Aetiogical Pattern of Adult Gastric Outlet Obstruction

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Abstract:
This prospective cross sectional study was conducted at Dhaka Medical College Hospital between November 2012 to November 2013. A total 150 patients were studied. In 96 cases (64%), malignancy is the cause of GOO and the rest of the cases are due to benign cause (36%). Among the malignant causes, ninety one cases (60.67%) were due to carcinoma stomach. Other malignant causes were carcinoma head of the pancreas (2.67%) and gastric lymphoma (0.67%). The benign causes were chronic duodenal ulcer with pyloric stenosis (34.67%) and corrosive ingestion (1.33%). The gender distribution was predominantly male, making up about 72.66% of the total patient. The mean age was 50.07 years; the range being 21 to 78 years. But the patients with malignancy ended to be older (mean age 55.3 years), whereas the mean age of patients diagnosed with benign causes was 40.74 years. All patients underwent upper GI endoscopy. Endoscopy was diagnostic in 145 cases. Among them, biopsy was needed in 91 cases. Five cases were diagnosed confirmatively on histopathology of operative specimen and these included carcinoma head of the pancreas and gastric lymphoma.

Key Words: Adult Gastric Outlet Obstruction, Gastric malignancy pyloric stenosis

Introduction
Gastric outlet obstruction (GOO) in adults is not a single entity; it is the pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying1. Two most common causes of GOO are gastric cancer and pyloric stenosis secondary to peptic ulceration. Previously the later was more common2. With the advent of proton pump inhibitors and Helicobacter pylori eradication therapy, this benign cause has become less common3. Until 1970s, benign disease was responsible for the majority cases of GOO in adults, while malignancy accounted for only 10 to 39% of cases4,5. By contrast in recent decades 50 to 60 percent cases have been attributable to malignancy4,6. 15 to 25 % of patients with pancreatic cancer present with GOO and they also have biliary obstruction7,8,9. Distal gastric cancer remains a relatively common cause of malignant GOO accounting for up to 35% of GOO8. Other malignant causes include periampullary tumors, lymphoma and metastases to the duodenum or jejunum10,11,12. In recent years the anatomical location of gastric cancer appears to have shifted from the antral portion to a more proximal part of stomach including involvement of gastro-oesophageal junction13.

Among the benign causes are peptic ulcer disease (PUD), caustic ingestion, post-operative anastomotic state and inflammatory causes such as Crohn’s disease and tuberculosis. Less often, chronic pancreatitis, annular pancreas and non-steroidal anti-inflammatory drug-included strictures result in GOO. PUD is the most common cause of benign GOO. After the association between Helicobacter pylori and peptic ulcer was recognized, less than 5% patients with complicated duodenal ulcer disease and less than 1%-2% with complicated gastric ulcer disease

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Among the benign causes are peptic ulcer disease (PUD), caustic ingestion, post-operative anastomotic state and inflammatory causes such as Crohn’s disease and tuberculosis. Less often, chronic pancreatitis, annular pancreas and non-steroidal anti-inflammatory drug-included strictures result in GOO. PUD is the most common cause of benign GOO. After the association between Helicobacter pylori and peptic ulcer was recognized, less than 5% patients with complicated duodenal ulcer disease and less than 1%-2% with complicated gastric ulcer disease
have developed this complication\textsuperscript{14,15}. Patients with ulcer related GOO often have a long history of symptoms\textsuperscript{16}. In a study carried out in the United States, only 14% ulcer-related GOO had acute disease and obstruction was the initial manifestation of the disease\textsuperscript{17}. It has been estimated that > 95% of cases of obstructing duodenal ulcer disease have the obstruction in the duodenal bulb, and the rest were in the post bulbar region\textsuperscript{15}. Caustic ingestion is another important cause, both acid and alkali ingestion can cause antral/pyloric scarring resulting in GOO\textsuperscript{18,19}. About one third of patients with ingestion of strong caustics end up having GOO\textsuperscript{19}.

