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JSM Nutritional Disorders

Review Article

Marasmus: An Update and Review of Literature

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Submitted: 24 October 2018

Accepted: 05 November 2018

Published: 09 November 2018

ISSN: 2578-3203

Keywords
- Marasmus
- Mortality and morbidity
- Standard protocolized management

Abstract

Marasmus is a form of severe malnutrition in children mostly occurring in developing countries. It is an important cause of less than 5 mortality and morbidity in developing countries. Due to energy and protein deficiency the child's fat and muscles are reduced significantly resulting in severe wasting and only skin and bone becomes visible. Marasmic child may develop edema called marasmic-kwashiorkor. Inadequate dietary intake of protein and energy rich diet due to poverty or lack of nutrition knowledge, inadequate mother and child health practice, poor health infrastructure, low birth weight and suffering frequently from various diseases are the important causes of marasmus. Marasmic children are vulnerable to various complications including various infectious disease, diarrhoea, hypoglycaemia, hypothermia, micronutrient deficiency (vitamin A, zinc, copper, iron etc.). Marasmic children must be treated appropriately to prevent morbidity and mortality. Forty case of management of marasmus is in important cause of treatment failure and consequently case fatality. Marasmic children without complication can be managed by community based management. While marasmic with complication were death rate are very high should be managed preferably at facility treatment at felicity based management comprises 7-steps of inpatient care (stabilization phase) and after that child can be transfer to community based care. In community children are given therapeutic food and routine medicine to treat simple medical condition at an outpatient community based center. Appropriate case management of marasmus by standard protocolized management with or without complication can reduces case fatality and improves health status marasmic children.

INTRODUCTION

Marasmus is one form of sever protein energy malnutrition (PEM). It is an important cause of under 5 deaths in developing countries. In 2009, over 9 million children died of disease and malnutrition globally and most child death occurs in developing countries. Sub-Saharan African accounts for around 4.8 million of all child death, where around 3.1 million are in south Asia [1]. Despite different development program and priority in several healths' and other program malnutrition remains a perennial problem in many developing countries. Infection, diarrhoea, low birth weight (LBW) associated with intrauterine growth restriction (IUGR) are associated with marasmus. Although incidence and mortality of severe malnutrition which include marasmus is declining slowly still, it is a threat to child health and child survival. Most of the child mortality occurs in severe acute malnutrition (SAM) which includes marasmus if it is not address properly. Faulty case management and weak health system inappropriate treatment strategies inadequately trained staffs, lack of support lead to high case fatality of severe malnutrition (marasmus) associated with complication. The article will provide awareness and information about the characteristics features of marasmus, its aetiology, pathogenesis and complications and its appropriate management at facility and community level.

Marasmus is the most common form of severe malnutrition in nutritional emergencies. The word marasmus is derived from the Greek word marasmos, which means wasting. It is characterized by severe wasting of fat and muscle which the body breaks down to make energy. Affected children exhibit extreme wasting and have the appearance of old man appearance with just skin and bones visible. The body of wasted child tries to conserve energy by poor activity and reducing metabolic process. Initially they look alert and active and have good appetite but as the disease progresses they become irritable, less active and develop food refusal [2].

UNDERLYING DETERMINANTS OF MALNUTRITION (AETIOLOGY)

Aetiology of malnutrition is multifactorial. As shown in the Figure 1. The important immediate causes of marasmus are inadequate dietary intake (deprivation), disease, particularly infectious disease and low birth weight particularly IUGR. Under nutrition makes children vulnerable to disease particularly infectious diseases. On the other hand children are likely to develop under nutrition following suffering from diseases like pneumonia, diarrhoea, measles tuberculosis etc. There are two well known vicious cycles involving malnutrition→ infection

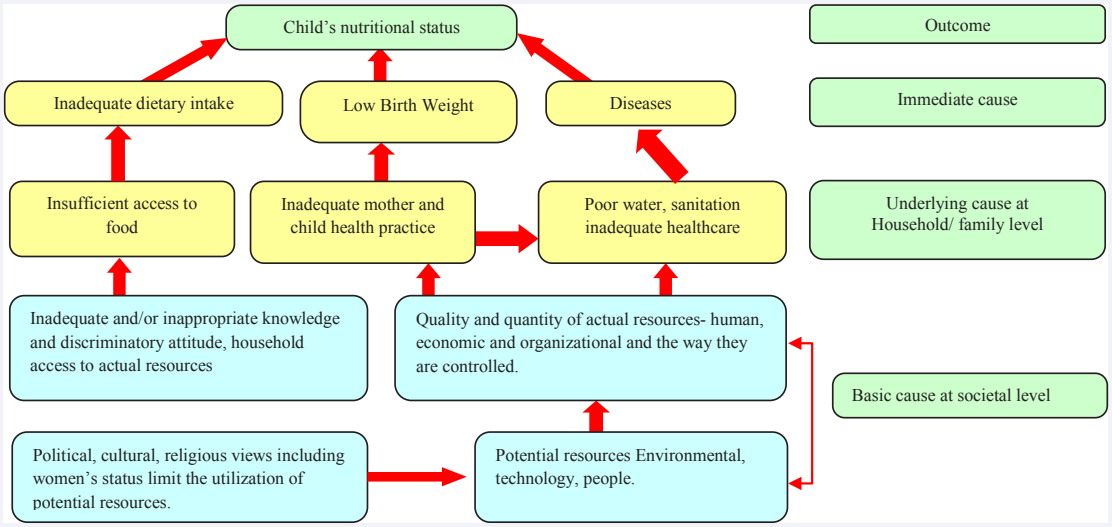

Figure 1 Aetiology of Malnutrition.

→malnutrition and malnutrition→ diarrhoea→ malnutrition. LBW associated IUGR may not show catch up growth and do not get weight properly and may remain under nourished. Nutrition sensitive factors for malnutrition are access to preventive and curative health service to all community members as well as hygienic and sanitary environment, an access to safe drinking water (Figure 1).

A grading of marasmus has been proposed as grade 1,loose skin fold in axilla and groin, Grade 2 loose skin fold in thigh, grade 3 wasting of chest and abdomen and grade 4 with wasting of buccal pad of fat giving the appearance of old man face. When marasmic children develop oedema it is termed as marasmic-kwashiorkor.

ASSESSMENT OF MALNUTRITION

Older classification depending on presence of under nutrition with oedema (Welcome Classification) is still used [3]. However to avoid confusion with clinical symptoms of kwashiorkor, which includes other features the term 'oedematous malnutrition' is preferred. According to Welcome classification a child is called marasmic if his/her weight is less than 60% of expected weight for his/her age (Table 1).

The Assessment of nutrients status is done according to weight for height or length (W/H), height (or length) for age (H/A) according to WHO classification as shown in Table-2 [4]. The WHO recommends (Table-II)the use of Z-scores or Standard Deviation (SD) Scores for evaluating anthropometric data, so as to accurately classify individuals with indices below extreme percentile. A Marasmic child usually falls under severe and moderate wasting.

