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Clinical case of calcifying idiopathic pancreatitis in a child

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ABSTRACT

The article describes a clinical case of idiopathic calcifying pancreatitis in a 14-year-old child, highlighting the challenges of diagnosing and treating chronic pancreatitis in pediatrics. Despite its relatively low prevalence among children (up to 0.5–1 cases per 100,000 population), the condition leads to significant disability. The complexity of diagnosis stems from the non-specific nature of symptoms and the lack of awareness among healthcare providers regarding this issue in children. There are very few studies on chronic pancreatitis in pediatrics available in international databases, and as of 2025, there are no active federal clinical guidelines for pancreatitis in children in Russia.

The presented clinical case outlines the early stages of idiopathic calcifying pancreatitis in a teenager presenting with non-specific symptoms at the onset of the disease. Comprehensive examination revealed atrophy of the pancreas, calcifications, and other characteristic features of chronic pancreatitis. A comprehensive diagnostic investigation was conducted to determine the cause of calcification, including molecular-genetic testing and exclusion of other potential causes such as cystic fibrosis, autoimmune processes, and duct obstruction. However, the exact etiology of the pancreatitis remained unclear.

A key aspect of the article is the discussion about the need to develop diagnostic and treatment protocols for chronic pancreatitis in children, given that individualized approaches are required for each patient. The conclusion emphasizes the importance of further research into the pathogenesis and identification of effective therapeutic strategies to prevent disease progression and maintain patients' quality of life.

Keywords: idiopathic pancreatitis, calcifying pancreatitis, hyperamylasemia, pancreatitis in children

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Клинический случай кальцифицирующего идиопатического панкреатита у ребенка

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АННОТАЦИЯ

В статье представлено описание клинического случая идиопатического кальцифицирующего панкреатита у ребенка 14 лет. Акцентируется внимание на трудностях диагностики и лечения хронического панкреатита в педиатрической практике. Несмотря на низкую распространенность хронического панкреатита среди детей (до 0,5–1,0 случая на 100 000 человек), заболевание имеет серьезные последствия, приводящие к инвалидности. Сложность диагностики обусловлена неспецифичностью симптомов и отсутствием настороженности врачей в отношении данной проблемы у детей. В международной литературе представлено крайне мало исследований по хроническому панкреатиту в педиатрии, а в России на 2025 год отсутствуют действующие федеральные клинические рекомендации по этому заболеванию.

Представленный клинический случай описывает раннюю стадию идиопатического кальцифицирующего панкреатита у подростка с неспецифичными симптомами в начале болезни. Проведенное обследование выявило атрофические изменения поджелудочной железы, кальцификацию и другие характерные признаки хронического панкреатита. Был проведен комплексный диагностический поиск для определения причины кальцификации, включая молекулярно-генетический анализ и исключение других возможных причин, таких как муковисцидоз, аутоиммунные процессы и обструкция протоков. Однако причина панкреатита осталась неясной.

Важным аспектом статьи является обсуждение необходимости разработки алгоритмов диагностики и лечения хронического панкреатита у детей, поскольку данное заболевание требует индивидуального подхода к каждому пациенту. В заключение подчеркивается значимость дальнейшего изучения патогенеза и поиска эффективных методов терапии для предотвращения прогрессирования заболевания и сохранения качества жизни пациентов.

Ключевые слова: идиопатический панкреатит, кальцифицирующий панкреатит, гиперамилаземия, панкреатит у детей

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INTRODUCTION

Chronic pancreatitis (CP) is defined as a long-term, multi-causal inflammatory disease of the pancreas (P) with persistent morphological changes that cause pain and persistent exocrine and endocrine insufficiency. CP may be a consequence of acute recurrent pancreatitis or develop asymptomatically, manifesting itself against the background of irreversible morphological and functional changes in the pancreas. Pancreolitiasis is a rare complication in children. However, if calcification is detected, it can be considered one of the important and reliable criteria for CP. The incidence of CP among adults varies between 27.4 and 50 cases per 100,000 people. In pediatric practice, this diagnosis is much less common, with some sources reporting up to 0.5–1.0 cases per 100,000 children [4, 5]. The most complete etiological and pathogenetic structure of CP is reflected in TIGAR-O classification (toxic-metabolic, idiopathic, genetic, autoimmune, recurrent, acute, obstructive).

