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Case Report

Report of two primary renal tumors with myxoid features. Differential diagnosis between benign and malignant entities

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Summary
Renal mesenchymal neoplasms are rare entities which can have a benign or a malignant behavior. Herein we describe two renal mesenchymal tumors with myxoid stroma, investigating the wide spectrum of differential diagnosis. With our first case we considered some benign entities such as myxoma, myxoid leiomyoma, and mixed epithelial and stromal tumor; with our second case we considered some sarcomas with myxoid features such as myxofibrosarcoma, low-grade fibromyxoid sarcoma, dedifferentiated liposarcoma, and myxoid liposarcoma. During the diagnostic process, it is important to integrate histopathological, immunohistochemical, and molecular data in order to avoid misdiagnosis. We concluded our second case report was a myxofibrosarcoma grade 1. To the best of our knowledge, we described the fourth primary renal myxofibrosarcoma reported in literature.

Key words: myxoma, myxoid leiomyoma, MEST, myxofibrosarcoma, renal myxoid neoplasm

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Conflict of interest
The Authors declare no conflict of interest.

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Introduction

Renal mesenchymal tumors with myxoid features are rare [1]. When approaching such lesions, both benign and malignant soft tissue neoplasms must be considered [2]. Among indolent tumors the differential diagnosis narrows to myxoma, myxoid neurofibroma, perineuroma, myxoid leiomyoma, and myxolipoma [3]. Primary renal sarcomas account for around 1% of all primary renal malignancies [4]. Many malignant entities may show myxoid stroma such as myxoid liposarcoma, myxofibrosarcoma (MFS), low-grade fibromyxoid sarcoma (LGFMS), leiomyosarcoma, rhabdomyosarcoma, and extra skeletal chondrosarcoma [4]. Besides mixed epithelial and stromal tumor (MEST), a biphasic entity behaving mostly in a benign fashion, may show myxoid change in the mesenchymal component [5]. For a correct diagnosis, the integration of histopathological, immunohistochemical, and molecular data is mandatory.

First case report

Our first case report is a 44-year-old woman with a left renal cystic mass on the lower pole (Bosniak category III-IV). The patient underwent enucleation of the renal lesion which broke during laparoscopic extraction due to its consistency. On gross examination we observed

multiple fragments with an overall axis of 18 cm and a gelatinous appearance except for a vorticoid and whitish nodular area (Fig. 1A).
Histologic examination revealed a mesenchymal lesion with fusiform neoplastic cells sometimes marked by microvacuole and with slight atypia (Fig. 1D). These elements were dispersed in an abundant myxoid stroma with no necrosis or significant mitotic activity (< 1 mitosis/HPF); rare elements were positive for Ki-67 (Fig. 1B-C).
We confirmed the mesenchymal nature of the spindle cell component using immunohistochemistry as they showed strong positivity for vimentin, desmin, and alpha-smooth muscle actin (α-SMA) (Fig. 2). The neoplastic spindle cells were negative for CD34, neurofilament protein, S100, and WT-1.
Although we evaluated the neoplasm in fragments, we did not recognize a tumoral pseudocapsule, but rather pushing borders with some occasional infiltrative foci of spindle cells.
There were also some tubular structures, isolated or combined in aggregates, composed of cells with epithelial morphology, without significant atypia and without cilia (Fig. 1B). We were not sure whether to interpret these tubular structures as embryological remnants, normal renal tubules entrapped by the neoplasia, or as an epithelial component of the tumor. Immunohistochemistry was not conclusive as the tubular structures were positive for CK 8-18, EMA, PAX8, and focally for GATA-3 (Fig. 2D), thus confirming their epithelial nature but not completely their origin. In fact, GATA-3 is normally present in collecting duct and distal renal tubules. However, it can also be co-expressed with PAX8 in the epithelial component of MESTs [6].
The remaining renal parenchyma was atrophic.

