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Case Report

Eosinophilia: a case series with review on different clinical presentations of eosinophilia

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ABSTRACT

In this article, the term eosinophilia is defined as an increase in peripheral blood eosinophilic leukocytes to more than 500 cells per microlitre of blood. Emphasis is placed on the number of eosinophils circulating in the peripheral blood, although an increase in eosinophils can be observed in other body fluids and many body tissues. Most cases of secondary eosinophilia are treated on the basis of underlying causes. Parasitic and fungal infections can be worsened or disseminated by use of steroids and should be ruled out. In patients with primary eosinophilia without organ involvement, no treatment may be necessary. Peripheral eosinophilia does not necessarily correlate with organ involvement. Steroid responsiveness should be evaluated, both for prognosis (steroid responsive patients do better) and to guide treatment when needed. We herein present a case series of three patients of blood eosinophilia who presented to the Sudha Multispecialty Hospital and Critical Care Centre and Shri. Bhausaheb Hire Govt Medical College, Dhule during 2012- 2014. We reported three cases of eosinophilia. First patient was 22 yr. male presenting with chronic fever and cough. He was prescribed antituberculous treatment for chronic fever and cough not responding to antibiotics. PBS revealed eosinophils, serum IgE levels increased. Treated with DEC and improved. Second patient was presented with abdominal pain and severe leg pain and cramps. Ultrasound abdomen showed appendicitis. Her CBC revealed increased TLC. Surgeon has advised surgery for appendicitis. Careful examination of PBS showed eosinophilia. She was prescribed DEC (Diethylcarbamazine) for 3 weeks and her symptoms were relieved. Third patient presented with fever of unknown origin (FUO). PBS and bone marrow showed eosinophilia. Serum IgE levels increased. Treated with DEC and steroids and improved. Patients presenting with common complaints such as fever, cough, breathlessness, weakness and associated increased leukocyte count does not always have bacterial infection. Careful examination of peripheral blood smear may reveal eosinophilia. All above mentioned patients were diagnosed late because increased eosinophils were never thought as a cause of raised TLC. Awareness about peripheral eosinophilia may help in early diagnosis of such patients.

Keywords: Eosinophilia, CBC, DEC

INTRODUCTION

Eosinophilia is the presence of > 500 eosinophil's per microliter of blood and is common in many settings besides parasitic infections. Significant tissue eosinophilia can occur without an elevated blood count. A common cause of eosinophilia is allergic reaction to drugs (iodides, aspirin, sulfonamides, nitrofurantoin, penicillins and cephalosporins). Allergies such as hay fever asthma, eczema, serum sickness, allergic vacuities and pemphigus are associated with eosinophilia. Eosinophilia also occurs in collagen vascular diseases and malignancies. The degree of eosinophilia is rarely helpful for identifying the cause, except at extremes of eosinophil counts. The disorders that can cause eosinophilia are best distinguished by patient's history,

clinical presentation and specific laboratory testing. This article will discuss various clinical presentations of eosinophilia.

CASE REPORTS

Case report 1:

22 yr. old male from (Maharashtra) India presented with dry cough, fever on and off since one and half year. No history of expectoration or hemoptysis and breathlessness. Was treated with antihistamines, antibiotics, cough suppressants & was started on antituberculous drugs by general practitioner. Referred to us for persistence of symptoms & high grade fever, increased leukocyte count & toxic look with respiratory distress. Examination revealed high grade fever (Temp-1020F), toxic look, PR-90/min, BP - 120/80 min RR-35/min. Rest general and systemic examination were unremarkable. Blood culture was sent on admission. Antituberculous treatment was stopped. His blood investigations showed Hb- 15.5gm%, TLC- 49,800 DLC-P-12: L-13; M- 03; E-72; B-0 with absolute eosinophil count 35, 856 /mm3. ESR - 10mm at the end of one Hr., malarial parasite- negative. Liver & renal parameters were normal. Chest X Ray- revealed bilateral lung fields showing reticulonodular pattern. Ultrasound abdomen & pelvis revealed bilateral minimal pleural effusion. Peripheral blood smear for microfilaria was negative. Filarial antibody IgG positive, serum IgE level were raised 327 (Normal in adults- upto 100 IU/mil).He was started on steroids & diethyl carbamazine (DEC) after CBC & PBS report & observed in hospital for next 72 hrs. Fever & cough subsided gradually. Patient was discharged & advised regular follow up in OPD. Repeat CBC revealed decrease in TLC & eosinophils. After 10 day TLC was 11,300 with 2% eosinophils, patient was afebrile & had no cough. Steroids tapered gradually & diethyli carbamazine was continued for 3 weeks. Patient has regular follow up in OPD for next three months.

