Asian Pac. J. Health Sci., **2019; 6(2):152-156** **e-ISSN: 2349-0659, p-ISSN: 2350-0964**

Document heading doi: 10.21276/apjhs.2019.6.2.22 **Case Report**

Miliary Tuberculosis and Acute Myocardial Infarction with left ventricular aneurysm in a 73 year old male seen at the National Hospital in Timor-Leste: A case report

C. Babua*, A. Monteiro, L. M. Dos Reis Seixas, J. Da Costa, H. Joao , C. A. G. Dos Santos

[1]Royal Australasian College of Surgeons
[2]Hospital Nacional Guido Valadares

Received: 15-04-2019 / Revised: 20-5-2019 / Accepted: 25-06-2019

Abstract

Background: The developing world is currently facing a double epidemic of communicable and non-communicable diseases. There is a growing body of knowledge recognizing an interaction between the two epidemics. Here we report a case of active pulmonary tuberculosis and acute myocardial infarction.
Case presentation: A 73 year old male who had recently been diagnosed with hypertension but with an otherwise unremarkable past medical history presented to the emergency department of the Timor-Leste National Referral Hospital with chest pain and shortness of breath of one weeks duration. There was a positive history of intermittent fever, dry cough and weight loss over the preceding 3-4 weeks. His 12-lead electrocardiogram showed ST-segment elevation in the inferior leads and chest X-ray was characteristic of miliary tuberculosis. Echocardiography showed low ejection fraction and left ventricular inferior wall aneurysm. Cardiac enzymes as well as cardiac catheterization were not available in the hospital. He was managed with medical therapy for myocardial infarction without reperfusion therapy. He initially showed improvement in his clinical parameters (Chest pain, Heart rate, Blood pressure, Oxygen saturations, and fever) but succumbed to sudden cardiac arrest after 10 days of hospital admission. Post mortem examination was not done. **Conclusion**: The presentation of acute myocardial infarction in this patient with active tuberculosis and an otherwise benign past medical history is in support of reports that tuberculosis plays an active role in the pathogenesis of acute myocardial infarction in some patients presenting with these two diseases.
Keywords: Acute myocardial infarction, Aneurysm, Electrocardiogram, Miliary Tuberculosis, Timor-Leste.

Introduction

Previously known to be afflicted predominantly with communicable diseases, and now with an increasing burden of non-communicable diseases(NCDs), the developing world is currently facing a double epidemic (communicable and non-communicable diseases)[1, 2]. Some of the most important communicable diseases in this regard include malaria, Human Immune Deficiency Virus/Acquired Immune Deficiency Syndrome (HIV/AIDS) and tuberculosis (TB). The emerging epidemic of NCDs in the developing world includes increasing burden of hypertension, cardiac diseases, stroke, chronic obstructive pulmonary diseases (COPD) and Chronic Kidney Disease (CKD) among others.

*Correspondence
C. Babua
Royal Australasian College of Surgeons
E-Mail: b2kriss@gmail.com

Various reasons have been advanced for this change in epidemiology, mainly an aging population coupled with urbanization and acquisition of a more western lifestyle[2]. This has resulted in an increase in the traditional cardiovascular risk factors including hypertension, diabetes mellitus (DM), dyslipidemia and obesity. However the increase in these traditional risk factors appears not to fully account for the increase in cases of ischemic heart disease thus necessitating the search for additional risk factors. Observational studies and case reports have been published previously suggesting significant interaction between communicable and non-communicable diseases. Infectious diseases including viruses and TB have been implicated as contributory factors in the chronic inflammatory process associated with athero-sclerosis [3-6]. Furthermore tuberculous coronary arteritis has been reported in at least one case of fatal myocardial infarction presenting with left ventricular aneurysm[7]. Here we report the case of a 73 year old male who presented to the emergency department with chest pain

and was diagnosed with inferior ST-elevation acute myocardial infarction and miliary TB.

