Superior mesenteric artery syndrome due to duodenal edema in a chronic renal failure patient: a case report

Sawako Goto · Susumu Ookawara ·
Masahiro Sugai

Received: 19 December 2012 / Accepted: 29 March 2013 / Published online: 19 April 2013
© Japanese Society of Nephrology 2013

Abstract A 74-year-old woman had been treated conservatively for type 2 diabetes mellitus (DM) and DM nephropathy since 2004. Her renal function gradually deteriorated, and she developed chronic renal failure (CRF) with a serum creatinine level between 2.5 and 3.0 mg/dl in 2010. She presented to our hospital with nausea and vomiting related to the progression of CRF in May 2011. An abdominal computed tomography (CT) scan showed distention of the upper digestive tract from the stomach to the third portion of the duodenum, ascites, and compression of the duodenum by the abdominal aortic artery and the overlying superior mesenteric artery (SMA), which was caused by edema along the gastrointestinal tract, in particular, at the duodenum. Therefore, she was diagnosed with SMA syndrome. Decompression of the distended upper digestive tract was achieved using a nasogastric tube, and hemodialysis was also initiated in order to adequately control her fluid balance disorder, which was thought to have induced the duodenal edema and ascites. After initiating these treatments, her condition gradually improved, and she fully recovered after approximately 2 months of treatment. Thus far, there has been no report of SMA syndrome caused by duodenal

edema associated with the progression of CRF that was successfully treated by conservative treatment and the initiation of blood purification.

Keywords Superior mesenteric artery syndrome · Chronic renal failure · Duodenal edema · Hemodialysis

Introduction

Superior mesenteric artery (SMA) syndrome was first reported in 1842 by Rokitsansky [1] and has been defined as compression of the third portion of the duodenum by the abdominal aortic artery (AA) and the overlying SMA.

Various causes of SMA syndrome were recently described in detail [2], and the aortomesenteric angle and distance were thought to be the most important factors in the development of SMA syndrome [3]. There are only a few reports on the relationship between dialysis and SMA syndrome in chronic renal failure (CRF) patients [4, 5]. In addition to the narrowing of the aortomesenteric angle, previous reports have shown that dialysis patients had specific backgrounds, such as severe vascular calcification in hemodialysis (HD) and peritoneal sclerosis in peritoneal dialysis [4, 5]. Furthermore, dialysis patients with SMA syndrome do not have a good prognosis because of their poor physiological condition and malnutrition [5].

In this report, we describe a patient with SMA syndrome induced by edema along the gastrointestinal tract, in particular, at the duodenum, associated with CRF progression. In addition to conservative treatment, including nasogastric tube placement, HD was immediately initiated to adequately control the fluid balance disorder. Thereafter, this patient gradually improved and fully recovered after approximately 2 months of treatment.

S. Goto
Department of Internal Medicine, Yamagata Prefectural Central Hospital, 1800 Aoyagi, Yamagata, Yamagata 990-2292, Japan

S. Ookawara (✉)
Department of Internal Medicine, Nishikawa Town Hospital, 581 Kaisyuu, Nishikawa-machi, Yamagata 990-0702, Japan
e-mail: su-ooka@hb.tp1.jp

M. Sugai
Department of Surgery, Nishikawa Town Hospital, 581 Kaisyuu, Nishikawa-machi, Yamagata 990-0702, Japan

Case report

A 74-year-old woman with diabetes mellitus (DM) and DM nephropathy had been treated conservatively since 2004 at the outpatient department in our hospital. Her renal function gradually deteriorated, and she developed CRF with a serum creatinine (Cr) level between 2.5 and 3.0 mg/dl in 2010.

She was admitted to our hospital on May 20, 2011, because of nausea and vomiting. On physical examination, she presented abdominal distention and bilateral pitting edema of the lower legs. On laboratory analysis, both blood urea nitrogen (BUN) and Cr concentrations had increased to 60.6 and 3.6 mg/dl, respectively, and these results indicated the aggravation of CRF. Upper gastrointestinal endoscopy showed oozing from the lower esophageal mucosa, and she was diagnosed with reflux esophagitis. Therefore, conservative treatment, which consisted of parenteral nutrition and a proton pump inhibitor, was initiated.