Gomez classification (Table 3) [5] is done according to weight for age in comparison to his/her weight for age median. If weight for age of a two year age male child whose median(50^{th} centile) weight is 12 kg, is 10 kg (less than 90%) than it is called 1^{st}degree malnutrition. If his weight is less than 75% (between 60-74%) than it is called 2^{nd} degree malnutrition. If his weight is only 5 kg that is less than 60% of his weight for age median than it is called 3^{rd} degree Malnutrition. A Marasmic child usually falls under 3^{rd} degree malnutrition.

A child aged 6 -59 months is classified as severely malnourished if she/he has one or more of the following:

- Weight for height median (WHM) < 70%
- Weight for height Z score (WHZ) < - 3SD
- Mid upper arm circumference < 110 mm
- Bipedal oedema (Kwashiorkor, marasmic kwashiorkor or oedematous malnutrition)

A child< 6 months should be classified as severely malnourished if he/she has (i) Visible wasting, (ii) WHM < 70% or – 3SD (iii) Bipedal oedema

GOMEZ CLASSIFICATION (DEPENDING ON WEIGHT FOR AGE)

Indian Academy of Paediatrics (IAP) divides underweight children in five groups. Children below 50% weight for age are graded IV or very severely malnourished. According to Gomez classification (Table 3) children with weight for age less than 60% of median without oedema are usually marasmic.

Depending on duration severe malnutrition is divided into acute severe and chronic severe malnutrition. In acute malnutrition (non-oedematous) weight for age and weight for height are reduced significantly, while height is not affected significantly (acute marasmus). If malnutrition continues for a long time (chronic), height for age is also significantly decreased causing significant stunting (chronic marasmus).

Pathogenesis of Malnutrition

Mild to moderately malnourished children, which constitute great portion of malnourished children, if not managed properly at community level with adequate protein and energy dense diet, undergo severe under nutrition, initially in the form of

Table 1: Welcome classification of malnutrition.

Weight for Age	With Oedema	Without Oedema
60-80% expected weight for age	Kwashiorkor	Undernutrition
<60% expected weight for age	Marasmic kwashiorkor	Marasmus

Table 2: WHO Classification of Malnutrition.

	Under Nutrition				Over Nutrition	
	Severe Malnutrition	Moderate Malnutrition	Mild Malnutrition	Well Nourished	Over weight	Obese
Symmetrical oedema	Yes	No	No	No	No	No
Weight for Height (SD)	< -3 SD (<70%) severe wasting	-2 SD to -3 SD (70% -79%) Moderate Wasting	-1SD to - 2 SD (80%- 89%) Mild Wasting	+2SD to -1 SD (90%- 120%)	+2 SD to +2.9 SD (121%-129%) Over weight	>+3.0 SD (>130%) Obese
Height for Age	< - 3SD (<85%) Severely Stunted	-2 SD to -3 SD (85%-89%) Moderately stunted	-1 SD to -2 SD (90% - 94%) Mildly stunted	+2 SD to – 1 SD (95% -110%)	>+2 SD > (110%) Tall	

Table 3: Gomez classification of malnutrition.

Weight for Age (Median)	Nutritional Status
>90	Normal Nutritional status
75-89	1st Degree Malnutrition
60-74	2nd Degree Malnutrition
<60	3rd Degree Malnutrition

wasting (severe wasting or marasmus). Severely malnourished (marasmic or non oedematous) children, who are immune-compromised suffer from recurrent infections, both clinically and sub-clinically. In response to recurrent infection, liver produces increased active phase proteins in the form of increased C-reactive protein (CRP), α-1 acid protein, α-1 anti-trypsin, macro globulin etc. at the cost of producing albumin in liver. As serum plasma albumin is decreased, plasma colloidal oncotic pressure is decreased; consequently more fluid is lost from intra vascular to extra vascular space, giving rise to oedema and oedematous malnutrition [6].

However development of oedema does not depend solely on decreased serum albumin. In normal healthy children there is balance between oxidant and anti-oxidant. In malnourished children, there is oxidative stress and free radical induced damage of tissue and anti-oxidants like zinc, β-carotene, and tocopheroletc are decreased. Therefore net increase of oxidants cause ongoing oxidative stress induced tissue damage and tissue damage induced oedema. Through Fenton reaction and lipid per oxidation in the presence of free iron, cell membrane and tissue are chemically damaged. Consequently increased free radical induced tissue damage also contributes to tissue oedema.

Oedematous malnutrition is also associated with increased anti- diuretic hormone (ADH), which causes fluid retention which further aggravates oedema. Increased serum ferretin, associated with oedematous malnutrition, also act as ADH, which also contributes to oedema.

Serum protein takes long time to decrease in severe marasmus and is not sensitive and early indicators of protein energy malnutrition (PEM).

In severely malnourished children serum albumin is initially decreased due to decreased protein intake and serum globulins are increased (albumin-globulin ratio is altered) maintaining normal total serum protein. If malnutrition status continues for long time without dietary intervention, total serum protein is finally also decreased. Serum albumin is not the earliest biochemical indicator of protein energy malnutrition (PEM). Decreased serum transferrin, serum pre-albumin and serum retinol binding protein are sensitive and early indicators of severe protein energy malnutrition which includes marasmus.

Table 4: Status of Hormones and biochemical indices in severe Protein energy Malnutrition [6].

Parameter Increased	Parameter Decreased
Serum Growth hormone	Somatostatin
Serum Cortisol	IGF 1 and IGF 2
Serum ADH	Serum Insulin
CRP	Serum T4 and TSH
α 1 anti-trypsin	Serum Albumin and pre-albumin
α 1 acid protein	Serum retinol binding protein (RBP)
Serum Ferretin	Serum Transferrin
Serum Globulin	

Table 5: Pathological Changes in Malnutrition.

Upper G.I.T.	• Mucosa flat atrophy
Small Intestine	• Mucosal and villous atrophy
Pancreas	• Exocrine secretion depressed • Endocrine less affected
Lympho-reticular System	• Thymus atrophied. • Lymphocyte proliferation depressed
C.N.S	• Head circumference and brain growth retarded
C.V.S.	• Myocardial function decreases due to myocardial structural changes • Degeneration of myocardial cells with formation of aschoff cell.

Both blood urea and serum Transferrin in PEM are increased early (in terms of days) with increased dietary intake of protein enriched diet in malnourished children. Serum total protein and albumin take long time (in terms of weeks) to increase in response to dietary intervention. Therefore serum transferrin estimation rather than serum albumin is an early indicator of dietary response of protein intake in malnourished children.

Serum creatinine and urine creatinine in particular, indicate endogenous protein (skeletal muscle and white muscle fibres) contents.

In severe malnutrition urinary creatinin height indices (CHI) is decreased as follows:

CHI =	24 hour urine creatinine
	24 hour urine creatinine of normal child with same height

Range 0.25-0.75 in kwashiorkor and 0.33-0.85 in marasmus, recovered value is 1.

