



CASE REPORT | SMALL BOWEL

Chilaiditi Syndrome Masquerading as Acute Cholecystitis

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ABSTRACT

Chilaiditi sign is an incidental radiological finding where the intestine is interposed between the diaphragm and liver. Chilaiditi syndrome (CS), characterized by gastrointestinal symptoms and Chilaiditi sign on imaging, is of important clinical significance despite its rarity given associated complications including intestinal obstruction, bowel ischemia, and perforation. While most cases involve the large intestine, we report a rare case of CS with ileal involvement complicated by small bowel obstruction, managed conservatively. Failure to recognize Chilaiditi sign or CS may prompt unnecessary surgical interventions, emphasizing the need for physician awareness to ensure accurate timely diagnosis and appropriate management.

KEYWORDS: Chilaiditi syndrome; small intestine; ileum; cholecystitis; hepatodiaphragmatic interposition

INTRODUCTION

Chilaiditi sign is an incidental radiological finding where the colon, or rarely, the small bowel is interposed between the diaphragm and liver in an asymptomatic patient.¹ When present along with bowel obstruction or gastrointestinal symptoms, it is considered Chilaiditi syndrome (CS).^{1,2} Gastrointestinal symptoms may range from a mild, intermittent complaint (nausea, vomiting, abdominal pain, and constipation) to a serious condition (acute abdomen).^{3,4}

The etiology of CS is thought to be elongation, laxity, or absence of the suspensory ligaments of the transverse colon or falciform ligament, with other risk factors including cirrhosis, ascites, obesity, diaphragmatic paralysis and chronic obstructive pulmonary disease.^{1,3} It most commonly involves the colonic hepatic flexure and transverse colon.^{2,3} Indeed, ileum involvement is seldomly reported. Here, we present a rare case of CS causing small bowel obstruction (SBO) masquerading as acute cholecystitis and highlight the importance of early diagnosis and management.

CASE REPORT

A 73-year-old woman with a history of gastroesophageal reflux presented to the emergency department with a 3-day history of severe right upper quadrant (RUQ) abdominal pain with radiation to the right shoulder, right back, and flank. It worsened postprandially, and when lying flat or onto her right side, but was constant otherwise. She reported passing a small amount of flatus the day before, nausea, and difficulty taking a deep breath due to the pain but denied fever, emesis, constipation, loose stools, hematochezia, and dysuria. She had no significant history of abdominal surgeries, smoking, and alcohol abuse.

Her vital signs were overall normal. On examination, when laid flat, she developed severe pain. There was considerable tenderness in the RUQ and epigastrium, but Murphy sign was negative. There were no peritoneal signs, scleral icterus, jaundice, and costovertebral angle tenderness. Acute cholecystitis was the highest on the differential diagnoses, given these clinical features.

Laboratory results revealed normal liver tests, lipase, lactate, and no pyuria. Ultrasound was negative for cholecystitis or cholelithiasis. Computed tomography (CT) scan of the abdomen and pelvis showed interposition of a gaseous-distended distal ileum between the liver and right upper anterior abdominal wall but without wall thickening or adjacent fat stranding (Figure 1).

Chilaiditi sign complicated by CS was recognized and given concerns for early SBO, Surgery was consulted. After the initial nasogastric decompression, she passed a Gastrografin challenge and had bowel movements, halting necessity for laparoscopic exploration. She tolerated diet advancement and was discharged. At 1-month follow-up, repeat CT showed resolution of Chilaiditi sign.

DISCUSSION

Chilaiditi sign, or hepatodiaphragmatic interposition, is an uncommon finding with an incidence between 0.25% and 0.28% on imaging.¹ Its clinical significance lies in the potential for confusion with pneumoperitoneum and the development of CS.

The pathogenesis of CS is thought to be multifactorial. Specifically, there are numerous anatomical and physiological factors predisposing for the development of Chilaiditi sign, and this often involves the liver, intestines, and/or the diaphragm, as outlined in Table 1.^{1-3,5-9} Importantly, CS has been found to be associated with colonic pseudo-obstruction and various pulmonary or gastrointestinal malignancies involving the colon, rectum, or stomach.³

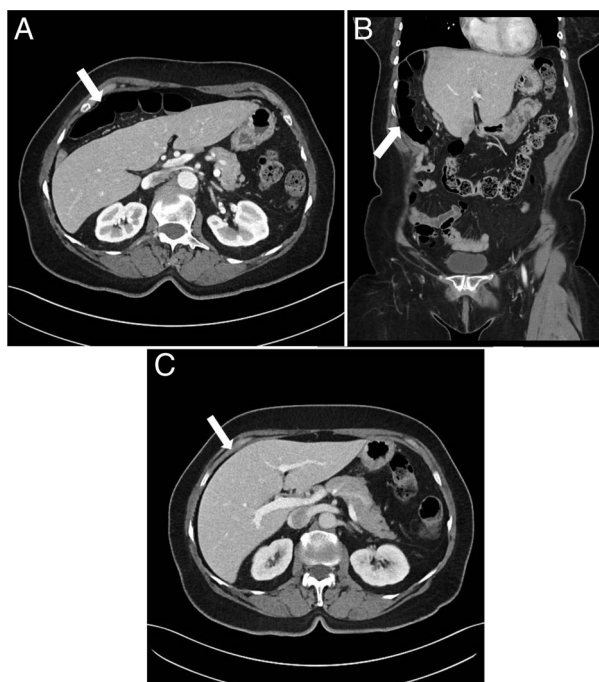


Figure 1. CT abdomen and pelvis showing interposition of gaseous-distended distal ileum (arrow) between the liver and the right upper anterior abdominal wall (Chilaiditi sign) but without wall thickening or adjacent fat stranding. (A) Axial view. (B) Coronal view. (C) Repeat CT at follow-up showing resolution of the interposition of bowel loops between the liver and right hemidiaphragm (arrow). CT, computed tomography.

