Case Report

Eosinophilic Gastritis – A case report

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Background: Eosinophilic gastritis is a rare chronic inflammatory disorder characterized by an eosinophilic infiltration of various layers from mucosa to serosa in the stomach wall, with peripheral eosinophilia, in the absence of any known cause for eosinophilia. It presents with varying symptoms depending on the layer of the stomach wall involved. Mucosal involvement presents with chronic dyspeptic symptoms whereas muscular involvement can presents with obstructive symptoms masquerading as gastric neoplasms. We describe a patient with isolated eosinophilic gastritis who presented with dyspeptic symptoms. Although it has been reported elsewhere, this is the first such case to be reported from a Sri Lanka. Case presentation: A 20 year old male presented with a history of epigastric burn and regurgitation for three weeks which was not related to meals without any other gastrointestinal symptoms. He poorly responded to different acid suppression medications. The examination was unremarkable except for mild epigastric tenderness. His full blood count revealed an eosinophilia (White blood cells-14 000/mm\textsuperscript{3} with 53\% of Eosinophils) with normal haemoglobin and platelets. The upper gastrointestinal endoscopy revealed areas of erythema and oedema in the stomach and was otherwise unremarkable up to the second part of duodenum. Gastric biopsy showed scattered areas of inflammatory infiltrate in the lamina propria with an eosinophil predominance; having 24 eosinophils per high power field. He was treated with prednisolon along with montelukast and he had a marked symptomatic improvement within a month. Conclusion: The aim of reporting this case is to make the physicians aware of this disease entity which should be included in the differential diagnosis when patients present with dyspeptic symptoms specially if there is no response to acid suppression. This is also to emphasize the necessity of research work to explain the aetiology and pathophysiology of eosinophilic gastritis as infective aetiological reason is possible in the tropics in particular.

Keywords: Eosinophilic gastritis, Dyspepsia, Eosinophilia

Background

Eosinophilic gastritis (EG) is a rare inflammatory disorder characterized by an eosinophilic infiltration in stomach with peripheral eosinophilia; in the absence of any known cause for eosinophilia. This was first described by Kaijser in 1937 and about 300 cases have been reported since then all over the world (Copeland et al., 2004). An extensive literature survey did not show any reported case of eosinophilic gastritis in Sri Lankan adult population, thus we report the first case.

Eosinophilic gastritis, enteritis and gastroenteritis are disorders with selective eosinophilic infiltration of the stomach, small intestine or both (Nguyen et al., 2012). They have two subtypes;

1. Primary / Idiopathic / allergic subtype
   - Atopic form
   - Non atopic form
   - Familial form
2. Secondary subtype
   - Hypereosinophilic disorders
   - Noneosinophilic disorders

List of abbreviations

EG, Eosinophilic gastritis; IL, Interleukin; GM CSF, Granulocyte macrophage colony stimulating factor; GI, Gastro-intestinal; HPF, High power field; UGIE, Upper gastro-intestinal endoscopy; CT, Computed tomography; WBC, White blood cells; E, Eosinophils
Eg: Celiac disease, inflammatory bowel disease, Vasculitis
EG can affect any age group but usually present in the third to fifth decades of life and it has a slight male preponderance (Nguyen et al., 2012). No clear association has been found between the eosinophilic gastritis and other idiopathic eosinophilic disorders like eosinophilic oesophagitis and colitis (Famularo et al., 2011).

Different hypotheses have been postulated to describe the pathogenesis of EG but convincing evidence for a definite aetiology is scarce. Underlying food & drug allergy or atopy have been noted in about 50% patients and this is even more common in children (Nguyen et al., 2012). Some studies say that altered mucosal integrity and impaired function of epithelial barrier are believed to facilitate the translocation of luminal bacteria and food products and localization of these substances across the gastro intestinal wall or in the peritoneum is thought to elicit an allergy like inflammatory response with eosinophilic infiltration (Famularo et al., 2011). Some researchers say that there is a partial IgE mediated food allergy response as well as a cell mediated response in EG (Lamabadusuriya, 2004). Eosinophil active cytokines like Interleukin (IL) 3, IL 5, Granulocyte macrophage colony stimulating factor (GM-CSF) and chemokines like eotaxin are also said to have a pivotal role in the pathogenesis (Nguyen et al., 2012). However a proper explanation for the aetiology of this condition is yet to be elucidated.

The disease presentation, course and the outcome depends on the layer of the stomach involved. It can be classified into 3 main subtypes (Lim et al., 2011).

**Predominant mucosal involvement**

This is the commonest form occurring in about 60%. Their presentation is with vomiting, dyspepsia, abdominal pain, diarrhea, malabsorption, failure to thrive or weight loss.

**Predominant muscle involvement**

This results in thickening of stomach wall and cause obstructive symptoms and present with cramping abdominal pain with nausea and vomiting. This may sometimes mimic pyloric stenosis or gastric carcinoma. Its occurrence is in about 30%

**Predominant serosal involvement**

This presents as eosinophilic ascites as a result of leakage of fluids due to serosal and visceral peritoneal inflammation. It is the least common type involving about 10%.

