Role of echocardiography in suspicion of cardiac sarcoidosis: A case of cardiac sarcodiosis

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Abstract

Sarcoidosis is a multi-organ granulomatous disease of unknown aetiology. Adverse outcome related with cardiac in-volvement, makes early diagnosis of cardiac sarcoidosis crucial. In a 60-year-old man presenting with epigastric pain, echocardiography showed thinned out basal inferior sptum and basal inferior wall. Further screening for sarcoidosis involvement showed multifocal cardiac involvement both on cardiac magnetic resonance (CMR) and 18-F-fluorothymidine -positron emission tomography-computed tomography (F18-FLT PET-CT). Because of the functional deterioration and clinical symptoms, steroid treatment was initiated and regular follow-up of cardiac abnormalities. This case shows the finding of cardiac sarcoidosis on echocardiogram in symptomatic untreated patient, and rises the awareness of possible severe cardiac damage, Combination of PET and CMR is appealing to better understand the evolution of cardiac sarcoidosis and may help in the management of such patients. The echocardiogram is often abnormal in manifest cardiac sarcodiosis (CS) disease, but is usually normal in clinically silent CS (1). Abnormalities are variable and usually nonspecific, although interventricular septal thinning, especially basal, can be a feature of CS (2). Less frequently, there may be an increase in myocardial wall thickness, simulating left ventricular (LV) hypertrophy or resembling hypertrophic cardiomyopathy (3). Other abnormalities include LV and/or RV diastolic and systolic dysfunction, isolated wall motion abnormalities, basal septal thinning, and aneurysms (4, 5). Regional wall motion abnormalities are usually seen in a non-coronary distribution.

Keywords: Echocardiography, Cardiac sarcodiosis, Left ventricular.

Introduction

Sarcoidosis may be a systemic granulomatous disease of unknown aetiology. The frequency of cardiac involvement (cardiac sarcoidosis (CS)) varies across countries, but it has been reported that this disease is a very important prognosis. Complete heart block is that the commonest, and ventricular tachycardia/ventricular fibrillation the second commonest arrhythmia during this disease, both of which are related to cardiac overtime.

Diagnosing CS is usually difficult due to the non-specific ECG and echocardiographic findings, and CS is usually misdiagnosed as dilated cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy or an idiopathic aneurysm, and thus, endomyocardial biopsy is vital, but features a low sensitivity.

MRI and PET F-fluorodeoxyglucose have recently been shown to be useful tools for the non-invasive diagnosis of CS also as therapeutic assessment tools, but are still unsatisfactory. Typically, corticosteroid therapy is used to treat CS to regulate inflammation, prevent fibrosis and protect against any deterioration of cardiac function, but the long-term outcome remains in discussion.

Case summary

60 years old male Patient , Known to have BA ,not smoker or diabetic or hypertensive presented to our hospital by epigastric pain and vomiting .Initial examination : BP 130/ 60 mmgh, PULSE 45 bpm Chest auscultation : normal vesicular breathing , Heart auscultaion: S1S2 ,no murmurs. ECG at admission revealed: complete Heart block (figure 1). Lab: within normal except elevated cardiac Tn. chest X ray: normal (figure 2).

Patient was admitted to our CCU as case of NSTEMI

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complicated by complete heart block.Temporary pace maker was inserted. Transthoracic Echocardiography showed impaired systolic function around 40%, thinning out of (basal inferior wall, basal septal wall suggesting of cardiac sarcoidosis (figure 3), Patient received full anti-ischemic medications. Patient was transferred to intervention Centre for revascularization Cornary angiography (13/6/2019) showed: 80% stenosis of mid LAD, PCI was done by Xience drug eluted stent 2.75x 12.

