Captopril-induced acute renal artery thrombosis and persistent anuria in a patient with documented pre-existing renal artery stenosis and renal failure

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Summary

We describe an elderly man, with pre-existing renal failure and atheromatous renal artery stenosis, who developed persistent anuria due to renal artery thrombosis after acute hypotension following captopril administration. Caution should be used when captopril is first administered to patients with impaired renal function in whom renal artery stenosis is known or suspected.

KEY WORDS: renal artery thrombosis, captopril, hypotension.

Introduction

Captopril is a potent pharmacological agent used in the treatment of renin-dependent hypertension. Its main activity is in inhibiting angiotensin-I-converting enzyme. Recent reports have indicated that the dramatic fall in blood pressure which may follow captopril therapy can cause reversible renal failure (Planz and Bundschu, 1980; Kawamura *et al.*, 1982). Reversible renal failure occurring without hypotension has been reported in three patients by Silas *et al.* (1983) all of whom were found to have renal artery stenosis affecting a single functioning kidney and in 11 patients by Hricik *et al.* (1983) with severe hypertension associated with either bilateral renal artery stenosis or renal artery stenosis in a single kidney. The mechanism of this decrease in renal function is thought to result from a disturbance of glomerular filtration consequent to blockade of the renin-angiotensin system in the presence of decreased renal artery perfusion pressure (Hricik *et al.*, 1983). Interstitial nephritis causing reversible renal failure secondary to captopril therapy has also been reported (Farrow and Wilkinson, 1981; Hooke *et al.*, 1982). We now report a patient with documented pre-existing poor renal arterial blood supply in whom

captopril induced renal artery thrombosis and subsequent persistent anuria.

Case report

A 66-year-old man was admitted to hospital in November 1982, for stabilization of his hypertension of 2½ years known duration. In June 1981, he had had an abdominal aortic bifurcation graft inserted for aneurysmal dilatation. Pre-operative angiography had revealed aneurysmal dilatation of the aorta from below the renal arteries to the common iliac vessels (Fig. 1). There was complete occlusion of the right renal artery. The right kidney was supplied by a branch of the right ureteric artery. The left renal artery was stenosed by atheromatous plaques. The pre-operative blood urea was 15.7 mmol/l and creatinine 0.157 mmol/l and showed no significant deterioration 4 months later.

When admitted to hospital in November 1982 his blood pressure was recorded as 240/120 mmHg (lying) and 180/100 mmHg (standing). Blood urea 18.4 mmol/l and creatinine 0.182 mmol/l. His usual antihypertensive medications (labetalol, clonidine, amiloride and hydrochlorothiazide) were continued and captopril, 25 mg thrice daily, was introduced. Three hours after the first dose the blood pressure was recorded as 80/55 mmHg (lying) and 75/55 mmHg (standing). The patient remained hypotensive but uncomplaining for the next 48 hr. After receiving seven 25 mg tablets the captopril was stopped. The patient was transferred to our care after 3 days of anuria. High dose intravenous urography failed to reveal any nephrograms and there was no uptake on screening with technetium diethylene triamine penta-acetic acid. Aortography revealed no evidence of patency in either renal artery and no nephrogram was observed in either kidney. Oblique films showed

FIG. 1. Abdominal aortogram showing aneurysmal dilatation. There is also atheromatous stenosis of the left artery and no evidence of a patent right renal artery. The right kidney is supplied by a branch (arrowed) of the right ureteric artery.

a blind stump of a renal artery on the left side. He was maintained on intermittent peritoneal dialysis but remained anuric until cardiac arrest 2 months later.

Discussion

The left renal artery thrombosis was almost certainly due to the fall in blood pressure caused by captopril treatment. As thrombosis of the abdominal aorta affecting the right renal artery had already occurred the result was anuria. Renal artery thrombosis of a single functioning kidney secondary to antihypertensive treatment has only been reported on one previous occasion; atenolol was incriminated in this case (Shaw and Gopalka, 1982). In patients with two functioning kidneys thrombosis of an already severely narrowed renal artery has been reported during treatment with the angiotensin converting enzyme inhibitor enalapril (Hodsman *et al.*, 1983a) and occluded renal arteries were found in three patients on captopril therapy by de Zeeuw (Hodsman *et al.*, 1983b) but this was not, at that time, related directly to the treatment. However, in a review by Hollenberg (1983) of 269 patients treated with captopril, no episodes of renal artery thrombosis were recorded.

Both distal aortic thrombosis and renal artery

thrombosis may be clinically silent (Sabiston, 1982). Thrombosis of a stenosed renal artery during antihypertensive treatment can easily be missed. This appears to have happened in the case we describe. It has been suggested that in patients with known renal artery stenosis, and high renin activity or salt depletion captopril is likely to cause acute hypotension (Collste *et al.*, 1979). We recommend that in the event of a profound fall in the blood pressure an immediate graded infusion with angiotensin as previously described (Hodsman *et al.*, 1983c), be given or, if this is not available, normal saline should be infused. Also following our experience with two other patients with renovascular hypertension, both of whom had precipitous falls in blood pressure after a single 25 mg dose of captopril, it is now our policy to administer an initial dose of 1 mg captopril (in a lactose carrier) and monitor blood pressure quarterly for 4 hr. Following this we continue on the second day with 3.125 mg captopril—increasing incrementally according to response. Twenty-five patients have been treated in this way and only one of these required specific corrective measures for treatment of an acute hypotensive episode following a mg dose of captopril.

Captopril is often used in the treatment of hypertension that has proved resistant to other available therapeutic agents. In this context the response to

captopril is good; approximately 75% achieving complete control with either captopril alone or in combination with a diuretic (Hollenberg, 1983). However, there is considerable evidence that the renin-angiotensin system plays a crucial part in regulating the glomerular filtration rate when renal perfusion pressure is low (Blythe, 1983). The administration of captopril to patients with bilateral renal artery stenosis or renal artery stenosis affecting a single kidney tends to result in acute deterioration in the glomerular filtration rate which is usually reversible if captopril administration is stopped (Blythe, 1983). We have shown that acute hypotension following captopril administration can cause irreversible renal failure in a patient with renal artery stenosis affecting a single functioning kidney. Captopril should be used with caution in hypertensive patients with known or suspected bilateral renal artery stenosis or renal artery stenosis and a solitary kidney.

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