IN INDIA ELSEWHERE


Esophageal ulcers and stricture occur with high frequency after endoscopic sclerotherapy (EST) using absolute alcohol (AA). The authors studied functional esophageal alterations after variceal eradication with AA.

Twenty-four patients (mean age 37.4 y, range 11-60; 18 men) with grade esophageal varices (cirrhosis-22, non-cirrhotic portal fibrosis-1, extrahepatic portal venous obstruction [EHPVO]-1) not having received EST in the past (pre-EST group), and 22 patients (mean age 35.4 y, range 11-56; 17 men; cirrhosis-21, EHPVO-1) in whom varices were eradicated (post-EST group) were studied. No patient had significant ascites.

Manometry was normal in 15 (62.5%) patients before and 2 (9%) after variceal eradication (p<0.001). Low amplitude of contraction in the distal esophagus (18.2 [14.3] vs 63.4 [24.9] mmHg, p<0.01), prolonged duration of contraction in the proximal (5.4 [2.6] vs 3.3 [0.8] s, p<0.001) and distal (6.6 [2.3] vs 4.3 [1.1] s, p<0.001) esophagus, and increased frequency of multi-level contraction (13 vs 3, p<0.001) were seen post-EST. The frequency of simultaneous contraction, non-transmitted contraction and spontaneous contraction was also more (p=ns) post-EST. LES pressure and velocity of contraction were lower (p=ns) in this group.

Seventeen of them had esophageal ulcers, 10 had transient dysphagia and 5 had mechanical dysphagia.

The authors conclude that esophageal dysmotility is frequent and marked after EST with AA. In most patients, the dysmotility remains asymptomatic.

Sarai SK, Shahi HM, Jain M, Jain AK, Issar SK, Murthy NS (Department of Gastroenterology, G B Pant Hospital, and Department of Biostatistics, Maulana Azad Medical College, New Delhi). The natural history of portal hypertension: asynergy: influence of variceal eradication. Am J Gastroenterol 2000;95:2888-93

In patients with portal hypertension, esophageal varices and portal hypertensive gastropathy (PHG) are the common causes of gastrointestinal (GI) bleeding. The authors prospectively studied the influence of sclerotherapy on the course of PHG existing before and that developing during or after variceal eradication.

Of 967 patients achieving variceal obliteration using absolute alcohol, 88 (9.1%); mean age 35.4 [17.2] y; cirrhosis-54, non-cirrhotic portal fibrosis [NCPF]-18, extrahepatic portal vein obstruction [EHPVO]-16 had PHG (n=78), gastric antral vascular ectasia (GAVE; 2) or both (8), 22 (26%) patients had PHG before (group A) and 64 developed PHG after eradication of varices (group B).

PHG was seen most often in the fundus of the stomach (70%) and was diffuse in 9 (10.4%) patients. It was transient in 34.9%, persistent in 55.8% and progressive in 9.3%. Patients with cirrhosis had persistent or progressive PHG more often (75% vs 40.4%, p<0.05). None of the patients with EHPVO had progressive PHG or bled due to PHG. During a follow up of 25.1 [14.2] months, PHG disappeared in 2 (9%) group A and 28 (44%) group B patients (p=0.05), and progressed more often in group A (p=ns). The frequency of bleeding was higher in group A (32% vs 4.7%, p=0.02), in patients with cirrhosis (p=ns), and in those with persistent or progressive PHG and severe PHG. GAVE did not regress or disappear in any patient: there was no significant blood loss in the 2 patients with only GAVE. One patient from group A died of uncontrolled bleeding from severe PHG while one patient with NCPF and bleed from severe PHG required shunt surgery.

The authors conclude that PHG present before sclerotherapy often persists or progresses after variceal obliteration, with likelihood of bleeding. PHG developing after variceal eradication is often transient and less severe.


Acute renal failure (ARF) associated with liver disease has a high mortality. The authors prospectively analyzed the outcome in patients with liver disease and ARF.

Of 221 patients with ARF and liver disease, 66 developed ARF secondary to cirrhosis (n=29, mortality 8), fulminant hepatic failure (25, 15) or obstructive jaundice (12, 7). The factors leading to ARF were volume depletion (24), gastrointestinal bleed (28), sepsis (34) and drugs (27), along with hyperbilirubinemia.

Patients with ARF with coexistent liver injury (n=155) had malaria (n=37, mortality 15), sepsis (36, 22), hypovolemia with ischemic hepatic injury (14, 5), acute pancreatitis (12, 4), rifampicin toxicity (10, no mortality), paroxysmal nocturnal hemoglobinuria (3, no mortality), copper sulphate poisoning (3, 2), recent abortion (11, 6) or delivery, including the HELLP syndrome (12, 4), and uncertain etiology (14, 4). Overall mortality was 42.5%, the maximum mortality (60%) being in patients with sepsis and toxic hepatitis with ARF.

Thus, coexistence of liver disease and ARF leads to high mortality. Correction of precipitating factors may prevent ARF. Prompt renal replacement therapy improves outcome.

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