https://doi.org/10.22416/1382-4376-2024-34-4-104-112
UDC 616.36-004-06:616.25-003.217

Recurrent Massive Hydrothorax in a Patient with Decompensated Liver Cirrhosis

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Aim: to demonstrate the need for a detailed differential diagnosis and selection of therapy in a patient with decompensated liver cirrhosis of combined etiology (HCV infection and primary sclerosing cholangitis).
Key points. The patient came to the clinic with complaints of shortness of breath with minimal physical activity, abdominal enlargement, swelling of the legs, yellowness of the skin, and severe weakness. The complaints arose two months after suffering from left-sided focal pneumonia. Laboratory tests revealed signs of systemic inflammation, liver failure, and acute kidney injury. According to the results of instrumental studies, massive hydrothorax was noted in the right pleural cavity. The patient underwent a series of thoracentesis, and a total of about four liters of non-inflammatory pleural fluid was evacuated. Differential diagnosis was based on the presence of dyspnea and respiratory failure. The patient received effective antiviral therapy with drugs using an interferon-free regimen. Subsequently, conservative therapy was carried out, against the background of which the symptoms regressed and the patient's condition improved.
Conclusions. Hepatopleural syndrome is a serious complication in patients with decompensated liver cirrhosis, although it does not always appear secondary to massive ascites. To resolve hepatic hydrothorax, it is necessary to carry out diuretic therapy, replacement transfusion therapy with albumin preparations, and if there is a large amount of fluid in the pleural cavities, therapeutic and diagnostic thoracentesis is recommended.
Keywords: liver cirrhosis, portal hypertension, pneumonia, hydrothorax, hepatopleural syndrome
Conflict of interest: the authors declare no conflict of interest.

For citation: Zemlyanukhina K.S., Tikhonov I.N., Zharkova M.S., Kiseleva O.Yu., Rzayev R.T., Ivashkin V.T. Recurrent Massive Hydrothorax in a Patient with Decompensated Liver Cirrhosis. Russian Journal of Gastroenterology, Hepatology, Coloproctology. 2024;34(4):104–112. https://doi. org/10.22416/1382-4376-2024-34-4-104-112

Рецидивирующий массивный гидроторакс у пациентки с декомпенсированным циррозом печени

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Цель: продемонстрировать необходимость подробного дифференциального диагноза, подбора терапии у пациентки с декомпенсированным циррозом печени сочетанной этиологии (HCV-инфекция и первичный склерозирующий холангит).
Основные положения. Пациентка обратилась в клинику с жалобами на одышку при минимальной физической нагрузке, увеличение живота в объеме, отеки ног, желтушность кожных покровов, выраженную слабость. Жалобы возникли через 2 месяца после перенесенной левосторонней очаговой пневмонии. Лабораторно выявлены признаки системного воспаления, печеночной недостаточности, острого повреждения почек. По результатам инструментальных исследований отмечался массивный гидроторакс в правой плевральной полости. Пациентке проведена серия торакоцентезов, суммарно эвакуировано около 4 литров плевральной жидкости невоспалительного характера. Дифференциальный диагноз проводился исходя из наличия одышки и дыхательной недостаточности. Пациентке проведена эффективная противовирусная терапия препаратами по безинтерфероновой схеме. В дальнейшем проводилась консервативная терапия, на фоне которой симптомы регрессировали, состояние пациентки улучшилось.
Заключение. Гепато-плевральный синдром является грозным осложнением у пациентов с декомпенсированным циррозом печени, при этом не всегда появляясь вторично на фоне массивного асцита. Для разрешения печеночного гидроторакса необходимо проводить диуретическую терапию, заместительную трансфузионную терапию препаратами альбумина, при большом количестве жидкости в плевральных полостях рекомендуется проведение лечебно-диагностического торакоцентеза.

