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A rare case of gastric inlet and outlet obstruction in a perimenopausal woman

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Abstract : A 42 year old lady presented with complaints of progressive dysphagia, vomiting and abdominal distension for five years. Patient was emaciated and anaemic on presentation. Endoscopy showed stricture oesophagus at 34 cm. Barium study showed bird beak appearance of lower end of oesophagus with an air fluid level and gross dilatation of stomach with delayed emptying of contrast. CT abdomen showed a grossly distended stomach suggesting partial obstruction due to growth or stricture. A diagnosis of achalasia cardia with partial gastric outlet obstruction was made. Patient underwent Hellers cardiomyotomy with Toupet fundoplication and a loop gastrojejunostomy with a jejunojejunostomy. Vagotomy was deferred as we were of the opinion that the gastric obstruction is due to dysmotility. Postoperative recovery was uneventful. There have been very few case reports of combined gastric inlet and outlet obstruction. Possibilities include achalasia cardia with antral web, distal carcinoma, obstruction due to chronic duodenal ulcer and adult onset hypertrophic stenosis. Lower oesophageal growth associated with any of the above can also present in a similar fashion. In such cases the possibility of connective tissue disorders should also be kept in mind though they tend to affect the oesophagus more than other organs.

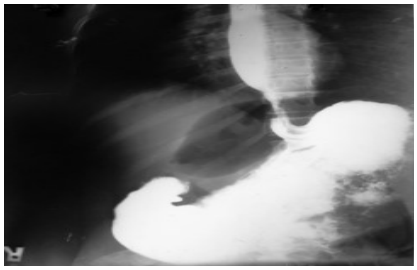
Keyword :achalasia cardia, GOO, gastric outlet obstruction

Introduction

Achalasia is a primary motor disorder of the oesophagus characterized by insufficient lower oesophageal sphincter relaxation and loss of esophageal peristalsis. The primary problem is neuronal in origin. Bolus transfer into the oesophagus is normal but the oesophageal musculature lacks the ability to propel its content down the oesophagus and into the stomach. Achalasia cardia may rarely present with gastric outlet obstruction1. It is very unusual to have simultaneous presentation of achalasia cardia with gastic outlet obstruction.

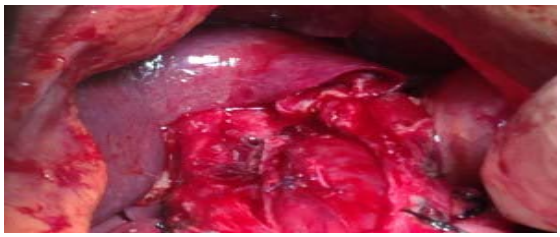
Case Profile

A 42 year old lady presented with complaints of retrosternal burning sensation, difficulty in swallowing and vomiting for 5 years. The retrosternal burning sensation occurred immediately following food intake and relieved with medications. The difficulty in swallowing was for solid foods initially but was present for both solids and liquids eventually. She had about 6-8 episodes of vomiting per week, which was immediately following food intake. Vomitus was foul smelling and contained food matter consumed more than 24 hours before. She also complained of abdominal distension noted for the last eighteen months. Also, history of decreased appetite and recent loss of weight was recorded.



Barium study

On general examination, vitals were stable, patient was pale and emaciated with no evidence of lymphadenopathy. Systemic examination revealed a distended abdomen with umbilicus flushed to the surface. Visual gastric peristalsis was observed. Percussion note was normal and succussion splash was noted on auscultation. Per rectal examination was normal. Haemogram revealed anaemia. Liver and renal function tests were within normal limits. Serum total protein was 4.7 g/dl and electrolyte panel revealed borderline hyperkalemia(5.2 Meq/L). Barium study showed bird beak appearance of lower end of oesophagus with an air fluid level and gross dilatation of stomach with delayed emptying of contrast. CT abdomen showed a grossly distended stomach suggesting partial obstruction due to growth or stricture. Upper gastrointestinal endoscopy showed a stricure oesophagus at 34 cm. A diagnosis of achalasia cardia with partial gastric outlet obstruction was made.



Fundoplication and loop GJ with JJ

Discussion

Patient underwent Heller's cardiomyotomy with Toupet fundoplication and loop gastrojejunostomy(GJ) with jejunojejunostomy(JJ). Vagotomy was deferred as we were of the opinion that the gastric obstruction is due to dysmotility. Postoperative recovery was uneventful. Patient was discharged and advised to continue proton pump inhibitors. The synchronized ability of upper and lower oesophageal sphincters to relax allows sufficient control of digestive transit. Relaxation of lower oesophageal sphincter is essential for oesophageal emptying and, similarly, pyloric relaxation permits gastric emptying. When the relaxation response of these sphincters is impaired, luminal transit is altered, as is the case of achalasia and chronic duodenal ulceration with healing by fibrosis. In achalasia, there is a disturbance of oesophageal motility. There is absence of peristalsis in the body of the oesophagus and failure of the lower oesophageal sphincter to relax in response to swallowing.

