ISSN 2221-2582

UDK 616.342-06-07-089]-053.5-055.23+616-006+616.136
DOI: 10.56871/CmN-W.2024.74.66.001

WILKIE SYNDROME AS A COMPLICATION OF MISDIAGNOSED CROHN'S DISEASE

© Maisa A. Amanova[2], Victoria V. Kholostova[1, 2], Alexander Yu. Razumovsky[1, 2], Alexey N. Smirnov[1, 2], Anatoly I. Khavkin[3, 4], Zorikto B. Mitupov[1, 2], Artur G. Mannanov[2], Maria S. Kuznetsova[1]

[1] Pirogov Russian National Research Medical University. Ostrovityanova str. , 1, Moscow, Russian Federation, 117997

[2] Filatov Children City Clinical Hospital. Sadovaya-Kudrinskaya str., 15, Moscow, Russian Federation, 123001

[3] Research Clinical Institute of Childhood, Ministry of Health of the Moscow Region. Bolshaya Serpukhovskaya str., 62, Moscow, Russian Federation, 115093

[4] Belgorod National Research University. Pobeda str. , 85, Belgorod region, Belgorod, Russian Federation, 308015

Contact information:
Anatoly I. Khavkin — Doctor of Medical Sciences, Professor, Head of the Department of Gastroenterology and Dietetics named after A.V. Mazurin, Research Clinical Institute of Childhood, Ministry of Health of the Moscow Region; Professor, Department of Pediatrics with a Course in Pediatric Surgical Diseases, Institute of Medicine, Belgorod National Research University.
E-mail: gastropedclin@gmail.com ORCID: 0000-0001-7308-7280 SPIN: 6070-9473

For citation: Amanova MA, Kholostova VV, Razumovsky AYu, Smirnov AN, Khavkin AI, Mitupov ZB, Mannanov AG, Kuznetsova MS. Wilkie syndrome as a complication of misdiagnosed Crohn's disease. Children's Medicine of the North-West (St. Petersburg). 2024;12(1):148-155. DOI: https://doi.org/10.56871/CmN-W.2024.74.66.001

Received: 01.12.2023 **Revised: 26.12.2023** **Accepted: 25.01.2024**

Abstract. Wilkie's syndrome or superior mesenteric artery (SMA) syndrome is a relatively rare condition due to abnormal branching of the SMA from the abdominal aorta due to the disappearance of adipose tissue that provides stability of the angle between the two major arterial vessels and, consequently, compression of the duodenum with subsequent impaired passage. The development of Wilkie's syndrome, given the data of the literature and our own experience, is always secondary. The peculiarity of our clinical case of Wilkie's syndrome in an adolescent girl is the child's prolonged treatment for misdiagnosed Crohn's disease of the colon, followed by the discovery of angiodysplasia and repeated surgical interventions, resulting in weight loss and, consequently, the development of syndrome or SMA.

Key words: *Wilkie's syndrome, superior mesenteric artery syndrome, intestinal angiomatosis, Crohn's disease.*

СИНДРОМ УИЛКИ КАК ОСЛОЖНЕНИЕ ОШИБОЧНО ДИАГНОСТИРОВАННОЙ БОЛЕЗНИ КРОНА

© Маиса Анизалиевна Аманова[2], Виктория Валерьевна Холостова[1, 2], Александр Юрьевич Разумовский[1, 2], Алексей Николаевич Смирнов[1, 2], Анатолий Ильич Хавкин[3, 4], Зорикто Батоевич Митупов[1, 2], Артур Габдулханнанович Маннанов[2], Мария Сергеевна Кузнецова[1]

[1] Российский национальный исследовательский медицинский университет имени Н.И. Пирогова. 117997, г. Москва, ул. Островитянова, 1

[2] Детская городская клиническая больница имени Н.Ф. Филатова Департамента здравоохранения г. Москвы. 123001, г. Москва, ул. Садовая-Кудринская, 15

[3] Научно-исследовательский клинический институт детства Министерства здравоохранения Московской области. 115093, г. Москва, Большая Серпуховская ул., 62

[4] Белгородский государственный национальный исследовательский университет. 308015, Белгородская область, г. Белгород, ул. Победы, 85

Контактная информация:
Анатолий Ильич Хавкин — д.м.н., профессор, заведующий кафедрой гастроэнтерологии и диетологии им. А.В. Мазурина; главный научный сотрудник отдела педиатрии Научно-исследовательского клинического института детства; профессор кафедры педиатрии с курсом детских хирургических болезней Медицинского института Белгородского государственного национального исследовательского университета. E-mail: gastropedclin@gmail.com ORCID: 0000-0001-7308-7280 SPIN: 6070-9473

