

CLINICAL REVIEW*Volume 10 Issue 1****Ogilvie's Syndrome: Acute Colonic Pseudo-obstruction.
A Review for Residents*****Tyler Bayliss, BS¹, Caleb Clark, BS¹,
Errington C. Thompson, MD, FACS, FCCM¹****ABSTRACT**

Ogilvie's syndrome (acute colonic pseudoobstruction) was first described in 1948. Acute colonic pseudoobstruction can occur in a variety of clinical settings, including post-surgical, obstetrics, pelvic surgery, critical care and sepsis. Clinicians need to recognize the syndrome early. Colonic distention without evidence of obstruction can be seen on plain films of the abdomen or CT scan. Successful therapies, including bowel rest, neostigmine and colonoscopic decompression, have been used. Avoiding respiratory compromise from abdominal distention and colonic perforation of the primary goals of treatment. Surgical intervention should be reserved for patients who are refractory to medical treatment or develops signs and symptoms of colonic ischemia or perforation.

Author affiliations are listed at the end of this article.

Corresponding Author:
Errington C. Thompson, MD
Marshall University
Joan C. Edwards
School of Medicine
thompsoner@marshall.edu

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INTRODUCTION

Ogilvie's syndrome, also known as acute colonic pseudo-obstruction (ACPO), was first described by Sir William Heneage Ogilvie in 1948.¹ His pivotal publication detailed a series of cases in which patients exhibited symptoms mimicking mechanical obstruction of the colon. Following surgical intervention, patients were found to harbor tumors encasing the celiac axis, infiltrating the liver and mesentery, and causing peritoneal studding. Ogilvie postulated that this neoplastic growth disrupted the sympathetic nerve supply to the bowel, leaving the parasympathetic nerves unchecked. As a result, he theorized that an elevated parasympathetic input to the bowel instigated the pseudo-obstruction.

This review aims to inform and educate medical students and residents on the pathophysiology, clinical presentation, complications, and treatment of Ogilvie's syndrome.

PATHOPHYSIOLOGY

The precise definition of Ogilvie's syndrome has been nebulous in the literature. It is most functionally defined as the manifestation of colonic obstruction symptoms without evidence of a mechanical blockage.²

Ogilvie's syndrome is generally categorized into primary and secondary pseudo-obstruction. Primary pseudo-obstruction may arise in conditions such as familial visceral myopathy^{3,4} or diffuse motility disorders affecting the autonomic innervation of the intestinal wall.⁵ Secondary pseudo-obstruction, initially described by Ogilvie and frequently encountered in clinical settings, is far more common.²

In seeking to comprehend acute colonic pseudo-obstruction, understanding the physiological role of the colon is pivotal. The colon extracts water and electrolytes from intestinal contents while storing these contents until evacuation.⁶ Myenteric plexus



clusters, dispersed throughout the small and large intestines, govern peristaltic contractions. Parasympathetic innervation, supplied by the right vagus nerve to the right and transverse colon and sacral nerves and S2-S4 to the left colon, predominantly influences peristalsis/mobility.⁷ Meanwhile, sympathetic stimulation, originating from the sacrum and supplied through the pelvic plexus, generally decreases mobility.⁷ Contrary to Ogilvie's original manuscript, sympathetic nerves seem to dampen, not stimulate, peristalsis.

Despite extensive research, the pathophysiology of Ogilvie's syndrome remains unclear. Typically, patients present with right and transverse colon dilation.⁸ Current theories propose an imbalance in sympathetic and parasympathetic innervation to the left colon as the cause of right colon dilation.^{8,9} Several indirect evidences support this theory, including that a considerable percentage of Ogilvie's syndrome patients have obstetric/gynecologic/urologic etiology.¹⁰⁻¹² In addition, neostigmine effectively resolves Ogilvie's syndrome symptoms,^{13,14} and epidural anesthesia reverses the effects of colonic pseudo-obstruction.¹⁵

While autonomic denervation theory is prevalent, alternative theories include the following: pharmacology (implicating medications with anticholinergic effects),⁹ infectious etiology (herpes zoster infecting enteric ganglia),¹⁶ vascular (reduced perfusion between superior and inferior mesenteric arteries),^{17,18} hormonal (suggesting a role for prostaglandin E),¹⁹ and metabolic (conditions such as hypokalemia or uremia).⁹

Ogilvie's syndrome has been associated with a plethora of medical abnormalities. Vanek and Al-Salti's 1986 retrospective analysis of over 400 Ogilvie's syndrome cases categorized patients into medical and postsurgical.⁸ Medical patients were further divided into subcategories: those with infections, cardiac issues, neurological disorders, cancers, pulmonary diseases, metabolic disturbances (such as electrolyte imbalances), renal diseases, and others. Postsurgical patients were divided into subcategories including trauma, obstetrics and gynecology, abdominal pelvic surgery, orthopedics, urologic surgery, thoracic and cardiovascular surgery, neurosurgery, and more. Such

categorization should theoretically assist clinicians in comprehending the disease process more efficiently.

