Diagnostic Pathology

CASE REPORT

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Perinephric myxoid pseudotumor of fat – histopathological and molecular characterization of 3 cases after renal transplantation

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Abstract

Background Perinephric myxoid pseudotumor of fat (PMPF) is a rare benign pseudo-neoplastic proliferation of the perinephric and renal sinus adipose tissue. Its pathogenesis is thought to be a reactive process typically associated with neoplastic and non-neoplastic end-stage kidney disease. The distinctive histopathological feature of PMPF is a myxoid process comprising bland, spindled stromal cells interspersed with mature adipose tissue. Macroscopically, it is characterized by tumorous lipomatous remodeling of the kidney, which may raise concerns of malignancy on imaging. To date, only seven cases of PMPF have been documented in the context of kidney transplantation.

Case presentation This report describes three cases of PMPF in patients following renal transplantation, involving both native and grafted kidneys. Macroscopically, all cases consisted of shrunken kidneys with thinned and atrophic renal parenchyma surrounded by massively hypertrophic perirenal fat with mass-forming nodules, which was in concordance with cross sectional imaging findings acquired before surgery. Histology of the remaining renal parenchyma showed end stage renal disease in all four surgically removed kidneys, with diffuse interstitial fibrosis, tubular atrophy and sclerosed glomeruli. Perirenal adipose tissue consisted of mature fat with areas of significant myxoid and collagenous stromal component, interspersed with bland spindle and stellate-shaped cells. Immunohistochemistry for S100, smooth muscle actin, desmin and IgG4 were negative. No *MDM2* gene amplification was identified by fluorescence in situ hybridization. Broad molecular profiling using the FoundationOne®Heme assay revealed no evidence of pathogenic alterations on DNA and RNA levels.

Conclusion PMPF is a rare benign condition typically associated with chronic kidney disease, occurring late in the course. The radiological findings may be mistaken for those of a malignant tumor, and histopathological examination is required to exclude a malignant neoplasm, in particular a well-differentiated or dedifferentiated liposarcoma of the retroperitoneum. Renal transplant recipients can be affected by PMPF, which can occur in both native and transplanted kidneys several years following renal transplantation.

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Keywords Perinephric myxoid pseudotumor of fat, Renal neoplasms, Kidney transplant, Chronic kidney disease, Diagnostic assessment, Liposarcoma, *MDM2*

Introduction

Perinephric myxoid pseudotumor of fat (PMPF) is a relatively recently described pathological entity characterized by tumor-like changes of the perinephric and renal sinus adipose tissue. Histologically, the lesion typically consists of mature adipose tissue, myxoid stroma, bland spindled cells, a mixed inflammatory cell infiltrate and arborizing vessels [1]. PMPF is associated with various chronic kidney diseases, including neoplastic diseases of the kidney, such as renal cell carcinoma and urothelial carcinoma, as well as non-neoplastic chronic kidney diseases, such as end-stage renal failure, diabetes mellitus, ureteral stricture, chronic pyelonephritis and congenital anatomical abnormalities [1–3]. The precise pathogenesis of PMPF remains unknown. However, it is hypothesized that PMPF is a reactive process in response to chronic "irritation" of the kidney, either due to the mass effect of neoplasia or the inflammation that frequently accompanies non-neoplastic chronic kidney disease [1].

In 2009, Tanas et al. were the first to describe this condition as "pseudosarcomatous fibroblastic/myofibroblastic proliferation" in perinephric tissue adjacent to renal cell carcinoma in 12 cases [4]. Dashti et al. introduced the term "perinephric myxoid pseudotumor of fat" in 2019 by the identification of similar lesions in perinephric adipose tissue in 11 cases primarily associated with non-neoplastic renal disorders [1]. In 2023, Hogan et al. published a large case collection describing 33 cases of PMPF, the majority of which (82%) were associated with malignancy, such as renal cell carcinoma, and urothelial carcinoma of the renal pelvis [2].