Для цитирования: Аманова М.А., Холостова В.В., Разумовский А.Ю., Смирнов А.Н., Хавкин А.И., Митупов З.Б., Маннанов А.Г., Кузнецова М.С. Синдром Уилки как осложнение ошибочно диагностированной болезни Крона // Children's Medicine of the North-West. 2024. Т. 12. № 1. С. 148–155. DOI: https://doi.org/10.56871/CmN-W.2024.74.66.001

Поступила: 01.12.2023 **Одобрена: 26.12.2023** **Принята к печати: 25.01.2024**

Резюме. Синдром Уилки, или синдром верхней брыжеечной артерии (СВБА) — относительно редкое состояние, обусловленное аномальным отхождением верхней брыжеечной артерии от брюшного отдела аорты из-за исчезновения жировой ткани, которая обеспечивает стабильность угла между двумя крупными артериальными сосудами, и, соответственно, сдавливанием двенадцатиперстной кишки с последующим нарушением пассажа. Развитие синдрома Уилки, учитывая данные литературы и собственный опыт, всегда вторично. Особенностью нашего клинического случая синдрома Уилки у девочки-подростка является длительная курация ребенка по поводу ошибочно диагностированной болезни Крона толстой кишки, с последующими обнаружением ангиодисплазии и неоднократными оперативными вмешательствами, приведшими к потере веса и, как следствие, развитию СВБА.

Ключевые слова: *синдром Уилки; синдром верхне-брыжеечной артерии; ангиоматоз кишечника; болезнь Крона.*

INTRODUCTION

Compression of the inferior or horizontal part of the duodenum (DU) between the abdominal aorta and the superior mesenteric artery (SMA) as a cause of intestinal obstruction was first described by Karl von Rokitansky in 1861 in a post mortem case. The syndrome was later studied in detail and described by Wilkie in 1927 [1].

The syndrome has many names, the most famous of which are: Wilkie's syndrome, superior mesenteric artery (SMA) syndrome, cast syndrome and aorto-mesenteric compass syndrome. The disease is very rare with only 500 reported cases in the literature and an estimated prevalence of 0.013%-0.3% according to the upper gastrointestinal tract radiography [2, 3]. It is more common among the female than male gender at a ratio 2:1 ratio, mainly between the second and fourth decades of life [3, 4].

Wilkie's syndrome is caused by compression of the third part of the duodenum between the aorta and the superior mesenteric artery branching from it at an acute angle, which in turn, leads to duodenal obstruction. Normally, the angle between the SMA and the aorta ranges from 25 to 60°, but with this syndrome it is narrowed (from 6 to 25°). An acute aortomesenteric angle is formed due to congenital anomalies (high insertion of the ligament of Treitz, low origin of the SMA from the abdominal aorta), significant weight loss, lumbar hyperlordosis, restorative proctocolectomy with ileaoanal anastomosis and other conditions. That is, this syndrome can be caused by various reasons, ranging from congenital anomalies to a hypercatabolic state or malnutrition. As a result, there is a loss of perivascular and retroperitoneal fatty cushion, which leads to narrowing of the aortomesenteric angle and subsequent compression of the duodenum [3]. However, 40.4% of cases are idiopathic [5].

The duodenal compression can be partial or complete, acute or chronic, resulting in the appearance of completely nonspecific symptoms, the most pronounced of which are the following: postprandial abdominal pain (59%), nausea (40%), vomiting with sudden weight loss and electrolyte imbalance (50%), early satiety (32%) and anorexia (18%). Symptoms are aggravated by lying supine after eating and are relieved by assuming the left lateral decubitus, prone or knee-chest position. In addition, symptoms can be masked under more common diseases such as peptic ulcer disease, biliary colic, pancreatitis, and mesenteric ischemia. During physical examination, an asthenic body habitus in this group of patients attracts attention.

The diagnosis of Wilkie syndrome requires a high degree of clinical suspicion, supported by radiographic studies demonstrating compression of the third portion of the duodenum. Computed tomography (CT) scan of the abdomen with contrast, being the gold standard for diagnosing SMA syndrome, allows to reveal not only the acute angle of the SMA from the aorta and the distance between the compressive vessels, but also the dilation of the stomach and duodenum.

