

Exceptional Case

Bilateral renal artery thrombosis secondary to acute necrotizing pancreatitis

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Abstract

Renal artery thrombosis is a rare, but serious and often under-diagnosed condition. We report a case of bilateral renal artery thrombosis secondary to acute necrotizing pancreatitis. A 66-year-old female presented with abdominal pain and acute kidney injury (AKI). A renal biopsy showed organized intraluminal thrombi and a computer tomography scan of the abdomen showed bilateral renal artery thrombosis. Emergent laprotomy showed necrosed pancreas. Doppler studies showed deep vein thrombosis of the lower extremities and internal jugular vein thrombosis. Workup for hypercoagulability was unremarkable. The final diagnosis was AKI secondary to bilateral renal artery thrombosis probably due to hypercoagulability of acute necrotizing pancreatitis.

Keywords: acute kidney injury; necrotizing pancreatitis; renal artery thrombosis

Background

Renal artery thrombosis is a rare, but serious and often misdiagnosed condition [1]. It can happen due to *in situ* thrombosis or thromboembolism [1]. Common causes include atrial fibrillation, infective endocarditis, anti-phospholipid antibody syndrome and trauma [1]. Bilateral renal artery thrombosis due to acute pancreatitis is rare and only one case has been reported so far [2]. We report a case of bilateral renal artery thrombosis secondary to acute necrotizing pancreatitis.

Case report

A 66-year-old female presented with abdominal pain, nausea, vomiting and fatigue for 5 days. She also noticed decreased urine output for 4–5 days prior to admission. Other significant medical history included hypertension for 5 years and use of 800–2400 mg Ibuprofen for low back pain for the last 3 years. Her past surgical history was significant for cholecystectomy. There was no family history of hypercoagulable state.

The pertinent findings on physical examination at admission included temperature of 37°C, pulse rate of 88 beats/min (regular), blood pressure of 160/90 mmHg, tenderness to palpation in the epigastrium with no rebound, guarding or distention and 2+ pitting edema in both lower extremities. Creatinine on admission was 362 µmol/L (53–97 µmol/L). The rest of the laboratory findings are shown in Table 1. A computed tomography (CT) scan of abdomen

and pelvis without contrast revealed normal pancreas, bilateral renal cortical scarring and mesenteric edema.

Hemodialysis was initiated because of anuria and hyperkalemia on Day 4. Percutaneous biopsy of the left kidney was performed the same day (Figure 1). The biopsy revealed normal glomerulus and interstitium with no significant inflammation or sclerosis. There were four interlobular-sized muscular arteries containing intraluminal thrombi with organization, although no vasculitis was present. Based on the biopsy findings of thrombosis without inflammation, renal artery thrombosis was suspected. On Day 5, the patient had worsening of her abdominal pain and developed severe lactic acidosis. A CT scan of the abdomen/pelvis with contrast was done which showed bowel perforation and bilateral renal artery thrombosis (Figure 2). She was immediately taken for surgery. Extensive pancreatic necrosis was noted on laparotomy and therefore she underwent necrosectomy of the pancreas. Heparin was started 2 days post-operatively. A Tc-99m MAG-3 scan was also done, which confirmed renal ischemia (Figures 3 and 4). Echocardiogram did not show any evidence of thrombus or vegetation. Doppler study of the lower extremities and neck revealed thrombosis of the bilateral deep veins and internal jugular vein (attached to dialysis catheter), respectively. Work up for vasculitis including ANA, anti-DsDNA, ANCA, hepatitis panel, Antisc1-70 were negative. Work up for hypercoagulability was also unremarkable (Table 2). The final diagnosis was acute kidney injury secondary to bilateral renal artery thrombosis. The etiology of renal artery thrombosis was postulated to be due to the hypercoagulable state induced by acute necrotizing pancreatitis and proximity of the inflamed pancreas

