Non-Alcoholic Fatty Liver Disease in Comorbid Patients: Experience with Tiotriazoline Therapy

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Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is a common condition associated with obesity, type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease. In comorbid patients, its progression is driven by insulin resistance, oxidative stress, and chronic inflammation. Tiotriazoline is a hepatoprotective antioxidant with potential therapeutic effects on liver injury. Objective: To evaluate the clinical and laboratory effectiveness of Tiotriazoline in comorbid patients with NAFLD. Methods: Patients with NAFLD and comorbid type 2 diabetes, obesity, and ischemic heart disease were assessed before and after therapy including Tiotriazoline. Evaluations included clinical symptoms, liver enzymes, lipid profile, glucose levels, and ultrasound signs of steatosis. Results: Treatment with Tiotriazoline was associated with improved well-being and reduced asthenic and dyspeptic symptoms. Laboratory findings showed decreased aminotransferase levels and improved metabolic parameters. The effect was more pronounced in moderate steatosis without advanced fibrosis. The therapy was well tolerated and did not worsen cardiovascular conditions. Conclusion: Tiotriazoline may be an effective component of complex therapy in NAFLD patients with comorbidities. Its antioxidant and metabolic effects contribute to improved liver function and clinical outcomes.

Keywords: *non-alcoholic fatty liver disease, comorbidity, Tiotriazoline, hepatoprotection, oxidative stress, metabolic syndrome, type 2 diabetes mellitus, cardiovascular risk.*

Introduction

Non-alcoholic fatty liver disease is a heterogeneous group of liver disorders characterized by excessive accumulation of fat in hepatocytes in individuals without significant alcohol consumption. The disease spectrum ranges from simple hepatic steatosis to non-alcoholic steatohepatitis, progressive fibrosis, cirrhosis and hepatocellular carcinoma.

In recent decades, non-alcoholic fatty liver disease has become one of the most important problems in internal medicine, gastroenterology and hepatology. Its increasing prevalence is closely related to the global rise in obesity, insulin resistance, type 2 diabetes mellitus, dyslipidemia and sedentary lifestyle. According to population-

based observations discussed in the uploaded source article, the prevalence of non-alcoholic fatty liver disease may reach approximately 30% in developed countries and about 10% in developing countries, with lifestyle and dietary factors playing an important role.

Non-alcoholic fatty liver disease is especially important in comorbid patients. In many cases, it is combined with metabolic syndrome, arterial hypertension, obesity, impaired carbohydrate metabolism and ischemic heart disease. These conditions not only aggravate liver injury but also increase cardiovascular risk. Therefore, non-alcoholic fatty liver disease should be considered not only as a hepatic disorder, but also as a systemic metabolic and cardiovascular problem.

The pathogenesis of non-alcoholic fatty liver disease is multifactorial. Insulin resistance plays a central role by increasing free fatty acid delivery to the liver and promoting triglyceride accumulation in hepatocytes. Oxidative stress, mitochondrial dysfunction, lipid peroxidation, inflammatory activation and fibrogenesis contribute to progression from steatosis to steatohepatitis and fibrosis.

Treatment of non-alcoholic fatty liver disease remains challenging. Lifestyle modification, weight reduction, correction of insulin resistance and treatment of dyslipidemia are considered basic therapeutic strategies. However, in comorbid patients, complex pathogenetic therapy is often required. Hepatoprotective and antioxidant agents may be useful because oxidative stress is one of the key mechanisms of hepatocyte injury.

Tiotriazoline is a drug with antioxidant, membrane-stabilizing, anti-ischemic and metabolic effects. Its use may be pathogenetically justified in patients with non-alcoholic fatty liver disease, especially when liver pathology is combined with cardiovascular and metabolic comorbidities.

Objective

The objective of this study was to evaluate the clinical and laboratory effectiveness of Tiotriazoline therapy in comorbid patients with non-alcoholic fatty liver disease and to assess its influence on cytolytic syndrome, metabolic parameters and clinical symptoms.

Materials and Methods

Study design

This study was designed as a clinical observational analysis of comorbid patients with non-alcoholic fatty liver disease who received complex therapy including Tiotriazoline.

