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# Acute Colonic Pseudo Obstruction

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## ABSTRACT

Acute colonic pseudo obstruction is an abdominal emergency seen in elderly hospitalised patients being treated for major orthopaedic or medical ailments. It has to be differentiated from mechanical obstruction. Identifying and treating medical and metabolic derangements is the first line of treatment. If the patient does not respond then prokinetic medications such as neostigmine should be administered. Colonoscopic decompression and finally surgical intervention are indicated in cases resistant to treatment. The morbidity and mortality associated with these modalities is quite high.

***Keywords:*** Acute colonic pseudo obstruction diagnosis management

## INTRODUCTION

Acute colonic pseudo obstruction (ACPO) is best described as acute colonic dilatation in the absence of intrinsic mechanical obstruction or any extrinsic inflammatory process. [1,2] It was described by Sir William Ogilvie in 1948. [3] Subsequently the term "Intestinal pseudo obstruction" was proposed by Dudley in 1958. [1] Inconsistency in definition and terminology has led to inconsistency in reporting and research on this topic. The incidence of ACPO is 100 cases per 100000 inpatient admissions. [1, 4] Colonic ischaemia followed by perforation is seen in 10-20% of patients with ACPO. Whereas the mortality associated with perforation is 45%. [1] The condition is complex with no uniform definitive pathological mechanisms seen.

### Basic structural physiology of colonic innervation:

The caecum is located in the right iliac fossa. The caecum and ascending colon are larger in diameter and have a thin wall as compared with rest of the colon. Maximum dilatation in ACPO is seen in the caecum. This is in conformity with Laplace's law. The intraluminal pressure needed to stretch the wall of the hollow viscus is inversely proportional to the diameter. Therefore the caecum with a larger diameter requires less pressure to increase in size and in wall tension. As the colonic wall becomes tensed the chances of ischaemia increases with longitudinal splitting of the colonic wall, herniation of the mucosa and eventually ischaemia and perforation. The colon has both sympathetic and parasympathetic innervation. Vagus nerve is the parasympathetic supply to the upper gastrointestinal tract up to the splenic flexure. The rest of the gut is supplied by sacral parasympathetic S2 to S5. The proximal colon has a rich sympathetic innervation. The lower six thoracic segments supply the sympathetic tone to the right colon whereas the lumbar segments 1-3 supply the left colon. The transition zone of innervation is the splenic flexure. Sympathetic stimulation results in inhibition of bowel motility and contraction of sphincters.

Normal colonic motor activity is regulated at various levels.

a. Colonic smooth muscle
b. Pacemaker activity generated by interstitial cells of Cajal (ICC).
c. Intrinsic control via the enteric nervous system (ENS).
d. Prevertebral and spinal reflex arcs.

e. Extrinsic modulation by the autonomic nervous system and the hormonal system.

**Aetiology:**

ACPO commonly affects elderly patients with co morbidities. These include a wide spectrum of surgical and medical diseases affecting multiple organ systems. (Table 1) There is a vast array of risk factors involved thereby supporting a multifactorial aetiology with a variety of pathways converging to a common end point of colonic motor activity.

***Possible causative mechanisms of ACPO:***

*Functional obstruction.*

Autonomic imbalance is the most acceptable theory for ACPO. Altered extrinsic regulation of colonic function by the sympathetic and parasympathetic nervous system is the most commonly suggested mechanism for ACPO. Excess of sympathetic and parasympathetic tone can lead to an atonic segment and functional obstruction. Majority of patients with ACPO have major illnesses which increase the systemic sympathetic tone contributing to autonomic imbalance at the level of the colon. Initial studies with guanethidine a sympatholytic followed by a parasympathomimetic drug triggers colonic high amplitude propagating sequences (HAPS). [5] This strongly supports the hypothesis of autonomic imbalance as the cause for ACPO.

