



Cystic Form of Duodenal Dystrophy (Clinical Case)

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Aim: the reason for the publication was the rare occurrence, as well as the non-specificity of symptoms of the cystic form of duodenal dystrophy. The listed features of this disease lead to difficulties in its differential diagnosis and the choice of the optimal method of treatment.

General statements. The report is devoted to the description of a case of successful treatment of a cystic form of duodenal dystrophy — a chronic inflammation of the pancreatic tissue, ectopic in the wall of the duodenum. A 47-year-old patient was admitted to the clinic with complaints of persistent abdominal pain, periodic vomiting, general weakness, weight loss of 20 kg in three months. With the help of computed tomography, the diagnosis was established, the tumor process was rejected, and chronic pancreatitis was detected in the orthotopic pancreas. Due to the presence of changes in the main pancreas, the patient underwent pancreatoduodenal resection. The features of the operation were pronounced infiltrative changes and pronounced vitreous tissue edema, which made it difficult to mobilize the hepatic flexure of the colon and duodenum.

Conclusion. Pancreatoduodenal resection is the optimal surgical intervention for the combination of cystic form of duodenal dystrophy with sub-/decompensated duodenal stenosis.

Keywords: chronic pancreatitis, duodenal dystrophy, pancreatic ectopia, pancreatoduodenal resection

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Кистозная форма дуоденальной дистрофии (клиническое наблюдение)

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Цель исследования: поводом для публикации послужила редкая встречаемость, а также неспецифичность симптомов кистозной формы дуоденальной дистрофии. Перечисленные особенности данного заболевания приводят к трудностям в ее дифференциальной диагностике и выборе оптимального способа лечения.

Основные положения. Сообщение посвящено описанию случая успешного лечения кистозной формы дуоденальной дистрофии — хронического воспаления ткани поджелудочной железы (ПЖ), эктопированной в стенку двенадцатиперстной кишки (ДПК). Пациент 47 лет поступил в клинику с жалобами на постоянную боль в животе, периодическую рвоту, общую слабость, потерю массы тела на 20 кг за 3 месяца. С помощью компьютерной томографии был установлен диагноз, отвергнут опухолевый процесс и выявлен хронический панкреатит в ортотопической ПЖ. Вследствие наличия изменений в основной ПЖ пациенту была выполнена панкреатодуоденальная резекция. Особенности операции были выраженные инфильтративные изменения и выраженный стекловидный отек тканей, затруднявшие мобилизацию печеночного изгиба ободочной кишки и ДПК.

Заключение. Панкреатодуоденальная резекция является оптимальным хирургическим вмешательством при сочетании кистозной формы дуоденальной дистрофии с суб/декомпенсированным стенозом ДПК.

Ключевые слова: хронический панкреатит, дуоденальная дистрофия, эктопия поджелудочной железы, панкреатодуоденальная резекция

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Duodenal dystrophy (DD) is a chronic inflammation of the pancreatic tissue, ectopic in the wall of the duodenum [1]. Ectopia or heterotopia of the pancreatic tissue is its atypical localization, which has its own blood supply and ductal system, without vascular, nervous or anatomical contact with a typically located (orthotopic) pancreas [2, 3].

Duodenal dystrophy is a rather rare disease, the proportion of patients with DD among patients with chronic pancreatitis is 6 % [4].

The disease was first described in 1970 by French authors F. Potet and N. Duclert, and they also proposed the term “duodenal dystrophy” [5].

DD is one of the forms of paraduodenal pancreatitis and is a postnecrotic cyst and/or dilated pancreatic duct with preserved or altered epithelial lining [6, 7].

Currently, according to the nature of the detected component, two forms of the disease are distinguished: cystic and solid variants of DD, which are also stages of the same pathological process. According to this classification, the form of the disease is considered cystic with a diameter of duodenal cysts of more than 1 cm, and solid — with a diameter of less than 1 cm. At present, another classification is used in the world, dividing DD into two types: 1) with concomitant calculous pancreatitis; 2) isolated — without significant changes in the orthotopic pancreas [1]. The cystic form of the disease is diagnosed more often, as a result of which, under the diagnosis of DD, some authors mean this stage of the inflammatory process [8].

According to the histological structure (Heinrich classification), pancreatic heterotopy is divided into three types: type 1 is represented by ducts, acini and islets of Langerhans, type 2 — by ducts and acini, and type 3 — only by pancreatic ducts. An ectopic pancreas that contains neither acinar nor endocrine cells is called myoeptithelial hamartoma, adenomyosis, or adenomyoma [9].

