

[CASE REPORT]

Superior Mesenteric Artery Syndrome Accompanied by Acute-onset Type 1 Diabetes Complicated with Graves' Disease

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Abstract:

A 35-year-old man experienced general fatigue and could not eat solid food because of nausea and vomiting. His weight abruptly decreased from 49 to 45 kg after 2 weeks. A detailed examination indicated superior mesenteric artery syndrome (SMAS) accompanied by acute-onset type 1 diabetes complicated by Graves' disease, referred to as autoimmune polyglandular syndrome type 3A (APS3A). Although SMAS has a good prognosis, some cases require emergency surgery, especially when complicated by gastric perforation. In our case, APS3A and SMAS developed rapidly and at approximately the same time, resulting in a cycle of mutual exacerbation.

Key words: autoimmune polyglandular syndrome, superior mesenteric artery syndrome, type 1 diabetes

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Introduction

Type 1 diabetes is often accompanied by autoimmune endocrine and non-endocrine diseases, referred to as autoimmune polyglandular syndrome (APS) (1). APS is classified into types 1 to 4, with APS3 being a combination of autoimmune thyroid disease and other autoimmune diseases without Addison's disease or hypoparathyroidism (2, 3). Among APS3 types, those involving type 1 diabetes and autoimmune thyroid disease [APS type 3A (APS3A)] are the most frequent (1, 3). Autoimmune thyroid disease includes Graves' disease, which causes hyperthyroidism, and Hashimoto's disease, which causes hypothyroidism. Excessive thyroid hormones promote glucose absorption from the intestinal tract and lipolysis, suggesting that ketoacidosis is likely to occur when hyperthyroidism occurs concurrently with type 1 diabetes (4).

Superior mesenteric artery (SMA) syndrome (SMAS) is a disease in which the horizontal portion of the duodenum is compressed between the SMA and the aorta or spine, resulting in impaired passage and duodenal obstruction (5).

SMAS develops when mesenteric and retroperitoneal adiposity is diminished due to rapid weight loss or metabolic disorders, and the aorta-intestinal distance decreases accordingly. Furthermore, SMAS may develop when anatomical variation, such as a short or high insertion of the Ligament of Treitz at the flexion of the duodenum, cause the duodenum to move more cranially to the vascular angle (5, 6). Vomiting and obstructive syndrome are also observed, and sometimes there are nonspecific symptoms, leading to a delayed diagnosis of SMAS (7).

Given that both diabetes and Graves' disease induce wasting among those affected by these disorders, a combination of these diseases can precipitate SMAS. However, there have only been a few studies concerning the incidence of SMAS due to diabetes and Graves' disease (8, 9). More importantly, such studies have shown that SMAS usually occurs after a long duration of diabetes. To our knowledge, a case of SMAS accompanied by acute-onset type 1 diabetes and Graves' disease has not been reported in the literature.

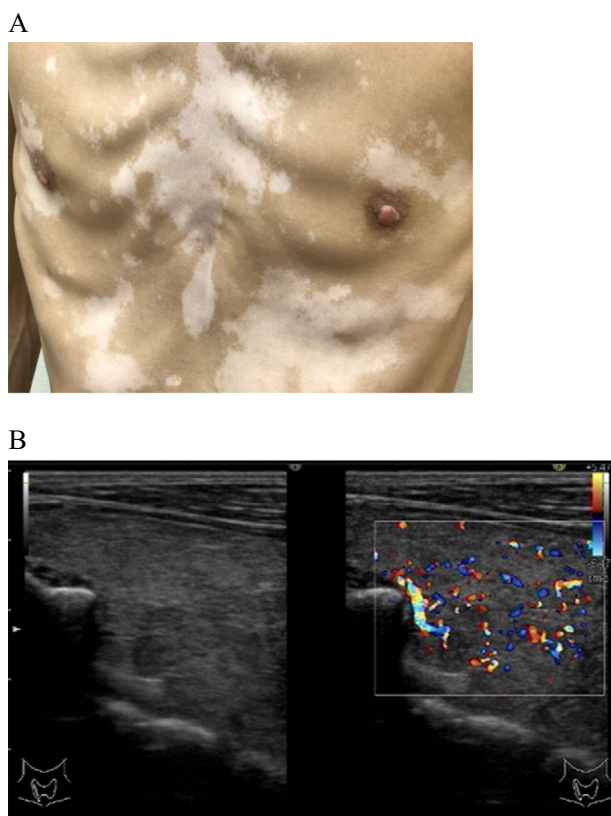


Figure 1. A: The patient, showing vitiligo vulgaris over his entire body. B: Ultrasonography of the thyroid shows diffuse goiter with hypervascularity.

