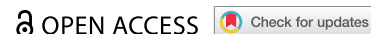


CASE REPORT



An infrequently encountered case of spontaneous subcapsular liver hematoma with hepatic artery pseudoaneurysm

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ABSTRACT

Hepatic artery pseudoaneurysm (HAP) is a rare complication of liver trauma and liver transplant, and spontaneous subcapsular liver hematoma is not frequently encountered outside the setting of preeclampsia and hemolysis, elevated liver enzyme and low platelet (HELLP) syndrome. We report a rare case of spontaneous subcapsular liver hematoma with hepatic artery pseudoaneurysm without any apparent liver trauma or recent interventional procedures of the hepatobiliary system. Although subcapsular hepatic hematoma and HAP are uncommon diagnoses, clinicians should be aware of these diagnoses to promptly diagnose and effectively treat them. Clinicians should also not forget these diseases could be masked by other common etiologies, such as gastritis.

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Hepatic hematoma; hepatic artery pseudoaneurysm; angioembolization; blood loss anemia; abdominal pain; transaminitis

1. Introduction



Hepatic artery pseudoaneurysm (HAP) is a rare complication of liver trauma and liver transplant, and spontaneous subcapsular liver hematoma is not frequently encountered outside the setting of preeclampsia and HELLP syndrome [1,2,3]. The incidence of HAP is estimated to be about 0.002% and is usually associated with percutaneous and endoscopic interventional procedures [4,5]. The incidence of subcapsular hematoma is 1/40,000 to 1/250,000 deliveries, but the incidence among those who are not pregnant has not been studied [6]. We report an infrequently encountered case of subcapsular liver hematoma with hepatic artery pseudoaneurysm without any apparent liver trauma, preexisting liver conditions, coagulopathy or interventional procedures of the hepatobiliary system.

2. Case description

A 66-year-old female presented to the emergency department with sudden onset of diffuse abdominal pain for 1 day. The pain was severe, constant, dull/achy, non-radiating, and aggravated by palpation. She also had nausea, multiple episodes of bilious vomiting, diarrhea, and melena. The patient denied fever, chills, hematemesis, or hematochezia. She had been on ferrous sulfate 325 mg daily for chronic microcytic anemia and pantoprazole 40 mg twice daily for gastroesophageal reflux disease at home. On admission,

her blood pressure was 76/38 mm Hg, heart rate 98 bpm, respiratory rate 16/minute, and temperature 98.8 Fahrenheit. On physical examination, she had lower abdominal tenderness to palpation with rebound tenderness. The examination of the heart and lungs was normal. Laboratory results are as follows: hemoglobin 11.2 g/dL, white blood cell count 18,470/ μ L, platelet count of 450,000/ μ L, PT 11.9 seconds, INR 1.05, sodium 127 mmol/L, potassium 5.4 mmol/L, chloride 95 mmol/L, bicarbonate 23 mmol/L, BUN 40 mg/dL, creatinine 2.07 mg/dL, glucose 155 mg/dL, AST 31 units/L, ALT 8 units/L, alkaline phosphatase 220 units/L, and total bilirubin 0.3 mg/dL. Lactate was elevated at 3.4 mmol/L. She tested negative for hepatitis B and C. A fecal occult blood test was positive. Her initial CT abdomen/pelvis without contrast on admission showed mild wall thickening of the stomach, likely from gastritis. The patient was started on intravenous fluid, pantoprazole, and piperacillin/tazobactam to treat her possible colitis. Colorectal surgery was consulted, and on third day of hospitalization, she underwent an esophagogastroduodenoscopy and flexible sigmoidoscopy, which showed partial gastric outlet obstruction/severe gastritis and diverticulosis with partial stricture in the sigmoid colon, respectively.

