Pathological physiology of the superior mesenteric artery syndrome. A review.

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Aim. To study the pathogenesis of the superior mesenteric artery syndrome (SMAS). **Material and methods.** We have analyzed 82 articles describing 227 cases of the SMAS from 1990 to 2019. Based on the clinical data, all patients were divided into 2 groups. **Results.** The 1st group consisted of 101 patients aged 25.8 ± 3.4 years with obstruction of the duodenum which appeared 8.2±1.9 days after severe stressful events. In 126 patients of the second group aged 36.7 ± 2.2 years, duodenal obstruction occurred after 17.2 ± 3.2 months of chronic peptic diseases. In more than 80% of patients with SMAS, the length of the duodenal obstruction zone was 3.30±0.15 cm, and it was located significantly cranial from the aortomesenteric angle, and therefore cannot be explained by the compression between the aorta and superior mesentery artery. Vitamin "C" was added to the barium in 8 patients with suspected gastroesophageal reflux disease. This led to a clear visualization of the Ochsner sphincter. Its location and length (3.20 ± 0.15 cm) were fully consistent with the length and position of the narrowed segment in SMAS. The aortomesenteric angle is proportional to the body mass index (BMI). There can be no norm. Because of this error, surgeons often operate on patients with duodenal dyskinesia only because their angle is < 25º, which only reflects their low BMI. **Conclusion.** In most cases of SMAS, the obstruction of the duodenum occurs because of the sphincter Ochsner dyskinesia in response to hypersecretion of the hydrochloric acid. Conservative treatment should be directed to hyperalimentation, draining of the duodenum, its irrigation with an alkaline solution, and suppression of hydrochloric acid excretion.

Keywords: *duodenal obstruction; physiology; sphincter Ochsner; superior mesenteric artery syndrome; Wilkie's syndrome.*

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). The aortomesenteric angle is normally 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, normally 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 [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 accepted 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. did 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 they cannot serve as the gold standard [10].

The purpose of the present study was to investigate the pathogenesis of the SMAS.

Material and methods. We have analyzed 82 articles from PubMed and PMC describing 227 cases of SMAS from 1990 to 2019 who underwent an X-ray examination of the stomach and duodenum and on frontal radiographs of which it was possible to measure the distance between the beginning of the narrowing of the duodenum and the midline of the spine. 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. Of the 101 patients in the 1st group, there were 14 (14%) children and adolescents aged 3 to 17 years (mean 12 years). In 126 patients of the second group aged 17–86 (36.7±2.2) years, including 8 patients with anorexia nervosa, 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 (**Table 1**). Among them were 3 teenagers aged 17-18 years [11].

Table. Distribution of the patients with SMAS depending on the duration of the disease.

Groups	Number of patients	Age (years)	M/W	Duration of the disease	Weight loss (kg)	BMI	Conservative treatment	Treatment time (days)	Surgery without conservative treatment
1st group	101	3 – 81 25.8±3.4	44/56	1 – 53 days 8.2 ±1.9	3 – 17 13.6±3.6	14 - 21 18.9±0.9	88.9%	2- 59 13.4±2.9	4%
2nd group	110	17 - 86 36.7±2.2	52/58	3 -72 months 17.2±3.2	3 - 29 18.0±4.3	15 - 28 16.0±3.6	39%	?	47%
	211	p <0.001		p <0.001	p >0.2	p >0.2			

The standard study of the upper digestive tract was carried out with an acidified barium suspension in 8 patients over 65 years of age with complaints of epigastric pain and/or heartburn with a presumptive diagnosis of gastroesophageal reflux disease (GERD). The purpose of this study was to determine the effect of acid on the duodenal function. For this purpose, we added 3 g of vitamin “C” to 200 ml of barium suspension.

