CASE REPORT

# A rare case of subcapsular liver haematoma following laparoscopic cholecystectomy

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## SUMMARY

Laparoscopic cholecystectomy is a commonly performed surgical procedure for the treatment of symptomatic cholelithiasis. As with all surgical procedures, it carries risk, with the most commonly reported complications including infection, bile leak and bleeding. One unusual complication is subcapsular liver haematoma, the diagnosis presented here. This is a rare occurrence; only a small number of cases have been reported in the literature and as yet no conclusive cause or management plan has been found. Iatrogenic liver trauma, the use of oral and intravenous non-steroidal anti-inflammatory drugs (NSAIDs) and anticoagulants have all been named as possible contributing factors. Particularly, the use of ketorolac has been associated with four reported cases of subcapsular haematoma following laparoscopic cholecystectomy. The case reported here refutes that hypothesis, as neither NSAIDs nor anticoagulants were used during the treatment of this patient.

## BACKGROUND

The incidence of symptomatic gallstones in the Western world is rising, with an estimated 20% of adults over 40 diagnosed with gallstones.[1] These are treated surgically by the removal of the gallbladder by routine laparoscopic cholecystectomy (LC).

The first UK LC was performed in Dundee by Nathanson and Cuschieri in 1989, and by 1992 10 000 LCs had been performed, largely replacing open cholecystectomy as a first-line treatment.[2] It is considered to be such a safe procedure that many patients are discharged home on the same day of surgery. In 2009, the alternative single-incision laparoscopic cholecystectomy was developed as a method to provide even less invasive surgery; this procedure is not currently in widespread use in the UK.[3] Despite the proven efficacy and safety of LC, the possibility of serious risks and side effects remains.[1] The most common complications include bleeding, infection, bile duct injury, intra-abdominal collections, port site hernia, ileus and pneumonia.[4–6]

We report an unusual complication, known as a hepatic subcapsular haematoma, of LC. Such a diagnosis is typically associated with blunt force hepatic trauma, such as from a road traffic accident or a stab wound to the right hypochondrium. A number of cases have been reported in the literature concerning post-LC subcapsular hepatic haematoma, but there is no agreement as to the cause. A number of cases have suggested the use of intraoperative non-steroidal anti-inflammatory drugs (NSAIDs) such as ketorolac and diclofenac as a cause, others postulate that accidental injury intraoperatively with laparoscopic instruments are the source. This case highlights this potential complication to other clinicians and in fact contradicts the view that NSAIDs, anticoagulation or intraoperative injury are the cause of subcapsular hepatic haematoma.

## CASE PRESENTATION

We report the case of a 60-year-old woman who presented to the emergency department 6 days following a day-case LC. She presented specifically with gradually worsening right upper quadrant abdominal pain, with associated fever and nausea. Her medical history included dyslipidaemia, hypertension and hypothyroidism, and although prescribed low-dose aspirin for primary prevention of cardiac disease, it was discontinued 2 weeks before surgery and she had not recommenced it at the time of readmission. Her operation was performed as a result of many years of recurrent biliary colic and chronic cholecystitis that culminated in a severe episode of gallstone pancreatitis requiring ERCP (endoscopic retrograde cholangiopancreatography) and sphincterotomy. No other abnormalities were noted regarding her liver or gallstone anatomy on the abdomen ultrasound scan (USS) or MRCP performed to make this diagnosis. On physical examination, she was noted to have a fever of >38°C but was otherwise haemodynamically stable. She was tender and guarding in her right upper quadrant on abdominal examination, with the remainder of her examination proving to be normal.

On review of her operation records, her procedure was largely uneventful and completed according to standard protocol. A Hasson subumbilical port was inserted under direct vision following skin infiltration with levobupivacaine. Following the establishment of pneumoperitoneum, one 10 mm operating port and two 5 mm assisting ports were introduced. The gallbladder was noted to be thick walled and difficult to dissect and two small gallbladder perforations were made with subsequent escape of bile, though no stones were lost. No intraoperative liver injury occurred, and blood and bile loss was noted to be minimal. It was noted intraoperatively that she had a small perforating artery that was clipped and no bleeding was noted. Subsequent gallbladder histopathology demonstrated evidence of chronic cholecystitis with no dysplastic change or malignancy, which was consistent with her preoperative clinical history. It was noted that this patient was not given intravenous or

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oral NSAID during her hospital stay and she denied taking any oral NSAIDs after discharge.

