

FIGURE 1 - Coronal computed tomography of the neck clearly showing the fish impacted in cervical esophagus

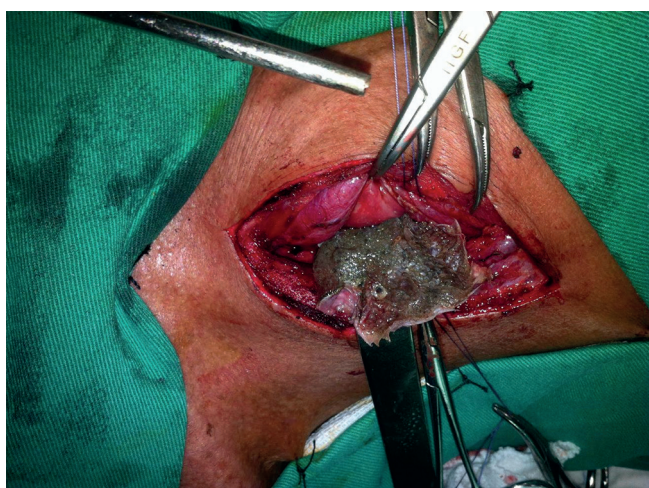


FIGURE 2 - Esophagotomy and removal of the intact fish

DISCUSSION

The greater part of foreign bodies (80%) pass through the gastrointestinal tract without difficulties, but 20% can obstruct the lumen, requiring endoscopic or surgical removal (1% of cases). As the esophagus is a narrow portion of the gastrointestinal tract, 28-68% objects are found in this region⁵. The symptoms depend on the location. Dysphagia, odynophagia and salivation suggest esophageal foreign body⁴. It can also present chest pain, cough, dyspnea, wheezing or stridor. In more severe cases, particularly in large or sharp foreign bodies, there may be intense pain, vomiting, refusal to eat, saliva ink with blood or shock¹.

A medical review of database present several accidents involving foreign bodies ingestion, including food-bolus impactions, coins, fish bones, dental prostheses, chicken bones, iron slices, lighters, little metallic foreign bodies, toothbrushes, needles, and spoons⁵, but no reports involving the ingestion of whole fish. Impaction events with fish bones includes 12.6% of

the accidents, the third highest in incidence⁵. As the majority of the bodies are radiopaque, the diagnosis can easily be done with plain radiography in posteroanterior and lateral projections. Endoscopy and contrasted study are needed in the case of radiotransparent objects. In all radiological exams it must be looked for signs of subcutaneous emphysema, which indicates drilling³. The treatment of choice is the endoscopic removal of the foreign body, which is successful with little or no complications for the patient². The surgical treatment should be performed when endoscopic management is not possible to solve the problem, or if there is impairment or progression in the gastrointestinal tract or complications such as perforation, obstruction and bleeding^{2,3}.

REFERENCES

1. Arana A, Hauser B, Hachimi-Idrissi S, Vandenplas Y. Management of ingested foreign bodies in childhood and review of the literature. *Eur J Pediatr*. 2001;160(8):468.
2. Brady PG. Esophageal foreign bodies. *Gastroenterol Clin North Am* 1991;20(4):691-701
3. Eisen GM, Baron TH, Dominitz JA, et al. Guideline for the management of ingested foreign bodies. *Gastrointest Endosc* 2002;55:802-6.
4. Hachimi-Idrissi S, Corne L, Vandenplas Y. Management of ingested foreign bodies in childhood: our experience and review of the literature. *Eur J Emerg Med* 1998;5:319-23.
5. Zhao-Shen Li, MD, Zhen-Xing Sun, MD, Duo-Wu Zou, MD, Guo-Ming Xu, MD, Ren-Pei Wu, MD, Zhuan Liao, MD. Endoscopic management of foreign bodies in the upper-GI tract: xperience with 1088 cases in China. Volume 64, No. 4: 2006 GASTROINTESTINAL ENDOSCOPY

