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CASE REPORT

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Right atrial tuberculoma: A diagnosis too late

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Abstract

Solitary intra-cardiac cavity tuberculoma is extremely rare and often only diagnosed during a post-mortem. We report a case of right atrial tuberculoma causing right atrial outflow tract obstruction in an immune-compromised man. The diagnosis of cardiac tuberculoma was made through the detection of mycobacterium tuberculosis DNA by tuberculosis-polymerase chain reaction in the pericardial fluid. The patient succumbed five days after admission but an autopsy was refused by his family. (Cardiol J 2011; 18, 5: 560–563)

Key words: cardiac mass, tuberculoma, HIV/AIDS, tuberculosis, polymerase chain reaction, pericardial effusion

Introduction

Extra-pulmonary tuberculosis accounts for 15–20% of mycobacterial infections. The commonest sites are the lymph nodes, pleura, abdomen and central nervous system [1]. Apart from the pericardium, the cardiovascular system is an extremely rare site of involvement. Myocardial tuberculosis was first reported in 1761 by Morgagni [2, 3]. Although tuberculosis is prevalent in Malaysia, an incidence of TB myocardium has never been reported. We report a 35 year-old immune-compromised Malay man who presented with right heart failure and echocardiographic finding of a solitary right atrial mass. He unfortunately succumbed to the complication of right atrial outflow obstruction after five days. The final diagnosis was made after obtaining a positive pericardial fluid tuberculosis-polymerase chain reaction (TB-PCR) result, as autopsy was not consented to.

Case report

A 35 year-old Malay man was referred for progressive dyspnoea over one month. This was associated with poor appetite and significant weight loss of 4 kg. He also complained of intermittent low grade fever for one week with worsening of orthopnoea, paroxysmal nocturnal dyspnoea, leg swelling and reduced effort tolerance for three days prior to admission. He was diagnosed as having HIV and having contracted hepatitis C three months prior to admission. He had been intravenously injecting illicit drugs, but had stopped this about one year previously. There was no family history of TB or recent contact with a tubercular patient.

He was in respiratory distress but alert at admission. He was tachypnoeic and required nasal prong oxygen supplement to achieve good oxygenation. He was bradycardic, with a pulse rate of 43 beats per minute but was hemodynamically stable with blood pressure of 120/88 mm Hg. He showed signs of heart failure evidenced by elevated jugular venous pressure with cannon 'a' wave, gross leg oedema, ascites and pulmonary oedema. The praecordium examination revealed displaced apex beat with distant heart sounds. There were no stigmata of chronic liver disease. There was no lymphadenopathy and a Bacillus Calmette-Guerin scar could

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be seen on the left forearm. Apart from that, he was also slightly dehydrated and oral thrush was present.

Laboratory investigations showed hypochromic microcytic anaemia (hemoglobin 10.6 g/dL) with elevated reticulocyte count. Leucocytosis was present with neutrophilia (leukocyte count 13.06 × × 10^9/L and 89.5% neutrophils). There was no monocytosis. Thrombocytopenia was also noted. Serum electrolytes, glucose and creatinine were normal. Alkaline phosphatase was mildly elevated (201 μ/L) and the transaminases were markedly raised; AST 1006 μ/L and ALT 292 μ/L. Serum creatinine kinase and lactate dehydrogenase were also increased, 578 μ/L and 1593 μ/L respectively. Hypoalbuminaemia (25.0 g/L) was present with an elevation of gamma globulin (36.1 g/L). The erythrocyte sedimentation rate was within normal range (13 mm/h). The coagulation profile was markedly prolonged (INR 5.0 and APTT ratio 1.8). Tumor markers were normal. The electrocardiogram showed small QRS complexes with complete heart block. A chest radiograph revealed globular heart with left sided pleural effusion. The mediastinum was not widened. The lung field was not suggestive of tuberculosis infestation or miliary TB (Fig. 1). Abdominal ultrasonography showed hyperechoeic hepatomegaly with ascites. The echocardiogram showed massive pericardial effusion with a diameter of 3.0 cm. There was evidence of cardiac tamponade with right ventricular collapse during diastole. A right atrial mass measuring 5.0 × 5.0 cm was found obstructing the tricuspid inflow. The mass arose from the right atrial free wall infiltrating into the myocardium and pericardial surface (Fig. 2). The inferior vena cava was dilated and clear of thrombus.

