Bile peritonitis—a report of fifteen patients

HAROLD ELLIS
M.A., M.Ch., D.M., F.R.C.S.

H. M. ADAIR
M.B., F.R.C.S.

Surgical Unit, Westminster Hospital, London

Summary
Fifteen patients with bile peritonitis are presented. One followed a gunshot wound, six resulted from acute cholecystitis and nine were consequent upon biliary tract surgery. The aetiology, clinical picture and management are reviewed.

Introduction
In 1960 the senior author was concerned with a review of all cases of bile peritonitis encountered over a period of 20 years at the Radcliffe Infirmary, Oxford (Ellis and Cronin, 1960). In this group of twenty-two patients the aetiology was: traumatic rupture of a choledochal cyst; perforation of an acutely inflamed gall bladder; acute cholecystitis without perforation; perforation of a stenosed common bile duct; rupture of a cholangitic liver abscess; perforation of a normal gall bladder or of a common bile duct; leak from the common duct after T-tube removal; idiopathic. The mortality was 50%.

This paper presents fifteen consecutive patients with bile peritonitis encountered over the past 10 years at Westminster Hospital, and reviews the publications in this field since the previous report.

Review of present patients
The patients in this series are summarized in Table 1. The aetiological groups were:

Traumatic rupture of the gall bladder
The only patient in this group was one who suffered a gunshot wound of the abdomen. The gall bladder was perforated and there were injuries to the right colon and left lobe of the liver. The traumatized gall bladder was removed and the patient survived.

Acute cholecystitis
There were six patients with perforation of an acutely inflamed gall bladder. Stones were present in three of these. There was no past history of gall bladder symptoms in any of the patients. All were treated by cholecystectomy or cholecystostomy, except for patient 4 who was admitted with a 5-day history and found to be in renal failure. Patient 5 was complicated by stenosis of the ampulla of Vater. A neoplasm was thought to be present and the ampulla was excised. Histology showed an adenoma. In patient 6 the perforation was in the cystic duct rather than in the gall bladder itself and in patient 7, no site of perforation could be found. The source of bile peritonitis in this case was thought to be a transudation across the inflamed gall bladder wall.

Post-operative leaks
Our cases can be divided into three groups.

(1) From T-tube site. There were five such patients and in two of these, leakage occurred within 24 hr of removal of the T-tube. Prior cholangiography had been normal. One of these was treated by simple drainage of the abdomen, the other by additional reinsertion of the T-tube. One patient 10 suffered postoperative pancreatitis and this will be described more fully later. Another patient 11 leaked following exploration of the common bile duct; a residual stone was present. A drain was inserted through one end of the wound and he was later re-explored. The fifth patient 12 underwent exploration of the common bile duct, cholecystectomy having been done previously. The patient died on the seventeenth postoperative day. Free bile was found in the abdomen post mortem, but its source could not be determined.

(2) Leak after cholecystectomy alone. There was one such patient where leak occurred 1 week postoperatively. At laparotomy the site could not be found.

(3) Leak after choledochoduodenostomy. Two patients came into this category. Patient 14 had this performed for a pancreatic lesion, either chronic pancreatitis or carcinoma. The gall bladder had been removed, on a previous occasion, for carcinoma. This leak progressed to fistula formation and eventual death. A post mortem was not performed. The second patient 15 underwent cholecystostomy and exploration of the common bile duct. Choledochoduodenostomy was performed but breakdown occurred.
### Table 1. Summary of cases of bile peritonitis

