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Antibiotic Resistance of Community and Hospital Acquired Methicillin-Resistant *Staphylococcus aureus* Isolates from Clinical Specimens

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Abstract: Problem statement: Methicillin-Resistant *Staphylococcus aureus* (MRSA) is responsible for an increasing number of serious hospital and community acquired infections. Increased emergence in MRSA resistance to antibiotics is a growing problem. **Approach:** The resistance of MRSA to 20 antibiotics agents were studied. Also comparison of antibiotics resistance of community and hospital acquired MRSA were performed. Meanwhile the profile of antibiotics resistance of different clinical specimens among community and hospital acquired MRSA were evaluated. The clinical specimens of wound, urine, diabetic foot, skin abscess and sputum were collected from 1189 patients from March 2008-2009 at Hawler, Maternity and Rizgary teaching hospitals in Erbil, Iraq, 377 of *Staphylococcus aureus* were isolated and identification by standard methods, 114 MRSA were detected by detection PBP2a. Antibiotics resistance for MRSA were determined by the agar dilution method according to CLSI and BSAC guidelines. **Results:** The percentages of resistance in all hospital acquired MRSA were higher than community acquired MRSA. Among community acquired MRSA, the highest percentage (73.33%) of wound specimens were resistance to tetracycline, erythromycin and azithromycin. About 14% of urine samples were resistance to tobramycin, levofloxacin, moxifloxacin and rifampicin, 12.5% of diabetic foot was resistance to tobramycin, moxifloxacin and rifampicin. The resistance to tobramycin and rifampicin among MRSA cause skin abscess were 10 and 75% of sputum specimens were resistance to azithromycin and ciprofloxacin. Among hospital acquired MRSA isolates, 92% of wound specimens were resistance to tetracycline, 85.71% of urine samples were resistance to erythromycin and azithromycin. All sputum specimens were resistance to erythromycin. **Conclusion/Recommendations:** The most antibiotics affected agents MRSA were gatifloxacin, moxifloxacin and rifampicin. Physicians should be aware about MRSA and order for diagnostic and antibiotics sensitivity test. The use of antibiotics on random scale without antibiotic sensitivity testing must be restricted.

Key words: Methicillin-Resistant *Staphylococcus aureus* (MRSA), antibiotic resistance, Penicillin-Binding Protein (PBP), nucleic acid, clinical specimens, soft-tissue infections, controlling nosocomial transmission, hospital acquired, antibacterial agents

INTRODUCTION

Antibiotics resistance among a variety of bacterial pathogens is now well documented and is an increasingly important consideration when evaluating therapeutic choice and healthcare cost (Resch *et al*., 2009; Amsterdam *et al*., 2010). Even control continuous use of antibiotics has resulted in multi-resistant bacterial strains all over the world. Antibiotic resistance is the major problem of global dimensions with a significant impact on morbidity, mortality and healthcare-associated costs (AL-Haj *et al*., 2010). The escalations in antibiotic resistance have presented challenges to healthcare providers, making the selection of effective empiric therapy increasingly difficult (Deasy, 2009).

Staphylococcus aureus is a leading cause of both hospital acquired and community acquired infections

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(Alp *et al*., 2009). They are transmitted among patients and visitors. The drug resistant strains are arising rapidly and thus making the treatment difficult. As a result, Methicillin-Resistant *Staphylococcus aureus* (MRSA) infection is a significant cause of high mortality and morbidity worldwide. Rapid identification of infected patients and interruption of strain transmission is very crucial in controlling the spread of infection (Bassetti *et al*., 2009; Baykam *et al*., 2009; Himabindu *et al*., 2009; Yamamoto *et al*., 2010).

The most significant resistance bacterium in terms of overall economic impact is MRSA (Koskinen *et al*., 2008). The differentiation of MRSA strains from other strains of *Staphylococcus aureus* has important implications for the treatment and management of patients with *Staphylococcus aureus* infections (Grisold *et al*., 2002; Garau *et al*., 2009; Amsterdam *et al*., 2010). There is a marked difference between the resistances profiles of Methicillin-Sensitive *Staphylococcus aureus* compared to MRSA isolates (Kim, 2009; Gould *et al*., 2010).

