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Malnutrition with Secondary Hypothyroidism in Children: A Case Report and Pathophysiological Review of Patients in One of the Hospitals in Surabaya

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Abstract

Malnutrition in children remains a global health problem that has a broad impact on growth, development and metabolic balance. One complication that receives less attention is secondary hypothyroidism, a condition that occurs due to metabolic adaptation to prolonged energy deficit. This situation can worsen the patient's prognosis and increase the risk of neurological complications and infections. This case report aims to describe the clinical course of a child with severe malnutrition who experienced secondary hypothyroidism and discuss the pathophysiological mechanisms underlying the link between malnutrition and thyroid dysfunction. Case: A 2 year 1 month old boy with a body weight of 5 kg (< -3 SD) and a height of 69 cm presented with complaints of diarrhea, fever, recurrent cough and episodes of seizures. Laboratory examination showed leukocytosis, increased CRP, and electrolyte disturbances. Evaluation of thyroid function revealed low FT4 levels (0.85 ng/dL) with depressed TSH (0.19 µIU/mL), leading to a diagnosis of secondary hypothyroidism. Metabolic adaptations due to malnutrition lead to decreased production of thyroid binding proteins as well as low TSH secretion in response to metabolic stress. This situation contributes to a slowdown in metabolism and worsens nutritional recovery, as well as increasing the risk of infectious and neurological complications. The literature supports that secondary hypothyroidism in children with malnutrition is reversible with optimal nutritional recovery. A multidisciplinary approach that includes clinical stabilization, electrolyte correction, and monitoring of thyroid function is very important in the management of children with malnutrition and secondary hypothyroidism.

Introduction

Malnutrition remains a global health problem that has a significant impact on children's growth and development, including the endocrine system. Prolonged malnutrition can cause disturbances in the hypothalamic-pituitary-thyroid (HPT) axis, which plays a role in metabolic regulation and the body's adaptation to chronic energy deficits. One consequence of this disorder is secondary hypothyroidism, which is a condition in which thyroid hormone production decreases due to impaired thyrotropin (TSH) secretion from the pituitary (Feldt-Rasmussen et al., 2021; Pirahanchi et al., 2023; Faglia & Beck-Peccoz, 2024). This condition not only slows metabolism, but also increases morbidity and mortality due to impaired immunity, growth retardation, and neurological deficits.

Epidemiologically, malnutrition remains a major problem in many developing countries. The World Health Organization (WHO) reports that around 45 million children under the age of five suffer from it, namely very low body weight compared to height, which can cause metabolic complications, including impaired thyroid function. Hypothyroidism secondary to malnutrition is often reversible with adequate nutritional restoration (Shulhai et al., 2024; Tahara et al., 1988; de Carvalho et al., 2018). However, if not treated immediately, this condition can cause persistent growth and development disorders and risks increasing morbidity and mortality rates in children.

Etiologically, secondary hypothyroidism in malnourished children is mainly caused by energy and protein deficiencies, which disrupt thyroid hormone regulation. Severe malnutrition inhibits the secretion of thyrotropin-releasing hormone (TRH) from the hypothalamus, which results in a decrease in TSH secretion from the pituitary, so that free thyroxine (FT4) levels in the blood decrease (Lee et al., 2015; Kluge et al., 2010; Feldt-Rasmussen et al., 2021; Teixeira et al., 2020). Apart from that, micronutrient deficiencies such as iodine, selenium and iron also play a role in disrupting thyroid hormone synthesis.

The pathogenesis of hypothyroidism secondary to malnutrition involves metabolic adaptation mechanisms aimed at conserving energy. One of the main mechanisms is the inhibition of the conversion of thyroxine (T4) to triiodothyronine (T3) in peripheral tissues, thereby increasing levels of reverse T3 (rT3), a biologically inactive form of thyroid hormone. In addition, impaired production of thyroid binding protein (thyroxine-binding globulin/TBG) due to liver dysfunction in children with poor nutrition also worsens thyroid hormone deficiency (Robinson et al., 2011; Sandeep, 2014). From a pathophysiological perspective, secondary hypothyroidism due to malnutrition occurs as the body's response to adapt to chronic energy deficits by reducing basal metabolism. Studies show that children with severe malnutrition have decreased expression of the enzyme deiodinase type 1 (D1) which converts T4 to active T3, while the enzyme deiodinase type 3 (D3) which converts T4 to rT3 is significantly increased (Dayal et al., 2014). This causes a slowing of metabolism that contributes to clinical symptoms such as bradycardia, hypothermia, and muscle weakness.