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>Aetiology</th>
<th>Special features</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22</td>
<td>M</td>
<td>Gunshot wound of abdomen, gall bladder perforated</td>
<td>Also injuries to small bowel, right colon and left lobe of liver</td>
<td>Cholecystectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>2</td>
<td>74</td>
<td>F</td>
<td>Acute cholecystitis with perforation</td>
<td>No stones</td>
<td>Cholecystectomy</td>
<td>Survived</td>
</tr>
<tr>
<td>3</td>
<td>57</td>
<td>M</td>
<td>Acute cholecystitis with perforation</td>
<td>Stones present, jaundiced, ischaemic heart disease</td>
<td>Cholecystostomy</td>
<td>Survived</td>
</tr>
<tr>
<td>4</td>
<td>78</td>
<td>M</td>
<td>Acute cholecystitis with perforation</td>
<td>Stones present, jaundiced, renal failure, atrial fibrillation</td>
<td>Dialysis</td>
<td>Died</td>
</tr>
<tr>
<td>5</td>
<td>77</td>
<td>F</td>
<td>Acute cholecystitis with perforation</td>
<td>Stenosis of ampulla of Vater, steroid therapy for rheumatoid arthritis, ischaemic heart disease</td>
<td>Cholecystectomy, excision of ampulla</td>
<td>Died</td>
</tr>
<tr>
<td>6</td>
<td>86</td>
<td>F</td>
<td>Acute cholecystitis, perforated cystic duct</td>
<td>No stones, atrial fibrillation</td>
<td>Cholecystectomy, exploration of common bile duct</td>
<td>Survived</td>
</tr>
<tr>
<td>7</td>
<td>85</td>
<td>M</td>
<td>Acute cholecystitis</td>
<td>Stones present, mild diabetic, asthmatic</td>
<td>Cholecystostomy</td>
<td>Died</td>
</tr>
<tr>
<td>8</td>
<td>51</td>
<td>M</td>
<td>Cholecystectomy for stones, leak following T-tube removal</td>
<td></td>
<td>Drainage</td>
<td>Survived</td>
</tr>
<tr>
<td>9</td>
<td>32</td>
<td>F</td>
<td>Cholecystectomy for stones, leak following T-tube removal</td>
<td></td>
<td>Drainage, re-insertion of T-tube</td>
<td>Survived</td>
</tr>
<tr>
<td>10</td>
<td>45</td>
<td>M</td>
<td>Cholecystectomy for stones, leak following T-tube removal</td>
<td>Post-operative pancreatitis, T-tube left in situ for 1 month</td>
<td>Drainage</td>
<td>Survived</td>
</tr>
<tr>
<td>11</td>
<td>49</td>
<td>M</td>
<td>Leak following cholecystectomy and exploration of common bile duct with T-tube drainage</td>
<td>Residual stone in common bile duct.</td>
<td>Drainage, later re-exploration</td>
<td>Survived</td>
</tr>
<tr>
<td>12</td>
<td>72</td>
<td>M</td>
<td>Exploration of common bile duct with T-tube drainage</td>
<td>Giant gastric ulcer, resuture of burst abdomen</td>
<td></td>
<td>Died 17 days post-operatively with bile peritonitis, source not found</td>
</tr>
<tr>
<td>13</td>
<td>66</td>
<td>M</td>
<td>Leak 1 week after cholecystectomy</td>
<td>Ducts not explored, ischaemic heart disease</td>
<td>Drainage, site of leak not found</td>
<td>Survived</td>
</tr>
<tr>
<td>14</td>
<td>84</td>
<td>M</td>
<td>Leak after choledochoduodenostomy for pancreatic lesion either chronic pancreatitis or neoplasm</td>
<td>Developed duodenal fistula, abdominal dehiscence</td>
<td>Drainage</td>
<td>Died</td>
</tr>
<tr>
<td>15</td>
<td>51</td>
<td>F</td>
<td>Previous cholecystostomy for empyema of gall bladder, cholecystectomy, exploration of common bile duct, choledochoduodenostomy</td>
<td>Steroids for rheumatoid arthritis</td>
<td></td>
<td>Died, anastomosis broken down post mortem</td>
</tr>
</tbody>
</table>
Typical cases from groups 2 and 3 were as follows:

Acute cholecystitis with perforation—laparotomy—cholecystectomy

A 78-year-old housewife (case 2) was admitted as an emergency with a 12-hr history of right-sided abdominal pain and vomiting.

