Original Article

Prevalence of community-acquired methicillin-resistant *Staphylococcus aureus* in patients with skin and soft tissue infections

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ABSTRACT

Introduction: In the early nineties, infections due to methicillin-resistant *Staphylococcus aureus* (MRSA) in patients without previous healthcare exposure were reported. The continued evolution of MRSA is illustrated by the infections caused by community acquired MRSA and the majority of these infections are nonlife-threatening infections of the skin and soft tissues. We carried out the retrospective study of bacterial isolates obtained from pus specimens of community-acquired skin and soft tissue infections in our set up with special reference to MRSA. **Materials and Methods:** The isolation rate of various organisms isolated from pus specimens was recorded for the years 2007 to 2012. The antibiotic susceptibility patterns of *S. aureus* strains (MRSA and MSSA) were also reviewed. **Results:** 47.48% of the total pus samples received from patients with a clinical diagnosis of community acquired SSTI during the period of 6 years, that is, from 2007 to 2012, showed culture positivity. Mixed organisms were isolated from five samples. 30.21% of them were *S. aureus* strains out of which 23.80% demonstrated the presence of methicillin resistance (MRSA). All the MRSA strains (100%) screened from clinical specimens were resistant to penicillin, cephalexin and cefazolin; 40% to erythromycin, clindamycin and amikacin; 80% to gentamicin; 90% to ofloxacin. The isolation rate of MRSA is far outnumbered by that of MSSA that remains fairly sensitive to the first line drugs against *S. aureus*. **Conclusion:** Abscesses are the most common clinical presentation caused by CA-MRSA in this study and we recommend that physicians should consider obtaining cultures and antimicrobial susceptibility tests in all such patients.

Key words: Antimicrobial susceptibility test, community-acquired MRSA, skin and soft tissue infections

INTRODUCTION

Methicillin-resistant *Staphylococcus aureus* (MRSA) are generated when methicillin-susceptible *S. aureus* (MSSA) acquire the mecA gene, which is carried on a mobile genetic element known as staphylococcal cassette chromosome mecA (SCCmecA).[1] Since their initial description in 1961, a number of clones of MRSA have spread widely throughout the world. Prior to the 1990s most MRSA were associated with hospitals or other healthcare units, but, beginning in the early nineties, infections due to MRSA in patients without previous healthcare exposure were reported from six continents, including Australia, where several outbreaks had been previously noted in Western Australia and the Northern Territory.[2,3] Those strains identified in

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the community among patients who may or may not have the predisposing factor for nosocomial MRSA infections are called community-acquired MRSA (CA-MRSA). Some studies arbitrarily suggest that if MRSA is identified 48-72 h after hospital administration, it is assumed that the patient brought the MRSA from the community into the hospital setting and this is commonly classified as CA-MRSA.[4] More frequent infections were noted in Taiwan, Canada, and especially the United States, where the epidemic of CA-MRSA infections took off with a vengeance. Initial infections in the USA were due to strains of ST1 lineage (also known as USA400 based on PFGE typing) and these were shown to contain an SCCmecA element (SCCmec IV) distinct from the elements I-III seen in most hospital-associated strains.[5] These organisms also contain genes encoding the Panton-Valentine leukocidin (PVL), which targets and damages the membranes of polymorphonuclear leucocytes. USA400 was rapidly replaced by another clone, ST8 (or USA300), which now accounts for 85% of the CA-MRSA isolates in the USA.[6] Studies have also shown the existence of CA-MRSA from Delhi, Mumbai, and other parts of India.[7-9] The continued evolution of MRSA is illustrated by the infections caused by CA-MRSA. Although these organisms are also capable of producing devastating disease in certain patients, the majority of these infections are nonlife-threatening infections of the skin and soft tissues.[10] Since most community-acquired SSTIs are primarily presented to a dermatologist in our set up, we reviewed the bacterial isolates of pus culture with special reference to MRSA, from OPD patients of the Department of Dermatology who were diagnosed with community acquired skin and soft tissue infections (SSTIs).