Given its strong association with chronic kidney disease, it is not surprising that PMPF can also manifest in the context of kidney transplantation. Chronic graft failure and end-stage kidney disease is a common scenario in long-term kidney transplant recipients [5]. However, only seven cases of PMPF in kidney transplants have been reported in the literature to date [6, 7, 11] (Table 1). Here we present two more cases of PMPF in transplanted kidneys and one case in which both native kidneys of a transplant patient were affected.

Case presentation

Case 1 was a 63-year-old male patient who underwent double kidney transplantation 28 and 16 years ago for chronic glomerulonephritis. Due to a persistent inflammatory condition of unknown etiology and microhematuria, a CT scan was performed. Two tumorous lesions in both native kidneys were found. The radiological differential diagnosis favored angiomyolipoma, but a malignant neoplasm could not be excluded with certainty. Bilateral nephrectomy of both native kidneys was performed to exclude malignancy and confirmed the diagnosis of PMPF in both kidney resections. Despite the patient's successful recovery from surgery, he eventually died two weeks later from a myocardial infarction.

Case 2 was a 61-year-old male patient with dialysis-dependent graft failure after double kidney transplantation 28 and 25 years earlier due to mesangioproliferative glomerulonephritis. Sonography and MRI detected an incidental solid and cystic mass in the first transplant kidney. As the lesion was suspicious for neoplasia on imaging, transplant nephrectomy was performed with a postoperative diagnosis of PMPF. Meanwhile, 12 years after the transplant nephrectomy, the patient shows no evidence of disease recurrence and has since undergone a successful re-transplantation.

Case 3 was a 61-year-old male patient who received a kidney transplant 20 years ago for focal segmental glomerulosclerosis. He had been on dialysis for 12 years after chronic graft failure. Due to recurrent pyelonephritis, a CT scan was performed that revealed several suspicious lesions in the transplant kidney. Biopsy showed normal mature adipose tissue next to connective tissue with a bland spindle cell proliferation in a loosely vascularized stroma, with no evidence of malignancy. *MDM2* fluorescence in situ hybridization assay of the biopsy excluded amplification of the *MDM2* gene. However, the diagnosis of PMPF was not made at the time of biopsy as this entity was not initially considered. Due to persistent graft site discomfort, the patient requested removal of the transplanted kidney and the surgical specimen was diagnosed with PMPF. Currently, the patient shows no complications and no signs of disease recurrence one year after the diagnosis of PMPF and surgery.

Radiological findings

In *case 1*, a renal mass protocol CT (non-contrast and contrast enhanced) showed a massive thinning of remaining renal parenchyma of both native kidneys, with a width < 4 mm. The perinephric tissue of both kidneys exhibited diffuse hypertrophy with focal nodular remodeling (bilateral masses of 15.0 × 10.0 × 8.0 cm), with an average density of approximately -60 to -75 Hounsfield units (HU), consistent with lipomatous tissue. The more nodular component displayed increased density (< 15 HU) and demonstrated contrast enhancement, which was suspicious for a malignant process (Fig. 1A).

Table 1 Cases of perinephric myxoid pseudotumor of fat reported in the literature

Author	Year	No. of Cases	Underlying Disease	Native Kidney vs. Transplant Kidney
Tanas, et al. [4]	2009	12	Renal cell carcinoma	Native kidney (12/12)
Dashti, et al. [1]	2019	11	Non-neoplastic chronic kidney disease	Native kidney (11/11)
Thoeni, et al. [6]	2021	1	Non-neoplastic chronic kidney disease, transplant failure	Transplant kidney (1/1)
Chen, et al. [10]	2021	1	Non-neoplastic chronic kidney disease	Native kidney (1/1)
Pham, et al. [7]	2022	2	Non-neoplastic chronic kidney disease, transplant failure	Transplant kidney (2/2)
Collins, et al. [34]	2022	1	Non-neoplastic chronic kidney disease	Native kidney (1/1)
Hogan, et al. [2]	2023	33	Malignancy (27/33 cases, 82%) Non-neoplastic chronic kidney disease (6/33 cases, 18%)	Native kidney (33/33)
Lee, et al. [8]	2023	4	Non-neoplastic chronic kidney disease (3/4 cases, 75%) No associated kidney disease reported (1/4 case, 25%)	Native kidney (4/4)
Liu, et al. [3]	2023	1	Horseshoe kidney with obstructing renal calculus	Native kidney (1/1)
Amato, et al. [35]	2023	1	Ureteropelvic junction obstruction	Native kidney (1/1)
Ortiz-Rey, et al. [36]	2023	1	Urothelial carcinoma in situ of ureter	Native kidney / ureter (1/1)
Broski, et al. [11]	2024	17	Non-neoplastic chronic kidney disease (11/17 cases, 65%) No associated kidney disease reported (6/17 cases, 35%)	Native kidney (13/17) Transplant kidney (4/17)
Kallen, et al. [37]	2024	1	Polycystic kidney disease	Native kidney (1/1)
Wu, et al. [38]	2024	13	End-stage renal disease and renal cysts (9/13 cases, 70%) Renal neoplasm (2/13 cases, 15%) Myeloma (1/13 cases, 7.5%) Lymphoma (1/13 cases, 7.5%)	Native kidney (13/13)
Total No. of Cases		**99**		**Native kidney (92/99)** **Transplant kidney (7/99)**

