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Epidemiology and Clinical Features of Methicillin-Resistant *Staphylococcus Aureus* (MRSA) at the University Hospital, Jeddah, Saudi Arabia

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ABSTRACT. This retrospective chart review describes the prevalence, demography, and clinical characteristics of patients colonized or infected with methicillin-resistant *Staphylococcus aureus* (MRSA) for the year 1998 at King Abdulaziz University Hospital (KAUH), Jeddah, Saudi Arabia. Results of MRSA positive cultures of clinical specimens obtained as part of investigations for suspected infections were retrieved from the Infection Control Department's records. Charts of patients were reviewed. Of 292 *S. aureus* identified, 111 (38%) were MRSA or 6.0 MRSA isolate per 1,000 admissions which represented a marked increase over MRSA prevalence in 1988 (<2%). Nosocomial acquisition occurred in 74.8% All age groups were affected, but 45.9% of patients were in the "extremes of age" group < 1 or > 60 years). The prevalence was highest in the medical ward (27%), followed by the pediatrics combined medical and surgical ward (20.7%), Out-patient Department (18%), the adult surgical ward (17.1%), and the intensive care unites (17.1%). Two thirds (66.7%) of cases represented infection and the rest represented colonization. Surgical wounds (31.1%), chest (27%), and endovascular catheters (20.3%) were the most common sites of infection. Bacteraemia occurred in 27% of patients. Local signs (68.9%), and fever (60.8%) were the most common clinical manifestations. Respiratory distress and septic shock occurred in 28.4% and 6.8% of cases, respectively. Of 74 patients with MRSA infection, and 37 patients with MRSA colonization, 91.9% and 56.8% received antibiotics in the preceding 6 weeks, respectively (P<0.0001). The total mortality of patients with MRSA infection was 60.8% (45/74). Mortality attributable to MRSA infection was 37.8% (28/74). The prevalence of MRSA is high and rapidly increasing at KAUH, as it is worldwide. Control measures to prevent the spread of MRSA in hospitals should continue with reinforcement of hygienic precautions and development of policies to restrict the use of antibiotics.

KEYWORDS: MRSA, Saudi Arabia, Infection, Colonization

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Introduction

Staphylococcus aureus is one of the most common pathogens well known for causing a variety of infections ranging from relatively benign skin infections to life-threatening systemic illnesses such as pneumonia, endocarditis, septic arthritis, osteomyelitis, and subcutaneous or visceral abscesses. *Staphylococcus aureus* is usually sensitive to cloxacillin (or methicillin), amoxicillin/clavulinate, first generation cephalosporins, erthromycin, clindamycin, and glycopeptides such as vancomycin and teicoplanin. Methicillin-resistant *Staphylococcus aureus* (MRSA) is important in that it is also usually resistant to the other antibiotics except glycopeptides which can only be administered through the intravenous route. MRSA is primarily a nosocomial pathogen that has emerged in the 1980s as a major cause of infection and colonization in hospitalized patients[1,2]. More recently, this organism has also been implicated as a cause of community-acquired infections in individuals with a recognized predisposing risk factor, such as recent contact with a health care facility, nursing home residence, or parenteral substance abuse[3-7]. Community-acquired MRSA infections in the absence of identified risk factors have also been increasingly reported[7-12]. The prevalence of MRSA has increased worldwide over the past decade with marked variations in different regions. It is generally high in the USA[13], southern European countries[14, 15], and Japan[16], but is low (less than 10% of *S. aureus* isolates) in Sweden, Denmark, and the Netherlands[14, 17, 18]. In the USA, the prevalence of MRSA increased from 2% in 1974 to approximately 50% in 1997[19-21]. In England and Wales, resistance to methicillin among *Staphylococcus aureus i*solated from blood or cerebrospinal fluid was stable at bout 1.5% of isolates during 1989-91, but increased thereafter to 13.2% in 1995[22]. Currently, in UK hospitals, prevalence of MRSA has reached epidemic levels and incidents involving MRSA has risen 12-fold since 1991 and were in 1999 responsible for 37% of all *S. aureus* infections, compared with only 3% in 1991[23]. A prevalence of over 30% was also observed in other southern European countries such as Spain, France, and Italy[14]. A high prevalence of MRSA has also been reported from Malaysia[24], Ethiopia[25], and other developing countries such as Kenya, Sri Lanka, and Tunisia[26].

