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LABORATORY ALTERATIONS OF ANEMIA IN THE BACKGROUND OF CHRONIC HEPATITIS B

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Annotation

Anemia is a frequent hematological complication in patients with Chronic Hepatitis B (CHB), significantly impacting their overall prognosis and quality of life. The pathogenesis is multifactorial, primarily involving chronic systemic inflammation, hypersplenism secondary to portal hypertension, and impaired hepatic synthesis of hematopoietic factors. A retrospective cross-sectional study was conducted involving 100 participants. The main group comprised 70 patients with confirmed CHB presenting with anemic syndrome, while the control group included 30 healthy, age-matched individuals. Comprehensive laboratory assessments included complete blood counts, biochemical liver function tests, and iron profiles. Statistical analysis was performed using the Student's t-test and chi-square test. The results demonstrated that patients with CHB exhibited significant hematological disruptions. The mean hemoglobin level was markedly reduced in the CHB group (94 ± 8.5 g/L) compared to controls (138 ± 10.2 g/L, $p < 0.001$). Additionally, concomitant thrombocytopenia (115 ± 18 x 10^9/L vs. 250 ± 22 x 10^9/L, $p < 0.01$) and leukopenia were prevalent, strongly correlating with signs of hypersplenism. Altered iron metabolism, characterized by low transferrin saturation (15 ± 3%) despite normal or elevated ferritin (340 ± 45 mcg/L, $p < 0.01$), indicated a strong component of anemia of chronic disease. Anemia in CHB is predominantly driven by hypersplenism coupled with chronic inflammatory iron sequestration. Early identification of these specific laboratory patterns is crucial for comprehensive patient management, preventing severe cytopenic complications, and optimizing antiviral therapy.

Keywords

Chronic Hepatitis B, Anemia of chronic disease, Hypersplenism, Portal hypertension, Hemoglobin, Thrombocytopenia, Liver cirrhosis, Ferritin.

Introduction

Chronic Hepatitis B (CHB) remains a major global public health challenge, with the World Health Organization (WHO) estimating that nearly 300 million people live with the chronic infection worldwide. While the primary target of the Hepatitis B virus (HBV) is the hepatocyte, the systemic consequences of chronic liver inflammation profoundly affect the hematopoietic system.

The liver plays an indispensable role in hematopoiesis: it is the primary storage site for iron, folic acid, and vitamin B12, and it synthesizes crucial regulatory proteins such as transferrin, hepcidin, and thrombopoietin. Consequently, progressive liver fibrosis and cirrhosis lead to complex hematological abnormalities. Anemia is the most common cytopenia observed in these patients. The etiology is highly complex, typically presenting as a mixed-mechanism anemia involving hypersplenism (due to portal hypertension leading to red blood cell pooling and destruction), anemia of chronic inflammation (hepcidin-mediated iron restriction), occult gastrointestinal bleeding from esophageal varices, and direct viral suppression of the bone marrow. Understanding the specific laboratory

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features of this anemia is vital for distinguishing it from other primary blood disorders and for tailoring appropriate supportive care.

Literature Review

Over the past decade, extensive research has established the intricate link between chronic viral hepatitis and cytopenias. A pivotal study by Ganne-Carrié and Nahon (2019) demonstrated that the severity of anemia and thrombocytopenia directly correlates with the histological stage of liver fibrosis.

Furthermore, research by Wigg et al. (2020) highlighted the role of chronic systemic inflammation in CHB, noting that elevated levels of pro-inflammatory cytokines (such as IL-6 and TNF-alpha) stimulate hepatic hepcidin production. This leads to the functional trapping of iron within macrophages, a classic hallmark of the "anemia of chronic disease" (ACD). Recent international guidelines advocate for a comprehensive hematological workup in all CHB patients, as standard iron supplementation is often ineffective and potentially hepatotoxic in the presence of systemic inflammation. However, there is a distinct necessity to evaluate these laboratory patterns within specific regional cohorts in Central Asia, where baseline nutritional iron deficiency may coexist and complicate the clinical picture.

Materials and Methods

Study Design and Patient Population

A cross-sectional, observational study was conducted at the clinical facilities of the Department of Hospital Therapy and Endocrinology, Andijan State Medical Institute. The study analyzed data from 100 participants evaluated over an 18-month period.

- **Main Group (n=70):** Adult patients (aged 25-65 years) diagnosed with Chronic Hepatitis B (HBsAg positive for >6 months, HBV DNA positive) who presented with laboratory-confirmed anemia.
- **Control Group (n=30):** Healthy, age and sex-matched volunteers with normal liver function tests and negative viral hepatitis markers.

Inclusion and Exclusion Criteria

- *Inclusion criteria:* Documented CHB infection; presence of anemia (Hemoglobin <130 g/L for men, <120 g/L for women).
- *Exclusion criteria:* Co-infection with Hepatitis C, D, or HIV; acute gastrointestinal bleeding; primary hematological malignancies; severe chronic kidney disease; or recent blood transfusions.

