CASE REPORT

Sclerosing stromal tumour of the ovary: A case report and the review of literature

Özhan Özdemir, Mustafa Erkan Sarı, Ertuğrul Şen, Aslıhan Kurt, Ayşe Burcu İleri[1], Cemal Reşat Atalay

Departments of Obstetrics and Gynecology and [1]Clinics Pathalogy, Ankara Numune Training and Research Hospital, Ankara, Turkey

Address for correspondence:
Özdemir Özhan, MD,
Department of Obstetrics and Gynecology, Ankara Numune Training and Research Hospital, Ankara - 06010, Turkey.
E-mail: seyozi@hotmail.com

ABSTRACT

Sclerosing stromal tumours are rare benign ovarian neoplasms of the sex cord stromal that occur predominantly in the second and third decades of life. To date, 208 cases have been recorded in the literature. Most patients have menstrual irregularities and pelvic pain. Infertility and virilisation have also been described. In this article, histopathological features and differential diagnosis of the benign sclerosing stromal tumour were described together with the literature data. It is imperative to consider the differential diagnosis of a sclerozing stromal tumour of the ovary in a young woman with an ovarian tumour. A combination of morphological, immunohistochemical, radiological and clinical findings is needed in differentiating the tumour from thecoma, fibroma/fibrosarcoma, lipoid tumours and Krukenberg tumour.

Key words: Ovary, pelvic pain, sclerosing stromal tumours

INTRODUCTİON

Sclerosing stromal tumour (SST), which were defined by Chalvardjan and Scully in 1973 for the first time, is an extremely rare ovarian sex cord stromal tumour with distinctive pathological features and benign nature.[1] Since it was first described by Chavarjian and Scully, fewer than 200 cases have been described in the literature. Most patients are young, with 70% of whom being between 14 and 29 years.[2] The most frequent presenting complaint is menstrual irregularity and pelvic pain. Macroscopically, they are usually observed as solid and typically unilateral tumours. SST is usually hormonally inactive, but it has been reported that some cases are related to pregnancy and androgenic symptoms.[3]

Awareness of such entity is crucial because of its histopathologic similarity with other neoplastic and non-neoplastic lesions of the ovary. SST should be distinguished from malignant tumours, but it is difficult to diagnose before surgery by imaging studies. It used to be diagnosed by pathological examination during surgery or after surgery. In this study, the case of ovarian sclerosing stromal tumour is described and its clinicopathologic and immunohistochemical features are reviewed together with the literature data.

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CASE REPORT

A 25-year-old woman was presented with pelvic pain during the last 4 months. Physical examination revealed a large, palpable abdomino-pelvic mass. There was no unusual symptoms such as hypermenorrhoea, menstrual irregularities and virilisation. Ultrasound examination showed a 15 cm heterogeneous left ovarian tumour consisting of predominantly solid tissue with several loculated cysts. Laboratory tests including tumour markers and serum hormonal assays were normal in case. The patient underwent laparotomical left oophorectomy and showed a normal uterus and right ovary with the left ovary replaced by a solid mass. Ascites was not present. On gross inspection, the removed left ovarian mass measured 15 × 14 × 13 cm. The mass was grey-white in color and had a smooth and well-encapsulated surface. The cut surface was a mostly solid and slightly oedematous [Figure 1]. The mass was described as benign by frozen analysis. The histologic features included a pseudolobular pattern with widespread areas of sclerosis and a two-cell population of spindled and round cells. Haemangiopericytoma-like vessels, myxoid to fibrotic stroma and focal cystic change were noted. Mitoses and necrosis were absent. Immunohistochemical studies showed positive inhibin [Figure 2]. The final diagnosis was that of sclerosing stromal tumour of the ovary. Post-operative recovery was uneventful.

