Prevalence and Antimicrobial Susceptibility of Methicillin-Resistant *Staphylococcus aureus* (MRSA) from Outpatients with Chronic Rhinosinusitis in Al-Kut/Wasit Province/Iraq

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Abstract

Staphylococcus aureus is a significant factor in the development of chronic rhinosinusitis (CRS). Prevalence of sinonasal methicillin-resistant *S. aureus* (MRSA) appears to be increasing among CRS patients. For that this study was designed to detect the prevalence and antimicrobial resistance of MRSA isolates from Iraqi patients with CRS living in Al-Kut city/Wasit Province. Out of 264 patients with CRS, *S. aureus* was isolated from 71 (one isolate per patient) (26.8%). Of these patients, 37 (52.1%) were females (age range 2 months to 61 year) and 34 (47.8%) were males (age range 3 to 70 year). Fifty two (73.2%) of *S. aureus* isolates were MRSA, of which 71.1% were *mecA*-positive. Distribution of MRSA was significantly ($P \leq 0.05$) associated with patient's age, whereas it was insignificantly associated with patient's gender. The highest resistance rates of MRSA were against β -lactams except carbapenems, whereas the highest susceptibility was for vancomycin, followed by imipenem, genatmycin, meropenem and ciprofloxacin. Twenty two (30.9%) isolates were multidrug resistant (MDR), of which 20 (28.1%) were MRSA and 2 (2.8%) were methicillin-sensitive *S. aureus* (MSSA). No isolate showed resistance or sensitivity to all antimicrobials included in this study. Highest rate of multidrug resistance was to 13 antimicrobials (1 isolate which was MRSA), whereas the lowest rate was to 6 antimicrobials (3 isolates which were MRSA).

Conclusions: These results indicate the high prevalence of MRSA among Iraqi patients with CRS and reveal its predominance in our community. Furthermore, high percentage of these isolates were MDR, which urge us to reevaluate the empiric treatment of these cases.

Keywords: MRSA; CRS; Prevalence; Antimicrobial resistance

Introduction

Chronic rhinosinusitis (CRS) is an inflammation of the nose and sinuses in which the signs and symptoms last for more than 12 weeks and characterize by nasal blockage/obstruction/congestion, nasal discharge, facial pain, and/or reduction of smell [1]. Cases of CRS are subdivided into cases with polyps (CRSwNP) and cases without polyps (CRSwtNP) [2]. Chronic rhinosinusitis represents a multifactorial inflammatory disorder, rather than simply a persistent bacterial infection [3]. *Staphylococcus aureus* and anaerobic bacteria are the main isolates in chronic sinusitis [4].

In healthy subjects the sinuses are nonsterile environments that are colonized with aerobic and anaerobic bacteria, including *S. aureus* [4]. *Staphylococcus aureus* colonization rates have been found to be around 33.3% and 27.3% in healthy individuals and in patients with CRS, respectively [2]. Others [5] reported that *S. aureus* isolation rates in CRS patients range from 15% to 70%. Furthermore, it was suggested that *S. aureus* is a significant factor in the development of CRS [6,7]. Increased recovery rate of methicillin-resistant *S. aureus* (MRSA) in patients with upper respiratory tract infections, including acute and chronic maxillary sinusitis, was reported [8,4], so that incidence of MRSA in these patients is becoming a clinical concern [9]. Methicillin-

resistant *S. aureus* (MRSA) is any strain of *S. aureus* that is resistant to all β -lactams due to acquisition of a transpeptidase, PBP2a, involved in cell wall synthesis that has low affinity for β -lactam antibiotics. This PBP2a is encoded by *mecA* gene. This gene is complex, contains insertion sites for plasmids and transposons that facilitate acquisition of resistance to other antibiotics (multidrug resistance: MDR), hence there is restricted number of treatment choices for infections caused by MRSA [10]. Sinonasal infection caused by MRSA usually treated by a combination of oral and topical antibiotics for 1 to 2 weeks. Trimethoprim/sulfamethoxazole or clindamycin are usually used orally. While intravenous antibiotics are limited to severe infections [11,12].

