CLINICAL VIGNETTE

Rectus Sheath Hematoma due to Rivaroxaban

Duong Tommy Hua, MD and Jaclyn Spiegel, MD

Case Report

A 63-year-old woman with a past medical history of asthma and morbid obesity presented to the emergency room with five days of shortness of breath, wheezing, and productive cough. She denied palpitations, chest pain, and dizziness. Her vitals on admission included temperature of 36.8 °C, heart rate 148/min, blood pressure 110/54 mm Hg, respiratory rate 30 breaths/min, oxygen saturation of 100% on BIPAP support with 50% FIO_2. Her weight was 138kg with Body Mass Index of 55.67 kg/m^2. Exam revealed irregular tachycardia, diffuse expiratory wheezes, and peripheral edema. Labs were notable for a hemoglobin of 15.6 g/dL and a respiratory viral panel that was positive for rhinovirus. Her chest x-ray showed cardiomegaly with interstitial edema. Electrocardiogram showed atrial flutter with rapid ventricular response (RVR). Echocardiogram showed a normal ejection fraction but evidence of moderate pulmonary hypertension and a dilated inferior vena cava.

The patient was treated for an asthma exacerbation, volume overload, and atrial flutter with RVR. She responded well to treatment with prednisone and ipratropium/albuterol, furosemide, and diltiazem, but remained hospitalized due to persistent hypoxia despite supplemental oxygen of up to 3 liters per minute and intermittent episodes of atrial flutter with RVR which were difficult to control despite use of high dose diltiazem. On hospital day five, cardiology was consulted for management of atrial flutter and added digoxin to her current regimen of diltiazem and daily rivaroxaban 20mg for stroke prophylaxis.

On hospital day nine, she developed another episode of atrial flutter with RVR for which she was given intravenous diltiazem and subsequently became hypotensive. She denied abdominal pain and her abdominal exam was benign. She was started on a dopamine drip and transferred to the Cardiac Care Unit where she then became obtunded and required intubation. Her hemodynamics continued to worsen requiring additional vasopressor support. Additionally, her hemoglobin significantly declined to 7.0 g/dL from 16.2 just three days prior. CT chest, abdomen, and pelvis with contrast were performed emergently to assess for a source of blood loss andrevealed a large rectus sheath hematoma measuring 25x20x8cm. Surgery was consulted, and recommended medical management. The patient continued to worsen with multi-organ failure and progressed to cardiac arrest and death on hospital day eleven.

Discussion

Rectus sheath hematoma (RSH) is an uncommon cause of abdominal pain or bleeding significant enough to lead to hemodynamic instability. In one study of 1,257 patients with acute abdominal pain, 1.8% were diagnosed with RSH.[1] The rectus sheath contains the rectus abdominis muscle, which is supplied by the epigastric arteries.[2] Damage to either the muscle or arterial supply leads to development of RSH.

In one study of 126 patients with RSH, the most common symptom was abdominal pain, which was seen in 84% of cases, followed by nausea or vomiting in 15% [3]. The same study, found 63% to have a palpable abdominal wall mass and 17% had abdominal wall ecchymosis. Patients with RSH may also have a positive Carnett's Sign (where abdominal pain is worsened or unchanged with tensing of abdominal wall) and a positive Fothergill's sign (where abdominal wall mass does not cross midline and remains palpable with tensing of abdominal wall).[2] Interestingly, only 55% of patients with RSH experienced a decrease in hemoglobin of >0.4 g/dL.[3]

Several risk factors for development of RSH have been described, including coughing, anticoagulant therapy, trauma, abdominal procedures, exercise, pregnancy, older age, and female gender.[2] Anticoagulation with warfarin, unfractionated heparin, or low-molecular-weight heparin is a well-described risk factor for RSH.[3] Theoretically, direct oral anticoagulants (DOACs) such as rivaroxaban could also be a risk factor for RSH, however data identifying rivaroxaban as a cause is limited.[4,5]

Diagnosing RSH can be difficult, particularly in morbidly obese patients like the patient presented here or those with other superficial abdominal anatomic abnormalities, but it is an important consideration in a patient with abdominal pain and a dropping hemoglobin. One study with 126 patients with RSH, found only 8.7% of cases diagnosed based on history and physical alone.[3] Most cases are diagnosed by CT scan with sensitivity and specificity for RSH approaching 100%, but ultrasound may be an alternative imaging modality with less sensitivity if CT scan is unavailable or unsafe.[2] However, in a morbidly obese patient, the limitations of physical exam are combined with suboptimal imaging like ultrasound in a patient with significant adiposity in the affected to the area.

RSH is usually managed conservatively as most cases are self-limited.[2,3] Overall, RSH carries a mortality of 4%, however in

patients on anticoagulation it could be as high as 25%.[6] Conservative therapy includes analgesics, local compression, rest, resuscitation with crystalloids or blood, and reversal of anticoagulation.[2] Aggressive management with embolization or surgery can be considered in patients who fail conservative therapy.[3]

Our patient had several risk factors for RSH, including her female gender, older age, significant coughing due to asthma exacerbation and rhinovirus, and anticoagulation with rivaroxaban. However, diagnosing RSH in this patient was difficult due to lack of abdominal pain and a benign abdominal exam. Her body habitus likely played a significant role in the difficulty to diagnose her condition, as physical exam was unreliable. However, her declining hemoglobin, hemodynamic compromise, and treatment with rivaroxaban prompted a CT scan, which definitively diagnosed her large RSH. Unfortunately, this patient's significant RSH led to hemorrhagic shock and death, which is still an uncommon yet more frequent outcome in anticoagulated patients. Although warfarin, unfractionated heparin, and low-molecular-weight heparin are established risk factors for RSH, anticoagulation with rivaroxaban should be considered as a risk factor for RSH as well.

REFERENCES

1. **Klingler PJ, Wetscher G, Glaser K, Tschmelitsch J, Schmid T, Hinder RA**. The use of ultrasound to differentiate rectus sheath hematoma from other acute abdominal disorders. *Surg Endosc*. 1999 Nov;13(11): 1129-34. PubMed PMID: 10556453.
2. **Hatjipetrou A, Anyfantakis D, Kastanakis M**. Rectus sheath hematoma: a review of the literature. *Int J Surg*. 2015 Jan;13:267-271. doi: 10.1016/j.ijsu.2014.12.015. Epub 2014 Dec 19. Review. PubMed PMID: 25529279.
3. **Cherry WB, Mueller PS**. Rectus sheath hematoma: review of 126 cases at a single institution. *Medicine (Baltimore)*. 2006 Mar;85(2):105-10. PubMed PMID: 16609349.
4. **Börekci E**. Rectus sheath hematoma and retroperitoneal bleeding due to rivaroxaban: a case report. *Afr Health Sci*. 2019 Jun;19(2):2290-2293. doi: 10.4314/ahs.v19i2.55. PubMed PMID: 31656515; PubMed Central PMCID: PMC6794513.
5. **Kocayigit I, Can Y, Sahinkus S, Aydın E, Vatan MB, Kılıc H, Gunduz H**. Spontaneous rectus sheath hematoma during rivaroxaban therapy. *Indian J Pharmacol*. 2014 May-Jun;46(3):339-40. doi: 10.4103/0253-7613.132193. PubMed PMID: 24987185; PubMed Central PMCID: PMC4071715.
6. **Osinbowale O, Bartholomew JR**. Rectus sheath hematoma. *Vasc Med*. 2008 Nov;13(4):275-9. doi: 10.1177/1358863X08094767. PubMed PMID: 18940904.