PREVALENCE AND MORPHOLOGIC PATTERN OF ANEMIA IN CHILDREN WITH SEVERE ACUTE MALNUTRITION TREATED IN JIMMA UNIVERSITY SPECIALIZED HOSPITAL: A CROSS-SECTIONAL STUDY

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Prevalence and Morphologic Pattern of Anemia in Children With Severe Acute Malnutrition Treated in Jimma University Specialized Hospital: A Cross-sectional Study

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Abstract

Background: Anemia is very common in severe acute malnutrition and multifactorial. It contributes for case fatality of severe acute malnutrition. But, the prevalence and types of anemia in malnourished children is not well established in the study area.

Objective: To determine the prevalence and morphologic pattern of anemia in children with severe acute malnutrition, admitted to Jimma University Specialized Hospital.

Methods and materials: A cross-sectional study was employed among children aged 6-60 months with severe acute malnutrition (WFH<70% or MUAC<11cm and/or bilateral pitting edema) treated in Jimma University Specialized Hospital during May-July, 2014. Weight, height and mid-upper arm circumference were measured using digital weighing machine, measuring board and tape meter, respectively, and then compared and interpreted using NCHS and WHO growth curves. Additionally, complete blood count by automation method and peripheral red blood cell morphology microscopically using Wright staining procedure were determined on admission and haematocrit by microhematocrit procedure. The data were double entered using EpiData 3.1 & analysed by SPSS 16.0. One sample t-test for continues variables, Chi square for categorical variables and multinomial logistic regression were used to adjust confounders and check significance.

Results: Fifty-two children, with median and IQR age, (19 months & 9-26 months) and 34 (65.4%) males, were included. Anemia(Hgb<11.0 g/dl) was found in 32 (61.5%) of the patients on admission. The commonest type of anemia on peripheral blood film was normocytic 18 (67.0%) and hypochromicity accounts for 34 (74%) of the patients. There was no difference in

the occurrence of anemia between edematous & non-edematous children at base line (61.5%vs 38.5%, p=0.16).

Conclusion: Anemia is highly prevalent in severely malnourished under-five children admitted to Jimma University Specialized hospital. Normocytic anemia is the predominant type. Most of anemic patients have hypochromic red blood cells. Similar studies are recommended as the present study is based on small sample size.

Keywords: Severe acute malnutrition, Anemia, Prevalence, Morphology

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List of Abbreviations

Hgb	Hemoglobin
Het	Hematocrit
MCV	Mean Corpuscular Volume
МСН	Mean Corpuscular Hemoglobin
МСНС	Mean Corpuscular Hemoglobin Concentration
JUSTH	Jimma University Specialized Hospital
EFMoH	Ethiopian Federal Ministry of Health
PEM	Protein Energy Malnutrition
SAM	Severe Acute Malnutrition
MUAC	Mid-upper Arm Circumference
WFH	Weight-for-Height
WFA	Weight for Age
HFA	Height for Age
WHO	World Health Organization
SC-UK	Save the Children – United Kingdom
JUCPHMS	Jimma University College of Public Health and Medical Sciences

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Background

Undernutrition is still a major public health problem in developing countries. It is associated with 50-60% of under-five mortality in poor countries [3]. Besides macronutrient deficiency, deficiencies in iron, iodine, vitamin A and zinc are the main manifestations of malnutrition in these countries [4, 5].

Formerly described as protein-energy malnutrion, severe acute malnutrion (SAM) describes severe forms of macronutrient deficiencies or malnutrion. It includes several overlapping syndromes of severe wasting (WFH < 70% NCHS median or MUAC < 11.0cm) or bilateral pitting edema [1].

Causes of SAM are multifactorial having a number of interwoven factors operating simultaneously. These causes could be categorized as immediate, underlying and basic [2].

Severe acute malnutrition is a multi-deficiency state and not simply a deficiency of macronutrients, like protein and calorie. It can have three forms, namely marasmus, kwashiorkor and marasmic-kwashiorkor. Whereas marasmus is considered a semi-starvation state, which includes the deficiency of energy, protein and other nutrients [2], there are several theories for kwashiorkor. One is low protein intake leading to hypoalbuminemia but different studies have shown that there was no edema in hypoalbumemic state. Dys-adaptation is the other theory that describes edema not to be determined only by diet but also by intrinsic differences among children with regard to their protein requirement and hormonal response and as a result kwashiorkor develops in children that poorly adapted and marasmus develops in those that are well adapted to the states of lower nutrient intake. The free radical theory explains edema results

from membrane damage by free radicals as there is lack of scavengers of free radicals which are of course micronutrients that the children are deficient in along with the macronutrient deficiency. The other ascribed theory in edema pathogenesis is the aflatoxins theory which was reported from study in Sudan by Hendricks that children with aflatoxins developed edema compared to those with no aflatoxin intake [2].

In Ethiopia, according to CSA rural nutrition survey in 1992, the highest prevalence of stunting(HFA< 5th centile on NCHS curve), wasting(WFH<70% of the median on NCHS curve) and underweight(WFA<5th centile on NCHS curve) recorded were 74.5%(Gondar), 14.2%(Tigray) and 59.9%(Tigray) respectively. According to this survey, the prevalence of moderate and severe forms of stunting and underweight showed an increasing trend over decade [2]. But, in the past few decades these rates have decreased quite a bit, most notably with mortality almost halving, according to EDHS2000-2011.