In *case 2*, a non-contrast CT scan revealed a markedly atrophied parenchyma of the left transplanted kidney, encased in multiple lobulated and round-shaped lesions of low density (< 20 HU), measuring up to 8.9 × 7.0 × 4.0 cm (Fig. 1C). These findings again raised concerns about a malignant neoplastic process.

MR imaging in *case 3* demonstrated large lobulated solid lesions up to 4.8 × 2.5 × 1.9 cm adjacent to the poles of the transplanted kidney in the right iliac fossa. Extended degenerative atrophy of the renal parenchyma as well as simple cortical parenchymal cysts (max. 0.6 cm) were observed (Fig. 1E). The perinephric masses showed a marked contrast enhancement and minimal diffusion restriction, so that a malignant process could not be excluded in the differential diagnosis.

Gross examination

In *case 1*, the explanted native kidneys weighed 667 g (right kidney) and 968 g (left kidney). The parenchyma was thin and atrophic with blurring of the cortico-medullary junction. The perirenal fat of both kidneys was massively hypertrophic and surrounded the shrunken kidneys (Fig. 1B). In *case 2*, the weight of the kidney was 1280 g, there was only a rim of residual renal parenchyma surrounded by nodular tissue with fatty appearance (Fig. 1D). In *case 3*, the kidney weighed 420 g. The renal parenchyma was markedly thinned and showed multiple cysts up to 0.6 cm in diameter, with a perirenal fatty mass with mucoid, glassy nodules enclosing the shrunken kidney (Fig. 1F). In all cases, PMPF was characterized by diffuse hyperplasia of the perirenal adipose tissue adjacent to atrophic renal parenchyma, rather than presenting as a sharply demarcated mass.

Histology, immunohistochemistry and molecular analysis

Histology of the remaining renal parenchyma showed end-stage kidney disease in all four surgically removed kidneys, with diffuse interstitial fibrosis, tubular atrophy and sclerosed glomeruli (Fig. 2A-C). Case 3 exhibited multiple cysts lined by flattened and cuboidal cells with clear, foamy or eosinophilic cytoplasm, and few oxalate crystals, consistent with acquired cystic kidney disease. Furthermore, there was chronic active C4d-negative antibody-mediated rejection in the residual parenchyma of case 3, presenting with transplant glomerulitis, transplant glomerulopathy, and chronic allograft arteriopathy. In all cases, perirenal adipose tissue consisted of mature fat as well as areas with a significant myxoid and collagenous stromal component, interspersed with bland spindled and stellate-shaped cells without cytologic atypia (Fig. 2D-J). Patchy chronic inflammation with lymphocytes, plasma cells and CD68 + macrophages was present. In areas of stromal myxoid change, thin-walled arborizing vessels were observed. Mitotic activity and necrosis were absent and there was no evidence of malignancy. Immunohistochemistry for S100, smooth muscle actin, desmin and IgG4 performed on all resections were negative. The Ki-67 proliferation index was < 5%. In all cases, no *MDM2* gene amplification by fluorescence in situ hybridization was identified (Fig. 2K-M). Broad

