Intermittent Early Postoperative Oliguria in a Patient after Renal Allotransplantation

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Oliguria immediately after renal allotransplantation is usually due to acute tubular necrosis, rejection, thrombosis of the renal vessels, or obstruction of the transplanted ureter (Carpenter & Austen, 1968). However, 'intermittent' early postoperative oliguria has not been described except in a report by McMillan (1968) in 5 cadaveric transplants. These cases presented with polyuria immediately after haemodialysis and this was attributed to the heparin administration during dialysis.

The patient described here (Figure 1) EC, male, aged 25, with chronic glomerulonephritis (terminal stage) received a kidney transplant from his father on September 2nd 1970. The warm ischaemia was prolonged due to technical difficulties with the vessels, and the urine volume gradually increased from 650 ml to 1250 ml during the first six days. On the 7th day the urine volume declined to 200 ml/24h (as little as he was passing postoperatively) and on the 9th day haemodialysis was commenced. After a 17-day

![Diagram](image-url)

Figure 1. Course of the patient after transplantation. Vu = urine volume, Scr = serum creatinine. The arrows represent haemodialyses.
period of oliguria and a normal renal angiogram (Figure 2) the patient started to pass a lot of urine with intervals of oliguria. It is worthwhile mentioning that the patient passed large quantities of urine at each voiding, and then hardly passed any urine until the next 'polyuric' period. On the 36th day after operation another renal angiogram was performed which was normal. All the remaining parameters did not show any evidence of rejection including the examination of the urinary sediment (Papadimitriou et al., 1970). On the 40th day the patient was explored. The transplanted kidney was hydronephrotic, the ureter was dilated and contained a large quantity of urine. The lower end of the ureter was narrowed and contained a number of clots which acted as a valve (Figure 3). Histology of the ureteric segment showed infiltration with lymphocytes and polymorphs with some necrotic and haemorrhagic areas. A recipient nephrectomy was performed. The urine volume gradually
increased and the serum creatinine fell to normal levels. The glomerular filtration rate ten months after transplantation was 65 ml/min.

DISCUSSION

It is well known that any oliguric episode after renal transplantation causes a lot of anxiety. The danger of rejection is always in the mind of the transplant team and routine tests are used to diagnose it (Chisholm et al, 1969) and treat it early. The case described above posed considerable difficulties. There were no local signs of obstruction and no pyrexia. However, exploration of the kidney was scheduled twice and postponed because the patient passed large urine volumes the day before. The ureteric stenosis was probably due to rejection (Haber & Putong, 1965; Swinney, 1970). In our series (Papadimitriou et al, 1970) we had the opportunity to study histologically ureteric segments from patients who had ureteric leaks. In two cases out of six, where there was no evidence of inflammation, the ureteric leak was thought to be due to necrosis of the ureteric wall following rejection.

We think that the present case is interesting for two reasons: firstly, from the clinical point of view (because of oliguric episodes due to 'intermittent' obstruction of the ureter) and secondly from the histological point of view (because of possible isolated rejection of the transplanted ureter).

REFERENCES


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