

Case Report

Cholesterol Emboli Syndrome — A rare complication of Cardiac Catheterization

Ayaz Hussain Shaikh, Bashir Hanif, Khurshed Hasan, Faiza Malik

Department of Cardiology, Tabba Heart Institute, Karachi.

Abstract

We are reporting the case of a 57 years old male, hypertensive, diabetic, dyslipidaemic who presented with exertional angina. He had a coronary artery bypass surgery, one year ago. He underwent left heart catheterization with graft study which showed critical native triple vessel disease with patent arterial graft to left anterior descending and occluded venous grafts to obtuse marginal and right coronary artery. The procedure was complicated by catheter induced dissection of the ascending aorta. Three days later he developed cholesterol emboli syndrome, that was treated symptomatically.

Introduction

Cholesterol embolization syndrome (CES) is a systemic atheroembolism of cholesterol which can involve brain, eyes, kidneys, and extremities. It is commonly caused by distal showering of cholesterol crystals from aortic atheromatous plaques.¹ CES is a complication of intravascular procedures with a reported incidence of 0.6% - 0.9%.² Our case is typical cholesterol emboli syndrome meeting the "definitive CES" criteria.⁶

Case Report

A 57 years, diabetic, hypertensive and dyslipidaemic male patient presented with recurrent typical anginal symptoms. He had an aortocoronary bypass surgery performed one year back for critical triple vessel coronary artery disease. He had Left Internal Mammary Artery (LIMA) grafted to Left Anterior Descending (LAD) artery and reverse Saphenous Vein Grafts (SVG) to Right Posterior Descending Artery (RPDA) and Obtuse Marginal branches. In view of recurrent anginal symptoms on optimum medications, he underwent coronary angiography. His pre-procedural serum creatinine and total leukocyte counts were in normal range.

Diagnostic cardiac catheterization revealed severe native vessel disease with, occluded venous grafts and patent LIMA to LAD. The procedure was complicated by mild ascending aortic dissection during occluded venous graft injection. He remained haemodynamically stable throughout the procedure and after.

A Transesophageal Echocardiogram was performed to assess the extent of aortic dissection which showed preserved

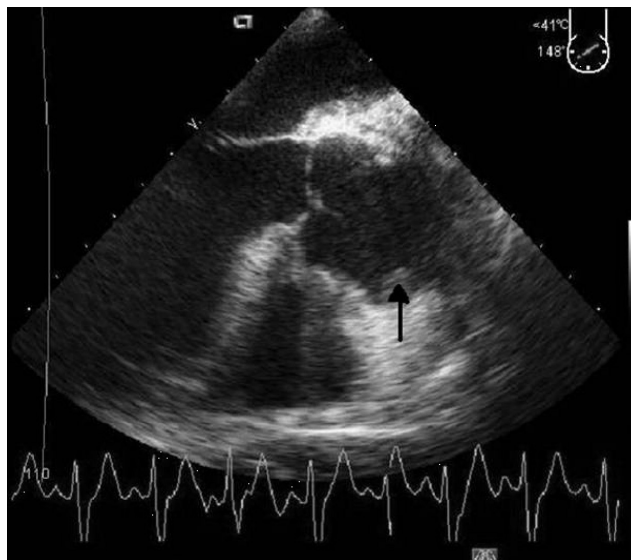


Figure-1: Transesophageal echocardiogram showing Grade III mobile atheroma in ascending aorta (arrow).

left ventricular systolic function with normal chamber dimensions. Ascending aorta and aortic arch were of normal size with grade III mobile plaque in the ascending aorta (Figure-1). No dissection flap or pericardial effusion was appreciated. Doppler examination was unremarkable.

On the third day of procedure patient complained of burning sensation in hand and feet along with low grade fever. Clinical examination revealed bluish discoloration of finger tips and toes (Figure-2a), along with erythematous macular non blanching rash over palms and sole with hyperaesthesia of affected areas (Figure-2b). Peripheral pulses were equally palpable bilaterally and no motor deficit was identified. However, the acral parts were very painful.

Laboratory data revealed a rise in serum creatinine

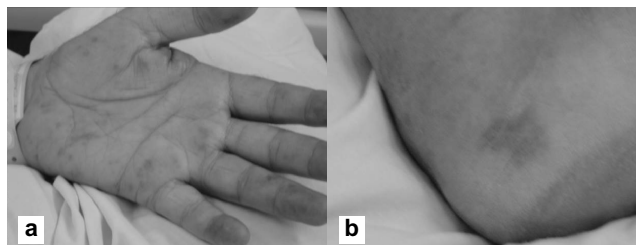


Figure-2a: Showing erythematous, non blanching rash on hand.

Figure-2b: Showing erythematous, non blanching rash on foot.

from normal value to 1.5mg/dl, ESR 65mm, CRP 7.0mg/dl, total leukocyte counts 16,000 with 12% eosinophils. His urine analysis revealed traces of protein. He was monitored in Coronary care unit with analgesics and intravenous hydration. Antianginal medications were optimized as he continued to have recurrent anginal symptoms. His symptoms improved over next one week, rash started fading with reduction in acral cyanosis, and decline in serum creatinine gradually.

He was discharged on 15th day of admission in stable condition with a plan for redo aortocoronary bypass surgery within a week.