Examination revealed signs of peritonitis, maximum on the right side of the abdomen. She was not jaundiced. The temperature was 37-4°C and the pulse 76/min.

A diagnosis of peritonitis probably due to acute appendicitis was made. At operation a normal appendix was found but free bile was present in the peritoneal cavity. The gall bladder was oedematous with a 3 × 2 cm gangrenous area in the body. A small perforation was present in this region. The duct system was normal and no stones were found. Cholecystectomy was performed and recovery was uneventful. Histology confirmed acute cholecystitis.

Bile leak after T-tube removal—drainage

A 45-year-old civil servant (case 10) had a routine cholecystectomy for gall stones together with negative exploration of the common bile duct, on the basis of a suspicious operative cholangiogram.

A T-tube cholangiogram at 10 days showed narrowing of the lower part of the common bile duct compatible with acute pancreatitis. The T-tube was left in situ for a further 4 weeks after which a second X-ray showed great improvement in the abnormal appearances.

Removal of the T-tube was followed by abdominal pain and signs of peritonitis. Laparotomy revealed a bile leak from the T-tube site with free bile in the peritoneal cavity. The peritoneum was drained and recovery was uneventful.

Discussion

Aetiology

The patients so far reported fall into three categories: peritonitis secondary to acute cholecystitis, postoperative peritonitis, and other miscellaneous causes.

Rupture of the acutely inflamed gall bladder may occur into the peritoneal cavity or adjacent visera. The incidence of bile peritonitis following acute cholecystitis is variously reported as 10-60% (MacDonald, 1960), 4% (Essenhigh, 1968), and 5% (Leborgne et al., 1972). Abu-Dalu and Urea (1971) divide their cases of perforation into those before and those after 1960 and give figures of 0-1 % and 2-1 % respectively. In our series six cases occurred in approximately 110 admissions, i.e. an incidence of 5-5%. All authors agree that this complication is seen most often in elderly patients and the mean age of 75 years in our patients correlates with this (Boglia, Mapatesta and Misiti, 1969). Perforation occurred before admission in all our patients. The possibility of perforation is often put forward as an argument for immediate surgery in acute cholecystitis. Our figures do not support a general policy of immediate operative treatment based on the risk of rupture of the acutely inflamed organ alone. Postoperative leaks are relatively common and indeed may be present to some extent after all biliary tract surgery. Stern (1967) enumerates the causes as: leak from an accessory cystic duct; trauma to the duct system at operation; slipped cystic duct ligature; leak around a T-tube or after its removal; common duct obstruction. Trauma to the biliary tree is recorded by Means (1964) in ten of thirty-six patients with bile peritonitis. The hazards of T-tubes are stressed by Winstone et al. (1965) who warn against polyvinyl tubes which cause less tissue reaction than rubber tubes. The source of leak may be difficult to find (McKenzie, 1955) and it may occur weeks or months after initial surgery. It is often associated with residual stones.

Other miscellaneous causes of bile peritonitis are uncommon. Trauma is usually of the closed type due to road traffic accidents (McCarthy and Picazo, 1968; Means, 1964). Open injury resulting in perforation may occur (Grimsehl and Penner, 1964) and in these cases there are usually multiple visceral injuries, as in our own patient 1. It is important to remember bile leak as a complication of percutaneous liver biopsy. Madden (1961) records two such cases in a series of seventy.

Perforation of an apparently normal gall bladder or duct may occur. Theories of ulceration by stone, mucosal vascular accident, and mucosal infection have been put forward (Ellis and Cronin, 1960). Lees and Mitchell (1964) describe seventeen cases of duct perforation in infancy falling into this category. Six of these proved to have stenosis at the lower end of the common bile duct and three had stones present. The authors note the relatively constant position of the perforation at the junction of the cystic duct and common hepatic duct.

