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Pelvic Lipoma: A Rare Cause of Occult and Progressive Obstructive Nephropathy in a Transplanted Kidney

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Abstract

Background: Pelvic lipoma leading to occult obstruction as a late failure in a kidney graft (KG) is an extremely rare and challenging diagnosis. **The Case:** A 38-year-old man with a KG for 23 years presented with progressive fluid overload and renal failure. Pelvic ultrasound revealed an 8 cm echogenic KG that lacked significant hydronephrosis. However, significant fat was seen surrounding its pelvis and ureter. Computerized tomography disclosed a pelvic mass with homogeneous fatty content and a well-defined capsule. The mass lacked hypermetabolism on PET scanning, and the KG function was poor on MAG III estimate. The mass, with its intact capsule and its encapsulated KG, was removed. Its biopsy showed rounded fat cells that lacked inflammation, fibrosis, granulomata, muscle, vascular proliferation, and mitosis. Biopsy of the KG showed end-stage features. For the past 2 years, the patient has been stable on maintenance hemodialysis and is awaiting future kidney transplantation. **Conclusion:** Pelvic lipoma can lead to occult and progressive KG loss due to chronic obstruction, without hydronephrosis, simulating chronic rejection. Its differential diagnosis includes liposarcoma, lipomatosis, teratoma/dermoid cysts, and granulomatous disease. Its removal with an intact capsule is essential to avoid fat leak and future recurrence.

Keywords

Lipoma, Graft Failure, Transplant, Pelvic, Obstruction, Rejection

1. Introduction

Compared to dialysis, kidney transplantation (KT) is the preferred treatment for patients with end-stage kidney disease due to its better long-term survival and morbidity [1]. The first successful KT was performed by Dr. Joseph Murray in 1954, and since then major developments in transplantation technique and immunology have been achieved, allowing safer and wider selection of acceptable donors and recipients [2]. Despite the substantial variations in KT incidence, prevalence, availability, accessibility, and quality worldwide, its incidence and prevalence were 14 and 255 per million population by 2022 [3]. Adjusted 5-year kidney graft (KG) survival from living donors reached an all-time high of 87.1% by 2015, while the corresponding percentage from deceased recipients was 75.6% [4]. Causes of KG loss include death with function due to cardiovascular diseases, cancer, and opportunistic infections, as well as chronic rejection, vascular thrombosis/stenosis, recurrence of primary nephropathy, and interstitial fibrosis due to calcineurin-inhibitors [5]. Obstruction accounts for 2% - 10% of KG loss. The culprits are 1) early ones due to ureteric ischemia, a narrow anti-reflux tunnel at the ureterovesical anastomosis, or external compression by a lymphocele or hematoma, and 2) late ones such as perinephric hematoma from periodic kidney biopsies leading to page kidney, hygroma due to lymphatic obstruction, renal cysts, and tumors [6]. Hydronephrosis is the hallmark of obstruction [7]. However, such a sign may be lacking in perirenal, peripelvic, and periureteric encapsulating masses/tumors, with KG loss mimicking progressive chronic rejection. In our case report, we present a patient with such a rare presentation and highlight its management.

2. The Case

A 38-year-old man presented with progressive shortness of breath for 2 months. The patient had KT 23 years ago for previous dysplastic kidneys. He had normal perioperative and follow-up since then. Unfortunately, for the past 9 months, he gradually developed fluid overload and progressive renal disease. His previous medications included Prednisone 5 mg daily, Rapimune 1 mg daily, Mycophenolate 250 mg twice daily, and Amlodipine 10 mg daily. He was afebrile and had normal blood pressure. His body weight was 60 kg. Physical examination was normal except for bilateral basal rales on chest auscultation, as well as bilateral sacral and lower limb oedema. Laboratory tests showed normal peripheral leucocytic and platelet counts. Hemoglobin was 10 g/L with normal transferrin saturation% and vitamin B12 level. Serum urea and creatinine were 36 mmol/L and 560 umol/L, respectively. Serum electrolytes, liver function tests, TSH, cholesterol, and triglyceride were normal. Urine routine and microscopy did not show abnormality. Chest x-ray and ECG were normal. Abdominal and pelvic ultrasound did not show abnormality except for bilateral small native kidneys and fat surrounding his 8 cm echogenic KG that lacked significant hydronephrosis (**Figure 1**). Plain computerized tomographic scan of the pelvis confirmed a 10 cm fat-density mass

