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Acute Colonic Pseudo-Obstruction (ACPO): An Expanding Colon with Unusual Risk Factors

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Abstract

The distention of the colon without mechanical or anatomical obstruction, Acute Colonic Pseudo-obstruction (ACPO), is a common condition occurring in the critically ill. ACPO in the setting of an acute pulmonary embolism and embolic stroke is a rarity. A 76-year-old female with shortness of breath, left hemiparesis and right-sided paresthesias presented with acute pulmonary embolism and acute infarcts of the left caudate nucleus, thalamus and occipital lobe. Her hospitalization was complicated with persistent distention of the large bowel without dilation of the small bowel. Empiric antibiotics were initiated without improvement and laboratory studies including *Clostridium difficile* were negative. She underwent nasogastric decompression and two decompressive colonoscopies with a resolution of her symptoms. This case illustrates an example of acute abdominal distension, without underlying etiology, in the setting of acute embolism of the pulmonary and cerebral vasculature. Early identification and action with decompressive colonoscopy were key to preventing further bowel damage or rupture.

Keywords

Acute Colonic Pseudo-Obstruction, ACPO, Ogilvie's Syndrome, Colonic Dilation, Acute Embolic Infarcts, Cerebrovascular Accident, Pulmonary Embolism, PE

1. Introduction

Acute colonic pseudo-obstruction (ACPO), or Ogilvie's syndrome, is distension of the colon without mechanical or anatomical obstruction [1]. It was first described by Sir William Heneage Ogilvie in 1948 in two patients with non-obstructive colonic dilation from retroperitoneal malignancy of the celiac plexus [2]. This condition now commonly occurs in older, debilitated patients with comorbidities

such as recent surgery, trauma, sepsis, electrolyte derangements, medications (eg. opioids and anticholinergics), and immobility [3]. ACPO is a rare but often fatal condition. A 2016 retrospective and cohort study analyzed 106,784 cases of ACPO from 1998 to 2011, each of which was managed with medical management alone, colonoscopy alone, surgery alone, or colonoscopy and surgery [4]. Logistic regression was used to determine predictors of adverse outcomes by treatment group, noting that increasing procedural invasiveness was associated with higher odds of medical complication, procedural complication, and mortality [4]. ACPO after a cerebrovascular accident is very rare with only a few cases reported in the literature and only one other case reported after a pulmonary embolism [5] [6]. We herein present a single case study of ACPO in the setting of both acute pulmonary embolism and acute embolic stroke of the left caudate, thalamus, and occipital lobes. Informed consent was obtained from the patient for publication of this case report and accompanying images.

2. Case Presentation

A 76 year old female with a history of pulmonary embolism, diabetes mellitus, right sided breast cancer, obstructive sleep apnea, and obesity (BMI of 47) presented with a 4 day history of progressive shortness of breath, generalized malaise, fatigue, numbness on the right side, and focal weakness on the left side. She had a prior pulmonary embolism 21 months before presentation and was treated with six months of anticoagulation with apixaban. Computed tomography (CT) showed acute pulmonary embolism and signs of right heart strain. Her initial pulmonary embolism severity index (PESI) score was 126, indicating a higher risk of mortality and further complications. Echocardiogram revealed an ejection fraction of 65% and normal global left ventricular function. Magnetic resonance imaging (MRI) of the brain revealed small acute infarcts in the left caudate nucleus, left thalamus, and left occipital lobe (**Figure 1**). Lower extremity non-invasive vascular assessment revealed an occlusive right posterior tibial vein deep venous thrombosis. Patient was admitted to the intensive care unit for close monitoring, started on a heparin infusion and later transitioned to apixaban.