Case report 2:

40 yrs. old female, R/O village from Maharashtra with complaints of intermittent abdominal pain, leg cramps, loss of appetite and weakness since 3-4 months. No h/o fever, loose motions or burning micturition. Abdominal pain was dull aching & occasionally associated with vomiting. Patient gives history of tuberculosis 10 yrs. back. Lab investigations showed Hb-10.4 gm. /dl, WBC 15,400 with P-88, L-10, E-01, M-01, B-00 and Platelets-4.3lacs. Patient was prescribed antibiotics & analgesics. Had partial symptomatic relief. After two months patient consulted to chest physician for similar complaints and investigated. CBC revealed Hb-10.9 gm. /dl, WBC count 18,500 with P-60, L-28, E-06, M-06, B-0 and platelets-5.6lacs. Ultrasound abdomen showed tubular, anechoic, non-compressible, aperistaltic tender structure measuring 5.6mms in right iliac fossa suggestive of an inflammed appendix. Chest X-ray & rest metabolic lab was normal. Surgical opinion in favour of laproscopic appendectomy. Patient was not willing for surgery. Patient took treatment & pulled on for next 15 days. Repeat CBC revealed Hb-9.6 gm. /dl, WBC-15,700 with P-38, L-59, E-02, M-01, B-00 and platelets-4.1 lacs. Patient referred to us for persistence of symptoms & increased WBC count. History confirmed and detail clinical examination done. On examination patient was afebrile, PR – 88/min, BP-110/70 mmHg weight 38 kg, thin built, rest general examination was normal, chest clear, CVS- Heart sounds normal, no murmur. P/A - soft, no distension, tenderness in right iliac fossa. The CBC showed WBC count of 15,200. Case was discussed with pathologist on phone & was told to look for eosinophilia on PBS. Before investigations surgery opinion was taken & surgeon has advised laproscopic surgery. The differential count showed 46% eosinophils. Absolute eosinophil count was 6992/mm3 Renal & liver function tests were normal. Patient was advised hospitalization for further investigations. But she was not willing so treated on OPD basic with Diethylcarbamazine for 3 weeks & was instructed to review if symptoms worsen. After 3 weeks patient came for follow up. She was symptomatically better & WBC count was 7300 with 11% eosinophils. Her abdominal pain & leg cramps were relieved and appetite was improved. After that patient had follow up with chest physician for three months & was symptom free.

Case report 3:

54 yr. old male, farmer resident of Dhule, Maharashtra and K/C/O hypertension on regular treatment with Tab Olmesartan 40 mg once daily came with complaints of mild fever, body-ache, cough and mild breathlessness since 8- 10 days. Clinical examination was unremarkable except bilateral ronchi in chest. CBC showed Hb-13.2 gm. /dl, TLC- 16,800 DLC (N-81, L-16, M-02, E-01, B-00), platelets 3.13 lacs, Sr Creatinine-2.3. Rest laboratory parameters were normal. Ultrasound abdomen and pelvis was normal. Tab Olmesartan was stopped because of raised creatinine and IV antibiotics were given on OPD basis. After 7 days CBC was repeated. It showed TLC-21,000, DLC (N-87, L-10, M-02, E-01, B-00), Sr. creatinine 1.9mg/dl, Sr. Uric acid-9.5 and chest X-ray S/O bronchitis. Patient had mild to moderate fever. So he was admitted. Broad spectrum antibiotics (piperacillin + Tazobactum) and empirical antimalarial were given for 5 days. Culture reports revealed no growth. CBC after 3 days revealed TLC- 12,300, DLC (N-87, L-10, M-01, E-02, B-00), platelets-2.86 lacs, Sr Creatinine-1.9, TSH was normal. Patient was symptomatically better so discharged. After 4 months patient came with complaints of fever, weakness, body-ache, and cough with scanty expectoration since 5 – 7 days. CBC revealed TLC-17,600, DLC (N-86, L-13, M-01, E-01, B-00), Sr. Creatinine-1.5. CT thorax showed enlarged right lobe of thyroid with two fluid attenuating non enhancing lesions with larger lesion showing peripheral calcification and measures 4.7 x 3.0cm compressing the trachea shifting it