CLINICAL PRESENTATION

Various clinical situations may precipitate acute colonic pseudo-obstruction. Current clinician guidance is that the syndrome typically emerges post-surgery but may also occur following childbirth, cardiac events, and systemic infections.²⁰ Symptom onset is often subtle rather than dramatic. Manifestations may include abdominal pain, nausea, and vomiting, with or without obstipation. Some patients may present with diarrhea, which can convolute the clinical picture.^{9,21} Early in the patient's course, the abdominal examination is typically benign and devoid of peritonitis signs. At this stage, abdominal X-rays may depict a nonspecific bowel gas pattern.

As pseudo-obstruction progresses, the signs and symptoms indicative of distal colonic obstruction become more prominent. Patient discomfort escalates as abdominal distension increases. Plain abdominal X-rays will exhibit right colon/cecum distention.^{9,20}

At this juncture, the clinician must diagnose the condition accurately, as eliminating mechanical obstruction is essential. This can be achieved through colonoscopy, a water-soluble contrast enema, or a CT scan of the abdomen and pelvis, which will assist in diagnosing colonic pseudo-obstruction.²

However, clinicians should not overemphasize the size of the cecum. Literature fails to concur on the extent of dilatation necessary for a definitive diagnosis of Ogilvie's syndrome. Some studies propose a minimum 9 cm colonic diameter, whereas others argue that this figure signals the onset of increased perforation risk.²¹ Certain studies even suggest a threshold of 12 cm before considering elevated perforation risk.^{8,9} These variances in cecal diameter are expected. Acute cecal dilatation may necessitate lesser distention before rupture, whereas gradual dilatation over days could potentially withstand greater expansion before rupture. The condition's chronicity may account



for the differences in cecal diameter. Hence, instead of focusing on the cecum's absolute size, clinicians should consider the overall appearance of the CT scan and the patient's overall condition. In general, the CT scan of a patient with acute colonic pseudo-obstruction exhibits dilation of the cecum, right colon, and transverse colon. In contrast, the left colon appears decompressed, starting approximately at the splenic flexure and including the sigmoid colon and rectum (Figures 1 and 2). Using our algorithm (Figure 3), if the patient has a cecum of 12 cm but is nontender with normal vital signs, one should consider a trial of neostigmine prior to taking the patient to the operating room.

COMPLICATIONS

The principal complication of Ogilvie's syndrome is colonic perforation, indicated by fever, leukocytosis,

and peritoneal signs. Colonic perforation incidence ranges between 15 and 20%, with mortality rates escalating to 40 to 50% following perforation. Factors such as age, cecal perforation, colonic ischemia, and a delay of over 6 days before colonic decompression are associated with a poor prognosis.²

TREATMENT

Ogilvie's syndrome management involves a comprehensive strategy, primarily encompassing a nasogastric tube, fluid resuscitation, correction of electrolytes, and colonoscopic decompression (Figure 3).²⁰ Clinicians must exclude a distal obstruction. If the patient has a distal obstruction, the obstruction is the cause of the patient's colonic dysfunction, and the obstruction must be addressed. If there is no distal obstruction, the clinician should review all the patient's medications. Any medications



FIGURE 1: Scout CT of the abdomen and pelvis. Marked dilation of the colon. Permission to use photo from Geetha Virupakshappa, Radiopaedia.org, rID: 155963



FIGURE 2: CT scan of the abdomen. Dilation of the transverse colon with several loops of decompressed small bowel seen. Permission to use photo from Bálint Botz, Radiopaedia.org, rID: 63714



