DIET AND NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD): ADVANCES AND MANAGEMENT STRATEGIES – A COMPREHENSIVE REVIEW

R. Sheetal[1,2], and Syeda Farha S[1]

[1]Department of Nutrition and Dietetics, JSS Academy of Higher Education and Research, Mysuru – Karnataka, India
[2]Current Designation: Teaching Assistant, PSG College of Arts and Science, Coimbatore, India.

ABSTRACT

Global public health is at risk from non-alcoholic fatty liver disease, or NAFLD. Fat accumulation in the liver is associated with chronic liver disease (NAFLD), even in the absence of significant alcohol consumption. It could have anything to do with liver inflammation and fibrosis. Because non-communicable diseases have become more common over the past few years, changing one's lifestyle has drastically altered the priorities for health. Between 1990 and 2017, the number of people worldwide with NAFLD grew from 19.34 million to 29.49 million. An estimated 35.4% of Indians are believed to have NAFLD. Nonalcoholic fatty liver disease is the term used when there is no alcohol intake, and ectopic fat deposition exceeds 5% of the weight of the liver (NAFLD). It encompasses a broad range of morphologically diverse liver abnormalities, from basic steatosis to severe forms like non-alcoholic steatohepatitis (NASH), a progressive form of the disease marked by fibrosis, hepatocyte ballooning degeneration, and lobule inflammation. Obesity, diabetes, dyslipidemia, and the so-called insulin resistance or metabolic syndrome are linked to primary NAFLD/NASH. Rarely, secondary NAFLD/NASH can be linked to a variety of illnesses, including pancreatic duodenal resection, endocrine disorders, polycystic ovary syndrome, and sleep apnea. Today's kids face an unparalleled dietary challenge. Food is abundant, yet a growing proportion of kids are overweight or obese due to inadequate nutrition. Hyperinsulinemia with insulin resistance seems to be crucial in pediatric NAFLD. Understanding pediatric NAFLD's pathophysiology is anticipated to enhance our understanding of the condition. Additionally, finding prevention treatments for pediatric NAFLD is imperative for the well-being of children.

ARTICLE HISTORY

Received: July, 22, 2024
Revision: September, 25, 2024
Accepted: November, 05, 2024
Online: June, 03, 2025

doi:
10.20473/jcmphr.v6i1.60756

KEYWORDS
Non-alcoholic fatty liver disease, dietary risk factors, lipid profile, liver enzymes, HOMA IR, non-communicable disease

Corresponding author
Syeda Farha S.
✉ syedafarhas@jssuni.edu.in
Department of Nutrition and Dietetics, JSS Academy of Higher Education and Research, Mysuru – Karnataka, India.

How to cite:
Sheetal, R., and S. Farha, S., 2025. Diet and Non-Alcoholic Fatty Liver Disease (NAFLD): Advances and Management Strategies – A Comprehensive Review. Journal of Community Medicine and Public Health Research. 6(1): 110-122.

Highlights:

1. One-third of people worldwide, in all age categories, suffer from nonalcoholic fatty liver disease (NAFLD), also known as MASLD. It is the primary cause of chronic

liver disease and is closely associated with metabolic syndrome, obesity, and insulin resistance.

2. Although NAFLD has historically been linked to obesity, metabolic dysfunction, genetic predisposition, and dietary variables can also cause it in lean people. This increases the risk of death, particularly in people who have several metabolic syndrome components.

INTRODUCTION

At least one-third of the general population across all age groups is afflicted with Non-Alcoholic Fatty Liver Disease (NAFLD), also known as Metabolic Dysfunction Associated Liver Disease (MASLD), one of the primary causes of chronic liver problems globally. The term NAFLD refers to a group of liver conditions that include cirrhosis, non-alcoholic steatohepatitis (NASH), and simple steatosis, which is defined as macrovesicular steatosis in hepatocytes without inflammation[1].

Globally, the most frequent cause of chronic liver disease is NAFLD. This disease is categorized as the hepatic manifestation of the Metabolic Syndrome due to its strong correlation with obesity, insulin resistance (IR), and dyslipidaemia. In the absence of alcohol usage, ectopic fat accumulation larger than 5% of the liver weight is predictive of NAFLD. In the last ten years, obesity and insulin resistance have emerged as the two primary risk factors for NAFLD. Even in individuals with comparable body mass indices (BMIs), NAFLD can present with a broad spectrum of clinical symptoms. Currently, liver biopsy is the most reliable method of diagnosing NAFLD.

There is not a single therapy strategy that is acceptable to everyone when treating illness. However, a mix of dynamic clinical characteristics impacted by pharmacological therapies and lifestyle choices may help identify valid score-based methods for predicting liver damage and customizing treatment options[2]. Although NAFLD is commonly linked to obesity, metabolic problems can also be the cause of it in non-obese persons. People with the advancing form of the disease and associated fibrosis, in addition to having NASH, are often more likely to have components of the metabolic syndrome, notably type 2 diabetes and visceral obesity. Patients with NAFLD who have multiple metabolic syndrome components are at a higher risk of dying[3].

NASH was discovered initially in adults in the late 1970s, and in children in the early 1980s. It was not until 25 years later that NAFLD's high prevalence in adults, kids, and teenagers was recognized. As the proportion of children who are obese rises, NAFLD is becoming the most common chronic liver condition affecting children. The dietary challenges that today's youth face are unsurpassed. Food is plentiful, but non-communicable diseases are becoming more common[1].