Etiology and pathogenesis

The mechanism of pancreatic heterotopia is not fully understood and is based on multiple theories. On the one hand, there is an opinion that pancreatic ectopia can occur as a result of metaplasia of pluripotent endodermal cells in situ (metaplasia theory) [10]. Other researchers believe that pancreatic heterotopia develops because of translocation of embryonic pancreatic cells to neighboring organs during embryogenesis (dystopia theory) [11].

Thus, the question of the origin of DD is still debatable. Cicatricial changes and postnecrotic cysts in the ectopic pancreas in 85 % are due to exacerbation of chronic pancreatitis on the background of alcohol abuse. However, these changes can also occur in the unaltered gland [12]. The severity of the chronic inflammatory process in aberrant and orthotopic pancreas varies and depends on the localization of heterotopia, as well as the influence of other risk

factors for chronic pancreatitis (alcohol load, smoking, cholelithiasis, previous episodes of acute pancreatitis) [13].

The pathogenesis of the disease also remains not fully understood. There is an opinion that the cystic form of DD occurs as a result of the direct toxic effect of alcohol metabolites on pancreatic cells. This leads to inflammatory infiltration of the ectopic tissue, production of pancreatic secretion, which is partially excreted through the pancreatic ducts. At the same time, the outflow of pancreatic secretions becomes difficult, the ductal system of the pancreas expands, and pancreatic cysts appear [14].

Epidemiology

According to autopsy data, the prevalence of duodenal dystrophy varies from 0.5 to 13.7 %, predominating in males aged 30 to 50 years [15]. Risk factors for the development of DD are alcohol abuse and smoking [7]. The most common localization of DD is the stomach (22–25 %), duodenum (17–36 %), jejunum (15–22 %) [15]. The literature describes single observations of pancreatic ectopia into the esophagus, common bile duct, gallbladder, mesentery of the small intestine, spleen, mediastinum, or fallopian tubes [16].

Clinical findings

The clinical manifestations of DD are very diverse. Basically, the symptoms are associated with compression of nearby organs or the pancreatic duct itself. This is possible when the cysts are larger than 1.5 cm [17].

All patients note recurrent or persistent pain in the epigastric region of varying intensity, 45 % have clinical and instrumental signs of acute pancreatitis, 73 % have weight loss, and 30 % of patients have vomiting due to mechanical stenosis of the duodenum. Biliary hypertension and obstructive jaundice associated with compression of the intrapancreatic part of the common bile duct occur in 13 % of cases [18].

The literature also contains descriptions of the syndrome of portal hypertension and extravasal compression of the inferior vena cava and/or thrombosis against the background of large postnecrotic cysts emanating from both the duodenal wall and the orthotopic pancreas [19].

The question of the existence of this disease as an independent isolated pathology remains debatable; without inflammatory changes in the tissue of the orthotopic pancreas. This is due to the fact that chronic pancreatitis against the background of DD is detected only in 71 % of cases [18].

Diagnostics

Despite the possibilities of modern radiation diagnostic methods, it is difficult to confirm the exact diagnosis of DD before surgery. This is due to the rarity of this pathology, low awareness of specialists

in diagnostic services, and the absence of specific clinical manifestations of the disease, which, as it progresses, acquires signs of chronic pancreatitis [20].

Mistakes in diagnostics lead to unreasonably long dynamic follow-up, ending in the development of complications, or to non-radical operations with further relapses of the disease or persistence of pain after surgery [6].

Laboratory research methods are usually not informative, and changes in indicators in clinical and biochemical blood tests confirm the presence of complications. At the same time, an increase in the level of alkaline phosphatase, blood amylase can be observed periodically, and it is also possible to increase enzymes that are markers of damage to hepatocytes, such as: AST, ALT, GGT. In a single observation with DD, an increase in the level of blood tumor markers, such as cancer antigen (CA 19-9) and carcinoembryonic antigen (CEA) was noted [21].

A number of modern domestic and foreign authors argue that the most informative and sensitive methods of instrumental diagnosis of DD are multislice computed tomography (MSCT) with intravenous bolus contrast enhancement, magnetic resonance imaging (MRI) with magnetic resonance cholangiopancreatography (MRCP), as well as endosonography of the pancreas and duodenum [22].