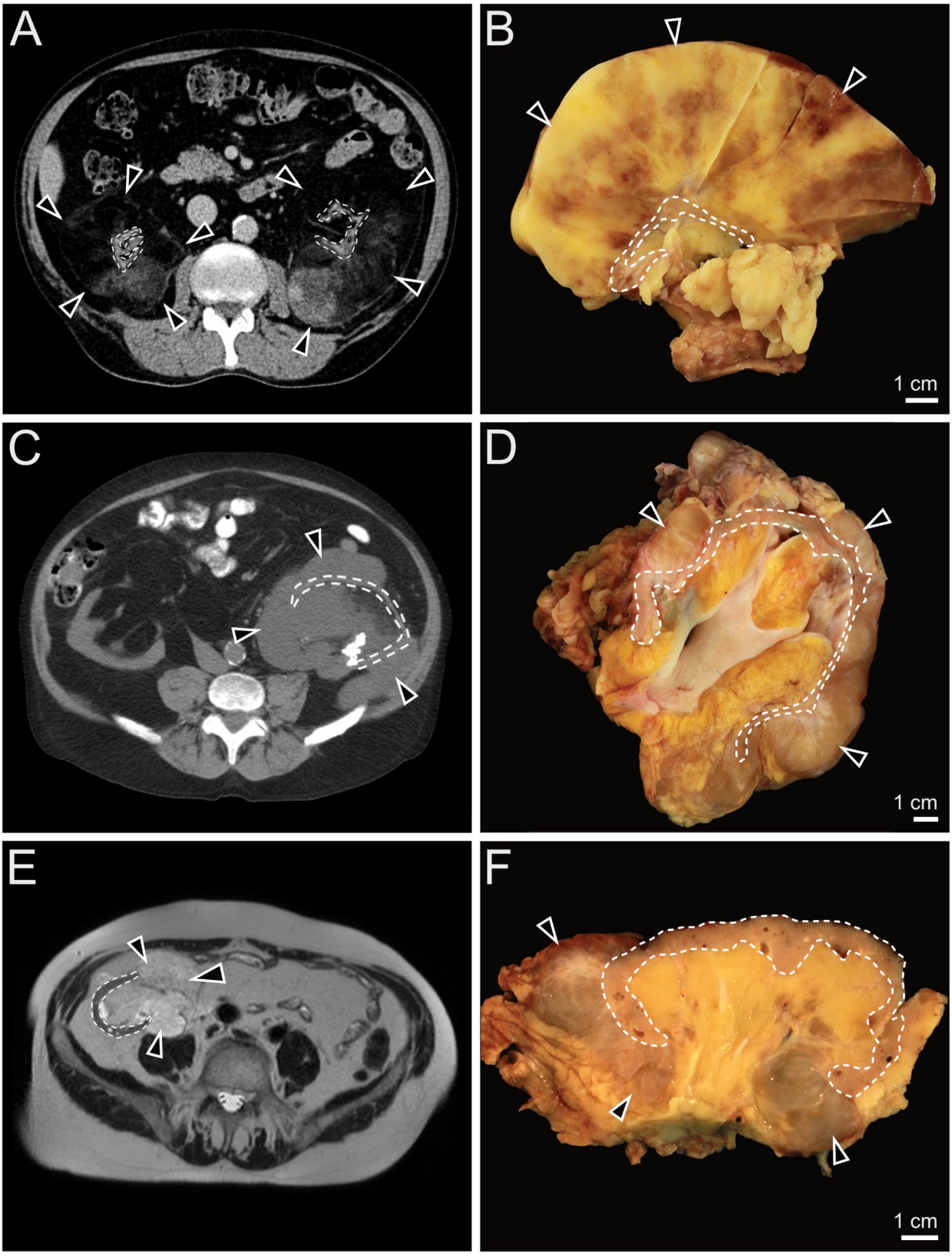

Fig. 1 (See legend on next page.)