Since obesity and metabolic disorders are frequently associated with NAFLD, it is thought that NAFLD represents the adult metabolic syndrome's hepatic manifestation. NAFLD has been seen in adults who are slender and underweight, although the condition was formerly thought to be the result of obesity. Since the conclusion of the previous decade, there has been an upsurge in the number of subjects categorized as lean and non-obese NAFLD. There is proof that malnutrition can result

in metabolic issues and eventually lead to NAFLD. Due to truncal fat accumulation, altered metabolism, and a loss of muscle mass, thin and underweight adults may also develop NAFLD. Even if an adult is not obese or overweight, their food intake, particularly a diet heavy in fat and sugar, can still have an impact on their chance of developing NAFLD.

It was discovered that patients with lean NAFLD had more pronounced metabolic disturbances. Reduced muscle mass, altered body composition, and reduced lipid turnover may all contribute to metabolic abnormalities in lean individuals. Dietary consumption, particularly a high-fructose, high-fat diet, may potentially contribute to the onset of non-alcoholic fatty liver disease, or NAFLD, in individuals who are not obese or overweight. NAFLD in underweight and thin individuals can also be attributed to genetic predispositions, type 2 diabetes mellitus, and other disorders associated with the metabolic syndrome. According to multicentre cohort research, those with NAFLD who are underweight or thin may also be more likely to die than those who are overweight or obese. Studies show that 5-8% of people with a BMI ≤ 25 kg/m2 may have NAFLD. Furthermore, it has been discovered that 8–19% of Asians with an average body mass index ≤ 25 kg/m2 have NAFLD[4].

OVERVIEW

PREVALENCE

One of the leading causes of chronic liver disorders in the world is NAFLD, commonly referred to as MASLD. According to estimates, 32% of people globally have NAFLD; men are more likely to have it (40%) than women (26%). Due to global differences in obesity rates, genetic, and socioeconomic factors, NAFLD incidence varies significantly by geography[5].

According to a children's autopsy study that employed liver histology to gauge the prevalence of non-alcoholic fatty liver disease (NAFLD) in the US, 9.6% of children between the ages of 2 and 19 had the condition. Fatty liver became more common as people grew older; at its highest, it was 17.3% among those in the 15- to 19-year-old range and 0.7% for people in the 2-4 age range[6]. NAFLD is now the second-leading cause of liver transplantation in the United States as a result of its increasing occurrence[3]. The prevalence of fatty liver rose with age, peaking at 17.3% for those between the ages of 15 and 19 and 0.7% for those between the ages of 2-4[6].

According to data from the World Health Organization, the number of overweight and obese individuals worldwide has surpassed that of underweight and malnourished individuals. A study done in the UK claims that in the last 40 years, the prevalence of obesity among kids and young adults (between the ages of 5 and 19) has risen tenfold[7]. The majority of NAFLD patients also have concomitant conditions such as dyslipidaemia, insulin resistance, obesity, and diabetes mellitus. It has also been discovered that non-alcoholic fatty liver disease increases the risk of extrahepatic conditions such as chronic renal disease and cardiovascular disease[8]. NAFLD is thought to be the most prevalent manifestation of liver illness in the paediatric population in developed nations, having a predicted mean rate of 7.6% in children in general and 34.2% in obese children[5]. Youngsters with NAFLD are more likely to develop

type 2 diabetes, frequently develop concurrent metabolic syndrome, and progress to end-stage liver disease[9].

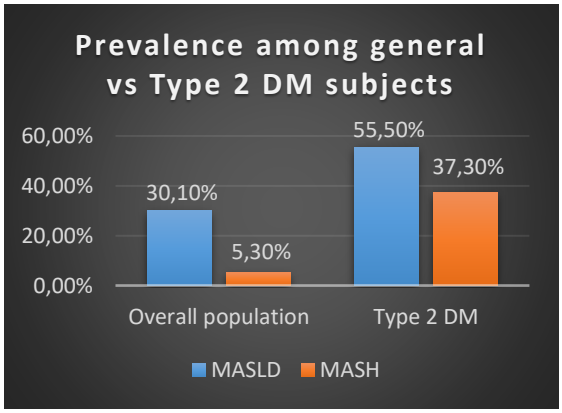

Figure 1 compares patients with Type 2 diabetes and the general population's NAFLD prevalence.

Asia has a diverse population of ethnic groups and socioeconomic backgrounds, which contributes to the significant variations in the incidence of NAFLD among its member nations. With a prevalence of 42%, Southeast Asia has the highest rate of NAFLD among Asian subregions. According to South Asian data, the prevalence of NAFLD was 24.74% in Sri Lanka, 26.2–33.86% in Bangladesh, and 25.7–32.74% in India[5].

The Global Burden of Disease Study (GBD) 2019 discovered that the rate of NAFLD increased from 12.4% in 1990 to 19.7% in Central Asia, despite the absence of data in the area. The high incidence of slender NAFLD (BMI<23) and non-obese NAFLD (BMI <25) is one characteristic of the NAFLD epidemic in Asia. Compared to other ethnic groups, Asians are more likely to have visceral obesity, which could explain up to 19% of non-obese Asians with NAFLD[5].

