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Case Report

Renal artery stenosis complicated by an intraoperative rupture of renal artery: a case study

Hardik Bakulkumar Mevawala*, Jash Kamlesh Shah, Yugam Rajeshkumar Oza, Shrinath Vasantbhai Gondalia, Nancy Mahesh Gor

Grodno State Medical University, Grodno, Belarus

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***Correspondence:**
Dr. Hardik Bakulkumar Mevawala,
E-mail: mevawalahardik@gmail.com

ABSTRACT

Renal artery stenosis (RAS) is a major contributor to the prevalence of secondary hypertension. Fibromuscular dysplasia and atherosclerosis are commonly responsible for the occurrence of the disease. Medical therapy is the primary means of treatment for RAS. However, surgical interventions for revascularization are also considered, in selected group of patients, which can effectively cure hypertension and chronic kidney disease. An older man presented at Venus hospital, Surat, Gujarat with the complaints of severe dyspnea, edema, uncontrolled hypertension and renal insufficiency. He was diagnosed RAS and was operatively managed with percutaneous transluminal renal angioplasty. Written consent was taken from the patient mentioned in the study. During the procedure, the renal artery got ruptured, which was managed by placing a covered stent. The patient was successfully treated for RAS, in spite of comorbidities and intraoperative complication. In the subsequent clinical follow-up, the patient was asymptomatic. There was marked reduction in serum creatinine levels and even the blood pressure improved significantly. Absence of post-operative complications and positive recovery of the patient signifies the fact that management of renal artery rupture with a covered stent is a convenient approach in acquiring effective haemostasis. This approach can be useful in managing any sort of vessel rupture, related to revascularization procedures.

Keywords: RAS, Percutaneous transluminal angioplasty, Covered stent, Hypertension, Renal artery rupture, Case-study

INTRODUCTION

A reduction in blood supply to the kidney, due to occlusion or narrowing of renal arteries, is known as RAS. There are multiple possible causes of this occlusion, which can be atherosclerosis, thromboembolism, vasculitis and fibromuscular dysplasia. It can occur unilaterally as well as bilaterally. For acute occlusion of the renal artery, thromboembolism is the most common cause. For chronic RAS, atherosclerosis is the most common cause, which usually occurs bilaterally. Fibromuscular dysplasia is less prevalent than atherosclerosis is seen unilaterally, in distal part of renal artery.[1,2]

In patients with RAS, decreased kidney perfusion leads to activation of renin-angiotensin-aldosterone-system (RAAS) which performs a major role in regulating the blood pressure. In short term, RAAS acts protective in nature in patients with RAS. But in the long term, in order to provide sufficient perfusion to the kidneys, RAAS can increase blood pressure via volume expansion and can cause hypertensive nephrosclerosis, renal fibrosis and glomerulosclerosis.[3,4]

RAS has a diverse presentation, from the patient being asymptomatic to acute end organ damage like flash pulmonary edema, aortic dissection, stroke, transient ischemic attack, hypertensive heart failure and acute

coronary syndrome. Chronic and progressive stenosis can present with hypertension at varying age, that can be less than 30 or after 50.[4,5] There can be presence of an abdominal bruit heard over the flank on physical examination of the patient.[3] Signs and symptoms of atherosclerosis (e.g., coronary artery disease, Peripheral artery disease, carotid artery stenosis) might also be present.[4]

Acute onset worsening hypertension which was controlled previously, worsening hypertension despite using three antihypertensive agents of different groups, hypertensive emergency and acute onset of unexplained pulmonary edema are some indications to investigate for RAS. For diagnosing RAS, laboratory investigations only act as a supportive evidence. Instrumental and imaging techniques are necessary to confirm its diagnosis.[6] Duplex sonography and MR angiography are the first-line investigation of choice. Catheter angiography is the gold standard for diagnosing RAS.[6,7]

Whether the patient is symptomatic or asymptomatic, medical therapy to control hypertension should be initiated in patients with RAS. Lifestyle modification, statin therapy, glycemic control and antiplatelet therapy might help along with antihypertensive drugs. Percutaneous transluminal balloon angioplasty with stenting, aortorenal bypass and endarterectomy are the surgical treatment options for treating RAS.[1,4,5]

Herein, we have described a case of an elderly man who underwent percutaneous transluminal renal angioplasty of left renal artery for treating RAS, which was complicated by an intraoperative renal artery rupture.

CASE REPORT

A 75-year-old male patient with history of diabetes mellitus, hypertension, chronic kidney disease, coronary artery bypass graft surgery performed 8 years ago presented at Venus hospital, Surat, Gujarat with complaints of severe breathlessness since half an hour and generalized edema.

