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A SELF-TEST ON A CLINICAL CASE

A 57-year-old woman with abdominal pain

A 57-year-old woman presented to the emergency department with left lower quadrant pain, which had started 1 week earlier and was constant, dull, aching, and nonradiating. There were no aggravating or alleviating factors. The pain was associated with low-grade fever and nausea. She reported no vomiting, no change in bowel habits, and no hematemesis, hematochezia, or melena. She did not have urinary urgency, frequency, or dysuria. She had no cardiac, respiratory, or neurologic symptoms.

Her medical history included hypothyroidism, type 2 diabetes mellitus, diverticulosis, and chronic obstructive pulmonary disease. Her medications included metformin, insulin, levothyroxine, and inhaled tiotropium. She had no allergies. She had never undergone surgery, including cesarean delivery. She was postmenopausal. She had two children, both of whom had been born vaginally at full term. She denied using alcohol, tobacco, and illicit drugs. Her family history was noncontributory.

On examination, she was not in acute distress. Her temperature was 36.7°C (98.1°F), blood pressure 130/90 mm Hg, heart rate 86 beats per minute and regular, respiratory rate 16 breaths per minute, and oxygen saturation 98% on ambient air. Examination of her head and neck was unremarkable. Cardiopulmonary examination was normal. Abdominal examination revealed normal bowel sounds, mild tenderness in the left lower quadrant with localized guarding, and rebound tenderness. Neurologic examination was unremarkable.

Initial laboratory data showed mild leukocytosis. Computed tomography with contrast of the abdomen and pelvis suggested acute diverticulitis.

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ATRIAL FIBRILLATION, AND THEN A TURN FOR THE WORSE

The patient was admitted with an initial diagnosis of acute diverticulitis. She was started on antibiotics, hydration, and pain medications, and her abdominal pain gradually improved.

On the third hospital day, she suddenly experienced shortness of breath and palpitations. At the time of admission her electrocardiogram had been normal, but it now showed atrial fibrillation with a rapid ventricular response. She also developed elevated troponin levels, which were thought to represent type 2 non-ST-elevation myocardial infarction.

She was started on aspirin, clopidogrel, and anticoagulation with heparin bridged with warfarin for the new-onset atrial fibrillation. Her heart rate was controlled with metoprolol, and her shortness of breath improved. An echocardiogram was normal.

On the seventh hospital day, she developed severe right-sided lower abdominal pain and bruising. Her blood pressure was 90/60 mm Hg, heart rate 110 beats per minute and irregularly irregular, respiratory rate 22 breaths per minute, and oxygen saturation 97% on room air. Her abdomen was diffusely tender with a palpable mass in the right lower quadrant and hypoactive bowel sounds. Ecchymosis was noted (**FIGURE 1**).

On day 7 she developed acute decompensation—what was the cause?

DIFFERENTIAL DIAGNOSIS

1 What is the likely cause of her decompensation?

- ☐ Acute mesenteric ischemia
- ☐ Perforation of the gastrointestinal tract
- ☐ Rectus sheath hematoma
- ☐ Abdominal compartment syndrome due to acute pancreatitis

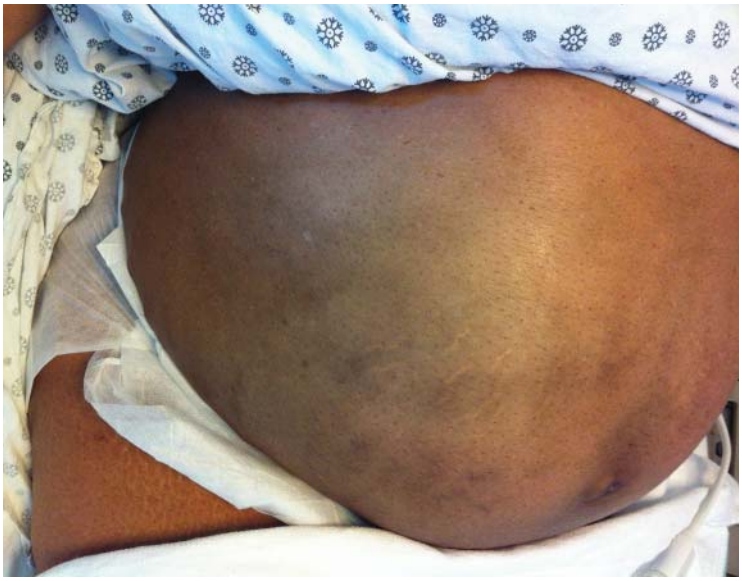


FIGURE 1. Ecchymosis of the abdominal wall, predominantly of the right flank (Grey Turner sign).

