
Reviews/Analyses

Why have mortality rates for severe malnutrition remained so high?

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A review of the literature that has appeared over the past five decades indicates that the median case fatality from severe malnutrition has remained unchanged over this period and is typically 20–30%, with the highest levels (50–60%) being among those with oedematous malnutrition.

A likely cause of this continuing high mortality is faulty case-management. A survey of treatment centres worldwide (n = 79) showed that for acutely ill children, inappropriate diets that are high in protein, energy and sodium and low in micronutrients are commonplace. Practices that could have fatal consequences, such as prescribing diuretics for oedema, were found to be widespread. Evidence of outmoded and conflicting teaching manuals also emerged. Since low mortality levels from malnutrition can be achieved using appropriate treatment regimens, updated treatment guidelines, which are practical and prescriptive rather than descriptive, need to be implemented as part of a comprehensive training programme.

Introduction

Protein–energy malnutrition (PEM) is a multi-deficiency state that includes a range of conditions, the most severe forms of which are marasmus, kwashiorkor, and marasmic kwashiorkor. The seminal investigation of PEM is widely regarded to be the work carried out by Williams in the 1930s; she identified oedematous malnutrition in the Gold Coast in 1933 and subsequently in 1935 introduced the term “kwashiorkor”.

In the 1950s the United Nations and its agencies became involved in evaluating the extent of the problem, and PEM became the subject of worldwide study. Since then, considerable resources have been directed towards improving the treatment of those suffering from this serious and complicated condition; despite this, for those who receive treatment, reported case-fatality rates currently range from 4.4% to 49.0%, with even higher rates for oedematous cases.

In May 1993 at a meeting attended by representatives from WHO, UNICEF, and Save the Children Fund (SCF), together with senior academics

and experienced doctors, it was considered what action, if any, should be taken to combat the continuing high case fatality associated with PEM. It was recommended that training should be improved and, in order to identify specific training requirements, that it would be helpful to have more information on current case-management practices and to review more fully the evidence for high case fatality. The present review article examines such evidence and seeks to explain why mortality remains high, by showing that outmoded and faulty case-management of PEM is widespread.

Methods

Evidence on case fatality from PEM was gathered by reviewing the literature that has appeared over almost the past 50 years. This resulted in 64 sets of data (1–62) to which three unpublished sets were added.^{a–c} Information on case-management was

^a Padlan AA. *A study of malnourished children in the National Children's Hospital, Quezon City, Philippines*. Dissertation, University of West Indies, Kingston, Jamaica, 1969.

^b Mohammed MA. *Evaluation of current strategies in the management of protein energy malnutrition among children under five years old, Balbala Hospital (Djibouti)*. MSc dissertation, London School of Hygiene and Tropical Medicine, University of London, 1993.

^c Pettifor J. University of Witwatersrand, unpublished data, 1993.

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gathered from two postal questionnaire surveys conducted from November 1993 onwards. The first questionnaire surveyed current practices in hospitals and nutrition rehabilitation units worldwide, asking for information about the treatment of severe PEM. Questionnaires were sent to the national offices of UNICEF and SCF for local distribution, and to hospitals and rehabilitation units known to us or colleagues. If severe PEM was common in their area, senior health personnel were asked to complete the questionnaire, and 79 responses were received.

The second survey provided anecdotal evidence of faulty practice. A simple questionnaire, designed to elicit information on specific clinical and dietary practices, was sent to experts who travel widely and observe treatment practice during the course of their work. Respondents were also invited to name the countries where they had observed faulty practices.

Results

Evidence of high case fatality from PEM

Table 1 provides case-fatality data covering the period from the 1950s to the 1990s collected from 67 hospitals and rehabilitation units treating children with severe PEM. Currently, the median case fatality rate is 23.5%, higher than the level for the 1950s. The evidence is striking. In no way does it reflect understanding about the etiology and pathophysiology of the condition gained in the 1950s and 1960s, nor more recent advances. Furthermore, it highlights the widespread failure to use treatments that have been known for many years.

Half of the sources examined also provided data disaggregated according to clinical diagnosis (Table 2), and for kwashiorkor and marasmic kwashiorkor, a higher proportion of children died. Even in the 1990s, case fatalities of 50–60% were reported for children with oedematous malnutrition. It is unlikely that these high rates are linked with acquired immunodeficiency syndrome (AIDS). Furthermore, wasting is more common than oedema in malnutrition that is secondary to AIDS. At Balbala Hospital in Djibouti,^d mortality was 12% among children with acute respiratory infections or gastroenteritis, but when children admitted with these infections had the added complication of PEM, death rates rose to nearly 25%, emphasizing the need to improve the treatment of PEM when it is associated with other conditions.