Although the patient had stopped the consumption of solid and liquid foods during the conservative treatment, her symptoms, including nausea, vomiting, and abdominal distention, had not been relieved completely. On May 23, CRF was further aggravated (BUN 74.4 mg/dl, Cr 4.52 mg/dl), and her chest X-ray showed dilatation with a cardiothoracic ratio (CTR) of 60.7 % and an enhanced shadow of the pulmonary vessels. It was difficult to maintain her body fluid levels with conservative treatment alone; therefore, we considered initiating blood purification to improve her fluid balance disorder. On May 24, her urinary output decreased to 400 ml/day, and she presented with nocturnal delirium caused by uremia. Her abdominal X-ray showed severe distention of the stomach; therefore, abdominal computed tomography (CT) was performed. The abdominal CT scan showed distention of the upper digestive tract from the stomach to the third portion of the duodenum, ascites, and compression of the duodenum by the abdominal AA and the overlying SMA (Fig. 1). The aortomesenteric distance was not narrow, but seemed to have increased (23 mm) because of edema along the gastrointestinal tract, in particular, at the duodenum. Therefore, she was diagnosed with SMA syndrome caused by duodenal edema resulting from a fluid balance disorder due to CRF. In addition to the conservative treatment, a nasogastric tube placement was performed for gastric and duodenal decompression. Furthermore, HD was started for correcting the uremia and fluid balance disorder, including pulmonary edema, ascites, and duodenal edema. After the treatments were initiated, the amount of drainage through the nasogastric tube was >1800 ml. The abdominal X-ray showed that the drainage had successfully relieved the stomach dilatation. After adding diuretics (furosemide

40 mg/day, hydrochlorothiazide 25 g/day), as well as the initiation of HD, her urinary output increased, and the uremia also resolved. Her gastrointestinal symptoms and fluid balance disorder gradually improved, and, thereafter, the nasogastric tube was removed. She was able to begin enteric nutrition with rice gruel. No evidence of SMA syndrome recurrence was observed. HD was continued two times per week to maintain her state, and she fully recovered after approximately 2 months of treatment. An abdominal CT was performed to re-examine the condition of the gastrointestinal tract and the relationship between the SMA and the abdominal AA. This abdominal CT scan indicated that the aortomesenteric distance and angle were 11 mm and 40°, respectively (Figs. 2 and 3), and both were within the normal range. Compared with the initial

Fig. 1 Initial abdominal computed tomography (CT) scan at the level of the duodenum crossing between the superior mesenteric artery (SMA; *single arrow*) and the abdominal aorta. The aortomesenteric distance is 23 mm, and a markedly dilated duodenum and duodenal edema are seen (*double arrow*)

Fig. 2 Abdominal CT scan after recovery at the level of the duodenum between the SMA (*single arrow*) and abdominal aorta. The aortomesenteric distance is 11 mm, and no duodenal dilation or edema is observed (*double arrow*)

Fig. 3 **a** Abdominal CT scan (sagittal view) showing severe aortic calcification. **b** Abdominal CT scan (3D view) showing an aortomesenteric angle of 40°

(a) Sagittal view

(b) 3D view

abdominal CT scan, the scan after 2 months also indicated that the duodenal edema had completely resolved.

Discussion

SMA syndrome is defined as vascular compression of the third portion of the duodenum between the abdominal AA and the overlying SMA. Duodenal obstruction causes nausea, vomiting, and abdominal distention. The main cause of this syndrome is a narrow aortomesenteric angle and numerous predisposing conditions with a potential impact on the aortomesenteric angle have been summarized into three categories: severe weight loss in catabolic states, external and intra-abdominal compression, or mesenteric tension [2].

It is useful and important to evaluate the image examination for diagnosing SMA syndrome. Abdominal X-ray shows the double-bubble sign due to obstruction of the third portion of the duodenum and, also, dilatation of the upper digestive tract from the stomach to the third portion of the duodenum [6]. Furthermore, abdominal CT scan is the most effective imaging technique for assessing the aortomesenteric angle and distance, fat tissue, and obstruction of the duodenum [2]. The normal range of the aortomesenteric angle and distance has been reported to be $38\text{--}65^\circ$ and $10\text{--}28$ mm, respectively [7, 8]. On the other hand, an aortomesenteric angle of $<22\text{--}25^\circ$ and a distance of <8 mm correlated well with the symptoms of SMA

