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CONGENITAL HYPOTHYROIDISM AND NEONATAL SCREENING: IMPACT OF EARLY DIAGNOSIS ON CHILD DEVELOPMENT

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ABSTRACT

Introduction: Congenital hypothyroidism (CH) is one of the most common endocrine disorders in the neonatal period and remains one of the leading preventable causes of intellectual disability in children.
Objective: To describe a clinical case of CH diagnosed early through newborn screening, emphasizing the impact of early diagnosis and timely therapeutic intervention on child development.
Methods: This was a retrospective, descriptive, observational case report based on a review of the electronic medical records of a patient treated at a hospital in the state of Goiás, Brazil.
Results: A term female newborn (39 weeks and 5 days of gestation; birth weight, 2,300 g) was admitted to the Neonatal Intensive Care Unit because of respiratory distress following cesarean delivery for acute fetal distress. Newborn screening revealed markedly elevated thyroid-stimulating hormone (TSH) levels (212.74 µIU/mL), subsequently confirmed by serum TSH >100 µIU/mL, triiodothyronine (T3) of 0.58, and free thyroxine (FT4) of 0.72, consistent with primary CH. Echocardiography demonstrated a patent ductus arteriosus (0.32 cm) and patent foramen ovale, with no abnormalities on transfontanellar ultrasonography and antithyroglobulin antibody levels <3.0. Following early initiation of levothyroxine replacement therapy, the patient showed favorable clinical evolution, with resolution of neonatal complications and subsequent transfer to the pediatric ward.
Conclusions: The absence of specific clinical manifestations during the first days of life may hinder the early recognition of CH. Newborn screening plays a central role in identifying thyroid dysfunction before the onset of clinical manifestations, enabling diagnostic confirmation and timely initiation of hormone replacement therapy. Early levothyroxine treatment substantially improves the prognosis of the disease.

Keywords: Brazil, Infant mortality, Pediatrics, Premature, Neonatal sepsis.

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INTRODUCTION

Congenital hypothyroidism (CH) is one of the most prevalent endocrine disorders in the neonatal period and is the leading potentially preventable cause of intellectual disability worldwide. In the absence of early diagnosis and appropriate therapeutic intervention, thyroid hormone deficiency can cause significant alterations in child growth and neuropsychomotor development, including delayed somatic development, cognitive impairment, and permanent intellectual deficits. Given its clinical and epidemiological relevance, Brazil established the Clinical Protocol and Therapeutic Guidelines for CH, formalized by Joint Ordinance No. 5, published in April 2021.[1]

CH is one of the most common endocrine disorders in the neonatal period and remains one of the leading preventable causes of intellectual disability in children. Deficient production or action of thyroid hormones during the early stages of development compromises fundamental processes of growth, brain maturation, and neuropsychomotor development, potentially resulting in irreversible cognitive impairment when diagnosis and treatment are not initiated early. Given its considerable clinical and epidemiological relevance, the Brazilian Ministry of Health published the Clinical Protocol and Therapeutic Guidelines for CH in 2021, establishing recommendations for the diagnosis, treatment, and longitudinal follow-up of these patients.[1]

In the context of Brazilian public health, the main strategy for the early identification of CH is universal newborn screening, performed through the heel prick test and provided free of charge by the Brazilian Unified Health System (SUS). This program enables the detection of the disease before the onset of clinical manifestations, allowing the timely initiation of hormone replacement therapy and significantly reducing the risk of permanent neurological sequelae. The consolidation of newborn screening represents one of the greatest advances in controlling this disorder, as most affected newborns appear clinically normal during the first weeks of life.[1,2]

The etiopathogenesis of CH is heterogeneous and involves different mechanisms responsible for reduced thyroid hormone synthesis or action. The main causes include thyroid gland dysgenesis, encompassing agenesis, ectopia, and hypoplasia, which account for most cases, as well as defects in hormone biosynthesis, central abnormalities of the hypothalamic-pituitary-thyroid axis, and transient forms associated with maternal or environmental factors. Studies have also described associations between the occurrence of the disease and factors such as advanced maternal age, gestational complications, prematurity, low birth weight, multiple gestation, female sex, and the presence of congenital malformations, highlighting the multifactorial nature of this condition.[2,3]