Prevalence of sinonasal MRSA appears to be increasing, and many unanswered questions remain regarding its role in sinonasal infection and its treatment [4,9]. Furthermore, the risk of MRSA presence in the infected sinus may not only lead to failure of antimicrobial therapy, but may also serve as a source for the spread of these organisms to other body sites, as well as an origin for dissemination to other individuals [13]. So that the sooner an MRSA infection is diagnosed, and the susceptibility to antimicrobial agents established, the sooner appropriate therapy and control measures are crucial steps in treating, controlling, and preventing MRSA infections [14]. In addition, as a result of changing resistance profiles of these bacteria, empiric treatment options need to be evaluated [15]. In Iraq, several studies

were carried out regarding the prevalence and antimicrobial resistance of MRSA from different clinical materials, but little is known about these bacteria in patients with CRS. For that this study was designed to detect the prevalence and antimicrobial resistance pattern of MRSA isolates from Iraqi patients with CRS living in Al-Kut city/ Wasit Province, using phenotypic and genotypic protocols.

Materials and Methods

Definition and diagnosis of CRS

Chronic rhinosinusitis can be defined as inflammation of the nose and paranasal sinuses with persistence of signs and symptoms for 12 weeks and more without total cure. The diagnosis of CRS depended on the above definition in addition to findings of two major factors, or one major and two minor factors [1] as follows: after full ENT history and examination the diagnosis of CRS was done according to the symptoms & signs that are characteristics of CRS. Major symptoms include: facial pain/pressure; facial congestion/fullness; nasal obstruction/blockage; nasal discharge/purulence/dicoloured; posterior drainage; hyposomia/anosmia; purulence on nasal examination and fever (acute RS only). Whereas minor symptoms are: headache; fever (nonacute); halitosis; fatigue; dental pain; cough; and ear pain/pressure. The diagnosis was confirmed by flexible nasopharyngoscopy to find signs of CRS like oedema, polypoidal mucosa, mucopus in the middle turbinate or mucopurulent discharge in the postnasal space around the Eustachian tube.

Patients

This study included 264 (150 females and 114 males) patients with CRS aged 2 months to 82 years. They were outpatients attending Otolaryngology clinic in Al-Kut City/Wasit Province/Iraq. History of antibiotic usage was documented. Some of the patients were on antibiotic treatment with amoxicillin for 1 to 2 days before specimen collection.

This work was approved by Wasit Health Administration/Wasit Province/Iraq. Also, all bacterial isolates in this study were collected and analysed anonymously. Therefore, consent from the patient was not required.

Specimen collection and processing

Specimens were collected during October 1st, 2013 to April 30th, 2014. Each patient had a specimen collected from the nares with a dry, unmoistened swab. The tip of the collection swab was inserted approximately 2.56 cm into the nares and rolled five times in each nostril. Collected specimens were inoculated directly onto mannitol salt agar plates (Oxoid) that were incubated for 24 to 48 h at 35°C and examined for growth and mannitol fermentation [16].

Identification of *S. aureus* species

Isolates that produced yellow colonies on mannitol salt agar screen were primarily identified as *Staphylococcus* spp. by colony morphology and pigmentation on tryptic soy agar, Gram stain, and catalase test. Biochemical identification of *S. aureus* was based on tube coagulase and hemolysin production [17].

Also all mannitol fermenting isolates were subjected to species identification by PCR protocol according to Ruzickova et al. [18]. Each isolate was subcultured on tryptic soy agar plates for 24 h at 37°C.

