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Solitary Fibrous Tumor of the Kidney: A Wide Spectrum of an Uncommon Entity from Benignity to Malignant Metastatic Disease

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Solitary fibrous tumour (SFT) is a spindle cell neoplasm that rarely occurs in the kidney. It has been reported mainly in the pleura, but it can also arise from almost every organ. Benign SFT of the kidney has been described in several cases, even with metastatic lesions, but only seven cases of malignant SFT including this case have been documented; and only three cases out of seven had distant metastasis. Clinical features are indistinguishable from other renal neoplasms. Diagnosis is based on inmunohistochemical analysis, showing positive staining for CD34, as a key for diagnosis. We report two different cases, with very different behaviour; showing the wide spectrum of malignancy of this entity; with a comprehensive review of the literature.

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1. INTRODUCTION

Solitary fibrous tumours (SFTs) are an unusual mesenchymal spindle cell neoplasm most commonly described in the pleura [1] and first reported by Kemplerer and Rabin in 1931 [2]. It has also been reported to occur at other extrapleural sites, even in soft tissues or parenchymatous organs such as liver, salivary gland, thyroid, breast, adrenal gland and urogenital tract (spermatic cord, seminal vesicles, urinary bladder, prostate and kidney) [3]. SFT arising at the kidney was first described by Gelb et al. in 1996 [4], and to our knowledge only 67 cases have been reported in the literature [5]. Despite most of the cases are benign, it is important to consider its presence in the kidney because it is usually diagnosed as a renal cell carcinoma and a differential diagnosis with a wide variety of monomorphous spindle cell tumours should be done. Only six cases of malignant SFT have been reported. The site of origin has been considered to be the renal capsule in a 15%, 6% in peripelvis connective tissue [6], and 3 % in renal pelvis; while 76% had an unknown site of origin [7]. It is estimated that 10% to 15% of intrathoracic SFTs will recur and/or metastasize [8]. This aggressive behaviour in 15% of the patients cannot be assessed by the histopathological findings; some malignant cases have behaved in a benign fashion after complete resection and viceversa; reason why an indefinite period of observation is mandatory [9-13]. Morphologically, SFT is generally characterized by spindle cell proliferation showing a pattern-less architecture, and the key to diagnosis is inmunohistochemical study using a monoclonal antibody directed against the human hematopoietic progenitor cell antigen (CD34) stain [14,15].

In this paper we present two cases with a very different behaviour. The first case represents a malignant and metastatic SFT, which is the third reported case of metastasis from a renal SFT and the first one with metastasectomy specimens showing malignant findings. The second one represents, initially, as a benign form of SFT.

2. CASE 1

A 57-year-old man who entered the emergency room by history of constitutional syndrome with a two month history of asthenia, anorexia and loss off weight of 20 kg in a month. He presented right hemiabdomen pain of moderate intensity, partially controlled with anti-inflammatory drugs, and occasional requirement for opioids. He has not urological symptoms. Physical examination showed a soft abdomen, depressible, painful tenderness in right upper quadrant, where a mass of about 30-35 centimeter (cm), hard consistency, not attached to deep structures was palpable. Laboratory data shown microcytic anemia.

Computed tomography shown a voluminous lesion in right kidney lower pole, 15.5 x 14.2 cm, causing a deformity of the inferior vena cava and displacement of renal vessels, compatible with neoformative process. Lymph nodes were within normal range. Magnetic resonance imaging (MRI) revealed no renal vein or inferior vena cava thrombosis (Fig. 1)

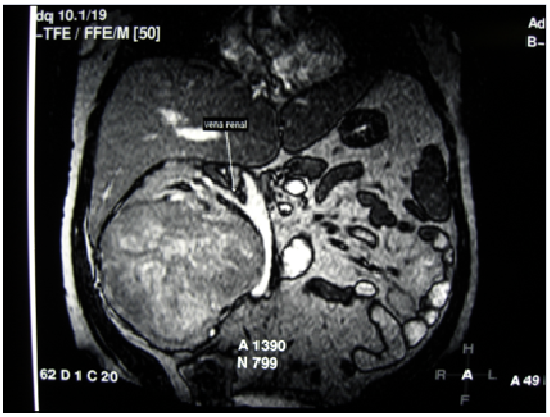

Fig. 1. MRI. Solid right renal mass

Given the large size of the tumour, arteriography and embolization of the main artery was done 24 hours before surgery. The patient underwent radical right nephrectomy.

