

De Novo Renal Cell Carcinoma in a Kidney Allograft Treated with Percutaneous Radiofrequency Ablation: A Case Report

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Keywords: *De novo*; Kidney allograft; Radiofrequency ablation; Renal cell carcinoma

Introduction

Renal cell carcinoma (RCC) represents a heterogeneous group of renal tumors. The incident rate has been increasing at 2-4% per year, such that RCC is now the 7th leading cancer type detected in males in the United States [1]. RCC accounts for 2-3% of all cancers in the general population compared to 5-7% of cancers diagnosed in kidney transplant recipients [2] However, the actual incidence of RCC in kidney transplant patients is low (0.5-1.6%), and the development of *de novo* tumor in the allograft is uncommon (0.1-0.3%) [3-7] Allograft tumors can either develop as a consequence of transmission of occult malignancy from the donor kidney or *de novo*; the former usually occurs early while the latter late following transplantation. We report herein a case of probable *de novo* RCC diagnosed in a renal allograft 58 months following kidney transplantation that was managed successfully with radiofrequency ablation.

Case Report

A 70 year-old African American male with a history of end stage renal disease secondary to polycystic kidney disease received an expanded criteria deceased donor kidney transplant on 8/26/08. He was on hemodialysis for 10 years prior to transplantation. The kidney transplant was performed using standard techniques including end-to-side vascular anastomoses between the renal vessels and recipient's right external iliac vessels followed by an extra-vesicle ureteroneocystostomy over a stent. Cold ischemia time was 26.5 hours. The patient received a single intra-operative dose of alemtuzumab induction (30 mg) in combination with post-operative tacrolimus, mycophenolate, and steroids. He required cystoscopy for persistent hematuria and clot retention with subsequent fulguration of a bleeding vessel in the bladder on post-operative day #24. He also experienced delayed graft function and remained dialysis-dependent until 10/2/08.

An ultrasound-guided percutaneous renal allograft biopsy performed one month following transplantation showed acute tubular necrosis, donor-transmitted glomerulomegaly and early chronic transplant arteriopathy but no evidence for acute rejection. Following a period of delayed recovery of renal allograft function, his renal function stabilized with a serum creatinine level in the 2.5-3.0 mg/dl range. A kidney transplant duplex ultrasound examination performed in July, 2011 (nearly 3 years following transplantation) during a hospitalization for acute kidney injury secondary to infection showed a small lower pole arteriovenous fistula in the kidney but no other vascular or parenchymal abnormalities. Because of recurrent infections, mycophenolate was stopped and the patient was placed on dual immunosuppressive therapy with tacrolimus and prednisone.

In June, 2013 (58 months following transplantation), the patient presented to his primary nephrologist with complaints of pain over the right lower quadrant transplant site for about 2 or 3 weeks. He denied fevers, chills, constitutional symptoms and any other signs or symptoms of either infection or acute rejection. He reported compliance with his immunosuppressive regimen, which included tacrolimus and prednisone. Physical exam revealed tenderness over

the allograft site and a bruit at the same location. Laboratory studies demonstrated stable renal allograft function and a normal white blood cell count. Subsequent kidney transplant ultrasonography showed a 2x1.9x1.7 cm hypoechoic mass in the upper pole of the renal allograft. He next underwent Magnetic Resonance Imaging (MRI) to better characterize the mass, which showed a round 18 mm lesion in the upper pole of the transplant kidney, with evidence for associated peri-lesional hemorrhage (Figure 1). The differential diagnosis at this point included RCC or a lipid poor angiomyolipoma.

He subsequently underwent ultrasound-guided fine needle aspiration of the mass. Cytopathology showed a group of polygonal cells with round nuclei, slight anisonucleosis, occasional conspicuous nucleoli and abundant eosinophilic cytoplasm, suggestive of RCC (Figure 2). Immunohistochemistry showed positive staining for vimentin and CYK 7, and negative staining for c-kit. This staining pattern, along with the cytology, was interpreted as being representative of RCC with clear and granular features.

He was determined to be a poor surgical candidate for partial nephrectomy because of advanced age and multiple co-morbidities including altered mental status, deconditioning with limited mobility, hypertension, history of varicella encephalitis with respiratory failure requiring tracheostomy and history of prostate cancer. Consequently, he underwent a technically successful percutaneous computerized tomographic (CT)-guided Radio Frequency Ablation (RFA) of the



Figure 1: Magnetic resonance imaging (MRI) examination showing a round 18 mm lesion in the upper pole of the transplant kidney, with evidence for associated hemorrhage.