Clinically, the manifestations of hypothyroidism secondary to malnutrition vary depending on the severity of thyroid hormone deficiency. Symptoms that often appear include lethargy, hypotonus, growth retardation, and neurocognitive disorders due to impaired myelination in the central nervous system. Digestive disorders such as constipation and flatulence are also common, which further worsens the patient's nutritional status (Vernia et al., 2021; Kalaitzakis et al., 2006). The diagnosis of secondary hypothyroidism in children with malnutrition is made based on the history, physical examination and laboratory tests. The main characteristics on laboratory examination are low FT4 levels with TSH levels that are also low or low normal, which indicates a disruption of the HPT axis at the hypothalamic or pituitary level. Examination of rT3 levels can help differentiate this condition from primary hypothyroidism, where TSH levels are usually elevated.

The differential diagnosis of hypothyroidism secondary to malnutrition includes non-thyroidal disease syndromes (non-thyroidal illness syndrome/NTIS), congenital hypothyroidism, as well as hypopituitarism due to genetic disorders or brain injury (Dayal et al., 2014). Therefore, evaluation of other endocrine functions, such as cortisol and growth hormone levels, is often necessary to more accurately establish a diagnosis. Complications of secondary hypothyroidism due to malnutrition include permanent growth retardation, impaired neurodevelopment, and increased risk of infection due to immune system dysfunction (Mousikou et al., 2023). Some studies show that low thyroid hormone levels correlate with increased levels of proinflammatory cytokines such as IL-6 and TNF-α, which can worsen the immune response to infection (Bhutta et al., 2017; Croce et al., 2021). Management of hypothyroidism secondary to malnutrition focuses on nutritional restoration as the main

strategy. Studies show that with proper nutritional rehabilitation, thyroid function can return to normal without the need for external thyroid hormone supplementation (Lippi et al., 2023). However, in cases with severe thyroid hormone deficiency and significant clinical symptoms, administration of levothyroxine may be considered with close monitoring for the risk of refeeding syndrome.

The prognosis for hypothyroidism secondary to malnutrition is generally good with appropriate intervention, especially if diagnosis and treatment are carried out early. Children with optimal nutritional recovery tend to experience recovery of thyroid function within a few weeks to months. However, delayed intervention may increase the risk of long-term impaired cognitive development, which has implications for the patient's future quality of life. With increasing understanding of the relationship between malnutrition and secondary hypothyroidism, further research is needed to develop more effective clinical guidelines for the diagnosis and management of this condition. A multidisciplinary approach that includes gradual nutritional intervention, monitoring of endocrine function, and infection prevention strategies is needed to ensure optimal recovery for children at high risk of developing this disorder (Dike et al., 2022).

Methods

This case report and pathophysiological review examined the association between severe malnutrition and secondary hypothyroidism in a pediatric patient, with the aim of elucidating clinical manifestations, diagnostic findings, and underlying metabolic adaptations. The study was conducted at a hospital in Surabaya, Indonesia, and employed a detailed, systematic approach to data collection, analysis, and interpretation while maintaining ethical standards.

The patient under investigation was a 2-year-1-month-old boy presenting with severe malnutrition, characterized by extreme anthropometric deficits (weight: 5 kg, height: 69 cm, both below -3 SD) and clinical symptoms including chronic diarrhea, fever, recurrent respiratory infections, and seizures. His medical history revealed prior episodes of diarrhea triggered by formula changes, with no familial history of endocrine disorders or seizures. The selection of this case was based on the pronounced clinical and biochemical evidence of malnutrition-related metabolic dysfunction, making it a pertinent subject for studying secondary hypothyroidism.

