A 12-year-old girl with chronic vomiting and epigastric pain

History:

A 12-year-old girl has presented with vomiting for 1 month. The frequency is about 2-3 times a day. The vomitus contains digested food without bile stain. The emesis is not related to meals. After vomiting, she can normally eat. Her bowel movement is once daily with normal stools. There is no fever. After 2 days of symptoms, she was treated with antibiotics and antiemesis with a diagnosis of gastroenteritis. However, the symptom persists. Three weeks prior to admission, the vomiting became worsening (7-8 times a day) and occurred a half an hour after meals. Its characteristics remained the same. At this time, she also complained of epigastric pain, weakness, and anorexia. She was admitted to a primary care hospital and treated with intravenous fluid. The symptoms seemed to be improved. However, the vomiting resumed with a history of foul-smell watery diarrhea 2 time a day for 1 weeks before admission to Children's hospital. She lost significant weight during the illness.

Past history:

She was admitted because of Dengue hemorrhagic fever 2 years ago. Perinatal and immunization history are normal. There is no genetic and atopic diseases in the family. The patient refuses a history of contact tuberculosis. She denies regular uncooked food ingestion.

Physical examination:

General appearance- Thai girl, obesity, alert
Body weight 80 kg, height 158 cm
Vital signs: T 37 C, PR 80/min, RR 18/min, BP 110/70 mmHg
HEENT: not pale, no jaundice, pharynx and tonsils-not injected, normal TM
Heart: regular rhythm, no murmur
Lungs: normal breath sound, no adventitious sound
Abdomen: soft, mild distension, mild tenderness at epigastrium, no guarding, no rigidity, active bowel sound, no abnormal mass, fluid thrill and shifting dullness can not be evaluated.
Ext: no pitting edema
PR: soft stool, sphincter tone-normal
CNS: intact

Basic investigations:

CBC : Hct 50%, WBC 23.000/cumm, N34, L16, Mono1, Eo48, platelet 546,000/cumm
UA : WBC 3-5/HPF, protein +1
UC : E. coli > 10^5
Stool concentration: negative for parasite x 1 day
Na 132 mEq/L, K 3.6 mEq/L, Cl 96 mEq/L, CO2CP 27 mEq/L
US abdomen: massive ascites
EGD : Esophagus- normal mucosa. Bile reflux into the antrum and lower esophagus is noted. The gastric and duodenal mucosa appears erythematous with speckle pattern. The gastric rugae are normal. The endoscopic diagnosis is gastroduodenitis.
CLO test : negative
Pathology : Mild chronic duodenitis, acute and chronic gastritis (antrum), and mild chronic gastritis (body). The Helicobacter pylori is negative.
Abdominal paracentesis under ultrasound guidance : The ascites fluid is straw color. WBC 672/cumm (Eosinophil 100%), RBC 87/cumm, pandy +4, sugar-trace. Protein 5.7 g/dl (albumin 3.38 g/dl), LDH 359 IU/L, gram stain-negative, AFB stain-negative, Latex-negative, culture-no growth.
Ascitic fluid cytology : negative for malignancy
Blood chemistry : BS 101mg/dl, albumin 4.04 g/dl, LDH 750 IU/L
Immunoglobulin levels: IgE 181.9 mg/dl (0-100), IgG 1,180 mg/dl (694-1618), IgA 99,2 mg/dl (68-378), IgM 142 mg/dl (60-263)
Zn level : 0.034 mg/dl (0.05-0.15)

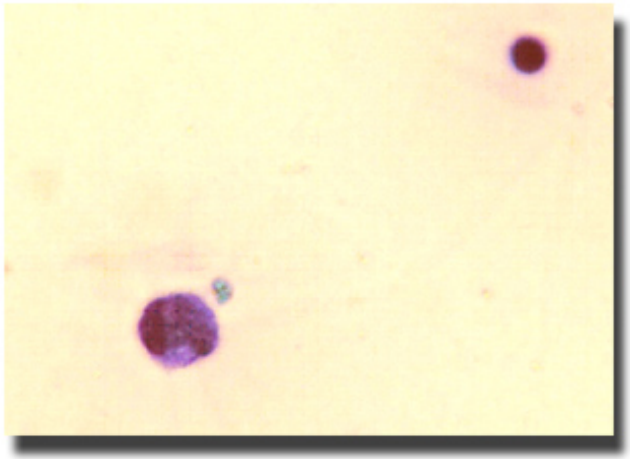

Figure 1: Eosinophils in the ascitic fluid (wright stain).

