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A Case of Exacerbation of Chronic Pancreatitis Against the Background of COVID-19 Complicated by Pancreaticopleural Fistula with Enzymatic Pleurisy

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Aim: to draw attention to the diagnosis and treatment of a rare complication of chronic pancreatitis — pancreaticopleural fistula with subtotal enzymatic exudative pleurisy, as well as the possible connection between exacerbation of chronic pancreatitis and pneumonia caused by SARS-CoV-2.
Key points. In primary chronic pancreatitis, pancreaticopleural fistulas develop extremely rarely, while abdominal symptoms may not be expressed, and pleural effusion syndrome comes first in the clinical picture, so it is difficult to establish the true cause of exudative pleurisy in such a situation. The causative agent of COVID-19 has a high affinity for angiotensin-converting enzyme 2 receptors, which are present in large numbers on acinar, ductal, and secretory cells of the pancreas. Obviously, this infection can influence the course of the inflammatory process in chronic pancreatitis.
This clinical observation presents a rare case of exacerbation of chronic pancreatitis with the formation of a pancreaticopleural fistula with subtotal enzymatic pleurisy on the left, which was preceded by pneumonia caused by SARS-CoV-2 in a 47-year-old man who abused alcohol. The study of exudate for amylase content helped to identify the enzymatic cause of pleurisy. Multislice computed tomography of the abdominal organs with bolus enhancement revealed a mass formation in the head of the pancreas with ectasia of the Wirsung duct and common bile duct. It was possible to restore the normal passage of bile and pancreatic secretions, as well as to stop the functioning of the pancreaticopleural fistula using pancreatoduodenal resection. A morphological examination of the macroscopic specimen revealed a diagnosis of pseudotumorous capitate chronic pancreatitis in the acute stage. After surgical treatment, according to ultrasound control, pleurisy was cured. The patient was examined a year later; his condition was consistent with the surgery. There were no clinical, physical, or instrumental data for left-sided pancreaticogenic pleurisy, hypertension of the extrahepatic and pancreatic ducts.
Conclusion. Recurrent exudative pleurisy in chronic pancreatitis with rapid accumulation of exudate in a large volume with a high amylase content is a sign of pancreaticopleural fistula. The development of exacerbation of chronic pancreatitis was influenced by a combination of factors, including coronavirus infection, which suggests further accumulation of clinical material with this combination of pathological processes.
Keywords: chronic pancreatitis, pancreaticopleural fistula, exudative pleurisy, COVID-19
Conflict of interest: the authors declare that there is no conflict of interest.

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Обострение хронического панкреатита на фоне COVID-19, осложнившееся панкреатоплевральной фистулой с ферментативным плевритом

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Цель: обратить внимание на диагностику и лечение редкого осложнения хронического панкреатита — панкреатоплевральной фистулы с субтотальным ферментативным экссудативным плевритом, а также на возможную связь обострения хронического панкреатита с перенесенной пневмонией, вызванной SARS-CoV-2.

Основные положения. При первичном хроническом панкреатите панкреатоплевральные фистулы развиваются крайне редко, при этом абдоминальные симптомы могут быть не выражены, а на первое место в клинической картине выходит синдром плеврального выпота, поэтому установить истинную причину экссудативного плеврита в такой ситуации трудно. Возбудитель COVID-19 обладает высокой тропностью к рецепторам ангиотензинпревращающего фермента 2, в большом количестве представленным на ацинарных, протоковых и секреторных клетках поджелудочной железы. Очевидно, эта инфекция может влиять на течение воспалительного процесса при хроническом панкреатите.
В представленном клиническом наблюдении описан редкий случай обострения хронического панкреатита с формированием панкреатоплевральной фистулы с субтотальным ферментативным плевритом слева, которому предшествовала пневмония, вызванная SARS-CoV-2 у мужчины 47 лет, злоупотребляющего алкоголем. Выявить ферментативную причину плеврита помогло исследование экссудата на содержание амилазы. При мультиспиральной компьютерной томографии органов брюшной полости с болюсным усилением обнаружено объемное образование в головке поджелудочной железы с эктазией вирсунгова протока и холедоха. Восстановить нормальный пассаж желчи и панкреатического секрета, а также прекратить функционирование панкреатоплевральной фистулы удалось с помощью панкреатодуоденальной резекции. При морфологическом исследовании макропрепарата установлен диагноз: псевдотуморозный головчатый хронический панкреатит в стадии обострения. После оперативного лечения по данным ультразвукового контроля наблюдалось излечение плеврита. Пациент осмотрен через год, состояние соответствует перенесенной операции. Клинических, физикальных и инструментальных данных за левосторонний панкреатогенный плеврит, гипертензию внепеченочных и панкреатических протоков не выявлено.
Заключение. Рецидивирующий экссудативный плеврит при хроническом панкреатите с быстрым накоплением экссудата в большом объеме с высоким содержанием амилазы является признаком панкреатоплевральной фистулы. На развитие обострения хронического панкреатита повлияла совокупность факторов, в числе которых была коронавирусная инфекция, что предполагает дальнейшее накопление клинического материала при таком сочетании патологических процессов.
Ключевые слова: хронический панкреатит, панкреатоплевральная фистула, экссудативный плеврит, COVID-19
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Introduction