Questions about the method of treating Wilkie's syndrome still remain controversial in the world literature. In some cases, the disease responds to successful conservative in the form of adequate nutrition by enteral/parenteral feeding and proper positioning of the patient after feeds.

ISSN 2221-2582

Surgery is resorted to when conservative measures are ineffective or in patients with a long history of progressive weight loss or pronounced duodenal dilatation with stasis and complications [6].

We present a clinical observation of a patient in whom the onset of SMA was caused by weight loss developed as a complication of surgical diseases of the upper gastrointestinal tract, which required surgery in the form of duodenojejunostomy.

CLINICAL OBSERVATION

A 16-year-old girl was admitted to the Filatov Children City Clinical Hospital with complaints of abdominal pain after eating, bloating, nausea, belching, episodes of vomiting, and lack of weight gain.

It is known from the anamnesis that she became acutely ill at the age of eleven, when, due to an intestinal infection, abdominal pain, weakness, diarrhea, blood in stool appeared. After treatment, blood and mucus from the rectum persisted. Due to existing complaints, the girl was repeatedly examined and treated in pediatric gastroenterology departments with a diagnosis of Crohn's disease, where anti-inflammatory therapy with Pentasa was carried out for a long time with a short-term effect; massive hormonal and cytostatic therapy had a negative effect. The discharge of blood and mucus in the stool persisted. During the next aggravation (bleeding), two years after the onset of the disease, a large rectal ulcer was found, which required laparoscopic Soave–Georgeson procedure, during which the rectum with the ulcerative defect was resected. In the postoperative period, anastomotic failure was noted and a retrorectal abscess occurred, which was relieved by drainage and antibiotic therapy. Three months later, the child underwent angiography (Fig. 1), which angiodysplasia of the distal colon, and therefore re-resection of the colon with pull-through procedure and ileostomy was performed. The postoperative period was complicated by the failure of the coloanal anastomosis, the recurrence of intestinal bleeding. The child was rushed to the Istanbul clinic, where a rectovaginal fistula and intestinal bleeding were detected. The repeated Soave pull-through procedure and fistulotomy were performed. However, a complication occurred again in the form of adhesive intestinal obstruction, which was resolved surgically. Two weeks after the operation, bright red blood appeared again from the rectum colonoscopies and rectal biopsies were performed several times (Fig. 2). No evidence of Crohn's disease or other inflammatory bowel diseases was identified; the resulting morphological picture was more consistent with angiodysplasia of the colon.

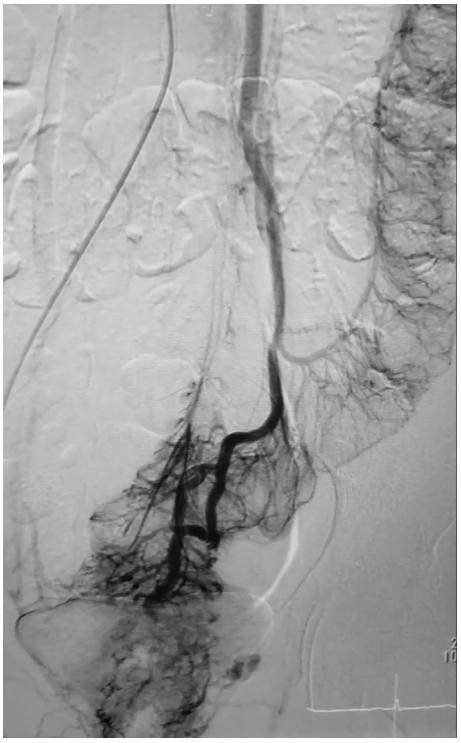

Fig. 1. Angiography showing foci of hypervascularisation in the distal colon, rapid discharge on the veins (before contrasting *V. portae*)

Рис. 1. Ангиография, на которой видны очаги гиперваскуляризаци в дистальных отделах толстой кишки, быстрый сброс на вены (до контрастирования *V. portae*)

