ACTA MEDICA LITUANICA. 2016. Vol. 23. No. 3. P. 155–164

Superior mesenteric artery syndrome

Giedrius Bernotavičius[1],

Kęstutis Saniukas[1],

Irena Karmonaitė[2],

Rimantas Zagorskis[2]

1 Centre of Children's Orthopaedics and Traumatology, Children's Hospital, Affiliate of Vilnius University Hospital Santariškių Klinikos, Vilnius, Lithuania

[2] Faculty of Medicine, Vilnius University, Vilnius, Lithuania

Background. An obstruction of the distal part of the duodenum can occur because of the superior mesenteric artery syndrome (SMAS) after a surgical correction of scoliosis. It is essential to evaluate the risk factors and diagnose the SMAS in time because complications of this condition are life-threatening and it is associated with a high rate of morbidity. Diagnostics of the SMAS is challenging, because it is rare and its symptoms are non-specific. Therefore, in order to better understand the essence of this pathology and to make diagnosis easier we present a rare clinical case of the superior mesenteric artery syndrome after a surgical correction of neuromuscular scoliosis.

The clinical case. A 12-year-old girl with a specific development disorder, sensory neuropathy and progressive kypho-scoliosis was admitted to Vilnius University Children's Hospital. The patient had right side 50-degree thoracic scoliosis and an 80-degree thoracic kyphosis. She underwent posterior spinal fusion with hooks and screws from Th1 to L2. On the fourth day after the surgery the patient developed nausea and began to vomit each day 1-2 times per day, especially after meals. The SMAS was suspected and a nasogastric tube was inserted, stomach decompression and the correction of electrolytes disbalance were made. After the treatment, the symptoms did not recur and a satisfactory correction and balance of the spine were made in coronal and sagittal planes.

Conclusions. It is extremely important to identify the risk factors of the SMAS and begin preoperative diet supplements before surgical correction of scoliosis for patients with a low body mass index. After the first episode of vomiting following the surgery, we recommend to investigate these patients for a gastrointestinal obstruction as soon as possible. Decompression of the stomach, enteral or parenteral nutrition, and fluid therapy are essential in treating the SMAS.

Keywords: superior mesenteric artery, scoliosis, aortomesenteric angle, abdominal discomfort

INTRODUCTION

The superior mesenteric artery syndrome (SMAS) is rare. However, it is a life threatening condition which occurs when the abdominal aorta and the superior mesenteric artery compress the distal part of the duodenum. It was first described in 1842 by the Austrian physician Rokitansky. The SMAS appears after spine surgeries or surgical treatment of scoliosis. The rate of the SMAS varies from 0.5 to 2.4% (1–3).

The superior mesenteric artery (SMA) rises from the anterior surface of the abdominal aorta,

Correspondence to: Giedrius Bernotavičius, Centre of Children's Orthopaedics and Traumatology, Children's Hospital, Affiliate of Vilnius University Hospital Santariškių Klinikos, Santariškių St. 7, LT-08406 Vilnius. E-mail: g.bernotavičius@gmail.com

just inferior to the origin of the celiac trunk at the level of L1 or L2 vertebra. It is covered in adipose and lymphatic tissue; initially it travels in an anterior/inferior direction and enters the small intestine tether (Fig. 1). The mean angle between the abdominal aorta and the SMA is from 38 to 56 degrees, and the distance between these blood vessels is from 10 to 28 mm (4). The SMAS appears because of the reduced aortomesenteric angle (6 to 16 degrees and distance from 2 to 8 mm) (4).

The SMAS can be caused by: fast body mass loss, some metabolic conditions which reduces mesenteric and retroperitoneal adiposal tissue amount, trauma, high anchoring of Treitz ligament, low anchoring of the mesenteric artery, a high-degree lumbar lordosis, neoplastic masses near SMA radix, a dissecting aortic aneurysm and some types of surgeries (5). A surgical correction of scoliosis is one of the most common causes of the SMAS. An asthenic body composition, kyphosis in the sagittal plane, a low body mass index, loss of body mass after surgery are risk factors for the developing SMAS after a surgical treatment of scoliosis (1, 2, 6). Earlier studies showed that if the percentiles of the weight in relation to sex and age are lower than 25, then the risk of developing SMAS is higher (2, 7).

Moreover, the changes in spinal column curvature in coronary and sagittal planes play an important role in the SMAS development. A higher correction causes an increase in the patient's height. Also, an older study shows that flexibility of the kyphotic curvature, the degree of scoliosis and spine balance are very important prognostic factors for developing the SMAS. A surgical correction of scoliosis significantly elongates the spinal column and increases external compression of the distal duodenum as the distal duodenum goes through a tapered angle which is formed of the aorta, the anterior wall of the spinal column, and the posterior wall of the superior mesenteric artery. Due to scoliosis correction surgery, lateral mobility of the SMA is decreased and so the aorto-mesenteric angle is changed. It is observed that the elongation of the spinal column, especially in the lumbar region, after the surgery is an important risk factor for developing the SMAS (6, 8).

