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EDITORIAL

- 5451 Wilson's disease: Prospective developments towards new therapies

Ranucci G, Polishchuck R, Iorio R

- 5457 Are our endoscopy patients at risk for pyogenic liver abscess?

Cerwenka H, Schemmer P

REVIEW

- 5460 Pancreatitis: Preventing catastrophic haemorrhage

Evans RPT, Mourad MM, Pall G, Fisher SG, Bramhall SR

MINIREVIEWS

- 5469 Treating children with inflammatory bowel disease: Current and new perspectives

Guariso G, Gasparetto M

- 5486 Influence of gut microbiota on neuropsychiatric disorders

Cenit MC, Sanz Y, Codoñer-Franch P

ORIGINAL ARTICLE

Basic Study

- 5499 Everolimus halts hepatic cystogenesis in a rodent model of polycystic-liver-disease

Temmerman F, Chen F, Libbrecht L, Vander Elst I, Windmolders P, Feng Y, Ni Y, De Smedt H, Nevens F, van Pelt J

- 5508 MicroRNA profile in neosquamous esophageal mucosa following ablation of Barrett's esophagus

Sreedharan L, Mayne GC, Watson DI, Bright T, Lord RV, Ansar A, Wang T, Kist J, Astill DS, Hussey DJ

- 5519 Expression of Interleukin-26 is upregulated in inflammatory bowel disease

Fujii M, Nishida A, Imaeda H, Ohno M, Nishino K, Sakai S, Inatomi O, Bamba S, Kawahara M, Shimizu T, Andoh A

- 5530 Autophagic cell death induced by reactive oxygen species is involved in hyperthermic sensitization to ionizing radiation in human hepatocellular carcinoma cells

Yuan GJ, Deng JJ, Cao DD, Shi L, Chen X, Lei JJ, Xu XM

- 5538 Yangzheng Sanjie decoction regulates proliferation and apoptosis of gastric cancer cells by enhancing let-7a expression

Deng HX, Yu YY, Zhou AQ, Zhu JL, Luo LN, Chen WQ, Hu L, Chen GX

Case Control Study

- 5549 Crohn's disease environmental factors in the developing world: A case-control study in a statewide catchment area in Brazil

Salgado VCL, Luiz RR, Boechat N, Schorr BC, Leão IS, Nunes T, Zaltman C

Retrospective Cohort Study

- 5557 Postoperative bleeding in patients on antithrombotic therapy after gastric endoscopic submucosal dissection

Sato C, Hirasawa K, Koh R, Ikeda R, Fukuchi T, Kobayashi R, Kaneko H, Makazu M, Maeda S

Retrospective Study

- 5567 Serous pancreatic neoplasia, data and review

Dietrich CF, Dong Y, Jenssen C, Ciaravino V, Hocke M, Wang WP, Burmester E, Möller K, Atkinson NSS, Capelli P, D'Onofrio M

- 5579 Pancreaticoduodenectomy for duodenal papilla carcinoma: A single-centre 9-year retrospective study of 112 patients with long-term follow-up

Lian PL, Chang Y, Xu XC, Zhao Z, Wang XQ, Xu KS

Clinical Trials Study

- 5589 Efficacy and safety of Xiangsha Liujunzi granules for functional dyspepsia: A multi-center randomized double-blind placebo-controlled clinical study

Lv L, Wang FY, Ma XX, Li ZH, Huang SP, Shi ZH, Ji HJ, Bian LQ, Zhang BH, Chen T, Yin XL, Tang XD

Observational Study

- 5602 Combination of acoustic radiation force impulse imaging, serological indexes and contrast-enhanced ultrasound for diagnosis of liver lesions

Sun XL, Yao H, Men Q, Hou KZ, Chen Z, Xu CQ, Liang LW

Prospective Study

- 5610 Incidents and adverse events of endoscopic ultrasound-guided fine-needle aspiration for pancreatic cystic lesions

Du C, Chai NL, Linghu EQ, Li HK, Sun YF, Xu W, Wang XD, Tang P, Yang J

SYSTEMATIC REVIEWS

- 5619 Systematic review of giant gastric lipomas reported since 1980 and report of two new cases in a review of 117110 esophagogastroduodenoscopies

Cappell MS, Stevens CE, Amin M

- 5634 Acute colonic pseudo-obstruction: A systematic review of aetiology and mechanisms

Wells CI, O'Grady G, Bissett IP

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Acute colonic pseudo-obstruction: A systematic review of aetiology and mechanisms

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Abstract

AIM

To critically review the literature addressing the definition, epidemiology, aetiology and pathophysiology of acute colonic pseudo-obstruction (ACPO).

