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COMPARATIVE CHARACTERISTICS OF STEATOSIS AND FIBROSIS SEVERITY IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

Non-alcoholic fatty liver disease represents a spectrum of pathological changes characterized by excessive lipid accumulation in hepatocytes (steatosis), subsequent inflammatory damage (steatohepatitis), and progressive fibrosis, which may ultimately lead to liver cirrhosis and hepatocellular carcinoma.

Currently, more than 400 million people worldwide are overweight, and this condition has reached epidemic proportions. NAFLD is detected in approximately 30–100% of individuals with excess body weight and accounts for 60–95% of the overall structure of chronic liver diseases. Recent studies indicate that NAFLD is not only closely associated with other liver disorders but also serves as an important risk factor for type 2 diabetes mellitus and an early marker of premorbid cardiovascular disease. Fatty liver is particularly vulnerable to exogenous damaging factors, including viral and toxic insults.

Keywords: Non-alcoholic fatty liver disease, hepatic steatosis, liver fibrosis fibrosis staging, steatosis severity, histopathological assessment, metabolic risk factors, liver disease progression.

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СРАВНИТЕЛЬНАЯ ХАРАКТЕРИСТИКА СТЕАТОЗА И СТЕПЕНИ ФИБРОЗА У ПАЦИЕНТОВ С НЕАЛКОГОЛЬНОЙ ЖИРОВОЙ БОЛЕЗНЬЮ ПЕЧЕНИ

Неалкогольная жировая болезнь печени (НАЖБП) представляет собой спектр патологических изменений, характеризующихся избыточным накоплением липидов в гепатоцитах (стеатоз), последующим воспалительным повреждением (стеатогепатит) и прогрессирующим фиброзом, который в конечном итоге может привести к циррозу печени и гепатоцеллюлярной карциноме.

В настоящее время более 400 миллионов человек во всем мире имеют избыточный вес, и эта проблема достигла масштабов эпидемии. НАЖБП выявляется примерно у 30–100% людей с избыточной массой тела и составляет 60–95% в общей структуре хронических заболеваний печени. Недавние исследования показывают, что НАЖБП не только тесно связана с другими заболеваниями печени, но и является важным фактором риска сахарного диабета 2 типа и ранним маркером предшествующих сердечно-сосудистых заболеваний. Жирная печень особенно уязвима к внешним повреждающим факторам, включая вирусные и токсические воздействия.

Ключевые слова: Неалкогольная жировая болезнь печени, печёночный стеатоз, фиброз печени, стадирование фиброза, тяжесть стеатоза, гистопатологическая оценка, метаболические факторы риска, прогрессирование заболеваний печени.

Relevance. Non-alcoholic fatty liver disease (NAFLD) is one of the most common chronic liver diseases worldwide and represents a growing public health problem. Its prevalence has increased in parallel with the global rise in obesity,

type 2 diabetes mellitus, and metabolic syndrome. NAFLD includes a wide spectrum of pathological conditions, ranging from simple hepatic steatosis to non-alcoholic steatohepatitis (NASH), progressive fibrosis, cirrhosis, and hepatocellular carcinoma.

Clinical outcomes in NAFLD patients largely depend on the severity of liver fibrosis, which is considered the most important predictor of liver-related and overall mortality. Therefore, comparative evaluation of steatosis severity and fibrosis stage is essential for early risk stratification, monitoring disease progression, and optimizing therapeutic strategies.

Aim. The aim of this study was to compare the severity of hepatic steatosis and the stages of liver fibrosis in patients with non-alcoholic fatty liver disease and to evaluate their association with metabolic risk factors.

Materials and Methods. This study included 120 patients diagnosed with NAFLD who were examined at a tertiary medical center. The diagnosis was based on clinical data, laboratory findings, and imaging studies, with exclusion of significant alcohol consumption and other chronic liver diseases.

All patients underwent physical examination, anthropometric measurements, and biochemical analysis, including liver enzymes, fasting glucose, lipid profile, and markers of insulin resistance. Hepatic steatosis was assessed using abdominal ultrasonography and graded as mild, moderate, or severe. Liver fibrosis was evaluated using transient elastography, and in selected cases, histopathological examination of liver biopsy specimens was performed. Fibrosis staging was classified according to the METAVIR scoring system.

Statistical analysis was conducted using standard methods, with correlation analysis applied to assess the relationship between steatosis severity, fibrosis stage, and metabolic parameters. A p-value <0.05 was considered statistically significant.

Results. Among the studied patients, the distribution of hepatic steatosis demonstrated marked heterogeneity, reflecting different stages of disease progression. Mild steatosis was identified in 45% of patients and was generally characterized by limited lipid accumulation within hepatocytes, minimal

inflammatory activity, and relatively preserved liver function parameters. This group predominantly included patients at an earlier stage of NAFLD, in whom timely lifestyle modification and metabolic control may effectively prevent disease progression. Moderate steatosis was observed in 35% of patients and was associated with more extensive fat deposition in liver tissue, often accompanied by biochemical signs of hepatocellular injury and early inflammatory changes. Severe steatosis, detected in 20% of patients, represented advanced fat infiltration of hepatocytes and was frequently associated with features suggestive of transition toward non-alcoholic steatohepatitis.

Assessment of liver fibrosis revealed that 50% of patients had no or minimal fibrotic changes (F0–F1), indicating either absence of structural remodeling or only early periportal fibrosis. Moderate fibrosis (F2) was identified in 30% of cases and reflected ongoing fibrogenesis with architectural distortion of liver tissue. Advanced fibrosis (F3) was present in 15% of patients and was characterized by bridging fibrosis and significant impairment of hepatic microcirculation, while 5% of patients were diagnosed with cirrhosis (F4), representing the terminal stage of fibrotic progression with irreversible structural and functional alterations of the liver.

Statistical analysis demonstrated a statistically significant positive correlation between steatosis severity and fibrosis stage ($r = 0.62$, $p < 0.01$), indicating that increasing hepatic fat accumulation is strongly associated with progressive fibrotic remodeling. This finding supports the concept that steatosis is not a benign condition but plays a critical role in triggering inflammatory pathways, oxidative stress, and activation of hepatic stellate cells, ultimately leading to fibrosis progression.

Furthermore, patients with advanced fibrosis (F2–F4) exhibited significantly higher body mass index values, reflecting a greater prevalence of obesity and central adiposity. Metabolic analysis revealed elevated fasting glucose levels, consistent with insulin resistance and impaired glucose metabolism, as well as increased triglyceride concentrations, indicating dyslipidemia. These metabolic

abnormalities were significantly less pronounced in patients with minimal fibrosis (F0–F1). Collectively, these findings confirm a strong and clinically relevant association between the progression of NAFLD and key components of metabolic syndrome, underscoring the systemic nature of the disease.

Overall, the results emphasize the importance of integrated assessment of hepatic and metabolic parameters in NAFLD patients, as individuals with advanced steatosis and fibrosis represent a high-risk group requiring intensified monitoring and targeted therapeutic interventions.

Conclusion. The results of this study demonstrate a close relationship between the severity of hepatic steatosis and the stage of liver fibrosis in patients with NAFLD. Advanced fibrosis is strongly associated with metabolic abnormalities, including obesity, dyslipidemia, and impaired glucose metabolism. Comprehensive assessment of both steatosis and fibrosis using non-invasive methods is crucial for early diagnosis, risk stratification, and prevention of disease progression and complications in NAFLD patients.

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