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Uterine myomas and sarcomas – clinical and ultrasound characteristics and differential diagnosis using pulsed and color Doppler techniques

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Abstract

Uterine tumors are a challenge encountered by every gynecologist in clinical practice. In the era of increasing incidence of endometrial cancer in the general population of women at reproductive age, compared to other genital malignancies, we should not forget about other tumors originating from the mucous and muscular layer of the uterus. Clear ultrasonographic differentiation of uterine tumors into benign (myomas) and malignant (sarcomas) lesions may sometimes prove impossible. Myomas, the most common uterine tumors, are characterized by discrete vascularization on color Doppler and high blood flow velocity as well as the lack of early diastolic notch on Doppler ultrasound. Sarcomas, on the other hand, show characteristic rich vascularization. Rapid tumor growth should also be noted when making the diagnosis. There are multiple known causes of uterine tumors. So far, no clear Doppler flow markers have been identified to characterize benign and malignant lesions.

Uterine myomas

Uterine myomas are the most common benign tumors of the female reproductive organs. They may affect even every fourth woman at reproductive age. It should be emphasized that uterine myomas occur most frequently in the population of peri- and postmenopausal women. They are detected in almost half of patients in their 50s[1,2]. The prevalence of myomas in the population and their health consequences make them one of the most common conditions encountered in gynecological practice.

These tumors are composed of uterine smooth muscle or vascular muscle layer, and fibrous stroma.

Table 1 shows the differentiation between myomas and uterine sarcomas.

Myomas may manifest as single or multiple lesions. Depending on their location, myomas can be classified as submucosal, intramural and subserosal. They are also rarely detected in the round, sacrouterine and broad uterine ligaments. Low-lying submucosal myomas may extend through the internal orifice of the cervix into its canal, appearing in the external orifice as so-called myomal polyps, otherwise known as nascent myomas[3,4].

Fig. 1, Fig. 2 Fig. 3, Fig. 4 and Fig, 5 show different types of uterine myomas.

In 2011, the International Federation of Obstetricians and Gynecologists (Fédération Internationale de Gynécologie et d'Obstétrique, FIGO) introduced a classification of myomas based on their location, which distinguishes eight classes (Fig. 6 and Tab. 2)[5].

Genetic factors, steroid sex hormones, as well as growth and angiogenesis factors are involved in the formation and growth of uterine myomas[6].

Risk factors for uterine myomas are well established and include African-American ethnicity, late onset of menopause,

Tab. 1. *Differentiation of uterine myomas/sarcomas*

Feature	Uterine myoma	Uterine sarcoma
Prevalence	250 : 1000 women	0.1 : 1000 women
	usually multiple tumors	single
Growth rate	slow	rapid
Age	perimenopausal and postmenopausal	50–65 years
5-year survival rate	100%	10%
The most common symptoms	heavy periods, abdominal pain, infertility	non-cyclic uterine bleeding
US characteristics:	round, solid, clearly demarcated, hyperechoic	oval, solid, heterogeneous tumors with mixed echogenicity
	tumor capsule, acoustic shadow, peripheral calcifications (usually postmenopausal)	no capsule, no acoustic shadow, no calcifications
Vascularization of the tumor	weak, peripheral, MUSA 1	strong, irregular, peripheral and central MUSA 3/4
Power Doppler	PI 0.7–0.9 RI 0.4–0.6 PSV 22.5 cm/sec.	 RI 0.37 ± 0.03 PSV 71 cm/sec.