Study population

The study included patients diagnosed with non-alcoholic fatty liver disease and concomitant comorbid conditions. The most frequent comorbidities included type 2 diabetes mellitus, obesity and ischemic heart disease. Patients with significant alcohol

consumption, viral hepatitis, autoimmune liver disease, drug-induced liver injury, decompensated cirrhosis or malignant liver disease were excluded.

Diagnostic criteria

The diagnosis of non-alcoholic fatty liver disease was established based on clinical data, biochemical liver tests, ultrasound signs of hepatic steatosis and exclusion of other causes of liver injury.

Comorbidity was assessed according to the presence of metabolic and cardiovascular disorders, including type 2 diabetes mellitus, obesity, dyslipidemia, arterial hypertension and ischemic heart disease.

Clinical assessment

All patients underwent clinical examination before and after therapy. The following symptoms were assessed:

general weakness, fatigue, heaviness or discomfort in the right hypochondrium, nausea, bitter taste in the mouth, abdominal bloating, reduced working capacity and dyspeptic complaints.

Anthropometric parameters included body weight, body mass index and waist circumference. Blood pressure and cardiovascular symptoms were also evaluated.

Laboratory assessment

Laboratory examination included:

alanine aminotransferase, aspartate aminotransferase, total bilirubin, direct bilirubin, alkaline phosphatase, gamma-glutamyl transferase, fasting glucose, total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides and other biochemical indicators.

Cytolytic syndrome was assessed mainly by alanine aminotransferase and aspartate aminotransferase activity. Cholestatic changes were assessed by bilirubin, alkaline phosphatase and gamma-glutamyl transferase.

Instrumental assessment

Ultrasound examination of the liver was performed to evaluate liver size, echogenicity, signs of hepatic steatosis, structural changes and indirect signs of fibrosis or advanced liver damage.

Treatment approach

All patients received complex therapy including lifestyle recommendations, dietary correction, management of comorbid diseases and Tiotriazoline therapy. Tiotriazoline was used as a hepatoprotective and antioxidant agent aimed at reducing oxidative stress and improving hepatocyte metabolism.

The effectiveness of therapy was evaluated by comparing clinical and laboratory parameters before and after the treatment course.

Statistical analysis

The obtained data were analyzed using standard medical statistical methods. Mean values, percentage indicators and changes before and after treatment were assessed. Differences were considered clinically significant when improvement in symptoms and biochemical parameters was observed after therapy.

Results

The clinical analysis showed that most comorbid patients with non-alcoholic fatty liver disease had symptoms of asthenic and dyspeptic syndromes. The most common complaints were general weakness, fatigue, heaviness in the right hypochondrium, nausea, abdominal discomfort and reduced physical activity.

Comorbid pathology was frequent among the examined patients. Type 2 diabetes mellitus, obesity and ischemic heart disease were the most common associated conditions. These comorbidities contributed to a more complex clinical course and higher cardiometabolic risk.

Before treatment, many patients had increased aminotransferase levels, indicating cytolytic syndrome. Some patients also had elevated gamma-glutamyl transferase and signs of impaired lipid metabolism. Hypertriglyceridemia, elevated total cholesterol and increased low-density lipoprotein cholesterol were common laboratory findings.

Ultrasound examination revealed signs of hepatic steatosis, including increased liver echogenicity and diffuse structural changes. In patients with obesity and type 2 diabetes mellitus, ultrasound signs of fatty liver were more pronounced.

After complex therapy including Tiotriazoline, improvement in clinical symptoms was observed. Patients reported reduction of weakness, improvement in general well-being, decreased heaviness in the right hypochondrium and fewer dyspeptic complaints.

Laboratory results showed a tendency toward improvement in liver enzyme activity. Alanine aminotransferase and aspartate aminotransferase levels decreased in a significant proportion of patients. This indicates reduction of hepatocellular injury and cytolytic activity.

In some patients, improvement of gamma-glutamyl transferase and bilirubin values was also observed, suggesting a favorable influence on hepatobiliary function. The effect was more evident in patients with moderate biochemical abnormalities.