METHODS

A systematic search was performed to identify articles investigating the aetiology and pathophysiology of ACPO. A narrative synthesis of the evidence was undertaken.

RESULTS

No consistent approach to the definition or reporting of ACPO has been developed, which has led to overlapping investigation with other conditions. A vast array of risk factors has been identified, supporting a multifactorial aetiology. The pathophysiological mechanisms remain unclear, but are likely related to altered autonomic regulation of colonic motility, in the setting of other predisposing factors.

CONCLUSION

Future research should aim to establish a clear and consistent definition of ACPO, and elucidate the pathophysiological mechanisms leading to altered colonic function. An improved understanding of the aetiology of ACPO may facilitate the development of targeted strategies for its prevention and treatment.

Key words: Acute colonic pseudoobstruction; Acute colonic pseudo-obstruction; Colonic; Intestinal; Pseudo obstruction; Ogilvie's syndrome; Pseudo-Obstruction; Pseudo-Obstruction

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Core tip: Acute colonic pseudo-obstruction is associated with considerable morbidity and mortality, though the underlying pathophysiology remains poorly understood.

An abundance of risk factors and associated conditions have been identified, strongly suggesting a multifactorial origin, and likely culminating in an imbalance in autonomic nervous supply to the colon. Future areas for research are identified and may allow for the development of novel therapeutic or preventative strategies.

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INTRODUCTION

Acute colonic pseudo-obstruction (ACPO) is a rare condition characterised by acute colonic dilatation in the absence of mechanical obstruction. It was first described by Sir William Ogilvie in 1948, in two patients with malignant infiltration of the pre-vertebral ganglia^[1].

ACPO usually occurs in hospitalised patients with severe illness or trauma, or following general, orthopaedic, neurosurgical, gynaecological or other surgical procedures, with an estimated incidence of 100 cases per 100000 admissions and a mortality rate of 8%^[2,3]. Colonic ischaemia or perforation occurs in up to 15%, and is associated with an estimated 40% mortality^[4-6]. Therefore, early recognition and appropriate therapy are important determinants of prognosis^[2].

The pathophysiology underlying ACPO is poorly understood, with the prevailing hypothesis being an imbalance in colonic autonomic innervation^[2,4]. Progress has been limited by a rudimentary understanding of the complex processes regulating colonic motility patterns, the lack of specific animal models for ACPO, and the poor quality of evidence arising from case reports and uncontrolled case series.

An improved understanding of the mechanisms underlying ACPO may aid the development of novel management strategies for this condition. Therefore, the aim of this systematic review was to critically review the literature addressing the definition, epidemiology, risk factors, aetiology and pathophysiology of ACPO with a view to informing knowledge gaps and identifying priority areas for future research.

MATERIALS AND METHODS

A systematic search of the Ovid MEDLINE and Embase databases was performed from inception to 25 May 2017, using the search terms "Ogilvie's syndrome", "pseudoobstruction", and "pseudo obstruction". Google Scholar was also searched using free text entries. Identified articles were screened by title and

abstract for inclusion, with subsequent acquisition of full texts. The reference lists of included papers were manually searched, and a hand search of the scientific literature was performed to identify additional relevant publications.

Non-English articles were excluded. There were no exclusion limits by study design; both primary research and review articles were eligible. Full-text articles were evaluated for evidence addressing the definition, epidemiology, risk factors, aetiology and pathogenesis of ACPO. Clinical management has recently been reviewed in depth^[7-9], and was beyond the scope of this study, though pathophysiological hypotheses drawn from this evidence were evaluated. A narrative synthesis of the identified evidence was undertaken.

