A 54-year-old woman was transferred to our hospital with disseminated intravascular coagulation, and was treated with heparin. On hospitalization day 13, she developed lower abdominal pain and mass followed by circulatory shock. She became oliguric and laboratory tests showed serum creatinine of 3.5 mg/dL and hemoglobin of 7.4 g/dL. Computed tomography showed hematoma in the left rectus sheath, compressing the urinary bladder exteriorly, which resulted in worsening of bilateral hydronephrosis. Conservative treatment resulted in resolution of the rectus sheath hematoma and improvement of renal function. Rectus sheath hematoma can be treated conservatively without surgical intervention even in complicated cases.

Key words: rectus sheath hematoma, shock, acute renal failure, obstructive uropathy, anticoagulant therapy

Before admission, her serum creatinine (SCr) level fluctuated around 1.5 mg/dL due to lupus nephritis and lupus cytisits, and hemoglobin (Hb) of around 10 g/dL due to renal anemia. The disease activity of SLE remained well controlled, because anti dsDNA and complement levels remained within the normal range with oral prednisolone (5 mg per day). Her medical history included three episodes of sepsis due to urinary tract infection. On admission, septic shock was treated with antibiotics, fluids and vasopressors. DIC was treated with fresh frozen plasma and heparin. An echocardiogram showed diffuse severe hypokinesis with an ejection fraction of 38% and a left ventricular apical thrombus, which was caused by antiphospholipid syndrome (APS) and cardiac disturbance due to sepsis. Heparin was administered at a dose of 10,000 units per day on the third day of admission. On hospitalization day 10, she was discharged from the ICU following improvement of the general condition. At that time, blood pressure hovered around 120/80 mmHg. Laboratory findings were as follows: leukocyte count 5,190/μL, Hb 9.2 g/dL, platelet count 61,000/μL, acti-
Figure 1. Computed tomography (CT) scan shows blood in the left rectus sheath (A). Note that the rectus sheath hematoma compressed the bladder (arrow) and showed a fluid-fluid level (arrowhead) within the mass in the lower abdomen (B), and resulted in worsening of bilateral hydronephrosis (C). At hospitalization day 65 (D), the hematoma had decreased in size following conservative treatment.

Abnormal partial-prothromboplastin time (APTT) 72.8 sec, blood urea nitrogen (BUN) 42 mg/dL, SCr 1.74 mg/dL, and C-reactive protein (CRP) 1.1 mg/dL. On hospitalization day 12, she complained of general fatigue and systolic blood pressure decreased to around 90 mmHg. On hospitalization day 13, she complained of lower abdominal pain and noticed a mass in the same area. The lower abdominal region was tender but abdominal guarding was absent. Systolic blood pressure diminished to around 80 mmHg, and urinary output decreased to 50 mL per 8 hours. Laboratory findings showed Hb 7.4 g/dL, BUN 59 mg/dL, and SCr 3.51 mg/dL. Ultrasonography (US) revealed a hypoechoic mass in the lower abdomen and bilateral hydronephrosis. Therefore, we suspected postrenal ARF due to obstruction of the urinary catheter. We attempted urethral restenting to release the obstructed urine flow, however no urine flow was noted. Since Hb and blood pressure continued to decrease rapidly, we suspected hemorrhagic shock and arranged for an urgent abdominal CT. The plain CT showed blood in the left rectus sheath, with thickening of the abdominal wall, and a fluid-fluid level within a mass in the mid-abdomen. The hematoma measured 12.8 × 8.0 cm transversely (Fig. 1A), and compressed the urinary bladder (Fig. 1B), with bilateral hydronephrosis (Fig. 1C). Based on these features, the diagnosis was established as rectus sheath hematoma that induced ARF due to hemorrhagic shock and obstructive uropathy. The first treatment step was discontinuation of heparin, followed by transfusion of packed red blood cells and fresh frozen plasma and treatment with dopamine as a vasopressor to correct blood pressure, which had decreased to around 80 mmHg (Fig. 2). The above measures increased blood pressure to 120 mmHg and Hb to 10 g/dL. Furthermore, the treatment also gradually increased urine volume and reduced SCr to baseline levels. Repeated CT scan, performed on hospitalization day 65, showed a significant decrease in the size of the hematoma (Fig. 1D). Though the anticoagulant therapy was restarted with warfarin on hospitalization day 45, rectus sheath hematoma did not show any sign of relapse. On hospitalization day 76, the left ventricular apical thrombus disappeared completely. Rectus sheath hematoma and ventricular apical thrombus were treated conservatively without surgical intervention.

