Abstract
Chilaiditi syndrome is a rare entity in which interposition of bowel between the liver and right hemidiaphragm manifests with abdominal pain, nausea, vomiting, anorexia, and constipation that can be easily misdiagnosed as bowel perforation or intestinal obstruction. Most patients with radiologic evidence of hepatodiaphragmatic interposition of bowel remain asymptomatic (i.e., Chilaiditi sign). We report the case of a patient with Chilaiditi syndrome who suffered from acute on chronic abdominal pain accompanied by nausea and vomiting, and underwent exploratory laparoscopy based on a working diagnosis of small bowel obstruction.

Case
50-year old Hispanic male presented to our hospital with severe 8/10 abdominal pain, localized to the right upper quadrant, and associated with nausea, vomiting and anorexia. His medical history included chronic kidney disease, rheumatoid arthritis, hypertension, and previous hospitalizations for unexplained abdominal pain. Initial evaluation included an abdominal ultrasound which revealed cholelithiasis without evidence of cholecystitis. This was followed by a CT of the abdomen without contrast that revealed dilated loops of small bowel, mostly of the jejunum and ileum, concerning for a small bowel obstruction. Subsequently, the patient underwent an exploratory laparoscopy with a preoperative diagnosis of small bowel obstruction. Laparoscopic exploration of the bowel did not reveal any obstruction or ischemia. However, the surgeons described segments of dilated small bowel and evidence of increased peristalsis.

Symptoms persisted after the exploratory laparoscopy and a new CT of the abdomen, and pelvis was obtained on post-operative day 5 that showed diffusely dilated colon without any evidence of mechanical obstruction suggesting colonic ileus or Ogilvie syndrome. In addition, a colonic loop at the hepatic flexure was interposed anterior to the liver and between the superior margin of the liver capsule and the diaphragm [figures 1-2]. This displaced colonic bowel loop is consistent with Chilaiditi sign, and explains the patient’s symptoms. Our patient was stable during ongoing observation post-operatively, but continued to have significant abdominal pain and food intolerance that recovered gradually and was able to be successfully discharged.

Discussion
Chilaiditi sign is a rare radiologic finding in which hepatodiaphragmatic interposition of the colon or small bowel occurs. This sign was first described in 1910 by Demetrius Chilaiditi, a Greek radiologist who practiced in Vienna, Austria. He described three cases of patients with bowel interposition between the liver and diaphragm. Chilaiditi sign is a radiographic term, as most patients with this anomaly will remain asymptomatic throughout their lives. Those who become symptomatic will develop Chilaiditi syndrome, which manifests with intermittent abdominal pain, distention, vomiting, anorexia, and constipation that may require surgical intervention.

The ability to recognize Chilaiditi sign is crucial, as it is commonly misinterpreted as free air under the diaphragm, pneumoperitoneum, which is an indication for immediate surgical exploration based on a working diagnosis of a perforated viscus; e.g. peptic ulcer or colonic diverticulum. The prevalence of Chilaiditi sign is around 0.025 to 0.28% of general population, more prevalent in males than females, and the incidence increases with age. The bowel segments, most commonly found interposed between the liver and diaphragm or abdominal wall are the colonic hepatic flexure and transverse colon, although interposition of the small bowel has been reported.

Recognition of this condition becomes especially important when...
performing interventional procedures such as hepatic biopsy and percutaneous gallbladder drainage.

Suspensory ligaments and fixation of the colon normally prevent interposition of the colon between the liver and diaphragm. Hence, any variations in normal anatomy can lead to the interposition of the colon. Anatomic distortions may predispose patients to the development of Chilaiditi sign, including decreased liver dimensions, elongation of the ligamentous suspension of the liver, and redundancy of the colon. Other described anomalies associated with this sign are right hepatic lobe segmental agenesis, relaxation or a genesis of the mesentery suspensory ligaments, and hypermobile transverse mesentery and transverse colon. Another predisposition to Chilaiditi sign is elongation of the lower thoracic cage in chronic obstructive pulmonary disease, leading to a broader space where colonic interposition can occur. The prevalence of Chilaiditi sign in cirrhotic patients without ascites is much higher than in the general population, possibly explained by the smaller volume of the cirrhotic liver. A retrospective study of the relationship between Chilaiditi syndrome and obesity concluded that obesity may be a contributing factor in the etiology of Chilaiditi syndrome, and the difference in fat deposition between men and women may account for the increased prevalence in men, who are more likely to have “central” or visceral organ obesity. Mental illness and schizophrenia may also increase incidence of Chilaiditi sign.

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Figure 2. Diffusely dilated colon with interposed colonic loop behind and superior to the liver, and no evidence of mechanical obstruction.
Chilaiditi sign has been associated with a variety of functional disorders, such as chronic constipation (colonic elongation and redundancy), aerophagia, cirrhosis, diaphragmatic paralysis, chronic lung disease, obesity, and multiple pregnancies.

Patients with Chilaiditi syndrome usually present with gastrointestinal symptoms followed by respiratory distress, and less frequently angina-like chest pain. Gastrointestinal symptoms include anorexia, nausea, emesis, abdominal pain, distension, potentially with serious complications including volvulus and bowel perforation. Our patient presented with these symptoms requiring hospitalization in the past for recurrent vomiting and abdominal pain episodes, which had no explanation. A working diagnosis of cyclic vomiting syndrome was being considered. Intestinal pseudo-obstruction which can overlap with Ogilvie syndrome, has also been described in a patient with Chilaiditi syndrome.

Diagnosis of Chilaiditi syndrome is based upon clinical findings and signs observed on plain radiographs and CT scans. CT scans of the abdomen enables clinicians to exclude diaphragmatic hernia and differentiate subphrenic fluid, true pneumoperitoneum, and air within the bowel lumen. This differentiation is of a critical importance because perforation can also complicate Chilaiditi syndrome when the involved bowel segment strangulates, and eventually perforates. The radiologic differential diagnosis is established by observing an elevation of the right hemidiaphragm due to caudal displacement of the liver, haustral markings between the liver and diaphragmatic surface, and the absence of image displacement with changes in the patient’s position. Pneumoperitoneum and subdiaphragmatic fluid collections have different characteristics being more mobile on lateral decubitus radiographs, and can be accompanied by pulmonary findings such as ipsilateral pleural effusion and basilar atelectasis.

In most cases of Chilaiditi syndrome, management is conservative and consists of bowel decompression, bowel rest, and aggressive fluid rehydration. Patients who fail conservative therapy should undergo an exploratory procedure. Colonoscopy should be performed with great caution considering the risk of gas being trapped in the acutely angulated and interposed bowel, potentially leading to perforation. Administration of carbon dioxide as the insufflating agent for colonoscopy is appropriate for decreasing this risk because it is nonexplosive, rapidly absorbed, and increases colon blood flow.

REFERENCES