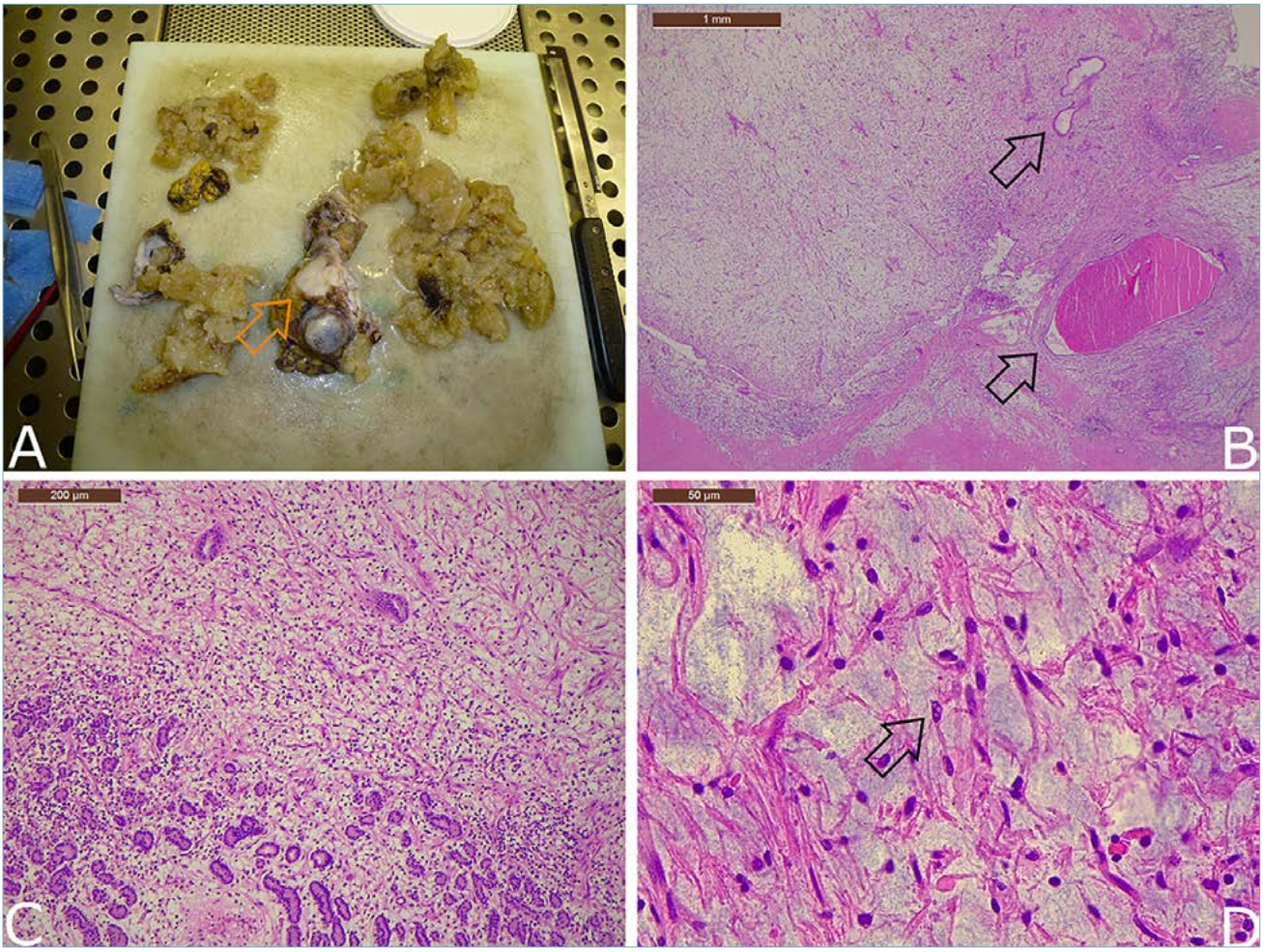

Figure 1. (A) Gross appearance: the arrow indicates the vorticoid and whitish nodular area; (B) Microscopic appearance: arrows indicate the epithelial component; (C) Myxoid stroma; (D) The arrow indicates some microvacuoles.

