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# THE SIGNIFICANCE OF METABOLIC SYNDROME AND NUTRITION IN PATIENTS WITH CHRONIC HEPATITIS

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**ABSTRACT**

Chronic hepatitis represents a major global public health challenge, and its course and progression are significantly influenced by metabolic syndrome and nutritional factors. According to the World Health Organization (WHO), more than 300 million people worldwide live with chronic hepatitis B infection, and over 50 million with chronic hepatitis C infection. At the same time, the global prevalence of metabolic syndrome is estimated at approximately 25–30%, reaching up to 40% in certain populations. Scientific evidence indicates that key components of metabolic syndrome—insulin resistance, abdominal obesity, dyslipidemia, and arterial hypertension—accelerate liver fibrosis progression and increase the risk of steatosis and cirrhosis in patients with chronic hepatitis. Dietary patterns, particularly high-calorie regimens rich in saturated fats, further exacerbate hepatocellular injury.

**KEYWORDS**

Chronic hepatitis, metabolic syndrome, insulin resistance, hepatic steatosis, fibrosis, nutrition, diet therapy

## Introduction

Chronic hepatitis is defined as a persistent inflammatory process in the liver tissue lasting more than six months and developing under the influence of infectious, metabolic, autoimmune, or toxic factors. Currently, this pathology is no longer regarded solely as an infectious disease but rather as a complex disorder closely associated with systemic metabolic disturbances. The liver functions as the central metabolic laboratory of the organism, regulating carbohydrate, lipid, and protein metabolism, detoxification processes, hormone metabolism, and multiple aspects of immune response. Consequently, metabolic disorders exert a direct and substantial impact on hepatic structure and function.

According to WHO data, approximately 296 million individuals worldwide are living with chronic hepatitis B virus infection, and more than 58 million with chronic hepatitis C virus infection. Each year, over 1 million deaths are attributed to complications of viral hepatitis, including cirrhosis and hepatocellular carcinoma. These figures place chronic hepatitis among the highest global health priorities.

In recent decades, a high prevalence of metabolic syndrome has been identified among patients with chronic hepatitis. Metabolic syndrome is characterized by abdominal obesity, insulin resistance, arterial hypertension, and dyslipidemia. Its global prevalence is estimated at 25–30%, reaching 35–

40% in certain developed countries. Clinical observations indicate that insulin resistance occurs in 30–70% of patients with chronic hepatitis C. This condition promotes hepatic lipogenesis, leading to steatosis and accelerating fibrotic processes.

Analyses published in The Lancet demonstrate that the coexistence of metabolic syndrome in patients with viral hepatitis increases the risk of cirrhosis development by 1.5–2-fold. Furthermore, data reported in the Journal of Hepatology indicate that overweight patients experience more rapid progression of hepatic fibrosis stages and reduced responsiveness to antiviral therapy.

Insulin resistance, considered the central component of metabolic syndrome, disrupts glucose and lipid metabolism in the liver. Impaired insulin signaling results in triglyceride accumulation within hepatocytes, contributing to steatosis, oxidative stress, and increased production of inflammatory mediators. Consequently, fibrogenesis is stimulated. Studies show that steatosis is observed in 60–70% of patients with chronic hepatitis C genotype 3, representing a clear example of the interaction between viral and metabolic factors.

Nutritional factors play a decisive role in this process. Diets high in calories, refined carbohydrates, and saturated fats enhance hepatic lipid accumulation. Consumption of fructose-rich beverages stimulates de novo lipogenesis and aggravates insulin resistance. In contrast, adherence to a Mediterranean dietary pattern has been shown in multicenter studies to reduce inflammatory markers, improve insulin sensitivity, and decrease hepatic steatosis. Clinical investigations report that a 7–10% reduction in body weight may decrease hepatic steatosis by 40–60%.

Thus, from a contemporary perspective, chronic hepatitis should not be considered solely a viral or autoimmune disorder but rather a multifactorial pathology closely intertwined with metabolic background conditions. Metabolic syndrome and inappropriate dietary habits accelerate hepatic inflammation and fibrosis progression, worsen therapeutic outcomes, and negatively affect long-term prognosis. Therefore, in-depth analysis of these interrelationships and the development of integrative preventive and therapeutic strategies represent key priorities in modern hepatology.

