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Haemophilus Influenzae Invasive Disease in Alaska

Haemophilus influenzae (HI) are gram-negative coccobacilli. There are six distinct encapsulated types, designated by serotyping as types A-F, and strains that are unencapsulated and grouped as nontypable. Serotype B(Hib) is the major cause of serious invasive disease such as meningitis, septic arthritis, pneumonia, epiglottitis, and cellulitis. Unencapsulated strains are implicated as causing otitis media, bronchitis, and sinusitis. Although HI is a major pathogen in young children, both encapsulated and unencapsulated strains of HI have been found to cause obstetrical infections, pneumonia, meningitis, occult bacteremia, and epiglottitis in adults.

A 1977 study conducted in Bethel by the Centers for Disease Control (CDC) showed that Yupik Eskimos have a high incidence of Hib meningitis and Hib invasive diseases. In 1979, the CDC and the Alaska State Department of Health and Human Services initiated statewide surveillance of invasive HI disease to determine the incidence of HI diseases throughout the state. Clinical laboratories, physicians, and infection control nurses have assisted in reporting cases to this surveillance system.

From January 1980 through December 1981, 189 cases of invasive Hib diseases occurred in Alaska. 50.9% were in Alaska natives. 90% of the cases were children under 60 months of age. Eskimo children, primarily those less than 12 months of age, had an incidence of disease ten times that seen in Caucasian children. Incidence of Hib meningitis and invasive Hib diseases by age and race are shown in Table 1.

Differences were noted in the manifestation of types of invasive Hib diseases between natives and non-natives (Table 2). Among Alaskan natives, cases of Hib pneumonia occurred more frequently 35%, then non-natives 7% ($P < .01$). Infectious arthritis also occurred more frequently in natives 12%, than non-natives 3%; but these differences were not shown to be statistically significant among non-natives, there were more cases of epiglottitis, 15% than in the natives 2% ($P < .01$).

Seasonal variations in the occurrence of Hib invasive diseases were observed in Alaska. Invasive Hib diseases were observed more frequently in the months March through May and in September than during the summer or winter periods.

There were no secondary cases among exposed children in nursery schools or day care centers. However, 2 brothers from one family were hospitalized one day apart, one with meningitis and the other with infectious arthritis. These two cases were considered co-primary cases rather than a result of secondary spread.

Table 1. Annual Incidence* of Hemophilus Influenza type b						
Disease in Alaska 1980-1981						
	Alaskan Native		Alaskan Non-Native			
	Eskimo	Indian	Caucasian	Black	Other	
Meningitis						
<12 months	1088 (20)	634 (6)	148 (20)	(0)	0	
<5 years	269 (22)	261 (11)	70.0 (42)	(0)	0	
All ages	27.2 (23)	25.3 (11)	6.9 (43)	(0)	0	
Other Invasive Disease						
<12 months	1415 (26)	317 (3)	133 (18)	167 (1)	0	
>5 years	562 (46)	214 (9)	63.3 (38)	37.8 (1)	0	
All ages	55.7 (47)	30.0 (13)	7.3 (45)	18.3 (5)	0	
All Diseases						
<12 months	2503 (46)	951 (9)	282 (38)	167 (1)	0	
<5 years	831 (68)	428 (18)	133 (80)	37.8 (1)	0	
All ages	85.3 (72)	55.3 (24)	14.2 (88)	18.3 (5)	0	

*Cases per 100,000 people per year () number of cases

Table 2. Invasive Hemophilus Influenza type b Infections in Comparative Population Groups					
Disease	Alaska Native		Alaska Non-Native		U.S. Reference*
Meningitis	35%	(34)	46%	(43)	53% (253)
Pneumonia	36%	(35)	7%	(7)	14% (67)
Cellulitis	9%	(9)	15%	(13)	9% (43)
Arthritis	12%	(12)	3%	(3)	6% (28)
Bacteremia	4%	(4)	9%	(8)	4% (19)
Other	2%	(1)	4%	(4)	2% (9)
Epiglottitis	2%	(1)	16%	(15)	13% (62)
Total	100%	(96)	100%	(93)	100% (481)

*Ref- Fresno, Calif. (Granoff, JID 141:40, 1980); Detroit, Mich, (Dajani, J. Pediat. 94:355, 1979); Denver, Col. (Todd, AJDC 129:607, 1974).

() Number of cases

Six native children had recurrent episodes of invasive Hib disease. The mean interval between hospital discharge and re-admission was 21.6 days (range 10-57) and the mean age of these children was 7.5 months (range 5-18 months). Three of the children had two episodes of pneumonia, and the remaining three had at least one episode of meningitis with a second episode of pneumonia or infectious arthritis.

Overall mortality due to invasive Hib diseases was 2.1% of all cases. Mortality in children less than 60 months of age was 2.3%. Three of the deaths in children were due to meningitis and one to pneumonia.

All of the cases were due to Haemophilus influenzae serotype b. Sixty-two isolates were forwarded to the Arctic Investigations Laboratory, Center for Infectious Diseases, Centers for Disease Control, Anchorage, for further evaluation. Eighty percent of the isolates were biotype 1 and 24% were beta-lactamase positive and ampicillin resistant.

Diagnosis

Recovery of the organism from blood, cerebral spinal fluid, joint fluid, wounds or other usually sterile body fluids is the most definitive method of identifying invasive Hib diseases. Because of the fastidious nature of this organism, wound cultures or body cultures should be plated as quickly as possible on chocolate agar supplemented with x and v factors. All isolates of H. influenzae should be tested for susceptibility to both ampicillin and chloramphenicol by the Kirby-Bauer disc method. While beta-lactamase production will identify 95% of H. influenzae which are ampicillin resistant, 5% of beta lactamase negative organisms will also be ampicillin resistant.

Latex particle agglutination may also be used to confirm the presence of the capsular polysaccharide, polyribitol phosphate (PRP), in cerebral spinal fluid, blood, or urine and assist in making a rapid diagnosis of invasive Hib diseases. Although highly sensitive in being able to detect up to 1.0ng of PRP/ml, certain strains of E. coli and streptococcus pneumoniae may cause false positive results. Duration of antigen excretion in urine and serum is variable and has not been correlated with persistence of infection. Clinical impression of the physician and the patient's clinical course should determine duration of antibiotic therapy.