

Case Report

Renal Pseudotumor Due to Perirenal Fat Necrosis

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We report the findings of an uncommon non-infectious cause of renal pseudotumor, perirenal fat necrosis, presenting as a native kidney mass in a renal transplant recipient. Resection was performed due to clinical suspicion for malignancy. To our knowledge this is the first report of a case of perirenal fat necrosis presenting in the transplant setting. This case emphasizes the need to continually expand our diagnostic considerations beyond epithelial and non-epithelial malignancies when confronted with native kidney masses in renal transplant recipients.

[N A J Med Sci. 2012;5(2):123-125.]

Key Words: renal pseudotumor, fat necrosis, renal transplant

INTRODUCTION

Perirenal fat necrosis is a rare renal pseudotumor often associated with a clinical history of acute pancreatitis or steroid use. Other important diagnostic features include a mass on computerized tomography or magnetic resonance with delayed post-contrast enhancement, and absence of adenopathy. In the clinical context of renal transplantation, these may pose diagnostic dilemmas due to the greatly increased risk for infections as well as epithelial and non-epithelial tumors in this population, all of which may present as renal lesions.

CASE REPORT

This 65-year-old obese, hypertensive, diabetic man with end stage renal disease (ESRD) received a deceased donor renal transplant two years after initiating hemodialysis. The transplanted kidney was inserted into the right lower quadrant with a neoureterocystostomy without complication. During the first postoperative week, the patient presented with atrial fibrillation, multiple bouts of vomiting and abdominal distension. Serum amylase and lipase studies performed on three separate occasions during that period were not elevated (Amylase 66 U/L, 77 U/L and 79 U/L; Lipase 28 U/L, 34 U/L, 31 U/L). After medical management, imaging studies of the thoracic and abdominal cavity were performed. Computed Tomography (CT) of the abdomen (Figure 1) demonstrated a slightly dilated small bowel loop with air level and atrophic kidneys with a partially calcified

exophytic 1.5 cm mass on the left native kidney. The transplanted kidney and rest of the organs, including the pancreas were unremarkable. The differential diagnosis for the renal mass included a complex cystic and solid mass and further work up was recommended because a malignancy could not be excluded. The patient improved and was discharged on azathioprine, tacrolimus, epoietin, prednisone, acyclovir and trimethoprim-sulfamethoxazole with a creatinine of 1.9 mg/dl. 56 days post-transplant he was admitted for excision of the native kidney mass. Left native nephrectomy was performed without complications.

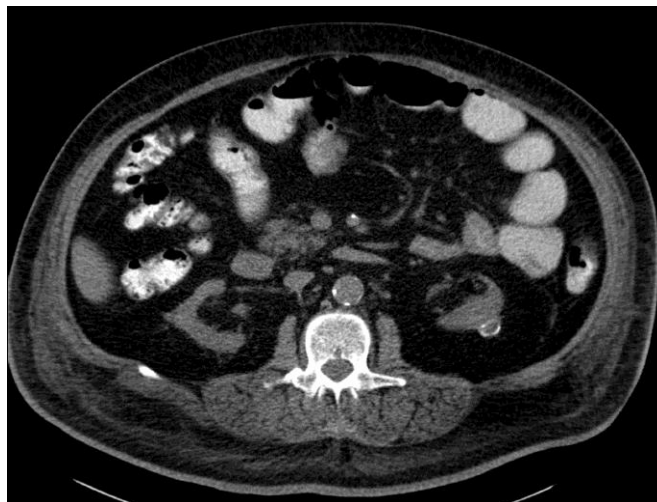


Figure 1. Computed Tomography (CT) of the abdomen demonstrated a slightly dilated small bowel loop with air level and atrophic kidneys with a partially calcified exophytic 1.5 cm mass on the left native kidney.

Received 12/17/2011; Revised 2/21/2012; Accepted 2/27/2012

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The 369 gm kidney had a puckered and irregular posterior surface with a well-circumscribed 2 x 1.5 x 1 cm white firm nodule (**Figure 2**). The nodule was filled with yellow pasty material surrounded by fibrosis. The rest of the kidney parenchyma was atrophic with two smooth cysts. Microscopic examination revealed perirenal fat necrosis surrounded by calcification and fibrosis of the capsule (**Figure 3** and **Figure 4**). The renal parenchyma showed atrophic changes and marked global and segmental glomerulosclerosis with thyroloidization of the parenchyma, but was negative for malignancy.



Figure 2. The 369 gm kidney image where a puckered and irregular posterior surface with a well-circumscribed 2 x 1.5 x 1 cm white firm nodule was shown.

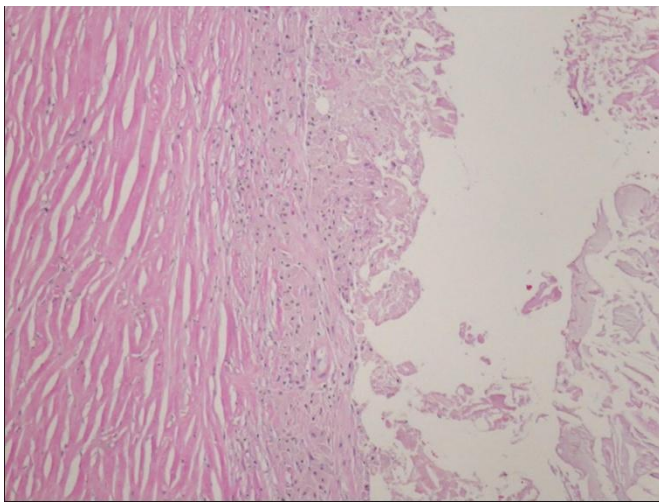


Figure 3. Perirenal fat necrosis and fibrosis of the capsule.