From hospitalization day 2 to 4, her blood pressure was elevated, ranging from SBP 140s to 180s and DBP 70s to 100s. On the fourth hospitalization day, she developed hypotension (BP 61/38) and was transferred to the intensive care unit (ICU) for closer

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observation. Her ICU stay was uncomplicated, and she was transferred out to the floor in 1 day. On the 6th hospitalization day, however, she started developing worsening abdominal pain, along with worsening leukocytosis and anemia, which required 2 units of packed RBC transfusions. She also developed transaminitis with AST 669 units/L, ALT 374 units/L, ALP 130 units/L, and total bilirubin 0.3 mg/dL. A repeat CT abdomen/pelvis with contrast was performed on

sixth day, and it showed a new large 5.28 cm subcapsular hematoma, surrounding the right lateral liver margin with displacement of the liver caudally into the left with small free intraperitoneal fluid collections, and a 1.5 × 1 cm pseudoaneurysm (Figure 1). The patient was treated with embolization of the pseudoaneurysm, located at the right hepatic artery (Figure 2). The patient's abdominal pain improved, and her hemoglobin remained stable at around 8 g/

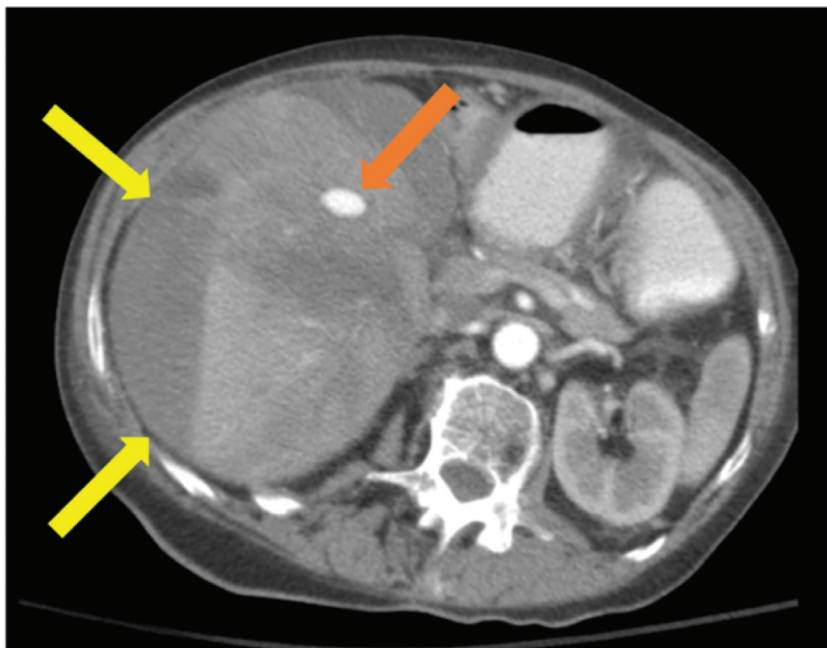


Figure 1. Computed tomography (CT) of the abdomen and pelvis with intravenous contrast showing subcapsular hematoma (Yellow arrows) and the dense arterial enhancement depicting pseudoaneurysm (Orange arrow).

