Jpn. J. Infect. Dis., 56, 54-56, 2003

Original Article

Methicillin-Resistant *Staphylococcus aureus* Prevalence in Community in the East Delhi Area

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(Received June 24, 2002. Accepted April 10, 2003)

SUMMARY: Methicillin-resistant *Staphylococcus aureus* (MRSA) continues to be an important nosocomial pathogen. Various hospital-based studies have described the incidence of MRSA and carriage of this organism in health care workers. Recently, even community acquired *S. aureus* strains have shown resistance to methicillin. This changing epidemiology prompted us to study the nasal carriage of MRSA amongst healthy individuals in a community. A total of 319 nasal swabs were taken from both anterior nares of healthy parents attending a well-baby clinic. Of these, 94 yielded growth of *S. aureus* (29.4%). Out of these 94 isolates, 17 (18.1%) were found resistant to oxacillin. These strains showed low level resistance only to clindamycin.

INTRODUCTION

Methicillin-resistant *Staphylococcus aureus* (MRSA) has proven to be one of the most widespread nosocomial pathogens of the late 20th century (1). Various hospital- based studies have described the incidence of MRSA causing such infections (2-5). In Guru Tegh Bahadur (GTB) Hospital, 33% *S. aureus* infections were found to be caused by MRSA, while the nasal carriage rate of this organism in health care workers was reported to be 39% in 1995 (6). Until a few years ago, only nosocomially acquired isolates were found to show such resistance, but recently even community-acquired strains have shown resistance to methicillin (7-12). Reports of increasing community- acquired MRSA from the United States, Canada, and Australia are evidence of the changing epidemiology of MRSA. Because of such changes it is important to assess the carriage rate of MRSA in the community amongst healthy individuals who have not been hospitalized nor have had antibiotic therapy in the recent past. It was with this background that this study was undertaken in the east Delhi area.

MATERIALS AND METHODS

This study was carried out at the dispensary attached to GTB Hospital in east Delhi. Nasal swabs from both anterior nares were obtained from healthy parents accompanying children at the well-baby clinic. Samples were also taken from healthy volunteers (included healthy adults of both sexes) residing in this area of east Delhi. Any person with a history of hospitalization, undergoing surgery or treatment of any kind and intake of antibiotics in the past 12 months was ruled out of the study.

The nasal swabs were first plated on mannitol salt agar (Difco, Detroit, Mich., USA) and were then placed in nutrient broth containing 7.5% sodium chloride. Subcultures from broth were made after overnight incubation on 5% sheep blood agar and mannitol salt agar. The plates were incubated for 24 h. Colony morphology suggestive of *S. aureus* was identified by Gram stain, catalase, slide, and tube coagulase tests and O-F tests by standard methods (13). Susceptibility testing on *S. aureus* was done by means of the agar screening method on Mueller Hinton agar (Difco) containing 6 mg/l of oxacillin and 4% sodium chloride. Plates were inoculated with a bacterial suspension matched with 0.5 McFarland standard using streak inoculation (14). The plates were incubated for exactly 24 h at 35°C. Drug-free plates were used as growth control. *S. aureus* ACTC 38591 was used in each plate as MRSA control (15). Growth of even a single colony was taken as indication of resistance. We used the guidelines of National Committee for Clinical Laboratory Standards for disc diffusion susceptibility in regard to trimethoprim-sulfamethoxazole (1.25 + 23.75 μg), amoxicillin-clavulanate (10 + 30 μg), erythromycin (15 μg), ciprofloxacin (5 μg), vancomycin (30 μg), clindamycin (2 μg) and gentamicin (10 μg). The plates were incubated at 35°C (15).

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RESULTS

A total of 319 nasal swabs were taken from 133 healthy male and 186 healthy females. Two hundred swabs were taken from parents of children attending the well-baby clinic, while 119 samples were from healthy volunteers. Out of 319 samples, 94 (29.4%) yielded growth of *S. aureus*. Eighty-two isolates grew on mannitol salt agar while 12 additional isolates were recovered from nutrient broth containing 7.5% sodium chloride. The cases were in the age group 19 - 26 years with a mean age of 22.5 years. Of these 94 isolates, 17 (18.1%) grew on oxacillin agar (Table 1).