Table 1: Median case fatality and mortality range among children treated for severe protein–energy malnutrition, by decade^a

	No. of studies	Median case fatality (%)
1950s	7	20.0 (9.0–50.0) ^b
1960s	24	25.7 (11.0–52.5)
1970s	6	24.5 (16.0–37.0)
1980s	20	13.7 (3.3–51.0)
1990s	10	23.5 (4.4–49.0)

^a Sources: ref. (1–62) and footnotes a–c, p. 223.

^b Figures in parentheses are the range.

Table 2: Median case fatality from protein–energy malnutrition, according to diagnosis, 1980s and 1990s

Diagnosis	No. of studies	Median case fatality (%)
<i>1980s</i>		
Kwashiorkor	12	30.1 (4.3–50.0) ^a
Marasmic kwashiorkor	9	32.3 (3.7–43.4)
Marasmus	10	24.0 (3.0–36.5)
<i>1990s</i>		
Kwashiorkor	6	24.0 (6.7–50.0)
Marasmic kwashiorkor	5	30.0 (5.3–60.8)
Marasmus	6	19.6 (6.8–35.0)

^a Figures in parentheses are the range.

Evidence of outmoded practice in treatment centres

Evidence of outmoded practice has been drawn from the 79 survey responses. In particular, information was sought on dietary management, initial antibiotic therapy, and treatment of dehydration. The current view is that early dietary management should involve restricted protein and energy intake (63). Daily consumption of 1–2g protein/kg body weight and 80–120kcal/kg body weight is recommended. Higher intakes are inappropriate and may be dangerous in the first few days of treatment because the malnourished child would be unable to deal with the extra metabolic stress involved. Carefully controlled amounts of protein and energy, given frequently throughout the day and night, are therefore needed to avoid overloading the heart, kidney, and intestine. Table 3 shows that for both protein and energy, over 50% of the institutions surveyed failed to administer amounts within the ideal ranges. This global figure, however, conceals regional trends of apparently better practice in Latin America, with Africa and Asia falling consistently behind. Scherbaum has concluded that therapeutic feeding has been predominantly influenced by the “protein dogma” of the past, i.e., a belief in the need for a high protein

^d See footnote b, p. 223.

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Table 3: Distribution of institutions, by region, according to their protein and energy targets for the first week of treatment for protein-energy malnutrition

	% of institutions:			
	All regions (n = 79)	Asia (n = 23)	Africa (n = 23)	Latin America (n = 18)
<i>Protein (g/kg/day)</i>				
1-2	42	26	31	83
3-4	30	52	29	6
>5	12	9	16	6
Unspecified	16	13	24	5
<i>Energy (kcal/kg/day)</i>				
<80	9	13	3	17
80-119	35	26	29	61
120-149	23	22	29	17
150-200	17	26	18	—
Unspecified	16	13	21	5

diet, with no consideration of recent scientific knowledge.^e

All severely malnourished children have potassium and magnesium deficiencies that adversely affect a number of metabolic functions, including fluid and electrolyte balance. In the absence of magnesium, potassium repletion is impaired. It is therefore recommended that supplements of both potassium and magnesium be given routinely. Table 4 shows that less than half of the centres surveyed gave potassium supplements and only 26% gave magnesium. Copper and zinc play an important role as cofactors in the scavenging of free radicals, thus preventing lipid peroxidation and cell membrane damage; copper and zinc supplements were, however, provided by only 14% and 25% of the centres, respectively (Table 4). Dietary intake of free iron catalytically generates highly reactive free radicals; although it is recommended that iron supplements are withheld in the initial phase of treatment, 31% of centres did not do so.

Multivitamin supplementation, which is important for the restoration of depleted tissues, was provided by 79% of the centres; folic acid and vitamin A were given by 60% and 66%, respectively. Disaggregation of the supplementation results by region indicated a similar pattern to that of protein and energy, with Latin America appearing to have consistently better practices than Asia or Africa.