Method. On radiographs, CT, and MRI from 35 articles we measured the distance from the sharp contraction in the 3rd part of the duodenum to the location of the superior mesenteric artery (SMA). On radiographs, all the values are greater than the real ones. We calculated the true value by multiplying the

value measured on the roentgenogram by the projection increase factor. It is equal to the ratio of the true height of the third lumbar vertebra (≈2.5 cm) to the value of its image on the roentgenogram. When analyzing CT and MRI, the coefficient is equal to the ratio of the true diameter of the abdominal aorta (≈2 cm) to the value of its image on the scan. **(Figure 1).**

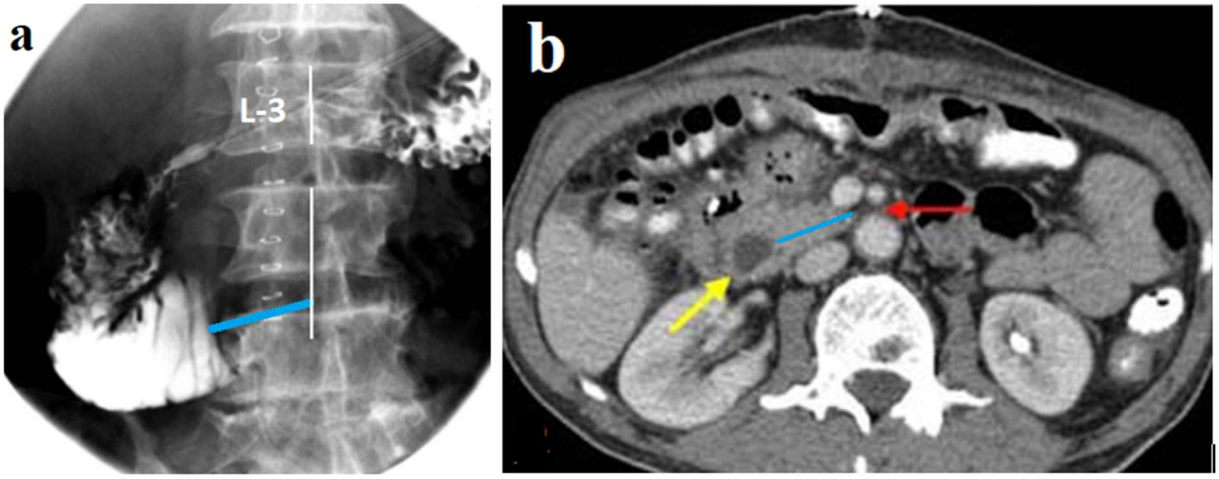

Figure 1. A 70-year-old man with a duodenal ulcer. On the 7th day after laparotomy and pyloroplasty, large residual volumes of stomach and vomiting appeared. Based on X-ray and CT studies, the SMAS was diagnosed. (From the article Chan DK. et al [11]. With the permission of the authors). **(a)** The distance from the beginning of the narrowing of the duodenum to the SMA projection is 3.5 cm (blue line). (b) The same distance on the CT is 3.7 cm. Aortomesenteric distance (red arrow) is projected into the center of the vertebra. Duodenum - yellow arrow.

Statistical analysis was performed by the method of the Student's t-test. The level of significance was set as $P<0.05$.

Results. In 20 transverse sections of MRI and CT, we determined the location of the SMA relative to L-3. In 12 (60%) cases, it was located on the midline of the vertebrae (see Figure 1, B). In 5 (25%) cases, SMA was to the left of the median line, and in 3 (15%) it was slightly to the right of the median line. The displacement from the median line was so insignificant that in all cases we calculated the length of the narrowed segment from the contraction line of the duodenum to the midpoint of the lumbar vertebra.

In 29 (83%) cases on X-ray examination or on CT and MRI, the length of the narrowed segment of the duodenum ranged from 2.5 to 4.6 cm (3.30 ± 0.15 cm) and always started far to the right of the median line (**Figure 2)**.

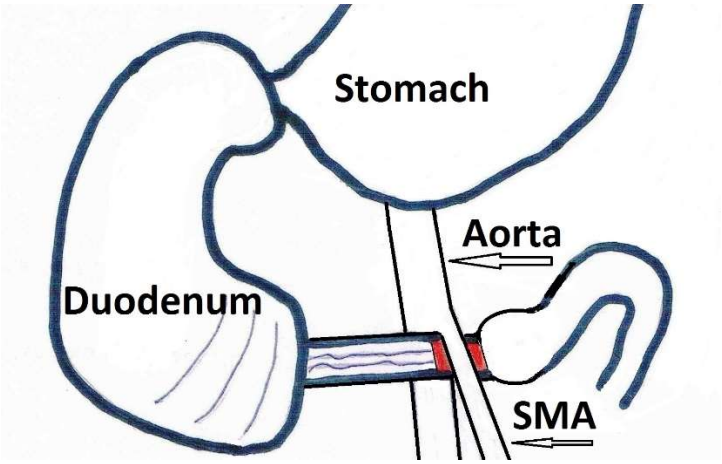

Figure 2. Scheme of anatomical relationships in patients with SMAS. The red area of narrowing corresponds to the length of the possible narrowing between the vessels in the aortomesenteric angle.

