Review Article

DOI: 10.7860/JCDR/2026/81975.23130

Acute Colonic Pseudo-obstruction: A Narrative Review of Pathophysiology, Advances in Diagnosis, and Management

Surgery Section

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ABSTRACT

Acute Colonic Pseudo-obstruction (ACPO), also known as Ogilvie's syndrome, is a clinical condition characterised by massive colonic dilatation without mechanical obstruction. It commonly occurs in postoperative, critically ill, or severely immobilised patients. The pathophysiology involves autonomic dysregulation, neuromuscular transmission defects, electrolyte imbalances, and inflammatory mediators. Clinically, ACPO presents with progressive abdominal distension, pain, and altered bowel movements, often mimicking true colonic obstruction. Diagnosis primarily relies on imaging modalities such as plain radiographs and Computed Tomography (CT), while advanced tools like CT colonography, colonic manometry, and Point-of-Care Ultrasound (POCUS) are emerging. Treatment includes conservative supportive care, administration of neostigmine, and colonoscopic decompression. Surgical intervention is reserved for complications such as ischaemia or perforation. Emerging methods, including Percutaneous Endoscopic Cecostomy (PEC) and neuromodulation, are under investigation for prompt treatment of ACPO. Early and timely diagnosis, along with individualised treatment strategies, are essential to prevent severe complications and improve patient outcomes. The present narrative review highlights current knowledge of ACPO, its dynamic pathophysiology, emerging diagnostic options, stepwise management, and novel therapies that may enhance patient outcomes.

Keywords: Colonic manometry, Cytokines, Enteric nervous system, Hypokalaemia, Neostigmine

INTRODUCTION

The ACPO, also known as Ogilvie's syndrome, is characterised by acute colonic dilatation in the absence of mechanical obstruction [1,2]. It was first described in 1948 by Sir William Heneage Ogilvie, a British surgeon, in two patients with metastatic cancer who developed massive colonic dilatation without any obstructing lesion [3]. ACPO occurs in various postoperative, medical, and traumatic contexts, primarily in immobile patients [1]. The pathophysiology involves an imbalance in autonomic innervation of the colon, which can lead to functional obstruction due to increased sympathetic or decreased parasympathetic activity [4]. Historically underdiagnosed, ACPO has gained increasing recognition due to advances in imaging particularly radiographs and CT which help differentiate it from mechanical large bowel obstruction [5,6]. Despite being a functional disorder, ACPO has a high potential for severe complications, including ischaemia and perforation, necessitating timely recognition and intervention [5].

Symptoms and Clinical Features of ACPO

The ACPO usually manifests over 3-5 days but can present abruptly within 48 hours [7]. The primary presentation is progressive abdominal distension, most pronounced in the caecum and right colon [7]. Nausea and vomiting may occur in some patients, while colicky or diffuse abdominal pain is common [5]. Bowel movements may cease, leading to constipation, although intermittent diarrhoea can occur in some cases [8]. These symptoms reflect a pseudo-obstructive pattern, mimicking true mechanical obstruction without a physical blockage [8].

On physical examination, abdominal distension and tympany are common findings [9]. Bowel sounds may range from hypoactive to normal, while hyperactive or high-pitched sounds are less typical [4,9]. Abdominal tenderness is usually diffuse and mild, although peritoneal signs such as rebound tenderness or guarding may indicate bowel ischaemia or perforation [10]. Laboratory findings often include mild leukocytosis, hypokalaemia, and other electrolyte imbalances [10]. Major complications include bowel ischaemia, caecal necrosis, and perforation, which are more likely if the caecal diameter exceeds 12 cm or if symptoms persist beyond 5-6 days [6,8]. Clinical features and complications of ACPO are detailed in [Table/Fig-1].

Category	Clinical findings
Onset of symptoms	Typically develops over 3 to 5 days, but may present suddenly within 48 hours
Abdominal symptoms	Progressive distension, most marked in the caecum and right colon
Gastrointestinal symptoms	Nausea and vomiting in some patients, colicky or diffuse abdominal pain in most
Bowel habits	Constipation is common due to halted peristalsis; some may experience intermittent diarrhoea
Obstruction pattern	Mimics mechanical obstruction, but without any physical blockage
Physical examination	Marked abdominal distension, tympany on percussion
Bowel sounds	Usually hypoactive to normal; hyperactive or high-pitched sounds are less typical
Tenderness and peritoneal signs	Diffuse mild tenderness; rebound tenderness or guarding may suggest ischaemia or perforation
Laboratory findings	Mild leukocytosis, hypokalaemia, and other electrolyte imbalances
Major complications	Bowel ischaemia, caecal wall necrosis, and perforation
Indicators of complications	Caecal diameter exceeding 12 centimeters or symptoms persisting longer than 5 to 6 days

[Table/Fig-1]: Clinical features and complications of ACPO.