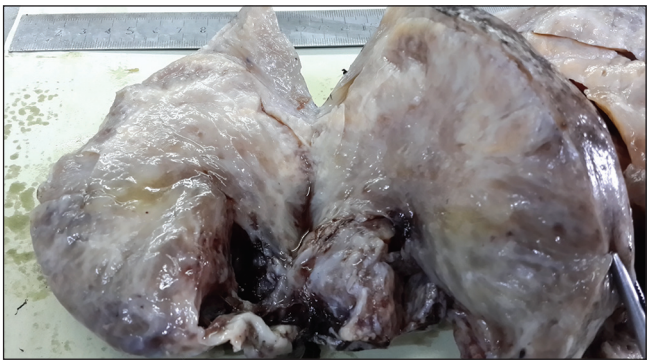

Figure 1: A, Macroscopically, the tumour involves the whole ovary and the sectioned surface is mostly solid, white and slightly edematous

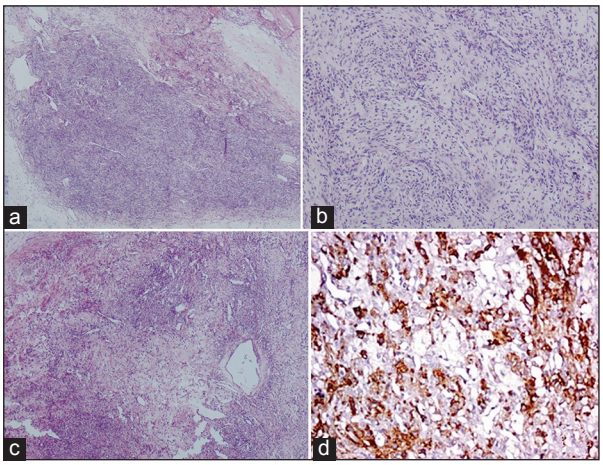

Figure 2: Sclerosing stromal tumour histopathologic features. (a) Pseudolobular pattern consisting of a hypercellular and hypocellular area (H and E, ×100). (b) Dual cell populations are seen; spindle cells and round cells (H and E ×200). (c) Hypocellular and hypercellular areas, and a conspicuous hemangiopericytoma-like vascular pattern (H and E, ×100). (d) Tumour cells show cytroplasmic staining for α-inhibin (IHC, ×200)

DISCUSSION

SST is attributable to theca cell-fibrous tumour subtypes of ovarian sex cord-stromal tumour from WHO-2003 classification that is distinctive clinical, pathologic and radiological features, which differentiates it from other stromal tumours.[4] The aetiology of SSTs is unknown. Based on the ultrastructural features, SSTs were thought to arise from pluripotent immature stromal cells of the ovarian cortex. However, SST has been proposed to stem from the perifollicular myoid stromal cells that are normally present in the theca externa.[5] The vascular, sclerotic and edematous stromal changes are constant features of these tumours and relate to the local elaboration of some vascular permeability and growth factors like vascular endothelial growth factor (VEGF).[6] On the other hand, Ismail *et al.* suggested that endocrine milieu might be responsible for the morphology of SST and they may be developed from preexisting ovarian fibromas.[3]

In the literature, reports of ovarian SSTs are rare. We undertook a MEDLINE® search using keywords ovarian neoplasms and sclerosing stromal tumour to obtain reports on this tumour in the English literature and then extended the search to related reports listed in their references. Until 2003, 114 cases had been reported by Peng *et al*.[7] We concluded that up to the writing of this paper, a total of 208 cases had been reported and cases identified between 2003 and 2014 were summarised in Table 1.

Ovarian SST occurs more commonly during the second to third decades of life with an average age of occurrence of 25, 9 years (4-73 years) and most of the reported cases have been unilateral. Bilateral SST was depicted in only four cases.[3,8-10] The most common signs and symptoms are a palpable pelvic mass, menstural irregularity, pelvic pain and non-specific symptoms related to the ovarian mass and our patient is complained of pelvic pain. Tumour size varies from 1cm to 31cm in diameter. Elevated serum CA125 level and/or ascites were depicted in some cases. Meigs' syndrome associated with SST of the ovary has been described in four cases.[11-14] Sclerosing stromal tumour with an ovarian torsion has been described in two cases.[15,16]

Sclerosing stromal tumours were reported in which the inactive tumours did not represent endocrine clinical symptoms. However, currently according to several reports, it is the active tumour that produce hormones (estrogenic or androgenic). These tumours synthesised dehydroepiandrosterone and that when steroidogenesis occurred, which caused irregular menses, amenorrhoea, infertility, precocious puberty and virilisation. Endometrial hyperplasia concomittent with SST have also been described which might indicate a status of excessive hormone production. Other authors have documented elevated levels of both estrogenic and androgenic hormones that were corrected after surgery. In several patients with irregular menses, normal menses following the excision of the tumour was noticed.[6] To date, 9 cases of virilising SST of the ovary have been described in the literature and three of the reported virilising SST were diagnosed during pregnancy [Table 2]. A virilising SST of the ovary in a young woman with Mc Cune Albright Syndrome was reported in 2013 by Bousssaid K *et al*.[17] In this case, neither hormonal activity nor virilisation was observed. Sclerosing stromal tumours are rarely seen together with pregnancies; only 15 reports of sclerosing stromal tumour of the ovary during pregnancy have been presented [Table 3].

