ACUTE LIVER FAILURE ON THE BACKGROUND OF CHRONIC LIVER DISEASE DUE TO HEPATITIS VIRUS REACTIVATION

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Abstract

Today, hepatitis B virus-associated acute liver failure remains the leading cause of liver failure (44% mortality in Asia and 41% in the United States). Studies show that among patients with hepatitis B-associated cirrhosis, acute liver failure develops in 10-20% of cases. Acute liver failure on chronic liver disease is a potentially reversible syndrome that occurs in patients with cirrhosis or chronic liver disease and is characterized by acute decompensation, organ failure, and high short-term mortality. Chronic hepatitis B virus infection is a leading cause of liver morbidity and mortality worldwide. When we talk about hepatitis B, there is a high risk of developing super infection hepatitis D, since hepatitis D remains infectious and can reactivate at very low titers that are not detected using modern analysis methods if HBsAg remains in the blood serum. The interaction of these viruses leads to accelerated progression of fibrosis and cirrhosis of the liver, which significantly increases the risk of developing acute liver failure against the background of chronic.

In our case, a patient diagnosed with liver cirrhosis as a result of viral hepatitis B with delta agent, class C according to Child–Pugh–Turcotte, MELD-36 points, the patient developed a severe form of acute renal failure, which required emergency intervention, so he was not included in the waiting list for a transplant from a cadaveric donor. His wife became the donor, which is an example of living donation, which provides higher chances of successful recovery due to a shorter waiting time and a lower risk of graft rejection. Timely examination of the donor and recipient, as well as prompt liver transplantation, contributed to a favorable outcome of the disease.

Introduction

In recent years, particular attention has been paid to the problem of viral reactivation, where an increase in viral activity in patients with chronic hepatitis may lead to disease exacerbation and liver injury.[1] Chronic hepatitis D (CHD) is a severe liver disease caused by the hepatitis D virus (HDV), which is prevalent globally.[2] The interaction between these viruses leads to accelerated progression of fibrosis and liver cirrhosis, significantly increasing the risk of acute-on-chronic liver failure (ACLF) development.[2] Relapses of hepatitis D after therapy are common and substantially reduce treatment efficacy.[3]

The prevalence and outcomes of ACLF vary depending on geographic region and disease etiology. According to a systematic review and meta-analysis, the global prevalence of ACLF among patients with decompensated cirrhosis is 35%.[4] In Asian countries, where HBV is highly endemic, viral activation frequently leads to the development of ACLF. Mortality among ACLF patients can reach up to 44%.[5] In the United States, patients with HBV-related decompensated cirrhosis who develop ACLF have a

30-day mortality rate of 41%, compared to 7% among patients without ACLF.[5] In Europe, where alcohol-related etiology predominates, the incidence of ACLF among cirrhotic patients ranges from 20% to 35%, with ACLF-related mortality between 30% and 50%.[6]

Classification of ACLF According to the EASL-CLIF Consensus

The classification of acute-on-chronic liver failure according to the EASL-CLIF (European Association for the Study of the Liver-Chronic Liver Failure) consensus is one of the most frequently cited classifications, proposed by the European Association for the Study of the Liver (EASL) and the CLIF research group. It classifies ACLF based on clinical criteria and the degree of damage to various organs (e.g., liver, kidneys, heart).[7] There are three types of ACLF related to organ failure, derived from the CLIF-SOFA score, which have been associated with high 28-day mortality rates: A (mild), B (moderate), and C (severe).

Type A: Liver failure with minimal dysfunction in other organs.[7] In a study conducted in Europe, the 28-day mortality rate among patients with Stage 1 ACLF was 23.3%, and the 90-day mortality rate was 55.2%.[8]

Type B: Manifest liver failure with progressive dysfunction of the organs. The 28-day mortality rate is 31.3%, and the 90-day mortality rate is 55.2%.

Type C: Severe liver failure with rapid progression of multi-organ failure requiring intensive treatment. The 28-day mortality rate is 74.5%, and the 90-day mortality rate is 78.4%.[7]

Thus, depending on the region and severity of the disease, the 28-day mortality rate in ACLF can range from 18% to 25%, and the 90-day mortality rate can range from 30% to 40%.[9]

The aim of this paper is to discuss a clinical case of a patient who underwent emergency liver transplantation due to acute liver failure on the background of chronic liver disease from aliving related donor.

