Superior mesenteric artery syndrome: Myths and reality.

I. Introduction

Superior mesenteric artery syndrome (SMAS) is a rare condition causing functional obstruction of the third portion of the duodenum [1]. This disease was first described by Professor Carl von Rokitansky in 1842 [2]. In 1861, he was the first to observe that superior mesenteric vessels may compress and obstruct the duodenum over the lumbar spine. In 1927 Wilkie published the first comprehensive series of 75 patients [3]. Since then, some authors have called this disease Wilkie's syndrome. This pathology is found in 0.2% to 0.78% of patients examined with barium [4]. It is believed that partial obstruction of the duodenum is the result of its compression in the angle between the aorta and the superior mesenteric artery (SMA). B Normally, the aortomesenteric angle is 25–60° and the mean aortomesenteric distance is 10–28 mm. Subjects presenting with an angle <25° and aortomesenteric distance < 8-10 mm may be affected by the SMAS. According to this hypothesis, the retroperitoneal fat and lymphatic tissue push the mesenteric artery away from the aorta. It is believed that conditions reducing the distance and decreasing the angle between the SMA and aorta may contribute to the compression of the horizontal segment of the duodenum [5,6]. The typical symptoms of the SMAS are nausea, vomiting, abdominal pain, early satiety, postprandial fullness, and anorexia. Upper gastrointestinal studies show a dilated proximal duodenum with an abrupt termination of the barium column in the third portion. Angiography has been suggested as the “gold standard” procedure for the assessment of the aortomesenteric angle and distance between the vessels [7].

This syndrome can present itself as acute small bowel obstruction or intermittent compression with chronic symptomatology. The syndrome has been reported in association with pancreatitis, peptic ulcer, intra-abdominal inflammation, and cancer; against the background of abdominal muscle

hypertrophy, prolonged immobilization, severe burns, anorexia nervosa, severe weight loss, tuberculosis, acute gastroenteritis, spinal cord injury, and scoliosis repair surgery [5]. Numerous predisposing conditions for SMAS can be summarized into three categories: severe weight loss in catabolic states, external and intra-abdominal compression, or mesenteric tension [6].

Once radiologic studies have established a diagnosis, first-line treatment is usually conservative with jejunal or parenteral nutrition for the restoration of the aortomesenteric fatty tissue. If conservative management fails, surgical options include open or laparoscopic duodenojejunostomy or duodenal mobilization and division of the ligament of Treitz. The overall success rate of medical management in adults was 71.3% [8] and in pediatric patients to 86%.[9]

An analysis of the literature revealed the following facts that contradict the above-described concept of the pathogenesis of the SMAS. 1) Low body weight is not a determining factor in the pathogenesis of the SMAS since 23.7% [8] to 50% [9] of the patients have a normal body mass index (BMI). 2) In third world countries, there are hundreds of millions of people with low BMI which does not increase the SMAS frequency. 3) Bhagirath Desai et al. produced a prospective study of 100 patients who had undergone a CT scan for various other complaints. A strong positive correlation was found between BMI and the angle between the aorta and SMA. With BMI increase, the angle also increases. In 25% of patients, these rates were less than the norm, which indicates that the value of the aortomesenteric angle cannot serve as the gold standard [10].

II. Material & Methods.

I selected 7 articles describing 14 cases of SMAS that were published by PubMed and PMC in 2016-2020. in open access. The main selection criterion was the availability of high-quality images (radiographs, CT, MRI). Typically, the articles describe from 1 to 4 cases of successful diagnosis and treatment of SMAS.

After I published a letter to the editor, which provided evidence that duodenal obstruction occurs because of the impaired function of the Ochsner's sphincter [11], I have received several requests from patients suffering from SMAS, including from the co-founder of the SMAS nonprofit association in the USA. From reviews of the patients, I realized that in some cases the treatment is not remarkably effective. Before turning to the analysis, I will dwell on the description of the normal physiology of the duodenum (**Figure 1**).

III. The normal physiology of the duodenum

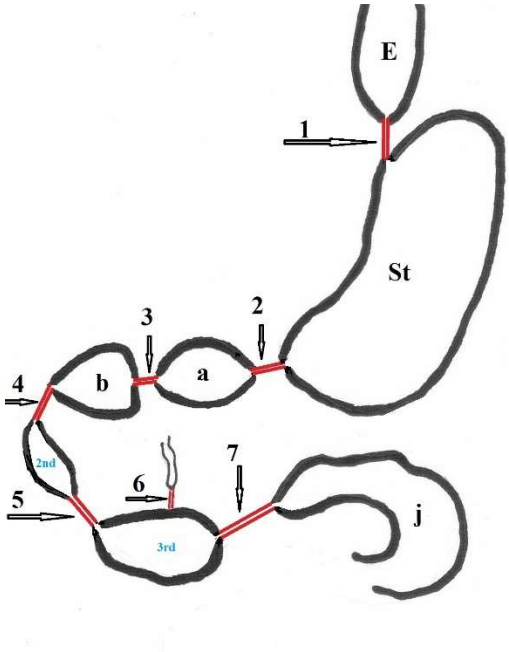


Figure 1. Scheme of gastroduodenal motility. Departments: e – esophagus; St – stomach; a- antral chamber; b – duodenal bulb (1st part of the duodenum); 2nd past; 3rd past of duodenum; j - jejunum. Sphincters: 1- lower esophageal sphincter; 2 – antral sphincter; 3 - pyloric sphincter; 4- bulbo-duodenal sphincter; 5 – sphincter of Kapandji; 6 – sphincter Oddi; 7 – sphincter of Ochsner.

