Improved cardiac function after renal transplantation

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Summary: There are few reports of the outcome of renal transplantation in patients with severe left ventricular (LV) impairment. We describe three men with chronic disabling heart failure associated with LV dysfunction in whom a remarkable improvement in cardiac function followed renal transplantation. Transplantation may offer the prospect of successful rehabilitation in these circumstances. Undue pessimism as to the prognosis in such patients is unwarranted.

Introduction

Patients without renal failure who have impaired left ventricular (LV) function have a poor prognosis (Nelson et al., 1975; Johnson & Palacios, 1982). When end stage renal failure (ESRF) and poor ventricular function coexist, there may be reluctance to refer a patient for renal replacement owing to pessimism about eventual outcome. We describe three men with ESRF and severe chronic LV dysfunction in whom a remarkable improvement occurred following renal transplantation. Undue pessimism about such patients for whom renal transplantation is, in all probability, the treatment of choice, is unwarranted.

Case studies

Patient 1

A man born in 1922 commenced haemodialysis for chronic renal failure due to atrophic pyelonephritis in December 1971. He had an inferior myocardial infarct in September 1977. Subsequently he gave up working as a furrier because of angina and dyspnoea. He was transferred from home dialysis to hospital-based haemodialysis because of repeated episodes of hypotension, LV failure and paroxysmal atrial fibrillation. Following an anterior myocardial infarct in July 1978 he deteriorated further. Episodes of LV failure were more frequent and, despite small interdialysis weight gains, dialyses were complicated by severe hypotension. Mean haemoglobin (Hb) was 10.3 g/dl, pre-dialysis plasma urea 26 mmol/l, creatinine 1.15 mmol/l and bicarbonate 16 mmol/l.

Cadaveric kidney transplantation was carried out in July 1979. He still has occasional episodes of ischaemic cardiac pain when working in the garden. He has an artificial leg prosthesis following trauma and his exercise tolerance is limited to approximately 150–200 yards walking on the flat by both pain in the stump and dyspnoea. There have been no further episodes of LV failure or hypotension and indeed, blood pressure levels of the order 150–160 mm Hg systolic and 90–105 mm Hg diastolic have been recorded from 30 months after transplantation. Current medication is azathioprine 150 mg, prednisolone 7.5 mg and disopyramide 150 mg b.d. Creatinine clearance (CrCx) is 58 ml/min and Hb 15.7 g/dl.

Patient 2

A man born in 1949 had rheumatic fever in 1965. In 1977 he developed infective endocarditis complicated by severe aortic regurgitation and renal failure presumed due to associated glomerulonephritis. Aortic valve replacement with a Carpenter Edwards xenograft was carried out in September 1979. At operation the LV was dilated and poorly contractile. Despite correction of the aortic regurgitation he had recurrent episodes of LV failure before and after commencing haemodialysis in February 1980. Chronic LV failure caused him to stop work as an electrician in December 1979. By March 1980 he had gross cardiomegaly and persistent symptomatic ventricular ectopic beats were present (9139 ventricular ectopics recorded on a 24-h ECG). Hb was 7.8 g/dl with pre-dialysis plasma urea 27 mmol/l, creatinine 1.15 mmol/l and bicarbonate 19 mmol/l. Cardiac dilatation was evident on echocardiography with left atrial diameter 6.6 cm (normal range 1.9–2.3 cm), LV end systolic diameter 6.5 cm (normal range 2.5–4.1 cm) and LV end diastolic diameter 8.2 cm (normal range 3.5–5.6 cm). Contrast angiography
revealed a very large and poorly contractile LV with an ejection fraction of 20–30% and a normally functioning aortic valve prosthesis. The prognosis appeared so poor that combined renal and cardiac transplantation was considered.

His cardiac state improved to a limited extent and kidney transplantation alone was carried out in August 1980. He has been asymptomatic and out of LV failure since then and returned to full-time work in November 1980. He still has ventricular ectopies but these are far less frequent and do not trouble him. Resting ejection fraction measured by nuclear angiography was 42% in October 1980. Current intracardiac dimensions (echocardiography) have returned to near normal – left atrial diameter 4 cm, LV end systolic diameter 4.8 cm, LV end diastolic diameter 6 cm. Current medication is hydralazine 50 mg t.d.s. (for hypertension), prednisolone 7.5 mg, azathioprine 200 mg and 2 cyclophosphamide K tablets/d. Hb is 14.9 g/dl, CrCx 101 ml/min.

