Acute gall bladder perforation – a dilemma in early diagnosis

C L Ong, T H Wong, A Rauff

Abstract
Gall bladder perforation is a rare complication of cholecystitis. A definitive diagnosis is uncommon before surgery and the morbidity and mortality associated with this condition are high. We report six patients with gall bladder perforation to show the difficulty of making an early diagnosis. The history and the clinical findings of these patients are reviewed to highlight diagnostic pitfalls.

In 1934, Neimeir presented his classic description of acute perforation of the gall bladder and proposed a classification based on his findings. He concluded that this rare condition ‘... demands eternal vigilance ...’ it would appear from our series that prompt recognition and treatment might lower this (mortality) significantly’. Half a century later and despite his recommendation this disease continues to be misdiagnosed and the mortality remains high.

Roslyn et al. reviewed the risk factors for gall bladder perforation. They found that patients aged over 60 years with acute perforation more commonly had associated severe systemic diseases such as diabetes and atherosclerotic heart disease. In this group perforated gall bladder was thought to be a result of a compromised blood supply. There was a second group of younger patients who were also at risk for acute perforations. Most of these patients were immunosuppressed for one reason or another and it was believed that the inability to combat infection played a major role in the perforation.

We reviewed the notes of all 487 patients who were admitted to this hospital for cholecystectomy between November 1987 and July 1989. Fifty-five were emergency admissions and 432 were elective cases. Six of the emergency cholecystectomies were done for acute gall bladder perforation and the others for empyemas and acute cholecystitis.

Patients and methods
The six patients with acute gall bladder perforations form the basis of this study. Each patient’s case notes were carefully examined and a detailed case history and the results of physical examination are presented below. The aim was to study the ways in which acute gall bladder perforation may present and to see if there are any pointers that may help in a correct preoperative diagnosis.

PATIENT 1
A 35 year old Malay man presented with a three day history of right sided abdominal pain. There had been no previous episodes. The pain was associated with nausea and vomiting. Physical examination showed considerable tenderness all over the right side of the abdomen with maximal pain in the right lower quadrant. He was afebrile. There was no free gas under the diaphragm and an abdominal x ray was reported as normal. A clinical diagnosis of acute appendicitis was made and he was operated on the same day. A Lanz incision was made and the appendix was found to be normal. It was noted through the limited incision, however, that there were adhesions in the gall bladder region. The appendectomy was completed, the incision was closed, and the patient was given prophylactic antibiotics. He was well in the immediate postoperative period. He became febrile and was especially tender in the right hypochondrial region on palpation. The presence of a calcified gall stone was picked up on review of the abdominal x ray. An urgent ultrasound showed an impacted gall stone at Hartmann’s pouch with a considerably distended gall bladder and there was localised fluid collection around the gall bladder which was thought to be caused by perforation. The patient was returned to theatre two days after the appendectomy and a cholecystectomy was performed. The perforated gall bladder had been sealed off by omentum.

PATIENT 2
A 27 year old Chinese woman presented with a five day history of epigastric pain which later shifted to the right hypochondrial region. This pain had become progressively severe and was associated with fever, chills, and rigors. She had a previous diagnosis of gall stones. Examination failed to show any symptoms of severe infection but she was in some distress from the pain. She had tenderness in the right hypochondrial region but palpation did not indicate any masses. A diagnosis of acute cholecystitis was made and she was treated with intravenous antibiotics. Ultrasound showed a thick walled gall bladder consistent with a diagnosis of empyema, with noticeably dilated intra- and extrahepatic ducts. She underwent surgery that day and a perforation in the body of the gall bladder was found. This was sealed off by omentum. The abscess cavity was filled with debris and pus. A choledochotomy was performed and a 0.5 cm stone impacted at the lower end of the common bile duct was removed.

PATIENT 3
This patient was a 29 year old Chinese woman who had systemic lupus erythematosus and was taking 7.5 mg of prednisolone per day. She
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presented with a one day history of right lower quadrant pain associated with fever, chills, and rigors but no other symptoms. There was no previous history of gall stone disease. On examination, she was found to have a temperature of 37.5°C. There was noticeable terness at McBurney’s point, with rebound. An abdominal and pelvic ultrasound was performed because there was some doubt about the diagnosis. Ultrasound showed a contracted but thick walled gall bladder with features suggestive of acute on chronic cholecystitis. There was no abscess collection in the pelvis. She underwent surgery the day after admission, at which time an adequate assessment of her medical condition had been made. The gall bladder was perforated in the body and it was sealed off by omentum, the transverse colon, and the duodenum. There were small sludge like stones in the gall bladder.

PATIENT 4
A 44 year old Chinese man presented with a one day history of acute severe epigastric pain. He had had previous episodes of jaundice 10 years earlier but was otherwise fit. Examination showed board-like rigidity in the upper abdomen with considerable tenderness. He had mild fever but no other signs. No free gas was seen under the diaphragm on the chest x ray. A clinical diagnosis of perforated duodenal ulcer was made. The patient was brought to theatre and a right paramedian incision was made. On entering the peritoneal cavity, a gangrenous gall bladder was seen with omentum stuck to its fundus where it had perforated. No stones were found in the gall bladder but there was sludge typical of oriental cholangiohepatitis. A cholecystectomy was performed.

PATIENT 5
A 72 year old Chinese woman presented with a one day history of right hypochondrial pain. There had been similar episodes in the past. She did not have any fever, chills, or rigors. On examination, there was very mild tenderness in the right hypochondrium without any evidence of guarding or rebound. A diagnosis of biliary colic was made and she was treated conservatively with intravenous antibiotics. One day after admission, however, her pain became more severe and more generalised. She was thought to have developed an empyema of the gall bladder and underwent surgery. Perforation of the gall bladder fundus with free bile in the peritoneal cavity was found and cholecystectomy was done.