Patients usually present with intermittent vomiting that progress until obstruction is complete. The vomitus is characteristically unpleasant in nature and is totally lacking in bile. Very often it is possible to recognize foodstuffs taken several days previously. The patient commonly complains of losing weight, and in the acute or chronic phase of obstruction, continuous vomiting may lead to dehydration and electrolyte abnormalities\textsuperscript{20}. It may be possible to see the distended stomach with visible peristalsis and a succussion splash may be audible. Anaemia is found in about 25% of patients\textsuperscript{21}. In malignant cases an epigastric mass, Virchow’s gland, ascites, hepatomegaly may also present. The means of diagnosis mostly contrast radiography and upper gastrointestinal endoscopy with biopsy. Sometimes repeat endoscopy with biopsy and brush cytology may require to established the true nature of disease\textsuperscript{22}.

Materials and Methods

This descriptive cross sectional study were conducted in surgery department of Dhaka Medical College Hospital (DMCH) from November 2012 to November 2013. Patients of both sexes (Age ≥ 18 years) admitted with postprandial vomiting due to mechanical obstruction diagnosed as GOO by upper G.I.T endoscopy were included. Whereas patients who refused to give consent and with inadequate information were excluded. Data was processed and analyzed using computer software SPSS version 17. The test statistics to be used are descriptive statistics, Chi-square (C2) and Student’s t-Test. Level of significance will be set at 0.05.

Results:

The age of patients at presentation ranged from 21-78 years with a mean age of 50.07 years. The mean age of patients with benign causes was 40.74 years (range 21-66 years), while that of malignant causes was 55.3 years (range 29-78 years). The difference in age distribution of the benign and malignant disease was statistically significant (p < 0.001). There were 109 (72.67%) males and 41 (27.33%) females, male to female ratio of 2.69:1. Both the benign and malignant GOO was found to be more common amongst the males. The male to female ratio in benign cases was 2.38: 1, while it was 2.84: 1 for malignant cases. This difference was statistically significant (p < 0.001). 85.33% cases came from low income group, 12.67% from lower middle income group and only 2.00% cases came from upper middle & above.

All the patients had complaints of vomiting. Haematemesis and/or melaena was found in 40% patients. 57.33% patients complained epigastric pain, anorexia 79.33% patients and 94% patients lost significant amount of their previous weight. 61.33% patients presented with epigastric fullness after taking meal and 28% patients presents with epigastric mass. Jaundice was found only 2.67% patients. The duration of problem ranged from 3 weeks to 8 years with a median duration of 1.26 years. The time interval between symptom onset and diagnosis was often more than 6 months (70.00%).

| Age (Years) | Frequency and percentage according to age group | Percentage of total |
|-------------|-----------------------------------------------|---------------------|
| ≤ 20        | 0 (0.00%)                                     | 0                   | 0.00%               |
| 21-30       | 5 (100.00%)                                   | 5                   | 3.33%               |
| 31-40       | 24 (88.89%)                                   | 27                  | 18.00%              |
| 41-50       | 16 (48.48%)                                   | 33                  | 22.00%              |
| 51-60       | 7 (11.67%)                                    | 60                  | 40.00%              |
| 61-70       | 2 (9.09%)                                     | 22                  | 14.67%              |
| ≥ 71        | 0 (0.00%)                                     | 3                   | 2.00%               |
| All age group | 54 (36.00%)                                   | 96 (64.00%)         | 150                 | 100.00%             |
Table-II
Distribution of Patients according to Clinical Presentation (n=150)

| Clinical Presentation          | Frequency | Percentage (%) |
|-------------------------------|-----------|----------------|
| Vomiting                      | 150       | 100.00%        |
| Haematemesis and/or Melaena    | 60        | 40.00%         |
| Epigastric Pain               | 86        | 57.33%         |
| Epigastric Fullness           | 92        | 61.33%         |
| Epigastric Mass               | 42        | 28.00%         |
| Anorexia/ Loss of Appetite    | 119       | 79.33%         |
| Weight Loss                   | 141       | 94.00%         |
| Yellow Coloration of skin and sclera | 4 | 2.67% |

Previous history suggestive of PUD was reported in 53.33% patients. Most of the patients had history of taking at least one form of anti ulcerant and majority of them took irregularly. 57.33% cases took antacid syrup/tablet, 48.67% cases H2 receptor blocker, 60.67% PPI and 22.00% cases took triple therapy.

