Abdominal Pain Mimics

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KEYWORDS

- Abdominal pain • Mimics • Differential diagnosis

KEY POINTS

- Abdominal pain is the most common reason for emergency department visits in the United States.
- Abdominal pain mimics are conditions that present with abdominal pain but are not caused by gastrointestinal abnormalities.
- Emergency physicians should keep abdominal pain mimics on the differential to avoid missing life-threatening diagnoses.

INTRODUCTION

Abdominal complaints comprise up to 10% of emergency department visits.[1] Abdominal pain can be the presenting symptom of potentially serious diagnoses that may not be caused by gastrointestinal pathology, but rather by mimics with extra-gastrointestinal origin (**Box 1**). In this article, we discuss some of these alternative causes of abdominal pain.

Although some of these pathologies cause abdominal pain because of their location within the abdominopelvic cavity, others have varying mechanisms. Some have shared visceral innervation, whereas others present with gastrointestinal symptoms such as nausea, vomiting, and diarrhea. Still, others involve cellular pathways resulting in irregularities to the gastrointestinal (GI) tract. When assessing a patient with abdominal complaints, it is important to have a broad differential diagnosis to identify these nongastrointestinal conditions.

CAN'T-MISS DIAGNOSES: ACUTE LIFE THREATS
Acute Coronary Syndrome

Coronary artery disease is consistently the leading cause of death in the United States.[2] Acute coronary syndrome (ACS) manifests as chest pain in 40% to 75% of cases, but can have atypical presentations including nausea, vomiting, and abdominal pain, leading to misdiagnosis and undertreatment.[3,4] Symptoms may vary based on

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Box 1
Selected abdominal pain mimics by category

Cardiovascular/Pulmonary
  Acute coronary syndrome
  Aortic dissection
  Congestive heart failure
  Pneumonia
  Pulmonary embolism
  Ruptured abdominal aortic aneurysm
Environmental
  Black widow spider bite
  Envenomation
  Heat stroke
  Mushroom toxicity
Functional
  Cyclic vomiting syndrome
  Irritable bowel syndrome
Hematologic
  Neutropenic enterocolitis
  Porphyria
  Sickle cell crises
  Spontaneous splenic rupture
Immunologic/Vasculitic
  Angioedema
  Food allergies
  Henoch-Schonlein purpura
  Polyarteritis nodosa
  Systemic lupus erythematosus

Genitourinary
  Ectopic pregnancy
  Ovarian torsion
  Pyelonephritis
  Testicular torsion
  Tubo-ovarian abscess
  Uremia
Infectious
  COVID-19
  Herpes zoster
  Lemierre's syndrome
  Lyme disease
  Pneumonia
Metabolic
  Adrenal crisis
  Alcoholic ketoacidosis
  Diabetic ketoacidosis
  Hypercalcemia
  Hyperglycemic emergencies
  Pheochromocytoma
  Thyrotoxicosis
Neurologic
  Abdominal epilepsy
  Abdominal migraine
Toxic
  Heavy metal poisoning
  Substance intoxication/withdrawal

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patient demographics, and certain patients, particularly the elderly, women, and diabetics can present with atypical symptoms. These atypical symptoms are likely due to the shared visceral sensory innervation between the heart and GI tract, making the location of pain less sensitive in the diagnosis of ACS. Emergency physicians should have a low threshold to rule out ACS in high-risk patients presenting with nonspecific abdominal pain. Work-up for ACS includes an electrocardiogram and cardiac enzymes. If cardiac etiology is ruled out, further investigation into other etiologies is warranted.

Aortic Emergencies

The major life-threatening acute aortic syndromes encountered by emergency physicians are aortic dissection (AD) and ruptured abdominal aortic aneurysm (AAA). Although uncommon, these entities carry a high mortality rate due in part to their variable presentations and absence of diagnostic laboratory testing.[5] Survival depends on rapid diagnosis and treatment.[6] ADs typically present with tearing chest and back pain, but can present with isolated abdominal pain, especially in type B dissections. Ruptured or symptomatic AAAs present primarily with acute, severe abdominal pain associated with hemoperitoneum, usually causing hemodynamic instability. Risk factors for aortic pathology include male gender and age more than 63 years.[7] History of hypertension, smoking, and known connective tissue disorders are also predisposing factors. Evaluation often begins with cardiac work-up, and a chest x-ray may show a widened mediastinum in type A AD. Bedside ultrasound should be considered to evaluate for flap in AD or for aortic enlargement with or without hemoperitoneum in AAA, particularly in unstable patients. This modality is 99% sensitive and 98% specific in identifying AAA greater than 3 cm in diameter.[8] In stable patients, contrast-enhanced computed tomography angiography (CTA) is the imaging modality of choice to diagnose and guide management of ADs and aneurysms.[9]