Upon admission, a thorough physical examination was conducted, documenting signs of dehydration, poor peripheral circulation, and neurological abnormalities. Anthropometric measurements were recorded and plotted on WHO growth charts to confirm severe acute malnutrition. Laboratory investigations were performed to assess systemic and endocrine disturbances, including a complete blood count (CBC) to detect infections, C-reactive protein (CRP) levels to evaluate inflammation, and electrolyte panels to identify imbalances such as hyponatremia and hypokalemia. Thyroid function tests (TFTs), specifically free thyroxine (FT4) and thyroid-stimulating hormone (TSH), were pivotal in diagnosing secondary hypothyroidism, revealing low FT4 with suppressed TSH levels.

Diagnostic imaging, including a chest X-ray, was utilized to investigate potential pulmonary complications given the patient’s respiratory symptoms. Continuous clinical monitoring was implemented to track vital signs, hydration status, and neurological function, ensuring early detection of complications such as refeeding syndrome. The management protocol adhered to WHO guidelines for severe malnutrition, emphasizing gradual rehydration, cautious electrolyte correction, and controlled enteral nutrition using a specialized formula to prevent metabolic overload. Empiric antibiotic therapy was administered due to suspected bacterial infections, supported by radiological and laboratory findings.

To evaluate the reversibility of thyroid dysfunction, follow-up thyroid function tests were conducted after nutritional stabilization. The multidisciplinary care team, including pediatric endocrinologists and nutritionists, collaborated to optimize metabolic recovery and monitor

endocrine function. A comprehensive review of existing literature was integrated to contextualize the findings, focusing on mechanisms such as hypothalamic-pituitary-thyroid (HPT) axis suppression, altered peripheral thyroid hormone conversion, and the role of thyroid-binding proteins in malnutrition. This synthesis highlighted the adaptive and reversible nature of secondary hypothyroidism in malnourished children, reinforcing the importance of nutritional rehabilitation as the cornerstone of treatment.

Result and Discussion

The intersection of malnutrition and endocrine dysfunction represents a critical yet understudied domain in pediatric medicine, particularly in resource-limited settings where nutritional deficiencies remain prevalent. Malnutrition induces a cascade of metabolic adaptations aimed at conserving energy, often at the expense of vital physiological processes, including thyroid hormone regulation. Secondary hypothyroidism in this context arises from hypothalamic-pituitary-thyroid (HPT) axis suppression, characterized by diminished thyrotropin-releasing hormone (TRH) secretion, blunted thyroid-stimulating hormone (TSH) response, and reduced peripheral conversion of thyroxine (T4) to triiodothyronine (T3). This endocrinopathy exacerbates the clinical sequelae of malnutrition—growth failure, neurodevelopmental delay, and immunosuppression—while complicating nutritional recovery due to its catabolic effects.

The present study examines these mechanisms through a paradigmatic case of severe protein-energy malnutrition (PEM) complicated by secondary hypothyroidism in a 25-month-old male. By correlating anthropometric deficits with biochemical and hormonal profiles, we elucidate the bidirectional relationship between nutritional deprivation and thyroid dysfunction. The case underscores the clinical imperative to recognize and address endocrine disturbances in malnourished children, as timely intervention may mitigate long-term morbidity. The following results delineate the patient's clinical trajectory, emphasizing the reversibility of thyroid dysfunction with nutritional rehabilitation and the implications for therapeutic strategies in analogous cases.