Acute mesenteric ischemia

Signs and symptoms of acute mesenteric ischemia can be vague

Signs and symptoms of acute mesenteric ischemia can be vague. Moreover, when it leads to bowel necrosis the mortality rate is high, ranging from 30% to 65%.[1] Hence, one should suspect it and try to diagnose it early.

Most patients with this condition have comorbidities; risk factors include atherosclerotic disease, cardiac conditions (congestive heart failure, recent myocardial infarction, and atrial fibrillation), systemic illness, and inherited or acquired hypercoagulable states.[2]

The four major causes are:

- Acute thromboembolic occlusion of the superior mesenteric artery (the most common site of occlusion because of the acute angle of origin from the aorta)
- Acute thrombosis of the mesenteric vein
- Acute thrombosis of the mesenteric artery
- Nonocclusive disease affecting the mesenteric vessels.[2]

Nonocclusive disease is seen in conditions in which the mesenteric vessels are already compromised due to background stenosis owing to atherosclerosis. Also, conditions such as septic and cardiogenic shock can compromise these arteries, leading to ischemia, which, if it persists, can lead to bowel infarction. Ischemic colitis falls under this category. It commonly involves the descending and sigmoid colon.[3]

The initial symptom of ischemia may be abdominal pain that is brought on by eating large meals ("postprandial intestinal angina."[2] When the ischemia worsens to infarction, patients may have a diffusely tender abdomen and constant pain that does not vary with palpation. Surprisingly, patients do not exhibit peritoneal signs early on. This gives rise to the description of "pain out of proportion to the physical findings" traditionally associated with acute mesenteric ischemia.[2]

Diagnosis. Supportive laboratory data include marked leukocytosis, elevated hematocrit due to hemoconcentration, metabolic acidosis, and elevated lactate.[4] Newer markers such as serum alpha-glutathione S-transferase (alpha-GST) and intestinal fatty acid-binding protein (I-FABP) may be used to support the diagnosis.

Elevated alpha-GST has 72% sensitivity and 77% specificity in the diagnosis of acute mesenteric ischemia.[5] The caveat is that it cannot reliably differentiate ischemia from infarction. Its sensitivity can be improved to 97% to 100% by using the white blood cell count and lactate levels in combination.[5]

An I-FABP level higher than 100 ng/mL has 100% sensitivity for diagnosing mesenteric infarction but only 25% sensitivity for bowel strangulation.[6]

Early use of abdominal computed tomography with contrast can aid in recognizing this diagnosis.[7] Thus, it should be ordered in suspected cases, even in patients who have elevated creatinine levels (which would normally preclude the use of contrast), since early diagnosis followed by endovascular therapy is associated with survival benefit, and the risk of contrast-induced nephropathy appears to be small.[8] Computed tomography helps to determine the state of mesenteric vessels and bowel perfusion before ischemia progresses to infarction. It also helps to rule out other common diagnoses. Findings that suggest acute mesenteric ischemia include segmental bowel wall thickening, intestinal pneumatosis with gas in the portal vein, bowel dilation, mesenteric stranding, portomesenteric thrombosis, and solid-organ infarction.[9]

Treatment. If superior mesenteric artery occlusion is diagnosed on computed tomography, the next step is to determine if there

is peritonitis.[10] In patients who have evidence of peritonitis, exploratory laparotomy is performed. For emboli in such patients, open embolectomy followed by on-table angiography is carried out in combination with damage-control surgery. For patients with peritonitis and acute thrombosis, stenting along with damage-control surgery is preferred.[10]

On the other hand, if there is no peritonitis, the thrombosis may be amenable to endovascular intervention. For patients with acute embolic occlusion with no contraindications to thrombolysis, aspiration embolectomy in combination with local catheter-directed thrombolysis with recombinant tissue plasminogen activator can be performed. This can be combined with endovascular mechanical embolectomy for more complete management.[10] Patients with contraindications to thrombolysis can be treated either with aspiration and mechanical embolectomy or with open embolectomy with angiography.[10]

During laparotomy, the surgeon carefully inspects the bowel for signs of necrosis. Signs that bowel is still viable include pink color, bleeding from cut surfaces, good peristalsis, and visible pulsations in the arterial arcade of the mesentery.