Pathogenesis Mechanism of ACPO

The most widely accepted pathophysiological mechanism of ACPO is autonomic dysregulation, primarily an imbalance between sympathetic and parasympathetic innervation of the colon [2,9]. Parasympathetic stimulation increases colonic motility, whereas sympathetic stimulation inhibits it [11]. In ACPO, there is increased sympathetic activity with impaired parasympathetic input, particularly in the left colon, leading to colonic atony and massive

dilation without mechanical obstruction [11]. This imbalance may result from medical or surgical conditions, trauma, or metabolic disturbances [11]. The positive response to parasympathomimetic agents such as neostigmine further supports this theory [2,11].

In addition to autonomic imbalance, some reports highlight defective neuromuscular transmission in the colonic wall [12]. Histopathological examinations in a few cases of ACPO show degeneration of enteric neurons, reduced Interstitial Cells of Cajal (ICCs), and altered smooth muscle cell function [12]. These findings suggest that primary myogenic or neurogenic defects contribute to the pathogenesis, particularly in chronic or recurrent cases [13,14]. Such abnormalities impair the activity of colonic pacemakers and peristalsis, predisposing the colon to pseudo-obstruction when exposed to systemic conditions like sepsis, electrolyte imbalances, or medications [13].

Several drugs, including opioids, anticholinergics, calcium channel blockers, and psychotropic medications, can also impair colonic motility and lead to ACPO [15,16]. These drugs act on neural or smooth muscle pathways, reducing peristalsis and promoting colonic dilation [16]. Additionally, electrolyte disturbances such as hypokalaemia, hypocalcaemia, and hypomagnesaemia are commonly observed in patients with ACPO. These abnormalities may act synergistically with other risk factors to impair muscular and neuronal function of the colon [17]. Correction of these metabolic derangements often leads to clinical improvement, highlighting their contributory role in ACPO pathogenesis [17,18].

The ACPO is also frequently observed in patients with Spinal Cord Injuries (SCI) and central nervous system disorders, suggesting a link between disrupted neurological pathways and colonic motility dysfunction [19,20]. SCI can interrupt parasympathetic outflow from the sacral region and induce reflex sympathetic overactivity, further reducing colonic propulsion [13,21]. This mechanism is supported by the high incidence of ACPO in paraplegic and quadriplegic patients, particularly in the early post-injury period [21,22].

Systemic inflammation and cytokine release in critically ill patients can impair gut motility. Inflammatory mediators such as Tumour Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6), and nitric oxide disrupt the neural circuitry of the enteric nervous system and reduce smooth muscle contractility [23,24]. Overproduction of nitric oxide via Inducible Nitric Oxide Synthase (iNOS) has been shown in experimental models to cause colonic hypomotility [25]. Therefore, in patients with sepsis, surgery, or trauma, inflammatory cascades may acutely impair colonic motility and contribute to the development of ACPO [23].

Disruption of local enteric reflex arcs, such as inhibitory and excitatory motor neurons within the myenteric plexus, can result in uncoordinated or absent peristalsis, causing functional obstruction [26,27]. This dysfunction may be secondary to ischaemia, trauma, or inflammation affecting the intrinsic enteric nervous system [28]. Experimental animal models demonstrate that disrupting neural inputs to specific colonic segments reproduces some features of pseudo-obstruction, supporting the local neurogenic theory [27]. Various pathophysiological mechanisms of ACPO are summarised in [Table/Fig-2].