Various hormonal and biochemical changes occur in severe malnutrition (marasmus/marasmic-kwashiorkor) some are increased and some are decreased. Some important hormonal and biochemical changes are given in Table (6) [6].

Significant pathological changes occurs in various system of the body. Some important pathological Changes of clinical significance are mentioned in Table-V. Some important pathological changes in various system in severely malnourished(marasmic) children are given in Figure (2-4) [7].

Clinical Significance of Pathological Changes

Mucosal and villous atrophy (Figure 2) is an important cause of frequent and persistent diarrhoea and mal absorption in severely mal nourished (marasmic) children. This is also the basis of offering initial low volume and low calorie (F-75) diet to severely malnourished children instead of initial high calorie (F-100) diet and high protein diet. Mucosal atrophy is more pronounced in oedematous malnutrition. This is one of the basis of offering low volume diet (9 ml/kg/feed) in oedematous malnutrition (marasmic-kwashiorkor and kwashiorkor) in comparison to non-oedematous (11 ml/kg/feed) marasmic children.

Heart muscle in malnourished children also undergoes degeneration and atrophy (Figure 3), therefore heart muscle cannot cope with initial sudden increase in protein, calorie and fluid intake.

Decreased pancreatic exocrine secretion contributes to malabsorption and food intolerance in malnourished children.

Thymic atrophy: Evidence of lympho-reticular depletion and impaired cell mediated immunity (CMI) in severe malnutrition (marasmic) can be seen on X-ray chest. Normally infants have large corrugated margined thymic shadow. In malnourished children, due to thymic involution thymic shadow cannot be seen and X-ray chest shows only narrow stalk of superior mediastinum instead of normal prominent thymic shadow of healthy infant (Figure 4).

Due to qualitative and quantitive reduction of T lymphocytes, cell mediated immunity (CMI) is impaired. This is clinically evidenced by anergy to tuberculin test, in spite of presence of active tuberculosis in children. More scientifically it can be tested by impaired CMI with candida albican antigen test which will be similarly non-reactive.

C.N.S.: Head circumference diminishes in severe PEM. CT scan/MRI of brain shows atrophy. There are also dendritic arborization defect in brain. Consequently children may have

Table 6: Difference between oedematous and non-oedematous malnutrition.

Clinical features	Non-oedematous (Marasmus)	Oedematous (Kwashiorkor)
Oedema	Absent	Present
Age of onset	Usually below1 year	Usually after 1 year
Occurrence	More common	Less common
Activity	Active	Apathetic
Appetite	Good	Poor
Hepatomegaly	±	+
Initial Therapeutic food	More11ml/kg/feed 2 hourly or130ml/kg/day	Less8ml/kg/feed 2 hourly or80-100ml/kg/day
Dermatosis	±	+
Infection	Less prone	More prone
Recovery	Recover early	Long to recover
Mortality	Less	More

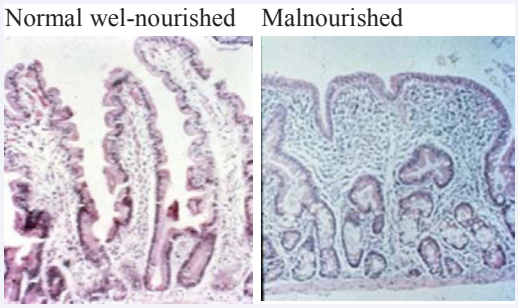

Figure 2 Histology of normal villous of gut mucosa epithelium (left) and submucous villous atrophy in malnourished child (right).

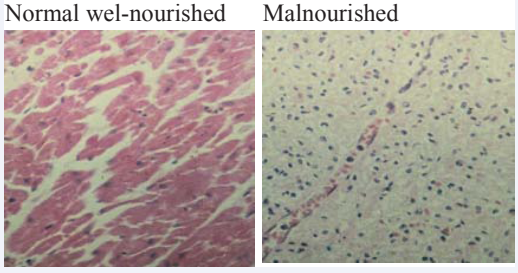

Figure 3 Histological slide of heart muscle showing normal heart muscle cells in well nourished child (left) and degenerated cells showing Aschoff cells in malnourished child (right).

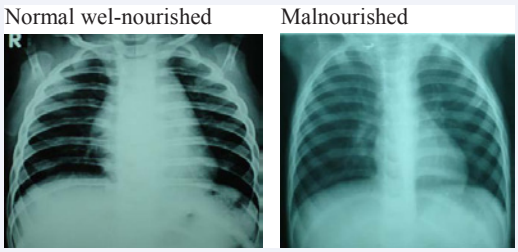

Figure 4 Chest x-ray of normal we nourished child (left) showing prominent thymicshadow, and atrophic thymic shadow (right) in malnourished child evidenced by narrow superior mediastinum.

developmental, learning and cognition problems when they are grownup.

Clinical features of Marasmus

Depending upon severity, duration of PEM presence of oedema in marasmic child, dermatoses and associated other nutritional deficiencies and complications associated with malnutrition, the presentation will be different

Clinical Features of malnutrition in Children

In severe acute under nutrition weight for Age and weight for height are significantly reduced, but height for age usually remains normal, as linear growth retardation or stunting due to under nutrition takes longer time to affect linear growth adversely. If acute malnutrition is not managed appropriately and under nutrition is prolonged, height for age eventually significantly reduced, and the child becomes both wasted and stunted (Figure 5). When a marasmic child develops edema it is called marasmic-kwashiorkor (Figure 6 showing wasted child with ankle edema).

Dermatoses of malnutrition include hypopigmentation or hyperpigmentation, desquamation, ulceration, spreading over limbs, thigh, groin and genitalia.

The skin lesions may look like mosaic of floor called crazy pavydermatosis or pigmented lesion may become confluent followed by flaking of skin called flaky paint dermatosis (Figure 7). Flaky paint dermatosis is more common features osedematous malnutrition.

Severe dermatoses which include ulcer, fissures and exudative lesion may resemble burn.

All Dermatoses may be complicated with secondary infection including candida.

Hair may be depigmented, lustreless. A flag sign is the alternate bands of hypo and normally pigmented bands of hair, when growth occurs in spurts. A severely malnourished child quite often presents with diarrhoea, dehydration, pneumonia with respiratory distress, septicaemia, loss of appetite, vomiting etc. and will require facility based treatment.

Difference between oedematous (kwashiorkor and marasmic kwashiorkor) and non-oedematous (Marasmus) malnutrition

The characteristic difference between oedematous and non-oedematous malnutrition are shown in the Table-VI. However such clinical features are not always consistent distinguishing features between oedematous and non-oedematous malnutrition. For example, liver enlargement may occur in marasmus and may be absent in kwashiorkor. A marasmic child may be more anorexic, while kwashiorkor child appetite may be reasonably preserved. Oedematous malnutrition develops usually in the second year of life usually after development of wasting (marasmus).As mentioned earlier marasmus may be acute staying for short duration and improved by clinical intervention. In acute marasmus the child is wasted and both weight for age and weight for height are significantly decreased but height for age is usually normal. However if it becomes chronic without receiving intervention height is also affected and height for age is also decreased and the child becomes both wasted and stunted. There is no edema in marasmus. Similarly edematous malnutrition (marasmic-kwashiorkor/kwashiorkor) may be acute or chronic and their anthropometric status will also be different. Dermatoses are more associated with edematous malnutrition. Anthropometric and clinical features of acute and chronic severe malnutrion (marasmus, kwashiorkor o are mentioned in Table (6).