Pleural effusion syndrome occurs in 300–320 cases per 100,000 population [1]. The structure of pleural effusion is presented as follows: 45.1 % of cases are malignant, 38.1 % are inflammatory (including 15.6 % of tuberculous cases), 7.4 % are non-inflammatory. Rare causes of inflammatory effusions include pancreatogenic (6.1 %) [2]. In surgical practice, pleural effusion in acute pancreatitis occurs in 3–17 % of cases, is an expected complication, and is not difficult to diagnose [3]. In chronic pancreatitis, pleural effusion caused by a pancreaticopleural fistula (PPF) is observed much less frequently (0.4 %) [4]. Thus, in the review by C. King et al. [5] over 38 years reported the presence of only 63 patients with pancreaticopleural fistula. E. Cazzo et al. [6] describe 40 cases over 20 years. We are mainly talking about patients with chronic pancreatitis who abuse alcohol.

The cause of pleural effusion associated with PPF is usually not recognized upon admission, and patients are taken to the therapeutic or pulmonology department with a diagnosis of pleurisy of unspecified etiology. In the Emergency Medical Hospital No. 1 of the city of Omsk during the COVID-19 pandemic from 2020 to 2022, out of 45 cases of such pleurisy, only one was a consequence of PPF. In pleural effusion, the diagnostic search is primarily focused on differentiating parapneumonic, tuberculous, oncological, and less often other causes. The enzymatic nature of pleurisy is most often established untimely [7].

In recent years, publications have appeared in which SARS-CoV-2 is presented as a new etiological factor of acute pancreatitis [8, 9]. The causative agent of COVID-19 has a high tropism for angiotensin-converting enzyme 2 (ACE2) receptors, which are present in large numbers on acinar, ductal and secretory cells of the pancreas. SARS-CoV-2 binds to ACE2 receptors and, with the help of the host enzyme transmembrane serine protease type 2, penetrates into a cell. Next, the virus associates with the endomembrane system by constructing double membrane vesicles from host cell components, where its enhanced replication occurs, while the cell cycle in the nucleus stops, pro-inflammatory factors are activated, and the effects of cytopathy with apoptosis of infected cells increase [10, 11].

In a retrospective cohort study performed by Y. Hadi et al. [12] between January 2020 and July 2021, compared 4420 patients with chronic pancreatitis and COVID-19 with 1,169,773 patients without chronic pancreatitis. They showed

higher mortality and risk of adverse outcomes after COVID-19, which may be due to additional damage to pancreatic tissue.

Aim of the study: to draw attention to the diagnosis and treatment of a rare complication of chronic pancreatitis — pancreaticopleural fistula with subtotal enzymatic exudative pleurisy, as well as to the possible connection of exacerbation of chronic pancreatitis with pneumonia caused by the SARS-CoV-2 virus.

In the case we describe, chronic pancreatitis exacerbation against the background of COVID-19 was complicated by the formation of a pancreaticopleural fistula (PPF) with enzymatic pleurisy.

Clinical case

Patient B., 47 years old, in mid-January 2021 was taken by emergency services to the Therapeutic Department of the City Clinical Emergency Hospital No. 1 due to left-sided pleurisy. Upon admission, he complained of weakness, shortness of breath and palpitations when walking, a dry cough accompanied by pain in the left half of the chest.

Shortness of breath appeared three days before hospitalization, progressively increased, and the abovementioned complaints joined. The local physician referred him for hospitalization in an emergency hospital with suspected exudative pleurisy on the left. For emergency assistance in Medical Unit No. 4, MSCT of the chest organs was performed and with a confirmed diagnosis of "exudative pleurisy" the patient was transported to City Clinical Emergency Hospital No. 1.

