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Indian Journal of Microbiology Research

Journal homepage: www.ipinnovative.com

Original Research Article

Clinico-microbiological study of community acquired MRSA from skin and soft tissue infections and its antibiogram in a tertiary care hospital in Karnataka

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ARTICLE INFO

Article history:
Received 04-05-2020
Accepted 06-05-2020
Available online 20-07-2020

Keywords:
Community associated methicillin resistant staphylococcus aureus (CA-MRSA)
Cefoxitin disc diffusion method
Double disk diffusion method (D test)
Inducible Clindamycin resistance

ABSTRACT

Background & Objectives: Methicillin resistant staphylococcus aureus (MRSA) has always been a worldwide problem, although its prevalence varies considerably among countries. The epidemiology of MRSA has changed over the years and infections are no longer confined to the hospital setting, but appear in healthy community dwelling individuals with no risk factors. From skin and soft tissue infection, common organism isolated is Community associated MRSA (CA-MRSA). Hence the study was done to know the prevalence of MRSA among community associated skin and soft tissue infections in Basaveshwar teaching and general hospital, attached to M.R. Medical College, Gulbarga.
Materials and Methods: Standard techniques were used to isolate Staphylococcus aureus from clinical specimens. Cefoxitin disc diffusion was used to find MRSA. Antibiogram of MRSA was detected by Kirby Bauer disc diffusion method. Inducible Clindamycin resistance was done by Double Disc Diffusion method (D test).
Results: From over 200 cases of CA-MRSA, total of (75.5%) staphylococcus aureus was detected. Out of them, CA-MRSA was 27 (17.9%). These showed high sensitivity to Vancomycin (100%), Linezolid (96.2%),Cindamycin (92.59%), moderately susceptible to TMP-SMX(85.1%), Rifampicin (88.1%), Tetracycline(81.4%),Gentamicin (70.3%), ciprofloxacin(62.9%) and a low susceptibility to Erythromycin (48.14%). 14.8% of CA-MRSA strains were D test positive (inducible MLS_B positive) and 29.63% were D test negative (MS phenotype). 7.4% of CA-MRSA were positive for constitutive MLS_B resistance.
Conclusion: There is a need for judicious selection of antimicrobial agents, as their indiscriminate use can exert pressure in selecting MRSA and other multi-drug resistant organisms. Further spread of community acquired infections can be done by effective infection control programs.

1. Introduction

Staphylococcus aureus is the foremost important and most studied human pathogen of the staphylococcus genus. It's nature can cause various diseases from mild infection realed to skin and soft tissue infections to severe infections such as necrotizing pneumonitis, osteomyelitis, and meningitis. It harbors several virulence factors including surface associated adhesions, secreted exoproteins and toxins. It also exhibits an important characteristic to acquire resistance to antimicrobial drugs.[1]

In the year 1961, Methicillin resistant staphylococcus aureus (MRSA) was first detected and has known for its important nosocomial pathogenicity worldwide. With increased rate of multidrug resistance, the infection has become predominant among people with increased frequency or recent contact with health care facilities. Hence, it is also named as health care associated methicillin resistant staphylococcus aureus (HA-MRSA).[1–3] Methicillin resistance is acquired by mecA gene, which regulates for an additional penicillin binding protein (PBP) namely 2a with reduced affinity to β-lactam agents. This gene is located in a small genetic element called staphylococcal cassette chromosome SCC mec.[1]

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https://doi.org/10.18231/j.ijmr.2020.027
2394-546X/

Other form of MRSA called community associated methicillin resistant staphylococcus aureus (CA-MRSA) is a prime etiologic agent for infection among healthy children and young adults with no exposure from hopsitals. CA-MRSA cause skin and soft tissue infections but can also cause serious systemic infections such as pneumonia and fasciitis.[2] These infections usually occur in immunocompetant persons without MRSA risk factors, have a type IV SCC mec genetic element that codes for mecA gene, usually contains PVL gene encoding Panton Valentine Leukocidin and susceptible to non β-Lactam antibiotics.[2]

CA-MRSA emergence has caused great impact world-wide since the presence of this pathogen in patients without risk factors represents a high risk to public health. Moreover CA-MRSA has caused outbreaks in the hospital setting suggesting that it may be replace HA-MRSA in the years to come with potentially catastrophic consequences. CA-MRSA strains are usually considered to be more virulent than HA-MRSA, leading to increased mortality and morbidity if they reach hospital population.[3–6]

Hence the present study was done to know prevalence of CA-MRSA from skin and soft tissue infections in patients attending skin and surgery out patients' department of HKE'S Basaveshwar Teaching and General Hospital, Gulbarga. This study is directed to create awareness among the clinicians regarding the prevalence and antibiotic sensitivity pattern of CA-MRSA and also to aid the clinicians in using appropriate antibiotics to treat such infections in future.