From a pathophysiological perspective, CH results from deficient production of thyroxine (T4) and triiodothyronine (T3), hormones essential for regulating cellular metabolism and the structural and functional differentiation of multiple organs and systems. During the neonatal period and the first years of life, these hormones play a crucial role in myelination, neurogenesis, synaptogenesis, and central nervous system maturation. Consequently, persistent hormone deficiency during critical stages of brain development may result in intellectual impairment, motor delay, and permanent cognitive abnormalities. The estimated worldwide prevalence ranges from one case per 1,500 to 4,000 live births, with a slight predominance among females and a higher frequency in individuals with Down syndrome.[3]

From a clinical perspective, recognizing CH solely on the basis of signs and symptoms presents a diagnostic challenge, as approximately 95% of newborns remain asymptomatic at birth due to

the transplacental transfer of maternal thyroid hormones. When present, initial manifestations are generally nonspecific and include excessive somnolence, difficulty sucking, constipation, hypotonia, and a hoarse cry. As the disease progresses, characteristic physical findings may emerge, such as macroglossia, umbilical hernia, dry skin, coarse facial features, delayed bone maturation, enlarged fontanelles, and delayed deep tendon reflexes. In cases resulting from dyshormonogenesis, the presence of goiter may constitute an important clinical indicator, while prolonged neonatal jaundice should prompt investigation for thyroid dysfunction and other associated endocrine disorders.[4]

Given the low sensitivity of clinical assessment during the neonatal period, diagnostic confirmation is based on laboratory screening performed through the heel prick test, preferably between the third and fifth days of life, when hormone levels exhibit greater physiological stability. Elevated thyroid-stimulating hormone (TSH) levels, together with free thyroxine (FT4) measurement, constitute the main criteria for diagnostic confirmation. Following diagnosis, immediate initiation of levothyroxine replacement therapy is recommended, generally at initial doses of approximately 10–15 µg/kg/day, followed by periodic clinical and laboratory monitoring to adjust treatment and assess child development.[5]

Differentiating between permanent and transient forms has important clinical and prognostic implications. While transient CH is characterized by temporary hormone deficiency, with spontaneous recovery of thyroid function after the first months or years of life, the permanent form requires continuous treatment for an indefinite period. Correctly distinguishing between these presentations prevents both premature discontinuation and unnecessary continuation of hormone replacement therapy.[3]

Although treatment is simple, safe, and highly effective when initiated early, delays in diagnostic confirmation or the initiation of hormone replacement therapy remain associated with cognitive deficits, behavioral abnormalities, and impaired neuropsychomotor development. In this context, multidisciplinary follow-up is essential to monitor growth, neurological development, therapeutic response, and treatment adherence, promoting better clinical outcomes and quality of life throughout child development.[6]

Thus, CH remains an important public health concern, not because of high mortality rates, but because of its potential to cause permanent neurological sequelae when not diagnosed promptly. Universal newborn screening, combined with early treatment and specialized longitudinal follow-up, constitutes the most effective strategy for preventing disease-related intellectual disability, reinforcing the need to continuously strengthen public policies aimed at newborn screening and improving the quality of care provided to affected children.[7]

Given the relevance of this topic, the present study aims to describe a clinical case of CH diagnosed early through newborn screening, emphasizing the impact of early diagnosis and therapeutic intervention on child development.

METHODS

Patient This was a retrospective, descriptive, observational case report based on a review of the electronic medical records of a patient treated at a hospital in the state of Goiás, Brazil.

The selected case involved a female child from Anápolis who was admitted to a Neonatal Intensive Care Unit (NICU) and diagnosed early with congenital hypothyroidism (CH) through newborn screening. The unit of analysis consisted of a single medical record,

selected because of the clinical relevance of the case.

The inclusion criteria comprised clinical records of patients diagnosed with CH who were followed up by the neonatology service. Medical records containing incomplete or inconsistent clinical information, as well as conditions that could interfere with the standardization of the clinical and therapeutic analysis, were excluded.

