Chapter 5:

NUTRITIONAL DISORDERS

Student Authors: Chiraag Seedat and Buncwane Mpakama

Specialist Advisor: Dr Lesego Ndhlovu

This chapter covers the following topics:

- Severe acute malnutrition (SAM)
- Failure-to-thrive (FTT)
- Rickets
- Vitamin and mineral deficiencies

SEVERE ACUTE MALNUTRITION (SAM)

Definition

SAM is diagnosed based on the presence of any one of the following:

- Weight-for-height below the -3 Z score on WHO standard growth charts
- Mid-upper arm circumference <11.5 cm
- Clinical signs of nutritional oedema

Pathophysiology

SAM affects many organs and organ systems. These include:

- Cardiovascular – decreased cardiac output, decreased stroke volume, decreased contractility, bradycardia, hypotension
- Urinary – decreased GFR, increased risk of UTIs
- Gastrointestinal – decreased gastric acid, decreased pancreatic digestive enzymes, decreased absorption of nutrients, loss of intestinal barrier function, atrophy of intestinal mucosa, diarrhoea (common)
- Liver – decreased albumin synthesis, decreased gluconeogenesis, decreased lipoprotein synthesis
- Immune – decreased cell-mediated immunity, decreased acute phase immune response, increased risk of sepsis
- Endocrine – decreased insulin, decreased cortisol and growth hormone levels
- Metabolism – impaired heat generation and heat loss, decreased basal metabolic rate (by 30%)
- Cells – decreased cell membrane permeability and sodium pump activity (result in increased intracellular sodium and decreased intracellular potassium levels)
- Haematological – increased risk of anaemia
- Neurological and psychological – increased irritability and apathy, decreased social responsiveness, attention disorders
- Dermatological and musculoskeletal – decreased skeletal muscle mass, decreased subcutaneous fat, atrophy of salivary glands

Clinical Features

The clinical features of a child with SAM can be divided into SAM with oedema or SAM without oedema or a combination of both (see also two types of malnutrition stated here).

Table 5.1: Comparison of the Clinical Features of SAM with Oedema to SAM without Oedema

SAM with Oedema (previously Kwashiorkor)	**SAM without Oedema (previously Marasmus)**

• Weight-for-age is variable
• Oedema:
  ○ May have facial oedema
  ○ Will have pitting oedema of the extremities
  ○ Ascites rarely occurs but the abdomen may be distended (due to hepatomegaly)
• Child is apathetic and listless
• Skin is hyperpigmented, dry and splits when stretched (crazy paving dermatosis)
• Hair is dry, dull and hypopigmented
See related image here.
	• Low weight-for-height
	• Head is large relative to body
	• Face has a "wizened" appearance and staring eyes
	• No oedema
	• Arms, thighs and buttocks appear emaciated due to loss of subcutaneous fat
	• Child is irritable and fretful
	• Skin is thin and dry
	• Hair is thin and sparse
	See related image here.

Children with SAM may also have features of vitamin and mineral deficiencies, dehydration, shock and/or infection. Clinical assessment of dehydration in children with SAM is difficult as the loss of subcutaneous fat makes the signs of dehydration (e.g. as skin turgor, sunken eyes) unreliable. Infection is also more difficult to diagnose as the features of infection (e.g. fever, leucocytosis) may also be absent in children with SAM due to the effect of the malnutrition on the body's immune system.

Investigations

Investigation choice should be guided by history and clinical examination.

Recommended tests include:

- Investigations for infections: blood culture, malaria blood film (in endemic areas), urine dipstick and Urine MCS, stool microscopy, especially if diarrhoea, chest X-ray if respiratory distress (for respiratory infections), HIV test and TB screening
- Blood glucose
- FBC and blood smear
- Electrolytes (Na, K, Ca, Pi, Mg)
- Serum albumin, LFTs
- Renal function tests

Complications

For the purposes of management, SAM may also be classified as complicated or uncomplicated.

Table 5.2: Features of Complicated and Uncomplicated SAM

Complicated SAM	Uncomplicated SAM
● Child < 6 months or less 4kg ● Pitting oedema (SAM with oedema) ● Dehydration ● Vomiting ● Refusing feeds or not eating well ● Hypoglycaemia ● Hypothermia ● Respiratory distress ● Convulsions ● Shock ● Lethargy ● Jaundice ● Weeping skin lesions ● Bleeding	● Child >6 months and >4 kg ● No pitting oedema ● Good appetite and feeding well ● Alert ● No danger signs

Management

SAM is managed according to the WHO Ten Steps. Management is divided into the stabilisation and rehabilitation phase. The stabilisation phase is the first few days of management, when one's focus is the restoration of metabolic and physical stability. The rehabilitation phase usually begins between day 3 and 7 of admission.

