

## Point-by point response to the Reviewers' comments

### Reviewer #1:

This is a well-written review about the Superior Mesenteric Artery Syndrome (SMAS). The author involved all the relevant topics and also included personal radiologic imaging with endoscopic imaging. Furthermore, the drafting imaging of the SMAS is extremely well described. The main flaw about this syndrome is the lack of relevant data about the right timing for the surgical approach after conservative treatment fail, which was well described in the text.

> Thank you so much for reviewing our manuscript and your kind words. We hope this article can help clinical managements of SMAS.

### Reviewer #2:

What is the meaning of the word " FD" that appears on the page 3, line 85 ?

> We appreciate your careful review and your kind comment. According to your comment, we would define FD as functional dyspepsia and state that acronym has been expanded on the revision (page 3, line 86).

### Reviewer #3:

1) Would you please kindly correct all your typos and grammar errors throughout the manuscript.

> Thank you so much for your review and thoughtful comments. According to your suggestion, we corrected all our typos and grammar errors by a native English speaker (Dr. Awoniyi). For major changes, we highlighted changes with yellow color.

2) I would generally suggest to elaborate on more information about possible therapy including surgery. It is well-known that there is no relevant guidelines but it might be nice to gather all the information that might be of importance.

> According to your suggestions, we added more information about possible therapy including surgery. Please see our modifications highlighted by yellow color in the text and the additional table (Table 6). Again thank you so much for your great suggestions.

*Opening page:*

## **Superior Mesenteric Artery Syndrome: Diagnosis and Management**

Oka A et al. SMA syndrome

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31

32 **Abstract**

33 Superior mesenteric artery syndrome (SMAS, also known as Wilkie's syndrome, cast syndrome,  
 34 or aorto-mesenteric compass syndrome) is an obstruction of the duodenum caused by extrinsic  
 35 compression between the superior mesenteric artery (SMA) and the aorta. The median age of  
 36 patients is 23 years old (range 0-91 years old) and predominant in females over males with a  
 37 ratio of 3:2. The symptoms are variable, consisting of postprandial abdominal pain, nausea and  
 38 vomiting, early satiety, anorexia, and weight loss and can mimic anorexia nervosa or functional  
 39 dyspepsia. Because recurrent vomiting leads to aspiration pneumonia or respiratory depression  
 40 via metabolic alkalosis, early diagnosis is required. The useful diagnostic modalities are  
 41 computed tomography as a standard tool and ultrasonography, which has advantages in safety  
 42 and capability of real-time assessments of SMA mobility and duodenum passage. The initial  
 43 treatment is usually conservative, including postural change, gastroduodenal decompression,  
 44 and nutrient management (success rates: 70-80%). If conservative therapy fails, surgical  
 45 treatment (i.e. laparoscopic duodenojejunostomy) is recommended (success rates: 80-100%).

46

47 **Key words:** Superior mesenteric artery syndrome, Wilkie's syndrome, Cast syndrome, Aorto-  
 48 mesenteric compass syndrome.

49

50 **Core tip:**

51 To summarize, SMA syndrome (SMAS) is a rare condition that occurs when the superior  
 52 mesenteric artery compresses the third part of the duodenum. This compression can cause  
 53 obstructive symptoms and weight loss. SMAS can be caused by a variety of factors, including  
 54 abnormal anatomy, rapid weight loss, and previous abdominal surgery. It is most commonly  
 55 seen in young, thin females, but can occur in males and people of any age or body type.  
 56 Treatment may involve dietary modifications, medications, and surgery to correct the  
 57 underlying cause. If left untreated, SMAS can lead to serious complications, including  
 58 malnutrition and intestinal damage.

## INTRODUCTION

Superior mesenteric artery (SMA) syndrome is a rare cause of duodenal obstruction by extrinsic compression between SMA and the aorta (SMA-Ao) (**Figure 1**), and a morbid entity when the diagnosis is delayed [1-3]. Von Rokitsansky first described this entity in his textbook with a case presentation in 1842 [1]. Later, Wilkie described pathological and diagnostic findings in details with 75 cases of his own in 1927 [2], thus SMA syndrome is also known as Wilkie's syndrome. The other names are reported as cast syndrome, aorto-mesenteric compass syndrome, or mesenteric duodenal obstruction [3]. A cast, which is used to treat certain congenital deformities such as scoliosis and hip displacement, is a major cause of SMA syndrome [4-6]. By the year 2022, more than 730 articles with approximately 2,400 cases of SMA syndrome had been reported (author's review in PubMed [7]). Initial conservative therapy occasionally fails, and surgical or, more recently, endoscopic surgical duodenojejunostomy is successfully performed. This review provides clinical information of SMA syndrome in details.

## EPIDEMIOLOGY AND ETIOLOGY

SMA syndrome frequently concerns young adult female [2,8-13]. Our review [7] found the median age is 23 years old (inter-quartile range [IQR]: 16-39) but any age can be affected (range 0-91 years old), with increasing trends of elder patients with SMA syndrome in recent literatures (**Figure 2**). The affected age seems to be related to underlying patients' conditions (i.e. congenital scoliosis at children or weight loss due to chemotherapy). Whereas the affected gender is, as reported previously, predominant in females over males with a ratio of 3:2 [7]. The incidence of SMA syndrome in the general population has been estimated at 0.013-0.78% based on radiographic studies [8,14-17], though an accurate prevalence of the disease is unknown, depending on under- or over-diagnosis in clinical practice and each patient's condition [11,18] (**Table 1**) [13,19-35]. A prospective case-control study conducted by Xu et al. found the incidence of SMA syndrome is 2.67% (26/973 admissions for 9 years) [28]. Scoliosis and burn injuries are well-known etiologies of SMA syndrome and clinicians should be aware of this entity. In contrast, in **functional dyspepsia** patients, the incidence is much higher (10.8%) than in the general population, which is explained by clinicians under-diagnosing.

