

A rare case of bilateral renal artery thrombosis in a patient with atrial fibrillation despite appropriate anticoagulation

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A 49-year-old man presented to our department with a 1-week history of 2 episodes of sudden-onset, self-limiting, sharp lumbar pain. His past medical history included hypertension and permanent atrial fibrillation. His general practitioner confirmed satisfactory anticoagulation with acenocoumarol. The patient was a nonsmoker and had no family history of thromboembolic events.

At presentation, the patient was hemodynamically stable and his physical examination was unremarkable. His blood pressure was 135/82 mmHg. Full blood count and the levels of urea and electrolytes were normal. Inflammatory markers were mildly raised. Serum creatinine levels were 97 mmol/l, yielding an estimated glomerular

filtration rate of 72 ml/min/1.73 m². The international normalized ratio on admission was within the therapeutic range (2.16). Neither microscopic hematuria nor microalbuminuria was present. A computed tomography (CT) angiogram of the abdomen demonstrated several thrombi within the segmental branches of both renal arteries, with associated areas of ischemia (FIGURE 1A and 1B). Both renal trunks were patent and the opacification of renal veins was symmetrical. A renal artery Doppler ultrasound was performed, revealing a decreased number of intrarenal vessels in ischemic areas that correlated with CT findings.

Due to late presentation, thrombolysis was not considered. The patient continued to receive

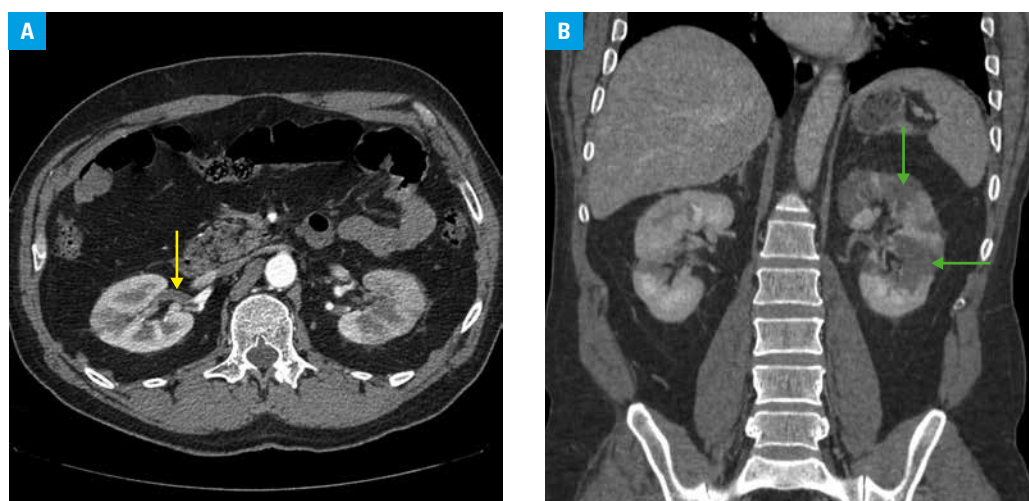
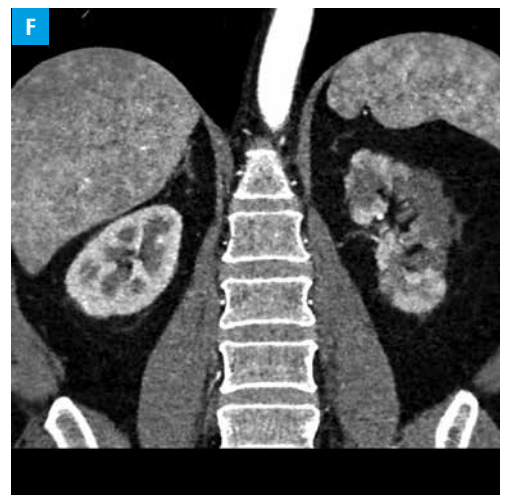
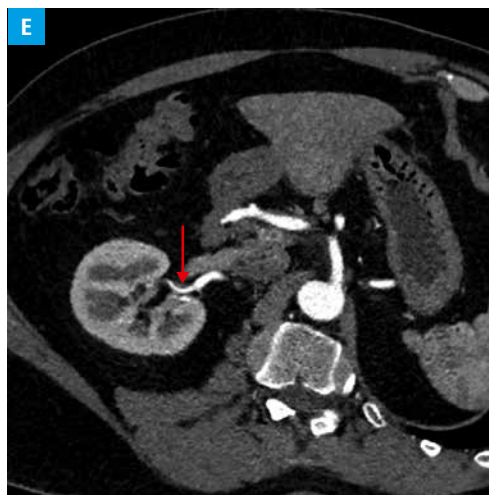
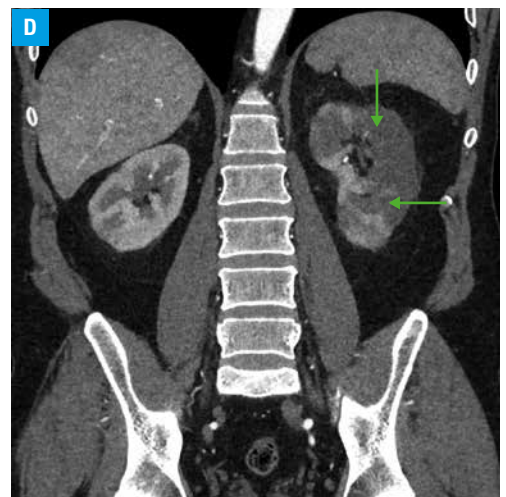
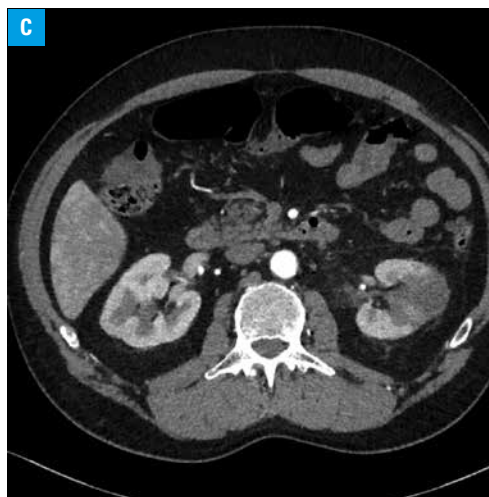