The mortality rate of children with complicated severe acute malnutrition that receive treatment in inpatient set ups has remained unacceptably high. Such high mortality in inpatient units has been attributed to co-morbidities such as infections and micronutrient deficiencies.

Anemia is found commonly as an associated feature of many pathological conditions. In most cases severe malnutrition is also accompanied by anemia, as integral part of the process of reductive adaptation associated with weight loss, reduced lean body mass and presence of edema. However, the specific cause of anemia is also complicated by associated deficiencies of specific micronutrients, increased red cell destruction and on-going suppression of red cell formation as a result of the inflammatory response to multiple infections. In oedematous malnutrition, it is usually associated with inability to effectively utilize iron, leading to an

increase in the iron stored and free form. Therapy with iron at this stage increases mortality. Because of the complexity of the possible interactions in the established condition, it can be very difficult to determine the sequence with which one factor might have acted as a primary exposure, subsequently interacting with the other factors, which later contribute and play a secondary role [6].

There are four key processes which separately and together contribute to anemia:

- 1. Reductive adaptation: anemia can be part of body's adaptation to reduce food intake and decreased metabolic activity.
- Specific nutrient deficiencies: formation of mature RBC is a complex process requiring the full complement of nutrients and metabolic intermediates. A limitation in any of these nutrients will limit the formation of red cells, their structural integrity and their functional capacity.
- 3. Infection: there is a complex interaction between infection and poor nutrition with each predisposing to, and making the other worse. In severe malnutrition, the effects of infection on anemia might be directly related to a specific infection or indirectly to more general inflammatory and immune response. Separately and together, these will limit the availability of nutrients for red cell formation and increase the likelihood of anemia.
- 4. Haemolysis, pro-oxidant damage: for the red cell, enhanced susceptibility to pro-oxidant damage will predispose to shortened life span, which increases the extent to which iron has to be held in an innocuous form. An increase in stored and/or free intracellular iron can act as the focus for on-going pro-oxidant stress and ensuing cellular pathology. An increased loss of red cells in the face of any limitation on red cell production inevitably leads to a reduction in red cell mass, and increased iron in storage.

The two most common micronutrient deficiencies associated with malnutrition, according to different studies, were found to be anemia and Vitamin D deficiency [7]. Severe anemia is one of the comorbidities responsible for increased mortality in severely malnourished children, yet it has not received the attention it should. In one study done in India, 25% of children with severe acute malnutrition required packed RBC transfusion and the most common type of anemia was microcytic(38.6%) followed by megaloblastic(30.5%)[8].

Even if there are few studies in the other part of the world, there are no adequate studies on prevalence and types of anemia in severe acute malnutrition in Ethiopia where there is already different degree of anemia in child population.

Study Rationale

The World Health Organization estimates that about 60% of all deaths, occurring among children aged less than five years in developing countries, could be attributed to malnutrition [9].

Sub-Saharan Africa bears the brunt of SAM in the world. On the average, the SAM associated mortality in sub-Saharan Africa is between 25 and 35% [10, 11]. In Nigeria, 22 to 40% of under-five mortality has been attributed to SAM [12]. SAM is also associated with a number of comorbidities such as lower respiratory tract infections including tuberculosis, diarrhea diseases, malaria and anemia [13, 14]. In other study on the nutritional status of Nigerian children revealed that 52% of child deaths are attributed to SAM making it the single greatest cause of infant mortality. The case fatality rate, despite inpatient treatment, remained high. In Africa, it's found to be as high as 20% even if acceptable fatality rate is expected to be 5%. Poorly trained staffs and faulty treatment is found to be the attributable reason. But, infections and micronutrient deficiencies were also found to be associated with mortality in severely malnourished children. The two most common micronutrient deficiencies are anemia and vitamin D deficiency [7]. In one of the studies done in India, 25% of children with severe acute malnutrition required transfusion [8].

Ethiopia is the second-most populous country in Africa, at nearly 84 million. Approximately 14% are children under five years of age [15]. These children and their mothers suffer disproportionately from the poor health and nutrition situation in the country. In fact, malnutrition is the underlying cause of 57% of child deaths in Ethiopia, with some of the highest rates of stunting and underweight in the world [16].

Micronutrient deficiency, also known as "hidden hunger", because it is less visible to the naked eye, is an additional, yet related issue in Ethiopia. The anemia rate among children 6-59 months remains high at over 44%, contributing to morbidity and mortality. Iron deficiency is the cause of half of all anemia cases [17]. Micronutrient deficiencies often co-exist with macronutrient deficiency. Factors associated with mortalities from severe acute malnutrion in our country are yet to be studied. As anemia is one of the factors associated with deaths from severe acute malnutrition in previous studies done in other parts of the world, it will be reasonable to look for this problem in Ethiopia.

The aim of this study is, therefore, to come up with prevalence and morphologic types of anemia occurring as co-morbidity with severe acute malnutrition so that it will be one stride for other studies.

Literature Review

Macronutrient deficiency, deficiencies in iron, iodine, vitamin A and zinc are the main manifestations of malnutrition in developing countries [4, 5].

Child anemia and malnutrition have both short- and long-term adverse consequences that have serious implications for individuals and societies. It continues to be a major health burden in developing countries and is a substantial contributor to childhood morbidity and mortality [18-20].

