Case reports

Effect of renal artery stenting on the progression of renovascular renal failure: a case of intravascular ultrasound-confirmed renovascular disease

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ACE-inhibitors; Angioplasty, peripheral; Ultrasounds.

We report the case of a 71-year-old male, submitted to percutaneous transluminal renal angioplasty (PTA) plus stent implantation following the confirmation, at intravascular ultrasound, of severe unilateral renal artery stenosis in the setting of a single functional kidney and of evidence of renal insufficiency (serum creatinine value 300 μmol/l). At 6 months of follow-up the serum creatinine levels had returned to normal (98 μmol/l). This case shows the role of direct PTA on the overall renal function in a case of global renal ischemia.

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Introduction

The high prevalence of obstructive renal vascular disease in adults is well documented, particularly in the presence of hypertension, diabetes mellitus, coronary heart disease and peripheral vascular disease[1]. Among patients with atherosclerotic renal artery stenosis (ARAS), a progressive narrowing was reported in 51% of renal arteries 5 years after the initial diagnosis; 9 to 16% of these stenoses progressed to total occlusion[2]. The risk of progression is highest among individuals with a preexisting high-grade ARAS, in which renal atrophy develops in 21% of patients with a renal artery stenosis occluding more than 60% of the arterial lumen and leading to the development of an "ischemic atrophic nephropathy" with chronic renal failure[3,4]. It is estimated that yearly, atherosclerotic renovascular disease is the cause of renal failure in 5 to 15% of adult patients who begin dialysis[1] and, among patients who are receiving dialysis, those with renovascular disease have the lowest survival rate[2]. Therefore, the effect that renal artery stenting may have on the subsequent renal function and on the need for dialysis in patients with atherosclerotic renovascular disease and renal impairment is of particular clinical and economic concern. Recently, endovascular stenting has partially substituted simple balloon angioplasty or surgical revascularization as the procedure of choice for the treatment of ARAS. Several reports have demonstrated an excellent technical success, low complication rates and low rates of restenosis after the procedure[5-7]. Despite these favorable reports, the ability of revascularization to preserve or salvage the function of the treated kidney has not been demonstrated completely. A major drawback on the study of the direct effect of treatment on the treated kidney has been the lack of simple methods to assess the function of the kidneys separately. We report a case that should prompt discussion of the problem regarding the direct beneficial effects of revascularization on the renal function after renal artery stenting of a hemodynamically significant ARAS.

Case report

A 71-year-old male was admitted to our hospital because of deteriorating renal function. The patient was a cigarette smoker, hypertensive and had a long history of dyslipidemia and peripheral vascular disease. At 61 years of age, in view of worsening claudication, he underwent a lumbar sympathectomy and, some years later, a surgical aortoiliac revascularization procedure. At the age of 66, he was sub-

mitted to atherectomy of the left internal carotid artery. When he was 70, after some episodes of paroxysmal constrictive pain induced by exertion, and after a stress test had showed evidence of ischemia in the lateral leads at 100 W, a diagnosis of stable angina was made and the patient placed under medical treatment. Three weeks later, he was admitted to our department because of a new episode of angina during a hypertensive bout (arterial pressure value of 210/110 mmHg) that was treated efficaciously with intravenous nitrates. An angiographic examination of the coronary arteries was performed and revealed a 45% reduction in the lumen of the left circumflex artery. Computer-based quantitative coronary angiography (QCA) revealed a 20% stenosis of the left anterior descending artery. Left ventricular angiography showed a preserved left ventricular systolic function with an ejection fraction of 55% and no segmental kinesis abnormalities. A subsequent abdominal aortography with a pig-tail catheter (Cordis, Johnson & Johnson, Miami, FL, USA) showed extensive atherosclerotic disease of the abdominal aorta which was characterized by a shaggy luminal surface, and an apparently moderate stenosis (40% at QCA) at the ostium of the left renal artery. The right renal artery was not visualized. A renal perfusion scintigraphy with technetium-labeled panteic acid (DTPA), confirmed the suspicion of a single kidney. Sequential analogic images showed a mild inhomogeneous radionuclide uptake of the upper pole of the left kidney during the perfusion phase; the left kidney had a normal morphology and dimensions and a normal down flow. Furthermore, the renographic curve of the left kidney was normal with a glomerular filtration rate of 71 ml/min/m^2, a time to peak activity of 220 s and a Y/2 of 900 s. The test was considered normal and the patient was discharged on a therapeutic regimen including progressively increasing enalapril doses (from 5 to 20 mg daily).

