Journal of Plastic, Reconstructive & Aesthetic Surgery (2006) **59**, 429–432

JPRAS
An International Journal of Surgical Reconstruction
www.JPRASurg.com

CASE REPORT

A case of skin necrosis as a result of cholesterol crystal embolisation

Melike Erdim, Erdem Tezel*, Nurdan Biskin

Department of Plastic and Reconstructive Surgery, Faculty of Medicine, Marmara University, Tophanelioglu Caddesi, Altunizade, Uskudar, Istanbul, Turkey

Received 7 May 2005; accepted 6 September 2005

KEYWORDS
Cholesterol crystal embolism;
Cholesterol embolism;
Cholesterol embolization syndrome;
Livedo reticularis

Summary Cholesterol crystal embolism is a multisystem disorder with a high mortality. It is usually seen following vascular surgery and long term anticoagulation therapy, but the diagnosis is often not considered. Skin manifestations are the first sign of cholesterol crystal embolism and recognition of the symptoms is a key element in early diagnosis and prevention of recurrences. We report a case who presented with acute renal failure, livedo reticularis and skin necrosis following angioplasty. Cutaneous biopsy revealed cholesterol crystals in the lumen of a vessel. Necrotic sites were treated with daily wound care and he was operated for reconstruction of tissue defects with cutaneous advancement flaps. One month after the operation healing was complete.

Cholesterol crystal embolism (CCE) is a life threatening, multisystem disorder resulting from atherosclerotic plaque embolisation from the aorta or its major branches.[1,2] While spontaneous occurance of CCE is possible, there are several causative factors such as invasive vascular procedures and anticoagulant therapies.[3] Most commonly affected organs are the kidney and skin although many organs could be involved.[1] CCE has a 1 year mortality rate of 64–87% and the cause of death is multiorgan failure in most of the cases.[1,4,5] The disease is difficult to be recognised in life and only 30% of the patients are diagnosed premortem.[6] We present a case who developed CCE following coronary angioplasty with cutaneous manifestations including skin necrosis and acute renal failure.

Case report

A 69-year-old male patient with known hyperlipidemia and hypertension, was admitted to a hospital with myocardial infarction. He underwent emergency right coronary artery stent angioplasty via the right femoral artery. One day after the angioplasty procedure the patient developed

* Corresponding author. Address: Tophanelioglu Caddesi, Marmara Universitesi Tıp Fakultesi Hastanesi, 13/15, Plastik Rekonstruktif Cerrahi Departmanı, Altunizade/Uskudar, Istanbul, Turkey. Tel.: +90 532 4220423; fax: +90 216 3267722.
E-mail addresses: erdem@erdemtezel.com (E. Tezel), erdemtezel@hotmail.com (E. Tezel).

S0007-1226/$ - see front matter © 2005 The British Association of Plastic Surgeons. Published by Elsevier Ltd. All rights reserved.
doi:10.1016/j.bjps.2005.09.033

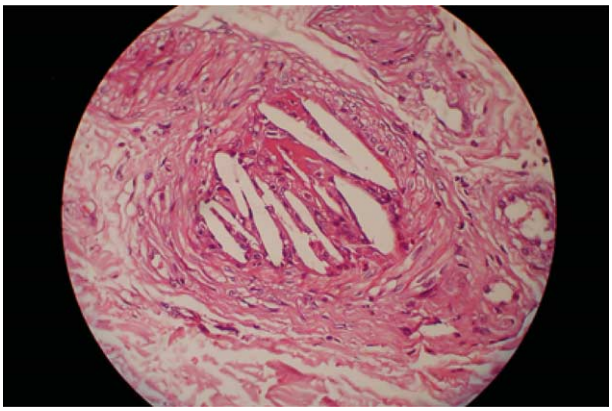

Figure 1 Skin biopsy showing cholesterol clefts in the lumen of a vessel.

purplish discoloration at the angiography catheterization site. On the following days livedo reticularis of the lower extremities was observed which were more prominent when standing. Moreover painful ecchymosis on the right superolateral thigh, suprapubic area, left lateral thigh and sacral region were detected. Labaratory workup was within normal ranges. He was discharged from the hospital 2 days after the procedure.