Discussion

We reported a case of hemorrhagic shock and obstructive uropathy due to a large rectus sheath hematoma in a patient on anticoagulant therapy. Hemorrhage into the rectus muscle sheath is a relatively uncommon complication of anticoagulant therapy. In this patient, an extremely large rectus sheath hematoma occurred in the lower abdomen. Because the posterior sheath is deficient below the arcuate line, hematomas in the upper abdomen tend to be small in size while those in the lower abdomen tend to be of large size (8). Furthermore, we considered that the large rectus sheath hematoma was enlarged for bleeding tendency caused by anti-coagulant
therapy and thrombocytopenia associated with DIC. Though the patient had mild hydronephrosis and renal dysfunction in association with lupus cystitis and nephritis on admission, the large rectus sheath hematoma worsened both the hydronephrosis and renal function. ARF, which is a sudden reduction in glomerular filtration rate (GFR), can be classified into prerenal, intrarenal and postrenal causes. In the present patient, prerenal ARF resulted from volume depletion and hypotension due to a large rectus sheath hematoma. Further, large hematoma in the lower abdomen induced postrenal ARF caused by bilateral hydronephrosis to compress the bladder. Thus, we considered that the rectus sheath hematoma induced ARF due to hemorrhagic shock and obstructive uropathy. Historically, the diagnosis of rectus sheath hematoma is often missed. Titone et al (11) correctly diagnosed 20 of 50 patients. The differential diagnosis includes incarcerated hernia, ovarian cyst, ruptured abdominal aneurysm, intraperitoneal tumor, and appendicitis. Recent advances in imaging techniques have markedly improved the rate of correct diagnosis of this condition. Several studies have reported the utility of US and CT for the diagnosis of hematoma (2). Hirai et al (12) reported that the combination of US and CT was more effective than US only, and the rate of the correct diagnosis was 93%, compared with 60% by US alone. In the present case, as she showed oliguria with a hypoechoic mass in the lower abdomen by US, we mistook the rectus sheath hematoma for bladder dilation induced by obstruction of urinary catheter. We could attain a correct diagnosis with CT.

With regard to the treatment, previous studies described various surgical procedures because of the difficulty of correct diagnosis preoperatively (13). Jones and Merendino (14) reported that surgical treatment is required to establish the diagnosis, to control the hemorrhage, and to evacuate the hematoma. In contrast, several recent studies have reported a satisfactory outcome of conservative treatments (15). In general, the prognosis is favorable and relapse is rare (16). Berna et al (17) recommend conservative treatment, such as withdrawal of anticoagulants, reversal of anticoagulation with vitamin K and transfusion of fresh frozen plasma and blood as needed, even for large hematomas, and indicate that surgical intervention should be avoided. Furthermore, they reported that thromboembolic or hemorrhage complications were not observed by the reintroduction of anticoagulant therapy. In the present case, we were able to establish the correct diagnosis, avoid surgical treatment and improve renal function by conservative treatment. Furthermore, though the anticoagulant therapy was restarted with warfarin for APS and left ventricular apical thrombus, rectus sheath hematoma showed no relapse.

In conclusion, we reported a case of hemorrhagic shock and obstructive uropathy due to large rectus sheath hematoma in a patient on anticoagulant therapy. We were able to establish a correct diagnosis, improve the hematoma-related obstructive uropathy and treat the patient conservatively even in such a complicated case. Physicians should be aware of the possibility of rectus sheath hematoma in patients on anticoagulant therapy.

References


© 2009 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imindex.html