Mechanism	Description	Supporting evidence
Autonomic dysregulation	Imbalance between increased sympathetic activity and impaired parasympathetic input, especially affecting the left colon, leading to colonic atony and massive dilation.	Common in postoperative states, trauma, and metabolic disorders; positive response to neostigmine supports this mechanism.
Neuromuscular transmission defect	Histological findings show enteric neuronal degeneration, reduced ICCs, and abnormal smooth muscle function.	Seen in chronic/recurrent cases; results in impaired peristalsis and pacemaker activity.
Drug-induced motility impairment	Drugs like opioids, anticholinergics, calcium channel blockers, and psychotropic agents reduce colonic peristalsis.	These act on smooth muscle and neural pathways, promoting colonic dilation.
Electrolyte disturbances	Hypokalaemia, hypocalcaemia, and hypomagnesaemia impair neuronal and muscular function	Correction of electrolytes often results in clinical improvement
Neurological disorders and Spinal Cord Injury (SCI)	Disruption of sacral parasympathetic outflow and reflex sympathetic overactivity reduce propulsion	High incidence of ACPO in SCI patients, particularly in early post-injury phase
Systemic inflammation and cytokine release	TNF-α, IL-6, and nitric oxide interfere with enteric neurons and smooth muscle	iNOS-derived nitric oxide shown in experimental models to impair motility. Common in sepsis, trauma, and surgery
Disrupted enteric reflex arcs	Damage to excitatory/inhibitory neurons in the myenteric plexus leads to uncoordinated peristalsis	Evidence from animal models supports localised neurogenic dysfunction

[Table/Fig-2]: Various pathophysiological mechanisms of ACPO.
Legend: ACPO: Acute colonic pseudo-obstruction; ICCs: Interstitial cells of cajal; TNF: α - Tumour necrosis factor-alpha; IL-6: Interleukin-6; iNOS: Inducible nitric oxide synthase; SCI: Spinal cord injury.

Emerging Diagnostic Methods for ACPO

The diagnosis of ACPO is primarily clinical and radiological. Plain abdominal radiography can reveal significant colonic distension, with a caecal diameter above 10-12 cm raising concern for imminent perforation [29,30]. CT is the most sensitive modality, as it not only confirms colonic dilation but also helps rule out mechanical causes such as volvulus or tumours and allows further assessment for complications, including ischaemia or perforation [13,31]. Contrast enemas, though less commonly used now, can also help demonstrate the absence of an obstructing lesion [6]. Laboratory findings often reveal electrolyte imbalances and signs of systemic illness, which further contribute to colonic dysmotility.

Recent advancements in the diagnostic approach to ACPO have focused on more refined, functional, and less invasive modalities. Contrast-Enhanced CT (CECT) and CT colonography have improved accuracy in differentiating pseudo-obstruction from true mechanical obstruction while detecting early ischaemia [32,33]. These techniques provide high-resolution images of colonic wall perfusion and luminal integrity, aiding risk identification and guiding therapy [32].

High-resolution colonic manometry has emerged as a tool to assess colonic neuromuscular function in suspected ACPO cases [34]. Colonic manometry provides insight into underlying dysmotility patterns, helping distinguish ACPO from Chronic Intestinal Pseudo-Obstruction (CIPO) and improving understanding of pathophysiological mechanisms [34].

Furthermore, POCUS is a rapid, bedside technique for evaluating colonic dilation in critically ill patients, offering dynamic assessment without radiation exposure [35,36]. When combined with traditional imaging, these evolving diagnostic methods allow comprehensive and earlier identification of ACPO, thereby improving outcomes through timely intervention [35]. Diagnostic approaches for ACPO are summarised in [Table/Fig-3].

Stepwise Management of ACPO

Management of ACPO follows a stepwise approach, progressing from conservative therapy to pharmacologic, endoscopic, and surgical interventions based on clinical features and the risk of complications [35,37].

Supportive conservative therapy is the initial course of management and includes cessation of oral intake, nasogastric decompression, and placement of a rectal tube to relieve colonic distension [37]. Intravenous fluid resuscitation is crucial to restore electrolyte balance, particularly correcting hypokalaemia and hypomagnesaemia, which are known to impair colonic motility [29,35]. Identification and withdrawal of precipitating factors, such

Modality	Description	Advantages	Limitations
Plain abdominal radiography	Shows significant colonic distension, especially with caecal diameter >10-12 cm.	Widely available; quick; first-line tool.	Low specificity; cannot differentiate mechanical vs. pseudo-obstruction.
Computed Tomography (CT)	Confirms colonic dilation; rules out volvulus or tumour; detects complications like ischaemia or perforation	High sensitivity; provides detailed anatomy; assesses for complications	Radiation exposure; requires contrast in some cases
Contrast enema	Delineates the colon and confirms absence of a mechanical lesion	Useful when CT is inconclusive or contraindicated	Less commonly used; risk of perforation; uncomfortable
Laboratory tests	Detects electrolyte imbalances and systemic signs of inflammation or sepsis	Helps identify contributing metabolic factors	Non-specific; adjunct to imaging
Contrast-enhanced CT/CT Colonography	Advanced imaging showing luminal integrity and wall perfusion; detects early ischaemia	Better diagnostic accuracy; guides management decisions	Requires advanced equipment; higher cost
High-resolution colonic manometry	Evaluates neuromuscular function of the colon; identifies dysmotility patterns	Distinguishes ACPO from CIPO; offers insights into pathophysiology	Limited availability; requires expertise; invasive
Point-of-Care Ultrasound (POCUS)	Bedside assessment of bowel dilation and peristalsis	Radiation-free; portable; dynamic evaluation; useful in unstable patients	Operator-dependent; limited in obese patients or gas-distended bowels

