



Subcapsular Liver Abscess Can Present As Subcapsular Hepatic Hematoma: Case Report

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Abstract

Subcapsular liver abscesses are serious clinical issues that can lead to life-threatening complications. We report a case of subcapsular liver abscesses that required multiple modes of investigation before a diagnosis could be made. This case outlines the steps taken to diagnose and identify the cause of subcapsular abscesses but is unique because the investigation results showed a high level of suspicion for subcapsular liver hematomas.

Background

A 63-year-old male with a past medical history of hypertension, hyperlipidemia, restless leg syndrome, rheumatoid arthritis, and a family history of deep venous thrombosis (DVT), was admitted to the hospital due to a left sided DVT, bilateral pulmonary embolisms (PE), and a small pneumoperitoneum that resulted in abdominal pain. The pneumoperitoneum was treated supportively and self-resolved. A coagulation panel was performed and showed no pathologies and the patient was discharged on anticoagulation (5 mg daily of warfarin, 7.5 mg of warfarin on Tuesday and Thursday) and scheduled to follow up with anticoagulation clinic. Because the patient's INR of 1.6 at the first follow up was subtherapeutic, the warfarin dose was adjusted to take 7.5 mg of warfarin on Saturday instead of the initial 5 mg. Five days later, the patient had an INR of 9.9 and was instructed to hold his warfarin and follow up with the coagulation clinic. The next day in clinic, the patient reported 2 to 3 days of abdominal distension and sharp, diffuse abdominal pain that increased with movement and had no relieving factors. The patient was then re-admitted to the ED after 3 weeks of his last admission where he was found to have an INR of 12.8. The patient also had tachycardia, a fever of 101.5, and an elevated procalcitonin of 158.2 on admission for which no primary source of infection was found. A sepsis work up showed negative blood cultures, normal lactic acid, and no acute changes on X-ray, and the patient was started on a 14-day course of Vancomycin and Zosyn after which the fever quickly resolved. An abdominal CT scan was done and showed 2 adjacent subcapsular collection on the liver of 5 cm and 2.8 cm that were both interpreted by the radiology report to be hematomas. In addition, stranding with a small hemoperitoneum and hyperdensity within the inferior collection were concerning for rupture

of the hematomas and hemorrhage in the right lobe of the liver, right paracolic gutter, and the pelvis. Ultimately, the fluid collections were managed with observation due to their size and lack of growth. In addition, bowel rest and NG tube placement were recommended. Five days after admission, the INR normalized, and the patient was started on Eliquis. A follow up CT scan 3 days later showed enlargement of the fluid collections and Eliquis was discontinued. At this time the patient was sent for placement of an IVC filter due to the inability to tolerate anticoagulation. Subsequently, an IR guided drainage of the collection was performed which yielded 300 cc of turbid, creamy, yellow/pink discharge, raising suspicion for a subcapsular hepatic abscess rather than a hematoma. The drainage tube was placed with the intention of not removing it until the drainage stopped, and the patient felt significant relief upon drainage of the abscess.

In addition, cultures that were drawn from the fluid collection grew *Proteus mirabilis*. Once the subcapsular hematoma was ruled out, the patient re-started apixaban. An EGD/colonoscopy was scheduled to investigate the cause of the infection, and the patient was discharged on ciprofloxacin 500 mg PO for 14 days.

Discussion

Subcapsular hepatic fluid collections can be defined as fluid collections in the space deep to the liver capsule and superficial to the parenchyma that have not ruptured. The liver capsule consists of the thick, inner layer called the Glisson's capsule and an outer serous layer derived from the peritoneum. In this potential space between the Glisson's capsule and the liver parenchyma, blood, pus, bile, and other fluids may accumulate. The liver is unique in that it has several factors that make it vulnerable to a variety

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of pathological conditions in the hepatic capsular and subcapsular areas. These factors include the negative subdiaphragmatic pressure in the hepatic area that can draw infected material or malignant cells towards the diaphragm, the connection to extraperitoneal sites and neighboring viscera through the perihepatic ligaments, and the systemic slow of blood to the liver in addition to the portal and hepatic arterial blood flow. These distinct anatomical and hemodynamic conditions in the hepatic area result in susceptibility to inflammatory, infectious, and metastatic processes. It is important to note that while hepatic fluid collections most often present with minimal symptoms due to their size being limited by the subcapsular space, they still possess the potential to rupture and lead to hemorrhage or infection.

The case we present is unique due to the high level of suspicion of the fluid collections being hematomas before the IR guided drainage was performed. The patient in our case presented with abdominal pain with an INR that significantly increased after his anticoagulation regimen was altered. In addition, the fluid collection was observed to have expanded when the patient was started on Eliquis, further raising suspicion for a hematoma. In the presence of these findings, in addition to the fever and elevated procalcitonin, one of the diagnoses that was considered was the possibility of an infected subcapsular hematoma. However, there were no other signs of bleeding and there was a negative fecal occult blood test.

The second diagnosis that was considered was that of a pyogenic liver abscess. Similar to a liver hematoma, pyogenic liver abscesses can arise in the same anatomical area, but the accumulated fluid is primarily pus rather than blood. A liver abscess in this patient could have appeared due to the hematogenous spread of infection from an undiagnosed pneumoperitoneum. While the CT scan results showed suspicion of subcapsular hematomas, this may have been a misdiagnosis. The patient had a history of taking meloxicam for rheumatoid arthritis,

and NSAID use has been associated with gastrointestinal complications such as peptic ulcer development. The perforation of a peptic ulcer could have led to a pneumoperitoneum, leading to the complications seen in our patient. Although the patient's blood cultures were negative and no clear source of infection was found, the clinical improvement of the patient after initiation of antibiotics and drainage of the fluid collection further supports this theory.

Conclusion

In a situation in which the etiology of a fluid collection in the liver is unclear, diagnosis of subcapsular fluid collections is usually done by ultrasound or CT, with CT having more specificity for the etiology of the fluid collection. While this imaging is usually sufficient for diagnosis, our case is a prime example of how this mode of diagnosis is not definitive since the CT report was consistent with a diagnosis of subcapsular hematomas while the clinical presentation and improvement with treatment shows more support for the diagnosis of a liver abscess. Proving one diagnosis doesn't exclude other co-presented disorders so meticulous exclusions of all other potentials is mandatory before concluding the course of therapy.

In the management of subcapsular fluid collections, conservative treatment with observation is indicated in the absence of expansion of the collection or severe infection. In event that intervention is required, the management of hematomas and abscesses is slightly different. Both can initially be managed conservatively and eventually may require CT-guided percutaneous drainage in the event of expansion of the fluid collection. However, abscesses must also be managed with antibiotics after drainage. The difference in treatments highlights the importance of identifying the fluid collection because failure to treat with antibiotics can lead to more severe complications such as peritonitis.