Medical Studies Series (1)

Severe Acute Malnutrition

Dr. Adil Abdalla Babiker

First edition 2024 AD

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Dedication

To my parents who gave much and earned little.

To my family who support me through all stages of writing this book.

To my teachers who made malnutrition a lovely subject.

To my students who need this book.

To my patients who inspire me and need young doctors to know how to manage them.

Table of contents

Abbreviations

PEM	Protein - energy malnutrition
SAM	Severe Acute Malnutrition
PCM	Protein - calorie malnutrition
WHO	World health organization
Alb	Albumin
BMI	Body Mass Index
Glo	Globulin
Ig	Immunoglobulin
Wt.	Weight
Ht	Height
SFT	Skin fold thickness
H.c	Head circumference
Ch.c	Chest circumference
MAC	Mid-arm circumference
Lab	Laboratory
Abd. G.	Abdominal Girth
CMI	Cell - Mediated immunity
DHSR	Delayed hypersensitivity reaction
MUAC	Mid Upper Arm Circumference
OTP	Out Patient Therapeutic Program
RUTF	Ready -To -Use-Therapeutic Food
TFC	Therapeutic Feeding Center
F-75	Therapeutic milk used in Phase 1 of treatment for severe Malnutrition
CMAM	Community-Based Management of Acute Malnutrition
MAM	Moderate Acute Malnutrition
F-100	Therapeutic milk used in Transition Phase and Phase 2 of the treatment of severe malnutrition
IMCI	Integrated management of childhood Illness

Preface

Malnutrition is a silent emergency affecting over 50 million children globally with increased risk of infections and increased morbidity and mortality causes leading to this problem are poverty, pathogens, pollution and politics with poverty as corner stone.

The size of malnutrition in the community like the top of iceberg appear only the top which present to hospitals and health facilities and the remaining at the community so community outreach to detect these cases and treat them at the community before develop complications also children with moderate acute malnutrition (MAM) need supplementary feeding to avoid occurrence of SAM.

Malnutrition affect all organs and even all cells so treating doctors need to know all changes for proper management as biochemical, hormonal, enzymes, digestion, absorption, hepatic, renal and cardiac functions. as malnutrition affect immunity both cellular and humeral make them prone to many infections and their response differ from well nourished child so the evaluation of malnourished differ from a well nourished child.

Assessment and treatment children with SAM differ because renal, hepatic and cardiac functions are affected so avoidance of intravenous fluids unless indicated so to keep malnourished by avoiding intravenous fluids.

Adil Abdalla Babiker

Chapter One

Introduction

Chapter one
1. Introduction

1.1. Definition:

Various terms are used to describe the individual nutritional state, the terms malnutrition and under nutrition are employed differently by different investigators. It is an impairment of health and physiological functions resulting from the failure of an individual to obtain all the essential nutrients in proper amount and balance

WHO defined protein-energy malnutrition (PEM) as a range of pathological conditions arises from coincidental lack, in varying proportions of protein and calories occurring most frequently in infants and young children and commonly associated with infections.

Once defined as chronic hunger PEM is self - perpetuating within the poor populations of socially backward and economically struggling nations, it must clearly be demarcated from generalized famine following massive disasters such as warfare, droughts, floods or earth quakes and its predominating social pathogenesis clearly differentiates PEM from individual malnutrition secondary to other diseases.

PEM may be understood as a multi factorial systemic pathological continuum of potentially progressive severity.

1.2. Nomenclature:

Historically, marasmus (from a Greek word) meaning to wasting was recognized for hundreds of years as being with gastro - enteritis a major contributor to high infant mortality . In the early part of this century reports from central Europe of the so-called starch dystrophy attracted little attention. In Latin America they used the name dysgraphia pluricarrncial for multiple deficiency growth failure .

The classic description by Cicely Williams in 1933 in Gold coast of a disease attributed to protein deficiency which she named kwashiorkor (taken from Ga language of Ghana) meaning the disease the first child gets when the second is on the way).

In Uganda the same disease called by the Baganda tribe obwosi meaning the disease the child gets when the mother becomes pregnant.

The disease kwashiorkor usually occurs during late infancy and early childhood 1-3 years, resulting from a diet low in protein with relative excess of carbohydrates, they presented with edema, growth retardation, muscle wasting and psychological changes. Occasionally they show hair and skin changes and hepatomegaly. While marasmus, usually occurring during infancy is characterized by growth retardation with severe wasting of muscle and subcutaneous fat.

These two distinct syndromes marasmus and kwashiorkor occur at either end of the spectrum, in between varying degrees of signs of each are found mixed together in what is termed marasmic-kwashiorkor.

In 1959 the term protein-calorie malnutrition was used to include the mild and moderate degrees and all the clinical types of the severe disease but this was abandoned by the expert group meeting in 1970, and since the adoption of the international system of unit (S1) with the replacement of the term calorie by Joule as unit (1 calorie = 4.1854 J) and energy for general use has resulted in protein-energy malnutrition.

1.3. Classification:

WHO classification:

Diagnostic criteria based on WHO standards for malnourished children aged 6 through 59 months:

● ***SAM***:

MUAC <115 mm, or

WHZ <–3, or

Bilateral pitting edema

● ***MAM***:

MUAC 115 to 124 mm, or

WHZ –2 to –3

●***Stunting (indicates chronic malnutrition)***:

Moderate stunting: WHZ –2 to –3

Severe stunting: WHZ <–3

1.3.1. Wellcome classification:

Is based on the presence or absence of edema and the deficit in body weight.

Percentage of normal wt. for age	Oedema	
	Present	Absent
60 – 80	Kwash	Under-weight marasmus
60 >	Marasmic-kwash	

It`s main Virtue is simplicity but cannot be applied when the age of the child is not known and it does not take into consideration the chronicity of the disease process.

1.3.2. Gomez classification:

This proposed by Gomez which classify weight deficit in degrees as follows:

First degree (mild)	76 - 90%
Second degree (Moderate)	61 - 75%
Third degree (Severe)	60% or less,

It is most widely used for nutritional surveys in communities but is less applicable in classifying the severe form of PEM.

1.3.3. Jelliffe classification:

Jelliffe modified Gomez classification by defining four groups at interval of 10% body weight deficit.

1.3.4. Bengoa classification:

Bengoa Included in third degree malnutrition all cases with oedema regardless of body weight.

1.3.5. McLaren classification:

McLaren Classified severe PEM by using clinical signs such as edema, dermatosis, edema plus dermatosis, hair change, and hepatomegaly which were combined with different serum albumin concentration. The score as follows:

	Point
Edema	3
Dermatosis	2
Edema + Dermatosis	6
Hair changes	1
Hepatomegaly	1
Serum albumin:	
< 1 g/dl	7
1 - 1.4 g/dl	6
1.5 - 2.4 g/dl	5
2.5 - 2.9 g/dl	4
3.0 - 3.4 g/dl	3
3.5 - 3.9 g/dl	2
≥ 4.0 g/dl	1

Score from 0 - 3 = Marasmus

From 4 - 8 = Kwashiorkor

No anthropometric measurements were used in this system except that an upper limit of 75% of expected weight for age was applied to patients with marasmus. WHO published recommendations for the analysis of data for children up to the age of 10 year' on a cross - sectional basis, using height for age and weight for height .

Chapter Two

Etiology

Chapter Two

2. Etiology

PEM has an extremely complex and multifactorial etiology in which the quantity and quality of food are only two of the involved elements.

2.1. Poverty:

Poverty was the corner - stone of PEM with poor diet, an increased

Susceptibility to disease, decreased birth rate, poor economic productivity and illiteracy.

2.2. Factors at the level of the child:

Factors that operating at the level of the individual child which can

be grouped under three headings: lack of food, infection and

Psycho social deprivation.

2.3. Weaning practices:

The time of supplementation and type of food and the way the mother wean her child may predispose to psycho social trauma and malnutrition.