Patients with the SMAS usually complain of acute or chronic (depending on the etiology of the SMAS or the degree of duodenum compression) abdominal pain. In both cases, there is a proximal small intestine obstruction. Patients with mild obstruction complain of pain in the epigastric region and early sensation of fullness after meals, whereas if the obstruction is severe patients complain of vomiting, vomiting bile and losing body mass that cannot be explained by any other causes (9, 10). During a physical examination such non-specific findings as abdominal tenderness and high frequency instestinal sounds could be observed. Lam et al. study described 14 patients with SMAS symptoms and evaluated their significance (Table 1) (11). Results of laboratory tests are usually within the normal ranges, only in the cases with severe vomiting a disbalance of electrolytes might be seen. Abdominal pain is one

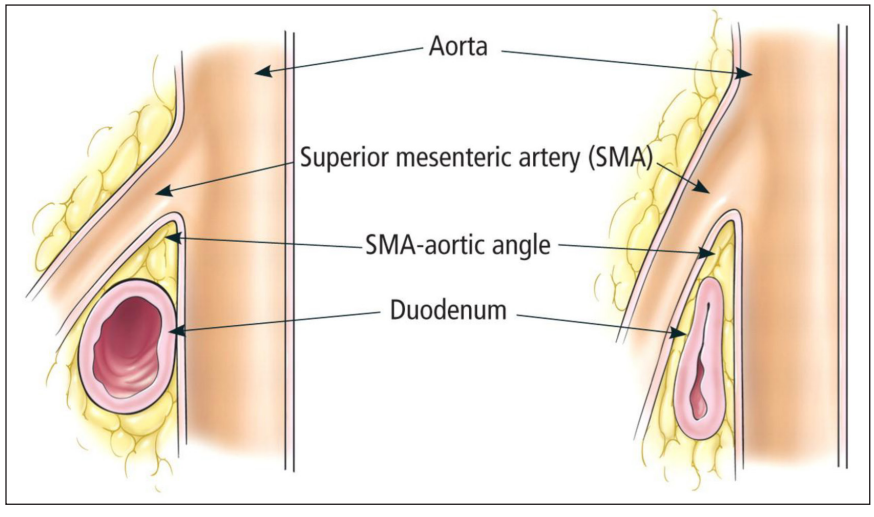

Fig. 1. Anatomy of the superior mesenteric artery syndrome

of the most common complaints of the patients who underwent surgical treatment of scoliosis. If the SMAS occurs, it usually develops one week after the surgery (1, 12).

Patients with a suspected SMAS must be thoroughly examined using radiographic tests to confirm or exclude the diagnosis. Computed tomography (CT), CT angiography, magnetic resonance imaging (MRI), MRI angiography, conventional angiography, sonoscopy and endoscopic diagnostic tests are used to diagnose the SMAS (13, 14). In the past, angiography was the gold standard for measuring the aortomesenteric angle and the distance between these two vessels. The traditional diagnostic test with barium still plays an important role in the diagnostics of the SMAS and it can be used to visualize the common, but non-specific image: the dilated proximal duodenum with break-up of barium in the distal third of the duodenum (Fig. 2) (15). In several studies, strict radiological criteria were published: dilatation of the first and second thirds of the duodenum with or without dilatation of the stomach, vertical or oblique compression of mucosal ridges, an antiperistaltic contrast flow towards the obstruction, evacuation of the contrast from the gastroduodenal region prolonged by 4–6 hours (16).

The aortomesenteric angle, the distance between the abdominal aorta and the SMA, the adiposal tissue, the obstruction of the duodenum and a possible cause of compression can be seen in CT images. Moreover, CT could be used to exclude other pathologies and diseases, to visualize the dilation of the duodenum, the anatomy of the SMA and its relation with other blood vessels. Lippl et al. have found that CT with contrast and MRI angiography are equally good for evaluating the aortomesenteric angle and distance (13). As these tests are non-invasive and can give a lot of anatomical information, nowadays they are considered to be the most valuable tests for diagnosing the SMAS. Now, the diagnostic criteria of the SMAS are the following:

- An obstruction of the duodenum with active peristalsis and sudden break-up of contrast in the distal third of the duodenum
- The aortomesenteric angle less than 25 degrees (it is the most sensitive factor, especially if the aortomesenteric distance is less than 8 mm or there is high anchoring of Treitz ligament or low position of the SMA) (4, 14).

A sonoscopy with a dopler can be used to find a reduced aortomesenteric angle (4). Also, during this test the position of the patient could be changed as it could help to recognize any changes

