CASE REPORT

Man with 27 year old was admitted with diffuse abdominal pain accompanied by stop in eliminating flatus and feces for three days and fever 38,3º C for 24 h. As history, had passed prior laparotomy seven years ago for acute appendicitis. He denied other symptoms, recent travel or infectious diseases. There was no recent use of medications or hospitalization. Denied alcohol or illicit drugs.

On examination, he was confused, agitated, dehydrated with clinical signs of sepsis. Was febrile (38,3º C), with tachycardia (112 beats per minute), tachypnea (20 breaths per minute) and hypotension (90x50 mmHg). The abdomen had prior infraumbilical laparotomy scar, very distended, painful diffusely, hypertimpanic and positive to sudden decompression. There was no evidence or clinical signs of liver disease or ascites. Rectal touch was normal without bleeding or mucus in the stool.

Initial investigation showed leukocytosis (18,600 leukocytes with 11% rod cells), metabolic acidosis signals, high C-reactive protein (38.6 mg/l) and abdominal radiography with air-fluid levels without pneumoperitoneum. Abdominal CT scan showed only distension and small amount of free fluid in the abdominal cavity; urinalysis and electrolytes unchanged. Differential diagnoses were acute inflammatory abdomen with diffuse peritonitis and acute obstructive abdomen.

Patient received treatment with appropriate volume expansion 20 ml/kg and antibiotic therapy with ciprofloxacin 400 mg 12/12 h and metronidazole 500 mg 8/8 h. It was referred to explorative laparotomy as urgency after 24 h after admission.

The intraoperative findings were only distension of the small bowel with the presence of thick flanges and thick purulent fluid in the abdominal cavity and pelvis. In the inventory of the cavity was not observed organized abscess and visceral perforation with no identifiable cause for the origin of pus. It was held lysis of adhesions and collection of purulent fluid to culture. The result of the culture was positive for *Neisseria meningitidis* group C, confirmed by polymerase chain reaction. The antibiogram was sensitive to ceftriaxone, meropenem and rifampicin.

Evolved on the 2nd day after surgery with worsening of confusion and positive meningeal signs besides diffuse petechiae and thrombocytopenia (88,000 platelets/mm^3). Spinal liquor resulted also be positive for *Neisseria meningitidis* group C (diplococci gram negative) with 33,000 cells/mm^3 (up to 5 cells/mm^3) 79% of neutrophils, 6 red blood cells (to 0/mm^3), total protein 172 mg/dl (up to 40 mg/dl) glucose and 1 mg/dl (40-80 mg/dl). It was referred to ICU with diagnosis of meningitis with meningococcemia; began treatment with ceftriaxone 1 g 12/12 h, resulting in improvement of neurological and abdominal symptoms after 72 h.

DISCUSSION

Neisseria meningitidis, Gram-negative diplococcus, was described in 1887 as major cause of meningitis and meningococcal bacteremia in all ages. The dissemination occurs through the nasopharynx with hematogenous spread to the meninges or other organs. It is not part of the normal gastrointestinal flora and isolated only in rectal secretions in combination with sexual transmission. Meningococcal spontaneous peritonitis have been reported in patients with preexisting ascites, but still little understood in patients without liver disease.

The first case was described in 1917 by Moeltoen[4] and the second with characteristics with appendiceal abscesses, was reported in 1938 by Turchetti[5]. In all cases, the peritonitis is associated with meningococcal disease in other distant sites.

Kelly in 2004 reported a case of peritonitis by *N. meningitidis* diagnosed after laparotomy[3] similar to acute peritonitis. The theory that can explain the pathophysiological mechanism for this condition is the spread of bacteria through the blood; however, patients with ascites and liver bacterial translocation can justify the isolation of bacteria in peritoneum[1,2,3,6].

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Letter to the Editor

WILKIE'S SYNDROME: A RARE CAUSE OF INTESTINAL OBSTRUCTION

Síndrome de Wilkie: causa rara de obstrução intestinal

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INTRODUCTION

Superior mesenteric artery (SMA) syndrome or Wilkie's syndrome is a rare but potentially life threatening gastrointestinal condition. This syndrome is a clinical phenomenon believed to be caused by compression of the third part of the duodenum between the SMA and the aorta, leading to obstruction. Patients may present symptoms of gastrointestinal obstruction, such as with recurrent episodes

vomiting, upper abdominal distension and epigastric tenderness[8]. Various etiology theories, clinical course and treatment options have hitherto been discussed[5]. An interdisciplinary teamwork provides the most beneficial diagnostic and therapeutic result in this often underestimated disease.

