

Case report

A 3 month old girl was referred to our unit because of recurrent life threatening events reported to have received mouth-to-mouth resuscitation. In one episode, bleeding from the nose and mouth was noted. Investigations in the referring hospital had failed to reveal a cause for these episodes.

All episodes involving resuscitation had begun in the presence of the mother only. She had given inconsistent histories to various carers. A 2 year old sibling had frequent attendances at the GP's surgery with minor illnesses, had developmental delay, and was being cared for by the maternal grandmother because she had marks possibly due to cigarette burns.

In hospital, a further life threatening event occurred and the multichannel physiological recordings showed a pattern suggestive of suffocation. Furthermore, nursing staff noted the mother to handle the child roughly and swear at her. Through the social services, a multi-agency planning meeting was held and a decision to institute covert video surveillance was made in order to examine the suspicion that the child's events were due to suffocation.

Under surveillance, the mother tampered with the recording equipment and tore up nursing records. After a period of physically rough play, the mother calmly and forcibly bent the child's arm backwards at the elbow. The baby screamed with pain and the mother pressed the alarm button. She reported to staff that the baby's arm had been caught in the cot side when she lifted the baby from the cot. Radiography confirmed a transverse fracture at the proximal end of the radius and ulnar of the left arm.

The mother was convicted and the children placed in care. No further life threatening events occurred. The siblings' development improved and progressed normally.

Even under close supervision in hospital, non-accidental injury may occur. Consideration should still be given to the possibility of non-accidental injury when fractures occur in hospital.

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Are cyclospora an important cause of diarrhoea in Bangladesh?

EDITOR,—Alam and colleagues have shown quite convincingly in their classical (double blind, randomised, placebo controlled) trial that co-trimoxazole is clinically beneficial in the treatment of Bangladeshi infants with persistent diarrhoea.¹ This is an important finding as chronic diarrhoea may be responsible for a third of all deaths in rural Bangladeshi children under 5 years old.²

In the study of Alam *et al* initial stool cultures were negative for *Vibrio cholerae*, salmonella, shigella, and as well, microscopic stool examination was negative for *Entamoeba histolytica*/*Giardia lamblia*. However, no mention is made of whether cyclospora were looked for or found. This parasite may cause chronic diarrhoea and is highly susceptible to treatment with co-trimoxazole.³ A modified acid-fast stain is required to detect the organism.

In a Nepali study where children with

tachypnoea or respiratory distress (and presumed pneumonia) were managed by village based health workers who dispensed co-trimoxazole, there was a major reduction in relative risk of death which was even greater in those children with diarrhoea, alone or in combination with pneumonia (36%; 95% confidence interval (CI) 23% to 48%), than in those with pneumonia alone (30%; 95% CI 3% to 50%).⁴ It has already been suggested that in the Nepali study, the treatment of children with co-trimoxazole for tachypnoea (presumed pneumonia) may serendipitously also have treated those children with respiratory distress from metabolic acidosis secondary to chronic diarrhoea caused by cyclospora infection.⁵

Could cyclospora be an important pathogen in Bangladeshi children? It would be very interesting to know if active efforts have been made to detect this newly described and seasonal pathogen in Bangladeshi children.

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- 1 Alam NH, Bardham PK, Haider R, Mahalanabis D. Trimethoprim-sulphamethoxazole in the treatment of persistent diarrhoea: a double blind placebo controlled clinical trial. *Arch Dis Child* 1995; 72: 483-6.
- 2 Faveau V, Yunus M, Zaman K, Chakraborty J, Sarder AM. Diarrhoea mortality in rural Bangladeshi children. *J Trop Pediatr* 1991; 37: 31-6.
- 3 Hoge CW, Shlim DR, Ghimire M, *et al*. Placebo-controlled trial of co-trimoxazole for cyclospora infections among travellers and foreign residents in Nepal. *Lancet* 1995; 345: 691-3.
- 4 Pandey MR, Daulaire NMP, Starbuck ES, Houston RM, McPherson K. Reduction in total under-five mortality in western Nepal through community-based antimicrobial treatment of pneumonia. *Lancet* 1991; 338: 993-7.
- 5 Booy R, Tudor-Williams G. Co-trimoxazole for cyclospora infection. *Lancet* 1995; 345: 1303-4.