Acute mesenteric artery thrombosis arising from chronic atherosclerotic disease can be treated with stenting of the stenotic lesion.[10] Patients with this condition would also benefit from aggressive management of atherosclerotic disease with statins along with antiplatelet agents.[10]

Mesenteric vein thrombosis requires prompt institution of anticoagulation. However, in advanced cases leading to bowel infarction, exploratory laparotomy with resection of the necrotic bowel may be required. Anticoagulation should be continued for at least 6 months, and further therapy should be determined by the underlying precipitating condition.[10]

Critically ill patients who develop mesenteric ischemia secondary to persistent hypotension usually respond to adequate volume resuscitation, cessation of vasopressors, and overall improvement in their hemodynamic status. These patients must be closely monitored for development of gangrene of the bowel because they may be intubated and not able to complain. Any sudden deterioration in their condition should prompt physicians to consider bowel necrosis developing in these patients. Elevation of lactate levels out of proportion to the degree of hypotension may be corroborative evidence.[4]

Our patient had risk factors for acute mesenteric ischemia that included atrial fibrillation and recent non-ST-elevation myocardial infarction. She could have had arterial emboli due to atrial fibrillation, in situ superior mesenteric arterial thrombosis, or splanchnic arterial vasoconstriction due to the myocardial infarction associated with transient hypotension.

Arguing against this diagnosis, although she had a grossly distended abdomen, abdominal bruising usually is not seen. Also, a palpable mass in the right lower quadrant is uncommon except when acute mesenteric ischemia occurs due to segmental intestinal strangulation, as with strangulated hernia or volvulus. She also had therapeutic international normalized ratio (INR) levels constantly while on anticoagulation. Nevertheless, acute mesenteric ischemia should be strongly considered in the initial differential diagnosis of this patient's acute decompensation.

Perforation of the gastrointestinal tract

Diverticulitis is the acute inflammation of one or more diverticula, which are small pouches created by herniation of the mucosa into the wall of the colon. The condition is caused by microscopic or macroscopic perforation of the diverticula. Microscopic perforation is usually self-limited (uncomplicated diverticulitis) and responds to conservative treatment, whereas macroscopic perforation can be associated with fecal or purulent peritonitis, abscess, enteric fistula, bowel obstruction, and stricture (complicated diverticulitis), in which case surgery may be necessary.

Microscopic perforation is usually self-limited, whereas macroscopic perforation may need surgery

Patients with peritonitis due to free perforation present with generalized tenderness with rebound tenderness and guarding on abdominal examination. The abdomen may be distended and tympanic to percussion, with diminished or absent bowel sounds. Patients may have hemodynamic compromise.

Plain upright abdominal radiographs may show free air under the diaphragm. Computed

tomography may show oral contrast outside the lumen and detect even small amounts of free intraperitoneal air (more clearly seen on a lung window setting).

Our patient initially presented with acute diverticulitis. She later developed diffuse abdominal tenderness with hypoactive bowel sounds. Bowel perforation is certainly a possibility at this stage, though it is usually not associated with abdominal bruising. She would need additional imaging to rule out this complication.

Other differential diagnoses to be considered in this patient with right lower-quadrant pain include acute appendicitis, incarcerated inguinal hernia, volvulus (particularly cecal volvulus), small-bowel obstruction, pyelonephritis, and gynecologic causes such as adnexal torsion, ruptured ovarian cyst, and tubo-ovarian abscess. Computed tomography helps to differentiate most of these causes.

Rectus sheath hematoma

Rectus sheath hematoma is relatively uncommon and often not considered in the initial differential diagnosis of an acute abdomen. This gives it the rightful term "a great masquerader." It usually results from bleeding into the rectus sheath from damage to the superior (more common) or inferior epigastric arteries and occasionally from a direct tear of the rectus abdominis muscle. Predisposing factors include anticoagulant therapy (most common), advanced age, hypertension, previous abdominal surgery, trauma, paroxysmal coughing, medication injections, pregnancy, blood dyscrasias, severe vomiting, violent physical activity, and leukemia.[11]

Rectus sheath hematoma is relatively uncommon and is not often considered in the initial differential diagnosis of an acute abdomen

Clinical manifestations include acute abdominal pain, often associated with fever, nausea, and vomiting. Physical examination may reveal signs of hypovolemic shock, a palpable nonpulsatile abdominal mass, and signs of local peritoneal irritation. The Carnett sign[11] (tenderness within the abdominal wall that persists and does not improve with raising the head) and the Fothergill sign[11] (a tender abdominal mass that does not cross the midline and remains palpable with tensing of the rectus sheath) may be elicited.

