International Journal of Medical Sciences And Clinical Research

VOLUME Vol.06 Issue04 2026
PAGE NO. 98-101
DOI 10.37547/ijmscr/Volume06Issue04-19

Clinical Course of Acute Myeloid Leukemia in A Patient with Chronic Hepatitis C

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Received: 09 March 2026; **Accepted:** 30 March 2026; **Published:** 25 April 2026

Abstract: This article presents a clinical case of a patient with relapsed acute myeloid leukemia (AML) and chronic HCV infection who developed severe infectious complications, including sepsis and multiple organ failure, as well as marked hepatic syndrome with signs of hemolysis during intensive chemotherapy and transfusion therapy. The dynamics of liver biochemical parameters, the clinical course of HCV infection, and the outcomes of direct-acting antiviral therapy administered during a critical condition are described.

Keywords: Acute myeloid leukemia (AML), hepatitis C virus (HCV), HCV reactivation, direct-acting antivirals (DAAs), immunosuppression, sepsis, multiple organ failure, antiviral therapy, hematologic malignancies, liver dysfunction.

Introduction: Hepatitis C virus (HCV) infection remains one of the leading causes of chronic liver disease and an important factor affecting prognosis in patients with hematologic malignancies. According to the World Health Organization, chronic HCV infection affects approximately 71 million people worldwide and is associated with high morbidity and mortality [1].

The prevalence of HCV infection among patients with hematologic malignancies ranges from 5% to 15%, which is substantially higher than in the general population [2,3]. In patients with acute myeloid leukemia (AML), the presence of HCV infection may adversely affect the disease course, limit the feasibility of intensive polychemotherapy, and increase the risk of hepatotoxic complications [4]. Immunosuppression caused both by the underlying disease and by chemotherapy creates conditions for HCV reactivation, accompanied by enhanced viral replication and the development of cytolytic syndrome [5,6].

In recent years, the introduction of direct-acting antivirals (DAAs) has significantly changed the management of HCV infection. These agents demonstrate high efficacy, with sustained virologic response achieved in more than 95% of cases, and have a favorable safety profile, including in immunocompromised patients [7,8].

Several studies have shown that DAA therapy in patients with hematologic malignancies is highly effective and well tolerated even during chemotherapy. Achieving sustained virologic response helps reduce the incidence of hepatotoxic complications and makes it possible to continue antitumor treatment in full [9,10]. However, most available data concern clinically stable patients, whereas experience with DAA therapy in critically ill individuals, including those with sepsis and multiple organ failure, remains limited.

In addition, the optimal timing for initiating antiviral therapy in patients with severe hematologic malignancies and life-threatening infectious complications remains controversial. In clinical practice, treatment is often delayed because of concerns regarding drug interactions, potential hepatotoxicity, and deterioration of the patient's general condition [11]. Therefore, clinical observations demonstrating the feasibility and safety of antiviral

therapy for HCV infection in severe and critical conditions are of particular importance.

Objective

The aim of this study was to demonstrate that severe infectious complications are not an absolute contraindication to antiviral therapy for HCV, particularly when treatment is well tolerated, and to assess the impact of timely viral control on liver function stabilization and on expanding the possibilities for antitumor treatment.

Case Presentation

A 29-year-old patient was diagnosed with acute myeloid leukemia (AML) on October 25, 2024, based on clinical and hematologic findings and bone marrow examination, which revealed 40.8% blasts. Immunophenotyping identified a population of myeloid blast cells consistent with minimally differentiated AML.

The patient received induction polychemotherapy according to the "7+3" regimen (cytarabine plus daunorubicin), resulting in partial remission with 7% blasts. Subsequently, additional therapy courses and high-dose cytarabine (HiDAC) were administered, leading to clinical and hematologic remission with fewer than 3% blasts. However, in November 2025, disease relapse was documented with 31% blasts, and salvage antirelapse therapy was initiated.

In March 2025, the patient was diagnosed with chronic hepatitis C virus infection. A 12-week course of direct-acting antiviral therapy with sofosbuvir/velpatasvir was prescribed. Sustained virologic response was achieved: HCV PCR was negative after 12 weeks and remained negative at the 24-week follow-up assessment.

Since March 2025, the patient had not received any blood component transfusions, which makes reinfection unlikely and supports the interpretation of subsequent viral detection as HCV reactivation. In January 2026, due to clinical deterioration, the patient was hospitalized. Laboratory evaluation revealed elevated transaminase levels (ALT 291 U/L, AST 127 U/L) and a high HCV viral load of 7.64 x 10^6 IU/mL, which was interpreted as reactivation of chronic HCV infection. A repeated 12-week course of antiviral therapy with sofosbuvir 400 mg plus daclatasvir 60 mg was therefore initiated.

