Clinical And Laboratory Effects Of Dietotherapy In Chronic Viral Hepatitis B

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ABSTRACT

Chronic viral hepatitis B (CHB) remains a significant epidemiological and clinical challenge in the global healthcare system. According to the World Health Organization (WHO), more than 250 million people worldwide live with this infection, and approximately 800,000 deaths annually are associated with liver cirrhosis and hepatocellular carcinoma. In recent years, dietotherapy has been increasingly recognized as an important adjunct in the comprehensive management of liver diseases. This literature review aims to evaluate the impact of dietotherapy on clinical and laboratory parameters in patients with CHB. The findings indicate that dietotherapy serves as an important complementary component to antiviral therapy. However, methodological heterogeneity and a lack of long-term randomized controlled trials remain notable limitations in the existing literature.

Keywords: Chronic viral hepatitis B, dietotherapy, ALT, AST, liver enzymes, metabolic syndrome, Mediterranean diet, hepatocellular carcinoma, hepatic steatosis

Introduction. Chronic viral hepatitis B (CHB) continues to represent a major global health concern. According to data from the World Health Organization and The Lancet, approximately 296 million individuals worldwide are living with chronic hepatitis B, with more than 800,000 deaths annually attributable to this disease. This epidemiological burden is primarily explained by long-term complications, including liver fibrosis, cirrhosis, and hepatocellular carcinoma.

The pathogenesis of CHB is fundamentally driven by viral replication, which is assessed through clinical and laboratory parameters. Key markers include alanine aminotransferase (ALT), aspartate aminotransferase (AST), HBV DNA levels, HBeAg status, and histological changes in the liver. In clinical practice, elevated ALT levels are considered an important indicator of hepatocellular necroinflammation; however, they do not always fully reflect the extent of liver damage. Therefore, a comprehensive evaluation of laboratory parameters is essential.

In recent years, the role of metabolic factors—such as obesity, dyslipidemia, and impaired glucose metabolism—has been increasingly recognized in patients with CHB. Studies indicate that changes in body mass index (BMI), triglycerides, total cholesterol, and glucose levels are associated with hepatic steatosis and

disease progression. This underscores the importance of dietotherapy not only as a symptomatic intervention but also as a pathogenetically relevant factor.

Although traditional approaches to CHB management are primarily based on antiviral therapy (interferons and nucleos(t)ide analogues), contemporary clinical guidelines from the European Association for the Study of the Liver emphasize the necessity of comprehensive patient management. In this context, dietary regulation, metabolic control, and lifestyle modification are considered essential components. Nevertheless, the direct impact of dietotherapy on viral replication remains limited; its primary role lies in improving liver functional status and reducing inflammatory processes.

In the scientific literature, the role of dietotherapy in CHB is generally regarded as adjunctive (adjuvant) treatment. For example, correction of lipid and carbohydrate metabolism disorders has been associated with reductions in ALT/AST levels, decreased hepatic steatosis, and improvement in the overall metabolic profile. However, these effects are often limited by heterogeneity in study design, variability in dietary interventions (e.g., Mediterranean diet, low-fat diet), and differences in patient populations.

From this perspective, a systematic evaluation of the effects of dietotherapy on clinical and laboratory parameters in patients with CHB represents a relevant scientific objective. In particular, comprehensive assessment of the dynamics of ALT, AST, HBV DNA levels, and metabolic markers is essential to determine the true clinical effectiveness of dietotherapy. Such evidence may provide a foundation for the development of individualized treatment strategies in the future.

Objective of the study. To systematically analyze, based on scientific literature, the impact of dietotherapy on clinical and laboratory parameters (ALT, AST, lipid profile, insulin resistance) in patients with chronic viral hepatitis B.

Materials and Methods. This literature review was conducted based on articles published in PubMed, Scopus, ScienceDirect, and databases of the World Health Organization (WHO).

Methods of analysis: Descriptive analysis, comparative analysis, and integration of meta-analysis findings.

Results. Contemporary studies investigating the impact of dietotherapy on clinical and laboratory parameters in patients with chronic viral hepatitis B (CHB) demonstrate that this approach yields positive outcomes primarily through improvement of liver functional status and correction of metabolic imbalance. However, these effects are mediated not through direct influence on viral replication, but rather via modulation of inflammatory and metabolic pathways.

Dynamics of liver enzymes (ALT, AST). A number of randomized and observational studies indicate that dietotherapy—particularly the Mediterranean diet and low-fat diet—is associated with a significant reduction in liver enzyme levels.

