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Opinion Article

Liver Disease with Regulation of Blood Glucose Levels and Insulin Resistance

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DESCRIPTION

Non-Alcoholic Fatty Liver Disease (NAFLD) is a condition characterized by excessive accumulation of fat in the liver that is not caused by alcohol consumption. NAFLD is the most common liver disease in the world and can range from simple steatosis (fat accumulation without inflammation) to Non-Alcoholic Steatohepatitis (NASH) (fat accumulation with inflammation and liver damage). NASH can progress to cirrhosis (scarring of the liver) and hepatocellular carcinoma (liver cancer). NAFLD is closely associated with metabolic syndrome, a cluster of risk factors that include central abdominal obesity, high blood pressure, high blood sugar, high triglycerides and low HDL cholesterol. These risk factors increase the risk of cardiovascular disease and Type 2 Diabetes Mellitus (T2DM). NAFLD is also considered a hepatic manifestation of insulin resistance, a condition in which the body does not respond properly to the hormone insulin, which regulates blood glucose levels.

Obesity is one of the major risk factors for NAFLD. Up to 80% of patients with NAFLD are obese, defined as a body mass index (BMI)>30 kg/m^2. However, not all obese individuals develop NAFLD, and some lean individuals can also have NAFLD. This suggests that the distribution of body fat plays a more important role than the total amount of body fat in the development of NAFLD. In particular, excess intra-abdominal fat or Visceral Adipose Tissue (VAT) is strongly linked to NAFLD. VAT is the fat that surrounds the internal organs in the abdomen. VAT is metabolically active and secretes various hormones and inflammatory molecules called adipokines that can affect liver function. VAT also releases Free Fatty Acids (FFAs) into the portal vein, which carries blood from the digestive organs to the liver. High levels of FFAs can cause lipid accumulation and oxidative stress in the liver cells, leading to inflammation and fibrosis.

Insulin resistance is another key factor in the pathogenesis of NAFLD. Insulin resistance means that the body's cells do not respond well to insulin, which normally stimulates glucose uptake from the blood into the cells for energy production. As a result, glucose levels remain high in the blood, triggering more insulin secretion from the pancreas. This leads to hyperinsulinemia (high insulin levels in the blood), which can have several adverse effects on the liver. Hyperinsulinemia can stimulate lipogenesis (fat synthesis) in the liver, increasing triglyceride production and storage. Hyperinsulinemia can also inhibit lipolysis (fat breakdown) in adipose tissue, reducing FFA oxidation and increasing FFA delivery to the liver. Hyperinsulinemia can also activate inflammatory pathways in the liver, such as nuclear factor kappa B (NF-κB), which can induce the expression of pro-inflammatory cytokines and chemokines that attract immune cells to the liver. Hyperinsulinemia can also promote fibrogenesis (scar formation) in the liver by stimulating hepatic stellate cells, which are responsible for collagen production.

Most patients with NAFLD are asymptomatic or have nonspecific symptoms such as fatigue, dyspepsia, dull pain in the right upper abdomen and hepatomegaly (enlarged liver). The diagnosis of NAFLD is usually made by imaging tests such as ultrasound or Magnetic Resonance Imaging (MRI), which can detect fat accumulation in the liver. Liver biopsy is the good standard for confirming NASH and assessing fibrosis stage, but it is invasive and has potential complications. The treatment for NAFLD and NASH involves weight reduction through lifestyle modifications, anti-obesity medications and bariatric surgery. Weight loss can improve insulin sensitivity, reduce VAT and FFA levels, decrease inflammation and fibrosis and reverse steatosis.

The American Association for the Study of Liver Diseases recommends a weight loss of at least 7% for patients with NASH. However, weight loss should be gradual and sustained to avoid rapid weight fluctuations that can worsen liver injury. There are currently no approved pharmacological therapies for NASH, but several drugs are under investigation in clinical trials. These include agents that target insulin resistance (such as pioglitazone), lipid metabolism (such as obeticholic acid), inflammation (such as cenicriviroc) and fibrosis (such as simtuzumab). The efficacy and safety of these drugs need to be further evaluated before they can be widely used for NASH treatment.

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CONCLUSION

NAFLD is a common and serious liver disease that is associated with obesity and insulin resistance. The pathogenesis of NAFLD involves complex interactions between genetic factors, environmental factors, dietary factors, hormonal factors and immune factors that affect lipid metabolism, inflammation and fibrosis in the liver. The treatment of NAFLD requires a multidisciplinary approach that aims to reduce weight, improve insulin sensitivity, decrease VAT and FFA levels, modulate adipokines secretion, inhibit inflammatory pathways and prevent fibrogenesis. Future research should focus on identifying novel biomarkers for diagnosis and prognosis of NAFLD, developing new therapeutic agents for NASH and preventing complications such as cirrhosis and HCC.