In 6 (17%) of 35 cases, the length of the narrowed segment of the duodenum was short. The place of obstruction was near 1 cm from the midline of the vertebra. In three of them on CT and MRI were rough rigid folds in the third part of the duodenum, indicating chronic duodenitis (**Figure 3**).

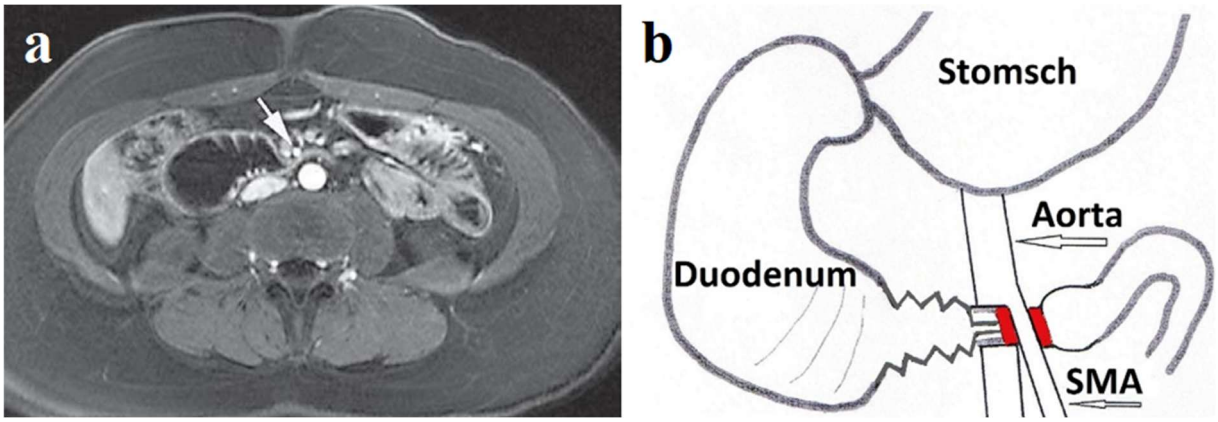

Figure 3. MRI of a patient with SMAS and its schematic. The second part of the rectum is expanded to 4 cm. Deep fixed folds in the third part of the duodenum. This picture, resembling a hedgehog, was the same in all three patients.

The first group consisted of 101 patients in whom the SMAS developed acutely after a few days (an average of 8 days) against the background of the severe catabolic process (after surgery on the spine - 16, other severe operations - 5, severe burns - 3, chemotherapy - 3, acute infectious diseases - 3, pancreatitis - 3, head injuries and heavy tetraparesis - 6, extreme physical loads - 2, etc. (see the Table). These were mainly young people with asthenic physique, who on the background severe stress have significantly lost weight over several days. In the 1st group, the conservative treatment was effective in 88.9% of cases. Non-operative treatment with gastric decompression was effective in most cases. In 6% of patients, the operation was performed without a preliminary attempt at conservative treatment, in 3% - after an unsuccessful attempt of the stomach decompression, and in 2% - after parenteral hyperalimentation only.

In patients of the 2nd group, the diagnosis of the SMAS was preceded by a long period of a disease (an average of 17.2 months). Among them were 10 patients with peptic ulcer disease, 12 patients with gastroesophageal reflux disease, 16 were taking medications that reduced the acidity of gastric juice, in 8 cases there was diagnosed dyspepsia, 4 patients had heart cachexia, the 4 patients were after the surgery due to obesity, as well as patients having chronic pancreatitis, recurrent urinary tract inflammation and diabetes mellitus. Five patients had not weight loss. In addition, 12 patients had BMI within normal limits. In patients of the 2nd group, the effect of the conservative treatment was significantly less than in patients of the 1st group - 39% and 89.9%, respectively. The more time lasted the disease, the fewer chances the conservative treatment had. Unfortunately, there is no data on the time the conservative treatment lasted in the patients of the 2nd group for several reasons. Firstly, in most articles, it is not indicated at all. Secondly, most surgeons consider the SMAS to be an indication for surgical treatment (47%). And thirdly, the conservative treatment in patients with a long-term disease is not standardized neither in terms nor in methods. Some patients were sent home

after a short period of intravenous parenteral nutrition with recommendations for postural treatment. In other cases, the surgical option was offered after 7-10 days of conservative treatment.

