cell count $8.6 \times 10^9/l$, serum IgA 3.15 g/l (18 months) and 2.65 g/l (20 months), antinuclear antibody titre 1:80. There was no IgG or IgM to Epstein-Barr or cytomegalovirus, no urinary light chains, a normal number and proportion of T and B cells, and normal whole blood proliferation to mitogen. Chest radiography and abdominal ultrasound were normal.

The pathogenesis of the paraprotein in our patient is unclear. The adult entity of benign monoclonal gammopathy has not been described in the paediatric population, and previous reports of monoclonal gammopathy in children have consistently associated such gammopathies with serious disease. Additionally, there are no reports of a monoclonal gammopathy as an incidental finding in an otherwise healthy child. In a 10 year review of 4000 highly selected children, 155 (3.9%) were found to have paraproteinaemia.¹ These children were suffering from primary and secondary immunodeficiency, malignancy, autoimmune disease, asthma, serious infection, and aplastic anaemia. In contrast, none of the 120 healthy paediatric controls used in this study (donors for bone marrow transplantation) had paraproteins identified.

Our case is also unusual in that her paraprotein is of the IgA type, as there are only four reports of IgA monoclonal gammopathy in the paediatric population. Two of these children had a severe combined immunodeficiency,¹ one had an unspecified malignancy,¹ and the other an unspecified chromosomal disorder.² Of the 155 children with monoclonal gammopathy in the 10 year study previously referred to, no IgA paraproteins were identified.

It is possible this abnormal production of IgA is related to a subtle T cell dysregulation. Such dysregulation of T cells has been implicated in another paediatric disorder associated with raised IgA, IgA nephropathy. In this condition abnormal T cell function and signalling is thought to be responsible for the aberrant B cell activity, where respiratory pathogens or common environmental antigens stimulate excessive production of IgA, in the presence of increased helped T cell activity³ and decreased suppressor T cell numbers.⁴

We are following up this infant, with much interest, for any alteration in her IgA paraprotein or immune function, and for the possible development of disease.

D E CAMPBELL
A S KEMP

Department of Immunology,
Royal Children's Hospital,
Parkville, Victoria 3052,
Australia

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Long term follow up to determine the prognostic value of imaging after urinary tract infections

EDITOR,—I read with interest the two papers by Merrick *et al* on the prognostic value of imaging in children with urinary tract infections (UTIs).^{1 2}

It would be helpful if the authors could tell us what proportion of the initial cohort of children with UTI, during 1975 to 1990, underwent initial imaging and later follow up imaging. We have shown that in an area of very good standard for general practice only a small minority of children with UTI had been referred for radiological investigations.³ The fact that this 'denominator' figure is not provided casts doubt on how representative their results are. The true natural history and prognostic value of different radiological abnormalities, further UTIs, etc, can only be determined on a large scale, prospective, hospital and community based study.

LYDIA JADRESIC
Department of Paediatrics,
Gloucestershire Royal Hospital,
Great Western Road,
Gloucester GL1 3NN

- 1 Merrick MV, Notghi A, Chalmers N, Wilkinson AG, Uttley WS. Long term follow up to determine the prognostic value of imaging after urinary tract infections. Part 1: reflux. *Arch Dis Child* 1995; 72: 388-92.
- 2 Merrick MV, Notghi A, Chalmers N, Wilkinson AG, Uttley WS. Long term follow up to determine the prognostic value of imaging after urinary tract infections. Part 2: scarring. *Arch Dis Child* 1995; 72: 393-6.
- 3 Jadresic L, Cartwright K, Cowie N, Witcombe B, Stevens D. Investigation of urinary tract infection in childhood. *BMJ* 1993; 307: 761-4.

EDITOR,—Merrick *et al* recommend micturating cystourethrography (MCU) be restricted to females who have not achieved bladder control.¹ Based on their data that suggest vesicoureteric reflux (VUR) is not a risk factor for progressive renal damage in boys less than 1 year of age, the authors excluded males.² As there is a possibility of type II statistical error with a sample size of only 205 children, basing evaluation strategies on such a small sample size warrants careful scrutiny.

The risk of renal damage due to pyelonephritis is considered greatest in the first few years of life.^{3 4} The most common indication for antibiotic prophylaxis to prevent pyelonephritis in infancy is the presence of VUR. We are concerned that the recommendation by Merrick *et al* might restrict the prescription of antibiotic prophylaxis in infant boys and thereby increase the risk of potentially avoidable episodes of pyelonephritis with attendant renal damage.