One and a half month later, the patient was readmitted to another hospital with angina pectoris and the angiography revealed obliteration of coronary vessels. He underwent emergency coronary angioplasty via the left femoral artery. Blood tests revealed eosinophilia and an increase in creatinine level. The previously ecchymotic areas had progressed to partially ulcerated lesions and skin biopsy was taken from the necrotic ulceration from the lateral thigh region. Histopathological examination revealed cholesterol crystals within the lumen of a small vessel, which are diagnostic for CCE. (Fig. 1) In the following weeks he developed acute renal failure (ARF) and was treated with intravenous prednisolone 40 mg/day with the diagnosis of ARF secondary to CCE. Renal function improved with treatment and the steroid dose was tapered. During this period he was referred to us for the necrotic ulcerations and physical examination revealed multiple areas of skin necrosis in the left and right lateral thigh regions, ecchymosis on the sacral region, and cyanosis of the toes bilaterally. At the time of the consultation it was decided to remain conservative and observe the progression of the lesions, while the patient recovered from ARF. The patient was not seen for 2 months, then he presented to our clinic with progression of necrotic sites and infection. Physical examination revealed necrosis on the left superolateral thigh (25$\times$10 cm), sacral region (3$\times$5 cm), right anterolateral thigh (35$\times$15 cm) (Fig. 2). He was hospitalised and was treated with appropriate antibiotics and daily debridement of the wounds. He was under oral steroid therapy at the time. It was found appropriate to delay the operation until the infection had subsided and the steroid therapy had been discontinued. Intraoperatively it was found that skin necrosis extended into the subcutaneous fatty tissue. All the necrotic ulcerations were debrided and some of the defects were primarly sutured while some of them were reconstructed by cutaneous advancement flaps. Remaining tissue defects underwent secondary healing. Almost 1 month following the initial operation healing was complete (Fig. 3).

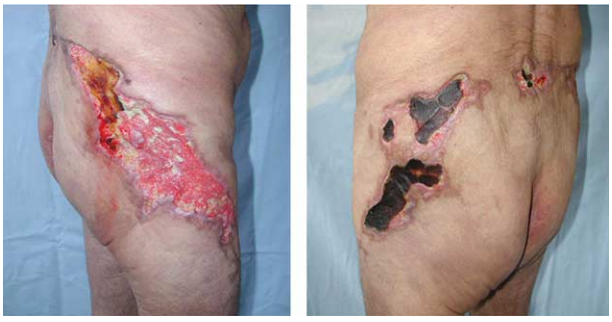

Figure 2 Preoperative view of the necrotic sites.

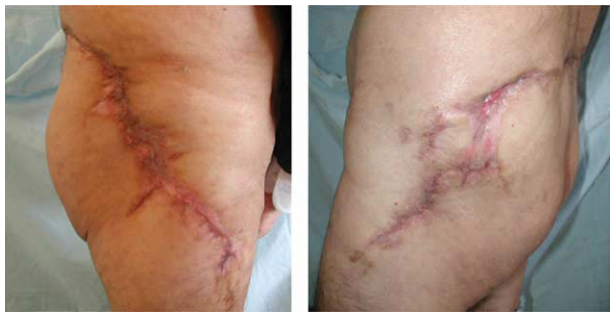

Figure 3 Postoperative photo of the patient upon complete healing.

Discussion

CCE is a multiorgan disorder which is caused by atheromatous plaque embolisation from major arteries. This embolisation may occur spontaneously as in aortic aneursym or may be precipitated by several factors such as vascular surgery, aortic manipulation (i.e. angiography, angioplasty), long term anticoagulation and thrombolytic therapy.[6,7] When the atheromatous plaques are disrupted, components are released into the blood stream and occlude the small arterioles in the