

Concurrent Rectus Sheath Hematoma and Iliopsoas Hematoma in a Cirrhotic Patient

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Abstract

Spontaneous muscle hematomas are rare in patients with cirrhosis and are associated with a high mortality rate. Hemostatic imbalances associated with cirrhosis are very complex and include primary and secondary hemostasis defects. Here we report a case of bilateral rectus sheath hematoma (RSH) with unilateral iliopsoas hematoma in a cirrhotic patient and review the literatures reported cases.

Keywords: Rectus sheath hematoma; Cirrhosis; Anticoagulation; Hyperfibrinolysis

Introduction

Muscle hematomas typically occur in patients with various hemostatic abnormalities or patients receiving anticoagulation therapy, and are usually associated with minor trauma. Spontaneous muscle hematomas are not very common in patients with cirrhosis [1]. Muscle hematomas are associated with an increased mortality in patients with cirrhosis as compared to patients receiving anticoagulation therapy [1]. Muscle hematoma in patients with cirrhosis is closely associated with the use of alcohol [1]. Given the high mortality rate clinicians should be aware of this important complication.

Case Report

A 61 year old African American man with chronic hepatitis C, cirrhosis, chronic alcohol use and hypertension was brought to the emergency department complaining of severe abdominal pain which occurred only when moving or coughing. He had been lying on the floor for the past 2 days and was drinking wine to relieve the pain. The pain was described as diffuse, sharp and stabbing. He denied any nausea or vomiting or recent trauma. His only medication was amlodipine and he was not taking any anticoagulants or aspirin.

Physical exam revealed a Temperature of 99.4 degrees Fahrenheit, heart rate of 105 beats per minute, BP 110/74 mmHg and respiratory rate of 22 breaths per minute. He had no tenderness with palpation of the abdomen and bowel sounds were active in all quadrants but the abdomen was much distended.

The initial labs revealed a white blood cell count of $10.9 \times 10^6/L$ with neutrophils of 65%, Hemoglobin, 6.7 g/dL; mean corpuscular volume (MCV) 101 femtoliter, platelet count $108,000/mm^3$, Alb 2.3 g/L, aspartate aminotransferase (AST), 421 U/L; Alanine aminotransferase (ALT), 127 U/L; alkaline phosphatase (ALP), 154 U/L; international normalized ratio (INR), 2.2; partial thromboplastin time (PTT), 33 seconds; lipase, 125 U/L; amylase, 92 U/L; BUN, 22 mg/dL; Creatinine, 1.2 mg/dL; ammonia, 73.3 $\mu mol/L$; CK level of 3258 U/L. Stool sample was grossly positive for occult blood. Calculated MELD score was 17 at the time of presentation.

Sonography of the abdomen showed a small stone in the neck of gallbladder with no pericholecystic fluid and common bile duct was not dilated. A CT scan of abdomen and pelvis with contrast demonstrated a large hemorrhage within the rectus abdominis muscle bilaterally plus hemorrhage extending to the left psoas muscle (Figures 1 and 2). Treatment was started immediately with 8 units of fresh frozen plasma and 2 units of PRBC. The patient was also hydrated with normal saline.

After stabilizing the patient, he subsequently underwent an upper endoscopy which disclosed gastritis and two large esophageal varices which were subsequently banded. The hemoglobin remained stable at 8.2 g/dL and patient was subsequently discharged.

Discussion

Spontaneous muscle hematoma is an uncommon condition and is known complication in patients with coagulopathies or on medical anticoagulation [1-5]. Rectus sheath hematoma can occur spontaneously or after trauma. Although cirrhotic patients have abnormal coagulation studies, it is unclear whether this increases the risk of RSH in these patients [6,7]. Alcoholic patients are known to sustain frequent minor trauma during bouts of intoxication that they frequently don't recall, trauma may have actually been a trigger in our patient despite patient denying any trauma history. A PubMed search revealed that intramuscular hematoma is uncommon in cirrhotic patients and in a review of literature there were only 11 cases, including our case, that have been reported. Seven out of 11 patients had pure alcoholic cirrhosis; two had mixed viral and alcoholic cirrhosis, one viral and one cryptogenic cirrhosis. Six patients had RSH, three had

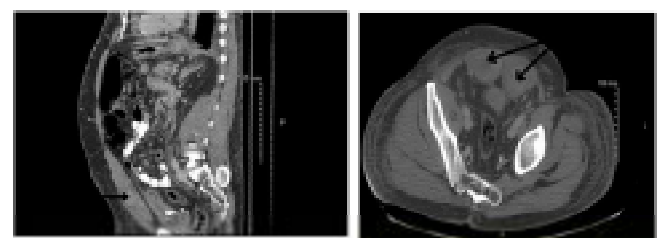


Figure 1: Rectus sheath hematoma (arrows) in sagittal and transverse plane.

