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Case Report

Testicular microcystic stromal tumor: a case report with MRI findings and literature review

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Abstract: Microcystic stromal tumor (MST) is an extremely rare subtype of sex cord-stromal neoplasm, with the majority of published literature consisting of isolated case reports or small case series, underscoring its scarcity. Most documented cases involve ovarian MST, whereas only two prior instances of testicular MST have been reported. The histological features of testicular MST are analogous to those of ovarian MST, and its diagnostic criteria are extrapolated from the well-established guidelines for ovarian MST. However, the clinical and imaging characteristics of testicular MST remain poorly understood, leading to frequent underdiagnosis or misdiagnosis. Herein, we present a rare case of MST arising in the right testis of a 54-year-old Chinese male. Histopathologically, the tumor exhibited characteristic microcystic and stromal architectures, with immunohistochemical positivity for vimentin, CD10, cyclin D1, WT-1, and CD56, and a low Ki-67 proliferation index. Scrotal ultrasonography revealed a heterogeneous hypoechoic intratesticular mass containing both solid and cystic components. Magnetic resonance imaging (MRI) demonstrated a complex cystic lesion in the right testis, which showed slightly heterogeneous hyperintensity on T2-weighted images and hypointensity on T1-weighted images. This represents the first reported case of testicular MST with MRI findings, and its distinctive imaging features correlate with the tumor's unique histological structure. In this study, we comprehensively analyzed the clinical, radiological, histological, and immunohistochemical characteristics of this case and compared them with previously documented features in the literature, aiming to enhance the understanding of this rare entity and facilitate accurate diagnosis.

Keywords: Microcystic stromal tumor, testis tumor, magnetic resonance imaging

Introduction

Microcystic stromal tumor (MST) is an exceptionally rare sex cord-stromal neoplasm of the ovary, first delineated as a distinct entity by Irving et al. in 2009 [1]. Histopathologically, MST shows a reproducible triad composed of variably prominent microcystic spaces, solid cellular nodules, and hyalinized fibrous stroma, with the relative proportions of these elements differing from case to case. Immunohistochemically, MST exhibits a highly specific profile including diffuse nuclear β-catenin staining, combined with robust expression of vimentin, CD10, WT-1, and cyclin D1, while typical sex cord-stromal markers (e.g., inhibin, calretinin) are usually negative. Molecular studies have demonstrated the Wnt/β-catenin pathway as the central driver of MST tumorigenesis [2-5]. Most sporadic cases harbor somatic CTNNB1 mutations (more frequent than APC alterations), whereas a small subset of patients carry germline APC mutations or 5q deletions, suggesting that MST may represent an extragastrointestinal manifestation of familial adenomatous polyposis in selected individuals [6-10]. Notably, CTNNB1 and APC mutations are mutually exclusive, and no consistent morphological correlates have been identified between tumors with either alteration to date [6]. Clinically, ovarian MST typically presents as a unilateral, localized lesion in adults, lacks endocrine activity, and follows a benign clinical course after surgical resection [6].

Because of its rarity, ovarian MST has been documented in fewer than 70 cases in the literature. Testicular MST is even more extraor-

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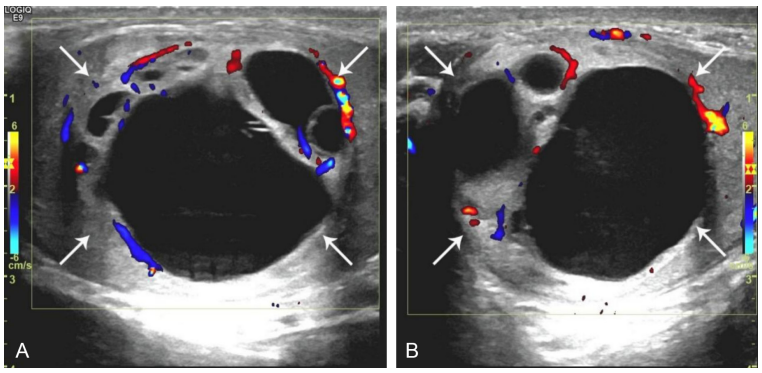

Figure 1. Ultrasound results. Ultrasonography revealed a multilocular cystic mass with solid components (shown by arrows). Significant aberrant blood flow indicators were observed in the solid regions of the bulk and the septa on color Doppler flow imaging. The tumor had distinct delineation. A. A longitudinal section image; B. A transverse section image.

