

The relationship between influenza and invasive pneumococcal disease in the Northern Territory, 2005–2009

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TO THE EDITOR: Following the 1918 influenza pandemic, during which an estimated 50 million people died,¹ the relationship between influenza and secondary bacterial infections such as *Streptococcus pneumoniae* (pneumococcus) became recognised as an area of considerable scientific importance. It has been shown that influenza infection results in epithelial damage, up-regulation and exposure of respiratory tract receptors, alterations in the immune response and increased adherence of pneumococcus.²

The most severe form of pneumococcal infection, invasive pneumococcal disease (IPD), has a mortality of 7%–9%.³ It occurs frequently in the Indigenous population of the Northern Territory, with an incidence of 70.5 per 100 000, compared with 7 per 100 000 in Australia overall.³ In population studies, 11%–20% of seasonal increases in IPD have been attributed to influenza.^{4,5}

During the increase in influenza notifications in 2009 in the NT, as a result of pandemic H1N1 (2009) influenza, a concurrent increase in IPD notifications was also recorded. Our study aimed to investigate whether there was a relationship between influenza and subsequent IPD.

We calculated the relative risk of IPD in the 4 weeks after laboratory-confirmed influenza compared with the background risk. Cases were defined as patients who were diagnosed with IPD within 4 weeks of laboratory-confirmed influenza being diagnosed. Data concerning cases of influenza and IPD between 2005 and 2009 were

extracted from the NT Notifiable Diseases System and merged by date of birth using Stata, version 11 (StataCorp, College Station, Texas, USA), leading to 75 matches. The 75 matched records were checked individually to determine whether they met the case definition, resulting in eight potential cases. Demographic characteristics (name, sex, Indigenous status and address) were then checked to confirm the match. One case was excluded, leaving seven cases eligible for analysis.

We identified 2567 cases of influenza and 346 cases of IPD in the study period. The risk of IPD within 4 weeks of influenza was calculated as 7/2567 (2.7×10^{-3}). To calculate the background risk of IPD in a 4-week period, we first calculated the annual risk of IPD occurring without previous influenza (339/1 076 470 person-years) and divided this by the number of 4-week periods in a year (13). This gave a background risk of 2.42×10^{-5} and a relative risk of 112.5 (95% CI, 48.9–224.8; $\chi^2 = 758.3$; $P < 0.001$).

This analysis is based on population surveillance data, and is therefore limited by the possibility of ascertainment bias due to variations in influenza testing. Nevertheless, our study provides additional evidence that IPD is an important complication of influenza, and reinforces the need for clinicians to promote influenza vaccination in high-risk individuals and to be aware of complications which may occur in the weeks after the initial viral infection. Additionally, achieving high coverage of pneumococcal vaccination in at-risk groups will reduce the impact of influenza.

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