Malnourished children are often anemic. Changes in the hematologic system are common, and anemia has always been a constant feature. The various factors that influence anemia in SAM are (a) metabolic changes in the red cell, (b) protein deficiency and adaptation anemia, (c) iron deficiency, (d) vitamin deficiency (vitamin B12, folate), or trace elements (copper, zinc), erythropoietin deficiency, (f) infections and (g) chronic disease [21].

Most often, malnutrition is associated with iron deficiency and anemia as a co-occurrence and that is a sinister combination. Each one aggravating the other, during infancy and early childhood culminates into reduced physical growth, which is more likely to be carried during the adolescence and adulthood. The prevalence of both conditions in the population (India) was higher than 40% [22].

In the study done on 150 subjects with malnutrition admitted in tertiary care hospital in Pakistan, 119(79%) were severely malnourished and 30(20%) were moderately malnourished. In this study anemia was the most common micronutrient deficiency seen in 117(78%) patients, out of these

88 had iron deficiency anemia. Co-existing rickets was found in 44% of severely stunted patients [23].

In another survey done in Pakistan (National Nutritional Survey-III, from 1985-1987), according to WHO criteria for weight-for-Age, 48% of young children were malnourished within which 10% were severely so. Anemia occurred in 65% of young children. In this study the number of under-5 children was 11,285[24].

In one of the studies done on India which included 131 cases of SAM with the age group varied between 6 and to 59 months. Of patients with SAM, 67.3% had severe anemia; 13.8% had moderate anemia. Of these patients, 25% required packed red blood cell transfusion. The most common type of anemia was microcytic (38.6%) followed by megaloblastic (30.5%)[8].

In another National survey done in India in 2005-2006, 20% of under-5 children were wasted of which 6% were having severe acute malnutrition, 43% were underweight while 48% were stunted. In this study, nearly 70% were anemic, of which 26% with mild anemia, 40% moderately anemic and 3% were severely so [25].

According to other Indian study done on 104 subjects hospitalized with severe acute malnutrition in Rewa District, 88.5% were anemic and out of which 7.6% mildly, 55.7% moderately and 24% severely anemic. In this study anemia and vitamin D deficiency were the two most common micronutrient deficiencies associated with malnutrition [7].

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Seventy percent of severe acute malnutrition patients in Africa were anemic at admission and no evidence of iron deficiency on admission. In this study, the incidence of megaloblastic anemia was found in 25% of patients with severe protein-energy malnutrition [26].

Another study in Shaanxi province in North Western China, among 118 infants with anemia, the prevalence of malnutrition was found to be 43.3% [27].

Similarly, in the study of prevalence of anemia in under five-year-old children in a children's hospital in Recife Brazil, when the Marascuilo procedure for multiple comparisons was applied, anemia was more prevalent among malnourished children than well-nourished children (p-value = 0.008) [28].

There are also studies in Africa. In the study done on risk factors in hospital deaths in severely malnourished children in Kampala Uganda, out of 220 severely malnourished children of less than sixty months of age, their mean hemoglobin was 8mg/dl, over 80% of 217 children for whom hemoglobin determined were anemic, out of which 14(6.5%) were severely anemic (Hgb<5g/dl) and 8(3.7%) were having severe anemia, Hgb<4g/dl. A total of 52(23%) children received blood transfusion during which 24(48%) died. This study concluded that the main risk factors of deaths among these children include blood transfusion and intravenous infusion [29]. Studies of red cell metabolism, erythropoietin concentration, iron and folate status were made in 48 children with protein-energy malnutrition in Johannesburg. Biochemical evidences showed that iron deficiency was present in 26% of cases on admission and developed in 90% of cases during recovery.

Folate deficiency was present in 14% of cases on admission and resolved on dietary therapy alone [30].

In other regional study which was conducted to investigate the prevalence and etiology of severe anemia in under five children in Abakaliki South Eastern Nigeria, malnutrition was associated with severe anemia in 10(7.1%) from 140 subjects with severe anemia, of which 3(30%) had severe malnutrition and 7(70%) were undernourished. These children had no other reason for severe anemia [31].

In a comparative study done on nine subject infants, in one of Egyptian Hospitals, of which four with marasmus, three kwashiorkor and two normal controls, to investigate plasma and red cell iron turnover in protein-energy malnutrition, it showed that hypochromic anemia was present in all infants with malnutrition. In kwashiorkor patients, bone marrow pictures demonstrated diminished cellularity with reduction in normoblasts. In one of kwashiorkor patients, marrow megaloblasts constituted 8% of count. The plasma and blood volume were increased in both marasmic and kwashiorkor patients compared to the normal controls. The plasma iron turnover was low in 2 infants with kwashiorkor and within normal range in the whole group of marasmus. The red cell iron uptake was slightly subnormal in kwashiorkor and in one marasmic infant. The apparent red cell iron turnover rate was increased and the corresponding apparent red cell survival was diminished in kwashiorkor and marasmus. The latter was, however, shortest in the marasmic infant showing a megaloblastic bone-marrow reaction [32].

In another study in this country on 73 infants with protein-energy malnutrion, the incidence of megaloblastic anemia was 48% (unpublished) .Other authors from similar country reported variable incidence of megaloblastosis in protein-calorie malnutrition[33, 34].

