Superior Mesenteric Artery Syndrome Treated with Percutaneous Radiologic Gastrojejunostomy

Jeong Woo Choi, Ju Young Lee, and Hyeon Geun Cho

Department of Internal Medicine, Sung Ae Hospital, Seoul, Department of Internal Medicine, Catholic Kwandong University College of Medicine, Incheon, Korea

Superior mesenteric artery (SMA) syndrome is a rare condition that must be differentiated from other gastrointestinal diseases manifesting as upper abdominal pain, nausea, or vomiting. The description of SMA syndrome is compression of the third portion of the duodenum by the SMA and the abdominal aorta. SMA syndrome is managed with nasoenteral nutrition or surgical strategies such as laparoscopic duodenojejunostomy. However, SMA syndrome treated using enteral nutrition by percutaneous radiologic gastrojejunostomy has not been reported. Here, we report our experience of successfully managing a case of SMA syndrome with percutaneous radiologic gastrojejunostomy. (Korean J Gastroenterol 2016;67:321-326)

Key Words: Superior mesenteric artery syndrome; Duodenal obstruction; Enteral nutrition; Gastrojejunostomy

INTRODUCTION

Superior mesenteric artery (SMA) syndrome, also known as Wilkie’s syndrome, is an uncommon, benign cause of duodenal obstruction by compression of the third portion of the duodenum due to narrowing of the angle between the SMA and the abdominal aorta. SMA syndrome manifests as nonspecific obstructive symptoms of nausea, vomiting, epigastric pain, and abdominal distension. Anorexia and weight loss can occur in chronic cases. SMA syndrome can result in significant electrolyte imbalances, dehydration, and malnutrition. In addition, recurrent vomiting may lead to aspiration pneumonia. Due to the life-threatening nature of some of these problems, prompt diagnosis and treatment are necessary. SMA syndrome is managed with total parenteral nutrition (TPN), nasojejunal enteral feeding, or surgical strategies such as laparoscopic duodenojejunostomy.1 To the best of our knowledge, there are no reports of enteral nutrition by percutaneous radiologic gastrojejunostomy (PRGJ) being used to treat SMA syndrome. Here, we describe a case of SMA syndrome successfully managed with PRGJ.

CASE REPORT

A 72-year-old woman who was bed-ridden for 20 years after deep brain stimulation for Parkinson’s disease presented at the emergency room complaining of abdominal distension and vomiting for three days. On admission, she was hypo-
tensive (blood pressure 55/30 mmHg) with a pulse rate of 96 beats per minute and a body temperature of 36.2°C. She was drowsy and had no focal neurologic deficits. On abdominal examination, her abdomen was distended and decreased bowel sounds were detected. She was positive for direct tenderness on the whole abdomen, but negative for rebound tenderness. She developed metabolic acidosis (pH 7.27) with a serum bicarbonate concentration of 11.5 mmol/L. A complete blood count revealed leukocytosis without anemia: white blood cells, 21,700/mm³ (polymorphonuclear leukocytes, 95%); hemoglobin, 14.9 g/dL; and platelets, 241,000 cells/mm³. Blood urea nitrogen, creatinine, and C-reactive protein levels were elevated (41.8 mg/dL, 1.7 mg/dL, and 4.97 mg/dL, respectively). Other laboratory values were as follows: serum sodium, 131 mmol/L; potassium, 4.3 mmol/L; chloride, 97 mmol/L; calcium, 8.4 mg/dL; glucose, 189 mg/dL; albumin, 3.7 g/dL; total bilirubin, 0.9 mg/dL; alkaline phosphatase, 158 IU/L; aspartate aminotransferase, 25 IU/L; alanine aminotransferase, 12 IU/L; and amylase, 131 IU/L. We could not assess body mass index as the patient was bed-ridden.

A plain abdominal radiograph demonstrated a distended and dropped stomach (Fig. 1). Abdominal computed tomography (CT) revealed complete obstruction of the third duodenal portion and confirmed that the stomach was distended in addition to being filled with gastric contents. The aortomesenteric angle was >22°, but compression of the duodenum was noted by the SMA and aorta, highly indicative of SMA syndrome (Fig. 2).