From the agar plate a loopfull was taken and suspended in 88 µl sterile distilled water, then 10 µl of extraction buffer and 2 µl of extraction enzyme were added according to the manufacturer's instructions (KAPA Express Extract, KAPA Biosystem, USA). Bacterial suspensions were run for 10 min at 75°C, followed by 5 min at 95°C in a DNA thermocycler (MultiGene, Labnet International, Inc., USA) and cell debris were removed by centrifugation (12,000 rpm for 1 min). Five µl of supernatant was used as a template DNA in PCR. Genomic DNA of bacterial isolates was amplified by PCR using primers targeted to conserved *S. aureus* sequence (SAU-JIRS10: F:5'-ATA AGA GAT GGC GGT ACT AAA -3' and SAU-JIRS11: R:5'-TAA GGC GGA TTA CAC GTT ACT -3', with amplicon size of 532 bp). PCR amplification reactions were performed in a final volume of 25 µl containing lyophilized Mastermix (AccuPowder PCR PreMix, Bioneer, Korea) dissolved in sterile distilled water, 20 pmol concentrations of each primer and 5 µl of DNA template. The cycling parameters were as follows: an initial denaturation at 94°C for 4 min; followed by 25 cycles of 94°C for 30 s, 54°C for 30 s, and 70°C for 90s; and with a final extension at 72°C for 7 min. The amplified PCR products were subjected to electrophoresis at a 2% agarose gel in 0.5X TBE buffer.

Phenotypic screening for methicillin resistance

Detection of MRSA was carried out using oxacillin screen agar and cefoxitin disc diffusion test according to CLSI instructions [19].

Oxacillin screen agar

Mueller-Hinton agar (MHA) plates containing 4% NaCl and 6 µg/ml of oxacillin (Sigma, USA) were prepared. Plates were inoculated with 10 µL of 0.5 McFarland suspension of the isolate by streaking in one quadrant and incubated at 35°C for 24 h. Plates were observed carefully in transmitted light for any growth. Any growth after 24 h was considered oxacillin resistant.

Cefoxitin disc diffusion test

All the isolates were subjected to cefoxitin disc diffusion test using a 30 µg disc (Bioanalyse). A 0.5 McFarland standard suspension of the isolate was made and lawn culture done on MHA plate. Plates were incubated at 37°C for 18 h and zone diameters were measured. An inhibition zone diameter of ≤ 21 mm was reported as oxacillin resistant and ≥ 22 mm was considered as oxacillin sensitive.

Genotypic screening for methicillin resistance (PCR amplification for detection of *mecA* gene)

All *S. aureus* isolates were screened for *mecA* gene by PCR. The *mecA* gene was amplified using primers as described by Murakami et al. [20] (F:5' AAA ATC GAT GGT AAA GGT TGG C 3', R: 5' AGT TCT GCA GTA CCG GAT TTG C 3'). PCR amplification reactions were performed in a final volume of 25 µl containing lyophilized Master mix (AccuPowder PCR PreMix, Bioneer, Korea) dissolved in sterile distilled water, 20 pmol concentrations of forward and reverse primers and 5 µl of DNA template. The cycling parameters were as follows: an initial denaturation at 94°C for 5 min; followed by 40 cycles of 94°C for 30 s, 55°C for 30 s, and 70°C for 1min; and with a final extension at 72°C for 5 min. PCR products were visualized on 2% agarose gel with ethidium bromide dye under UV transilluminator. Amplicons of 533 bp were consistent with *mecA* gene amplification.

Susceptibility testing

Disk-diffusion tests were carried out with antibiotic-containing disks (Bioanalyse) on Mueller-Hinton agar plate (Himedia). The results were expressed as susceptible or resistant according to the criteria recommended by CLSI [19]. The following antimicrobial agents were tested: β -lactams [penicillin (P: 10 IU), amoxicillin (AX: 25 μ g), amoxicillin-clavulanic acid (AMC: 20/10 μ g), cefoxitin (FOX: 30 μ g), cefotaxime (CTX: 30 μ g), ceftazidime (CAZ: 30 μ g), ceftriaxone (CRO: 30 μ g), imipenem (IMP: 10 μ g), and meropenem (MEM: 10 μ g)], macrolides [clindamycin (DA: 10 μ g), azithromycin (AZM: 15 μ g) and clarithromycin (CLR: 15)], polypeptides [vancomycin (VA: 30 μ g)], sulfonamides [trimethoprim-sulfamethoxazole (SXT: 1.25/23.75 μ g)], fluoroquinolones [ciprofloxacin (CIP: 5 μ g)], and aminoglycosides [gentamicin (CN: 10 μ g) and amikacin (AK: 10 μ g)].