2. Materials and Methods

The study was done in Microbiology department attached to Mahadevappa Rampure Medical college, Gulbarga. 200 samples were collected from patients with skin and soft tissue infections attending the Dermatology and out patients from Surgical speciality.

Patients from skin department with pyoderma, patients from surgical speciality with soft tissue infections and samples patients within 48 hours of hospitalization were included in the study. Patients with high risk points for MRSA like recent hospitalization, surgery, dialysis, long term cure, indwelling catheter, percutaneous medical device, history of MRSA infection in the past and clinical specimen from patients after 48 hours of hospitalization were excluded from the study.

2.1. Method of collection of samples

All the clinical specimen were collected under aseptic precautions. Specimens such as pus and exudates were collected from patients of all ages and both sexes with skin and soft tissue infections. For collecting exudate samples, sterile swabs soaked in sterile saline were used whereas for collecting pus samples, sterile swab or disposable syringe and needle for aspiration were used.

2.2. Laboratory procedures

All the samples were processed within two hours of collection. Smear were made from each sample and examined with gram staining for pus cells and presence of gram positive cocci in clusters.

2.2.1. Culture

Specimens were streaked on nutrient agar, blood agar, milk agar and mannitol salt agar and incubated at 37°C for 18 to 24 hrs and observed for growth on next day.

1. On nutrient agar- presence of large (2-4mm), circular, smooth, opaque, golden yellow colonies were noted.
2. On blood agar- presence of beta hemolytic colonies noted.
3. On milk agar- presence of golden yellow pigment noted.
4. On mannitol salt agar- presence of yellow colored colonies noted.

All the suspected colonies were subjected to gram stain to look for the presence of gram positive cocci, around 1μm in size and arranged in clusters.

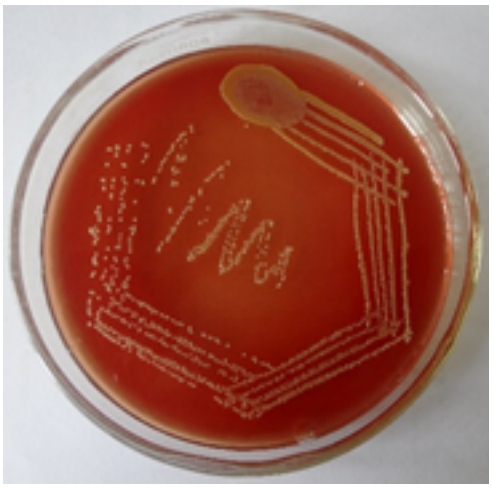

Fig. 1: β-Hemolytic colonies on blood agar

2.2.2. Biochemical reactions

2.2.2.1. Catalase test. A suspected colony was taken with a clean sterile glass rod and transferred onto a slide containing 3% hydrogen peroxide and looked for effervescence.

2.2.2.2. Test for differentiation of staphylococci from micrococci. Modified hugh and Leifsons o/f test was performed where the culture under test was inoculated into two tubes of O/F dextrose medium by stabbing down their