dinary. Only two cases have been reported worldwide to date [11, 12]. The histopathologic features of testicular MST closely mirror those of its ovarian counterpart, and thus its diagnosis relies on the criteria originally established for ovarian MST. However, due to the extreme rarity of testicular MST, its clinical presentation, imaging characteristics, and biological behavior remain poorly elucidated. This knowledge gap poses significant challenges for preoperative diagnosis, often leading to underrecognition or misclassification as other testicular neoplasms (e.g., seminoma, Sertoli cell tumor, or cystic trophoblastic tumor). Many reports focused on gross and microscopic findings, with minimal data available on radiologic features, particularly magnetic resonance imaging (MRI), which is pivotal for preoperative tumor characterization and staging.

Radiologic information on MST is sparse. A PubMed search identified only three reports describing MRI findings in ovarian MST [13-15]. To the best of our knowledge, no case of testicular MST with detailed MRI characterization has been published. We recently encountered a case of testicular MST with classic morphologic and immunohistochemical features consistent with this entity. Herein, we present this case in detail, with particular emphasis on its MRI findings, and compare its clinicopathologic profile with the two previously reported testicular MSTs.

Case report

A 54-year-old male presented to the urology department with a painless enlargement of the right hemiscrotum, which had progressively increased in size over the past three years. The general physical examination yielded no significant findings. The patient reported no lower urinary tract symptoms, such as frequency, urgency, dysuria, or gross hematuria. The patient's medical history was significant for hypertension and cerebrovascular disease. The patient reported no history of cryptorchidism, scrotal trauma, scrotal infection, or genitourinary tuberculosis, and there was no known family history of neoplastic or hereditary disorders.

Upon examination, the right testis exhibited enlargement, firmness, and a lack of tenderness, with the patient indicating a sensation of heaviness. A palpable solid intratesticular mass exhibited an indistinct interface with the epididymis. The results of the transillumination and lifting tests were negative. The left testis appeared clinically normal; however, a varicocele of the left spermatic vein was identified, with no associated tenderness. No palpable inguinal lymphadenopathy was observed.

Scrotal ultrasonography demonstrated a predominantly cystic, heterogeneous hypoechoic mass that occupies a significant portion of the right testis, measuring approximately 3.2 × 2.7 cm. The lesion comprised multilocular cystic spaces interspersed with hypoechoic solid components. Several irregularly thickened septa were observed within the cystic portion. Color Doppler imaging revealed blood flow signals along the septa and cyst wall, as well as within the solid regions, indicative of a vascularized solid-cystic tumor. The mass exhibited a distinct margin and a regular overall contour (**Figure 1**).

An MRI was conducted for additional characterization. A cystic-solid intratesticular mass was observed in the right testis, measuring approximately 2.8 × 3.2 × 3.0 cm. On T2-weighted imaging, the lesion exhibited a multilocular cystic structure with thick septa and a somewhat identifiable solid component of relatively low signal strength, displaying a moderately heterogeneous look (**Figure 2A**, **2B** and **2D**). The primary cystic region exhibited uniform low signal