MANAGEMENT OF SEVERE ACUTE MALNUTRITION (SAM)

Severe acute malnutrition, particularly associated with complications is an important cause of under five mortality in developing countries. Faulty case management and weak

Figure 5 Child with wasting and stunting are compared with normal one.

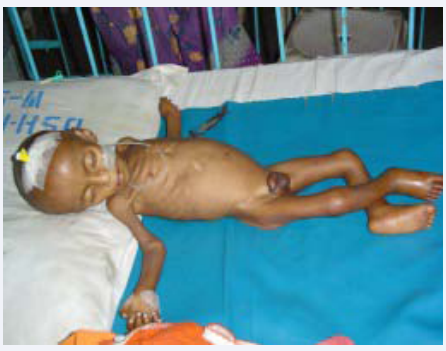

Figure 6 Wasting & edema.

health system, inappropriate treatment strategies, inadequately trained staffs, lack of support lead to high case fatality of severe malnutrition [8,9].

In order to maximize coverage and access to therapeutic care for severely malnourished children, an approach that combines the following components is most appropriate which include-

- Management of SAM with complication at facility level
- Management of SAM without complication at community level.

Underlying conditions and management strategy of SAM and chronic malnutrition

Severe acute malnutrition (SAM) characterized by severe wasting is an unstable condition resulting from absolutely short duration of nutritional deficit that is often complicated by concurrent infective illness. The child with SAM has a limited ability to respond to stresses (infectious and environmental), is highly vulnerable to infectious disease and has a high mortality rates. It is thus vital to treat SAM proactively with short duration, highly intensive treatment regimen, aiming to rehabilitate the child in a few weeks. By contrast chronic malnutrition characterized by significant stunting is relatively stable condition is the result of slow progression of prolonged episodes of under nutrition, both of the pregnant mother and of the young infant, importantly during the first two years of life. Management of Severe Acute Malnutrition (SAM) in Facility based inpatient care [10].

GENERAL PRINCIPLE FOR ROUTINE CARE

The general principles of management of SAM are derived on the basis of following physiological and metabolic conditions of severely malnourished children.

- **Reductive adaptation**

This is physiological and metabolic slow down in order to preserve energy. This mechanism is relevant to hypothermia, hypoglycaemia and initial low calorie therapeutic food in malnourished children.

- **Not to use diuretics for oedema**

Use of diuretics may aggravate already existing intravascular hypovolemia, leading to hypovolemic shock. Diuretics may cause further potassium loss of potassium depleted malnourished children.

- **Avoid initial intake of high protein and high calorie diet**

Death may occur if high protein and high calorie diet is given early, as due to reductive adaptation theory, cardiovascular and gastrointestinal system cannot cope with sudden increase in proteins, calorie and fluid intake. Heart muscle (Figure 3) of malnourished children undergoes degeneration and GI system is associated with subtotal villous atrophy (Figure 2). The malnourished children may develop heart failure, loose motion and vomiting from food intolerance and malabsorption, with increased case fatality, associated with initial high calorie and high protein therapeutic food.

- **Minimum use of IV fluid**

Intravenous fluid may be administered only for 2 hours and slowly in shock only. IV fluid may cause heart failure due to fluid overload in severely malnourished children.

- **Low sodium and high Potassium containing fluid**

In spite of low serum sodium in malnourished children, there is sodium retention and decreased tissue potassium which is not always reflected in serum electrolytes level. This is the basis of providing half strength polyelectrolyte solution (cholera saline) to malnourished children with diarrhoea and shock. Oral rehydration saline for malnourished children (ReSoMal) similarly contains low sodium and high potassium.

- **Frequent feeding**

Due to reductive adaptation and loss of appetite, malnourished children are vulnerable to hypoglycaemia. This is the basis of 2 hourly feeding, including feeding at night for PEM children. Similarly ReOsMal also contain high sugar than previous conventional ORS.

- **Give antibiotics empirically [11]**

Since the severely malnourished children are immune compromised, they are prone to develop intercurrent infection, some of which are clinically obvious like pneumonia, gastroenteritis etc. for which admission in facility based care are sought. However, some are not clinically obvious. Since infections in malnourished children are catastrophic, SAM children are given routine broad spectrum antibiotics on admission.

- **Iron should not to be given immediately**

Iron is less utilized in malnourished children for synthesis of haemoglobin. The free unused iron acts as a free radical which promotes bacterial growth and oxidative tissue damage. Iron in malnourished children is converted to ferritin, which acts as anti diuretic hormone and helps in developing oedema. There is evidence to suggest that increased ferritin is associated with increase case fatality in malnourished children.

THE 10 STEPS IN THE MANAGEMENT OF CHILDREN WITH SAM WHICH INCLUDES MARASMUS [10]

These 10 steps are divided into 2 parts-

- Initial Stabilization phase (containing 7 steps)
- Rehabilitation phase (containing 3 steps)

A. Initial Stabilization phase

There are 7 steps in initial stabilization phase, where life threatening conditions are identified and treated and specific deficiency is corrected. Usually achieved in 1st week. Seven steps are the following:

Step 1	Treat/ prevent Hypoglycaemia
Step 2	Treat/ prevent Hypothermia
Step 3	Treat/ prevent Dehydration
Step 4	Correct electrolyte imbalance

Step 5	Treat/ prevent Infection
Step 6	Correct Micronutrient deficiency
Step 7	Start cautious feeding including breast feeding

B. Rehabilitation Phase

Usually achieved in 2-6 weeks. Three stages of rehabilitation are following:

Step 8	Achieve catch up growth
Step 9	Provide sensory stimulation and emotional support
Step 10	Prepare for discharge and follow up regularly

Steps of Management

A. Initial Stabilization Phase

Step 1	**Hypoglycaemia**

- **Blood glucose:**

< 3 mmol/L or 54 mg/dl

- **Clinical features:**

Lethargy, hypothermia, altered level of consciousness

- **Management:**

○ **If the baby is conscious**

▪ 50 ml of 10% Glucose orally or by NG tube

▪ F-75 Diet half hourly for 2 hours (giving one-quarter of 2 hourly feed)

▪ Keeping the child warm

▪ Antibiotics

▪ 2 hourly feed, day and night.

○ **If the child is unconscious**

▪ I.V. 10% glucose (10 ml/kg) followed by 10% glucose 50ml by NG tube, followed by F-75 diet as mentioned above.

Blood sugar should be carefully monitored. If blood sugar is persistently <3 mmol/L, in spite of above management more severe underlying cause including septicaemia should be considered and appropriate management and if possible referral to a higher facility should be done.