CAUSES AND RISK FACTORS RELATED TO NAFLD

Numerous metabolic risk factors, such as insulin resistance, obesity, dyslipidaemia, and cardiovascular disease, have been shown to have a substantial correlation with NAFLD. NAFLD is believed to be the hepatic manifestation of a more widespread and fundamental metabolic illness. While cases of NASH-related cirrhosis and juvenile NAFLD have been documented involving individuals who were two and eight years old, respectively, nearly all of these patients frequently manifest clinically beyond the age of ten. The typical age limit for a diagnosis is between 11 and 13[10].

NAFLD is frequently seen in people who are obese or have type 2 diabetes. While NAFLD is typically diagnosed in obese individuals, metabolically unwell non-obese patients may also have it. In addition to NAFLD, patients with multiple metabolic syndrome components also have an increased chance of dying[3]. NAFLD often goes undiagnosed until there is substantial damage to the liver's cells and/or other organs, until a concurrent acute liver injury presents with worse clinical outcomes than anticipated, or until NAFLD-related diseases like resistance to insulin and type 2 diabetes manifest. Children may report non-specific symptoms, including headaches, tiredness, irritability, and difficulty concentrating, in addition to specific symptoms. The expansion of the liver capsule could be the reason for the latter. While there is a slight chance of cirrhosis and liver failure in adults with simple steatosis, the problem appears to progress more quickly in children, with many acquiring NASH and hepatic fibrosis in their early adolescence or adulthood. Paediatric patients with more significant liver damage typically have a worse prognosis and worse liver repercussions[10].

One important risk factor for NAFLD is obesity. Therefore, it is essential to consider that when deciding whether to get a follow-up workup. Compared to 21% and 34% of the non-NAFLD population, respectively, 71% of NAFLD patients had hyperlipidaemia, and nearly 93% had insulin resistance[3].

An individual with a metabolic disorder is more inclined to acquire coronary artery disease with atherosclerosis (ASCVD), type 2 diabetes, and chronic renal failure. Both non-alcoholic fatty liver disease and metabolic syndrome are significantly influenced by insulin resistance. Abdominal weight gain, atherogenic dyslipidaemia, hypertension, increased plasma glucose, a prothrombotic condition, and a pro-inflammatory state seem to be the primary underlying risk factors[11]. According to estimates, 8% of children and adolescents (6 years old) are classified as severely obese, while 20% of them are overweight or obese. In addition, type 2 diabetes (T2DM) has also become much more common in this age group, with rates jumping from 0.34 per 1,000 in 2001 to 0.46 per 1,000 in 2009. This suggests that type 2 diabetes is becoming more common in this age group overall (30.5%)[3].

RISK FACTORS OF PAEDIATRIC NAFLD

Many factors, including genetics and lifestyle, probably need to coexist for steatosis and fibrosis to develop. Many risk variables have been found, such as visceral fat, age, gender, race/ethnicity, obesity, and insulin resistance.

Obesity and visceral adiposity: Being overweight is linked to high BP, hyperinsulinemia, dyslipidaemia, and raised fasting glucose levels, primarily as central obesity. Children who have a higher body mass index are far more likely to develop liver fibrosis, and individuals who are considered obese possess a threefold higher risk of hepatic fibrosis than people who are not obese.

Visceral body fat: Compared to abdominal obesity, which is linked to abdominal obesity, muscularity has less effect. Due to the adipocytes' enhanced lipolytic activity and capacity to transfer FFAs straight into the portal vein, which is needed for the liver to convert to TGs, subjects with considerable visceral obesity have higher plasma FFA levels. Overindulgence in FFA causes hepatocyte injury.

Insulin resistance: Insulin resistance in the liver and throughout the body contributes to elevated levels of FFAs in the blood. Hepatic triglycerides (TG) arise due to the skeletal muscle and fat tissue's reduced ability to metabolize TG due to an elevated level of free fatty acids (FFAs). Adipokines, including adiponectin and leptin, have been linked to the development of NAFLD and are known to modify insulin resistance. Adolescents with NAFLD were found to have lower levels of adiponectin and higher amounts of leptin in their blood.

Gender: According to research utilizing population-based and clinical series methods, men are more inclined than women to acquire NAFLD. Type 2 NASH is more prevalent in boys and early prepubertal girls (mean age 10.5 years), but type 1 NASH is most prevalent in older pubertal females (mean age 13.3 years). The impact of sex hormones is one reason for the gender differences in the occurrence of fatty liver and/or the beginning of NAFLD[6].

PATHOGENESIS OF NAFLD

Over the past 20 years, the prevalence of NAFLD has nearly doubled in the US, and during the past few decades, paediatric obesity has increased globally. NAFLD affects boys twice as often as girls, and its development is significantly influenced by age, sex, and ethnicity[12]. Histology is necessary to make a conclusive diagnosis of NAFLD. Adults and children exhibit very different NASH features and patterns. In children, traditional adult traits are categorized as "type 1"; these findings include pericellular fibrosis and/or lobular inflammation, mainly in the acinar zone, ballooning degeneration, and macro-vesicular steatosis. Macro-vesicular steatosis with systemic inflammation and/or fibrosis is the

hallmark of pediatric "type 2" NASH; lobular inflammatory and/or cellular damage are usually absent[6].

