Reminder of important clinical lesson

Unusual presentation of spontaneous splenic haematoma due to severe pancreatitis: a cautionary tale

Karthik Purushothaman,¹ David W Borowski²¹Department of General Surgery, University Hospital North Tees, Stockton on Tees, UK²Department of Colorectal Surgery, University Hospital North Tees, Stockton on Tees, UK

Correspondence to David W Borowski; DWBorowski@AOL.com

Summary

We report the case of a patient with multiple splenic complications from chronic pancreatitis with pseudocyst formation, including splenic vein thrombosis, subcapsular splenic haematoma and splenic artery pseudoaneurysm. The initial presentation was associated with pleuritic chest pain, clinically resembling symptoms of pulmonary embolism. The patient was treated with therapeutic low-molecular-weight heparin, without confirmatory imaging. However, the latter arranged computed tomographic pulmonary angiogram was negative, while the abdominal sequences of the CT revealed the splenic haematoma as causative pathology. The patient was initially treated conservatively, and discharged from inpatient care. On a subsequent CT, a pseudoaneurysm of the splenic artery was found and treated with coil embolisation. The patient is currently awaiting definitive management of the pancreatic pseudocyst.

BACKGROUND

Pancreatitis is a common presentation to surgical emergency services in the western world, with increasing incidence in the UK.¹ While most cases are mild and self-limiting, few patients progress to severe pancreatitis as defined by the presence of multiorgan dysfunction, or local complications.² Splenic complications from pancreatitis are comparatively rare, and include splenic vein thrombosis, splenic haematoma or rupture and splenic artery pseudoaneurysms.

The anatomical location of the spleen, and its proximity to the diaphragm, can predispose to atypical presentations, giving rise to the possibility of misdiagnosis.

We present the case of a patient who not only suffered multiple splenic complications from his pancreatitis, but also presented in a rather atypical fashion; this resulted in initial misdiagnosis and inappropriate treatment with systemic anticoagulation.

CASE PRESENTATION

A 45-year-old man was admitted to the local Accident and Emergency department with sudden onset of left shoulder tip and left-sided chest pain of 6 h duration. The pain appeared pleuritic in nature, worse on inspiration, and was associated on expiration with palpitations, shortness of breath and nausea. There was no history of abdominal pain, vomiting, cough or haemoptysis. The patient had a history of alcohol-induced pancreatitis requiring hospitalisation several months earlier.

On examination, the patient was found to be tachycardic but normotensive, and had normal oxygen saturation on room air (99%). Initial chest and abdominal examination revealed no particular abnormality. ECG showed sinus tachycardia only.

Based on the initial blood results, with a significantly elevated D-dimer level at 2448 µm/l, a clinical working diagnosis of pulmonary embolism (PE) or acute coronary

syndrome was made and the patient was started on body-weight-adjusted therapeutic low-molecular-weight heparin (LMWH).

However, the 9 h later performed computed tomographic pulmonary angiogram (CTPA) was negative, while the abdominal sequences of the CT revealed the splenic haematoma as causative pathology for the patient's pain.

INVESTIGATIONS

On admission, the patient's blood results included haemoglobin: 12.4 gm/dl, white cell count: 7.9×10^9 , troponin I: 0.01 µm/l, D-dimer: 2448 µm/l, with an otherwise normal coagulation profile. C reactive protein was raised at 130 mg/l, serum pancreatic amylase was normal at 30 U/l (normal range 0–50 U/l).

ECG indicated sinus tachycardia only. Arterial blood gas at room air showed pH: 7.46 nmol/l, pCO₂: 4.53 kPa, pO₂: 11.1 kPa, bicarbonate: 25.0 meq/l and base excess: 1.1 mmol/l.

CTPA was negative for PE, but the CT of the abdomen revealed a pseudocyst in the tail of pancreas, splenic vein thrombosis and a large subcapsular splenic haematoma.

DIFFERENTIAL DIAGNOSIS

Clinical pre-CT differential diagnoses: PE, acute coronary syndrome, Perforated duodenal ulcer, splenic pathology; pancreatitis

TREATMENT

The patient was initially given a high dose of subcutaneous LMWH (11375 U) for suspected PE; as the CTPA did not confirm this diagnosis, and indeed showed the cause of the presentation to be locally complicated severe pancreatitis pseudocyst formation in the tail of the pancreas, splenic vein thrombosis and subcapsular splenic haematoma (figure 1), the patient was referred to the surgical on-call team for further management. After discussion

Figure 1 CT showing subcapsular splenic haematoma (arrow) and pancreatic tail pseudocyst (broken arrow) at initial presentation.

with the regional pancreatic centre, a decision was made for conservative management.

