Malnutrition in developing countries

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Abstract

Although now rare in industrialized countries, severe acute malnutrition is unfortunately still common throughout the developing world and is a key contributor to both global childhood morbidity and mortality. This review describes the epidemiology of malnutrition and the presentation and pathophysiology of the severe syndromic forms — marasmus and kwashiorkor. The gold standards for diagnosis and management are detailed and the challenges of implementation in the basic healthcare systems of the developing world are discussed. As the leading cause of ill health in the world today, more effective treatment and prevention of malnutrition must be a priority for the global healthcare community.

Keywords kwashiorkor; marasmus; protein-energy malnutrition; severe acute malnutrition; underweight

Underweight: the leading cause of ill health in the world today

In 2008, of the 8.8 million global deaths of children under 5 years of age, 93% occurred in the developing countries of Africa and Asia. The highest rates of childhood mortality are found in sub-Saharan Africa where one in seven children die before their fifth birthday. Outside the neonatal period, the most common primary causes of death are pneumonia (19%) and diarrhoea (17%) but over a third of all deaths can be attributed to underlying undernutrition (see Figure 1). It is estimated that there are 148 million underweight children: 78 million live in South Asia and 36 million in sub-Saharan Africa. In the developing world, 20% of children are underweight and 3.5% (19 million) are severely malnourished. Millennium Development Goal 4 (a reduction in global under-5 mortality by 2/3 between 1990 and 2015) will only be achieved if significant gains in both the prevention and management of malnutrition are made.

As well as causing loss of life, malnutrition results in substantial morbidity and loss of quality of life; long-term developmental problems and educational underachievement diminish the ability to work thereby reducing potential for national development. When calculated as disability-adjusted life years, underweight in childhood is the leading global risk factor for ill health in the world today.

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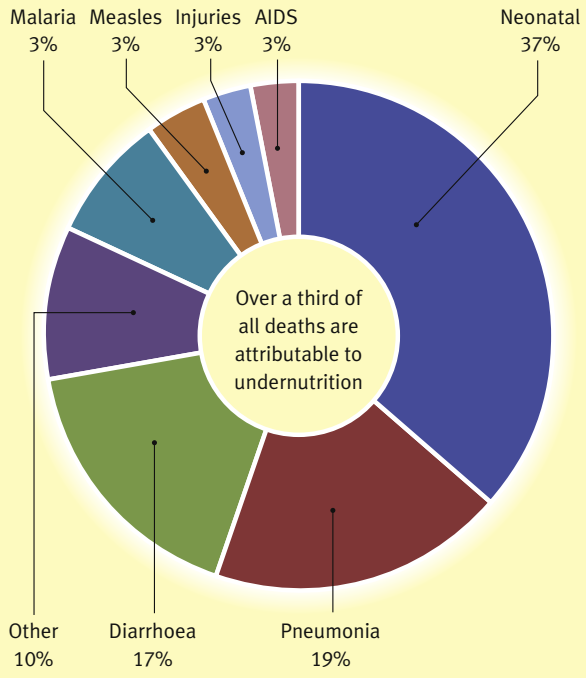

Based on data of the Child Mortality Estimation Group used in the UNICEF report 'The State of the World's Children 2008'

Figure 1 Global cause specific mortality in children under 5 years of age.

Definitions and diagnosis

The diagnosis of malnutrition is based on measurement of body size (anthropometry) and clinical signs (see Box 1).

Based on the 2006 WHO Child Growth Standards, underweight is defined as a weight that is greater than 2 standard deviations (or "z scores") below the median expected weight for age. This could be due either to stunting (low height for age) or wasting (low weight for height). Moderate wasting is weight for height z score (WHZ) less than −2 and severe wasting WHZ less than −3.

