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Popular Article

Non Alcoholic Fatty Liver Disease: An Overview

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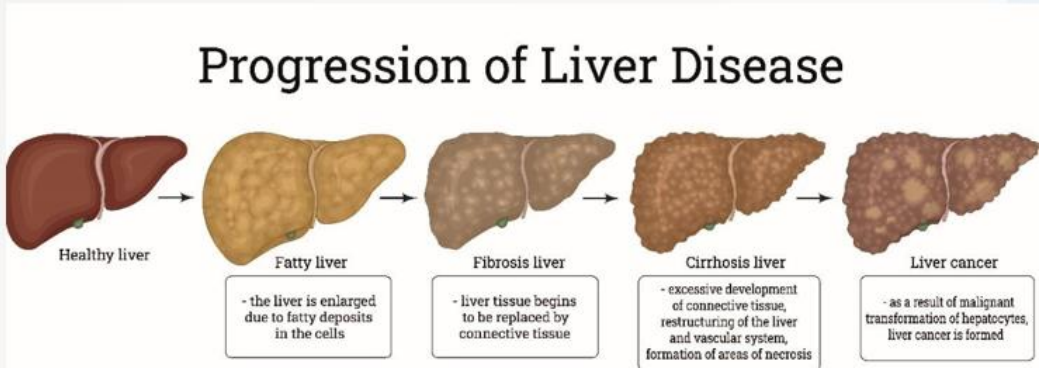
Non-Alcoholic Fatty Liver Disease (NAFLD) has emerged as an important cause of liver disease globally from the past two decades. NAFLD represents a progressive liver disorder ranging from simple liver steatosis to nonalcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and ultimately hepatocellular carcinoma, in the absence of excessive alcohol intake. NAFLD is strongly associated with metabolic abnormalities such as obesity, insulin resistance (IR), and type 2 diabetes. Patients with NAFLD are at high risk of dying from cardiovascular disease and other metabolic diseases (Ma J *et al.*,2017). Liver biopsy followed by histological analysis is the gold standard for confirming the presence and activity of NAFLD, which is histologically diagnosed when hepatic triglyceride accumulation occurs in more than 5% of hepatocytes. The size of fat droplets can differ which can be termed as macro or microvesicular, where macrovesicular steatosis is the predominant pattern seen in NAFLD and is characterized by large vacuoles that occupy the whole cytoplasm and push the nucleus to one side of the cell.Whereas, in some patients of NAFLD present with multiple small lipid vacuoles in the cytoplasm and the nucleus remains unmoved, which is termed “microvesicular steatosis.” .



Pathogenesis of NAFLD

The major factors contributing to NAFLD development are inflammatory responses characterized with lipid accumulation in hepatocytes, cellular stress, and cell death (Gual *et al*., 2017). In NAFLD intra cytoplasmic lipid accumulation with triglycerides is the most important feature in the liver pathogenesis associated with over nutrition and Insulin Resistance (Kitade *et al*., 2017). Fatty acid accumulation and the associated hyperinsulinemia causes inflammation and steatosis. Over nutrition increases Free Fatty Acid influx from diets, resulting in *de novo* lipogenesis in the liver. Over nutrition also induces chronic inflammation and promotes Insulin resistance. Lipotoxicity is due to activation of the c-Jun N-terminal kinase (JNK) signaling pathway resulting in mitochondrial damage and hepatocyte injury (Wong *et al*., 2016). Molecules released from damaged hepatocytes further promote changes in signaling pathways that regulate cellular stress (such as oxidative stress; endoplasmic reticulum stress) and inflammatory responses, thus perpetuating hepatocellular injury and subsequent cell death and promoting NAFLD development.

Conclusion

Once the condition of NAFLD is initiated in the body no specific cure available, to prevent mortality and morbidity it is advised to target weight reduction, health promotion and control of risk factors. NAFLD is related to multiple pathologies such as simple fatty liver to simple steatosis to fibrosis, cirrhosis to hepatic carcinoma. As the liver disease continuous multiple treatment regimens are followed. Recently followed treatment regime is the use of nutraceuticals having antioxidant, anti-inflammatory and immunomodulatory action as **single medication and supplementary medication** at various levels are required to combat different stages of disease progression from fatty liver to cellular stress further to cell death .

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