A 62-year-old man presented to Mayo Clinic Rochester with a history of steady weight loss during the previous 16 months and worsening weakness during the past 3. He was admitted to the hospital gastroenterology service with a diagnosis of “weight loss and abdominal distension.” Notably, he had undergone an abdominoperineal resection followed by chemoradiation for rectal cancer 2 years before presentation. At presentation, his symptom complex was remarkable for abdominal distention and diffuse, nonradiating, and continuous pain. The pain was notably exacerbated by eating, and he stated that within 3 to 4 hours after a meal he would have generalized cramps throughout the abdomen. The patient had an end colostomy from his surgical intervention, and he stated that, because of increased output from the stoma, he was changing his colostomy appliance over 6 times a day instead of his usual 1 to 2 times. He had no nausea or vomiting, fevers, or chills but did have a 16 kg weight loss during the 3 months before admission. His medical history was remarkable for type 2 diabetes mellitus, the stated vomiting, and diffuse, nonradiating, and continuous pain. The symptom complex was remarkable for abdominal distension by food points more toward an obstructive process than acute infectious gastroenteritis. Malabsorption is also not a likely etiology for this patient’s presentation because his surgery involved large bowel resection; most absorptive processes occur in the small bowel, which was left intact in this patient. However, his history of radiotherapy compels a consideration of radiation enteritis, and, given the lapse in time between his original treatment for rectal cancer and his presentation to our institution, chronic radiation enteritis is a possibility. Its physiologic consequences can include altered intestinal transit, reduced bile acid absorption, increased intestinal permeability, bacterial overgrowth, and malabsorption. The resulting clinical manifestations include nausea, vomiting, lactose intolerance, obstructive symptoms, diarrhea, weight loss, malnutrition, and bleeding (usually in patients with colonic involvement).1 Although malabsorption may have contributed to the patient’s condition at presentation, his primary underlying problem was obstruction. Dumping syndrome2 is also unlikely given the lack of vasomotor symptoms. Defined as the poorly regulated dumping of gastric contents into the small intestine, dumping syndrome is often associated with a pylorus rendered incompetent by surgery. Since our patient had no surgery that manipulated the structure or innervation of the stomach, dumping syndrome is highly unlikely. On examination, our patient was awake, alert, and oriented. He had a pronounced cachectic appearance, with wasting of the temporal muscle as well as of the thenar and hypothenar. His abdomen was distented, with an end colostomy in the left lower quadrant. No parastomal herniation was observed; however, visible peristalsis occurred every 5 minutes, and borborygmi were present.

1. Which one of the following is the most likely etiology of this patient’s symptom complex?
   a. Paralytic ileus
   b. Partial small bowel obstruction
   c. Acute gastroenteritis
   d. Malabsorption secondary to radiation enteritis
   e. Dumping syndrome

   A paralytic ileus is in the differential diagnosis for this presentation, but lack of other causative conditions for paralytic ileus such as pancreatitis or peritonitis makes this diagnosis unlikely. Additionally, before paralytic ileus is assumed, a mechanical obstructive process needs to be excluded because the management strategies for these 2 distinct pathophysiologic processes are different. At the time of presentation, the patient’s symptoms had been progressive over several months, were associated with postprandial pain and cramping, and occurred after surgery for rectal carcinoma. Given such a history, partial small bowel obstruction would be the most likely cause of his symptom complex. Gastroenteritis is unlikely in this setting because the patient had no history of such a syndrome. Additionally, the cyclical nature of his pain complex and the exacerbation by food points more toward an obstructive process than acute infectious gastroenteritis. Malabsorption is also not a likely etiology for this patient’s presentation because his surgery involved large bowel resection; most absorptive processes occur in the small bowel, which was left intact in this patient. However, his history of radiotherapy compels a consideration of radiation enteritis, and, given the lapse in time between his original treatment for rectal cancer and his presentation to our institution, chronic radiation enteritis is a possibility. Its physiologic consequences can include altered intestinal transit, reduced bile acid absorption, increased intestinal permeability, bacterial overgrowth, and malabsorption. The resulting clinical manifestations include nausea, vomiting, lactose intolerance, obstructive symptoms, diarrhea, weight loss, malnutrition, and bleeding (usually in patients with colonic involvement).1 Although malabsorption may have contributed to the patient’s condition at presentation, his primary underlying problem was obstruction. Dumping syndrome2 is also unlikely given the lack of vasomotor symptoms. Defined as the poorly regulated dumping of gastric contents into the small intestine, dumping syndrome is often associated with a pylorus rendered incompetent by surgery. Since our patient had no surgery that manipulated the structure or innervation of the stomach, dumping syndrome is highly unlikely.

