

# Nosocomial Meningitis: Moving beyond Description to Prevention

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See Article on Page 171-179

Nosocomial bacterial meningitis may result from invasive procedures (for examples, such as craniotomies, the placement of internal or external ventricular catheters, lumbar puncture, intrathecal infusions of medications, or spinal anesthesia), head trauma, or hospital-acquired bacteremia complicated by metastatic infection [1]. The specific bacteria that cause nosocomial meningitis vary according to the pathogenesis and timing of infection after the predisposing event, so the choice of empirical antimicrobial therapy depends on the pathogenesis of the infection. In particular, the antimicrobial susceptibility profiles for locally common gram negative bacilli must be considered in the approach to empirical therapy [2]. However, there has been no report of nosocomial meningitis in Korea, apart from a number of case series.

In this issue of the Korean J Intern Med, Kim et al. [3] described the epidemiology of nosocomial meningitis and its outcomes. They reported that the most common organisms by rank were coagulase-negative staphylococci, *Acinetobacter* species, and *Staphylococcus aureus*. Most (78/91, 86%) of the infections were related to the placement of an external ventricular drain (EVD).

In the recent literature, the incidence of EVD-related infections ranges from 2% to 27% [4]. In Korea, the re-

ported rate of surgical site infection (SSI) was 3.09-3.68 per 100 operations in nationwide prospective multicenter studies of craniotomy in 2008 and 2009 [5,6]. While the incidence of EVD-related infections and the SSI rates after craniotomy in Kim et al. [3], were not given, the prevention of EVD-related ventriculomeningitis is of paramount importance because it is a significant cause of morbidity and mortality in critically ill neurological patients. Relevant risk factors have been identified as the duration of catheterization, frequency of EVD manipulation, intraventricular hemorrhage, and insertion techniques [4]. Therefore, the avoidance of modifiable risk factors should be emphasized.

*Acinetobacter* species are nosocomial pathogens of increasing importance, even in post-neurosurgical meningitis, with mortality from this infection exceeding 15% [7]. Alarmingly, *Acinetobacter* was the second most common cause of nosocomial meningitis in this study. In fact, *Acinetobacter* can survive in a wide range of environments and persist for extended periods of time on surfaces, which make it a frequent cause of outbreaks of infection and an endemic, healthcare-associated pathogen [8]. *Acinetobacter* is easily transmitted via direct contact with colonized patients or the hands of hospital staff, and even by respiratory droplets. Furthermore, antimicrobial therapy for these pathogens has become problematic, as they are frequently resistant to expanded-spectrum cephalosporins and carbapenems. Given the emergence of carbapenem-

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resistant *Acinetobacter* species, the therapeutic options are limited and no optimal treatment has been established [9]. Consistent with these findings, Kim et al. [3] reported that 26% of *Acinetobacter* species were resistant to imipenem and 27% of the patients infected with them died. Therefore, infection-control measures should be enforced vigorously to prevent person-to-person spread and cross-infection from the environment.

In conclusion, nosocomial bacterial meningitis poses a substantial challenge because of the emergence of disease caused by multidrug resistant organisms (MDROs) and severely limited treatment options. Encouraged by recent progress in reducing the incidence of infections associated with invasive devices, we should now turn our attention to preventing and controlling infection due to MDROs. The time is right to make effective prevention the new status quo.

#### Conflict of interest

No potential conflict of interest relevant to this article was reported.

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