2.4. Infections:

There is an interaction between infection and malnutrition, infection reduces appetite, anorexia may be a central phenomenon or local causes such as oral thrush, sore tongues and aphthous ulcers. Infections either enteral or parenteral results in a negative nitrogen balance bringing out into the open latent cases of malnutrition.

2.5. Social factors:

Some practices in some societies affect the child nutrition example customary habits of ceasing breast feeding once a new pregnancy has

started. Food taboos also play their role in some communities including stopping milk and other food during gastro - enteritis

2.6. Factors at the national level:

These include inequalities between and within countries. According to the World Bank, the re-allocation of only 2-3% of world income per year would eradicate poverty by the year 2000 .

According to UNICEF reports that adequate investments in human develop, including health services, nutrition, education and housing would amount to 30 - 50 billion dollars a year which is only 5% of world military spending. So the long run of malnutrition can only be eliminated through political actions by distributing wealth between northern and southern hemisphere and within countries.

Chapter Three

Pathogenesis

Chapter Three

3. Pathogenesis

There is yet no clear explanation as to why children present with such different patterns of malnutrition - marasmus and kwashiorkor. Kwashiorkor is common in areas where high energy - low protein foods are the staple diet eg maize, cassava and rice. In animal studies hypoalbuminemia occurs most rapidly with high energy low protein diets especially when the energy source is sucrose.

3.1. Failure to adapt theory:

This theory proposes that, whereas a child who develops marasmus in the presence of a protein energy deficient diet has been able to utilize his own proteins (From muscle) to supply amino acids to the liver, the kwashiorkor child cannot mobilize sufficient essential amino acids, and thus the liver is unable to maintain serum albumin production, and hypoproteinemia and edema develop.

3.2. Hormones:

Variation in hormonal levels e.g., cortisol, insulin and growth hormone have been proposed, which facilitate mobilization of amino acid from body stores in marasmus and inhibit this response in kwashiorkor. In PEM, growt hormone is high in kwashiorkor and cortisol tends to be higher in marasmic rather than edematous cases. Elevated cortisol may be due to decreased clearance and or increased stress due to infection. Thyroxine (T4) is usually normal but T3 may be depressed. Reduced activity of thyroid hormones may depress the metabolic rate and conserve energy. In PEM there is a glucose intolerance which may be related to decreased insulin release or insulin resistance.

3.3. Micronutrient deficiency and free radical damage:

A free radical is an atom or molecule with an unpaired electron and most are unstable and highly reactive. Free radicals production occurs during cell damage from infection and toxins such as endotoxins and aflatoxins . There are many systems in the body which mop up these excess free radicals assuperoxide and glutathione peroxidase.

Vitamins such as A and E and trace element, such as copper, zinc, manganese, iron, selenium and Sulphur amino acids play an important part in the enzyme system necessary to protect against free radical damage . Dietary levels of these materials are low in areas where kwash is common. This will result in accumulation of free O2 radicals which may then damage liver epithelial as well as other cells giving rise to kwash. Radical oxidant stress results in low levels of blood glutathione and reduced glutathione peroxidase which have been detected in kwashiorkor and marasmic- kwash. than marasmus.

3.4. Aflatoxins:

Aflatoxins are mycotoxins produced by the fungus Aspergillus flavus and are found in many foods in tropical countries as maize, sorghum and groundnut, they have been associated with liver damage and one of the results of liver damage is Hypoalbuminaemiathey also demonstrate that aflatoxins in the blood more often in higher levels in children with kwashiorkor than marasmus and least often in healthy controls. Aflatoxin have been detected in the livers of kwash. But not as yet in marasmic children.

3.5. Incidence, morbidity and mortality rates:

Data based on anthropometric measurements estimated that in developing countries ten million of children under 5 years of age

suffered from malnutrition, eighty million from moderate and 120 million from milder forms of malnutrition .

In community surveys done in some developing countries showed that the prevalence of kwashiorkor and marasmus in the range of 0 - 7.6% while the prevalence of children weighing less than 60% of the standard weight in the ranges of 0.5 -4.6%.

The same studies showed that the prevalence of severe and moderate cases of PEM on the range of 20% and in few countries it is over 30% With regard to the number of children in the world who suffer from malnutrition it has been estimated that in 1966 of the 667 million children under the age of 14 years living in developing countries, 269 million were undernourished.

Malnutrition affects the host immunity predisposing him to various infectious diseases. Such as gastro – enteritis, pneumonias and sepsis. Concerning mortality rates, Gomez found that the mortality rate was 31%the major cause of death are diarrheal diseases and bronchopneumonia. Pats stated that the average mortality rate for children up to one year old is more than five times as large in the developing countries as in the developed countries. The difference is far greater in children between the ages of one to four years where the mortality rate is ten to forty times higher in developing than in developed countries.

3.6. Magnitude of malnutrition in the world:

The first clinical description of kwashiorkor was given by Procter in 1927 although popularization of the syndrome was attributed to Williams in 1933. Is well described in Asia in the early 1950. It is also described in Europe during the last world war in 1940 from ltaly, Germany, Greece, France and England to which the name]flour feeding injury was given. Until the third decade of this century, it was certainly a common scourge in the now affluent industrial nations. It has been

eradicated wherever an authentic social development was achieved and whenever sanitary conditions are improving at an optimal pace.

Marasmus has been recognized for centuries, it is far commoner in many parts of the world. It is estimated that more than a third of under five-year old children are malnourished WHO data from a cross-section of population in developing countries indicate both regional and global trends in malnutrition.

3.7. Magnitude of malnutrition in developing countries:

PEM is indeed one of the silent emergencies of the world. It is now recognized that half of mankind are underfed. food]production appear to be fundamental because in developing countries where PEM is prevalent, the production of food, especially that rich in protein is insufficient to meet the nutritional need of the population. Low food production results mainly from a failure to employ modern agricultural techniques as well as an additional reduction in productive capacity because of debilitating infections and nutritional diseases. Inadequate transportation and preservation facilities may result in the availability of food in some areas, whereas serious deficiencies may exist in other areas and among certain population groups. People in tropics and subtropics were severely affected than others. Over two third (80%) of the world malnourished children live in Asia especially the southern part, 15% in Africa and 5% in Latin America.

3.8. Magnitude of malnutrition in Sudan:

The exact incidence is not available. The percentage of admissions of PEM to the pediatric units in Khartoum in 1966 was 10%. However, it is thought that the number currently is much more than this. Some reports have shown an incidence of malnutrition as high as 50% in welfare clinics. Marasmus and Kwashiorkor which are the severe form of PEM, represent only the top of the iceberg. For every case that

presents, there are 10 - 20 more who did not a veil themselves to medical attention and 10 - 20 more of grade one and two PEM . Two thousand and hundred cases were admitted to Medani hospital during 1966 - 67, one third (700 cases) were diagnosed as marasmus and kwashiorkor in the ratio of 3:4 . The majority of cases occurred between the age of 6 months and 36 months. Marasmus, usually occurring at an earlier age, is reaching a peak of maximum incidence earlier than kwashiorkor. Hospital mortality was found to be as high as 20%. Yousif in a field survey at the Hag Yousif Village examining 304 children between the age 1 - 4 year, has found that incidence of marasmus is 3.9% and that of kwashiorkor is 2.6% of the cases. Corkill reported that breast feeding continued for up to three years in Sudanese children, while Culwick reported a range of 13 - 32 months breast feeding. Munoz in a specific PCM prevalence survey which was conducted in parts of Khartoum, Northern and Blue Nile provinces reported the following incidence related to age:

6 months	39.6%
1 year	64.7%
2 years	28.2%
2½ yeare 2	3.7%
3 years	52.5%

Munoz has recommended comprehensive PEM surveys at the community level where previous surveys show an incidence of more than 30%.He has also reported on the nutritional

status of a sample of 5356 children from the same provinces aged 3-18years, classifying 53.3% of them as cases of malnutrition. These figures are close to the results of surveys in the Eastern meditarenian region which show that as many as 6- 80% of the children between the age of 6 months and two years are malnourished from the commencement of the weaning period.

