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# ACUTE COLONIC PSEUDO OBSTRUCTION (OGILVIE Syndrome) in a patient with hemorrhagic Stroke: A case report.

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#### **ABSTRACT:**

Acute colonic pseudo-obstruction (ACPO) or Ogilvie syndrome is a functional motility disorder of the colonic bowel without any organic or mechanical obstruction. Autonomic disturbance which is one of the underlying factors that can lead to Ogilvie syndrome, is caused by stroke. Acute colonic pseudo- obstruction (ACPO) in the setting of stroke is not often reported in medical literature. Various modalities of treatment strategies are recommended including conservative approaches, medical therapies, endoscopic and surgical procedures. We will discuss a patient suffering from a hemorrhagic stroke in the right basal ganglia that developed Acute Colonic Pseudo Obstruction and improved subsequently with conservative management.

#### **Case report:**

We received a 61 year old male, who was a known diabetic and hypertensive for the last 4 years. He arrived at the emergency department with complaint of sudden onset weakness on the left side of the body for the last 12 hours. The patient had previously suffered from hemorrhagic stroke in 2009. On examination he had a pulse of 77/min, blood pressure of 180/120 mmhg, respiratory rate of 20/min and oxygen saturations of 97%. On neurological examination, tone was reduced on left side with power 3/5 on left upper and lower limbs (MRS scale) and positive left Babinski sign. Baseline laboratory tests were ordered. A CT scan was ordered which showed a hematoma in the right basal ganglia (figure A). The patient was admitted to the acute stroke unit, and kept under strict observation. During his stay in the stroke unit, the patient had severe hypertension; for which he received injectable labetalol and Irbesartan. After spending two days in the acute stroke unit, he had symptomatic improvement in power on left side. His blood pressure was controlled by increasing the dose of injectable labetalol and then he was shifted to the ward on the 3rd day of admission. The patient had not passed any stools in the stroke unit, but his abdomen was soft and bowel sounds were present. Another significant issue during his stay in the acute stroke unit was persistent hypokalemia despite replacement with injectable potassium chloride. and Despite receiving oral potassium I/V supplementation, the patient had a potassium level of 3.2mmol/l. On the 4thday of admission, the patient started complaining of epigastric discomfort, bloating and headache. On examination, the abdomen was soft and bowel sounds were present. The only abnormality was high blood pressure of 180/100 which was still being managed via injectable labetalol. On the 5thday, blood pressure was well controlled but the patient developed intractable hiccups. On examination, the abdomen was still soft and non-tender with normal bowel sounds. On day 6, patient started vomiting a greenish fluid and a nasogastric tube was inserted for drainage. Patient was shifted to the high dependency unit. Abdominal examination showed mild epigastric distension, bowel sounds were sluggish on auscultation. Despite receiving regular oral and injectable potassium supplementation, the patient continued to remain hypokalemic with current potassium level of 3.1mmol/L. Opinion was taken from the surgical team, and they advised a CT abdomen to assess the state of the bowels. The CT abdomen showed distended gut loops, but no signs of

obstruction or impending perforation. The surgical team advised conservative management and he received laxatives and enema, in addition to the routine medications. Over the course of the next three days of admission there was marked improvement in abdominal distention with NG tube output decreased from 100, 70 and down to 50 ml on day 9. Furthermore, the patient was able to pass soft stools and bowel sounds became more regular. However, following these improvements, on the 9th day, the patient became febrile and developed significant tachypnea. Physical examination and investigations done suggested a hospital acquired pneumonia, for which he was started on tazobactam-piperacillin and shifted back to the stroke unit. His anti-biotic was changed to meropenem due to a lack of a clinical response to tazobactam-piperacillin. On day 12, there was an improvement in fever, however the patient developed significant thrombocytopenia which was 90,000 on the 12th day, 57,000 on the 13th day and 62,000 on the 14th day. Sputum culture results showed growth of Escherichia Coli which was resistant to everything except for amikacin. Consequently, the patient was switched to Amikacin. On day 15, patient was shifted back to the ward where he started taking oral sips and small boluses of food. By day 17, patient had markedly improved and was discharged from the ward, with a further review in two weeks' time. Family members were counseled in detail about the ACPO and its potential relapse. They were informed to remain vigilant and watchful for developing symptoms and to bring the patient back to the hospital if needed.