ABCD DV/1173

ABCD Arq Bras Cir Dig Letter to the Editor

2016;29(1):67

DOI: /10.1590/0102-6720201600010019

NEISSERIA MENINGITIDIS PERITONITIS SEROTYPE C: CASE REPORT

Peritonite por Neisseria meningitidis sorotipo C: relato de caso

João Kleber de Almeida **GENTILE**¹, Maurice Youssef **FRANCISS**^{1,2}, Hamilton Ribeiro **BRASIL**^{1,2}

From the ¹Departamento de Cirurgia Geral da Irmandade da Santa Casa de Misericórdia de São Paulo, São Paulo, SP e ²Hospital Geral de Guarulhos (Departamento of General Surgery of the Irmandade da Santa Casa de Misericórdia de São Paulo, São Paulo, SP and ²Guarulhos General Hospital, Guarulhos, SP), Brazil

Financial source: none

Conflicts of interest: none

Correspondence:

João Kleber de Almeida Gentile

E-mail: joaokleberg@gmail.com

Received for publication: 04/02/2015

Accepted for publication: 15/12/2015

 This is an open-access article distributed under the terms of the Creative Commons Attribution License.

INTRODUCTION

The meningococcal disease manifestation as acute abdomen with meningococcal peritonitis is rare. Is reported primary peritonitis and bacteremia by *Neisseria meningitidis* serotype C occurring in conjunction with the obstructive acute abdomen.

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 38,3° 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 (38,3° 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/mm³). Spinal liquor resulted also be positive for *Neisseria meningitidis* group C (diplococci gram negative) with 33,000 cells/mm³ (up to 5 cells/mm³) 79% of neutrophils, 6 red blood cells (to 0/mm³), 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 meningococemia; 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 Moeltoen⁴ and the second with characteristics with appendiceal abscesses,

was reported in 1938 by Turchetti⁵. 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 laparotomy³ 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}.

REFERENCES

1. Bannatyne RM, Lakdawalla N and Ein S. Primary meningococcal peritonitis. *Can Med Assoc J.* 1977;117(5):436.
2. Bar-Meir S, Chojkier M, Groszmann RJ, Atterbury CE and Conn HO. Spontaneous meningococcal peritonitis. *The American Journal of Digestive Diseases.* 1978;23(2):119-122.
3. Kelly SJ and Robertson RW. *Neisseria meningitidis* peritonitis. *ANZ Journal of Surgery.* 2004;74:182-183. doi: 10.1046/j.1445-1433.2003.02850.x.
4. Moeltoen MH. Meningococcal peritonitis. *Zentralbl Chir.* 1917;44: 94.
5. Turchetta A. Considerazioni cliniche su un caso di peritonite meningococcica circoscritta in adulto apparentemente idiopatica. *Minerva Med.* 1938;(2):570.
6. Wendlandt D, King B, Zielbell C and Milling T. Atypical presentation of fatal meningococemia: peritonitis and paradoxical centrifugal purpura fulminans of late onset. *The American Journal of Emergency Medicine.* 2011;29(8):960.

ABCD DV/1174

ABCD Arq Bras Cir Dig

2016;29(1):68

DOI: /10.1590/0102-6720201600010020

Letter to the Editor

WILKIE'S SYNDROME: A RARE CAUSE OF INTESTINAL OBSTRUCTION

Síndrome de Wilkie: causa rara de obstrução intestinal

Ayşe KEFELİ, Adem AKTÜRK, Bora AKTAŞ, Kerim ÇALAR

From the Kecioren Training Hospital, Gastroenterology Department, Pınarbaşı St. Sanatoryum Av. No:25 Kecioren and Siirt State Hospital, Radiology Department Abdullah Özgür Yeniova Gaziosmanpaşa University, Gastroenterology Department, Ankara, Turkey

Correspondence:

Ayşe Kefeli
aysekefeli@hotmail.com

Financial source: none

Conflicts of interest: none

Received for publication: 09/12/2014

Accepted for publication: 19/11/2015

 This is an open-access article distributed under the terms of the Creative Commons Attribution License.

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