
Original Article

Methicillin Resistant *Staphylococcus aureus* in Children

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ABSTRACT

Background: The changing epidemiology of Methicillin Resistant *Staphylococcus aureus* (MRSA) became evident in the 1990s when community-acquired MRSA cases were first reported. Increasing prevalence of MRSA will inevitably increase the use of vancomycin, adding further to the problem of antimicrobial resistance.

The previous retrospective study during 1996-1998 in Rasool Akram Hospital determined the increasing prevalence of MRSA. The goal of this prospective descriptive study was to determine the antibiotic resistance pattern of *Staphylococci* spp responsible for upper respiratory infections in children.

Materials and Methods: From Dec 2001 to Dec 2003, we surveyed 73 *Staphylococci* spp (78.1%, *S. aureus*, 21.9% coagulase negative) obtained from children (1 month- 14 yrs) with upper respiratory infections (otitis media; mastoiditis; sinusitis; tracheitis,...). All isolates (blood; CSF or other sterile body fluids) after culturing and antibiogram were first evaluated by disc diffusion and then by Etesting for MIC detection.

Results: The results showed an increasing resistance to penicillin (100% vs 70%); and gentamicin (56.3% vs 30%); and a decreased resistance to erythromycin (47% vs 66%); oxacillin (11.6% vs 40%); and chloramphenicol (15.4% vs 22%). Only 6.8% of *S.aureus* and 25% of coagulase negative staph are MRSA.

MRSA prevalence in this study is 6.4% similar to the previous study (5.4%) and there has been no significant increase during 4 years. By using penicillinase inhibitor or other non beta lactam antibiotics more than 80% antibiotic coverage will be achieved. In a minority of cases (6.8%) vancomycin was needed.

Conclusion: We conclude that the prevalence of MRSA is rare in the present study. Therefore, vancomycin is not efficient for the empiric therapy of all *Staphylococcal* suspected infections. Penicillin is not appropriate for the treatment of children with suspected *Staphylococcal* infections. PRP plus one of the gentamicin; rifampin; clindamycin; chloramphenicol or Trimethoprim/ Sulfamethoxazole are recommended in severe cases. When *staphylococci* may be involved in more extensive infections, the empirical use of clindamycin provides appropriate coverage including the majority of community-acquired MRSA strains.

Limiting broad spectrum antibiotic use will minimize the antibiotic pressure that favors selection of resistant strains. In severe, invasive *staphylococcal* infections, such as severe pneumonia or toxic shock syndrome, inclusion of vancomycin in an empiric antibiotic regimen may be prudent initially, particularly among children with predisposing risk factors for MRSA carriage.

Keywords: MRSA (Methicillin Resistant *Staphylococcus aureus*), Antibiotic resistant, *Staphylococcal* infection, Upper respiratory infections

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INTRODUCTION

After the introduction of antibiotics in the mid-20th century, clinicians soon witnessed clinical failures secondary to bacterial resistance (1-10). Despite the scientists' efforts to synthesize more potent antibiotics during the last five decades, bacterial resistance continues to evolve in large part due to the overuse and misuse of antibiotics. The treatment of several pathogens, including methicillin-resistant staphylococcus aureus, penicillin-resistant streptococcus pneumoniae and vancomycin-resistant enterococci, is problematic (3,4).

Staphylococcus aureus is a ubiquitous environmental organism, with a predilection for skin, particularly for face, nose and hands and is routinely found in one-third of adults as normal flora. *S.aureus* organisms are easily recognized in the laboratory by their tendency to produce classic golden pigmented colonies with beta- hemolysis on blood agar and the appearance of clustered, grape-like Gram-positive cocci on Gram staining (3,4,8). Staphylococcal carriage rates as high as 90% have been found in health care workers, in those with dermatologic diseases, dialysis patients, intravenous drug users and diabetics (5,11). Hospital spread isolates in surgical units, intensive care units, nurseries and burn units are well-documented (9, 8, 12, 13). Formation of abscess is the trademark of *S.aureus* clinical infection and involvements of skin, skin structures and lymph nodes are most commonly encountered. Invasive disease involving lungs, liver, bones, joints, kidney, endocardium and foreign body device infections are potentially life- threatening (5).