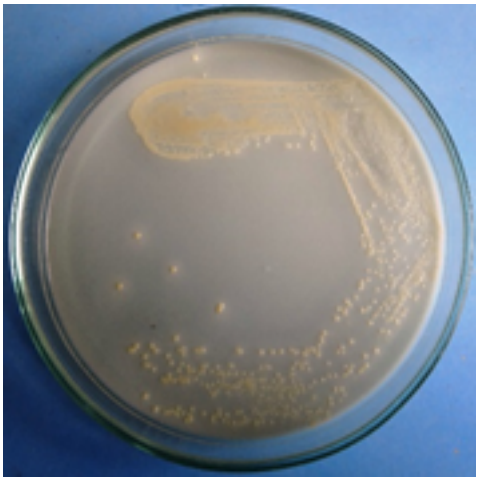

Fig. 2: Golden yellow colonies on milk agar

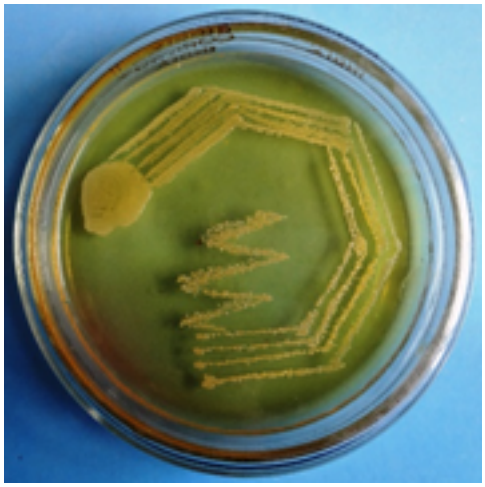

Fig. 3: Yellow colonies on mannitol salt agar

whole length with a long wire loop. One tube was covered with a layer of sterile liquid paraffin of at least 2.5 cm depth, after inoculating the culture into it and both tubes were incubated at 37ºc. Staphylococci produce acid by fermentation throughout the depth of medium both in the anaerobic tube sealed with paraffin oil and in the aerobic tube. Micrococcus fails to produce acid in both the tubes or produce acid in the aerobic tube only.

2.2.3. Coagulase test

2.2.3.1. Slide coagulase test. This test is to detect clumping factor, which is present in staphylococcus aureus. Human plasma was used as the reagent, which was stored at 4ºc and brought to room temperature before use. Clumping factor was detected by making a heavy suspension of cells in saline and stirring the mixture to a homogenous composition and then adding a drop of plasma. The mixture was examined for the presence of clusters seen to naked eye within 10 seconds and tested with positive and negative controls.

2.2.3.2. Tube coagulase test. Diluted plasma (1 in 6) was taken, 1ml was added to small tubes. The test colony was mixed in the diluted plasma along with both positive and negative controls. The tubes were kept at 37ºc and were seen for clot formation after 4 hours. If it was negative, they were at room temperature for next 12-16 hours and were re-examined for the presence of clot.

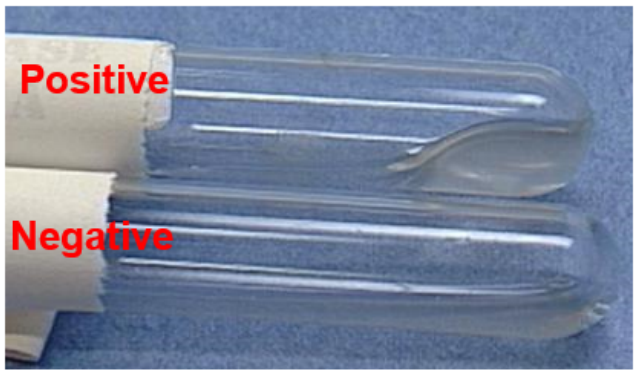

Fig. 4: Tube coagulase test

2.2.3.3. Deoxyribonuclease test (DNAse test). DNA agar plates were dried of all the moisture and the plates were divided into sections and strains under test were touched with an inoculating loop and spot inoculation was done. Positive and negative controls were used. The plates were incubated at 37ºc for 24 hours and then they were flooded with a few ml of 3.6% HCl to precipitate unhydrolysed DNA. After few minutes plates were examined. Spot cultures that were surrounded by a clear cloudy zone comparable in width to the zone around positive controls were considered positive.

2.2.4. Detection of methicillin resistant staphylococcus aureus (MRSA) by Cefoxitin disc diffusion test

All the isolates of staphylococcus aureus identified phenotypically by different biochemical test, were subjected to susceptibility testing using cefoxitin (30μg) disc diffusion method for detection of methicillin resistance. A sterile swab was dipped in staphylococcus suspension (Mc farland 0.5) and was plated on Muller Hilton agar using cefoxitin 30μg and plates were kept in incubator for 18-24 hours at 37^{o}C. The zone of inhibition was noted down in mm. To tell the strain as sensitive zone diameter should be $\geq$22mm and to tell the strain as resistant the zone diameter should be $\leq$21mm to cefoxitin.

