Case Report

Ogilvie's syndrome in a case of myxedema coma

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ABSTRACT

Ogilvie's syndrome [acute colonic pseudo-obstruction (ACPO)] presents as massive colonic dilatation without a mechanical cause, usually in critically ill patients due to imbalanced sympathetic and parasympathetic activity. The initial therapy remains conservative with supportive measures (correction of metabolic, infectious or pharmacologic factors) followed by neostigmine and decompressive colonoscopy. Surgery is reserved for patients with clinical deterioration or with evidence of colonic ischemia or perforation. A 60-year-old lady presented with fever, altered sensorium, obstipation, bradycardia and abdominal distension. Investigation revealed hyponatremia and acute colonic pseudo-obstruction. Supportive measures and decompressive colonoscopy were not of great benefit. Thyroid profile was suggestive of primary hypothyroidism. Colonic motility was restored only on starting thyroxin. The case is illustrative of the need to consider hypothyroidism, a common endocrine disorder, in the differential diagnosis of Ogilvie's.

Key words: Acute colonic pseudo-obstruction, hypothyroidism, myxedema, Ogilvie's syndrome

Introduction

Ogilvie described in 1948[1] a syndrome of acute colonic obstruction associated with retroperitoneal malignancy. Dudley [2] in 1958 recognized the obstruction to be due to functional rather than mechanical causes and named it as acute colonic pseudo-obstruction (ACPO). It is postulated to occur due to imbalance between sympathetic and parasympathetic innervations of the large bowel.[3] It has been documented to occur with underlying medical and surgical disorders like recent surgery, general anaesthesia, medications, heart failure and electrolyte abnormalities.[4,5] We present Ogilvie's syndrome in a patient with myxedema coma.

Case Report

A 60-year-old lady with no previous co-morbidities presented with history of fever of 6 days, non-projectile vomiting, obstipation and altered sensorium of 2 days duration. Clinically, at admission, the patient had a pulse of 120/min, BP of 172/102 mm Hg and a temperature of 100°F. The patient had pallor, with no icterus, cyanosis and pedal edema. Abdomen was distended; there was no guarding or rigidity. Bowel sounds were not heard, tympanitic note was present all over abdomen. Digital rectal examination did not reveal any impacted faeces. On neurological evaluation, the patient was conscious but drowsy, and was unable to recognize the relatives. Release reflexes or cranial nerve deficit were not present, gag reflex was preserved, the patient was moving all four limbs, cerebellum could not be assessed, there was no neck rigidity. Hematological investigations showed hemoglobin of 12.2 gm%, total lymphocyte count (TLC) of 7300/mm^3 with polymorphs 77%, and platelets of 2.24 × 10^5/mm^3. Serum electrolytes and arterial blood gas analysis showed hyponatremia (Na^+ 122 mEq/l) with potassium level of 4 mEq/l. Renal and liver functions were normal. Electrocardiogram (ECG) on Day 2 of admission revealed junctional rhythm [Figure 1]. Abdominal radiograph revealed gas-filled loops of large intestine [Figure 2a]. On contrast-enhanced computed tomography (CECT) abdomen, the colon appeared distended with maximum cecal diameter of 12 cm [Figure 2b]. Magnetic resonance imaging (MRI)

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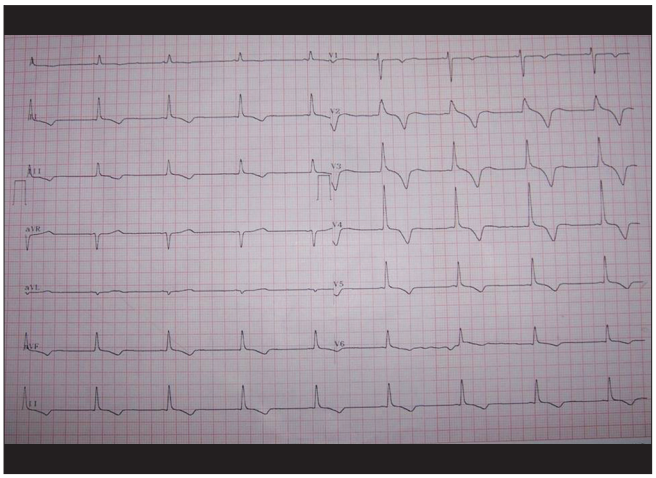

Figure 1: Electroencephalogram showing idioventricular junctional rythm

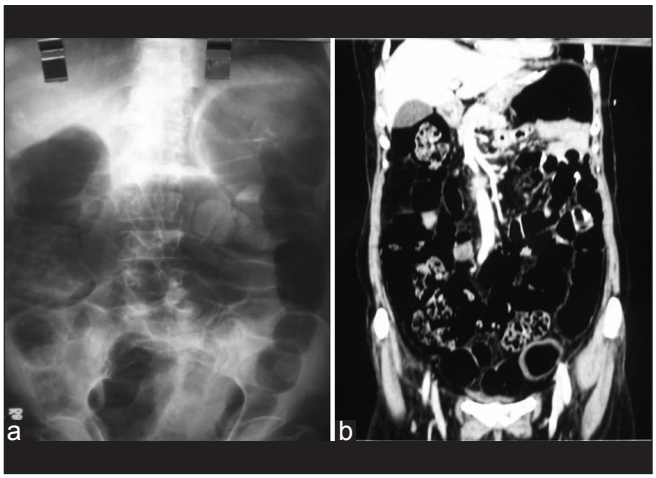

Figure 2: Distended bowel loops on (a) plain X-ray abdomen and (b) coronal reformat

