Community-acquired Pneumonia Among Children in Taiwan

Community-acquired pneumonia is a common disease and the leading cause of death among children worldwide. In Taiwan, Streptococcus pneumoniae, Mycoplasma pneumoniae, and viruses are equally common etiologic pathogens in healthy children with community-acquired pneumonia. Historically, pneumonia caused by Streptococcus pneumoniae usually resolved completely without destructive sequelae in spite of intense inflammation during pneumococcal infection. However, the incidence of complications of pneumococcal pneumonia, including necrotizing pneumonia and/or empyema, has increased among children in the past decade. Before the use of pneumococcal conjugate vaccine in Taiwan, serotype 14 was the most common serotype that caused necrotizing pneumonia and/or empyema. Since 2009, there has been a shift from serotype 14 being the predominant serotype to serotype 19A. The same shift occurred for the genotype ST46, a clone associated with necrotizing pneumonia and empyema, being the predominant genotype previously to genotype ST320. Serotype 19A ST320 pneumonia had a significantly higher risk of lung necrosis and air leak. The evolution of serotype 19A ST320 into a highly virulent strain with high-level resistance to antimicrobial agents is a great challenge in treating pneumococcal disease. The optimal pneumococcal conjugate vaccine must contain serotype 19A to prevent this serious complication in children in Taiwan and elsewhere.

M. pneumoniae is a common pathogen responsible for pediatric community-acquired pneumonia, accounting for 10–40% of cases. The illness is usually self-limiting, with symptoms lasting several weeks, but sometimes it causes severe pneumonia. Although there is still insufficient evidence to show that antibiotics are effective in children with pneumonia caused by M. pneumoniae, macrolides are the drug of choice for treating children with M. pneumoniae infection. Since 2000, macrolide resistance in M. pneumoniae strains has been increasing since the report of a pediatric patient infected by macrolide-resistant M. pneumoniae in Japan. Disease caused by macrolide-resistant M. pneumoniae has a longer duration of fever than that caused by macrolide-susceptible M. pneumoniae. The mechanism of resistance to macrolides in M. pneumoniae frequently involves mutations in the 23S rRNA including transitions of A2063G and A2064G. The mutation at A2063G is more common than A2064G, but the mutation at A2064G confers resistance to a broader spectrum of other antimicrobial agents. Wang et al reported that M. pneumoniae was the most frequent cause of fatal community-acquired pneumonia in a medical center in Taiwan. A proportion of M. pneumoniae pneumonia was noted to present with persistent fever, hypoxemia, and radiographic deterioration in spite of macrolide usage. Pleural effusion, necrotizing pneumonitis, lung abscess, bronchiolitis obliterans organizing pneumonia, and acute respiratory distress syndrome have been reported in association with M. pneumoniae in children. Severe lung injury in M. pneumoniae pneumonia was presumed to be a consequence of overproduction of cytokines and an exuberant cell-mediated immune response. Clinical experiences and animal models suggest that the use of both antimicrobial therapy and immunomodulatory agents is important to improve the outcome of severe M. pneumoniae pneumonia. In contrast, extracorporeal membrane oxygenation therapy could be considered to treat patients with acute respiratory distress syndrome caused by M. pneumoniae. The percentage of macrolide-resistant M. pneumoniae strains in Taiwan was 23% in a multicenter study. Given the increase of macrolide-resistant M. pneumoniae in recent years, effective antimicrobial treatment should be a critical component of managing severe cases of macrolide-resistant strain infection. More studies to evaluate the optimal treatment in severe cases of M. pneumoniae pneumonia regardless of macrolide resistance are warranted.

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