MRSA becomes resistant to antibiotics by acquiring a foreign DNA called SCCmec. The SCCmec region contains several genes, including the *mecA* gene, which confers resistance against methicillin (Kaito *et al*., 2011). Methicillin resistance is imparted by the 2.1-kb *mecA* gene, which encodes the 78-kDa PBP2a. PBP2a, as a transpeptidase, facilitates cell wall synthesis and bacterial growth at concentrations of β-lactams inhibitory to native penicillin-binding proteins (Kim, 2009). PBP2a acts as a surrogate transpeptidase that takes over the biosynthetic functions of the normal PBPs when the bacteria encounter β-lactam antibiotics in the environment (Oliveira *et al*., 2002; Pereira *et al*., 2009). MRSA has persisted and implies cross-resistance to all available β-lactam antibiotics (Glikman *et al*., 2008). The MRSA isolates were resistant to antibiotics other than β-lactam antibiotics (Cardoso *et al*., 2007).

The spectrum of infections due to MRSA varies from mild skin infections to serious and invasive diseases such as surgical site infections, lower respiratory tract infections, urinary tract infections and skin infections (Niederman, 2009; Fadeyi *et al*., 2010; Yamamoto *et al*., 2010; Dugal and Mamajiwala, 2011).

MATERIALS AND METHODS

During the period March 2008-2009, the clinical specimens including wound, urine, diabetic foot, skin abscess and sputum were collected from 1189 patients at Hawler, Maternity and Rizgary teaching hospitals in Erbil, Iraq using standard bacteriological methods (Masaadeh and Jaran, 2009; Mohammadi *et al*., 2010). The infections were classified into the community and hospital acquired MRSA.

Specimens were inoculated onto blood agar and mannitol salt agar. The plates were incubated aerobically at 37°C for 18-24 h, 377 of *Staphylococcus aureus* (231 community acquired and 263 hospital acquired) was identified on the basis of a positive Gram stain, tube coagulase test and API STAPH system (bioMérieux, France) (Mahon *et al*., 2006).

Detection 114 of MRSA (56 community acquired and 58 hospital acquired) by detection of PBP2a by PBP2a kit (Oxoid, Japan) was performed according to the manufacturer's instructions using colonies from Mueller-Hinton agar (Brown *et al*., 2005; Mohanasoundaram and Lalitha, 2008).

Antibiotics resistance was determined by the agar dilution method for Penicillin G (Sigma-Aldrich), Cefotaxime (Sigma-Aldrich), Ceftriaxone (Mepha), Cefepime (Exir), Tetracycline (Sigma-Aldrich), Doxycycline (Sigma-Aldrich), Amikacin (Sigma-Aldrich), Tobramycin (Sigma-Aldrich), Erythromycin (Sigma-Aldrich), Azithromycin (Fluka), Clarithromycin (Sigma-Aldrich), Ciprofloxacin (Fluka), Gatifloxacin (Cipla), Levofloxacin (Sigma-Aldrich), Moxifloxacin (Bayer), Ofloxacin (Sigma-Aldrich), Clindamycin (Sigma-Aldrich), Rifampicin (Sigma-Aldrich) and Chloramphenicol (Sigma-Aldrich) according to Wikler (2006) and BSAC (2011) guidelines and breakpoint.

Data were analyzed with SPSS software. Chi-square test was used to compare differences in antibiotics resistance. A p-value of <0.05 was considered for calculating statistical significance.

RESULTS

Community and hospital acquired MRSA combined different resistance phenotypes were noted, the percentages of resistance in all hospital acquired MRSA were higher than community acquired MRSA, but statistically the different were not significant except amikacin ($P = 0.024$). The total of phenotypic resistance in community and hospital acquired MRSA are listed in Table 1.

Table 2 shows that the antibiotic resistance patterns of MRSA isolated from clinical specimens were found to be variable. Highest percentages of the isolates showed resistance to β-lactam antibiotics. The effects of gentamicin, amikacin, tobramycin, erythromycin, levofloxacin and moxifloxacin were significantly different on the clinical specimens.