The patient was transferred to the intensive care unit; under invasive monitoring of blood pressure and central venous pressure, he dropped his haemoglobin and received repeated blood transfusions (a total of 5 units packed cells) until haemoglobin remained stable. Within 48 h, the patient developed full-blown alcohol withdrawal and had to be sedated and ventilated for a few days, but recovered well from this without signs of ongoing intra-abdominal blood loss.

After a total hospitalisation of 25 days, the patient was discharged, with out-patient follow-up at 6 weeks.

OUTCOME AND FOLLOW-UP

At out-patient follow-up, the patient remained well. However, on the follow-up CT at 10 weeks following discharge, the pseudocyst had increased in size, and a new splenic arterial pseudoaneurysm was discovered (figure 2). However, the splenic haematoma had regressed. The patient was urgently readmitted for successful coil embolisation of the pseudoaneurysm (figure 3), and is currently awaiting drainage of the pancreatic pseudocyst.

DISCUSSION

Spontaneous splenic haematoma is a rare complication of pancreatitis.^{3–5}

An estimated prevalence of 0.4% has been reported in the literature,³ and few of these cases documented in the literature have focused on conservative management.

The anatomical proximity of the pancreatic tail to the splenic hilum provides a potential for bleeding complications in pancreatitis.^{6–7} These complications occur in 1–5% of cases of pancreatitis,⁷ and can include splenic vein thrombosis, arterial pseudoaneurysms, bleeding in a pseudocyst, splenic haematoma and splenic rupture.^{5–8}

The classic presenting symptoms are pain, tenderness and guarding in the left upper quadrant, and hypovolaemic shock.⁹ Left shoulder tip pain, particularly when lying flat (Kehr's sign), is caused by blood irritating the

Figure 2 CT showing subcapsular splenic haematoma (arrow) and pancreatic pseudocyst with splenic artery pseudoaneurysm (broken arrow) at follow-up CT 10 weeks following discharge.

left hemi-diaphragm, and is said to be present in 50% of cases of splenic rupture.¹⁰ However, symptoms can be subtle, and the condition has occasionally presented with pleuritic symptoms.^{10–12}

Spontaneous subcapsular haematomas can potentially occur in both normal and diseased spleens, and require urgent surgical intervention as they can lead to fatalities caused by the dreaded splenic rupture. A subcapsular splenic haematoma is a type of haematoma that occurs within the splenic capsule, and can be the result of both traumatic and non-traumatic situations.^{13–16}

Causes of spontaneous splenic haematoma and rupture

Gastrointestinal

- ▶ Pancreatitis
- ▶ Liver cirrhosis
- ▶ Portal hypertension

Figure 3 Interventional angiogram demonstrating splenic artery pseudoaneurysm before (a) and after coil embolisation (b).

- ▶ Inflammatory bowel disease.
 - ▶ Infective
 - ▶ Epstein-Barr virus
 - ▶ Viral hepatitis
 - ▶ Bacterial endocarditis
 - ▶ Typhoid
 - ▶ Tuberculosis
 - ▶ Malaria
 - ▶ Visceral Leishmanniosis
 - ▶ Brucellosis
 - ▶ Syphilis
 - ▶ Tularemia .
- ▶ Granulomatous and metabolic
 - ▶ Amyloidosis
 - ▶ Sarcoidosis
 - ▶ Gaucher's disease
 - ▶ Felty's syndrome.
- ▶ Haematologic and neoplastic
 - ▶ Hemophilia
 - ▶ Anticoagulation
 - ▶ Hemolytic anaemia
 - ▶ Myeloid metaplasia/fibrosis
 - ▶ Lymphoma and leukaemia
 - ▶ Multiple myeloma
 - ▶ Metastatic carcinoma.
- ▶ Miscellaneous
 - ▶ Pregnancy
 - ▶ Raised intra-abdominal pressure (prolonged vomiting, chronic cough)
 - ▶ Polyarteritis nodosa.
- ▶ Colonoscopy

A few hypotheses have been proposed to explain the pathogenesis of spontaneous splenic haematoma and rupture associated with pancreatitis; the most common cause is splenic vein thrombosis with subsequent local hypertension, leading to haematoma formation. Splenic vein thrombosis in acute or chronic pancreatitis results from perivenous inflammation caused by the anatomic location of the splenic vein along the entire posterior aspect of the pancreatic tail, where it lies in direct contact with the peripancreatic inflammatory tissue. The exact mechanism of thrombosis is likely multifactorial, including both intrinsic endothelial damage from inflammatory processes and extrinsic damage secondary to venous compression from fibrosis, adjacent pseudocysts or oedema.¹⁷ Obstruction of the splenic vein may also be caused by enlarged retroperitoneal lymph nodes, or by pancreatic or perisplenic nodes that are located near the splenic artery, superior to the splenic vein. These nodes lie adjacent to the pancreas and the splenic vein, and may compress the splenic vein when involved in an inflammatory process.¹⁸