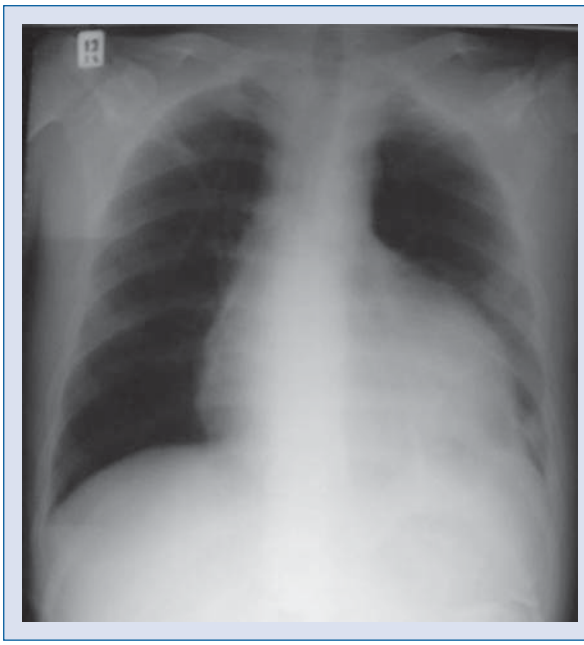

Figure 1. Chest X-ray of the patient showing globular heart with left pleural effusion. The mediastinal is not widened and lungs parenchyma was normal.

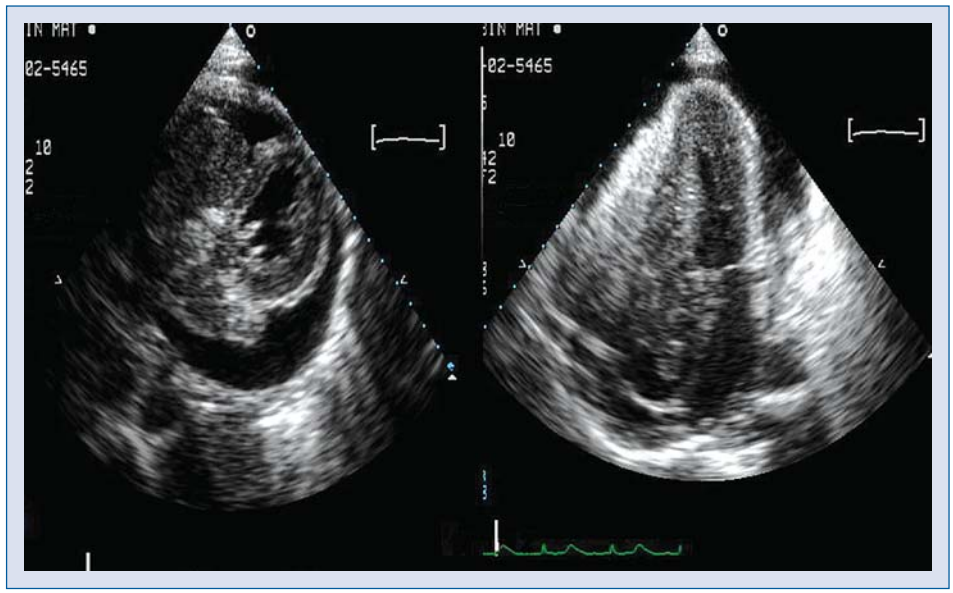

Figure 2. Transthoracic two-dimensional echocardiography showing a homogenous, well-circumscribed and defined mass obstructing the tricuspid inlet at parastemal short axis view and apical four-chamber view.

Figure 3. Bottle showing the pericardial fluid which is yellowish-coloured and remained clotted *ex-vivo*. The mycobacterium tuberculosis polymerase chain reaction was positive from the fluid.

Pericardial aspiration was performed and the pericardial fluid was sent for laboratory analysis. The initial sample showed a gelatinous fluid which clotted on contact with air (Fig. 3). Pericardial fluid analysis showed mature lymphocytes and foamy macrophages with reactive mesothelial cells. Centrifugation of the pericardial fluid did not yield any malignant cell. A diagnosis of myocardial tumor with an inter-current pneumonia was initially entertained. The patient was started on carbapenem antibiotic while waiting for myocardial biopsy. However, he rapidly deteriorated, requiring intubation and mechanical ventilation. He succumbed on the fifth day after admission. We were unable to biopsy the mass or perform an autopsy examination as the family did not consent to either. The pericardial fluid for TB-PCR detected mycobacterium tuberculosis DNA.

Discussion

Tuberculosis presenting as an intra-cardiac mass is an unusual finding, especially in immunocompetent persons. The differential diagnosis of right atrial mass includes thrombus, right atrial myxoma, lymphoma, myosarcoma, rhabdomyosarcoma, vascular tumor and secondary deposit. In Western case series, lymphomas and secondary deposits are the more common causes, followed by a primary cardiac tumor like myxoma. Therefore, even in our patient, the diagnosis of tuberculoma was not initially suspected. The striking feature of our patient was the absence of pulmonary tuberculosis, with the heart being the primary site. A clearly visible mass arising from the right atrium free wall infiltrating the myocardium may lead one to suspect cardiac tumor rather than tuberculoma.