This is due to the fact that, during the first six month, of life the breast milk is completely a adequate as well as it is adapted to the needs of the infant. It is also reported that up to 91.59%.of babies are breast fed until they are one year old, and 35% until they are two years old . A study conducted at the Children Emergency Hospital (CEH) , Concluded that pure kwashiorkor represents only a minority of the total admissions for severe malnutrition, this pattern is similar to other reports from northern Sudan by Omar and Zumrawe and the Middle East and opposite to that seen in tropical countries where kwashiorkor is often the predominant form of PEM admitted to hospitals.

This study also showed the marked seasonal prevalence, with most kwashiorkor admissions occurring in the wet as well as dry seasons which is similar to other results from Africa . There is no hunger period in urban and preurban areas of the north.

Infections, particularly diarrhoea are important precipitating factor, also concluded that, poor socioeconomic circumstances and lack of education are major factors in the aetiology of PEM. Family instability, decline in breast feeding and the late introduction of mixed feeding, which are important factors in other countries, do not appear to play a prominent role in the aetiology of malnutrition in the Sudan. The Study also suggested that marasmus may be more related to poverty ,reduced food availability and lack of maternal education than Kwashiorkor. The study did not reveal any socio - economic factors, specific deficiencies or imbalances in the diets that shed light on the aetiology of kwashiorkor. Hassan and Rabie conducted a study in Sudanese children for detecting the earliest manifestations of PEM to facilitates their management, reduces their duration of stay in hospital and to increase their rate of recovery, they showed that steady reduction in weight is an early manifestation of PEM, they prefer to apply local standard of growth for normal in Khartoum.

Chest / head circumference ratio measurements were done for 200 children, any child with a ratio of one or above is considered to be well - nourished. Early cases of PEM have a ratio of 0.9-1. A ratio of less than 0.9 indicates well established PEM. The triceps skin folds of the same 200 children were measured, the range of readings was as follows:

Normal Children	8 - 13 mm
Early cases of PEM	5 - 7 mm
Severe cases of PEM	2.4 - 5 mm

They also estimated the Kyneurenine (Which is a product of tryptophan catabolism) in the sera of 23 cases with malnutrition, the results were as follow:

Normal Level	10 - 16 mg/100 ml
Early malnutrition	8 - 10 mg/100 ml
Moderate malnutrition	8 - 10 mg/100 ml
Severe malnutrition	2 - 5 mg/100 ml

Durria (43) showed that undernutrition is widespread across north Sudan. Overall, one in seven children was thin relative to their age. The highest levels of chronic undernutrition were in central and Eastern regions which are the major agricultural areas.

The rural and nomadic children are more undernourished than urban children. She also concluded that growth retardation in children begins at an early age and continues up to the age of 2½ years after which the nutritional status improves. The main causes of malnutrition are the late introduction of supplementary feeding, poor access to food as well as ignorance of the importance of appropriate feeding for children less than 5 years . Alawia in a paper published in 1988 concluded that the type of grain consumed associated with nutritional status which was better when wheat rather than sorghum or millet was the staple.

Kamal presented a paper on mortality levels and health in relation to nutritional status, he showed that mortality rates in under five years old are variable. The overall rate of 121 / 1000 is probably on the conservative side and reflects the difficulty of collecting mortality data. This is due to the fact that mothers are either reluctant to admit their child, death or they have recalled problems. He also showed that mortality rates were highest in Red sea and Kassala provinces where undernutrition was the worst, and lowest in Khartoum, Northern and Blue Nile provinces Children in the later provinces also had the least number of illnesses. the data also showed that children who had diarrhea, vomiting and fever were more chronically undernourished than those who did not. In addition children with multiple illnesses were more vulnerable to undernutrition. The prevalence of PEM in preschool children in Sudan was conducted by National Surveys 1988-87 in Six northern regions which included 79595 children. Results of their study showed:

Wasting in	12.4%
Severe wasting in	1.7%
Stunting in	32.1%
Severe stunting in	12.6%

So they concluded that 58.8% of children are malnourished. Another study conducted in Red sea and North Kordofan during 1990 showed:

Red sea	W/H - 2 SD	22-2%
	W/H - 3 SD	8 - 8%
N.Kordofan	W/H - 2 SD	15 - 4%
	W/H - 3 SD	2.3%

In 1991 another three surveys were done in North. Darfur, Eastern state and displaced camps in Khartoum showed the following:

N. Darfur	W/H - 2 SD	16 - 9%
	W/H - 3 SD	3.4%
Eastern state	W/H - 2 SD	14 - 1%
	W/H - 3 SD	4.6%
Displaced in Khartoum	W/H - 2 SD	17.7%

Hendrickse et al recorded high level of aflatoxins in children with kwash. Several studies were done onthe plasma lipid and lipoproteins levels in Sudanese children.

Elhag, El shafa Eltome and Mahagoub documented no difference in lipoprotein levels between cases and control.

Abdalla found that anaemia in PEM was associated with iron and folic acid deficiency.

Mineral and some trace elements were studied by Ahmed (1991) and Hashim who found selenium level to be lowin children with oedematous form of PEM.

Hassain found 29% of hospitalized children with PEM to have xerophthalmia. Omer found significant high level of lgG, lgA and lgM in malnourished children compared to control.

Chapter Four

Biochemistry of protein-energy malnutrition

Chapter Four

Biochemistry of protein-energy malnutrition

Kwashiorkor and marasmus are two advanced forms of PEM. It is suggested that marasmus characterized by severe growth retardation but remarkably well preserved metabolic processes and represents a state of good adaptation to the stress of PEM. Whereas kwashiorkor in which there is outbreak of adaptation, resulting in the characteristic biochemical and clinical picture of kwashiorkor.

4.1. Protein and amino acids:

4.1.1. Albumin:

In severe PEM total body protein was reduced. In marasmus the serum protein concentration is either normal or slightly reduced.

Plasma proteins are reduced in kwashiorkor, the greatest reduction is in the albumin fraction albumin levels below normal results in development of oedema.

In marasmic - kwashiorkor total protein and albumin were also found to be significantly reduced compared to the control values. The metabolic rate of albumin in severely PEM is reduced to about half the rate found after recovery.

4.1.2. γ globulin:

Unlike albumin, globulin was not affected by nutritional state, and their rate of synthesis may even be increased in the presence of infection. Plasma transferring concentration is remarkably reduced in severe PEM

.

4.1.3. Immunoglobulin:

There is significant reduction of lgG in children with kwashiorkor, while the concentration of serum 1gA and lgD were significantly increased, elevated levels of lgM noted in children with marasmus and combined PEM, all these changes returned to normal values with renutrition.

Taddesse showed that in kwash lgG and lgM values were lower than control, while lgA levels were higher. Several other studies showed normal or higher levels of lgG, lgM, lgA . Hassan showed lgG , lgM and lgA levels to be significantly higher in PEM compared to the control group.

Reed stated that serum albumin was significantly reduced in children with kwashiorkor, both marasmic and kwash group had significantly lower concentrations of serum transferrin and it was also significantly lower in the group with kwash than marasmus. Furthermore, children who died had significantly lower concentrations of serum transferrin than those who survived.

Taiwo studied plasma proteins in Nigerian children with PEM , he showed significant ($P < 0.05$) increase in plasma albumin levels in all the 4 types of PEM due to the increased synthetic activity of the liver when the protein is available.

4.1.4. Amino acid metabolism:

In severe PEM the total plasma amino acids concentration is reduced to one half the normal value. In kwashiorkor there is a fall in the plasma concentration of most of the essential amino acids. The concentration of the non - essential amino acids was fairly well maintained or even increased the branched chain amino acids show the largest reduction in plasma concentration .