The prevalence of MRSA in Saudi Arabia is not well defined and published data are limited[27, 28]. This study describes the prevalence of MRSA and the demographic and clinical characteristics of patients colonized or infected with this organism for the year 1998 at King Abdulaziz University Hospital (KAUH), Jeddah, Saudi Arabia.

Materials and Methods

Institution and patient population: KAUH is a tertiary care teaching hospital with a bed capacity of 265 bed and 18,492 admissions in 1998, the time of this retrospective review. Hospital units included adult and pediatric medical, surgical, and intensive care units, and obstetrics and gynecology unit. The hemodialysis unit was not opened during the study period and there is as yet no burn unit. Patients with MRSA positive cultures from any body specimen were identified from January 1st, 1998 to December

31st, 1998 for this reveiw.

Data collection: During the study period, specimens for bacterial culture were obtained as part of septic screen for suspected infections. Surveillance cultures specific for MRSA colonization were not done during this period. All MRSA positive culture results were obtained from the Infection Control Department's records. Charts of all patients with these positive cultures were reviewed with standardized data collection. Information collected included patient demographics, mode of acquisition (nosocomial- or community-acquired), hospital units where patients stayed, comorbidities, surgery and other invasive procedures, presence of foreign devices, antibiotic use, previous hospitalization, clinical significance of MRSA (colonization versus infection), site and clinical manifestations of infections, complications, and outcome.

Microbiological methods: Susceptibility testing of *Staphylococcus aureus* isolates to oxacillin was perfromed using 1-microgram oxacillin disk diffusion method (Oxoid Limited, Basingstoke, Hampshire, England) according to published guidelines[29]. Oxacillin resistance was demonstrated by a zone of inhibition of 10 mm or less.

Definitions: MRSA isolates were considered community-acquired if they were recovered within 72 hours of admission, and nosocomial, if they were recovered beyond that period.

The clinical significance of MRSA isolation from different body specimens was classified into either infection or colonization based on the presence or absence of a potential source of MRSA infection, the patient's clinical status, and other relevant data. In the absence of any potential source or clinical evidence of infection, MRSA was considered to be colonizing the site from which a specimen was obtained.

The source of infecion was determined on the basis of clinical evidence and recovery of MRSA from an infected site.

Outcome of patients with MRSA infection was classified into 4 categories; recovery without complications, recovery following complications such as septic shock or respiratory failure, death due to MRSA infection, or death unrelated to MRSA infection. MRSA-attributable mortality was defined as death related to clinically and microbiologically documented MRSA infection or any of its complications (*e.g.*, septic shock and acute respiratory distress syndrome).

Data analysis: The Statistical Package of Social Sciences (SPSS) program was used for data analysis. Yates-corrected Chi-square test was used for comparison of proportions (categorial data).

Results

The total number of *Staphylococcus aureus* isolated during the study period was 292

TABLE 1. Clinical characteristics of 111 patients with MRSA infection or colonization.

Characteristics	Number of Patients	Percentage
Nosocomial Acquisition	83	74.8
Community Acquisition	28	25.2
Comorbidities	53	47.7
Diabetes Mellitus	24	21.6
Malignancy	12	10.8
End Stage Renal Failure	10	9.0
Cerebrovascular accident	8	7.2
Heart Failure	7	6.3
Chronic obstructive pulmonary disease	5	4.5
Human Immunodeficiency Virus (HIV)	2	1.8
Past history of MRSA infection or colonization	21	18.9
Previous hospitalization	20	18.0
Unit of Stay:		
Medical Ward	30	27.0
Pediatrics (Medical and Surgical) Ward	23	20.7
Out-Patient Department	20	18.0
Surgical Ward	19	17.1
Intensive Care Unit	12	10.8
Neonatal Intensive Care Unit	7	6.3
Clinical Significance		
MRSA Infection	74	66.7
MRSA colonization	37	33.3
Central venous catheter infection	10	13.5
Peripheral venous line infection	5	6.8
Surgical site infection	23	31.1
Pneumonia	20	27.0
Urinary tract infection	3	4.1
Others	13	17.6
Bacteremia (n: 74 patients)	20	27.0
Clinical manifestations of MRSA infection (n: 74 patients)		
Fever	45	60.8
Shock	5	6.8
Respiratory distress	21	28.4
Local signs	51	68.9
Outcome (n: 74 patients)		
Recovery	29	39.2
Death Due to MRSA infection	28	37.8
Death Due to other causes	17	23.0