[Table/Fig-3]: Diagnostic approaches for Acute Colonic Pseudo-Obstruction (ACPO).
CIPO: Chronic intestinal pseudo-obstruction

as opioids, anticholinergic drugs, or infections, is essential, as ACPO often resolves after correction of these underlying causes [6]. In many patients, these measures alone are sufficient for resolution within 24-72 hours [25].

If conservative management fails within 48-72 hours and colonic dilation exceeds 10-12 cm, pharmacologic therapy is initiated for ACPO [38]. Neostigmine, a reversible acetylcholinesterase inhibitor, is the most extensively studied and effective agent. It is administered intravenously at 2-2.5 mg over 3-5 minutes under continuous cardiac monitoring and induces colonic decompression in most patients, often within 30 minutes [38]. Neostigmine enhances peristalsis by increasing the availability of acetylcholine at neuromuscular colonic junctions [38]. However, it is contraindicated in patients with suspected mechanical obstruction, ischaemia, or bradyarrhythmias due to the risk of severe bradycardia and hypotension [39]. Atropine should be available to counteract these adverse effects, and repeated low doses or continuous infusions may be effective in partial responders or patients with recurrent symptoms [40,41].

Colonoscopic decompression is a useful therapeutic option when pharmacologic therapy is ineffective or contraindicated [42]. This procedure allows direct visualisation and evacuation of colonic gas and contents, resulting in rapid symptomatic relief [42]. Placement of a decompression tube during the procedure helps prevent recurrence. However, colonoscopic decompression carries a risk of perforation, particularly in patients with severe colonic dilation or underlying ischaemia, and should therefore be performed by experienced endoscopists under close monitoring [43,44].

Surgical intervention is reserved for patients with complications such as bowel ischaemia, necrosis, or perforation, or when all other treatment options have failed [45]. Surgical options include segmental colectomy, cecostomy, or colostomy, depending on the location and severity of the colonic pathology [45,46]. Surgery carries significant risk, with high morbidity and mortality, especially in elderly or critically ill patients [46].

New therapeutic approaches, such as Percutaneous Endoscopic Cecostomy (PEC), are useful in patients with recurrent or refractory ACPO [47]. PEC offers a minimally invasive alternative to surgical cecostomy and has shown favourable outcomes in selected patients [47]. Additionally, new pharmacologic agents and neuromodulation techniques, including sacral nerve stimulation, are under investigation but remain experimental [48].

Future Directions in ACPO

Future research on ACPO should address several key aspects [49]. First, a standard definition and classification system are needed to enhance diagnostic accuracy and allow for comparative studies [49,50]. Second, further investigation of the underlying pathophysiology using advanced imaging and motility studies may provide deeper insight into the mechanisms of colonic dysfunction [49]. Third, the effectiveness and safety of various treatment modalities, including pharmacologic agents and endoscopic procedures, should be assessed through well-designed clinical trials [49]. Finally, predictive models should be developed to identify patients at high risk of complications such as ischaemia or perforation, enabling timely and appropriate intervention [50]. Addressing these aspects will improve understanding, management, and outcomes in ACPO [50].

CONCLUSION(S)

The ACPO is a life-threatening functional disorder characterised by colonic dilation without mechanical obstruction. Prompt diagnosis through clinical assessment and advanced imaging is crucial to prevent serious complications such as ischaemia and perforation. Management follows a stepwise approach, beginning with conservative strategies, progressing to pharmacologic agents such as neostigmine, and advancing to endoscopic or surgical interventions if necessary. Novel therapies, including PEC and neuromodulation, are valuable in recurrent cases. Timely diagnosis, correction of underlying factors, and individualised treatment are essential to improve patient outcomes and reduce morbidity and mortality associated with ACPO.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jul 25, 2025
- Manual Googling: Nov 15, 2025
- iThenticate Software: Nov 18, 2025 (3%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? NA
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Jul 11, 2025**
Date of Peer Review: **Oct 07, 2025**
Date of Acceptance: **Nov 20, 2025**
Date of Publishing: **Apr 01, 2026**