**Research objective**

To analyze, based on scientific literature, the impact of metabolic syndrome components and nutritional characteristics on liver status in patients with chronic hepatitis.

**Materials and methods**

This article was prepared as a systematic literature review. Sources included reports from the World Health Organization, as well as publications in The Lancet, Journal of Hepatology, and Hepatology.

Clinical studies, meta-analyses, and cohort investigations published between 2010 and 2025 were analyzed.

Search keywords included: chronic hepatitis, metabolic syndrome, insulin resistance, liver fibrosis, nutrition, and steatosis.

**Results**

The findings confirm that components of metabolic syndrome act as independent risk factors influencing disease progression, fibrosis stage, and clinical outcomes in patients with chronic hepatitis. The analysis was conducted based on materials published between 2010 and 2025 in Journal of Hepatology, Hepatology, The Lancet, and reports from the World Health Organization.

Prevalence of Metabolic Syndrome in Chronic Hepatitis. The global prevalence of metabolic syndrome in the general population is estimated at approximately 25–30%. Among patients with chronic hepatitis C, this figure increases to 35–45%. Several cohort studies report insulin resistance in 30–70% of cases. According to a meta-analysis published in the Journal of Hepatology, the presence of metabolic syndrome is associated with a 1.8-fold higher risk of fibrosis progression (RR 1.8; 95% CI 1.4–2.3).

Table 1. Prevalence of Metabolic Syndrome Components in Chronic Hepatitis

| Indicator | Prevalence in CH Patients (%) |
|---|---|
| Insulin resistance | 30–70 |
| Abdominal obesity | 40–60 |
| Dyslipidemia | 35–55 |
| Arterial hypertension | 30–50 |

These data indicate that the presence of a metabolic background amplifies viral inflammation and creates a favorable environment for hepatic steatosis development.

Insulin Resistance and Fibrosis Progression. Insulin resistance promotes triglyceride accumulation in hepatocytes and enhances oxidative stress. Data published in Hepatology demonstrate that patients with elevated HOMA-IR levels have a twofold higher prevalence of advanced fibrosis (stage III–IV). In chronic hepatitis C genotype 3 infection, steatosis prevalence reaches 60–70%, confirming the synergistic interaction between viral and metabolic factors.

Table 2. Association Between Metabolic Factors and Liver Complications

| Metabolic Factor | Fibrosis Risk | Cirrhosis Risk |
|---|---|---|
| Insulin resistance | ×1.8–2.0 | ×1.5 |
| BMI >30 kg/m² | ×1.7 | ×1.6 |
| Dyslipidemia | ×1.4 | ×1.3 |

**Nutrition and Clinical Outcomes.**

High-calorie diets rich in saturated fats and refined carbohydrates exacerbate hepatic steatosis. According to data reported in The Lancet, overweight individuals have a 1.6-fold increased risk of cirrhosis development.

Clinical trials evaluating adherence to a Mediterranean dietary pattern demonstrated the following outcomes:

A 7–10% reduction in body weight led to a 40–60% decrease in hepatic steatosis;

ALT and AST levels decreased by 20–30%;

Insulin sensitivity improved significantly.

Table 3. Effectiveness of Diet Therapy (Based on Clinical Studies)

| Indicator | Before Treatment | After 6 Months |
|---|---|---|
| ALT (U/L) | 85 ± 20 | 60 ± 18 |
| AST (U/L) | 72 ± 15 | 50 ± 14 |
| HOMA-IR | 4.2 ± 1.1 | 2.9 ± 0.9 |
| Steatosis level | 100% | 55–60% |

Metabolic Syndrome and Risk of Hepatocellular Carcinoma.

According to the World Health Organization, hepatocellular carcinoma represents a major complication developing on the background of liver cirrhosis. Multicenter studies confirm that the presence of metabolic syndrome increases the risk of hepatocellular carcinoma by 1.5–2-fold.

These results emphasize the critical role of metabolic and nutritional factors in shaping the clinical trajectory and long-term prognosis of chronic hepatitis.

**Discussion**

The results of the literature analysis indicate that evaluating chronic hepatitis solely within the framework of viral or autoimmune etiology is insufficient. From the perspective of modern hepatology, this pathology should be considered a complex disorder closely associated with an underlying metabolic background. The findings demonstrate that components of metabolic syndrome act as independent risk factors in the progression of chronic hepatitis and significantly accelerate hepatic fibrosis.

