Clinical challenges and treatment options for carbapenem-resistant acinetobacter infections.

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Introduction

Acinetobacter species have gained increasing recognition as significant pathogens involved in hospital-acquired and healthcare-associated infections on a global scale. These infections manifest in various forms, including ventilator-associated pneumonia, bloodstream infections, wound infections, and urinary tract infections. Notably, Acinetobacter is a leading causative agent of ventilator-associated pneumonia in the United States. Within the genus Acinetobacter, comprising over 30 genomic species, Acinetobacter baumannii stands out as the most clinically relevant due to its relative virulence and high degree of multidrug resistance. Additionally, Acinetobacter nosocomialis and Acinetobacter pittii are increasingly implicated in healthcare-associated infections [1].

However, conventional biochemical identification methods in clinical microbiology laboratories often cannot distinguish A. baumannii from other Acinetobacter species. This necessitates genetic methods, which are not routinely available in clinical settings. It's important to note that in clinical studies, "A. baumannii" may refer to the genomic species itself or, more commonly, the broader A. baumannii complex, encompassing all four genomic species mentioned above [2].

A hallmark characteristic of A. baumannii is its remarkable ability to develop resistance to multiple classes of antimicrobial agents. While the term Multidrug Resistance (MDR) is commonly used, it technically denotes non-susceptibility to at least one agent in at least three antimicrobial categories. This definition may apply even to relatively antimicrobial-susceptible A. baumannii isolates. For instance, resistance to ceftriaxone, ciprofloxacin, and trimethoprim-sulfamethoxazole is sufficient to classify an isolate as MDR, leaving several treatment options available. In contrast, Extensive Drug Resistance (XDR) signifies non-susceptibility to at least one agent in all but two antimicrobial categories, carrying more significant clinical implications. For example, an isolate that is susceptible to colistin and tigecycline but resistant to all other tested categories would be deemed XDR. These XDR isolates are increasingly encountered in clinical practice [3].

There is a growing apprehension regarding the development of resistance when using tigecycline as a monotherapy for treating A. baumannii infections. Notably, two distinct cases have been documented in the literature, both involving tigecycline monotherapy. In one instance, a patient with ventilator-associated pneumonia received tigecycline monotherapy for an initially susceptible A. baumannii isolate, only to later develop a resistant isolate in the lungs, marked by a significant. In the second case, a patient with a susceptible urinary isolate was treated with tigecycline monotherapy, but subsequently faced a recurrent infection, presenting as pneumonia and an epidural abscess, with a tigecycline-resistant isolate. These occurrences raise concerns about the effectiveness of tigecycline monotherapy in eradicating A. baumannii infections [4,5].

Conclusion

It is possible that sub therapeutic concentrations at the infection sites, influenced by tigecycline's pharmacokinetics, contributed to the emergence of resistance in these cases. Given these apprehensions, it is imperative to reconsider the role of tigecycline in pathogen-specific therapy for A. baumannii infections, especially in the context of immunotherapy and at the presently approved dosage.

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