Screening transabdominal ultrasound of the abdominal cavity is considered an insufficiently informative diagnostic method, since it is often difficult to distinguish the wall of the duodenum and determine the exact localization of the cyst against the background of pronounced inflammatory and infiltrative changes in the area under study [23]. Ultrasound visualizes an unevenly compacted, thickened duodenal wall (ranging from 16.4 to 67.0 mm) due to the presence of cystic formations with thin septa that deform the intestinal lumen. When performing duplex scanning, the blood flow in the capsule and septa of formations is not detected [19]. The length of changes varies from 23.2 to 76.0 mm. Also, signs of chronic pancreatitis of varying severity are often diagnosed. In cases of cystic form of DD against the background of chronic pancreatitis, in 51 % of cases, changes in the head of the pancreas are noted. At the same time, pancreatic hypertension is detected in 47 % of patients, postnecrotic cysts — in 45.1 %, the presence of stones in all parts of the pancreas — in 26 %.

The most informative and sensitive diagnostic method is MSCT with intravenous bolus contrast. The study determines a pronounced thickening of the wall of the duodenum with the presence of cystic changes in the muscle layer. Duodenal cysts are often oblong or bilobed [24]. The use of intravenous contrast agents will make it possible to identify the solid component more clearly and exclude the presence of tumor changes. Cystic changes in the duodenum can be either homogeneous with a density of 5 to 17 HU, and inhomogeneous with a density of

up to 24 HU [23]. Another important criterion for DD in the MSCT is to determine the displacement of the gastroduodenal artery anteriorly and to the left (normally, it is located between the wall of the duodenum and the head of the pancreas), which is one of the specific signs of the disease, as well as to determine the relationship of a cystic or solid component with the tissue of the orthotopic pancreas. In addition, MSCT reveals complications of the disease, such as dilatation of the stomach (which is due to developed duodenal stenosis), as well as an increase in the head of the pancreas (in the presence of exacerbation of chronic pancreatitis), biliary, pancreatic hypertension and calcifications [19]. According to the world literature, the sensitivity of the method is 95 %, and the specificity is 94 % [25].

Another sensitive diagnostic method is MRI with MRCP. The study identifies cystic formations with varying degrees of severity of cystic and solid components. The solid component is under-intensified on T1 and T2 modes, while the cystic component is often hyperintense on T2 and STIR modes and hypointense on T1 mode. Cysts are more often determined in the submucosal layer, have an irregular rounded shape, and periodically merge with each other. In most of them, thin septa are detected that do not accumulate a contrast agent [23]. Another advantage of MRI is the ability to simultaneously perform MRCP, which is necessary to determine the connection of cysts with the pancreatic duct or the pancreas itself, as well as to detect biliary hypertension due to compression of the bile ducts [21]. The sensitivity of the method is 84 %, the specificity is 86 % [25].

For the purpose of differential diagnosis of tumor and inflammatory diseases of the pancreas and duodenum, endosonography of the pancreas and duodenum is often used [26]. The main objective of the study is to identify the thickened wall of the duodenum with the presence of hypoechoic cystic changes, which are mainly localized in the submucosal and muscular layers. Changes characteristic of DD are visualized, such as circular duodenal stenosis, starting from the bulb of the duodenum, calcifications and pseudocysts of the pancreas, stenosis of the common bile duct and/or expansion of the pancreatic duct. If ductal adenocarcinoma is suspected, the diagnosis can be confirmed by fine needle biopsy with histological or cytological studies [21]. The sensitivity of the method is 94 %, and the specificity is 94 % [25].

To detect duodenal stenosis and assess the degree of damage to the duodenum and stomach, esophago-gastroduodenoscopy is performed [1]. During the study, the possibility of installing a nasointestinal probe for nutrition in decompensated duodenal stenosis is evaluated. The advantage of this research method is the possibility of performing a biopsy if an oncological process is suspected in the region of the major duodenal papilla (MDP), pancreatic head or duodenum. In 47.5 % of cases, the medial wall of the duodenum bulges into its lumen with its

pronounced narrowing. In 3.4 % of patients, it is not possible to pass the apparatus distal to the constriction. Concomitant lesions are found in 32 % of patients: erosive esophagitis — in 18.6 %, erosive-ulcerative duodenitis — in 10 %, erosive gastritis — in 18.6 % [1].

Confirmation of duodenal stenosis is an X-ray examination of the upper digestive tract, which diagnoses a slowdown or complete absence of evacuation (more than 24 hours) of the contrast agent from the stomach or duodenum. During stomach radiography, the expansion of the arch of the duodenum is detected in 5.1 % of patients, signs of sub- and decompensated stenosis — in 12.7 % of cases [1].

