

Renal Transplant Hypertension Caused by Iliac Artery Stenosis

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A captopril renal study performed with both radiohippuran and $^{99m}\text{Tc-MAG}_3$ demonstrated the typical changes of a hemodynamically significant renal artery stenosis in a hypertensive renal allograft recipient. Arteriography demonstrated high grade stenosis not of the renal artery but of the iliac artery. After successful angioplasty, the patient's hypertension resolved.

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Hypertension in renal transplant recipients is a very common finding. It correlates negatively with graft survival, and hypertensive cardiovascular disease is a leading cause of death. The causes of hypertension in transplant patients are multiple and often difficult to separate from each other (1). They comprise acute and chronic rejection, cyclosporine toxicity, native kidney hypertension, renal artery stenosis (RAS), urinary obstruction and hypercalcemia. It is not uncommon to find more than one contributing factor (2). It is therefore important to identify the causes that are curable, such as hypertension caused by native kidneys (treated by nephrectomy) or graft RAS (treated by percutaneous transluminal angioplasty (PTA) or surgery) (3).

In our institution, hypertension in transplant recipients has been studied for many years. A differential diagnostic algorithm has been recommended by Laskow and Curtis (2). Nuclear medicine evaluation, which includes imaging with ^{131}I -iodohippurate (150 μCi) and more recently with $^{99m}\text{Tc-MAG}_3$ (5 mCi) complemented by effective renal plasma flow (ERPF) calculations, has been routinely used. In patients suspected of having RAS, captopril renography has been employed (3), using a protocol that consists of a baseline radionuclide study to exclude acute rejection or obstruction followed by a second study performed one hour after oral administration of 25 mg of captopril. Because of reports that diuretic administration reduces the

risk of false positive results, we routinely inject furosemide 15 minutes after injecting the tracer (4).

This report deals with an unusual finding of vascular hypertension due to stenosis not in the renal artery, but in the iliac artery.

CASE REPORT

The patient was a 48-yr-old female who received a cadaver transplant on 6/23/86. The initial graft function was excellent (ERPF = 333 ml/min) and the post-transplant course was uneventful. In July 1990, 1 wk before admission, she was seen by her physician who found high blood pressure (225/125 mmHg) and an abdominal bruit. At that time the BUN was 19 mg/dl, plasma creatinine 1.7 mg/dl. The patient was treated with Cyclosporin, Prednisone, Imuran, Lasix and Catopress.

Baseline and captopril renal studies were both performed on 7/17/90 with $^{99m}\text{Tc-MAG}_3$ (using 1 mCi for the baseline and 10 mCi for the subsequent post-captopril study). The captopril phase demonstrated findings typical of hemodynamically significant RAS, namely prolonged parenchymal transit time of the tracer resulting in marked parenchymal retention (Fig. 1). The postcap-

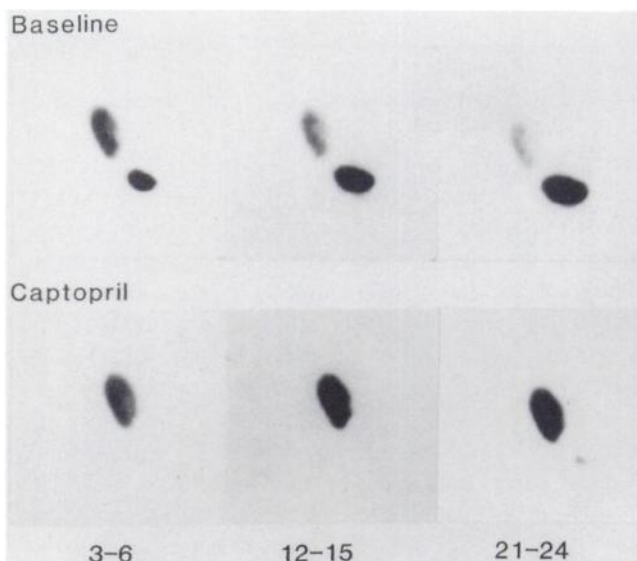


FIGURE 1. Selected frames of the baseline study (top) performed with $^{99m}\text{Tc-MAG}_3$ (1 mCi) and repeated 1 hr later (10 mCi) demonstrated prominent prolongation of parenchymal transit time of the tracer resulting in prominent cortical retention.

