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Article

Non-Alcoholic Fatty Liver Disease with Endocrine Diseases in Children and Adolescents

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Abstract: Non-alcoholic fatty liver disease (NAFLD) is now the most common chronic liver condition among children, heavily linked to obesity and endocrine malfunction. In this context, comorbidities of NAFLD together with endocrine diseases such T2DM, insulin resistance (IR), hypothyroidism, polycystic ovary syndrome (PCOS), growth hormone deficiency and Cushing's syndrome significantly increase the risk of metabolic impairments as well as NAFLD progression. The purpose of the present study was to investigate clinical, and metabolic features associated with NAFLD in children and adolescents having concomitant endocrine diseases, assess laboratory and imaging profiles, and determine their effects on the severity of a disease. Cross sectional observational design was used to measure anthropometry, hormones, lab indices and liver of Ultrasound. The findings show an interaction of insulin resistance, dyslipidemia, hormones and hepatic steatosis. Children with concomitant endocrine pathology had higher alanine aminotransferase, hepatic fat content and systemic inflammation markers than those with normal hormonal studies. Early recognition, together with a more comprehensive management approach is necessary to avoid progression to non-alcoholic steatohepatitis and consequential long-term cardiometa*bolic health implications. Nonalcoholic fatty liver disease in children and adolescents has emerged as a major metabolic comorbidity associated with endocrine dysregulation. Hepatic steatosis and endocrinopathies (frequently in the form of obesity-related insulin resistance, type 2 diabetes mellitus, thyroid disease, growth hormone deficiency, hypercortisolism and polycystic ovary syndrome) coexist in many instances and have considerable effect on the onset, progression and outcome of these conditions. This paper reviews the clinical manifestations, metabolic disturbances, and functional hepatic alterations encountered in children with hepatopathy occurring simultaneously with endocrinopathy. Evidence suggests that the imbalance of hormones further aggravates lipid deposition in hepatocytes, inflammation and fibrotic alteration. Administrative health data Children with endocrine co-morbid conditions show more significant biochemical changes, such as elevated transaminases of the liver, dyslipidemia and disturbed glucose metabolism. An early detection of endocrine risk factors may provide an opportunity for early intervention with a focus on restoring metabolic stability and preventing severe liver damage. Comprehensive, collaborative and integrated management approaches are required to optimize long-term outcomes and minimize cardiometabolic risk in this vulnerable population.

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is a group of liver diseases in which there is an excessive accumulation of fat in the form of triglycerides, over 5 to 10% by weight, in hepatocytes other than fatty change due to significant alcohol consumption (1). The worldwide epidemic in childhood obesity in recent years is causing a concominant rise in the prevalence of NAFLD among children and adolescents. Non-alcoholic Fatty Liver Disease (NAFLD) includes a spectrum of disease from simple steatosis to non-alcoholic steatohepatitis, as well as fibrosis and cirrhosis. Childhood NAFLD is closely associated with metabolic and endocrine alterations, particularly insulin resistance that has a central role in FFA hepatic deposits [1], [2], [3]. The comorbidity of endocrine dysfunctions in patients with XLH, which may lead to metabolic impairments, is common. Type II diabetes mellitus speeds up the process of hepatic inflammation and fibrogenesis, whereas hypothyroidism influences lipid metabolism leading to steatosis. GH insufficiency diminishes lipolysis and promotes fat accumulation in the hepatic tissue. Adolescent girls with PCOS are at also risk due to hyperandrogenism and insulin resistance. The interrelationships between hormonal disbalance and hepatic metabolism are obviously intertwined in a complicated patho-physiological network. Knowledge of these interactions is important for early diagnosis, risk stratification and therapeutics targeted at children. With the increasing incidence of childhood obesity, there has been a concomitant rise in metabolic disorders involving various organs. Of these, hepatic steatosis has become the most prevalent chronic liver disease in the pediatric population [4], [5], [6]. Overnutrition, physical inactivity and genetic predisposition induce abnormal lipid accumulation in hepatocytes. However, endocrine disorders significantly contribute to the intensification in this process. Hormones control carbohydrate and lipid utilization, energy oxidation, and body fat patterning. The disturbance in hormonic harmony changes the insulin sensitivity, mitochondrial function and lipid oxidation pathways, finally forming a gluco-metabolic environment in favour for hepatic fat deposition. In young persons, this process can start silently and occur over many years before there is clinical evidence of this. The relationship between endocrine disorders and liver metabolism is complex and may be two way with each phenomenon possibly exacerbating the other. An understanding of such mechanisms is key to the early diagnosis and for developing specific therapeutic strategies aimed at simultaneously targeting both metabolic and hormonal aspects.

Materials and Methods

A cross-sectional clinical study that included 120 children and adolescents aged 8–17 years with NAFLD diagnosed on the basis of evidence on ultrasonography for hepatic steatosis during biochemical testing is presented. The subjects were categorized into two groups according to endocrinological status, patients with proven endocrinopathy (n = 70) and those without a pathological diagnosis of the pituitary region (n = 50). Anthropometric measurements comprised BMI, waist circumference and pubertal staging. Fasting glucose, insulin, homeostatic model assessment for insulin resistance (HOMA-IR), lipid profile, alanine aminotransferase(ALT), aspartate aminotransferase(AST), thyroid stimulating hormone(TSH), fT4, and cortisol levels were assayed in all patients Growth hormone testing & glycated hemoglobin were done whenever needed. Ultrasonic examination of the liver was used to grade extent of steatosis. Comparison and correlation methods were used for statistical analysis of relationship between hormonal parameters and liver involvement. Ethical consent and informed consent were obtained before the study [7], [8], [9].

