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Original contribution

Perinephric myxoid pseudotumor of fat: a distinctive pseudoneoplasm most often associated with non-neoplastic renal disease☆

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Summary In 2009, Tanas et al reported unusual changes in the perinephric fat, mimicking well-differentiated liposarcoma. We report 11 perinephric masses showing similar changes but chiefly arising in patients with non-neoplastic renal disease. Tissue from 11 perinephric masses was retrieved, and immunohistochemistry for IgG/IgG4 and fluorescence in situ hybridization (FISH) for *MDM2* amplification was performed. Clinical information was obtained. Cases occurred in 10 males and 1 female (43-84 years of age; median, 63.5 years). Ten patients presented with perinephric masses (size range, 2-28 cm), and one was an incidental finding. Four patients had bilateral or multiple masses. Underlying renal disease included diabetes mellitus (n = 3), end-stage kidney (n = 2), diabetes and end-stage kidney disease (n = 1), chronic pyelonephritis (n = 1), and non-invasive high-grade papillary urothelial carcinoma of the renal pelvis (n = 1). Three patients were not known to have renal disease. Most tumors were submitted as "well-differentiated liposarcoma." The masses consisted of mature fat, myxoid stroma, moderately variable spindled to stellate cells and a mixed inflammatory cell infiltrate. Enlarged, hyperchromatic stromal cells were absent. IgG4-positive plasma cells and *MDM2* amplification were absent in all tested cases. Clinical follow-up (11 patients; range, 1-120 months; median, 24 months) showed absent or stable disease in 9 patients; 2 died of unrelated causes. This distinctive pseudoneoplasm usually occurs in association with non-neoplastic renal disease, although similar changes may be identified in the perinephric fat of patients with renal carcinoma. Morphologic evaluation and FISH for *MDM2* amplification should allow its distinction from liposarcoma and other mimics.

1. Introduction

The differential diagnosis of perinephric soft tissue masses, in particular those composed at least in part of adipose tissue, centers on liposarcoma, with roughly 25% of well-differentiated and 75% of dedifferentiated liposarcomas occurring in the retroperitoneum, often adjacent to the kidney [1-3]. Although the diagnosis of most perinephric well-differentiated and dedifferentiated liposarcomas is usually possible on morphological grounds alone, demonstration of *MDM2* amplification and/or overexpression by fluorescence in situ hybridization (FISH) or immunohistochemistry, respectively, may be very valuable in morphologically challenging cases or in the setting of limited biopsies [4-8]. Subtypes of well-differentiated liposarcoma that may be particularly

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challenging include the myxoid variant [9], which may simulate a variety of other myxoid soft tissue tumors including true myxoid liposarcoma (characterized by *FUS/EWSR1-DDIT3* fusions) [10] and the inflammatory variant, which may simulate an inflammatory process or hematolymphoid neoplasm [11,12].

In 2009, Tanas and colleagues published their experience with 12 patients with renal cell carcinoma, in which careful examination of the perinephric fat disclosed changes simulating well-differentiated liposarcoma (in particular the presence of atypical stromal cells), and labeled this process "pseudosarcomatous fibroblastic/myofibroblastic proliferation in perinephric adipose tissue adjacent to renal cell carcinoma" [13]. Two of these 12 cases had originally been seen in consultation as possible well-differentiated liposarcomas with synchronous renal cell carcinomas, and the remaining cases were identified through sequential study of radical nephrectomy specimens performed for renal cancer. To the best of our knowledge, there are no similar subsequent reports of these types of changes in the perinephric fat.

Over the past several years, we have seen in consultation several unusual perinephric lesions, showing microscopic changes similar to those described by Tanas et al, but presenting as mass lesions in patients without renal cell carcinoma. We studied 11 examples of this rare pseudotumor, which we have termed "perinephric myxoid pseudotumor of fat" in order to better understand its clinicopathologic, immunohistochemical and molecular cytogenetic features, and to raise awareness of this potential liposarcoma mimic.

