Splenic laceration after endoscopic retrograde cholangiopancreatography

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Endoscopic retrograde cholangiopancreatography has been part of clinical practice for over 35 years. The procedure itself carries risks. Most complications associated with the procedure have been described in the literature. Splenic injury is an extremely rare complication after endoscopic retrograde cholangiopancreatography and only nine such cases have been reported to our knowledge since 1988. We report on a patient who presented with abdominal pain and was found to have abnormal liver function and a dilated pancreatic duct. An endoscopic retrograde cholangiopancreatography was performed and a splenic laceration was noted subsequently. As this operation is an increasingly important diagnostic and therapeutic modality for pancreatico-biliary disease, clinicians must be aware of this complication in order to make an early diagnosis and begin appropriate management.

Introduction

Endoscopic retrograde cholangiopancreatography (ERCP) is an invasive procedure that has been commonly used to diagnose and treat hepatobiliary and pancreatic diseases for the past 35 years. The procedure itself carries a complication rate of between 5 and 10%. Splenic injury is an extremely rare but potentially lethal complication of ERCP. Here we report on a patient who had a splenic laceration after ERCP.

Case report

A 63-year-old woman with a history of hypertension treated with beta-blockers, and a laparotomy done many years ago for an unknown disease, presented to the hospital complaining of intermittent upper abdominal pain for 2 days. A physical examination revealed a soft abdomen with mild tenderness over the left upper quadrant. A liver function test showed a bilirubin level of 46 µmol/L (reference level, <17 µmol/L), an alkaline phosphatase level of 607 U/L, and an amylase level of 857 U/L. Her clotting profile was normal with a prothrombin time of 9.8 s (reference range, 8.5-11.5 s), an activated partial thromboplastin time (APTT) of 40.2 s (reference range, 27.5-40.5 s), and an international normalised ratio (INR) of 1.0. An abdominal ultrasound indicated that her pancreatic duct was dilated. A fully accredited endoscopist performed an ERCP with the patient lying in a prone position throughout the procedure. The endoscopist encountered difficulties when trying to cannulate the common bile duct and the pancreatic duct was the only duct successfully cannulated. The endoscope was bowed in the ‘long’ position in an attempt to cannulate the common bile duct and no epigastric hand pressure was used. The pancreatographic showed a stricture of the pancreatic duct at the head of pancreas with a distorted and dilated distal duct. No obvious bleeding tendency was observed during papillotomy. The procedure took 90 minutes and the patient was transferred back to a general ward immediately after the procedure. She appeared well and oral intake was resumed 4 hours after the procedure. Her vital signs and physical examination were unremarkable.

Eighteen hours after the procedure, the patient became hypotensive with a systolic and diastolic blood pressure of 70 and 40 mm Hg respectively. She complained of epigastric and nausea. She was afebrile with a pulse rate of 70 beats/min. A physical examination revealed mild epigastric and left upper quadrant tenderness without peritoneal signs. Intravenous crystalloid was administered and her systolic blood pressure was stabilised at 90 mm Hg. Her haemoglobin level was 92 g/L and her haematocrit was 27%; the platelet count was 230 x 10^9/L, prothrombin time was 11.1 s, APTT was 30.0 s, and INR was 1.1. Abdominal computed tomography (CT) demonstrated a large haemoperitoneum and extravasation of contrast in the splenic region (Fig). An emergency laparotomy revealed approximately 4 L of fresh blood and clots. Active bleeding from a laceration on the superioposterior aspect of the spleen was noted. The laceration transected the superioposterior quadrant of the spleen with the quarter completely avulsed from the rest. Features of chronic pancreatitis
were present. No adhesions were noticed, despite the history of a previous laparotomy. A splenectomy was performed and examination of the spleen showed a splenic tear, a subcapsular haematoma but otherwise normal splenic tissue. The patient had an unremarkable recovery after the operation.

**Discussion**

Endoscopic retrograde cholangiopancreatography is associated with a spectrum of well-defined complications. As ERCP has become more widely used, rare complications of the procedure have been reported more often in recent years. Cases of splenic injury after colonoscopy are well described in the literature but splenic injury after ERCP remains rare. The first report of splenic injury appeared in 1988 and a total of nine cases have been documented in the literature since (Table 2-10). In the past 10 years approximately 70 000 ERCP procedures have been done in Hospital Authority hospitals in Hong Kong but we have not found any other reported cases with post-ERCP splenic injuries.

Although the cases reported in the literature have been categorised as post-ERCP splenic injuries, they actually have variable pathological findings. They include avulsion of the short gastric vessel(s) in two,\(^2,3\) avulsion of the splenic capsule in two,\(^4,5\) subcapsular haematomas (or abscesses resulting from haematomas) in two,\(^6,7\) and splenic lacerations in two.\(^8,9\) Whether these different pathological findings can be attributed to different or similar aetiological causes remain unclear.