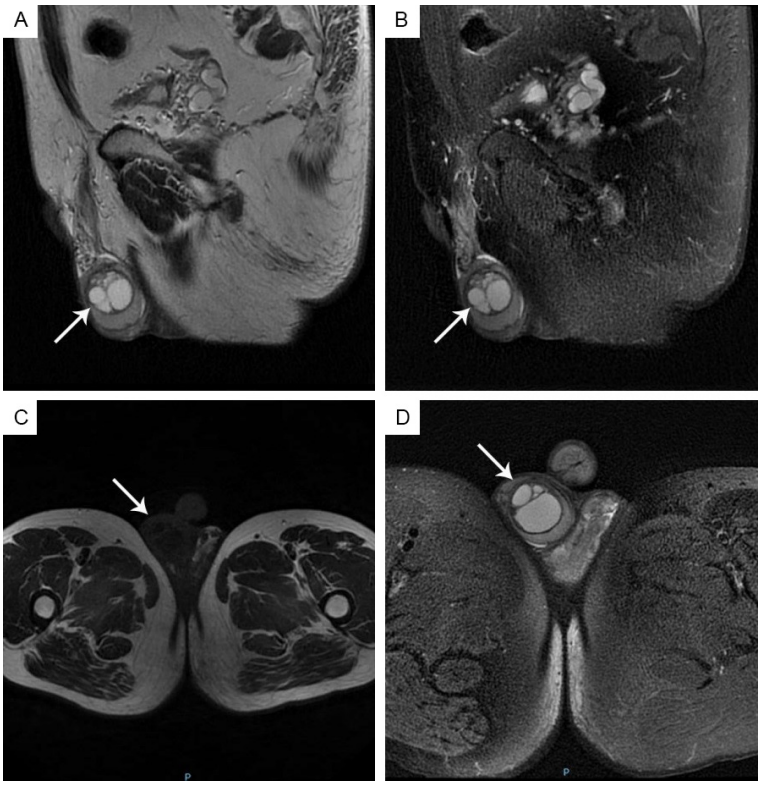

Figure 2. Magnetic resonance imaging depicting a complicated multilocular cystic-solid mass in the right testis. The images are (A) a sagittal T2-weighted picture, (B) a sagittal T2-weighted image with fat saturation, (C) an axial T1-weighted image, and (D) an axial T2-weighted image with fat saturation.

strength on T1-weighted images (**Figure 2C**). The contralateral testis appeared normal, and a left-sided varicocele of the spermatic vein was observed once more. No hypertrophied inguinal lymph nodes were detected. Diffusion-weighted pictures exhibited inferior quality and were not amenable to reliable evaluation.

Laboratory analyses indicated normal serum concentrations of carcinoembryonic antigen (CEA), alpha-fetoprotein (AFP), β-human chorionic gonadotropin (β-HCG), and lactate dehydrogenase (LDH). Renal and hepatic function tests, together with standard blood and urine studies, were within reference ranges. The patient was admitted for surgical intervention and had a right inguinal radical orchiectomy with high ligation of the spermatic cord.

A cystic-solid tumor affecting the right testis was found intraoperatively. The right epididymis and spermatic cord seemed macroscopically normal. The gross examination of the resected testis revealed a multilocular cystic-solid mass, approximately 3 cm in the greatest dimension, encompassing the majority of the testicular parenchyma. The cyst walls and septa exhibited irregular thickness.

The lesion exhibited the typical features of MST (**Figure 3**). Tumor cells exhibited a proliferative growth pattern and were intermingled with bands of hyalinized stroma. The tumor exhibited microcystic gaps, a reticular framework, and sheet-like regions of neoplastic cells. The cells presented relative uniformity, characterized by moderate quantities of coarsely granular eosinophilic cytoplasm and mostly bland oval nuclei, some of which displayed visible nucleoli. Mitotic figures were infrequent, and tumor necrosis was minimal. Stromal bleeding was observed. The spermatic cord and epididymis were devoid of neoplasm.

Immunohistochemical staining revealed widespread strong positivity for CD10, vimentin, and WT-1, together with nuclear expression of cyclin D1. CD56 exhibited focused positivity. The tumor cells tested negative for sex cord-stromal markers (inhibin and calretinin) and germ cell tumor markers (AFP, OCT4, SALL4). Supplementary negative markers were epithelial markers (CK, CK5/6), neuroendocrine markers (S-100, NSE), vascular markers (CD31, CD34, ERG), and a collection of other markers (desmin, SMA, GPC-3, D2-40, CD117). The Ki-67 labeling index was almost 1%. A diagnosis of testicular MST was established based on these findings.

Based on the histopathological diagnosis, we advised genetic counseling and the evaluation of germline testing for APC and associated genes. Currently, targeted sequencing of CTNNB1/APC is not regarded as a standard clinical assay for sex cord-stromal cancers, and further molecular testing would not have affect-