Pulmonary Embolism

Venous thromboembolism (VTE) accounts for 100,000 to 180,000 deaths annually in the United States.[10] Mortality approaches 30% if undiagnosed compared to a 2.5% to 10% when appropriately diagnosed and treated.[11,12] Patients with pulmonary embolism (PE) can present with a variety of symptoms, including pleuritic chest pain, dyspnea on exertion, hemoptysis, or even circulatory collapse.[13] However, nearly 6.7% of patients with PE seek medical attention for vague gastrointestinal complaints.[12] The mechanism by which PE causes abdominal pain is unclear, but may be related to hepatic congestion from right heart strain and distention of the liver capsule or diaphragmatic irritation from pulmonary infarction.[14–16]

Multiple clinical decision tools can be implemented to risk-stratify patients for PE. Known malignancy, use of exogenous estrogens, prolonged immobilization, and known hypercoagulable state raise the concern for VTE. In patients with atypical presentations where there is a high concern for PE, clinical decision rules should not be applied. Notably, thrombosis is a relatively common complication of COVID-19 infection, with a disproportionate incidence of PE, believed to be caused by pulmonary microthrombi.[17,18] Instead chest CTA should be pursued to avoid missing this life-threatening diagnosis.

Ectopic Pregnancy

Ectopic pregnancy (EP) is the implantation of a fertilized egg outside the endometrium, most commonly in the fallopian tube. It occurs in 1% to 2% of all pregnancies and can cause hemorrhagic shock and maternal death.[19] The classic symptoms are abdominal/pelvic pain, vaginal bleeding, or amenorrhea, but presentation is often nonspecific.[19–21] Hypotension with vaginal bleeding in a woman of reproductive age is highly concerning for ruptured EP. Risk factors include age over 35 years, smoking, prior history of EP, tubal luminal injury from prior infections, and pregnancy with assisted reproduction.[19,22] A beta-human chorionic gonadotropin (beta-hCG) above the discriminatory zone without ultrasound evidence of intrauterine pregnancy should prompt further evaluation for EP. Management in hemodynamically stable patients consists of medical treatment with methotrexate. Surgical intervention is indicated in hemodynamically unstable patients or those with contraindications to methotrexate.

Ovarian Torsion

Ovarian torsion (OT) represents 2.7% of all gynecologic emergencies, with an incidence of 4.9 per 100,000 before the age of 20 years.[23] This diagnosis is often missed because of its nonspecific presentation of acute onset abdominal pain associated with nausea and vomiting.[24] Physicians should maintain a high index of suspicion for OT, as late or missed diagnosis can result in ovarian loss and reduced fertility. Gynecologic and surgical history is of utmost importance, as OT remains a clinical diagnosis confirmed only with laparoscopy. Physical examination is usually nonspecific but can elicit an enlarged abdominal mass. Transvaginal ultrasound with doppler is the initial test of choice to evaluate for OT, and the most consistent finding is an enlarged edematous ovary with a "string of pearls" sign.[25,26] Ultrasound may also show a lack of blood flow to the affected ovary (**Fig. 1**).[27] Other findings include a "whirlpool sign," large ovarian cysts, and pelvic free fluid.[25–27] Computed tomography (CT) and MRI can be used when abdominal pathology is still on the differential. However, laparoscopy remains the diagnostic gold standard for OT.[28] Laparoscopy for detorsion and oophoropexy is the treatment of choice to salvage the ovary in premenopausal women.[22–28]
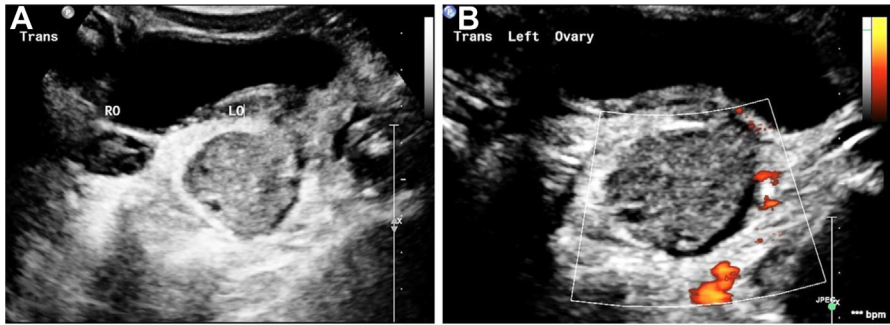
Fig. 1. Ovarian torsion in a prepubescent female. (*A*) Left ovary (LO) and right ovary (RO) on gray-scale transverse US. Note the significant difference in size, with the left ovary measuring 18.8 mL while the right measures 3.9 mL. The left ovary also has peripheral follicles and hyperechoic fat, suggesting mesenteric fat stranding. (*B*) Image of the left ovary with no intraovarian flow as seen on power Doppler. (From Ngo AV, Otjen JP, Parisi MT, et al. Pediatric ovarian torsion: a pictorial review. Pediatr Radiol 2015;45(12):1845-55; with permission.)

