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Congenital Hypothyroidism in a 7-Month-Old Infant with Developmental Delay and Stunting

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Abstract. *Congenital hypothyroidism is a preventable cause of growth, developmental, and intellectual disorders when diagnosis and treatment are initiated early. However, clinical manifestations may be subtle during the early stages, resulting in delayed diagnosis when neonatal screening is not performed. This case report describes the clinical presentation, diagnosis, and management of congenital hypothyroidism diagnosed after the neonatal period. A 7-month-old female presented with developmental delay, poor weight gain, constipation, and reduced responsiveness to her environment. Physical examination revealed macroglossia and growth and developmental delays. Anthropometric measurements showed a weight of 5 kg, height of 60 cm, weight-for-age of -2.95, height-for-age of -2.61, and weight-for-height of -1.66. Laboratory evaluation revealed a free thyroxine (FT4) level of 5.15 pmol/L and thyroid-stimulating hormone (TSH) >100,000 µIU/mL, confirming congenital hypothyroidism with stunting. Treatment consisted of levothyroxine at an initial dose of 10 µg/kg/day, nutritional intervention, and physical therapy. After two months, FT4 increased to 11.06 pmol/L and TSH decreased to 12.906 µIU/mL, accompanied by increased activity, weight gain, and developmental improvement. This case emphasizes the importance of neonatal screening, early recognition of developmental abnormalities, and prompt treatment to minimize the adverse consequences of congenital hypothyroidism.*

Keywords: *Congenital Hypothyroidism, Congenital Hypothyroidism Screening, Diagnosis, Treatment*

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INTRODUCTION

Congenital hypothyroidism is a deficiency in thyroid hormone production in newborns resulting from anatomical abnormalities of the thyroid gland, metabolic disorders in thyroid hormone synthesis, and iodine deficiency (Anggraini, D., & Fharel, 2025; Mukhlisatunnafsi, 2024; Darma, 2023). Congenital hypothyroidism (CH) is one of the causes of mental retardation in children that can be prevented if detected and treated early. Thyroid hormones play a role in the development of the central nervous system (including migration and myelination) (Stepien & Huttner, 2019; Calzà et al., 2015; Sawicka-Gutaj et al., 2022).

95% of CH cases do not exhibit characteristic clinical signs and symptoms at birth, and the window for early intervention to prevent mental retardation is very brief (Hadders-Algra,

2021; Vargason et al., 2019). Therefore, most developed countries have implemented neonatal screening programs for the early detection of CH. The incidence of CH varies among countries. Worldwide, the incidence of congenital hypothyroidism is 1 in 3,000 births (Darma, 2023; Mukhlisatunnafsi, 2024; Liu et al., 2023; Dayal & Prasad, 2015; Kiess et al., 2018).

Based on data from the Down Syndrome Registry of the Pediatric Endocrinology Coordination Unit of the Indonesian Pediatric Society (IDAI), sourced from several specific hospitals in Indonesia, the majority of individuals with Down syndrome experience a delayed diagnosis, leading to growth and motor development delays as well as intellectual disabilities (Esbensen etal., 2024; Korlimarla et al., 2021; Horvat et al., 2016; Locatelli et al., 2021; Hendrix et al., 2021). A study in Indonesia by Pulungan et al. showed that delays in initiating early therapy affect IQ, with an average score of 51 in cases that received early therapy at 1.5 years of age (Lumeno et al., 2026; Nugraha & Pradiptha, 2023).

This study also showed that normal FT4 levels support better intellectual development during the remaining period of brain development. Congenital hypothyroidism can be transient or permanent and is classified based on the location of the disorder: primary (in the thyroid gland) or secondary/central (in the pituitary gland and/or hypothalamus); the severity of hypothyroidism: (serum TSH levels > 100 mIU/L are considered severe; and the age of onset of hypothyroidism intrauterine cases are more severe). The most common form is permanent primary congenital hypothyroidism (elevated serum TSH levels) due to thyroid dysgenesis. In permanent congenital hypothyroidism, treatment must be continued for life, whereas treatment is not necessary for the transient form.

METHODS

The methods used in this report consist of patient observation based on medical history, physical examination, and ancillary tests.