The indications for MCU and antibiotic prophylaxis are evolving. Since pyelonephritis and renal scarring can develop in infants who do not have VUR, perhaps an episode of pyelonephritis in infancy justifies antibiotic prophylaxis, regardless of the presence or absence of VUR.

WM LANE M ROBSON
University of South Carolina School of Medicine,
Paediatric Nephrology,
The Children's Hospital,
Greenville Hospital System,
Memorial Medical Office Building,
890 W Faris Road, Suite 250, Box #1,
Greenville, SC 29605-4253, USA

ROBIN KELLEY
Paediatric Infectious Disease,
Greenville Memorial Hospital,
Greenville Hospital System,
Greenville, SC, USA

- 1 Merrick MV, Notghi A, Chalmers N, Wilkinson AG, Uttley WS. Long term follow up to determine the prognostic value of imaging after urinary tract infections. Part 1: reflux. *Arch Dis Child* 1995; 72: 388-92.
- 2 Merrick MV, Notghi A, Chalmers N, Wilkinson AG, Uttley WS. Long term follow up to determine the prognostic value of imaging after urinary tract infections. Part 2: scarring. *Arch Dis Child* 1995; 72: 393-6.

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Dr Uttley comments:

I would like to thank Dr Jadresic for her interest in the two papers by Merrick *et al*. The vast majority of children referred to the Edinburgh hospitals with UTI, either acutely or after treatment, would have undergone imaging of their urinary tract in some form. This has been standard hospital practice and teaching throughout the period of the review. Indeed the need for investigation after a single UTI has been the formal recommendation to undergraduates, postgraduates, and general practitioners throughout. Needless to say we have no information regarding children not referred by their general practitioners and we agree with Dr Jadresic that some patients may not have been referred. Large scale prospective hospital and community based studies are indeed required, and perhaps the impending era of national guidelines and audit will help to answer these thorny questions in the future.

With regard to the letter of Drs Robson and Kelley we do not recommend MCU to be restricted solely to females who have not achieved bladder control. Although it has not emerged as an independent variable for progression of renal disease in young boys, an MCU is clearly an important part of their work-up, particularly after febrile UTI and where abnormalities other than VUR could well be present. However, we do feel the indirect voiding study to be a more sensitive, and a substantially less invasive and traumatic way of assessing the presence or absence of VUR in all children who have achieved bladder control and that this now should be seen as the initial test for reflux.

The data presented reaffirm the importance of reflux as a risk factor for progressive renal damage generally and particularly so when associated with infection.

The fact that VUR did not come out as a risk factor for progressive renal damage in boys under 1 year of age, must be due in part to the fact that this particularly vulnerable group did in general receive appropriate antibiotic prophylaxis. A further issue is that renal damage identified at presentation in these young males will represent renal dysplasia, sometimes profound, and for which evidence of progression was difficult to elucidate.

Overall it would be a pity if debate over appropriate investigation was to overshadow the importance of appropriate clinical follow up and antibiotic treatment and prophylaxis and to that extent we are in total agreement with the final paragraph of Drs Robson and Kelley's letter.

Non-accidental fracture occurring in hospital

EDITOR,—Recent reports have implied that fractures occurring to infants in hospital are due to natural causes, whereas those occurring out of hospital may be due to non-accidental injury. The inference is that fractures occurring within a place of safety cannot be non-accidental. We report a case of a child who sustained a non-accidental fracture of the left radius and ulnar while in hospital.

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.

H KLONNIN
M P SAMUELS
D P SOUTHALL
Academic Department of Paediatrics,
University of Keele,
North Staffordshire Hospital,
Stoke on Trent ST4 6QG

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.

ROBERT BOOY

Department of Paediatrics, Infectious Diseases,
Queen Elizabeth The Queen Mother Wing,
St Mary's Hospital,
South Wharf Road,
London W2 1NY

- 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.
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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.

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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.

TOSHIKAZU SHIMIZU
YUICHIRO YAMASHIRO
KEIJIRO YABUTA

Department of Paediatrics,
University of Fukuoka,
3-1-3, Hongo, Bunkyo-ku, Tokyo, 113,
Japan

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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.

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