Case presentation

A 35-year-old male patient was diagnosed with: Cirrhosis of the liver as a result of chronic hepatitis B with the delta agent, Class C by the Child-Pugh Score (CPS). MELD score: 36. Acute hepatic cell failure on the background of chronic liver failure (ACLF). Portal hypertension syndrome. Esophageal varices of grade 3. Ascites of grade 2 according to the International Ascites Club (IAC).

According to the patient, he considered himself ill starting from July 2023, when he noticed a moderate increase in abdominal size. He was examined at a private medical center, where a Fibroscan revealed liver fibrosis at stage F4. Subsequently, the patient underwent further examination at the A.N. Syzganov National Scientific Center of Surgery (NSC). PCR for hepatitis D on October 4, 2023, was positive, while PCR for hepatitis B was negative. Due to worsening of his condition, on January 8, 2024, the patient was hospitalized at the NSC for further examination and preparation for liver transplantation from a living donor.

Upon examination, ascites (biochemical analysis shown below in Table 1), esophageal varices grade 3 (E varix F3, Lm, CB, RCS (+)), and mild portal hypertensive gastropathy were found. Ultrasound on January 9, 2024, showed splenomegaly and bilateral hydrothorax (370 ml). CT from March 10, 2023 (1 a, b, c) revealed liver cirrhosis, splenomegaly, splenorenal and splenomesenteric shunts, recanalization of the umbilical vein, esophageal and gastric varices, and ascites. Based on these findings, the following diagnosis was made: Cirrhosis of the liver due to chronic hepatitis B with the delta agent, Class C by CPS. MELD score: 36. Acute hepatic cell failure on the background of chronic liver failure (ACLF). Portal hypertension syndrome. Esophageal varices grade 3. Ascites grade 2 according to IAC.

The patient was diagnosed with hepatitis D reactivation on the background of liver cirrhosis resulting from chronic hepatitis B with the delta agent, which led to decompensation of liver function and acute metabolic disorders. Due to a high total bilirubin level in the blood, plasma exchange sessions were initiated. However, due to the severity of

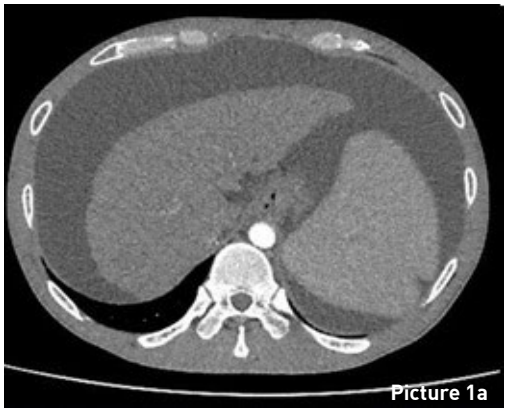
the patient's condition and progression of liver failure, it was recommended to proceed with liver transplantation from a living related donor. On January 18, 2024, an emergency liver transplantation surgery was performed from a living donor, with the donor being the patient's wife (K, 27 years old).

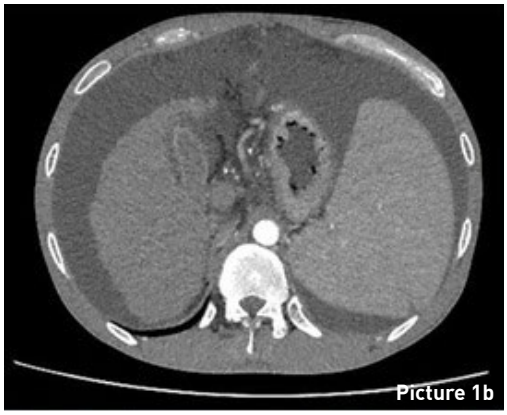
CT findings from October 9, 2023, showed: Liver volume–1345 cm³ (6,140 cm³). Volume of the left lobe of the liver +segment I–446.9 cm³ (33.2%).

