Case Report

Interventional Cardiology

Tuberculous pericarditis: An exceptional STEMI association: Case report

Among all the complications of myocardial infarction, pericardial effusion remains correlated with a high morbidity and mortality rate. Tuberculosis in Morocco is a public health problem with 25,000 new cases per year, 47% of which are extra pulmonary, we present an exceptional case of a 70 year old man presenting with an extensive anterior myocardial infarction complicated with tuberculous pericarditis effusion, highlighting the importance of thinking of this aetiology in a specific context.

Keywords: Tuberculous pericarditis; STEMI

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Case Description

A 70 year-old diabetic and hypertensive man with no family history of TB or chronic chest condition, was admitted within 4 hours of an extensive anterior ST elevation myocardial infarction (STEMI) (Figure 1). He was immediately transferred to the cathlab for primary percutaneous intervention. Coronary angiography analysis showed an occlusion of the mid left anterior descending artery (LAD) (Figure 2). The case was manged by stenting with predilatation with a satisfying result (Figure 3). Thransthoracic echocardiography (TTE) showed an akinesia in the territory of the LAD (apex, anteroseptal, anterior and anterolateral segments) with mild systolic left ventricle dysfunction. During the hosptalisation, we noted the onset of dyspnea and bilateral lower limb swelling of lower limb with no friction rub. Systematic TTE revealed small to moderate pericardial effusion but no sign of tamponade, with elevated left ventricular (LV) filling pressure. We decided to manage the case as myocardial infarction complicated by congestive heart failure by salt restriction and diuretics. Patient showed clinical improvement of his condition and was discharged with a slight pericardial effusion, with a close follow-up in consultation. Three months later, the patient was seen in consultation for dypnea at rest with signs of right heart failure, fever, night profuse sweating and a weight loss amounts to 12 kg. The chest radiography revealed a pleural effusion with cardiomegaly, without pericardial calcification. TTE showed a circumferential abundant pericardial effusion, without evidence of tamponade with a persistent mild systolic left ventricle dysfunction and normal (LV) filling pressure (Figure 4). Pericardial liquid analysis revealed positive QuantiFERON-TB assay (7.95 UI/mL) and elevated Adenosine Desaminase level. Microscopic examination was not contributive. Serology showed negative HBV, HCV, HIV and syphilis. The patient was treated by ant tuberculosis chemotherapy. Successive TTE revealed the disappearance of the pericardial effusion after 2 months of treatment.

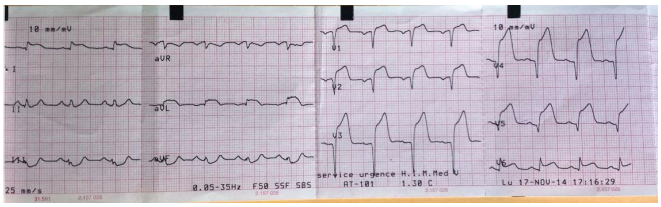

Figure 1: A 12-leads ECG showing extensive anterior STEMI.

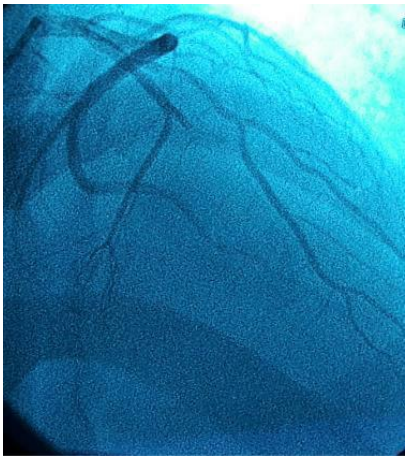

Figure 2: Coronary angiography showing an occlusion of the mid left artery descending (LAD).

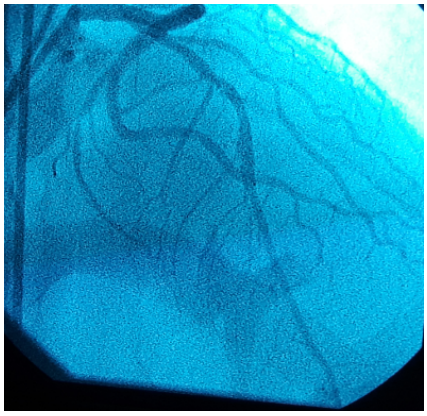

Figure 3: Successful angioplasty of the occluded LAD with the drug eluting stent deployed.

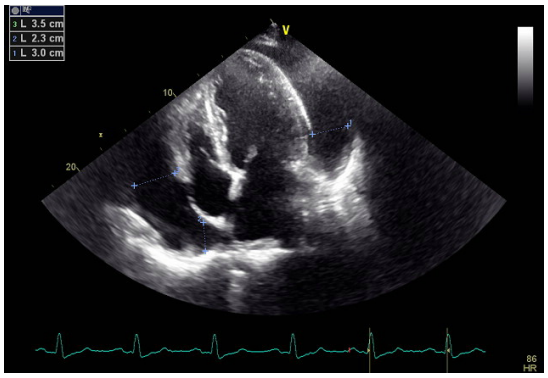

Figure 4: Apical view in transthoracic echocardiography showing the circumferential pericardial effusion with no tamponade sign.

