

# Bilateral Renal Arterial Embolisation in a Patient with Mitral Stenosis and Atrial Fibrillation: an Uncommon Reason of Flank Pain

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## Introduction

Thromboembolism is a well known complication of mitral stenosis especially when it is associated with atrial fibrillation (1). But renal embolization generally remains an undiagnosed clinical condition because renal function disorders are only noticed when patients have bilaterally affected arteries, previous chronic renal insufficiency or involvement of a solitary functioning kidney (2). In this article we report a case of bilateral renal embolisation in a patient with mitral stenosis and atrial fibrillation with spontaneous recovery of renal function and treated with balloon valvuloplasty.

## Case

A 31-year-old male was admitted to our hospital with bilateral flank pain and hemoptysis for the last one week. Physical examination showed a normal arterial blood pressure, an increased intensity of the first heart sound, a diastolic mitral rumble, a mitral opening snap supporting the diagnosis of mitral stenosis. He had an atrial fibrillation with a normal ventricular response, and his telecardiogram was consistent with mitral stenosis and pulmonary hypertension. On his complete blood count, WBC was 19100/mm<sup>3</sup>, Htc was 39%. The sedimentation rate was 60 for 30 minutes and 80 for 1 hour. His biochemical examination showed that: SGOT: 102 U/L, SGPT: 81 U/L, LDH:

4351 U/L, CK-MB: 36 U/L, total bilirubine: 2,16 mg/dl, BUN: 30 mg/dl, creatinine: 1,54 mg/dl. On urine specimen there was a macroscopic hematuria, density was 1025, protein 150mg/dl, erythrocyte 250 /microL and sediment contained large numbers of RBC's and WBC's. Abdominal ultrasound performed in the emergency room, was not diagnostic. A contrast-enhanced CT scan showed that there was a clear perfusion difference with a demarcation line especially on the ventral ramus of the right renal artery. Another "wedge-shaped" opacification defect was seen on the upper posterior pole of the left kidney (Figure 1). On renal angiograms renal embolism of the right kidney was diagnosed but perfusion of the left kidney was quite normal (Figure 2).

Patient has been hospitalized and treated with heparin infusion (1000U/hour) and aspirine (1x100mg/day) for one week. Because of the late admission and relatively well preserved renal functions no thrombolytic treatment was considered. At the end of the first week, his renal functions were recovered to normal without a need for dialysis. On echocardiogram, his mitral valve area was 1cm<sup>2</sup>, mean gradient was 11mmHg with a minimal mitral regurgitation, echocardiographic score was considered as 7 and left ventricular functions were normal. On transesophageal echocardiogram, there was a severe spontaneous echo contrast in the left atrium but no thrombi or vegetation were observed. On his cardiac catheterisation, systolic pulmonary artery pressure was 58 mmHg, and 14mmHg of mitral gradient was measured. A mitral balloon valvuloplasty was performed with Inoue technique by using a 28 size balloon. Echocardiographic controls showed a successful dilatation without any complication. After this procedure,

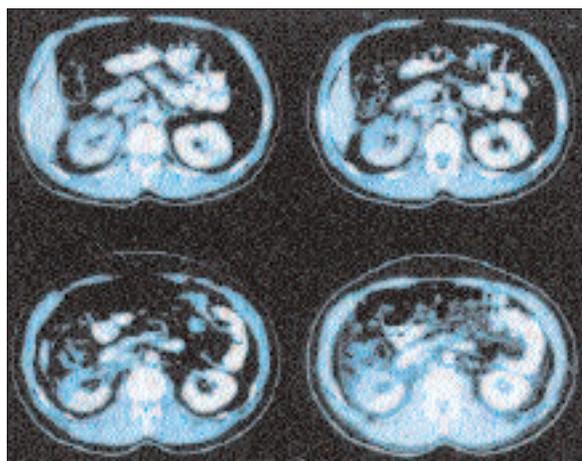
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the patient was discharged on coumadine 1x 2,5mg and aspirine 1x100 mg.

## Discussion

The first diagnosis of renal infarction is attributed to Traube, in 1856 (3). The major causes of renal infarction include emboli secondary to cardiac disease like valvular heart disease, atherosclerosis, myocardial infarction, ventricular or interatrial septal aneurysms, bacterial endocarditis, heart tumours, and dilated cardiomyopathy. Particularly, atrial fibrillation either rheumatic or nonrheumatic increases the risk of embolic phenomena. Because of the renal arteries are end-arteries, acute occlusion always results in infarction. Other aetiological factors for thromboembolism include trauma, polycythemia vera, fibromuscular dysplasia, extraadrenal pheochromocytoma, dissection of aorta or renal artery. Moreover, renal infarctions have been reported in patients with connective tissue diseases as, systemic lupus erythematosus, primary antiphospholipid antibody syndrome, polyarteritis nodosa, systemic vasculitis, mixed connective tissue disease and Behcet's disease (3).