## PATHOPHYSIOLOGY

Decreasing SMA-Ao angle causes compression to the third part of the duodenum (see DIAGNOSIS). Decreases in the SMA-Ao angle can be either congenital or acquired (**Table 2**) [2,4-6,18-20,23,26,31,34-61]. The major causes of SMA syndrome involve body weight loss and resulting loss of mesenteric fat tissue between SMA-Ao, which in turn, makes a narrower angle between the vessels. The reasons for weight loss include several types of dietary conditions (eating disorders and malabsorptive diseases), hypermetabolism (drugs and burns), and cachexia causing conditions (tuberculosis and malignancy). Especially in severe injuries and burns, prolonged bedrest in a supine position increases risk of compression of the duodenum. Scoliosis treatments (surgery and cast) and scoliosis itself are well-known causes of SMA syndrome. The lengthening of the spine during scoliosis surgery is thought to be the underlying pathophysiology. Intestinal surgeries including ileal pouch-anal anastomosis and colectomy are also well-described causes, reducing the SMA-Ao angle due to pulling on the mesentery. Congenitally short or hypertrophic ligament of Treitz is a major cause in children.

Although it is not pure SMA syndrome, aortic artery aneurysm (AAA) and surgery near or around the SMA and 3rd duodenum induces “pseudo-” SMA syndrome. AAA-related SMA syndrome was first reported by Dr. Osler as aortoduodenal syndrome [58-60].

## SYMPTOMS

Patients with SMA syndrome suffer from vague and nonspecific symptoms, such as nausea, vomiting, epigastric pain, early satiety and post-prandial discomfort, bloating (abdominal distension), and weight loss, which can mimic anorexia nervosa and functional dyspepsia [9,13,62]. The epigastric pain and discomfort are more severe in a supine position and relieved in the lateral decubitus position (positioning knees to the chest) which reduces tension on the small bowel mesentery [62]. Especially in acute phase, severe duodenal obstruction leads to severe symptoms and life-threatening dilatation of the stomach [9,13,62]. In contrast, in chronic phase, the recurrent nausea and vomiting leads to inadequate food intake, resulting in severe weight loss and thus, aggravation of the syndrome [9,13,62].

## COMPLICATIONS AND COMORBIDITIES

Various complications of SMA syndrome have been reported (**Table 3A**) [36,63–87]. Notably, unrecognized or severe cases may progress to life-threatening complications, such as hypovolemic shock, aspiration pneumonia, and sudden death, even in young patients. Mechanisms of sudden death remain unclear, and several hypotheses, however, can be raised based on published cases including autopsies - arrhythmia by severe hypokalemia, severe compression of the inferior vena cava by dilated duodenum, or severe pulmonary depression induced by alkalosis and increased abdominal pressure. Thus, immediate corrections of blood election and volume and early reduction of intestinal pressure should be required in severe cases. The most frequent complication is gastrointestinal injury caused by retained or refluxed peptic acid and bile acid as well as elevated intraluminal pressure. The incidence of mucosal injury has been reported as 25-59% in patients with SMA syndrome [63,74]. Inadequately treated or chronic mucosal injuries may progress to emphysema, necrosis, portal venous gas, and pneumoperitoneum. Elevated intraluminal pressure at the second portion of the duodenum disturbs the flow of pancreatic juice, occasionally resulting in elevated pancreatic enzymes and acute pancreatitis. Vomiting itself can increase serum amylase (mainly from salivary glands), so pancreatic amylase isozyme and lipase should help to recognize pancreatic abnormalities. Recurrent vomiting also leads to aspiration pneumonia, dehydration, electrolyte abnormalities, and severe malnutrition. SMA syndrome sometimes co-exists with other vascular compression diseases (**Table 3B**) [88–91]. Of these, nutcracker phenomenon is the most frequent based on anatomic location to the SMA. It is a condition that occurs when the left renal vein becomes compressed between the aorta and SMA with similar symptoms as SMA syndrome.

## DIAGNOSIS

Due to its non-specific symptoms, SMA syndrome might be overlooked in clinical practice [27,92]. Even in radiologists, the duodenum seems to be a neglected segment in the intestine [93]. SMA syndrome requires a high degree of clinical suspicion and few teaching methods have been reported [94,95]. The diagnosis is based on clinical symptoms supported by radiological evidence of duodenal obstruction. Traditional criteria for SMA syndrome are based on barium X-ray studies (**Figure 3**): 1) dilatation of the first and second parts of the duodenum with or without