Case report

A 73 year old male was seen at the emergency department of Hospital Nacional Guido Valadares (HNGV) in December 2017 with complaints of a non-radiating crushing central chest pain, moderate to severe in intensity for one week associated with shortness of breath, worsened by only minimal physical activity. There was positive history of dry cough, fever, drenching night sweats and progressive weight loss in the preceding 3-4 weeks. Other symptoms included nausea, dizziness and general body weakness. Micturition habits were normal and review of the central nervous system was unremarkable. He had been recently diagnosed with hypertension and started on nifedipine at a peripheral health centre (blood pressure control could not be ascertained). There was no history of dyslipidemia or diabetes. There was no family history of cardiac disease or any other familial illnesses. No history of cigarette smoking, alcohol consumption or use of any other recreational substances. On physical examination he was noted to be ill looking in moderate painful distress, non-obese, wasted but with no pallor of mucous membranes or palms, no jaundice and no enlarged lymph nodes. He had no peripheral edema and no stigmata of immune deficiency. On admission at the emergency unit, vital signs were within normal limits as follows: blood pressure: 131/67mmHg, heart rate: 90 beats/min., respiratory rate: 18 breaths/min., SPO_2:91 % (on room air), and temperature: 36.2˚C.

He had no jugular venous distension, heart sounds were normal and lungs clear. Abdominal examination and central nervous system evaluations were unremarkable. A complete blood count obtained showed a normal hemoglobin concentration of 152 g/L, mild leucocytosis of 11.6 X 10^9/L, and normal platelet count of 158 X 10^9/L. He had normal random blood glucose of 4.1 mmol/L, sodium was low at 133mmol/L with normal potassium of 4.8mmol/L. Urea and creatinine were mildly elevated at 7.5mmol/L and 132µmol/L respectively. Chest X-ray showed diffuse bilateral millet seed like infiltrates (Fig. 1) Consistent with miliary T.B.12-lead electrocardiogram (ECG) showed ST-segment elevation with pathological Q-waves in inferior leads (leads II, III and aVF) with reciprocal ST-segment depression in the anteroseptal leads (V1-4) (Fig. 2 and 3).