isolates, of which 111 (38%) isolates were MRSA isolated from 111 patients, which translates to 6 (111/18,492 x 1,000) MRSA isolate per 1,000 patients. Twenty-eight (25.2%, or 1.5 per 1,000 patients) isolates were community-acquired and 83 (74.8%) isolates were nosocomial. Sixty-five (58.6%) patients were male and 46 (41.4%) were female, with a mean age of 31.8 ± 25.8 years. Twenty (18%) patients were below 1 month of age, 9 (8.1%) were between 1 and 12 months, 60 (54.1%) were between 1 and 60 years, and 22 (19.8%) were more than 60 years of age. Fifty-three (47.7%) patients were Saudi citizens and 58 (52.3%) were non-Saudi. The clinical characteristics of pa-

tients are summarized in Table 1. MRSA caused infection in 74 (66.7%) of the cases, and in the remaining 37 (33.3%) patients, it represented colonization. Surgical site infections (31.1%), pneumonia (27%), and endovascular catheter infections (20.3%) were the most common types of infection. Bacteraemia occurred in 20 (27%) patients. Local signs (68.9%) such as erythema, purulent discharge, or tenderness of wounds or endovascular catheters' sites, and fever (60.8%) were the common clinical manifestations of MRSA infection. Respiratory distress and septic shock occurred in 28.4% and 6.8% of cases, respectively. Of 74 patients with MRSA infection, and 37 patients with MRSA colonization, 68 (91.9%) and 21 (56.8%) patients received antibiotics in the preceding 6 weeks, respectively, (P<0.0001, Odds ratio: 8.6, CI: 2.7-28.7). Of 111 patients with MSRA infection or colonization, 53 (47.7%) had at least one comorbidity; 39 (35.1%) patients had one, and 14 (12.6%) had 2 comorbidities.

A total of 29 (39.2%) of 74 patients with MRSA infections completely recovered from their infections; 21 (28.4%) patients had uneventful recovery, whereas the remaining 8 (10.8%) patients recovered following complications such as septic shock and/or respiratory failure requiring mechanical ventilation. The total morality of patients with MRSA infection was 60.8% (45/74). Mortality attributable to MRSA infection was 37.8% (28/74).

Discussion

This study at KAUH demonstrated a high prevalence of MRSA (38% of all *S. aureus* isolates). The prevalence has gradually increased from less than 2% in 1988 (unpublished data) to the current rate of 38%. The organism affected all age groups, but almost half (45.9%) the patients were in the "extremes of age" group (<1 or > 60 years). There was no predilection for any gender or nationality. Three-quarters (74.8%) of cases were nosocomial and the rest (25.2%) were community-acquired. The prevalence was highest in the medical ward (27%), followed by the pediatrics combined medical and surgical ward (20.7%), Out-Patient Department (18%), the adult surgical ward (17.1%), the intensive care unit (10.8%), and the neonatal intensive care unit (6.3%), in descending order of frequency. Approximately, two-thirds (66.7%) of cases represented infection and one-third (33.3%) represented colonization. This high infection to colonization ratio is similar to what has been observed by other researchers [30]. For instance, in an American MRSA outbreak, 260 of 286 (90.9%) affected patients were infected and not simply colonized[30]. Health care facilities that routinely perform MRSA surveillance cultures, which were not undertaken at KAUH during this study period, obviously have substantially lower infection to colonization ratio due to detection of more colonized patients. Therefore, this information is essential for appropriate comparison of MRSA prevalence and infection to colonization ratio of different centers.