148 gastric dilatation, 2) abrupt vertical or oblique compression of the third part of the duodenum,  
 149 3) reverse flow of contrast proximal to the obstruction, 4) significant delay (4-6 h) in  
 150 gastroduodenal transit, and 5) relief of obstruction after postural changes (the prone knee-chest  
 151 or lateral decubitus position) [18,19,96]. Recently, in addition to barium studies, various imaging  
 152 modalities have been used to confirm SMA syndrome, such as computed tomography (CT),  
 153 abdominal ultrasound (US), magnetic resonance imaging (MRI), endoscopy and endoscopic  
 154 ultrasonography (EUS) etc. (**Table 4**). Many cases are diagnosed by these modalities, which can  
 155 directly visualize the SMA compression of the duodenum without barium studies. The standard  
 156 diagnostic modality is CT scan (**Figure 4**) which allows for both diagnosis of SMA syndrome  
 157 with measurement of the SMA-Ao angle and distance as well as detection of complications, such  
 158 as gastric necrosis, portal vein gas, acute pancreatitis etc. 3D-CT is more helpful in recognizing  
 159 the anatomy of SMA, the aorta and duodenum [97]. The normal SMA-Ao angle is between 38 to  
 160 65 degrees and has a distance of 10 to 33 mm [5,98]. Unal et al. reported the cutoff value is 22  
 161 degrees on the SMA-Ao angle and 8 mm on a distance with a 42.8% sensitivity and 100%  
 162 specificity [98]. Abdominal US is another modality that provides a convenient, quick, noninvasive  
 163 tool to diagnose SMA syndrome [98-100]. The sensitivity of abdominal US in diagnosis of SMA  
 164 syndrome has been confirmed in a comparison study with CT findings [98]. Abdominal US  
 165 benefits from an improvement of image resolution and can clearly visualize the duodenum and  
 166 SMA-Ao angle (**Figure 5 and Video**). Endoscopy can detect gastrointestinal complications, such  
 167 as mucosal injury, bleeding, and bezoar, etc. (**Figures 6A and 6B**). It can also reveal extrinsic  
 168 compression (by SMA) at the 3<sup>rd</sup> portion of duodenum (**Figure 6C**). Further, EUS with mini-  
 169 probes can be used to confirm the compression by SMA and also measure SMA-Ao distance  
 170 [37,101]. Recently, linear EUS has been used for measuring the SMA-Ao angle and also endoscopic  
 171 duodenojejunostomy (see TREATMENTS). Laboratory tests are not diagnostic, but they are  
 172 necessary to identify the presence of electrolytic complications and pancreato-biliary  
 173 abnormalities. As for differential diagnosis, almost all disorders mimicking SMA syndrome are  
 174 summarized in **Table 5** [15,27,45,58-60,87,102-128]. Patients with eating disorders, functional dyspepsia,  
 175 and peptic ulcer disease present non-specific symptoms masquerading as SMA syndrome. In  
 176 addition, many diseases that potentially involve or compress duodenum should be suspected of  
 177 and ruled out by CT, abdominal US, or other modalities.

178

179 **TREATMENTS**

180 The therapeutic options are summarized in **Table 6**. The initial treatment is usually managed  
 181 conservatively by decompression of dilated stomach and duodenum by postural change and/or  
 182 nasal gastric tube suction [129,130]. Positioning the patient in the left lateral or sitting position  
 183 should be helpful [96,131]. However, the best position for each patient may vary because recent  
 184 studies revealed there is a variation of the SMA position and movement [132–134]. In addition to  
 185 gastric tube suction, intravenous metoclopramide can enhance gastrointestinal motility and help  
 186 decompression [135,136]. After decompression therapy, gaining weight to increase adipose tissue  
 187 between SMA and aorta should be considered. Nasal gastric feeding is effective, but the jejunal  
 188 tube is more ideal while endoscopic assistant should be considered [137]. Total parenteral  
 189 nutrition is a useful option for initial nutrient treatment and if the intestinal feedings are  
 190 impossible. These nutrition managements contribute to the restoration of adipose tissue to  
 191 increase the angle at the origin of the SMA [29].

192 Surgical therapy, however, can be recommended if conservative therapies fail especially  
 193 in elder patients with multiple abdominal operation histories, immobility (bed rest), long history  
 194 of SMA syndrome and arteriosclerosis of SMA. Surgical therapy might be considered earlier  
 195 before a patient's conditions worsen and complications occur [130]. The best timing for transition  
 196 to surgical options is not clear. Shin et al. recommend 6 weeks at least of conservative therapy  
 197 based on the average response rate to this method [138]. There is currently no randomized study  
 198 conservative versus surgical therapy. A recent large cohort with 80 patients with SMA syndrome  
 199 by Lee et al. revealed the overall success and recurrence rates of conservative therapy were 71.3  
 200 and 15.8%, respectively [139]. The need for surgical therapy was 18.7% of patients (15/80 cases),  
 201 which is similar to other recent cohorts 11.5–22.2% [9,140,141]. These recent operation rates are lower  
 202 than previously reported (70%) in 1974 [142], likely due to advances in nutritional therapies and  
 203 medications [139]. Various surgical procedures include laparoscopic, laparotomic, or robotic  
 204 gastrojejunostomy, gastroduodenostomy and duodenojejunostomy, Strong's procedure (a  
 205 division of the ligament of Treitz), anterior transposition of the third part of duodenum,  
 206 duodenal lowering, Ladd's procedure, and transabdominal duodenojejunostomy [143–147]. Since  
 207 1998, when the first successful laparoscopic duodenojejunostomy was performed, most



surgeons prefer laparoscopic duodenojejunostomy because of its safety and effectiveness (success rates: 80-100%) [143-145]. Laparoscopic approach has been reported to shorten post-operative length of hospital stay [130,144]. Most recently, a new technique of endoscopic gastrojejunostomy (so called lumen-apposing metal stent: LAMS) has also been reported in several case reports as a safe and effective therapeutic option [148-150].

