STUDY OF NON-ALCOHOLIC FATTY LIVER DISEASE IN PATIENTS OF HYPOTHYROIDISM IN WESTERN UTTAR PRADESH.

Abstract

Background: Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as the hepatic manifestation of metabolic dysfunction. Hypothyroidism contributes to dyslipidaemia, insulin resistance and altered lipid metabolism, potentially increasing NAFLD risk. This study evaluated the prevalence of NAFLD among patients with hypothyroidism and explored associated clinical and biochemical factors.

Methods: A hospital-based cross-sectional study was conducted at Muzaffarnagar Medical College. Adult patients with hypothyroidism underwent clinical assessment, thyroid function testing, liver function testing, lipid profile analysis and ultrasonography of the liver. Demographic variables, metabolic risk factors and biochemical parameters were analysed in relation to ultrasonographic evidence of NAFLD.

Results: NAFLD was frequently identified among hypothyroid patients and was associated with higher thyroid stimulating hormone levels, adverse lipid profiles and elevated liver enzymes. Increasing severity of thyroid dysfunction showed a positive relationship with hepatic steatosis. Metabolic risk factors including obesity, dyslipidaemia and associated comorbidities further increased the likelihood of NAFLD.

Conclusion: Hypothyroidism is strongly associated with NAFLD. Early screening of hypothyroid patients using liver ultrasonography and metabolic assessment may facilitate timely intervention and reduce long-term hepatic and cardiovascular complications.

Keywords: Hypothyroidism, Non-alcoholic fatty liver disease, NAFLD, Thyroid stimulating hormone, Dyslipidaemia, Ultrasonography.

Introduction

Non-alcoholic fatty liver disease represents a spectrum of hepatic disorders ranging from simple steatosis to non-alcoholic steatohepatitis, fibrosis, cirrhosis and hepatocellular carcinoma. The condition has emerged as one of the most common chronic liver diseases worldwide and parallels the increasing prevalence of obesity, diabetes mellitus and metabolic syndrome. Recent evidence suggests that endocrine disorders, particularly hypothyroidism, may play an important role in NAFLD pathogenesis.

Thyroid hormones regulate basal metabolic rate, lipid handling, glucose homeostasis and energy expenditure. Reduced thyroid hormone activity promotes weight gain, insulin resistance and dyslipidaemia, factors closely linked to hepatic fat accumulation.

Experimental and clinical studies demonstrate that impaired thyroid hormone signalling within the liver contributes to altered lipid metabolism and steatosis. Consequently, hypothyroidism has gained attention as a potentially modifiable risk factor for NAFLD.

India carries a substantial burden of both thyroid disorders and metabolic disease. Despite increasing recognition of the association between hypothyroidism and NAFLD, regional data remain limited. Understanding this relationship is important because early identification of hepatic involvement may improve clinical outcomes and reduce future complications. The present study therefore evaluated the prevalence of NAFLD among patients with hypothyroidism and examined demographic, biochemical and metabolic correlates associated with hepatic steatosis.

Materials and Methods

This observational cross-sectional study was conducted in the Department of General Medicine, Muzaffarnagar Medical College. Adult patients diagnosed with hypothyroidism were included after informed consent. Clinical history, demographic information and relevant comorbidities were recorded using a structured proforma.

All participants underwent thyroid function testing, liver function testing and lipid profile analysis. Thyroid status was assessed using serum thyroid stimulating hormone and thyroid hormone levels. Liver function parameters included serum transaminases and other standard biochemical markers. Lipid profile assessment included total cholesterol and triglyceride levels.

Abdominal ultrasonography was performed to identify fatty liver changes. Patients were categorised according to ultrasonographic findings. Statistical analysis was undertaken to determine associations between NAFLD and thyroid function, biochemical parameters, demographic characteristics and risk factors. A p value below 0.05 was considered statistically significant.

Results

The study population consisted of adult hypothyroid patients representing a broad age distribution and both genders. Ultrasonography identified a substantial burden of fatty liver disease among participants. NAFLD was more frequently observed among patients with higher thyroid stimulating hormone concentrations, suggesting a relationship between worsening thyroid dysfunction and hepatic steatosis.

Biochemical analysis demonstrated that patients with NAFLD generally exhibited less favourable lipid profiles. Triglyceride and cholesterol values tended to be higher among those with ultrasonographic evidence of fatty liver. Elevated liver enzymes were also more common, supporting the presence of metabolic and hepatic dysfunction.

