

## NOTES

### Pneumonia Due to *Haemophilus influenzae* (*H. aegyptius*) Biotype 3

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*Haemophilus influenzae* (*H. aegyptius*) biotype 3 was isolated from eye, nasopharyngeal, and sputum cultures of a 23-month-old male and from sputum and transtracheal aspirate cultures of his 39-year-old mother, both with diffuse bronchopneumonia.

The literature contains recent reports concerning *Haemophilus influenzae* and other species of this genus as the etiological agents in a variety of clinical syndromes (1, 3). However, a review of the literature has failed to reveal any report concerning the isolation and identification of *H. influenzae* (*H. aegyptius*) biotype 3 associated with clinical cases of diffuse bronchopneumonia. Therefore, to our knowledge, this is the first time that  $\beta$ -lactamase-producing *H. influenzae* (*H. aegyptius*) biotype 3 has been reported as the causative agent of diffuse bronchopneumonia in two related cases.

#### CASE REPORTS

**Case 1.** A 23-month-old male was admitted to the Wilford Hall Medical Center, Lackland Air Force Base, Tex., on 3 January 1977. Prior to admission, the patient had a 4-week history of upper respiratory congestion, with rhinorrhea and coughing, and had been placed on appropriate supportive therapy, including oral ampicillin and decongestants. However, due to a markedly increased productive cough, wheezing, and respiratory distress, he was admitted for further evaluation and treatment. Physical examination revealed a classical Down's syndrome, previously confirmed by chromosomal analysis. Rectal temperature was 100.6°F (38.2°C). There was much purulent nasal discharge and congestion, accompanied by posterior pharyngeal drainage. The admission leukocyte count was 15,600 cells per mm<sup>3</sup>. The differential showed 73 segmented neutrophils, 2 bands, 15 lymphocytes, and 10 monocytes. The radiographic report of the chest film indicated a right upper lobe infiltrate and some suggestion of a right and left lower lobe infiltrate. The patient was

placed on appropriate dosages of methicillin and gentamicin. During the next several days, the clinical condition of the patient deteriorated despite vigorous management procedures and the addition of chloramphenicol to his antibiotic coverage. His temperature reached a maximum of 105°F (40.5°C), and generalized tonic-clonic seizures and two cardiorespiratory arrests occurred. On day 4, a third arrest occurred from which the patient could not be resuscitated, and he was pronounced dead on 7 January 1977.

From the day of admission, multiple specimens were submitted for culture. Blood and cerebrospinal fluid cultures failed to reveal the presence of microorganisms. Cultures of the eye, nasopharynx, and sputum, however, grew *H. influenzae*, with chocolate agar containing GC agar base (Baltimore Biological Laboratory [BBL], Cockeysville, Md.), 1% IsoVitaleX (BBL), and 5% sheep erythrocytes as the growth medium. The organisms exhibited typical tinctural and morphological characteristics on Gram stain, fastidious growth requirements, and an X- and V-factor requirement (6), as determined on brain heart infusion agar (BBL) with X-, V-, and X- and V-factor strips (BBL). Hemolysis was negative on 5% rabbit blood agar (4). Serological typing by slide agglutination with *H. influenzae* poly and types A through F antisera (Difco Laboratories, Detroit, Mich.) showed the isolates to be nontypable. Penicillin  $\beta$ -lactamase enzyme was demonstrated from all isolates by the method of Rosenblatt and Neumann (Program Abstr. Intersci. Conf. Antimicrob. Agents Chemother. 15th, Washington, D.C., Abstr. no. 388, 1975). The modified Bauer-Kirby technique (2) con-

firmed the resistance of the isolates to ampicillin and their sensitivity to chloramphenicol and erythromycin. Isolates were forwarded to a local reference laboratory for confirmation and taxonomic biotyping by the method of Kilian (5). The use of this schema (Table 1) permitted identification of these organisms as *H. influenzae* biotype 3.

**Case 2.** A 39-year-old female, the mother of case 1, was admitted to this medical center on 9 January 1977. Before admission, the patient had a 2-week history of shortness of breath and coughing, with yellow sputum. Appropriate supportive therapy was instituted, and, during the next 6 outpatient days, the fever reached 102°F (38.9°C) and the coughing increased. At this time, a chest film confirmed a diffuse bronchopneumonia, and ampicillin was added to the medication regimen. Over the next 3 days, the cough resolved somewhat and the fever disappeared. On approximately 1 January 1977, the patient experienced a return of the fever, and the cough and sputum production were much worse. On 9 January 1977, a chest film showed a progression of the diffuse bronchopneumonia, and admission was advised. Temperature on admission was 102°F (38.9°C), and the leuko-

cyte count was 16,000 with a left shift. Findings of the physical examination were compatible with diffuse bronchopneumonia. Multiple sputum specimens and transtracheal aspirates, when cultured and identified as previously described (case 1), yielded growth of ampicillin-resistant,  $\beta$ -lactamase-positive *H. influenzae* (*H. aegyptius*) biotype 3. The patient recovered promptly on intravenous chloramphenicol and was discharged on 17 January 1977, after being afebrile for 46 h with chest X-ray evidence of resolution.

The implication of this microorganism as the etiological agent of diffuse bronchopneumonia is a significant finding. These cases indicate that the clinical microbiologist must be more aware of the *Haemophilus* species and their possible role in disease. Moreover, the resistance of these organisms to ampicillin suggests that all significant *Haemophilus* isolates should be examined for the elaboration of  $\beta$ -lactamases.

#### LITERATURE CITED

1. Buck, L. L., and G. W. Douglas. 1976. Meningitis due to *Haemophilus influenzae* type e. *J. Clin. Microbiol.* 4:381.
2. Center for Disease Control. 1975. Ampicillin-resistant *Haemophilus influenzae*. *Morbid. Mortal. Weekly Rep.* 24:205-206.
3. Jones, R. N., J. Slepach, and J. Bigelow. 1976. Ampicillin-resistant *Haemophilus paraphrophilus* laryngoepiglottitis. *J. Clin. Microbiol.* 4:405-407.
4. Kilian, M. 1974. A rapid method for the differentiation of *Haemophilus* strains. The porphyrin test. *Acta Pathol. Microbiol. Scand. Sect. B* 82:835-842.
5. Kilian, M. 1976. A taxonomic study of the genus *Haemophilus*, with the proposal of a new species. *J. Gen. Microbiol.* 93:9-62.
6. Young, Y. M. 1974. *Haemophilus*, p. 302-307. In E. H. Lennette, E. H. Spaulding, and J. P. Truant (ed.), *Manual of clinical microbiology*, 2nd ed. American Society for Microbiology, Washington, D.C.

TABLE 1. Abbreviated biochemical characteristics of *H. influenzae* biotypes

<i>H. influenzae</i> biotype	Indole production	Urease activity	Ornithine decarboxylase activity
1	+	+	+
2	+	+	-
3	-	+	-
4	-	+	+
5	+	-	+