Methicillin resistance among staphylococci isolated from patients in northern Egypt has escalated alarmingly in the past decade. Data about the prevalence of fusidic acid (FA) resistance in Egyptian clinical isolates are limited. This work investigates the prevalence and mechanism of FA resistance among 81 methicillin-resistant staphylococcal isolates from major hospitals of Alexandria, Egypt. Some combinations for treating infections due to resistant isolates were studied. Twenty-six isolates (32.1%) were FA resistant (minimum inhibitory concentrations [MICs] = 2-1,024 mug/ml), and fusB and fusC genes coding for FA resistance were detected in 30.77% and 34.62% of the FA-resistant strains, respectively. One highly resistant isolate, S502 (MIC = 1,024 mug/ml), possessed both genes. Plasmid curing resulted in fusB loss and MIC decrease by 16-64 folds. Conjugation caused acquisition of FA resistance among susceptible isolates. Serial passages in subinhibitory FA concentrations produced mutants with increased MIC by 4-32 folds. The combination of FA with rifampin, gentamicin, or ampicillin/sulbactam, in a subinhibitory concentration, was synergistic against the isolates, including serial passage mutants, decreasing number of survivors by an average of 2-4 logs. A relatively moderate rate of FA resistance was detected in Alexandria hospitals. Combination therapy with gentamicin, rifampin, or
ampicillin/sulbactam is crucial to preserve the effectiveness of FA.