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Vanishing Bile Duct Syndrome as a Manifestation of Paraneoplastic Syndrome in Hodgkin's Lymphoma

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Aim: to present a clinical observation of paraneoplastic manifestations of Hodgkin's lymphoma — liver damage in the development of vanishing bile duct syndrome.

Clinical case. Patient Sh., 18-years-old female, was admitted with complaints of yellowing of the skin and sclera, severe general weakness, and fever. Mechanical jaundice, viral hepatitis, Wilson — Konovalov disease, autoimmune hepatitis, and infections were excluded. A hypothesis was put forward about drug-induced hepatitis; some improvement was noted against the background of therapy with ursodeoxycholic acid and prednisolone, but fever and neutrophilic leukocytosis persisted. Physical examination and imaging revealed enlarged lymph nodes in various groups on both sides of the diaphragm, enlarged liver and spleen. PET-CT revealed active accumulation of ^{18}F-fluorodeoxyglucose in the enlarged lymph nodes and bone marrow; lymphoproliferative disease was suspected. During follow-up, laboratory signs of cytolysis and cholestasis persisted, severe jaundice, decreased liver protein-synthetic function, and signs of portal hypertension were noted. A liver biopsy was performed, which revealed ductopenia in most portal tracts, without signs of inflammation (vanishing bile duct syndrome). The presence of splenomegaly, supra- and subdiaphragmatic lymphadenopathy contradicted the diagnosis of "drug-induced cholestasis". A septic process was excluded. According to the examination, including histological examination of the enlarged lymph node, Hodgkin's lymphoma, nodular sclerosis, with damage to the supra- and subdiaphragmatic lymph nodes, liver ("vanishing bile duct syndrome" — paraneoplastic reaction) and spleen, stage IIIB according to the Ann Arbor classification, were diagnosed. After polychemotherapy, the fever resolved, and laboratory parameters showed significant positive dynamics. Control PET-CT did not reveal foci of pathological accumulation of ^{18}F-fluorodeoxyglucose. The patient continues to take ursodeoxycholic acid.

Conclusion. Liver involvement in Hodgkin's lymphoma may manifest as vanishing bile duct syndrome, which is essentially a manifestation of paraneoplastic syndrome. Achieving complete remission and ursodeoxycholic acid therapy are considered to be the key to resolving ductopenia.

Keywords: vanishing bile duct syndrome, Hodgkin's disease, paraneoplastic syndrome, cholestasis

Conflict of interest: the authors declare no conflict of interest.

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Синдром исчезающих желчных протоков как проявление паранеопластического синдрома при болезни Ходжкина

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Цель: представить клиническое наблюдение паранеопластического проявления лимфомы Ходжкина — поражения печени при развитии синдрома исчезающих желчных протоков.

Клиническое наблюдение. Пациентка Ш., 18 лет, поступила с жалобами на желтушное окрашивание кожи и склер, выраженную общую слабость, лихорадку. Исключены механическая желтуха, вирусные гепатиты, болезнь Вильсона — Коновалова, аутоиммунный гепатит, инфекции. Выдвинуто предположение о лекарственном гепатите, на фоне терапии препаратами урсодезоксихолевой кислоты и преднизолоном отмечено некоторое улучшение, однако сохранялись лихорадка и нейтрофильный лейкоцитоз. При физикальном ос-

мотре и по данным методов визуализации обнаружено увеличение лимфатических узлов различных групп по обе стороны диафрагмы, увеличение печени и селезенки. При ПЭТ-КТ обнаружено активное накопление ^{18}F-фтордезоксиглюкозы в увеличенных лимфатических узлах и костном мозге; заподозрено лимфопролиферативное заболевание. При последующем наблюдении сохранялись лабораторные признаки цитолиза и холестаза, выраженная желтуха, отмечены снижение показателей белковосинтетической функции печени и появление признаков портальной гипертензии. Выполнена биопсия печени, при которой выявлена дуктопения в большинстве портальных трактов, без признаков воспаления (синдром исчезающих желчных протоков). Наличие спленомегалии, над- и поддиафрагмальной лимфаденопатии противоречило диагнозу «лекарственный холестаз». Исключен септический процесс. По данным проведенного обследования, включая гистологическое исследование увеличенного лимфатического узла, диагностирована лимфома Ходжкина, нодулярный склероз, с поражением над- и поддиафрагмальных лимфатических узлов, печени («синдром исчезающих желчных протоков» — паранеопластическая реакция) и селезенки, стадия IIIB по классификации Энн-Арбор. После проведения полихимиотерапии разрешилась лихорадка, отмечена выраженная положительная динамика лабораторных показателей. При контрольной ПЭТ-КТ очагов патологического накопления ^{18}F-фтордезоксиглюкозы не выявлено. Пациентка продолжает прием урсодезоксихолевой кислоты.

