Is prolapse gastropathy a cause of upper gastrointestinal bleeding?

Prolaps gastropati üst gastrointestinal sistem kanama nedeni midir?

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Background!aims: Prolapse gastropathy is a clinical syndrome involving the invagination of a part of the gastric mucosa into the lower esophagus resulting in well demarcated hemorrhagic mucosa and sometimes bleeding. The importance of this syndrome is that it has been reported as a cause of hematemesis. As this syndrome is considered to be seen in patients with retching and vomiting and to be one of the causes of upper gastrointestinal bleeding, this study was undertaken to evaluate its frequency and importance in our patients. Methods: The study groups comprised of 941 patients with hematemesis and 54 with recurrent retching and vomiting. Biopsies were obtained from the prolapsing mucosa when detected. Hematoxylin and eosin stained specimens were reviewed by an experienced histopathologist. **Results:** Prolapse gastropathy was detected in 20 patients (2%), of whom six (0.6%) were in the hematemesis group and 14 (25.9%) in the retching and vomiting group. The location of the lesion was the greater curvature in 10 patients (50%), lesser curvature in seven patients (35%) and anterior wall in three patients (15%). In all six cases from the hematemesis group, bleeding was not related to prolapse gastropathy because other causative lesions were identified. Histologic evaluation of the biopsies revealed acute inflammation, chronic inflammation and submucosal hemorrhage. Conclusions: According to the results of this study, prolapse gastropathy is not a cause of hematemesis. Other causes should be identified when prolapse gastropathy is detected in a patient with hematemesis.

Key words: Prolapse gastropathy, gastrointestinal bleeding, retching, vomiting

Amaç: Prolaps gastropati gastrik mukozanın bir kısmının distal özofagus içine prolabe olması sonucu sınırları belirgin hemorajik mukoza ve bazen de kanama ile seyreden klinik sendromdur. Bu sendromun önemi hematemez nedeni olarak rapor edilmiş olmasıdır. Prolaps gastropatinin öğürme-kusma yakınmaları olan hastalarda görülmesi ve üst gastrointestinal sistem kanama nedeni olarak tanımlanması nedeniyle hastalarımızda bu sendromun sıklığını ve önemini araştırdık. Yöntem: Çalışmaya hematemez yakınması olan 941 hasta ile tekrarlayan öğürme-kusma yakınması olan 54 hasta alındı. Endoskopi sırasında prolaps gastropati tespit edilen vakalardan biyopsiler alındı. Preparatlar hematoksilen eosin ile boyanarak incelendi. Bulgular: Hematemez grubunda 6 (%0.6) hasta, öğürme-kusma yakınmaları olan grupta 14 (%25.9) hasta olmak üzere toplam 20 (%2) hastada prolaps gastropati saptandı. Lezyonlar 10 (%50) hastada büyük kurvatur yönünde, 7(%35) hastada küçük kurvatur yönünde, 3(%15) hastada ön duvar yönünde yerleşmişti. Hematemez grubundaki 6 hastada prolaps gastropatiyi primer kanama nedeni olarak düşünmedik, çünkü bu hastaların hepsinde kanamayı izah edecek başka patolojiler mevcuttu. Biyopsilerin histolojik incelenmesinde akut inflamasyon, kronik inflamasyon ve submukozal kanama bulguları mevcuttu. Sonuç: Bizim çalışmamızda prolaps gastropatinin hematemez nedeni olmadığı görülmektedir. Hematemez mevcut olan bir hastada prolaps gastropati görülse bile diğer kanama nedenleri araştırılmalıdır.

Anahtar kelimeler: Prolaps gastropati, gastrointestinal kanama, bulantı, kusma

INTRODUCTION

Prolapse gastropathy (PG) is a clinical syndrome involving the invagination of a part of the gastric mucosa into the lower esophagus, resulting in retrosternal pain, hemorrhagic mucosa, and sometimes bleeding. According to Miller, gastroesophageal prolapse was first described by Enderlen in 1903, in an autopsy (1). Since then, many case reports defining similar lesions have been published (2-4). In 1984, Shepherd described the syndrome as a result of recurrent retching and named it as prolapse gastropathy (5). The etiology is suggested to be related to ethanol ingestion (6, 7), nonsteroidal anti-inflammatory drug use (8, 9), gastroenteritis, hyperemesis gravidarum, uremia, malignancy, and duodenal ulcer (9).

The importance of this syndrome is that it has been reported as a cause of upper gastrointestinal bleeding in adults (7-9), especially in children (10, 11). It can also be a cause of chronic hemorrhage in patients with hiatal herniation and recurrent prolapses may be the cause of gastric or esophageal tears. Recently, it has been defined in a gastroenterology atlas (12) and has been described in a major textbook as a cause of mild upper gastrointestinal hemorrhage often seen in alcoholic patients (13). As this syndrome is considered to be seen in patients with retching and vomiting and to be one of the causes of upper gastrointestinal bleeding, this study was undertaken to evaluate its frequency and importance in our patients undergoing endoscopy for hematemesis and retching, vomiting.