Complicated SAM should be managed from WHO step 1 while uncomplicated SAM may be managed from the rehabilitation phase.

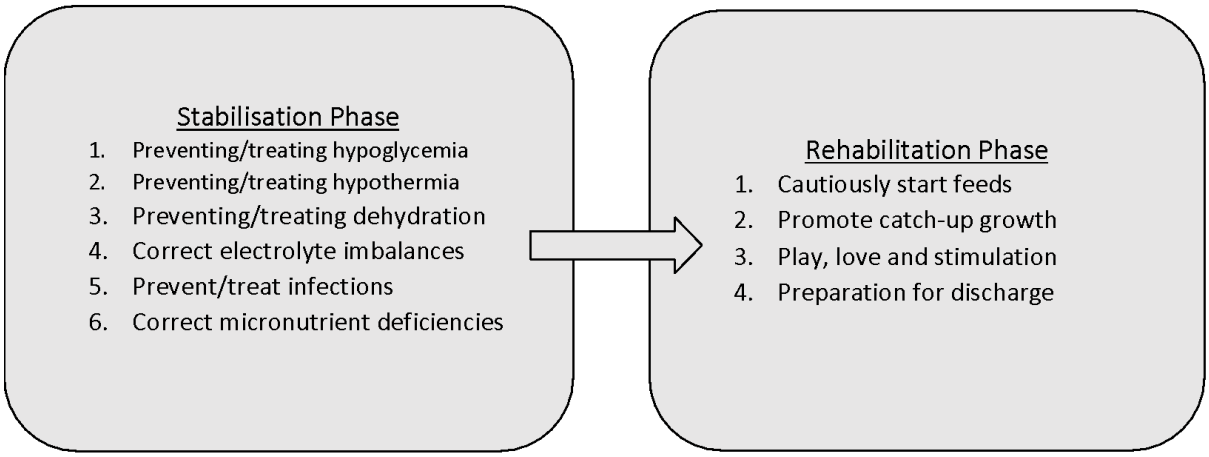

Figure 5.4: WHO Ten Steps

Table 5.3: WHO Ten Steps and Management Plans

WHO Ten Steps		
Step		**Management**
1	Treat/prevent hypoglycaemia (glucose <3mmol/l)	Treatment – give 50 mL 10% dextrose solution orally or by nasogastric tube (NGT if not tolerating oral feeds) every 30 mins for 2 hours; if unconscious or lethargic, give a 5mL/kg bolus of IV dextrose 10% Prevention – feed two hourly (including throughout the night)
2	Treat/prevent hypothermia (axillary temp <35°C)	Treatment – feed immediately; rewarm with clothes, blankets, a heater lamp or skin-to-skin contact (do not use a hot water bottle on direct skin); during rewarming, monitor temperature two hourly until >36.5°C

		Prevention – feed 2 hourly (day and night); keep the child covered and dry; change wet nappies, clothes and bedding; avoid exposure to cold environments; let the child sleep with his/her caregiver
3	Treat/prevent dehydration	Treatment – give 5 mL/kg ORS or rehydration solution for malnutrition (ReSoMal) every 30 mins for two hours and then give 5-10 mL/kg/hr; also replace the fluid lost from diarrhoea and vomiting; if the child is shocked, IV fluid is required (see *Shock* in *Feeding and Fluid Management* chapter) Prevention – give 10 mL/kg ORS after each watery stool and give maintenance fluids (e.g. continue with breastfeeds)
4	Correct electrolyte imbalances	All children with SAM have excess body sodium, even if plasma sodium is low. Deficiencies of potassium, magnesium and phosphate are common and require correction.
5	Treat/prevent infection	Broad-spectrum antibiotics are routinely given as the signs of infection are often absent in these children. Oral amoxicillin may be used in uncomplicated cases but IV gentamycin and ampicillin are used in complicated/severe SAM.
6	Correct micronutrient deficiencies	Children with SAM have multiple micronutrient deficiencies. Supplementation recommendations include multivitamins (especially vitamin A), folic acid (1 mg/day) and zinc (2 mg/kg/day). Copper (0.3 mg/kg/day) and iron (3 mg/kg/day) supplementation should be given for two weeks once the child has been stabilised.
7	Cautiously start feeds	Feeding should be started as soon as possible, but caution is needed due to the child's fragile state. Feeds should be small and frequent and should be given orally

		or via NGT. The WHO recommends use of the F75 formula.
8	Promote catch-up growth	Feeding is increased to achieve weight gain of 10 g/kg/day. The WHO recommends the use of the F100 formula.
9	Provide sensory stimulation and emotional support	Children with SAM may have delayed mental and behavioural development. They require care, a stimulating environment, play and physical activity.
10	Prepare for follow-up after discharge	Children can be discharged if they are not oedematous, have good appetites, show good weight gain, do not have infections, are playful and are alert. Parents/guardians need to be counselled on good feeding practices and the provision of structured playtime. A follow-up plan should be arranged to ensure that the child is growing well.