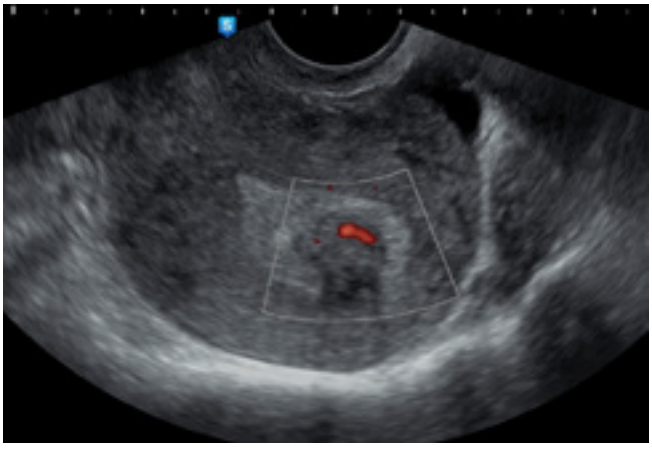

Fig. 1. *Uterine myoma. Discrete flow on color Doppler (stage 1)*

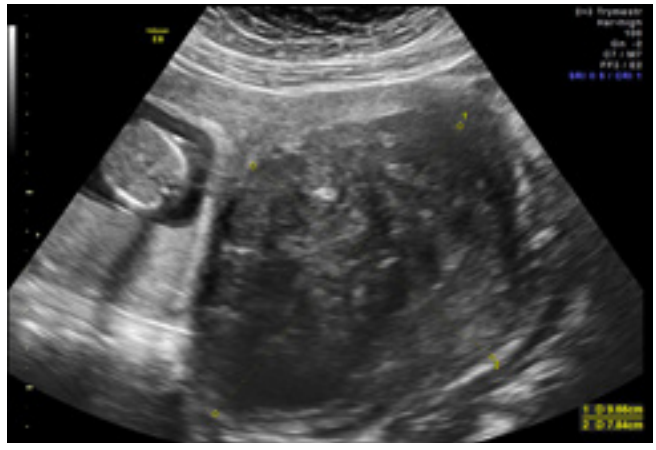

Fig. 2. *Uterine myoma at 18 weeks gestation*

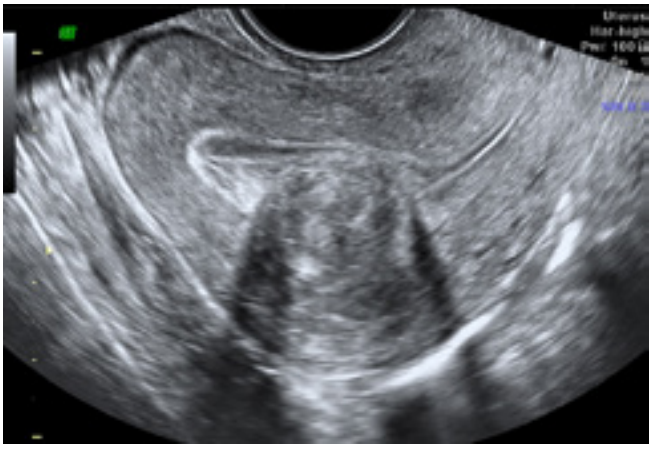

Fig. 3. *Submucosal myoma*

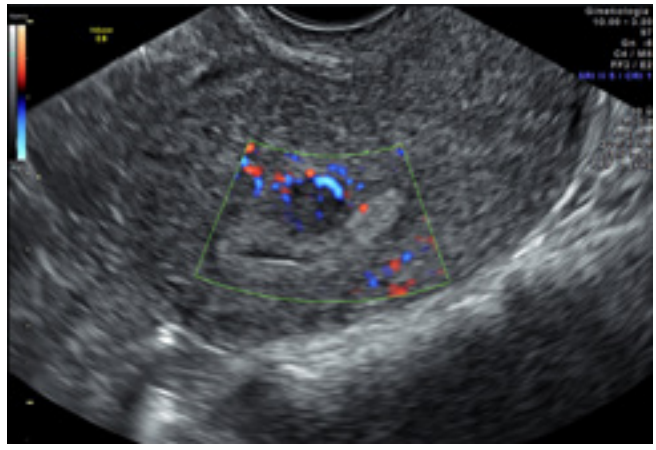

Fig. 4. *Submucosal myoma – centrally located hypoechoic lesion with abundant vascularization, grade 3 flow in color Doppler*

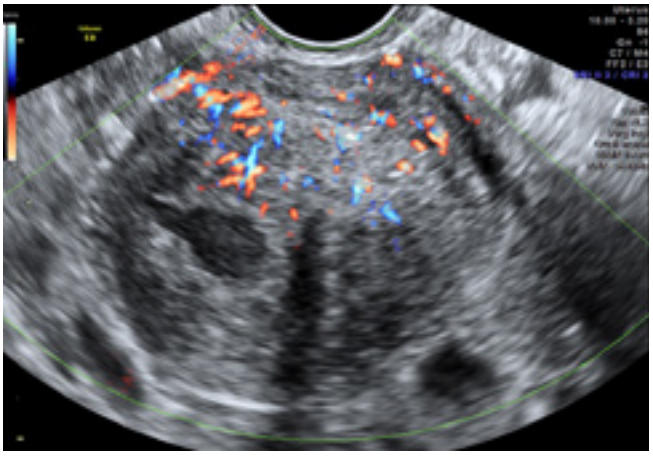

Fig. 5. *Intramural myoma – uterine tumor with mixed echogenicity, rich vascularization in color Doppler*