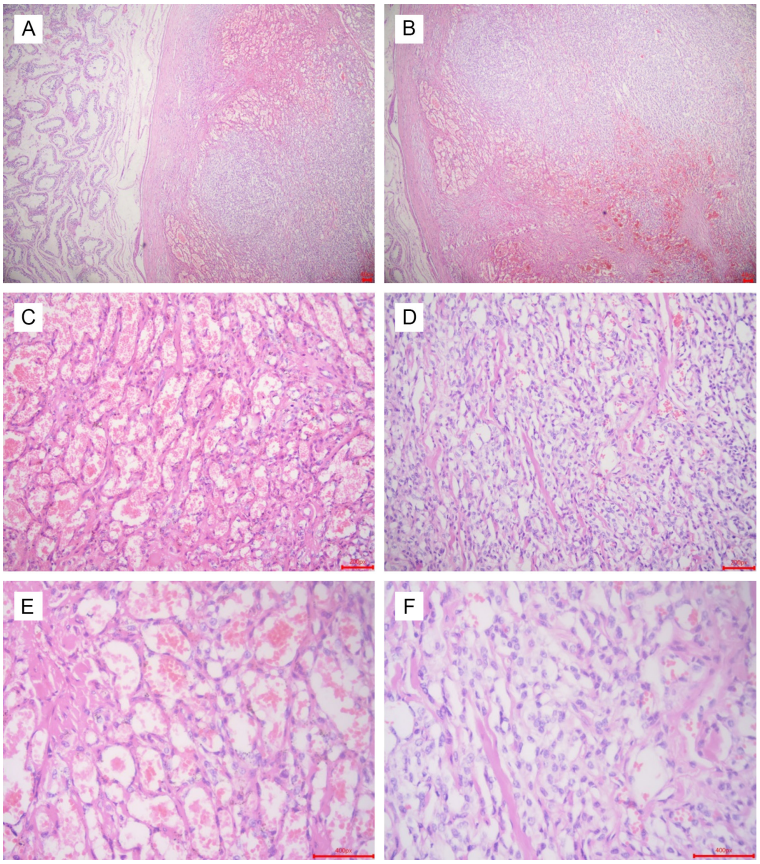

Figure 3. Photomicrographs of the right testicular microcystic stromal tumor. The lesion in the right testis consisted of microcysts, solid cellular regions, and fibrous/hyalinized stroma. The neoplasm comprised homogeneous cells featuring mild cytoplasm and uniform oval nuclei. The tumor cells exhibit microcystic, reticulated, and sheet-like arrangements, with associated hemorrhaging in the stroma. Mitotic activity was limited. No tumor necrosis was present. (A) and (B) at 40× magnification; (C) and (D) at 200× magnification; (E) and (F) at 400× magnification.

ed the initial surgical therapy. Consequently, no molecular testing was conducted. This signifies a constraint of the current findings, since we were unable to directly exhibit CTNNB1 or APC mutations to validate the activation of the Wnt/β-catenin pathway in this tumor. Nonetheless, the diagnosis of MST was deemed definitive due to the presence of distinctly microcystic architecture, hyalinized stroma, and inconspicuous stromal cells exhibiting a characteristic immunophenotype (positive for CD10, vimentin, WT-1, and cyclin D1, alongside the absence of sex cord and germ cell markers), consistent with contemporary WHO diagnostic standards. The postoperative course was unremarkable, no adjuvant therapy was provided, and the patient remained disease-free three months post-surgery.

Discussion

Sex cord-stromal tumors may develop in both male and female gonads; however, they are rare in the testis, comprising only approximately 2-5% of all testicular neoplasms [16]. Microcystic stromal tumor is a newly identified and exceedingly rare entity within this category. Since the initial series of 16 ovarian cases published by Irving and Young in 2009, less than 70 ovarian MSTs have been recorded globally [1]. The 2020 WHO classification of female genital malignancies categorizes MST as a unique pure stromal tumor within the range of ovarian stromal neoplasms [17]. In this context, the incidence of MST in the testis is exceptionally uncommon, and each new case significantly enhances our comprehension of its clinicopathological spectrum.

MST is histologically defined by a consistent triad of architectural and cytological characteristics: a microcystic or reticular arrangement, interspersed solid cellular regions, and notable hyalinized or fibrous stroma [1]. The tumor cells generally exhibit homogenous, coarsely eosinophilic cytoplasm and bland, round to oval nuclei with inconspicuous nucleoli, whereas mitotic figures are sparse. Immunohistochemically, MST exhibits a unique profile characterized by widespread expression of vimentin, CD10, WT-1, and cyclin D1, alongside nuclear accumulation of β-catenin. In contrast, markers indicative of sex cord differentiation (including inhibin and calretinin) and germ cell markers (AFP, OCT4, SALL4) are absent [2, 3, 18, 19]. The amalgamation of these symptoms enables MST to be identified with considerable confidence in standard practice, as evidenced by the existing WHO diagnostic criteria [17].

