Congestive Gastropathy And Other Upper Endoscopic Findings in 250 Portal Hypertensive Patients

PORTAL HİPER TA NSİYONLU 250 HASTADA KONJESTİF GAS TROPA Tİ VE DİĞER ÜST GASTROINTESTINAL ENDOSKOPIE BULGULAR

Sadakat ÖZDİL, M.D., Fatih **BEŞIŞIK,** M.D, Güngör **BOZTAŞ, M.D.*, Zeynel** MUNGAN, **M.D.*, Oktay YEĞİNSÜ, M.D.", Yılmaz ÇAKALOĞLU, M.D.*, Sabahattin KAYMAKOĞLU, M.D., Prof.Atilla ÖKTEN, M.D.",** Prof.Nurten **EROL, M.D",** Prof.**Süleyman YALÇIN, M.D.**, Prof.Hauf SEZER,** M.**D."**

* **Associate Professor of Gastroenterohepatoiogy**
** **Professor of Gastroenterohepatoiogy**
Istanbul Medical Faculty, Department of Gastroenterohepatoiogy, İSTANBUL

SUMMARY

Upper endoscopic finginds in 200 cirrhotic (142 males mean age: 46,5+13.4 years, 58 females, mean age: 45.1+15.3 years) and 50 non cirrhotic (20 males, mean age: 29.1 + 13.4 years; 30 females, mean age: 39.4+13.5 years) portal hypertensive patients were evaluated to determine the spectrum of abnormalities encountered, with particular attention to mucosal changes consistent with congestive gastropathy. 190 of 200 (95%) cirrhotics, 44 of 50 (88%) non-cirrhotics had findings compatible with esophageal varices. The prevalance of gastric varices was 25.5% and 48% in cirrhotic and non-cirrhotic patients, respectively (p<0.01). Congestive gastropathy was the second most common abnormality (76%) identified after esophageal varices, in both groups.

Key Words: Portal hypertension, Upper gastrointestinal endoscopy

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ÖZET

*200 sirozlu (142 erkek, **ortalama yaş:** 46.5±13.4 yıl; 58 kadın, ortalama yaş: 45.1 ±15.3 yıl) ve 50 non-sirotik (20 erkek, ortalama yaş: 29.1x13.4 yıl; 30 kadın, ortalama yaş: 39.4+13.5 yıl) portal hipertansiyonlu hastada, özellikle, konjestif gastropati başta olmak üzere üst gastrointestinal endoskopik bulgular araştırıldı. Sirozlu 200 hastanın 190'ında (%95), non-sirotik hastaların ise 44'ünde (%88) özofagus varisleri tespit edildi. Mide varisleri prevalansı ise sirozlu hastalarda %25.5, non-sirotiklerde %48 idi (p<0.01). Her iki grupta da özofagus varislerinden sonra en sık karşılaşılan ikinci anomali konjestif gastopati idi (%76).*

Anahtar Kelimeler: Portal hipertansiyon, Üst gastrointestinal endoskopi

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Portal hypertension may give rise to different upper gastrointestinal complications, in addition to gastroesophageal varices. There is increasing clinical and experimental evidence indicating that the portal hypertensive (PHT) gastric mucosa is morphologically and functionally different from the normotensive mucosa and more susceptible to endogenous and exogenous detrimental agents (1). Congestive gastropathy, the pathogenesis of which thought to involve venous congestion with gastric mucosal capillary dilatation is a frequent finding in these subjects.

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Correspondence: Rauf SEZER, M.D.
Professor of Medicine, Chairman
Department of Gastroenterohepatoiogy
Istanbul Medical Faculty, ISTANBUL

We, therefore, have evaluated endoscopic findings of 250 consecutive portal hypertensive patients to determine the spectrum of abnormalities encountered, with particular attention to mucosal changes consistent with congestive gastropathy.