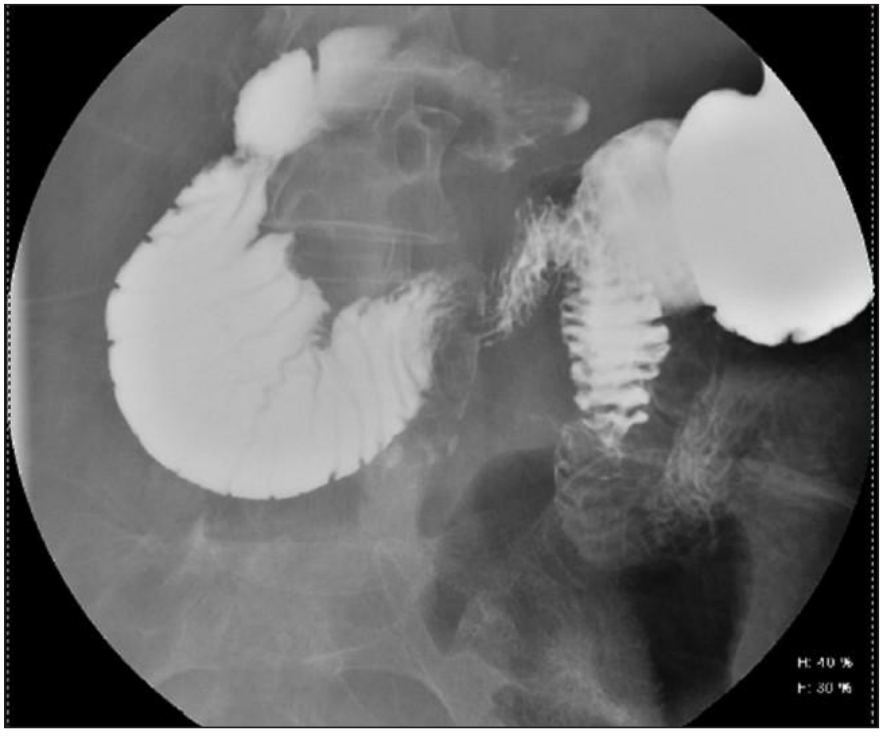

Fig. 2. Fluoroscopy of the duodenum with barium

of the aortomesenteric angle while changing body positions (14). Moreover, an endoscopy of the upper gastrointestinal tract can help to eliminate an intestinal obstruction, a gastric or duodenal ulcer, all of which could manifestat as symptoms similar to the SMAS.

Table 1. **Symptoms of the SMAS**

Symptoms	Number of patients (%)
Any type of vomiting	13 (92.9)
Abdominal pain/sensitivity	8 (57.1)
Abdominal tenderness	6 (42.9)
Vomiting bile	5 (35.7)
Hypoactive peristaltic movement	4 (28.6)
Anorexia	3 (21.4)

Treatment of the SMAS is usually started with conservative means. The main principles for treating the SMAS are: rehidration, elimination of obstruction, and maintenance of an optimal body weight. If the progression of the symptoms is sudden, a nasogastric tube for gastric and duodenum decompression, positioning of the patient (on abdomen or left side of the body with knees bend to chest) could be an effective part of treatment (16). The described specific compulsory position releases tension on the SMA and increases the space between the aorta and the SMA. It is necessary to recover the balance of fluids and electrolytes as profuse vomiting can be the cause of hypovolemia, hypokalemia, or even metabolic alcalosis. Furthermore, enteral or parenteral nutrition is needed to maintain and increase the body mass. It is important, because the increase of the retroperitonial adiposal tissue increases the aortomesenteric angle. Enteral nutrition with a nasogastric tube situated distally to the obstruction site is preferred. A recent study showed a positive response to non-surgical treatment as symptoms of 86% of the patients improved (9). Most of the patients with the SMAS that developed after surgical treatment of scoliosis made a full recovery after conservative treatment (1).

When symptoms do not improve while treating conservatively, surgical treatment is suggested (17, 18). Surgical treatment of the SMAS involves: gastrojejunostomy, duodenojejunostomy, lysis of Treitz ligament, or the surgical method of Strong. Previously, open duodejejunostomy was the gold standard as it was then the most successful and the safest surgical intervention. However, there is an even better alternative nowadays – a minimally invasive laparoscopic duodenojejunostomy (19). Gastrojejunostomy ensures adequate decompression but the obstruction is not always eliminated. Consequently, the symptoms can persist because the obstruction can cause ulcers and bile reflux. During Strong's surgery, Treitz ligament is separated, then the transverse and ascendant parts of the duodenum are mobilized. The duodenum is drawn to the right of side of the SMA. However, if there are numerous adhesions the procedure becomes very hard or even impossible to perform.

Due to a high risk of complications and relatively high mortality, it is essential to assess risk factors for the developing SMAS and to diagnose or exclude the diagnosis of the SMAS as soon as possible. Since many physicians are not aware of this syndrome due to its rarity, the SMAS is diagnosed just after a long period of time while a patient is suffering from abdominal pain or discomfort. Late diagnosis can end in complications: death because of the disbalance of electrolytes, a perforation of the stomach, gastric pneumatosis or obstruction caused by duodenum benzoar. Because of the rarity of the SMAS and its non-specific symptoms, diagnostics of this syndrome is a really big diagnostic challenge for physicians. Therefore, to better understand the risk factors of the SMAS and to facilitate diagnostics, we present a recent clinical situation during which the SMAS developed after the surgical treatment of neuromuscular scoliosis.

The clinical case

A 12-year-old girl with mixed specific developmental disorder and sensory neuropathy was hospitalized in the Vilnius University Children's Hospital for progressive neuromuscular kyphoscoliosis. The patient had not been treated surgically before. At the time of arrival, her weight was 38 kilograms and her height was 150 centimetres. The weight and height were both at the 40th percentile according to age and gender. The body mass index was 16.89 kg/m^2. There was no family history of gastrointestinal tract diseases.

The patient had right thoracic scoliosis from Th1 vertebra to L2 vertebra. The Cobb angle in the radiograph was 55 degrees, and kyphosis of the thoracic region was 80 degrees (Figs. 3, 4).

