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Case Report

Acute Pulmonary Edema after Endovascular Treatment for Renal Artery Stenosis - Clinical Evolution after Angioplasty and Stenting in Moderate Stenosis: A Case Report

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Abstract

Renal Artery Stenosis (RAS) is frequent in hypertensive patients undergoing coronary angiography. We present a case of a 42-years-old woman with hypertension, dyslipidemia, type-2 diabetes, chronic kidney disease and previous myocardial infarction, who was admitted at the emergency department with typical angina. She was diagnosed with acute myocardial infarction. Coronary angiography showed severe stenosis in right coronary artery and successfully submitted to angioplasty with stent insertion. Renal ultrasound was performed with a Doppler study of renal arteries and showed bilateral chronic nephropathy and signs of significant stenosis of the left renal artery. The blood pressure was controlled during hospital admission and renal function was normal the patient was discharged for brief outpatient evaluation. However, she was admitted again with Hypertensive Acute Pulmonary Edema (HAPE) and was indicated renal angiography. It demonstrated obstruction of 50% and 70% in the ostium of the right and left renal arteries, respectively. Left renal artery angioplasty was performed without complications. Twenty-four hours after angioplasty and stent insertion the patient evolved with new HAPE and even after medical adjustments the blood pressure persisted high. Because this clinical evolution after angioplasty it was suspected of renal stent stenosis. A new renal ultrasound with Doppler was performed; the left renal artery stent was patent with no stenosis and normal velocity at the origin of the right renal artery. Although the renal ultrasound with Doppler did not demonstrate signs of significant stenosis at right renal artery, was decided perform angioplasty considering clinical features and evolution with recurrent HAPE. After the second angioplasty, the patient had a good clinical evolution, improvement of renal function and blood pressure levels.

Keywords: Atherosclerotic Renovascular Disease; Hypertensive Acute Pulmonary Edema; Renal Artery Angioplasty; Renal Artery Stenosis

Introduction

Renal Artery Stenosis (RAS) affects about 5% of hypertensive patients [1], more than 20% of patients undergoing coronary angiography [2] and up to 33% of cases of aortic atherosclerotic disease [3]. There are three major clinical syndromes related to RAS: incidental finding in an asymptomatic individual, ischemic nephropathy, and renovascular hypertension. The main etiologies are atherosclerotic disease (approximately 90% of cases) and fibromuscular dysplasia, which is more common in young women. Atherosclerotic renovascular disease is more common in men [4], above 50 years old, with multiple atherosclerotic risk factors. RAS is one of the most frequent causes of secondary hypertension and it is challenging to determine whether elevated blood pressure is related to RAS taking into account the degree renal artery obstruc-

tion. We report a case of a patient who underwent a successful percutaneous interventional treatment in renal arteries, despite non-significant stenosis based on clinical features.

Case Report

A 42-years-old woman was admitted to the Emergency Department (ED) of the Heart Institute, São Paulo Medical School, Brazil, with typical angina. The patient reported diagnosis of hypertension since she was 30 years old, a history of poor drug adherence and worsening of blood pressure control for the last eight months. Furthermore, she reported dyslipidemia, type-2 diabetes mellitus with insulin therapy, chronic kidney disease and previous myocardial infarction. Her initial vital signs were as follow: regular heart rate of 87 beats per minute, Blood Pressure (BP) of 180x110 mm Hg, and respiratory rate of 20 breaths per minute. The oxygen saturation was 97% while breathing in ambient air. The electrocardiogram showed sinus rhythm, left ventricle hypertrophy and no ST segment abnormality. Serum troponin was 27.89 ng/mL (reference value < 0.04), creatine kinase MB 11.26 ng/mL (reference value < 3.8 ng/mL), hemoglobin 10.6 g/dL, hematocrit 33%, leukocytes 8120 /mm^3, platelets 466.000 /mm^3, total cholesterol 199 mg/dL, HDL cholesterol 45, LDL cholesterol 132, triglycerides 108 mg/dL, glucose 87 mg/dL, urea 91 mg/dL and creatinine 2.05 mg/dL. The admission diagnosis was consistent with acute myocardial infarction. On the echocardiogram, the left ventricular systolic function was mildly reduced with ejection fraction of 45%. Coronary angiography showed severe stenosis in the right coronary artery (Figure 1).

