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COMPRESSION OF THE CELIAC TRUNK SYNDROME. HISTORY OF THE PROBLEM

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The article presents the main stages of the development of ideas about a fairly rare pathology in clinical practice - the syndrome of compression of the ventral trunk. Until the mid-nineteenth century, reports of the clinical picture of chronic abdominal ischemia were more descriptive. Serial pathoanatomic studies, comparison of anatomical features revealed at autopsy with clinical manifestations accompanying these changes, contributed to the isolation of chronic ischemic disease of the digestive system in a separate nosological form. The discovery of angiography contributed to the significant development of vascular surgery and the isolation of the ventral trunk compression syndrome as an independent disease, since it allowed for a lifetime comparison of the detected changes in blood vessels with the clinical manifestations of the disease. The term "ventral trunk compression syndrome" was first introduced by P. Harjola when describing a case of abdominal pain in a patient with compression of the ventral trunk by the neuroganglionic tissue of the ventral plexus. Later, J. Dunbar confirmed the Association of clinical manifestations of chronic abdominal pain with compression of the ventral trunk by the median arched ligament of the diaphragm. In English-language literature, the disease was named "Dunbar syndrome". The effectiveness of eliminating the symptoms of this disease by surgical dissection of compression structures has been proven. Further study of this problem developed in parallel with the development of surgery in General in accordance with the emergence and introduction of new surgical technologies. The purpose of numerous studies was to study the etiopathogenetic mechanisms of the occurrence of a variety of clinical manifestations of this syndrome and improve the results of its treatment by improving known surgical techniques and introducing new ones.

Keywords: compression of the celiac trunk syndrome; chronic abdominal ischemia; surgical treatment.

СИНДРОМ КОМПРЕССИИ ЧРЕВНОГО СТВОЛА. ИСТОРИЯ ВОПРОСА

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В статье рассмотрены основные этапы развития представлений о достаточно редкой в клинической практике патологии — синдроме компрессии чревного ствола. До середины XIX в. сообщения о клинической картине хронической абдоминальной ишемии больше носили описательный характер. Серийные патологоанатомические исследования, сопоставление анатомических особенностей, выявленных на аутопсии, с клиническими проявлениями, сопровождающими эти изменения, способствовали выделению хронической ишемической болезни органов пищеварения в отдельную нозологическую форму. Открытие ангиографии способствовало значительному развитию сосудистой хирургии и выделению синдрома компрессии чревного ствола как самостоятельного заболевания, так как позволило проводить прижизненное сопоставление выявленных изменений сосудов с клиническими проявлениями заболевания. Термин «синдром компрессии чревного ствола» впервые был введен Р. Нагјоla при описании случая абдоминальной боли у пациентки со сдавлением чревного ствола нейроганглионарной тканью чревного сплетения. Позднее J. Dunbar подтвердил связь клинических проявлений хронической абдоминальной боли с компрессией чревного ствола срединной дугообразной связкой диафрагмы. В англоязычную литературу заболевание вошло под названием «Dunbar syndrome». Была доказана эффективность устранения симптомов данного заболевания путем хирургического рассечения сдавливающих структур. Дальнейшее изучение этой проблемы шло параллельно с развитием хирургии вообще в соответствии с возникновением и внедрением новых хирургических технологий. Цель многочисленных исследований - изучение этиопатогенетических механизмов возникновения многообразия клинических проявлений этого синдрома и улучшение результатов его лечения путем усовершенствования известных хирургических методик и внедрения новых.

Ключевые слова: синдром компрессии чревного ствола; хроническая абдоминальная ишемия; хирургическое лечение.

Celiac trunk compression syndrome is currently understood as the symptom complex arising from extravasal compression of the celiac trunk by the diaphragm's median arcuate ligament, nerve fibers, and/or solar plexus neurofibrous tissue.

This pathology belongs to the group of vascular diseases leading to visceral circulation disorders. The symptom complex caused by stenosing or occlusive lesions of the unpaired visceral arteries is called "angina abdominalis" since it is expressed by the appearance of pain bouts in the abdomen, increasing the functional load on the digestive organs. There are other names for this condition caused by a lesion of the unpaired visceral arteries: chronic intestinal ischemia, "visceral angina," chronic intestinal ischemia, chronic abdominal ischemia syndrome, and others [5].

Until the 19th century, reports devoted to the particular disease's clinical picture were more descriptive. Nevertheless, even then, attempts were made to link clinical manifestations to pathomorphological changes. So, the first mention of the pathology, later known as celiac trunk compression syndrome, most likely occurred in the Italian physician and anatomist G. Lancisi's monograph, "De motu cordis et aneurysmatibus," published in 1728, in which celiac trunk dilatation was present in combination with the disease. The main clinical manifestations of the disease were abdominal pain, flatulence, belching, constipation, and rumbling. The work noted that this pathology's clinical manifestations were inherent in persons with a hypochondriacal and hysterical nervous system structure. The author associated celiac artery dilatation of an aneurysmal nature with a spasm of circular spiral fibers of the artery caused by nerve plexuses. Even earlier, in the middle of the 17th century, the works of the French physician L. Riviere, the section "De Melancholia hypochondrica," mentions pathologic clinical manifestations associated with celiac artery aneurysmal dilatation. Later, when the celiac trunk compression syndrome acquired a separate nosological unit status, one manifestation of this pathology in the form of various neurovegetative disorders (headaches, dizziness, fainting, palpitations, increased sweating, episodes of sudden weakness, feeling of lack of air during household stress) allowed several authors to highlight another sign of this disease - depressive astheno-hypochondriac syndrome [4, 9, 25, 32].