Table 1: Antibiotic resistance of community and hospital acquired MRSA

	Community acquired n = 56		Hospital acquired n = 58		Statistical analysis	
Antibiotic	n	(%)	n	n	X^2	P-value
Penicillin G	56	100.00	58	100.00	0.02	0.895
Cefotaxime	54	96.43	56	96.55	0.02	0.893
Ceftriaxone	55	98.21	57	98.28	0.02	0.894
Cefepime	55	98.21	57	98.28	0.02	0.894
Tetracycline	34	60.71	49	84.48	1.38	0.241
Doxycycline	19	33.93	27	46.55	0.70	0.402
Gentamicin	24	42.86	43	74.14	2.68	0.096
Amikacin	16	28.57	39	67.24	5.07	0.024
Tobramycin	9	16.07	13	22.41	0.37	0.545
Erythromycin	34	60.71	52	89.66	1.91	0.168
Azithromycin	38	67.86	48	82.76	0.58	0.445
Clarithromycin	27	48.21	35	60.34	0.52	0.472
Ciprofloxacin	29	51.79	47	81.03	2.16	0.141
Gatifloxacin	1	1.79	2	3.45	Not done	
Levofloxacin	9	16.07	14	24.14	0.56	0.454
Moxifloxacin	7	12.50	11	18.97	0.57	0.451
Ofloxacin	18	32.14	35	60.34	2.82	0.093
Clindamycin	25	44.64	38	65.52	1.37	0.242
Rifampicin	6	10.71	15	25.86	2.06	0.151
Chloramphenicol	15	26.79	22	37.93	0.68	0.411

Table 2: Antibiotic resistance profiles of the community and hospital acquired MRSA isolates from clinical specimens

	Wound n = 54		Urine n = 21		Diabetic foot n = 16		Skin abscess n = 10		Sputum n = 13		Statistical analysis	
Antibiotic	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	X^2	P-value
Penicillin G	54	100.00	21.00	100.00	16.00	100.00	10.00	100.00	13.00	100.00	0.00	1.000
Cefotaxime	54	100.00	18.00	85.71	15.00	93.75	10.00	100.00	13.00	100.00	0.82	0.936
Ceftriaxone	54	100.00	20.00	95.24	16.00	100.00	10.00	100.00	12.00	92.31	0.29	0.991
Cefepime	54	100.00	20.00	95.24	16.00	100.00	10.00	100.00	12.00	92.31	0.29	0.991
Tetracycline	47	87.04	14.00	66.67	9.00	56.25	7.00	70.00	6.00	46.15	7.32	0.120
Doxycycline	25	46.30	9.00	42.86	5.00	31.25	3.00	30.00	4.00	30.77	3.13	0.537
Gentamicin	36	66.67	16.00	76.19	5.00	31.25	4.00	40.00	6.00	46.15	13.64	0.009
Amikacin	32	59.26	13.00	61.90	6.00	37.50	3.00	30.00	1.00	7.69	30.94	<0.001
Tobramycin	16	29.63	3.00	14.29	2.00	12.50	1.00	10.00	0.00	0.00	20.12	<0.001
Erythromycin	46	85.19	17.00	80.95	3.00	18.75	7.00	70.00	13.00	100.00	36.88	<0.001
Azithromycin	43	79.63	16.00	76.19	10.00	62.50	7.00	70.00	10.00	76.92	4.57	0.335
Clarithromycin	33	61.11	11.00	52.38	7.00	43.75	6.00	60.00	5.00	38.46	4.05	0.400
Ciprofloxacin	42	77.78	12.00	57.14	6.00	37.50	6.00	60.00	10.00	76.92	9.45	0.051
Gatifloxacin	2		3.70	0.00	0.00	1.00	6.25	0.00	0.00	0.00	0.00	Not done
Levofloxacin	14	25.93	2.00	9.52	3.00	18.75	3.00	30.00	1.00	7.69	10.57	0.032
Moxifloxacin	11	20.37	3.00	14.29	2.00	12.50	2.00	20.00	0.00	0.00	15.98	0.003
Ofloxacin	29	53.70	9.00	42.86	4.00	25.00	4.00	40.00	7.00	53.85	7.37	0.118
Clindamycin	32	59.26	11.00	52.38	8.00	50.00	4.00	40.00	8.00	61.54	2.93	0.569
Rifampicin	10	18.52	5.00	23.81	2.00	12.50	1.00	10.00	3.00	23.08	4.58	0.333
Chloramphenicol	20	37.04	6.00	28.57	5.00	31.25	2.00	20.00	4.00	30.77	2.77	0.597

