September 2014 Critical Care Case of the Month: Bad Case of Colic

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History of Present Illness:

A 70 year old man with a past medical history of chronic kidney disease, bipolar disorder, benign prostatic hypertrophy, hypertension and diabetes presented to the emergency department with constipation associated with bloating for 15 days. He denies flatus. He tried over the counter laxatives (polyethylene glycol) with no relief. He has no recent history of colonoscopy or recent antibiotic use. He denies chills, diarrhea, dysuria, fever, hematochezia, hematuria, melena, nausea or vomiting. In the emergency department, he is tachypneic with a grossly distended abdomen.

Past Medical History:

- Diabetes
- Hypertension
- Chronic kidney disease
- Bipolar disorder
- Benign prostatic hypertrophy
- Hyperlipidemia

Past Surgical History:

- Cholecystectomy 2012

Medications:

- Aspirin 81 mg daily
- Furosemide 20 mg daily
- Quetiapine 300 daily
- Doxazosin- 4 mg daily
- Clonazepam 1 mg – twice daily as needed
- Simvastatin 20 mg – daily
- Pioglitazone 15 mg daily

Social History:

He is a retired farm laborer and worked in a cannery. He is married and has two adult children.

He was a former smoker and quit in 2010 He denies any alcohol or illicit drug use

Physical Exam:

- **Vital signs** – Temperature 37.2 °C, heart rate 84 beats/min, respiratory rate 18-24 breaths/min, blood pressure 121/83 mmHg, SpO2 94 % on 4 L NC

- **General** – Average build, well-nourished, in mild distress
- **HEENT** – Unremarkable
- **Neck** - Supple, no jugular venous distention
- **Chest** – Decreased breath sounds right base more than left base
- **Heart** - Regular rate, normal S1/S2, no murmur
- **Abdomen** – hypoactive bowel sounds, soft, distended, non-tender to palpation but diffusely tympanic.
- **Neurological** - Appropriately moves all 4 extremities, CN II-XII grossly intact
- **Extremities** - No edema
- **Skin** - No rash or palpable nodules

Laboratory:

- **CBC:** WBC $6.4 \times 10^3 /\mu\text{L}$, hemoglobin 15.3 g/dL, hematocrit 45%, Platelets 121,000 / μL .
- **Chemistries:** Na^+ 141 mmol/L, K^+ 4.5 mmol /L, Cl^- 105 mmol /L, CO_2 25 mmol /L, blood urea nitrogen (BUN) 24 mg/dL, creatinine 1.2 mg/dL, glucose 95 mg/dL, calcium 9.9 mg/dL, albumin 4.2 g/dL, liver function tests within normal limits. hemoglobin A1C 5.1%. lactic acid 1.8 mmol/L
- **Coagulation:** Prothrombin time (PT) 16.6 sec, international normalized ratio (INR) 1.3

Radiography:

A CT scan abdomen and pelvis was done and a representative coronal view is shown in Figure 1.

Panel 1. Coronal cut of computed Tomography (CT) of the abdomen and pelvis on admission.

Which of the following are **characteristics** of acute colonic pseudo-obstruction (Ogilvie's syndrome)?

1. Antibiotics and abnormal bacterial overgrowth are implicated in the pathogenesis
2. Immediate surgical decompression is required
3. It is a complication of inflammatory bowel disease
4. It occurs in the absence of an anatomic lesion that obstructs the flow of intestinal contents

Correct!

4. It occurs in the absence of an anatomic lesion that obstructs the flow of intestinal contents

Acute colonic pseudo-obstruction (Ogilvie's syndrome) is a disorder characterized by gross dilatation of the cecum and right hemicolon (although occasionally extending to the rectum). The approximate risk of spontaneous perforation is 3 percent, with an attendant mortality rate of 50 percent (1).