Nevertheless, until the middle of the twentieth century, the study of celiac trunk compression syndrome occurred within the pathological framework associated with impaired visceral circulation. The first studies of the pathology of the aorta's visceral branches occurred in the field of anatomy during pathological studies. The first report on intestinal infarction, preceded by the clinical picture of intestinal ischemia, was made by the French physician J. Despre in 1834. The message was published in the Bulletin of the Paris Anatomical Society. In 1843, a German pathologist and morphologist, F. Tiedemann, at autopsy, revealed that superior mesenteric artery occlusion caused intestinal infarction. The first description of atherosclerotic occlusion of all three unpaired visceral arteries was made in 1869 by C. Chiene. In 1883, C. Thane, first reported isolated obliteration of the celiac trunk. All these changes were found during the autopsy and were not systemic [3, 5, 8, 12, 36].

Messages describing the clinical picture of the digestive system ischemia were also sporadic. From the second half of the 19th century, the number of publications describing chronic abdominal ischemia symptoms increased (G. Bacelli, 1867, H. Huchard, 1893, others). This increase, along with the analysis of pathomorphological changes accompanying these clinical manifestations, allowed clinicians to isolate a separate nosological pathology. These reports traced the authors' attempt to compare visceral vascular lesions' clinical manifestations with intermittent claudication, which was well studied by that time. G. Carriere in 1900 considered this pathology as "intermittent stomach claudication.". The clinical manifestations of the unpaired visceral arteries obliteration were first described in 1901 by J. Schnitzer, who systematized the symptoms in patients with chronic abdominal ischemia and highlighted the symptom triad: abdominal pain, intestinal dysfunction, and progressive weight loss. In 1903, the outstanding Italian physician, G. Bacelli, proposed the name "angina abdominalis" to denote intestinal chronic circulatory disorders, thus highlighting the leading pain syndrome and emphasizing the pathogenetic mechanism's similarity with angina pectoris [3, 5, 13, 16, 21].

At the beginning of the 20th century, largerscale, serial pathological celiac trunk compression syndrome studies began to be performed in which topographic and anatomical changes characteristic of this pathology were traced (B. Lipshutz, 1917; R. George, 1934; N. Michels, 1955). In a study performed in 1917 by B. Lipshutz on 65 corpses to study the variant anatomy of the abdominal cavity vessels. Variants were described when the celiac trunk's proximal part was blocked by the diaphragm. In his study, R. George also reported eight diaphragm imposition cases on the celiac trunk out of 38 autopsies performed. In addition, it was noted that in these cases, the constricting action of the diaphragm reduces the caliber of the celiac trunk at its origin. The publications were purely descriptive, anatomical, and topographic [19, 26, 28].

A significant step that contributed to vascular surgery development, in general, was the discovery of angiography. The first angiography was performed in 1923. A. Moniz from the University of Lisbon was the first to describe the technique of puncture cerebral angiography and the data of the world's first contrast study of the head vessels in a patient with a brain tumor. Several other specialists succeeded in performing contrast angiography of the aorta and various peripheral arteries [15, 35].

The development of angiographic studies made it possible to perform in vivo studies of vascular changes, comparing them with diseases' clinical manifestations. In 1963, P. Harjola described a case of abdominal pain in a young woman, which he regarded as a manifestation of ischemia caused by external compression of the celiac trunk. An angiogram showed stenosis of the celiac trunk. During the operation, a narrowing of the artery by fibrous-altered ganglionic tissue of the celiac plexus was revealed. The artery was released, and the symptoms were arrested. The author called this pathology "celiac axis syndrome" (celiac trunk compression syndrome). Based on this clinical report, many researchers of the problem agreed that celiac plexus neurofibrous tissue may be one of the etiological factors of extravasal compression of the celiac trunk [14, 20].

In 1965, the American radiologist J. Dunbar in collaboration with surgeons F. Beman and S. Marable, described the results of observations in 27 patients with the clinic of chronic abdominal ischemia. In 15 people, the cause of ischemia was extravasal compression of the celiac trunk by the median arcuate ligament, confirmed by angiography and during surgery. Celiac trunk decompression was performed in 13 patients with a positive clinical effect in 12 of them. Control angiography showed a normal vessel course. In the English-language literature, this disease is called "ligamentum arcuatum syndrome" (median arcuate ligament syndrome) or "Dunbar syndrome". Later, in 1966, Marable et al. described the combined compression of the celiac trunk by the median arcuate ligament and the celiac plexus as a new clinical substrate. Several studies have reported the success of surgical treatment by dissecting the arcuate ligament and neuroganglionic tissue of the solar plexus [18, 27].