Table 1. Serum and urine laboratory values at admission

Laboratory test	Value	Reference range
Hemoglobin	109 g/L	115–155 g/L
WBC	$10.1 \times 10^9/L$	$3.4\text{--}10.4 \times 10^9/L$
Platelets	$505 \times 10^9/L$	$150\text{--}425 \times 10^9/L$
ESR	46 mm/h	0–4 mm/h
Sodium	134 mmol/L	136–145 mmol/L
Potassium	5 mmol/L	3.5–5.1 mmol/L
BUN	12.85 mmol/L	2.5–7.1 mmol/L
Creatinine	362 $\mu\text{mol/L}$	53–97 $\mu\text{mol/L}$
Albumin	22 g/L	34–48 g/L
ALT	50 U/L	0–55 U/L
AST	35 U/L	0–34 U/L
Bilirubin	6.84 $\mu\text{mol/L}$	3.42–20.52 $\mu\text{mol/L}$
Calcium	2.2 mmol/L	2.15–2.65 mmol/L
Lactate	1.6 mmol/L	0.5–2.2 mmol/L
LDH	398 U/L	125–243 U/L
Creatine kinase	73 U/L	29–168 U/L
C3	1.52 g/L	0.83–1.93 g/L
C4	0.34 g/L	0.15–0.57 g/L
Urine analysis	Protein 100 Specific gravity 1.017 WBC 75 RBC 6 No RBC/WBC/granular cast	
Urine sodium	<20 mmol/L	

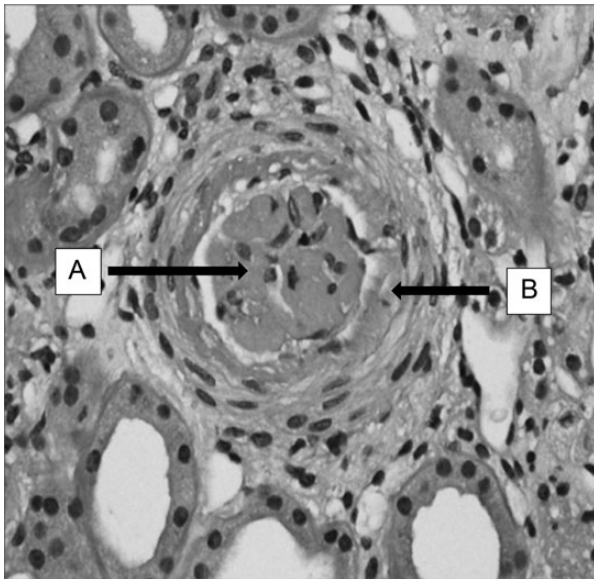


Fig. 1. H&E staining showing intraluminal thrombus with organization within inter lobular muscular arteries (A). No inflammation noted in the vessel walls (B).

to the renal arteries. There are no signs of renal recovery at 6-month follow-up and the patient continues to be on hemodialysis.

Discussion

Renal artery thrombosis was first described by von Reclinghausen in 1861 [3]. The true incidence of renal artery thrombosis is difficult to estimate because of the vague clinical presentation and rarity of the condition [4]. The common symptoms include diffuse abdominal pain, flank pain, nausea, vomiting and renin-mediated elevation in

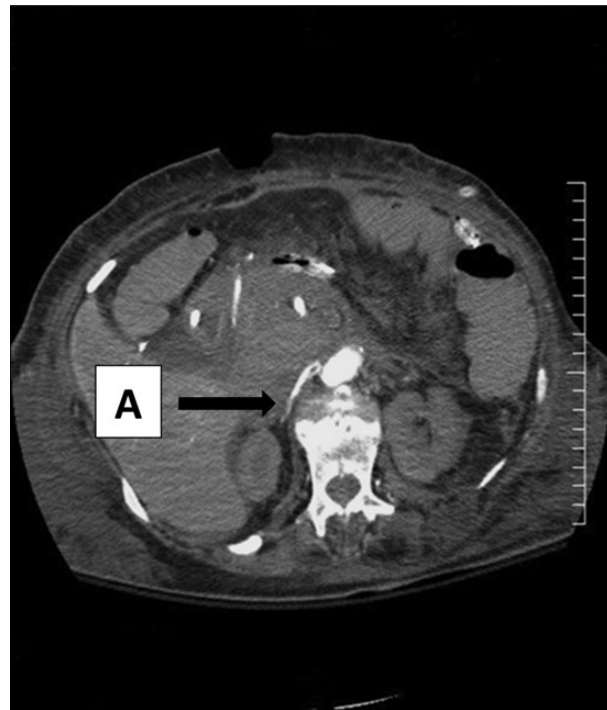


Fig. 2. Filling defect within right renal artery consistent with thrombosis (A).

blood pressure [5]. Bilateral occlusion of the renal arteries can cause anuria [5]. Elevation in creatinine can be a late feature as creatinine levels may not rise until 50% of the kidney function is lost. However, the creatinine level may not correlate with parenchymal loss [6]. Common laboratory findings include leukocytosis, proteinuria, hematuria, elevated lactate dehydrogenase (LDH) with minimal or no rise in serum aminotransferase [7]. Elevated LDH usually indicates infarction of the kidneys [8].

