The relationship between nonalcoholic fatty liver and age, obesity, blood lipids, blood glucose, and dietary principles

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Abstract: This article discussed the close relationship between nonalcoholic fatty liver disease (NAFLD) and blood lipids, blood glucose and dietary principles. The study found that dyslipidemia, hyperglycemia and poor dietary habits are important risk factors for NAFLD. This article elaborates how these factors affect liver fat metabolism, inflammatory response and injury process, and points out that correcting these factors is of great significance for the prevention and treatment of NAFLD. In addition, this article also introduces the dietary intervention strategies for NAFLD, such as low-fat diet and Mediterranean diet, which have shown positive effects in improving liver function and quality of life. Based on the above findings, future research can further explore new dietary intervention strategies, the application of multi factor comprehensive intervention in the treatment of NAFLD and the pathogenesis of NAFLD.

Keywords: nonalcoholic fatty liver disease (NAFLD); Dyslipidemia; Hyperglycemia; Dietary principles; Dietary intervention strategies

1. Introduction

Nonalcoholic fatty liver disease (NAFLD), as an increasingly prominent global health problem, has attracted extensive attention from the medical community and the public health field. The characteristic of this liver disease is that its pathogenesis is not caused by alcohol intake or other known liver damage factors, but abnormal fat deposition in hepatocytes. With the change of modern lifestyle, the incidence of NAFLD has shown an upward trend year by year, which not only seriously affects the quality of life of patients, but also poses a huge challenge to the social health system.[1]

In the past few decades, the global obesity rate has been rising, at the same time, the incidence of NAFLD has also increased. This obvious correlation has caused people to think deeply about the relationship between the two. In addition to obesity as a significant risk factor, age, blood lipid levels, blood glucose status and dietary habits are also considered to be important factors affecting the onset and development of NAFLD.

As an irreversible physiological process, age is closely related to the occurrence and development of NAFLD. With the growth of age, the metabolic function and organ function of human body gradually decline, and the liver's ability to regulate fat metabolism also decreases, which increases the risk of fat deposition in hepatocytes.

Obesity is another important cause of NAFLD. Under the condition of obesity, the adipose tissue in the human body will secrete a series of bioactive substances, which act on the liver through the blood circulation, causing steatosis and inflammatory reaction of hepatocytes, and ultimately leading to NAFLD.

In addition, abnormal metabolism of blood lipids and blood glucose also plays an important role in the pathogenesis of NAFLD. Under the condition of hyperlipidemia and hyperglycemia, the balance of lipid synthesis and oxidation in the liver is broken, and the deposition of fat in hepatocytes is intensified, which accelerates the progress of NAFLD.[2]

As a controllable lifestyle factor, dietary habits are of great significance for the prevention and treatment of NAFLD. Reasonable dietary structure and habits can effectively control weight, blood lipids and blood glucose levels, thereby reducing the risk of NAFLD. Therefore, to explore the relationship between NAFLD and age, obesity, blood lipids, blood glucose and dietary principles has
important theoretical and practical value for formulating effective prevention and treatment strategies.

2. Relationship between nonalcoholic fatty liver disease and age

2.1 Incidence and characteristics of NAFLD in different age groups

The incidence and characteristics of nonalcoholic fatty liver disease (NAFLD) showed significant differences in different age groups. This difference is mainly affected by age-related physiological changes, lifestyle, environmental factors, genetic factors and other factors.

2.1.1 Childhood and adolescence

Although it is traditionally believed that NAFLD mainly affects adults, the incidence of NAFLD in children and adolescents is also rising in recent years. This is mainly related to the change of modern lifestyle, such as high calorie diet, lack of exercise, long-term use of electronic equipment, etc. These factors lead to an increase in obesity rates in children and adolescents, which in turn increases the risk of NAFLD.[3]

NAFLD in children and adolescents is characterized by steatosis of hepatocytes, usually without obvious fibrosis or cirrhosis. However, without timely intervention, the disease may gradually worsen, affecting the growth and development of children and their long-term health.

