

**SEVERE ACUTE MALNUTRITION.
A BRIEFING FOR DOCTORS AND NURSES.**

Medical practice for the management of Severe Acute Malnutrition has dramatically changed in the last 20 years. This affects all aspects, from diagnosis to treatment, and includes a more profound understanding of its pathophysiology, the mechanisms of development of complications (natural and iatrogenic) and even its name.

Severe Acute Malnutrition used to be referred to in medical books as “Protein Energy Malnutrition (PEM)”. This name implied that malnutrition was deficiency of protein or energy and, as for other specific deficiencies, suggested that treatment should consist on proteins or energy. Usually textbooks went on to say that Marasmus (extreme thinness) was due to lack of energy, and Kwashiorkor (oedematous malnutrition) to lack of proteins. This has been now proved incorrect and even dangerous.

Since there is little activity around this condition in western countries, some medical textbooks keep referring to it in the old – outdated – way, maintaining old concepts and treatment. These treatments are associated to a mortality of more than 50 %. Modern treatment of malnutrition can reach mortality rates as low as 1 to 3 percent.

Here are some misconceptions that need to be eradicated:

- Marasmus is lack of energy: It can be true, but it can be due as well to lack of other essential micronutrients needed for growth and tissue renovation (the sort of nutrients that are present in every cell). Marasmus can happen with excess energy if no nutrients are taken.
- Kwashiorkor is lack of protein: The oedema of malnutrition is not due to osmotic causes, as has been long thought. In fact, it is due to membrane leaking, after damage produced by free radicals. These are in turn the consequence of an external noxa (usually infection) and specific deficiencies of antioxidants (probably Selenium). Kwashiorkor can happen with protein excess in diet.
- Treatment consists in big amounts of proteins and energy: Since the metabolism of the malnourished child is slowed down, excessive proteins and energy may be deleterious. For example, the ability of the liver to detoxify proteins is very limited, and a protein-rich diet may become toxic.

In fact, the modern treatment of acute malnutrition is based in an initial treatment with low protein, low energy diet, enriched with essential micronutrients. Only after metabolic equilibrium is obtained (without weight gain) the patient can start to recover with high quantities of energy, proteins and the right balance of macro and micronutrients to promote tissue growth.

Malnutrition is usually diagnosed through anthropometry (weight-for-height, in which the weight and height of the child is compared to those of an international reference; MUAC, or other). However, the main symptom that reveals the metabolic status of the child is “lack of appetite”. Patients with no appetite usually present a severe complication -visible or not- that need specialised treatment. Malnourished children without complications – and with appetite- can be treated at home.

Pathophysiology of Severe Acute Malnutrition

Severe acute malnutrition can result in profound metabolic, physiological and anatomical changes. Virtually all physiological processes are altered due to severe acute malnutrition. Every organs and systems are involved in reductive adaptation.

Reductive adaptation is the physiological response of the body to under nutrition i.e. systems slowing down to survive on limited macro and micro-nutrients intake. The system *reduces* activity, to *adapt* to the lack of nutrients and energy.

This results in profound **physiological and metabolic changes**, some of which can be observed by the clinician, and other which are not. The initial reductions will not alter normal function of the body BUT will affect its capacity to adapt to any other new situation (an infection, cold, or even to an IV infusion or excessive oral liquids). For example, the circulatory system may be still working correctly, with no signs or symptoms of presence of a problem... BUT it may not be able to adapt to a sudden increase of circulatory volume (after an infusion or a transfusion, for example). Since the adaptive mechanisms to increased volume cannot be mobilised, a simple infusion may result in cardiac overload and lethal pulmonary oedema. Similar situations occur with: digestive system, and the amount of proteins and other nutrients that can be absorbed in one meal; immune system, and its ability to respond to infection; liver ability to detoxify and kidney ability to excrete, etc.

Children with malnutrition do not show usual symptoms and signs:

In addition, some of the changes mentioned result in unusual signs and symptoms. For example, a child with severe acute malnutrition may not be able to present fever in face of an infection. In fact, very often the infection will present with hypothermia ! You can see other examples in the following page.

Usual life-saving actions may be dangerous in the malnourished child !!!!

These are the reasons why it is so important to follow standard protocols for the treatment of severe acute malnutrition and its complications (like this one). The changes in metabolic and physiological responses in the malnourished child are so important that therapeutic decisions that are live-saving in a well nourished child can be potentially mortal in the malnourished child.

The following page presents some of the main alterations in each of the body systems. Knowing them can help understand the evolution and therapy of severe acute malnutrition and its complications.

Cardiovascular system:

- Cardiac output and stroke volume are reduced.
- Infusion of saline may cause an increase in venous pressure.
- Any increase in blood volume can easily produce acute heart failure.
- Any decrease will further compromise tissue perfusion.
- Blood pressure is low.
- Renal perfusion and circulation time are reduced.
- Plasma volume is usually normal and red cell volume is reduced.

Gastro-intestinal system

- Production of gastric acid is reduced.
- Intestinal motility is reduced.
- Pancreas is atrophied and production of digestive enzymes is reduced.
- Small intestinal mucosa is atrophied; secretion of digestive enzymes is reduced.
- Absorption of nutrients is reduced.