The exact mechanism causing splenic injuries after ERCP remains unresolved. ‘Bowing’ of the endoscope in the ‘long’ position with torsion on the greater curvature of the stomach (while attempting to pass the endoscope through the narrowed duodenum or to cannulate the papilla) is the causative mechanism postulated by most of the authors.\(^2,5,9,10\)

**TABLE. Features of cases with splenic injuries after endoscopic retrograde cholangiopancreatography (ERCP)**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Intervention</th>
<th>Time to diagnosis</th>
<th>Pathological findings</th>
<th>Therapy</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewis et al(^2)</td>
<td>ERCP, stenting</td>
<td>9 hours</td>
<td>Avulsion of short gastric vessels</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Zyromski and Camp(^3)</td>
<td>ERCP, sphincterotomy</td>
<td>72 hours</td>
<td>Avulsion of short gastric vessels</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Trondsen et al(^4)</td>
<td>ERCP, sphincterotomy</td>
<td>15 hours</td>
<td>Spleen ‘decapsulated’</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Wu and Katon(^5)</td>
<td>ERCP</td>
<td>68 hours</td>
<td>Splenic capsular avulsion</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Furman and Morgenstern(^6)</td>
<td>ERCP, sphincterotomy</td>
<td>Not available</td>
<td>Splenic abscess</td>
<td>Conservative</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Lo et al(^7)</td>
<td>ERCP, sphincterotomy</td>
<td>48 hours</td>
<td>Subcapsular haematoma</td>
<td>Conservative</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Ong et al(^8)</td>
<td>ERCP</td>
<td>48 hours</td>
<td>Splenic laceration</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Badaoui et al(^9)</td>
<td>ERCP</td>
<td>20 minutes</td>
<td>Splenic laceration</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
<tr>
<td>Kingsley et al(^10)</td>
<td>ERCP, stenting</td>
<td>24 hours</td>
<td>Not reported</td>
<td>Splenectomy</td>
<td>Death from multi-organ system failure</td>
</tr>
<tr>
<td>Present case</td>
<td>ERCP</td>
<td>18 hours</td>
<td>Splenic laceration</td>
<td>Splenectomy</td>
<td>Full recovery</td>
</tr>
</tbody>
</table>
This manoeuvre may cause splenic capsular tears or vascular avulsion by traction on the short gastric vessels. Inflation of the stomach has been suggested as another possible mechanism exerting tension on the greater curve of the stomach.5,6,10 These postulated mechanisms seem a good explanation for avulsion of the short gastric vessels during ERCP. It is, however, unlikely that these manoeuvres alone create sufficient force to injure the splenic parenchyma.

Difficulty with cannulation of the common bile duct or ampulla of Vater may lead to a prolonged procedure requiring more manipulation of the endoscope and thus transmission of excessive torque to the patient. This is probably an important factor predisposing to post-ERCP splenic injury. Use of gentle epigastric hand pressure to help ease the long-bowing of the endoscope and alleviate the mechanical stress on the spleen may help to decrease the risk of splenic injury. At the same time, it is worthwhile noting that a diagnostic ERCP with no therapeutic procedure was the leading event in two of the 10 cases. Another factor contributing to splenic injury after ERCP is the presence of abdominal adhesions due to prior abdominal surgery.5,6,10 Patients with cirrhosis, pancreatitis, and those who are on anticoagulants are expected to be more prone to splenic injury after ERCP.5,6 Features of pancreatitis were found in two out of the 10 cases, including our patient. The association between the occurrence of a post-ERCP splenic injury and pancreatitis cannot be explained by chance alone. Pancreatitis, especially chronic pancreatitis, may lead to calcification and fibrosis of the supporting ligaments between the pancreas and the spleen, resulting in reduced relative mobility between the stomach and spleen. This phenomenon may, in turn, increase the chance of splenic injury during ERCP.

Delayed diagnosis was a common feature in the post-ERCP patients with splenic injuries, due to the lack of awareness of this rare complication. Splenectomy may not be necessary for all patients. Two of the 10 patients were successfully managed conservatively. Early diagnosis may increase the chances of preserving the spleen. The clinical features and reasons for performing a laparotomy are the same as for splenic rupture from non-endoscopic causes. The diagnosis requires a high index of suspicion. The occurrence of sudden abdominal pain, haemodynamic instability, or a drop in haematocrit in a post-ERCP patient should alert the clinician to the possibility of intra-abdominal bleeding including splenic injury. Another obstacle to early diagnosis is the variable time interval between ERCP and presentation of symptoms. One of the patients presented with a syncopal episode 2 days after discharge from hospital. This 'lag' varies from 20 minutes to 72 hours in the series. The diagnosis and management of our patient was straightforward. She presented with shock, abdominal pain, and a drop in her haematocrit after ERCP. Abdominal CT showed unambiguous features of active bleeding from the lacerated spleen and a splenectomy was necessary to control the haemorrhage.

Most patients recover fully after either splenectomy or conservative management. But this complication is potentially fatal; one patient died of multi-organ failure after splenectomy.10

In summary, post-ERCP splenic injury is a rare complication. Recognition of the condition requires a high index of suspicion and laparotomy is often needed for effective management of splenic injury after ERCP.

References