Заключение. Поражение печени при лимфоме Ходжкина может проявляться синдромом исчезающих желчных протоков, по сути, представляющим собой проявление паранеопластического синдрома. Достижение полной ремиссии и терапия урсодезоксихолевой кислотой рассматриваются как залог разрешения дуктопении.

Ключевые слова: синдром исчезающих желчных протоков, болезнь Ходжкина, паранеопластический синдром, холестаз

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Introduction

The course of Hodgkin's lymphoma can be accompanied by paraneoplastic manifestations known as "B-symptoms". The most common among them are fever, neutrophilia, cerebellar degeneration, and neuropathy [1]. Cases of acute liver failure, chronic cholangitis, and liver cirrhosis have also been described [2]. Of particular interest is liver injury during the development of vanishing bile duct syndrome (VBDS).

Clinical case

Patient Sh., 18-years-old female, was admitted to the Hepatology Department of the V.Kh. Vasilenko Clinic of Propaedeutics of Internal Diseases, Gastroenterology and Hepatology (Sechenovskiy University), with complaints of jaundice of the skin and sclera, fever, and marked general weakness. According to medical history, two years prior she developed pain in the right ankle joint with joint deformity; later, pain in the right knee joint appeared. Tendinitis of the quadriceps femoris muscle was suspected, and Sirdalud (tizanidine) and celecoxib in the maximum daily doses were prescribed. On day 10 of taking these medications, her body temperature rose to 38.5–39 °C, a dull pain in the upper abdomen appeared along with nausea and scleral icterus. The patient was hospitalized for evaluation: elevated serum transaminases and cholestatic enzymes were found, along with pronounced neutrophilic leukocytosis. Viral hepatitis (A, B, C), Wilson disease, autoimmune hepatitis, obstructive jaundice, and infections were excluded. MRI of the abdomen revealed enlarged para-aortic lymph nodes. Computed tomography of the abdomen and chest showed enlargement of lymph nodes in the supraclavicular and subclavicular regions, mediastinum, and retroperitoneal space, as well as moderate hepatomegaly and splenomegaly (Fig. 1). Initially, drug-induced hepatitis was suspected, and therapy was administered with ursodeoxycholic acid (UDCA) preparations, prednisone at 30 mg per day, and plasma exchange sessions. Against this background, some decrease in liver transaminase activity and cholestasis markers was noted and the arthritis regressed; however, neutrophilic leukocytosis and fever persisted. Given the presence of generalized lymphadenopathy, PET-CT was performed, which revealed active uptake of ^{18}F-fluorodeoxyglucose in the enlarged lymph nodes and diffusely increased metabolism in the bone marrow (Fig. 2), leading to suspicion of a lymphoproliferative disease. An oncology consultation concluded that the nature of the liver lesion should be clarified before proceeding with a lymph node biopsy.