Computed tomography is more sensitive than abdominal ultrasonography in differentiating rectus sheath hematoma from an intra-abdominal pathology.[11] In addition, computed tomography also helps to determine if the bleeding is active or not, which has therapeutic implications.

In our patient, rectus sheath hematoma is a possibility because of her ongoing anticoagulation, findings of localized abdominal bruising, and palpable right lower-quadrant mass, and it is high on the list of differential diagnoses. Rectus sheath hematoma should be considered in the differential diagnosis of lower abdominal pain particularly in elderly women who are on anticoagulation and in whom the onset of pain coincides with a paroxysm of cough.[12] Women are twice as likely as men to develop rectus sheath hematoma, owing to their different muscle mass.[13] In addition, anterior abdominal wall muscles are stretched during pregnancy.[13]

Abdominal compartment syndrome

Abdominal compartment syndrome has been classically associated with surgical patients. However, it is being increasingly recognized in critically ill medical patients, in whom detecting and treating it early may result in significant reduction in rates of morbidity and death.[14]

Abdominal compartment syndrome is of three types: primary, secondary, and recurrent. Primary abdominal compartment syndrome refers to the classic surgical patients with evidence of direct injury to the abdominal or pelvic organs through major trauma or extensive abdominal surgeries. Secondary abdominal compartment syndrome refers to its development in critically ill intensive care patients in whom the pathology does not directly involve the abdominal or pelvic organs.

Various medical conditions can culminate in abdominal compartment syndrome and result in multiorgan failure. Recurrent abdominal compartment syndrome refers to its development after management of either primary or secondary intra-abdominal hypertension or abdominal compartment syndrome.[15] Clinicians thus must be aware of secondary and recurrent abdominal compartment syndrome occurring in critically ill patients.

The normal intra-abdominal pressure is around 5 to 7 mm Hg, even in most critically

ill patients. Persistent elevation, ie, higher than 12 mm Hg, is referred to as intra-abdominal hypertension.[16–18] Intra-abdominal hypertension is subdivided into four grades:

- Grade I: 12–15 mm Hg
- Grade II: 16–20 mm Hg
- Grade III: 21–25 mm Hg
- Grade IV: > 25 mm Hg.

The World Society of the Abdominal Compartment Syndrome (WSACS) defines abdominal compartment syndrome as pressure higher than 20 mm Hg along with organ damage.[18] It may or may not be associated with an abdominal perfusion pressure less than 60 mm Hg.[18]

Risk factors associated with abdominal compartment syndrome include conditions causing decreased gut motility (gastroparesis, ileus, and colonic pseudo-obstruction), intra-abdominal or retroperitoneal masses or abscesses, ascites, hemoperitoneum, acute pancreatitis, third-spacing due to massive fluid resuscitation with transfusions, peritoneal dialysis, and shock.[18,19]

Physical examination has a sensitivity of only 40% to 60% in detecting intra-abdominal hypertension.[20] The gold standard method of measuring the intra-abdominal pressure is the modified Kron technique,[18] using a Foley catheter in the bladder connected to a pressure transducer. With the patient in the supine position, the transducer is zeroed at the mid-axillary line at the level of the iliac crest, and 25 mL of normal saline is instilled into the bladder and maintained for 30 to 60 seconds to let the detrusor muscle relax.[15] Pressure tracings are then recorded at the end of expiration. Factors that are known to affect the transbladder pressure include patient position, respiratory movement, and body mass index, and should be taken into account when reading the pressure recordings.[15,21] Other techniques that can be used include intragastric, intra-inferior vena cava, and intrarectal approaches.[15]

The WSACS recommends that any patient admitted to a critical care unit or in whom new organ failure develops should be screened for risk factors for intra-abdominal hypertension and abdominal compartment syndrome. If a patient has at least two of the risk factors suggested by WSACS, a baseline intra-abdominal pressure measurement should be obtained. Patients at risk for intra-abdominal hypertension should have the intra-abdominal pressure measured every 4 to 6 hours. However, in the face of hemodynamic instability and worsening multiorgan failure, the pressure may need to be measured hourly.[18]