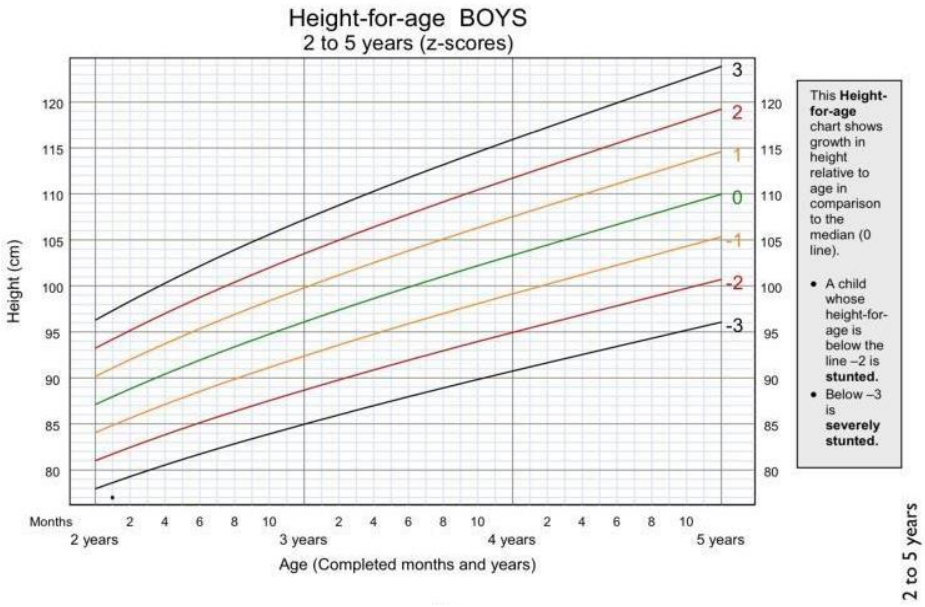

Figure 1. Height-for-age boys

This figure illustrates the patient's height-for-age trajectory plotted against WHO growth standards. The child's height (69 cm) falls below the -3 standard deviation (SD) line, indicating severe stunting, a hallmark of chronic malnutrition. Stunting results from prolonged nutrient deprivation, recurrent infections, and metabolic disturbances, all of which impair linear

growth. The comparison with normative percentiles (e.g., 3rd, 50th, 97th) underscores the severity of growth retardation. Given the patient's age (2 years 1 month), this deviation suggests long-standing nutritional deficiencies, likely compounded by secondary hypothyroidism, which further suppresses growth hormone activity and bone maturation.

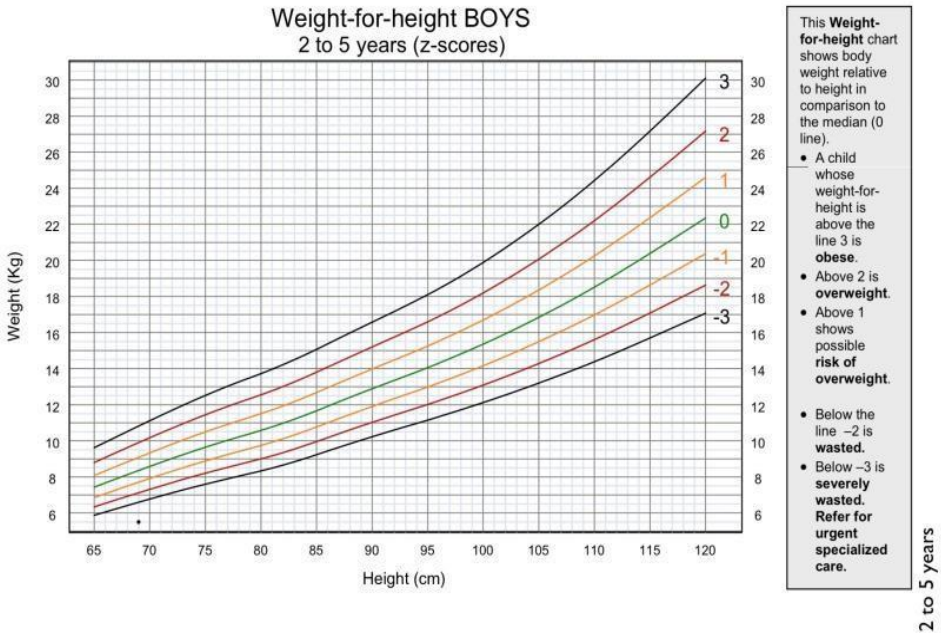

Figure 2. Weight-for-height boys

The weight-for-height graph demonstrates the child's acute malnutrition status. His weight (5 kg) relative to height is critically low, placing him below the -3 SD cutoff, consistent with severe wasting. Wasting reflects acute energy deficiency and catabolism, often exacerbated by infections (e.g., diarrhea, respiratory illnesses). The flat or declining curve suggests ongoing metabolic stress, where the body breaks down muscle and fat for energy. Notably, secondary hypothyroidism worsens wasting by reducing basal metabolic rate (BMR), slowing tissue repair, and impairing protein synthesis. This figure highlights the urgency of nutritional rehabilitation to reverse catabolism.