Hyperglycemic Emergencies

Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) are 2 conditions that are life-threatening if not recognized and managed promptly. DKA accounts for 50% of deaths in diabetics under the age of 21 years; however, overall mortality rates for DKA remain less than 1%.[29] HHS, on the other hand, has an inpatient mortality rate of up to 16%.[29] DKA and HHS present with abdominal pain, nausea, and vomiting, which often resolve with the correction of the underlying metabolic derangements. Pain does not correlate with the severity of hyperglycemia, but may be related to acidosis or precipitating factors such as toxic ingestions, pancreatitis, infection, or dehydration.[30] Management of both conditions involves aggressive hydration, insulin, electrolyte replacement, and addressing the underlying cause.[31]

Adrenal Crisis

Acute adrenal crisis has an incidence of up to 10 per 100 patient-years and a mortality rate approaching 0.5/100 patient-years. It is more likely to occur in patients with primary adrenal insufficiency than in patients with secondary adrenal insufficiency. Acute adrenal crisis is often incited by (commonly viral gastrointestinal illness) or abrupt cessation of chronic steroids. Early symptoms include fever, nausea, vomiting, and lower abdominal pain. Physical examination may reveal signs of Cushing's disease and even findings of an acute abdomen. If left untreated, patients will progress to hypotension and altered mental status, consistent with shock, and ultimately to cardiovascular collapse.[32] Laboratory findings include hyponatremia, hypoglycemia, and hyperkalemia, sometimes reflected on EKG. Adrenocorticotropic hormone and cortisol levels can confirm diagnosis, though the treating clinician should not wait for these results. Management includes directed therapy for shock, expeditious glucocorticoids, and supportive care.[33]

SHOULDN'T-MISS DIAGNOSES: REQUIRE URGENT AND TIMELY INTERVENTION
Congestive Heart Failure Exacerbation

Heart failure is a common presentation to the emergency department. Acute exacerbation of heart failure is the gradual or rapid decompensation of cardiac function,

resulting from either fluid overload or maldistribution. Common presenting symptoms include dyspnea, orthopnea, and edema. A subset of patients present with nonspecific abdominal pain, especially children and adolescents with dilated cardiomyopathy.[34] These symptoms, which include right upper quadrant (RUQ) pain, nausea, and vomiting, are thought to be due to congestive hepatopathy from fluid overload and can mimic biliary colic, cholecystitis, and hepatitis. Fluid overload causing splanchnic congestion leads to bowel wall edema, ileus, and possibly bowel ischemia. Severe heart failure exacerbation resulting in cardiogenic shock can present with abdominal pain due to nonocclusive mesenteric ischemia. A proposed mechanism is the combination of preferential shunting of blood from mesenteric circulation to cardiac and cerebral circulation and the concomitant mesenteric vasospasm.[35] If serious abdominal pathologies are ruled out and symptoms are attributed to heart failure, the typical treatment includes acute diuresis and cardiac optimization.

