Journal of Comprehensive Clinical Practice
An official Journal of Vivekananda Polyclinic and Institute of Medical Sciences, Lucknow, UP, India

CASE REPORT

Acute Colonic Pseudo-obstruction (Ogilvies Syndrome) in Thalamic Hemorrhage

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Abstract

Ogilvies syndrome, also known as acute colonic pseudo-obstruction (ACPO), is a severe side effect of numerous neurological conditions, notably parkinsonism, dementia stoke, and spinal cord injury. A 55 years old man on the third day of left thalamic hemorrhage developed ACPO. He had severe pain, abdominal distension, and reduced bowel sounds. His abdominal radiograph revealed air fluid levels and computerized tomography dilated bowel loops. He was treated with intravenous fluids and nasogastric suction but because of worsening condition on day nine, he was prescribed inj neostigmine 2 mg IM. He responded by passing large amount of flatus within one hour of injection. His serum Catecholamine levels declined on day 15 (Serum dopamine on day 5 and 15 was 92.5 and 19.8 µg/L, epinephrine 47.8 and 19.8 µg/L and nor epinephrine 64.2 and 32.2 µg/L, respectively) suggesting the role of increased sympathetic activity in ACPO.

ARTICLE INFO

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Dates:
Published: 30-12-2023

Keywords:
Acute colonic pseudo obstruction, Neurological disorders, Ogilvie s syndrome

Conflict of Interest:
The authors have no conflict of interest

How to Cite:
Misra UK, Sarkar GG, Upadhyaya V, Bhatt A, Tripathi A, Thakur RK. Acute colonic pseudo-obstruction (Ogilvies syndrome) in thalamic hemorrhage. Journal of Comprehensive Clinical Practice. 2023;17(2):42-44

Introduction

The first record of acute colonic pseudo-obstruction (ACPO), reported two cases of colonic pseudo-obstruction being linked to malignant infiltration of the celiac plexus, which resulted in sympathetic denervation.[1] Stroke is commonly associated with constipation but rarely leads to ACPO. A rare case of ACPO in a patient with hypertensive thalamic intra-cerebral hemorrhage is reported and its pathogenesis and management is discussed.

Case Report

The male patient, aged 55, had a history of impaired sensorium. A few hours following a family feud, the patient was found in an unconscious state in his room and rushed to the hospital. On admission he was confused, talking incoherently there was no focal weakness or cranial nerve palsy. His biceps, triceps, knee, and ankle reflexes were normal. He was not able to cooperate for sensation and coordination testing.

His white cell count was 7700/mm^3, platelets 2.50000/mm^3, serum creatinine 0.9 mg/dL, urea 21 mg/dL, sodium 132 mEq/L, potassium 4.92 mEq/L, bilirubin

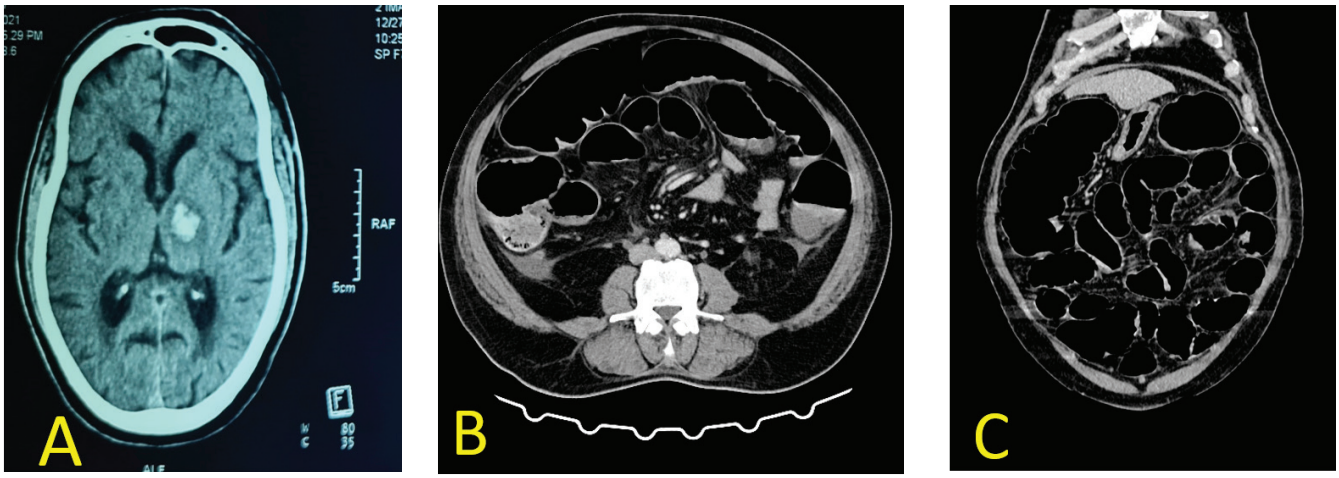

Figure 1: Showing a 55 year old man with acute colonic pseudo obstruction on day 5 of thalamic haemorrhage A)) Non-contrast enhanced CT scan of brain axial section showing left thalamic haemorrhage B) Axial contrast enhanced CT image of the abdomen showing dilated bowel loops with air-fluid levels and ascites C) Coronal reformatted contrast enhanced CT image of the abdomen showing multiple dilated bowel loops.