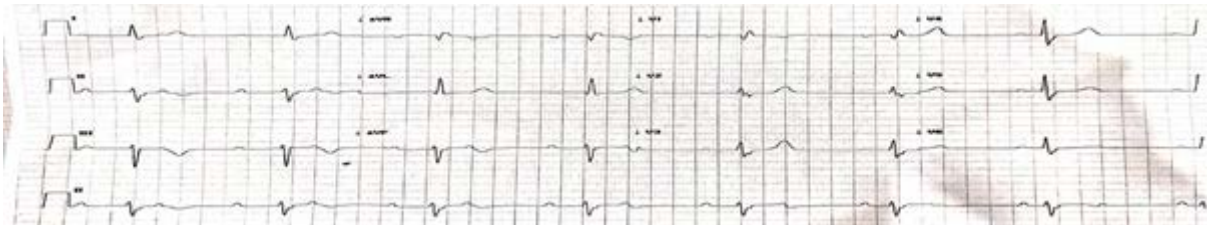

Figure 1: electrocardiogram on admission revealed complete heart block

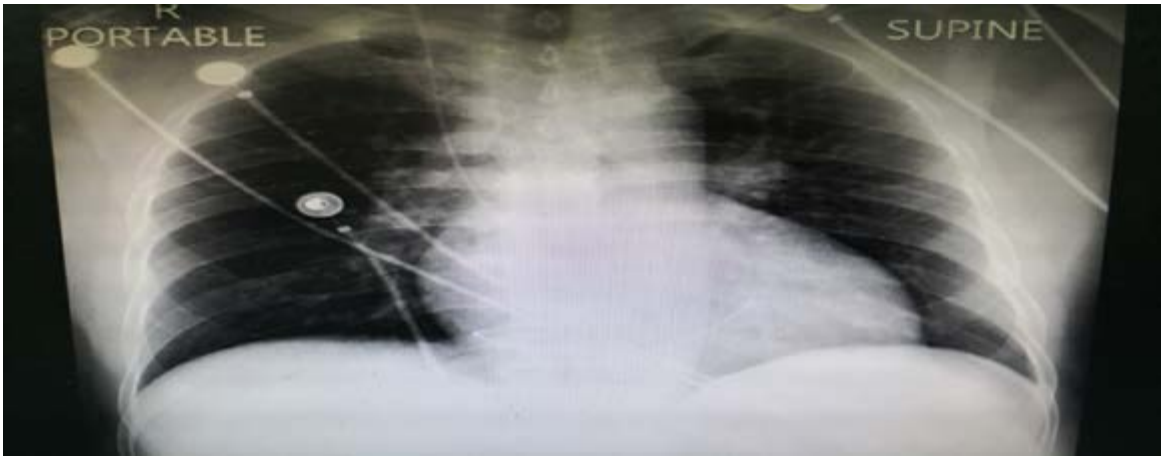

Figure 2: chest radiographon admission: normal xray Figure 2: chest radiographon admission: normal xray

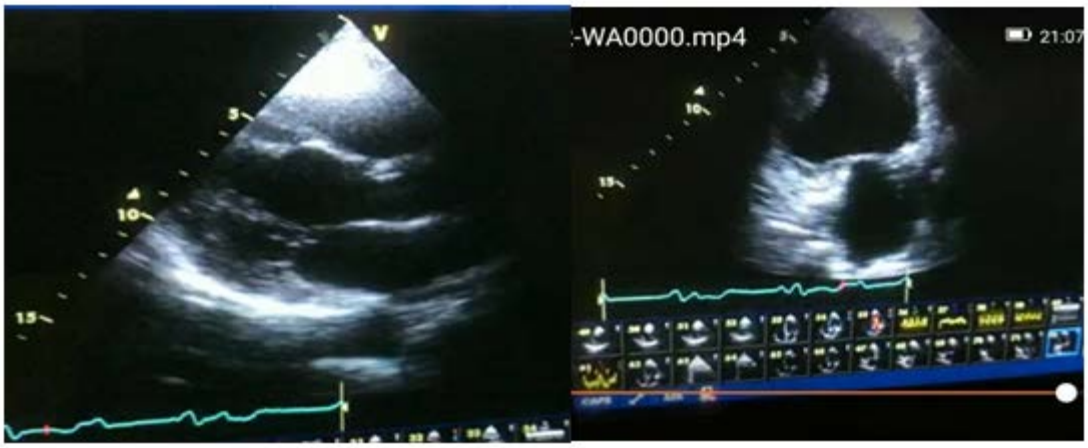

Figure 3: echcardiography showed thinning out basal anterior septum, and basal inferior wall

Temporary pacemaker was removed after PCI. 10 days later ,CMR was done that showed : Impaired systolic function EF 40% , RWMA (hyokinesia of mid , basal inferior septum , inferolateral wall , thinned out basal inferior and basal anterosptal segment), LGE showed intense transmural scar of basal anteroseptal , inferior segment , anterior wall showed focal sub-endocardial and mid wall fibrosis suggesting cardiac sarcoidosis.

One day later after CMR, Patient again developed asymptomatic Complete Heart Block ….CRT D was implanted Respiratory team reviewed the case and recommended for high resolution CT (showed scattered mediastinal lymphadenopathy without specific CT finding for sarcoidosis, 2 days later , PET scan was done showed : positive F-18FLT PET/CT scan involving basal inferior , basal anteroseptal segment , Rt paratracheal , Rt hilar lymph nodes suggesting cardiac sarcoidosis with lymph nodes involvement Patient was finally discharged from hospital on full anti-ischemic medications plus corticosteroids (prednisolone 60 mg /day), lymph node biopsy is planned

after 6 months as patient cannot stop dual antiplatelets.

Discussion

Cardiac sarcoidosis is a difficult diagnosis to make. As this case illustrates, the disease is a great mimicker of many common clinical entities. A high index of suspicion and early recognition of atypical features of common cardiac syndromes may offer first clues to the diagnosis.

Lack of risk factors (not smoker not diabetic or hypertensive), normal lipid profile. Thinning of the basal anterior septum as well as the tapered edges of the culprit stenosis on angiography rather than abrupt edges as with a ruptured atherosclerotic plaque, recurrence of complete heart block after PCI raise suspicion for a non-atherosclerotic cause of inflammation.

Once there is diagnostic suspicion, efforts should be made to confirm the diagnosis of cardiac sarcoidosis. This, too, can be very challenging because the gold standard, endomyocardial biopsy, performed ‘‘blindly’’ at the right ventricular septal wall, has a sensitivity of only 25 % [7]. The low diagnostic sensitivity is likely largely a result of focal involvement [8] with the resultant sampling error.

CMR is currently considered the gold standard as it is more sensitive for detection of cardiac involvement.[9] It provides high sensitivity by using T2 weighted imaging for detection of acute inflammation, T1 weighted imaging to assess wall motion abnormalities, hypertrophy, infiltrative processes, wall thinning and heart failure; and late gadolinium enhancement (LGE) for assessment of fibrosis or scar tissue. FDG-PET should also be considered when the diagnosis of cardiac sarcoidosis has not been firmly established.

Epicardial coronary artery involvement is rare functional impairment due to presumed coronary vasculitis was evidenced by abnormal wall motion or thinning of the affected portions of the left ventricular Myocardium [10].

Conclusion

This case further broadens the spectrum of presentations of cardiac sarcoidosis and reiterates the importance of considering this disorder in the differential diagnosis of cardiac pathology that involves both the myocardium and coronary arteries.

Acknowledgements

None

Conflict of Interest

None

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