THE RELATION BETWEEN RETINOIDS AND NON-ALCOHOLIC FATTY LIVER: A REVIEW

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ABSTRACT

Non-alcoholic fatty liver (NAFLD) is the most common liver disease. It carries the risk of progressing to liver fibrosis, cirrhosis and hepatocellular carcinoma. The accumulation of triglyceride in hepatocytes is the hallmark of the disease. NAFLD is defined as liver fat exceeding 5–10% triglyceride of liver weight. Diacylglycerol acyltransferase (DGAT) is the main enzyme for hepatic triglyceride synthesis. A retinoid is a form of vitamin A stored mainly (80%) in the liver of healthy individuals, while diseased liver with NAFLD lacks their retinoid content. The leading cause of death in the NAFLD is a cardiovascular disease due to triglyceride accumulation in the liver of NAFLD patients. Therefore, decreasing triglyceride biosynthesis effect of retinoid could have a beneficial effect in prolonging the life expectancy of NAFLD patients beside improving the hepatic steatosis and insulin resistance. The article summarises the current knowledge of (NAFLD) and its relation to retinoid, providing a better understanding to the readers by discussing the recent finding present in research papers. A keywords search of Medline and PubMed was performed for NAFLD, retinoids, and triglyceride. The search was limited to the English language published papers between 2004-2019. Sixty-nine journal articles were found, and after reviewing the content of each article, 13 had relevant trials for animal and human with NAFLD and included in this review. NAFLD patients and animal model of NAFLD had reported having a decreased serum retinol level, and decreased hepatic antioxidant enzymes, with an increased hepatic level of triglyceride. Additionally, a human study of NAFLD reported that serum retinoid inversely correlates with triglyceride level. Retinoid deficient animals, either transgenic or dietary, had developed steatosis, steatohepatitis and even hepatic tumours. Many researchers reported a decrease in hepatic triglyceride level in NAFLD animal model in response to RA treatment also reported a decrease in the body weight, hepatic steatosis, and increase antioxidant enzymes. Moreover, animals with DGAT2 antisense oligonucleotide significantly inhibited triglyceride accumulation. In conclusion, the retinol deficiency can be the leading cause for the development of NAFLD, it can be used in the future for the treatment of obesity and NAFLD, and the role of retinoids in decreasing hepatic steatosis and triglyceride is not entirely understood. One of the suggested mechanism is through inhibition of hepatic triglyceride synthesis.

Keywords: Non-alcoholic fatty liver, Retinoid, Triglyceride.