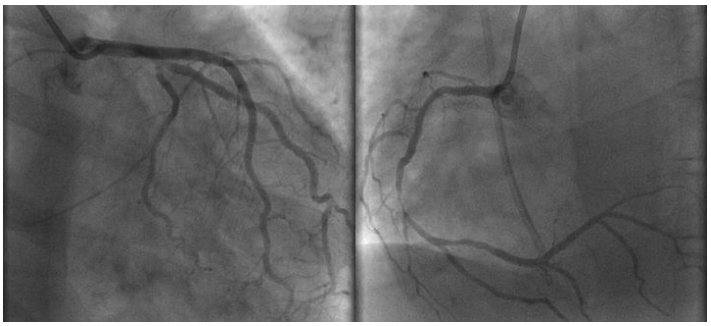

Figure 1: Coronary angiography showing severe stenosis on proximal third of the right coronary artery.

that was successfully submitted to angioplasty with stent insertion. Due to the high blood pressure, there was necessary the use of nitroglycerin and the patient was referred to the Coronary Unit. There was difficulty in weaning the intravenous vasodilator, requiring the use of three classes of oral antihypertensive (Carvedilol 25 mg twice daily, Amlodipine 5 mg twice daily, Clonidine 0.1 mg twice daily) beyond aspirin 100 mg and Atorvastatin 80 mg once daily. Renal ultrasound was performed with Doppler study of renal arteries and showed bilateral chronic nephropathy and signs of significant stenosis of the left renal artery. The peak flow velocity in the left renal artery was 294 cm/s (reference value <150 cm/s) and in the right renal artery was 94 cm/s (reference value <150 cm/s). Because BP was under control during the hospital admission and renal function remained stable, the patient was discharged from the hospital and referred for early outpatient evaluation. However, while waiting for the assessment, she was admitted again at the ED of the Heart Institute, due to acute pulmonary hypertensive oedema (BP-190/120 mmHg). Because of the failure to drug treatment, persistently elevated BP and acute pulmonary hypertensive oedema (APHE) renal angiography was indicated. It showed stenosis of 50% in the right renal artery ostium and 70% in the left renal artery ostium. Left renal artery angioplasty was performed without complications.

Twenty-four hours after angioplasty and stent insertion the patient evolved with new APHE, requiring non-invasive ventilation, vasodilator, intravenous diuretic and the need to increase oral antihypertensive doses (Figure 2).

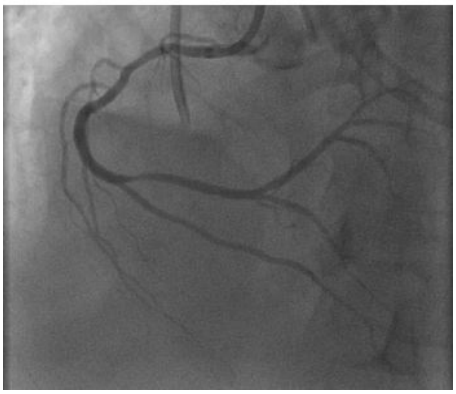

Figure 2: Coronary angiography showing the final view after angioplasty and stent insertion.

With the introduction of Enalapril 5 mg twice daily it was registered the worsening renal function (creatinine: 2.6 mg/dL) and hyperkalemia, with improvement after with draw. The patient remained with elevated BP, even with four antihypertensive drugs combination. Face to this clinical evolution after angioplasty was suspected of renal stent stenosis. A new renal ultrasound with Doppler was performed and demonstrated increasing in systolic velocity at the origin of the left renal artery (300 cm/s; normal value <150 cm/s) and normal velocity at the origin of the right renal artery (102 cm/s; normal value <150 cm/s). Patient was referred to renal angiography. The left renal artery stent was patent with no stenosis (Figure 3).

Figure 3: (A) Renal angiography showing severe stenosis in the ostium of the left renal artery. (B) final view after angioplasty and stent insertion.

Although the renal ultrasound with Doppler did not demonstrate signs of significant stenosis at right renal artery, was decided perform angioplasty considering clinical features and evolution with recurrent APHE. After the second angioplasty, the patient had a good clinical evolution, improvement of renal function and control of BP levels. She was discharged three days after the procedure, with 3 antihypertensive. Currently, she is been following-up at the ambulatory of the Heart Institute with controlled BP and no further complications.