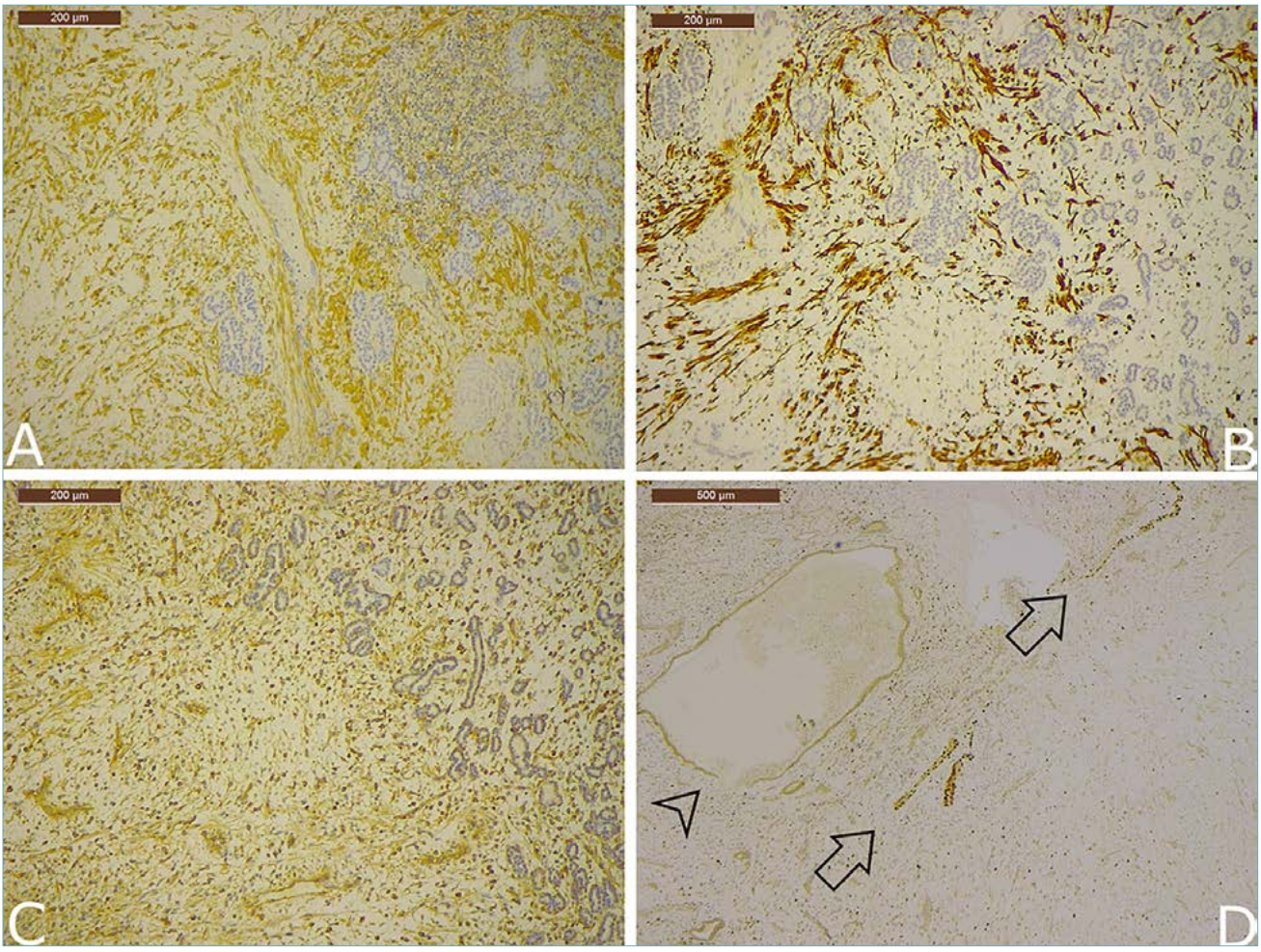

Figure 2. (A) α-SMA; (B) desmin; (C) vimentin; (D) GATA-3. Arrows indicate tubular structures positive for GATA-3; arrowhead indicates a cystic structure negative for GATA-3.

Second case report

Our second case report is about a 61-year-old man whose clinical history began with hypertensive peaks. The physical examination revealed a palpable mass in the left flank and the computed tomography (CT) scan of the abdomen described some voluminous ipodense cystic lesions (the major one measuring 21x16x21 cm in dimensions) both in the cortex and pelvis of the left kidney. Other clinical-radiological findings were hydronephrosis, calculi, and some minute swollen para-aortic lymph nodes. The patient underwent open radical left nephrectomy, sub hilar para-aortic lymphadenectomy, and splenectomy without complications. Nephrectomy specimen weighed 1750 g including the perirenal fat. On gross examination we found a well-circumscribed, encapsulated, exophytic, and globoid tumor measuring 17x11 cm. The mass was indented and intruded into the renal sinus from which it seemed to develop. It completely occupied the entire kidney and abutted on the remaining parenchyma. The tumor was homogeneous, gray white, semi translucent, and gelatinous. Mucinous glistening liquid dripped from the solid cut surface (Fig. 3A).

Histologic examination revealed a heterogeneous cellularity with focal atypia and an abundant myxoid matrix with occasional fine strands of fibrous tissue (Fig. 3B). The mesenchymal cells frequently showed binucleation (Fig. 3D), eosinophilic globules, and a patternless growth. We also found cells with multiple cytoplasmic mucin-containing vacuoles and nonscalloped nuclei (pseudolipoblasts) (Fig. 3D) and other sparse and slender histiocytic-like cells.