RESULTS

Definitions of ACPO

ACPO is characterised by acute colonic dilatation in the absence of intrinsic mechanical obstruction or extrinsic inflammatory process^[10]. ACPO should be distinguished from other acute conditions, such as toxic megacolon, which may have inflammatory or infective causes, as well as chronic intestinal pseudo-obstruction (CIPO) and other causes of megacolon.

Many overlapping terms have been used in the literature to describe ACPO since its original description, reflecting the uncertainty regarding its precise aetiology (Table 1). The term "intestinal pseudo-obstruction" was first proposed by Dudley in 1958^[11], with "ACPO" not appearing for another twenty years^[4]. Use of "Ogilvie's syndrome" has been discouraged due to ambiguity regarding its meaning^[12], though considerable heterogeneity in terminology still exists in recent literature^[3,13,14].

Inconsistent terminology has contributed to inconsistency in research and reporting. No standardised clinical definition or diagnostic criteria for ACPO were identified^[15], preventing reliable synthesis of data on risk factors or therapeutic strategies^[7,8]. Some studies failed to distinguish ACPO from CIPO and other forms of megacolon^[10,16], despite these being distinct entities with different mechanisms and therapies^[17,18].

Epidemiology

ACPO is uncommon, with an identified incidence of approximately 100 cases per 100000 inpatient admissions^[3]. A recent US study reported a declining mortality rate associated with ACPO from 9.4% in 1998 to 6.4% in 2011, although over-diagnosis may have contributed^[3]; historically mortality rates were as high as 30%^[4]. Colonic perforation and ischemia occur in 10%-20%, with an associated mortality of up to 45%^[2,4]. Rates of surgical and endoscopic intervention have declined in recent decades, due to an increasing focus on conservative management and pharmacological therapy with neostigmine^[3].

ACPO disproportionately affects elderly and

Table 1 Terms found used to describe acute colonic pseudo-obstruction

Term	Ref.
Large intestine colic	[1]
Ogilvie's syndrome	[4, 153, 155-157]
Pseudo-megacolon	[158]
Adynamic ileus	[159, 160]
Paralytic ileus	[14, 142]
Functional obstruction of the intestinal tract	[161]
Idiopathic large bowel obstruction	[154]
Colonic ileus	[160, 162-164]
Intestinal pseudo-obstruction	[11, 111, 146, 165, 166]
Non-mechanical large bowel obstruction	[167]
Pseudo-obstruction of the large bowel, pseudo-obstruction of the colon	[23, 86, 140, 141, 168, 169]
Acute colonic pseudo-obstruction	[4]
Non-obstructive colonic dilatation	[170, 171]
Malignant ileus	[144]
Cecal ileus	[164]
Acute megacolon	[81, 172]

Table 2 Prevalent medical and surgical risk factors for acute colonic pseudo-obstruction

Category	Risk factors
Surgical	Cardiac surgery, solid organ transplantation, major orthopaedic surgery, spine surgery
Cardiorespiratory	Shock, myocardial infarction, congestive heart failure, chronic obstructive pulmonary disease
Neurological	Dementia, Parkinson's disease, Alzheimer's disease, stroke, spinal cord injury
Metabolic	Electrolyte imbalance, diabetes, renal failure, hepatic failure
Medications	Opiates, anti-Parkinson agents, anticholinergics, antipsychotics, cytotoxic chemotherapy, clonidine
Obstetric/gynaecological	Caesarean section, normal vaginal delivery, instrumental delivery, preeclampsia, normal pregnancy, pelvic surgery
Infectious	Varicella-zoster virus, herpes virus, cytomegalovirus
Miscellaneous	Major burns/trauma, severe sepsis, idiopathic

comorbid patients; often those with an acute illness on a background of chronic cardiac, respiratory or neurological disease. ACPO also occurs following general, orthopaedic, neurosurgical, cardiothoracic and gynaecological procedures^[19]. Vanek *et al.*^[2] reported 400 cases of ACPO, with approximately 50% resulting from acute medical illness and 50% from post-surgical patients. Incidence rates have been described in spinal or orthopaedic surgery (1%-2%)^[13,20], cardiac bypass (up to 5%)^[21], and following burn injuries (0.3%)^[22]. An abundance of other conditions associated with ACPO were found to have been reported in the literature, the most common of which are summarised (Table 2).

