Original research article

A study on micronutrient deficiency in children with protein energy malnutrition

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Abstract

Two major clinical syndromes, kwashiorkor and nutritional marasmus are widely recognized. These syndromes constitutes only tip of iceberg of the problem of the problem of protein energy malnutrition. Children's also suffer from mild to moderate malnutrition while they hidden in the community. Several theories have been put forward to explain the etiology of the severe forms of PEM. WHO criteria was used to define protein energy malnutrition. Detailed history and systemic examination was done. The clinical signs of micronutrient deficiency such as bitot's spots, xerophthalmia, keratomalacia and corneal opacities in vitamin A deficiency, glossitis, angular stomatitis, seborrheic dermatitis in vitamin B deficiency, anorexia, pallor, irritability, bleeding from gums and conjunctiva in vitamin C deficiency, deformities in forearm, bow legs (genu varum) or knock knees (genu valgum), craniotabes, poor growth in height and weight, frontal bossing of the skull, swelling of the wrist and knees in vitamin D deficiency. Poor physical growth, delayed sexual maturation anemia, anorexia, diarrhea hair loss dermatitis impaired immune response, poor wound healing in Zinc deficiency was assessed. In our study, the prevalence of anemia is 74.88%, prevalence of Vit. D deficiency 32.70%, Vit. B deficiency 30.81% and Vit.

Keywords: Micronutrient deficiency, children, protein energy malnutrition

Introduction

It is obvious that malnutrition occurs because of a deficiency in protein and calorie intake. There are two distinct entities; the edematous malnutrition represented by kwashiorkor and marasmic kwashiorkor and non-edematous malnutrition represented by marasmus. The reasons for the differing manifestations are still not very clear; however, various theories have been proposed^[1].

Two major clinical syndromes, kwashiorkor and nutritional marasmus are widely recognized. These syndromes constitutes only tip of iceberg of the problem of the problem of protein energy malnutrition. Children's also suffer from mild to moderate malnutrition while they hidden in the community. Several theories have been put forward to explain the etiology of the severe forms of PEM^[2].

The development of marasmus and kwashiorkor has been linked to difference in diet and metabolism. The term was used because the marasmus was thought to be due to energy deficiency and kwashiorkor was thought to be due to deficiency of proteins. Marasmus was understood a response from starvation and shortage of energy leading to wasting of muscle and subcutaneous fat. This development was considered as physiological adaptive process, as energy is unavailable from food, energy is made available from the body^[3]. Through wasting important body structures are preserved, muscle protein is broken down and amino acids become available which are important for the synthesis of glucose in particular, in the early phases of energy deficiency, glucose is crucial for brain metabolism. Children with marasmus are characterized by an absence of metabolic abnormalities and they are able to maintain homeostasis even during long periods of little or no food ^[4]. This is because through the process of wasting important amino acids and other compounds crucial for homeostasis become available. This hypothesis was supported by findings showing that children with marasmus have normal levels of serum albumin. Furthermore the concentration of lipoprotein is upheld in marasmus and fatty liver does not therefore develop in marasmus. According to protein energy hypothesis, children who develop kwashiorkor unlike marasmic children fail to maintain homeostasis. Kwashiorkor was seen as a result of children consuming a diet high in energy but low in protein. It was suggested that this consumption pattern lead to pathological abnormalities such as fatty liver and edema. A shortage of dietary protein means that the amount of amino acids required for essential synthesis is inadequate. [35] The protein energy hypothesis has been challenged. A longitudinal study comparing the diet of Indian children with

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marasmus and kwashiorkor, did not find any differences in the intake of energy and protein in children with marasmus and kwashiorkor^[5, 6].

Methodology

Study design: Hospital based observational study.

Study population: All Children of age 6 months to 60 months admitted to the Pediatric ward.

Sample size: All Children of age 6 months to 60 months admitted to the Pediatric ward fulfilling the inclusion criteria, during the study period, was included in the study.

Inclusion criteria

All the children of 6 months to 60 months having protein energy malnutrition according to WHO criteria.

Exclusion criteria

- Children of age < 6 months and >60 months.
- Children suspected to have congenital malformation.
- Children with genetic disorder including thalassemia.
- Patient party not giving consent for examination.

WHO criteria was used to define protein energy malnutrition. Detailed history and systemic examination was done. The clinical signs of micronutrient deficiency such as bitot's spots, xerophthalmia, keratomalacia and corneal opacities in vitamin A deficiency, glossitis, angular stomatitis, seborrheic dermatitis in vitamin B deficiency, anorexia, pallor, irritability, bleeding from gums and conjunctiva in vitamin C deficiency, deformities in forearm, bow legs (genu varum) or knock knees (genu valgum), craniotabes, poor growth in height and weight, frontal bossing of the skull, swelling of the wrist and knees in vitamin D deficiency. Poor physical growth, delayed sexual maturation anemia, anorexia, diarrhea hair loss dermatitis impaired immune response, poor wound healing in Zinc deficiency was assessed. We looked for clinical evidence of associated infections like acute gastroenteritis, acute respiratory infections, meningitis, sepsis, tuberculosis malaria, measles, skin infections. Investigations was done as and when necessary.

