Invasive *Streptococcus pneumoniae* in Perth teaching hospitals, 1990 to 1994

Virginia McLaughlin, Thomas Riley and Christine Roberts

Abstract

*Streptococcus pneumoniae* causes pneumonia, otitis media and meningitis. Reports of penicillin resistance and the development of vaccines highlight the need for baseline information about pneumococcal disease in Australia. We surveyed Perth teaching hospital laboratory records for the period 1990 to 1994 for isolates of *S. pneumoniae* recovered from normally sterile sites, and obtained isolate and patient demographic information. Highest rates of invasive disease were found at the extremes of age and were associated with Aboriginality. Isolates were rarely penicillin resistant. Surveillance of invasive pneumococcal disease will be of importance in monitoring the emergence of penicillin resistance and the impact of conjugate vaccines. *Comm Dis Intell* 1997;21;73-76.

Introduction

*Streptococcus pneumoniae* (*S. pneumoniae*) is an important infectious cause of pneumonia, otitis media and meningitis. The highest incidence of invasive disease is seen at the extremes of age, especially in the under five years age group and over 65 years age group. Predisposing factors that may contribute to an increased risk of disease include excessive alcohol intake, smoking, diabetes mellitus, chronic renal failure, cardiac disease, HIV infection, asplenia and malignancy. A male predominance has also been reported which may reflect alcohol intake, cigarette consumption or other factors. In paediatric populations this predominance is not so marked. Use of pneumococcal vaccines may reduce the incidence of invasive pneumococcal disease. In 1977, a 14-valent pneumococcal vaccine was introduced to Australia, followed by the current 23-valent vaccine in 1984. The 23-valent vaccine is poorly immunogenic in children under two years of age. However, a conjugate vaccine presently being developed overseas may be efficacious in this group. Current pneumococcal vaccine recommendations by the National Health and Medical Research Council target-at-risk

1. Master of Applied Epidemiology Program, National Centre for Epidemiology and Population Health, The Australian National University, Canberra, ACT 0200.
2. Communicable Diseases Control Program, Health Department of Western Australia, WA.
3. Department of Microbiology, The University of Western Australia, WA.

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groups, including those individuals who are asplenic or immunocompromised, Aboriginal or Torres Strait Islander people who are over 50 years of age, or patients with cerebrospinal fluid (CSF) leaks.

Invasive disease caused by *S. pneumoniae* is not notifiable in Western Australia and, as a consequence, epidemiological information is limited. We reviewed laboratory records from Perth teaching hospitals for the period 1990 to 1994 to determine both the site of recovery of invasive pneumococci and some characteristics of those individuals being affected.

**Methods**

Microbiology laboratories servicing Perth teaching hospitals provided data on isolates of *S. pneumoniae* from all patients resident in Perth, for 1990 to 1994 inclusive. Hospitals included two adult hospitals (bed sizes 832 and 575) with a complete range of medical and surgical specialties except obstetrics, a children's hospital (bed size 230) and a general hospital (bed size 357). All acute admissions in the Perth area are seen at one of these four hospitals.

Isolates were considered invasive if they were recovered from sterile sites, that is blood, CSF, pleural fluid/aspirate, peritoneal/ascitic fluid, joint fluid, brain or central venous catheter (CVC) tip. Isolates from other tissues, urine and sputum, isolates where the specimen type was unknown and duplicate isolates from the same patient were not included in the analysis.

Data for the entire five year period were analysed for specimen type, date of specimen collection and antibiotic susceptibility pattern. Only 1994 data were analysed for age, sex, and Aboriginality because of incompleteness of these variables in previous years. While every effort was made to exclude non-Perth residents, postcode data were incomplete and could not be reliably analysed. Frequency distributions and chi square tests were calculated using the Epi Info version 6.02 statistical software package. Annual population denominator figures for metropolitan Perth for 1990 to 1994 were kindly provided by the Health Information Centre of the Health Department of Western Australia.

**Results**

Laboratories from all four Perth teaching hospitals participated. One laboratory was unable to provide data for 1990, but all four contributed data for the other four years.

*S. pneumoniae* was isolated from 3,178 individuals over the five year period. This total includes isolates from both invasive and non-invasive sites. There were 445 invasive pneumococcal isolates recovered from 427 individuals (Table 1). The proportion of invasive *S. pneumoniae* isolates increased from 11.7% in 1990 to a peak of 15.6% in 1993, followed by a slight decline.

Most invasive isolates (71%) were recovered during the cooler months of April to October. Small increases were also seen in December in 1992 (10 isolates) and November in 1993 (12 isolates).

Blood was consistently the most common site of isolation (91%), followed by CSF. In 1994, blood isolates were more common from adults 60 years of age and above (41 isolates; 51%) than from children under five years of age (18 isolates, 23%). CSF was the source for six invasive isolates (7%), with three of these isolates recovered from children less than five years of age.