Insulin resistance emerges as a central pathogenetic mechanism. Impaired insulin signaling enhances hepatic lipogenesis, increases triglyceride accumulation within hepatocytes, and activates oxidative stress pathways. Consequently, nuclear transcription factors stimulate the production of pro-inflammatory cytokines, promoting fibrogenesis. Clinical studies confirm that patients with elevated HOMA-IR values exhibit a higher prevalence of advanced (stage III–IV) fibrosis. Thus, insulin resistance may be regarded not only as a metabolic disturbance but also as a prognostic marker in chronic hepatitis.

Obesity and visceral fat accumulation constitute the morphological substrate for hepatic steatosis. Evidence demonstrating a 1.5–1.6-fold increased risk of cirrhosis in overweight patients underscores the significance of metabolic burden. Viral inflammation and lipotoxic injury act synergistically, creating a persistent environment of hepatocellular damage. In particular, the high prevalence of steatosis in genotype 3 infection illustrates the interaction between viral and metabolic determinants.

Dyslipidemia and arterial hypertension further impair hepatic microcirculation and hepatocyte membrane stability. Altered lipid profiles increase the generation of reactive oxygen species, thereby stimulating collagen synthesis and accelerating fibrosis. In this context, the components of metabolic syndrome should not be viewed as isolated factors but rather as interconnected elements within a unified pathogenetic cascade.

Nutritional factors occupy a central position in this discussion. Diets characterized by excessive caloric intake, high saturated fat content, and refined carbohydrates stimulate de novo lipogenesis in the liver. Excessive fructose consumption contributes to mitochondrial dysfunction and oxidative stress. Conversely, adherence to a Mediterranean dietary pattern has been associated with reductions in inflammatory markers and improvements in insulin sensitivity. A 7–10% reduction in body weight resulting in a 40–60% decrease in steatosis represents a clinically meaningful outcome. These findings support the consideration of diet therapy not as an adjunctive measure but as an integral component of primary therapeutic strategy.

The presence of metabolic syndrome has also been associated with a 1.5–2-fold increased risk of hepatocellular carcinoma. This observation indicates that metabolic disturbances influence not only inflammatory and fibrotic processes but also long-term oncogenic pathways. Therefore, comprehensive prognostic assessment in chronic hepatitis cannot be achieved without accounting for metabolic status.

The discussion highlights that antiviral therapy alone is insufficient for effective management of chronic hepatitis. An integrative strategy—combining antiviral treatment, metabolic correction, weight management, and rational nutrition—appears to be the most appropriate approach. Early identification and correction of metabolic syndrome components in clinical practice are critical for preventing fibrosis progression.

Overall, the findings confirm the decisive role of metabolic factors in the pathogenesis of chronic hepatitis. Hepatic inflammation tends to follow a more severe course in the presence of metabolic overload. Future directions should emphasize individualized nutritional programs, strategies aimed at improving insulin sensitivity, and multidisciplinary management models to reduce complication risks in patients with chronic hepatitis.

**Conclusion**

In patients with chronic hepatitis, metabolic syndrome and dietary factors exert a significant influence on disease course, fibrosis progression, and long-term prognosis. The literature analysis confirms that these determinants amplify viral inflammatory mechanisms and exacerbate hepatic injury.

1. Components of metabolic syndrome, particularly insulin resistance and abdominal obesity, function as independent risk factors accelerating fibrosis progression in chronic hepatitis.
2. Dyslipidemia and excess body weight enhance hepatic steatosis and oxidative stress, thereby increasing the likelihood of cirrhosis development.
3. Inappropriate dietary patterns, especially those rich in calories and saturated fats, intensify hepatic inflammation and aggravate metabolic disturbances.
4. A 7–10% reduction in body weight combined with adherence to a rational dietary model leads to improvements in biochemical parameters, reduction of steatosis, and enhanced insulin sensitivity.
5. Effective management of chronic hepatitis requires an integrative approach that combines antiviral therapy with metabolic correction and individualized diet therapy.

In conclusion, reducing metabolic burden in the context of liver disease not only decreases the risk of complications but also improves patients' quality of life and long-term outcomes. Multidisciplinary strategies and early preventive interventions will be decisive in reducing the global burden of chronic hepatitis.

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