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LETTER TO THE EDITOR

Ochsner's Sphincter Dyskinesia Is the Cause of Superior Mesenteric Artery Syndrome

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Dear Editor,

Recently, your journal published an article by Ganss and co-authors *Superior Mesenteric Artery Syndrome: a Prospective Study in a Single Institution*.[1] In this work, all the mistakes of previous researchers brought to the point of absurdity. References to previous studies cannot be proof of truth. Only undeniable scientific facts are accepted as evidence.

1. The criterion for the diagnosis of the superior mesenteric artery syndrome (SMAS), the authors consider the aortomesenteric angle ≤ 22° and distance ≤ 8 mm. The decrease in the distance between the aorta and the superior mesenteric artery (SMA) reflects only one of the symptoms of the disease—drastic slimming—which is caused by the disappearance of the fat pad in the aortomesenteric angle. These parameters cannot be considered the gold standard because

   (a) An analysis of the literature revealed the following facts that contradict the accepted concept of the pathogenesis of the SMAS. Low body weight is not a determining factor in the pathogenesis of the SMAS, since from 23.7[2] to 50%[3] of the patients have normal body mass index (BMI).
   (b) In third world countries, there are hundreds of millions of people with low BMI which does not increase the SMAS frequency.
   (c) Bhagirath Desai et al. did a prospective study of 100 patients who had undergone 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 indicate that they cannot serve as the gold standard.[4]
   (d) In those patients whose conservative treatment for 2–7 days resulted in complete relief of symptoms, it could not change and did not change the aortomesenteric angle.

2. Decrease of the aortomesenteric angle cannot be the cause of the SMAS, because the duodenal narrowing is located proximal to the angle and the length of the narrowing of the duodenum significantly exceeds the length that the vessels can create.

   (a) The diameter of the aorta at the level of L-3 is 2 cm. The diameter of the SMA is 0.5 cm. Thus, the length of the narrowed part of the duodenum between these vessels cannot be more than 1 cm. On the axial CT and MRI images, the aorta is most often located anteriorly along the midline L-3. SMA is in front of the aorta in the midline or slightly to the left or to the right (Fig. 1).

Since in most cases the aortomesenteric angle is located along the midline of L-3, on frontal abdominal radiographs, we measured the length of the narrow segment of the duodenum from the place of a sharp vertical band of the contrasted duodenum to the middle of L-3 (Fig. 2). Only in 6 (17%) of 35 cases, the narrow portion of duodenum was located directly between the aorta and SMA, and its length was about 1 cm. In the remaining 29 cases, the beginning of the narrow segment was 2.5–4.6 cm (average 3.30 ± 0.15 cm) proximal of the SMA, i.e., most of the narrowed duodenum was

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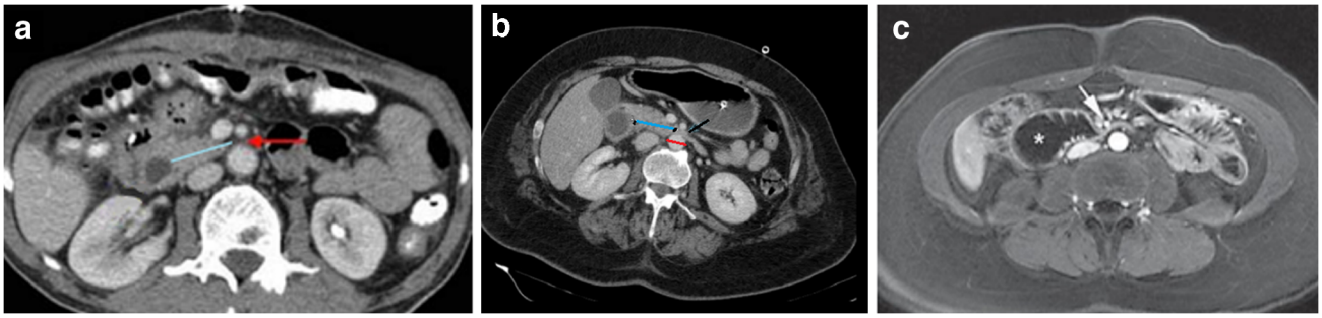

Fig. 1 Axial scans in patients with SMAS diagnosis. **a** The stomach and duodenum are not dilated. The authors' red arrow indicates the distance between the aorta and SMA as the cause of SMAS. Meanwhile, the area of the duodenum between the second portion and this intervascular gap has no contents due to spasm (my blue line). Its length is about 4 cm. (see Fig. 2a. of this patient). **b** The stomach and duodenum are dilated. The black arrow indicates the distance between the aorta and the SMA. My blue line is conducted through the spasm segment of the 3rd part of the duodenum. Its length is more than 3 cm. **c** The third part of the duodenum opened. The rough folds of the mucous are visible in this portion, which indicate duodenitis

out of the aortomesenteric angle. The location and length of the narrowed segment of duodenum corresponded to the location and length (3.2 ± 0.15 cm) ($P > 0.2$) of the functional Ochsner sphincter.[5]

Ochsner Sphincter normally contracts in response to the penetration of the acidic gastric contents into the duodenum and prevents penetration of chyme with a low pH into the jejunum. During its contraction, a chyme discarded retrogradely to the sphincter of Kapandji, located between the bulb and the second part of the duodenum. 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 mixing of the acidic gastric contents with the bile and pancreatic secretions. 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 the original.[6,7] These sphincters are not visible when examined with barium, since the contrast agent has a high pH. The addition of vitamin "C" provokes a contraction of the sphincter Ochsner (Fig. 3).