Global pandemic of staphylococcal disease was well- reported through the 1950s and 1960s when the first reports of methicillin-resistant *S-aureus* (MRSA) emerged. By the late 1970s, MRSA was clearly established as an important nosocomial pathogen (1). Within the last decade virtually all hospitals have identified MRSA within their facilities and have recognized nosocomial spread and

subsequent infection in high risk hospitalized patients (13- 24).

Unfortunately the increasing prevalence of MRSA will inevitably increase the use of vancomycin, adding further to the problem of antimicrobial resistance (25-30). Thus it is important to discontinue vancomycin if no MRSA is identified and the suitable antibiotic therapy should be substituted based on susceptibilities (26, 27). Limiting broad spectrum antibiotic use will minimize the antibiotic pressure that favors selection of resistant strains (28-33).

The increased prevalence of MRSA was documented in the previous retrospective chart review in 214 Staphylococci spp isolates during 1996-1998 in Hazrat Rasool Akram Hospital (unpublished). There was no change in resistance to penicillin (70%); to PRP and cephalotin resistance was 40-60%, to gentamycin was 20-30%; but resistance to erythromycin was increasing 25% in 1996; 36% in 1997 and 66% in 1998. Resistance to chloramphenicol was 0% in 1996, 15%- 22% in 1997 and to ciprofloxacin was 0% in 1996.

Only 12 cases (5.6%) were resistant to all drugs except vancomycin by disc diffusion methods. Therefore, to determine whether MRSA infections in children are a continuing problem, we prospectively surveyed the clinical microbiology laboratory data in Hazrat Rasool Hospital from Dec 2001, to Dec 2003, for all *S.aureus* isolates obtained from children with upper respiratory infections (otitis media, mastoiditis, sinusitis, tracheitis, ...) at the pediatric ward of the hospital. We collected the clinical data regarding patients from whom isolates were obtained and now report an updated profile of MRSA disease.

MATERIALS AND METHODS

A list of all hospitalized patients younger than 14 years of age with upper respiratory infection caused by *S.aureus* isolate between Dec 2001 and Dec 2003 was compiled from their records in Hazrat Rasool Akram Hospital in Tehran. Isolates collected from

these patients were transported to Clinical Microbiology Laboratory of Hazrat Rasool Hospital for further microbiologic evaluation. Susceptibility data from the laboratory were used to define the subset of MRSA. For each *S.aureus*, isolate identified, patient's demographic (age, sex, date of admission) and relevant clinical data (site of culture specimen, antimicrobial therapy before the hospitalization, underlying medical conditions and sample site) were collected through reviewing the medical records.

A disease-associated isolate was defined as the one responsible for the clinical syndrome as determined from consideration of the site from which *S.aureus* was isolated, the physical examination findings and other relevant clinical data. Isolates not associated with disease were said to be colonizing. All SA isolates obtained during the study period were collected from the clinical microbiology laboratory and subcultured onto trypticase soy agar. After a 24-h incubation the isolate was stored at -70°C in skim milk as described (20-22).

The disc diffusion method was used for initial susceptibility testing in the clinical microbiology laboratory. All isolates were further evaluated by Etesting and MIC detection by methods described by the AB BIODISC. The antimicrobial agents tested are all discussed in table 1.

Table 1. NCCLS interpretive MIC criteria (mic/ml)

Antibiotic	S	I	R
B.penicillin	<0.12	-	>0.25
Ciprofloxacin	<1	2	>4
Amoxi/Clav.	<4	-	>8
Chloramphenicol	<8	16	>32
Clindamycin	<0.5	1-2	>4
Erythromycin	<0.5	1-2	>2
Gentamycin	<4	8	>16
Methicillin	<8	-	>16
Oxaci (S.Aureus)	<2	-	>4
Oxaci (S.Epiderm)	<0.25	-	>0.5
Rifampin	<1	2	>4
Tetracycline	<4	8	>16
Trim/Sulfa	<2	-	>4
Vancomycin	<4	8-12	>32

RESULTS

Seventy three staphylococci spp. were identified during the period of surveillance: 16(21.9%) were coagulase negative and 57(78.1%) were coagulase positive.

Age, sex and site of isolation in patients are present in table 2-4.

Table 2. Age distribution of patients

Age	Percentage
<1 y old	43.9
2-10 y	42.5
>10	13.6

Table 3. Sex distribution of patients

Sex	Percentage
Male	50.7
Female	49.3

Table 4. Site of staph isolation in patients

Site of isolation	Percentage
Blood	44.4
CSF	1.2
Other (sinus, Middle, Ear ...)	54.4

Antibiotic susceptibility of *S.aureus* and coagulase negative Staph are shown in table 5-7.

