Eosinophilic myocarditis: A rare case presentation

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SUMMARY

22-year-old gentleman presented to the emergency department with palpitation and pre-syncope. Initial investigation revealed leukocytosis with a predominant eosinophil count and an exceptionally high troponin level. His electrocardiogram (ECG) showed supraventricular tacycardia with aberrancy. The patient was given intravenous tenecteplase and subsequently underwent coronary angiogram. He was diagnosed with eosinophilic myocarditis after extensive investigation.

INTRODUCTION

Myocarditis is an inflammation of the heart muscle. It is believed that it is most often caused by viruses, bacteria, fungi, parasites or protozoa. They are also known causes of infectious myocarditis. There are other causes of myocarditis such as immune-mediated causes (e.g., systemic lupus erythematosus, Churg-Strauss syndrome, sarcoidosis or Wegener's granulomatosis), toxic causes (e.g., drugs, heavy metals, and snake or scorpion venom), and damage from ionising radiation or electricity.

Eosinophilic myocarditis (EM) is a rare subtype of myocarditis characterised by infiltration of eosinophil which subsequently causes either focal or diffuse myocardial inflammation. To date there are less than 30 published case reports of EM.[2]

CASE REPORT

A 22-year-old gentleman with a previous history of appendicectomy and multiple previous admission for typhoid fever and hypereosinophilic enterocolitis which was confirmed with trephine biopsy presented with vomiting for 2 days duration. There were two episodes of vomiting, and it was non-projectile in nature. The vomiting was associated with pre-syncope prior to presentation. He also experienced palpitation, profuse sweating and dizziness prior to the pre-syncopal episode. The patient denied other symptoms such as fever, shortness of breath, abdominal pain, loose stool and chest pain.

Upon arrival at the emergency department, he was alert, conscious, mildly dehydrated and septic looking. His blood pressure was on the lower side (108/62 mmHg). His heart rate was at 87 bpm and SpO_2 98% under room air. Respiratory and cardiovascular examination was normal.

Initial electrocardiogram (ECG) revealed ST-segment depression over leads I, II, III, aVF and V4-V6. His white cell count was 36.5($x10^9$/L) with eosinophil of 24.47(70%), haemoglobin 15.3 g/dL, while his platelet was 398($x10^9$/L). Troponin level was 3108ng/dL, while renal and coagulation profile was normal. He was empirically treated as typhoid carditis and was started on intravenous normal saline, pantoprazole 40 mg, ceftriaxone 3 g and metronidazole 500 mg.

Patient then developed supraventricular tachycardia with aberrancy which aborted with carotid massage and intravenous calcium gluconate 10% and intravenous magnesium sulphate 2 g.

He was admitted to the coronary care unit (CCU) for close monitoring. Bedside transthoracic echocardiogram showed normal heart with an estimated ejection fraction of 58% with normal valves. Day 2 of admission, computed tomography (CT) abdomen and CT pulmonary angiogram was done which turned out to be normal. He was treated as type II myocardial infarction in view of persistent widespread ST-segment depression and was given intravenous tenecteplase by the cardiology team as emergency coronary angiogram was not available at the time. ECG post thrombolysis showed resolution of ST depression over the anterolateral lead by more than 50%.

The patient also underwent coronary angiogram to find the culprit for the arrhythmias which turned out to be normal. He also had persistent high eosinophil throughout admission and was referred to haematology team for further investigation. Patient was counselled for endocardial biopsy to confirm the diagnosis of eosinophilic myocarditis however patient refused the procedure. He was started on tablet cetirizine 10 mg once daily (OD) and tablet prednisolone 20 mg OD by the haematology team and subsequently patient's eosinophil level reduced to a normal range. He was discharged on day 7 of his admission with oral cetirizine and prednisolone 20 mg once a day, and appointment for cardiac magnetic resonance imaging (MRI). MRI cardiac done later showed myocardial inflammation and fibrosis of basal/mid-apical inferoseptal left ventricle wall with extension to subepicardial layer of apicoseptal indicative of myocarditis.

DISCUSSION

Eosinophilic myocarditis (EM) is a rare form of myocardial inflammation with wide variety of aetiology. In developed

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country, the most common causes of EM are allergic reaction or hypersensitivity. Other possible cause includes infection, malignancies, vasculitis and drugs.

It is characterised pathologically by diffuse focal myocardial inflammation with abnormally high eosinophil infiltration. This infiltration plays an important role in the pathogenesis of eosinophilic myocarditis via release of eosinophilic granule proteins such as eosinophil cationic protein and major basic protein which subsequently causes dysfunction of myocyte mitochondria leading to myocardial lesions such as endocardial necrosis.

Three stages have been reported in EM. The first stage includes myocardial infiltration of eosinophil causing acute necrosis. This is followed by hyper-coagulation state leading to thrombus formation either within the coronary vasculature or the ventricles. Finally stage three which cause permanent cardiac dysfunction due to the formation of scar tissue.