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2. Which one of the following is the best approach to the initial management of this patient?
   a. Ordering a human immunodeficiency virus (HIV) test and obtaining an infectious disease consultation
   b. Placing a nasogastric tube that is set at low intermittent suction

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c. Placing intravenous (IV) lines, starting fluids, and assessing electrolyte parameters
d. Administering morphine for pain control
e. Obtaining a surgical consultation for immediate surgical intervention

This patient’s presentation is most consistent with a small bowel obstruction. Although cachexia, thenar, hypothenar, and temporal wasting may be manifestations of HIV, other more plausible differentials should be investigated first. An HIV test may be performed later if clinically warranted; however, it should not be the initial step in managing this patient. Placement of a nasogastric tube would be an initial step in management only in the case of active nausea and vomiting. However, obtaining IV access, starting fluids, and assessing for electrolyte abnormalities would be integral in the initial evaluation and management of this patient’s problems. Patients with small bowel obstruction are predisposed to abnormalities in potassium, magnesium, and phosphorous metabolism. Because such abnormalities can affect a variety of organ systems, they need to be assessed and corrected promptly. Administration of narcotic agents in the setting of bowel obstruction is contraindicated because it could worsen the underlying problem. Although obtaining a surgical consultation may be reasonable for this patient, immediate surgical intervention is not indicated.

Intravenous access was obtained, and fluids were initiated. Initial laboratory studies revealed a slight leukocytosis of 13.6 × 10⁹/L (reference ranges given parenthetically) (3.5-10.5 × 10⁹/L); hemoglobin of 13.2 g/dL (13.5-17.5 g/dL); platelet count of 383 × 10⁹/L (150-450 × 10⁹/L); and a mean corpuscular volume of 90.9 fl (81.2-95.1 fl). The electrolyte panel provided the following levels: 129 mEq/L of sodium (135-145 mEq/L); 3.4 mEq/L of potassium (3.6-4.8 mEq/L); 86 mEq/L of chloride (100-108 mEq/L); 31 mEq/L of bicarbonate (22-29 mEq/L); 0.8 mg/dL of creatinine (0.8-1.2 mg/dL); 2.5 mg/dL of phosphorous (2.5-4.5 mg/dL); 3.5 g/dL of albumin (3.5-5.0 g/dL); and 3.24 mg/dL of ionized calcium (4.65-5.3 mg/dL). An arterial blood gas test showed a pH of 7.58 (7.32-7.42), a PCO₂ of 37 torr (41-51 torr), an arterial PO₂ of 24.8 mg/dL (19-9). The higher-than-expected pH value indicates alkalemia and suggests an alcalotic process; respiratory and metabolic acidosis can thus be excluded. Primary respiratory alkalosis alone is unlikely because the higher-than-expected bicarbonate value suggests a metabolic component. In a metabolic alkalosis with respiratory compensation, the pH would be closer to normal, and the measured carbon dioxide would be high to compensate for the high bicarbonate. In contrast, the measured carbon dioxide value for this patient was within the normal range, suggesting concomitant respiratory alkalosis instead of primary metabolic alkalosis. Therefore, a diagnosis of concurrent metabolic alkalosis and respiratory alkalosis is the most consistent with this patient’s presentation. Prolonged metabolic alkalosis can cause arteriolar constriction, leading to tissue hypoperfusion as well as to widespread effects including coronary artery hypoperfusion and decreased cerebral blood flow. Consequently, the underlying cause of metabolic alkalosis must be determined and corrected. This patient’s blood gas findings were secondary to a contraction alkalosis, which was subsequently corrected with appropriate fluids.