SST can not predict its presence preoperatively on the basis of clinical and ultrasonographic findings alone. It is difficult to distinguish SST consisting of solid and cystic areas from ovarian malignancies on the basis of radiological and macroscopic examination, as these tumours additionally appear very vascular giving the impression of malignant

Table 1: Overview of all case reports on SST between 2003-2014

Case no and article	Age	Side	Clinic and symptom	CA-125	Tumour size (cm)	Gross apperance	Immunohistochemical staining
Peng HH *et al*., 2003	24	Left	İM	High	8,4	Solid	
Kim JY *et al*., 2003 (3 cases)	16	Left	İM	Normal	6	Solid	SMA(+), vimentin(+), PR(+)
	26	Left	İM	Normal	6	Solid	SMA(+), vimentin(+)
	39	Left	İM, pelvic pain	Normal	5,5	Cystic	
Kuscu E *et al*., 2003	34	Right	Hirsutism, İM	Normal		Solid	SMA(+), CK(-), S100(-), desmin(-)
Yerli H *et al*., 2003	34	Right	Amenorrhea, hirsutism	Normal	12,5	Solid	SMA(+), CK(-), S100(-), desmin(-)
Deval B *et al*., 2003	29	Right		Normal	4,5	Solid-cystic	
Huang SC *et al*., 2003	31		Pregnancy	High			
Calabrese M *et al*. 2004	30	Right	Pregnancy	Normal	14	Cyst	
Bildirici K *et al*.,2004	17		Meigs' syndrome				
Gurbuz A *et al*., 2004	21		Pregnancy		7	Solid	Vimentin(+), SMA(+), desmin(+)
Akbulut M *et al*., 2004	17	Right	İM, pelvic pain	Normal	10	Solid-cystic	İnhibin(+), vimentin(-), SMA(-), CK(-), ER(-), PR(-)
Bouraouis *et al*., 2004 (3cases)	15,26,56						
Akyildiz EU *et al*., 2004 (3 cases)	23,24,28						—
Mathur SR *et al*., 2004 (4 cases)							—
Kostopoulou E *et al*., 2004 (3 cases)							
Kurt G *et al*., 2004 (6 cases)	16-24	3 Right 3 Left	Pelvic pain, pregnancy (2)		6-14	Solid-cystic	ER(-), İnhibin 4 cases (+); calretinin, SMA, PR 3 cases (+)
Popovska S *et al*., 2005	26						—
Pai RR *et al*.,2005 (4 cases)	Mean 22,2	All case unilateral	İM		Mean 10	Solid-cystic (2); solid (1); unilocular cystic (1)	—
Korczyinski J *et al*.,2005	30	Right	İM, pelvic pain	Normal			
Terauchi F *et al*.	18	Right	Abdominal distention	High	20	Solid, cystic	CA125 (-)
Pai R *et al*., 2005 (4 cases)	Mean 22		Aymptomatic (2), İM (2)			Solid-cystic (2) Solid (1) Cystic(1)	
Arora R *et al*., 2008	25	Left	Abdominal pain		6	Solid-cystic	SMA(+),CD99(+), demsin(-), S-100 (-), EMA(-), CD34 (+)
Chang W *et al*., 2006	11	Bilateral		Normal	Left 8,5 right 17	Solid-cystic	SMA (+)
Sen N *et al*., 2006	25	Left	Aymptomatic		4,5	Solid	—
Jung NH *et al*. 2006	50	Right	Meigs' syndrome	High	18	Solid-cystic	Vimentin(+), reticulin(+), calretinin (+), CD34(-), SMA(-), S-100(-), inhibin (-)
Darghouth CL. *et al*., 2007	15						
Sharma M *et al*., 2007 (2 cases)	19	Left	Polymenorrhagia	Normal	6	solid	SMA (+)
	25	Left	Abdominal pain	Normal	6	Solid-cystic	SMA (+)
Iravanloo G *et al*., 2008	26	Left	Pelvic pain, İM	Normal	23	Solid	
Ergeneli MH *et al*., 2008	11						
Tomsová *et al*., 2008	17						
Stankovic Z *et al*., 2008			Ovarian torsion				
Youm HS *et al*., 2008	71	Left	Abdominal distention	Normal	44	Solid-cystic	Desmin(+), SMA(+),S100(-)
Arrora R *et al*., 2008							
Chang YW *et al*., 2009	12	Bilateral	Palpable mass	Normal	Right 13 Left 2	Solid	—
Wada H *et al*., 2009	52	Left	Abdominal distention	Normal	31	Solid	—
Uguralp *et al*., 2009	15	Right	İM, Pelvic pain,	High	4	Solid-cystic	—
Gwin K *et al*., 2009 (4 cases)							
Gulati A *et al*., (2009)							
He Y *et al*., 2010	4	Right	Premature thelarche				
Amorim-Costa C *et al*., 2010			Meigs' syndrome	High			
Zekioglu O *et al*., 2010 (14 cases)	16-54				6-21	8 Solid 6 Solid-cystic	İnhibin, vimentin, SMA, demsin, all cases (+); CD 99, 4 cases (+); ER all cases (-); PR 2 cases (+)
Qureshi *et al*., 2010 (16 cases)	23-40	All cases unilateral		7 cases High			All cases mucin (-)
Ismail SI *et al*., 2010							