Pneumonia

Pneumonia is a common respiratory infection, and its diagnosis is among the mainstays of emergency medicine. Although classic symptoms include cough, chest pain, and fevers, it is important to remember that at extremes of age, the presenting features may vary. In these groups, symptoms are more likely to include headache, nausea, and abdominal pain.[36] The diagnosis of pneumonia can be made based on symptoms, imaging, and clinical examination findings, and treatment should be targeted toward the most likely organisms in the particular patient. Pneumonia is a common cause of sepsis and early and aggressive treatment should be considered to minimize the risk of deterioration.[36]

COVID-19

The SARS-CoV-2 virus has been associated with a wide array of symptoms which cause the syndrome known as COVID-19. Among the manifestations of COVID-19 are diarrhea, nausea, vomiting, and abdominal pain, which can be present in up to 50% of patients evaluated for COVID-19. Symptoms are hypothesized to be due to binding of viral particles to angiotensin-converting enzyme-2 (ACE-2) receptors within the GI tract.[37] Notably, GI symptoms may present later in disease course than respiratory symptoms. Patients with COVID-19 may exhibit elevated liver function tests (LFTs) and bilirubin, with higher elevations in more severe illness.[38] ACE-2 receptors are also expressed in pancreatic islet cells, and a pancreatitis-like presentation may thus be seen.[39] Management strategies for COVID-19 are rapidly evolving as knowledge about the syndrome improves.

Pelvic Inflammatory Disease

Pelvic inflammatory disease (PID) is the inflammation of the upper female reproductive tract induced by an infection ascending from the vagina or cervix.[40] *Chlamydia trachomatis* and *Neisseria gonorrhea* are the most commonly implicated organisms. Patients present with pelvic or abdominal pain of varying severity, vaginal discharge, dyspareunia, and postcoital bleeding.[40] In severe cases, patients can present with fever and RUQ pain due to liver capsule inflammation and adhesions, known as Fitz-Hugh-Curtis syndrome. Timely clinical diagnosis of PID is crucial, as prolonged infection and inflammation can lead to infertility and increase the risk of EP. PID is a clinical diagnosis based on pelvic examination findings of cervical motion tenderness, adnexal tenderness, and mucopurulent cervical discharge. Transvaginal ultrasound can be used to confirm the diagnosis.[41] Laparoscopic diagnosis remains the gold standard but is not routinely performed. Most patients can be

treated as outpatients, except during pregnancy, presence of tubo-ovarian abscess, or severe illness with inability to tolerate oral antibiotics. A single dose of intramuscular ceftriaxone followed by oral doxycycline for 2 weeks is the current recommended outpatient treatment, while parenteral treatment consists of cefotetan or cefoxitin with doxycycline.[40,42,43]

Sickle Cell Crises

Patients with sickle cell disease (SCD) often present to the emergency department due to complications of their disease. SCD is caused by morphologically abnormal erythrocytes. Abdominal pain is attributed to microvascular occlusion causing infarcts of the visceral and mesenteric structures within the abdomen. Most common among these is vaso-occlusive pain crisis. Abdominal pain is one of the most frequent symptoms associated with these pain crises and often mimics an acute abdomen.[44] The pain of vaso-occlusive crisis can progress to hepatic, splenic, and renal infarcts, with laboratory and imaging studies reflecting these pathologies. Once intra-abdominal pathology has been excluded, it is reasonable to treat the patient for vaso-occlusive crisis. Analgesia is paramount, and intravenous fluids should be considered only if the patient shows signs of hypovolemia.[45]

Multiple RUQ pathologies are associated with SCD. The hemolysis in SCD leads to the formation of pigmented gallstones, which in turn increase the risk of symptomatic cholelithiasis and acute cholecystitis. These sickled red blood cells can also occlude hepatic sinuses and ducts. The resultant acute intrahepatic cholestasis can lead to derangements in hepatic and renal function or coagulation abnormalities. A severe consequence of SCD is acute sickle hepatic crisis, which affects up to 10% of those admitted for abdominal pain crises and often mimics acute cholecystitis.[45] In patients with SCD and RUQ, the clinician should have a low threshold to obtain hepatic function tests, coagulation panels, and appropriate imaging, especially with associated physical examination findings.