## CONCLUSION

In conclusion, superior mesenteric syndrome is a serious condition that requires prompt diagnosis and treatment to prevent long-term complications. Diagnosis can be challenging and may involve imaging studies, such as CT or MR, and upper gastrointestinal endoscopy. Due to the non-specific nature of clinical obstructive presentations, recognition of risk factors such as rapid weight loss, previous abdominal surgery (typically bariatric surgery), trauma or congenital anomalies can predispose patients toward the development of SMA syndrome. These conditions are typically driven by a reduction in the mesenteric fat pad or an abnormal angle between the SMA and duodenum. Early diagnosis and treatment are essential to prevent complications and ensure a successful outcome.

## ACKNOWLEDGMENTS

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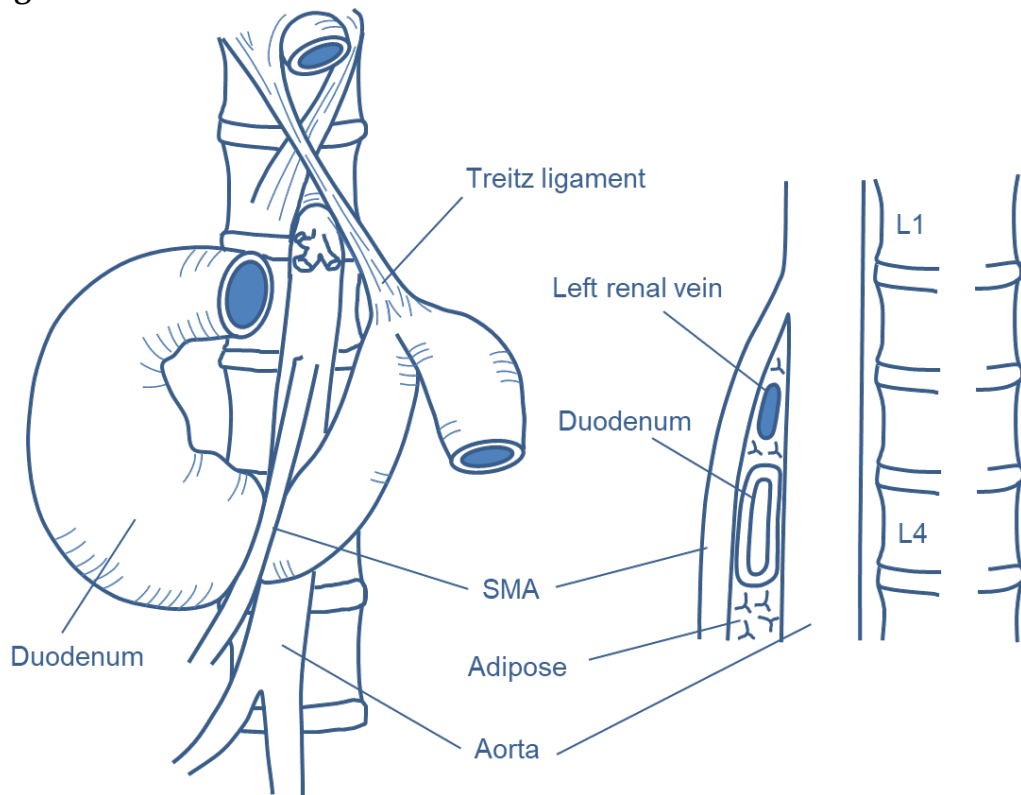
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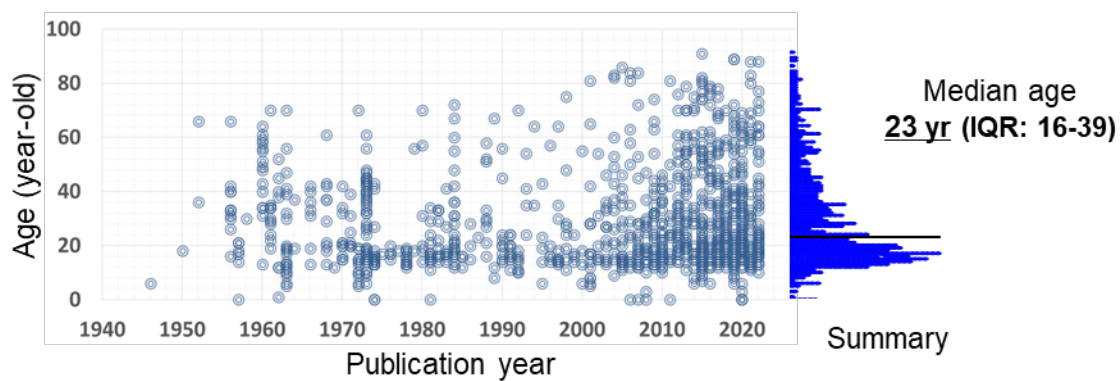