Data collection was preceded by the signing of the Informed Consent Form (ICF) by the patient's legal guardian in a private setting, after appropriate explanations regarding the study objectives, the use of clinical information, and the guarantees of confidentiality and anonymity. Subsequently, the electronic medical record available in the hospital's institutional system was reviewed.

Information regarding demographic characteristics, clinical history, medical history, physical examination findings, and complementary examinations was extracted. All information was recorded using a data collection instrument developed by the researcher exclusively for this study, ensuring the patient's confidentiality and anonymity throughout all stages of the investigation.

As a complementary strategy, a narrative review of the scientific literature was conducted to contextualize the clinical findings and discuss the results in light of the available evidence. The literature search was conducted in the MEDLINE/PubMed, LILACS, and Scientific Electronic Library Online (SciELO) databases, as well as through consultation of books, dissertations, theses, and technical documents considered relevant to the topic. Priority was given to recent publications and studies with higher levels of evidence related to CH.

The data obtained were subjected to descriptive analysis and presented in narrative form and, when appropriate, organized into tables and figures to facilitate understanding of the clinical characteristics, the therapeutic approach employed, and the clinical course of the case.

This study was approved by the institution's Research Ethics Committee under Certificate of Presentation for Ethical Consideration (CAAE) No. 00229126.7.0000.5076 and approval opinion No. 8.776.888, in accordance with the Guidelines and Regulatory Standards for Research Involving Human Subjects, as established by National Health Council Resolution No. 466/2012.

RESULTS

Atypical A term female newborn (39 weeks and 5 days of gestation), appropriate for gestational age, was admitted to the Neonatal Intensive Care Unit (NICU) due to respiratory distress during the first hours of life. Delivery was performed by cesarean section because of acute fetal distress, with cephalic presentation, clear amniotic fluid, a birth weight of 2,300 g, and Apgar scores of 8 and 9 at the first and fifth minutes, respectively.

The patient required neonatal resuscitation maneuvers and subsequently developed persistent respiratory distress. Initially, oxygen therapy was administered using an oxygen hood with a fraction of inspired oxygen (FiO_2) of 40%, without a satisfactory clinical response. Admission to a neonatal intensive care unit was therefore requested for monitoring and specialized treatment.

The mother, aged 31 years, had an obstetric history of G3P2A0, blood type O Rh-positive, and had attended 12 prenatal consultations. A urinary tract infection occurred late in pregnancy and was appropriately treated. The newborn had blood type A Rh-positive.

During the patient's clinical investigation, echocardiography revealed a patent ductus arteriosus (PDA) measuring 0.32 cm, with mild to moderate flow, associated with a patent foramen ovale (PFO).

Concurrently, newborn screening identified markedly elevated TSH levels, confirming the diagnosis of CH. Following laboratory confirmation, levothyroxine sodium (Puran T4) replacement therapy was immediately initiated according to the institutional protocol.

During her stay in the NICU, the newborn remained in a heated incubator under continuous pulse oximetry monitoring and received nutritional support through a nasogastric tube. Due to neonatal hyperbilirubinemia, she underwent phototherapy for 3 days, with satisfactory resolution of the condition.

On physical examination, the patient was in fair general condition, with normal skin coloration, adequate hydration, no fever, cyanosis, or jaundice, and reduced responsiveness to stimuli. Cardiovascular examination revealed a regular heart rhythm, normal heart sounds, the presence of a cardiac murmur, and adequate peripheral perfusion. Pulmonary auscultation revealed preserved bilateral vesicular breath sounds, without adventitious sounds or clinical signs of respiratory distress. The abdomen was mildly distended, soft, and nontender to superficial and deep palpation, with no organomegaly or palpable masses. The lower limbs exhibited palpable and symmetrical peripheral pulses, without relevant abnormalities. The external genitalia were consistent with female sex.

During hospitalization, diagnoses of neonatal jaundice secondary to ABO incompatibility, which resolved after phototherapy, successfully treated late-onset neonatal sepsis, and an episode of neonatal respiratory distress with a favorable clinical course were also recorded.