2. Materials and methods

Approval for this study was granted by the Mayo Clinic Institutional Review Board. Our institutional and consultation archives were searched for perinephric/retroperitoneal masses showing microscopic changes similar to those described by Tanas et al [13], initially yielding 14 cases with available slides and tissue blocks. Because of the retroperitoneal location of these lesions and the frequent presence of plasma cells (see below), immunohistochemistry for IgG (rabbit polyclonal, 1:15000; Dako Corp, Carpinteria, CA) and IgG4 (clone MRQ-44, prediluted; Ventana, Santa Clara, CA) was performed using standard laboratory protocols. Re-review of one case disclosed a fibrotic nodule with an increased number of IgG4-positive plasma cells (up to 30 in a high-powered field); this case was excluded. Fluorescence in situ hybridization for *MDM2* amplification was performed according to previously described protocols [14]. Clinical information including patient follow-up was obtained from medical charts and from

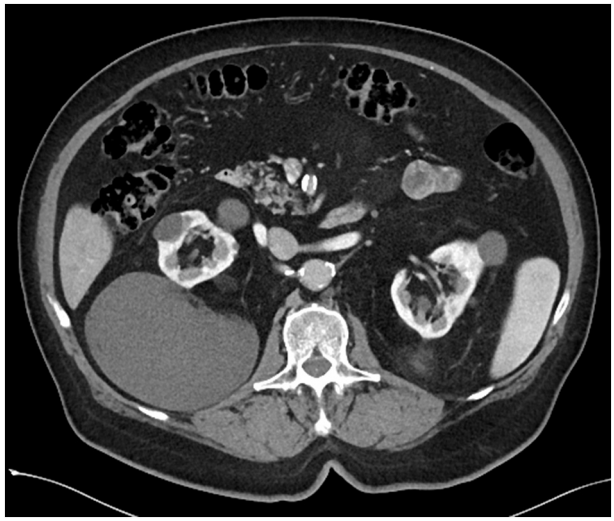

Fig. 1 Computerized tomography scan of a perinephric pseudotumor of fat, presenting as a 14 × 12 × 10 cm low-to-intermediate density mass in the right perirenal space of a 79-year-old male with diabetes mellitus–associated renal disease. Similar, smaller masses were also present in right and left perirenal spaces.

Table Summary of clinicopathologic, immunohistochemical and molecular cytogenetic features.

Case	Age (y)	Sex	Clinical presentation	Size of largest mass (cm)	Follow-up duration (mo)	Underlying renal disease	IgG4-positive plasma cells	*MDM2* FISH
1	63	Male	Multiple, bilateral masses	4.1	1 mo, DOC	DM	Not increased	Negative
2	69	Male	Multiple, bilateral masses	12	24 mo, ANED	DM	Not increased	Negative
3	79	Male	Multiple, bilateral masses	14	4 mo, ANED	DM and ESRD	Not increased	Negative
4	59	Male	Multiple, bilateral masses	16	48 mo, ANED	ESRD	Not increased	Negative
5	50	Male	Left perirenal mass	28	24 mo, ANED	NA	Not increased	Negative
6	45	Male	Left perirenal mass	6.5	26 mo, ANED	ESRD	Not increased	Negative
7	43	Female	Right perirenal mass	NA	120 mo, ANED	Chronic pyelonephritis	Not increased	Negative
8	58	Male	Left perirenal mass	2.9	48 mo, ANED	No disease	Not increased	Negative
9	84	Male	Left perirenal mass	15	3 mo, ANED	No disease	Not increased	Negative
10	78	Male	Right perirenal mass	4.5	DOC soon after procedure	DM	Not increased	Negative
11	75	Male	Right perirenal mass	NA	3 mo, ANED	Ipsilateral renal pelvis urothelial carcinoma	NA	Negative

Abbreviations: DOC, died of other causes; DM, diabetes mellitus; ANED, alive with stable disease or no evidence of disease; ESRD, end-stage renal disease; NA, not available; FISH, fluorescence in situ hybridization.

referring pathologists and clinicians. For two patients, review of the clinical history revealed prior diagnoses of IgG4-disease in other locations; these patients were excluded. The final study population thus consisted of 11 patients.

