

Ogilvie's syndrome: an uncommon gastrointestinal complication following coronary artery bypass graft surgery

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ABSTRACT

Ogilvie's syndrome or acute colonic pseudo-obstruction is characterized by an acute distention of the large bowel in the absence of any mechanical obstruction usually occurring in critical illness or following an extensive surgery. It is a rare gastrointestinal complication of open heart surgery. Massive distention of the colon may cause perforation in the cecum which causes subsequent fecal peritonitis and associated with a high mortality rate. In this article, we report a case of acute colonic pseudo-obstruction occurring following coronary artery bypass graft surgery and our treatment approach is discussed.

Keywords: Coronary artery bypass graft surgery; neostigmine; Ogilvie's syndrome.

Ogilvie's syndrome also called as colonic pseudo-obstruction is an acute colonic dilatation without any mechanical obstruction which may develop after surgery or systemic illness.^[1] It is seen in less than 3.5% of patients after cardiovascular or thoracic surgery.^[2,3] This syndrome is a type of megacolon syndrome and it remains as a poorly understood condition which is characterized by massive dilatation of the colon and the presence of fluid levels on abdominal X-ray.^[4] It is also associated with a broad range of medical and surgical conditions including open heart surgery, trauma, other surgical interventions, malignancy, and pregnancy.^[5] It is a rare, but lethal postoperative gastrointestinal complication following coronary artery bypass graft (CABG) surgery with a mortality rate as high as 50%.^[4] In this report, a patient who developed an acute colonic pseudo-obstruction (ACPO) following CABG and the treatment approach was discussed.

CASE REPORT

A 67-year-old male patient with no significant history except hypertension for the past five years admitted to the cardiology department with unstable angina pectoris. Coronary angiography revealed multivessel coronary artery disease and he was scheduled for CABG surgery. He had a smoking history of two-packs per day for the past 50 years.

Preoperative blood tests were normal. Echocardiographic evaluation revealed a reduced

ventricular function with a left ventricular ejection fraction of 35% with apical dyskinesia and no significant valvular pathology. His routine anti-hypertensive prescription included an angiotensin receptor blocker monotherapy.

After performing four vessel CABG surgery, the patient was transferred to the intensive care unit with an infusion of moderate doses of positive inotropic agents. The intra-aortic counter pulsation was established in the postoperative seventh hour due to the low cardiac output. Hemodynamically-stabilized patient was extubated on the postoperative first day. The inotropic agents were initiated to attenuate gradually. The otherwise stable patient developed an abdominal distention during the end of postoperative first day. Gastrointestinal symptoms worsened on the second day. All blood tests and blood gas analysis produced normal results without any electrolyte imbalance.

On physical examination, diminished bowel sounds with a markedly distended abdomen were inspected. The abdominal palpation was painful. However, there was no rebound, an indicator of the peritoneal irritation. The erected position plain abdominal roentgenogram revealed a generalized significant

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colonic flatus (Figure 1). Abdominal ultrasound investigation showed no intra-abdominal pathology regarding neither an organomegaly nor a peritoneal free fluid. There was no evidence of a mechanical bowel obstruction or an intestinal perforation, either. A nasogastric (NG) and a rectal tube were introduced following a useless rectal enema. After the constitution of nil per os (NPO), parenteral nutrition (Kabiven Peripheral™, Fresenius Kabi AG, Germany) was initiated to maintain a daily calorie intake. The pharmacologic agents which promote the bowel motility such as metoclopramide and simethicone/alverin citrate were initiated with a spasmolytic agent hyoscine-N-butylbromide. Intravenous fluid and electrolyte replacement continued for hydration as the oral intake had been totally stopped. The intra-aortic balloon pump was retrieved as the hemodynamic parameters were normal. He was mobilized to reconstitute the intestinal motility. Despite all these supportive measures, no fecal passage or discharge of flatus through the rectal tube were achieved. Purgation, enemas, decompression of the flatus via the gastric tube failed.

In the postoperative fourth day, abdominal distention progressed to a risky level and roentgenogram showed a further distention of the colon (Figure 2). Continued medical treatment failed and the patient was unable to be mobilized due to pain. As the

diagnosis was considered to be Ogilvie's syndrome, we decided to administrate neostigmine methylsulfate, a parasympathomimetic agent. At two hours following the initial intravenous bolus dose of 2.5 mg neostigmine methylsulfate, the bowel gas output was observed. The patient was able to defecate at six hours following the initial dose and the abdominal distention gradually resolved. No side effects except a slight bradycardia (heart rate of 65/bpm) were observed. Despite the reconstitution of the intestinal motility, the medication was continued for two more days with a daily dose of 2.0 mg intravenously.

The patient was discharged in the postoperative eight day with a well overall hemodynamic condition with no residual abdominal symptoms.

