Vagal dysfunction following endoscopic variceal sclerotherapy

FERSOSH P MISTRY, D SREENIVASA, NITIN M NARAWANE, PHILIP ABRAHAM, SHOBNA J BHATIA

Department of Gastroenterology, K E M Hospital, Mumbai 400 012

Background: Sclerotherapy is associated with complications which involve adjacent structures like the pleura. The effect of sclerotherapy on function of the vagus nerve, which lies in close proximity to the thoracic esophagus, is not clear. Aim: To study gastric acid secretion as a marker of vagal function in portal hypertensive patients who have undergone sclerotherapy. Methods: Portal hypertensive patients who had undergone at least three sessions of sclerotherapy were evaluated by mapping gastric acid-secreting mucosa by the Congo red test and by estimating gastric acid secretion using the modified sham feeding test. Patients with portal hypertension who had never been subjected to endoscopic sclerotherapy were recruited as controls. Results: On Congo red test, complete or substantial reduction in acid-secreting mucosa was observed in eight patients in comparison to none of the controls. Significantly lower acid secretion on modified sham feeding test was observed in those eight patients. Conclusion: A lower gastric acid secretion, probably secondary to vagal dysfunction, is seen in patients who have undergone multiple sessions of sclerotherapy; vagus nerve involvement may be secondary to peri-esophageal inflammation. [Indian J Gastroenterol 1998; 17: 22-23]

Key words: Congo red test, gastric acid secretion, modified sham feed

The vagus nerve is situated in close proximity to the thoracic esophagus. Apart from control of gastrointestinal motility, this nerve plays a role in basal and meal-stimulated gastric acid secretion; the latter accounts for almost 75% of peak gastric acid output.1

Vagal denervation is an integral part of any surgery for peptic ulcer disease. Several methods have been used to evaluate completeness of vagotomy; among the ones commonly used are measuring gastric acid output after modified sham feeding and mapping the acid secreting mucosa by the Congo red test.

Endoscopic sclerotherapy is routinely used in the management of bleeding esophageal varices. The sclerosant induces a neuroinflammatory response in the perivascular area and later leads to fibrosis and subsequent obliteration of varices. This inflammation can extend to the periesophageal tissue and possibly involve the vagus nerve, leading to vagal dysfunction. Mascler et al2 evaluated pancreatic polypeptide secretion in response to insulin hypoglycemia, a well known stimulus for vagal nerve function. They demonstrated a transient and reversible reduction in pancreatic polypeptide secretion immediately after sclerotherapy; this observation was not seen 6 months after sclerotherapy.

Our study was conducted to evaluate vagal function in portal hypertensive patients who had undergone multiple sessions of sclerotherapy.

Methods

Portal hypertensive patients who had undergone at least three sessions of endoscopic variceal sclerotherapy were recruited for the study after obtaining valid, informed consent. The etiology of portal hypertension was established by biochemical tests, abdominal ultrasonography and isotopic scan, and confirmed by liver biopsy if necessary. Sclerotherapy was done by the free hand technique, with 3% phenol in water as sclerosant; 3 mL sclerosant was injected per site. Attempt was made to inject intravariceally; no tamponade was applied.

Patients with portal hypertension who had never been subjected to sclerotherapy or variceal ligation were recruited as controls. Patients receiving drugs which could modify gastric acid secretion and those who had prior esophageal or gastric surgery or recent variceal bleed were excluded.

Protocol

The study protocol was approved by the Institution’s Ethics Committee. Gastric acid secretion was measured at least 2 weeks after the last sclerotherapy session. Controls were evaluated immediately on recruitment. Congo red test: A diagnostic gastroscopy was performed, and residual gastric secretions were aspirated. Sixty mL of 0.5% sodium bicarbonate was sprayed on the gastric mucosa and was aspirated after 1 minute contact with the gastric mucosa. Thirty mL of 2% freshly prepared Congo red was then sprayed evenly to cover the entire gastric mucosa. After 3 minutes, the mucosa was observed for change in color from red to black, signifying acid secretory mucosa; persistence of red color indicated non-secretory mucosa.