Once confined mainly to hospitals, MRSA has recently been increasingly implicated in community-acquired infections and colonization in patients with predisposing risk factors such as recent contact with a health care facility, nursing home residence, or pa-

renteral substance abuse[3-7], as well as in patients without any recognized predisposing risk factor[7-12]. For instance, in two hospitals in the USA in the early 1990s, 28-67% of the patients with MRSA colonization had probable community acquisition[31, 32]. In five Canadian tertiary acute-care teaching hospitals in 3 provinces, patients with MRSA isolates present at admission accounted for 62% of MRSA isolations from 1990-1992 [11]. In a university hospital in the USA, 36 of 87 (41%) patients with MRSA had community acquisition; of these, 8 (22%) had no identified risk factors [33]. In a pediatric hospital in the USA, 8 and 35 cases of community-acquired MRSA infections were identified in the time periods 1988-1990 and 1993-1995, respectively. One (12.5%) and 25 (71.4%) cases had no identified risk increased from 0.1 per 1,000 admissions in 1988-1990 to 2.6 per 1,000 admissions in 1993-1995[12]. At KAUH, the prevalence of community acquisition of MRS (25.2% or 1.5 per 1,000 admissions) is moderately high when compared to published data. These studies, collectively, suggest the MRSA may be more widespread in the general population than has been previously appreciated.

Risk factors that have been associated with MRSA acquisition include older age, prolonged hospitalization, prior antibiotic therapy, more severe underlying disease and degree of disability, surgical procedures, presence in an intensive care or burn unit, having surgical wound infection, intravascular devices, mechanical ventilation, tracheotomy. pressure ulcers, or exposure to other infected or colonized individuals[1, 2, 34-39]. Not only does antibiotic therapy predispose to colonization with MRSA, but it also increases the risk of invasive disease and infection, as demonstrated by this study where significantly more patients with MRSA infection than those with MRSA colonization received antibiotics prior to positive MRSA culture (91.9% versus 56.8%, $P < 0.0001$). Other host factors associated with progression from colonization to infection include recent prior hospitalization, preceding surgical or wound debridement, and the number of invasive procedures[40].

The body sites most frequently affected by overt MRSA infection were surgical site infections (31.1%), pneumonia (27%), and endovascular catheter infections (20.3%) in descending order of frequency. Approximately, one-fourth of all patients with MRSA infections had bacteraemia but only 6.8% had overt septic shock. The total mortality of patients with MRSA infections was high (60.8%), and so was the mortality attributable to MRSA infection (37.8%). It was generally believed that MRSA strains were not more virulent than methicillin-susceptible (MSSA) ones[20, 41, 42]. Recent data, however, suggest that MRSA bacteraemia is associated with a significantly higher mortality rate than MSSA bacteraemia[43-45]. For instance, Romero-Vivas *et al* compared 100 cases of MSSA bacteraemia and 84 cases of MRSA bacteraemia; the mortality rates were 32% and 58.3%, respectively ($P < 0.01$), and methicillin resistance was found to be independently associated with mortality[43].

Conclusions

In conclusion, the prevalence of MRSA is high and rapidly increasing at KAUH as it is worldwide. One can foresee a time in the near future when the majority of *Staph-*

ylococcus aureus isolates would be resistant to methicillin, as happened with penicillin, to which almost all isolates of *Staphylococcus aureus* are currently resistant. Attempts to control the spread of MRSA in hospitals should continue with reinforcement of hygienic precautions and infection control measures[46]. Hospitals should also develop policies to restrict the use of antibiotics and establish monitoring systems for rapid identification of epidemics and determination of factors responsible for spread and colonization to allow for more targeted approach[47-49].

References

[1] **Thompson RL, Cabezudo I, Wenzel RP.** Epidemiology of nosocomial infections caused by methicillin-resistant *Staphylococcus aureus. Ann Intern Med* 1982; **97(3)**: 309-317.

[2] **Boyce JM.** Methicillin-resistant *Staphylococcus aureus.* Detection, epidemiology, and control measures. *Infect Dis Clin North Am* 1989; **3(4):** 901-913.

[3] **Saravolatz LD, Markowitz N, Arking L, Pohlod D, Fisher E.** Methicillin-resistant *Staphylococcus aureus.* Epidemiologic observations during a community-acquired outbreak. *Ann Intern Med* 1982; **96(1):** 11-16.

