the clinical relevance of our findings. It would be easier to respond had Dr. Stollerman provided precise definitions of the terms “virulent strains” and “less virulent strains.” The strains included in our studies were all clinical pharyngitis isolates. In fact, strains 556, 558, and 568 were all clinical isolates from patients with uncomplicated streptococcal pharyngitis. These isolates were not associated with severe or systemically invasive infections. Furthermore (and most importantly), these 3 strains, which were studied in detail, were from individuals who experienced treatment failure and, thus, might well have been associated with intracellular invasion—just the kind of group A streptococcal (GAS) isolates that are met daily by clinicians. We wonder how Dr. Stollerman would define these strains and what criteria, in this situation, he would use to distinguish “virulence”?

Dr. Stollerman indicates that “penicillin is extremely effective in terminating clinical GAS infection” [p. 763, 1]. No one would dispute this; there has never been a clinical isolate of GAS that has proven to be resistant to penicillin in vitro. However, at the same time, our previous studies have recorded almost a 40% rate of failure by penicillin (either oral penicillin VK or intramuscular benzathine penicillin G) to eradicate GAS from the upper respiratory tract [3]. This can be interpreted, if we are correct, to mean that perhaps there are a significant number of individuals presenting with uncomplicated pharyngitis with GAS isolated from throat cultures who have GAS that is intracellular.

We appreciate Dr. Stollerman’s concern about “low-grade (nonexudative) GAS pharyngitis” [p. 763, 1], although he does not define this state. Krause and Rammelkamp [4] brought this issue to attention >40 years ago in their studies involving monkeys. Yet, even strains that appeared to be less virulent in monkeys recovered their capacity to express M protein after in vivo passage. So, even if we adopt that partial definition of virulence, there is little documentation to promote the belief that so-called “nonvirulent” clinical isolates would be permanently fixed in that physiological and/or pathogenetic state. Very little is known about the physiological state of intracellular GAS.

Finally, we fully agree that clinicians should look before they leap with regard to clinical implications. The initial sentence of the second to last paragraph of our article clearly states that “our data, however, should not be interpreted as indicating that macrolides or azalides are clinically more effective in eradicating GAS from the upper respiratory tract” [p. 1405, 2].

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References


IMP-4–Producing Pseudomonas aeruginosa in a French Patient Repatriated from Malaysia: Impact of Early Detection and Control Measures

To the Editor—We read with great interest the article by Peleg et al. [1] that reported on an outbreak of bacterial strains that produced the metallo-β-lactamase (MBL) gene IMP-4 in an Australian hospital. In 2004, they identified the blα IMP-4 gene—which had been previously described in Acinetobacter species and Citrobacter youngae isolates from Hong Kong and China [2, 3]—in a Pseudomonas aeruginosa isolate in Australia [4]. After this identification, the gene rapidly disseminated in their hospital, resulting in an outbreak involving 5 different gram-negative species [1]. As indicated by Rossolini in the editorial commentary of the same issue [5], blα IMP-4 was likely imported from Southeast Asia via international travelers, which underscores the role of human movement in the intercontinental spread of MBL genes. In this letter, we report an IMP-4–producing P. aeruginosa isolate from a French patient repatriated from Malaysia, and we describe strategies of detection and infection control used for preventing its intrahospital dissemination.

Before being repatriated to France in August 2006, the 75-year-old patient had been hospitalized in an intensive care unit in Malaysia for 23 days following a cerebrovascular accident and had received multiple antibiotic therapies, including a 12-day course of meropenem for pneumonia. On admission to the emergency department of our hospital, the patient presented with right hemiplegia; had a tracheotomy canula, a urinary catheter, and a nasogastric tube; and was receiving oral amoxicillin-clavulanate. Because of his prior hospitalization, a rectal swab was immediately performed for detection of multiresistant bacteria. Culture yielded a multiresistant P. aeruginosa isolate with an unusual β-lactam phenotype that was characterized by resistance to ticarcillin,
The MBL determinant was subsequently identified as \( \text{bla}_{\text{IMP-4}} \) by molecular techniques [7]. The patient, who had just been transferred to the neurosurgery department, was moved to a single room, and contact barriers were implemented. Antibiotic treatment was discontinued, and invasive devices were removed as soon as possible to decrease the risk factors for \( P. \text{aeruginosa} \) infection. Forty days after admission, when discharge from the hospital was scheduled, a rectal swab was performed and determined to be negative for IMP-4–producing isolates. However, isolation measures were maintained until discharge, 20 days later. No further MBL-producing isolate has been identified in our hospital so far.

MBL genes are widespread in some geographical areas, particularly Southeast Asia, whereas they remain uncommon in other countries [8]. So far, very few MBLs have been reported in France, and all of those that have been identified were Veronese imipenem–type enzymes [9, 10]. Our report highlights the importance of an active surveillance strategy in countries where MBLs are absent or uncommon. Patients transferred from high-risk areas should be screened on admission, and isolation precautions should be implemented until culture results are available. Such measures are critical to prevent these enzymes from spreading worldwide, as occurred with extended-spectrum \( \beta \)-lactamases.

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Community-Acquired \( Listeria \) \( monocytogenes \) Meningitis in Adults

To the Editor—Brouwer et al. [1] should be congratulated for their evaluation of 30 episodes of listerial meningitis in adults, which was the first prospective study of this problem to be published and is a valuable addition to the literature.

Brouwer et al. [1] state that the symptoms and signs of patients with \( Listeria \) \( monocytogenes \) meningitis were “not different from those found in the general population of patients with community-acquired bacterial meningitis,” and claim that this “is in contrast with previous reports, which stressed the importance of atypical presentation” [1, p. 1237]. A publication I authored [2] is cited as one of the “previous reports.” In my article, I specifically stated, “Meningitis due to \( L. \) \( monocytogenes \) is usually clinically similar to that due to more common etiologic agents” [2, p. 4]. I then summarize, in tabular form, some features particular to listerial meningitis. These features do not differ in any significant way from those reported by Brouwer and colleagues. For example, my table shows that the presentation of \( L. \) \( monocytogenes \) meningitis is usually acute but may be subacute; in the Brouwer and colleagues series, 27% of patients had symptoms for >4 days prior to presentation. My table shows that nuchal rigidity is less common in \( L. \) \( monocytogenes \) meningitis than in other more common bacterial etiologies and cites an absence of nuchal rigidity of 15%–20%; Brouwer and colleagues report an absence of neck stiffness for 27% of patients with \( L. \) \( monocytogenes \) meningitis. My table states that the CSF Gram stain result was negative for most patients (organisms were seen in ~40%); in the Brouwer and colleagues series, the CSF Gram stain result was negative for 60% of the patients.

My point is that I did not emphasize “the importance of atypical presentation,” but rather attempted to draw attention to some ways in which listerial meningitis may differ from other causes of bacterial meningitis. Regarding this point, the data presented by Brouwer et al. [1] do not differ in any meaningful way from my assessment from almost 10 years ago.