Hassan and Rabie found that the amino acid concentrations were imbalanced in cases of kwashiorkor where the amount of essential amino acids are reduced considerably, while

that of non -essential amino acids remain unchanged. They also found that the level of kyneurenine, a product of tryptophan catabolism in serum is lower and is directly proportional to the degree of malnutrition.

Alanin concentration is found to be reduced in severe kwashiorkor while plasma lysine is maintained within normal, Phenylanine concentration in plasma is well maintained, possibly because of the decreased phenylanime hydroxylase activity but there is also a marked fall in tyrosine concentration, thus the phenylalanine tyrosine ratio is law .

4.2. Carbohydrate metabolism:

Fasting blood glucose is lower in malnourished than recovered or normal children.In marasmus Alleyne and Scullard found that there is a 50% reduction in glycogen stores. They also showed that, in Jamaican children hepatic glycogenolysis was not impaired, the activity of hepatic glucose - 6 phosphatase was increased and that of liver phospharylase activity was normal. This was thought to be a remarkable adaptation to fasting which resulted in the production of glucose rather than utilization of energy. In kwashiorkor the incidence of hypoglycaemia (blood sugar concentration less than 40 mg / 100ml) Vary from area to area. Mild hypoglycaemia (blood sugar between 20 and 40 mg) is relatively common due to impairment of blood glucose and homeostasis in the first week of treatment. Severe hypoglycaemia (blood sugar less than 20 mg/100 ml) Carries a poor prognosis and is found with hypothermia, coma and infections .

4.3. Lipid metabolism:

In marasmus, normal or increased concentrations of plasma triglycerides, cholesterol and β Lipoprotein . Recently it was reported that plasma

total lipids and lipid fractions were normal in marasmic patients both on admission and during recovery, but α - lipoprotein levels and phospholipids are depressed . Plasma free fatty acid (FFA) concentration in marasmus has variously been reported as increased, variable or normal. In washiorkor low fasting levels of total lipids measured as low concentrations of triglycerides , cholesterol and phospholipids.

The fasting plasma concentraion of β lipoprotein is reduced while that of α lipoprotein has been reported as normal or reduced .

Truswell Showed a strong negative correlation between the serum concentrations of β - lipoprotein, total cholesterol, triglycerides and the degree of fatty liver in patients with marasmus and kwashiorkor.

On re - feeding with a low fat diet, most workers have shown that there is an abrupt early rise in triglyceride concentration which then falls more slowly to normal values. Serum total cholesterol and phospholipid show a similar but less pronounced patternto normal values. Other studies conducted among Jamaican children with kwashiorkor and fatty liver have shown that fasting serum triglyceride levels may sometimes be normal and remain so during recovery .

In Sudan, Ahmedfound that the serum total lipids levels in kwashiorkor and marasmic - kwash groups was significantly reduced.

Also El tom and Ahmed found that the plasma triglycerides were significantly lower in kwashiorkor and marasmic- kwashiorkor than that of the controls and following treatment the plasma triglycerides were not significantly different from that of the control.

4.4. The fatty liver:

Due to fat accumulation in the liver parenchyma, the cells surrounding the portal tracts at the periphery of the liver lobules are affected first then fatty changes progress towards the centre. The liver increases in size, become pallor in colour and firmer in consistency. It has been

consistently associatedwith low concentrations of plasma lipids especially triglycerides. This fat could accumulate in the liver of patients with kwashiorkor because of the excessive influx of free fatty acids from adipose tissue, or by incorporation into plasma lipoproteins. Excessive synthesis of fat in the liver from dietary carbohydrates has been suggested.

4.5. Blood urea and electrolytes:

There is a decrease in total body potassium in malnourished children, the lowest values were found in the severest malnourished children.

In Nigerian children with PEM Taiwo and Thomas showed that plasma electrolytes potassium, sodium, chloride and bicarbonate levels are significantly ($P = 0.05$) higher after recoveryfor all 4 types of PEM. These results support the reports of significant decrease in serum electrolytes in PEM.Potassium depletion alters renal function, including a profound reduction in the capacity of the kidney to concentrate. It is estimated that malnourished patients have a decreased total body potassium, however the plasma potassium concentration was normal . Magnesium deficiency has been reported and may be responsible for tetany observed in some malnourished children, serum iron and copper are also low in kwashiorkor due to reduction in transport protein. The iron content of the liver may be decreased in accordance with the previous dietary history of the child.

The blood urea in PEM is invariably low, unless the patient is dehydrated the low urea may be a reflection to a low protein intake and it rises with recovery. In Nigerian children with PEM Thaiwo and Thomas showed that plasma urea levels were significantly ($P< 0.05$) higher after treatment than before. plasma creatinine levels after treatment are significantly ($P< 0.01$) lower for marasmic- kwash than on admission.

4.6. Anemia in PEM:

In PEM mild to moderate reduction in haemoglobin levels occur, this anaemia, (Hb 8 - 10 g / dl) usually normochromic normocytic slightly hypochromic which correlated well with total plasma protein and resolved on treatment with milk protein folic acid deficiency varies very much from country to country, which is reflected by white cells abnormalities (hyper segmented neutrophils, gaint metamyelocytes) and with a good response to folic acid treatment.

Iron deficiency anemia is more likely to occur in marasmic infants in the first year of life. The erythron is micro normoblastic or normoblastic with hypochromia and anisocytosis. Latent iron deficiency begins 3 or 4 weeks after treatment. Serum B 12 is not affected in PEM and may even be actually raised.

A decrease in red cell survival time has been described in PEM, which may be due to folic acid deficiency. Coward has described an intrinsic defect in the red cell membrane]in kwashiorkor.

4.7. Infections:

Chronic infections depresses bone marrow function, also fever]has shown to depress iron absorption. Malaria as well as hookworm infestations contribute significantly to anaemias in PEM.

Hypoplasia of the erythron may develop, Kondi described two types**:** The first occurs early in treatment, transient and may be due to infection. The second type is more severe, occurs later in recovery and sometimes have a prolonged duration.

Kondi suggested that this aplasia might be due to riboflavin deficiency on the basis of a response to riboflavin.

In Sudan Abdalla , studied anaemia as a manifestation of PEM, Iron deficiency anaemia was found to be the commonest, mainly due to poor diet, infections and parasitic infestations, megaloblasic anaemia has also been described .

Chapter Five

Malnutrition and infection

Chapter Five

Malnutrition and infection

5.1. Interaction between infection and malnutrition:

The interaction between infection and malnutrition is well established. The interaction is complex and is not easy to separate the effect of infection on nutrition from that of nutrition on infection. The most striking investigations, which showed the impact of nutrition and infection on the health of children carried by Puffer and Serrano from a data collected in13 field projects in Latin America. They showed that 57% of the children who died under the age of 5years had immaturity or nutritional deficiencies as a primary or associated cause of death. Nutritional deficiency was the associated cause of 60.9% of the death from infectious diseases as compared with only 32.7% of deaths from other causes. This relation becomes obvious when a natural or man-made disaster drastically curtails the production and distribution of food and simultaneously causes breakdown in personal hygiene, environmental sanitation, and provision of medical care.

The resultant overcrowding of people and the social disorganization favour the spread of infectious diseases. Far more relevant scientifically than these secondary associations, are the

clinical observations as well as experimental studies providing the inter - relationship between infections and malnutrition are direct and causal. These direct interactions fall into two basic patterns: malnutrition generally alters resistance of the host to infection on the other hand, infectious disease exaggerate existing malnutrition.

The combined effect of malnutrition and infection cannot be predicted from the occurrence and characteristic of either alone. Infectious disease nearly always makes Co - existing malnutrition worse.