Case Report

A 7-month-old female infant was brought to the pediatric outpatient clinic at Daha Sejahtera Regional General Hospital by her mother with complaints that the infant did not respond well when her name was called or when spoken to. The patient also exhibited developmental delays in laughing when teased, returning smiles, or babbling. She showed no interest in reaching for toys around her, could not sit independently, and required parental assistance when placed on her stomach. The patient's mother also reported that her daughter's weight gain was very slow each month and that the patient had infrequent bowel movements, occurring approximately every 5–7 days. A history of previous illnesses such as nausea, vomiting, diarrhea, seizures, and swelling in the child's neck was denied. The patient had not previously received any treatment. The patient's mother has no history of taking medication for thyroid disease. The area where the patient lives is not endemic for iodine deficiency. A family history of similar diseases was denied. Pregnancy history: G3P0A0. The mother attended 3 antenatal care (ANC) visits at the community health center and 1 visit with an obstetrician; she regularly took vitamins and iron supplements; there were no illnesses during pregnancy; and she reported no fever, trauma, asthma, or allergies.

Delivery history: The patient gave birth at the community health center with a status of P4A0 via spontaneous occipital delivery, assisted by a general practitioner and a midwife. Gestational age was 38 weeks; birth weight was 2,800 grams; length was 48 cm; head circumference was 32 cm; the infant cried vigorously; the amniotic fluid was clear; and the umbilical cord was fresh. The patient had no history of prolonged labor. The patient's immunization history showed that she had received age-appropriate vaccinations.

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Table 1. Nutritional status history based on WHO guidelines

AGE	Weight	Height	Weight/Age	Height-for-Age	Weight/Height
7 months	5 kg	60 cm	-3.57	-3.26	-1.82
8 months	5.2 kg	61 cm	-3.57	-3.37	-1.85
9 months	5.5 kg	63 cm	-3.42	-3.06	-2.09
10 months	6.2 kg	64 cm	-2.69	-3.13	-1.11
11 months	6.8 kg	65 cm	-2.12	-3.17	-0.45
12 months	7.2 kg	69 cm	-1.89	-2.04	-1.12

Physical examination revealed general weakness, alert and oriented, pulse 95 bpm, respiratory rate 22 bpm, temperature 36.5°C, SPO2 99%. Head size was normocephalic; hair was black and not dry; conjunctiva was slightly pale; sclera were not icteric; the nose had discharge; the mouth showed macroglossia (Figure 1); and the neck was within normal limits. Vesicular lung sounds were present bilaterally. Examination of the heart, abdomen, and genitalia was within normal limits. Supportive tests including routine blood work, FT4, TSH-S, TORCH, and initial hearing screening yielded the following results: hemoglobin 9.3 g/dL, hematocrit 29.2%, white blood cells 5.4 thousand/µL, platelets 328 thousand/µL, MCV 89.8 fl, MCH 28.6 pg, MCHC 31.8%, SGOT 131 U/L, SGPT 42 U/L, FT4 <5.15 pmol/L, and TSHs >100,000 uIU/mL (Table 2). The initial hearing screening was within normal limits (Figure 2, Table 2, Table 3). Based on the medical history, physical examination, and supportive tests, a working diagnosis of congenital hypothyroidism accompanied by stunting was established. The patient was managed with levothyroxine hormone therapy. The initial dose of levothyroxine was 10–15 µg/kg body weight/day. The patient began levothyroxine therapy at 7 months of age, and FT4 and TSH levels were reevaluated at 9 months of age. Laboratory results after 2 months of treatment showed FT4 levels of 11.06 pmol/L and TSH levels of 12.906 IU/mL, which were significantly improved compared to the previous laboratory results (Table 5). Management of stunting involved providing supplemental food, and the patient was referred to the medical rehabilitation clinic for sensory-motor and speech stimulation.

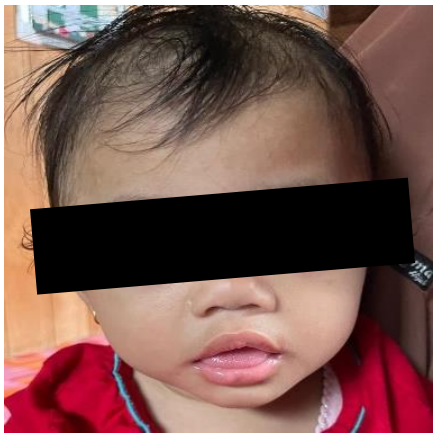

Figure 1. Macroglossia and Flat Facial Features

Table 2. Laboratory Results for FT4, TSHs, and TORCH

Examination	Results	Reference Range	Unit	Method
SGOT	131	5 – 34	U/L	NADH (WITHOUT P-5'-P)
SGPT	42	0–55	U/L	NADH (WITHOUT P-5'-P)
THYROID				
FT4	<5.15	10.60 – 19.40	pmol/L	ELFA
TSHs	>100,000	0.730 – 8.350	uIU/mL	ELFA
TORCH				