Case Report

A 35-year-old man experienced general fatigue and hyperhidrosis for 6 months and eventually experienced polydipsia. He found he was unable to eat solid food because of nausea and vomiting. Consequently, he visited a clinic and was transferred to a hospital due to nausea and vomiting that was refractory to initial antiemetic treatment. At the hospital, his point-of-care blood glucose level was 616 mg/dL. In addition, he had tachycardia (heart rate >150/min) and a high fever (38°C). Since the patient also had hyperthyroidism [thyroid-stimulating hormone (TSH) <0.02 mIU/L, free T3 (fT3) 8.18 pg/mL, free T4 (fT4) 3.94 ng/dL] and potential thyroid crisis, he received not only insulin but also 45 mg of thiamazole and 200 mg of potassium iodide. The patient was subsequently referred to our hospital for a further evaluation and management of severe hyperglycemia and hyperthyroidism.

Upon an examination, his height and body weight were 178 cm and 45 kg, respectively. His weight was noted to have suddenly decreased from 49 to 45 kg in a span of 2 weeks prior to visiting the hospital. Previously, he had not undergone regular weight determinations nor any medical examinations. It was noted, however, that at 20 years old, his weight had been 56 kg, and this value had been maintained (for a consequent BMI of 17.7 kg/m²) until 6 months

before hospitalization. The patient was not aware of any significant weight loss. Furthermore, the patient had no significant medical history and did not drink regularly. He had smoked 20 cigarettes per day for 15 years but had become unable to smoke 1 month prior to admission due to his poor physical condition. He was self-employed with his own business and did not exercise regularly. His axillary temperature, pulse, and blood pressure were 37.1°C, 81 bpm (irregular), and 133/85 mmHg, respectively, upon sufficient rehydration therapy. Since he was in a clear state of consciousness, he did not meet the diagnostic criteria for thyroid storm.

Electrocardiography revealed atrial fibrillation, and he had mild thyromegaly and finger tremors. He had no diabetic complications but had vitiligo vulgaris over his entire body (Fig. 1A). Laboratory data showed mild ketoacidosis with a slightly high blood glucose level (215 mg/dL) and hyperthyroidism (TSH <0.05 mIU/L, fT3 3.37 pg/mL, fT4 2.82 ng/dL) (Table 1). Furthermore, based on the antibody test results showing positivity for islet cell antibodies and thyroid autoantibodies, he was diagnosed with APS3, accompanied by type 1 diabetes and Graves' disease (Table 1).

Ultrasonography (US) of the thyroid showed diffuse goiter with hypervascularity (Fig. 1B). Abdominal dynamic computed tomography (CT) revealed marked dilation of the stomach and fluid retention up to the horizontal leg of the duodenum (Fig. 2). Furthermore, abdominal US revealed an SMA bifurcation angle of 22° and an SMA aortic distance of 7 mm, values that were lower than those in healthy subjects (Fig. 3). Given these findings, we established a clinical diagnosis of SMAS accompanied by APS3A.

Continuous insulin infusion improved ketosis, and the symptoms of thyrotoxicosis improved promptly with thiamazole. Only extracellular fluid infusion (1,500 mL) was given on the first and second days of hospitalization. At three days after admission, he started to eat food while avoiding the forward bending position, since the stenotic part of the SMA was prone to open in that position. With these conservative treatments, he was able to resume his dietary regimen, and his general condition gradually improved (Fig. 4). He was discharged on the 22nd day of hospitalization.