Ключевые слова: цирроз печени, портальная гипертензия, пневмония, гидроторакс, гепато-плевральный синдром
Конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

Для цитирования: Землянухина К.С., Тихонов И.Н., Жаркова М.С., Киселева О.Ю., Рзаев Р.Т., Ивашкин В.Т. Рецидивирующий массивный гидроторакс у пациентки с декомпенсированным циррозом печени. Российский журнал гастроэнтерологии, гепатологии, колопроктологии. 2024;34(4):104–112. https://doi.org/10.22416/1382-4376-2024-34-4-104-112

Introduction

Liver cirrhosis (LC) in the stage of decompensation is characterized by the development of complications, among which the most common are jaundice, variceal bleeding, hepatic encephalopathy, liver failure and infections. The prognosis of patients with cirrhosis is determined precisely by the development of complications, which are the first symptoms of chronic liver disease (CLD) in more than 70 % of patients [1].

This article presents a clinical observation of a patient with liver cirrhosis of autoimmune and viral etiology and massive hydrothorax, which required a detailed differential diagnosis, repeated thoracentesis and selection of conservative therapy.

Clinical case

Patient G., female 58 years old, was admitted to the V.Kh. Vasilenko Clinic of Propaedeutics of Internal Medicine, Gastroenterology and Hepatology (Sechenov University) in April 2023 with complaints of inspiratory shortness of breath with minimal physical activity (such as housework, walking along the corridor), enlarged abdomen, swelling of the legs, yellowing of the skin, severe general weakness. The leading complaint was inspiratory dyspnea.

From the medical history it is known that in 1998, during a routine examination of the patient, markers of hepatitis C were first detected. The route of infection, its duration and genotype of the virus were unknown. She was observed at her place of residence, did not receive antiviral therapy, and the patient's health remained satisfactory.

In 2020, the patient suffered a moderate COVID-19 infection, after which the woman developed nosebleeds, and a clinical blood test revealed deep thrombocytopenia (< 50,000 U/μL) for the first time. She had not undergone a detailed examination at the time of the onset of symptoms. Subsequently, bleeding did not recur.

In February 2022, the patient suffered a second COVID-19 mild infection and received symptomatic therapy. At the end of the month, she noticed an increase in the volume of the abdomen, swelling of the lower extremities up to the middle third of the legs, a decrease in diuresis, and inspiratory shortness of breath appeared with moderate physical activity (climbing stairs). According to ultrasound examination of the abdominal organs, hepatomegaly, uneven contours of the liver, and ascites were noted. From the beginning of March 2022, the patient independently began taking diuretic drugs: spironolactone 200 mg and furosemide 80 mg everyday. Against this background, the severity of edematous-ascitic syndrome decreased, but shortness of breath and weakness persisted.

For further treatment, the patient was hospitalized in a hospital at her place of residence, where she was first diagnosed with liver cirrhosis of viral HCV etiology, class C according to Child — Pugh. Clinical blood test revealed normochromic mild normocytic anemia, thrombocytopenia up to 45,000 U/μL, ESR increased up to 53 mm/h. In a biochemical blood test — an increase in the activity of serum transaminases by 4 times (aspartate aminotransferase > alanine aminotransferase, which is observed at the stage of liver cirrhosis, although it also occurs with alcoholism; the patient denies drinking alcohol), an increase in cholestasis markers: total bilirubin level — up to 53.1 μmol/L due to the direct fraction (normal level — up to 21 μmol/L), gamma-glutamine transpeptidase — up to 375.5 U/L (normal level — up to 73 U/L), alkaline phosphatase — up to 709 U/L (normal level — up to 360 U/L), as well as hypoalbuminemia up to 32 g/L (normal level — more than 35 g/L) as a manifestation of protein synthetic insufficiency (along with hypocoagulation according to standard tests — increased INR, decreased prothrombin index. Increased ferritin level to 850.7 mcg/L (normal level — up to 150 mcg/L) and increased level of C-reactive protein (up to 2.5 norms) can be interpreted as a sign of systemic inflammation in a patient with decompensated cirrhosis. Tests for HBsAg (PCR, anti-HBs, HBsAg) and HIV were negative.