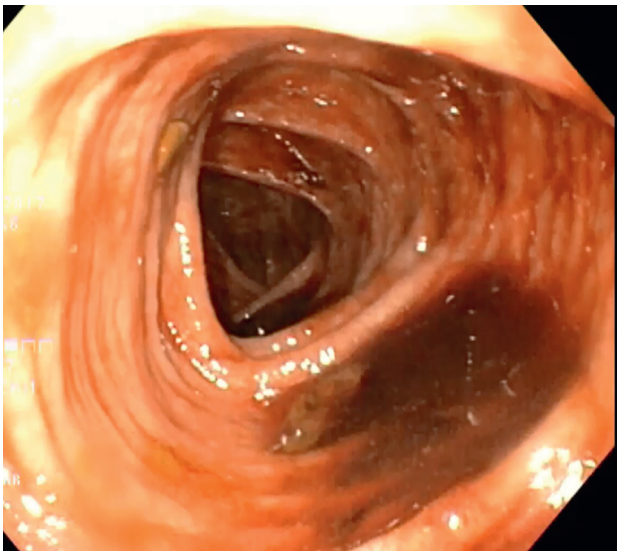

Fig. 2. Colonoscopy. Haemorrhagic content is detected in the lumen of the large intestine

Рис. 2. Колоноскопия. В просвете толстой кишки определяется геморрагическое содержимое

Over the next two months, the girl was treated at the Filatov Children City Clinical Hospital with a diagnosis of angiodysplasia of the colon; multiple ulcers of the colonic mucosa; chronic intestinal bleeding; posthemorrhagic anemia of I–II degree; ileostomy carrier; a follicular cyst of the right ovary. Operated: midline laparotomy, adhesiolysis, resection of the right ovarian cyst, colectomy. Ileorectal anastomosis, ileostomy. The postoperative period was difficult. A year later, the child underwent ileostomy closure. There were no complications. The wound healing is complete. The intestinal passage recovered in the first 24 hours. Two months later, the girl was hospitalized again in an emergency with a diagnosis of chronic ulcerative proctitis, rectovaginal fistula, intestinal bleeding. During sigmoidoscopy and vaginoscopy, a punctate fistula was identified in the vagina, communicating with the rectum in the area of the intestinal anastomosis, and therefore relaparotomy, adhesiolysis, and the end ileostomy formation were performed. The postoperative period proceeded without complications, the intestinal passage was recovered on the first postoperative (po) day.

Upon admission, the child's condition was severe due to the underlying disease. The girl has an asthenic body habitus, the skin is pale, dry in the limb area, the nails are layered, the tongue is covered with a white coating, the teeth are yellow with plaque. On the stoma — liquid small intestinal discharge. Body weight — 36 kg, height — 163 cm. The child has severe malnutrition: BMI — 13.5, average deviation from BMI — 3.69. Physical development is moderate, dysharmonious due to low body weight, the deficiency of which corresponds to the III degree of malnutrition.

Examined: an ultrasound examination of the abdominal organs (abdominal ultrasound) revealed an expansion of the duodenum up to 29 mm. The angle between the SMA and the aorta was 10°, and the diameter of the intestine in the area of the aorto-mesenteric tweezers was 2.5 mm. There were echo signs of adhesive disease, mild ascites and reactive changes in the pancreas. Esophagogastroscopy revealed gastroduodenitis and cardiac insufficiency.

According to the results of upper gastrointestinal tract radiography with barium suspension, there were the following changes (Fig. 3): the gastroesophageal reflux grade II; the stomach is hook-shaped, elongated, the lower part is sac-like dilated, located below the pectinate line, peristalsis is deep, spastic, during which the stomach

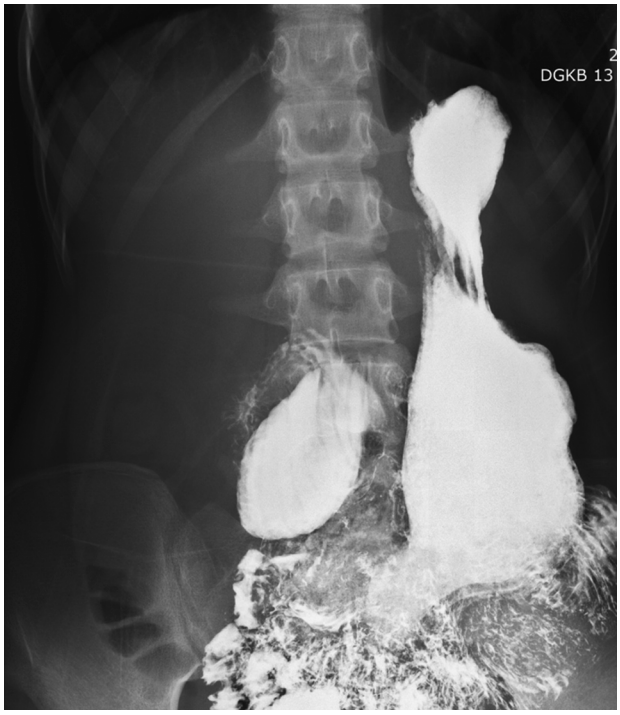

Fig. 3. The duodenum is dilated, positive "double bubble" symptom, the stomach has an hourglass shape