Discussion

APS is a syndrome that induces organ dysfunction from a combination of multiple endocrine diseases caused by autoimmunity (1). Organ-specific autoantibodies to multiple endocrine glands are observed in APS; in addition, such autoimmune diseases are also associated with the digestive tract, liver, skin, and nervous system (10). APS3 has the highest frequency among the APS types and is characterized by the absence of autoimmune Addison's disease (2). There are various combinations of diseases in APS3; however, among them, APS3A, characterized by the combination of autoimmune thyroid disease and type 1 diabetes, has a high morbidity rate. Furthermore, APS3A is reported to be accompa-

Table 1. Laboratory Data on Admission.

	Clinical values (normal range)		Clinical values (normal range)		Clinical values (normal range)
Complete blood count		Blood chemistry (continued)		Diabetes	
WBC (μL)	9,900 (4,000-9,600)	sFe ($\mu\text{g/dL}$)	77 (94-178)	Blood glucose (mg/dL)	215 (80-110)
Neutrophil (%)	83.4 (40-69)	UIBC ($\mu\text{g/dL}$)	95 (120-250)	HbA1c (NGSP)(%)	14.0 (4.6-6.2)
Eosinophil (%)	0.1 (0-5)	Ferritin ($\mu\text{g/dL}$)	429.7 (40.7-335.8)	Urinary ketone	3+ (-)
Basophil (%)	0.1 (0-2)	Serology		Total blood ketone bodies ($\mu\text{mol/L}$)	1,930 (26-122)
Monocyte (%)	3.7 (3-9)	CRP (mg/dL)	2.17 (<0.1)	Acetoacetic acid ($\mu\text{mol/L}$)	477 (13-69)
Lymphocyte (%)	12.7 (26-46)	Anti-intrinsic factor antibody	(-)	3 hydroxybutyric acid ($\mu\text{mol/L}$)	1,450 (0-76)
RBC ($10^6/\mu\text{L}$)	6.34 (4.0-5.0)	Anti-smooth muscle antibody	(-)	pH	7.239
Hb (g/dL)	16.9 (11.8-15.1)	HLA haplotypes	DRB1*09:01:02- DQB1*03:03:02- DQA1*03:02	HCO ₃ ⁻ (mEq/L)	13.5
Hct (%)	47.7 (35.0-45.0)			Serum C-peptide (ng/mL)	0.58 (1.1-3.3)
Platelets $\times 10^4$ (μL)	25.5 (16-35)			Urinary C-peptide ($\mu\text{g/day}$)	31.24 (22.8-155.2)
Blood chemistry		Urinalysis		Anti-GAD antibody (U/mL)	>2,000 (<5.0)
Total protein (g/dL)	6.4 (6.3-7.9)	Color	Yellow	Anti-IA-2 antibody (U/mL)	9.6 (<0.6)
Albumin (g/dL)	3.8 (3.9-5.0)	Specific gravity	1.030 (1.005-1.030)	Anti-ZnT8 antibody (U/mL)	512 (<15.0)
Total bilirubin (mg/dL)	1.3 (0.3-1.2)	pH	5.0 (5.0-8.5)	Insulin antibody concentration (nU/mL)	699 (<125)
AST (U/L)	38 (13-33)	Protein	- (-)	Insulin antibody binding rate (%)	2.2 (<0.4)
ALT (U/L)	18 (6-27)	Glucose	4+ (-)	Thyroid	
LDH (U/L)	124 (119-229)	Ketone	3+ (-)	TSH (mIU/L)	<0.05 (0.35-4.94)
ALP (U/L)	556 (115-359)	Urobilinogen	+/- (-)	Free T ₃ (pg/mL)	3.37 (1.88-3.18)
AMY (U/L)	19 (49-136)	Bilirubin	- (-)	Free T ₄ (ng/dL)	2.82 (0.70-1.48)
CPK (U/L)	25 (24-195)	Nitrite	- (-)	Thyroglobulin (ng/mL)	50.84 (0-32.6)
Blood urea nitrogen (mg/dL)	20.1 (8-20)	WBC	- (-)	TRAb (IU/L)	9.4 (0-1.99)
Creatinine (mg/dL)	0.54 (0.46-0.79)	blood	- (-)	TSAb (%)	207 (\leq 120)
Na (mEq/L)	131 (137-145)			Anti-thyroglobulin antibody	<100 (<100)
K (mEq/L)	5.7 (3.5-4.8)			Anti-microsome antibody	6,400 (<100)
Cl (mEq/L)	90 (100-107)			Anti-thyroid peroxidase antibody (IU/mL)	253.2 (<16.0)
Ca (mg/dL)	9.4 (8.0-10.0)				
IP (mg/dL)	6.6 (2.5-4.5)				
T-Cho (mg/dL)	125 (128-219)				
LDL-Cho (mg/dL)	49 (70-139)				
HDL-Cho (mg/dL)	43 (40-)				
TG (mg/dL)	90 (0-149)				