Morphology

An important role in the differential diagnosis is played by an urgent intraoperative morphological study, in which it is necessary to exclude a malignant lesion of the duodenum, the head of the pancreas. In a planned morphological study, in addition to identifying a lesion in the duodenum, the need to assess the damage to the tissue of the orthotopic pancreas is determined, i.e., the need to exclude an isolated form of the disease [9]. The mucous and submucosal layers of the duodenal wall are often with fibrous changes and polymorphocellular infiltration. The muscular layer is thickened, it has areas of pancreatic heterotopia in the form of acini and ducts, with signs of chronic inflammation around. In addition, cystic formations are detected, located in the muscular membrane, lined with a cylindrical epithelium, which in structure resembles the normal epithelium of the pancreatic ducts. In large cysts, the walls do not have an epithelial lining for a greater extent, and in some areas, they are lined with glandular epithelium, surrounded by fibrous and inflammatory changes. In these cases, we are talking about dilated ducts. With large sizes of ectopic tissue, it is also located in the submucosal layer of the duodenum in close proximity to the MDP. Pancreatic tissue with severe sclerosis, dilated ducts, and signs of chronic inflammation. The submucosa in front of the cysts contains a large number of hyperplastic Brunner's glands [18].

Differential diagnosis

Differential diagnosis of DD is carried out with tumors, inflammatory diseases and congenital anomalies of the pancreas, duodenum, and bile ducts. It is extremely difficult to distinguish between cystic forms of DD and pancreatic diseases such as cystic mucinous tumors, acute pseudotumor pancreatitis, ductal adenocarcinoma, or pancreatic neuroendocrine tumor [26]. DD in its clinical and instrumental picture may also be similar to diseases of the bile ducts, such as cholangiocarcinoma, choledochal cysts [27].

Treatment

Throughout the world, duodenal dystrophy is considered a benign disease, so there are several approaches to the treatment of this pathology. In the case of an uncomplicated form of duodenal dystrophy, the clinical manifestations of which are a non-permanent, moderately pronounced pain syndrome, conservative treatment is used, the basis of which is a strict diet (restriction of fatty, fried, high-protein foods) and the exclusion of alcohol and smoking [18]. Drug therapy includes the appointment of painkillers (non-steroidal anti-inflammatory analgesics, with severe pain — narcotic analgesics), proton pump inhibitors, enzyme preparations [13].

In a more progressive form of DD (i.e., in chronic pancreatitis with the presence of minor cystic dystrophy), the use of a prolonged synthetic analogue of somatostatin, octreotide, is described [28]. Such treatment is prescribed for a long period (from 3 to 9 months), with full compliance with the recommendations, it leads to a significant clinical improvement (pain syndrome decreases, cysts are reduced, weight is restored) [29].

Currently, with improvement of medical and diagnostic equipment, an alternative method of treating DD is used — endoscopic fenestration or cystoduodenostomy, which are used for strictly defined indications. These indications include certain sizes of duodenal cysts, their number and superficial location [30]. Some researchers believe that endoscopic procedures are not justified because it is sometimes not possible to remove all of the diseased tissue. Endoscopic techniques are effective, for example, in the presence of one or two very large cysts in the wall of the duodenum, and provide an opportunity for histological verification of the diagnosis [31, 32].

With the ineffectiveness of conservative and minimally invasive therapy, surgical intervention remains the only radical method of treatment [33]. The choice of surgical tactics depends on the severity of changes in the duodenum and orthotopic pancreas, as well as on the progression of complications of the disease. The greatest efficiency of surgical treatment is achieved when the substrate of the disease itself is removed, i.e., ectopic tissue of the pancreas and duodenal cysts [33].

A number of modern domestic surgeons (V.I. Egorov et al.) perform pancreatic-preserving surgical interventions in patients with no changes in the orthotopic pancreas [25]. These interventions are resection of the stomach with cysts, resection of the vertical branch of the duodenum with single-row continuous duodeno-duodenal anastomosis, total duodenectomy, resection of the duodenum with its replacement with an intestinal insert with suturing of the pancreatic and common bile ducts into the "neoduodenum" [34, 35].

On the other hand, according to Japanese surgeons, segmental resection of the duodenum is permissible just in cases where it is affected only above

the duodenal papilla [22]. This is due to the fact that the reconstruction of a large defect near the MDP contributes to a high risk of anastomotic failure, leakage of bile and pancreatic juice, which leads to serious complications. In addition, the authors are of the opinion that MDP reimplantation is rather complicated and pancreatoduodenectomy is a safer and simpler method of surgical treatment [36].