Discussion

Tuberculosis (TB) is one of the most widespread infections in the world [1], and considered as public health problem with 9 million cases in the world in 2012 [1], and 27.000 new cases per year in Morocco, with a non-negligible prevalence of 14% of the extrapulmonary form [2]. Tuberculous pericarditis (TBP), remains one the main cause of hospitalization pericardial effusion and heart failure in developing countries [3]. Three forms of presentation of tuberculous pericarditis: The effusive form (80%), the constrictive (5%), and the effusive-constrictive pericarditis (15%) [4]. Clinical presentation is heterogeneous. The symptomatology may variate from simple coughing to exertional dyspnea with congestive heart failure, orthopnea and lower limb swelling. Common symptoms may also include fever with night sweats, weight loss [5-7]. Signs of chronic right heart failure due to effusive cardiac compression remains the most common type of presentation [6,8]. Making the diagnosis of TBP relaying only on symptomatology remains a difficult task, indeed signs and symptoms are generally not specific the echocardiography is the gold standard to make a certain diagnosis of the pericardial effusion. Computed tomography (CT) and Magnetic resonance imaging (MRI) are more efficient but they may generally be missing in developing and poor countries [5-7]. The chest radiography may look for pulmonary lesions suggestive of active tuberculosis, pleural effusion and cardiomegaly [9,10]. In the majority of cases of TBP, the ECG is abnormal [11]. The modifications concerns usually the ST-T–wave but they remain nonspecific [11,12]. The ST and PR-segment variations are found in 10% to 12% of cases [12]. The presence of microvoltage has a good sensibility but is not specific of the pericardial effusion [12]. Less frequently transient atrial fibrillation occurs in 4% of cases, and electrical alternans which is more specific of swinging heart and cardiac tamponade [9]. All this electrical abnormalities can be hidden in case of ST elevation myocardial infarction (STEMI). The fibrinous strands as seen in TTE are not specific for a tuberculous etiology [9,13].

There are two methods diagnosis of TBP: Direct and indirect methods: The Direct Method is Pericardiocentesis which is recommended every time TBP is suspected. Macroscopic analysis finds blood stained fluid in 80% of cases. Microscopic analysis shows typically exudative fluid with high protein content, cytology is remarkable for a predominance of monocytes and lymphocytes count. Light's criteria [14] remain the most accurate way to identify pericardial exudates effusions [15]. Anatomopathology of the pericardial biopsy specimen make certitude diagnosis of TB, but thoracotomy and all its complications with increased morbidity and marked prolongation of the hospital stay may limit the procedure [16]. However the sensitivity diagnostic of pericardial biopsy for TP ranges only from 10% to 64% [17,18]. In other more valuable diagnosis tool, we find the polymerase chain reaction (PCR) for detecting Mycobacterium tuberculosis DNA in pericardial effusion fluid [19]. In tuberculous endemic countries, the high incidence of TB, the probability of cross-sensitization from mycobacterium, and mass

BCG vaccination, make tuberculin skin testing not very useful [20].

The other method of TBP diagnosis is the indirect one: Dosing the Adenosine desaminase (ADA) activity in pericardial liquid can make the diagnostic of TBP as showed in several studies [21-23]. Elevated ADA activity ranging from 30 to 60 U/L is suggestive of tuberculosis with a specificity and sensitivity diagnosis rate of 74% and 90% [23]. High levels of the ADA are known to be a reliable prognostic indicator for the development of constrictive pericarditis in TBP. Pericardial lysozyme is also described as a diagnostic tool for TBP. Cutoff levels of 6.5 μg/dL has been described to make the diagnosis of TBP with a sensitivity and specificity rates of 100% and 91% respectively [22]. Elevated interferon-γ (IFN-γ) levels in pericardial liquid can be used in early diagnosis of TBP with a sensitivity and specificity rate respectively of 92% and 100% [22]. Being a rapid and highly specific test of TBP diagnosis, the IFN-γ is now the test of choice for the diagnosis, but technical and financial constraints may limit its use in many developing countries [23].

Before the advent of antibiotics mortality was 80 to 90%, with the antituberculosis chemotherapy, mortality rate decreased significantly [24]. The mortality rate ranges from 8% to 17% in HIV-negative patients [25,26] and 17% to 34% in HIV-positive patients [27]. The antituberculosis protocol consists on a quadruple therapy of pyrazinamide, rifampicin, ethambutol, and isoniazid, for two months, followed by isoniazid and rifampicin for four months [28,29]. No data had shown advantages of lengthening treatment duration although for 9 months or longer, on the contrary some cases of treatment poor compliance was described [29]. The effectiveness of Short-course chemotherapy is also proved in TBP in HIV-positive patients [30]. Association of corticosteroids was described to be effective in constrictive form but remains controversial in the effusive form of tuberculous pericarditis [31].

Conclusion

Early pericardial effusion due to left ventricular free wall rupture and congestive heart failure are the main causes for pericardial effusion post-ST-elevation MyocardiaI infarction (MI). It is important to think about tuberculosis etiology in a context of endemic countries.

Executive Summary

Among all the complications of myocardial infarction, pericardial effusion remains correlated with a high morbidity and mortality rate.

Tuberculosis in Morocco is a public health problem with 25,000 new cases per year, 47% of which are extra pulmonary.

We present an exceptional case of a 70 year old man presenting with an extensive anterior myocardial infarction complicated with tuberculous pericarditis effusion, highlighting the importance of thinking of this aetiology in a specific context.

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