

A Complication of Acute Pancreatitis: Lumbar Vessel Rupture

A De Rosa, K Oyewole, M Hanief, R Vashisht

Citation

A De Rosa, K Oyewole, M Hanief, R Vashisht. *A Complication of Acute Pancreatitis: Lumbar Vessel Rupture*. The Internet Journal of Surgery. 2009 Volume 22 Number 2.

Abstract

Severe cases of acute pancreatitis are associated with a high mortality rate, secondary to complications that develop as a consequence of the inflammatory and necrotic process. Haemorrhage is one of the most life-threatening complications. We report the case of a 63-year-old woman who presented with acute gallstone pancreatitis, complicated by lumbar vessel rupture. To our knowledge this is the first case of reported lumbar vessel rupture associated with acute pancreatitis. We emphasise the value of CT in the early diagnosis of bleeding and the value of angiography for control of bleeding from ruptured vessels.

INTRODUCTION

Approximately 25% of acute pancreatitis cases are severe¹, and severe pancreatitis is associated with a mortality rate of up to 9.4 %². Complications of pancreatitis can be categorised into systemic or local. Systemic complications include hyperglycaemia, hypocalcaemia, Multi Organ Dysfunction Syndrome, Disseminated Intravascular Coagulation and Adult Respiratory Distress Syndrome. Local complications include pancreatic pseudocysts, abscess formation, duodenal obstruction and vascular complications such as haemorrhage and vein thrombosis. Haemorrhage is one of the most life-threatening complications, often due to bleeding from local blood vessels. Bleeding secondary to vascular injuries is more frequent in chronic pancreatitis; however, the mortality rate following bleeding in acute pancreatitis is higher, 60.4% compared with 22% in chronic pancreatitis in a study by Flati et al³. Of patients who die of pancreatitis, haemorrhage occurs most frequently into the retroperitoneum or peritoneal cavity³.

CASE REPORT

A 63-year-old female presented to the Accident and Emergency Department with a two-hour history of epigastric pain, radiating to the back, associated with nausea and vomiting.

On initial examination she was afebrile and haemodynamically stable. Abdominal examination demonstrated localised peritonitis in the epigastric region and a 'rigid abdomen.'

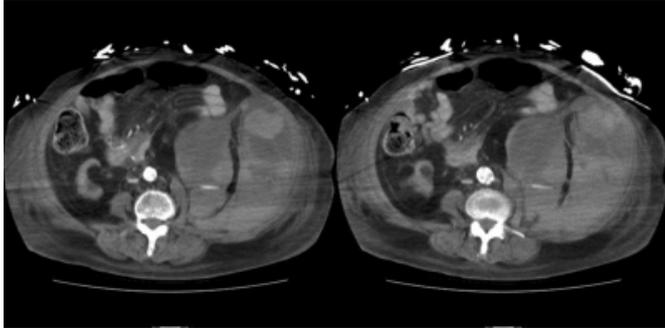
She had no history of pancreatitis, or alcohol excess, but had ultrasound evidence of gallstones. Past medical history included severe rheumatoid arthritis, controlled with steroids, methotrexate and rituximab, borderline diabetes mellitus, hypertension, osteoarthritis of the spine, chronic renal impairment and tobacco smoking of 15 cigarettes a day for more than 30 years.

She scored four on the Glasgow Prognostic Criteria for; age >55 years, WCC >15x10⁹/litre (43.6x10⁹/L), AST >200IU/L (356IU/L) and glucose >10mmol/L (15.6mmol/L). In addition, amylase on admission was 4209IU/L, and creatinine 142umol/L. Abdominal CT showed a gallstone in the gallbladder and possibly in the common bile duct and patchy pancreatic necrosis. She was admitted to HDU for invasive monitoring.

Subsequently, she developed respiratory failure and metabolic acidosis and was moved to ITU for intubation. Nine days post presentation, the haemoglobin dropped from 8.7 to 4.8 in six hours. CT scan of the chest and abdomen showed active bleeding from a lumbar vessel on the left side at L3 and a massive left abdominal retroperitoneal haematoma extending from the upper abdomen to the pelvis (Image 1&2). Arteriography performed hours later showed no active contrast medium extravasion; however, in view of the CT findings, the left 4th lumbar artery was embolised. The haematoma was drained in theatre on two occasions and she was discharged 11 weeks later. Ten months post discharge a laparoscopic cholecystectomy was performed and at follow-up 15 months later she reports being well.

Figure 1

Image 1&2. CT scan slices through the pelvis showing a large left-sided retroperitoneal haematoma within which is some linear density in keeping with enhancement in a lumbar vessel, and at the anterior edge is a blush of increased density demonstrating an active bleed from the lumbar vessel.