Evacuation from the stomach occurs because of a contraction of the antrum. Each 3rd - 5th peristaltic wave closes in the antrum. The place where the wave completely closes the lumen of the stomach is called the antral sphincter. At this moment, a closed cavity arises between the antral and pyloric sphincters, which is called the antrum chamber. The advancement of the peristaltic wave leads to an increase in pressure in the antrum chamber. When it reaches a threshold value,

the pyloric sphincter opens, and a bolus of chyme is injected into the duodenal bulb. The length of the pyloric sphincter is 0.7 cm.[12]

When the bulb is filled with chyme, a contraction of the bulbo-duodenal sphincter occurs. This leads to an increase in pressure in the bulb, which causes a contraction of the pyloric sphincter and stops evacuation from the stomach. The volume of the antrum chamber is approximately equal to the volume of the duodenal bulb. In this way, the bulbo-duodenal sphincter provides the evacuation of the chyme from the stomach in portions of a certain volume.

After the closure of the pyloric sphincter, the bulbo-duodenal sphincter opens, and the bolus enters the 3rd part of the duodenum. In response to irritation of the intestine with low pH hydrochloric acid, a sphincter of Ochsner contracts. Its length is 3.2 ± 0.15 cm. This prevents the penetration of a chemically aggressive bolus into the jejunum. The bolus is discarded cranially, but the contraction of the Kapanji sphincter with a length of 2.05 ± 0.09 cm prevents the penetration of the bolus into the 2nd part of the duodenum. This sphincters reaction is repeated several times. So, there is a pendulum-like movement of a bolus in the intestine between these two sphincters. The acidic chyme also causes the secretion of secretin and cholecystokinin by the duodenal epithelium. These enzymes regulate the volume and rate of secretion of bile and pancreatic juice, which enter this part of the gut from the Vater's papilla. Thanks to contractions of these sphincters, the chyme mixes with digestive juices, and when the chyme's pH rises to a certain level, the Ochsner sphincter opens, passing a less aggressive bolus into the jejunum. Since there is no acid in barium suspension, in patients without pathology of the duodenum, the Kapandji and Ochsner sphincters are not contracted and not detected. In such cases, the true width of the duodenum is almost the same throughout and equal to about 2 cm. In patients with duodenal dyskinesia, these sphincters can be found. The 3rd part of the duodenum between these sphincters, as a rule, is wider than other parts. Its width reaches 3 cm.

IV. Duodenal dyskinesia

Dyskinesia of the duodenum is characterized by the impaired of its motor function, especially its sphincters. Strengthening the tone of the sphincters of Kapandji and Ochsner leads to a periodic increase in pressure in the third part of the duodenum between these sphincters, as evidenced by the expansion of the 3rd part of this gut and the appearance of primary diverticula, which are prolapse of the mucous through a weakened muscle layer (**Figure 2)**.