In mid-December 2020, he suffered COVID-19 on an outpatient basis with damage to the lungs, a moderate course, verified by chest MSCT and SARS-CoV-2 RNA PCR. X-rays described areas of compaction of the lung tissue like "ground glass" in the lower lobe of the right lung involving 25 % of the parenchyma, the temperature increased to 38.6 °C, objectively the respiratory rate was up to 22 per minute. Drugs that did not have significant pancreatic toxicity were prescribed: favipiravir according to the standard regimen, dexamethasone intramuscularly 8 mg per day for 10 days, levofloxacin 500 mg 2 times a day orally, ambroxol 30 mg 1 tablet 3 times a day. For dexamethasone, pancreatitis is indicated as an undesirable effect, but there are no contraindications for use and conditions for careful use of chronic pancreatitis, the dose and duration of administration are small. X-ray monitoring of convalescence was not performed. From the anamnesis it was known that for 5 years the patient had been suffering from chronic pancreatitis, exacerbations occurred two to three times a year, without pronounced abdominal symptoms, and the patient was mainly bothered by moderate pain in the epigastrium associated with errors in the diet. He did not follow a diet; in the deterioration in the condition, he was treated on an outpatient basis with antispasmodics and proton pump inhibitors; the last exacerbation was noted in October 2020. There was no acute destructive pancreatitis during the course of the disease. The patients abuses alcohol.

Upon admission to the hospital, the patient's condition was relatively satisfactory, consciousness was clear, BMI — 18.6 kg/m^2, pale skin. On auscultation, the breathing was harsh, could not be heard from the third rib, and there was a shortening of the percussion sound. Respiratory rate — 22 per minute. Heart sounds were rhythmic, heart rate — 120 per minute. Blood pressure — 130/80 mmHg. The abdomen was soft and painless on palpation. The liver and spleen were not enlarged. No peripheral edema was observed. Body temperature 37.2 °C.

General blood test: hemoglobin — 111 g/L (norm — 110–160 g/L), erythrocytes — 4.1 × 10^{12}/L (norm — (3.5–5.5) × 10^{12}/L), leukocytes — 12.2 × 10^9/L (norm — 4.0–10.0 × 10^9/L), segmented — 75 % (norm — 50–70 %), monocytes — 5 % (norm — 3–12 %), lymphocytes — 20 % (norm — 20–40 %), ESR — 68 mm/h (norm — 0–15 mm/h), glycemia — 6.0 mmol/L (norm — up to 6.1 mmol/L). Biochemical blood test: AST — 12.4 U/L (norm — 0–40 U/L), ALT — 12.6 U/L (norm — 0–40 U/L), creatinine — 77.5 μmol/L (norm — 58–115 μmol/L), total bilirubin — 6.6 μmol/L (norm — 5.0–21.0 μmol/L), serum amylase — 171.8 U/L (norm — up to 100 U/L), CRP — 297 mg/L (norm — 0–5 mg/L), procalcitonin — less than 0.5 ng/mL (norm — 0–0.064 ng/mL), GGT — 70 U/L (norm — up to 55 U/L), serum iron — 7 μmol/L (norm — 34.5–5.83 umol/L), other indicators are normal. Electrophoresis of proteins: albumins — 31 % (norm — 54–65 %), alpha1 — 6.64 % (norm — 2–5 %), alpha2 — 15.8 % (norm — 7–13 %), beta — 10.29 % (norm — 8–15 %), gamma — 35.53 % (norm — 12–22 %). Urine diastasis (January 15, 2020) — 120 U/L (norm — 10–125 U/L).

ELISA testified to a previous coronavirus infection: cutoff index for IgM for COVID-19 — 0.84, for IgG — 17.1, SARS-CoV-2 PCR from the nasopharynx was negative. Mantoux test with 2 TE PPD-L, Diaskin test, HIV ELISA, HBsAg, Anti-HCV were negative.

ECG: sinus tachycardia (100 per minute), the electrical axis of the heart is not deviated, diffuse changes in the myocardium. To identify a possible connection between pleural effusion and myocardial dysfunction, echocardiography was performed — the dimensions of the cavities are not expanded, the systolic and diastolic functions of the myocardium were not impaired, myocardial contractility is satisfactory (ejection fraction — 59 %); along the perimeter of the heart there was a homogeneous echo-free space of up to 3–4 mm in diastole.

Abdominal ultrasound: the pancreas was not visualized (shielded by gas), only a large hydrothorax on the left was described.

Chest MSCT showed signs of left-sided exudative pleurisy of large volume (thickness of the layer between the pleural layers was 75 mm), subtotal compression of the left lung, enlargement of the intrathoracic lymph nodes — up to 18 mm. In the right lung there were single focal compactions (up to 4 mm in size), areas of heavy compactions of a post-inflammatory nature. Taking into account the X-ray picture, the absence of signs of heart failure, as well as the presence of clinical and laboratory inflammatory response syndrome (low-grade fever, moderate leukocytosis, increased ESR and CRP level, hypergammaglobulinemia), the impression was of tuberculous pleurisy. In addition, tuberculosis can be accompanied by a moderate increase in isoamylase levels, which was observed in the patient.

