Letter to the Editor

Nephron 1985;40:120-121

Percutaneous Transluminal Dilatation of Graft Renal Artery as Treatment for Posttransplantation Hypertension

C. Claudio Campieri
V. Vittorio Bonomini
Institute of Nephrology, S. Orsola University Hospital, Bologna, Italy

Vittorio Bonomini, MD, Institute of Nephrology, S. Orsola University Hospital, I-40138 Bologna (Italy)

Dear Sir,

benefited twice from percutaneous transluminal dilatation (PTD) of the graft renal artery. Posttransplant renal artery stenosis is a frequent cause of hypertension [1] and may sometimes mimic a rejection been controlled with 0.150 mg of clonidine daily during of the graft by reducing renal blood flow [2]. We present a dialysis treatment, exhibited a severe hypertensive crisis case of a patient who, under these circumstances, has (standing BP 180/110 mm Hg) and an increase in serum Fig. 1. Arteriograms before (a) and after (b) the first PTD. Arteriograms before (c) and after (d) the second PTD.

Repetition of Angioplasty in Graft Arterial Stenosis

Fig. 2. Clinical course of B.I., 40-year-old male. HD = Hemodialysis; = serum creatinine. PTD = Dilatation.

12-16n
200-10
Transplantation
1
11–10.
ng BP, mm
160–120-
HD
creatinine up to 1.7 mg% at about 3 months after transplantation without clinical and humoral signs of rejection. His previous clinical course after surgery had been uneventful. Antihypertensive therapy was gradually administered and increased up to 200 mg of captopril and 0.450 mg of clonidine daily. Despite these drugs, BP remained in the range of 180/110 mm Hg with episodic hypertensive crisis, and creatinine reached the level of 3.5 mg%. Therefore an arteriogram was performed that demonstrated a 75% stenosis of the graft artery with a polar branch stemming from the stenosed tract (fig. 1). 3 days later, the patient underwent PTD according to the technique of Grüntzig et al. [3]. Poststenotic systolic BP rose from 15 to 115 mm Hg. An arteriogram showed a 20% residual stenosis (fig. 1), and 5 days later BP went down to 140/80 mm Hg and creatinine to 1.3 mg%. Both remained at this level for the following 8 months. At 12 months after surgery an increase in BP was detected again (standing BP 175/100 mm Hg), and 0.150 mg of clonidine daily were readministered. Over the following 4 months clonidine had to be increased up to 0.450 mg daily because of resistant hypertension. At 18 months, because of still uncontrolled hypertension and slowly deteriorating renal function (creatinine 2.4 mg%), a new arteriogram was performed that demonstrated an 85% stenosis of the transplanted artery (fig. 1). PTD was performed during the same radiological session with a more striking result as compared with the previous procedure. A residual 15% stenosis was documented (fig. 1) and BP went down to 120/80 mm Hg on the third day without medication, persisting in this range over the next 6 months.
The clinical course, shown in figure 2 is typical for posttransplant renal artery stenosis that manifests itself early after surgery with the onset of hypertension. The latter is particularly severe and resistant to antihypertensive drugs. The increase in hypertension despite therapy, associated with a sharply declining renal function, must suggest a worsening of the stenosis. At this point an angiographic study is mandatory, preferably intravenous digital subtraction angiography, for the correct diagnosis and for the most appropriate treatment. In our case, PTD twice permitted a quick correction of hypertension and renal impairment without any of the side effects elsewhere reported [4]. This technique can be repeated unlike corrective surgery which is generally considered a high-risk procedure [5].

References