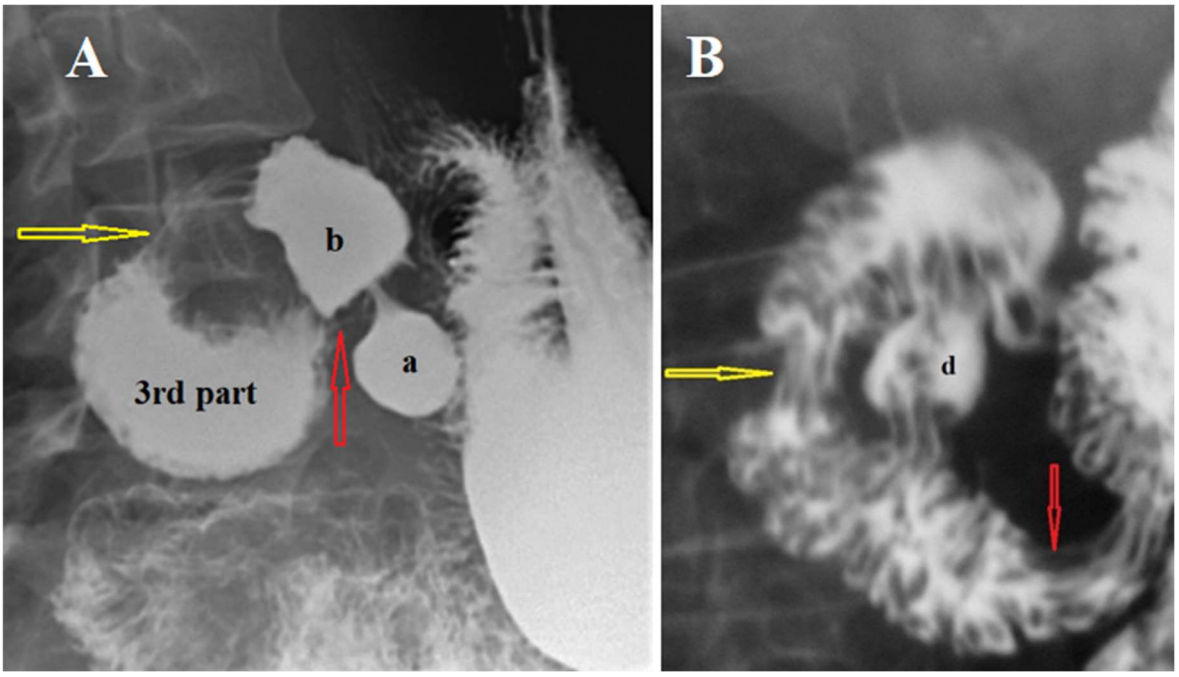


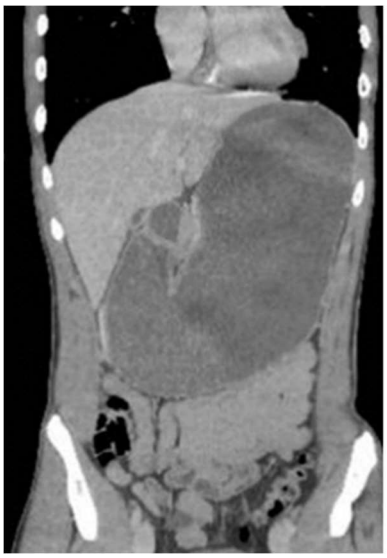
Figure 2. Radiographs of patients with duodenal dyskinesia. (**A**) Vertical radiograph of an elderly woman suffering from diabetes with complaints of heartburn and epigastric pain. The radiograph was done a minute after taking a barium. Good evacuation from the stomach is determined. The antrum chamber (**a**) extrudes barium into the duodenal bulb (b) through the open pyloric sphincter. The third part of the duodenum is expanded between the contracted sphincters (Kapandji - the yellow arrow; Ochsner - the red arrow). (**B**) Radiograph of an elderly woman with juxtapapillary diverticulum. Narrow sections with longitudinal folds are the duodenal sphincters: Kapandji (the yellow arrow) and Ochsner (the red arrow).

The clinical symptoms of duodenal dyskinesia are not specific: belching, heartburn, epigastric pain. Since the pH-meter does not diagnose GERD in them,

various diagnoses are given to these patients: functional dyspepsia, hypersensitive esophagus, etc. X-ray studies show a contraction of sphincters, even though barium does not contain hydrochloric acid. However, following the contraction, the disclosure of the sphincters, including Ochsner's sphincter is always observed. The extension of the 3rd part of the duodenum (> 2 cm) is not combined with a violation of the evacuation of barium into the jejunum and there are no signs of chronic or acute obstruction. The frequent detection of cholelithiasis in these patients can be explained by high pressure in the duodenum, which can provoke the dysfunction of the sphincter of Oddi and the injection of duodenal contents into the common bile duct [13].

V. Analysis of published cases of SMAS.

Case 1. A 15-year-old boy presented to the emergency department with acute bilious vomiting and epigastric with no weight loss or other common risk factors after the acute consumption of a meal and excessive quantities of water [14] (**Figure 3).**



The patient was diagnosed with SMA syndrome based on the findings of contrasted CT of the abdomen (The aortomesenteric angle and distance were 14° (normal range: 38°–65°4) and 4 mm (normal range: 10–28 mm5), respectively. In addition, the abdominal fat was significantly low. After the CT scan, the patient lay in prone and knee-chest position for about an hour, and then his symptoms resolved spontaneously.

Figure 3. On the CT section a massively distended stomach is determined. There are no symptoms of the duodenal enlargement.

Discussion. The young athlete had an acute stomach expansion. As shown in a study by Bhagirath Desai et al [10], the aortomesenteric angle is proportional to the body mass index (BMI). This means, firstly, that the norm for the average American, the average Hindu, and the average athlete cannot be the same. Secondly, the aortomesenteric angle cannot serve as a diagnostic test for SMAS.

Case 2. "A 12-year-old, previously healthy, girl presented to our hospital with a 3-day history of epigastric pain and vomiting. She also reported having frequent epigastric pain for the past 2 years. Physical examination revealed a body mass index of 13.5 kg/m² and abdominal distension. Abdominal contrast-enhanced CT revealed a bloated stomach and a sandwiched horizontal portion of the duodenum between the aorta and superior mesenteric artery (SMA). The angle between the aorta and SMA was 17°, and the aorta–SMA distance was 5.2 mm (**Figure 4**), which met the diagnostic imaging adult criteria for SMA syndrome (SMAS). An upper gastrointestinal series also revealed that the contrast agent smoothly reached the proximal duodenum, but stopped at the horizontal portion of the duodenum where stenosis was suspected based on CT (**Figure 5**). Decompression using a nasogastric tube improved her clinical symptoms, and she was discharged" [15].

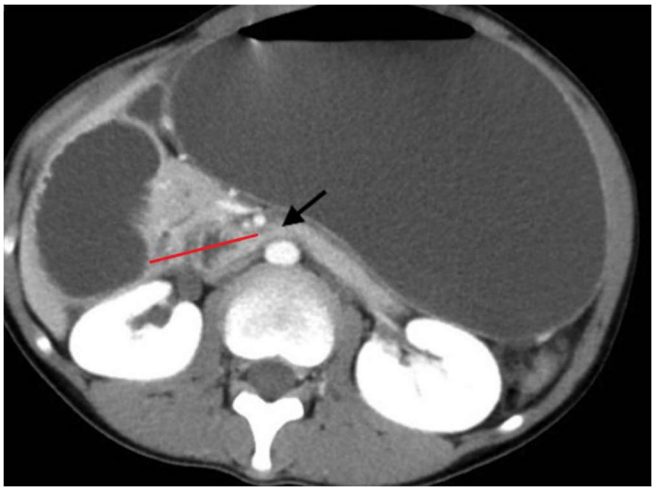


Figure 4 from an article by Kogawa et al [15]. The authors marked with a black arrow the gap between the aorta and the superior mesenteric artery. I drew a red

line from the expanded 3rd part of the duodenum to the gap between the vessels. The entire section of the duodenum, which is marked with a red line, is in a contracted state.

