



## Symptoms

The clinical presentation of marasmus varies greatly depending on the severity and duration of calorie restriction. Furthermore, the age of the patient and whether or not the marasmus is associated with vitamin and mineral deficiencies will contribute to different presentations of marasmus.

Marasmus will present with a failure to thrive. In infants, it may be associated with irritability and apathy. Furthermore, infants may have sunken fontanelles as a result of dehydration. The general appearance is shrunken and wasted due to reduced levels of subcutaneous fat. Weight loss is initially most noticeable in the groin or axilla, later on in the buttocks, face, and thigh. Wasting of fat in the face leads to a characteristic 'old man' appearance. Marasmus may also be associated with hypotension, hypothermia, and bradycardia.

The child will be less than 60% of the weight for age. Long-standing marasmus is associated with growth stunting; this may lead to the weight for height being within the normal range. Marasmus is frequently associated with symptoms of anemia and rickets.

#### *BioTalk on Symptoms, Diagnosis & Treatment of Marasmus*

As marasmus progresses, several body systems may be affected. An impairment in cardiovascular function can contribute to hypotension, hypothermia, and bradycardia.

As marasmus tends to be associated with other micronutrient deficiencies, it is important to recognize that these may also present in a child with marasmus. The child may suffer from dry eyes and the development of Bitot spots secondary to vitamin A deficiency. The nails may have a spoon-shaped appearance (koilonychia) secondary to iron deficiency and anemia. Hypocalcemia may lead to the presentation of Chvostek or Trousseau signs. Over a long period of time, a deficiency in calcium and vitamin D may lead to the development of associated rickets or other bone deformities.

It is important to note that in marasmus, the signs indicative of kwashiorkor such as edema, scarcity, dermatosis, depigmentation of hair, and cheilosis are not present.

## Diagnosis

Anthropometry is essential in the diagnosis of marasmus; this may be difficult to perform in a reliable and repeatable manner in low-income countries. This involves accurate measurement of vertical length to a precision of 0.5 cm, weight to a precision of 100 g, and middle-upper arm circumference (MUAC) to a precision of at least 2 mm or less. Pitting

edema should also be investigated to rule out kwashiorkor.

The diagnosis of marasmus is made if there is a MUAC of less than 115 mm or if the weight for height Z score is greater than three standard deviations below the mean.

Laboratory investigations can be used in the diagnosis of protein-energy malnutrition along with the investigation of associated mineral deficiencies. Laboratory investigations recommended by the WHO include hemoglobin and blood smear, blood glucose, serum albumin, electrolytes, stool microscopy, and culture, including investigation of parasites, human immunodeficiency virus testing, and urine microscopy and culture.

A full blood cell count can help to identify low levels of hemoglobin or abnormalities in the red cell indices; furthermore, a blood film smear can be used in the diagnosis of anemia and malaria in some circumstances. Iron studies, folic acid, and B12 levels are also frequently used.

Other investigations frequently used as part of a nutrition profile include specific tests for plasma proteins such as transferrin, albumin, and thyroxine-binding prealbumin. Albumin levels are the most frequently used biochemical marker of nutritional status; however, the long half-life of albumin (19 days) means that it is less useful in monitoring the short-term nutritional changes, for example, in response to treatment. Other proteins, such as thyroxine-binding prealbumin (2 days) and retinol-binding protein (10 hours), have a shorter half-life and are more useful in determining a response to treatment.

## Treatment / Management

The main causes of death in severe malnutrition include infection, dehydration, electrolyte imbalance, and heart failure; furthermore, there is a risk of death due to the onset of refeeding syndrome. The treatment of marasmus may be in a hospital or in the community, however, it has been shown that community-based treatment in children with uncomplicated

severe malnutrition has better outcomes compared to hospital care.

The management of marasmus is divided into three main phases:

1. Resuscitation and stabilization
2. Nutritional rehabilitation
3. Follow up and prevention of recurrence

### Resuscitation and Stabilization

As the main causes of death in marasmus include dehydration and infection, the main aim during the resuscitation and stabilization phase is to rehydrate, prevent infections that may progress to sepsis, and avoid the complications of the treatment of marasmus such as refeeding syndrome. This phase lasts for approximately one week and is the phase during which patients are most susceptible.

Dehydration can be treated with an intravenous isotonic solution, in circumstances where the child is suffering from hypovolemia plasma or blood may be used. The child should be in a warm room as they are susceptible to hypothermia. Furthermore, as there is an atypical response to infections a child with marasmus may not have overt signs indicating they are suffering from sepsis, antibiotics may be given following blood cultures in those who are suspected to be suffering from sepsis.

To prevent the development of refeeding syndrome, nutrition should be delivered slowly and carefully with caloric intake between 60-80% of the calorie requirement for age. A potential risk of refeeding is the development of hypoglycemia, this can be avoided by continuous nasogastric feeding at night or small meals during the nighttime. Vitamins such as thiamine and oral phosphate should be administered to prevent the development of hypophosphatemia which is associated with refeeding.

During this phase of treatment, the recognition of refeeding syndrome is of great importance. Electrolyte abnormalities developed as a consequence of refeeding syndrome

may lead to arrhythmia or sudden death, weakness, and rhabdomyolysis, confusion, and death. Thiamine deficiency may lead to encephalopathy or lactic acidosis. Fluid overload may result in acute heart failure and edema.

### Nutritional Rehabilitation

Once the acute complications of marasmus have been treated and the child's appetite begins to return along with the correction of electrolyte abnormalities and sepsis the nutritional rehabilitation phase can start. This includes the gradual increase of caloric intake, vaccination, and increased motor activity. Children may need 120% to 140% of their required caloric intake to maintain a growth rate similar to their peers. The nutritional rehabilitation phase may last from 2 to 6 weeks.

During this phase, it is important to encourage mother and child interaction to help reverse developmental delay.

### Follow up and Prevention of Recurrence

As there is a risk of relapse, it is important to follow up on patients who have presented with marasmus. Education should be provided to mothers with regards to breastfeeding and supplemental feeding.

Furthermore, other ways in which marasmus can be reduced include the provision of a supply of uncontaminated drinking water, adequate food supplies, and control of infectious diseases.

### Source

*Titi-Lartey OA, Gupta V. Marasmus. [Updated 2022 Jul 25]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK559224/>*



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