The differential diagnosis for this disorder includes causes of acute surgical abdomen and non-surgical causes such as mesenteric ischemia, nephrolithiasis and pyelonephritis [1]. A CT scan with contrast can confirm renal artery thrombosis [8]. The risks and benefits of the use of intravenous contrast with concomitant renal failure have to be carefully assessed. However, if there is high pre-test probability of renovascular thrombosis, benefits outweigh risks as prompt intervention could be lifesaving and also help recover renal function. A radioisotope scan can show segmental or generalized decrease in renal perfusion and can be used as an additional non-invasive modality to confirm the diagnosis [1]. Renal arterial duplex Doppler ultrasound study has low sensitivity for the diagnosis of renal arterial thrombosis [1]. Renal arteriography is the gold standard for making the diagnosis of renal arterial thrombosis [4]. The work-up should also include echocardiogram to identify the source of thromboembolism and laboratory testing for hypercoagulability.

The etiology of thrombosis is multifactorial in our patient: (i) injury to endothelium and increase in the levels of procoagulants like fibrinogen and Factor V and VIII due to release of proteolytic enzymes [9], (ii) increased thromboxane levels, platelet-endothelium interaction and platelet activation and adhesion [10] and (iii) anatomic proximity of renal vessels to the inflamed pancreas [2].

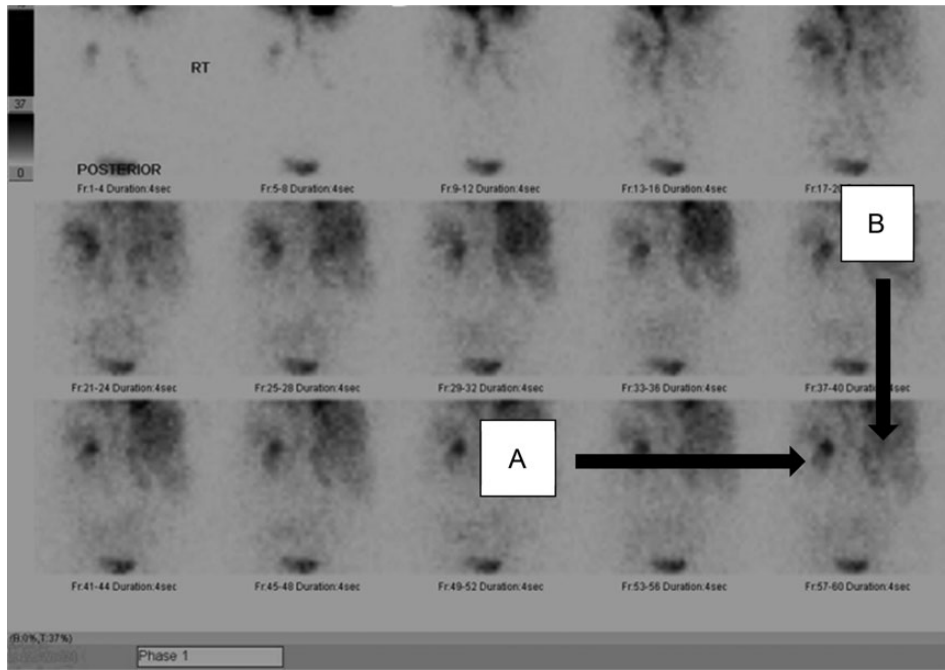


Fig. 3. Tc-99m MAG-3 scan showing delayed uptake of tracer in both kidneys except upper poles (A). Lateral aspect of right kidney showing no uptake consistent with infarct (B). Split cortical function is 61% left and 39% right.