Three or more of the characteristics listed below define the metabolic syndrome: belly fat (waist circumference >102 cm for men and >88 cm for women); low HDL-C (less than 40 mg/dl for men, less than 50 mg/dl for women, or on drug therapy for low HDL-C); poor fasting glucose levels (100–125 mg/dl or anti-diabetic treatment); and high triglycerides (>150 mg/dl or on drugs therapy for elevated triglycerides). The development of MS is primarily caused by compensatory hyperinsulinemia and insulin resistance. Additionally, it has been claimed that the leading cause of multiple sclerosis onset is obesity. Insulin resistance is a key player in the pathophysiology of fatty liver. When regular insulin levels either fall too low to trigger an appropriate metabolic response or require higher than usual concentrations, this is one of the two circumstances that characterize insulin resistance[11].

There appears to be a causal connection between adult fatty liver and obesity. When extrapolating this association to the pediatric age range, especially for young children, care should be used because inherited metabolic diseases (IMD) can mimic or coexist with a diagnosis of NAFLD. When hepatocytes are seen under a light microscope, either micro-vesicular or macro-vesicular, fat defines steatosis. Large fat droplets accumulate due to excess free fatty acid supply in macro-vesicular steatosis, and the nucleus is moved to the cell's periphery. Although most of the cell is occupied by large fat droplets, there are also tiny droplets of macro-vesicular steatosis, which exhibit one or more distinct little fat droplets that may not displace any nuclear material. Both macro-vesicular and micro-vesicular steatosis can coexist with NAFLD[13].

Research on both humans and animals has demonstrated that hypertriglyceridemia, which is primarily brought on by the liver's excess production of very low-density lipoprotein (VLDL), is one of the primary features of multiple sclerosis (MS)[11]. Because multiple factors cause non-alcoholic fatty liver disease (NAFLD), its etiology is yet unknown. Steatosis is facilitated by the dysregulation of hepatic lipid absorption, oxidation, synthesis, and secretion, as well as the movement of very low-density lipoproteins[14].

Studies using the "two-hit theory" previously described the pathophysiology of NAFLD. The first hit is a rise in liver fat linked to hepatic triglyceride buildup and insulin resistance. The second hit includes oxidative damage, inflammatory cytokines, mitochondrial failure, and adipokines.

The "multiple-hit model," which has become the most widely accepted explanation, proposes that altered crosstalk between multiple organs and tissues, such as the gut, liver, pancreas, and adipose tissue, along with an array of genetic and environmental variables, may be the reason why metabolic dysfunction is more common[12].

DIAGNOSIS AND ASSESSMENT OF THE CONDITION

Many people are not diagnosed until the disease has advanced, rendering risk factor modification and current or experimental treatments ineffective. This is because, up until they reach an advanced stage, neither NAFLD nor NASH exhibits any symptoms. (16) Hepatic steatosis, the exclusion of other liver diseases, excessive alcohol consumption (≥30 g/day for men and ≥20 g/day for women), and other manifestations of fatty liver (e.g., methotrexate, systemic steroids) are the basis for a diagnosis of NAFLD. Based on hepatic steatosis and metabolic risk factors, the finding of NAFLD was reclassified as metabolically-associated fatty liver disease (MAFLD) in

2020. The test most commonly used to diagnose hepatic steatosis is abdominal ultrasonography because of its accessibility and price.

Basic steatosis scores, like the fatty liver index, take metabolic risk variables and liver enzymes into account[15]. To diagnose, evaluate, and monitor non-alcoholic fatty liver disease (NAFLD) for an extended duration, non-invasive imaging techniques should be utilized to quantify hepatic fibrosis and steatosis[6].

Liver histology is necessary to evaluate non-alcoholic fatty liver disease in adults and children accurately. The most common kind of pediatric NAFLD, SS, is not always benign. In cirrhosis linked to NAFLD, steatosis is not readily seen. It is generally thought that a significant factor in the development of non-alcoholic fatty liver disease (NAFLD) is hepatic insulin resistance. While the etiology of pediatric non-alcoholic fatty liver disease (NAFLD) may not be the same as that of adult NAFLD, hepatic insulin resistance in conjunction with hyperinsulinemia is a key component of NAFLD[1].

Research on adult hepatic steatosis frequently uses computed tomography (CT). However, CT should not be used in clinical therapy or pediatric NAFLD studies due to the ionizing radiation it produces and the availability of alternative modalities. Various methods for evaluating NAFLD use magnetic resonance imaging (MRI)[6].

A significant portion of NAFLD patients are obese. It is an independent risk factor substantially correlating to the illness's advancement. The primary source of triglycerides that cause steatosis is visceral fat. This likely explains why some people with NAFLD have central obesity despite generally being thin. Both lower glucose tolerance (43%) and a more significant prevalence (62%), respectively, are associated with a recent diagnosis of diabetes mellitus 40. In a prospective study of 100 individuals with type 2 diabetes mellitus, the 49% prevalence of NAFLD indicates that diabetes mellitus is a significant independent risk factor for NAFLD. The test results most closely linked to individuals with NAFLD are insulin resistance and hyperinsulinemia[11].

Liver enzymes are not accurate or dependable indicators on their own. Although unintentionally abnormal liver enzymes are frequently reported in NAFLD patients, up to 80% of these people may have regular liver testing. Moreover, those with significant liver illness had reduced alanine aminotransferase levels. The NAFLD Liver Fat Score (NLFS), which has shown sufficient accuracy in diagnosing NAFLD, is used to measure the fat content in the liver. The Fatty Liver Index (FLI) is derived from the serum triglyceride and gamma-glutamyltransferase (GGT) levels, waist circumference, and BMI. It has proven to be quite effective at identifying fatty liver[16].

