

# Abdominal Pain Mimics



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## KEYWORDS

• Abdominal pain • Mimics • Diagnoses

## KEY POINTS

- Emergency department providers have become skilled at triaging patients with abdominal pain requiring surgical interventions.
- Abdominal pain mimics, medical conditions that cause the sensation of abdominal pain without abdominal abnormality, continue to puzzle the best physicians.

As any emergency physician can attest, abdominal pain makes up a significant portion of chief complaints. Eleven percent of emergency department (ED) visits are attributed to abdominal pain each year.<sup>1</sup> Fortunately, with improvements in technology and advancements in imaging, ED providers have become skilled at triaging patients with abdominal pain requiring surgical interventions. However, abdominal pain mimics, medical conditions that cause the sensation of abdominal pain without abdominal abnormality, continue to puzzle the best physicians. In this article, abdominal pain mimics, which includes diagnoses that cannot be missed, conditions that require urgent evaluation, and additional conditions to consider when broadening a differential diagnosis, are discussed (**Box 1**).

### Box 1

#### Abdominal pain mimics

##### *Metabolic*

Diabetic ketoacidosis

Alcoholic ketoacidosis

Calcium abnormalities

Thyrotoxicosis

Uremia

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Porphyria

Pheochromocytoma

Adrenal crisis

*Hematologic*

Sickle cell disease

Neutropenic enterocolitis

Spontaneous splenic rupture

*Immunologic*

Angioedema

Henoch-Schonlein purpura

Systemic lupus erythematosus

Polyarteritis nodosa

Food allergy

*Infectious*

Pneumonia

Tuberculosis

Lyme disease

Pharyngitis (Lemierre syndrome)

*Toxic*

Mushroom intoxication

Alcohol intoxication

Metal poisoning

Envenomation

Opioid withdrawal

*Cardiopulmonary*

Atypical acute coronary syndrome

Pulmonary embolism

Pneumonia

Congestive heart failure

*Functional*

Abdominal migraine

Cyclic vomiting

Irritable bowel syndrome

*Neurologic*

Herpes zoster

Abdominal epilepsy

*Environmental*

Heat stroke

*Adapted from* Fields JM, Dean A. Systemic causes of abdominal pain. *Emerg Med Clin North Am* 2011;29:196–7; with permission.

## **CANNOT MISS DIAGNOSES**

Atypical acute coronary artery syndrome, diabetic ketoacidosis, pulmonary embolism (PE), and congestive heart failure make up a group of diagnoses associated with significant morbidity and mortality if missed. These patients may present with abdominal pain as the sole complaint, and the practitioner must assess their cardiac, metabolic, and thromboembolic risk factors to ensure an accurate, timely diagnosis.

### ***Acute Coronary Syndrome***

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Coronary artery disease is the number one cause of death in the United States.<sup>2</sup> Acute coronary syndrome (ACS), a clinical diagnosis that includes ST segment elevation myocardial infarction, non-ST segment myocardial infarction, and unstable angina, accounts for 3.5 million hospital admissions worldwide each year.<sup>3</sup> The typical presentation of ACS includes chest pain, shortness of breath, diaphoresis, and often pain in the left arm or jaw. ACS with an atypical presentation is an oft-missed diagnosis in the ED with grave consequences.<sup>4</sup> Symptoms are often dependent on a patient's age and gender. Younger patients (less than age 70) with ACS tend to present with typical symptoms such as chest pain and present earlier in the progression of symptoms.<sup>5</sup> On the other hand, the older patient population, greater than 70, who tend to be female and diabetic, often present only with gastrointestinal symptoms: abdominal pain, nausea, and vomiting. The pathophysiology behind this atypical presentation stems from autonomic neuropathy that occurs with longstanding diabetes mellitus. Researchers are still unsure why women tend to experience atypical symptoms more commonly than men. To avoid diagnostic delays, an early cardiac evaluation should be considered in high-risk patients (elderly female patients with history of diabetes, hypertension, and congestive heart failure) with nonfocal abdominal pain. An electrocardiogram with cardiac enzymes should be considered in addition to electrolytes, a liver function panel, and lipase. With a normal cardiac workup, the practitioner should pursue other abdominal pain causes. Keeping atypical ACS in the differential will prevent late treatment and reduce morbidity and mortality.

