to 50% of cases [34]. The levonorgestrel-releasing intrauterine device (Mirena®) is the only effective and well-tolerated treatment for menometrorrhagia in adenomyosis [32]: it markedly reduces uterine enlargement, pain, and the significance of dysmenorrhoea [35]. After three years, tachyphylaxis has been demonstrated and its replacement is suggested [36]. NSAIDs are also used. GnRH agonists cause hypoestrogenism and therefore reduce uterine enlargement, JZ thickness, and the endometrial deposits that cause dysmenorrhoea. They can be used preoperatively in order to make it easier to carry out resections of sites of adenomyosis when a conservative approach is used, or to reduce uterine size before hysterectomy. They can also be prescribed to women who are interested in future child bearing and they may or may not be combined with surgery (adenomyoma resection) [32]. Danazol is less commonly used because it has androgenic side effects, and no clinical studies have proved it to be effective in adenomyosis [32]. In an in vitro model [13], danazol and medroxyprogesterone acetate (MPA) reduced secretion of interleukin-6 from endometrial stromal cells.
Figure 14. Deep adenomyosis of the anterior myometrium on magnetic resonance imaging with haemorrhagic foci, associated with a number of leiomyomata (here they are intracavitary and in the wall of the posterior isthmus): a, b: sagittal T2-weighted sequence; c: axial T2-weighted sequence; d: axial T1-weighted sequence; e: axial T1-weighted FS sequence; f: gadolinium-enhanced sagittal T1-weighted sequence.

Embolisation of uterine arteries

Consideration is made of whether this procedure is indicated on a case-by-case basis. It consists of selective embolisation of a uterine artery on each side using non-calibrated microparticulate PVA (polyvinyl alcohol) or a calibrated microparticulate agent (trisacryl microspheres, 500–700 µm), with or without a gelatin sponge (Spongé® or Curaspon®) [37–39]. The clinical results show short-term resolution of symptoms (particularly for menorrhagia, but less so for pain) [40]. Medium and long-term efficacy varies depending on the study [41,42] (approximately 50% long-term success) but there is significant recurrence of symptoms after two years have passed [43]. A repeat treatment or a hysterectomy may be required. Bilateral uterine artery ligation by abdominal endoscopy seems to be less effective [32].

MRI can show post-treatment changes (Figs. 25 and 26): reduced uterine size, decreased JZ thickness, and full
Figure 15. Deep diffuse adenomyosis on magnetic resonance imaging (thickened junctional zone and foci of high T2 signal intensity) associated with deep pelvic endometriosis of the torus uterinus invading the myometrium (foci of high signal intensity on T1-weighted FS images): a, b: sagittal and axial T2-weighted sequences; c, d: axial T1-weighted FS sequences.

Figure 16. Focal adenomyosis (normal junctional zone on the left image/thickened on the right with foci of high T2 signal intensity). Lesions associated with anterior and posterior DPE with foci of high T2 signal intensity at the uterine surface (“external adenomyosis”): a: Sagittal T2-weighted sequence; b: Coronal T2-weighted sequence.

or partial infarction of the lesions with non-vascularised areas of low signal intensity on T2-weighted images [44,45]. There is not usually any fall in the maximum JZ-myometrium ratio. However, only limited numbers of cases have been described combining both adenomyosis and leiomyoma, so the improvement in symptoms may be attributable to treatment of either one of these abnormalities.
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Figure 17. Deep diffuse adenomyosis that is predominantly posterior with foci of high T2 signal intensity associated with interstitial leiomyomata of the anterior and right posterolateral uterine corpus, deep pelvic endometriosis of the torus uterinus, and bilateral endometriotic cysts: a: sagittal T2-weighted sequence; b: axial T2-weighted sequence.

Figure 18. Adenomyosis and endometrial cancer: does the adenomyosis limit the spread of the cancer within the myometrium? a, b: sagittal T2-weighted magnetic resonance imaging sequence: thickened junctional zone, subendometrial microcysts; c: sagittal sonographic view: is there invasion of the anterior subendometrial microcyst?
Figure 19. Adenomyoma of the right uterine corpus with a nodular appearance and subendometrial microcysts. Note the hypervascularity shown on MRI (d): a, b: sagittal and axial T2-weighted sequences; c, d: contrast-enhanced sagittal and axial T1-weighted Fat-Sat sequences; e: transvaginal sonogram of the same patient. Note the microcysts.