Tuberculosis is again prevalent due to the increasing number of HIV cases worldwide. In our country, the incidence of intracardiac tuberculosis is difficult to ascertain. From our literature search, we believe there had been no previously reported case of cardiac tuberculoma in Malaysia.

Primary TB of the heart is uncommon. Tuberculosis pericarditis has been reported in up to 1% of patients presenting with active pulmonary tuberculosis, while involvement of other parts of the heart is extremely rare [4]. Cardiac tuberculomas have been reported (mainly during post-mortem) in fewer than 0.3% of all patients prior to the introduction of anti-TB therapy [3, 5]. Isolated cardiac tuberculomas as in our case are extremely rare [6]. Single or multiple cardiac tuberculomas are often visualised in the right cardiac chambers, particularly in the free wall of the right atrium [7]. They are usually solitary, well defined and sharply demarcated from the surrounding tissue. The mass may infiltrate the underlying myocardium, causing ulceration of the surface leading to thrombus formation and subsequent thromboembolism. This phenomenon may give rise to hematogenous seeding and disseminated tuberculosis. Cardiac tuberculoma can be found in all four cardiac chambers and simultaneous manifestation of tuberculomas at different locations has been reported. The right heart chambers, particularly the right atrium free wall, are often affected, probably because of the frequent involvement of the right mediastinal lymphatic drainage with consequent spread to the pericardium and myocardium [8].

Several mechanisms may explain the involvement of the heart in active tuberculosis. The involvement of the myocardium could be the result of extension of the pericardial TB, infiltration from the mediastinal lymph node or hematogenous dissemination. Thus far, three distinct histological subtypes of myocardial TB have been recognized. The commonest is the diffused infiltrating form which is characterized by microscopic giant cells and lymphocytes. Hematogenous dissemination

results in miliary form appearance. The last subtype is the nodular form which is characterized by central caseation.

In the past, cardiac tuberculoma was exclusively diagnosed at necropsy. Advances in medical technology have allowed us to make an early diagnosis. Transthoracic and trans-oesophageal echocardiography, computed tomography and magnetic resonance imaging scanning are non-invasive and may contribute to optimal morphologic visualisation and description. However, they cannot help in distinguishing a tuberculoma from an intra-cardiac tumor.

Generally, tissue diagnosis by endomyocardial biopsy and Ziehl-Neelsen staining are the most specific tests. However, this often fails to reveal the acid fast bacilli, and a definitive diagnosis depends on seeing the typical histological changes. A culture of the organism can be done for definitive diagnosis. In addition, PCR for mycobacterium tuberculosis DNA can confirm the suspected diagnosis. The diagnosis of our patient was made only after obtaining TB-PCR from the pericardial fluid, as we were unable to perform the biopsy; the diagnosis was made from finding mycobacterium tuberculosis DNA in the sterile pericardial fluid and the supportive cellular finding of mature lymphocytes and foamy macrophages in the pericardial fluid. The patient was also an immunocompromised individual who was at high risk of tuberculosis infection.

Reported cases of tuberculoma show that it can present in many clinical manifestations. They may be asymptomatic or may present with arrhythmias [6, 9, 10]. They can also cause severe hemodynamic disturbances resulting from acute severe aortic regurgitation when ulceration of the aortic annulus occurred. Other mechanisms include mitral regurgitation caused by submitral tuberculoma, coronary artery occlusion, superior vena cava obstruction, formation of ventricular aneurysm or rupture of ventricular free wall. Tuberculoma involvement of the right atrium is commonly present with atrial fibrillation or flutter. In our patient, the tuberculoma caused obstruction of the tricuspid inlet, therefore leading to the devastating right heart failure and his eventual demise.

Complete regression of tuberculoma treated with standard anti-tuberculous chemotherapy has been reported [11]. Surgical intervention may be required in large tuberculoma when pharmacotherapy alone is inadequate. The timing of surgical resection depends on the hemodynamic status of the patient. Early resection may be required in cases when the tuberculoma is causing hemodynamic instability or when the diagnosis is uncertain. In our patient, we were unable to proceed with surgical resection due to the rapid deterioration of the patient, hindering transfer to the tertiary center.

Although cardiac tuberculoma is an extremely rare entity in modern clinical practice, it should be suspected in patients presenting with intra-cardiac mass who have risk factors for tuberculosis, or have had exposure to tuberculosis. Given a prompt diagnosis, these patients can be successfully treated with standard anti-tuberculous chemotherapy, although the duration of treatment remains a subject of debate. In an era of an increasing number of acquired immune-compromised patients, and with increasing numbers of diagnoses of tuberculosis, a diagnosis of intracardiac tuberculoma should be considered in patients presenting with intracardiac mass which is mimicking a tumor. Our patient is a case where such a diagnosis came too late.

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