WBC: white blood cells, RBC: red blood cells, Hb: hemoglobin, Hct: hematocrit, AST: aspartate aminotransferase, ALT: alanine transferase, LDH: lactate dehydrogenase, ALP: alkaline phosphatase, AMY: amylase, CPK: creatine phosphokinase, IP: ionized phosphate, T-Cho: total cholesterol, LDL-Cho: low-density lipoprotein cholesterol, HDL-Cho: high-density lipoprotein cholesterol, TG: triglycerides, sFe: serum ferritin, UIBC: unsaturated iron binding capacity, CRP: C-reactive protein, HLA: human leukocyte antigen, HbA1C: hemoglobin A1C, anti-GAD: anti-glutamate decarboxylase, anti-IA-2: anti-tyrosine phosphatase-related islet antigen 2, anti-ZnT8: anti-islet specific zinc transporter 8, TSH: thyroid stimulating hormone, T3: triiodothyronine, T4: tetraiodothyronine, TRAb: thyroid receptor antibodies, TSA: thyroid-stimulating antibodies

nied by skin symptoms, such as vitiligo, hair loss, gastrointestinal symptoms, and rheumatoid diseases (1, 11, 12). In this regard, our patient presented with type 1 diabetes, Graves' disease, and vitiligo vulgaris.

It has been reported that DRB1*04:05-DQB1*04:01, DRB1*08:02-DQB1*03:02, and DRB1*09:01-DQB1*03:03 are disease-sensitive human leukocyte antigen (HLA) haplotypes associated with type 1 diabetes among Japanese persons (13, 14). Among these HLA haplotypes, DRB1*04:05-DQB1*04:01 was associated with APS3A, and DRB1*09:01-DQB1*03:03 was not associated with APS3A (15). Furthermore, the frequency of HLA DRB1*04:05-DQA1*03:03-DQB1*04:01 and DRB1*08:02-DQA1*03:01-DQB1*03:02 in patients with APS3A, with both Graves' disease and type 1 diabetes, was reported to be significantly higher than

that in the control group (16). Interestingly, the haplotype in our case was DRB1*09:01:02-DQA1*03:02-DQB1*03:03:02, which was reported to be a disease-sensitive haplotype for type 1 diabetes but not for APS3A (Table 1).

SMAS is a disease in which the horizontal part of the duodenum is sandwiched between the SMA anteriorly and the spine or aorta posteriorly, causing obstructive duodenal symptoms, such as abdominal pain and vomiting (7). The most important causes of SMAS include decreased visceral fat due to malnutrition, hypercatabolism under highly invasive conditions, and anorexia nervosa (17). SMAS is reportedly caused by duodenal compression associated with the loss of mesenteric adipose tissue maintaining the angle and distance between the aorta and SMA. SMAS among adults is most often associated with severely debilitating diseases,

