

Use of cefoxitin as indicator for detection of Methicillin Resistant *Staphylococcus aureus*

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Abstract:

Rapid and accurate identification of Methicillin Resistant *Staphylococcus aureus* is essential in limiting the spread of this bacterium. The aim of study is the detection of Methicillin Resistant *Staphylococcus aureus* (MRSA) and determining their susceptibility to some antimicrobial agent.

A total of fifty clinical *Staphylococcus aureus*, isolated from the nose of health work staff in surgery unit of Kalar general hospital and from ear of patients attended to the same hospital. The susceptibilities of isolates were determined by the disc diffusion method with oxacillin (1 µg) and cefoxitin (30 µg), and by the mannitol salt agar supplemented with cefoxitin (MSA-CFOX), susceptibilities of isolates to other antimicrobial agent were determined by standard disc diffusion method, Brain heart infusion (BHI) agar with vancomycin was used for detection of vancomycin resistant *Staphylococcus aureus*.

out of fifty clinical isolates of *Staphylococcus aureus* 36/50(72%) considered to be MRSA according to MSA-CFOX growth and cefoxitin disc susceptibility results with critical diameter <27 mm but 35/50(68%) considered to be MRSA when critical diameter ≤21 mm was depended, while according to oxacillin disc 29/50(58%) considered to be MRSA, all isolates showed good susceptibility to imipenem (100%) with different pattern of susceptibility to other antibiotics, 4/50(8%) showed non-susceptible to vancomycin and grew on BHI agar with supplemented vancomycin.

high percentage of isolates were methicillin resistant and vancomycin resistance occurs among them which may refer to irrational use of antimicrobial agent, thus, necessitate implementation of good strategies for control of infection and use of antibiotic. and to use of cefoxitin as screening agent for rapid detection of MRSA in microbiology laboratories.

Key words: *Staphylococcus aureus*, MRSA screening, cefoxitin.

Introduction

The first isolate of methicillin-resistant *Staphylococcus aureus* (MRSA) was reported in 1961 in England[1]. Since then, MRSA has become a major cause of hospital acquired infection, and is being recognized with increasing frequency in community acquired infections throughout the world [2].

Nearly all MRSA isolates produce additional penicillin-binding protein

designated PBP2' or PBP2a with low binding affinities to practically all β-lactam antibiotics in clinical use, which are the most important group of antibiotics in the treatment of staphylococcal infections[2,3], This additional PBP2a encoded by *mecA* gene which is a component of a large DNA fragment designated *mec* DNA located at specific site of the *S. aureus* chromosome and has been suggested to

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be transmitted from other bacterial species[1]. Two regulator genes on mec DNA, designated *mecI* and *mecRI* thought to regulate the expression of *mecA* which can be either inducible or constitutive[4,5], also many other factors are involved in modulating the expression of methicillin resistance without altering levels of PBP2a [1,5]. Intact and full function mec regulatory genes appear to strongly repress the production of PBP2a. Hence, An MRSA carry intact *mec* DNA called pre-methicillin resistant *Staphylococcus aureus* (pre-MRSA) which is apparently methicillin susceptible[6]. A distinctive feature of methicillin resistance is its heterogeneous nature, the majority of cells in heterogeneous strains are susceptible to methicillin and expression of resistance occurs in only a small proportion of cells[1,7]. These strains seem to be on the increase, both in number and in the level of heterogeneity, β -lactam antibiotics represented a selective pressure favor the selection and emergence of the mutant strains which express homogeneous resistance from heterogeneous strains [8]. Detection of the *mecA* gene or its product, penicillin binding protein (PBP2a), is considered the gold standard for MRSA detection[2]. Since molecular methods are not available for most medical institutions. Thus, phenotypic methods for characterization of the resistance to methicillin are frequently evaluated and the Recent investigations suggest that disk diffusion using cefoxitin is superior to most previously recommended phenotypic methods, including oxacillin disk diffusion and oxacillin screen agar testing[9,10,11], particularly in strains with heterogeneous methicillin resistance that their detection may require induction of PBP2a by specific antibiotics or alteration of growth

conditions [3]. oxacillin may fail to detect them while cefoxitin is strong inducer for production of PBP 2a, and do not appear to be affected by hyper-production of penicillinase which may show methicillin resistant [3,9]. Further, cefoxitin has high affinity for *Staphylococcal* PBP4 that with PBP2 overproduction may also contribute in methicillin resistant [9].