The pathology involved in achalasia is the degeneration of ganglion cells in myenteric plexus of oesophageal body and the LES. Although the cause for the degenerative process is unclear, the end result is loss of inhibitory neurotransmitters nitrous oxide and vasoactive intestinal polypeptide and consequently imbalance between the excitatory and inhibitory neurons. This results in unapposed cholinergic activity that leads to incomplete relaxation of the LES and peristalsis due to loss of latency gradient along the oesophageal body2. The problem is easily diagnosed with typical complaints and radiological signs. There is difficulty in swallowing especially of liquid and cold food with regurgitations of food at a stage. The radiological features include a dilated oesophagus with a bird beak-like lower end. There is little or no barium in the stomach. The oesophagus may even reach a great proportion that it can even be seen on plain chest radiograph.

Oesophagoscopy is invaluable in the diagnosis. Helier's operation with the different modifications is usually adequate for its treatment. The outcome is good for early cases of achalasia. Gastric outlet obstruction (GOO) represents a clinical and pathophysiological consequence of any disease process which produces mechanical impediment to gastric emptying. Intrinsic or extrinsic obstruction of the pyloric channel or duodenum is the usual pathophysiology of GOO and the mechanism of obstruction depends upon the underlying etiology. Classification of diseases causing GOO into two welldefined groups of benign and malignant facilitates management and treatment. Historically GOO has been considered a disease process synonymous with chronic peptic ulcer disease. However since the advent of proton pump inhibitors, the complications from peptic ulcer disease have drastically decreased with a change in ratio between benign and malignant gastric outlet obstruction4. Most patients with GOO present with vomiting as their cardinal symptom and tend to develop dehydration and dyselectrolytemia if untreated. Malnutrition and weight loss are frequent when the condition approaches chronicity and are most significant in patients with malignant etiologies. Outlet obstruction secondary to chronic duodenal ulceration is usually due to a combination of fibrosis from repeated cycles of ulceration and healing, oedema, inflammation, and pylorospasm associated with current activity of the ulcer. Pyloric obstruction usually occurs after a long history of duodenal ulcer distress and is almost always resulting from severe cicatricial duodenal deformity and there is little expectation from conservative management. The cicatrisation results in the impairment of the propulsive motility of the antrum and the active relaxation of the pyloric sphincter. Such patients present with features of gastric outlet obstructions consequence upon failure of relaxation response of the pyloric sphincter. Barium meal exanfination is helpful in the diagnosis. Treatment should relieve the obstruction and control the ulcer diathesis. There have been very few case reports of combined gastric inlet and outlet obstruction. Possibilities include achalasia cardia with antral web, distal carcinoma, obstruction due to chronic duodenal ulcer and adult onset hypertrophic stenosis. Lower oesophageal growth associated with any of the above might also present in a similar fashion. In such cases the possibility of connective tissue disorders should also be kept in mind though they tend to affect the oesophagus more than other organs1.3. Corrosive acid ingestion may present with such a picture. Here, gastric outlet obstruction presents before or along with oesophagal obstruction5. Heller's myotomy has remained the surgical treatment of choice for Achalasia for many years. Recent guidelines have unanimously demonstrated that fundoplication following myotomy is both cost effective and superior to myotomy alone. Among the approaches available, Toupet or posterior fundoplication has shown to have a slight, albeit insignificant advantage over Dor or anterior fundoplication2.

Our patient was treated with Heller's cardiomyotomy with Toupet fundoplication and loop GJ with JJ.Surgery forms the final option for patients presenting with refractory GOO. Most common surgeries for peptic strictures include vagotomy and antrectomy, vagotomy and pyloroplasty, truncal vagotomy and gastrojejunostomy, pyloroplasty and laparoscopic variants. Gastrojejunostomy (Billroth II reconstruction) can be considered in patients with preserved anatomy like peptic GOO, however with altered anatomy, Rouxen Y jejunostomy would be the preferred option. Placement of a jejunostomy tube at the time of surgery should be considered, as patients with GOO are already malnourished. Also in chronically dilated conditions the stomach is slow to recover the normal rate of emptying. In peptic GOO gastrojejunostomy can be combined with truncal vagotomy and antrectomy. Laparoscopic gastrojejunostomy for the relief of GOO is associated with a smoother and more rapid postoperative recovery

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