Perforation of acquired small bowel diverticulum

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A 77-year old woman was seen with an unusual pathologic entity after emergent abdominal exploration—a ruptured small bowel diverticulum. This patient had a known previous history of colonic diverticulosis when she had acute onset of severe abdominal pain. The patient underwent an exploratory laparotomy with resection of representative segments of small and large bowel. The large bowel had evidence of diverticulosis, while the small bowel resected segment had evidence of diverticulitis with rupture. An extensive review of the literature revealed a very small number of reported cases in the world literature (less than 150 cases). We reviewed the history of reported cases of ruptured and nonruptured small bowel diverticular disease, as well as this case.

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In the United States, diverticulosis of the large intestine is a relatively common condition in many individuals of advancing age. According to estimates, roughly one third of the population aged 50 years or more will develop some form of diverticular disease. Diverticulosis, although often asymptomatic, is associated with colicky abdominal pain. The disease may present as diverticular bleeding or with sequelae of acute/chronic diverticulitis. Diverticulosis is traditionally thought of as a large bowel disease with more than 90% originating in the left colon. The remaining 10% are usually attributed to the right ascending and transverse colon. Orr and Russell, in their study, found a 0.42% incidence of small bowel diverticulosis diagnosed by barium contrast radiography. We report the case of a patient who developed an acute surgical abdomen from a ruptured small bowel diverticulum.

Report of case
A 77-year-old woman came to the hospital emergency department complaining of abdominal pain. The symptoms started 2 days earlier and had increased in severity and intensity at presentation. The pain was characterized as intermittently sharp, without radiation. The patient had no other physical complaints. She denied chest pain, shortness of breath, nausea, vomiting, anorexia, fever, chills, hematochezia, hematemesis, melena, acolic stools, or dark-colored urine.

The patient’s medical history was positive for diverticulosis (diagnosed by colonoscopy), hypercholesterolemia, coronary artery disease, atherosclerotic heart disease, and non-Q wave myocardial infarction. Surgical history was positive for a left nephrectomy, bladder suspension, coronary artery bypass surgery, and a previous colonoscopy. The patient’s medication list consisted of diltiazem, lovastatin, digoxin, and aspirin.

Physical examination revealed an elderly woman who appeared healthy, was 70 kilograms in weight, and 5 feet, 2 inches in height. Clinical test results yielded the following values: blood pressure, 194/92 mm Hg; pulse rate, 71 beats per minute; respiratory rate, 14 per minute; and temperature, 99°F. The heart and lungs were normal. Abdominal examination revealed positive bowel sounds, soft abdomen, lower abdominal guarding, and tenderness to palpation. Percussion tenderness was appreciated throughout, and rebound tenderness was present in the lower abdomen. A rectal examination revealed good sphincter tone and guaiac negative stool.

An abdominal radiograph series was obtained and found to be within normal limits, with no evidence of obstruction or free intraperitoneal air. Laboratory test findings disclosed normal values for complete blood count (white blood cell count [wbc], 8.6 x 10³), liver function tests, amylase, lipase, and lactate acid. Serum electrolyte evaluation showed the following values: sodium, 144 mEq/dL; potassium, 4.6 mEq/dL; chloride, 103 mEq/dL; bicarbonate, 30 mEq/dL; blood urea nitrogen, 24 mg/dL; creatinine level, 1.5 mg/dL; and random blood glucose, 100 mg/dL. A urinalysis performed upon admission revealed 3+ bacteria, and 5 to 10 wbc/high power field.

The patient was admitted to the hospital with a diagnosis of acute diverticulitis. She was placed on a regimen of intravenous fluids only, nothing by mouth, and given intravenous ampicillin sodium/sulbactam sodium, 1.5 grams every 6 hours. A cardiology consultation was requested because of the patient’s cardiac medical history.