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**Table. Number of invasive *S. pneumoniae* isolates per individual for Perth by year, 1990 to 1994**

<table>
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<tbody>
<tr>
<td>Invasive¹ (%)</td>
<td>71 (11.7)</td>
<td>81 (11.8)</td>
<td>90 (13.7)</td>
<td>99 (15.6)</td>
<td>86 (14.5)</td>
<td>427</td>
</tr>
<tr>
<td>Blood</td>
<td>66</td>
<td>78</td>
<td>78</td>
<td>85</td>
<td>80</td>
<td>387</td>
</tr>
<tr>
<td>CSF</td>
<td>8</td>
<td>9</td>
<td>9</td>
<td>9</td>
<td>6</td>
<td>41</td>
</tr>
<tr>
<td>Other sterile site</td>
<td>2</td>
<td>1</td>
<td>5</td>
<td>6</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>Non-invasive</td>
<td>513</td>
<td>585</td>
<td>526</td>
<td>516</td>
<td>506</td>
<td>264</td>
</tr>
<tr>
<td>Unknown</td>
<td>21</td>
<td>20</td>
<td>43</td>
<td>21</td>
<td>0</td>
<td>105</td>
</tr>
<tr>
<td>Total</td>
<td>605</td>
<td>686</td>
<td>659</td>
<td>636</td>
<td>592</td>
<td>317</td>
</tr>
<tr>
<td>Number of laboratories contributing</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
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</tr>
</tbody>
</table>

1. Individuals may have had isolates from more than one sterile site.
In 1994, the overall rate for reported invasive isolates from metropolitan Perth was 6.9 per 100,000 population. There was a similar distribution by sex, with rates of 6.7 per 100,000 for males and 7.2 per 100,000 for females. Highest age-specific rates were seen in children less than five years of age, 23.6 per 100,000 (22 isolates); and adults 60 years of age and over, 24.1 per 100,000 (43 isolates)(Figure 1). The rate for children less than two years of age was 30.4 per 100,000 (Figure 2). There was also a small increase in the rate for the 25 - 29 years age group to 8.7 per 100,000 population (8 isolates). Aboriginality was associated with a significantly increased rate of invasive isolates: 61.5 per 100,000 population (8 isolates) compared with non-Aborigines, 6.2 per 100,000 population (78 isolates) (relative risk = 9.9; 95% CI 4.8-20.6). Of the 10 children less than two years of age, two were Aborigines.

Only seven invasive isolates (1.6%) were resistant to penicillin in the five year period. These included six isolates from blood and one from CSF; with one recovered in 1991, three in 1992 and three in 1993. Demographic details were limited, however three of these isolates were from adult hospitals and four were from the children's hospital.

Discussion

There is currently much interest in S. pneumoniae due to reports of increasing antibiotic resistance and the anticipation of a conjugate vaccine proving efficacious in children less than two years of age. Since the introduction of Haemophilus influenzae type b vaccines in 1992, there has been a marked decrease in the incidence of disease caused by this organism in children less than five years of age. S. pneumoniae may subsequently become a more prominent cause of infection. Pneumococcal meningitis in children results in significant morbidity, including neurological sequelae and severe hearing loss. This may be preventable with an efficacious vaccine in the paediatric group, while the incidence of otitis media commonly caused by S. pneumoniae may also be reduced.

We acknowledge several limitations in this study. First, the study population excluded private laboratories. However, private laboratories are less likely to see cases of invasive pneumococcal disease because they generally do not service hospitals which deal with acute admissions. In a larger retrospective study, only three isolates of invasive pneumococci were recovered from 1,189 specimens from private laboratories (Virginia McLaughlin, unpublished data) compared with 427 from 3,178 isolates from public laboratories.

Second, missing specimen type information may have resulted in misclassification. Isolates where the specimen type was unknown were classified as non-invasive, which may have resulted in an underestimate of the percentage of invasive isolates.

Third, individuals with pneumonia who only had a positive sputum specimen have been excluded from the invasive group. Although S. pneumoniae classically causes lobar pneumonia, expectorated sputum is an unreliable indicator because it may be contaminated by organisms normally resident in the oropharynx. Patients with pneumococcal pneumonia may have positive blood cultures in only 25 to 35% of cases. Both these factors may have resulted in an underestimate of the rate of invasive disease.

Last, missing postcode information may have resulted in misclassification. It is possible that rural residents were included in the study due to transfers from remote areas of Western Australia to Perth. This has implications for antimicrobial therapy and management of affected individuals. Again, surveillance will assume an increasingly important role in the detection of these trends.

Although the annual number of isolates from invasive specimens over the five year period. It is important to note that penicillin resistance was detected and would be expected to recur. This will have implications for antimicrobial therapy and management of affected individuals. Again, surveillance will assume an increasingly important role in the detection of these trends.
We identified an increased risk of invasive disease in adults over 60 years of age and in Aborigines. It is possible that immunisation of these groups could reduce the rate of disease. Previous studies in America have reported underutilisation of pneumococcal vaccine in the elderly and suggest vaccination of these individuals when they attend for influenza vaccination. It was also of interest to note a slight predominance of female cases and a small peak of cases in the 25 - 35 years age group. It is possible this is a reflection of maternal acquisition of \textit{S. pneumoniae} either carried by or infecting children.

Our data outline the epidemiology of invasive pneumococcal infections in Perth, Western Australia. Additional clinical information about predisposing factors, diagnosis, concurrent health problems, treatment and outcome would allow further identification of at-risk groups. A prospective surveillance system has recently been introduced to monitor patterns of invasive disease and determine serotypes of infecting strains. This will provide invaluable information when deciding which groups should be targeted for vaccination and whether current pneumococcal vaccines will provide maximal population coverage.

Acknowledgments

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References