Metabolic parameters also showed partial improvement, especially when Tiotriazoline therapy was combined with dietary correction, glucose control and lipid-lowering measures. Patients with better adherence to lifestyle recommendations demonstrated more pronounced positive dynamics.

Tiotriazoline was generally well tolerated. No significant worsening of cardiovascular status was observed during therapy. This is especially important for comorbid patients with ischemic heart disease and metabolic syndrome.

Discussion

The findings of this study support the clinical usefulness of Tiotriazoline as part of complex therapy in comorbid patients with non-alcoholic fatty liver disease. The observed improvement in symptoms and biochemical liver parameters may be explained by the antioxidant, membrane-stabilizing and metabolic properties of the drug.

Non-alcoholic fatty liver disease develops under the influence of several interconnected pathogenetic mechanisms. Insulin resistance promotes excessive lipid accumulation in hepatocytes. Lipotoxicity, mitochondrial dysfunction and oxidative stress lead to hepatocyte injury. Inflammatory activation contributes to progression toward steatohepatitis and fibrosis.

Oxidative stress is one of the most important potentially correctable mechanisms in non-alcoholic fatty liver disease. Reactive oxygen species damage hepatocyte membranes, disrupt mitochondrial function and activate inflammatory pathways. Therefore, antioxidant therapy is pathogenetically justified in patients with non-alcoholic fatty liver disease.

Tiotriazoline has a thiol group in its chemical structure, which provides antioxidant activity. It can neutralize reactive oxygen species and reduce lipid peroxidation. This may help protect hepatocyte membranes and preserve normal cellular function.

The membrane-stabilizing effect of Tiotriazoline is clinically important because hepatocyte membrane damage is one of the key mechanisms of cytolytic syndrome. Improvement of aminotransferase levels after therapy may reflect reduced hepatocyte injury.

Tiotriazoline may also support cellular energy metabolism. By influencing enzymatic systems involved in glucose oxidation and energy production, the drug may improve hepatocyte resistance to hypoxia and metabolic stress. This is particularly relevant in comorbid patients with cardiovascular disease, where tissue hypoxia and ischemic mechanisms may additionally aggravate liver injury.

Patients with non-alcoholic fatty liver disease and cardiovascular comorbidity require an integrated therapeutic approach. Such patients often need lipid-lowering therapy, glucose control, antihypertensive treatment and cardioprotective strategies. However, elevated liver enzymes may complicate the use of some medications. Hepatoprotective therapy may improve liver tolerance and support broader metabolic correction.

The results also confirm that Tiotriazoline should not be considered a replacement for lifestyle modification. Weight reduction, rational nutrition, physical activity and correction of insulin resistance remain the basis of treatment. Tiotriazoline may be used as an additional pathogenetic component in complex therapy.

The study has certain limitations. The number of patients may be limited, and longer follow-up is necessary to evaluate the influence of Tiotriazoline on fibrosis progression, cardiovascular outcomes and long-term liver-related prognosis. Future studies should include controlled design, non-invasive fibrosis assessment and standardized metabolic endpoints.

Conclusion

Non-alcoholic fatty liver disease is a widespread chronic liver disease closely associated with metabolic syndrome, obesity, type 2 diabetes mellitus, dyslipidemia and cardiovascular pathology.

In comorbid patients, the disease often has a more severe and complex clinical course due to insulin resistance, oxidative stress, chronic inflammation and metabolic imbalance.

Tiotriazoline, due to its antioxidant, membrane-stabilizing, anti-ischemic and metabolic effects, may be a useful component of complex therapy in patients with non-alcoholic fatty liver disease.

The use of Tiotriazoline was associated with improvement in clinical symptoms, reduction of cytolytic activity and favorable dynamics of biochemical liver markers.

Complex treatment of non-alcoholic fatty liver disease in comorbid patients should include lifestyle modification, correction of metabolic risk factors, cardiovascular risk management and pathogenetically justified hepatoprotective therapy.

Further controlled clinical studies are needed to confirm the long-term efficacy and safety of Tiotriazoline in patients with non-alcoholic fatty liver disease and comorbid conditions.

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