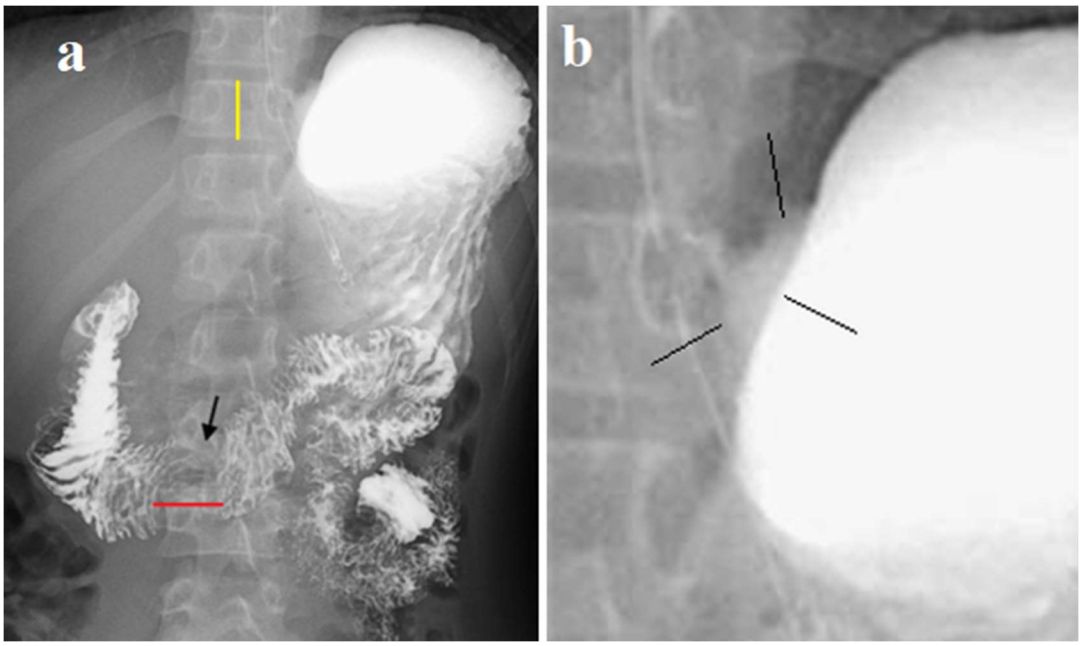
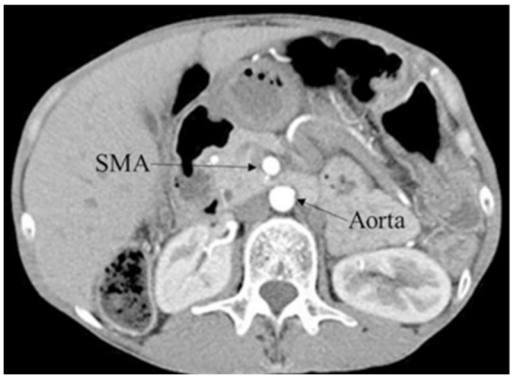


Figure 5. (a) from an article by Kogawa et al. **(b)** increased gastroesophageal junction. (**a**). The black arrow shows the longitudinal folds of the contracted intestine, which is supposedly squeezed between the vessels. I drew a red line parallel to the contracted segment. It begins to the right of the vertebra and its length is approximately equal to the height of D-11, i.e. about 2 cm. Since the aorta is in the midline of the vertebra and the diameter of the superior mesenteric artery is not more than 5 mm, the contracted segment of the intestine cannot be caused by vascular compression. (**b**) Black lines show the penetration of barium into the open abdominal part of the lower esophageal sphincter, which is convincing evidence of GERD.

Conclusion During exacerbation of GERD, which is accompanied by hypersecretion of hydrochloric acid, an Ochsner's sphincter spasm occurred. Removal of gastric contents ceased sphincter irritation. GERD and duodenal dyskinesia are frequent companions, as they are caused by the same reason - hypersecretion of hydrochloric acid.

Cases 3. Of the 46 patients with functional dyspepsia syndrome (bothersome postprandial fullness, early satiation, epigastric pain, and epigastric burning) 5

(11%) based on ultrasonography, or computed tomography **(Figure 6)** were diagnosed with SMAS. All 5 were women with a body mass index significantly lower than the remaining 41 patients (18.7 vs. 24.0 kg/m2, p=0.003). In addition, all 5 patients had 105/mL or more bacteria in the duodenum [16].



On the abdominal transverse image there is no expansion of the stomach and duodenum. The gap between the aorta and superior mesenteric artery is 8 mm, which correlates with low MBI. The only result of this study is confirmation of the fact that the patient is thin. This study has nothing to do with the diagnosis of SMAS.

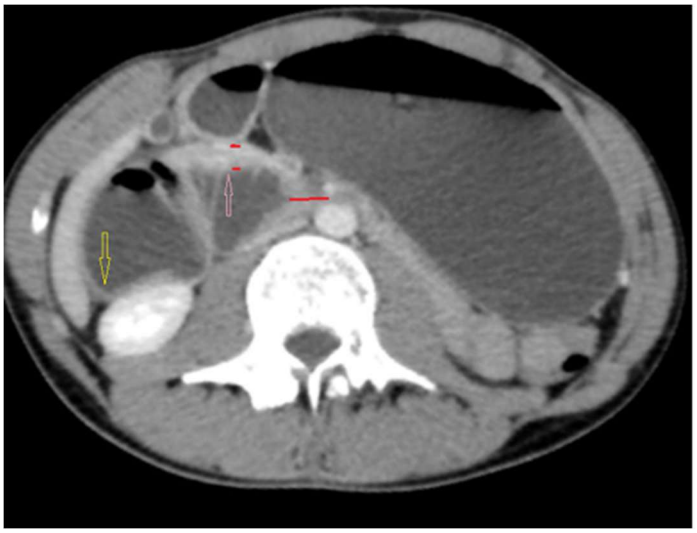
Fifure 6. On the abdominal transverse image of CT the distance between the SMA and aorta is 9 mm. The stomach and duodenum are not dilated.

Conclusion Functional dyspepsia is not a diagnosis. Its clinical picture is not specific and does not differ from GERD. The criteria of Rome IV are not a scientific method. They are established by voting. They can be used only during primary care in order to treatment by PPI and, in the absence of effect, decide which department to send the patient for examination. The absence of clinical, and radiological signs of duodenal obstruction completely exclude the of SMAS. Dimension of the aortomesenteric angle is pointless and dangerous since doctors who consider this method the gold standard operate on patients only because they are thin.