syndrome [3, 8]. In this case, the initial abdominal CT scan showed that the aortomesenteric distance was rather large (23 mm), which was caused by the duodenal edema. It has been previously reported that endoscopic examination of the duodenum showed pale mucosa, edema, congestion, and ulceration in patients with CRF [9, 10]. In the present case, since edema of the interstitial space and extremities and pulmonary congestion were observed, the probable cause of SMA syndrome was duodenal edema due to the fluid balance disorder that was induced by CRF. In fact, the abdominal CT scan after the recovery from SMA syndrome showed the resolution of duodenal edema, normalization of the aortomesenteric distance (11 mm), and a normal range of the aortomesenteric angle (40°). Moreover, there have been some reports about SMA syndrome with CRF [4, 5]. In these reports, the onset of SMA syndrome had been attributed to vascular calcification in HD patients, peritoneal sclerosis, and adhesion in peritoneal dialysis. The vascular calcification at the abdominal AA was recognized with abdominal CT scans in this case (Fig. 3a). Therefore, a relationship between the onset of SMA syndrome and vascular calcification might also be possible.

As to the treatment, three treatment categories have been postulated. These treatments include drainage with nasogastric tube placement and mobilization into the prone or left lateral decubitus position, parenteral nutrition to correct the fluid and electrolyte imbalance, and surgical treatment [2]. In this case, nasogastric tube placement for gastrointestinal decompression was performed, and

parenteral nutrition was initiated. Furthermore, HD was also performed for managing the fluid balance disorder due to CRF. After initiating these treatments, the patient demonstrated clinical improvement, which included resolution of the gastrointestinal and uremic symptoms, increased urine output, and relief of the edematous conditions. Most importantly, the initiation of HD was considered to be effective in resolving the duodenal edema, which was the main cause of SMA syndrome in this case.

In conclusion, we reported a case of SMA syndrome caused by duodenal edema due to CRF. Previous reports about the relationship between the onset of SMA syndrome and gastrointestinal edema are scarce. However, according to our experience, SMA syndrome may be due to duodenal edema. Especially, in edematous patients with CRF, it should be taken into consideration to perform HD treatment for managing the fluid balance disorder, as well as the conservative treatments, including nasogastric tube placement and parenteral nutrition.

Conflict of interest All the authors have declared no competing interest.

References

1. Rokitsansky C. *Lehrbuch der pathologischen Anatomie*. Vienna: Braunmüller und Seidel; 1861. p. 187–91.
2. Welsch T, Büchler MW, Kienle P. Recalling superior mesenteric artery syndrome. *Dig Surg*. 2007;24:149–56.
3. Unal B, Aktaş A, Kemal G, Bilgili Y, Güliter S, Daphan C, Aydinuraz K. Superior mesenteric artery syndrome: CT and ultrasonography finding. *Diagn Interv Radiol*. 2005;11:90–5.
4. Kitaura K, Harima K. Superior mesenteric artery syndrome with vascular calcification in a maintenance hemodialysis patient. *Clin Nephrol*. 2009;71:228–30.
5. Yap DY, Ma MK, Lai AS, Chan SY, Seto WK, Lam MF, Chan TM, Lai KN. Superior mesenteric artery syndrome complicating dialysis patients with peritoneal failure—report of 3 cases. *Clin Nephrol*. 2011;75:37–41.
6. Wu MC, Wu IC, Wu JY, Wu DC, Wang WM. Superior mesenteric artery syndrome in a diabetic patient with acute weight loss. *World J Gastroenterol*. 2009;15:6004–6.
7. Derrick JR, Fadhli HA. Surgical anatomy of the superior mesenteric artery. *Am Surg*. 1965;31:545–7.
8. Neri S, Signorelli SS, Mondati E, Pulvirenti D, Campanile E, Di Pino L, Scuderi M, Giustolisi N, Di Prima P, Mauceri B, Abate G, Cilio D, Misseri M, Scuderi R. Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. *J Intern Med*. 2005;257:346–51.
9. Misra V, Misra SP, Shukla SK, Jaiswal PK, Agarwal R, Tondon S. Endoscopic and histological changes in upper gastrointestinal tract of patients with chronic renal failure. *Indian J Pathol Microbiol*. 2004;47:170–3.
10. Kang JY. The gastrointestinal tract in uremia. *Dig Dis Sci*. 1993;38:257–68.