"A COMPARATIVE STUDY TO KNOW THE HAEMATOLOGICAL PROFILE AND BIOCHEMICAL PROFILE AMONG CHILDREN WITH SEVERE ACUTE MALNUTRITION AND HEALTHY CHILDREN"

By
REG. NO: BN0117003

Dissertation

Submitted to the
KLE Academy of Higher Education and Research,
Belagavi, Karnataka

In partial fulfilment
of the requirements for the degree of

DOCTOR OF MEDICINE
IN
PATHOLOGY

DEPARTMENT OF PATHOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELAGAVI, KARNATAKA

APRIL-2020

K.L.E ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA.

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Sub: Acceptance Letter

Sir/Madam,

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Yours sincerely,

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ABSTRACT

BACKGROUND: Malnutrition is India's silent crisis as the maximum number of malnourished children less than five years of age and related deaths twice to that of sub-Saharan Africa are reported here.[1] In India, more than five millions children are estimated to die every year as a result of undernutrition directly or indire ctly due to which succumbs in the loss of one child every ten seconds.[2]SAM is the main public health problem, which affects around 7.5% of under-five children in India according to NFHS-4 (2015-16).[3] Severely malnourished children are more prone to long term developmental delay and have weakened immunity making them susceptible to infections. Children with SAM can suffer consequences for their future health, learning and economic performance. SAM results in numerous pathophysiological changes in the body systems including alterations in hematological parameters.

OBJECTIVES:

PRIMARY OBJECTIVE:

1. To compare the haematological profile among children with severe acute malnutrition and healthy or well nourished children.
2. To compare the biochemical profile among children with severe acute malnutrition and healthy or well nourished children.

SECONDARY OBJECTIVE: Comparison of anthropometric parameters in children with severe acute malnutrition and healthy or well nourished children.

METHODOLOGY: A total of 60 subjects in the age group of 6 months to 5 years, with 30 cases diagnosed as severely acute malnourished and 30 controls at Nutrition Rehabilitation Center, KLES Dr.Prabhakar Kore Charitable Hospital and Medical research centre, Belagavi from January 2018- December 2018. Under aseptic conditions, 5 ml of venous blood was collected in vacutainers, 2.5 ml in EDTA vacutainers for haematological investigations and 2.5 ml in plain vacutainers for biochemical investigations. All the samples were then analyzed using an automated analyser.

RESULTS: In the present study, for identification of SAM cases, all the 30 cases (100%) have <-3SD weight for height and 10 cases (33.33%) have MUAC <11.5 cm. The most common age group of the children that were enrolled in the study was between 13-36 months with females (53.33%) outnumbering males (46.67). The male : female ratio was 1:1.42.Most of the SAM children had marasmus (90%) followed by marasmic-kwashiorkar (6.66%) and kwashiorkor (3.33%).Majority of the SAM cases had anaemia (90%), out of which majority had moderate anaemia (50%) followed by severe anaemia (26.67%), mild anaemia (13.33%) and normal blood picture (10%).The most common type of anaemia observed was microcytic hypochromic anaemia (63.33%) followed by dimorphic anaemia (23.33%), normocytic hypochromic anaemia (3.3%) and normal blood picture (10%) .Most of the cases which had microcytic hypochromic anaemia (63.33%) majority of them had decreased serum ferritin levels (84.21%) and rest had normal ferritin levels. In our study the mean serum sodium, mean serum albumin and mean serum total proteins were significantly lowered in severely malnourished subjects as compared to controls.

CONCLUSION: The findings of this study highlights the significant haematological and biochemical changes occurring in cases of severe acute malnutrition in children aged between 6- 60 months as compared to healthy children. This study also highlights “weight for age” as more sensitive marker than MUAC for identification of SAM children in children aged between 6 – 60 months of age.

KEY WORDS: MUAC ; NFHS; SAM; W/H and SD.

LIST OF ABBREVIATIONS

CC	Chest circumference
EDTA	Ethylene diamine tetra acetate
FAO	Food and agriculture organization
H/A	Height for age
IAP	Indian Academy of Paediatrics
MUAC	Mid upper arm circumference
MCV	Mean corpuscular volume
MCH	Mean corpuscular haemoglobin
MCHC	Mean corpuscular haemoglobin concentration
NFHS	National family health survey
OFC	Occipito-frontal circumference
PEM	Protein energy malnutrition
RBC	Red blood cells
SAM	Severe acute malnutrition
SD	Standard deviation
UNICEF	United Nations International Children's Emergency Fund
W/H	Weight for height
WHO	World Health Organisation

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INTRODUCTION

India, with a population of 1.37 billion, faces many challenges in refining the well-being and nutrition of its people. Malnutrition is India's silent crisis as the maximum number of malnourished children less than five years of age and related deaths twice to that of sub-Saharan Africa are reported here.[1] Of the total 132 million children under 5 years of age, it is observed that around nine millions are suffering from severe acute malnutrition (SAM). This accounts for nearly 50% of children with SAM across the whole world. Also, India inhabits more than one-third of the world's undernourished children; i.e, around 146 million. In India, more than five millions children are estimated to die every year as a result of undernutrition directly or indirec tly due to which succumbs in the loss of one child every ten seconds.[2]

Childhood malnutrition is an important public health issue and development challenge in India. It is also one of the leading causes of morbidity and mortality in children less than 5 years of age in developing countries. Apart from increasing the risk of disease and death, under nutrition also leads to growth delay and compromised psychosocial and cognitive development. In India, the prevalence of severe acute malnutrition in children remains high regardless of the overall economic growth and development.[2]

SAM is the main public health problem, which affects around 7.5% of under-five children in India according to National Family Health Survey-4 (NFHS-4, 2015-16).[3]

India's third National Family Health Survey (NFHS-3, 2005-06) indicated that the prevalence of severe wasting was 7.9% as per World Health Organisation

(WHO) Child Growth Standards.[1] Thus, at any point in time, an average eight million Indian children under five years are severely wasted[4] and are hazardously undernourished to survive, grow and develop to their full potential in adulthood.

SAM remains a major killer of children as mortality rates in children with severe wasting is high. Children with SAM are at nine times higher risk of death as compared to well-nourished children.[6]

SAM mainly affects preschool-age children, mostly from the WHO South-East Asia Region and African Region.[7]In the year 2016, it has been estimated that approximately 52 million children less than five years of age suffered from acute malnutrition and 17 million suffered from SAM with Southern regions of Asia and Sub-Saharan Africa having maximum number of severely acute malnourished children.[8]

In the year 2011, it has been estimated that twenty million children worldwide suffered from SAM with less than two million children received treatment and around one million children die each year with SAM.[9]

Severely malnourished children are more prone to long term developmental delay and have weakened immunity making them susceptible to infections as compared to well-nourished children.[10]SAM can be a direct cause of child death, or it can act as an indirect cause by increasing the case fatality rate in children suffering from common childhood illnesses such as diarrhoea, pneumonia or measles. Children with SAM can suffer consequences for their future health, learning problems and reduced performance in adulthood.[11]

SAM results in numerous pathophysiological changes in the body systems including alterations in hematological parameters. Low red cell counts resulting in anaemia has always been a consistent feature of protein energy malnutrition(PEM) and may be normochromic normocytic, microcytic hypochromic or macrocytic.[12,13] White blood cell changes also demonstrates the synergistic relationship between SAM and infections.[14]

The purpose of this study is to understand the changes in haematological profile, biochemical profile and anthropometric assessment of SAM children. This study will help in the proper diagnosis and appropriate management of SAM children and the results can be further generalised to whole paediatric population.

OBJECTIVES

PRIMARY OBJECTIVE:

- To compare the haematological profile among children with severe acute malnutrition and healthy or well nourished children.
- To compare the biochemical profile among children with severe acute malnutrition and healthy or well nourished children.

SECONDARY OBJECTIVE:

- Comparison of anthropometric parameters in children with severe acute malnutrition and healthy or well nourished children.

REVIEW OF LITERATURE

MALNUTRITION :

Malnutrition is a disease created by humans and is usually found to "*start in the womb and ends in the tomb.*"[15] It is a pathological state which results from an abnormal intake of nutrients. An inadequate dietary intake of energy and protein results in under nutrition or protein energy malnutrition and an excessive energy intake results in over-nutrition. Thus, the term malnutrition may refer to under nutrition or over nutrition.[16]

Malnutrition and the associated retarding influences like infections, insanitary environment and lack of hygeine causes a lot of illness, reduced growth , delayed development and death among children. Most of the children in India living below poverty line usually suffers from starvation and multi-deprivation leading to physical and developmental retardation. It has been evaluated that around eighty million children, i.e., about sixty-five percent children under five years of age in India suffered from the ill effects of varying degrees of malnutrition.

Socio-demographic factors like lack of child spacing, large household and disregard of the female child have an unfavourable impact on child survival and development. Environmental factors like low education of parents, poor socioeconomic status, improper sanitation and improper child rearing practices causes reduced growth and development of children. Nutritional factors like improper breastfeeding practices, weaning practices and diet during illness also influences the growth and development of children. There are other important factors like maternal malnutrition, low birth weight and repeated infections that predisposes to malnutrition.[17]

Historical perspective

The term 'Malnutrition' is derived from the Latin words '*mal'* and *'nutritic'*, meaning ill and nourishment.[18] It is assumed that *Hippocrates*, the Father of Medicine, paid stringent attention to the nourishment of his patients as an important part of their treatment . His nutritional medicaments revealed a proximity of effects of individual foods on healthy as well as sick individuals. He advised consuming pulses along with cereals and heavy labor work with little drink to obese people. Remedial foods were recommended for pyrexia, 'hot intestines', diarrhea, melancholic syndromes etc. "Aahara" or the dietary philosophy remained crucial to the system of ancient Indian system of medicine, Ayurveda. Thus, ancient Indian literature provides indication about the significance of diet and nutrition. Count Rumond in the year 1795 coined the phrase "Science of nutrition" in an essay on feeding poor people. [19]

The knowledge of nutrition radiated in the 20th century when the importance of vitamins and amino acids was studied. The nutritional necessities of humans were proven during this period and the association between nutrition, diet and the human body in health and disease were documented.[19] Kwashiorkor was first described by Ciceley Williams (England) in 1933 which meant red boy in the "Ga language" of the Accra region (Ghana).[19,20] K. Fronuis and Cravioto also contributed significantly on protein calorie deficiency. D. Jelliffe, a pioneer in nutrition acquaint with the term "protein calorie malnutrition", which was later modified to protein energy malnutrition and the same was adopted by the FAO/WHO committee in 1971 to describe both kwashiorkor and marasmus.[19]

BURDEN OF MALNUTRITION:

Childhood Malnutrition is of significance because of its significant contribution to mortality and morbidity. Childhood is a period of rapid growth and development which requires a high protein and energy intake and failure to meet them results in malnutrition. Malnutrition in turn makes children more vulnerable to infectious diseases and is a contributing factor currently associated with more than one third of all deaths among children below the age of five years across the whole world.[20]

Malnutrition in the world

According to WHO Global Health Observatory (GHO) data 2017, 151 million (22%) children under five years of age were stunted; 51 million (7.5%) of under five years of age were wasted, of which 17 million were severely wasted. Around 45% of deaths among children under the age group of five years are mainly linked to under nutrition. These occurs mostly in low and middle-income countries.[22,22]

There are mainly two forms of malnutrition through which it is expressed; stunting and wasting. In the recent years, stunting is declining too slowly, i.e, 198.4 million (2000) to 151 million (2017), while wasting still affects the lives of too many young children (51 million), thus keeping the high rates of malnutrition. [22]

Overview of malnutrition in developed countries

Though the magnitude of malnutrition is comparatively less than that of the developing countries, the effects on the child is the same. In North America, 0.5 million and 0.1 million children under the age of five were stunted and wasted. About

5 million and 0.7 million under five children are stunted and wasted in Latin America and Carribean.[22]

Overview of malnutrition in developing countries

It has been found that around 19 million preschool children, typically from the regions of South-East Asia and Africa, are suffering from severe wasting. Under nutrition in children is a key health problem worldwide contributing to childhood illness, diminished intellectual development and suboptimal work capability associated with anaemia in adulthood.[23] Of the 7.6 million deaths occuring every year in children less than five years of age,[24] approximately 35% are due to nourishment associated factors and 4.4% of deaths have been shown to be explicitly due to severe wasting. [23]

According to UNICEF, WHO, World Bank Group joint malnutrition estimates, Africa and Asia bears the greatest share of all forms of malnutrition. In 2017, more than half of all stunted children under 5 years of age lived in Asia (83.6 million,i.e., 55%) and more than one third lived in Africa (58.7 million,i.e., 39%). In 2017, more than two thirds of all wasted children under 5 years of age lived in Asia (35 million,i.e., 69%) and more than one quarter lived in Africa (13.8 million,i.e.,27%).[22]

Asia

The stunted rate among under-five-children in Asian region is 83.6 million. Two out of five stunted children in the world live in Southern Asia,i.e., 58.7 million of under five children are stunted in southern Asia. South East Asia constitutes 14.9

million, followed by Eastern Asia (4.8 million), Western Asia (4.2 million) and Central Asia (0.9 million).[22]

India

India is also one of the developing countries where under nutrition in children less than five years of age is a major public health problem. This is revealed by the statistics that in India, under-weight children are among the maximum in the whole world, and is exactly double to that of Sub-Saharan Africa.[25,26] In spite of economic development and growth in India, the frequency of severe wasting among children is increasing.[27]

SAM is a foremost public health concern, which affects 7.5% of under-five children in India according to NFHS-4(2016-17) survey.[28]

According to NFHS-3 (2005-06), 7.9% of under-five children in India suffered from SAM.[29] The NFHS-3 also documented the prevalence of severe underweight, and severe stunting among children in India as 16% and 24%, respectively.[30]

Karnataka

In Karnataka, 36.6% of under-five children are stunted, 35.2% are underweight, 26.1% are wasted and 10.5% are severely wasted.[31]

Belgaum

36.7% of under five children are stunted, 31.7% are wasted with 16% of them severely wasted and 38.5% are underweight.[32]

ETIOLOGY OF UNDERNUTRITION:[33]

1. Primary causes: It includes lack of adequate intake of food which can be due to following:

a. Poverty.
b. Ignorance.
c. Food fads- reservations for eating specific foods.
d. Traditional habits- continuation of breast milk without introduction of complementary feeding at appropriate age, use of over diluted food formulas and restriction of food intake during periods of certain illness like diarrhea.
e. Social and cultural factors- males are given more food than females.
f. Congenital defects like cleft palate and cleft lip, which interferes with food intake.
g. Intrauterine growth retardation (IUGR) and maternal malnutrition predispose child to under nutrition later in life.

2. Secondary causes : It includes the following factors which leads to undernutrition despite adequate intake of food.

a. Chronic illness and infections like tuberculosis, human immunodeficiency virus (HIV), chronic diarrhoea, pneumonia.
b. Increased metabolic needs and lack of appetite.
c. Malabsorption/ impaired utilization.
d. Excessive loss - Gastroenteritis.
e. Drugs- Isoniazid predisposes to pyridoxine deficiency, phenytoin predisposes to folic acid deficiency.

Overpopulation is another risk factor associated with under nutrition and is seen commonly in developing countries. This can reduce production of foods, leading to insufficient food intake or intake of foods with deprived nutritional quality. On the other end, the effects of malnutrition on individuals can create and maintain poverty, which can additionally weigh down economic and social development. Both malnutrition and poverty together makes the individual more prone to disease. This vicious cycle of poverty and illness can end in death.[34,35]

Classification of Malnutrition:

Malnutrition can be classified biochemically, clinically (qualitatively) or anthropometrically (quantitatively). For quantification of malnutrition in communities and for planning preventive measures, a quantitative classification according to the severity of malnutrition is required. Anthropometry is used to provide this classification.[36]

A. Anthropometric Classification of Malnutrition:

Malnutrition in childhood is classified anthropometrically into 3 forms:[36,37]

1. Wasting – In wasting, the child has a low weight as equated to that expected of a healthy child of the similar height and sex. It usually results from acute malnutrition[36] and sometimes it may result from an acute shortage of food as seen in emergency settings such as wars and natural disasters. In non emergency settings, it can result from underlying medical conditions.[37]

2. Stunting – In stunting, the child has a low height for that particular age and sex. It is defined as failure to achieve the biological potential for growth which results from chronic malnutrition or past malnutrition.[36,37]

3. Underweight – A child is said to be underweight when it has a low weight as compared to healthy children of the similar age and sex. It may be due to wasting or stunting or both and is thus a composite of wasting and stunting.[36]

B. Clinical Classification of Malnutrition:

Different clinical classifications of PEM are available. It is usually classified according to weight for age.

Acute malnutrition is usually classified according to weight for height while chronic malnutrition is classified according to height for age.[38]

a) Classification according to weight for age: Weight for age is the most frequently used parameter to classify nutritional status.

i) **Gomez's classification**: Gomez and his colleagues provided the first classification of malnutrition which came in 1956.The disadvantage of this classification is it does not tells whether the malnutrition is of recent or past onset. It has three degrees.

Table 1: Gomez's classification according to weight for age:[38]

Nutritional status	**Weight for age (Harvard)**
Normal	>90 % of expected
First degree Protein energy malnutrition	75-90 % of expected
Second degree Protein energy malnutrition	60-75 % of expected
Third degree Protein energy malnutrition*	<60 % of expected

*All cases with oedema should be included in third degree Protein energy malnutrition irrespective of weight for age.

ii) **Jelliffe's classification:** It was proposed in the year 1965.

Table 2:Jelliffe's classification according to weight for age:[38]

Nutritional status (Harvard)	Weight for age
Normal	>90 % of expected
First degree Protein energy malnutrition	80-90 % of expected
Second degree Protein energy malnutrition	70-80 % of expected
Third degree Protein energy malnutrition	60-70 % of expected
Fourth degree Protein energy malnutrition	<60 % of expected

iii) **Wellcome Trust or International classification:** The Wellcome Trust provided a clinical classification in the year 1970 which is based on the percentage of expected weight for age and whether the oedema is present or absent. The disadvantage of this classification is that it does not takes account of stunting and hence the duration of malnutrition.

Table 3: Wellcome Trust classification of malnutrition:[39]

Weight for age (Boston)	Oedema	Clinical type of PEM
60-80 % of expected	+	Kwashiorkor
60-80 % of expected	-	Underweight
<60 % of expected	-	Marasmus
<60 % of expected	+	Marasmic-kwashiorkor

iv.)**The IAP classification of malnutrition (1972):**

The National Sub-committee of the Indian Academy of Paediatrics has accepted the standard value (100%) of 50th percentile of Harvard Standard. This classifies PEM into four grades which are as follows:

TABLE 4– IAP classification of malnutrition:[39]

Nutritional status*	**Weight for age**
Normal	>80 % of expected
Grade I Protein energy malnutrition	71-80 % of expected
Grade II Protein energy malnutrition	61-70 % of expected
Grade III Protein energy malnutrition	51-60 % of expected
Grade IV Protein energy malnutrition	< 50 % of expected

* If the patient has oedema of nutritional origin, the letter "K" is placed along with the grade of PEM in order to denote kwashiorkor.

b.)Classification according to height for age:

In the year in 1972, McLaren from Beirut and Water low from London, individually gave the height for age classification and weight for height classification to indicate stunting and wasting respectively.

Table 5 - Classification based on height for age :[38]

Height for age	**Waterlow's**	**McLaren's**	**Visweshwara Rao's**
Normal	>95% of expected	> 93 of expected	> 90% of expected
First degree	90-95% of expected	80-93% of expected	80-90% of expected (stunting/short*)
Second degree	85-90% of expected	-	- (stunting)
Third degree	< 85% of expected	< 80% of expected	< 80% of expected (stunting/dwarf*)

*Terminology used in McLaren's classification

c) Classification according to weight for height: It is used to grade wasting. Wasting indicates recent or acute malnutrition.