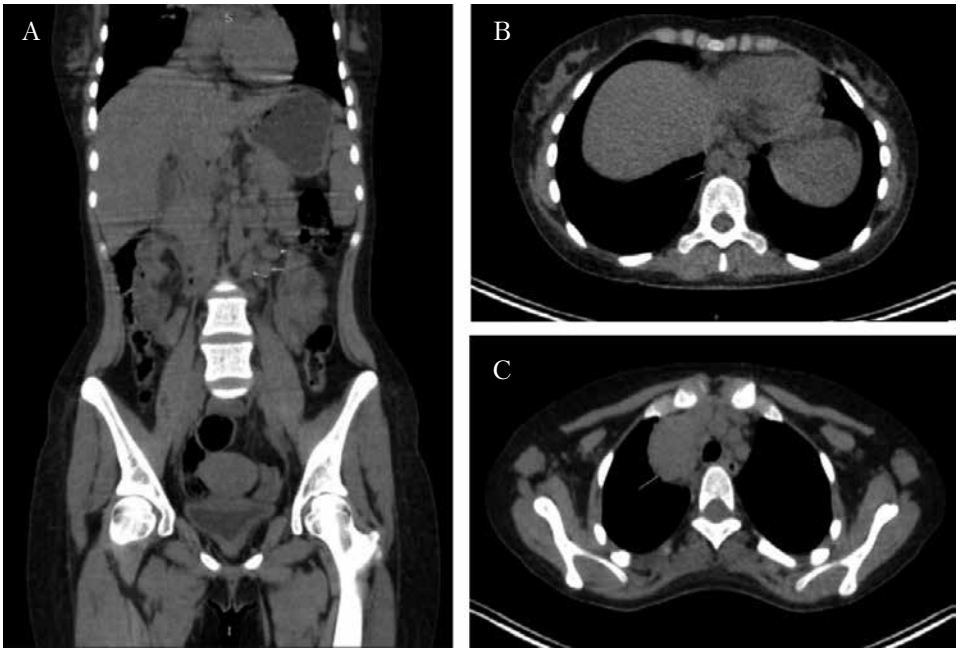

Figure 1. Computed tomography of the abdominal cavity and chest (two months after the onset of fever): A (coronal section) — enlarged mesenteric lymph nodes, length > 1.5 cm, multiple (indicated by arrows); B (horizontal section) — enlarged paraaortic and retrocrural lymph nodes (indicated by an arrow): C (horizontal section) — large paratracheal lymph node (indicated by an arrow)

Рисунок 1. Компьютерная томография органов брюшной полости и грудной клетки (через 2 мес. от манифестации лихорадки): A (корональный срез) — увеличенные мезентериальные лимфатическое узлы, длинник > 1,5 см, множественный характер (указаны стрелками); B (горизонтальный срез) — увеличенные парааортальные и ретрокруральные лимфатическое узлы (указаны стрелкой): C (горизонтальный срез) — крупный паратрахеальный лимфатический узел (указан стрелкой)

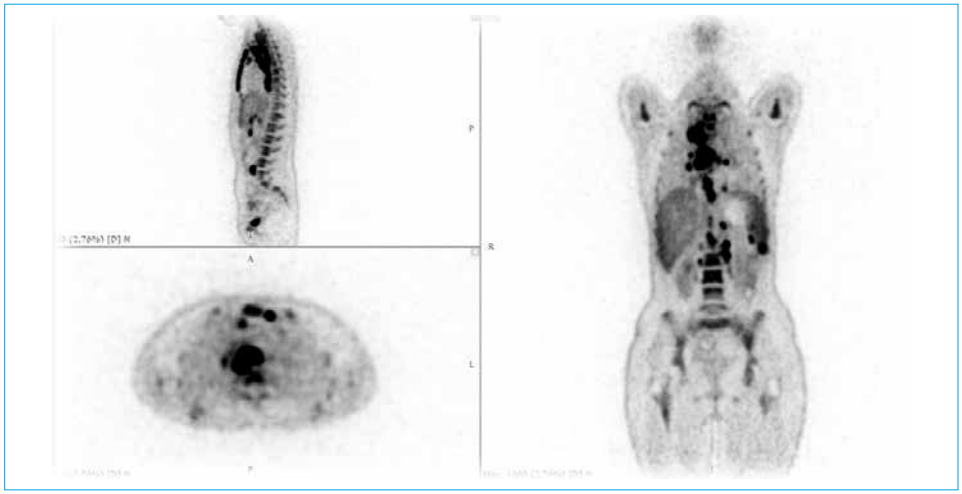

Figure 2. PET-CT data 5 months after the manifestation of fever: widespread lymphadenopathy with active uptake of ^{18}F-fluorodeoxyglucose involving the lower jugular and supraclavicular lymph nodes, paratracheal lymph nodes on the right, intrathoracic, retroperitoneal, retrocrural lymph nodes, iliac lymph nodes on the right; moderate enlargement of the spleen; diffusely increased metabolism in the bone marrow