Step 2	**Hypothermia**

- Rectal temperature <35.5˚C, axillary < 35˚C
- Co-exists with hypothermia and sepsis
- Re-warm the child

○ Cover the child with warm blanket and increase the ambient temperature with safe heat source, or put the baby on mother's bare chest (skin to skin) and cover them, the Kangaroo mother care.

○ Start antibiotics and 2 hourly feed.

Step 3	**Diarrhoea and Dehydration**

It is difficult to estimate dehydration in severely malnourished children as positive skin pinch sign and sunken eye may occur due to loss of fat and muscle wasting without dehydration. Similarly dehydration may be over estimated in oedematous malnutrition. Therefore it is assumed that malnourished children with diarrhoea have dehydration. Clinical assessment of dehydration in severe PEM are shown in Figure (8,9).

Dehydration correction oral solution for malnourished children called ReSoMal contains low sodium and high potassium [12]. Correction of dehydration is done in the following manner [13]:

- Give ReSoMal 5ml/kg every 30 minutes for 2 hours. Then 5-10 ml/kg/hour every alternate hour for 4-6 hours.
- F- 75 in alternate hour
- If diarrhoea is severe modified/ Hypo-osmolar WHO ORS, containing more sodium (75mmol sodium/L) than

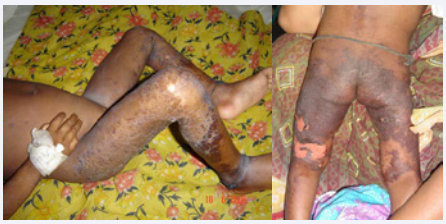

Figure 7 Picture showing Flaky paint dermatoses.

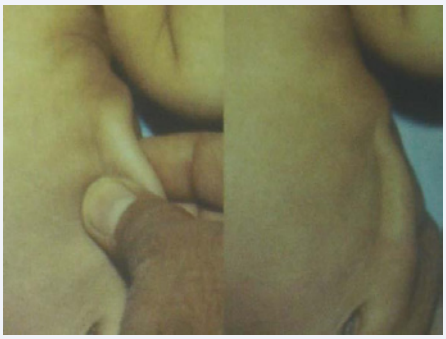

Figure 8 Technique of skin (full thickness) pinch to assess dehydration in malnutrition.

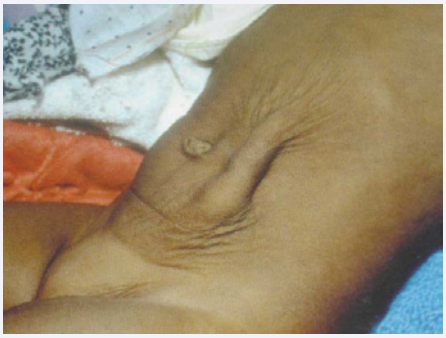

Figure 9 Skin pinch (full thickness) goes back very slowly in a malnourished child with diarrhoea.

of ReSoMal (sodium 45mmol/L) may be used to prevent symptomatic hyponatremia.

Signs of over hydration (due to injudicious use of fluid resuscitation)

- Increasing pulse rate
- Increase respiratory rate
- Oedema (puffy face)
- Distended neck veins.

A positive skin pinch sign may be found in malnourished child due to loss of subcutaneous fat, even without dehydration. So a full thickness skin pinch (Figure 8,9) is required to assess dehydration.

Step 4	Correct Electrolyte Imbalance

SAM children have excess body sodium with low tissue potassium and magnesium. Serum electrolytes do not reliably reflect tissue electrolytes contents rather it may act as supporting role of clinical dyselectrolytemia.

Treatment

Until stabilization introduce-

- Extra potassium 3- 4 mmol/kg/day
- Extra magnesium 0.4 – 0.6 mmol/kg/day

When rehydrating, give low sodium rehydration fluid (ReSoMal)

- Prepare food with less salt
- Do not treat oedema with diuretics

Symptomatic tissue potassium deficiency may be associated with ileus and abdominal distension, which aggravates feeding difficulty. Intra muscular magnesium injection help improving body potassium utilization, thereby improving abdominal distension and feeding difficulty. Injection 50% magnesium sulphate 0.3ml/kg IM should be given on 1st day and 0.1 ml/kg on 2nd and 3rd day.

Step 5	Treat and prevent infection

Signs of infection such as fever are absent and subacute bacterial infections are common in malnourished children which may be asymptomatic. Therefore routine broad spectrum antibiotics are used on admission [10,11].

Early use of antibiotics also-

- Improves nutritional response to feeding
- Prevents shock
- Reduce mortality

I. First line treatment

With no complication (WHO guide line) oral Amoxycillin 15mg/kg 8 hourly for 5 days.

If the child is severely sick with lethargy or complication IV/IM Ampicillin (50 mg/kg) 6 hourly for 2 days, then oral Amoxycillin 15mg/kg 8 hourly for 5 days and IV/IM Gentamicin 7-8mg/kg once daily for 7 days.

II. Second line treatment

If the child doesn't improve with first line of treatment within 48 hours or deteriorates after 24 hours or if the child present with septic shock or meningitis then infuse IV/IM Inj. Ceftriaxone 100mg/kg/day with gentamicin for 5 days.

Micronutrient deficiency

Avitaminosis causing angular stomatitis frequently found in marasmus (Figure 10) similarly xerophthermia due to vitamin A deficiency is also associated with PEM (blinding malnutrition) (Figure 11).

Step 6	Correct Micronutrient deficiency

Vitamin A orally on D1,D_2,D_{14} (If not received within one month) in following dose according to age	
< 6 months of age	50,000 IU
6 – 12 months of age	1,00,000 IU

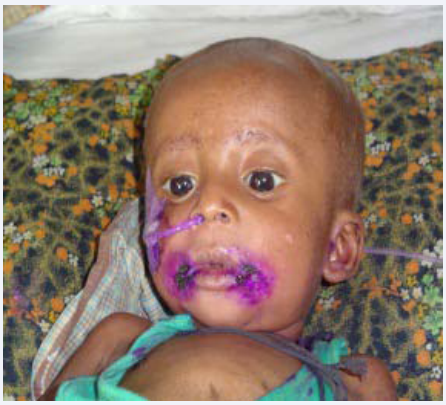

Figure 10 Evidence of avitaminosis (angular stomatitis) in severely malnourished child contributing feeding difficulty. Discoloration of lip due to zention violate application.

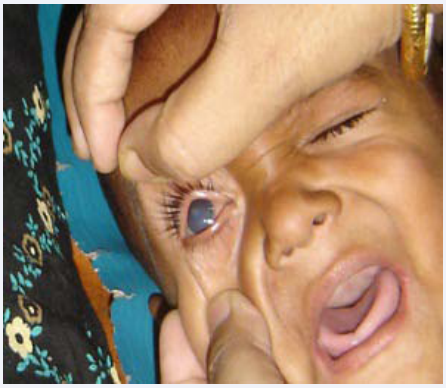

Figure 11 Eye sign (corneal opacity) of vitamin a deficiency in a malnourished child.