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topril renogram was repeated with iodohippurate and again prominent cortical retention developed while ERPF remained within normal limits (Fig. 2, upper panel).

An arteriogram performed on 7/18/90 revealed a normal graft renal artery but high grade stenosis of the right iliac artery proximal to the renal graft with an 80 mm systolic pressure gradient. The stenosis was treated by angioplasty with immediate improvement in the blood flow to the iliac, renal and femoral arteries (Fig. 3).

Another captopril renal study was performed on 7/20/90 (Fig. 2, lower panel) and demonstrated normal graft hemodynamics. The patient's antihypertensive medication was gradually withdrawn and blood pressure was found to be 135/88 mmHg. A repeat scintigram and arteriogram in October 1990, were judged to be normal.

DISCUSSION

The captopril renogram has been used since 1982 as a noninvasive test to diagnose hemodynamically significant

renal artery stenosis in patients suspected of having renovascular hypertension (4-6). Most of the published series deal with unilateral and bilateral stenoses in patients with native kidneys. Only a few laboratories have investigated the role of captopril renography in the diagnosis of renovascular hypertension in patients with graft artery stenosis (3,7-10). Before introduction of ACE inhibitor renography, the differential diagnosis between graft RAS, chronic rejection and hypertension originating in native kidneys in transplant recipients was possible only by arteriography (3).

Captopril induces efferent arteriole vasodilatation which leads to decline in filtration pressure, sharp fall in glomerular filtration and prolonged parenchymal transit of the renal tracers best seen with tubular agents such as OIH and $^{99m}\text{Tc-MAG}_3$. It has been suggested that such findings indicate a curable stage of the disease, while images unchanged after captopril even in the presence of anatomic

