Chronic Active Hepatitis-Rare Association with Secondary Eosinophilia

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Sir,

Hypereosinophilia is characterised by marked increase in eosinophil count. In most cases it is reactive and found in association with parasitosis, allergic manifestations, pulmonary infiltrates, malignancies, collagen vascular diseases.1 It may be primary due to clonal proliferation of eosinophils. Some infrequent rare secondary causes should also be kept in mind while evaluating case of hypereosinophilia, like chronic active hepatitis, acute pancreatitis, chronic dialysis, post radiation and hypopituitarism.2

A 55 year old male patient, residing at Ahmedabad, was admitted in L.G. General hospital with distension of abdomen over a period of 20 days. Patient was chronic alcoholic since last 30 years. No history of previous blood transfusion, major surgery or exposure to multiple sexual partners.

On examination, vitals were normal. Patient was pale. Respiratory, cardiovascular and nervous system examinations were normal. Per abdomen examination was suggestive of globular distension of abdomen with dilated veins on flanks. Liver and spleen were not palpable.

After admission routine blood investigations were sent.

Haemoglobin was 5.8 Gm%, Total count was 51,000 cells/cumm, Neutrophil count was 36%, Lymphocyte count was 10%, Monocyte count was 01%, Eosinophil count was 53%, Basophil count was 00%. Platelet count was 1.35 lacs. Peripheral smear was suggestive of moderate hypochromia, anisocytosis, macro-ovalocyte, and eosinophilia. Random blood sugar, serum urea and serum creatinine were within normal limits. S. bilirubin was 1.0 mg%, serum glutamic pyruvic transaminase was 60 IU/L, alkaline phosphatase was 256 IU/L, prothrombin time with INR was 2.1.

HIV was non-reactive, while HBsAg, IgGHBcAb and HBeAg were reactive.

USG Abdomen was showing shrunken liver with coarse echo texture suggestive of cirrhosis.

Absolute eosinophil count was 20,000/cumm. The significantly elevated eosinophil count is usually found in malignancy and primary clonal eosinophilia.

Bone marrow examination was suggestive of eosinophilic myeloid hyperplasia. Other myeloid cell series were normal in maturation and distribution. The bone marrow findings along with normal LDH level and negative ascitic fluid cytological screening rule out malignancy as a cause of hypereosinophilia.

S.IgE level was 2000 IU/ml. Although patient had no past history of allergic manifestations.

Three consecutive samples of stool for ova, cysts and parasites were sent, which were negative.

Serum rheumatoid arthritis factor was positive, but patient was not having any symptoms suggestive of rheumatoid arthritis and anti cyclic citrullinated peptide antibody was negative.

ANA was negative which ruled out connective tissue disease as a cause for hypereosinophilia.

So in our patient we ruled out primary and common secondary causes for eosinophilia, and found only chronic active hepatitis as to be associated with hypereosinophilia.

References