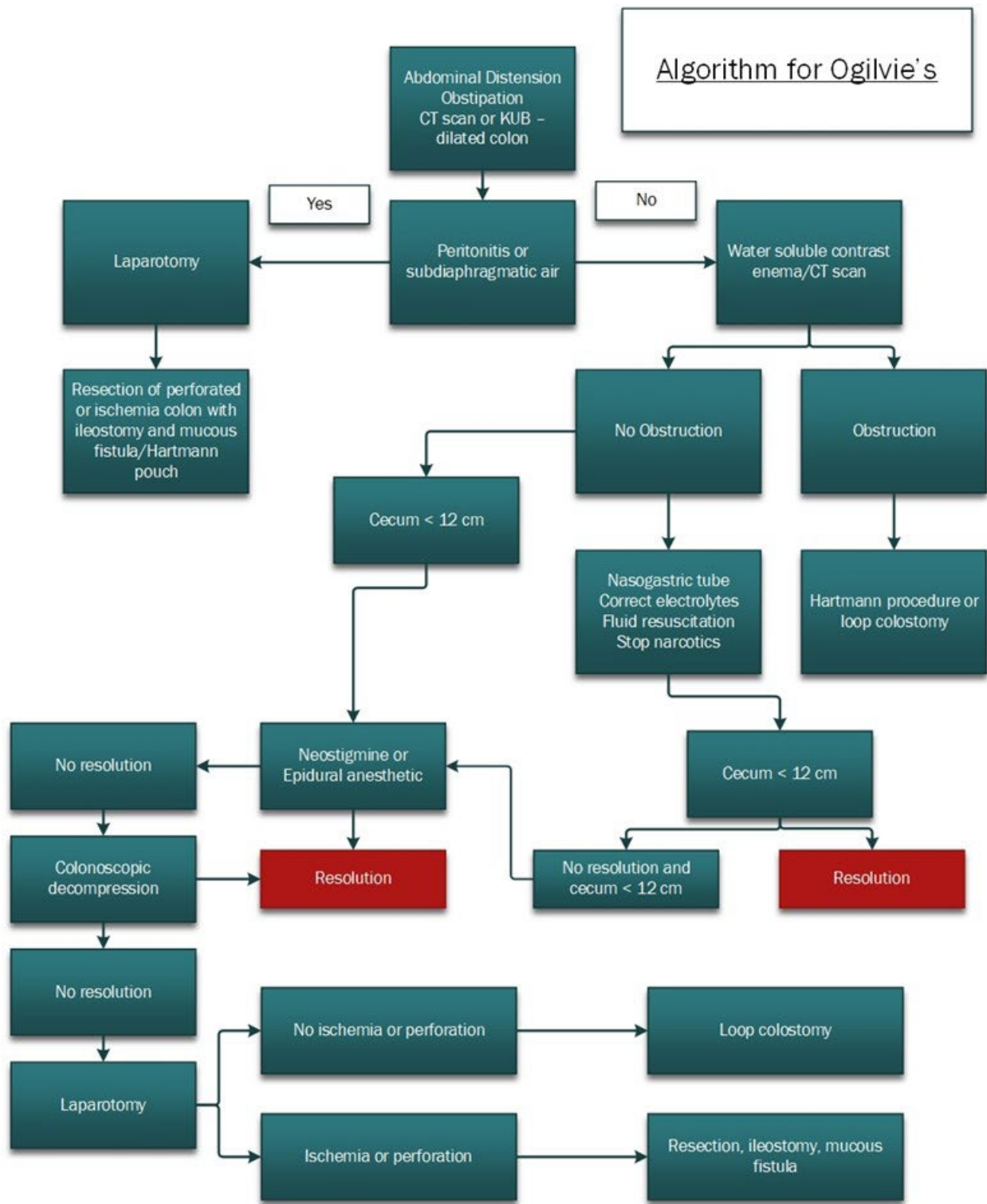


FIGURE 3: Algorithm for Ogilvie's Syndrome



associated with Ogilvie's syndrome that slow colonic transit should be discontinued, such as neuroleptics, clonidine, antiparkinsonian medications, opioids, anticholinergics, and some antihypertensives.^{9,22}

Following NG tube decompression, distal obstruction exclusion, and cessation of contributing medications, patients requiring further intervention should undergo a trial acetylcholine-esterase inhibitor, such as neostigmine^{13,14} or pyridostigmine⁹, or undergo colonoscopy.²³ Colonoscopic decompression has proven effective in select patients. The procedure's success hinges on the endoscopist's skill, introducing the scope and maneuvering through the unprepared colon while decompressing the right colon/cecum without over-insufflation.²⁴ One of the procedure's benefits is the early detection of colonic ischemia (Figure 4). If ischemia is observed, the procedure is terminated, and the patient is immediately transferred to the operating room.

Although neostigmine use has proven beneficial, the drug is not without risks. Its adverse side effects include arrhythmia, bradycardia, cardiac arrest,

heart block, and severe hypotension, necessitating its administration in a monitored setting such as the intensive care unit.²⁵ The standard 2-2.5 mg dose is delivered via IV push, with patients typically responding within 20 to 30 minutes. Neostigmine can be repeated 3 to 4 hours post-initial dose, and an abdominal X-ray can determine the therapy's effectiveness through observed cecal diameter reduction.⁹

Epidural anesthesia, using Bupivacaine or lidocaine, has shown quick results in patients with colonic pseudo-obstruction but has not yet gained widespread acceptance in major medical centers.^{15,26}