Anti-Toxoplasma IgM	0.06 (Negative)	Negative: <0.56; Pos: ≥ 0.65	-	ELFA
Anti-Toxoplasma IgG	0 (Negative)	Neg: <4; Pos >= 6	IU/mL	ELFA
Anti-Rubella IgG	0	Negative: <10	IU/mL	ELFA
Anti-Rubella IgM	0.12	Negative TV: <0.80	-	ELFA
Anti-CMV IgM	0.07	Negative: <0.70; Pos: >= 0.90	-	ELFA
Anti-CMV IgG	<4	Negative: <4; Positive: >=6	IU/mL	ELFA

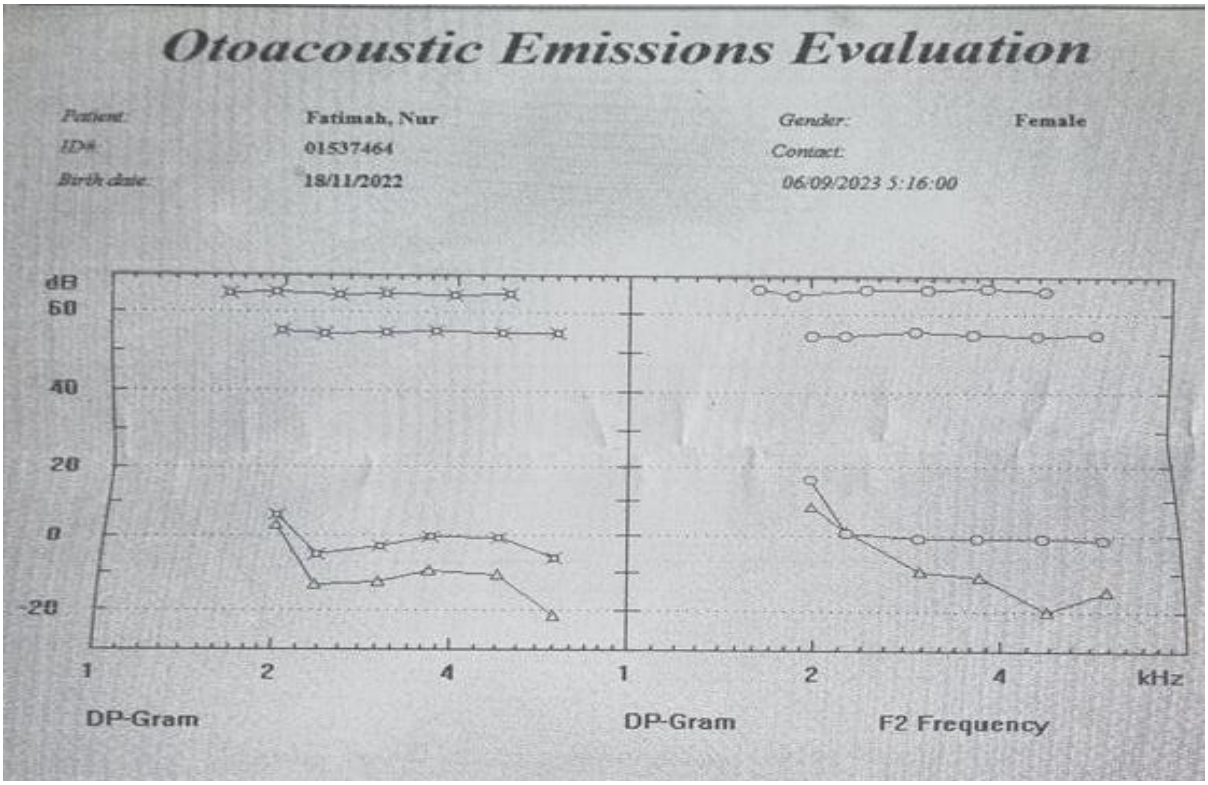

Figure 2. Hearing Screening Results

Table 3. Left Ear Audiometric Test

Result: PASS Ear: Left								
Protocol: 2–6 kHz; Screen, 4/6 for Pass Time: 5:09:02								
L1 (dB)	L2 (dB)	F1 (Hz)	F2 (Hz)	GM (Hz)	DP (dB)	NF (dB)	DP-NF (dB)	Result
65.1	54.9	4922	6,000	5,434	-6.5	-21.7	15.2	Pass
64.6	54.8	3,938	4,781	4,339	-0.7	-11.0	10.3	Pass
64.8	55.4	3,000	3,656	3,312	-0.6	-9.9	9.3	Pass
64.7	55.0	2,484	3,000	2,730	-3.2	-12.6	9.4	Pass
65.4	54.7	1922	2,344	2122	-5.4	-13.6	8.2	Pass
64.8	55.5	1,594	1969	1771	5.9	3.0	2.9	Reference

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Table 4. Left Ear Audiometric Test

Result: PASS Ear: Right								
Protocol: 2–6 kHz; Screen, 4/6 for Pass Time: 5:14:03								
L1 (dB)	L2 (dB)	F1 (Hz)	F2 (Hz)	GM (Hz)	DP (dB)	NF (dB)	DP-NF (dB)	Result
66.1	54.8	4922	6,000	5,434	-1.1	-14.8	13.7	Pass
66.9	54.6	3,938	4,781	4,339	-0.9	-20.1	19.2	Pass
66.5	54.8	3,141	3,750	3,432	-0.8	-11.2	10.4	Pass
66.3	55.7	2,484	3,000	2,730	-1.0	-9.7	8.7	Pass
65.1	54.5	1875	2,297	2075	0.6	0.9	-0.3	Reference
66.6	54.5	1641	2016	1818	16.4	8.1	8.3	Pass

Table 5. Laboratory Test Results After 2 Months of Levothyroxine Treatment

Test	Results	Reference Range	Unit	Method
THYROID				
FT4	<11.06	10.60 – 19.40	pmol/L	ELFA
TSHs	>12.906	0.730 – 8.350	uIU/mL	ELFA

RESULT AND DISCUSSION

Descriptive Analysis

Congenital hypothyroidism is a deficiency of thyroid hormones caused by the thyroid gland not forming properly, not forming at all, or by a disorder in the production or function of thyroid hormones present from birth. The global incidence of congenital hypothyroidism based on neonatal screening results is 1 in 3,000 to 1 in 4,000, whereas in the pre-screening era, the incidence was 1 in 6,700 live births. The female-to-male incidence ratio for congenital hypothyroidism is 2:1.6. However, if diagnosis and treatment are delayed, symptoms will develop gradually, characterized by macroglossia, umbilical hernia, and developmental delay.

