

THE CLINICAL PICTURE

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A 68-year-old man with a blue toe



FIGURE 1. Signs of blue-toe syndrome were apparent on the patient's mottled distal right hallux.

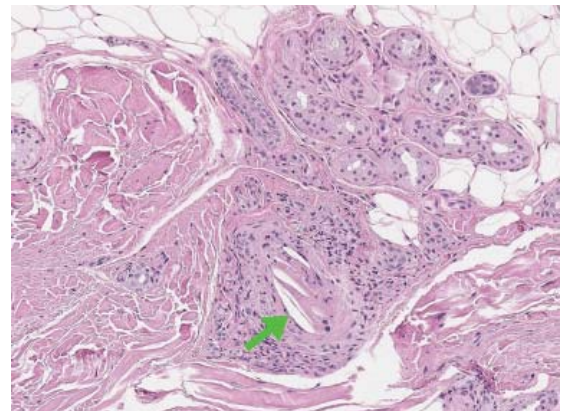


FIGURE 2. Biopsy of the cutaneous lesion on the toe revealed cholesterol clefts (arrow) (hematoxylin and eosin, $\times 100$).

The differential diagnosis includes contrast nephropathy and infectious endocarditis

A 68-YEAR-OLD MAN presented with concern about a bluish toe. Several months earlier he had undergone total aortic arch replacement and coronary artery bypass grafting. Since then his renal function had declined and he had been losing weight.

He had hypercholesterolemia, hypertension, and a 20-pack-year smoking history. Physical examination confirmed that his right great toe was indeed bluish (**Figure 1**). Peripheral, neck, and abdominal vascular examinations were normal. Laboratory testing revealed:

- Serum creatinine concentration 5.15 mg/dL (reference range 0.61–1.04)
- C-reactive protein level 1.5 mg/dL (0–0.3)
- Eosinophil count $0.58 \times 10^9/L$ (0–0.50)
- Serum complement level normal
- Urine sediment unremarkable.

Transthoracic echocardiography revealed no evidence of vegetation, and a series of blood cultures were negative. The right toe was biopsied, and study revealed cholesterol clefts (**Figure 2**), confirming the diagnosis of cholesterol crystal embolism.

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He was treated with prednisolone 20 mg/day, and his weight loss and renal function improved.

■ CHOLESTEROL CRYSTAL EMBOLISM

Cholesterol embolization typically occurs after arteriography, cardiac catheterization, vascular surgery, or anticoagulant use in men over age 55 with atherosclerosis.¹ It presents with renal failure, abdominal pain, systemic symptoms, or, most commonly (in 88% of cases), skin findings.²

“Blue-toe syndrome,” characterized by tissue ischemia, is seen in 65% of patients.² Lesions can appear anywhere on the body, but most commonly on the lower extremities. Most are painful due to ischemia. The condition can progress to necrosis.

Patients may have elevated C-reactive protein, hypocomplementemia (39%), and eosinophilia (80%).^{3,4} The diagnosis is confirmed only with histopathologic findings of intravascular cholesterol crystals, seen as cholesterol clefts.

The differential diagnosis includes con-

trast nephropathy and infectious endocarditis. However, contrast nephropathy begins to recover within several days and is not accompanied by skin lesions. Repeated blood cultures and echocardiography are useful to rule out infectious endocarditis.

Treatment includes managing cardiovas-

cular risk factors and end-organ ischemia and preventing recurrent embolization. Surgical or endovascular treatment has been shown to be effective in decreasing the rate of further embolism.² Corticosteroid therapy is assumed to control the secondary inflammation associated with cholesterol crystal embolism.^{1,5} ■

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