Statistical analysis

Differences in the distributions of the studied determinants were tested by Chi square [21]. A P value of ≤ 0.05 was considered to indicate statistical significance.

Results

Prevalence of MRSA among CRS patients

Staphylococcus aureus was isolated from 71 (one isolate per patient) out of 264 (26.8%) patients with CRS. Of these patients, 37 (52.1%) were females (age range 2 months to 61 years) and 34 (47.8%) were males (age range 3 to 70 years).

Fifty two (73.2%) *S. aureus* isolates were oxacillin resistant (MRSA) by screen test. *mecA*-mediated oxacillin resistance among these isolates was noted in 37 isolates (71.1%) by both cefoxitin disc diffusion and PCR tests.

As a whole, distribution of MRSA was significantly ($P \leq 0.05$) associated with patient's age. The highest rate of MRSA isolation was in the young age groups (11-20 and 21-30 year) in both females (26.9% vs. 42.3%, respectively) and males (23.0% vs. 30.7%, respectively) (Table 1). Whereas the isolation rate of MRSA was similar in females and males (50% each). So that prevalence of MRSA was insignificantly associated to the patient's gender.

Patient's age (year)	No. of <i>S. aureus</i> isolates (%)								
	MSSA			MRSA			Total		
	Females (n=11)	Males (n=8)	Total (n=19)	Females (n=26)	Males (n=26)	Total (n=52)	Females (n=37)	Males (n=34)	Total (n=71)
≤ 10	1 (9.0)	1 (12.5)	2 (10.5)	1 (3.8)	4 (15.3)	5 (9.6)	2 (5.4)	5 (14.7)	7 (9.8)
11-20	3 (27.2)	1 (12.5)	4 (21.0)	7 (26.9)	6 (23.0)	13 (25.0)	10 (27.0)	7 (20.5)	17 (23.9)
21-30	2 (18.1)	3 (37.5)	5 (26.3)	11 (42.3)	8 (30.7)	19 (36.5)	13 (35.1)	11 (32.3)	24 (33.8)
31-40	1 (9.0)	0	1 (5.2)	3 (11.5)	1 (3.8)	4 (7.6)	4 (10.8)	1 (2.9)	5 (7.0)
41-50	0	1 (12.5)	1 (5.2)	2 (7.6)	2 (7.6)	4 (7.6)	2 (5.4)	3 (8.8)	5 (7.0)
51-60	4 (36.3)	1 (12.5)	5 (26.3)	1 (3.8)	2 (7.6)	3 (5.7)	5 (13.5)	3 (8.8)	8 (11.2)
≥ 60	0	1 (12.5)	1 (5.2)	1 (3.8)	3 (11.5)	4 (7.6)	1 (2.7)	4 (11.7)	5 (7.0)
Total	11	8	19	26	26	52	37	34	71

Table 1: Distribution of *S. aureus* isolates among patients with CRS according to patient's gender and age.

Antimicrobial susceptibility of MRSA

All *S. aureus* isolates were subjected to antimicrobial susceptibility test against 17 antimicrobial drugs referred to 6 classes. All of *S. aureus* isolates were sensitive to vancomycin (100%) followed by imipenem (98.5%), Gentamycin (95.7%), meropenem (91.5%), and ciprofloxacin (83.0%) (Figure 1). However, only one isolate was sensitive to penicillin. Accordingly, the highest resistance (including resistant and intermediate resistant) was observed for penicillin (98.5%), followed by amoxicillin (95.8%), ceftriaxone (94.2%), ceftazidime (80.2%),

cefotaxime (76.0%), amoxicillin-clavulante (59.1%), azithromycin (56.3%), amikacin (53.4%), cefoxitin (52.1%), clindamycin (49.2%) and trimethoprim-sulfomethoxazole (42.2%).