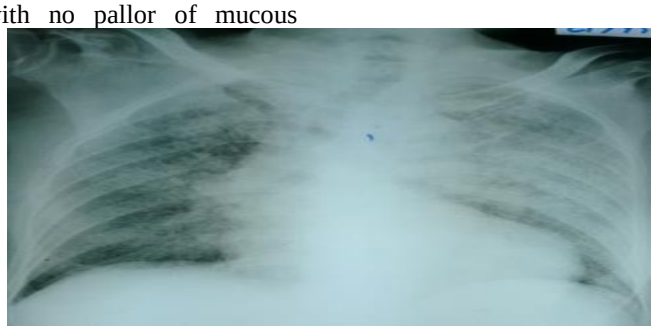

Fig. 1: Chest X-ray on admission

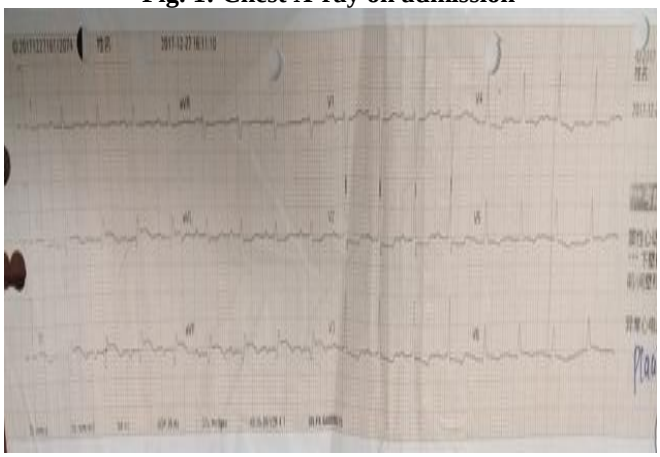

Fig. 2: 12 lead ECG obtained on admission

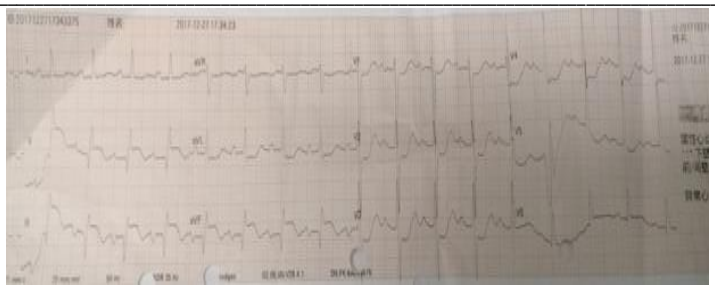

Fig. 3: 12 lead ECG obtained 2 hours after admission

Bedside echocardiography performed by the cardiologist showed Spontaneous Echo Contrast (SEC) in the left ventricle with impaired systolic function (left ventricular ejection fraction of 20%) and left ventricular inferior wall aneurysm. Cardiac enzymes and lipid profiles were not available at the hospital. He was treated with dual antiplatelet therapy using aspirin and clopidogrel as well as an anticoagulant(enoxaparin), analgesics (morphine), oxygen, simvastatin, intravenous fluids (normal saline) and anti-tuberculous medication (isoniazid, rifampicin, pyrazinamide and ethambutol combination tablets). Other medications included an angiotensin converting enzyme inhibitor (captopril) and a potassium sparing diuretic (spironolactone). On day 2 of admission, patient reported a reduction in the intensity of chest pain to mild and non-persistent. He was noted to be having a fever of 39° C, other vital signs remained normal with blood pressure: 131/87mmHg, heart rate: 100 beats/min., respiratory rate: 20 breaths/min., SPO_2 :95 % (on three litres of oxygen via nasal canula). On third day of admission, patient reported feeling better, fever free with only mild chest discomfort. Apart from low oxygen saturation (88%, corrected upon increasing flow from three to five litres by nasal canula), all vital signs were within normal limits. The following three days were largely uneventful with only low grade fevers and largely pain free. Repeat ECG showed return of the ST-segment towards the iso-electric line with persistence of the Q-waves in the inferior leads during the entire period of admission (Fig 4).

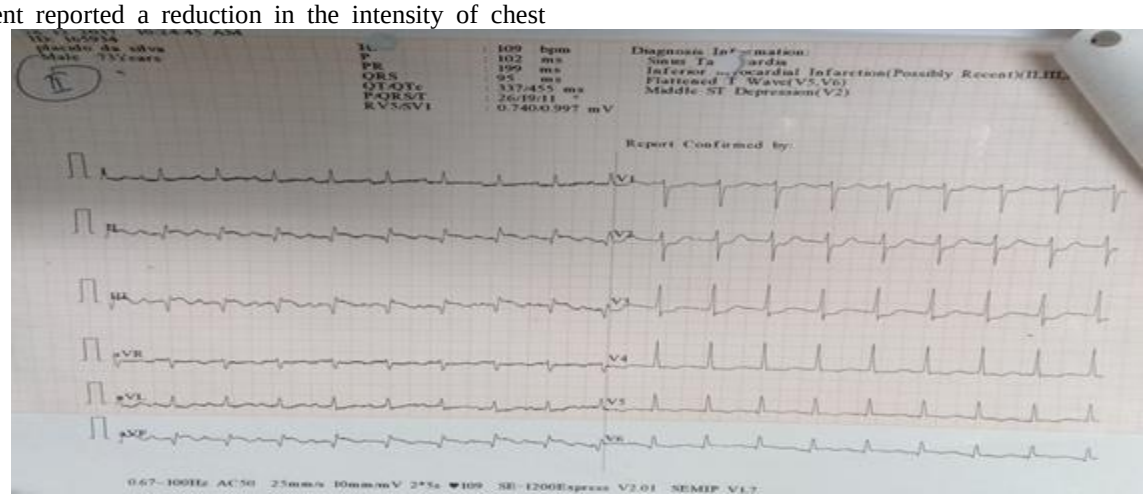

Fig. 4: 12 lead ECG on day 3 of hospital admission.

On day seven of admission, patient developed worsening of shortness of breath with low oxygen saturation at 77% and elevated blood pressure of 167/112 mmHg and tachycardia of 146 beats/min. These were corrected by switching to oxygen by mask and escalation of dose of angiotensin converting enzyme inhibitor (ACEI). Three days later (day 10 of admission) the patient died due to sudden cardiac arrest. Post mortem was not done.

