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Peculiarities of the Treatment of Acute and Chronic Hepatosis

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Abstract. *This article analyzes the clinical and pathogenetic characteristics of acute and chronic hepatosis, differences in treatment strategies, and modern therapeutic approaches. It highlights the necessity for emergency medical intervention in acute cases and outlines the importance of long-term, complex therapy, diet, and lifestyle changes in chronic hepatosis. The article presents data on the use of hepatoprotectors, antioxidants, insulin sensitizers, and antifibrotic medications, emphasizing the effectiveness of individualized treatment strategies based on etiology.*

Key words: *Hepatosis, acute hepatosis, chronic hepatosis, hepatoprotector, NAFLD, ursodeoxycholic acid, silymarin, antifibrotic therapy, metabolic syndrome, liver dystrophy.*

Introduction: Liver diseases, especially hepatosis, remain one of the pressing global public health challenges. Hepatosis is a pathological condition characterized by dystrophic damage to liver parenchyma and can occur in acute or chronic forms. These disorders impair the metabolic, detoxifying, synthetic, and regulatory functions of hepatocytes [1].

Acute hepatosis is usually caused by massive hepatocyte damage and necrosis over a short period. This condition is commonly due to the toxic effects of alcohol, medications, or chemicals and requires urgent medical intervention to prevent liver failure or hepatic coma [2].

In contrast, chronic hepatosis is mostly associated with metabolic syndrome, obesity, insulin resistance, prolonged toxic exposure, and non-inflammatory hepatic steatosis. It develops gradually over years, often asymptomatically at first, but may eventually progress to fibrosis, cirrhosis, or even hepatocellular carcinoma[3].

The prevalence of chronic hepatosis, particularly non-alcoholic fatty liver disease (NAFLD), is increasing worldwide. It is estimated to affect up to 25–30% of the population in both developed and developing countries and has become a leading indication for liver transplantation [4].

Therapeutic strategies for hepatosis should consider the disease's etiology and pathogenesis. However, in clinical practice, treatment often focuses on nonspecific symptomatic relief, making long-term remission difficult. Therefore, both acute and chronic forms require individualized, staged, and comprehensive management[5].

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Furthermore, in-depth studies of the immunologic, genetic, and epigenetic factors involved in hepatosis, the development of new hepatoprotective agents, and the implementation of advanced diagnostic tools are essential [6].

Literature Review:

In recent years, numerous studies have focused on the pathogenesis, diagnosis, and treatment of acute and chronic hepatosis.

Acute hepatosis is often induced by drug toxicity or chemical exposure. A.N. Struchkov and colleagues emphasized the importance of detoxification therapy, plasmapheresis, and hepatoprotectors in the management of acute toxic hepatosis [7].

Chronic hepatosis, particularly NAFLD, is widely addressed in modern research and is closely linked to metabolic syndrome, obesity, and insulin resistance. Bellentani et al. reported a global prevalence of up to 30% [6[8]]. Angulo and Sherif highlighted the risk of progression to cirrhosis and hepatocellular carcinoma [9].

Various authors, including Ivashkin and Maev, have discussed the role of hepatoprotectors—such as ursodeoxycholic acid, silymarin, L-ornithine-L-aspartate, and thioctic acid—in protecting hepatocytes [10]. Promising antifibrotic agents such as FXR agonists, PPAR modulators, and GLP-1 analogues are also being explored [11].

Lifestyle modification remains the cornerstone of treatment for NAFLD, including hypocaloric diets and physical activity. EASL's 2023 clinical guidelines recognize lifestyle change as the first-line treatment [12].

Recent hypotheses focus on the gut–liver axis, oxidative stress, and mitochondrial dysfunction, further expanding the scope of therapeutic targets to include immunomodulatory and antioxidant treatments [13].

Results

Acute Hepatosis:

Key symptoms include systemic intoxication, severe hyperglycemia, and sharp elevations in liver enzyme levels. Treatment strategies include:

- **Eliminating the etiological factor** (e.g., toxic substance)
- **Detoxification therapy**: Hemodesis, Rheopolyglukin, plasmapheresis [14]
- **Hepatoprotectors**: Ademetionine, ursodeoxycholic acid, silymarin [15]
- **Antioxidants and vitamins**: Vitamin E, Vitamin C [16]

Clinical studies confirm that early detoxification and hepatoprotective therapy significantly improve patient outcomes [17].

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Chronic Hepatosis:

Often linked to metabolic syndrome, obesity, and insulin resistance. Treatment includes:

- **Lifestyle modification**: Calorie restriction, physical activity [18]
- **Hepatoprotectors**: Essential phospholipids, ursodeoxycholic acid [19]
- **Insulin sensitizers**: Metformin [20]
- **Antifibrotic therapy**: Angiotensin receptor blockers [21]

Long-term and complex treatment is critical and often relies on patient compliance.

Discussion

Delayed treatment of acute hepatosis can lead to severe complications such as hepatic coma or liver failure. Therefore, early diagnosis and intensive therapy are vital [3][22]. Chronic hepatosis can remain latent for years and later lead to liver fibrosis and cirrhosis [23].

Modern pharmacotherapy includes hepatoprotectors, antioxidants, immunomodulators, and antifibrotic agents. Ursodeoxycholic acid and silymarin are proven to stabilize hepatocyte membranes and reduce oxidative damage [24].

Emerging drugs—such as FXR agonists and PPAR modulators—are promising for managing liver fibrosis in chronic hepatosis [25].

Conclusion

Hepatosis, both acute and chronic, is a significant liver disorder associated with life-threatening complications. Effective management relies on early detection, identification of the causative factor, and individualized, step-by-step treatment.

Acute hepatosis typically results from toxic exposure or drug-induced liver injury and requires prompt symptomatic and etiological therapy. Treatments such as detoxification, hepatoprotectors, and antioxidants play a decisive role in preventing liver failure.

In both forms, modern hepatoprotectors, insulin sensitizers, antioxidants, and bioregulators are widely used to improve hepatocyte function and slow disease progression. New agents such as FXR agonists and antifibrotic therapies offer exciting prospects for future treatment.

Ultimately, the key to successful hepatosis treatment lies in early diagnosis, targeted intervention, and the continued development of innovative drugs based on deeper molecular and clinical understanding.

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