Spontaneous Erosion of a Lost Intra-Abdominal Gallstone Through the Back Eight Months Following Laparoscopic Cholecystectomy

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ABSTRACT

Background and Objectives: Gallbladder perforation during laparoscopic cholecystectomy with spillage of bile and gallstones occurs in up to 40% of patients. Several reports have recently been published describing complications related to these lost gallstones. The purpose of this study was to determine the incidence of this complication in our patients.

Methods: A prospectively maintained database of 856 laparoscopic cholecystectomies performed between 1989 and 1996 by a single surgeon was analyzed.

Results: The number of perforations resulting in loss of stones in the abdominal cavity was 16% (165 patients). Of these 165 patients, only a single patient could be identified as having a long-term complication.

Conclusions: Intra-abdominal lost gallstones can produce complications secondary to migration and erosion. It is prudent to make a concerted effort to remove spilled gallstones by every possible means but conversion to laparotomy is not justifiable.

Key Words: Cholecystectomy, Laparoscopic, Laparoscopic adverse effects, Cholelithiasis surgery, Male, Human, Case, Report, Abscess etiology, Foreign bodies, Postoperative complications

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INTRODUCTION

Gallbladder perforation during laparoscopic cholecystectomy with spillage of bile and gallstones occurs in a substantial number of patients (up to 40%).[15,21] Most surgeons believe that free intraperitoneal stones are not a justification for conversion to laparotomy even if a large number of stones are left in situ. There are, however, a number of reports demonstrating that, on occasion, these stones may cause infection or abscess, inflammation, fibrosis, adhesions, cutaneous sinuses, small bowel obstruction, or generalized septicemia.[3-5,8,17-19,22,24,25]

One of our patients suffered a late sequelae of a retained intra-abdominal gallstone which spontaneously eroded through the patient's back eight months following laparoscopic cholecystectomy. The purpose of this study was to review our series of laparoscopic cholecystectomy to determine the incidence of long-term complications of lost gallstones in our patients.

CASE REPORT

A prospectively maintained database of 856 laparoscopic cholecystectomies performed between 1989 and 1996 by a single surgeon (RJF) was analyzed. Gallbladder perforations resulting in loss of stones in the abdominal cavity occurred in 16% (165 patients). Of these 165 patients, only a single patient could be identified as having a long-term complication. This was a 63-year-old Caucasian male who presented to us with an acute episode of right upper quadrant (RUQ) abdominal pain of approximately six hours duration. The patient stated that the pain was constant and associated with nausea and vomiting. The patient suffered 2-3 attacks of similar abdominal pain in the past.

Past medical history was significant for a hiatus hernia, with gastroesophageal reflux disease and esophageal stricture for which he underwent regular dilatations by his gastroenterologist. He also suffered from peptic ulcer disease for which he was treated by oral ranitidine 150 mg twice a day. Furthermore, he was taking oral Azulfidine 500 mg four times a day for suspected inflammatory bowel disease the

nature of which was indeterminate. He also suffered from pericarditis in 1979 for which he was treated with steroids. Past surgical history included a hemorrhoidectomy, but no abdominal surgery.

Physical examination revealed a well nourished male in no acute distress. Vital signs were within normal limits. Abdominal examination was within normal limits except for some tenderness in the RUQ which was not associated with rebound or guarding.

Preoperative laboratory evaluations revealed a hemoglobin and a hematocrit of 16.9 (14-18) gm/dl and 48.9 (40-54)% respectively, white blood cell count of 15.9 (4-11) k/μl, platelets 270 (150-400) k/μl, sodium 139 (135-145) mmol/l, chloride 103 (98-107) mmol/l, potassium 5.0 (3.5-5) mmol/l, CO_2 28 (22-31) mmol/l, blood urea nitrogen and creatinine of 11.7 (9-21) mg/dl and 1.6 (0.8-1.5) mg/dl respectively, and blood glucose of 126 (65-110) mg/dl. Urinalysis disclosed a specific gravity of 1.27, positive ketones, negative white blood cell esterase, some mucus and +1 white blood cells. The radiologist opinion of the plain film of the abdomen was that of a normal bowel gas pattern and no gross abnormality. Abdominal ultrasound revealed cholelithiasis with several medium-sized stones but no dilatation of biliary ducts. Dilatation of the large bowel with edematous bowel walls was also noted.