Discussion

Cholesterol embolization syndrome (CES) is a systemic atheroembolism involving brain, eyes, kidneys, and extremities, caused by distal showering of cholesterol crystals from aortic atheromatous plaques.¹ Aorta is one of the most heavily involved area with atherosclerotic plaques, therefore, procedures involving mechanical injury by catheters to this region could potentially disrupt plaque material and induce CES.³ Large retrospective studies of patients undergoing intravascular procedures have reported a 0.6% to 0.9% incidence of CES.²

Major risk factors for CES include advanced age, female gender, repeated vascular procedures and peripheral vascular disease. The presence of identifiable aortic atherosclerotic plaque by noninvasive means appears to increase the risk, nearly every organ system has shown histological involvement in autopsy studies.⁴ Renal, neurological, and cutaneous manifestations tend to dominate the clinical picture after vascular interventions. Obstruction of cutaneous vessels results in the mottled purple pattern of livedo reticularis, ulcers, cyanosis, purpura, often referred as blue toe syndrome, purple-toe syndrome, or trash foot.⁵

CES may occur spontaneously but often follows cardiovascular surgery or coronary angiography and angioplasty. Due to the crystal morphology, occlusion of the vessels is not always complete, giving rise to ischaemic atrophic changes rather than necrosis. Subsequently, a "foreign body" type response occurs. The classic triad of syndrome is ischaemic skin lesions, acute renal failure and eosinophilia,⁶ which were all present in our patient.

The criteria for "Definitive" and "Possible" CES have been defined. "Definite CES" is defined as development of cutaneous signs including livedo reticularis, blue toe syndrome, and digital gangrene with or without renal impairment, while "Possible CES" is defined as presence of only renal impairment i.e., a post-catheterization serum creatinine >1.3 mg/dl, two weeks after the procedure in the presence of normal preprocedural renal function without skin lesions.⁶

The diagnosis can be confirmed by biopsy of the skin, muscle or kidney, which demonstrates the characteristic appearance of cholesterol crystals with giant cell, however diagnosis is mostly made on clinical grounds and laboratory tests.⁷ Transesophageal echocardiography can be helpful in demonstrating the anatomy of aorta where mobile atheroma can also be visualized.⁸

Awareness of syndrome and detailed clinical history are of great importance for correct diagnosis. Blood tests reveal renal dysfunction, eosinophilia, which is encountered in 70 to 80% of cases; erythrocyte sedimentation rate is often elevated with urine analysis showing modest proteinuria with or without haematuria.⁹

Treatment options are limited. A conservative approach with the avoidance of any further vascular intervention to prevent embolism recurrence has been considered the best option if possible.¹⁰ Skin lesions are very painful and good analgesia has pivotal importance in treatment. Statins might stabilize plaques, while steroid treatment remains controversial.¹¹

The diagnosis in our patient was based on clinical and laboratory parameters. He classically described painful skin lesions, however renal dysfunction was fortunately not marked and recovery was achieved with conservative therapy.

Acknowledgement

We are thankful to all the staff members of Tabba Heart Institute especially Miss Tadeeb Anwar for her cooperation, assistance and efforts.

References

1. Scolari F, Tardanico R, Zani R, Pola A, Viola BF, Movilli, et al. Cholesterol crystal embolism: A recognizable cause of renal disease. *Am J Kidney Dis* 2000; 36: 1089-109.
2. Nasser TK, Mohler ER 3rd, Wilensky RL, Hathaway DR. Peripheral vascular complications following coronary interventional procedures. *Clin Cardiol* 1995; 18: 609-14.
3. Rosansky SJ, Deschamps EG. Multiple cholesterol emboli syndrome after angiography. *Am J Med Sci* 1984; 288: 45-8.
4. Orr WP, Banning AP. Aortic atherosclerotic debris detected by transesophageal echocardiography - a risk factor for cholesterol embolization. *QJM* 1999; 92: 341-6.
5. Motegi S, Abe M, Shimizu A, Tamura A, Hatori T, Nakano A, Hasegawa A, et al. Cholesterol crystal embolization: skin manifestation, gastrointestinal and central nervous symptom treated with corticosteroid. *J Dermatol* 2005; 32: 295-8.
6. Bashore TM, Gehrig T. Cholesterol emboli after invasive procedures. *J Am Coll Cardiol* 2003; 42: 217-8.
7. Fernandez-Samos R, Suarez D, Ortega JM, Zorita A, Vasquez J, Moran CF, et al. Multiple cholesterol embolization syndromes: a lethal complication of vascular procedures Report of two histologically proven cases. *J Cardiovasc Surg* 1995; 36: 87-91.
8. Sheikhzadeh A, Ehlermann P. Atheromatous disease of the thoracic aorta and systemic embolism. Clinical picture and therapeutic challenge. *Z Kardiol* 2004; 93: 10-7.
9. Izumi C, Kondo H, Tamura T, Inoko M, Kitaguchi S, Himura Y, et al. Clinical evaluation of cholesterol embolization syndrome after cardiac catheterization. *J Cardiol* 1998; 31: 201-6.

10. Meyrier A. Cholesterol crystal embolism: diagnosis and treatment. *Kidney Int* 2006; 69: 1308-12.
 11. Matsumura T, Matsumoto A, Ohno M, Suzuki S, Ohta M, Suzuki E, et al. A case of cholesterol embolism confirmed by skin biopsy and successfully treated with statins and steroids. *Am J Med Sci* 2006; 331: 280-3.
-