There have been several reports of spontaneous pancreatitis-induced subcapsular haematoma in the literature, but the management of this complication remains controversial. Some authors suggest a conservative, spleen-preserving approach in cases of haemodynamically stable patients with continuing signs of improvement, while others would favour early surgical intervention with splenectomy.

Patel *et al*¹⁹ reported a case of large subcapsular splenic haematoma which was managed conservatively with a favourable outcome; the CT scan at 4 months showed

marked resolution of the haematoma. Kuramitsu *et al*²⁰ reported a large subcapsular splenic haematoma, which was initially managed conservatively but later required surgical intervention for a flare-up of pancreatitis. Indeed, Rypens *et al*⁸ suggested that most of splenic complications from pancreatitis may potentially regress and should, therefore, be managed conservatively. As part of a conservative approach, radiological percutaneous drainage may be used.^{21 22} In contrast, Thompson and Ashley²³ advocated early splenectomy to prevent the rupture of the splenic haematoma and its associated morbidity.

One of the important lessons that can be learned from the here presented case is the possibility of a significant intra-abdominal bleeding complication without suggestive clinical signs. Our patient was treated for suspected PE without confirmatory imaging.

According to the British Thoracic Society (BTS) guidelines,²⁴ heparin should be given to patients with intermediate or high clinical probability prior to imaging. Unfractionated heparin (UFH) should be considered only as a first-dose bolus, in massive PE or where rapid reversal of effect may be required. Otherwise, comparatively long-acting LMWH is preferable to UFH, due to its equivalent efficacy, safety and ease of use. In contrast, a Spanish study by Galipienzo *et al*²⁵ reported that withholding anticoagulant therapy in patients with suspected venous thromboembolic disease without confirmatory CTPA seems to be safe. This latter approach would have allowed imaging prior to treatment, and prevented a not indicated treatment in our patient.

Blood D-dimer assay should only be considered following assessment of clinical probability; and are not required for patients with high clinical probability of PE. A negative D-dimer test reliably excludes PE in patients with low or intermediate clinical probability; and such patients do not require imaging for venous thromboembolism.

In a study of the predictive value of D-dimer assay for PE, Perrier *et al*²⁶ confirmed a negative test result to be less than 500 µm/l in their laboratory. Whereas there is no clear upper range for the D-dimer test in the context of PE, a unusually high lever—such as seen in the case presented here in order of a 10-fold increase above normal range according to our hospital laboratory—should raise suspicion of clotting process that would exceed the size of a pulmonary embolus.

In summary, this patient did not have the classic clinical signs of splenic haematoma during the initial presentation, and was started on therapeutic dose of LMWH on clinical suspicion of PE. However, there were no risk factors in this patient to suggest PE. This could have had a detrimental effect on the outcome if the splenic haematoma had not been recognised in the abdominal CT scan. While it is perceivable that the systemic anticoagulation in this case may have precipitated the splenic bleeding, the sudden onset of pain prompting the patient to seek medical attention would probably indicate otherwise.

Systemic anticoagulation should be started with an element of caution, and differential diagnoses must be considered.

Learning points

- ▶ Pancreatitis can have a number of splenic complications, including splenic haematoma, rupture, splenic vein thrombosis and pseudoaneurysms of the splenic artery.
- ▶ These complications may present without classic abdominal signs and symptoms; upper abdominal and splenic pathologies should be considered as important differential diagnoses in patients with chest pain and no other clinical indications for chest pathology.
- ▶ D-dimer levels substantially higher than the usual values seen in the context of pulmonary embolism should raise suspicion of other pathology.
- ▶ Therapeutic anticoagulation should be used with caution, preferably after confirmation of the diagnosis