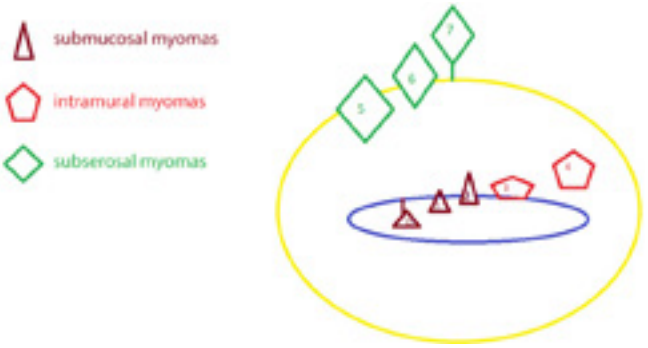

Fig. 6. *Types of uterine myomas according to FIGO[5]. The numbering for the subtypes of myomas is given in Tab. 2*

positive family history, long interval since last delivery, hypertension, and consumption of dietary supplements and soy milk. Factors that reduce the risk of uterine myomas include multiple births, use of oral contraceptives or depot medroxyprogesterone acetate, and smoking in women with low body mass index [weight (kg) / height (m)^2].

Current studies indicate that mutations in the mediator complex subunit 12 (*MED12*) gene are the most common mutation leading to myomas, present in approximately 70% of all these lesions. *MED12* mutations result in a phenotype of smaller tumors containing more extracellular matrix. Myomas in the *MED12 mutation* group are composed in equal parts of smooth muscle and fibroblasts, whereas 90% of cells in *HMGA2* mutation myomas, the second most common group, are smooth muscle cells[7].

Autosomal dominant mutations are usually associated with a mutation in the fumarate hydratase (*FH*) gene – these families are associated with an increased risk of uterine and renal cancer, i.e. hereditary leiomyomatosis and renal cell cancer[7,8].

Mutations in genes encoding non-histone high mobility group proteins, missense mutations and fumarate hydratase gene deletions are common causes of myomas. Overexpression of 14–27 of the 22,000 genes assessed to date, as well as down-regulation of genes that modulate the synthesis of retinoids, insulin-like growth factor (IGF) and those responsible for the synthesis of extracellular matrix (ECM) components, may significantly contribute to the risk of uterine myomas. The tendency to develop these tumors may be hereditary. This may be seen in some syndromes, such as the Reed's syndrome (myomas of the uterus and subcutaneous tissue) and Bannayan-Zonana syndrome (myomas of the uterus, lipomas and hemangiomas)[6].

Tab. 2. *FIGO staging of myomas[5]*

Submucosal myoma (SM)	**Other myoma (O)**	**Hybrid leiomyomas – modeling both endometrium and serosa**
0 – submucosal myoma in the uterine cavity	3 – myoma contacts endometrium, but 100% intramural	Two types of myomas. The first number indicates the degree of contact with the endometrium, and the second number indicates the degree of contact with the serosa
1 – myoma located in the uterine cavity, but *intramural* extension <50%	4 – intramural myxoma	
2 – myoma located in the uterine cavity, but *intramural* extension ≥50%	5 – subserosal intramural extension ≥50%	
	6 – subserosal intramural extension <50%	
	7 – subserosal pedunculated	
	8 – other (parasitic myoma – growing from the peritoneum of organs other than the uterus, cervical myoma)	
FIGO (*Federation Internationale de Gynecologie et d'Obstetrique*) – International Federation of Gynecologists and Obstetricians		

The genetic background of myomas is also supported by their higher incidence (approximately 2.9 times more frequent) in African-American vs Caucasian women[6,9–13].

Excess estrogen and the relationship between high levels of estrogen during the premenopausal period, secondary to the presence of amenorrheic cycles, and during pregnancy are the most thoroughly investigated factors in the formation of myomas. High sensitivity of tumor cells to estrogen during the follicular phase of the menstrual cycle and their ability to bind 20% more hormone per milligram of cytoplasmic protein compared to cells of a "healthy" myometrium is their characteristic feature[3,14].

Production of female sex hormones decreases after menopause. During this period, inhibition of the growth of myomas, and sometimes their involution and calcification, can be expected. Estrone, the main estrogen produced by adipose tissue, increases the risk of myomas in obese women.

