

Renal vein thrombosis in a renal allograft

Case Report

A 61-year-old woman with end-stage renal disease secondary to post-streptococcal glomerulonephritis received a live related transplant from her brother in 1980. For 23 years she had no medical or nephrological problems and had excellent transplant function with creatinine ranging from 60–80 $\mu\text{mol/litre}$. In 2004 a cystic lesion was noted on her right breast, biopsy of which revealed a high grade B cell non-Hodgkin's lymphoma.

Ten days before admission she noticed pain over her transplant kidney and over the ensuing days she reported passing progressively less urine. Initial blood tests were suggestive of pyelonephritis with a neutrophil leucocytosis (white cell count 19.1×10^9 cells/litre) and acute phase response (C-reactive protein 58 mg/litre). Her creatinine was 368 $\mu\text{mol/litre}$. An ultrasound scan of the transplant kidney demonstrated globally poor perfusion and the renal vein could not be demonstrated (Figure 2). This was in stark contrast to a scan performed 1 year earlier (Figure 1). A transplant nephrectomy was performed uneventfully that night and confirmed the diagnosis of renal vein thrombosis.

Discussion

Although renal venography is regarded as the gold standard for the diagnosis of renal vein thrombosis, duplex ultrasonography is now frequently used to make the diagnosis (Duckett et al, 1991). Renal vein thrombosis is a rare complication of acute renal transplantation but a common cause

of early graft loss. It has an incidence of approximately 1% and usually manifests 3–9 days post transplantation (Jones et al, 1988). However, it is an extremely rare cause of graft loss in chronic stable transplant patients.

Renal vein thrombosis takes a completely different course in native and

transplant kidneys. In allografts, which have no capsular venous anastomoses, there is no collateral venous drainage to maintain blood circulation or to enable spontaneous recanalization. The imbalance between arterial influx and the decreasing venous outlet leads to an elevation in vascular impedance, and finally the arterial supply of the graft is compromised. The only possibility of preserving the graft from necrosis is immediate (within an hour of thrombosis) surgical restoration of the venous outflow. All other cases should undergo emergency nephrectomy to minimize the chances of graft rupture and haemorrhage (Nerstrom et al, 1972).

The authors presume that the cause of the transplant renal vein thrombosis in this patient was an acquired thrombophilia from her underlying haematological malignancy. **BJHM**

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Figure 1. Ultrasound image demonstrating normal colour and spectral Doppler flow in the renal transplant vein.



Figure 2. Ultrasound image showing very little colour Doppler flow in the renal transplant and no spectral trace at the pedicle. These findings are consistent with a diagnosis of renal vein thrombosis in the transplanted kidney.