Furthermore, the consequences of infection are likely to be more serious in malnourished host than in a well - nourished. When infection aggravates malnutrition or malnutrition lowers resistance to infection the relationship between the two can be classified as synergistic. When malnutrition is more likely to discourage multiplication of the agent than to affect the resistance of the host, this interaction is classified as antagonistic.

***Effects of infection on nutrition**:*

Infections may affect nutritional status by diminishing intake and by directly affecting the metabolism of the host . Reduction of intake, usually accompanies many infections. Anorexia may be a central phenomenon or due to local causes such as oral thrush which occurs frequently in malnourished children, a sore tongue, aphthous ulcers and infected cheilosis may

also make eating a difficult task .

***Effects of infection on metabolism**:*

5.2. Effects on proteins and amino acids:

Infection is associated with an increased excretion of nitrogen and a negative nitrogen balance irrespective of the nutritional status of the individual. An increase in tissue catabolism is the initial effect of infection. With increased excretion of urinary nitrogen and prolonged infection plasma aminoacids fall which represents urinary loss with continuing hepatic ureogenesis and gluconeogenesis. In a study done in Uganda Frood showed that combination of respiratory tract infections, malaria and gastro - enerities led to a a dramatic fall in serum albumin, these changes in serum albumin antecedated the clinical appearance of kwashiorkor.

5.3. Effects on carbohydrate metabolisn:

Marked low levels of blood glucose are common due to reduced caloric intake. During severe infection there is a rise in serum cortisol and a fall in insulin level, This high cortisol will stimulate hepatic gluconeogenesis.

5.4. Effects on lipid metabolism:

Severe infections may lead to accumulation of fat in the liver possibly mediated through increased levels of cortisol, also it may be associated with a reduction in plasma levels of β. lipoproteins which may lead to fatty liver. Intestinal infections decrease absorption of fat resulting in steatorrhoea.

5.5. Effects of infections on vitamins:

B complex vitamins:

Experimental studies in man have proved that infections have an effect on B Complex vitamins such as thiamine, niacin, pyridoxine, folic acid and vitamin B12.

Vitamin A:

Concentrations of vitamin A in the blood are appreciably reduced in pneumonia, rheumatoid arthritis, acute tonsillitis and rheumatic fever. Intestinal absorption of vitamin A may also be impaired in the presence of Giardia lamblia.

Rodger have emphasized the lower serum carotene and vitamin A values in patients with hookworm disease compared to those in the same communities who are free of infection. It

has also been observed that xerophthalmia and keratomalacia occur sporadically after infections among malnourished infants in developing countries.

Iron:

The effects of acute infections on Iron metabolism in man]are well documented. Infections influence Iron metabolism most directly through loss of blood resulting in anaemia.Necator

Americanus causes daily loss of 0.05 ml blood lost per worm per day and loss of Ancylostoma duodenale was five to ten times greater than that cited for Necator, they reported a loss per worm per day of 0.26 + 0.045 ml.

An outstanding feature of chronic malaria is the resultant anaemia. This is apparently due to the fact that the malaria parasites meet their high protein requirements by spiliting haemoglobin, leaving large amounts of unused heme as a malaria pigment.

The anaemia associated with chronic infectious disease varies greatly with the nature and severity of the infection. It is not the result of blood loss, overt hemolysis or Iron deficiency, and does not respond to folic acid or vitamin B12.

So Infections affect nutrition through altering absorption, metabolism as well as the excretion of specific nutrients and by reducing appetite also, a frequent customaryhabit is to withdraw of solid food or the change in dietaryhabit during illness which thus resulted in a reduced nutritional intake.

Consequently, it is not surprising that severe or prolonged illness has an adverse effect on growth and nutrition especially when the child is already in a malnourished state.

Several investigators failed to demonstrate effect of the usual infectious diseases on the growth of well nourished children . Other long- term studies of well nourished children have identified

instances in which growth appeared to have been depressed .

Chapter Six

Effects of malnutrition on immunity

Chapter Six

Effects of malnutrition on immunity

6.1. Introduction:

The effects of malnutrition on immunity was observed since ancient times, it was observed that during famine and lack of food there is increased incidence of many infectious diseases this relation between diet and host resistance was mentioned in puranicscriptures in India.

In PEM there is a significant impairment of several aspects of immunity. Including cell-mediated immune responses, secretorylgA, antibody production, phagocytic function, complement system, antibody affinity and cytokine production .

6.2. Cell-mediated immunity:

The initial suggestion that malnutrition could lead to a disturbance of immunity in children came perhaps from the work of Vint who showed that there was thymic atrophy in

children who died of kwashiorkor. This finding passed unnoticed because the role of the thymus in immunity was not well establishedat that time.

Profound structural changes in lymphoid tissues in PEM were observed by Symythe who showed that the thymus issmall with an ill - defined demarcation between cortex and medulla.

There are fewer than normal lymphoid cells and Hassal corpuscles are crowded, dilated, degenerated and occasionallyeven calcified. There is cellular depletion of thymus dependent areas in the spleen and lymph - nodes. In the intestine, lymphoid aggregates are small, and the number

of intraepithelial lymphocytes and of submucosal plasma cells were reduced.Clinically the effects of malnutrition on lymphoid organs was reflected in the small size of the tonsils . The depressed cell-mediated immunity was due to thymic dysfunction with the elevation of the percentage of immature T- lymphocytes (CD1a), it may be postulated that thymic atrophy results in a lack of the epithelial functions responsible for the chemotaxis and differentiation of cortical thymocytes (CD1 a). This was reflected by a decrease in the percentage of mature effector T- Lymphocytes (CD3) and concomitant with a slight increase in the percentage of suppressor - cytotoxic lymphocytes (CD 8) that decrease the CD4 / CD8 Lymphocyte ratio .The effect of thymulin, a hormone secreted by thymic

epithelial cells results in maturation of T Lymphocytes with a decrease in (CD1a), appearance of new mature effector T Lymphocytes (CD3) and a parallel increase in T helper - Inducer (CD4) and T suppressor cytotoxic (CD8)antigen carrying cells.

Other thymic hormones showing the same effects are thymopoiotin and thymosin fraction. Zinc supplementation permitted the recovery of normal thymus size in a acutely malnourished children , it also enhance growth during recovery with improved host defenses .

6.3. Delayed cutaneoushypersensitivity reaction:

It tests CMI, it composed of at least three separate components the sensitization (afferent) Limb entails immunization of thymus derived T. lymphocytes against a macrophage processed antigen, the recognition (efferent) limb is characterized lymphocytes after they recognised and interacte with the antigen deposited in the skin. The inflammatory reaction, probably induced by lymphokines released at the skin site and it is read as a positive skin test. All the three components are found to be affected in severe PEM Fakhir demonstrated that the response to

1. chloro, 2-4 dinitrobenzine (DNC B) was impaired in malnourished children and the skin test returned to normal in most of the patients. Also tuberculin sensitivity was found to be significantly impaired in malnourished children who receivedBCG at birth ($P < 0.01$) . Sinha , in a study found that this delayed hypersensitivity reaction to be significantly impaired in severe malnutrition with kwashiorkor showed the maximum depression than marasmic - kwash, while the marasmic affected little or not at all. In another study David concluded that moderate malnutrition showed a reduction in CMI.Smythe studied lymphocyte transformation test after phytohaemagglutinin stimulation. The distribution for the three cell types Untransformed, blastoid cells and mitotic cells were studiedin children with PEM who showed significantly more unresponsive cells and fewer blastoid or mitotic cells than the control group. In another study David tested the blastogenic response to the mitogen PHA, and including the total peripheral lymphocyte counts and the blastogenic indices in normal, midly and moderately malnourished children. He concluded that, grade II malnourished children demonstrated a significant reduction in the number of circulating lymphocytes although non was markedly lymphopenic. In addition, there was a marked reduction (nearly 50%) in the mitogenic response to PHA by the lymphocytes of those moderately undernourished children. The grade one group, while maintaining normal levels of circulating lymphocytes, also sufferedan impaired inVitro blastogenic response. The values obtained for all underweight children were quite similar, regardless of the degree of malnutrition.