>12 months of age	2,00,000 IU
Give daily at least for 2 weeks	
• Multi vitamin supplement (without iron)	
Folic Acid- Give 5mg on day one	1 mg/kg/day
• Zinc	2 mg/kg/day
• Copper	0.3 mg/kg/day
• Iron	3mg/kg/day
But only given when child is gaining weight (start at rehabilitation phase)	

Calcium is also added in therapeutic food. Although calcium and vitamin D deficiency are also associated with severe malnutrition, clinical rickets is very unusual. This is because rickets occurs due to mineral deficiency in growing bone. Calcium and vitamin D supplement are essential during rehabilitation phase of malnutrition when the child grows fast.

WHO recommended therapeutic diets-

- F- 75 (100 ml containing 0.9 gm protein and 75 Kcal energy)
- F- 100 (100 ml containing 2.9 gm protein and 100 Kcal energy)
- Prepared from milk powder, sugar, soybean oil
- Combined minerals and vitamins (CMV if available) or electrolytes/mineral solution if CMV not available commercially.

Frequency of feeding during the acute phase

- Start with therapeutic feed every 2 hours (12 feeds in 24 hour)
- Night feeds are extremely important.

Step 7	**Start cautious feeding including breast feeding**

Start feeding with F-75 containing 75 Kcal and 0.9 gm protein/100ml feed from cup/spoon/syringe.

The following is the usual recommended schedule:

Days	**Frequency**	**Vol/kg/feed**	**Vol/kg/day**
1-2	2 hourly	11 ml	130 ml
3-7	3 hourly	16 ml	130 ml
8+	4 hourly	22 ml	130 ml

For children with severe oedema, the volume/feed and volume/day (100ml/kg/day) are reduced until oedema disappears. If intake is <80 Kcal/kg/day, give remaining by N

Breast feeding

- Breast feeding is encouraged in between feeds
- Required amounts of therapeutic diet are ensured even if the child is breast fed, that is required amount of F-75/ F-100 diet are not curtailed if the child breast feeds.

TREATMENT OF ASSOCIATED CONDITIONS

1. Emergency management of shock

Severe dehydration / dehydration shock and septic shock are difficult to differentiate on clinical signs alone. Signs of septic shock may include

a. Signs of shock but without history of watery diarrhoea. Don't drink eagerly like severe dehydration.

b. Hypothermia or hypoglycaemia

2. Diagnosis of shock is based on following criteria

a. Lethargy and unconscious and

b. Cold clammy hand, feet and plus either

I. Slow capillary refill time (>3 sec) or

II. Weak fast pulse (>160/min in 2 -12 months of age, >140/ min in 1 - 5 years of age).

3. Treatment of Shock

5 important components-

a. Give oxygen

b. Give sterile 10% glucose (5ml/kg) IV route

c. Keep the child warm

d. Give an antibiotic

e. Give IV fluid at 15ml/kg over 1 hour. Use Ringer's lactate with 5%dextrose or half strength normal saline with 5% dextrose.

f. Measure and record pulse and respiration rate every 30 min.

If the shock is due to severe diarrhoea

Use half strength cholera saline (15ml/kg for first 2 hours to prevent symptomatic hyponatremia)

If there are signs of improvement after 1 hour (pulse and respiratory rate decreasing)

a. Repeat IV fluid 15ml/kg for one hour (total 2 hour).

b. Switch to oral or NG rehydration with ReSoMal 10ml/ kg/hr in alternate hours with F- 75 diet.

c. Continue feeding with F- 75 diet

If the child fails to improve (pulse and respiratory rate remains high) after 1 hour assume septic shock:

In this case

a. Give maintenance IV fluid (3ml/kg/hour) while waiting for blood.

b. Transfuse whole blood at 10ml/kg slowly over 3 hours

c. Stop infusion if signs of over hydration appears (pulse suddenly increases by >25/min or resp. rate increases by >5/min from existing condition).

4. Anaemia in malnourished children

Anaemia, particularly iron deficiency is commonly associated with severe PEM. In majority of cases, normocytic normochromic anaemia is common. However associated vitamin and mineral

Table 7: Clinical features of under nutrition in children.

	Oedema	Wasting	Dermatosis	Reduced Weight for age	Reduced Height for age	Reduced Weight for height
Severe Acute Malnutrition (SAM)						
Marasmus (acute)	0	++	0	++	0	++
Kwashiorkor (acute)	++	0	±	±	0	±
Severe Chronic Malnutrition (SCM)						
Marasmus (chronic) (Chronic wasting and stunting)	0	++	0	+++	++	+++
Kwashiorkor (Chronic)	++	+	±	++	++	++
Chronic Mild Malnutrition (Nutritional dwarfism)	0	0	0	++	++	±

Table 8: Composition of oral rehydration salts solution for severely malnourished children (ReSoMal) showing low sodium and high glucose and potassium content.

Component	Concentration (mmol/l)
Glucose	125
Sodium	45
Potassium	40
Chloride	70
Citrate	7
Magnesium	3
Zinc	0.3
Cooper	0.45
Osmolarity	300

deficiencies, including iron deficiency and ongoing sepsis may modify the picture. The normocytic normochromic anaemia in severe PEM is associated with decrease in circulatory erythrocytic mass [14]. The metabolic changes in red blood cell, decrease in erythrocytes, and fall in erythropoietin production cause erythroid hypoplasia with increase in myeloid/ erythroid ratio. However associated iron deficiency, may cause iron deficiency anaemia. The incidence of iron deficiency anaemia is variable and depends on number of factors such as dietary habits, parasitic infestation, chronic blood loss etc.

Although majority of hypochromic microcytic anaemia are due to iron deficiency but other conditions like ongoing infection is frequently associated with PEM. Infection decreases haemoglobin synthesis and iron is less utilized for haemoglobin synthesis and it is eliminated rapidly from blood to reticulo-endothelial system in the form of ferritin. Characteristically in iron deficiency anaemia serum iron is decreased with increase of total iron binding capacity (TIBC) and decrease in serum ferritin. However in anaemia with severe PEM, serum irons though less, TIBC is also less and serum ferritin may be increased.

Not only iron is unutilized in PEM, the unaltered ferrous ion catalyzes the reaction of superoxide and hydrogen peroxides (H_2O_2) to produce highly reactive hydroxyl (OH) ion through Fenton reaction, which is capable of producing chemical injury to cell membrane. Lipid per oxidation has been proposed as the primary mechanism for cellular dysfunction and tissue injury. In malnourished children, oxidative process overwhelms the anti-oxidant protection. These facts are the basis of withholding of iron supplementation in the early phase of management of severely malnourished children. In mild to moderate anaemia, iron should be given for 3 months to replace the iron store, but this should not be started until after the initial stabilization phase has been completed.

EMERGENCY TREATMENT OF SEVERE ANAEMIA

A Blood transfusion is required

- If haemoglobin is <5 gm/dl
- If haemoglobin is between 5 -7 gm/dl with respiratory distress.