In the emergency department, the patient's left pleural cavity was catheterized, and 2000 mL of free fluid of a light brown color was evacuated, after which the state of health improved: there was no discomfort in the chest and no palpitations, shortness of breath decreased. Examination of the exudate: slightly turbid, light brown, protein — 8.1 g/L, specific gravity — 1020, Rivalta test is positive, acid-fast mycobacteria and atypical cells were not detected. Microscopy of the drug natively: leukocytes — 30–35, macrophages — 1–3, mesothelial cells — 0–1, erythrocytes — up to 5 per field of view. Microscopy of a stained specimen: segmented leukocytes — 63 %, lymphocytes — 28 %, monocytes — 9 %. Culture for microflora was negative. PCR for Mycobacterium tuberculosis DNA was negative.

Despite the treatment, shortness of breath increased, a progressive accumulation of fluid in the left pleural cavity was physically determined, and a control radiograph on the seventh day of hospitalization showed left-sided hydrothorax to the posterior segment of the forth rib, the median shadow was shifted to the right. During pleural puncture on January 22, 2100 mL of free light brown fluid was obtained. An episode of recurrent large-volume hydrothorax, the absence of acid-fast mycobacteria and signs of bacterial infection, negative PCR for mycobacterial DNA in the pleural contents dated January 15, 2021, the absence of a cardiac history, edema, a normal ECG, the presence of moderate hyperfermentemia and a history of chronic pancreatitis served as the basis for an extensive study of effusion with by determining the level of α-amylase — it was increased to 3651 U/L.

The patient was transferred to the surgical department with suspected left PPF, and therefore MSCT of the PPF with bolus contrast enhancement was performed. The liver was of normal size, homogeneous. The common hepatic duct was dilated to 14 mm. No stones were revealed in the gallbladder. The common bile duct was dilated to 16 mm, and its sharp narrowing is detected in the intrapancreatic section (Fig. 1). Pancreas dimensions: the head — up to 36 mm; in the structure of the head there was a rounded area with clear contours that intensively accumulated a contrast agent in the arterial phase, up to 120 HU, dimensions — 21 × 25 mm; the body of the gland — up to 18 mm; the tail — up to 15 mm, contrast density of the body and the tail of the gland was diffusely reduced (Fig. 2). The Wirsung duct was expanded to 8 mm in the body and tail of the gland, but was not traced in the area of the head (Fig. 3). No free fluid was found in the abdominal cavity.

At the captured levels of the chest in the pleural cavity on the left, the liquid layer thickness was up to 47 mm, the lower lobe of the left lung is reduced in volume due to the area of consolidation of the lung tissue (Fig. 4).

Conclusion: CT picture of a space-occupying lesion in the head of the pancreas; in the differential series, the pseudotumorous form of pancreatitis and cancer of the head of the pancreas should be considered. Choledocho- and virsungectasia. Hepatomegaly. Left-sided hydrothorax, compression atelectasis of the lower lobe of the left lung.

After receiving the MSCT results, the left pleural cavity was drained with a silicone tube with an internal diameter of 6 mm according to Bulau. Within three days, a yellowish effusion of up to 450–510 mL per day was observed with a high amylase content of up to 3472 U/L, which confirmed the presence of a functioning PPF. Due to the reasonable suspicion of pancreatic head cancer and the determination of active surgical tactics, further MRI and magnetic resonance cholangiopancreatography (MRCP) studies were not performed to search for a fistula and a detailed assessment of the condition of the biliary and pancreatic ducts.

Because of the combination of morphological phenomena (the presence of a tumor of the head of the pancreas, pronounced ectasia of the common bile duct and the main duct of the pancreas with a cicatricial stricture of their terminal sections, the presence of signs of PPF) on February 2 the patient underwent pancreaticoduodenectomy. After the operation, the diagnosis was formulated: "chronic fibrosing pancreatitis with predominant damage to the head of the pancreas. Cicatricial stricture of the terminal portion of the common bile duct. Biliary hypertension. Pancreaticopleural fistula on the left".

