Gastric outflow obstruction caused by gall stones and leading to death by complex metabolic derangement

C O Wight, M Seed, W W Yeo, T A McCulloch

Abstract

A 67 year old woman was admitted with a three week history of vomiting, having become increasingly confused for three days. Investigations revealed deranged serum biochemistry consistent with a combination of a diabetic non-ketotic hyperosmolar state and a metabolic alkalosis consistent with gastric outflow obstruction. She was treated with intravenous saline, intravenous insulin, and subcutaneous heparin, but did not improve clinically and had an asystolic cardiac arrest the following day; she was transferred to the intensive care unit and despite treatment with inotropes she died 40 hours after admission. Necropsy revealed that the stomach was massively dilated with gas and stomach contents, and contained many small black faceted gall stones. In addition a large non-faceted brown-yellow gall stone was wedged in the pyloric antrum causing total obstruction. The patient had died from a complex metabolic derangement including the cause of intrauterine death. We have not been able to find an account of rupture of the heart secondary to this lesion, but given the histogenesis and frequent presentation in early childhood, it is not altogether surprising that a foregut cyst may cause intrauterine death.

non-ketotic hyperosmotic diabetic coma and metabolic alkalosis precipitated by the acute gastric outflow obstruction complicated by previously undiagnosed type II diabetes mellitus.

Keywords: gastric outflow obstruction; gall stones; diabetes mellitus

Gall stone ileus occurs when a gall stone erodes into part of the gastrointestinal system and causes intestinal obstruction. It is a well recognised but rare complication of cholecystitis, resulting in 1–2% of all adult small bowel obstructions.1 The importance of gall stone ileus lies in its high mortality (12–27%), which is thought to be due to the patients being elderly and suffering from concomitant diseases.2 The most frequent site of impaction of the gall stone is in the terminal ileum following erosion of the gall stone into the duodenum.1 Gastric outlet obstruction by gall stones is a very rare but recognised complication of cholecystitis resulting from erosion of a gall stone into the gastric antrum.3

We report a case of death by non-ketotic hyperosmolar coma resulting from pyloric obstruction caused by perforation of a large gall stone into the pyloric antrum and complicated by previously undiagnosed non-insulin dependent diabetes. To the best of our knowledge, this is the third reported case of gastric outlet obstruction by a gall stone and the first complicated by non-insulin dependent diabetes.

Case report
A 67 year old woman was admitted with a three week history of vomiting, having become increasingly confused for three days. She had longstanding hypertension that was treated with methyl-dopa, atenolol, and a combination of amiloride and cyclopenthiazide. Otherwise, past medical history was unremarkable.

On examination she was obese, drowsy, and very dehydrated. Her blood pressure was 70/40 mm Hg and she had atrial fibrillation. There was clinical suspicion of an epigastric mass, which was thought to be a tumour, but her abdomen was non-tender and bowel sounds were present.

Investigations revealed deranged serum biochemistry consistent with a combination of a diabetic non-ketotic hyperosmolar state (calculated osmolality 347 mmol/l) and a metabolic alkalosis consistent with gastric outflow obstruction (sodium 118 mmol/l, potassium 3.9 mmol/l, bicarbonate 43 mmol/l, urea 69.5 mmol/l, creatinine 441 mmol/l, glucose 41.5 mmol/l).

She was treated with intravenous saline, intravenous insulin, and subcutaneous heparin, but did not improve clinically and had an asystolic cardiac arrest the following day after an episode of vomiting. Cardiac output was restored with adrenaline and atropine and she was transferred to the intensive care unit. Despite treatment with inotropes she steadily deteriorated and died 40 hours after admission.

PATHOLOGICAL EXAMINATION
At postmortem examination the stomach was massively dilated with gas and stomach contents, and found to contain many small black faceted gall stones, each approximately 5 mm in maximum diameter. In addition a large non-faceted brown-yellow gall stone measuring $5 \times 3.5 \times 3.5$ cm was wedged in the pyloric antrum causing total obstruction (fig 1). There was a 2 cm diameter fistula running directly from the gall bladder into the pyloric antrum, the mucosa of which was eroded and the wall appeared fibrotic. The gall bladder also contained multiple small faceted black gall stones. The common bile duct and left and right hepatic ducts were dilated but contained no gall stones. The liver appeared normal on external surface but was congested on slicing. Histology was unremarkable. The rest of the gastrointestinal system was within normal limits. The only other postmortem findings were a minimal degree of atheroma in the large arteries and some pulmonary oedema.