FAILURE-TO-THRIVE (FTT)

Definition

There is no formal definition for FTT. However, the term is generally used to describe children who have unsatisfactory weight gain (growth curve flattening or weight loss) or a low weight for age but a z-score of > -2 on WHO standard growth charts or the Road-to-Health charts (used in South Africa) when corrected for age, gender, genetic potential and medical condition.

Aetiology

There are many causes of FTT. A thorough history and examination is essential as one must find the underlying aetiology. The causes can be divided into four categories:

- Inadequate intake:
    - Feeding/social issues e.g. poverty, incorrect feeding techniques
    - Mechanical issues e.g. cleft palate, nasal obstruction
    - Inability or difficulty swallowing e.g. HIE, cerebral palsy
    - Gastroeosophageal reflux disease

- Inadequate nutrient absorption:
    - Malabsorption e.g. infection, cystic fibrosis, lactose intolerance, coeliac disease
    - Intestinal obstruction e.g. hernia, pyloric stenosis
    - Short bowel syndrome
- Increased nutrient utilisation:
    - Chronic diseases and infections e.g. TB, HIV, chronic cardiac and respiratory diseases
    - Genetic diseases
    - Metabolic conditions e.g. storage diseases
    - Endocrine disorders e.g. diabetes mellitus, hyperthyroidism
- Excess nutrient loss
    - Chronic gastroenteritis

Clinical Features

The child will have:

- Weight <3rd percentile for gestational age when plotted on the appropriate chart e.g. growth chart for the child with Down syndrome
- Weight for height <10th percentile when plotted on the appropriate chart
- Weight loss of ≥2 percentiles
- Daily weight gain less than expected for age

The child may also have features of underlying diseases causing FTT and/or features of vitamin and nutrient deficiencies.

Investigations and Management

Investigations should be guided by history and examination.

Management depends on the underlying aetiology. Outpatient treatment can be considered if the cause is impaired intake and enteral nutrition can meet patient requirements. Inpatient management is generally required for patients with other medical conditions, with severe malnutrition, who have suffered child abuse/neglect and/or significant psychosocial issues. Management should involve a

multidisciplinary team that may include a dietician, social worker and psychiatrist, depending on the needs of the child.

RICKETS

Definition

Rickets refers to the failure of mineralisation of bones in growing children.

Table 5.4: Pathophysiology of Vitamin D Metabolism

Vitamin D Metabolism

Vitamin D has two forms – Vitamin D2 (ergocalciferol) and Vitamin D3 (cholecalciferol). Vitamin D is either acquired directly from diet or through the conversion of 7-dehydrocholesterol to vitamin D3 when dehydrocholesterol in the skin is exposed to UV light. The vitamin D is then stored (in muscle or fat) or transported to the liver where it is hydroxylated to 25-hydroxyvitamin D (calcidiol – an inactive compound is inactive).

Calcidiol is hydroxylated in the kidney to its active form, 1,25 hydroxyvitamin D (calcitriol). Calcitriol promotes calcium and phosphate absorption in the gut and increases renal absorption of both calcium and phosphate. Parathyroid hormone (PTH) is responsible for the regulation of calcitriol. In response to low calcium levels, PTH increases the activity of 1-α-hydroxylase, an enzyme that is responsible for this conversion of calcidiol to calcitriol. High calcitriol levels act in a negative feedback mechanism and decrease PTH secretion.

Calcium and Phosphate Homeostasis

Calcium and phosphate are absorbed in the GIT (under the influence of calcitriol) and are mostly stored in the bone as hydroxyapatite. Ionised (free, non-protein-bound) calcium and phosphate are filtered by the kidney. Over 95% of filtered calcium is reabsorbed. (See a diagram showing the mechanisms of maintenance of calcium homeostasis here.)

PTH and calcitonin play an important role in calcium regulation. PTH is secreted in response to low serum calcium levels. It increases the calcium concentration by

increasing intestinal absorption and promoting renal reabsorption of calcium. It is worth noting that PTH has the opposite effect on phosphate as it decreases phosphate absorption by the kidney. PTH also causes bone resorption by increasing osteoclast activity resulting in the release of calcium from the bone.

Calcitonin is released when serum calcium levels are high. It is released from C cells in the thyroid gland and works to suppress calcium release from bone and decrease calcium absorption from the intestines and kidneys.

Aetiology

Calcium and phosphate play an important role in bone mineralisation. Deficiencies in either of these may, therefore, cause rickets. Dietary deficiency is the most common cause.