Transfuse

- Whole blood 10 ml/kg slowly over 3 hours
- Furosemide 1 mg/kg at the start of transfusion

If signs of cardiac failure appear, transfuse

Packed cell 5-7ml/kg body weight rather than whole blood

5. Vitamin – A deficiency

Vitamin A on day 1, 2 and 14

If there is corneal clouding or ulceration

a) Chloramphenicol or tetracycline eye drop

b) Atropine eye drop

c) Cover with eye pads soaked in saline solution and bandage

6. Dermatoses

a) Apply gauze soaked in 1% potassium permanganate solution over affected area and keep it for 10 minutes twice daily.

b) Omit nappies so that perineum can remain dry

c) Zinc oxide paste/ointment (Figure 12)

d) Antifungal (Clotrimazole) twice daily for candidiasis, oral nystatin (1, 00,000 IU), 4 times daily for oral candidiasis, which also acts as reservoir for gut and skin candidiasis.

7. Helminthiasis

Helminthiasis is frequently associated with malnourished children. Anthelminthic given during rehabilitation phase.

a) Single dose of 200mg of Albendazole if age is >3- 23 months, 400mg of age is >24 months

b) 100 mg of Mebendazole twice daily for 3 days for children > 24 months.

For Giardiasis

Metronidazole

(7.5 mg/kg, 8 hourly for 7 days.)

8. Tuberculosis

If tuberculosis is suspected due to contact with adult TB patient, chronic cough (> 2 weeks), chest infection not responding to conventional antibiotics, perform a Montaux test. In malnourished children the interpretation of Montaux test is made with caution. It may be false negative or mildly positive (if induration <5mm) in spite of presence of active tuberculosis due to impaired cell mediated immunity.

Treatment with evidence of TB, should be given accordingly to National TB Guideline of Bangladesh.

9. Continuing diarrhoea and dysentery

Loose or poorly formed stool are frequently associated with malnourished children, particularly in rehabilitation phase requiring no treatment provided the child is not sick and weight gain is satisfactory. Similarly food intolerance like lactose intolerance, milk protein allergies etc. are frequently over diagnosed in malnourished children. Rarely diarrheoa is due to lactose intolerance. Treat only if continuing diarrhoea is preventing general improvement. In that case substitute normal milk with non-milk formula (Rice suji, comminuted chicken soup) [see annex]

Osmotic diarrhoea: Some malnourished children cannot tolerate high osmolar diet during rehabilitation phase (F-100). In that case low osmolar cereal based F-75 diet should be continued for long time and F-100 should be gradually introduced.

Persistent Diarrhoea: Diarrhoea associated with severe malnutrition is a special entity and require special approach. Persistent diarrhoeacommonly associated with severely malnourished children not only responsible for treatment failure with poor weight gain, but also associated with high mortality. It should be treated with easily digestible protein and energy rich diet (Rice suzy, comminuted chicken soup, elemental and pre-elemental diet etc.), together with appropriate micronutrients (Zinc, vitamin A, copper, potassium) and appropriate antibiotic when required.

10. HIV/ AIDS

It may hinder recovery and may be associated with food intolerance (like lactose intolerance) and persistent diarrhoea. Lactose free diet may be tried.

11. Pneumonia

Pneumonia is one of the most frequent medical complications contributing to increased case fatality. Characteristic clinical features of pneumonia (tachypnoea, lower chest in drawing, cough) may not be evident due to poor host response associated with poor intercostal, subcostal and diaphragmatic muscle mass. For a given sensitivity and specificity they produce 5 breaths fewer respiratory rate than well nourished children. A number of children do not have fever and X-ray chest finding may not be conclusive. Therefore high index of clinical suspicion of pneumonia should be adopted with mild cough and suboptimal tachypnoea and even when characteristic WHO defined features of pneumonia are absent. Failure to recognize the above facts may cause failure to diagnose pneumonia early in malnourished children and also can delay timely treatment for pneumonia which may be potentially catastrophic.

C. Rehabilitation Phase

Step 8	Achieve catch-up growth

Signs of entrance to the rehabilitation phase are return of appetite and loss of oedema in oedematous ma

It should be gradual and take usually 1 week.

Recommended food: F -100 (every 100ml containing 100 Kcal energy and 2.9 gram of protein)

To change from Starter (F- 75) to Catch –up formula-

a) Replace F- 75 with same amount of F -100 every 4 hours for 48 hours

b) Increase each successive feed by 10ml until some feed remains uneaten

c) The point when some remain unconsumed after most feeds is likely to occur when intake reach about 30ml/kg/feed (200ml/kg/day)

d) In place of F-100 diet, non-milk formula like khichuri, halua, modified porridge or modified family food can be used, provided they have comparable energy, protein and micronutrient concentration.

Step 9	Sensory stimulation and emotional support

Malnourished children suffers from psychosocial deprivation

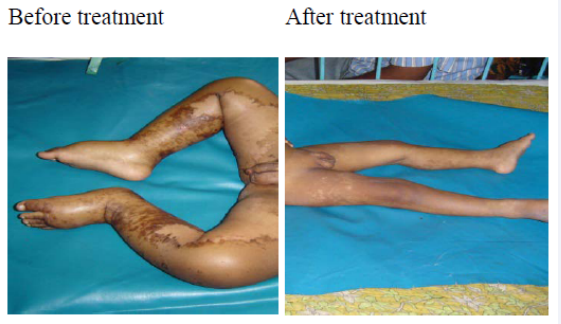

Figure 12 Disappearance of both oedema and dermatoses in malnutrition after protocolised management of SAM including skin treatment with zinc oxide and potassium permanganate.

are requires emotional support [15].

Step 9 Provide

a) Tender loving care

b) A cheerful, stimulatory environment

c) Toys made of locally available discarded materials

d) Physical activity as soon as the child is well enough

e) Parental involvement when possible, comforting, bathing, play and to be continue

Step 10	Prepare for discharge and follow up regularly

Criteria for discharge from inpatient care in areas where there is no community based outpatient care:

Child factor:

- WHM >80% or >WHZ > - 2 SD
- Oedema has resolved
- Good appetite and gaining weight
- Child has been provided with appropriate micronutrients

Mother factor:

- Mother can prepare appropriate food and feed for child
- Has financial resource to feed the child
- Can recognize danger sign and early access to hospital for urgent re-admission
- Can be visited weekly

Failure to respond to treatment

Indicators:

a. High mortality

b. Poor weight gain

A. High Mortality

Unacceptable	>20%
Poor	11 – 20%
Moderate	5 – 10%
Good	<5%

Death occurring within 24 hours of admission:

Consider untreated or delayed treatment of sepsis, pneumonia, severe anaemia, hypothermia, incorrect rehydration fluid, over use of IV fluids.

Within 72 hours:

Low volumes to high volume feed or feeding with wrong formula

Death Occurring at night:	Hypothermia • Not covered with blanket • draught coming from nearby window/ door • No night feed.