Ultrasound of the abdominal organs shows signs of liver cirrhosis, portal hypertension (splenomegaly, ascites). An endoscopic examination revealed grade 2 varicose veins of the esophagus without the threat of bleeding.

From the life history it is known that patient was born in 1964, she grew and developed normally as a child and did not lag behind her peers. She does not smoke, does not drink alcohol. The woman has higher education, specialty — obstetrician-gynecologist. There is no allergic history. The patient is a widow, two adult daughters are healthy. The mother has a peptic ulcer of the stomach and duodenum, cholelithiasis. The patient's father has a history of arterial hypertension and acute cerebrovascular

accident at the age of 70 years. There are no brothers or sisters. There were no injuries or operations. On the skin of the trunk and upper extremities there are areas of depigmentation similar to vitiligo.

She first contacted the Clinic of Propaedeutics of Internal Medicine in May 2022 with complaints of severe skin itching mainly at night, decreased muscle mass, and general weakness.

During an objective examination, attention was drawn to traces of scratching on the lower extremities, icterus of the sclera; with deep palpation of the abdomen, the liver was moderately painful, protruded from under the edge of the costal arch by 2 cm along the right midclavicular line, dense, lumpy, the edge was rounded.

According to laboratory tests, signs of normochromic anemia, and bilinear cytopenia, liver failure persisted: hyperbilirubinemia — up to 2.5 norms due to both fractions, hypoalbuminemia — up to 30 g/L, decrease in prothrombin level up to 49 %, increase in INR — 1.5. Moderate biochemical activity remained and the level of immunoglobulins class M and G increased to 1.5 norms. For the first time, the genotype of the hepatitis C virus was determined — 1b.

Ultrasound of the abdominal organs showed hepatomegaly, unevenness and tuberosity of the contours of the liver (as one of the signs of cirrhosis), signs of portal hypertension syndrome — splenomegaly 128 × 53 mm, dilatation of the veins of the portal system (portal and splenic), recanalization paraumbilical vein, the presence of porto-systemic shunts, minimal ascites. There were no signs of focal liver formations or thrombosis in the portal vein system.

Noteworthy was the increase in markers of cholestasis [2]. Along with skin itching, female gender and the presence of an autoimmune skin disease (vitiligo), this suggested a combined etiology of liver damage — autoimmune (in particular, primary biliary cholangitis or primary sclerosing cholangitis, possibly in combination with autoimmune hepatitis). The patient's titer of autoantibodies was examined: antimitochondrial (AMA-M2, anti-GP210, anti-SP100), antibodies to liver and kidney microsomes, to smooth muscles — within normal limits, antinuclear factor on the Hep2 cell line — 1 : 160 (clinically insignificant increase). This allowed us to exclude autoimmune hepatitis and primary biliary cholangitis as causative factors of cirrhosis. To further exclude bile duct involvement, the patient underwent magnetic resonance cholangiopancreatography.

An alternation of areas of uneven narrowing and expansion of the intrahepatic bile ducts is determined: segmental ducts of the right lobe — up to 2.8 mm; sectoral ducts — up to 2.2 mm; segmental ducts of the left lobe — up to 2 mm; the right lobar duct — up to 3.5 mm; the left lobar duct — up to 3.7 mm; the common hepatic duct — up to 1.8 mm, the common bile duct — up to 2 mm. There is unevenness of the contours of the bile ducts, the signal from the wall is reduced at T2. This MRI picture made it possible to diagnose a primary cholestatic liver disease — primary sclerosing cholangitis (PSC).

Based on complaints, medical history, and research results, a detailed clinical diagnosis was formulated:

— *Primary disease:* liver cirrhosis of combined etiology (primary sclerosing cholangitis + viral HCV, genotype 1b), class B according to Child — Pugh (8 points), MELD-Na — 14 points, portal hypertension: splenomegaly with hypersplenism syndrome, grade 2 varicose veins of the esophagus, porto-systemic shunts, expansion of portal veins, minimal ascites.

— *Complications of the underlying disease:* liver failure (coagulopathy, hyperbilirubinemia, hypoalbuminemia).