Conclusion

1. The reduction of the aortomesenteric angle reflects the patient's low weight. This indicator is not related to the diagnosis of SMAS.
2. The selection of 39 out of 162 cases of chronic "bowel movement disorders" has no objective criteria. Firstly, the reduction of the aortomesenteric angle is mistakenly taken as a gold standard. Secondly, there is no reason to believe that a symptom score is less than the minimum value (25) which indicates some other disease.

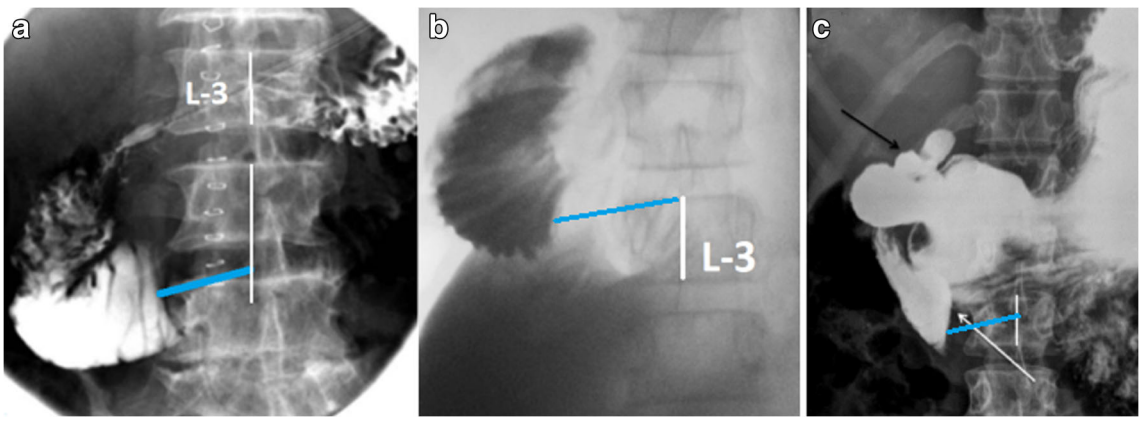

Fig. 2 Radiographs of patients who were diagnosed with SMAS. The closed segment of the duodenum (blue line) has no relation to the aortomesenteric angle. By location and length, it corresponds to the contracted Ochsner sphincter.[6] **a**, **b** Evacuation from the stomach is not broken. **c** Ulcer duodenum (black arrow)

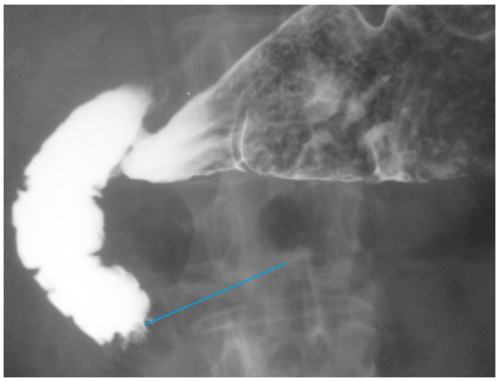

Fig. 3 Study of a patient with functional dyspepsia with the addition of vitamin "C" to barium suspension. The contracted segment of the 3rd part of the duodenum is not related to the aortomesenteric angle

3. The statement that "Superior mesenteric artery syndrome is a rare cause of chronic duodenal obstruction" is not true, since SMAS also occurs as an acute condition in association with pancreatitis, intra-abdominal inflammation, severe burns, acute gastroenteritis, spinal cord injury, and scoliosis repair surgery.
4. The cases described (5 cases per year) cannot be called a rare cause of "chronic duodenal obstruction", as there were no other more frequent diseases. These cases cannot be fully called "chronic duodenal obstruction", because, firstly, the limits of the norm beyond which gastroduodenal dilation is diagnosed are not described. Secondly, gastroduodenal dilation was only in 57% of patients, and a delayed gastroduodenal emptying only in 38%.
5. We believe that in 39 of the 162 cases of functional dyspepsia, there was a more pronounced dysfunction (dyskinesia) of the Ochsner's sphincter due to hypersecretion of hydrochloric acid against the background of chronic peptic disease.
6. An analysis of the literature shows that in the acute form of SMAS, which occurs after 8.2 ± 1.9 days after surgery or severe diseases, conservative treatment was effective in 88.9% of patients. In patients with a disease duration of 17.2 ± 3.2 months, conservative treatment was effective in 39% of cases. However, in most of this category of patients, conservative treatment was either not applied at all or lasted only a few days.
7. The authors of the article do not describe the methods of conservative treatment in 39 cases of patients. However, the lack of effect from conservative treatment in these patients cannot be an excuse for a difficult and meaningless operation, since it is impossible to carry out pathogenetic treatment without knowing the cause of the disease. The long-term result of the operation is dangerous by the development of cronic enteritis and irritable bowel syndrome, since the operation excludes the function of Ochsner's sphincter, with the result that the low-pH chyme immediately flows from the stomach into the jejunum.

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