Non Alcoholic Fatty Liver: A new lifestyle disease

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Abstract

**Background:** Non-alcoholic fatty liver disease (NAFLD) is the accumulation of lipid, primarily in the form of triacylglycerols in individuals who do not consume significant amounts of alcohol. The other known causes of steatosis, such as certain drugs and toxins, have been excluded. The rising incidence of obesity is associated with health complications. The non-alcoholic fatty liver disease is increasingly being recognized as a major cause of liver related morbidity and mortality among 15-40% of the general population. Currently, a liver biopsy is the gold standard method for diagnosing NAFLD. Ultrasonography is relatively inexpensive and widely available in clinical settings. NAFLD is considered to be an integral part of the metabolic syndrome. The present study is designed to study the clinical profile of patients with NAFLD with varying degrees of severity as diagnosed by Ultrasonography and evaluate the relationship between the non-alcoholic fatty liver disease and the metabolic syndrome.

**Methods and Result:** A cross-sectional study was conducted with a total of 100 cases that were included and investigated for NAFLD. **Results:** On physical examination mean BMI was 27.3±3.8kg/m2. Mean diastolic blood pressure was 80.2±8.9 mm Hg and mean systolic blood pressure mm Hg 138.1±15.6. The correlation was significant for fasting plasma glucose, diastolic blood pressure, triglycerides, high-density lipoprotein and waist circumference (table 1). Waist circumference was 90.2±9.1cms, Serum LDL, Serum HDL, Serum VLDL, & Serum triglycerides were (mg/dl) 126.2±37.3, 42.6±5.9, 26.7±9.6, 270.4±90.3 respectively.

Introduction

The term “fatty liver” usually refers to a non-alcoholic fatty liver disease (NAFLD), distinguishing it from other liver diseases stemming from high alcohol consumption or other causes (1). NAFLD affects people of all ages and is currently one of the leading causes of chronic disease. It is not related to alcohol consumption; in fact, obesity is the most common trait of people with fatty liver. It is due to eating an excess of fatty or high-calorie foods which can cause fat to build up in the liver (2).

Non-alcoholic fatty liver disease (NAFLD) represents a spectrum of disease from non-alcoholic fatty liver (NAFL), characterized by hepatic fat accumulation without inflammation, to non-alcoholic steatohepatitis (NASH), characterized by hepatic fat deposition with
inflammation, accumulating fibrosis, and ultimately liver cirrhosis (3). NASH-related cirrhosis is currently a leading cause of chronic liver disease and is associated with hepatocellular cancer (4).

**Causes and risk Factors Associated with NAFLD/NASH**

Exactly how and why fatty liver develops is not clear. It occurs when the body produces too much fat, or when it cannot process fat properly. Obesity is a clear risk factor. Around 70 percent of people with obesity have the condition, while 10 to 15 percent of people with a normal weight have it. Regardless of their weight, a person with "deep" abdominal fat is more likely to have a fatty liver (5). Other risk factors include:

(a) **Obesity:** Body mass index (BMI) indicates whether a person is a healthy weight for their height. The World Health Organisation (WHO) recommends BMI of 23 kg/m² and 27.5 kg/m² to define overweight and obesity respectively in Asians.

(b) **Diabetes:** Limit consumption of high-sugar food and drinks to prevent excessive caloric intake.

(c) **Metabolic Syndrome:** This is a combination of high blood pressure and blood sugar levels, obesity and high cholesterol; similar to risk factors for cardiovascular disease (CVD). NAFLD is one of the most common forms of liver disease. It is often linked to insulin resistance but is more frequently associated with type 2 diabetes mellitus and obesity. NAFLD patients are at higher risk of CVDs such as coronary heart disease and stroke. However, the links between fatty liver and cardiovascular risk may vary after accounting for factors such as obesity, hypertension, or diabetes (6).

(d) **Elevated Triglyceride Levels**

The symptoms can be vague, and they can resemble those of a number of other problems. Tests may be carried out to eliminate other conditions like blood test (to identify specific causes of liver issues (e.g. viral hepatitis), liver imaging such as ultrasound, computerized tomography (CT) scanning, transient elastography (Fibro Scan), or magnetic resonance imaging (MRI).

While there are clear links between obesity and fatty liver, some people develop NAFLD without obesity. This suggests that there are other factors. These include genetic influences, smoking, older age, certain medications, such as steroids, and tamoxifen for cancer treatment, rapid weight loss, infections, such as hepatitis, exposure to some toxins. However, research suggests that "excess fat mass remains the most common background condition."

