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INTESTINAL MICROBIOCENOSIS IN NON-ALCOHOLIC FATTY LIVER DISEASE (LITERATURE REVIEW AND OWN DATA)

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Abstract

In recent years, there has been a steady increase in diseases associated with changes in the normal microbiocenosis of the gastrointestinal tract. Intestinal microbiocenosis is important in the pathogenesis and development of various diseases of the digestive system, in particular, nonalcoholic fatty liver disease (NAFLD). NAFLD is a predictor of metabolic diseases and the most common disease among liver diseases, attracting the attention of specialists in our country and abroad. According to the latest data, the prevalence of Western Europe is 30-40%, and 20% in Asian countries [1,3,7,11,18,20]. From the modern point of view, the development of NAFLD in the metabolic syndrome is visceral (abdominal) obesity and insulin resistance [2,4,10,14,21]. It is known that abdominal adipose tissue actively secretes many different mediators, including free fatty acids (FFA), tumor necrosis factor-alpha (FNO-α), growth factor V1 (TGF B1), resistin, adiponectin, leptin, interleukins, etc. produces [5,9,16,22,25]. Today, it cannot be denied that there is a connection between intestinal microbiocenosis activity in the development of NAFLD and the origin of NAFLD when the activity of intestinal microbiocenosis is disturbed. A number of studies show that the development of the level of intestinal microbiocenosis is accompanied by increased manifestations of metabolic syndrome (MS) and increased insulin resistance (IR) [1,6,12,17,23]. It is known that there are two theories about the pathogenesis of NAFLD. In the case of obesity, especially visceral obesity, the amount of free fatty acids in the liver increases and liver steatosis is formed. Nowadays, one of the main reasons for the increase of FFA in the liver is the increased permeability of the intestinal mucosa, as a result of which FFA moves into the submucosa, then into the bloodstream and into the liver. The progressive development of steatosis lays the groundwork for steatohepatitis. Additional oxidized stress, peroxyoxidized lipids lead to disruption of cellular defense mechanisms, inflammation, and necrosis. NAFLD cytochrome R450 (CYP) 2E1. CYP) 2E1 stimulates the formation of free radicals from endogenous ketones, food nitrosamines, aldehydes [2,11,19,24]. Ketones and fatty acids can be cytochrome mediators. The inflammatory process can develop endotoxinemia in intestinal dysbacteriosis. Lipopolysaccharide, gram-negative bacteria enter the portal vein, activate type 4 immune response, Toll-like receptors,

inflammation and fibrosis develop. In NAFLD, endotoxemia increases the expression of inflammatory cytokines (FNO-α), interleukin-6, 8 and these cytokine receptors. Extraordinary oxide in the liver, endotoxins in high concentration and various aggressive factors (stress, dietal disturbance, drug disorder) lead to an increase in bowel barrier [2,7,11,13,17,22,25]. The intensity of the intestinal microflora and the overthrow of bacteria in the dislocation or small intestine leads to the development of endotoxemia, which enhances inflammation changes in the liver. In increasing the permeability of the intestinal wall, increases the activity of Tolls in the bowel wall, increases the risk of intestinal cavity and the intestinal lymphoid tissue provides the immune response [11,14,21,24].

The digital system epithelium is a natural barter, nutrients, nutrients in the physiological condition, the ions provide a small amount of bacteria and toxins permeability. Selective binders are specialized non-cellular structures - under the supervision of hard compounds, and they were considered not less valued in the transportation of nutrients in the gastrointestinal tract. The permeability of the intestinal wall is administered by interrepile structure - the control of hard compounds. In a healthy person, the intestinal barer consists of 3 floors. 1). Millerly, Mechanical and Immunological (Mutsin and Iga Production)) mucosa to provide functions; 2 epithelium 3) of epithelial 3), which consists of epithelisms and non-incurrished parts - part of the immune system, produces macrophags and lymphocytes [2,9,11,18,21]. As a result of intestinal microbiocentosis, the risk of NAFLD development occurs based on inflammation and metabolic mechanisms. The mechanism of inflammation is the expression of the expression of solid compounds and an increase in ethanol products. Metabolic mechanism is short chain precumbers (SCFAS), the change in the status of liabopy factors, the increase in the amount of grass acids, and the increase in the breed metilamin [3,6,11,22]. The decline in hard combination increases the amount of intestinal barer permeability and fat lipopolisaccharides and fatty acids, and intensifying chronic inflammation of the liver. The increase in chronic inflammation of the liver accelerates the release of ethanol in the bowel. The increase in ethanol causes endokanabinoid regulation and increases the risk of the NAFLD. Endogenol is metaboli in many bowel microflora, after absorption, after absorption, followed by the gate veil, then oxenately and acetaldelegidi. Thus, as a result of etanol metabolism, triglycerides accumulate and oxidative stress. As the amount of Ethanole increases, obesity sharply in the patients and the risk of developing the SG development from NAFLD increases. Ethanol also increases the permeability of the bowel muclut, endotoxemia increases. In short chain

fatty acids (SCFASs), G-protein is connected to the G-protein 43 (GPR43) and brake the lyolizus and enhances the accumulation of fats in the liver fat tissue [6,12,17,20]. From the learned publications, many inspections have been proven to be an integral connection between the bowop microbiocentials and steatogepatitus (SG). The main types of proteobacteria, Actinobacteria, Bacteroides, Bacteridates, Bacteroides, the main types of fireworks on NAFLD and SG were determined. There are large amounts of proteobacteria in NAFLD and SG patients (Esterichia and other generations enteroobacteriacea E) actinobacteria and (bacteroides and prevevotella). Other inspections were seen in the Bacteride species in NAFLD and SG [7,12, 22].

Conclusion

The state of intestinal microbiosenosis is important in the development of NAFLD. Studies show that the corruption of the bowel microbiocenosis increases the risk of steatosis, inflammation (SG), hepatitis and fibrosis risk. Non-alcoholic fatty liver disease and intestinal microbiocenosis are related to each other, and changes in intestinal microbiocenosis increase the risk of liver disease and intestinal microbiocenosis.

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