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"Left-sided" Chilaiditi sign? A large gastric perforation with secondary pancreatitis

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Abstract. – BACKGROUND: Although the Chilaiditi sign correctly underestimates the radiological presence of air-under-the-diaphragm, in few cases it has lead to delays in the diagnosis of underlying pneumoperitoneum. In this article we report the case of a young lady presenting with acute pancreatitis and radiographic findings of "left-sided" Chilaiditi sign with underlying a large gastric perforation.

CASE REPORT: A 35 year old Caucasian female presented to the Emergency Department with a twenty-four hour history of severe epigastric pain radiating into her back. Stable observations, mildly raised white cell count, increased amylase and subdiaphragmatic radiolucency were interpreted as acute pancreatitis with Chilaiditi sign and treated accordingly. Twenty-four hours later the deterioration of the clinical conditions required a CT scan and an emergency laparotomy that lead to the diagnosis of a large gastric perforation. However, after several days she died of a disseminated intravascular coagulation in the Intensive Care Unit.

CONCLUSIONS: In our case the young age of the patient and lack of underlying comorbidities initially compensated the severity of the perforation until it became evident from the absent resolution of the pancreatitis. Bearing this in mind, radiological appearances similar to Chilaiditi sign with significant symptoms or signs should be investigated with a CT scan in order to reach promptly a correct diagnosis.

Key Words:
Chilaiditi sign, Gastric perforation, Acute pancreatitis.

Introduction

The incidental radiographic finding of subdiaphragmatic radiolucency due to bowel interposed between the liver and the right hemidiaphragm is named Chilaiditi sign, first described by Demetrius Chilaiditi in 1910 when he was working in Vienna[1,2]. The occurrence is reported as 0.025-0.28% of the general population[2] and its importance lies in the fact that it can be misdiagnosed as a pneumoperitoneum and leads to unnecessary surgical interventions. Chilaiditi syndrome is recognised as the association of the radiological sign with complications such as abdominal pain[3], volvulus[4] or respiratory distress[5,6].

Chilaiditi sign has been reportedly linked to anatomical, pathological factors and certain diseases. Anatomical factors range from malfixation or malposition of bowel, redundant or elongated bowel, laxity of hepatic suspensory ligaments[7-9]. In all these cases the underlying anatomical variant allows for augmentation of the space between the liver and the diaphragm, thus, allowing for interposition of bowel in this space. Pathological factors include conditions such as liver cirrhosis, pregnancy and chronic obstructive airways disease as these result in reduced liver volume. An increased abdominal pressure or enlargement of the lower thoracic cavity are also postulated mechanisms indicated in the predisposition to Chilaiditi sign[7-9]. More recently, Chilaiditi sign has been described in a case of acute pancreatitis probably due to the paralytic ileus often associated with this disease ("sentinel loop") and the consequent bowel dilatation and eventual interposition between the diaphragm and the liver[10]. In this article we report the case of a young lady presenting with a clinical picture of acute pancreatitis and radiographic findings similar to the Chilaiditi sign that masked a large gastric perforation.

Case Report

A 35 year old Caucasian female presented to the Emergency Department with a twenty-four hour history of severe epigastric pain radiating into her back. She had complained of feeling nauseous for one day and had two episodes of vomiting prior to attending the Emergency Department.

Since the summer of 2011 she had noted symptoms of early satiety and inability to take liquids first thing in the morning. She noted that

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shortly after ingesting solids she would begin to vomit. Occasionally she described waking in the morning with a sensation of vomit in her mouth. Over a period of six months from October 2011, she had lost four stones. On March 2012 she was admitted to the Accident and Emergency Department for multiple episodes of vomiting and received an esophagastroduodenoscopy that showed significant looping in the stomach and inability to pass the scope into the duodenum, signs suspicious for paraoesophageal hernia and gastric volvulus. She was discharged home with the request for an urgent barium meal. This was done few days later and showed the antrum located anteriorly and superiorly and the posterior surface of the stomach anteriorly, in keeping with a mesentero axial volvulus. One week later she presented to the Emergency Department with recurrent vomiting being unable to tolerate solids or liquid. She underwent an emergency laparotomy for an incarcerated diaphragmatic hernia containing the fundus of the stomach. It was noted there was a congenital opening in the diaphragm approximately 6 × 6 cm which was closed using Ethilon sutures. A Nissen fundoplication was carried out and feeding jejunostomy tube inserted. Post-procedure she progressed well with removal of the feeding tube and discharged home two weeks post-operatively. One month after the operation she was seen in an Outpatient Clinic and stated that she had nausea on eating quickly and had to avoid certain foods.

Six months after her surgery, on the present admission, she had significant amounts of abdominal pain, located in the epigastrium and radiating into her back. Her abdomen was tender in the epigastrium, otherwise soft and non-distended in the remaining quadrants. The blood tests revealed an elevated amylase 652 IU/L (normal < 101 iU/L), a mildly elevated white cell count 11.78 × 10^9/L (normal 4.00-11.00 × 10^9/L) and neutrophils count 9.54 × 10^9/L (normal 2.00-7.00 × 10^9/L). Chest X-ray showed "left-sided" Chilaiditi sign and abdominal X-ray no specific bowel pattern (Figure 1); therefore, she was diagnosed with acute pancreatitis and scored mild using the Glasgow prognostic criteria. Initial treatment consisted in supplemental oxygen administered via nasal cannula, fluid resuscitation with IV fluids, catheterisation with urine output monitoring, and patient-controlled-analgesia (PCA) using morphine. Her condition deteriorated overnight when she developed sinus tachycardia, tachypnoea and a raised temperature (39.0°C).