When hepatocytes are seen under a light microscope, the presence of fat, which can be either micro-vesicular or macro-vesicular, defines steatosis. Furthermore, over 50% of NAFLD patients may have normal ALT levels. Since fibrosis assessment is the histological characteristic most substantially correlated with liver-related outcomes, focusing on it among individuals with NAFLD15 makes sense. The diagnosis and risk-level-based classification of the patients are shown in Figure 2.

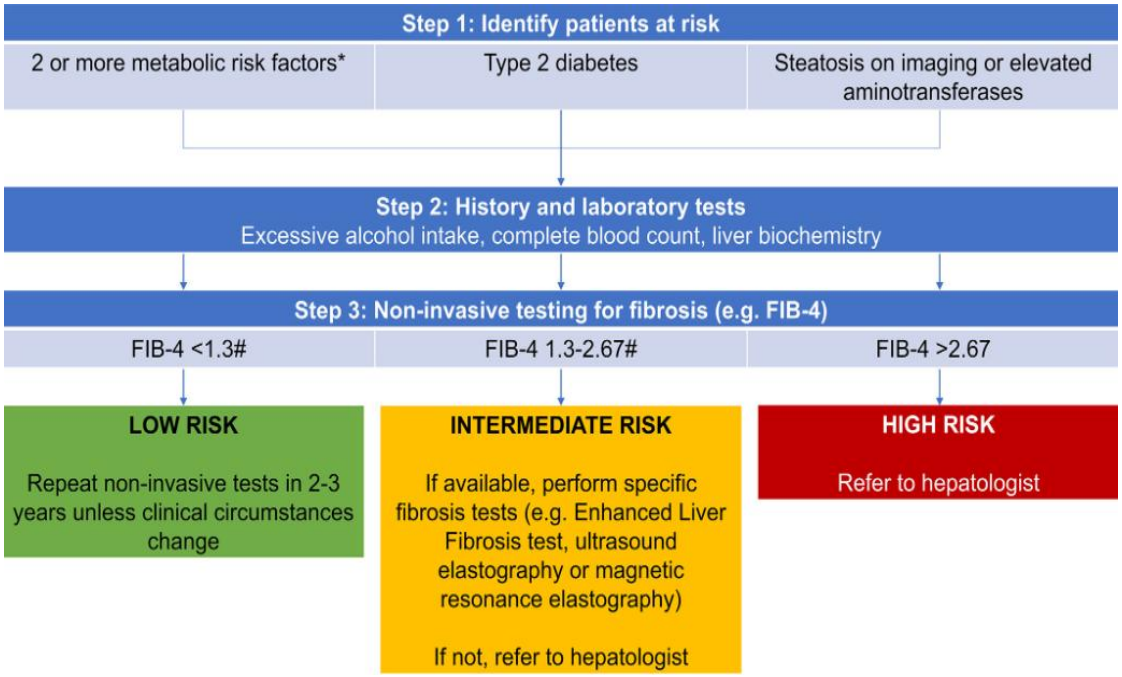

Figure 2: Diagnosis of the patients at risk and their classification based on level of risk.

RELATIONSHIP BETWEEN INSULIN RESISTANCE AND NAFLD

Insulin resistance is a significant contributing factor to hepatic steatosis and steatohepatitis. It is defined as a condition whereby an increased-than-average insulin dose is needed to induce a quantitatively normal response. Many years later, it was discovered that the pathophysiologic mechanism of endogenous hyperinsulinemia causes anomalies in the metabolism and endocrine system[14]. Insulin resistance (IR) is the impairment of the appropriate cascade impact of insulin signalling in target tissues, primarily the liver, adipose tissue, and muscle. It is believed that when IR is considered to begin, up to 70% of glucose disposal occurs in muscle tissue. Excess free fatty acids (FFA) and immune-mediated inflammatory alterations are the causes of ectopic lipid accumulation.

Steatosis and IR in muscle tissue are worsened by increased circulating free fatty acid (FFA) and lipolysis in adipocytes caused by IR in adipose tissue. Insulin limits the postprandial rise in glucose and inhibits glycogenolysis during calorie ingestion, reducing hepatic glucose synthesis. This feedback system is compromised in IR, increasing hepatic glucose synthesis even in postprandial glucose elevation[17].

Two recent studies found a connection between an elevated risk of NAFLD and resistance to insulin and excessive intake of red or processed meat. An increased risk of NAFLD was shown in a study including a cross-sectional sample of patients receiving screenings at a gastroenterology department who consumed considerable amounts of animal protein, especially meat[18].

DIETARY FACTORS INFLUENCING THE RISK OF NAFLD

Diet has been demonstrated to be a significant pathogenic component in NAFLD within the last several decades. Research looking at the effects of certain foods or nutrients has shown that diet and the prevalence of NAFLD are related. However, meals and nutrients are swallowed in various combinations rather than absorbed separately[19]. Diet is a significant contributing factor to the onset of NAFLD. The initial hit of NAFLD is mainly influenced by genetics and positive energy balance

(NAFLD). But the second "hit" and the severity of NAFLD are influenced by the makeup of the diet, underscoring the need for NAFLD management and therapy.