On the second day of admission, the patient reported improvement in her previous somatic complaints. Abdominal examination result was unchanged, with persistent left lower quadrant tenderness, guarding, and rebound. Laboratory tests were repeated and revealed the following values: lactate acid, 2.6 mmol/L (normal range, 2.0 mmol/L),
and WBC, 12.5 x 10^3. A computed tomography (CT) scan of the abdomen and pelvis was performed that revealed nonspecific soft tissue density adjacent to the sigmoid and descending colon, findings consistent with diverticulitis.

On the next hospital day, the patient’s consent was taken, and she underwent exploratory laparotomy. Abdominal exploration disclosed the presence of a diverticulitis site perforation of the small bowel for which a segmental resection with end-to-end anastomosis was performed. In addition, a sigmoid colon resection was carried out secondary to marked inflammation and edema of the bowel wall. Evidence of perforation of the large intestine was absent. A transverse loop colostomy was also performed at the time of surgery.

The patient had an uneventful postoperative course and was discharged from the hospital on the 10th day.

Discussion

Much of what is known about small bowel diverticula is from isolated historical cases in the medical literature. The first reports of non-Meckel’s small bowel diverticula were initially described by Cooper in 1807.2 In 1906, Gordonier and Sampson3 reported the first case in which surgical treatment of small intestinal diverticula was needed, secondary to a bowel obstruction. In 1920, Case radiographically demonstrated small bowel diverticula, and surgical excision was first carried out by Hunt and Cook in 1921.4

Small bowel diverticula are of two subtypes, namely, the congenital and the acquired. Meckel’s diverticulum, congenital in origin, is a true diverticulum, possessing all layers of the bowel wall. It arises approximately 2 feet from the ileocecal valve on the antimesenteric border of the intestine. Acquired, or false, diverticulum lacks the muscularis layer of the bowel wall and is usually thin-walled, round, elliptical, or multi-lobulated.1 In contrast to Meckel’s diverticulum, acquired diverticula form between the leaves of the mesentery and rarely produce any symptoms. However, when complications arise from these diverticula, serious sequelae may develop. Williams1 reported a 17% incidence of serious medical and surgical complications arising from small bowel diverticula. These complications include hemorrhage, inflammation with perforation, fistulization, abscess formation, and pancreatic-biliary duct obstruction. The branch of a mesenteric vessel, coursing through the fundus of a diverticular sac, can be responsible for the occurrence of a spontaneous hemorrhage. False diverticula may occur in any age group, but the majority of cases have been encountered after the fifth or sixth decade of life with a male-to-female ratio of 1.5:1. Approximately 50% of patients with small bowel diverticula also harbor colonic diverticula.5

Duodenal diverticula occur along the border of the pancreas in close apposition to the second, third, and fourth portion of the duodenum. They are often located near the common bile duct or the pancreatic duct. The ducts may even drain directly into a diverticulum.5 Duodenal diverticula are usually solitary in nature and are multiple only 20% of the time. They are demonstrated radiologically with an incidence of 0.016% to 5.76%.5 In Munnell and Preston’s series, an overall mortality with complicated duodenal diverticular disease was 33%, with perforation being the most commonly encountered morbidity.5 Jejunal and ileal diverticula usually are multiple and occur in clusters. Most occur in the proximal jejunum, with a decreasing incidence toward the ileocecal valve.5 Larger diverticula are found between the duodenum and jejunum near the region of the ligament of Treitz. Lesions of the jejunum and ileum are represented radiologically at approximately 0.073% to 1.3%.5

Small bowel diverticula are usually asymptomatic and could remain undiagnosed unless discovered by autopsy or by surgical exploration. The extent of complications seems to be directly related to the number of lesions present. Chronic symptoms have been associated with a single large multiloculated diverticulum. In Altemeier’s series of sixteen patients, the most common complication of jejunal diverticulosis was “…chronic or recurrent jejunal obstruction.”6 These patients suffered from nausea, left-sided/epigastric pain, vomiting, intestinal hypermotility, and distention or thickening of the jejunal bowel wall. The incidence of pseudo-obstruction of the small bowel lumen secondary to jejunal diverticulosis is 10% to 25%.7 Bacterial overgrowth and abnormal intestinal motility have been postulated as the cause of pseudo-obstruction. Previous authors have noted abnormal peristaltic activity of the affected segment of small bowel, termed “jejunal dyskinesia.”8

