Distal biliary stricture as a complication of sclerosant injection for bleeding duodenal ulcer

W Luman, N Hudson, C P Choudari, M A Eastwood, K R Palmer

Abstract
A patient undergoing repeated endoscopic injection therapy for important bleeding from a duodenal ulcer developed intestinal perforation followed by extrahepatic obstructive jaundice resulting from benign biliary stricture. It is proposed that these complications were a consequence of the use of ethanolamine olate as part of the injection regimen and caution against the use of this material is needed, particularly as current clinical trials suggest that sclerosants offer no advantage over injection therapy with dilute adrenaline alone.

Endoscopic injection therapy improves the prognosis of patients presenting with peptic ulcer haemorrhage.1-6 Published studies have used a variety of injection agents including dilute adrenaline, polidocanol, absolute alcohol, and ethanolamine olate in a variety of combinations and concentrations, but the safest and most effective regimen is unclear. Sclerosants are potentially dangerous7-12 and studies are conflicting as to whether they are a necessary adjunct to adrenaline; for example Lin et al reported that a combination of 1:10 000 adrenaline and absolute alcohol was a more effective treatment than dilute adrenaline,13 while others have not found this.14 15

Endoscopic injection therapy seems remarkably safe but we report a hitherto unrecognised complication of sclerosant injection for bleeding duodenal ulcer.

Case history
An eighty year old woman was admitted with a 12 hour history of haematemesis and melaena. She had been taking slow release diclofenac 100 mg every day for arthritic back pain and prednisolone 5 mg every day for polymyalgia rheumatica for three years. Three months earlier an emergency hysterectomy for pyometrium was complicated by left ventricular failure. In addition she had a previous history of chronic obstructive airways disease and idiopathic thrombocytopenic purpura. On admission the pulse rate was 120 beats per minute, the systolic blood pressure was 80 mm Hg, and she displayed epigastric tenderness. The haemoglobin concentration was 76 g/l, platelets $205 \times 10^9/l$, prothrombin time 1·0, urea 8·7 mmol/l, liver function tests were normal. After resuscitation with intravenous colloid and blood, she had an endoscopy, which showed a 15 mm anterior duodenal ulcer with a visible spurting vessel in its base. The ulcer was injected with 3 ml of 1:100 000 adrenaline and the vessel stopped bleeding. She received four units of blood and was discharged on the fifth day while taking omeprazole 40 mg every day. The following day she was readmitted with further haematemesis. On this occasion she was haemodynamically stable and her haemoglobin concentration was 96 g/l. She had a further endoscopy at which the bleeding duodenal ulcer was again seen. The vessel was re-injected with 8 ml of adrenaline 1:100 000. After consultation with our surgical colleagues it was decided to treat her conservatively and endoscopy was repeated 48 hours later. The vessel in the ulcer base remained protuberant and was again injected with 3 ml adrenaline 1:100 000 and on this occasion, an additional injection of 0·5 ml of 5% ethanolamine was placed directly into the vessel. She remained haemodynamically stable after this third therapeutic endoscopy. Five days later, however,

Figure 1: Endoscopic retrograde cholangiography showing complete obstruction in the lower common bile duct compounding to displacement of the main pancreatic duct.
she developed fever, confusion, abdominal tenderness and guarding. A chest radiograph showed free subdiaphragmatic air. The presumed ulcer perforation was treated conservatively with intravenous fluids, nasogastric suction, and antibiotics and she was discharged on the 15th day.

On review in outpatients eight weeks later the patient complained of tiredness and general malaise but denied abdominal pain or vomiting. On examination she was mildly icteric. Serum liver function tests showed: alkaline phosphatase 1100 U/l, bilirubin 40 mmol/l, alanine transaminase 579 U/l. An upper abdominal ultrasound scan showed normal liver texture but the common bile duct was dilated to 16 mm in diameter. Endoscopic retrograde cholangiopancreatography showed a tight smooth stricture in the distal common bile duct and the distal pancreatic duct was displaced towards the common bile duct (Fig 1). After endoscopic retrograde cholangiopancreatography there was spontaneous improvement in her liver function tests; alkaline phosphatase 618 U/l, bilirubin 12 μmol/l, alanine transaminase 107 U/l. At percutaneous transhepatic cholangiography the biliary stricture was again noted but a fistulous tract had formed between the common bile duct proximal to the stricture and the first part of the duodenum (Fig 2). The stricture was dilated with a balloon and a 12 F double mushroom biliary stent was inserted percutaneously (Fig 3). Subsequently the liver function tests normalised and the stent was removed six weeks later.

**Discussion**

Endoscopic injection therapy is now commonly used to treat bleeding peptic ulcer. The most effective and safest injection regimen is not yet established and different groups have reported comparable results using dilute adrenaline alone, sclerosants alone, and combination treatment. Injection therapy is generally regarded as safe but isolated reports of complications have appeared and these have usually occurred after the use of sclerosants. Furthermore, studies from our own group and from Hong Kong have suggested that the addition of sclerosants to dilute adrenaline confers no benefit in terms of stopping bleeding or preventing rebleeding. This case report adds to the increasing number of complications associated with sclerosant injection. We suggest that both the intestinal perforation and the biliary stricture were a complication of ethanolamine olate therapy. Clearly we cannot exclude a spontaneous ulcer perforation and subsequent inflammatory reaction leading to the biliary problem but this seems unlikely. The patient’s treatment was consistently conservative, principally because her poor general condition precluded surgical intervention. We would argue that her initial presentation at a great age with important bleeding from a giant duodenal ulcer with a background of chronic obstructive airways disease and ischaemic heart disease put her at a very high risk of postoperative death should surgical intervention have been undertaken. Avoidance of surgery by endoscopic injection almost certainly prevented death, but it might be argued that a surgical operation after haemostasis and resuscitation would have avoided the series of complications described in this report. The timing of surgery is clearly difficult and we do not yet have sufficient information to make reasoned decisions in such cases.

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