CASE REPORT

Cullen’s sign in amoebic liver abscess

A Misra, D Agrahari, R Gupta

A 45 year old woman was admitted to our hospital with a two week history of pain in the right upper abdomen and fever. On examination she was febrile and had mild pallor but no icterus; her right hypochondrium was tender and bowel sounds were normal. Cullen’s sign was present (see fig 1).

Investigations showed haemoglobin 90 g/l (reference range 120–160), total lymphocyte count 14.6 × 10⁹/l, differential leucocyte count polymorphs 87%, leucocytes 10%, and eosinophils 3%, platelets 240 × 10⁹/l (13–400), serum urea concentration 15.6 mmol/l (2.5–6.7), creatinine 247.5 µmol/l (<133), bilirubin 15.4 µmol/l (5.1–17), alkaline phosphatase 682 IU/l (37–147), alanine aminotransferase 48 IU/l (<35), and a prothrombin time of 16 seconds against control of 14 seconds (international normalised ratio 1.15). Chest radiography in the erect posture did not show any gas under the diaphragm.

Ultrasound of the abdomen revealed a 100 mm hypoechoic lesion in the right lobe of the liver with a small subhepatic collection. A diagnosis of ruptured ectopic pregnancy was suggested and a catheter was placed in situ draining the abscess cavity. Around 40 ml creamy pus was removed at that time. The subhepatic collection was aspirated under ultrasound guidance. Around 40 ml white creamy pus was taken out.

The patient was treated with intravenous metronidazole 0.5 g three times daily and intravenous cefotxime 1 g four times daily. Her condition improved quickly and clinical and biochemical parameters normalised in three days. The catheter continued to drain for 11 days, and it was then removed. The subhepatic collection also resolved. Her amoebic serology was positive.

Cullen’s sign slowly disappeared over two weeks.

DISCUSSION

This patient had ruptured an amoebic liver abscess, which was successfully managed by percutaneous catheter drainage. She had Cullen’s sign. In the literature there is no mention of Cullen’s sign in relation to liver abscess.

Although Hofstatter first reported the occurrence of periumbilical ecchymosis in 1909, he did not relate it to any underlying disorder, and it was Cullen (1918) who first recognised its relevance to ruptured ectopic pregnancy.⁴ A similar discoloration in the flanks was also described in acute pancreatitis by Turner in 1920, and it bears his name.⁵ Haemorrhagic pancreatitis and ruptured ectopic pregnancy are the conditions usually associated with Cullen’s sign but it has also been described in association with retroperitoneal haemorrhage, splenic rupture, and leaking aortic aneurysm.⁶ In fact any process causing haemoperitoneum may cause periumbilical ecchymosis.

Cullen’s sign has rarely been described in relation to liver diseases. In 1922 Sternberg reported its occurrence in association with adenocarcinoma of the liver, and it was described in a case of possible cirrhosis with portal hypertension by Hallendal in 1934.⁷ Mabin described it in two cases of hepatocellular carcinoma both having haemorrhagic ascitic fluid.⁸ Metastatic hepatic lesions and intra-abdominal non-Hodgkin’s lymphoma have also been reported with Cullen’s sign.⁹ Cullen’s sign in liver abscess has not been reported previously.

This sign occurs due to accumulation of blood in subcutaneous tissues. A test with Perls’ stain shows a large amount of haemosiderin in subcutaneous tissues indicating free blood in periumbilical tissues.⁶ The proposed mechanism of accumulation of blood is diffusion along fascial planes or via the falciform ligament secondary to intraperitoneal or retroperitoneal haemorrhage and has been associated with ruptured ectopic pregnancy and haemorrhagic pancreatitis.¹ This sign has rarely been described except under those conditions. We report its occurrence in a patient with amoebic liver abscess.

Summary points

• Cullen’s sign has been described in association with the conditions causing haemoperitoneum—for example, haemorrhagic pancreatitis, ruptured ectopic pregnancy, etc.
• Cullen’s sign was seen in this case of amoebic liver abscess; this has not been described previously.
• Inflammation may be the causative mechanism.
bleeding. In this case there was no intraperitoneal haemorrhage, neither was there any obvious bleeding diathesis. Platelet counts and prothrombin time were normal. During the process of abscess formation with destruction of hepatic parenchyma and sinusoids there may have been extravasation of blood, causing the Cullen’s sign.

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Authors’ affiliations
A Misra, R Gupta, Gastroenterology Unit, Department of Medicine, MLN Medical College, Allahabad, India
D Agrahari, Department of Surgery

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