6.3. T-Lymphocytes:

The absolute lymphocyte count significantly low in malnourished children, small lymphocytes count were also reduced significantly in malnourished childrenwith kwashiorkor group exhibited maximum reduction in T.cell percentage and absolute T cell count when compared with marasmic- kwashiorkor or marasmus.

Hassan studied lymphocyte sub population by usingmonoclonal antibodies: mature T Lymphocytes (CD3+), HelperT Lymphocyles (CD4+) and suppressor T lymphocytes (CD8+)

all are found to be decreased in children with PEM but the ratio of CD4 /CD8 did not show a statistically significant difference.

This decreased level of T Lymphocytes and their sub-population may be attributed to lympholysis induced by high levels of cortisol in the serum, impaired differentiation and

maturation of T Lymphocyte precursors as well as the distribution of T Lymphocytes to new sequestration areas. Another explanation for the low level of T lymphocytes sub-population is the decreased density of T cell antigen molecules. In another study Chandra showed that patients with PEM have a pronounced reduction in the proportion of T4 helper cells and a moderately reduced proportion of T8 cytotoxic suppressor cells,proportion return to normal when these patients recover . These findings suggest a reduction in either the number of such cells or the density of antigen molecules on the cell surface.Itis recognized that cell surface glycoproteins may be altered in PEM and other nutritional deficiencies.

Co - culture experiment with the reverse haemolytic plaque technique for enumeration of 1gG producing cell in Vitro demonstrated abnormalities in T cell function.

B lymphocytes of patients with PEM in the presence of T cells of well - nourished controls function normally.This may suggest that PEM alters immunoregulatory mechanisms.

6. 4. Complement system:

The complement system is invariably affected in PEM the total haemolytic complement activity may be reduced and levels of C3, factor B, and other complements are low. Infection may produce an

acute phase reactive increase in complement activity, but more often it further depresses the concentration of complement proteins partly as a result of consumption in antigen - antibody reactions. Owing partly to changes in the complement system the opsonic function of plasma is reduced.David, studied two complement components C3 and C4 in malnourished children on admission and at discharge. In patients with kwashiorkor the level of C3 was significantly reduced while concentration of C4 complement were not affected by the nutritional status. Feeding a mixed diet high in protein and calories for 4 - 5 weeks was accompanied by improvement of immunological parameters and increase in C3 level in most of the patients.

Chemotactic migration of neutrophils may be slower than normal in PEM. Ingestion of bacteria is a normal process, but intracellular bacterial and candida killing capacity is impaired. The metabolic basis for such abnormalities are not defined, but the activity of several enzyme systems involved in bactericidal processes is reduced in PEM.

6. 5. Humeral immunity:

Humeral immunity plays an important role in the defense against bacteria viruses and other pathogens. Frequent episodes of infection in malnourished children suggest that resistance to infection may be lowered due to the effect of malnutrition on humoral immunity.

Studies in childhood malnutrition have been along two main lines:

1. Antibody production in response to antigen.
2. Immunoglobulins and other proteins involved in the immune mechanism.

Chandra Showed that the antibody response to tetanus toxoid was lower but did not reach statistical significance. Cohen and Hansen measured the synthesis role of γ - globulin and found that it was increased in

malnutrition, and the presence of infection led to an increase in the rate of synthesis to a level three times higher than in uninfected children.

There have been several studies on the levels of immnuoglobulins in malnutrition. Watson and Freesmann showed that in kwashiorkor there is a rise levels of 1gG , 1gM and 1gA . Chandra showed that in kwashiorkor without infection there is normal levels of 1gM , 1gA with raised level of 1gG, while in kwashiorkor with infection there is a raised level of all the three immunoglobulins. Keet and Thomas showed thatin malnourished children with infection compared to controls with infection alone there was no difference in the levels of lgG and lgM although serum lgA levels were higher in the malnourished group whereas malnourished children without infection had hypogamaglobulinemia.

Taddesse studied immunoglobulins in kwashiorkor, he found that 1gG and IgM values are lower than values for controls These differences were statistically significant only for IgG concentration.He postulated that the lowered levels of IgG in kwashiorkor was due to the fact that IgG has been known to suffer in catabolism during the catabolic states of the body. It`s degradation can rise up to nine times the normal rates.The plasma cell synthesis rate cannot be completed and the serum level of IgG can fall as low as 100 mg /100 ml such a type of catabolic hypogamaglobulinemia characteristically affects the usually long-lived IgG much more than normally short-lived IgA and IgM.

In malnutrition the body goes into a catabolic state along with degradation of it`s own proteins. Involvement of IgG in such a phenomenon may probably take place in cases of kwashiorkor resulting in reduced levels of IgG.

Watson showed that no changes was detected in the concentration of free secretory component (FSC) in the tears of moderately malnourished (Grade I and II) children. There was a significant difference between normal and severely malnourished children. Chandra studied the effect

of PEM on immune competence, the lymphoid tissues, particularly the thymus which was found to be atrophied. There was a reduction in delayed cutaneous hypersensitivity reaction, fewer T cells especially T helper cells, decreased thymulin activity, impaired secretory IgA, decreased antibody response and affinity, reduced concentration and activity of complements and phagocytic dysfunction.

6.2. Effects of Vitamins and Mineral deficiency on immunity:

Vitamins:

Isolated vitamin deficiencies in man are rare thus most of the data obtained from animal experiments. Beisel demonstrated that deficiencies of folic acid, pyridoxine and vitamin A consistency impair cell - mediated immunity and T. cell dependant antibody response.

Chardra postulated that severe deficiency of vitamin C resulted in impairment of cell - mediated immunity. The effect of vitamin E on immunity is closely linked to the presence or absence of other antioxidants particularly selenium. The mechanism by which vitamin deficiencies produce immuno- deficiency are not uniform, pyridoxine deficiency result in thymic epithelial dysfunction and reduction in serum thymic factor activity, whereas vitamin A deficiency alterations in surface- membrane glycoproteins of lymphocytes may interfere with antigen binding .

Trace elements:

The role of trace elements in regulation of immunity and infection was well recognized . Human zinc deficiency epitomized by the inherited syndrome of acrodermatitis enteropathica, results in pronounced impairment of cell mediated immunity. Zinc supplementation reverse the changes within a few weeks, zinc deficiency increases vulnerability to listeria, salmonella, Coxsacki virus and other pathogens .

Iron:

Iron deficiency is associated with an impaired lymphocyte stimulation, response to mitogen and decreased neutrophil bactericidal capacity . Mc Farlane have shown that the serum transferrin level is a sensitive prognostic indicator of the outcome from malnutrition. He postulated that transferrin may have a bactericidal action and in its absence free unbound iron absorbed from the gastrointestinal tract may favour bacterial multiplication.

Prokaska demonstrated that copper deficiency results in a decreased antibody forming cell response in mice, and a raised vulnerability to avariety of micro-organisms. Chandra stated that selenium deficiency impairsTcell dependent antibody responses, especially in association

with vitamin E deficiency, he also stated that most of the heavy metals depress immunity and increase susceptibility to infectious challenge.

Excessive intake of certain essential trace elements such as zinc also depresses immune responses possibly through reduction in thymic factor activity, changes in lymphocyte subpopulation, sequestration of cells, altered metabolic pathways and changes in hormonal homoeostasis.

Chapter Six

Effects of malnutrition on immunity

Chapter seven

Evaluation of the malnourished child

This can be done by taking history. Perform clinical examination and do some investigations. But in very sick patients avoid frequent handling and necessary investigations taken while patient at bed.

