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Co-Occurrence of Antibiotic Resistance in Extensively Drug-Resistant *Staphylococcus aureus* Isolated from Wounds in Tikrit Teaching Hospital

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Abstract: ***Background***: Much of the recent literature in this context has focused on tracing of highly multidrug-resistant (MDR) which might be disseminated in hospital environment particularly through areas of highly susceptible patients like intensive care unit, burn and surgery wards and children's lounge. The assessment of the disease burden is of priority for policymakers and public health officials to perform evidence of resource allocation and consequently to plan for the mitigation of threats to health. Methodology: The present work was conducted on 200 patients with wounds. Three hundreds were hospitalized and the other two hundreds were non-hospitalized attended the outpatient's clinic after operation for removal of stitches. Their ages ranged from 1-40 years. Wound swab was taken on the third postoperative day from hospitalized patients and on the seventh postoperative day from patients attended outpatient clinic. The isolated Staphylococcus aureus isolates were subjected to susceptibility diffusion discs of penicillin, benzathine penicillin, chloramphenicol, streptomycin, gentamicin, tetracycline, erythromycin, colistin, nitrofurantoin, Fucidin and clindamycin. ***Results***: The present study revealed that the percentage matrices were not symmetrical as determined by the considered resistance, e.g. 30% of strains resistant to tetracycline were resistant to streptomycin while 100% of the strains resistant to streptomycin were resistant to tetracycline. It was also found that 20% of strains resistant to erythromycin were resistant to gentamicin, while 83.3% of strains resistant to gentamicin were resistant to erythromycin at the same time. ***Conclusions***: It was seen that there were large numbers of trains resistant to pairs of antibiotics at the same time even of different antibiotics groups, e.g. colistain sulfate and erythromycin, erythromycin and tetracycline, clindamycin and penicillin. Nitrofurantoin showed no shared resistance with any of the antibiotics used in the present study.

Keywords: Wounds, *S. aureus*, Antibiotics, Co-Resistance.

Research Paper

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INTRODUCTION

Because of the wide usage of antibiotics in hospitals, it become a serious problem all over the world, especially in developing countries. This type of usage led to emergence of antibiotic-resistant bacteria in the hospital environment. Most of hospital infections are caused by multiply resistant microorganisms [1-5]. A significant continuous pose of threat has been made by infectious diseases on human life and property. Revealing of the true patterns of transmission of infectious diseases among society and planning a real effective prevention and control should be made to minimize the widespread of large-scale infectious disease [6]. Much of the recent literature in this context has focused on tracing of highly multidrug-resistant (MDR) which might be disseminated in hospital environment particularly through areas of highly susceptible patients like intensive care unit, burn and surgery wards and children's lounge [6, 7]. The assessment of the disease burden is of priority for policymakers and public health officials to perform evidence of resource allocation and consequently to plan for the mitigation of threats to health. The Burden of Communicable Diseases in Europe (BCoDE) report aimed to provide a practical policy for this burden assessment. A policy which was followed later by many national and international studies and analyses to be built [7-12].

Resistance to penicillin G by *S. aureus* appeared rapidly after introduction the drug. Later on there was increasing proportion of *S. aureus* strains that resist streptomycin and tetracycline within the first few years of introduction of these drugs. An outbreak of infection caused by strains of *S. aureus* resistant to gentamicin and methicillin occurred in special care baby unit [13]. Methicillin- resistant *S. aureus* (MRSA) strain has been greatly increased in recent years, and patients are likely to be infected with MRSA include elderly peoples and those with postoperative infection such as orthopedic, vascular surgery, patient with spinal injury, chronic skin ulcer, burn and chronic disease of respiratory and urinary tracts [14]. It has been found that erythromycin and clindamycin are substitutes to the use of B-lactam antibiotics or aminoglycosides, either alone or in combination with some of these drugs, however, resistance to these drugs arised as a problem during therapy, the so-called macrolide and lincosamide resistance phenotype which characterized by cross-resistance to all macrolides, lincosamides and streptogramins B. This resistance phenotype is, in many cases, plasmid and/or transposon mediated, and they found that there is no relationship between methicillin and macrolide-lincosamide resistance [15,16]. An outbreak of infection caused trimethoprim-resistant Enterobacteriaceae occurred at the University college hospital of London. It was found that geriatric patients previously treated with trimethoprim were the main source of this outbreak and this was plasmid-mediated and easily transferred from strain to another [17]. Resistance transfer studies and plasmid screening experiments demonstrated that multi-resistance phenotype was due to transmissible plasmid and/or transposons. These strains also shown resistance to cephalosporins which is chromosomaly encoded [18]. Most of hospital infections caused by multiple resistant organisms [13, 14], especially multiple resistant *S. aureus* which has been frequently isolated from patients.