Using contrast radiography, Cooke and colleagues9 described hypermotility within the area of jejunal diverticula and poor peristaltic activity. With this in mind, chronic and intermittent changes in small bowel wall kinetics may be the reason that jejunal diverticulosis often is undiagnosed or asymptomatic. The lesion is usually discovered incidentally by upper gastrointestinal radiography or CT scanning. Occasionally, a patient may only have the symptoms of chronic indigestion, irritable bowel, diffuse abdominal pain, postprandial bloating, or malabsorption. The acute complications of jejunal diverticula are obstruction, hemorrhage, or perforation, as seen in 6.5% to 10.4% of cases.2

Perforated jejunal diverticulosis is extremely rare; Babcock and coinvestigators7 found only eight reported cases in their series review. The type of patient that develops acute surgical abdomen is expected to have the more common causes of peritoneal irritation. These include perforated peptic ulcers, large bowel diverticulitis, appendicitis, small or large bowel obstructions, or even acute pancreatitis. The diagnosis of perforated small bowel diverticulitis is discovered intraoperatively. The mortality rate of 21% for jejunal diverticulosis is probably secondary to delays in diagnosis. Many authors have stated difficulty in recognizing the presence of small bowel diverticula at the time of surgical abdominal exploration.2 Our patient had a history of sigmoid diverticulosis. However, upon abdominal exploration, the perforated segment was actually discovered.

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within the region of a small bowel diverticulum, not in the large intestine. The most common complications detected at laparotomy are hemorrhage and inflammation. However, jejunal diverticulitis can still perforate without the presence of inflammation. Perforation can occur secondary to trauma, nasal cocaine ingestion, and foreign body impaction.⁷

Although there is currently no proven etiology for the formation of small bowel or jejunal diverticulosis, several theories exist. Increased intraluminal pressure, also a major cause of large bowel diverticulosis, is the most common accepted cause of small bowel disease. Other etiologies that have been examined are obesity, venous stasis, and constipation.⁷ Krishnamurthy and coauthors⁷, in four cases of jejunal diverticulosis, discovered fibrosis of the circular and longitudinal smooth muscle of the jejunum. In addition, they suggested that abnormalities of the muscle or myenteric plexus accounted for the diskinetic activity of the bowel wall. They believed this state eventually resulted in the protrusion of a diverticulum.

Unfortunately, at this time, the treatment of small bowel diverticulosis can not be directed at the cause because of the absence of any proven etiologic factor. Asymptomatic patients with small bowel disease incidentally discovered by radiography are not recommended to undergo any specific treatment protocol. However, emergent surgical exploration is indicated when “acute abdomen” is seen on physical examination. During laparotomy, the small and large intestines should be adequately evaluated for gross abnormalities and perforations. A ruptured small bowel diverticulum, if discovered, can be managed using either of two technical options that have been reported. Limited local excision of the diverticulum has been suggested by some of the authors; however, a high incidence of postoperative complications exists with this treatment protocol.⁷ The general consensus is to perform a limited resection of the involved diseased segment with end-to-end anastomosis.

Conclusion
Although very rare, small bowel diverticulosis has increased in incidence with the advancing age of the population. The patient in our hospital underwent surgical exploration and that led to a clinical diagnosis of ruptured diverticulitis. This was confirmed by pathologic analysis of the specimens. This type of patient must be treated with the appropriate standard of care and very selectively evaluated for the need for surgical intervention. Small bowel diverticulosis and diverticulitis certainly exist and, therefore, must always be included in the differential diagnosis of the patient with abdominal complaints when a definitive etiology cannot be identified.

References