(*Continued*)

Table 1: (*Continued*)

Case no and article	Age	Side	Clinic and symptom	CA-125	Tumour size (cm)	Gross apperance	Immunohistochemical staining
Park SM *et al*., 2011	11	Left	Hirsutism		9	Solid	Vimentin(+), SMA(+), inhibin(+), S100(-), CK(-)
Liou JH *et al*., 2011	18	Right	İM, pelvic pain, Meigs' Syndrome	High	16,5	Solid	İnhibin (+), Ca-125 (-), CK(-)
Dilbaz B *et al*., 2011	14	Right	Pelvic pain	Normal	7	Solid	—
Akbulut M *et al*., 2011	73	Left	Ovarian torsion		14	Solid-cystic	Calretinin(+), inhibin(+), ER(+), PR(+), CK 7(-), CD34(-), vimentin (-), SMA(-), S100(-), chromogranin (-), sinaptophysin (-)
Banik T *et al*., 2012 (3 cases)	19	Left	İM, abdominal pain		5	Solid	
	21	Right	İM		12	Solid-cystic	
	18		Abdominal pain	High	8,5	Solid	Inhibin(+), CK(-), vimentin(-), SMA(-), desmin (-), EMA(-)
Chung CP, *et al*., 2012	59	Left	İM		1,5	Solid	
Khanna M *et al*., 2012	32		İM, pelvic pain	High	16	Solid-cystic	SMA(+)
Suraweera P *et al*., 2012	33	Right	Hirsutism, virilisation	Normal	6	Solid-cystic	
Fotedar V *et al*., 2012	23		Abdominal pain, İM	High	4,7	Solid-cystic	Vimentin(+), inhibin(+), desmin (-)
Duzcu SE *et al*., 2012	17			Normal		Solid	Vimentin(+), calretinin(+), İnhibin (+), PR(+), SMA(+), ER(-), EMA(-), CK 7(-), CK 20(-), AFP(-)
Kim D *et al*., 2012	26	Right	Pregnancy	High	6,5	Solid	—
Mahadevappa A *et al*., 2012	16	Left	İM, pelvic pain, Meigs' Syndrome	High	17	Solid	—
Sayilgan AT *et al*., 2012	19	Right	Pelvic pain	High	10	Solid	Vimentin(+), SMA(+), CA125(-), CD34(-), CD99(-), CD10(-), EMA(-), VEGF(-), PLAP(-), CK(-), PAS (-)
Parlakgumus HA *et al*., 2013	24	Right	Pelvic pain	Normal	2	Solid	İnhibin(+), calretinin(+), vimentin (+), SMA(+), ER(+), PR(+), CD34 (+), desmin(-), CK(-), EMA(-)
Boussaid K *et al*., 2013	24	Left	McCune Albright syndrome, hirsutism, acne, amenorrhea			Solid	Inhibin(-), vimentin(-), CK(-), WT1(+)
Limaiem F *et al*., 2013 (2 cases)	16	Left	Pelvic pain	Normal		Solid	SMA (+), inhibin (+), vimentin (+), cytokeratin (-)
	45	Right	Pelvic pain	Normal		Solid-cystic	SMA(+), inhibin(+), vimentin(+), CK(-)
Kutuk MS *et al*., 2013							
Kaygusuz EI *et al*., 2013 (7 cases)	18-25	5 Right 2 Left	Pregnancy (3), İM (2), abdominal discomfort (2)	All cases normal	6-12	Solid (5), Solid-cystic (2)	Vimentin, SMA, desmin, inhibin, calretinin, PR all cases (+); ER, CK, CK-7 all cases (-) ; c-kit, melan-A 4 cases (+); CD-10 3 cases (+)
Amal Abd. 2014	19	Right	İM, pelvic pain		8,6	Solid	Calretinin(+), inhibin(+)
Liang YF *et al*., 2014	25	Right	Ectopic pregnancy	High	5	Solid-cystic	CD34(+), desmin(+), SMA(+), CD31(-), S-100(-), ER(-), PR(-)