2.1.2 Adulthood

The incidence of NAFLD is the highest in adulthood. This is mainly related to the lifestyle, work pressure, metabolic changes and other factors of this age group. NAFLD in adulthood is characterized by steatosis, ballooning and fibrosis of hepatocytes. With the development of the disease, some patients may progress to cirrhosis and liver cancer.

2.1.3 Old age

The incidence of NAFLD in the elderly is relatively low, but the characteristics of NAFLD are complex. Elderly NAFLD patients tend to have more chronic diseases, such as cardiovascular disease, diabetes and so on. These diseases and their therapeutic drugs may cause certain damage to the liver, thereby increasing the risk of NAFLD. In addition, the immune system function of the elderly is relatively weak, and the ability to clear pathogens is reduced, which may increase the risk of liver infection, thus inducing or aggravating NAFLD.

In addition to hepatocyte steatosis and fibrosis, the pathological manifestations of NAFLD in the elderly may also be accompanied by hepatocyte atrophy, hepatic sinususctasia and intrahepatic vascular lesions. These changes make the condition of elderly patients with NAFLD more complex, and the treatment difficulty increases accordingly.[4]

2.2 Effect of age-related physiological changes on NAFLD

Age related physiological changes play an important role in the occurrence and development of NAFLD. Here are some major physiological changes and their effects on NAFLD:

2.2.1 Metabolic decline

With the growth of age, the metabolic function of human body gradually declines. This includes fat metabolism, sugar metabolism and protein metabolism. The decline of metabolic function may lead to the increase of fat deposition in the liver, which may lead to NAFLD. In addition, the decline of metabolic function may also affect the metabolic ability of the liver to drugs and toxins, thereby increasing the risk of liver injury.

2.2.2 Weakened immune system function

The immune system function of the elderly is relatively weak, and the ability to clear pathogens is reduced. This makes the elderly more vulnerable to infection by viruses, bacteria and other pathogens, thus inducing or aggravating NAFLD. In addition, the weakening of immune system function may also affect the ability of liver repair and regeneration, making it more difficult to recover from liver injury.

2.2.3 Cumulative effects of chronic diseases

With the growth of age, people's risk of chronic diseases increases gradually. These chronic diseases
include cardiovascular disease, diabetes, hypertension and so on. These diseases and their therapeutic drugs may cause certain damage to the liver, thereby increasing the risk of NAFLD. In addition, the cumulative effect of chronic diseases may also increase the burden on the liver, making the liver more vulnerable to damage.[5]

2.2.4 Change of living habits

With the growth of age, people's living habits will also change. For example, the amount of exercise of the elderly is relatively reduced, and their eating habits may also change (such as eating more high calorie foods). These changes in living habits may lead to weight gain and high blood lipid levels, thus increasing the risk of NAFLD. In addition, changes in living habits may also affect the composition and function of intestinal microbiota, and further affect the health status of the liver.

3. Relationship between nonalcoholic fatty liver disease and obesity

3.1 Epidemiological relationship between obesity and NAFLD

As a global health problem, obesity has a close epidemiological relationship with nonalcoholic fatty liver disease (NAFLD). A large number of studies have shown that the risk of NAFLD in obese people is significantly higher than that in normal weight people. This association is reflected in people of different regions, ages and genders, suggesting that obesity may be an important independent risk factor for NAFLD.

In obese people, the incidence of NAFLD increased with the increase of body mass index (BMI). A large sample size epidemiological survey showed that the prevalence of NAFLD could be as high as 50% among obese people with BMI over 30. This data is much higher than the prevalence rate of normal weight people, which further confirms the close relationship between obesity and NAFLD.