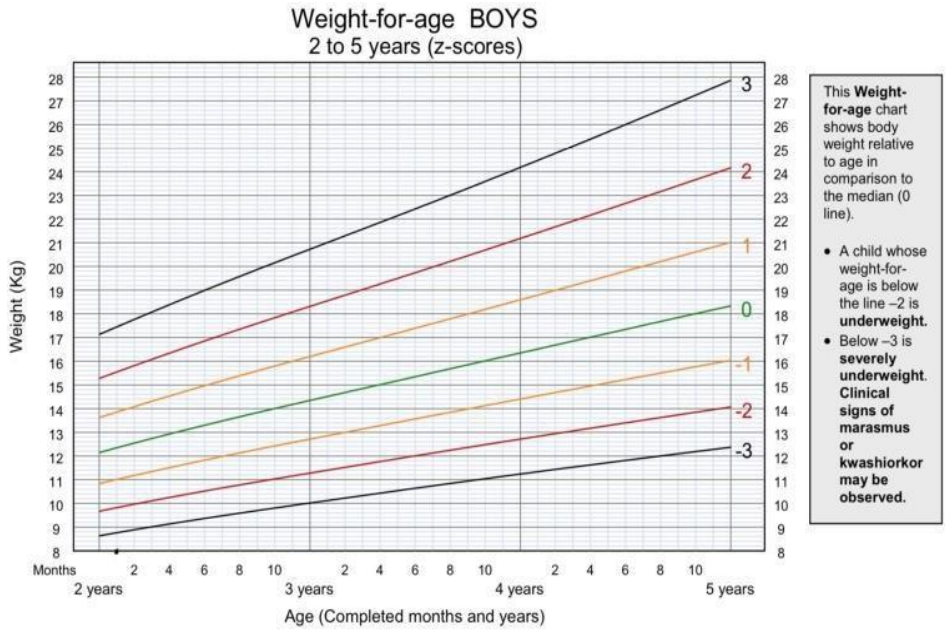

Figure 3. Weight-for-age boys

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Weight-for-age is a composite indicator of both acute and chronic malnutrition. The patient's weight (5 kg) is below -3 SD, classifying him as severely underweight. This deficit reflects cumulative impacts of inadequate caloric intake, malabsorption (from chronic diarrhea), and endocrine dysfunction. The divergence from expected growth curves (e.g., 50th percentile) signals prolonged nutritional deprivation. Secondary hypothyroidism exacerbates this by blunting appetite and slowing anabolic processes. The graph's utility lies in tracking recovery; post-intervention weight gain would suggest improved metabolic and thyroid function.