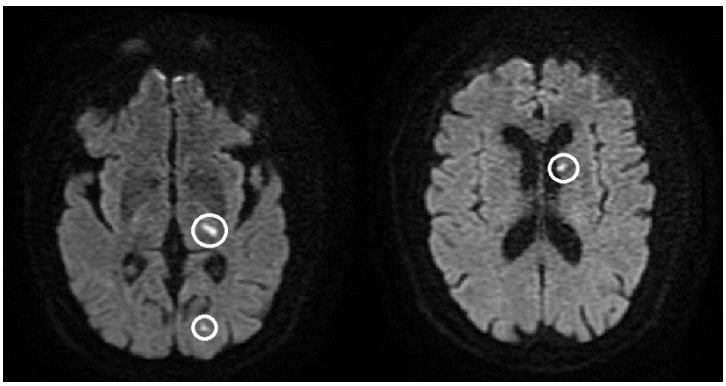

Figure 1. MRI brain with two infarcts (circled) in left image and one infarct (circled) in right image.

During her hospitalization, the patient developed intermittent encephalopathy and an uptrending white blood cell count. Ceftriaxone was started for empiric coverage. Urine culture was positive for *Escherichia coli.* Eleven days after admission, the patient had a persistently elevated white blood cell count and ongoing encephalopathy. CT scan of the head showed evolving subacute infarct of the left thalamus. CT scan of the chest with contrast demonstrated no acute findings, however generalized gaseous distention was noted. Antibiotics were therefore broadened to cover a possible intra-abdominal source of infection. Serial abdominal x-rays (Figure 2) showed persistent distension of the large bowel and CT showed maximum dilation of cecum of 12.5 cm in diameter without small bowel dilation, suggestive of colonic pseudo-obstruction (Figure 3 and Figure 4).

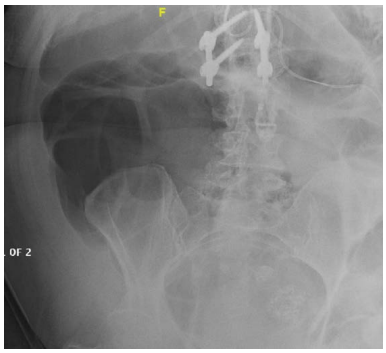

Figure 2. Upright abdominal x-ray with notable dilation of the cecum.

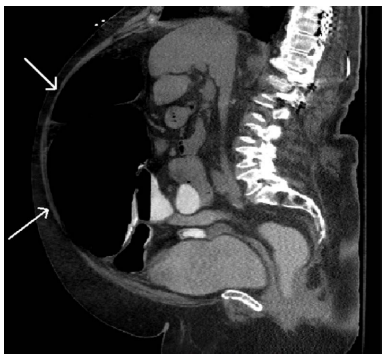

Figure 3. Abdominal CT scan sagittal view of colon distension (arrows) with cecum diameter of 12.5 cm.

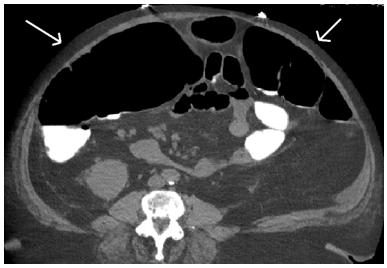

Figure 4. Abdominal CT scan axial image with gaseous distension of cecum and colon (arrows). Contrast is seen in colon.

Lab results were significant for persistent hypokalemia (3.3 mmol/L), persistent leukocytosis (>15 × 10^9/L), acute kidney injury (creatinine 1.5 mg/dL), elevated C-reactive protein (17.4 mg/dL), normal lactate level (1 mmol/L), and negative *C. difficile* toxins. Physical examination was notable for progressively worsening abdominal distension without tenderness to palpation. She had no nausea or vomiting and continued to pass flatus with small bowel movements.

Given her persistently dilated cecum with concerns for perforation, the patient underwent an emergent decompressive colonoscopy. General surgery was consulted, but was deemed as a poor surgical candidate given her comorbidities and recommended conservative measures first. A nasogastric tube was placed for additional decompression, in addition to a nil per os (NPO) diet and electrolyte replacement. Metoclopramide and bisacodyl suppositories were also administered to promote gut motility. She was not prescribed any opioid or anticholinergic medications during her hospitalization. Her abdominal distension resolved after a second decompressive colonoscopy.

