ABDOMINAL AORTIC ANEURYSM – A CONTRA-INDICATION TO ANGIOTENSIN CONVERTING ENZYME INHIBITOR THERAPY

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INTRODUCTION

Angiotensin converting enzyme inhibitors are becoming increasingly popular as first line agents for the treatment of hypertension and for earlier use in the management of congestive cardiac failure. They are, however, not without side effects which include hypotension, angio-oedema, cough, electrolyte disturbance, blood dyscrasias and rashes. In this article we will discuss acute renal failure caused by bilateral renal artery thrombosis patients with severe atheromatous disease of the aorta taking A.C.E. inhibitors. Firstly three clinical cases will be reviewed.

CASE 1

A 67-year-old man was admitted with a five day history of increasing intermittent claudication with rest pain at night, associated with epigastric and back pain. He was an ex-smoker with hypertension for the preceding eight years and a history of congestive cardiac failure. Five years previously he had been started on enalapril due to difficulty in controlling his hypertension. Four months later an intravenous pyelogram had been performed which was normal despite mild renal impairment (urea 10.9 mmol/l, creatinine 139 μmol/l). Two years prior to admission an abdominal aortic aneurysm was diagnosed, at which time a further I.V.P. showed a small left kidney. His other medications were frusemide, spironolactone, cimetidine, aspirin and salbutamol inhalers.

Examination revealed an ill and distressed man with a regular pulse of 100 beats per minute, blood pressure 70/50 mm.Hg., a raised jugular venous pressure and gross pulmonary oedema. His abdomen was tender with guarding and rigidity but his femoral pulses were present. He had a blood urea of 54 mmol/l and the serum potassium was 8.6 mmol/l, hence he was treated acutely with insulin and dextrose whilst peritoneal dialysis was established. Despite some biochemical improvement this was not accompanied by any change in his clinical condition and he died on dialysis three days after admission.

Post mortem examination revealed an abdominal aortic aneurysm filled by blood clot and atheromatous debris, extending down to the common iliac vessels with both origins of the renal arteries occluded.

CASE 2

A 73-year-old man presented initially with accelerated hypertension. He was an ex-smoker with a past history of duodenal ulceration and congestive cardiac failure. Five years previously an abdominal aortic aneurysm had been diagnosed leading to an aortic-left iliac and right femoral bypass graft for severe and extensive atheromatous disease. He made a good recovery from this operation with return of all peripheral pulses. Investigations on this admission revealed a urea of 13.3 mmol/l and creatinine 213 μmol/l with an I.V.P. showing a mass near the left ureteric orifice in the bladder and a non-functioning left kidney. His blood pressure was established and he was discharged on frumil and slow release nifedipine, to be readmitted several weeks later for further urological investigation. In the interim period his treatment was changed to frusemide and captopril because of poor blood pressure control.

On readmission he was generally unwell, in heart failure with a urea of 56.8 mmol/l and creatinine 1129 μmol/l. Ultrasound scan showed the collecting systems were not dilated. Peritoneal dialysis was instituted but despite an improvement in his biochemistry, he became dialysis-dependent and was referred to the Renal Unit for haemodialysis. Isotope dimercaptosuccinimide (DMSA) scan revealed no function in either kidney and on cystoscopy a papillary tumour was seen near the left ureteric orifice but not obstructing it. In spite of haemodialysis he remained very poorly and subsequently deteriorated and died one month after admission to the Unit.

His immediate cause of death was shown, at autopsy, to be a perforated duodenal ulcer with peritonitis. His kidneys were small and scarred with both renal arteries totally occluded by atheroma.

CASE 3

A 67-year-old man was initially admitted for the management of his congestive cardiac failure. He was an ex-smoker with a past history of perforated duodenal ulcer, hypertension for ten years and repair of his abdominal aortic aneurysm had been performed four years previously. He was taking frusemide for his heart failure.