Table 5.5: Causes of Rickets in Children

Causes of Rickets in Children	
Calcium-deficiency rickets	● Vitamin D deficiency (more common in infants): ○ Dietary deficiency of Vitamin D ○ Inadequate exposure to sunlight ○ Impaired absorption of Vitamin D: Fat malabsorption, coeliac disease ○ Impaired hydroxylation of Vitamin D to 25-hydroxyvitamin D: liver immaturity, prematurity ○ Decreased renal synthesis of 1,25 dihydroxy vitamin D: renal failure ● Calcium deficiency (more common in older children): ○ Decreased dietary intake
Phosphate-deficiency rickets	● Decreased intake of phosphate ● Decreased intestinal absorption of phosphate: aluminium hydroxide ingestion ● Increased renal loss of phosphate: genetic disorders (X-linked), Fanconi syndrome, mesenchymal tumours.

Clinical Features

The child with rickets (see also an image of a child with rickets here) may have:

- Delayed closure of fontanelles:
    - The posterior fontanelle closes by 3 months, while the anterior fontanelle should be closed by 24 months
- Frontal and parietal bossing (protuberance of the skull):
    - Children with frontal bossing will have an unusually prominent forehead
- Craniotabes (softening of the skull bones)
- Rachitic rosary:
    - This is the widening of the ends of the anterior ribs at the costochondral junctions
    - It may be visible as beading or palpable as nodules at the costochondral junction
- Harrison's sulcus (groove in the lower margin of the thorax due to the pull of the diaphragm on the lower ribs)
- Widening of the wrist and bowing of the distal radius and ulna
- Lateral bowing of the femur and tibia (see figure 3.2)

The child may also have features of hypocalcaemia.

Diagnosis and Investigations

X-rays can be used to diagnose rickets (see X-ray here and here). Rachitic changes are best seen at the metaphysis of the knee (distal femur and proximal tibia), wrist (especially the distal ulna) and the anterior ribs (rachitic rosary). Features on X-ray include:

- Cupping and fraying of metaphysis (see figure 5.8)
- Widened epiphyseal plates.
- Poorly mineralised epiphyseal plates
- Cortical spurs
- Coarse trabeculation
- Deformities: bowing of long bone, fractures, frontal bossing

Calcium, phosphate, PTH and alkaline phosphatase (ALP) levels should also be measured. In calcium-deficiency rickets, calcium and phosphate levels are low while

ALP and PTH levels are high. In phosphate-deficiency rickets, phosphate levels are low while calcium, ALP and PTH levels may be raised or normal.

Management

Calcium-deficiency rickets is generally treated with Vitamin D supplementation of 1000 to 5000 IU/day for 4-6 weeks, as the most common cause of this rickets is dietary deficiency. If hypocalcaemia is present, this should be corrected. An X-ray should be done after 8-12 weeks of treatment to ensure adequate bone healing. Failure to respond to treatment requires further investigation and management will depend on the underlying cause.

Phosphate-deficiency rickets will generally require further investigations and management will, therefore, depend on the cause.

VITAMIN AND MINERAL DEFICIENCIES

Classification of Nutrients

Nutrients in food can be classified based on how much of the nutrient is needed by the body or based on the body's response to deficiency of the nutrient.

Classification Based on Quantity Required

Nutrients may be classified as:

- Macronutrients – carbohydrates, protein and fat
- Micronutrients – vitamins, macrominerals and microminerals

Classification Based on Response to Deficiency (Golden Classification)

Table 5.6: Golden Classification of Nutrients

TYPE I NUTRIENT DEFICIENCIES	TYPE II NUTRIENT DEFICIENCIES
● Deficiencies of these nutrients manifest with early clinical signs without abnormal anthropometry ● Examples include vitamins, iron, copper and iodine	● Deficiencies of these vitamins do not present with obvious, early clinical features and may therefore go unrecognised ● However, they lead to growth failure (stunting and wasting) ● Examples include protein, zinc, magnesium and potassium

Vitamins

Vitamins can be divided into two groups – fat-soluble vitamins (vitamins A, D, E and K) and water-soluble vitamins (vitamin B complex and vitamin C). Deficiencies of water-soluble vitamins tend to develop after weeks to months of malnutrition. However, deficiencies of fat-soluble vitamins take longer than a year to develop because the body is able to store larger amounts of these vitamins.

Minerals

Minerals can be divided into macrominerals and microminerals (also known as trace minerals). Macrominerals are needed in milligram amounts on a daily basis and include sodium, potassium, chloride and calcium. Microminerals are needed in smaller quantities and include copper and zinc. Chromium, fluoride, iodide, selenium and cobalt are also important minerals but are needed in even smaller amounts and are therefore known as ultra-trace minerals.