### ***Diabetic Ketoacidosis***

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Hyperglycemic crises are becoming increasingly common as the incidence of diabetes mellitus continues to increase. Abdominal pain occurs in a significant proportion of patients with diabetic ketoacidosis. Patients are often unable to delineate between abdominal pain as a cause for their hyperglycemia (ie, a viral gastroenteritis with increasing insulin requirements or reduced insulin administration) or abdominal discomfort caused by worsening hyperglycemia and ketoacidosis. Umpierrez and Freire,<sup>6</sup> in a prospective 2003 study, found a direct correlation between abdominal pain and metabolic acidosis in patients with diabetic ketoacidosis. Patients with abdominal pain, nausea, and vomiting had significantly lower bicarbonate levels and a lower pH. Interestingly, there was no association between abdominal pain and severity of hyperglycemia or dehydration. Very rarely did these patients have intra-abdominal abnormality, and the researchers recommended hydration and insulin therapy to close the anion gap before performing an aggressive abdominal pain workup or exploratory laparotomy, because often symptoms resolved with resolution of the metabolic acidosis. Patients with hyperglycemic hyperosmolar nonketotic state were significantly less likely to have abdominal pain. Gastroparesis from poorly controlled blood glucose, ileus from the metabolic derangements associated with diabetic ketoacidosis, and pancreatitis may also contribute to abdominal pain in patients

with diabetic ketoacidosis. Clinicians should aim to manage electrolyte derangements and pain, in addition to performing gastric emptying studies, if applicable.

### ***Pulmonary Embolism***

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Clinicians consider PE when patients present with cardiopulmonary symptoms, such as pleuritic chest pain, shortness of breath, or even simply anxiety. However, abdominal pain can also be the presenting symptom of this diagnosis associated with tremendous morbidity and mortality. Nearly 7% of PEs have associated gastrointestinal complaints that range from vague discomfort to an acute abdomen.<sup>7</sup> Although the mechanism still remains unclear, pain may be due to hepatic congestion (due to right heart strain), distention of Glisson capsule, or even diaphragmatic irritation from pulmonary infarction.<sup>8,9</sup> It is crucial to maintain a high index of suspicion for PE in high-risk patients: those with active malignancy, known hypercoagulable states, patients on exogenous estrogens, or patients who present with concerning histories such as shortness of breath, chest pain, or lower extremity edema following injuries, recent surgeries, or prolonged sedentary periods. Clinical decision-making rules such as the Geneva, Wells, or PERC (pulmonary embolism rule-out criteria) criteria should not be used in patients with abdominal pain, for which the provider has a high index of suspicion, because this may result in significant morbidity and mortality from a missed diagnosis. Clinicians should consider a computed tomographic (CT) angiogram of the chest to rule out a PE in high-risk patients. If positive, patients should receive therapeutic dosing of anticoagulation before admission for further monitoring.

### ***Congestive Heart Failure***

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Congestive heart failure is an increasingly common end-stage cardiac disease caused by a functional or structural cardiac defect impairing the heart's ability to fill and pump the ventricles.<sup>10</sup> Patients typically present with cardiopulmonary complaints such as chest pain, palpitations, or worsening shortness of breath. Interestingly, many patients experience abdominal pain: both localized and general. Patients may present with right upper quadrant pain that mimics that of hepatitis or cholecystitis. Similar to patients with abdominal pain and PE, patients in decompensated congestive heart failure can have hepatic congestion from right heart strain causing pain over the right upper quadrant on abdominal examination. In patients with new onset congestive heart failure, increasing distention of the splanchnic circulation can lead to bowel edema and a resultant adynamic ileus. These patients typically present with abdominal distention and nonfocal abdominal pain on examination, similar to a bowel obstruction. It is important to assess for cardiac risk factors, in addition to ruling out intra-abdominal abnormality before attributing abdominal pain to decompensated heart failure. In the pediatric population, children and adolescents with a new presentation of heart failure from dilated cardiomyopathies tend to present with abdominal complaints such as pain, anorexia, nausea, and vomiting.<sup>11</sup> Practitioners should have a higher index of suspicion in this younger patient population and incorporate cardiac laboratory tests, a chest radiograph, and even a bedside echocardiogram, into their initial workup.

## **CONDITIONS REQUIRING URGENT EVALUATION**

Several conditions requiring urgent but noncritical evaluation may also present with abdominal pain. Diseases such as community-acquired pneumonia, sickle cell disease, hereditary angioedema, mushroom intoxication, black widow spider

envenomation, adrenal crisis, and hematologic malignancies require urgent evaluation by a practitioner.

