Case Report

Stretching of renal artery in a functionally solitary kidney: an unusual case of ischaemic nephropathy

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Key words: Ischaemic nephropathy; renovascular disease; renal failure; renal artery stenosis

Introduction

A variety of pathological lesions can cause renal artery stenosis [1]. Renovascular disease is most commonly the result of atherosclerosis but may also be caused by fibromuscular dysplasia, mainly in young females [1]. Rarer causes include atheroembolism, thromboembolism, arteritis, neurofibromatosis, renal artery aneurysm, dissection of the aorta, and arterial trauma. Extrinsic compression of renal artery by tumours or parapelvic cysts have also been described [1–3].

Renal artery stenosis is associated with two important potentially curable clinical syndromes, renovascular hypertension and ischaemic nephropathy [1,3–5]. The latter generally results either from bilateral renal artery stenosis or unilateral artery stenosis in a solitary kidney [1,3]. Ischaemic nephropathy is a frequent cause of renal failure and hypertension of the one-kidney, one-clip model in the ageing population with atherosclerotic cardiovascular disease [1,3,5].

Adding to the spectrum of renal vascular disease, we report a case of ischaemic nephropathy resulting from mechanical distortion by stretching of the renal artery of a solitary kidney in a patient with an atherosclerotic aortic aneurysm.

Case report

A 63-year-old white woman was referred to our hospital for hypertension, renal failure, and suspected renal artery stenosis on a solitary kidney. Six months before, the patient had been treated for acute pulmonary oedema associated with severe hypertension and myocardial ischaemia. Left ventricular (LV) failure was treated successfully. A mild renal dysfunction with a serum creatinine (Scr) of 173 μmol/l was also observed. The patient had no history of diabetes mellitus or hyperlipaemia but was a long-time smoker. She was not aware of having suffered from high blood pressure or renal insufficiency but an atrophic left kidney had been documented by intravenous pyelography 20 years earlier. Investigation revealed severe LV hypertrophy, moderate LV dilatation, and a LV ejection fraction of 50%. Abdominal echography revealed a left atrophic kidney (bipolar diameter of 8 cm) with marked cortical hyperechogenicity, a right kidney measuring 12 cm with normal cortical echogenicity, and an aortic aneurysm (4.5 × 3.0 cm). She was discharged with a calcium-channel antagonist (diltiazem 270 mg/day) and a diuretic (indapamide 2.5 mg/day).

During the following months, the patient’s blood pressure was poorly controlled, with values around 180/100 and frequent modifications of antihypertensive therapy. The diuretic was changed for an ACE inhibitor (enalapril, up to 7.5 mg/day) with transient improvement of blood pressure. Three weeks later, Scr was found to be increased at 218 μmol/l. Her angina worsened. A beta blocker and a long-acting nitrate were added and the dosage of the ACE inhibitor was decreased.

Upon admission in our centre, the patient’s medication consisted of nifedipine 40 mg/day, acebutolol 200 mg/day, enalapril 5.0 mg/day, and a long-acting nitrate. On examination, blood pressure was 200/95, a loud mid-abdominal bruit irradiating to the right flank was audible, and a pulsatile epigastric mass was palpable. Blood electrolytes and urinalysis were normal. Scr was 291 μmol/l and blood urea 24 mmol/l, giving an elevated urea/creatinine ratio of 0.083. Creatinine clearance was 25 ml/min. Enalapril was stopped and renal function improved. Scr decreased from 291 to 173 μmol/l in 12 days. Thereafter, two successive angiographies (a coronarography and an abdominal aortography) were carried out (Figure 1).

The coronarography revealed a one-vessel disease for which a medical treatment was suggested. Abdominal aortography confirmed the presence of an
aortic aneurysm with two successive dilatations of 3.1 and 4.5 cm in diameter and extending 10 cm below the renal arteries. It also revealed a left renal artery thrombosis and absent late ipsilateral nephrogram. The aorta was remarkable at the level of renal arteries for its kinked appearance and tapered narrowing of the proximal segment of the right renal artery with high-grade stenosis (Figure 2a).

