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Paralytic Ileus Secondary to Electrolyte Imbalance: A Case Study in a 16 Year Old Female

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Abstract

Paralytic ileus is a metabolic state in which the intestines fail to transmit peristalsis due to failure of the neuromuscular mechanism in the small intestines and colon. It is a major cause of morbidity in hospitalized patients especially during late presentations and points of mismanagement. The causes include infections, electrolyte imbalance (hypokalemia, hyponatremia), surgeries and medications. When the exact cause of the disease condition is identified and corrected, paralytic ileus is usually resolved. This case report is that of a 16 year old female who was admitted and managed as a case of paralytic ileus. The patient presented with symptoms of fever, abdominal pain, abdominal distension, vomiting and inability to pass stool or flatus. There was associated body weakness, reduced urine output and weight loss. She was properly examined clinically and sent for various investigations. Investigations such plain abdominal X-Ray, serum electrolyte estimation, chest X-Ray and full blood count were carried out. The results of the investigations done were in keeping with the diagnosis of paralytic ileus, electrolyte imbalance and ongoing sepsis. She was subsequently managed through nil per oral, adequate fluid rehydration, antibiotics and correction of electrolyte imbalance. Following stable clinical state and investigation results, she was discharged and advised on follow-up.

Keywords Paralytic ileus, Hypokalemia, Potassium, Intestinal Obstruction, Electrolyte Imbalance

Major classifications Health Science, Public Health

1. Introduction

Paralytic ileus is a clinical syndrome due to acute and transient disturbance of the transportation of the content of the intestinal lumen due to cessation of smooth muscle motor activity in the small intestines and colon with the potential to return to normal (Jones et al 1989). Paralytic ileus is seen due to the loss of muscle activity. However, paralytic ileus can be caused by a lot of factors such as abdominal operations, infections, inflammation, medications (opiates, calcium channel blocker and tricyclic antidepressant), electrolyte imbalance (hypokalemia, hyponatremia, hypomagnesemia and hypermagnesemia). Following abdominal operations, the mechanisms that are responsible for the dysmotility include surgical stress-induced sympathetic reflexes, inflammatory response medication release and anesthetic/analgesic side effect, each of which can inhibit intestinal motility (Doorly et al 2012). There is reflex inhibition of intestinal motility due to sympathetic hyperactivity and/or depression of parasympathetic excitation.

Disturbance of intracellular and extracellular potassium levels causes abnormal neuromuscular functions. Mild hypokalemia (<3.5mmol/L) may be asymptomatic or linked to nonspecific symptoms such as mental status changes or muscular dysfunction (Gennan FJ 1998). Further losses (<3.0mmol/L) are linked to progressive muscular dysfunction from weakness to paralysis (Cohn et al 2000). Hence, many conditions associated with acute and chronic hypokalemia can be linked to paralysis of skeletal muscle.

The clinical presentation of paralytic ileus includes inability to tolerate oral fluid/solid, nausea, vomiting, lack of flatus or bowel movement, abdominal distension, reduced or absent bowel sounds.

To make a diagnosis of paralytic ileus as well as identify the cause, several investigations may need to be performed such as laboratory evaluation, radiological examination, electrocardiography, ultrasonography and CT scan (Summers et al 1995). The investigations requested include serum electrolyte estimation (with emphasis on serum potassium level), electrocardiography, abdominal x-ray (erect and supine views), ultrasound of abdomen etc. Full Blood Count is done to show the hemoglobin level and identify if infection is present. Chest X-ray may be done to detect pneumonia and the presence of free air in the sub-diaphragmatic space which may be seen due to perforation of the gut (Kumar D 1993). Radiological examination of the abdomen is usually done in three positions (upright, supine and lateral). The accuracy of diagnosis is approximately 85% (Kumar D 1993). Electrocardiography is useful to detect hypokalemia in paralytic ileus and also monitor the patient during its correction.

Up to now, the management of paralytic ileus is aimed at the causative illness (Markum et al 1996). It consists of limiting oral intake and correcting the underlying causes. If vomiting and abdominal distension is present, the stomach should be decompressed using nasogastric tube. Common teaching is to replace electrolyte according to strict guidelines after intestinal surgery, often due to the claim that hypokalemia is a cause of ileus (Benson et al 1993).Treatment of hypokalemia involves the use of potassium chloride (KCl) intravenously since patients are unable to tolerate any oral administration in cases of paralytic ileus. The dose of KCl is generally no more than 10mEq/hr per infusion. Serum potassium should be evaluated every 3-6 hours due to the risk of cardiac arrthymia (Burgess et al 1993).

2. Pathophysiology

There is progressive distension of the paralyzed gut, especially the small intestine, from accumulation of gas and gastrointestinal secretion. The increasing intestinal pressure further increases intestinal secretion and exudation of extracellular fluid from the congested veins leading to further distension. A viscous circle is thus setup leading to loss of water and electrolyte trapped in the gut. The abdominal distension disturbs respiration and respiratory acidosis may occur. The patient may therefore develop dehydration, shock, acidosis, hypokalemia, hyponatremia and renal failure.

3. Case Study

This case study is about a 16 year old single female secondary school student who was admitted to the emergency department complaining of fever, abdominal pain, abdominal distension, vomiting and inability to pass flatus or feces.