The most common causes of CP in children are genetic factors (mutations), abnormalities in pancreatic duct development, effects of trauma and surgery, infectious lesions of the gland, gallstone disease, or chronic cholecystitis with obstruction of excretory ducts [1, 7, 9, 11]. Genetic factors predisposing to the development of CP include following mutations: cationic trypsinogen (encoded by the *PRSS1* gene); transmembrane regulator of the chloride channel CFTR; serine protease inhibitor 1 Kazal (SPINK1), transmembrane regulator CTRC, chymotrypsin C (CTRC). These mutations disrupt the balance of activation and inactivation of trypsinogen in acinar cells and pancreatic ducts, leading to direct damage to the pancreas. Mutations are more often inherited in an autosomal dominant manner, but autosomal recessive forms have also been described. The incidence of hereditary pancreatitis varies from 14 to 79% among patients with chronic and recurrent pancreatitis [7]. Congenital anomalies of *pancreatic ducts* (annular shape, pancreas divisum, etc.) may not manifest themselves clinically and often remain undiagnosed or are discovered by chance; they are less likely to lead to the development of CP than inflammatory processes and obstruction of the excretory ducts. For example, in the United States, according to data for 2022, annular pancreas was diagnosed in only 3.4 cases per 100,000 people. However, if congenital anomalies are present, CP is significantly more likely to be diagnosed in childhood [12].

Pathomorphological processes of CP are based on inflammation with progressive destruction of the acinar apparatus. Morphologically, these changes are characterized by inflammatory infiltration, atrophy, fibrosis, and calcification. According to the Marseille-Rome classification, the most common form is calcifying CP. It is much less common in children than in adult patients, who develop it mainly as a result of systematic alcohol consumption. Calcifications of the pancreas consist mainly of calcium salts and collagen, which acts as a matrix for concernment formation. Inflammation is a key link in calcification pathogenesis. The release of transforming growth factor beta 1 (TGF-β1) by macrophages and lymphocytes triggers the transformation of pancreatic mesenchymal stem cells into fibroblasts, adipocytes, osteoblasts. It also stimulates the release of parathyroid hormone-like protein by acinar cells and fibroblasts, which ultimately leads to concernment formation. Moreover, it is important to note that vitamin D_3 deficiency reduces the activity of osteocalcin, an inhibitor of calcification, and increases the activity of parathyroid hormone-binding protein, which promotes the formation of calcifications, i.e., vitamin D_3 deficiency is a contributor to calcification [8].

For more than 40 years, researchers have been paying special attention to pancreatic stone protein (PSP), also known as lithostatin or Reg1 protein. Initially, it was assumed that PSP inhibits stone formation and that its reduction caused calcific pancreatitis, but in 2006, this theory was called into question [6]. Recent studies have established the lithogenic, rather than lithostatic, role of PSP [14]. The possibility of using this protein as a biomarker for sepsis is also currently being investigated [13, 15].

At present, there is no clear algorithm for diagnosing and treating children with CP. Usually, the main goal in treating any CP is to compensate insufficiency of the exocrine function, so patients receive long-term enzyme replacement therapy. At the same time, indications for surgical treatment may be underestimated. There are also difficulties in timely diagnosis in pediatric patients due to nonspecific complaints. Most often, they are bothered by pain in the epigastric region and left hypochondrium, pain radiating to the back, a feeling of heaviness after eating, nausea, and vomiting. Parents note insufficient weight and height gain in their child due to exocrine pancreatic insufficiency and maldigestion [2, 3]. The most reliable indicator for assessing pancreatic exocrine function at present is the level of fecal elastase-1. The structure of the pancreas can be qualitatively assessed using imaging methods such as endosonography, computed tomography, magnetic resonance cholangiopancreatography (MRCP), and magnetic resonance imaging (MRI).