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## Figure Legends

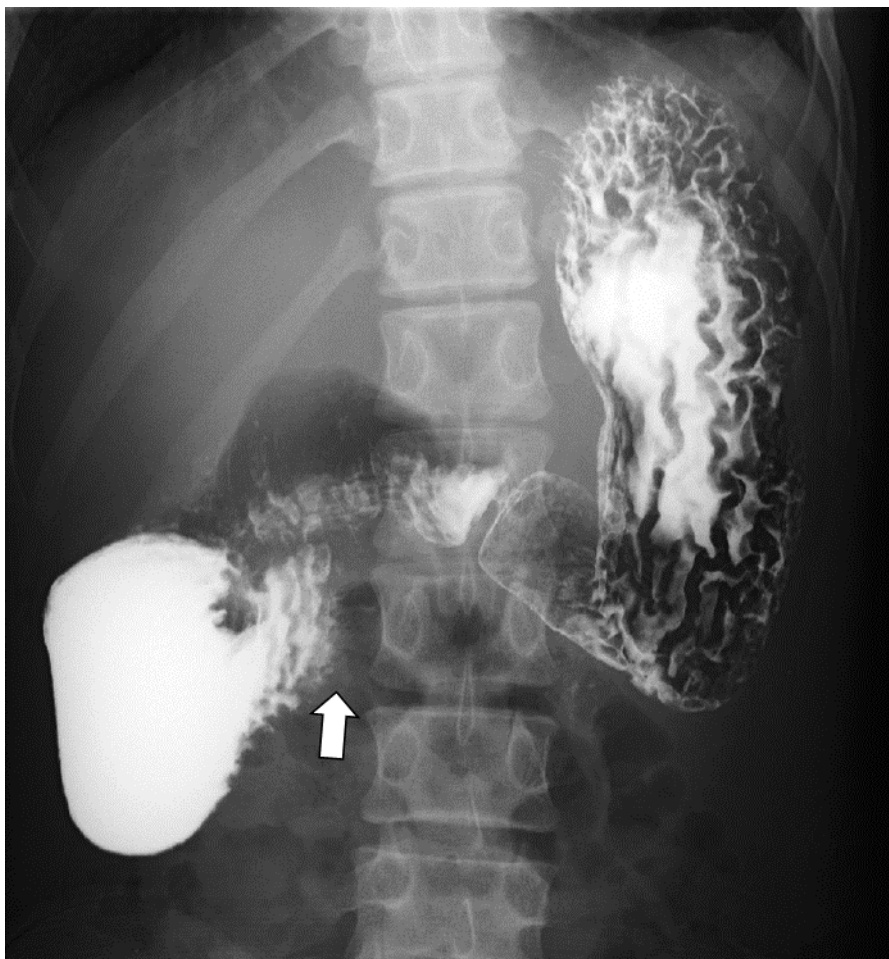


**Figure 1 Anatomy related to superior mesenteric artery syndrome.** SMA: superior mesenteric artery.

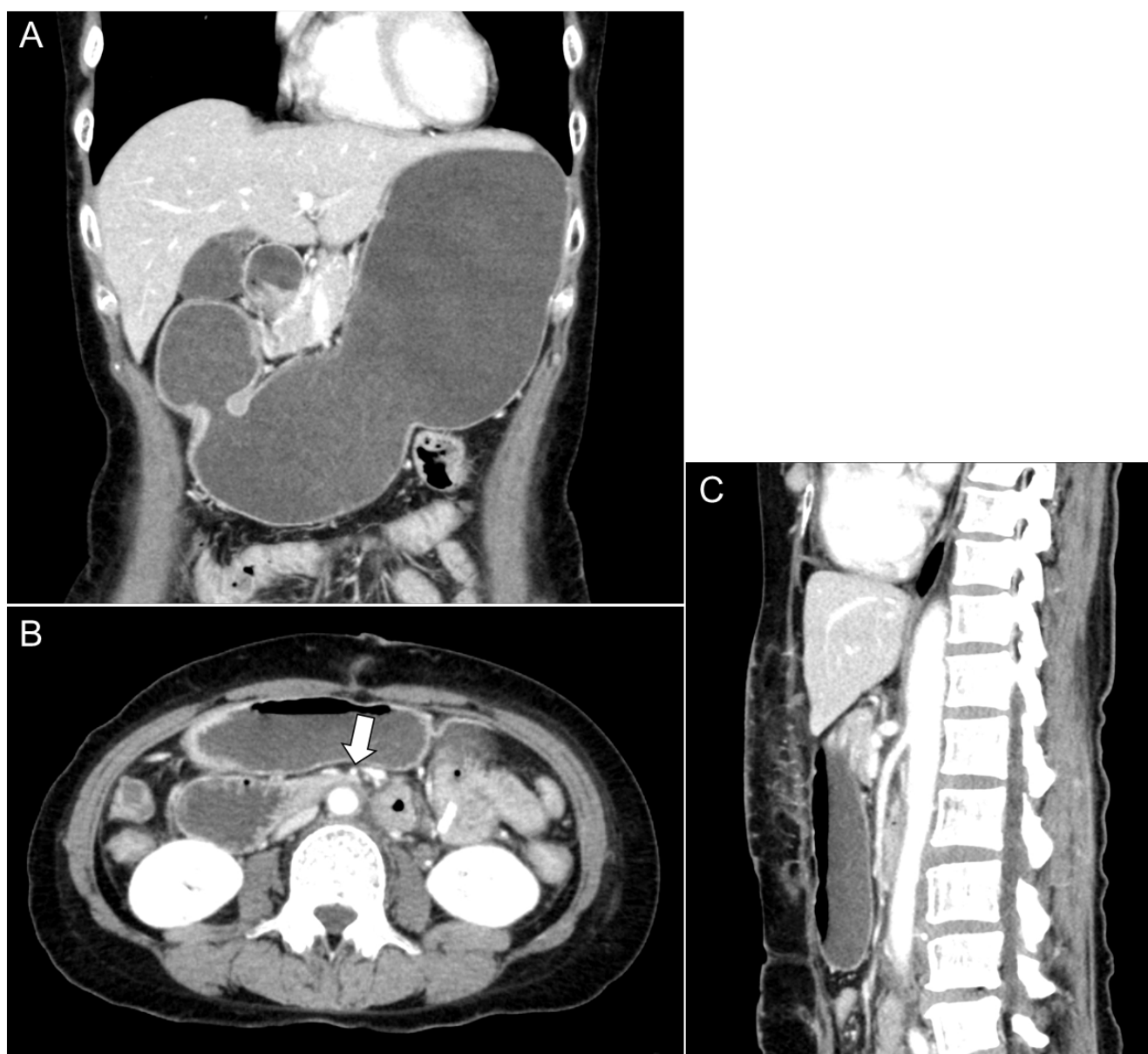


