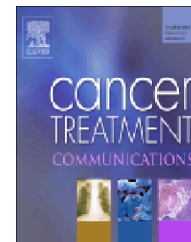




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De novo undifferentiated pleomorphic sarcoma arising from a renal allograft: A case report



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KEYWORDS

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Abstract

To the best of our knowledge, this is the first report of an undifferentiated pleomorphic sarcoma arising from a renal graft. Transplantectomy was performed in a 47-year old woman presenting to the emergency room because of general weakness. Preoperative workup revealed a 5.5 cm malignant mass of the graft which was not present on routine ultrasound performed 12 months earlier. Following transplantectomy, local recurrence developed despite complete tumor resection and interruption of immunosuppression. Despite radiation therapy, the outcome was ultimately fatal. Genetic analysis revealed that the tumor had arisen from donor tissue. Annual ultrasound surveillance might not be enough effective to screen for these rare high grade neoplasms.

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Abbreviations: 18-FDG PET, 18-fluodeoxyglucose positron emission tomography; 99-Tc-HDP, Technetium 99 m-hydroxymethylene diphosphonate; CT, computed tomography; FNLCC, Fédération Nationale des Centres de Lutte Contre le Cancer; LOH, loss of heterozygosity; MRI, magnetic resonance imaging; R0 resection, complete resection with no microscopic residual tumor (margins are microscopically negative according to the pathologist); RCC, renal clear cell carcinoma; RTR, renal transplant recipient; STR, short tandem repeat

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1. Introduction

Renal transplant recipients (RTR) are at an increased risk of developing renal cancer [1,2]. *De novo* renal neoplasms develop after renal transplantation and they arise either from a native kidney or from the graft. This latter situation is much less frequent [1]. Less than 100 cases have been reported. Roupret evaluated a renal clear cell cancer (RCC) cumulative incidence of 0.24% among RTR population [3]. Most of these malignancies are RCC but papillary types 1 and 2, tubulopapillary, chromophobic, sarcomatoid, epidermoid and urothelial carcinomas and oncocytomas have been described [4-8].

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Kidney graft tumors represent a particular therapeutic dilemma. On the one hand, radical treatment requires allograft nephrectomy and return to dialysis. On the other hand, interruption of immunosuppression may conceptually favor the host's immune response. Current guidelines recommend that any solid renal tumor should be treated with partial or radical nephrectomy [9]. Percutaneous radiofrequency and cryoablation represent alternative treatments in specific cases [10-12].

Many uncertainties on allogenic tumors remain due to their rarity. In this context, we report the case of an undifferentiated pleomorphic sarcoma arising from a renal transplant. To the best of our knowledge, no such a case has been reported up to now.

2. Material and methods (molecular identification of the tumor's origin)

Extraction of DNA was performed from paraffin blocks by the phenol-chloroform technique. DNA extracts were quantified using a Nanodrop spectrophotometer ND-100. 0.5 ng of extracted DNA was amplified on a GeneAmp[®] 9700 PCR thermal cycler (Applied Biosystems), using the AmpF/STR[®] SMG Plus[®] PCR amplification kit (Applied Biosystems), according to the manufacturer's instructions. Amplified products were separated on a 3130XL Genetic Analyser (Applied Biosystems) and analysis of DNA profiles was undertaken using GeneMapper TM ID version 3.2 (Applied Biosystems).

3. Case report

A 43-year-old woman underwent deceased renal transplantation for terminal renal failure secondary to polycystic kidneys disease. She was placed on tacrolimus and mycophenolate and her evolution was unremarkable during 4 years. Then, she showed up at the Emergency Department of our institution with general weakness. Physical examination was normal. Laboratory studies revealed acute kidney failure. A non-enhanced abdominal CT-scan showed a suspicious 5.5 cm mass of the inferior pole of the grafted kidney compressing the excretory system and two enlarged right external iliac lymph nodes. Abdominal MRI confirmed a tissular heterogeneous mass responsible for hydronephrosis. Chest X-ray and 99 m-Tc-HDP bone scintigraphy were negative. The patient underwent ureteropyelography with double-J stenting and biopsies which revealed a malignant undifferentiated cancer with a distinct immunomarker pattern (Table 1). She underwent transplantectomy, iliac and ilio-obturator lymphadenectomy and segmental iliac artery resection. A 2-cm tumor thrombus was extracted from the iliac vein.

Gross findings of the surgical specimen consist of a whitish heterogeneous mass $5.5 \times 5.5 \times 5 \text{ cm}^3$, studded with hemorrhagic areas, with a capsular effraction and perirenal adipose tissue infiltration. The tumor compresses the pyelocaliceal system and the proximal ureter. Large sampling of the tumor showed a diffuse highly cellular proliferation of pleomorphic and fusiform cells arranged often patternless. Mitosis were abundant (43 mitosis/10 high power fields; Mib1 was approximately 60%). Confluent necrosis was observed. Peritumoral parenchyma was atrophic. Immunohistochemical

Table 1 Immunomarker pattern.

Marker	Biopsy	Specimen
Vimentin	+	+
NSE	n.a.	+
CD99	+	+
VS38C	+	n.a.
t22q12	-	n.a.
EMA	-	-
Total keratin	-	-
CK 7	-	n.a.
CK 20	-	n.a.
CK 903	-	n.a.
Uroplakin	-	n.a.
LCA	-	n.a.
CD 3	-	n.a.
CD 20	-	n.a.
CD 34	n.a.	-
CD 56	-	n.a.
CD 57	n.a.	-
CD 128	-	n.a.
S-100	-	-
Melan A	-	n.a.
HMB 45	-	n.a.
AML	n.a.	-
Desmin	n.a.	-

analysis shows diffusely or focally positive stainings for vimentin, NSE, and CD99. Epithelial (keratin, EMA), muscular (desmin, AML) and vascular (CD34) markers were all negative, as CD 57 and S-100 (see Table 1). Four iliac lymph nodes were invaded by the tumor. It could be explained by the local seeding of tumoral cells. We considered the surgical specimen as an undifferentiated renal pleomorphic sarcoma, grade 3 (FNCLCC classification). The TNM stage was pT2 pN1 cM0 pV1 pR0. The transplantectomy specimen and histology slides are shown in Figure 1.

We used STR loci analysis to determine the tumor's origin (graft versus host). The tumoral tissue showed LOH at the D3S1358, D16S539 and vWA loci. Microsatellite instability was seen with a new allele 13 at the vWA locus (Figure 2). No STR instability was observed at the other 7 loci analyzed. As the tumor mirrored the genetic instability of the donor's DNA and not that of the receiver's DNA, we concluded that the cancer originated from the donor's kidney.

Four weeks following surgery, an 18-FDG thoraco-abdominal PET-CT showed an intense hypermetabolic signal infiltrating the right psoas muscle and the iliac vessels with suspect retroperitoneal lymph nodes suggesting local recurrence and lymphatic spread. Ten weeks postoperatively, the patient underwent a local radiation therapy (60 Gy in 30 fractions). One year postoperatively, PET-CT showed persistent retroperitoneal infiltration with a differential diagnosis of actinic alteration or tumor persistence but no adenomegaly or distant metastases. The patient died abroad 18 months after transplantectomy. Unfortunately, it was not possible to obtain further details about the medical cause of death and a possible dissemination of the disease.

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