DISCUSSION

Progressive colonic dilatation following surgery is clinically indicative of the diagnosis of Ogilvie's syndrome in the absence of a mechanical obstruction.^[4] In 1948, Sir William Heneage Ogilvie, the Chilean-born British gastrointestinal surgeon and orthopedist, initially described a novel clinical syndrome with a report of two cases.^[6] This new syndrome which is characterized by acute abdominal pain, constipation and large bowel distention without a mechanical obstruction was then named as Ogilvie's syndrome.



Figure 1. The plain abdominal roentgenogram showing moderate colonic distention (postoperative day 2).



Figure 2. The plain abdominal roentgenogram showing severe colonic distention (postoperative day 4).

Although the exact pathophysiology of the ACPO is still obscure, a parasympathetic deprivation is thought to be responsible.^[7]

Ogilvie's syndrome is mostly seen in sexagenarians and septuagenarians. Also, it is two times higher in males than females.^[4] The initial studies investigated the etiology were concentrated on cesarean sections. However, later studies revealed that cardiothoracic surgery as well as lumbar spine and hip procedures were dominantly responsible.^[4,8]

Although the pathophysiological mechanism of Ogilvie's syndrome following cardiovascular surgery still remains to be elucidated, it is often thought to be neurogenic. The possible scenario explaining the mechanism of the colonic pseudo-obstruction seems to be the lack of parasympathetic innervation to the colon.^[4] The success of a parasympathomimetic drug neostigmine in the treatment of the ACPO also proves this theory of neuropraxia.^[1,5] If it is a type of a neurological dysfunction, what could be the cause of this parasympathetic neuropraxia? Is it related to a trauma of the parasympathetic ganglia? Alternatively, is it solely because of excessive sympathetic discharge due to the pain or stress during the operation?

An intraoperative trauma or direct manipulation of the ganglia or its branches seems to be impossible in cardiothoracic, orthopedic, and the lumbar spinal cord operations. However, the vagus nerve, which is the main parasympathetic trunk of the gastrointestinal system, may be injured during CABG surgery due to local hypothermic slush application or during extensive lateral pericardiotomy.^[9] Besides, sympathetic discharge may occur, when the patient accidentally awakes due to inadequate anesthesia and this excessive sympathetic surge may easily cause relative loss of parasympathetics.^[4] Although it is still unproven, the extracorporeal circulation during CABG surgery may contribute to neurogenic disarrangement.^[4] An elongated extracorporeal circulation may also lead to increased ischemia of the parasympathetic ganglia due to hypotension, thereby, eventually cause synaptic retardation.^[10] In our case, a reasonable cardiopulmonary bypass time was achieved comparing to a four-vessel CABG surgery. The circulation pressure of the heart-lung machine was around 70 mmHg during the extracorporeal circulation period which corresponds to the normal mean value. In the light of all these discussed hemodynamic parameters, the possibility of a mechanical or ischemic trauma can be excluded.

Furthermore, the diagnosis depends on the physical examination, follow-up and imaging with an erected abdominal roentgenogram, irrespective of the pathophysiology. During the postoperative period, a massive distention of the abdomen with impaired flatus or stool outflow should always remind the Ogilvie's syndrome.^[4]

In the treatment of Ogilvie's syndrome, the NPO, NG, and rectal tubes should be the first to constitute by means of immediate colonic decompression. The supportive intravenous medication including fluid and electrolyte replacement should be applied afterward. Follow-up may lead to complete resolution of the ACPO or may show further progression. In this presented case, as the supportive measures did not improve the patient's overall status, neostigmine was initiated as an advanced treatment.^[11] Neostigmine is a parasympathomimetic which inhibits the acetylcholinesterase enzyme, which is responsible for the breakdown of the acetylcholine (Ach) molecule. As it blocks the binding site of the acetylcholinesterase, the enzyme can no longer interfere the Ach before it interacts with the receptors of the postsynaptic membrane.^[12] It eventually helps the threshold to be reached and the impulse can be triggered in the next neuron. Increase in the parasympathetic activity further initiates the intestinal motility resulting the resolution of the abdominal distention.^[13]

When any other medical treatments are useless to resolve the state of ACPO, colonic decompression via colonoscopy, percutaneous decompression or laparotomy should be considered.^[8,14] If left untreated, colon perforation due to necrosis of the cecum may cause fecal peritonitis and death, eventually.^[15]

In conclusion, in case of an acute megacolon following the CABG surgery, the diagnosis should be considered as Ogilvie's syndrome. Immediate diagnosis and effective treatment are necessary to prevent colonic necrosis and the eventual perforation. In addition to the supportive treatment, neostigmine, a parasympathomimetic, can be used to reconstitute the intestinal motility. Surgical option should be reserved as the last chance due to its high potential for mortality.

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