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SHORT REPORTS

Persistent intestinal protein loss after measles

Measles is one of the commonest precipitating factors in kwashiorkor.¹ There is appreciable intestinal protein loss during acute measles infection in underweight children with diarrhoea,23 and I have investigated the possibility that if this persists it might cause kwashiorkor.

Patients, methods, and results

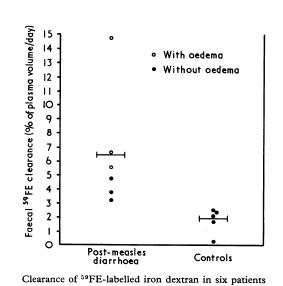
Six children with persistent diarrhoea after measles were studied. The history of measles was confirmed either by documentation during the acute illness or by post-measles skin staining on examination. None had diarrhoea before the onset of measles. Their ages ranged from 20 months to 9 years (mean age 38 months). All weighed less than 80 % of the Harvard standard growth curve (mean value 59 \pm SD 10%). Their mean serum albumin level was 23 ± 4 g/l. Three of the children had developed oedema since the acute illness. Stool culture grew no important pathogens. One child had ova of *Schistosoma mansoni* in the stool. Controls were five children who had recovered from measles but were being reinvestigated after intestinal protein loss had been found during acute measles, as described previously.2 Their mean age was 30 ± 13 months, and their mean weight $81 \pm 15\%$ of the Harvard standard growth curve. ⁵⁹FE-labelled iron dextran (⁵⁹FE) was used to measure intestinal protein

loss. A dose of $0.1 \,\mu \text{Ci/kg}$ was injected intravenously, and all stools over the next three days were collected. Plasma was sampled daily. The faecal clearance of 5% FE thus calculated correlated closely with plasma protein loss into the gut.⁴ With the assumption that albumin is cleared similarly to ⁵⁹FE,⁴ the absolute albumin loss was estimated from the faecal ⁵⁹FE clearance and calculated total intravascular albumin pool. The mean faecal clearance in the patients with post-measles diarrhoea was $6.5 \pm 4.2\%$ of the plasma volume daily. This was significantly higher than in the controls, whose clearance was 2.0 ± 0.9 % of the plasma volume daily (t = 2.6; P < 0.05). Those with oedema had a greater clearance than those without oedema (see figure). The mean absolute albumin loss was significantly (P < 0.05) greater in the patients with post-measles diarrhoea $(0.9 \pm 0.7 \text{ g/day})$ than in the controls $(0.4 \pm 0.3 \text{ g/day})$.

Discussion

The patients with post-measles diarrhoea continued to lose protein in the stool two to four weeks after measles, having a similar ⁵⁹FE clearance to that found in acute measles² and a significantly higher clearance than children who had recovered from measles.

Kwashiorkor developed in the three patients with the highest protein losses. The mean absolute albumin loss in all the patients with post-measles diarrhoea was almost 1 g daily-an important loss, since their mean total intravascular albumin pool was only 13 ± 4 g. The mean loss of 0.4 g albumin daily in the controls was from a mean total intravascular albumin pool of 24 ± 7 g. Shukry et al⁵ found that children with kwaskiorkor lost little protein in the stool in the absence



with post-measles diarrhoea and five controls (t=2.6; P < 0.05).

of diarrhoea, and our unpublished results confirm this. Thus the protein loss in the patients with post-measles diarrhoea was related to the diarrhoea and not to the poor state of nutrition.

We do not know whether measles virus has any specific role in the syndrome of post-measles diarrhoea. Superinfection by bacteria or fungi in the large bowel or small bowel is a possible explanation, since secondary infections after measles are common in other tissues. Possibly underweight children with diarrhoea from any cause may lose similar amounts of protein to the children in this study.

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