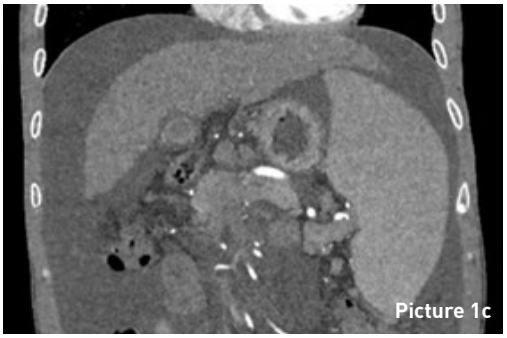
Table1.
Biochemical Indicators of the Patient Before Liver Transplantation

	January, 2024							
	08/01	**10/01**	**11/01**	**12/01**	**14/01**	**15/01**	**16/01**	**17/01**
ALT	60.40	46,60	41.70	33.70	32.70	35.30	46,0	51.80
AST	117.60	92,60	82.0	68.20	63.30	69.40	89.20	103.80
Total bilirubin	727.0	789	797	760	737.70	746.4	757.4	871.0
PTI	24.10	29.10	32.70	30.30	23.20	27.20	24.00	26.00
INR	2.83	2.39	2.16	2.30	2.94	2.53	2.70	2.63

Picture 1 (a, b, c).
CT from March 10, 2023: Liver cirrhosis, splenomegaly, portal hypertension, splenomesenteric and splenorenal shunts, recanalization of the umbilical vein, esophageal and gastric varices, ascites.

