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Cholesterol Crystal Embolisms Complicating Coronary Artery Bypass Surgery: One Case Report

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Abstract: ***Introduction: Cholesterol crystal emboli (CCE) affect small caliber arteries, 100 to 400µ, and originate from unstable plaques usually located on the aorta and sometimes lower extremity arteries. Depending on the level of origin of the emboli, the symptomatology is highly variable, ranging from blue toes to systemic cholesterol crystal embolism disease to renal and/or digestive involvement. Observation: We report the observation of a 60 - year - old patient with multiple cardiovascular risk factors admitted for management of tritruncal coronary artery disease. The arterial doppler of the neck and lower limb vessels found diffuse atheromatous overload without significant stenosis. The patient underwent coronary artery bypass grafting under extracorporeal circulation with aortic clamping. The postoperative course was marked by the occurrence of bilateral distal ischemia of the toes with preservation of the distal pulses, the arterial doppler showed no change compared to the preoperative one. The biological blood test showed progressive renal failure with preserved dieresis. The diagnosis retained is a distal ischemia due to cholesterol embolism, which evolved into toe necrosis requiring toe amputation. Treatment with antiplatelet agents and statin was reinforced with a spectacular clinical improvement. Conclusion: We report through this observation in the light of the literature data the severity of cholesterol emboli and their therapeutic management.***

Keywords: cholesterol crystal emboli, aortic clamping, distal ischemia

1. Introduction

Cholesterol crystal emboli (CCE) affect small caliber arteries, 100 - 400µ, and originate from unstable plaques usually located on the aorta and sometimes lower extremity arteries [1]. Depending on the level of origin of the emboli, the symptomatology is highly variable, ranging from blue toes to systemic cholesterol crystal embolism disease to renal and/or digestive involvement.

2. Clinical Case

This is a 58 - year - old patient, diabetic, hypertensive and chronic smoker (50 packs/year). The patient presented with neglected chest pain until the onset of NYHA class III dyspnea, orthopnea and the occurrence of a myocardial infarction in the inferior territory of the heart. Coronary angiography showed a stenosis of (70 - 90%) the proximal anterior interventricular artery (AIV) and a subocclusive stenosis (90 - 99%) of the distal right coronary artery (RCA) with a poor downstream bed.

Preoperative transthoracic echocardiography showed high filling pressures and left ventricular dysfunction with an estimated ejection fraction of 40%.

The preoperative workup and the biological workup were unremarkable.

Fifteen days after his myocardial infarction (MI), the patient underwent surgical myocardial revascularization under cardiopulmonary bypass (CPB) and aortic clamping (AC). As the intraoperative verification of the coronary vessels showed a very small and calcified right coronary network with a small downstream bed, a coronary monobypass of the middle VIA through the left internal mammary artery was performed.

The cardiopulmonary bypass (CPB) and aortic clamping (AC) times were 51 and 26 min, respectively. The weaning from the CPB was easy under 5 ɣ/kg/min. The stay in the intensive care unit lasted 43 h with a good evolution.

During the hospital stay, the patient was under the usual postoperative treatment of coronary artery bypass surgery: converting enzyme inhibitor (CEI), β blocker, antiplatelet agent, statin, diuretic (post CEC), iron, proton pump inhibitor (PPI) and preventive anticoagulation by low molecular weight heparin.

The evolution was marked by the occurrence of cyanosis and coldness of the toes of both feet, of the lateral surfaces and soles of the feet {Fig 1, 2, 3} at 5 days post - op. In view of this situation, which suggested the onset of ischemia, the anticoagulant treatment was reinforced. Despite the preservation of distal pulses (especially pedal pulses) of the lower limbs, the clinical symptomatology rapidly worsened towards gangrene and amputation of all the toes of both feet, in parallel with the rapid worsening of renal function (previously normal): creatinine level: 315 µmol/l and glomerular filtration rate (GFR) at 18.9 ml/min/1.73 m². A significant elevation of CRP also accompanied the biological and clinical worsening of the patient.

The introduction of intravenous corticosteroid therapy based on 80 mg of solumedrol per day, the discontinuation of anticoagulation and the increase in the dose of statin, led to a dramatic improvement in renal function and clinical symptomatology (lateral surfaces and soles of both feet).

