Uterine sarcoma subtypes and their differentiation from non-sarcomatous entities using MRI.

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Learning objectives

- Review the main subtypes of uterine sarcomas.
- Review imaging features, particularly with regards MRI using imaging from our regional Gynaecological Cancer Network.
- Discuss ways in which the above can be differentiated from benign or non-sarcomatous entities using MRI from the literature.
Background

The preoperative diagnosis of uterine sarcoma can be extremely challenging. These tumours are uncommon malignancies and each of the sarcoma subtypes display features common to both benign neoplasms and non-sarcomatous malignancies. The main recognised subtypes are leiomyosarcoma (LMS), endometrial stromal sarcoma (ESS) and high grade undifferentiated uterine sarcoma (HGUS), the latter historically classed as a high grade ESS. Although carcinosarcoma (previously known as malignant mixed Mullerian tumour or MMMT) is separately classified as a high-grade endometrial carcinoma, it is included in this review as imaging features often overlap and mimic uterine sarcoma. Each of these entities will be separately described in depth in this overview. Smooth muscle tumours of uncertain malignant potential (STUMP) lesions are also discussed as they may enter the radiological differential diagnosis when imaging the above conditions. There are few publications on the topic of uterine sarcoma within the general radiological literature so this overview aims to describe the clinical and radiological features of these rare tumour subtypes, while raising radiological awareness of the more recent separate FIGO sarcoma classification.

Historically, patients with significant vaginal bleeding and/or an enlarging uterine mass underwent hysterectomy and, only at the time of pathological diagnosis, was it clear that the patient had an underlying sarcoma (1). This is still a common occurrence and many patients have only ultrasound assessment prior to surgery and require post-operative staging with CT. There are now many non-surgical treatments available as an alternative to invasive surgery for presumed fibroid disease. These include hormonal therapy, endometrial ablation and interventional radiology procedures such as fibroid embolisation. Clearly these treatments do not provide histology. Furthermore, clinical features commonly thought to be indicative or worrying for underlying sarcoma when clinical examination was the mainstay of assessment, have also been shown to occur in benign conditions - particularly presentation with a rapidly enlarging uterus in a woman of reproductive age in whom pregnancy has been excluded. A literature review of 26 studies in 1994 demonstrated that a history of rapid uterine growth was observed in only 2.6% of patients diagnosed with sarcoma (2). Further evidence of this poor correlation was presented in a paper which showed fibroids increased and decreased in size, at varying rates in the same woman when followed up with imaging at regular intervals (3). During minimally invasive operations to remove presumed fibroids, there is the concern that in the presence of an undiagnosed sarcoma, seeding or spread to the peritoneal cavity can occur during morcellation of the "fibroid" (4). Furthermore, due to the heterogeneity of this type of tumour, frozen sections analysed at time of surgery are not reliable in diagnosing sarcoma (1). Hysteroscopic sampling also has varying success in diagnosing uterine sarcoma, with reports ranging from 38-64% (5-7). There is therefore increasing reliance on pre-treatment imaging to differentiate between these uncommon
sarcomas and other pathology. It is also important to differentiate between ESS/HGUS and endometrial cancer as the FIGO staging, patterns of spread and prognosis greatly differ. The separate FIGO sarcoma staging is not widely publicised in the radiological literature and is therefore outlined in this review.

The investigation of women with possible uterine malignancy begins with history and clinical examination, followed by ultrasound (trans-abdominal and/or trans-vaginal) and hysteroscopy with biopsy. MRI is then commonly performed and the radiological features of these individual tumour subtypes will be discussed in this overview. The review is illustrated using imaging from patients treated within our regional Gynaecological Cancer Network.
Findings and procedure details

MRI Protocol for gynaecological disorders

As uterine sarcomas are uncommon, the aim is to try to differentiate these tumours from other far commoner pathology, such as fibroids in the cases of LMS or endometrial carcinoma in the case of ESS, HGUS or carcinosarcoma. Therefore, MRI protocols used in many institutions are based on those for endometrial carcinoma or fibroid assessment.