Table 6: Classification according to weight for height:[38]

Weight for height	Waterlow's classification	McLaren's classification
Normal	> 90 % of expected	> 90 % of expected
First degree wasting/	80-90% of expected	85-90% of expected mild wasting*
Second degree wasting	70-80% of expected	75-85% of expected moderate wasting
Third degree wasting/	< 70% of expected	< 75% of expected severe wasting*

*Terminology used in McLaren's classification

d.)WHO Classification:

It is based on weight for height (wasting), height for age (stunting) and presence of oedema. The WHO recommends the use of Z scores or standard deviation (SD) scores to indicate how far away the child's measurement falls from the median (Z score = 0). The calculations are based on the Z-score percentile charts developed by the WHO.[39]

WHO cut-off for assessment of protein energy malnutrition in community: The WHO cut-off to estimate malnutrition in population analysis is the mean value minus two standard deviations (SD). As adopted from Waterlow's classification, the combined position of two indicators, i.e., weight for height and height for age distinguishes between wasting caused by acute malnutrition and stunting caused by chronic malnutrition (Table 7).

Table 7.WHO-cut-off for assessment of PEM in the community[38]

Cut-off	H/A	W/H	H/A & W/H
> Mean - 2 SD	Normal	Normal	Normal
< Mean - 2 SD	Stunted	Wasted	Stunted & wasted

H/A - Height for age, W/H - Weight for height

e) Standard deviation (SD) score/Z score:

The SD score is used in population studies. Percentage of the median is calculated first to interpret data at population level and Z score is then calculated.

Practically, 80% of the reference median for weight for age and weight for height and 90% for height for age correspond to 2 SD below the median. Third centile corresponds to median minus 2 SD.[38]

f) **WHO classification of malnutrition:**[38]

i. **Acute and chronic malnutrition:**

Table 8 - WHO classification for acute and chronic malnutrition:

W/A	H/A	W/H	Interpretation
Normal	Normal	Normal	Normal
Decreased	Normal	Decreased	Acute malnutrition
Decreased	Decreased	Normal	Chronic malnutrition
Decreased	Decreased	Decreased	Acute-on-chronic malnutrition

ii. **Moderate and severe under nutrition:**

Table 9: WHO classification for moderate and severe malnutrition:

Features	**Moderate**	**Severe**
Oedema	No	Yes
Weight-for-height (wasting)	70-79%	< 70%
Height-for-age (stunting)	85-89%	< 85%.

Spectrum of Protein Energy Malnutrition:

It comprises of severe as well as mild forms of Protein energy malnutrition. The severe forms of Protein energy malnutrition are marasmus, kwashiorkor and marasmic-kwashiorkor.[40]

WHO Guidelines for the Identification of Severe Acute Malnutrition (SAM):[41]

In the year 2009, the WHO and UNICEF defined SAM for the purpose of categorizing children who are at greatest danger of mortality due to severe wasting and who would benefit the most from special nutritional therapy. The diagnostic norms for SAM children aged 6 to 60 months are as follows:

1. Weight for height Z score should be less than -3 SD of the 2006 WHO Child Growth Standards

OR

2. Mid Upper Arm Circumference less than 115 mm

OR

3. Bilateral pedal oedema.

If any one of these criteria are fulfilled then a child is classified as having severe acute malnutrition (SAM). The above definition of SAM is a modification of the 1999 WHO definition of severe malnutrition.

Types of Severe Acute Malnutrition: It can take two extreme forms, i.e, marasmus and kwashiorkor. Out of these, marasmus is by far the more common and less serious, the maximum mortality being found when both are present together, i.e, marasmic-kwashiorkor.

A) **Kwashiorkor:** It was first documented by Professor Cicely Williams from Gold Coast in the year 1933 who ascribed it to protein deficiency. She concluded that this was the disease of the first child when the second was on the way shifting the first child from the breast and named it kwashiorkor. The term "**Kwashiorkar**" is taken from the "**Ga language"** of Ghana, which means 'kwa-ni-oshi korkor' inferring *'pretend not to mind the korkor (second one)*', the disease of the first child; 'red boy', owing to the typical pigmentary changes, was however alternative term for kwashiorkor.[40] Later on, the term was assumed as the disease of the 'deposed child', when the second one is born.[41]

Certain other researchers identified a disorder similar to kwashiorkor with bulging cheeks and oedema and recommended the term '*sugar baby*' in order to point towards the dietary origin of the disease. A classical case of kwashiorkor is typically miserable, lack of interest in the surrounding, reduced growth, and has oedema, enlarged liver, anaemia, hair and skin changes.[40]

The kwashiorkor is characterised by the triad of growth retardation, odema and mental changes.[40]

Grading of kwashiorkor :[40]

Grade 1 - Pedal oedema.

Grade 2 - Grade 1along with oedema of the face.

Grade 3 - Grade 2 along with oedema of chest wall and back.

Grade 4 - Grade 3 along with ascites.

b) **Marasmus**: The term "marasmus" is originated from the Greek word *marasmos*, which means 'wasting'. The children suffering from marasmus shows extreme wasting and have an "old man appearance" with just skin and bones.[40] Marusmus is the commonest type of severe protein energy malnutrition that occurs in preschool children and results in growth retardation and muscle wasting without oedema.[43]

Predisposing Factors for Marasmus:[43]

• Early abrupt weaning.

• Intake of diet with inadequate calories and protein.

• lnfections- gastroenteritis.

• Malabaorption disorders.

Grading of marasmus:[40]

The wasting starts mainly in the brown fat since it is metabolically more active and is essential in thermogenesis. Children with marasmus usually have good appetite and are alert initially as compared to kwashiorkar. Later on these children may become irritable. Marasmus has been graded as follows :

Grade 1 - Wasting starts in the region of axilla and groin.

Grade 2 -Wasting extending to thigh and buttocks.

Grade 3 - Chest and abdomen.

Grade 4 - Wasting of buccal pad of fat also which is less active metabolically.

C) **Marasmic kwashiorkor:**When children suffering from marasmus develops oedema, it is referred to as marasmic kwashiorkor.[40] This condition is associated with features of both marasmus and kwashiorkor. The child is said to have this condition when all of the following features are present :

1. Weight <60% of the expected weight.
2. Oedema.
3. Marked wasting and stunting.
4. Anaemia.
5. Mental apathy.

Wasting is more obvious in the upper part of the body and oedema in the lower part of the body. Skin and hair changes associated with kwashiorkor may be seen.[42]

Clinical Features of Marasmus :[43]

Salient features of marasmus are as follows:

- Weight - decreased to less than or equal to 60% of expected.
- Muscle wasting- seen best at the temporalis and scapular muscles.
- Measurement of mid upper arm circumference also indicates muscle wasting.
- Growth retardation- leads to marked stunting.
- Loss of subcutaneous fat - leads to old man appearance. Loose folds of skin can be seen over the glutei and inner side of thigh.

Head-to-Toe Examination

1. Appearance :
   - Old-man appearance.
   - Cry is good.

2. Head :

• Microcephaly.

• Delayed closure of anterior fontanelle.

3. Neck :

• May appear long due to reduction of subcutaneous fat and wasting involving the muscles.

4. Abdomen :

• Scaphoid.

• Abdominal distension can be seen, due to diarrhoea causing hypokalaemia and decreased muscle tone.

5. Extremities :

• Thin due to wasting.

Complications of Marasmus :

- Hypoglycaemia.
- Hypothermia.
- Infections.
- Electrolyte imbalance.
- Growth retardation (both physical and mental).
- Behavioural changes.
- Poor school performance.
- Motor coordination affected.
- Intelligence is also affected.

Clinical Features Of Kwashiorkar:[43]

Changes should be identified in the early stages only and should be treated accordingly. One should not wait for the typical changes to occur, which may be seen only in florid kwashiorkor.

Features Usually Present

1. Growth retardation :

- Decreased weight - 60%-80% of the expected weight.
- Stunting –decreased height.

2. Oedema-generalised, pitting, fast oedema.

3. Behavioural changes :

- Irritability.
- Anorexia.
- Lethargy.
- Apathy.
- Cry- moaning.
- Lack of interest in the surroundings.

4. Hair changes - These include hair that are hypopigmented (blond hair, reddish or grey), thin, dry, brittle, lustureless, sparse and easily pluckable. Flag sign is positive. There are associated changes in texture and straightening of curly hair.

5. Skin changes- These are commonly seen on the areas exposed to continuous pressure and irritation.

- Bullous lesions.
- Bleeding purpura, petechiae and ecchymoses in severe cases.

- Dermatosis- It may be crazy pavement dermatosis/paddy field dermatosis/ flaky paint dermatosis.
- Erythema.
- Mucocutaneous lesions - smooth tongue and stomatitis.
- Infections - secondary skin infection may occur.

6. Muscular changes: Muscle wasting can be seen in the shoulders and upper arms. The presence of oedema may mask the muscle wasting.

7. CNS Changes: Mental changes in kwashiorkor are due to:

- Alteration in neurotransmitter synthesis and release.
- Brain oedema.
- Decreased calories.
- Degenerative changes and reduction in dendritic arborisation.
- Electolyte imbalances such as hypokalaemia and hypomagnesaemia.

8.Cardiac Changes :

- Anaemia.
- Congestive cardiac failure (cardiomegaly).
- Pericardial effusion (due to hypoproteinaemia).

Features Occasionally Present:

- Hepatomegaly due to fatty infiltration.
- Associated vitamin deficiencies like vitamin A deficiency.
- Pot belly due to hypotonia and weakness of the abdominal muscles.

Associated Problems:

- Diarrhoea.
- Respiratory infections.

Anthropometric Examination :[44]

Anthropometry is a simple and valuable tool for assessing the status of nutrition in children.

a) **Weight**: In children, weight is usually estimated via a beam scale or Salter type scale with pants in which the child is placed. The shoes are removed first and children is weighed with as little clothes as possible. The weight is either recorded directly or by balancing the beam once the beam reaches its balance point. For older children, the weight is measured correctly to approximately 500 gm and for smaller children to 100 gm.

b) **Height:** For children less than two years, a horizontal measuring rod or infantometer is used. Height measured in lying down posture is called length. Length is measured for children who cannot stand. The shoes are removed first and the child is placed on the back on a flat surface. One person, usually the mother, maintains the child's head against the fixed vertical head board with eyes of the child directed upwards. The other person firmly presses the knees together and down so that they touch the horizontal surface and then moves the mobile foot board so that it touches the heels when the feet are at right angle. Accuracy should be nearest to 0.5 cm.

For children more than two years, a vertical measuring rod or anthropometer is used. The child is allowed to stand without shoes and the heels, buttocks, shoulders and occiput lies in one plane touching the wall and the child is asked to look straight.

After that, the reading is taken directly after placing a horizontally held book or wooden board in order to touch the top of the head with hairs being flattened. The reading is measured accurately to the nearest 0.5 cm.

c) **Mid upper arm circumference (MUAC):** The circumference of the arm remains fairly constant in children between one to five years of age. By convention, it is usually taken on the left arm at the mid- point between the tip of the shoulder and the tip of the elbow. While recording MUAC, the measuring tape is held gently without pressing the soft tissues. The reading is measured accurately to 0.1 cm.

MUAC is a good test to identify children with risk of dying. But it is not suitable for continued growth monitoring as it increases very slowly during the age group of one to five years.

d) **Head circumference**: For assessing the head circumference, the maximum occipitofrontal circumference (OFC) is measured by keeping the non- stretchable and flexible tape over the occipital protuberance and frontal crests. The reading is then recorded to the nearest 0.1 cm.

e) **Chest circumference (CC)**: It is usually recorded at the level of nipple and is linked to occipitofrontal circumference (OFC).

Relationship between CC and OFC:

- Early infancy- OFC is more than CC.
- At one year of age- OFC becomes equal to CC.
- After one year of age- CC is more than OFC.

In children with PEM, chest circumference may continues to be reduced as compared to OFC, i.e., OFC to chest circumference ratio becomes more than one.

PATHOPHYSIOLOGY OF SEVERE ACUTE MALNUTRITION:[45]

The basic pathophysiological changes in severe acute malnutrition arises due to difference between the demand and supply of major nutrients and micronutrients. When the child's intake is compromised, the metabolic and physiological changes takes place to conserve energy and prolong life. This process of slowing down is called as reductive adaptation and includes reduced physical activity and growth, reduced basal metabolism and decreased immune and inflammatory response.

During the initial stages, the fat stores are mobilized to meet the demand. When these fat stores gets exhausted along with ongoing imbalance, the proteins are also mobilized from various organs like muscles, skin, and gastrointestinal tract. During this period, the body tries to conserve energy by limiting the use. Finally, these physiological and metabolic derangements leads to various consequences and are responsible for clinical features and complications seen in SAM children.

Various studies have been done and theories have been brought forward to explain the pathogenesis of severe acute malnutrition. The important ones are :

1. **Gopalan's Theory of Adaptation :**

Some children develop marusmus while others develop kwashiorkor even though there is no quantitative and qualitative differences in their diet. This is explained by the Gopalan's theory which says that marasmus develops due to good adaptation to poor diet while kwashiorkor is the result of adaptation failure.

In marasmus, decreased calorie intake leads to decreased insulin levels and increased cortisol levels. Increased cortisol level in turn leads to tissue catabolism, which causes muscle wasting. In this process, glucose and amino acids are released

into the circulation. The glucose is utilised by the brain and amino acids are required and utilised for the synthesis of albumin and beta-lipoproteins. Thus, the albumin level is maintained in the blood and hence no oedema occurs in marasmus. The beta lipoprotein helps in mobilising the fat from the liver; hence, the fat is not accumulated in the liver. In kwashiorkor, there is increase in insulin level which prevents tissue catabolism. Hence, the amino acids are not available for albumin synthesis and beta lipoproteins are not formed. Hence, oedema and fatty liver develops.

2. **Golden's Theory of Free Radicals :**

According to this theory, free radicals has a significant role in the pathogenesis of oedema in cases of protein energy malnutrition. The depletion of the antioxidants leads to kwashiorkor and therefore antioxidant supplementation can prevent kwashiorkar.

Investigations in Kwashiorkar :[46]

In kwashiorkor, the diagnosis is mainly clinical and based on anthropometry. The investigations are usually done to find out associated problems.

1. To find out the associated infection :

- Blood culture (for sepsis).
- Peripheral smear for malaria.
- Stool examination for ova, cyst.
- Sputum examination or resting gastric juice examination for acid-fast bacilli of tuberculosis.

2. To find out the complications:

- Blood sugar (hypoglycaemia).

- Serum electrolytes (hypokalaemia, hypernatraemia or dilutional hyponatraemia).
- Chest X-ray (heart failure, bronchopneumonia).
- ECG (heart failure).

Investigations in Marasmus :[47]

- Complete blood counts.
- Serum proteins.
- Peripheral smear.
- Blood sugar.
- Serum electrolytes.
- Routine and microscopic examination of urine.
- Routine and microscopic examination of faeces.
- Investigations to find out associated infection & urine culture, blood culture, Mantoux test, chest X-ray.

Ferritin and its importance in SAM children:

Ferritin is the major intracellular iron storing protein and is also seen in small amounts in the blood circulation. The ferritin levels in the serum generally correlates with total body iron. However, it has been categorised as an acute phase reactant also and known to be increased by infections, inflammatory processes and some malignant conditions. It has also been observed that few conditions like vitamin C deficiency or scurvy can be a potential cause of low ferritin.[48]

The WHO recommends that a serum ferritin concentration of less than 12 ng/ml indicates depleted iron stores in children in less than 5 years of age, while a concentration more than 12 ng/ml indicates depleted iron stores in those more than 5

years of age.[49] However, both thresholds may be too low during an acute phase response or when there is chronic disease, and a serum ferritin concentration between 30 and 100 ng/ml may better represent depleted stores of iron in such conditions.[49,50]

Kapur D et al.(1997) conducted a population based study to measure the extent and etiology of anaemia and iron deficiency in children between 9 to 36 months of age. The results showed that the prevalence of anaemia using WHO cut-off values among children was 64% in children between 9-36 months of age and of these 7.8% had severe anaemia. Using 10.0 g/dl as the Hb cut-off point 44% children less than eighteen months of age in the present study population were anemic. On a sub-sample study, eighty-eight percent children were estimated to be iron deficient, with serum ferrtin concentration less than twelve µg/L. The red cell morphology on peripheral smear revealed 33.9% as microcytic hypochromic and 37.1% as dimorphic. 55% of children in moderate anaemia group had dimorphic anaemia. In this study the parasitic infection was not related to the prevalence or severity of anaemia.[51]

Later on, a descriptive study with controls conducted by **Mishra SK et al.(2007**) in children between 6 months to 59 months of age at Maharajgunj, Kathmandu, Nepal showed that the education level of parents of children with PEM was found to be significantly decreased as compared to their non-PEM counterparts. They found that mean serum glucose, sodium, potassium, cholesterol, haemoglobin was statistically insignificant in both groups while mean total protein, albumin, and calcium were statistically significant in both the groups. There was significantly higher frequency of hypoproteinemia and hypoalbuminaemia in PEM group when compared to control group.[52]

Aguayo VM et al. (2009–2011) in their study to evaluate the efficacy of current cut off of MUAC in the identification of children with severe acute malnutrition in India observed that children in the age group between six months and twenty three months represented around eighty percent of the subjects diagnosed with SAM. MUAC<115mm was as effective as WHZ<−3 SD in identifying 6–59-months old children with medical complications and is also the most important risk factor of death among oedema-free children. Furthermore, death rates in children with MUAC<115mm were higher than in children with WHZ<−3 SD and 91 % of the deaths among oedema free children were deaths of children with MUAC<115 mm.[53]

Another study conducted by **Thakur N. et al.** (**2010-11**) to estimate the prevalence and type of anaemia in severely acute malnourished children. They found that approximately 67.3 % SAM children in their study were having severe anaemia and 13.8% were having moderate anaemia. In this study, the most common type of anaemia observed was microcytic anaemia (38.6%) followed by megaloblastic anaemia (30.5%). Of these patients, 25% required packed red blood cells transfusion due to severe anaemia. [54]

A year later, **Basheir HM et al.(2011)**conducted a study with controls in Sudan to assess the haematological parameters in malnourished children less than five years of age. They observed that all the haematological parameters of children was considerably different as compared to controls. Haemoglobin levels, Serum iron and serum ferritin levels of malnourished children was significantly reduced as compared to controls. However, the platelet count and white blood cell counts of test group was significantly increased as compared to controls. They also found marasmus in 82% of malnourished children.[55]

During the same year, **Tadesse AW et al.(2011-12)** conducted a population based survey in children between 6-59 months to assess the usefulness of MUAC and weight-for-height (WHZ) in diagnosing severe acute malnutrition in rural districts of Ethopia. They used WHO criteria of MUAC less than 115 mm and WHZ less than -3 SD to define severe wasting . The kappa coefficient was then calculated. They found that there was fair agreement between the MUAC and WHZ definitions of severe wasting in boys and children less than one year of age but poor agreement in girls and children aged two years or more.[56]

A year later **Badi MA et al.(2012-13)** conducted a study over 622 children diagnosed with SAM. They observed that a higher number of patients were having marasmus (94.2%) and as compared to kwashiorkor (5.8%). In their study marasmus was seen commonly in less than 1 year of age (59.2%) while kwashiorkor was seen between 1 - 5 years (61.1%) patients. Males (54%) outnumbered females (46%) with a male to female ratio of 1.18:1 in SAM but no significant differences were observed in males and females. The main illnesses associated with SAM were gastroenteritis (71.1%) followed by pneumonia (16.6%). They concluded a high prevalence of SAM among male children who were less than 5 years of age with high risk of gastroenteritis and pneumonia thereby signifying the critical need to improve the nutritional status of children.[57]

Another cross-sectional study was conducted by **Bhadoria AS et al. (2012–14)** in Meerut district of Northern India to identify the prevalence of severe acute malnutrition (SAM) among children aged 6–60 months. Out of total 18,463 children enrolled in the study, the mean age of patient was 32.6 ± 15.4 months with majority of the patients as males (53.4%). Their results showed that the prevalence of SAM was

lower in their district (2.2%) as compared to the national prevalence (7.9%). They also concluded that younger age, nuclear family, lower parental education, and poor occupation of the head of the family predisposes a child to SAM.[58]