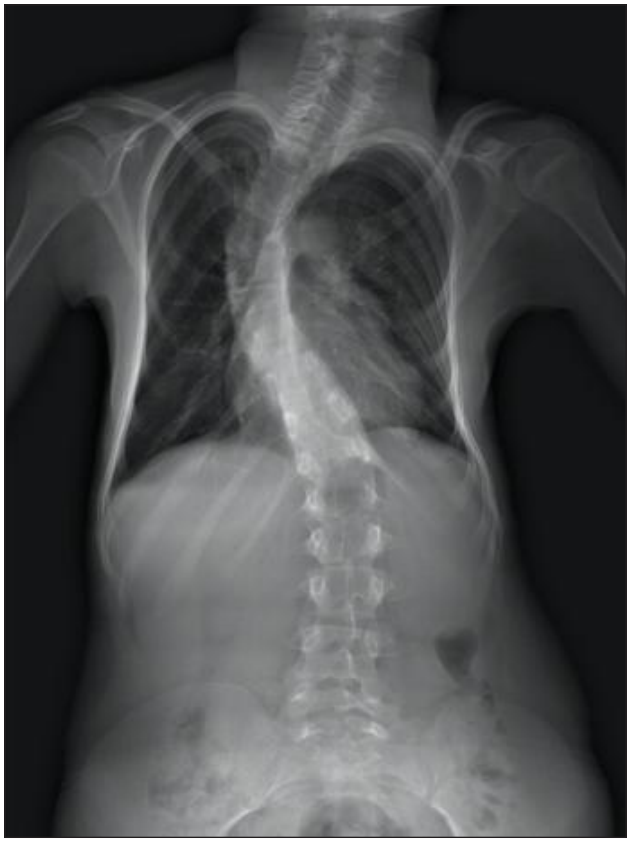

Fig. 3. Thoracic scoliosis

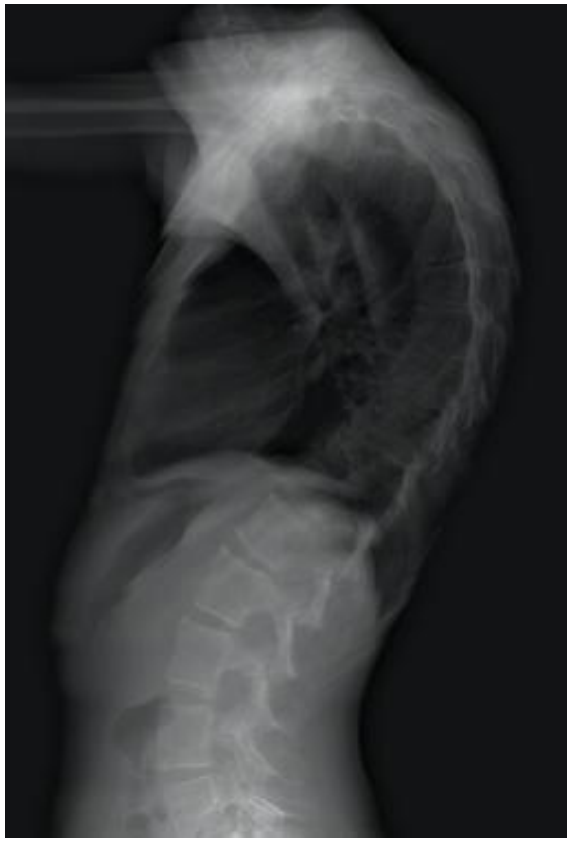

Fig. 4. Kyphosis in the thoracic region of the spine

The patient's gait was impaired as she was dragging her feet. Clinically, obvious kyphoscoliosis with the costal hump of 3 cm was seen. Her heels were in valgus deformation. Blood tests taken before the surgery showed no pathology. A whole spine MRT was performed and showed no other abnormalities except kyphoscoliosis.

Posterior spondylodesis with screws and hooks at Th1-L2 level using Expedium implants was performed. Facet joints were removed at Th2-L2 level, ligamenta flava were removed from the region of spondilodesis. Th1 vertebra was fixed with hooks, and all the other vertebrae were fixed with pedicular screws. Shevron type osteotomy was performed at the apex of kyphosis. Further, two rods were mounted, and compression and distraction at the corresponding segments and decortication with high-speed burr were performed. Bone autografts were added to the site of decortication, the wound was closed layer-by-layer and one drain was left. The duration of the whole surgery was five hours during which 300 ml of blood were lost. Haemoglobin, saturation and blood pH were within the normal range. Results of the correction were satisfying: after the surgery, the Cobb angle was 24 degrees, and coronal and sagittal balances were fully recovered.

At first, postoperative care was not complicated and during the first day the patient began to eat. Three days after the surgery, the drain was removed and the thoracolumbar brace was put on for better spine balance. During the fourth day, the patient became nauseous and later started vomiting 1 to 2 times per day, usually after meals. The brace was removed to take a closer look: the abdomen was soft without any tenderness, a bit sensitive in the epigastric region, peristalsis was normal while auscultating. Hypokalemia and metabolic alkalosis were found in blood tests. Gastric decompression with a nasogastric tube for 2 days and correction of electrolytes with intravenous fluids were performed because of a suspected SMAS. After the treatment, the condition of our patient improved and she started to eat on her own again. The patient was discharged 24 days after the surgery, a consultation of a gastroenterologist was recommended. After one month she came back for a consultation of an orthopaedist: symptoms of the SMAS during that period did not recur and the balance of the spine was satisfying.