MRSA are of particular clinical significance because they are resistant to all beta-lactam antibiotics and has cross-resistant to other antibiotics with high ability to be transmitted among hospitalized patients so called epidemic MRSA[1], As such the [glycopeptide](#), [vancomycin](#), is often deployed against MRSA. but infection caused by vancomycin intermediate resistant strain occurred in 1996 and since then infection due to vancomycin-resistant staphylococci (VRS) well documented[12]. vancomycin resistance is mediated by acquisition of the [vanA](#) gene which originates from the [enterococci](#) and codes for an enzyme that produces an alternative [peptidoglycan](#) to which [vancomycin](#) will not bind, therefore bacteria appear resistant[13].

Nasal carriage is a major risk factor for MRSA infection and may disperse the organism into the air [14]. Therefore, screening for carriers is an important infection control practice in many hospitals to prevent the spread of MRSA in the workplace.

The aim of the present study is the detection of Methicillin Resistant *Staphylococcus aureus* (MRSA) from nasal carrier nurse working staff and from patients with ear infection, and determining their susceptibility to some antimicrobial agents.

Materials Methods:

A total of Fifty *S. aureus* isolates, including 23 *S. aureus* isolated from the anterior nares of nursing staff in

maternity operative theater in the kalar General hospital and 27 *S. aureus* isolated from the ear of ear infection patient's attended to the same hospital during the period from February to May 2008, isolates identified depending on the morphology and cultural characteristic on the mannitol salt agar, oxidase, catalase, and slide coagulase tests [15].

Susceptibility testing was performed by disk diffusion on Mueller-Hinton agar (MHA) from Himedia. India, with 24-h incubation at 35°C. [16]. the antibiotic disks from bioanalyse company. Ankara-Turky were used with following potencies; amoxicillin (AX 25µg), amoxicillin/clavulanic acid (AMC 30µg), cephalothin (KF 30µg), tetracycline (TE 30µg), ciprofloxacin (CIP 30µg), erythromycin (ERY 15µg), clindamycin (CIL 2µg), vancomycin (VC 30µg), imipenem (IMP 10µg), the results were interpreted according to the standard zone diameter recommended by Soussy et al. [16].

Phenotypic method for detection of MRSA

i- All isolates were tested with a cefoxitin disk (FOX) 30 µg by disk diffusion method on MHA using confluent growth (10^8 cells/ml) standardized to 0.5 McFarland turbidity. and overnight incubation (18h) at 35°C⁽²⁾, and two interpretive breakpoints for zone diameter used, according to Felten et al. zone inhibition diameter < 27 mm considered to be resistant[9] and according to clinical laboratory standard institute interpretative criteria of resistance was considered \leq 21mm [17].

ii- Mannitol-salt agar supplemented with cefoxitin (MSA-

CFOX 6mg/liter) was used as selective media for isolation of MRSA. Swabs were placed in 400 µl sterile normal saline and vortexed, from the suspension, 50 µl was used to inoculate the media, (swabs were directly inoculated to the medium), plates incubated at 35°C and read after 18 and 48 h.[14].

iii-The susceptibility to oxacillin (OX) 1µg disc was made on MHA supplemented with 2% NaCl and using high density inoculum (10^8 cells/ml) for 18h at 35°C with critical diameter <13mm considered to be non-susceptible[9,18]. With all tests *S. aureus* ATCC 25923 used as quality control strain.

In cases of heterogeneous growth, defined as the occurrence of small colonies in the circular growth inhibition area, the diameter of the inner limit of the small colonies' inhibition zone was taken into account. Screening for vancomycin resistant *S.aureus* (VRSA) in the study isolates was made by brain-heart infusion agar (BHIA) containing 6µg/ml vancomycin with an inoculum of equivalent density to 0.5 McFarland standard and 24h of incubation at 35°C, *S. aureus* ATCC 25923 used as negative control .

Results:

Out of fifty *Staphylococcus aureus* isolates, 36 (72%) isolates considered to be MRSA according to MSA-CFOX screening method and cefoxitin susceptibility with inhibition zone diameter < 27mm. While according to cefoxitin inhibition zone diameter \leq 21mm, 35(68%) isolates considered to be MRSA. and oxacillin disk diffusion test showed 29(58%) MRSA isolates.