MATERIALS AND METHODS

Patients

This study was carried out in teaching hospital of Tikrit. The present work was conducted on 200 patients with wounds. Three hundreds were hospitalized and the other two hundreds were non-hospitalized attended the outpatient's clinic after operation for removal of stitches. Their ages ranged from 1-40 years. The majority of these patients were from rural areas or referred to this hospital from other town's hospitals. General informations such as including demographic survey were listed. The acceptance for participation in the present study was taken from all the participants whose native language is Arabic. They were not mentally retarded and they were completely healthy considering hearing and speaking.

Sampling

Wound swab was taken on the third postoperative day from hospitalized patients and on the seventh postoperative day from patients attended outpatient clinic. Samples were taken by using sterile cotton swabs moistened with nutrient broth carried in test tubes contained 2 ml broth liquid [4]. Wound swabs were enriched in nutrient broth at 37 °C for 18 hours. Each sample was sub-cultured on mannitol salt agar and incubated at 37°C for 24 hours. Pure cultures were obtained after isolation on appropriate selective media. The suspected colonies were purified twice then sub-cultured on nutrient agar slants and kept at 4 0C for full identification and further studies.

Antibiotic Susceptibility Testing

A loopful growth from isolates of *Staphylococcus aureus* were inoculated into nutrient broth and incubated at 37°C for 18 hours. The bacterial suspensions were diluted with ringer solution. The proportion of dilution was 1:1000 [14]. Diluted bacterial suspension were poured onto the surface of the Muller-Hinton agar plates. The excess of bacterial suspensions were discarded using Pasteur pipette and plates were left for one hour at room temperature to dry. The antibiotic discs were selectively applied by using sterile forceps which was flamed after being cleansed with alcohol. The plates were incubated at 37 0C for overnight. The size of zones of inhibition were measured from edge of disc to the edge of inhibition of growth and the result was compared with standard diameter of inhibition zones for each antibiotic utilizing the method of Bauer *et al*., [15]. The following standard strain *Staphylococcus aureus* ATCC25923 was used as a reference strain.

Statistical Analyses

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics such as means, standard deviations, and frequency distributions were computed to summarize the data [14-16].

RESULTS

The degree of association between the antibiotic resistance among the 40 strains of Staphylococcus aureus was studied (Table 1). The present study showed a shared resistance between pairs of antibiotics tested. It was seen that there were large numbers of trains resistant to pairs of antibiotics at the same time even of different antibiotics groups, e,g. colistain sulfate and erythromycin, erythromycin and tetracycline, clindamycin and penicillin. Nitrofurantoin showed no shared resistance with any of the antibiotics used in the present study. The distribution of interaction scores was non-normal and Mann–Whitney statistical analysis justified significant difference indicates

($P<0.05$). Resistance patterns were not randomly distributed. Certain antibiotic classes showed stronger co-resistance associations. Clinically, the present results suggested linked resistance mechanisms, e.g., plasmid-mediated or cross-resistance.

Table 1. Matrix showed the association between phenotypic resistance to pairs of antibiotics among 40 strains of *Staphylococcus aureus* isolated from wounds.