The specimen weighted 2950 gr. and measured 24 x 17 x 11 cm. On cut section two thirds of the kidney were replaced by well defined large mass of a whitish tissue with multiple haemorrhagic areas. The residual parenchyma was displaced to the apical end, along with the adrenal gland and showed a good cortico-medullary differentiation with a congestive marrow. Vessels showed thrombosis. The tumor had a pseudocapsula, moving the fatty tissue in the renal sinus.

Microscopically examination revealed a high grade sarcoma, with spindle cells partially necrotic by previous embolization, whose morphological and immunohistochemical features were suggestive of renal malignant solitary fibrous tumor (Vimentin , CD34 and Bcl-2 positive; CKs, S100, HNB45, melan A, actin, ML, desmin, epithelial membrane antigen (EMA), CD99, CD117, negative) (Figs. 2 and 3).

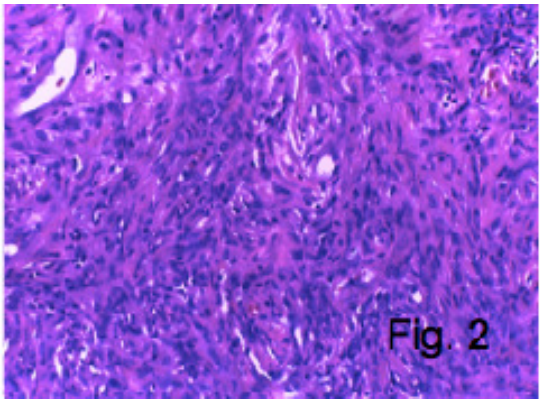

Fig. 2. Benign SFT. Hematoxylin –eosin, magnification x 40. Mesenchimal spindle cells, without atypia or mitosis

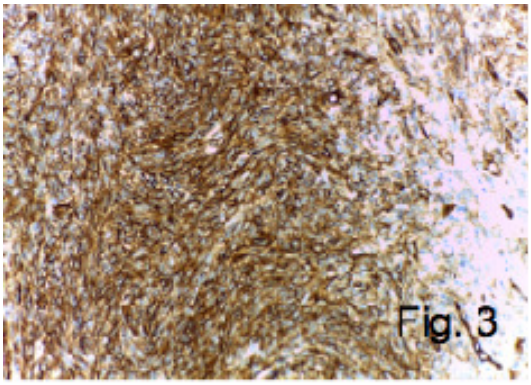

Fig. 3. Benign SFT. Immunohistochemistry, diffuse positive staining for CD 34. (40x)

Two years after nephrectomy, follow-up CT detected three right lung nodules and hemothorax. After biopsy and pathological confirmation of SFT metastasis, upper right lobectomy and atypical segmentectomy was performed. Resected tissue from the lungs was positively stained for CD 34 vimentin, ki-67 proliferation index of 12% and overexpression of p53 and negative for CK7, CK20, RCC and CD 10. Two years after lung surgery, patient underwent urgent surgery for bowel obstruction related to a new metastatic lesion placed at small intestine. Inmunohistochemically, CD 34 labelling was observed, also positive bcl2, vimentin and desmin. However, no area of SFT were described, even though is compatible with the diagnosis of dedifferentiated SFT. Synchronously, two new lung nodules were found at CT and resected. Neither chemotherapy nor radiation therapy was given. Patient is now currently free of disease 50 months after first surgery.

3. CASE 2

A seventy two year old woman complained of two-month history of abdominal pain. No lumbar tenderness, haematuria or other constitutional symptoms were found. Laboratory findings were unremarkable. Physical examination revealed a palpable left flank mass. Ultrasonography of the abdomen showed a 12.8 cm solid mass in the left kidney. CT confirmed a voluminous primary tumour in the left kidney, with ectasia of the upper caliceal group by extrinsic compression and dilated left renal and gonadal veins, probably by vascular hiperflow (Fig. 6). She underwent radical left nephrectomy. Pathologic findings in nephrectomy piece were a 13x7 x9 cm specimen; the cut section of the nephrectomy piece identifies a tumour mass of 12 x 7.5 cm that appeared encapsulated without affecting the renal capsule macroscopically as well as renal sinus structures, which were compressed by the tumour. It had a whitish surface with an increased elastic consistency and whitish-orange coloration. Microscopically, it consisted of mesenchymall cells with little atypia and low mitotic index (Fig. 4).