3. Discussion

Acute colonic pseudo-obstruction (ACPO) can acutely affect patients with respiratory, cerebral, cardiovascular, or chronic renal diseases [1]. Patients will typically present with sudden onset and progressive abdominal distension, discomfort, nausea and vomiting with or without altered bowel function [7]. The abdomen will typically be tympanic and non-tender on presentation [1].

While the pathophysiology has not yet fully been elucidated, there is believed to be a etiology stemming from an imbalance of the enteric parasympathetic and sympathetic nervous systems. Both decreased parasympathetic activity and increased sympathetic activity contribute to the colon distension from the dysmotility primarily in the cecum and ascending colon [2] [8]. The sympathetic-vagal balance is also altered from an acute ischemic stroke by elevating sympathetic activity while suppressing the parasympathetic activity [9]. The wall tension will increase relatively to the colon diameter. Such increases the risk of complications of ischemia, gut translocation of bacteria, and perforation [7]. Perforation has the propensity to occur whenever the colon diameter is greater than 12 cm for longer than 6 days [7]. Bacterial gut translocation also presents the opportunity for patients to be more susceptible to sepsis and its complications in addition to the risk of ischemia and perforation [10].

ACPO can be diagnosed with imaging such as an upright abdominal x-ray and the preferred modality of abdominal computed tomography with contrast [2]. This can help exclude mechanical causes of obstruction. Colonic distension can be identified with a diameter length of >9 cm on radiographic imaging [3]. Initial management includes addressing the reversible causes and early bowel decompression. Reversible causes include pharmacologics that affect gut motility (opiates, anticholinergics, antidepressants, antipsychotic among others), electrolyte derangements, immobility, infections, and surgery [2] [3]. Treatment op-

tions include conservative management followed by pharmacological, endoscopic and/or surgical interventions (Figure 5).

Conservative management includes bowel rest with nil per os (NPO) and intravenous hydration, correcting electrolytes, discontinuing contributing medications, early ambulation while addressing the underlying cause [7]. Conservative therapy can be continued up to 72 hours if there is no progression of disease and no concern for perforation [2] [7]. If conservative therapy fails, pharmacological, endoscopic, and surgical interventions are available. Prokinetic agents including metoclopramide and erythromycin revealed inconsistent disease response and thus further reviews supported against the use of prokinetic agents for ACPO [11]. The preferred pharmacological intervention is neostigmine, an acetylcholinesterase inhibitor, which stimulates the parasympathetic nervous system and promotes colonic contractility [12]. Endoscopic intervention includes colonic decompression without a bowel preparation or gas insufflation to provide immediate relief and reduce subsequent complications. Success rates reported as 61% - 95% after initial decompression and further procedural decompressions improving success rates to 73% - 88% [13]. Complications of endoscopic colonic decompression include iatrogenicperforation. However, if conservative interventions fail to resolve the obstruction, surgical intervention may be warranted.

