Case Report

Primary adenocarcinoma of the renal transplant

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Key words: kidney transplant; renal cell carcinoma

Introduction

Primary tumours occurring in the renal graft itself have been reported only rarely [1–8]. We present a case of renal cell carcinoma (RCC) diagnosed by ultrasound, 7 years after renal transplantation.

Case

A 15-year-old girl presented with renal failure of unknown aetiology in 1989. After 9 months of haemodialysis, she was transplanted in January 1991. The graft donor was a 35-year-old woman who died of a cerebral glioma without known metastasis and not treated by ventriculoatrial shunting. Initial immunosuppressive treatment included prednisolone and azathioprine associated with antilymphocyte globulin for 20 days. Cyclosporine was then introduced for long-term therapy (5 mg/kg per day). Two episodes of acute rejection were successfully treated by steroid bolus. A large post-biopsy fistula needed to be treated by embolization.

For 6 years, her clinical status was good, but renal function then worsened progressively with an increase in creatinine serum from 2 to 4 mg/dl in 1997. The US examination of the graft performed yearly showed moderate parenchymal atrophy and several small cortical cysts.

In May 1997 US examination revealed a 2.2-cm ovoid mass in the superficial cortex of the renal graft (Figure 1). This mass appeared solid, with cystic areas and an hypoechoic peripheral rim. The superficial location of the mass allowed scanning with a high-frequency probe and high-resolution images of the tumor vasculature were obtained using Color and Power Doppler modes, with 3D reconstruction before and after administration of Levovist (Schering™, Berlin, Germany) (Figures 2A, B). Large, regular vessels were seen within the peripheral rim of the lesion, while smaller, irregular, branching vessels were seen in the centre. The Duplex study did not revealed any arteriovenous shunting.

The hypervascular nature of the mass was confirmed by an enhanced CT scan (Figure 3) and by T1-weighted MRI images after administration of gadolinium. On T2-weighted sequences, a hypointense rim, cystic areas within the lesion and small adjacent cortical cysts were seen (Figure 4). Angiography with selective catheterization showed a close correlation between the vascular architecture of the lesion and the 3D power reconstruction images (Figure 5). There was no adenopathy and no metastases were seen.

Because the vasculature of the lesion shown on Doppler images could correspond to a ‘spoke-wheel’ pattern and suggest an oncocytoma, partial nephrectomy was first discussed. However, as the renal function was poor, nephrectomy was performed.

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Fig. 4. T2-weighted MRI images demonstrates a low signal within the peripheral rim, cystic areas within the lesion and a small surrounding cortical cyst.

Fig. 5. A selective angiogram reveals a vascular distribution that correlates well with the 3D Power mode US reconstruction. The two coils are related to the large arteriovenous fistula embolization.

Macroscopically, a solid, well-marginated nodule was found in a subcapsular location. The tumour was composed of yellowish tissue with cystic and hemorrhagic areas. Microscopic findings were typical of a well-differentiated RCC of predominantly alveolar type, grade I according to the classification of Fuhrman. It was surrounded by a pseudocapsule containing malignant cells and dystrophic neovessels which connected with vessels arising from both the tumour and the surrounding parenchyma (Figure 6). The rest of the renal parenchyma showed a severe chronic allograft nephropathy.

Hybridization techniques were performed and showed that the tumour’s origin was the donor.

The patient’s status 3 months after surgery is good. The contralateral kidney and the liver from the donor were also transplanted. No tumour has developed in them.
Primary adrenocarcinoma of the renal transplant

Fig. 6. Pathologic examination (HE ×3.5). The tumour, of alveolar architecture contained many small vessels, and cystic and haemorrhagic areas. It is separated from the surrounding parenchyma by a pseudo capsule (arrowhead) corresponding to the peripheral rim seen on US and MRI. This pseudocapsule is a broad fibrous strip containing many large vessels that communicate with both the vessels of the tumour and those of the surrounding renal parenchyma.

Discussion

Renal cell carcinoma occurring in the transplanted kidney has been described only rarely. Seventeen tumours occurring in the renal allograft were reported by the Cincinnati Transplant Tumor Registry from 1968 to 1991, but cases diagnosed during the early era of transplantation were included [2]. To our knowledge, only six other cases of RCC and one case of undifferentiated carcinoma arising in renal transplants have been reported [3–8].

Such a tumour may already be in the kidney at the time of transplantation from either a cadaver or living related donors, as illustrated by cases reporting a mass discovered within 2 years after transplantation [4,5,8]. Careful pre-transplant imaging of organs should usually prevent such a transmission [2].

However, most malignancies in transplanted organs are thought to be de novo cancers. In the current case pathology excluded a late metastasis from the cerebral tumour of the donor and hybridization techniques indicated allograft origin. The diagnosis of a small mass 7 years after transplantation also suggests de novo allograft origin, as previously reported in other cases with long post-transplantation intervals, up to 19 years [3,5]. Nevertheless the overall growth rate of native kidney small RCC can be low, with a large range of 0–1.1 cm/year which suggests two types of RCC, one growing slowly and the other more rapidly [7]. The same findings have also been reported in the case of immunosuppressive treatment [1]. In a patient followed for 3 years before allograft nephrectomy, Heinz-Peer et al. [5] showed that the linear diametric growth rate was approximately 0.5 cm/year.

Early diagnosis of the tumour is essential because these tumours may have a high incidence of metastases [1], even when the mass is small [2]. The 17 cases reported from the Cincinnati Transplant Tumor Registry were diagnosed after transplant removal for rejection or technical problems. Of the seven cases published more recently, three have been diagnosed preoperatively by greyscale ultrasound [3,5,7], two by CT [6–8] and one by scintigraphy [4].

The current case emphasizes the important role of periodic follow-up by ultrasound for the early diagnosis of tumour. The superficial position of the transplanted kidney allows the use of high-frequency transducers, which improves the signal-to-noise ratio and the spatial resolution, and makes analysis of the tumour content more accurate. Doppler techniques including Power mode, 3D reconstruction and post-contrast study, clearly showed the vascularity of the lesion, with large peripheral vessels (within the pseudocapsule on pathology) and central tumour vessels. However, these findings were also described in small RCC with other imaging modalities [8] and illustrate the low specificity of the ‘spoke wheel’ pattern previously reported in oncocytoma. However, on greyscale US, as well as on subsequent CT and MRI, the mass had a solid component with a pseudocapsule and internal cystic changes which have also been described in small alveolar RCC in native kidneys [8]. The association of cortical cysts and worsening renal function is another feature of the current case, similar to ACD reported in native kidneys in patients with renal failure. The possible association of ACD and RCC has been previously suspected in patients receiving cyclosporine [1].

Differential diagnosis also includes lymphoproliferative disorders, which mainly arise within the hilum or pedicle of the graft, or poorly differentiated urothelial carcinoma [4].

The treatment of RCC in a transplanted kidney is usually transplant removal and discontinuation of immunosuppressive therapy which can result in complete regression, including cases with small pulmonary metastases [6]. Partial nephrectomy has also been performed in one case of RCC, without recurrence [2].

Acknowledgements. The authors thank Robert L Lebowitz for his advice and review of the manuscript.

References


Received for publication: 13.5.98
Accepted for publication: 20.5.98