2.2.5. Determination of antibiogram for methicillin resistant staphylococcus aureus

Antibiogram was done on Mueller Hinton agar by Kirby Bauer disc diffusion method as per CLSI guidelines. Below

Fig. 5: Clear Halo- DNAse positive

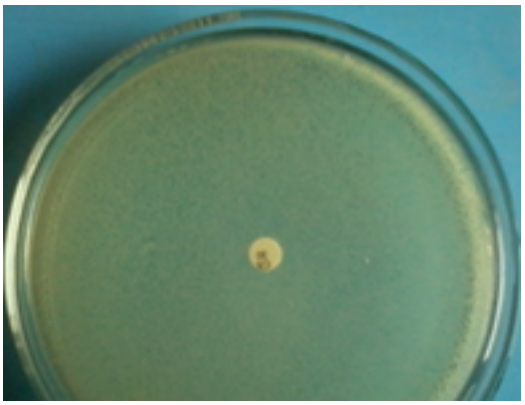

Fig. 6: Methicillin resistant S.aureus

mentioned antibiotic discs were used for our study as shown in Table 1.

Table 1: Antibiotic discs and their concentrations used for the study

Antibiotic disc	Concentration
Erythromycin	15μg
Clindamycin	2μg
Ciprofloxacin	5μg
Gentamicin	10μg
Tetracycline	3μg
Co-trimoxazole	25μg
Rifampicin	5μg
Trimethoprim-Sulfamethoxazole	1.25/23.75μg
Vancomycin	30μg
Linezolid	30μg

2.2.6. Determination of inducible Clindamycin resistance (D test)

Antibiogram of MRSA isolates showing Clindamycin sensitive and Erythromycin resistant, The D test was done. To identify inducible Clindamycin resistance, 15μg Erythromycin and 2μg Clindamycin disc were placed 15-20 mm apart for Staphylococcus aureus strains. Staphylococcus aureus 25923 was used as control for these tests and the plates were incubated at 37ºc for 18-24 hours. In the double discs diffusion test, if there is inducible Clindamycin resistance, the Erythromycin will diffuse throughout the agar, and resistance to Clindamycin will be induced resulting in flattening or blunting of Clindamycin zone of inhibition adjacent to the Erythromycin disc giving the shape of D to the zone.

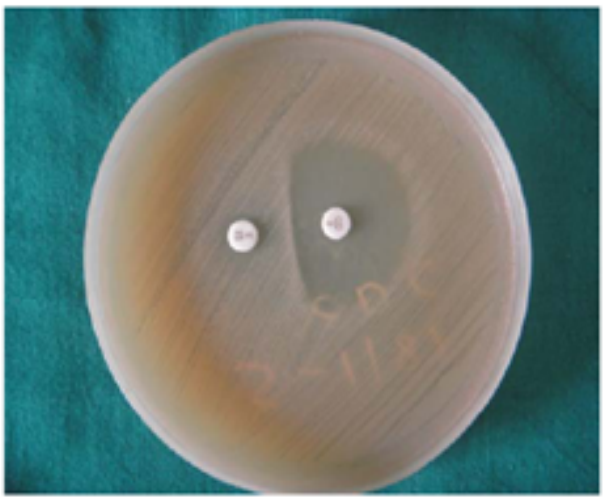

Fig. 7: D test- positive

3. Results

In the present study, 200 samples were examined from patients with community acquired skin and soft tissue infections. The bacteriological profile of the organisms is shown in Table 2. Most common isolate was Staphylococcus aureus accounting for 75.5% (151) of isolates. This was followed by streptococcus pyogenes 14% (28), Escherichia coli 5.5% (11), and klebsiella 5% (10).

Table 2: Bacteriological profile of community associated skin and soft tissue infections

Isolates	Cases (%) (N=200)
Staphylococcus aureus	151 (75.5%)
Streptococcus pyogenes	28 (14%)
Escherichia coli	11 (5.5%)
Klebsiella pneumoniae	10 (5.0%)

Among the various isolates, prevalence of CA-MRSA and methicillin sensitive S. aureus (MSSA) is shown in Table 3. 17.9% (27) of community associated staphylococcus aureus were found to be Methicillin resistant by the cefoxitin disc diffusion method. 82.1% (124) were found to be Methicillin sensitive staphylococcus aureus (MSSA).

