Isolated perforation of the gall bladder following blunt abdominal trauma

D. A. LAFFEY
B.Sc., M.B., Ch.B.

D. J. HAY
M.B., F.R.C.S.

Department of Surgery, University of Manchester Medical School, Hope Hospital, Salford M6 8HD

Summary
Isolated perforation of the gall bladder as a consequence of blunt abdominal injury is rare. A single case is described which illustrates several features which may characterize this lesion.

Introduction
Rupture of the gall bladder is a well recognized complication of acute cholecystitis. The gall bladder may be damaged along with other viscera in cases of massive abdominal injury or when penetrating wounds occur. Isolated perforation of the gall bladder as a result of non-penetrating injury to the abdomen is a very much rarer occurrence. In his review of the literature, Gayen (1973) found 51 previously reported cases. An apparent increase in the incidence of this lesion has been ascribed by several authors (Schecter, 1969; Fletcher, 1972) to a rise in the numbers of automobile accidents with rapid deceleration and impact against a steering wheel or seat belt as the probable mechanism of injury.

Case report
A 51-year-old man was admitted to hospital as an emergency. An itinerant with a long history of heavy alcohol abuse, he was a resident in a reception centre. Two and a half weeks before admission, whilst under the influence of alcohol, he had been attacked and kicked in the abdomen. Shortly after the incident he was seen by a doctor who noted some tenderness and guarding in the right upper quadrant and as the patient was otherwise well, a presumptive diagnosis of superficial injury was made and no specific treatment was offered.

In the period up to his admission, the patient became anorexic and began to vomit all solid food and for the week before his admission to hospital, his intake had been restricted to fluids. In addition, his abdomen had become more painful and had swollen progressively. During this period, his bowels had continued to function at least once daily, with the production of watery green or yellow stools. He also suffered sweating attacks.

On examination he was cachectic, afebrile, not shocked or anaemic and with no jaundice or stigmata of liver disease. His abdomen was distended with fluid and was generally tender with some guarding. Bowel sounds were present although reduced and rectal examination revealed yellow, foul-smelling stools. Investigations on admission showed: haemoglobin 14-4 g/dl, PCV 0-402, WBC 11-5 x 10⁹/l, with a normal differential. Urea and electrolytes were normal and the serum amylase was 266 Somogyi units/100 ml. The chest X-ray was clear and the abdominal X-rays served only to confirm the presence of ascites. Paracentesis abdominis was performed and more than 3 litres of dark yellow fluid were removed. Following this procedure, the patient's symptoms were much relieved and enabled a more thorough clinical examination of the abdomen, which was found to be minimally tender with no rigidity. No masses were detectable and bowel sounds were normal. It was decided to treat this patient conservatively, at least until the results of the analyses of the ascitic fluids were known. He was at first able to tolerate a light diet, but over the course of the next 4 days, with the rapid re-accumulation of ascites came a return of the vomiting and abdominal pain together with the appearance of jaundice. Liver function tests during this period showed total bilirubin 52 mmol/l (3–17), alkaline phosphatase 640 u/l (20–95) and albumin 31 g/l (36–52).

In view of the rapid deterioration of the patient's condition, it was decided to perform a laparoscopy which showed gross bile staining of the peritoneal cavity. Laparotomy was performed immediately and this showed gross biliary contamination of the whole peritoneal cavity with multiple collections of fibrinous exudate between loops of bowel, in the pelvis, in both paracolic gutters and beneath both hemi-diaphragms. The thin walled, otherwise normal gall bladder was perforated by a small tear in its fundus. There were no gall stones. The pancreas was very thickened and there was marked fat necrosis. A pre-operative cholangiogram showed no other abnormality in the biliary tree and cholecystectomy was performed, together with drainage of the common bile duct with a T-tube. Drains were
Case reports

The gall bladder has been well summarized in previous reviews (Estrada and Sutherland, 1969; Schecter, 1969). Characteristically, following the initial injury, symptoms are few, usually relatively mild and there is little constitutional upset. The initial symptoms often regress and there may follow what Schecter has termed a 'period of illusion' in which the patient's condition may improve to an extent to lead to discharge from hospital (Estrada and Sutherland, 1969; Breen, 1975). This period may last for a matter of hours, several days or even weeks and gradually gives way to increasing abdominal discomfort and collection of ascites.

There have been several reports of delayed perforation of the gall bladder following blunt trauma (Fielding and Strachan, 1975; Brickley et al., 1960), in which the clinical course and operative findings have been such as to suggest that gross biliary contamination of the peritoneal cavity occurred some considerable time after the initial trauma. This has been explained in terms of late perforation of a contused area of gall bladder or of loss of omental sealing as a late event.

Delay in making the diagnosis is the rule rather than the exception. Many cases have been reported where the delay is a matter of weeks rather than hours (Fletcher, 1972). Diagnosis is rarely made before operation.

Non-traumatic perforation of the gall bladder as may occur in acute cholecystitis is much commoner than traumatic perforation. Spillage of infected bile gives rise to the picture of 'classical' biliary peritonitis with acute onset of toxaemia and shock, and associated very high mortality variously estimated at between 30% and 90% (Ellis and Adair, 1974; MacDonald, 1966). In the great majority of recorded cases of traumatic perforation, however, there has been no pre-existing biliary tract disease. Leakage of sterile bile gives rise to a clinical picture, exemplified by the case described, which bears little resemblance either in symptomatology or in prognosis to the more common 'classical' biliary peritonitis. The overall mortality in traumatic perforation of the gall bladder has been estimated at about 5% (Schecter, 1969). Breen (1975) in his search of the literature was unable to find recorded a single death attributable to traumatic rupture of the gall bladder as an isolated injury.

Biochemical tests are of very limited value in making an early diagnosis. Liver function tests may be of value in revealing diversion of the bile stream. Peritoneal lavage offers the greatest prospect of reaching the diagnosis with the provisos that false-positive results may be obtained in cases of ruptured foregut or even in acute pancreatitis and false negative results are possible if bile leakage is entirely retropitoneal.
In several important respects the case presented accords with previous examples of this rare condition, namely the association with alcohol ingestion, the protracted course with delay in diagnosis and the comparatively benign nature of this form of biliary peritonitis. It is suggested that a wider appreciation of the essential differences between the clinical picture resulting from contamination of the peritoneal cavity with sterile bile as compared with infected bile might lead to earlier diagnosis in these cases.

Acknowledgment
We wish to thank Mr G. Ingram, under whose care this patient was, and Mrs E. W. Robinson, for typing the manuscript.

References

Isolated perforation of the gall bladder following blunt abdominal trauma.

D. A. Laffey and D. J. Hay

doi: 10.1136/pgmj.55.641.212

Updated information and services can be found at:
http://pmj.bmj.com/content/55/641/212

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/