DISCUSSION

The pathogenic process in acute pancreatitis is initiated by a number of stimuli, the most common being alcohol and obstruction, secondary to gallstones. Such stimuli cause damage to the acinar cells of the pancreas either directly or indirectly. The damaged cells release exocrine, proteolytic and lipolytic enzyme-rich fluids, initiating an intense inflammatory response. This results in a self-perpetuating process of extravasation of activated enzymes, inflammation and necrosis, causing disruption and erosion of the walls of visceral and vascular structures. Vascular complications occur via numerous mechanisms; enzymatic erosion can cause direct rupture of the vessel and haemorrhage, the vessel wall can become weakened with the formation of a true aneurysm, which is prone to rupture, erosion of the vessel wall by the enzyme-rich fluid contained within pseudocysts can cause sanguination from the vessel into the lumen of the pseudocyst resulting in a pseudoaneurysm and eventual rupture. The necrotising and inflammatory process may cause thrombosis of the portal vein, secondary portal hypertension with subsequent variceal formation (e.g. oesophageal) and eventual bleeding. Depending on the location of the vessel and pathogenesis of vessel rupture, bleeding can occur into the gastrointestinal tract, peritoneal cavity or retroperitoneum.

In the order of decreasing incidence the splenic, gastroduodenal and superior pancreaticoduodenal arteries are the most commonly involved in pancreatitis. To our knowledge, we are the first to report lumbar artery rupture in acute pancreatitis. The head and body of the pancreas as well as the lumbar vessels lie in the retroperitoneum. It is thus

feasible that extravasation of pancreatic elastolytic enzymes caused erosion and subsequent rupture of the lumbar vessels, by a similar mechanism that occurs in splenic artery rupture.

Radiological modalities such as US, CT and angiography can diagnose vascular complications of pancreatitis by demonstrating active bleeding, the formation of arterial pseudoaneurysms, haemorrhage into a pseudocyst, portal venous thrombosis with development of varices, and fluid/haematoma formation. In this case, CT identified bleeding from a lumbar vessel on the left side at L3, although arteriography did not show extravasation of contrast medium; due to the significant drop in haemoglobin we deemed that it was appropriate to embolise the fourth lumbar artery. Following this, the patient's haemodynamic status improved and the haemoglobin stabilised at 8.5mmol/L.

The increasing use of diagnostic and interventional radiology appears the least invasive and safest approach in the management of arterial bleeding in acute pancreatitis. Transcatheter arterial embolisation may result in permanent control of the bleeding or temporary control, stabilising the patient, allowing for definitive operative management. A study of 19 patients by Zhou et al. showed that definitive haemostasis was achieved in 89.5%, the incidence of re-bleeding was 36.8% and the success rate of haemostasis by a second attempt following a re-bleed was 71.4%⁴. Transcatheter arterial embolisation is especially useful in the unstable, acutely ill, or septic patient who would have a poor operative risk such as this case, where, given the poor clinical and metabolic state, surgery would have had a high mortality rate. The evidence in support of the use of angiography in controlling bleeding in acute pancreatitis is in cases bleeding from pancreatic and peripancreatic vessels; we propose that angiography is a feasible method for controlling bleeding in vessels distant to the pancreas because of its success in this case.

CONCLUSION

Haemorrhage is an important consideration in the context of acute deterioration, haemodynamic instability and a drop of haemoglobin in a patient with acute pancreatitis. Early recognition enables timely intervention preventing further clinical deterioration, thus avoiding the need for more invasive operative management, and thus reducing the mortality rate associated with such bleeding complications. A high index of suspicion and lateral thinking is crucial in order to diagnose and treat bleeding from an unusual source.

References

1. Steinberg W and Tenner S. Acute Pancreatitis. *The New England Journal of Medicine* 1994;330:1198-1210
2. Corfield AP, Cooper MJ, Williamson RCN, Mayer AD, McMahon MJ, Dickson AP, Shearer MG, Imrie CW. Prediction of severity in acute pancreatitis: prospective comparison of three prognostic indices. *Lancet* 1985;2:403-407
3. Flati G, Andrén-Sandberg A, La Pinta M, Porowska B, Carboni M. Potentially fatal bleeding in acute pancreatitis: pathophysiology, prevention and treatment. *Pancreas* 2003;26(1):8-14
4. Zhou F, Wang C, Xiong J, Wan C, Zheng C. Experience in diagnosis and treatment of bleeding complications in severe acute pancreatitis by TAE. *J Huazhong Univ Sci* 2005;25(2):182-4

Author Information

Antonella De Rosa, MB ChB

Department of General Surgery, West Middlesex University Hospital

Kehinde Oyewole, MB ChB

Department of General Surgery, West Middlesex University Hospital

Mohamed Rubnawaz Hanief, MD MRCS

Department of General Surgery, West Middlesex University Hospital

Rajiv Vashisht, FRCS

Department of General Surgery, West Middlesex University Hospital