We concluded that the patient had died from a complex metabolic derangement including non-ketotic hyperosmotic diabetic coma and metabolic alkalosis precipitated by the acute gastric outflow obstruction complicated by previously undiagnosed type II diabetes mellitus.

Discussion
Gall stone ileus has a high mortality, which is thought to result from the elderly age of many patients,4 concomitant disease, and delay in seeking medical care after intestinal obstruction. In one study 50% of patients presenting with gall stone ileus had significant pulmonary disease, 33% had cardiac disease, and 40% had diabetes mellitus.5 In another study the number of patients having concomitant diabetes mellitus was as high as 50%.6 In this case diabetes mellitus was previously undiagnosed and the combination of the gastric outflow obstruction and diabetes led to a complex metabolic picture resulting in death. Furthermore, less than half of patients presenting with gall stone ileus have known biliary tract disease, and so there is often a delay in diagnosis5 once medical help is sought.

Figure 1 A large gall stone is impacted in the gastric antrum (arrow). A probe is present in the common bile duct (ST, stomach; L, liver).
Short reports

Systemic reactive amyloidosis associated with Castleman’s disease: serial changes of the concentrations of acute phase serum amyloid A and interleukin 6 in serum

Shu-ichi Ikeda, Hisanao Chisuwa, Seiji Kawasaki, Junko Ozawa, Yoshinobu Hoshii, Tadaaki Yokota, Tuneto Aoi

Abstract
A case is reported of a 21 year old woman who suffered from Castleman’s disease and systemic reactive amyloidosis. The serum concentrations of serum amyloid A (SAA) and interleukin 6 (IL-6) were extremely high and amyloid protein was immunohistochromically identified as AA. After surgical excision of a large retroperitoneal lymph node with the pathological findings of plasma cell type of Castleman’s disease, both serum SAA and IL-6 declined, showing a similar pattern of reduction curves. All clinical symptoms and laboratory abnormalities greatly improved. The biochemical feature of Castleman’s disease is abnormal production of IL-6 and this cytokine continuously may stimulate the synthesis of an amyloid precursor, SAA, causing systemic reactive (AA) amyloidosis. This pathogenetic theory is strongly supported by the present study.
(J Clin Pathol 1997;50:965–967)

Keywords: Castleman’s disease; amyloid; serum amyloid A; interleukin 6

Systemic reactive (AA) amyloidosis usually occurs in patients with chronic inflammatory disorders. Serum amyloid A (SAA), which is an acute phase reactant of hepatic origin, is an amyloid precursor in this type of amyloidosis. It is well known that the serum concentration of SAA dramatically increases in the inflammatory state and this response is mediated by actions of some cytokines including interleukin 1 (IL-1), IL-6, and tumour necrosis factor (TNF).

Castleman’s disease is a unique form of lymphoproliferative disorder characterised pathologically by the presence of giant lymph node hyperplasia with plasma cell infiltration.

Patients with this disease commonly have fever, anaemia, hypergammaglobulinaemia, and an increase in the serum concentrations of acute phase reactant proteins, all of which are ascribed to the large amount of IL-6 produced in the hyperplastic lymph nodes. We examined serial changes of serum concentrations of SAA and IL-6 in a patient with Castleman’s disease and systemic reactive amyloidosis who was treated with surgical removal of an involved large solitary lymph node.

Case report
The patient was a 21 year old woman with a nine year history of general fatigue, arthralgia, and slight fever. In the past three years she had been suffering from epigastric discomfort and poor appetite. On examination, she looked sick and had an enormously enlarged liver with hard consistency, other physical findings were unremarkable. Abnormal laboratory findings are summarised in table 1. Briefly, she had a raised erythrocyte sedimentation rate (152 mm in the first hour), raised C reactive protein (CRP), anaemia, thrombocytosis, hypoalbuminaemia, hypergammaglobulinaemia, and slight proteinuria. Biopsies of liver tissues and gastric mucosa showed severe amyloid deposits with alkaline Congo red staining; amyloid deposition diffusely involved the parenchymal sinusoidal space of Disse and this was also seen in the lamina propria and muscularis mucosae of the gastric mucosa. After pretreatment with KMnO4, these amyloid deposits lost the affinity for Congo red dye (data not shown). Computed tomography of the abdomen revealed a mass with small calcifications that mimicked a liver tumour in the caudate lobe. Surgical

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Accepted for publication 15 July 1997

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*J Clin Pathol* 1997 50: 963-965
doi: 10.1136/jcp.50.11.963

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