Cecostomy was suggested as an effective decompression method for the right colon in several papers published in the late 1980s and early 1990s.²⁷ Despite its relative ease of performance, either laparoscopically or percutaneously by radiology, cecostomy carries a high complication rate, including leakage around the cecostomy tube leading to fecal peritonitis. Though the cecostomy tube's removal spontaneously closes the fistula in most patients,

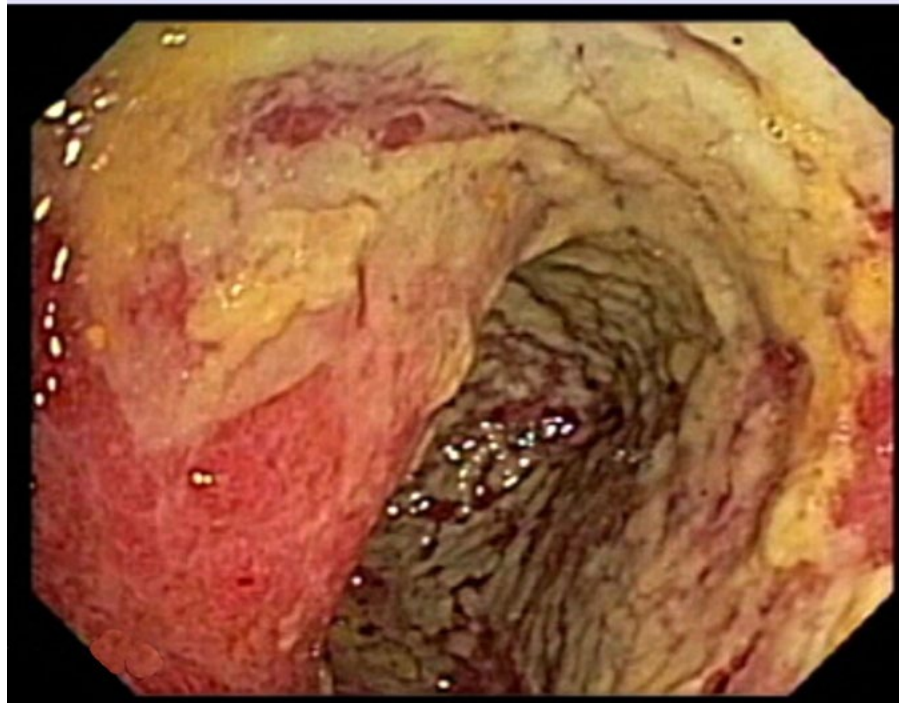


FIGURE 4: Colonoscopic view of ischemic colitis. Photo from www.gastrointestinalatlas.com. Permission granted by Julio Murra-Saca, MD



some require additional surgery for fistula closure, relegating cecostomy to historical reference.²⁰

If conservative therapy or perforation necessitates surgical intervention, surgeons should consider laparoscopic intervention versus exploratory laparotomy. In the absence of perforation and extreme distension, the patient can be examined laparoscopically for ischemia evidence. If no ischemia is detected, a transverse loop colostomy is performed. Conversely, if ischemic changes or perforation are present, the surgeon must determine the most suitable procedure considering the patient's hypotension. A subtotal colectomy with an ileostomy may be the best choice, considering the patient's pre-existing health and intraoperative hemodynamic stability.^{2,20,28}

CONCLUSION

Ogilvie's syndrome presents a unique diagnostic challenge due to its ambiguous nature and lack of definitive diagnostic tests. A laboratory test or X-ray cannot conclusively diagnose acute colonic pseudo-obstruction (Ogilvie's syndrome). Consequently, practitioners need to maintain an acute sense of vigilance. The diagnosis of Ogilvie's syndrome should be contemplated in any patient exhibiting abdominal distention, nausea, and vomiting sans evidence of mechanical obstruction.

Diagnostic criteria rely heavily on clinical presentation. Exclusion of a distal obstruction is requisite, with the patient subsequently undergoing a CT scan of the abdomen and pelvis. Colonic pseudo-obstruction can be addressed through colonoscopy-based decompression or the application of neostigmine to enhance motility. Patients presenting with escalating abdominal pain and leukocytosis should be suspected of having a perforation. Such patients require immediate operative intervention.

In conclusion, Ogilvie's syndrome remains a complex medical mystery that calls for astute clinical acumen and swift decision-making for diagnosis and management. Clear clinical guidelines based on patient symptoms and reactions, alongside the application of suitable treatment strategies, are crucial to managing this condition effectively. The key

to successful intervention lies in prompt detection and treatment, highlighting the need for further research to demystify this condition and develop more definitive diagnostic tools.

AUTHOR AFFILIATIONS

1. Marshall University Joan C. Edwards School of Medicine, Huntington, West Virginia

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