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OGILVIE SYNDROME UNDER THE CLOAK OF SIGMOID VOLVULUS : A STALEMATE

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ABSTRACT

Acute colonic pseudo-obstruction, also known as Ogilvie syndrome is characterized by acute dilatation of colon in the absence of any mechanical obstruction. Several mechanisms have been implicated in the pathogenesis behind Ogilvie syndrome including autonomic imbalance, disrupted colonic reflex arcs, chronic disease, and medications. Ogilvie's syndrome can often be managed by conservative therapy. However, unrecognized and untreated, the continued distension associated with this can lead to perforation peritonitis that is associated with a higher mortality rate. Thetreatment of colonic pseudo-obstruction comprises of a series of escalating interventions contingent on the degree of distension, risk of perforation, and the patient's response. Treatment options include supportive care, pharmacological therapy with neostigmine , endoscopic decompression (colonoscopy), and surgery.

Acute colonic pseudo-obstruction, also termed as Ogilvie syndrome was initially described by Sir William Heneage Ogilvie in 1948. It is characterized by acute dilatation of colon in the absence of any mechanical obstruction. Ogilvie syndrome is a very rare disorder, thereby, there are lesser known facts about this in medical literature. Dysregulation of the colonic autonomic innervation is hypothesized to play a vital part. Several mechanisms have been implicated in the pathogenesis behind Ogilvie syndrome including autonomic imbalance, disrupted colonic reflex arcs, chronic disease like lupus, scleroderma, and medications like anti-Parkinsonian drugs, clonidine, anti-cholinergics, anti-psychotics and opiates.

Acute colonic pseudo-obstruction is also found to be associated with congestive heart failure, myocardial infarction, Parkinson's disease, Alzheimer's disease. It can also be seen following major orthopedic or spinal surgery or trauma , cesarean section or instrumental delivery.[1,2] Ogilvie's syndrome can often be managed by conservative therapy. However, unrecognized and untreated, the continued distension associated with this can lead to perforation peritonitis that is associated with a higher mortality rate.

Here, we present a case report of a teenager with delayed milestones who was diagnosed as a case of sigmoid volvulus and was operated, but after surgery, later in the post operative period was identified as a case of Ogilvie's syndrome instead.

Case Presentation

A seventeen year old teenager presented to the casualty department in GB. Pant hospital in Portblair, Andaman and Nicobar Islands, with chief complaints of abdominal distension, difficulty in breathing and obstipation for past 10 days. Abdominal distension was insidious in onset, initially was mild but gradually increased over a period of 7-8 days. She gave history of not passing flatus and motion for past 10 days. Along with abdominal distension, she developed difficulty in breathing. She had difficulty in lying down and sleeping. There was no history of vomiting, fever, loose stools, pica or habitual ingestion of inedible things like hair or raw vegetables. Her parents gave history that she had delayed milestones. She started to utter bisyllables at 1 year of age and began to speak meaningful sentences only at 4 years. She had a delayed menarche , at 16 years of age.

On examination, her pulse rate was 140 beats per minute, blood pressure was 110/66 mm Hg in right arm sitting position. Her abdomen was grossly distended with prominent dilated veins over the abdomen.

There was no visible fullness or mass. There was no visible peristalsis. On palpation, there was no local rise of temperature or tenderness. Bowel sounds were absent. On per rectal examination , the rectal mucosa was felt bulging suggestive of raised intra-abdominal pressure.

Routine blood panel along with radiological investigations were ordered for. Her hemoglobin was 9 g/dl and total white blood cell count was 11,600 cells/cubic millimeters.

Her renal function tests were deranged with blood urea 95 mg/dl and serum creatinine 1.9 mg/dl suggestive of renal compromise probably due to dehydration. Plain radiograph of her abdomen revealed dilated bowel loops, mainly large bowel, indicating colonic obstruction. (Figure 1).

To rule out mechanical causes of obstruction, a contrast enhanced CT scan of her abdomen was asked for. Meanwhile, her CT scan reports came, an attempt at per rectal decompression of colon with the help of a soft rectal tube was made. Ryles nasogastric tube was also inserted to aid in relieving distension.

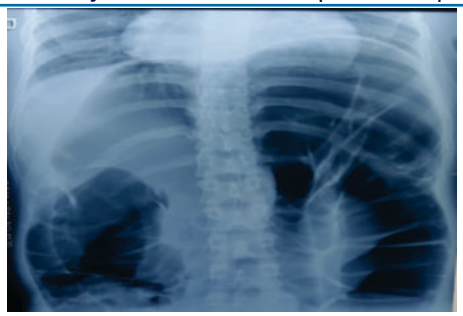

FIGURE 1 : Dilated large bowel loops suggestive of colonic obstruction.