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Fig. 1 Radiological and macroscopic appearance of perinephric myxoid pseudotumor of fat (**A,C,E**) Radiologically, massive thinning of the remaining renal parenchyma (dashed lines) with hypertrophy and partially nodular remodeling of the perinephric tissue (arrowheads) gives the impression of mass-forming lesions. (**B,D,F**) Nephrectomy specimen show hypertrophied, ill-defined and nodular perirenal fat (arrowheads) adjacent to thinned, atrophic kidney parenchyma (dashed line). (**A**-**B**) Case 1: PMPF in both native kidneys (in B only the left larger kidney is shown, however, macroscopic findings of the right kidney were comparable). (**C**-**D**) Case 2: PMPF in kidney transplant. (E-F) Case 3: PMPF in kidney transplant

molecular profiling using the FoundationOne®Heme assay in all three cases did not reveal any pathogenic alterations on DNA (e.g. mutation) and RNA (e.g. fusion) levels.

Discussion

Our three cases of PMPF in kidney transplant recipients share many similarities with previously described cases of PMPF. All three patients presented with chronic graft failure and end-stage kidney disease. In all of them, imaging revealed perinephric lesions with a tumor-like appearance, favoring a neoplastic process. Diagnosis of PMPF on imaging is challenging due to the wide range of radiological findings and the limited literature describing the imaging appearance of PMPF. Several potential mimics of malignancy (e.g. liposarcoma) and a wide range of non-neoplastic differential diagnoses, including infectious conditions, cysts, or IgG4-related disease have been reported in the context of PMPF [8–11]. Radiology has been described as challenging to clearly distinguish between an exophytic process arising from the kidney itself and a lesion arising from perinephric fat [8]. Due to this ambiguity in imaging, histological examination remains the most accurate diagnostic tool for identifying PMPF and is required in the majority of cases to exclude malignancy. This is particularly important for kidney transplant recipients, who are at increased risk of developing malignant tumors in both their native and transplanted kidneys [12, 13]. However, the findings in a biopsy of PMPF may be inconclusive, as PMPF consists of different histological components, not all of which may be seen in the biopsy material. In addition, PMPF is an unusual and rare entity and PMPF might not be considered initially.

The main histological features prompting to the diagnosis of PMPF are a very prominent myxoid stroma with loosely arranged spindle cells without marked atypia and low mitotic activity, interspersed with unsuspicious mature adipose tissue. A mixed inflammatory infiltrate and delicate blood vessels may be present. In our cases, PMPF consisted of tumorous, mass-forming myxo-lipomatous remodeling exclusively of the perirenal tissue, without involvement of the atrophic renal parenchyma itself. This finding is consistent with previously published cases [1, 2]. However, other authors also described extension and infiltration of PMPF into the renal parenchyma [6].

The characteristic myxoid stroma distinguishes PMPF from other potential differential diagnoses, such as retroperitoneal lipoma or angiomyolipoma [14]. However, one of the most important differential diagnostic considerations for a retroperitoneal fat containing lesion is a well-differentiated or dedifferentiated liposarcoma [15, 16]. The morphological spectrum of liposarcoma is wide, including a prominent myxoid appearance [17]. The amplification of the mouse double minute 2 homolog (*MDM2*) gene is the leading oncogenic pathway and diagnostic hallmark of well-differentiated and dedifferentiated liposarcoma [18]. We and others suggest that a definitive diagnosis of PMPF requires demonstration of absence of *MDM2* gene amplification by fluorescence in situ hybridization (FISH) analysis [1]. Myxoid liposarcoma is another mesenchymal malignancy that shares morphological characteristics with PMPF. However, this entity is genetically defined by the presence of *DDIT3* gene fusions and appears to be exceedingly rare in the retroperitoneum [19, 20].

