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Case Report
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Ogilvie Syndrome due to Methicillin Sensitive *Staphylococcus aureus*

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Abstract

We describe a case of Ogilvie syndrome that developed in a 50-year-old patient with a VSD who developed hospital acquired pneumonia and methicillin sensitive *Staphylococcus aureus* after being treated for leptospirosis. The patient had developed acute kidney injury and electrolyte imbalance which contributed to the abdominal distension.

Case Presentation

Patient is a 50-year-old with a VSD who suffered from infective endocarditis in 1999 presented to Jayawardenapura Hospital with a history of fever, myalgia and arthralgia for 5 days. They empirically started the patient on ceftriaxone for the treatment for leptospirosis. The patient also developed acute kidney injury the serum creatinine rising to 2. During the hospital stay the patient developed shortness of breath and a new fever was started on treatment with piperacillin tazobactam and blood cultures were taken which showed *Staphylococcus aureus* methicillin sensitive. Patient was then transferred to NHSL due to financial problems. In the ward at NHSL he became significantly more dyspneic and was transferred to Medical Intensive care unit as he desaturated and he was mechanically ventilated for 5 days. His kidney functions started to improve and the patient developed hyponatremia and hypokalemia as the patient became polyureic. Simultaneously the patient developed abdominal dissension and paralytic ileus. He did not pass stool for 3 days. Abdominal supine X-ray showed dilated haustra of the large and small intestine. The patient was referred to the surgical team who advised to keep the patient nil by mouth and nasogastric decompression. The abdominal girth of the patient increased by 5 cm in the next two days and the patient didn't pass flats or stool. The surgical team inserted a rectal tube which reduced the abdominal dissension. During this time the patient electrolytes are corrected with intravenous replacement.

The surgical team also advised a contrast enhanced CT scan of the abdomen did not show any obstructive lesions or mucosal defects there is dilation of the intestine from the cecum to the descending colon. After the CT scan the patient's kidney functions worsened. We suspected a contrast neuropathy. IV hydration was carried out before the CT scan was carried out and afterwards. The renal function began recover slowly.

Vancomycin was started as the patient blood cultures showed *Staphylococcus aureus* and since his fever did not subside. As there was a suspicion of infective endocarditis repeated transthoracic echo is were carried out which did not show any vegetation.

The patient's abdominal dissension came down and the rectal tube was removed however the abdominal dissension worsened. Patient was kept nil by mouth and total parental nutrition was started. Nasogastric tube was inserted and abdomen was decompressed and the patient improved. Patient developed polyuria and electrolyte imbalance which were corrected again intravenously. The fever also subsided after the initiation of vancomycin.

After the abdominal distension came down enteral feeding was started initially with tropic feeds and shifted to full feeds after the patient started tolerating oral. The patient was transferred to the ward after he recovered.

Discussion

Ogilvie syndrome or acute colonic pseudo-obstruction is caused by acute dilatation of the colon that involves the cecum and right hemi-colon and it occurs in the absence of mechanical obstruction. Severe illness/infection or surgery, mostly caesarean section are the usual causes [1].

Acute pseudo-colonic obstruction appears to be more common in men than women and

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patients over 60 years of age [2].

Electrolyte disturbances, debilitation, medications such as narcotics and anticholinergics appear to increase the incidence of Ogilvie syndrome. The clinical manifestations range from abdominal distension and pain to nausea and vomiting and obstipation. To reach a diagnosis one must exclude all structural and known causes of colonic obstruction as well as there should be clinical and radiological evidence [3].

The pathophysiology of Ogilvie syndrome is not known. The imbalance between sympathetic and parasympathetic innervation which culminates in the overall excess of sympathetic supply is postulated [4].

About 90% of patients have bowel sounds present. Distension of the caecum and the ascending colon are noticed in plain films and CT scans help to exclude obstruction [1]. CT scan has the added advantage of demonstrating possible etiology that might have precipitated the event such as retroperitoneal hemorrhage. In the absence of the availability of a CT scan, a contrast enema using a water-soluble contrast can be used to establish the diagnosis provided there are no signs of peritonitis on the physical exam. Contrast enema can cause osmotically driven evacuation of the bowel and relieve the pseudo-obstruction but it could also increase the intra-colonic pressure and cause perforation [5].

The aim of management of pseudo-colonic obstruction is minimizing the risk of colonic perforation and ischemia by decompressing the abdomen. Given this risk patients should be monitored by serial abdominal X-rays every 12 h to 24 h to see the colonic diameter and by laboratory test in the form of full blood count and electrolytes. Initially patients are conservatively managed with IV fluids keeping them nil orally and on NG suction which was performed for our patient [6]. Some of his symptoms were relieved by these actions.

Supportive care by the removal of aggravating agents is the first step in the management of Ogilvie syndrome. Supportive care includes treatment of the underlying condition. Stopping medications that could decrease gut motility such as anticholinergics, calcium channel blockers and opiates. Correction of electrolyte abnormalities with the maintenance of normovolemia with intravenous fluids as the patients are kept nil by mouth [7].

Decompression can also be initiated with the form of intermittent nasogastric tube suctioning and insertion of rectal tube for gravity drainage. Gentle tap water enemas can be administered but their use should be limited due to the risk of perforation. Rectal tube insertion for gravity drainage and nasogastric tube were implemented in our patient and this finally led to the resolution of his symptoms.

Early ambulation of the patient is also encouraged to relieve symptoms patients can be placed prone with their hip elevated on a pillow or placed in a knee chest position [8].

There have been some case reports of the usage of erythromycin 250 mg IV every eight hours for three days or orally 250 mg four times daily for 10 days for treatment but the results have been inconsistent with only gradual improvement in 12 h to 24 h of therapy [9].

If the colonic diameter is more than 12 cm and if 48 h to 72 h has passed with poor resolution to therapy with intravenous fluids, being kept nil orally and NG tube suctioning. Neostigmine can be tried. Studies have been done for both bolus dosing and continuous infusion and some have shown greater colonic diameter reduction with continuous infusion all the later requires more studies for validation. Some have shown however that there is no difference between the two methods [10]. Neostigmine is an anticholinesterase inhibitor and it targets ineffective colonic motility as a result of either excessive sympathetic stimulation or parasympathetic dysfunction or both simultaneously. Continuous cardiac monitoring due to risk of bradycardia should be present with the availability of atropine to be administered if the neostigmine is to be reversed [1].

For patients who fail or have contraindications to neostigmine colonic decompression is performed. For Ogilvie syndrome that has been precipitated by the use of opiates methyl-naltrexone can be administered before the commencement of percutaneous or surgical decompression [11]. When the caecum is dilated more than 9 cm colonic decompression is usually preferred as it reduces the risk of ischemia and perforation but Ogilvie syndrome in itself has the risk of perforation due to difficulty in performing the procedure in pseudo-obstruction [1].

Surgical intervention is usually reserved for candidates who fail pharmacological and endoscopic methods and those who at a risk of peritonitis and perforation [8]. Surgical intervention in the absence of perforation is surgically placed cecostomy tube or a segmental or subtotal resection with primary anastomosis. If there is perforation a total colectomy, ileostomy, and Hartmann procedure are performed with the future intent of performing an ileorectal anastomosis [12].

Learning Points

Ogilvie syndrome should always be thought of in a patient who is critically ill and presents with abdominal distension especially in the absence of obvious mechanical obstruction. Various methods can be utilized in treatment such as intermittent nasogastric suctioning, rectal tube insertion, endoscopic methods and neostigmine administration. Electrolyte imbalances should always be corrected.

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