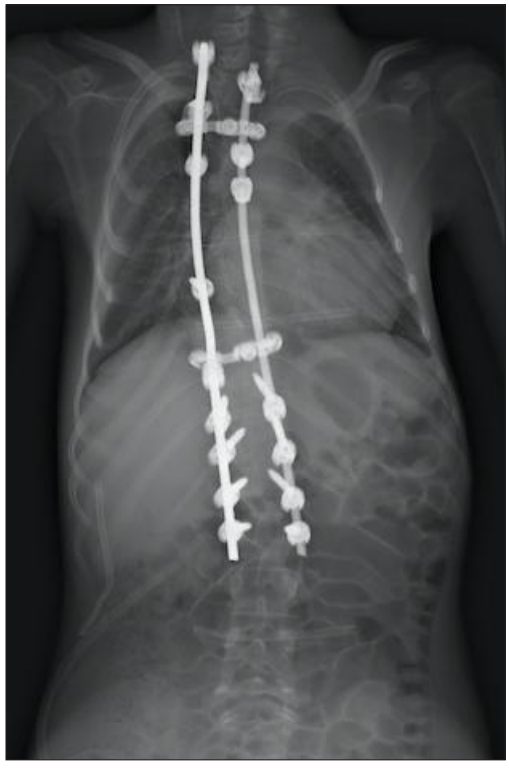

Fig. 5. A coronal view of fused spinal column

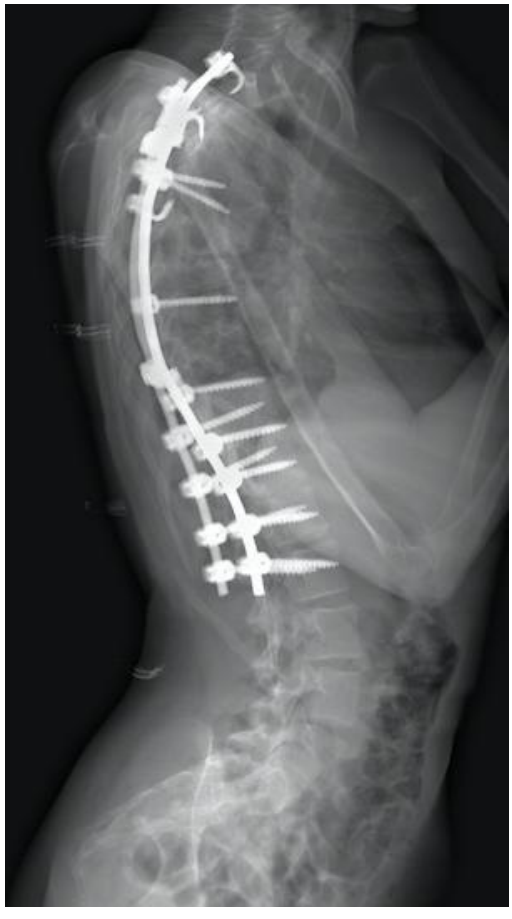

Fig. 6. A lateral view of fused spinal column

DISCUSSION

Postoperative sickness and vomiting are not very rare phenomena in children after long surgeries, thus physicians often do not evaluate these symptoms correctly as they are not very specific. For example, vomiting and sickness can occur because of the postoperative consumption of opioid drugs used to relieve pain. For patients who undergo posterior spondylodesis, sickness and vomiting can occur because of postoperative ileus and late mobilization, or because of both of these causes (11). Known causes of postoperative ileus include general anaesthesia, analgesics, electrolytic disbalance, and injury of the greater splanchnic nerve. Postoperative ileus typically manifests itself on day 1 after the surgery and resolves between the 3rd and the 5th days thereafter (15). Distinguishing postoperative ileus from a mechanical small bowel obstruction is of a critical importance. Although both can initially be managed conservatively with bowel rest, a prolonged or worsening small bowel obstruction ultimately requires surgery to prevent intestinal ischemia, necrosis, perforation, and subsequent peritonitis and sepsis. However, clinical differentiation is difficult given the common set of symptoms and signs that the conditions share (11). We recommend prompt radiological evaluation once the diagnosis of gastrointestinal obstruction is made (11).

Therefore, recognising a child at risk of developing the SMAS following a posterior spinal instrumentation can be a challenge. An asthenic body structure, kyphosis in the sagittal plane, a low body mass index are the risk factors of developing the SMAS after surgical treatment of scoliosis. Some authors conclude that an increase in the body mass before the surgery can lower the chance of developing the SMAS (3). Our patient had all of these risk factors, and, as she developed postoperative vomiting, the SMAS was consequently suspected. Recent studies show that the aortomesenteric angle correlates with the body mass index (22). Kim et al. (23) found during their study that percentile of the body mass according to gender and age helps to foresee the SMAS better than the body mass index alone. Nowadays, it is possible that known risk factors could be indicators of severity of the SMAS. However, more studies should be carried out to confirm that.

In our case, the patient started vomiting during the fourth day after the surgery. According to

literature, the SMAS most often develops during the first week after the surgery, which coincides with our case. Moreover, the patient's symptoms were relieved by compulsory position (lying on the left side, knees bent to chest). Disbalance of electrolytes was found while analysing blood. On the basis of the symptoms and risk factors, the SMAS was suspected. However, treatment was started immediately and no radiograph was taken before that, although studies recommend to perform abdominal radiograph with barium contrast first (24, 25). The condition of the patient improved significantly after gastric decompression with a nasogastric tube and a correction of electrolyte imbalance. A considerable loss of the body mass is observed in patients with the SMAS, but body mass of our patient was intact because of early treatment.

Lam et al. (11) described 14 cases of the SMAS after surgical treatment of scoliosis. Three of these cases were analysed. They assessed clinical symptoms and presented the algorithm how to diagnose and treat the SMAS (Fig. 7). One of the patients had no

