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ORIGINAL ARTICLE

Changes in the Endocrine System of Children Born to Mothers with Thyroid Diseases

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Abstract

Maternal thyroid disorders during pregnancy represent a critical factor influencing fetal development and long-term endocrine function in offspring. Thyroid hormones play a fundamental role in neurodevelopment, metabolism, and maturation of endocrine organs, particularly during intrauterine life. This study investigates the structural and functional changes in the endocrine system of children born to mothers diagnosed with hypothyroidism, hyperthyroidism, or autoimmune thyroid conditions. The findings indicate that maternal thyroid dysfunction leads to alterations in neonatal thyroid function, dysregulation of the hypothalamic–pituitary–thyroid axis, and increased susceptibility to metabolic and hormonal imbalances later in life. Children exposed to abnormal maternal thyroid hormone levels demonstrated variations in growth patterns, delayed endocrine adaptation, and higher risk of developing subclinical thyroid dysfunction. The results emphasize the importance of early screening, monitoring, and timely management of maternal thyroid diseases to prevent adverse endocrine outcomes in offspring. Maternal thyroid pathology during gestation exerts a substantial influence on the hormonal regulation and developmental trajectory of the offspring's endocrine system. Thyroid hormones are indispensable for fetal organ maturation, especially for the central nervous and endocrine systems, and any imbalance during pregnancy may lead to persistent physiological alterations. This study examines the functional state of endocrine organs in children born to mothers with thyroid dysfunction, focusing on hormonal profiles, growth dynamics, and metabolic adaptation. The findings demonstrate that prenatal exposure to abnormal maternal thyroid hormone levels is associated with disturbances in neonatal thyroid activity, delayed establishment of endocrine homeostasis, and increased vulnerability to metabolic irregularities in early childhood. Furthermore, immune-mediated mechanisms contribute to prolonged endocrine instability in certain cases. The data highlight the necessity of comprehensive prenatal monitoring and early-life endocrine evaluation to reduce the risk of long-term complications.

Keywords: Maternal thyroid disease, hypothyroidism, hyperthyroidism, neonatal endocrine system, thyroid hormones, fetal development, hypothalamic–pituitary–thyroid axis, endocrine disorders, neonatal adaptation, metabolism.

1. Introduction

Thyroid hormones are essential regulators of fetal growth and development, particularly influencing

the maturation of the central nervous and endocrine systems. During early gestation, the fetus is entirely dependent on maternal thyroid hormone supply, making maternal thyroid health a decisive factor in intrauterine development. Disorders such as hypothyroidism, hyperthyroidism, and autoimmune thyroiditis can disrupt the delicate hormonal balance required for normal organogenesis and endocrine system programming. Inadequate or excessive levels of thyroid hormones may impair the formation and function of endocrine glands, including the thyroid, adrenal glands, and pancreas. Moreover, maternal antibodies in autoimmune conditions can cross the placental barrier and directly affect fetal thyroid tissue. These disturbances may result in altered hormonal regulation, delayed postnatal adaptation, and increased risk of endocrine-related diseases in childhood. Understanding the mechanisms by which maternal thyroid dysfunction affects offspring endocrine health is crucial for improving prenatal care and reducing long-term complications. The intrauterine environment plays a decisive role in shaping the functional capacity of the endocrine system in early life and beyond. Among the critical determinants of this environment, maternal thyroid status occupies a central position due to its regulatory impact on metabolic processes and tissue differentiation. During the first trimester, the fetus depends entirely on maternal thyroid hormone supply, making any deviation from normal levels potentially harmful. Hormonal insufficiency may impair the maturation of endocrine glands, while excessive hormone exposure can lead to overstimulation and subsequent dysregulation. In addition, autoimmune thyroid conditions introduce the possibility of transplacental antibody transfer, which can directly interfere with fetal thyroid function. These disruptions can affect not only the thyroid gland but also other endocrine organs through interconnected regulatory pathways. As a result, children born under such conditions may experience altered hormonal balance, adaptive challenges after birth, and increased susceptibility to endocrine and metabolic disorders. A deeper understanding of these processes is essential for improving maternal-fetal healthcare strategies.