In addition to BMI, waist circumference, waist hip ratio and other body parameters are also associated with the risk of NAFLD. Central obesity (abdominal fat accumulation) is considered to be a particularly important risk factor for NAFLD. This is because abdominal adipose tissue has higher metabolic activity and can secrete more bioactive substances, such as free fatty acids, inflammatory factors and adipokines, which play an important role in the pathogenesis of NAFLD.[6]

3.2 Pathophysiological mechanism of NAFLD caused by obesity

3.2.1 Insulin resistance

Insulin resistance is one of the key pathophysiological mechanisms of NAFLD caused by obesity. In obesity, excessive adipose tissue will lead to the secretion of excessive free fatty acids and inflammatory factors by adipocytes, which will interfere with the normal signal transduction of insulin, thus reducing the sensitivity of target organs to insulin. The occurrence of insulin resistance will further aggravate the deposition of fat in the liver, thus causing or aggravating NAFLD.

3.2.2 Disorder of fat factor secretion

Adipose tissue is not only an energy storage organ, but also an endocrine organ with a variety of biological activities. Under the condition of obesity, the endocrine function of adipose tissue is disordered, which is manifested by the abnormal secretion of a variety of fat factors. Among them, some adipokines (such as adiponectin, leptin, etc.) can improve insulin resistance and protect the liver, while others (such as resistin, visfatin, etc.) may aggravate insulin resistance and liver injury. The secretion disorder of these adipokines plays an important role in the pathogenesis of NAFLD.

3.2.3 Oxidative stress and lipid peroxidation

Under the condition of obesity, excessive adipose tissue will lead to the intensification of oxidative stress in the liver. Oxidative stress refers to the imbalance of oxidation and antioxidation in the body, which leads to the production and accumulation of reactive oxygen species, leading to cell damage. In the liver, oxidative stress can lead to increased lipid peroxidation, which can lead to hepatocyte injury and inflammatory response. These changes play a key role in the pathogenesis of NAFLD.

3.2.4 Intestinal microbiota changes

Recent studies have shown that intestinal microbiota also plays an important role in the pathogenesis of obesity and NAFLD. Under the condition of obesity, the composition and function of
intestinal microbiota change, which is manifested by the increase of harmful bacteria, the decrease of beneficial bacteria and the impairment of intestinal barrier function. These changes will lead to the increase of endotoxin and inflammatory factors in the intestinal tract, and enter the liver through the portal vein system, thus causing or aggravating NAFLD.

There are close epidemiological links and complex pathophysiological mechanisms between obesity and nonalcoholic fatty liver disease. These mechanisms include insulin resistance, lipid factor secretion disorder, oxidative stress and lipid peroxidation, and intestinal microbiota changes. In order to effectively prevent and treat NAFLD, we need to deeply understand these mechanisms and formulate corresponding intervention strategies. For example, by improving lifestyle (such as diet adjustment, increasing exercise, etc.) to reduce weight and improve body size indicators; Regulating the secretion of adipokines and improving insulin resistance through drug therapy; Through antioxidants and probiotics to reduce oxidative stress and intestinal microbiota changes. The implementation of these strategies will help to reduce the risk of NAFLD and improve the quality of life of patients.

4. Relationship between nonalcoholic fatty liver disease and blood lipids

Nonalcoholic fatty liver disease (NAFLD), as a liver disease closely related to metabolism, is closely related to dyslipidemia. Dyslipidemia is not only one of the important characteristics of NAFLD, but also plays a key role in the pathogenesis and development of NAFLD. This article will discuss the correlation between dyslipidemia and NAFLD in detail, as well as the role and mechanism of different lipid components in the pathogenesis of NAFLD.

4.1 Correlation between dyslipidemia and NAFLD

A large number of studies have shown that there is a significant correlation between dyslipidemia and NAFLD. On the one hand, NAFLD patients are often accompanied by dyslipidemia, which is manifested by the increase of serum triglyceride (TG), the decrease of high density lipoprotein cholesterol (HDL-C) and the increase of low density lipoprotein cholesterol (LDL-C). On the other hand, dyslipidemia is also one of the independent risk factors of NAFLD, which can directly promote the deposition of fat in the liver and the occurrence of inflammatory reaction, thereby aggravating liver injury.

