

## CORRESPONDENCE

## Necrotising pneumonia, pneumatoceles and the pneumococcus

We commend Principi and Esposito for their timely review of severe community-acquired pneumonia in children.<sup>1</sup> They state that the leading cause of necrotising pneumonia and pneumatoceles within the context of community acquired pneumonia is *Staphylococcus aureus*. We must disagree with this claim.

Historically, *S aureus* has been the principal organism associated with necrotic or cavitary pneumonia in children, but recent data demonstrate *Streptococcus pneumoniae* is now a much more important cause.<sup>2-4</sup> Pneumococcal infection was the leading cause of necrotic or cavitary pneumonia in two recent studies by Sawicki *et al* in Boston and McKee *et al* in London, accounting for 89% (28/32) of cases in the latter;<sup>2 3</sup> this figure will underestimate the true incidence of pneumococcal infection as over 90% of invasive pneumococcal disease is culture negative, and culture negative techniques for determining pneumococcal infection and serotyping are still not widely available.

The occurrence of severe pneumococcal necrotising pneumonia in children was first reported by Kerem *et al* in 1994.<sup>5</sup> This problem has become progressively more common globally over the last 2 decades.<sup>2 3</sup> These changes are linked to increases in infection with certain pneumococcal serotypes, specifically serotypes 1, 3, 14 and 19A.<sup>3 4 6</sup> Hsieh *et al* have demonstrated that the lung necrosis and subsequent cavitary changes are microangiopathic resulting from thrombosis of intrapulmonary blood vessels and subsequent pulmonary gangrene.<sup>6</sup> It is tempting to speculate that earlier recognition of these haematological changes could offer the opportunity for therapeutic intervention to minimise lung damage.

Worryingly, the rise in 19A infection in the USA has been associated with the spread of several multi-antibiotic resistant clones. We have also recently observed an increase in severe 19A disease in the UK within the UK paediatric empyema surveillance programme, but we have not found any evidence of antibiotic resistance.

Staphylococcal disease due to methicillin-resistant strains and Pantone-Valentine leukocidin related disease remain important, but most reports are of sporadic outbreaks and the overall incidence of staphylococcal pneumonia in children in developed countries is now very low. These findings have significant implications for patient management. For many years, our first choice antibiotic has been Clindamycin which has excellent antipneumococcal activity and antistaphylococcal activity. When managing patients with proven staphylococcal pneumonia, it is also now our practice to

exclude important underlying immunological causes. Paediatricians need to be ever aware of the rapidly changing epidemiology of pneumococcal disease and that the consequences of pneumococcal infection can be extremely serious.

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