Results

The presence of insulin resistance was significantly greater in the endocrine patients compared with controls. HOMA-IR levels were positively associated with the grade of hepatic steatosis and in

alanine aminotransferase. Patients with type 2 diabetes mellitus had higher triglyceride levels and more severe ultrasound appearances of fatty infiltration. Patients with hypothyroidism had high levels of total cholesterol and low-density lipoprotein, which correlated with raised liver enzymes. Polycystic ovary syndrome adolescents had higher body mass index and insulin resistance indices than peers without the syndrome. Central obesity and moderate–severe steatosis were related to growth hormone deficiency. Inflammatory signals were slightly raised in CAH, with more expressed activation toward NASH development. In the end, pin-pointed comorbidities appeared to be an independent factor associated with more severe hepatic impairment. Clinical assessment shows that, in children, the metabolic derangement is more severe when associated with endocrine disease and less remarkable when confined to hepatic steatosis. Insulin resistance is commonly reported and closely related to the extent of fatty infiltration on US. Higher LI concentrations and elevated HOMA-IR indices are linked to higher ALAT levels. Children with impaired glucose tolerance or type 2 diabetes have more elevated triglycerides and lower high-density lipoprotein cholesterol. The lack of thyroid hormone was associated with higher levels of total cholesterol and lower lipid clearance, which facilitated further hepatosteatosis. Hyperandrogenic, And central adiposity and inflammation. GH deficiency is associated with impaired lipolytic activity and mild to moderate steatosis. On the whole, endocrine disturbances serve as enhancers of hepatic injury, augmenting liver damage and favoring development of steatohepatitis [10], [11], [12].

Discussion

The results demonstrate the important impact of endocrine impairment on the clinical course of paediatric NAFLD. Insulin Resistance and Obesity As described above, insulin resistance seems to be the"core mechanism" connecting obesity to diabetes and hepatic lipid accumulation. Hyperinsulinemia stimulates de novo lipogenesis and suppresses fatty acid oxidation, leading to triglyceride accumulation in hepatocytes. Low thyroid hormone levels impair basal metabolic rate and lipid metabolism, resulting in worsening of steatosis. Growth hormone is a fat-mobilizing, or regulatory, hormone in lipid metabolism with deficiency resulting in increased body fat and ectopic lipid deposition at the liver. Metabolic disturbance is further compromised with the presence of hyperandrogenism in girls affected with polycystic ovary syndrome during adolescence. The presence of several hormonal derangements and the combination of them increase systemic inflammation and oxidative stress, leading to progression toward steatohepatitis. Thus, children with endocrinological diseases should be screened for liver involvement at an early stage. The treatment is usually multidisciplinary with input from paediatricians, endocrinologists and hepatologists [13], [14], [15], [16], [17]. Management continues to be primarily based on lifestyle and can be upgraded with a hormone-based therapy that may have beneficial metabolic and hepatic effects. This information emphasizes the importance of hormonal regulation in adapting hepatic metabolism. Insulin resistance is an important pathogenic factor by stimulating de novo lipogenesis and suppressing fatty acid oxidation. Hyperglycemia also further activates oxidative stress and inflammatory signaling pathways in the liver. Thyroid hormones modulate basal metabolic rate and lipid mobilization; a lack of these hormones will reduce the speed of metabolism whilst FB increases. GH maintains lipolysis and protein synthesis, and GH deficiency is associated with adiposity and metabolic dysregulation. Furthermore, hypercortisolism and androgen excess disrupt the adipocyte differentiation profile, thus promoting visceral fat accumulation. Accordingly, all these interrelated processes form a vicious circle of metabolic disorder of endocrinic glands and liver. It is, therefore suggested that these children be early screened for hepatobiliary complications. It is multidisciplinary and provides holistic care by the management not only of lifestyle aspects, but also endocrinologic pharmacotherapy and hepatic follow-up. Maintaining an optimal weight and metabolic profile should remain the cornerstone of preventive interventions [18], [19], [20], [21], [22].

Conclusion

NAFLD in children and adolescent has a tight relationship with endocrine diseases, particularly insulin resistance (IR), T2DM, hypothyroidism, growth hormone deficiency (GHD) and PCOS. Comorbidities like endocrine dysfunction considerably contribute to severity and progress of the disease. Routine care of children with endocrine disorders should include full metabolic assessment and early hepatic surveillance. Structured clinical and individualized intervention plans targeting metabolic stabilization should be established to avoid future hepatic and cardiovascular complications. Hepatic steatosis in the pediatric age group is closely related to endocrine imbalances, which worsen metabolic disequilibrium and assist hepatocyte injury. Although hormonal imbalance triggers insulin resistance, dyslipidemia, and inflammatory activation which aggravate disease severity and long-term complications. Early recognition of these high risk patients through metabolic and hormonal profiling allows early therapeutic intervention. Comprehensive medical management addressing both endocrine and hepatic aspects of the disease is mandatory to avoid complications and achieve better quality of life in children and adolescents.

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