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Case Series

Pathology Section

Endometrial Stromal Tumour: Clinicopathological Series of Seven Cases

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ABSTRACT

Endometrial Stromal Tumour (EST) mimics other neoplasms and is difficult to diagnose due to its wide range of morphologies. This is a clinicopathological study of seven cases of EST, which includes Endometrial Stromal Nodule (ESN), Low-grade Endometrial Stromal Sarcoma (LGESS), and High-grade Endometrial Stromal Sarcoma (HGESS). The age ranges from 34 to 75 years. Five out of seven cases presented with abnormal uterine bleeding, abdominal pain, and were radiologically suspected to be leiomyomas. After histopathological and Immunohistochemical (IHC) examination, one was diagnosed as ESN, three as LGESS, and the remaining three cases as HGESS. One case was initially diagnosed as a cellular leiomyoma and experienced multiple recurrences, eventually being diagnosed as HGESS with a fatal outcome within 36 months of the onset of the first symptoms. ESN and LGESS should be differentiated from leiomyoma and leiomyosarcoma. IHC plays an important role in distinguishing these tumours from the more common smooth muscle counterparts.

Keywords: Cluster of differentiation 10, Mimics, Wilms tumour gene 1

INTRODUCTION

The EST are a rare group of tumours, accounting for <1% of all uterine tumours [1]. However, accurate diagnosis is important as most of them fall under the malignant category unlike their smooth muscle counterparts. There are four main categories, include ESN, LGESS, HGESS and Undifferentiated Stromal Sarcoma (USS). LGESS is the most common type [2]. While a few case reports are available [2-6], there is a lack of large case series with follow-up data. The present study describes a series of seven EST cases, detailing gross findings, morphology, IHC findings, and follow-up data.

CASE SERIES

The present case series describes a study of seven ESTs collected over a period of four years (from 2018 to 2022), with ages ranging from 34 to 75 years. Detailed clinical, radiological, and treatment histories of each case were recorded before histopathological examination. IHC staining was done with formalin-fixed paraffin-embedded sections using a detection system based on the peroxidase-antiperoxidase method. Heat-induced epitope retrieval and enzymatic activation of chromogen were conducted for visualising the antigen-antibody reaction product. A positive tissue control and the same tissue for internal negative control were used for each case. The clinicopathological profiles of the cases are summarised in [Table/Fig-1].

Case 1

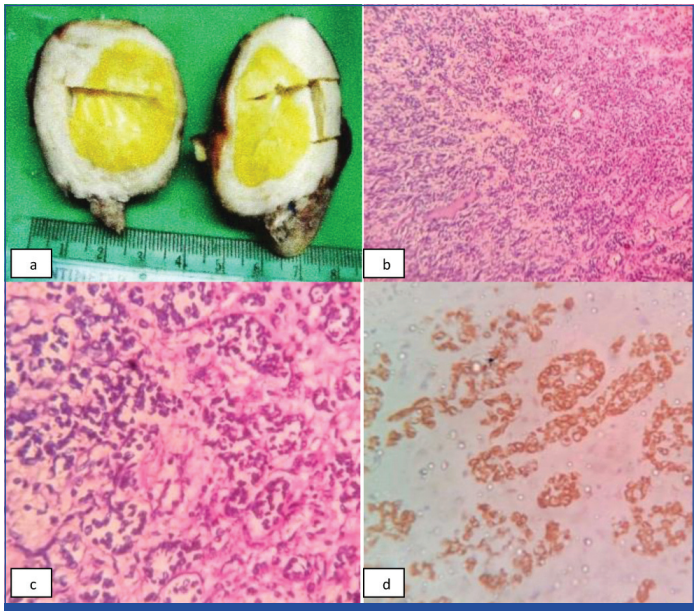
The first case involved a 35-year-old parous woman who presented with abnormal uterine bleeding for two years, with no significant

