

Nephroquiz for the Beginner
(Section Editor: M. G. Zeier)

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Exercise-induced anuria in a renal allograft recipient

Case

A 47-year-old renal allograft recipient presented 14 months post-transplant with anuria, of sudden onset. He had autosomal dominant polycystic kidney disease (ADPKD) and had been on hospital haemodialysis for 3 years prior to transplantation, when he received a live unrelated renal allograft from his partner. The graft was a 1,2,2 mismatch and his immunosuppression was tacrolimus (5 mg/day) led triple therapy with azathioprine (75 mg/day) and prednisolone. His immediate post-transplant function was good (Table 1), but at day 21 he had a deterioration in function and required a diagnostic renal transplant biopsy. Two samples were taken from the medial pole under real-time ultrasound guidance and histological examination revealed acute cellular rejection (Banff grade 1). He had no haematuria or drop in haemoglobin post-biopsy. The deterioration in renal function reversed after treatment with methyl prednisolone. Following this episode graft function remained stable, with a blood urea of 8.8 mmol/l and creatinine of 139 µmol/l and normal blood pressure on a single anti-hypertensive agent (betablocker) at routine review, 13 months post-transplant, just prior to his current presentation.

Fourteen months post-transplant he presented to the accident and emergency department of a district general hospital, while on a weekend away. He gave a history of not passing urine for 10 h and complained of a dull aching pain over his transplant kidney. He had been feeling fit and well, and had been lifting heavy 'flat pack furniture' as well as working-out intensively in a gym in the days preceding his presentation. He was urgently transferred to the transplant unit. There was no history of trauma and he had been compliant with immunosuppression, confirmed by trough tacrolimus level of 5 µg/l.

On presentation he was very anxious regarding his graft function. He was hypertensive at 150/110 mmHg and had no increase in temperature. His graft in the left iliac fossa was enlarged and tender, his plasma creatinine had risen to 311 µmol/l (Table 1) and his bladder was empty. Emergency investigations were undertaken.

Question

What is your diagnosis?

Table 1. Renal function, haematology, clotting screen and blood pressure immediately post-transplant

	Day 1	Day 21*	Day 26	13 months**	14 months
Urea mmol/l	9.2	9.9	11.1	8.8	13.9
Creatinine µmol/l	146	190	145	139	311
Haemoglobin g/l	127	122	129	170	161
WBC ($\times 10^9$)	11.8	5.4	12.5	8.6	11.2
Platelets ($\times 10^9$)	240	223	259	228	203
PT (ratio)		1.1			1.0
APTT ratio		0.9			1.1
Blood Pressure	120/75	130/80	120/80	116/80	150/110

*At day 21 with mild acute cellular rejection; ** post-treatment, at follow up at 13 months; and on presentation with anuria.

Answer to the quiz on the preceding page

Ultrasound with a Doppler examination and CT scan (Figure 1a, b and c) demonstrated a dense perinephric collection, suggestive of haematoma over the lateral aspect of the transplant kidney with displacement of the hilum anteriorly and medially. The vascular supply to the kidney was preserved but compressed. Given these findings, he was prepared for theatre and consented exploration of the graft and possible graft nephrectomy.

In theatre the transplant kidney was explored with evidence of fresh clots and fresh blood within the pseudo-capsule. Patchy ischaemia over the lower pole was seen. Clots were evacuated and the cavity was washed with saline. On leaving theatre he had already started to pass urine. Over the following few days his renal function gradually improved, no further clot or haematoma was seen on repeat ultrasound and his baseline creatinine returned to normal. He is now 32 months post-renal allograft and continues to have stable renal function with plasma creatinine of 122 $\mu\text{mol/l}$ and well controlled blood pressure (Table 2).

Discussion

This case demonstrates an acute bleed which occurred within the fibrous pseudocapsule and caused renal dysfunction in a renal allograft of 14 months, with full reversal of function on evacuation of the haematoma. This haematoma occurred in the absence of any definite history of injury. However, our patient had carried out heavy strenuous exercise in the days preceding the bleed. It seems likely that this resulted in the spontaneous pseudocapsular bleed. Renal allografts are superficially placed in the iliac fossa and all patients are cautioned regarding contact sports. Despite this, there are very few case reports describing non-biopsy related renal allograft injury, indeed any injury reported seems to follow blunt trauma to the anterior abdominal wall.

