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Perspective

Role of Thyroid Hormone in Hyperthyroidism Patients Suffering with Non-Alcoholic Fatty Liver Disease

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DESCRIPTION

Nonalcoholic Fatty Liver Disease (NAFLD) is prevalent to the extent of 20%–30%, and obesity and metabolic disease are emerging as major worldwide health concerns. Non-Alcoholic Simple Fatty Liver (NASFL), Non-Alcoholic Steatohepatitis (NASH), liver cirrhosis, and hepatocellular cancer are all included in the vast range of disorders known as NAFLD. NAFLD has been one of the major causes of hepatocellular carcinoma and is increasingly the main cause of cirrhosis. Although the pathogenesis of NAFLD is not fully understood, it is generally agreed that NAFLD results from the interaction of complex genetic background and environmental factors. Insulin resistance increases the lipolysis of adipose tissue, which impacts a large amount of Free Fatty Acids (FFA) into the liver, causing FFA and triglyceride deposition in the liver. The lipid metabolism, insulin resistance, and glucose metabolism are all significantly influenced by the thyroid hormone. New studies have suggested a link between NAFLD and thyroid hormone levels. The correlation between hypothyroidism and insulin resistance was originally noted. Other researches have shown that in populations with hypothyroidism or euthyroidism, the morbidity of NAFLD has an inverse relationship with thyroid hormone levels. In animal models, liver fat content has been decreased using thyroid receptor beta antagonists and thyroid hormone analogues. Thyroid hormone analogues have been shown in clinical investigations to potentially improve NAFLD.

NAFLD is more common in people with hypothyroidism (35.7%–36.3%) than it is in people with euthyroidism (27.4%–33.1%). On the frequency of NAFLD in the hyperthyroid population, there are, nevertheless, incredibly little publications available. According to the Rotterdam Study, NAFLD prevalence ranged from 21.5% in hyperthyroid people to a declining tendency in subjects with hypothyroid, euthyroid, and hyperthyroid thyroid status. The risk of NAFLD steadily decreased as hyperthyroidism replaced hypothyroidism. However, only 7 cases of clinical hyperthyroidism were included in the Rotterdam Study; 114 patients were in a subclinical state. According to our data, clinically hyperthyroid patients had a prevalence of NAFLD of 11.95%, which was lower than that of subclinical hyperthyroid subjects and other thyroid status populations. Uncertainty exists regarding the processes by which thyroid hormone levels affect the amount of liver fat and NAFLD. The thyroid receptor agonist MB078 has been shown in studies using rodent models to lessen hepatic steatosis. Intrahepatic lipolysis is induced by thyroid hormone through activating autophagy. Previous research on hypothyroidism patients suggested that increased insulin resistance, metabolic problems, and NAFLD were brought on by low thyroid hormone levels. As a result of hyperthyroidism, our findings showed that thyroid hormone further decreased the amount of liver fat. The thyroid hormone may encourage the consumption of body fat, help people lose weight, and have a direct effect on the liver, increasing the removal of intrahepatic fat. These effects are independent of both metabolic and inflammatory variables. This study still has certain restrictions. 1) This cross-sectional study lacked follow-up data; 2) The sample size wasn't calculated beforehand and was small. The gender distribution of the patients included was uneven according to the characteristics of the condition; the study was only conducted in a clinic setting, which could have influenced the results. 4) Neither liver Magnetic Resonance Spectroscopy (MRS) nor liver biopsy was employed to precisely measure the amount of liver fat in the study.

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