brain and cerebrospinal fluid (CSF) studies were normal. Electroencephalogram (EEG) revealed diffuse slowing suggestive of a metabolic encephalopathy. An impression of encephalopathy secondary to hyponatremia was made. Intravenous 3% saline was instituted after calculating the deficit of sodium, with an aim to raise the serum sodium concentration by not more than 8–10 mEq/l per day. She was started on broad-spectrum antibiotics empirically due to the presence of fever; however, no focus of sepsis could be identified. Patient was kept nil per orally (NPO) and started on parenteral nutrition. Despite these measures, the altered sensorium and abdominal distension persisted. Gut decompression was attempted with colonoscope on Day 4 of hospitalization, but remained unsuccessful. A possibility of hypothyroidism was considered due to the presence of bradycardia, hyponatremia and altered sensorium. Thyroid profile was done and gave the following results: T3 0.37 ng/ml (0.6–1.8 ng/ml), T4 1.6 μg/dl (5.6–13.7 μg/dl) and thyroid stimulating hormone (TSH) 341.57 IU/l (0.35–5.5 IU/l). Anti-thyroid peroxidise (anti-TPO) antibodies were raised (86 U/ml with normal of 0.5–20 U/ml). She was started on replacement with thyroxin from Day 5. Ultrasonography (USG) thyroid revealed normal-sized thyroid and USG-guided fine needle aspiration cytology (FNAC) revealed lymphocytic thyroiditis. Abdominal girth was monitored for signs of peritonism on a daily basis. The abdominal distension regressed gradually and sensorium improved. By Day 11 she was feeding orally and constipation was relieved. The ECG reverted to sinus rhythm. There were no distended bowel loops on abdominal radiograph on follow-up.

Discussion

We have described a patient of hypothyroidism presenting as Ogilvie's syndrome (ACPO). ACPO is diagnosed by excluding mechanical large bowel obstruction (LBO) characterized by more severe abdominal symptoms and signs. Other differential diagnoses include toxic mega colon and ischemic colitis.[5] ACPO can occur at any age, but occurs more frequently in the elderly. It usually presents in association with underlying medical or surgical disorders such as recent surgery, recent general anesthesia, medications, heart failure, infection, and electrolyte abnormality.[4] It mostly occurs in hospitalized patients who are frequently debilitated.[6]

The mechanism of Ogilvie's syndrome is poorly understood and likely to be multifactorial. The main theories involve an imbalance of autonomic influences, which produces a hypotonic bowel, either through increased sympathetic activity or through decreased parasympathetic activity.[3] Neostigmine improves bowel motility in patients with ileus, suggesting that parasympathetic deficiency causes colonic hypotonia producing stasis and dilatation by inefficient expulsion of gas and stool.[7,8]

About 75% of cases resolve spontaneously with conservative therapy in a median of 4 days. In patients who do not resolve, intravenous neostigmine can be instituted. About 80% of patients with pseudo-obstruction respond to 2 mg neostigmine infused intravenously rapidly.[9-12] Colonoscopic decompression relieves symptoms in up to 80% of cases.[13-15] Administration of polyethylene glycol after acute colonic decompression may help prevent relapse.[16] Precipitating conditions, particularly electrolyte abnormalities such as hypokalemia, hypomagnesaemia, and hypocalcaemia, are treated Supportive care includes NPO, intravenous hydration, and discontinuation of narcotic, sedative, or anticholinergic medications. Antibiotics are administered if sepsis is suspected. Ambulation, if possible, or frequent patient repositioning may help move intestinal

gas. Laxatives are generally avoided because fluid tends to accumulate in the bowel. Conservative management is continued for 72 hours if cecal diameter is <12 cm and there are no signs of bowel or peritoneal complication.[17]

Hypomotility and atony of the gastrointestinal tract, mostly involving colon, occurs in myxedematous patients. Patients frequently have symptoms of bloating, flatulence, and constipation. As the hypothyroid state becomes more severe, intestinal hypomotility may progress to atony, ileus, prolapse or volvulus.[18] Chronic pseudo-obstruction has been described in hypothyroidism,[19] but *de novo* presentation as Ogilvie's syndrome (acute colonic pseudo-obstruction) has not been reported. One study on the prevalence of ACPO as a postoperative complication of hip arthroplasty recorded 3 out of 30 cases to have underlying hypothyroidism prior to surgery.[20] Possible mechanism of hypomotility in hypothyroidism is autonomic neuropathy and altered impulse transmission at myoneural junction, intestinal ischemia, intestinal myopathy and glycosaminoglycan (GAG) deposition.[21]

Our patient presented with altered sensorium and abdominal distension. The encephalopathy was thought to be resulting from hyponatremia. We considered diagnosis of ACPO as no mechanical cause could be identified as responsible for the obstruction and duration of disease was less than 6 days. The possible precipitants leading to ACPO could have been dyselectrolytemia and sepsis. Initial measures like administration of 3% saline and large bowel decompression with colonoscopy were not successful in improving her symptoms. Hypothyroidism was not recognized at presentation as it was masked by the sepsis and dyselectrolytemia. The bradycardia and junctional rhythm were unmasked only after treatment of the sepsis. Subsequently, when we diagnosed hypothyroidism and instituted thyroxin replacement, gut motility was restored and her sensorium improved. No previous case report has documented myxedema coma presenting as ACPO.

Conclusion

We conclude hypothyroidism to be a cause of Ogilvie's syndrome. Ogilvie's syndrome should be diagnosed only after excluding mechanical causes for colonic obstruction. We emphasize the need to consider hypothyroidism in the differential diagnosis of any patient with acute onset LBO. If laparotomy is performed on the patient with ileus, the complications of myxedema coma and death may follow. Thus, it is important to consider the possibility of myxedema ileus in cases of marked colonic distension before subjecting them to surgery.

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