Unusual Complication of Superior Mesenteric Artery Syndrome: Spontaneous Upper Gastrointestinal Bleeding with Hypovolemic Shock

Kai-Hsiung Ko¹, Shih-Hung Tsai², Chih-Yung Yu¹, Guo-Shu Huang¹, Chang-Hsien Liu¹, Wei-Chou Chang¹*

Departments of ¹Radiology and ²Emergency Medicine, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, R.O.C.

Superior mesenteric artery (SMA) syndrome is an unusual form of duodenal obstruction. Complications of SMA syndrome may sometimes develop and are usually associated with marked gastric dilatation, although most complications can be corrected by supportive treatment. In this article, we report a case of severe SMA syndrome with hypovolemic shock in a 24-year-old man. Multidetector-row computed tomography with reconstructed images was performed to establish the diagnosis. Spontaneous gastrointestinal bleeding is an extremely uncommon complication of SMA syndrome, and emergent surgical intervention was unavoidable in our patient. To our knowledge, no other such case has been reported in the English-language literature. [J Chin Med Assoc 2009;72(1):45–47]

Key Words: complications, esophageal tear, multidetector-row computed tomography, superior mesenteric artery syndrome, upper gastrointestinal bleeding

Introduction

Superior mesenteric artery (SMA) syndrome is a well-defined disorder that was first described by von Rokitansky. Although severe complications are uncommon among the limited number of reported cases, development of a severe complication is potentially fatal and requires immediate surgical intervention. Altere, we present an unusual case of SMA syndrome with severe complications of acute spontaneous gastrointestinal bleeding and hypovolemic shock. Esophageal submucosal tear and active arteriolar bleeding over the esophagogastric junction were noted during surgical intervention. The patient underwent surgical repair of the bleeding and duodenojejunostomy with a favorable outcome.

Case Report

A 24-year-old man was admitted to our emergency room due to severe vomiting with sudden hematemesis.

He had been robust prior to this sudden event. Tracing the patient's history, he had experienced weight loss of about 10 kg in the recent 2 weeks and had begun to suffer from intermittent abdominal fullness and poor appetite. Because of the persistent abdominal symptoms, he consulted a doctor at a local clinic. Conservative treatment was prescribed for presumed acute gastritis or peptic ulcer, but the treatment was ineffective, with no change in symptoms.

On physical examination, the patient was pale, was vomiting and had diffuse abdominal pain. His vital signs, including pulse rate of 80 beats/minute, blood pressure of 105/60 mmHg, and respiratory rate of 15 breaths/minute, were relatively stable. On abdominal radiography, the abdomen was mildly distended. Bowel sounds were normal. Laboratory investigation revealed mild anemia (hemoglobin of 11.3 mg/dL) and markedly positive occult blood (4+) in gastric contents. Other laboratory values were within the normal ranges. After the series of laboratory studies and initial management, admission was recommended because peptic ulcer with acute upper gastrointestinal bleeding was suspected.



*Correspondence to: Dr Wei-Chou Chang, Department of Radiology, Tri-Service General Hospital, 325, Section 2, Cheng-Kung Road, Taipei 114, Taiwan, R.O.C.

E-mail: chougo2002@yahoo.com.tw • Received: January 23, 2008 • Accepted: August 29, 2008

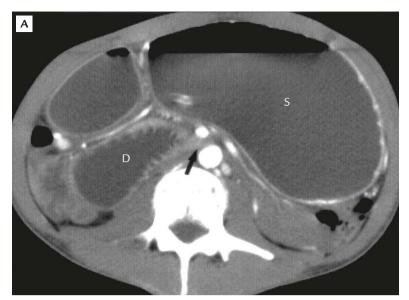




Figure 1. (A) Axial computed tomography and (B) reformatted coronal images of the abdomen reveal marked distension of the stomach (S) and dilatation of the second portion of the duodenum (D). The abruptly narrowed third portion of the duodenum (black arrow) is compressed between the superior mesenteric artery and abdominal aorta.

On admission, emergent upper gastrointestinal endoscopy was performed, and acute gastrointestinal bleeding was confirmed. However, there was no ulceration of the stomach and duodenum, but there was marked distension of the stomach. The procedure was not completed owing to the patient's intolerance, but active bleeding over the esophagogastric junction was noted. The active bleeder could not be stopped by endoscopy. The patient's blood pressure had dropped to 80/50 mmHg and hemoglobin had dropped to 8.5 mg/dL.

After resuscitation with isotonic fluid and blood transfusion, his vital signs stablized temporarily. Abdominal computed tomography (CT) was then performed, which showed marked distension of the stomach and proximal duodenum with abrupt narrowing of the third portion of the duodenum (Figure 1). The abruptly narrowed third portion of the duodenum was obviously compressed between the SMA and abdominal aorta. Preoperative diagnosis was SMA compression syndrome. The aortomesenteric angle measured 11° and the aortomesenteric distance was 3 mm on reformatted sagittal CT images. There was no evidence of abnormal free air, focal mass or bowel wall thickening of the stomach and duodenum. In addition, CT scan at the esophagogastric junction level (Figure 2) showed edematous thickening and enhancement of the distal esophageal wall, indicating submucosal injury of the distal esophagus.

