very detailed search for an aetiology was made but no cause could be found except for the fact that both the infants were born to mothers who came from south eastern Saudi Arabia where the diet is very deficient in vitamins, and both mothers had clinical evidence of panhypovitaminosis.

We think that this congenital abnormality in the infants may be due to chronic maternal riboflavine deficiency, as reported by Warkany and Nelson (1941) in experimental animals. Could I, through your columns, ask other paediatricians if they have come across this abnormality, and, if so, what was its aetiology?

References

- Warkany, J., and Nelson, R. C. (1941). Skeletal abnormalities in offspring of rats reared on deficient diets. *Anatomical Record*, **79**, 83–93.

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Acute inflammatory bowel disease in childhood: a new disease?

Sir,

We should like to report on the subsequent progress of the child described as Case 1 in the paper by Manuel *et al.* (*Archives*, 1978, **53**, 642).

This 6-year-old boy made a complete recovery after his initial illness and remained well for the next 2½ years. He then had an acute episode of diarrhoea and vomiting requiring no specific treatment, but the anorexia and diarrhoea persisted for 2 weeks. Six weeks later he became constipated. This did not respond to treatment with laxatives nor did it respond to repeated enemata over the next 4 months. He also began to have abdominal

pains, which were thought to be due to his severe constipation, and he developed painful anal fissures.

Physical examination at this time showed no abnormality apart from a rather scarred anus with two fissures, but colonoscopy revealed scattered aphthoid ulcers throughout the colon characteristic of Crohn's disease, and an ulcerated terminal ileum.

The typical noncaseating granulomata, found in Crohn's disease, were present in the biopsies from the terminal ileum, confirming this diagnosis. He was started on sulphasalazine. He responded satisfactorily, with return of his stools to normal, disappearance of his abdominal pain, and healing of the anal fissures. He has since remained well.

It is interesting to speculate what relationship the initial illness reported in the *Archives* may have had to the subsequent one. Was this an acute presentation of Crohn's disease? This was considered at that time as it is rare to have a diarrhoeal illness of such severity in a child 6 years of age in this country. However at that time none of the usual diagnostic features of Crohn's disease was present—namely the barium follow-through examination did not suggest Crohn's disease, and the abnormality on small intestinal biopsy was severe but nonspecific, with an acute inflammatory cell reaction. Was the initial illness in fact a severe attack of acute gastroenteritis which, in some way, has precipitated the development of Crohn's disease in a susceptible individual? The answer to these questions is uncertain, and it remains quite possible that the two illnesses were unrelated. Nevertheless this case illustrates that although spontaneous resolutions can occur in acute inflammatory bowel disease for which no cause has been found, the eventual diagnosis of chronic inflammatory bowel disease is not thereby excluded, and long-term follow-up is necessary in children in whom this diagnosis is made.

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