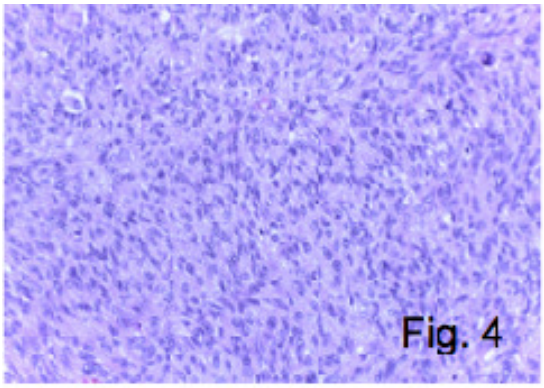

Fig. 4. Malignant SFT. Hematoxylin –eosin, magnification x 40. Increased cellularity with overlapping nuclei, cellular pleomorphism, and high mitotic index.

No necrosis or haemorrhage was observed. A characteristic hemangiopericytoid vascular pattern was identified. The tumour was well circumscribed and encapsulated. Inmunohistochemical techniques resulted in CD 34 and bcl-2 positive and HMB45, actin, S100, EMA and CD 99 negative (Fig. 5). Findings were consistent with SFT. The patient has no evidence of disease 7 months after surgery.

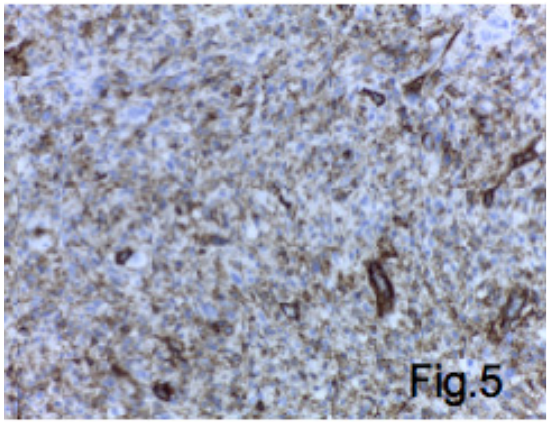

Fig. 5. Malignant SFT. Immunohistochemistry, positive staining for CD 34. (40x)

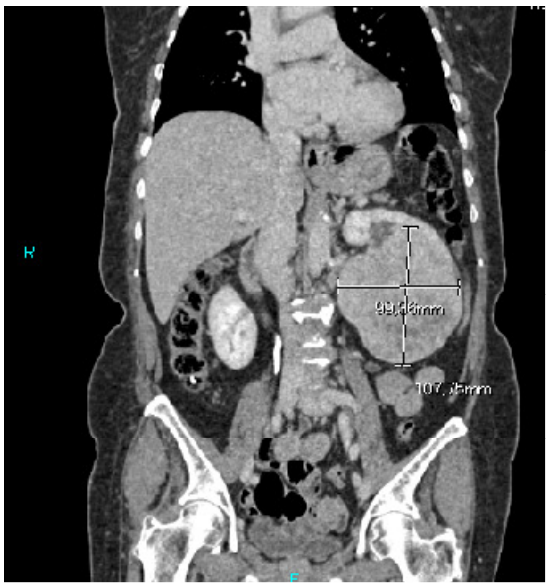

Fig. 6. CT showing ectasia of the upper caliceal group by extrinsic compression by the solid mass

4. DISCUSSION

SFT are mesenchymal spindle cell neoplasms originally described in the pleura; however, there have been reports of extrapleural origin, including urogenital tract. SFT clinically appears to be asymptomatic, and in most of the cases, does not metastasize. Although 10 -15% of extrathoracic SFT has been reported to show an aggressive behaviour. SFTs of the kidney are characterized by spindle cells proliferation showing a pattern-less architecture with patchy hypocellular and hypercellular areas. Cellularity is variable and is composed of a mixture of storiform, short fascicular arrangements of bland spindle cells and collagenous bands [16]. They are characterized by increased vascularity, with hemangyopericytoma-like patterns. The criteria for malignant SFTs was first proposed by England et al. [17] and included increased cellularity with crowded/ overlapping nuclei, cellular pleomorphism, and mitotic count of more than 4 per 10 high-power fields. Clinical outcome of this tumours are not necessarily poor in presence of a histologically malignant diagnosis and they can have a benign behaviour [18,19]. Contrariwise, benign histological findings can show an aggressive growth invading neighbouring tissues [20].