Clinicians managing patients in the intensive care unit should think of intra-abdominal pressure alongside blood pressure, urine output, and mental status when evaluating hemodynamic status. Clinical manifestations of abdominal compartment syndrome reflect the underlying organ dysfunction and include hypotension, refractory shock, decreased urine output, intracranial hypertension, progressive hypoxemia and hypercarbia, elevated pulmonary peak pressures, and worsening of metabolic acidosis.[22]

Treatment. The standard treatment for primary abdominal compartment syndrome is surgical decompression. According to WSACS guidelines, insertion of a percutaneous drainage catheter should be advocated in patients with gross ascites and in whom decompressive surgery is not feasible. A damage-control resuscitation strategy used for patients undergoing damage-control laparotomy has been found to increase the 30-day survival rate.[23] A damage-control resuscitation strategy consists of increasing the use of plasma and platelet transfusions over packed red cell transfusions, limiting the use of crystalloid solutions in early fluid resuscitation, and allowing for permissive hypotension.

The gold-standard method of measuring intra-abdominal pressure is through the bladder

Secondary abdominal compartment syndrome is treated conservatively in most cases, since patients with this condition are very poor surgical candidates owing to their comorbidities.[18] However, in patients with progressive organ dysfunction in whom medical management has failed, surgical decompression should be considered.[18] Medical management of secondary abdominal compartment syndrome depends on the underlying etiology. Strategies include nasogastric or colonic decompression, use of prokinetic agents, paracentesis in cases with gross ascites, and maintaining a cumulative negative fluid balance. The WSACS does not recommend routine use of diuretics, albumin infusion, or renal replacement strategies. Pain should be adequately controlled to

improve abdominal wall compliance.[18,24] Neuromuscular blockade agents may be used to aid this process. Neostigmine may be used to treat colonic pseudo-obstruction when other conservative methods fail. Use of enteral nutrition should be minimized.[18]

Our patient might have abdominal compartment syndrome, but a definitive diagnosis cannot be made at this point without measuring the intra-abdominal pressure.

WHICH IMAGING TEST WOULD BE BEST?

2 Which imaging test would be best for establishing the diagnosis in this patient?

- ☐ Plain abdominal radiography
- ☐ Abdominal ultrasonography
- ☐ Computed tomography of the abdomen and pelvis with contrast
- ☐ Magnetic resonance imaging of the abdomen and pelvis

Plain abdominal radiography

Plain abdominal radiography can help to determine if there is free gas under the diaphragm (due to bowel perforation), obstructed bowel, sentinel loop, volvulus, or fecoliths causing the abdominal pain. It cannot diagnose rectus sheath hematoma or acute mesenteric ischemia.

Ultrasonography is widely available, safe, effective, and tolerable

Abdominal ultrasonography

Abdominal ultrasonography can be used as the first diagnostic test, as it is widely available, safe, effective, and tolerable. It does not expose the patient to radiation or intravenous contrast agents. It helps to diagnose rectus sheath hematoma and helps to follow its maturation and resolution once a diagnosis is made. It can provide a rapid assessment of the size, location, extent, and physical characteristics of the mass.

Rectus sheath hematoma appears spindle-shaped on sagittal sections and ovoid on coronal sections. It often appears sonolucent in the early stages and sonodense in the late stage, but the appearance may be heterogeneous depending on the combined presence of clot and fresh blood. These findings are sufficient to make the diagnosis.

Abdominal ultrasonography has 85% to 96% sensitivity in diagnosing rectus sheath hematoma.[25] It can help diagnose other causes of the abdominal pain, such as renal stones and cholecystitis. It is the preferred imaging test in pediatric patients, pregnant patients, and those with renal insufficiency.

Abdominal computed tomography

Abdominal computed tomography has a sensitivity and specificity of 100% for diagnosing acute rectus sheath hematoma with a duration of less than 5 days.[25] It not only helps to determine the precise location and extent, but also helps to determine if there is active extravasation. Even in patients with renal insufficiency, noncontrast computed tomography will help to confirm the diagnosis, although it may not show extravasation or it may miss certain abdominal pathologies because of the lack of contrast.

Acute rectus sheath hematoma appears as a hyperdense mass posterior to the rectus abdominis muscle with ipsilateral anterolateral muscular enlargement. Chronic rectus sheath hematoma appears isodense or hypodense relative to the surrounding muscle. Above the arcuate line, rectus sheath hematoma has a spindle shape; below the arcuate line, it is typically spherical.