NAFLD has been linked to an overly fattening diet, particularly one high in animal protein, refined carbohydrates, and saturated fats. Several investigations have confirmed this link. Adult NAFLD is also associated with increased meat and carbonated beverage consumption[20]. Global observations show that both adults and children are becoming obese at an increasing pace. While genetic predisposition may play a role, unhealthy eating conditions that restrict access to and the cost of nutritious food are primarily responsible for the significant rise in obesity rates observed globally[7].

In particular, being overweight, driven by excessive consumption of food, has an impact on the development of NAFLD. Individuals with NAFLD often have deficiencies in vitamins, minerals, and polyunsaturated fatty acids in addition to increased intake of calories, carbs, and fat[21]. Dietary hazards included excessive amounts of red meat, processed meat, drinks with added sugar, trans fatty acids, and sodium, along with reduced consumption of vegetables, fruits, lentils, whole grain foods, nuts/seeds, milk, fiber, calcium, and omega-3 fatty acids found in seafood[7].

Impact of High-Calorie Foods: Fast food, takeout, and fried foods are a few examples of diets high in calories. Young individuals who ingested 2273 ± 558 KCal (fat: 36% ± 5.7%, sugar: 95 ± 42 g) per day received 5753 ± 1495 kcal (fat: 43% ± 6.8%, sugar: 285 ± 117 g) of hyperalimentation for four weeks as part of a study assessing the effect of fast food on liver function. The body weight rose to 74.0 kg from 67.6 kg, whereas serum ALT levels increased to 97 ± 103 U/L from 22.1 ± 11.4 U/L. These results suggested that a diet high in energy density can simply and significantly raise the risk of obesity and NAFLD.

Carbohydrates: Consuming excessive amounts of simple carbohydrates, such as fructose and sucrose, represents one of the primary manifestations of NAFLD. Globally, there is a dramatic rise in the use of soft drinks, particularly those with added sugar. However, eating complex carbohydrates in moderation—especially those found in whole grains—may stop the onset of NAFLD or prevent it from worsening when entire grain foods contain dietary fiber, antioxidant vitamins, and minerals[21].

Fructose-rich foods: The body uses glucose as its primary energy source, but fructose metabolism mainly occurs in the liver. Global fructose consumption has increased over the last 10 years, which could contribute to the growth in cases of non-alcoholic fatty liver disease (NAFLD). Consuming fructose can affect the influx of portal endotoxins, the functioning of the liver, inflammatory and metabolic functions, and gut flora[22].

Lipid and Cholesterol content in the food: A high-fat diet caused inflammation, insulin resistance, hepatic steatosis, and increased tumor necrosis factor α (TNFα). Thus, overindulging in meals heavy in cholesterol and saturated fats is one of the primary risk factors for developing NAFLD. Examining the food records from both obese and non-obese persons with the illness, the findings revealed that NAFLD patients consumed significantly more cholesterol than healthy controls. This implies that the development of NAFLD requires nutrition, regardless of weight, and dietary cholesterol consumption is necessary.

Polyunsaturated Fatty acids: Patients with NAFLD exhibited decreased PUFA ingestion compared to healthy persons, even with increased fat intake. Moreover, we found that

PUFA intake was significantly lower in non-obese NAFLD patients than in obese patients. These findings suggest that NAFLD patients have an unbalanced diet and that PUFA deficiency contributes to the onset and progression of the illness.

Intake of Nuts and Seeds: Nut and seed consumption and lowered risk of NAFLD: Nuts are an excellent source of nutrients and additional components supporting general wellness despite their image as a high-calorie snack. Nuts are a great source of minerals, high-quality protein, fiber, and water-soluble vitamins, including folic acid and folate. Condensed amounts of oxidative stress, insulin resistance, inflammation, and metabolic syndrome—all of which are components of the pathophysiology of non-alcoholic fatty liver disease—appear to be caused by the soluble bioactive elements (tocopherols, tocotrienols, phytosterols, sphingolipids, carotenoids, and chlorophylls) and essential fatty acids (MUFA and PUFA) found in nuts[23]. Owing to their correlation with decreased visceral fat and general adiposity, nuts are probably protective against the excessive buildup of hepatic fat and, consequently, against NAFLD[10,11]. A mainstay of the Mediterranean diet, nuts are a rich source of fiber, protein, unsaturated fats, antioxidants, and polyphenols. These nutrients might offer defense against inflammation and oxidative stress, two things known to speed up the onset of NAFLD[24].

MANAGEMENT OF NON-ALCOHOLIC FATTY LIVER DISEASE

Treatment strategies are desperately needed because NAFLD can manifest in childhood and has been shown to adversely affect patients' prognoses for some illnesses. Nutrition therapy is the cornerstone of care for those with NAFLD and those at risk of acquiring the sickness. is something that most people have[21]. Regardless of the diet type utilized to achieve that goal, there is a relation between the percentage of fat loss and the enhancement in the histology of the liver that follows lifestyle modification. Both low-fat and low-carb diets can help reduce liver fat[15].

NAFLD is now treated with diet and lifestyle modifications aimed at weight loss, management of underlying metabolic risk factors, and use of pharmaceuticals in cases of severe fibrosis or NASH. A patient with NAFLD usually eats too much meat, soft drinks, sugary foods, and saturated fat but not enough fiber, fish oil, omega-3 fatty acids, or specific vitamins. The good news is that significant decreases in liver fat occur in conjunction with improvements in hepatic IR, even with relatively little weight loss[25]. The Mediterranean diet, low-carb, low-fat, and Dietary Approaches to Stop Hypertension (DASH) diet are the four most popular dietary regimens[26].

