Captopril and Transplant Renal Artery Stenosis

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Dear Sir,

The recent excellent review of posttransplantation hypertension by Waltzer et al. [1] suggests that deterioration of renal function with captopril therapy may be indicative of the severity of transplant artery stenosis. We report a case in which labile renal dysfunction was precipitated by the use of captopril, in the presence of a narrowing of the transplant artery that was of dubious functional significance.

mm Hg) developed, which was controlled with metoprolol 25 mg b.i.d. Her renal function and hypertension control have now remained stable for 9 months after transplantation.

Alpha methyl

DOPA 250mg t.i.d.

1000-
- 800

Captopril 50mg t.i.d

Metoprolol 25mg b.i.d.

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=*■ 300H

“ 200

100-

60 50-40-30

20

Case Report

E.S. is an 11-year-old Caucasian female who received a cadaveric renal transplant 3 months after presentation in end stage renal failure associated with small sclerotic kidneys without evidence of immunologic, infective or vascular disorder. Prior to transplantation, she was maintained on continuous ambulatory peritoneal dialysis. Hypertension was controlled with captopril, 75 mg t.i.d.

At operation, there was a very short arterial segment on the donor organ. A postoperative renal scan showed poor perfusion, and an arterial thrombectomy and repair of an intimal tear with partial dissection of the arterial wall were performed, restoring good perfusion.

Immunosuppression was achieved with ciclosporin and prednisone. Hypertension was controlled with α -methyldopa 250 mg t.i.d., and nifedipine 10 mg p.r.n. After 2 weeks of nonfunction without rejection, graft function was established and plasma creatinine stabilized between 100 and 120 μ mol/l (1.2–1.4 mg/dl). α -Me-thyldopa was discontinued 4 weeks after transplantation

due to excessive sedation. Captopril (50 mg t.i.d.) was recommenced, which maintained adequate blood pressure control. Several days later, her plasma creatinine rose abruptly to 190 $\mu\text{mol/l}$ (2.2 mg/dl). Her plasma creatinine was subsequently unstable (fig. 1) without evidence of other clinical disorder and with therapeutic ciclosporin A levels. An angiogram showed arterial narrowing in one oblique view (fig. 2). Antihypertensive therapy was withdrawn to observe if renal function would stabilize at a higher arterial pressure. Her blood pressure remained within normal limits for 4 days after withdrawal of captopril; her plasma creatinine stabilized at approximately 100 $\mu\text{mol/l}$ (1.2 mg/dl). Mild hyperension (to 140/95

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CAPD

Angiogram

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100

20 40 60 80

DAYS POST TRANSPLANT

Fig. 1. A semi-logarithmic plot of plasma creatinine versus time and antihypertensive therapy in patient E.S.

Fig. 2. Oblique of renal transplant angiogram demonstrating relative narrowing at anastomotic site.

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Discussion

References

Most case reports of captopril-induced azotemia in transplant renal artery stenosis (TRAS) describe patients with refractory hypertension with persistent renal insufficiency subsequent to captopril therapy [2, 3]. The labile dysfunction described here has not been a feature of such reports. The use of angiotensin-converting enzyme inhibition with saralasin in the evaluation of posttransplant hypertension has failed to discriminate between high renin states associated with acute and chronic rejection and TRAS [4]. Nevertheless, the association of captopril therapy, renal insufficiency and significant TRAS has been reported with sufficient frequency that a concept has evolved that the former conditions imply the third. In our patient, renal function was

maintained by normal blood pressure, and the mild hypertension that did develop was more easily controlled than prior to transplantation. These observations imply that the angiographic abnormality was not of functional significance. Although impairment of transplant renal function by captopril therapy may be a sensitive indicator of arterial narrowing, the phenomenon lacks specificity and may exaggerate the significance of minor degrees of vascular stenosis.

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