Ogilvie syndrome secondary due to underlying hypokalaemia and anticholinergics: Case report and brief review of the literature

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Abstract
Ogilvie’s syndrome is a rare but potentially life-threatening condition characterised by massive dilation of the colon without a mechanical obstruction. It typically affects older adults and those with underlying medical conditions, such as neurological or cardiovascular diseases, and may result in severe complications such as perforation or sepsis. Diagnosis is based on clinical presentation and radiological studies, and treatment involves a combination of conservative measures, such as bowel rest and pharmacological agents, and interventional procedures, such as endoscopic decompression or surgery.

Here we present the case of a 67 year old male who presented with Ogilvie’s syndrome after changes in his antipsychotic medications. He was given laxatives which led to persistent hypokalemia contributing to worsening distention. This case report highlights the important aspects in management such as cautious use of secretory laxatives (causing worsening Hypokalemia) and combination of motility agents in pseudo colonic obstruction.

Keywords: Ogilvie Syndrome, Hypokalemia, Anticholinergic Agents Gastrointestinal Motility Disorders.

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Introduction
Ogilvie’s syndrome, also known as acute pseudo-colonic obstruction, due to absence of mechanical obstruction. It is an uncommon entity usually seen secondary to Myocardial infarction, Parkinson’s disease and antimotility drugs. Symptoms may be severe enough, requiring total colectomy. A very important aspect to consider is, to exclude persistent hypokalemia as a precipitating factor (as the loss of potassium in the GI tract is difficult to detect due to reduced/absent bowel movements) in order to avoid morbidities associated with surgical intervention. Prompt recognition and management of Ogilvie’s syndrome are essential to prevent morbidity and mortality.¹

This case report highlights the important aspects in management such as cautious use of secretory laxatives (causing worsening Hypokalemia) and combination of motility agents in a patient with pseudo obstruction.

Case Report
We present the case of a 67 year old gentleman, with background of Hypertension, Parkinson’s disease, Anxiety disorder and Herpetic Neuralgia, who came to the emergency department of Ziauddin Hospital Clifton, Karachi with progressive abdominal distention for the last 20 days. He was seen on 21st of February 2022. His distention was associated with dull, intermittent, non-radiating abdominal pain, nausea and decreased oral intake. He also complained of altered bowel habits with on and off passage of flatus, but no history of bleeding per rectum. There was no weight loss and no fever. He had generalized weakness and decreased mobility.

His regular long-term medications included Gabapentin, Quetiapine, Ropinirole, Amlodipine, Procyclidine, Clonazepam, Sertraline, Carbidopa/Levodopa and Amantadine. He was a non-smoker and had no known drug allergies. Clinical examinations showed vitals within normal limits. His Head, eyes, ears, nose, and throat (HEENT), Cardiovascular and respiratory examination were unremarkable. Abdominal examination revealed gross distention of the abdomen which was tense and non-tender. Bowel sounds were sluggish but audible. Digital Rectal Examination revealed good anal tone and impacted stool which was removed.

On initial labs, infective markers were within limits, The blood Urea and Serun Creatinine were in the normal range but the serum Electrocytes showed Hypokalaemia (2.9mEq). ECG and chest Xray were also normal. Initial abdominal Xray and CT scan of the abdomen revealed massive dilatation of the colon (Caecal diameter of > 12cm - normal <9cm)) without any obvious mechanical obstruction. (Figure 1 and 2).

To relieve the discomfort of distention, a rectal tube was inserted to assist the decompression following which large
amount of gas and liquid stool filled the bag. Fluid and Potassium were replaced. However despite replacement he had persistent Hypokalaemia (maximum potassium attained after replacement was 3.2) and no real cause could be ascertained as the patient had no diarrhoea and was not on any medications to explain the potassium loss. Liquid Stool from rectal tube was sent for electrolyte’s level which revealed a Sodium concentration of <10mEq/l and a Potassium concentration of 93 mEq/L (20-80mEq/L). Spironolactone was added to treat his persistent hypokalaemia after which potassium levels increased only up to 3.5mEq/L. As Potassium replacement, rectal tube insertion and spironolactone did not improve the caecal diameter and remained at >12 cm, colonic decompression was repeated to relieve the dilatation. After the colonic decompression the patient’s distention improved mildly. His medications were reviewed and only anticholinergics were stopped. Two days following the decompression and trials of enema his distention increased again. A diagnosis of gastrointestinal potassium wasting resulting in colonic pseudo-obstruction (Ogilvie’s syndrome) was made. This was the diagnosis of exclusion as no obstructive cause could be found to explain the massive dilatation of the Colon. He was given a trial of Pyridostigmine 30mg TDS following which he had bowel movements however his distention did not resolve. A second trial of colonic decompression was given after a week of Pyridostigmine which improved his distention significantly. Serial abdominal x rays were advised which gradually showed improved abdominal distention and the patient was discharged in an improved state.

