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A Narrative Review of Secondary Osteoporosis Diagnosis, Follow-Up and Treatment in Vulnerable Children

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Introduction

The coordination and operation of several developmental processes are necessary for the function, synthesis, and secretion of thyroid hormone. Any stage of this process can experience dysfunction. The maturation, differentiation, and cell migration of the thyroid gland itself are required processes. The hypothalamic-pituitary-thyroid axis must mature properly, and the thyroid gland must reach sexual maturity, according to the systemic-endocrine perspective. The foetal thyroid gland reaches maturity at around 11–12 weeks of gestation, but thyroid hormone is not secreted until the middle of the second trimester, at about 16–17 weeks. Given that the foetal central nervous system (CNS) develops during the first trimester, an adequate source of maternal thyroid hormone is necessary prior to this period. Numerous studies on animals have demonstrated that the placental barrier is permeable to maternal thyroid hormones. In addition, the placenta contains deiodinases that can convert T4 to T3. Cretiny, which manifests as deaf-mutism, mental retardation, and spasticity, is a result of low thyroid hormone levels during foetal development in populations with severe maternal hypothyroidism [1].

Cretinism, which is characterised by severely stunted physical growth and mental disabilities, can result from congenital hypothyroidism. Dysgenesis, hypoplastic thyroid, pituitary dysfunction, normal thyroid tissue with dysfunctional thyroid hormone production, and absence of the thyroid gland are all possible causes of congenital hypothyroidism (dyshormonogenesis) [2].

Additionally, syndromic conditions like Pendred syndrome, Bamforth-Lazarus syndrome, or brain-lung-thyroid syndrome may manifest as congenital hypothyroidism. An autosomal recessive disease called Pendred syndrome is brought on by mutations in the SLC26A4 (pendrin) gene on chromosome 7. A cellular ion transport protein called pendrin, its dysfunction causes hearing loss and a decrease in the incorporation of iodine into the thyroid. Loss of thyroid transcription factor 2 (FOXE1), which is encoded by the FOXE1 gene, leads to Bamforth-Lazarus syndrome (TTF2). TTF2 loss causes abnormal thyroid morphogenesis. Last but not least, a mutation in the NKX2-1 gene, which codes for the homeobox protein Nkx-2, causes the Brain-lung-Thyroid Syndrome. Because homeobox proteins like Nkx-2 are essential for embryogenesis, their loss causes organ dysmorphogenesis.

Description

The ideal pharmacokinetics of amniotic uptake and conversion are currently unknown, despite the fact that numerous different protocols and dosages have been reported. The optimal gestational age to begin treatment is also debatable, with some women starting it as soon as the diagnosis is made and others delaying it until closer to the third trimester because of the risk of infection. Therapeutic monitoring is frequently carried out using a variety of techniques, including serial ultrasounds to measure the size of the goitre, cordocentesis to measure foetal hormones directly, and amniotic fluid to measure them inferentially. After intra-amniotic therapy, success rates as high as 70% have been reported. When goitre shrinkage is not noted, inefficiency should be carefully taken into account [3].

Compared to foetal hypothyroidism, foetal hyperthyroidism is much less frequent. Thyroid-stimulating antibodies pass through the placenta and cause foetal hyperthyroidism, most frequently when maternal Grave's disease is present. Fulminant neonatal hyperthyroidism is a rare occurrence (1% of mothers with Grave's disease), despite being a life-threatening condition. These antibodies cause the foetal thyroid to activate in an uncontrolled manner. Anti-thyroid antibodies that are circulating (and therefore transplacental) continue to exist after definitive maternal therapy, such as thyroidectomy or radioablation. While foetal overt hyperthyroidism may cause intrauterine growth restriction (IUGR), goitre formation, tachycardia, and heart failure, subclinical hyperthyroidism is not linked to poor foetal outcomes. For patients with uncontrolled hyperthyroidism and TRAb levels three times the normal value, screening and diagnosis for foetal findings of hyperthyroidism are advised. Growth evaluation, brain, heart, and skeletal development, amniotic fluid volumes, and thyroid goitres are all included in ultrasound screening. Additionally, skilled perinatalologists should be consulted for prenatal care coordination.

A careful balance between the health of the mother and the foetus is the therapeutic approach to foetal hyperthyroidism. PTU can be administered to the mother while being closely monitored to prevent the previously described iatrogenic hypothyroidism. Hepatotoxicity and leukocytosis are additional conditions linked to PTU. Levothyroxine add-back therapy is not advised once euthyroidism has been achieved due to the increased side effect profile and difficulty identifying the underlying cause of side effects. Given reports of teratogenicity like choanal atresia, aplasia cutis, and hearing loss, PTU is preferred over MMI. However, maternal levothyroxine supplementation is necessary in the case of mothers receiving effective treatment for Grave's disease [4].

The idea that thyroid hormones are transferred from the mother to the foetus has been called into question by developments in recent years. The natural iodothyronines T4, T3, and reverse triiodothyronine (rT3) are difficult to cross the placenta, according to early animal studies. Both T4 and T3 are actively deiodinated by the placenta's inner ring (5-deiodinase), which keeps them from sufficiently reaching the foetal compartment. However, research on chickens and embryos has revealed that thyroid hormones both T4 and T3 are effectively transferred from the mother to the foetus even before the start of foetal thyroid function, indicating a critical role for maternal thyroid hormones in foetal growth early in pregnancy. T3 receptors are additionally present in the rat brain at an early stage.

More debatable is the transfer of thyroid hormone from the mother to the foetus in humans. Early research with thyroxine and triiodothyronine given at term gestation revealed that the transfer was slow and that T3 appeared to cross the placenta more readily than T4. Thyroid hormone levels were significantly lower in cord serum than in maternal serum, even when high doses of T4 were infused into pregnant women at term. To ascertain the amount of maternal transfer of T4, Vulsma and associates124 recently studied 25 congenitally hypothyroid neonates who were born with a complete organification defect.

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Compared to normal ranges of 80 to 170 nmol/L, the serum T4 concentration was between 35 and 70 nmol/L. These studies suggest that, at least during the last trimester [5].

Conclusion

The clinical symptom of a developing fetus's thyroid gland dysfunction is goitres. According to estimates, there are between 1:30,000 and 1:50,000 live births per year that have foetal thyroid goitre. Thyroid Receptor Antibodies (TRAbs) come in two varieties: Thyroid Stimulating Antibody (TSAb) and Thyroid Stimulating Blocking Antibody (TSBAb). TSAB results in hypothyroidism, while TSBAb causes hyperthyroidism. Less frequently does TSBAb result in foetal thyroid goitres .

Fetal thyroid goitres are known to have both mechanical (mass effect) and biochemical effects. Due to the mass's general size and location, possible (although rare) complications include dystocia during labour and esophageal and tracheal compression leading to polyhydramnios or asphyxia. The aetiology of the goitre affects the biochemical effects of thyroid goitres. Cardiac failure, growth restriction, and mental retardation are all problems associated with hyperthyroidism. Deafness and delays in developmental milestones related to motor and mental function are both seen in hypothyroidism cases. Both of these will be described later. Ultrasonography is used to initially diagnose foetal thyroid goitres. The second or third trimester is when this is most likely to happen.

Acknowledgement

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Conflict of Interest

None.

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