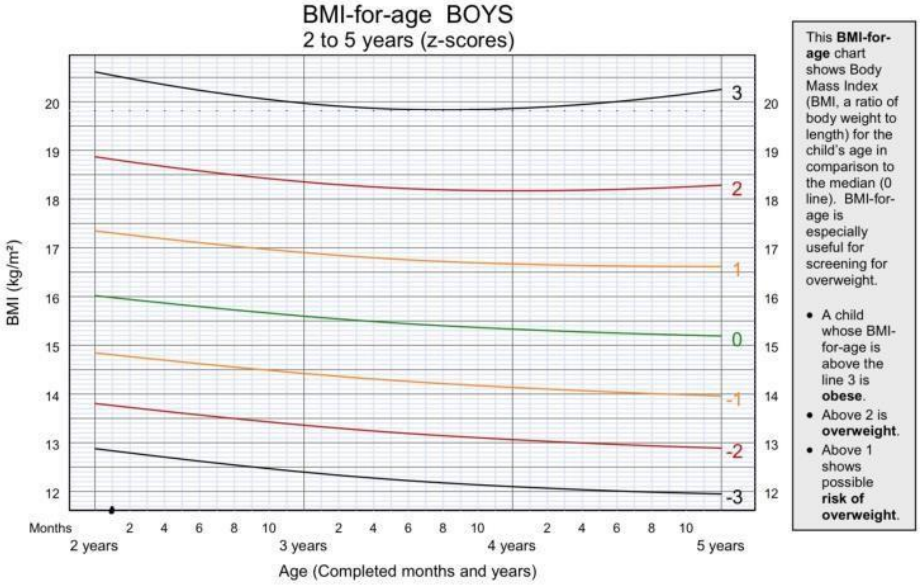

Figure 4. BMI-for-age boys

An. MM / boy / 2 years 1 month / 5 kg / 69 cm
BB/U : <-3 SD (*underweight* weight)
TB/U : < -3 SD (very short)
BMI : 10.5
BMI/U : 10.5 (< -3 SD) P 75th (*underweight*)
Nutritional status : *underweight*

The BMI-for-age plot (BMI = 10.5 kg/m², < -3 SD) confirms severe thinness, aligning with wasting and stunting. BMI is a critical metric in malnutrition as it integrates weight and height deficits. The extremely low BMI underscores systemic metabolic adaptation, where energy conservation mechanisms (e.g., reduced T3, elevated reverse T3) dominate. This figure also indirectly reflects the risk of complications like hypoglycemia and infections, as low BMI correlates with weakened immunity and impaired organ function. Monitoring BMI during treatment helps assess nutritional recovery and thyroid hormone normalization. Laboratory investigations revealed leukocytosis, increased C-reactive protein (CRP) which indicated an active inflammatory process, as well as electrolyte disturbances in the form of hyponatremia (130 mmol/L) and hypokalemia (2.5 mmol/L). Thyroid function evaluation revealed low FT4 levels (0.85 ng/dL) with depressed TSH (0.19 µIU/mL), consistent with a diagnosis of secondary hypothyroidism.

Patient management is carried out with rehydration therapy according to WHO guidelines for malnutrition, gradual correction of electrolytes, and provision of enteral nutrition with a special formula for children with severe malnutrition. The patient also received empiric antibiotic therapy due to suspicion of bacterial infection as seen on radiological examination of the chest, as well as close monitoring of neurological status considering the history of seizures.

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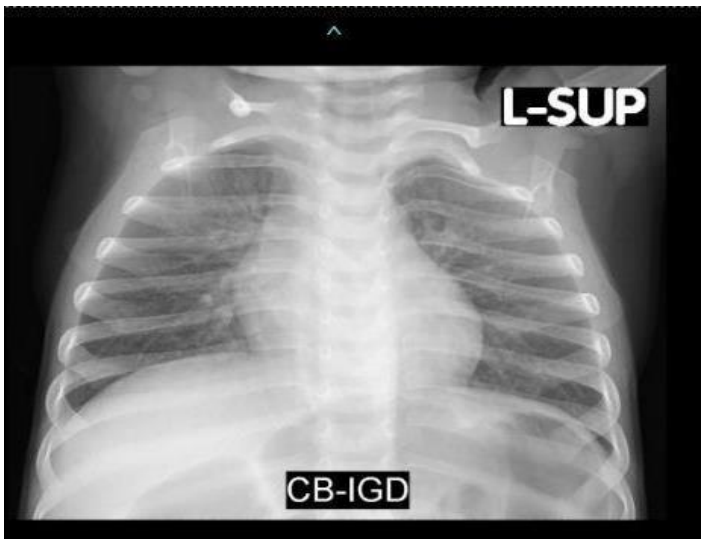

Figure 5. Radiological Interpretation of Neonatal Thorax with Evaluation of Lung Position and Condition

During treatment, the patient showed gradual clinical improvement with an increase in body weight of up to 6.5 kg within two weeks. Bowel pattern improved, and there were no further episodes of seizures during hospitalization. The results of evaluation of thyroid function after improving nutritional status showed an increase in FT4 levels, indicating that the thyroid dysfunction that occurred was reversible and related to severe malnutrition. Secondary hypothyroidism that occurs in children with poor nutrition is part of the body's adaptive response to chronic energy deficits. This condition can be explained by a metabolic adaptation mechanism that causes a decrease in thyroid hormone synthesis in an effort to conserve energy. A decrease in FT4 levels with low TSH indicates a disruption in the regulation of the hypothalamic-pituitary-thyroid (HPT) axis, which is also often found in conditions of severe malnutrition and other systemic diseases. Severe malnutrition has a significant impact on thyroid hormone regulation through several mechanisms, including decreased expression of deiodinase type 1 (D1) which is responsible for the conversion of T4 to T3 in peripheral tissues (Robinson et al., 2011). This decrease in D1 activity leads to the accumulation of inactive rT3, thus worsening the secondary hypothyroid state. In addition, disturbances in thyroid hormone transport due to low levels of thyroid binding proteins such as TBG (thyroxine-binding globulin) are also found in patients with poor nutrition.