extending from the subhepatic region to the right side of the pelvic cavity. It was displacing adjacent bowel loops and urinary bladder contralaterally, yet had a well-defined capsule without adjacent tissue invasion and lymphadenopathy. It extended into the KG sinus and encapsulated its pelvis and ureter (Figure 2). PET scan did not show hypermetabolic pelvic uptake. On MAG III scan, KG function was extremely poor. Hence, the patient was subjected to total excision of the mass with the KG and ureter till the urinary bladder. On grossing, the mass was homogeneous, soft, yellow, with an intact capsule and without evidence of tissue invasion and lymphadenopathy. Histopathological examination of multiple random biopsies showed rounded fat cells that lacked inflammation, fibrosis, vascular proliferation, and mitosis (Figure 3). Biopsy of the KG revealed severe interstitial fibrosis and global glomerulosclerosis without immune deposits (Figure 4). The patient was placed on maintenance hemodialysis and immunosuppressive drugs were withdrawn. On follow-up, he remained stable without disease recurrence, on follow-up MRI scans, for more than 2 years. He is planned to receive future KT.

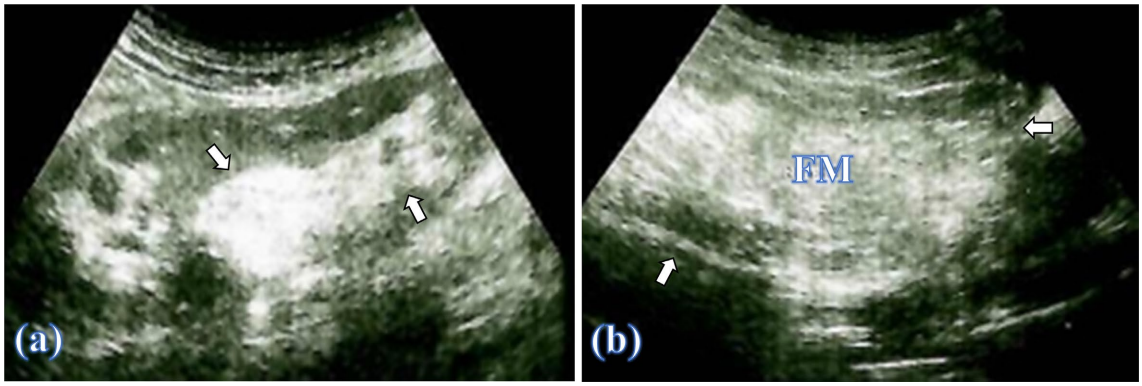

Figure 1. Right pelvis ultrasound picture showing: (a) a 9 cm kidney graft without hydronephrosis yet with fat in its sinus (Arrows) and (b) its surrounding pelvic fatty mass (FM) within a capsule (arrows).

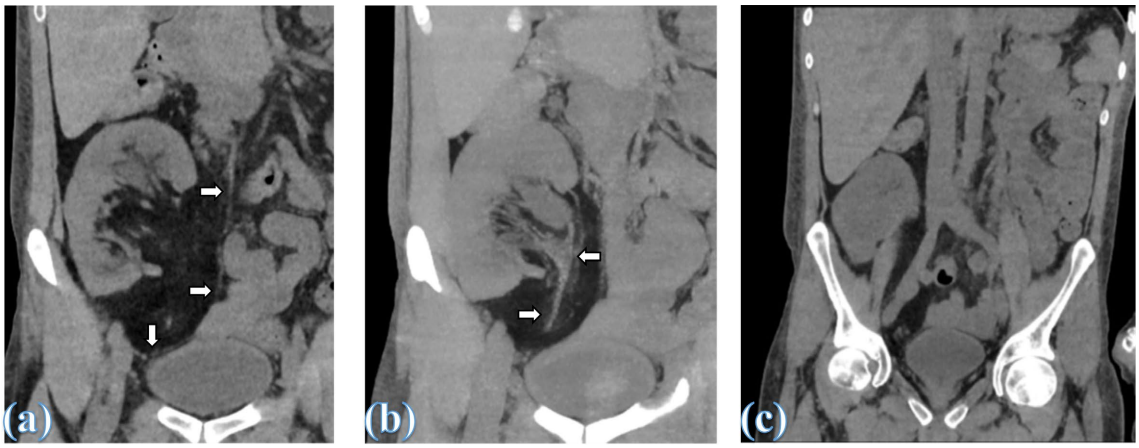

Figure 2. CT scan pictures of the pelvis showing: (a) a large homogeneous fatty tissue extending into its renal sinus and with an intact capsule (Arrows), (b) its encapsulation of the transplanted kidney pelvis and its ureter (Arrows), and (c) absence of iliac and para-aortic lymphadenopathy.