Further investigation confirmed laboratory abnormalities consistent with primary CH. The heel prick test revealed a TSH concentration of 123.99 µIU/mL. Subsequently, repeat serum testing demonstrated TSH levels greater than 100 µIU/mL, associated with reduced triiodothyronine (T3 = 0.58) and free thyroxine (FT4 = 0.72) levels. Antithyroglobulin antibody testing yielded a result below 3.0, while transfontanellar ultrasonography showed no structural abnormalities. Blood cultures collected from two independent samples were negative, ruling out active bacterial infection. The main examinations performed during hospitalization are summarized in Table 1.

Table 1. Newborn laboratory and imaging test results.

Date	Tests	Results
06/11/2025	Echocardiography	PFO + PDA (0.32 cm) with mild to moderate flow
06/18/2025	Blood culture	Negative (two samples)
06/23/2026	Newborn screening	TSH 212.74 µUI/mL
06/30/2025	TSH	Greater than 100 µIU/mL
06/30/2025	T3/ FT4	0.58/ 0.72
06/30/2025	Antithyroglobulin antibody	< 3,0
07/01/2025	Transfontanellar ultrasonography	No abnormalities

Note: TSH: thyroid-stimulating hormone; PFO: patent foramen ovale; PDA: patent ductus arteriosus; T3: triiodothyronine; FT4: free thyroxine; NR: normal.
Source: Patient's medical records.

Following clinical stabilization, progressive improvement in respiratory status, resolution of neonatal complications, and initiation of hormone replacement therapy for CH, the patient showed satisfactory clinical progress, allowing transfer from the neonatal intensive care unit (NICU) to the pediatric ward, where she remained under clinical monitoring until hospital discharge.

DISCUSSION

The present report describes a case of CH diagnosed early through newborn screening, enabling the timely initiation of hormone replacement therapy and favorable clinical evolution. This finding reinforces the importance of universal newborn screening programs, since most affected newborns present with initial physical examination findings that are not strongly suggestive of the disease, making diagnosis based exclusively on clinical evaluation unfeasible. In the present case, despite the need for neonatal hospitalization due to respiratory distress and the presence of associated clinical complications, the diagnosis of CH was established based on the markedly elevated TSH levels identified through newborn screening, allowing therapeutic intervention before the onset of irreversible neurological manifestations.

This clinical presentation is consistent with the pathophysiology of CH described in the literature. During the first weeks of life, the transplacental transfer of maternal thyroid hormones exerts a transient protective effect on the newborn, partially masking the effects of hormone deficiency. Consequently, only a small proportion of patients exhibit evident clinical manifestations at birth, while most remain oligosymptomatic until maternal hormone stores are progressively depleted. When diagnosis is delayed, manifestations such as prolonged jaundice, macroglossia, hypotonia, umbilical hernia, constipation, feeding difficulties, impaired weight gain and linear growth, and progressive impairment of neuropsychomotor development may occur, culminating, in untreated cases, in permanent intellectual disability.[8]

Another aspect observed in this report was the concomitant presence of patent ductus arteriosus (PDA) and patent foramen ovale (PFO). Although these cardiac abnormalities may occur independently during the neonatal period, recent studies have demonstrated a higher frequency of congenital malformations among individuals with CH, particularly structural heart defects associated with forms resulting from thyroid dysgenesis. The reported abnormalities include septal defects, PDA, renal anomalies, and other congenital malformations, suggesting possible shared biological mechanisms during embryogenesis, although this relationship has not yet been fully elucidated.[9,11]

In the present case, the TSH concentration identified through newborn screening (123.99 µIU/mL), subsequently confirmed by persistently elevated serum levels exceeding 100 µIU/mL, together with reduced T3 and free thyroxine (FT4) levels, was consistent with severe primary CH. International recommendations establish that newborns with significantly elevated TSH levels should be immediately referred for diagnostic confirmation and specialized follow-up. Whenever possible, additional laboratory tests and imaging studies should be performed before treatment initiation; however, when TSH levels are markedly elevated or there is a risk of therapeutic delay, the immediate initiation of levothyroxine should be prioritized and should not be postponed for imaging studies.[12]