Antimicrobial susceptibility studies of MRSA isolates by disc diffusion methods showed 88% of the isolates resistant to trimethoprim-sulfamethoxazole and amoxicillin-clavulanate followed by 76% showing resistance to gentamicin, 71% to ciprofloxacin, and 53% to erythromycin. Low resistance was seen to clindamycin (23%), while none of the isolates showed reduced susceptibility to vancomycin (Table 2).

DISCUSSION

Strains of MRSA have emerged as important pathogens

Table 1. Prevalence of MRSA and MSSA in the community

	Male (n = 133)	Female (n = 186)	Total (n = 319)
Number of *S. aureus* isolated (*S. aureus*: total swabs)	40 (1:3.3)	54 (1:3.4)	94
Number of MRSA isolated (MRSA: total swabs)	5 (1:26.6)	12 (1:15.5)	17
Number of MSSA isolated (MSSA: total swabs)	35 (1:3.8)	42 (1:4.4)	77

MRSA: Methicillin-resistant *Staphylococcus aureus*.
MSSA: Methicillin-sensitive *Staphylococcus aureus*.

Table 2. Resistance of 17 MRSA isolates

	Number of resistant isolates (%)
Trimethoprim-sulfamethoxazole	15 (88)
Amoxicillin-clavulanate	15 (88)
Clindamycin	4 (23)
Erythromycin	9 (53)
Ciprofloxacin	12 (71)
Gentamicin	13 (76)
Vancomycin	0

over the last 20 years affecting primarily hospitalized patients (16). A few reports have described the acquisition of MRSA among individuals who had little or no direct contact with hospitals or other health care facilities (8,17,18). In the hospital, inpatients and hospital personnel are the potential reservoirs for this organism. However, studies regarding the prevalence of this organism in communities beyond the hospital have been few (11,12).

The largest populations of *S. aureus* are found in the regions of the skin and mucous membranes surrounding openings in the body surface. These include the anterior nares, inguinal and perineal areas. Anterior nares constitute the most common site of colonization and are also responsible for the dissemination of this organism. This area is well ventilated and usually remains moist. However, colonization usually remains undetected as this area is seldom cultured for clinical purposes (2). In this study we demonstrate the nasal carriage rate of MRSA in a general population with no previous exposure to hospital or antibiotic usage. The nasal carriage of *S. aureus* was 29.4%. The colonization rate may range from 10% to more than 40% in a normal adult population (19). Our figure of 29.4% correlates well within this. Although nasal carriage of *S. aureus* has been suggested as the source of infections, previous studies have been in defined patient groups or health care workers (2,3,6). Not many studies have investigated the nasal carriage of MRSA in an otherwise healthy population. O'Brien et al. studied the colonization rate of MRSA in two remote communities in Australia and found colonization rates of 68% and 65% (12). Such high rates could have been due to the combination of hand, nasal, and skin swabs used to screen MRSA carriage.

There have been a number of reports of community MRSA from other parts of the world (12,16). However, it is not always clear whether these strains have arisen in the community or are hospital strains that have spread to the community. Molecular techniques may help in solving this problem. Our results indicate the existence of MRSA even in a healthy population with no recent exposure to hospital or health care workers. Larger community-based studies are needed to confirm that transmission is occurring more frequently in community settings.

