Reversible Acute Renal Insufficiency with Combination of Enalapril and Diuretics in a Patient with a Single Renal-Artery Stenosis

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Dear Sir,

Acute renal insufficiency induced by angiotensin-converting enzyme inhibitors has been previously reported in patients with bilateral renal-artery stenosis or renal-artery stenosis in a solitary kidney [1, 2]. Recently, reversible acute renal failure has been described with combination of enalapril and diuretics in patients without renal-artery stenosis [3]. Renal impairment with such a combination in patients with single renal-artery stenosis has not been reported.

A 68-year-old hypertensive patient was admitted for dyspnea on exertion. He had been previously treated by nifedipine (30 mg daily). His blood pressure was 260/130 mm Hg; plasma creatinine concentration was 110 $\mu\text{mol/l}$ and examination showed a grade II hypertensive retinopathy. Nifedipine was withdrawn and clonidine (0.150 mg daily) was started in addition to altizide (15 mg daily) and spironolactone (25 mg daily). As his blood pressure was not under control after 6 days, enalapril (10 mg daily) was introduced. 24 h later the 24-hour urinary output decreased from 1,400 ml to 300 ml while blood pressure gradually decreased from 200/120 to 120/80 mm Hg; serum creatinine level rose to 397 $\mu\text{mol/l}$. Spironolactone, altizide and enalapril were discontinued; within 48 h urinary output increased to 1,700 ml and serum creatinine level fell over 4 days to 109 $\mu\text{mol/l}$. An intravenous pyelogram showed a 9 cm right kidney and a 12.5 cm left

kidney. Digitalized intravenous angiography showed a right renal-artery stenosis with a normal left renal-artery. On day 9 enalapril was reintroduced (without diuretics) in increasing doses to a maximum of 5 mg twice daily. Blood pressure came under control and no changes were observed in serum creatinine levels.

The fall of glomerular filtration rate in patients with renovascular hypertension treated with angiotensin-converting enzyme inhibitors is thought to be the result of a failure in renal autoregulation: in the hypoperfused kidney, pharmacologic blockade of the renin-angiotensin system impairs the angiotensin II-mediated efferent arteriolar constriction. Thus, the occurrence of renal impairment needs the presence of bilateral renal-artery stenoses or renal-artery stenosis in a solitary kidney [1]. However, renal insufficiency in patients without large renal vascular disease has been reported [3, 4]; small renal vessel lesions could play a role. In our patient, it seems likely that nephroangiosclerosis was present in the left kidney. Moreover, sodium depletion

and volume contraction induced by diuretic therapy can enhance the effects of the renin-angiotensin system inhibition on glomerular filtration rate; Hricik [5] demonstrated a marked attenuation of the adverse renal effects of captopril after sodium repletion in a patient with transplant renal-artery stenosis. The previously reported patients without large renal vessel disease in whom angiotensin-converting enzyme inhibitors induced renal insufficiency

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were under diuretics [3] or presented with severe hyponatremia [4]. In our patient renal function remained normal when enalapril was reintroduced after withdrawal of the diuretics. We think that renal impairment was due to an increased renal hemodynamic effect of enalapril on the glomerular filtration rate in a sodium-depleted state. A combination of enalapril and diuretics should be used with caution, even in patients with a single renal-artery stenosis.

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