3. Results

The Table summarizes the clinicopathologic, immunohistochemical and molecular cytogenetic features of the 11 studied cases of perinephric myxoid pseudotumor of fat. The masses occurred in 10 men (91%) and 1 woman (9%) ranging from 43 to 84 years of age (median, 63.5 years of age). Diagnostic procedures included needle core biopsy (n = 4), perinephric mass resection (n = 1) and nephrectomy (n = 6). Ten patients presented with a perinephric mass (size range, 2-28 cm; median, 9.3 cm) (Fig. 1); in one patient the lesion was an incidental finding submitted as a "perihilar lymph node" during ureteronephrectomy for urothelial carcinoma. Four patients had bilateral or multiple masses. Underlying renal disease was known to be present in 8 patients, including diabetes with clinically impaired renal function (n = 3), end-stage kidney disease (n = 2), diabetes and end-stage kidney disease (n = 1), chronic pyelonephritis (n = 1), non-invasive high-grade papillary urothelial carcinoma of the renal pelvis (n = 1) and contralateral renal cell carcinoma 3 years previously (n = 1). Two patients did not have known renal disease. Cases were most often submitted in consultation to "rule out"

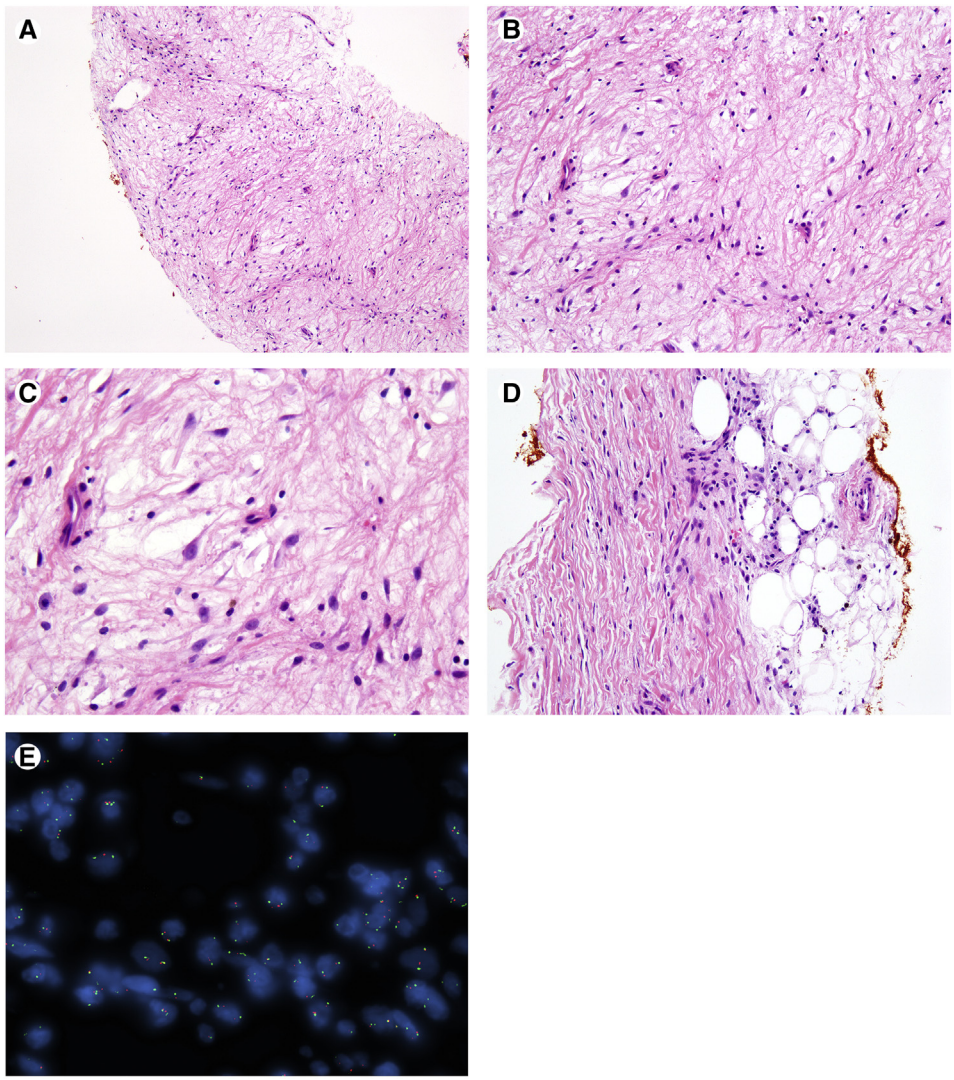

Fig. 2 A, CT-guided core needle biopsy of a perinephric pseudotumor of fat, presenting as multiple perinephric masses in a 63-year-old male with diabetes mellitus and clinical evidence of renal dysfunction (hematoxylin and eosin, ×40). B, The mass consisted of a hypocellular proliferation of somewhat variable fibroblastic cells in a fibromyxoid background, with an arborizing vasculature (hematoxylin and eosin, ×100). C, Higher power magnification of moderately variable spindled cells and interspersed lymphocytes (hematoxylin and eosin, ×200). D, Areas of mature fat containing patchy chronic inflammation, with lymphocytes and plasma cells, were present (hematoxylin and eosin, ×200). Immunohistochemistry for IgG4 was negative (not shown). E, Fluorescence in situ hybridization for *MDM2* amplification was negative (CEN 12(gr)/MDM2(or)[12q15], Image indicates nuclei with 2 red and 2 green signals).

well-differentiated liposarcoma (n = 7); other suggested diagnoses were angiomyolipoma, spindle cell lipoma, "myxoid neoplasm," "low grade sarcoma," IgG4-related disease, fibromatosis, aggressive angiomyxoma, myxoid liposarcoma and a reactive process. In some cases, the referring pathologists suggested more than one possible diagnosis.