The European Union suggests the Mediterranean diet plan to treat NAFLD. This dietary pattern is an example of nutritional therapy for NAFLD, which outperforms weight loss, improving both hepatic and extrahepatic health. The most decisive proof supporting the advantageous effects of two variations of the Mediterranean diet comes from the two most prominent and most prolonged experiments. First, an 18-month randomized controlled study (RCT) showed that a low-carb Mediterranean diet was superior to a low-fat diet in reducing intrahepatic fat[15]. Probiotics have been demonstrated to improve the pathogenesis of non-alcoholic fatty liver disease by changing the internal environment. Probiotic treatment had positive therapeutic outcomes in animal models, including improvements in serum ALT, cholesterol, oxidative stress, insulin resistance, inflammation, and liver fibrosis in addition to reductions in liver fat[21].

NAFLD affects about one-third of inactive individuals, and half of them never exercise. It has been demonstrated that physical activity reduces the risk of insulin resistance, metabolic syndrome, dyslipidemia, hypertension, and type 2 diabetes[25]. Even without weight reduction, raising physical activity levels in NAFLD patients lowers their hepatocellular damage indicators and intrahepatic lipid concentration. Exercise was found to lower intrahepatic triglyceride (IHTG) levels in people with NAFLD without significantly changing weight, according to a systematic review and meta-analysis of 17 research on the topic[26].

Strength and limitations

The study presents an extensive review of Non-Alcoholic Fatty Liver Disease (NAFLD), with strengths in its exhaustive discussion on dietary risk factors, pathogenesis, prevalence, and therapeutic methods. It is pertinent to clinical and public health contexts because it incorporates contemporary epidemiological data, emphasizes the role of insulin resistance, and addresses pediatric NAFLD. Its practical implications are further enhanced by incorporating dietary intervention tactics like probiotics and the Mediterranean diet. The study's shortcomings, however, include its reliance on secondary sources, lack of original data, and less attention to the efficacy of pharmacological therapies. Furthermore, despite emphasising dietary changes, it offers neither a comprehensive quantitative analysis nor the findings of randomised controlled trials to support its assertions.

CONCLUSION

This review emphasizes the prevalence of NAFLD, which affects one-third of the world's population and is closely associated with obesity, insulin resistance, and metabolic syndrome, even in those who are lean. High consumption of animal protein, sugars, and saturated fats, as well as PUFA deficiencies, are identified as major dietary hazards, and the significance of poor diets and increase inactivity in pediatric cases is emphasized. The review's usefulness as a practical manual for early clinical and public health interventions stems from its concise synopsis of the mechanisms behind NAFLD, its diagnostic difficulties, and its successful non-pharmacological treatments, particularly those involving Mediterranean diets and probiotics.

ACKNOWLEDGMENT

The authors would like to acknowledge the support of the JSS Academy of Higher Education and Research in carrying out the research.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

FUNDING

The authors thank the JSS Academy of Higher Education and Research for providing financial support for the original research.

AUTHOR CONTRIBUTION

All authors have contributed to all processes in this research, including preparation, data gathering, drafting, and approval of this manuscript for publication.

REFERENCE

1. Roberts EA. Pediatric nonalcoholic fatty liver disease (NAFLD): a "growing" problem?. Journal of hepatology. 2007 Jun 1;46(6):1133-42. DOI: 10.1016/j.jhep.2007.03.003
2. Meroni M, Longo M, Tria G, Dongiovanni P. Genetics is of the essence to face NAFLD. Biomedicines. 2021 Sep 30;9(10):1359. DOI: 10.3390/biomedicines9101359
3. Arshad T, Paik JM, Biswas R, Alqahtani SA, Henry L, Younossi ZM. Nonalcoholic Fatty Liver Disease Prevalence Trends Among Adolescents and Young Adults in the U nited S tates, 2007-2016. Hepatology Communications. 2021 Oct;5(10):1676-88. DOI: 10.1002/hep4.1760
4. Fahim SM, Chowdhury MA, Alam S. Non-alcoholic fatty liver disease (NAFLD) among underweight adults. Clinical nutrition ESPEN. 2020 Aug 1;38:80-5. DOI: 10.1016/j.clnesp.2020.06.002
5. Teng ML, Ng CH, Huang DQ, Chan KE, Tan DJ, Lim WH, Yang JD, Tan E, Muthiah MD. Global incidence and prevalence of nonalcoholic fatty liver disease. Clinical and molecular hepatology. 2022 Dec 14;29(Suppl):S32. DOI: 10.3350/cmh.2022.0365
6. Barshop NJ, Sirlin CB, Schwimmer JB, Lavine JE. epidemiology, pathogenesis and potential treatments of paediatric non-alcoholic fatty liver disease. Alimentary pharmacology & therapeutics. 2008 Jul;28(1):13-24. DOI: 10.1111/j.1365-2036.2008.03703.x
7. Paik JM, Mir S, Alqahtani SA, Younossi Y, Ong JP, Younossi ZM. Dietary risks for liver mortality in NAFLD: global burden of disease data. Hepatology Communications. 2022 Jan;6(1):90-100. DOI: 10.1002/hep4.1707
8. Jasirwan CO, Lesmana CR, Hasan I, Sulaiman AS, Gani RA. The role of gut microbiota in non-alcoholic fatty liver disease: pathways of mechanisms. Bioscience of microbiota, food and health. 2019;38(3):81-8. DOI: 10.12938/bmfh.18-032
9. Jain V, Jana M, Upadhyay B, Ahmad N, Jain O, Upadhyay AD, Ramakrishnan L, Vikram NK. Prevalence, clinical & biochemical correlates of non-alcoholic fatty liver disease in overweight adolescents. Indian Journal of Medical Research. 2018 Sep 1;148(3):291-301. DOI: 10.4103/ijmr.IJMR_1966_16
10. Temple JL, Cordero P, Li J, Nguyen V, Oben JA. A guide to non-alcoholic fatty liver disease in childhood and adolescence. International journal of molecular sciences. 2016 Jun 15;17(6):947. DOI: 10.3390/ijms17060947
11. Paschos P, Paletas K. Non alcoholic fatty liver disease and metabolic syndrome. Hippokratia. 2009 Jan;13(1):9. https://www.hippokratia.gr/2018/09/14/non-alcoholic-fatty-liver-disease-and-metabolic-syndrome/
12. Fang YL, Chen H, Wang CL, Liang L. Pathogenesis of non-alcoholic fatty liver disease in children and adolescence: From "two hit theory" to "multiple hit model". World journal of gastroenterology. 2018 Jul

