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Case report

Acute acalculous cholecystitis in intensive care

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Acute acalculous cholecystitis is increasingly being recognised as a complication of critical illness. The disease usually develops on a background of prolonged illness following multiple trauma, extensive burns, severe sepsis, major surgery or drug overdosage. The clinical picture is virtually indistinguishable from acute obstructive cholecystitis and the diagnosis is often made at laparotomy.[1] We report three cases which occurred over a nine-year period in a six bedded intensive care unit.

CASE 1. A 39-year-old male with longstanding ankylosing spondylitis underwent manipulative spinal osteotomy to correct spinal deformity. Four days later he became profoundly shocked, with respiratory and renal failure. Initial laparotomy revealed a large perforated gastric ulcer. A long complicated illness ensued, with prolonged respiratory failure necessitating artificial ventilation via a tracheostomy, inotropic support, and haemodialysis. Another laparotomy was performed on the seventeenth day to drain a subphrenic abscess; a distended gangrenous gallbladder was found and removed. Four months after the start of his illness, a third laparotomy was done because of persistent vomiting. There were widespread adhesions and infected bile escaped during mobilisation of the right colonic flexure, which was adherent to the remains of the gallbladder bed. *Candida albicans* was cultured from a specimen of bile, and the same organism was isolated from blood cultures. This man was nearly six months in intensive care, and we were later able to identify a deficiency of the trace element selenium.[2]

CASE 2. A 22-year-old female developed acute renal failure secondary to postoperative hypovolaemia and hypotension following small-bowel resection for adhesions and gangrenous bowel. She was transferred to this hospital for dialysis. A second laparotomy, one week after the first, revealed further necrotic bowel which was resected. There were about two litres of green fluid in the abdominal cavity and the gallbladder was noted to be stained a deep green colour but otherwise seemed healthy. She remained acutely ill after this operation with paralytic ileus, large naso-gastric aspirates, tachycardia and hypertension. She

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developed tenderness over the gallbladder which was removed at a third laparotomy one week later, and found to be still stained dark green, slightly distended and flabby. Some adhesions were mobilised during this operation, and she subsequently made a slow but satisfactory recovery.

CASE 3. A 42-year-old male suffered very severe chest and shoulder trauma during a road traffic accident. There was a marked flail segment and a major degree of surgical emphysema which extended as far as the pubis. He required prolonged ventilatory support. On the twelfth day he became critically ill with refractory hypotension, disseminated intravascular coagulopathy and severe jaundice. He gave every appearance of septicaemia although no organism was isolated. An ultrasound scan showed biliary ducts and gallbladder of normal calibre. One week later CT scan of chest and abdomen was normal. After a further week the patient was able to indicate that he had abdominal pain. His white cell count rose from $26 \times 10^9/1$ to $60 \times 10^9/1$. Repeat ultrasound scan suggested cholecystitis with a dilated tender gallbladder. This was confirmed at laparotomy, and a tense gallbladder with a patchy gangrenous wall was removed. Eight days later he required wound exploration for evacuation of a haematoma but otherwise he made a gradual recovery.

DISCUSSION

Acute acalculous cholecystitis accounts for up to 8% of acute cholecystitis.[1] It is not a new disease, the first recorded case being in 1844.[3] Sporadic reports appeared over the next century, and there has been a dramatic and linear increase in incidence since 1950. Such increase would seem to coincide with the advent of intensive care. The incidence of the disease has been estimated at between 0·5–1·6% in critically ill patients.[4] In our series we estimated that 383 patients were at risk, having stayed in intensive care for more than seven days, giving an incidence of 0·8%.

Our patients suffered from many of the risk factors known to be associated with acute acalculous cholecystitis. These have been listed as shock, septicaemia, prolonged fasting, prolonged ventilatory support, use of total parenteral nutrition, dehydration, prolonged infusion of opiate drugs, and multiple blood transfusions.[4] Many of those risk factors are known to diminish gallbladder function. Positive pressure ventilation has been shown to increase pressure within the common bile duct, while the physiological effects of prolonged starvation and opiate drugs are well recognised inhibitors of gallbladder contraction. Inspissation of bile and perhaps obstruction of the cystic duct predispose to some degree of gallbladder ischaemia and the risk of secondary infection.[5] Orlando and colleagues[3] discussed the concept of gallbladder perfusion pressure (mean arterial pressure minus intraluminal pressure) in the development of the syndrome. Mean arterial pressure is decreased by hypotension and the use of vaso-active drugs, while intraluminal pressure is increased by biliary stasis, inspissated bile and oedema of the ampulla. A decrease in gallbladder perfusion pressure would lead to ischaemia and necrosis, the causes of which can be considered to be multifactorial. The epithelial damage that occurs during low-flow states thus shares a common cause with stress ulceration, renal tubular necrosis and haemorrhagic enteritis, and can be considered to be yet another manifestation of multiple organ failure. In one small study, 45% of 25 critically ill patients had bile sludge or a thickened gallbladder

wall demonstrated by ultrasonography, although none of the patients required operation.[6] Another report hinted at an association between acute gallbladder disease and multiple organ failure following major long bone trauma, but this has not been confirmed.[7]

The role of total parenteral nutrition in the aetiology of the disease is difficult to understand. Presumably the majority of patients would not survive long enough to develop acute gallbladder disease without intravenous feeding. However, Roslyn and colleagues[8] have documented a very high incidence of gallbladder disease (45%) in a series of patients with short bowel syndrome or inflammatory bowel disease who received total parenteral nutrition for a period of two months or more. The average period of intravenous feeding in their patients was 18 weeks, and the majority of patients with gallbladder disease had gall stones. Other workers[9] have compared the histological appearance of intense vascular injury in the muscularis and serosa of the gallbladder to that which can be produced in some animals experimentally following activation of factor X11 (Hageman factor) dependent pathways by intravenous injection of bacterial endotoxins.

Pathological examination of the diseased gallbladders in our cases showed the classical appearance[1] of enlarged organs with thickened oedematous walls, marked cellular infiltration, and varying degrees of mucosal ulceration, necrosis and patchy thrombosis of vessels. Gangrenous areas showed complete destruction of the normal architecture. In none of our cases were bacteria or fungi seen microscopically or isolated on culture. An increased index of suspicion for this disease is required if the high mortality associated with late diagnosis and delayed treatment is to be avoided.[3] Serial ultrasound investigations in high-risk patients are of proven value, and an aggressive surgical philosophy aimed at early laparotomy where intra-abdominal sepsis is suspected is recommended.[4]

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