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Case Report

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Complaint of Persistent Nausea Resulting with Superior Mesenteric Artery Syndrome Diagnosis: A Case Report

Hilal Aksoy, Seval Tunç, İzzet Fidancı, Duygu Ayhan Başer

Department of Family Medicine, Hacettepe University Faculty of Medicine, Ankara, Türkiye

ABSTRACT

Superior mesenteric artery syndrome (SMAS) is a rare syndrome that is generally seen in adolescents and young adults. In this case, a 22-year-old female patient applied to the outpatient clinic with complaints of persistent nausea and vomiting. After evaluation of the physical and laboratory findings of the patient, contrast-enhanced abdominal computed tomography was requested. The findings were considered as SMAS and the patient was referred to the gastroenterology clinic.

Keywords: Duodenum, gastrointestinal tract, superior mesenteric artery syndrome, Wilkie's syndrome

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Address for correspondence: Dr. Hilal Aksoy. Department of Family Medicine, Hacettepe University Faculty of Medicine, Ankara, Türkiye

Phone: +90 533 337 71 12

E-mail: hilal.aksoy35@gmail.com

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INTRODUCTION

Superior mesenteric artery syndrome (SMAS), also known as Wilkie's syndrome, is a rare clinical phenomenon that occurs as a result of compression of the third part of the duodenum between the aorta and the superior mesenteric artery.[1] The prevalence of SMAS was estimated at around 0.013–0.3% and it is seen 2 times more frequently in women than in men.[2] It is generally seen in adolescents and young adults between the ages of 10 and 39.[3] Patients have gastrointestinal system symptoms such as early abdominal satiety, nausea, bilious vomiting, bloating, postprandial abdominal pain, and weight loss. Since the symptoms are non-specific, they can be confused with other gastrointestinal pathologies such as ileus, gastroesophageal reflux, and pancreatitis.[4] In this case report, a case of SMAS detected in a patient who applied to a university hospital family medicine outpatient clinic with a complaint of nausea was evaluated.

CASE REPORT

A 22-year-old female patient applied to the outpatient clinic with complaints of persistent nausea and vomiting for 6 months. When the patient's anamnesis was deepened, it was learned that nausea had been present for a long time but had worsened in the past 6 months and did not regress with antiemetics. Postprandial bilious vomiting had occurred at least 4 days a week. The patient had cramp-like pain in the left upper quadrant which was relieved when the patient pressed this area with her hand. The patient had no diarrhea or constipation and also stated that she had been at the same weight for a long time and could not gain weight. The patient, whose last menstrual period was 5 days ago, did not have a known disease and regular medication use. The patient had no past medical or surgical history. There was no feature in her family history.

Physical examination of the patient was weight 49 kg, height 168 cm, and body mass index (BMI) 17.36 kg/m^2 and vital signs were fever 36°C, pulse 80/min, and blood pressure 112/78 mmHg. In the abdominal examination, bowel sounds were normoactive, and there was no tenderness, defense, or rebound. No organomegaly was detected. Other system examinations were normal. Laboratory tests of the patient at the time of admission are summarized in Table 1.

Contrast-enhanced abdominal computed tomography (CT) was requested in terms of pancreatobiliary pathologies due to the presence of amylase elevation and nausea-vomiting symptoms in the patient. The abdominal CT scan of the patient is shown in Figure 1. It was reported as "aorto-mesenteric angle: 20° (normal: 28°–65°), aorto-mesenteric distance: 6 mm (normal: 10–28 mm), subcutaneous and intra-abdominal fat tissue decreased and these signs are important for SMAS." Due to the clinical and imaging findings of the patient, SMAS was considered and the patient was referred to the gastroenterology clinic. Afterward, it was learned that the patient was given treatments for her symptoms by the gastroenterology department, and the patient was referred to the general surgery clinic for the operation, but the patient did not accept surgery.