Discussion
Two per cent of patients undergoing cholecystectomy are found to have perforation of the gall bladder.1 It occurs in 10% of patients who are being treated conservatively. In our study, there was a gall bladder perforation rate of 1·23%.

In 1942, Glenn2 reported a mortality rate of 42%, but more recent studies by Lennon and Green3 and Roslyn and Bussuttil4 have reported mortality to be between 12 and 16%. We had no deaths among our six patients. This would seem to be fortunate since in none of the six was a correct diagnosis made preoperatively. This can be attributed to the fact that all our patients were operated on early, and, more importantly, in five of the six the perforation was restricted to a limited area. Only one patient had free bile in the peritoneal cavity. Furthermore, unlike most studies in which the patients are generally aged 60 or more,6 four of our six patients were below the age of 45 years.

Three of our patients had previous symptoms associated with disorders of the hepatobiliary system. In this context, it is interesting that all six patients had fairly acute presentations. The main symptom was pain and in only one of them had the pain lasted longer than five days. Three had had the pain for only one day. This seems to

### Table 1 Summary of the patients presented

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (y)</th>
<th>Symptomatic diseases</th>
<th>Symptom duration (d)</th>
<th>Site of pain</th>
<th>Fever</th>
<th>Total white cell count (×10⁹/l)</th>
<th>Polymorphs (%)</th>
<th>Preoperative diagnosis</th>
<th>Complications</th>
<th>Duration of stay in hospital (dys)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>35</td>
<td>Nil</td>
<td>3</td>
<td>RIF</td>
<td>No</td>
<td>12·4</td>
<td>84</td>
<td>Acute appendix</td>
<td>Nil</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>27</td>
<td>Nil</td>
<td>3</td>
<td>RHC</td>
<td>Yes</td>
<td>9·3</td>
<td>80</td>
<td>Empyema gall bladder</td>
<td>Nil</td>
<td>9</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>29</td>
<td>SLE</td>
<td>1</td>
<td>RIF</td>
<td>Yes</td>
<td>12·7</td>
<td>86</td>
<td>Acute cholecystitis</td>
<td>Nil</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>44</td>
<td>Nil</td>
<td>1</td>
<td>Epi</td>
<td>Yes</td>
<td>17·5</td>
<td>94</td>
<td>Perforated duodenal ulcer</td>
<td>Wound infection</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>72</td>
<td>Nil</td>
<td>1</td>
<td>RHC</td>
<td>No</td>
<td>8·6</td>
<td>95</td>
<td>Biliary colic</td>
<td>Nil</td>
<td>11</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>75</td>
<td>Toxic goitre</td>
<td>4</td>
<td>RIF</td>
<td>Yes</td>
<td>10·2</td>
<td>80</td>
<td>Perforated appendix</td>
<td>PAT, AF</td>
<td>14</td>
</tr>
</tbody>
</table>

RIF = right iliac fossa; RHC = right hypochondrium; Epi = epigastrium; PAT = paroxysmal atrial tachycardia; AF = atrial fibrillation.
correlate well with the type of perforation that we saw in this series. Five were type II and only one was type I. There were no type III perforations. Most other series also showed that type II perforations were the most common (Table II). Type III perforation with fistulation to adjacent organs would obviously be associated with long standing symptoms as the fistula require a considerable amount of time to form.

The mean age of our patients was 47 years. This is much younger than the mean age in most series, which is about 65. This observation seems to support the findings of Roslyn et al. that types I and II perforations occur more commonly in the younger people, especially men below 50 years of age.

Only one of our patients was immunosuppressed. It seems that apparently healthy young people are also susceptible to gall bladder perforation. Three of them were being treated for what was initially thought to be acute uncomplicated cholecystitis.

Isch et al. showed that the clinical manifestations of acute gall bladder perforations were similar to those of acute cholecystitis and they felt this could account for the delay in the diagnosis. Our data, however, show that the site of maximal pain in these patients can often be sufficiently misleading to cause the clinician to make diagnoses such as acute appendicitis and perforated duodenal ulcer. The site of maximal pain was in the right lower quadrant in three of the patients and epigastric in one. In the other patients, in whom the pain was in the right hypochondrial region, the clinical picture was that of acute cholecystitis or biliary colic. The patient may look sufficiently well and may not seem to be infected so that the perforation is completely unsuspected.

Although most of the patients had omentum stuck to the site of perforation and the gall bladder was reported to be distended, no gall bladder mass was documented in any.

The total white cell count may not be raised at all in these patients. Three of them had counts less than 11 (the upper limit of normal in our hospital). All of them, however, had raised polymorph percentages, indicating an acute bacterial infection.

Liver function tests were abnormal in three of the patients and in these consisted of non-specific increases in liver enzyme activities. This finding did not help in differentiating between gall bladder perforation and simple acute cholecystitis just as it did not assist in differentiating between empyema and acute cholecystitis.

The ultrasound scan was not helpful either. Ultrasound was used in three patients and suggested acute cholecystitis in two and empyema in one.

The best form of management is early surgery, this would concur with the findings of those of Larmi et al., Addison and Finan advocated early and urgent cholecystectomy for acute gall bladder disease. They reported the risk of perforation to be between 3 and 12% in patients treated conservatively for acute cholecystitis. They also showed that the mortality and morbidity for emergency cholecystectomies compared favourably with those for elective surgery and concluded that in well selected patients, emergency cholecystectomy for acute cholecystitis should be advocated as a safe procedure.

We agree with this view.

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