

Post-coronary stenting renal cholesterol embolism treated by renal artery stenting

Koroner stent uygulaması sonrası gelişen renal kolesterol embolisinin renal stent uygulaması ile tedavisi

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Introduction

Cholesterol crystal's embolism (CCE) is an important yet underdiagnosed medical problem resulting from shedding of cholesterol crystals from eroded atherosclerotic plaques of the aorta and/or large feeder arteries into the small vessels of multiple organs. We report the case of a 56-year-old woman who following coronary angioplasty with stenting developed acute atheroembolic renal infarction successfully treated by renal artery stenting.

Case report

A 54-year-old woman was referred to our hospital for coronary angiography due to typical angina pectoris. The results of the physical examination and routine blood tests were unremarkable. Coronary angiography showed a significant proximal right coronary artery stenosis and percutaneous transluminal coronary angioplasty with stenting was successfully performed. Ten minutes after the procedure the patient experienced sudden-onset of severe back and left flank pain accompanied by nausea and vomiting. The abdominal examination revealed moderate to severe tenderness, localized mainly over the left upper quadrant. Her lower extremities were well perfused with no ischemic or gangrenous lesions. At first, we believed that the patient's initial symptoms were probably related to retroperitoneal hemorrhage due to severe back pain, and emergency abdominal computer tomography angiogram was planned, which revealed no retroperitoneal hemorrhage, but acute renal infarction in lower pole of the left kidney (Fig. 1). Repeated laboratory studies showed that the elevated levels of C-reactive protein (292 mg per liter), lactate dehydrogenase (from 338 to 2125 IU/L), aspartate aminotransferase (from 22 to 120 IU/L), alanine aminotransferase (from 30 to 110 IU/L), creatine kinase (from 74 to 300

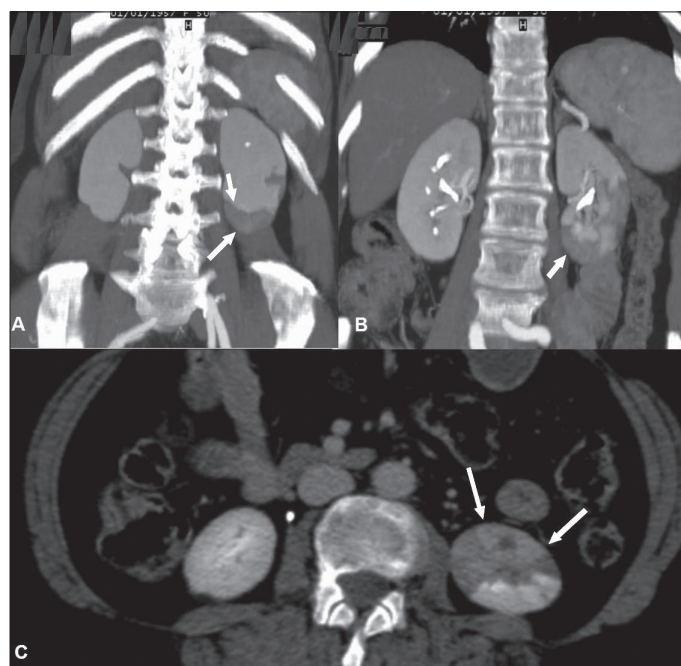


Figure 1. An abdominal CT angiography view of well demarcated areas of cortical hypoperfusion representing infarction (arrows) in the lower pole of left kidney

IU/l), D-Dimer (0.46 to 2.18 ug/dl), but normal levels of blood urea nitrogen (9.4 mg/dl) and creatinine (0.60 mg/dl). Blood testing showed no evidence of eosinophilia. Since she continued to have severe left flank and back pain in spite of iv high doses of narcotic analgesics, we decided to perform renal angiography. Selective left renal angiogram showed two left renal arteries arising from the aorta. The main left renal artery angiograms showed normal perfusion of upper and middle pole, but no perfusion to the lower pole (Fig. 2A, 2B). The selective

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catheterization of the accessory lower pole renal artery showed a cut-off of perfusion to lower pole (Fig. 2C). The vascular surgery team discounted surgery as an option because renal function was not compromised and limited perfusion defect to lower lobe; and the percutaneous transluminal renal angioplasty performed (Fig. 2D, 2E), and provided lower lobe perfusion despite residual filling defect (Fig. 2F). Immediately afterwards, her flank pain dramatically resolved and the procedure was stopped. Five hours after renal angioplasty, the patient's severe left flank pain and nausea repeated, and she underwent angiography laboratory again. The selective angiogram of the accessory lower pole renal artery showed diminished perfusion due to bifurcational filling defect (Fig. 3A), which was managed successfully with percutaneous transluminal renal angioplasty and stenting (2.5x18mm) (Fig. 3B, 3C, 3D). Her symptom's dramatically improved again. She was discharged from hospital 2 days after the procedure without renal impairment and has been uneventful for 2 months of the follow-up.

