

Clinical Report

Allograft dysfunction in a patient with an odd-looking kidney: case of renal lipomatosis and review of literature

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Abstract

Renal lipomatosis was diagnosed in a kidney transplant recipient who presented with acute kidney injury (AKI) several years after transplantation. The patient had an odd-looking kidney transplant on ultrasound and computed tomography (CT) scan, showing a medullary mass with resultant compression of the surrounding renal parenchyma. A biopsy of the renal medulla confirmed fatty infiltration of the renal parenchyma. The patient underwent percutaneous nephrostomy and AKI resolved with relief of the obstruction. Renal lipomatosis is a rare condition that should be differentiated from other neoplasms of the kidney. When it occurs in a functioning transplant kidney, the treatment approach proves to be very challenging.

Keywords: kidney transplant; lipomatosis

Case report

A 66-year-old African-American female with a history of end-stage renal disease secondary to diabetes and hypertension underwent a cadaveric kidney transplant. The post-operative course was unremarkable with excellent graft function and serum creatinine ranging from 1.4 to 1.7 mg/dL for many years. Immunosuppression was maintained with tacrolimus, mycophenolate mofetil and prednisone. There were no episodes of rejection or recurrent urinary tract infection. Seven years later, serum creatinine was noted to increase to as high as 3.6 mg/dL over a few days from presentation. There were neither specific complaints nor any localizing physical examination findings. A kidney ultrasound showed a hyperechoic solid mass in the renal medulla, with central compression and resultant hydronephrosis. Follow-up imaging with computed tomography (CT) of the abdomen showed an 11 × 10 × 8.5 cm fatty mass at the center of the transplant kidney, causing compression of the central renal pelvis and dilatation of peripheral calyces (Figure 1). Mycophenolate mofetil was held for initial suspicion of malignancy and was then restarted once malignancy was ruled out after the medullary mass biopsy.

Due to the obstruction and renal deterioration, a percutaneous nephrostomy was placed with subsequent nephrostogram showing dilatation and distortion of the renal allograft collecting system. After decompression, the patient had an improvement in renal function with serum creatinine decreasing to 1.6 mg/dL (Figure 2).

A biopsy of both the renal cortex and medullary mass was performed. Renal cortex biopsy showed low-range borderline acute tubulointerstitial rejection with mononuclear infiltrates, extensive arteriolar hyalinosis and moderate interstitial fibrosis and tubular atrophy. There was no evidence of polyoma virus nephropathy. A biopsy of the renal medullary mass revealed mature adipocytes with no evidence of hyperchromasia and increased vascularity (Figure 3). A dose of methylprednisolone was administered intravenously followed by an oral steroid taper for the acute rejection changes seen in the biopsy. Mycophenolate mofetil was restarted once malignancy was ruled out.

Discussion

Renal sinus lipomatosis refers to a moderate increase in sinus fat. In some cases, the whole renal parenchyma may be replaced by fat and is known as renal replacement lipomatosis [1]. Renal replacement lipomatosis is a rare condition, characterized by fatty proliferation of the renal sinus with atrophy of the renal tissue. The extent of renal involvement is variable, with renal sinus lipomatosis being the mildest form and renal replacement lipomatosis being the most severe [2]. Renal lipomatosis may mimic and may be confused with neoplasm of the kidney. Some differential diagnoses include liposarcoma, lipoma, angiomyolipoma and transitional cell carcinoma of the renal sinus [3]. In contrast to renal lipomatosis, these tumors are usually located intrarenally or extrarenally.

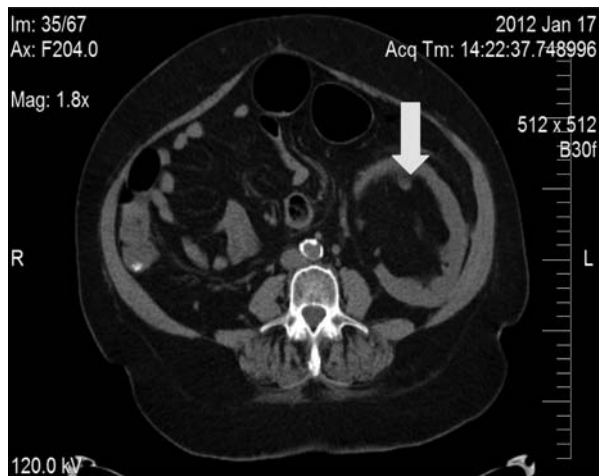


Fig. 1. CT of the abdomen showing a fatty mass (arrow) at the center of the transplant kidney.