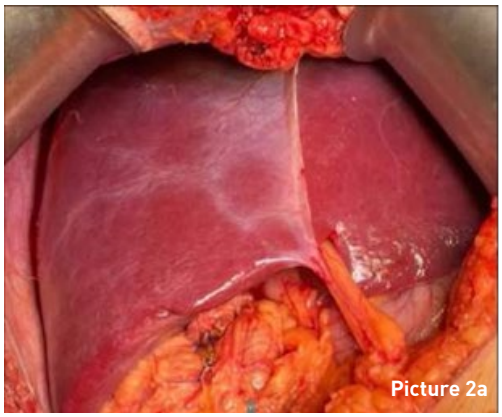

Picture 1a

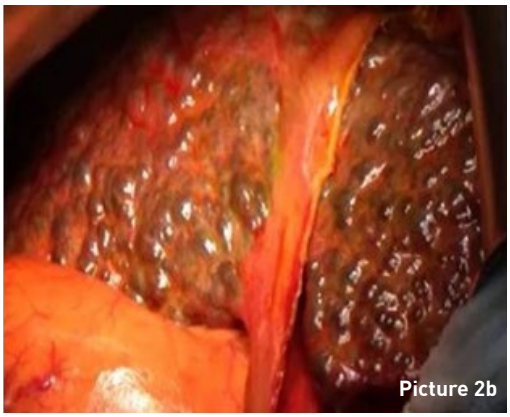

Picture 1b

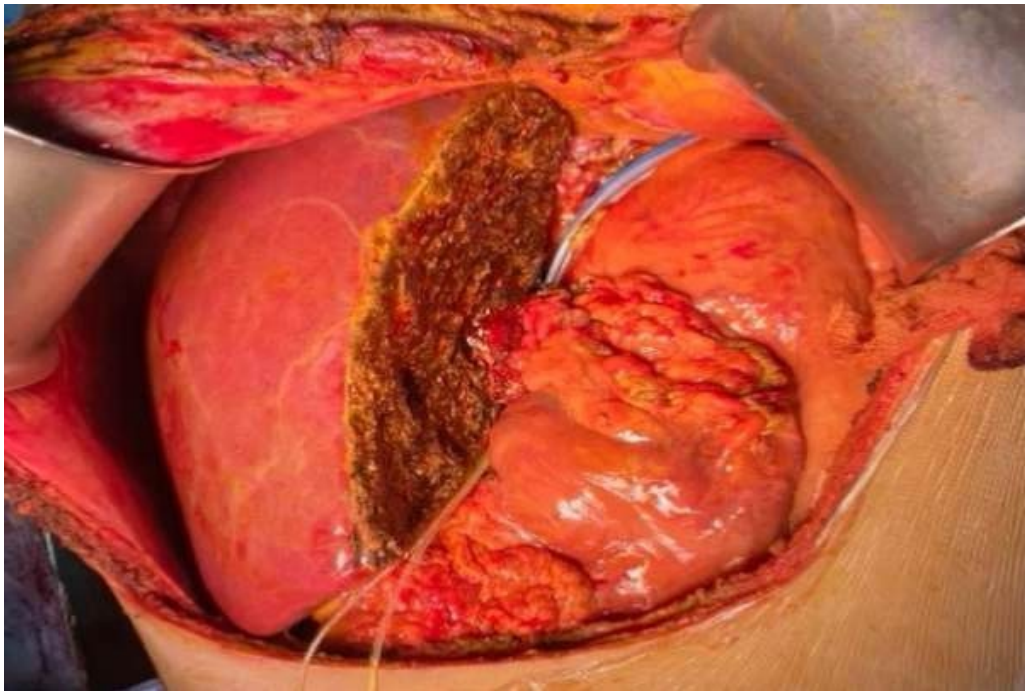

Picture 1c

Picture 2a.
ntraoperative Period Donor Liver

Picture 2b.
Cirrhotic Liver of the Recipient


Picture 2a


Picture 2b



Picture 2 c.
Final Appearance of the Transplanted Liver

	January 2024 year							
	19/01	**20/01**	**22/01**	**25/01**	**29/01**	**03/02**	**07/02**	**16/02**
ALT	93.60	92.50	58.30	66.80	52.40	52.20	68.20	31.50
AST	152.20	77.70	26.60	39.00	15.20	33.30	31.30	36.7
Total bilirubin	478.40	187.60	221.60	301.10	183.10	114.0	107.20	19.90
PTI	50.60	44.30	48.30	82.00	79.60	84.90	86.30	105.50
INR	1.49	1.66	1.55	1.11	1.13	1.09	1.08	0,97

Table 1.
Postoperative Period: A Decrease in Biochemical Parameters Was Observed

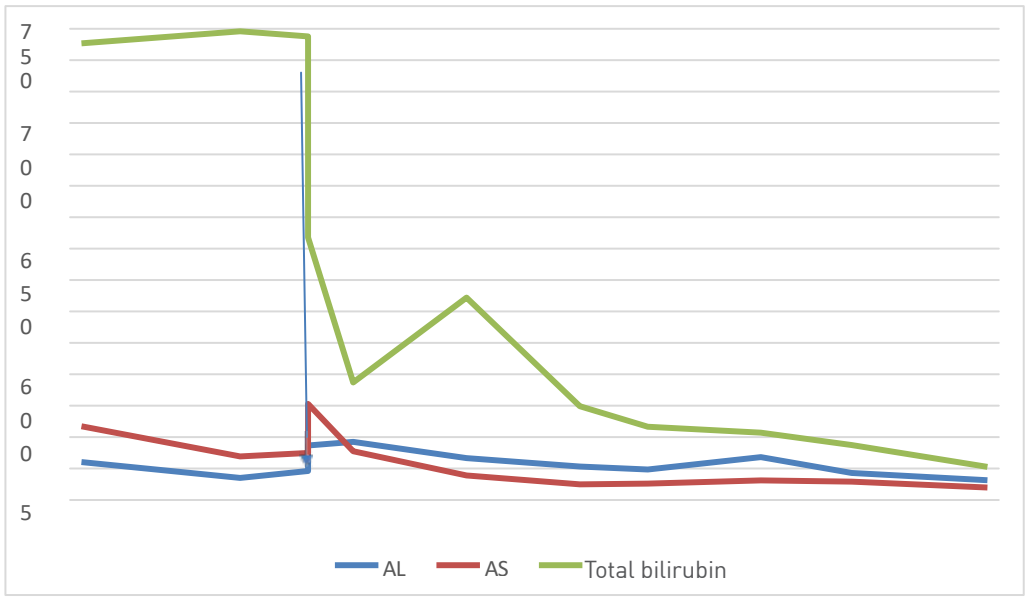
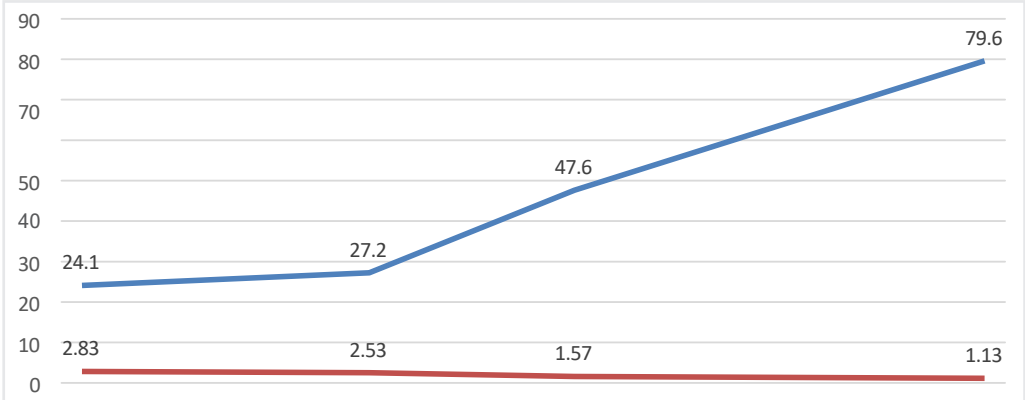