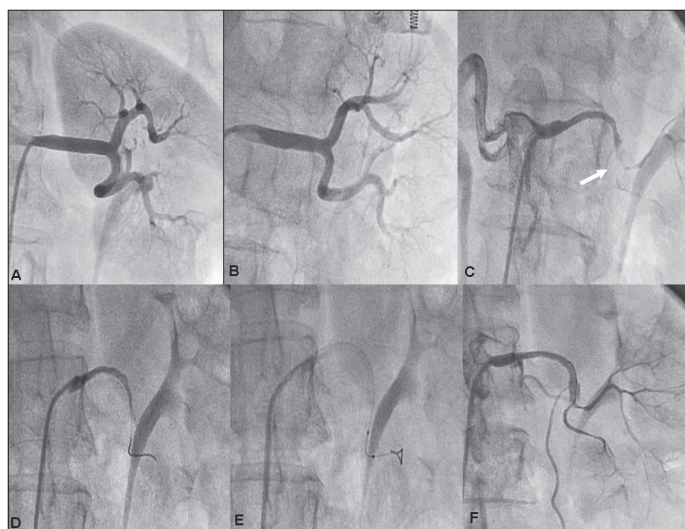


Figure 2. Selective left renal angiogram. Panel A and B show selective the main left renal angiogram with normal perfusion. Panel C-E show percutaneous angioplasty of the accessory lower pole renal artery occlusion

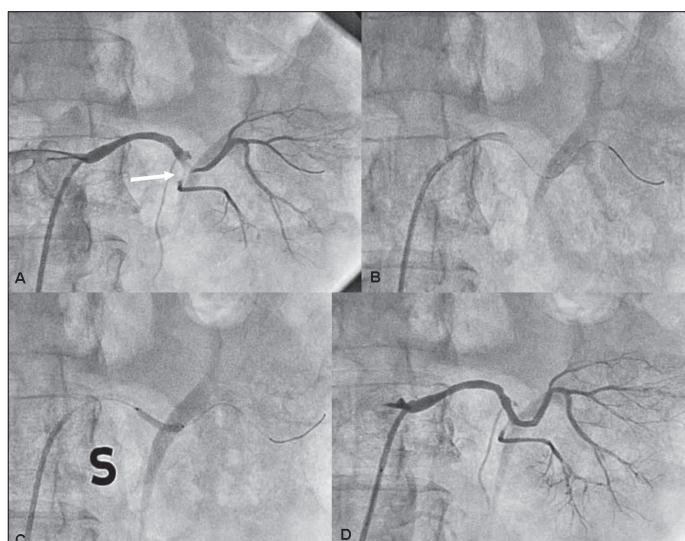


Figure 3. Repeated selective left accessory lower pole renal angiogram shows succesful recanalisation with stenting

Discussion

Recent studies showed that this entity is not a rare event in everyday clinical practice, and requires a high index of suspicion (1). The presence of a classic triad characterized by a precipitating event, acute or subacute renal failure, and peripheral cholesterol crystal embolization in skin/retina strongly suggests the diagnosis (2, 3). It is increasingly recognized as a serious complication from invasive cardiovascular procedures, such as manipulation of the aorta during angiography or vascular surgery, or after anticoagulant and fibrinolytic therapy. Because of proximity of the renal arteries to abdominal aorta and it receive an enormous amount of blood flows, the kidney is a frequent target organ (3). Renal infarction, although rare in the setting of atheroembolization, typically leads to an increase in serum aspartate aminotransferase and lactate dehydrogenase levels (4).

The aim of treatment should be to halt the progression of tissue ischemia and prevent repeated showers of renal cholesterol crystal embolism. There are currently no effective treatments for atheroembolism; symptomatic and preventive measures have been advocated.

Various treatment strategies have been pursued including with low-density lipoprotein apheresis, statins, pentoxifylline, corticosteroids and cyclophosphamide (2, v5-7). Supportive measures include angiotensin blockade for predialysis patients and hemodialysis support for those who reach end-stage renal disease. Other important preventive measures include, secondary prevention of atherosclerotic risk factors, avoiding anticoagulant therapy, forbidding any new radiologic and/or vascular surgery procedure (8). The possibility of using a brachial approach for aortogram or coronary angiogram to prevent atheroembolism needs to be considered (1). In presented case, patient's intractable pain resolved only percutaneous renal revascularization with stenting, and this should be considered in patients with intractable pain.

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