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OGILVIE'S SYNDROME IN SURGICAL PRACTICE: A NARRATIVE REVIEW OF ETIOLOGY AND THERAPEUTIC APPROACHES

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ABSTRACT Ogilvie's syndrome, or acute colonic pseudo-obstruction, is a rare but serious condition characterized by colonic dilation without mechanical obstruction. It primarily affects hospitalized and postoperative patients, especially those with metabolic disturbances or exposed to medications that impair intestinal motility.This narrative review summarizes the current understanding of the etiology, risk factors, clinical presentation, diagnostic approach, and therapeutic strategies for Ogilvie's syndrome in surgical practice.A comprehensive literature review was conducted using PubMed, Scopus, Embase, and Web of Science. A total of 15 high-quality references were selected for inclusion.Early identification and escalation from conservative to invasive management reduce morbidity and mortality. Neostigmine remains the pharmacological cornerstone, while colonoscopic decompression and surgery are reserved for refractory or complicated cases.

KEYWORDS : Ogilvie's syndrome, Acute colonic pseudo-obstruction, Neostigmine, Surgical complications, Postoperative care.

INTRODUCTION

Acute colonic pseudo-obstruction, also known as Ogilvie's syndrome, is a rare but potentially life-threatening condition characterized by massive colonic dilatation without an underlying mechanical cause. Initially described by Sir William Ogilvie in 1948, this syndrome typically affects hospitalized or postoperative patients, particularly those with underlying cardiac, neurologic, or metabolic disorders (1). In surgical settings, its incidence is increasing due to the frequent use of medications such as opioids, calcium channel blockers, and immunosuppressants, all of which can impair gut motility (2). Despite its clinical relevance, Ogilvie's syndrome remains under-recognized, often misdiagnosed, and inadequately managed, leading to serious complications including ischemia and perforation. Early recognition and appropriate treatment—ranging from conservative measures to pharmacologic and endoscopic decompression—are essential to prevent morbidity and mortality. This narrative review aims to explore the current understanding of the etiology, risk factors, clinical features, and therapeutic strategies of Ogilvie's syndrome in surgical practice.

METHODS

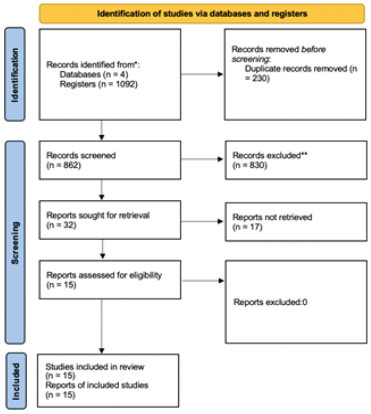
This narrative review was conducted to synthesize current knowledge on Ogilvie's syndrome in the surgical setting. A comprehensive literature search was performed across four electronic databases: PubMed, Scopus, Embase, and Web of Science. The search included articles published up to March 2025, without language restrictions. The keywords used were: "Ogilvie's syndrome," "acute colonic pseudo-obstruction," "surgical complications," "postoperative ileus," and "neostigmine therapy." Boolean operators (AND, OR) were applied to refine the search. Inclusion criteria focused on original articles, clinical trials, case series, systematic reviews, and expert guidelines that addressed the etiology, diagnosis, or management of Ogilvie's syndrome in surgical patients. Studies exclusively involving pediatric populations or chronic pseudo-obstruction were excluded. Titles and abstracts were screened for relevance, followed by full-text review. Data were extracted narratively, emphasizing clinical applicability. A total of 15 references were selected and cited in the final version of this review based on their quality and relevance.