Table 5. Antimicrobial susceptibility of 58 *S. aureus* isolates.

Antibiotic/ susceptibility (%)	Amoxicillin	Trimethoprim/Sulfamethoxazol	Erythromycin	Oxacillin	Chloramphenicol	Gentamycin
Sensitive	54.3	60.3	47.1	88.4	84.6	56.3
Intermediate	5.7	-	1.5	-	7	-
Resistant	40.0	39.7	51.5	11.6	7.7	43.7

Table 6. Number of antibiotic resistant isolates in 58 *S.aureus*.

Antibiotic/Number of resistant isolates	Tetracycline	Penicillin	Erythromycin	Trimetho/Sulfamethoxazol	Gentamycin	Amoxil/Clav	Oxacillin	Choloram/Rif.	Vanco/Cipro
23 isolates	+	+	-	-	-	-	-	-	-
6 isolates	+	+	+	-	-	-	-	-	-
2 isolates	+	+	-	+	-	-	-	-	-
3 isolates	+	+	-	+	+	-	-	-	-
1 isolates	+	+	+	+	-	-	-	-	-
1 isolates	+	+	-	-	+	-	-	-	-
3 isolates	+	+	+	-	+	-	-	-	-
1 isolates	+	+	-	-	-	+	-	-	-
2 isolates	+	+	+	-	-	+	-	-	-
12 isolates	+	+	+	+	+	+	-	-	-
4 isolates	+	+	+	+	+	+	+	+	-

Table 7. Number of antibiotic resistant isolates in sixteen coagulase negative staph

Antibiotic/ Number of Resistant isolates	Tetracycline	Penicillin	Erythromycin	Trimetho/Sulfamethoxazol	Gentamycin	Amoxil/Clav	Oxacillin	Chloram/ Rifampin	Vanco-cipro
3 isolated	+	-	-	-	-	-	-	-	-
3 isolated	+	+	-	-	-	-	-	-	-
2 isolated	+	+	-	+	-	-	-	-	-
1 isolated	+	+	+	-	-	-	-	-	-
1 isolated	+	+	-	+	+	-	-	-	-
1 isolated	+	+	+	-	+	-	-	-	-
1 isolated	+	+	+	+	+	-	-	+ chlor	-
4 isolated	+	+	+	+	+	+	+	-	-

There are no significant difference between the sensitivity to oxacillin ($x^2=0.26$, $p=0.609$) and cloramphenicol ($x^2=0.25$; $p=0.616$); with vancomycin (gold standard); but a significant difference was detected between sensitivity to erythromycin ($x^2=7.86$; $p=0.005$); amoxicillin/clav ($x^2=5.54$; $p=0.018$); Thrimethoprim/sulfamethoxazole ($x^2=3.87$; $p=0.04$).

DISCUSSION

The changing epidemiology of MRSA became evident in the 1990s when community- acquired MRSA cases were first reported (10-17). Embil et al (2). reported that 63% of MRSA isolates were identified within 72 hours following admission in a review of five Canadian university hospitals from 1990 to 1992. Moreno et al (3). also reported a high rate of community MRSA cases (99 of 170; 58%) with an incidence of 0.2 per 1000 patients. There are no risk factors for the differentiation of patients with community MRSA from those with methicillin-susceptible *S.aureus* (MSSA), and pulse field gel electrophoresis confirmed that 68% had unique pulse field gel electrophresis patterns (10, 14, 15). Whereas Layton et al (6) noted that community MRSA acquisition was associated with recent hospitalization, previous antimicrobial therapy, nursing home residence and IV drug use; they also observed that 22% of patients had no discernible risk factors.

Although MRSA disease has been increasingly recognized in children without traditional risk factors, it is not clear that to what extent MRSA colonization has become pervasive in the community at large (16-18).

MRSA risk factors should be delineated in all cases in which MRSA is documented. Those factors include prior hospitalization, surgery or use of antimicrobial drugs within the last 6 months, day-care center attendance and/ or day- care or household contact with health care workers, or those with chronic underlying diseases (3-13).

