CASE REPORT

Acute renal artery embolisation: role of local catheter-based intra-arterial thrombolysis

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SUMMARY

A 45-year-old man without previous comorbidity presented to us with acute onset right-sided flank pain for last 14 hours. His general physical and systemic examination was unremarkable, and there were no clinical signs of peritonitis. The ultrasonography did not reveal any evidence of nephrolithiasis or hydronephrosis. His contrast-enhanced CT scan revealed hypoattenuated areas of right kidney and evidence of right renal artery thrombosis. He was immediately shifted to cardiac catheterisation lab, and his renal angiography showed thrombotic occlusion of right renal artery. The bolus dose of streptokinase (250 000 IU) was given locally in renal artery by right judkins catheter followed by systemic infusion of streptokinase (100 000 IU/hour) for 24 hours. After that he was started on low molecular weight heparin. Repeat renal angiography done after 5 days showed completely normal right renal artery. His cardiac and thrombophilia work up was negative, and he was discharged on antiplatelets, oral anticoagulants and statins.

BACKGROUND

Renal artery thrombosis is rare cause for acute abdomen. The largest case series of emergency department patients gives a prevalence of about 2/100 000.¹ High degree of clinical suspicion, early diagnosis and emergent treatment prevent the loss of renal functions and development of hypertension.² The contemporary data on treatment options for the renal artery thrombosis is scanty. This report represents successful outcome in renal artery thrombosis by use of local intra-arterial thrombolysis followed by systemic thrombolysis with streptokinase.

CASE PRESENTATION

A 45-year-old man presented to emergency department with acute onset right-sided flank pain that started 14 hours prior to admission. The pain was severe, continuous, non-radiating and without any aggravating or relieving factors. Except for smoking, he has no other atherosclerotic risk factors. There was no history of fever, vomiting, jaundice, constipation, diarrhoea, burning micturition, haematuria, trauma, drug intake, alcohol intake, malena and weight loss. He underwent coronary angiography through right femoral artery approach 4 days prior to onset of symptoms in some local private hospital for atypical angina, which revealed normal epicardial coronaries. Physical examination

showed a temperature of 37.4°C, blood pressure of 132/78 mm Hg, pulse 87/min regular good volume; respiratory rate of 14/min and oxygen saturation was 95%. His cardiac and respiratory examination was unremarkable. The abdomen was soft, not distended, moving normally with respiration, no tenderness, no guarding or rigidity, no organomegaly, no free fluid in abdomen and normal bowel sounds. His urine output was maintained.

INVESTIGATIONS

His blood investigations revealed normal: haemogram, blood sugar, serum lipase, serum amylase levels, liver functions, urine examination, lipid profile and ECG. His serum lactate dehydrogenase (LDH) was >1200 (<247 IU). His renal parameters at time of admission showed blood urea nitrogen (BUN) of 8 (8–20 mg/dL) and serum creatinine of 1.2 (0.67–1.17 mg/dL). X-ray abdomen standing and ultrasound abdomen were normal. In view of severe abdominal pain in absence of signs of peritonitis, the possibility of mesenteric ischaemia was kept, and contrast-enhanced CT abdomen along with CT angiography of abdominal vessels were ordered, which showed area of non-enhancement in right kidney (figure 1A) and multiple calcified atherosclerotic plaques in abdominal aorta and no flow in right renal artery (figure 1B).

DIFFERENTIAL DIAGNOSIS

After ruling out all the common causes for acute abdomen and based on the raised serum LDH levels and CT findings, the final diagnosis of renal artery thrombosis was made. His thrombophilia work up was negative. The transthoracic echocardiography did not reveal any valvular heart disease or thrombus in left-sided chambers. The cause for thrombosis at abnormal site was thought to be due to embolisation of cholesterol emboli released during cardiac angiography, which was done 4 days prior to the onset of symptoms and which further leads to thrombus formation in renal artery.

TREATMENT

He was immediately shifted to cardiac catheterisation lab for selective renal angiography. The left-sided renal angiography was normal (video 1), and right renal angiography showed thrombotic occlusion of renal artery (figure 2A) (video 2). The streptokinase (250 000 IU) was given directly into right renal artery with help of judkins right catheter. After that, systemic thrombolysis was done with

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Figure 1 CT scan images. (A) Coronal section showing multiple areas of non-enhancement on right-sided kidney. (B) Abdominal aorta showing multiple calcified plaques (blue arrow) and cut-off right renal artery.

streptokinase (100 000 IU/hour) for 24 hours. Although his pain was relieved, but there was worsening of renal parameters on second day. On second day, he had BUN of 15 (8–20 mg/dL) and serum creatinine of 1.89 (0.67–1.17 mg/dL). Then he was started on low molecular weight heparin.