Most of the reviewed literature is in agreement that patients should void one hour prior to examination and that peristaltic activity from bowel should be reduced to a minimum, by administering an anti-spasmodic such as Buscopan, if there is no contraindication to administration (8-11).

At least two T2 weighted sequences should be performed, usually in the sagittal and axial oblique planes (8-11). A T1W sequence of the pelvis allows assessment of pelvic lymphadenopathy and bone marrow signal (10). This is often performed in the axial plane.

Diffusion weighted sequences display differing signal intensity based on the Brownian movement of water. They are increasingly used routinely in gynaecological MRI. The b-values can be used to calculate the ADC (apparent diffusion coefficient) value for a given pixel. Low ADC values indicate increased risk of malignancy (12). Cellularity is directly proportional to restricted diffusion which lends itself to cancer imaging as high cellularity is often seen in solid tumours. Care must be taken however to assess the degree of necrosis in a tumour as this may give a false negative value of restriction (12).

Contrast enhancement with a gadolinium-based agent is key to assessing the degree of myometrial invasion for tumours which involve or expand the endometrial cavity. It can aid the differentiation of ESS or carcinosarcoma from endometrial carcinoma. There is conflicting advice in the literature on optimum contrast protocols. Dynamic contrast enhancement (DCE) is suggested by many, as early enhancement allows visualization of the subendometrial zone (corresponding to the inner junctional zone) for assessment of early myometrial invasion, the equilibrium phase assesses for deep myometrial invasion and delayed phase allows cervical stromal assessment (10). However, the European Society of Urogenital Imaging suggested that a single post contrast sequence at 2 min 30 sec after contrast administration allowed improved spatial resolution which was lost in favour of temporal resolution in DCE (13). Some centres also routinely use DCE in the workup of patients for potential fibroid embolisation.
Endometrial Stromal Sarcoma/HGUS

Background

ESS represents less than 10% of all uterine sarcomas and was previously staged by the same criteria as other tumours of the uterine corpus (14). The need for revision of staging for uterine sarcomas was recognised recently and in 2009, a new FIGO staging for ESS was formulated and published (15). See figure 1 below.

ESS/HGUS typically presents with abnormal vaginal bleeding and/or abdominal pain (8) and on imaging appears as a polypoidal mass in the endometrial cavity (8,16). Alternatively ESS can display myometrial invasion to such a degree that it can mimic an intramural mass such as intramural myoma (17). Pathologically, the tumour cells resemble endometrial stroma in the proliferative phase (14). Previously, it was subclassified as low grade or high grade ESS depending on the mitotic figures present in the pathology specimen (8). Low grade ESS occurs in a younger age group (mean age at diagnosis of 39 years) and tends to infiltrate the myometrium with worm like projections and lymphatic permeation, contrary to the very well-demarcated outline seen in similar benign pathology such as endometrial stromal nodule (14). High grade ESS tends to be far more destructive and infiltrative than low grade with only a 50% 5-year survival rate and a tendency to recur at a late stage after treatment (mean 65 months for stage I disease and mean 9 months for stage III/IV disease) (18). Peritoneal seeding is commonly present at the time of diagnosis and metastases occur most commonly in the peritoneum and lungs. More recent literature has suggested that due to their widely disparate behaviour, low and high grade ESS should be thought of as completely different entities (19).

Tumours with similar histological characteristics that were previously classified as high grade ESS are high-grade undifferentiated uterine sarcomas (HGUS). They tend to exhibit both stromal and smooth muscle components on pathological analysis. There is now the consensus that HGUS is a separate entity to ESS but very little in the way of literature currently exists due to changes in diagnostic criteria and rarity of the disease. What is clear however is that the prognosis for HGUS is poor with a mean survival of only 11.8 months and with 58% of patient having distant metastases at diagnosis (20). Survival rates are lower than in leiomyosarcoma, carcinosarcoma and high grade ESS (20). Imaging features for ESS discussed below are similar for HGUS. Treatment includes hysterectomy and bilateral salpingo-oophorectomy with peritoneal exploration, omentectomy and lymphadenectomy. Despite lymphatic involvement being common, lymphadenectomy does not seem to improve survival (19). Adjuvant chemotherapy and/or hormone treatment can be considered depending on hormone receptors present within the tumour (19). In particular, low grade ESS are commonly ER or PR positive, and status should routinely be performed. There is conflicting advice in the literature on the benefit of adjuvant chemotherapy as standard.
**Imaging findings**