## INVESTIGATIONS

Blood investigations confirmed raised inflammatory markers with white cell count (WCC) 13.6 (e9/L) and C reactive protein (CRP) 285 mg/L, consistent with her presentation and fever. Full blood count also demonstrated a mild normocytic anaemia, with haemoglobin 113 g/L, notably lower than her preoperative level of 138 g/L. Despite the history of pancreatitis, her liver function tests remained normal throughout her admission—bilirubin 11 μmol/L, serum alkaline phosphatase 89 U/L and both transaminases within normal limits. A contrast-enhanced CT of the abdomen and pelvis demonstrated a large, inhomogeneous fluid collection measuring 12×7×8 cm located posteriorly and inferiorly to the liver, with evidence of pneumobilia (figure 1). Expert radiology advice was sought who felt that there was a subcapsular haematoma, with early evidence of infection.

## TREATMENT

Conservative management with broad-spectrum intravenous antibiotics was initiated, with the patient's WCC level showing initial improvement to 12 e9/L and CRP falling to 235 mg/L. Despite 6 days treatment, the patient continued to demonstrate ongoing low-grade fever and right upper quadrant pain with her haemoglobin level falling very gradually over this time period to 95 g/L, where it then remained static. A repeat CT scan was arranged, which demonstrated persistent subcapsular collection with reduced attenuation, liquefaction of previous blood products and increasing pneumobilia, consistent with an infected haematoma. Following further consultant with radiology, non-operative management was continued and an US-guided pigtail catheter was inserted into the collection on day 14 postoperation, draining a large volume of altered blood (figure 2). The patient's pigtail drain remained in situ for 1 week where it continued to drain liquefactive haematoma; her blood investigations thereafter revealed a steady fall in WCC and CRP while her haemoglobin remained static. Although microscopy and culture failed to demonstrate an infective source, the patient was treated clinically as an infected haematoma, and antibiotics were continued for 48 h following removal of the drain.

## OUTCOME AND FOLLOW-UP

The patient was discharged from hospital day 25 post-LC after an 18-day postoperative readmission with a plan for outpatient follow-up. She attended for routine review 6 weeks later and was found to be fully recovered with normal blood investigation and no residual haematoma on USS. She has now been discharged from routine follow-up.

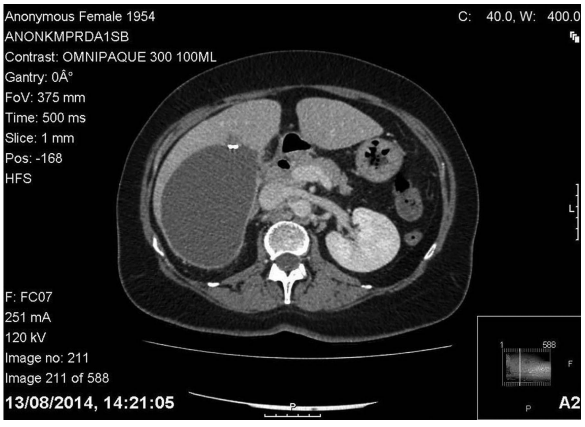


**Figure 1** Contrast-enhanced CT of the abdomen and pelvis demonstrating a large subcapsular haematoma with evidence of pneumobilia in biliary tree.

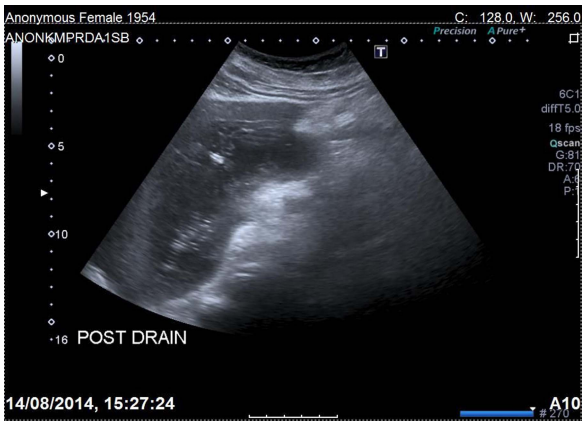


**Figure 2** Ultrasound image of subcapsular haematoma following percutaneous drainage.

## DISCUSSION

Subcapsular haematoma is a rare, recognised complication following LC. It is characterised by a fluid collection, such as blood, bile or pus, forming between the thick, fibrous inner layer of the liver and the outer serous layer, the visceral peritoneum.[7] A small number of case reports have been published detailing the potential contributing factors to their formation; such as NSAID use, anticoagulant use and the possibility of intraoperative trauma. Conservative, surgical and radiological management of post-LC subcapsular haematomas have all been reported; despite this, aetiology and best practice management remain unclear.