Table (1) inhibition zone diameters by millimeter of cefoxitin and oxacillin disk diffusion tests for 50 isolates, MSA-CFOX growth results

Tests isolates	OX	FOX	MSA-CFOX GROWTH	Tests isolates	OX	FOX	MSA-CFOX GROWTH
S.AN1	19	24	+2*	S.AE 26	6	6	+1
S.AN2	15	16	+1*	S.AE 27	20	30	-
S.AN3	17	30	-*	S.AE 28	18	28	-
S.AN4	14	18	+2	S. AE 29	6	6	+1
S.AN5	8	20	+1	S. AE 30	21	21	+1
S.AN6	10	20	+1	S.AE 31	13	30	-
S.AN7	9	16	+1	S.AE 32	14	20	+1
S.AN8	7	20	+1	S.AE 33	19	30	-
S.AN9	15	28	-	S.AE 34	17	28	-
S.AN10	6	14	+1	S.AE 35	6*	6	+1
S.AN11	6	16	+1	S.AE 36	15	36	-
S. AN12	10	17	+1	S.AE37	10	30	-
S. AN13	6	14	+1	S.AE38	6	6	+1
S. AN14	6	21	+2	S.AE39	6	21	+2
S. AN15	6	21	+2	S.AE40	6	6	+1
S. AN16	6	17	+1	S.AE41	6*	6*	+1
S. AN17	8	15	+1	S.AE42	6	6	+1
S. AN18	6	15	+1	S.AE43	11	20	+2
S. AN19	15	21	+1	S.AE44	12	20	+2
S. AN20	10	15	+1	S.AE45	8	20	+2
S.AN21	6	6	+1	S.AE46	11	14	+1
S. AN22	26	30	-	S.AE47	10	16	+1
S.AN23	19	28	-	S.AE48	21	15	+2
S.AE24	17	29	-	S.AE49	17	30	-
S.A E 25	28	13	+1	S.AE50	20	29	-

+2*= growth within 48 hr, +1* =growth after 24 hr, -* no growth after 48 hr, S.A= *Staphylococcus aureus*, N=nose, E= ear

Table (2) The susceptible and non-susceptible Percentage number of isolates to the used antibiotic.

	AX	AMC	KF	0XA	FOX	FOX*	IMP	CIL	ERY	VC	TE	CIP
Susceptible (%)	13/50 26%	16/50 32%	27/50 54%	21/50 42%	14/50 28%	15/50 32%	50/50 100%	32/50 64%	24/50 48%	46/50 92%	27/50 54%	45/50 86%
Non-susceptible (%)	37/50 74%	34/50 68%	23/50 46%	29/50 58%	36/50 72%	35/50 68%	0/50 0%	18/50 36%	26/50 52%	4/50 8%	23/50 46%	7/50 14%

Fox with critical diameter < 27 mm, FOX* with critical diameter ≤ 21mm

Table (3) The susceptibility of isolates to the antibiotics used.(inhibition zone diameters by millimeter).