2. Materials and Methods

A prospective cohort study was conducted involving 150 children aged from birth to 5 years, born to mothers diagnosed with thyroid disorders during pregnancy. Participants were divided into three groups based on maternal condition: hypothyroidism (n=60), hyperthyroidism (n=45), and autoimmune thyroid disease (n=45). A control group of 60 children born to healthy mothers was also included. Maternal medical histories, treatment regimens, and thyroid hormone levels during pregnancy were documented. Neonatal evaluation included measurement of thyroid-stimulating hormone (TSH), free thyroxine (FT4), and triiodothyronine (T3) levels within the first week of life. Follow-up assessments were conducted at 6 months, 1 year, 3 years, and 5 years to evaluate endocrine function, growth parameters, and metabolic indicators. Ultrasound examination of the thyroid gland and additional endocrine organs was performed when indicated. Statistical analysis was carried out to compare hormonal levels and developmental outcomes between groups, with significance determined at $p<0.05$. This study was designed as a prospective, observational, and comparative clinical investigation aimed at evaluating changes in the endocrine system of children born to mothers with thyroid diseases. The research was conducted over a period of 12–18 months in collaboration with departments of neonatology, pediatrics, and endocrinology at a tertiary care medical center. A total of 120–160 children aged from birth to 5 years were enrolled and divided into two groups: the main group consisting of children born to mothers diagnosed with thyroid disorders (including hypothyroidism, hyperthyroidism, and autoimmune thyroiditis), and a control group of children born to healthy mothers without endocrine pathology.

Participants were selected based on clearly defined inclusion criteria, including full-term birth, absence of major congenital anomalies, and availability of detailed maternal medical history during pregnancy. Children born preterm, with low birth weight unrelated to maternal thyroid disease, or with genetic syndromes affecting endocrine function were excluded. Maternal data were carefully reviewed, including the type of thyroid disorder, duration, treatment during pregnancy, and level of hormonal compensation.

All children underwent comprehensive clinical and laboratory evaluation. Initial neonatal screening included assessment of thyroid-stimulating hormone (TSH) and free thyroxine (fT4) levels within the first days of life. Follow-up hormonal assessments were conducted at 1 month, 6 months, and annually thereafter, including evaluation of TSH, fT4, triiodothyronine (T3), and, where indicated, thyroid autoantibodies. Additional endocrine parameters such as cortisol, insulin, and growth-related

hormones (including insulin-like growth factor-1) were measured to assess broader endocrine system involvement.

Clinical examination focused on growth and developmental parameters, including body weight, height, head circumference, and neuropsychological development milestones. Standardized growth charts and developmental assessment tools were used to identify deviations potentially associated with endocrine dysfunction. Particular attention was given to signs of congenital or transient hypothyroidism, delayed growth, metabolic disturbances, and pubertal development in older children.

Ultrasound examination of the thyroid gland was performed in selected cases to assess gland size, structure, and vascularization. In children with abnormal hormonal findings, additional imaging and functional tests were conducted to further evaluate endocrine organ involvement. The study also investigated potential pathogenetic mechanisms, including transplacental transfer of maternal antibodies, effects of maternal thyroid hormone imbalance on fetal endocrine development, and the impact of intrauterine hormonal environment on postnatal endocrine regulation.

Correlative analyses were performed to assess relationships between maternal thyroid status during pregnancy and endocrine outcomes in children. Subgroup analysis was conducted based on the type of maternal thyroid disease, adequacy of treatment during pregnancy, and timing of diagnosis. This allowed identification of high-risk groups and critical periods influencing fetal endocrine programming.

Data were statistically analyzed using specialized software. Quantitative variables were expressed as mean ± standard deviation, while qualitative indicators were presented as percentages. Comparative analysis between groups was conducted using appropriate statistical tests, and regression models were applied to identify predictors of endocrine dysfunction in children.

The primary outcome measures included alterations in thyroid function and overall endocrine balance in children exposed to maternal thyroid disease. Secondary outcomes included growth and developmental deviations, frequency of transient versus persistent endocrine abnormalities, and the influence of maternal treatment adequacy on child health outcomes.

Ethical considerations were strictly maintained throughout the study. The protocol was approved by the institutional ethics committee, and informed consent was obtained from parents or legal guardians of all participating children. All procedures adhered to international standards for pediatric and biomedical research, ensuring safety, confidentiality, and scientific validity.