Her CT scan revealed coffee bean sign suggestive of evolving sigmoid volvulus. (Figure 2)

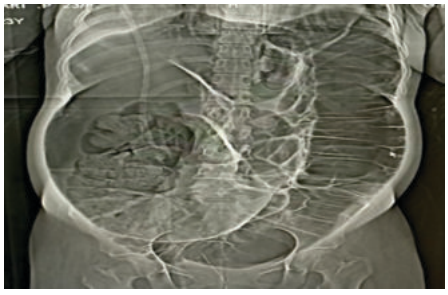

FIGURE 2 : COFFEE BEAN SIGN : Sigmoid Volvulus

As the attempt at rectal decompression of colon failed and her increasing abdominal distension measured by her abdominal girth which increased from 98 to 106 cm, and her agonising abdominal discomfort prompted to plan her for an emergency exploratory laparotomy. CT scan of her abdomen also pointed towards sigmoid volvulus, and due to fear of inevitable gut gangrene or perforation peritonitis, she was posted for surgery. General anesthesia was administered in monitored setting in the operation theatre. Midline exploratory laparotomy incision was given. Abdomen was opened in layers. There was a large sigmoid volvulus. The whole of sigmoid colon was twisted 360 degree upon its own axis. (Figure 3)

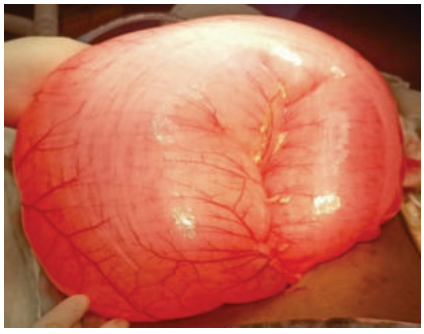

Figure 3 : Sigmoid Volvulus

Though there was a sigmoid volvulus, there was no impending perforation or gangrene. To our surprise, the rest of colon also seemed pathological. The whole of transverse colon was also dilated and distended with gas. (Figure 4). The cecum and ascending colon were also dilated and they contained large amount of soft fecal matter. The whole of colon seemed edematous and distended. The decision to resect whole of sigmoid colon and to bring the descending colon out on to the abdomen as colostomy was made.

The distal stump was closed and left inside the abdomen. Sigmoidectomy with colostomy was done. Patient was shifted to intensive care unit. Her post-operative recovery period was uneventful. She was kept nil by mouth till 48 hours post surgery. She was allowed sips of water from the third post operative day.

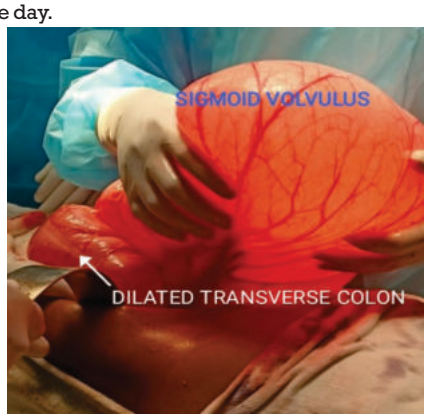

FIGURE 4 Dilated transverse colon

But even till 5 days after surgery, that is nearly 120 hours post surgery, no gut or colonic content was visible from the colostomy site. The colostomy was non functional. What was alarming was her increasing abdominal distension. Her abdominal girth had now risen to 85 cm. On the seventh post operative day, there was marked abdominal distension, abdominal girth measured 96 cm and the patient was again complaining of abdominal discomfort and difficulty breathing. The colostomy was still non-functional. A plain radiograph of her abdomen was taken again on the seventh post operative day. The radiograph showed marked distension of large gut, probably of the transverse colon this time. (Figure 5)

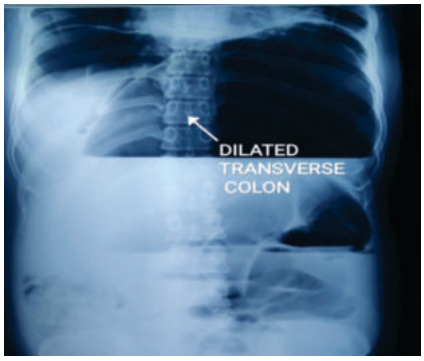

FIGURE 5 Dilated transverse colon in plain radiograph.

A probable diagnosis of acute colonic pseudo-obstruction or Ogilvie syndrome was made as there was colonic obstruction in the absence of a mechanical cause. Other pathologies of colon was ruled out intra-operatively. So this pointed towards one of the rare conditions in clinical scenario, acute colonic pseudo-obstruction or Ogilvie syndrome.