S. No.	Age (y)	Signs and symptoms	Clinicoradiological diagnosis	Surgery performed	Gross	Histological diagnosis	IHC	Follow-up
1	35	Abnormal uterine bleeding	Submucosal/ intramural nodule	Myomectomy	Well circumscribed nodule measuring 4×4 cm, cut surface solid yellow to tan color	ESN (Mitotic activity <3/10HPF)	CD10(+) Desmin(-)	Uneventful following surgery.
2	40	Abnormal uterine bleeding, pelvic pain	Fibroid uterus	Myomectomy followed by total hysterectomy	8×6×3 cm on myomectomy, later TAH and BSO done(intramural masses, largest 7×4×3 cm)	LGESS (Mitotic activity >4/10 HPF)	CD10(+) WT1(+) Desmin(-)	Doing well after 24 months postsurgery.
3	75	Pelvic pain and mass	Fibroid uterus	Hysterectomy	Total 8×6×4 cm, two globular masses 6×4 cm and 4×3 cm	LGESS (Mitotic activity >4/10 HPF)	CD10(+) WT1(+) Desmin(-)	Doing well after 30 months postsurgery.
4	52	Abnormal uterine bleeding	Fibroid uterus	Hysterectomy	Two irregular tissue pieces 8×4 cm and 4×3 cm	LGESS (Mitotic activity 7/10 HPF)	CD10(+) WT1(+) Desmin(-)	Doing well after 50 months postsurgery.
5	60	Abnormal uterine bleeding, pelvic pain and pelvic mass	Malignant lesion	Total abdominal hysterectomy	Total 11×8×7 cm with a polypoid mass 6×5 cm	HGESS (Expansile, permeative growth with Mitotic activity 15/10 HPF necrosis lymphvascular invasion)	CD10(+) Desmin(-) Cyclin D1(+)	Patient was lost within one month of surgery and initial chemotherapy.
6	60	Abnormal uterine bleeding, pelvic pain and abdominal mass	Malignant lesion	Resection of growth with attached small bowel segment.	6×5×4 cm	HGESS (Expansile permeative growth with Mitotic activity 15/10 HPF necrosis lymphvascular invasion)	CD10(+) Desmin(-) Cyclin D1(+)	Lost during follow-up.

7	34	Pain abdomen and abdominal swelling	Fibroid uterus with degeneration	Myomectomy with cholecystectomy (2018) Followed by total abdominal hysterectomy (2019). Followed by debulking (2020)	5 kg mass on debulking operation	HGESS (Expansile, permeative growth with Mitotic activity>10/10 HPF necrosis and lymphvascular invasion)	CD10(+) Desmin(-) Cyclin D1(+)	Patient expired 36 months after first surgery.

[Table/Fig-1]: Clinicopathological profile of all the cases.
BSO: Bilateral salpingo-oophorectomy; TAH: Total abdominal hysterectomy; LGESS: Low-grade endometrial stromal sarcoma; HGESS: High-grade endometrial stromal sarcoma

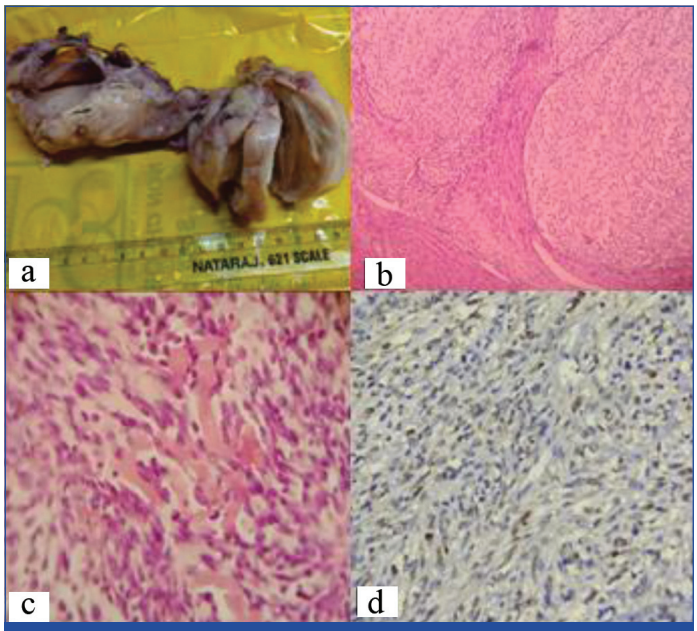
past medical history. Ultrasonography (USG) revealed an intramural mass suggestive of a leiomyoma. On gross examination, a well-circumscribed yellowish nodule measuring 4×4 cm was observed [Table/Fig-2a]. Microscopically, densely packed round to oval cells resembling the proliferative phase of the endometrium were seen, with low mitotic activity (<3/10HPF) [Table/Fig-2b,c]. Periarteriolar whorling arrangement and expansile non infiltrative margins were noted. The neoplastic cells were positive for CD10, leading to a diagnosis of ESN [Table/Fig-2d]. The patient had an uneventful long-term postoperative follow-up period.