NSAIDs are a class of painkiller that are commonly used postoperatively for their analgesic, antifeverish and anti-inflammatory effects. A number of case reports propose that their use may be responsible for the formation of subcapsular haematoma following LC. Shibuya *et al*[8] and Vuilleumier and Halkic[9] report cases of patients who were treated for hypovolaemic shock with emergency laparoscopic intervention following a ruptured subcapsular haematoma. Both papers attribute a combination of presumed intraoperative trauma and the use of NSAIDs for postoperative pain relief as potential causes. Guercio *et al*[10] report a case of subcapsular haematoma that they also attribute to a combination of intraoperative trauma and the administration of NSAIDs, in this case intravenous ketorolac. This view has been echoed in three further cases of subcapsular haematoma following the administration of intravenous ketorolac.[11 12] Ketorolac is a non-selective COX (cyclooxygenase inhibitor) that acts to inhibit prostaglandin synthesis thereby supressing the inflammation cascade. Strom *et al*[13] found that high-dose ketorolac, particularly in the elderly, was associated with an increased risk of surgical site and gastrointestinal bleeding; consequently, its use in the UK is now restricted. Concluding that the use of NSAIDs could contribute to subcapsular haematoma following LC is reasonable; however, the patient we report here did not receive any NSAID medication at any point in her intraoperative or postoperative care.

De Castro *et al* describe the case of an unstable subcapsular haematoma presenting 6 weeks postoperatively with active bleeding that required initial embolisation and subsequent

radiological drainage of an infected haematoma. This patient had been given an NSAID, diclofenac sodium, in the days following her initial surgery and, due to extensive cardiac comorbidities, was anticoagulated with low molecular weight heparin (LMWH) preoperatively and postoperatively.[14] Both of these medications could be implicated in a subcapsular bleed. Our patient received only one injection of 40 mg LMWH (enoxaparin) during her initial admission; though it is possible that LMWH could be implicated, as she received only one dose this seems unlikely. Other antiplatelets and anticoagulants, such as clopidogrel and warfarin, are routinely discontinued prior to any operative intervention, and thus far they have not been attributed to any subcapsular haematomas. The patient we report had a regular prescription of low-dose aspirin. This is not routinely stopped before LC procedure but it was discontinued voluntarily by the patient prior to surgery and was not restarted afterwards.

Proposing a 'best-practice' management plan for post-LC laparoscopic haematoma is difficult. We describe the use of US-guided drainage of the haematoma rather than a surgical intervention. The decision to avoid surgery was motivated by a reluctance to disturb the haematoma resulting in catastrophic bleeding and laparotomy, but also by the wishes of the patient, who was keen to avoid surgery. Percutaneous drainage has been shown to be a safe and effective intervention for the drainage of intra-abdominal collections following gastrointestinal surgery and other surgeries in the majority of patients.[15–17] Draining a haematoma surgically would necessitate either laparoscopic or open surgery; the less invasive nature of a percutaneous drain may be preferred by patients, even if the outcome is a slightly longer hospital admission.

In this case, it is difficult to conclude an aetiology behind the development of this subcapsular haematoma—no NSAIDs were administered, and anticoagulant and antiplatelet use was minimal. The most likely cause in this case could be a small bleed from an accessory perforator artery or unidentified trauma, however, the consultant surgeon performing the procedure did not report any intraoperative trauma or immediate complications. Three months preoperatively, the patient required ERCP treatment for a common bile duct stone that caused an episode of pancreatitis and this may explain some of the pneumobilia found on postoperative imaging. In this case, the cause of the subcapsular haematoma remains unexplained, but clinically, the patient responded very well to percutaneous drainage and antibiotics, excluding the need for surgical intervention. As presentation of subcapsular haematoma is both rare and variable, we would recommend initiating a treatment plan on a case by case basis and accept that although conservative management was the best choice here, some patients should have surgical intervention.

## Learning points

- Despite the minimally invasive nature of laparoscopic surgery and short recovery time, this procedure still carries significant risks and surgeons should be alert to this.
- This case contradicts the previous cases, which cite intravenous ketorolac and low molecular weight heparin as the cause of subcapsular haematoma postlaparoscopic cholecystectomy.
- It shows the important role radiological guidance of percutaneous drainage plays in the management of patients with abdominal collections.

**Competing interests** None declared.

**Patient consent** Obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.

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## Findings that shed new light on the possible pathogenesis of a disease or an adverse effect