Original Research Article

The functioning of Thyroid among severely malnourished children

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Received: 29-10-2021 / Revised: 19-11-2021 / Accepted: 12-12-2021

Abstract

Background: Protein energy malnutrition (SAM) continues to be a major public health problem in the tropical and subtropical regions of the world. It affects several aspects of the secretion and metabolism of thyroid hormones. The present study has been conducted to study the effect of SAM on thyroid hormone. **Objective**: To study the level of T3, T4, TSH in severely malnourished children and healthy control. **Design:** Case-control study. **Methods:** The children in the study are very malnourished and range in age from 6 months to 60 months. There was a total of 40 cases in total. Details were gathered using a pre-made proforma. The levels of triiodothyronine (T3), thyroxine (T4), and thyroid stimulating hormone (TSH) were measured. Using an appropriate statistical technique, the parameters were compared between cases and controls. **Results:** T3 and T4 levels were significantly low in SAM children as compared to controls. TSH levels were similar in both groups. **Conclusions:** T3 and T4 levels are lower in those with severe acute malnutrition (SAM), but TSH levels remain unchanged. The altered thyroid hormone level seen in children with PEM could be a protective mechanism, limiting protein catabolism and lowering energy demands.

Keywords: Severe Acute Malnutrition, Thyroxine, Triiodothyronine, Thyroid stimulating hormone.

Introduction

Protein Energy Malnutrition (PEM) is one of the most common dietary problems in underdeveloped nations, and it is a primary cause of childhood mortality and morbidity, resulting in long-term physical and mental damage[1,2]. According to estimates, there are around 162 million children worldwide that suffer from PEM in various forms. PEM is thought to be the primary or secondary cause of about half of the 3 million fatalities in children under the age of five. South Asia and Sub-Saharan Africa are home to three-quarters of the world's stunted children, while India is home to approximately one-third of the world's malnourished children[3].

Malnutrition is defined as "the cellular mismatch between the supply of nutrients and energy and the body's demand for them to maintain development, maintenance, and certain functions," according to the World Health Organization (WHO). Because the body's supply of protein and energy is limited in PEM, it attempts to utilise them more efficiently by lowering basal caloric expenditure[2]. PEM has been linked to several endocrine disorders, including alterations in growth hormone, insulin, glucocorticoids, and thyroid hormones. Changes in thyroid homeostasis have received insufficient attention. Thyroid hormone secretion and metabolism, as well as the anatomy of the thyroid gland, are all altered in PEM (Abroe et al)[6]. This results in a reduction of the activity of the gland[7], as the body tries to adapt to low calorie intake.

Materials and methods

The research was carried out at Fakir Mohan Medical College and Hospital, Balasore ,Medicine OPD. It included 40 youngsters ranging in age from 6 months to 60 months. These children were included based on WHO's severe acute malnutrition criteria, which included children with weight for height (W/H) or length (W/L) with a Z score less than 3 standard deviations, and/or W/H or W/L with a Z score less than 2 SD and a mid-upper arm circumference (MUAC) of 11.5% and/or bilateral pitting edoema.

After obtaining informed written agreement from parents/guardians, subjects were included in the current study. The information was entered into a pre-designed proforma. An anthropometric assessment and a systemic examination were carried out. Weight was recorded to the nearest 100g, length/height of the child was measured to the nearest cm, mid arm circumference (MAC) was measured to the nearest mm.

40 controls matched for age and sex were taken from children of normal weight for height/age attending hospital for either checkup for minor ailments or brother and sister of patients admitted to the pediatric ward. All children with a maternal history of thyroid dysfunction, children with clinical evidence of endocrinal abnormality, especially thyroid and infants with clinical goitre were excluded from the study. 3ml of venous blood was taken aseptically and stored in an EDTA (Ethylene Diamine Tetraacetic Acid) vacutainer and test tube. The serum obtained from a blood sample spun at 5000 rpm (rotation per minute) for 5 minutes was used to calculate T3, T4, and thyroid stimulating hormone (TSH). T3, T4, and TSH were calculated using the chemiluminescence method.

The data was entered into a Microsoft Excel spreadsheet, and the results were expressed as mean standard deviation (SD) for continuous variables and percent (%) for categorical data. Epi Info software version was used to statistically evaluate the observations. For categorical data, descriptive statistics were used. We utilised a one-way ANOVA and an independent sample t-test. A statistically significant P value of 0.05 was used.

Results

Table 1: Mean value of serum T3

	Range (ng/dl)	Number	T3 mean (ng/dl)	t-value	p-value
Control	110-192	40	142.3		
Cases	**69-172**	**40**	**106.40**	**2.79**	**<.01**

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Table 2: Mean value of serum T4

	Range (µg/dL)	Number	T4 mean(µg/dL)	t-value	p-value
Control	7.28-9.1	40	8.12		
Cases	**5.01-8.92**	**40**	**7.04**	**2.53**	**<.02**

The study involved 40 children ranging in age from 6 months to 60 months. Males made up 25 of the children in the study, while females made up 15 of them (sex ratio M: F:1.6:1). [Table 1,2,3] shows the mean T3, T4, and TSH values of patients and controls. The mean T3, T4, and TSH levels in the SAM group were 106.4ng/dL, 7.04g/dL, and 2.28 mIU/L, respectively, while the controls had 143.2ng/dL, 8.12g/dL, and 2.66mIU/L. When compared to controls, mean T3 and T4 levels were considerably lower in cases (p0.05 for both parameters). Cases and controls had essentially equal mean TSH levels.

Discussion

Thyroid gland shape and function are altered as a result of protein energy deficiency[8]. As previously indicated, the effects of PEM on the body are complex, affecting nearly all organ systems[3]. SAM is linked to a decrease in plasma protein synthesis. Hormones play a crucial part in PEM's energy and protein metabolism[10]. The purpose of this study was to determine the levels of thyroid hormones in children with SAM and compare them to age-matched controls. The mean T3 and T4 levels in the current study (Table 1, 2) were substantially lower in cases compared to controls (p0.05). Graham et al (1973), has undertaken research[11] Ingenbleek (1986)[4] Turkay'at al[15]. and Abrol et al[16]. also showed similar inference. Similar results are reported by a study done by Kumar S et al[12]. Orbak Z et al[13]. and a study conducted by Das BK et al[14]. found that mean T3 levels were significantly lower in malnourished children as compared to controls, however, in their study they found no significant difference in mean T4 levels of cases and controls. Low T3 levels in children with PEM are likely due to low binding proteins, impaired thyroxine monodeiodination in the liver, which results in the decreased peripheral conversion of T4 to T3, and elevated corticosteroids, which are commonly seen in children with malnutrition (act by inhibiting the 5' deiodinase system). Low T4 levels in children with PEM are likely due to a decrease in thyroid secretion rate, depletion of reserves, and failure of the mean TSH levels in patients and controls were similar in this investigation. In contrast to the current investigation, Orbak Z et al discovered that mean TSH levels of children with PEM were greater than controls. T4 undergoes intracellular monodeiodination to produce T3 at the pituitary level, causing negative feedback inhibition, resulting in normal TSH levels in children with PEM.

Conclusion

To summarise, protein energy deficiency is linked to lower T3 and T4 levels without a change in TSH levels. The altered thyroid hormone level seen in children with PEM may be a defence mechanism against excessive metabolic activation and energy use, protecting malnourished children with low calorie reserves from dying young. Normal TSH levels are explained by the fact that T4 undergoes intracellular monodeiodination to T3 at the pituitary level, preserving the central feedback system and permitting optimal thyroid adaptation.

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Conflict of Interest: Nil Source of support: Nil