### ***Community-Acquired Pneumonia***

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Community-acquired pneumonia has been associated with abdominal pain in select patient populations. Although adult patients with community-acquired pneumonia tend to present with productive cough, fever, and malaise, pediatric patients often complain solely of abdominal pain. The pathophysiology behind abdominal pain in patients with pneumonia is multifaceted. In patients with lower lobar pneumonias, diaphragmatic irritation from infiltrates can cause upper abdominal pain and occasionally mimic cholecystitis, particularly in adults.<sup>12</sup> Authors of case reports in the pediatric population of acute abdomens from lower lobar and retrocardiac pneumonias attributed the abdominal complaint to innervation by the lowest intercostal nerves of both the lower costal pleura and the anterior abdominal wall.<sup>13</sup> In addition, there are case reports of reactive mesenteric adenitis from lobar or segmental pneumonias, often mimicking acute appendicitis in the pediatric population.<sup>14</sup> It is important to consider chest radiographs in patients who present with fevers and productive cough, despite their complaint of abdominal pain, given the possibility of referred pain from an extra-abdominal cause.

### ***Sickle Cell Disease***

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Sickle cell disease, a hemoglobinopathy characterized by a genetic variation in the  $\beta$ -globin chain of hemoglobin, is a common hematologic condition. During periods of illness, dehydration, or hypoxia, sickle red blood cells tend to become inflexible, occluding small venules and leading to microinfarcts. Patients experience severe pain, often localized to the abdomen. Abdominal pain in the sickle cell population should be approached similarly to abdominal pain in other patient populations. In the sickle cell population, a complete blood count with differential and reticulocyte count should also be drawn in addition to traditional abdominal pain laboratory tests.

Sickle cell hemoglobin C and hemoglobin SC-thalassemia patients tend to be prone to splenic infarction due to high viscosity blood from normal hemoglobin counts. These patients present with left upper quadrant pain, nausea, and vomiting and may have a friction rub on cardiac examination.<sup>15</sup> Splenic sequestration occurs when high volumes of red blood cells pool in the spleen due to an unclear cause. Patients tend to be young and have splenomegaly, hypotension, anemia, and very high reticulocyte counts.<sup>16</sup> Definitive treatment is splenectomy.

Hepatic sequestration can also occur. Patients complain of right upper quadrant pain due to microinfarction of the liver. Patients tend to have a mild transaminitis that improves over 1 to 2 weeks without intervention. Patients can have mesenteric microthrombi precluding them to bowel necrosis, particularly during pain crises. These patients tend to present similarly to patients with mesenteric ischemia. Abdominal pain is nonfocal, often out of proportion to examination. In patients who appear toxic, resuscitation is key, as is early surgical intervention. In patients with less clear-cut symptoms, a thorough evaluation including complete blood counts with differentials and reticulocyte counts, in addition to abdominal laboratory tests: complete metabolic profile, liver function panel, and lipase, must be considered. Hydration is crucial, as is pain control. The practitioner should consider advanced imaging such as CT scans of the abdomen and pelvis with intravenous (IV) contrast to evaluate for evidence of bowel necrosis.

Patients with sickle cell disease are also susceptible to pneumonias from encapsulated organisms; therefore, as mentioned above, a chest radiograph, especially in

patients with upper respiratory complaints, is reasonable. Only when intra-abdominal abnormality is ruled out should the practitioner attribute a sickle cell patient's pain to a pain crisis.

### ***Hereditary Angioedema***

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Hereditary angioedema (HAE) is a condition characterized by either a defect or a deficiency in the C1 receptor resulting in excess bradykinin production. Angioedema attacks may be precipitated by stress, trauma, infection, or hormonal fluctuations. As a result, patients have vasodilation and edema, particularly affecting the skin, upper airways, and gastrointestinal system. These patients present with vague abdominal pain, nausea, and vomiting, as well as airway complaints. Because of excessive vasodilation, patients tend to feel light-headed. They will not have pruritus or hives, because HAE does not affect the histamine pathway or mast cells; these symptoms separate HAE from allergic reaction or anaphylaxis. Providers should initially ensure a patient's airway is intact without evidence of oral edema. Patients with airway concerns should be intubated early.