We were aware about the beneficial effects of neostigmine, an acetyl-choline esterase inhibitor in cases of colonic pseudo-obstruction but were reluctant to use it due to its adverse effects of bronchospasm, bronchorrhea and severe bradycardia. But as options for endoscopic decompression were not available readily in this remote island, we decided to go with pharmacological decompression of colon with neostigmine, but with caution and in a monitored setting. A low dose of neostigmine, 1 mg, was administered to the patient intravenously. Within 2 minutes of administering it, we observed that her base line heart rate dropped from 138 to 96 beats per minute. It was hovering around 90 beats per minute, but never dropped dangerously low. But around 45 minutes since neostigmine administration, the patient complained of breathing difficulty. Her oxygen saturation also dropped upto 60 percent. On auscultation there were coarse rhonchi. As expected, the patient developed bronchospasm and severe

bronchorrhea. But she was actively managed with intravenous injections of deriphylline and hydrocortisone. She was nebulised frequently with budesonide and salbutamol. Around two hours later, her oxygen saturation improved upto 98 percent and her chest signs reduced. Her heart rate also stabilized. We were startled to see the passage of colonic mucus secretions and large amounts of colonic gas from the colostomy site within 3 hours of administering neostigmine. Ryles tube was inserted expecting to drain gastric secretions and bile, due to parasympathomimetic and secretomotor actions of neostigmine. As we anticipated, Ryles tube drained 2 litres of gastric secretions and bile, 24 hours later. Passage of colonic gas and mucus secretions from the colostomy began to increase from the second day of administering neostigmine. Liquid fecal matter appeared in the colostomy on the third day post neostigmine therapy. The Ryles tube stopped draining gastric secretions, hence, was removed. The patient was allowed liquids and soft diet from the fourth day. Well formed semi-solid feces started to appear in the colostomy from the fifth day. The patient started to tolerate oral feeds since then. Her abdominal distension also gradually decreased and her abdominal girth was only 76 cm on the fifth day post neostigmine therapy. There was no need for repeat administration of neostigmine. The patient was eventually discharged and kept on regular follow up.

DISCUSSION

Sir W.H. Ogilvie first described Acute Colonic Psuedo-obstruction in two cases of patients with retroperitoneal malignancy who had an acute onset of non-obstructive colonic dilatation.[3] In both patients, the tumor had invaded the celiac plexus, causing Ogilvie to suggest sympathetic deprivation as the etiology of the massive distension.[4] Advances in gastrointestinal physiology have further elucidated the functions of the autonomic nervous system of the colon.

Colonic motor and secretory functions are mediated by the autonomic nervous system. The sympathetic nervous system arises from the spinal cord at the level of the thoracic and lumbar vertebra. Parasympathetic supply to the colon is delivered from two nerve trunks. From the ascending colon upto the level of the sigmoid receives parasympathetic innervation from the medulla oblongata via the vagus nerve. Distal to the splenic flexure, lumbar plexus from spinal segments S2 to S4 supply the parasympathetic innervation.[5] In general, the parasympathetic nervous system increases gut motility and the sympathetic system decreases motility.[6] Abnormalities of the autonomic nervous system, characterized by sympathetic dysfunction, parasympathetic dysfunction, or a combination of both, have been used to explain the etiology of Acute Colonic Psuedo-obstruction . In contrast to Ogilvie's initial theory of sympathetic deprivation, the success of neostigmine suggests that parasympathetic dysfunction is the likely etiology for Ogilvie's syndrome.[7]

Typical patient is a elderly with multiple co-morbidities who is hospitalized for an acute medical event or has undergone any sort of surgery, need not necessarily be abdominal. But in our case the patient was a teenager with history of delayed milestones with no other significant co-morbidities. The presenting symptoms of the condition commonly include abdominal distension, pain, nausea, and vomiting. Obstipation is a common association, but some patients may have diarrhea due to hypersecretion of water. Lack of intestinal contractility is often associated with decreased or absent bowel sounds, but high-pitched, tinkling bowel sounds may also be encountered. Systemic toxicity and peritoneal signs are uncommon and their presence should raise a suspicion of ischemia and perforation.[1,2] Raised total white blood cell counts indicate peritonitis. The renal function values could be deranged due to dehydration.

Plain abdominal radiographs typically demonstrate a distended colon , with the largest diameter usually encountered in the cecum and right colon, which can reach 10 to 12 cm in diameter. Dilation and gas continuing all the way down to the distal rectum support the suspicion of pseudo-obstruction in contrast to a mechanical obstruction in which there is a paucity of gas distal to the obstruction. Gas shadow was seen continuing upto the distal rectum in our patient too. Nowadays, abdominal CT is utilized as standard investigation as it has ability to distinguish the type of obstruction as well as to assess signs of ischemia and impending perforation. Abdominal tenderness, leukocytosis, fever, and dilatation of cecum more than 12 cm are signs indicative of colon ischemia, perforation or impending perforation.