The primary molecular event in MST seems to be the deregulation of the Wnt/β-catenin sig-

Table 1. Overview of clinical characteristics of patients with testicular MST

Case	Age (yr)	Clinical presentation	location	Tumor size	Imaging finding	Surgy status	Association	Follow-up (months)
1	33	No symptoms	R testis	3 cm	Solid-cystic	UO	None	NED (12 m)
2	Late 30s	Palpable mass	R testis	3.9 cm	Solid-cystic	UO	None	NED (12 m)
3	54	Scrotal magnify	R testis	3.2 cm	Solid-cystic	UO	None	NED (3 m)

Note: UO, unilateral orchidectomy; NED, no evidence of disease.

naling pathway. Somatic missense mutations in CTNNB1 affecting exon 3 have been found in most documented ovarian MSTs, but a minority cases have mutually exclusive changes in APC [2, 9, 20-23]. Both genes are involved in the β-catenin destruction complex; their disruption results in the stability and nuclear accumulation of β-catenin, subsequently promoting the transcription of downstream targets, such as cyclin D1 [5]. Nuclear β-catenin and pronounced cyclin D1 staining serve as surrogate indicators of Wnt pathway activation [2]. Molecular investigation of CTNNB1 or APC was not conducted in this instance, as such testing is not routinely performed for female cord-stromal cancers at our institution, and the findings would not have influenced the immediate surgical strategy. This signifies a constraint regarding the genotype-phenotype connection, as we are unable to accurately identify the underlying mutation in this specific tumor. The diagnosis of MST is predominantly based on morphological and immunophenotypic characteristics rather than molecular analysis. The presence of a classic microcystic-stromal architecture coupled with a distinctive immunoprofile (positivity for CD10, vimentin, WT-1, and cyclin D1, without sex cord and germ cell markers) meets the current diagnostic criteria. The absence of molecular testing limits mechanistic interpretation but does not compromise the diagnostic integrity in this patient.

MST is often localized to the ovary, with its presence in the male gonad having been acknowledged very lately. Yet, only two cases of testicular origin have been documented in the literature. Zhu et al. reported a 33-year-old Chinese male with a 3-cm cystic-solid mass in the right testis, exhibiting the characteristic triad of microcysts, solid stromal regions, and fibrous stroma, alongside the expression of vimentin, CD10, nuclear β-catenin, and cyclin D1; targeted sequencing revealed a CTNNB1 c.110C>G mutation [11]. Hoogland et al. subsequently documented a right testicular MST in a Caucasian man in his late forties, exhibiting characteristic microcystic stromal histology and positivity for CD10, vimentin, and β-catenin, along with a CTNNB1 exon 3 c.98C>T point mutation identified through molecular analysis [12]. This tumor is, to our knowledge, the third recorded instance of testicular MST. **Table 1** delineates and contrasts the clinical characteristics of the three instances.

The morphological results in our patient closely resemble those observed in previously documented testicular cases and in ovarian MST. The tumor consisted of microcystic and reticular regions interspersed with solid cellular zones inside hyalinized stroma. The malignant cells exhibited cytological blandness, mild eosinophilic cytoplasm, oval nuclei, and few mitoses, with an absence of necrosis. The lesion exhibited strong diffuse immunohistochemical staining for CD10, vimentin, WT-1, and cyclin D1, although markers indicative of sex cord differentiation (inhibin, calretinin) and germ cell origin (AFP, OCT4, SALL4) were absent. Focal CD56 positivity was noted, consistent with the sporadic expression documented in ovarian MST [10, 18, 22, 24]. The Ki-67 labeling index was roughly 1%, akin to the modest proliferation rates reported in the majority of ovarian MSTs [6, 18, 20, 25]. The immunohistochemistry profile of the three testicular cases (**Table 2**) is thus remarkably consistent with that of ovarian MST and successfully rules out additional microcystic or cystic stromal and germ cell tumors of the testis. When these distinctive histological and immunophenotypic attributes are evident, MST is unlikely to create a significant diagnostic difficulty.

Preoperative serum tumor indicators are essential in the evaluation of testicular neoplasms. In our instance, AFP, β-hCG, LDH, and CEA levels were all within normal ranges, consistent with the prior two testicular MST cases

Table 2. Synopsis of immunohistochemistry observations in patients with testicular MST

Case	VIM	CD10	Cyclin D1	βcat	WT-1	CD56	AR	SF-1	CK	S100	Ki-67
1	+	+	+	+	-	-	NA	NA	-	NA	< 10%
2	NA	+	NA	+	NA	NA	+	+	+F	+F	NA
3	+	+	+	NA	+	+W	NA	NA	-	-	1%+

Note: VIM, vimentin; CD10, cluster designation 10; βcat, β-Catenin; WT-1, Wilms tumor 1; AR, androgen receptor; SF-1, steroidogenic factor 1; CK, cytokeratin; NA, not applicable; F, focal expression; W, weak expression.

