Gastric Ulcer and Cholestasis Following Injection Therapy of Bleeding Duodenal Ulcers

In a recent issue of Endoscopy Loperfido et al. (1) reported a case of extensive necrosis of the gastric mucosa following injection of polidocanol and epinephrine into a bleeding peptic ulcer. In the same issue another case of gastric necrosis following injection of ethanolamine oleate was described (2). We report on two cases in which gastric ulcers occurred after injection therapy of bleeding duodenal ulcers, one accompanied by hepatic cholestasis.

The first patient (case no. 1) was a 58-year-old woman admitted to our intensive care unit (ICU) with hematemesis and a history of arterial hypertension. Several weeks before admission the patient had experienced epigastric cramp-like pain. The onset of bleeding was sudden and massive. On admission her pulse rate was 130/min and her blood pressure 70/45 mmHg. The haemoglobin level was 3.0 g/dl. Other tests, including tests of hepatic function were normal. After resuscitation with intravenous fluid and eight units of blood, emergency endoscopy was performed. The oesophageal and gastric mucosa were normal. A large ulcer was present on the posterior wall of the duodenum in which a large vessel was visible. Endoscopic sclerotherapy (ES) was performed, 4 cc of epinephrine F(1/10 000) being injected around the visible vessel followed by injection of 6 cc of polidocanol (1.5%) into the vessel. There was no rebleeding and the patient was discharged on day 6. Six weeks later another endoscopy was performed; the duodenal ulcer had healed but there were numerous small superficial gastric ulcers, mainly in the antrum along the greater curvature. Laboratory investigations revealed a cholestasis syndrome (alkaline phosphatase: 3 x N, gamma GT: 5 x N). Unfortunately the patient did not report for follow-up.

The second patient (case no. 2) was a 77-year-old woman admitted in our department with melena. Eight days before she had undergone surgery for insertion of a hip prosthesis. Three days after surgery she developed melena and thoracic pain, investigation revealing a myocardial infarction. On examination the patient was pale, with a pulse rate of 140/min, and blood pressure 80/40 mmHg; the hemoglobin level was 5.0 g/dl. The patient was given eight units of blood. Five days later rebleeding occurred and the patient was admitted to our ICU. Emergency endoscopy revealed a large duodenal ulcer on the posterior wall with a large visible vessel. The gastric antrum was normal. On ES, 5 cc of adrenaline (1/10 000) was injected around visible vessel and 8 cc of polidocanol (1.5%) was injected into the vessel. The patient's further course was uneventful and she was discharged on day 10. Four weeks later the duodenal ulcer had practically healed but a 1 cm long linear gastric ulcer was found along the greater curvature in the antrum (Figure 1); the surrounding mucosa was erythematous and antral folds were attracted towards the ulcer. No biochemical tests were performed. Two weeks later the ulcer was replaced by a long white scar. Four months later the scar was still evident and the mucosa was normal but the antrum seemed deformed; the duodenal ulcer had healed completely.

Injection therapy is an effective means of controlling bleeding peptic ulcers, as demonstrated in several recent randomized controlled trials. In these studies, no side effects related to injection therapy were reported. However, several isolated cases of complications due to ES were reported, including one case of perforation following ES using absolute ethanol (3), and one case of dissecting duodenal haematoma (4) after injection of epinephrine. A case of pancreaticoduodenal necrosis following endoscopic treatment of a bleeding duodenal ulcer with epinephrine and polidocanol and then injection of 1-butyl 2-cyano acrylate (Histoacryl) was also reported (5). Histoacryl was found to be present in the gastro-duodenal and right hepatic arteries, which were thrombosed.

Reflux of the sclerosing agent in hepatic and gastro-duodenal arteries could explain the abnormalities observed in our two cases. The small gastric ulcers (case no. 1) and the long linear ulcer (case no. 2) were located in the antrum, primarily along the greater curvature. It would seem possible that the sclerosing agent injected into the visible vessel might flow back into the gastro-duodenal artery and then into the arteries feeding the right portion of the great curvature. Such a
vascular route was described in the case of injection therapy of esophageal varices complicated by gastric ulcers (6).

Cholestasis, as observed in case no. 1, could be induced by the sclerosing agent. In a previous paper (7) we reported that 29 of 32 patients experienced hepatic cholestasis after injection sclerotherapy of esophageal varices using polidocanol. This cholestasis could be related to hepatic dissemination of polidocanol via a vascular route.

There remains no doubt that ES for bleeding ulcers is a highly valuable treatment modality, but as these reports show this simple technique can be associated with several complications.

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References