OUTCOME

After 4 days of anticoagulation, his renal functions were normalised. The check angiography showed normal patency in right renal artery with no evidence of thrombosis or atherosclerotic narrowing in it (figure 2B) (video 3). The renal scintigraphy confirmed the complete patency of the right renal artery and the

regular function of the kidney. He was discharged on warfarin, ecosprin and rosuvastatin.

DISCUSSION

The renal artery thrombosis is rare disease with life-threatening outcome in the form of renal infarction. The autopsy studies reported the prevalence of 14 per 1000.³ The incidence of renal artery thrombosis based on emergency department admissions was 0.004%–0.007%.^{4,5} Atrial fibrillation has strong association with the renal artery thrombosis and is responsible for 64% of the cases.⁶ The overall rate of systemic embolic events was 11% in the patients of non-valvular atrial fibrillations.⁷ The systemic embolic event rate involving kidney was 6%.⁷ According to Virchow triad the vessel wall injury, blood hypercoagulability and stasis of blood are the precursors of thrombosis at any unusual site. So hypercoagulable states, trauma to renal vessels by any endovascular therapy, renal artery dissection and atherosclerotic renal artery narrowing or vasculitic involvement of vessel wall are the factors responsible for the renal artery thrombosis. In observational study of the 27 patients of renal artery thrombosis, 16 (59%) of the patients were idiopathic.⁸

The clinical presentation includes acute onset flank pain, haematuria, absence of signs of peritonitis, increased creatinine and increased levels of serum LDH.⁹ The serum LDH, although not specific, is found in 99% of cases of renal infarction.^{2,6} Contrast-enhanced CT is the diagnostic test of choice in this condition as demonstrated in our case.

There are no available guidelines for the treatment of this rare entity. Either the systemic anticoagulation or the percutaneous interventions including local thrombolysis,^{10–13} balloon angioplasty and stent placement are the available options. Thrombolytic agents used in different reports were urokinase, alteplase and streptokinase. The window period from the onset of symptoms to onset of irreversible renal injury was 3 hours.¹⁴ However, in catheter-based thrombolysis, as in our case and in the case series of four patients¹⁵ reported successful perfusion of renal arteries in 14 hours and 24 hours, respectively.

Video 1 Left-sided renal angiography showing normal patency of left renal artery.

Figure 2 Renal angiography. (A) Right renal angiography showed thrombotic occlusion of renal artery. (B) Check angiography after completion of thrombolysis revealed normal flow in right renal artery.

There is no randomised control trial comparing systemic anticoagulation, catheter-directed thrombolysis and renal artery stenting. In dogs, renal ischaemia for 2 hours was followed by gradual recovery of renal function over 2–3 weeks. Ischaemia for 4 hours caused irreversible renal damage.¹⁶ However, there are case reports about the recovery of kidney function even after weeks of total occlusion.¹⁷ In another report, recovery of renal functions was seen with renal artery stenting done after 2 weeks of thromboembolic occlusion of renal artery.¹⁸ Another report showed unsuccessful outcome after renal artery stenting, done

after 7 days of symptom onset in the transplanted kidney.¹⁹ Systemic anticoagulation with enoxaparin started after 5 hours, resulted in a successful outcome in a patient of oral contraceptive-induced renal artery thrombosis.²⁰

The viability of kidney after a prolonged period of ischaemia depends on collaterals from lumbar, suprarenal and ureteral vessels²¹ and also the degree of obstruction, as subtotal obstruction resulted in hibernation of renal parenchyma. Our case confirmed that early thrombolytic therapy is safe and beneficial when compared with surgery in renal artery thrombosis. By

Video 2 Right-sided renal angiography showing thrombotic occlusion of right renal artery.

Video 3 Right-sided renal angiography after thrombolysis showing normal patency of right renal artery.

giving early thrombolytic therapy, we can prevent the irreversible renal damage and subsequent nephrectomy.²²

Learning points

- In patients of acute onset severe flank pain in the absence of clinical signs of peritonitis, the possibility of renal artery thrombosis should be considered.
- The raised levels of serum LDH will be clue to the diagnosis, and contrast-enhanced CT is the diagnostic test of choice.
- Early diagnosis and urgent catheter-based thrombolysis will prevent the onset of renal infarction and any further surgical intervention.

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