Original Research Article

A study on fatty liver in patients admitted to medical wards in tertiary hospital

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ABSTRACT

Background: Fatty liver is a common clinical condition seen in medical wards. It can be either alcoholic fatty liver disease or non-alcoholic fatty liver disease. It is an iceberg phenomenon which can be associated with metabolic syndrome, cardiovascular disease and hepatocellular carcinoma. The aim of the present study is to observe and analyse the prevalence of fatty liver diseases in general population admitted to general wards in department of general medicine and to help in the timely prevention of associated risk factors and complications

Methods: It is a prospective cross-sectional study conducted in Narayana medical college and hospital a tertiary care hospital for a period of 6 months from November 2017 to May 2018 and includes a total of 250 patients. Patients were routinely screened with ultrasound abdomen and those having fatty liver changes were included in this study and their clinical and biochemical parameters were examined.

Results: Out of the 250 patients admitted to medical wards during the study period, 70 patients (28%) had fatty liver. Out of these, 23 (34%) were males and 47 (67%) were females, alcoholics were 9 (12.8%) and non-alcoholics were (87.2%), 35 (50%) were diabetics, 11 (15.7%) had CAD (coronary artery disease), 29 (41.4%) were obese with BMI >30 and 26 (37.1%) were hypertensive.

Conclusions: Fatty liver is a common incidental finding in ultrasound abdomen in patients admitted to medical wards and when neglected, which when given importance and with proper intervention and life style modifications can prevent metabolic syndrome, cardiovascular disease and hepatocellular carcinoma.

Keywords: Alcoholic fatty liver disease, Cardiovascular disease, Hepatocellular carcinoma, Metabolic syndrome, Non-alcoholic fatty liver disease

INTRODUCTION

Fatty liver disease is a common condition seen in medical wards. Term fatty liver means build-up of fat in liver. It can be either alcoholic fatty liver disease or non-alcoholic fatty liver disease. History of alcohol intake differentiates between alcoholic fatty liver disease and non-alcoholic fatty liver disease. Other causes of fatty liver are hepatitis c infection, inborn errors of metabolism, Reyes syndrome, acute fatty liver of pregnancy, after surgical procedures like biliopancreatic diversion, extensive small bowel resection, gastric bypass and drugs like L- asparaginase, methotrexate, amiodarone, glucocorticoids, estrogens, tamoxifen, tetracycline.

The amount of fatty acid in the liver depends on the balance between the process of delivery and removal. In some patients, fatty liver may be accompanied by hepatic inflammation and liver cell death (steatohepatitis). Hepatic steatosis describes the accumulation of fat, mostly as triglycerides, cholesterol and phospholipids,
excess of 5-10% of liver weight.\textsuperscript{1} Alcoholic liver disease (ALD) can be divided into the following 3 groups:

- Alcoholic fatty liver (simple steatosis)
- Alcoholic hepatitis
- Alcohol-related cirrhosis

Alcohol is the world’s third largest risk factor for disease burden. Quantity and duration of alcohol intake are the most important risk factors involved in the development of alcoholic liver disease. Alcoholic fatty liver is an early and reversible consequence of excessive alcohol consumption. Fatty liver develops in every individual who consumes more than 60g of alcohol per day.\textsuperscript{2} The accumulation of fat within perivenular hepatocytes coincides with location of alcohol dehydrogenase, the major enzyme responsible for alcohol metabolism. The hallmark of alcoholic hepatitis is hepatocyte injury characterized by ballooning degeneration, spotty necrosis, polymorphonuclear infiltrate and fibrosis in periventricular and perisinusoidal space of disease.

Ingestion of alcohol initiates an inflammatory cascade by its metabolism to acetaldehyde. Steatosis from lipogenesis, fatty acid synthesis, and decreased fatty acid oxidation appears secondary to effects on sterol regulatory transcription factor and peroxisome proliferator activated receptor alpha. Intestinal derived endotoxin initiates a pathogenic process through toll like receptor 4 and tumor necrosis factor alpha that facilitates hepatocyte apoptosis and necrosis. Hepatocyte injury and impaired regeneration following chronic alcohol ingestion are ultimately associated with stellate cell activation and collagen production, which are the key events in fibrogenesis.

