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Serum Albumin Level in Sudanese Children with Edematous and Non-Edematous Malnutrition

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Abstract

Introduction

Edema formation in malnourished children is multifactorial and not necessarily caused by hypoalbuminemia. In Sudan, studies that evaluate serum albumin level in malnourished children are scarce, if any. The aim of this study is to assess serum albumin concentrations in Sudanese children with edematous and non-edematous malnutrition.

Material and Methods

The study involved 23 males matched for age and weight with 17 females. The ages of studied subjects ranged between 6 – 36 months. Weight and serum albumin levels were measured according to the standards. Patients were classified according to the presence of edema, Wellcome and Gomez classifications. In addition, patients were classified according to their levels of albumin into normal (3.5 - 4.8 g/dl), mild (2.8 - 3.4 g/dl), moderate (2.1 - 2.7 g/dl) and severe (< 2.1 g/dl) hypoalbuminemia.

Results

Serum albumin levels were significantly lower in malnourished subjects with edema (M±SD = 2.4±0.8 g/dl) compared to those with malnutrition but no edema ((M±SD = 3.1±0.8 g/dl, P = 0.026) However, classifying studied malnourished children according to their albumin levels revealed no significant differences in the mean of albumin when albumin levels of edematous patients were compared with non-edematous subjects in each class (P> 0.05). There was no significant correlation between weight and serum albumin level (correlation coefficient (CC) = 0.156, P = 0.337).

Conclusion

The current study demonstrates the tendency of edematous malnourished children to have hypoalbuminemia; nevertheless, some children with significantly low albumin are still not suffering from edema. These paradoxical results display the possible variations in the pathophysiological response to food deprivation.

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INTRODUCTION

World health organization (WHO) defines severe acute malnutrition as a very low weight for height (below 3Z scores of the median WHO growth standards), by visible severe wasting, or by the presence of nutritional edema[1].There are many classifications for malnutrition. In Gomez classification, the child's weight is compared to that of a normal child (50th percentile) of the same age. Accordingly three categories can be distinguished: mild (75%-90% weight for age); moderate (60%-74% weight for age) and severe (<60%weight for age) malnutrition[2]. Alternatively, Wellcome classification considers both weight and presence edema as follows: kwashikor (weight for age 60-80% with edema), undernutrition (weight for age 60-80% without edema), marasmic-kwashiorkor (weight for age < 60% with edema) and marasmus (weight for age < 60% without edema) [3]. The major clinical syndromes of severe childhood malnutrition (SCM) are marasmus (non-edematous SCM), kwashiorkor and marasmic-kwashiorkor (edematous SCM).

Edema formation in edematous malnutrition is multifactorial. Hypoproteinemia and electrolytes disturbance are claimed to be among the major contributing factors [4]. These facts were supported by the work of Gopalan and others [5, 6] who demonstrated clinical and biochemical cure of Kwashiorkor patients with diet rich in protein. However, a study conducted by Golden et al showed no difference in the concentration of plasma albumin before and after the loss of edema when patients were put on restricted diet [7]. Furthermore Golden demonstrated improvement of edema in these children treated with high energy diets and not with protein rich meals [8], it was reported that Indian children on poor diet can develop either kwashiorkor or marasmus regardless of the quantitative or qualitative differences in their diet [9]. Chronic food deprivation children with non-edematous malnutrition can maintain body protein breakdown at the same rate as when they are well nourished but children with edematous malnutrition cannot [10].

Nutritional edema is associated with increased secretion of an antidiuretic substance that prevent normal execratory response to water intake[11].The functional and structural hepatic changes associated with malnutrition may lead to in activation of antidiuretic (ADH) and aldosterone hormones and hence water retention[12]. Other factors that may contribute to nutritional edema include antioxidant depletion[13-15], low hepatic transaminases[16], excessive lipid peroxidation[17], free circulating iron[18] and ferritin[19].

In Sudan, studies that evaluate serum albumin level in malnourished children are scarce, if any. The aim of this study is to assess serum albumin concentrations in Sudanese children with edematous and non-edematous malnutrition.