										7	C*
									4	5	S
								4	4	4	CN
							6	6	9	10	TE
						17	5	6	9	10	E
					13	15	6	6	10	10	P
				16	19	14	4	4	8	9	CT
			0	0	0	0	0	0	0	0	F
		0	4	5	5	5	1	2	3	4	FU
	4	0	15	15	15	14	5	6	11	9	CL
14	5	0	15	14	16	14	5	6	10	10	TMP
CL	FU	F	CT	P	E	TE	CN	S	C	BP	

*= P, penicillin; BP, benzathine penicillin; C, chloramphenicol; S, streptomycin; CN, gentamicin; TE, tetracycline; E, erythromycin; CT, colistin; F, nitrofurantoin; FU, Fucidin; CL, clindamycin.

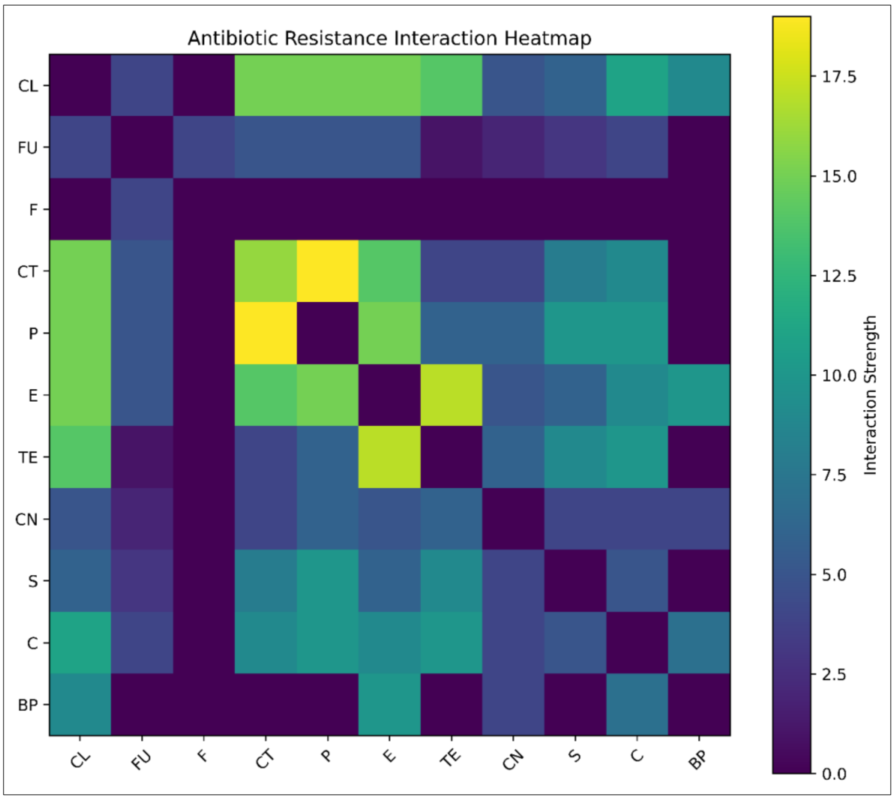

Figure 1: Heatmap of Phenotypic Resistance Associations among Antibiotics in *Staphylococcus aureus* Isolates. P, penicillin; BP, benzathine penicillin; C, chloramphenicol; S, streptomycin; CN, gentamicin; TE, tetracycline; E, erythromycin; CT, colistin; F, nitrofurantoin; FU, Fucidin; CL, clindamycin

The present study revealed that the percentage matrices were not symmetrical as determined by the considered resistance, e.g. 30% of strains resistant to tetracycline were resistant to streptomycin while 100% of the strains resistant to streptomycin were resistant to tetracycline (Table 2). It was also found that 20% of strains resistant to erythromycin were resistant to gentamicin, while 83.3% of strains resistant to gentamicin were resistant to erythromycin at the same time. The Mann–Whitney U test revealed significant differences in resistance distributions among antibiotics. Penicillin, tetracycline, trimethoprim, and clindamycin demonstrated significantly higher resistance levels compared to gentamicin, streptomycin, and fucidin ($p < 0.05$). Nitrofurantoin exhibited the lowest resistance, differing significantly from all other antibiotics ($p < 0.001$). These findings indicate a non-random, structured pattern of antimicrobial resistance, likely reflecting differential usage and underlying resistance mechanisms.