Third group. As a result of the application of barium with vitamin C, in all the patients of the third group clearly revealed two functional constrictions, the contractions of which created pendular barium movements between the narrowing in the 2nd and 3rd parts of the duodenum. They disappeared after the evacuation of barium into the jejunum (**Figures 4**).

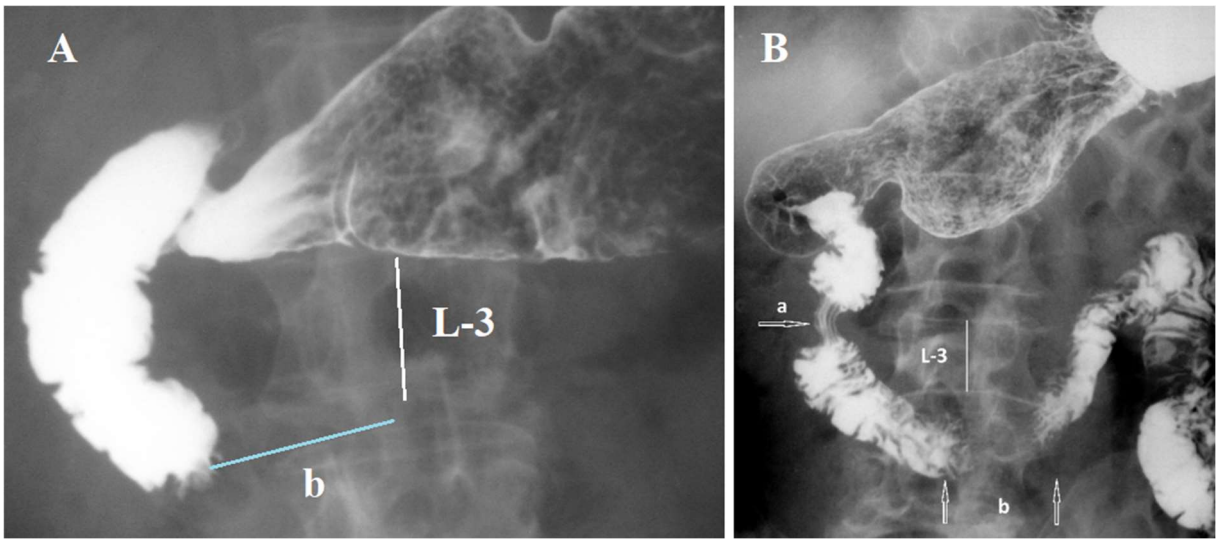

Figure 4. UGI study with Vitamin C of two elderly patients with gastroesophageal reflux disease without duodenal obstruction. (A). The short-term sphincter Ochsner contraction (b). (B). Simultaneous contraction of sphincter Kapandji (a) and sphincter Ochsner (b). The length of the sphincter Ochsner is 2.9 cm.

The length of the Ochsner sphincter was measured in 8 patients of the 3rd group and in 15 patients examined with suspected gastroesophageal reflux disease, on whose radiographs the length of the Ochsner sphincter could be measured. Its length ranged from 2.0 to 4.2 cm (3.2 ± 0.15 cm) [12].

Discussion. Currently, it is believed that the violation of patency in the 3rd part of the duodenum is due to compression of the gut in the aortomesenteric

angle. It is assumed that fatty tissue in this corner pushes the SMA away from the aorta, which increases the aortomesenteric angle and makes room for the duodenum. Conditions accompanied by the loss of fatty tissue lead to a decrease in the angle and the duodenum is compressed between the vessels. An analysis of the literature revealed the following facts that contradict the accepted 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 BMI. 2) In third world countries, there are hundreds of millions of people with low BMI but the SMAS frequency does not increase. 3) Bhagirath Desai et al. did 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 angle cannot serve as the gold standard for the diagnosis of SMAS [10]. 4) 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. In addition, it was shown that during the remission this angle does not change [13]. 5) If we consider SMAS because of mechanical compression, then an increase in adipose tissue in the aortomesenteric angle is a factor that aggravates bowel compression, since it takes up more space.