Another study by **Gohain EK et al.(2013-14)** in Gauhati Medical College showed that anaemia was a constant feature of Protein energy malnutrition (PEM) in the age group of 6 months to 59 months of age. The cases have lower mean value of hemoglobin and hematocrit as compared to controls. They found that children with PEM were having statistically significant lower mean values for RBC indices like RBC count, MCH and MCHC as compared to the controls. In their study, they observed noteworthy leucocytosis, neutrophilia and thrombocytosis among children with PEM as compared to controls. Ninety-one percent of the children with PEM are anaemic and most of them were moderate to severely anaemic (73.6%). The cases had almost equal distribution of microcytic(34%), macrocytic(32.9%) and normocytic (31.8%) anaemia whereas in the controls most common anaemia found was microcytic anaemia(46.8%).[59]

A year later in **2014**, **Dukhi N et al.** conducted a survey in South Africa by means of WHO child growth standard to compare MUAC and weight for height (W/H) as indices of acute malnutrition in children aged between 0–59 months of age. Their study showed that, out of all child participants, 7.7% were malnourished using W/H measurements in comparison to 6.6% using MUAC. There was around 54% agreement between the two indices when considered in SD bands and the significant kappa statistic value of 0.27 constituted fair agreement. Similar percentages of male (1.1%) and female (1.1%) children under five years of age were detected with severe acute malnutrition (SAM) using W/H. In children aged less than months W/H identified ten children as malnourished compared to only one child identified as

malnourished using MUAC. They concluded W/H (7.7%) as a more sensitive measure of child malnutrition and measured more than twice for the children with SAM compared to MUAC. W/H also appeared as a more reliable indicator in children less than one year old as compared to MUAC but in age group of three to less than five years, a disagreement appeared whereby MUAC was a more sensitive measure than W/H as the child's age is increased.[60]

The study conducted by **Agarwal A et al. (2014)** in Mumbai to know the haematological profile and anthropometric assessment showed that out of total 77 children enrolled in the study, 47 were males and 30 were females. The results showed that 21 children (27.27%) were anemic with a Hb of less than 11.5%. 65 children (84.41%) had normal leucocyte count for their reference range, whereas 3 children (3.91%) suffered from leucopenia and 9 (11.68%) from leucocytosis. 57 children (74.02%) had markedly increased absolute eosinophilic counts (AECs) as compared to only 20 children(25.97%) with normal AECs. 53 children(68.83%) had normal platelet count while 23 children(29.87%) showed evidence of thrombocytosis. The results of anthropometry showed that 18 children belonged to Grade 1, 28 children for Grade 2, 11 children for Grade 3 and 3 children for Grade 4 Protein energy malnutrition according to the IAP.[61]

Arya AK et al.(2014-2015) later on conducted a case control study to understand the haematological profile of severely malnourished in Kanpur. They found that 95% of the SAM children enrolled in their study had anaemia, out of which around 52% were severely anaemic and 28% were moderately anaemic. They also observed that the children with SAM had statistically significant lower mean values for haemoglobin, red cell indices like RBC counts, MCV, MCH and MCHC as

compared to controls. The mean value of white blood cells in SAM children was increased as compared to controls. The cases also had higher mean value for neutrophils and lower mean value for lymphocytes.[62]

Chizoba ON et al. conducted a study to know the prevalence of severe acute malnutrition and the prevalence of wasting, stunting and underweight in children aged 6 - 60 months from the medical records of the Mother of Christ hospital Ogui, Enugu. They observed that SAM mainly occurs in families that have limited access to nutritious food and are living in insanitary conditions, which increases risk of repeated infections. Malnutrition was common in the initial two years of life as a result of inadequate breastfeeding of the child and increased rate of infection. The study also revealed that large family size had adverse impact on the nutritional status of the children as compared to those with small family size. [63]

Yaikhomba T et al. in their study over 50 SAM children aged between 6 months to 5 years to evaluate iron, folate and vitamin B_{12} status in hospitalized children. They found that 78 %SAM children had weight for height Z score<−3SD, 72 % had MUAC<11.5 cm and 22% of them had oedematous malnutrition. Anaemia was prevalent in 94 % of SAM children and there was significant correlation between weight for height Z score<−3SD and vitamin B_{12} deficiency. Significantly, majority of the SAM children were having vitamin B_{12} deficiency as compared to folate and iron deficiency.[64]

Choudhary M et al. conducted a prospective study to know the clinical profile of SAM children in Rajasthan. The results showed that incidence of SAM was 3.28%. The average age of admitted patients was 14.92 ± 7.48 months. In their study, they found that 41.3%, 32.1%, 21.3% and 5.3% patients were in PEM grade IV, III, II

and I respectively. Female patients outnumbered males (84.2% v/s 68.21%). The most common presenting complaint was pyrexia followed by vomiting and other illness associated with PEM was gastrointestinal (60%) followed by respiratory tract infection (52%). The average duration of exclusive breastfeeding was 2.6 ± 1.5 months and mean age of weaning was 8.4±3.9 months.78.7% children were still on breast feed at the time of hospitalisation and among them 40.7% of children were above twelve months of age. They also observed that most of the caretakers were uneducated and in all cases, caretakers were mothers.[65]

Pravana NK et al. (July-Dec 2014) conducted a case control study in Nepal to evaluate the determinants of SAM among children aged between six months and fifty- nine months. Their study results showed that the prevalence of SAM in children less than five years of age was 4.14%. They observed that low socioeconomic status, age at birth less than 20 years or more than 35 years, birth interval of less than 24 months, illiterate father and bottle feeding were significantly associated with SAM. They found no significant differences with regards to mother's educational level, initiation of breastfeeding, colostrum feeding, and exclusive breastfeeding in between the groups.[66]

Another study was conducted by **Bobby J et al.** to know the prevalence of malnutrition in children aged 12- 60 months attending anganwadis in rural areas of Karnataka in South India. They also studied the importance of using various field-based formulae and various anthropometric indicators used for classification of malnutrition. The results showed that the prevalence of wasting, stunting, and wasting and stunting was 31.2%, 9.4%, and 29.2% respectively. Wasting was more predominant among the younger age groups. They concluded that to diagnose acute

malnutrition, the best indicator was a comparison with the reference weight calculated using Weech's formula and indicators such as MUAC needed to be used carefully since they are not sensitive for identification of all cases of malnutrition. But, MUAC-for height method could be used due to its increased sensitivity. They also concluded that for recognition of stunting, if reference tables are not accessible, Weech's formula can be used for calculation of expected height although the sensitivity of this indicator is not very high.[67]

Dakshayani B et al.(2016-17) conducted a cross-sectional observational study for estimation of serum electrolytes in SAM children between the age group of 1 month to 5 years . The results showed that the mean value of serum sodium was 134.58±5.45 meq/L, potassium was 4.29±0.75 meq/L, and chloride was 103.31±7.16 meq/L. Hyponatremia was seen in 43.4% and hypokalemia in 7.1% of children. They observed no significant differences in the mean values of serum electrolytes and frequency of hyponatremia and hypokalemia between groups. They concluded that dyselectrolytemia occurs in SAM children with and without complications and serum electrolyte levels may need to be measured in all SAM cases to detect asymptomatic hyponatremia and hypokalemia.[68]

MATERIALS AND METHODS

The present study entitled "**A COMPARATIVE STUDY TO KNOW THE HAEMATOLOGICAL PROFILE AND BIOCHEMICAL PROFILE AMONG CHILDREN WITH SEVERE ACUTE MALNUTRITION AND HEALTHY CHILDREN"** was conducted in the department of Pathology and KLES Dr. Prabhakar Kore Hospital and Medical research centre, Belagavi in the year 2017-18 .

TYPE OF STUDY: Prospective observational study.

DURATION OF STUDY: January 2018- December 2018.

STUDY DESIGN : Case control study.

STUDY POPULATION: This study was done on all admitted cases in the age group of 6 months to 5 years which were diagnosed as severely acute malnourished based on WHO classification at Nutrition Rehabilitation Centre, KLES Dr. Prabhakar Kore Hospital and Medical research centre, Belagavi from January 2018- December 2018. The age, sex and anthropometrically assessed healthy children, attending hospital for immunisation or from community were taken as controls. For every severely acute malnourished case, a control was taken. A detailed history including dietary, immunisation and socioeconomic status was obtained. Thorough clinical examination including anthropometry and relevant investigations were done and documented in preformed proforma.

INCLUSION CRITERIA:

1. All children diagnosed with severe acute malnutrition in between the age group of 6 months to 5 years were taken as cases.
2. Age matched, sex matched and anthropometrically assessed healthy children, attending hospital for immunisation or from community were taken as controls.
3. Consenting parents or child's guardian.

EXCLUSION CRITERIA:

1. Children less than 6 months or more than 5 years of age.
2. Unconsenting parents.
3. Children with mild and moderate malnutrition.
4. Children with lesions like lymphoma, tuberculosis, leukemia, dehydration, liver cirrhosis, nephrotic syndrome, cardiac failure or other systemic disease leading to weight loss were excluded from the study.
5. Children with chronic illnesses and history suggesting ongoing haemolysis and haemoglobinopathies.

METHODOLOGY: After taking written informed consent, detailed history, thorough clinical examination including anthropometry and relevant investigations was done in all cases and controls.

Under aseptic conditions, 5 ml of blood (venous blood) was collected in vacutainers, 2.5 ml in EDTA (Ethylene diamine tetra acetate) vacutainer for haematological investigations and 2.5 ml in plain vacutainer for biochemical investigations.

All the sample were then analyzed using an automated analyser. In subjects, where platelet count is less via automated analyser, it was verified by manual recounting in peripheral blood smear.

The following investigations were done in all cases and controls:

1. Haemoglobin estimation.
2. Packed cell volume.
3. Red blood cells count.
4. Total leucocyte count.
5. Platelet count.
6. RBC indices (Mean corpuscular volume, Mean corpuscular haemoglobin, Mean corpuscular haemoglobin concentration and Red cell Distribution width).
7. Serum albumin.
8. Serum total proteins.
9. Serum sodium and potassium.
10. Serum ferritin.

SAMPLING PROCEDURE:

Two groups were undertaken: Group A and B.

Group A and B constitutes samples from severely acute malnourished children and healthy or well nourished healthy children respectively and were analysed at Central haematology and biochemistry lab at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

SAMPLE SIZE: A total of 60 children in the age group of 6 months to 5 years of age were analysed with 30 cases in Group A and 30 controls in Group B.

Sample size was calculated as follows:

Sample size formula:

The minimum sample size formula based on mean and standard deviation is

$$n = \frac{(z_\alpha + z_\beta)^2(s_1^2 + s_2^2)}{(\bar{X}_1 - \bar{X}_2)^2}$$

where z_α is linked with the level of significance and z_β is linked with the power of the test. For 5% level of the significance z_α =1.96 and z_β =0.84 for 80% power of the test.

$\bar{X}_1$ is the mean of the first group (8.40) and $\bar{X}_2$ is the mean of the second group (9.98).

s_1 is the standard deviation of the first group (2.35) and s_2 is the standard deviation of the second group (1.54).

With these values the sample size, in each group, obtained is 25.

To make the study more confirmative, the sample size will be raised to 30.

Anthropometric Examination: The height/ length, weight of children, head circumference, chest circumference, mid upper arm circumference and weight for height was assessed by using growth standards given by World Health Organisation.

a) Weight. It has been measured using a beam scale or Salter type scale with pants in which the child is placed. It has been measured accurately to the nearest 100 gms.

b) Height: For children less than two years of age, a horizontal measuring rod or infantometer was used. For children more than two years of age, a vertical measuring rod or anthropometer is used. Accuracy has measured to the nearest 0.5 cm.

c) Mid upper arm circumference (MUAC): The reading was taken on the left arm, halfway between the tip of the shoulder and the tip of the elbow. The measuring tape was held gently without pressing the soft tissues. Accuracy was measured to the nearest 0.1 cm.

INVESTIGATIONS	**INSTRUMENT USED**
Complete blood counts	Sysmex XN-350 six part fully automated haematology analyser.
Serum sodium and potasium	Roche 9180 Electrolyte Analyser.
Serum albumin and total proteins	Falcon mini fully automated analyser byARK diagnostics Pvt. Limited.
Serum ferritin	Cobas E411 biochemistry analyser.

ETHICAL CONSIDERATION:

A written informed consent was obtained from parents/ guardian of both cases as well as control group before sample collection.

PHOTOGRAPHS

Photograph 1: Sysmex XN-350 six part fully automated hematology analyser for complete blood counts.

Photograph 2: Roche 9180 Electrolyte Analyzer by ARK diagnostics Pvt. Limited for the estimation of serum electrolytes.

Photograph 3: Falcon mini-fully Automated Random Access Chemistry Analyzerby ARK diagnostics Pvt. Limited for the estimation of serum albumin and total proteins.

Photograph 4:Cobas E411 biochemistry analyser for serum ferritin estimation.

RESULTS

In this study, a total of 60 children in the age group of 6 months to 60 months were enrolled with 30 cases diagnosed as severely acute malnourished children and 30 controls. Of the 30 cases, 10 cases (33.33%) each belonged to the age group of 13-24 months and 25-36 months, followed by 6 cases (20.0%) in 6-12 months, 3 cases (10.0%) in 37-48 months and 1 case (3.33%) in 49-60 months. The most common age group of the children that were enrolled in the study was between 13-36 months (66.67%).

Table 10: Age wise distribution of study subjects.

Age (in months)	No. of Participants	Percentage (%)
6-12	6	20
13-24	10	33.33
25-36	10	33.33
37 - 48	3	10.0
49 -60	1	3.33
Total	30	100.0

Graph 1: Age wise distribution of study subjects.

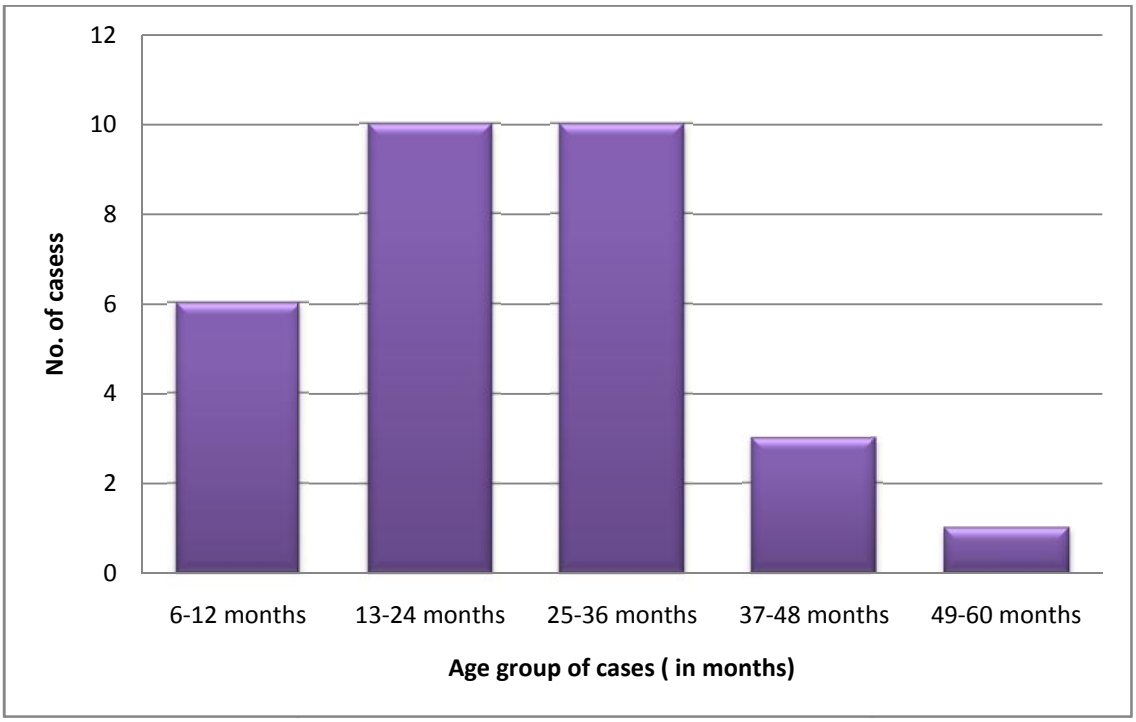


Table 11: Gender wise distribution of study subjects:

Gender	No. of Participants	Percentage (%)
Boys	14	46.67
Girls	16	53.33
Total	30	100.0

Majority of the study subjects were females , i.e, 16 (53.33 %) and the rest were males, i.e., 14 (46.67 %). The male : female ratio was 1:1.42.

Graph 2: Gender wise distribution of study subjects.

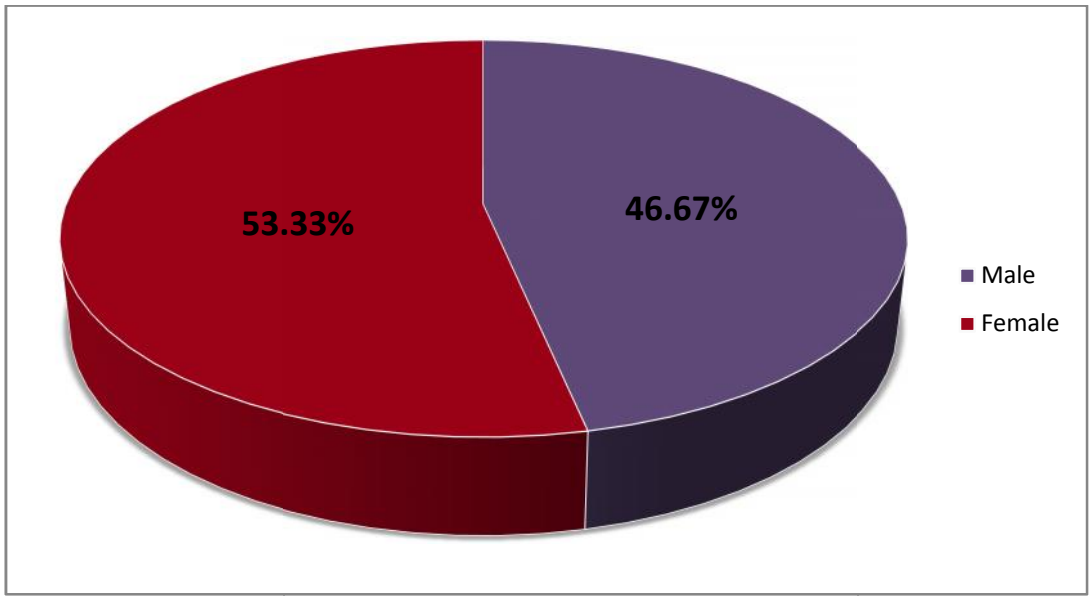


Table 12 : Comparison of mean of various anthropometric parameters among SAM cases and conrols.