MATERIALS AND METHODS

The endoscopic reports of 200 cirrhotic (142 males, mean age: 46.5+13.4 years: 58 females, mean age: 45.1+15.3 years) and 50 non-cirrhotic (20 males, mean age: 29.1 ±13.4 years; 30 females, mean age: 39.4+13.5 years) patients who had been evaluated in our endoscopy unit as a part of their routine examination, between 1988 and 1990 were reviewed. Esophageal varices were graded by extent into the lumen. Grade I was less than 1 mm, grade II was up to 2 mm, grade II was up to 3 mm, and grade IV was over 3 mm (2). In addition, distribution, color and red color

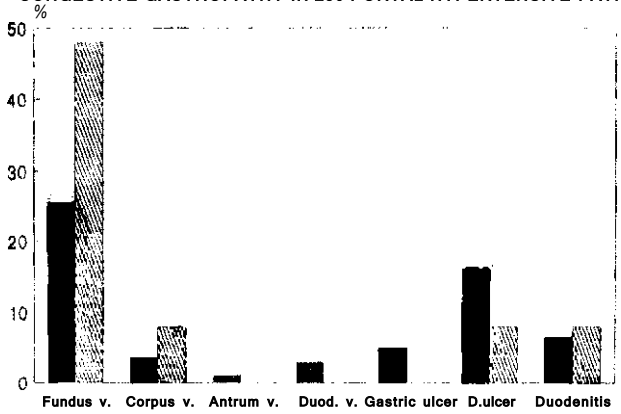

Figure 1. The Prevalence of esophageal varices in portal hypertensive patients (Dark bars: Cirrhotics, Hatched bars: Non-cirrhotics).

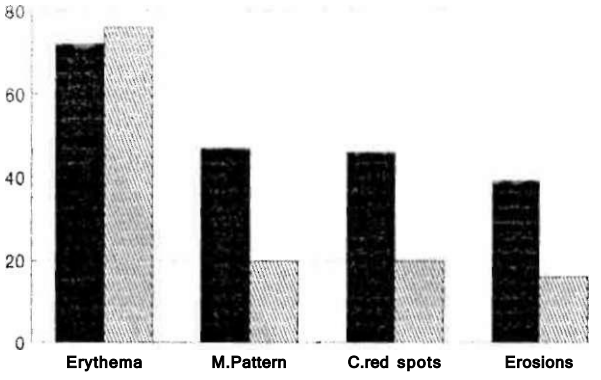

Figure 2. The prevalence ot findings suggestive of congestive gastropathy (Dark bars: Cirrhotics, Hatched bars:Non-cirrhotics).

signs (the red wale sign, small cherry red dots, a hematocystic spot, a diffuse redness) of esophageal varices, gastric and duodenal varices were also noted. Specific attention was directed at findings consistent with portal hypertensive gastropathy. These included petechiae, focal or diffuse intense erythema with or without erosions, edematous mucosa with a fine white reticulated or mosaic pattern, and cherry red spots resembling vascular malformations.

For endoscopic evaluation, Olympus GIF 1T10 and Olympus GIF XQ20 end-viewing endoscopes were used with the patient in left lateral position.

Statistical analysis was conducted using chi-square test.

RESULTS

190 of 200 (Grade I: 26 (13%), Grade II: 46 (23%), Grade III: 56 (28%), Grade IV: 62 (31%), total: 95 %) cirrhotics, 44 of 50 (Grade I: 11(22%), Grade 11:11 (22%), Grade III: 9 (18%), Grade IV: 13 (26%), total: 88%) non-cirrhotics had findings compatible with esophageal varices (Figure 1),

The prevalance of gastric varices was 25.5% and 40% in cirrhotic and non-cirrhotc patients, respectively. The difference between these two groups were statistically significant ($p<0.01$).

Congestive gstropathy was the second most common abnormalitiy (76%) identified after esophageal varices, in both groups. Findings suggestive of congestive gastropathy in cirrhotics included erythema (76%), erosions (16%), mosaic pattern (20%), cherry red spots (20%). Erythema in 76%, erosions in 16%, mosaic pattern in 20%, cherry-red spots in 20% of non-cirrhotic patients were detected (Figure 2). These signs of congestive gastropathy were significantly more prevalent in cirrhotics when compared with non-cirrhotic patients ($p<0.01$).