Broad-spectrum antibiotics should be administered routinely on admission to severe cases of PEM, but were provided by only 24% of the study centres;

^e **Scherbaum V.** *Management and rehabilitation of protein energy malnutrition in Ethiopia.* MSc dissertation, Institute of Child Health, University of London, 1992.

regional proportions were 4% in Asia, 32% in Africa, and 33% in Latin America.

Rehydration is often a necessary initial step in the treatment of severe PEM. The type of oral rehydration fluid is important, since solutions with a high sodium content may induce heart failure (64). Respondents were asked which rehydration fluid they used; the high sodium WHO/UNICEF fluid was employed by 84%, 74%, and 56% of centres in Africa, Asia, and Latin America, respectively (Table 5). This is contrary to current medical opinion which recommends a modified, lower sodium content solution (64). Use of routine intravenous rehydration was high in Latin America (22% of centres), contrary to recommendations (63).

Anecdotal evidence of faulty practice

The results of the small survey to gather information on faulty case-management are given in Table 6. The data were collected by 27 expert practitioners with field experience of severe PEM in 34 countries. In support of Scherbaum's evidence, 56% of the experts reported that kwashiorkor was viewed only as protein deficiency, and that this belief was the basis for prescribing very high protein diets. Between 11% and 56% of respondents reported practices such as

Table 4: Distribution of institutions that gave dietary supplements, by region

Supplement	% of institutions in:			
	All regions (n = 79)	Asia (n = 23)	Africa (n = 23)	Latin America (n = 18)
Potassium	48	35	47	61
Magnesium	26	22	18	44
Copper	14	9	3	44
Zinc	25	30	5	56
Multivitamin	79	74	74	89
Folic acid	60	52	61	61
Vitamin A	66	83	53	67
Iron: week 1	31	30	39	11
Iron: week 2	49	52	37	67

Table 5: Distribution of the type of rehydration fluid administered, by region

Type of fluid:	% of institutions in:			
	All regions (n = 79)	Asia (n = 23)	Africa (n = 23)	Latin America (n = 18)
Intravenous	5	—	—	22
WHO/UNICEF oral rehydration solution	75	74	84	56
Other	20	26	16	22

Table 6: Distribution of faulty practices reported by the expert practitioners

Faulty practice	% of practitioners (n = 27)
Diuretics given to treat oedema	41
Specific treatment for "acute phase" not recognized	41
Kwashiorkor regarded as protein deficiency—high protein diet given immediately	56
High dose vitamin A not routinely given even where deficiency exists	37
Anaemia treated with iron from admission	56
Intravenous albumin/amino acids given	11
Food not measured	61
No feeding at night	41
No blanket provided even where hypothermia is a risk	33

failure to feed and keep the child warm at night, which could have serious consequences including death.

Discussion

The evidence for the continuing high mortality from PEM and widespread faulty practice is clear. Although there has been a global decline in the overall prevalence of PEM (65) associated with improved levels of primary health care and community awareness, there has been no concomitant improvement in survival among those who develop severe PEM.

The unacceptably high mortality levels that we have reported here may even have been underestimated. For example, in some hospitals, especially in Africa, parents may take home a dying child. Furthermore, no attempt was made to obtain a representative sample of institutions, and it is likely that the respondents represent a greater proportion of the better managed, rather than the poorer managed centres.

Low mortality from PEM is certainly attainable. Of the 67 data sets examined, 15% had mortality levels below 10%, the lowest being 3.3%. Such reports, although few in number, are important because they highlight the feasibility of significantly reducing mortality associated with severe PEM. In Bangladesh, at the Children's Nutrition Unit, Dhaka, case fatality fell from 20% to 5% over a 4-year period (66). No change in severity on admission occurred and the reduction in mortality was ascribed to the following modifications in case-management: routine prescribing of broad-spectrum antibiotics on admission; transfusion of packed cells for severe anaemia; with-

holding iron supplements in the first week of treatment; avoiding intravenous rehydration whenever possible; cautious refeeding; use of a low sodium diet; and daily monitoring for signs of fluid overload.

Examination of the regimens on a worldwide basis indicated that better practice appeared to be more widespread in Latin America. The starting points for the distribution of questionnaires in Latin America included rehabilitation units with an international reputation for success in research into the treatment of PEM. Hence the regional difference observed for Latin America may therefore have arisen because of sample bias.

