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HYPEREOSINOPHILIC SYNDROME SIMULATING AN ACUTE CORONARY SYNDROME (ACS)

Medicine

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ABSTRACT

To report a case of Hypereosinophilic Syndrome (HES) which presented clinically as ACS. We present a case of 56 years old male patient with ACS with peripheral hypereosinophilia. The condition occurred after an event of ACS. Coronary angiography revealed Normal & secondary causes of hypereosinophilia were excluded.

KEYWORDS

INTRODUCTION:

HES is defined as peripheral blood eosinophil count above 1500 /µl associated with damage to end organ such as heart & lungs when no cause found for eosinophilia & no evidence of malignancy.

Cardiac involvement is major cause of morbidity & mortality and affects around 50% of patients of HES. Clinical manifestations include sign & symptoms of Heart Failure, intra-cardiac thrombus, myocardial ischemia, arrhythmia & less frequently pericarditis. The outcome is variable & depends upon progression of endomyocardial fibrosis with estimated mortality of 5 years of 30%.

CASE REPORT:

A 60 year old male patient, admitted to Civil hospital Ahmedabad with clinical presentation of Acute Coronary syndrome with criteria of Non ST segment elevation Myocardial infarction (NSTMI), atypical chest pain, ST depression & T wave inversion in V3,V4,V5 on ECG. S. Troponin I level 2.69 (N <0.03), coronary angiography showed normal, which did not confirm ACS, Moreover blood tests revealed eosinophilia with eosinophil count 15700/µl. The patient was discharged asymptomatic under antiplatelet therapy. Two months later patient returned to the emergency department with low grade fever with occasional chest pain since 2 days. The ECG revealed Right bundle branch block with ST depression in V3-V6 leads with S. Troponin level 0.08 (N <0.03).2D Echo was Normal, ANA Profile Negative. The eosinophil count raised up to 2530/µl. Bone marrow biopsy and flow cytometry were normal which excluded haematological malignancy (normal T cell population and no clonal cells). Cytogenetic analysis demonstrated a normal karyotype. The patient was further evaluated & search for secondary causes of HES showed no evidence of parasitic infection, HIV infection, or any other malignancy. Patient was chronic smoker since last 35 years & k/c/o Hypertension since 5 years. Patient was treated with oral steroids Tab. Prednisolone given in a dose of 1 mg/kg/d for 5 days then was tapered. Patient improved on steroids, Eosinophil counts start decreasing from the 2nd day of therapy. Patient was discharged after 2 weeks with oral steroids with no symptoms, normal trop-I level and normal ECG.

DISCUSSION

HES is divided into primary neoplastic HES, secondary HES and idiopathic HES.As there is no evidence of any secondary cause in this case, Idiopathic type of HES is kept by exclusion.

When eosinophilia presents with cardiac involvement, some causes of Eosinophilic myocarditis must be excluded in the differential diagnosis, such as allergic disorders, hypersensitivity to drugs, parasitic infections, infectious diseases (HIV infection), malignancy (acute or chronic eosinophil leukaemia, T cell lymphoma, and some solid tumours involving the lungs and colon) and Autoimmune disorder. Cardiac involvement has three different pathological stages: the necrotic stage, the thrombotic stage and the fibrotic stage. Some patients remain asymptomatic until the latter, when they develop a restrictive or dilated cardiomyopathy. Presentation of HES is not specific & in most cases, dyspnoea is the only symptom. Chest pain, cough and palpitations can also present. Cardiac involvement most frequently presents as heart failure, intracardiac thrombus, myocardial ischaemia, arrhythmias or pericarditis.

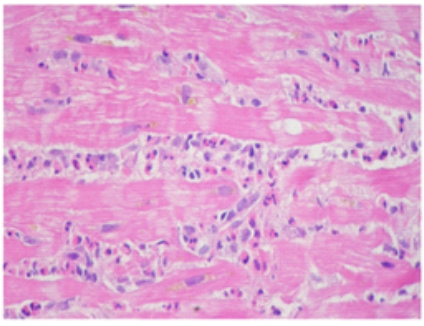

Figure 1 Endomyocardial biopsy suggestive of eosinophilic infiltration

There are some reported cases of eosinophilic myocarditis clinically presenting as ACS. The clinical manifestations mimicking an ACS may be related to coronary artery spasm, coronary aneurysms, occlusive coronary thrombi or coronary artery dissection. ECG frequently shows T-wave inversion and ST-T wave abnormalities, probably due to endomyocardial fibrosis and inflammation. The serum troponin level increase may indicate the initial phase or the necrotic stage, with normalization after initiation of high dose corticosteroids, which may suggest that troponin I might be a sensitive marker for cardiac damage and cardiac decompensation. The management of HES includes conventional therapy for heart failure and immunosuppressive therapy for the underlying eosinophilia. Several cases have shown good outcomes with prednisolone prescribed at a starting dose of 1 mg/kg/day (as used in conventional heart failure therapy), with improvement of symptoms, increased ejection fraction

and resolution of eosinophilia. Our patient had a good response with prednisolone 1 mg/kg/day. After prednisolone was tapered and then stopped, the patient was still asymptomatic at the 2 month follow up, had stable heart function under treatment and had a normal serum eosinophil count. Follow-up should be maintained in order to control the eosinophil level and reassess the need to restart immunosuppressive therapy. There is no evidence for the use of anticoagulation therapy for prophylaxis of thrombotic complications. Glucocorticoids remain the treatment of choice. Tyrosine kinase inhibitor Imatinib also can be used. Interferon alpha is a second line drug of choice. Hydroxyurea also has efficacy for steroid refractory cases. Valve replacement is indicated in patient with hypereosinophilia & regurgitating valve. Alternatively in refractory cases, Hematopoetic stem cell transplantation reverses the organ Dysfunction.

CONCLUSION

This case report describes a rare, potentially fatal disease; however, early initiation of corticosteroid therapy contributed to the favourable outcome. Since hypereosinophilia can sudden worsen the condition of patient, possible diagnosis of HES should not be delayed until other diagnosis have been excluded & should be as fast as ACS excluded.

REFERENCES:

(1) Monograph on Common Haematological Diseases in India by Association of Physicians of India
(2) Rezaizadeh H, Sanchez-Ross M, Kaluski E, Klapholz M, Haider B, Gerula C. Acute eosinophilic myocarditis: diagnosis and treatment. Acute Card Care 2010;12:31–36.
(3) Hypereosinophilia by Dr. Venkata Anuradha Samavedi, MEDSCAP
(4) Blood 2015 126:1069-1077;doi:https//doi.org/10.1182 by Amy D. Kilon
(5) Amini R, Nielsen C. Eosinophilic myocarditis mimicking acute coronary syndrome secondary to idiopathic hypereosinophilic syndrome: a case report. J Med Case Rep 2010;4:40.