Research Article

A Coronary Septic Embolism: An Unusual Complication of Infective Endocarditis.

El Ghacham El Amrani Sihame, Majdoub Kawtar, Fatima-zahra El Hattab, Abakar Bachar, Qat Amin, Raiss Zakaria, Amri Rachida, Zarzur Jamila and Cherti Mohamed.

Departement of cardiology B, CHU Ibn Sina, Mohammed V University, Rabat, Morocco.

We report a case of a 60-year-old man with diagnostic of infective endocarditis involving both the aortic and the mitral valves. Empiric treatment with oxacillin and gentamicin was started. Two days after admission, the patient sustained an acute ST elevation myocardial infarction in inferior. The coronary angiography revealed an occlusion at the end of left anterior descending (LAD) artery without recovery, while the remaining coronary arteries were completely free from any stenosis/atherosclerotic lesion. After a period of further antibiotic therapy, the patient received mitral and aortic valve replacement.

Introduction:
Acute myocardial infarction from septic embolization is a very common complication of infective endocarditis carrying a high risk of mortality and morbidity. Diagnosis is based on clinical, imaging, coronary angiography and blood cultures. The treatment strategy differ from those of atherothrombotic ST elevation myocardial infarction.

In the present article, an unusual case of inferior ST elevation myocardial infarction as a consequence of infective endocarditis is reported.

Case report:
A 60-year-old man, with no cardiac risk factors and had no history of disease, admitted in our emergency for three weeks history of fever. Empiric treatment with amoxicillin + clavulanica acid for one week was initiated at another clinic, but this fever had persisted. At the time of presentation, he was febrile. The cardiovascular examination revealed a murmur of aortic and mitral regurgitation. The electrocardiogram showed a left ventricular hypertrophy and a left atrial hypertrophy (Figure 1).
The biological results found a white blood cell count of 13240/mm³ and C-reactive protein was detected as 135 mg/dl. The blood cultures was negative.

The transthoracic echocardiography revealed a rheumatic severe aortic regurgitation with a small mobile mass compatible with vegetation at the ventricular slope of the right coronary cusp, a rheumatic moderate mitral stenosis with moderate mitral regurgitation, an oscillating mass 14x6 mm at the edge of the anterior leaflet of mitral valve, and moderate tricuspid regurgitation (Figure 2). Left ventricular was dilated with a normal systolic function. Transesophageal echocardiography was performed, confirmed the infective endocarditis. So the patient was treated by antibiotics.

The evolution was marked by the appearance on the second day of treatment of chest pain. The electrocardiogram showed a acute ST segment elevation in the inferior leads with Q waves of necrosis (Figure 3). The troponine level was 6.8 ng/ml. Emergency cardiac catheterization revealed an occlusion at the end of the LAD next to the tip without recovery, while the remaining coronary arteries were completely free from any stenosis/atherosclerotic lesion (Figure 4). The diagnosis of a septic coronary embolism on infective endocarditis was considered for the patient.

After being stabilized with medical treatment, mitral and aortic replacement was performed by the Departement of Cardiovascular Surgery.
Figure 3: Electrocardiogram showed acute ST elevation in inferior leads with Q waves of necrosis.

Figure 4: Left coronary angiography showed an occlusion at the end of left anterior descending (LAD) artery without recovery.

Discussion:
Septic coronary embolism was first established through autopsystudies in patient with infective endocarditis (1,2). It complicates diagnosed infective endocarditis in approximately 7% of cases (3). On the other hand, the discovery of an infective endocarditis following a septic coronary embolism is less frequent.

The most embolizations occurs on the left main coronary and left anterior descending artery due to favorable anatomy. During infective endocarditis, the incidence of embolic complications is greater in mitral valve disease especially for the vegetations present on the anterior mitral leaf left. Besides, the risk of complication is higher in the case of infective endocarditis include the presence of a mobile vegetation, vegetation length of more than 10 mm and infection due to Staphylococcus aureus, Candida, HACEK organisms (Haemophilus, Actinobacillus, Cardiobacterium, Eikinella, Kingella) and Abiotrophia microorganisms. Also, the risk of embolism decreases after the commencement of antibiotictherapy (4).

The optimal treatment of acute myocardial infarction in the context of infective endocarditis is controversial. Thrombolytic therapy is not recommended due to the high risk of intracranial bleeding, because
lesions such as mycotic aneurysms (common in infective endocarditis), have a high risk of bleeding. In addition to the presence of a bacteremia that causes a disturbance of hemostasis in case of sepsis.

It seems safe to perform coronary angiography in the setting of infective endocarditis. Abrupt occlusion of a coronary artery on angiography, despite the patency of other vessels and the absence of collaterals, should suggest the possibility of embolism (5).

There appears to be a trend toward the use of primary percutaneous intervention, however evidence regarding the safety and efficacy of this approach is also limited. Such interventions may be complicated by a high failure rate, which appears to be the case with balloon angioplasty, and increased risk of further embolic phenomena, coronary artery mycotic aneurysm formation or stent infection (5, 6, 7, 8).

Conclusion:--

The patients with infective endocarditis who develop chest pain and ischemic modifications in the electrocardiogram, should suggest the possibility of septic coronary embolism. The management of acute myocardial infarction due to infective endocarditis constitutes a therapeutic dilemma. There is currently little evidence to support systemic thrombolysis or primary percutaneous intervention in this situation. The safest option, as well as possible, is to stabilize the patients with medical treatment and early valvular surgery.

Bibliographie:--

1. Brunson JG. Coronary embolism in bacterial endocarditis. Am J Pathol. 1953;29(4):689–701 [PubMed]
2. Wenger NK, Bauer S. Coronary embolism: review of the literature and presentation of fifteen cases. Am J Med. 1958; 25(4):549–57. [PubMed]
3. Garvey GJ, Neu HC. Infective endocarditis—an evolving disease. A review of endocarditis at the Columbia-Presbyterian Medical Center, 1968–1973. Medicine (Baltimore) 1978;57(2):105–27 [PubMed]
4. Hill EE, Herijgers P, Claus P, Vander schueren S, Peetermans WE, Herregods MC. Clinical and echocardiographic risk factors for embolism and mortality in infective endocarditis. Eur J Clin Microbiol Infect Dis. 2008;27:1159–64 [PubMed]
5. Roxas C.J., Weekes A.J. Acute myocardial infarction caused by coronary embolism from infective endocarditis J Emerg Med. 40 (2011), pp. 509-514 [PubMed]
6. Dubois CL, Desmet W. Septic embolism to the right internal mammary artery causing acute myocardial infarction. Catheter Cardiovasc Interv. 2004; 62:341–2. [PubMed]
7. Glazier JJ, McGinnity JG, Spears JR. Coronary embolism complicating aortic valve endocarditis: Treatment with placement of an intracoronary stent. Clin Cardiol. 1997; 20:885–8. [PubMed]
8. Khan F, Khakoo R, Failinger C. Managing embolic myocardial infarction in infective endocarditis: Current options. J Infect. 2005; 51:e101–5. [PubMed]