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Case report

Hepatic infarction and acute pancreatitis: a case report and review of the literature

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Abstract

Hepatic infarction is rare due to the unique dual hepatic blood supply from the hepatic artery and the portal vein. Herein, we report a case of hepatic infarction that occurred as a complication of acute pancreatitis. The patient was a 58-year-old male with past medical history of chronic alcoholism, who presented with epigastric abdominal pain, nausea, and vomiting. Hepatic infarction was diagnosed with computed tomography of the abdomen and pelvis without contrast, which revealed suspicion of splenic vein thrombosis and peripancreatic fat stranding along with a wedge-shaped, peripheral hypo density in the right hepatic lobe with typical morphology for hepatic infarction.

Key words: outcome, hepatic infarct, acute pancreatitis.

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Introduction

The release of inflammatory markers and digestive enzymes makes acute pancreatitis a systemic disease. Presence of pro-coagulant inflammatory mediators, stasis, vessel spasm and mass effects from the surrounding inflamed pancreas causes thrombosis in acute pancreatitis [1]. Hepatic infarction is rare due to liver's unique dual blood supply, however, may occur from thrombosis of hepatic artery and/or portal vein. We present a unique case of hepatic infarction from acute pancreatitis with splenic vein thrombosis.

Case report

A 58-year-old patient with chronic alcoholism presented with epigastric pain, nausea, and vomiting. His vital signs revealed tachycardia, tachypnoea, and hypotension. On examination, he appeared to be in distress, and abdominal distension with tenderness over the epigastric region was observed. Laboratory work showed leukocytosis, thrombocytopaenia, acute kidney injury, and lactic acidosis. His lipase was elevated at 130,000 U/l. His liver chemistries were significant for bilirubin 2.4 mg/dl, aspartate transferase (AST) 322 U/l, alanine transferase (ALT) 125 U/l, and alkaline phosphatase of 93 U/l. His coagulation factors were pertinent for INR 1.5, prothrombin time 17.7 seconds, Aptt 17 seconds, fibrinogen 506 mg/dl, and fibrin degradation products (FDP) 20-40. A computed tomography (CT) of the abdomen and pelvis without contrast revealed peripancreatic fat stranding along with a wedge-shaped, peripheral hypodensity in the right hepatic lobe with typical morphology for hepatic infarction along with necrotising pancreatitis (Figs. 1-2). Splenic vein hyperdensity observed on the CT led to suspicion of splenic vein thrombosis, but there was no evidence of portal vein thrombosis. Doppler ultrasound of the liver confirmed patency of the hepatic vessels. CT-guided peripancreatic abscess aspiration grew *Enterococcus* and *Enterobacter cloacae*. Intravenous (IV) piperacillin-tazobactam was initiated until he underwent a pancreatic necrosectomy after 90 days. Serial CT scans during the course of his hospitalisation confirmed gradual resolution of the hepatic infarction as his pancreatitis was resolving. Other aetiologies such as hepatitis B were excluded.

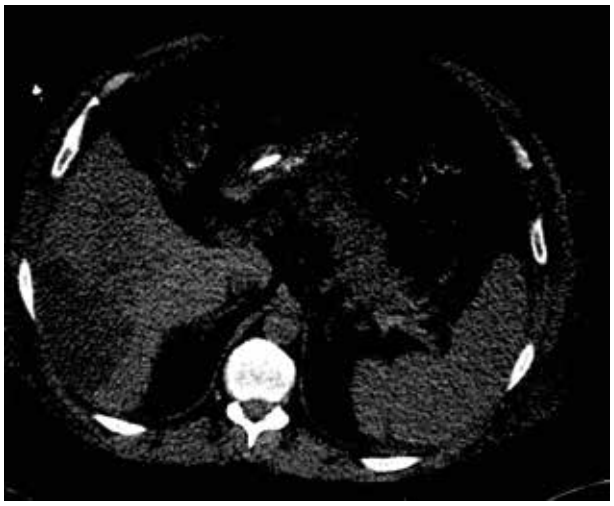

Fig. 1. Noncontrast transverse section of the computed tomography scan depicting wedge-shaped, peripheral hypo-density in the right hepatic lobe with the typical morphology for hepatic infarction

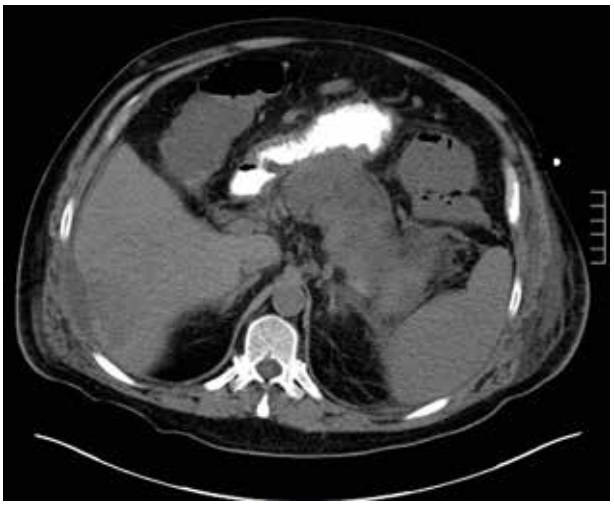

Fig. 2. Noncontrast transverse section of computed tomography scan depicting peri-pancreatic fat stranding, along with necrotising pancreatitis

Unfortunately, his hospital course was complicated by septic shock, acute respiratory distress syndrome, upper gastrointestinal bleeding, and acute renal failure, ultimately resulting in his death.

Discussion

Hepatic infarction is a rare phenomenon due to the unique dual blood supply of the liver, provided by the hepatic artery and portal vein. Some of the described aetiologies include liver transplantation [2], chemoembolisation of the hepatic artery [3], blunt abdominal trauma resulting in portal vein and hepatic artery thrombosis [4], antiphospholipid syndrome [5], and sickle cell disease [6].

Pancreatitis has also been reported with hepatic infarcts, although chronic pancreatitis has been identified more commonly. One patient with chronic pancreatitis developed extensive hepatic infarction from thrombosis of portal venous radicles along with portosystemic shunting and systemic hypotension [7]. Portal vein thrombosis resulted in liver infarction in another patient with chronic pancreatitis [8], whereas thrombosis of both splenic vein and portal vein resulted in multiple hepatic infarcts in another patient with chronic pancreatitis [9].

As mentioned above, acute pancreatitis can also lead to thrombosis that can affect the splanchnic venous system. The splenic vein, either isolated or in combination with the portal vein or superior mesenteric vein, is the most commonly thrombosed vessel [10]. However, acute pancreatitis appears to be the culprit leading to hepatic infarction in the literature only twice previously. In Japan, a patient with acute pancreatitis developed portal vein and hepatic vein thrombosis that subsequently resulted in infarction of the liver [11]. Necrotising pancreatitis was found in one patient as the aetiology of hepatic infarction in a study done in Italy reviewing 23 patients with hepatic infarction over a 10-year period [12].

Our case suggests that hepatic infarction should be considered in a patient with acute pancreatitis. No specific management strategies have been effective and the underlying condition should be treated appropriately.

Disclosure

Authors report no conflict of interest.

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