Patients with acute renal infarction commonly present with persistent abdominal/flank/lower back pain. Most patients have a history associated with a high risk of thromboembolism. Within 24 hours after onset of symptoms, most patients may show elevated serum levels of LDH and frequently they will have hematuria (4).

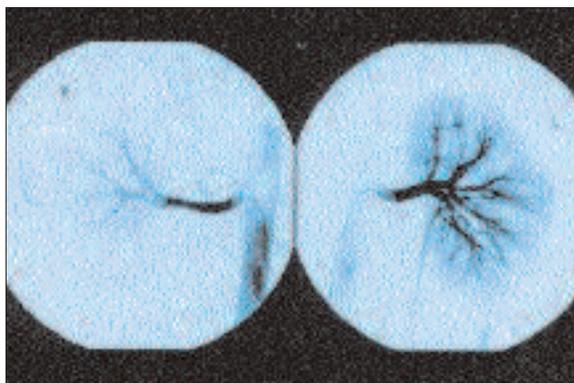


**Figure 1:** A clear perfusion difference with a demarcation line especially on the ventral ramus of the right renal artery can be seen in the right kidney. A "wedge-shaped" opacification defect was seen at the upper posterior pole of the left kidney.

Acute mesenteric thromboembolism must be considered in the differential diagnosis. Sometimes it can be together with renal embolism and generally it produces the acute abdomen symptoms.

In the literature, there are cases of spontaneous recovery (2,5) or successful late embolectomies after 43 days from onset of symptoms (6). Return of renal function in humans was believed to be successful when revascularization occurred before 90 min (7) to 18 h after onset of symptoms (8). Although encouraging results with intra-arterial fibrinolytics, complications may include uncontrolled bleeding, pseudoaneurysms, distal embolization, pericatheter thrombosis, allergic reactions, and strokes (2). In our case, because of the hemoptysis, late admission, bilateral but partial renal infarction and relatively well preserved renal functions, we did not consider to give any thrombolytics. Because of the suitable conditions of the mitral valve and no evidence of intracardiac trombi and vegetations, balloon valvuloplasty was the preferred treatment for mitral stenosis. After the first month, the patient was doing well with oral anticoagulation, renal functions and biochemical parameters were within normal limits.

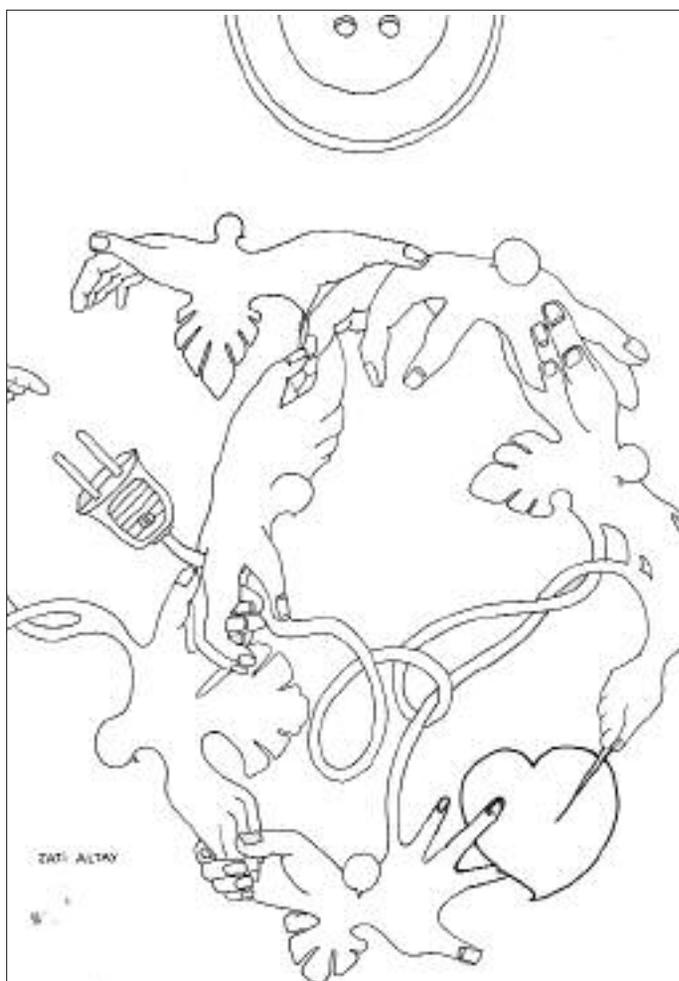
Hall gave a good definition of acute renal vascular occlusion, calling it an "uncommon mimic" (9). All patients seen for persistent or sudden abdominal/flank/lower back pain with history of cardiovascular and/or connective tissue diseases should be considered at high risk for renal vascular occlusion. If the patient has an elevated LDH and hematuria, an abdominal contrast-enhanced CT scan should be performed as soon as possible.



**Figure 2:** Selective renal angiograms show a clear difference of perfusion and severe intrarenal occlusion in the right kidney. In spite of a defect seen on CT scan, left kidney vascularity and perfusion were quite normal.

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