It is important to note that endosonography is as sensitive as computed tomography [8]. IgG4 is used as a diagnostic marker for autoimmune pancreatitis, but in some cases, even when this pathology is present, IgG4 may remain within normal limits. It is also important to note the need for a detailed and thorough medical history, given the possibility of toxic damage to the pancreas [2, 10].

CASE REPORT

Patient Ch., 14 years old, was admitted to the gastroenterology department on a scheduled basis for examina-

tion, differential diagnosis of the disease, assessment of the functional integrity of the pancreas, dynamics of structural changes, and decision on further treatment tactics. She was of average, harmonious physical development, grew and developed according to her age, and had no chronic diseases.

The first and only clinical manifestation of the disease was back pain, for which the girl sought medical help at her local clinic. An X-ray of the spine revealed radiopaque concretions in the gallbladder and pancreas. The patient was referred to the gastroenterology department of the hospital, where she underwent a comprehensive examination. Multislice computed tomography (MSCT) of abdomen with standard intravenous contrast visualization showed a normally located gallbladder. Attention was drawn to pronounced atrophic changes in the parenchyma as it was significantly reduced in volume and had multiple fatty inclusions, multiple widespread calcifications up to 7.5 mm in size, irregular in shape, localized mainly in the projection of the ductal system (Fig. 1). The parapancreatic tissue was intact. The intra- and extrahepatic bile ducts were not dilated. No free fluid or signs of lymphadenopathy were detected.

The examination revealed a decrease in fecal elastase-1 to 143 μg/g, which was considered a sign of exocrine pancreatic insufficiency. Coprocytogram revealed indirect signs of malabsorption in the form of fatty acid excretion, isolated altered muscle fibers, and iodophilic flora. No endocrine insufficiency was detected. Blood levels of amylase and lipase, transaminases, alkaline phosphatase, and gamma-glutamyltranspeptidase were within the reference range.

An extensive diagnostic search for the cause of pancreatic calcification was conducted. Inherited disease was ruled out by molecular genetic analysis of the most common mutations: cationic trypsinogen (PRSS1), CFTR, serine protease inhibitor 1 Kazal (SPINK1), chymotrypsin C (CTRC), and anionic trypsinogen *PRSS2*. Primary hyperparathyroidism was excluded, as serum parathyroid hormone, calcium, and phosphorus levels were within normal limits. No clinical or anamnestic data suggesting a toxic etiology of pancreatitis were identified. The girl grew up in a well-to-do family, had no concomitant chronic diseases, and did not take any medications on a regular basis. Kidney function was completely preserved, and no metabolic disorders were found. Cystic fibrosis, one of the most common causes of CP in children, was also excluded by means of a sweat test. No conclusive evidence of an autoimmune process was found.

A comprehensive examination revealed a decrease in 25-OH vitamin D levels to 7 ng/ml, which, given the pathogenetic mechanisms, is a predisposing factor for calcification. IgG antibodies to the capsid antigen of the Epstein–Barr virus and to cytomegalovirus were detected, which probably indicated a previous viral infection that could have triggered the development of an autoimmune process or had a direct cytolytic and cytopathic effect. No conclusive evidence of an autoimmune process was obtained. Celiac disease and inflammatory bowel disease were also ruled out during the examination and differential diagnosis.