[4] **Saravolatz LD, Pohlod DJ, Arking LM.** Community-acquired methicillin-resistant *Staphylococcus aureus* infections: a new source of nosocomial outbreaks. *Ann Intern Med* 1982; **97(3):** 325-329.

[5] **Levine DP, Cushing RD, Jui J, Brown WJ.** Community-acquired Methicillin-resistant *Staphylococcus aureus.* endocarditis in the Detroit Medical Center. *Ann Intern Med* 1982; **97:** 330-338.

[6] **Craven DE, Rixinger AI, Goularte TA, McCabe WR.** Methicillin-resistant *Staphylococcus aureus* bacteraemia linked to intravenous drug abusers using a "shooting gallery". *Am J Med* 1986; **80(5):** 770-776.

[7] **Moreno F, Crisp C, Jorgensen JH, Patterson JE.** Methicillin-resistant *Staphylococcus aureus* as a community organism. *Clin Infect Dis* 1995; **21(5):** 1308-1312.

[8] **Berman DS, Eisner W, Kreiswirth B.** Community-acquired methicillin-resistant *Staphylococcus aureus* infection. *N Engl J Med* 1993; **329(25):** 1896.

[9] **Pate KR, Nolan RL, Bannerman TL, Feldman S.** Methicillin-resistant *Staphylococcus aureus* in the community. *Lancet* 1995; **346:** 978.

[10] **Hollis RJ, Barr JL, Doebbeling BN, Pfaller MA, Wenzel RP.** Familial carriage of methicillin-resistant *Staphylococcus aureus* and subsequent infection in a premature neonate. *Clin Infect Dis* 1995; **21(2):** 328-332.

[11] **Embil J, Ramotar K, Romance L, Alfa M, Conly J, Cronk S, Taylor G, Sutherland B, Louie T, Henderson E, *et al.*** Methicillin-resistant *Staphylococcus aureus* in tertiary care institutions on the Canadian prairies 1990-1992. *Infect Control Hosp Epidemiol* 1994; **15(10):** 646-651.

[12] **Herold BC, Immergluck LC, Maranan MC, Lauderdale DS, Gaskin RE, Boyle-Vavra S, Leitch CD, Daum RS.** Community-acquired methicillin-resistant *Staphylococcus aureus* in children with no identified predisposing risk. *JAMA* 1998; **279(8):** 593-598.

[13] **Boyce JM.** Increasing prevalence of methicillin-resistant *Staphylococcus aureus* in the United States. *Infect Control Hosp Epidemiol* 1990; **11(12):** 639-642.

[14] **Voss A, Milatovic D, Wallrauch-Schwarz C, Rosdahl VT, Braveny I.** Methicillin-resistant *Staphylococcus aureus* in Europe. *Eur J Clin Microbiol Infect Dis* 1994; **13(1):** 50-55.

[15] **Cookson BD**. Aspects of the epidemiology of MRSA in Europe. *J Chemother* 1995; **7(Suppl 3):** 93-98.

[16] **Oguri T.** Incidence and antimicrobial susceptibility of clinical isolates of MRSA from 1988 to 1990, from the results of 26 clinical laboratories in Tokyo and the surrounding area. *Nippon Rinsho* 1992; **50(5):** 952-960.

[17] **Vincent JL, Bihari DJ, Suter PM, Bruining HA, White J, Nicolas-Chanoin MH, Wolff M, Spencer RC, Hemmer M.** The prevalence of nosocomial infection in intensive care units in Europe. *JAMA* 1995; **274(8):** 639-644.

[18] **Vandenbroucke-Grauls C.** Management of methicillin-resistant *Staphylococcus aureus* in the Netherlands. *Rev Med Microbiol* 1998; **9:** 109-116.

[19] **Panlilio AL, Culver DH, Gaynes RP, Banerjee S, Henderson TS, Tolson JS, Martone WJ.** Methicillin-resistant *Staphylococcus aureus* in U.S. hospitals, 1975-1991. *Infect Control Hosp Epidemiol* 1992; **13(10):** 582-586.

