Case report

Severe cutaneous cholesterol emboli syndrome after coronary angiography

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Abstract

Cholesterol embolization syndrome is due to dislodgment of cholesterol crystals from the atherosclerotic plaques lining the walls of major arteries resulting in an occlusion of small arteries. We describe a case of severe cutaneous cholesterol emboli syndrome following repeat coronary angiography showing by our observation that this syndrome is often unrecognized or misdiagnosed and that a better evaluation of risks factors in patients undergoing invasive procedures could prevent this severe complication. © 1999 Elsevier Science B.V. All rights reserved.

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1. Introduction

The cholesterol syndrome is due to dislodgment of cholesterol crystals from the atheromatous aorta resulting in an occlusion of small arteries. Such emboli may occur spontaneously but often follow vascular surgery or radiological investigation especially coronary diagnostic or interventional procedures [1].

The organs most frequently involved are the skin and the kidneys but any organ can be affected [2]. The cutaneous manifestations include livedo reticularis, indurated firm violaceous painful plaques and noduli tending to show central necrosis and tender blue toes [3]. The latter can be the only clinical manifestation, also known as blue toe syndrome [4]. Suggestive symptoms include pain, often very severe, myalgia and restless legs [3].

We describe a case of severe cutaneous cholesterol emboli with mild impairment of renal function following repeat coronary angiography.

2. Case report

A 65-year-old woman with a history of severe unstable angina, abdominal aortic aneurysm, claudicatio intermittens and non-insulin-dependent diabetes had undergone a coronary angiography revealing a triple vessel disease. A few days after the procedure the patient complained of pain in the left foot followed by the appearance of a blue left big toe. These symptoms, being diagnosed as arterial embolus, were treated with i.v. heparin and analgesics.

One week later she presented at our institution to be submitted to a hybrid revascularization. A percutaneous transluminal coronary angioplasty (PTCA) on the right coronary artery was followed 2 days later by a left internal mammary artery bypass to the left anterior descending artery (LAD) through a left anterior thoracotomy. The postoperative period was uneventful. As a part of a clinical study the patient underwent a control angiography which showed an occlusion of the left internal mammary artery (LIMA) to LAD anastomosis. Three days later she complained of severe pain in the calves diagnosed as a deep thrombophlebitis and was treated with subcutaneous injections of Calci-parin. In the next days the patient showed exacerbation of...
Fig. 1. Hemorrhagic nodules and plaques with central necrosis on the lower abdomen and inferior limbs showing a reticular vascular pattern.

Fig. 2. Biopsy of skin showing small artery containing cholesterol clefts with foreign body giant-cell reaction. (Hematoxylin-eosin stain, original magnification: x100).
the pain followed by the acute appearance of livedo reticularis on the legs, acrocyanosis of the feet and large tender ecchymotic nodules and plaques which were localized on the lower abdomen and limbs where they showed a reticular vascular pattern. In the following days most of these lesions underwent massive central necrosis with the formation of black eschars (Fig. 1). A deep cutaneous biopsy was performed on an ecchymotic area on the left thigh which showed typical intraluminal cholesterol clefts and giant cells diagnostic for cholesterol emboli (Fig. 2).

Abnormal laboratory values include a raise in creatine phosphokinase (466), lactate dehydrogenase (835), creatinine (2.33), sedimentation rate (57) and blood eosinophilia (15%).

An abdominal computerized axial tomography confirmed the presence of a 4 cm aneurysm of the subrenal aorta extending to the iliaca arteries. The optic fundi were normal. Distal pulses were present.

The anticoagulant treatment was immediately stopped. A supportive therapy was undertaken to relieve the pain by means of a continuous epidural infusion with marcain with only slight relief of the pain as a result.

A follow-up period of 5 months showed normalization of the renal function within 4 weeks after the onset of the cholesterol syndrome while the cutaneous embolization went on for more than 3 months with persistence of severe pain in the lower limbs and the development of new hemorrhagic lesions which showed very slow healing, leaving atrophic scars.

Patient died about 5 months from the onset of the disease likely of cardiac failure.

3. Discussion

Cholesterol emboli may occur spontaneously but more often follow vascular surgery or radiological investigation especially coronary interventional procedures. Major predictive factors include advanced age, repeat vascular procedures, female gender and peripheral vascular disease. Minor predictors include rate of anticoagulation, use of thrombolitics, elevated creatinine levels, low platelet counts, longer periods of anticoagulation and use of increased sheath size of the catheters [5].

The reported incidence of cholesterol emboli after coronary interventional procedures is about 0.08% [6]. Since vascular procedures are likely to grow in number we should expect to observe cholesterol embolization with greater frequency. However the disorder is frequently underdiagnosed especially when the clinical symptoms are limited to the presence of acrocyanosis or a mild impairment of the renal function.

Various laboratory tests may support the diagnosis of cholesterol embolization such as eosinophilia and a raised sedimentation rate. In patients with multisystem involvement there may be abnormal renal function tests, increased amylase levels, hematuria, melena and anemia.

However, the diagnosis is only established with a deep skin biopsy showing the typical (cholesterol) needle-shaped clefts of cholesterol infiltrates and giant cells into the arteriolar lumen [7].

Our case confirms that cholesterol emboli are often unrecognized or misdiagnosed. Actually the pain in the toes and acrocyanosis following the first coronary angiography which were suggestive for the blue toe syndrome had been interpreted as a thrombotic embolus and treated with anticoagulants which increased greatly the risk for other cholesterol emboli as they interfere with the stabilization of cholesterol crystals embedded within atheromatous plaques by platelet-fibrin thrombi [2]. A more accurate evaluation of the risks factors present in our patient such as the presence of an aneurysm of the abdominal aorta, repeat vascular procedures, female gender, peripheral vascular disease and use of anticoagulants, could have led to an earlier diagnosis and prevent unwarranted procedures such as anticoagulation and repeat coronary angiography.

In conclusion, a better assessment of risks factors in patients undergoing vascular procedures and a heightened awareness of the cholesterol emboli syndrome as a possible complication of vascular procedures, may lead to its correct identification and prompt management.

References