Also, a more rapid growth of myomas with increasing doses of gestagen has been observed in women using hormone replacement therapy[13,15]. This effect on tumor cells results from the effects of sex hormones on the expression of genes for growth factors and apoptosis. Estrogens have been shown to stimulate the development of myomas by increasing the expression of genes for the epidermal growth factor receptor *(*EGFR), transforming growth factor beta (TGF-β) and platelet-derived growth factor *(*PDGF), while decreasing the expression of the proapoptotic protein p53. The effect of progesterone on accelerated myoma growth is through increased expression of the genes coding for EGFR, TGF-β and bcl-2 protein with decreased expression of the gene encoding the tumor necrosis factor alpha (TNF-α)[6].

Some authors also draw attention to reports on the influence of lifestyle on the formation of uterine muscle tumors. Excess intake of red meat promotes the formation of myomas, whereas smoking has a protective effect[13].

Recently, it has been shown that vitamin D deficiency may be one of the risk factors for the development of uterine myomas; and what is more, it may correlate with tumor size. Both women already diagnosed with and those at high risk of uterine myomas have reduced levels of vitamin D. The protective effect of vitamin D results from its action on Wnt4/B-catenin inhibition, inhibition of mTOR pathway (*mammalian target of rapamycin*) signaling and modulation of action and expression of metalloproteinases. Increasing consideration is given to the possibility of using vitamin D as a non-invasive treatment for uterine myomas[16].

Increasing attention is being paid to endocrine disrupting chemicals (EDCs), e.g. DDT, cadmium, phthalates, dioxins. These chemicals are commonly found in the environment (air, soil, or water), food, or cosmetics, disrupting normal endocrine function. Some EDCs "mimic" the action of hormones, while other block them. They can alter the endocrine function by changing blood hormone levels, the rate at which they are produced, metabolized, or degraded. These chemicals have been linked to multiple adverse human health effects, including changes in sperm quality and fertility, impaired embryo development, endometriosis, early puberty, metabolic diseases, diabetes and obesity, and uterine myoma formation.

Recent analyses investigating redox potentials have clearly shown that the process of formation of benign and malignant uterine lesions involves significant disturbances at the level of Nrf2 (nuclear factor) expression, which consequently leads to molecular changes already at the level of transcription and translation under the influence of AOE (oxidative enzymes). Increased lipid peroxidation and an altered mechanism of AOE action in the myometrium were observed in endometrial abnormalities. Proteins and mRNA levels of copper-zinc superoxide dismutase (CuZnSOD), catalase (CAT), glutathione peroxidase (GPx), glutathione reductase (GR), and transcription factor Nrf2 were investigated. The study found that the advancement of uterine lesion formation depends on both the type of enzymes involved and the course of endometrial transformation. These observations will contribute to the understanding of the molecular mechanisms underlying oxidative stress mediated disorders[17].

Small and scarce lesions usually do not produce clinical symptoms. If they do occur, however, their type and severity depends on the size of the tumor, its location and coexisting degenerative changes within the myoma itself. The most common clinical signs of uterine myomas include:

- heavy menstrual bleeding that results from endometrial vascular changes, dilatation of small endometrial veins, increased endometrial surface area, dysregulated action of local growth factors, and impaired angiogenesis;
- pain associated with torsion of a pedunculated tumor, dilatation of the cervical canal by a nascent myoma, degenerative changes in the tumor during pregnancy, or rare neoplastic transformation to sarcoma;
- symptoms of gastrointestinal or urinary compression, which may be caused by cervical myomas, myomas lodged in the pouch of Douglas, or rarely by large tumors arising from the sacrouterine ligaments;
- infertility, which occurs in 1–2.4% of uterine cases with myomas and is associated with deformation of the uterine cavity, impaired sperm transport and blastocyst implantation by submucosal tumors, and impaired fallopian tube patency due to intramural myomas[3,18–20].

The benign nature of myomas is due to their inability to metastasize via blood and lymphatic routes, with a relatively low risk of transformation to malignant sarcoma (about 0.2–0.5%).

Uterine myomas on ultrasound

As described by Mendelson, Boehm, and Velez, uterine myomas usually present in ultrasound images as

round, solid, clearly demarcated lesions with lower echogenicity in relation to the surrounding myometrium (hypoechoic)[21].