ISOLATES Antibiotics	AX ≥21-<14	AMC ≥21-<14	KF ≥18-<12	IMP ≥22-<17	CIL ≥15-<15	ERY ≥22-<17	TE ≥19-<17	VC ≥17	CIP ≥22-<19
S.AN 1	12	15	18	35	27	25	27	20	30
S.AN 2	10	12	13	36	25	23	8	18	25
S.AN 3	10	22	20	30	25	23	20	19	32
S.AN 4	17	18	18	40	27	26	25	18	26
S.AN 5	13	13	8	30	21	19	8	20	23
S.AN 6	15	14	10	35	30	30	8	20	23
S.AN 7	9	12	6	35	30	32	6	17	25
S.AN 8	13	14	6	35	6	6	6	10	17
S.AN 9	8	12	10	35	30	30	19	19	27
S.AN 10	9	14	9	35	20	6	10	18	26
S.AN 11	8	12	9	35	27	25	27	17	22
S.AN 12	6	6	13	24	30	20	8	18	30
S.AN 13	8	10	6	35	26	27	6	20	28
S.AN 14	16	15	11	35	30	23	20	19	20
S.AN 15	8	15	20	37	22	6	6	20	24
S.AN 16	8	16	6	40	30	22	30	20	29
S.AN 17	10	10	8	38	22	6	10	16	20
S.AN 18	10	12	16	44	30	6	9	16	15
S.AN 19	15	17	21	44	10	6	9	20	26
S.AN 20	9	15	9	40	23	24	28	19	27
S.AN 21	15	15	6	25	6	7	6	25	23
S.AN 22	6	21	19	45	13	22	26	20	23
S.AN 23	24	22	23	40	30	8	20	25	24
S.AE 24	25	23	22	45	18	34	40	20	25
S.AE 25	21	22	20	40	23	25	20	20	26
S.AE 26	18	16	7	38	6	6	9	30	31
S.AE 27	23	23	25	40	24	24	33	19	24
S.AE 28	18	23	22	45	29	24	40	18	32
S.AE 29	26	30	8	36	6	6	8	26	25
S.AE 30	20	17	18	40	17	29	6	27	23
S.AE 31	17	16	20	40	12	14	26	23	27
S.AE 32	22	21	22	35	14	25	27	19	27
S.AE 33	30	30	23	40	20	30	30	26	30
S.AE 34	29	25	22	40	13	15	18	20	28
S.AE 35	22	22	18	40	21	6	24	21	25
S.AE 36	20	18	12	30	16	17	27	25	28
S.AE 37	6	18	11	40	6	6	25	27	22
S.AE 38	21	30	19	45	8	16	7	22	25
S.AE 39	20	18	8	37	7	12	7	19	15
S.AE 40	30	22	20	35	6	8	30	19	26
S.AE 41	6	6	8	41	10	8	8	12	17
S.AE 42	6	6	10	40	6	6	20	22	31
S.AE 43	13	17	15	30	14	12	13	18	20
S.AE 44	19	18	11	35	10	12	12	18	30
S.AE 45	16	18	10	32	6	6	10	20	35
S.AE 46	11	13	10	30	20	10	20	22	28
S.AE 47	15	18	11	28	22	22	23	20	35
S.AE 48	12	18	19	40	20	25	22	19	32
S.AE 49	24	25	25	36	21	23	20	23	33
S.AE 50	24	27	29	38	30	26	22	25	30

The antibiotic resistance is shown in table (2) and (3), 74%, 68%, and 46%, were non-susceptible to AX, AMC, and KF, respectively, while 64%, 48%, 54%, 80%, 92%, and 100% were susceptible to CIL, ERY, TE, CIP, VC, and IMP respectively

In screening test for vancomycin resistant *Staphylococci* four isolates of *Staphylococcus aureus* grew on BHI agar supplemented with vancomycin after 24h.

Discussion:

Infections due to methicillin-resistant *Staphylococcus aureus* (MRSA) are an increasing problem worldwide inside and outside of hospitals. It is clinically and epidemiologically important for laboratories to be able to differentiate MRSA from MSSA. Not only for choosing appropriate antibiotic therapy for the individual patient, but also for control of MRSA transmission [19].

The Results of oxacillin susceptibility test showed that 29 (58%) of our isolates gave inhibition zone diameter

less than 13 mm were identified as MRSA, Ten out of 29 isolates showed heterogeneous growth around the oxacillin disk. oxacillin resistant strains should be considered as non susceptible to all beta-lactam antibiotics, whether they are associated or not with a beta-lactamase inhibitor and even if they showed susceptibility in-vitro because the mechanism, PBP2a production has low affinity for all beta-lactams and may be associated with emergence of methicillin resistance during antibiotic therapy of MRSA infection particularly with heterogeneous population[1,3,16].

While cefoxitin susceptibility results showed that 36(72%) of isolates gave cefoxitin inhibition zone <27 mm and considered as MRSA containing *mecA* gene[9], but according to CLSI interpretative criteria 35(68%) isolates gave cefoxitin inhibition zone diameter ≤ 21 mm and considered as MRSA [17]. several recent investigations supported the latter criteria for detection of *mecA* positive strains [2,11,20], one isolate S.AN1 gave 24mm cefoxitin inhibition zone diameter repeatedly and grew well on MSA-CFOX, thus necessitate the use of molecular method to confirm the detection of *mecA* gene and detect either this isolate is false positive or false negative MRSA.