Over-expression of lipin-1 could remarkably suppress very low-density lipoprotein -triacylglyceride (VLDL-TAG) secretion which has a potent anti-inflammatory property. Increased synthesis of TAG, decreased fatty acid oxidation, impaired VLDL-TAG secretion and activated inflammatory factors act together to exacerbate the development of AFLD.\textsuperscript{3} Hepatic deletion of SIRT1 promotes steatosis, inflammation, and fibrosis in response to ethanol challenge. Ethanol-mediated impairment of hepatic SIRT1 signaling via lipin-1 contributes to development of alcoholic steatosis and inflammation. Reagents designed to increase SIRT1 regulation of lipin-1 can be developed to treat patients with alcoholic fatty liver disease.\textsuperscript{4} AST and ALT are elevated but AST will be more elevated than ALT. Complete abstinence of alcohol is the corner stone in treatment of alcoholic liver disease.

NAFLD is the most common chronic liver disease. The spectrum of NAFLD ranges from simple steatosis without evidence of cell injury, which tends to be stable over time, to steatohepatitis progressing to cirrhosis.\textsuperscript{5} The mechanisms underlying gut microbiota-mediated development of NAFLD include modulation of host energy metabolism, insulin sensitivity, and bile acid and choline metabolism.\textsuperscript{6} Obese adipose depots also produce excessive soluble adipokines that inhibit tissue insulin sensitivity. Thus, hyperinsulinemia occurs and lipid uptake, fat synthesis and storage results in hypertriglyceridemia. Its precursors fatty acids, diacylglycerol and reactive oxygen species damage hepatocytes and its regeneration causes fibrosis. NAFLD is related to the metabolic syndrome and insulin-resistant states (e.g. diabetes mellitus type 2) and cardiovascular disease.\textsuperscript{7,8} NAFLD causes endothelial dysfunction and causes increase in carotid intimal thickness and increases number of plaques in carotid and coronary arteries. Up to 80% of obese people have this disease.\textsuperscript{9} NAFLD is a premalignant condition. Once cirrhosis develops due to NAFLD, liver cancer risk is 1%. Patients will be usually asymptomatic but may have vague abdominal pain. Liver enzymes will be elevated.

A low normal ALT value does not guarantee freedom from underlying steatohepatitis with advanced fibrosis.\textsuperscript{10} Effective risk stratification and management of NAFLD requires evaluation of hepatic parenchymal fat, fibrosis, and inflammation. Liver biopsy remains the current gold standard; however, non-invasive imaging methods are rapidly evolving and may replace biopsy in some circumstances. These methods include well-established techniques, such as conventional ultrasonography, computed tomography, and magnetic resonance imaging and newer imaging technologies, such as ultrasound elastography, quantitative ultrasound techniques, magnetic resonance elastography, and magnetic resonance-based fat quantitation techniques.\textsuperscript{11} Proton magnetic resonance spectroscopy has appropriate sensitivity for quantification of the intrahepatic lipid content. But liver biopsy should be done to confirm the diagnosis.\textsuperscript{12,13} Pharmacological intervention with orlistat, topiramate, phentermine, metformin, thiazolidinediones, vitamin D, vitamin E, omega 3 fatty acids and statins are tried.\textsuperscript{14} If there is no response to this treatment, bariatric surgery and liver transplantation are done.\textsuperscript{15}

\textbf{METHODS}

This study is a prospective cross-sectional study which was conducted in Narayana medical college and hospital, a tertiary care hospital, from November 2017 to May 2018 for a period of six months and included a total of 250 patients. History was collected from all the patients admitted to general wards in the Department of General Medicine and these patients were examined and screened for fatty liver by diagnostic ultrasonography method. Those who were found to have fatty liver were included in this study and their biochemical parameters like Fasting lipid profile were done.

\textbf{Inclusion criteria}

All patients in the age group 18-65 years who are admitted to medical wards.
**Exclusion criteria**

- Patients less than 18 years of age and more than 65 years of age.
- Patients with inborn errors of metabolism.
- Patients who are pregnant.
- Patients who are on drugs like tamoxifen, methotrexate, estrogens, corticosteroids, tetracyclines.

**RESULTS**

Out of the 250 patients admitted to wards during the period between November 2017 and May 2018, there were 70 patients with fatty liver i.e., 28% of the total patients.

Out of the 70 patients with fatty liver, 23 were males i.e., 33% and 46 are females i.e., 67% (Table 1).