Рисунок 2. Данные ПЭТ-КТ через 5 мес. от манифестации лихорадки: распространенная лимфаденопатия с активным захватом ^{18}F-фтордезоксиглюкозы с вовлечением нижнеяремных и надключичных лимфатических узлов, паратрахеальных лимфатических узлов справа, внутригрудных, забрюшинных, ретрокруральных лимфатических узлов, подвздошных лимфатических узлов справа; умеренное увеличение селезенки; диффузно повышенный метаболизм в костном мозге

On admission to the Sechenovskiy University clinic, the patient's general condition was relatively satisfactory, she was alert, and she performed the number-connection test in 30 s. Body mass index was 18.0 kg/m^2. The skin, visible mucous membranes, and sclera were icteric. Supraclavicular, axillary, and inguinal lymph nodes up to the size of a hazelnut were palpable, painless, firm, and not matted together. No edema was present. Respiratory rate was 16 per min. Lung fields were normal on percussion; breath sounds were vesicular with no rales. Heart borders were within normal limits; heart sounds were clear and rhythmic, with no murmurs. Heart rate was 84 per min, blood pressure — 115/70 mmHg. The abdomen was of normal shape, soft and non-tender on palpation; the segments of the colon were unremarkable. The liver extended 2 cm below the costal margin, with a rounded, painless, elastic edge. The elastic, painless lower pole of the spleen was palpable; percussion indicated spleen dimensions of 16 × 8 cm. Laboratory findings on admission were as follows: hemoglobin — 97 g/L, leukocytes — 16×10^9/L, neutrophils — 13.5×10^9/L, platelets — 520×10^9/L, erythrocyte sedimentation rate — 96 mm/h. Urinalysis and stool analysis were unremarkable. Blood biochemistry showed signs of hepatocellular injury (alanine aminotransferase — 55 U/L, aspartate aminotransferase — 109 U/L) and cholestasis (elevated alkaline phosphatase up to 703 U/L, gamma-glutamyl transferase — up to 551 U/L, bile acids — up to 203.3 μmol/L, total bilirubin — up to 106.5 μmol/L, direct bilirubin — up to 57.5 μmol/L); total protein — 69 g/L, albumin — 29 g/L, cholesterol — 9 mmol/L. Coagulation studies indicated moderate hypocoagulation (international normalised ratio — 2.25, prothrombin index — 27 %). C-reactive protein was elevated to 114 mg/L. Abdominal ultrasound revealed multiple enlarged para-aortic lymph nodes confluent into conglomerates, as well as solitary enlarged paracaval lymph nodes; spleen size was 180 × 57 mm, portal vein diameter 14 mm, splenic vein diameter up to 8 mm (signs of portal hypertension). Esophagogastroduodenoscopy showed non-erosive reflux esophagitis and superficial gastritis.