Pathological examination of surgical material revealed that in the parenchyma of the pancreas there were many false lobules formed due to the diffuse proliferation of connective tissue between the lobules and in their stroma. The stroma was focally represented by proliferations of wide layers of connective tissue of varying degrees of maturity (in some places young, edematous, in others — coarse

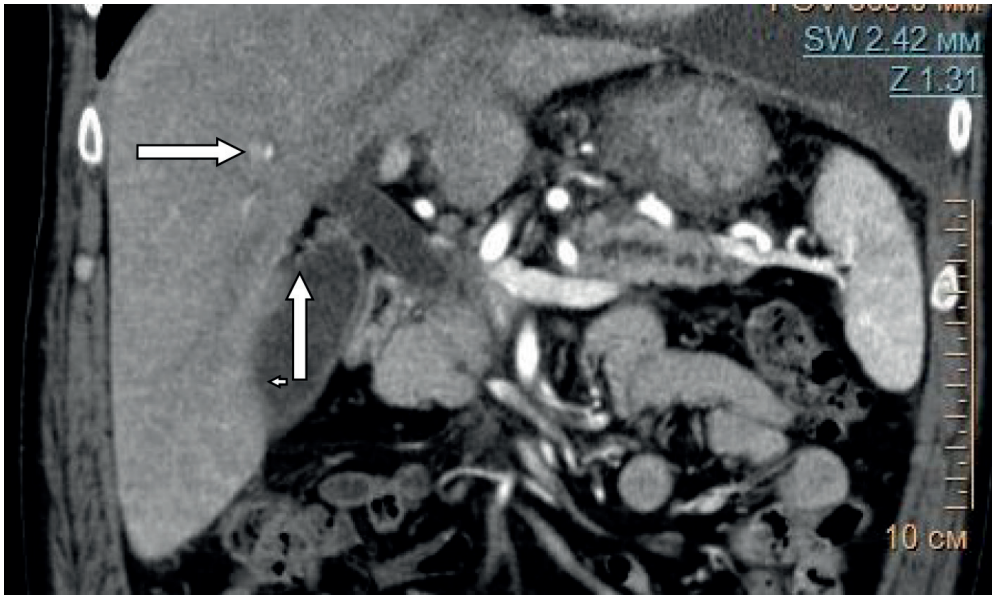

Figure 1. MSCT of the abdominal cavity from January 27, 2021, with bolus contrast enhancement. arterial phase: the common hepatic duct is dilated to 14 mm (top arrow), the common bile duct is dilated to 16 mm, sharply narrowed in the intrapancreatic region (lower arrow)

Рисунок 1. МСКТ брюшной полости от 27.01.2021 г. с болюсным контрастным усилением, артериальная фаза: общий печеночный проток расширен до 14 мм (верхняя стрелка), общий желчный проток расширен до 16 мм, резко сужен в интрапанкреатическом отделе (нижняя стрелка)

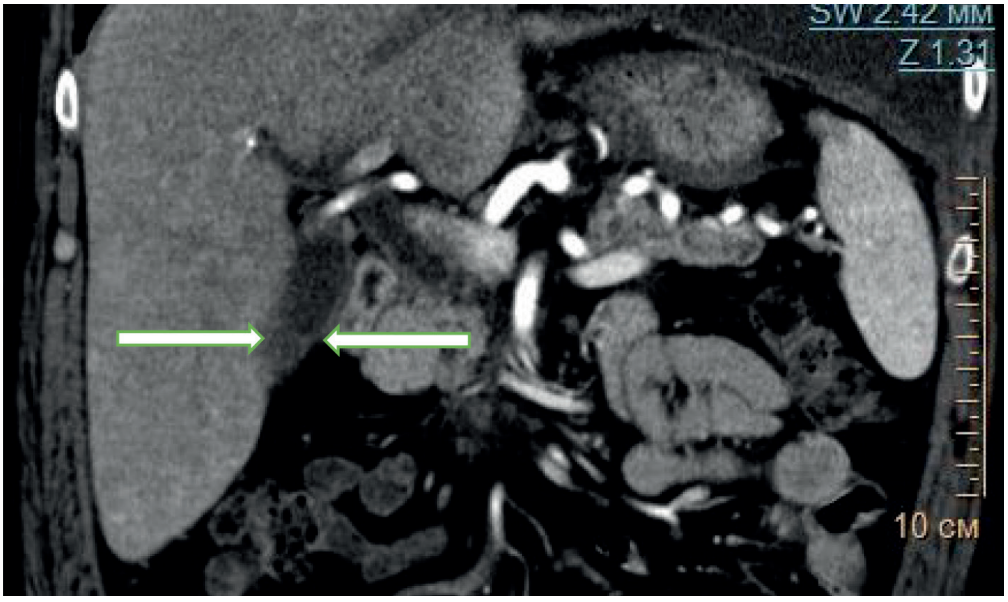

Figure 2. MSCT of the abdominal cavity from January 27, 2021, with bolus contrast enhancement. arterial phase: volumetric formation of the head of the pancreas with clear contours, intensively accumulating a contrast agent in the arterial phase, up to 120 HU, dimensions 21 × 25 mm (between the arrows)