Pathophysiology

Review of the literature revealed a vast array of risk factors for ACPO, supporting a multifactorial aetiology with several pathways leading to a common effect

on colonic motor function^[23] (Figure 1). However, few published studies were found directly investigating the pathophysiological mechanisms underlying ACPO, with the majority of evidence inferred from basic physiology and other disease states such as post-operative ileus (POI). No animal models of ACPO were identified.

Normal colonic motor activity is regulated at several levels; (1) colonic smooth muscle; (2) pacemaker activity generated by interstitial cells of Cajal (ICC); (3) intrinsic control *via* the enteric nervous system (ENS); (4) prevertebral and spinal reflex arcs; and (5) extrinsic modulation by the autonomic nervous system and hormonal systems. Being a "functional obstruction", ACPO is presumed to result from aberrations in colonic motor activity, though physiological data specifically characterising these abnormalities was not identified.

Autonomic imbalance

Altered extrinsic regulation of colonic function by the sympathetic and parasympathetic nervous systems is the most commonly suggested mechanism for ACPO^[10,24-29]. This mechanism was first postulated by Ogilvie, who proposed a "sympathetic deprivation" of the colon^[1]. Current theory favours a relative excess of sympathetic over parasympathetic tone, though it is unclear whether this is due to increased sympathetic activity, reduced parasympathetic signalling, both, or either. The prevailing hypothesis remains that ACPO is the result of reduced parasympathetic innervation to the distal colon, leading to an atonic segment and functional obstruction^[2,10,27,28,30-32]; however no published physiological data was found to directly support or refute this claim^[28,29].

The colon receives sympathetic innervation from the prevertebral ganglia (proximally *via* the superior mesenteric ganglion; distally *via* the inferior mesenteric ganglion). Nerve fibres follow the respective arterial routes^[33], and the proximal colon appears to have a richer sympathetic innervation^[23]. The parasympathetic supply to the mid-gut is *via* the vagus nerve while the hind-gut is innervated by sacral parasympathetic outflow (S2-4)^[33], although animal studies have demonstrated some overlap between these distributions^[34]. The transition point from distended to normal colon in ACPO usually occurs near the splenic flexure^[2,10,35], where there is a transition in the innervation for both sympathetic and parasympathetic supply to the colon, supporting autonomic imbalance as a key step in pathogenesis.

Most patients with ACPO are unwell with major illness, therefore having increased systemic sympathetic drive^[23,36,37], potentially contributing to autonomic imbalance at the level of the colon. Other reported cases have involved pathology potentially disrupting the autonomic supply to the colon, such as retroperitoneal tumours or haemorrhage^[38,39]. Interestingly, ACPO cannot be reproduced in humans or animals by splanchnic or pelvic nerve transection^[40,41], thus the pathogenesis appears more complex than excess or

