Late bleeding after endoscopic sphincterotomy for bile duct calculi

I A Finnie, M V Tobin, A I Morris, I T Gilmore

Haemorrhage after endoscopic sphincterotomy for gall stones may occur unexpectedly days or weeks later

The most common complication of endoscopic sphincterotomy for bile duct calculi is haemorrhage.¹ Although this usually occurs at the time of the procedure or within hours, this may not always be so. We describe five patients who presented with late bleeding, which has implications when advising patients about possible adverse events.

Case histories

Case 1—A 45 year old woman with jaundice due to a 2 cm ductal calculus underwent endoscopic sphincterotomy. The calculus could neither be delivered through the sphincterotomy nor be crushed. There were no immediate complications and she was allowed home after 48 hours with a view to surgery the next week. Seventy two hours after the sphincterotomy she was readmitted with hypovolaemic shock after a massive melaena. The bleeding settled with a combination of somatostatin infusion and endoscopic diathermy of the sphincterotomy site using bipolar electrocoagulation. A subsequent laparotomy, common duct exploration, and stone extraction were uncomplicated.

Case 2—An 82 year old man presented with abdominal pain, fever, jaundice, and impaired renal function. The clinical picture and ultrasonic appearances suggested ductal calculi and, although no definite stones were seen at endoscopic retrograde cholangiography, a sphincterotomy was made. No stones were retrieved on trawling the duct. His general condition initially improved, but 72 hours later he developed melaena and hypovolaemia. He eventually required duodenotomy and underrunning of the sphincterotomy site for continued bleeding, but no ductal stones were found. Nineteen days after sphincterotomy he died of multiorgan failure.

Case 3—A 65 year old woman with jaundice due to a 3 cm ductal calculus underwent endoscopic sphincterotomy. It was not possible to deliver or crush the stone, and therefore laparotomy, common duct exploration, and stone extraction were carried out 48 hours later. Five days after that operation she developed melaena. The bleeding continued and further laparotomy was performed. The source of the bleeding was the sphincterotomy site. This was underrun and there were no further stones. The patient subsequently recovered uneventfully.

Case 4—A 48 year old woman with a previous cholecystectomy had endoscopic sphincterotomy and duct clearance for retained ductal calculi. No stricture was seen at cholangiography. Bleeding at the time of sphincterotomy required transfusion, and she was allowed home when her haemoglobin concentration was stable and stools had returned to normal colour. Twelve days after sphincterotomy she was readmitted with melaena and hypotension. The sphincterotomy site was cauterised and bleeding stopped. She was allowed home but a further bleed occurred at 21 days. The sphincterotomy site was again cauterised and a somatostatin infusion was given. The bleeding stopped, and one

year later she was well with no further episodes of bleeding.

Case 5—A 72 year old jaundiced man underwent endoscopic sphincterotomy and stone extraction for ductal calculi. He was well initially, but 72 hours after the procedure he developed melaena and hypotension. He was managed conservatively.

Discussion

Haemorrhage after endoscopic sphincterotomy has been reported in 4-10% of patients,^{1,2} although the definition of what constitutes a serious bleed varies. All our patients required transfusion (mean 7 units, range 2-12), and in all cases endoscopic or operative confirmation, or both, was obtained that the sphincterotomy site was the source of bleeding. The five patients were seen over 24 months, during which endoscopic sphincterotomy was performed in 200 patients for ductal calculi. A further patient needed transfusion (2 units) for an immediate haemorrhage after sphincterotomy during this time. Only one patient died (case 2) after sphincterotomy for calculi.

We do not know why some patients bleed after endoscopic sphincterotomy. Retained calculi may be a risk factor, and it is routine to achieve duct clearance whenever possible. Retained calculi may have been a contributory factor in case 1, but in the other cases the bile duct was free of calculi at the time of haemorrhage. In all the above cases prothrombin time and platelet count were normal, and a recent study found no correlation between coagulation measurements and haemorrhage after endoscopic sphincterotomy.³ The risk of bleeding may be related to the size of the sphincterotomy.¹

Immediate haemorrhage is likely to be from branches of the pancreaticoduodenal artery² whereas late bleeding may be due to separation of the coagulum from the sphincterotomy site.⁴ This delayed haemorrhage occurs at a time when patients usually have been discharged from hospital after an apparently uneventful procedure. Endoscopists should warn them that bleeding may occur as a late complication. General practitioners are advised to refer patients back to hospital urgently should they develop melaena after sphincterotomy; such haemorrhage may be torrential but sometimes settles with conservative management.

Part of this material was presented in abstract form at the spring meeting of the British Society of Gastroenterology, 1990.

- 1 Vaira D, Ainley C, Williams S, *et al*. Endoscopic sphincterotomy in 1000 consecutive patients. *Lancet* 1989;ii:431-3.
- 2 Goodall RJR. Bleeding after endoscopic sphincterotomy. *Ann R Coll Surg Engl* 1985;67:87-8.
- 3 Hill J, Maxwell AJ, Tweedle DEF, Martin DR. Do measured parameters of blood clotting help predict haemorrhage after endoscopic sphincterotomy? [Abstract]. *Gut* 1990;31:A608.
- 4 Leese T, Neoptolemos JP, Carr-Locke DL. Successes, failures, early complications and their management following endoscopic sphincterotomy: results in 394 consecutive patients from a single centre. *Br J Surg* 1985;72:215-9.

(Accepted 12 March 1991)

Gastroenterology Unit,
Royal Liverpool Hospital,
Liverpool L7 8XP
I A Finnie, MRCP, registrar
M V Tobin, MRCP, senior
registrar
A I Morris, FRCP, consultant
physician
I T Gilmore, FRCP, consultant
physician

Correspondence to:
Dr Gilmore.

BMJ 1991;302:1144