Portal hypertensive gastropathy and gastric varices before esophageal variceal sclerotherapy and after obliteration

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Objectives: To evaluate the frequency and clinical importance of portal hypertensive gastropathy (PHG) and gastric varices (GV) before endoscopic sclerotherapy (EST) and after esophageal variceal obliteration. Methods: Patients with portal hypertension (PHT) with variceal bleed were prospectively evaluated for PHG and GV before EST with intravariceal injection of absolute alcohol and after esophageal variceal obliteration. Gastric varices and PHG were characterized and graded according to previously established criteria. Patients were followed up for 12-48 (mean 37) months after variceal obliteration. Results: Of 70 patients with PHT 26 had PHG before (severe in two) [18/37 in cirrhosis, 6/20 in non-cirrhotic portal fibrosis (NCPF), and 2/13 in extrahepatic portal vein obstruction (EHPVO)] and 50 had GV after variceal obliteration (severe in 22) (27/37 in cirrhosis, p<0.03 before versus after esophageal variceal obliteration; 16/20 in NCPF, p<0.01; and 7/13 in EHPVO, p=ns). Type I GV (continuation of esophageal varix into the stomach) was found in 25/70 before and 5/70 after esophageal variceal obliteration (p<0.001); in contrast, other types of GV were seen in 14/70 before and 25/70 after (p<0.01). Overt bleeding from GV and PHG during follow-up after variceal obliteration occurred in 6 and 4 patients, respectively. Conclusions: Esophageal variceal obliteration by EST increases the frequency of PHG and GV (except type I GV which get obliterated); both PHG and GV have potential to cause rebleeding. [Indian J Gastroenterol 1998; 17: 10-12]

Key words: Portal hypertension

Endoscopic sclerotherapy (EST) is a well established method for controlling variceal bleeding in patients with portal hypertension (PHT); however, EST is associated with several complications.1 Mucosal changes of PHT, termed portal hypertensive gastropathy (PHG), have been reported to increase following EST in some studies;2,3 the results of other studies are controversial.4,5 Most workers studied patients with cirrhosis who are likely to have progressive deterioration of liver functions over long follow-up period; such deterioration may influence the development of PHG.6

Authors who found that PHG increased following EST suggested that esophageal variceal obliteration increased collateral blood flow through the stomach; if this is true then gastric varices (GV) should also increase following EST. Studies on this issue show conflicting results.7,8,9

We studied cirrhotic and non-cirrhotic patients (to obviate the role of deteriorating liver function in causing PHG) with esophageal variceal bleeding before EST and following esophageal variceal obliteration to determine (a) the role of EST in causing PHG, (b) the frequency and types of GV before EST and after esophageal variceal obliteration, and (c) the frequency of bleeding from GV and PHG after esophageal variceal obliteration.

Methods

Seventy consecutive patients with variceal bleeding due to PHT of various etiologies attending our tertiary care center during a three year period (September 1992 to September 1995) were prospectively studied before starting EST and after esophageal variceal obliteration. The cause of PHT was established by obtaining specific history and biochemical liver profile, ultrasonography, hepatitis virus serology, and liver biopsy. Twenty six other patients were excluded from the final analysis because of not undergoing liver biopsy (10), irregular follow-up (6), presence of other potential source of gastrointestinal bleeding, i.e., peptic ulcer (7) and presence of isolated GV on initial endoscopy requiring surgical therapy (3).

Endoscopy protocol

Sclerotherapy was performed weekly for the initial three weeks, and subsequently fortnightly till variceal eradication, with ethyl alcohol (Bengal Chemical, Calcutta; 100% for adults, diluted to 50% in children) using a forward-viewing fiberscope (GIF Q10; Olympus, Japan) and injector needle (NM 1K; Olympus); 0.5-1.5 mL of alcohol was injected intravariceally at each site. No patient was treated with beta blocker or nitrates during the study period.

Patients were followed up during EST and after variceal eradication for a total period of 12 to 48 (mean 37) months. Surveillance endoscopy was performed after variceal eradication every three months during the first year and subsequently every six months; endoscopy was also performed whenever patients presented with upper gastrointestinal bleed. Demonstration of active bleeding from a source at endoscopy was considered as definite proof of the lesion being the cause of the bleeding; in the absence of such proof demonstration of a single possible source at endoscopy was considered as a likely cause of bleeding.

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Evaluation for PHG and GV

During each session of EST the gastric mucosa was carefully inspected including a retroverted view for evaluation of the fundus. The presence, distribution and types of PHG and GV were recorded. PHG was characterized and graded as described previously. Briefly, scarlatinina rash, mosaic pattern and stripe pattern were classified as mild PHG and cherry-red spots and diffuse hemorrhagic pattern were considered as severe PHG. GV were classified as described by Hosking et al.

Statistical analysis

The differences between proportions in the different groups were analyzed using the χ² test, with Yates' correction where applicable.

Results

The demography of the patients is shown in Table 1.

