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Original Research Paper

Pathology

A RARE CASE OF CARDIAC TUBERCULOSIS WITH INCIDENTAL FINDING OF MYOCARDIAL BRIDGING

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ABSTRACT Cardiac tuberculosis and myocardial bridging both are an extremely rare finding. Myocardial involvement by tuberculosis is less common and mostly associated with pericardium. Morphologically three forms of cardiac tuberculosis seen are nodular, miliary and diffusely infiltrative form. Myocardial bridging is a congenital anomaly where a segment of a major coronary artery takes an intramuscular route and tunnels through the myocardium for some distance before returning to its normal course. Clinically myocardial bridging is difficult to diagnose and often detected incidentally, at autopsy. We hereby present a rare autopsy case in a 31 year old female, where we discuss about cardiac tuberculosis and myocardial bridging and there possible outcomes.

KEYWORDS : Myocardial bridging, cardiac tuberculosis, myocardium

1. INTRODUCTION

Tuberculosis can be seen in all age groups and is one of the most common morbidity seen among the population of developing countries. An increased incidence of tuberculosis is often noted among immunocompromised individuals. Mostly it occurs as pulmonary tuberculosis followed by secondary involvement of other organs. The overall incidence of cardiac tuberculosis is 1-2% [1]. Myocardium involvement is mostly seen in association with pericardial disease, isolated involvement of myocardium is rare since the lactic acid produced in them and the continuous pumping action by the muscles inhibits the tubercle bacilli to settle. Prevalence of isolated myocardial involvement is less than 2% of cardiac tuberculosis [1][2]. Apart from heart other organs which are rarely involved by tuberculosis are thyroid, pancreas and skeletal muscle. The mode of spread of tubercle bacilli to heart is either hematogenous, lymphatic or contiguous from surrounding organs or tissues [1][2].

Myocardial bridging was first recognized by Reyman at autopsy in 1737 and first described angiographically by Portmann and Iwig in 1960 [3][4], it is a congenital anomaly involving major coronaries where a segment of it de-routes from its epicardial course and takes an intramuscular path before returning back to the epicardium. The myocardial band that overlies the de-routed segment of the artery is labelled as myocardial bridge. Such cases are frequently asymptomatic, however may lead to adverse complications like angina, myocardial ischemia, acute coronary syndromes, left ventricular dysfunction, arrhythmias, and even sudden cardiac death [3][4]. Complications mainly occur due to the narrowing of the tunneled segment of the artery at systole thus hampering the normal hemodynamics which may lead to formation of a plaque proximal to the tunneling and compromising the blood supply further. Myocardial bridging most commonly involves the middle segment of the left anterior descending (LAD) artery [3][4].

2. Case history

The deceased individual is a 31 year old female, mentally retarded and visually impaired since childhood and also diabetic as per her clinical history. She presented at the casualty in a disoriented state with complaints of fever, dyspnea (grade-3), cough with scanty expectoration and pleuritic chest pain since 5-6 days. On further clinical examination, bilateral pedal edema was present along with mild pallor. On lung auscultation crackles were heard with broncho-vescicular breathing. As per her previous echocardiography report, she was a known case of dilated cardiomyopathy with decreased left ventricular ejection fraction & generalized left ventricular hypokinesia. Her chest radiography showed evidence of patchy consolidation. Since she expired within few hours of her admission no further clinical work-up was done, and therefore to know the exact cause of her death a complete autopsy was performed where organs were grossly examined and kept for histo-pathological examination.

At autopsy, the heart appeared mildly enlarged and weighed 450 grams, the pericardium appeared unremarkable. On dissection the left and right ventricular wall had evidence of multiple ill- defined, grey-white lesion measuring from 1 × 1 cm to 0.3 x 0.3 cm (Figure 1). LAD had a 90% block one cm away from the origin while other major coronaries were patent.

On examination of lung, bilateral lungs weighed 500 grams each and pleura was thickened focally. On cut section lung showed multiple grey white patchy consolidated areas suggestive of bronchopneumonia. (Figure 2)

Other prominent findings on gross examination of other organs were chronic passive congestion of liver, congested spleen and superficial scars on cortical surface of kidney. Rest all organs appeared unremarkable on gross examination.

On histo-pathological examination of the heart wall, myocardium showed extensive areas of caseating necrosis with langhans type giant cells and at places extending upto the epicardium (Figure 3). Ziehl-Neelsen stain was done to look for acid fast bacilli Within the heart wall, but it was negative. Other prominent findings seen in the heart were, block due to atherosclerotic plaque within the LAD artery,

vasculitis in the coronary vessels, subendocardial and myocardial fibrosis along with tunneling of LAD with fibrin thrombi through the myocardium suggestive of myocardial bridging. Verhoeff- Van Gieson (VVG) stain was done for the same to confirm the presence of epicardial coronary artery within the myocardium (Figure 4).

Lung on microscopic examination, had evidence of tuberculous bronchopneumonia with langhans type giant cell and epithelioid cell granulomas(Figure 5), for the same Ziehl-Neelsen stain was done which turned out to be positive, unlike the section from the heart wall. Other additional findings seen in lung were features of acute lung injury, which showed evidence of intra-alveolar fibrin.

The final cause of death after thorough evaluation of gross and histo-pathological findings was framed as "Cardiac tuberculosis and tuberculous bronchopneumonia with acute coronary insufficiency in a known case of dilated cardiomyopathy with chronic ischemic heart disease".