All articles on SMAS refer to an article by Neri et al [13] as a source of standards for the magnitude of the aortomesenteric angle. The reliability of the data published in an article by Neri et al is highly doubtful. US detected reduced angle <25° in 29 of the 950 patients with dyspepsia and/or abdominal pain who, according to their assessment, had SMAS during hypotonic duodenography with barium [13]. Firstly, the authors claim that "the aortomesenteric angle is normally 25–60° [2, 3, 6, 7, 10-12] and the mean aortomesenteric distance of 10–28 mm [1-3, 6, 7, 10-12]". However, in these links, there are no studies of

the normal parameters of the aortomesenteric angle. These numbers first appeared in an article by Neri et al. Secondly, the authors claim that in all 29 patients with an aortomesenteric angle < 25º, SMAS was diagnosed, which was confirmed by X-ray examination. It is known that SMAS is one of the forms of duodenal obstruction. Any violation of patency is accompanied by the expansion of the stomach and duodenum. The main clinical symptom is recurrent vomiting. These symptoms are not mentioned in the article. The X-ray diagnosis of SMAS is based only on "duodenal stenosis". On the presented X-ray there is no expansion of the stomach and duodenum. The narrowing in the 3rd part of the duodenum begins to the right of the vertebra and cannot be associated with the aortomesenteric angle **(Figure 5).**

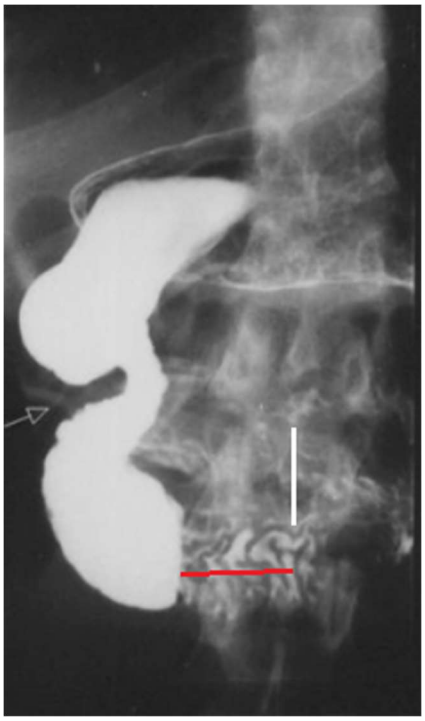

Figure 5. The only one of 29 radiographs, which was supposed to prove the diagnosis of SMAS, indicates duodenal dyskinesia. The stomach and duodenum are not dilated despite hypotonic duodenography. The constriction, about 2.5 cm long, which starts to the right of the vertebra, is caused by the contraction of the Ochsner sphincter. The fine-wavy contour of the duodenum indicates duodenitis.

Obviously, the length of the narrowing of the duodenum between the aorta (≈ 2 cm diameter) and the SMA (≈ 0.5 cm diameter) cannot be longer than 1 cm. However, the narrowing of such length was observed only in 6 (17%) of 35 cases. In 29 (83%) of the 35 radiographs, the length of the compression zone was within 2.5-4.6 cm (3.30 ± 0.15 cm). This zone of the duodenal contraction begins to the right of the vertebra (L-3) and therefore in no way could be caused by these vessels .

It is the zone of the sphincter Ochsner functions, which normally contracts in response to the penetration of the acidic gastric contents into the duodenum [14]. This sphincter prevents penetration of chyme with a low pH into the jejunum. During the contraction of the sphincter Ochsner, a chyme is discarded retrogradely to the sphincter of Kapandji, located between the bulb and the second part of the duodenum. The contraction of the sphincter of Kapandji prevents the penetration of the chyme into the bulb. The repetition of this process is described in the literature as a pendulum motion, leading to the mixing of the acidic gastric contents with the bile and pancreatic secretions, which have a high pH. When the pH of the chyme between these sphincters increases, the sphincter of Ochsner opens, allowing the bolus to enter the jejunum in a less aggressive state than originally [12]. The sphincter of Ochsner is rarely detected during upper gastrointestinal studies because acid, which provokes its contraction, is absent in the barium suspension. The use of Vitamin C greatly increased the detection of the sphincter of Ochsner and allowed to calculate its length, which ranges from 2 to 4.2 cm (3.2±0.15 cm). Thus, the length and arrangement of the area constriction of the duodenum in 29 patients with SMAS fully coincides with the length and location of the sphincter Ochsner ($P > 0.2$). Vitebski showed that the narrowing of the duodenum in patients with SMAS completely disappears under the high pressure created proximal to the duodenal constriction as if it did not exist [15]. It is possible only because of the disclosure of the sphincter under the threshold pressure. Normally, the

contraction of the sphincter Ochsner lasts only a few seconds. In the SMAS patients, the sphincter may be in a contracted state for a long time, which indicates its dyskinesia.