Electron microscopy shows ultraestructural features consistent with fibroblastic characteristics. Abundant collagen appears surrounding spindle cells proliferation. Electron microscopy seems to benefit diagnosis differentiating this entity from other neoplasm with nervous and muscular components.

Hypothesis for the development of malignant SFT has been proposed in two ways; " de novo " or from transformation or dedifferentiation within benign form. Origin of the tumours has not been elucidated although it seems to originate from primitive mesenchymal cells at the renal capsule [21].

Symptoms are inespecific and similar from those related by patients with renal cell carcinoma. It can be diagnose in asymptomatic patients as a radiological finding, or appear as a palpable mass. Haematuria, flank or abdominal pain may be present. Doege Potter syndrome associated with non-islet cell is traduced in paraneoplasic syndrome due to hypoglycaemia and it's correlated with bad prognosis forms [22]. Most of the tumours are diagnosed preoperatively as renal cell carcinoma.

Risk factors predicting a poor prognosis of a SFT have been described by Mosquera et al. [23]: incomplete resection, size larger than 10 cm,

presence of malignant cellularity or extrathoracic site.

Differential diagnosis is challenging for the pathologist and is based on inmunohistochemically findings. This entity should be differentiating from other primary monomorphus malignant and benign spindle cell lesions.

Inmunohistochemical study is the key to diagnosis. CD 34 inmunorreactivity is characteristic of SFT, although is not specific, is indispensable in the diagnosis, and its presence with CD 99 is high suspicios for the differential diagnosis with other mesenchymal tumours [24-26]. CD 34 is a glycoprotein that is present in haematopoietic progenitor cells and endothelial cells of small blood vessels [27], as consequence, it is also positive in hemangioperycitoma as well as other malignancies arising from vessels and fibroblasts as elastofibroma [28]. CD 34 is present in acute nonlymphocitic leukaemia, dermatofibrosarcoma protuberans, neurofibroma, epithelioid sarcoma, gastrointestinal stromal tumours and SFT [29]. Positive inmunorreactivity for CD34 and CD 99 may not be present in case of dedifferentiation or atypia. Bcl-2 protein expression is usually preserved in all SFT from the benign forms to the aggressive ones [30]. CD 99 and Bcl-2 are expressed in 70% of SFTs, being only from 20 to 35% positive for epithelial membrane antigen and smooth muscle actin [31]. S-100 protein is uncommon but may be present, specially associated to sarcomatoid components in aggressive tumours. α-SMA, CD31, c-kit and desmin are most times negatives, but its negativity does not exclude the diagnosis of SFT. P53 is a bad prognosis marker and it can be present in cases with poor outcome, high recurrence rate, nuclear atypia and malignant histological criteria. Ki67 expression is found from 1% to 44% of the malignant SFT cases.

Differential diagnosis includes both benign and malignant entities. Cellular forms of SFT are indistinguishable from hemangiopericytoma, containing thin-walled vessels with little fibrosis [32] and haemorrhagic areas. Hemangiopericytoma positivity of CD 34 is less marked than in SFT. Hemangiopericytoma positivity of CD 34 is less marked than in SFT. Authors have suggested that these two entities represent a spectrum of the same tumour, especially in lipomatous hemangiopericytoma and SFT with fat in its composition. Presence of collagen in areas alternating hypocellular and hypercellular patterns are more likely to be related to SFT while the presence of round or oval nuclei and a diffuse and widespread staghorn vascular pattern come up more towards hemangiopericytoma.

