Acute Colonic Pseudo-obstruction (The Ogilvie syndrome): A Case Report and Review of Literature

N Agarwal, A Mishra, Z Kayali

Citation

Abstract
Acute colonic pseudo-obstruction first described by Sir Heneage Ogilvie in 1948 is a poorly understood syndrome and is characterized by signs of large bowel obstruction without a mechanical cause. This condition usually develops in hospitalized patients and is associated with a range of medical and surgical conditions. If inappropriately managed, may cause ischemic necrosis and colonic perforation, with a mortality rate as high as fifty percent. An imbalance in the autonomic innervations (sympathetic over activity and parasympathetic suppression) has been thought to be the pathophysiological factor in the causation of this condition. This article describes the case of an 81-year old man with multiple medical problems who developed Ogilvie syndrome after a hip surgery. The clinical characteristics, diagnostic methods and management of Ogilvie syndrome are reviewed.

Department and institution or hospital where the work was done:
Department of Internal Medicine, University of Iowa Hospital, Iowa City, Iowa, 52246. USA

INTRODUCTION
Acute colonic pseudo-obstruction first described by Sir Heneage Ogilvie in 1948 is a poorly understood syndrome and is characterized by signs of large bowel obstruction without a mechanical cause. This condition usually develops in hospitalized patients and is associated with a range of medical and surgical conditions. If inappropriately managed, it may result in ischemic necrosis and colonic perforation, with a mortality rate as high as fifty percent. An imbalance in the autonomic innervations (sympathetic over activity and parasympathetic suppression) has been thought to be the pathophysiological factor in the causation of this condition.

CASE REPORT
An 81-year-old male with history of hypertension, chronic obstructive pulmonary disease, and depression was initially admitted for the workup of a syncopal episode. During his hospital stay, one night he tripped while walking to the bathroom and sustained an intertrochanteric fracture of the right femur. He was started on morphine 2 mg intravenously every 4-hour prn and bulk laxatives. He was also on bupropion and sertraline for depressions and haloperidol for agitation. Day # 1 after the accident he underwent hip surgery (open reduction and internal fixation). Day #5 morphine was stopped, as he no longer complained of pain. On day #11 he complained of dull abdominal pain and was found to have slight abdominal distension. By day #12 he had developed marked abdominal distension. Examination showed distended and tense abdomen on palpation with mild RLQ tenderness. Auscultation of abdomen revealed high pitched and markedly diminished bowel sounds. There was no evidence of any rigidity and rebound tenderness. Laboratory results showed a leukocyte count of 4800/ mm$^3$ with normal differential and serum electrolytes with in the normal range (sodium 137 meq/l, chloride 100meq/l, potassium 4.2 meq/l, bicarbonate 27meq/l, magnesium 2.0meq/l and anion gap of 10meq/l). Abdominal x-ray revealed massive dilatation of cecum (cecal diameter was measured to be 17 cm)(figure 1), diffuse dilatation of ascending colon and transverse colon, with presence of gas all the way to lower rectal region without any small bowel dilatation. There was no radiological evidence of any free air in the peritoneal cavity or within the bowel wall.
On the basis of clinical signs and symptoms and the classical abdominal x-ray findings, patient was diagnosed to have developed colonic pseudo obstruction (Ogilvie syndrome). Treatment was started with nasogastric tube suction, tap water enema and periodic change of position. Antidepressant (bupropion and sertraline) and antipsychotic (haloperidol) were stopped. This was followed by administration of neostigmine 2mg intravenously over 10 minutes under continuous EKG monitoring and atropine at bedside. Within few minutes of receiving neostigmine patient passed a flatus followed by a small liquid stool. Slow rectal tube suction was also started. On day # 13 examination showed improvement in abdominal distension and x-ray showed decrease in cecal dilatation to 11 cms. He was continued on nasogastric and rectal suctioning with close follow up of serum electrolytes. On day #14 (that is 48 hours after onset of symptoms) examination showed soft mildly distended abdomen with almost normal bowel sound. Abdominal film revealed a cecal diameter of 5cm with nonspecific bowel gas pattern (figure 2).

DISCUSSION

Though Ogilvie syndrome is a rare condition, its importance lies in its ability to cause dangerous complications in affected patients with high mortality rate\(^4\). Predisposing conditions include trauma, infection, surgery, drugs (narcotic analgesics, antidepressants, antipsychotic, calcium channel blockers, narcoleptics) and several other ill-defined factors\(^5\).

Colonic motility is under autonomic regulation, being increased by parasympathetic and inhibited by sympathetic nervous system. The above-mentioned conditions can alter the fine balance between these two systems leading to excessive parasympathetic suppression, sympathetic stimulation or both. This imbalance there by results in extreme suppression of colonic motility resulting into colonic atony or pseudo-obstruction.

The clinical presentation is fairly classic and diagnosis relatively easy, requiring only an abdominal x-ray. Patient typically presents with abdominal distension, diminished or absent bowel sounds and/or abdominal pain and vomiting. Abdominal x-ray shows dilated caecum (more than ten centimeter) and diffuse dilatation of other parts of colon (ascending and transverse colon)\(\_\_\_\)\. Presence of gas shadows
in rectum is the definitive evidence, which rules out any mechanical obstruction. In the absence of rectal gas shadows with high clinical suspicion of Ogilvie syndrome, a radiographic enema is all that is required to rule out distal obstruction.

For many years the treatment has included conservative management like withholding enteral feeding; correcting electrolyte imbalance (e.g., hypokalemia, hypomagnesaemia); withholding any drugs known to decrease colonic motility; decompression using a nasogastric tube, rectal tube (e.g., Levine catheter); positional changes (e.g. placing the patient in a prone position or right lateral position) and colonoscopic decompression. The pharmacologic management has consisted of promotility agents (e.g., cisapride and erythromycin) and lately parasympathomimetic agents like neostigmine. However, traditionally these pharmacological agents were superseded by the above mentioned non pharmacological management both in literature and in practice till recently when a randomized case controlled trial established the relative superiority, efficacy and ease of treatment of Ogilvie syndrome with parenteral neostigmine (in doses of 1-2 mg intravenously over a period of three to five minutes and to be repeated once if required in 2-3 hours). Neostigmine reversibly inhibits acetyl cholinesterase and thus potentiates the activity of acetylcholine, resulting in increase in colonic motility (a parasympathomimetic effect). As kidneys mainly excrete it, its serum half-life is prolonged and side effects accentuated in patients with renal dysfunction. Because of its potential of causing bradycardias and bronchospasm it may be used cautiously in susceptible patients under continuous EKG monitoring and readily available atropine.

**CONCLUSION**

Colonic pseudo-obstruction (Ogilvie syndrome) is a rare but potentially dangerous condition in hospitalized patients, resulting due to parasympathetic suppression. The ease with which it can be diagnosed early because of its classical presentation (both clinical and radiographic) makes it amenable to an early treatment, which can prevent its fatal complications. The treatment with intravenous neostigmine has proved very effective, preventing in many cases prolonged periods of uncomfortable traditional conservative management and even diminishing the need for surgical interventions. This case demonstrates the subtlety with which it can rapidly progress to massive cecal dilatation (with minimal symptoms) thereby highlighting the need for high index of awareness to diagnose this condition.

**References**

Author Information

Neeraj Agarwal, MD
Resident, Department of Internal Medicine, University of Iowa Hospital and Clinics

Archana Mishra, MD
Postdoctoral Fellow, Department of Pathology, University of Iowa Hospital and Clinics

Zeid Kayali, MD
Fellow, Division of Gastroenterology & Hepatology, University of Iowa Hospital and Clinics