Expect the unexpected: Rectus sheath hematoma comes without a notice

UMAMA GORSI, MD; VISHNU PRIYA MALLIPEDDI, MD

INTRODUCTION
Rectus sheath hematoma (RSH) is an unusual clinical entity that results from bleeding into the rectus sheath. RSH was first described nearly 2500 years ago, by Hippocrates and Galen who described it as a consequence of abdominal trauma (1). The first published case of RSH in the United States was in 1857 (2). The most common predisposing factors are anticoagulants, strenuous activities (e.g., cough, vomiting, exercise) and blunt abdominal trauma (3). There is an estimated rise in the RSH cases based on the increasing use of anticoagulants (2). Delayed recognition of RSH may result in complications like hemodynamic instability, abdominal compartment syndrome, multi-organ dysfunction and even death (4). We present a case of spontaneous rectus sheath hematoma resulting in hemodynamic instability, during anticoagulation therapy for acute pulmonary embolism, in a middle-aged female. We highlight the need for the physicians to consider RSH in the differential diagnosis list in high-risk patients on anticoagulants.

CASE SUMMARY
A 57-year-old obese woman with a history of hypertension, renal artery stenosis which was stented, presented with the complaints of worsening left-sided chest pain and shortness of breath for two days. She was taking clopidogrel as her only medication. She had a blood pressure of 108/56 mm Hg, pulse rate of 66 beats/min, respiratory rate of 22 breaths/min and oxygen saturation of 97%. Lungs were clear and auscultation of the precordium revealed no murmurs. The abdomen was soft, non-tender but an ecchymosis was noted in the left lower quadrant. The remainder of the physical examination was unremarkable. Laboratory investigations showed elevated D-dimer level. Computed tomography (CT) of the chest showed a right upper lobe and right lower lobe segmental pulmonary embolism. Immediately, IV heparin was started. But within 24 hours, the patient’s blood pressure dropped to 80 mm Hg and heart rate rose to 110 beats/min. There was a drop in hemoglobin from 11.3 to 8.2 g/dl in the preceding three hours. The patient was given intravenous fluids. IV heparin and clopidogrel were held temporarily. She had developed a 20 cm ecchymosis in the left lower abdominal wall, severe tenderness in the left lower abdominal quadrant and severe abdominal pain on straight leg raise (positive Carnett’s sign) in a supine position. CT of the abdomen confirmed the diagnosis of rectus sheath hematoma (RSH). The patient was transferred to the intensive care. Fluid resuscitation was started. IV Heparin and clopidogrel were discontinued. An inferior vena cava filter was placed and anticoagulation was resumed. Close monitoring of bleeding and coagulation markers for 3 months was planned along with a decision to remove the filter at that time.

DISCUSSION
Rectus sheath hematoma is the most common primary non-neoplastic disorder of the rectus abdominis muscle (5). It occurs due to the accumulation of blood within the rectus abdominis muscle either due to bleeding from the inferior
epigastric artery or the superior epigastric artery or their branches, or occasionally from direct tears of the rectus abdominis muscle [6]. RSH affect women twice as often as men, generally in the fifth and seventh decades of life [7] due to a smaller rectus abdominis muscle mass and an inability to tamponade the bleeding [2]. The main risk factors for RSH are anticoagulant therapy, hematological disorders, trauma, strenuous physical activity, coughing, sneezing, and pregnancy [8]. In a review of 126 cases of rectus sheath hematoma, almost 70% of the patients were on anticoagulation therapy, while 24% of them were on simultaneous anticoagulation and antiplatelet therapies [8].

Eliciting signs on physical examination helps in differentiating abdominal wall pathologies [9]. Carnett and Fothergill signs are elicited by flexing the neck with the patient supine. In Carnett’s sign, the pain and tenderness persist or increase with palpation of the abdominal mass in RSH and decrease with intra-abdominal pathology. In Fothergill sign, the hematoma remains fixed and palpable in RSH whereas, impalpable in an intra-abdominal mass [10]. The patient had positive Carnett’s and Fothergill signs.

Although ultrasonography of the abdomen is preferred in pregnant women, pediatric population and in patients with acute renal failure, its sensitivity for RSH is only 71%. CT abdomen with IV contrast is the diagnostic imaging modality of choice with 100% diagnostic success rate [11] and is considered superior to ultrasonography. A hyperdense mass posterior to the rectus abdominis muscle with ipsilateral anterolateral muscular enlargement are characteristic of acute RSH, although chronic RSH may present as an isodense or hypodense mass relative to the rectus abdominis muscle on CT scan abdomen [12].

Three tiers of RSH severity have been proposed. Type I RSH is intramuscular, does not cross the midline or dissect along the fascial planes. Type II RSH is intramuscular, may cross the midline, with blood seeping between the muscle and the transversalis fascia excluding prevesical space. Type III RSH may or may not involve muscle but blood is found between the muscle and the transversalis fascia, in the peritoneum or prevesical space of Retzius [13]. Although rectus sheath hematoma is self-limiting, it is associated with an overall mortality of approximately 4% whereas for those on anticoagulant therapy, it is 25% [7].

RSH is most commonly managed conservatively in the majority of RSH cases [14]. It consists of bed rest, analgesia, hematoma compression, ice packs application, fluid resuscitation and most importantly, discontinuation of anticoagulants. Type I and type II RSH are managed conservatively. Type III RSH is usually managed by blood transfusion and invasive treatment [13]. We encountered a Type III RSH in our patient who was hemodynamically unstable, managed conservatively by aggressive fluid resuscitation, discontinuation of IV heparin and clopidogrel and placing an IVC filter.

Reinitiating anticoagulation therapy is always a concern and the decision must be individualized.

References

Authors
Umama Gorsi, MD, Memorial Hospital of Rhode Island, Warren Alpert Medical School of Brown University; Division of Cardiology, Mayo Clinic.
Vishnu Priya Mallipeddi, MD, Division of Cardiology, Mayo Clinic

Correspondence
Umama Gorsi, MD Preventive Cardiology Fellow, Mayo Clinic 1805 Quarry Ridge Rochester, MN 55901 umamasardar@hotmail.com

References