Abdominal pain initially begins as mild discomfort. With worsening edema, patients experience abdominal distention and even ascites. Nausea and vomiting follow thereafter, with progression of intestinal edema leading to obstruction. Patients may have dilated loops of bowel on radiography.<sup>17</sup> Eventually the nausea begins to subside, but patients experience an exponential increase in the severity of their pain. Patients may appear to have an acute abdomen and often undergo multiple unnecessary surgical procedures in their lifetime. Symptoms peak around 1 to 3 days and resolve spontaneously thereafter. In addition to monitoring electrolytes, providers should acquire imaging to rule out any surgical cause. ED care is primarily supportive with hydration, antiemetics, and pain control. Several treatment options currently exist, including C1 and kallikrein inhibitors. These treatment options are very expensive and, if used, should be administered early and with the assistance of a pharmacist. Traditionally, fresh frozen plasma (FFP) was used, because it contains C1 esterase inhibitor to replenish low supplies. FFP, similar to all blood products, carries a risk of blood-borne pathogens, and there is anecdotal evidence that angioedema can be worsened by the kinin, which is also found in FFP. Regardless, FFP should be used when C1 and kallikrein inhibitors are not available, because it is effective during HAE attacks.<sup>18</sup> There is no role for antihistamines or steroids in these patients.

### ***Amanita Intoxication***

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Several toxic ingestions may present with abdominal pain. There are at least 50 species of mushrooms that are poisonous to humans. *Amanita* mushrooms account for more than 90% of fatal mushroom poisonings in the world, but are rare in the United States<sup>19</sup> and limited to the coastal Pacific Northwest, Pennsylvania, New Jersey, and Ohio. Amatoxins are found in several mushrooms, including *Amanita*, *Lepiota*, and *Galerina* species. Amatoxins primarily include  $\alpha$ -,  $\beta$ -, and  $\gamma$ -amanitins, with the  $\alpha$ -amanitin being most dangerous. This toxin is absorbed intestinally before being excreted into bile and shunted into the enterohepatic circulation for clearance. The toxin has a lengthy enterohepatic clearance, absorption by hepatocytes, and hepatocellular necrosis that places patients at risk for fulminant hepatic failure. Of the *Amanita* species, *Amanita phalloides* is associated with the highest morbidity and mortality.

*A phalloides* poisoning occurs in 3 stages. The initial stage, or incubation stage, is an asymptomatic stage that occurs within 6 to 12 hours of mushroom consumption. The second stage, typically 6 to 16 hours after consumption, is known as the

gastrointestinal stage. In this stage, phalloidin, another enterotoxin, causes gastroenteritis-like symptoms due to alteration in the cell membrane of enterocytes.<sup>20</sup> These patients will present with nonspecific gastrointestinal symptoms such as non-focal abdominal pain, nausea, vomiting, and diarrhea. Symptoms last approximately 24 hours. The final stage, known as the cytotoxic stage, is associated with highest morbidities and mortalities. This stage is  $\alpha$ -amanitin-mediated, and patients present in hepatic failure with jaundice, coagulopathy, and encephalopathy. To worsen the clinical picture, the enterotoxin continues to cycle between the intestinal and enterohepatic circulation, perpetuating hepatocyte necrosis. Practitioners should have a high index of suspicion in patients with known mushroom consumption or new onset hepatic failure. It is important to check electrolytes, liver function tests, a coagulation profile, and ammonia in patients with suspected amanita poisoning.

Treatment includes repeated activated charcoal (as often as every 2–4 hours), because this may prevent intestinal absorption of the toxin. Because of the latency phase, patients tend to present later in their course, and most sources still advise giving activated charcoal even if a significant amount of time has passed since ingestion.<sup>21</sup> Additional treatment regimens center on agents that compete with the amatoxin for binding sites. Penicillin G competes with amatoxin for binding sites on serum proteins and prevents uptake by hepatocytes. IV dosing is 1,000,000 IU/kg for the first day, followed by 500,000 IU/kg for 2 days. Silibinin, a silymarin derivative, acts similarly to penicillin G by competing with amatoxin for binding sites on transmembrane transporters, preventing the amatoxin from penetrating hepatocytes. Silibinin may also reduce enterohepatic recirculation. It must be used within 48 hours of ingestion to be effective. IV dosing is 20 to 50 mg/kg/d for 48 to 96 hours. Studies have not shown a benefit to combining silibinin with penicillin G. *N*-acetylcysteine, known for its hepatic free radical scavenging effects, may be beneficial, despite the lack of confirmatory data. The IV dose is 150 mg/kg over 15 minutes, followed by 50 mg/kg over 4 hours, followed by 100 mg/kg over 16 hours. In patients with worsening liver failure despite the aggressive use of antidotes, liver transplantation may be the only solution.