FIGURE 1 Evolving changes of bilateral renal artery thrombosis visible on consecutive computed tomography scans at presentation (A, B) and at 1 (C, D) and 3 months (E, F). Transverse sections on the left (A, C, E), coronal sections on the right (B, D, F). Images on the left show evidence of complete occlusion of the right superior segmental artery (A, yellow arrow) with visible progressive recanalization (C and E, red arrow). Images on the right show diffuse ischemic changes (B and D, green arrows) in the left kidney with evidence of diffuse parenchymal thinning (F).

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oral anticoagulation. Further diagnostic work-up with transesophageal echocardiography excluded the presence of mural thrombi and spontaneous echo contrast in all heart chambers. CT scans at 1 month (FIGURE 1C and 1D) and 3 months (FIGURE 1E and 1F) indicated the evolution of ischemic areas and evidence of progressive recanalization. Due to the unusual presentation, we performed further diagnostic testing, which excluded autoimmune and hematological disorders. The outcome at 1 year remained favorable, and blood pressure remained well controlled while staying on 1 antihypertensive drug.

Renal artery thrombosis is a rare, serious, and often misdiagnosed condition of multiple etiologies, including atherosclerosis, trauma, fibromuscular dysplasia, or spontaneous dissection of a renal artery or aorta. However, the most common cause of renal artery thrombosis reported by far is atrial fibrillation (AF).^{1,2} The mechanism of thrombosis in AF is a complex interaction of such factors as abnormal structural defects (dilated left atrium/atrial appendage) in the endothelium, distorted coagulation, and inflammation, and cannot be attributed only to blood stasis within the left atrium/atrial appendage that leads to peripheral embolization.³ This case is unusual as our patient was appropriately anticoagulated and had no evidence of intramural thrombi

or underlying clotting disorders. It is unlikely that an embolus originating from a mural thrombus would have caused symmetrical, bilateral renal artery embolization.

With this case, we would like to highlight several points. First, renal artery thrombosis is a rare but possible thromboembolic consequence of AF. Second, thrombosis in AF does not necessarily occur due to blood stasis within the left atrium, and the mechanism leading to thrombosis is more complex.

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REFERENCES

- 1 Hazanov N, Somin M, Attali M, et al. Acute renal embolism: forty-four cases of renal infarction in patients with atrial fibrillation. *Medicine*. 2004; 83: 292-299.
- 2 Ciećwierz D, Skarżyński P, Mielczarek M, et al. Manual aspiration thrombectomy complemented with local thrombolysis for acute renal artery embolism. *Pol Arch Med Wewn*. 2015; 125: 473-474.
- 3 Watson T, Shantsila E, Lip G. Mechanisms of thrombogenesis in atrial fibrillation: Virchow's triad revisited. *Lancet*. 2009; 373: 155-166.