Figs. 2–4 illustrate the morphological features of 3 representative cases. Histologically, perinephric myxoid pseudotumor of fat consisted of an admixture of mature fat, myxoid stroma, spindled to stellate stromal cells and a variably intense mixed inflammatory cell infiltrate, consisting predominantly of lymphocytes and small aggregates of plasma cells (Figs. 2A, 3A and 4A). A well-developed, thick-walled, arborizing vasculature was present in some cases, particularly in areas of extensive stromal myxoid change (Figs. 2B and 3B). Thickened fibrous septa, reminiscent of those seen in well-differentiated liposarcoma, were occasionally present, although these lacked the finely fibrillar collagen usually present in that entity (Figs. 2D and 4B). The nuclei of the stromal cells were generally small, with evenly dispersed chromatin and occasional small nucleoli (Figs. 2C, 3C and 4B). However, scattered stromal cells in essentially all cases showed moderate nuclear enlargement and irregularity, albeit to a lesser degree than is usually seen in well-differentiated liposarcoma. Lipoblasts were absent, although rare "pseudolipoblastic" stromal cells with cytoplasmic distension by myxoid secretions were present (Fig. 3D). Typical features of IgG4-disease, such as hyalinized nodules, perivascular inflammation, fat necrosis and large number of plasma cells were absent. Mitotic activity and necrosis were not seen.

Ten tested cases showed no more than very rare IgG4-positive plasma cells. Fluorescence in situ hybridization for *MDM2* amplification was negative in all 11 cases (Fig. 2E).

Clinical follow-up was available for 11 patients (range, 1-120 months; median, 24 months) and showed absent or stable disease in 9 patients. Two patients died of unrelated causes.

4. Discussion

The 11 unusual pseudotumors that comprise the present series show morphological features quite similar to those seen in the lesions previously reported by Tanas and colleagues as "pseudosarcomatous fibroblastic/myofibroblastic proliferation in perinephric adipose tissue adjacent to renal cell carcinoma" [13]. However, there are some important differences between the cases that comprise our series and those reported by Tanas et al. In particular, our cases presented clinically as mass lesions, concerning for some type of retroperitoneal neoplasm, and a history of renal disease was only obtained retrospectively. This is in contrast to the lesions reported by Tanas et al, which either represented incidental findings in patients