Complete obstruction of the duodenum is rarely observed. This means that in most cases there is a periodic opening of the Ochsner sphincter, during which the chyme bolus passes from the duodenum into the jejunum. It is likely that in 6 (17%) of 35 cases, where the length of the duodenal narrowing from the beginning to the L-3 midline was about 1 cm, the moment of the Ochsner sphincter opening was recorded.

Since Ochsner's sphincter is normal reacts by a short-term contraction in response to irritation with hydrochloric acid, it is natural to assume that a prolonged and strong sphincter contraction, i.e., its dyskinesia, is due to the excessive release of hydrochloric acid. Two different processes can lead to the appearance of duodenal obstruction. Sudden acute development of symptoms is typical for the stressful conditions (group 1), which in the catabolic stage are accompanied by significant weight loss (severe injuries, burns, malignant formations, and after severe operations). This is especially often observed in adolescents after surgery on the spine [15,16]. 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 increasing gastric pH [17].

In patients of the 2nd group cause functional dyspepsia, postprandial syndrome, and peptic diseases (gastroesophageal reflux disease, gastritis, gastric or duodenal ulcer), is the hypersecretions of hydrochloric acid. They, in contrast to the patients in the 1st group, have a long history of the disease. Disturbance of the duodenal patency increases gradually: from slight disruption without duodenal dilatation to complete obstruction. For example, in GERD, delayed gastric evacuation may be due to duodenal dyskinesia. However, a

moderate expansion of the duodenum without stagnation in the stomach and presence of a short-term spasm of the sphincter Ochsner cannot serve as a diagnostic sign of the SMAS and an indication for surgery [18]. On the background of a long-term process occur change in the wall of the duodenum. It becomes rigid including at the level of the sphincter Ochsner (see Figure 3) [11,12]. This may be the reason for the lower effectiveness of the conservative treatment in patients of the 2nd group. Analysis of the literature returns us to the previous diagnostic criteria SMAS: a combination of clinical symptoms of intestinal obstruction (vomiting, abdominal pain, bloating, weight loss, rapid saturation) with radiologic signs of obstruction in the third part of the duodenum (expansion of the stomach and duodenum with a long contraction of the sphincter Ochsner and very slow evacuation of barium to the intestine). The study of the aortomesenteric angle and the distance between the aorta and the SMA does not make any sense, because it duplicates the BMI. The decrease of the aortomesenteric angle leads to erroneous conclusions. For example, in a study by Kawanishi with co-authors in 5 (11%) from 46 women with functional dyspepsia the aortomesenteric angle was ≤22 ° with a distance between vessels of ≤8 mm. All 5 were women with a BMI significantly lower than the remaining 41 patients, which does not provide a basis for change in the diagnosis of functional dyspepsia to SMAS [19].

As the literature analysis has shown, the most effective treatment for SMAS is nasogastric decompression with nasojejunal feeding. Based on our hypothesis, the dyskinesia of the sphincter Ochsner is caused by hypersecretion of hydrochloric acid and the pathogenetic treatment should be aimed at the reducing of the hydrochloric acid release and removing it from the duodenum. Intravenous administration of the proton pump inhibitors may have a positive effect, especially in patients of the 2nd group. The duodenal irrigation with an alkaline solution is theoretically justified [20]. In patients with a long history,

which suggests duodenal wall rigidity, Ochsner sphincter dilatation may be effective.

Conclusion.

In most cases of SMAS, the obstruction of the duodenum occurs because of the sphincter Ochsner dyskinesia in response to hypersecretion of the hydrochloric acid. Conservative treatment should be directed to hyperalimentation, draining of the duodenum, its irrigation with an alkaline solution, and suppression of hydrochloric acid excretion.

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