On basic MRI sequences, the soft polypoid mass, usually within the endometrial cavity, returns heterogeneous but mainly high T2-weighted signal (8,14,17) (figure 2). Fronds of high signal extend into the myometrium in low-grade ESS, which has been shown to correspond pathologically with lymphatic extension (14). High-grade tumour can have the appearance of invasive endometrial carcinoma (EC) with high/intermediate signal involving a proportion of the myometrium with loss of the normal low signal junctional zone. The tumour can be invasive to such an extent that the myometrial component predominates despite originating from the endometrial cavity (figure 3). ESS is more likely to present with a larger tumour mass than seen in EC. It has been shown to more commonly contain haemorrhage and necrosis, have irregular margins with nodular areas and nodular intramyometrial extension (14). Whilst EC can also be irregular in outline, nodular areas around the tumour margin are uncommon (14). Contrast enhancement can also help to separate ESS from EC in that many cases of ESS enhance to a similar degree as the normal myometrium, whereas EC very rarely enhances as much as the adjacent myometrium on delayed phase imaging (10,14). Difficulty can occur in cases of less hypervascular ESS although dynamic contrast enhancement is superior to a single delayed phase sequence in these cases (14). A further diagnostically helpful MRI finding in ESS is that of low signal bands on T2-weighted sequences, thought to represent bundles of preserved myometrial fibres separated by tumour and these correspond with the appearances seen on pathological specimens (18). ESS and HGUS show restricted diffusion. Restricted diffusion however cannot be used to confidently differentiate high-grade sarcomas (both high grade ESS and HGUS) from EC as restriction is seen in all of these conditions (21). A table summarising features of ESS/HGUS and how these compare with EC, carcinosarcoma and leiomyosarcoma are shown in figure 4.

**Leiomyosarcoma**

**Background**

Leiomyosarcoma (LMS) constitute one third of all uterine sarcomas and can rarely occur in pre-existing fibroids or, most commonly, de novo (17). Most are thought to arise de novo, with only 0.1-0.8% of benign fibroids undergoing malignant transformation (17). The prevalence of sarcoma in pathological specimens from patients with hysterectomy for presumed benign fibroid disease is thought to be in the order of 0.2-0.5% (2,5,22). The 5-year survival rates for leiomyosarcoma in the literature vary greatly from 25-75% (23). Spread is to the adjacent myometrium, adjacent pelvic organs and via haematogenous spread to distant organs, primarily the lungs. As for ESS, the FIGO staging protocol for LMS was revised in 2009 and is shown in figure 5.
Treatment includes hysterectomy with or without bilateral salpingo-oophorectomy, depending on age of patient. Adjuvant systemic therapy has shown little proven additional benefit (23). Lymphadenectomy is not indicated unless nodes are clinically involved as only 7% of cases in a large case series by Major et al had positive nodes at time of lymphadenectomy (24).

**Imaging findings**

The main differential when reviewing MRI imaging for LMS is the differentiation from a leiomyoma (fibroid). LMS on MRI appears as a myometrial mass which tends to express heterogeneous T2 hyperintensity with areas of necrosis and haemorrhage. Haemorrhage will usually demonstrate high T1 signal in addition to the high T2 signal (17). Irregular outline was initially thought to indicate malignancy but the specificity of this finding has not been proven (25). Heterogenous enhancement is present in the solid components of the sarcoma. Lack of calcification is documented in the literature as a consistent finding in LMS (19). Diffusion weighted imaging (DWI) displays high signal intensity (SI) with associated low ADC values in the sarcoma (4,12). Care must be taken not to calculate these values in necrotic or cystic areas as they may give a false negative result (12).