Fever was said to have started about one week prior to presentation, low grade, intermittent, no known aggravating factor but said to be relieved by oral analgesics. She also developed abdominal pain the same time which was colicky in nature, intermittent, gradual in onset, aggravated by feeding, generalized, no known relieving factor and not known to radiate to any other part of the body. There was associated abdominal swelling that same period which gradually increased in size up to the time of presentation. Patient started having episodes of vomiting about three days prior to presentation. She had about

two episodes per day of bilious vomitus made up of recently ingested meals. The vomitus was projectile, not mucoid and not blood stained. She also stopped passing feces initially and later stopped passing flatus about the same period. There was associated generalized body weakness, headache, reduced urine output, weight loss and anorexia. There was associated history of frequent passage of loose stools, which was managed at home before the presenting symptoms developed. There was no history of foreign body ingestion, trauma to the abdomen, dysuria, vaginal discharge or itching.

There was no history of abdominal or gynecological surgery in the past, no history of previous protruding abdominal mass and no history of similar conditions in the past. There was no history of menorrhagia or dysmenorrhea. There was no known drug allergy. There was also no record of alcohol or drug use. Family history was not significant.

On examination, she was acutely ill looking, warm to touch with temperature of 37.9°C, in obvious painful distress, not pale, moderately dehydrated, no peripheral lymphadenopathy, no pedal edema. The vital signs were normal with blood pressure of 120/70mmHg, pulse rate of 105 beats per minute and respiratory rate of 20 cycles per minute. Abdominal examination revealed a distended abdomen that moved minimally with respiration, generalized abdominal tenderness, no palpable organomegaly, absent bowel sounds. Digital rectal examination revealed good anal hygiene, normal sphincter tone and empty rectum. Cardiovascular system and respiratory system examinations revealed normal findings.

Investigations were requested to aid in making a diagnosis. Plain Abdominal X-Ray was done; Supine view revealed gaseous dilated bowel loops while the erect view revealed multiple air fluid levels. Serum electrolyte estimation revealed hypokalemia with potassium level of 2.1mmol/L (normal of 3.5-5.0); otherwise normal. Chest X-Ray revealed elevated diaphragms; otherwise normal. Full Blood Count done revealed a PCV of 45% with marked neutophilia. Viral screening for HIV I&II, VDRL, HBsAg and HCV were negative. Random Blood Glucose level was normal (6.4mmol/L). Urinalysis revealed normal findings.

Following counseling on the diagnosis and line of management, she was admitted into the ward and placed on nil per oral. She was resuscitated with intravenous fluids. Nasogastric tube was placed draining bilous effluent while urethral catheter was inserted and draining clear urine. Potassium deficit was corrected using intravenous KCl and potassium levels monitored as expected. She was also placed on intravenous antibiotics. Following treatment, bowel sounds returned gradually, she was commenced on graded oral sips, gradually passed flatus and then stool and had a normal serum electrolyte estimation with potassium level of 3.8mmol/L. She was subsequently discharged on oral antibiotics and other medications and advised on need for routine followup.

4. Discussion

This case study focuses on paralytic ileus that is caused by electrolyte imbalance (particularly hypokalemia). In the patient's history, there were preceding episodes of passage of loose stools that led to dehydration and hypokalemia. This is in keeping with Burgess et al 1993 "Hypokalemia often occurs or should be suspected in cases of diarrhea, chronic or excessive use of non-potassium sparing diuretic agents, respiratory alkalosis in patients with hypokalemic paralysis". The preceding episodes of passage of loose stools were then followed by days of constipation and inability to pass stool. This is also in keeping with Markogiannakis et al 2007 "The overt illness is often preceded by a long phase of altered bowel habits and worsening constipation." The patient had a serum electrolyte estimation done with potassium level of 2.1mmol/L that is in keeping with Cohn et al 2000 "Further potassium losses (<3.0mmol/L) are linked to progressive muscular dysfunction from weakness to paralysis." The PCV of 45% may be false raised due to dehydration while the elevated neutrophil count is possible due to bacteremia and infection. The abdominal x-ray finding are in keeping with Summers et al 1995 which says "there is usually an evenly spread accumulation of air in the gaster, intestine and colon in paralytic ileus". The management of the patient involved the passage of nasogastric tube which is in keeping with Oyasiji et al 2010 "Patients who are vomiting should undergo placement of nasogastric tube for gastrointestinal decompression." It also involved the administration of intravenous potassium chloride (KCl) which is in keeping with Benson et al 1993 "Common teaching is to replace electrolyte according to strict guidelines after intestinal surgery, often due to the claim that hypokalemia is a cause of ileus." The use of antibiotics was also in keeping with Dellinger et al 2013 "If there is any clinical or laboratory evidence of infection (or even sepsis), antibiotics should be given early, as per recommendations of The Surviving Sepsis Campaign."

5. Conclusion

Paralytic ileus is usually characterized by symptoms and signs of intestinal obstruction in the absence of a lesion causing

mechanical obstruction. The diagnosis is made based on clinical presentation, physical examination and investigations that are in keeping with the diagnosis of paralytic ileus. Appropriate line of management can be selected based on the findings. The basic treatment is to treat the underlying cause following which the paralytic ileus will spontaneously resolve. Proper treatment depends on the timely determination of the pathogenesis and on close interdisciplinary collaboration.

Recommendations

Following presentation to the hospital, the cause of the paralytic ileus should be identified and treated accordingly. Failure of conservative measures can be backed up by surgical approach.

More case reports and literature reviews should be encouraged, written and published.

Research works should also be encouraged to elaborate on other causes and line of management for expert care of the patients.

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