[11, 12]. This observation highlights that normal tumor markers do not rule out the existence of a stromal tumor like MST. The European Association of Urology endorses scrotal ultrasonography as the principal imaging technique for assessing testicular masses, as it accurately evaluates tumor size, location, echotexture, and cystic components [26]. All three documented testicular MSTs had imaging results indicating a unilateral, well-defined lesion in the right testis characterized by a mainly cystic structure and a varied solid component, with negative serum markers. Notwithstanding these indicative characteristics, ultrasonography alone lacks the requisite specificity to differentiate MST from other benign or malignant conditions, hence impeding effective organ-sparing therapy.

Radiologic data regarding MST, including in the ovary, is limited. Only three ovarian MSTs with MRI characteristics have been documented, exhibiting primarily cystic lesions with elevated signal intensity on T2-weighted imaging and iso- to hypointense signal on T1-weighted sequences, frequently accompanied by septations and little solid components [13-15]. To our knowledge, no comprehensive MRI characterization of a testicular MST has been previously published before this publication. The MRI of our patient revealed a multilocular cystic-solid intratesticular mass exhibiting consistently low T1-weighted and heterogeneously high T2-weighted signals, accompanied by thickened septa and a peripheral solid component with a slightly lower T2 signal. These characteristics reflect the fundamental microcystic structure and fibrous stroma identified histologically. While CT was not conducted in our case, Zhu et al. documented a mixed solid-cystic mass in the right testis via CT in their patient [11]. The available data indicate that testicular MST typically appears radiologically as a well-defined, predominantly cystic lesion with a solid stromal component. Although this appearance is not pathognomonic, it may prompt radiologists to consider a benign stromal process when correlated with clinical and laboratory findings.

However, the imaging characteristics of MST coincide with various other mixed solid-cystic testicular diseases, necessitating meticulous differential diagnosis. Leydig cell cancers generally manifest as solid hypoechoic nodules on ultrasound and exhibit isointensity on T1-weighted MRI and hypointensity on T2-weighted MRI compared to normal testicular parenchyma [27]. Teratomas and mixed germ cell tumors frequently exhibit a complicated mixture of cystic regions, solid tissue, bleeding, and necrosis, characterized by a more varied echotexture and enhanced patterns compared to MST. Epidermoid cysts typically have a “target” or “onion-skin” appearance on ultrasonography and MRI, which was absent in our case. Testicular tuberculosis typically manifests alongside systemic symptoms, may exhibit calcifications and impaired diffusion, and is often associated with hydrocele. Testicular lymphoma often affects both testes, manifests as a mostly solid mass, and exhibits low T2-weighted signal intensity with significant diffusion restriction. Consequently, while imaging helps refine the differential diagnosis, a conclusive diagnosis of MST necessitates histopathological and immunohistochemical evaluation.

Clinically, all three patients with testicular MST exhibited painless unilateral scrotal enlargement or a palpable mass, and none displayed particular symptoms or serum markers to differentiate MST from other testicular neoplasms. Currently, there are no evidence-based protocols for the management of testicular MST. Radical orchiectomy has been the preferred treatment in all documented cases, including ours [11, 12]. The limited follow-up data is encouraging. Both previously documented patients were alive without recurrence at 12 months, and our patient is disease-free 3 months post-surgery. Based on these data and

by analogy with other benign or low-grade sex cord-stromal tumors, a practical surveillance strategy would entail physical examinations and serum measurements of AFP, β-hCG, and LDH every three months during the first year, every six months in years two to three, and annually thereafter for a minimum of five years. Scrotal ultrasonography may be conducted at 6 and 12 months during the initial year and annually thereafter, or sooner if new symptoms develop. Cross-sectional imaging of the abdomen and pelvis should be limited to patients exhibiting unusual characteristics or clinical indications of relapse. This systematic follow-up procedure balances the early diagnosis of uncommon relapse with the low but unclear malignant potential of this tumor.

A crucial translational inquiry is whether testis-sparing surgery may be safely administered to specific individuals with suspected MST, especially younger men for whom the preservation of endocrine and reproductive function is paramount. The consistently favorable clinical trajectory observed thus far, along with the characteristic imaging profile - a small, unilateral, well-defined cystic-solid lesion with negative tumor markers - indicates that organ-preserving surgery may be viable in selectively chosen instances. In cases of a solitary testis, bilateral lesions, or significant reproductive issues, enucleation of the mass with a margin of normal parenchyma, along with intraoperative frozen-section analysis, may be contemplated when a benign sex cord-stromal tumor is highly suspected. If frozen-section analysis indicates MST or a similar benign stromal tumor, testis-sparing surgery may ensure sufficient local control while preserving hormonal and reproductive function. Nonetheless, existing evidence is confined to radical orchiectomy instances, and there are no published data regarding outcomes following organ-preserving surgeries in testicular MST. The anticipated collection of further cases, along with comprehensive recording of surgical techniques, margin status and long-term outcomes, is crucial prior to endorsing testis-sparing surgery as a normal practice.