Cildren with protein-energy malnutrition showed significant changes in thyroid hormone levels, including decreased T3 and increased rT3, indicating impaired peripheral conversion of thyroid hormone. This is in line with the concept of non-thyroidal illness syndrome (NTIS), where the body experiences a decrease in basal metabolism as a form of protection against prolonged nutritional stress. The decrease in TSH production observed in this case is most likely related to impaired TRH (thyrotropin-releasing hormone) secretion from the hypothalamus due to prolonged energy deficit (Warner & Mittag, 2012; Sainsbury & Zhang, 2012; Mariotti & Beck-Peccoz, 2021; Sawicka-Gutaj et al., 2022). The decrease in T3 and FT4 levels that occurs in conditions of malnutrition can be corrected through adequate nutritional intervention. A study by (Geme, 2020) shows that restoration of nutritional status can restore thyroid function without the need for external thyroid hormone supplementation. Adequate enteral nutrition therapy was able to increase FT4 and T3 levels within 4-6 weeks, confirming that thyroid disorders in malnutrition are reversible.

Apart from the impact on the metabolic system, secondary hypothyroidism can also affect the immune system and inflammatory response. In research by Economidou et al. (2011), it was found that impaired thyroid function in critically ill patients was associated with an increased

risk of infection and impaired tissue healing. This is supported by a recent study from Rodríguez-Pérez et al. (2022) who found that low thyroid hormone levels were associated with increased inflammatory biomarkers such as IL-6 and TNF-α in patients with severe malnutrition. Hypothyroidism also contributes to growth and development disorders in children with poor nutrition. Long-term thyroid hormone deficiency is associated with delayed bone growth and impaired brain development in children. A longitudinal study by (Lischinsky et al., 2016) showed that children with hypothyroidism secondary to malnutrition had a significant reduction in cerebral cortex volume compared to healthy controls.

Providing thyroid hormone therapy to children with secondary hypothyroidism due to malnutrition is still a matter of debate. Some studies show that L-T4 supplementation can accelerate the recovery of metabolic function, but in conditions of severe malnutrition, a rapid increase in thyroid hormone can increase the risk of refeeding syndrome. Therefore, current recommendations emphasize a gradual nutritional approach as the main strategy in restoring thyroid function. This case confirms that impaired thyroid function in children with malnutrition is reversible and closely related to prolonged energy deficit. Appropriate nutritional intervention and close monitoring of endocrine function are the main keys in the management of children with this condition. Further studies are needed to better understand the pathophysiological mechanisms and clinical implications of secondary hypothyroidism in the malnourished pediatric population.

Conclusion

Secondary hypothyroidism in children with malnutrition is a complex condition and involves various metabolic adaptation mechanisms aimed at conserving energy. This case shows that impaired thyroid function in children with malnutrition is reversible and can improve with improved adequate nutrition. Early diagnosis and monitoring of thyroid function is very important to prevent long-term complications that can affect a child's growth and development. The primary treatment in these cases is gradual nutritional intervention with close monitoring of metabolic status and thyroid function. Additional thyroid hormone therapy should be considered on an individual basis, taking into account the risks and benefits. Further research is needed to establish clearer clinical guidelines in the management of hypothyroidism secondary to malnutrition, especially in the context of the role of thyroid hormones on metabolic and neurological recovery. As understanding of the relationship between malnutrition and thyroid disorders increases, it is hoped that a more integrated clinical approach can be implemented to improve outcome health of children experiencing severe malnutrition. In the future, more extensive research and clinical trials are needed to optimize intervention strategies and ensure better recovery for patients with similar conditions.

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