[20] **Lowy FD.** *Staphylococcus aureus* infections. *N Engl J Med* 1998; **339(8):** 520-532.

[21] **Gaynes R, Culver D.** National Nosocomial Infections Surveillance System. Nosocomial methicillin-resistant *Staphylococcus aureus* (MRSA) in the United States, 1975-1996 (abstract #727). *Program and Abstracts of the IDSA 35th Annual Meeting, 1997.*

[22] **Speller DC, Johnson AP, James D, Marples RR, Charlett A, George RC.** Resistance to methicillin and other antibiotics in isolates of *Staphylococcus aureus* from blood and cerebrospinal fluid, England and Wales, 1989-95. *Lancet* 1997; **350:** 323-325.

[23] **Mayor S.** England sets standard to reduce hospital acquired infection. *BMJ* 1999; **319:** 1392.

[24] **Hanifah YA, Hiramatsu K, Yokota T.** Characterization of methicillin-resistant *Staphylococcus aureus* associated with nosocomial infection in the University Hospital, Kuala Lumpur. *J Hosp Infect* 1992; **21(1):** 15-28.

[25] **Geyid A, Lemeneh Y.** The incidence of methicillin-resistant *Staphylococcus aureus* strains in clinical specimens in relation to their beta-lactamase producing and multiple-drug resistance properties in Addis Ababa. *Ethiop Med J* 1991; **29(4):** 149-161.

[26] **Hart CA, Kariuki S.** Antimicrobial resistance in developing countries. *BMJ* 1998; **317:** 647-650.

[27] **Alghaithy AA, Bilal NE, Gedebou M, Weily AH**. Nasal carriage and antibiotic resistance of *Staphylococcus aureus* isolates from hospital and non-hospital personnel in Abha, Saudi Arabia. *Trans R Soc Trop Med Hyg* 2000; **94(5):** 504-507.

[28] **Zaman R, Dibb WL.** Methicillin-resistant *Staphylococcus aureus* (MRSA) isolated in Saudi Arabia: epidemiology and antimicrobial resistance patterns. *J Hosp Infect* 1994; **26(4):** 297-300.

[29] **National Committee for Clinical Laboratory Standards.** Performance Standards for Antimicrobial Disk Susceptibility Tests. *Approved Standard M2-A5.* National Committee for Clinical Laboratory Standards, Villanova, PA 1993.

[30] **Myers JP, Linnemann CC Jr .** Bacteraemia due to methicillin-resistant *Staphylococcus aureus. J Infect Dis* 1982; **145(4):** 532-536.

[31] **Linnemann CC Jr, Moore P, Staneck JL, Pfaller MA.** Reemergence of epidemic methicillin-resistant *Staphylococcus aureus* in a general hospital associated with changing staphylococcal strains. *Am J Med* 1991; **91(suppl 3B):** 238S-244S.

[32] **Nettleman MD, Trilla A, Fredrickson M, Pfaller MA.** Assigning responsibility: using feedback to achieve sustained control of methicillin-resistant *Staphylococcus aureus. Am J Med* 1991; **91(suppl 3B):** 228S-232S.

[33] **Layton MC, Hierholzer WJ Jr, Patterson JE.** The evolving epidemiology of methicillin-resistant *Staphylococcus aureus* at a university hospital. *Infect Control Hosp Epidemiol* 1995; **16(1):** 12-17.

[34] **Crossley K, Loesch D, Landesman B, Mead K, Chern M, Strate R.** An outbreak of infection

caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. I. Clinical Studies. *J Infect Dis* 1979; **139(3):** 273-279.

[35] **Strausbaugh LJ, Jacobson C, Sewell DL, Potter S, Ward TT.** Methicillin-resistant *Staphylococcus aureus* in extended-care facilities: experiences in a Veterans' Affairs nursing home and a review of th literature. *Infect Control Hosp Epidemiol* 1991; **12(1):** 36-45.

[36] **Peacock JE JR, Marsik FJ, Wenzel RP.** Methicillin-resistant *Staphylococcus aureus:* introduction and spread within a hospital. *Ann Intern Med* 1980; **93(4):** 526-532.