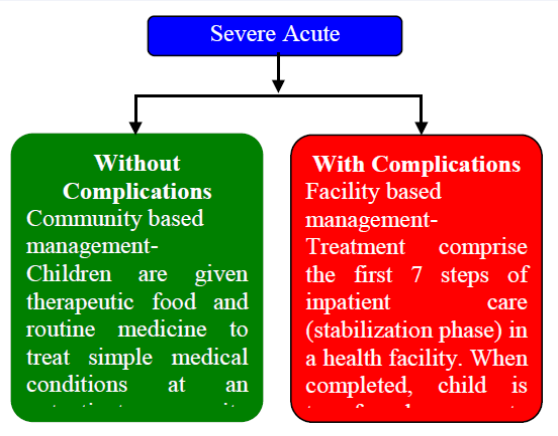

Figure 13 Management of SAM with or without complication.

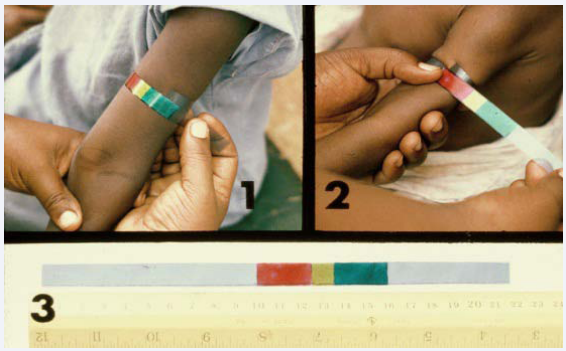

Figure 14 Assessment of malnutrition in community using MUAC. Well nourished child's MUAC falls in green zone (1), where as malnourished child's MUAC in red (<110mm) zone (2), Measurement scale (3).

Death during rehabilitation:	Too rapid treatment with F -100 diet
After 7 days	Consider hospital acquired sepsis. Persistent diarrhoea among malnourished children is associated with high mortality

C. Poor Weight gain during rehabilitation phase:

Consider weight gain as-

Poor	<5 gm/kg/day
Moderate	5- 10 gm/kg/day
Good	>10 gm/kg/day

If weight gain is poor, then major changes of management and overhauling of department of nutrition will be necessary.

Undiagnosed infections (TB, asymptomatic UTI) may also be considered.

Other factors involved in poor weight gain are:

a) Inadequate feeding, particularly night feed, wrong feeding technique and wrong preparation of food.

b) Specific nutrient deficiency, particularly not providing zinc and potassium to diet. Zinc and potassium

particularly required during catch-up growth, as growing muscles require zinc and potassium

c) Psychological problems and psychosocial problem are frequently associated with malnourished children. They are quite often emotionally deprived due to dysfunctional family unit and functionally single parent family. The psychological problems are characterized by stereotyped movements, rocking, rumination etc.

Treat by providing extra care, love and attention.

ROLE OF COMMUNITY BASED MANAGEMENT OF SEVERE MALNUTRITION

Where sufficient resources are made available, the WHO inpatient medicalized treatment model for SAM can achieve low case fatality rate (CFRs). However, exclusive inpatient treatment strategies are resource intensive, requiring large number of skilled staff. As the prevalence of SAM is highest in resource poor environment, there is usually substantial mismatch between the large numbers of patients requiring treatment and small number of skilled staff and limited resources available to treat them. The HIV/AIDS has further aggravated in sub Saharan Africa [16,17].

Community based management (CBM) compliments the existing WHO inpatient protocol.

A growing number of countries and international relief agencies have adopted a community based model for the management of acute malnutrition called community based therapeutic care (CTC).

The WHO also recommends treatment of uncomplicated SAM at community level. The model provides a framework for an integrated public health response to acute malnutrition, treating most patients with SAM solely as outpatient and reserving inpatient care for the few with SAM associated with complications. The model also aims to integrate treatment with various other interventions designed to reduce the incidence of malnutrition and improve public health and food security. Programme designed attempts to take into account the socioeconomic factors particularly poverty, high workload of women and factors that contribute to late presentation of cases of SAM. The design minimises the cost of families and maximizes access to treatment. The decentralised design also means that in non-emergency situation, there are few cases of SAM at any one access point and the quantities of ready to use therapeutic food required to treat SAM are therefore small.

SAM is classified on the basis of whether there is co-existent life threatening complications. Children presenting with SAM complicated by life threatening illness receive inpatient care according to WHO treatment protocol. Those with SAM but without life threatening complications are treated through weekly or fortnightly therapeutic programme.

The key components of community based management are

a) The introduction of technique to engage community to promote early presentation and compliance

b) Handing over the identification of SAM to community through use of MUAC (Fig-14) and

c) The development of Ready to Use Therapeutic Foods (RUTF) based on local capacity

This model can easily be implemented and resourced even in impoverished environment.

In outpatient therapeutic program they receive a ration of takehome ready to use therapeutic food (RUTF) to provide energyof 200Kcal/kg/day, a course of oral broad spectrum antibiotic, folic acid, anthelminthic and if appropriate anti-malarial.

READY TO USE THERAPEUTIC FOOD (RUTF)

Development of Ready to Use Therapeutic Food has greatly eased the difficulties associated with providing a suitable energy high-nutrient dense food that is safe to use in outpatient program [18]. RUFT is an energy dense food enriched with minerals and vitamins with similar nutrient profile but greater energy and nutrient density than F-100, the diet recommended by WHO in the recovery phase of treatment of SAM. The original RUTF recipes contains five ingredients- Peanut-butter, vegetable oil, powdered sugar, dried skimmed milk and a vitamin mineral mix. In contrast to water based F-100, RUTF is an oil based paste with extremely low water activity. As a result RUFT food does not grow bacteria, allowing it to be kept unrefrigerated in simple packaging for several months. As the food is eaten uncooked, heat labile vitamins are not destroyed and labour, fuel and water demands on poor household are minimised. The production process is simple and is RUTF can be made from local crops with basic technology that is readily available in developing countries.

The development of RUTF food has allowed much of the management of SAM to move out of hospital by shortening of duration of inpatient treatment; the move towards using RUTF food in the recovery phase of treatment reduces the resources to treat SAM which improves cost effectiveness.

Currently imported, commercially produced RUTF are used. Therefore RUTF needs to be more easily accessible and affordable for the approach to be sustainable. Local production of RUTF needs to be promoted for increase access and availability to RUTF through reducing cost.

Commercially available nutrient dense food is expensive. RUTF itself is used in acute phase of rehabilitation and prescribed as therapeutic item not as food. Therefore locally made RUTF in the community based treatment of childhood malnutrition is feasible and desirable [19,20]. The success of home based treatment of severe malnutrition will require the provision of homemade nutrient dense food supplement which can be safely stored and administered without much preparation by caregiver.

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Cite this article

Shakur S, Afroze S, Shakur S (2018) Marasmus: An Update and Review of Literature. JSM Nutr Disord 2(1): 1008.