The scientists predict it to become the leading cause of liver pathology, liver failure and indication for liver transplantation in childhood and adolescence in the Western world within next 10 years. Around 25 percent of pediatric patients with NASH will go on to develop cirrhosis within 10 years. Among those with obesity, the risk is higher.

Despite the alarming rate of non-alcoholic liver disease, there are limitations in knowledge and unmet needs in the management of NAFLD among medical providers. In a survey conducted among primary care physicians, 58% expressed a lack of confidence in their knowledge and management of fatty liver disease (7). The challenge in diagnosing NAFLD may stem from the fact that most patients are asymptomatic and are typically only identified by routine blood tests showing elevated liver enzymes. Nevertheless, a subset of patients can have normal liver enzymes and thus remain undiagnosed.

Numerous studies have looked at the management of fatty liver disease; however, there is no consensus on the optimal management of NAFLD. Currently, there are no drug therapies approved by the Food and Drug Association (FDA) for the treatment of NAFLD (8). This article aims to review current knowledge on NAFLD, with emphasis on identifying gaps in its diagnosis and management, and proposes future directions to address these limitations.

**Materials and Methods**

In the present cross-sectional study, a total of 100 cases were included. The patients diagnosed as NAFLD on USG seeking treatment in the Department of Medicine IPD and OPD were included and investigated for metabolic syndrome according to the NCEP ATP 3 criteria. The data was collected during OPD/IPD treatment and was recorded in predesigned and pretested proforma and analyzed. Inclusion criteria were: All patients diagnosed as NAFLD by abdominal Ultrasonography, Age more than 18 years. Exclusion criteria were: Patients with a history of alcohol intake more than 30 grams/day in males and
more than 20 grams/day in females. Patients with a history of jaundice or HBsAg positive, Patients with history of following drug intake - steroids, synthetic estrogens, heparin, and calcium channel blockers, amiodarone, valproic acid, antiviral agents, Unwilling patients. Detailed history, anthropometry, and clinical examinations were carried out after taking informed consent of the patient. All patients in the study underwent routine investigations including complete blood counts, blood sugar, liver function tests, blood pressure, and lipid profile.

Results

Table 1: Showing distribution of patients according to their clinical and biochemical profiles (n=100).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age(years)</td>
<td>45.6±7.4</td>
</tr>
<tr>
<td>BMI(body mass index)</td>
<td>27.3±3.8</td>
</tr>
<tr>
<td>Blood pressure(systolic, mm Hg)</td>
<td>138.1±15.6</td>
</tr>
<tr>
<td>Blood pressure(diastolic, mm Hg)</td>
<td>80.2±8.9</td>
</tr>
<tr>
<td>Fasting blood sugar(mg/dl)</td>
<td>120.2±48.9</td>
</tr>
<tr>
<td>Waist circumference(cm)</td>
<td>90.2±9.1</td>
</tr>
<tr>
<td>Total Cholesterol(mg/dl)</td>
<td>235.3±38.2</td>
</tr>
<tr>
<td>Serum LDL(mg/dl)</td>
<td>126.2±37.3</td>
</tr>
<tr>
<td>Serum HDL(mg/dl)</td>
<td>42.6±5.9</td>
</tr>
<tr>
<td>Serum VLDL (mg/dl)</td>
<td>26.7±9.6</td>
</tr>
<tr>
<td>Serum triglycerides (mg/dl)</td>
<td>270.4±90.3</td>
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Discussion

We are experiencing a worldwide epidemic of obesity, which is associated with different co morbidities, such as non-alcoholic fatty liver disease (NAFLD), which is currently considered the most common hepatopathy worldwide.

The purpose of this study is to provide an overview of the role of different nutrients and dietary models on the development, treatment, prevention, and reversal of NAFLD. A high-calorie diet, particularly rich in saturated fatty acids and cholesterol, sugary drinks with high fructose content, increases visceral adiposity inducing the accumulation of lipids in the hepatocytes and triggers other systemic and tissue specific responses driving the progression of liver damage. These events could be exacerbated by a genetic predisposition. In this context, the reduction of caloric intake, obtained with lifestyle interventions or with dietary supplements (i.e., omega-3 fatty acids, vitamins, probiotics) may contribute to reverse metabolic derangement and liver damage occurring in NAFLD (9-11).

