Rectus sheath hemorrhage due to oral anticoagulant therapy


Department of Emergency Medicine, Ondokuz Mayis University, Faculty of Medicine, Samsun, Turkey

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* Correspondence to:
Ahmet Baydın
Ondokuz Mayis University
Faculty of Medicine
Department of Emergency Medicine
Kurupelit Samsun / Turkey
E-mail: abaydin@omu.edu.tr

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Rectus sheath hemorrhage (RSH) is an uncommon condition characterized by abdominal pain. It is an accumulation of blood in the sheath of the rectus abdominis muscle, secondary to rupture of an epigastric vessel or muscle tear. It is usually located infraumblically and often misdiagnosed as acute abdomen. This case report describes a patient with RSH and retroperitoneal hemorrhage due to anticoagulant therapy, who was admitted to our Emergency Department with complaints of abdominal pain, emesis, nausea, and abdominal wall ecchymosis. Computerized tomography must be chosen for diagnosis. Main therapy is conservative management.


1. Introduction
Rectus sheath hemorrhage (RSH) is a relatively uncommon condition but important disease causing abdominal pain. RSH is an accumulation of blood in the anterior rectus abdominis muscle due to disruption of epigastric vessels or rectus muscle (Henzel et al., 1966; Siu et al., 2003). RSH usually can occur at any age but the fifth and sixth decade is the most common (Casey et al., 2000). As RSH can be seen spontaneously, it can be seen due to trauma, previous abdominal operations, subcutaneous drug injections, hematological diseases, hypertension, coughing, physical exercise, pregnancy, iatrogenic causes, and anticoagulation therapy (Maharaj et al., 2002; Siu et al., 2003; Cherry and Mueller, 2006; Karabulut et al., 2006).

The number of patients with RSH associated with warfarin therapy is uncommon in the literature. RSH is a medical emergency that requires early diagnosis and treatment to prevent morbidity. Primary management of the RSH is conservatism. This case report describes a patient with RSH and retroperitoneal hemorrhage secondary to anticoagulant therapy, who was initially presented with acute abdomen.

2. Case Report
A 64-year-old female was admitted to our Emergency Department with complaints of abdominal pain, emesis, nausea, and abdominal wall ecchymosis. She had a history of atrial fibrillation diagnosed 10 years ago and she was taking 7.5 mg of anticoagulant drug (warfarin sodium) daily for 18 months for atrial fibrillation. At the same time she used to take oral antidiabetic drugs for Diabetes Mellitus for six years. Her previous INR measurements were within the therapeutic range. On presentation, her vital signs were stable. Mean arterial pressure was 120/70 mmHg, respiratory rate was 20/min, pulse rate was 80/min. On physical examination revealed painful mass in the left and right lower quadrant of the abdomen. The abdomen was tender on palpation, but there was no rebound tenderness and muscular rigidity. Other physical examination findings were normal.

The main laboratory test results were as follows: white blood cell count: 13,3 thousand/uL (reference range, 4.3-10.3), haemoglobin level: 7,8 g/dL (reference range, 13.6-17.2), platelet count: 189 thousand/uL (reference range, 165-352), creatinine: 1.69 mg/dL (reference range,
0.4-1.4), glucose: 179 mg/dL (reference range, 70-110), CPK: 258 U/L (reference range, 35-195), prothrombin time: 52.5 seconds (control, 10-14), and international normalized ratio: 5.21 (0.85-1.15 INR) while the other biochemical tests were within normal limits. There was atrial fibrillation on her electrocardiogram. Ultrasound (US) examination of the abdomen revealed a heterogeneous hematoma extending from both rectus abdominis muscle under umbilicus to 15 cm inferior and free fluid around liver and spleen. Computerized Tomography was performed with a suspicion of intraabdominal bleeding. Abdominal computerized tomography (CT) revealed a hematoma (rectus sheath hematoma) on the abdominal wall (Fig. 1a). A minimal free fluid around liver and in retroperitoneal space was observed (Fig.1b).

![Fig. 1a, 1b.](image)

Fig. 1a, 1b: Hematoma 4.5 cm in diameter expanding from the level of umbilicus to symphisis pubis in both rectus abdominis muscles.

After clinical and radiological evaluation, RSH was diagnosed and medical treatment (fresh frozen plasma and vitamin K) was administered in the emergency department. During this period 4 units of fresh frozen plasma and 3 units of erythrocyte suspension was administered. The INR came down to 2.1 after 9 hours. Complete blood cell count examination on the 12th hour of admission revealed an increase in the hemoglobin and hematocrit levels (9.4 g/dl and 28.3% respectively). Then the patient was transferred to the emergency observation unit. She was successfully treated. The patient discharged after 3 day.

