

## IDIOPATHIC PERFORATION OF THE GALLBLADDER

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Gallbladder perforation is an infrequent but potentially fatal disease. It is usually a complication of acute cholecystitis with or without gallstones and has rarely been reported in association with trauma, neoplasia, chemotherapy, vascular disease or pregnancy.<sup>1-6</sup> However, idiopathic perforation of the gallbladder without any demonstrable cause is an extremely rare occurrence,<sup>7</sup> and is the subject of this report.

### Case Report

A previously healthy 30-year-old Nepalese man presented to the emergency department of Dar Al-Shefa Hospital, Riyadh, as a result of two days' history of severe pain in the epigastrium, right hypochondrium and right lumbar region. The patient vomited once and had no urinary trouble. There was no history of trauma or drug abuse. His vital signs were as follows: pulse 99/min; blood pressure 140/100 mm Hg; respiration 24/min; and temperature 37.8°C. Physical examination revealed tenderness with rebound pain in the epigastrium, right upper quadrant and right lumbar region. Bowel sounds were faint, and no significant distention of the abdomen was noted. Laboratory studies revealed white blood cell count to be 18,500/mm<sup>3</sup> with predominance of neutrophils; hemoglobin 16 g/dL; hematocrit 43.8% and platelets 311,000/mm<sup>3</sup>; SGPT 75 U/L (normal up to 42); SGOT 84 U/L (normal up to 37); alkaline phosphatase 166 U/L (normal up to 306); serum amylase 41 U/L (normal up to 90); BUN 11 mg/dL (normal 9-20); Na<sup>+</sup> 138 mmol/L (normal 135-155); and K<sup>+</sup> 3.5 mmol/L (normal 3.5-5.5). The Widal test, Sickling test and blood cultures were all negative. Urine analysis revealed no significant abnormality. The flat and upright abdominal radiographs were interpreted as suggestive of an adynamic ileus pattern and revealed no gas under the diaphragm. Ultrasonographic examination revealed a slight thickening of the gallbladder wall but no stones or distention. A small amount of free fluid was observed in the pelvis. With a provisional diagnosis of peritonitis secondary to perforated duodenal ulcer, acute cholecystitis or sub-hepatic retrocecal



FIGURE 1. Gross photograph showing a small perforation in the body of the gallbladder.

appendicitis, an emergency laparotomy was performed using a midline vertical incision about 12 hours after the admission and after initial hydration. The abdominal viscera were stained yellowish green. Bile was seen leaking through a small hole in the body of the gallbladder near the fundus. The gallbladder wall was slightly thickened, and it was neither distended nor inflamed. Other abdominal viscera were unremarkable. A small amount of bile-stained fluid was noted in the right paracolic gutter and pelvis. Bile culture was obtained and cholecystectomy was performed.

Pathological examination of the gallbladder confirmed the presence of a perforation (Figure 1). Microscopic examination of the gallbladder showed localized necrosis (Figure 2) with minimal inflammation. No vascular occlusion or inflammation was noted. A mild-to-moderate degree of arteriosclerotic changes were noted in the arterioles of the gallbladder wall (Figure 3). There were no stones in the gallbladder and the bile culture was negative. A diagnosis of idiopathic perforation of the gallbladder was established. The postoperative recovery was uneventful and the patient went home on the seventh postoperative day. He was symptom-free at the last follow-up visit six weeks after the surgery.

### Discussion

Perforation of the gallbladder occurs in 3%-10% of cases of acute cholecystitis, and is usually associated with the

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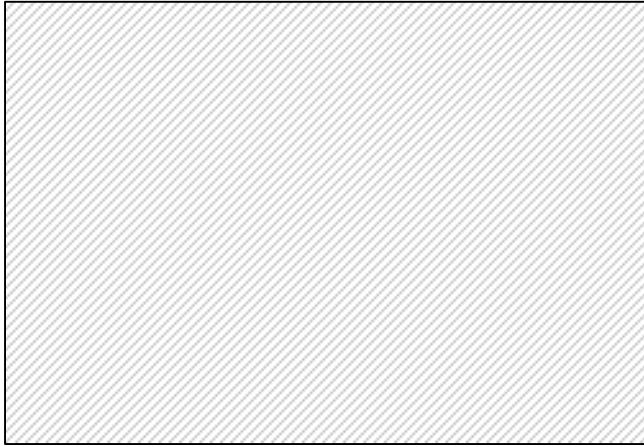


FIGURE 2. Microphotograph showing localized necrosis of the gallbladder wall at the site of perforation.

presence of stones. The mortality rate is in the range of 12%-16%.<sup>6</sup> It is believed that the initial inflammation is chemically induced. Apparently, a concentration of bile due to outflow obstruction by a stone acts as an irritant, leading to inflammation. Secondary bacterial infection may then occur. The organisms are thought to reach the gallbladder via the lymphatics, and are most commonly *Escherichia coli* and *Streptococcus faecalis*. In rare cases, acute cholecystitis may occur without stones, and is then usually associated with a source of infection elsewhere.<sup>8</sup> Mucosal inflammation and ischemia may progress to necrosis and eventual perforation.

Gallbladder perforation without any apparent cause is thought to be very rare,<sup>9</sup> and presents a difficult problem. Patients are usually elderly, with right upper abdominal pain usually of less than three days' duration. The usual signs are right upper abdominal tenderness, guarding, fever, and leukocytosis. The gallbladder may or may not be palpable, but it is usually difficult to ascertain.

Our patient was relatively young and had no fever. Elevated serum amylase and alkaline phosphatase have been reported in some cases of gallbladder perforation, however, they were normal in our case. Various imaging procedures (e.g., ultrasonography and CT scan), along with a high index of suspicion are helpful in obtaining a diagnosis.<sup>10</sup> However, laparotomy usually plays a decisive role in the diagnosis as well as management, as it did in our case. Early diagnosis of gallbladder perforation is important for the treatment and prognosis. Delay in surgical intervention is the major reason for increased morbidity and mortality associated with gallbladder perforation.<sup>11</sup> Fortunately in our case, the decision to perform laparotomy was made early on and probably played a major role in the favorable outcome. In one review, a correct diagnosis was established preoperatively in only one of the nine (11.1%) patients.<sup>7</sup> The fundus is the most common site of perforation, probably because it is the most distal part with regards to blood supply.



FIGURE 3. Microphotograph showing moderate arteriosclerotic changes in the arterioles of the gallbladder wall.

Furthermore, some systemic diseases, such as atherosclerotic heart disease, may induce ischemia of the gallbladder wall, leading to necrosis and perforation. Our patient had no known history of heart disease, but it is interesting that moderate atherosclerotic changes were noted in the gallbladder wall arterioles. Whether this played any role in the causation of this perforation is not clear. This case helps to emphasize the importance of considering this disease and the need for early laparotomy in patients presenting with peritonitis of unknown etiology.

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