Splenic sequestration (and eventual infarct) is commonly seen in SCD, particularly in children. Sequestration is defined as splenomegaly with a hemoglobin drop of ≥ 2 g/dL from baseline. It can rapidly progress and lead to shock. Repeated episodes may require splenectomy. Oftentimes, patients undergo splenic autoinfarction and atrophy before the age of 5 years.[46] Asplenia leaves patients with SCD at increased risk of infection by encapsulated organisms. In patients who present for evaluation with a history of SCD, other pathologies including acute chest and aplastic crisis should also be considered before assuming vaso-occlusive crisis.[47]

Thyrotoxicosis

Thyrotoxicosis is another uncommon diagnosis that can present with abdominal pain. The annual incidence of 30 cases per 100,000. If left untreated, it can lead to thyroid storm, which has a mortality rate of up to 50%.[48] Thyrotoxicosis is associated with gastroparesis, resulting in nausea, vomiting, and abdominal pain. Other symptoms can be consistent with a hypermetabolic state with increased sympathetic drive.[49] The causes of vomiting in thyrotoxicosis are unclear, but may be related to the action of thyroid hormones on GI motility.[48] A common presenting sign is tachycardia, and laboratory derangements may include abnormal LFTs in addition to thyroid studies consistent with hyperthyroidism.[50] Excess thyroid hormone is thought to have direct and secondary effects on the liver, leading to hepatocellular damage. Treatment includes beta-blockade and antithyroid agents such as propylthiouracil and methimazole, with relatively rapid return to normal function.[48]

Uremia

Uremia describes the spectrum of conditions and illnesses associated with kidney failure that are not otherwise explained by the disease state. It is the accumulation of metabolic waste products that are typically cleared by normal kidneys. The treatment of uremia is primarily via dialysis, and 5-year survival for dialysis patients is under 35%.[51] Early signs and symptoms of uremia are vague, and may include fatigue, anorexia, nausea, and anemia, among many other symptoms.[51] As the disease progresses, multiple organ systems may become involved. The initiation of dialysis is often heralded by worsening gastrointestinal symptoms, and symptoms such as dysgeusia and distaste for specific foods may suggest the need to initiate dialysis. Patients may also manifest signs and symptoms consistent with malnutrition. Although dialysis can temper metabolic derangements and improve survival, the long-term prognosis is poor without successful renal transplantation.[52]

WILL MISS IF NOT CONSIDERED: UNCOMMON PRESENTATIONS AND RARE DIAGNOSES

Acute Intermittent Porphyria

Porphyrias are a group of syndromes which arise from a defect in heme synthesis. The most common and severe of these syndromes is acute intermittent porphyria (AIP). It is an autosomal dominant condition with incomplete penetrance; as such, symptomatic disease is only seen in 1 to 2/100,000.[53] Triggers include drugs, infection, alcohol, steroids, and fasting. In AIP, neurovisceral crises due to failures in the heme synthetic pathway present as severe, colicky, epigastric pain with vomiting and constipation. Patients may progress to having psychiatric symptoms as well as peripheral and autonomic neuropathies mimicking Guillain-Barre Syndrome. The diagnosis can be confirmed with the measurement of urine porphobilinogen, aminolevulinic acid, and porphyrins from a sample during an acute attack. This sample should be shielded from light, as these molecules are photosensitive. Patients may present with hyponatremia, hypomagnesemia, slight leukocytosis, and elevated aminotransferases.[54] If AIP is suspected, consider the patient's urine color; with this condition, colorless or yellow urine will turn dark red or purple within hours if exposed to light.[55] AIP flares are managed conservatively with sugar or carbohydrate boluses and definitive treatment is via heme transfusions.

Lead Toxicity

Lead toxicity is a rare cause of abdominal pain; however, missing the diagnosis can lead to severe morbidity and mortality. Abdominal pain due to lead toxicity is usually subacute, intermittent, located in the upper quadrants, and associated with constipation.[56] Lead toxicity can also present with anemia, hearing loss, neurotoxicity, and death. Blood lead level (BLL) >5 μg/dL is considered elevated.[57] As lead exposure is commonly occupational and environmental, treatment starts with limiting exposures. BLL over 70 μg/dL requires chelating agents such as dimercaprol, DMSA, and D-penicillamine.