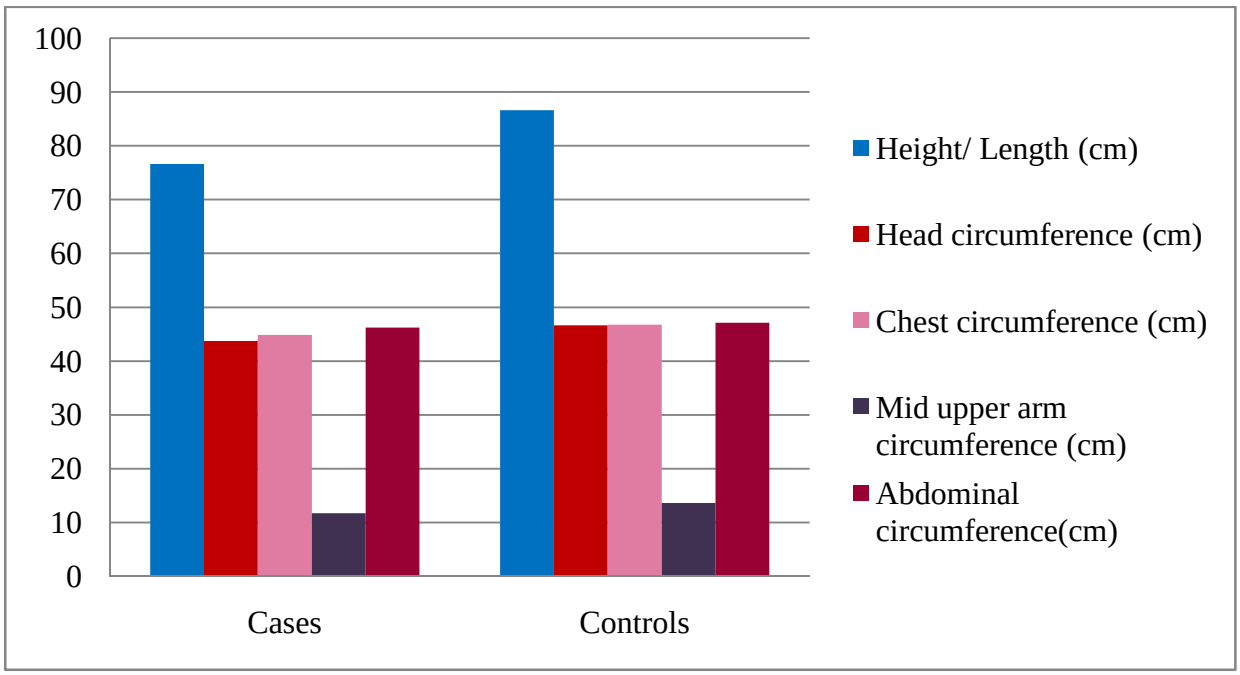
Anthropometric parameters	SAM mean ±SD	Control mean ±SD	p value*
Weight (kgs)	7.36±1.27	12.05±2.11	<0.0001
Height/ Length(cm)	76.63±6.58	86.60±8.56	<0.0001
Head circumference (cm)	43.72±1.95	46.62±1.59	<0.0001
Chest circumference (cm)	44.83±3.20	46.73±2.90	0.0190
Mid upper arm circumference (cm)	11.71±0.88	13.63±2.84	<0.0001
Abdominal circumference (cm)	46.21±3.81	47.09±2.84	0.3115
Weight for height (SD)	-3.59±0.70	0.00±0.97	<0.0001

In the above comparison the *p value is obtained using Student's Unpaired t Test.

The comparison of different anthropometric parameters among cases and controls is depicted in the table 3. The mean weight, mean height/length, mean head circumference, mean mid upper arm circumference and mean weight for height among cases and controls were statistically significant.

However, the mean chest circumference and mean abdominal circumference among cases and controls were statistically not significant.

Graph 3 : Comparison of mean height/length, head circumference, chest circumference, mid upper arm circumference and abdominal circumference among SAM cases and controls.



Graph 4 : Comparison of mean weight (in kgs) among SAM cases and controls.

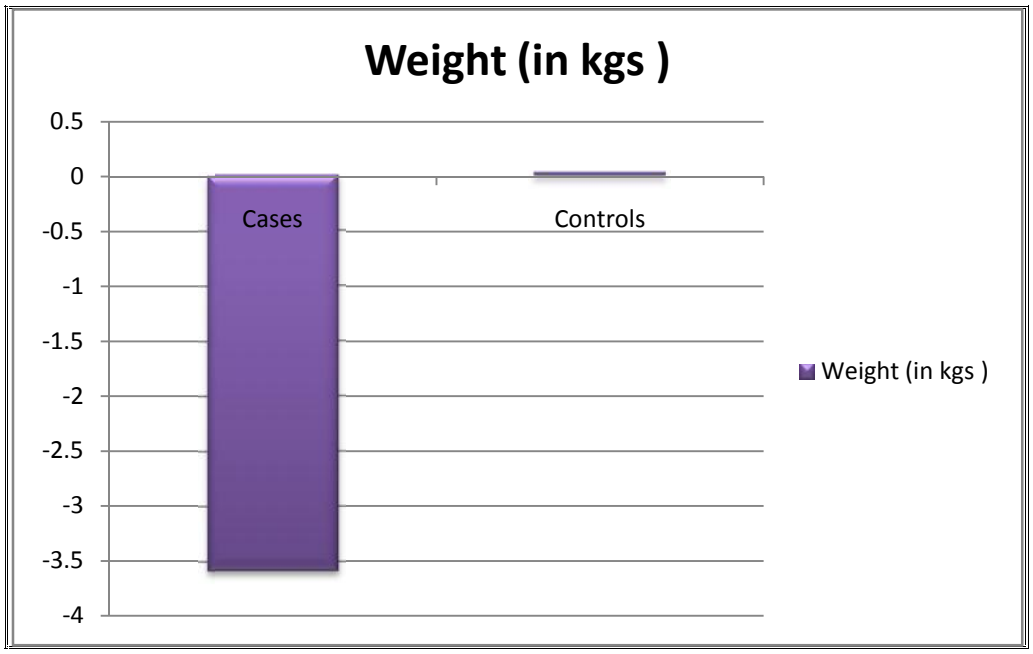


Table 13: Distribution of cases according to type of severe acute malnutrition.

Type of severe acute malnutrition	Number of cases	Percentage
Marasmus	27	90%
Kwashiorkar	1	3.3%
Marasmic-kwashiorkar	2	6.6%
Total	30	100%

Graph 5: Distribution of cases according to type of severe acute malnutrition.

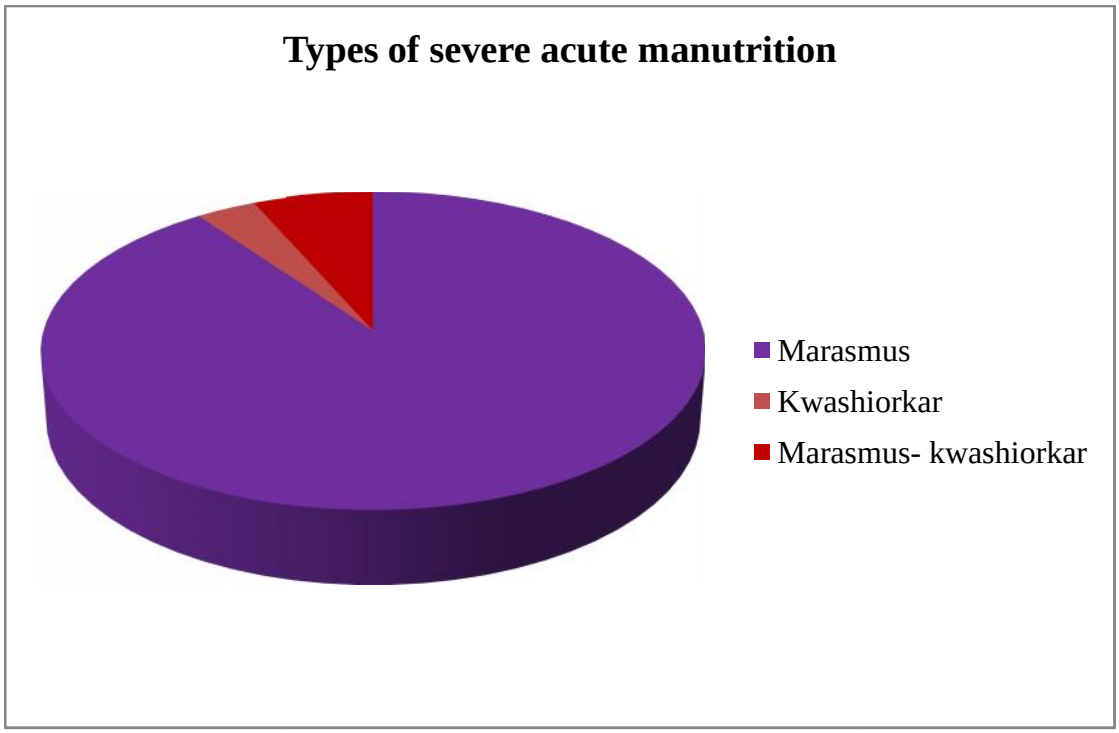


Table 14: Comparison of mean Haemoglobin (gm/dl) among cases and controls.

Group	Mean ± SD	p value*	Inference
Cases	8.33 ± 2.16	<0.0001	Highly Significant
Controls	12.55 ± 1.30		

In the above comparison the *p value is obtained using Student's Unpaired t Test.

Graph 6: Comparison of mean haemoglobin among cases and controls.

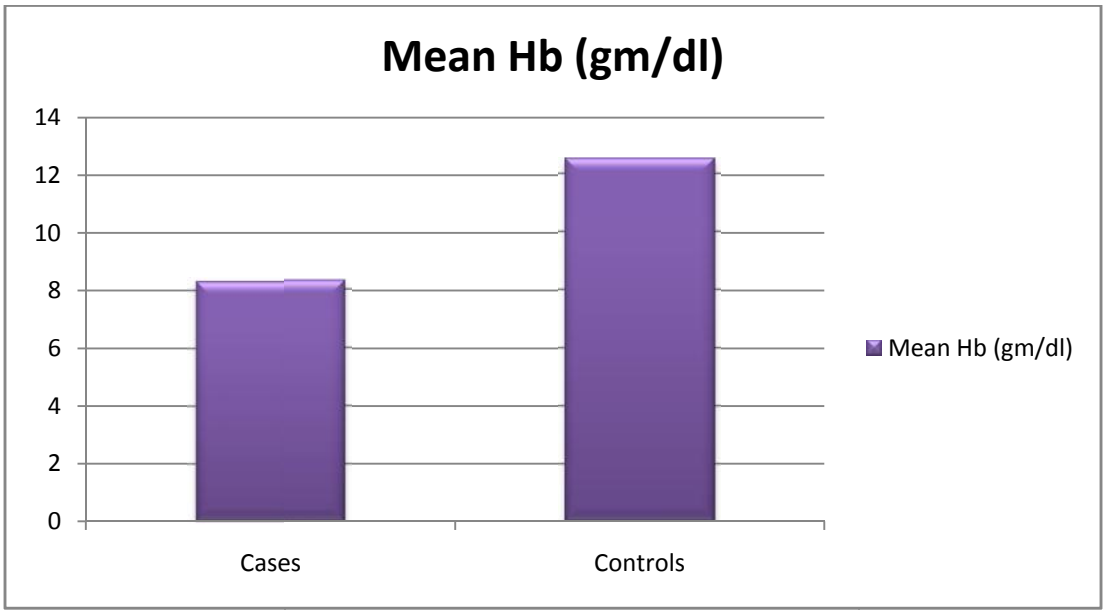


Table 15 : Comparison of mean packed cell volume (%) among cases & controls.

Group	Mean ± SD	p value*	Inference
Cases	28.11 ± 6.93	<0.0001	Highly Significant
Controls	39.04 ± 3.74		

In the above comparison the *p value is obtained using Student's Unpaired t Test.

Graph 7: Comparison of mean packed cell volume among cases & controls.

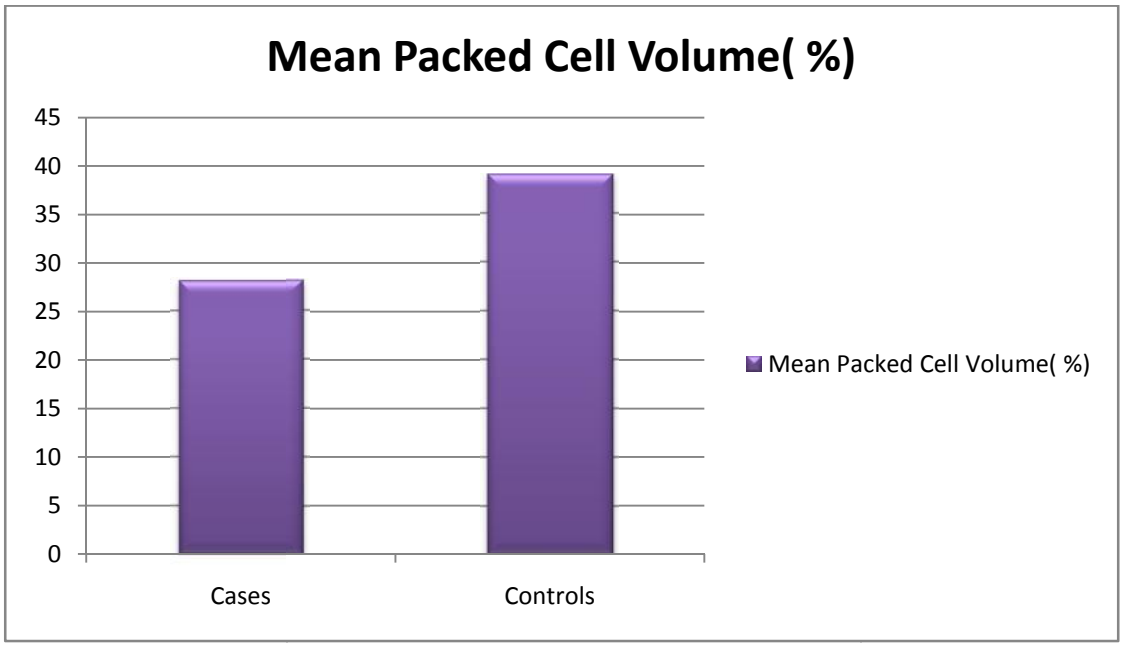


Table 16: Comparison of mean RBC count among cases & controls.

Group	Mean ± SD	p value*	Inference
Cases	4.30 ± 0.95	0.0863	Not significant
Controls	4.64 ± 0.49		

In the above comparison the *p value is obtained using Student's Unpaired t Test.

Graph 8: Comparison of mean RBC count among cases and controls.

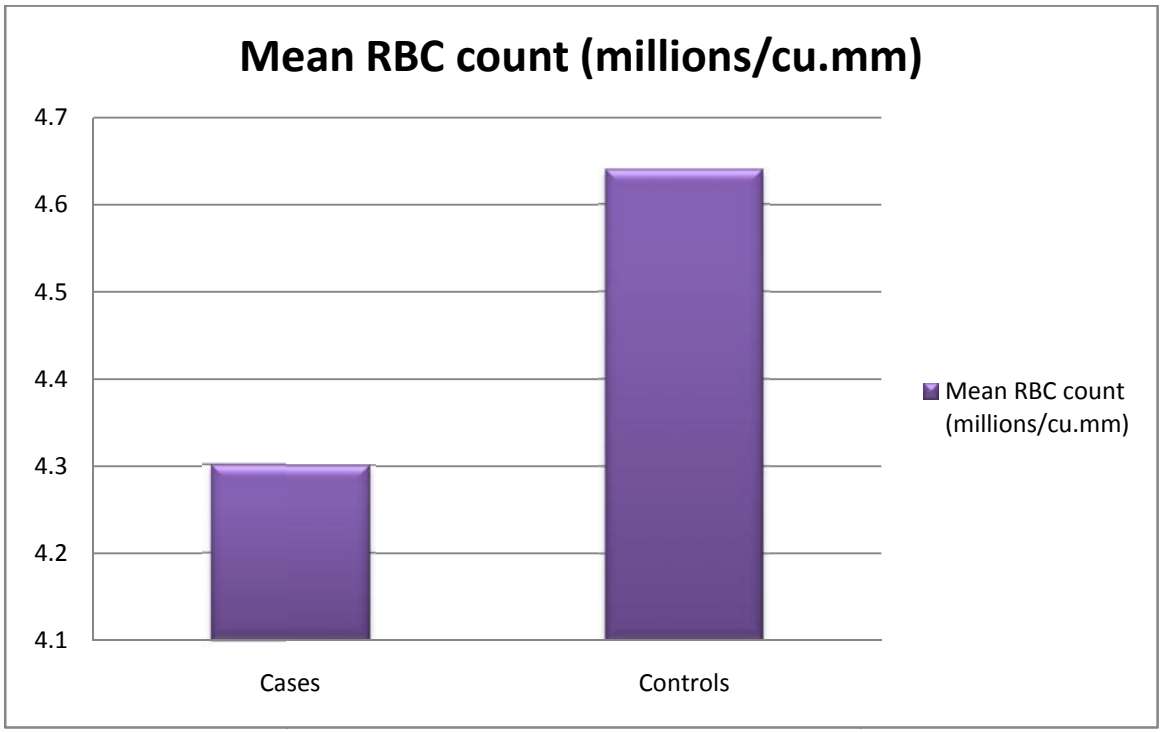


Table 17 : Comparison of mean reticulocyte count among cases & controls.

Group	Mean ± SD	p value*	Inference
Cases	0.94 ± 1.28	0.3609	Not significant
Controls	0.72 ± 0.12		

In the above comparison the *p value is obtained using Student's Unpaired t Test.

Graph 9 : Comparison of mean reticulocyte count among cases and controls.

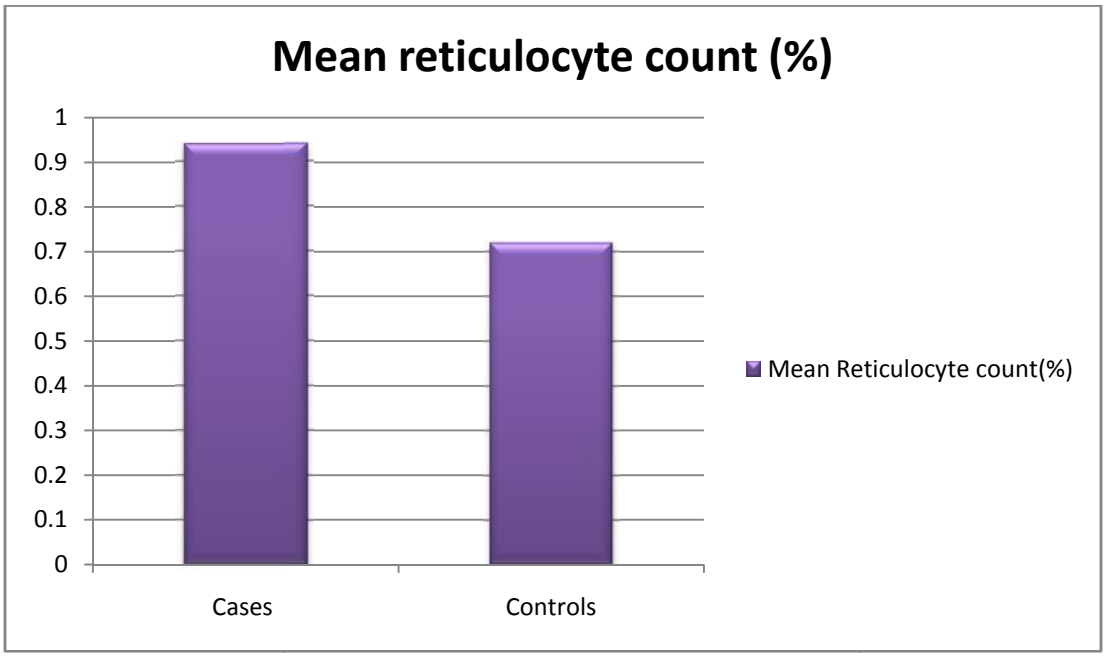


Table 18: Comparison of mean platelets count among cases & controls.

Group	Mean ± SD	p value*	Inference
Cases	4.29 ± 1.38	0.0007	Highly significant
Controls	3.19 ± 0.95		

In the above comparison the *p value is obtained using Student's Unpaired t Test.

Graph 10: Comparison of mean platelet count among cases and controls.