Gastric acid analysis by modified sham feeding: A nasogastric tube was positioned in the antrum after overnight fast. The residual gastric contents were aspirated and discarded. The basal acid output was estimated by aspirating gastric juice in aliquots of 15 minutes each using a
continuous low-pressure suction device. The patients were then asked to chew and spit out a fixed meal (vegetable sandwich) without swallowing. Following this sham feed, gastric acid was again collected in aliquots of 15 min each for a total of 1 hour. The volume of gastric acid in each aliquot was noted and the pH determined using a pH meter.

Results

Nineteen patients (12 men, 7 women; mean (SD) age 31.8 (13.3) years) comprised the study group. Seven patients (3 men, 4 women; mean age 22.9 (6.3) years) who had never been subjected to sclerotherapy were recruited as controls.

The etiology of portal hypertension in the study group was cirrhosis of liver (7), non-cirrhotic portal fibrosis (3) and extrahepatic portal venous hypertension (9), as compared to 3, 2 and 2 controls, respectively. Patients from the study group had undergone a median 10 (range 3-52) sessions of sclerotherapy. Fourteen patients in the study group had portal hypertensive gastropathy (8 mild, 6 severe) in comparison to 4 controls (all mild).

On Congo red test, complete or substantial reduction in the acid secreting mucosa was observed in eight patients from the study population; none of the controls had any reduction in acid secreting mucosa (p<0.05).

The results of the sham feed stimulated gastric acid output are shown in the Table.

The results of the Congo red test and gastric acid analysis in the study group were independent of gender, age, etiology of portal hypertension and the number of prior sclerotherapy sessions.

Discussion

Esophageal variceal sclerotherapy (EVS) is a mainstay in the management of esophageal varices. Complications such as esophageal ulceration, stricture and perforation are known. Several studies have documented altered esophageal motility following sclerotherapy, possibly occurring due to local irritation or neural damage.

The necroinflammatory response following sclerotherapy may involve structures adjacent to the esophagus; pleuritis, mediastinitis and pericarditis have been described following EVS. Chaudhary et al and Mathur et al noted dense periesophageal fibrosis during devascularization surgery in patients who have undergone multiple sessions of sclerotherapy. The vagus, which runs in close proximity to the esophagus, is also liable to be damaged following EVS. Masclay et al demonstrated transient vagal dysfunction after sclerotherapy. We studied gastric acid secretion as a marker of functional integrity of the vagus.

The Congo red test is a qualitative test which allows mapping of the acid-secreting mucosa. Measurement of basal (BAO) and modified sham feed (SAO) stimulated gastric acid output is a quantitative test of vagal function. Eight of our 19 patients who had undergone EVS had complete or substantial reduction in the acid-secreting mucosa on Congo red test. These patients also had significantly lower BAO and SAO as compared to the controls. The 11 patients whose acid-secreting mucosa was normal also had lower BAO and SAO as compared to the controls, though this difference was not significant.

Gender, age, etiology of portal hypertension and the number of prior sclerotherapy sessions were not found to affect the development of vagal dysfunction following EVS.

We have shown that EVS is associated with lower gastric acid secretion as compared to controls. This could be due periesophageal inflammation following EVS, leading to vagal dysfunction. The duration and long-term consequences of this lower acid secretion are not known. The effect of vagal dysfunction following EVS on the rest of the gastrointestinal tract also needs to be studied.

References


Table: Gastric acid output (mmol/h) after modified sham feed stimulation

<table>
<thead>
<tr>
<th>Acid study</th>
<th>Study group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Congo abnormal (n=5)</td>
<td>Congo normal (n=11)</td>
</tr>
<tr>
<td>BAO</td>
<td>0.34 (0.42)*</td>
<td>1.11 (1.48)</td>
</tr>
<tr>
<td>SAO</td>
<td>0.52 (1.00)**</td>
<td>1.65 (1.66)</td>
</tr>
</tbody>
</table>

Values are mean (SD)
P*<0.01; **<0.05 as compared to control group (Student's t test for unpaired data)

Correspondence to: Dr Abraham, Professor and Head. Fax: (22) 414 3495

Received July 1, 1997. Received in final revised form September 16, 1997. Accepted September 20, 1997

Indian Journal of Gastroenterology 1998 Vol 17 23