Early hormone replacement therapy remains the main strategy for preventing the neurological complications of CH. Guidelines from the European Society for Paediatric Endocrinology and

the American Academy of Pediatrics recommend that levothyroxine be initiated immediately after diagnostic confirmation, preferably before the second week of life, at initial doses ranging from 10 to 15 µg/kg/day. This approach promotes rapid normalization of serum thyroxine concentrations, generally within a few days, followed by a progressive reduction in TSH levels over the subsequent weeks, substantially reducing the risk of permanent cognitive sequelae.[11,13]

Despite the high efficacy of hormone replacement therapy, longitudinal follow-up remains essential. Recent studies have demonstrated that patients with more severe forms of the disease or extremely low T4 levels before treatment frequently require more intensive therapeutic adjustments during the first months of life. Furthermore, individuals with thyroid dysgenesis tend to require higher doses of levothyroxine throughout follow-up, a characteristic that may assist in etiological differentiation when imaging studies are not readily available.[14]

Although early treatment is associated with excellent neurocognitive outcomes in most patients, recent evidence indicates that children affected by severe forms of CH may exhibit subtle deficits in attention, memory, language, and motor coordination, even after adequate hormone replacement therapy. These findings reinforce that the follow-up of these children should not be restricted to laboratory monitoring, and periodic multidisciplinary assessment of neuropsychomotor development throughout childhood is recommended.[11]

In recent years, some authors have begun discussing the possibility of individualizing the initial levothyroxine dose, since excessively high doses could be associated with potential future metabolic and neurobehavioral effects. However, to date, the evidence remains insufficient to modify current recommendations, and the prevailing consensus is that the risks associated with delayed treatment far outweigh the potential effects of overtreatment, provided that clinical and laboratory monitoring is performed systematically.[15]

Another relevant aspect highlighted in the literature concerns the impact of newborn screening programs on the epidemiological profile of CH. The expansion of screening program coverage, combined with the reduction of TSH cutoff values and the implementation of reassessment strategies in high-risk groups, has contributed to increased identification of mild and transient forms of the disease. Although these cases are clinically less severe, their recognition remains important to prevent diagnostic delays and ensure individualized follow-up, particularly among premature infants, low-birth-weight newborns, and infants from multiple pregnancies.[16]

In the present case, the early identification of CH through newborn screening enabled the immediate initiation of levothyroxine, promoting favorable clinical evolution and significantly reducing the risk of permanent neurological impairment. Thus, this report reinforces the importance of maintaining and strengthening universal newborn screening programs, ensuring timely access to specialized services, and providing continuous multidisciplinary follow-up, all of which are considered essential to ensuring adequate growth, neuropsychomotor development, and quality of life for children affected by this endocrinopathy.

CONCLUSIONS

The present report demonstrates that the absence of specific clinical manifestations during the first days of life hinders the early recognition of CH when evaluation is restricted to the neonatal physical examination. In this context, newborn screening plays a central role in identifying thyroid dysfunction before the onset of clinical manifestations, enabling

diagnostic confirmation and timely initiation of hormone replacement therapy. Early levothyroxine treatment significantly improves the prognosis of the disease, reducing the likelihood of neurological impairment and promoting adequate neuropsychomotor development, as observed in this case.

The clinical evolution described reinforces that the benefits of early diagnosis depend not only on initial detection but also on continued specialized follow-up, with periodic monitoring of thyroid function and individualized therapeutic adjustments throughout childhood. This approach contributes to maintaining euthyroidism and ensuring adequate somatic growth, neurological maturation, and cognitive performance, minimizing the occurrence of permanent functional limitations.

From a public health perspective, the findings of this report reaffirm the importance of strengthening newborn screening strategies within the Brazilian Unified Health System (SUS), particularly through expanded coverage and reduced intervals between sample collection, release of results, diagnostic confirmation, and treatment initiation. Optimizing these stages of care represents an essential measure to reduce preventable adverse outcomes, promote greater equity in access to healthcare, and ensure better prospects for development, autonomy, and quality of life among individuals affected by CH.

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