Figure 1.
Dynamic Changes in Biochemical Parameters

Figure 2.
Coagulation Profile in Dynamics



	08/01/2024	15/01/2024	20/01/2024	30/01/2024
PTI	24.1	27.2	47.6	79.6
INR	2.83	2.53	1.57	1.13

Discussion

Orthotopic liver transplantation (OLT) remains the only curative intervention capable of significantly improving outcomes in acute-on-chronic liver failure (ACLF), particularly at advanced stages where supportive therapy fails to ensure survival.[10] In the CANONIC study, 4.9% and 15% of ACLF patients underwent transplantation within 28 and 90 days of admission, respectively, with post-transplant survival for grades 2–3 reaching ~80% versus ~20% with conservative management.[11] Subsequent studies confirm one-year survival above 70%.[8]

This case is notable for both etiology and its rapid progression. Chronic HBV with HDV superinfection is known to accelerate fibrosis, cause earlier cirrhosis, and increase acute decompensation risk.[2] HDV reactivation—even at low, undetectable levels—poses diagnostic challenges and can delay treatment. In our patient, swift deterioration led to multiorgan failure, including severe acute kidney injury—a poor prognostic factor in ACLF.

Given the patient's critical state and ineligibility for deceased donor listing, living donor liver transplantation (LDLT) was performed. LDLT offers distinct advantages in emergencies: immediate graft availability, minimal cold ischemia, and in some reports, superior short- and medium-term graft survival.[9] In this case, using the patient's spouse as donor reduced immunologic risk and expedited surgery.

Postoperative labs (Table 2) showed steady improvement in liver and synthetic function, consistent with literature indicating optimal outcomes when transplantation occurs before irreversible extrahepatic organ failure.[12]

This case emphasizes the need for vigilant HDV monitoring in HBV-related cirrhosis, especially in endemic areas, and illustrates LDLT's role as a viable alternative in organ shortage settings. While limited by its single-case design, the scenario demonstrates that early ACLF recognition, rapid donor–recipient assessment, and timely transplantation can yield favorable short-term results even in severe HBV/HDV-associated disease.

Limitations. This is a single case report, which limits the generalizability of the results. In addition, long-term follow-up data are not yet available, as the patient received a liver transplant only 1 year ago. The level of maintenance of graft function to date has been assessed as satisfactory. In the future, monitoring of organ function indicators will be carried out on an ongoing basis.

What's Known? OLT remains the only curative treatment for ACLF. HBV/HDV coinfection is associated with accelerated progression of fibrosis, earlier cirrhosis, and increased risk of acute decompensation. Multiorgan failure, particularly acute kidney injury, is recognized as a poor prognostic factor in ACLF.

What's New? This report highlights

the successful use of LDLT as a life-saving option in HBV/HDV-related ACLF with multiorgan failure. It emphasizes that timely LDLT, even in patients with severe extrahepatic complications, can result in meaningful short-term recovery when deceased donor grafts are unavailable.

Conclusion

The presented clinical case demonstrates the critical importance of liver transplantation as the only radical method for treating patients with severe liver failure. Emergency transplantation significantly reduces waiting time and lowers the risk of organ rejection, which greatly improves the patient's prognosis. This case highlights the importance of early intervention and the use of living donor liver transplantation as a key aspect in the treatment of severe forms of liver failure.

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