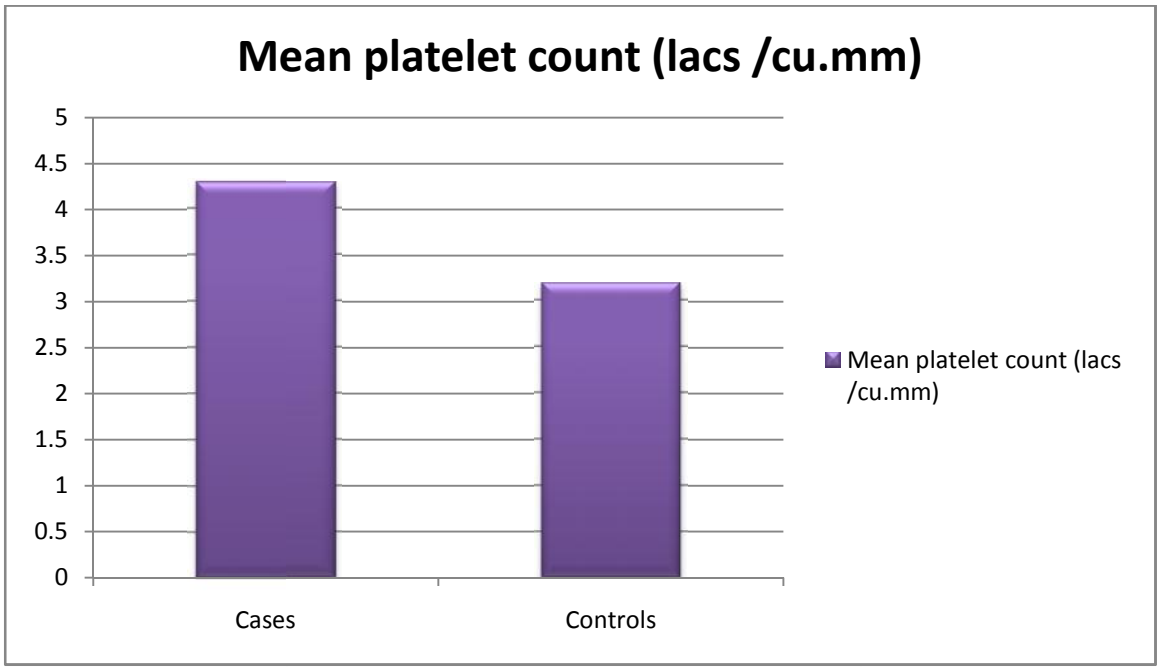


Table 19: Comparison of mean total leucocyte count among cases & controls.

Group	Mean ± SD	p value*	Inference
Cases	12.55 ± 4.90	0.0002	Highly significant
Controls	8.43 ± 2.83		

In the above comparison the *p value is obtained using Student's Unpaired t Test.

Graph 11 : Comparison of mean total leucocyte count among cases & controls.

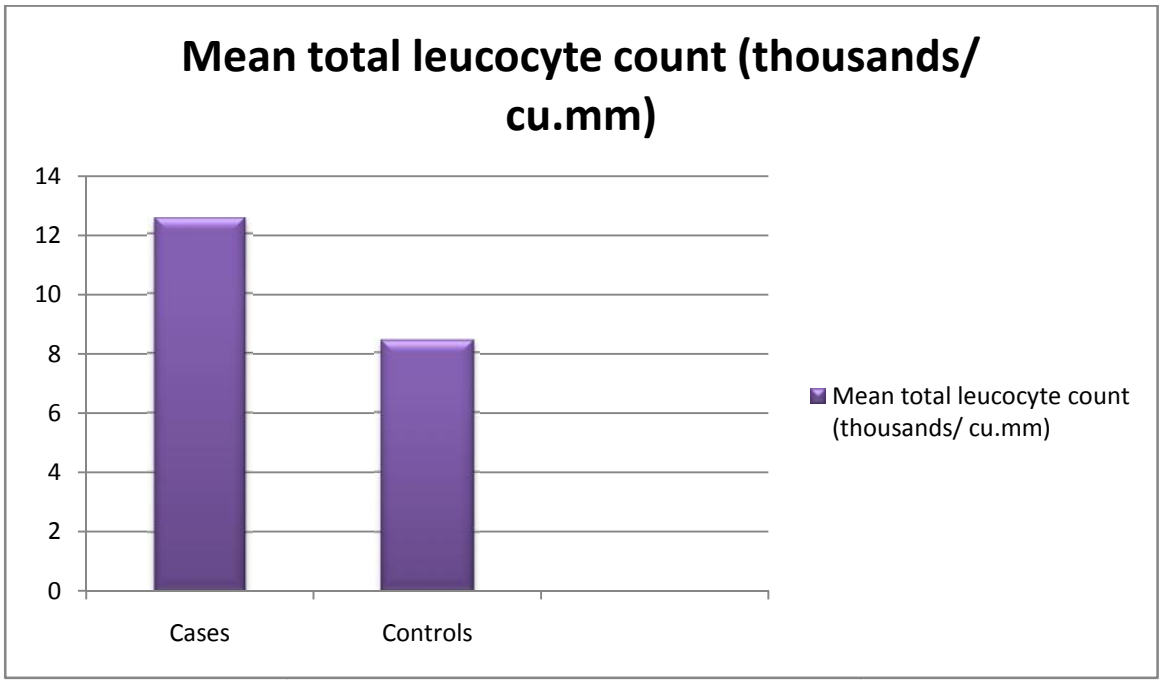


Table 20: Comparison of different RBC indices among cases and controls.

RBC Indices	Cases mean ±SD	Control mean ±SD	p value*
MCV (fL)	70.01±14.68	84.04±4.04	<0.0001
MCH (pg)	21.09±4.83	27.21±1.08	<0.0001
MCHC (gm/dl)	29.76±3.22	31.74±1.19	0.0025
RDW (%)	18.16±3.98	13.13±0.76	<0.0001

In the above comparison the *p value is obtained using Student's Unpaired t Test.

As depicted in the table 20, MCV and MCH among cases was significantly decreased as compared to controls. The mean RDW among cases was significantly increased in cases as compared to controls. However, the MCHC among cases and controls showed no statistically significant differences.

Graph 12 : Comparison of different RBC indices among cases and controls.

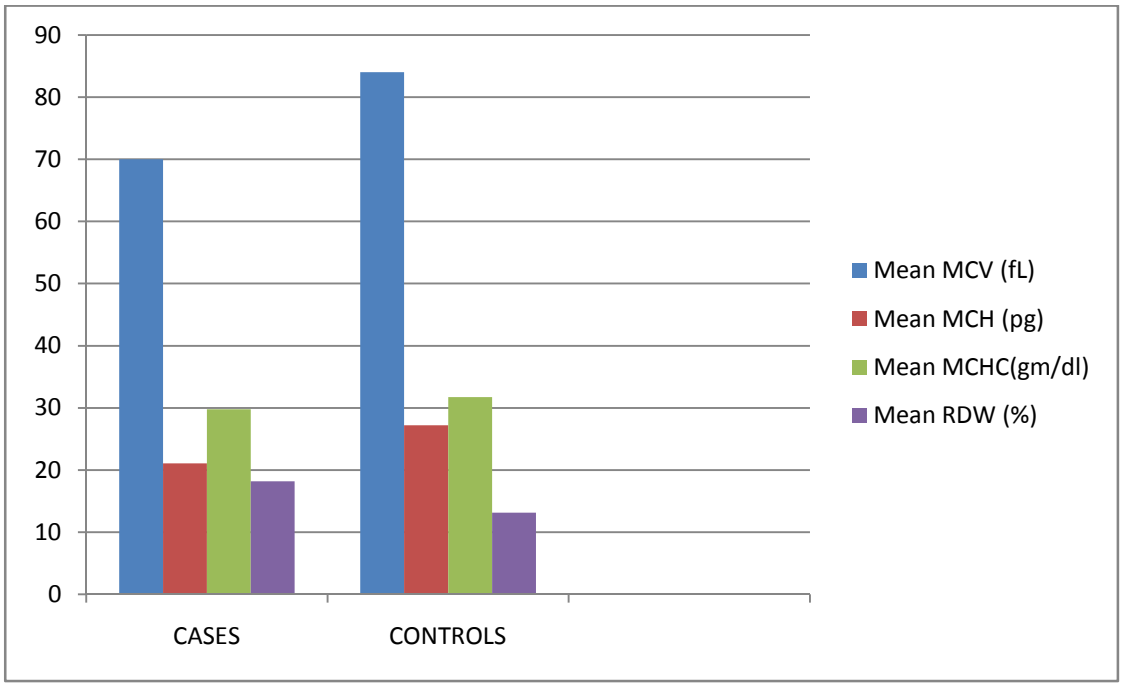


Table 21: Distribution of cases according to type of anaemia.

Type of anaemia	Number of cases	Percentage
Normal blood picture	3	10%
Microcytic hypochromic anaemia	19	63.33%
Dimorphic anaemia	7	23.33 %
Normocytic hypochromic anaemia	1	3.3%
Total	30	100%

Graph 13 : Distribution of cases according to type of anaemia.

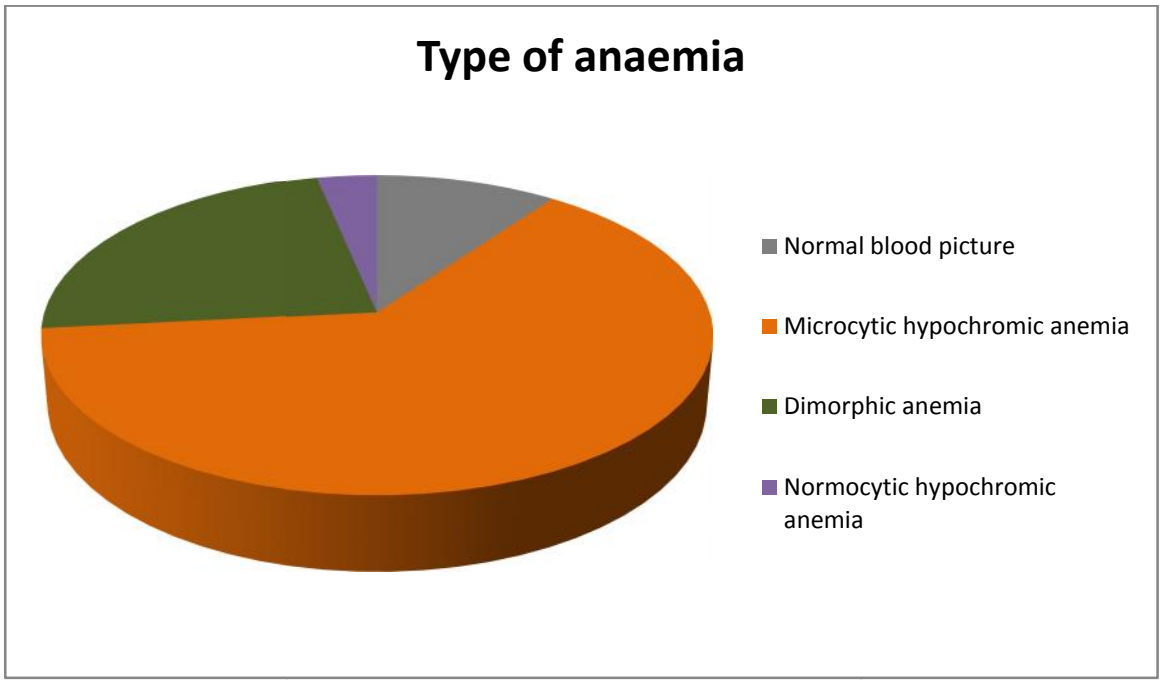


Table 22: Distribution of cases according to severity of anaemia.

Severity of anaemia	Number of cases	Percentage
No anaemia	3	10%
Mild anaemia	4	13.33%
Moderate anaemia	15	50.0%
Severe anaemia	8	26.67%
Total	30	100%

The severity of anaemia in children aged 6 months to 5 years of age was graded as follows :[64]

a. Mild anaemia : Haemoglobin = 10–10.9 gm/dl.
b. Moderate anaemia : Haemoglobin = 7.0–9.9 gm/dl.
c. Severe anaemia : Haemoglobin < 7.0 gm/dl.

Graph 14: Distribution of cases according to severity of anaemia.

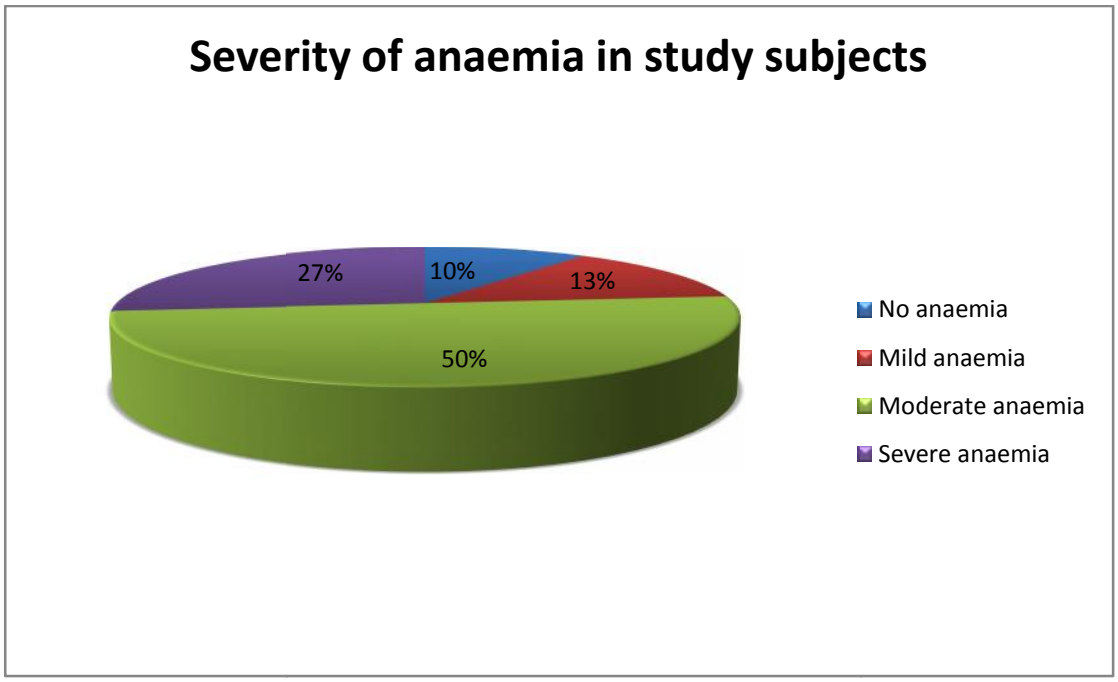


Table 23: Comparison of biochemical parameters among SAM cases and controls.

Biochemical parameters	SAM mean ±SD	Control Mean ±SD	p value*
S.sodium(mEq/L)	129.57±9.16	131.30±5.75	0.3836
S.potassium(mEq/L)	3.84±0.99	3.57±0.45	0.1736
S. albumin(gm/dl)	3.38±0.61	4.22±0.45	<0.0001
S. total proteins(gm/dl)	5.87±1.08	7.54±0.45	<0.0001
S. ferritin(ng/ml)	65.86±112.79	218.33±51.76	<0.0001

In the above comparison the *p value is obtained using Student's Unpaired t Test.

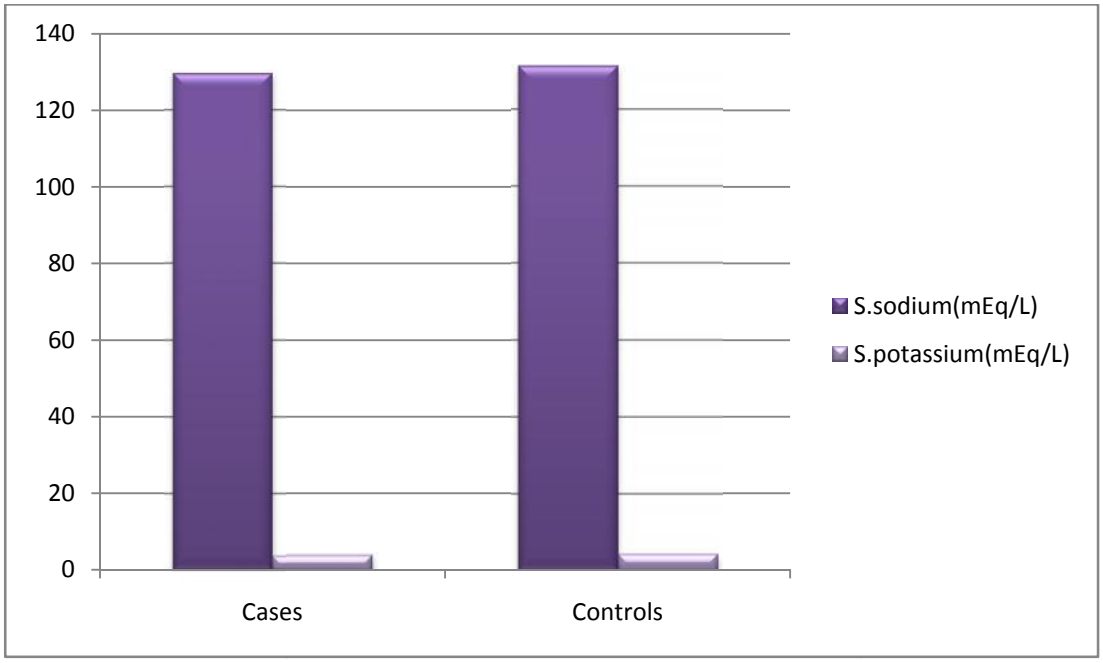
As depicted in the table 22, the mean serum sodium levels among study and control population was statistically not significant.

The mean levels of serum albumin, serum total proteins and serum ferritin among among cases were significantly reduced as compared to controls.

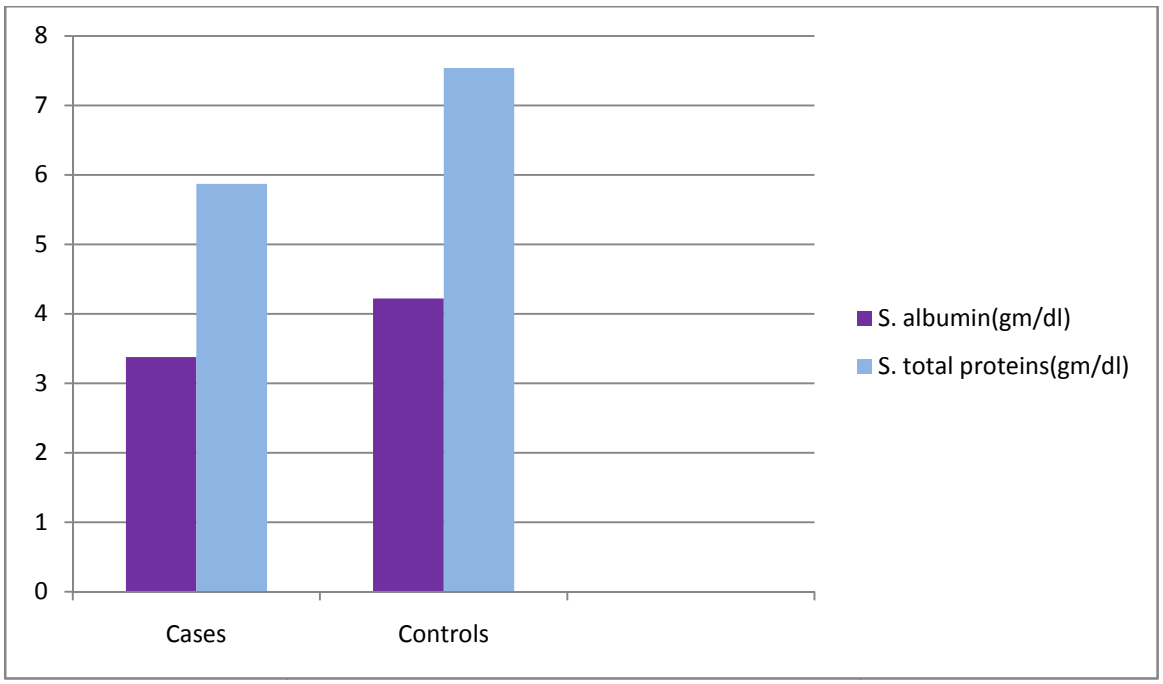
Of all the 30 cases, 17 cases (56.66 %) had reduced serum albumin and 13 cases (43.33 %) had normal serum albumin levels.

Of all the 30 cases, 16 cases (53.33%) had reduced serum ferritin levels (< 12 ng/ml) followed by 10 cases (33.33%) with normal ferritin levels and 4 cases (13.33%) had increased ferritin levels.