Рис. 3. Нижняя горизонтальная ветвь ДПК расширена, положительный симптом «двойного пузыря», желудок имеет форму песочных часов

takes the shape of an hourglass; dilatation of the lower horizontal branch of the duodenum up to 40 mm, the presence of antiperistaltic waves in it, persistent duodenogastric reflux.

After an examination by a council of doctors, a decision was made to correct protein-energy deficiency fluid and electrolyte imbalance. A course of parenteral nutrition was started under the control of basic indicators of homeostasis. However, against the background of infusion therapy, allergic reactions were observed in the form of a rash, breathing difficulty and tachycardia. Vomiting persisted during enteral load. The weight curve was without dynamics. Taking into account the above, it was decided to perform a surgical intervention on vital indications.

An operation was performed — the relaparotomy; adhesiolysis, Roux-en-Y duodenojejunostomy, enterostomy. The postoperative period proceeded without complications. Due to the severity of the condition, the child was in the intensive care unit (ICU) for 6 days, where she received partial parenteral nutrition — only protein. Upon administration of fat emulsions, the child developed a rash and itchy skin. Infusion, antibacterial, analgesic and symptomatic therapy were also carried out. There was an increase in blood amylase

 ISSN 2221-2582

levels to 174, which required proteolytic therapy with a positive effect. On the second postoperative day, enteral feeding by enterostomy with a medical formula was started. On the same day, passage through the gastrointestinal (GI) tract was restored. On the fourth postoperative day, conservative therapy and feeding by enterostomy were continued, and at the same time, oral feeding was started. When trying to increase the rate of enteral nutrition by enterostomy, an increase in losses through the ileostomy was noted, and therefore feeding by enterostomy was stopped. The child began to eat orally with a gradual increase in the single volume of 80–100 ml. She digested the food, there was no nausea or vomiting. She was discharged on the eighth postoperative day.

Three months after the operation, the child still had reflux complaints (nausea, heartburn). When performing upper GI radiography with contrast, gastroesophageal reflux (GER) persisted, which reached stage III, while the passage was satisfactory, and during endoscopic examination there were phenomena of a catarrhal esophagitis in the lower third of the esophagus. And in endoscopic examination there were phenomena of a catarrhal esophagitis in the lower third of the esophagus. The child underwent Nissen fundoplication.

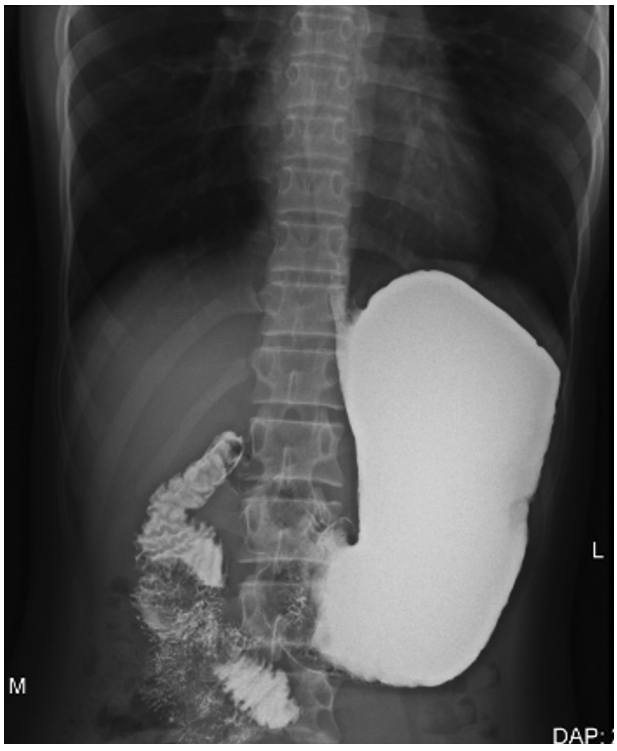

Fig. 4. The duodenum is not dilated, evacuation of contrast agent from the duodenum to the underlying sections is satisfactory, the stomach has a normal shape and size

Рис. 4. ДПК не расширена, эвакуация контрастного вещества из ДПК в нижележащие отделы удовлетворительная, желудок имеет обычную форму и размеры

After 1.5 years, the child had no complaints. According to the tested upper gastrointestinal radiography with contrast, the passage of barium from the stomach and duodenum was satisfactory, there was no gastroesophageal reflux, and the stomach had normal shape and size (Fig. 4).