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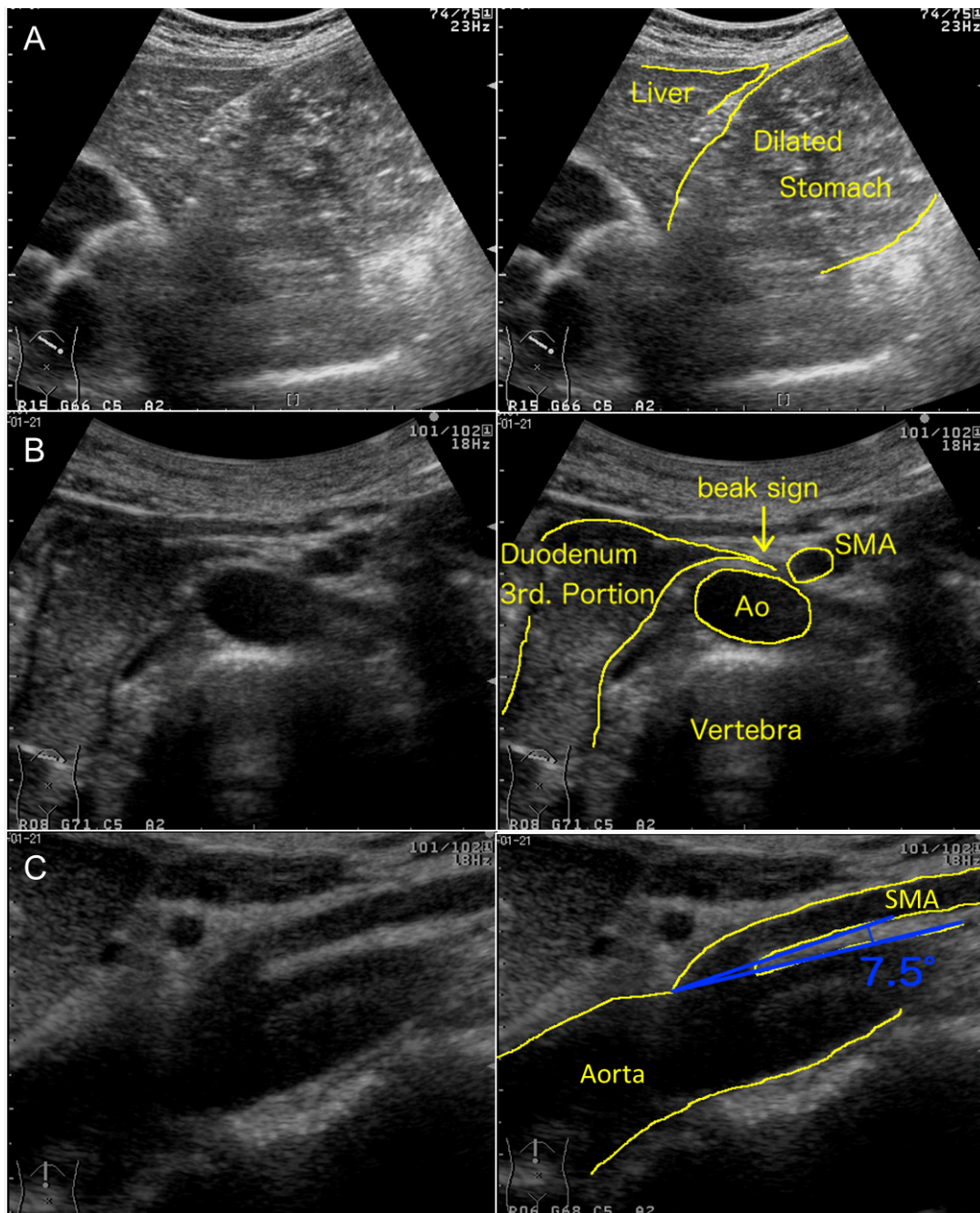
695 **Figure 2 Published patient age.** IQR: inter-quartile range. Dot indicates an individual case. The  
 696 data is based on our review [7].