In other Nigerian study, it was observed that the hemoglobin, packed cell volume and serum ferritin $(8.70\pm1.10g/dl, 0.26\pm0.04L/L$ and $15.10\pm13.45ng/ml$) of protein energy malnourished children were significantly lower (P<0.05) than values obtained for control subjects $(11.00\pm0.90g/dl, 0.33\pm0.02L/L, 53.50\pm30.13ng/ml$). These low values could be attributed to poor diet due to low socio-economic status of the parents. It was observed from the administered questionnaire that these children were from very poor background, not well breast-fed or abandoned. The significant reduction in hemoglobin and packed cell volume in this study could be explained by the fact that synthesis of hemoglobin is impaired in protein-depletion conditions [35]. Indirect evidence that protein deficiency is the cause of anemia in SAM was shown by Allen and Dean (1993)[36]. However, it has been shown that an erythropoietic adaptation rather than anemia occurs in children with SAM as a consequence of a reduction in lean body mass, as measured indirectly by urinary creatinine excretion [37].

According to Viteri and Alvarado (1970), total circulating hemoglobin is reduced in proportion to the degree of depletion of the lean body mass [36].

In the study done in West Indies on the acute-phase protein response to infection in edematous and

nonedematous protein-energy malnutrition, there were no significant differences in white blood cell counts or hemoglobin and albumin concentrations between the 3 groups (marasmus ($85.9 \pm$

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2.9), marasmic-kwashiorkor (89.7 \pm 5.1) and kwashiorkor (90.6 \pm 9.2) in terms of hemoglobin)[38].

Objectives

General Objective

To determine prevalence and morphologic pattern of anemia in children with severe acute malnutrition treated in JUSH

Specific Objectives:

1. To determine prevalence of anemia in children with severe malnutrition.

2. To describe the morphologic types of anemia in children with severe acute malnutrition.

Methods

Study Design: Cross-sectional study design was used.

Setting

Jimma University Specialized Hospital is one of the few tertiary hospitals in the country. It is located in Jimma Zone in southwest Ethiopia. There are neonatal intensive unit, general ward with critical care room and nutritional rehabilitation unit in the pediatric ward of the hospital. The nutritional rehabilitation unit is exclusively used for severely malnourished children that fulfill in-patient admission criteria or those coming with referral paper from other facilities including out-patient therapeutic program. Patients who were admitted to this unit had relatively stable condition. Critically ill children were stabilized in the critical care room before they were transferred to this unit. Any child, who became critically ill in the rehabilitation unit, was transferred to the critical care room until stabilized. The admission criteria were based on the national management guideline for severe acute malnutrition (SAM). The standard of care was based on the guideline of management for SAM, 2007[1].

Participants

All severely malnourished children between 6-60 months of age admitted to nutritional rehabilitation unit during the study period, March – July, 2014 G.C, were included in the study. The eligibility criteria were weight-for-height below 70% of the median of the NCHS growth reference or MUAC less than 11.0 cm or bilateral pitting edema. Children with congenital disorders, other hematologic or immunologic disorders and history of treatment with therapeutic food or hematinics before admission were excluded.

Variables

Dependent variables

Anemia: hemoglobin level below 11mg/dl, according to WHO (2001) criteria.

Type of anemia

Normocytic: MCV=70-81fL for 6-24 months of age and 75-86fL for 24-60 months of age or RBC size similar to nucleus of resting small lymphocyte on peripheral film.

Microcytic: MCV < 70fL for 6-24 months and < 75fL for 24-60 months or RBC size less than nucleus of small lymphocyte on peripheral film. See Annex-II

Macrocytic: MCV > 81fLfor 6-24 months and > 86fL for 24-60 months or RBC size bigger than nucleus of small lymphocyte. See Annex-II

Normochromic: MCH=24-30pg for 6-23 months and 25-31pg for 23-60 months or central pallor area is less than or equal $to1/3^{rd}$ of the entire RBC diameter on peripheral film.

Hypochromic: MCH<24pg for 6-23 months and < 25pg for 23-60 months or central pallor area is greater than $1/3^{rd}$ of the entire RBC diameter on peripheral film.

Hypersegmented neutrophils: > 5% with 5 lobes or > 1% with 6 or more lobes Independent variables

Severe acute malnutrion: WFH<70% or MUAC<11.0cm or bilateral pitting edema

Gender and age

Parental education

Comorbidity

Treatment

Data source and measurement

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Data were collected using data collection format which includes sociodemographic characteristics by interviewing the parents/caretakers, measuring weight (to nearest 0.1) using digital weighing scale, height (to the nearest 0.1) using measuring board (supine for <24 month and erect for above 24 month old), and MUAC (to the nearest 0.1) using tape meter. CBC on admission was also done using sysmx analyser(see above) which is available in the Hospital after specimen collected to 3-mL K2 EDTA tube from peripheral veins and run within 4hours after sample collection. Haematocrit was determined by microhematocrit method in accordance with SOPs. Peripheral morphology examination was performed after smear prepared and application of Write staining procedure. RBC size on CBC has been determined based on RDW shifting and chromicity was also defined in comparison with MCHC value from CBC.

Potential biases

There was an effort to reduce selection bias by including all subjects who fulfil admission criteria and no need of selection from among eligible candidates who pass inclusion criteria, was required. Anthropometric measurements were done after calibration of the scale.