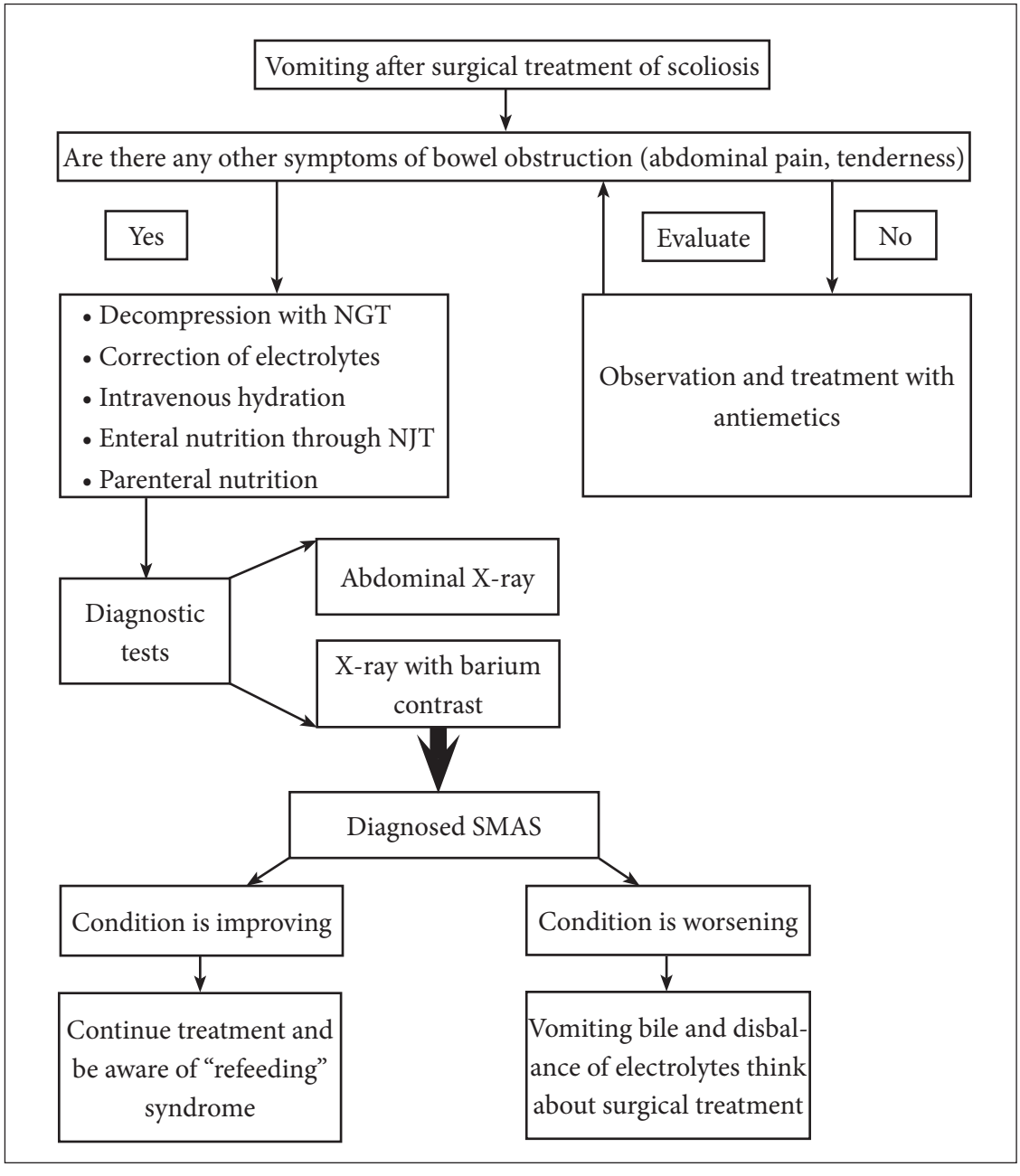

Fig. 7. The algorithm of the diagnosis and treatment of the SMAS (NGT – nasogastric tube, NJT – nasojejunal tube)

clinical symptoms until discharge thus he suffered from anorexia and low nutrition. The patient came back to hospital on the 27th day after the surgery because of recurrent vomiting which began on the 13th day. In other study, Tsirikos et al. (26) described a clinical case in which postoperative care was not complicated and even during the first day the patient began to eat normally. After the scheduled treatment, she was discharged although her body mass was lower than the 20th percentile. A high nutritional value diet was recommended to the patient. She was hospitalised because of sickness and constant vomiting 45 days after the surgery. She was dehydrated with oliguria and disbalance of electrolytes, and her body mass decreased by 7 kilograms and was lower than the 3rd percentile. An abdominal radiograph with barium contrast was taken and the diagnosis of the SMAS was established. Examples of such a late onset of symptoms have also been described in other studies (27). It is possible that before discharge patients had already had some degree of proximal duodenum obstruction which was not suspected, and with the decrease of the body mass the obstruction increased and caused the symptoms. This just confirms how important it is to suspect and diagnose the SMAS. Parents should also be informed about possible symptoms of the SMAS: loss of the body weight, abdominal pain, vomiting before discharge from hospital.

Frequently, symptoms of the SMAS create a vicious circle: food is not tolerated, then the body weight is lost, and as a result the aortomesenteric angle decreases, the duodenum is compressed even more, and it is followed by sickness and vomiting. The aim of treating the SMAS is to break this circle.

The SMAS is often more acute in children than in adults; also, conservative treatment is often more successful for children (9). The course of disease is usually more chronic in adults and surgical treatment is more effective for them as well (28). In our clinical case, the symptoms of the patient improved significantly after conservative treatment. We performed gastric decompression with a nasogastric tube for two days. However, nasojejunal enteral nutrition, or even parenteral nutrition, is necessary in more severe cases to obtain the required level of nutrition (5). A patient can return to eating normally when his or her condition is improved and the compression of the duodenum is decreased. Of course, if the body mass is low, high in calories nutritious food is prescribed.