Table 3: Prevalence of community associated methicillin resistant staphylococcus aureus (CA-MRSA)

Isolate	Cefoxitin Disc Diffusion Method		Total
	MRSA (%)	MSSA (%)	
Staphylococcus aureus	27 (17.9%)	124 (82.1%)	151 (100%)

The age and sex distribution of CA-MRSA infections are shown in Table 4 and Table 5. They were more commonly seen in the age group of 21-30 years, followed by 11-20 years, 0-10 years, 31-40 years, 41-50 years, 51-60 years, and 61-70 years respectively. Males were more commonly affected and accounted for 62.97% (17 cases) of isolates against 37.03% (10 cases) in females.

Table 4: Age distribution of Ca-Mrsa infection

Age in years	Number of cases (27)	Percentage
0-10	4	14.81
11-20	6	22.22
21-30	10	37.03
31-40	3	11.11
41-50	2	7.40
51-60	1	3.70
61-70	1	3.70

Table 5: Sex wise distribution of CA-MRSA infection

Sex	Number of cases (27)	Percentage
Males	17	62.97
Females	10	37.03

CA-MRSA infection presented clinically with abscess (51.86%) in most of the cases, followed by cellulitis (22.22%), furunculosis (11.11%), wound infection (7.4%) and carbuncle (7.4%) respectively, as shown in Table 6.

Table 6: Clinical presentation of CA-MRSA skin and soft tissue infections

Clinical presentation	Number of cases (27)	Percentage
Abscess	14	51.85
Cellulitis	6	22.22
Furunculosis	3	11.11
Wound infection	2	7.4
Carbuncle	2	7.4

Most of the CA-MRSA isolates were resistant to Erythromycin. 51.86% of the isolates were resistant to Erythromycin. 92.59% of the isolates were susceptible to Clindamycin and 7.41% of the isolates were resistance to Clindamycin as shown in Table 7.

The macrolide resistance of CA-MRSA as determined by disc diffusion method is shown in Table 8. Of the total isolates, 13(48.14%) isolates were found to be susceptible to both Clindamycin and Erythromycin – S phenotype, 2(7.40%) isolates were found to be resistant to both Clindamycin and Erythromycin – R phenotype (constitutive resistance) and 12(44.14%) isolates were found to be resistant to Erythromycin and Clindamycin sensitive.

The resistance to inducible clindamycin as determined by D-test is shown in Table 9. 12 isolates were found to be resistant to Erythromycin and sensitive to Clindamycin by disc diffusion test. D test was done on these 12 isolates to look for inducible Clindamycin resistance. 4(14.8%) isolates were found to be positive by D test- inducible Clindamycin resistance (iMLS$_B$). 8 (29.63%) of the isolates were found to be negative by D test- MS phenotype.

Antibiotic sensitivity profile of CA-MRSA is shown in Table 10. All the isolates were susceptible to Vancomycin (100%), followed by Linezolid (96.2%), Rifampicin (88.8%), TMP-SMX (85.1%), Tetracycline (81.4%), Gentamicin (70.3%) and Ciprofloxacin (62.9%) in that order.

4. Discussion

The present study was performed to assess prevalence of CA-MRSA in patients with skin and soft tissue infections. This was designed keeping in mind its usefulness in creating awareness among the clinicians regarding CA-MRSA and in treatment planning for infected patients.

It was observed from the samples that most common organism was Staphylococcus aureus and accounted for 75.5% (151) of the infections. In a similar community-based Indian study, out of the 250 cases of pyoderma studied, S. aureus was isolated in 80.8% of cases.[7] Moran et al. reported that Staphylococcus aureus was isolated in 76% of patients with similar skin conditions compared to our study.[8] One study from Mumbai reported Staphylococcus aureus to be the predominant pathogen (81.4%) among community associated skin infections.[9]

Cefoxitin Disc diffusion method is used to find Methicillin resistance. CA-MRSA accounted for 17.9% (27 cases) of the staphylococcus aureus isolates. Study done by Nagaraju et al., showed 11.8% CA-MRSA which is lower than our finding.[7] Study conducted by saxena et al., showed 18.1% which is similar to our finding.[10]

The prevalence of CA-MRSA varies worldwide. Investigations of two MRSA outbreaks in Native American communities found that 55% and 80% of staphylococcal infections were caused by MRSA.[8] In a study conducted