An aortic aneurysm resection and a right aortorenal bypass were therefore planned. At surgery, prior to aneurysm resection, a thrill and weak pulse were palpable on the right renal artery. The aneurysm was resected and an infrarenal synthetic graft was installed. When the right renal artery was again palpated before proceeding to the aortorenal bypass, the thrill had disappeared and a strong pulse was felt. Right renal artery pressure was then measured by intra-arterial cannulation and was identical to radial artery pressure. The aortorenal bypass was thus omitted. The postoperative period was remarkable for an improvement of renal function with decrease of Scr, urea (Figure 1) and urea/creatinine ratio. An aortography performed 10 days after surgery showed straightening of the aorta and disappearance of the proximal renal artery stenosis (Figure 2b). Isotopic renal plasma flow also improved from 268 ml/min preoperatively to 375 ml/min postoperatively. At discharge, 12 days after surgery, the patient had a blood pressure of 130/70 and a Scr of 127 μmol/l. Her medication was nifedipine 40 mg/day, acebutolol 200 mg/day, and a long-acting nitrate. One year after surgery, the patient was doing well with Scr of 100 μmol/l and creatinine clearance of 48 ml/min. Four years after surgery, blood pressure remains well controlled and Scr unchanged at 100 μmol/l.

**Discussion**

Ischaemic nephropathy and/or renovascular hypertension usually results from obliteration of the renal artery lumen by atheroma or fimuscular dysplasia [1,3]. In addition, a number of other clinical entities can occasionally produce renal artery stenosis. These include arterial embolism, cholesterol embolism, vasculitis, and aortic dissection or thrombosis [1–3]. Furthermore, unusual causes of renovascular disease secondary to external compression of the renal artery have been reported [1,6]. Among these cases, only two were secondary to compression by an aortic aneurysm [7,8]. The first patient was a young man with a left suprarenal syphilitic saccular aneurysm [7] while the second was a middle-aged woman with a right infrarenal atherosclerotic saccular aneurysm [8]. The former patient had severe hypertension with mild elevation of Scr (227 μmol/l); the latter had severe renin-dependent renovascular hypertension with normal Scr. In both

![Fig. 1. Variation of blood urea and serum creatinine before and after aneurysm resection. Indicated by arrows are the end of ACE inhibitor administration, the coronarography (C), the aortography (A), and aortic surgery (S).](image)
Ischaemic nephropathy and stretching of renal artery cases, renal artery stenosis was caused by lateral compression and associated stretching of the vessel. Both had a contralateral functional kidney, which presumably assumed most of the renal function and represent examples of a two-kidney, one-clip Goldblatt model of renovascular hypertension.

We report herein the unusual occurrence of ischaemic nephropathy secondary to pure longitudinal stretching, without external compression, of a renal artery by an infrarenal aortic aneurysm in a woman with a functionally solitary kidney. Stretching of the right renal artery and narrowing of the vascular lumen resulted from kinking and left lateral displacement of the juxtarenal aorta by the saccular aneurysm. Resection of the aneurysm with pulling back of the aorta toward the midline relieved traction on the renal artery and reopened the vascular lumen by simple recoil. The proximal high-grade stenosis disappeared and the renal ischaemia resolved. Blood pressure control also improved, as expected in a one-kidney, one-clip model of renovascular hypertension.

This case represents a rare cause of ischaemic nephropathy, angiographically similar to a proximal atherosclerotic renal artery stenosis, but due entirely to the extreme stretching of the vessel. Careful exploration of the stenotic segment before and after aneurysm resection resulted in the avoidance of an unnecessary aortorenal bypass. Postoperative angiography and favourable evolution confirmed the surgical finding that vascular obstruction was completely relieved by the aneurysm resection.

References
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Received for publication: 2.6.97
Accepted: 12.6.97