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Young Ah Kim, M.D., Ph.D.
Department of Laboratory Medicine, National Health Insurance Service Ilsan Hospital, Goyang, Korea

Importance of the Molecular Epidemiological Monitoring of Carbapenem-Resistant *Pseudomonas aeruginosa*

Pseudomonas aeruginosa, a non-fermentative, aerobic gram-negative bacillus, is one of the leading causes of severe healthcare-associated infections in immunocompromised patients [1]. It is the etiologic agent of various nosocomial infections, including sepsis, pneumonia, and urinary tract infections [1]. *P. aeruginosa* frequently exhibits resistance to carbapenems [2, 3]. Carbapenem-resistant *P. aeruginosa* (CRPA) is considered a critical-priority pathogen for the development of new antimicrobials [4]. Common resistance mechanisms in CRPA isolates include porin loss, efflux pump expression along with extended-spectrum β-lactamase production, and AmpC hyperproduction [5]. In addition, resistance can be mediated by the production of carbapenemase from acquired resistance genes [5].

In this issue, Kim *et al*. [6] provide a compelling look into the molecular epidemiology of CRPA isolates from three Korean hospitals in the Gyeongsang and Jeolla provinces collected between 2011 and 2019. Their analysis of 155 carbapenem-non-susceptible *P. aeruginosa* isolates elucidates the clonal distribution and resistance mechanisms of these isolates, revealing a complex scenario wherein high-risk clones, such as ST235 and ST111, are predominant; however, sporadic sequence types are more prevalent. The study by Kim *et al*. [6] underscores the critical role of molecular epidemiological monitoring in understanding and combating the spread of CRPA. By identifying specific resistance mechanisms, such as the presence of the metallo-β-lactamase genes bla_{IMP-6}, bla_{VIM-2}, and bla_{NDM-1}, and their associations with high-risk clones, this study not only maps the current landscape but also aids in predicting future trends of antibiotic resistance.

In a previous study, ST235 and ST111 were identified as particularly worrisome strains that produced not only class B but also class A and D carbapenemases [5]. ST111 has been identified in all six continents except Oceania [5]. The widespread clone ST235 is often associated with poor clinical outcomes because of its multidrug resistance and virulence factors [5]. ST235, which is a dominant clone in Korea, harbors bla_{IMP-6} and therefore, shows extensive multidrug resistance [7]. The prevalence of this clone limits therapeutic options in Korea [7]. The frequent use of meropenem in clinical settings may have contributed to its spread.

Recently, a clonal shift was observed with the emergence of *P. aeruginosa* ST773 harboring bla_{NDM-1} in hospitals in the Gyeongsang province [8]. This clone additionally harbors *rmtB*, which confers resistance to amikacin; this complicates treatment as the combination of carbapenems and amikacin has become a crucial option to combat multidrug-resistant or extensively drug-resistant *P. aeruginosa* [8].

Antimicrobial resistance because of carbapenemase genes poses greater challenges than resistance mechanisms such as membrane impermeability because of the possibility of horizontal gene transfer. Therefore, nationwide monitoring of carbapenemase genotypes of CRPA isolates is essential for establishing

an efficient strategic policy to control antimicrobial resistance. Further attention is required to curb the emergence and spread of new CRPA clones.

AUTHOR CONTRIBUTIONS

Kim YA contributed to all processes of this manuscript.

CONFLICTS OF INTEREST

None declared.

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Corresponding author: Young Ah Kim, M.D., Ph.D.
Department of Laboratory Medicine, National Health Insurance Service Ilsan Hospital, 100 Ilsan-ro, Ilsandong-gu, Goyang 10444, Korea
E-mail: yakim@nhimc.or.kr

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