Discussion

Ogilvie’s syndrome also referred to as acute colonic pseudo-obstruction was first reported in 1948. It refers to massive dilatation of the colon without underlying mechanical obstruction. Dilatation is confined mostly to the caecum and ascending colon. It is most commonly reported amongst elderly men after the sixth decade and in hospitalized patients with severe comorbidities such as Parkinson’s disease, Myocardial infarctions, trauma and sepsis. Furthermore, Electrolyte imbalance and medications as anti-cholinergic narcotics (56 %), H-2 blockers (52 %), phenothiazine (42 %), calcium-channel blockers (27 %), steroids (23 %), tricyclic antidepressants (15 %), and epidural analgesics (6 %).

According to the literature review, acute colonic dilatation could be secondary to decreased parasympathetic activity arising from sacral plexus S2-S3 resulting in distal colonic atony. It has been observed that patients with Ogilvie’s Syndrome have underlying hypokalaemia which resolves after initiation of Potassium sparing diuretics such as spironolactone. Usually in such patients, aldosterone levels are normal.

Clinical symptoms vary from abdominal pain and distention to Nausea and vomiting constipation and diarrhea. Although constipation is more common, around 20-40% of patients were also observed to have diarrhea.

Hypokalaemia may result from the loss of potassium rich fluids from the lower GI tract (such as diarrhea or laxative abuse) due to overexpression or increased responsiveness of the potassium channels in the colon. This may be worsened by giving secretory laxatives to treat constipation. When we assessed the stool sample collected through the rectal tube, it had high potassium content. Explanation that could lead to this possibility was increased secretions in the gut due to the excessive laxative/enema use as our patient presented with absolute constipation. The lack of gut motility caused the pooling of potassium rich secretions in the Gut. Keeping this in consideration, laxatives should be used cautiously as they could possibly worsen the hypokalaemia which is difficult to identify.
The criteria to diagnose Ogilvie’s syndrome include epigastric pain, abdominal distention, constipation, nausea and vomiting (symptoms of obstruction). CT and X ray of abdomen are used to rule out any obstruction. Radiographic imaging should show massive dilatation of the colon (mostly caecum and ascending colon) without any mechanical cause leading to obstruction.3

Different approaches to manage Ogilvie’s include surgical decompression or medical therapy. Medical management of Ogilvie includes nasogastric decompression, rectal tube insertion, electrolyte replacement and discontinuation of any medications leading to this bowel dysfunction. Mobilization and frequent position changing can also help with the treatment.7 Potassium sparing diuretics and Neostigmine/Pyridostigmine has shown promising results in a number of clinical trials. Neostigmine/Pyridostigmine acts by enhancing cholinergic and parasympathetic activity in the colon. In emergency conditions, it has shown clinical improvement.8 Decompressive colonoscopy is also used to provide immediate colonic decompression.

Caecal diameters of 12cm or above are recommended for surgical decompression. The risk of developing ischaemia in such cases is 15%.5

It is essential to highlight that Ogilvie’s syndrome should be an important differential diagnosis in patients who present with Parkinson’s disease, MI, or motility disorders secondary to drugs. The use of non-potassium laxatives should be done carefully so as to avoid worsening of hypokalaemia and the pseudo obstruction. Osmotic and stimulant laxatives should generally be avoided as they can cause worsening of hypokalaemia. Alternative options can include fiber supplements, stool softeners and lubricants.

Our patient had caecal diameters of more than 12 cm. According to the literature review9 such patients end up requiring surgical intervention, however our patient was managed medically by replacing electrolytes, adding Spironolactone and Pyridostigmine to his treatment regime along with multiple sessions of colonic decompression which eventually resolved his dilatation and the patient was safely discharged home. The case reflects that with methodical medical management, surgery can be avoided.

**Conclusion**

In conclusion, Ogilvie’s syndrome is still a rare disorder which can occur without any predisposing cause. Our patient developed Ogilvie’s syndrome because of his medical condition and medications being an underlying cause of his disease but excess use of laxatives causing hypokalaemia further worsened the disease. It is of utmost importance to timely recognize the disease to avoid laxative abuse and to medically intervene before proceeding to the surgical intervention.

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**References**