The differentiation from simple fibroids is usually simple enough on MRI as these are typically hypointense on T1 and T2 sequences with low SI on DWI and homogenously enhance post contrast (25,27). They also tend to be very well defined as a result of their pseudocapsule (26). However, fibroids can undergo degeneration or be of a cellular type, the latter containing smooth muscle cells packed tightly together with little in the way of intervening collagen. These behave in a similar way on MRI to LMS and differentiation is difficult and not always possible (25). Red degeneration (when the fibroid outgrows its blood supply) can have areas of haemorrhage within it but does not typically show restricted diffusion (12). Fibroids with myxoid degeneration can have increased T2 signal but in general show only minimal enhancement in contrast to very vascular LMS (25). Cystic fibroids have internal T2 hyperintensity but do not enhance. Using multivariate analysis, one study by Naggara et al has shown that the combination of low ADC values, high SI on the b1000 DWI sequence and high T2 signal have a 92.4% diagnostic accuracy for leiomyosarcoma (4). This study showed only a small overlap in ADC values with cellular fibroids. Tamai et al showed a larger overlap in ADC values between fibroid and sarcomas, however T2 signal and DWI signal intensity were lower in fibroids than sarcomas (12). There has been a large standard deviation in ADC values reported in benign fibroids due to histological variety but even degenerated fibroids have higher ADC values and lower SI on DWI than sarcomas (12). Rapid increase in size of a fibroid, although less reliable in pre-menopausal women, should raise suspicion and concern regarding underlying sarcoma in post menopausal women (1) (figure 6). Clearly, evidence of metastatic spread or invasion of adjacent organs is indicative of sarcoma, as this does not occur in fibroid disease (figure 7).
STUMP (smooth muscle tumours of uncertain malignant potential) are difficult to diagnose pre-operatively and can display features of sarcoma or of a fibroid which is cellular or undergoing degeneration. Features suggesting myxoid degeneration in particular should raise concern as these overlap with STUMP lesions (28) (figure 8). These are a heterogenous group of uterine smooth muscle tumours which are difficult to distinguish pathologically in terms of predicting clinical outcome. A study by Tanaka et al could not identify features on MRI which were indicative of STUMP lesions in their study due to significant overlap with both fibroids and leiomyosarcomas (28). Uniform, early enhancement is thought to be a useful MR characteristic in these lesions although this also occurs in fibroids (figure 9). Close follow-up following surgery is required to assess for malignant recurrence or spread in these cases.

**Carcinosarcoma**

**Background**

Previously termed malignant mixed Mullerian tumour (MMMT), carcinosarcoma is now classified as a high grade endometrial carcinoma as they have are felt to have the similar aetiological factors and it is felt that they have different biological behaviour in comparison to other uterine sarcomas (29). It is felt likely that the tumour undergoes focal clonal evolution and sarcomatous degeneration but begins as an endometrial carcinoma and is staged using the same protocol as EC. Both sarcomatous and carcinomatous elements are present in the tumour. They account for approximately 2% of uterine malignancies and up to 50% of uterine sarcomas (29). When metastasis occur, they are more commonly epithelial rather than sarcomatous.

Again, although considered very similar entities, it is important to differentiate EC from carcinosarcoma as the prognosis and survival differ greatly (30). The 5-year survival is 18-39% with many patients presenting with lymphatic / peritoneal spread and distant spread, most commonly to the lungs (27). Pre-operative diagnosis with imaging is also highly valuable because it is common for this entity to be incorrectly diagnosed by pipelle biopsy, thought to be in part due to the greater adherence of the sarcomatous portions of the tumour compared to the carcinomatous elements (30).