Table 7: Susceptibility pattern of the clinical CA-MRSA isolates to Erythromycin and Clindamycin by disc diffusion method

Antibiotic	Sensitive	Percentage	Resistant	Percentage
Erythromycin	13	48.14	14	51.86
Clindamycin	25	92.59	2	7.41

Table 8: Macrolide resistance of the isolates based on disc diffusion method

Organism	Total No of Ca-Mrsa Isolates	Both Erythromycin and Clindamycin sensitive	Erythromycin Resistant and Clindamycin Sensitive	Both Erythromycin And Clindamycin Resistant
CA-MRSA	27	13 (48.14%)	12 (44.44%)	2 (7.40%)

Table 9: Inducible Clindamycin resistance among CA-MRSA isolates based on D test

Isolate	No of strains	Inducible Clindamycin resistance (D test)	
		Positive	Negative
CA-MRSA (Erythromycin resistant and Clindamycin sensitive)	12	4(14.8%)	8(29.63%)

Table 10: Antibiotic susceptibility pattern of CA-MRSA

Antibiotic	Sensitive	Percentage	Resistant	Percentage
Ciprofloxacin	17	62.9	10	37.1
TMP-SMX	23	85.1	4	14.9
Tetracycline	22	81.4	5	18.6
Rifampicin	24	88.8	3	11.2
Gentamicin	19	70.3	8	29.7
Linezolid	26	96.2	1	3.8
Vancomycin	27	100	0	0

in Japan reported 21% prevalence of MRSA of 229 Stapylococcus aureus.[11]

The finding in our study is consistent with the significant increase of CA-MRSA all over the world and has proved the association of CA-MRSA has spread in our community. Most of the CA-MRSA strains were isolated from patients of age group 21-30 years followed by 11-20 years, 0-10 years and 31-40 years respectively. It was most commonly seen in young adults and children. Propensity of young people to share belongings, activities and close physical contact put them at high risk. Majority of the infection were seen in males (62.97%) compared to females (37.03%). This is similar to the findings of other studies.[7,12]

Majority of patients with CA- MRSA infections presented with abscesses (51.85%) and cellulitis (22.22%) followed by furunculosis (11.11%), wound infection (7.4%) and carbuncles (7.4%) respectively. The findings are similar to studies done by Paul et al. and Crawfold et al.[13,14] In our study, CA-MRSA strains showed a susceptibility of 48.14% to Erythromycin which was similar to results obtained by Fridkin et al. and Timothy et al. (44% sensitivity).[15,16] Huang et al. and Frazee et al. had recorded a much lower susceptibility of 7% and 3.6% respectively which is in contrast to our study.[17,18]

A susceptibility of 92.59% to Clindamycin was seen in our study. A similar result of 96% and 94% sensitivity was observed by Huang et al. and Frazee et al. respectively.[17,18] A study by Mandelia C et al. reported a Clindamycin sensitivity of 93.3%.[19] Majorities CA-MRSA infections are sensitive to Clindamycin, hence Clindamycin sensitivity is used as a surrogate marker for diagnosing CA-MRSA infections by some authors.

In the present study, a rate of 14.8% was observed for inducible Clindamycin resistance (iMLS$_B$). Out of the total isolates, 29.63% were negative by D test. Isolates that were resistant to both Erythromycin and Clindamycin (constitutive MLS$_B$ resistance) constituted 7.4%. In studies conducted by Sattler et al. and Frank et al., the rate of inducible Clindamycin resistance was reported to vary widely from 8 to 94%.[20,21] It was observed to be 33% among CA-MRSA isolates in a study by Mukesh Patel et al. which is higher than that observed in our study.[22] Another study done in Mangalore reported 15.65% and 7.23% of CA-MRSA to be iMLS$_B$ and constitutive MLS$_B$ resistant respectively, which is similar to our study.[2] A retrospective study on CA-MRSA by Hsing Huang et al. reported inducible Clindamycin resistance of around 10% which is slightly lower than our finding.[17]

An MRSA strain that is Erythromycin resistant and Clindamycin sensitive should be followed with a D test which indicates the ability of MRSA strains to become resistant to Clindamycin during antibiotic therapy. Clindamycin is active against CA-MRSA strains as well as against group A streptococci, and is therefore an appealing

therapeutic choice. But the number of CA-MRSA strains harboring this inducible type of resistance is increasing, hence Clindamycin cannot be prescribed for treating MRSA infections without conducting an appropriate D test for iMLS$_B$.