— *Concomitant disease:* vitiligo.

The patient received hepatoprotective therapy, transfusion of a 20 % albumin solution as replacement therapy. For the prevention of hepatic encephalopathy, according to the recommendations of the Ministry of Health of the Russian Federation from 2021, — lactulose and rifaximin alfa, for the primary prevention of variceal bleeding — non-selective beta-blockers [1]. Given the diagnosis of PSC, the patient was prescribed ursodeoxycholic acid drugs, and to reduce skin itching, therapy with selective serotonin reuptake inhibitors (sertraline).

The patient was recommended to start antiviral therapy using an interferon-free regimen at her place of residence.

Next hospitalization in the Clinic of Propaedeutics of Internal Medicine due to increasing shortness of breath and edematous-ascitic syndrome took place in November 2022 after suffering an acute respiratory infection.

There was an increase in edema of the legs to the middle third, abdomen in volume, dullness of percussion sound over the lower parts of both lungs, weakening of breathing below the angle of the scapula on the right, increased vocal tremor above this zone, as well as the appearance of the phenomenon of egolalia in the projection of the Skoda stripe. In laboratory tests — an increase in the level of C-reactive protein.

According to the results of an ultrasound examination, in comparison with the previous hospitalization, the condition of the abdominal organs was without dynamics, however, at least 1000 mL of anechoic fluid was found in the right pleural cavity — it was regarded as the first-time occurrence of hepatic hydrothorax.

A computed tomography scan of the chest organs was performed, which confirmed the presence of effusion in the right pleural cavity, there were no inflammatory changes in the lungs, and the presence of hepatopleural syndrome was stated. Given the absence of severe respiratory failure, it was decided to refrain from performing thoracentesis. A decision was made on conservative diuretic therapy

to relieve the condition. She was discharged with improvement — shortness of breath, ascites and edema decreased.

After discharge as an outpatient, from January 2023, the patient began antiviral therapy according to the regimen of sofosbuvir + velpatasvir 400 mg/100 mg 1 tablet once a day without ribavirin for 12 weeks. At week 12 of the treatment, aviremia was achieved (HCV RNA — negative).

Early February 2023, the patient again developed shortness of breath, a nonproductive cough, and a periodic increase in body temperature to a maximum of 39.5 °C with chills. Blood tests revealed neutrophilic leukocytosis and an increase in the level of C-reactive protein to 68 mg/L. Computed tomography of the chest organs showed right-sided hydrothorax, left-sided focal pneumonia. As therapy, she took a drug from the group of second generation cephalosporins (cefuroxime) for 5 days. There were obvious positive clinical dynamics in the form of a decrease in the severity of respiratory failure, cough, regression of fever and improvement in general well-being.

After suffering from pneumonia for a month, the patient began to notice an increase in jaundice, the appearance of edema in the lower extremities, and an increase in the volume of the abdomen. Objectively: increasing signs of liver failure (hypoalbuminemia — up to 26.9 g/L; coagulopathy — INR 1.6, prothrombin index 50 %). An ultrasound examination showed an increase in ascites to grade 2 and the volume of pleural effusion on the right — about 3.5 liters.

The patient was readmitted to the hepatology department of the Clinic of Propaedeutics of Internal Medicine. An objective examination revealed icterus of the skin and sclera, the presence of multiple telangiectasia, and palmar erythema. Noteworthy were severe shortness of breath, an increase in the frequency of respiratory movements to 24 per minute, a decrease in oxygen saturation to 94 % (when breathing air) as signs of respiratory failure. On examination, there was asymmetry of the chest, lag of the right half during breathing. On auscultation, there was harsh breathing over the lungs, sharply weakened below the angle of the scapula on the right. From the cardiovascular system: the tones were clear, rhythmic. Blood pressure 90/55 mmHg, heart rate/pulse — 64 beats per minute.