The first celiac trunk operation in Russia was performed by an outstanding Russian surgeon, academician A.V. Pokrovsky, on May 25, 1962, in a patient with a clinical picture of abdominal angina. Decompression of the celiac trunk and the common hepatic artery was performed. In 1962, for the first time globally, Pokrovsky used a retroperitoneal approach through the chest and diaphragm to perform manipulations on the thoracoabdominal aorta and visceral arteries. This advanced technique quickly gained popularity among domestic surgeons. It became the primary method of performing reconstructive interventions on the aorta and its branches, having received the name "Russian access" abroad. Long enough developed by Pokrovsky's techniques were used for many years to perform surgical interventions for celiac trunk decompression [6, 7].

Since the 1970s, the study of various aspects of the celiac trunk compression syndrome began to develop within the walls of the 1st Leningrad Medical Institute named after academician I.P. Pavlov (now the Pavlov St. Petersburg State Medical University). The founder and the permanent leader of this direction was the prominent Russian surgeon, Professor A.M. Ignashov. Over the half-century history of the development of this direction, the institution's staff have conducted many experimental and clinical studies that have provided answers to many theoretical and practical questions on the development of celiac trunk compression syndrome. Created by A.M. Ignashov's team today has the largest and most recognized worldwide experience in this pathology's surgical treatment [2].

The introduction of noninvasive diagnostic methods into medical practice, such as Doppler ultrasound, spiral computed tomography, and magnetic resonance angiography, significantly advanced vascular surgery development, enabling expanded diagnostic searches and conducting larger studies on the prevalence and structure of vascular diseases [1, 11].

Celiac trunk compression stenosis using B-mode ultrasound scanning was first diagnosed by J. Kaude and P. Wright in 1981. In 1982, J. Woodcock and in 1983 Y. Nimura, for the first time, developed and introduced into practice the duplex ultrasound scanning method of the visceral arteries, enabled the nature of vascular lesions to be determined more accurately. Over the past 25 to 30 years, several signs and calculated indicators have been proposed to assess the degree of stenosis of the celiac trunk and mesenteric arteries with Doppler ultrasound. However, there are still no generally accepted criteria for hemodynamically significant stenosis of unpaired visceral arteries, and the data among various authors differ. The most recognized indicators of hemodynamically significant celiac trunk stenosis among clinicians are the criteria proposed by G. Moneta in 1991 [10, 24, 29].

New multi-detector thin section tomographs have significantly improved the ability to obtain highresolution images of the aorta and its branches. The mesenteric vessels are well visualized, and in many cases, computed tomographic angiographic examination avoids traditional angiography [30].

The possibilities of vascular surgery have significantly expanded because of the active introduction of new technologies, including those arising at the intersection of various disciplines. X-ray endovascular surgery has become one of these promising areas in medicine. However, the expectations of this technology's good effect from turned out to be unjustified precisely with extravasal compression of the celiac trunk. The literature reports poor endovascular balloon angioplasty results with or without stenting of the celiac trunk without prior decompression. Complications in the form of restenosis rapid development, occlusion, and clinical manifestation recurrence after endovascular intervention in compression of celiac trunk syndrome due to deformation, migration, and stent fracture because of unfavorable results precluded using this method as an independent method without preliminary celiac trunk decompression. Nevertheless, high efficiency of the hybrid approach was noted in patients with residual stenosis, restenosis, as well as in patients with combined celiac trunk stenosis (compressional and atherosclerotic) after performing surgery to decompress the celiac trunk [17, 34].

The introduction of minimally invasive surgical technologies also affected celiac trunk compression syndrome. For the first time, elimination of extravasal compression of the celiac trunk by a laparoscopic method was performed by S. Roayaie in 2000. This surgical intervention method has not yet acquired large-scale character due to the relatively high risk of massive intraoperative bleeding; however, the literature data analysis enables discussing clinical results comparable to open methods [23, 33].

The literature also contains data on using automated methods in the surgical treatment of celiac trunk compression syndrome. A pioneer in this field was a team of American surgeons led by N. Jaik, who performed a surgical intervention in 2007 using the DaVinci TM surgical system (Institute Surgical, Sunnyvale, California, USA). They performed a robotic laparoscopy in 2007. However, this technique has not yet been adopted due to the technology's high cost of the [22].

A separate treatment area for celiac trunk stenosis has become reconstructive surgery, usually performed by a combined occlusion – compression and atherosclerotic procedure. With isolated compression stenosis of the celiac trunk, reconstructive surgery's indication is degenerative changes in the celiac trunk's wall in the zone of prolonged exposure to compressing tissues. The basic principles of surgical treatment, in this case, do not differ from reconstructive operations performed for atherosclerotic vascular lesions and comprise replacing or bypassing the altered area by restoring disrupted blood flow. The results of such surgical interventions are comparable to those of operations for celiac trunk decompression [31].

These are the main stages in forming modern ideas about the causes and mechanisms of celiac trunk compression syndrome development, approaches to its diagnosis, and treatment of its pathology. This presented brief review does not exhaust the whole variety of events in the study of such a narrow and specific clinical medicine problem. However, it provides a general idea of the main historic milestones in its development.

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