Mucosal Abnormalities of the Upper Gastrointestinal Tract in Patients with Portal Hypertension: A Reappraisal

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Abstract

In a retrospective study of upper gastrointestinal endoscopy in patients with portal hypertension, abnormalities other than varices were detected in 90 (30.9%) of 304 patients with a past history of haematemeses and/or melena. These lesions were most common in cirrhotics, both alcoholic (42.1%) and non-alcoholic (36.2%), and were less common in non-cirrhotic portal fibrosis (16.2%) and in extrahepatic portal hypertension (8.3%). Moreover, similar lesions were found in 38 (18.6%) of 216 patients with portal hypertension who had never bled in the past, and were again most common in cirrhotics. These findings suggest that the possibility of bleeding originating from lesions other than varices should be seriously considered in patients with portal hypertension (especially of cirrhotic origin) who present with upper gastrointestinal haemorrhage.

Key words: Portal hypertension, non-variceal bleeding lesions.

Introduction

The introduction of endoscopy has facilitated the diagnosis and management of upper gastrointestinal (UGI) haemorrhage. In a number of studies from the West, a high incidence of UGI mucosal lesions has been documented in patients with portal hypertension presenting with bleeding. In fact, between 37.5% and 80% of these patients were found to be bleeding not from varices but from erosive mucosal lesions, gastric or duodenal ulcers or Mallory-Weiss tears. In contrast, studies from India suggest that such lesions are uncommon in patients with portal hypertension, and in the event of a bleed the source is almost invariably the varices. This difference has been ascribed to the fact that in the West the majority of patients are cirrhotics, while in India non-cirrhotic portal fibrosis is the most common cause of portal hypertension. The present retrospective study was carried out to analyse the incidence of mucosal lesions and their association with the etiological cause of portal hypertension.

Material and Methods

The endoscopy records for a five year period between July 1980 and June 1985 were retrospectively analysed. The case records of all patients with portal hypertension were scrutinised. We included only those patients who had been diagnosed as having extrahepatic portal hypertension, noncirrhotic portal fibrosis or cirrhosis of the liver. The diagnosis of extrahepatic portal hypertension was made by splenorenovenography; non-cirrhotic portal fibrosis was diagnosed on the basis of clinical and laboratory data, splenorenovenography and liver biopsy; cirrhosis was diagnosed on the basis of clinical and laboratory data, nuclear scans and, whenever feasible, liver biopsy.

Three hundred and four patients with a history of UGI bleeding in the past ("bleeders") were included in the study. During the same period, 216 patients without a past history of UGI bleeding ("non-bleeders") were also examined. In all these patients, oesophageal varices were present at endoscopy, and the following additional data were recorded:

1. The presence of another lesion such as duodenal and gastric ulcers, duodenal and gastric erosions or Mallory-Weiss tears. Erosions were diagnosed only if a distinct mucosal break was observed. For the purposes of this study, we excluded reports of 'mild' or 'moderate' gastritis or duodenitis. If the same patient had two or more of these lesions (e.g. duodenal ulcer along with gastric erosions) he was listed only under one category (in this case, duodenal ulcer).

2. The association between the aetiology of portal hypertension and the incidence of these lesions.

3. The relationship of the detection rate of such lesions and the timing of endoscopy.

The data were analysed separately for the "bleeders" and the "non-bleeders".

Results

Of the 304 patients who had a history of UGI bleeding, 198 (65.6%) had cirrhosis, 70 (23%) noncirrhotic portal fibrosis and 36 (12.2%) extrahepatic portal obstruction. Of the cirrhosis, 81 (41%) were alcoholic, 36 (19%) had chronic active hepatitis with cirrhosis, 2 (1%) had Wilson's disease, and the other 77 (39%) had cryogenic cirrhosis. Ninety (30%) of these patients had an associated lesion of the UGI tract. The most common abnormality was gastric erosions in 26 (8.5%) patients, followed by duodenal ulcer in 23 (7.6%), duodenal erosions in 17 (5.6%), and gastric ulcer in 12 (4%) patients. Other lesions detected were haemorrhagic gastritis, oesophageal ulcers and Mallory-Weiss tears.

These abnormalities were most common in patients with cirrhosis, occurring in 38% of these patients, and were seen less frequently in patients with non-cirrhotic portal fibrosis (16%) and extrahepatic portal obstruction (8%). Among the cirrhotics, such lesions were equally common in patients with alcoholic (42%) and those with non-alcoholic (36%) cirrhosis.
The detection rate of these mucosal abnormalities was 33% in 43 patients endoscoped within 24 hours of a bleed, 35% in 38 patients endoscoped between 24 and 72 hours, 29% of 35 patients examined within a week, and 29% of 188 patients examined more than a week after the last bleed.

Of the "non-bleeders", 188 (87%) had cirrhosis, 23 (11%) had non-cirrhotic portal fibrosis and 5 (2%) had extraportal portal obstruction; 38 (18%) of these patients had an associated mucosal abnormality. Gastric erosions and duodenal erosions were most common, being seen in 12 (6%) patients each, followed by duodenal ulcer in 9 patients (4%) and gastric ulcer in 5 patients (2%). These mucosal abnormalities were most common in patients with cirrhosis (19%), while only one patient (4%) with non-cirrhotic portal fibrosis and one (20%) with extraportal portal obstruction had such abnormalities.

There was no significant difference in the mean serum albumin and bilirubin levels and prothrombin time between the bleeders and non-bleeders.

Discussion
In studies from the UK and the USA, upper GI lesions other than varices were reported to be the site of bleeding in 37% to 82% of patients with portal hypertension. In many of these studies, alcoholic cirrhosis was the predominant or the only cause of portal hypertension. It was presumed that the harmful effect of alcohol on the gastric mucosa was responsible for the high incidence of these lesions. This interpretation is supported by a study in which non-alcoholic patients with portal hypertension were found to bleed only rarely from a duodenal or gastric ulcer. In one of these studies, however, the mucosal abnormalities were equally common in alcoholic and non-alcoholic cirrhotics. In a recent study of 140 patients, most of whom were alcoholic, mucosal abnormalities were the source of bleeding in only 10%. Another study found that associated mucosal lesions were very common in patients with portal hypertension (39%) but only rarely were they the source of bleeding.

Studies from India have stressed the rarity of such lesions in patients with portal hypertension, and this difference has been ascribed to the fact that most of the patients had non-cirrhotic portal fibrosis rather than cirrhosis. However, in one study from India, only 5 of 185 patients with portal hypertension had associated UGI lesions such as ulcer or gastritis, despite the fact that cirrhosis (81 patients, 44%) was the commonest cause of portal hypertension.

The present study confirms that associated lesions of the UGI tract are common in our patients with portal hypertension. This perhaps reflects the fact that the majority of our patients with portal hypertension had cirrhosis, unlike centres elsewhere in India where non-cirrhotic portal fibrosis is more common. The mucosal abnormalities were equally common in alcoholic and non-alcoholic cirrhosis, indicating that alcohol is not the aetiological factor in the production of these lesions. They may be related to the underlying liver disorder since these lesions were seen with equal frequency in non-cirrhotic portal fibrosis and extraportal portal obstruction.

As this is a retrospective study, we cannot determine whether or not these lesions contributed to the bleeding episode or whether they occurred as an epiphenomenon caused by the stress of bleeding. However the fact that these lesions were seen less commonly in patients with portal hypertension without any history of bleeding suggests that they may have had a temporal relationship with the bleeding episode. In patients with cirrhosis and varices the possibility that a lesion other than the varices may be responsible for or may contribute to the bleeding should always be considered.

References

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