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PERICARDIAL CALCIFICATION ASSOCIATED WITH LEPROSY: A RARE COMBINATION

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Abstract
Majority of pericardial calcifications in the tropics are claimed to be of tuberculous origin. Other known causes include among other things uraemia, asbestosis, and post traumatic/post operative. We report a case of pericardial calcification not associated with constrictive pericarditis seen in a patient with established leprosy. Although a direct causal relationship cannot be established between leprosy and pericardial calcification, the well known condition of cardiac amyloid, a complication of leprosy might have contributed in the pathophysiology of the development of pericardial calcification in our patient. The inclusion of leprosy in the differential diagnosis of the aetiology of pericardial calcification in the tropics is suggested.

Key words: Pericardium, calcification, leprosy

Introduction

Pericardial calcification is associated with uraemia, asbestosis, post trauma/post operative and post pericarditis especially tuberculous or pyogenic. [1, 2] The presence of pericardial calcification occurs in about 50% of patients with constrictive pericarditis, [2] implying that not all calcified pericardium are constricted. Previous report from Nigeria[3] of patients with calcific constrictive pericarditis failed to show the cause of the pericardial disease even on histology. It has been suggested that the majority of cases of chronic pericarditis in this environment may be tuberculous even if histopathology fails to prove it[4]. Dystrophic calcification of peripheral nerves in leprosy is a well known phenomenon. [5] However, the association of pericardial calcification with leprosy has not been reported.

Case report

A 55-year-old Nigerian woman was admitted to the University of Ilorin Teaching Hospital on account of leg swelling and dyspnoea on exertion of 3 weeks and 4 days respectively. These were associated with unproductive cough, palpitation, orthopnoea and paroxysmal nocturnal dyspnoea. She was earlier seen 18months ago with hypopigmented, hypoanaesthetic maculo-papular skin lesions on both forearms and hands. Previous lepromin test and ZN stain of the skin smear specimen were in favour of lepromatous leprosy and since then she has been receiving treatment accordingly. Her past medical history revealed no evidence of previous trauma, tuberculosis or incapacitating acute pericardial disease. There was also no previous history of exposure to asbestos.

On examination she was found to be chronically ill-looking with bilateral pitting pedal oedema up to the knees. Clawing of both hands with thenar and hypothenar wasting, bilaterally thickened but non tender great auricular (palpated along the outer border of the sternomastoid muscle), median and ulnar nerves were noted. Her blood pressure was 130/80mmHg, pulse rate was 120 per minute and was irregularly irregular. The apex beat was at 6th left intercostal space anterior axillary line. There were few bilateral basal crepitations in the chest. The liver and spleen were enlarged measuring 5cm and 2cm below the costal margins respectively and were non tender. Examination of other systems revealed no abnormality.

Results of investigations performed were as follows: Urinalysis, urea, electrolytes, creatinine, serum calcium and fasting blood sugar were within

normal limits. ECG showed atrial fibrillation with rapid ventricular response and low voltage complexes, suggestive of biventricular failure. Frontal chest radiograph revealed left ventricular cardiac enlargement, fullness of the hilar vasculature/ early upper lobe blood diversion bilaterally and calcific curvilinear opacity along the left heart border. Patient could not be further investigated neither with 2D Echocardiography nor with lateral chest radiograph due to non functioning facilities throughout the time of managing her case.

Because there were no symptoms of constrictive pericarditis in this patient she was accordingly treated for the heart failure with low salt diet, frusemide, slow K, digoxin and bed rest. Two weeks later, she showed remarkable improvement and was discharged to be seen in the medical outpatient department.

Discussion

All organs have a finite number of ways of reacting to injury, and the pericardium is no exception. Calcifications of the visceral and parietal pericardium are believed to represent the end stage of a previous pericardial insult, such as tuberculosis, purulent pericarditis, haemopericardium, asbestosis, rheumatic fever or uraemia. [1, 2] Large calcifications of the pericardium may be present in the absence of the clinical syndrome of constrictive pericarditis. Depending on the series, only 28 – 50% of calcified pericardium are constricted. [2] It has been shown that echocardiography is the simplest and the most widely used technique for imaging the pericardium. [6] It is unfortunate that our patient could not be evaluated even with simple 2D Echocardiography, which under normal circumstances will show pericardium as a dense echo behind the posterior left ventricular wall, while the presence of calcification will show a rather brightly echo, often with distal acoustic shadowing. [7] It is also worthy of note that, our patient had no clinical features of constriction which if present could have been suggested by the findings of an elevated venous pressure that fail to fall with inspiration – Kussmaul's sign, absence of cardiac murmurs and the presence of a loud early-diastolic sound known as the pericardial knock sound. [8] On echocardiography absence of respiratory variation in vena caval diameter was found to be an indicator of constriction.[7]

The clinical signs and symptoms of pericardial disease are often diagnostic. However, other differential diagnosis, such as myocardial infarction, pleurisy and spontaneous pneumothorax must be considered in evaluating patients with such clinical features. In these conditions and in those directly affecting the pericardium, chest radiograph and certain CT/ Fluoroscopic techniques may play an important role. The normal pericardium cannot be distinguished from the heart on chest radiograph. Only in the rare instance when air occupies the pericardial sac can one appreciate this thin membrane or as demonstrated in our case when the pericardium is calcified. Lateral view, if available localise the calcification better to the pericardium. Fluoroscopy and CT are not only sensitive but are also able to locate calcification to the pericardium.

Leprosy has a wide geographical distribution, occurring in Africa, Asia, the Caribbean and Pacific Islands. The lesions seen radio graphically mostly affect the hands, feet and face, and are caused by infection by Mycobacterium leprae. [2, 9] While the association of nerve calcification with leprosy is well known, [5] the association of pericardial calcification with leprosy is unknown. Previous reports[10] however, have noted that secondary amyloidosis may arise as a complication of leprosy and that cardiac amyloidal is a cause of pericardial effusion, impaired myocardial contractility and conduction defect all leading to heart failure. Although a direct causal relationship cannot be established in the case presented above, it may explain the noted association of the clinico-radiograhic cardiovascular presentation (high cardiac output, pulmonary venous congestion and pericardial calcification) and leprosy. With the normal serum calcium levels in our patient, the noted pericardial calcification is presumed to be of dystrophic nature. Moreso, similar dystrophic calcification of nerves has already been reported in association with leprosy. [5]

Pericardial calcification is treated by pericardiectomy if associated with constrictive pericarditis. [4] This may explain why our patient responded very well and rapidly on conventional antifailure therapy without being subjected to the hazardous procedure of pericardiectomy which has an attendant reported mortality of 6 - 25%. [3, 11] In addition, the absence of features of constriction both clinically and radiographically further confirms the believe that not all calcified pericardium are constricted. [2, 5]

Since the exact mechanism of the changes in the calcification of the pericardium is still unknown[12] and the claim that tuberculosis, a chronic granulomatous disease is the leading cause of pericardial calcification in the tropics. [4]

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