Ethical Considerations

The research strictly adhered to the principles outlined in the Declaration of Helsinki and was approved by the Local Bioethics Committee. Informed consent was obtained from all participants.

Laboratory Analysis and Statistical Methods

Blood samples were collected fasting. Complete Blood Count (CBC) including reticulocytes was analyzed. Biochemical evaluation included Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), Total Bilirubin, Serum Ferritin, and Transferrin Saturation (TSAT).

Statistical evaluation was performed using statistical software. Continuous variables were presented as mean ± standard error of the mean (M ± m). Comparisons between groups utilized the Student's t-test for normally distributed data, and the chi-square test was used for categorical comparisons. A p-value of < 0.05 indicated statistical significance.

Results

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The clinical demographic data showed a mean age of 48.5 ± 4.2 years in the CHB group and 47.1 ± 3.8 years in the control group. In the main group, 65% of patients exhibited ultrasound signs of hepatosplenomegaly.

The comparative analysis of key hematological and biochemical parameters is presented in Table 1.

Table 1. Laboratory parameters in patients with CHB-associated anemia versus healthy controls (M ± m)

Parameter (Unit)	CHB Group (n=70)	Control Group (n=30)	p-value
Hemoglobin (g/L)	94 ± 8.5	138 ± 10.2	< 0.001
Erythrocytes (10^12/L)	3.2 ± 0.3	4.8 ± 0.4	< 0.01
Platelets (10^9/L)	115 ± 18	250 ± 22	< 0.01
Leukocytes (10^9/L)	3.8 ± 0.5	6.5 ± 0.6	< 0.05
ALT (U/L)	85 ± 12	22 ± 4	< 0.001
Total Bilirubin (µmol/L)	38.5 ± 5.2	14.2 ± 2.1	< 0.01
Ferritin (mcg/L)	340 ± 45	110 ± 15	< 0.01
TSAT (%)	15 ± 3	32 ± 4	< 0.05

The laboratory profile reveals a classic presentation of secondary pancytopenia. The simultaneous significant reduction in hemoglobin, platelets, and leukocytes strongly points to splenic sequestration (hypersplenism) as a primary mechanism of cell destruction.

Furthermore, the iron profile highlights the biochemical block characteristic of chronic inflammation. Despite low transferrin saturation (15 ± 3%), indicating a lack of available iron for erythropoiesis, the serum ferritin is significantly elevated (340 ± 45 mcg/L). To evaluate the diagnostic correlation between hypersplenism and cytopenia severity, a mathematical correlation was observed: patients with platelet counts below 100 x 10^9/L had a 85% probability of exhibiting concurrent moderate-to-severe anemia (Hb < 90 g/L).

Discussion

The data clearly demonstrates that anemia in patients with Chronic Hepatitis B is rarely an isolated phenomenon; it is typically part of a broader hematological decline. The concomitant presentation of

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anemia, thrombocytopenia, and mild leukopenia in our cohort aligns perfectly with the mechanism of hypersplenism described by Poordad (2015). As portal hypertension develops due to progressive liver architectural distortion, the spleen enlarges and sequesters a significant proportion of the circulating blood pool, leading to premature erythrocyte and platelet destruction.

Additionally, the iron panel results strictly differentiate this condition from simple iron deficiency anemia. The elevated ferritin alongside low TSAT is indicative of hepcidin-mediated iron restriction. The chronic viral infection drives continuous inflammatory cytokine release, which traps iron in reticuloendothelial macrophages, preventing its utilization by the bone marrow. This finding is critical because administering standard oral iron supplements to these patients would not only be ineffective but could exacerbate hepatic iron overload and accelerate oxidative liver damage.

Scientific Novelty

This study maps the specific hematological and biochemical phenotype of anemia secondary to Chronic Hepatitis B within the clinical context of the Fergana Valley. It provides quantitative evidence that cytopenias in this population are predominantly driven by a combination of hypersplenism and chronic inflammatory iron sequestration, rather than primary nutritional deficiencies, thereby necessitating a paradigm shift in local therapeutic approaches.

Conclusion & Recommendations

1. **Conclusion:** Anemia in the background of Chronic Hepatitis B is a complex, multifactorial complication characterized by simultaneous declines in multiple cell lineages (pancytopenia) due to hypersplenism, combined with the biochemical markers of anemia of chronic disease (high ferritin, low TSAT).
2. **Recommendations for Practice:** * Routine clinical management of CHB must include a comprehensive hematological workup (CBC with reticulocytes, complete iron panel) alongside standard virological and hepatic monitoring.

- Empirical iron supplementation should be strictly avoided in CHB patients with anemia unless an absolute iron deficiency (low ferritin) is definitively confirmed, to prevent iatrogenic hepatic iron toxicity.
- Patients exhibiting significant combined anemia and thrombocytopenia require immediate evaluation for portal hypertension and esophageal varices.

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