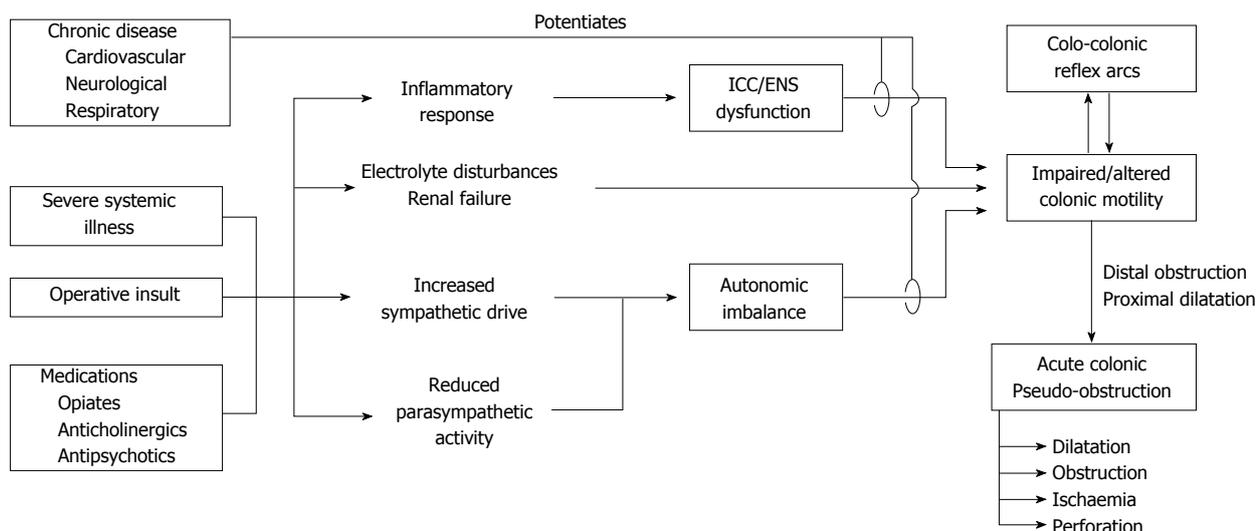


Figure 1 Pathophysiological factors that have been implicated in the development of acute colonic pseudo-obstruction. ICC: Interstitial cells of Cajal; ENS: Enteric nervous system.

deficiency of sympathetic or parasympathetic activity alone.

Neostigmine is an acetylcholinesterase inhibitor and parasympathomimetic commonly used in management of ACPO, further supporting autonomic imbalance as a key pathophysiological mechanism. Early studies investigated use of guanethidine, a sympatholytic, followed by neostigmine^[42]. Guanethidine was later shown ineffective^[43], while neostigmine has been proven efficacious in several randomised trials^[7,44-46]. Prior studies have shown that parasympathetic stimulation with neostigmine triggers colonic high amplitude propagating sequences (HAPS) in both dogs and humans^[47,48], which has been proposed as a possible mechanism for its decompressive effect in the functionally obstructed colon^[49]. However, HAPS primarily arise in the proximal colon, and it is unknown what effect neostigmine has on other prominent colonic motor activity such as cyclic events, or the role of these events in ACPO^[50].

Colonic reflex arcs

Several spinal and ganglionic reflex arcs are involved in regulating intestinal motor function. The colo-colonic inhibitory reflex refers to inhibition of proximal colonic motor activity in response to distal colonic distention^[51,52]. Conversely, proximal distension also causes a reduction in basal intraluminal pressure in the distal colon^[51]. Evidence from animal models suggests these reflex arcs are mediated *via* afferent mechanoreceptors synapsing with adrenergic efferent neurons in the prevertebral ganglia and spinal cord^[18,51,52].

These reflexes provide a possible mechanism to explain how disordered motility and distension of one colonic region may potentiate dilatation of other regions, contributing to ACPO^[18,26,28,30]. Some authors have pointed to the therapeutic success of epidural anaesthesia and splanchnic nerve block in

case reports as evidence for this mechanism^[10,53], though it is difficult to ascertain whether this is due to disruption of the efferent limb of these reflex arcs, or simply reduction in the extrinsic sympathetic supply to the colon. Furthermore, epidural anaesthesia has been implicated as both a cause of and a therapy for ACPO^[18,53,54]. The lack of high-quality evidence identified in this area makes it difficult to determine the effects of spinal or ganglionic blockade in ACPO and the precise role of the colo-colonic inhibitory reflex.

Intrinsic colonic dysfunction

ICC are the pacemaker responsible for generating electrical slow waves, which are modulated by the ENS, resulting in the rhythmic contractile activity of the intestine^[55,56]. Despite their recognised importance in the control of gastrointestinal motility, few studies were identified that specifically investigated the roles of enteric neurons or ICC in ACPO.

Permanent impairment of the ENS, ICC, and/or myopathy characterises many forms of CIPO^[24,57-59]. However, the acute onset, reversibility, and different epidemiology of ACPO implies a distinct pathophysiological process from CIPO, hence these findings should not be extrapolated to the acute form of pseudo-obstruction.