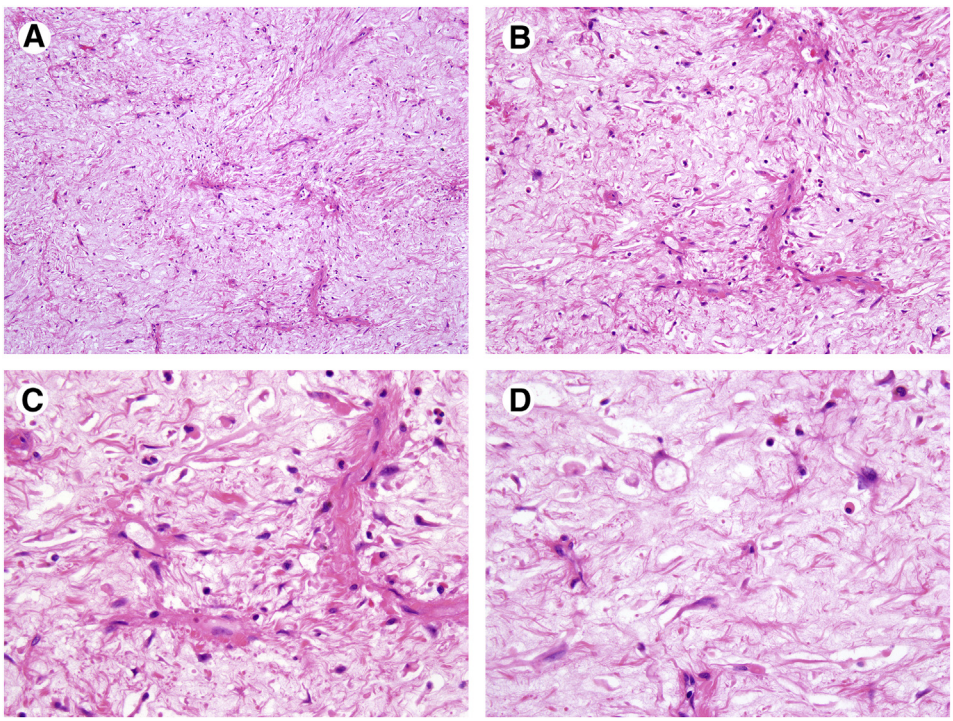

Fig. 3 A, Predominantly myxoid perinephric mass in a 45-year-old male with diabetes mellitus and end-stage kidney (hematoxylin and eosin, ×100). B and C, Although the arborizing, thick-walled vasculature raised the question of a myxoid sarcoma (hematoxylin and eosin, ×200), close inspection showed the spindled to stellate lesional cells to lack significant pleomorphism or hyperchromatism (hematoxylin and eosin, ×400). D, Rare "pseudolipoblasts" were present, as were scattered plasma cells; true lipoblasts were absent (hematoxylin and eosin, ×400). This case was negative for IgG4-positive plasma cells and for *MDM2* amplification (not shown).