Although the incidence of the SMA syndrome after scoliosis surgery is generally low (1–4.7%) (2, 3, 7, 12), it is associated with significant morbidity. Hence, there should be a high index of suspicion, especially for high-risk patients. Classically, the symptoms of the condition include nausea, bilious vomiting or increased bilious nasogastric aspirates, postprandial abdominal distension, and epigastric pain. However, the diagnosis of the SMA syndrome may not be easy, given the common clinical symptoms that it shares with several other conditions.

CONCLUSIONS

It is necessary to identify risk factors for the SMAS before surgical treatment of scoliosis for patients, especially those with a low body mass index. While diagnostics of the SMAS is difficult as the symptoms are non-specific and the syndrome itself is rare, it is essential to recognise the "red flags" after the surgery and to observe body mass of a patient.

The most common symptoms of the SMAS are abdominal pain, vomiting episodes, and abdominal tenderness. We suggest testing for gastrointestinal obstruction immediately if vomiting starts after surgery. An abdominal x-ray with barium contrast should be the first choice test for patients with vomiting episodes. The SMAS can develop up to one month after surgical treatment of scoliosis, so if there are symptoms of a gastrointestinal obstruction, the SMAS must be included as a possible cause.

Gastric decompression, enteral or parenteral nutrition, and infusion therapy comprise emergency treatment for the SMAS. If the condition does not improve over one week, surgical treatment is recommended. Surgical methods of SMAS treatment include: Strong surgery, gastrojejunostomy, or duodenojejunostomy. A delay in using surgical treatment can increase mortality rates because of malnutrition and a disbalance of electrolytes. Duodenojejunostomy has shown best results so far. When the SMAS is diagnosed too late or not diagnosed at all, life-threatening complications can appear.

Received 11 April 2016
Accepted 27 September 2016

References

1. Altiok H, Lubicky JP, DeWald CJ, Herman JE. The superior mesenteric artery syndrome in patients with spinal deformity. Spine. 2005 Oct 1; 30(19): 2164–70.
2. Zhu Z-Z, Qiu Y. Superior mesenteric artery syndrome following scoliosis surgery: its risk indicators and treatment strategy. World J Gastroenterol WJG. 2005 Jun 7; 11(21): 3307–10.
3. Tsirikos AI, Jeans LA. Superior mesenteric artery syndrome in children and adolescents with spine deformities undergoing corrective surgery. J Spinal Disord Tech. 2005 Jun; 18(3): 263–71.
4. Neri S, Signorelli SS, Mondati E, Pulvirenti D, Campanile E, Di Pino L, et al. Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. J Intern Med. 2005 Apr; 257(4): 346–51.
5. Welsch T, Büchler MW, Kienle P. Recalling Superior Mesenteric Artery Syndrome. Dig Surg. 2007; 24(3): 149–56.
6. Shah MA, Albright MB, Vogt MT, Moreland MS. Superior mesenteric artery syndrome in scoliosis surgery: weight percentile for height as an indicator of risk. J Pediatr Orthop. 2003 Oct; 23(5): 665–8.
7. Braun SV, Hedden DM, Howard AW. Superior mesenteric artery syndrome following spinal deformity correction. J Bone Joint Surg Am. 2006 Oct; 88(10): 2252–7.
8. Crowther MAA, Webb PJ, Eyre-Brook IA. Superior mesenteric artery syndrome following surgery for scoliosis. Spine. 2002 Dec 15; 27(24): E528–33.
9. Biank V, Werlin S. Superior mesenteric artery syndrome in children: a 20-year experience. J Pediatr Gastroenterol Nutr. 2006 May; 42(5): 522–5.
10. Cohen LB, Field SP, Sachar DB. The superior mesenteric artery syndrome. The disease that isn't, or is it? J Clin Gastroenterol. 1985 Apr; 7(2): 113–6.
11. Lam DJL, Lee JZJ, Chua JHY, Lee YT, Lim KBL. Superior mesenteric artery syndrome following surgery for adolescent idiopathic scoliosis: a case series, review of the literature, and an algorithm for management. J Pediatr Orthop B. 2014 Jul; 23(4): 312–8.
12. Hod-Feins R, Copeliovitch L, Abu-Kishk I, Eshel G, Lotan G, Shalmon E, et al. Superior mesenteric artery syndrome after scoliosis repair surgery: a case study and reassessment of the syndrome's pathogenesis. J Pediatr Orthop Part B. 2007 Sep; 16(5): 345–9.
13. Lippl F, Hannig C, Weiss W, Allescher H-D, Classen M, Kurjak M. Superior mesenteric artery syndrome: diagnosis and treatment from the gastroenterologist's view. J Gastroenterol. 2002; 37(8): 640–3.
14. Unal B, Aktaş A, Kemal G, Bilgili Y, Güliter S, Daphan C, et al. Superior mesenteric artery syndrome: CT and ultrasonography findings. Diagn Interv Radiol Ank Turk. 2005 Jun; 11(2): 90–5.
15. Girotra M, Shah HR, Rego RF. An intriguing cause of intractable nausea and vomiting. Saudi J Gastroenterol Off J Saudi Gastroenterol Assoc. 2013 Aug; 19(4): 190–1.
16. Dietz UA, Debus ES, Heuko-Valiati L, Valiati W, Friesen A, Fuchs KH, et al. [Aorto-mesenteric artery compression syndrome]. Chir Z Für Alle Geb Oper Medizen. 2000 Nov; 71(11): 1345–51. German.
17. Shiyanagi S, Kaneyama K, Okazaki T, Lane GJ, Yamataka A. Anterior transposition of the third part of the duodenum for the treatment of superior mesenteric artery syndrome. J Pediatr Surg. 2008 Feb; 43(2): e1–3.
18. Okugawa Y, Inoue M, Uchida K, Kawamoto A, Koike Y, Yasuda H, et al. Superior mesenteric artery syndrome in an infant: case report and literature review. J Pediatr Surg. 2007 Oct; 42(10): E5–8.
19. Singaporewalla RM, Lomato D, Ti TK. Laparoscopic duodenojejunostomy for superior mesenteric artery syndrome. JSLS J Soc Laparoendosc Surg Soc Laparoendosc Surg. 2009 Sep; 13(3): 450–4.
20. Fuhrman MA, Felig DM, Tanchel ME. Superior mesenteric artery syndrome with obstructing duodenal bezoar. Gastrointest Endosc. 2003 Mar; 57(3): 387.
21. Lim JE, Duke GL, Eachempati SR. Superior mesenteric artery syndrome presenting with acute massive gastric dilatation, gastric wall pneumatosis, and portal venous gas. Surgery. 2003 Nov; 134(5): 840–3.
22. Ozkurt H, Cenker MM, Bas N, Erturk SM, Basak M. Measurement of the distance and angle between the aorta and superior mesenteric artery: normal values in different BMI categories. Surg Radiol Anat SRA. 2007 Oct; 29(7): 595–9.
23. Kim JY, Kim HS, Moon ES, Park JO, Shin DE, Lee GK, et al. Incidence and Risk Factors