Neoangiogenic vessels focally had a curvilinear arrangement (Fig. 3C). Atypical mitoses were not detectable (< 3 mitoses/50 HPFs) and necrosis was absent. The diagnostic workflow with immunohistochemistry in-

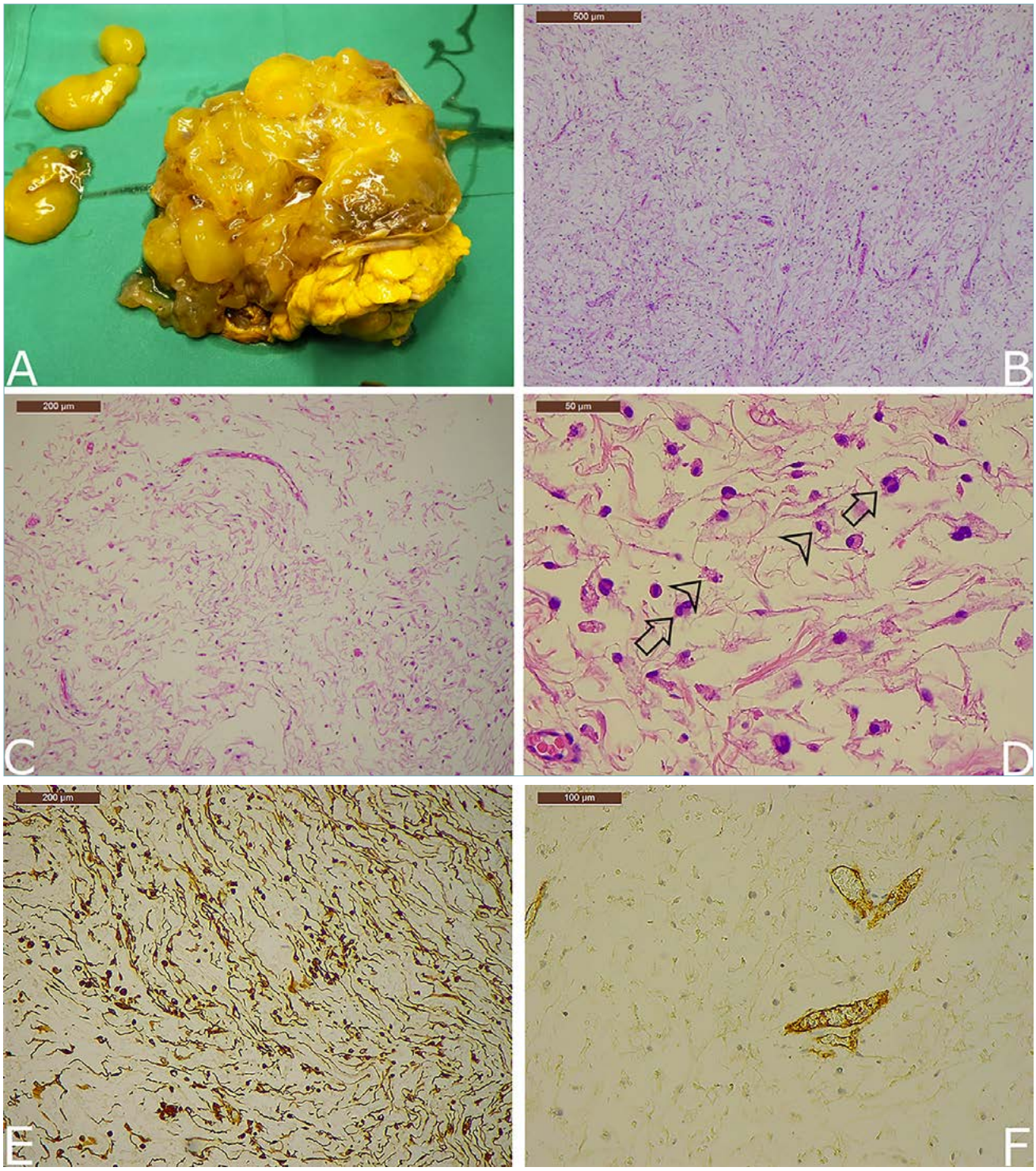

Figure 3. (A) Gross appearance; (B) Microscopic appearance; (C) Arcuate vessels; (D) Arrows indicate binucleated tumor cells; arrow heads indicate pseudolipoblasts; (E) Vimentin; (F) MUC-4.

cluded vimentin, bcl-2, CD34, CD99, alpha-methylacyl coenzyme A racemase (AMACR) (as they were reported to be strongly positive in a previous case of renal MFS by Val-Bernal et al.) [4], desmin, heavy caldesmon, α-SMA (in order to rule out smooth muscle differentiation and LGMFS), CD68 (in order to show fibroblastic

differentiation), S100 (in order to rule out a nerve sheet tumor and liposarcoma), MDM2 (in order to rule out liposarcoma), and MUC-4 (in order to rule out LGFMS). Additionally expression of bcl-2, CD99, and AMACR served as markers of tumor aggressiveness [4].