They generally have a heterogeneous, "striated" or "banded" echostructure characterized by the presence of radial acoustic shadows. The presence of shadows is due to the tissue density in these tumors. In cases of acute myomal necrosis, heterogeneous complex structures appear within the tumor. Usually a hyperechoic capsule is also visible, which allows defining the exact borders of the lesion. As a result of retrograde changes in myomas in the form of necrosis, vitrification, fluid fields and microcalcifications, these tumors may change their echostructure. They then become hypo- or hyperechoic. After menopause, peripheral calcifications may appear. In the case of the presence of tumors in the myometrium, the image of the arch vascular system is disturbed. Uterine myomas are characterized by peripheral vascularization. Vascular anomalies, such as variceal vasodilatation and arteriovenous anastomoses, can be observed within the lesions themselves. The presence of the so-called *brigding sign* between the myoma and the surrounding myometrium is a symptom that helps differentiate uterine myomas, especially those located peripherally, from colorectal or adnexal tumors[22–24].

Uterine myomas can be distinguished from adnexal tumors by the presence of a simple vessel around the myoma periphery[25].

Ultrasonographic differentiation of myoma and adenomyosis is also extremely important. Adenomyosis often manifests as a thickening of one of the uterine walls or diffuse hypoechoic myometrial lesions, but it can also resemble a myoma, creating a mass in the uterine myometrium (*focal adenomyosis*).

Imaging diagnosis of uterine myomas

Ultrasonography (US; including sonohysterography with saline infusion) and magnetic resonance imaging (MRI) are considered the most effective tools for the diagnosis of uterine myomas in terms of number, echostructure, volume, or location in the uterine muscle. These modalities allow precise tracing of tumor vascularization and facilitate differentiation between benign, i.e. adenomyosis, and malignant lesions[26].

Simplicity, low cost, wide availability and, most importantly, non-invasiveness are great advantages of ultrasound in gynecology. The Doppler technique enables assessment of hemodynamic changes in lower pelvic vessels, characteristic of both physiological and pathological states. Characteristics of vascular flow is possible owing to the use of parameters enabling its qualitative and quantitative assessment. These include resistance index (RI), pulsality index (PI) and systolic-diastolic ratio (S/D)[27].

Transabdominal imaging offers a wide field of view, high signal penetration, and the ability to examine adjacent organs beyond the pelvis minor. It is therefore more effective for visualization of large tumors, submucosal or parasitic myomas penetrating deep into the abdominal cavity. Imaging is hampered by retroverted uterus, scarring, and excessive fatty tissue.

Transvaginal ultrasound (TVS) allows for detailed imaging as the transducer is placed adjacent to the tumor, which means that a high-frequency ultrasound wave can be used. It is now a routine examination in gynecology. The combination of transabdominal and endocavitary ultrasound is the most commonly used technique for detecting, mapping, and evaluating myomas[28].

Ultrasonography combined with sonoangiography allows detection of vessels as small as 100–200 μm in diameter. Using color Doppler technique, it is possible to visualize the ascending branch of the uterine artery. Using low pulse repetition frequency (PRF) filters in the range of 600–800 Hz and wall motion filters (WMF) in the range of 50–100 Hz, arch vessels at the border of the outer and middle myometrial layers can be visualized[22–24].

Based on uterine arterial blood flow analysis by color Doppler, steroid sex hormones were found to be the major regulators of uterine perfusion. Estrogen and progesterone receptors are present in the uterine arteries and myometrium. Cyclic changes in the number of these receptors alter uterine blood flow. Estrogen acts as a vasodilator, increasing uterine perfusion, therefore it is more intense in premenopausal women. Progesterone has the opposite effect, decreasing uterine blood flow[29].

Based on a feedback from a panel of clinicians (Morphological Uterus Sonographic Assessment, MUSA), a common classification was proposed to describe ultrasonographic blood flow characteristics in myometrial pathology[26].

The MUSA scale is interpreted as follows:
- 1 – no flow;
- 2 – minimum flow;
- 3 – moderate flow;
- 4 – strong flow.

It should be noted that myomas are usually sparsely vascularized. Color Doppler or power Doppler imaging shows myomas as structures with abundant peripheral vascularization or with poorly visible circular vascularization pattern, whereas central flow is rarely observed. The afferent branches reach the center of the tumor through peripheral arteries. The peripheral flow in the tumor is slightly more increased compared to normal myometrium and the central portion of the myoma. Doppler techniques make it easier to distinguish an endometrial polyp with a single feeding vessel from a myoma and adenomyosis, where vascularization is much more diffuse[30–32].

Vascular myomas are rare. They are characterized by the presence of multiple thick-walled and dilated venous and lymphatic vessels, which increase in diameter along with the tumor growth.