CASE REPORT

A 27 years old woman was referred to our hospital, with recurrent episodes of profuse vomiting and upper abdominal pain associated with loss of appetite and dyspepsia since two years. She had no other comorbidities. Had been treated at another hospital with proton pump inhibitors, analgesics and intravenous fluids. She had a history of chronic anorexia and progressive loss of weight along with recurrent episodes of vomiting and upper abdominal pain. Clinical examination revealed dehydration, asthenicity (body mass index 19,5 kg/m^2, weight: 50 kg, length:160 cm), abdominal distension, epigastric tenderness. Laboratory investigations showed a total white cell count of 9 500 mm^3 and hypokalaemia (serum potassium: 3 mEq/l). Plain radiograph of the abdomen revealed gastric dilation. Ultrasonography was unremarkable. Upper gastrointestinal endoscopy showed dilated stomach and duodenum. Contrast-enhanced *computerized tomography* scan revealed grossly distended stomach and duodenum proximal to the third part of the duodenum at the level of the origin of superior mesenteric artery with abrupt narrowing at this level, suggestive of Wilkie's syndrome. While, normally, the angle between the SMA and the aorta is 22° to 60°, in this case, the aortomesenteric angle was 13,5°(Figure 1). In this case, conservative management was inefficient, so surgical treatment aiming to bypass the obstruction by an anastomosis between the jejunum and the proximal duodenum (duodenojejunostomy) was successful.

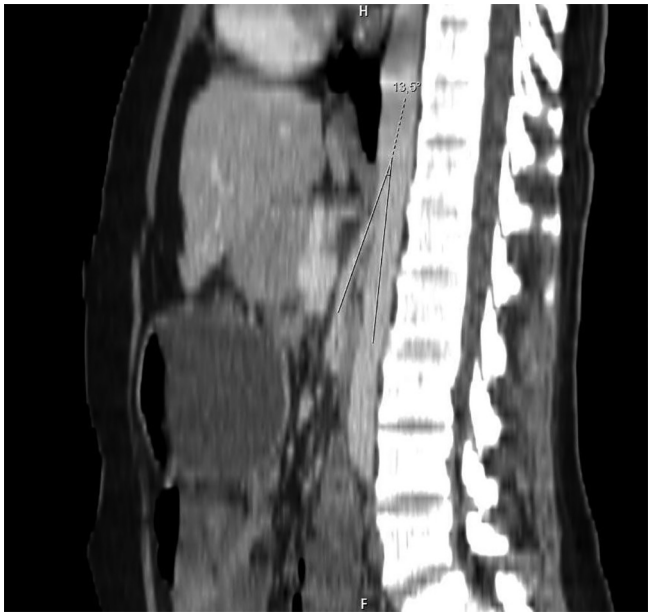

FIGURE 1 - CT of the abdomen showing reduced angle between the superior mesenteric artery and the aorta, with compression of the duodenum

DISCUSSION

Wilkie's syndrome occurs when the third portion of the duodenum is compressed between the SMA and the aorta. While, normally, the angle between the SMA and the aorta is 25° to 60°, it is narrowed in this syndrome[7]. The aortomesenteric angle may be narrowed because of congenital anomalies, significant weight loss, lumbar hyperlordosis, restorative proctocolectomy with ileal–anal anastomosis[1,2,6]. Clinical features of Wilkie's syndrome are entirely vague and non-specific. The most prominent symptoms are post-prandial abdominal pain (59%), nausea (40%), vomiting (50%), early satiety (32%), and anorexia (18%). These symptoms are aggravated by lying supine after eating and are relieved by assuming the left lateral decubitus, prone or knee-chest position[3]. These symptoms are compatible with more common conditions such as peptic ulcer disease, biliary colic, pancreatitis, and mesenteric ischemia. Physical examination generally reveals an asthenic body habitus.

The diagnosis of Wilkie's Syndrome requires a high degree of clinical suspicion confirmed by radiographic studies demonstrating compression of the third portion of the duodenum. CT of the abdomen typically shows gastric and duodenal dilation and narrowed aortomesenteric angle[9]. Wilkie's syndrome responds to conservative management in the form of adequate nutrition by enteral/parenteral feeding and proper positioning of the patient after feeds. Surgery is resorted to when conservative measures are ineffective or in patients with long history of progressive weight loss or pronounced duodenal dilatation with stasis and complications[4].

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