Stunting is a result of chronic undernutrition and these children require multiple interventions to improve their health and well-being. Wasting and/or nutritional oedema signifies acute malnutrition and requires immediate and intense intervention. All degrees of malnutrition impact negatively on health. Because of the large numbers affected, the great majority of malnutrition-associated deaths occur in mild and moderately underweight children. However, compared with children with a WHZ greater than −1, the odds ratio for mortality is estimated to be 3.0 for

Criteria for the diagnosis of severe acute malnutrition

One or more of:-

- Weight for height z score of less than −3
- Presence of bilateral pitting pedal oedema
- Mid upper arm circumference (MUAC) of less than 11.5 cm

Box 1

WHZ less than −2 and 9.4 for WHZ less than −3. Therefore, severely wasted children are the focus of in-patient treatment programmes.

In the resource limited settings where malnutrition is common, accurate measurement of weight and height may not be possible and calculation of age and access to, and correct use of, the reference norms may also be difficult. MUAC (mid upper arm circumference) may be more appropriate in these situations as it can be measured more easily. MUAC is relatively constant from 6 months to 5 years avoiding the requirement for accurate calculation of age. MUAC of less than 11.5 cm and WHZ of less −3 identify similar proportions of children and are associated with similar risks of mortality.

In the most basic settings where no measurements are possible, diagnosis is based on the presence of visible signs of severe wasting and nutritional oedema. There are two well-recognized malnutrition syndromes. Children with marasmus (see Figure 2) have severe muscle wasting and minimal adipose tissue; they are often noted to be irritable. Children with kwashiorkor (see Figure 3) present with oedema and may show other classical features including dermatitis, sparse depigmented hair and hepatomegaly; they are typically described as apathetic. Nutritional oedema (i.e. pitting oedema of both feet with no identifiable cause such as nephrosis) increases weight and, therefore, may result in a misleadingly high WHZ score. Many children present with clinical features of both syndromes.

Whichever diagnostic criteria are used, they must be applied consistently. All children who present to a health facility, whatever the reason, should have their nutritional status assessed. If treatment is prescribed for the presenting condition but there is a failure to identify and address underlying malnutrition then an opportunity to reduce long-term mortality has been lost.

Figure 3 Child with kwashiorkor (lower limb oedema, sparse depigmented hair, dermatitis with areas of hypo- and hyperpigmentation, angular stomatitis).

Aetiology and pathophysiology

Impaired growth may result from a combination of inadequate nutrient intake, increased losses (diarrhoeal episodes, vomiting) and increased energy expenditure (usually due to infections). Kwashiorkor is from the Ghanaian Kwa language meaning 'the deposed child' and relates to the child being displaced from the breast by a newborn sibling. Indeed, malnutrition often presents around the time of weaning. This is a critical period when breast milk no longer provides adequate calories for growth but weaning foods may be nutritionally incomplete and also a source of infection — especially enteric infections. It is usually difficult to determine the exact sequence of events in any one child; rather, malnutrition and infection appear to exist in a vicious circle with each increasing susceptibility to the other.

Figure 2 Child with marasmus (marked wasting, prominent ribs, increased axillary skin folds, 'old man' face).

Several specific pathological mechanisms have been identified in malnourished children. Even in the absence of overt infection, micro-organisms may still play a crucial role. In unhygienic environments, it is postulated that small bowel bacterial overgrowth leads to a T-cell mediated enteropathy with variable degrees of villous atrophy and crypt hyperplasia. This enteropathy impairs nutrient digestion and absorption and, as a result of increased mucosal permeability, may result in persistent stimulation of the systemic inflammatory response and sepsis from bacterial translocation.

Although the nutritional oedema of kwashiorkor was first described in the 1930s, the underlying pathological mechanism is still not fully understood. The long held assumption was that children with kwashiorkor had a disproportionate lack of protein in their diet resulting in lower plasma albumin concentrations and reduced oncotic pressure. This caused fluid leak into the interstitium and the contracted intravascular volume triggered salt and water retention by the kidney, so further worsening the oedema. However, the diets of children with marasmus and kwashiorkor have not been found to be significantly different and a low protein diet has not consistently produced a kwashiorkor-like syndrome in animal models.