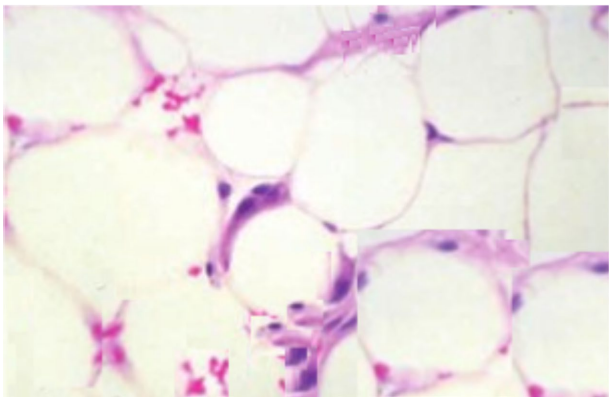

Figure 3. Photomicrograph of post-operative tissue biopsy of pelvic mass showing rounded Mature fat cells that lack inflammation, fibrosis, vascular proliferation, and mitosis.

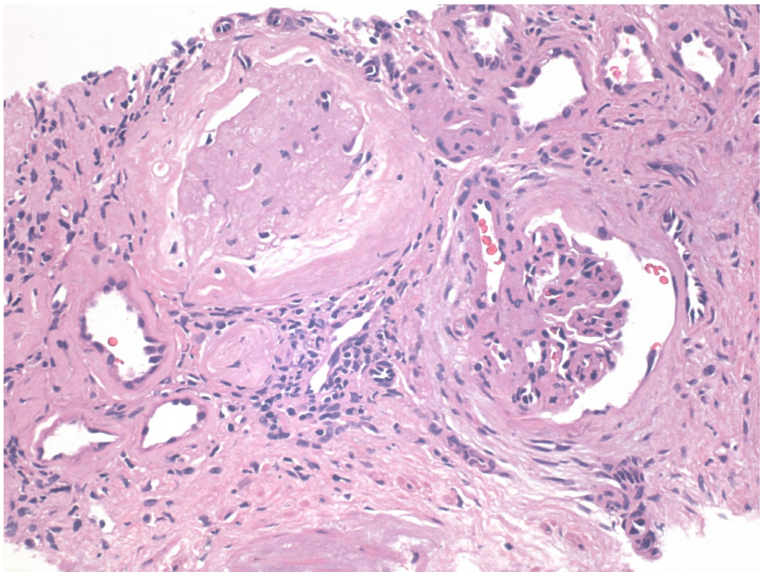

Figure 4. Photomicrograph of a kidney biopsy showing tubular thyroidization and atrophy. with interstitial fibrosis and globally sclerosed glomeruli with periglomerular fibrosis (H & EX400).

3. Discussion

Lipomata are benign encapsulated mesenchymal tumors of adipose tissue that occur in almost all parts of the body where fat normally exists. However, deep-seated pelvic lipomata (PL) are extremely rare [8]. Moreover, reports on its association with transplanted kidneys and their failure are lacking. It is a dysplastic disorder associated with proliferation of mesenchymal primordial fat cells, not the adult ones, raising an issue of hereditary predisposition [9]. Its differential diagnosis is summarized in **Table 1**. They include pelvic lipomatosis, liposarcoma, teratoma/dermoid cyst, retroperitoneal fibrosis, and granulomatous disease viz. idiopathic (autoimmune) or infections (tuberculosis and bilharziasis) [10]. PL has the following features: 1) a non-enhancing homogeneous fatty mass with a distinct capsule that lacks significant septa by CT scanning, 2) composed of normal

Table 1. Differential diagnosis of retroperitoneal masses.

