



Fig. 1. CT angiography revealed a dilated left renal vein after passing between the aorta and superior mesenteric artery.

dominal aorta, was first named in 1972 by De Schepper [2]. Renal Doppler ultrasound, CT angiography, magnetic resonance angiography or venography can be used for the documentation of the degree of left renal vein compression [5]. NCS presented with FMF was first reported in 2009 by Ozcan *et al.*, which was similar to our case [6]. Here, we presume that actually, from the beginning, our patient's proteinuria was due to NCS. For mild haematuria or proteinuria, conservative treatment is proposed, whereas for recurrent severe haematuria or flank pain, endovascular stent surgical treatment is proposed for NCS [2]. Here, we proposed the conservative treatment. Mild proteinuria is a benign condition, but it can be persistent, and therefore, conservative treatment should be continued for several years. Although amyloidosis should be considered first in clarifying the aetiology of proteinuria in FMF, NCS, another rare entity, should be kept in mind and should also be excluded by non-invasive techniques when possible or by invasive techniques if necessary in FMF patients.

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Blue toe syndrome as a clue to the underlying cause of acute renal failure

To the Editor,

We present here a scenario of acute renal failure in the setting of blue toe syndrome. A 62-year-old male with a past medical history of hyperlipidaemia, hypertension and stroke presented with a 2-week history of severe diarrhoea, generalized weakness and myalgias. His home medications included Lipitor, Coumadin, Lopressor, Aspirin and lisinopril. During his prior hospitalization, one and a half months ago, he had undergone coronary artery bypass graft which was complicated by an ascending and descending aortic dissection post-operatively. The ascending dissection was surgically repaired. The origin of the celiac axis, superior mesenteric artery and renal arteries was from the true lumen, and thus, the blood flow was not compromised from the descending aortic dissection (Figure 1a). On examination, the patient's blood pressure was 157/91 mmHg and pulse 85 beats/minute of equal strength in both upper and lower extremities. His systemic examination was significant for painful bluish discoloration of the toes bilaterally (Figure 1b). Initial laboratory results revealed blood urea nitrogen 111 mg/dL, serum creatinine 10.8 mg/dL, potassium 5.8 mEq/L, bicarbonate 15 mEq/L and creatinine phosphokinase 12 893 IU/L. Complement C3 level was decreased. Urine analysis did not reveal muddy brown casts. The remainder of the laboratory panel was also unremarkable. Lipitor was discontinued. Despite adequate hydration, the patient remained oliguric with no improvement in his renal function. In the setting of surgical history and blue toe syndrome, an atheroembolic phenomenon as the cause of renal failure was considered. Direct ophthalmoscopy and slit lamp examination of the eye revealed cholesterol crystal emboli in the retinal arterioles (Hollenhorst plaques, Figure 1c) and thus confirmed the diagnosis of atheroembolic disease. The patient was started on haemodialysis.

Atheroembolic disease may present with general symptoms of fever, myalgias, headache, weight loss and diarrhea [1]. Though sometimes subtle, the pathological process of dislodging multitude of cholesterol crystals from atherosclerotic plaques in the arteries (often post-operatively) can manifest symptomatically diversely. The

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Fig. 1. (a) The aortic dissection/aneurysm at the level of the left renal artery measured 3.5 cm, and the renal arteries originated from the true lumen. In addition, there was no evidence of aortic leak, and at the level of the origin of the celiac artery and SMA, approximately one-third of the anterior aspect of the aorta was patent. (b) Blue toes suggestive of atheroembolic disease. (c) Cartoon image of the patient's retina as seen on direct ophthalmoscope. It revealed cholesterol crystal emboli in the retinal arterioles—Hollenhorst plaques.

emboli may travel to the capillary beds of the renal, mesenteric, retinal, tibial and peroneal arteries capable of producing digital or skin ischaemia, and even overt organ failure. Therefore, a high degree of clinical suspicion is warranted since, in the setting of gastrointestinal symptoms, abnormal renal parameters may be confused for acute renal failure secondary to severe dehydration. While the cholesterol showers to the lower extremities are often labeled as 'blue toe syndrome', the actual cutaneous manifestations of the bluish discoloration are uncommonly seen in only 5% of patients with atheroembolic disease [1]. In our patient, the blue toes were the sole dermatological manifestation. Other more classical manifestations include cord-like purplish cutaneous discoloration in the lower extremities (livedo reticularis). Acute renal failure is present in 25–50% of atheroembolic cases [2]. Although the disease commonly occurs after invasive vascular procedures (i.e. coronary angiography via femoral artery), instances of spontaneous embolization have also been reported. In the latter cases, the diagnosis may be difficult to establish without a renal biopsy confirmation. However, other embolic signs may be used to reach a presumptive diagnosis. In our patient, the presence of Hollenhorst pla-

ques in the retina with the recent history of aortic surgery was thus diagnostic of atheroembolic renal disease.

Blue toe syndrome may be the sole dermatological manifestation of atheroembolic renal disease. Hollenhorst plaques in the retina may aid in the diagnosis.

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