MATERIALS AND METHODS

This was a retrospective study of the bacterial isolates of pus culture with special reference to MRSA, from OPD patients of the Department of Dermatology who were diagnosed with community acquired SSTIs, carried out at the Routine Laboratory of Department of Microbiology, Maulana Azad Medical College, New Delhi. Patients were classified to have community-acquired SSTI if there had not been any previous hospitalisation or visit to a hospital for a period of one year. The causative organisms obtained from specimens of such patients and their antimicrobial susceptibility patterns were reviewed for the period 1st January 2007 through 31st December 2012. Our laboratory received pus samples requested for culture and antibiotic susceptibility test from all departments of Lok Nayak Hospital which is the associated teaching hospital of the college. Pus specimens were received along with the requisition slips in either a sterile container or a sterile swab and were inoculated onto 5% sheep blood agar and MacConkey agar media as well as brain heart infusion (BHI) broth. The clinical diagnosis of each specimen received was also recorded. The plates were examined for the growth of bacteria after 24 h of aerobic incubation of plates at 37°C (microaerophilic and anaerobic culture were not done unless it was requested for suspected cases by the clinicians). If no growth were observed on the plates, subcultures were made from the glucose broth onto 5% sheep blood agar and MacConkey agar which were observed after overnight incubation. The colonies of organisms were identified based on the colony morphology on blood agar, MacConkey's agar and the findings of Gram staining and were further subjected to a series of biochemical tests like catalase test (3% hydrogen peroxide reagent), oxidase test (disc from HiMedia), and sugar fermentation tests. All Staphylococcal strains were further tested for the production of free coagulase enzyme using tube coagulase test as per standard methods.[11] *S. aureus* NCTC 6571 of known coagulase production was included as control strain for the coagulase test. All confirmed *S. aureus* strains were subsequently tested for methicillin resistance based on Kirby–Bauer disk diffusion method using Cefoxitin discs (30μg) obtained from Hi-Media Laboratories Pvt. Ltd. The isolates were considered methicillin resistant if the zone of inhibition was less than 21 mm. Further, the antibiotic susceptibility patterns of methicillin resistant *S.aureus* strains were determined on the day of their isolation by the modified Stoke's disc diffusion method on Muller Hinton agar comparing the zones of inhibition of the test strain with that of the control strain to define sensitivity or resistance to different antimicrobials. *S. aureus* NCTC 6571 was used as reference strain for the standardization of antibiotic susceptibility testing. The antibiotics used were Penicillin-G (10 unit); Cephalexin (30 μg); Cefazolin (30 ug); Erythromycin (15 μg); Clindamycin (02 ug); Gentamicin (10 μg); Amikacin (30 μg); Ofloxacin (5 μg); Vancomycin (30 μg); Teicoplanin (30 ug); Linezolid (30 μg), and Chloramphenicol (30 ug). The test was interpreted as Sensitive (S), Intermediate susceptible (IS), or Resistant (R) in accordance with standard recommendation.[12] D-test for *S. aureus* was performed and the results were interpreted using NCCLS guidelines.[13]

RESULTS

During the 6-year study period from 1st Jan 2007 to 31st Dec 2012, our laboratory received a total of 139 specimens of pus from clinically diagnosed cases of community acquired SSTI from the Department of Dermatology. Of these, 66 (47.48%) specimens showed culture positivity including five specimens yielding >1 organisms, 73 specimens (52.51%) did not show any growth after 48 h of aerobic incubation. The age of the patients ranged from 6 years to 65 years with a mean age of 32 and a standard deviation of 15. The number of each organism isolated in [Table 1].

The Table 1 shows the isolation of 42 Staphylococcus aureus strains (30.21%) out of which 10 strains (23.80%) demonstrated the presence of methicillin resistance (MRSA)

Table 1: Year-wise distribution of various organisms isolated from pus specimens (SSTI)

Year	No of OPD samples	MSSA	MRSA (P) (%)	Pseudomonas aeruginosa	Streptococcus pyogenes	Members of Enterobacteriaceae	Acinetobacter spp
2007	15	05	00 (0)	01	01	02	00
2008	21	08	01 (4.76)	01	01	01	00
2009	13	00	01 (7.69)	03	00	01	00
2010	21	04	02 (9.52)	02	00	02	01
2011	38	07	02 (5.26)	01	00	04	00
2012	31	08	04 (12.90)	02	01	05	00
Total	139	32	10 (7.19)	10	03	15	01

P: Prevalence rate; MRSA: Methicillin-resistant *Staphylococcus aureus*; MSSA: Methicillin-sensitive *Staphylococcus aureus;* OPD: Out-patient department; SSTI: Skin and soft tissue infections.

and the remaining strains were considered as methicillin sensitive *S. aureus* (MSSA). The other organisms isolated were Pseudomonas spp. (7.19%), Streptococcus pyogenes (2.16%), Acinetobacter spp. (0.71%), and members of the Enterobacteriaceae family (10.79%). The most common infection type caused by CA-MRSA is an abscess which accounted for seven (70%) cases. The other infection types associated with CA-MRSA were cellulitis (20%) and impetigo (10%). The prevalence of MRSA rose from 0% in 2007 to 12.90% in 2012.