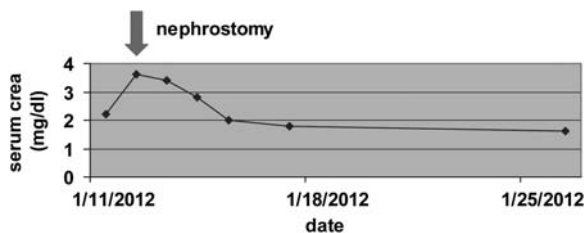


Fig. 2. Trend of patient's serum creatinine level over time.

ally outside the renal sinus [4]. Patients with liposarcoma, lipoma and angiomyolipoma also tend to have preserved renal function [5].

The formation of renal lipomatosis may be due to chronic inflammation, ~70% of which are associated with a calculus [6]. Renal calculi have been found to cause obstruction of the renal collecting system, which subsequently results in hydronephrosis, chronic infection and eventual atrophy of the renal parenchyma. This is often followed by replacement of the destroyed renal parenchyma by fatty tissue [1]. The development of renal lipomatosis has also been attributed to long-term steroid administration, chronic leakage of urine in the perinephric tissue, renal tuberculosis [1] and renal infarction. Other possible risk factors include aging, obesity, atherosclerosis [7] and chronic urinary tract infection [1]. Lipomatosis in the transplant kidney had been reported within 6 months to 8 years of transplantation [8]. In the case of our patient, her renal lipomatosis was detected 7 years after transplantation.

The most common investigational tools used to detect renal lipomatosis include a kidney ultrasound and a CT scan [1], both of which were done on our patient. Ultrasound often shows a hypoechoic rim of residual renal parenchyma with a hyperechoic mass at the center, representing the lipomatous tissue. The CT scan demonstrates the destroyed renal parenchyma and the lipomatous mass with a negative attenuation similar to normal adipose tissues [4].

When present in a native kidney that is already deemed non-functional, nephrectomy is often the adequate treatment [1]. This principle, however, may not necessarily

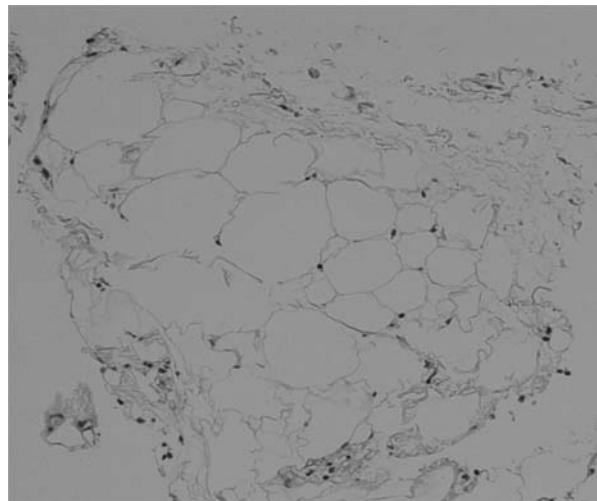


Fig. 3. Biopsy of the renal medullary mass showing mature adipocytes.

apply to a functional transplant kidney. In the case of our patient, since there was note of compression of the surrounding renal tissue with concomitant dilatation of the renal pelvis, nephrostomy tube placement was deemed to be necessary. In fact, the patient's serum creatinine level gradually decreased after nephrostomy tube placement relieved the urinary obstruction. The presence of a mass infiltration in the patient's renal medulla compelled us to obtain a tissue biopsy in order to rule out malignancy. In this case, pathology showed mature adipocytes with no malignant features.

Incidentally, a biopsy of the patient's renal cortex revealed low-range borderline rejection, which we addressed with steroid therapy. This may be related to holding of mycophenolate mofetil during the period when malignancy was not ruled out yet.

Although percutaneous nephrostomy placement is not an ideal solution to relieve the obstruction of the renal collective system, a more permanent solution remains elusive. Residual renal function in a 7-year-old allograft is an important consideration. Impact of lipomatosis on the renal cortex and the long-term prognosis in other situations suggest a dismal prognosis. Utilizing the native ureter to bypass obstruction can be a possibility if it is not involved in the pathology. In this case, however, extensive intrarenal involvement effectively precludes surgical excision of the lipomatosis as well as distal relief of obstruction with an indwelling stent, thereby leaving proximal diversion with a nephrostomy tube as the only option.

In summary, renal lipomatosis in a kidney transplant is a very rare entity with limited or no treatment options. The exact etiology is unclear, and it remains a consideration whether the type of immunosuppression can contribute or reverse the formation. Early diagnosis and relieving calyceal obstruction can improve renal function and hopefully prevent progressive cortical atrophy.

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Conflict of interest statement. None declared.

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