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A DEVASTATING BOTTLENECK: SEVERE GASTRIC OUTLET OBSTRUCTION WITH PROFOUND ANAEMIA AND MALNUTRITION IN A 16-YEAR-OLD MALE

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ABSTRACT Upper gastrointestinal obstruction in adolescent patients typically raises immediate concern for peptic ulcer disease, foreign body ingestion, or aggressive neoplastic processes. We present the case of a 16-year-old male who presented with chronic, progressive abdominal pain, post-prandial vomiting, and an alarming 19 kg weight loss over two years, resulting in severe cachexia with severe anaemia. Diagnostic endoscopy revealed extreme pyloric and duodenal ulceration with structural narrowing so profound that the endoscope could not be negotiated past the first part of the duodenum. Beyond the mechanical gastric outlet obstruction, further investigation revealed severe microcytic hypochromic anaemia secondary to chronic mucosal blood loss. Rather than a mechanical or neoplastic culprit, histopathological examination of biopsy tissue obtained during endoscopy demonstrated 25–30 eosinophils per high-power field (HPF), establishing a definitive diagnosis of Eosinophilic Gastritis (EG). Following collaborative discussion with a gastroenterologist, targeted therapy with oral corticosteroids was initiated, alongside aggressive mucosal protection and parenteral nutrient restoration. This combined regimen led to dramatic clinical improvement, successful endoscopic resolution with complete luminal patency, and eventual dietary advancement to a normal diet.

KEYWORDS : Duodenal narrowing, Gastric outlet obstruction, Cachexia, Microcytic hypochromic anaemia, Eosinophils, Oral steroids, Eosinophilic gastritis.

CASE PRESENTATION

A 16-year-old male college student presented with a one-year history of dull, progressive, diffuse abdominal pain that was characteristically aggravated after meals. Over the preceding 3 months, he experienced a severe loss of appetite, culminating in Intractable, non-projectile vomiting of partially digested food material. Clinical history was notable for profound, unintentional weight loss, dropping from 56 kg to 37 kg over a two-year period. He reported no history of fever, hematemesis, melena, altered bowel habits, passage of worms in stool, or known food allergies.

On physical examination, the patient was thin, cachectic, and exhibited marked pallor. His recorded weight was 37.7 kg, corresponding to an extremely low Body Mass Index (BMI) of 14.73 kg/m^2.

INVESTIGATIONS

	Hb (mg/dl)	Stool occult blood
Day 1	9.4	Present
2nd month	11.8	Absent
3rd month	13.3	Present
4th month	14.7	Absent
6th month	15.2	Absent
7th month	15.4	Absent

Diagnostic Endoscopy & Biopsy

An urgent Esophagogastroduodenoscopy (EGD) was performed under local anaesthesia to evaluate the persistent vomiting and gastric outlet obstruction symptoms. It reported:

- Stomach: Gastric stasis (retained food) with marked erythema and deep pyloric ulcers.
- Duodenum & Obstruction: Severe oedema and ulceration in D1 causing critical duodenal narrowing (endoscope couldn't be negotiated beyond).
- Histopathology: Dense inflammatory infiltrate with 25–30 eosinophils/HPF, confirming Eosinophilic Gastritis.

MANAGEMENT & CLINICAL COURSE

Due to the severe duodenal narrowing and secondary gastric outlet obstruction, a combined medical and nutritional stabilization plan was executed in consultation with a gastroenterologist:

1. **Anti-inflammatory & Immunosuppressive Therapy:** Treatment with oral steroids was initiated. This therapeutic intervention led to a gradual, sustained improvement in the patient's clinical condition. The patient remains on oral steroid therapy, which is currently being gradually tapered.
2. **Acid Suppression & Mucosal Protection:** The patient was placed on a proton pump inhibitor (Rabeprazole) and an alginate oral suspension (Gaviscon) to shield the ulcerated pyloric and duodenal surfaces.
3. **Helicobacter pylori Eradication:** Empirical triple therapy consisting of oral Amoxicillin and Clarithromycin was completed. A course of deworming too was given.
4. **Nutritional & Parenteral Micronutrient Correction:** Due to severe malabsorption and mechanical narrowing, oral iron was avoided. The patient was managed with intravenous Ferric Carboxy Maltose under close hospital supervision, alongside weekly oral Vitamin D3 and intramuscular Methyl cobalamin injections.
5. **Dietary Progression:** The patient was initially supported on liquid nutrition to bypass the narrowing. As the mucosal swelling and obstruction resolved under corticosteroid therapy, the patient was gradually weaned off the liquid diet and currently tolerates a normal, solid diet without difficulty.

Follow-up Endoscopy

During the course of the ongoing, tapered steroid therapy, a repeat upper GI endoscopy was performed to assess mucosal healing and structural patency:

- **Scope Negotiation:** In stark contrast to the initial evaluation, the endoscope was easily and successfully negotiated beyond the D1 and D2 duodenal junction, confirming complete resolution of the prior mechanical obstruction.