The patient was conscious with temperature in a normal range, pulse 90/min, respiration rate 18 bpm, blood pressure 190/90 mmHg, SpO_2 80% and random blood glucose 256 mg/dL. A 2D ECHO was done which showed mild ischemic heart disease, mild left ventricular systolic dysfunction and pulmonary hypertension. Pulmonary edema was seen on X-ray. Serum creatinine was 2.44 mg/dL, pH 7.29 and lactate 4.1 mg/dL.

The patient was diagnosed with accelerated hypertension with left ventricular failure and trifascicular block. He was treated with furosemide (40 mg/4ml), $NaHCO_3$ (8.4%), heparin (5000 IU/ml), nitroglycerine (25 mg/5 ml) and was stabilized via BiPap. Medical therapy with Prazosin (5 mg), telmisartan (40 mg), aspirin (75 mg), clopidogrel (75 mg), atorvastatin (10 mg), metolazone (2.5 mg), cefexime (400 mg), clonidine (100 mcg), febuxostat (40 mg) was provided over the next four days and the patient was discharged.

The next day after discharge, patient came with the complaint of vomiting. On examination, the blood pressure was 200/110 mm Hg, pulse 50/min, respiratory rate 18 bpm, SpO_2 98%, random blood glucose 375 mg/dL, serum creatinine-2.6 mg/dL and bilateral wheezing was present on chest auscultation.

So, he was readmitted with the diagnosis of hypertensive urgency and was treated with nitroglycerin (3 ml/hr), $NaHCO_3$ (8.4%), amlodipine (5 mg), clonidine (100 mcg), furosemide (40 mg), tolvaptan (15 mg), aspirin (75 mg), clopidogrel (75 mg), atorvastatin (10 mg), metoprolol (12.5 mg), nicardipine (20 mg), prazosin (5 mg) and moxonidine (0.2mg) were added over the following next six days. On the fourth day, serum creatinine was 2.82 mg/dL and sodium 129 mEq/L. The next day a nephrologist was consulted who suggested undergoing renal Doppler ultrasound, which showed bilateral RAS and renal parenchymal abnormalities. For further confirmation, MR angiography of renal artery was done which showed 90% stenosis in left renal artery.

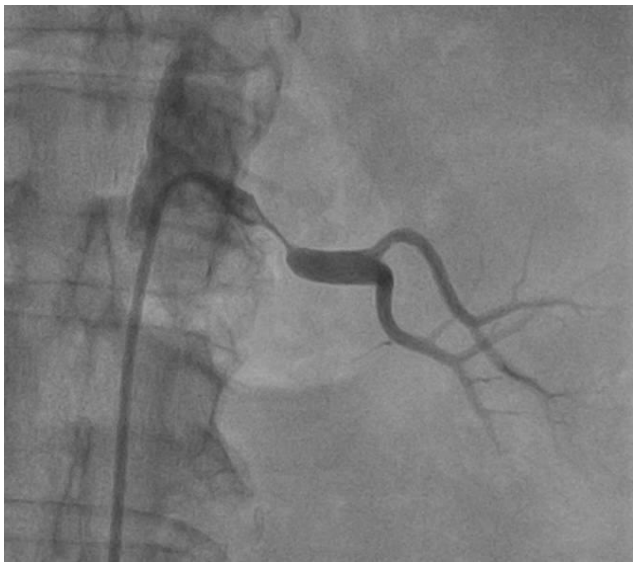

Figure 1: Angiography of left RAS before performing revascularization.

Multiple calculi in common bile duct (CBD) up to 8 mm with dilation of CBD and few small gall bladder calculi. Based on the results of MRA, Percutaneous transluminal renal angioplasty was suggested. Before the procedure, serum creatinine was 2.72 mg/dL.

Percutaneous transluminal renal angioplasty of left renal artery through right femoral arterial route was done with 6x15 mm stent at 16 at ATM pressure. As the lesion was very fibrotic in nature, higher than normal pressure was applied to open the lesion, which led to arterial rupture due to high pressure dilation.