Рисунок 2. МСКТ брюшной полости от 27.01.2021 г. с болюсным контрастным усилением, артериальная фаза: объемное образование головки поджелудочной железы с четкими контурами, интенсивно накапливающие контрастный препарат в артериальную фазу (до 120 HU), размерами 21 × 25 мм (между стрелками)

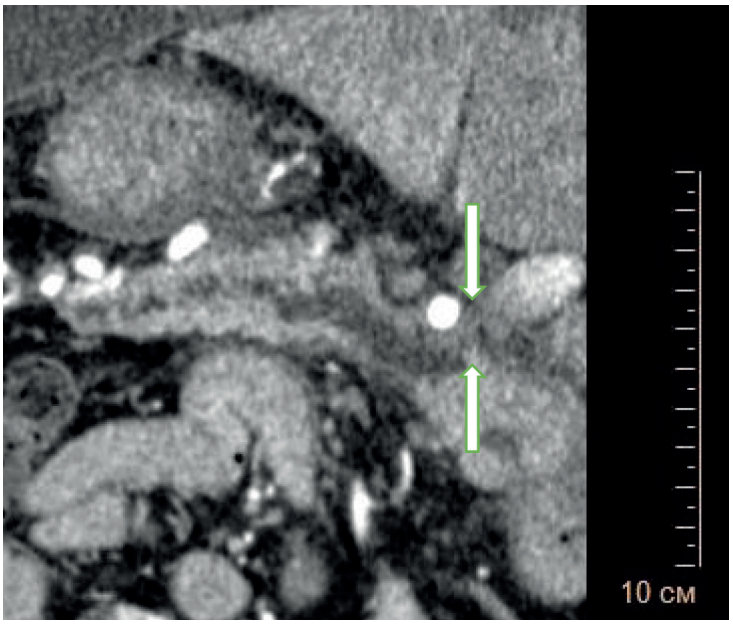

Figure 3. MSCT of the abdominal cavity from January 27, 2021, with bolus contrast enhancement, arterial phase, reconstruction of the pancreas: the main pancreatic duct is dilated up to 8 mm (between the arrows), in the body and tail of the gland, in the area of the head of the gland — is not traced

Рисунок 3. МСКТ брюшной полости от 27.01.2021 г. с болюсным контрастным усилением, артериальная фаза, реконструкция поджелудочной железы: главный проток поджелудочной железы расширен до 8 мм (между стрелок), в теле и хвосте железы, в области головки железы — не прослеживается

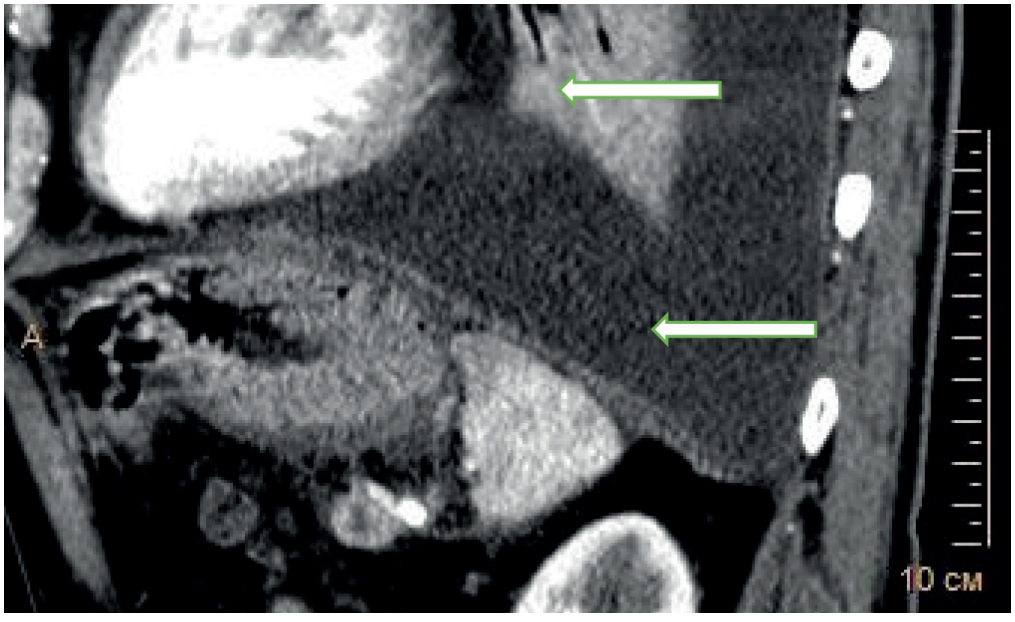

Figure 4. MSCT of the abdominal cavity with the capture of the lower chest from January 27, 2021, with bolus contrast enhancement, arterial phase: atelectasis of the lower lobe of the left lung (top arrow); left-sided hydrothorax (lower arrow)