[Table/Fig-2]: a) Gross photograph of well circumscribed Endometrial Stromal Nodule (ESN) with characteristic yellow cut surface. b) Photomicrograph showing cellular round cell tumour with prominent small arterioles (100X). c) Proliferation of endometrial stromal cells without atypia (400X). d) Tumour cells show strong diffuse CD10 positivity (400X).

Case 2

A 40-year-old woman who presented with menorrhagia, pelvic pain, and an abdominal mass lasting for one year. The sonographic diagnosis was a fibroid uterus, and she underwent myomectomy [Table/Fig-3a]. However, she returned with menorrhagia after three months. Subsequently, a total abdominal hysterectomy and omental sampling were performed. The provisional clinical diagnosis was leiomyosarcoma. On gross examination, multiple grayish-white intrauterine masses were observed, with the largest measuring 7×4×3 cm. Microscopically, the tumour masses consisted of proliferating round to oval stromal cells arranged in sheets with prominent vascularity and the presence of spiral vessels. Hyalinised stroma with myxoid change, as well as lymphatic invasion and myometrial invasions, were noted [Table/Fig-3b]. The neoplastic cells exhibited mild to moderate pleomorphism, moderate nuclear atypia, and increased mitotic activity (>4/10 HPF). The presence of focal atypical spindle cells and collagen deposition [Table/Fig-3c] made it challenging to differentiate between LGESS, leiomyosarcoma, and sex cord stromal tumour. Omental tissue sampling revealed the presence of sarcomatous deposits. IHC showed diffuse positive staining for CD10, WT1 [Table/Fig-3d], Estrogen Receptor (ER), Progesterone Receptor (PR), and negative staining for desmin, inhibin, and CD56. The final diagnosis was LGESS.



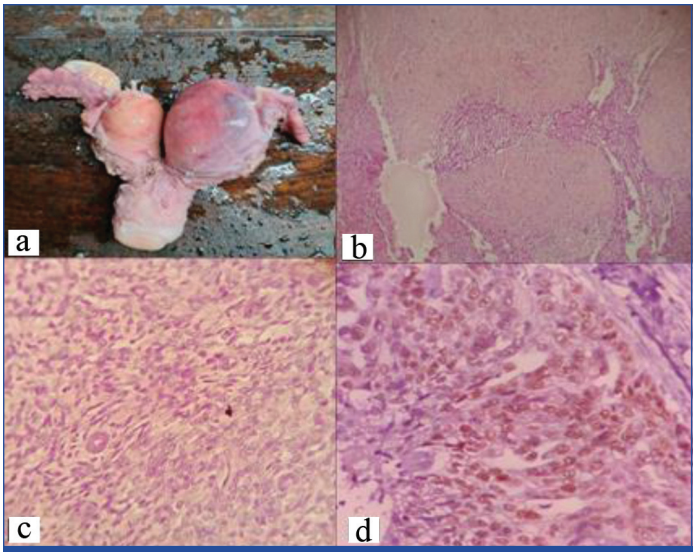
[Table/Fig-3]: a) Gross photograph of low-grade ESS after removal of tumour in suspected fibromyoma. b) Photomicrograph showing LGESS with myometrial invasion (100X). c) Spindle cell morphology with presence of collagenous band in low-grade ESS (400X). d) Low-grade ESS shows WT1 immunostain positivity (400X).