Discussion

In this case report we present the net benefit of renal revascularization in a patient with no significant renal stenosis but with clinical features of renovascular hypertension. The aim of revascularization in RAS is to reduce obstruction to renal perfusion, thereby improving downstream ischemia and reducing activation of Renin-Angiotensin-Aldosterone System (RAAS), resulting in improved blood pressure control and preservation of renal function. The difficulty lies in knowing which patients will benefit from revascularization as a high degree of stenosis may not correlate with significant hemodynamic effects and RAS may be an incidental finding [5]. In our patient we have seen an improvement in the blood pressure levels control, renal function and no more recurrent pulmonary edema. Data from studies in the United States have indicated that revascularization is performed in 16% of patients with newly diagnosed atherosclerotic renovascular disease [6]. Since endovascular interventions are associated with substantial morbidity [7], inconvenience, and cost, with little apparent benefit, the widespread use of such procedures outside of clinical trials have been questioned.

Data from randomized clinical trials, including the Angioplasty and Stenting for Renal Artery Lesions (ASTRAL) trial [8] and the Stent Placement and Blood Pressure and Lipid Lowering for the Prevention of Progression of Renal Dysfunction Caused by Atherosclerotic Ostial Stenosis of the Renal Artery (STAR) trial [9], that assessed the usefulness of renal artery stenting with respect to kidney function showed no significant difference with respect to renal function, blood pressure, renal or cardiovascular events, or mortality compared endovascular revascularization plus medical therapy with medical therapy alone in patients with atherosclerotic renovascular disease. These studies have been criticized for enrolling some participants who did not have clinically significant renal-artery stenosis and for not having their findings confirmed by core laboratories [10]. The Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) study included patients with severe renal artery stenosis that was defined angiographically as stenosis of at least 80% but less than 100% of the diameter or stenosis of at least 60% but less than 80% of the diameter of an artery, with a systolic pressure gradient of at least 20 mm Hg. It was found no benefit of stenting with respect to the rate of the composite primary end point or any of its individual components, including death from cardiovascular or renal causes, stroke, myocardial infarction, congestive heart failure, progressive renal insufficiency, and the need for renal-replacement therapy (Figure 4).

Figure 4: (A) Renal angiography showing moderate stenosis in the ostium of the right renal artery. (B) final view after angioplasty and stent insertion.

Current clinical consensus would generally not advocate screening for atherosclerotic RAS in everyone, particularly where it will not the alter management plan [11]. Suggested criteria for which patients to investigate are outlined below: rapid onset of hypertension over the age of 55 years with additional adverse features (difficult-to-control blood pressure, association with decline in renal function, association with heart failure); rapid, unexplained decline in renal function (more than 50% increase in serum creatinine concentration over 12 months) or significant deterioration in renal function after introduction of an ACE inhibitor or angiotensin II receptor blocker; and repeated hospital admissions for heart failure with preserved left ventricular function on echocardiography.

The management goals in RAS are to control hypertension, preserve renal function and prevent recurrent flash pulmonar edema. The use of revascularization in management of flash pulmonar edema is supported by some evidence from older case series [12,13]. In one series of patients with proven RAS, 41% of patients with bilateral disease had a history of pulmonar edema, and following stenting, 77% of these patients had no further episodes of

pulmonary edema [14]. A recent systematic review included data from 173 patients and concluded that the quality of evidence was low and justified only a weak recommendation for Percutaneous Transluminal Renal Artery Angioplasty (PTRAA) with or without stenting in patients with atherosclerotic RAS and either flash pulmonar edema or chronic heart failure and renal insufficiency [15]. The interpretation of the results of the large trials so far in the management of atherosclerotic RAS has been limited by patient selection and methodological issues including crossover from medical to intervention arms and analysis based on intention to treat (when not all randomized patients actually underwent a procedure).

Conclusion

Well-selected patients with moderate renal arterial stenosis and clinical features of renovascular hypertension may benefit from percutaneous transluminal renal artery angioplasty for the treatment of atherosclerotic RAS.

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