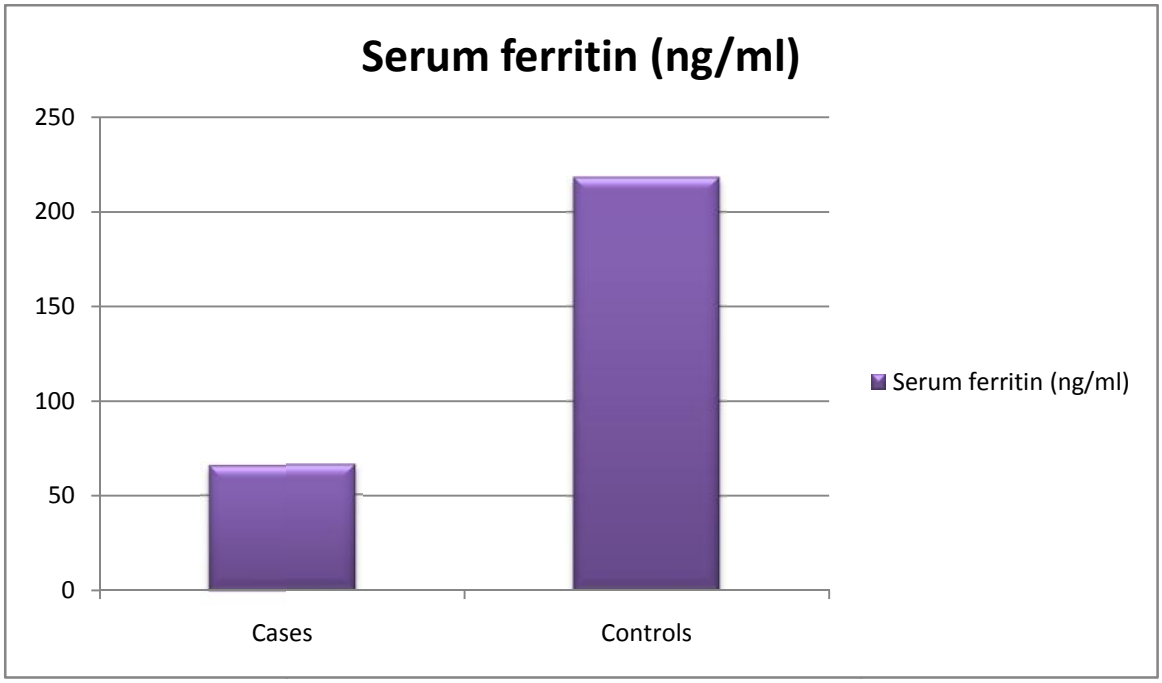
Graph 15: Comparison of mean of serum sodium and potassium among SAM cases and controls.



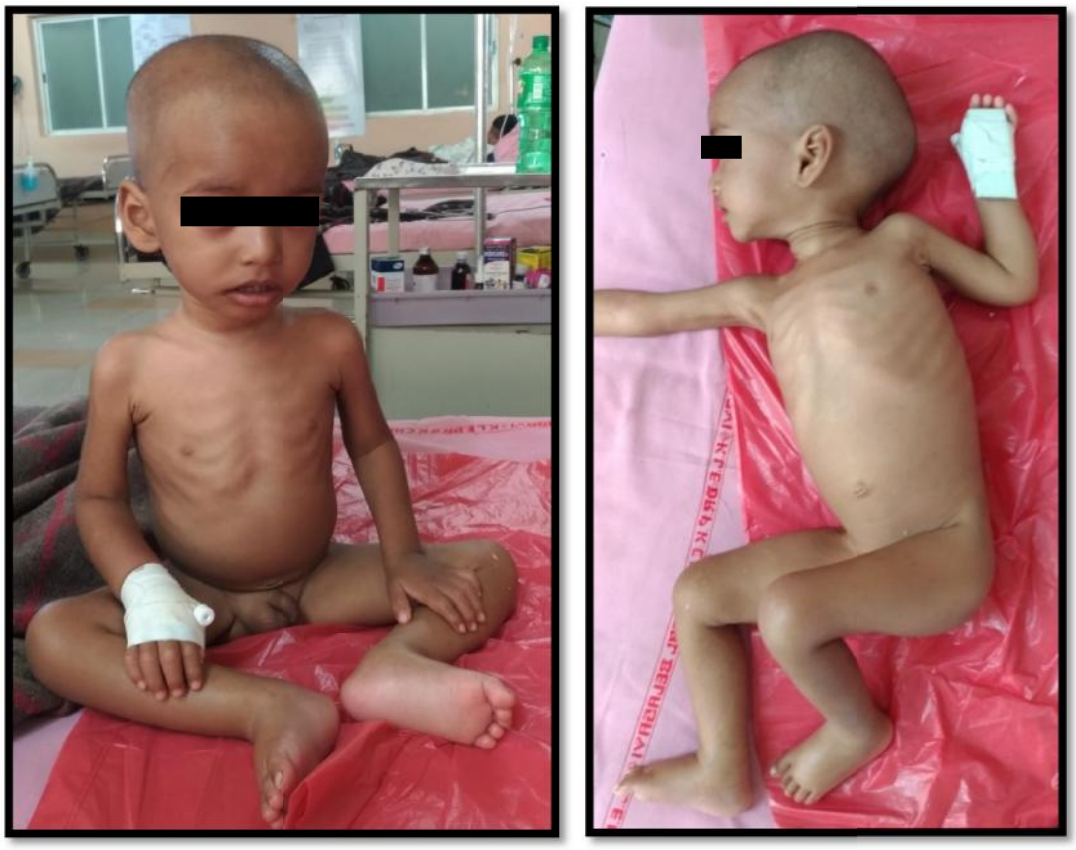
Graph16: Comparison of mean of serum albumin and total proteins among SAM cases and controls.



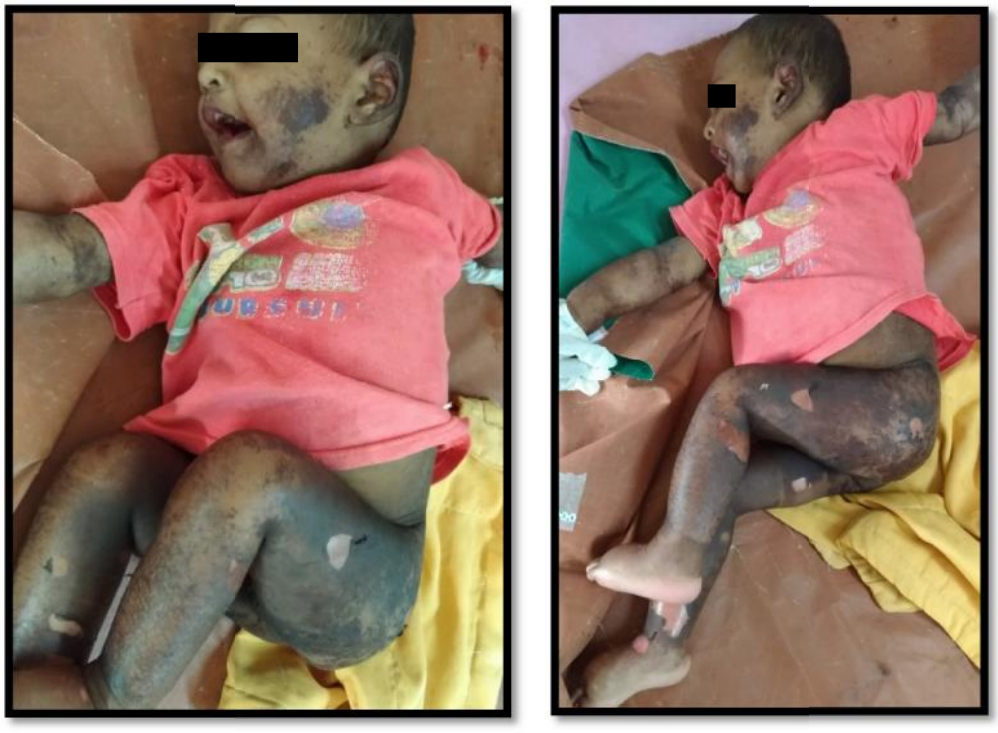
Graph 17: Comparison of mean of serum ferritin levels among SAM cases and controls.



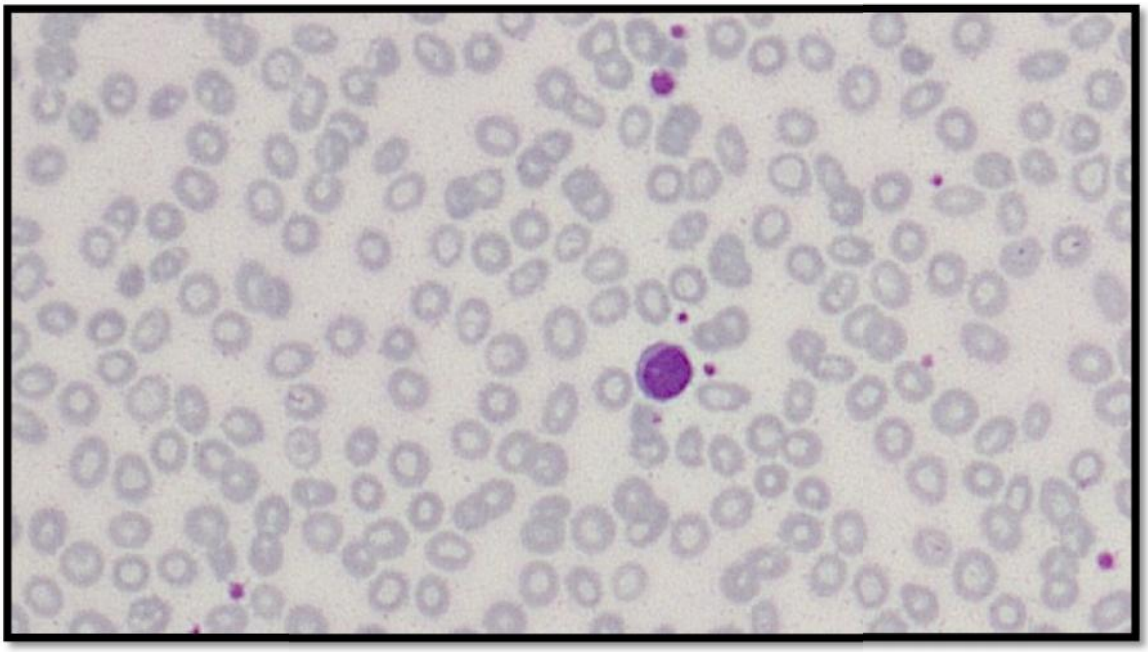
PHOTOGRAPHS



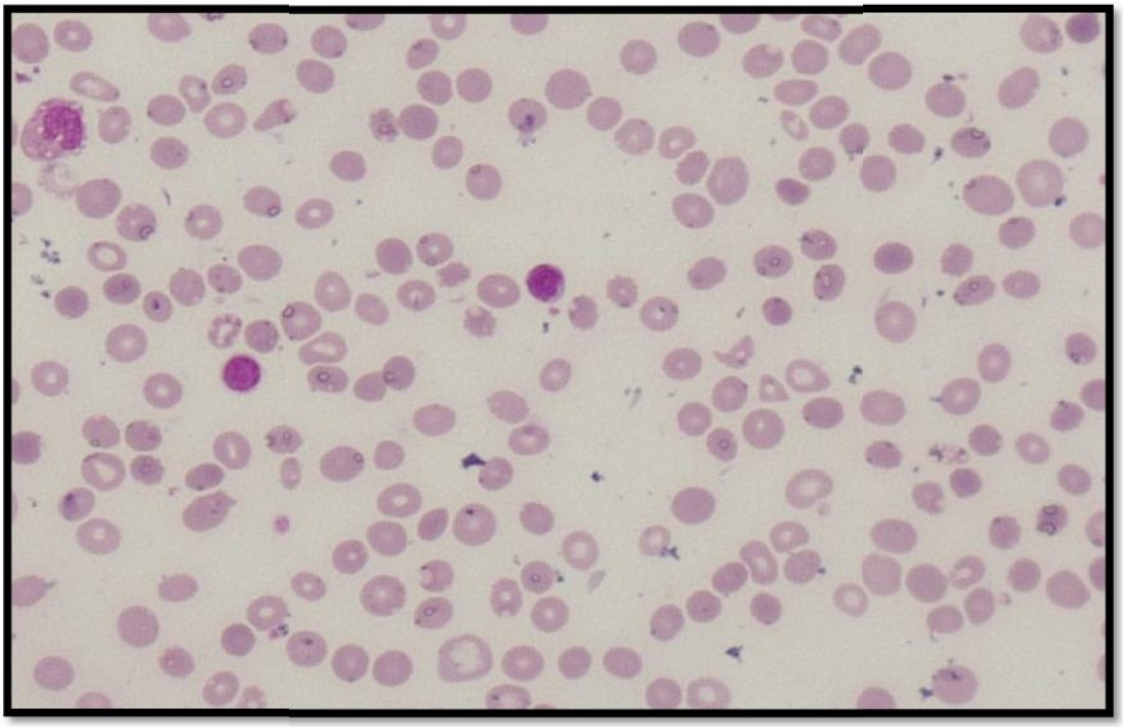
PHOTOGRAPH 5: A 2 year old boy diagnosed with marasmic – kwashiorkor with pedal oedema.



PHOTOGRAPH 6: A 3year old boy diagnosed with kwashiorkor with flaky paint dermatosis.



Photograph 7: Smear showing a case of microcytic anaemia with few pencil shaped cells (Wright stain, 100X)



Photograph 8: Smear showing a case of dimorphic anaemia with macrocytes, polychromatophils and few microcytes (Wright stain, 100X)

DISCUSSION

The burden of malnutrition is massive and is mostly seen in low and middle income countries.[69,70]

Malnutrition if not curbed at the right age affects growing children by its adverse consequences. India is one of the developing countries where under nutrition among children less than five years of age is still a major public health concern. This is revealed by the fact that in India, under-weight children are among the maximum in the world, and is nearly twice to that of Sub-Saharan Africa region.[71,72]

The present study was conducted among children aged 6 months to 5 years with Severe Acute Malnutrition (SAM) admitted into the Nutritional Rehabilitation Centre at KLES Dr. Prabhakar Kore Hospital and Medical research centre and Department of Pathology, JNMC, Belagavi between January 2018 to December 2018. A total of 60 children were enrolled in this study, with 30 cases and 30 controls.

Of the 30 study subjects, majority were in the age group of 13-24 months and 25-36 months. This was similar to other studies conducted by Choudhary M et al.[65] where the most common age group was 6-24 months and Singh K et al. [73] where the most common age group was 12-23 months. But this observation was in contrast with the findings of other studies where the high percentage of admitted children belonged to the age group of 6-12 months done by Yaikhomba T et al., (50.0%)[64] and Thakur N et al.(45.8%)[54] and 49-60 months of age group in a study done by Chaturvedi A et al. (45.3%)[74]. Majority of the literature suggests than Severe Acute Malnutrition was mainly observed in the age group of 1-3 years[75] and the findings of our study were in line with the same.

In the present study, majority of the study subjects were females, i.e, 16 (53.33 %) and the rest were males, i.e.,14 (46.67 %). The male:female ratio is 1:1.42. These results were consistent with the findings of Choudhary M et al. (Females- 84.2%)[65] and Kumar R et al. (Females -51.9%, Males- 49.1%).[76]

This is in contrast with the study done by Tiwari AK et al. where higher percentage of boys were seen (56.4% were males and 43.6% were females).[77]

In our study, majority of the SAM cases had anaemia (90%), out of which majority had moderate anaemia (50%) followed by severe anaemia (26.67%), mild anaemia (13.33%) and no anaemia(10%). Similar results were obtained in the study done by Dwivedi D et al.[78] where they found that forty two percent of SAM children had moderate anaemia and nineteen percent SAM children had severe anaemia.

In the present study, among the SAM children, marasmus being most common was observed in 90% of SAM children followed by marasmic-kwashiorkar (6.66%) and kwashiorkor (3.33%). Similar results were observed by Basheir HM et al.[55]. These findings concluded that marasmus poses a great threat in developing countries like India where poverty along with and inadequate food supplies are prevalent. [79]

In this study, the mean haemoglobin levels (gm/dl) in cases and controls are 8.33±2.16 and 12.55±1.30 respectively. The p value was <0.0001 which was statistically significant. These findings were similar with the studies conducted by Gohain et al.[59], Basheir HM [55]and Arya AK et al.[62]

In our study the most common type of anaemia was microcytic hypochromic anaemia (63.33%). Similar finding was observed in the study done by Thakur N et al.[54]

In the current study, SAM children had statistically significant lower mean values for red cell indices MCV, MCH, and RDW as compared to control group. These findings were similar with the studies conducted by Arya AK et al.[62] and Gohain EK et al.[59]

In our study, children with SAM had statistically significantly higher mean values for total leucocyte counts as compared to controls. These findings were consistent with the studies conducted by Arya AK et al.[62] and Gohain EK et al.[59]

In our study, the mean serum sodium, mean serum albumin and mean serum total proteins were significantly lowered in cases as compared to control group. Similar results were obtained by Nagle D et al.[80] where serum albumin and serum total proteins levels were significantly lowered in severely malnourished subjects.

However, in our study the mean serum potassium in study and control population was statistically insignificant. In the present study, hypokalemia was observed in 14 cases (46.67%), hyperkalemia in 5 cases (16.67%) and normal potassium levels in 11 cases (36.67%).

In another study conducted by **Dakshayani B et al.[68]**, hyponatremia was seen in 43.4% and hypokalemia in 7.1% of children. They found no statistically significant differences in the mean values of serum electrolytes and frequency of hyponatremia and hypokalemia between groups. They concluded that dyselectrolytemia occurs in SAM children with and without complications. Serum electrolyte levels may need to be measured in all SAM cases to detect asymptomatic hyponatremia and hypokalemia. This will help in triaging those with asymptomatic hyponatremia and hypokalemia to inpatient care.

In the current study, out of total 30 SAM cases, 19 cases (63.33%) had microcytic hypochromic anaemia with clinical features suggestive of iron deficiency anaemia. Amongst these, 16 cases had reduced serum ferritin levels (<12 ng/ml) and 3 cases had normal serum ferritin levels. Similar association between serum ferritin and iron deficiency anaemia was observed by **Kapur D et al.**[51]

Out of 7 cases (23.33%) which had dimorphic anaemia, 3 cases had reduced serum ferritin levels (<12 ng/ml), 3 cases had normal serum ferritin levels followed by 1 case with increased serum ferritin levels. This variation could be probably due to associated iron deficiency, vitamin C deficiency or infections as serum ferritin is also an acute phase reactant.[51]

In our study for identification of SAM cases, we found all the 30 cases (100%) have <-3SD weight for height and 10 (33.33%) cases were having MUAC <11.5 cm. These findings were consistent to those observed by **Dukhi N et al.**[60] where they identified weight for height as a more sensitive measure of child malnutrition and measured more than twice for children with SAM compared to MUAC.

CONCLUSION AND SUMMARY

We summarize the results of our prospective observational study conducted at Nutritional Rehabilitation Center, KLES Dr. Prabhakar Kore Hospital and Research centre and Department of Pathology, JNMC, Belagavi in which 60 subjects were enrolled during a period of one year from January 2018 to December 2018. All the information collected were analysed using various statistical tools and following conclusions were drawn :

1. The most common age group of the children that were enrolled in the study was between 13-36 months (66.67%).

2. Majority of the study subjects were females accounting for 16 cases (53.33 %) and the rest were males accounting for 14 cases (46.67 %). The male : female ratio was 1:1.42.

3. In our study, for identification of SAM cases, we found all the 30 cases (100%) have <-3SD weight for height as compared to 10 cases (33.33%) with MUAC <11.5 cm.

4. Most of the SAM children had marasmus (27 cases, 90%) followed by marasmic-kwashiorkar (2 cases, 6.66%) and kwashiorkor (1 case, 3.33%).

