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A Brief Report Oxidative Stress in Non-alcoholic Fatty Liver Disease

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Introduction

The most common and rapidly expanding chronic liver disease, both in developed and developing nations, is non-alcoholic fatty liver disease (NAFLD). In the overall adult population, its prevalence is thought to be between 23 and 25%. The load varies between and within regions, with the Middle East (32%) and South America (30%) having the highest frequency and Africa having the lowest (13%). Furthermore, prevalence rates for type 2 diabetes were revealed to be 24.1%, 23.7%, and 27.4%, respectively, in North America, Europe, and Asia. These rates rose to 55.5 % in people who had the disease. NAFLD is a metabolic condition marked by hepatocyte steatosis and fat accumulation without a history of binge drinking (i.e., less than 14 and 21 standard drinks per week on average for men and women, respectively). Simple steatosis (SS) to steatohepatitis (NASH), which is characterised by the presence of lobular inflammation, hepatocyte damage, and ballooning and that can ultimately lead, in a minority of subjects, to hepatic fibrosis, are just a few of the histological alterations that the disease presents with. SS is regarded as a benign disorder because it has minimal to no active inflammation and no fibrosis, none of which contribute to the aggravation or advancement of the disease. However, in a small number of people, namely those with NASH, the condition can worsen and eventually lead to cirrhosis, which increases the risk of hepatocellular cancer and/or liver-related death (HCC). NASH is currently the second most frequent cause of liver transplantation in Europe and the US, and it is anticipated that it will soon be cured of hepatitis C. The increased percentage of non-cirrhotic patients in whose NASH can proceed immediately into HCC is also cause for concern [1].

Description

T Abdominal ultrasonography is typically used to diagnose NAFLD and has been shown in studies that compare it to the gold standard exam (liver biopsy) to have strong sensitivity and high specificity for the diagnosis of steatosis. The examination of steatosis is now carried out using magnetic resonance imaging, particularly in the research field. In the absence of liver biopsy, the diagnosis of NASH is determined using standardised criteria based on the assessment of the NAS (NAFLD activity score), which accounts for independent scores for steatosis, balloon-shaped degeneration, inflammation, and fibrosis. For ethical considerations, the majority of NAFLD patients do not need a liver biopsy, nevertheless. In actuality, a biopsy is not advised, especially in people who do not exhibit severe liver disease [2].

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According to the conventional "two hits" theory, the first stages of fatty liver are primarily caused by increased insulin resistance, which is thought to happen as a result of significant modifications to the body's normal lipid metabolism, such as increased mobilisation of the aforementioned fatty acids (FA) from visceral adipose tissue to the liver. By controlling blood sugar levels and directing nutrients from the bloodstream to cells after meals through a complicated pathway in which nuclear receptors like the liver X receptor (LXR), farnesoid X receptor (FXR), and nuclear factor erythroid 2-related factor 2 (Nrf2) play a crucial role, insulin has an impact on the intestinal-liver-fat axis. While liver tissue is mostly unaffected, insulin resistance typically develops in adipose and muscular tissue. Less glucose is given to muscle and adipose tissue when they lose their sensitivity to the effects of insulin. This leads to a catabolic state in which peripheral adipose tissue is broken down and free FA are released into the bloodstream. Hyperinsulinemia is the outcome of pancreatic beta cells secreting higher amounts of insulin to make up for the elevated blood glucose levels. The liver develops a hyper-anabolic state and continues to generate and store lipids because it has remained mostly insulin sensitive and is exposed to rising amounts of blood glucose, serum triglycerides, and insulin [3,4].

The pathophysiology of NAFLD may also be influenced by the gut microbiome. According to one study, NAFLD patients had considerably higher intestinal permeability and small intestine bacterial overgrowth. It was determined that the intestinal tight connection integrity of these patients was damaged. Colon bacteria can harm intestinal tight junctions by producing alcohol and acetaldehyde on their own. Increased intestinal permeability makes it possible for endotoxins produced by intestinal bacteria to reach the portal circulation and trigger toll-like receptor (TLR)-4 signalling in Kupffer cells, which in turn causes an upsurge in pro-inflammatory cytokines to be released later. TLR-4 can activate pathways that are dependent or independent of Myeloid Differentiation Primary Response 88 (MyD88), with MyD88-dependent signalling being more important in the aetiology of NASH than alcoholic liver disease. Both routes result in the activation of nuclear factor kappa B (NF-B), which is followed by the generation of type I interferon (IFN) and pro-inflammatory cytokines (which are indicative of MyD88-dependent signalling) (being characteristic of MyD88-independent signaling) [5].

Conclusion

The interpretation and connection of some results from experimental and clinical investigations remain the most significant difficulty for OS in NAFLD at the current state of knowledge. Due to a lack of compelling data supporting their use in the treatment of this disease, many of these approaches to restoring a "healthy" lipid profile and/or bolstering the antioxidant status are challenging to implement into routine clinical practise. Undoubtedly, we must acknowledge that understanding of the processes supporting NAFLD has advanced extremely quickly in the past ten years, but at the same time, the disciplines under consideration represent some of the most promising areas for future scientific study.

Conflicts of Interest

The authors declare no conflict of interest.

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