We inserted a nasogastric tube to decompress the stomach, then drained 2.5 L of gastric contents. After five days of conservative treatment using nil per os and nasogastric drainage, an additional nasojejunal tube was inserted to commence enteral nutrition (Fig. 3). Upper gastrointestinal endoscopy showed irregular ulcerations on the body of the stomach and narrowing of the third portion of the duodenum without intrinsic duodenal pathology (figure not shown). On day 18 after admission, percutaneous gastrojejunostomy was performed with a double-lumen catheter (Carey-Alzate-Coons gastrojejunostomy set; Cook, Bloomington, IN, USA) for simultaneous feeding via the jejunum and aspiration of the gastric content of the stomach (Fig. 4). We performed the
procedure under monitored sedation with midazolam and propofol. After percutaneously puncturing the air-filled stomach through the abdominal wall using an introducer needle, we advanced a guidewire into the antrum of the stomach under fluoroscopic guidance. We removed the introducer needle, advanced a radiopaque seeking catheter over the guidewire, and placed the tip beyond the ligament of Treitz through the obstructive site. We then removed the catheter and inserted an introducer needle. We placed a gastrojejunostomy catheter over the guidewire, and the introducer needle sheath into the jejunum. After the sheath and the guidewire were removed, we injected contrast dye to identify the position of the catheter tip in the jejunum. No procedure-related complications occurred, and oral intake was allowed after the procedure, beginning with a liquid diet. A plain abdominal radiograph two days after the procedure showed a normal gastrointestinal gas pattern without regurgitation of contrast dye into the stomach (Fig. 5). The PRGJ tube was well tolerated and the patient was discharged 16 days after PRGJ without any delayed complications. At five months post-PRGJ, an upper gastrointestinal series was performed to reassess the passage of material through the stomach and duodenum, and revealed that the obstruction of the duodenum had resolved without regurgitation into the stomach (figure not shown). We replaced the PRGJ tube with a percutaneous radiologic gastrostomy (PRG) tube to allow for nutrition more reflective of a normal diet. After replacing the PRG tube, the patient remains tolerant of treatment without any obstructive symptoms.
DISCUSSION

SMA syndrome was first described by the Austrian professor Carl von Rokitansky\(^2\) in his anatomy textbook published in 1842, but remained pathologically undefined until 1927, when Wilkie\(^3\) published the first comprehensive series of 75 patients. Several causes of SMA syndrome are proposed, including significant weight loss, prolonged bed rest, external abdominal compression, anatomic variation, surgical alterations to the anatomy such as spinal surgery, and eating or psychological disorders.\(^4\) We considered prolonged immobilization to be the cause of SMA syndrome in the present case. We could not monitor the patient’s changes in body weight, but a CT scan helped to exclude other causes.

Upper barium studies or conventional angiography have been used to diagnose SMA syndrome. However, the enhanced CT scan is the current gold standard because it is non-invasive and does not induce vomiting in patients with obstruction. Furthermore, it can demonstrate the etiology of the gastrointestinal obstruction and evaluate the aortomesenteric anatomy.\(^7\) In our case, abdominal CT showed that the aortomesenteric angle was $>22^\circ$. However, compression of the duodenum by the SMA and aorta was noted, strongly suggestive of SMA syndrome.

The treatment of SMA syndrome is aimed at managing the precipitating factor, which is usually related to weight loss. Loss of intra-abdominal fat (mesenteric fat) around the SMA will decrease the aortomesenteric angle and the distance between the SMA and the aorta causes mechanical compression of the duodenum.\(^8\) Therefore, conservative treatment with nasogastric decompression and nutritional supplementation to facilitate weight gain and resolve electrolyte imbalances and dehydration are attempted before surgical bypass, which is reserved for those who do not respond to medical treatment. Nasoenteral or nasogastric tube feeding has been widely and safely used to provide adequate nutrition in patients who are prohibited from ingesting food and fluids until the obstructive lesion resolves or their enteral function returns.\(^9,10\) A retrospective study reported that the overall success rate of medical treatment for SMA syndrome was 71.3% (57 of 80 patients) during a median observation period of five months (range, 1-84 months), while the recurrence rate was 15.8% (9 of 57 patients).\(^7\) Another retrospective study showed a success rate for initial nasojejunal tube placement of 91.5% and a tube-related late complication rate of 23% in patients without SMA syndrome.\(^11\) However, the success rate of nasaonteral treatment in SMA syndrome patients was not evaluated, because most studies did not divide the medical treatment group into subgroups, such as TPN and nasoenteral groups.

