Cholesterol crystal embolisation is a frequently underdiagnosed condition. While coronary catheterisation is safe and commonly performed, the reported patient developed very painful trash feet after undergoing this routine procedure. Ulceration and gangrene occurred after catheter manipulation during cardiac angiography, which caused occlusion of the small arteries in his feet. The triad of pain, livedo reticularis, and intact peripheral pulses is pathognomonic for cholesterol embolisation. The prognosis depends on the extent of the systemic disease and a high rate of mortality (75–80%) is observed. Prognosis is poor and the treatment is only supportive. It is suggested that while cardiac catheterisation is largely safe and a very commonly performed procedure, it can still lead to complications with serious side effects and can even prove fatal.

CASE REPORT

A 65 year old man presented to our accident and emergency department with patchy mottling of both his feet. The following day this progressed to painful blisters and then to ulcers and gangrene (fig 1).

Three weeks before this event, he had presented to the accident and emergency department with bilateral painful thighs following a routine uneventful cardiac angiography. He was

Figure 1 Trash feet after coronary angiography from different views.

Cholesterol crystal embolisation (CCE) is a clinical disorder that is still frequently underdiagnosed. The incidence of this disorder is approximately 0.08%. Though Flory described it as early as 1945, its diagnosis is still a dilemma for clinicians. Moreover, there is no cure for the disease and the treatment is mainly supportive. The prognosis of the condition is very poor with a mortality rate of 72–80%.

Coronary catheterisation is a safe and commonly performed procedure. We present a case report of a patient who underwent a routine coronary catheterisation, which resulted in very painful trash feet.

CASE REPORT

A 65 year old man presented to our accident and emergency department with patchy mottling of both his feet. The following day this progressed to painful blisters and then to ulcers and gangrene (fig 1).

Three weeks before this event, he had presented to the accident and emergency department with bilateral painful thighs following a routine uneventful cardiac angiography. He was
admitted on that occasion and treated with anticoagulant. He was discharged home on the 10th day with warfarin.

His other medical history included unstable angina, chronic obstructive arterial disease, aortic aneurysm, and borderline hypercholesterolaemia, and he was a known former smoker. He was not known to be diabetic and his family history was not notable.

Physical examination found tender distal toes with intact distal pulses. Both his feet were warm and well perfused other than the gangrenous patches. Further neurovascular examination of the lower limb found no abnormalities.

Laboratory investigation showed an eosinophilic count of $1.03 \times 10^9/l$, erythrocyte sedimentation rate of 34 mm in the first hour, urea concentration of 8.4 mmol/l, and creatinine concentration of 138 µmol/l. His creatine kinase was 1363 IU/l. His dorsalis pedis resting pressure indices were 0.99 and 0.97 for the left and right foot, respectively.

He was further managed conservatively with analgesics and anticoagulation. His ulcers did not worsen and he is settling with the current treatment.

DISCUSSION

The condition is CCE. The ulceration and gangrene in his feet resulted after catheter manipulation during cardiac angiography and caused occlusion of the small arteries in the feet. The triad of thigh and foot pain, livedo reticularis, and intact peripheral pulses is considered to be pathognomonic for cholesterol embolisation.

An eosinophilia, raised erythrocyte sedimentation rate, and decreased complement concentration may support the diagnosis; however, a final diagnosis can be established only with tissue biopsy evidence of cholesterol crystals in the involved skin, muscle, and kidney.

This condition predominantly affects the elderly (mean 66 years), frequently those with a history of hypertension (61%), atherosclerotic cardiovascular disease (44%), renal failure (34%), and aortic aneurysm (25%) at presentation.

Clinical presentation varies and depends on the origin of the atheroma. Microembolism of the cholesterol crystals and atherosclerotic debris results from manipulation by cardiac catheters. If the proximal aorta has been disturbed then symptoms may occur in the central nervous system, abdominal organs, and extremities. The lower extremities are involved if the site is below the renal arteries.

Skin is involved in 35% of patients with CCE. Livedo reticularis is the most common skin disorder (49%), followed by gangrene (35%), cyanosis (28%), and ulceration (17%). Other manifestations are transient ischaemic attacks, amaurosis fugax, acute and chronic renal failure, and rarer manifestations such as hypertension, bowel ischaemia and infarction, pancreatitis, and adrenal insufficiency. However, painful limbs and cutaneous manifestation along with intact peripheral pulses are clinically pathognomonic for CCE.

Laboratory tests that may aid in the diagnosis of CCE include eosinophilia, eosinophiluria, increased sedimentation rate, and decreased complement concentrations. Final diagnosis is established with biopsy and the aforementioned clinical findings.

Multiple treatment regimens have been generally unsuccessful in altering the course of the disease process. The most significant impact on the disease can be made by its prevention. Thrombolytic and anticoagulant treatments are not indicated and only supportive care for such symptoms as hypertension, ulceration, and gangrene is advocated. The prognosis depends on the extent of the systemic disease and a high rate of mortality (75–80%) is recorded due to multifactorial, cardiac, and renal aetiologies.

Prognosis is poor with high mortality and the treatment is only supportive.

REFERENCES