This research possesses multiple limitations. The MRI methodology lacked a dynamic contrast-enhanced series, which could have offered supplementary insights into vascularity and enhancement kinetics. Secondly, as previously mentioned, molecular testing for CTNNB1 or APC was not conducted because these tests are not routinely accessible for female cord-stromal malignancies at our hospital and would not have impacted the initial surgical therapy. Therefore, we cannot provide direct genotypic data to the expanding literature on Wnt/β-catenin pathway modifications in MST. The follow-up duration in our study remains brief. Due to the extraordinary rarity of testicular MST and the little observation period for all three described patients, our comprehension of its genuine long-term behavior is still inadequate. Ongoing monitoring of current cases and documentation of new instances, preferably incorporating histological, immunohistochemical, molecular and radiological data, will be essential to enhance risk stratification, improve follow-up strategies and assess the safe adoption of organ-preserving methods in selected patients.

Disclosure of conflict of interest

None.

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References

[1] Irving JA and Young RH. Microcystic stromal tumor of the ovary: report of 16 cases of a hitherto uncharacterized distinctive ovarian neoplasm. Am J Surg Pathol 2009; 33: 367-375.

[2] Rizzo NM, Sholl LM, Kao CS, Cornejo KM, Sangoi AR, Hirsch MS, Collins K, Gordetsky JB, Reyes Curcio FA, Fletcher CDM, Ulbright TM and Acosta AM. Molecular correlates of aggressive behavior and biological progression in testicular sertoli cell tumors. Mod Pathol 2023; 36: 100152.

[3] Sanceau J, Poupel L, Joubel C, Lagoutte I, Caruso S, Pinto S, Desbois-Mouthon C, Godard C, Hamimi A, Montmory E, Dulary C, Chantalat S, Roehrig A, Muret K, Saint-Pierre B, Deleuze JF, Mouillet-Richard S, Forné T, Grosset CF, Zucman-Rossi J, Colnot S and Gougelet A. DLK1/DIO3 locus upregulation by a β-catenin-dependent enhancer drives cell proliferation and liver tumorigenesis. Mol Ther 2024; 32: 1125-1143.

[4] Zhang R, Li S, Schippers K, Li Y, Eimers B, Lavrijsen M, Wang L, Cui G, Chen X, Peppelenbosch MP, Lebbink JHG and Smits R. Analysis