*Colonic Reflex Arc:*

Several spinal and ganglionic reflex arcs are involved in regulating intestinal motor activity. Colo-colic inhibitory reflex is inhibition of proximal colonic motor activity in response to distal colonic distension. Conversely proximal distension causes a reduction in basal intraluminal pressure in the distal colon. Reflex arcs are mediated via afferent mechanoreceptors synapsing with adrenergic efferent neurons in the prevertebral ganglia and spinal cord. These reflexes provide a possible mechanism to explain how one region may potentiate disordered motility and distension of one colonic region may potentiate dilatation of other regions contributing to ACPO. [6] Some researchers have claimed therapeutic success of epidural anaesthesia and splanchnic nerve blocks in a few anecdotal case reports supporting this hypothesis. Whether it is due to disruption of the efferent limb of these reflex arcs or simply reduction in the extrinsic sympathetic supply of the colon continues to be a debatable issue. Therefore epidural anaesthesia has been implicated as both a cause and a therapy for ACPO.

*Intrinsic Colonic dysfunction:*

Interstitial cells of Cajal (ICC) are the pacemaker responsible for generating electrical slow waves, which are moderated by the ENS resulting in the rhythmic contractile activity of the intestine. [7] Permanent impairment of the ENS, ICC and myopathy characterizes many forms of chronic pseudo obstruction. Few studies demonstrated a reduction and degeneration of enteric ganglion cells in the resected colon specimens of pseudo obstruction patients. Whether the histological abnormalities represent a cause or effect on the colonic dilatation and pseudo obstruction is questionable.

Nitric oxide (NO) is an important inhibitory neurotransmitter released by the colonic enteric neurons and is implicated in colonic dilatation and dysfunction in toxic megacolon and colitis. [7] However there is no substantial evidence to support the role of NO. PEG (polyethylene glycol) an osmotic laxative reduces NO production and also reduces the relapse rates after decompression in ACPO.

*Chronic disease and pharmacologic factors:*

ACPO patients are elderly individuals with chronic cardiac, respiratory and neurologic diseases such as Parkinsonism. Chronic stress conditioning causes effects of chronic disease. It potentiates excitatory and inhibitory neurotransmitters potentially explaining this association.

Effects on ENS and extrinsic regulation are seen in patients suffering from diabetes,

Parkinson's disease and Alzheimer's disease. Effect on ENS and ICC degeneration with age explains the preponderance of elderly population. [7]

*Patients with chronic conditions on medications:*

These medications include anticholinergics, opiates, calcium channel blockers and psychotropic drugs. Antimotility agents inducing ACPO is a predictor of poor response to neostigmine. Management associated with ACPO modulate the autonomic nervous system. Clonidine and amitraz are alpha 2 adrenergic agonists. Alpha 2 adrenergic signalling reduces release of acetylcholine from enteric neurons resulting in a relative imbalance of sympathetic or parasympathetic supply consistent with the current theory regarding the pathophysiology of ACPO. [7, 8]

*Obstetric aetiology:*

The commonest operation leading to ACPO is caesarean section. However it can occur even after normal and instrumental vaginal delivery as well. Other causes are pre-eclampsia, multiple pregnancies, anti-partum haemorrhage, and placenta previa. Possible mechanisms of ACPO developing after caesarean section are compression of parasympathetic plexus by the gravid uterus or the fall back of the uterus into the pelvis following delivery causing mechanical obstruction at the recto sigmoid junction. Pregnant state causes increase in the progesterone and glucagon levels. Both diminish the tone of the large bowel and predispose to ACPO especially when combined with acute physiologic disturbances such as surgery, pre-eclampsia, peripartum sepsis or haemorrhage. Resting sympathetic outflow is increased in the third trimester even in women with normal blood pressure. Autonomic dysfunction and sympathetic over activity are features of pre-eclampsia. [1, 6]