With such complications of the disease as duodenal stenosis, obstructive jaundice against the background of biliary stenosis, as well as chronic calculous pancreatitis in the orthotopic pancreas, the question of surgical treatment is not in doubt [2, 37]. Pancreatoduodenal resection is performed (with preservation or resection of the pylorus, which is determined by the level of the lesion), or removal of the pancreatic head with resection of the vertical duodenal branch and duodeno-duodenal anastomosis or resection of the pancreatic head with excision of cystic changes in the duodenum [34].

Considering the rarity of the disease, the difficulties of differential diagnosis and determining the optimal surgical tactics, we present our own clinical case report of the successful treatment of a patient with cystic form of DD.

Patient Sh., 47 years old, was admitted to the A.V. Vishnevsky National Medical Research Center of Surgery with complaints of persistent pain in the epigastric region, assessed by a visual analogue scale (VAS) of 6 points, periodic vomiting (1 time in 2–3 days), general weakness, weight loss of 20 kg in 3 months.

From the *anamnesis* it was known that pain in the epigastric region bothered the patient for six months. During the examination in the out-patient clinic at the place of residence, no pathology was detected. After 3 months the patient noted the addition of a feeling of heaviness and fullness in the stomach after each meal, nausea, periodic vomiting, he began to lose body weight. The patient was examined at the district hospital, where, according to the results of ultrasound MRI, MSCT of the abdominal organs with intravenous bolus contrast, a neoplasm of the head of the pancreas was suspected. The man denied alcohol intake; smoking experience — 20 years, 1–2 packs a day. The patient sought medical consultation in A.V. Vishnevsky National Medical Research Center of Surgery.

On examination, the patient's condition was satisfactory. Skin and visible mucous membranes were of normal color. There was no hyperthermia. Breathing was vesicular, breath sounds heard throughout all lung fields, there were no wheezing, respiration rate is 16 per minute. Heart sounds are clear, rhythmic, blood pressure — 130/80 mm Hg. Art. Pulse — 70 per minute. The tongue was clean and moist. The abdomen was not bloated, soft on palpation, painful in the epigastric region, participated in the act of breathing. Peristalsis was heard. There were no peritoneal symptoms. Murphy's percussion test was

negative on both sides. Stool and diuresis — adequate. The patient's height was 180 cm, body weight was 72 kg, BMI — 22 kg/m².

Laboratory tests in admission: complete blood count detected iron deficiency anemia of mild severity (Hb — 104 g/L), biochemical blood test revealed moderate hyperamylasemia (alpha amylase — 184 U/L, pancreatic amylase — 113 U/L), increased level of GGT — 123 U/L, moderate hypokalaemia — 3.4 mmol/L. CEA — 0.8 ng/mL, CA 19-9 < 2 U/mL.

MSCT of the abdominal organs with contrast enhancement. The contour of the pancreas was clear, dimensions: head — up to 28 mm, body — up to 13 mm, tail — up to 24 mm. The main pancreatic duct was not differentiated. Parapancreatic tissue was not infiltrated. Parapancreatic lymph nodes were not enlarged. The wall of the vertical branch of the duodenum was diffusely thickened, edematous with the presence of cysts up to 15 mm and perifocal infiltration (Fig. 1). Conclusion: duodenal dystrophy, signs of duodenal stenosis.

Esophagogastroduodenoscopy. In the wall of the vertical branch of the duodenum, the mucous membrane is pronouncedly edematous, the intestinal lumen is slit-like narrowed, in some places — up to complete overlap, difficult to pass with the apparatus. Conclusion: stenosis of the vertical branch of the duodenum.

X-ray examination of the esophagus, stomach, duodenum. The evacuation of the contrast agent from the stomach at the time of the study did not occur. When examined after 2 hours, the bulk of the contrast agent remained in the stomach, and only a small part was in the duodenum. The vertical branch of the duodenum was narrowed over 42 mm. Conclusion: subcompensated stenosis of the vertical branch of the duodenum with a delay in the passage of the contrast agent.

Endosonography. The structure of the tail and body of the pancreas is somewhat heterogeneous with hyperechoic inclusions up to 2.5 mm in size, but with clear and even contours. The structure of the pancreatic head is heterogeneous, predominantly hypoechoic, the contours were ill-defined and uneven. The size of the zone of infiltrated pancreatic parenchyma reached 45 mm. Infiltrative changes passed to the duodenum (Fig. 2) with thickening of its wall up to 11.0 mm in the bulb and up to 10.0 mm in the vertical branch. Conclusion: signs of paraduodenal pancreatitis; secondary changes in the wall of the duodenum; an increase in the lymph nodes of the hepatoduodenal ligament of an inflammatory nature; ascites; stenosis of the vertical branch of the duodenum.