Cases 4. The authors describe 4 cases of SMAS, which they operated on without using conservative treatment [17].

The first case of 4. A 17-year-old male with epigastric pain, bloating and a prolonged history of vomiting. CT showed a massively distended stomach and proximal duodenum with an acute caliber change at the proximal D3 level (**Figure 7)**. Esophagogastroduodenoscopy revealed grade D oesophagitis. Magnetic resonance angiography revealed an aortomesenteric angle of 34 degrees

but an aortomesenteric distance of only 3.5mm. Laparoscopic duodenojejunostomy was performed.



The yellow arrow shows the wall of the duodenum of normal thickness. A sharp thickening of the wall is determined at the level of the incompletely disclosed Ochsner's sphincter (pink arrow). The red line shows the closed part of the sphincter. Conclusion: duodenitis.

Figure 7. My designations.

Conclusion A young man with GERD and duodenitis developed an exacerbation of the disease. Based on CT and MRI without process dynamics, it is impossible to exclude SMAS. In such cases, conservative treatment is indicated. The overall success rate of medical management in pediatric patients to 86% [9].

Second case of 4. A 45-year-old female with oesophagitis, epigastric pain, and vomiting. A barium meal investigation was normal and EGD initially showed reflux esophagitis. Laparoscopic duodenojejunostomy was uncomplicated.

Third case out of 4. A 21-year-old female with poorly controlled type 1 diabetes mellitus complained of daily, frequent vomiting, and the need to lie on her left side for comfort after eating. Abdominal ultrasound and barium meal investigations were unremarkable and EGD initially showed reflux esophagitis. Laparoscopic duodenojejunostomy was uncomplicated. After seven months her vomiting returned, accompanied by postprandial diarrhoea. A diagnosis of diabetic gastroparesis was made.

The fourth case of 4. A 69-year-old female with open cholecystectomy, pyloroplasty with episodes of postprandial epigastric pain, and nausea without vomiting. A barium meal investigation confirmed significant delayed transit from D3 to D4, with dilatation of the third part of the duodenum to 4.5cm (**Figure 8**). Open gastrojejunostomy was undertaken.

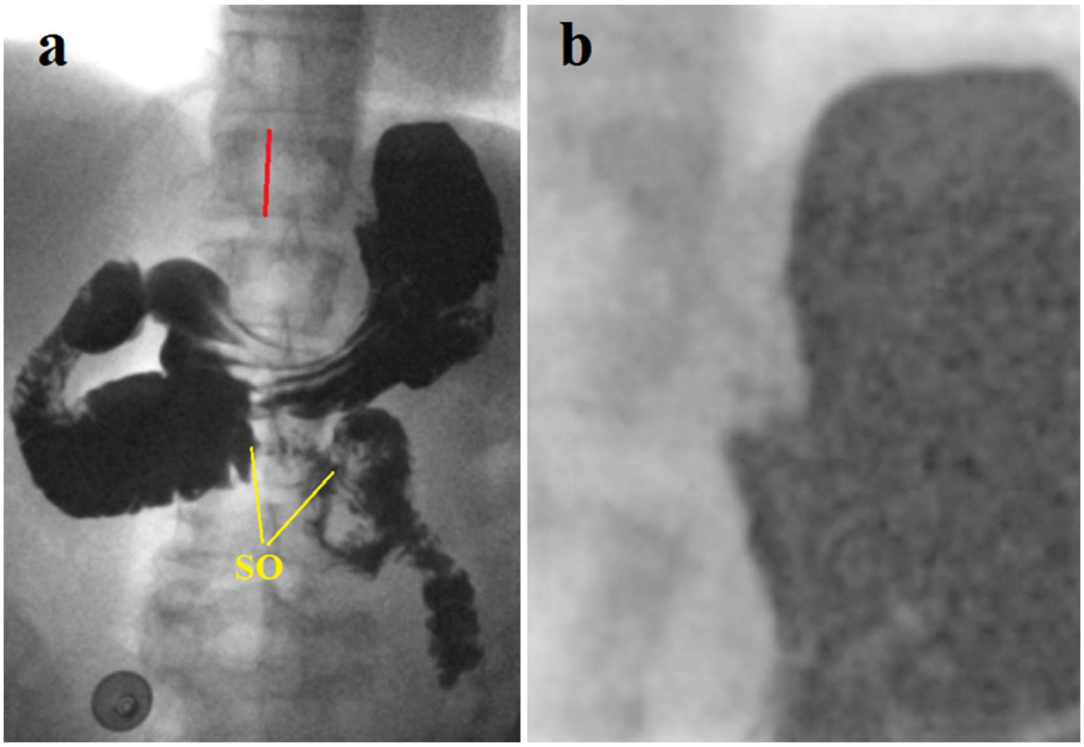


Figure 8. On the radiograph (**a**), the normal evacuation of barium to the jejunum is determined. No signs of intestinal obstruction. The red line marks the height of the first lumbar vertebra, the true height of which is 2.2 cm. The moment of contraction of the Ochsner's sphincter (SO) is fixed. Its length is 2.2 cm. The width of the 3rd section of the duodenum is 3.5 cm. The angular deformation of the cardiac section of the stomach (increase in Figure 2 b) is due to the opening of the abdominal part of the lower esophageal sphincter. Conclusion: GERD and combination with duodenal dyskinesia.

Discussion. Each of the four patients in this study had strong evidence of GERD. Only in the 1st case, there was a sharp expansion of the stomach and duodenum, which suggests a diagnosis of SMAS. However, surgical treatment without attempting conservative treatment is a mistake since the success of conservative treatment in pediatric patients reaches 86% [18]. The remaining three patients did not have obstruction of the duodenum. The diagnosis of SMAS

was erroneously established based on low aortomesenteric angle and the distance between the aorta and superior mesenteric artery. These patients with GERD were operated on only because they were thin. Duodenojejunostomy allows the aggressive gastric chyme to enter the jejunum, causing irritation, which is dangerous for the development of enteritis.