Рисунок 4. МСКТ брюшной полости с захватом нижней части груди от 27.01.2021 г. с болюсным контрастным усилением, артериальная фаза: ателектаз нижней доли левого легкого (верхняя стрелка); левосторонний гидроторакс (нижняя стрелка)

fibrous), sparsely infiltrated with lymphocytes, histiocytes, and plasmacytes. Ducts of all sizes were unevenly ectatic to varying degrees, their walls show signs of chronic inflammation and sclerosis. PCR testing of SARS-CoV-2 RNA was not performed.

Drug therapy was carried out in the following volume: octreotide, parenteral omeprazole, cefoperazone sulbactam, antispasmodics, crystalloids. The postoperative period proceeded without complications. On control ultrasound of the pleural cavities, no fluid was detected. The patient was discharged for outpatient treatment in satisfactory condition on day 14 after the surgery. He was examined a year later — the condition was consistent with the surgery. There were no clinical, physical, or instrumental data for left-sided exudative pleurisy, hypertension of the extrahepatic and pancreatic ducts.

Discussion

The described observation is interesting as a variant of the course of chronic pancreatitis against the background of COVID-19. Exacerbation of chronic pancreatitis was complicated by the formation of PPF with enzymatic pleurisy on the left, which was the reason for seeking medical help. The clinical picture suggested a differential diagnosis of tuberculous pleurisy, pleural mesothelioma, and a complicated course of coronavirus pneumonia (pleural effusion has been described in 7 % of COVID-19 cases [13]), which were not confirmed by the results of laboratory tests.

Difficulties in diagnosis were associated with the latent course of exacerbation of chronic pancreatitis: the absence of characteristic complaints and increased levels of enzymes in the blood and urine. Intraductal hypertension did not translate into pain due to the outflow of contents into the pleural cavity. In such cases, patients are treated and examined for a long time for pleurisy, and even with a confirmed diagnosis of chronic pancreatitis, a cause-and-effect relationship between these diseases is rarely established. In this observation, the enzymatic genesis of pleurisy was indicated by the rapid accumulation of a light brown effusion in a large volume after pleural puncture, and high levels of amylase in it, corresponding to those in the pancreatic secretion, indicated the presence of PPF. Therefore, with such a combination of diseases, it is necessary to examine the pleural exudate for the content and concentration of amylase and lipase.

Another feature of this clinical situation is that the exacerbation of chronic pancreatitis with the formation of PPF coincided in time with the disease with coronavirus pneumonia. Recent COVID-19 with lung damage is confirmed by the presence of post-inflammatory pneumosclerosis of the right lung and the profile of anti-COVID antibodies (IgM, IgG), and exacerbation of chronic pancreatitis is proven by histological examination of the surgical material: stromal edema and proliferation of young connective tissue of the head of the pancreas, which caused obstruction of the pancreatic part of the common bile duct and Wirsung duct in the area of the head of the pancreas with the formation of biliary and pancreatic hypertension, which was realized in a microfistula with discharge of contents into the pancreas. It was not possible to visualize PPF, but the regression of effusion after surgical elimination of intraductal hypertension with clinical cure confirms the connection between PPF and the course of chronic pancreatitis.

It is possible that the exacerbation of inflammation in chronic pancreatitis with the formation of PPF occurred as a result of additional damage to the pancreas by the SARS-CoV-2 virus due to the combined effect of endogenous and exogenous ligands on immunocompetent cells in the pancreas tissue.

Conclusion

Recurrent exudative pleurisy in chronic pancreatitis with rapid accumulation of exudate in a large volume with a high amylase content is a sign of pancreaticopleural fistula. The development of exacerbation of chronic pancreatitis was influenced by a combination of factors, including coronavirus infection, which suggests further accumulation of clinical symptoms with this combination of pathological processes.