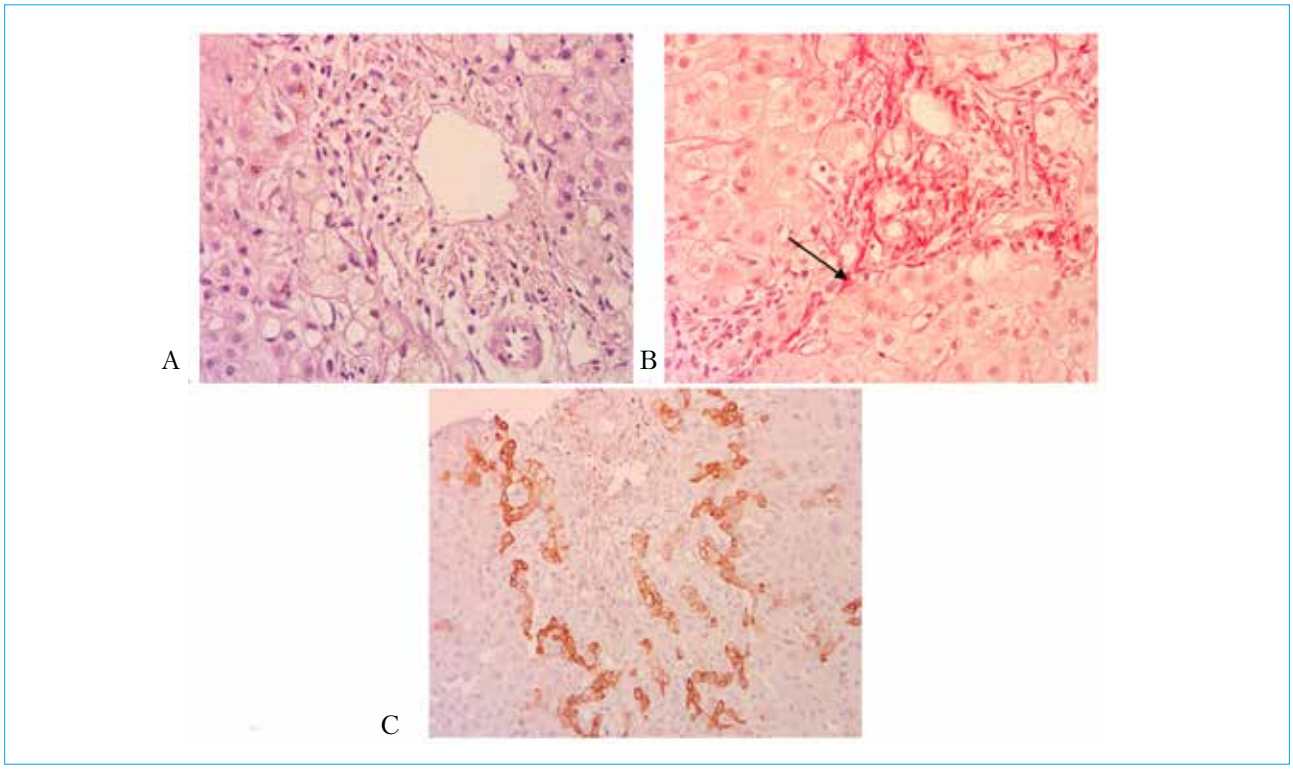

Figure 3. Liver biopsy: A — the interlobular bile duct is absent in the portal tract (hematoxylin and eosin, ×400); B — moderate sclerosis of the portal tract, forming fibrous septum (indicated by the arrow) (picrosirius, ×400); C — CK7+ periportal hepatocytes, formation of small bile ducts periportally (IHC study with antibodies to CK7, ×200)

Рисунок 3. Биопсия печени: А — в портальном тракте междольковый желчный проток отсутствует (гематоксилин и эозин, ×400); В — умеренный склероз портального тракта, формирующаяся фиброзная септа (указана стрелкой) (пикросириус,×400); С — СК7+ перипортальные гепатоциты, формирование мелких желчных протоков перипортально (ИГХ-исследование с антителами к СК7, ×200)

To clarify the cause of cholestasis, a percutaneous liver biopsy was performed (Fig. 3). In biopsy specimens stained with hematoxylin-eosin, picrosirius red, and CK7, the lobular and trabecular architecture of the liver was not preserved throughout. Foci of marked focal parenchymal-canalicular cholestasis were observed in the central and intermediate parts of all lobules, and there was slight expansion of moderately sclerosed portal tracts with forming fibrous septa extending from them. In 7 of 9 portal tracts (78 %), interlobular bile ducts were absent (ductopenia); in two portal tracts the bile ducts were deformed and reduced in size, with no signs of inflammation. Immunohistochemical examination showed membranous CK7 expression in periportal hepatocytes and progenitor cells, which can be considered an initial stage of a compensatory-adaptive process of bile duct formation aimed at resolving biliary hypertension. VBDS was suspected, and further clarification of the nature of the underlying disease was required.