EMA – Epithelial membrane antigen; CK – Cytokeratin; SMA – Smooth muscle actin; ER – Estrogen receptors; PR – Progesterone receptors; İM – Irregulary menstruation

Table 2: Case of virilising sclerosing stromal tumour (SST) of the ovary in the literature

Case	Age	Side	Clinic and symptom	CA–125	Tumour size (cm)	Gross apperance
Ismail *et al*., 1990	29	Bilateral	Pregnancy, virilisation		R 14; L 10,5	Solid
Cashell *et al*., 1991	27	Left	Pregnancy, hirsutism		3	Solid
Duska LR *et al*., 1998			Pregnancy, virilisation			
Andrade *et al*., 2001	18	Accessory ovary	Pelvic pain, hirsutism		16	Solid-cystic
Kuscu E *et al*., 2003	34	Right	Hirsutism, oligomenorrhea	N		Solid
Yerli H *et al*., 2003	34	Right	Hirsutism Amenorrhea	N	12,5	Solid
Park SM *et al*., 2011	11	Left	Hirsutism		9	Solid
Suraweera P *et al*., 2012	33	Right	Hirsutism, viriisation	N	6	Solid-cystic
Boussaid K *et al*., 2013	24	Left	McCune Albright syndrome, hirsutism, acne, amenorrhea			Solid

tumours. Ultrasonography is useful for distinguishing between cystic and solid masses, but can be undetermined in lesion characterisation, such that a differential diagnosis from malignant ovarian neoplasms is not always possible.

Table 3: Sclerosing stromal tumour (SST) of the ovary during pregnancy in the literature

Case	Age	Side	Clinic and symptom	CA–125	Tumour size (cm)	Gross apperance
Tiltman, 1985	18	Right	Pregnancy		20	Cystic
Tiltman, 1985	32	Right	Pregnancy		4	Solid
Ismail *et al*., 1990	29	Bilateral	Pregnancy, virilisation		R 14; L 10,5	Solid
Cashell *et al*., 1991	27	Left	Pregnancy, hirsutism		3	Solid
Duska LR *et al*., 1998			Pregnancy, virilisation			
Huang SC *et al*., 2003	31		Pregnancy	High		
Calabrese M *et al*. 2004	30	Right	Pregnancy, pelvic pain	Normal	14	Cyst
Gurbuz A *et al*., 2004	21		Pregnancy		7	Solid
Kurt G *et al*., 2004 (2/6 cases pregnant)	16-24 (mean 21)	3 Right 3 Left	Pregnancy (2 cases)		6-14 (mean 11)	Solid-cystic
Kim D *et al*., 2012	26	Right	Pregnancy	High	6,5	Solid
Kaygusuz EI *et al*., 2013	19	Left	Pregnancy	Normal	6	Solid
Kaygusuz EI *et al*., 2013	21	Right	Pregnancy	Normal	8,5	Solid
Kaygusuz EI *et al*., 2013	19	Right	Pregnancy	Normal	6	Solid-cystic
Liang YF *et al*., 2014	25	Right	Ectopic pregnancy	High	50	Solid-cystic

Ultrasonography and computed tomography findings of SST show an increased peripheral vascular as seen in malignant tumours. Magnetic resonance imaging findings include typical signal patterns such as hypointense nodules, hyperintense stroma, lobulation, strong enhancement with gadolinium and a peripheral hypointense rim are present.[18] The distinct histopathological appearance and immunohistochemistry of SST are important in aiding diagnosis.