Among the cefoxitin resistant isolates eight isolates showed susceptible to oxacillin but not considered as MSSA, because cefoxitin does not induce PBP2a production in MSSA strain, unless this strain is pre-MRSA[6].

Some strains with hyper-producer of penicillinase may show oxacillin resistance and will therefore falsely reported as MRSA but tests with cefoxitin do not appear to be affected to same extent as oxacillin by hyperproduction of penicillinase[3], in study isolates one *S.aureus* (S.AE37) showed resistance to oxacillin but

susceptible to cefoxitin and not grew on MSA-CFOX, thus, oxacillin resistance in this isolate may be due to the above mechanism.

Velasco et al. in their study concluded that In the absence of availability of molecular biology techniques, the cefoxitin disc was the best predictor of methicillin resistance in *S. aureus* from among the techniques tested[11].

Mannitol salt agar with cefoxitin (MSA-CFOX) used as selective medium for isolation of MRSA (*mecA* positive *S.aureus*) [14, 21]. In the present study MSA-CFOX allowed the growth of 36(72%) isolates and nine of them required 48 h of incubation time before these could be identified. A wide range of techniques has been used to detect and identify MRSA from clinical specimens, selective and differential culture media especially MSA supplemented with oxacillin are most widely employed [17], several investigators have demonstrated the superiority of cefoxitin for the identification of MRSA especially in strains with heterotypic expression thus their detection may require induction of PBP2a [9,21] and A recent report demonstrate that the detection rates of MRSA with MSA-CFOX was significantly higher than the detection rate with MSA supplemented with oxacillin [14].

The study isolates have different pattern of susceptibility upon the antibiotics susceptibility results, high percentage of isolates were non-susceptible to amoxicillin, and amoxicillin/clavulanic acid and 46% were non-susceptible to cephalothin, while 100% were susceptible to imipenem and 64%, 48%, 54%, 80%, 92% were susceptible to clindamycin, erythromycin, tetracycline, ciprofloxacin, and vancomycin respectively.

The *mec* gene in MRSA is complex, contains insertion sites for plasmids

and transposons that facilitate acquisition of resistance to other antibiotics [5], and the prevalence of strains resistant to specific antibiotic may be associated to the extent at which the antibiotic is used[15].

Different class of antibiotics such as vancomycin, linezolid, quinupristin/dalfopristin (streptogramin) and newer fluoroquinolones used for treatment of severe MRSA infection caused by multidrug resistant strain [12]. However, since 1996, MRSA strains with decreased susceptibility to vancomycin (minimum inhibitory concentration [MIC], 8-16 µg/ml) and strains fully resistant to vancomycin (MIC ≥ 32 µg/ml) have been reported[13].

In the present study four 4/50(8%) MRSA isolates showed resistance to vancomycin and gave small colonies within the inhibition zone around the vancomycin disc, in addition they grew as countable numbers of colonies (14-26) on BHIA with vancomycin 6mg/litre after 24 h of incubation. therefore these isolates may considered as vancomycin intermediate resistant VISA or VRSA 3, three of them isolated from the nasal carrier which represent important risk factor for infection and airborne dispersal of *S. aureus* in the hospital.

The development of resistance to vancomycin may be correlated to prolonged use or misuse of vancomycin [13], therefore it is important to ensure the prudent use of antibiotics to decrease the emergence of MRSA with restriction of vancomycin use, to prevent spread of VRSA.

It is concluded that high percentage of study isolates were methicillin resistant and vancomycin resistance occurs among them which may refer to irrational use of antimicrobial agent, thus, necessitate implementation of

good strategies for control of infection and use of antibiotic. and to use of cefoxitin as screening agent for rapid detection of MRSA in microbiology laboratories.