Urea was 11.3 mmol/l with a creatinine of 159 μmol/l and echocardiography showed a poorly functioning left ventricle. He made a good improvement when his diuretics were increased and with the addition of enalapril. Over the next few months his urea remained between 17.5 and 19.5 mmol/l, his main complaint being of intermittent claudication at 200 yards. After a further three months his renal function had deteriorated with a urea of 45.8 mmol/l, creatinine 894 μmol/l, having had no change in his medication. His enalapril was discontinued and he was admitted to hospital for conservative management of his renal failure. He responded quite well to a low protein diet and reduction in his frusemide
and therefore was allowed home. Unfortunately he suffered a myocardial infarction complicated by supraventricular tachycardia two weeks later, which led to end stage renal failure. Despite peritoneal dialysis he died nine days after admission.

Post mortem examination revealed severe nephrosclerosis of both kidneys and almost complete bilateral renal artery stenosis with confluent mural thrombosis from the ascending to abdominal aorta.

DISCUSSION

There are at least three mechanisms by which A.C.E. inhibitors cause renal damage: by reducing glomerulo-filtration pressure; by causing systemic hypotension and by induction of glomerulonephritis. It is the first of these mechanisms that is of major importance in the three cases illustrated above. Uremia occurs mainly in patients who have either bilateral reno-vascular disease or a stenosis in the artery of a solitary functioning kidney. It is believed that renal artery stenosis decreases pressure in the afferent glomerul-arteriole and filtration pressure is then maintained by angiotensin 2 induced constriction of the efferent arteriole. When and A.C.E. inhibitor is given the efferent arteriole relaxes, the glomerulo-filtration pressure falls and renal failure results.

In a prescription event monitoring study of patients on enalapril who had died, 75 out of 913 cases examined showed a rise in the urea or creatinine concentration of 50% or more above pre-treatment values. Enalapril appeared to have contributed to a decline in renal function and subsequent death in ten of these patients. A recent retrospective study of 530 consecutive patients presenting to a Renal Unit as acutely uremic emergencies found 85 patients had reno-vascular disease considered responsible for their loss of renal function; 21 had uraemia which could be closely attributed to A.C.E. inhibitor treatment with 18 of these having significant reno-vascular pathology. Overall 3 of the 21 died.

Renal artery stenosis may be unsuspected. In this same study 400 consecutive hypertensive patients were examined in a specialist hypertension clinic and vascular imaging was performed when clinically indicated. Fifty-eight (i.e., 14.5%) were shown to have reno-vascular pathology. A group of London Radiologists met with interesting results when they carried out renal arteriography on 100 patients referred for investigation of their peripheral vascular disease. Most patients (59) were shown to have either stenosis (28 unilateral; 24 bilateral) or total occlusion (7) of the renal arteries. Interestingly out of those who had normal renal function (53 out of 100) the majority (31) had abnormal renal arteries. They concluded that peripheral vascular disease seems to be the best clinical marker for the presence of anatomical renal artery stenosis.

CONCLUSION

Many patients with hypertension and congestive cardiac failure have widespread atheromatous disease as shown in the examples cited and the research quoted. It is important to assess a patient clinically (smoker, intermittent claudication, ischaemic heart disease, arterial bruises), to check the renal function before and during therapy with A.C.E. inhibitors and to request vascular imaging as appropriate. This careful patient selection and subsequent monitoring should decrease the morbidity and mortality from renal insult induced by A.C.E. inhibitor treatment.

REFERENCES

1 Ferna RE. Adverse Effects on Angiotensin Converting Enzyme Inhibitors. The Adverse Drug Reaction Bulletin 141: April 1990


From the minutes of the medical committee meeting 29th October 1895

The medical staff unanimously recommended that a bicycle be purchased for the use of the house surgeon, owing to the extended area of the town.

C. N. Dean    W. Hall    C. A. Rayne    J. H. Irvin

Note: It was part of the house surgeon's duties to attend emergencies at all times day or night within the borough.