Characteristic histological finding of the SST of ovary is the pseudolobular pattern that is formed by the cellular nodules that are separated from each other by hypocellular, oedematous and collagenous stroma. The hemangiopericytomatous pattern-like dilated vascular structures are the characteristics of cellular areas, and the luteinised theca-like cells with vacuolised cytoplasm and fusiform fibroblast-like are the characteristics of hypercellular areas.[19] Characteristic pathological findings of the SST of ovary were observed both macroscopically and microscopically in all the cases reported in literature.

Several immunohistochemical markers of the sex-cord stromal tumours were studied in SST. Immunohistochemical analysis for inhibin, smooth muscle actin (SMA), vimentin, estrogen receptors (ER) and progesterone receptors (PR) using formalin-fixed and paraffin-embedded materials showed predominant positivity for a SMA, consistent positivity for inhibin and vimentin, and negativity for S-100 protein and epithelial markers, suggesting a stromal origin of the SST.[16,19]

The differential diagnosis of SSTs should include thecoma/fibroma, metastases, and malignant epithelial ovarian tumours. In some cases, the differential diagnosis between SSTs and juvenile granulosa cell tumour with pronounced stromal sclerosis was difficult. However, the characteristic vascular pattern and mitotic activity were used in favor of SST, whereas tumours with follicular structures, higher mitotic activity and characteristic granulosa cell morphology were rather classified as Juvenile granulosa cell tumour.[20] Infrequently, the vacuolated cells and the presence of signet ring cells in association with oedematous stroma may be misdiagnosed as Krukenberg tumours, which could be differentiated by recourse to immunohistochemistry.[19] In the literature inhibin, calretinin, CD34, alpha glutathione S-transferase positivity (α-GST), melan-A, müllerian-inhibiting substance, WT-1 and CD99 was reported to be useful to differentiate STT from thecoma, fibroma and other sex cord stromal tumours.[3,19]

Inhibin has been shown to be useful marker for ovarian sex cord stromal tumours. Inhibin is a specific, but less sensitive marker than calretinin in the diagnosis of ovarian sex cord-stromal tumours. In addition, a correlation was observed between the calretinin and α-inhibin expressions and the luteinisation level of tumour cells.[19] Also, inhibin and calretinin have been shown to be more sensitive and specific marker than CD99, A103 (melan-A), CD10 and WT-1for ovarian sex cord stromal tumours.[3,19] CD34 stains the endothelium of often dilated and branching vascular architecture and clearly distinguishes SST from thecoma and fibroma. αGST positivity within scattered cells appears to be useful in the distinction of SST from diffuse staining thecomas and no staining fibromas.[21] Lifschitz–Mercer *et al*., proved that PR stained positively in SST cells.[22] Kostopoulou E *et al*., defined that a positivity for ER beta was observed in a significantly larger number of cells than that for ER alpha.[23] In addition, three copies of chromosome 12 in 13-21% of all examined SSTs tumour cells was reported using fluorescence *in situ* hybridisation (FISH) analysis.[6] One researcher described a patient with SST with monosomy of chromosome 16.[24] Although many studies showed variable immunohistochemical analysis

for sclerosing stromal tumours, a predominant positivity for inhibin, calretinin, smooth muscle actin and vimentin is a well-known immunohistochemical panel suggesting a stromal origin of the SST.

In conclusion, the patient's young age (generally, the second or third decade of life), the unilaterality of the tumour and the characteristic macroscopic and histopathological appearance of the tumour are essential characteristics for the diagnosis of SST of the ovary. SST is a very rare tumour but tumour markers, hormone tests and ultrasonography, in addition to MRI should be performed when the women are under the age of 30 and the visual inspection reveals 5 cm or more of a solid tumour. Frozen biopsies should also be performed during surgery. The distinct histopathological appearance and immunohistochemistry of SST are important in aiding diagnosis. All cases were diagnosed as benign except for one patient with lowgrade malignancy reported in 1990.[24] Surgical resection of the tumour is curative since to date, no local or distant recurrences have been reported in literature. A combination of morphological, immunohistochemical, radiological and clinical findings is needed in differentiating the tumour from thecoma, fibroma/fibrosarcoma, lipoid tumours and Krukenberg tumour.

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