to the left suggestive of benign nodules? Colloid nodules. Bilateral mild to moderate pleural effusion. Few mildly enlarged right paratracheal and carinal lymph nodes is seen largest node measuring 10mm, no focal lung parenchymal abnormality detected. 2D echo showed thin rim of pericardial effusion. ESR- 60mm at the end of one hour. TSH-1.10uIU/ML (normal range-0.25 to 6.0 uIU/ML). Repeat CBC revealed TLC-25,800 DLC (N-86, L-12, M-01, E-01, B-00). So patient referred to Nashik for bone marrow examination. Bone marrow aspiration showed normocellular bone marrow with increased mature eosinophils consistent with Hypereosinophilic syndrome. Retrospective PBS examination showed predominant eosinophils with Absolute Eosinophil Count (AEC) of 13,000. Test for detection of translocation involving the FIP1L1/PGDFRA by FISH (Fluorescence in situ Hybridization) technique to rule out eosinophilic leukemia was done. It was negative, no evidence of deletion or translocation seen. Serum IgE levels were 2,077.30IU/ml (normal adult's upto 100IU/ml). Anti-nuclear antibody (ANA) was negative. Patient was prescribed Tab. Wysolone 1mg/kg for 28 days with tapering doses and Tab. Hetrazan (DEC) 6mg/kg for 3 weeks. Repeat CBC after 10 days showed TLC- 18600 DLC (P-70, L-19, E-10, M-01, B-00) with AEC-1860/cumm. Patient was symptomatically much better. Sr. creat normalized to 1.2mg/dl. After 30 days CBC was normal with TLC- 9880 and AEC 467/cumm. Patient has regular follow up in OPD for last 2 yrs. for hypertension. CBC and Sr. creatinine done every 6 monthly showing normal reports.

DISCUSSION

Eosinphilic bronchitis (EB) may present as isolated chronic cough. EB without asthma was described by Gibson et al in 1989.[1] Cough variant asthma (CVA) is characterized by eosinophilia in sputum,[2,3] bronchoalveolar lavage (BAL) and bronchial biopsy specimens.[4] Upto 50% of patients with CVA have EB and degree of eosinophilia is similar to asthma. CVA progresses to typical asthma in 17-37% of cases. Eosinophilia may present with episodic respiratory symptoms of cough, wheeze, chest tightness, dyspnoea and sputum production but whose lung function measurements do not fulfill the criteria for asthma are often left without a diagnosis and effective treatment. EB is a key feature of asthma where it forms part of the current definition and is believed to be responsible for airway hyper responsiveness and asthma symptoms. EB is not a universal feature of asthma, however. The prevalence of EB in asthma ranges from 66% to 100%. EB may be absent during exacerbations of asthma and also in stable disease.[5,6]

Patients with seasonal allergic rhinitis and atopic subjects without asthma may have EB demonstrated either by sputum analysis or in bronchial biopsy samples. Approximately 50% of subjects with allergic rhinitis have EB; the level of EB can be similar to that seen in asthma and correlates with the degree of airway responsiveness. Some patients with COPD have eosinophilic inflammation detected in sputum, bronchial washings, BAL fluid and bronchial biopsy specimens. Eosinophilic inflammation in COPD is associated with the degree of airflow obstruction and mortality.[7] The group of primary (idiopathic) eosinophilic pneumonias consists of diseases of varying severity. Loefflers syndrome was originally reported as a benign, acute eosinophilic pneumonia of unknown cause characterized by migrating pulmonary infiltrates and minimal clinical manifestations. In some patients these clinical characteristics prove to be secondary to parasites or drugs. Acute eosinophilic pneumonia is an idiopathic acute febrile illness of, < 7 days duration with severe hypoxemia, pulmonary infiltrates, pleural effusions and no history of asthma. BAL fluid reveals greater than 25% eosinophils (normally less than 2% in nonsmokers) however; the peripheral eosinophilia tends to develop later in the course and may not be apparent on initial presentation. Chronic eosinophilic pneumonia presents with significant systemic symptoms including fever, chills, night sweats cough, anorexia and weight loss of several weeks to months duration. The chest x-ray clinically shows peripheral infiltrates, and pulmonary function tests reveals obstruction. Peripheral blood and BAL eosinophilia is more pronounced than in acute form. For both acute and chronic disease, dramatic clearing of symptoms and chest X ray is often noted after initiation of glucorticoid therapy. In contrast to acute eosinophilic pneumonia, chronic eosinophilic pneumonia tends to recur and may require repeated treatment.

The hypereosinophilic syndrome is characterized by presence of >1500 eosinophils per microliter of peripheral blood for 6 months or longer ; lack of evidence for parasitic, allergic or other known causes of eosinophilia; and signs or symptoms of multisystem organ dysfunction. Consistent features are blood and bone marrow eosinophilia with tissue infiltration by relatively mature eosinophils. The heart may be involved with tricuspid valve abnormalities or endomyocardial fibrosis and a restrictive biventricular cardiomyopathy. Other organs affected typically include the lungs, liver, spleen, skin and nervous system. Therapy of disorder consists of glucocorticoids and /or hydroxyurea, plus therapy as needed for cardiac dysfunction, which is frequently responsible for much of the mortality and morbidity in this syndrome. Pulmonary eosinophilia has also been associated with T cell lymphoma and has been reported following lung and bone marrow transplantation.