Gastric ulcer was identified in 10 (%5), duodenal ulcer in 32 (16%) of cirrhotic patients. These figures in

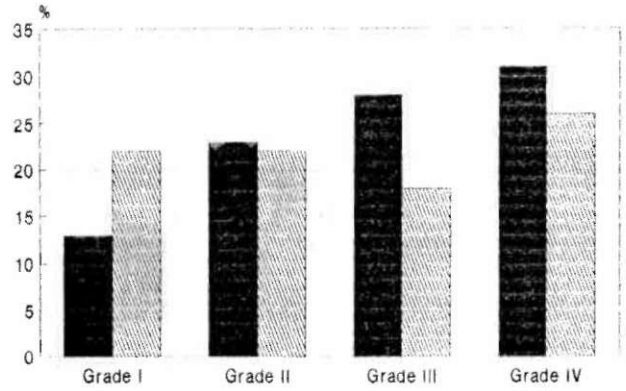

Figure 3. Miscellaneous findings observed during endoscopic examination (Dark bars: Cirrhotics, Hatched bars: Non-cirrhotics).

noncirrhotics were 0 and 4 (8%), respectively (Figure 3). The difference was not statistically significant ($p>0.05$).

DISCUSSION

Endoscopy is the best method of visualizing a wide range of upper gastrointestinal abnormalities encountered in portal hypertensive patients.

Portal hypertension, defined as an increase in portal vein pressure above the normal range of 5 to 10 milimeters of mercury, is characterized by remarkable collateral circulation that carry portal blood into the systemic veins (3-5). The collaterals that lead to the greatest clinical problems lie within the mucosae of the stomach and esophagus. When dilated, they form gastric and esophageal varices and confirm the presence of portal hypertension. Patients with cirrhosis and esophagogastric varices have a 25% to 33% risk of initial varieceal bleeding, and an associated mortalitiy of up to 50% (3,4). Large size, tortuosity, diffuse redness, hematocystic spots, proximal extension, and presence of esophagitis are associated with high prob-

ability of bleeding (2), This means that endoscopic evaluation is also necessary for prognostic assessment and therapeutic decision. In our study, 95% of cirrhotics and 88% of non-cirrhotics had findings compatible with esophageal varices. These results are concordant with other investigators' (3,5). The prevalence of gastric varices was 25.5% and 48% in cirrhotics and non-cirrhotics, respectively. The difference between these two groups were statistically significant ($p<0.01$). Portal or splenic vein thrombosis was the antecedent of portal hypertension in 32% of our non-cirrhotic population. In this group of patients, gastric varices are usually more prominent as confirmed in our study (6,7).

Focal or diffuse erythema was included among those observations thought to be suggestive of congestive gastropathy, because it has been previously similarly described (8,9). It should be noted that half the patients in whom these were observed had other supporting findings of congestive gastropathy (Figure 2). These signs of congestive gastropathy were significantly more prevalent in cirrhotics when compared with non-cirrhotic patients. That may be related to endotoxamia found in many patients with cirrhosis of the liver. A decrease in phagocytic function of the liver, disturbance of venous outflow due to portal hypertension and formation of portosystemic shunts result in diffusion of endotoxin through the gut wall. A close correlation between endotoxemia and erosive changes of gastric mucosa was reported in cirrhotic patients (10).

Biopsies in these patients might have been useful to support the diagnoses, particularly since characteristic abnormalities have been described frequently. Albeit, routine biopsy was not a feature of this retrospective study, It should be included in ongoing prospective investigation of this disorder.

Portal hypertensive gastroduodenal mucosa is morphologically and functionally different from the normotensive gastroduodenal mucosa. The portal hypertensive gastric mucosa has reduced luminal acidity and potential difference, extensive submucosal edema, distinctive gross appearence (endoscopically) and increased susceptibility to severe damage by noxious agents such as aspirin, alcohol and bile acids (1,11,12). In our study, gastric ulcer was identified in 5%, duodenal ulcer in 16% of cirrhotic patients. In literature, these figures range between 10-15%. This pervalance is five times more than the normal population (5,13).

There is certain shortcomings of this study such as the subjective descriptions made by different endoscopists, the lack of histologic correlation, and insufficiency of routine follow-up. We have commenced a prospective study free of these limitations. The results of that study may permit a more objective means to evaluate the abnormalities observed in portal hypertensive patients.

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