In the epidemiological survey, dyslipidemia and NAFLD prevalence showed a significant positive correlation. A large sample size study found that the increase of serum TG level was closely related to the prevalence of NAFLD, while the decrease of HDL-C level further increased the risk of NAFLD. These findings suggest that dyslipidemia may be an important predictor of NAFLD.

4.2 The role and mechanism of different lipid components in the pathogenesis of NAFLD

4.2.1 Triglycerides (TG)

Triglyceride is one of the main lipids in the blood, and its increase is closely related to the pathogenesis of NAFLD. Hypertriglyceridemia can lead to the excessive deposition of fat in the liver, leading to hepatocyte steatosis and inflammatory reaction. In addition, triglycerides can also promote the occurrence of oxidative stress and lipid peroxidation in the liver by activating a variety of signaling pathways and transcription factors, and further aggravate liver injury.

4.2.2 Cholesterol (TC)

Cholesterol is one of the important components of cell membrane, and it is also the raw material for the synthesis of bile acids, vitamin D and other physiological active substances. However, when the blood cholesterol level is too high, it will lead to the increase of its deposition in the liver, which will cause or aggravate NAFLD. Cholesterol can regulate the expression of genes related to fat metabolism and inflammatory response by activating nuclear receptors and other signaling pathways, and then affect the pathogenesis of NAFLD.

4.2.3 High density lipoprotein (HDL)

High density lipoprotein (HDL) is a kind of lipoprotein particles with anti atherosclerotic effect. Its main function is to transport cholesterol in peripheral tissues to the liver for metabolism and excretion. In NAFLD patients, HDL levels tend to decrease, which hinders the reverse transport of cholesterol and further aggravates the lipid deposition and inflammatory reaction in the liver. In addition, HDL also has
antioxidant, anti-inflammatory and other biological activities, which can directly protect hepatocytes from damage.

4.2.4 Low density lipoprotein (LDL)

Low density lipoprotein (LDL) is a kind of lipoprotein particles rich in cholesterol. Its main function is to transport the cholesterol synthesized by the liver to the peripheral tissues for use or storage. In NAFLD patients, LDL levels tend to increase, which increases the deposition of cholesterol in peripheral tissues, and then aggravates the condition of fatty liver. In addition, oxidized LDL also has a stronger pro-inflammatory effect, which can directly activate immune cells and promote the release of inflammatory factors, thereby aggravating the inflammatory reaction and damage of the liver.

5. Relationship between nonalcoholic fatty liver disease and blood glucose

5.1 Comorbidity between diabetes and NAFLD

Nonalcoholic fatty liver disease (NAFLD) is closely associated with diabetes mellitus. Diabetes is a chronic metabolic disease characterized by hyperglycemia, while NAFLD is a liver disease characterized by liver fat deposition and inflammatory reaction. The comorbidity relationship between the two is not only reflected in the correlation of the prevalence rate, but also related to the common pathogenesis and risk factors.

Epidemiological survey showed that the prevalence of NAFLD in diabetic patients was significantly higher than that in non-diabetic patients. On the contrary, NAFLD patients are often accompanied by diabetes. This correlation may be due to their common pathogenesis, such as insulin resistance, obesity, dyslipidemia and other factors.

5.2 Metabolic changes of liver under hyperglycemia and its effect on NAFLD

Under hyperglycemia, a series of changes have taken place in liver metabolism, which play an important role in promoting the occurrence and development of NAFLD. The metabolic changes of liver under hyperglycemia and their effects on NAFLD will be described in detail below.

5.2.1 Insulin resistance

In hyperglycemia, the sensitivity of liver to insulin is reduced, leading to the occurrence of insulin resistance. Insulin resistance weakens the role of insulin in regulating glucose metabolism, leading to further increase in blood glucose levels. At the same time, insulin resistance also promotes the deposition of fat in the liver and aggravates the pathogenesis of NAFLD.