5. Majority of the SAM cases had anaemia (27 cases, 90%), out of which majority had moderate anaemia (15 cases, 50%) followed by severe anaemia (8 cases, 26.67%), mild anaemia (4 cases, 13.33%) and normal blood picture (3 cases, 10%).

6. The most common type of anaemia observed was microcytic hypochromic anaemia (19 cases, 63.33%) followed by dimorphic anaemia (7 cases, 23.33%) and normocytic hypochromic anaemia (1 case, 3.3%). Normal blood picture was seen in 3 cases which constituted a total of 10% .

7. Among the cases which had microcytic hypochromic anaemia (19 cases, 63.33%) majority of them had decreased serum ferritin levels (16 cases, 84.21%) and rest (3 cases, 10%) had normal ferritin levels. These findings conclude decreased serum ferritin levels in frank cases of SAM with iron deficiency anaemia.

8. Out of 7 cases (23.33%) which had dimorphic anaemia, out of which 3 cases (42.85%) had reduced serum ferritin levels (<12 ng/ml), 3 cases (42.85%) had normal serum ferritin levels followed by 1 case (14.28%) had increased serum ferritin levels .

9. Our study also concluded that electrolyte imbalance occurs in SAM children. Majority of the cases accounting for 14 cases (46.67%) had hypokalemia, followed by 11 cases (36.67%) which had normal potassium levels and 5 cases (16.67%) which had hyperkalemia. So, serum electrolyte levels may need to be measured in all SAM cases to detect asymptomatic hyponatremia and hypokalemia.

10. In our study, mean serum albumin and mean serum total proteins were significantly lowered in severely malnourished subjects as compared to controls.

BIBLIOGRAPHY

1. Thakur N, Chandra J, Pemde H, Singh V. Anaemia in severe acute malnutrition. Nutrition. 2014;30(4):440-2.

2. Pelletier DL, Frongillo EA, Schroeder DG, Habicht JP. The effects of malnutrition on child mortality in developing countries. Bull World Health Organ. 1995;73:443-8.

3. International Institute for Population Sciences (IIPS). National Family Health Survey (NFHS-4), 2015-2016. IIPS, Mumbai, India. 2017.

4. United Nations Children's Fund (UNICEF). Tracking progress on child and maternal nutrition. A survival and development priority. United Nations Children's Fund (UNICEF), 2009; New York.

5. United Nations Children's Fund (UNICEF). Tracking progress on child and maternal nutrition. A survival and development priority. New York: United Nations Children's Fund (UNICEF); 2009.

6. Hobbs B, Bush A, Shaikh B, Azmat S, Mazhar A, Solter S, et al. Acute malnutrition: an everyday emergency. A 10-point plan for tackling acute malnutrition in under-fives. JPMA J Pak Med Assoc. 2013;63(4 Suppl 3):S67–72.

7 .World Health Organization. Nutrition. [7 December 2018].URL: www.who.int/topics/ nutrition/ en/index.html.

8. UNICEF/WHO/ World Bank group, Levels and trends on child malnutrition –Joint Child Malnutrition Estimates, 2017, https://data.unicef.org/wp-

content/uploads/2017/06/JME-2017_brochure_June-25.pdf, Assessed 18 December 2018.Google Scholar.

9. Report on the IAEA technical meeting on 'Severe acute and moderate malnutrition in South East Asia' (jointly with IMTF), 27–29 May 2013, Bangkok, Thailand, p.1.

10. WHO; UNICEF; WFP; UN System Standing Committee on Nutrition, 2007; Community-based management of severe acute malnutrition - A joint statement;, Accessed 17 December 2018.Google Scholar.

11. World Health Organization. Management of severe malnutrition: a manual for physicians and other senior health workers. Geneva: World Health Organization; 1999.Google Scholar.

12. Lukens JN. Iron metabolism and iron deficiency. In: Miller DR, Bodner RL, Miller LP, eds. Blood diseases of infancy and childhood. Philadelphia:Mosby;1995:193-219.

13. Warrier RP. The anaemia of malnutrition. In: Suskind RM, Suskind LL, eds. The malnourished child. New York:Lippincott-Raven. 1990;19:61-72.

14. Nathan DG. Regulation of haematopoiesis. Pediatric research. 1990;27:423-7.

15 .Elizabeth KE. Nutrition and Child Development. New Delhi : Paras medical publishers. 4th edition, 2010.p.163.

16. WHO Global Database on Child Growth and Malnutrition. [cited 2018 Sep 17]. Available from: http://www.who.int/nutgrowthdb/about/introduction/en/index.html)

17. Elizabeth KE. Nutrition and Child Development. New Delhi : Paras medical publishers. 4th edition, 2010.p.163.

18. Nix Staci. Williams Basic Nutrition and Diet Therapy. 12th ed. Mosby, Elsevier. Missouri, USA. 2005.

19. KG Rajul. Introduction to Nutrition in Public health, Text book of Public Health and Community Medicine. 2nd ed. Pune: Department of community medicine, Armed Forces Medical College; 2009. p 713.

20. WHO | Children: reducing mortality [Internet]. WHO. [cited 2018 Aug 17]. Available from: http://www.who.int/mediacentre/factsheets/fs178/en/index.html.

21. Palmer PES, Reeder MM. Tropical sprue. Kwashiorkor in The imaging of tropical diseases, with epidemiological, pathological and clinical correlation; vol 2, 2nd ed, Williams and Wilkins company, London, 1981.

22. Levels and trends in child malnutrition. UNICEF/ WHO / World Bank Group Joint Child Malnutrition Estimates Key findings of the 2018 edition. available from Lhttp://www.who.int/nutgrowthdb/2018-jme-brochure.pdf ?ua=1

23. Black R et al. Maternal and child undernutrition and overweight in low-income and middle income countries. Lancet, 2013;382:427–51.

24. United Nations Interagency Group for Child Mortality Estimation. Levels and trends in child mortality. Report 2012. New York, United Nations Children's Fund, 2012.

25. A Monica et al., Malnutrition Analysis of Anganwadis.2016, DU Journal Of Undergraduate Research and Innovation Volume 2, Issue 2 pp 72-79. Available from http://journals.du.ac.in/ugresearch/pdf-vol5/9.pdf.

26. Children aged <5 years underweight Data by country. Global Health Observatory data repository. World Health Organisation. Available from- http://apps.who.int/gho/data/ node.main. CHILDUNDERWEIGHT?lang=en

27. Chatterjee P. Child malnutrition rises in India despite economic boom. Lancet 2007;369:1417-8.

28. National Family Health Survey 4, 2015-16, India Fact Sheet, International Institute for Population Sciences, Ministry of Health and Family Welfare, Government of India.

29. National Family Health Survey (NFHS-3), 2005 - 2006: India: Volume I [Internet]. International Institute for Population Sciences (IIPS) and Macro International; 2007. Available from: http://www.rchiips.org/NFHS/chapters.html

30. International Institute for Population Sciences and Macro International National Family Health Survey (NFHS-2). Mumbai; IIPS: 1998 99; 2000. Available from: http:// www.dhsprogram.com/pubs/pdf/frind2/frind2.pdf. [Last accessed on 2018 Sep 15].

31. National Family Health Survey 4, 2015-16, State Fact Sheet, Karnataka. International Institute for Population Sciences, Ministry of Health and Family Welfare, Government of India.

32. National Family Health Survey 4, 2015-16, District Fact Sheet, Belgaum, Karnataka. International Institute for Population Sciences, Ministry of Health and Family Welfare, Government of India[cited2019, Aug27]. Available from: http://rchiips.org/nfhs/factsheet_ NFHS-4 .shtml.

33. Lakshamanswamy A. Clinical Paediatrics History Taking And Case Discussion. New Delhi: Wolters Kluwer (India) Pvt. Ltd. 3rd Edition,2012.p.199.

34. Harohalli R Shashidhar. Malnutrition medscap, Updated: Feb 11, 2013. Available from http://emedicine.medscape.com/article/985140-overview#aw2aab6b2b3aa.

35. Turning the tide of malnutrition. Responding to the challenge of the 21st century. Nutrition for Health and Development, Sustainable Development and Healthy Environments, World Health Organization.Available from:http://apps.who.int/iris/bitstream/handle/ 10665/ 66505/ WHO_ NHD_00.7.pdf;jsessionid=70BEA82A538C9A02FE0A86C0AE98FCFB?sequence=1.

36. Waterlow JC. Classification and definition of protein-calorie malnutrition. British Medical Journal. 1972 Sep 2;3(5826):566–9.

37. Global Database on Child Growth and Malnutrition. [cited 2018 December 17]. Available from: http://www.who.int/nutgrowthdb/about/introduction/en/index2.html

38. Elizabeth KE. Nutrition and Child Development. New Delhi: Paras medical publishers. 4th edition, 2010.p.175-8.

39. Pocket Book of Hospital Care for Children: Guidelines for the Management of Common Childhood Illnesses. 2nd edition. Geneva: World Health Organization; 2013. 7, Severe acute malnutrition. Available from: https://www.ncbi.nlm.nih.gov/books/NBK154454/.

40. Elizabeth KE. Nutrition and Child Development. New Delhi : Paras medical publishers. 4th edition, 2010.p.181-2.

41. Williams CD. Kwashiorkor: a nutritional disease of children associated with a maize diet. Lancet. 1935;229:1151-2.

42. Lakshamanswamy A. Clinical Paediatrics History Taking And Case Discussion. New Delhi: Wolters Kluwer (India) Pvt. Ltd. 3rd Edition,2012.p.212-3.

43. Lakshamanswamy A. Clinical Paediatrics History Taking And Case Discussion. New Delhi: Wolters Kluwer (India) Pvt. Ltd. 3rd Edition, 2012.p.206-11.

44. Elizabeth KE. Nutrition and Child Development. New Delhi : Paras medical publishers. 4th edition, 2010.p.169-71.

45. Lakshamanswamy A. Clinical Paediatrics History Taking And Case Discussion. New Delhi: Wolters Kluwer (India) Pvt. Ltd. 3rd Edition,2012.p.206.

46. Lakshamanswamy A. Clinical Paediatrics History Taking And Case Discussion. New Delhi: Wolters Kluwer (India) Pvt. Ltd. 3rd Edition,2012.p.211.

47. Lakshamanswamy A. Clinical Paediatrics History Taking And Case Discussion. New Delhi: Wolters Kluwer (India) Pvt. Ltd. 3rd Edition,2012.p.208. 48. Thompson WG, Meola T, Lipkin MJR, Freedman ML. Red cell distribution width, mean corpuscular volume, and transferrin saturation in the diagnosis of iron deficiency. Archieves of Internal Medicine. 1988; 148: 2128–30.

49. WHO, UNICEF, UNU. Iron deficiency anaemia: assessment, prevention, and control. A guide for programme managers. Geneva, World Health Organization, 2001. WHO/ NHD/01.3.

50. Witte DL. Can serum ferritin be effectively interpreted in the presence of the acute phase response. Clinical Chemistry, 1991, 37:484–5.

51. Kapur D, Agarwal KN, Sharma S, Kela K and Kaur I. Iron status of children aged 9-36 months in an urban slum Integrated Child Development Services project in Delhi. Indian Pediatrics 2002; 39:136-44 .

52. Mishra SK, Bastola SP, Jha B.Biochemical nutritional indicators in children with protein energy malnutrition attending Kanti Children Hospital, Kathmandu, Nepal. Kathmandu University Medical Journal,2009 , Vol. 7, No. 2, Issue 26, 129-34.

53. Aguayo VM, Aneja S, Badgaiyan N and Singh K. Mid upper-arm circumference is an effective tool to identify infants and young children with severe acute malnutrition in India. Public Health Nutrition.2015; p 1-5. doi:10.1017/S1368980015000543.

54. Thakur N, Chandra J, Pemde H, Singh V. Anaemia in severe acute malnutrition. Nutrition. 2014;30(4):440-2.

55. Basheir HM, Hamza KL. Hematological Parameters of Malnourished Sudanese Children Under 5 Years – Khartoum State – 2011. Clinical Medicine Journal Vol. 1, No. 4, 2015, p. 152-6.

56. Tadesse AW, Tadesse E, Berhane Y, Ekstrom EC. Comparison of Mid-Upper Arm Circumference and Weight-for-Height to Diagnose Severe Acute Malnutrition: A Study in Southern Ethiopia. Nutrients. 2017; 9(3):267.

57. Badi MA , Ba-Saddik IA. Severe Acute Malnutrition among Hospitalized Children, Aden, Yemen. Open Journal of Epidemiology, 2016, 6, 121-7. Published Online May 2016 in SciRes. http://www.scirp.org/journal/ojepi http://dx.doi.org/ n10.4236/ojepi.2016.62012.

58. Bhadoria AS, Kapil U, Bansal R, Pandey RM, Pant B, Mohan A. Prevalence of severe acute malnutrition and associated sociodemographic factors among children aged 6 months–5 years in rural population of Northern India: A population-based survey. J Family Med Prim Care 2017;6:380-5.

59. Gohain EK , Pathak K, Choudhury B. A Case Control Study of Hematological Changes in Children with Protein Energy Malnutrition Attending Gauhati Medical College and Hospital. IOSR Journal of Dental and Medical Sciences. 2016;15(10):25-9.

60. Dukhi N, Sartorius B & Taylor M. Mid-upper arm circumference (MUAC) performance versus weight for height in South African children (0–

59 months) with acute malnutrition. South African Journal of Clinical Nutrition;2017;30(2), 49-54, DOI: 10.1080/16070658.2016.1255483.

61. Agarwal A, Tambekar MY & Dhar R. Hematological Profile and Anthropometric Assessment of a Pediatric Mumbai Slum Population. Internatioal Journal of Current Medical And Applied Sciences, 2016, August, 11(3),184-7.

62. Arya AK, Kumar P, Midha T , Singh M.Hematological profile of children with severe acute malnutrition: a tertiary care centre experience.International Journal of Contemporary Pediatrics.2017 .Sep;4(5):P-1577-80.

63. Chizoba ON, Aloysius M, Otiora C, Uche C. Prevalence of severe acute malnutrition in children aged 6-60 months admitted at Mother of Christ specialist hospital Ogui, Enugu, Nigeria. European Journal of Preventive Medicine. Vol. 2, No. 4, 2014, pp. 45-51. doi: 10.11648/j.ejpm.20140204.12.

64. Yaikhomba T, Poswal L, Goyal S. Assessment of Iron, Folate and Vitamin B12 Status in Severe Acute Malnutrition. The Indian Journal of Pediatrics. 2014;82(6):511-4.

65. Choudhary M, Sharma D, Nagar RP, Gupta BD, Nagar Tand Pandita A. (2015) Clinical Profile of Severe Acute Malnutrition in Western Rajasthan: A Prospective Observational Study from India. J Pediatr Neonatal Care 2(1): 00057. DOI: 10.15406/jpnc.2015.02.00057

66. Pravana NK, Piryani S, Chaurasiya SP, Kawan R, Thapa RK ,Shrestha S. Determinants of severe acute malnutrition among children under 5 years of

age in Nepal: a communitybased case–control study. BMJ Open 2017;7:e017084. doi:10.1136/ bmjopen-2017-017084.

67. Bobby J, Rebello A, Kullu P & Raj VD. (2002). Prevalence of Malnutrition in Rural Karnataka, South India: A Comparison of Anthropometric Indicators. Journal of health, population, and nutrition, Sept 2002(3): p.239-44.

68. Dakshayani B, Ravi M , Premalatha R. A study of serum electrolytes in severe acute malnourished children with and without complications. Indian Journal of Child Health. 2018; 5(2):120-3.

69. Malnutrition. Key facts. World Health Organisation. Available from http://www.who.int/news-room/fact-sheets/detail/malnutrition. Accessed on 4th June, 2018.

70. Levels and trends in child malnutrition. UNICEF / WHO / World Bank Group Joint Child Malnutrition Estimates Key findings of the 2018 edition. available from Lhttp://www.who.int/nutgrowthdb/2018-jme-brochure.pdf ? ua=1.

71. Monica A et al., Malnutrition Analysis of Anganwadis.2016, DU Journal of Undergraduate Research and Innovation Volume 2, Issue 2 pp 72-79. Available from http://journals.du.ac.in/ugresearch/pdf-vol5/9.pdf.

72. Children aged <5 years underweight.Data by country. Global Health Observatory data repository. World Health Organisation. Available from http://apps.who.int/gho/ data/node.main.CHILDUNDERWEIGHT?lang=en

73. Singh K, Badgaiyan N, Ranjan A. et al. Management of Children with Severe Acute Malnutrition: Experience of Nutrition Rehabilitation Centers in Uttar Pradesh, India [Internet]. Indianpediatrics.net. 2019 [cited 16 July 2019]. Available from: https://www.indianpediatrics.net/jan2014/jan-21-25.htm

74. Chaturvedi A et al., Progress of children with severe acute malnutrition in the malnutrition treatment centre rehabilitation program: evidence from a prospective study in Jharkhand, India. Nutrition Journal (2018) 17:69, p 1-9.

75. Nutrition. UNICEF. Available from http://unicef.in/Story/1124/Nutrition.

76. Kumar R, Singh J, Joshi K, Singh HP, Bijesh S. Co-morbidities in Hospitalized Children with Severe Acute Malnutrition Indian Pediatrics (2014) 51: p 125-7.

77. Tiwari AK, Jaiswal AK, Saurabh K, Priyanka, Supriya..A study of clinical profile and outcome of SAM children admitted in nutritional rehabilitation centre, Patna Medical College and Hospital, Patna, Bihar, India. **International Journal of Contemporary Pediatrics,** [S.l.], v. 5, n. 4, p. 1497-1504, june 2018. ISSN 2349-3291. Available at: <https://www.ijpediatrics.com/index.php/ijcp/article/view/1640>. Date accessed: 17 july 2019. doi:http://dx.doi.org/10.18203/2349-3291.ijcp20182553.

78. Dwivedi, D., Singh, V., Singh, J., & Sharma, S. (2018). Study of Anaemia in Children with Severe Acute Malnutrition. Journal of Nepal Paediatric Society, 37(3), 250-253. https://doi.org/10.3126/jnps.v37i3.18480.

79. Health grades Editorial Staff. Marasmus. Food, Nutrition and Diet. Medical Reviewers: William C., Lloyd III, MD, FACS; 2013.

80. Nagle D, Dubey A, Agrawal BK. Study of serum electrolytes and proteins level in severe malnourshed within 24 hours of admission (age group 6 month to 5 year). International Journal of Biological & Medical Research.,2014; 5(4): 4539-45.

ANNEXURE I-CONSENT FORM

INFORMED CONSENT FOR CHILD'S PARENTS/ GUARDIAN

TITLE OF THE STUDY: "A COMPARATIVE STUDY TO KNOW THE HAEMATOLOGICAL PROFILE AND BIOCHEMICAL PROFILE AMONG CHILDREN WITH SEVERE ACUTE MALNUTRITION AND HEALTHY CHILDREN".

Purpose of the study: You are being asked to enroll your child/ ward in this study as your child/ ward is eligible for participation in this study. The purpose of this study is to determine haematological profile, biochemical alterations and anthropometric assessment in severe acute malnourished children and its comparison with healthy or well nourished children. This study will help in knowing the haematological profile, biochemical alterations and anthropometric assessment of severe acute malnutrition which helps in the treating the patients and in decreasing the morbidity and mortality in children with severe acute malnutrition .

Procedure: During this study, you will be asked questions regarding history and background about your child/ ward and you are supposed to answer to the best of your knowledge . The principal investigator of the study is Dr. _____ under the guidance of Dr. _____ (guide) and Dr. _____ (co- guide).

If you agree to enroll your child/ ward in this study, you will be interviewed regarding your ward details including present, past and family history etc .

Risks and benefits: There are no risks involved in taking part in this study and benefit is we will be able to know the maternal and fetal morbidity and mortality so that we can reduce the same.

Alternatives: Taking part in this study is voluntary. You may choose to withdraw your child/ ward anytime during the study, or if you decide to take part now, you can later change your mind and withdraw from the study. The study doctor or sponsor may terminate your participation in this study anytime.