There are no structural differences in the vasculature of sparsely and richly vascularized myomas. According to the majority of authors, resistance and pulsation indices in myoma vessels are useless, as both low- and high-resistive flow can be observed. Such a mixed type of vascularization is found mainly in pedunculated myomas. The ranges of values for resistance and pulsatility indices in the vessels of myomas are RI = 0.4–0.6 and PI = 0.7–0.9, respectively.

Flow cytometry showed an inverse correlation between RI (0.27–0.73, median 0.44) and myoma volume. The Doppler flow waveform in the vessels of myomas is similar to that observed in the uterine arteries. In most cases, it is possible to visualize the diastolic flow. The lack of early diastolic *notch* seen in high-resistance vessels is a hallmark. High amplitude of systolic and diastolic flow velocity may occur in submucosal myometrial vessels, in which pathological vessels mimicking those found in malignant neoplasms may form[22].

The phase of the menstrual cycle and the duration of menopause do not affect the profile of blood flow in myometrial vessels, hence it is believed that there are different mechanisms regulating flow in the uterine arteries and myomal vessels. However, the influence of patient's hormonal status on this profile is noticeable. Resistance indices in premenopausal women are lower than in menopausal patients, regardless of tumor size.

Literature reports suggest the impact of lesion location and size on the Doppler blood flow profile. Larger and peripherally located myomas with features of degenerative changes are characterized by higher diastolic blood flow velocity and lower resistance index in the tumor vessels. Some authors note that an increase in lesion diameter in both pre- and postmenopausal women improves blood flow visualization regardless of tumor location, whereas a decrease in RI is seen only in the vessels of submucosal and subserosal lesions. This effect was not observed in the case of intramural myomas. Such observations are attributed to a different physiology of the surrounding tissues (endometrium, uterus) and their direct influence on myomas (pressure exerted by the myometrium) as well as their distance from the larger branches of the uterine arteries. Large myomas, characterized by central foci of necrosis, show moderate RI in peripheral vessels on Doppler, and low RI in vessels inside the tumor, due to vasoactive factors released in this region[33].

In 1992, Kurjak compared blood flow profiles in the uterine arteries of healthy patients and those diagnosed with uterine myomas[33]. The measurements of blood flow parameters in uterine arteries revealed lower resistance and pulsation indices and higher blood flow velocities in patients with uterine myomas. The study demonstrated that myomas alter uterine blood flow regardless of their size or the presence of clinical symptoms.

The assessment of blood flow parameters in the uterine arteries and in the vessels of myomas, which are increasingly easy to visualize due to advances in imaging techniques and the sensitivity of ultrasound equipment, is extremely important to attempt differentiation from malignant lesions arising from the myometrium.

Diagnostic difficulties may be encountered in the evaluation of interligamentous myomas. Ambrosio et al. showed that acoustic shadowing was present in 89% of these myomas (63% of interstitial myomas, of which 100% had shadowing within the lesion, which was the most characteristic feature of this group of tumors). Up to 89% of these myomas showed increased flow within the tumor (MUSA grade 3 or 4), compared with the control group. Interligamentous myomas were solid in 84% of cases[34].

Uterine sarcomas

Nonepithelial neoplasms of the uterine corpus are rare, accounting for approximately 1% of female genital malignancies and less than 5–10% of uterine malignancies[35]. Sarcomas occur in older women (age at diagnosis is usually 62–67 years). The risk is twofold higher in black women compared to white women[36].

The prognosis for patients diagnosed with uterine sarcoma depends on tumor size and stage. Five-year survival rates for women are less than 10% except when the tumor is confined to the uterus (then it is 50%). The prognosis for uterine sarcomas is much worse than for endometrial cancer. This is due to the more rapid formation of both local and distant metastases[3,37].

Sarcomas are tumors composed of mesenchymal tissue. Simple (homogenous) and complex sarcomas, including homologous (derived from uterine tissue), heterologous (containing tissue not normally found in the uterus), and mixed forms (composed of a squamous or glandular, benign or malignant epithelial component and a mesenchymal component – always malignant) have been distinguished.

Similar risk factors may promote the development of sarcoma or uterine cancer. Both cancers are associated with obesity, nulliparity, and excess endogenous and exogeneous estrogen[36].

Pelvic exposure to direct radiation, especially during radiation therapy, is now a significant risk factor of uterine sarcomas.

So far, the etiopathogenesis and molecular abnormalities underlying these tumors have not been fully understood. The World Health Organization (WHO) classification of endometrial sarcomas distinguishes leiomyosarcoma (LMS – 40% of endometrial sarcomas,

as well as non-infiltrating and infiltrating low-grade (LG ESS – 10–15% of sarcomas) and high-grade (HG ESS) endometrial stromal sarcoma (ESS).