Spontaneous renal rupture was frequently described in the early years of transplantation, it is nearly always

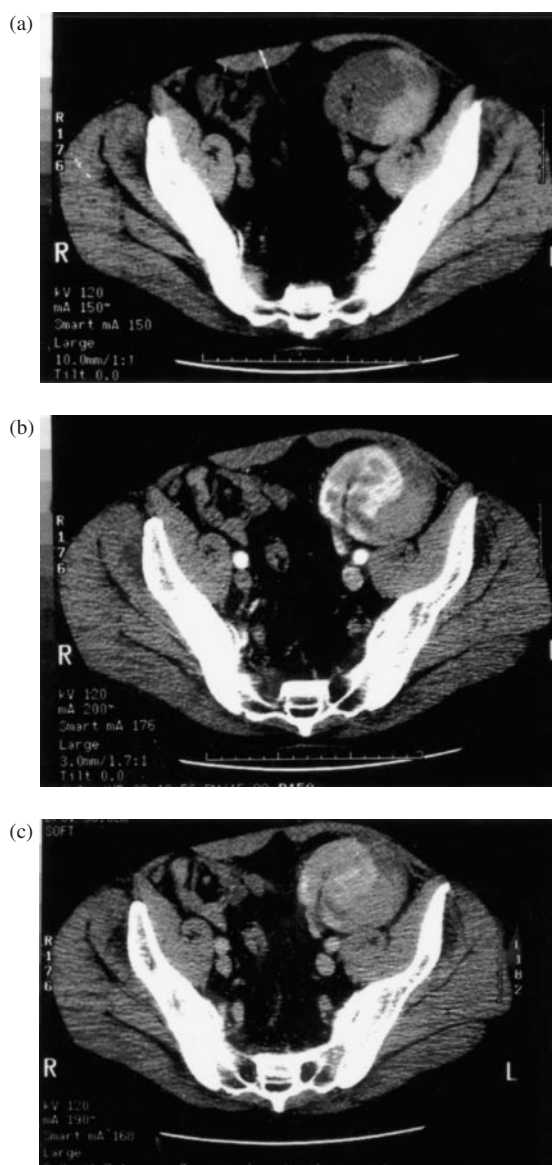


Fig. 1. The pre contrast CT image (a) in which the blood has higher attenuation (brighter) compared with the kidney. The second image (b) is post-contrast in the arterial phase. The renal cortex is enhanced and the haematoma appears relatively lower in attenuation (darker) than the renal parenchyma. In the parenchymal phase (c) there is still no contrast in the collecting system and the kidney continues to appear a little brighter.

Table 2. Renal function, haematology, clotting screen and blood pressure on presentation with anuria and recovery following evacuation of haematoma along with present day results, 32 months post-transplant

	14 months	Post-op Day 1	Post-op Day 2	Post-op Day 3	32 months
Urea mmol/l	13.9	14.0	11.3	9.3	6.7
Creatinine $\mu\text{mol/l}$	311	233	189	156	122
Haemoglobin g/l	161	148	131	133	156
WBC ($\times 10^9$)	11.2	11.3	10.7	10.7	6.8
Platelets ($\times 10^9$)	203	155	138	159	227
PT (ratio)	1.0				
APTT ratio	1.1				
Blood Pressure	150/110	150/90	140/90	130/90	125/85

associated with severe early rejection and is now rarely seen [1,2]. There is one report of a lymphocele occurring in an established renal allograft, following a squash ball induced blunt injury [3]. A further two case reports have described seat belt compression injuries presenting late with hypertension and a rise in creatinine. One of these cases documents a lower pole infarct [4] and the other resulted in a lymphocele of the transplant [5]. A case of anuria in a renal allograft following blunt trauma to the abdomen demonstrated an extensive subcapsular haematoma, with recovery of renal function following evacuation [6]. In 1939, Page described an experimental model of wrapping cellophane around the renal capsule resulting in a tight fibrous sheath around the kidney, leading to hypertension and impairment of function due to increases in intrarenal pressure [7]. This has been described in native kidneys following blunt trauma and is known as Page kidney [8].

In a renal allograft, there is a risk of spontaneous subcapsular bleeding which may be brought on by strenuous exercise. Renal allografts are particularly prone to this phenomenon due to the formation of a tight fibrous pseudocapsule which seems to occur universally following graft insertion, and seems to be a similar mechanism to the fibrous encasement of a foreign body. This case illustrates that in a patient with stable graft function who presents with oligo/anuria for which no obvious cause is apparent, the possibility of subcapsular haematoma should be considered with urgent evacuation of the capsule if renal function is to be restored.

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Gemma Browne¹

Paul Allan²

K. K. Madhavan³

Robin Winney¹

¹Department of Renal Medicine

²Department of Radiology

³Scottish Liver Transplant and

Renal Transplant Unit

Lothian University Hospitals NHS Trust

UK