Late rupture of the renal graft: not always graft rejection

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Introduction

The fate of failed renal grafts returning to dialysis programs is usually uneventful except for the patients who develop fever and graft tenderness not responsive to low dose steroids [1]. We describe a patient who required urgent nephrectomy after graft rupture which began during one of his periodic haemodialysis sessions.

Case Report

A 28-year-old male patient was diagnosed as having type II mesangiocapillary glomerulonephritis in March 1984. Nine months later he started periodic haemodialysis treatment after progressive chronic renal failure. In November 1986 he received a cadaveric renal allograft immediately functioning with good evolution. In November 1986 he received a cadaveric renal allo-
graft immediately functioning with good evolution. Immunosuppression was based on prednisone 1 mg/kg per day progressively tapered to 0.1 mg/kg per day during 1 year, and cyclosporine-A (CyA), 12 mg/kg p.o. daily adjusted to obtain whole blood levels of 150–250 ng/ml. Renal function was good (serum creatinine 1.1 mg/dl after 1 month). During the first year after renal transplantation microhaematuria and mild renal function deterioration without proteinuria was detected. Renal biopsy disclosed recurrent type II mesangiocapillary glomerulonephritis and vasculopathy induced by CyA. Progressive CyA dose reduction (to 3 mg/kg per day) and addition of azathioprine was prescribed [2]. After transient improvement of 12 months duration, again slow and progressive renal function deterioration was observed in subsequent years. The patient began periodic haemodialysis treatment on December 1996.

Forty days after returning to his haemodialysis program, he was receiving a scheduled haemodialysis session and required urgent admission with acute and important pain over the graft associated with a fall in haemoglobin concentration and haemodynamic instability. Graft ultrasonography showed subcapsular and intraparenchymatous haematoma. Some hours later, he underwent graft nephrectomy. During surgery, a 3–4 cm solid and ruptured mass on the upper pole of the graft was observed. Several similar nodules of 0.8–1 cm diameter and a number of small cysts were also discovered in the remainder of the graft (Figure 1). Histologic examination revealed a multicentric, low-grade renal cell tumour arising on an acquired renal cystic disease (Figure 2). Severe chronic rejection was also noted. Extension studies were entirely negative, including toracoabdominal computerized tomography and bone gammagraphy. The patient has completed six uneventful postoperative months and he is stable on haemodialysis.

Discussion

Solid-organ recipients are at an increased risk of malignant tumours. Excluding skin and cervix tumours, de novo renal cancer constitutes the most frequent type according to both the International Registry [3] and our own series [4]. The prevalence of these renal tumours varies between 0.6 and 1.7% of all recipients, a much higher percentage than that found in the general population [5]. Among the risk factors known to be related to the development of renal cell cancers in renal patients, acquired renal cystic disease is probably the most relevant [6,7]. Acquired renal cystic disease arises in 24–47% of dialysis patients and renal cell carcinomas in 6–10% of them [6,7]. It is debated whether a functioning renal transplant allows or promotes acquired renal cystic disease regression [8,9]. The possibility that renal transplantation reduces the incidence of renal cell tumours with respect to the incidence observed in dialysis is unknown. Probably, the protective effect that regression of acquired cystic renal disease might exert on cancer development is negatively bal-

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Fig. 1. Gross examination disclosed a $10 \times 6 \times 3$ cm kidney graft with two well delimited nodules, the biggest one with a diameter of 2.5 cm and placed close to the graft surface. Both nodules were friable and haemorrhagic.

Fig. 2. Microscopically the tumour cells were large, with clear cytoplasms and small and centrally located nuclei (H&E $\times 450$).

anced with the intense drug immunosuppression regimens that renal transplantation implies. Particularly long-term CyA treatment has been associated with an increased incidence of renal cancer, higher than that observed with other immunosuppressants. Among many distinctive features of renal cell carcinoma in this group, it is frequent to note that renal cysts are either absent or very small [7]. Our patient had not acquired renal cystic disease in native kidneys and showed acquired cystic renal disease with very small cysts within the graft. Prolonged immunosuppression associated with long-term dysfunctioning graft were then probably the most important factors for tumour development.

Graft rupture is a severe complication usually super-vened during the first weeks after renal transplantation [10]. It is frequently related to acute rejection, venous thrombosis and/or severe acute tubular necrosis and to our knowledge has not been described arising in the context of renal cell carcinoma of the graft. It is widely accepted that periodic screening of so-called high-risk dialysis patients should be performed mainly with
sonogram and/or CT scan [6,7,11]. Cost-efficacy of these screening programmes is poorly known. Even more questionable is the place for periodic imaging screening in failed renal grafts returning to dialysis. Our patient illustrates the possibility of renal tumours arising in a failed renal graft and suggests the need for close surveillance in case of this possibility. Acute rupture with haemorrhage unmasked the otherwise silent and dangerous neoplastic disease.

References


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