Correspondence

Kawasaki disease in Canterbury

Sir,

We should like to report what we believe to be the first recorded case of mucocutaneous lymph node syndrome (MLNS) in a white English child resident in this country.

A 9-year-old boy presented in January 1978 with a severe cough, was diagnosed as having bronchitis and treated with ampicillin by his general practitioner. Four days later, after an apparent recovery, he developed a sore throat and tender cervical lymph nodes. Diarrhoea and vomiting followed 2 days later with some abdominal discomfort. A rash appeared on his hands and feet, spreading to his trunk. He was febrile and 2 days later, when there was no improvement, he was admitted to hospital.

On admission, 9 days after the onset, he was an ill, febrile child (peak temperature 40°C), with strikingly congested conjunctiva and a florid erythematous rash, more marked on the extremities. There was vague abdominal tenderness, and anorexia and vomiting which persisted for some days. His pyrexia persisted despite active measures and on the 3rd day in hospital his liver became palpably enlarged and tender. Because of concern about his fluid intake, intravenous fluids were started. Despite careful monitoring, later the same day he went into cardiac failure with gallop rhythm and pulmonary oedema. This was controlled during the next 24 hours but occasional ectopic beats persisted and his pulse remained high for some days. During the course of the illness he developed a sore strawberry tongue and complained of occasional joint pain although there were no objective signs of joint involvement. On the 5th day, general improvement began, his temperature fell and his rash began to fade. The liver enlargement gradually diminished. On the 9th day in hospital an early systolic murmur was heard for the first time. The hands started desquamating on the 13th day and the feet the day after. After 3 weeks in hospital he was discharged with no remaining signs or symptoms, apart from the murmur. He remains completely well at follow-up 6 months later.

The relevant laboratory investigations were: bilirubin—27 μmol/l (1.6 mg/100 ml); SGOT—46 IU/l; LDH—567 IU/l; electrophoresis of plasma protein—increased α2-globulin; gamma GT—71 IU/l; WBC—25.5×10⁹/l (25 500/mm³); neutrophils—81%; ESR—80 mm in 1st hour; viral studies—negative; ASO titre—normal.

The MLNS was first described in Tokyo (Kawasaki, 1967). In 1972 the Japanese Mucocutaneous Lymph Node Syndrome Research Committee (Tanaka et al., 1976) stipulated that before a diagnosis could be made, a child must have a fever for at least 5 days with no response to antibiotics, and of the following: (1) bilateral congestion of the ocular conjunctiva; (2) changes in the peripheral limbs including indurative peripheral oedema and erythema of the palms and feet, followed later in the illness by membranous desquamation of the fingertips; (3) changes in the lips and mouth including dry red fissured lips, strawberry tongue, and red oral and pharyngeal mucosa; (4) a polymorphonuclear xanthoma of the trunk without crusts or vesicles.

Other findings reported are acute nonpurulent cervical lymphadenopathy, mild diarrhoea, arthritis or arthralgia, proteinuria and pyuria, leucocytosis with neutrophilic shift to the left, increased ESR, and positive C-reactive protein. Evidence of aseptic meningitis, mild jaundice, increased in serum transaminase and α2-globulin, and evidence of carditis have also been described.

Our case fulfilled all these criteria and many of the others.

Previously reported cases in the UK are of a girl of Italian parentage in Nottingham (Hewitt, 1977), a Japanese in Birmingham (Fossard and Thompson, 1977), and 3 West Indians in London (Scopes and Hulse, 1977; Lyen and Brook, 1978). With widespread recognition of the MLNS it seems likely that the diagnosis will be made with increasing frequency in children of all ethnic groups. The differential diagnosis (Kawasaki et al., 1974) includes erythema multiforme, scarlet fever, Stevens-Johnson syndrome, and Still's disease. In the past MLNS may well have been diagnosed as any of these. The prognosis is generally excellent but there is the occasional report of a child meeting a sudden cardiac death due to arteritis of the coronary system (Kegel et al., 1977), making follow-up of cases desirable.

We would like to thank Drs W. J. Appleyard and C. A. Porter for their help in the preparation of this case.