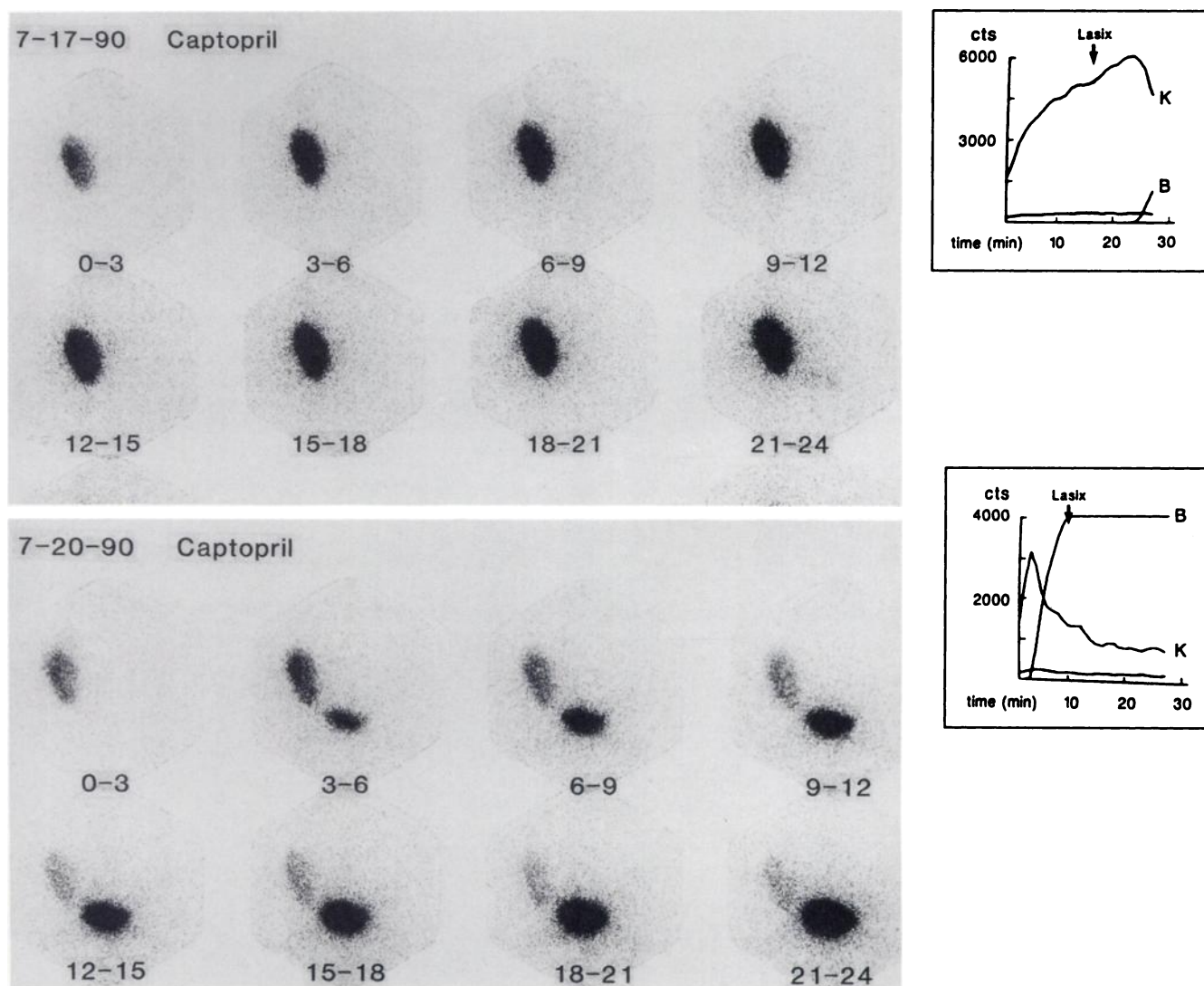


FIGURE 2. (Top) Captopril renal study performed with ^{131}I OIH (150 μCi) led to similar marked cortical retention (left) that had not resolved after Lasix injection (40 mg), indicated by an arrow (right). (Bottom) The captopril renal study repeated two days after angioplasty of the stenotic iliac artery demonstrated normal tracer kinetics.

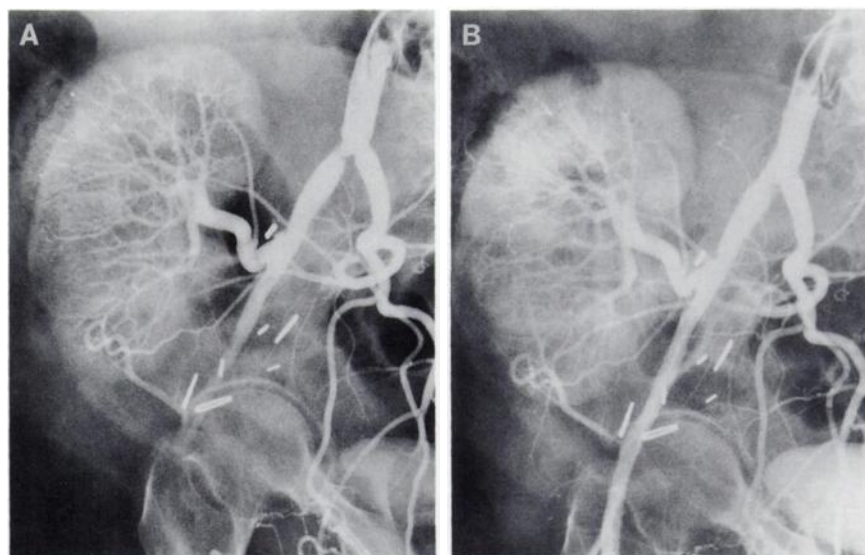


FIGURE 3. (A) Arteriogram demonstrates normal graft renal artery and high-grade right iliac artery stenosis immediately below the aortic bifurcation. (B) Repeat arteriogram after angioplasty that resulted in the normalization of the iliac artery lumen.

RAS predict poor response to angioplasty or surgery. In a series of renal transplant patients with renal artery stenosis, Drane (10) correlated captopril renography results with abnormal arteriograms and the outcome after intervention. He showed that cure correlated better with captopril-induced changes on scintigrams than with arteriograms. Our patient, in whom hypertension resolved after angioplasty of the high-grade stenosis of the iliac artery, showed characteristic captopril-induced changes on OIH and ^{99m}Tc -MAG₃ studies. This demonstrates that stenotic lesions not only in the renal artery but also more proximally must be considered as a source of the hypertension in interpreting a positive captopril renogram and recommending therapeutic interventions.

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