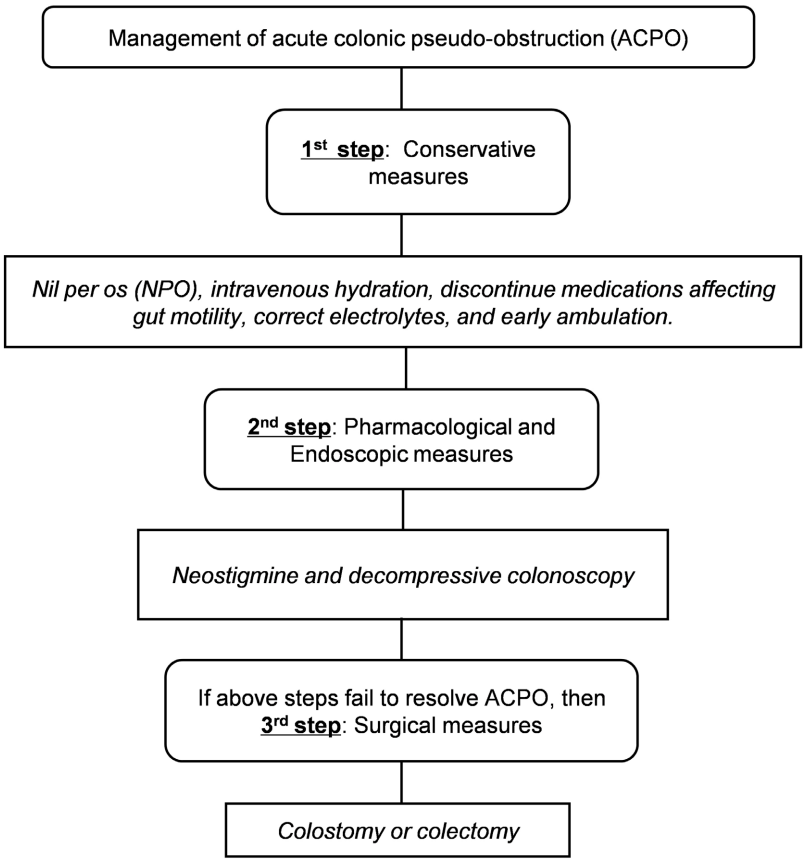

Figure 5. Flowchart of management of ACPO.

Surgical interventions include colostomy or colectomy [3]. Authors Kling and Goldstein note in their case series that colonic decompression may be a more appropriate initial treatment in cases of Ogilvie's caused by strokes [8]. This was also pursued with our patient at the recommendations of both general surgery and gastroenterology. Neostigmine was not considered like the other case studies with strokes given the urgent potential of colon perforation [5] [8]. Our patient thus underwent colonic decompression as the initial treatment due to the large colon diameter. Patient had tolerated the procedure well without complications with subsequent resolution of this clinical presentation.

Compared to the Polish case report of Ogilvie's, our patient did not have to undergo a decompressive stoma as her obstruction did not recur after the decompressive colonoscopies [6]. Furthermore, the patient was deemed as a poor surgical candidate given her comorbidities. However, there is currently no standardized treatment for ACPO. The exact recurrence rate of ACPO remains unknown. One study found a recurrence rate of 50% with prokinetic agents like erythromycin [11]. A recurrence rate of up to 40% after colonic decompression was reported [14]. Author O'Dea found success with long-acting acetylcholinesterase inhibitor, pyridostigmine for treatment of recurrent ACPO, however further studies are needed [15].

4. Conclusions

We present a rare case of acute colonic pseudo-obstruction (ACPO), also known as Ogilvie's Syndrome, precipitated by autonomic dysfunction in the setting of acute embolic stroke, as well as cardiac strain secondary to acute pulmonary embolism. ACPO should be suspected in patients with new onset abdominal distention, nausea and vomiting, with or without altered bowel function, in which abdominal imaging is without obvious signs of mechanical obstruction [7]. Other differential diagnoses to consider include small bowel obstruction, toxic megacolon, and mechanical obstruction in the setting of recent surgery, recent trauma, electrolyte imbalance, medications, infection, stroke or cardiac strain. Reversible causes for altered gut motility should be assessed as well.

The primary goal of therapy for ACPO is colonic decompression [3]. Early recognition of APCO is imperative to prevent complications such as perforation and bowel ischemia. It is recommended to consult gastroenterology and surgery early for better outcomes. Conservative management includes bowel rest with NPO and intravenous hydration, as well as pharmacological therapy, primarily neostigmine [2]. If conservative therapy fails, decompression can be achieved endoscopically. Surgical intervention with colectomy or colostomy can be considered as a last resort.

Disclaimer

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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