Kidney at risk: 11-year course of renal artery stenosis

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Case history

In 1990, a 49-year-old woman with unstable angina was admitted for coronary angiography, which showed two-vessel disease. She had a 33-year history of hypertension and so renal angiography was also performed which revealed a non-stenosing atherosclerotic plaque in the proximal left renal artery (Figure 1A). Blood pressure was controlled with two antihypertensive drugs. The calculated creatinine clearance was 61 ml/min, and was unchanged since 1974.

In 2001, aged 60, she was admitted again because of unstable angina. A coronary angiogram revealed three-vessel disease. Renal arteriography now showed a high-grade stenosis of the left renal artery where the plaque was seen 11 years before (Figure 1B). Blood pressure was still well controlled with three antihypertensive drugs. Renal function was unchanged. Captopril scintigraphy with $^{99m}$Tc-mercaptoacetyltriglycine (MAG$_3$) showed equal perfusion of both kidneys, but severely impaired glomerular filtration on the left side indicated by massively enhanced tracer accumulation in a later phase (Figure 2A). This confirmed the haemodynamic significance of the angiographic finding. Percutaneous transluminal angioplasty with stent implantation was performed.

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Fig. 1. (A) Angiogram of the left kidney from 1990 with non-stenosing atherosclerotic plaque (arrowhead). (B) Angiogram of the left kidney from 2001 with high-grade stenosis of the proximal left renal artery (arrowhead). (C) Angiogram of the left kidney 2 months later after percutaneous transluminal angioplasty with stent implantation (arrowhead).
Discussion

Atherosclerotic renal artery disease causes renovascular hypertension and progressive kidney failure. Progression from a non-stenosing plaque to renal artery stenosis (>60% of diameter) occurs in 60% of patients over 5 years. Renal artery occlusion happens in less than 10% of patients within the same period [1]. Treatment options are aggressive antihypertensive and lipid-lowering therapy, percutaneous transluminal angioplasty with or without stent implantation, and surgical reconstruction. Revascularisation may prevent disease progression by restoration of arterial patency, but can cause renal infarction, contrast nephropathy or cholesterol embolism. Therefore the benefit of revascularisation and the optimal time of intervention is still a matter of debate [2,3], since no long-term follow up investigations are available.

Our patient illustrates: (i) the slow progression of atherosclerotic renal artery disease over 11 years with stable renal function for 25 years, after 35 years of well controlled hypertension; (ii) a different progression rate of atherosclerosis in coronary compared with renal arteries; (iii) the normalisation of angiographic and scintigraphic findings and reduction of antihypertensive treatment after angioplasty with stent implantation. We suggest careful follow-up of such patients with aggressive medical treatment of hypertension and hyperlipidaemia and with intervention only when renal artery occlusion is impending according to scintigraphic and angiographic findings.

References


Fig. 2. (A) Captopril scintigraphy of both kidneys with $^{99m}$Tc-labelled MAG3 from 2001 (at the same time as the angiogram of Figure 1B): massive tracer accumulation in the left kidney during the later phase due to severely impaired glomerular filtration with decreased urine flow on the left side compared to a normal scintigram on the right side. (B) Normalisation of the scintigraphic findings one month after angioplasty with stent implantation (angiogram of Figure 1C).