REFERENCES

1. Nimmo, G. R., Schooneveldt, J., O'Kane, G., McCall, B. and Vickery, A. (2000): Community acquisition of gentamicin sensitive methicillin-resistant *Staphylococcus aureus* in South East Queensland, Australia. J. Clin. Microbiol., 38, 3926-3931.
2. Thompson, R. L., Cabezudo, I. and Wenzel, R. P. (1982): Epidemiology of nosocomial infection caused by methicillin-resistant *Staphylococcus aureus*. Ann. Intern. Med., 97, 309-317.
3. Layton, M. C., Heirholzer, W. J. and Patterson, J. E. (1995): The evolving epidemiology of methicillin-resistant *Staphylococcus aureus* at a university hospital. Infect. Control Hosp. Epidemiol., 16, 12-17.
4. Saravolatz, L. D., Pohlod, D. J. and Arking, L. M. (1982): Community acquired methicillin-resistant *Staphylococcus aureus* infections: A new source for nosocomial outbreaks. Ann. Intern. Med., 97: 325-329.
5. Salaria, M. and Singh, M.(2001): Methicillin-resistant *Staphylococcus aureus*. Indian Pediatr., 38, 29-36.
6. Vardhan, M. S., Aggarwal, D. S. and Gupta, R. L.: In press.
7. Herold, B. C., Immergluck, L. C., Maranan, M. C., Lauderdale, D. S., Gaskin, P. E., Boyle, V. S., Leitch, C. D. and Daum, R. S. (1998): Community acquired methicillin- resistant *Staphylococcus aureus* in children with no identified predisposed risk. JAMA, 279: 593-598.
8. Berman, D. S., Eisner, W. and Kreiswirth, B. (1993): Community acquired methicillin- resistant *Staphylococcus aureus* infection. N. Engl. J. Med., 329, 1896.
9. Rosenberg, J. (1995): Methicillin-resistant *Staphylococcus aureus* (MRSA) in the community: who's watching ? Lancet, 346, 132-133.
10. Saravolatz, L. D., Markowitz, N., Arking, L. M., Pohlod, D. J. and Fisher, E. (1982): Methicillin-resistant *Staphylococcus aureus*. Epidemiologic observations during a community acquired outbreak. Ann. Intern. Med., 96, 11-16.
11. Moreno, F., Crisp, C., Jorgenson, J. H. and Patterson, J. E. (1995): Methicillin-resistant *Staphylococcus aureus* as a community organism. Clin. Infect. Dis., 21, 1308-1312.
12. O'Brien, F. G., Pearman, J. W., Gracy, M., Riley, T. V. and Grubb, W. B. (1999): Community strain of methicillin-resistant *Staphylococcus aureus* involved in a hospital outbreak. J. Clin. Microbiol., 37, 2858-2862.
13. Baird, D. (1996): *Staphylococcus*: cluster forming cocci. p. 245-261. *In* Collee, J.G., Fraser, A.G., Marimion, B.P. and Simmons, A. (ed.), Practical Medical Microbiology. 14th ed. Churchill Livingstone, Ltd., Edinburgh.
14. Thornsberry, C., McDoughal, L. K. (1983): Successful use of broth microdilution in susceptibility testing for methicillin resistant (heteroresistant) *Staphylococci*. J. Clin. Microbiol., 18, 1084-1091.
15. National Committee for Clinical Laboratory Standards (1997): Performance standards for antimicrobial disc susceptibility test. 6th ed. M2-46. National Committee for Clinical Laboratory Standards, Villanova, Pa.
16. Boyce, J. M. (1998): Are the epidemiology and micro-

biology of methicillin-resistant *Staphylococcus aureus* changing? JAMA, 379, 623-624.
17. Hollis, R. J., Barr, J. L., Doebbeling, B. N., Pfaller, M. A. and Wenzel, R. P. (1995): Familial carriage of methicillin-resistant *Staphylococcus aureus* and subsequent infection in premature neonate. Clin. Infect. Dis., 21, 328-32.
18. Pate, K. R., Nolan, R. L., Bannerman, T. L. and Feldman, S. (1995): Methicillin- resistant *Staphylococcus aureus* in the community. Lancet , 346, 978.
19. Kloos, W. E. (1998): *Staphylococcus*. p. 577-632. *In* Collier, L., Balows, A. and Sussman, M. (ed.), Topley & Wilson's Microbiology and Microbial Infections. Vol. 2. 9th ed. Oxford University Press, New York.