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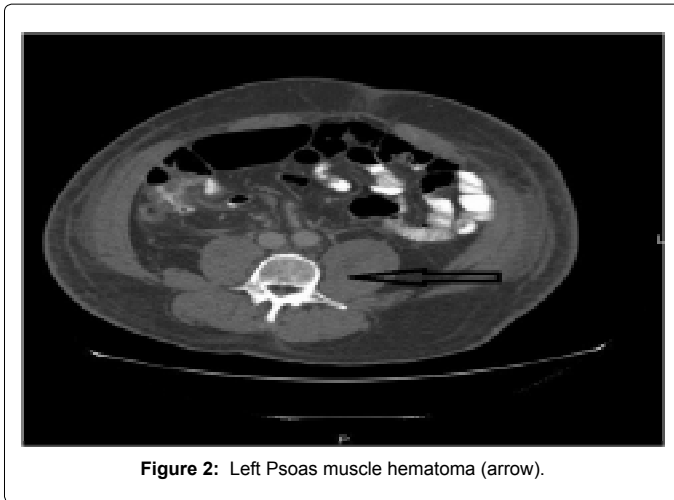


Figure 2: Left Psoas muscle hematoma (arrow).

iliopsoas hematoma, one had gluteus maximus & biceps femoris & pectoralis muscle hematoma and our case had mixed RSH and iliopsoas hematoma. MELD score was calculated on only 5 patients with a range between 16-29. Three patients had to have embolization of the bleeding vessel, one patient had to undergo liver transplantation and the remainders were managed medically [1,6,8].

It has been documented that the mortality rate of muscle hematoma in patients with cirrhosis is more than 70%. (75% [1], 100% [6] and 100% [8]). In contrast to patients with cirrhosis, Cherry & Mueller reported that RSH is rarely fatal in patients receiving anticoagulation therapy with a mortality rate of only 1.6% [3]. In cirrhotic patients, coagulopathy is characterized by coagulation factor deficiencies and accelerated fibrinolysis [9]. In these patients there is an increased endothelial release of tissue plasminogen activator (t-PA), decreased hepatic clearance, decreased plasminogen activator inhibitor and alfa-2 antiplasmin [9,10] and factor XIII [11]. In Spoerke et al. study, it was revealed that consumption of alcohol correlates with a slower rate of fibrin formation and slower rate of fibrin cross linking in alcoholic patients [12].

Mukamal et al. showed Fibrinogen, vWF levels and factor VII concentration were lowest among chronic heavy drinkers [13]. Similar results were seen in Wannamethee et al. study which showed decrease in plasma fibrinogen and vWF concentrations in heavy drinkers [14].

Recently, there is ongoing attention towards the thrombin activatable fibrinolysis inhibitor (TAFI), which is reduced in patients with chronic liver disease as a possible explanation for the hyperfibrinolytic state described in this setting. The role of hyperfibrinolysis in the occurrence of bleeding in cirrhotics is still unclear [15,16]. There are multiple medications in the literature such as epsilon amino caproic acid (EACA) which is a synthetic derivative of the amino acid lysine-binding site of fibrinogen and blocks the binding of fibrin [17], and aprotinin used for hyperfibrinolysis post liver transplant [18] which are used to treat cirrhotic patients with hyperfibrinolysis; more studies are needed to verify their effectiveness in treating muscle hematoma in cirrhotic patients. Assessing plasminogen activation and clot lysis may also assist to differentiate hyperfibrinolysis from DIC and other causes of bleeding in patient with cirrhosis.

The high mortality rate of spontaneous muscle hematoma in cirrhosis compared with patients receiving anti-coagulation therapy or hemophiliacs may be related to defects of secondary hemostasis, the

effect of alcohol on platelet aggregation, inhibition of platelet adhesion to fibrinogen or a combination of factors discussed above or may be related to poor nutritional status [1].

RSH can mimic a wide variety of intra-abdominal disorders and is frequently misdiagnosed initially. Failure to make a prompt diagnosis can lead to delay in treatment or unnecessary surgery [19].

Conclusion

Spontaneous muscle hematoma should always be kept in the differential diagnosis of abdominal pain in a patient with cirrhosis and anemia. Hemostatic defects associated with cirrhosis, namely defects in primary and secondary hemostasis, as well as fibrinolysis and DIC can cause bleeding in these patients. More studies are needed to determine whether EACA or aprotinin may be useful in the treatment of RSH in cirrhotic patients. Early detection of muscle hematoma in patients with cirrhosis is critical due to the high mortality rate, compared to non-cirrhotic patients, and may result in a significant decrease in the mortality rate. CT scanning may be used liberally in search of internal hematomas when other sources of bleeding have been excluded in patients with known or suspected coagulopathies.

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