3. Discussion

Nowadays, anticoagulant agents such as warfarin, unfractionated heparin, and low-molecular-weight heparin are used extensively to prevent deep vein thrombosis, pulmonary embolism, acute ischemic stroke, in the setting of acute myocardial infarction, valvular heart diseases, and atrial fibrillation. Bleeding is the most common complication of anticoagulant agents (Landeafeld and Beyth, 1993). In the literature, there are many complications due to anticoagulant therapy such as subcapsular renal hematoma, retroperitoneal and intraperitoneal hemorrhage, hemotherax, cutaneous hemorrhage, intramural bowel hematoma, lingual hematoma, spinal epidural hematoma, gastrointestinal hemorrhage, epistaxis, hematuria. (Lewin and Patterson, 1980; Balci et al., 2001; Mrug et al., 2002; Polat et al., 2003; Kirazli et al., 2004; Dolin et al., 2006; Ozpolat et al., 2007). In addition, RSH and intraperitoneal hemorrhage secondary to anticoagulant therapy is a rare complication. It has been described in patients treated with warfarin, intravenous unfractionated heparin, and subcutaneous low-molecular-weight heparin (Berna et al., 2000; Cherry and Mueller, 2006; Osinbowlwale et al., 2008).

The presenting symptoms and signs of RSH are sudden abdominal pain, abdominal wall mass, and rarely abdominal wall ecchymosis, nausea or vomiting (Berna et al., 2000; Cherry and Mueller, 2006). Thus the patients with RSH may present to emergency department with complaint in relation with acute abdomen. In our case, the patient presented to our emergency department with abdominal pain, abdominal wall ecchymosis, nausea and vomiting. With these findings our initial diagnosis was intraabdominal hemorrhage. Because of the history of coumadin therapy and determination of abdominal mass in the physical examination, an USG was performed and RSH was diagnosed.

The frequency of bleeding resulting from anticoagulant therapy increases with long term usage (Lewin and Patterson, 1980). The risk of bleeding is correlated with the clinical condition of the patient, the patient’s age, the patient’s gender, and intensity of anticoagulation (Berna et al., 2000). Bleeding episodes are reported to occur in 20% of cases (Choudari et al., 1994). The rate of major bleeding in Turkey was reported as 21.6% (Unverir et al., 2006). The authors commented the high incidence of major bleeding episodes in Turkey could be attributed to social and cultural diversity, difficulties in follow-up of bleeding profiles and genetic factors (Denizbas et al., 2006; Unverir et al., 2006). Our case was a 64 year-old woman and was under anticoagulant therapy for a long time.

RSH is a rarely seen condition in patients under anticoagulant therapy and clinical diagnosis of the disease is quite difficult. Imaging can provide the correct diagnosis and exclude an intraabdominal disorder. Ultrasonography (USG) can be used as a screening modality for initial evaluation of patients because it is simple, rapid, noninvasive, inexpensive, and widely available but with less sensitivity, ranging from 70% to 90% in published reviews (Cherry and Mueller, 2006). USG is a subjective screening method dependent to the performer’s experience and misdiagnosis of RSH is possible, so an abdominal CT scanning is more reasonable in such patients (Daves et al., 1996). Computed tomography (CT) is superior to USG in localisation, extension and evaluation of the size of the hematoma. Moreover CT imaging can give the classification of the hematoma. CT of the abdomen is useful in excluding other intra-abdominal processes and is the gold standard with virtually 100% sensitivity and specificity for RSH (Cherry and Mueller, 2006). In our patient RSH was found by USG but abdomen CT was performed for a detailed detection.

In RSH, unlike other acute intraabdominal pathologies, despite severe abdominal pain, general condition of the patient doesn’t fail (Linhares et al., 1999).
Conservative treatment is the mainstay of management in hemodynamically stable patients with non-expanding hematoma (Siu et al., 2003; Karabulut et al., 2006). Surgery is indicated only in progressive and large painful hematomas or when the diagnosis is in doubt. In our patient with RSH and intraabdominal hemorrhage, warfarin therapy was ceased and conservative therapy with fresh frozen plasma and blood transfusion was performed.

In conclusion, acute abdominal pain associated with RSH in a patient receiving anticoagulant therapy should raise suspicion for intraabdominal bleeding. Early diagnosis is crucial, because most patients are treated nonoperatively with good outcome. Our case illustrates that prompt recognition of this condition by emergency physicians can be achieved with USG.

REFERENCES