Three months after having been discharged from the hospital, the patient presented with a yet worsened renal function: urea nitrogen and creatinine levels rose from 122 μmol/l and 7.31 mmol/l respectively to 300 μmol/l and 10 mmol/l. On the basis of the suspicion that renal failure was attributable to the ACE-inhibitor treatment (in a patient with unilateral renal artery stenosis and a single kidney), a selective renal arterial angiography was proposed with the aim of verifying the need of percutaneous transluminal angioplasty (PTA). The patient was re-admitted for left renal artery catheterization using the femoral approach. A right Judkins catheter (Cordis, Johnson & Johnson, Miami, FL, USA) was used to cannulate, via the right femoral artery, the left renal artery and an intravascular ultrasound (IVUS) examination was performed. A 3.0F, 40 MHz transducer, IVUS catheter usually used for the coronary artery (Clearview Ultra System, Boston Scientific, Galway, Ireland) showed a calcified, eccentric atherosclerotic plaque at the origin of the proximal left renal artery and allowed a more clear picture of the stenosis seen during angiography (Fig. 1). Therefore, a primary stenting of the left renal artery was performed (HerculinK^{TM} plus 6/12 mm, Guidant, Santa Clara, CA, USA) at the ostium of the left renal artery. The residual intra-stent stenosis was minimal, as confirmed at angiography (10% at QCA) and at IVUS (Fig. 2). Sequential serum creatinine and urea nitrogen determinations were made at 1, 6 and 12 hours after stenting: the serum creatinine level progressively fell to 281, 248 and 187 μmol/l and that of urea nitrogen to 10.5, 10.0 and 9.3 mmol/l. Urinalysis was normal. The patient was discharged the day after, and the only change in therapy was the addition of clopidogrel for 1 month. Within 6 months of follow-up, the serum creatinine levels had fallen to a normal value of 98 μmol/l (Fig. 3).

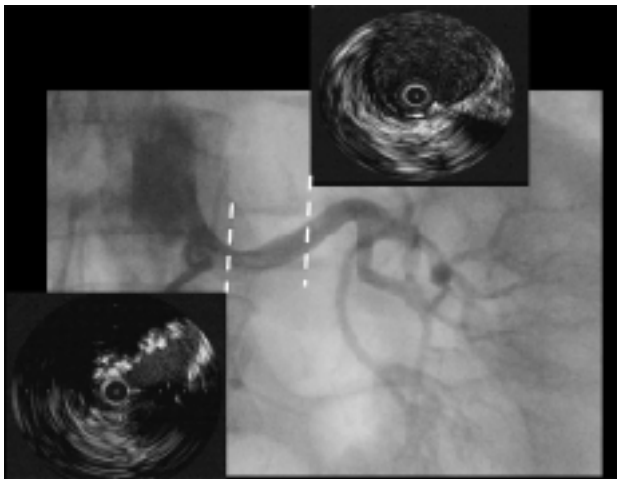

Figure 1. *Angiogram and corresponding intravascular ultrasound cross-sections obtained before stent placement. The lower intravascular ultrasound image shows the extent of renal artery narrowing.*

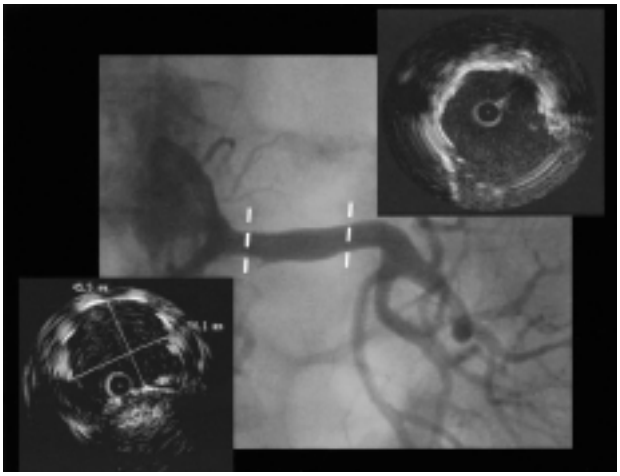

Figure 2. *Angiogram and corresponding intravascular ultrasound cross-sections obtained after stent placement. The lower intravascular ultrasound image shows the results of percutaneous transluminal angioplasty and stenting.*