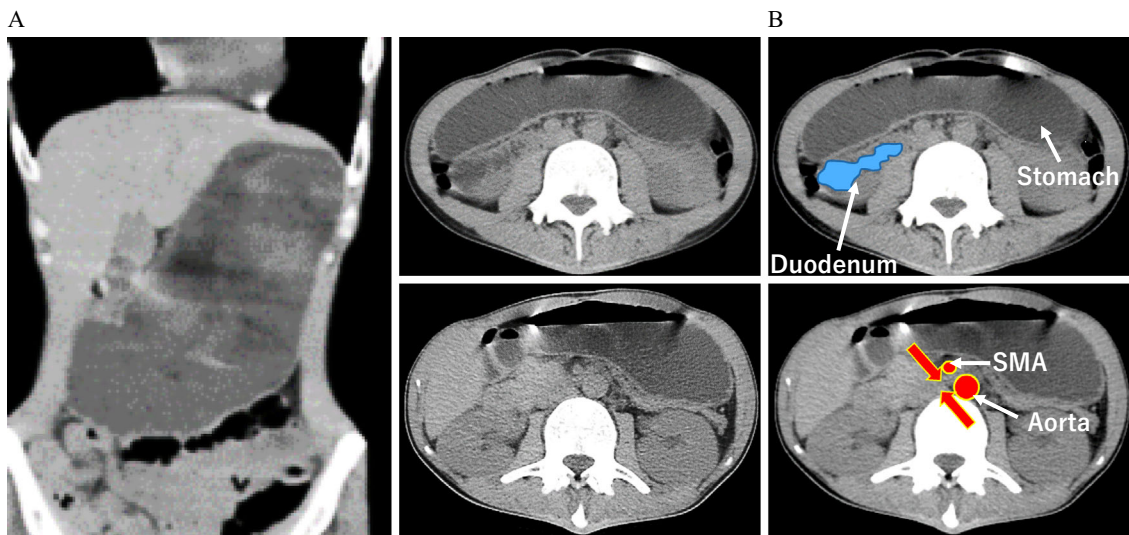


Figure 2. Abdominal dynamic computed tomography shows marked dilation of the stomach and fluid retention up to the horizontal leg of the duodenum.

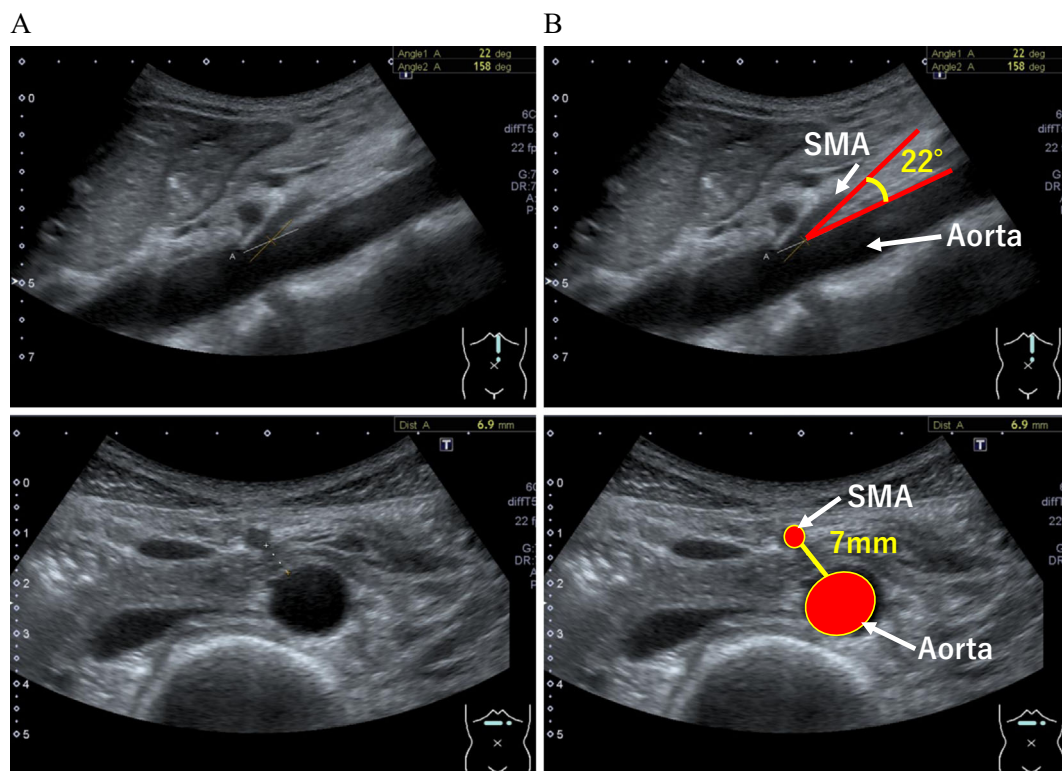


Figure 3. Abdominal ultrasonography reveals a superior mesenteric artery (SMA) bifurcation angle of 22° and SMA aortic distance of 7 mm, which is lower than that of healthy subjects.

