condition had deteriorated. On examination at this stage his abdomen was tender with guarding. His CAPD fluid was tested using ‘dipstick’ testing and was found to be positive for bilirubin. The patient also had a chest X-ray, which showed air under the diaphragm. This can be normal in the CAPD population, but the chest X-ray was repeated after a temporary dialysis line was inserted and the air was no longer present. Due to the clinical picture and patient’s condition he was taken to theatre where a perforated duodenal ulcer was found. He had a protracted post-operative course with an ITU stay and multiple intra-abdominal abscesses, but has now recovered.

Normal methods for diagnosis including erect chest X-rays have been shown to be of limited use in these patients and are not a reliable indicator of visceral perforation [3]. One of the more common indicators of visceral perforation is multiple enteric organisms on culture of CAPD fluid. This in itself can take a few days, which is also delaying the patient’s treatment. We feel that the bedside testing of CAPD fluid for bilirubin may help with the diagnosis of visceral perforation. If we can shorten the time to surgery for these patients we may be able to make an impact on the mortality and morbidity.

Conflict of interest statement. None declared.

Manchester Institute of Helen Scarborough
Nephrology and Transplantation Sivanandam Shrikanth
Manchester Royal Infirmary Ram Gokal
Manchester, UK
Email: hscarborough@ntlworld.com


doi:10.1093/ndt/gfh765

Advance Access publication 15 March 2005

**Recurrent hydronephrosis causing acute uraemia in a renal transplant donor without the presence of stones or stricture**

Sir,

When the kidney donor has been thoroughly examined according to the usual standards [1], complications in the short and the long term are seldom seen. This, together with the favourable outcome for the recipient, justifies living donor transplantation.

However, a presumed complication could have been fatal to a 63-year-old woman who donated her right kidney to her brother in January 2003. In the following May and June, she had severe pain in the left flank and, when vomiting, general oedema and ultimately anuria occurred, she was admitted to the urological department, where the serum level of creatinine was found to be 841 μmol/l. Ultrasound showed hydronephrosis and, after nephrostomy, the serum creatinine dropped to 150 μmol/l. Antegrade pyelography revealed no stones or stricture and the catheter was removed. Unfortunately, the patient was readmitted with similar symptoms some weeks later. Nephrostomy was again performed and no abnormalities in the urinary tract were seen on the pyelogram. The woman had had no problems whatsoever from the urinary tract before donation, and hydronephrosis of the kind caused by a valvular effect over an aberrant artery passing the ureteropelvine junction was suspected. After a period with a JJ stent, she underwent a pyeloplasty operation in August, and 1 year after donation was well with a serum creatinine of 123 μmol/l.

Before donation, the patient reduced her weight from 102 to 79 kg to achieve a body mass index (BMI) of 31. Intravenous pyelography showed a small extrarenal pelvis on the left side, which expert radiologists judged to be completely normal, as was the renogram. Arteriography showed two arteries on the left side.

The 65-year-old recipient had no urological complications, and 1 year after transplantation his serum creatinine was 142 μmol/l.

This kidney donor, who developed acute uraemia 5 months after donation, was at first believed to have passed a stone unknowingly. After a second similar episode with no stone or abnormalities in the urinary tract, another less obvious explanation had to be sought. She was used to drinking about four litres a day; all urine had to pass through one kidney, and it was suspected that a slight hydronephrosis was kinked over the aberrant artery, permitting complete obstruction now that surplus water could not be eliminated by a second kidney [2].

1. The weight loss with a decrease in the surrounding fatty capsule may have resulted in less physical support for the kidney, thus adding to the problem.

In any case, although kidney donors have a life expectancy longer than that of the general population and should not, in our opinion, have limitations set on their lives [3], health personnel, including physicians in primary care, must be very thorough in their diagnostic work-ups, when a person with a single kidney presents with symptoms from that region. Obstruction might have caused serious damage to the remaining kidney within a relatively short time.

We do not think that this potential complication has a frequency that should lead to limitation of kidney donation. However, when the remaining kidney has multiple vessels and an extrarenal pelvis, the risk of ureteropelvic junction obstruction may be slightly increased [4,5].

Conflict of interest statement. None declared.

1Department of Nephrology
Odense University Hospital
DK-5000 Odense C

2Department of Urology
Skejby Hospital
DK-8200 Aarhus N
Denmark
Email: bjesper@dadlnet.dk

Aggressive renal cell carcinoma in a 27-year-old kidney transplant

Sir,
The late development of a primary tumour in an allograft kidney is a rare event: the longest reported interval period, to our knowledge, is 21 years. In the case described below, we present the finding of an aggressive renal cell carcinoma in a transplanted kidney after 27 years.

A 29-year-old female underwent cadaveric renal transplantation in April 1976 for renal failure secondary to chronic reflux nephropathy. The donor was a ventilated 15-year-old male who sustained a subarachnoid haemorrhage following trauma. The graft displayed excellent function on low-dose immunosuppressants for 27 years. Late follow-up consisted of clinic review only: routine ultrasound screening was not performed. However, during investigation for anaemia and abnormal liver function tests in September 2003, an abdominal ultrasound detected the presence of a 5 × 6 cm lesion in the upper pole of the transplanted kidney. A transcutaneous renal biopsy revealed a high-grade primary renal cell carcinoma and a transplant nephrectomy was subsequently performed. At operation, a large multicentric tumour was seen to invade through the renal capsule; no lymphadenopathy was present. Macroscopic pathological examination showed multiple yellow/cream nodules diffusely involving the whole kidney (Figure 1). Histology revealed the presence of a high-grade type II papillary renal cell carcinoma with areas of sarcomatoid change (Figure 2). The duration from renal transplantation to formal histological diagnosis was 330 months. Sadly, despite surgery, the patient rapidly developed multiple bony and subcutaneous metastases and died 5 months post-nephrectomy.

Fig. 1. Section through renal allograft showing multiple deposits of pale tumour.

Fig. 2. Micrograph showing the typical findings of high grade renal cell carcinoma. Other areas displayed a more conventional grade II papillary subtype, whereas others were representative of sarcomatoid change.