For all antimicrobials included in this study resistance rate of MRSA was higher than that of MSSA (Figure 1). All (100%) isolates of MRSA were resistant to both penicillin and amoxicillin followed by ceftriaxone (94.1%), cefotaxime (92.3%), ceftazidime (84.5%), cefoxitin (71.1%), amoxicillin-clavulanate (63.4%), azithromycin (61.4%), amikacin (59.6%), clarithromycin (57.6%), trimethoprim-

sulfamethoxazole (50.0%). and clindamycin (46.1%). On the other hand, the highest susceptibility of MRSA was observed for vancomycin (100%), followed by imipenem (98.0%), genatmycin (94.2%), meropenem (90.3%), and ciprofloxacin (82.6%).

Twenty two (30.9%) isolates were MDR, of which 20 (28.1%) were MRSA and 2 (2.8%) were MSSA. No isolate showed resistance or sensitivity to all antimicrobials included in this study. Highest rate of multidrug resistance was to 13 antimicrobials (1 isolate which was MRSA), whereas the lowest rate was to 6 antimicrobials (3 isolates which were MRSA) (Table 2).

Figure 1: Percentages of antimicrobial resistance of 71 *S. aureus* isolates from patients with CRS.

Discussion

Chronic rhinosinusitis remains a highly prevalent disease with a major impact on overall quality of life [12]. Previous investigations [8,9] suggested that there is a significant etiological role of MRSA in CRS to which there is restricted number of treatment choices [15]. Therefore, we investigated prevalence and antimicrobial resistance of MRSA isolates from Iraqi patients with CRS. In this study, *S. aureus* was isolated from 26.8% of outpatients with CRS, of these isolates 73.2% were MRSA. Similar figures were reported from other parts of Iraq among patients with different clinical cases other than CRS. In the western city Ramadi, Lafi et al. [22] demonstrated MRSA in 73.1% of *S. aureus* isolates from blood culture and wound swabs. Also, in north Iraq, Mohammed [23] isolated MRSA in frequency of 72.0% from the nose of health work staff in surgery unit of Kalar General Hospital and from ear of patients attended to the same hospital. In Baghdad, it was found that 55.5% of *S. aureus* isolates from urinary tract infection (UTI) patients were MRSA [24]. High isolation percentages of MRSA from CRS patients were demonstrated in other parts of the world as in the USA where Brook et al. [8] compared the rate of recovery of MRSA between the periods 2001–2003 and 2004–2006. They found *S. aureus* in 15% of the patients with CRS between 2001 and 2003, 27% of which were MRSA, whereas during the period from 2004–2006 *S. aureus* was found in 20% of the patients with CRS, 61% of which were MRSA. In Nigeria, Ologe & Nwabuisi [25] demonstrated *S. aureus* in 47.1% of CRS patients. Also, Niederfuhr et al. [26] isolated *S. aureus* from 23.0%, and 38.7% of patients with CRS without and with nasal polyp, respectively. Our results and those of above mentioned workers reveal the significant increase in the rate of recovery of MRSA from patients with CRS. This may reflect the irrational and intensive use of

antimicrobial agents, especially β -lactams, in our community, as β -lactam antibiotics represent a selective pressure favor the selection and emergence of the mutant strains which express homogeneous resistance [23,27]. Others [8,28] reported that previous antibiotic treatment seems to be a risk factor for CRS infection with MRSA, as they isolated MRSA more often from treated individuals than from those not treated previously. This indicates a possible role for antibiotic usage to the emergence of MRSA-causing CRS [29]. Furthermore, Didier et al. [30] and Sachithanandam [28] explained the high prevalence of multiresistant MRSA by intensive use of topical and systemic antimicrobial agents which represent a highly selective pressure for antibiotic-resistant bacterial clones. In addition, bacterial exposure to some antibiotic classes may potentially induce endogenous, resistance-conferring mutations in bacterial genes that encode drug targets.