### ***Black Widow Spider Envenomation***

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Black widow spider bites, another rare toxidrome, may present with abdominal pain. Black widow spiders (*Latrodectus mactans*) are the most toxic of the *Latrodectus* genus. The female black widow spiders are approximately 2 cm long with a red-orange hourglass on their abdomen. Bites tend to be small and often go unnoticed by patients.  $\alpha$ -Latrotoxin, the toxin released with black widow envenomations, opens cation channels in presynaptic neurons, resulting in a massive release of various neurotransmitters (primarily norepinephrine and acetylcholine), leading to neurologic and autonomic symptoms. Symptoms typically begin within 1 hour of envenomation and include pain, anxiety, agitation, and autonomic dysfunction, including hypertension, tachycardia, and diaphoresis.<sup>22</sup> Patients experience muscle cramping of large muscle groups, including extremities, back, and abdomen. Often patients can have abdominal rigidity that mimics that of an acute abdomen. Initial laboratory tests aim to rule out other causes of a patient's symptoms, and any pertinent imaging should be used in a similar fashion. Care is primarily supportive. Antivenin (*L mactans*) is immunoglobulin G derived from horses inoculated with black widow venom. Relief is typically experienced after a single (2.5 mL) vial, but no prospective trials have confirmed an outcome benefit of the administration of antivenin.<sup>23</sup> Opioids are frequently needed for pain control. Calcium gluconate and benzodiazepines were historically used but few studies support their usage. Deaths from *L mactans*

envenomations are quite rare, and patients tend to require hospital admission solely for pain control.

### ***Adrenal Crisis***

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Adrenal crisis is primarily a state of mineralocorticoid deficiency.<sup>24</sup> It is precipitated by stress or infection, particularly in patients who have chronic undiagnosed adrenal insufficiency or patients who are unable to take their stress doses of glucocorticoids during illness due to protracted nausea and vomiting. Patients on chronic oral or inhaled steroids who abruptly stop their steroid regimens may also experience a crisis.

There are case reports of patients in adrenal crisis who present with acute abdomens; the mechanism is unclear. Similarly to other abdominal pain mimics, clinicians who encounter patients with known adrenal insufficiency and abdominal pain should rule out other pathologic causes of abdominal pain. Assessing electrolytes, a liver function panel, and lipase, in addition to performing a thorough abdominal examination and acquiring imaging, as the data and examination suggest, is imperative to ensuring that an intra-abdominal cause for a patient's abdominal pain is not missed. Untreated adrenal crises end in significant morbidity and mortality. These patients are very ill and necessitate admission for further evaluation, including hypothalamic-pituitary axis testing, and treatment.

As a result, patients present most commonly with hypovolemia and hypotension. Resuscitation, in these patients, is the priority. Patients should receive 1 to 3 L of normal saline, unless they require dextrose solutions (5% dextrose) for hypoglycemia, within the first 12 to 24 hours of presentation. Steroids also play a major role in resuscitation. In patients with known adrenal insufficiency in crisis, the clinician should administer 100 mg IV hydrocortisone promptly. The patient should receive 50 mg IV hydrocortisone every 8 hours thereafter. Hydrocortisone has mineralocorticoid properties, which eliminate the need for additional mineralocorticoid administration. In patients with undiagnosed adrenal insufficiency, the provider should consider giving 4 mg IV dexamethasone instead of hydrocortisone because it does not alter serum cortisol levels. Electrolyte monitoring also is crucial in the patient presenting with adrenal crisis. These patients have hyponatremia and hyperkalemia from aldosterone deficiency. With normal saline boluses and mineralocorticoid properties of hydrocortisone, patients tend to improve their electrolyte abnormalities within a few days.

