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SPONTANEOUS TRANSHEPATIC RUPTURE OF THE GALLBLADDER WITH MASSIVE HEMOPERITONEUM

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Perforation of the gallbladder has an incidence of 1.6 to 2.8% in gallbladder disease. Among these cases transhepatic rupture is a very rare event. We report a case associated with secondary massive hemoperitoneum, free gallstone spillage, partial portal vein thrombosis and secondary pulmonary embolism.

Key-words: **Gallbladder, perforation.**

Transhepatic rupture of the gallbladder is rare, with fewer than 20 cases reported in the literature (1).

We report an unusual case in which the patient initially presented with right basic thoracic pain evocating pulmonary embolism. The definite emergency diagnosis was obtained during pulmonary angio-CT.

Case report

A 68-year-old patient presented in the emergency department with complaints of retrosternal and epigastric pain. Pain had suddenly led the patient to hypotensive syncope at home. There was a history of increasingly more frequent episodes of epigastric pain evolving for several days and for which the family practitioner had prescribed proton pump inhibitor for a week.

At physical examination tenderness was found in the right upper quadrant and in the epigastric area but pulmonary examination appeared normal. Laboratory tests revealed a CRP level 118 mg/l (nl < 5 mg/l), D-Dimeres were > 4.0 mg/L (nl < 0.5), Got at 140 U/l (nl < 40), GPT at 118 U/l (nl < 41).

Because of the association of an episode of hypotensive syncope at home and a retrosternal pain with elevated D-Dimeres, the patient was first considered as having pulmonary embolism. Pulmonary angio-CT was performed (not illustrated) and fresh clots were found in about 30% of the segmental branches of the right lower pulmonary artery. On the lower views of CT (not illustrated) ascite was found in both upper quadrants. Subphrenic stratified fresh blood clots were also visible in the hepatic area. For these reasons the patient was readmitted in the radiologic department for complementary abdominal CT.

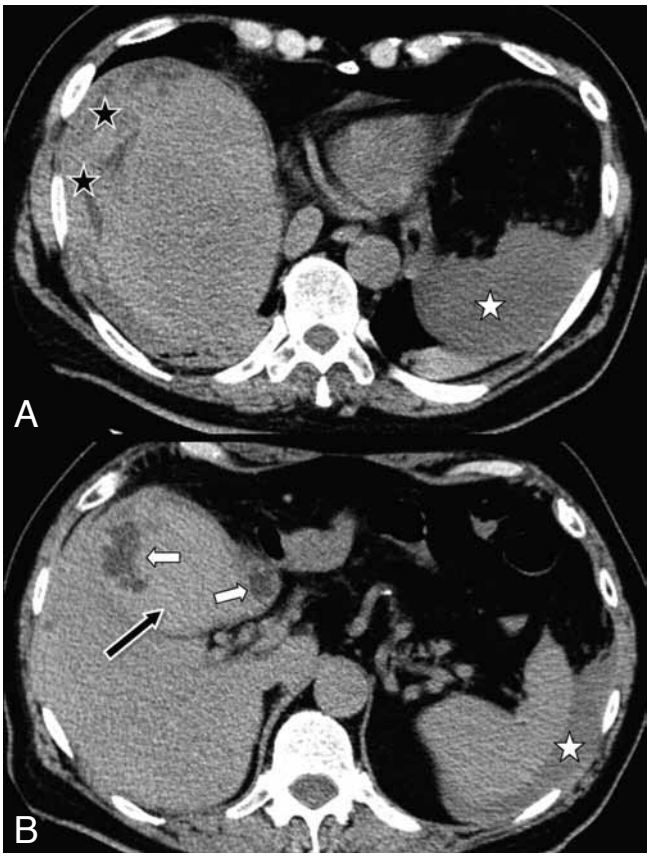

Fig. 1. — A, B: unenhanced abdominal CT views show stratified perihepatic fresh blood clots (black stars). Free ascitic fluid is found in the left perisplenic space (white star). The distended gallbladder (black arrow) appears spontaneously dense due to the presence of fresh blood. Groups of radiolucent gallstones are spontaneously visible in the hemorrhagic bile (small white arrows).