MATERIAL AND METHODS

The study involved 40 malnourished children; 23 males matched for age and weight with 17 females. The volunteers were recruited mainly from Albuluk and Ibraheem Malik teaching hospitals – Khartoum –Sudan. The ages of studied subjects ranged between 6 – 36 months. Weight scale (Health O-Meter - USA) was used for measuring weight. Venous blood samples were obtained from the femoral vein. Spectrophotometer BTS 310 (Bio System Company - Germany) was used to measure serum albumin using bromcresol (SPINREACT). Patients were classified according to the presence of edema, Wellcome and Gomez classifications. In addition, patients were classified according to their level of albumin into normal (3.5 - 4.8 g/dl), mild (2.8 - 3.4 g/dl), moderate (2.1 - 2.7 g/dl) and severe (< 2.1 g/dl) hypoalbuminemia.

Statistical evaluation was performed using SPSS (SPSS for windows 19.0). Normal distribution of studied variables was examined using Kolmogorov-Smirnova and Shapiro-Wilk tests. Unpaired T-test and Mann-Whitney U test were used to assess significant difference in the means of albumin levels in the different groups. Screening for significant correlations between albumin levels and weight were performed using bivariate correlations. $P < 0.05$ was considered significant.

RESULTS

The study involved 17 (42.5%) females and 23 (57.5%) males. The females age (M±SD = 13.1±4.8 months) and weight (M±SD = 5.6±1.2 Kg) were not significantly different when compared to the males (M±SD = 16.8±8.0 months and 6.1±1.1 Kg respectively, $P > 0.05$). Serum albumin level was significantly lower in malnourished subjects with edema (M±SD = 2.4±0.8 g/dl) compared to those with malnutrition but no edema ((M±SD = 3.1±0.8 g/dl, $P = 0.026$) (figure 1 and 2). However, classifying studied malnourished children according to their albumin levels revealed no significant differences in the mean of albumin concentration when albumin levels of edematous patients were compared with non-edematous subjects in each class (figure 3). Moreover, there was no significant correlation between children weights and serum albumin levels (correlation coefficient (CC) = 0.156, $P = 0.337$) (figure 4).

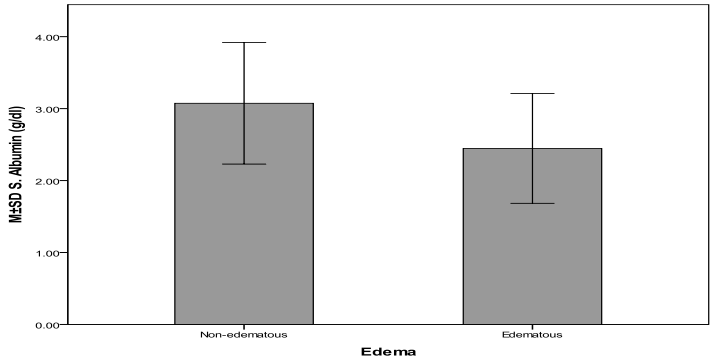

Figure-1: Distribution of serum albumin level in malnourished subjects grouped according to the presence of edema

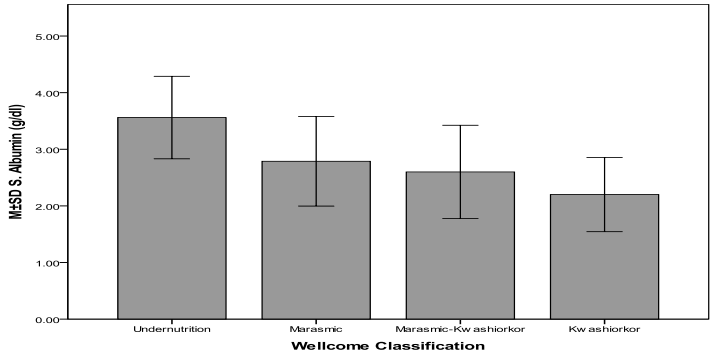

Figure-2: Distribution of serum albumin level in malnourished subjects grouped according to Wellcome classification

