



Clinical factors responsible for gastric outlet obstruction in patients from Bihar region

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Abstract

From the standpoint of pathology, the term pyloric stenosis is usually inaccurate at least in adult patients, since the site of obstruction is rarely situated at the pylorus itself but, is more often placed immediately proximal to the sphincter where the diagnosis of carcinoma is most probable or more distally in the duodenal bulb where the cause is almost invariably a duodenal ulcer. Hence present study was planned to evaluate the clinical factors responsible for gastric outlet obstruction in patients from Bihar region.

The present study was planned in Department of Gastroenterology, Ruban Memorial Hospital, Patna, Bihar. The total 25 cases of the gastric outlet obstruction referred to our hospital were enrolled in the present study.

Gastric Outlet Obstruction is the commonest disease with significant morbidity and mortality. Patients with gastric outlet obstruction due to cicatrized duodenal ulcer require truncal vagotomy with posterior gastrojejunostomy. Vagotomy is optional in view of better response with drugs for APD. Antral carcinoma cases require curative or palliative surgery depending on the stage of the disease.

Keywords: gastric outlet obstruction, paralytic ileus, recovery, Bihar region, etc

Introduction

Gastric outlet obstruction (GOO) is a medical condition where there is an obstruction at the level of the pylorus, which is the outlet of the stomach. Individuals with gastric outlet obstruction will often have recurrent vomiting of food that has accumulated in the stomach, but which cannot pass into the small intestine due to the obstruction. The stomach often dilates to accommodate food intake and secretions. Causes of gastric outlet obstruction include both benign causes (such as peptic ulcer disease affecting the area around the pylorus), as well as malignant causes, such as gastric cancer. Causation related to ulcers may involve severe pain which the patient may interpret as a heart condition/attack. Treatment of the condition depends upon the underlying cause; it can involve antibiotic treatment when *Helicobacter pylori* is related to an ulcer ^[1], endoscopic therapies (such as dilation of the obstruction with balloons or the placement of self-expandable metallic stents), other medical therapies, or surgery to resolve the obstruction.

Gastric outlet obstruction (GOO, also known as pyloric obstruction) is not a single entity; it is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying. Clinical entities that can result in GOO generally are categorized into two well-defined groups of causes: benign and malignant. This classification facilitates discussion of management and treatment. In the past, when peptic ulcer disease (PUD) was more prevalent, benign causes were the most common; however, one review showed that only 37% of patients with GOO have benign disease and the remaining patients have obstruction secondary to malignancy ^[2].

Gastric outlet obstruction can be a diagnostic and treatment dilemma. Despite medical advances in the acid suppression mechanism, the incidence of GOO remains a prevalent

clinical problem in benign PUD. Also, an increase in the number of cases of GOO seems to be noted secondary to malignancy; this is possibly due to improvements in cancer therapy, which allow patients to live long enough to develop this complication. As part of the initial workup, exclude the possibility of functional nonmechanical causes of obstruction, such as diabetic gastroparesis. Once a mechanical obstruction is confirmed, differentiate between benign and malignant processes because definitive treatment is based on recognition of the specific underlying cause.

Carry out diagnosis and treatment expeditiously, because delay may result in further compromise of the patient's nutritional status. Delay will also further compromise edematous tissue and complicate surgical intervention. Orient initial management to identification of the primary underlying cause and to the correction of volume and electrolyte abnormalities. Barium swallow studies and upper endoscopy are the main tests used to help make the diagnosis. Tailor treatment to the specific cause. The stomach is located mainly in the left upper quadrant beneath the diaphragm and is attached superiorly to the esophagus and distally to the duodenum. The stomach is divided into four portions: cardia, body, antrum, and pylorus. Inflammation, scarring, or infiltration of the antrum and pylorus are associated with the development of GOO.

The duodenum begins immediately beyond the pylorus and mostly is a retroperitoneal structure, wrapping around the head of the pancreas. The duodenum classically is divided into four portions. It is intimately related to the gallbladder, liver, and pancreas; therefore, a malignant process of any adjacent structure may cause outlet obstruction due to extrinsic compression. Intrinsic or extrinsic obstruction of the pyloric channel or duodenum is the usual pathophysiology of GOO; the mechanism of obstruction depends upon the underlying etiology. Patients present with intermittent symptoms that progress until obstruction is

complete. Vomiting is the cardinal symptom. Initially, patients may demonstrate better tolerance to liquids than solid food. In a later stage, patients may develop significant weight loss due to poor caloric intake. Malnutrition is a late sign, but it may be very profound in patients with concomitant malignancy. In the acute or chronic phase of obstruction, continuous vomiting may lead to dehydration and electrolyte abnormalities. When obstruction persists, patients may develop significant and progressive gastric dilatation. The stomach eventually loses its contractility. Undigested food accumulates and may represent a constant risk for aspiration pneumonia.