The treatment of colonic pseudo-obstruction comprises of a series of escalating interventions contingent on the degree of distension, risk of perforation, and the patient's response. Treatment options include supportive care, pharmacological therapy with neostigmine, endoscopic decompression (colonoscopy), and surgery.

Non-operative, supportive care is initiated for patients with a cecal diameter that is less than 12cm without evidence of ischemia or perforation. This includes keeping the patient nothing by mouth (NPO), correction of electrolyte disturbances and discontinuation of medications that decrease gut motility like opiates, anticholinergics, anti-Parkinsonian agents, anti-depressants, neuroleptics, clonidine, and atropine. Insertion of a nasogastric tube and rectal tube for decompression may be of help.[1,2] In our case too, even before the radiological investigations revealed a sigmoid volvulus, rectal tube was inserted and an attempt to decompress the colon per-rectally was made, but not with much success, as the abdominal distension kept on increasing.

Neostigmine is the cornerstone of pharmacological therapy. It is an acetylcholine-esterase inhibitor that stimulates the muscarnic receptors and enhances colonic motor activity. Neostigmine is given as a 2 to 2.5 mg intravenous bolus injection over 3 to 5 minutes. But in our case neostigmine at a lower dose of 1 mg was tried due to fear of uncontrolled parasympathetic stimulation.[1,2] But neostigmine even at a lower dose of 1mg could fetch us the desired results as it helped the patient gain colonic motility, and secretions of the colon along with passage of flatus and gas was visible in the colostomy bag.

Neostigmine results in significant parasympathetic stimulation and causes strong colonic peristalsis that usually leads to subsequent flatus and bowel movements as seen in our case. Neostigmine is found to be a safe and effective option for patients with acute colonic pseudo-obstruction who have failed conservative management. Success rates for neostigmine treatment range from 60% to 94% with recurrence rates in upto 31% of patients , with some patients requiring administration multiple times. Neostigmine is contraindicated in mechanical bowel obstruction and in patients with signs of ischemia or perforation.[1,2] It should be used with caution among patients with asthma, chronic obstructive pulmonary disease as it can cause severe bronchospasm and bronchorrhea. The patient discussed here also developed bronchospasm and breathing difficulty due to secretions as a result of bronchorrhea. Her blood oxygen saturation levels even dropped to 60%, following which she was managed actively with oxygen supplementation, nebulisation with salbutamol and budesonide and intravenous injections of hydrocortisone and deriphylline. This is the reason why neostigmine should be given in a monitored setting with atropine readily available. It can also cause severe bradycardia, so it should not be used in patients with coronary artery disease. Other common side effects include vomiting, crampy abdominal pain and excessive salivation.

Colonoscopic decompression should be considered in patients with contraindications to neostigmine or for those who are unresponsive to it. The aim of endoscopic decompression is to advance the scope to the right colon with minimal insufflations and place a colonic decompression tube while removing as much as gas possible from the colon. Patients who do not respond to other lines of treatment or those who demonstrate signs of systemic toxicity, ischemia or perforation, require surgery. Surgical options are determined according to the condition of the patient and the colon.[1,2]

In our case, the query, whether the patient had a primary sigmoid volvulus and developed Ogilvie syndrome in the post-operative period or she had primary acute pseudo-obstruction following which she developed sigmoid volvulus due to massive distension of whole sigmoid and transverse colon, still remains a mystery that is unanswered.

CONCLUSION

Ogilvie's syndrome or acute colonic pseudo-obstruction is a clinical syndrome arising with marked abdominal distension without any evidence of mechanical obstruction. It also is an established post-operative complication too. However, because it is rare, it may be overlooked or treated as adynamic paralytic ileus. Surgeons who perform cardiovascular, thoracic, spinal or orthopedic procedures should include Ogilvie syndrome in their differential diagnosis when their post-operative patient's abdomen becomes markedly distended. Diagnosis is confirmed by radiological scans of abdomen. Prompt treatment is important to avoid the complication of perforated colon, particularly of the cecum. Treatment should include an initial trial of conservative measures with nasogastric decompression, bowel rest, and correction of electrolytes. Cessation of medications which possess the potential to exacerbate the condition, such as opioids, anti-cholinergics and anti-psychotics is also important. After a 24- to 48-hour period, if there is no improvement, the patient should have a trial of neostigmine provided there are no contraindications. Use of colonoscopy, decompression tube placement in the ascending colon, and surgery should be reserved for patients who do not respond to neostigmine pharmacotherapy. In the presence of peritoneal signs of perforation, surgery is the appropriate first line intervention.

Conflict of interests: There is no conflict of interests

Ethics committee approval: Approval was obtained from the institutional ethics committee.

Consent: Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

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