697  
698 **Figure 3** Upper gastrointestinal series (barium X-ray) of a 16-year-old male with superior  
699 **mesenteric artery syndrome**. Arrow indicates obstructive compression of the third portion of  
700 the duodenum.  
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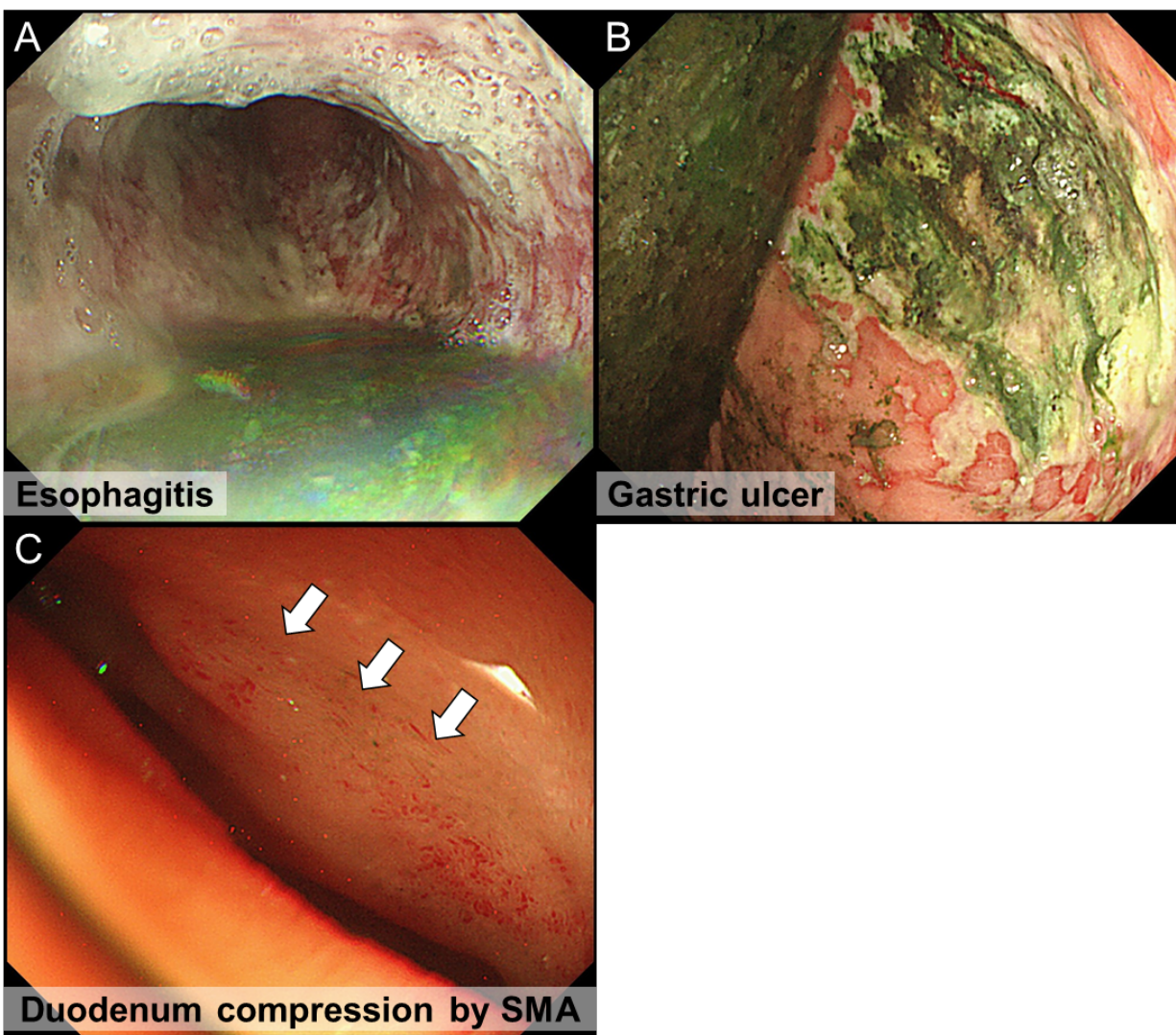


**Figure 4** Enhanced computed tomography images of a 56-year-old female with superior mesenteric artery syndrome. Computed tomography images (A: coronal, B: axial, C: sagittal) show a markedly distended stomach and proximal duodenum by extrinsic compression between the superior mesenteric artery (arrow in panel B) and aorta.



**Figure 5** Abdominal ultrasonographic images of a 53-year-old female with superior mesenteric artery syndrome. Upper abdominal ultrasonography shows a markedly dilated stomach (A) and obstruction of duodenum (which looks like beak, beak sign) by extrinsic compression between the superior mesenteric artery (SMA) and aorta (Ao). C: The SMA-Ao angle (7.5 degree) and distance (5 mm) are decreased.





715

716 **Figure 6 Endoscopic findings of patients with superior mesenteric artery syndrome.**

717 Esophagitis (A) and gastric ulcer (B) with retained luminal contents. C: compression area in the

718 third portion of the duodenum.

719

**TABLE 1. Incidence of SMA syndrome in several populations**

<b>Population</b>	<b>Incidence (%)</b>	<b>References</b>
Acute general hospitals	0.001-0.0052	19
Chronic-care hospital	0.097	19
Hospital admissions	0.05-2.67	13,28,29
Upper gastrointestinal endoscopy	0.48	30
Post-scoliosis surgery + cast	0.5-3.4	20,31-35
Spinal cord injury	0.53	22
Burn injury	1.0-1.12	23-25
Anorexia nervosa (admitted)	2.73	26
Functional dyspepsia	10.8	27
SMA: superior mesenteric artery		

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**TABLE 2. Etiology of SMA syndrome**