In both females and males distribution of MRSA was significantly ($P \leq 0.05$) associated with the patient's age where the highest rate of MRSA isolation was in the young age groups (11–20 and 21–30 year) in both females and males. Whereas the prevalence of MRSA was insignificantly associated to the patient's gender. These results were in partial agreement with Brook et al. [8] who found no differences in the age distribution, ethnicity or gender of the patients. Also, Niederfuhr et al. [26] reported that *S. aureus* detection rates were not influenced by gender, atopic state, or presence of asthma. Although the age distribution of MRSA was significantly different, our estimation may be not real as the number of patients at different age groups was not equal. About half of patients (127/264: 48.1%) were at the young age groups (11–20 and 21–30 year) in both females and males.

Overall resistance patterns of MRSA isolates included in this study were consistent with those obtained by other Iraqi researchers [23,31], where the highest resistance rates were against β -lactam antibiotics, except carbapenems. These high resistance rates were expected as most MRSA isolates in this study were *mecA* producers (71.1%). Our results were consistent with other researchers in Iraq [23,31] and across the world [4, 10,12,14, 19] where the most clinically important resistance mechanism in staphylococcal isolates to β -lactam antimicrobials is the acquisition of a *mecA* gene encoding a modified penicillin-binding protein (PBP), known as PBP2a. This PBP2a is intrinsically resistant to inhibition by β -lactams [10,14]. In addition, *mecA*-negative MRSA have another mechanisms of methicillin resistance, such as changes in affinity of PBPs for oxacillin [14,19]. Lack of control over antibiotic use in our country is the main reason for the development of increasing resistance to different antimicrobials among our clinical isolates including MRSA. Also, overuse of β -lactams for treating different cases in our hospitals is another reason for this high prevalence of MRSA. Emergence of antibiotic-resistant bacterial clones reflects the intensive use of antimicrobial agents [30]. So that there must be a scheduled rotation of β -lactams use with other antimicrobials in the study area to reduce this high antibiotic resistance among our isolates.

Amoxicillin-clavulanate, clindamycin, trimethoprim/sulfamethoxazole, and levofloxacin or ciprofloxacin represent the commonly used agents for treatment of CRS caused by MRSA [12]. In the study area, amoxicillin- clavulanate, cefotaxime, ceftriaxone and trimethoprim-sulfamethoxazole were the most commonly used antimicrobials for treatment of CRS patients as explained by Otolaryngologists. In this study resistance of MRSA to these antimicrobials were 63.4%, 71.1%, 92.3%, 94.2% and 50.0%, respectively. Also, 46.1% of MRSA were resistant to clindamycin.

Whereas 11.5% were resistant to ciprofloxacin. So that ciprofloxacin is most appropriate antimicrobial for treating CRS patients in the study area. For isolates that are resistant to ciprofloxacin the suitable alternative is gentamicin, since 3.8% of MRSA isolates were resistant to this antimicrobial. Amoxicillin-clavulanate, clindamycin, cefoxitin, cefotaxime, ceftriaxone and trimethoprim-sulfamethoxazole must be stopped in order to prevent distribution of resistance among *S. aureus* isolates. Gilmore et al. [10] reviewed that reduction of antibiotic use within the hospital could decrease nosocomial acquisition of multi-resistant bacteria, in addition to scheduled rotation of antibiotic use. Furthermore, strict compliance with infection control policies can aid in the reduction of nosocomial spread of multidrug-resistant MRSA.

