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Short Communication

Non-Alcoholic Fatty Liver Disease in Relation to Pulmonary Function Decline

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DESCRIPTION

Non-Alcoholic Fatty Liver Disease (NAFLD) has emerged as one of the most common liver disorders worldwide, driven by rising rates of obesity, type 2 diabetes and sedentary living. Initially considered a condition limited to the liver, it is now increasingly understood as a disorder with widespread systemic effects. The progression of NAFLD is strongly associated with cardiovascular disease, kidney dysfunction, endocrine disturbances and other extrahepatic outcomes. More recently, attention has turned toward the respiratory system, as evidence points to an inverse association between NAFLD and lung function.

Pulmonary function plays a vital role in determining overall health status, influencing physical performance, quality of life and mortality across a wide range of diseases. Declines in measures such as Forced Expiratory Volume in one second (FEV1), Forced Vital Capacity (FVC) and diffusion capacity are predictors of morbidity even in individuals without established lung disease. The finding that NAFLD patients often present with impaired pulmonary function highlights a growing intersection between hepatology and respiratory medicine. Understanding this relationship is necessary for framing NAFLD as a multisystem condition that extends beyond the liver, with implications for prevention and management strategies.

Epidemiology of NAFLD and lung function decline

The global prevalence of NAFLD is estimated to be approximately 25–30% among adults, with higher rates reported in regions where obesity and type 2 diabetes are particularly common. The burden is rising steadily in both developed and developing countries, reflecting changes in dietary habits, urbanization and reduced physical activity. NAFLD affects not only middle-aged adults but also increasingly younger populations, raising concerns about long-term outcomes [1-3].

Several population-based studies have confirmed that individuals with NAFLD display significantly reduced lung capacity compared with those without fatty liver. Cross-sectional studies from East Asia, for example, have shown that ultrasound-diagnosed fatty liver correlates with lower predicted values of FEV1 and FVC, even after controlling for obesity, smoking and other confounders. Longitudinal research has added further weight, demonstrating that NAFLD predicts accelerated decline in lung function over time. Such findings suggest that the association is not merely incidental but may reflect direct biological interactions between hepatic and pulmonary systems.

NAFLD and reduced pulmonary function

Obesity is among the most significant contributors. Excess adiposity, particularly visceral fat, is a strong driver of hepatic fat accumulation and simultaneously restricts diaphragmatic movement, limiting lung expansion. Central obesity also contributes to obstructive sleep apnea, further impairing respiratory function.

Insulin resistance is another central factor. Elevated circulating insulin levels promote hepatic fat storage while also altering pulmonary vascular regulation, reducing perfusion and potentially affecting gas exchange efficiency. This dual influence makes insulin resistance a unifying mechanism linking metabolic dysfunction in the liver and the lungs.

Environmental exposures such as smoking and air pollution further exacerbate both conditions. While smoking is not a primary cause of NAFLD, it amplifies oxidative stress and systemic inflammation, worsening both hepatic and pulmonary injury. Similarly, exposure to fine particulate matter has been associated with increased prevalence of both fatty liver and reduced lung function in urban populations [4-7].

Mechanisms connecting NAFLD and lung function decline

Oxidative stress is another central pathway. Hepatic mitochondrial dysfunction in NAFLD produces excessive reactive oxygen species. These molecules not only injure hepatocytes but also damage vascular endothelium and pulmonary parenchyma, impairing oxygen exchange and accelerating decline in respiratory function.

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Insulin resistance also plays an important role. Altered insulin signaling reduces vascular reactivity and impairs microcirculatory flow. In the lungs, this compromises alveolar capillary perfusion, contributing to reduced diffusion capacity and progressive decline in function [8-10].

CONCLUSION

The relationship between nonalcoholic fatty liver disease and pulmonary function reflects the multisystem nature of metabolic disorders. Evidence from epidemiological studies, clinical research and mechanistic investigations consistently demonstrates that individuals with NAFLD experience reduced lung capacity and diffusion efficiency, independent of traditional risk factors such as obesity and smoking.

Inflammation, oxidative stress, insulin resistance and ectopic fat deposition provide biologically plausible explanations for this association, while the overlap of lifestyle and environmental determinants further reinforces the systemic impact of NAFLD. Recognizing this connection has practical implications. Clinicians should consider lung function when evaluating patients with NAFLD, while respiratory specialists should remain aware of potential hepatic involvement in their patients.

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