The patient underwent laparoscopic cholecystectomy under general anesthesia. Prophylactic antibiotics were administered at the time of induction of anesthesia. An open laparoscopy was performed and a Hasson cannula was introduced infraumbilically. A pneumoperitoneum was created and a 10 mm laparoscope with attached video camera was introduced into the abdomen. Generalized exploration of the abdomen disclosed that the small bowel was dilated more than usual but no visible organic cause such as stricture or tumor in the small bowel were noted. This was felt to be consistent with his diagnosis of indeterminate inflammatory bowel disease. The gallbladder was markedly distended. Following this initial inspection, three additional cannulas were placed in the abdomen. An 11 mm cannula was placed in the upper midline and two 5 mm cannulas were placed along the right costal margin. The cystic duct was traced down to its junction with the common bile duct. The gallbladder was very friable and was torn on numerous occasions during mobilization from the hepatic bed. This resulted in spillage of bile and gallstones intraperitoneally. Most of these stones were recovered using mechanical means and a large bore irrigation-suction device. However, because of the number of stones involved in the spill, it was not reasonable to assume that all stones had been retrieved. An operative laparoscopic transcystic cholangiogram was obtained which was entirely normal. The cystic duct and artery were doubly clipped and divided. The gallbladder was dissected from its liver bed and removed via the umbilical port. The abdomen was then irrigated with saline and inspected for hemostasis prior to release of the pneumoperitoneum and removal of the cannulas. All skin incisions were closed subcuticularly using plain catgut and Steri-Strips and Bupivacaine was injected as local anesthesia. The patient tolerated the procedure well and left the operating room in stable condition. Estimated blood loss was 150 cc. The pathological evaluation of the gallbladder revealed chronic cholecystitis with cholelithiasis but no evidence of malignancy.

The patient's postoperative recovery was complicated by a pseudo-obstruction of the small and large intestine which resolved gradually over the next five days with conservative management. Plain films of the abdomen failed to show an acute process in the immediate postoperative period which could account for the patient's ileus. Subsequent abdominal films continued to show resolution of the pseudo-obstruction. Postoperative laboratory evaluation during this period revealed a white cell count of 7 k/μl, hemoglobin/hematocrit of 10.8 g/dl/31.1%, platelets 152 k/μl, CO_2 of 23 mmol/l, Chloride 98 mmol/l, sodium 134 mmol/l, potassium 3.6 mmol/l, glucose 122 mmol/l, blood urea nitrogen and creatinine of 8 mg/dl and 1.2 mg/dl respectively. These slightly abnormal electrolyte values were thought to be secondary to the overzealous intravenous rehydration therapy which also may have had a dilutional effect on the patients hemoglobin and hematocrit. Eventually the patient was started on a regular diet on the 5th postoperative day and was discharged home the next day on oral Azulfidine 500 mg qid, oral Colace 100 mg tid, and Pepcid 20 mg qid. He was seen in the outpatient clinic two weeks later with no obvious complaints.

Two months after his laparoscopic cholecystectomy, the patient presented with complaints of intermittent pyrexia and abdominal pain. Physical examination was within normal limits except for an elevated temperature of 102.4° and a large tender lump in the right axilla. Laboratory evaluation at the time revealed a hemoglobin of 11.3 gm/dl, hematocrit of 34.1%, platelet count of 261 k/μl and white cell count of 5.4 k/μl with differential of 83% granulocytes (45-80%), 10% lymphocytes (15-40) and 6% monocytes (2-11%). Erythrocyte sedimentation rate was elevated at 70 mm/hr. Routine coagulation screening, urea and electrolytes, creatinine, total protein, albumin, calcium, phosphate, cholesterol and uric acid were all within normal limits. Liver function tests revealed normal bilirubin and lactic

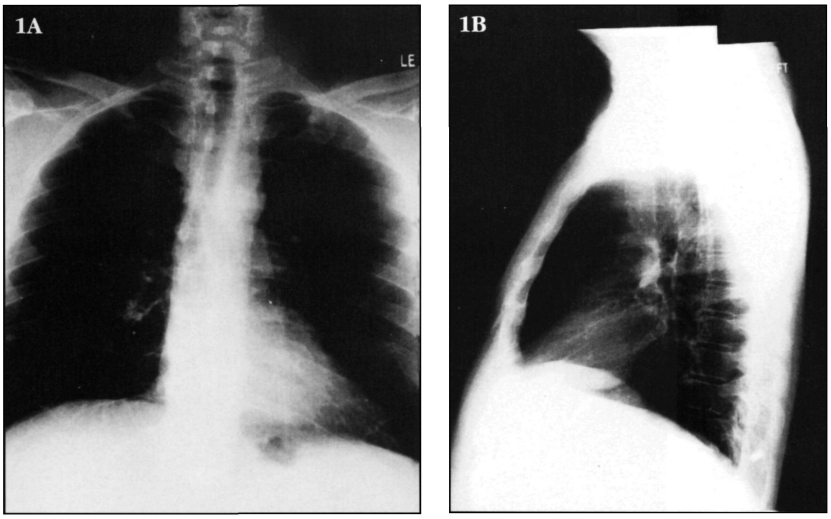

Figure 1 A & B. Postero-anterior and lateral chest films showing no active pathology expect for granulomatous disease on the right side.