Jain *et al*^[57] investigated ICC using immunohistochemistry in patients with pseudo-obstruction syndromes, showing two patients with ACPO had a normal number and distribution of ICC. However, whether ICC function is affected in ACPO remains unknown. Choi *et al*^[35] reported a reduction and degeneration of enteric ganglionic cells in the resected colonic specimens of four patients with pseudo-obstruction, but it is unclear whether these patients had ACPO or CIPO, and whether these histological abnormalities represent cause or effect of the colonic

dilatation and pseudo-obstruction. Without further pathophysiological studies, the role of the ENS and ICC in the development of ACPO remains unknown.

Nitric oxide (NO) is an important inhibitory neurotransmitter released by colonic enteric neurons, and has been implicated in colonic dilatation and dysfunction in toxic megacolon^[37,60,61] and colitis^[62]. Several authors have speculated whether NO may also have a role in ACPO, however no physiological evidence was found to support this claim. However, polyethylene glycol (PEG), an osmotic laxative that may also reduce NO production^[63,64], has been shown to reduce relapse rates after decompression in ACPO^[65]. This finding could be explained by either the laxative effects of PEG or its effect on nitrinergic signalling. A number of novel pharmacological treatments targeting NO regulation have recently been discovered, though no studies were identified that applied these therapies in patients with ACPO^[66].

Chronic disease and pharmacological factors

Notably, many ACPO patients are elderly and have chronic cardiac, respiratory or neurological disease, with onset often occurring in the context of a further acute physiological insult^[2,3]. Chronic stress conditioning, similar to the effects of chronic disease, potentiates excitatory and inhibitory neurotransmission^[67], potentially explaining these associations. The ENS and its extrinsic regulation are affected in several conditions commonly associated with ACPO, including diabetes mellitus^[68,69], Parkinson's^[70,71] and Alzheimer's disease^[70,72,73]. Furthermore, both the ENS and ICC have been shown to degenerate with age^[74-76], which may partly account for the elderly preponderance of this disorder^[2,3,29,77].

Patients with chronic conditions are also more likely to be on multiple medications affecting colonic motility^[78,79], and may therefore be predisposed to the development of ACPO. Numerous medications have been associated with ACPO, including anticholinergics, opiates, calcium channel blockers, and psychotropic drugs^[80-82]. Furthermore, use of anti-motility agents is predictive of poor response to neostigmine^[83], while methylnaltrexone has been successfully used in a patient with ACPO who failed to respond to two doses of neostigmine^[84].

Many medications associated with ACPO modulate the autonomic nervous system, supporting disruption of these pathways as a key pathophysiological mechanism. Clonidine^[18,85-87] and amitraz^[88] are α_2 -adrenergic agonists that have been associated with ACPO. At the level of the colon, α_2 -adrenergic signalling reduces release of acetylcholine from enteric neurons, resulting in a relative imbalance of sympathetic over parasympathetic supply^[89,90], consistent with current theory regarding the pathophysiology of ACPO.

Obstetric causes

The operation most commonly resulting in ACPO is

caesarean section^[2,26,91-99], but ACPO also occurs after normal and instrumental vaginal delivery^[100-102]. A recent systematic review identified that preeclampsia, multiple pregnancy, and antepartum haemorrhage/placenta praevia appear to be more common in women who develop ACPO following caesarean section^[103]. However, it remains unclear how these events result in acutely perturbed colonic function. Some authors have proposed that compression of parasympathetic plexuses by the gravid uterus may contribute^[26,27,104], while others have hypothesised that the uterus may fall back into the pelvis following delivery, causing a mechanical obstruction at the rectosigmoid colon^[36,105].

Pregnancy is also associated with high levels of progesterone and glucagon, both of which have been shown to diminish the tone of the large bowel^[92], and may predispose to ACPO when combined with acute physiological disturbances such as caesarean section, preeclampsia, peripartum sepsis, or haemorrhage. Resting sympathetic vasomotor outflow is increased during the third trimester, even in women with normal blood pressure^[106,107], while autonomic dysfunction and sympathetic overactivity are a feature of severe preeclampsia^[108,109]. It is unknown whether sympathetic supply to the colon is also affected in these states, but this could plausibly contribute to autonomic imbalance and ACPO. Finally, prostaglandins are intimately involved in parturition^[110], and have also been suggested as a possible contributor to ACPO in obstetric patients^[92] (*discussed in next section*).