Other soft tissue sarcomas, including striated cell sarcoma and undifferentiated types characterized by nuclear pleomorphism, very rarely occur in the uterine body. Smooth muscle tumors of uncertain malignant potential (STUMP) include LMS of uncertain malignant potential, usually with good prognosis[38].

Leiomyosarcomas accounts for approximately 1.3% of malignant uterine tumors and ¼–1/3 of uterine sarcomas. The mean age of incidence is 52 years. Five-year survival rates for LMS range from 19–65%. Metastases most often occur in the lungs[37].

Poorly differentiated invasive sarcoma has an incidence of 1.8 per 1,000,000 women in the general population. The mean age at onset is 52 years. The characteristic feature of this tumor is infiltration of myometrial vessels in the form of finger-like microextensions. It is a hormone-dependent sarcoma. More than 80% of ESS have receptors for estrogen, progesterone or gonadolyserine. These receptors may be responsible for autocrine tumor growth. ESS cells express aromatase[1]. Five-year survival rates for ESS range from 50–65%[37].

Undifferentiated uterine sarcoma (UUS) is a tumor with no differentiated cells in the stroma. It is a highly malignant tumor that infiltrates the myometrium. It has a poor prognosis.

Uterine sarcomas usually remain asymptomatic for a long time. Accurate diagnosis is delayed when other myometrial lesions coexist (adenomyosis, myomas) and is usually made during histopathological evaluation after hysterectomy for other indications. Abnormal, usually heavy, uterine bleeding is one of the most frequently observed symptoms, occurring in 70–90% of patients. Uterine enlargement can be found in 20–30% of patients. Lower abdominal pain occurs when the uterus is significantly enlarged or when the tumor is locally advanced. Sarcomas metastasize mainly through the bloodstream. Lymphatic spread is rare. In the case of dissemination, the symptoms depend on the location and size of the metastases[1,29,35].

The rare occurrence and histopathologic heterogeneity of endometrial sarcomas mean that the knowledge of them comes mainly from retrospective studies.

Sarcomas on ultrasound

Leiomyosarcomas are usually described on ultrasonography as solid, oval tumors with heterogeneous internal echoes, containing areas of mixed and low echogenicity.

Uterine sarcomas are shown in Fig. 7 and Fig. 8. The diagnosis was confirmed by postoperative histopathological examination.

The endometrium is usually thin, atrophic, with mucosal pattern depending on the phase of the cycle in young women. Retrograde changes in the form of centrally localized necrosis are very common. On the other hand, the hypoechogenic areas in sarcomas due to the presence of focal necrosis are also found in myomas. Therefore, myomas and sarcomas are often difficult to differentiate on ultrasound. The size of the tumor is very important. Rapid growth of the tumor (doubling of the lesion diameter within 6 months may raise suspicion of sarcoma) may be one of the most important alarming symptoms. The lack of calcifications inside the tumor and the absence of acoustic shadowing is the most characteristic US feature of sarcomas, although definitive diagnosis cannot be based on US image.

On ultrasound, endometrial stromal sarcoma appears as a polypoid, hypoechoic, pronounced tumor of the uterine cavity originating from the endometrium, enlarging the uterine cavity and the entire uterine body. The tumor usually does not involve the entire mucosa and may infiltrate the myometrium at the base, which is also seen on ultrasound.

Doppler diagnosis of uterine sarcomas

Doppler ultrasonography can visualize an irregular distribution of vessels in the tumor; they are thin, randomly scattered or dilated, with multiple interconnections. The vessels may be located in the marginal and central areas of the tumor, with the mean resistance index (RI) values of 0.37 ± 0.03. Analyzing the vascularization pattern of lesions arising from the myometrium, it should be noted that low resistance to blood flow in the tumor vessels (RI <0.5) occurs in 9% of premenopausal and 4% of postmenopausal women[29,33].

Sarcoma-like lesions are characterized by MUSA grade 3 or 4 vascularization[39]. Pathologic vascularization is found in 100% of sarcomas.

Peak systolic velocity (PSV) may be an additional parameter allowing for more precise diagnosis. PSV was found to be higher in uterine sarcomas than in myomas (mean: 71 cm/s vs 22.5 cm/s, respectively). PSV assessment may be a helpful indicator in differentiating between malignant and nonmalignant tumors of the uterine body.

The vascularization of angiosarcomas is irregular, centrally and peripherally localized. Blood flow indices in the tumor are low.