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Received October 17, 2014; Accepted October 28, 2014; Published October 30, 2014

Citation: Shraideh YA, Farney AC, Rogers J, Orlando G, Farooq U, et al. (2014) *De Novo* Renal Cell Carcinoma in a Kidney Allograft Treated with Percutaneous Radiofrequency Ablation: A Case Report. J Clin Case Rep 4: 410. doi:10.4172/2165-7920.1000410

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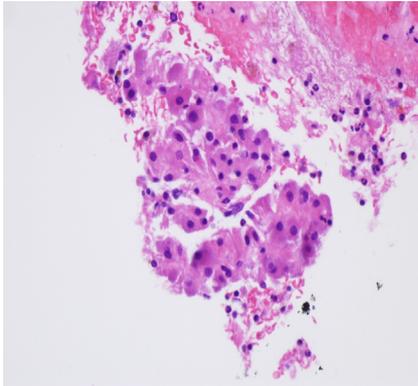


Figure 2: Fine needle aspiration biopsy with subsequent cytopathology demonstrating a group of polygonal cells with round nuclei, slight anisonucleosis, occasional conspicuous nucleoli and abundant eosinophilic cytoplasm, suggestive of renal cell carcinoma.

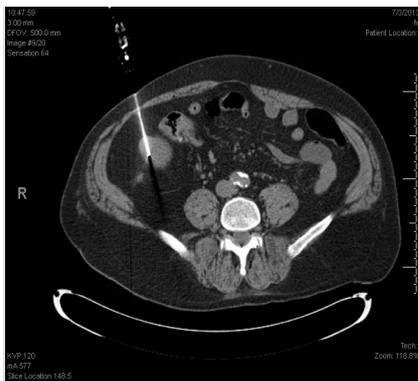


Figure 3: Technically successful percutaneous computerized tomographic (CT)-guided radiofrequency ablation (RFA) of the lesion approximately 2 months after presentation.

lesion approximately 2 months after presentation (Figure 3). Two months post-RFA, a repeat MRI showed post-procedural changes of the primary lesion and no new focal parenchymal masses. His serum creatinine level at the time of RFA was 2.8 mg/dl and subsequently stabilized in the 3.0-3.5 mg/dl range. The patient did well for approximately 7 months following RFA, at which time he was transferred to hospice care because of moderate dementia and generalized debility unrelated to his kidney transplant. He subsequently died with a functioning allograft and without evidence of recurrent RCC.

Discussion

Because of the requisite immunosuppression following transplantation, recipients of renal allografts have a 3-5 times higher risk of developing cancer than the general population [2]. The most frequent cancers that develop post-transplant are skin cancers (both malignant melanoma and non-melanoma skin cancers) and non-Hodgkin's lymphoma [2]. Although there has not been an overall increase in risk in transplant recipients of the most common cancers seen in the general population (including carcinomas of the lung, breast, prostate, and colon), kidney transplant recipients have a 5-fold higher risk of developing RCC compared to the general population [2-7].

De novo RCCs are found in 1-5% of renal transplant recipients; however, most (90%) are present in the native kidneys. Very few renal cancers are diagnosed in the kidney allograft, with an incidence of <0.5% post-transplant [2-7]. The average length of time from transplantation to tumor presentation is >5 years, suggesting that many of these are *de novo* tumors rather than tumors derived from the donor [2-7]. The estimated growth of *de novo* allograft tumors is between 0.5 and 1 cm per year, similar to RCC behavior in non-transplant patients [8] Typically, these cancers have been detected incidentally, are usually small and confined to the allograft, are low grade, and 50+% have clear cell histology [3-10].

There are multiple treatment options for the treatment of RCC including total nephrectomy, partial nephrectomy, and RFA [3-10]. Partial nephrectomy (or nephron sparing surgery) can keep patients off dialysis; however, clear surgical margins must be obtained to prevent recurrence [3-7]. These options are invasive, however, and may not be appropriate for patients who are considered to be at high or prohibitive surgical and anesthetic risk. In these instances, percutaneous RFA is another effective alternative [7-10]. This option is very precise, allows preservation of the renal parenchyma, and maintains GFR levels [8]. Short term outcomes following RFA are good, and it is suggested that these patients be followed with serial CT or MRI scans every 6 to 12 months [7].

In summary, we report herein an unusual case of *de novo* RCC developing in a renal allograft that was treated successfully with RFA. Because this is a rare occurrence, it is difficult to determine the best therapies for individuals in these situations. Therefore, it is important to continue to record these events so that eventual evidence-based guidelines may be developed. In our patient, the RFA method was chosen because of his advanced age, overall low functional status and the localized nature of the primary lesion. This treatment option should be reserved for similar cases in the future until more data are available that demonstrate the long-term safety and efficacy of this approach in chronically immunosuppressed transplant recipients.

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