[37] **Linneman CC Jr, Mason M, Moore P, Korfhagen TR, Staneck JL.** Methicillin-resistant *Staphylococcus aureus*: experience in a general hospital over four years. *Am J Epidemiol* 1982; **115(6):** 941-950.

[38] **Cohen SH, Morita MM, Bradford M.** A seven-year experience with methicillin-resistant *Staphylococcus aureus. Am J Med* 1991; **91(suppl 3B):** 233S-237S.

[39] **Coello R, Jimenez J, Garcia M, Arroyo P, Minguez D, Fernandez C, Cruzet F, Gasper C.** Prospective study of infection, colonization and carriage of methicillin-resistant *Staphylococcus aureus* in an outbreak affecting 990 patients. *Eur J Clin Microbiol Infect Dis* 1994; **13(1):** 74-81.

[40] **Longfield JN, Townsend TR, Cruess DF, Stephens M, Bishop C, Bolyard E, Hutchinson E.** Methicillin-resistant *Staphylococcus aureus* (MRSA): risk and outcome of colonized vs. infected patients. *Infect Control* 1985; **6(11):** 445-450.

[41] **French GL, Cheng AF, Ling LM, Mo P, Donnan S.** Hong Kong strains of methicillin-resistant and methicillin-sensitive *Staphylococcus aureus* have similar virulence. *J Hosp Infect* 1990; **15(2):** 117-25.

[42] **Harbarth S, Rutschmann O, Sudre P, Pittet D.** Impact of methicillin-resistance on the outcome of patients with bacteraemia caused by *Staphylococcus aureus. Arch Intern Med* 1998; **158(2):** 182-189.

[43] **Romero-Vivas J, Rubio M, Ferandez C, Picazo JJ.** Mortality associated with nosocomial bactermia due to methicillin-resistant *Staphylococcus aureus. Clin Infect Dis* 1995; **21(6):** 1417-1423.

[44] **Conterno LO, Wey SB, Castelo A.** Risk Factors for mortality in *Staphylococcus aureus* bacteraemia. *Infect Control Hosp Epidemiol* 1998; **19(1):** 32-37.

[45] **Blot S, Vandewoude K, Colardyn F.** *Staphylococcus aureus* Infections. *N Engl J Med* 1998; **339 (27):** 2025-2027.

[46] **Mulligan ME, Murray-Leisure KA, Ribner BS, Standiford HC, John JF, Korvick JA, Kauffman CA, Yu VL.** Methicillin-Resistant *Staphylococcus aureus:* a consensus review of the microbiology, pathogenesis, and epidemiology with implications for prevention and management. *Am J Med* 1993; **94(3):** 313-328.

[47] **Weinstein RA.** Controlling antimicrobial resistance in hospitals: infection control and use of antibiotics. *Emerging Infect Dis* 2001; **7(2):** 188-192.

[48] **Landman D, Chockalingam M, Quale JM.** Reduction in the incidence of methicillin-resistant *Staphylococcus aureus* and ceftazidime-resistant *Klebsiella pneumonia* following changes in a hospital antibiotic formulary. *Clin Infect Dis* 1999; **28(5):** 1062-1066.

[49] **Monnet DL.** Methicillin-resistant *Staphylococcus aureus* and its relationship to antimicrobial use: possible implications for control. *Infect Control Hosp Epidemiol* 1998; **19(8):** 552-559.

الخصائص الوبائية و السريرية لجرثومة ستافيلوكوكس أوريوس ذات المناعة للميثيسيلين في مستشفى جامعة الملك عبد العزيز بجدة في المملكة العربية السعودية

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المستخلص. يهدف البحث الى وصف النمط الوبائي و الخصائص السريرية للمرضى المصابون بجرثومة ستاف أوريوس ذات المناعة للميثيسلين لسنة ١٩٩٨ في مستشفى جامعة الملك عبد العزيز بجدة في السعودية .

تم استخراج نتائج العينات التي ثبت إيجابيتها لجرثومة ستاف أوريوس ذات المناعة للميثيسلين من قسم مكافحة العدوى و من ثم تم مراجعة ملفات جميع المرضى الإيجابيين لهذه الجرثومة .