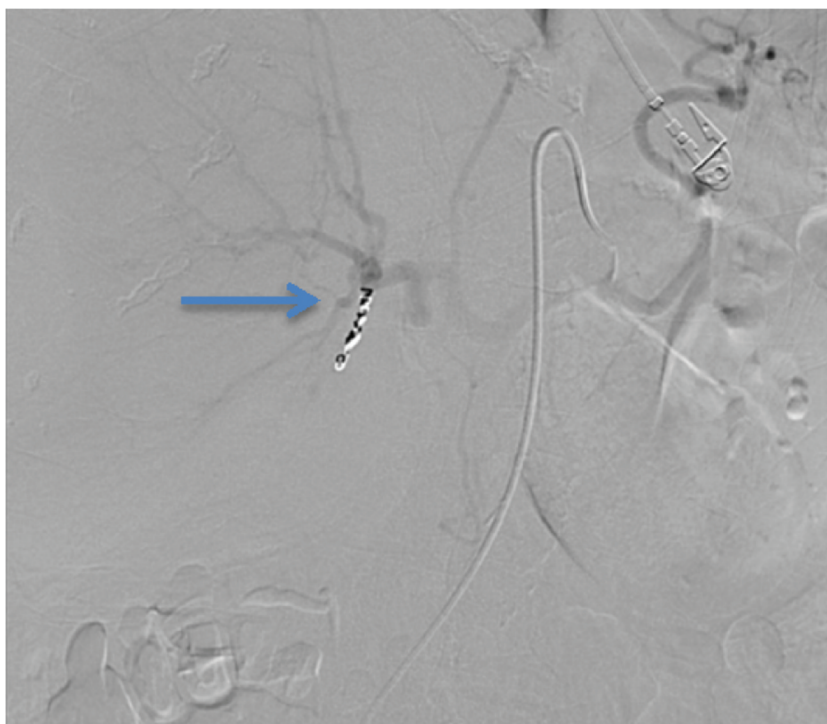


Figure 2. Embolization coil deposited within the hepatic artery branch leading to the pseudoaneurysm (Blue arrow).

dL for the next 10 days in the hospital. Prior to discharge, her hemoglobin was 8.2 g/dL, WBC 9,190/ μ L, platelets 700,00/ μ L.

3. Discussion

A subcapsular liver hematoma is an uncommon disease that is described as blood between the Glisson capsule and the hepatic parenchyma [7]. A hepatic artery pseudoaneurysm (HAP) is the dissection of the hepatic artery wall between the tunica media and the tunica adventitia, and the incidence is increasing, as there are more patients undergoing percutaneous and endoscopic interventional procedures of the hepatobiliary system [5]. There has been no association between subcapsular liver hematoma and HAP, but both diseases have been associated with a drop in hemoglobin levels, which leads to their high mortality rates [8,9,10,11]. Subcapsular liver hematoma commonly presents as abdominal pain, but HAP could present asymptotically [5,12]. Subcapsular liver hematoma can be easily diagnosed with CT of the abdomen and pelvis, and HAP is diagnosed with CTA of the abdomen and pelvis [5,12]. Management of both subcapsular liver hematoma and HAP has been a challenge. For subcapsular liver hematoma, conservative treatment is common when patients have hemodynamic stability, but if there is hemodynamic instability or the hematoma rapidly expands with decreasing hemoglobin levels, angioembolization should be attempted [3,13,14]. If angioembolization fails or patient is hemodynamically unstable, surgical intervention would be considered [14]. For HAP, as in our case, embolization of the affected artery is the initial choice of management, as it is better at controlling bleeding, requires less transfusions, and correlates to a shorter duration of hospitalization [15,16].

Our patient was admitted with an unspecific, diffuse, severe abdominal pain. She did not have common risk factors for subcapsular liver hematoma (pregnancy – preeclampsia or HELLP) or HAP (percutaneous or endoscopic procedures of the hepatobiliary system). There was a case report of liver hematoma in the left lobe of the liver after laparoscopic Nissen fundoplication from retraction injuries, but the patient in our case had the surgery 2 years ago and has a hematoma in the right lobe of the liver [17]. She initially presented with hemodynamic instability and anemic hemoglobin levels, but the diagnosis could not be made initially as the diagnosis was masked by partial gastric outlet obstruction/severe gastritis and diverticulosis with partial stricture in the sigmoid colon, and the initial CT of the abdomen and pelvis did not show any abnormalities. However, close monitoring of the patient enabled the team to order a repeat CT of the abdomen and pelvis with

and without contrast and diagnose the patient with subcapsular liver hematoma and HAP. Angioembolization stabilized her hemoglobin levels.

4. Conclusion

Spontaneous subcapsular hepatic hematoma and concomitant presence of HAP are not routinely encountered in clinical practice. Liver hematoma can be an important differential diagnosis in cases with abdominal pain, blood loss anemia with or without signs of shock. Thus, clinicians should be aware of these diagnoses to promptly diagnose and effectively treat them in a timely manner.

Authors' contributions

All authors actively participated in patient management, literature search and write up of this case report. All authors read and approved the final version of this manuscript.

Disclosure statement

No potential conflict of interest was reported by the authors.

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