7.1. History and examination:

This done in a recording form by checklist his current complaint, detailed nutritional history, breast feeding duration, weaning, supplementary feeding type and amount of food, milestones, and immunization.

Examination:

Weight and length or height, Oedema, severe pallor, jaundice, signs of vitamin deficiency especially vitamin A oral thrush, Enlargement or tenderness of liver, Signs of circulatory collapse: cold hands and feet, weak radial pulse, diminished consciousness, Temperature: hypothermia or fever, then examen all systems chest for signs of pneumonia or heart failure, abdomen for hepatomegaly .

Investigations:

If available may help to diagnose specific problems but they are not needed to guide treatment, these are:

- Blood glucose, Glucose concentration <54 mg/dl (3mmol/l) is indicative of hypoglycemia.
- Hemoglobin or packed-cell Hemoglobin <40g/l or packed-cell volume <12% is

- Volume indicative of very severe anemia.
- Blood film for malaria.
- Culture of blood and urine.
- Chest X-ray.
- Stool analysis for dysentery and parasitic infestation.

Criteria for admission to feeding programs:

Mid-Upper Arm Circumference (MUAC):

It is simple to use, cheap and easy and can be used by non-medical person.

Moderate Acute Malnutrition (MAM):

MUAC > 115 and < 125 mm.

7.2.3. Severe Acute Malnutrition (SAM):

Children 6–59 months are classified as severely wasted based on MUAC if their MUAC < 115 mm.

The Presence of Bilateral Pitting Oedema:

Bilateral pitting oedema is a form of SAM. It is a characteristic of kwashiorkor. A combination of bilateral pitting oedema and wasting is a severe condition.

Weight-for-Height s (WHO standards):

Admission Criteria for SAM in Children 6–59 Months for Treatment in Inpatient Care:

- Poor appetite.
- SAM with any of the following medical complications:
- Intractable vomiting - Convulsions

- Lethargy, not alert
- Unconsciousness - Hypoglycemia
- High fever (> 38.5° C maxillary)
- Hypothermia (< 35° C maxillary)
- Severe dehydration - Persistent diarrhea
- Lower respiratory tract infection
- Severe anemia
- Eye signs of vitamin A deficiency
- Skin lesion
- IMCI General Danger signs.
- Bilateral pitting oedema +++

7.2. Treatment of Sever Acute Malnutrition:

Initial treatment:

Begins with admission to hospital and lasts until the child's condition is stable and appetite returned, which is usually after 2–7 days, the aims are:

- To treat or prevent hypoglycemia and hypothermia;
- To treat or prevent dehydration and restore electrolyte balance;
- To treat septic shock, if present;
- To treat infection;
- To identify and treat any other problems, including vitamin deficiency, severe anemia and heart failure.

7.3. Treatment of acute complications:

Treatment can be divided into three phases:

Phase I: Treatment of acute complications:

The major cause of death in severe PEM is dehydration, electrolyte disturbance, hypoglycemia, hypothermia and Infections. It is there for these conditions that require immediate attention on the admission of a malnourished child.

To keep child with SAM a life by avoiding blood transfusion and intravenous fluids.

7.4. Fluid and electrolyte disturbances:

Assessment of hydration is difficult because a number of signs that are normally used are unreliable as sunken eyes, depressed anterior fontanel and skin pinch. So, Thirst. Drinking eagerly is a reliable sign of "some" dehydration. In infants this may be expressed as restlessness. Severe dehydration can be diagnosed by significant losses, prolonged capillary refill and weak or absent pulses. Dry mouth dry eyes can be found in malnourished child without dehydration due to atrophy of salivary and lacrimal glands.

Dehydration should be by mouth or nasogastric tube using ReSoMal. Intravenous fluids should not be used because it easily causes over hydration and heart failure, unless the patient is severely ill.

Amount of ReSoMal to give Between 70 and 100 ml of ReSoMal per kg of body weight is usually enough to replace the losses. . This amount given over 12 hours, starting with 5 ml/kg every 30 minutes for the first 2 hours orally or by NG tube, and then 5–10 ml/kg per hour.

Dehydration in which I.V. fluids given over a short duration. Till pulse appear to shift to ReSoMal. Many signs of dehydration are also seen in septic shock so any malnourished child with severe dehydration should be treated for septic shock.

Fluids used:

- Half- strength Darrow's solution with 5% glucose.
- Ringer's lactate solution with 5% glucose.
- 0.45% (half-normal) saline with 5% glucose.

Give 15 ml/kg IV over 1 hour and monitor the child carefully for signs of over hydration. While the IV drip is being set up, insert an NG tube and give ReSoMal through the tube (10 ml/kg per hour). Reassess the child after 1 hourif still repeat, if improve continue ReSoMal.

7.2.3.1.3. ReSoMal Concentration (mmol/l):

- Glucose 125
- Sodium 45
- Potassium 40
- Chloride 70
- Citrate 7
- Magnesium 3
- Zinc 0.3
- Copper 0.045
- Osmolality 300

Local and generalized infections:

Due to defects in both humoral and cellular immunity Infections such as bronchopneumonia, otitis media, urinary tract infection and septicemia are frequently present, the response of malnourished to infection is poor so the classical symptoms and signs may not present making diagnosis difficult. Because bacterial infections are common early treatment of bacterial infections will improve the nutritional response to feeding, prevents septic shock and reduces mortality.

Children with no apparent signs of infection and no complications should be given cotrimoxazole (25 mg of sulfamethoxazole + 5 mg of trimethoprim/kg) orally twice daily for 5 days or amoxicillin syrup15 mg/kg orally every 8 hours for 5 days.

Children with complications or severely ill should be given:

Ampicillin, 50 mg/kg IM or IV every 6 hours for 2 days, followed by amoxicillin, gentamicin, 7.5 mg/kg IM or IV once daily for 7 days.

Other combinations ceftaxime plus penicillin. If no response or the child remains very ill, tuberculosis should be suspected and treated.

All severely malnourished children should be treated for septic shock especially if they had:

- Hypothermia
- Hypoglycemia
- Signs of dehydration, but without a history of significant losses
- Edema with dehydration.

It is difficult to differentiate between severe pneumonia and heart failure because many signs of heart failure can be found SAM without heart failure as edema, hepatomegaly significant weight gain found in heart failure.

Other parasitic infections such as malaria, giardiasis, ankylostomiasis and ascariasis were treated accordingly.

Hypoglycemia:

All severely malnourished children are at risk of developing hypoglycemia (blood glucose <54 mg/dl or <3 mmol/l).

Common presentations are pallor and apneic spells, less commonly were hypothermiabody temperature (<36.5 °C), lethargy, limpness, rolling of the eyes and Convulsions. If hypoglycemia is suspected, treatment should be given immediately without laboratory confirmation.

The frequency varies from one region to another from 10% to 24%, mostly marasmic - kwashiorkor or kwashiorkor, it is thus better to prevent by providing a high concentration of glucose (50 g/L) in the fluid given in the initial phase. If the patient is conscious or can be roused and is able to drink, give 50 ml of 10% glucose or sucrose, or Give F-75 diet by mouth.

For severe hypoglycemia (level below 20 mg/dl) or patient unconsciousness, cannot be aroused or has convulsions, give 5 ml/kg of body weight of sterile 10% glucose intravenously (IV), followed by 50 ml of 10% glucose or sucrose by nasogastric (NG) tube. Then Continue frequent oral or NG feeding with F-75 diet to prevent a recurrence.

Stay with the child until he or she is fully alert.

All malnourished children with hypoglycemia should be treated for septicemia.

