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Hypertension and renal failure

D J A Goldsmith, G Hamilton

A 74-year-old man with a history of intermittent claudication, cigarette-smoking, hypertension, and a successful recent right nephro-ureterectomy for a right ureter transitional cell carcinoma, presented with abnormal renal function and hypertension (plasma creatinine 228 μmol/l and blood pressure 200/100 mmHg on quinapril 20 mg). Fundoscopy showed grade II hypertensive changes. There was no abdominal bruit. There was no blood and only a trace of protein on urine reagent strip testing; phase-contrast urinary sediment microscopy was normal; 24-h protein excretion was 0.2 g (normal < 0.2 g/24 h), and creatinine clearance was 36 ml/min. Prior to the right nephrectomy, renal function had been abnormal (plasma creatinine 184 μmol/l).

Ultrasound of his abdomen showed a nonobstructed 9.3 cm left kidney with increased echogenicity and reduced cortical thickness. Isotope renography while on an angiotensin-converting enzyme (ACE) inhibitor showed no activity on the right (nephrectomy) and definite but sluggish excretion by the left kidney. Frusemide 40 mg/day was added, which reduced blood pressure a little (175/90 mmHg), but caused plasma creatinine to rise from 241 to 312 μmol/l.

Quinapril was continued at 20 mg daily and lacidipine 4 mg was added. The patient could not tolerate any strength of any other calcium channel blocker, nor a beta- or an alpha-blocker. Blood pressure rose further. Quinapril was increased to 40 mg/day, and plasma creatinine rose again to 386 μmol/l. Echocardiography showed significant left ventricular hypertrophy and diastolic dysfunction.

Questions

1 Is it likely that this patient has glomerulonephritis?
2 What is the most probable reason for the raised blood pressure and worsening renal function?
3 What options are available for effecting an improvement?

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Answers

QUESTION 1

It is unlikely that this patient has glomerulonephritis, as there was negligible proteinuria, no microscopic haematuria, and normal urinary sediment.

QUESTION 2

The most probable reason for the raised blood pressure and worsening renal function is renal arterial stenosis in a solitary kidney.

QUESTION 3

Angioplasty, arterial stenting and reconstructive surgery can all be useful in this condition.

Clinical course

Magnetic resonance angiography of his left renal artery strongly suggested an osteal renal artery lesion, confirmed at formal angiography, by which time the patient was experiencing breathlessness on exertion, orthopnoea and angina, and plasma creatinine was 420 μmol/l, creatinine clearance 20 ml/min, and blood pressure typically 200–210/90 mmHg. The degree of stenosis was so severe that the osteal meatus was a pinhole, and was occluded by the guide wire, so angioplasty with arterial stenting was abandoned. Quinapril was stopped and blood pressure controlled using lacidipine 6 mg, verapamil 240 mg, celiprolol 400 mg, and frusemide 80 mg. He was anticoagulated with heparin to forestall renal artery occlusion. He then underwent a left splenorenal bypass to relieve 95–99% left renal artery stenosis. Postoperatively there was a brisk diuresis, a marked improvement in renal function, and a marked fall in blood pressure. Some six months after the bypass surgery the patient is very well and has typical blood pressure values of 140/84 mmHg on lacidipine 4 mg daily. Plasma creatinine is 164 μmol/l with creatinine clearance of 46 ml/min. Exercise tolerance is limited only now by claudication; orthopnoea, breathlessness and angina have all resolved.

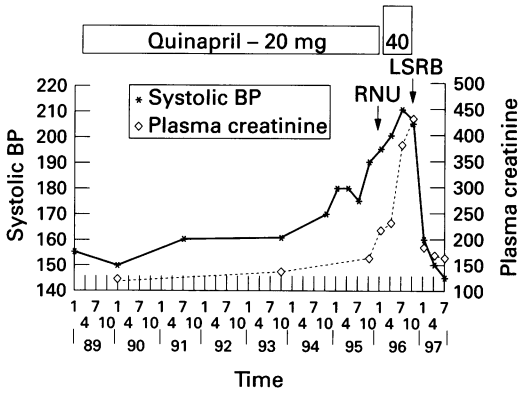

Figure 1 Time-plot of renal function and average blood pressure (BP). Nephro-ureterectomy (RNU) and renal arterial bypass (LSRB) are marked with arrows

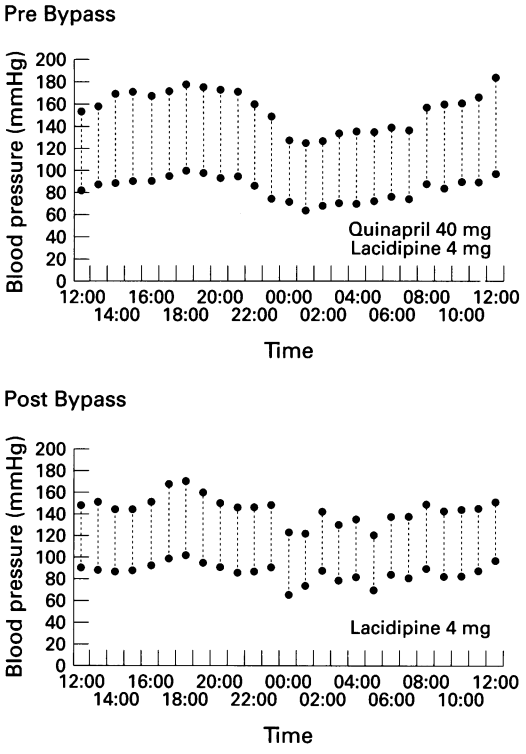

Figure 2 24-h Ambulatory blood pressure traces for subject six months before renal bypass (when on quinapril 40 mg, lacidipine 4 mg), and three months after renal bypass (when on lacidipine 4 mg alone)

The course of blood pressure and renal function is given in figure 1. Figure 2 shows the results of ambulatory blood pressure monitoring before and after the renal bypass.

