

## Mycoplasmal pneumonia preceding Henoch-Schönlein purpura

Sir,

Mycoplasmal pneumonia and Henoch-Schönlein purpura are well-defined clinical entities. We report a case in which the two conditions occurred in probable causal association.

### Case report

An 8-year-old Caucasian boy presented with the history of a cough and a low grade fever for one week. This was followed by painful swelling of the right elbow and left ankle together with central abdominal pain associated with vomiting for one day. On the morning of admission, a nonirritating rash developed on the buttocks and lower limbs. He was a well-nourished boy with an evident, nonspasmodic cough and slight dyspnoea. There was no cyanosis. The blood pressure was normal and the axillary temperature was 37.5 °C. The typical Henoch-Schönlein maculopapular purpuric rash was noted on the buttocks and the flexor aspect of both lower limbs. No rash was evident on the trunk or the upper limbs. Crepitations were heard over the right middle lobe region. There were evident painful swellings of the right elbow and left knee joints. Other systems were normal.

Hb 12.4 g/100 ml; WBC 9600/mm<sup>3</sup>; neutrophils 71%, lymphocytes 24%, monocytes 5% (no atypical cells seen); platelets 240,000/mm<sup>3</sup>, ESR 20 mm/hr. Cold agglutination titre, positive at 1 in 16 at 4 °C.

*Mycoplasma pneumoniae* CFT, >1 in 2560.

Chest x-ray showed a patch of pneumonia at the right hilar region radiating outwards along the course of the bronchovascular markings. Stools for occult blood, positive. The following were normal or negative. Urinalysis, blood urea and electrolytes, throat swabs, bleeding time, prothrombin time, KCT, blood culture, antinuclear factor by fluorescent dye test.

He was treated with oral ampicillin. The abdominal pain, with vomiting and arthritis, resolved within 48 hours. The rash faded within the first week, but a fresh crop appeared in the second week. He continued to have a troublesome cough and the chest signs persisted. He was then started on erythromycin. The second crop of the rash faded after a further week and did not recur. The respiratory signs and symptoms resolved after about 2 weeks. The ESR became normal in 2 weeks. There was no evidence of overt renal involvement during his 2 weeks in hospital, though he had microscopical haematuria on one occasion.

*Mycoplasma pneumoniae* CFT 2 months later was 1 in 160. The repeat chest x-ray was clear. Repeat urine analysis was normal.

### Comment

In pneumonia caused by the organism *Mycoplasma pneumoniae*, the clinical illness may be a result of hypersensitivity to the organism or one or more of its products (Couch, 1971). This could explain the various associated complications of mycoplasmal pneumonia, e.g. urticaria, erythema nodosum, erythema multiforme, Stevens-Johnson syndrome, Guillain-Barré syndrome, and encephalitis, all of which are probably hypersensitivity diseases.

Although Henoch-Schönlein purpura has long been regarded as due to some form of hypersensitivity, the origin of the antigen is rarely apparent. The evidence for drug, food, and other allergies is unconvincing in the majority of cases (*British Medical Journal*, 1971). The infective aetiology has always been an attractive one because the illness is usually preceded by an upper respiratory tract infection (Allen, Diamond, and Howell, 1960; Bywaters, Isdale, and Kempton, 1957). Gairdner (1948) implicated haemolytic streptococci as an aetiological agent, a view which was refuted by Bywaters *et al.* (1957) and Vernier *et al.* (1961).

In this case the clinical pattern of Henoch-Schönlein purpura was obvious. The diagnosis of mycoplasmal pneumonia was first suggested by the prominent cough and the suggestive chest x-ray findings (Mufson *et al.*, 1961), and the serology was also positive. It is not uncommon for the untreated respiratory symptoms to recur (Couch, 1971). Tetracycline and erythromycin are the antibiotics of choice. In this case it did appear likely that the *Mycoplasma pneumoniae* was the aetiological agent for the Henoch-Schönlein purpura.

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## The 'grey toddler': chloramphenicol toxicity

Sir,

I read the article by Craft *et al.* (*Archives*, 1974, **49**, 235) on the 'grey toddler' with interest. However, there are certain points worthy of comment.

Chloramphenicol, when given orally, provides effective therapeutic antimicrobial blood levels with good penetration in the CSF (Weinstein, 1970) and does not necessitate the intravenous route which may increase the risk of toxicity. Sulphonamides and chloramphenicol to some extent share a common metabolic pathway of glucuronide conjugation in the human liver (Seneca, 1971). This pathway is likely to be overburdened with large dosages of chloramphenicol and sulphonamides when given intravenously, which may be a contributory factor to chloramphenicol toxicity.

Two children who neither had bacterial isolation from the CSF, nor had any other supportive evidence of bacterial meningitis were continued on large doses of these drugs. The third child with *Haemophilus influenzae* meningitis was treated with a combination of sulphadimidine, streptomycin, and chloramphenicol; in fact chloramphenicol alone would have been sufficient for an effective therapy for this type of meningitis (Smith, 1972).

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Dr. Craft replies as follows:

On behalf of my co-authors I would like to reply to Dr. Haider's letter regarding our article on the 'grey toddler'.

Our aim in reporting these 3 cases was to suggest that chloramphenicol may occasionally cause toxicity in toddlers, as well as in neonates, when given intravenously

in a dose of about 100 mg/kg per day. We deliberately avoided the wider issue of the optimum management of a sick child with presumptive meningitis of unknown aetiology. However, we would defend the use of intravenous therapy in the initial management of any child thought to have meningitis whatever the antibiotic regimen chosen.

Chloramphenicol is excreted after glucuronation and sulphonamides following acetylation and we know of no evidence that the one interferes with the other.

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## Storage of sulphatides in gastric mucosa in the late infantile form of sulphatidosis

Sir,

The extraneural storage of sulphatides in sulphatidosis is well known. Its presence has been reported repeatedly in the liver, gall bladder, kidneys, pancreas, adrenals (Wolfe and Pietra, 1964), and sometimes in other sites, such as the hypophysis and reticulo-endothelial system. In a histochemical study of the distribution of the storage in a case of late infantile form of this disease, in a male aged 3 years, the storage of sulphatides was found in the chief cells of gastric glands predominantly in their basal portions, in addition to the above mentioned tissues. The lipid was present in a moderate amount in a form of minute, birefringent, and easily extractable droplets, staining typically brown in the Hirsch-Peiffer method. Occasionally it could be detected even in the glandular lumen. It was also present in the interstitial histiocytes, together with lipopigment. The finding of sulphatide storage in the gastric mucosa increases the list of sites of extraneural storage, and confirms the generalized nature of the metabolic defect (Hagberg and Svennerholm, 1960). If confirmed in other cases, the finding would be useful in the diagnosis of the condition during life.

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