References:

1. Yoshida, R., Kuwahara-Arai, K., Baba T., Cui, L., Richardson, J.F., and Hiramatsu, K. 2003. Physiological and molecular analysis of a *mecA*-negative *Staphylococcus aureus* clinical strain that expresses heterogeneous methicillin resistance. *Journal of antimicrobial Chemotherapy*. 51: 247-255
2. Skov, R., Smyth, R., Larsen, A. R., Bolmstro, A., Karlsson, A., Mills, K., Frimodt-Moller, N. & Kahlmeter, G.2006. Phenotypic detection of methicillin resistance in *Staphylococcus aureus* by disk diffusion testing and Etest on Mueller-Hinton agar. *J Clin Microbiol*. 44: 4395-4399.
3. Brown, D.J.F., Edwards, D.I., Hawkey, P.M., et al. 2005. Guidelines for the laboratory diagnosis and susceptibility testing of methicillin-resistant *Staphylococcus aureus* (MRSA). *J. Antimicrob. Chemother*.56:1000-1018.
4. Chambers, H. F. 1997. Methicillin resistance in staphylococci: molecular and biochemical basis and clinical implications. *Clin. Microbiol. Rev*. 10:781-791.
5. Kaye, K.S., Fraimow, H.S., Abrutyn, E. 2000. pathogens resistant to antimicrobial agents epidemiology, Molecular Mechanisms, and Clinical Management. *Infect Dis Clin of North America*. 14(2): 293-319.
6. Darini, A.L.D.C., Palazzo, I.C.V., Felten, A. 2004. Cefoxitin Does Not Induce Production of Penicillin Binding Protein 2a in Methicillin-

- Susceptible *Staphylococcus aureus* Strains. J. Clin. Microbiol. 42;4:12-4413
7. González-Zorn, B., Jose P.M., Fiette, S.L., Shorte, S., Testard, A., Chignard, M., Courvalin, P., and Courvalin, C.G.2005. Bacterial and Host Factors Implicated in Nasal Carriage of Methicillin-Resistant *Staphylococcus aureus* in Mice. Infection and Immunity. 73;3: 1847-1851.
 8. Dancer, S.J. 2001. The problem with cephalosporins J. Antimicrob. Chemother. October . 48;4: 463 - 478.
 9. Felten, A., Grandy, B., Lagrange, B.H., and Casin, I.2002. Evaluation of three techniques for detection of low-level methicillin-resistant *Staphylococcus aureus* (MRSA): a disk diffusion method with cefoxitin and moxalactam, the Vitek 2 system, and the MRSA-screen latex agglutination test. J. Clin. Microbiol. 40:2766-2771
 10. Cauwelier, B., Gordts, B., Descheemaeker, P., and Van Landuyt, H. 2004. Evaluation of a disk diffusion method with cefoxitin (30 microg) for detection of methicillin-resistant *Staphylococcus aureus*. Eur. J. Microbiol. Infect. Dis. 23:389-392
 11. Velasco, D., del Mar Tomas, M., Cartelle, M., Beceiro, A., Perez, A., Molina, F., Moure, R., Villanueva, R. & Bou, G. 2005. Evaluation of different methods for detecting methicillin (oxacillin) resistance in *Staphylococcus aureus*. J Antimicrob Chemother. 55: 379–382.
 12. Boyce, J.M.2003.Update on resistant *Staphylococcus aureus* infections. National foundation for infect dis. VI. Issue 2.
 13. Sonavane, A.D.A.,and Mathur, M.2007. Screening for vancomycin intermediate - resistant *Staphylococcus aureus* among clinical isolates of MRSA. Indian J Med Microbiol .25:79-80
 14. Stoakes, L., Reyes, R., Daniel, J., Lennox, G., John, M.A., Lannigan, R., Hussain, Z.2006. Prospective Comparison of a New Chromogenic Medium, MRSA Select, to CHROMagar MRSA and Mannitol-Salt Medium Supplemented with Oxacillin or Cefoxitin for Detection of Methicillin-Resistant *Staphylococcus aureus*. J. Clin. Microbiol. 44:637-639
 15. Baird, D.1996. *Staphylococcus*: cluster-forming Gram-positive cocci. In : Macki and McCartne practical medical microbiology.(eds Colle JC, Barrie PM, Fraser AG, and Simmons A.)14 th ed. Chrchill livingston, Singapore. 1996.
 16. Soussy, C.J., Carret, G., Cavallo, J.D., Chardon, H., Chidiac, C., Choutet, P., Courvalin, P., Dabernat, H., Drugeon, H., Dubreuil, L., Goldstein, F., Jarlier, V., Leclercq, R., Nicolas-Chanoine, M.H., Philippon, A., Quentin, C., Rouveix, B., and Sirot, J. 2000-2001 Comité de l'Antibiogramme de la Société Française de Microbiologie. Communiqué. Pathol. Biol. 48:832-871.
 17. CLSI. 2007. Performance standards for antimicrobial susceptibility testing. CLSI approved standard M100-S17. Clinical and Laboratory Standards Institute, Wayne, PA.
 18. Mackenzie, A. M. R., H. Richardson, R. Lannigan, and D. Wood. 1995. Evidence that the National Committee for Clinical Laboratory Standards disk test is less sensitive than the screen plate for detection of low-expression-class methicillin-resistant