On the "almost" unenhanced views (Fig. 1) – pulmonary angio-CT having been performed only 30 minutes before – stratified fresh blood clots were confirmed in the perihepatic space. A very dense and distended hemorrhagic gallbladder was also found. Numerous radiolucent hypodense gallstones were seen floating in the dense hematic bile. On contrast enhanced MPR views (Fig. 2) a large defect was diagnosed in the hepatic side of the thickened wall of the hemorrhagic gallbladder.

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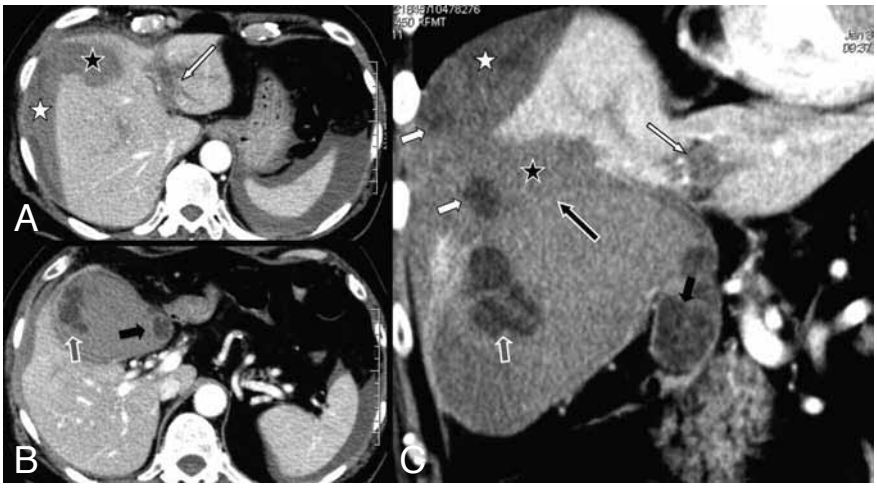

Fig. 2. — Axial CT views (A,B) and coronal MPR view (C) obtained after contrast enhancement. A large defect appears in the hepatic side of the thickened wall of the hemorrhagic gallbladder (black arrow). This defect appears in close relation with an hypodense area of dilacerated hepatic parenchyma (black star) in communication with the perihepatic clots (white star). A group of radiolucent hypodense gallstones obstruct the infudibulum (small black stars). Another is found in the fundus of the gallbladder (grey arrows). Free stone spillage has produced in the dilacerated hepatic parenchyma and in the perihepatic space (small white arrows). There is a thrombosis of the left intrahepatic portal vein (long white arrow).

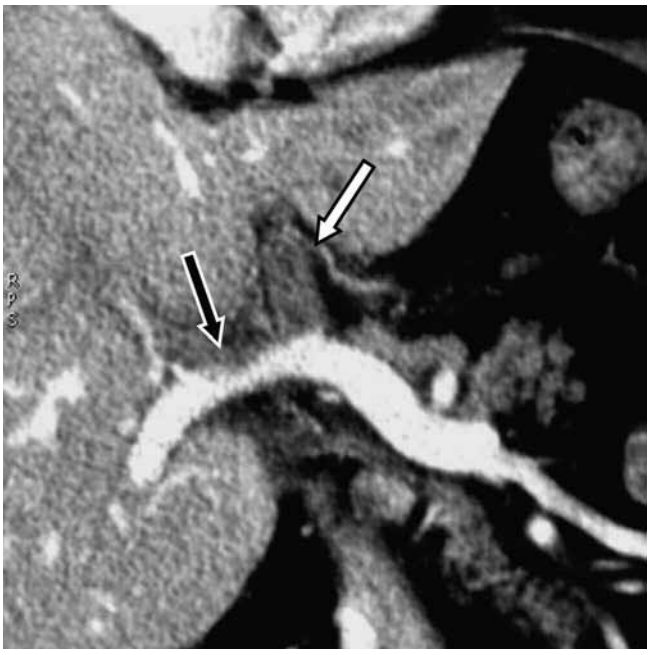

Fig. 3. — Coronal oblique MPR view illustrating the partial thrombosis of the hilar portal vein (black arrow) and the complete thrombosis of the left intrahepatic portal vein (white arrow).