Case 3

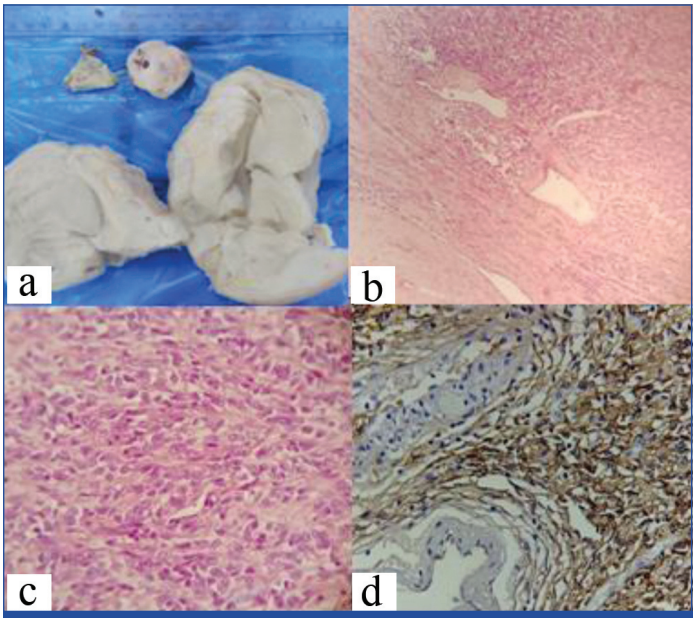
A 75-year-old postmenopausal woman presented with pelvic pain and a mass that had been present for two and a half years. She had a history of myomectomy 15 years ago. The clinical and radiological diagnosis was a fibroid uterus, and the patient underwent abdominal hysterectomy and Bilateral Salpingo-Oophorectomy (BSO). On gross examination, multiple intramural fleshy white masses were identified, with the largest measuring 8×6 cm [Table/Fig-4a]. Morphologically, it shared similar features with the second case. It was a cellular round cell tumour that exhibited tongue-like myometrial invasion, moderate nuclear atypia, brisk mitoses, and areas of vascular prominence [Table/Fig-4b,c]. The differential diagnosis was LGESS versus leiomyosarcoma. Immunohistochemically, the tumour cells were positive for CD10, WT1, ER [Table/Fig-4d], PR, and negative for desmin. Therefore, the final diagnosis was LGESS.

Case 4

A 54-year-old woman presented with complaints of postmenopausal bleeding lasting for eight months. She underwent hysterectomy following a radiological diagnosis of a fibroid uterus. On gross examination, intramural fleshy masses were noted, with the largest measuring 8×4 and 4×3 cm [Table/Fig-5a]. Microscopically, there was proliferation of round to oval stromal cells arranged in sheets, with lymphovascular and myometrial invasion [Table/Fig-5b]. The neoplastic cells exhibited nuclear atypia and increased mitotic activity (7/10 HPF) [Table/Fig-5c]. The diagnosis of LGESS was made after conducting an IHC study using a similar antibody panel as in previous two cases. The tumour cells were positive for CD10, WT1 [Table/Fig-5d], ER, PR, and negative for desmin. In all three cases of LGESS, the initial differential diagnosis was leiomyosarcoma. The postoperative follow-up period was unremarkable in these cases.



[Table/Fig-4]: a) Gross photograph of TAH&BSO in low-grade ESS shows two uterine masses. b) Tongue-like myometrial invasion in low-grade ESS (100X). c) Cellular round cell proliferation with spiral vessel (400X). d) Low-grade ESS shows diffuse ER immunopositivity (400X).