Benign differential diagnoses to consider include renal replacement lipomatosis (RRL), renal sinus lipomatosis (RSL) and renal fibrolipomatosis (RFL). These terms are often used synonymously in the literature and refer to a spectrum of entities characterized by varying degrees of accumulation of fatty tissue within the renal sinus and replacement of destroyed renal parenchyma by adipose tissue [21, 22]. While RSL is defined as an only moderate increase in renal sinus fat, RRL and RFL appear to be more severe forms with marked fatty tissue proliferation within the renal sinus, renal atrophy or destruction of the renal parenchyma [23–25]. Calculus disease, infection, non-inflammatory chronic kidney diseases and obesity have been discussed as underlying causes [22]. Macroscopic and radiological appearance and some histological features of RRL may be similar to PMPF. However, RRL is histologically composed of fibrofatty tissue with mature adipocytes, but lacks the characteristic myxoid matrix and spindled-cell component of PMPF [26, 27]. Cross-sectional imaging may reveal findings suggestive of either PMPF or RRL/RFL, yet it can usually not confirm either diagnosis definitely. While RRL is generally associated with renal calculi and renal parenchymal destruction, PMPF is considered to be an independent low density mass or masses with HU values ranging from fat-like (negative HU) to slightly higher than water (low positive HU), which may show minimal contrast enhancement. Additionally, chemical shift imaging MRI