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References


Gentamicin dosage schedules

Sir,

The paper by Yoshioka et al. (Archives, 1978, 53, 334), gives a guide to gentamicin dosage for an age group missed by the popular adult nomograms. Such a guide is necessary when choosing initial dosage, but we believe that subsequent maintenance therapy should be monitored by determining serum gentamicin levels in all patients rather than just in ‘difficult’ cases as suggested by the authors. The disadvantage of determining the half-life of gentamicin from creatinine clearance is that the latter should be corrected to the patient’s lean body mass (Hull and Sarubbi, 1976). The lean body mass of an acutely-ill oedematous child with chronic renal disease is difficult to estimate.

Gentamicin clearance is a sensitive indicator of renal function, and is logically best predicted by measuring serum levels of the drug itself. Sawchuck and Zaske (1976) described a method for calculating the half-life and distribution volume of gentamicin. We have adapted this method for a programmable calculator instead of the computer. Our experience suggests that the one-compartment model used in this method is perhaps too simple: the half-life derived from serum levels of gentamicin in the first 4 hours after a dose overestimates the subsequent rate of clearance and slightly underestimates the interval between doses. A better model could be made by fitting a 2- or 3-term exponential function to later serum levels (Kahlmeter et al., 1978) but the one-compartment model does provide useful insight into the effects of varying dosage regimens.

We question whether a dosage of 1 mg/kg is enough to treat serious infections, especially by IM injection. The peak serum levels achieved in the 3 patients treated by the schedule were all below 5 μg/ml. The longer the dose takes to enter the circulation, the lower the interstitial fluid levels will be (Kozak et al., 1977).

There appears to be no alternative to repeated measurement of serum gentamicin levels, and our efforts should be directed towards improving the accuracy and availability of our assays. Micromethods using small quantities of blood would be particularly valuable for very young children for whom venepuncture is often difficult or impossible. There is one factor in our favour: the interval between doses for many patients with renal failure is longer than our slowest bio-assay.

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References


Dr Yoshioka and co-workers comment:

We welcome the comments from Dr Mayon-White and Miss Perks. Their opinion is acceptable as a principle that serum levels should be monitored in all patients receiving gentamicin treatment. But unfortunately many hospitals do not provide antibiotic assay systems, and even in hospitals in which this service is available, the reproducibility of measured levels is reported to be poor (Reeves and Bywater, 1975). In such a situation we believe proposed nomograms or other pharmacokinetic data are still a useful guide in planning gentamicin treatment.

The statement about correcting the creatinine clearance value to the patient’s lean body mass seems misleading. Obviously, endogenous creatinine clearance is a value which is determined on the basis of the serum level and the amount of creatinine excreted in urine in a unit of time. Rapid evaluations of creatinine clearance or glomerular filtration rate are proposed on the basis of age, body weight, and serum creatinine level for adults (Hull and Sarubbi, 1976), and of body length and plasma creatinine level for children (Schwartz et al., 1976). However, we do not agree with the use of values obtained by these expedients until it is otherwise proved that these values actually correlate with serum half-life of gentamicin. Only values determined in individual patients should be used for our dosage schedule at the present time.

As far as the serum half-life determination of gentamicin is concerned, we do not argue with their statement that it should be done after 4 hours of dosing. However, absorption of gentamicin from the injected site is rapid even in patients with renal insufficiency. Peak levels were attained within 2 hours in all our patients (Table I) after intramuscular dosing, and therefore we do not believe the lower blood level in the 3 patients was due to delayed absorption.

The serum concentration of gentamicin was once reported to be unpredictable (Kaye et al., 1974). But recently it became evident that a series of factors affect the serum concentration. These include obesity of the patient, unstable renal function, haematocrit values, administration of other drugs, or certain disease states of the patient. The accuracy of prediction of gentamicin level was reported to have markedly improved by taking account of these factors (Hull and Sarubbi, 1976). We believe that satisfactory treatment can be achieved, with few exceptions, by using dosage schedules; however, it

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*Arch Dis Child* 1978 53: 916-917
doi: 10.1136/adc.53.11.916

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