Age, gender distribution and place of residence were analysed in relation to ultrasonographic findings. Although demographic characteristics contributed to variability

in prevalence, metabolic abnormalities appeared to exert a stronger influence on the occurrence of NAFLD. Individuals with obesity, dyslipidaemia and related metabolic risk factors showed a greater likelihood of fatty liver changes.

The findings reinforce the concept that hypothyroidism and NAFLD share overlapping pathophysiological mechanisms. Progressive thyroid dysfunction was associated with biochemical abnormalities that favour hepatic lipid accumulation and contribute to steatosis development.

Discussion

The current study highlights a clinically important association between hypothyroidism and NAFLD. Thyroid hormones play a central role in lipid metabolism through regulation of cholesterol synthesis, fatty acid oxidation and hepatic energy balance. Reduced thyroid hormone activity promotes lipid accumulation within hepatocytes, creating a favourable environment for fatty liver development.

Several previous studies have reported similar findings. Observational studies and meta-analyses have demonstrated a significant association between overt hypothyroidism and NAFLD. More recent investigations have suggested that even subclinical hypothyroidism may contribute to hepatic steatosis. The present findings are therefore consistent with growing international evidence supporting the role of thyroid dysfunction in metabolic liver disease.

One plausible mechanism linking hypothyroidism and NAFLD is dyslipidaemia. Hypothyroid patients frequently exhibit elevated cholesterol and triglyceride concentrations due to reduced lipid clearance and altered hepatic metabolism. These changes increase the delivery of fatty acids to the liver and facilitate triglyceride accumulation within hepatocytes. Insulin resistance, another common feature of hypothyroidism, further contributes to hepatic steatosis through increased lipolysis and altered glucose metabolism.

The relationship between thyroid dysfunction and NAFLD may also involve direct hepatic effects. Thyroid hormone receptor-beta signalling is essential for normal hepatic lipid regulation. Impaired receptor activity or reduced intrahepatic thyroid hormone availability may produce a state of functional hepatic hypothyroidism. This phenomenon has been increasingly recognised in contemporary studies and may explain why thyroid dysfunction remains associated with NAFLD even after adjustment for conventional metabolic risk factors.

The present study additionally demonstrated associations between fatty liver and adverse biochemical markers. Higher liver enzyme levels among patients with NAFLD suggest ongoing hepatocellular injury. Elevated triglycerides and cholesterol further indicate a metabolic milieu favourable to disease progression. These observations support the concept

that NAFLD should be viewed not merely as a hepatic disorder but as a manifestation of systemic metabolic dysfunction.

From a clinical perspective, the findings emphasise the need for heightened vigilance in patients with hypothyroidism. Many individuals remain asymptomatic during early stages of NAFLD, resulting in delayed diagnosis. Routine assessment of metabolic risk factors, liver enzymes and ultrasonography may facilitate earlier detection. Such an approach is particularly relevant in resource-limited settings where advanced imaging or liver biopsy may not be feasible.

The study has important public health implications. India is experiencing a rapid rise in obesity, diabetes and thyroid disorders. As these conditions frequently coexist, the burden of NAFLD is likely to increase substantially. Integrating liver health assessment into the routine evaluation of hypothyroid patients may represent a practical strategy for identifying high-risk individuals and preventing long-term complications.

Certain limitations should be acknowledged. The cross-sectional design precludes definitive conclusions regarding causality. Ultrasonography, although widely available and non-invasive, may underestimate mild steatosis compared with histological assessment. Future multicentre prospective studies incorporating advanced imaging modalities and long-term follow-up are warranted to clarify causal pathways and evaluate the impact of thyroid hormone replacement on NAFLD outcomes.

Despite these limitations, the study contributes valuable data from a North Indian population and supports existing evidence linking hypothyroidism with NAFLD. Recognition of this association may improve screening strategies and encourage a more comprehensive metabolic evaluation of patients with thyroid dysfunction.

Conclusion

NAFLD is common among patients with hypothyroidism and is associated with adverse metabolic and biochemical profiles. Higher thyroid stimulating hormone levels, dyslipidaemia and elevated liver enzymes appear to correlate with hepatic steatosis. Routine screening for NAFLD in hypothyroid individuals may permit earlier diagnosis, risk stratification and targeted intervention. A multidisciplinary approach addressing thyroid dysfunction, weight management and metabolic risk factors is likely to provide the greatest clinical benefit.