Case 5. A 19-year-old male was admitted 4 months postoperative C5 fracture-dislocation with recurrent abdominal distension, nausea, and reduced appetite, a significant weight loss of 13 kg over 6 weeks, and voluminous bilious vomiting (3.5 l/day), eructation, and gastric distension. A subsequent CT scan of the abdomen with contrast revealed classical radiological signs of SMA syndrome (**Figure 9**). The patient had free gastric drainage and high-calorie supplements (3000 kcal/day) with a triluminal nasojejunal (NJ) feeding tube (to bypass the obstruction and to build up the mesenteric retroperitoneal fat) [18].

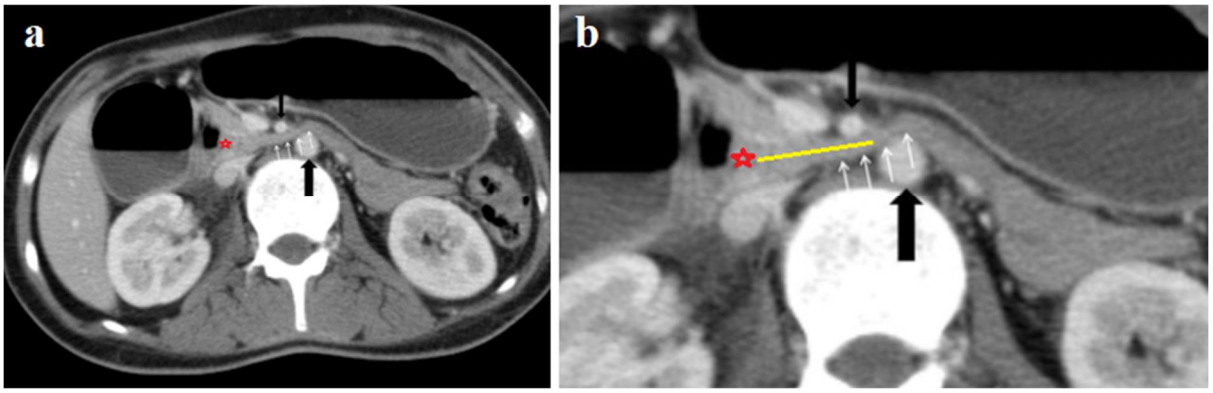


Figure 9. Axial post intravenous contrast enhancement CT image demonstrating distension of the stomach and proximal duodenum. The authors believe that the third part of the duodenum (small arrows) compressed between the abdominal aorta (large black arrow) and the SMA (small black arrow). However, the picture clearly shows that the narrowing of the duodenum begins to the right of the vertebra (red asterisk) and its length to the gap between the vessels (yellow line) is about 3.5 cm.

Discussion. Clinical symptoms and radiological signs are consistent with the diagnosis of SMAS. Conservative treatment was justified and successful. However, the interpretation of images and the pathological physiology of SMAS

contrary to known facts. First, the authors adhere to the hypothesis of Professor Carl von Rokitansky (1847), contrary to the obvious fact that the narrowed segment of the duodenum in length and location cannot be the result of compression between the vessels. In terms of length (3.5 cm) and location, this narrowed segment of the duodenum corresponds to the contracted Ochsner's sphincter. Secondly, the assumption that the cure occurs because of the accumulation of adipose tissue between the vessels, which pushes the vessels away and increases the distance between them, has no confirmation. The weight loss is not observed in all patients. A strong positive correlation exists between BMI and the angle between the aorta and SMA in patients without SMAS]10]. This means that hundreds of millions of underweight people of the third world have a "pathologically small" aortomesenteric angle. However, there is no evidence that SMAS more prevailing in the third world than in developed countries. It has obvious that the disappearance of symptoms after conservative treatment of 2-59 (13.4±2.9) days is not associated with the appearance of a fat pad in the aortomesenteric angle. It was shown that during the remission this angle does not change [19]. If we assume that vascular bifurcation is a rigid system, then the increased volume of adipose tissue in the aortomesenteric angle reduces the residual volume, which can lead to compression of the duodenum. If this system is flexible, then both the duodenum and fat pad increase the angle equally. An analysis of the literature indicates that the volume of the fat pad does not play any role in the pathogenesis of SMAS. There is reason to believe that the cessation of the provocation of the release of hydrochloric acid and its removal from the stomach leads to a decrease in the tone of the Ochsner's sphincter. Ultimately, this leads to the relaxation of the sphincter and recovery of passage.

Case 6. "A 32-year-old female patient presented to the gastroenterology department with complaints of gradually severe bloating, epigastric and left flank ache, nausea, and occasional vomiting of 1 month’s duration. A body mass index

was 18 kg/m2. Contrast-enhanced abdominal computed tomography demonstrated gastroduodenal dilatation (**Figure 10 a)**. There was a narrowing of the third portion of the duodenum compressed by SMA and AA. The upper gastrointestinal double-contrast radiograph (**Figure 10 b**) showed a vertical band of extrinsic compression (arrow) on the mid transverse part of duodenum caused by SMA with proximal duodenal dilatation (star). Therefore, a diagnosis of SMA syndrome was confirmed. Nasogastric tube was placed for decompression. Fluid resuscitation with parenteral and enteral nutritional support was managed conservatively to improve weight gain. The patient underwent a gastroduodenoscopy after the condition was relieved, revealing no intrinsic obstructions" [20].