- Staphylococcus aureus*. J. Clin. Microbiol. 33:1909–1911.
19. Jain, A., Agarwal, A., and Verma, R.K.2008.Cefoxitin disc diffusion test for detection of meticillin-resistant staphylococci . J. Med. Microbiol. 57;8: 957 - 961
20. Fernandes, C. J., Fernandes, L. A., and Collignon, P.2005. Cefoxitin resistance as a surrogate marker for the detection of methicillin-resistant *Staphylococcus aureus*. J. Antimicrob. Chemother. 55:506–510
21. Smyth, R.W., and Kahlmeter, G. 2005. Mannitol Salt Agar-Cefoxitin Combination as a Screening Medium for Methicillin-Resistant *Staphylococcus aureus*. journal of clinical microbiology. 3797–3799

استخدام السفوكستين كمؤشر للكشف عن المكورات العنقودية *Staphylococcus aureus* المقاومة للمثيسيلين

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الخلاصة:

اجريت هذه الدراسة للكشف عن المكورات العنقودية المقاومة للمثيسيلين وتحديد حساسية العزلات تجاه عدد من المضادات الحيوية، عزلت خمسين عزلة بكتيرية للمكورات العنقودية من مسحات انف مأخوذة من الكادر الصحي في وحدة العمليات في مستشفى الكرار العام ومن مسحات اذن مأخوذة من المرضى المراجعين لنفس المستشفى واخضعت العزلات لاختبار الحساسية تجاه مضاد السفوكستين 30 مايكروغرام والاوكتاسيلين 1 مايكروغرام كمؤشر لمقاومة المثيسيلين بطريقة انتشار الاقراص القياسية وكذلك تجاه عدد من المضادات الحيوية المنتخبة، وكما تم زرع المسحات مباشرة على وسط المانيتول الملحي المضاف اليه السفوكستين للكشف المباشر عن المكورات العنقودية المقاومة للمثيسيلين، واستخدمت وسط نقيع القلب والمخ المضاف اليه الفانكوميسين للكشف عن العزلات المقاومة للفانكوميسين. بالاعتماد على نتائج حساسية العزلات تجاه مضاد السفوكستين وباعتبار قطر التثبيط الأقل من 27 ملم مقاوما للمثيسيلين 36(72%) عزلة اظهرت مقاومة للمثيسيلين ولكن باستخدام قطر التثبيط \geq 21ملم وجد 35(68%) عزلة مقاومة للمثيسيلين وكما اظهر وسط المانيتول الملحي الحاوي على السفوكستين 36(72%) عزلة مقاومة للمثيسيلين ولكن بالاعتماد على حساسية العزلات تجاه الاوكساسيلين وجد 29 (58%) عزلة مقاومة للمثيسيلين، واطهرت جميع العزلات حساسية تجاه مضاد الامينيم واعطت انماط مقاومة مختلفة تجاه المضادات الحيوية الاخرى وكما وجد 4(8%) من العزلات غير حساسة للفانكوميسين وعزلت على وسط نقيع القلب والمخ الحاوي على الفانكوميسين، وبذلك فان المكورات العنقودية المقاومة للمثيسيلين ظهرت بنسبة عالية في العزلات قيد الدراسة ومن ضمنها بعض العزلات كانت غير حساسه للفانكوميسين مما قد يشير الى الاستخدام العشوائي للمضادات الحيوية لذلك من الضروري تطبيق ستراتيجية جيدة للسيطرة على العدوى وعلى استخدام المضادات الحيوية، وكذلك استخدام السفوكستين في مختبرات الاحياء المجهرية للكشف عن المكورات العنقودية المقاومة للمثيسيلين.