[Table/Fig-5]: a) Gross photograph of low-grade ESS mimics fibromyoma. b) Low-grade ESS shows myometrial and lymph vascular invasion (100X). c) Neoplastic cells show atypia with high mitotic activity in low-grade ESS (400X). d) Photomicrograph shows hyalinised vessels and WT1 positivity (400X).

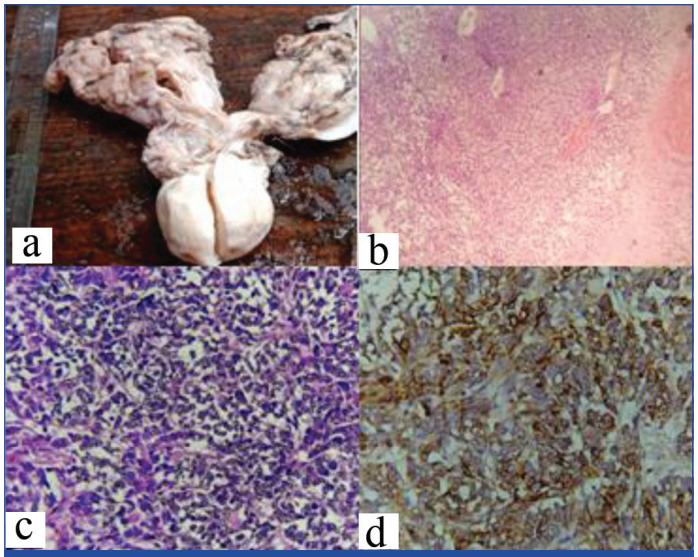
Case 5

A 60-year-old woman presented with postmenopausal bleeding and a pelvic mass lasting for two months, without any significant past medical history. Her serum CA-125 level was mildly elevated (45 U/mL), and the radiological diagnosis was a possibly malignant endometrial polyp.

Following hysterectomy and BSO an irregular ulcerated polypoid lesion measuring 6×5 cm was observed, filling up the uterine cavity [Table/Fig-6a]. Microscopically, there were sheets of neoplastic round to oval cell proliferation with areas of necrosis, haemorrhage, arborising vasculature, and a high mitotic count (15/10 HPF) [Table/Fig-6b,c]. The tumour had invaded into the myometrium. The differential diagnoses were HGESS and poorly differentiated carcinoma. IHC study showed focal CD10 positivity [Table/Fig-6d], positive staining for Cyclin D1, and negative staining for WT1, ER, PR, EMA, and desmin. Therefore, the final diagnosis of HGESS was made. Unfortunately, the patient was lost within one month of the operation despite her receiving initial chemotherapy.

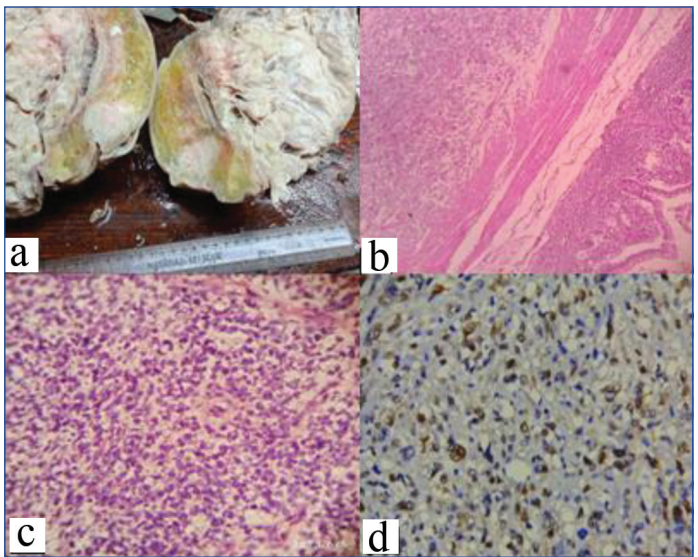
Case 6

A 60-year-old woman with a clinical suspicion of intraabdominal malignancy. The CT abdomen revealed a tumour arising from the