0.23 mg/dL, SGPT 75U/L, electrocardiogram was normal. Computerized tomography (CT) of the brain revealed left thalamic hemorrhage (2.0 x 4.2 cm) (Figure 1A). On day three he developed pain and distension in the abdomen. Bowel sounds were feeble and there was no tenderness or free fluid. On day five abdominal distension persisted (abdominal girth 46"). He was put on nasogastric decompression and IV fluid. An erect radiograph of the abdomen revealed multiple air-fluid levels. Dilated small and large bowel loops, measuring up to 7 cm, were observed on the abdominal CT scan (Figures 1B, C), as mild ascites and right pleural effusion. On day nine he was drowsier, and abdominal distension increased. Because of their worsening clinical condition, he was prescribed 2 mg neostigmine IM, and within 1-hour, he passed a large amount of flatus and his abdominal distension decreased (41.7"). Because of improvement, colonoscopic decompression was deferred and he was allowed oral feeding. The patient improved by day 10 and was discharged on day 19. His serum catecholamine levels decrease as he improved, dopamine on day 5 and 15 were 92.5 and 19.8 µg/L, epinephrine 47.8 and 19.8 µg/L and norepinephrine 64.2 and 32.2 µg/L, respectively

Discussion

The patient developed ACPO on the third day of thalamic hemorrhage which responded to supportive treatment and neostigmine on ninth day. Several neurological disorders such as Parkinson's disease, dementia, spinal cord injury and stroke can result in ACPO.[2] In a review of 15 patients with intracranial disease with ACPO, there were two patients with subarachnoid hemorrhage, five with intra-cerebral hemorrhage, six with ischemic stroke, and two with cerebral contusion. The predictors of ACPO are a history of abdominal surgery, sepsis, pneumonia, myocardial infarction, rib fracture, and peritoneal infection.[3] Nifedipine is an effective antihypertensive agent commonly used to control BP in stroke. The property of smooth muscle relaxation affects not only the intracranial vessels but also the gastrointestinal tract and may contribute to ACPO.[4]

It has been suggested that a difference between sympathetic and parasympathetic activity causes APCO and gut motility is increased by parasympathetic activity and reduced by sympathetic activity (Barret 2005). A functional blockage and decreased tone in the proximal colon (adynamic colon) may result from increased sympathetic tone and decreased parasympathetic activity.[6] In our study dopamine, epinephrine, and norepinephrine were elevated during ACPO and declined on recovery suggesting its possible role in the pathogenesis of ACPO.

The ACPO syndrome has also been linked to the inhibition of GI hormone, which promotes colon

motility when it is affected by neurohypophysis. Because of this mechanism of action, somatostatin and octretide are being used to treat ACPO. Timely recognition and regular monitoring are essential to prevent complications of ACPO such as ischemia, perforation, and peritonitis which may require surgical intervention. In uncomplicated cases, conservative management includes clinical monitoring including abdominal girth charting, abdominal radiography and 12 to 24 hourly laboratory evaluation. Worsening or lack of improvement for 72 hours should lead to reconsideration of the management plan.[7] Persistence of ACPO after six days is associated with high risk of perforation. The conservative management includes nil orally, nasogastric decompression, fluid and electrolyte resuscitation, antibiotics for suspected infection, and removal of offending drugs (nifedipine, calcium channel blocker, osmotic laxative). A review of 1027 patients with ACPO revealed that 70% of patients responded to conservative treatment, complications occurred in 6% and mortality of 10%.[8] The patients, refractory to conservative treatment may be treated by colonoscopic decompression. Neostigmine has been used in ACPO, it increases the level of acetylcholine at the synapse and increases colonic motility. In a randomized controlled trial comparing 2 mg neostigmine in 3 to 5 min IV versus standard treatment, 10 out of 11 (91%) responded compared to none in standard treatment arm.[9] Hypotension, bradycardia, asystole, convulsions, bronchospasm, salivation, vomiting, and increased GI motility, which can cause cramping in the abdomen and diarrhea, are among the side effects of IV neostigmine. Atropine should be used to address severe adverse effects, such as bradycardia, in patients receiving neostigmine medication. Patients should also be constantly watched throughout this time. Pyridotigmine 10–30 mg twice a day showed positive results in all seven of the patients who had previously failed neostigmine and endoscopic decompression treatment. Comparing pyridostigmine to neostigmine, pyridostigmine has less severe side effects.[10] Colonic decompression is successful in 61–95%.[2] We have used intramuscular neostigmine, and was effective with 60 minutes and may be tried before more invasive techniques.

References

1. Laine L (1999) Management of acute colonic pseudo-obstruction. N Engl J Med 341(3): 192-193.
2. Jain A, Vargas HD. Advances and challenges in the management of acute colonic pseudo-obstruction (ogilvie syndrome). Clin Colon Rectal Surg. 2012 Mar;25(1):37-45. doi: 10.1055/s-0032-1301758. PMID: 23449274; PMCID: PMC3348732.
3. Imai K Paralytic ileus associated with intracranial disease. Surgery for cerebral stroke 2001, 29:104-108
4. Karaki S, Weis GB. Calcium Channels in smooth muscles Gastroenterology 87:960-970, 1984
5. Barrett KE. Intestinal motility. In: Barrett KE, ed. Gastrointestinal Physiology. New York, NY: McGraw-Hill; 2006:154–158
6. Durai R. Colonic pseudo-obstruction. Singapore Med J 2009;50 (3):237–244
7. De Giorgio R, Knowles CH. Acute colonic pseudo-obstruction. Br J Surg 2009;96(3):229–239
8. Wegener M, Börsch G. Acute colonic pseudo-obstruction (Ogilvie's syndrome). Presentation of 14 of our own cases and analysis of 1027 cases reported in the literature. Surg Endosc 1987;1(3): 169–174
9. Ponec RJ, Saunders MD, Kimmey MB. Neostigmine for the treatment of acute colonic pseudo-obstruction. N Engl J Med 1999;341 (3):137–141
10. O'Dea CJ, Brookes JH, Wattchow DA. The efficacy of treatment of patients with severe constipation or recurrent pseudo-obstruction with pyridostigmine. Colorectal Dis 2010;12(6): 540–548