	Lipoma	Lipomatosis	Liposarcoma	Retroperitoneal fibrosis	Teratoma/dermoid cyst	Chronic granulomatous disease	
						Idiopathic	Secondary
Etiology	Dysplasia	Autosomal dominant inflammatory reaction	Malignancy	Autoimmune	Germ cell benign tumor (rarely malignant)	Autoimmune	(TB/Bilharziasis)
Incidence (100,000 individuals/year)	Rare case reports	0.6 - 1.7	0.25	1.4	3	0.5	No report
Main Location	Retroperitoneal	Pelvic	Retroperitoneal	Periureteric	Pelvic (ovaries & testicles)	Ureters, bladder and prostate	
Presentation & imaging (US/CT)	Encapsulated fat	Non-capsulated fibro-adipose (mesh-appearance)	Enhancing heterogenous mass with multiple septa	Hyperechoic diffuse peri-ureteric mass with hydronephrosis CT enhancement: early not late (fibrosis)	Cystic mesh/cyst Heterogeneous echogenic mass with calcifications and Fat fluid	Mass+ obstruction Enhancing solid mass	
Histology	Small rounded fat cells	Sheets of Fat ± skeletal muscle	Large spindle-shaped fat cells	Fibroblasts, storiform collagen, lymphocytic and plasma cells (IgG4-positive)	Mature tissues of 3-mesenchymal layers with calcification, sebum, and hair	Granulomatous mass	
Treatment	Surgery	Surgery	Surgery	Immunosuppression /ureterolysis	Surgery	immunosuppression	Specific antibiotics
Recurrence	IIRx	Common	IIRx	Common	IIRx	IIRx	IIRx

Abbreviations: TB: tuberculosis, IIRx: if inadequately treated, US: ultrasonography, CT: computerized tomography.

fat cells (small, rounded, and without mitosis) on histopathology, 3) can cause slow-compressive complications as seen in our patient, and 4) should be removed with the intact capsule to avoid fat leak and future recurrence (5%) [11]. Moreover, pre-operative mass biopsy is inadequate to exclude liposarcoma since the latter can be focal. Liposarcoma is the most common pelvic fatty tumor (80%) and is malignant. Liposarcoma tends to have rapid progression, evidence of local invasion and metastasis, as well as diffuse heterogeneous echotexture (with thick septa > 2 mm) and enhancement with contrast CT [12]. On histology, it is composed of large, spindle-shaped cells with mitosis. The second similar lesion is teratoma in female ovaries, which can present as a cyst (dermoid cyst) or as a heterogeneous solid mass with echogenic focus causing acoustic shadowing due to calcification, hair, and sebum (hair-fluid), as well as fat fluid and hair fluid levels. Rarely, it can progress to teratosarcoma. The third pelvic lesion is pelvic lipomatosis. It is a rare benign non-capsulated fibro-adipose inflammatory response to pelvic inflammation and cystitis glandularis [13]. It is a familial disease with autosomal dominant inheritance. On histopathology, five types were reported: 1) conventional lipoma, 2) angiolipoma, 3) fibrolipoma, 4) fusiform cell lipoma, 5)

myelolipoma, and pleomorphic ones [8]. On histopathological examination, and contrary to simple PL, it exhibits 1) dense, vascular lipomatous tissue, and 2) 30% inflammation and vascular proliferation [13]. Moreover, immunohistochemical stains, though positive for S-100 at mature adipocytes, are also positive for desmin and smooth muscle actin in the fibromatous areas [9]. Pelvic lipomatosis, though benign, can present as an abdominal mass associated with both pressure and invasion manifestations to adjacent organs, viz. hematuria, urinary stone symptoms, painful ejaculation, constipation, tenesmus, rectal bleeding, lower limb edema, and deep vein thrombosis [13]. In management of fatty pelvic masses, three rules should be respected: 1) liposarcoma should be considered until exclusion by adequate histopathology, 2) preoperative percutaneous biopsy should be avoided for risk of neoplastic or inflammatory cell leak with risk of future recurrence, and 3) surgery offers the definitive approach [12]. Moreover, two solid urinary tract obstructive lesions can be encountered: 1) retroperitoneal fibrosis, and 2) granulomatous disease. Lesions of the former are mainly abdominal rather than pelvic and its treatment indicates ureterolysis and immunosuppression [14]. On the other hand, granulomatous disease can affect the ureters and prostate [15]. Diagnosis can be established by lesional biopsy. The idiopathic disease shows non-caseating granulomata which is amenable to corticosteroids and mycophenolate. On the other hand, secondary disease can develop after specific infections viz. tuberculosis (caseating granulomata) or bilharziasis (ova-associated) and indicates specific therapy for each [16].

4. Conclusion

In KG, PL can lead to obstructive uropathy without significant hydronephrosis if it encapsulates the kidney and/or its pelvis and ureters.