This defect appeared contiguous to a hypodense area of dilacerated hepatic parenchyma in close relation with the perihepatic fresh clots. Radiolucent hypodense gallstones were also found obstructing the infudibulum. Others were visible in the fundus of the gallbladder and free gallstone spillage had also produced in the dilacerated hepatic parenchyma and in the perihepatic space. Additionally a thrombosis of the left intrahepatic portal vein was found (Fig. 2B and 3).

The radiological diagnosis of spontaneous transhepatic hemorrhagic rupture of the gallbladder caused by gallstone obstruction was proposed.

Emergency laparotomy was performed. A large hemoperitoneum was present and more than 1, 7 liter of blood was aspired. The right perihepatic space was packed with clots and during aspiration a large tear was found in the hepatic parenchyma at the level of the segment 6. This tear was in close relation with a perforation of thickened inflammatory gallbladder wall. Multiple spilled gallstones were found in the right upper quadrant. The partial thrombosis of the portal vein was considered to be the result of the septic dilaceration through the liver. Similarly the partial pulmonary embolism was considered as a collateral damage due to the migration of clots from sushepatic veins.

Discussion

In the presence of fresh perihepatic hematoma on emergency abdominal CT various hypotheses can be proposed included hepatic tumor (carcinoma or adenoma), a gallbladder cause or rupture of an arterial branch in the hepatic area (hepatic, cystic or pancreatic artery) (2).

Perforation of the gallbladder occurs in 1.6-2.8 % of cases of gallbladder disease (3) and is one of the most life-threatening complications of acute cholecystitis with a reported prevalence of 2-11% (4-5).

Risk factors include old age, male gender, previous cholecystitic attacks, association of severe systemic diseases in the same patient, arteriosclerosis, immunosuppression or prolongated corticoterapy (4, 6). The proposed mechanism of gallbladder perforation is stone impaction in the infudibulum or cystic duct, which leads to retention of secretion from mucus glands and distention leading to vascular compromise, followed by necrosis and perforation. During this process bleeding can occur that results in hemorrhagic cholecystitis with hemoperitoneum, a rather rare event (5-6).

A gallstone impaction resulting in gallbladder distention and followed by massive transhepatic hemorrhagic rupture with hemoperitoneum and gallstone spillage was the proposed physiopathologic sequence for the reported case. Secondary partial pulmonary embolism was considered being caused by the migration of clots from suprahepatic veins to the inferior vena cava.

In cases of bleeding of a perforated gallbladder, the source of bleeding is usually the gallbladder wall or the cystic artery, due to local extension of the inflammatory process, leading to focal necrosis, rupture and bleeding (1). Very rarely however – as seen in the reported case – the major cause of hemorrhage is the liver parenchyma through which the gallbladder has eroded (1, 4-5).

Rare cases of perforation of the gallbladder due to blunt injuries to the abdomen with sometimes delayed presentation have also been reported (8-9).

Finally the different causes of hemorrhagic cholecystitis are numerous and frequently associated : gallstones (50% of cases), anticoagulotherapy (5-6, 10) or coagulopathy, gallbladder cancer, hepatic or cystic aneurysm, trauma, portal hypertension, corticotherapy. A gallbladder tumor or an aneurysm of the cystic artery may also rupture into the gallbladder (2, 5).

Gallbladder hemorrhage may first be evacuated through the cystic canal causing painful acute obstructive jaundice and/or with subsequent hematemesis. On the other side when this canal is not permeable because obstructed by a gallstone or by blood clots, hemorrhage may distend the gallbladder and finally provoke rupture with free hemoperitoneum (5-6). The gallbladder fundus is the most common site of perforation due to poor vascular supply. Sometimes as in the reported case the rupture may produce directly through the hepatic parenchyma (2-4).

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