Associated with Superior Mesenteric Artery Syndrome following Surgical Correction of Scoliosis. Asian Spine J. 2008 Jun; 2(1): 27–33.
24. Wyten R, Kelty CJ, Falk GL. Laparoscopic duodenojejunostomy for the treatment of superior mesenteric artery (SMA) Syndrome: case series. J Laparoendosc Adv Surg Tech A. 2010 Mar; 20(2): 173–6.
25. Record JL, Morris BG, Adolph VR. Resolution of Refractory Superior Mesenteric Artery Syndrome with Laparoscopic Duodenojejunostomy: Pediatric Case Series with Spectrum of Clinical Imaging. Ochsner J. 2015; 15(1): 74–8.
26. Tsirikos AI, Anakwe RE, Baker AD. Late presentation of superior mesenteric artery syndrome following scoliosis surgery: a case report. J Med Case Reports. 2008 Jan 19; 2: 9.
27. Smith BG, Hakim-Zargar M, Thomson JD. Low body mass index: a risk factor for superior mesenteric artery syndrome in adolescents undergoing spinal fusion for scoliosis. J Spinal Disord Tech. 2009 Apr; 22(2): 144–8.
28. Merrett ND, Wilson RB, Cosman P, Biankin AV. Superior mesenteric artery syndrome: diagnosis and treatment strategies. J Gastrointest Surg Off J Soc Surg Aliment Tract. 2009 Feb; 13(2): 287–92.

Giedrius Bernotavičius, Kęstutis Saniukas, Irena Karmonaitė, Rimantas Zagorskis

VIRŠUTINĖS PASAITO ARTERIJOS SINDROMAS

Santrauka

Įžanga ir tikslas. Po chirurginės skoliozės korekcijos gali išsivystyti dvylikapirštės žarnos obstrukcija dėl viršutinės pasaito arterijos sindromo (VPAS). Dėl gresiančių komplikacijų ir santykinai didelio mirštamumo itin svarbu laiku diagnozuoti arba atmesti viršutinės pasaito arterijos sindromą ir įvertinti rizikos veiksnius sindromui išsivystyti. Šio sindromo retumas ir nespecifiniai simptomai – tikras diagnostinis iššūkis gydytojams. Todėl siekiant geriau suprasti ligos esmę, išsiaiškinti rizikos veiksnius ir palengvinti diagnostiką, darbe aprašoma neseniai įvykusi ir reta klinikinė situacija, kurios metu išsivystė viršutinės pasaito arterijos sindromas po neuroraumeninės skoliozės korekcijos.

Klinikinis atvejis. 12 metų mergaitė su specifiniu mišriu raidos sutrikimu ir sensorine neuropatija atvyko į Vilniaus universiteto vaikų ligoninę dėl progresuojančios neuroraumeninės kifoskoliozės. Tiriamajai pacientei išsivystė dešinioji krūtininė 50 laipsnių skoliozė ir 80 laipsnių kifozė. Pacientei atlikta nugarinė spondilodezė sraigtais ir kabliukais Th1-L2 lygyje. Ketvirtą parą po operacijos mergaitę pradėjo pykinti, ji pradėjo vemti kiekvieną dieną po 1–2 kartus, dažniausiai pavalgius. Įtarus viršutinės pasaito arterijos sindromą, atlikta skrandžio dekompresija, 2 dienas įvestas nazogastrinis zondas ir koreguotas elektrolitų disbalansas. VPAS būdingi simptomai nesikartojo, ir buvo pasiekta patenkinama stuburo korekcija bei balansas.

Išvados. Būtina identifikuoti viršutinės pasaito arterijos sindromui būdingus rizikos veiksnius ir pradėti priešoperacinę dietą pacientams su mažu kūno masės indeksu, kuriems planuojama atlikti chirurginę skoliozės korekciją. Atsiradus pirmajam vėmimo epizodui po operacijos siūlome, kad šie pacientai būtų nedelsiant patikrinti dėl virškinamojo trakto obstrukcijos. Skrandžio dekompresija, enterinis arba parenterinis maitinimas, skysčių terapija yra būtinos priemonės gydant VPAS.

Raktažodžiai: viršutinė pasaito arterija, skoliozė, aortomezenterinis kampas, pilvo diskomfortas