### ***Hematologic Malignancies***

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Patients with hematologic malignancies including leukemia and lymphoma can experience abdominal pain related to their underlying cancer. Neutropenic enterocolitis (NEC) and spontaneous splenic rupture are 2 conditions associated with hematologic malignancies that may present to the ED with abdominal pain. NEC, also known as typhlitis, is a condition caused by breakdown of the gut membrane due to chemotherapy. The condition was first identified in the pediatric population undergoing chemotherapy for leukemia, but has been seen in neutropenic patients of all ages, particularly those with underlying hematologic malignancies. The pathophysiology of NEC is multifactorial. Cytotoxic medications lead to compromise of the bowel wall integrity. Neutropenia (absolute neutrophil count <500 cells/ $\mu$ L) causes patients to have a poor immune response to injury and promotes the spread of microorganisms, both bacterial and fungal. As a result, they have a polymicrobial intestinal infection, typically of the cecum (thought to be due to its distensible nature and poor vascularity), that frequently leads to bowel necrosis. Patients present classically with right lower quadrant pain (although theoretically both the small and the large bowels can be affected) and a prolonged fever (tends to occur 2–3 weeks after

chemotherapy) and are often bacteremic. Providers should consider NEC in febrile patients with abdominal pain on active chemotherapy, especially patients who recently received induction chemotherapy, severely neutropenic individuals, or patients on immunosuppressants. NEC is diagnosed radiographically. A CT scan with both oral and IV contrast should be performed, as a patient's renal function allows. Practitioners should avoid the use of barium enemas and colonoscopy in this population, because patients are prone to bowel perforation and subsequent bacteremia. CT scans will show evidence of bowel wall thickening, pneumatosis, or free air.<sup>25</sup> In patients with thrombocytopenia, intramural hemorrhage is also possible. Patients should have blood and urine cultures performed and started on an antibiotic regimen that covers *Pseudomonas aeruginosa*, *Escherichia coli*, other enteric gram-negative species, including anaerobes.<sup>26</sup> In patients without evidence of perforation, peritonitis, or severe bleeding, treatment should be conservative with bowel rest, IV hydration, pain control, and nasogastric tube insertion. Although neutropenic and immunocompromised populations are considered poor surgical candidates, if a patient has free air, uncontrolled hemorrhage from underlying coagulopathy, or clinical deterioration attributed to bowel necrosis, surgical intervention is indicated.

Spontaneous splenic rupture is a rare condition associated with hematologic malignancies that presents with abdominal pain. Occasionally, splenic rupture precedes a patient's cancer diagnosis. The pathophysiology of spontaneous splenic rupture is unclear but thought to be due to 3 mechanisms. First, the spleen is infiltrated by malignant lymphoproliferative and myeloproliferative cells. Because the splenic capsule is not distensible, it becomes prone to capsular rupture and subsequent splenic hemorrhage. Second, thrombocytopenia, common with myelodysplastic, predisposes these patients to hemorrhage. Finally, patients with hematologic malignancies are prone to splenic infarctions, which alter the vasculature of the spleen, increasing the likelihood of rupture.<sup>27</sup> Patients present with severe left upper quadrant pain and are often in hemorrhagic shock. Clinicians should have a high index of suspicion for spontaneous splenic rupture in patients with known hematologic malignancies who recently received induction chemotherapy who present with left upper quadrant pain and hypotension. These patients may decompensate quickly. Bedside ultrasound can assess for hemoperitoneum, but providers may consider CT scans to assess hemodynamically stable patients. Clinicians should check a complete blood count with differential, in addition to basic abdominal laboratory tests: electrolytes, a liver function panel, and lipase. Providers should use blood products to transfuse hypotensive patients and involve surgery early, as splenectomy is frequently necessary.

## UNUSUAL CAUSES TO CONSIDER

Beyond the life-threatening and urgent causes, there are a wide variety of unusual causes of abdominal pain. These causes include acute intermittent porphyria, rheumatologic diseases, infectious causes, neurologic causes, and functional causes.

### ***Acute Intermittent Porphyria***

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Porphyrias encompass 8 metabolic disorders of heme biosynthesis. They lead to skin lesions, neurologic symptoms, and visceral symptoms. Porphyrias are separated into acute porphyrias, chronic cutaneous porphyrias, and rare recessive porphyrias. A subset of acute porphyrias, the acute intermittent porphyrias, present with intermittent attacks of severe abdominal pain.<sup>28,29</sup> Other signs and symptoms include nausea, vomiting, dark reddish urine, constipation or diarrhea, sweating, tachycardia, and