Accurate diagnosis of EM is challenging. The diagnosis is made based on the medical history, clinical findings and laboratory investigation results. However, in some cases it is difficult to differentiate between myocarditis and acute myocardial infarction because of similar clinical presentation. Back to our patient, he was treated as type 2 myocardial infarction in view of the persistent ST-segment depression and changes in his cardiac enzyme level. The patient was given fibrinolytic therapy as coronary angiogram was not available at the time, before further investigation revealed the diagnosis of EM.

The presence of peripheral eosinophilia may indicate a diagnosis of EM. However, in some patients with a confirmed diagnosis of EM, they never develop peripheral eosinophilia throughout the course of the disease. Relying on peripheral eosinophilia to diagnose EM and treat the disease may be misleading. Inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate are often raised together with myocardial injury markers such as troponin T and creatine kinase. This patient was previously diagnosed with hypereosinophilic enterocolitis with histopathological sample taken from trephine biopsy which made the diagnosis of eosinophilic myocarditis likely.

ECG findings in EM may mimic of those with acute coronary syndrome which can cause the delay in arriving to the exact diagnosis of myocarditis. Common ECG findings include sinus tachycardia, ST-T segment abnormalities and conduction delay.[1] However ECG changes were not specific or sensitive for myocarditis. For example, our patient had widespread ST depression and few episodes of arrhythmia which delay an accurate diagnosis of myocarditis. Echocardiography is usually the most readily available imaging modality in most institutions. Common findings on 2D echocardiography include left ventricular dysfunction in up to 69% of cases as evidenced by segmental wall motion abnormalities. Reversible left ventricular hypertrophy can also be observed in 15% of cases while left ventricular cavity dilatation is usually minimal or absent. In addition, only 23% will have right ventricular involvement.[5] In comparison to our patient, the patient had no heart abnormalities in the echocardiography. Cardiovascular magnetic resonance (CMR) is the only non-invasive imaging modality that can assess for endomyocardial involvement and aid in the initial diagnosis of EM prior to endomyocardial biopsy (EMB).[9] Myocarditis is usually characterised by extensive myocardial hyperintensity on T-2 weighted imaging together with subendocardial delayed enhancement. The good diagnostic accuracy of MRI in myocarditis was highlighted in pooled controlled trials with a sensitivity and specificity of 67 and 91% respectively along with positive and negative predictive values of 91 and 69% respectively.[8]

Nuclear imaging has high sensitivity in detecting evidence of myocarditis. However, they are not frequently recommended for the diagnosis in view of its limited availability and risk of radiation exposure to patients and staff involved.

EMB remains the gold standard investigation for the diagnosis of eosinophilic myocarditis. EMB findings include diffuse myocardial necrosis associated with extensive eosinophilic infiltration of the myocardial interstitium, perivascular infiltration, focal myocyte dissolution and myocardial interstitial fibrosis. However, EMB has a low sensitivity (50%) as eosinophilic infiltration is often focal and this can give rise to sampling errors and false-negative results.[1] According to journal of the American College of Cardiology, EMB is associated with a risk of severe complication such as cardiac tamponade (0.5%), perforation (0.1%) and overall risk of complication is 6%.

Initial treatment goal of patient with EM is haemodynamic stability. Treatment of EM include identifying the underlying cause of EM such as stopping the offending drug and treating the parasitic infection as soon as possible. Standard cardiac failure medication and high dose corticosteroid remain therapy of choice. Corticosteroids therapy has been successfully documented in various case reports such as in three cases presented by Wong et al., all the patients demonstrated complete recovery and normalisation of cardiac contractility after treatment with high-dose oral steroids with gradual tapering.[10]

The usage of intravenous methylprednisolone bolus (1 g/day for 3 days) followed by 1 mg/kg/day oral prednisolone, with gradual tapering for one year demonstrated an improvement in symptoms, such as reduction of eosinophil count and increased ejection fraction as reported by a case report.[11] Our patient was started with tablet prednisolone 20 mg for two months and then continued with tapering dose of prednisolone 10 mg. Some patients with milder form of EM may not require corticosteroid therapy. There is no clear guideline regarding the use, dose and duration of corticosteroid therapy in EM.

CONCLUSION

Eosinophilic myocarditis (EM) remain rare type of myocarditis and possibly underdiagnosed. EM characterised by endocardial injury with elevation of eosinophil count. In setting of patient with persistent eosinophilia and presenting with non-specific cardiac symptoms and findings

(electrocardiogram (ECG), troponin elevation and echocardiogram), EM should be added to the differentials. CMR may assist in earlier diagnosis of EM in patient with peripheral eosinophilia. Treatment modality for EM remain to be finalise.

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DECLARATION

The authors declare no conflict of interest

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