On initial endoscopy 30 (75%), 10 (50%) and 8 (66%) cases had grade III to IV esophageal varices among patients with cirrhosis, NCPP and EHPVO, respectively; the other patients had grade II varices. The esophageal varices were eradicated during 5 sessions (range 1-10).

PHG and GV before and after EST

The frequency of PHG increased following esophageal variceal obliteration in patients with cirrhosis and NCPP (Table 2). Disappearance of PHG was not noted in any patient after variceal obliteration by EST. Severe PHG was detected in two patients before and in 22 patients after EST (p=0.01).

Type I GV (continuation of esophageal varix into the stomach) was found in 25/70 before and 57/70 after EST (p<0.001). Type II GV was detected in 14 patients before and 28 patients after variceal obliteration (p<0.01) (Table 2). Type III GV appeared in one patient with cirrhosis after esophageal variceal obliteration.

Rebleeding

Sixteen patients (22.8%) had rebleeding during follow-up after esophageal variceal obliteration. The causes of rebleeding included recurrent esophageal varices (6 patients), GV (8) and PHG (4); in only 3/6 patients with GV was active bleeding documented at the time of endoscopy.

Discussion

In this study PHG increased in frequency and severity after esophageal variceal eradication by EST; type I GV obliterated whereas other types of GV increased. Both these lesions have the potential to cause rebleeding in the long run after esophageal varices are obliterated. EST has replaced surgically created portasystemic shunts as the primary modality of therapy for management of esophageal variceal bleeding. As shunts reduced the pressure in the portal circulation, later development of GV and PHG was not important; in contrast, esophageal variceal obliteration by sclerotherapy diverts the blood flow from esophageal varices to other collaterals including those through the stomach, causing development of GV and PHG.

Most previous workers reported that PHG increases following EST; however, others have shown PHG to regress in some patients. We found it to increase in frequency and severity in cirrhosis and NCPP, with a trend towards increase in patients with EHPVO. Increase in frequency and severity of PHG after esophageal variceal obliteration by sclerotherapy in EHPVO has been reported by us earlier. This increase in PHG non-cirrhotic patients could be attributed to EST, since deterioration of liver function which could influence the development of PHG is obviated in this group of patients.

Type I GV, which are in continuation with esophageal varices were obliterated by sclerotherapy for esophageal varices; this can be explained by the possible flow of sclerosant into the GV. A flow of sclerosant into peri-esophageal collaterals through perforator has been shown by endoscopic ultrasonography. The other types of GV, which are not in continuation with the esophageal varices, increased after esophageal variceal obliteration. The presence of gastroesophageal venous anastomoses in the normal esophagus leads us to believe that sclerotherapy for esophageal varices with consequent blockage of the esophageal variceal collaterals might increase the upstream pressure and therefore open up gastric veins and capillaries.

Table 2: Incidence of PHG and GV before and after EST

<table>
<thead>
<tr>
<th></th>
<th>Pre-EST</th>
<th>Post-EST</th>
<th>Pre-EST</th>
<th>Post-EST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cirrhosis (n=37)</td>
<td>18 (1)</td>
<td>27* (8)</td>
<td>6</td>
<td>13</td>
</tr>
<tr>
<td>NCPP (n=20)</td>
<td>6 (1)</td>
<td>16** (11)</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>EHPVO (n=13)</td>
<td>2 (0)</td>
<td>7 (3)</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Total (n=72)</td>
<td>26 (2)</td>
<td>50** (22**</td>
<td>14</td>
<td>29**</td>
</tr>
</tbody>
</table>

Figures within parenthesis denote number of patients with severe PHG p<0.003, **<0.01, *=<0.01 as compared to corresponding pre-EST values. D: disappeared after EST; NA: newly appeared after EST.

Table 1: Demographic and clinical parameters

<table>
<thead>
<tr>
<th></th>
<th>Cirrhosis</th>
<th>NCPP</th>
<th>EHPVO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51 (18)</td>
<td>22 (13)</td>
<td>13 (8)</td>
</tr>
<tr>
<td>M/F</td>
<td>22:15</td>
<td>16:4</td>
<td>7:6</td>
</tr>
<tr>
<td>Alcoholic</td>
<td>6</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>HBsAg+ve</td>
<td>12</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Child’s class A/B/C</td>
<td>25/10/2</td>
<td>18/2/0</td>
<td>13/0/0</td>
</tr>
</tbody>
</table>
Our study shows that both PHG and GV have the potential to cause rebleeding in the long run.

Patients with NCPF and EHPVO are expected to have longer life expectancy than those with cirrhosis; therefore, the strategy for the management of these patients needs to be re-evaluated and further studies need to be undertaken comparing the results of shunt surgery and sclerotherapy in the light of the results of our study.

References

Correspondence to: Prof Guha Mazumder
Received March 25, 1997. Received in final revised form October 7, 1997. Accepted October 14, 1997

12 Indian Journal of Gastroenterology 1998 Vol 17