Type 1 free perforation of the gallbladder

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Summary: A series of 4 cases of free perforation of the gallbladder into the peritoneal cavity associated with peritonitis are reported. Two were diagnosed at laparotomy and 2 at post-mortem. The 3 patients who died were men who were either elderly or had serious concomitant diseases, including diabetes, atherosclerosis or alcoholism. The sole survivor was a fit young woman.

Introduction

Perforation of the gallbladder, particularly in association with stones, is a well recognized entity found in up to 2% of patients undergoing cholecystectomy. Usually the perforation is localized. Free perforation into the peritoneal cavity is distinctly uncommon. It usually presents as an abdominal emergency and is only diagnosed at laparotomy. Occasionally the presentation may be chronic and may be overlooked as conventional diagnostic techniques are of little help. For these reasons the mortality is usually high. We report 4 cases who were only diagnosed at surgery or post-mortem and who illustrate some of the difficulties in successful management.

Case reports

Case 1: A 38-year-old woman presented with a six-month history of intermittent upper abdominal pain which for 14 hours prior to her admission had been severe and associated with nausea and vomiting. She had abdominal guarding and an absence of bowel sounds. The serum amylase was 140 iu/l. Despite normal radiological examination of the abdomen and chest a diagnosis of perforated duodenal ulcer was made and an emergency laparotomy performed. An acutely inflamed and perforated gallbladder with a stone in Hartmann's pouch was found in association with generalized peritonitis. Cholecystectomy was performed. A wound swab from the peritoneum grew *E. coli* and she was treated with cefuroxime. Subsequent progress was uneventful and she was discharged home after 11 days.

Case 2: A 78-year-old man, who had been unwell for two weeks prior to admission, collapsed an hour before his admission with diffuse abdominal pain. He had no bowel sounds and a chest X-ray showed consolidation of the right lower lobe. There were multiple fluid levels on the abdominal films. The serum amylase was 210 iu/l. He was resuscitated with intravenous fluids and treated with antibiotics. Laparotomy 24 hours later revealed a ruptured gallbladder containing many stones. There was more than 500 ml of bile in the peritoneal cavity, which grew *E. coli* on culture. The *E. coli* was sensitive to cephalosporins. Postoperative progress was complicated by renal failure and hypotension, and 9 days after admission he died from a pulmonary embolus which was confirmed at post-mortem examination.

Case 3: A 78-year-old man was admitted with a diagnosis of acute cholecystitis with a perforated gallbladder. The patient had had right upper quadrant pain for a week and on admission was unconscious and hypotensive. He had abdominal guarding, absent bowel sounds and neck stiffness. He had maturity onset diabetes for five years and was receiving

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Table 1. Mortality from gall bladder perforation in England and Wales, 1980–1982

	All deaths ($\times 10^3$)		Gall bladder perforations●	
Year	Male	Female	Male	Female
1980	292	290	5	9
1981	290	289	7	16
1982	290	292	18	17
Total	872	871	30	42

●Deaths from perforation of the gallbladder include all cases reported to the Registrar General, including those in whom the disease was not the primary cause of death

treatment with tolbutamide 1 g daily. Twenty years prior to admission he had had a subarachnoid haemorrhage and three years before a right-sided stroke, but he had made a good recovery from both of these cerebrovascular incidents. His blood glucose on this final admission was 38.5 mmol/l and he had ketonuria. The arterial pH was 7.14, and serum amylase 1800 iu/l. Initial resuscitation was directed towards correcting his acidosis, but death occurred within sixteen hours of admission. At post-mortem there was 200 ml of free bile in the peritoneal cavity and the gallbladder, which contained 5 stones, was perforated in the fundus.

Case 4: A 56-year-old man was referred because of a four-week history of backache associated with night sweats, anorexia and weight loss. He had 12-year history of hypertension treated with atenolol 100 mg daily and a two-year history of alcoholic liver disease. On admission he had a low-grade pyrexia (38°C) with a soft non-tender abdomen. There was loss of lumbar lordosis and extreme limitation of lumbar movement. Spinal radiology suggested osteomyelitis of L3 and L4. Bone biopsy confirmed this diagnosis and *E. coli* was cultured from the sample. The source of this infection was sought with abdominal ultrasound which demonstrated no abnormality apart from a single gallstone in the gallbladder. Intravenous urography was normal. During the subsequent 14 days he remained septicaemic and confused, with a leukocytosis of $24\,000 \times 10^9$/l which failed to respond to gentamicin, ampicillin, cefotaxime and metronidazole. A peritoneal tap showed green turbid fluid with an amylase content of 17 000 iu/l, and a diagnosis of acute pancreatitis was made. He died 26 days after admission. At post-mortem a necrotic gallbladder was found to be associated with an abscess cavity which communicated directly with the peritoneal cavity. The peritoneal cavity contained 4.5 litres of green fluid.

Discussion

Perforation of the gallbladder is a rare condition which in 3 of the 4 cases reported here was fatal. Between 1980 and 1982 it was responsible for 72 of the 1 700 000 deaths in England and Wales (Table 1); the male to female ratio was 1:1·4 and most patients were aged over 60 (Figure 1). Of the 4 patients seen in the Queen's Medical Centre, Nottingham, the 3 fatalities were all men, 2 of whom were elderly (78 years), and the younger man (aged 56 years) had coincident hypertension and alcoholism. The young woman who survived the perforation had no associated diseases and was relatively well on admission. In 3 of the 4 cases the perforation was associated with an *E. coli* infection.