Liver function

- Synthesis of all proteins is reduced.
- Abnormal metabolites of amino acids are produced.
- Capacity of liver to take up, metabolize and excrete toxins is severely reduced.
- Energy production from galactose and fructose is much slower than normal.
- Gluconeogenesis is reduced, with high risk of hypoglycemia during infection.
- Bile secretion is reduced.

Genitourinary system

- Glomerular filtration is reduced.
- Capacity of kidney to excrete excess acid or a water load is greatly reduced.
- Urinary phosphate output is low.
- Sodium excretion is reduced.
- Urinary tract infection is common.

Immune system

- All aspects of immunity are diminished.
- Lymph glands, tonsils and thymus are atrophied Cell-mediated immunity is severely depressed.
- Ig-A levels in secretions are reduced.
- Complement components are low.
- Phagocytes do not kill ingested bacteria efficiently.
- Tissue damage does not result in inflammation or migration of white cells to the affected area.
- Acute phase immune response is diminished.
- Typical signs of infection, such as an increased white cell count and fever, are frequently absent.
- Hypoglycaemia and hypothermia are signs of severe infection usually associated with septic shock

Endocrine system

- Insulin levels are reduced and the child has glucose intolerance.
- Insulin growth factor 1 (IGF-1) levels are reduced.
- Growth hormone levels are increased.
- Cortisol levels are usually increased.

Circulatory system

- Basic metabolic rate is reduced by about 30%.
- Energy expenditure due to activity is very low.
- Both heat generation and heat loss are impaired.
- The child becomes hypothermic in a cold environment and hyperthermic in a hot environment.

Taking the child's medical history and conducting the physical examination. Check list.

Medical history:

1. Usual diet before current episode of illness,
2. Breastfeeding history,
3. Food and fluids taken in the past few days,
4. Recent sinking of eyes,
5. Duration and frequency of vomiting or diarrhoea, appearance of vomit or diarrhoeal stools,
6. Time when urine was last passed,
7. Contact with people with measles or tuberculosis,
8. Any deaths of siblings,
9. Birth weight,
10. Milestones reached (sitting up, standing, etc.)
11. Immunisations.

Physical examination:

1. Weight and length or height,
2. Oedema,
3. Appetite: anorexia,
4. Enlargement or tenderness of the liver, jaundice,
5. Abdominal distension, bowel sounds, "abdominal splash" (a splashing sound in the abdomen),
6. Severe pallor,
7. Signs of circulatory collapse: cold hands and feet, weak radial pulse, diminished consciousness,
8. Temperature: hypothermia or fever,
9. Thirst,
10. Eyes: corneal lesions indicative of Vitamin A deficiency,
11. Ears, mouth, throat: evidence of infection,
12. Skin: evidence of infection or purpura,
13. Respiratory rate and type of respiration: signs of pneumonia or heart failure,
14. Appearance of faeces.

WARNING: NEVER DO ANY OF THE FOLLOWING:

- Never give diuretics against malnutrition oedema. The oedema is partially due to potassium and magnesium deficiency, that can easily recover in two weeks. Oedema disappears with appropriate feeding adding a micronutrient solution. Giving diuretics would aggravate the electrolyte imbalance and would risk death.
- Do not give Iron in the first days of treatment (until Phase 2 or Rehabilitation phase). It risks having toxic effects and reduce defense against infections.
- Do not give preparations rich in proteins (more than 1.5 g of protein per kg/day). Any excess in the first days can be dangerous, because the severely malnourished child is not able to assume the metabolic effort needed to deal with them. An excess of proteins can overload the liver, the heart and kidneys and provoke death.
- Do not give liquids in perfusion. In the child with severe malnutrition liquids in perfusion can easily produce cardiac overload. These are only given when there is a diagnosis of septic shock.
- Do not give blood transfusion. Most anaemia in malnourished children under treatment is in fact an hemo-dilution, due to the return to the blood stream of liquid accumulated as oedema, or retained in cells (marasmus). This is resolved in less than 2 – 4 days . Wrong treatment with transfusion often results in cardiac overload and death from pulmonary oedema.



Some references for those who would like to know more:

Most manuals are not up to date in treatment of malnutrition. Here are some that are:

Oxford Handbook of Tropical Medicine. 3rd Edition. All should read page 64 and following. Actually, a great bedside reference for medicine in the tropics (not just a parasitology manual).

<http://www.oup.com/us/catalog/general/subject/Medicine/ImmunologyInfectiousDisease/?view=usa&ci=9780199204090#Features>

WHO protocols for treatment of Severe Acute Malnutrition.

www.who.int/nutrition/publications/malnutrition/en/

Malnutrition and HIV/AIDS :

Several documents can be found here: www.fantaproject.org/focus/hiv_aids.shtml

And here: www.unsystem.org/scn/

Look nutrition and HIV on the left side of the screen to access the material by main subject

Physiopathology and treatment of severe malnutrition:

And here is a brief paper on the physiopathology of severe acute malnutrition, that is free access on the web. **Ashworth, A. 2001, 'Treatment of severe malnutrition', J Pediatr Gastroenterol Nutr, vol. 32, no. 5, pp. 516-8. (www.jpagn.org).**