In a review of 400 cases, the most common associations were: trauma (nonoperative) 11%, infection (pneumonia, sepsis most common) 10%, cardiac (myocardial infarction, heart failure) 10%, obstetric or gynecologic disease 10%, abdominal/pelvic surgery 9%, neurological (Parkinson disease, spinal cord injury, multiple sclerosis, Alzheimer disease) 9%, orthopedic surgery 7%, miscellaneous medical conditions (metabolic, cancer, respiratory failure, renal failure) 32%, and miscellaneous surgical conditions (urologic, thoracic, neurosurgery) 12% (2).

In the literature, Cesarean section (even in the absence of bowel injury), normal vaginal delivery and spinal anesthesia are associated with acute colonic pseudo-obstruction. It also occurs as a rare complication during the post-operative period of cardiac surgery, occurring in 0.06% of patients in one series (3). An increasingly recognized association of acute colonic pseudo-obstruction is chemotherapy. Vincristine, all transretinoic acid and methotrexate are most commonly associated (4,5).

The pathogenesis of acute colonic pseudo-obstruction is unknown. There is no proposed mechanism to explain colonic dilation in those patients without obvious involvement of the parasympathetic nerves.

Case continued:

Gastroenterology was consulted for urgent decompression. However, he was deemed a high risk for perforation given the severity of colonic dilatation. A rectal tube was placed and a large amount of air and loose stool was evacuated.

What is the role of **pharmacologic therapy** in patients with acute colonic pseudo-obstruction?

1. Erythromycin binds to motilin receptors in the intestine and stimulates smooth muscle relaxation
2. Neostigmine, an acetylcholinesterase inhibitor, may be effective in producing rapid colonic decompression
3. Pharmacologic therapy should only be attempted if colonic decompression fails
4. There is good evidence that methylnaltrexone in the setting of post-operative acute colonic pseudo-obstruction is efficacious

Correct!

2. Neostigmine, an acetylcholinesterase inhibitor, may be effective in producing rapid colonic decompression

Neostigmine, an acetylcholinesterase inhibitor, may be effective in producing rapid colonic decompression. In a prospective, double-blind, placebo-controlled trial of neostigmine in acute colonic pseudo-obstruction of 21 patients, 11 patients received 2.0 mg of neostigmine intravenously and 10 patients received intravenous saline. Prompt decompression was observed in 11 patients (91 percent) who received neostigmine compared to none receiving placebo, with a time to respond ranging from 3 to 30 minutes and a median of 4 minutes. The most frequent adverse effect was mild to moderate transient crampy abdominal pain. Excessive salivation and vomiting were also noted in a few patients. Symptomatic bradycardia requiring atropine was observed in two patients. Bronchospasm and hypotension have been reported (1). Therefore, neostigmine should be used with caution in patients with bronchial asthma, recent myocardial infarction, and concurrent therapy with beta-blockers. In addition, atropine should be available at the bedside and patients should receive continuous electrocardiographic monitoring with vital sign for 30 minutes. The use of neostigmine in pregnancy has not been well-studied (6). The rate of recurrence of acute colonic pseudo-obstruction after neostigmine ranges from 5 to 33% (7).

Other pharmacologic agents include erythromycin and methylnaltrexone. Erythromycin binds to motilin receptors in the intestine and stimulates smooth muscle contraction. It can be administered intravenously (250 mg every eight hours for three days) or orally (250 mg by mouth four times a day for ten days) (8). Methylnaltrexone has been reported in a case report to be effective in patients with acute colonic pseudo-obstruction that occurs post-operatively, secondary to opioid use, or persists after two injections of neostigmine (9). However, large prospective studies are needed to determine the role of methylnaltrexone in the treatment of patients with acute colonic pseudo-obstruction associated with opioid use.

An algorithm for the approach to patients with acute colonic distention has been proposed (Figure 2, next page) (10). In pseudo-obstruction, if conservative management has a partial/no response, neostigmine should be administered. If neostigmine fails, colonic decompression is recommended. Colonic decompression resolves 70% of acute colonic pseudo-obstruction as measured by reduction in cecal diameter radiographically (2). Surgical cecostomy/percutaneous cecostomy is the last resort if all measures fail to resolve acute colonic pseudo-obstruction because it is associated with high morbidity and mortality (11).