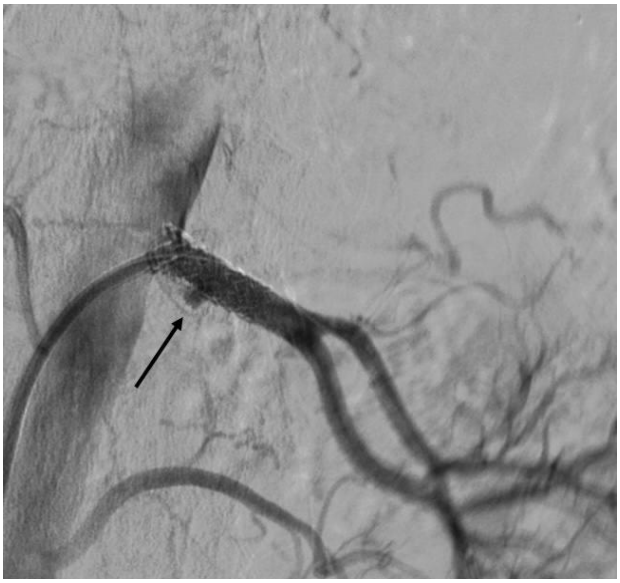

Figure 2: Extravasation of contrast (arrow) from the main renal artery, indicating the rupture of renal artery in the inferior part.

This was associated with severe pain in the left flank region. Immediately, a balloon was inflated to stop the bleeding. The damage was inspected on the monitor to locate the ruptured site, which was in the inferior part of the vessel. A covered stent was needed for cessation of bleeding and was ordered from another regional hospital as it wasn't available. It was decided to keep the balloon inflated for 10 minutes. Heparin was reversed with protamine sulfate. Roughly after 45 minutes, a covered stent of 4.5x19 mm was at hand and was introduced into the vessel at 20 atm pressure, followed by its placement on the ruptured area.

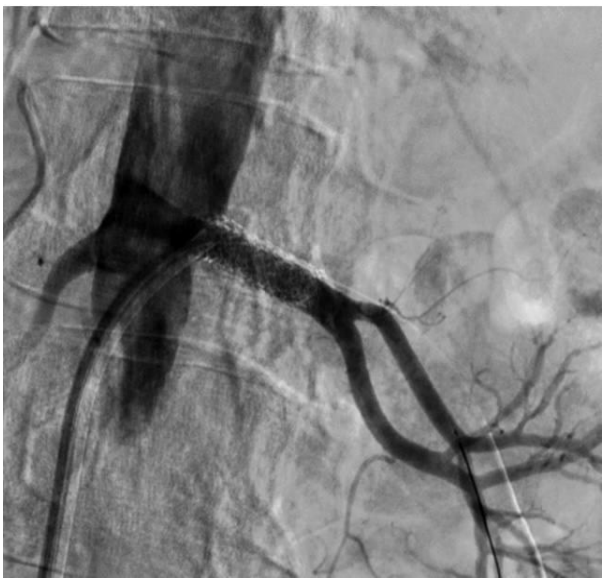

Figure 3: Left renal artery angiogram, obtained after placement of a covered stent showing no sign of continued extravasation.

By the time a covered stent was available, the procedure of inflating the balloon for 10-minutes, deflating it and checking the patency of ruptured vessel was repeated 4 times. The rupture didn't heal by primary and secondary haemostasis. On completion of the surgery, 100 ml of contrast non-ionic dye was used.

After the procedure, the patient was sifted to ICU for monitoring. Ryle's tube and foley's catheter were inserted. The patient still complained of left flank pain and vomiting. Pain resolved within 3-4 hours, but vomiting continued. Next day acetone levels were 40 mg/dL, serum creatinine 3 mg/dL, uric acid 9.3 mg/dL and lactate 1.8 mg/dL. Treatment with dextrose and normal saline (DNS) + insulin IV was started. Over the next three days serum creatinine levels were 1.5 mg/dL, 2.7 mg/dL and 2.8 mg/dL respectively.

DISCUSSION

The narrowing of renal arteries is called as RAS, which can occur unilaterally or bilaterally. Fibromuscular dysplasia, atherosclerosis and vasculitis are the common causes of RAS. Fibromuscular dysplasia is the underlying cause commonly seen in young women. Whereas, in elderly patients, atherosclerosis is the cause in 90% of the cases and this is called Atherosclerotic RAS (ARAS). Vasculitis as a cause of RAS is seen in <1% of all the patients with RAS.[1] The involvement of renin-angiotensin-aldosterone system (RAAS) is the major contributor of renovascular hypertension and nephropathy.[2] This patient had chronic hypertension and chronic kidney disease. The patient ultimately presented with hypertensive urgency, hypertensive left ventricular heart failure and flash pulmonary edema. He was given amlodipine, clonidine, furosemide, metoprolol, nicardipine, prazosin and moxonidine right away and the patient was kept under supervision. There wasn't any notable improvement in blood pressure with these antihypertensive agents even after four days, which led to a suspicion of a secondary cause of hypertension that can be RAS.