One case of perforated carcinoma of the gall bladder is described by Kotorac (1972). He claims this to be very rare, the disease usually pursuing a chronic course with internal fistula formation.

Finally, bile peritonitis may be idiopathic. Various theories have been put forward—ulceration by a tiny calculus, mucus gland rupture, a minute sealed perforation, retroperitoneal perforation with peritoneal transudation, and rupture of a small cholangitic liver abscess. Calculi may cause pressure necrosis and ulceration in the biliary system but there is no evidence that this happens with calculi so small as to leave no trace. Mucus glands in the gall bladder may become infected and one of these may rupture.
method of commencing. Failure.

Operation relatively slow leak of bile who patient presented ‘pre-operatively’ patients with acute pancreatitis and free intraperitoneal bile.

Clinical picture

Two types of clinical picture are described which can be correlated, to some extent, with the main aetiological groups mentioned.

Perforation complicating acute cholecystitis invariably presents acutely. This is borne out by our own cases where there was acute pain with signs of widespread peritonitis. Three patients were jaundiced: two were clinically shocked. This is in contrast to the cases described by Singh (1972) who quotes the syndrome of tachycardia, hypotension, abdominal distension, jaundice and signs at the right lung base. Had treatment been withheld from our cases, these florid signs might have developed. The often non-specific nature of the symptoms and signs, and the rarity of the condition make diagnosis difficult pre-operatively (MacDonald, 1966; Abu-Dalu and Urea, 1971; Essenhijgh, 1968).

Postoperative bile peritonitis usually presents with a similar clinical picture of acute pain and peritonism. This was so in the present series. However, one patient presented more insidiously with only mild pain and tenderness, but with abdominal distension and signs of free fluid. Rosato, Berkowitz and Roberts (1970) describe two similar indolent presentations. This seems to be the usual manner of presentation in infants (Borde and Cotoni, 1966; Lees and Mitchell, 1966) and may be due to the relatively slow leak of bile and the absence of infection.

Treatment

Patients presenting ‘pre-operatively’ with bile peritonitis should be treated surgically. This was the method of treatment in our cases, apart from one patient who was referred at a late stage with renal failure. Operation is preceded by resuscitation with intravenous fluids, and a broad spectrum antibiotic is commenced. The principles of surgery are evacuation of bile, drainage of the peritoneal cavity, and treatment of the biliary tract lesion (Leborgne et al., 1972). A diseased or injured gall bladder should be removed if this can be done safely. Alternatively, cholecystostomy is performed.

There are many postoperative cases where a small leak of bile occurs. Provided the area has been drained this is usually unimportant and the collection issues from the drain site. Should the patient have localized or generalized peritonitis, then laparotomy is indicated on the same principles as above.

Treatment is more likely to be successful if the diagnosis is made early and acted upon promptly.

Prognosis

Ellis and Cronin (1960) quote a mortality of 50% in their series. Various authors since then have given figures of 70% (MacDonald, 1966), 39% (Essenhijgh, 1968), 29% (McEarchern and Sullivan, 1963) and 25% (Leborgne et al., 1972). Means (1964) gives figures of 87-5% mortality in pre-operative generalized peritonitis and 11% for pre-operative localized peritonitis. His figures for the postoperative groups are 80% and 20% respectively.

The mortality in our group of patients was 40%. In our five deaths, one patient was admitted in renal failure, two patients had complicated courses with abdominal dehiscence and duodenal fistula, and two patients were under treatment with steroids.

Various theories have been put forward to explain the high mortality associated with this condition. Some authorities favour fluid imbalance, bacterial contamination and toxicity of bile salts as the important factors (Means, 1964; Conn, Chavez and Sain, 1970). Review of the publications suggests that the high mortality is related to age of the patient, length of history, and promptness of treatment. Our own experience would support the importance of those, together with the presence of intercurrent disease.

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References