The tumor cells showed diffuse immunoreactivity for vimentin (Fig. 3E) and CD68, and weak positivity for MDM2 and bcl-2. Negative reactivity was observed for desmin, heavy caldesmon, α-SMA, S100, MUC-4 (Fig. 3F), CD34, CD99, and AMACR.

The proliferation index, valuated with Ki-67 immunohistochemistry, was mostly low (less than 1-2%), with some isolated areas 20%. Fluorescence *in situ* hybridization (FISH) for *MDM2*, using a Poseidon Repeat-Free MDM2 (12q15)/SE12 control probe (Kreatech Diagnostics, NL), did not show amplification.

Discussion

Our first case report was of difficult interpretation. We considered three main differential diagnoses: myxoma, myxoid leiomyoma, and MEST. We have no doubt it was not a highly aggressive neoplasia as it had no necrosis, few mitoses, and low cellularity. The features favoring a diagnosis of myxoma were the abundant myxoid matrix on histology and the absence of immunoreactivity for WT-1 (which is considered a well-expressed marker in retroperitoneal leiomyoma [7] and MEST [6]). However, the presence of a vorticoid whitish nodular area on gross examination and the immunohistochemical findings of our case (positivity for vimentin, desmin, and α-SMA) could be consistent with a diagnosis of myxoma resulting from a leiomyoma with myxoid degenerative changes. In fact, no previous case of renal myxoma reported immunohistochemical positivity of neoplastic cells for desmin, although its expression was described for acral myxoma along with α-SMA. Occasional staining for α-SMA in renal myxoma was reported only by 3 authors [3,15,19]. To the best of our knowledge, 22 cases of renal myxoma have been reported to date [1-3,8-25] (Tab. I). Data were available for 19 cases [1-3,8,10-12,15-25], with a median age at diagnosis of 50 years and a female/male ratio of 10/9. On the contrary, immunohistochemistry can reliably demonstrate the smooth muscle lineage of leiomyomas as their cells are positive for α-SMA, desmin, heavy caldesmon, and calponin [26-28]. Moreover, diffuse expression of ER and PR in leiomyomas was reported in 87% and 67% of cases, respectively, in the series by Gupta et al. [26] and Patil et al. [27]. Similarly, WT-1 expression was seen in 73% of leiomyomas by Gupta et al.[26].

Nevertheless, myxoma and myxoid leiomyoma are benign entities that do not contemplate the presence of slight cytologic atypia and infiltrative margins as in our case. In addition, we observed some epithelial tubular structures of unknown origin among the mesenchymal neoplastic component. These last three characteristics reminded us of MESTs. Most of them are benign [5], but 14 reported cases have been associated with aggressive behavior [29]. They typically occur in perimenopausal women, often with a history of hormone therapy, with a mean patient age of 52 years[5] . These tumors are solitary and involved the medulla bulging into the renal pelvis or the cortex especially in the lower renal pole [5]. They are typically unencapsulated, but well circumscribed neoplasms [5]. In a series of 53 MESTs by Caliò et al. [6], 75% did not have a pseudocapsule, but instead a pushing border where spindle cells infiltrated the renal parenchyma as we reported for our case. MESTs display variable proportions of solid and cystic components, although the latter often predominates [5]. The solid areas are usually firm, white, and vorticoid [5]. Histologically, they are complex neoplasms, composed of a variety of epithelial elements embedded in a stroma of variable composition[6]. Cytological atypia is usually minimal in both components and mitoses are rare, as are necrosis and hemorrhage [5,6]. The stroma ranges from hypocellular (with a collagenous predominance or a rarer myxoid change) to markedly cellular in particular around cystic components [5,6]. The epithelial component consists of medium-sized round cysts (lined by flat to cuboidal, hobnail, ciliated, mucinous, columnar, or rarely squamous cells), spatulate papillae reminiscent of phyllodes tumor, nephrogenic adenoma-like glands, and complex papillae [5,6]. Besides showing positivity for markers of smooth muscle differentiation, stromal cells are usually positive for PR and ER. In the series by Caliò et al. [6], 95% and 88% of MESTs stained for PR and ER, respectively. 26% of 34 MESTs showed positivity for WT-1 in the stromal component [6]. Concluding, MEST could be a good diagnosis for our case. However, the main hitch was the absence of spindle cells immunopositivity for WT-1. Thus, after these considerations, we made a diagnosis of mesenchymal neoplasia with low grade of malignancy. A definitive diagnosis either of myxoma or myxoid leiomyoma or MEST was not possible.