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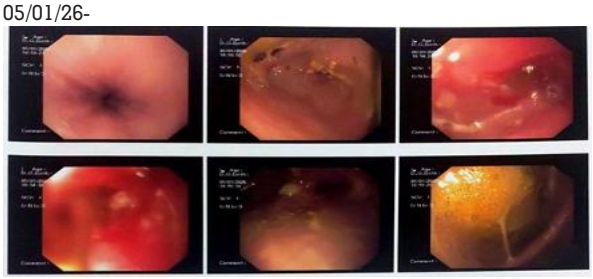

- Food material seen in stomach

- Erythema and ulceration in pylorus
- D1 oedematous and scope could not be negotiated beyond D1-D2 junction

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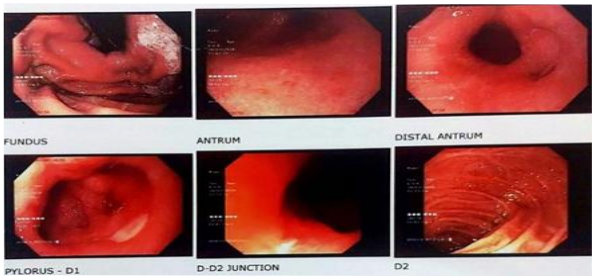

- No food residue in stomach.
- Paediatric scope could be negotiated through D1-D2 junction.
- Erosive gastritis, duodenal ulcer, D1-D2 junction stricture noted.

- **Histopathology:** Microscopic examination of the follow-up biopsy tissue showed a highly significant decrease in mucosal inflammation and a marked reduction in tissue eosinophil infiltration.

DISCUSSION

This patient's dramatic clinical presentation was driven by Eosinophilic Gastritis (EG), a rare, benign inflammatory disease characterized by prominent eosinophilic infiltration of gastric and duodenal tissues. Its clinical presentation depends heavily on which histological layer is primarily affected: mucosal, muscularis, or subserosal[3].

In this case, the patient suffered from a severe combination of mucosal and muscularis-layer disease:

1. **Mucosal Infiltration:** Led to severe mucosal friability, pyloric and duodenal ulceration, and chronic micro-bleeding. This explains his chronic microcytic hypochromic anaemia and early positive stool occult blood.
2. **Muscularis Infiltration:** Caused extensive inflammatory thickening, muscle hypertrophy, and tissue oedema. In this patient, this manifested as severe duodenal narrowing effectively creating a gastric outlet obstruction[2]. This blockage led to post-prandial epigastric pain, delayed gastric emptying, persistent vomiting, and a devastating state of cachexia (loss of 19 kg).

The histopathological confirmation of 25–30 eosinophils/HPF was the diagnostic turning point[1]. While mucosal healing and nutritional support stabilized the patient, targeted therapy with oral corticosteroids—selected after specialist gastroenterology consultation—directly addressed the deeper muscularis inflammation . This resulted in the rapid reduction of duodenal wall oedema, allowing the patient to progress from a restricted liquid diet to normal solid food, and facilitating a clean follow-up endoscopy (performed during the ongoing, tapered steroid regimen) where the luminal narrowing was no longer present.

CONCLUSION

Severe upper gastrointestinal narrowing and profound weight loss in an adolescent can easily mimic malignant processes or advanced cicatricial peptic ulcer disease. Eosinophilic gastritis should be kept high on the differential diagnosis for patients presenting with gastric outlet obstruction, persistent microcytic anaemia, and cachexia. A multidisciplinary approach utilizing specialist gastroenterology consultation, targeted oral corticosteroid therapy with a gradual taper, and parenteral nutrient restoration can safely reverse severe duodenal strictures, precluding the need for aggressive surgical interventions.

Learning Points

1. **Obstruction Mimics:** Inflammatory disorders like eosinophilic gastritis can cause structural narrowing in the duodenum severe enough to prevent endoscopic passage, mimicking malignancy or chronic peptic cicatrisation.
2. **Avoid Premature Surgical Intervention:** Even when upper GI narrowing is severe enough to cause objective mechanical obstruction, clinicians must resist the urge to rush into surgical decompression or bypass procedures. A trial of targeted conservative medical management should always be prioritized, as inflammatory and eosinophilic strictures can be highly reversible5.
3. **Histopathological Key:** A finding of elevated eosinophils (25–30 eosinophils/HPF) on mucosal biopsy is crucial to distinguishing eosinophilic gastritis from classic peptic ulcer disease or gastric malignancies in patients presenting with outlet obstruction.
4. **Steroid Response in EG:** Systemic oral corticosteroids are highly effective at reducing the transmural inflammation and muscularis oedema responsible for duodenal narrowing, allowing for complete structural resolution under a planned, ongoing steroid taper.
5. **Objective Monitoring:** Repeat endoscopy and repeat mucosal biopsies serve as excellent, objective parameters to confirm both macroscopic luminal patency and microscopic resolution of eosinophilic inflammation during active therapy.
6. **Strangely:** Peripheral blood eosinophilia is NOT a mandatory feature for deep tissue/ gastrointestinal eosinophilic infiltration. 20-80% patients show peripheral eosinophilia meaning that normal blood tests DO NOT rule out infiltration of deeper tissues. A high index of suspicion must be maintained.

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