Measles and the State of Nutrition

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SUMMARY
The severity of an attack of measles is largely determined by the underlying state of nutrition at the time of the attack. Evidence is presented which suggests that, conversely, measles may be responsible for the precipitation of malnutrition in undernourished children, by a combination of several different mechanisms.


The severity of measles in undernourished children has been well recognized in recent years. It has also been accepted that the difference in mortality and morbidity from measles between patients living in a tropical climate and those living in temperate climates is based on differences in underlying nutritional status, and is not caused by a more virulent strain of measles virus in the tropics. The possibility that measles may precipitate well-fed and underweight children into a state of malnutrition is not so well documented, and little has been written about the mechanisms which might bring this about.

Creighton first drew attention to what was probably kwashiorkor in English children when he described the effects of a measles epidemic. Numbers (of children) who recovered from measles were afterwards affected with debility . . . and oedematous swellings of the face and extremities, which were very difficult to remove. From West Africa, Gans and Murphy both reported that admissions to hospital for kwashiorkor were more common 2-3 months after an epidemic of measles. There was a recent history of measles in 50% of children with kwashiorkor in Uganda, and in 30% of children in Rhodesia. An average acute weight loss of 500 g during the week of active measles has been reported by Scrimshaw et al., while Morley et al. showed that the mean length of time taken to regain the weight lost during an attack of measles was 7 weeks. It was more significant that, after 3 months, 15% of children had failed to regain their former weight. Poskitt measured serum albumin levels before, during, and 6 weeks after measles in children with suboptimal nutrition. Mean albumin levels fell from 3.34 g/100 ml before the onset of measles to a mean of 2.98 g/100 ml during the acute illness, a difference which is statistically significant. Six weeks after recovery, albumin levels had returned to normal.

MECHANISMS BY WHICH MEASLES MIGHT AFFECT NUTRITIONAL STATE
There are many possible mechanisms by which measles might exert an effect on nutritional state:

Increased Needs for Protein and Energy
Any disease which is accompanied by fever increases the body's need for protein and energy. Additional protein is presumably necessary for the repair of epithelial surfaces damaged during measles. If these needs are not met at the time of the illness, or soon after, there would be weight loss, or at least failure to gain weight.

Decreased Intake of Food
Many illnesses, including measles, are accompanied by anorexia, and the mouth ulcers so frequently present during and after measles would compound the feeding difficulties. In some cultures protein is traditionally eliminated from the diet of children with measles.

The Effect of Measles on the Alimentary Tract
Measles virus infection affects most tissues of the body, and the alimentary tract is no exception. Sydenham, in 1674, noted diarrhoea in association with measles, while 3 recent reports from developing countries showed that 20-40% of children with measles had had diarrhoea at some time during, or immediately after, their illness. Visible blood was present in the stools of 13% of children in Nigeria, while microscopical examination showed red blood cells in the stools of 80% of malnourished children with measles in Rhodesia.

The onset of the diarrhoea is variously reported to be during the prodrome, coinciding with the period of maximum skin desquamation, or up to 14 days after the appearance of the measles rash. Evidence for the direct involvement of the gut by measles comes from reports of multinucleate giant cells in the intestinal lymphoid tissue, in jejunal biopsy specimens, and in the stools. The site of the blood loss is unlikely to be the small bowel, since intestinal specimens from a series of malnourished children, taken during the 4 days immediately after the appearance of the measles rash, failed to show more than the known changes associated with malnutrition.

Koplik spots in the colon and ileocolitis have both been reported, suggesting that the diarrhoea and red cell loss may originate from the colon. Red, friable, rectal mucosa was seen in 9 out of 14 children on whom proctoscopy was performed within 2 days of the onset of the measles rash, and rectosigmoid biopsy specimens...
taken from these same children showed vascular congestion in 12, and superficial ulceration in 3.

Lactose intolerance, precipitated by measles, was first reported by Carpentier et al. in another study, lactase, maltase and sucrase levels in jejunal biopsy specimens from malnourished children with measles were significantly lower than the levels in equally-malnourished children without measles. Short periods of stress or starvation are known to affect intestinal enzyme levels, and it may be through this mechanism that measles has its effect, rather than by direct action on the small-bowel mucosa. Dossetor and Whittle performed xylose absorption tests on 11 children with acute measles, and repeated the tests 5-22 weeks later, by which time a statistically significant improvement had occurred. However, the majority of the children whom they tested were malnourished, and improvement in xylose absorption is known to occur within a few weeks of starting nutritional rehabilitation. It is possible, therefore, at least during the acute phase of measles, that malabsorption may contribute to the precipitation of malnutrition.

Faecal protein loss during acute measles has been estimated, using intravenous Fe-labelled iron dextran, and intravenous "chromium chloride." In the first study, the mean daily loss of albumin in the stool was 1.7 g. Retesting after recovery showed considerable improvement, with albumin loss reduced to almost normal values. In the second study, albumin loss was estimated in 10 malnourished children during the 4 days immediately after the onset of the rash of measles, and in 6 control children with moderate-to-severe kwashiorkor. The children with measles had an estimated albumin loss equivalent in magnitude to the losses reported in moderate protein-losing enteropathy. The children with kwashiorkor themselves had an albumin loss that was slightly, but not significantly, greater than normal adult values. The results of these two studies suggest that protein loss from the gut during measles may reach significant levels, and contribute to the fall in serum albumin previously reported. Dossetor and Whittle calculated that in the acute phase of measles, up to 20% of protein intake may be lost in the stool, assuming normal intake (which is unlikely, because of anorexia).

The histological changes described in the rectosigmoid colon would point to this area as the source of protein loss, rather than the small bowel.

DISCUSSION

The evidence presented strongly suggests that, at least in undernourished children, an attack of measles can precipitate overt malnutrition by a combination of the following: an increased need for protein and energy, a decrease in the intake of food through anorexia, and through direct or indirect effects on the small and large bowel.

In well-nourished children, who receive an adequate diet during the attack of measles, the effects are probably minimal and transient. In the undernourished, who may be inadequately fed during the acute phase of the disease, and who may return to a diet with marginal amounts of energy and protein after the illness, the effects are more prolonged. A diet which was just adequate to allow growth before the illness, may become inadequate during the recovery phase, for the child needs protein and energy for the repair of damaged tissues and protein lost from the body, in addition to the normal requirements for growth.

Measles is thus one of the many diseases which can initiate, or sustain, the vicious circle of malnutrition and infection which is the major problem in children under 5 years old in all developing countries.

REFERENCES