Clinically, the following syndromes were noted in this case: articular syndrome (at disease onset), generalized lymphadenopathy, progressive liver failure, neutrophilic fever, and hepatolienal (hepatosplenic) syndrome. The presence of splenomegaly and supra- and sub-diaphragmatic lymphadenopathy was inconsistent with a diagnosis of drug-induced cholestasis. Given the neutrophilic leukocytosis, the patient's transfers between multiple hospitals, and glucocorticoid therapy, a septic process was deemed unlikely (three sets of blood cultures obtained at the height of fever were negative; echocardiography revealed no vegetations or masses on the heart valves or within the chambers). Lymphoma was considered the most likely diagnosis, and a biopsy of an enlarged supraclavicular lymph node was performed (Fig. 4). The diagnosis was Hodgkin's lymphoma, nodular sclerosis subtype, with involvement of supra- and subdiaphragmatic lymph nodes, liver (with "vanishing bile duct syndrome" as a paraneoplastic reaction), and spleen, stage IIIB by the Ann Arbor classification.

Over the next four months, 4 cycles of polychemotherapy were administered according to the BEGEV regimen (bendamustine, gemcitabine, vinorelbine) in combination with nivolumab. During treatment, the fever resolved, and the jaundice and general weakness significantly diminished. Laboratory tests showed a marked positive trend: levels of bilirubin and markers of hepatocellular injury and cholestasis decreased

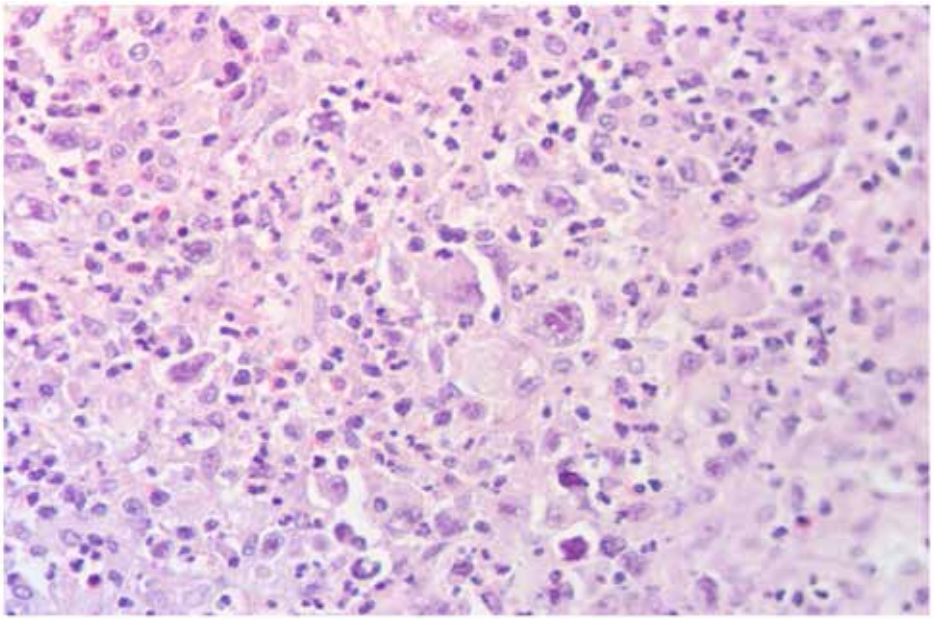

Figure 4. Biopsy of the supraclavicular lymph node: the structure is blurred due to the proliferation of tumor tissue formed by atypical cells of the lymphoid series (Sternberg — Reed, Hodgkin) against the background of diffuse infiltration by lymphocytes, eosinophils, plasma cells with areas of sclerosis (hematoxylin and eosin, ×400)

Рисунок 4. Биопсия надключичного лимфатического узла: рисунок строения стерт из-за разрастания опухолевой ткани, образованной атипичными клетками лимфоидного ряда (Штернберга — Рида, Ходжкина) на фоне диффузной инфильтрации лимфоцитами, эозинофилами, плазмоцитами с участками склероза (гематоксилин и эозин, ×400)