Diagnosis of eosinophilic gastroenteritis is characterized by the following features (Lim et al., 2011).
1. Presence of abnormal gastrointestinal(GI) symptoms
2. Biopsy evidence of eosinophilic infiltration in one or more areas of gastro intestinal tract having >20 eosinophils per high power field(HPF)
3. Absence of an identified cause for eosinophilia
4. Exclusion of eosinophilic involvement in organs other than GI tract

UGIE appearance may vary from normal mucosa to mild erythema, nodularity, thick mucosal folds and ulcers. Histopathologic features play a major role in the diagnosis (Lim et al., 2011). This usually shows increased eosinophils in the lamina propria (often more than 50/HPF) and also in the muscularis and serosal layers. Mast cell infiltrates and hyperplastic mesenteric lymph nodes infiltrated with eosinophils may also be present. Sometimes this is seen in macroscopically normal sites too. Due to their patchy distribution there is a high chance for these lesions to be missed, so multiple biopsies should be taken (Famularo et al., 2011).

There are no pathognomonic radiological findings in EG. Ultrasound scans, CT abdomen provide additional aids for diagnosis especially in the case of muscular or serosal involvement. They show narrowing of the antrum with gastric retention, lymphadenopathy or ascites if present.

Steroids play a major role in the treatment of EG. About 90% show a response to this; especially for the ones with obstructive symptoms and eosinophilic ascites (Lim et al., 2011). Mast cell stabilizers and Leukotriene receptor antagonists have also been proven to be effective (Lim et al., 2011). Remission without treatment is said to be possible in some patients (Famularo et al., 2011). Even though the natural course of the disease is unclear, some literature says that it may progress to eosinophilic leukaemia rarely (Famularo et al., 2011). Fatal outcomes are uncommon with this disease.

**Case presentation**

A 20 year old male presented with a history of epigastric burn and regurgitation for three weeks. It was not related to meals and was not subsided with acid suppression medications. He did not have loss of appetite, loss of weight, haematemesis or melena. There were no episodes of vomiting with radiating back pain and the bowel habits were normal. He was a teetotaler and a non smoker. He denied a history or a family history of allergy or atopy.

On examination, he was afebrile, not pale or icteric and his vital signs were normal with a heart rate of 88bpm, blood pressure of...
110/70mmHg and a respiratory rate of 14/min. His abdominal examination was unremarkable except for mild epigastric tenderness and the cardiovascular, respiratory and nervous system examination was normal.

During investigation, his full blood count showed an elevated white cell count (WBC- 14000/mm$^3$) with 53% of eosinophils(E). The absolute eosinophil count was 7420/mm$^3$. The Erythrocyte sedimentation rate was 04mm/1st hour. His stool full report was negative for parasitic ova and cysts and the filarial antigen testing too was negative. Blood picture showed an eosinophilia with normal morphology while the other cell lines were normal. His serum amylase levels, X-ray abdomen were normal. The ultrasound scan of the abdomen didn’t show organomegaly, lymphadenopathy, abdominal or pelvic masses or free fluid. His ECGs, renal and liver functions were normal and the chest X ray didn’t show any abnormality.

An upper gastro intestinal endoscopy (UGIE) was performed and areas of erythema and oedema were noted in the stomach while the oesophagus and the duodenum were normal. Gastric biopsy showed scattered areas of inflammatory infiltrate in the lamina propria with an eosinophil predominance; having 24 eosinophils per high power field. His oesophageal and the duodenal biopsies were normal with no evidence of eosinophilia. Thus the diagnosis of eosinophilic gastritis was made.

He was started on oral prednisolone 10mg 8hourly along with montelukast 10mg nocte. Within a period of one month he had a remarkable symptomatic improvement and the full blood count showed a reduction in the absolute eosinophil count. (WBC- $9.92 \times 10^9$ E-32% absolute eosinophil count was 3174/mm$^3$). Repeat Upper GI endoscopy done in one month revealed a dramatic improvement of the gastritis macroscopically. Having planned to continue steroids and leukotriene receptor antagonists under careful monitoring; patient lost to follow up.

**CONCLUSION**

Index case had all features of eosinophilic gastritis with predominant mucosal involvement. He showed a dramatic improvement to the steroids and leukotriene receptor antagonists. Our aim of reporting this case is to make the physicians aware of this disease entity which can easily be overlooked. Thus EG should be considered as a differential diagnosis when patients present with dyspeptic symptoms especially with poor response to acid suppression. Requesting a full blood count for the patients with dyspepsia would be beneficial in detecting this condition. This case further emphasize the necessity of research work to enlighten the aetiopathogenesis of isolated eosinophilic gastritis as it remains elusive.

**REFERENCES**