There is some evidence from Africa, however, that increased impoverishment in hospitals results in a failure to provide essential medical supplies and clinical services that could limit good practice (67; and A. Burgess, personal communication, 1995)

Notwithstanding these reservations, a major cause of high PEM case fatality is probably faulty practice as a result of inadequate knowledge; it is therefore appropriate to improve the training methods for medical practitioners and the information they are given. All the widely available guidelines have been produced by WHO (68, 69) and nongovernmental organizations including Oxfam (70) and SCF (71). The guidelines differ in their clarity, foundation on current knowledge, and instructions for treatment. A definitive set of user-friendly, practical guidelines is lacking. Although such an absence cannot be construed as a direct cause of the high case fatality, it is plausible that it may contribute to the problem. There is an urgent need for clear, accessible and authoritative information that is prescriptive rather than descriptive in order to aid rapid and appropriate decision-making.

The knowledge of medical and paramedical workers could be improved through short-term training courses and in the longer term by updating and modifying courses and curricula in tertiary education institutions. In parallel with this, institutions could review their own clinical management procedures and performance with a view to improving practice. The process must start with the preparation of educational materials and simple, clear field guidelines based on the experience of successful, existent regimens. WHO and UNICEF are contributing to this process through their programme Integrated Management of the Sick Child, in which referral criteria, technical guidelines and training materials are being developed for the treatment of common childhood illnesses (diarrhoea, acute respiratory infections, measles,

malaria, and malnutrition). Improved case-management has the potential to avert approximately 0.5 million malnutrition-related deaths annually; hence there is an urgent need for progress to be made.

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Résumé

Pourquoi les taux de mortalité par malnutrition sévère sont-ils toujours aussi élevés?

Cet article expose les résultats de recherches visant à mettre en évidence la forte mortalité associée à la malnutrition sévère et à en chercher les explications dans une prise en charge inadaptée. Les taux de létalité ont été tirés de 65 séries de données, et les renseignements sur la prise en charge des cas de deux enquêtes par questionnaire postal réalisées en 1993. La première enquête portait sur les méthodes de traitement actuellement appliquées dans les hôpitaux et centres de réadaptation partout dans le monde. La deuxième a permis de recueillir auprès de 27 experts spécialisés dans le travail sur le terrain des témoignages sur les pratiques inadaptées en matière de prise en charge des cas.

Un examen des articles publiés a fait ressortir un taux de létalité médian de 23,5% dans les années 90, c'est-à-dire plus que dans les années 50. Si l'on examinait les distributions de fréquence par décennies, ce taux était généralement de 20 à 30%. Les taux observés actuellement vont de 4,4% à 49,0%. D'après les études faisant une distinction suivant le diagnostic clinique, une plus forte proportion d'enfants (50 à 60%) décèdent de kwashiorkor et de kwashiorkor avec marasme. Dans les années 90, les taux de létalité médians pour ces deux affections étaient respectivement de 24% et 30%, contre 19,6% pour le marasme seul. L'enquête sur les centres de traitement montre que les pratiques inadaptées voire dangereuses en matière d'alimentation sont répandues. Chez les enfants gravement atteints, il est fréquent de voir des régimes alimentaires riches en protéines, en énergie et en sodium

et pauvres en potassium, en magnésium et en micronutriments. Dans l'enquête sur les erreurs observées, 11 à 56% des répondants signalaient des traitements susceptibles d'avoir des conséquences graves et même mortelles.

L'absence de directives formelles sur le traitement de la malnutrition protéino-énergétique n'est pas directement la cause des taux de létalité élevés, mais s'il existe par ailleurs des instructions peu claires, il se peut que ce facteur joue un rôle. Un examen de manuels récents montre que les instructions sont d'une sécurité, d'une clarté et d'une cohérence variables, par exemple en ce qui concerne l'administration de fer et d'antibiotiques.

Bien que la mortalité soit dans l'ensemble toujours élevée et que les pratiques inadaptées soient très répandues, nous avons trouvé une mortalité faible (taux de létalité 10%) dans 15% des études examinées. Ces résultats sont importants car ils montrent qu'il est possible de réduire fortement la mortalité par malnutrition protéino-énergétique sévère. Il semble que la cause principale des taux de létalité élevés associés à cette affection réside dans l'application de pratiques inadaptées; il est par conséquent nécessaire que tout programme complet de formation dispose de ressources suffisantes et de manuels pratiques de traitement clairs et à jour.

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