The objectives of the congenital hypothyroidism screening program include early identification of infants with thyroid dysfunction, providing thyroid hormone treatment as soon as possible, preferably before the infant is 2 weeks old, and preventing long-term effects such as mental retardation, which cannot be reversed if treatment is delayed.

Table 6. Classification of Congenital Hypothyroidism

Classification	Type	Causes
Primary hypothyroidism	Permanent primary	Thyroid gland dysgenesis; dishormonogenesis
Primary hypothyroidism	Transient/temporary primary	Pregnant women undergoing radioactive iodine therapy; associated with congenital nephrotic syndrome; use of antithyroid drugs or goitrogens in pregnant women; iodine deficiency; exposure to iodine-containing substances; a mother with autoimmune thyroiditis; idiopathic
Secondary hypothyroidism	—	Anatomical defect in the midline of the central nervous system; destruction of the pituitary gland
Tertiary hypothyroidism	—	Multiple hypothalamic hormone deficiencies; head trauma; isolated TRH deficiency

In addition to the conditions mentioned above, hypothyroidism can be transient or permanent and is classified according to the location of the disorder: primary (in the thyroid gland) or secondary/central (in the pituitary and/or hypothalamus); severity of hypothyroidism (serum TSH levels >100 mIU/L are considered severe); and age at onset of hypothyroidism, with

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intrauterine cases being more severe. The most common form is permanent primary congenital hypothyroidism (elevated serum TSH levels) due to thyroid dysgenesis. In permanent congenital hypothyroidism, treatment must be continued for life, whereas treatment is not necessary for the transient form.