Considering the severity of the patient's condition due to respiratory failure as part of hepatopleural syndrome, severe edematous-ascitic syndrome, increasing liver failure and intoxication, it was decided to transfer the patient to the intensive care unit for thoracentesis and complex detoxification and symptomatic therapy. Among the causes of decompensation, repeated infection or its long-term complications, pulmonary embolism were considered

At the time of the initial examination, there was a tendency towards hypotension. Its genesis could be due to the development of pulmonary embolism (there was no clear data for the development of thromboembolism), acute myocardial infarction (there was no characteristic clinical picture and data for focal myocardial damage on the ECG), drug-induced hypotension (the patient took large doses of diuretics, beta-blockers — carvedilol 25 mg/day). It was decided that the patient's examination plan had to include a general clinical blood test, a biochemical blood test, a coagulogram with a mandatory study of D-dimer levels, troponin T and I levels.

After receiving the results of a biochemical blood test (in particular, the level of creatinine with the mandatory determination of the glomerular filtration rate), it was necessary to discuss the possibility of conducting a CT scan of the chest and abdominal cavity with intravenous injection of a contrast agent [3].

According to laboratory data at the time of the examination, acute myocardial infarction was excluded; a clinical blood test showed macrocytic anemia of mild severity, absence of leukocytosis, and thrombocytopenia up to 40,000 U/μL. According to the results of a biochemical blood test, the patient maintained hyperbilirubinemia due to both fractions (total bilirubin — 83.3 μmol/L, direct bilirubin — 44.2 μmol/l), severe hypoalbuminemia — 24.6 g/L. For the first time, a pronounced increase in creatinine level was detected (up to 251 mmol/L), glomerular filtration rate — 18 mL/min, which gave reason to suspect the development of acute kidney injury (hepatorenal syndrome) [4]. A significant increase in the level of C-reactive protein (up to 49.1 mg/L) and ferritin (up to 397 ng/mL) could indicate the presence of a bacterial infection, including infection of the pleural fluid (spontaneous bacterial hydrothorax). According to the coagulogram, hypocoagulation was noted (INR — 1.56, prothrombin time — 17.4 s), increasing the level of D-dimer to 36 norms.

Diuretic therapy was discontinued and infusions of 20 % albumin solution were continued.

Clinically, the patient's condition remained unchanged; severe shortness of breath and O_2 desaturation up to 80 % were still noted.

According to the results of computed tomography performed without contrast injection showed an accumulation of free fluid with a volume of up to 1300 mL in the right pleural cavity; no free liquid or gas in the left pleural cavity; atelectasis

in the lower lobe and third segment of the right lung (Fig. 1). There were no pathological changes in the left lung. Focal infiltrative changes were not detected. The mediastinum was shifted to the left by 10 mm. There was no effusion in the pericardial cavity. In the abdominal cavity, there was an accumulation of fluid up to 500 mL near the liver with streaks along the flanks and in the pelvic cavity. The liver was not enlarged, the surface was finely lumpy, and there was an enlargement of the left lobe. The portal vein was not contrasted; it was difficult to assess in a native study. Splenomegaly up to 128 × 61 × 161 mm was noted. In the large intestine, there was an extensive circular thickening of the wall of the cecum and ascending and proximal third of the transverse sections of the colon up to 15 mm with edema of the adjacent tissue (right-sided segmental colitis?).

In connection with the clinical picture, radiological data, and a pronounced increase in acute phase proteins, it was necessary to exclude the presence of active inflammation in the colon, particularly *Clostridioides difficile* infection, which in patients with liver cirrhosis may manifest itself not as diarrhea and hematochezia, but as an increase in liver failure, encephalopathy and an increase in the blood levels of acute phase proteins. The toxin test came back negative. A colonoscopy was performed showing a picture of portal colopathy; no signs of inflammatory bowel diseases or tumor growth were detected.

The patient underwent evacuation of pleural fluid on the right under ultrasound navigation. About 2500 mL of transparent light-yellow liquid without pathological impurities was obtained, the biomaterial was sent for laboratory testing (primarily to exclude exudate/infection of the pleural effusion). Empirical therapy with a sixth-generation cephalosporin (cefepime) was started intravenously. Based on the results of bacteriological, cytological, and biochemical examinations of the pleural fluid, there was no evidence for the presence of infection. The nature of the fluid corresponded to a transudate; serum-ascitic gradient of albumin concentration was more than 11 g/L.