Dr Alam comments:

The clinical trial of trimethoprim-sulphamethoxazole in the treatment of persistent diarrhoea in Bangladeshi children was not aimed to treat persistent diarrhoea associated with cyclospora infection. So, the presence of cyclospora in the stool was not looked for carefully. The role of cyclospora in the pathogenesis of persistent diarrhoea in infants and children in Bangladesh has not been studied extensively. Recently, six cases of chronic diarrhoea associated with cyclospora infection were reported from Bangladesh.¹ However, all the subjects in this report were above 2 years of age. Our study subjects were below 2 years of age. Thus, cyclospora as a causal agent of persistent diarrhoea in our subjects is uncertain. Nevertheless, the possibility of cyclospora infection in a few cases cannot be ruled out. The suggestion made with reference to the Nepali study² that the treatment of children with co-trimoxazole for tachypnoea (presumed pneumonia) may serendipitously also have treated those children with respiratory distress from metabolic acidosis secondary to chronic diarrhoea caused by cyclospora infection is a speculation. Further epidemiological studies are needed to identify cyclospora as a causative agent associated with persistent diarrhoea in Bangladesh.

- 1 Albert MJ, Kabir I, Azim T, Hossain A, Ansaruzzaman M, Unicomb L. Diarrhea associated with *Cyclospora* sp in Bangladesh. *Diagn Microbiol Infect Dis* 1994; 19: 47-9.

- 2 Pandey MR, Daulaire NMP, Starbuck ES, Houston RM, McPherson K. Reduction in total under-five mortality in western Nepal through community-based antimicrobial treatment of pneumonia. *Lancet* 1991; 338: 993-7.

Aspirin treatment and increased generation of cysteinyl leukotrienes in Kawasaki disease

EDITOR,—We read with great interest the recent article by Dr Mayatepek and Dr Lehmann in which they demonstrate that cysteinyl leukotrienes may be involved in the pathophysiology of Kawasaki disease and leukotriene synthetase inhibition or receptor antagonism may offer a new potential therapeutic approach.¹ Aspirin combined with high intravenous doses of gammaglobulin are presently the most commonly used treatment for Kawasaki disease. Considering that non-steroidal anti-inflammatory drugs, including aspirin, can augment the 5-lipoxygenase pathway by blocking cyclo-oxygenase in some pathological conditions,^{2,3} administration of aspirin may contribute to an increase in leukotriene generation in Kawasaki disease. The authors did not refer to the generation of cysteinyl leukotrienes during aspirin treatment, but examination of this may be necessary in assessing the safety of implementing aspirin treatment in Kawasaki disease.

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- 1 Mayatepek E, Lehmann WD. Increased generation of cysteinyl leukotrienes in Kawasaki disease. *Arch Dis Child* 1995; 72: 526-7.
- 2 Knapp HR, Sladek K, Fitzgerald GA. Increased excretion of leukotriene E₄ during aspirin-induced asthma. *J Lab Clin Med* 1992; 119: 48-51.
- 3 Rainsford KD. Leukotrienes in the pathogenesis of NSAID-induced gastric and intestinal mucosal damage. *Agents Actions* 1993; 39 (special No): C24-6.

Dr Mayatepek comments:

In our study on the role of cysteinyl leukotrienes in Kawasaki disease we examined patients during the acute phase before any treatment. A possible effect of non-steroidal anti-inflammatory drugs, such as aspirin, on cysteinyl leukotriene generation in Kawasaki disease was not the subject of this article. However, it was well known that doses of up to 2.5 g of aspirin had no effect on urinary leukotriene E₄ (LTE₄) excretion.¹ Furthermore, administration of other non-steroidal anti-inflammatory drugs, such as indomethacin, in daily doses of 50 mg also had no effect on the baseline and allergen stimulated LTE₄ excretion in urine.² Performing the above mentioned study, we were able to measure urinary excretion of LTE₄ in three of these patients with Kawasaki disease during aspirin and gammaglobulin treatment and found no increase in leukotriene generation. It seems therefore unlikely that aspirin in the dosage used for treatment in Kawasaki disease may increase generation of cysteinyl leukotrienes in vivo.

- 1 Fauler J, Thon A, Tsikas D, von der Hardt H, Frölich JC. Enhanced synthesis of cysteinyl leukotrienes in juvenile rheumatoid arthritis. *Arthritis Rheum* 1994; 37: 93-7.