One of the most common causes of CP is obstruction of pancreatic ducts. This obstruction can appear at the point where the bile duct or pancreatic ducts enter the duodenum as a result of a tumor or ulcerative lesion, swelling of the major duodenal papilla, diverticulitis, and a number of other causes. In order to rule out pathology in the duodenum, the girl underwent esophagogastroduodenoscopy with biopsy. Signs of superficial antral inactive gastritis and superficial duodenitis were detected. A small

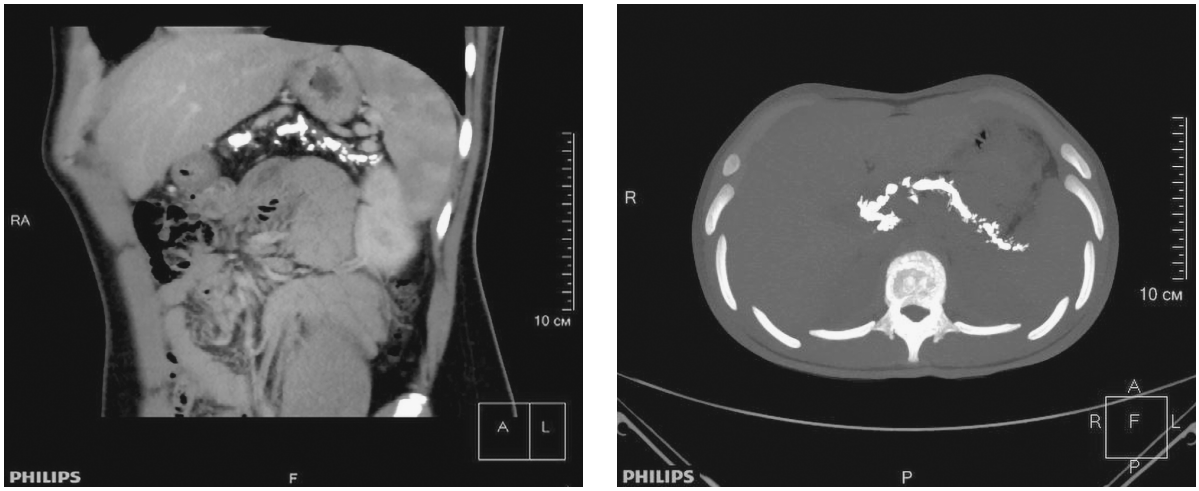

Fig. 1. Calcifications in the projection of the pancreatic duct system in patients with chronic pancreatitis on MSCT of the abdominal cavity

Рис. 1. Кальцинаты в проекции протоковой системы поджелудочной железы у пациентки с хроническим панкреатитом на МСКТ органов брюшной полости

amount of *Helicobacter pylori (HP*) was found in the biopsy samples from the antral section of the stomach.

Based on the results of the examination, the patient was diagnosed with: CP (atrophy, lipomatosis of the pancreas). Pancreolitiasis. Chronic *HP*-associated gastroduodenitis (superficial) in remission.

It is difficult to say what reasons caused the pancreatitis. If calcification was primary and caused obstruction of the ducts, which could have led to atrophy and lipomatosis of the gland, or if calcification was secondary to chronic inflammation. During a long and thorough examination, some aggravating factors were found, like vitamin D_3 deficiency, persistent herpes and Helicobacter infection, and a slow-moving inflammatory process in the stomach and duodenum lining. Taking into account diffuse pancreolitiasis and strictures of the pancreatic ducts, the patient underwent combined stone extraction and drainage of the pancreatic duct. The patient was advised to follow a diet that limits fatty meats, fried and smoked foods. It was recommended to adhere to a normal-calorie, balanced diet enriched with vitamins and minerals. Given the moderate exocrine insufficiency of the pancreas, replacement enzyme therapy was prescribed.

CONCLUSION

When a patient complains of back pain, pancreatitis should be included in the differential diagnosis. Only a targeted collection of complaints and medical history will allow a specialist to suspect this condition in a child with nonspecific complaints at the onset of the disease. Early diagnosis and treatment are the path to long-term and sustained remission.

ADDITIONAL INFORMATION

Author contribution. 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 author declares that he has no competing interests.

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

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в концепцию исследования, сбор, анализ, интерпретацию данных для работы, составление и пересмотр статьи, окончательное утверждение версии для публикации и соглашаются нести ответственность за все аспекты исследования.

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.

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