Henoch–Schönlein Purpura

Henoch–Schönlein Purpura (HSP) is a systemic, small vessel vasculitis caused by the deposition of IgA in vessel walls of the skin, gastrointestinal tract, joints, and kidneys. It is the most common vasculitis in children, with an incidence of 10 to 20 cases per 100,000 children/year.[58] It is a self-limited condition that lasts an average of 4 weeks.[59] The classic HSP tetrad is cutaneous palpable purpura, joint pain, gastrointestinal

complaints, and renal involvement. GI presentations range from colicky abdominal pain to intussusception, bowel ischemia, and perforation.[60] Prognosis and long-term sequelae depend on the extent of renal involvement, ranging from isolated microscopic hematuria, to nephritis complicated by nephrotic syndrome and end-stage renal disease. Treatment for HSP without renal involvement is symptomatic, including fluids and pain control. HSP nephritis is commonly treated with corticosteroids or other immunomodulators.

Hereditary Angioedema

Hereditary angioedema (HAE) is a rare autosomal dominant disorder, caused by C1-inhibitor deficiency. This deficiency contributes to a pathway that ultimately leads to vascular permeability and extravasation of plasma into interstitial tissue, causing non-pitting edema of the subcutaneous and submucosal tissue of the gastrointestinal and respiratory tracts and skin.[61,62] HAE attacks are precipitated by trauma, stress, and hormonal dysregulation. These attacks can appear as skin swelling, upper airway edema, and obstruction, and most commonly abdominal attacks, which occur in 80% of patients with HAE due to intestinal wall edema.[63] Symptoms include severe pain, nausea, vomiting, and distention. Attacks last up to 8 days and often resolve spontaneously.[64]

Work-up for HAE includes measuring plasma C4 and C-1 inhibitor levels. CT scans of the abdomen during an attack will show bowel wall edema and ascites but are nondiagnostic.[65]

In addition to symptomatic management, the first-line treatment for acute attacks includes C1-inhibitor replacement, a bradykinin B2-receptor antagonist, or a kallikrein inhibitor. Fresh frozen plasma is second-line therapy.[66]

Shingles

In herpes zoster, pain often precedes the presence of rash. Although the pain typically involves a single dermatome, it can also involve 2 or 3 consecutive dermatomes. It does not typically cross the midline.[67] The vesicles that develop will usually crust over in 7 to 10 days. Outbreaks can be managed with antivirals such as acyclovir or valacyclovir. Patients will often present with postherpetic neuralgia, which follows the same dermatomal distribution but persists for at least 90 days after acute infection. Symptoms can be managed with topical agents, oral gabapentin, or pregabalin. Vaccination should be considered if clinically appropriate.[68]

Abdominal Migraine

Abdominal migraine is a diagnosis of exclusion. It is typically seen in childhood and is characterized by episodic central abdominal pain associated symptoms usually associated with migraines: nausea, vomiting, pallor, and photophobia. However, headaches are not usually present. Adults are not likely to have a first-time diagnosis of abdominal migraine unless there is a strong family history.[69] It is often associated with other cyclic conditions such as cyclic vomiting syndrome. Acute symptoms resolve in more than 80% of patients with rest in a dark, quiet room.[70] Some limited evidence shows that pizotifen may be of benefit if medication is needed.[70]

SUMMARY

Non-gastrointestinal causes of abdominal pain should be considered before assigning a diagnosis of nonspecific abdominal pain. This article is by no means exhaustive and there are many diagnoses to be considered. Although some of these can be made in

the emergency department, some will require appropriate outpatient follow-up. A high index of suspicion is needed when evaluating patients with abdominal pain.

CLINICS CARE POINTS

- Thorough history and physical examination are mainstays of diagnosis and should help guide any interventions and testing.
- Serial abdominal examinations can help with both diagnosis and treatment.
- If a diagnosis is in doubt, strong return precautions and expeditious follow-up are essential.
- Patients, particularly those at extremes of age may present with atypical presentations of life-threatening conditions.
- Having a broad differential across multiple organ systems can help prevent anchoring bias in the evaluation of patients.

DISCLOSURES

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