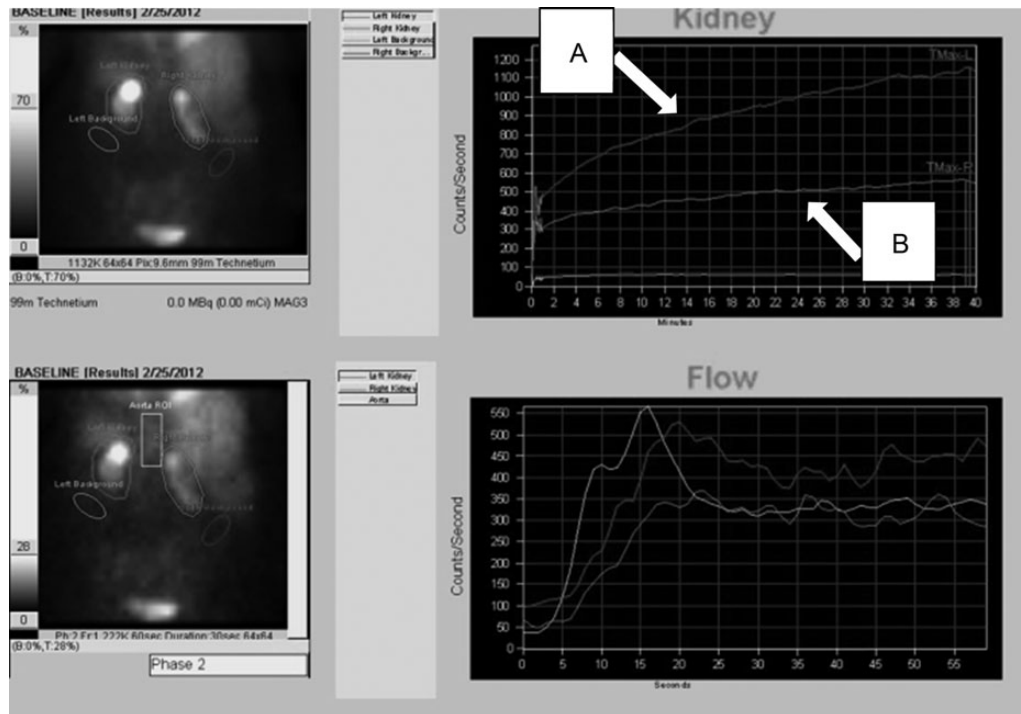


Fig. 4. Renogram showing delayed perfusion phase and function phase consistent bilateral reno-vascular obstruction and parenchymal damage. (Right kidney = B, left kidney = A.)

Bilateral renal artery thrombosis due to acute pancreatitis is rare. Few cases of renal vein thrombosis secondary to pancreatitis have been reported previously [11]. The presence of deep vein thrombosis in the lower extremities and thrombus in the right jugular vein support the hypercoagulable state in our patient.

Anticoagulation with heparin should be initiated for renal artery thrombosis if revascularization is not an option [5]. The optimal duration of anticoagulation is uncertain; however, since we postulated that the etiology of our patient's thrombosis was due to pancreatitis, we decided to treat her for 3 months. The decision to perform

Table 2. Work up for hypercoagulability

Laboratory test	Value	Reference range
Plasma Homocysteine(total)	14 µmol/L	5–15 µmol/L
β ₂ -Glycoprotein 1 Ab, IgG	<4 U/mL	<10 U/mL negative
β ₂ -Glycoprotein 1 Ab, IgM	<4 U/mL	<10 U/mL negative
β ₂ -Glycoprotein 1 Ab, IgA	<4 U/mL	<10 U/mL negative
Anti-cardiolipin IgA	0 APL	0–11 APL
Anti-cardiolipin IgG	5 APL	0–14 APL
Anti-cardiolipin IgM	4 APL	0–12 APL
Lupus anticoagulant screen	44 s	28–45 s
Factor V Leiden R506Q mutation	Negative	Negative
Prothrombin (F2)G20210A mutation	Negative	Negative
Anti-thrombin III activity	86	85–128%
Anti-thrombin antigen	92	82–136%
Protein C activity	84	84–171%
Protein C total antigen	77	63–153%
Protein S activity	56	54–132%
Protein S total antigen	71	63–126%

endovascular or surgical revascularization will be based on the presence of collateral circulation to the kidneys and time since the event. Patients with chronic atherosclerotic disease of the aorta and renal arteries usually have adequate collateral circulation. These patients can have relatively preserved renal function even with complete occlusion of the renal arteries [12]. The optimal period for revascularization of thrombosed renal arteries is yet unclear; however, revascularization data from other arterial territories would suggest earlier revascularization for optimal results. Revascularization therapy can be performed endovascularly or surgically based on the timing of diagnosis and patient comorbidities [6, 13]. Endovascular revascularization can be performed using intra-arterial streptokinase/urokinase infusion, t-PA power-pulse spray rheolytic thrombectomy and transluminal balloon angioplasty with or without stenting [6, 14, 15]. Surgical techniques include aorto-renal bypass graft, *in situ* repair, arteriotomy with thrombectomy and auto-transplantation of the kidney to the hypogastric vessels [14]. Revascularization was not attempted in our patient because of the delay in diagnosis and unstable post-surgical course following the pancreatic necrosectomy.

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Conflict of interest statement. None declared.

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