Here, we reported a rare case of CS involving the ileum. A comprehensive literature search of Medline and Embase revealed only 4 similar cases.^{2,10-12} Incorporating our case, 4 of 5 patients were female with an average age of 58.2 years, with the most common presentation being SBO (4/5 cases) and RUQ abdominal pain. Three cases, excluding ours, had a known conventional predisposing factor for CS (pneumatosis cystoides intestinalis, adhesions). The remaining published case involved heterotopic endometriosis involving the right hemidiaphragm, a possible risk factor.¹⁰ In addition, our case was unique in that surgical management was not warranted likely due to the early recognition of this entity.

Table 1. Risk factors for the development of Chilaiditi sign^{1-3,5-9}

Hepatic causes	Atrophy/reduced liver volume due to: Cirrhosis Right lobe segmental agenesis Hepatectomy Ptotic liver Absence/laxity/elongation of the falciform ligament Congenital liver split Fixation of the liver due to adhesions
Diaphragmatic causes	Abnormally high diaphragm due to: Muscular degeneration Phrenic nerve injury-paralytic right hemidiaphragm
Intestinal causes	Colonic hypermobility Elongated or redundant colon with long mesentery Absence/laxity/elongation of the mesentery, suspensory ligaments Malrotation or congenital malpositioning of the bowel Dolichocolon Redundant and hypermobile transverse mesentery and transverse colon Chronic constipation (colonic elongation and redundancy) Megacolon, volvulus, and meteorism Intraperitoneal adhesion, caused by widespread tumor metastasis or previous surgery
Miscellaneous causes	Increased intra-abdominal pressure (obesity, multiple pregnancies, and ascites) Aerophagia (abnormal gas accumulation) Significant weight loss Enlargement of the lower thoracic cavity (chronic obstructive pulmonary disease) Mental retardation and schizophrenia Psychotropic medication and iatrogenic factors, such as endoscopic procedures, bariatric surgery, enteral feeding tube insertion, and colonoscopy Congenital hypothyroidism, pneumatosis cystoides intestinalis, paralytic ileus, melanosis coli, sigmoidal or rectal tumors, scleroderma

Chilaiditi sign is important to recognize given that differentials include pneumoperitoneum, diaphragmatic hernia, and subphrenic abscess.³ However, the presence of normal plicae circulares or haustral markings of the colon under the diaphragm can help rule out these more serious conditions. Furthermore, unlike in cases of free air, changing the position of a patient with Chilaiditi sign will not alter the position of the radiolucency. Pneumoperitoneum and subdiaphragmatic fluid collections may also be accompanied by pulmonary findings such as ipsilateral pleural effusion and basilar atelectasis. CT effectively distinguishes the presence of bowel interposed between the liver and diaphragm, as was observed in our patient.¹³ Specifically, it allows differentiation between subphrenic fluid, pneumoperitoneum, and intraluminal bowel gas. Identification of Chilaiditi sign is also crucial before certain procedures. Its recognition may prevent the risk for bowel perforation during percutaneous transhepatic interventions or liver biopsies, especially in cirrhotic patients, who are predisposed to development of Chilaiditi sign.⁶ In addition, colonoscopy should be performed with caution due to risk of progressive air entrapment in an acutely angulated, interposed bowel, which may lead to perforation.³ Colonoscopy, therefore, should ideally be utilized after the resolution of CS when associated colorectal malignancy is suspected. In addition, there is some evidence that colonoscopy may predispose for CS.^{6,14}

The key to management of CS is accurate timely diagnosis. No intervention is required for an asymptomatic patient with Chilaiditi sign.³ In most cases of CS, management is conservative consisting of bowel rest, bowel decompression, stool softeners and/or enemas, and aggressive rehydration.¹³ Repeat imaging following bowel decompression showing resolution of the air below the diaphragm confirms both the diagnosis and success of the treatment, and should be performed especially given that the recurrence rate is not well elucidated in the literature.³ Surgical management is reserved for patients who do not respond to conservative measures or develop serious complications including intestinal obstruction/ischemia, perforation, and cecal/colonic volvulus.^{6,15} Indeed, the strength of our case was that we expeditiously suspected early SBO in the context of CS, which may have prevented necessity for surgery.

In conclusion, awareness of this condition expedites early and accurate diagnosis and reduces unnecessary exploratory laparotomies for misdiagnosed pneumoperitoneum. While this patient presented with symptoms suggestive of cholecystitis, early recognition of SBO in the context of CS likely prevented surgical intervention and improved outcomes. In summary, while Chilaiditi sign is typically benign, expeditious recognition of CS is crucial to prevent possible complications.

DISCLOSURES

Author contributions: B. Saha: Conceptualization, data extraction, writing original draft, reviewing and editing final

manuscript. A. Verma: writing original draft, reviewing and editing final manuscript. N. Coelho-Prabhu and D. Cabrera: reviewing and editing final manuscript, supervision. ZS Kelm: data extraction, reviewing and editing final manuscript. B. Saha and A. Verma are the article guarantors.

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Written informed consent was obtained from the patient for their de-identified information to be published.

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