The major benign causes of GOO are PUD, gastric polyps, ingestion of caustics, pyloric stenosis, congenital duodenal webs, gallstone obstruction (Bouveret syndrome), pancreatic pseudocysts, and bezoars. PUD manifests in approximately 5% of all patients with GOO. Ulcers within the pyloric channel and first portion of the duodenum usually are responsible for outlet obstruction. Obstruction can occur in an acute setting secondary to acute inflammation and edema or, more commonly, in a chronic setting secondary to scarring and fibrosis. *Helicobacter pylori* has been implicated as a frequent associated finding in patients with GOO, but its exact incidence has not been defined precisely. Within the pediatric population, pyloric stenosis constitutes the most important cause of GOO. Pyloric stenosis occurs in 1 per 750 births. It is more common in boys than in girls and also is more common in first-born children. Pyloric stenosis is the result of gradual hypertrophy of the circular smooth muscle of the pylorus.

Pancreatic cancer is the most common malignancy causing GOO. Outlet obstruction may occur in 10-20% of patients with pancreatic carcinoma. Other tumors that may obstruct the gastric outlet include ampullary cancer, duodenal cancer, cholangiocarcinomas, and gastric cancer. Metastases to the gastric outlet also may be caused by other primary tumors. The incidence of GOO has been reported to be less than 5% in patients with PUD, which is the leading benign cause of the problem. Five percent to 8% of ulcer-related complications result in an estimated 2000 operations per year in the United States [3]. The incidence of GOO in patients with peripancreatic malignancy, the most common malignant etiology, has been reported as 15-20%.

Once the diagnosis of gastric outlet obstruction (GOO) is suspected, request a surgical consultation. GOO due to benign ulcer disease may be treated medically if results of imaging studies or endoscopy determine that acute inflammation and edema are the principal causes of the outlet obstruction (as opposed to scarring and fibrosis, which may be fixed). If medical therapy conducted for a reasonable period fails to alleviate the obstruction, then surgical intervention becomes appropriate. Typically, if resolution or improvement is not seen within 48-72 hours, surgical intervention is necessary. The choice of surgical procedure depends upon the patient's particular circumstances; however, vagotomy and antrectomy should be considered the criterion standard against which the efficacy of other procedures is measured [4].

In cases of malignant obstruction, weigh the extent of surgical intervention for the relief of GOO against the malignancy's type and extent, as well as the patient's anticipated long-term prognosis. As a guiding principle, undertake major tumor resections in the absence of metastatic disease in a patient who can withstand such a

procedure from a nutritional standpoint. In patients with largely metastatic disease, determine the degree of surgical intervention for palliation in light of the patient's realistic prognosis and personal wishes. Contraindications for surgery relate to the underlying medical condition. Most patients benefit from an initial period of gastric decompression, hydration, and correction of electrolyte imbalances. In patients who are severely malnourished, postponing surgical intervention until the nutritional status has been optimized may be wise. In selective cases, some patients may benefit from total parenteral nutrition (TPN) or distal tube feeding (eg, placed via a percutaneous jejunostomy). One of the relative contraindications for surgery is the presence of advanced malignancy; in these cases, in which life expectancy may be limited to a few months, palliation via endoscopically placed stents should be considered. Overall, every patient with GOO deserves evaluation by a surgeon. Even if the patient has unresectable disease, palliative surgical measures may improve the quality of life [5, 6].

From the standpoint of pathology, the term pyloric stenosis is usually inaccurate at least in adult patients, since the site of obstruction is rarely situated at the pylorus itself but, is more often placed immediately proximal to the sphincter where the diagnosis of carcinoma is most probable or more distally in the duodenal bulb where the cause is almost invariably a duodenal ulcer. Hence present study was planned to evaluate the clinical factors responsible for gastric outlet obstruction in patients from Bihar region.

Methodology

The present study was planned in Department of Gastroenterology, Ruban Memorial Hospital, Patna, Bihar. The total 25 cases of the gastric outlet obstruction referred to our hospital were enrolled in the present study.

Gastric contents were aspirated through Ryle's tube after an overnight fast. Saline load test was performed in all cases. 750ml of normal saline was infused through Ryle's tube, which was then clamped and released after half an hour, volume of aspirate was noted down. Any volume >400 ml was considered significant. Detailed history, physical examination and investigation for pre-operative assessment was done in all cases. Upper Gastro-Intestinal endoscopy was done in all cases for diagnostic confirmation. Biopsies were taken wherever required. Barium meal examination was done in few cases of corrosive stricture as the scope couldn't be passed beyond. Intra operative findings were noted down and case was followed up in the post-operative period.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion criteria: Presence of projectile vomiting of undigested food material, succussion splash heard 3-4 hours after meal, visible gastric peristalsis, presence of mass with above features, Gastric overnight aspirate of >200ml in fasting state. Positive saline load test: Retention of more than 400 ml of normal saline 30minutes after administration of 750ml of NS, OGD demonstrating Gastric outlet obstruction

Exclusion criteria: Patients below 18 years, pregnant

women, Patients with any history of previous cancer.