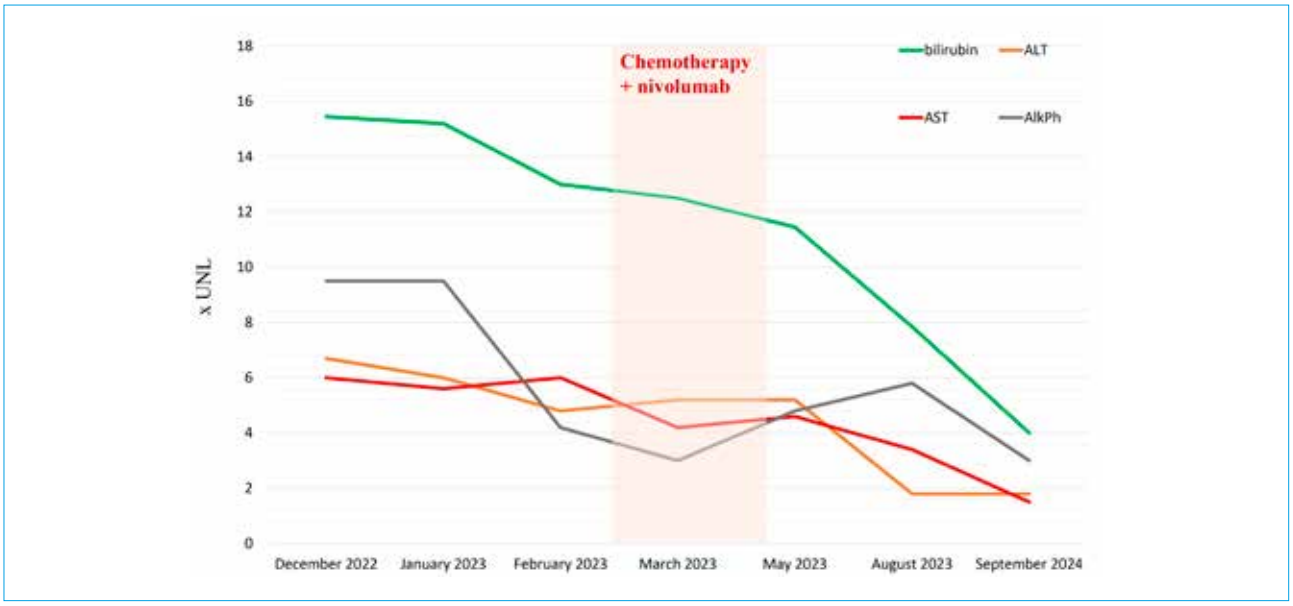

Figure 5. Dynamics of laboratory parameters of cytolysis and cholestasis
Рисунок 5. Динамика лабораторных показателей цитолиза и холестаза

(Fig. 5). Follow-up PET-CT revealed no foci of pathological ^{18}F-FDG uptake; an increase in spleen size was noted (which can be explained by the development of portal hypertension). At present, Hodgkin's lymphoma remains in remission. The patient continues to take UDCA at 15 mg/kg/day; moderate elevation of liver enzymes persists (roughly 1.5–3–fold above normal), as does mixed hyperbilirubinemia (2–3–fold elevation). Ongoing follow-up by a hematologist and a gastroenterologist-hepatologist is indicated.

Discussion

Vanishing bile duct syndrome (VBDS) is characterized by progressive destruction of intrahepatic bile ducts and their disappearance, resulting in cholestasis. This syndrome was first described in 1988 as "idiopathic adulthood ductopenia". Subsequent studies confirmed the autoimmune nature of the process; however, its pathogenesis is not fully understood. VBDS has been reported in patients after liver transplantation, in the context of various medications (ibuprofen, levofloxacin, allopurinol, ampicillin, amoxicillin, erythromycin, doxycycline, co-trimoxazole, etc.), in HIV-infected patients, and in malignancies, including lymphoma [3, 4]. Despite the possibility of VBDS arising in various pathological conditions, the syndrome is rare. VBDS is considered a diagnosis of exclusion; during differential diagnosis, viral, autoimmune, and other diseases that lead to ductopenia due to inflammatory and destructive bile duct injury must be ruled out. To confirm the diagnosis, a liver biopsy is required; the presence of ductopenia is evidenced by the absence of interlobular bile ducts in more than 50 % of portal tracts [5]. Clinically milder forms are referred to as "idiopathic cholestasis". In such cases, signs of pericholangitis or destructive cholangitis are not found in the biopsy. The prognosis of this condition is not well understood and is often unpredictable; according to the literature, it may depend on the etiology of VBDS and the patient's initial overall condition [3].