Sample Size Determination

Considering 90% of prevalence (p) from previous studies with 95% confidence level (Z) and 5% margin of error (d), required sample size (n) was calculated by the formula: $N = z^2 pq / d^2 \Rightarrow N = (1.96)^2 (0.9) (0.1) / (0.05) = 138$ Since N = 138 is < 10,000, given N' large sample > 10,000, then n can be calculated by: n = N / (1 + N/N') = 138 / (1+138/10,000) = 138/1.0138 = 136 With 10% contingency, n = 150

But, only fifty-two subjects were recruited because of a very short study period and significant budget shortage. Data collection can be continued in the future.

Quantitative variables handling

The quantitative variables in the study were age, weight, height, MUAC, Hct, and RBC indices. All of them were measured to the nearest 0.1. Hct and RBC indices values were transformed to categorical data using SPSS 16.0 for analysis purpose.

Statistical methods application

One sample t test was used for all continuous variables. Chi square test was employed to analyse categorized variables. Multinomial logistic regression was applied to see the impact of certain variables (edema, age, wasting, stunting and sex) on the development of anemia as well as to adjust confounders. In all cases, p<0.05 was considered significant.

Limitations and challenges encountered

There was significant time and financial limitation to undertake this study on a larger sample size; additionally more important laboratory tests, like bone marrow examination, serum level of micronutrients, plasma protein level and volume should have been used to meet the whole objectives of the study. There was also interruptions of data collection because absence of therapeutic food in the hospital stock.

Ethical considerations

This study has passed ethical clearance and approved by ethical committee of Jimma University College of Public Health and Medical Sciences Post-graduate Research Project. Data were collected after consent is granted from every caretaker/family and candidates were excluded when their families fail to give consent, but they had received appropriate care as per the standard.

Results

General Characteristics

Fifty-two children with SAM (34 males and 18 females) age 6-60 months with a median age of 19 months and IQR of 9-24 months, were studied. The general characteristics of the subjects are shown in Table 1. The majority 45 (86.5%) of the mothers of the studied children had no formal education (illiterate), while 42 (82.7%) of fathers had a similar level of education. The mean of participants' mid-upper arm circumference was 11.39cm. Twenty-nine(55.8%) of patients were severely wasted(WFH<70% or MUAC <11cm), while 38(73.1%) and 32(61.5%) of them were stunted and edematous, respectively. In terms of clinical forms of severe acute malnutrition, 32(61.5%) edematous and 20(38.5%) marasmus cases were admitted to the nutritional rehabilitation unit and participated in the study. The major comorbidity identified was anemia which accounts 61.5% of the patients, while diarrheal diseases, pneumonia, and urinary tract infections account 34.6%, 26.9% and 15.4% of patients. Admission hemoglobin was determined for all subjects, the mean hemoglobin of which was 10.31 ± 0.2728 . 32 of 52 patients were anemic.

Complete blood count and peripheral morphology was determined for reasonable number of patients, see table 2. All patients had received F-75, of which 40 treated with F-100 and only 3 patients got RUTF as transition and phase two treatment. The means of MCV, MCH and MCHC of all severely malnourished children in the study were 77.4 ± 11.5 fl, 24.3 ± 4.6 pg and 31.1 ± 2.6 %, respectively.

Characteristics, N=52	Frequency
Age, months	
12-60	21(40.4%)
6-12	31(59.6%)
Median	19
IQR	9-24
Sex, Male	34(65.4%)
MUAC, in centimeters	
Mean \pm SD	11.39±1.95
Median	11.0
Severe wasting (WFH<70% or	
MUAC <11cm)	29(55.8%)
Stunting (HFA <5 th centile)	38(73.1%)
Type of malnutrition	
Non-edematous	20(38.5%)
Edematous	32(61.5%)
Maternal Education	
Illiterate	45(86.5%)
Grade 1-6	3(5.8%)
Grade 7-8	2(3.8%)

Table 1. Characteristics of severely malnourished children participating in the study, Jimma University Specialized Hospital, Jimma, 2014.

Grade 9-12	2(3.8%)
Paternal Education	
Illiterate	43(82.7%)
Grade 1-6	3(5.8%)
Grade 7-8	2(3.8%)
Grade 9-12	4(7.7%)
Comorbidity	
Diarrheal diseases	18(34.6%)
Pneumonia	14(26.9%)
UTI	8(15.4%)
Others	12(23.1%)
Treatment	
F-75	52(100%)
Antibiotics	52(100%)
Folic acid	52(100%)

Characteristics, N=52	Mean \pm SD or N (%)	p-value ^a
Admission hemoglobin, g/dl	10.3±0.27	0.007
MCV(fL) (N=39)	77.4±11.5	0.004
MCH(pg) (N=39)	24.3±4.6	0.34
MCHC,% (N=39)	31.1±2.6	0.02
Peripheral morphology, N=46		
Normocytic	34(73.9%)	
Microcytic	12(26.1%)	
Normochromic	12(26.1%)	
Hypochromic	34(73.9%)	

Table 2. Laboratory profiles of severely malnourished children, Jimma University Specialized Hospital, Jimma, 2014.