5.2.2 Abnormal fat metabolism

Under hyperglycemia, liver fat metabolism is also abnormal. On the one hand, due to the existence of insulin resistance, lipolysis is weakened, resulting in the accumulation of free fatty acids in the liver. On the other hand, hyperglycemia also promotes the synthesis of fat in the liver and further aggravates the deposition of fat. These changes provide favorable conditions for the occurrence of NAFLD.

5.2.3 Increased inflammatory response

Under hyperglycemia, the inflammatory reaction in the liver is also significantly aggravated. Inflammatory response is one of the important links in the pathogenesis of NAFLD, and hyperglycemia promotes the release of inflammatory factors and the spread of inflammatory response by activating a variety of inflammatory signaling pathways and transcription factors. These changes not only aggravate the degree of liver injury, but also may lead to liver fibrosis and cirrhosis.

5.3 Importance of blood glucose control in NAFLD treatment

In view of the significant impact of hyperglycemia on NAFLD, blood glucose control is particularly important in the treatment of NAFLD. The following will elaborate the importance of blood glucose control in the treatment of NAFLD from several aspects.

5.3.1 Improve insulin resistance

Through effective blood glucose control measures (such as diet adjustment, increased exercise, drug treatment, etc.), the state of insulin resistance can be improved, and the sensitivity of the liver to insulin
can be improved. This helps to reduce blood glucose levels and reduce fat deposition in the liver, thereby alleviating NAFLD.

5.3.2 Correct abnormal fat metabolism

Blood glucose control can also correct the abnormal state of liver fat metabolism, reduce the fat content in the liver by reducing the accumulation of free fatty acids and inhibiting the process of fat synthesis, which is helpful to improve the liver function and prognosis of NAFLD patients.

5.3.3 Inhibition of inflammatory response

Effective blood glucose control can also inhibit the inflammatory response in the liver, reduce the degree of liver injury and promote the repair process by inhibiting the release of inflammatory factors and reducing the infiltration of inflammatory cells, which helps to prevent NAFLD from developing into more serious stages such as liver fibrosis and cirrhosis.

6. Relationship between nonalcoholic fatty liver disease and dietary principles

6.1 Effect of dietary factors on NAFLD

The formation and development of nonalcoholic fatty liver disease (NAFLD) are closely related to dietary factors. Long term unreasonable dietary habits, such as excessive energy intake, high-fat diet, low dietary fiber intake, can lead to the occurrence of NAFLD or aggravate its condition. The influence of dietary factors on NAFLD will be discussed in detail below.[7]

6.1.1 Energy intake

Excess energy intake is one of the important risk factors for NAFLD. Long term intake of excessive energy, especially from high-fat and high sugar foods, will lead to excessive deposition of fat in the liver, leading to NAFLD. Therefore, maintaining the balance of energy intake and consumption is essential for the prevention and treatment of NAFLD.

6.1.2 Fat type

Different types of fat have different effects on NAFLD. Excessive intake of saturated fatty acids and trans fatty acids can significantly increase the risk of NAFLD (ω-3 fatty acids) can protect the liver and reduce the occurrence of NAFLD. Therefore, we should reduce the intake of saturated fatty acids and trans fatty acids and increase the intake of polyunsaturated fatty acids in our daily diet.

6.1.3 Dietary fiber

Dietary fiber is a kind of polysaccharide that is not digested and absorbed by the human body. It has a good effect on regulating blood glucose, blood lipids and intestinal function. Studies have shown that insufficient dietary fiber intake is closely related to the increased risk of NAFLD. Increasing dietary fiber intake helps to improve intestinal microecology, reduce the production and absorption of endotoxin, so as to reduce the burden of liver and improve its function.

6.2 Dietary intervention strategies for NAFLD

Based on the influence of dietary factors on NAFLD, it is of great significance to formulate reasonable dietary intervention strategies for the prevention and treatment of NAFLD. The following will introduce several common dietary intervention strategies for NAFLD.