Metabolic factors

A disrupted "*milieu interieur*" is common in ACPO, which may precipitate or exacerbate the effects of altered autonomic function or other mechanisms. A host of metabolic factors have been shown to influence colonic motility, though few studies have investigated these specifically in ACPO. However, the isolated colonic dysfunction characterising ACPO makes it unlikely that metabolic abnormalities are the sole pathophysiological explanation.

Renal failure and electrolyte disturbances often accompany ACPO, although it has been disputed whether this is a cause or effect of the pseudo-obstruction^[4,16,32,39,83,111]. Alterations in potassium and other electrolyte concentrations affect ion channel function and may alter ICC pacemaker or smooth muscle activity^[112,113]. Furthermore, electrolyte imbalance has been identified as a predictor of a poor clinical response to neostigmine^[83].

Prostaglandins and cytokines are both well-established inflammatory mediators of gastrointestinal motility^[114,115]. Pro-inflammatory cytokines such as TNF- α , IL-6, IL-8, and IL-1 β have implicated in POI^[114,115], but have not yet been investigated in ACPO. Prostaglandins have been implicated in POI^[115,116], CIPO^[117,118], acute small intestinal dysmotility^[119], and also affect ICC function and slow wave frequency^[120]. However, no studies were identified investigating the

role of prostaglandins in the development of ACPO. Cyclooxygenase-2 (COX-2) expression is increased in the distended colon of mice with experimentally-induced mechanical obstruction, and it has been suggested that this is responsible for reduced motility *via* the effects of prostaglandin E₂^[121]. However, caution must be taken when extrapolating these results to humans and to ACPO^[122].

Viral enteroneuropathy

ACPO has also been associated with several viral infections, most commonly herpes zoster reactivation in low thoracic or lumbar distributions^[123-130], but also disseminated varicella zoster^[131-134], acute cytomegalovirus^[135], and severe dengue^[136].

Several mechanisms have been proposed to explain these associations, all involving autonomic dysfunction. Reactivation of herpes virus in enteric ganglia may result in a sympathetic autonomic neuritis^[123,124,137,138], presumably diminishing colonic sympathetic supply. Local segmental colonic inflammation with afferent stimuli to the sacral nerve roots and blockage of colonic parasympathetic supply has also been suggested^[138], while other authors have hypothesised that viral spread from the dorsal root ganglia to the thoracolumbar or sacral lateral columns may interrupt sacral parasympathetic pathways^[137]. Involvement and inflammation of the intrinsic enteric nervous system, and post-viral dysautonomia have also been suggested^[136,137,139].

Other hypotheses

Several alternate hypotheses regarding the pathophysiology of ACPO were identified. Compromised vascular supply to the colon was suggested in many early reports^[11,140,141], but now thought to represent an exacerbating complication rather than a cause. "Hinge-kinking" of the colon at the transition from retro- to intra-peritoneal structures has also been proposed^[142], however, intra-operative and radiological findings generally suggest a "gradual" transition in colonic calibre^[23]. Other identified hypotheses lacking supporting evidence included the "air-fluid lock syndrome"^[143-145], and colonic distension due to aerophagy in chronic respiratory disease^[146,147].

DISCUSSION

Despite considerable speculation, this comprehensive and systematic review of the literature finds that the mechanisms underpinning ACPO still remain uncertain. Limited physiological evidence from patients with ACPO was found to exist, and the lack of an animal model has required extrapolation of data from other disease states. The abundance of identified risk factors and associated conditions supports a multifactorial hypothesis, with aberrations in function at any of several levels seemingly able to lead to common or similar effects on the colon.

Overall, autonomic imbalance is the most strongly supported pathophysiological contributor, and likely plays a key role in the development of ACPO. Evidence supporting a major role for autonomic imbalance includes the observation that most patients with ACPO are systemically unwell, or have other pathology disrupting the autonomic supply to the colon. Furthermore, the transition point from distended to normal colon in ACPO usually occurs at the splenic flexure, corresponding to the change in autonomic innervation of the mid-gut and hind-gut. However, the precise aberrations in autonomic function leading to ACPO remain unclear.