According to specimen source among community acquired MRSA (Table 3), the highest percentage (73.33%) of wound specimens were resistance to tetracycline, erythromycin and azithromycin. about 14% of urine samples were resistance to tobramycin, levofloxacin, moxifloxacin and rifampicin. Only one sample (6.25%) of diabetic foot was resistance to gatifloxacin and 12.5% were resistance to tobramycin, moxifloxacin and rifampicin. The resistance to tobramycin and rifampicin among MRSA cause skin abscess were 10%. However 75% of sputum specimens were resistance to azithromycin and ciprofloxacin and all of the sputum specimens were resistance to erythromycin. Statistical analysis revealed that the differences of clinical specimen's resistance among community acquired MRSA were significant for gentamicin, amikacin, tobramycin, erythromycin, ciprofloxacin and moxifloxacin were significant.

Table 3: Antibiotic resistance profiles of community acquired MRSA isolates from clinical specimens

	Wound n = 15		Urine n = 7		Diabetic foot n = 16		Skin abscess n = 10		Sputum n = 8		Statistical analysis	
Antibiotic	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	X^2	P-value
Penicillin G	15	100.00	7	100.00	16	100.00	10	100.00	8	100.00	0.00	1.000
Cefotaxime	15	100.00	6	85.71	15	93.75	10	100.00	8	100.00	0.82	0.936
Ceftriaxone	15	100.00	7	100.00	16	100.00	10	100.00	7	87.50	0.61	0.961
Cefepime	15	100.00	7	100.00	16	100.00	10	100.00	7	87.50	0.61	0.961
Tetracycline	11	73.33	4	57.14	9	56.25	7	70.00	3	37.50	7.08	0.132
Doxycycline	5	33.33	4	57.14	5	31.25	3	30.00	2	25.00	7.61	0.17
Gentamicin	8	53.33	5	71.43	5	31.25	4	40.00	2	25.00	14.85	0.005
Amikacin	4	26.67	3	42.86	6	37.50	3	30.00	0	0.00	32.65	<0.001
Tobramycin	5	33.33	1	14.29	2	12.50	1	10.00	0	0.00	22.39	<0.001
Erythromycin	11	73.33	5	71.43	3	18.75	7	70.00	8	100.00	33.75	<0.001
Azithromycin	11	73.33	4	57.14	10	62.50	7	70.00	6	75.00	1.70	0.790
Clarithromycin	8	53.33	3	42.86	7	43.75	6	60.00	3	37.50	3.15	0.533
Ciprofloxacin	9	60.00	2	28.57	6	37.50	6	60.00	6	75.00	14.01	0.007
Gatifloxacin	0	0.00	0	0.00	1	6.25	0	0.00	0	0.00	Not done	
Levofloxacin	1	6.67	1	14.29	3	18.75	3	30.00	1	12.50	8.431	0.077
Moxifloxacin	2	13.33	1	14.29	2	12.50	2	20.00	0	0.00	14.23	0.007
Ofloxacin	4	26.67	3	42.86	4	25.00	4	40.00	3	37.50	3.91	0.418
Clindamycin	6	40.00	2	28.57	8	50.00	4	40.00	5	62.50	2.86	0.582
Rifampicin	1	6.67	1	14.29	2	12.50	1	10.00	1	12.50	1.60	0.810
Chloramphenicol	4	26.67	2	28.57	5	31.25	2	20.00	2	25.00	1.41	0.843

Table 4: Antibiotic resistance profiles of hospital acquired MRSA isolates from clinical specimens