Other benign tumours should be discarded. Benign fibromatous tumours or fibromas has not the hipercelullarity of SFT and is negative for CD 34 [33,34]. Benign fibrous histiocitytoma is rare in the kidney and the absence of CD34 in addition to a characteristic growth pattern besides the storiform growth makes the diagnosis [35]. Inflammatory myofibroblastic tumour is an entity that can behave in a benign pattern as well as in an aggressive way with metastatic potential, so it should be regarded as low-grade sarcoma [36]. It is characterized by the presence of myofibroblastic cells in a myxoidhialinized stroma and associated with a polymorphous inflammation [37]. Angiomyolipoma of the kidney shows a spindle cell smooth muscle component may be misinterpreted as SFT [38]; however, its immunoreactivity to HMB45, a-smooth muscle actin and desmin, and CD34 negativity helps with diagnosis. Leyomioma of the kidney can be easily distinguished by histological analysis [39]. Benign peripheral nerve sheath tumours (schwannoma, neurofibroma and perineurioma) are rare but may occur in the kidney [40,41]. Perineurioma is a tumour that can show a storiform growth pattern and positivity for CD 34 in 50% of cases with EMA immunoreactivity that helps in the diagnosis.

S-100 protein positive tumours should be excluded; diagnosis of SFT should be kept in mind when a sarcomatous tumour is found in a metastatic lesion in a patient with history of SFT or at local remanent after SFT removal. Follicular and interdigitating dendritic cell tumours mark S-100 but not CD 34, showing at microscopy small mature lymphocytes and plasma cells and CD 68 positive in inmunohistochemically analysis.

Synovial sarcoma is a rare tumour that grows in the kidney is also composed by spindle cells and high cellularity involving renal collecting ducts and disposed occasionally as cytokeratin cysts and a hemangiopericytoma –like growth pattern [42,43]. Synovial sarcomas could express bcl-2 and CD99, and sometimes cytoqueratin and EMA but are negative for desmin, actin, S-100 protein and the most differential, CD34 negative. This entity has also cytogenetic features than

differentiates from SFT, such as expression of SYT-SSX gene fusion and t (t; 18) translocation.

Primary fibrosarcoma of the kidney express vimentin alone and herringbone growth pattern with a high mitotic rate in absence of hypocellular component and negative for CD 34 and Bcl-2 [44,45].

Malignant peripheral nerve sheat tumours are rare, shows bcl-2 in more than 30 % of the cases [46] and S- 100 in approximately a half, being expression of CD 34 very uncommon [47].

Sarcomatoid renal cell carcinoma may simulate a SFT because of a hemangiopericytoma-like appearance. The spindle cells of sarcomatoid components reacted with antikeratin antibody and showed ultrastructural features of epithelial origin that may be also absent [48]. Leiomiosarcoma is recognized by the presence of smooth muscle and muscle markers.

Mesoblasticnephroma is extremely rare in adults and contains a gene fusion ETV6-NTRK3 that differentiates it from other spindle cell neoplasms [49-51].

Treatment has not been established but it seems that a complete surgical resection of the primary tumour and metastatic lesions should be recommended with longer follow-up. Other therapies with antiangiogenics drugs based on its possible origin of perycites [52] and the rich vascularity has been proposed. Park et al. [53] reported results from 14 patients with advance, recurrent or metastatic hemangioperycitoma and malignant solitary fibrous tumour who received a combination of temozolamide and bevazicumab. eighty-six percent of the patients had previous treatment and antiangiogenic therapy was starting because of symptomatic disease, neoadjuvant treatment to downstage tumour and enable surgical resection or progression after prior therapy. Response criteria was assessed according to Choi criteria [54], measuring response to treatment according to decrease in tumour volume. The overall response rate was 79%, with a 6-months progression-free rate of 78, 9% and an estimated median progression-free survival of 9, 7 months with a median follow-up of 34 months. The most frequent toxic effect was myelosupression. Another case of metastatic malignant SFT received interferon after resection of the kidney and liver metastasis achieving stable disease for 20 months. Imatinib mesilate has been reported to have benefitial effect in pleural SFT expressing PDGFR-β [55,56].

This is the seventh case described of a malignant renal SFT and the third one with documented metastasis, although some benign forms are also capable to metastasize. No previous reports of sequential surgeries of multiple metachronous metastasis have been found. It is remarkable the needed of a long follow-up in this patients, with development of metastasis in our patient two and four years after removal of the primary lesion. With these two cases, only 69 solitary fibrous tumours from the kidney have been reported.

5. CONCLUSION

SFT of the kidney is an uncommon neoplasm that can behave like a benign process or like a malignant tumor with high metastatic potential. Immunohistochemistry is the key for diagnosis and surgery of the primary lesion and metastasis is the only curative treatment. Long term follow up is necessary to diagnose local recurrences or distant metastasis.

CONSENT

Patients authorize the use of their clinical data by signing the informed consent form.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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