*Metabolic factors:*

Disrupted homeostasis is common in ACPO. This can precipitate or exacerbate the effects of altered autonomic function. A variety of electrolyte disturbances accompany ACPO. These include alterations in Na and K ions. Electrolyte imbalances have been identified as a predictor of poor clinical response to neostigmine. Prostaglandin and cytokine release can cause acute small intestinal motility, altered ICC function and slow wave frequency. Increased expression of COX 2 is seen in distended colon of mice in experimental models. The effect is mediated through PGE2. However similar results are not seen in human studies. [9]

*Viral enteroneuropathy:*

ACPO is associated with viral infections. These include herpes zoster reactivation involving the lower thoracic and lumbar segments, disseminated varicella zoster, acute cytomegalovirus and severe dengue infections. The possible explanation is autonomic dysfunction. Herpes infection affects enteric ganglion thereby leading to sympathetic autonomic neuritis causing decrease in sympathetic activity. Local segmental colonic inflammation will cause stimulation of the sacral nerve roots leading to blockage of the colonic parasympathetic supply. Viral spread from the dorsal root ganglion to the thoracolumbar or sacral columns could interrupt the sacral parasympathetic pathways. Involvement and inflammation of the intrinsic enteric nervous system can also lead to post viral dysautonomia. [9]

Other possible hypothesis which have been proposed are compromise in the blood supply to the colon, " Hinge-Kinking" of the colon at the transition from retro to intraperitoneal junction, air fluid lock syndrome and colonic distension due to aerophagy in COPD patients.

Having reviewed all postulated theories it is evident that that the precise mechanism is still unclear. However autonomic imbalance seems to be the most accepted hypothesis for the aetiology of ACPO.

ACPO patients are systemically unwell or have lesions disrupting the autonomic supply of the colon. The transition point for distended to normal colon in ACPO occurs

at the splenic flexure corresponding to the change in autonomic innervation of the mid gut and hind gut.

**Clinical presentation:**

ACPO occurs in debilitated, hospitalised patients who have multiple medical problems associated with surgical conditions.

The onset of symptoms is insidious, usually 3-5 days postoperatively. Abdominal pain, nausea and vomiting are seen in 80% of patients. There is history of obstipation and accompanying fever. Vital parameters will be altered in a select few patients.

Physical examination will reveal rapidly developing abdominal distension and tenderness. If there is ischaemia or perforation patient will have rebound tenderness, guarding or even rigidity depending on the progression of peritonitis. [10] Bowel sounds will be absent or hypoactive high pitched in nature. Digital examination of the rectum will reveal an empty rectum.

**Table 1 Risk factors for acute colonic pseudo obstruction.**

| | |
|---|---|
| Surgical | Cardiac surgery, spine surgery, orthopaedic surgery |
| Neurological | Parkinson's disease, dementia, Alzheimer's disease |
| Metabolic | Electrolyte imbalance, diabetes, renal failure, hepatic failure |
| Medications | Opiates, anti-Parkinson drugs, anticholinergics, antipsychotics, clonidine |
| Gynaecologic | Caesarean section, instrumental delivery, pregnancy, pelvic surgery |
| Infections | Varicella zoster, herpes virus, cytomegalovirus |
| Cardiorespiratory | Shock, myocardial infarction, CCF, COPD |
| Other causes | Sepsis, major burns, trauma |