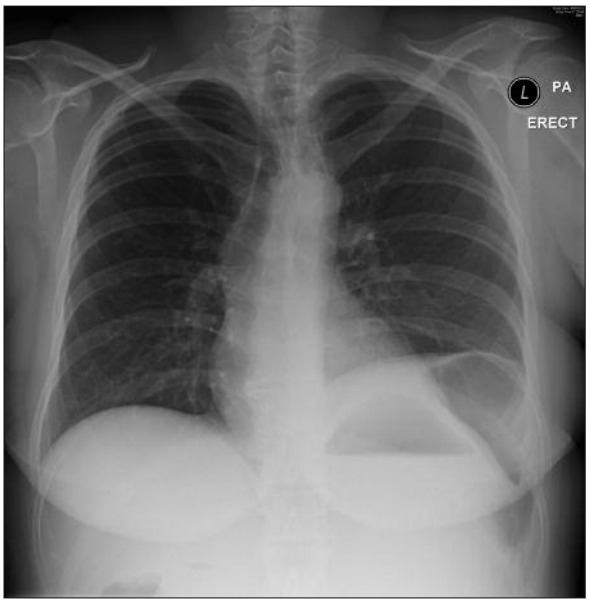

Figure 1. Chest X-ray on admission. Although diagnosis on admission was mistaken for Chilaiditi sign, a postoperative review of the images found an elevated left hemidiaphragm and free air under the left hemidiaphragm consistent with perforation of the bowel.

She also manifested severe abdominal pain and abdominal examination showed generalised guarding with a distended abdomen. An arterial blood gas revealed a pH 7.17, $PaCO_2$ 6.5 kPa, PaO_2 14.4 kPa, Na 130 mmol/L, K 4.6 mmol/L, lactate 4.7 mmol/L, BE –11.3 mmol/L, HCO_3 17.5 mmol/L. A CT abdomen and pelvis scan revealed extensive ascites and free intraperitoneal air, indicative of bowel perforation and features suggestive of pancreatitis.

She was taken to the operating room for an emergency exploratory laparotomy. Laparotomy revealed an acute gastric perforation of approximately 10 cm x 4 cm on the fundus of the stomach towards the greater curvature with significant extravasation of gastric contents. The gastric perforation appeared necrotic in origin without any other gastric pathology. An emergency sleeve gastrectomy was performed and the abdomen was washed out and closed. Post-operatively she remained on the intensive care unit (ICU) for three days where she was supported with maximum dose inotropes and intravenous antibiotics. She remained septic with temperature of 40.0°C and eventually became anuric and developed DIC disseminated intravascular coagulation (noted on post mortem examination). Three days later she died.

Discussion

Hepatodiaphragmatic interposition of the colon can be confused with pneumoperitoneum and subphrenic abscesses radiologically and lead to unnecessary surgical intervention if not recognised correctly. There are several points of distinction to note between this condition and that of a pneumoperitoneum or subphrenic abscess. Haustral markings in the subdiaphragmatic air are present and the positional orientation of the patient will not change the location of the subdiaphragmatic air in the hepatodiaphragmatic interposition of the colon[11,12].

In our patient the chest radiograph showed subdiaphragmatic radiolucency on a clinical background of stable vital parameters, raised amylase and mildly raised white cell count. This lead to the diagnosis of acute pancreatitis with "left-sided" Chilaiditi sign, as previously reported[10]. The Chilaiditi sign has been introduced to correctly interpret the clinical meaning of the radiological sign of air-under-the-diaphragm on the right side and avoid an erroneous diagnosis of perforation. However, there have been various cases where an underlying pathology was not promptly detected[1,13-15]. The presence of normal vital parameters does not necessarily exclude bowel perforation and the Chilaiditi syndrome *per se* in certain patients may mask an underlying significant pathology. Where in doubt, suspicious symptoms should require further evaluations to rule out underlying perforations.

Our patient had a previous laparotomy where a Nissen fundoplication was carried out to repair a congenital incarcerated diaphragmatic hernia, increase the lower oesophageal sphincter pressures and decrease episodes of reflux[16]. The Nissen procedure has low post-operative complication rates as well as a low morbidity and mortality[17] but there have been cases where gastric or oesophageal perforations have resulted during a Nissen fundoplication. A review by Perkidis et al[18] highlighted 25 patients (1%) with an intraoperative gastric perforation and 15 patients (0.6%) requiring conversion from a laparoscopic to an open procedure out of 2453 cases of patients undergoing a Nissen fundoplication. No study or review literature was found identifying gastric perforation as a late post-operative complication. During post-mortem examination of our patient, attempts were made to look at the previous diaphragmatic repair and Nissen fundoplication as well as any anatomical abnormality that may explain her sequence of events. However, no definitive outcome was reached.

One potential explanation for the gastric perforation and acute pancreatitis is the occurrence of closed loop obstruction on a background of the Nissen fundoplication and possible co-existence of superior mesenteric artery (SMA) syndrome. The syndrome is characterised by compression of the third, or transverse portion of the duodenum against the aorta by the SMA secondary to a narrowing of the angle between these two vessels[19]. It leads to acute or chronic intermittent partial or complete duodenal obstruction[20,21] with an approximate incidence of 0.013-0.3%[22,23]. However, at post-mortem examination, no such evidence was found supporting this diagnosis in our patient.

Conclusions

In this patient the young age and lack of underlying comorbidities initially compensated the severity of the perforation until it became evident from the absent resolution of the pancreatitis. Bearing this in mind, radiological appearances similar to Chilaiditi sign with significant symptoms or signs should be investigated with a CT scan in order to reach promptly a correct diagnosis.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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