EPIDEMIOLOGY

Acute colonic pseudo-obstruction, or Ogilvie's syndrome, predominantly affects hospitalized or postoperative patients and is more commonly observed in males over the age of 60 (2). Although the exact incidence is difficult to determine due to underreporting and misdiagnosis, large retrospective series estimate an occurrence of approximately 100 cases per 100,000 hospital admissions annually (2). The syndrome typically involves the cecum and right colon but may extend to the rectum in severe cases. Postoperative patients, particularly those undergoing orthopedic, obstetric (e.g., cesarean section), or abdominal surgeries, are at elevated risk. For instance, in a systematic review of 66 postpartum cases, 92% occurred after cesarean section (3).

Additionally, certain comorbidities such as trauma, cardiac disease, infection, renal failure, and metabolic disturbances further increase susceptibility (2). Medications that impair colonic motility—like opioids, calcium channel blockers, anticholinergics, and immunosuppressants—have also been linked to its onset (4). In transplant recipients, especially after renal transplantation, the cumulative dose of corticosteroids and the use of mycophenolate mofetil appear to be contributing factors (4). While rare in the pediatric population, cases have been reported, particularly in immunocompromised children or those with neurologic impairments, underscoring the syndrome's broad yet selective distribution.

Figure. PRISMA.



ETIOLOGY AND RISK FACTORS IN SURGICAL PATIENTS

The pathogenesis of Ogilvie's syndrome is believed to involve an imbalance between sympathetic and parasympathetic innervation of the colon, leading to colonic atony and functional obstruction (5). This autonomic dysregulation may

be triggered by surgical stress, systemic illness, or specific pharmacologic agents. In surgical patients, especially those undergoing orthopedic procedures (e.g., hip replacement), cesarean sections, or abdominal operations, the incidence increases due to factors such as postoperative immobility, electrolyte disturbances, and opioid use (5,6).

Postoperative ileus is a common differential, but Ogilvie's syndrome should be suspected when colonic dilatation is disproportionate or persists despite standard supportive care. Risk factors include advanced age, male sex, major trauma, and comorbid conditions such as heart failure, sepsis, or chronic kidney disease (6).

Medication-induced hypomotility is a significant contributor in the perioperative context. Drugs with anticholinergic properties, such as butylscopolamine, as well as calcium channel blockers and narcotics, have been implicated (7). Furthermore, immunosuppressive regimens, especially those including corticosteroids and mycophenolate mofetil, are notable triggers in transplant recipients (7). Recognition of these risk factors is critical for early diagnosis and timely intervention, especially in surgical patients who may already be at increased risk of complications such as ischemia and perforation.

CLINICAL MANIFESTATIONS AND DIFFERENTIAL DIAGNOSIS

Ogilvie's syndrome typically presents with progressive abdominal distension, often within 3 to 7 days postoperatively, though it may occur as rapidly as 24 to 48 hours after a triggering event (7). Patients may report diffuse abdominal discomfort or cramping pain, nausea, and vomiting. Constipation is common, although paradoxical diarrhea may also occur in up to 40% of cases due to overflow around fecal impaction (7). In severe presentations, the distension can lead to respiratory compromise, especially in older adults or those with underlying cardiopulmonary disease. Physical examination frequently reveals a tympanitic, distended abdomen with preserved bowel sounds in up to 90% of patients (8).

Laboratory tests are generally nonspecific but may show electrolyte imbalances such as hypokalemia or hypomagnesemia. Leukocytosis may indicate impending ischemia or perforation. Imaging is essential to confirm the diagnosis. Abdominal radiographs typically reveal massive colonic dilation, particularly of the cecum and right colon, with air-fluid levels. A computed tomography (CT) scan is the preferred modality as it can exclude mechanical obstruction and identify complications (9).

The differential diagnosis includes mechanical large bowel obstruction, toxic megacolon, and chronic intestinal pseudo-obstruction. Mechanical obstruction can usually be differentiated by identifying a transition point or obstructing lesion on imaging. Toxic megacolon, in contrast, is associated with systemic toxicity, fever, bloody diarrhea, and a loss of haustral pattern on imaging (10). Other considerations include volvulus, especially of the sigmoid or cecum, and postoperative ileus, which more commonly involves small bowel dilatation.

