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Case report

Acute epiglottitis: a case cluster

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Acute epiglottitis is most commonly recognised in children as an infection with *H. influenzae* and may be a severe and rapidly fatal disease. Less commonly, adults, compromised by chronic bronchitis, may develop acute epiglottitis with *H. influenzae* or with a variety of other organisms including *Streptococcus pneumoniae, Streptococcus pyogenes, Staphylococcus aureus,*[1] beta-haemolytic group C Streptococci[2] or *Klebsiella* spp.[3] The peak incidence in children is 2 – 6 years of age, due to the fact that infants receive passive immunity to *H. influenzae in utero* which decreases after birth. Susceptibility to infection then increases until a natural immunity to the micro-organism is developed later in childhood. There may be a seasonal predominance in the winter months. The main focus of infection is the supraglottic area, but the acute phase of the infection is more generalised, producing a state of toxaemia. The presentation is usually of sudden onset with dyspnoea, stridor, and profuse drooling. The severity of the infection usually requires immediate medical attention. Prompt treatment is directed towards relief of any airway obstruction and the eradication of the aetiological agent by the use of appropriate antibiotics.

PATIENTS

A cluster of four patients with acute epiglottitis presented to the Royal Belfast Hospital for Sick Children between September and December 1985. Three were less than 21 months and one was five years of age. Three were female and one male. All lived within 20 miles of the hospital. They were all previously healthy and had normal developmental milestones. They presented with sudden onset of inspiratory stridor, which in two cases followed a reported upper respiratory tract infection. The diagnosis of acute epiglottitis rather than laryngtracheitis was suspected due to the severity of presentation. All patients had pyrexia (up to 40°C), raised leucocyte count (up to 27 $\times$ $10^9/l$), and increasing, severe stridor. One patient had a history of penicillin allergy. Diagnosis was confirmed clinically at intubation and confirmed microbiologically by the isolation of *H. influenzae* from blood culture. All patients responded to chloramphenicol within five days with no complications. One patient received ampicillin in addition to chloramphenicol.

ORGANISMS

The organisms were all isolated from blood cultures using an automated radiometric blood culture detection system (Bactec) on chocolate blood agar

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in 5–10% CO_2. Identification was based on X (Haemin) and V (Nicotinamide Adenine Dinucleotide) growth factor utilisation on nutrient agar. Slide agglutination (Wellcome Ltd) showed the strains to be Pittman serotype b. Antibiotic susceptibility testing was by disc diffusion using the Stokes method[4] with the Oxford Staphylococcus NCTC 6571 as control. Minimum inhibitory concentrations were obtained in broth dilutions of antibiotics using inocula of 10^8 colony forming units/litre. Beta-lactamase production was detected using a chromogenic nitrocefin test.

All the isolates were sensitive to chloramphenicol (10 µg disc). Two isolates were resistant to ampicillin (10 µg). In one of these beta-lactamase production was demonstrated, but the other ampicillin resistant strain was not a beta-lactamase producer. Both had minimum inhibitory concentrations >8 µg/ml to ampicillin.

DISCUSSION

A cluster of four cases occurring in a three month period was remarkable as the frequency of cases brought to the attention of bacteriologists through positive blood culture is much lower. Most series report an incidence of about one per thousand paediatric admissions.[5] Incidence rates are erratic which may reflect the cluster-like occurrences over time. There was no reason to suppose that these cases were epidemiologically related.

Of *H. influenzae* isolates in Northern Ireland, 9·3% are resistant to ampicillin due to production of beta-lactamase.[6] One of the strains reported was ampicillin resistant but not a beta-lactamase producer. This form of resistance is uncommon and is probably due to permeability barriers or structural alterations in penicillin binding proteins. A much lower incidence of chloramphenicol resistance, 1·7% has been reported in a United Kingdom survey,[7] and this is therefore the drug of choice in life-threatening *H. influenzae* infection such as epiglottitis. Chloramphenicol is also more rapidly bactericidal than ampicillin against *H. influenzae.* Indeed even those rare strains producing enzymes which inactivate chloramphenicol by transacetylation have been successfully treated with high doses of chloramphenicol.[8] High initial doses of 100 mg/kg/day should be used, which can be reduced later when quantitative susceptibility tests are available and a clinical response is shown. Chloramphenicol assays of trough (pre-dose) and peak (1 hr post dose) serum concentrations should be performed to determine maintenance intravenous dosage aimed at a peak serum level of 15–25 mg/l, as there are large variations in pharmacokinetic responses due to age, liver enzyme induction, and protein binding displacement. A peak at two hours post dose should be taken when using oral administration. Marrow toxicity may also be anticipated by reticulocyte, haemoglobin, neutrophil, and platelet counts.[9] Some of the newer cephalosporins such as cefuroxime and cefotaxime which show good in vitro activity against *H. influenzae* have been used in the treatment of *H. influenzae* meningitis and epigolottitis,[10] and provide a suitable therapeutic alternative to chloramphenicol in epigolottitis without the potential problems of associated toxicity.

Household contacts under the age of six have a 500 times increased risk of *H. influenzae* infection,[11] and rifampicin prophylaxis should be considered in this risk group. Pharyngeal colonisation of contacts is reported to be up to 70%, so that culture from contacts is not helpful in identifying those at risk. In the United States of America, familial spread has more commonly been reported and antibiotic prophylaxis of close contacts is recommended. In this country the

incidence and rate of spread is much lower and the debate on antibiotic prophylaxis of contacts has not yet been resolved: there is, however, a reasoned trend towards antibiotic prophylaxis for close contacts of acute cases.

Bacterial capsular polysaccharide vaccines are now widely used in the United States of America at 24 months of age, and some studies report significant reductions in the incidence of *H. influenzae* type b infection in children over 24 months.[12] Protein polysaccharide conjugate vaccines are being investigated with the hope of improved protection to include children less than 24 months who are at greater risk from life-threatening infection.

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