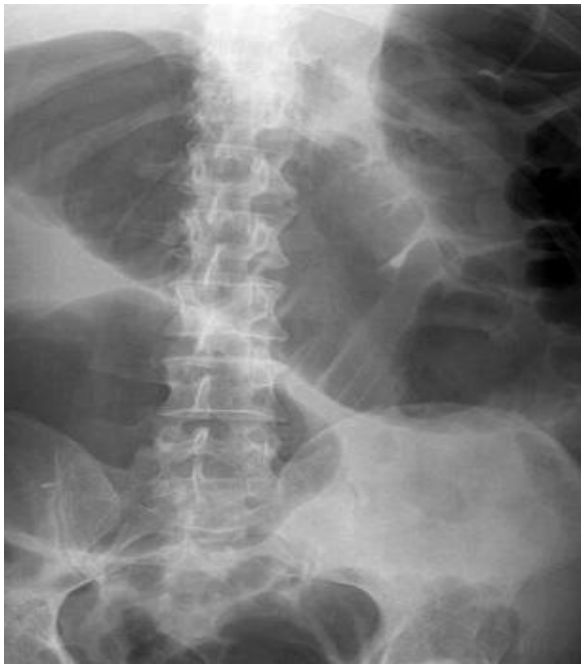

**Figure 1: Plain X ray abdomen showing distended colon**

**Investigations:**

Complete blood count and renal profile is necessary in all patients. Leucocytosis is seen in most of the patients. Serum electrolyte examination is necessary in all patients as most of the patients have altered levels. Renal insufficiency is also a common accompaniment in most patients. Correction of metabolic derangements is therapeutic in a few cases. Plain x ray of the abdomen is diagnostic. (Figure 1) The only issue is to rule out a mechanical cause for obstruction. Plain x ray can also help in this respect. The patient can be placed in right lateral decubitus position for several minutes. The gas will pass into the distal colon. Gaseous filling of the rectum can be facilitated by positioning the patient in prone lateral view of the pelvis. This progression of gas in the colon has a 75% success rate in excluding mechanical obstruction and gaseous filling which do not occur in patients with mechanical obstruction. [11, 12] A transverse diameter of the caecum exceeding 12 cms is worrisome and calls for more proactive or aggressive treatment. Abdominal CT scan will be necessary to exclude mechanical obstruction and toxic megacolon. Contrast enema has also been suggested. However perforation needs to be ruled out before considering this option. Meglumine diatrizoate a water soluble contrast is usually used. This has to be a low pressure enema where no air should be used. The examination is terminated when the dilated colon is reached. Hyperosmolarity results in fluid

shifts into the lumen which increases colonic motility thereby evacuation of air from the distended colon. [12]

Flexible colonoscopy is another diagnostic modality. It is helpful in differentiating pseudo obstruction from mechanical obstruction. It is therapeutic in achieving colonic decompression. If mechanical obstruction is diagnosed then it enables biopsy of the lesion. The only pitfall is that the visibility is poor as prior colonic preparation cannot be done before the procedure. [12]

**Treatment:**

The priorities in management include

1. Exclude mechanical obstruction.
2. Aggressive correction of metabolic derangements.
3. Optimization of medical co morbidities.
4. Discontinuation of all potential medications which interfere with motility of the colon. (Anti-parkinsonism medications, calcium channel blockers etc.)
5. Nasogastric tube insertion with continuous aspiration and drainage.
6. Correction of fluid and electrolyte depletion.

Quite a few number of patients will respond to this treatment.

If the response is poor then cholinesterase inhibitors is the next therapeutic option. Neostigmine is the drug of choice. [13]

Contraindications to the use of neostigmine are

Heart rate < 60 beats /minute.

Systolic BP < 90 mm of HG

Acute bronchospasm needing active treatment.

Neostigmine is administered in the dose of 2 to 2.5 mgs intravenously slowly over three minutes. The dose can repeated after 3 to 4 hours. If there is no response to two successive doses then colonoscopic decompression is attempted. It is effective in 85% of cases and can be repeated. It may be technically difficult due to an unprepared colon. The colon in ACPO is papery thin which increases the chances of perforation. Gas cannot be insufflated in ACPO.