Over the course of 1.5 years, the girl gained 7 kg, her weight was 43 kg. BMI — 16.2, z-score — 2.14, which corresponds to stage II malnutrition.

In addition, the patient's appetite and skin condition improved significantly, and the normal menstrual cycle was restored.

DICSUSSION

Wilkie's syndrome is characterized by a narrowing of the aortomesenteric angle (from 6 to 25°), and the distance between the compressive vessels is less than 10 mm (can be reduced to 2 mm), whereas normally it is from 10 to 28 mm [7, 8]. In a physiological state and in a straight position, the aorta-SMA angle ranges from 38 to 65° and is maintained by the presence of perivascular adipose tissue [8].

Etiological factors can be either congenital or acquired. Congenital causes include abnormally short or high insertion of the ligament of Treitz, dislocating the duodenum to a cranial position; (promoting the approach of the third part of the duodenum to the apex of the aortomesenteric angle and its compression); a low branching of the SMA from the aorta or the presence of embryonic adhesions [9]. Acquired factors may be multi-etiological. Potential causes include catabolic states such as tumors or burns, as well as diseases causing severe weight loss, such as anorexia nervosa or malabsorption syndrome. Severe trauma or lesions associated with prolonged bedtime rest like brain trauma or spinal cord have also been implied. Finally, postoperative states such as spinal surgery (cast syndrome), esophagectomies, or abdominal aorta aneurism repairs can also be in the genesis of this syndrome [9].

Regardless of the associated factors, a depletion of the fatty cushion in the area of the aorto-mesenteric tweezers occurs, leading to the anatomical changes, mentioned above.

In our case, the patient had a prolonged history without the identification of a specific trigger. She had no history of psychiatric disorders lea-

ding to weight loss, but had major past surgeries or conditions that resulted not only in severe malnutrition but also lengthy bedtime rest.

Patients with SMA syndrome may present in the acute period, with chronic insidious symptomatology or with an acute exacerbation of a chronic disease [10]. The acute form is usually less common and is characterized by signs and symptoms of intestinal obstruction, which quickly resolves with conservative therapy. As a rule, these patients experience a severe loss of body weight (within 1–2 months), and therefore have a positive response to the therapy. Chronic cases may present with progressive, non-specific symptoms that may last for years. The most commonly reported symptoms are long-term abdominal pain, nausea, vomiting bile or food content, vague postprandial indisposition, early satiety, regurgitation, food intolerance, and lack of weight gain or loss weight are the most commonly referred [9]. Persistent vomiting can also lead to dehydration, severe hypovolemia, oliguria, electrolyte imbalances such as hypokalemia and metabolic alkalosis, and ultimately the development of Mallory-Weiss syndrome [9]. Symptoms can aggravate with meals and prone position and relieved with the knee-elbow position or lateral left decubitus[1].

These symptoms may also mimic other diseases such as pancreatitis, peptic ulcers, retroperitoneal or duodenal tumors, eating disorders, or diseases characterized by slow peristalsis such as dermatomyositis or systemic lupus erythematosus [5, 9]. On physical examination, the signs are usually unclear: the abdomen is soft, accessible to deep palpation, abdominal distension and high-pitched bowel sounds are detected [8], which was not the case in our patient.

Due to the low specificity of signs and symptoms, clinical diagnosis requires a high index of suspicion, especially in patients with postprandial pain, vomiting and severe weight loss [11]. Thus, diagnosis is made based not only on clinical evidence but also on radiological findings.