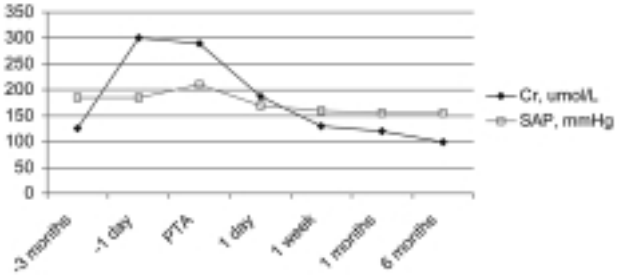

Figure 3. *Systolic arterial pressure (SAP) and serum creatinine (Cr) levels before percutaneous transluminal angioplasty (PTA) and at follow-up.*

Discussion

On reviewing the literature supporting ARAS as the cause of progressive renal failure, two major problems come to mind. First, the coexistence of ARAS and chronic renal failure or end-stage renal disease is assumed to be the cause and effect[8] and secondly, the accuracy of the diagnosis of ARAS is, as shown by the creatinine clearance and glomerular filtration rate, sometimes questionable[1-4,7].

In the first case, many interventions had been increasingly performed and reported, but despite the technical success and the introduction of stenting for recalcitrant ostial lesions, the results, in terms of the overall renal function (creatinine or glomerular filtration rate), were at best discordant[1-4,7]. In many of these cases a unilateral ARAS coexisted with advanced chronic renal failure, suggesting that the concomitant parenchymal dysfunction of the companion "non-stenosed" kidney obscured any direct effects of revascularization on the function of the treated kidney and rendering an overall renal function recovery after PTA unlikely. This confused picture can largely be explained by hypothesizing that these patients present with two distinct modes of renal impairment with a common background: arteriolosclerosis and large vessel atheroma. To circumvent this problem and isolate the direct effect of revascularization on the overall renal function, Watson et al.[1] studied patients with chronic renal impairment and ARAS involving all the renal arteries or with unilateral ARAS in the setting of a solitary or single functional kidney (global renal ischemia) and provided evidence that stenting of the renal artery is effective in either improving or stabilizing the renal function or in preserving the kidney size. The point is to identify how important ARAS is as a cause of renal disease. With regard to this, the accuracy of available invasive and non-invasive tests remains questionable. Nuclear imaging tests with DTPA do not provide anatomic information about the renal arteries. Moreover, their accuracy is reduced in patients with an impaired renal function. Magnetic resonance angiography and duplex ultrasonography are probably the most promising and accurate non-invasive screening tests, even in the presence of renal failure[9]. In our case, nuclear imaging with DTPA failed to provide evidence of significant ARAS. The diagnostic accuracy of renal scintigraphy approximates 65% and unfortunately is not improved by the use of captopril (68%)[10]. In our case, renal artery angiography was not of diagnostic value. However, the increase in the plasma creatinine concentration after the increment in the dosage of the ACE-inhibitor, and in the absence of any other apparent cause, unmasked the subsequent IVUS-confirmed severe ARAS. Moreover, IVUS is also particularly useful for the correct positioning and optimal expansion of the stent. This is important in order to minimize the incidence of subacute thrombosis and of late restenosis[11]. With regard to treatment, only a few studies comparing percutaneous treatment and surgical therapy are available[12]; the outcome of these highlight the fact that the technical and clinical results of these approaches are similar, although lower rates of complications are associated with percutaneous treatment since it does not require general anesthesia, is significantly less invasive, and since patients recover within a shorter time[13]; in addition, percutaneous treatment is substantially less costly than surgery, which however remains the treatment of choice for patients with specific vascular conditions[14,15]. Nevertheless, in these studies percutaneous treatment was associated with a higher restenosis rate, requiring a close follow-up and further intervention. Results reported in a recent randomized trial in patients with ostial ARAS demonstrated that stenting and PTA have similar complication rates, although PTA is associated with a lower primary success rate and a higher restenosis rate[14]. In conclusion, we suggest, in the presence of global renal ischemia with an indication for ACE-inhibitors, a more detailed diagnostic work-up, followed, when necessary, by endovascular intervention in an attempt to prevent or delay the progression to end-stage renal failure.

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