95.33% patients were anaemic, 5.33% were icteric, 85.33% patients were dehydrated, 18.67% patients had oedema and 12(8%) patients had Virchow’s gland. Abdominal examination revealed visible peristalsis in 54% patients, 28% patients had palpable lump in epigastrium, succussion splash elicited in 71.33% patients. Ascites was found in 32 (21.33%) cases and hepatomegaly in 11 (7.33%) patients.

In 45.33% cases endoscopist failed to negotiate the duodenum. Antral growth was found in 91 (60.67%) cases, deformed duodenal bulb in 34.67% cases. Ulcerative lesions in stomach were found in 27.33% cases; 7.33% in duodenum and in pyloric channel 8.67% cases. Endoscopic findings of mucosa were normal in 5 (3.33%) cases.

Table-III
Causes of Gastric Outlet Obstruction (n=150)

| Cause                                | Number | Percentage (%) |
|--------------------------------------|--------|----------------|
| Malignant Cause                      | 96     | 64.00%         |
| CA Stomach                           | 91     | 60.67%         |
| CA Head of Pancreas                  | 4      | 2.67%          |
| Gastric Lymphoma                     | 1      | 0.67%          |
| Benign Cause                         | 54     | 36.00%         |
| Chronic Duodenal Ulcer with          | 52     | 34.67%         |
| Pyloric Stenosis                     |        |                |
| Corrosive Ingestion                  | 2      | 1.33%          |

In ultrasonography of whole abdomen 8.67% patients had hepatic secondaries, 28.67% patients had ascites, 4 (2.67%) patients had mass in head of the pancreas, antral wall thickening or mass in the antral wall found in 37.33% cases. In 52 (34.67%) cases, no abnormalities were detected. Some incidentals findings like cholecystitis, renal cyst, BEP were also being found in 16 (10.67%) in cases. Computed Tomography of abdomen was done in 54 cases. In 92.59% cases there were antral wall thickening or mass in the antral wall, ascites were found in 53.70% cases, in 42.59% cases there were lymph node deposits, hepatic secondaries in 14.81% cases and in 4 (7.41%) cases there were pancreatic mass.

After all the investigations before operative procedure 96.67% cases were diagnosed confirmatively. Only 5 (3.33%) cases were confirmatively diagnosed via histopathological findings of operative specimens, 4 cases were diagnosed as carcinoma head of the pancreas and 1 case was gastric lymphoma. Out of the 150 cases, 64% were diagnosed as malignant and 36% were benign cause of GOO. Out of malignant cases 60.67% were adenocarcinoma of the stomach, 2.67% cases were carcinoma head of the pancreas and 1(0.67%) was gastric lymphoma. Out of benign cases 34.67% were due to chronic duodenal ulcer with pyloric stenosis and 2(1.33%) cases were due to ingestion of corrosive.

In cross comparison between endoscopy without biopsy and with biopsy towards diagnosis reveals that, 27.33% cases were diagnosed by endoscopy of upper GIT without biopsy and all of them were diagnosed as benign cause. Among the 104 cases where biopsy needed 8.67% were diagnosed as benign and 60.67% were diagnosed as malignant causes. In 5 (2.88%) cases diagnosis remained in a dilemma and it needed the open biopsy.

Fig.-1: Drug History of study population (n=150):
Discussion

Gastric outlet obstruction has traditionally been considered synonymous with pyloric stenosis as a result of peptic ulcer disease in adults, accounting for up to 90% cases in the 1960’s and early 70’s. A 10 years study from 1970-79 showed peptic ulcer to be the etiology amongst 81% of the GOO patients. Now with the decrease incidence of peptic ulceration due to the advent of potent medical treatment, the malignancy is the commonest cause of GOO especially in the western countries.

In this study, out of 150 patients, 64% had malignancy as the cause of obstruction, while 34.67% had benign disease due to chronic duodenal ulcer with pyloric stenosis. Another study from India in 1998 showed malignancy as the cause of GOO in 76% of the patients. Interestingly a study of 64 patients from Nigeria during 1991-96 periods did not show this change in aetiology and reported chronic duodenal ulcer as the commonest cause (66%) of GOO, while carcinoma of the stomach was seen only in 15% cases.