Our case suggests the importance of macroscopic and microscopic description in the differential diagnosis between benign mesenchymal tumors with myxoid features along with the immunohistochemical support (α-SMA, desmin, and WT-1 can be useful in routine diagnostic activity).

Our second case report did not show necrosis, high mitotic activity, or atypical mitoses. However, the pres-

Table I. Clinicopathological data of 22 cases of renal myxoma.

Authors	Age/Sex	Site/ Location	Symptoms	Tumor size (cm)	Treatment	Year of publication
Appel et al.	NA/NA	Right/ parapelvic	Hematuria for 2 months	8 cm	Enucleation of mass	1968
Shenansky et al.	62/male	Right/ lower pole	Hematuria for 6 months	4 cm	Nephrectomy	1973
Melamed et al.	52/female	Left/ lower pole	Renal colic	7 cm	Nephrectomy	1994
Melamed et al.	68/female	Right/ upper pole	Asymptomatic	10 cm	Nephrectomy	1994
Kundu et al.	36/male	Left	Hypochondrium mass for 2 months	28 cm	Nephrectomy	1995
Nishimoto et al.	NA/NA	NA	NA	NA	NA	1996
Koike et al.	NA/NA	NA	NA	NA	NA	2004
Val-Bernal et al.	37/male	Right	Asymptomatic	6 cm	Nephrectomy	2005
Owari et al.	62/male	Right/ middle portion	Asymptomatic	8 cm	Nephrectomy	2006
Bolat et al.	27/female	Left/ lower pole	Asymptomatic	15 cm	Nephrectomy	2007
Nishimoto et al.	36/male	Left/ lower pole	Asymptomatic	9 cm	Nephrectomy	2007
Hakverdi et al.	59/male	Right/ upper pole	Lower urinary tract infection	6 cm	Nephrectomy	2010
Chan et al.	47/female	Right/ lower pole	Abdominal pain for 4 months	12 cm	Nephrectomy	2011
Yildirim et al.	82/male	Left/ renal sinus	Dysuria, urinary obstruction, and flank pain	9 cm	Nephrectomy	2012
Shah et al.	43/female	Left/ mid-upper portion	Asymptomatic	4.9 cm	Nephrectomy	2013
Gomez-Gonzalez et al.	29/female	Left/ interpolar region	Asymptomatic	4.5 cm	Enucleation of mass	2014
Souza et al.	73/female	Left/ middle third	Recurrent cystitis	11.9 cm	Nephrectomy	2015
Suthar et al.	48/female	Right/ mid-lower pole	Abdominal pain for 15 days	7.4 cm	Nephrectomy	2015
Tenkorang et al.	50/female	Right/ mid-portion	Right dull flank pain	4 cm	Nephrectomy	2017
Thakker et al.	55/female	Right/ upper pole	Abdominal pain	1.8 cm	Enucleation of mass	2017
Tutman et al.	17/male	Transplant kidney/ mid-lower pole	Incidental finding during follow-up for renal insufficiency	5.4 cm	Nephrectomy	2017
Salehipour et al.	56/male	Right/ lower pole	Right flank pain, hematuria	8.5 cm	Partial nephrectomy	2019

ence of binucleated neoplastic cells and pseudolipoblasts suggested a diagnosis of a low-grade sarcoma. We considered three different possible malignant mesenchymal tumors: MFS, LGFMS, and dedifferentiated liposarcoma. Beyond histological features, we excluded LGFMS due to absence of immunoreactivity for MUC-4 and dedifferentiated liposarcoma due to absence of *MDM2* amplification. Moreover, the Italian referent pathologist for soft tissues' tumors confirmed our case was a MFS grade 1 according to the French Federation of Cancer Centers Sarcoma group (FNCLCC) as it had focal atypia (score 1), less than 0-9 mitoses for 10 HPF (score 1), and no necrosis (score 0). The term MFS was originally proposed by Angervall et al. [30]. This sarcoma mainly affects elderly patients (in the sixth to eighth decades of life) [31]. Origin in the limbs is more frequent than that in the retroperitoneum and abdominal cavity [31] and local recurrences are unrelated to histological tumor grade [30]. Intermediate (grade 2) and high grade (grade 3) neoplasms are capable of metastasiz-

Table II. Clinicopathological data of 3 cases of renal myxofibrosarcoma.