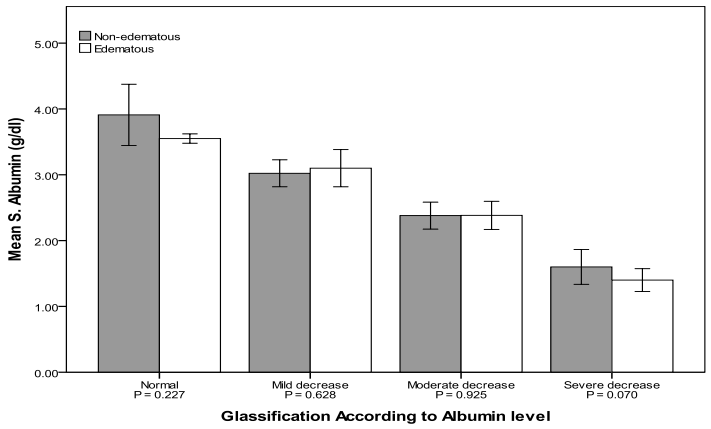

Figure-3: Comparison of malnourished patients with and without edema when grouped accordingto serum albumin levels

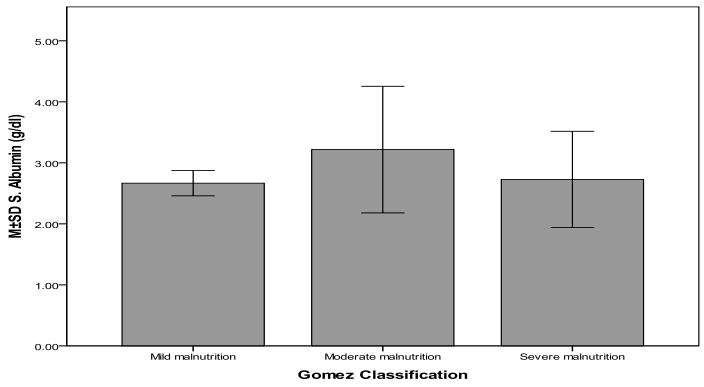

Figure-4: Distribution of serum albumin level in malnourished subjects when grouped according to Gomez classification

DISCUSSION

It is evident from the current results that serum albumin concentrations tend to be lower in edematous malnourished children, however, classifying studied children according to their albumin levels revealed no significant differences in the mean when albumin levels of edematous patients were compared with non-edematous subjects in each group. These two findings are not necessarily contradictory; in contrast they demonstrate the variations of the pathophysiological response of malnourished children to food deprivation [20. 21]. In addition the current study weaken the general believe that edema of malnutrition is solely an outcome of the decreased oncotic pressure caused by associated hypoalbuminemia[22].

The explanation for decreased serum albumin concentrations in edematous malnourished children is not simply attributable to poor food intake. There are several studies that confirmed the variations in the rate of protein metabolism in patients with edematous malnutrition compared to non-edematous conditions. By the end of the last century, Manary et al reported that kwashiorkor patients had slower rates of whole-body protein breakdown than did patients with marasmus [21]. Six years later, Jahoor and his group were able to prove that these differences in the rate of protein catabolism were because of inherent differences in protein turnover among corresponding patients and not due to dissimilar physiological adaptation to low food intake[20]. Therefore, Serum albumin may not be a useful marker for malnutrition especially in patients with chronic illnesses[23]. Pediatricians should pay more attention to the clinical manifestations of malnutrition and anthropometric measurements while evaluating malnourished children.

In general, edema of malnutrition is easy to explain by starling principle [24] which declares that capillary filtration rate across the capillaries is proportional to difference between hydrostatic and colloidal osmotic pressure. Low plasma albumin associated with malnutrition is expected to decrease plasma colloidal osmotic pressure and induce generalized edema [25]. However, previous reports showed that the edema secondary to hypoalbuminemia would not appear unless plasma albumin decreased to as low as 2-2.5 g/dl [26]. In addition, the extend edema might not correlate well with plasma protein concentration[27]. These reports preclude from drawing a conclusion that edema of malnutrition is mainly due to the decreased oncotic pressure caused by associated hypoalbuminemia.

In conclusion, the current study demonstrates the tendency of edematous malnourished children to have hypoalbuminemia; nevertheless, some children with

significantly low albumin are still not suffering from edema and some with normal serum albumin were still edematous. These paradoxical results display the possible variations in the pathophysiological response to food deprivation in those suffering from malnutrition. In addition the current study attenuate the general believe that edema of malnutrition is solely an outcome of the decreased oncotic pressure caused by associated hypoalbuminemia.

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