Surgical treatment

Surgery may be radical (hysterectomy: HST) or conservative (hysteroscopy and endometrial curettage, adenomyomectomy, resection of the site of adenomyosis with or without treatment for associated endometriosis). The age of the patient and whether she is interested in future child bearing will influence the type of surgery chosen. The choice between a total and subtotal HST is made based principally on the health of the cervix, pouch of Douglas, and rectovaginal septum [32]. The decision on whether to preserve the adnexal structures depends on age, and the presence of endometrioma or associated deep peritoneal endometriosis.
Figure 20. Initial suspicion of endometrial cancer on computed tomography (CT) (a). On sonography (b, c) and magnetic resonance imaging (MRI) (d), it in fact appears to be an anterior adenomyoma: nodular appearance within the myometrium, isosignal intensity from the junctional zone with foci of high T2 signal intensity, and a moderate mass effect on the cavity. Note the hypervascularity shown on MRI (e): a: sagittal plane CT; b, c: sonography views of the uterus; d: sagittal T2-weighted MRI sequences; e: plus contrast-enhanced T1-weighted sequences.

Experimental US guided treatments

One case of focal adenomyosis treated with ultrasound-guided aspiration and sclerotherapy with intracavitary alcohol instillation [46] has been reported for a recurrent symptomatic lesion.

The feasibility of laparoscopic high-intensity focused ultrasound (HIFU) ablation has been studied in seven patients with focal adenomyosis [47]. Anatomical pathology investigations confirmed that thermal ablation of the sites of adenomyosis had been successful, with healthy margins.
Figure 21. Intracavitary polypoid adenomyoma on sonography and magnetic resonance imaging: a, b: sagittal and axial sonography views of the uterus; c, d: sagittal and coronal T2-weighted sequences; e, f: sagittal T1-weighted Fat-Sat sequences before and after contrast enhancement.
Figure 22. Haemorrhagic cystic adenomyoma on magnetic resonance imaging: peripheral halo of low T2 signal intensity and central haemorrhagic fluid contents exhibiting high signal intensity on T1-weighted images that persist with fat-saturation: a: sagittal T2-weighted view; b: sagittal T1-weighted view; c: sagittal T1-weighted Fat-Sat view.

Figure 23. Haemorrhagic cystic adenomyoma of the left uterine horn on a bicornate uterus with a double cervix seen on magnetic resonance imaging: a: coronal T2-weighted view; b: axial T1-weighted view; c: axial T1-weighted Fat-Sat view.
Figure 24. Decidualisation of adenomyosis lesions: a: right uterine horn, sagittal sonographic view; endometrium in a pregnant woman and adenomyosis lesions. Correlates on magnetic resonance imaging to: adenomyosis of the right uterine horn and intrauterine pregnancy in the rudimentary left horn; b: sagittal T2-weighted sequence; c: axial T2-weighted sequence.

Figure 25. Adenomyosis before and after embolisation shown on two successive monitoring magnetic resonance imaging scans, sagittal T2-weighted sequences (a–c): reduced thickness of the junctional zone and zone of post-embolisation change in the uterine fundus exhibiting low T2 signal intensity.
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Conclusion

Adenomyosis is a common condition that is asymptomatic in one third of cases, although in others it can cause dysmenorrhea and/or menometrorrhagia.

The purpose of imaging is to make the diagnosis, to determine the extent of disease spread, and to look for any associated pathologies, especially endometriosis in an infertile patient.

A transvaginal pelvic sonogram is the first line investigation. Sonohysterography may be useful for diagnosis in some specific kinds of cases. MRI is the second line investigation to carry out, and it offers the best performance when there is any doubt over diagnosis and in terms of looking for any of the pathologies associated with adenomyosis.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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rectovaginal septum are three different entities. Ref Gynecol Obstet 1995;3:121–3.