of tumor-associated AXIN1 missense mutations identifies variants that activate β-catenin signaling. Cancer Res 2024; 84: 1443-1459.
[5] Erazo-Oliveras A, Muñoz-Vega M, Mlih M, Thiriveedi V, Salinas ML, Rivera-Rodríguez JM, Kim E, Wright RC, Wang X, Landrock KK, Goldsby JS, Mullens DA, Roper J, Karpac J and Chapkin RS. Mutant APC reshapes Wnt signaling plasma membrane nanodomains by altering cholesterol levels via oncogenic β-catenin. Nat Commun 2023; 14: 4342.
[6] Parra-Herran C. Endometrioid tubal intraepithelial neoplasia and bilateral ovarian microcystic stromal tumors harboring APC mutations: report of a case. Int J Gynecol Pathol 2022; 41: 337-342.
[7] Parra-Herran C and McCluggage WG. Ovarian microcystic stromal tumour: from morphological observations to syndromic associations. Histopathology 2022; 80: 898-904.
[8] Sharma A, Zalejski J, Bendre SV, Kavrokova S, Hasdemir HS, Ozgulbas DG, Sun J, Pathmasiri KC, Shi R, Aloulou A, Berkley K, Delisle CF, Wang Y, Weisser E, Buweneka P, Pierre-Jacques D, Mukherjee S, Abbasi DA, Lee D, Wang B, Gevorgyan V, Cologna SM, Tajkhorshid E, Nelson ER and Cho W. Cholesterol-targeting Wnt-β-catenin signaling inhibitors for colorectal cancer. Nat Chem Biol 2025; 21: 1376-1386.
[9] Mikkelsen MK, Pinto SN, Chiang J, Shimony N and Gajjar A. Evolving biology and therapy of WNT-activated medulloblastoma. J Natl Compr Canc Netw 2025: 23: e257061.
[10] McCluggage WG, Chong AS, Attygalle AD, Clarke BA, Chapman W, Rivera B and Foulkes WD. Expanding the morphological spectrum of ovarian microcystic stromal tumour. Histopathology 2019; 74: 443-451.
[11] Zhu P, Duan Y, Ao Q and Wang G. Microcystic stromal tumor of testicle: first case report and literature review. Cancer Res Treat 2018; 50: 1452-1457.
[12] Hoogland M, Bleumer I and Suurmeijer A. Microcystic stromal tumour of testis. BMJ Case Rep 2022; 15: e248498.
[13] Murakami M, Wroblewski J and Kawagoe H. Microcystic stromal tumor resected by laparoscopic surgery. Gynecol Minim Invasive Ther 2017; 6: 135-138.
[14] Arafah MA, AlBreacan L, Akkour K and Alomar S. A 46-year-old woman with primary infertility and a diagnosis of microcystic stromal tumor of the ovary confirmed by histology and gene sequencing: a case report and review of the literature. Ame J Case Rep 2021; 22: e933528.
[15] Tsuji T, Oki N, Maeda T, Watanabe T, Inagaki M and Yoshida S. Huge ovarian microcystic stromal tumor presenting as acute abdomen in a patient with familial adenomatous polyposis: a case report with magnetic resonance imaging findings. Cureus 2024; 16: e72422.
[16] Acosta AM, Idrees MT, Berney DM and Colecchia M. Contemporary updates on sex cord-stromal tumors of the testis. Adv Anat Pathol 2024; 31: 126-135.
[17] Höhn AK, Brambs CE, Hiller GGR, May D, Schmoeckel E and Horn LC. 2020 WHO classification of female genital tumors. Geburtshilfe Frauenheilkd 2021; 81: 1145-1153.
[18] Deng L, Feng D, Liang J, Luo J and Ling B. Ovarian microcystic stromal tumor: a case report and literature review. Front Med (Lausanne) 2020; 7: 58.
[19] Bandhon BZ, Navaneethan S and Patton D. A case report on the microcystic stromal tumor of the ovary: a rare type of ovarian tumor. Int J Gynaecol Obstet 2024; 167: 455-460.
[20] Donthi D, Chen H, Peng Y and Niu S. Ovarian microcystic stromal tumor with intraovarian recurrence and peritoneal and omental spread: a case report with morphological, immunohistochemical, and molecular analysis. Int J Gynecol Pathol 2023; 42: 491-495.
[21] Kojima N, Yoshida H, Uno M, Hiranuma K, Naka T, Shiraishi K and Kato T. Microcystic stromal tumor of the ovary: a recurrent case with somatic CTNNB1 missense mutation. Virchows Arch 2022; 481: 799-804.
[22] Dantzer C, Dif L, Vaché J, Basbous S, Billottet C and Moreau V. Specific features of ß-catenin-mutated hepatocellular carcinomas. Br J Cancer 2024; 131: 1871-1880.
[23] Gill SJ, Pirzada A, Power P and Neveu J. A constellation of rare gynecological malignancies and familial adenomatous polyposis gastrointestinal adenocarcinoma: a case report. Int J Gynecol Pathol 2022; 41: 622-627.
[24] Chen Q, Lu W and Lv W. Overlap of microcystic stromal tumor and primary solid pseudopapillary neoplasm of the ovary. Int J Clin Exp Pathol 2015; 8: 11792-11797.
[25] Wu B, Wu C, Li D, Yang Z, Liu Y, Zhang HX, Xin HW and Bai Y. Ovarian microcystic stromal tumor with significant nestin expression: a unique case. Intern Med 2024; 63: 2781-2785.
[26] Albers P, Albrecht W, Algaba F, Bokemeyer C, Cohn-Cedermark G, Fizazi K, Horwich A, Laguna MP, Nicolai N and Oldenburg J; European Association of Urology. Guidelines on testicular cancer: 2015 update. Eur Urol 2015; 68: 1054-1068.
[27] Cassidy FH, Ishioka KM, McMahon CJ, Chu P, Sakamoto K, Lee KS and Aganovic L. MR imaging of scrotal tumors and pseudotumors. Radiographics 2010; 30: 665-683.