Authors	Age/sex	Site/Location	Tumor size (cm)	Treatment	Histological grade (FNCLCC system)	Year of publication
Val Bernal et al.	70/female	Right/upper two thirds	12 cm	Nephrectomy	2	2015
Resorlu et al.	62/male	Left/upper two thirds	20 cm	Nephrectomy	2	2017
Yakirevich et al.	62/male	Right	NA	Nephrectomy	2	2019

Table III. Clinicopathological data of 7 cases of renal LGFMS.

Authors	Age/Sex	Site/ Location	Tumor size (cm)	Treatment	MUC-4	FISH	Year of publication
Silverman et al.	70/male	Bilateral	NA	Right partial nephrectomy	NA	NA	2000
del Valle González et al.	28/male	Left	25 cm	Nephrectomy	NA	NA	2009
Arancio et al.	83/female	Right	18.5 cm	Nephrectomy	NA	NA	2010
Alevizopoulos et al.	48/male	Left/pelvis	4.6 cm	Nephrectomy	NA	NA	2012
Rubistein et al.	6/male	Right	16.4 cm	Nephrectomy	Yes	*EWSR1-CREB3L1*	2014
Bhattar et al.	35/male	Left/lower pole	7 cm	Nephrectomy	No	NA	2018
Mok et al.	10/female	Left/lower pole	10 cm	Open excision biopsies	Yes	*EWSR1-CREB3L1*	2018

ing [30]. MFS can arise de novo or be induced by radiation [32,33]. If MFS is radiation-induced, it usually occurs many years after exposure and is associated with poor prognosis [32,33]. Tearada et al. described a case of high-grade MFS in the spermatic cord after radiotherapy for prostate cancer 7 years before [32]. Ruo et al. reported a radiation-induced MFS in the left temporal-parietal scalp developed at the site of a previous basal ganglia germinoma treated with radiotherapy 14 years before [33]. Although rare, it should always be kept in mind that MFS may occur after a long period following radiotherapy [32,33]. Thus, anamnesis and clinical data are always important. Histologically, MFS comprises a spectrum of malignant fibroblastic neoplasms with variable cellularity, pleomorphism, and proliferative activity [31]. However, all cases share distinct morphological features such as multinodular growth, myxoid stroma, and a curvilinear vascular pattern [31]. Pseudolipoblasts and multinucleated giant cells are noted in low-grade and high-grade MFS, respectively [31]. Pseudolipoblasts are neoplastic cells resembling lipoblasts as a result of cytoplasmic vacuolization due to dilatation of endoplasmic reticulum [30]. The vacuoles contain acid mucin rather than neutral fat as in lipoblasts [30]. To the best of our knowledge only three cases of renal MFS have already been reported [4,34,35] (Tab. II). The best described case, with histological and immunohistochemical details, is that by Val-Bernal et al. [4]. Their tumor appeared as a 12 cm mass in the upper two-thirds of the right kidney of a 70-year-old woman. Unlike our case, that renal MFS was considered grade 2 as it showed transition from hypocellular myxoid areas to solid fasciculate components, focal necrosis, and a mitotic count of 5 mitoses/10 HPFs. They observed the presence of curvilinear capillaries, pseudolipoblasts, and multinucleated giant cells. Additionally the tumor cells of their MFS were positive for vimentin, bcl-2, CD34, CD99, and AMACR. FISH for *MDM2* and *DDTI3* showed no amplification and a normal pattern, respectively. Our case and the one by Val-Bernal et al. [4] confirmed MFSs are heterogenous neoplasms with different grades of malignancies due to the presence or absence of specific histological features. Molecular analysis seems to be more relevant than immunohistochemistry in the formulation of this diagnosis. The most difficult distinction to make is that between MFS and LGFMS [30]. This latter is also known as Evans tumor and it occurs typically in young adults with the same frequency in men and women [36]. It arises in the skeletal muscle of proximal extremities or trunk, although sometimes it can be centered in subcutaneous tissues [30]. Classical LGFMS shows an admixture of heavily collagenized, hypocellular zones, and more cellular myxoid nodules [36]. Approximately 30% of cases show giant collagen rosettes composed of a central core of eosinophilic collagen surrounded by rounded to ovoid cells with no nuclear atypia [36]. Neoplastic cells are very bland with small hyperchromatic nuclei and one to several nucleoli [30]. The tumor vasculature consists of branching capillary-sized vessels[30]. Mitotic figures are scarce [36]. MUC-4, a transmembrane glycoprotein that plays a role in cell growth signaling pathways, is consistently expressed in LGFMS [30]. The majority of these tumors are also positive for EMA, CD99, and bcl-2 [30]. The cytogenetic hallmark, identified in 76-96% of

