Austrian Syndrome: Triad of the Past or Harbinger of the Future?

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Austrian Syndrome: Triad of the Past or Harbinger of the Future?

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Abstract

Introduction
Austrian Syndrome is a rare complication of disseminated Streptococcus pneumoniae infection, consisting of pneumonia, meningitis, and endocarditis.

Case Description
A 64 year old male with a history of alcohol and tobacco abuse presented with a fever of 101.5°F and acute change in mental status. According to his wife, he had been coughing for 3 weeks. Physical examination noted left pleural and mydriatic decrease, decreased RUL breath sounds, and an irregular pulse. Chest x-ray confirmed RUL pneumonia and EKG showed atrial flutter. CT brain was negative for mass or herniation. Lumbar puncture was performed and blood cultures were obtained. Vancomycin and ceftriaxone were then initiated. He was intubated and transferred to the intensive care unit.

The cerebrospinal fluid and urine antigen tests were positive for Streptococcus pneumoniae, so dexamethasone was started as pneumococcal meningitis treatment. Subsequently, the blood cultures were noted to be positive for gram positive cocci. Transesophageal echocardiogram was ordered to rule out endocarditis, and it showed probable mitral valve vegetation. Later, multiple cranial nerve palsies and a brain empyema developed, with thoracic and lumbar emboil resulting in quadriparesis. The patient expired on hospital day 25.

Discussion
The lungs are the usual portal of entry for pneumococcus leading to pneumonia. There are many predisposing risk factors for invasive pneumococcal disease. Alcoholism is one of the strongest risk factors for pneumococcal endocarditis. The complications of Austrian syndrome include systemic embolization, valve perforation and abscess formation. Transesophageal echocardiography is preferred to transthoracic echocardiography in detecting vegetations. Treatment begins empirically with vancomycin and ceftriaxone until perivalvular abscess formation is confirmed. Surgery is usually performed for vegetations >10 mm, 1 or more embolic events during 2 weeks of antimicrobial therapy, increase in vegetation size despite appropriate antimicrobial therapy, valve perforation or rupture.

C) Perivalvular extension (valvular dehiscence/rupture, or fistula, new heart block, large abscess or extension to the abscess despite appropriate antimicrobial therapy).

Significance of Pneumococcal Antimicrobial Resistance
Currently, 15 to 30% of S. pneumoniae worldwide are multidrug-resistant (MDR). Despite increase in antimicrobial resistance worldwide over the past few decades, mortality rates for IPD have not increased.

Clinical failures often include factors independent of the pneumococcal antimicrobial susceptibility. These include:
A) Host factors (e.g, comorbidities: extremes of age, or underlying immunosuppression).
B) Pneumococcal virulence (e.g, capsular subtype).
C) Mortality rates are also higher in the presence of multilobar involvement, hypoxemia, renal insufficiency, and the need for ICU care.

Given the above confounding factors, dissecting out the impact of antimicrobial resistance on clinical outcomes is difficult, if not impossible.

Vaccination and its Importance
In the United States, about 39,750 cases of IPD and 4,000 deaths occur annually. The clinical spectrum from colonization to IPD depends on the pneumococcal capsular serotype. Currently, 94 capsular serotypes have been identified. Six serotypes (i.e., 4, 6B, 9V, 14, 19F, and 23F) account for >80% of IPD in children and >50% of IPD in adults in the United States. They also account for the majority of IPD in Europe.

Since the introduction of the PCV7 (4, 6B, 9V, 14, 18C, 19F, 23F), the rate of IPD due to PCV7 serotypes has declined significantly in many countries. In the US, it decreased from 64% of invasive and 50% of noninvasive isolates in 1999–2000 to 3.8% and 4.2%, respectively, in 2010–2011.

The PCV7 also has indirect (herd) effects that have led to decreased incidence of vaccine serotypes disease in unvaccinated children and adults.

However, there have been reports of an increase in non-PCV7 serotypes, especially 19A. This phenomenon is termed ‘replacement’.

PCV13 adds pneumococcal serotypes 1, 3, 5, 6A, 7F, and 19A to PCV7’s serotypes to provide coverage for over 85% of epidemiologically significant pneumococcal serotypes in the United States and throughout the world.

Austrian Triad and Vaccination
Reports of pneumococcal endocarditis often failed to provide adequate data on either the infecting pneumococcal serotype or patients’ vaccination histories.

Among the limited data we have, Aronin et al showed that the most common capsular serotypes that were identified causing Austrian triad were 12, 1 and 8. Hence, PCV 13 could potentially prevent a significant number of Austrian syndrome cases.

Future Prospects
The development of effective conserved pneumococcal protein vaccines (CPPV) that would not target the polysaccharide capsule are currently in trials. These may prevent pneumococcal disease and carriage without resulting in the selective pressure that is thought to drive serotype replacement.

References

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