No risk factors differentiated patients with community MRSA from those with methicillin-susctible *S.aureus* (MSSA). *Staphylococcus aureus* (MRSA) as a pathogen confined to the hospital environment in patients with well- described risk has recently been challenged with the recognition of community- acquired MRSA (CA-MRSA) in

children and adults who lack these predisposing risk factors (10,14,15). Investigators have noted that community-acquired MRSA infections are more likely to be susceptible to clindamycin, and the types of clinical infections encountered are similar to that of MSSA (23).

As in other educational centers, vancomycin is the first drug for empiric therapy of staphylococcal infections in all patients admitting to the pediatric ward of our referral center.

Most of staph-spp isolates in pediatric ward are sensitive to oxacillin (88.4%). Chloramphenicol (84.6%), and trimethoprim/ sulfamethoxazole (60.3%) but are less sensitive to amoxicillin/clav. (54.3%) gentamycin (56.3%) and erythromycin (47.1%). No significant difference was seen between the sensitivity to oxacillin and chloramphenicol with vancomycin as gold standard, but significant difference was seen in sensitivity to erythromycin; trimethoprim/ sulfamethoxazole; and amoxicillin/clav. Similar results were seen in at least one study (5).

As discussed above, 72.5% and 37% of *S.aureus* and coagulase negative staph isolates are only resistant to penicillin and tetracycline; 20.7% and 25% of them are sensitive to all drugs except penicillin, and tetracycline. Probably; resistance (>50%) of Staph.spp is due to the penicillinase producing by these organisms. MRSA is 6.8% and 25% for *S.aureus* and coagulase negative staph. Rate of MRSA is (6.4%) similar to the previous study (5.4%), and no significant increase is seen during 4 years. More than 80% antibiotic coverage will be achieved by using penicillinase inhibitor (amoxicillin/clav) or other non beta lactam antibiotics (cotrimoxazole; erythromycin; gentamycin; rifampin; clindamycin; Chloramphenicol and PRP (oxacillin) in staphylococcal infections; in minority of cases (6.8%) vancomycin was needed.

Between 2001-2002 at Texas Children's Hospital the outcome of therapy for MRSA with that of methicillin-susceptible (MSSA) invasive infections in children treated with clindamycin, vancomycin or beta-lactam antibiotics was compared. It was concluded that Clindamycin was effective in treatment of children with invasive infections caused by susceptible CA-MRSA isolates (33). Clindamycin is another choice in resistant suspected staphylococcal infections. The CA-MRSA isolates obtained from children irrespective of identified predisposing risk factors were more likely to be susceptible to clindamycin and erythromycin compared with the nosocomially acquired MRSA isolates. Resistance to trimethoprim/ sulfamethoxazole was infrequent in all MRSA isolate groups. Resistance to gentamicin occurred in only 1 of the 10 CA-MRSA isolates, a patient with a known risk factor; only a single nosocomially acquired isolate was resistant to rifampin (32-33).

We recommend never to use Penicillin in children with suspected Staphylococcal infections. Other drugs (single or in combination), such as PRP, erythromycin, thrimethoprim/ sulfamethoxazole; gentamycin are suitable for use in admission; depending on the severity of infection.

Therefore empiric treatment of mild to moderate infections with standard anti-staphylococcal therapy (PRP; Erythromycin; Cephalothin) is still adequate for the majority of our patients. Because MRSA is rare (6.8%) in our study, Vancomycin is not appropriate for empiric therapy of all Staphylococcal suspected infections except in high-risk patients.

Treatment failure will be resulted if an anti-staphylococcal beta-lactam antibiotic is used for therapy of MRSA infection. In cases with treatment failure, identification of a specific isolate is important, allowing appropriate antibiotic treatment adjustments to be made on the basis of antimicrobial susceptibility testing.

We prefer PRP plus one of the gentamycin, rifampin; clindamycin; chloramphenicol or trimethoprim/Sulfamethoxazole for cases with severe disease. Where staphylococci may be involved in more extensive infections, the empiric use of clindamycin provides appropriate coverage, including the majority of community-acquired MRSA strains. In severe, invasive staphylococcal infections, such as severe pneumonia or toxic shock syndrome, inclusion of vancomycin in an empiric antibiotic regimen may be prudent initially, particularly among children with predisposing risk factors for MRSA carriage.

Unfortunately, the recent recognition of MRSA strains with intermediate resistance to vancomycin associated with treatment failure (25-31) suggests that such a strategy may not be successful for long.

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