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Short Communication

Iron Deficiency Anemia; What If The Etiology is Not That Common?

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SHORT COMMUNICATION

A 4-year-old boy presented with pallor and fatigue for 7 months. During the last year, he also complained of intermittent, dull, postprandial abdominal pain, and early satiety. He required a blood transfusion 6 months ago because an episode of severe anemia (Hemoglobin (Hgb) level 3.2g/dL).

Physical examination revealed tachycardia and mucocutaneous pallor. His body weight and height were consistent with moderate malnutrition. His weight and height were stuck in the 10 percentile since he was 6 months old.

Laboratory test showed severe microcytic anemia (Hgb 2.88g/dl, Hematocrit 10,10%, mean corpuscular volume 56,40 fL, red cell width 20.70%, Ferritin 3,44 ng/ml) without signs of hemolysis. A peripheral blood smear was compatible with iron deficiency anemia; no schistocytes, white-cell, or platelet alteration were seen. Hgb electrophoresis showed unremarkable findings. He received a packed red blood cell transfusion and started treatment with oral iron tablets 6mg/kg/day.

He had a positive H Pylori (HP) breath test, so he was subsequently treated and HP eradicated. The fecal occult blood test was repeatedly negative.

Upper endoscopy showed hiatal hernia with the majority of the stomach identified above the diaphragm. No signs of bleeding or gastritis were seen, and mucosal biopsies were normal. Upper gastrointestinal series showed both the gastroesophageal junction and the gastric fundus herniated above the diaphragm, confirming a type III hiatal hernia.

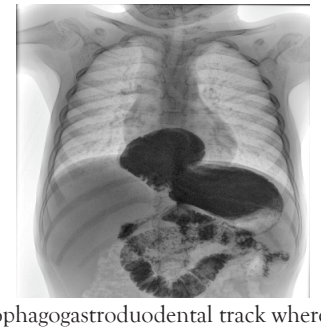

Figure 1: Esophagogastroduodental track where we can see a type III hiatal hernia

After 5 months of oral iron treatment, he reached a maximum Hgb level of 10.20g/dL, so weekly intravenous iron treatment was started. However, his Hgb levels dropped again after its interruption.

Hiatal hernia was surgically repaired by excision of the hernia sac and closure of diaphragmatic crura. A Nissen fundoplication was added. IDA resolved after the corrective surgery with no need for further iron treatment. Six months after surgery, the patient is still asymptomatic with height and weight in 50th percentile for age and sex.

DISCUSSION

In adults, hiatal hernia is commonly seen as a consequence of obesity and high intrabdominal pressure, or esophageal-diaphragmatic membrane hyperlaxity.

However, HH is rarely seen in children. Olsen et al. reviewed the incidence of HH during 9 years in Mayo Clinic; only 20 children were diagnosed compared with more than 17000 adults [1].

In the pediatric population, most of HH are congenital. In children, a surgical approach should be made initially, even though the patient is asymptomatic [2].

The exact mechanism by which HH causes IDA is not clear: HH could cause acid-reflux that could damage the esophageal wall with subsequent bleeding. Bleeding could come from Cameron ulcers, little linear erosions localized in herniated gastric mucosa. However, in our case, there was no evidence of bleeding. Shih et al. published a similar case in 2016. They proposed that IDA could be caused by malabsorption due to herniated stomach [3-6]. Our theory is that early satiety could be the cause of the malnutrition. Intrathoracic stomach dilates when the patient eats, causing discomfort and decreasing food intake. This could explain our patient's hyporexia, which resolved soon after surgical treatment.

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CONCLUSION

There are very few reported cases of IDA and HH in children, and, to our knowledge, none of them had a negative fecal occult blood test and no evidence of active bleeding.

Once common causes of IDA in children are ruled out, HH must be considered as a possible cause of severe refractory IDA.

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