With the persistent gastrointestinal bleeding and progression to hypovolemic shock, emergent surgical

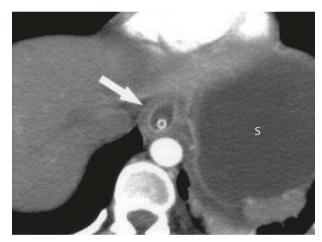


Figure 2. Axial computed tomography at the esophagogastric junction level shows edematous thickening and enhancement of the distal esophageal wall (white arrow). Submucosal esophageal tear was confirmed at surgery.

intervention was performed. During the operation, the active bleeder was identified as longitudinal submucosal lacerations of the distal esophagus with arteriolar bleeding over the left aspect of the esophagogastric junction. Also noted was that the ligament of Treitz was in a higher than normal position with narrowing of the aortomesenteric angle. SMA syndrome with duodenal compression and obstruction was confirmed at surgery. Eventually, the bleeder was repaired, and the patient received duodenal mobilization and duodenojejunostomy to release the SMA compression.

The patient achieved an 8-kg weight gain during admission and was discharged in a stable condition. The patient remained well over 1 year of follow-up.

Discussion

SMA syndrome is an unusual form of intestinal obstruction caused by severe vascular compression of the third portion of the duodenum. This syndrome is often precipitated by conditions that narrow the angle, including possible immobilization, external compression due to body cast treatment of spinal fracture, and rapid weight loss for any reason. ^{1,4,5} With vascular compression, a variety of gastrointestinal symptoms, including nausea, vomiting, postprandial abdominal pain and weight loss, usually develop when the aortomesenteric angle is less than 22° and/or the distance is less than 8 mm. ⁶⁻⁸

Severe complications that have been reported to result from SMA syndrome include acute gastric dilatation, gastric pneumatosis, gastric rupture, portal venous gas, and cardiovascular collapse. 2,3,9 When intragastric pressure from gastric distension gradually exceeds 30 cmH₂O over a period of time, intramural blood flow is impaired and results in gastric ischemia and necrosis. CO2 gas may develop as a result of mucosal ischemia; the mucosal ischemia also allows the gas to pass into the bowel wall or portal vein. 10,11 An interesting aspect in our case was that the stomach became massively distended over a relatively short period of time, but no full-thickness necrosis or perforation occurred; instead, we found an esophageal submucosal tear with active arteriole bleeding at surgery. The possible cause of the esophageal tear and active bleeding over the esophagogastric junction may have been related to the severe coughing and vomiting.

Establishing the diagnosis of SMA is usually based on clinical suspicion and confirmed by radiologic studies such as upper gastrointestinal barium study or conventional angiography. Traditionally, upper gastrointestinal series and conventional angiography are suggested as diagnostic modalities, but disadvantages of nonspecific radiologic appearances of barium studies and invasiveness of angiography often preclude using these modalities. However, with improved technique of multidetector-row CT and the advantage of reconstructed images, this modality has not only become a rapid, available and noninvasive technique to facilitate diagnosis, but it can also detect potentially fatal complications that may require immediate surgery.

In conclusion, SMA syndrome may result in potentially fatal complications such as acute gastrointestinal bleeding and hypovolemic shock. Multidetectorrow CT with reformatted images is a reliable modality for early diagnosis of SMA syndrome and its complications, leading to a proper clinical intervention, including gastric decompression and possible surgical correction.

References

- Welsch T, Buchler MW, Kienle P. Recalling superior mesenteric artery syndrome. *Dig Surg* 2007;24:149–56.
- Lim JE, Duke GL, Eachempati SR. Superior mesenteric artery syndrome presenting with acute massive gastric dilatation, gastric wall pneumatosis and portal venous gas. Surgery 2003; 134:840–3.
- Sakamoto Y, Mashiko K, Matsumoto H, Hara Y, Kutsukata N, Yamamoto Y. Gastric pneumatosis and portal venous gas in superior mesenteric artery syndrome. *Indian J Gastroenterol* 2006;25:265–6.
- Huang IF, Wu TC, Wang KS, Hwang B, Hsieh KS. Upper gastrointestinal endoscopy in children with upper gastrointestinal bleeding. *J Chin Med Assoc* 2003;66:271–5.
- Crowther MA, Webb PJ, Eyre-Brook IA. Superior mesenteric artery syndrome following surgery for scoliosis. Spine 2002; 27:E528–33.
- Agrawal GA, Johnson PT, Fishman EK. Multidetector row CT of superior mesenteric artery syndrome. *J Clin Gastroenterol* 2007;41:62–5.
- Hines JR, Gore RM, Ballantyne GH. Superior mesenteric artery syndrome: diagnostic criteria and therapeutic approaches. Am J Surg 1984;148:630–2.
- 8. Unal B, Aktas A, Kemal G, Bilgili Y, Guliter S, Daphan C, Adyinuraz K. Superior mesenteric artery syndrome: CT and ultrasonography findings. *Diagn Interv Radiol* 2005;11:90–5.
- Lay CS, Yu CJ, Tyan YS. Abdominal aortic dissection with acute mesenteric ischemia in a patient with Marfan syndrome. *J Chin Med Assoc* 2006;69:326–9.
- Turan M, Sen M, Canbay E, Karadayi K, Yildiz E. Gastric necrosis and perforation caused by acute gastric dilatation: report of a case. Surg Today 2003;33:302–4.
- Liebman PR, Patten MT, Manny J, Benfield JR, Hechtman HB. Hepatic-portal venous gas in adults: etiology, pathophysiology and clinical significance. *Ann Surg* 1978;187:281–7.