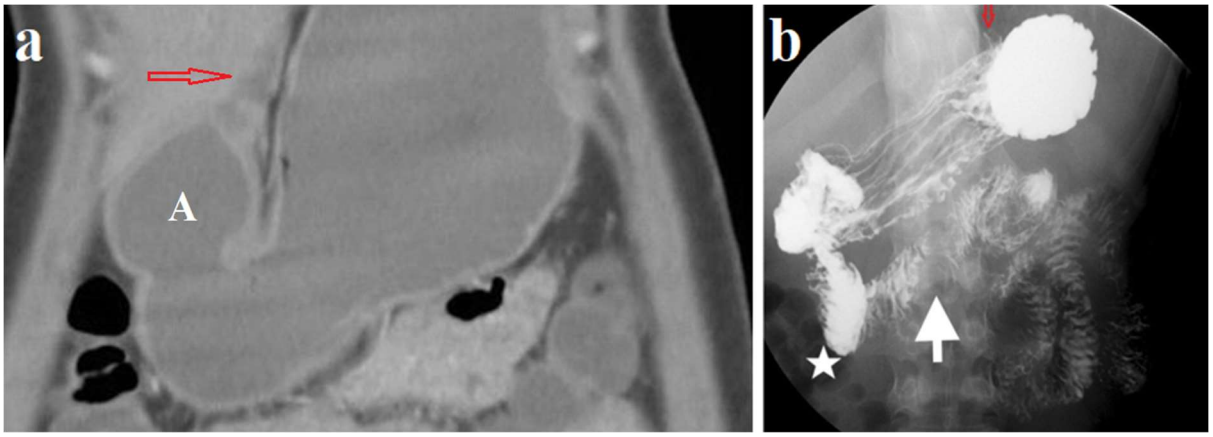


Figure 10. The captions to the figures do not correspond to the radiological data. On the longitudinal section of CT (**a**), a sharp expansion of the stomach is determined (A - antrum). The duodenum (red arrow) is not expanded. **(b)** On the x-ray, performed in a horizontal position on the back, there are no contents in the stomach. There is no expansion of the stomach and duodenum. There is no narrowing of the duodenum. The evacuation of barium into the jejunum is not impaired. The red arrow shows rough folds at the level of the lower esophageal sphincter.

Conclusion: a patient with GERD had an acute expansion of the stomach, which was successfully eliminated by drainage. The narrowing of the aortomesenteric angle (13 degrees), which corresponds to BMI = 18, was the basis for unjustified diagnosis.

Case 7. "A 57-year-old male had a 3-year history of heartburn and a 1-year history of cough. The endoscopy result was esophagitis LA-C and atrophic gastritis with erosion. The patient was diagnosed as having GERD and received treatment including PPI. The symptoms of cough, heartburn, and vomiting were relieved. Symptoms were aggravated in the past 2 months and reoccurred after he is stopping using medicines. He had a 5-kg weight loss. The DeMeester score was 1.56 and this result did not meet the standard of acid reflux disease. In high-resolution manometry the lower esophageal sphincter pressure was lower than normal.

The results of the upper gastrointestinal radiography revealed esophageal reflux disease and possible gastritis ", as well as an abrupt cutoff of the third portion of the duodenum (white arrow) and dilatations of the first and second portions of the duodenum (**Figure 11**). Abdominal CT scan revealed a 25 ° angle between the SMA and the aorta, and the gap between vessels was 7.8 mm. The patient was diagnosed as having SMA syndrome. Authors combined laparoscopic Toupet fundoplication with duodenojejunostomy to manage SMA syndrome complicating extraesophageal symptoms [21].

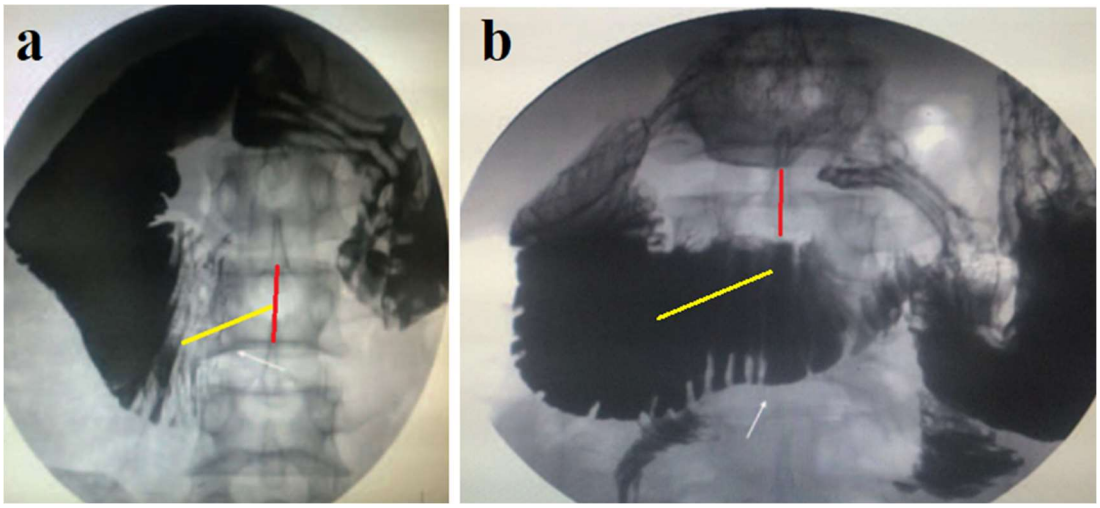


Figure 11. On the x-ray performed before surgery (**a**), the narrowing of the duodenum is determined, which begins to the right of the vertebra in 3 cm from the midline where the aortomesenteric angle is located. After surgery (**b**), this segment of the duodenum has a normal size (equal to a height of L-3 ≈ 2.4 cm).