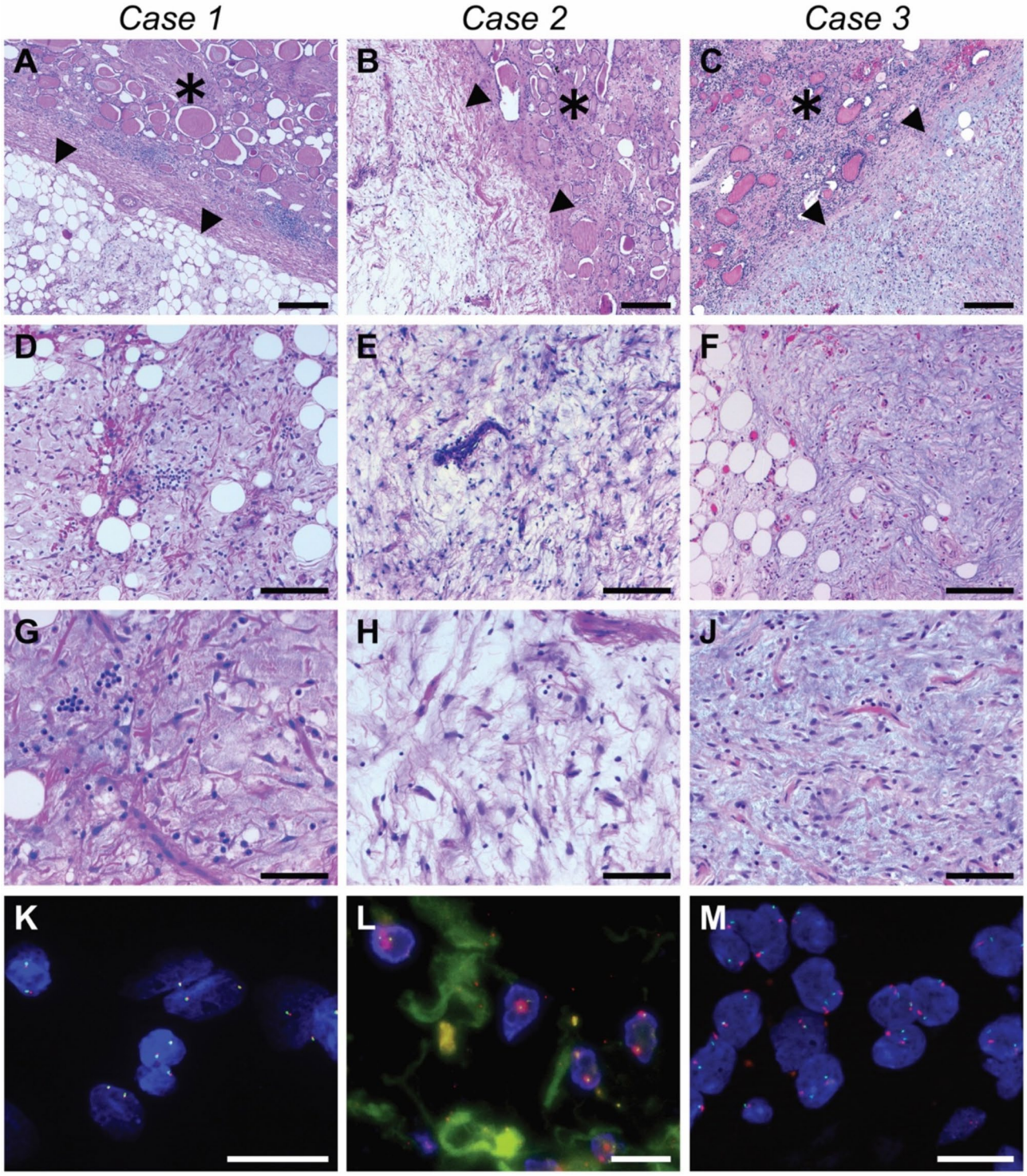

Fig. 2 Histological appearance of perinephric myxoid pseudotumor of fat
(**A**-**C**) Renal parenchyma shows the picture of an end-stage-kidney with sclerosis of the glomeruli, interstitial fibrosis and tubular atrophy (✻). PMPF is directly adjacent to the renal capsule (▼) (HE, scale bar = 250 µm). (**D**-**F**) PMPF consists of an admixture of mature fat, fibromyxoid stroma and spindled to stellate stromal cells with a variably intense mixed inflammatory cell infiltrate (HE, scale bar = 100 µm). (**G**-**J**) Spindled to stellate cells of PMPF without cytological atypia (HE, scale bar = 50 µm). (**K**-**M**) *MDM2* fluorescence in situ hybridization: No amplification of the *MDM2* gene in all three cases (scale bar = 10 µm).

typically identifies both macroscopic fat masses and fat-suppressed T2-weighted MRI irregularities of myxoid components within suspected PMPF [8, 10, 11]. Of note, imaging features may differ in native and transplanted kidneys [11].

Renal myxoma is another condition that may exhibit morphological overlap with PMPF. This rare kidney tumor has only been reported in a limited number of cases [28–30], including one transplant kidney [31]. Typical histological features of renal myxoma include a hypervascular myxoid stroma with spindle cells [29, 32, 33]. However, myxomas typically do not contain mature adipose tissue, which is the main distinguishing factor from PMPF [28, 33].

The prognosis of PMPF is generally benign, and no cases of recurrence or malignant transformation have yet been reported. In our cases, all patients had a favorable immediate post-operative outcome. While one patient died shortly after surgery due to an unrelated illness, the remaining two patients show no signs of disease recurrence in the medium and long term.

In conclusion, PMPF is a benign condition typically associated with end-stage kidney disease, occurring late in the course. Radiological findings may be mistaken for those of a malignant tumor, and a histopathological examination is usually required to rule out a malignant neoplasm, particularly retroperitoneal well- or dedifferentiated liposarcoma. Renal transplant recipients can be affected by PMPF, which can occur in both native and transplanted kidneys.

Abbreviations

CT	Computer tomography
DDIT3	DNA Damage Inducible Transcript 3
FISH	Fluorescence in situ hybridization
HU	Hounsfield unit
MDM2	Mouse double minute 2 homolog
MRI	Magnetic resonance imaging
PMPF	Perinephric myxoid pseudotumor of fat
RFL	Renal fibrolipomatosis
RRL	Renal replacement lipomatosis
RSL	Renal sinus lipomatosis

Author contributions

CS: Conceptualization (supporting); project administration (equal); visualization (lead); writing – original draft (lead). AG: Conceptualization (equal); supervision (lead); validation (equal); writing – original draft (supporting); writing – review and editing (lead). MB: Providing clinical data (lead); review and editing (equal). FAH: Providing radiological data (lead); review and editing (equal). CP: Providing molecular data (lead); review and editing (equal). BMH, TF, BBL: Writing – review and editing (equal). All authors read and approved the final manuscript.

Funding

This research received no specific grant from any funding agency in the public, commercial, or non-for-profit sectors.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent for publication

This case series has been assembled in accordance with the Declaration of Helsinki. Written informed consent was obtained from patients 2 and 3 for publication. Patient 1 died more than 12 years ago and no relatives could be contacted at this time.

Competing interests

The authors declare no competing interests.

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Received: 9 November 2024 / Accepted: 10 February 2025
Published online: 01 March 2025

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