Prevalence of anemia and its morphological pattern

As indicated in table 2 and figure 1, 32 of 52 patients (61.5%) were found to be anemic. Of these anemic patients, 12(37.5%) mildly, 20(62.5%) moderate to severely anemic. 19(59.4%) of them were edematous, out of which 12(63.1%) had moderate to severe anemia. A child who was infant or had male gender or edema, was more likely to develop anemia as compared to her/his counterpart, but statistically was not significant(p>0.05 and 95% CI of aOR includes 1), see table 4. RBC indices on complete blood count and peripheral morphology indicated the morphologic pattern of the anemic patients to be, 15(48.39%) normocytic-hypochromic, 10(32.26%)

microcytic-hypochromic, and 3 (9.68%) normocytic-normochromic anemia. Hypochromicity was found in 81.48% of the patients, indicating the importance of iron deficiency which could be of multifactorial origin (p<0.05) even though causes of anemia was not part the objective of the study. In two of the cases, hyperchromia with bigger RBCs was found.

Table 3. Prevalence of anemia among severely malnourished children in Jimma University Specialized Hospital, Jimma, 2014

Anemia status	Frequency	p-value	
On admission (N=52)			
No anemia	20(38.5%)		
Anemia	32(61.5%)	%)	

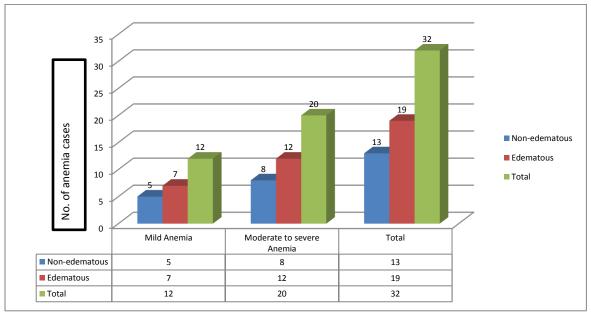


Figure 1. Distribution of anemia by type of severe acute malnutrition, Jimma University Specialized Hospital, Jimma, 2014.

RBC indices or peripheral morphology		Degree of anemia		Total	p-value
		Mild	Moderate/severe		
MCV	Normocytic	8(32%)	6(24%)	14(56%)	0.09
	Microcytic	1(4%)	5(20%)	6(24%)	
	Macrocytic	0	5(20%)	5(20%)	
MCH	Normocromic	6(24%)	7(28%)	13(52%)	0.26
	Hypochromic	3(12%)	7(28%)	10(40%)	
	Hyperchromic	0	2(8%)	2(8%)	
MCH & MCV	Normocytic-				
	normochromic	6(24%)	4(16%)	10(40%)	
	Normocytic				
	hypochromic	2(8%)	2(8%)	4(16%)	
	Microcytic				
	hypochromic	1(4%)	4(16%)	5(20%)	
	Macrocytic				
	normochromic	1(4%)	2(8%)	3(12%)	
	Other				
	combination	0	3(13%)	3(12%)	
Peripheral morphology					
(N=27)					
RBC size	Normocytic	7(25.9%)	11(40.7%)	18(67%)	0.04

Table 4. Morphologic types of anemia in severely malnourished children participating in the study, Jimma University Specialized Hospital, Jimma, 2014.

	Microcytic	3(11.1%)	6(22.2%)	9(33.3%)	
	Macrocytic	0	0	0	
Chromicity	Normochromic	1(3.7%)	4(14.8%)	5(18.5%)	
	Hypochromic	9(33.3%)	13(48.1%)	22(81%)	0.0
By RBC indices and	Normocytic-				
peripheral morphology	normochromic	0	3(9.7%)	3(9.7%)	
(N=31)					
	Normocytic-				
	hypochromic	7(22.6%)	8(25.8%)	15(48%)	
	Microcytic-				
	hypochromic	4(12.9%)	6(19.4%)	10(32%)	
	Macrocytic-				
	normochromic	1(3.2%)	1(3.2%)	2(6.5%)	
		`` '	× ,	、	
	Macrocytic-	0	1/2 00/ >	1(2,00())	
	hyperchromic	0	1(3.2%)	1(3.2%)	

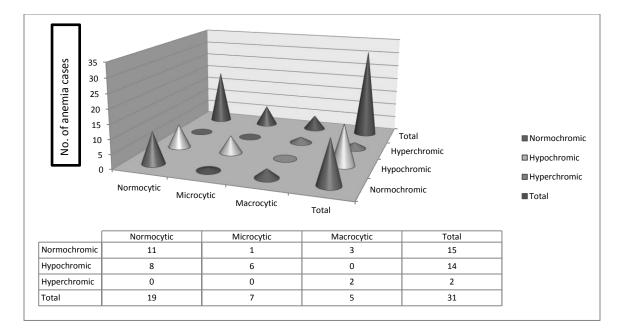


Figure 1. Peripheral morphology pattern of anemia in severe acute malnutrition patients, Jimma University Specialized Hospital, 2014.

Table 5. Independent predictors of anemia in severely malnourished children, Jimma University Specialized Hospital, Jimma, 2014.

	Crude OR(95% CI)	Adjusted OR(95% CI)
Age group, 6-12mo/12-60mo	2.1(0.6-6.7)	3.5(0.8-14.9)
Gender, male/female	1.5(0.5-4.7)	1.8(0.5-6.4)
Severe wasting, yes/no	0.8(0.2-2.3)	1.0(0.2-5.4)
Stunting, yes/no	0.8(0.2-3.0)	0.8(0.2-3.6)
Nutritional Edema, yes/no	1.9(0.6-6.0)	3.3(0.6-17.9)

Discussion

Undeniably, malnutrion remains one of the most common causes of morbidity and mortality throughout the World, particularly in Sub-Saharan African countries and South and Southeastern part of Asia. Ethiopia is among those countries that primarily contributes the overall global burden of malnutrition in children. Malnutrition has been responsible, directly or indirectly, for more than half of the 10.9 million deaths annually among under five children.