A day later, the patient again noticed an increase in shortness of breath; a CT scan of the chest was performed showing an increase in the volume of fluid in the right pleural cavity to 500 mL. Thoracentesis was performed again under ultrasound guidance. In total, about 4 liters of fluid were evacuated during two procedures.

After 3 days, the patient was transferred from the ICU with a marked improvement in her condition — shortness of breath, manifestations of respiratory failure, indicators of kidney damage, and the severity of edematous-ascitic syndrome decreased. Respiratory support was indicated to continue in the Hepatology Department.

Considering clinical picture, medical history, examination data in the hospital, and research

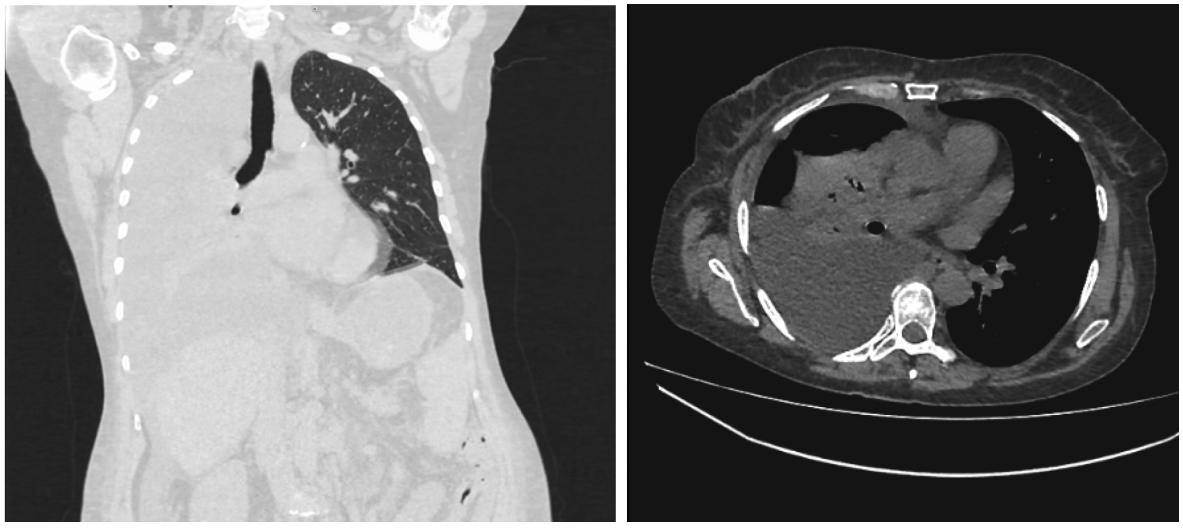

Figure 1. MSCT of the chest organs of Patient G. Massive right-sided hydrothorax: in the right pleural cavity the effusion is up to 1300 mL, in the left pleural cavity the effusion is not detected; there is a shift of the mediastinum to the healthy side; atelectasis of the right lung due to compression by free fluid

Рисунок 1. МСКТ органов грудной клетки пациентки Г. Массивный правосторонний гидроторакс: в правой плевральной полости выпот до 1300 мл, в левой плевральной полости выпот не определяется; отмечается смещение средостения в здоровую сторону; ателектаз правого легкого за счет сдавления свободной жидкостью