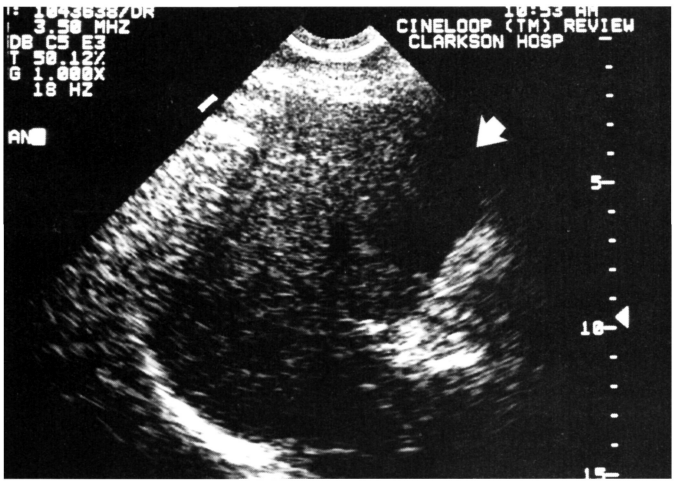

Figure 2. Ultrasound scan of the abdomen raising the suspicion of a poorly defined fluid collection in the area of the gallbladder fossa (arrow).

dehydrogenase but slightly raised alkaline phosphatase, AST and GGT. Amylase was also slightly elevated. Blood cultures were negative. Postero-anterior and lateral chest films revealed no active disease expect for granulomatous disease on the right side **(Figure 1 A & B)**. The patient underwent an ultrasound scan of the whole abdomen which raised the suspicion of a poorly defined fluid collection in the area of the gallbladder fossa measuring 3 x 4 x 4 cm **(Figure 2)**. The possibility of an abscess was entertained. However, computerized tomography (CT) scan of the abdomen with contrast failed to substantiate the findings of ultrasonography as no fluid collection was seen **(Figure 3)**. As the patient continued to spike a temperature, a technetium-99m HMPAO white cell scintigraphy was arranged which did not demonstrate any evidence of focal inflammation or infection **(Figure 4)** which could account for his pyrexia. In view of these negative findings the patient was started on a course of antibiotics, erythromycin 500 mg qid. This failed to resolve his pyrexia after seven days of treatment and therefore he was switched to cefuroxime 250 mg bd for a further three days but without any improvement. He was then given Augmentin 500 mg tid for two weeks which led to the gradual return of the temperature to normal. Repeat liver function tests and amylase showed normal values. It was concluded that the patient's pyrexia may have been secondary to inflammation of the RUQ due to a hematoma or other low grade inflammatory process. Following the normalization of his temperature and laboratory work-up, the patient was discharged back to his family practitioner. At the time of discharge the right axillary node had decreased quite significantly and was non-tender. It was speculated that this enlarged node represented reactive phenomenon related to his pyrexia of unknown origin.

After discharge, the patient continued to experience intermittent low-grade pyrexia and abdominal pain. A repeat CT scan of the abdomen two months later revealed changes around the gallbladder bed indicating an inflammatory process although no definite fluid collection was noted **(Figure 5)**. He was once again treated conservatively with oral antibiotics, Augmentin 500 mg tid with improvement in his temperature, albeit temporarily.

Eight months following his laparoscopic cholecystectomy, the patient noticed a painful mass on the right side of his back. This mass initially did not bother him and or interfere with sporting activities such as golf. However, some

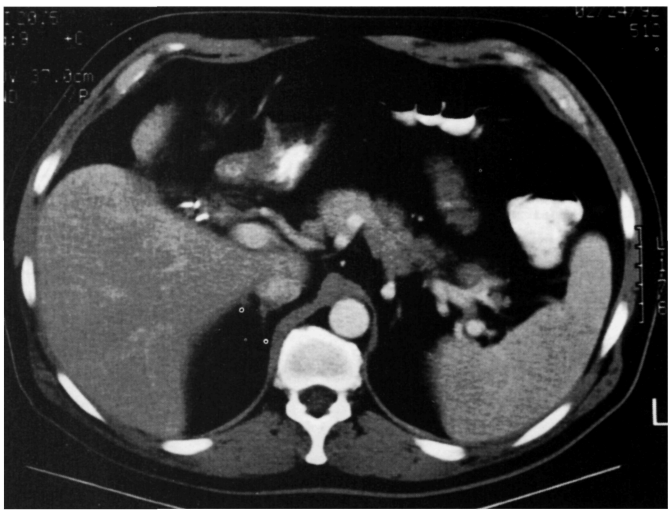

Figure 3. Computerized tomography (CT) scan of the abdomen with contrast failing to demonstrate any fluid collection in the area of gallbladder fossa.