**Imaging features**

The typical appearance of a carcinosarcoma is a large, solid, heterogeneously hyperintense mass replacing the endometrial cavity. (29). There is commonly deep myometrial invasion (figure 10) with areas of hemorrhage and necrosis, features uncommon in EC (29). These malignancies have a tendency to occur at the uterine fundus (figure 11) although rarely can protrude through the cervical os (29). Flow voids
around the tumour are highly indicative of carcinosarcoma and again, this has not been shown to be a prominent feature in EC (30). Bharwani et al showed that 50% of carcinosarcomas displayed early contrast enhancement equal to or greater than that of the outer myometrium and 62% showed delayed enhancement (9). Again this is in comparison with EC which usually shows mild enhancement, less than that of the myometrium. However, 88% of the patients they studied could not be differentiated on imaging findings from EC. Additionally, if pelvic or retroperitoneal lymphadenopathy are present, sarcoma should be considered as this is more common than in EC.
<table>
<thead>
<tr>
<th>Stage</th>
<th>Diagnosis</th>
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<tbody>
<tr>
<td>I</td>
<td>Tumour confined to uterus</td>
</tr>
<tr>
<td>IA</td>
<td>Limited to endometrium/endocervix with no myometrial invasion</td>
</tr>
<tr>
<td>IB</td>
<td>&lt; 50% invasion of myometrium</td>
</tr>
<tr>
<td>IC</td>
<td>&gt; 50% invasion of myometrium</td>
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<tr>
<td>II</td>
<td>Tumour extends to the pelvis</td>
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<tr>
<td>IIA</td>
<td>Adnexal involvement</td>
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<tr>
<td>IIB</td>
<td>Tumour extends to extrauterine pelvic tissue</td>
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<tr>
<td>III</td>
<td>Tumour invades abdominal tissues</td>
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<td>IIIA</td>
<td>One site</td>
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<td>IIIB</td>
<td>&gt; one site</td>
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<tr>
<td>IIIC</td>
<td>Metastases to pelvic and/or para-aortic lymph nodes</td>
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<tr>
<td>IVA</td>
<td>Tumour invades bladder and/or rectum</td>
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<tr>
<td>IVB</td>
<td>Distant metastases</td>
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</tbody>
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**Fig. 1:** FIGO staging of ESS and adenosarcoma. Adapted from FIGO staging of uterine sarcomas (15).


**Fig. 2:** HGUS. (A) Sagittal T2W sequence shows heterogenous and bizarre mass replacing/expanding vagina and cervix and extending into endometrial cavity, proven pathologically to be high-grade undifferentiated uterine sarcoma following TAH and BSO.
Urinary catheter noted in situ. (B) Contrast enhanced CT of abdomen for follow up purposes performed at 5 months post surgery shows ill-defined low attenuation masses throughout liver, consistent with metastases.

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Fig. 3: HGUS. (A) Fat suppression T1W sequences with areas of high internal signal corresponding to intra-tumoural haemorrhage and (B) T2W sequences showing complete replacement of uterus by a large heterogenous mass. Exophytic component noted at uterine fundus (yellow arrow) due to serosal breach, demonstrating aggressive and invasive nature of HGUS. (C) DWI (b=800 s/mm2) and D) ADC map displaying restricted diffusion within almost the entire mass replacing uterus, including the exophytic component extending through the fundal serosal breach.
### Fig. 4: Summary of MR characteristics of ESS/HGUS v carcinosarcoma, LMS and EC.

© Adapted from information in body of text.
<table>
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<tbody>
<tr>
<td>I</td>
<td>Tumour limited to uterus</td>
</tr>
<tr>
<td>IA</td>
<td>&lt;5 cm</td>
</tr>
<tr>
<td>IB</td>
<td>&gt;5 cm</td>
</tr>
<tr>
<td>II</td>
<td>Tumour extends to pelvis</td>
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**Fig. 5:** FIGO staging of Leiomyosarcoma. Adapted from FIGO staging of uterine sarcomas (15).