A computed tomographic (CT) scan of the abdomen and pelvis, obtained using IV contrast, revealed a postoperative abdominoperineal resection with end colostomy in the left lower quadrant. The numerous loops of the small bowel filling the abdominal cavity were markedly distended. An apparent transition point was identified within the deep pelvis, which is indicative of a mechanical obstruction.

4. Which one of the following is the most important next step in the management of this patient?

a. Obtaining carcinoembryonic antigen and cancer antigen 19-9 levels
b. Obtaining a surgical consultation and recommending immediate surgical exploration
c. Continuing medical management of the small bowel obstruction
d. Obtaining an oncology consultation for immediate initiation of chemotherapy
e. Obtaining an oncology consultation for immediate initiation of chemotherapy

Although carcinoembryonic antigen and cancer antigen 19-9 levels may be helpful in the future management of this patient, they are not of particular value at this time. These levels are followed to assess the progression of a diagnosed gastrointestinal malignancy, particularly after treatment to assess for recurrence of malignancy. However, these tumor markers are not of diagnostic value. The clear transition point on the CT scan of the abdomen and pelvis provides evidence of an ongoing mechanical small bowel obstruction. Therefore, surgical consultation is necessary to clarify and correct the cause of the mechanical obstruction. An oncology consultation may eventually be useful; however, the initiation of che-
motherapy without a tissue diagnosis of malignancy is premature and potentially dangerous. A flat-plate abdominal x-ray film will not add any more information than that provided by the CT scan; therefore, it is unnecessary at this time.

The patient underwent an exploratory laparotomy, with findings indicative of chronic obstruction. The obstruction was secondary to adhesions, which were lysed during the operation. A diverting ileostomy was created, and the patient initially did well postoperatively. Because of severe malnutrition, total parenteral nutrition (TPN) was initiated, and an oral diet was started. Both were started simultaneously because several days would be required for oral feeding alone to meet the caloric needs of the patient, given his severely malnourished state. On the next day, the patient experienced generalized weakness, diffuse myalgias, and shortness of breath. Laboratory studies showed hypophosphatemia, hyperglycemia, hypokalemia, and an elevated creatinine phosphokinase level.

5. Which one of the following is the most likely etiology of this patient’s new symptom complex?
   a. Refeeding syndrome
   b. Addison disease
   c. Allergic reaction to TPN
   d. Selenium deficiency
   e. Polymyositis

This patient’s presentation is consistent with refeeding syndrome. Refeeding syndrome describes a complex of symptoms seen most commonly when enteral or parenteral nutrition is started after prolonged states of starvation. This syndrome is characterized by multiple metabolic derangements, including hypophosphatemia, hypokalemia, and hyperglycemia. Hypophosphatemia-induced adenosine triphosphate depletion results in muscle weakness, fatigue, and rhabdomyolysis. Addison disease may be considered with the clinical syndrome observed in our patient; however, because it is usually associated with hyperkalemia, it is unlikely given the observed hypokalemia of the patient. Although an allergic reaction to a component of the infusate is a possibility, the electrolyte abnormalities observed in our patient would not be the result of such a reaction. Selenium deficiency, a rare cause of cardiomyopathy, and skeletal muscle dysfunction can manifest symptomatically as dyspnea and weakness, as in our patient. The deficiency is historically associated with long-term use of TPN that is not supplemented with trace minerals. Selenium deficiency is uncommon with current TPN formulations, has no associated electrolyte abnormalities, and is therefore an unlikely etiology of our patient’s presentation. Polymyositis presents as a symmetric, proximal muscle weakness and is often associated with an elevated creatinine phosphokinase level. Patients do not usually present with acute polymyositis; however, a subtype associated with the presence of anti-synthetase antibody can have an abrupt onset. Because hypophosphatemia, hyperglycemia, and hypokalemia are not associated with polymyositis, it is not the likely cause of the patient’s new symptoms.

The marked electrolytic abnormalities were corrected, and feeding was reinstated at a slower rate. The rest of the patient’s clinical course was unremarkable, and he was discharged from the hospital several days later.