DISCUSSION

Tumors of the urinary tract are among the four most common malignant neoplasms in transplant recipients. Transplanted patients have a 15 times greater frequency of renal tumors

than the general population, felt in part to be related to immunosuppression and long term dialysis. Usually the tumor is diagnosed by ultrasound and is treated with partial or total nephrectomy.^{1,2}

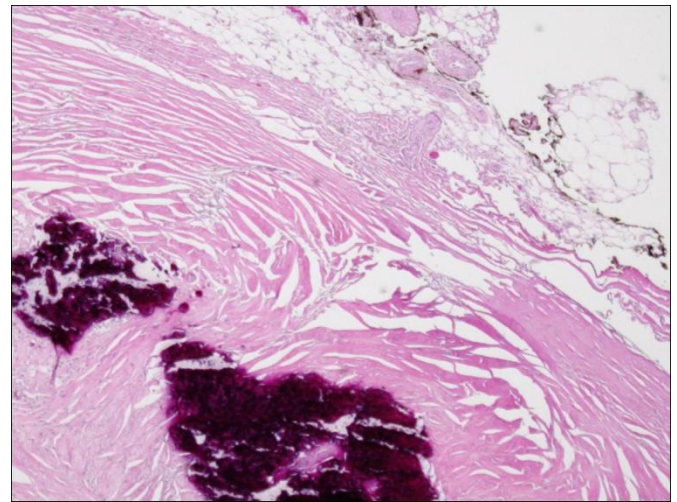


Figure 4. Calcification and fibrosis of the capsule.

In general, radiologic studies can differentiate surgical from non-surgical renal masses.³ Enhancing, solid renal masses are felt to represent neoplasm. However, lymphoma, angiomyolipoma, metastatic disease, renal anomalies and other pseudotumors can mimic renal cell carcinoma radiographically.

Renal pseudotumors are uncommon entities that may simulate renal neoplasms and therefore lead to unnecessary surgical procedures. Bhatt proposed a classification of renal pseudotumors into five types: developmental, infectious, granulomatous, vascular and miscellaneous.³ The developmental type includes prominent renal columns of Bertin, renal dysmorphism, dromedary humps, splenorenal fusion, cross-fused renal ectopia and persistent fetal lobulation. Infectious pseudotumors include focal pyelonephritis, renal abscess or scarred kidney. Granulomatous pseudotumors include xanthogranulomatous pyelonephritis, sarcoidosis, malakoplakia and tuberculosis (which can also be classified as infectious). Vascular pseudotumors include extramedullary hematopoiesis, arteriovenous malformation, renal pelvic hematomas and anticoagulant-induced subcapsular hemorrhage. Among miscellaneous, regenerating nodule after reflux has been reported as renal pseudotumor and also we would include perirenal fat necrosis in this category. Doppler, CT and MRI characteristics have been described in an attempt to correctly diagnose renal pseudotumors.³⁻⁶

Pancreatitis and steroids have been associated with pseudorenal tumor due to fat necrosis. In 2001 Pedrosa *et al.* reported the first renal pseudotumor due to fat necrosis associated with pancreatitis.⁷ Fat necrosis of the abdominal adipose tissue is a known complication of severe acute pancreatitis. It is hypothesized that lipase can be release into

the lymphatic or vascular system during acute episode of pancreatitis causing peripheral or metastatic fat necrosis. The most common locations of fat necrosis in the presence of pancreatitis are the peripancreatic adipose tissue, the omentum and mesentery, but a predilection for the retroperitoneum has been reported with greater involvement of left kidney, especially the posterior pararenal space without penetration of the peritoneal space or fat; explaining cases reported as mimickers of renal cell carcinoma and liposarcoma.^{8,9} Since Pedrosa's original report there has been one case of perirenal fat necrosis secondary to alcoholic pancreatitis reported by Fumado Ciutat et al.¹⁰ Persistence of radiologic findings for more than one year following the acute episode prompted surgical resection.

In the current case the exact etiology of the fat necrosis is unclear. The episode of nausea and vomiting suggested pancreatitis but this was not confirmed serologically (three separate amylase and lipase measurements were normal). The possibility of more remote pancreatitis episode remains in the differential diagnosis as a cause for this mass. A radiographic finding of chronic pancreatitis would have supported that finding but was not noted.

Retroperitoneal fat necrosis can also be caused by long-term steroid use due to alteration of lipid metabolism.⁹ Given the short duration of steroids we doubt that this is the etiology of the lesion. Perirenal fat necrosis may also be associated with underlying renal cell carcinoma.¹⁰ Extensive analysis of the current case failed to reveal an underlying renal cell carcinoma. Therefore, we consider the etiology of the fat necrosis in the current case to be unknown but perhaps related to a more remote bout of pancreatitis without development of chronic pancreatitis.

To our knowledge this is the first case of fat necrosis mimicking renal cell carcinoma in the native kidney of a

transplanted patient. Clinicians should be aware of this entity in order to avoid native nephrectomy and its attendant morbidity, and also because prolonged steroid use, which many transplant patients incur, may lead to the development of this lesion. Pathologists should also be aware of the rarely noted co-occurrence of perirenal fat necrosis with an underlying renal cell carcinoma.

CONFLICT OF INTEREST

None.

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