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Gurusamy KS**, Farouk M, Tweedie JH. UK guidelines for the management of acute pancreatitis: is it time to change? *Gut* 2005;**54**:1344–5.
2. **Bradley EL III**. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Georgia, United States; September 11–13, 1992. *Arch Surgery* 1993;**128**:586–90.
3. **Malka D**, Hammel P, Lévy P, *et al*. Splenic complications in chronic pancreatitis: prevalence and risk factors in a medical–surgical series of 500 patients. *Br J Surg* 1998;**85**:1645–9.
4. **Lankisch PG**. The spleen in inflammatory pancreatic disease. *Gastroenterology* 1990;**98**:509–16.
5. **Mauro MA**, Schiebler ML, Parker LA, *et al*. The spleen and its vasculature in pancreatitis: CT findings. *Am Surg* 1993;**59**:155–914.
6. **Calvo E**, Cigüenza R, Alvarez-Sala JL, *et al*. Spontaneous rupture of the spleen in chronic pancreatitis: an often forgotten complication. *Gastroenterol Clin Biol* 1992;**16**:194.
7. **Fishman EK**, Soyer P, Bliss DF, *et al*. Splenic involvement in pancreatitis: spectrum of CT findings. *AJR Am J Roentgenol* 1995;**164**:631–5.
8. **Rypens F**, Deviere J, Zalcmán M, *et al*. Splenic parenchymal complications of pancreatitis: CT findings and natural history. *J Comput Assist Tomogr* 1997;**21**:89–93.
9. **Swirsky D**. The spleen and its disorders. In: Warrell DA, Cox TM, Firth JD, eds. *Oxford Textbook of Medicine*. 4th edn. Oxford: Oxford University Press, 2004:3609–16.
10. **Eowenfels AB**. Kehr's sign: a neglected aid in rupture of the spleen. *N Engl J Med* 1966;**274**:1019.
11. **Pillai JM**, Mitchener P, Smith S, *et al*. Pleuritic pain and a rare complication. *Postgrad Med J* 2002;**78**:436–7.
12. **MacKenzie KA**, Soiza RL. Spontaneous splenic rupture mimicking pneumonia: a case report. *Cases J* 2008;**1**:35.
13. **Brown J**, Msarchant B, Zumla A. Spontaneous subcapsular splenic haematoma formation in infectious mononucleosis. *Postgrad Med J* 1989;**65**:427–8.
14. **Croft DN**, Dawson JL. Spontaneous subcapsular splenic haematoma in acute malaria. *Postgrad Med J* 1962;**38**:123.
15. **Gandhi V**, Philip S, Maydeo A, *et al*. Ruptured subcapsular giant haematoma of the spleen—a rare complication of acute pancreatitis. *Trop Gastroenterol* 2010;**31**:123–4.
16. **Amonkar SJ**, Kumar EN. Spontaneous rupture of the spleen: three case reports and causative processes for the radiologist to consider. *Br J Radiol* 2009;**82**:111–13.
17. **Bernades P**, Baits A, Levy P. Splenic portal venous obstruction in chronic pancreatitis. *J Dig Dis Sci* 1992;**37**:340–6.
18. **Moosa AR**, Gadd MA. Isolated splenic vein thrombosis. *World J Surg* 1985;**9**:348–90.
19. **Patel VG**, Eltayeb OM, Zakaria M, *et al*. Spontaneous subcapsular splenic hematoma: a rare complication of pancreatitis. *Am Surg* 2005;**71**:1066–9.
20. **Kuramitsu T**, Komatsu M, Ono T, *et al*. Ruptured subcapsular giant hematoma of the spleen as a complication of chronic pancreatitis. *Intern Med* 1995;**34**:564–8.
21. **Tseng CW**, Chen CC, Chiang JH, *et al*. Percutaneous drainage of large subcapsular hematoma of the spleen complicating acute pancreatitis. *J Chin Med Assoc* 2008;**71**:92–5.
22. **Siu TL**. Percutaneous drainage of spontaneous subcapsular hematoma of the spleen complicating chronic pancreatitis. *Surgeon* 2004;**2**:52–5.
23. **Thompson JE Jr**, Ashley SW. Subcapsular hematoma of the spleen associated with acute pancreatitis. *Surgery* 1997;**121**:231–3.
24. **British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group**. British Thoracic Society guidelines for the management of suspected acute pulmonary embolism. *Thorax* 2003;**58**:470–83.
25. **Galipienzo J**, García de Tena J, Flores J, *et al*. Safety of withholding anticoagulant therapy in patients with suspected pulmonary embolism with a negative multislice computed tomography pulmonary angiography. *Eur J Intern Med* 2010;**21**:283–8.
26. **Perrier A**, Desmarais S, Goehring C, *et al*. D-dimer testing for suspected pulmonary embolism in outpatients. *Am J Respir Crit Care Med* 1997;**156**:492–6.

Copyright 2012 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Purushothaman K, Borowski DW. Unusual presentation of spontaneous splenic haematoma due to severe pancreatitis: a cautionary tale. *BMJ Case Reports* 2012;10.1136/bcr-2012-007271, Published XXX

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow