Haemophilus influenzae type b pneumonia

ELLEN R. WALD AND MYRON M. LEVINE

From the Department of Pediatrics and Division of Infectious Diseases, University of Maryland Hospital and School of Medicine, Baltimore

SUMMARY Thirteen patients with *Haemophilus influenzae* type b pneumonia are reported to emphasise the clinical, radiographic, and therapeutic aspects of this illness. All but one patient was under 2½ years of age. The presenting complaint was a variable duration of upper respiratory infection and fever in most cases. One-third of patients had radiographic evidence of pleural involvement; one-third showed a patchy bronchopneumonia on roentgenogram; and the remainder had segmental or lobar infiltrates. Clinical response to antibiotic therapy was prompt in patients without pleural involvement.

The relative importance of *Haemophilus influenzae* type b as a pulmonary pathogen in the antibiotic era is unknown. Since 1955, reports have stressed the rarity of primary *H. influenzae* type b pneumonia with or without empyema (Nyhan *et al.*, 1955; Riley and Bracken, 1965; Vinik *et al.*, 1966; Honig *et al.*, 1973; Faden and Overall, 1976). However, recent paediatric textbooks (Smith, 1972; McCracken and Eichenwald, 1975) note that pneumonia and empyema due to *H. influenzae* type b may be clinically and radiographically indistinguishable from pneumococcal pneumonia. Furthermore, the apparent therapeutic responses of patients with pneumonia to penicillin may not distinguish between these two aetiological agents (Honig *et al.*, 1973). Our recent experience with 13 patients is presented to emphasise the clinical, radiographic, and therapeutic aspects of *H. influenzae* type b pneumonia.

Case reports

The case histories of the 13 patients (8 boys, 5 girls) with *H. influenzae* type b pneumonia are summarised in the Table. They all presented during a 4-year period, 1972-1976. Diagnosis was based on the recovery of *H. influenzae* type b from the culture of blood or pleural fluid in a patient with pulmonary infiltrates on roentgenogram. These patients were not evaluated for simultaneous viral infection by either cultures or serology. None of the patients had any other focus of infection. 12 of the 13 patients were under 2½ years of age. The white blood cell count was $>12.0 \times 10^9/l$ for all, but the differential counts were variable. The presenting complaints varied

from a preceding upper respiratory infection of 1 day to 3 weeks' duration in 12 patients, to acute onset of fever and right-sided chest pain in 1.

Four patients had radiographic evidence of pleural involvement; diagnostic thoracentesis performed in 3 showed empyema in each. Closed thoracotomy drainage was necessary in only one patient (Case 3) because of reaccumulation of fluid and continued fever. 2 patients (Cases 1, 3) did not become afebrile until after 2 weeks and were discharged with persistent pleural reaction. Subsequently, both have had complete resolution of the pleural reaction by radiological examination. One patient (Case 4) had only a small right-sided effusion that did not require drainage; both he and Case 2 responded promptly to antibiotic therapy. Roentgenograms in the 9 patients without pleural effusion included patchy bronchopneumonia in 4 and segmental or lobar involvement in the others.

Four patients were treated with intramuscular procaine penicillin G; each responded promptly to therapy. Case 8 responded to penicillin G, 100 000 U/kg intravenously. Case 3 was begun on intravenous methicillin and became afebrile within 24 hours of therapy. Cases 5 and 12 responded promptly to intravenous ampicillin. Case 11 was treated with erythromycin and sulphafurazole because of alleged penicillin allergy. In the remainder, treatment was initiated with intravenous ampicillin and methicillin because of pleural effusion.

Discussion

H. influenzae type b may cause fulminating pneumonia or be responsible for a more indolent presentation. Radiographically, lobar distribution is more

Table Clinical details of 13 patients with *H. influenzae* type b pneumonia

Case no.	Age (yr) (m)	Sex	History	Temp. on admission (°C)	WBC (diff) ($\times 10^9/l$)	X-ray	Isolation of <i>H. influenzae</i> b	
							Blood	Pleural fluid
1	1	4 F	URI 3 d	40.3	12.6; 72P, 2B 17L, 9M	RLL pneumonia R effusion	+	+
2	1	7 M	URI 1 w	38.9	21.7; 69P, 16B 14L, 14M	LLL pneumonia L effusion	+	Gram (—) cocci bacilli seen on smear
3	2	6 F	Fever and URI 4 d	39.7	29.9; 34P, 36B 10 Met, 1E 13L, 6M	RLL pneumonia R effusion	—	+
4	4	M	Fever and URI 10 d	38.6	17.8; 41P, 2B 39L, 10M	RLL pneumonia R effusion	+	Not done
5	2	M	URI 7 d	38.3	23.0; 51P, 30B 9L, 10M	RLL, LLL (broncho-pneumonia)	+	
6	11	F	URI 3 d	38.9	13.2; 56P, 44L	RUL, RLL (broncho-pneumonia)	+	
7	7	M	URI 1 w	38.6	25.4; 69P, 26L 5M	LUL pneumonia	+	
8	15	M	Sudden onset chest pain	38.6	12.1; 85P, 3B 9L, 3M	RUL pneumonia	+	
9	8	F	Fever and URI 1 d	38.9	16.6; 26P, 6B 64L, 8M	RUL (broncho- pneumonia)	+	
10	6	M	Fever and URI 6 d	40.3	15.5; 50P, 2B 40L, 8M	RUL pneumonia	+	
11	1	8 M	Fever and URI 3 d	38.3	14.2; 77P, 8M 14L	RUL pneumonia	+	
12	6	M	URI 3 w	40.0	19.7; 61P, 17B 12L, 10M	LUL pneumonia	+	
13	2	F	Fever and URI 9 d	39.7	Not done	LLL (broncho- pneumonia)	+	

URI = upper respiratory infection; P = polymorphonuclear leucocytes; B = band forms; Met = metamyelocytes; L = lymphocytes; M = monocytes.

common than bronchopneumonia in infants and children. 'Classical' pneumococcal disease with fever, chest pain, rigor, and lobar distribution is less common in infancy than indolent disease with non-specific upper respiratory complaints and patchy bronchopneumonia on radiography. Therefore, the specific bacteriological pathogen responsible for pneumonia cannot be predicted on the basis of clinical or radiographic presentation. In addition, pneumonia due to *H. influenzae* type b has responded to intramuscular penicillin G (Nyhan *et al.*, 1955). This may be explained by data which indicate that the minimal inhibitory concentration of penicillin for *H. influenzae* type b is approximately one dilution higher than that of ampicillin (McLinn *et al.*, 1970; Sabath *et al.*, 1970). Because of their apparent brisk therapeutic response to penicillin or methicillin, 5 of these 13 patients may not have been recognised as having *H. influenzae* type b pneumonia without the routine use of blood cultures.

There are therapeutic implications in the recognition of *H. influenzae* type b as a cause of pneumonia. The recent documentation of ampicillin resistance in strains of *H. influenzae* type b has resulted in altered recommendations for the treatment of meningitis and other life-threatening illnesses caused by the organism (Katz, 1975). Failure of clinical response

in pneumonia may indicate an ampicillin-resistant *H. influenzae* type b and a need to change antibiotic regimens. To document the possible bacterial aetiology of pneumonia an aggressive diagnostic approach to all pneumonias should include the routine use of cultures of blood and pleural fluid (when available). Lung aspirates may be necessary to diagnose the aetiological agent in those patients who do not have bacteraemia and respond poorly to conventional therapy.

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Correspondence to Dr Ellen R. Wald, Department of Pediatrics, 22 South Greene Street, Baltimore, Maryland 21201, USA.