The recurrence rate with colonic decompression is 18-65%. Placement of an indwelling colonic tube up to the level of the transverse colon can help in reducing the recurrence rate. Post procedure administration of PEG (polyethylene glycol) helps in reducing the recurrence rate. [11, 12]

In patients who were on opiate analgesia and have developed ACPO, methyl naltrexone a peripherally acting opioid antagonist is useful if neostigmine fails to relieve ACPO. Other promotility agents have been tried such as erythromycin, cisapride and metoclopromide. However the results with these medications are not promising. [12, 13]

If all conservative methods fail then surgery is indicated. [14, 15] A tube cecostomy may be performed. However the thinning and friability of the distended caecum can limit a successful outcome in such patients. The next surgical option is subtotal colectomy. However the morbidity and mortality with this procedure is high. [16]

## CONCLUSION

Acute colonic pseudo obstruction is rare condition seen in elderly hospitalised patients with an accompanying surgical or medical disease. Plain X ray abdomen is diagnostic. However mechanical obstruction needs to be ruled out by CT scanning.

Aggressive conservative treatment including supportive care with correction of metabolic derangements is the first line of treatment. A suboptimal response necessitates the use of other options which include neostigmine therapy, colonic decompression and eventually surgery.

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## REFERENCES


1. Coulie B, Camilleri M. Intestinal pseudo-obstruction. Annu Rev Med. 1999. 50:37-55.
2. Alwan MH, van Rij AM. Acute colonic pseudo-obstruction. Aust N Z J Surg. 1998 Feb. 68(2):129-32.
3. Ogilvie H. Large-intestine colic due to sympathetic deprivation; a new clinical syndrome. Br Med J. 1948 Oct 9. 2(4579):671-3.
4. Ohkubo H, Iida H, Takahashi H, et al. An epidemiologic survey of chronic intestinal pseudo-obstruction and evaluation of the newly proposed diagnostic criteria. Digestion. 2012. 86(1):12-9.
5. Saunders MD. Acute colonic pseudo-obstruction. Best Pract Res Clin Gastroenterol. 2007. 21(4):671-87.
6. De Giorgio R, Knowles CH. Acute colonic pseudo-obstruction. Br J Surg. 2009 Mar. 96(3):229-39.
7. Bachulis BL, Smith PE. Pseudoobstruction of the colon. Am J Surg. 1978 Jul. 136(1):66-72.
8. Lee JT, Taylor BM, Singleton BC. Epidural anesthesia for acute pseudo-obstruction of the colon (Ogilvie's syndrome). Dis Colon Rectum. 1988 Sep. 31(9):686-91.
9. Mashour GA, Peterfreund RA. Spinal anesthesia and Ogilvie's syndrome. J Clin Anesth. 2005 Mar. 17(2):122-3.
10. Spira IA, Wolff WI. Gangrene and spontaneous perforation of the cecum as a complication of pseudo-obstruction of the colon: report of three cases and speculation as to etiology. Dis Colon Rectum. 1976 Sep. 19(6):557-62.
11. Dorudi S, Berry AR, Kettlewell MG. Acute colonic pseudo-obstruction. Br J Surg. 1992 Feb. 79(2):99-103.
12. Vanek VW, Al-Salti M. Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. Dis Colon Rectum. 1986 Mar. 29(3):203-10.
13. Hutchinson R, Griffiths C. Acute colonic pseudo-obstruction: a pharmacological approach. Ann R Coll Surg Engl. 1992 Sep. 74(5):364-7.
14. Shi XZ, Lin YM, Powell DW, Sarna SK. Pathophysiology of motility dysfunction in bowel obstruction: role of stretch-induced COX-2. Am J Physiol Gastrointest Liver Physiol. 2011 Jan.300(1):G99-G108.
15. Clarke HD, Berry DJ, Larson DR. Acute pseudo-obstruction of the colon as a postoperative complication of hip arthroplasty. J Bone Joint Surg Am. 1997 Nov. 79(11):1642-7.
16. Nanni G, Garbini A, Luchetti P, Nanni G, Ronconi P, Castagneto M. Ogilvie's syndrome (acute colonic pseudo-obstruction): review of the literature (October 1948 to March 1980) and report of four additional cases. Dis Colon Rectum. 1982 Mar. 25(2):157-66.


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