## **Discussion:**

Acute colonic pseudo-obstruction (ACPO), also known as Ogilvie's syndrome, is a condition characterized by massive colonic distension in the absence of mechanical obstruction. Patients presenting with Ogilvie's syndrome have underlying medical and surgical conditions predisposing themto the syndrome. <sup>(1)</sup> Sir Heneage Ogilvie in 1948, who reported two cases of bowel obstruction, without finding any organic etiology for the underlying obstruction.<sup>(2)</sup> Stroke is one of the leading cause of functional immobility which results in autonomic disturbances of the colonic bowel motility i.e. Constipation and fecal incontinence. (3) The exact underlying cause of this abnormality is unknown, but it is presumed that stroke leads to abnormal autonomic instability, which results in reduced vagal tone of the colonic bowel.<sup>(4)</sup>

The actual incidence of acute colonic pseudo obstruction is unknown. However electrolyte imbalance, medications (e.g. anticholinergic) and recent abdominal surgery are the most common underlying predisposing conditions associated with this syndrome.<sup>(5, 6)</sup> Certain neurological disorders such as Parkinson's disease, spinal cord injury, multiple sclerosis and dementia, account for the 9% of this syndrome, with rare cases attributed to acute stroke.<sup>(5, 6)</sup> We have found a similar case report documenting this, in a case of hemorrhagic strokes involving the right frontal cortex.<sup>(7)</sup> The second reported case has been reported in a patient with brainstem hemorrhage.<sup>(8)</sup>

Management options include conservative measures initially and more invasive measures as we progress down the management tree. Conservative includes correction of electrolyte management disturbance (usually potassium, magnesium and calcium) and modifying current medications. In 70 percent of the cases, it is resolved by flatus tube insertion and pharmacological management is responsible in the remaining 30 percent of the cases. (6) Neostigmine, an acetyl cholinesterase inhibitor increases colonic contractility and results in immediate decompression in 80 percent of patients, with recurrence in 3-5 percent of patients.<sup>(6)</sup> An infusion of neostigmine is recommended and effective in refractory cases.<sup>(9)</sup> In this patient, conservative management with electrolyte imbalance correction worked effectively and his obstruction was relieved subsequently. His stay was complicated further by hospital acquired pneumonia due to E. Coli and was treated successfully according to the sputum cultures sensitivity. Prucalpride is a 5-hydroxytryptamine (5-HT) agonist that promotes colonic contractility has been used in refractory cases with some promising results. (10) Endoscopic procedure (flexible sigmoidoscopy) that decompresses the colon was effective in the range of 61% to 95% but with high risk of recurrence. Surgical modality is reserved in refractory and complicated cases of Ogilvie syndrome (e.g. ischemia or perforation). (6)

## Imaging

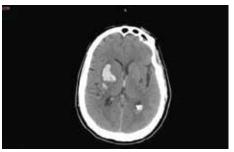


Figure A (1): CT brain plain showing hematoma in the right basal ganglia with ventricular extension

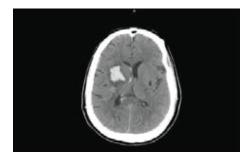


Figure A (2): CT brain plain showing hematoma in the right basal ganglia with ventricular extension

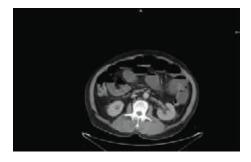


Figure B (1): CT abdomen with contrast performed after symptoms of Obstruction

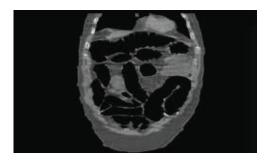


Figure B (2): coronal view of the CT abdomen done after symptoms of obstruction

#### **References:**

1. Maloney N, Vargas HD. Clin Colon Rectal Surg. 2005 May; 18(2):96-101.

2. Ogilvie H, Large intestine colic due to sympathetic deprivation: a new clinical syndrome. Br Med J 1948; 2:671–3.

3. Kumar S, Selim MH, Caplan LR. Medical complications after stroke. The Lancet Neurology. 2010;9(1):105-18.

4. Barron SA, Rogovski Z, Hemli J. Autonomic consequences of cerebral hemisphere infarction. Stroke. 1994;25(1):113-6.

5. De Giorgio R, Knowles CH. Acute colonic pseudo-obstruction. British Journal of Surgery. 2009;96(3):229-39.

6. Jain A, Vargas HD. Advances and challenges in the management of acute colonic pseudo-obstruction (ogilvie syndrome). Clinics in colon and rectal surgery. 2012;25(1):37-45.

7. Wanklyn PD, German A. A case of refractory Ogilvie syndrome following a haemorrhagic stroke. Clin Med (Lond). 2013;13(6):623-4.

8. Lee SJ, Na IH, Choi ES, Jung SH, Yoon JS. Occurrence of Intestinal Pseudo-obstruction in a Brainstem Hemorrhage Patient. Annals of rehabilitation medicine. 2012;36(2):278-81.

9. White L, Sandhu G. Continuous neostigmine infusion versus bolus neostigmine in refractory Ogilvie syndrome. The American Journal of Emergency Medicine.29(5):576.e1-.e3.

10. Smart CJ, Ramesh AN. The successful treatment of acute refractory pseudo-obstruction with prucalopride. Colorectal disease : the official journal of the Association of Coloproctology of Great Britain and Ireland. 2012;14(8):e508.

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