DISCUSSION

A common clinical presentation, small bowel obstruction is any lesion that disrupts the natural flow of luminal contents through the small intestine. The blockage may be mechanical, ie, due to a physical barrier, or nonmechanical, ie, due to a neuromuscular failure of peristalsis in the absence of mechanical interference per se. Mechanical obstructions can be further stratified into simple obstruction, in which the intestinal lumen alone is occluded, and obstruction with strangulation, in which the intestinal blood supply is also impaired, leading to intestinal ischemia and possibly necrosis.

Given the many etiologies of small bowel obstruction, the clinician would be advised to begin by classifying the nature of the lesion, thereby narrowing the range of possible causes. Lesions are categorized as those extrinsic to the intestinal wall (adhesions secondary to surgery or radiation, hernias, malignancy), those intrinsic to the intestinal wall (primary tumors, intussusception, radiation-induced strictures), and those intraluminal in nature (gallstones, enteroliths, foreign bodies).

Although the pathophysiologic effects of a small bowel obstruction differ depending on the point of obstruction, distention of the small bowel with gas and fluid near the point of obstruction is almost universally observed. As the bowel dilates, water and electrolytes accumulate both intraluminally and within the bowel wall, accounting for the dehydration and hypovolemia that accompanies this condition. Other consequences of bowel obstruction are increased intraabdominal pressure, decreased venous return, and elevation of the diaphragm, which can lead to compromised ventilation. As the intraluminal pressures increase, mucosal blood flow decreases, potentially leading to ischemia and necrosis of the intestinal lining. These pathophysiologic changes must be addressed when managing a patient with small bowel obstruction.

A thorough history and examination are cardinal in the recognition of small bowel obstruction. The most common symptoms of small bowel obstruction are abdominal distention, vomiting, crampy abdominal pain, and inability to pass flatus. The degree of distention is determined by the location and severity of the obstruction. Other possible symp-
toms are fever, which may be suggestive of strangulation, and tachycardia and hypotension, which are indicative of a high degree of dehydration. Nausea and vomiting can be more severe in proximal than distal obstruction, but distention of the abdomen can be less severe because the proximal intestine acts as a reservoir as it dilates. While examining the patient, the clinician should be vigilant to clues to the underlying etiology. For example, old surgical scars could point to adhesions as the underlying cause. Similarly, interrogation of the hernial orifices could lead to the finding of an incarcerated hernia, and a rectal examination may be remarkable for intraluminal masses or occult blood, potentially indicating a malignancy, intussusception, or infarction.

The initial laboratory evaluation includes both a complete blood cell count, which may reveal a leukocytosis, and an electrolyte panel, which may demonstrate abnormalities secondary to the significant fluid shifts between intravascular and extravascular spaces that occur in this condition. Supine and upright plain abdominal films reveal a ladderlike pattern of dilated small bowel loops with air-fluid levels. Computed tomography is rarely needed to make the clinical diagnosis of bowel obstruction but may be useful in determining extraluminal causes of obstruction. Because it reveals underlying intra-abdominal anatomy, CT may also be useful before definitive surgical intervention in patients with obstructive symptoms.

Because surgical intervention may be required at any time, an early surgical consultation should be obtained. In all cases, aggressive fluid resuscitation and electrolyte repletion should be initiated. Nasogastric tube insertion is mandatory to relieve vomiting, avoid aspiration, and reduce the contribution of swallowed air to abdominal distention. Partial small bowel obstruction can be treated expectantly as long as there is continued passage of flatus and stool. However, even an incomplete obstruction may require surgery if it persists for several days. Surgical intervention is almost always indicated with complete small bowel obstruction because strangulation, which increases mortality markedly, cannot be excluded. However, immediate surgery is not recommended when there is a postoperative obstruction or a history of radiation therapy, inflammatory bowel disease, or numerous operations for obstruction. In such cases, both medical and surgical evaluation and judgment are vital to deciding when best to operate.

REFERENCES

Correct answers: 1. b, 2. c, 3. e, 4. b, 5. a