cases of LGFMS, is t(7;16)(q33;p11) that results in a *FUS-CREB3L2* fusion [36]. A minority of cases bear the t(11;16)(p11) *FUS-CREB3L1* or the t(11;22)(p11;q12) *EWSR1-CREB3L1* gene fusions [37]. We found 7 reported cases of primary renal LGFMS in literature [37-43] (Tab. III). Unfortunately, only 3 cases were studied for MUC-4 expression [37,42,43]: 2 LGFMS showed strong positivity [37,42] and one did not [43]. Only 2 cases were analyzed for translocations with FISH and both LGFMS had the *EWSRI-CREB3L1* gene fusion [37,42].

In our diagnostic process, we could have considered the entity of myxoid liposarcoma (MLS). Its peak incidence is in the fourth and fifth decades of life and it occurs more commonly in the deep soft tissues of the extremities [44]. It appears as a well-circumscribed mass with a glistening and gelatinous cut surface in low-grade tumors and a fleshy tan appearance in high-grade neoplasms [44]. Microscopically, they are characterized by uniform round to oval-shaped non-lipogenic cells and small signet ring lipoblasts in a prominent myxoid stroma with a delicate and arborizing vasculature[4 4]. In MLS with histological progression, there is a greater number of round cells, positive for S-100 and vimentin, organized in solid sheets with no intervening myxoid stroma [44]. These tumors usually show either *FUS-DDIT3* or *EWSR1-DDIT3* rearrangement [44]. However, our case did not show any lipoblasts or solid areas. Findeis et al. [45] described a case of a renal myxoid neoplasm of difficult interpretation as both myxoma and MLS were considered.

Our case suggests the importance of immunohistochemistry and molecular analysis in the differential diagnosis between renal sarcomas with myxoid features. A diagnostic workflow with immunohistochemistry should include at least vimentin, bcl-2, CD34, CD99, AMACR, MUC-4, and MDM2. Besides, the presence of a specific genomic abnormality (such as a *FUS-CREB3L2* fusion for LGMFS or *MDM2* amplification for dedifferentiated liposarcoma) can be useful in the diagnostic process.

Although MFS belongs to the subgroup of non-translocation-related sarcomas with higher chromosomal instability, it deserves genomic profiling as it can reveal clinically relevant genomic alterations for tailored treatments [35].

Yakirevich et al. conducted comprehensive genomic profiling (CGP) in 13 adult renal sarcomas including a case of MFS [35]. Their MFS had amplifications of two adjacent receptor tyrosine kinase genes (*KIT* and *PDGFR*) encoding KIT and PDGFR-α proteins that can be modulated by tyrosine kinase inhibitors such as pazopanib [35]. Olaratumab, a monoclonal antibody against PDGFR-α, can improve overall survival [35]. The MFS reported by Yakirevich et al. had also homozygous deletion of tumor suppressor genes *CDKN2A* (that encodes p16INKa and p14ARF) and *CDKN2B* (that encodes p15INK4b) [35]. p16INK4, p14ARF, and p15INK4b are negative regulator of the cell cycle checkpoints kinases CDK4 and CDK6[35]. When *CDKN2A/B* are lost, the activity of cyclin-dependent kinases 4 and 6 can be inactivated by Palbociclib [35].

Other recent studies, such as those by Ogura et al. [46] and Ma et al. [47], revealed other genomic alterations in MFSs (including mutations in *TP53*, *RB1*, *NF1*, *ATRX*, *NTRK1*, novel oncogenic *BRAF* fusion gene [46], and MET overexpression due to chromosome 7 polysomy) [47]. Their results provide a valuable basis for the development of precision medicine approaches in MFS [46,47].

Conclusions

Our two cases highlighted the difficulty in diagnosing primary myxoid lesions of the kidney.

Utilization of immunohistochemical stains and molecular testing is imperative in narrowing the differential diagnosis, especially between benign and malignant entities. However, ancillary methods are not always conclusive as they may be in contrast with the morphological appearance of the tumor as we saw in our first case.

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