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3. Discussion

An atheromatous aorta can be a source of cholesterol crystal embolism (CCE). This embolization is responsible for obstruction of distal arterioles around which a granulomatous giant cell inflammatory reaction develops [2].

Diagnosis is often delayed or unrecognized due to various or misleading clinical signs, such as renal failure, digestive or neurological symptoms, or both [3].

Although CCE can occur spontaneously, it is increasingly recognized as an iatrogenic complication of an invasive vascular procedure, such as manipulation of the aorta during angiography or vascular surgery. It has also been reported to occur after anticoagulant therapy or thrombolysis. Patients undergoing coronary artery bypass grafting (CABG) often have a combination of these factors: anticoagulation, intra - arterial angiography, and intraoperative aortic clamping.

These multiple factors could explain the clinical condition as well as the biological disorder in our patient postoperatively.

The incidence of CCE is reported to be 0.9% after coronary angiography and about 5% after angioplasty of arteriosclerotic stenosis of the renal arteries. Its prevalence on all renal biopsies is 3.9% after 65 years of age. CCE are responsible for 3% to 4% of hospitalizations for acute renal failure (ARF). CCE occur in men over 50 years of age (mean age: 67 ± 5 years), smokers, hypertensive and in 50% of cases with heart failure. They occur in 87% of cases after a free interval (very short after aortic clamping, 29 ± 24 days after angiography, 90 ± 75 days after anticoagulant treatment) or one of the triggering factors. Renal involvement is the most common after skin involvement and is the major prognostic factor. Usually, renal failure develops in a subacute mode. It worsens in bouts and leads to refractory cardio - renal failure with cachexia. Elsewhere, it appears abruptly a few hours after the triggering factor and immediately threatens life through severe multivisceral damage. It can also develop quietly, in which case the diagnosis is rarely made and the disease is largely under - diagnosed. A renal biopsy may be necessary [4].

Treatment is based on simple principles: discontinuation of anticoagulants, treatment of heart failure (HF) with converting enzyme inhibitor (CEI) /sartan/diuretics and hypertension, extra renal replacement therapy without heparin in case of refractory heart failur or poorly tolerated renal insufficiency [5]. Statins are useful. Corticosteroids appear to improve renal and vital prognosis.

In an effort to stabilize plaques, it is proposed that patients with CCE be treated with antiplatelets and statins, or iloprost in the acute phase of blue toe syndrome [1, 6].

4. Conclusion

The occurrence of CCE is rare, but if it occurs it can be rapidly fatal. The diagnosis is rarely evoked because of the diversity of clinical signs and the evolution which can be insidious. It is necessary to think about it in front of any blue toe with preserved pulses or normal arterial echodoppler of the lower limbs, especially in post vascular catheterism, vascular surgery or aortic clamping and all the more if deterioration of the renal function.

Unstable atheromatous plaques are often the site of fibrin deposition and it is thought that the prescription of anticoagulants and fibrinolytics is likely to destabilize these plaques.

The real treatment must be preventive and in case of CCE occurrence, the treatment is based on simple principles: stop anticoagulants, treatment of heart failure if it exists (CEI /Sartan/diuretics) and hypertension, extra renal replacement therapy without heparin if renal insufficiency is poorly tolerated, reinforce statin treatment and use of corticosteroids.

References

[1] J. Constans. Embolies de cristaux de cholestérol et traitements antithrombotiques. Journal des maladies vasculaires.2010; 35: 301
[2] Elsherbeny AM1, Shawky A, El - Sharkawy M, Mahfouz A. Cholesterol crystal embolization in a Saudi patient after cardiac surgery - - a case report. Middle East J Anaesthesiol. 2010 Jun; 20 (5): 747 - 52.
[3] V. PIRIOU, J. - PH. CLAUDEL, O. BASTIEN, S. ROSS AND J. - J. LEHOT. Severe systemic cholesterol embolization after open heart surgery. British Journal of Anaesthesia 1996; 77: 277–280
[4] M. Roland, E. Guiard, C. Jacquot, A. Karras. Risque rénal et embols de cholesterol (EC). Journal des maladies vasculaires.2010; 35: 301
[5] Smith SC Jr, Allen J, Blair SN, Bonow RO, Brass LM. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update: endorsed by the National Heart, Lung and Blood Institute. Circulation.2006; 1132363 - 2372.
[6] Jucgla A, Moreso F, Muniesa C, Moreno A, Vidaller A. Cholesterol embolism: still an unrecognized entity with a high mortality rate. J Am Acad Dermatol.2006; 55: 786 - 793.