	Wound n = 39		Urine n = 14		Sputum n = 5		Statistical analysis	
Antibiotic	n	(%)	n	(%)	n	(%)	X^2	P-value
Penicillin G	39	100.00	14	100.00	5	100.00	0.00	1.000
Cefotaxime	39	100.00	12	85.71	5	100.00	0.70	0.704
Ceftriaxone	39	100.00	13	92.86	5	100.00	0.17	0.919
Cefepime	39	100.00	13	92.86	5	100.00	0.17	0.919
Tetracycline	36	92.31	10	71.43	3	60.00	3.50	0.174
Doxycycline	20	51.28	5	35.71	2	40.00	1.38	0.502
Gentamicin	28	71.79	11	78.57	4	80.00	0.25	0.882
Amikacin	28	71.79	10	71.43	1	20.00	20.50	<0.001
Tobramycin	11	28.21	2	14.29	0	0.00	18.67	<0.001
Erythromycin	35	89.74	12	85.71	5	100.00	0.59	0.757
Azithromycin	32	82.05	12	85.71	4	80.00	0.11	0.945
Clarithromycin	25	64.10	8	57.14	2	40.00	3.01	0.222
Ciprofloxacin	33	84.62	10	71.43	4	80.00	0.65	0.722
Gatifloxacin	20	5.13	0	0.00	0	0.00	Not done	
Levofloxacin	13	33.33	1	7.14	0	0.00	23.49	<0.001
Moxifloxacin	90	23.08	2	14.29	0	0.00	15.60	<0.001
Ofloxacin	25	64.10	6	42.86	4	80.00	5.75	0.056
Clindamycin	26	66.67	9	64.29	3	60.00	0.20	0.907
Rifampicin	90	23.08	4	28.57	2	40.00	2.39	0.303
Chloramphenicol	16	41.03	4	28.57	2	40.00	1.29	0.525

Among hospital acquired MRSA isolates (Table 4), the spectrum of phenotypes resistance ranged from resistance to all β-lactams antibiotics to variable resistance to non-β-lactams antibiotics according to clinical specimens. About 92% of wound specimens were resistance to tetracycline and 5.13% was resistance to gatifloxacin. Out of 14 MRSA collected from urine sample, 85.71% were resistance to erythromycin and azithromycin and 7.14% was resistance to levofloxacin. All sputum specimens were resistance to erythromycin, while sensitive to tobramycin, gatifloxacin, levofloxacin and moxifloxacin. Statistical analysis revealed that the differences of clinical specimens resistance to amikacin, tobramycin, levofloxacin and moxifloxacin were significant among hospital acquired MRSA.

DISCUSSION

The increasing prevalence of multi-drug resistant organisms with few or no treatment options such as

MRSA in hospitalized patients and to a lesser extent, in the community are a serious cause for concern and have become a global problem (AL-Haj *et al*., 2010).

The epidemiology of drug resistance can exhibit remarkable geographical variability and rapid evolution over time, due to a complex interplay of factors involved in the selection and spread of different resistant genes and MRSA (Rossolini and Mantengoli, 2008; Yamamoto *et al*., 2010). Moreover, antibiotic resistance phenotype were differed strikingly between hospital and community acquired MRSA found by present study, which is in agreement with other studies (Enayet *et al*., 2006; Nimmo *et al*., 2006; Daum, 2007; Martino *et al*., 2008; Rehm, 2008; Nastaly *et al*., 2010). The different data may be referred to overuse and/or abuse of surgical antibiotic prophylaxis in hospitals are common. Antibiotics are extensively used as growth promoters in poultry production or to control infectious disease. Anti-microbial exercise and/or especially abuse is considered to be the most vital selecting force to antibiotics resistance of bacteria (Akond *et al*., 2009). Although new antibiotics can effectively treat some resistant pathogens and more research is needed to develop novel antibiotics, bacteria will eventually develop resistance to any antibiotic with time. The misuse and overuse of antibiotics drive the emergence and spread of resistance.

Measures for controlling nosocomial transmission of MRSA include prospective laboratory-based surveillance; placing patients colonized or infected with MRSA in isolation; use of barrier precautions such as gloves and gowns; hand washing and hand antisepsis; and careful environmental cleaning in patient rooms. Reducing overuse of broad-spectrum antimicrobial agents may also contribute to efforts to control MRSA. Screening patients to determine whether they are colonized with MRSA has also been shown to be useful in the number of settings (Coia *et al*., 2006; Humphreys *et al*., 2009). Similarly, screening and eradication of MRSA from colonized healthcare workers have been recognized and recommended as an important part of a comprehensive infection control policy for this organism (Fadeyi *et al*., 2010).

CONCLUSION

The following conclusions can be drawn from the studies summarized here: (1) Antibiotic resistance patterns of hospital acquired MRSA were found to be higher than community acquired MRSA; (2) The most antibiotics affected agents MRSA were gatifloxacin, moxifloxacin and rifampicin and (3) The study information's can be used to assist in design of treatment and to plan for preparing a hospital infection control guidelines.

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