Diagnosis :

Eosinophilic ascites and urinary tract infection

Treatment :

Cetriaxone x 7 days for UTI
Prednisolone x 2 weeks for eosinophilic ascites

Clinical course :

>>At 2 weeks follow up, the patient was fine. Her body weight was 73 kg. There was no vomiting or abdominal pain. CBC showed Hct 42.9%, WBC 17,200/cumm, N81, L19, platelet 525,000/cumm. LFT included albumin/globulin 4.82/2.82 g/dl, cholesterol 243 mg/dl, TB/DB 1.23/0.30 mg/dl, AST/ALT 82/170 U/L, AP 100 U/L. The prednisolone ws then taperred off in 1 month. She was also treated with ZnSO4 cap 75 mg elemental Zn daily.
>>One month later, the symptoms remained controlled. She gained 1 kg of weight. LFT was improved, including albumin/globulin 4.8/2.9 g/dl, cholesterol 144 mg/dl, TB/DB 1.08/0.25 mg/dl, AST/ALT 39/37 U/L, AP 116 U/L. The steroid and ZnSO4 were then discontinued.
>>Two month later, she has been still fine. At this time, her weight is 79 kg. The Zn level returns to normal (0.2 mg/dl).

Eosinophilic Gastroenteropathy

This is an uncommon disease, characterized by presence of abnormal GI symptoms, evidence of eosinophilic infiltration (>20 cells/HPF) in more than one area of the GI tract, absence of identified causes of eosinophilia, and exclusion of eosinophilic involvement outside the GI tract. Its etiology has remained unknown, but it is associated with history allergic diseases in 0-70% of the cases. Food allergy is usually undetermined in most cases. The eosinophilic recruitment and activation are induced by 3 major cytokines, including IL-3, IL-5, and GM-CSF.

History and physical examination:

The symptoms depend on the specific area and depth of bowel wall of involvement. These include abdominal pain, anorexia, bloating, postprandial nausea/vomiting, weight loss, diarrhea, edema, dysphagia, and GI bleeding. Fify percent of the cases may have a history of atopy. If the predominant site of involvement is gut mucosa, the patient will present mainly with abdominal pain, nausea/vomiting, diarrhea, weight loss, GI bleeding, protein-loosing enteropathy, and malabsorption. In contrast to muscular involvement, the principle presentation is intestinal obstruction, Eosinophilic ascites, the least common form, can be seen in a patient with serosal layer involvement, similar to the present patient.

Laboratory findings:

Seventy-a hundred per cent of the cases is associated with peripheral eosinophilia. In some cases, there may be presence of bone marrow precursor of the eosinophils in the absence of peripheral eosinophilia. The serum IgE might be elevated. Iron-deficiency anemia and hypoalbuminemia can be seen in the patient with GI bleeding and protein-loosing enteropathy, respectively. Imaging studies, including GI contrast study, ultrasound, and CT scan, are not specific. Endoscopy with biopsy is the most helpful diagnostic tool. Sometimes, surgical specimen is required for establishing a correct diagnosis, particularly in a case with muscular involvement.

Treatment:

If specific food hypersensitivity can be identified, dietary elimination is the first step treatment. In infant, elemental formula might be useful and strongly recommended because of high incidence of cow's milk allergy. Unfortunately, the specific food antigen is usually unidentified, particularly in the older child. Therefore, corticosteroids, cromolyn (mast cell stabilizer), and montelukast (leukotriene receptor antagonist) are frequently precribed. In case of eosinophilic esophagitis, the topical use of swallowed fluticasone propionate (via inhaler without spacer) is a treatment option.

Learning points in the present case

> Detection of ascites by physical examination is sometimes difficult, particularly in an obese patient with minimum ascitic fluid.
> In such case with high index of suspicious, imaging study, for instance, abdominal ultrasonography should be considered.
> Endoscopy with tissue biosy is of limtted value in case of muscular and serosal involvement.
> Intetinal capsule biopsy might be helpful in this case.