4. DISCUSSION

Cardiac tuberculosis is a rare finding with only 1-2% cases and mostly reported incidentally at autopsy [1][2]. Clinically, most cases being asymptomatic often goes under-diagnosed, they can present as anything like coronary vasculitis, ventricular arrhythmias or sudden cardiac death due to the secondary changes caused by tuberculosis within the heart wall[2]. Imaging studies of heart can be helpful but endomyocardial biopsy is gold standard for diagnosis. Three primary morphologic forms of cardiac tuberculosis seen are diffuse infiltrative form rich in lymphocytes and giant cells, nodular type with caseation and a miliary variant seen with hematogenous spread as described by Horn & Saphir [1][2]. Present case shows an evidence of an infiltrative type of morphology within the heart wall that is mostly limited upto the myocardium at places extending upto the epicardium, but sparing the pericardium. Like the present case, other case reports where a similar type of morphology was noted in the heart wall was done by Cowley A et al..,(2017) [5]. After studying the gross and histological findings in the present case a secondary involvement of heart wall from a lung primary is suggested.

Our case also had an incidental finding of congenital myocardial bridging, myocardial bridging has an incidence of 4.6% to as high as 80% at autopsy [6][7]. Coronary angiography, intravenous ultrasound and doppler CT (computed tomography) scan are the common modalities that can reveal the presence of a myocardial bridging antemortemly [4]. The proximal segment to the bridging vessel is more vulnerable to endothelial damage and develops an atherosclerotic plaque due to the hemodynamic changes that occurs within it, which further complicates with narrowing of lumen, as seen in the present case. Similar studies like the study by Thej et al..,(2012) also had myocardial bridging with atherosclerosis [8]. Similarly other findings like subendocardial and myocardial fibrosis as seen in the present case and the study by Morales et al..,(1993) can also occur in association with myocardial bridging [9].

Therefore to summarize the present case, being a diabetic she was immunocompromised which possibly predisposed her to tuberculosis in both lung and heart, likewise diabetes can also predispose to dilated cardiomyopathy. Similarly myocardial bridging, diabetes as well as chronic vasculitis, all have the potential to predispose to atherosclerosis followed by ischemic changes of myocardium, as suggested by the presence of myocardial and sub-endocardial fibrosis in the present case. Likewise secondary changes induced by tuberculosis infection in myocardium can lead to vasculitis or fibrosis which can be followed by arrhythmia or sudden cardiac death. Thus to conclude, we see in some or the other way all the pathologies and there complications in the present case are interlinked to each other.

Images


figure 1 - Left ventricular wall with multiple grey white lesions.

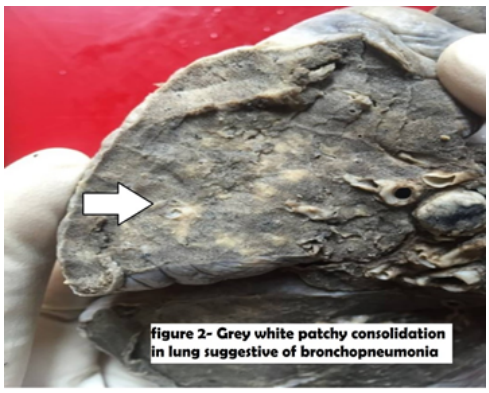
figure 2- Grey white patchy consolidation in lung suggestive of bronchopneumonia

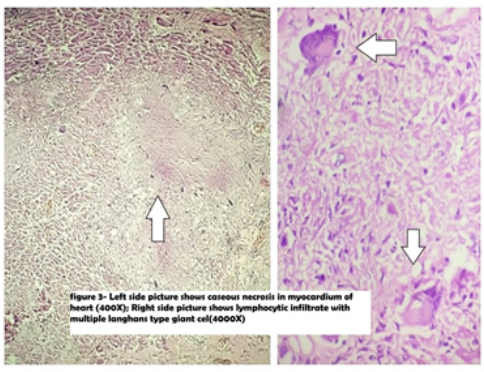
figure 3- Left side picture shows caseous necrosis in myocardium of heart (400X); Right side picture shows lymphocytic infiltrate with multiple langhans type giant cell(4000X)

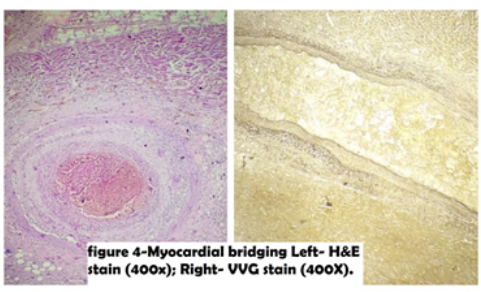
figure 4-Myocardial bridging Left- H&E stain (400x); Right- VVG stain (400X).

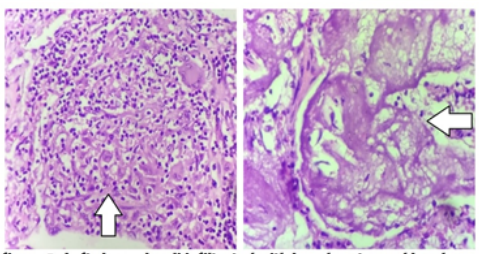
figure 5- Left- lung alveoli infiltrated with lymphocytes and langhans type giant cells (4000X); Right- Intra alveolar fibrin (4000X)

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