The diagnostic testing of any intestinal obstruction usually begins with a plain abdominal radiography with contrast. The upper gastrointestinal tract radiography with barium reveals such pathognomonic signs of SMA syndrome as dilatation of the lower horizontal branch of the duodenum; a clear cut line that demarks the obliteration of the duodenal lumen by the superior mesenteric artery; pendulum movement of contrast agent proximal to the obstruction; delay of 4 to 6 hours in gastroduodenal transit time, with relief of the obstruction when the patient is placed in the knee-chest position, left lateral decubitus, or with the Hayes maneuver [1, 8–10]. Fibroesophagogastroduodenoscopy (FEGDS) of the upper gastrointestinal tract is usually performed to exclude an intrinsic mechanical bowel obstruction. The only symptom of SMA syndrome during endoscopy of the upper gastrointestinal tract is an extrinsic regular and sometimes pulsatile compression of the duodenal wall in the transition in the middle and lower third. FEGDS does not have high diagnostic accuracy, specificity and sensitivity and, and usually allows the detection of secondary inflammatory changes, which gives a complex view of the disease, as well as on the basis of the identified endoscopic changes allows to develop a set of measures for preoperative preparation and the postoperative period. However, abdominal multi-slice computed tomography (MSCT) scan with intravenous contrast is the gold standard of diagnosis [8], allowing to determine the aortomesenteric angle and the distance between the compressive vessels, extent of duodenal distension and exact point of obstruction, assess of the amount of retroperitoneal fat and exclude other frequent causes of intestinal obstruction (tumors, annular pancreas, aneurysms, etc.) [1, 4].

The purpose of the treatment is to interrupt the pathological cascade composed of weight loss, loss of retroperitoneal fat, narrowing of the aortomesenteric angle, external duodenal compression. Most authors believe that patients with a short period of illness, mild symptoms and incomplete duodenal obstruction should be offered conservative medical treatment [1]. Such measures include insertion of a nasogastric tube for gastric decompression, correction of electrolyte disturbances, and provision of nutrition in the form of a high-calorie oral diet if tolerated, and parenteral nutrition if necessary. The diet can be supplemented with postural maneuvers and means that improve intestinal motility [10].

Surgical intervention is indicated in cases of conservative treatment failure, a prolonged disease with progressive weight loss, and recurrent upper gastrointestinal symptoms (at least once per week for more than six months). The presence of secondary complications such as peptic ulcers and pancreatitis due to biliary reflux against the background of duodenal hypertension is also a reason

for choosing a surgical approach [1, 8–9]. To date, no period has been established during which it would be possible to determine the ineffectiveness of drug treatment [10]. No time limit has yet been set to determine the medical treatment's ineffectiveness [10]. However, some studies report failure rates of 50–70% [1]. The authors came to the conclusion that the severity of the existing symptoms and the presence of secondary complications cannot be corrected in a timely manner using a conservative method, which significantly worsens the patient's quality of life. In such situations, surgical treatment is most appropriate. The operative options include Strong's procedure, gastrojejunostomy, and duodenojejunostomy [5]. Strong's procedure is preferably used in the pediatric population and consists of lysing the ligament of Treitz allowing for mobilization of the duodenum. Although the Strong's procedure is simpler and less invasive than alternatives (no anastomoses are required), it has a high failure rate of 25% [10].

Gastrojejunostomy allows for gastric decompression, but does not eliminate duodenal (DU) obstruction [4]. This method of surgical intervention for chronic duodenal obstruction (CDO) is non-physiological and carries a large percentage of failures and complications: stomach peptic ulcers as a result of biliary reflux, and recurrence of symptoms due to non-decompression of the duodenum, the development of blind loop syndrome, etc. [1, 10]. It is used extremely rarely, and, as a rule, in patients who have previously been repeatedly operated on for CDO, when traditional methods of surgical treatment were not successful.

Duodenojejunostomy was first introduced by Starley in 1910 and over the years it is has become the most frequent treatment with a success rate of 90% [1, 12, 13]. The use of open and laparoscopic approaches has been previously reported, with the latter having advantages such as shorter hospital stay, less trauma and good cosmetic effect [5]. The first laparoscopic duodenoneuneustomy was described in 1998 by Gersin and Heniford, who proved it to be a safe and reproducible technique [9]. As noted by Escaño et al. in their case series analysis, laparoscopic duodenojejunostomy is an effective minimally invasive treatment, with an acceptable rate of postoperative complications and favorable long-term results [6]. This is why it is considered the treatment of choice. In this case, the procedure was performed uneventfully, provided immediate symptomatic relief and significantly improved the patient's quality of life.