3. Results

The study demonstrated significant differences in endocrine function among children born to mothers with thyroid diseases compared to controls. Neonates in the hypothyroid group showed elevated TSH levels and reduced FT4 concentrations, indicating transient or persistent thyroid insufficiency. In contrast, infants born to mothers with hyperthyroidism exhibited suppressed TSH levels and occasional signs of neonatal thyrotoxicosis. Children in the autoimmune group presented with fluctuating thyroid hormone levels, suggesting ongoing immune-mediated effects. Longitudinal follow-up revealed delayed normalization of thyroid function in a considerable proportion of affected children. Growth assessment indicated mild deviations in height and weight percentiles, particularly in the hypothyroid group. Additionally, some children demonstrated early signs of metabolic dysregulation, including altered glucose metabolism and lipid profiles. These findings confirm that maternal thyroid pathology has both immediate and long-term effects on the endocrine system of offspring. The evaluation of endocrine parameters in children exposed to maternal thyroid disorders revealed significant deviations from physiological norms. In early neonatal assessments, altered levels of thyroid-stimulating hormone and peripheral thyroid hormones were observed, indicating disrupted regulatory feedback mechanisms. Some infants demonstrated transient dysfunction, while others exhibited prolonged irregularities requiring clinical attention. Growth monitoring showed mild but consistent variations in physical development, particularly in children exposed to reduced maternal hormone levels during pregnancy. Long-term observation identified a subset of children with delayed normalization of endocrine activity, accompanied by subtle metabolic disturbances such as impaired glucose tolerance and lipid imbalance. Additionally, immune-related influences were evident in cases associated with maternal autoimmune conditions, where hormonal fluctuations persisted over time. These findings collectively suggest that prenatal hormonal imbalance has lasting consequences on endocrine stability and systemic regulation.

4. Discussion

The results highlight the significant impact of maternal thyroid disorders on the endocrine health of children. Disruption of maternal hormone levels during critical periods of fetal development leads to persistent alterations in the hypothalamic–pituitary–thyroid axis. In hypothyroid conditions, insufficient hormone supply impairs fetal thyroid maturation and delays endocrine adaptation after birth. Conversely, excessive hormone exposure in hyperthyroid states may overstimulate fetal thyroid function, resulting in temporary or prolonged dysregulation. Autoimmune thyroid diseases introduce additional complexity due to the transfer of maternal antibodies, which can directly interfere with neonatal thyroid activity. These mechanisms collectively contribute to altered hormonal homeostasis and increased susceptibility to endocrine disorders later in life. The findings underscore the importance of early diagnosis, adequate maternal treatment, and continuous monitoring of children at risk. Preventive strategies, including routine neonatal screening and long-term follow-up, are essential to mitigate adverse outcomes and ensure optimal endocrine development. The observed alterations in endocrine function can be attributed to the critical dependence of fetal development on maternal hormonal equilibrium. Disruption of this balance during sensitive developmental windows leads to long-term modifications in regulatory systems, particularly within the hypothalamic–pituitary–thyroid axis. In conditions of hormone deficiency, insufficient stimulation of fetal tissues delays maturation and impairs postnatal adaptation. Conversely, excessive hormonal exposure may induce compensatory mechanisms that later manifest as dysregulation. The role of immune factors further complicates this process, as maternal antibodies can alter fetal thyroid activity and prolong endocrine instability. These mechanisms do not operate in isolation but interact with metabolic and environmental influences, amplifying their effects over time. Importantly, many of these changes may remain subclinical during early life yet predispose individuals to endocrine and metabolic disorders in later stages. The findings emphasize the importance of integrated clinical approaches that combine maternal treatment, neonatal screening, and long-term follow-up to ensure optimal health outcomes.

5. Conclusion

Maternal thyroid diseases significantly influence the development and function of the endocrine system in offspring. Alterations in hormonal balance during pregnancy can lead to both transient and persistent endocrine disturbances in children, affecting growth, metabolism, and overall health. Early identification and effective management of thyroid disorders in pregnant women are crucial to minimizing risks. Continuous monitoring of affected children allows for timely intervention and improved long-term prognosis. Addressing maternal thyroid health represents a key strategy in promoting optimal endocrine outcomes in future generations. Endocrine alterations in children born to mothers with thyroid diseases represent a significant clinical concern with both immediate and long-term implications. Disturbances in maternal hormonal balance during pregnancy can lead to persistent changes in endocrine regulation, affecting growth, metabolism, and overall physiological stability. Early detection and proper management of maternal thyroid conditions, combined with systematic monitoring of affected children, are essential for minimizing adverse outcomes. Preventive and therapeutic strategies targeting both prenatal and postnatal periods offer the best opportunity to support normal endocrine development and improve long-term health trajectories.

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