Privacy and confidentiality: All information collected about your child/ ward during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify your child/ ward in this research record. Information from this study will be published but your identity will be confidential in any publication. No information about you or information provided by you during research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

Financial incentives for participation: You and your/ ward will not be paid/offered any gift /incentives for participating in this study.

Authorisation to publish results: The results of this study would be forwarded to the KLE University, Belagavi as a part of requirement towards the completion of MD degree, review and publishing.

Name of the child/ ward: _____

Name of parent/ guardian : _____

Signature of the parents/ guardian : _____

Questions: In case you have any questions related to the study in future you can contact:

Name of witness: _____

Signature of the witness : _____

Name of the researcher: _____

Signature of the researcher: _____

Date: Place :

ANNEXURE II – ETHICAL CLEARANCE

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/02 **Date: 22/11/2017**

To,

PG student in Pathology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A COMPARATIVE STUDY TO KNOW THE HAEMATOLOGICAL PROFILE AND BIOCHEMICAL PROFILE AMONG CHILDREN WITH SEVERE ACUTE MALNUTRITION AND HEALTHY CHILDREN",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE III – PROFORMA

"A ONE YEAR CASE CONTROL STUDY ON HAEMATOLOGICAL PROFILE, BIOCHEMICAL PROFILE AND ANTHROPOMETRIC ASSESSMENT IN SEVERE ACUTE MALNUTRITION AT JNMEDICAL COLLEGE, KLE PRABHAKAR KORE CHARITABLE HOSPITAL,BELAGAVI- 590010".

NAME:

AGE:

SEX:

I.P./O.P NUMBER:

OCCUPATION:

COMPLETE ADDRESS:

PHONE NO:

PRESENT HISTORY:

PAST HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

IMMUNISATION HISTORY :

CLINICAL DIAGNOSIS:

TREATMENT GIVEN/ADVISED:

GENERAL PHYSICAL EXAMINATION :

Pulse

RR

BP

Temperature

Pallor

Cyanosis

Icterus

Clubbing

Lymphadenopathy

Oedema

ANTHROPOMETRY :

Age

Sex

Height/ Length

Weight

Head circumference

Chest circumference

Mid upper arm circumference

Abdominal circumference

Weight for height

HAEMATOLOGICAL INVESTIGATIONS:

- Blood group
- Hb
- Red cell count
- PCV
- MCH
- MCH
- MCHC
- RDW
- Reticulocyte count
- Total leucocyte count
- Differential leucocyte count

BIOCHEMICAL INVESTIGATIONS:

- Serum Sodium
- Serum Potassium
- Serum ferritin
- Serum albumin
- Total proteins

ANNEXURE IV – WRIGHTS STAINING PROTOCOL

Procedure:

1. Allow the slides to dry and place on the staining rack.
2. Put sufficient Wrights stain on the smear and wait for 3 minutes.
3. Add equal quantity of buffer (distilled water or phosphate buffer pH-6.5)
4. Blow gently
5. Wait for 5 minutes and then discard the stain and the buffer.
6. The back of the slide is cleaned with gauze
7. Excess of water is drained off
8. The stained smear is dried.
9. **RESULTS:**

NUCLEUS	
Chromatin	Purple
Nucleoli	Light blue
CYTOPLASM	
Erythroblast	Dark blue
Erythrocyte	Dark pink
Reticulocyte	Grey- blue
Lymphocyte	Blue
Monocyte	Grey- blue
Promyelocyte	Blue

Myelocyte	Pink
Metamyelocyte	Pink
Neutrophil	Pink
Basophil	Blue
GRANULES	
Promyelocyte	Red / purple
Basophil	Purple black
Neutrophil	Purple
Eosinophil	Red orange
Toxic granules	Dark blue
Platelet	Purple
OTHER INCLUSIONS	
Auer rods	Purple
Cabot ring	Purple
Howell- Jolly body	Purple
Dohle body	Light blue

ANNEXURE – V - KEY TO MASTER CHART

AC - Abdominal circumference

CC - Chest circumference

DA - Dimorphic anaemia

DLC - Differential leucocyte count

Hb - Haemoglobin

HC - Head circumference

MCV - Mean corpuscular volume

MCH - Mean corpuscular haemoglobin

MCHC - Mean corpuscular haemoglobin concentration

MHA - Microcytic hypochromic anaemia

MUAC - Mid upper arm circumference

NBP - Normal blood picture

PCV - Packed cell volume

PS - Peripheral smear

RDW - Red cell distribution width

SD - Standard deviation

TIBC - Total iron binding capacity

TLC - Total leucocyte count

S.NO	Hb (gm%)	PCV (%)	RBC (millions/cumm)	RETIC (%)	PLATELETS(lac/cumm)	TLC(/cumm)	DLC	PS	MCV (fL)	MCH (pg)	MCHC (gm/dl)	RDW (%)	Serum Ferritin (ng/m)	Serum Albumin (gm/dl)	Total proteins (gm/dl)	Weight (Kg)	Height/ Length (cm)	HC (cm)	CC (cm)	MUAC (cm)	AC (cm)	Oedema	Weight for Height (SD)	Serum sodium (mEq/L)	Serum Potassium (mEq/	Serum Iron	Vitamin B12 (pg/ml)	TIBC	Transferrin saturation
Case 1	3.3	11.9	1.78	1	8.8	7000	N46L47E2M5	DA	110	28	36	22.5	10.07	3	5.2	6.3	70.5	41	46	11.1	50	Absent	-3.15	138	3.97		50		
Case 2	9.9	32.7	5.11	0.6	4.51	10020	N60L35E2M3	MHA	64.1	19.4	30.3	14.9	5.13	3.6	5.6	7.6	79	45	44	13	46	Absent	-3.24	120	3				
Case 3	6.6	26.3	4.7	0.7	4.96	10120	N35L60E2M3	MHA	56	14.1	25.1	16.9	3.88	3.3	6	7.2	76	43.7	41	11.5	42	Absent	-3.88	129	3.3				
Case 4	6.2	23.8	4.43	0.7	4.23	10570	N30L63E2M3	MHA	53.8	14	25.9	18	5.84	3.5	7.2	8	81	46	46	11.7	45.5	Absent	-3.14	125	2.8				
Case 5	6.6	21.5	3.57	0.7	4.88	15170	N35L60E1M4	MHA	60.2	18.5	30.8	18.9	5.35	4.6	6	4	64	42	34.7	9.5	37	Absent	-5.77	126	3.1				
Case 6	8.9	27.5	3.55	0.6	5.36	7160	N40L55E1M4	DA WITH THROMBOCYTOSIS	77.4	21	32.4	17.5	7.25	3.8	4.7	7.8	76	42	46	13	45	Absent	-3.09	125	3				
Case 7	9.8	37.7	5.42	0.7	4.42	10820	N38L55E2M5	MHA	63	20	31.1	17.8	6.7	3.2	7.4	7	74	43	44	12	45	Absent	-3.05	129	3.3				
Case 8	7.5	30.1	4.84	0.7	3.77	15030	N62L35E2M1	DA WITH NEUTROPHILIA	62.3	15.5	24.8	21.9	57.5	2.8	4.8	5.9	72	45	42.5	10	39.8	Present	-5.13	137	4.44		538		
Case 9	6.9	22.5	2.56	0.9	2.56	13600	N16L77E1M6	DA	88.1	27	30.6	16.4	10	3.1	4.5	6	67	42	44	11	43	Present	-3.22	136	4.2				
Case 10	10.3	33.4	5.05	0.6	5.35	14290	N70L26E1M3	DA WITH NEUTROPHILIA AND THROMBOCYTOSIS	66.1	20.4	30.9	23.9	65.5	4.5	7.6	9.72	90	47	46	13	49	Absent	-3.1	138	3.8				
Case 11	9	31.7	5.1	0.6	4.03	10430	N40L55E2M3	MHA	62.1	17.7	28.6	16	7.68	3.5	7	7.9	80	43	46	11.6	47	Absent	-3.04	128	3				
Case12	7.7	28.9	4.78	0.7	3.3	6220	N58L37E2M3	MHA	60.5	16.1	24.6	16.3	10	4.1	5.9	8.7	83	44	47	12	47.5	Absent	-3.05	150	5.8				
Case 13	8	24	4.35	0.6	3.76	11240	N38L60E1M1	MHA	67.4	22.1	23.1	17.6	34	3	5.8	8	81	45	47	12.2	56.9	Absent	-3.72	114	4.2				
Case 14	10	30.5	5.23	0.7	4.13	10650	N39L45E12M4	MHA WITH EOSINOPHILIA	62	20	31	18	6.9	3.4	5.9	7.8	80	44	45	12.3	45.5	Absent	-3.19	122	3.1				
Case 15	6.3	19	2.66	0.8	4.32	15950	N26L70E2M3	MHA	66.5	23.8	35.8	18	508	2.6	4	6.2	70	40	41	10.2	43	Absent	-3.19	136	6.45				
Case 16	7.6	24.4	3.76	0.8	1.6	12000	N46L50E2M3	DA	105.7	34.8	33	19	105.3	2.8	5	6	70	42	44	12.9	48	Present	-4.43	134	4.6		125		
Case 17	8	25.7	3.12	0.6	1.73	8900	N32L59E3M6	MHA	68.5	20.8	30.8	18	50.8	2.6	3.8	8	80	44	48	12.6	47	Absent	-3.19	150	5.8				
Case 18	10.2	32.4	4.21	0.6	3.5	12920	N46L50E1M3	NHA	76.9	24.3	31.6	15.4	126	4.4	6	9	70	45	46	11.2	48	Absent	-3.38	130	4.2				
Case 19	8.6	31	4.81	0.8	6.28	20400	N23L67E4M6	MHA WITH THROMBOCYTOSIS	60	24.9	32	17	46.73	3.7	5.6	5.8	68	48	41	11.1	44	Absent	-3.98	136	4.97				
Case 20	8.6	28.2	4.24	0.7	4.06	11340	N40L55E2M3	MHA	66.4	20.3	30.6	15.7	44	3.2	6.1	8.1	86	46	44	11.9	47	Absent	-4.79	139	3.5				
Case 21	9.2	30.7	5.22	0.5	3.69	10150	N24L68E3M5	MHA	59.7	18.1	30.3	17.9	3.2	2.5	6	7	80	40	48	11	47	Absent	-4.37	122	2.9	15		450	3
Case 22	6.4	20.1	4.9	0.6	3.8	10000	N41L54E1M4	MHA	53.7	12.7	23.7	16.8Q	19.86	2.8	6.8	7.7	76	44	48	11.9	48.2	Absent	-3.08	142	3.8	12		351	3
Case 23	9.2	31.7	4.73	0.9	5.74	12510	N45L55E2M4	MHA WITH THROMBOCYTOSIS	64	20	30	16.9	6.6	3.1	7.2	9.2	86.1	44	45.1	12.5	46.2	Absent	-3.34	132	5.33				
Case 24	12.6	40.6	4.92	0.7	3.18	17540	N47L49E1M3	NBP	82.5	25.7	31.1	15	50.5	3	4.9	9.5	87	43	53	12.4	54.5	Absent	-3.18	122	3.1				
Case 25	11.4	37.2	5.07	0.7	3.6	14390	N35L58E3M4	NBP	70.3	20.4	29	15.8	300.5	4.6	7.9	7.6	79	45	43	11.2	45	Absent	-3.88	122	3.2				
Case 26	3.2	11.3	2.5	7.7	3.35	32700	N69L23E0M8	DA WITH NL	97.5	28.2	28.9	35	236.9	3.4	6.7	7.7	79.3	44	46.6	12	46	Absent	-3.8	118	3.6				
Case 27	7.8	27.8	4.46	0.5	5.27	9830	N25L70E2M3	MHA	61	22	29	16.2	5.8	3.29	6	6	68	41	42	12	43	Absent	-3.58	120	3.3				
Case 28	11.7	37	4.5	0.8	3.73	12000	N30L65E2M3	NBP	88	24	33	13	220	4.1	6.8	6	70	44	44	12	45	Absent	-3.55	122	3.6				
Case 29	10.4	34.3	4.34	0.8	5.22	10000	N30L65E2M3	MHA WITH THROMBOCYTOSIS	69	23	31	17	6.7	3.1	5.6	7.3	77	43	44	11.1	45.1	Absent	-3.1	124	2.9				
Case 30	8.1	29.3	5.09	0.8	5.54	13620	N36L60E1M3	MHA WITH THROMBOCYTOSIS	57.5	15.9	27.7	20.1	9.23	3	4.2	7.7	79	46	48	11.4	50	Absent	-3.1	121	2.9				
Control 1	13.8	45.5	5.1	0.6	3.9	13070	N44L50E2M4	NBP	89.1	27	30.3	14.3	150	4.2	7.1	8.8	78	46	44.5	12.9	46	Absent	-1.11	132	4.13				
Control2	13.6	39	4.8	0.6	1.91	8000	N40L52E2M6	NBP	98	29.3	32	12.9	210	4.2	8.1	13.8	92	46.5	50	15	48	Absent	0.41	139	3.9				
Control 3	11.3	36.1	4.25	0.7	2.6	7130	N46L45E3M6	NBP	83.1	25.9	31.2	12.8	140	4.2	7	9.9	80	46	47	12.8	47	Absent	-0.81	138	4				
Control 4	12	37.3	3.5	0.8	2.15	8500	N44L50E2M4	NBP	81	26.8	30.5	13.4	280	4	8	13.8	94.5	47.4	45	12	46	Absent	0.03	136	3.2				

S.NO	Hb (gm%)	PCV (%)	RBC (millions/cumm)	RETIC (%)	PLATELETS(lac/cumm)	TLC(/cumm)	DLC	PS	MCV (fL)	MCH (pg)	MCHC (gm/dl)	RDW (%)	Serum Ferritin (ng/m)	Serum Albumin (gm/dl)	Total proteins (gm/dl)	Weight (Kg)	Height/ Length (cm)	HC (cm)	CC (cm)	MUAC (cm)	AC (cm)	Oedema	Weight for Height (SD)	Serum sodium (mEq/L)	Serum Potassium (mEq/	Serum Iron	Vitamin B12 (pg/ml)	TIBC	Transferrin saturation
Control 5	11.3	34.1	4.27	0.7	2.4	7.79	N46L46E2M6	NBP	78	26.2	31.2	13.3	300	4.3	7.8	8.4	72	42.5	42.5	13	42	Absent	-0.67	128	3.6				
Control 6	11.2	34.5	3.8	0.7	2.9	8950	N41L51E2M6	NBP	82	27	29.8	12.8	225	3.1	6.8	9.7	80.5	45.8	45	12.6	45	Absent	-0.72	124	3.4				
Control 7	11.8	35.2	4.44	0.8	2.23	6200	N47L47E2M4	NBP	80.7	24.7	30.1	12.9	200	3.9	7.2	13.4	93	47	46	13	47.6	Absent	0.01	130	3				
Control 8	11.4	34.1	3.55	0.9	2.15	8330	N46L45E3M6	NBP	83.9	27	32.2	14.5	238	3.7	6.8	13	93	46.8	47	13.1	48	Absent	0.01	126	3.1				
Control 9	15.4	45.7	4.36	0.6	3.37	13650	N34L63E1M2	NBP	84	28	32	13.7	135	5	7.9	10.7	81	44	45	11.9	46	Absent	0.07	137	4.5				
Control 10	13.2	40.2	4.7	0.7	4.88	14950	N60L30E3M7	NBP	85.4	28.1	32.9	13.3	225	4.7	7.5	17	110	50	53.4	15.1	56	Absent	-0.32	139	3.7				
Control 11	11.6	38.9	4.79	0.7	4.2	9120	N45L48E2M5	NBP	79	26	31	12.3	160	3.9	7.8	12.3	89	46	42	15	45	Absent	-0.09	140	3.9				
Control 12	16.8	50	5.57	0.5	2.69	6980	N45L50E3M2	NBP	89.1	29.4	33	13	250	4	7.92	12.5	88	46	45	13	46	Absent	0.09	132	3.2				
Control 13	12.4	39.1	4.8	0.8	1.67	9280	N48L50E1M1	NBP	82	27	32.4	12.7	225	4.2	7.5	14.2	93.3	48	46	14	47	Absent	0.06	126	3				
ontrol 14	14.2	42.7	5.36	0.5	2.7	7900	N60L33E2M5	NBP	79.7	26.5	33.5	12.1	149	4.7	8.1	15	81	46	50	13.8	46	Absent	3.89	139	4				
Control 15	12.9	41.9	5.11	0.7	3.24	8700	N54L39E2M5	NBP	82	25.2	30.8	12.8	280	4.5	7.9	10.6	81.4	45	45.5	14	46.4	Absent	0.1	136	3.8				
Control 16	11.6	37.1	4.97	0.8	2.46	10890	N31L63E3M3	NBP	85	28	33	12.8	300	4	7.6	9.7	82	50	54	15	49.5	Absent	-1.53	136	2.9				
Control 17	12.7	38.6	4.5	0.9	4.3	7580	N43L51E2M4	NBP	86	28.2	31	13	320	3.9	7.9	12.9	90	48	49	15	50	Absent	0.22	130	4				
Control 18	12.2	38.1	4.74	0.6	2.9	8000	N58L35E2M5	NBP	83.4	27.2	30	12.8	280	3.6	7.7	13.6	96.8	48	50	17	51	Absent	-0.8	122	3				
Control 19	11.2	37.5	4.56	0.8	4.4	9350	N40L45E4M9	NBP	80	26.1	32.3	13.1	190	4.1	7.8	12.5	90	47	43	13.3	42	Absent	-0.38	136	3.7				
Control 20	12.5	36.1	4.65	0.6	2.9	6770	N45L53E1M1	NBP	77.6	26.9	34.6	12.5	140	4.8	7.8	10.7	82	47	46	12.6	48	Absent	0.03	136	3.5				
Control 21	13	41.7	4.89	0.5	2.8	8260	N43L49E3M5	NBP	86.2	28.3	33.2	12.2	175	4.4	7.1	11.8	87	45.4	45	13	46	Absent	-0.38	125	3.1				
Control 22	11.8	37.1	5.41	0.8	4.4	8000	N45L48E2M5	NBP	84	27.3	32	13	235	4.2	7.9	14	88.3	47.5	47	14	43	Absent	1.4	134	4.52				
Control 23	12.3	40.2	4.64	0.8	3.82	8230	N46L44E2M8	NBP	86.8	27.1	31.2	12.9	270	4	6.9	15	103	47.2	49	13	50	Absent	-0.95	130	3.5				
Control 24	11.9	36.3	4.36	0.7	2.87	6160	N55L35E4M6	NBP	83.3	27.3	32.8	12.6	201	5.4	8	12	89	48	50	15	51	Absent	-0.66	128	3.8				
Control 25	12	35.4	4.87	0.9	4.32	7150	N40L55E1M4	NBP	84.1	28	31	13	190	4.6	7	11.3	83.3	44	45	13	45	Absent	0.04	130	4				
Control 26	12.4	39.6	4.7	0.9	2.45	8763	N57L39E1M3	NBP	86	29	32	12	195	4.1	7.1	12.5	88.3	46	46	14	46.1	Absent	0.25	122	3				
Control 27	13.2	42.3	4.98	0.8	2.33	6550	N39L55E2M4	NBP	82	27.4	33	14	208	4.4	7.4	9.2	73.5	47	48	14	47.2	Absent	0.01	120	3.3				
Control 28	13	41.7	4.89	0.7	3.56	5150	N43L47E4M6	NBP	85.6	27	31.2	13.8	250	4.1	8	11.9	80	48	47	12	49	Absent	1.53	130	3.6				
Control 29	11.2	35.4	4.15	0.9	5.34	13560	N267L65E3M5	NBP	85.3	27.1	31.6	15.5	224	4.7	7.88	8.6	72	46.5	45	13	48	Absent	0.04	132	3.5				
Control 30	12.1	39.8	4.48	0.6	3.96	7850	N45L46E3M6	NBP	88.9	27.3	30.3	14	205	3.8	6.7	12.6	89	46	44	13.7	45	Absent	0.18	126	3.1				