CONCLUSION

Wilkie's syndrome poses a really challenging diagnostic task due to its rarity and non-specific symptoms. A high index of suspicion in cases of severe weight loss and upper gastrointestinal symptoms is of utmost importance. Intravenous contrast-enhanced computed tomography is the gold standard for diagnosing SMA syndrome and should be performed in all patients with radiological features of duodenal obstruction at the level of its lower horizontal branch. Early detection can not only avoid the syndrome-associated complications but also improve the prognosis, making conservative measures more likely to be effective. Surgical intervention should be considered in more severe, chronic cases or whenever medical treatment fails. Laparoscopic duodenojejunostomy has proven to be the best modality of choice warranting the best outcomes with a good safety profile. If this group of patients has severe gastroesophageal reflux grade III and esophagitis that are not amenable to conservative therapy, duodenojejunostomy must be supplemented with fundoplication.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

Funding source. This study was not supported by any external sources of funding.

Consent for publication. Written consent was obtained from the patient's legal representatives for publication of relevant medical information within the manuscript.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Информированное согласие на публикацию. Авторы получили письменное согласие законных представителей пациента на публикацию медицинских данных.

REFERENCES / ЛИТЕРАТУРА

1. Lorentziadis M.L. Wilke's syndrome. A rare cause of duodenal obstruction. Ann Gastroenterol. 2011; 24(1): 59–61. PMID: 24714262; PMCID: PMC3959462.
2. Jain N., Chopde A., Soni B. et al. SMA syndrome: management perspective with laparoscopic duodenojejunostomy and long-term results. Surg Endosc. 2021; 35(5): 2029–38. DOI: 10.1007/s00464-020-07598-1. Epub 2020 Apr 27. PMID: 32342220.
3. Chang J., Boules M., Rodriguez J. et al. Laparoscopic duodenojejunostomy for superior mesenteric artery syndrome: intermediate follow-up results and a review of the literature. Surg Endosc. 2017; 31: 1180–5.
4. Silva E., Ribeiro C., Guerreiro S., Domíngues A. Síndrome de Wilkie — a propósito de um caso clínico. Revista Portuguesa de Cirurgia. 2016; 37: 25–8. https://revista.spcir.com/index.php/spcir/article/view/420.
5. Molina Proaño G.A., Cobo Andrade M.M., Guadalupe Rodríguez R.A. et al. Wilkie's syndrome, a missed opportunity. J Surg Case Rep. 2018; 2018(9): rjy246. DOI: 10.1093/jscr/rjy246. PMID: 30254733; PMCID: PMC6149230.
6. Escaño E.A., Valero A.P., de la Iglesia M.T. et al. Superior mesenteric artery syndrome (Wilkie syndrome): Analysis of a series of 7 cases. Cir Esp (Engl Ed). 2020; 98(1): 48–50. English, Spanish. DOI: 10.1016/j.ciresp.2019.02.011. Epub 2019 Apr 12. PMID: 30987762.
7. Claro M., Sousa D., Abreu da Silva A. et al. Wilkie's Syndrome: An Unexpected Finding.Cureus. 2021; 13(12): e20413. DOI: 10.7759/cureus.20413. eCollection 2021 Dec. PMID: 35047254.
8. Bin Waqar S.H., Khan A.A., Mohiuddin O. et al. Perplexing case of Wilkie's syndrome: a rare presentation in a young patient. Cureus. 2019; 11. DOI: 10.7759/cureus.5085.
9. Marinheiro A. Medicina da Universidade de Lisboa. Faculdade de Medicina Universidade de Lisboa. Síndrome de Wilkie: Caso clínico. 2015.
10. Farina R., Foti P.V., Cocuzza G. et al. Wilkie's syndrome. J Ultrasound. 2017; 20(4): 339–42. DOI: 10.1007/s40477-017-0257-2. PMID: 29204239; PMCID: PMC5698186.
11. Ali T., Tomka J., Bakirli I., Bakirov I. Surgical Treatment of Wilkie's Syndrome by Vascular Transposition. Cureus. 2022; 14(4): e24251. DOI: 10.7759/cureus.24251. PMID: 35475250; PMCID: PMC9018456.
12. Güngörer V., Öztürk M., Arslan Ş. A rare cause of recurrent abdominal pain; the coexistence of Wilkie's syndrome and nutcracker syndrome. Arch Argent Pediatr. 2023; 121(1): e202102373. English, Spanish. DOI: 10.5546/aap.2021-02373.eng. Epub 2022 Oct 27. PMID: 36287212.
13. Cienfuegos J.A., Vivas Pérez I., Rotellar F. Co-occurrence of compression syndromes: celiac axis stenosis, superior mesenteric artery and nutcracker syndrome. Rev Esp Enferm Dig. 2020; 112(11): 885. DOI: 10.17235/reed.2020.6945/2020. PMID: 32755144.