Liver involvement in Hodgkin's lymphoma can be multifactorial and may be caused by: (a) tumor infiltration of the hepatic parenchyma; (b) chemotherapy; (c) compression of the bile ducts by enlarged lymph nodes at the liver hilum, leading to intrahepatic bile duct ectasia and ascending cholangitis; or a combination of several factors [1]. S.G. Hubscher et al. were the first to report VBDS in Hodgkin's lymphoma, describing three patients with lymphoma who developed intrahepatic cholestasis and ductopenia [6]. In Hodgkin's lymphoma complicated by VBDS, jaundice develops in 100 % of cases; overall, jaundice occurs in 3–13 % of Hodgkin's lymphoma patients. Proposed mechanisms of VBDS in Hodgkin's lymphoma include: (a) a direct cytotoxic effect of tumor cells on the biliary epithelium (when liver metastases are present); (b) damage due to production of tumor cytokines and inflammatory mediators; (c) development of immunopathological reactions, including autoantibody formation [4, 5]. The latter two mechanisms can be considered manifestations of paraneoplastic syndrome. It is

believed that under the influence of damaging factors, accelerated "aging" of cholangiocytes occurs, the rate of apoptosis increases, and apoptosis begins to predominate over regenerative processes [1]. In some cases, VBDS may be an adverse outcome of chemotherapy, as evidenced by the absence of characteristic changes in liver biopsy specimens obtained before treatment. In the development of cholestasis syndrome in patients with Hodgkin's lymphoma in the absence of combined independent diseases with bile duct damage, additional examination is justified, including liver biopsy, in order to exclude vanishing bile duct syndrome [3]. In differential diagnosis, imaging methods — especially PET — play an important role, as they allow exclusion of extrahepatic biliary obstruction and hepatic infiltration by lymphoma cells.

Overall, regarding paraneoplastic "B-symptoms" in Hodgkin's lymphoma, it should be noted that they are very common — recorded in more than 40 % of cases, and in one of every six patients they precede the main manifestations [2]. The more pronounced these symptoms, the greater the probability of their concurrence; in the presented clinical case there was a combination of high fever, arthritis, and paraneoplastic cholestasis. During the diagnostic workup, hepatic tumor infiltration and mechanical cholestasis were ruled out, leading to the diagnosis of VBDS.

When liver involvement is suspected in Hodgkin's lymphoma, due to the risk of progressive liver failure, initiation of therapy should not be delayed while awaiting liver biopsy results. In early-stage disease (I–II), radiotherapy — or otherwise combination chemotherapy — can be started without waiting for a biopsy [1]. In addition to acute liver failure, there have been cases with an outcome of liver cirrhosis [2]. The dose of chemotherapeutic agents, especially doxorubicin, should be adjusted with consideration of the potential risk of hepatotoxicity. In Hodgkin's lymphoma complicated by VBDS, after radiotherapy and chemotherapy, a sustained remission of the underlying disease is observed in most cases. In the present case, secondary damage to hepatocytes due to cholestasis manifested as a cytolytic syndrome and acute liver failure. To protect hepatocytes from the damaging action of bile acids under cholestatic conditions, UDCA therapy is indicated. Achieving complete remission of the lymphoma and administering UDCA are considered the key to resolving ductopenia, although the long-term consequences of this paraneoplastic syndrome remain not well studied [1, 7]. In our case, one year after polychemotherapy, the lymphoma is in remission and there is significant (though incomplete) improvement in liver function test results. UDCA therapy is ongoing, and the patient continues to be monitored by a hematologist and a gastroenterologist-hepatologist.

Conclusion

Liver involvement in Hodgkin's lymphoma can manifest as vanishing bile duct syndrome, which may represent one of the paraneoplastic syndrome manifestations. This syndrome is usually accompanied by other lymphoma "B-symptoms" (in this case — fever, neutrophilia, arthritis). Secondary injury to hepatocytes is reflected by a cytolytic syndrome, and liver failure can develop. Imaging methods, including PET-CT, allow exclusion of biliary obstruction and infiltration of the liver by lymphoma cells. Active polychemotherapy can be initiated without waiting for the results of liver biopsy. This approach, supplemented by UDCA, contributes to remission of the primary disease and regression of paraneoplastic liver injury.

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