<table>
<thead>
<tr>
<th>Table 1: Sex distribution of fatty liver.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>Female</td>
</tr>
</tbody>
</table>

Out of the 250 patients admitted to wards -51 patients were in the age group of 20-40 years, out of which 9 were having fatty liver; out of the 150 patients in age group 41-60 years, 44 were having fatty liver; out of the 40 patients in age group 61-80 years, 16 were having fatty liver; out of the 9 patients in age group >80 years, 1 was having fatty liver (Table 2).

<table>
<thead>
<tr>
<th>Table 2: Age distribution of fatty liver.</th>
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</thead>
<tbody>
<tr>
<td>Age groups</td>
</tr>
<tr>
<td>20-40 yrs</td>
</tr>
<tr>
<td>41-60 yrs</td>
</tr>
<tr>
<td>61-80 yrs</td>
</tr>
<tr>
<td>&gt;80 yrs</td>
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</tbody>
</table>

Out of the 250 patients admitted to wards; 70 patients were having fatty liver, out of which 9 patients have history of alcohol consumption i.e., 12.85% and 61 patients do not have history of alcohol consumption i.e., 87.15% (Table 3).

<table>
<thead>
<tr>
<th>Table 3: Fatty liver and alcohol consumption.</th>
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</thead>
<tbody>
<tr>
<td>Alcohol consumption</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>No</td>
</tr>
</tbody>
</table>

Out of the 70 patients with fatty liver on usg abdomen, 35 patients had history of diabetes, 26 had history of hypertension, 11 had coronary artery disease, 6 had chronic kidney disease and 12 were smokers (Table 4).

**Table 4: Fatty liver and comorbidities.**

<table>
<thead>
<tr>
<th>Associated factors</th>
<th>Fatty liver</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>35</td>
</tr>
<tr>
<td>Hypertension</td>
<td>26</td>
</tr>
<tr>
<td>CAD</td>
<td>11</td>
</tr>
<tr>
<td>CKD</td>
<td>6</td>
</tr>
<tr>
<td>Smoking</td>
<td>12</td>
</tr>
</tbody>
</table>

Out of the 70 patients with fatty liver, 48 were having dyslipidemia; 29 patients were obese (Table 5).

**DISCUSSION**

Over the past many years, patients admitted to authors’ hospital with cardiovascular diseases, diabetes, hypertension and obesity already had fatty liver in their previous ultrasound abdomen reports which is neglected. Fatty liver is an iceberg phenomenon which can be associated with metabolic syndrome, cardiovascular diseases and hepatocellular carcinoma. Proper intervention and life style modification can prevent metabolic syndrome and cardiovascular disease.

The prevalence of fatty liver among patients in present study group is 28%; which is comparable to the study by Bellentani S et al, (25-35%).

The sex demographics involving more females in our study correlate with the study by Duseja A et al, which indicates that females were significantly overrepresented compared to males.

The reason for this appears to be the incidence of more NAFLD cases in the female sex because most of the female patients present very late to the hospital by the time they were diagnosed and restraining themselves to the household, making them less prone to physical activity.

The age level prevalence is present in all age groups but more in the age group of 40-80 years. Among fatty liver disease patients, alcoholic liver disease accounted for 12.85% and non-alcoholic liver disease accounted for 87.15% which suggests that non-alcoholic fatty liver is more common and leads to metabolic syndrome and cardiovascular diseases.
Diabetes is the most common comorbidity followed by hypertension which suggests that patients with diabetes have more propensity to develop non-alcoholic fatty liver disease. Smoking and coronary artery disease are associated with non-alcoholic fatty liver disease.

Dyslipidemia is seen in 68.5% of fatty liver disease patients. Obesity is seen in 41.4% of fatty liver disease patients. Liver enzymes (SGOT and SGPT) were elevated in 20% of patients. Patients with fatty liver disease showed a markedly higher risk of developing liver-related death compared to the general population. The AFLD group had higher liver-related mortality and had a worse survival than the NAFLD group. Patients with more severe fibrosis at baseline showed a worse survival than patients with none or mild fibrosis at baseline.18

Present study analysed that majority of patients with fatty liver when detected by simple ultrasound abdomen followed necessary health education and lifestyle modifications, can prevent development of metabolic syndrome, hepatocellular carcinoma and death as most of them belong to low socioeconomic status with limited means to afford high priced diagnostic modalities and treatment.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES


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