References / Литература

1. *Чучалин А.Г.* Пульмонология. Национальное руководство. Краткое издание. М.: ГЭОТАР-Медиа, 2020. [*Chuchalin A.G.* Pulmonology. National guidelines. Moscow: GEOTAR-Media Publ., 2020. (In Russ.)].
2. *Плаксин С.А., Фаршатова Л.И.* Диагностические и лечебные возможности видеоторакоскопии при плевральных выпотах различной этиологии. *Пермский медицинский журнал.* 2017;34(2):20–5. [*Plaksin S.A., Farshatova L.I.* Diagnostic and therapeutic opportunities of videothoracoscopy in pleural effusion of different etiology. *Perm Medical Journal.* 2017;34(2):20–5. (In Russ.)]. DOI: 10.17816/pmj34220-25
3. *Котельникова Л.П., Плаксин С.А., Кудрявцев П.Л., Фаршатова Л.И.* Легочно-плевральные осложнения панкреатита. *Вестник хирургии им. И.И. Грекова.* 2017;176(3):28–31. [*Kotelnikova L.P., Plaksin S.A., Kudryavtsev P.L., Farshatova L.I.* Pulmonary-pleural complications of pancreatitis. *Grekov's Bulletin of Surgery.* 2017;176(3):28–31. (In Russ.)]. DOI: 10.24884/0042-4625-2017-176-3-28-31
4. *Mihai C., Floria M., Vulpoi R., Nichita L., Cijevschi Prelipcean C., Drug V., et al.* Pancreaticopleural fistula — from diagnosis to management. A case report. *J Gastrointestin Liver Dis.* 2018;27(4):465–9. DOI: 10.15403/jgld.2014.1121.274.ple
5. *King J.C., Reber H.A., Shiraga S., Hines O.J.* Pancreatic-pleural fistula is best managed by early operative intervention. *Surgery.* 2010;147(1):154–9. DOI: 10.1016/j.surg.2009.03.024

6. *Cazzo E., Apodaca-Rueda M., Gestic M.A., Chaim F.H.M., Saito H.P.A., Utrini M.P., et al.* Management of pancreaticopleural fistulas secondary to chronic pancreatitis. Arq Bras Cir Dig. 2017;30(3):225–8. DOI: 10.1590/0102-6720201700030014
7. *Красильников Д.М., Матвеев В.Ю., Абдульянов А.В., Малова И.И., Имамова А.М.* Хронический панкреатит, осложненный панкреатоплевральным свищом. *Анналы хирургической гепатологии*. 2016;21(4):30–4. [*Krasilnikov D.M., Matveev V.Yu., Abdulyanov A.V., Malova I.I., Imamova A.M.* Chronic pancreatitis complicated by pancreaticopleural fistula. *Annaly khirurgicheskoy gepatologii = Annals of HPB Surgery*. 2016;21(4):30–4. (In Russ.)]. DOI: 10.16931/1995-5464.2016430-34
8. *Pandanaboyana S.* Exploring Koch's postulate for SARS-CoV-2-induced acute pancreatitis: Is it all about the ACE? *Br J Surg*. 2021;108(8):879–81. DOI: 10.1093/bjs/znab178
9. *Jabłońska B., Olakowski M., Mrowiec S.* Association between acute pancreatitis and COVID 19 infection: What do we know? *World J Gastrointest Surg*. 2021;13(6):548–62. DOI: 10.4240/wjgs.v13.i6.548
10. *Liu F., Long X., Zhang B., Zhang W., Chen X., Zhang Z., et al.* ACE2 expression in pancreas may cause pancreatic damage after SARS-CoV-2 infection. *Clin Gastroenterol Hepatol*. 2020;18(9):2128–30.e2. DOI: 10.1016/j.cgh.2020.04.040
11. *Shaharuddin S.H., Wang V., Santos R.S., Gross A., Wang Y., Jawanda H., et al.* Deleterious effects of SARS-CoV-2 infection on human pancreatic cells. *Fron Cell Infect Microbiol*. 2021;11:678482. DOI: 10.3389/fcimb.2021.678482
12. *Hadi Y., Shah-Khan S.M., Sohail A.H., Jannat F., Syed A., Humphries C.E., et al.* Su1270: Chronic pancreatitis and COVID-19: Incidence and outcomes. A multicenter research network analysis. *Gastroenterology*. 2022;162(7):564–5. DOI: 10.1016/S0016-5085(22)61337-4
13. *Салимов Д.Ш., Глушков И.В., Воробьев А.А., Крайнюков П.Е.* Поражение плевры при COVID-19: опыт хирургического лечения на протяжении полутора лет пандемии. *Оперативная хирургия и клиническая анатомия (Пироговский научный журнал)*. 2022;6(2):26 31. [*Salimov D.S., Glushkov I.V., Vorobiev A.A., Kraynukov P.E.* Pleura damage in COVID-19, experience of treatment during eighteen months of the pandemic. *Russian Journal of Operative Surgery and Clinical Anatomy*. 2022;6(2):26–31. (In Russ.)]. DOI: 10.17116/operhirurg.2022602126

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