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Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Wolfe, R.A., Ashby, V.B., Milford, E.L., Ojo, A.O., Ettenger, R.E., Agodoa, L.Y.C., *et al.* (1999) Comparison of Mortality in All Patients on Dialysis, Patients on Dialysis Awaiting Transplantation, and Recipients of a First Cadaveric Transplant. *New England Journal of Medicine*, **341**, 1725-1730. https://doi.org/10.1056/nejm199912023412303

[2] Poggio, E.D., Augustine, J.J., Arrigain, S., Brennan, D.C. and Schold, J.D. (2021) Long-Term Kidney Transplant Graft Survival—Making Progress When Most Needed. *American Journal of Transplantation*, **21**, 2824-2832. https://doi.org/10.1111/ajt.16463

[3] Mudiayi, D., Shojai, S., Okpechi, I., Christie, E.A., Wen, K., Kamaleldin, M., *et al.* (2022) Global Estimates of Capacity for Kidney Transplantation in World Countries and Regions. *Transplantation*, **106**, 1113-1122. https://doi.org/10.1097/tp.0000000000003943

[4] United States Renal Data System (2022) Chapter 7 (Transplantation). https://usrds-adr.niddk.nih.gov/2022/end-stage-renal-disease/7-transplantation

[5] El-Zoghby, Z.M., Stegall, M.D., Lager, D.J., Kremers, W.K., Amer, H., Gloor, J.M., *et al.* (2009) Identifying Specific Causes of Kidney Allograft Loss. *American Journal of Transplantation*, **9**, 527-535. https://doi.org/10.1111/j.1600-6143.2008.02519.x

[6] Berger, P.M. and Diamond, J.R. (1998) Ureteral Obstruction as a Complication of Renal Transplantation: A Review. *Journal of Nephrology*, **11**, 20-23.

[7] Rishor-Olney, C.R. and Hinson, M.R. (2023) Obstructive Uropathy. StatPearls Publishing.

[8] Martinez, C.A.R., Palma, R.T. and Waisberg, J. (2003) Giant Retroperitoneal Lipoma: A Case Report. *Arquivos de Gastroenterologia*, **40**, 251-255. https://doi.org/10.1590/s0004-28032003000400010

[9] Chander, B., Krishna, M., Thakur, S., Mahajan, N., Vij, A. and Diwakaran, J. (2012) Extremely Rare Giant Retroperitoneal Fibrolipoma: A Case Report. *Journal of Cancer Research and Therapeutics*, **8**, 314-316. https://doi.org/10.4103/0973-1482.99002

[10] Miglani, U., Sinha, T., Gupta, S.K., Doddamani, D., Sethi, G.S., Talwar, R., *et al.* (2010) Rare Etiology of Obstructive Uropathy: Pelvic Lipomatosis. *Urologia Internationalis*, **84**, 239-241. https://doi.org/10.1159/000277606

[11] Barut, I., Tarhan, O.R., Ciris, M. and Tasliyar, E. (2006) Lipoma of the Parietal Peritoneum: An Unusual Cause of Abdominal Pain. *Annals of Saudi Medicine*, **26**, 388-390. https://doi.org/10.5144/0256-4947.2006.388

[12] Shaaban, A.M., Rezvani, M., Tubay, M., Elsayes, K.M., Woodward, P.J. and Menias, C.O. (2016) Fat-Containing Retroperitoneal Lesions: Imaging Characteristics, Localization, and Differential Diagnosis. *Radio Graphics*, **36**, 710-734. https://doi.org/10.1148/rg.2016150149

[13] Heyns, C.F. (1991) Pelvic Lipomatosis: A Review of Its Diagnosis and Management. *Journal of Urology*, **146**, 267-273. https://doi.org/10.1016/s0022-5347(17)37767-4

[14] Engelsgjerd, J.S., Leslie, S.W. and LaGrange, C.A. (2024) Retroperitoneal Fibrosis. StatPearls Publishing.

[15] El-Reshaid, K., Allam, A. and Mohamad, S. (2023) Prostatic Abscess Due to Idiopathic Granulomatous Disease: A Rare Complication of a Chameleon Disorder. *Open Journal of Nephrology*, **13**, 6-12. https://doi.org/10.4236/ojneph.2023.131002

[16] Williams, O. and Fatima, S. (2022) Granuloma. StatPearls Publishing.