SHORT REPORT

Sudden infant death due to disseminated pneumococcal infection

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The Office of National Statistics, England has reported approximately 13 deaths in children per year as a result of invasive pneumococcal disease (meningitis, septicaemia, pneumonia) between 1989 and 1999 in England and Wales. This is thought to be an underestimate; the actual incidence is supposed to be at least three times higher.¹ However, sudden infant death syndrome as a result of pneumococcal disease in a previously healthy child is extremely rare.²

A 6 month old infant with a 10 day history of coryzal symptoms suddenly became cyanotic and apnoeic while playing at home. She was immediately taken to the emergency department where cardiopulmonary resuscitation was unsuccessful. Previously she had been a thriving infant growing along the 75th centile. She had been seen three times by a health care provider in the past week, including a one hour stay in the paediatric ward, seven days prior to her collapse. She remained afebrile, eating well and playing, and was discharged home with a diagnosis of upper respiratory tract infection. She had been immunised with three doses of diphtheria-pertussis-tetanus and HiB vaccine, oral polio vaccine, and meningococcal C vaccine.

Blood samples taken at the time of resuscitation showed haemoglobin 83 g/L, white blood cells $27.7 \times 10^9/L$ (neutrophils $12.5 \times 10^9/L$, lymphocytes $15.2 \times 10^9/L$), platelets $215 \times 10^9/L$. Peripheral blood film showed Tuerk cells suggestive of viral infection. Tests for antibody titres (Epstein-Barr virus, influenza virus, parainfluenza virus, adenovirus, and respiratory syncytial virus) were negative. Mycoplasma antibody titres were not raised. Blood culture was negative. A full skeletal survey prior to postmortem examination showed bilateral pleural effusions and enlarged heart and liver.

Postmortem examination showed a grossly enlarged heart with dilated right atrium. The myocardium was soft and flabby with small pale areas. Lungs were firm in consistency and were oedematous. No haemorrhage was seen. The liver was enlarged and had a mottled appearance. Kidneys, gall bladder, spleen, thymus, and adrenals appeared normal. Meninges appeared thickened and gelatinous.

On histology, extensive acute inflammatory infiltrates involving meninges were seen. Focal area of necrosis and inflammation in the brain substance suggestive of encephalitis were also present. Extensive inflammatory infiltrates over the pericardium, and focal abscesses in the myocardium, especially in the region of the mitral valves, were seen. In the lungs, diffuse lymphocytic infiltrate and macrophages were seen in the alveolar walls, indicating a viral infection. Lung substance was not involved by the type of acute inflammatory infiltrate seen in the heart and meninges, but on the pleural surface of the right lung there was a dense inflammatory infiltrate, reflecting infection in the pleural cavity. The thymus showed notable stress reaction and focal abscesses were seen

in the adjoining connective tissue. No abnormality was seen in the spleen, kidneys, and adrenals. The liver showed extensive fatty change, but no inflammation was seen. Culture of brain tissue, cerebrospinal fluid, and subcutaneous tissue around the neck, pleura, and myocardium grew *Streptococcus pneumoniae* sensitive to penicillin.

In summary, the child had evidence of a viral chest infection but also more significantly a disseminated pneumococcal infection involving the meninges, brain substance, pleura, pericardium, myocardium, and soft tissues of the neck, which was thought to be the cause of death. Fatty change in the liver was thought to be a consequence of a disseminated bacterial infection.

An increase in invasive pneumococcal infections requiring intensive care admission has been reported recently.³ The antibiotic resistance to pneumococcal infections is also reported to be increasing.⁴ Immunisation programmes targeted at *Haemophilus influenzae* type b and *Neisseria meningitidis* group C have resulted in substantial reductions in mortality in vaccinees. A 7 valent pneumococcal conjugate vaccine licensed for use in European infants has been shown to lower the incidence of all invasive pneumococcal infections (approximately one case avoided per 400 children immunised), and also those caused by the seven serotypes covered by the vaccine.^{5,6} This case raises the issue of whether pneumococcal immunisation for infants should become a routine in the UK, rather than immunising the high risk group only.

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REFERENCES

- McIntosh EDG, Booy R. Invasive pneumococcal disease in England and Wales: what is the true burden and what is the potential for prevention using 7 valent pneumococcal conjugate vaccine? *Arch Dis Child* 2002;86:403-6.
- Sharief N, Khan K, Conlan P. Overwhelming sepsis presenting as sudden unexpected death. *Arch Dis Child* 1993;69:381-2.
- Pierce CM, van Saene HKF, Petros AJ. Increase in severe *S. pneumoniae* infection requiring paediatric intensive care and a decrease in antibiotic prescribing. *Arch Dis Child* 2001;84(suppl 1):a10.
- Cartwright K. Pneumococcal disease in western Europe: burden of disease, antibiotic resistance and management. *Eur J Pediatr* 2002;161:188-95.
- Anonymous. 7-valent pneumococcal conjugate vaccine: new preparation. Prevents rare invasive infections in infants. *Prescrire Int* 2002;11(57):7-10.
- Finn A, Booy R, Moxon R, et al. Should the new pneumococcal vaccine be used in high-risk children? *Arch Dis Child* 2002;87:18-21.