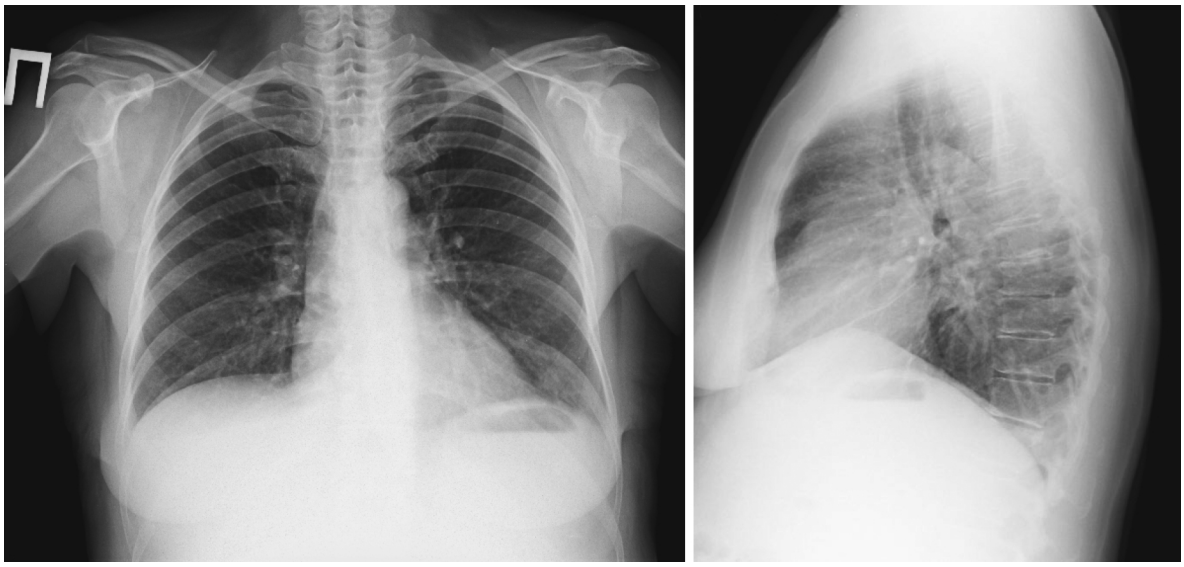

Figure 2. Control radiography of the chest organs of Patient G. (anterior and lateral projections): positive dynamics are noted — a decrease in the amount of free fluid in the right parts of the lungs, as well as a decrease in the severity of mediastinal displacement

Рисунок 2. Контрольная рентгенография органов грудной клетки пациентки Г. (передняя и боковая проекции): отмечается положительная динамика — уменьшение количества свободной жидкости в правых отделах легких, также уменьшение выраженности смещения средостения

data, the cause of decompensation of cirrhosis can be considered an infection suffered in February.

With further treatment in the Hepatology Department, a gradual decrease in the level of creatinine to 68 umol/L was noted — resolution of acute kidney injury, the severity of edematous-ascitic syndrome decreased, there was a tendency to reduce the severity of liver failure: hypoalbuminemia was stopped — the albumin level by the end of hospitalization was 40.8 g/L, a gradual decrease in bilirubin levels and coagulopathy indicators continued. During the period of her stay in the Clinic, the patient did not interrupt antiviral therapy, the selection of all drugs was carried out taking into account drug-drug interactions.

A final clinical diagnosis was made

— *Primary disease:* liver cirrhosis of combined (PSC + HCV) etiology (HCV RNA — negative, antiviral therapy — sofosbuvir/velpatasvir), class C according to Child — Pugh (10 points), MELD-Na — 30 points, portal hypertension: splenomegaly with hypersplenism, grade 2 varicose veins of the esophagus, porto-systemic shunts, expansion of the veins of the portal system, grade 2 ascites.

— *Complications of the underlying disease:* liver failure: coagulopathy, hyperbilirubinemia, hypoalbuminemia. Massive right-sided hydrothorax as part of hepatopleural syndrome. Atelectasis S3, S6-S10 of the right lung. Respiratory failure stage 2 mild. Macrocytic anemia. Protein-energy deficiency. Acute kidney injury from 03.23.2023.

— *Concomitant diseases:* vitiligo.

At the moment, antiviral therapy has been completed, a stable virological response has been achieved — 12 weeks after the end of treatment, HCV RNA is not detected in the blood. The patient feels well; skin itching, shortness of breath do not bother, there is no swelling. She continues to take ursodeoxycholic acid, carvedilol, lactulose, and courses of rifaximin alfa. Compensation of the underlying disease is noted, hydrothorax is absent.