hypertension.<sup>29–31</sup> Patients often become dehydrated and develop electrolyte abnormalities, particularly hyponatremia, which is seen in 40% of cases.<sup>29</sup> Neurologic complications may also occur, including seizures, neuropathy, paresis, and alterations of consciousness ranging from apathy to agitation.<sup>29,32</sup> Testing a 24-hour urine sample for porphyrins makes the diagnosis. Attacks can be triggered by hormonal changes during menstruation, fasting, smoking, alcohol abuse, sun overexposure, infections, and certain medications.<sup>28–30,32</sup> Management includes discontinuing any contributing medications and supportive care. Electrolyte abnormalities should be corrected.<sup>28</sup> Acute intermittent porphyrias are rare autosomal-dominant disorders, although they have low penetrance, with only 10% to 20% of carriers exhibiting symptoms.<sup>30–32</sup> The chronic cutaneous porphyrias and rare recessive porphyrias typically do not cause abdominal pain.<sup>29,32</sup>

### ***Systemic Lupus Erythematosus***

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Up to 40% of patients with systemic lupus erythematosus may develop abdominal pain secondary to intestinal vasculitis, otherwise known as lupus enteritis.<sup>33,34</sup> The pain typically presents insidiously, developing pain over hours to days and without peritoneal signs, with associated nausea, vomiting, diarrhea, fever, and tachycardia.<sup>33,35</sup> Occasionally, bowel ischemia can result, leading to perforation and hemorrhage.<sup>35,36</sup> Diagnosis can be made with CT scanning, which shows bowel wall thickening, a “target sign” of increased bowel wall enhancement, intestinal segment dilation, enlarged mesenteric vessels, and increased attenuation of mesenteric fat.<sup>34</sup> Treatment consists of high-dose corticosteroids or cyclophosphamide.<sup>33–35</sup> Patients with an acute abdomen, and patients who do not respond to treatment, should have evaluation to look for other causes of abdominal pain, such as peptic ulcer disease, pancreatitis, pelvic inflammatory disease, appendicitis, and cholecystitis.<sup>33,36</sup>

### ***Henoch-Schönlein Purpura***

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Henoch-Schönlein purpura is the most common vasculitis in children and is associated with gastrointestinal complaints in 50% to 75% of patients. The most common manifestation is colicky abdominal pain, which is thought to be due to bowel ischemia, although patients may have bleeding, ulcers, intussusception, or pneumatosis intestinalis.<sup>37,38</sup> Abdominal pain may precede or follow the classic rash of palpable purpura on the abdomen, buttocks, and lower extremities.<sup>38</sup> After exclusion of significant abdominal abnormality, treatment consists of corticosteroids.<sup>37,39</sup>

### ***Polyarteritis Nodosa***

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Polyarteritis nodosa is a necrotizing vasculitis, which affects medium-sized vessels.<sup>40</sup> It commonly involves the renal arteries, resulting in hypertension, but may affect multiple organ systems.<sup>41</sup> When the gastrointestinal tract is involved, patients may exhibit abdominal pain and gastrointestinal bleeding.<sup>40</sup> Diagnosis can be difficult in the ED because patients have nonspecific symptoms and laboratory findings, usually requiring testing for specific antibodies.<sup>41</sup> Treatment with corticosteroids and cyclophosphamide is effective.<sup>40,41</sup>

### ***Lemierre Syndrome***

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Lemierre syndrome (LS) is a rare complication of tonsillopharyngitis, which was more common in the preantibiotic era.<sup>42</sup> LS is caused by *Fusobacterium necrophorum* in 70% to 80% of cases, but *Streptococcus*, *Bacteroides*, *Peptostreptococcus*, and *Eikenella* species may also lead to its development. In LS, the bacteria penetrate

the parapharyngeal space to the posterior lateral compartment of the neck, seeding the internal jugular vein. The resulting thrombophlebitis of the vein then leads to septic emboli to the lungs, liver, and spleen.<sup>42–44</sup> Abdominal pain may come from primary abdominal abnormality as well as from referred pain from pleural complications. Patients present with signs of severe sepsis, and symptoms of a throat infection may have resolved by the time of presentation, which may lead to a delay in diagnosis.<sup>42</sup> In patients in whom LS is suspected, the internal jugular vein should be examined by ultrasound for evidence of thrombophlebitis, although CT and MRI may also be used.<sup>42,44</sup> Although LS was fatal in the preantibiotic era, recovery is good with appropriate antibiotic treatment, with a 6% mortality rate.<sup>42,44,45</sup> The use of anticoagulants is controversial.<sup>44,45</sup>