MATERIALS AND METHODS

Upper gastrointestinal endoscopy was performed on 5054 patients between October 2000 and January 2002 (15 months). The incidence of PG was calculated in patients with hematemesis and those with retching and vomiting. Nine hundred and forty one patients had been admitted with hematemesis, while 54 had recurrent retching and vomiting.

A careful history of alcohol consumption, drug intake, gastrointestinal diseases, other systemic diseases, pregnancy, malignancy and full blood count were recorded in patients with prolapse gastropathy syndrome.

Endoscopy was performed using either the Olympus GIF 240 or Pentax 2800 T endoscopes after routine topical lidocaine spray to the pharynx and sedation with midazolam when needed (increments of 2.5 mg IV).

Biopsies were obtained from the prolapsing mucosa. Hematoxylin and eosin stained specimens were reviewed by an experienced histopathologist who was unaware of the endoscopic findings.

RESULTS

Upper gastrointestinal endoscopy was performed on 5054 patients with various upper gastrointestinal symptoms between October 2000 and January 2002. Nine hundred and forty one patients (18.6%) had complaints of hematemesis while 54 patients (1.1%) had retching and vomiting.

Prolapse gastropathy was detected in 20 patients. This group was 0.4% of all endoscopies performed and 2% of the study groups (995 patients) which consisted of 18 males (90%) and two females (10%), aged 15-69 years (mean 33 years). There were six patients (0.6%) in the hematemesis group and 14 patients (25.9%) in the retching and vomiting group. The characteristics of all of these patients are shown in Table 1-2.

No	Age	Gender	NSAID	Localization and	Other endoscopic findings	Other	Transfusion
			ETOH	size of PG		medical	
						conditions	
1	33	М	None	GC-3 cm	Esophagitis	None	No
2	34	М	None	GC-2 cm	None	None	No
3	35	М	ETOH	LC- 2 cm	Antral gastritis	None	No
4	39	М	None	GC-2-3 cm	None	None	No
5	22	М	None	GC-3 cm	Esophagitis	Malignancy	No
6	25	М	None	GC- 4-5 cm	Hiatal hernia	None	No
7	15	F	None	ASW- 3 cm	None	None	No
8	69	М	None	GC-3 cm	Duodenitis	Subileus	No
9	29	М	None	ASW-3 cm	Duodenal ulcer scar	None	No
10	37	F	None	LC-2-3 cm	Esophagitis, Hiatal hernia	None	No
11	26	М	None	GC-1-2 cm	None	None	No
12	25	М	ETOH	LC-2-3 cm	Antralgastritis	None	No
13	25	М	None	LC- 1-2 cm	Duodenitis, antral gastritis	None	No
14	33	М	None	GC-2 cm	Antral gastritis	None	No

Table 1. Characteristics of prolapse gastropathy patients in retching and vomiting group

NSAID: Nonsteroidal anti-inflamatory drug ; ETOHialcohol; PG: Prolapse gastropathy; GC: Greater curve; LC:Lesser curve; ASW: Anterior stomach wall.

					e 1		
No	Age	Gender	NSAID	Localization and	Other endoscopic	Other	Transfusion
			ETOH	size of PG	findings	medical	
						conditions	
1	37	М	NSAID	LC-3 cm	Esophagitis, Erosive gastritis	None	No
2	22	М	None	ASW-3 cm	Duodenal ulcer (Fill)	None	4 unit
3	37	М	None	LC-2cm	Duodenal ulcer (FIIC)	None	1 unit
4	33	М	None	LC-2 cm	Duodenal ulcer	None	No
5	42	М	None	GC-3 cm	Duodenal ulcer, hiatal hernia	None	No
6	40	М	None	GC-2-3 cm	Duodenal ulcer	None	No

Table 2. Characteristics of prolapse gastropathy patients in hematemesis group

Abbreviations: NSAID: Nonsteroidal anti-inflamatory drug; ETOH: alcohol; PG: Prolapse gastropathy; LC: Lesser curve; ASW: Anterior stomach wall; GC: Greater curve; F: Forrest classification of bleeding ulcers.

One patient in the study group had a history of non-steroidal anti-inflammatory drug (NSAID) intake and two patients alcohol intake. None of the female patients were pregnant. One patient was undergoing chemotherapy due to renal cell carcinoma and one other had findings of subileus. Two patients received blood transfusions due to low blood count levels.

During endoscopy, a tense knuckle of gastric mucosa forcefully and repeatedly prolapsed into the distal esophageal lumen was seen in some patients (Figure 1). The mucosal knuckle was inflamed and covered with multiple petechial hemorrhage. In all patients well-defined areas of mucosal hemorrhage (1-5 cm diameter) surrounded by normal mucosa were detected (Figure 2-3). The location of the lesions were: the greater curvature in 10 patients (50%), lesser curvature in seven patients (35%) and anterior wall in three patients (15%).