Discussion

According to the Timor Leste ministry of health, Tuberculosis (TB), both pulmonary and extra pulmonary, is endemic in the country with 139 new smear positive cases notified in 2002 per 100,000 of the population [8]. A more recent publication by the World Health Organization (WHO)puts the burden of TB in Timor Leste at 789 per 100 000 population [9]. Although a common presentation at the national hospital, the exact burden of ischemic heart disease

including acute myocardial infarction is not known. Infectious diseases including TB are increasingly being recognized as contributory factors in atherosclerosis and thus cardiovascular disease (CVD). Two main mechanisms have been suggested including increase in inflammation resulting in plaque formation and/or rapture and autoimmune phenomena[10].Our patient had some traditional risk factors for myocardial infarction including male gender, older age and hypertension and was diagnosed with active TB. There are several case reports of active TB and acute myocardial infarction including one case of a young female from Kosova with no known risk factors for ischemic heart disease suggesting TB as a potential pathogenic factor[11]. The presentation of TB with acute myocardial infarction (AMI) and aneurysm of the cardiac wall has as well been reported previously. One such case is of a 19 year old male who presented with tuberculous inflammation of the epicardium and endocardium (as seen on postmortem) with involvement and subsequent occlusion of the anterior descending coronary artery. The patient developed ventricular aneurysm as a complication of acute myocardial infarction with subsequent fatality [7]. The current case presented with X-ray evidence of miliary TB and ST-elevation myocardial infarction (STEMI) involving the inferior wall and left ventricular aneurysm (as seen on echocardiography). The patient did not receive reperfusion therapy but initially showed improvement in symptoms as well as ECG changes before sudden death on day 10 of hospital stay. Multiple factors contribute to mortality among patients with acute myocardial infarction, sudden death as in this case could be due to ventricular arrhythmias or cardiac rupture (especially in a patient presenting with aneurysm of the ventricular wall). The most accurate way to diagnose TB is either by microbiological test (microscopy or culture) or through histology. A chest X-ray is another useful tool for pulmonary TB especially in situations where microbiological and histological tests are not available or patient does not have sputum as in the current case. Furthermore miliary TB is usually a radiological diagnosis based on X-ray finding of millet seed-like infiltrates in the bilateral lung fields. In this case the typical history of several weeks of cough with fevers, night sweats and progressive weight loss in a country with high TB prevalence was suggestive of the diagnosis. A typical X-ray with a miliary pattern and response to anti TB with resolution of fever at the end of first week of therapy helped to refine the accuracy of the diagnosis of TB. With similar previous report of TB presenting with AMI and left ventricle (LV) aneurysm, more studies are needed to determine whether TB increases risk of cardiac aneurysm in patients presenting with acute myocardial infarction. Furthermore studies are needed to outline the precise mechanisms of pathogenesis of AMI in patients presenting with active TB. This is potentially helpful in modifying treatment strategies (potential role for anti-inflammatory agents) for patients presenting with this combination of disease entities and especially for developing countries still facing a double burden of both communicable and non-communicable diseases. There were several limitations in the current case ranging from inadequate diagnostic tests (lipid profiles, cardiac enzymes) to patient management (lack of reperfusion therapy, cardiac intensive care unit). However with the limited diagnostics we were able to confirm the diagnosis and initiate therapy, other risk factors for ischemic heart disease including dyslipidemia could have been missed. Lack of a cardiac unit with reperfusion therapy and cardiac monitoring facilities could have further contributed to the fatal outcome in this case. A post mortem examination in this case was as well a missed opportunity that could have further elucidated the potential mechanisms of cardiac injury and cause of death.

Conclusion

With infectious agents increasingly being recognized as possible contributors to the chronic inflammatory process associated with acute coronary syndromes, it is likely that the occurrence of miliary TB and acute myocardial infarction in this 73 year old male with a relatively benign past medical history was not a coincidence but rather a case of Acute Myocardial Infarction with Tuberculosis as a contributing risk factor. The finding of a left ventricular aneurysm on echocardiography similar to one reported previously may be suggestive of TB as a risk factor for this complication.

Acknowledgements

The authors acknowledge the contribution of all HNGV staff who participated in the care of the patient reported and thus helping to generate all clinical and investigation data published in this report.

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Conflict of Interest: None
Source of Support: The Royal Australasian College of Surgeons (RACS) with funding from the Australian Government