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### **Tuberculosis**

Abdominal tuberculosis is a rare manifestation of extrapulmonary tuberculosis, affecting 3% to 5% of patients.<sup>46</sup> It can affect any part of the gastrointestinal system, including the bowel, peritoneum, and hepatobiliary system.<sup>47</sup> Patients have nonspecific symptoms, but most commonly present with abdominal pain, ascites, abdominal distension, fever, and weight loss.<sup>46,47</sup> Complications include bowel perforation, bowel obstruction from strictures, and abscess formation.<sup>47,48</sup> Ascitic fluid, if present, can be sent for mycobacterial cultures, although the results may not be available for 6 weeks.<sup>46</sup>

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### **Lyme Disease**

Lyme disease is caused by *Borrelia burgdorferi*, transmitted by an Ixodid tick bite. Patients present with a variety of musculoskeletal, neurologic, and cardiovascular symptoms. Neurologic symptoms typically cause pain, which can include abdominal pain. The diagnosis is supported when the history includes a tick bite and the classic rash of erythema migrans; however, 50% of patients do not recall a tick bite and cutaneous findings are absent in 20%.<sup>49</sup> Lyme disease typically resolves with antibiotic treatment using doxycycline, amoxicillin, or cefuroxime.<sup>50</sup>

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### **Varicella Zoster**

Varicella zoster, or shingles, is an acute reactivation of the latent varicella zoster virus, which resides in dorsal root ganglia, cranial nerve, and enteric ganglia cells after chickenpox.<sup>51,52</sup> Patients typically report onset of pain before onset of the pathognomonic rash. If the dermatome affected overlies the abdomen, patients may present to the ED complaining of abdominal pain.<sup>51</sup> In addition, although rare, patients may develop visceral zoster, which may mimic an acute abdomen or lead to complications such as colonic pseudo-obstruction.<sup>51–53</sup> Treatment with antivirals is effective.<sup>52,53</sup>

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### **Abdominal Epilepsy**

Abdominal epilepsy is an unusual cause of chronic, recurrent abdominal pain. Diagnosis is made by 4 criteria: paroxysmal gastrointestinal complaints that are unexplained by other testing (pain, nausea, vomiting, bloating, diarrhea), central nervous system complaints (headache, confusion, fatigue, dizziness), electroencephalogram with findings specific for seizure disorder (temporal lobe seizures, generalized spike and wave discharges, and frontal lobe seizures), and improvement with anticonvulsant medications.<sup>54–56</sup> Treatment can be initiated with carbamazepine, phenytoin, and valproic acid.<sup>56</sup>

### ***Abdominal Migraine***

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Abdominal migraines are a variant of migraine headaches, which typically appear during childhood and adolescence.<sup>57,58</sup> Diagnostic criteria include the following:

- a. At least 5 attacks meeting criteria B–D
- b. Attacks of abdominal pain lasting 1 to 72 hours
- c. Pain which is midline, periumbilical, or poorly localized, dull or sore in nature, and of moderate to severe intensity
- d. At least 2 associated symptoms of anorexia, nausea, vomiting, or pallor
- e. Symptoms are not attributable to another cause.

Patients should avoid triggers, such as alcohol, stress, and certain foods, and may be treated with abortive migraine medications.<sup>58</sup>

### **PEARLS AND PITFALLS**

- Abdominal pain mimics are medical conditions that cause the sensation of abdominal pain without abdominal abnormality.
- Clinicians should initially rule out all intra-abdominal causes of abdominal pain.
- Cannot miss diagnoses, which may present with abdominal pain, include
  - Atypical ACS
  - Diabetic ketoacidosis
  - PE
  - Congestive heart failure
- Some abdominal pain mimics require urgent intervention, including
  - Lower lobe pneumonia
  - Sickle cell disease
  - HAE
  - Amanita poisoning
  - Black widow spider envenomation
  - Adrenal crisis
  - Hematologic malignancies
- Once the life-threatening abdominal pain mimics have been ruled out, the ED practitioner may want to consider some unusual causes of abdominal pain, including porphyrias, lupus enteritis, Henoch-Schonlein purpura, polyarteritis nodosa, Lemierre syndrome, tuberculosis, Lyme disease, herpes zoster, abdominal epilepsy, and abdominal migraine.

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