Results & Discussion

Gastric Outlet Obstruction is a disorder wherein there is an obstruction in the opening of the stomach (Pylorus), blocking the entrance of ingested food coming from the stomach to the duodenum. Gastric Outlet Obstruction is the one of the most common problem encountered in general surgery, Predominant cause for Gastric Outlet Obstruction have changed substantially with identification of H. Pylori and the use of proton pump inhibitor. Until the late 1970's benign disease was responsible for the majority of Gastric Outlet Obstruction in adult, while malignancy accounted for only 10-39% of causes, By contrast in recent decades 50-80% of cases have been attributed to malignancy. In this study the various aspects and management of Gastric Outlet Obstruction are analysed.

Gastric outlet obstruction is the more accurate term for the commonly used term pyloric stenosis. From the standpoint of pathology, the term pyloric stenosis is usually inaccurate atleast in adult patients, since the site of obstruction is rarely situated at the pylorus itself but is more often placed immediately proximal to the sphincter where the diagnosis of carcinoma is most probable or more distally in the duodenal bulb where the cause is almost invariably a duodenal ulcer.

Table 1: Causes of gastric outlet obstruction

Causes	Number of Cases
Infantile hypertrophic pyloric stenosis	6
Cicatrised duodenal ulcer	12
Carcinoma pyloric antrum	7
Total	25

Table 2: Distribution according to symptoms

Symptoms	Cicatrised duodenal ulcer	Carcinoma antrum	Number of Cases
Pain	12	6	18
Vomiting	12	7	19
Anorexia	8	7	15
Weight loss	7	7	14
Hematemesis	2	3	5
Malena	6	3	9

Table 3: Distribution as per the signs

Number	Duodenal ulcer	Carcinoma pyloric regions	Total
Pallor	10	7	17
VGP	12	6	18
Succession splash	12	5	17
Palpable mass	9	2	11
Dehydration	8	5	13

Table 4: Types of surgical procedures adopted in the study.

Cases type	Procedure	Number
Duodenal ulcer	Truncal vagotomy with posterior gastro jejunostomy	12
Carcinoma antrum	Billroth II gastrectomy	4
	Anterior gastro jejunostomy	3

Surgical treatment has better results on long term follow up but it cannot be offered, initially, to patients with poor clinical status because of increased morbidity and mortality. Based on the fact that less than 40% of patients who require

palliative care are fit to undergo a surgical procedure, the need to achieve this objective with a less invasive, safer and effective method has been made clear.

In agreement with other studies [7, 8] the diagnosis of gastric outlet obstruction in this study was based on clinical presentation, an upper gastrointestinal barium study, and/or an inability during upper endoscopy to intubate the second portion of the duodenum (Upper gastrointestinal endoscopy) and confirmed by histology and intra-operative findings. Other diagnostic investigations included abdominal ultrasound and computerized tomography (CT) scan.

GOO results from either an intrinsic or an extrinsic obstruction of the pyloric channel or duodenum. The underlying disease process dictates the precise mechanism of the obstruction. In benign causes, the obstruction is typically the result of inflammation, edema, or scarring in the region of the pyloric channel or duodenal bulb. With PUD, the obstruction typically results from acute inflammation and edema or chronic fibrosis and scarring of the pyloric channel or duodenal bulb. Similarly, gastroduodenal involvement with Crohn's disease can lead to a Crohn's-related stricture. With pancreatitis, intense peri-pancreatic inflammation and edema can result in inflammation and fibrosis of the adjacent duodenum. In contrast, obstruction secondary to a large gastric polyp, gastric bezoar, gastric volvulus, or migration of a gastrostomy tube represents a discrete mechanical problem [9].

All the patients were subjected to a standard preoperative treatment, which included stomach wash twice a day for three days prior to surgery. Preoperatively stomach was dilated in majority of the cases. Postoperatively Ryle's tube aspiration continued till bowel movements established by noting bowel sounds, passing of flatus and gross reduction in quantity of Ryle's tube aspiration. Later on majority of patients were allowed to take oral fluids on 6th day followed by semisolid and solid diet. The average hospital stay in this series was 10 days. This is almost similar when compared to the series of Ralph A. Matteis and Robert E. Hermann where the average hospital stay was 8.3 days and of Fisher *et al.* where it was 6.8 days [10, 12].

Conclusion

Gastric Outlet Obstruction is the commonest disease with significant morbidity and mortality. Patients with gastric outlet obstruction due to cicatrized duodenal ulcer require truncal vagotomy with posterior gastrojejunostomy. Vagotomy is optional in view of better response with drugs for APD. Antral carcinoma cases require curative or palliative surgery depending on the stage of the disease.

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