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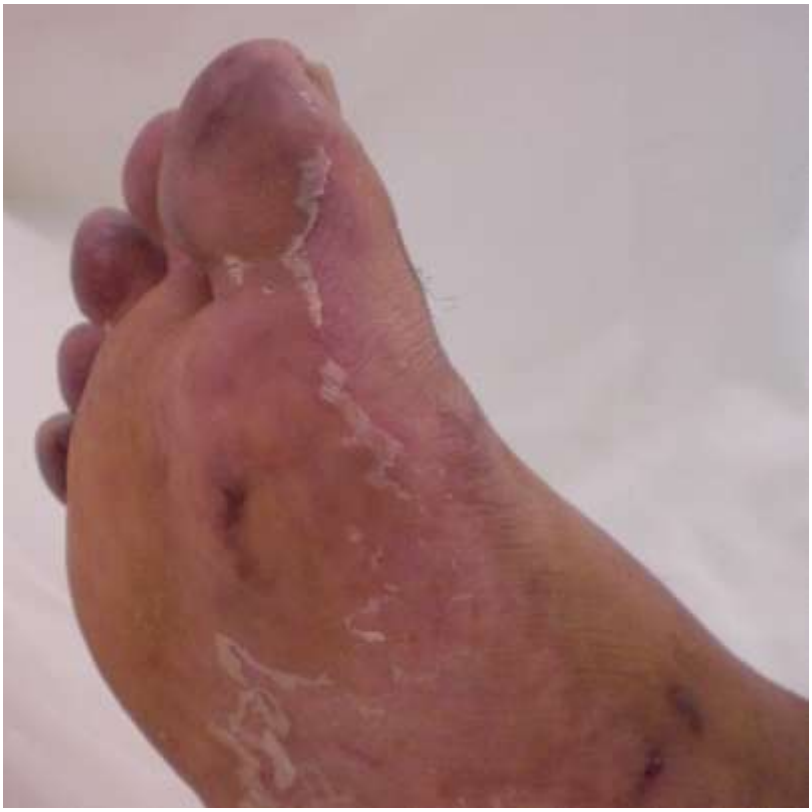

Figure 2: Cyanosis of the toes, lateral surfaces and soles of the feet

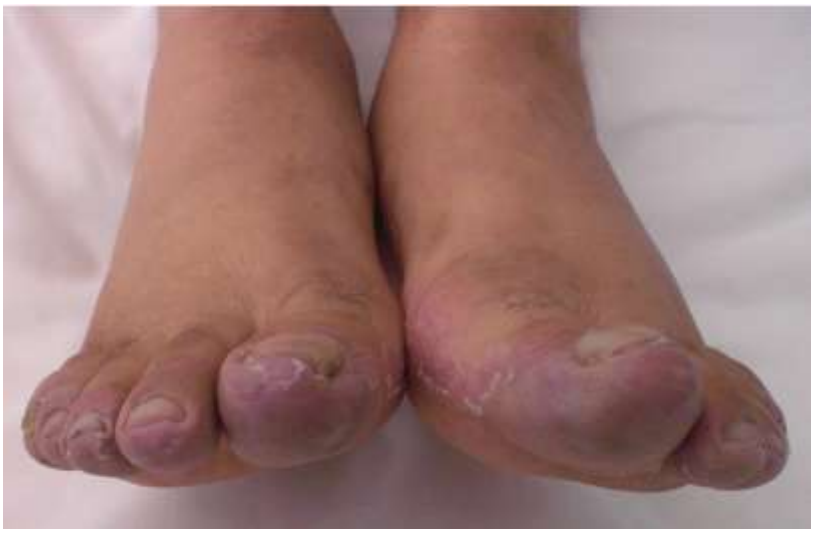

Figure 3: Cyanosis of the toes of both feet

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