Methicillin-resistant Staphylococcus lugdunensis carrying SCCmec type V misidentified as MRSA

ABSTRACT

Staphylococcus lugdunensis is a rare cause of severe infections and clinical manifestations are similar to those related to S. aureus infection. We describe a hospital-acquired bacteremia due to methicillin-resistant Staphylococcus lugdunensis, misidentified as methicillin-resistant S. aureus. The oxacillin MIC was 16 µg/mL and the mecA gene and SCCmec type V were determined by PCR. Although treatment had been appropriated, the patient died after rapid progressive respiratory failure and another nosocomial sepsis. It is important not only to identify S. lugdunensis in view of its clinical course, but also to determine its susceptibility to oxacillin by detecting the mecA gene or its product.

Keywords: Staphylococcus; methicillin resistance; bacteremia.

CASE REPORT

A 73-year old female with systemic arterial hypertension and chronic obstructive pulmonary disease (COPD) GOLD IV was admitted to the Emergency Department of the University Hospital ‘Clementino Fraga Filho’ (HUCFF), in March 2005. HUCFF, a tertiary-care teaching hospital with 490 beds, is affiliated to the Federal University of Rio de Janeiro, Rio de Janeiro City, Brazil. The patient had asthenia, cough, fever, purulent secretions and worsening of dyspnea, but the chest x-ray showed no pulmonary infiltrates. Diagnosis of acute exacerbation and upper respiratory infection were made. Bronchodilators, glucocorticoids, gatifloxacin and non-invasive ventilation were initiated. Respiration did not recover, and mechanical ventilation was needed. After completing the course of antimicrobial therapy the patient recovered. However, two cultures from blood collected at day 7 of therapy detected Gram-positive bacteria. Bacterial contamination of blood was initially suspected since the patient had improved and precise identification of the sites of blood collection was not possible. Initially, methicillin-resistant S. aureus (MRSA) and Streptococcus alpha hemolytic were identified in the hospital laboratory three days after blood collection and the strains were sent to our laboratory for confirmation. After the initial clinical improvement, the patient presented fever, leukocytosis and hypotension. The central line catheter was removed, and vancomycin and amikacin were included in the treatment, according to the local microbiology pattern and the previous isolation. The nosocomial sepsis was probably related to the central line catheter, although abdominal imaging were not performed. The transthoracic echocardiogram result was normal. Another set of blood cultures drawn on the day when sepsis was diagnosed detected Escherichia coli and S. epidermidis. The patient had fast and progressive respiratory failure and despite intensive medical treatment she expired on day 18 of hospitalization.

The isolate obtained from the first blood culture was characterized as MRSA in the hospital laboratory by VITEK (BioMerieux). Gram staining, catalase test and latex agglutination clumping factor and DNAse detection were performed as complementary tests. Antimicrobial susceptibility testing (VITEK, BioMerieux) revealed resistance to oxacillin, penicillin and chloramphenicol.
In our laboratory, the isolate presented slightly yellow colonies and showed beta-hemolysis on sheep blood agar. It gave a positive reaction for clumping factor, but the tube coagulase test was negative. Even, the isolate was evaluated using the method based on the conventional reference tests and the ornithine decarboxylase production, an enzyme produced only by S. lugdunensis strains. To confirm the species identification, the fbl gene, which encodes a fibrinogen binding protein of S. lugdunensis was amplified by PCR as previously published. A 425 bp amplicon was obtained after PCR amplification of template DNA extracted from reference strain S. lugdunensis DSMZ 4804 and the clinical isolate. Antimicrobial susceptibility was evaluated according to CLSI guidelines. The isolate was resistant to oxacillin, clindamycin, tetracycline, chloramphenicol, and gentamicin. Oxacillin MIC, determined by the E-test method, was 16 μg/mL. Initially, the mecA gene was detected by PCR, and the SCCmec typing was performed by multiplex PCR of the ccr type and mec class, using the method described by Kondo et al. The e isolate was ccr type 5 and mec class C, characterizing the SCCmec type V. The e 804 bp amplicon (corresponding to ccr5 gene) and 518 bp (corresponding to class C mec gene) generated in PCR reaction were confirmed by sequencing and comparison with GenBank database.

The e is the first case report of a methicillin-resistant S. lugdunensis (MRSL) isolate carrying the SCCmec type V causing a systemic infection. To our knowledge, this is the fourth report in the literature describing S. lugdunensis carrying the mecA gene. Kawaguchi et al. detected the mecA gene in one of two strains of S. lugdunensis isolated from blood during a laboratory surveillance in Japan, but no description of clinical infection was provided by them. In 2003, Tee et al. reported a case of MRSL causing bloodstream infection in a neonate. In 2008, Tan, Ng and He found e (4.7%) S. lugdunensis strains carrying the mecA gene in a collection of 106 clinical isolates. In Brazil, this is the first report of a MRSL isolate. S. lugdunensis was first described by Frenery and is associated with several infections, mainly skin/flora tissue infections and endocarditis. In the clinical setting it frequently causes illness that has been described as acute, aggressive and destructive - more in keeping with coagulate-positive staphylococci than characteristically indolent coagulate-negative staphylococcal infections, especially endocarditis, where the majority of infections involve native or prosthetic valves, being the mortality rates high. Therefore, its aggressive nature resembles S. aureus. S. lugdunensis should be suspected when a staphylococcal isolate is positive in the clumping factor test but negative in the tube coagulase test. E isolation rates of S. lugdunensis are about 10% for non-S. epidermidis coagulate negative isolates. However, in a recent study, Böcher et al. showed that the misidentification of S. lugdunensis could be contributing to this low rate. They improved the identification of S. lugdunensis using the Columbia sheep-horse agar inoculation as an additional test. To reinforce this fact we evaluated the bacterial diagnostic provided for 5,360 blood cultures performed in our hospital, between 2000 and 2009 (data not shown). No S. lugdunensis isolate was detected, suggesting that this pathogen is being misidentified.

In the present case report we observed a clear misidentification of a MRSL isolate as community MRSA. Both species present similar colony morphology and blood-agar hemolysis. Interestingly, in addition to being positive by the clumping factor agglutination test, S. lugdunensis strains are also positive for DNase, like S. aureus strains. E is shows the importance to employ tube coagulase testing for staphylococcal characterization. For confirmation the ornithine-decarboxylase test is also important, since S. lugdunensis is the only staphylococcus positive in this assay. E susceptibility pattern of the isolate indicated a community-associated pathogen. Only resistance for β-lactams and chloramphenicol were observed. Moreover, this non-multiresistance has been related to the SCCmec type V, often found in community isolates. E is case report confirms the isolation of non-multiresistant S. lugdunensis carrying the SCCmec V as a cause of a serious hospital-acquired infection which was misdiagnosed as non-multiresistant MRSA in a setting with nosocomial transmission of the latter.

REFERENCES