Table 7. Clinical Manifestations of Congenital Hypothyroidism by Age

Age	Clinical manifestations
2 weeks	Prolonged neonatal jaundice; edema; difficulty feeding; hypothermia; protruding abdomen; wide anterior fontanelle
>1 month	Dry skin; *failure to thrive*; constipation; lethargy
>3 months	*Delayed developmental disorder*; umbilical hernia; constipation; dry skin with carotenemia; macroglossia/enlarged tongue; myxedema; loud, hoarse voice
>6 months	*Delayed developmental disorder*; delayed linear growth; characteristic facial features; dry skin; deep, hoarse voice; macroglossia/large tongue; umbilical hernia
Older children	With or without goiter; delayed linear growth; short stature; delayed motor, mental, dental, and skeletal development, as well as delayed puberty; constipation; reduced physical activity; dry skin; myxedema

In this case, a 7-month-old infant presented with several clinical symptoms as listed above, namely *delayed developmental disorder and* slowed linear growth. In addition, the umbilicus was protruding, the patient had a characteristic facial appearance, macroglossia (enlarged tongue), short stature, and dry skin. The patient also experienced recurrent constipation. A diagnosis of congenital hypothyroidism in this patient was established based on clinical symptoms and thyroid function test results. In this case, FT4 and TSH testing was not performed until the patient was 7 months old, after the patient's mother complained that the child was unresponsive to the surrounding environment, had recurrent constipation, and exhibited motor delays, specifically an inability to sit up at the appropriate age. Based on these clinical symptoms, the doctor suspected congenital hypothyroidism and conducted a thyroid function evaluation.

In this patient, there was a delay in diagnosis because the family was unaware of the symptoms and the Newborn Screening Program (NSP) had not been conducted. However, in practice, the reach of the NSP in Indonesia remains limited and tends to be concentrated in large hospitals or urban areas. This limited coverage is influenced by various factors, including a lack of public awareness and education among the general public and healthcare workers, limited understanding among community health workers and pregnant women regarding the benefits of the newborn screening program, and the insufficient integration of the newborn screening program into antenatal care services at community health centers (Zizlavsky et al., 2023; Perry et al., 2017; Nishimwe et al., 2021; Miller et al., 2021). At birth, most infants with congenital hypothyroidism appear normal because they are still receiving maternal thyroid hormones transferred through the placenta. After maternal hormone levels decline within the first few days to weeks of life, the clinical manifestations of congenital hypothyroidism develop gradually, such as prolonged jaundice, lethargy, difficulty breastfeeding, constipation, macroglossia, umbilical hernia, hypotonia, and delayed growth and development if not treated promptly (Eng & Lam, 2020; Sonia et al., 2025).

By 7 months of age, infants are generally able to sit without assistance or with minimal assistance, reach for and transfer objects between hands, begin repetitive babbling (canonical babbling), and demonstrate interaction with their surroundings (Nasution et al., 2025; Zubler et al., 2022). In this case, the patient is not yet able to sit independently, has not begun babbling, and shows limited responsiveness to the environment, indicating delays in gross motor, language, and social development. These findings are consistent with the effects of thyroid hormone deficiency on central nervous system development, particularly the processes of myelination, neuronal migration, neuronal differentiation, and synapse formation during early life (Hartati et al., 2025; Rose et al., 2023). Thyroid hormone deficiency is also known to disrupt the processes of

myelination, neuronal migration, neuronal differentiation, and synaptogenesis, which play a crucial role in the maturation of the central nervous system (Bernal, 2007; Margareta, 2025; Sugihanawati, 2026).

Laboratory test results showed HB 9.3, FT4 <5.15, TSHs >100,000, and TORCH (-). These lab results indicate a very high TSH level and low FT4, supporting a diagnosis of primary congenital hypothyroidism. This pattern suggests a disorder of the thyroid gland, causing the pituitary to increase TSH secretion via a negative feedback mechanism; therefore, thyroxine must be administered immediately (Choirunnisa et al., 2026; Mukhlisatunnafsi, 2024). In this patient, the FT4 level increased more rapidly than the TSH level. This finding is consistent with the physiological response commonly observed in primary congenital hypothyroidism, in which FT4 levels typically improve within 1–2 weeks after levothyroxine administration, whereas TSH normalization takes longer due to the recovery of the hypothalamic-pituitary-thyroid axis. Therefore, TSH levels may remain elevated for several weeks to several months even after FT4 has returned to the normal range (van Trotsenburg et al., 2021).