All the 10 MRSA strains (100%) screened from clinical specimens were resistant to penicillin, cephalexin and cefazolin; four strains (40%) to erythromycin, clindamycin, and amikacin; eight strains (80%) to gentamicin; nine strains (90%) to ofloxacin. However, all (100%) MRSA strains recorded sensitivity to vancomycin, teicoplanin, linezolid, and chloramphenicol and none of the MRSA strains showed intermediate susceptibility against any of the drugs tested. D-test was positive for four MRSA strains. In a pattern similar to MRSA strains, the MSSA strains (100%) isolated in our study too were penicillin resistant and susceptible to vancomycin, teicoplanin, linezolid, and chloramphenicol. The MSSA strains showed 84.37% sensitivity to Cephalexin; 96.87% to Cefazolin; 68.75% to Erythromycin; 93.74% to Clindamycin; 71.87% to Gentamicin; 87.50% to Amikacin; 31.25% to Ofloxacin.

DISCUSSION

Our study demonstrates that the proportion of patients with SSTI caused by community acquired MRSA is increasing on a yearly basis, and CA-MRSA is now a very common cause of community-acquired SSTIs in our centre which is New Delhi's largest and busiest tertiary care hospital. Recent reports suggest that CA-MRSA is becoming more common in many geographic areas in the United States and Europe.[14,15] The first Indian report of CA-MRSA infection was from this hospital complex reporting 14% isolation rate of CA-MRSA from infected patients.[7] Two years later Umashankar *et al.* reported 10.90% isolation rate of CA-MRSA from a study conducted at the Southern part of India.[8] Whereas a study conducted by Patil *et al.* showed only one out of 70 community-acquired *S. aureus* strains isolated to be methicillin resistant.[9] Not a single request for anaerobic culture was made in this study due to absence of clinical features suggestive of anaerobic infections. This clearly shows that the bacterial causes of common community-acquired SSTIs are generally facultative anaerobic organisms such as *S. aureus* and occasionally gram negative bacilli. Because of the predictable etiology of these infections, most physicians do not routinely obtain cultures from these patients. Obtaining cultures of SSTIs is now of greater importance to monitor the extent of CA-MRSA infections in one's community and guide therapy in areas in which CA-MRSA is already prevalent. Most community-acquired SSTIs are treated with antimicrobial drugs such as cephalexin. Patients requiring intravenous therapy are most commonly given agents such as cefazolin. In areas with a high prevalence of CA-MRSA, empiric treatment for SSTIs with β-lactam agents such as cephalexin may no longer be appropriate. Agents such as vancomycin, teicoplanin, linezolid and chloramphenicol should be considered for CA-MRSA. Inducible clindamycin resistance (D-test positivity) was found in few samples of our study and clinical failure due to inducible clindamycin resistance among CA-MRSA has been reported.[16] Macrolides, quinolones, and aminoglycosides have inconsistent activity against the MRSA isolates identified in our study and other reports of CA-MRSA.[14]

One of the limitations of our study is that our hospital serves a large area with low-income population which makes it difficult to follow up the patients. The previous hospitalisation records of the patients are often not available due to which a community-based prospective study would be an ideal approach to reflect the statistics of the general population. Moreover, it is common for the general population in our set up to self-prescribe antibiotics before presentation to the OPD, which is most likely the reason for a high percentage of non-cultureable SSTIs (52.51%) in this study. Abscesses are the most common clinical presentation caused by CA-MRSA in our study which is in accordance with the findings of Ruhe *et al.*[17] and Miller *et al.*[18] Although MRSA is now one of the most common pathogens isolated from patients with community-associated SSTIs, it is far outnumbered by the isolation rate of MSSA that remains fairly sensitive

to the first line drugs against *S. aureus* like Cefazolin, Clindamycin, and Amikacin. Abscesses are the most common clinical presentation caused by CA-MRSA in this study and we recommend that physicians should consider obtaining cultures and antimicrobial susceptibility tests in all such patients.

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