In 1996, Berná et al[26] classified rectus sheath hematoma into three grades based on findings of computed tomography:

- Grade I is intramuscular and unilateral
- Grade II may involve bilateral rectus muscles without extension into the prevesicular space
- Grade III extends into the peritoneum and prevesicular space.

Magnetic resonance imaging

Magnetic resonance imaging is useful to differentiate chronic rectus sheath hematoma (greater than 5-day duration) from an anterior abdominal wall mass. Chronic rectus sheath hematoma will have high signal intensity on both T1- and T2-weighted images up to 10 months after the onset of the hematoma.[27]

Back to our patient

Since our patient's symptoms are acute and of less than 5 days' duration, computed tomography of the abdomen and pelvis would be the best diagnostic test, with therapeutic implications if there is ongoing extravasation.

Computed tomography of the abdomen with contrast showed a new hematoma, measuring 25 by 14 by 13.5 cm, in the right rectus sheath (FIGURE 2), with no other findings. The hematoma was grade I, since it was intramuscular and unilateral without extension elsewhere.

Laboratory workup showed that the patient's hematocrit was falling, from 34% to 24%, and her INR was elevated at 2.5. She was resuscitated with fluids, blood transfusion, and fresh-frozen plasma. Anticoagulation was withheld. In spite of resuscitation, her hematocrit kept falling, though she remained hemodynamically stable.

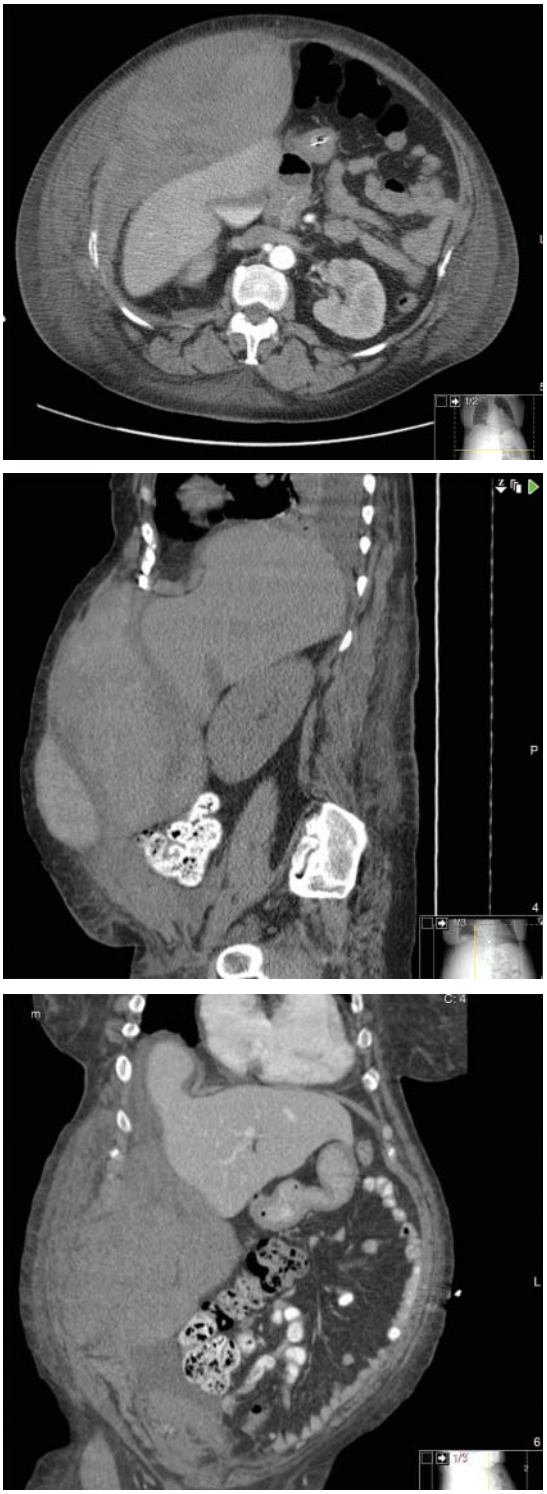
THE WAY FORWARD

3 At this point, what would be the best approach to management in this patient?

- ☐ Serial clinical examinations and frequent monitoring of the complete blood cell count
- ☐ Urgent surgical consult for exploratory laparotomy with evaluation of the hematoma and ligation of the bleeding vessel
- ☐ Repeat computed tomographic angiography to identify a possible bleeding vessel; consideration of radiographically guided arterial embolization
- ☐ Measuring the intra-abdominal pressure using the intrabladder pressure for abdominal compartment syndrome and consideration of surgical drainage