After the examination and discussion of the patient at the case conference, a clinical diagnosis was made: chronic pancreatitis, duodenal dystrophy, complicated by subcompensated duodenal stenosis.

Taking into account the survey data, the patient underwent surgery — gastro-pancreaticoduodenal resection.

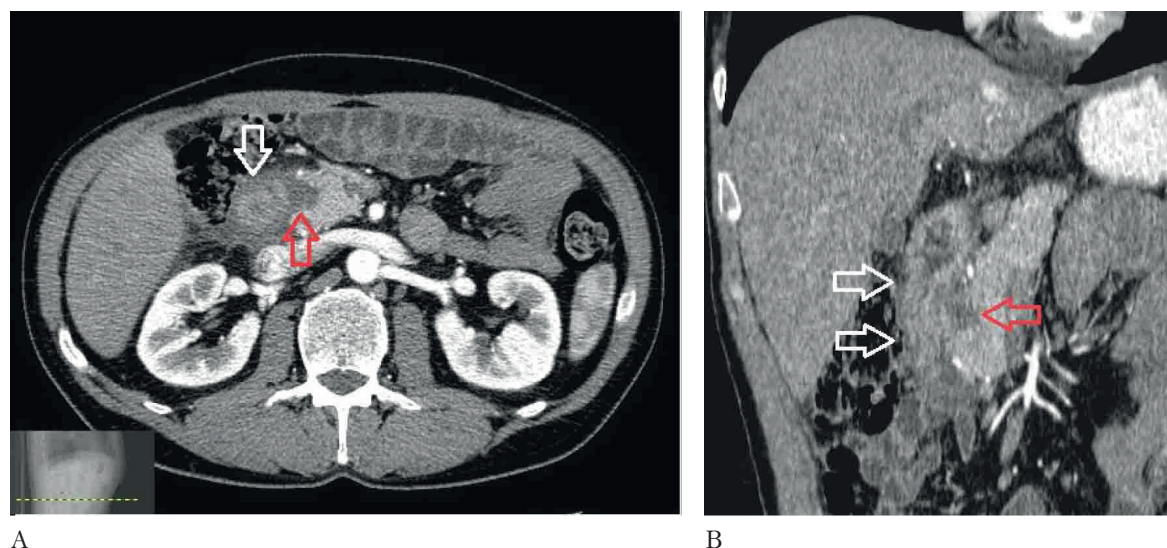


Fig. 1. MSCT with intravenous bolus contrast, arterial phase: A — axial view; B — coronary view (the white arrow indicates a circular thickening of the walls of the duodenum with perifocal infiltration, the red arrow indicates a cyst in the wall of the vertical branch of the duodenum)

Рис. 1. МСКТ с внутривенным болюсным контрастированием, артериальная фаза: А — аксиальная проекция, В — коронарная проекция. Белой стрелкой указано циркулярное утолщение стенок ДПК с перифокальной инфильтрацией, красной стрелкой указана киста в стенке вертикальной ветви ДПК.

With technical difficulties due to pronounced infiltrative changes and vitreous tissue edema, the hepatic flexure of the colon and duodenum was mobilized. The vertical branch of the duodenum was sharply expanded and edematous with a thickened wall up to 1.5 cm. Palpation of the head of the pancreas revealed its dense texture without obvious focal formations. The body and tail of the pancreas were soft, lobed, “juicy”. In the area of the MDP — pronounced edema and tissue infiltration. Hepaticocholedochus was transected above the cystic duct. The antrum of the stomach was resected (2 cm above the pylorus). The jejunum was transected 5 cm from the ligament of Treitz, the pancreas — at the level of the border of the body and the isthmus (Fig. 3). The diameter of the main pancreatic duct was 2 mm. An end-to-end invagination pancreatojejunostomy (PJA) was formed on an isolated loop of the small intestine with a single-row interrupted suture. Forty centimetres away from it, a gastrojejunostomy was formed with a single-row continuous suture. On a loop of the jejunum, put behind the colon, a single-row hepaticojejunostomy “end-to-side” with interrupted sutures was formed, lower — an inter-intestinal anastomosis with a loop of the small intestine carrying pancreatojejunostomy and gastroenteroanastomosis.

Histological examination. In the pancreatic tissue at the border with the duodenum, foci of steatonecrosis with hemorrhages were visualized, in parapancreatic tissue — lymph nodes with reactive changes and foci of steatonecrosis. Focal-spread polymorphocellular chronic inflammatory infiltration, fibrosis of the submucosa, hypertrophy and fibrosis of the muscular membrane were found in the duodenum, with the presence of cystic cavities in it, the walls of which were represented by granulation and fibrous tissue, with diffuse, predominantly lymphoplasmacytic, inflammatory infiltration, and focal hemorrhages (Fig. 4A, B). In adipose tissue — lymph nodes with reactive changes. Conclusion: paraduodenal pancreatitis with severe inflammation of the cystic cavities.

