ceftazidime, and imipenem (MIC, >32 mg/L); intermediate susceptibility to aztreonam; and susceptibility to piperacillin. Such a phenotype suggested MBL production, which was confirmed by the immuno-EDTA double-disk synergy test [6]. The MBL determinant was subsequently identified as bla<sub> extended</sub>-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 <i>P. aeruginosa</i> 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 imipenemase–type enzymes [9, 10]. Our report highlights the importance of an active surveillance strategy in countries where MBLs areAbsent 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 b-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 <i> Listeria monocytogenes </i> 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 <i>L. monocytogenes</i> is usually clinically similar to that due to more common etiologic agents” [2, p. 4]. I then summarize, in tabular form, several 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 <i>L. monocytogenes </i> 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 <i>L. monocytogenes </i> 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 <i>L. monocytogenes </i> 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.

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High Colonization Pressure Might Compromise the Efficiency of Routine Methicillin-Resistant Staphylococcus aureus Screening

To the Editor—Routine screening for methicillin-resistant Staphylococcus aureus (MRSA) in the intensive care unit is a widely recommended [1] and quite well-studied [2–4] intervention. Yet, the recent study by Huang et al. [5] finally imparts a clinical imperative (the reduction of bacteremias) to the old epidemiological rationale of MRSA transmission control.

The study’s sequential design allows for the assessment of multiple interventions, and its unique and very astute monitoring of methicillin-susceptible S. aureus bacteremias as a control excludes the possibility of natural fluctuations or other confounding factors, which were not accounted for in previous studies [2–4].

Thus, it is all the more deplorable that Huang and colleagues did not provide an estimation of the MRSA colonization pressure during the study interval. Colonization pressure is an important risk factor for MRSA acquisition in the intensive care unit [6]. A study of vancomycin-resistant Enterococcus transmission [7] concludes that a high colonization pressure may supersede the effect of other transmission variables, including infection-control measures. It does not seem unreasonable to extrapolate this phenomenon to MRSA, especially in light of high rates of gut carriage of the organism in colonized patients [8].

The MRSA carriage prevalence and incidence reported in the article by Huang et al. [1] evoke low colonization pressure. Indeed, no successful control of the spread of MRSA has been achieved by screening strategies in an environment of high colonization pressure (i.e., >50%).

Future studies of MRSA control in the intensive care unit need to report the colonization pressure that is prevalent in the study population, because the findings reported might not be applicable to intensive care units with high colonization pressure. In these cases, other, more aggressive measures might be required [8].

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