7;24(27):2974.
DOI: 10.3748/wjg.v24.i27.2974

13. Hegarty R, Deheragoda M, Fitzpatrick E, Dhawan A. Paediatric fatty liver disease (PeFLD): all is not NAFLD–pathophysiological insights and approach to management. Journal of hepatology. 2018 Jun 1;68(6):1286-99. DOI: 10.1016/j.jhep.2018.02.006
14. Marušić M, Paić M, Knobloch M, Liberati Pršo AM. NAFLD, insulin resistance, and diabetes mellitus type 2. Canadian Journal of Gastroenterology and Hepatology. 2021;2021(1):6613827. DOI: 10.1155/2021/6613827
15. Wong VW, Zelber-Sagi S, Cusi K, Carrieri P, Wright E, Crespo J, Lazarus JV. Management of NAFLD in primary care settings. Liver International. 2022 Nov;42(11):2377-89. DOI: 10.1111/liv.15404
16. Piazzolla VA, Mangia A. Noninvasive Diagnosis of NAFLD and NASH. Cells. 2020 Apr 17;9(4):1005. DOI: 10.3390/cells9041005
17. Fujii H, Kawada N, Japan Study Group of Nafld (JSG-NAFLD). The role of insulin resistance and diabetes in nonalcoholic fatty liver disease. International journal of molecular sciences. 2020 May 29;21(11):3863. DOI: 10.3390/ijms21113863
18. Noureddin M, Zelber-Sagi S, Wilkens LR, Porcel J, Boushey CJ, Le Marchand L, Rosen HR, Setiawan VW. Diet associations with nonalcoholic fatty liver disease in an ethnically diverse population: the multiethnic cohort. Hepatology. 2020 Jun;71(6):1940-52. DOI: 10.1002/hep.30967
19. Liu X, Peng Y, Chen S, Sun Q. An observational study on the association between major dietary patterns and non-alcoholic fatty liver disease in Chinese adolescents. Medicine. 2018 Apr 1;97(17):e0576. DOI: 10.1097/MD.0000000000010576
20. Mirmiran P, Amirhamidi Z, Ejtahed HS, Bahadoran Z, Azizi F. Relationship between diet and non-alcoholic fatty liver disease: a review article. Iranian journal of public health.2017Aug;46(8):1007. https://ijph.tums.ac.ir/index.php/ijph/article/view/10599/5246
21. Yasutake K, Kohjima M, Kotoh K, Nakashima M, Nakamuta M, Enjoji M. Dietary habits and behaviors associated with nonalcoholic fatty liver disease. World Journal of Gastroenterology: WJG. 2014 Feb 2;20(7):1756. DOI: 10.3748/wjg.v20.i7.1756
22. Alwahsh SM, Gebhardt R. Dietary fructose as a risk factor for non-alcoholic fatty liver disease (NAFLD). Archives of toxicology. 2017 Apr;91:1545-63. DOI: 10.1007/s00204-016-1892-7
23. Asbaghi O, Emamat H, Kelishadi MR, Hekmatdoost A. The association between nuts intake and non-alcoholic fatty liver disease (NAFLD) risk: a case-control study. Clinical nutrition research. 2020 Jul;9(3):195. DOI: 10.7762/cnr.2020.9.3.195
24. Cardoso BR, Tan SY, Daly RM, Dalla Via J, Georgousopoulou EN, George ES. Intake of nuts and seeds is associated with a lower prevalence of nonalcoholic fatty liver disease in US adults: findings from 2005–2018 NHANES. The Journal of Nutrition. 2021 Nov 1;151(11):3507-15. DOI: 10.1093/jn/nxab253

25. Munteanu MA, Nagy GA, Mircea PA. Current management of NAFLD. Clujul medical. 2016;89(1):19. DOI: 10.15386/cjmed-539

26. Cardoso AC, de Figueiredo-Mendes C, A. Villela-Nogueira C. Current management of NAFLD/NASH. Liver International. 2021 Jun;41:89-94. DOI: 10.1111/liv.14869