Figure 1. Algorithm for the approach to patients with acute colonic distention. (Reproduced with permission from: Eisen GM, Baron TH, Dominitz JA, et al. Acute colonic pseudo-obstruction. *Gastrointest Endosc* 2002; 56:789. Copyright © 2002 Elsevier).

Case continued:

Despite the rectal tube, NPO and NG decompression, the patient continued to have abdominal distention. He developed progressive dyspnea and hypoxemia requiring more supplemental oxygen. On exam, he is tachypneic, tachycardic and hypertensive. His oxygen saturation is 92% on 6 liters nasal cannula. On auscultation of his chest, coarse breath sounds at the right bases greater than left base. His abdominal exam is unchanged. The rest of his exam is unremarkable. His laboratory studies are significant for hypernatremia (Na^+ 150 mmol/L), hypokalemia (K^+ 3.2 mmol/L) and thrombocytopenia (Platelets 81,000 / μL).

Which of the following **laboratory abnormalities are seen** in acute colonic pseudo-obstruction?

1. Hypercalcemia and hypermagnesemia
2. Hypocalcemia and hypomagnesemia
3. Hypokalemia
4. Both 2 and 3

Correct!
4. Both 2 and 3

Metabolic abnormalities such as hypokalemia, hypocalcemia, and hypomagnesemia, are common and occur in more than 50% of patients (2). Hypokalemia may be due to a secretory diarrhea with high potassium content. The proposed mechanism for this is up regulation of apical big potassium (BK) channels throughout the surface-crypt axes of the colon (12). Colonic BK channels play a significant role in intestinal potassium secretion in a variety of disease processes (13). The main purpose of treating hypokalemia is to prevent life-threatening complications, such as arrhythmias, paralysis, rhabdomyolysis, and diaphragmatic weakness. Several forms of potassium repletion exist. Estimating the potassium deficit assumes that there is a normal distribution of potassium between the intracellular and the extracellular space. The preferred replacement form is potassium chloride because patients with hypokalemia and metabolic alkalosis are often chloride depleted. In addition, potassium chloride raises the serum chloride at a much faster rate than potassium bicarbonate.

Case continued:

He became progressively hypoxemic and a chest x-ray was obtained prior to his transfer to the intensive care unit (Figure 3).

Figure 3. Panel A: Most recent AP film (four days after presentation). Panel B: AP film on admission.

What is the most significant **change noted on the chest X-ray** that can account for his progressive hypoxemia?

1. Increasing pulmonary vasculature, suggesting pulmonary edema
2. Right lower lobe atelectasis and subsequent pneumonia
3. Suboptimal film due to poor inspiratory effort
4. None of the above

Correct!

2. Right lower lobe atelectasis and subsequent pneumonia

This patient likely developed right lower lobe atelectasis secondary to splinting from his abdominal pain. This, in turn, led to the development of pneumonia. Most of the morbidity and mortality of atelectasis is due to the primary disorder. Hypoxemia, which is usually transient, is the main complication. Within 24 to 48 hours, blood is shunted away from atelectatic areas. If the atelectasis is extensive, it may cause enough hypoxemia to require supplemental oxygen or ventilator support.

Case continued:

Neostigmine was administered on admission to the intensive care unit. Broad spectrum antibiotics for health care associated pneumonia (vancomycin and piperacillin-tazobactam) were administered. Chest percussive therapy, incentive spirometry, and bronchodilators were administered to improve the atelectasis. A CT scan of the chest, abdomen and pelvis was obtained (Figure 4).

Figure 4. Panels A and B: Coronal views of CT demonstrating right lower lobe posterior segment consolidation. Panels C and D: CT demonstrating marked distention of the colon with large amount of fecal matter.

Despite these measures, the patient continued to require more supplemental oxygen. Bilevel (bipap) was initiated with improvement in hypoxemia. Intubation was discussed with the patient, however the patient adamantly declined. His code status was changed to do not resuscitate/do not intubate (DNR/DNI). The patient had some improvement in abdominal distention with conservative measures.

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