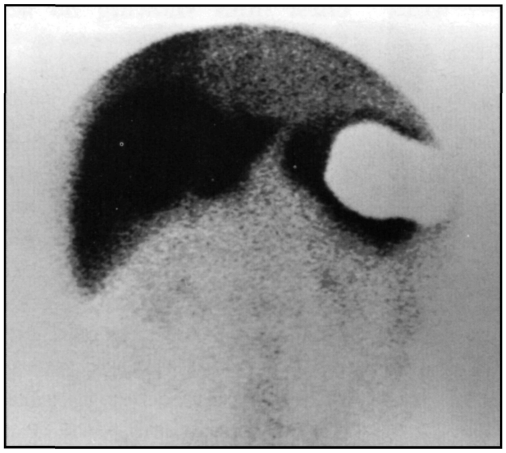

Figure 4. Technetium-99m HMPAO white cell scintigram showing no evidence of focal inflammation or infection.

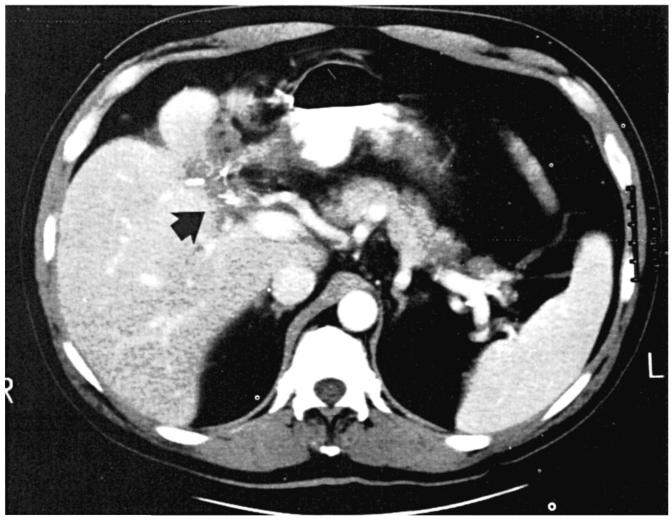

Figure 5. Repeat CT scan of the abdomen two months later showing changes around the gallbladder bed indicating an inflammatory type process (arrow) although no definite fluid collection can be seen.

days later he noticed increasing pain at the side of his lesion associated with elevation of temperature. Physical examination revealed a fluctuant swelling in the right flank. Incision and drainage of this area revealed an abscess cavity containing a small foreign body. Pathologic analysis revealed the foreign body to be a mixed pigmented calculus consistent with a gallstone surrounded by granulation tissue. The patient made an uneventful postoperative recovery and is now completely asymptomatic.

The patient was mailed a questionnaire as a part of the department of surgery's routine long term follow-up program. He displayed unhappiness by stating that "the small gallstone left in his body cost him eight months of his life and that he would not recommend laparoscopic cholecystectomy to a friend or family member."

DISCUSSION

Laparoscopic cholecystectomy has become the gold standard for the treatment of gallstone disease. Although spillage of both bile and stones occurs frequently during this procedure, most surgeons do not feel that this is a reason to convert to a laparotomy. Our data would support this practice, since only one related complication has been identified in 165 patients (0.6%). However, an increasing number of case reports in the literature have produced compelling evidence that retained/lost gallstones are not completely innocuous and can lead to serious complications.[1-14,17-20,22-28] It is therefore essential that every effort be made to recover all spilled stones at the time of laparoscopic cholecystectomy. This can be a laborious process, especially if the stones are numerous, small, and fall between loops of bowel. The large and medium-sized stones can be retrieved individually by mechanical means such as forceps, or can be collected in a plastic retrieval bag intracorporeally and removed en masse. In cases of multiple small stones, a wide bore suction-irrigation device is extremely helpful but not completely successful. It is also important that an effort be made to close holes in the gallbladder during dissection using either clips or sutures to minimize further spillage of bile and stones.

Although the natural history of spilled intra-abdominal gallstones in humans is unknown, the potential for complications is real. Our case highlights the fact that intra-abdominal lost gallstones can produce complications in the future due to migration and erosion. This can be a source of long-term morbidity for the patient and a diagnostic dilemma for the surgeon.[2,16,23] Every effort, therefore, should be made to rule out stone-related delayed complications in patients presenting with vague symptoms several months following laparoscopic cholecystectomy. It is prudent to make a concerted effort to remove spilled gallstones when performing a laparoscopic cholecystectomy by every possible means short of converting to a laparotomy.

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