Introduction

Staphylococcus aureus is a major cause of hospital- and community-associated infections worldwide. This microbe has the capacity to develop resistance to antibiotics through a large variety of resistance mechanisms. Traditional antibiotic susceptibility testing reveals only the phenotypic characteristics. Due to inducible phenotypes and variable protocols, interpretation of phenotypic susceptibility data is complicated. DNA microarray technology allows simultaneous detection of many different resistance genes. Some bacteria do not express their resistance genes unless by tight regulation and presence of selective antibiotic pressure. As genotyping techniques become realistic options for routine laboratory susceptibility testing, inconsistencies between phenotypic and genotypic resistance patterns are important to detect and resolve because they may have therapeutic consequences. Our objective was to examine whether antibiotic susceptibility patterns determined by disk diffusion reflect the genotypic resistance patterns determined by DNA microarray technology in S. aureus blood culture isolates.

Material and Methods

We included 108 S. aureus isolates detected in blood cultures taken from bacteremic patients in 2011-12 at Akershus University Hospital, Norway. Antibiotics for 11 common antibiotics obtained using the EUCAST disk diffusion sensitivity testing protocol (1) were compared to the presence of genes associated with resistance towards the same antibiotics. Genotypes were obtained from diagnostic DNA microarrays (Alere Technologies, Jena, Germany) as previously described (2). Beta-lactamase production was also assessed using a standard acidometric agar plate method (3), and both zone diameter and sharpness of the zone edge was taken into account in the disk diffusion test for benzylpenicillin. Discrepancies between genotype and phenotype resulted in repeated phenotypic testing with the same methods on isolates recovered from frozen storage.

Results

The agreement between the disk diffusion method and genotyping was 93.5% before phenotypic reanalysis and 97.2% after. Six of seven isolates showing discrepancies regarding beta-lactamase, were corrected after reanalyses. The remaining isolate contained the blaZ gene, but was susceptible to penicillin according to both disk diffusion and acidometric tests. Five isolates showed phenotypic resistance to fusidic acid without harbouring the genes far1 (fusB) or Q6GD50 (fusC), but this was reduced to one isolate after reanalysis. Two isolates changed from resistant to susceptible phenotypes towards erythromycin and trimethoprim-sulfamethoxazole when being retested – the isolates contained no resistance genes towards the two antibiotics.

Conclusion

Phenotypic and genotypic resistance profiles were coherent in all S. aureus isolates with two exceptions. One isolate harboured the blaZ gene, but no beta-lactamase production was detected phenotypically. The remaining isolate carried the far1(fusB) or Q6GD50 (fusC), but this was reduced to one isolate after reanalysis. Two isolates changed from resistant to susceptible phenotypes towards erythromycin and trimethoprim-sulfamethoxazole when being retested – the isolates contained no resistance genes towards the two antibiotics.