As mentioned above, nasojejunal tubes are safe, but they can be uncomfortable and easily dislodged.\(^15\) In addition, the long-term use of nasoenteral tubes is associated with several problems such as tube blockage, gastroesophageal reflux, and tracheoesophageal fistula.\(^12\) Other possible complications due to feeding tube misplacement include pneumothorax, intrapleural infusion of the enteral diet, esophageal perforation, and necrosis of the nasal ala.\(^12,13\) While the nasoenteral tube is acceptable when enteral nutrition is indicated for one month or less, for more prolonged nutritional replacement experts recommend percutaneous or surgical enterostomy.\(^14\)

Laparoscopic duodenjejunostomy is the surgical treatment of choice if conservative management fails, and is successful in up to 90-100% of cases.\(^1,7\) However, patients with serious comorbidities are at increased risk of postoperative complications such as pneumonia, confusion, stroke, myocardial infarction, and acute kidney injury. These comorbidities include old age, malnutrition, chronic obstructive pulmonary disease, advanced liver cirrhosis, heart failure, and ischemic heart disease.\(^15-18\) Long-term medical treatment should be considered in these high-risk patients instead of surgical options.

PRG, also known as transgastric jejunostomy, is performed for nutritional support or gastrointestinal decompression as percutaneous gastrostomy with the benefit of reducing gastroesophageal reflux or aspiration.\(^19\) Primary percutaneous gastrojejunostomy is a procedure in which a tube tip is placed into the jejunum through a puncture site in the stomach wall. As with PRG, major complications include peritonitis, gastric perforation, bleeding requiring transfusion, deep stomal infection, septicemia, and aspiration. Minor complications include superficial wound infection, peristomal leakage, and tube dislodgement. Minor tube-related complications such as migration, blockage, and balloon rupture can occur.\(^19,20\) PRGJ may reduce the risk of aspiration, but gastrojejunostomy catheters are longer and narrower.

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than those used in gastrostomy, and this can lead to complications such as tube blockage. 21 A retrospective study reported that clinical success of PRGJ was achieved in all 29 patients, and no procedure-related complication occurred except for a case of pneumoperitoneum. 19 Several studies report that PRG and PRGJ entail fewer major complications (0% vs. 5.3%) and a higher success rate (100% vs. 84-89%) than endoscopic placement. 22,23 Major complications occurred less frequently after radiologic gastroscopey (5.9% vs. 9.4% for percutaneous endoscopic gastrostomy [PEG] and 19.9% for surgery) and 30-days procedure-related mortality rates were highest for surgery (2.5% vs. 0.3% for radiologic gastrostomy and 0.53% for PEG) in a meta-analysis. 24 However, no comparative studies of PRGJ and laparoscopic duodenojejunostomy are available.

Combined gastrostomy and gastrojejunostomy tubes can be used for simultaneous enteral feeding and gastric drainage with a decreased risk of aspiration. 25 Double-lumen gastrojejunostomy tubes have been used for simultaneous enteral feeding and gastric decompression. 26,27 These tubes may be particularly useful in patients with SMA syndrome. Long-term gastric decompression is accomplished by continuous or intermittent drainage through the aspiration (gastric) port without nasopharyngeal discomfort and complications associated with prolonged nasal intubation. Because feeding through gastrostomy is unavailable until the duodenal obstruction has resolved, nutritional support can be maintained through the feeding (jejunal) port.

The patient in this report was at high risk for surgical complications due to her advanced age and history of being bed-ridden due to a neurological disease. The patient’s condition improved with long-term nutritional therapy by PRGJ using a double-lumen gastrojejunostomy tube to avoid the risks of surgical intervention under general anesthesia.

In conclusion, PRGJ can be a feasible alternative medical option in high-risk patients with chronic SMA who have comorbidities that make them unsuitable for surgical management. Further case reports are needed to allow high-quality comparisons of the available treatment modalities for SMA syndrome.

REFERENCES