Hypothermia:

Hypothermia rectal temperature below 35.5 °C (95.9 °F) or the underarm temperature is below 35.0 °C (95.0 °F), is often an unrecognized problem leading to increased mortality the incidence of hypothermia to be up to 20% in the first week after admission, it is mainly due to decreased Subcutaneous tissue and deficient energy intake. It is prevented by closing windows at night. The child should be properly covered with clothes, including a hat, and blankets. Washing should be kept to a minimum and, if necessary, done during the day. and treated with physical warming. If the rectal temperature is below 35.5 °C (95.9 °F) or the underarm temperature is below 35.0 °C (95.0 °F), the child

should be warmed. Either use the "kangaroo technique" by placing the child on the mother's chest or abdomen (skin-to-skin) and covering both of them. All hypothermic children must also be treated for hypoglycemia and for septicemia.

Anemia:

Malnourished children tolerates anemia well because oxygen demands are low, thus blood transfusion should be undertaken as a last resort using packed cells in a dose 15- 20 ml/kg. In severely ill children who do not respond to treatment small transfusion of whole blood (10 ml / kg) may have dramatic effects possibly via supplying essential micro nutrients.

Folic acid 5 mg / day should be given routinely for all malnourished children. Do not give iron during the initial phase of treatment, as it can have toxic effects and may reduce resistance to infection.

Vitamin supplementation:

All malnourished children should receive 5 mg of folic acid orally on day 1 and then 1 mg orally per day thereafter. Many malnourished children are also deficient in riboflavin, ascorbic acid, pyridoxine, thiamine and the fat-soluble vitamins D, E and K. All diets should be fortified with these vitamins by adding the vitamin mix

Vitamin A:

- <6 months of age 50000 IU
- 6–12 months of age 100000 IU
- >12 months of age 200000 IU

For prophylaxis single dose for therapy three doses day one, day two and after two weeks

Vitamin D:

All patients recovering from PEM should receive vitamin D10 micro gram daily, those who have evidence of rickets should be treated accordingly.

7.5. Phase II Initiation of cure:

Criteria to enter SAM management for children 6–59 months:

- Weight-for-height/length <–3 Z-score of the WHO growth standards.
- Mid-upper arm circumference <115 mm.
- Bilateral pitting edema.
- Visible severe wasting.
- Body mass index (BMI) below 16.
- Criteria for inpatient care:
- Any complication of SAM.
- Any danger sign according to IMCI.
- Poor feeding or fail the appetite test.
- Any medical condition admitting a well-nourished child.
- Severe edema (+++).

Children with SAM who have appetite (pass the appetite test) and are clinically well and alert should be treated as outpatients.

Malnutrition affect all organs (even all cells) as gastro intestinal, liver, kidneys, cardiopulmonary, endocrine and brain all these changes are reversible except brain. These changes should be considered in the initial phase of feeding by giving low solutes diet F75 (75 kcal or 315 kJ/100 ml).

Constituent Amount per 100 ml F-75:

- Energy 75kcalth (315kJ)
- Protein 0.9 g
- Lactose 1.3 g
- Potassium 3.6 mmol
- Sodium 0.6 mmol 1.9 mmol
- Magnesium 0.43 mmol
- Zinc 2.0 mg
- Copper 0.25 mg

Percentage of energy from:

- Protein 5%
- Fat 32%

Osmolarity 333mOsmol/l.:

Patient should be fed from a cup, spoon, dropper, syringe or nasogastric tube. Initially start by frequent and small amounts to be tolerated.

The initial phase of treatment ends when appetite improve and able to complete his feeds.

The volume of F-75 feed at 130 ml/kg per day, gradually decrease the frequency of feeding and increase the volume of each feed until you are giving the child feeds 4-hourly (6 feeds per day).

7.2.4. Phase III: Rehabilitation:

The child is entered the rehabilitation phase when his or her appetite has returned. The aim in this phase is to restore normal weight for height. Indications of entrance into this phase are recovery of appetite and changes of expression, at this stage, the child is moved to a nutritional

rehabilitation center (NRC), and attached to thehospital which provide mothers with some instructions in child feeding and hygiene.

Supplementary feeding programs:

Objective:

The general objective of a Supplementary Feeding Program (SFP) is to treat moderate malnutrition, to prevent severe malnutrition and avoid relapse after discharge from therapeutic feeding.

Admission Criteria:

- Children 6 months to 59months
- MUAC 11 to 11.9 cm and/or
- Weight/Height (W/H) between 70 – 79.9% and/or
- All children discharged from a TFP/ TFU/OTP

Discharge Criteria:

- MUAC >12.5 cm for two consecutive measurements for those children admitted on MUAC 11.0-11.9 and/or
- W/H >85 % for two consecutive measurements
- After a total three months/ 12 weeks in the program if the child is not responding

TFC/TFU/OTP discharges, after 8 weeks if the weight has remained >85% wt./ht.

Community-Based Management of Acute Malnutrition (CMAM):

Community-based management of severe acute malnutrition (SAM) in children 6-59 months includes community outreach, outpatient care and inpatient care for the management of SAM. Community outreach is essential part in CMAM since early detection of SAM before development of complications improve the outcome and hence reduce

morbidity and mortality due to SAM. To detect SAM at community by using simple method the mid-upper arm circumference (MUAC) .

MUAC involves measuring the circumference of a child's left mid-upper arm. MUAC < 115

millimetres (mm) for children 6-59 months indicates SAM or in health facility can use weight for height WFH WHO standards , WFH < -3 z-score indicate severe wasting. Also the presence of bilateral pitting oedema and visible severe wasting indicate SAM.

In the presence of anthropometric confirmation of SAM, it is essential to assess the following clinical signs:

- Anorexia, poor appetite
- Intractable vomiting
- Convulsions
- Lethargy, not alert
- Unconsciousness
- Hypoglycaemia
- High fever
- Hypothermia
- Severe dehydration
- Lower respiratory tract infection
- Severe anaemia
- Eye signs of vitamin A deficiency
- Skin lesion .

Children with SAM without medical complications can start treatment for SAM in outpatient care by using ready use theraputic feeding RUTF Criteria to enter CMAM:

- Bilateral pitting oedema + and ++

or

- Severe wasting (MUAC < 115 mm or WFH <-3 z-score) and Appetite test passed
- No medical complication
- Child clinically well and alert

Discharged cured:

- 15 percent weight gain for two
- Consecutive visits
- Sustained weight gain
- Oedema free for two consecutive visits
- Child clinically well and alert

Children are referred to Supplementary Feeding if available

Community outreach:

Is essential to detect children with SAM before occurrence of complications, it also mobilizes communities.

SUPPLEMENTARY FEEDING FOR CHILDREN WITH MAM

Children with MUAC ≥ 115 mm and < 125 mm for children 6-59 months indicates MAM. moderate acute malnutrition or by a WFH ≥ -3 and < -2 z-score.

7.6. Provision of RUTF:

Provide 200 kcal/kg body weight/day

The child is fully recovered if the following discharge criteria are met:

.

- Fifteen percent weight gain for two consecutive visits
- Sustained weight gain; child has been gaining weight during the last three visits
- No bilateral pitting oedema for two consecutive visits
- Child is clinically well and alert

Chapter Eight

Prevention

Chapter Eight

Prevention

1. **Education**: This was a priority to food, water and sanitation; there is a correlation between female literacy and child survival. Thus education about breast feeding, supplementary feeding and weaning as well as the use of locally available proteins is vitally essential.

2. **Water supply and sanitation**: Improvement in water supplies, Sanitation and hygiene results in a reduction in the incidence of diarrhea.

3. **Immunization**: most of the immunizable diseases predispose to malnutrition such as measles and Tuberculosis.

4. **Breast feeding**: supply adequate nutrient for the child for the first 6 months, protect against infection and prolong the birth interval.

5. Family planning and control of fertility.

6. **Political and economic background**: Political actions to reduce the inequalities between and within countries.

7. Rise the standard of living at a national level through development and constructing schemes and projects.

8. **Nutritional rehabilitation centres**: To educate mothers about diet, feeding techniques, animal husbandry and harvesting local food.

Chapter Nine

References

Chapter nine
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