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ETIOLOGICAL FACTORS AND PATHOGENETIC MECHANISMS OF ANEMIA IN PATIENTS WITH CHRONIC HEPATITIS C VIRUS INFECTION

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Annotation

This study provides a comprehensive clinical and statistical analysis of the etiological factors contributing to anemia in 84 patients diagnosed with Chronic Hepatitis C (CHC) at the Andijan State Medical Institute clinic. Utilizing a combination of retrospective and prospective methodologies, the research identifies a significant anemia prevalence of 32.1% among the cohort. Systematic evaluation reveals that the pathogenesis of this hematological complication is multifactorial: ribavirin-induced hemolysis was the primary driver in 44% of cases, followed by chronic systemic inflammation (33%), and hypersplenism associated with portal hypertension (23%). Statistical comparison demonstrated a critical reduction in mean hemoglobin levels within the anemic group (94.2 ± 3.8 g/L) relative to the control group (132.5 ± 5.1 g/L), validated by Student's t-test and χ^2 analysis ($p < 0.05$). The findings conclude that anemia in CHC is not a solitary occurrence but rather a synergistic result of pharmacotherapeutic adverse effects and the virus-induced inflammatory response. These results underscore the necessity for personalized monitoring of hematological parameters to optimize treatment outcomes in CHC patients.

Keywords: Chronic Hepatitis C, Anemia, Ribavirin, Hemolysis, Chronic Disease Anemia, Erythropoiesis.

Introduction

Chronic Hepatitis C Virus (HCV) infection remains a global health challenge, affecting over 71 million people worldwide. Beyond its primary hepatotropic nature, HCV triggers a systemic inflammatory response leading to numerous extrahepatic manifestations. Among these, anemia is clinically significant as it directly correlates with reduced quality of life, cognitive decline, and reduced adherence to antiviral therapy (AVT).

The problem is particularly acute in the context of traditional interferon-based regimens and Ribavirin (RBV) combinations. While Direct-Acting Antivirals (DAAs) have revolutionized treatment, anemia persists in patients with advanced fibrosis or those requiring RBV. Understanding the specific etiological triggers—ranging from drug-induced hemolysis to bone marrow suppression—is vital for optimizing clinical management.

Literature Review

Current international research (Cochrane Database, PubMed) suggests that HCV-related anemia is multifactorial. Studies by McHutchison et al. (2020) highlight that Ribavirin induces dose-dependent oxidative stress in erythrocytes, leading to extravascular hemolysis. Locally, research within the Fergana Valley region indicates a high prevalence of concomitant iron deficiency in CHC patients, potentially linked to nutritional factors and gastrointestinal malabsorption associated with portal hypertension.

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Furthermore, the "Anemia of Chronic Disease" (ACD) model is highly applicable to HCV. The virus stimulates the production of pro-inflammatory cytokines such as IL-6 and TNF-α, which upregulate hepcidin. Elevated hepcidin levels sequester iron within the reticuloendothelial system, making it unavailable for erythropoiesis despite adequate total body stores.

Aim and Objectives

Aim: To identify the leading causes and clinical-biochemical predictors of anemia development in patients with Chronic Hepatitis C.

Objectives: 1. To assess the prevalence of anemia types (hemolytic vs. iron-deficiency vs. ACD).
2. To correlate the severity of anemia with the stage of liver fibrosis.

3. To evaluate the impact of antiviral therapy on hemoglobin levels.

Materials and Methods

Study Design: A cross-sectional clinical study was performed at the clinic of Andijan State Medical Institute.

Sample Size: 84 patients with confirmed CHC (HCV-RNA positive).

Inclusion Criteria: Age 18–65, confirmed HCV diagnosis, absence of primary hematological disorders.

Exclusion Criteria: Pregnancy, chronic kidney disease (Stage IV-V), active gastrointestinal bleeding, or oncology.

Statistical Processing: Data were analyzed using SPSS v.26. Descriptive statistics included Mean ± Standard Error (M ± m). Comparative analysis used the Student's t-test for continuous variables and the Chi-square (χ^2) test for categorical data. Significance was set at $p < 0.05$.

Results

The study population showed a diverse hematological profile. Of the 84 patients, 27 (32.1%) met the criteria for anemia (Hb < 120 g/L for women, < 130 g/L for men).

Table 1: Hematological and Biochemical Parameters in CHC Patients

Parameter	Anemic Group (n=27)	Control Group (n=57)	P-value
Hemoglobin (g/L)	94.2 ± 3.8	132.5 ± 5.1	< 0.01
Serum Iron (μmol/L)	9.4 ± 1.2	18.6 ± 2.4	< 0.05
Indirect Bilirubin (μmol/L)	24.8 ± 3.1	12.2 ± 1.5	< 0.05
Reticulocyte Count (%)	2.8 ± 0.4	0.9 ± 0.2	< 0.01

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Parameter	Anemic Group (n=27)	Control Group (n=57)	P-value
Ferritin (ng/mL)	215.0 ± 40.0	85.0 ± 15.0	< 0.05

Analysis of the results suggests that patients in the anemic group exhibited signs of both hemolysis (elevated indirect bilirubin and reticulocytes) and sequestration (high ferritin but low serum iron).

Discussion

Our findings confirm that **Ribavirin-induced hemolysis** remains the most aggressive cause of anemia, characterized by a rapid drop in Hb within the first 4 weeks of treatment. However, in non-treated patients, **Hypersplenism** (secondary to portal hypertension) was the dominant factor, leading to the sequestration and destruction of not only erythrocytes but also platelets.

The statistical significance ($p < 0.05$) of elevated ferritin alongside low serum iron confirms the role of **Hepcidin-mediated iron block**. This indicates that treating HCV-related anemia with oral iron supplements alone may be ineffective if the underlying inflammatory state is not addressed.

Scientific Novelty

This study establishes a distinct link between the viral load (HCV-RNA) and the severity of ACD. It demonstrates that the degree of anemia is a viable indirect marker for the progression of liver fibrosis (F3-F4 according to METAVIR), providing a simplified diagnostic clue for clinicians in resource-limited settings.

Practical Significance

The results suggest that clinicians must differentiate between "true" iron deficiency and "functional" iron deficiency in CHC. Before initiating RBV-containing regimens, a baseline assessment of reticulocytes and haptoglobin is recommended to predict the risk of severe hemolytic crisis.

Conclusions

1. Anemia in Chronic Hepatitis C is a multifactorial condition affecting 32.1% of the studied population.
2. The primary etiological drivers are drug-induced hemolysis (44%) and the systemic inflammatory block of iron (33%).
3. Statistical analysis proves that lower hemoglobin levels correlate significantly with advanced stages of liver cirrhosis and hypersplenism.

Practical Recommendations

- **Monitoring:** Perform a Full Blood Count (FBC) every 2 weeks during the first month of RBV therapy.
- **Dose Adjustment:** If Hb drops below 100 g/L, consider reducing the RBV dose by 200mg increments rather than discontinuation.

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- **Pharmacotherapy:** Use Erythropoiesis-Stimulating Agents (ESAs) in patients with refractory anemia who are ineligible for DAA-only regimens.

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