ACPO is difficult to rigorously investigate due to its acute and sporadic nature and heterogeneity. The plethora of case reports identified in the literature is testament to the large number of illnesses associated with this condition. It is also clear that a categorical clinical definition of ACPO is needed to standardise research, and improve the quality of published evidence. Distinct subtypes of ACPO have recently been proposed according to gut wall thickness on cross-sectional imaging^[148], and further work should investigate the aetiology and prognostic significance of this phenomenon.

Importantly, the precise aberrations in colonic motor activity underlying ACPO have not been characterised. Despite the common assumption that the distal colon is atonic, direct physiological evidence supporting this claim could not be identified, and it remains unknown whether ACPO is fundamentally due to atony, dysmotility, or spasm in part or all of the colon, or is due to heterogeneous motility states. Furthermore, although many authors have argued that the sympathetic system decreases colonic motility and the parasympathetic increases contractility^[10,18,28,30,43], this is now recognised as overly simplistic, with further layers of modulation within the ENS, ICC and smooth muscle^[27,33]. It is also important to note that the therapeutic success of neostigmine also does not specifically prove interrupted parasympathetic supply to the colon as the sole pathophysiological mechanism underlying ACPO^[29], despite this being suggested by many authors^[2,42,43,149,150]. Indeed, provocation of HAPS and colonic emptying by neostigmine may be sufficient to overcome an alternative pathophysiological mechanism and result in decompression of the functionally obstructed colon.

High-resolution colonic manometry has recently been used to characterise the abnormalities underlying colonic dysmotility in the early post-operative period, demonstrating increased cyclic motor activity in the left colon, rather than atony, and an absence of HAPS^[151,152]. Applying similar research methods to ACPO would be a particularly interesting research avenue, with potential to rigorously define the altered motility patterns underlying the disorder. It is interesting to note that transient "ring[s] of spasm" were noted intra-operatively in the distal colon of several of the original

descriptions of this syndrome^[1,153,154], though the pathophysiological significance of these remains unclear. Other areas for future pathophysiological investigation include the development of an animal model of ACPO, histological examination of resected specimens, and rigorous investigation of the impact of preventative or therapeutic strategies on colonic function.

This systematic review and appraisal of the literature surrounding ACPO has identified limited evidence investigating the mechanisms underlying this condition. A number of areas for further study have been identified. It is hoped that an improved understanding of the pathophysiology of ACPO will lead to the development of targeted strategies for its prevention and treatment.

COMMENTS

Background

Acute colonic pseudo-obstruction (ACPO) is an uncommon condition, characterised by acute colonic dilatation in the absence of a mechanical obstruction. Colonic ischaemia or perforation may occur in up to 15% of patients, and is associated with considerable morbidity and mortality.

Research frontiers

The pathophysiology underlying ACPO is poorly understood, with a plethora of associated conditions reported in the literature. The prevailing hypothesis is an imbalance in colonic autonomic innervation, though few articles have thoroughly examined the evidence supporting this claim.

Innovations and breakthroughs

This systematic review finds that the mechanisms underpinning ACPO still remain uncertain. Autonomic imbalance is the most strongly supported pathophysiological factor, though the precise aberrations leading to ACPO are unclear. A number of other factors may contribute to ACPO, including colonic reflex arcs, intrinsic colonic dysfunction, chronic disease, and pharmacological or metabolic disturbances.

Applications

A number of areas for future investigation have been identified, including the use of high-resolution colonic manometry to rigorously define the altered motility patterns underlying ACPO, the development of an animal model, and histological examination of resected specimens. An improved understanding of the pathophysiology of ACPO may lead to the development of novel preventative or therapeutic strategies.

Terminology

Pseudo-obstruction is characterised by a clinical presentation suggestive of intestinal obstruction, in the absence of a mechanically obstructing lesion. ACPO is the acute form of colonic pseudo-obstruction, while chronic intestinal pseudo-obstruction is a distinct entity with different mechanisms and therapies. "Ogilvie's syndrome" has also been used to refer to ACPO, though this term is now discouraged due to ambiguity regarding its meaning.

Peer-review

This is a very interesting review regarding the etiology of acute colonic pseudo-obstruction. The author introduced a number of articles regarding this issue.

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