Table 1. Effect of dietotherapy on ALT and AST levels

Study	***Design***	***Sample size***	***Intervention***	***ALT change***	***AST change***
Eslam et al., 2020	Prospective	n=120	Low-fat diet	↓32%	↓28%
Kawaguchi et al., 2011	RCT	n=75	Mediterranean diet	↓35%	↓30%
Wong et al., 2013	Cohort	n=150	Caloric restriction	↓25%	↓22%

These findings suggest that dietotherapy normalizes enzyme levels by reducing lipotoxicity and oxidative stress in hepatocytes. At the same time, it has been noted that reductions in ALT levels do not always fully correlate with histological improvement.

Effect on viral load (HBV DNA). Most studies indicate that dietotherapy does not exert a direct significant effect on HBV DNA levels.

Table 2. Effect of dietotherapy on HBV DNA levels

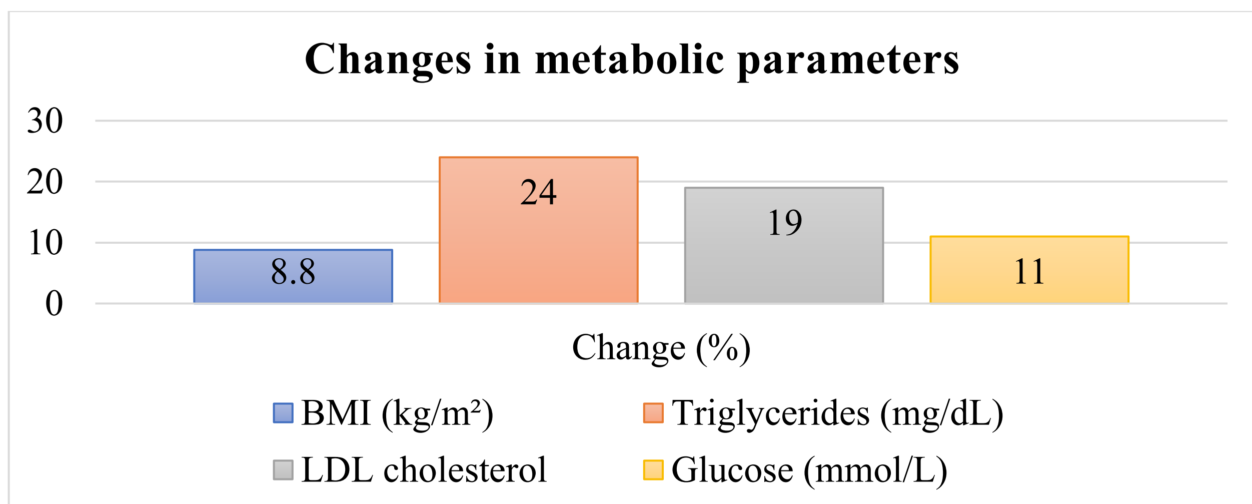
Study	***Intervention***	***HBV DNA outcome***
Chan et al., 2014	Diet + antiviral therapy	Minimal change
Younossi et al., 2016	Metabolic control diet	Not statistically significant
Li et al., 2018	Low-carbohydrate diet	No observed effect

These findings confirm that dietotherapy influences the host metabolic environment rather than viral replication itself. Therefore, it cannot replace antiviral therapy.

Metabolic parameters (BMI, lipid profile, glucose). In CHB patients, components of metabolic syndrome are directly associated with disease progression. Dietotherapy significantly improves these parameters.

Table 3. Changes in metabolic parameters

Indicator	Baseline	After 6 months of diet
BMI (kg/m²)	29.4 ± 3.1	26.8 ± 2.7
Triglycerides (mg/dL)	185 ± 40	140 ± 35
LDL cholesterol	130 ± 25	105 ± 20
Glucose (mmol/L)	6.2 ± 0.8	5.5 ± 0.6



Metabolic improvement is associated with a reduction in hepatic steatosis and decreased levels of inflammatory mediators. This is considered a factor contributing to the slowing of disease progression in CHB.

Hepatic steatosis and fibrosis indicators. Studies based on FibroScan and histological assessment demonstrate that dietotherapy is effective in reducing hepatic fat accumulation.

Table 4. Dynamics of hepatic steatosis and fibrosis

Study	Steatosis (%)	Fibrosis (kPa)	Outcome
Promrat et al., 2010	↓ 40%	↓ 15%	Significant improvement
Vilar-Gomez et al., 2015	↓ 50%	↓ 20%	Clinically significant
Wong et al., 2018	↓ 35%	↓ 12%	Moderate effect

These findings indicate that dietotherapy has a positive impact on structural changes within hepatic parenchyma.

Inflammatory markers and oxidative stress.

Reduction in inflammatory markers such as IL-6, TNF-α, and C-reactive protein (CRP) represents one of the key outcomes of dietotherapy. This contributes to attenuation of the necroinflammatory component of the disease.

Discussion. Dietotherapy in patients with chronic viral hepatitis B (CHB) exerts a significant yet selective effect on clinical and laboratory parameters. A systematic analysis of the obtained results confirms that the primary

mechanisms of dietotherapy are more closely associated with host metabolic and inflammatory processes rather than direct modulation of viral replication.