Non-invasive method for evaluating RAS are assessing the overall functioning of the kidney and RAAS, perfusion studies and captopril renography. But in case of elderly patients these tests are non-conclusive.[8,9] As there is a limited use of these non-invasive studies for diagnosing ARAS in elderly patients, imaging techniques are more preferable. Imaging and renal blood-flow velocity assessment can be provided by duplex ultrasonography. Whereas, renal circulation and aorta can be evaluated by Gadolinium-enhanced MRI and CT angiography.[7,10,11] Doppler ultrasonography was performed, which showed bilateral RAS with renal parenchymal abnormalities. MR angiography confirmed the diagnosis, revealing 90% stenosis of the left renal artery.

As per the standard guidelines, ARAS with multi-drug resistant malignant hypertension and flash pulmonary edema, denotes class 1 indication for stenting the renal arteries. If the patient have ARAS + unstable angina + progressive renal dysfunction + resistant hypertension, it is categorized into class 2A indication for artery stenting and class 2B category includes the patients who have unilateral/bilateral ARAS or CKD with unilateral ARAS.[12] Renal artery stenting was a class 1 indication in our patient, percutaneous transluminal angioplasty (PTA) of left renal artery was decided to carry out.

Renal angioplasty was performed via right femoral artery with 6x15 mm stent. Radial artery access is preferred, in order to avoid vascular complications and bleeding, which are seen commonly in femoral artery access. Though, the decision of the access site is made according to the preferences of the physician and individualized by the anatomical orientation of renal arteries of the patient.[1] No complications like femoral site hematoma/aneurysm related to stent insertion site were seen in patient.

Procedural renovascular complications like embolization, perforation or rupture, branch occlusion, occlusive dissection, subcapsular hematoma, renal infraction or complications in which nephrectomy is required are possible but are very rare.[13] In this man, due to fibrotic nature of the lesion higher than normal pressure was applied to re-perfuse the kidney, which led to inferior wall rupture of the left renal artery. Extravasation of the contrast was visible on the angiogram. The rupture was acutely managed by balloon tamponade. The patient underwent multiple procedures of 10-minute balloon inflation to minimize the blood loss, and in the meantime a covered stent was made available. A case similar to this patient was witnessed in a retrospective study performed by Morris et al. which demonstrated 308 PTA or stent placement procedures, out of which 13 patients sustained injury to the renal artery and five patients developed renal artery rupture. Out of these five patients, one patient was managed by 10-minute balloon inflation procedure followed by stent-graft deployment, after which no contrast extravasation was visible.[14] Similarly, we also obtained hemostasis, as soon as the covered stent of 4.5×19 mm was introduced.

There was intra-operative and post-operative flank pain noted which resolved 3-4 hours after the operation was completed. During the whole procedure, 100ml non-ionic dye was used. Contrast induced nephropathy is one of the complication and can be defined as 25% or more rise in serum creatinine levels, after contrast exposure compared to pre-operative creatinine levels.[15] The pre- and post-operative serum creatinine levels in this elderly man were 2.82 mg/dL and 3 mg/dL indicating an absence of contrast induced nephropathy.

Aortorenal bypass can be carried out instead of stent placement as another treatment option in patient with ARAS. Dua et al. compared the inpatient mortality between aortorenal bypass and renal artery stenting, which was 2.5% and 0.6% ($p<0.01$). 23.5% of the patients, who survived, faced complications in case of aortorenal bypass. Whereas the complication rate in stenting procedure was 47.4%.[16] Although, stenting showed higher complications, the mortality rate was lower compared to aortorenal bypass.

In this case, there was absence of any postoperative complications. Over the next three days serum creatinine levels decreased gradually and on the follow-up after seven days it was found to be 1.37 mg/dL which was the lowest level noted since past eight years. On follow-up, the blood pressure was 131/82 mm hg, 137/80 mm/hg and 129/85 mm hg.

On the basis of our study, we can say that, placing a covered stent is an efficacious method to control the bleeding and stabilizing the patient. But, PTA is not free of complications, though proven successful for this patient. We propose that, a provision of a covered stent should be made preoperatively for all kinds of stenting procedures, which can reduce mortality, if the procedure is complicated by vessel rupture. The major step in an effective renal revascularization is meticulous selection of the candidate after performing conscientious examination. Therefore, well-designed trials are necessary, for identifying the specific group of population which will be most likely benefited by this intervention, in order to reduce the complications related to the procedure.

CONCLUSION

Renal artery angioplasty is an effective method to treat hypertension caused by RAS and to regain the kidney function. Covered stent placement can effectively control bleeding, if angioplasty is complicated by rupture of the renal artery. Therefore, in order to bring haemostasis in any sort of revascularization procedures, covered stent placement might be an effective method for which further studies are needed.

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