Levothyroxine treatment should be initiated as soon as possible, no later than two weeks after birth or immediately after confirmation from thyroid function (serum) testing in the newborn (van Trotsenburg et al., 2021; Wardiah et al., 2025). These patients should also undergo early hearing screening, as thyroid hormone deficiency can have serious effects on the development of the auditory system. Low or absent hormone levels, such as those seen in congenital hypothyroidism, are often associated with hearing loss. However, the incidence of hearing loss among individuals with congenital hypothyroidism remains unclear; it is estimated to affect approximately 20% of patients, either alone or in conjunction with vertigo and tinnitus (Dewi et al., 2025). The patient's initial screening results showed that hearing in both the right and left ears was within normal limits.

Table 8. Levothyroxine Dosage by Age

Age	Levothyroxine (µg/kg/day)
0–3 months	10–15
3–6 months	8–10
6–12 months	6–8
1–3 years	4–6
3–10 years	3–4
10–15 years	2–4
>15 years	2–3

Patients are treated with levothyroxine (LT-4) at an initial dose of 10–15 mcg/kg/day. Levothyroxine can be administered orally after crushing the tablet and mixing it with water. It can be taken before or with a meal, provided it is taken in the same manner and at the same time every day. Levothyroxine must be taken as prescribed and should not be consumed with soy milk, iron, or calcium.

After two months of treatment, the TSH level decreased to >12.906 and FT4 increased to <11.06 (Table 5). These results indicate a significant improvement in FT4 and TSH levels, and the patient demonstrated developmental improvements, including increased activity, better responsiveness to the environment, the onset of babbling, and progress in gross motor skills, language, and personal-social development. This disorder is believed to be related to thyroid hormone deficiency, which plays a crucial role in central nervous system maturation, myelination, and synaptic development during the first year of life. The clinical improvement following levothyroxine administration demonstrates that adequate therapy can still benefit a child's development, even when the diagnosis is established after the neonatal period (Esposito et al., 2022; Wassner, 2017; Leung & Leung, 2019; Rodriguez et al., 2022; Casey et al., 2017).

In addition to hormone therapy, physical therapy is also considered important for stimulating the child's motor, sensory, and speech skills. Assessment of clinical response and

serum thyroxine levels is performed 2–4 weeks after the start of treatment, every 1–2 months during the first year of treatment, every 1–3 months during the second and third years of treatment, and every 6–12 months once growth is complete. Changes in the clinical signs and symptoms of hypothyroidism and whether growth and development are proceeding normally or not.8

Early detection through SHK leads to a better prognosis, whereas delayed detection of HK results in symptoms such as mental retardation, motor disorders, hypotonia, and ataxia.8 This case illustrates that a delay in diagnosis until 7 months of age resulted in significant impairments in growth and development. The positive hormonal response following levothyroxine administration underscores how crucial it is to recognize clinical symptoms early in infants who have not undergone screening for congenital hypothyroidism.

CONCLUSION

This case of congenital hypothyroidism in a 7-month-old infant with developmental delay and *stunting* demonstrates that thyroid dysfunction from the earliest stages of life can have far-reaching effects on a child's physical growth and development. Delayed diagnosis has the potential to cause growth retardation as well as motor, cognitive, and neurological developmental impairments that become increasingly difficult to correct as the child ages. The clinical manifestations of congenital hypothyroidism in early life can be nonspecific, making the condition at risk of going undetected through clinical observation alone. Therefore, screening for congenital hypothyroidism in newborns is a crucial step to enable early diagnosis and intervention. An evidence-based approach in these cases underscores the importance of integrating clinical findings, anthropometric measurements, developmental assessments, and thyroid function tests in determining diagnosis and management. Appropriate and sustained thyroid hormone replacement therapy is a key component in correcting hormone deficiency and supporting improved growth and development in children. For infants who have experienced developmental delays and *stunting*, management must not focus solely on hormonal correction but must also include monitoring of nutritional status, developmental stimulation, and periodic growth evaluations. Collaboration among physicians, nutritionists, rehabilitation specialists, and families is necessary to optimize treatment outcomes and address developmental delays. This case report underscores the importance of early detection of congenital hypothyroidism as part of efforts to prevent persistent growth and developmental disorders in children.

SUGGESTION

The implementation of newborn screening must be strengthened by increasing screening coverage, ensuring prompt follow-up on screening results, and educating parents on the importance of adherence to treatment and regular follow-up. Long-term evaluation of growth, neurocognitive development, nutritional status, and thyroid function is also necessary to comprehensively assess the success of therapy. Prompt, appropriate, consistent, and multidisciplinary management is expected to improve the prognosis and enhance the quality of life for children with congenital hypothyroidism.

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