Discussion

Renovascular disease is an important cause of acute and chronic renal failure. It is in part remediable by angioplasty, stenting or surgery.[1 2] In younger patients the pathology is fibromuscular hyperplasia, and can affect several arterial segments of both kidneys. In older patients the pathology is atherosclerosis, often contiguous with aortic disease and maximal peri-osteally.[3]

Early diagnosis and intervention are important; clinical clues are refractory hypertension, 'flash pulmonary oedema',[4] worsening renal function with the start or increase in ACE inhibitor (often reversible with ACE inhibitor withdrawal), renal bruit (neither a sensitive nor a specific sign) and contextual (age, smoking, atherosclerosis elsewhere[5]). Unilateral disease is often accompanied by renal size and functional asymmetry, the latter accentuated by test doses of an ACE inhibitor. No noninvasive method of diagnosis is as accurate as renal angiography; although spiral computed tomography has a sensitivity and selectivity of 95%.

Final diagnosis

Renal artery stenosis in a single kidney following nephrectomy.

Keywords: renal artery stenosis; hypertension

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Pleural effusion and a pelvic mass

D Agranoff, D May, C Jameson, G K Knowles

A 56-year-old woman presented with a nine-month history of lassitude, weight loss, an unproductive cough and increasing breathlessness. There was no complaint of chest pain or haemoptysis and she had never smoked. Examination revealed signs of a massive right pleural effusion, confirmed on chest X-ray (figure 1), and a large pelvic mass. There was no clinically detectable ascites, hepatosplenomegaly or lymphadenopathy. Full blood count, erythrocyte sedimentation rate, urea and electrolytes, liver function tests and blood calcium were all normal.

Approximately four litres of yellow fluid were removed by pleural drainage and multiple pleural biopsies were obtained. At laparotomy, a large mass was discovered in the right ovary (figure 2). The uterus contained multiple fibromyomata but no peritoneal nodules were present and there was no ascites.

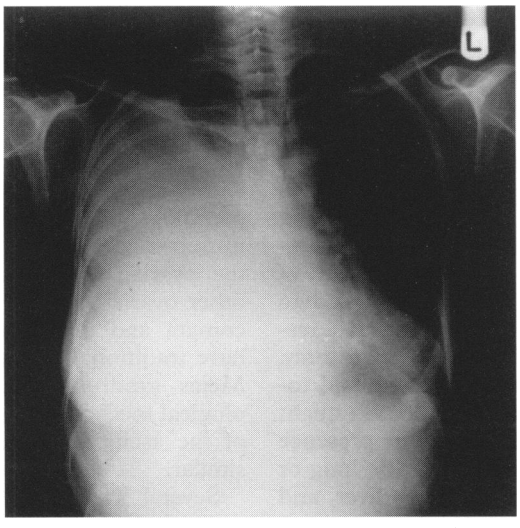

Figure 1 Chest X-ray showing right pleural effusion

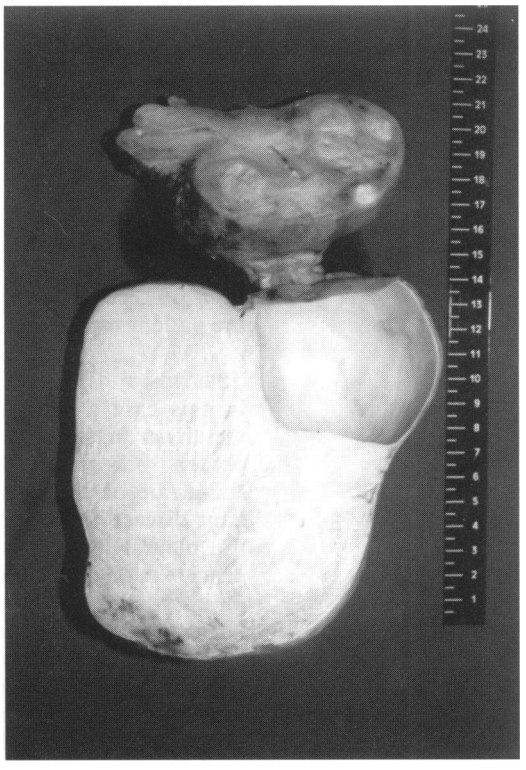

Figure 2 Right ovarian tumour attached to uterus containing multiple fibromyomata

Questions

1 What is the most probable diagnosis?
2 What additional diagnoses should be considered?
3 What is the correct management in this situation?

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