Results

Micronutrient Deficiencies	Number (n)	Percentage (%)
Vitamin A	63	29.86
Vitamin B	65	30.81
Vitamin C	28	13.27
Vitamin D	145	68.72

Table 1: Micronutrient Deficiencies

In present study Vitamin D deficiency accounts for 68.72%, followed by Vitamin B 30.81%, Vitamin A 29.86% and Vitamin C which accounts for 13.27%.

Miana N-4-10-14	Tatal	Age Group (in months)									
Micro-Nutrient		6-12		12-23		24-35		36-47		48-60	
Deficiency	(n)	n	%	n	%	n	%	n	%	n	%
Vitamin A	63	7	11.11	24	38.10	6	9.52	14	22.22	12	19.05
Vitamin B	65	5	7.69	18	27.69	16	24.62	15	23.08	11	16.92
Vitamin C	28	4	14.29	9	32.14	5	17.86	5	17.86	5	17.86
Vitamin D	145	15	10.34	45	31.03	29	20.00	23	15.86	32	22.07

Table 2: Micronutrient Deficiency According to Age Group

Table 3: PEM According to Micronutrient Deficiency

	T-4-1	Micronutrient Deficiency									
Grade	Total	Vit	Vitamin A		Vitamin B		amin C	Vitamin D			
	(n = 211)	n	%	n	%	n	%	n	%		
	Stunting										
Moderate	145	39	26.90	44	30.34	23	15.86	99	68.28		
Severe	66	24	36.36	21	31.82	5	7.58	45	68.18		
Under Weight											
Moderate	145	39	26.90	44	30.34	23	15.86	99	68.28		

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Severe	66	24	36.36	21	31.82	5	7.58	45	68.18
Wasting									
Moderate	83	19	22.89	24	28.92	11	13.25	56	67.47
Severe	41	15	36.59	13	31.71	2	4.88	29	70.73

Anaemia	Number (n)	Percentage (%)
Present	158	74.88
Absent	53	25.12
Total	211	100.00

Table 4: Anaemia

In our study Anaemia present in 74.88% children with protein energy malnutrition and absent in 25.12% children.

Discussion

In our study, the prevalence of anemia is 74.88%, prevalence of Vit.D deficiency 32.70%, Vit.B deficiency 30.81% and Vit.A deficiency 29.86%. The study done by Kumar R^[7] also shows the prevalence of vitamin D (15.4%) is more common followed by vitamin B (14.4%) And vitamin C (1.9%).Study done by Arya A K^[8] also shows the prevalence of anemia (95%) Vitamin D (25%), Vitamin B (10.5%), and Vitamin A (12%) in his study.

Conclusion

The most common micro vitamin deficiencies were Vitamin D, Vitamin B, Vitamin A. of Vitamin D 32.70%, Vitamin B 30.81% and Vitamin A 29.86% and 74.88% children affected with anaemia.

References

- 1. Hulst JK, Joosten L, Zimmermann Hop W, Van Buuren S, Buller H, *et al.* Malnutrition in critically ill children: from admission to 6 months after discharge. Clin Nutr., 2004, 223-232.
- 2. Pinstrup-Andersen P, Burger S, Habicht JP, Peterson K. Protein-energy malnutrition. In Disease control priorities in developing countries, 1993.
- 3. Zere E, McIntyre D. Inequities in under-five child malnutrition in South Africa. Int. J Equity Health. 2003;2:7.
- 4. Duggan B WJ, aW WA. Nutrition in Pediatrics. Basic science, clinical application, 2004.
- 5. Neumann GC, Jelliffe DB, Zerfas AJ, Jelliffe EF. Nutritional assessment of the child with cancer. Cancer Res. 1982;42:699s-712s.
- 6. Goulet O, Lebenthal E, Branski D, Martin A, Antoine JM, Jones PJH. Nutritional solutions to major health problems of preschool children: how to optimise growth en development. J Pediatr Gastroenterol Nutr. 2006;(3-43):1-3.
- 7. Kumar R, Singh J, Joshi K, Singh HP, Bijesh S. Co-morbidities in Hospitalized Children with Severe Acute Malnutrition. Indian Pediatrics. 2014 Feb;51:125-127.
- 8. Arya AK, Lal P, K. Co-morbidities in Children with Severe Acute Malnutrition-A Tertiary Care Centre Experience. IJCMR, 2017 May, 4(5).