A rapid virologic response was achieved after 4 weeks of treatment, with negative PCR results; however, after discharge, the patient discontinued therapy on his own and did not complete the full course. Beginning on February 25, 2026, due to refractory AML, the patient underwent intensive polychemotherapy according to the FLAG-Ida protocol. This was followed by severe post-chemotherapy cytopenia, with leukocytes decreasing to 0.01 x 10^9/L and neutrophils to 0 x 10^9/L, which on March 6, 2026, was complicated by febrile neutropenia, sepsis, and septic shock.

Repeated examination in March 2026 showed that HCV PCR had become positive again. Given the premature discontinuation of antiviral therapy, this finding indicates viral persistence and reactivation of infection.

Table 1. Dynamics of Biochemical Parameters

Date (2026)	ALT (U/L)	AST (U/L)	Bilirubin (umol/L)	Clinical Event
15.01	291	127	8	Before initiation of antiviral therapy
10.03	97	28	175	Sepsis
12.03	29	21	530	Hemolysis, peak hyperbilirubinemia
19.03	19	13	312	Clinical stabilization

Despite the extremely severe condition of the patient, including septic shock, multiple organ failure, and agranulocytosis, antiviral therapy was tolerated satisfactorily and was not associated with any significant worsening of liver function. A downward trend in transaminase levels was observed, whereas hyperbilirubinemia appeared to be multifactorial, likely related to sepsis, hemolysis, and massive transfusions.

Interruption of the antiviral therapy course, despite the achieved early virologic response, likely contributed to HCV reactivation. Under conditions of profound immunosuppression, this may have additionally aggravated the severity of the patient's condition and the course of the infectious complications.

DISCUSSION

The management of patients with hematologic malignancies and concomitant chronic HCV infection represents a complex clinical challenge, especially in the setting of intensive polychemotherapy and severe immunosuppression. With the introduction of direct-acting antivirals, the possibilities for HCV eradication have expanded significantly, including in patients with severe comorbid conditions.

According to the literature, DAA therapy is highly effective and well tolerated in patients with hematologic malignancies, allowing sustained virologic response to be achieved in more than 95% of cases. The presence of a hematologic malignancy itself is not considered a contraindication to HCV treatment, particularly when interferon-free regimens are used. However, these patients remain at risk for HCV reactivation, especially in the context of pronounced immunosuppression caused by both the underlying disease and cytotoxic therapy. HCV reactivation may manifest either as reappearance of viral RNA in the blood after sustained virologic response has been achieved or as a significant increase in viral load in patients with previously controlled infection.

In the present clinical case, after sustained virologic response had been achieved during therapy with sofosbuvir/velpatasvir at 12 and 24 weeks, HCV reactivation was later documented in the setting of AML relapse and immunosuppression, accompanied by high viral load and elevated transaminases. An important contributing factor was likely the premature discontinuation of the repeated antiviral therapy course with sofosbuvir plus daclatasvir despite the achievement of a rapid virologic response.

It is well known that interruption of DAA therapy may result in incomplete viral eradication and viral persistence, which, under conditions of immunosuppression, creates a basis for reactivation. In this case, the absence of blood component transfusions during the interval between treatment courses makes reinfection unlikely and supports the interpretation of a true reactivation process.

It should also be emphasized that HCV reactivation in immunocompromised patients may contribute to deterioration of liver function and may indirectly influence the course of the underlying disease and infectious complications. Although no pronounced cytolytic syndrome was observed in this case, active viral replication could have aggravated the patient's overall condition in the context of sepsis and multiple organ failure.

At the same time, this clinical case demonstrates that even in an extremely severe condition, including septic shock and agranulocytosis, antiviral therapy with direct-acting agents is feasible and is not associated with significant worsening of liver function. This is consistent with current understanding of the safety of DAA therapy and its priority role in the treatment of HCV infection in complex patients.

Thus, a key issue is not only timely initiation of antiviral therapy but also ensuring its complete course. Premature discontinuation of treatment may negate the achieved effect and lead to infection reactivation, especially in high-risk groups such as patients with hematologic malignancies.

CONCLUSIONS

1. Direct-acting antiviral therapy in patients with acute myeloid leukemia is effective and may result in sustained virologic response even in the setting of immunosuppression.

2. Premature discontinuation of antiviral therapy, despite the achievement of a rapid virologic response, is associated with the risk of viral persistence and subsequent HCV reactivation.

3. In patients with hematologic malignancies, HCV reactivation is likely associated with severe immunosuppression and may worsen the course of the underlying disease and infectious complications.

4. Antiviral therapy can be administered even in severe clinical conditions, including sepsis and multiple organ failure, without significant deterioration of liver function.

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