

NON-TRAUMATIC SPLENIC RUPTURE SECONDARY TO PANCREATIC ADENOCARCINOMA

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INTRODUCTION

Splenic rupture is a rare presentation of pancreatic cancer and may be challenging to diagnose in the acute setting. In this poster, we present a case of splenic rupture secondary to a pancreatic adenocarcinoma invading the splenic vein. The pathophysiology underlying the splenic rupture is discussed as well as management options.

CASE PRESENTATION

A 55-year old man presented with severe left upper quadrant pain without a prior history of trauma. The initial CT was reported as showing a pancreatic mass but no splenic abnormality was described. One day later, the patient became haemodynamically unstable with a 4 unit drop in haemoglobin. A repeat CT was performed – this time showing large volume haemoperitoneum (Figure 1) and a small capsular breach. The pancreatic mass was again noted, together with a dilated pancreatic duct, pancreatic atrophy and splenic vein occlusion. These findings were consistent with a pancreatic adenocarcinoma invading the splenic vein, resulting in venous engorgement of the spleen and finally, rupture. Retrospective review of the initial CT showed that a small subcapsular haematoma had been present but was missed (Figure 2). The patient underwent an emergency splenic artery embolisation to reduce arterial inflow to the spleen. Haemodynamic stability was achieved with gradual resolution of the haemoperitoneum on subsequent CTs. However, follow-up imaging also revealed increase in size of the pancreatic mass along with new liver metastases (Figure 3). Palliative chemotherapy was subsequently instituted.

DISCUSSION

The etiology of non-traumatic splenic rupture can be broadly divided into infectious, haematological, neoplastic, metabolic and rheumatological causes, in order of reported incidence. In our case, splenic vein invasion and occlusion by a pancreatic tumour with sparing of the splenic artery resulted in a mismatch between arterial inflow and venous outflow. This led to increased intraparenchymal pressure followed by a subcapsular haematoma and eventually splenic rupture. Splenic vein occlusion usually results in gastrosplenic collaterals, portal gastropathy and gastric varices (often fundal). These were not present in our case. It is also worth noting that direct invasion of the spleen by a pancreatic mass can also result in splenic rupture. In a haemodynamically unstable patient, management options include splenic artery embolisation or a splenectomy. A non-selective splenic artery embolisation was performed in this case due to the presence of haemodynamic instability.

In conclusion, splenic rupture in the absence of trauma should always prompt a search for an underlying cause. When reporting a non-traumatic splenic rupture, radiologists should actively exclude concomitant pancreatic pathology.

REFERENCES

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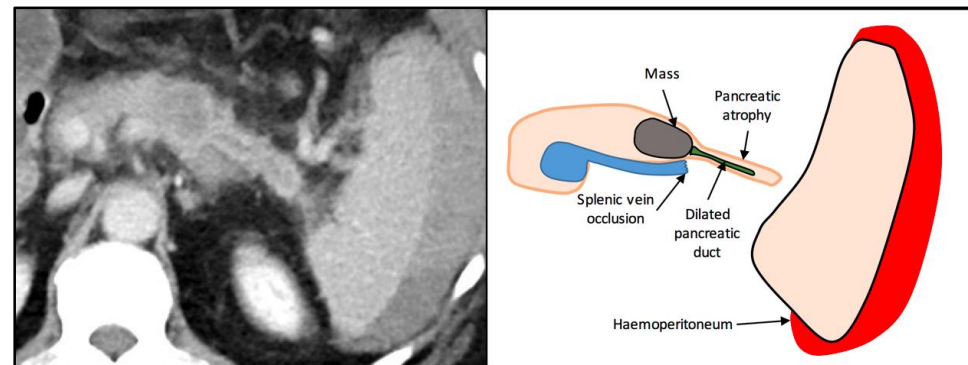


Figure 1. Axial portal venous phase CT showing a hypodense mass in the mid-body of the pancreas – resulting in splenic vein occlusion, pancreatic duct dilatation and parenchymal atrophy. There is a large volume haemoperitoneum as a result of venous engorgement from the splenic vein occlusion.

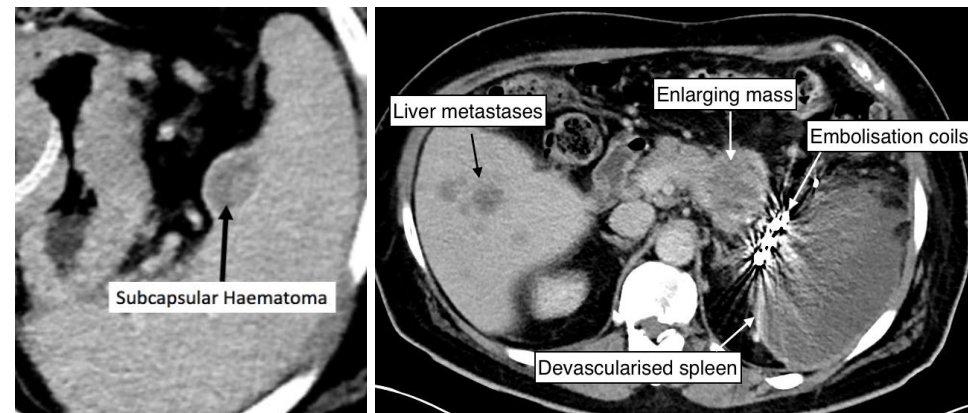


Figure 2. CT performed at presentation shows the splenic subcapsular haematoma which was initially missed.

Figure 3. Follow-up CT shows progressive increase in size of the pancreatic mass and new liver metastases. A completely devascularised spleen is noted following splenic artery embolisation.