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PEDIATRIC ONCALL
CHILD HEALTH CARE

TEACHING FILES (GRAND ROUNDS)

RECURRENT ABDOMINAL PAIN - DIAGNOSTIC DILEMMA

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Clinical Problem:

A 6-year-old boy presented with recurrent abdominal pain for 1 ½ years. He had history of soiling of his pants on passing stools on consumption of certain foods such as milk, cookies and chocolates. There was no history of diarrhea, constipation or eczema. No family member had history of allergies. He had history of pica as an infant. On examination, weight was 18.3 kg (between the 25th and 50th centile as per Indian Academy of Pediatrics growth charts). General and systemic examination were normal. Investigations showed hemoglobin 11.9 gm/dl, white cell count of 8100 cells/cumm (36% polymorphs, 50% lymphocytes, 12% eosinophils) as well as serum IgE of 189 IU/ml (Normal <90 IU/ml). Stool examination was normal but was acidic in pH. Antiendomysial antibodies (IgA, IgG) were negative and anti tissue translutaminase (TTG) IgA was also negative. Upper gastrointestinal endoscopy showed erosive gastritis with a positive rapid urease test for H. pylori from antral biopsy. On histopathology, there was presence of erosive gastritis in the stomach without H.pylori like organisms and features of chronic duodenitis with a normal villous to crypt ratio. There were no raised eosinophil counts on the biopsy samples. The patient was advised to avoid milk in the diet and advised regular follow up. He was also treated with triple therapy for H.pylori in form of proton pump inhibitor for 28 days + Clarithromycin for 10 days + metronidazole for 10 days but there was no improvement. His cow's milk protein allergy (CMPA) IgE was sent and was positive.

Is this eosinophilic gastritis or H.pylori gastritis or Cow milk protein allergy?

Discussion:

Primary eosinophilic gastrointestinal diseases (EGIDs) are a diverse group of conditions that affect the various parts of the digestive tract and are characterised by eosinophilic inflammation in the absence of eosinophilic causes. While there are currently no universally accepted guidelines for diagnosing EGIDs, a definitive diagnosis can be made based on persistent digestive issues, the presence of increased eosinophils in biopsy samples and the absence of any other known cause for gastrointestinal eosinophilia.[1] Although peripheral eosinophilia is common in Eosinophilic Gastroenteritis (EoGE), it is not required for diagnosis. The majority of EoGE patients, including the patient being discussed, experience a temporary increase in peripheral blood eosinophilia, with eosinophil levels greater than 500/mm^3.[1] Endoscopic findings in patients with EoGE may appear normal or non-specific. Histology is the gold standard for diagnosing EGIDs in which eosinophils predominate over GI cells. There is currently no agreement on the threshold for intestinal eosinophils to be considered pathological. However, the accepted threshold for stomach gastritis is greater than 30 cells/hpf. In addition, the epithelium may exhibit degenerative and regenerative changes, as well as foveolar and crypt hyperplasia.[1] Except for the presence of eosinophilia (nonspecific), none of these findings are present in our patient, suggesting a different diagnosis. In addition, Eosinophilic gastritis is highly responsive to dietary restriction therapies in children[2] however our patient's symptoms continued despite change in diet. The other striking feature is the positive rapid urease test (RUT) on endoscopy which indicates a possibility of H.pylori infection. Recurrent abdominal pain is highly associated with H. pylori gastritis.[3] As per the guidelines of ESPGHAN, the diagnosis of H. pylori infection can be made by either confirming the presence of H. pylori-positive gastritis through histopathology and at least one other positive biopsy-based test or by obtaining a positive culture result.[4] In this child histopathology did not show H.pylori like organisms on the histopathology and a culture could not be done in the child. Hence H.pylori also could not be proven in the child. The child was treated with for H.pylori triple regimen but there was no improvement. CMPA is an immune response to the proteins found in cow's milk, which can be classified into two types: immunoglobulin E (IgE) mediated and non-IgE mediated. The IgE mediated response is caused by a type 1 hypersensitivity reaction, whereas the non-IgE mediated response is caused by a delayed type 4 hypersensitivity reaction.[5] The diagnosis of cow milk protein allergy is typically

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made by taking a detailed history of symptoms, conducting a skin prick test and measuring serum specific IgE levels to cow's milk protein, using an elimination diet and conducting an oral food challenge.[6] Although the negative predictive value of skin prick tests and specific IgE tests for immediate reactions is high, some patients may still experience a clinical reaction. If there is a strong suspicion of cow milk protein allergy despite negative IgE test results, an oral food challenge may be necessary to confirm the absence of clinical allergy. In cases where the diagnosis of food allergy in children is uncertain, an oral food challenge, whether open or blind, is still considered the most reliable method.[6] IgE mediated CMPA is treated by removing cow's milk protein (CMP) from the diet while for Non–IgE mediated: remove cow's milk protein as well as soy due to cross reactivity with CMP.[5] In our patient, thus the most likely diagnosis seems to be CMPA as there is a history of pain on drinking milk and milk products and also positive CMPA IgE. Confirmation of the same can only be ascertained when milk and milk products are completely eliminated from the diet and there is resolution of the symptoms.

Compliance with ethical standards

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