Acute Colonic Pseudo-obstruction Caused by Acute Gastroenteritis
A Case Report
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ABSTRACT

Acute colonic pseudo-obstruction is known as Ogilvie’s syndrome with marked colonic distension in the absence of any mechanical obstruction. The mechanism of this disease is still unclear. Acute colonic pseudo-obstruction may be caused by infection, traumatic disease, postoperation or neurological disease. The index patient presented with acute colonic pseudo-obstruction secondary to acute gastroenteritis. She responded to conservative treatment including diet restriction, gastrointestinal decompression, intravenous fluid and electrolyte supplement. Therefore, early diagnosis and conservative treatment are important for acute colonic pseudo-obstruction, or colectomy is inevitable.

Keywords: Acute colonic pseudo-obstruction, gastroenteritis conservative treatment, Ogilvie’s syndrome

INTRODUCTION

Colonic pseudo-obstruction (CPO) is characterized by severe dilatation of the colon without any provable mechanical obstruction in the previously healthy colon. Acute colonic pseudo-obstruction (ACPO), also known as Ogilvie’s syndrome (1), presents with abdominal pain and abnormal intestinal motility and is not uncommon in patients with critical illnesses, electrolyte imbalances, anticholinergic medication regimens and recent surgery. Conservative treatment is recommended in the first instance. Otherwise, surgery is the only treatment for the cases with colonic necrosis or perforation. So, it is important to improve outcome by early recognition.

CASE REPORT

A 63-year-old female presented with complaints of severe paroxysmal pain in the hypogastrium of four hours duration. The pain was acute in onset, colicky, and gradually involved the whole of the abdomen. The patient had diarrhoea, nausea and recurrent episodes of nonbilious vomiting since the onset of pain. She had no history of other medical problems. Her body temperature was 38.5 °C and blood pressure was 138/85 mmHg.
An abdominal examination detected abdominal distension, marked tenderness all over without rebound tenderness, and hypoactive bowel sounds. Laboratory findings: total leukocyte count: 10.11 \times 10^9/L, neutrophil count: 8.00 \times 10^9/L, serum potassium: 3.20 mmol/L, serum sodium: 125.0 mmol/L, serum chloride: 90.0 mmol/L, routine stool and faecal occult blood tests: negative, Vibrio cholerae detection: negative. The computed tomography (CT) of the whole abdomen showed severe dilatation and pneumatosis of the total colon in the absence of any obstructive lesion (Figure).

A variety of causes lead to the sympathetic and parasympathetic autonomic nervous system dysfunction which can lead to Ogilvie’s syndrome (2). Usually, this syndrome is secondary to traumatic disease, postoperation, infection, metabolic diseases, neurological diseases, use of drugs etc. The diagnosis of Ogilvie’s syndrome is based on the clinical and radiographic findings (3). The diagnostic criteria: (1) abdominal distension and pain, nausea and vomiting, status reduction, some patients may have diarrhoea (2) the main signs are abdominal distension, mild tenderness, without peritonitis diminished or absent bowel sounds (3) X-ray and CT scan demonstrate dilatation and pneumatosis of colon (4). No provable mechanical obstruction. Early recognition and treatment of this syndrome are important in order to improve the outcome. Supportive therapy should be the initial management. Improvement of clinical symptoms occurs in several days. However, if ischaemia or perforation occurs, surgery should be performed (4). In this patient, we believe the syndrome was related to acute gastroenteritis. We considered that intestinal infection and electrolyte disorder induced enteric nervous system imbalance, and further induced serious disorder of colon function. Although ACPO is well-known clinically, it remains poorly understood and continues to challenge physicians and surgeons in many respects. Therefore, we alert physicians to be aware of this disease in order to improve the outcome of ACPO.

AUTHORS’ NOTE
The authors declare that they have no conflict of interest.

REFERENCES