Accurate diagnosis is crucial, as management strategies differ significantly between these entities. Early identification and differentiation of Ogilvie's syndrome can prevent unnecessary surgical interventions and reduce the risk of complications such as colonic ischemia or perforation.

THERAPEUTIC APPROACHES: FROM CONSERVATIVE TO INVASIVE

The management of Ogilvie's syndrome aims to decompress the colon and prevent complications such as ischemia or perforation. Treatment is guided by clinical stability, cecal diameter, and response to initial interventions (10). In stable patients with mild to moderate symptoms and no evidence of ischemia or perforation, conservative therapy is the first-line approach. This includes bowel rest (nothing by mouth), intravenous fluid and electrolyte correction, discontinuation of motility-impairing drugs (e.g., opioids, anticholinergics), and gentle decompression via nasogastric or rectal tubes (10). Positional changes and mobilization are also encouraged. This approach leads to resolution in 70–90% of cases within 72 hours (10).

If conservative measures fail or if the cecal diameter exceeds 12 cm, pharmacologic treatment with neostigmine is recommended. Neostigmine, a reversible acetylcholinesterase inhibitor, enhances colonic motility by increasing acetylcholine availability (11). A single 2–2.5 mg intravenous dose has shown rapid efficacy in over 80% of patients, often producing bowel movement within minutes (11). Patients must be closely monitored for bradycardia, and atropine should be readily available during administration. Neostigmine is contraindicated in patients with bradyarrhythmias, asthma, or suspected perforation (12).

For patients unresponsive to pharmacologic treatment or those with contraindications, colonoscopic decompression is the next step. This procedure allows direct evacuation of gas and placement of a decompression tube. Although successful in up to 70% of cases, recurrence rates remain high, and perforation risk, though rare, must be considered (13).

If all previous interventions fail or if signs of perforation or peritonitis are present, surgical intervention—typically a cecostomy—is required. Surgery carries the highest morbidity and is reserved for life-threatening cases. Thus, timely recognition and escalation of therapy are essential to prevent complications and improve patient outcomes.

COMPLICATIONS, PROGNOSIS, AND PREVENTION OF RECURRENCE

If not promptly diagnosed and managed, Ogilvie's syndrome can progress to severe complications. The most feared outcomes are colonic ischemia and perforation, particularly when cecal diameter exceeds 12 cm or distension persists beyond six days (13). These complications dramatically increase morbidity and mortality, with perforation associated with mortality rates of up to 50% in some series. Signs such as fever, marked abdominal tenderness, leukocytosis, and peritoneal irritation warrant immediate intervention due to the risk of bowel necrosis (13).

Prognosis is generally favorable with early recognition and appropriate therapy. Most patients respond well to conservative measures or a single dose of neostigmine. However, delayed diagnosis or inadequate management can lead to prolonged hospitalization, need for surgical intervention, and long-term gastrointestinal dysfunction. Recurrence is not uncommon and has been reported in 6–14% of cases, especially in patients with persistent risk factors or inadequate follow-up care (14).

To prevent recurrence, it is critical to address modifiable risk factors. This includes avoiding unnecessary use of motility-impairing medications and ensuring correction of electrolyte imbalances. In some cases, maintenance therapy with osmotic laxatives like polyethylene glycol (PEG 3350) has shown promise in reducing recurrence, especially in patients with colonic hypomotility (14,15). Additionally, structured postoperative care involving early mobilization, bowel regimen protocols, and clinical awareness of Ogilvie's syndrome may reduce incidence and recurrence in surgical

patients. Ultimately, multidisciplinary management involving surgeons, gastroenterologists, and intensivists is often necessary to ensure optimal outcomes. Timely intervention, individualized treatment escalation, and careful monitoring not only reduce acute complications but also minimize the risk of recurrence, supporting a better long-term prognosis for patients affected by this uncommon yet significant condition.

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