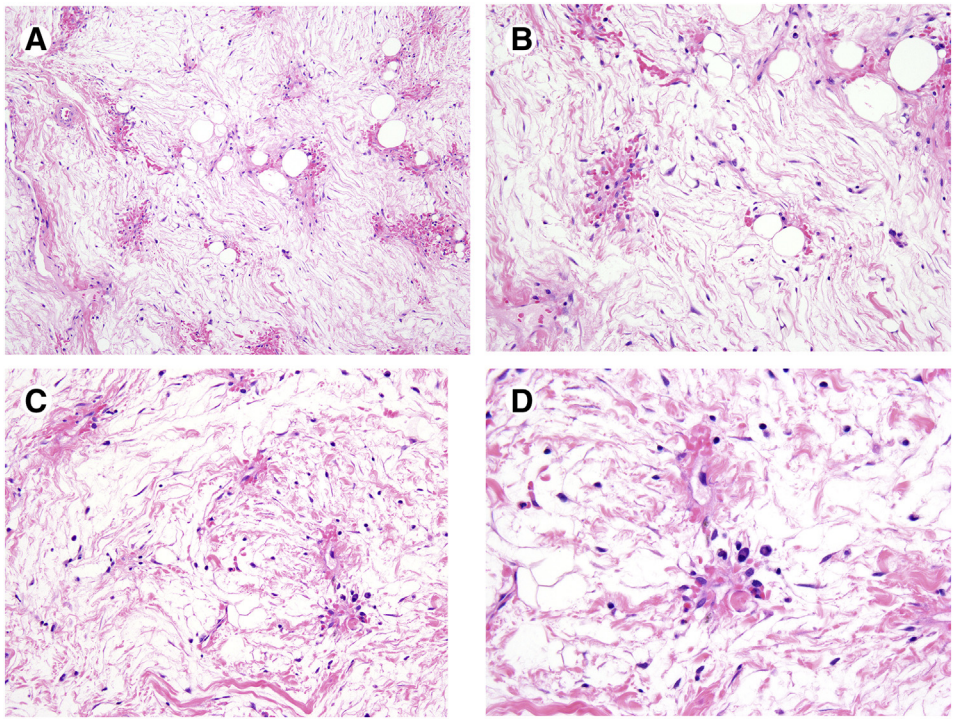

Fig. 4 A, A large, variably myxoid perinephric mass from a 59-year-old male with end-stage renal disease (hematoxylin and eosin, ×100). B, A needle biopsy from this mass had been interpreted at another institution as representing "well-differentiated liposarcoma," on the basis of the liposarcoma-like admixture of mature fat, collagenous septa and slightly enlarged stromal cells (hematoxylin and eosin, ×200). C and D, Other areas in this mass showed more extensive myxoid change, without fat (hematoxylin and eosin, ×40) and small clusters of plasma cells (hematoxylin and eosin, ×100). As in all other cases, IgG4 and *MDM2* were negative.

undergoing nephrectomy for renal cell carcinoma, or were only identified after retrospective close inspection of perinephric fat in radical nephrectomy specimens. Additionally, although the present cases were frequently associated with some type of renal disease, none of the patients in the present series had a history of renal cell carcinoma. The differences between these two studies are almost certainly related to the method of case ascertainment [13]. Since these lesions are not always renal cell carcinoma-associated, we have termed them "perinephric myxoid pseudotumor of fat" to better describe their unique clinicopathologic features. The pathogenesis of perinephric myxoid pseudotumor of fat is obscure, but is presumably related in some way to "irritation" of the kidney, either by mass effect in the case of carcinoma, or by the inflammation that often accompanies non-neoplastic renal disease. Although the study of Tanas and colleagues suggests that these changes may be relatively common in the perinephric fat of radical nephrectomy specimens, when searched for carefully, they must be in most instances be quite subtle and "non-mass-forming," as we do not recall seeing them in our routine surgical pathology practices.

The chief significance of perinephric myxoid pseudotumor of fat lies in its potential for confusion with well-differentiated liposarcoma of myxoid or inflammatory subtype, true myxoid liposarcoma, myxofibrosarcoma, fibromatosis, IgG4-related disease, angiomyolipoma, and rare retroperitoneal lipomas. Tanas et al stressed potentially mimicry of well-differentiated liposarcoma by these distinctive perinephric pseudotumors, emphasizing the frequent presence of enlarged, atypical stromal cells [13], and "liposarcoma" was the diagnosis most often suggested by referring pathologists for the lesions that comprise the present series. Although the morphological features of perinephric myxoid pseudotumor of fat are somewhat reminiscent of well-differentiated liposarcoma, in particular those showing myxoid change and chronic inflammation, there are some important differences. Specifically, the stromal cells of these pseudotumors lack the marked nuclear enlargement and hyperchromasia usually seen in well-differentiated liposarcoma. While these cells may show a moderate degree of nuclear enlargement, they are characterized by evenly dispersed chromatin with small, visible nucleoli. Myxoinflammatory fibroblastic pseudotumors also lack the finely fibrillar, "gelatin-like" collagen present in the fibrous septa of well-differentiated liposarcomas, and do not contain lipoblasts (an infrequent but potentially diagnostic feature of liposarcoma). However, given the relative frequency and clinical significance of well-differentiated liposarcoma in this anatomic location, we believe that definitive diagnosis of perinephric myxoid pseudotumor of fat requires demonstration of absent *MDM2* amplification by fluorescence in situ hybridization. We do not use immunohistochemistry for MDM2 in our clinical practice, as this test may be positive in non-neoplastic histiocytes, a potential pitfall particularly in the setting of fat necrosis [15].