Fig. 6: LMS. Sagittal T2W sequences in 62 year old patient with known multifibroid uterus. (A) Initial MRI study, and (B) repeat study performed 4 months later due to clinically noted increase in size of pelvic mass. The posterior mass (blue arrows), compressing the endometrial cavity has grown dramatically in size and contains high signal, consistent with necrosis/fluid. This was later pathologically proven to be a leiomyosarcoma. Note the anterior fibroid has reduced in size over the interval (yellow arrows). Axial (C) and coronal (D) T2W sequences performed during the second MRI study with large flow voids (red arrows) corresponding to prominent uterine vessels, around the periphery of the ill-defined, heterogenous sarcoma, illustrating the hyper-vascular nature of the tumour.

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Fig. 7: LMS. (A) T1W sequence showing high signal centrally within mass, consistent with haemorrhage (blue arrow). (B) T2W sequence shows cystic/necrotic areas within the mass and endometrial cavity compressed between mass and bladder (yellow arrow). (C) T2W axial sequence displaying heterogenous but reasonably well-defined LMS with less well-defined area along the right postero-lateral aspect of the tumour (yellow arrow). (D) Follow up CT chest showed nodule in left upper lobe (blue circle) which gradually increased in size, in keeping with a metastatic deposit.

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Fig. 8: STUMP (A) Sagittal T2W sequence and (B) post contrast T1W fat sat sequence in 54 year old patient with STUMP lesion. High T2 signal in (A) corresponds with areas of cystic change/necrosis and the lesion is well-defined peripherally. Enhancement is patchy and heterogenous and similar to a fibroid undergoing myxoid degeneration, in itself a worrying feature for STUMP.

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Fig. 9: STUMP. Well-demarcated round myometrial lesion close to cervical os, hyperintense to myometrium on T2W imaging (A), isointense on T1W sequence (B). Fat sat pre (C) and post-gadolinium (D) sequences demonstrate uniform enhancement.

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Fig. 10: Carcinosarcoma. (A) T2W and (B) 3D T1W post gadolinium sequences showing low/isointense endometrial mass invading >50% of myometrium (yellow arrows) and extending through the os, with parts of the tumour enhancing to similar degree as myometrium. C) DWI (b=800 s/mm2) and D) ADC map showing restricted diffusion in the endometrial tumour mass invading myometrium.

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Fig. 11: Carcinosarcoma. (A) Sagittal T1W fat sat and (B) post-gadolinium sequences showing haematometra (blue arrow). After contrast, the solid tumour within the superior aspect/fundal region of the retroverted uterus enhances avidly (yellow arrow).

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Conclusion

Uterine sarcomas are relatively rare with limited published radiological literature on this topic. Most series are of very limited patient numbers. On imaging, the presence of a complex uterine mass with bizarre architecture and spread out with the uterus at initial presentation should raise the suspicion of sarcoma.

Diffusion weighted imaging allows differentiation in most cases between malignant and benign entities although overlap occurs in the case of leiomyosarcoma and cellular fibroid as they are both pathologies which are highly cellular. Also, care must be taken to assess DWI in conjunction with standard anatomical MRI sequences to reduce the risk of false positive values in areas of tumour necrosis.

The differentiation between ESS/HGUS/carcinosarcoma and endometrial carcinoma can be difficult and commonly not possible prior to surgery. However, the presence of intratumoral haemorrhage and necrosis, flow voids, myometrial worm-like extension and peritumour nodules are documented as more likely in sarcomas. DWI is unlikely to be of great benefit in differentiation as malignant entities in general, whether sarcomatous or carcinomatous, tend to display restricted diffusion. The pattern of enhancement may assist differentiation, as parts of sarcomas tend to enhance at least as much as myometrium. This feature is uncommon in endometrial carcinoma.

There remain no pathognomonic MRI features for any of the subtypes of uterine sarcomas but, as discussed, certain features may raise suspicion of the diagnosis pre-operatively. In particular, great care should be taken when imaging fibroids for eligibility for fibroid embolisation. It is essential to be vigilant and highlight suspicious features within the fibroids as these patients are now commonly managed by non-invasive treatments with no means of pathological assessment.
References