Histologic evaluation of the biopsies obtained from the lesions revealed acute inflammation, chronic inflammation and submucosal hemorrhage (Figure 4). There was no ulceration.



Figure 1. Gastric mucosal prolapse into the esophagus viewed through the endoscope



Figure 2. Congested, hemorrhagic mucosa at cardia secondary to prolapse gastropathy



Figure 3. Close view of the hemorrhagic mucosa



Figure 4. Biopsy specimen showing superficial mucosal hemorrhage (H&E; X 100).

DISCUSSION

Imagination of a part of the stomach into the esophagus has been described in autopsies since the beginning of the 19th century. Case reports on this subject have described the condition as "herniation of gastric mucosa into the esophagus" (2), "mucosal prolapse at the esophagogastric junction" (3) and "retrograde prolapse of gastric mucosa into the esophagus" (4). Young et al, identifying the condition as inflamed gastric mucosa below the esophagogastric junction, reported it to be a cause of hematemesis (6). Thomas and Khatak reported a case of hematemesis after alcohol ingestion, who had previously undergone multiple endoscopies where a similar lesion was identified as gastritis and they pointed out that the lesion may sometimes be confused with gastritis (7). A case of recurrent hematemesis due to mucosal prolapse in association with hiatus hernia has been reported by Laforret (8).

In 1984 Shephard, proposing the term "prolapse gastropathy syndrome", reported 22 patients who presented with epigastric pain alone or with hematemesis associated with a previous history of recurrent early morning retching or postprandial retching (5). He defined the characteristic endoscopic finding in these patients as a knuckle of inflamed and sometimes bleeding gastric mucosa which repeatedly prolapses into the esophagus lumen during retching. In 1999, Byfield reported seven cases with similar findings (9).

Endoscopically there is a localized patch of hyperemic, congested, bright red mucosa varying in size from 1 to 6 cm in diameter. The lesion is found in the proximal stomach several centimeters distal to the gastroesophageal junction, frequently on the lesser curve. Active oozing of blood may be seen along with coffeee ground material in the stomach (4).

It has been suggested that the force of the repeated retching causes gastric mucosal trauma. The prolapsing gastric mucosa becomes edematous and inflamed, and a localized lesion in the knuckle is caused by a pressure effect on the mucosa and mucosal capilleries while it is being jammed into the esophagus lumen. Gastric irritability caused by this inflammation and from the retching itself probably perpetuates the syndrome.

The lesions detected have commonly been located in the greater curve (46%), lesser curve (43%), anterior wall (7%) and sometimes posterior wall (5,6). In the present study the location of the lesions was the greater curve in 10 patients (50 %), lesser curve in seven patients (35 %) and anterior wall in three patients (15 %).

A history of NSAID intake and alcohol ingestion has been reported at a rate of 46% in patients with PG but in this study, it was only 15%. Other causes such as gastroenteritis, hyperemesis gravidarum, uremia, malignancy, and duodenal ulcer have been reported (9). In the present study, it was found that chemotherapeutic medication and intestinal obstruction can also cause PG. The incidence of PG has been reported as between 2% and 5.5% (5). Shephard found PG in 22 (2.4 %) of 914 upper gastrointestinal endoscopies, but it was found in only 20 (0.4 %) of 5054 endoscopic examinations in the present study.

Prolapse gastropathy has been reported in almost 2% of adults who undergo upper endoscopy for the evaluation of hematemesis and Bishop described it as a cause of hematemesis in seven of 27 pediatric patients (25.9%). We detected six cases (0.6%) among 914 patients on whom endoscopy was performed for evaluation hematemesis, but in none of these cases was hematemesis related to PG, because other lesions were identified as the cause. Our findings correlate with the results of large series evaluating 3294 and 2225 cases for upper gastrointestinal bleeding, which failed to mention PG as a cause of bleeding (14, 15).

Prolapse gastropathy is detected mostly in patients with retching and vomiting and when this group is considered, the incidence was 25.9%,

with none of the patients having upper gastrointestinal bleeding.

Evaluation of other studies suggests that PG is mostly found in male patients at a rate of 87% (5, 9-11). Our results were similar, with a male predominace of 90%.

The need for blood transfusion has been reported in only five patients with PG (6-8). None of the 14 patients in our retching and vomiting group required blood transfusion, while two out of six patients in the hematemesis group required blood transfusion, but both of these patients had duodenal ulcers, which led to the need for transfusion.

In conclusion although PG has been reported as a cause of hematemesis, in this study it was found that it was not a cause of hematemesis when patients with hematemesis accompanied by retching and vomiting were evaluated. The present authors suggest that when PG is detected in a patient with hematemesis, there should be evaluation for other causes.

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