Resistance pattern	No. (%) of <i>S. aureus</i> isolates	
	MRSA (n=20)	MSSA (n=2)
P, AX, AMC, FOX, OX, CTX, CAZ, CRO, CLR, DA, AZM, SXT, AK	1 (5.0)	0
P, AX, AMC, FOX, OX, CTX, CAZ, CLR, DA, AZM, SXT	1 (5.0)	0
P, AX, AMC, FOX, CTX, CAZ, CRO, CLR, AZM, SXT, AK	1 (5.0)	0
P, AX, AMC, FOX, CTX, CAZ, CRO, CLR, AZM, CN, AK	1 (5.0)	0
P, AX, AMC, FOX, CTX, CAZ, CRO, CLR, DA, AZM, CIP	1 (5.0)	0
P, AX, AMC, OX, CTX, CRO, CLR, DA, AZM, SXT	2 (10.0)	0
P, AX, FOX, OX, CTX, CRO, CLR, DA, AZM, SXT	1 (5.0)	0
P, AX, AMC, OX, CTX, CAZ, CRO, CLR, AZM, SXT	0	1 (50.0)
P, AX, AMC, FOX, OX, CRO, CLR, AZM, AK, CIP	1 (5.0)	0
P, AX, AMC, FOX, OX, CTX, CAZ, CRO, SXT, CIP	1 (5.0)	0
P, AX, OX, CTX, CRO, CLR, DA, AZM, SXT	4 (20.0)	0
P, AX, FOX, OX, CLR, DA, AZM, SXT	1 (5.0)	0
P, AX, AMC, CLR, DA, AZM, SXT, CIP	0	1 (50.0)
P, FOX, CRO, CLR, DA, AZM, CIP	1 (5.0)	0
P, AX, OX, CLR, DA, AZM, SXT	1 (5.0)	0
P, AX, FOX, CRO, SXT, AK	1 (5.0)	0
P, OX, CLR, DA, AZM, AK	1 (5.0)	0
P, OX, CLR, DA, AZM, SXT	1 (5.0)	0

Table 2: Antimicrobial resistance patterns of multidrug resistant *S. aureus* isolates from patients with CRS.

For all antimicrobials included in this study resistance rate of MRSA was higher than that of MSSA. Also, 38.4% (20/52) of MRSA were MDR compared to 10.5% (2/19) of MSSA. Cross-resistance of MRSA to other antibiotics (in addition to their resistance to β -lactams) is due to *mecA* and its associated DNA which act as a trap for integration of other determinants, including genes for resistance to fluoroquinolones, aminoglycosides, tetracyclines, macrolides, trimethoprim-sulfamethoxazole and erythromycin [10,14,15]. Also, many MDR MRSA strains are presently only susceptible to a single class of clinically available bactericidal antibiotic, the glycopeptides

(vancomycin and teicoplanin), and the widespread acquisition of the *vanA* or *vanB* determinants from enterococci would be a potential public health disaster [10]. All our *S. aureus* isolates (MRSA and MSSA) were sensitive to vancomycin which is not used for treatment of CRS patients in the study area. In other parts of Iraq, it was found that 92.0% [23] and 100% [31] of MRSA isolates were sensitive to vancomycin. So that vancomycin may be a drug of choice for serious cases caused by MDR MRSA. In the light of our results there is an obvious need for more effective antibiotic therapy for infections with MRSA. However, new therapeutic agents alone will not provide a long-term solution, and our attention to prevention must remain constant. Strict adherence to hospital infection-control practices, as well as appropriate use of antibiotics and improved surveillance systems to track the emergence of resistance patterns, are of primary importance as we look to the future usefulness of antibiotic therapy against this extremely adaptive organism [10].

Conclusions

Methicillin-resistant *S. aureus* had high prevalence among Iraqi outpatients with CRS, which indicate their predominance in our community. Furthermore, a significant percentage of these isolates were MDR, which urge us to reevaluate the empiric treatment of these cases.

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