

Effects of Thyroid Autoimmunity on Non-Alcoholic Fatty Liver Disease in Euthyroid Girls with Hashimoto's Thyroiditis

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has recently become the most common cause of chronic liver disease in children and adolescents. While often benign and self-limiting, steatosis (fatty liver) can progress with hepatocyte injury into non-alcoholic steatohepatitis (NASH) in 3%-5% of patients. NAFLD is closely associated with traits of metabolic syndrome (MS) such as obesity, hypertension, dyslipidemia and insulin resistance. For this reason NAFLD is considered to be the liver presentation of the MS. Although the pathogenesis of the disease is still unclear, it is widely accepted that multiple factors including ethnic, environmental, metabolic and stress-related factors influence the development and progression of the disease.

Hypothyroidism has also been identified as a risk factor for the development of NAFLD, because of thyroid dysfunction could lead to obesity, dyslipidemia, insulin resistance and lipid peroxidation, all of which are related to NAFLD. Previous studies have reported higher prevalence of NAFLD among patients with overt and even subclinical hypothyroidism.

AIM

The aim of this study was to investigate whether autoimmune Hashimoto's thyroiditis (HT) increases the incidence of NAFLD, regardless of thyroid dysfunction. In addition, the relationship between autoimmunity and the following factors was evaluated: Body Mass Index (BMI), body parameters measured by Bioelectric Impedance Analysis (BIA), and metabolic syndrome parameters.

METHODS

43 newly diagnosed euthyroid girls with HT with a mean age of 14.4 ± 2.1 years were included in the study. The control group consisted of 41 age- and BMI-matched healthy girls. At enrollment, all subjects underwent physical examination including blood pressure, standing height, weight, waist circumference (WC), and hip circumference measurements. The lipid profile, liver function tests, blood glucose, insulin, high sensitivity C-reactive protein (hs-CRP), thyroid functions, and thyroid antibodies were measured. Thyroid and liver ultrasonography (US) were performed and body parameters were measured by BIA.

RESULTS

In our study, Grade 1 steatosis was detected by liver US in 3 patients (7%) in the HT group while the control group was completely normal. There was no significant difference between the two groups in terms of NAFLD ($p = 0.085$). There was no significant difference between the two groups in terms of anthropometric variables except for systolic and diastolic blood pressures, which were significantly higher in patients with HT even though they were still within the normal range. The median thyroid stimulating hormone (TSH) value of the patient group was higher [$2.88 (0.43-5.57) \mu\text{LU/mL}$] than the control group [$1.98 (0.96-4.24) \mu\text{LU/mL}$] ($p = 0.017$). However, once again, these higher values were still within the normal range. There was no statistically significant difference in metabolic parameters (ALT, AST, GGT, cholesterol, triglyceride, glucose, insulin and HOMA-IR) between the two groups. When we compared the BIA parameters between patient and control groups, there was no statistically significant difference ($p > 0.05$).

A multivariate logistic regression analysis did not find that the independent variables BMI-SDS, age, waist circumference, hip circumference, TSH, Anti-TPO, anti-Tg antibodies, and systolic blood pressure affect the presence of NAFLD.

CONCLUSION

In conclusion, our study revealed that HT patients had increased NAFLD compared to the control group, but this difference was not statistically significant. The two groups were considerably homogeneous in terms of thyroid function, metabolic risk factors, and anthropometric variables except for systolic and diastolic blood pressures, which were significantly higher in patients with HT patients. These observations suggest an atherogenic role of thyroid antibodies. As thyroid autoimmunity increases atherosclerosis via an inflammatory mechanism, it could also have a role in NAFLD development in a similar manner. Since the identification of risk factors is imperative for the prevention of NAFLD development, further studies with larger cohorts are needed.