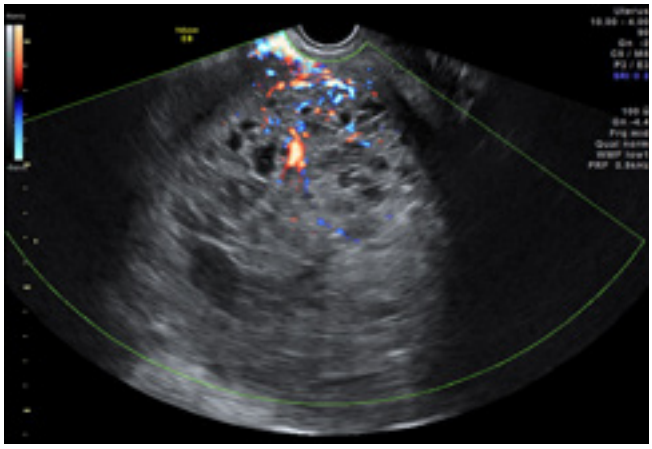

Fig. 7. *Sarcoma. Mixed echogenicity, richly vascularized, grade 4 color Doppler lesion*

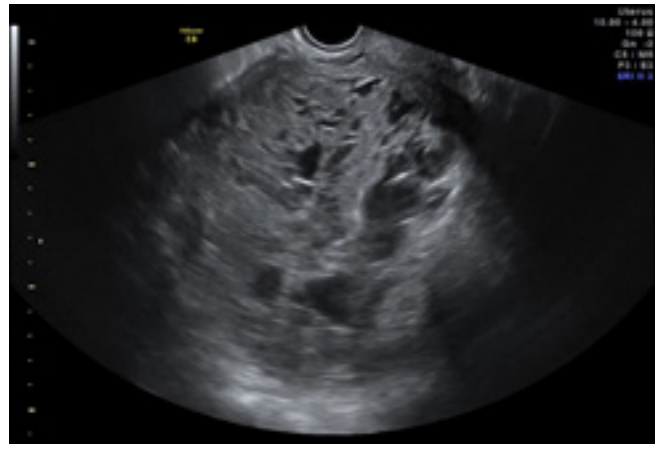

Fig. 8. *Mixed echogenicity sarcoma lesion*

Conclusions

Differentiation between myomas and uterine sarcomas is difficult and not always possible. Ultrasonography and magnetic resonance imaging are the gold standard for the diagnosis of uterine tumors.

It can be generally accepted that the finding of a large inhomogeneous uterine tumor on ultrasound in a postmenopausal patient should raise the suspicion of sarcoma even when the sparse vascularization of the lesion is shown in imaging[21,37].

It is now known that neither RI nor PI analysis can clearly distinguish between myomas and uterine sarcomas. It is estimated that RI is not significantly different between myomas and leiomyosarcomas (mean: 0.49 ± 0.18)[31].

The diagnosis of uterine tumors is actually limited to transabdominal and transvaginal ultrasound. Both methods are widely used and readily available. To distinguish whether the lesion is benign or malignant, in case of diagnostic difficulties, Doppler imaging should be used, following the rule that benign lesions are usually hyperechoic, less vascularized – most often in the periphery (MUSA grade 1).

Since sarcomas are usually single, large tumors, often rapidly growing, the recommended ultrasound follow-up of suspicious lesions should be performed after 3 months. Virtually all sarcomas have increased vascular flow (MUSA grade 3 or 4) and cystic degeneration is found within the tumor in half of the cases. Women under the age of 40 years are at a low risk of sarcomas.

Ultrasonography using color Doppler appears to be a sensitive and specific method in experienced hands. However, further studies are needed to introduce sonographic terminology and methodology to the study of the uterine muscle and to clarify the imaging parameters that differentiate typical myomas from smooth muscle tumors of unknown malignant potential (STUMP) and leiomyosarcomas.

The above literature review and our own experience have shown that the flow velocity is higher in uterine sarcomas than in myomas, but no unambiguous norms or values of flow parameters have been developed so far. This topic requires further studies and larger groups of women undergoing diagnosis.

Conflict of interest

The authors report no financial or personal relationships with other individuals or organizations that might adversely affect the content of the publication and claim ownership of this publication.

Author contributions

Original concept of study: PG. Writing of manuscript: KW, TG, MH, PC, EW. Analysis and interpretation of data: KW, TG, PC, EW. Critical revision of the manuscript for important intellectual content: KW, AS, GP. Collection and assembly of data: KW, AS, GP.

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