<b>Etiology</b>	<b>References</b>
Congenital	
Short or high insertion of Treitz ligament	61
Low origin of the SMA	36
Spinal deformity (Scoliosis, Marfan etc.)	37,38
Familial	39,40
Malrotation of SMA and SMV	41,42
Malrotation of intestine	2
Body weight loss	
Diet and obesity surgery (sleeve surgery)	5,43
Eating disorders (anorexia nervosa, anorexia bulimia)	26,37
Malabsorption	18
Malignancy	18,19,36
Tuberculosis	45
Chemotherapy	46,47
Trauma (Burn injury, brain injury, spinal cord injuries etc.)	23,48,49
Neural disorders (ALS, MELAS, paraplegia, cerebral palsy etc.)	50,51
Drug or alcohol abuse	37
Rheumatoid arthritis	52
Scoliosis surgery	20,31-35
Intestinal surgery (IPAA, colectomy etc.)	37,53,54
Aging (bed rest, frail, vascular calcification etc.)	55,56
Body cast	4-6
<hr/>	
<b>"Pseudo-" SMA syndrome</b>	
Aortic artery aneurysm (Aortoduodenal syndrome)	57-59
Surgery near or around the SMA and 3rd duodenum	60
SMA: superior mesenteric artery, SMV: superior mesenteric vein, ALS: amyotrophic lateral sclerosis, MELAS: mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes, IPAA: ileal pouch-anal anastomosis	

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723



**TABLE 3. Complications and comorbidities of SMA syndrome**

<b>A. Complications</b>		<b>References</b>
Gastrointestinal complications	25-59% in SMA syndrome	63,74
Esophageal inflammation, bleeding, and ulcer		61,81,82
Gastric inflammation, ulcer, emphysema, ischemia, necrosis, perforation, and bezoar		83-86
Duodenal inflammation, ulcer, mucosal necrosis, emphysema, and bezoar		64,65,87
Portal venous gas and thrombosis		66,67,85
Pneumoperitoneum and pneumomediastinum		68,69
Pancreato-biliary complications		
Elevated serum pancreatic and/or biliary enzymes		70
Acute and chronic pancreatitis		86
Jaundice		71
Pulmonary complications		
Aspiration pneumonia		72
Adult respiratory distress syndrome		73
Dehydration		
Low blood pressure		76
Acute kidney injury		76
Shock		76
Electrolytes and gas abnormalities		
Hypokalemia		77
Hyponatremia		77
Metabolic alkalosis		77
Severe malnutrition		78
Recurrent pregnancy loss		79
Sudden death		83
<b>B. Comorbidities related to SMA syndrome</b>		<b>References</b>
Nutcracker phenomenon		88,89
Celiac axis compression syndrome (Median arcuate ligament syndrome)		90,91
SMA: superior mesenteric artery		

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**TABLE 4. Diagnostic modalities for SMA synddrome**

<b>Modalities</b>	
	Plain film X-ray
	Barium X-ray
	Angiogram
	CT
	Plain
	Enhanced (3D-CT)
	Abdominal ultrasound
	B-mode
	Doppler-mode
	MRI
	MR angiography
	MR enterography
	Endoscopy
	White liight imaging
	Ultrasonography (EUS)
	Gastric-emptying scintigraphy
	Multi-channel manometory
SMA: superior mesenteric artery, CT: computed tomography, 3D: 3-demension, MRI: magnetic resonance imaging, EUS: endoscopic ultrasonography	

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727

**TABLE 5. Differential diagnoses of SMA syndrome**

<b>Disorders mimicking SMA syndrome</b>	<b>References</b>
<b>Similar symptoms by...</b>	
Eating disorder	
Anorexia nervosa, Anorexia bulimia	126
CIPO	127
Peptic ulcer disease	128
Reflux esophagitis	102
Functional dyspepsia	27
Cyclic vomiting syndrome	103
Pancreatitis	104
Gastric outlet obstruction	105
<b>Involvement of duodenum by... (other disorders)</b>	
Tubercular infection	106,107
Megaduodenum (localized CIPO)	108
Henoch-Schönlein purpura	109-111
Crohn's disease	112,113
Celiac disease	87
Ectopic pancreas	114
Duodenal diverticula	115
Duodenal edema	116
Tumor	
Primary or metastatic duodenal cancer, pancreatic cancer, lymphoma etc.	15,117
Anatomical abnormality (web, diaphragm)	118
Foreign body (bezoar etc.)	119
<b>Extrinsic compression by... (non-SMA)</b>	
Aortic artery aneurysm (Aortoduodenal syndrome)	57-59
Stent or filter	
Mesenteric artery, aorta, IVC etc.	120,121
Horseshoe kidney	122
Lymphnode	123
Abscess	124
Traumatic false aneurysm	125

SMA: superior mesenteric artery, CIPO: chronic idiopathic intestinal pseudo-obstruction, IVC: inferior vena cava

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**TABLE 6. Treatments for SMA syndrome**

<b>Therapeutic methods</b>	
Conservative therapy	
	Decompression of dilated stomach and duodenum by
	Postural change (left lateral, sitting position)
	Nasal gastric tube suction
	Medication (metoclopramide)
	Gaining weight by
	Giving multiple small feeds
	Feeding tube (nasal gastric or jejunal)
	Total parenteral nutrition
Surgical therapy	
	Anterior transposition of the third part of duodenum
	Gastroduodenostomy
	Gastrojejunostomy
	Duodenojejunostomy
	Strong's procedure (a division of the ligament of Treitz)
	Duodenal lowering
	Ladd's procedure
Endoscopic therapy	
	Lumen-apposing metal stent* by
	EUS-guided gastrojejunostomy
SMA: superior mesenteric artery, *LAMS: potential option based on case reports, EUS: endoscopic ultrasonography	

732 **Video Legends**

733 **Abdominal ultrasonographic images of a 53-year-old female with superior mesenteric artery**  
734 **syndrome.** Upper abdominal ultrasonography shows a markedly dilated stomach and proximal  
735 duodenum as well as obstructive compression of the third portion of the duodenum between  
736 the superior mesenteric artery and aorta.