Table 1. Laboratory tests of the patient at the time of admission

	Results	Normal Range
Sodium (mEq/L)	140	136-146
Potassium (mEq/L)	4.5	3.5-5.1
Chlorine (mEq/L)	106	101-109
Fasting glucose (mg/dL)	79	70-100
Creatinine (mg/dL)	0.65	0.51-0.95
Urea nitrogen (mg/dL)	6.3	6-20
Estimated glomerular filtration rate (mL/min)	>60	>60
Albumin (g/dL)	4.86	3.5-5.2
Alanine aminotransferase (U/L)	16	<35
Aspartate transaminase (U/L)	18	<35
Alkaline phosphatase (U/L)	60	30-120
Gamagglutamyl transferase (U/L)	13	<38
Total bilirubin (mg/dL)	0.62	0-0.2
Direct bilirubin (mg/dL)	0.47	0-1.2
Amylase (U/L)	209	28-100
Triglyceride (mg/dL)	55	<150
Total cholesterol (mg/dL)	121	<200
HDL cholesterol (mg/dL)	43	>50
LDL cholesterol (mg/dL)	72	<130
VLDL cholesterol (mg/dL)	11	<40
Total calcium (mg/dL)	10.04	8.8-10.6
Inorganic phosphorus (mg/dL)	3.66	2.5-4.5
Ferritin (µg/L)	16	11-307
Erythrocyte (10^6/µl)	4.88	3.83-5.08
Hemoglobin (gr/dL)	12.7	11.7-15.5
Hematocrite (%)	39.1	34.5-46.3
Leukocyte (10^3/µl)	9.2	4.1-11.2
Platelet (10^3/µl)	338	159-388

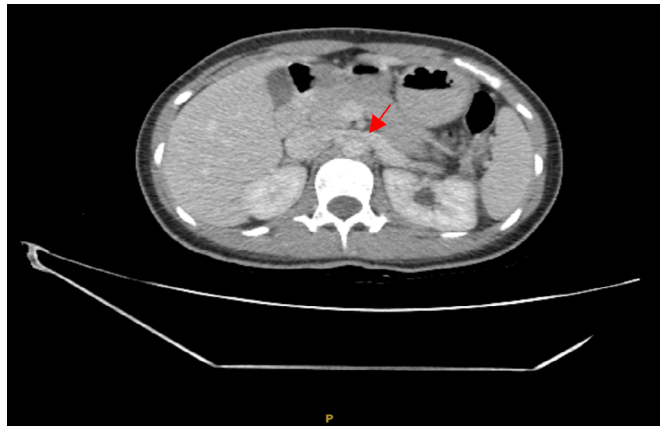

Figure 1. Abdominal computed tomography scan of the patient.

DISCUSSION

Although SMAS is rare in the community, it is an entity that should be kept in mind in the differential diagnosis of patients with persistent nausea and vomiting.[5] This patient had these symptoms for a long time. Abdominal pain seen in SMAS is usually localized above the umbilicus and to the left, and the patient is relieved when pressure is applied. In this case, the patient stated that her pain was relieved when she applied pressure to the left upper quadrant with his hand. Plain abdominal radiography, barium upper gastrointestinal system radiography, CT, CT angiography, magnetic resonance angiography, ultrasonography, and endoscopy are helpful images in the diagnosis of SMAS.[2] Abdominal CT to rule out pancreatobiliary diseases was evaluated. Aorto-mesenteric angle and aorto-mesenteric distance were decreased in abdominal CT as expected in SMAS. Although the etiology of the disease is not known exactly, the causes may be acquired or congenital, and no cause was found in 40.4% of the cases.[6] While anatomical variations such as the shortness of the ligament of Treitz are among congenital causes, the acquired causes include cerebral palsy, cachexia, extensive burns, dietary disorders such as anorexia nervosa leading to severe weight loss, post-operative conditions such as bariatric surgery, and surgical correction of scoliosis.[7]

In this case, abdominal CT revealed decreased subcutaneous and intra-abdominal adipose tissue. Retroperitoneal adipose tissue and lymphatic tissue act as a cushion under the superior mesenteric artery, keeping it away from the vertebral column and thus preventing the duodenum from being compressed between the aorta and the SMA.[8] Patients with SMAS usually have a low BMI. In a case series of 80 patients performed by Lee et al., the mean BMI of the patients was found to be 17.4 kg/m^2.[9] Welsch et al. suggested that individuals with low BMI or who have lost weight are more prone to SMAS.[2] The case was underweight too. In the treatment of acute cases, conservative treatment is applied. The target of treatment is to help the patient gain weight and to restore the loss of fatty tissue pad, which is thought to cause narrowing of the aorto-mesenteric angle. Surgical treatment is applied in cases whose complaints persist for a long time and do not respond to conservative treatments.[10]

CONCLUSION

A family physician should have a comprehensive approach to the patient. The patient should constantly monitor the patient's complaints and be able to resolve the underlying condition by making a more detailed evaluation in cases where there is no improvement in the complaints.

Disclosures

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