Case Report

A 75-year-old man with renal insufficiency and eosinophilia after coronary angiography

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Case presentation

A 75-year-old male patient received esophageal carcinoma surgery in Oct 2005. The next day of the operation, he had dyspnea, chest discomfort and sweating when he was on some activities. ECG showed ST segment elevation and T wave depression in leads V1–6. Biochemical markers of myocardial necrosis were elevated. A diagnosis of acute myocardial infarction was made. After anticoagulant, antiplatelet and vasodilator therapy, his symptoms relieved in 3 hours. One week before the admission, a visible edema of the left lower extremity occurred. Doppler ultrasound showed thrombotic occlusion of the left superficial femoral vein and popliteal vein. With further anticoagulant and vasodilator therapy, the edema disappeared. He was admitted to our hospital for further assessment and treatment on Nov 3, 2005. Diagnosis on admission were coronary heart disease, acute myocardial infarction of anterior wall, esophageal carcinoma after surgery and thrombosis of left lower extremity vein. Blood, urine and stool routine tests, liver and renal functions were normal. Doppler ultrasound showed atherosclerosis of both lower extremities, severe narrowed inferior part of right superficial femoral artery and unclear image of right posterior tibial artery (occlusion?). Echocardiography showed segmental wall motion abnormalities in distal part of anterior wall of left ventricle and the apex. LVEF (Simpson method) was 48%. The coronary angiography performed on Nov 14 showed total occlusion of the ostium of LAD. Arteriography of both renal arteries showed no stenosis. Percutaneous coronary intervention was tried but failed. In early December he began to have sour lower extremities with petechia on toes and soles. From Dec 11, his serum creatinine level elevated progressively, with the maximum being 403 μmol/L, and a gangrene occurred on the right heel. Blood examination showed WBC >10,000/μL, eosinophil 8%–15%, ESR 63–108 mm/h, IgM 0.36 g/L, other immunoglobulins in the normal ranges, complement C3 0.84 g/L and CRP 9.22 mg/L. Skin biopsy showed thrombosis in most small vessels of subcutaneous panniculus adiposus and no cholesterol crystals (Fig.1). His renal function recovered gradually and serum creatinine decreased to 166–203 μmol/L. The gangrene of right heel healed up by changing dressings. The petechia on toes and soles faded away. He recovered well and was discharged.

Clinicopathological discussion

Dr. Shao Geng: In this case, we can find the evidences of atherosclerosis of coronary and peripheral arteries. The patient had multiple petechia on lower extremities after anticoagulant therapy and CAG, with normal peripheral pulses and normal skin temperature, indicating the embolism at the site of small arteriole but not artery. The occurrence of the renal dysfunction which was manifested by the elevated level of serum creatinine indicated the embolism of the kidneys. In the laboratory examination, acceleration of ESR, elevation of WBC count and especially eosinophilia all reflected the activation of inflammatory reaction. When we...
take all the clinical manifestations into account, the diagnosis of cholesterol crystal embolism is established. Furthermore we should pay attention to that the embolism and inflammatory activation were not merely the effects of cholesterol crystal, other contents in the atheroma also played a role in the progress. Therefore someone suggests that it is suitably called “atheroembolism”. There is at present no special treatment for this syndrome. Statins may prevent the recurrence of embolism through stabilization of the cholesterol rich plaque. This patient is suitable to receive intensive lipid lowering therapy for this syndrome. Statins may prevent the recurrence of embolism, the surgery should be considered after the intensive lipid lowering therapy for a period. Although the patient is in the situation of inflammatory reaction caused by the activated immune system, it is better not to offer the treatment with corticosteroid for following reasons: 1) There is no evidence of improving the prognosis by corticosteroid treatment; 2) The patient had the history of venous thrombosis and heel ulcer.

Dr. Huo Yong: Though the biopsy of the skin lesion found no cholesterol crystals but thrombus, this case should still be diagnosed as cholesterol crystal embolism. The reasons are as follows: First, according to the onset time, the symptoms of thrombotic embolism take place just after the procedures, while the symptoms of cholesterol crystal embolism, which are caused not only by the mechanical embolism, but also by the immune response, often occur about 1 to 4 weeks after the procedures which conforms to the patient’s condition. Second, thrombotic embolism usually localized to a site, and is difficult to explain the widespread lesions of the skin, kidneys and muscles (he had sour lower extremities) in this patient. The third and the most important point is that, a thrombotic embolism cannot induce the elevation of WBC count, especially the elevation of acidophils, which is characteristic of cholesterol crystal embolism. On the other hand, the biopsy sampling has its own deficiency, and we should not exclude the possibility of cholesterol crystal embolism just depending on pathological examination. The symptoms of cholesterol crystal embolism are related to inflammatory reaction, and the reaction may have individual variations. This may be why the cholesterol crystal embolism is rarely seen despite of the great deal of intervention therapy.

Dr. Ding Wenhu: There is still no report of cholesterol crystal embolism in Chinese literature. According to reports from other countries, the incidence of cholesterol crystal embolism after coronary angiography and PCI varied from 0.08% to 4% in different groups of patient. Persons who have multiple artery atherosclerosis are more likely to suffer from the cholesterol crystal embolism. Most cases are related to anticoagulant, thrombolytic therapy, transluminal intervention or vascular surgery. For these patients embolism occurs in 1-4 weeks after these treatments or operations. However, in some cases embolism results from spontaneous of rupture atheromatous plaques without any causes. Except the lesions of skin, renal failure, the increase in ESR, the elevation of WBC count and eosinophilia present in this case, clinical manifestations also include symptoms of nervous system and digestive system, anemia, hypertension, hypocomplementemtamia etc., depending on the organs affected and degree of affection. Biopsies of affected organs including skin, kidney, intestine and muscle can show the needle shaped clefts left by the dissolved cholesterol crystals in lumina of arteriole. The positive rate of the skin biopsy is about 1/3 to 2/3. The specificity is high, but sensitivity is not 100% yet.

**Summary**

With the rising number of the cases of atherosclerosis and PCI, the occurrence of cholesterol crystal embolism would be increasing accordingly. Owing to different affected organs, the clinical manifestations are various, so physicians may ignore the diagnosis easily. It calls for physicians to intensify the knowledge about this syndrome. In this case, evidence of atherosclerosis in many sites, embolism in many sites 3-4 weeks after anticoagulant therapy and PCI and signs of immunological activation, such as elevation of the counts of WBC and eosinophils, acceleration of ESR and hypocomplementemtamia, strongly suggest the diagnosis of cholesterol crystal embolism. The result of skin biopsy can further support the diagnosis but the negative result can not exclude the diagnosis. There is no specific treatment. Symptomatic treatment can be used according to the affected organs. The indication and effectiveness of corticosteroid and plasmapheresis are still not clear. Statins could prevent further embolism as showed in some reports. However what is more important is to avoid using anticoagulant, thrombolysis, the operation of PCI or vascular surgery.