[Table/Fig-6]: a) Gross photograph of fragile mass in high-grade ESS. b) Cellular tumour with areas of necrosis in high-grade ESS (100X). c) Tumour cells show high-grade atypia with brisk mitotic activity (400X). d) HGESS shows focal CD10 positivity (400X).

fundus of the uterus and adhering to a segment of the small bowel. Palliative surgery was performed, which involved resection of the mass along with a portion of the attached small bowel. On gross examination, a fragile mass measuring 6x5x4 cm with necrotic areas was observed [Table/Fig-7a]. Microscopically, it demonstrated an atypical proliferation of round to spindle cells, tumour giant cells, hyalinised vessels, lymphovascular invasion, myxoid areas, necrosis, haemorrhage, and a high mitotic activity (15/10HPF) [Table/Fig-7c]. The tumour invaded the small bowel wall through the muscularis propria [Table/Fig-7b]. The closest differential diagnoses included Gastrointestinal Stromal Tumour (GIST), spindle cell sarcoma, HGESS and desmoplastic small round cell tumour. The tumour showed focal positivity for CD10, Cyclin D1 [Table/Fig-7d], and negativity for desmin, myogenin, WT1, S-100, ER, PR, and EMA. The final diagnosis was HGESS; however, the patient did not return for follow-up.

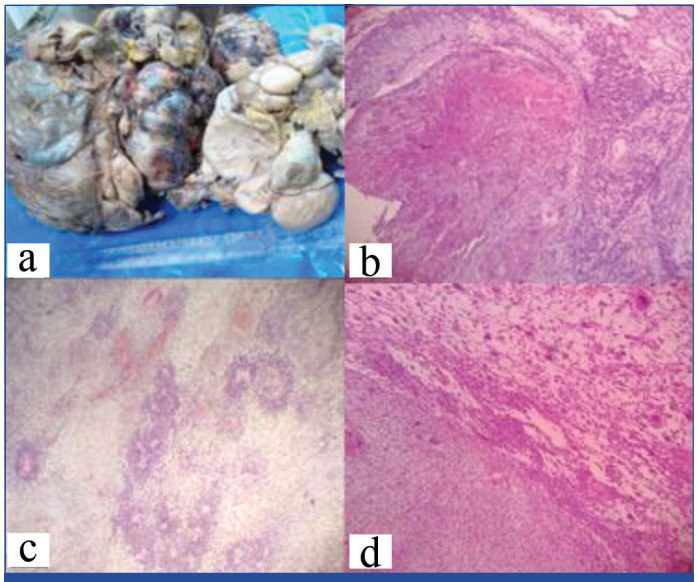


[Table/Fig-7]: a) Gross photograph of high-grade ESS with yellowish area of myxoid change. b) HGESS showing bowel wall involvement (100X). c) Neoplastic cells show hyperchromatic nuclei with high mitotic activity (400X). d) HGESS shows Cyclin D1 positive tumour cells (400X).

Case 7

A 34-year-old female presented in 2019 with abdominal swelling and pain lasting for eight months. She had a history of cholecystectomy with myomectomy in 2018, with a histopathological report of chronic cholecystitis and leiomyoma. Following abdominal hysterectomy in June 2019, a histopathological diagnosis of cellular leiomyoma was

given. She returned back in February 2020 with a large recurrent mass and an Magnetic Resonance Imaging (MRI) showed a pelvic mass with mixed intensity and solid cystic areas, herniating into subcutaneous tissue. The mass was separated from the liver, pancreas, kidney, and spleen, and it displaced the stomach and bowel loops. The patient underwent debulking surgery, and a 5 kg tumour mass was removed [Table/Fig-8a]. A provisional histopathological diagnosis of desmoplastic small round cell tumour was made. Microscopically, it was a hypercellular oval to spindle cell tumour with areas of necrosis, lymphovascular invasion, high mitotic count, and focal fibro-myxoid appearance [Table/Fig-8b-d]. She underwent chemo-radiation but eventually presented with abdominal and lung metastasis in November 2020. IHC study was conducted, which showed negative staining for Cytokeratin, EMA, desmin, and WT1. Vimentin and cyclin D1 were positive, and CD10 was focally positive. Finally, the diagnosis of HGESS was made, but unfortunately, the patient had already succumbed to death within a month of the last surgery.



