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 baby's surgery with no 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 juridically 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.

As cyclospora an important cause of diarrhoea in Bangladesh?

Dr Alan 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 recent 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 below 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 for the presence of cyclospora in the stool is not looked for found. The 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.

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.


Dr Mayatepe comments:

In our study on the role of cyclospora in diarrhoea in our western Nepali study, in 3 of these patients with Kawasaki disease, reduced dosage of gammaglobulin treatment and found no increase in leukocytosis. It seems therefore unlikely that aspirin in the dosage used in our treatment in Kawasaki disease in increase generation of cyclosporin leukotrienes in vivo.


Aspirin treatment and increased generation of cycloxylin leukotrienes in Kawasaki disease

EDITOR,—We read with great interest the recent article by Dr Mayatepe and Dr Lehmann in which they demonstrate that cyclospora leukotrienes may be involved in the pathophysiology of Kawasaki disease and leukotriene synthetase inhibition or receptor antagonism may offer a new potential therapeutically 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 cycloxylin leukotrienes during aspirin treatment, but examination of this may be necessary in assessing the safety of implementing aspirin treatment in Kawasaki disease.

Recruiting patients to clinical trials: lessons from studies of growth hormone treatment in renal failure

EDITOR—We were interested in Professor Campbell’s commentary on our recent paper.1 Many of the important points he raises such as entry criteria, trial design, and patient selection were dealt with in the accompanying paper.2 It would have been helpful if we had made it clear that these two papers were companion papers by including more cross references. Neither report makes it clear that the studies had ethical committee approval and as Professor Campbell raises this point we can confirm that the protocols for both studies had been submitted separately to our local ethics committee and received its approval.

We disagree that ‘some recruitment bias must have been introduced by the rather complex process of obtaining “staged” informed consent’. Our experience was the reverse. Inevitably it does take time to discuss such complicated issues adequately but the process does not need to be very complex: about 50% of our families required only a single interview. Thirty of 31 eligible patients took part in the Psychological study and 28 of 31 eligible patients opted for treatment with growth hormone. Thus it was possible to involve families in the decision without biasing recruitment whilst protecting against families accepting uncritically prior decisions of the professionals.

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Osteogenesis imperfecta, non-accidental injury, and temporary brittle bone disease

EDITOR—The commentary which followed the paper by Smith prompts me to comment.1 The most widely used classification of osteogenesis imperfecta is that of Sillence.2 Type IVA is the type most likely to be confused with inflicted injury. At the time of the first fracture the skeleton may be normal and there are no stigmata of osteogenesis imperfecta. Metaphysal injuries are regarded as specific of inflicted injury in infancy.3 However, Paterson in a study of type IVA osteogenesis imperfecta noted that metaphysal fractures do occur.4 The validity of his work has been both vigorously criticised5 and defended6 and remains controversial.

In the commentaries Hobbs and Wynne and Carry state that the metaphysal fractures of osteogenesis imperfecta do not cause diagnostic confusion as the skeleton will always indicate obvious bone disease. In making that assertion they refer to the work of Astley.7 Their argument is specious. Astley's paper was received by the journal in 1978. The type IVA variant was first described by Sillence in 1979. It is therefore not surprising that Astley concluded ‘in all of these there was generalised bone disease so that confusion with non-accidental injury did not occur’.

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