such as malignancies, malabsorption syndromes, acquired immunodeficiency syndrome, trauma, and burns (18-20). To our knowledge, there are no diagnostic criteria for SMAS, and it is often a diagnosis of exclusion. Abdominal radiography and upper gastrointestinal angiography are imaging tests typically performed as initial investigations. Abdominal radiography reveals a double bubble sign characteristic of gastric bubble enlargement and duodenal obstruction, often nonspecific (7). Upper gastrointestinal angiography reveals a linear rupture of the horizontal duodenum, marked dilation

of the gastric to the descending duodenum, and stasis of the contrast medium (6, 21). Abdominal US, enhanced CT, and magnetic resonance imaging reveal a sharpening of the SMA bifurcation angle and a narrowing of the abdominal aorta-SMA distance (6). The imaging criteria in the diagnosis of SMAS include a distance between the abdominal aorta and SMA of 8 mm or less and an SMA bifurcation angle of $\leq 25^\circ$, which is the most sensitive indicator for a diagnosis (6). Our case report showed that the distance between the abdominal aorta and SMA was 7 mm, and the

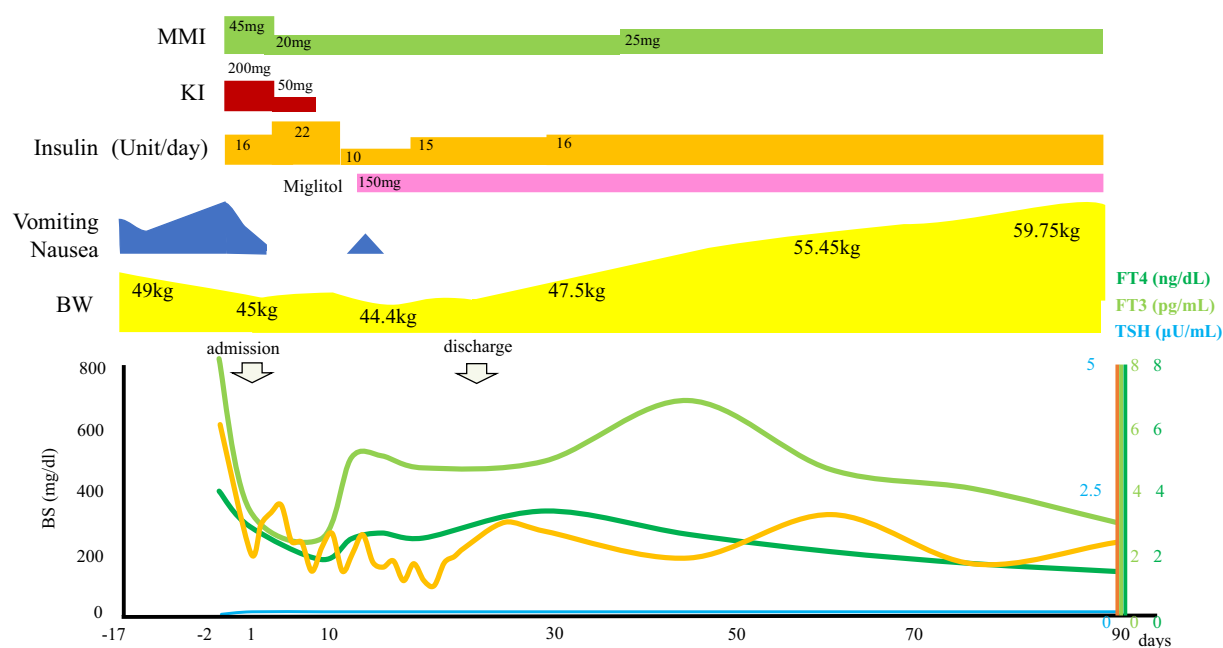


Figure 4. The whole clinical course of the patient. MMI: methimazole, KI: potassium iodide, BW: body weight, FT3: free triiodothyronine, FT4: free thyroxine, TSH: thyroid-stimulating hormone

Table 2. Clinical Description of Superior Mesenteric Artery Syndrome Associated with Diabetic Patients in the Literature.