[Table/Fig-8]: a) Gross photograph of recurrent high-grade ESS after debulking operation. b) Hypercellular spindle cell morphology with hyalinised vessels and lymph vascular invasion (100X). c) High-grade ESS shows extensive areas of necrosis and haemorrhage with focal preservation of tumour cells (100X). d) Photomicrograph shows myxoid areas, tumour giant cells and atypia with high mitotic activity (400X).

DISCUSSION

Endometrial Stromal Tumours (ESTs) are a rare group of tumours that typically occur in perimenopausal women. Due to their rarity, diagnosing ESTs can be challenging. LGESS tends to occur at a younger age than HGESS and USS with a median age ranging from 45 to 55 years [3]. The most common clinical presentation of EST is abnormal uterine bleeding, although patients may also experience pelvic pain and dysmenorrhoea [7]. ESNs are clinically considered benign, while LGESSs are tumours of low malignant potential, often exhibiting indolent clinical behaviour. Some cases may experience late recurrence after hysterectomy, as seen in two cases. HGESS, on the other hand, is a tumour of high malignant potential with a more aggressive clinical outcome. UUS displays high-grade morphological features and exhibits very aggressive clinical behaviour [8]. The differential diagnosis for ESN includes cellular leiomyoma and LGESS. ESNs typically exhibit uniform bland ovoid cells resembling proliferative phase endometrial stroma. The vascular pattern, composed of typical arterioles, is not a prominent feature of cellular leiomyoma. While ESNs may contain large blood vessels, a characteristic of cellular leiomyoma, they are not as conspicuous as in cellular leiomyoma [9]. Additionally, CD10 can be useful in differentiating ESN from cellular leiomyoma. The presence of a pushing margin and the absence of lymphovascular invasion help differentiate ESN from LGESS. Therefore, a definitive diagnosis of ESN can be rendered on resected specimens, allowing for extensive sampling, and cannot be confidently established on small biopsies [8].

The differential diagnosis for LGESS includes HGESS, cellular leiomyoma, leiomyosarcoma with an extensive intravascular component, Uterine Tumours Resembling Ovarian Sex-Cord Tumours (UTROSCT), and adenosarcoma [10]. LGESS is morphologically characterised by uniform ovoid cells with mild to moderate atypia, tongue-like invasion into the myometrium, lymphovascular permeation, and a mitotic activity ranging from 1-13/10HPF [11]. IHC panel including CD10, inhibin, calretinin, and CD56 can be useful in distinguishing ESS from sex cord-stromal tumours, although it is important to note that markers of sex cord-like differentiation may be strongly positive in sex cord-like areas of ESS. CD56 is typically positive in sex cord-stromal tumours but negative in ESS. In the abdominal cavity, GIST is an important differential consideration [12]. Morphologically, both tumours are usually distinct. GIST demonstrates cellular, plump, and uniformly spindle cell proliferation with nuclear palisading and characteristic perinuclear vacuolisation, in contrast to the plump, round to oval cell proliferation of EST. GISTs typically show limited atypia, with mitotic activity rarely exceeding 10/50 HPF [13]. GISTs are positive for c-Kit, CD34, and DOG1, while LGESS shows diffuse positivity for ER, PR, and CD10.

There can be significant overlap in staining for CD10 and desmin between ESS and smooth muscle tumours, making it helpful to include additional smooth muscle markers, such as h-caldesmon or calponin, which are usually negative in ESS. In cases presenting with urinary symptoms and a bladder mass, the differential diagnosis includes Solitary Fibrous Tumour (SFT), synovial sarcoma, carcinoid, Primitive Neuroectodermal Tumours (PNET) and large nested variants of urothelial carcinoma. The focal presence of spindle cell morphology, bland cytology, and abundant collagen in LGESS may lead to a differential diagnosis of SFT. Additionally, endometrial stromal sarcoma can occasionally show stag horn-like vessels similar to those seen in SFT. However, these findings are seen only in focal areas of LGESS, and the presence of typical small round arterioles helps establish the diagnosis. CD34 and STAT6 are positive in SFT and negative in EST.