The postoperative period was complicated by acute pancreatitis. The department carried out complex conservative treatment: prevention of stress gastric ulcers (omeprazole), thromboembolic complications (low molecular weight heparins), as well as substitution infusion (isotonic glucose solution, sodium chloride), antibacterial (amoxicillin + clavulanic acid), antisecretory (octreotide), symptomatic therapy (multimodal analgesia, prokinetics). Against the background of the treatment, positive dynamics was noted in the form of pain relief, normalization of blood amylase parameters (on day 1 — 500 U/L, on

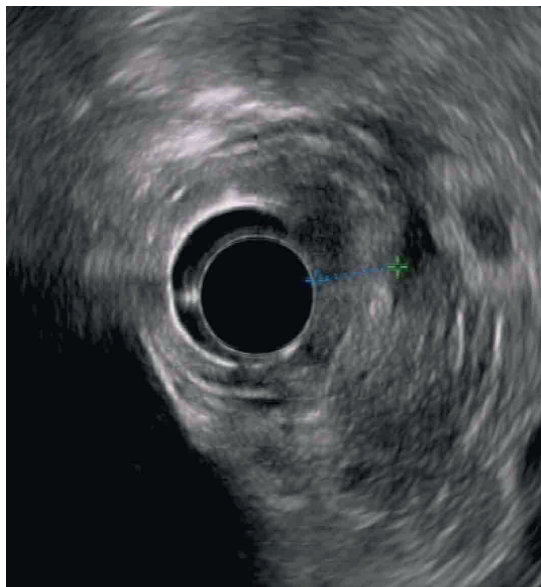


Fig. 2. Endosonographic image of thickening of the duodenal wall up to 1.04 cm and heterogeneous structure of the pancreatic head (the size of the zone of infiltrated pancreatic parenchyma reaches 45 mm)

Рис. 2. Эндосонографическое изображение утолщения стенки ДПК до 1,04 см и неоднородной структуры головки ПЖ (размер зоны инфильтрированной паренхимы ПЖ достигает 45 мм)

day 8 — 50 U/L) and water and electrolyte balance. The drainage installed to the PJE zone was removed on day 4 as the discharge stopped and the level of amylase in it decreased (on day 1 — more than 1000 U/L, on day 3 — 340 U/L). On day 8, the pain syndrome according to VAS was 1 point.

The patient was discharged from the hospital on day 9 after the surgery in a satisfactory condition. After four months he was examined in the out-patient department, the general condition was recognized as satisfactory.

Discussion

Despite numerous works devoted to the study of the criteria for various forms of paraduodenal pancreatitis, currently, most authors note difficulties in making a diagnosis, partly owing to the rarity of the disease. As a result, there are many errors in the diagnosis and treatment of this pathology. In the presented observation, the cystic form of DD clinically manifested as a complication of the disease — increased symptoms of duodenal obstruction without signs of chronic pancreatitis. This indicates the non-specific manifestation of the disease in this particular case, although most patients with DD suffer from chronic pancreatitis of the orthotopic pancreas.



Fig. 3. Macropreparation photo. The lumen of the duodenum is narrowed to 3.5 cm in diameter, its wall in this zone is thickened to 1.2 cm, in the muscle layer there are a large number of cysts filled with hemorrhagic contents, with a diameter of 0.1 to 0.5 cm

Рис. 3 Фото макропрепарата. Просвет ДПК сужен до 3,5 см в поперечнике, ее стенка в этой зоне утолщена до 1,2 см, в мышечном слое большое количество кист, заполненных геморрагическим содержимым, диаметром от 0,1 до 0,5 см

Our example indicates the complexity of the differential diagnosis of patients with cystic duodenal dystrophy at the preoperative stage, which must be taken into account in clinical practice. Before surgical treatment, even if there are convincing data for a postnecrotic cyst of the pancreatic head, a cystic form of duodenal dystrophy cannot be completely excluded. Therefore, in order to most accurately confirm the diagnosis, it is necessary to apply a unified algorithm of therapeutic and diagnostic measures, including MSCT or MRI of the abdominal cavity, endoscopy, endosonography with biopsy (if necessary, exclusion of a malignant process).