Fatalities from severe acute malnutrition are not explained solely by the undernutrition, rather additional contribution from common comorbidities which have intricate relationship with malnutrition. Micronutrient deficiency is top coexistent that plays its role in the causation and pathogenesis of anemia, as one of the multiples of factors. Anemia is among the most common comorbid condition in severe acute malnutrition, which may contribute for the fatalities [4, 5, 10, 22, 23, 29]. There are studies in Ethiopia on anemia in the school children and child population in general, but there is little information on anemia of severe acute malnutrition even if it is one of the causes of mortality in these patients.

Regarding the associated comorbidities, as indicated in previous study done in Rewa district in India, on 104 severely malnourished subjects in Ghandi Memorial Hospital, the common associated illnesses excluding anemia, in decreasing order of frequency were diarrhea (54%), acute respiratory tract infections (27.8%) and tuberculosis (2.2%). Anemia was reported as micronutrient deficiency accounting 92(88.3%) of the cases, followed by Rickets and vitamin A deficiency[39]. The current study also showed comparable results that include diarrheal diseases, pneumonia, and urinary tract infections account 34.6%, 26.9% and 15.4% of patients, respectively. The major comorbidity identified was anemia which accounts 61.5% of the

patients, which is lower than the Indian study, possibly explained by smaller sample size. Additionally, in other similar study done in India on 131 subjects of the same age group, demonstrated a higher prevalence of anemia as the previous one; 67. 3% severe anemia and 13.8% moderate anemia. It has also depicted the common types of anemia to be microcytic (38.6%) and macrocytic (30.5%), while the current study showed different figures and the commonest type of anemia being normocytic-hypochromic 15(48.4%) accompanied by microcytic-hypochromic 10(32.4%) and normocytic-normochromic 3(9.7%) anemia[7]. The lack of reproducibility may be due partly to large difference in sample size and additionally, behavioural and cultural variation in nutrition and child raising practices can also contribute, in spite of relatively similar socio-demographic characteristics.

Evaluation of the RBC indices demonstrated that the admission mean Hgb was 10.3mg/dl, mean MCV 77.4fL, and mean MCHC 31.1 %(p<0.05). The peripheral RBC morphology has also shown hypochromicity in 22 of 27 films (81.5%), with the evidence of the low mean MCHC value; this may indicate that most patients were in iron deficiency state. In a case control study in Nigeria with 90 SAM patients and 90 controls, children with SAM had lower mean values for Hgb, Hct and MCV (p<0.05)[40].

The present study has also shown that from 32 cases of anemia, 61.5% had edematous type of malnutrition (either kwashiorkor or marasmic-kwashiorkor), but there was no statistically significant association (aOR=3.3, 95% CI 0.6-17.9), see table 4. There is another study in Egypt which was conducted on 40 severely malnourished children between the ages of 6-15 months to evaluate serum erythropoietin level, and revealed that kwashiorkor & marasmic-kwashiorkor patients had more fragile red cells than marasmic patients and control groups, but difficult to draw conclusion from this small sample based study. Although erythropoietin was increased and

appropriate for the degree of anemia in cases of protein energy malnutrition, there was no concomitant significant increase in the absolute reticulocyte count[41]. At the onset it is worth noting that although anemia is usually mild or moderate in degree on admission, it may become severe after the commencement of dietary treatment. In one previous study done in McCord Zulu hospital (in 1955) on 776 patients, demonstrated that megaloblastic anemia, with bone marrow examination, is found in 42 cases and out of these 52% were kwashiorkor patients. There was decline in hematocrit in these patients with dietary treatment, in 15 of them there was sudden decline requiring transfusion[42]. In another study done in Abadan Nigeria on 9 infants with kwashiorkor, it was found that all patients were mildly anemic and macronormocytic normochromic in type. There was decline in Hgb with treatment and there was rise in Hgb concomitantly with increase in serum protein level over a period of weeks and months, indicating that one of the factor in causation of anemia may be that Hgb in its production can draw on plasma proteins and thus when these proteins are abnormal or deficient, the production of Hgb may be affected[43]. But, the present study didn't try to see changes with treatment.

The specific cause of anemia is also complicated by associated deficiencies of specific micronutrients, increased red cell destruction and on-going suppression of red cell formation as a result of the inflammatory response to multiple infections as well hemodilution following establishment of dietary treatment. It can be very difficult to determine the sequence with which one factor might have acted as a primary exposure, subsequently interacting with the other factors, which later contribute and play a secondary role [6, 21, 44]. Even if it was not the aim of the present study, the abundance of normocytic anemia and hypochromicity can be indicative of multifactorial origin, but there were signs of iron deficiency. In a study done in 1969 on 148 infants with kwashiorkor from Africa and India, it has demonstrated that anemia due mainly to

protein deficiency, but there was some evidences that deficiency of iron and folic acid also play a role[45].

Generalizability of findings of the present study is questionable as a result of certain limitations. Primarily, the small sample size significantly limits external validity of the study results. Secondly, as it is a facility based investigation, the study is not immune from biases. Additionally, precision and accuracy of anthropometric measurements and validity of laboratory tests are difficult to monitor and control perfectly.