**Patient 3**

A man born in 1926 reached end stage renal failure due to mesangial proliferative glomerulonephritis in July 1983 and was subsequently managed with diuretics, dietary protein restriction and intermittent peritoneal dialysis. He had recurrent LV failure and angina pectoris which was not fully controlled by nitrates and nifedipine. By October 1983 he was in chronic congestive cardiac failure with a resting tachycardia, gallop rhythm, an elevated JVP (4–10 cm), hypotension (100/70 mm Hg) and cardiomegaly. Attempts to remove fluid during peritoneal dialysis failed owing to severe hypotension. Haemoglobin (Hb) was 8.5 g/dl, plasma urea 22.4 mmol/l, bicarbonate 17 mmol/l and creatinine 0.8 mmol/l. His ECG (Figure 1) showed widespread T-wave inversion with voltage evidence of LV hypertrophy. Nuclear angiography revealed LV dilatation with diffuse hypokinesia and a global ejection fraction of 17% (normal >50%). It was considered that renal transplantation offered the sole realistic prospect of satisfactory rehabilitation but, in view of the heart disease, the ethical justification for live donor renal transplantation was considered carefully before allowing the operation to proceed. The prospective donor was warned that the patient might not survive surgery.

In December 1983 he underwent live donor renal transplantation. Apart from one rejection episode and a cytomegalovirus infection, his postoperative course has been unremarkable. He has no chest pain or dyspnoea and there are no abnormal cardiovascular physical signs. Current daily medication is prednisolone 20 mg, azathioprine 100 mg and nifedipine (slow release) 20 mg b.d. (for hypertension). CrCx is 114 ml/min and Hb 11.1 g/dl. By February 1984 his ECG (Figure 2) showed normal T-waves but LV hypertrophy persists. Ejection fraction, measured by nuclear angiography, had increased to 54%.

**Discussion**

Heart disease is a frequent cause of death in patients on maintenance haemodialysis (Jacobs et al., 1977).
Echocardiographic and radionuclide studies commonly provide evidence of impaired LV function in haemodialysed patients even in the absence of symptoms and signs of heart failure (Lai et al., 1982). Studies of patients selected either at random from a dialysis population or because LV function appeared normal have shown that renal transplantation is followed by a reduction in LV mass (Ikaheimo et al., 1982) and volume (Ikaheimo et al., 1982; Montague et al., 1982). Some (Lai et al., 1982; Ulmer et al., 1982) but not all (Ikaheimo et al., 1982) authors have reported improved LV function following transplantation. The outcome after kidney transplantation in patients with clinically evident severe chronic LV dysfunction has been little reported. Ianhez et al. (1975) described 7 patients with heart failure before commencement of regular renal replacement therapy. Two improved after institution of haemodialysis and 5 following transplantation. These latter 5 patients received haemodialysis for an average period of less than 1 month before transplantation and it is impossible to know whether more prolonged haemodialysis would have controlled their heart failure. The three patients we describe had chronic disabling LV dysfunction associated in two with myocardial ischaemia and in the third with previous rheumatic valvular disease. In each, a remarkable and gratifying improvement in cardiac function followed kidney transplantation.

Possible contributory causes of LV dysfunction which might be corrected by transplantation include anaemia, chronic volume overload, presence of an arteriovenous fistula, hypertension, acidosis and retention of nitrogenous waste products. It is difficult to single out any of these as being of overriding importance in our patients. None was severely anaemic (mean pre-transplant Hb 8.9 g/dl rising to 13.8 g/dl postoperatively). In no patient could removal of salt and water during dialysis procedures correct LV failure and indeed this often led to a precipitous fall in blood pressure. Arteriovenous fistulae are still functioning in patients 1 and 2 while none was created in patient 3. All three patients had been hypertensive at some time but uncontrolled hypertension was clearly not the cause of the severe LV failure present shortly before transplantation. The uraemic state and acidosis were well controlled by treatment in each patient. Drug treatment might conceivably have played a part in improving cardiac function in our patients. Corticosteroid and immunosuppressive treatment may be of benefit in some cardiomyopathies (O'Connell et al., 1981; Melvin et al., 1982). Two patients received agents (nifedipine and hydralazine) that reduce ventricular afterload. We consider it unlikely that such medication is mainly responsible for our observations. No patient was alcoholic and cobalt treatment for anaemia was not employed.

Whatever the mechanism of improvement we describe it is clear that cardiac performance can improve dramatically after successful kidney transplantation. Too pessimistic an attitude towards referring or transplanting patients with severe LV dysfunction is unwarranted.

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References