Eosinophilic esophagitis (EoE) is increasingly recognized in adults and children around the world. EoE often presents with dysphagia, food impaction, regurgitation or vomiting and decreased appetite. In addition, young children with EoE may present with feeding difficulties and poor weight gain. It is more common in men.[8] EoE is diagnosed based on typical esophageal symptoms and esophageal mucosal biopsies demonstrating esophageal

squamous epithelial infiltration with eosinophils. An atopic history of food allergy, asthma, eczema, or allergic rhinitisis present in majority of patients. Treatments for EoE include dietary restrictions, proton pump inhibitors, systemic or topical glucocorticoids, montelukast, immuno-modulators and endoscopic dilatation of strictures. Topical glucocorticoids are the most commonly used treatment in adults, but dietary restrictions has proven effective primarily in pediatric studies.

Eosinophilic gastritis, an autoimmune disorder first described by Kaisjer in 1937.[9] This inflammatory condition involves the infiltration of various layers of the stomach wall by eosinophils. There is no known cause but many therapies, including allergic reaction and parasitic infestation have been proposed. The disorder affects both genders equally, with a typical age at presentation of 35 to 45 years. Clinical manifestations typically include early satiety, epigastric pain, diarrhea, nausea and vomiting but can culminate in more serious symptoms such as gastrointestinal bleeding, weight loss and anaemia. Preferential involvement of different layers of gastric wall can produce specific clinical effects. When the predominant involvement is mucosal the presentation tends to be pain and bleeding; when the submucosa or muscularis is affected, patients usually present with obstruction. If the eosinophilic inflammation extends from stomach to the duodenum, patients may exhibit malabsorption, resulting in hypoproteinemia and or ascites.[10] Although approximately 80% - 90% of patients have some degree of peripheral eosinophilia, definitive diagnosis of eosinophilic gastritis rests on obtaining histopathologic specimens via endoscopic biopsy.[11] Because eosinophilic gastritis does not respond to therapies designed to counteract acid secretion or the presence of *H. Pylori*, endoscopic investigation with antral biopsy should be considered in patients whose dyspepsia is relucalcitrant to typical therapy. If eosinophilic gastritis is diagnosed, treatment with a short course of corticosteroids is indicated.

Eosinophilia-myalgia syndrome is a multisystem disease, with prominent cutaneous, hematologic and visceral manifestations, that frequently evolves into a chronic course and can occasionally be fatal. The syndrome is characterized by eosinophilia (eosinophil count > 1000 per microliter) and generalized disabling myalgia without other recognized causes. The disease is caused by ingesting contaminants in L-tryptophan containing products.[12] Contaminated L-tryptophan may not be the only cause of EMS. According to one estimate 14% of EMS cases were not related to L- tryptophan. Non L-tryptophan related cases were more likely to be associated with peripheral edema, rash, scleroderma like skin changes, alopecia, neuropathy and lower mean eosinophil count, fewer pulmonary symptoms and a better prognosis than L- tryptophan cases. Treatment is withdrawal of products containing L-tryptophan and the administration of glucocorticoids.

Very rarely eosinophilia may present as chronic eosinophilic leukemia. The term eosinophilic leukemia can be used to describe any haematological neoplasm in which a raised eosinophil count is the dominant abnormality; eosinophils are increased in blood and bone marrow, and the eosinophils are a part of the neoplastic clone. An eosiophil count greater than 1.5x 10.9/l is often used as one of the criteria for making this diagnosis. Chronic eosinophilic leukemia is a rare disease and its natural course can vary considerably between individuals. The disease may remain stable for years, even decades, or it may quickly progress and transforms to acute leukemia. Treatment may include corticosteroids, chemotherapy drugs such as hydroxyurea, and interferon therapy. Some patients may respond to imatinib mesylate. A stem cell transplant is newer option of therapy.

Eosinophilic fasciitis is a rare idiopathic disorder associated with induration of the skin that generally develops rapidly. Adults are primarily affected. The skin has a coarse cobblestone ‘’peau d orange’’ appearance. Skin involvement spares the fingers. Full thickness excisional biopsy of the lesion reveals fibrosis of the subcutaneous fascia, and is generally required for the diagnosis. Inflammation and eosinophil infiltration in the fascia are variably present. In acute phase of the illness, peripheral blood eosinophilia may be prominent. MRI appears to be a sensitive tool for the diagnosis of eosinophilic fasciitis. Treatment with glucocorticoids leads to prompt resolution of the eosinophilia. In contrast skin changes generally show slow and variable improvement. The prognosis of patients with eosinophilic fasciitis is good.

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