Conclusion

With all its limitations, the following three conclusions can be drawn from the current study. First, the prevalence of anemia is significant in severely malnourished children. Second, there is no difference in the occurrence of anemia between edematous and non-edematous malnutrition. Lastly, hypochromicity is very common in malnourished children; even though anemia can come from interaction of multiple interwoven factors.

Recommendation

The prominent findings of this study are generally similar with studies done in other settings, outside Ethiopia. Therefore, further studies should be done to see the real picture of anemia status of malnourished children and the morphologic pattern of anemia with adequate samples and standard laboratory investigations.

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Annex-II: Procedures and Reference Tables

	Hemog (g per d					orpuscular (fL)
Age	Mean	2 SDs below mean	Mean	2 SDs below mean	Mean	2 SDs below mean
26 to 30 weeks' gestation	13.4	11.0	41.5	34.9	118.2	106.7
28 weeks' gestation	14.5	NA	45	NA	120	NA
32 weeks' gestation	15.0	NA	47	NA	118	NA
Full term (cord sample)	16.5	13.5	51	42	108	98
1 to 3 days	18.5	14.5	56	45	108	95
2 weeks	16.6	13.4	53	41	105	88
1 month	13.9	10.7	44	33	101	91
2 months	11.2	9.4	35	28	95	84
6 months	12.6	11.1	36	31	76	68
6 months to 2 years	12.0	10.5	36	33	78	70
2 to 6 years	12.5	11.5	37	34	81	75
6 to 12 years	13.5	11.5	40	35	86	77
12 to 18 years (male)	14.5	13.0	43	36	88	78
12 to 18 years (female)	14.0	12.0	41	37	90	78
Adult (male)	15.5	13.5	47	41	90	80
Adult (female)	14.0	12.0	41	36	90	80

Table-IIA: Age-specific Normative Red Blood Cells Values

NA = not available; SD = standard deviation.

Adapted with permission from Robertson J, Shilkofski N, eds. The Harriet Lane Handbook. 17th ed. Philadelphia, Pa.: Mosby; 2005:337.

Table-IIB: Cut-off values used by the World Health Organization to define anemia

Reference group	cut-off (g/L)	Categorie	Categories of anemia (g/L)				
		Mild	Moderate	Severe			
Pregnant women	110	100-110	70-99	<70			
Non-pregnant women	120	110-119	80-109	<80			
Children 6 - 59 months of age	110	100-109	70-99	<70			
Children 5 - 11 years of age	115	110-114	80-1-9	<80			
Children 12 - 14 years of age	120	110-119	80-109	<80			
Men	130	110-129	80-109	<80			

Adapted from: Iron Deficiency Anaemia Assessment, Prevention and Control. A guide for programme managers. Geneva: World Health Organization, 2001.

Annex – III

Data Collection Format

Stu	ıdy	ID:
Da	te o	f Admission:
Da	te o	f Enrolment:
A)	SO	CIODEMOGRAPHIC CHARACTERISTICS
	1.	Age in Months:
	2.	Sex:- 0) Male 1) Female
	3.	Maternal Education: 0) Illiterate1) Read and write2) Grade1-63) Grade7-8
		4) Grade 9-12 5) College/University/Vocational
	4.	Paternal Education: 0) Illiterate1) Read and write2) Grade1-63) Grade7-8
		4) Grade 9-12 5) College/University/Vocational
B)	AN	THROPOMETRIC INDICES
	1.	Weight in Kilograms:
	2.	Height in centimetres:
	3.	MUAC in centimetres:
C)	CL	LINICAL PARAMETERS
	1.	Bilateral pitting edema:- 0) Yes 1) No
	2.	Comorbidity on admission or during hospital stay:
		0) Urinary tract infection 1) pneumonia 2) Tuberculosis
		3) Diarrheal disease4) Intestinal parasitosis
		5) Others (specify):

D) LABORATORY PARAMETERS

- 1. Hematocrit in percent:
 - a) On admission: -----
 - b) With therapeutic Feeding:
 - i) In 1st 3 days in transition phase: ------
 - ii) In 1st 3days in phase two: -----
- 2. RBC Indices on Admission:
 - a) MCV: ----- fL c) MCHC: -----g/dL
 - b) MCH: ----- pg d) RDW: -----
- 3. Peripheral Morphology Pictures
 - a) RBC size:- 0) Microcytosis 1) Normocytosis 2) Macrocytosis
 - b) RBC chromicty:- 0) Normochromic 1) Hypochromic
 - c) Hypersegmented Neutrophils:- 0) Yes 1) No

E) TREATMENT PROVIDED

	Phase One/ Day			Transition phase/Day				Phase Two/Day											
	1	2	3	4	5	6	1	2	3	4	5	6	1	2	3	4	5	6	7
Anthropometry						•				1	1			1	1				
Weight (cm)																			
Height (cm)																			
MUAC(cm)																			
Oedema(Grade)																			
Comorbidity				1	1	I				1				1					
Antibiotics																			

	Start Date	End Date	Start Date	End Date	Start Date	End Date
1.						
2.						
3.						
Feed Type		I		I		
F-75						
F-100						
RUTF						
Iron						
Folic Acid						
Deworming						
Others						

Note: Oedema if absent write 0 and present put grade as +, ++ or +++ when applicable

For medication write/mark:-

- 1) Continued if remain on similar drug in different phase(s)
- 2) Same date in the end date column if the drug is single dose