Discussion. The patient had a typical picture of GERD without symptoms of intestinal obstruction. Short-term treatment with PPI led to the complete disappearance of symptoms. Fundoplication is not indicated if conservative treatment relieves the patient of symptoms. The x-ray taken before the operation fixed the moment of contraction of the sphincter of Ochsner. This functional state is due to dyskinesia of the duodenum and, as a rule, is replaced by the opening of this sphincter. Otherwise, there would be a clinical picture of obstruction. This is confirmed by an x-ray taken after surgery, where the opening of the Ochsner's sphincter is determined. The diagnosis of SMAS and surgery are not substantiated.

VI. Results

From published in 2016-2020 of articles on SMAS, 7 papers were selected in which there was enough information about the symptoms and examination methods. A total of 14 cases were analyzed. Only in 2 (17%) of them, the combination of clinical and radiological signs corresponded to the diagnosis of SMAS. One patient was cured by the conservative method, and the second patient, aged 17 years, was operated on without conservative treatment. In 12 cases, the diagnosis of SMAS had no justification since there was no picture of bowel obstruction. In 2 cases there was an acute expansion of the stomach, and in the remaining cases, dyskinesia of the duodenum in combination with GERD. Four patients with duodenal dyskinesia were operated on without conservative treatment. The cause of the erroneous diagnosis was a decrease in the aortomesenteric angle in patients with low BMI.

VII. Pathological physiology of the SMAS

The SMAS is an obstruction in the third part of the duodenum, resulting in the expansion of the duodenum and the stomach. The assumption that the SMAS arises because of weight loss, which leads to the disappearance of the fat pad in

the aortomesenteric angle and, therefore, the decrease of this angle, is completely unreasonable. First, weight loss is not observed in all patients. Secondly, a strong positive correlation exists between BMI and the angle between the aorta and SMA in patients without SMAS]10]. This means that hundreds of millions of underweight people of the third world have a "pathologically small" aortomesenteric angle. However, there is no evidence that SMAS more prevailing in the third world than in developed countries. Thirdly, it has obvious that the disappearance of symptoms after conservative treatment of 2-59 (13.4±2.9) days is not associated with the appearance of a fat pad in the aortomesenteric angle. It was shown that during the remission this angle does not change [14]. From the point of view of mechanics, the larger the volume of the fat pad in the interval between the aorta and SMA, the less space remains for the duodenum. This means that the pressure on the duodenum will increase. Hence it follows that neither the decrease in angle, nor the fat pad plays a role in the pathogenesis of SMAS. They are proportional to the total amount of adipose tissue in the human body, i.e. BMI.

In a previous study, we have analyzed 79 articles from PubMed describing 227 cases of SMAS [22]. Based on clinical data, all 227 patients from selected articles were divided into 2 groups. The 1st group consisted of 101 patients aged 3 – 81 (25.8 ± 3.4) years with obstruction of the duodenum which appeared 1–53 (8.2±1.9) days after severe stressful events: complicated surgeries, burns, trauma, chemotherapy, etc. Acute development of symptoms is typical for the stressful conditions, which in the catabolic stage are accompanied by significant weight loss. It is known that stress states are accompanied by a decrease in the pH of gastric contents. In such cases even high doses of proton pump inhibitors do not influence improving gastric pH [23]. In 126 patients of the second group aged 17–86 (36.7±2.2) years duodenal obstruction occurred after 3 -72 (17.2±3.2) months of the chronic diseases. Most of these patients had peptic disorders with

hypersecretion of hydrochloric acid or other disorders corresponding to the concept of dyspepsia.

The zone of the duodenal constriction both in length and location corresponds to the contraction of the Ochsner's sphincter. This sphincter normally reacts by a short-term contraction in response to irritation with hydrochloric acid. A prolonged and strong sphincter contraction, i.e., dyskinesia, is probably due to the excessive release of hydrochloric acid (**Figure 12**).

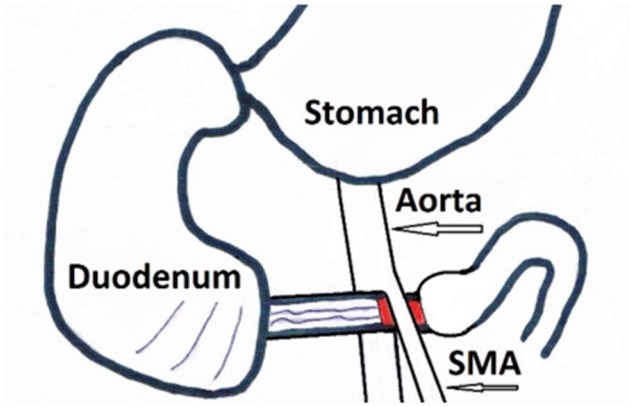


Figure 12. Scheme of anatomical relationships in patients with SMAS. The red area of narrowing is in the aortomesenteric angle.

Obviously, the clinical and x-ray manifestations depend on the amount and pH of hydrochloric acid, as well as the duration of the provocation. Ochsner's sphincter reaction can be divided into 3 degrees. Grade 1 is characterized by nonspecific symptoms of dyspepsia (abdominal pain after eating, belching, heartburn, etc.), which are characteristic of GERD. An X-ray examination determines the short-term contraction of the Ochsner's sphincter. These patients need treatment for GERD despite negative pH data. With the 2nd and 3rd degrees, vomiting occurs, rapid weight loss. On radiographs, the expansion of the stomach and duodenum, as well as a steady contraction of the Ochsner's sphincter, are determined. These symptoms are characteristic of SMAS. They have the same treatment initiation: drainage of the stomach and irrigation of duodenum with a slightly alkaline solution, feeding through a nasojejunal tube, or parenterally, as well as the appointment of PPI. Young people with a short history (2nd degree) can achieve a complete cure. In patients with a long history of the disease,

Ochsner's sphincter achalasia may occur like achalasia of the lower esophageal sphincter. In such cases, balloon expansion of the sphincter is justified pathophysiologically. Surgical treatment should be a rare exception, after a long (at least 3 weeks) attempt at conservative treatment.

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