NAFLD has emerged as the most common cause of chronic liver disease worldwide. NAFLD can lead to hepatocellular carcinoma. NAFLD is an independent determinant of cardiovascular disease (CVD). NAFLD is therefore a complex problem with complications far beyond the liver. Diabetes is not only associated with NAFLD but may also be a risk factor for development of progressive fibrosis (12-14).

A total of 100 cases diagnosed ultrasonographically as NAFLD were included in the study. In the present study, it was observed that mean age of the patient was 45.6±7.4years. On physical examination mean BMI was 27.3±3.8 kg/m² while mean waist circumference was 90.2 ±9.1cm. Mean Diastolic blood pressure (mm of Hg) was 80.2±8.9and mean Systolic blood pressure (mm Hg) 138.115.6. These results are consistent with studies by Rakesh Gaharwar et al and Animesh Deb et al. (15,16) The mean Fasting blood sugar (mg/dl) was 120.2±48.9and mean total cholesterol (mg/dl) was 235.3±38.2while mean Serum triglycerides (mg/dl) were 270.4±90.3 .These findings are similar to study by Shivaram Prasad Singh et al and Kwon YM et al.(17,18) The study shows that 53% patients had
fasting plasma glucose >100 mg/dl, while 38% patients were hypertensive similar to studies by Rakesh Gaharwar et al and Animesh Deb et al.(15,16) Maximum 70% patients had Triglycerides >150 mg/dl while low Serum HDL level was seen in 51% patients and increased waist circumference was found in 44% patients which were also observed by Yang KC et al and the difference was statistically significant (19). In the present study, it was observed that mean Fasting plasma glucose was 120.2±48.9 mg/dl.

**Tips for Patients with Fatty Liver:**

Increased physical activity is a key component of managing NAFLD. Patients should do thirty minutes of moderate exercise five times a week and aim to lose between 1 and 1.6kg per week.

Dietary alteration also plays an essential role in NAFLD treatment. Patients should take 600 calories less than their daily caloric requirement and reduce their intake of sugar – including fructose, or fruit sugar – as well as saturated and trans fats, such as those found in fast food. Patients are advised to increase their intake of polyunsaturated fat, especially omega-3 fatty acids. This can be achieved by consuming more fish and high-fibre food, as well as more soy protein and whey. Choline, fibre, coffee and green tea can also help stave off liver damage.

NAFLD patients with obesity should take steps to alleviate metabolic risk factors. On the other hand, patients with steatohepatitis and fibrosis are at highest risks of developing the progressive liver disease.

Treatment could be Medications have a mild benefit. Lifestyle modification including weight loss, dietary changes and physical exercise is first-line treatment. Treatments of any associated metabolic disease are important: e.g. control of diabetes, hypertension and dyslipidaemia.

NASH can be associated with progressive liver damage. Even simple steatosis, or excess liver fats, can escalate to fibro-cirrhotic disease.

Cirrhosis can be complicated by end-stage liver disease (liver failure) and hepatocellular carcinoma (HCC). NAFLD/NASH-related cirrhosis is one of the most common indications for liver transplantation candidates in developed countries.

Liver transplantation is the most effective treatment for the end-stage liver disease and improves recipients’ survival and quality of life.

**Conclusion**

From our study, it can be concluded that symptoms and signs of NAFLD are non-specific and occur later in the course of the disease hence the physician should have a high index of suspicion in order to detect NAFLD early in the course of the disease. Prognosis depends on the extent of liver damage. Steatosis alone is not fatal, but asymptomatic NASH can progress to liver cirrhosis. Cardiovascular disease is the leading cause of illness and death in NAFLD patients, who should minimize their overall risk by giving up smoking and reducing alcohol consumption. Higher prevalence of all the components of metabolic syndrome in cases of NAFLD was observed. Liver biopsy is considered the gold standard for diagnosing NAFLD but is not practical and most patients are not willing to undergo the test. Thus, patients must be evaluated for the presence of NAFLD by abdominal Ultrasonography and blood test. Early detection would help in modifying the disease course and delaying its complications.

In general, patients with NAFLD should Get regular check-ups from a doctor specialising in liver care, Maintain a healthy diet and regular exercise to attain a normal BMI, Lower triglyceride and cholesterol levels through diet management and/or medication, Control any diabetes via diet management, insulin or medication, Eliminate or reduce alcohol consumption.

**References**