Recent interest in this topic has been limited. A classification for gallbladder perforation was first proposed as long ago as 1934 by Niemeier who suggested three groups: Type 1 – acute free perforation into the peritoneal cavity; Type 2 – subacute localized perforation with abscess formation; Type 3 – chronic perforation into an adjacent hollow viscus or on to the skin.

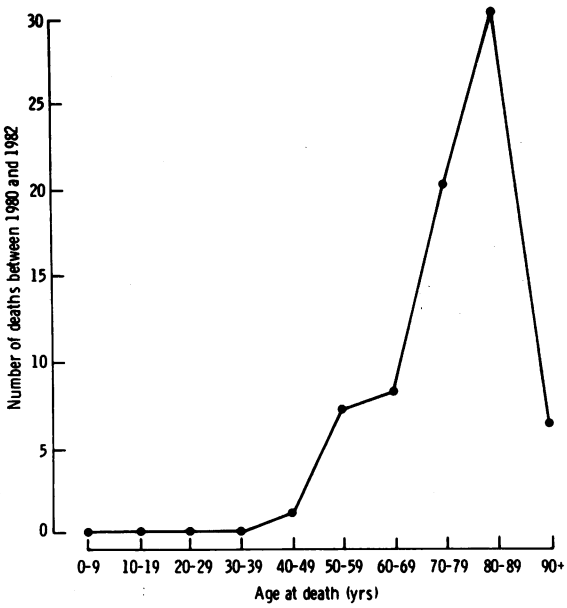

Figure 1. Age at death of patients with a perforated gall bladder in England and Wales between 1980 and 1982 (Source: Registrar General 1980–82)

One of the largest series from Pittsburgh reported 113 non-traumatic perforations in 3260 cholecystectomies performed over 20 years; the most common problem was a subacute localized abscess (Type 2), whereas only 11 cases of acute free perforation into the peritoneum were reported (Smith 1981). There was no mortality (see Table 2) but the series was highly selected and contained only cases admitted for either routine or acute cholecystectomy. In other series mortality has ranged from 8% to as high as 67% (Table 2).

In general patients have been elderly (Figure 1) and many reports have stressed the association with other diseases, in particular diabetes and atheroma. In 1979 Zupunski reviewed 14 deaths from gallbladder perforation and found that 11 had diabetes. Strohl *et al.* (1962) suggested that the mechanism of perforation depended upon atheroma in the vessels supplying the gallbladder, which may explain the association with diabetes.

Few centres have extensive experience with free perforation of the gallbladder. The diagnosis is seldom made until surgical exploration has been undertaken. Death may be due to endotoxic shock or associated disease such as diabetes. At present there is little prospect of

Table 2. Published reports of Type 1 free perforations of the gallbladder (cases identified at emergency or routine cholecystectomy)

Author	Country	No. of cases	No. of deaths	Mortality (%)
Lennon & Green 1983	England	12	1	8%
Smith 1981	USA	11	0	0%
Paul 1980	USA	8	2	25%
Roslyn & Busuttill 1979	USA	6	3	50%
Williams and Scobie 1976	Canada	3	1	33%
Isch *et al.* 1971	USA	9	6	67%
Abu-Dalu & Urca 1971	Israel	23	2	9%
McCubbrey & Thieme 1960	USA	3	1	33%
Gallagher 1960	USA	4	2	50%
Morse *et al.* 1957	USA	10	6	60%
Fletcher & Ravdin 1951	USA	5	2	40%
Cowley & Harkins 1943	World review	99	30	30%

improvement in techniques of diagnosis and management but it is to be hoped that physicians and surgeons will become more aware that the condition does not necessarily present as an acute abdomen.

References

Abu-Dalu J & Urca I (1971) *Archives of Surgery* **102,** 108–110
Cowley, L L & Harkins H N (1943) *Surgery, Gynaecology and Obstetrics* **77,** 661–668
Fletcher A G & Ravdin I S (1951) *American Journal of Surgery* **81,** 178–185
Gallagher W B (1960) *American Journal of Surgery* **100,** 407–411
Isch J H, Finneran J C & Nahrwold D L (1971) *American Journal of Gastroentology* **55,** 451–458
Lennon F & Green W E R (1983) *Journal of the Royal College of Surgeons of Edinburgh* **28,** 169–173
McCubbrey D & Thieme E T (1960) *Archives of Surgery* **80,** 204–207
Morse L, Krynski B & Wright A R (1951) *American Journal of Surgery* **81,** 178–185
Niemeier O W (1934) *Annals of Surgery* **99,** 922–924
Paul G J (1980) *Journal of the Medical Society of New Jersey* **77,** 97–99
Registrar General's Statistical Review of England and Wales (1980–82) HMSO, London
Roslyn J & Busuttill R W (1979) *American Journal of Surgery* **137,** 307–312.
Smith E B (1981) *Journal of the National Medical Association* **73,** 333–335
Strohl E L, Diffenbach W G & Baker J H (1962) *International Abstracts of Surgery* **114,** 1–7
Williams N F & Scobie T K (1976) *Canadian Medical Association Journal* **115,** 1223–1225
Zupunski A (1979) *Medicinski Pregled* **32,** 51–54