Carcinoid tumours and ESS can exhibit overlapping IHC staining. ESS is focally positive for CD56 and synaptophysin, while carcinoid tumours variably stain positive for ER, PR, or CD10. However, the characteristic organoid pattern and salt and pepper nuclear chromatin in carcinoid tumours, along with diffuse ER, PR, and CD10 positivity in LGESS, aid in distinguishing these two entities. LGESS typically shows negative CD99 staining, which is seen in PNET. The wormlike pattern of invasion observed in LGESS is similar to the infiltration seen in the large, nested variant of urothelial carcinoma. GATA3 and thrombomodulin are positive in large, nested variants of urothelial carcinoma and negative in ESS [12]. WT1 positivity has been observed in non neoplastic endometrial stroma. Diffuse WT1 positivity is characteristic of endometrial stromal neoplasms and can aid in the differential diagnosis [14]. All three of the LGESS cases showed positive staining for WT1.

The term HG-ESS has been reintroduced in the classification of EST following the discovery of t(10;17)(q22;p13) resulting in YWHAE-NUTM2A/B fusion, which is associated with distinct morphological characteristics [15]. The characteristic morphological features of HG-ESS include a tumour with high mitotic activity (>20-30 mitoses/10 HPF), a fibrous or myxoid appearance, extensive lymphovascular invasion, diffuse positivity for cyclin D1, and negative staining for smooth muscle markers and WT1 [16]. Genetic analysis may assist in identifying HG-ESS in cases where the diagnosis is challenging [15]. Patients with HG-ESS often lack expression of ER and PR. Therefore, it remains unclear whether the ovaries should be preserved in premenopausal women with HG-ESS. In a study by Zhang YY et al., six patients underwent ovarian preservation surgery, and four of them were in Stage I without recurrence. Two additional patients, who were misdiagnosed as uterine leiomyoma, underwent hysterectomy

and BSO. Both patients died due to the disease (survival was 10 and 12 months, respectively). Furthermore, the research found that the prognosis of postmenopausal patients was poor [17].

UUS is a rare uterine sarcoma that encompasses a diverse group of neoplasms with no specific line of differentiation, and its diagnosis is made by exclusion [8]. UUS typically shows positive staining with p16 and focal positivity with CD10, PR, cyclin D1, and beta-catenin [18]. The most common cytogenetic abnormality observed in LGESS is a recurrent translocation involving chromosomes 7 and 17, t(7;17)(p15;q21), resulting in a fusion between JAZF1 and SUZ12. High-grade endometrial stromal sarcoma typically harbours the YWHAE-FAM22 genetic fusion as a result of t(10;17)(q22;p13) [1].

CONCLUSION(S)

Endometrial stromal neoplasms are rare uterine tumours that can have a fatal outcome. They can mimic a variety of benign and malignant neoplasms, making accurate diagnosis challenging. In the present case series, an equal number of Low-grade and High-grade Endometrial Stromal Sarcomas (LGESS and HGESS) were observed, while Endometrial Stromal Nodules (ESN) were the rarest. The age of presentation ranged from 34 to 75 years. Given that EST can resemble various abdominopelvic tumours, thorough pathological sampling is necessary to identify the characteristic histomorphology. Cyclin D1 and WT1 play important roles in differentiating LGESS from HGESS, as WT1 positivity is seen in LGESS and Cyclin D1 positivity is seen in HGESS. Tumour size and grade are two important factors for disease progression. Therefore, an integrated morphological and immunohistochemical study, including a large number of cases in the future, will be helpful in planning specific therapeutic strategies for each subcategory.

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