TMP-SMX is usually preferred for treatment of CA-MRSA skin and soft tissue infections. In our study, 85.1 % of CA-MRSA strains showed sensitivity to TMP-SMX. Studies conducted by Huang et al. and Rice LB have observed very high rates of sensitivity (100%) to TMP-SMX by CA-MRSA strains.[17,23] Frazee et al. reported a sensitivity of 86% whereas Timothy et al. reported a sensitivity of 95% to TMP-SMX.[18] However, it may be ineffective against Cellulitis or other skin and soft tissue infections caused by Group A Streptococci, allergy to sulfa drugs, etc.

Tetracycline sensitivity in our study was 81.4% which is similar to studies done by Miller, Moran and Frazee et al.[8,18,24] They are also unlikely to be useful when there is a high suspicion for Group A Streptococcus infections. In CA-MRSA strains, resistance to Tetracycline is mostly associated with tetK, which encodes a Tetracycline specific efflux pump. This pump does not efflux doxycycline and minocycline. Thus, the long acting Tetracyclines may be active even when resistance to Tetracycline is detected. In our institution, we routinely test only for Tetracycline sensitivity and do not use doxycycline and minocycline disc for antibiotic testing. Hence further study is needed to know about the use of longer acting doxycycline /minocycline in case of Tetracycline resistance.

We found a low level of sensitivity (62.9%) to Ciprofloxacin, which is similar to studies done by Frazee et al and Timothy et al. Louis B Rice et al reported a low sensitivity of 20% and Mandelia C et al. reported 18.3% sensitivity which is low compared to our study. This may be attributed to the inadvertent use of Ciprofloxacin for various infections.

The present study showed a sensitivity of 88.8% to Rifampicin. Similar results were reported by Timothy and Louis et al. Rifampicin should not be used as a sole agent in therapy of CA-MRSA infections because of high rate of emergence of resistance. As Rifampicin achieves high concentration in mucosal surfaces, it may promote eradication of MRSA carriage theoretically. In India, use of Rifampicin as an Anti- MRSA drug should be discouraged owing to the high prevalence of tuberculosis.

Gentamicin sensitivity in our study was 70.3 %. Higher degree of susceptibility was shown to Vancomycin (100%) and Linezolid (96.2%) in our study. This is similar to studies done by Hsing et al, timothy et al and Louis et al. Vancomycin should be used as a reserve drug and used mainly to treat invasive / complicated skin and soft tissue infections not responding to other drugs. Vancomycin resistant strains are also emerging.

Oral therapy with Linezolid, a bacteriostatic oxazolidinone is also effective. The use of Linezolid is limited by the high cost, limited bioavailability of the suspension, the occurrence of thrombocytopenia with prolonged use and the availability of less expensive, effective oral antimicrobial drugs. It is reserved for treatment of serious MRSA infections only.

CA-MRSA isolates have typically been susceptible to most non β-Lactam antimicrobial drugs. This enables the clinicians to have a number of options when selecting empirical treatment of putative CA-MRSA infections. Judicious use of antimicrobials, particularly in the outpatient setting could help control the emergence of CA-MRSA strains and limit the acquisition of additional Antimicrobial resistance genes in existing strains.

5. Limitation

The main limitation of our study is that it is a hospital based study, hence many patients in the community with CA-MRSA infections who do not present to the hospital may have led us to underestimate the prevalence of infection in the community. We did not look for the MRSA colonization in these CA-MRSA infected individuals. The sample size is small, due to time constraint; hence more studies are required to determine the risk factors and establish preventive measures within the community.

6. Conclusion

There is a need for judicious selection of antimicrobial agents, as their indiscriminate use can exert pressure in selecting MRSA and other multi-drug resistant organisms. Effective infection control programs for the community should be considered to prevent the spread of community acquired infections.

7. Source of Funding

None.

8. Conflict of Interest

None.

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Cite this article: Thilagavathy , Chavan SKD. Clinico-microbiological study of community acquired MRSA from skin and soft tissue infections and its antibiogram in a tertiary care hospital in Karnataka. *Indian J Microbiol Res* 2020;7(2):146-153.