Dynamic monitoring continues — regular screening for hepatocellular carcinoma (ultrasound of the abdominal organs and testing the level of alpha-fetoprotein every 6 months) [5].

Discussion

There are four major pulmonary complications that can occur in patients with chronic liver disease: hepatopleural syndrome, hepatopulmonary syndrome, portopulmonary hypertension, and pneumonia. Pulmonary complications of cirrhosis can occur hidden against the general background

of multiorgan/systemic dysfunction in a patient with decompensated cirrhosis and often remain undiagnosed, which worsens the patient's prognosis, as well as outcomes after orthotopic liver transplantation [6, 7].

Hepatopleural syndrome is an accumulation of transudate in the pleural cavities in patients with decompensated cirrhosis in the absence of diseases of the heart, pleura and lungs (20 % of cases of hydrothorax). It can lead to severe respiratory failure and be complicated by spontaneous bacterial empyema (13–16 % of cases). The difference (gradient) of albumin concentration in pleural puncture and blood serum allows one to distinguish transudate from exudate, just as with ascites. If the indicator is more than 11 g/L, it is more likely to be a transudate, in particular, hepatic hydrothorax.

Hepatopleural syndrome occurs in 5–15 % of patients with cirrhosis and is associated with a worse prognosis. Symptoms (shortness of breath, cough, hypoxia) usually appear with an effusion of more than 500 mL. Most often, the effusion is right-sided (up to 85 % of cases) but can also be left-sided (13–17 %) or bilateral (8–24 %).

Ascitic fluid enters from the abdominal cavity into the pleural cavities (mainly on the right) through micropores in the diaphragm due to increased intra-abdominal pressure and negative intrathoracic pressure during inspiration. This explains the likelihood of developing hydrothorax in the absence of ascites.

Hydrothorax can be diagnosed by the presence of shortness of breath at rest and with exertion and the following physical findings: asymmetry/lag of one half of the chest during breathing, dullness to percussion, weakening/absence of vocal tremor and bronchophony, absence of respiratory sounds. The diagnostic accuracy of physical methods is approximately 60 % (with large effusions it reaches 88 %) [1].

Treatment of hydrothorax in liver cirrhosis is similar to that for ascites. A diet with salt limitation to 2 grams per day, diuretic therapy (spironolactone 50–400 mg/day in combination with loop diuretics, usually furosemide at a dose of 40–160 mg/ day) is required. If necessary, thoracentesis can be performed [8–12].

Therapeutic thoracentesis (pleural puncture) is necessary to relieve shortness of breath, clarify the nature of the fluid, and carry out a differential diagnosis with tuberculosis and cancer, but its effectiveness in refractory hydrothorax is limited. Pleural effusion is a low protein transudate; infection of the pleural fluid with the development of spontaneous pleural empyema is possible (equivalent to spontaneous bacterial peritonitis). Repeated (and large-volume) thoracentesis is undesirable, as this increases the risk of complications (inexhaustible hydrothorax, pneumothorax, infection of pleural fluid and soft tissues, bleeding) [13].

Other treatments for hepatic hydrothorax include the installation of a transjugular portosystemic shunt (TIPS), which is effective in 70–80 % of cases but is associated with certain complications. Also, in some cases, pleurodesis and surgical interventions are performed to restore the integrity of the diaphragm. Liver transplantation remains a radical treatment option [14–19]. Eradication of the hepatitis C virus reduces the risk of disease progression, decompensation of cirrhosis, hepatocellular cancer, however, the presence of an autoimmune disease (primary sclerosing cholangitis), impaired renal function and port-pulmonary complications adversely affect the patient's prognosis, and therefore in the described clinical case the patient was placed on the waiting list for liver transplantation.

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Submitted: 18.11.2023 Accepted: 02.02.2024 Published: 30.08.2024
Поступила: 18.11.2023 Принята: 02.02.2024 Опубликована: 30.08.2024