It should be noted that surgical intervention is the main method of treatment, and the most rational amount of surgery depends on the level and extent of duodenal damage. When the process spreads to the MDP area and the bulb of the duodenum, it is necessary to perform pancreatoduodenal resection, because reconstruction of a large defect near the MDP is accompanied by a high risk of anastomotic leakage, bile and pancreatic juice leakage.

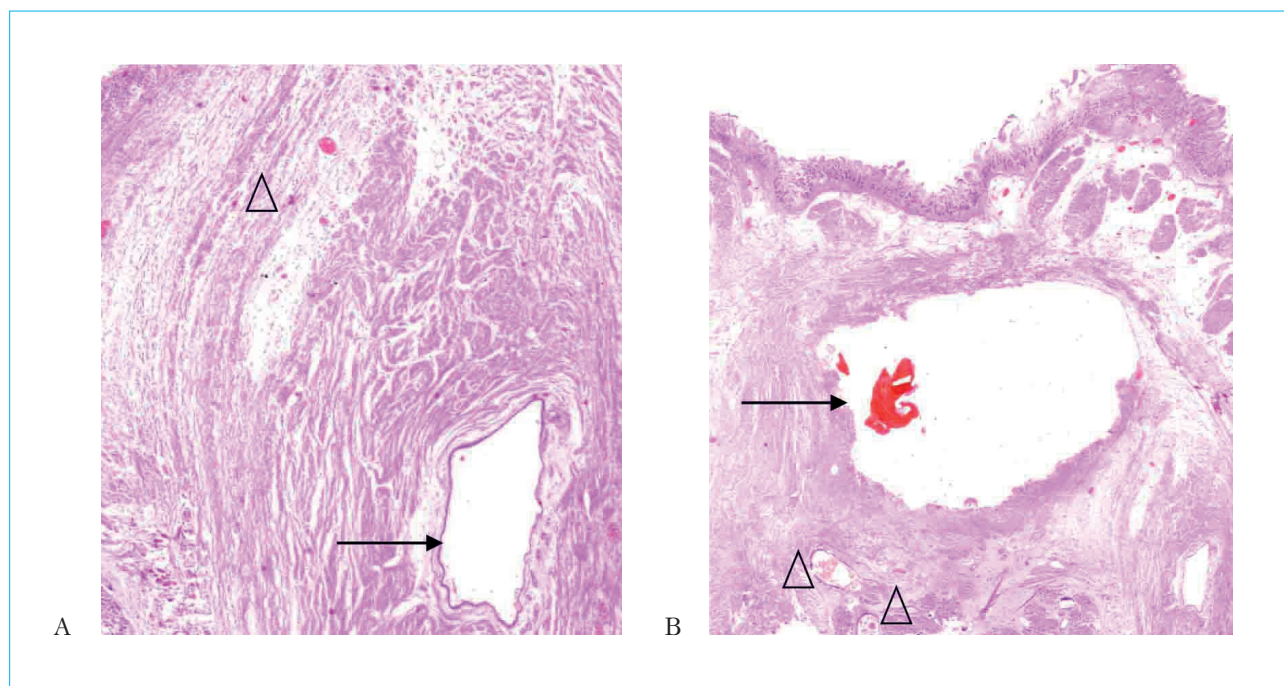


Fig. 4. Micropreparations, stained with hematoxylin and eosin, magnification $\times 50$. A — in the muscle layer of the duodenal wall — dilated heterotopic pancreatic ducts (\rightarrow), lined with cuboidal epithelium, edema and fibrosis of the submucosa (Δ); B — duodenal wall with hypertrophy, edema and fibrosis of its muscle layer containing pseudocysts (\rightarrow) and dilated heterotopic pancreatic ductal structures (Δ)

Рис. 4. Микропрепараты, окраска гематоксилином и эозином, увеличение $\times 50$: А — в собственном мышечном слое стенки ДПК расширенные гетеротопические панкреатические протоки (\rightarrow), выстланные кубическим эпителием, отек и фиброз подслизистой основы (Δ); В — стенка ДПК с гипертрофией, отеком и фиброзом собственного мышечного слоя, содержащего псевдокисты (\rightarrow) и расширенные гетеротопические панкреатические протоковые структуры (Δ)

Conclusion

If a cyst formation is detected in the region of the pancreatic head or duodenum without signs of chronic pancreatitis, it is more likely that an isolated form of DD should be assumed. When confirming

the diagnosis of DD, it is necessary to assess the level of damage to the duodenum and perform either pancreatoduodenal resection or resection of the pancreatic head with excision of cystic changes in the duodenum to prevent severe complications of this disease.

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