The key clinical concern in a patient with a rectus sheath hematoma who is hemodynamically stable is whether the hematoma is expanding. This patient responded to initial resuscitation, but her falling hematocrit was evidence of ongoing bleeding leading to an expanding rectus sheath hematoma. Thus, serial clinical examinations and frequent monitoring of the complete blood cell count would not be enough, as it could miss fatal ongoing bleeding.

Radiographically guided embolization with Gelfoam, thrombin, or coils should be attempted first, as this is less invasive than exploratory laparotomy.[28] It can achieve hemostasis, decrease the size of the hematoma, limit the need for blood products, and prevent



In most cases, rectus sheath hematoma resolves within 1 to 3 months

FIGURE 2. On computed tomography of the abdomen and pelvis with contrast, the transverse section (top), sagittal section (middle), and coronal section (bottom) show a right rectus sheath hematoma measuring 25 by 14 by 13.5 cm. The inferior epigastric artery is a possible source of the hematoma.

rupture into the abdomen. If this is unsuccessful, the next step is ligation of the bleeding vessel.[29]

Surgical treatment includes evacuation of the hematoma, repair of the rectus sheath, ligation of bleeding vessels, and abdominal wall closure. Surgical evacuation or guided drainage of a rectus sheath hematoma on its own is not normally indicated and may indeed cause persistent bleeding by diminishing a potential tamponade effect. However, it may become necessary if the hematoma is very large or infected, if it causes marked respiratory impairment, or if abdominal compartment syndrome is suspected.

Abdominal compartment syndrome is very rare but is associated with a 50% mortality rate.[30] It should be suspected in patients with oliguria, low cardiac output, changes in minute ventilation, and altered splanchnic blood flow. The diagnosis is confirmed with indwelling catheter manometry of the bladder to measure intra-abdominal pressure. Intra-abominal pressure above 25 mm Hg should be treated with decompressive laparotomy.[30] However, the clinical suspicion of abdominal compartment syndrome was low in this patient.

The best choice at this point would be urgent computed tomographic angiography to identify a bleeding vessel, along with consideration of radiographically guided arterial embolization.

TREATMENT IS USUALLY CONSERVATIVE

Treatment of rectus sheath hematoma is conservative in most hemodynamically stable patients, with embolization or surgical intervention reserved for unstable patients or those in whom the hematoma is expanding.

Knowledge of the grading system of Berná et al[26] helps to assess the patient's risk and to anticipate potential complications. Grade I hematomas are mild and do not necessitate admission. Patients with grade II hematoma can be admitted to the floor for 24 to 48 hours for observation. Grade III usually occurs in patients receiving anticoagulant therapy and frequently requires blood products. These patients have a prolonged hospital stay and more complications, including hypovolemic shock, myonecrosis, acute coronary syndrome, arrhythmias, acute renal failure, small-bowel infarction, and abdominal compartment syndrome—all of which increases the risk of morbidity and death. Thus, patients who are on anticoagulation who develop grade III rectus sheath hematoma should be admitted to the hospital, preferably to the intensive care unit, to ensure that the hematoma is not expanding and to plan reinstitution of anticoagulation as appropriate.

In most cases, rectus sheath hematomas resolve within 1 to 3 months. Resolution of large hematomas may be hastened with the use of pulsed ultrasound.[31] However, this treatment should be used only after the acute phase is over, when there is evidence of an organized thrombus and coagulation measures have returned to the target range. This helps to reduce the risk of bleeding and to prevent symptoms from worsening.[31]

OUR PATIENT'S COURSE

Our patient underwent urgent computed tomographic angiography, which showed a modest increase in the size of the rectus sheath hematoma. However, no definitive blush of contrast was seen to suggest active arterial bleeding. Her hematocrit stabilized, and she remained hemodynamically stable without requiring additional intervention. Most likely her bleeding was self-contained. She had normal intra-abdominal pressure on serial monitoring. She was later transferred to acute inpatient rehabilitation in view of deconditioning and is currently doing well. The hematoma persisted, decreasing only slightly in size over the next 3 weeks.

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