Primary myxoid liposarcoma of the retroperitoneum is exceedingly rare [16], with the overwhelming majority of purported cases in this location either representing metastases from a primary tumor elsewhere (often the thigh) [10] or incorrectly diagnosed myxoid variants of well-differentiated and dedifferentiated liposarcoma [9,17]. Morphologically, true myxoid liposarcoma typically shows an exceptionally well-developed capillary vasculature, very uniform, bland cells and numerous lipoblasts, features lacking in myxoinflammatory fibroblastic pseudotumor. In particularly problematic cases, molecular genetic analysis for the myxoid liposarcoma-specific *FUS/EWSR1-DDIT3* fusions may be helpful in establishing the correct diagnosis [18]. Similarly, myxofibrosarcoma is exceedingly rare in the retroperitoneum, with the great majority of suspected cases representing instead variants of dedifferentiated liposarcoma with myxofibrosarcoma-like histology [9,19].

The clinical and morphological features of myxoinflammatory fibroblastic pseudotumor may also raise the question of an "early" or myxoid variant of IgG4-related fibrosclerosing disease. Indeed, we excluded 3 morphologically generally similar cases from this study because we could not be certain that they did not represent early manifestations of IgG4-disease (see Methods). IgG4-positive plasma cells were not a feature of the remaining lesions, and on balance we believe it unlikely that the pathogenesis of these unusual perinephric pseudotumors is related to IgG4-disease. In general, IgG4-related fibrosclerosing lesions show a greater amount of hyalinized collagen, perivascular inflammation, and a large number of IgG4-positive plasma cells [20]. Clinical and serological correlations are also helpful in patients with suspected IgG4-disease involving the retroperitoneum. Because of the potential morphological overlap between IgG4-disease and myxoinflammatory fibroblastic pseudotumor, we recommend immunohistochemistry for IgG4 on lesions of this type.

Myxoid areas may be seen in intra-abdominal fibromatoses, but typically co-exist with zones showing more typical histology, with long, sweeping fascicles of uniform, "active-appearing" myofibroblastic cells arrayed about a thin-walled, dilated vasculature. Immunohistochemical studies to demonstrate aberrant nuclear accumulation of β-catenin protein [21] or molecular genetic studies for *CTNNB1* mutations [22] may be of value in cases where the differential diagnosis includes fibromatosis.

Finally, the lipomatous variant of angiomyolipoma and true retroperitoneal lipomas should also be considered in the differential diagnosis of myxoinflammatory fibroblastic pseudotumor. Lipomatous angiomyolipomas may show stromal myxoid change, but also contain small aggregates of cytologically bland perivascular epithelioid cells, showing a characteristic "myomelanocytic" immunophenotype, with co-expression of myoid and melanocytic markers [23]. Lipomas in any location, including the retroperitoneum, may show myxoid change but lack the moderately atypical stromal cells seen in myxoinflammatory fibroblastic pseudotumor. Like myxoinflammatory fibroblastic pseudotumors, retroperitoneal lipomas lack *MDM2* amplification, a negative finding crucial in their distinction from well-differentiated liposarcoma [24].

In summary, we have described the clinicopathologic features of 11 cases of a rare, mass-forming, perinephric pseudotumor usually associated with non-neoplastic renal disease, which we propose terming "perinephric myxoid pseudotumor of fat." The etiology of this process is not clear but may reflect a localized connective tissue reaction to inflammation or neoplasia within the kidney. Similar, non-tumefactive changes can be seen in the perinephric fat surrounding renal cell carcinomas, if looked for closely, and it is likely that the present cases represent a more advanced stage of this same unusual reactive process. Although it is in theory possible that recognition of this distinctive pseudoneoplastic process might serve to identify patients with unrecognized renal disease, its chief significance more likely lies in its ability to mimic other more ominous perinephric tumors, in particular myxoid and inflammatory variants of well-differentiated liposarcoma. Awareness of this unusual process, careful morphological evaluation, and appropriate ancillary testing (eg, *MDM2* FISH, IgG4, β-catenin and melanocytic marker immunohistochemistry) should allow for its confident diagnosis.

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