Another study from Nigeria described a similar figure of 14% for gastric carcinoma amongst cases of GOO. This reflects that pyloric stenosis due to chronic duodenal ulcer is still more common in developing countries.

The mean age of the patients was 50.07 years with a peak incidence in 6th decade, which is consistent with another study where 52 patients of GOO were studied and the peak incidence were in 5th and 6th decade of life. Again, in malignant cases, the mean age was 55.3 years which is slightly higher than another study carried out in Bangladesh where mean age of malignant gastric carcinoma was 51.48 years. In this study males are seen to be affected more than female. This signifies and consistent with that the peptic ulcer and gastric cancer is still more common in male.

The present of other confounding factors like the socio economic status of patients admitted in the study hospital are usually from low income group, has made the analysis not statistically significant. But it is proved in other study, that antral carcinoma is more common in lower socioeconomic condition. Chronic duodenal ulcer is also common in this group.

All patients in this series presented with characteristic vomiting which drew attention to the possibility of clinical diagnosis of GOO. Bleeding was present in 40%, 61.33% of the patients complaint of epigastric fullness and 23.33% epigastric mass which indicates advanced malignant lesion.

The average duration of problem was more in benign cases (4.76 years) and less in malignant cases (1.42 years).

In this study 80 patients (53.33%) had past history suggestive of PUD, but all patients had history of taking anti ulcerant. Most of them took these drugs irregularly and not completed a full course. It is documented that effective medical treatment can cause a reduction in the incidence of GOO due to duodenal ulceration. It is clear from the present study that still a great incidence of long term complication of peptic ulcer disease is contributed by incomplete or maltreatment of peptic ulcer.

95.33% patients were clinically anaemic, whereas only 5.33% patients were icteric. 12 malignant patients had Virchow’s gland, visible peristalsis was seen in 54% patients, succussion splash in 71.33% cases, ascitis 38.67% and hepatomegaly 7.33% of cases, which represents the well advanced malignancy and signs of inoperability.

These also indicates negligence and late presentation to surgeons which is a common picture in our poor people.

Endoscopy was done in every case and detected 91 (60.67%) antral growth, 41 (27.33%) suspicious cases of stomach ulcer, 13 (8.67%) pyloric channel ulcer. Histopathology of specimen taken through endoscopic biopsy confirmed 91 cases adenocarcinoma of stomach. It was also seen that some patients have active gastric and duodenal ulcers in addition to the obstructive lesions.

Endoscopy also detect 52 (34.67%) deformed duodenal bulb with narrowing and 11 (7.33%) chronic duodenal ulcer from which biopsy were not taken as it look like a benign stricture or narrowing and malignancy in this region is so uncommon that under normal circumstances surgeons can be confident that they are dealing with benign disease.

Endoscopy with or without biopsy is diagnostic in 96.66% cases, remaining 3.33% cases had normal mucosa with GOO and these cases were diagnosed with the aid of radiological investigations and confirmed by per-operative biopsy. So it can be stated that performing biopsy in all cases of GOO improved the rate of correct preoperative diagnosis and so did the management of the patients.

However, some cases may need open biopsy to confirm the diagnosis.

Conclusion

The principle aim of this study is to find out the cause of GOO in the era of advanced medical treatment. The relative incidence of GOO due to malignancy is more than that of chronic duodenal ulcer with pyloric stenosis is observed in this series. On the other hand malignant cases present in advanced stage due to lack of proper diagnostic facilities and referral system especially in low socioeconomic groups and as there no national screening program to detect early gastric carcinoma.
Limitations:
Study was conducted in a single hospital and with small sample size.

Recommendations:
The present study evidently shows that the incidence of GOO due to malignancy is notably increased. As early diagnosis in any form of a malignancy is essential to improve survival rate and to prevent complications, screening programme for gastric cancer should be started. It is also clear that still a significant incidence of long term complication of PUD is contributed by incomplete or mal-treatment of peptic ulcer, so complete medical treatment and follow up of PUD is necessary.

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