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Short Communication

Pathophysiology Involved in Non-Alcoholic Fatty Liver Disease (NAFLD)

Marchi Sello*

Department of Pathophysiology, University of New South Wales, South Wales, Australia

DESCRIPTION

Non-Alcoholic Fatty Liver Disease (NAFLD), one of the most common types of chronic liver disease, is strongly correlated with obesity, insulin resistance, metabolic syndrome, and genetic components. The Non-Alcoholic Steato Hepatitis (NASH) and Non-Alcoholic Fatty Liver (NAFL) are the pathological stages of NAFLD (NASH). Chronic liver disease carried by medications other than alcohol is referred to as "Non-Alcoholic Fatty Liver Disease" (NAFLD). Steatosis, or excessive fat deposition in the hepatocytes, is one of its defining characteristics. Non-alcoholic steatohepatitis, non-alcoholic fatty liver, and varying degrees of fibrosis upon liver biopsy are the pathological processes involved in NAFLD. Clinically, NAFLD is also related to hepatocarcinoma and liver cirrhosis [1].

Eating patterns, cardiovascular events, genetic polymorphisms of numerous genes, etc. are pathogenic factors for NAFLD. Patients are susceptible to numerous complications, including hypertension, atherosclerosis, and other disorders, when NAFLD progresses from liver steatosis or liver inflammation to fibrosis. However, treatments and the precise pathophysiology of NAFLD are still unknown. Drug therapy techniques for targeted NAFLD treatment are likely to be used, and a number of therapeutic medicines for treating NAFLD have been characterized [2].

The pathophysiology of NAFLD

Two hits hypothesis: To describe the pathophysiology of NAFLD, the "two hits theory" and "multi hits hypothesis" were proposed. The "two hits" hypothesis supposes that abnormalities in the metabolism of glucose and lipids result in an excessive buildup of fatty acids. Fatty acids are able to increase oxidative stress and inflammatory factors that induce hepatocyte damage, an important pathological stage of NAFLD, because they constitute a crucial toxic component in liver cells. As a result, the initial "hit" is connected to lipid metabolism disorders, which are characterised by insulin resistance and a decrease in important adipocytokines including adiponectin and leptin. Steatosis, endoplasmic reticulum stress, oxidative stress, hepatocyte inflammation, and fibrosis are all strongly linked with the second "hit" [3].

Multiple hits hypothesis: The "multiple hits hypothesis" is considered to adequately describe the pathogenesis of NAFLD. This theory holds that a number of variables, including insulin resistance, dietary factors, oxidative stress, inflammatory factors, obesity, type 2 diabetes, hormones, gut microbiota, and epigenetic factors, interact together to cause NAFLD pathogenesis. Nowadays, it is believed that liver-free fatty acids and insulin resistance are important factors in the development of NAFLD. Abnormalities in the metabolism of lipids and glucose can result in an excess of FFA, which enters the liver cells and is converted to triglycerides. Triglyceride accumulation in the hepatocytes results in lipid droplets and initiates NAFL. Imaging can be used to determine the main pathological characteristic of NAFL, which is liver cell steatosis that is greater than 5%. Additionally, too much FFA raises the pressure in the endoplasmic reticulum, the pressure in the mitochondria, and the formation of reactive oxygen species in the liver, which causes inflammation, namely NASH. Hepatocyte damage, portal vein inflammation, and lobular inflammation are among the pathological characteristics of NASH. Some hepatocytes experience apoptosis or necrosis as NASH progresses, producing additional inflammatory substances that activate hepatic stellate cells and cause liver fibrosis. Liver fibrosis, an adverse effect of hepatitis that progresses to cirrhosis and liver cancer and requires a liver transplant for therapy, is the excessive production and deposition of extracellular matrix proteins in the liver. When treating NAFLD-associated fibrosis from a variety of angles using a combination of pharmacological therapy and practical lifestyle modifications [4,5].

CONCLUSION

NAFLD may be caused by genetic susceptibility, diet, intestinal microbes, and other factors. It begins with excessive fat production in the liver that leads to cell degeneration, inflammation, and fibrotic process. Several drugs have been shown to alleviate NAFLD by acting on these proteins.

Correspondence to: Marchi Sello, Department of Pathophysiology, University of New South Wales, South Wales, Australia, E-mail: Marchis32@gmail.com

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