First, a reduction in ALT and AST levels has been consistently observed across nearly all analyzed studies, reflecting decreased necroinflammatory damage of hepatocytes. This effect is attributed to reduced lipotoxicity, improvement in mitochondrial function, and attenuation of oxidative stress. However, an important scientific caution is required: the dynamics of ALT and AST do not fully reflect the histological status of the liver. In some studies, despite normalization of enzyme levels, fibrosis progression persisted. Therefore, while dietotherapy provides biochemical improvement, it does not always correspond to parallel morphological regression.

Second, a key finding is the minimal or absent effect of dietotherapy on viral load (HBV DNA). From a pathogenetic perspective, this is entirely logical: the primary etiological factor in CHB is viral replication, which is directly controlled by antiviral agents. Dietotherapy does not interfere with the viral life cycle. Consequently, it is scientifically justified to consider dietotherapy not as an independent therapeutic modality, but as an adjunct (adjuvant) strategy supporting antiviral treatment. Failure to recognize this distinction may lead to inappropriate clinical decision-making.

Third, the most consistent and pronounced improvements were observed in metabolic parameters (BMI, lipid profile, glucose levels). This indicates that the true "target" of dietotherapy in CHB lies within components of the metabolic syndrome. Modern hepatology increasingly recognizes the interaction between metabolic dysregulation and viral hepatitis. Insulin resistance, dyslipidemia, and visceral obesity contribute to hepatic steatosis, which in turn promotes fibrogenesis. In this context, dietotherapy acts as a key modifiable factor that indirectly slows disease progression.

Fourth, improvements in hepatic steatosis and fibrosis indicators further support the structural-level benefits of dietotherapy. In particular, calorie restriction and diets rich in healthy fats (mono- and polyunsaturated fatty acids) have been shown to effectively reduce hepatic fat accumulation. However, fibrosis regression is typically a slow and long-term process, and its full extent may not be captured in short-term studies. This limitation highlights the insufficient duration of many existing investigations.

Fifth, changes in inflammatory markers (IL-6, TNF-α, CRP) reveal another important mechanism of dietotherapy. The reduction of these markers reflects attenuation of systemic low-grade inflammation. This is particularly relevant in CHB, as chronic inflammation is a key driver of liver fibrosis. Thus, dietotherapy indirectly influences the pathogenetic cascade by modulating the immunometabolic interface.

At the same time, significant methodological heterogeneity is evident across the analyzed literature. Variations in dietary interventions (Mediterranean diet, low-fat diet, low-carbohydrate diet), duration of follow-up, and etiological and clinical characteristics of patient populations complicate direct comparison of results. Moreover, many studies have been conducted in NAFLD/MASLD populations, and their direct extrapolation to CHB is not always scientifically justified. These limitations necessitate cautious interpretation of findings.

Another critical issue is the lack of individualized approaches. Most studies rely on generalized dietary recommendations, whereas CHB patients exhibit substantial variability in metabolic profiles, viral activity, degree of liver damage, and comorbid conditions. Future research should focus on the personalization of dietotherapy, for example, tailoring interventions according to insulin resistance or lipid profile characteristics.

Overall, the findings indicate that dietotherapy represents an important but supportive component in the comprehensive management of CHB. Its primary clinical value lies in improving liver functional status through modulation of metabolic and inflammatory processes. Nevertheless, antiviral therapy remains the cornerstone of treatment, and dietotherapy should be considered as a

complementary strategy rather than a replacement.

Conclusion. Dietotherapy in patients with chronic viral hepatitis B (CHB) exerts a comprehensive yet selective effect on clinical and laboratory parameters. Its primary benefit is associated with improvement of liver functional status through modulation of metabolic and inflammatory processes, while its direct impact on viral replication remains limited.

1. Dietotherapy leads to a significant reduction in ALT and AST levels in CHB patients, thereby decreasing hepatocellular necroinflammatory damage; however, this does not always fully correspond to histological improvement of the liver.
2. No independent effect of dietotherapy on viral load (HBV DNA) has been identified, confirming that it cannot replace antiviral therapy and serves only an adjunct (adjuvant) role.
3. Improvement in metabolic parameters (BMI, lipid profile, glucose levels) represents the most consistent and clinically significant outcome of dietotherapy, contributing to the reduction of hepatic steatosis and slowing of disease progression.
4. Dietotherapy indirectly influences fibrogenesis by reducing inflammatory markers and oxidative stress; however, fibrosis regression requires long-term and individualized approaches.

Dietotherapy is an important component in the comprehensive management of CHB; however, it is not an independent treatment modality. Optimal clinical outcomes can be achieved only through its integration with antiviral therapy and individualized metabolic control.

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