6.2.1 Low fat diet

Low fat diet is a dietary pattern that limits fat intake, aiming to reduce fat deposition in the liver and improve its function. Low fat diet should control the intake of total fat and saturated fatty acids, appropriately increase the intake of polyunsaturated fatty acids, and ensure adequate carbohydrate and protein supply to meet the energy needs of the body. In addition, low-fat diet should also pay attention to the balanced collocation of food and diversified choices to ensure the comprehensive intake of nutrients.

6.2.2 Mediterranean diet

The Mediterranean diet is a diet model with olive oil, fruits, vegetables, whole grains, beans, nuts and fish as the main ingredients, which has significant anti-inflammatory, antioxidant and
cardiovascular health effects. Studies have shown that the Mediterranean diet also has a good effect on the prevention and treatment of NAFLD. Olive oil in Mediterranean diet is rich in monounsaturated fatty acids, which helps to reduce blood lipids and improve liver function; Rich fruits, vegetables and whole grains provide sufficient dietary fiber and antioxidant substances, which help to reduce the burden of liver and promote its repair and regeneration. In addition, moderate intake of high-quality protein sources such as fish and beans can also help maintain the normal structure and function of the liver.

7. Conclusions

As a complex metabolic liver disease, the pathogenesis of nonalcoholic fatty liver disease (NAFLD) involves many aspects, including dyslipidemia, blood glucose disorders and unreasonable eating habits. In this paper, the relationship between NAFLD and blood lipids, blood glucose and dietary principles was discussed in detail.

First of all, there is a significant correlation between dyslipidemia and NAFLD. Abnormal fluctuations of triglycerides, cholesterol and other lipid components not only participate in the pathogenesis of NAFLD, but also aggravate liver injury by affecting fat metabolism and inflammatory reaction. Therefore, correcting dyslipidemia is of great significance for the prevention and treatment of NAFLD.

Secondly, hyperglycemia is also one of the important risk factors of NAFLD. Hyperglycemia promotes the occurrence and development of NAFLD by affecting insulin resistance, fat metabolism and inflammatory reaction. Effective blood glucose control is essential to improve the prognosis of NAFLD patients.

Finally, dietary habits play an important role in the pathogenesis and prevention of NAFLD. Excessive energy intake, high-fat diet and insufficient dietary fiber intake can increase the risk of NAFLD. Reasonable dietary intervention strategies such as low-fat diet and Mediterranean diet can effectively prevent and treat NAFLD and improve liver function and quality of life of patients.

Based on the above findings, the following aspects can be further explored in the future research and treatment of NAFLD:

Development and application of new dietary intervention strategies: Although the existing dietary intervention strategies have achieved certain results, there are still problems such as individual differences and long-term compliance. In the future, we can develop more personalized and operable new dietary intervention strategies according to the characteristics and needs of different populations, and verify their effectiveness and safety through clinical trials.

Application of multifactor comprehensive intervention in the treatment of NAFLD: the pathogenesis of NAFLD involves multiple risk factors and pathogenesis, and a single intervention measure is often difficult to achieve the desired effect. In the future, multiple factors such as blood lipid control, blood glucose management and diet adjustment can be integrated for intervention, in order to achieve better effect in the treatment of NAFLD. At the same time, we can also explore the comprehensive treatment mode of combining drug and non drug intervention to improve the treatment compliance and quality of life of patients.

In depth study of the pathogenesis of NAFLD: Although there is a certain understanding of the pathogenesis of NAFLD, there are still many unknown areas need to be further explored. In the future, high-throughput omics technology, animal models, clinical trials and other means can be used to further study the key molecules and signaling pathways in the pathogenesis of NAFLD, so as to provide a theoretical basis for the development of new therapeutic targets.

Strengthen international cooperation and exchange: NAFLD has become a global health problem, and countries have accumulated rich experience and resources in research and treatment. In the future, we can strengthen international cooperation and exchange, share research results and lessons learned, and promote the progress of prevention and control of NAFLD worldwide.

References

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