Case	Age	Sex	Type of diabetes	Duration of diabetes	HbA1c (more recent)	Diabetic complications	Body weight loss	Comorbidities	Ref
1	28	F	Type 1	17 years	10 % over 10 years	Retinopathy Neuropathy	-16 kg (16 years old) -4 kg/2 months		25
2	65	M	Type 2	25 years	Unknown	Retinopathy Neuropathy	-50 kg (95→45)/27 years		25
3	18	F	Type 1	12 years	11.5 %	Albuminuria	-22.7 kg/unknown	Duodenojejunostomy	26
4	41	M	Type 2	4 years	11.4 %	Unknown	-26 kg/3 months		27
5	48	F	Type 1	13 years	15.3 %	Retinopathy Neuropathy Albuminuria	-15 kg (57→42)/6 months	Graves' disease (APS3 susp.)	9
6	58	F	Type 2	20 years	8-9 %	Retinopathy Neuropathy	-10 kg (40→30)/3 weeks		28
7	30s	M	Type 1	0 years	14 %	-	-4 kg (49→45)/2 weeks	APS3	Our case

SMA bifurcation angle was 22°, which aided in the diagnosis of SMAS. Conservative management, including decompression of the stomach and duodenum by inserting a nasogastric tube, correction of electrolyte imbalance, fluid resuscitation, and nutritional management, constitute the first line of management. Surgical treatment may be undertaken if conservative treatment results in failure or subsequent relapse (6, 22). In this regard, our patient gradually improved with conservative treatment, thus avoiding surgery.

The onset of SMAS is precipitated by a decrease in visceral fat, and weight loss can be a risk factor for SMAS (6). Our patient presented with marked weight loss (body mass index, 14.2 kg/m²) at the time of admission. When Graves' disease is complicated with APS3A, it causes further weight loss from hyperthyroidism, and 60.7% of affected individuals lose weight (23). Weight loss can also occur in patients

with type 1 diabetes (24). Furthermore, when diabetes is combined with Graves' disease, serious pathological conditions, such as ketoacidosis and thyroid storm, may occur. In our case, co-occurrence of type 1 diabetes and Graves' disease caused mutual exacerbation, leading to marked body weight loss and SMAS.

To date, six cases of SMAS associated with diabetes (three reports of type 1 diabetes and two reports of type 2 diabetes) have been reported (Table 2) (9, 25-28). Of note, a recently reported case of SMAS in a diabetic patient from weight loss due to SGLT2 inhibitors implied that body weight loss is a pivotal aspect for this pathology (28). It has been reported that SMAS is often misdiagnosed as diabetic gastroparesis. Three patients with SMAS had diabetic neuropathy and possibly co-existing diabetic gastroparesis, exacerbated by decreased peristalsis of the gastrointestinal tract.

More importantly, all reports had a relatively long history of diabetes. In the present case, our patient was diagnosed with diabetes and SMAS at the same time, and no diabetic neuropathy was observed. To our knowledge, this is the second reported case of APS3 resulting in SMAS; however, this is the first case of SMAS accompanied by Graves' disease and type 1 diabetes, which resulted in the extremely rapid evolution of disease without complications such as diabetic neuropathy. In particular, our case demonstrated that diabetic ketoacidosis fueled further weight loss and precipitated SMAS onset.

In summary, we presented the first case of SMAS caused by the acute onset of APS3A with type 1 diabetes and Graves' disease. It is crucial to consider the possibility of SMAS developing in cases of APS3A and rapid weight loss with gastrointestinal symptoms. Although SMAS has good prognosis, there are some cases in which emergency surgery is required for gastric perforation. A small number of patients die from consequent gastric perforation caused by SMAS and electrolyte abnormalities. Thus, a prompt diagnosis is required.

The principles outlined in the Declaration of Helsinki were followed, and the subjects provided their informed consent prior to relevant data collection and publication.

The authors state that they have no Conflict of Interest (COI).

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