Table 2: The percentage matrix showing the results as percentage of the total number of *Staphylococcus aureus* strains resistant to each antibiotic

Percentage of strains resistant to indicated pairs of drugs*:												
F	**CT**	**TMP**	**BP**	**FU**	**CN**	**CL**	**E**	**C**	**S**	**TE**	**P**	
0	84.2	73.6	52.6	0	31.5	78.9	68.4	52.6	31.5	78.9	-	P
0	70	70	50	25	30	70	85	45	30	-	75	TE
0	66.6	100	83.3	33.3	66.6	100	100	66.6	-	100	100	S
0	72.7	90.9	63.6	27.2	36.3	100	81.8	-	36.3	81.8	90.9	C
0	76	64	40	20	20	60	-	45	24	68	52	E
0	93.7	87.5	56.2	25	31.2	-	93.7	68.7	37.5	87.5	93.7	CL
0	66.6	83.3	66.6	16.6	-	83.3	83.3	66.6	66.6	100	100	CN
0	80	100	80	-	20	80	100	60	40	100	100	FU
0	90	100	-	40	40	90	100	70	50	100	100	BP
0	83.3	-	55.5	27.7	27.7	77.7	88.8	55.5	33.3	77.7	77.7	TMP
0	-	55.5	33.3	14.8	14.8	55.5	70.3	29.6	14.8	51.8	59.9	CT
0	0	0	0	0	0	0	0	0	0	0	0	F

*= P, penicillin; BP, benzathine penicillin; C, chloramphenicol; S, streptomycin; CN, gentamicin; TE, tetracycline; E, erythromycin; CT, colistin; F, nitrofurantoin; FU, Fucidin; CL, clindamycin.

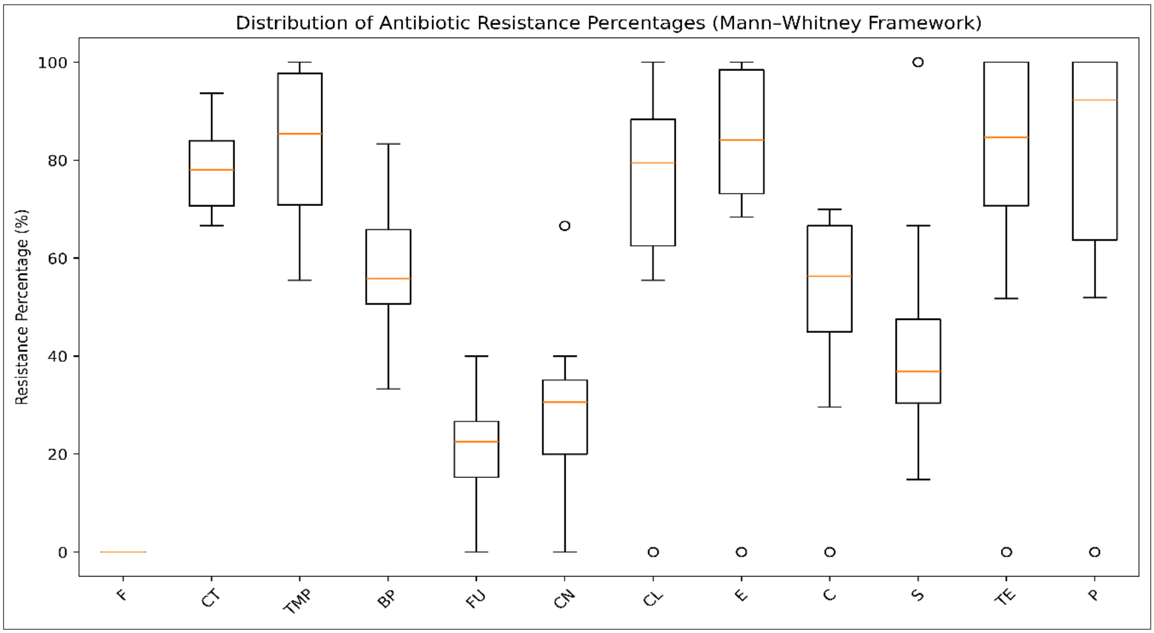

Figure 2: Distribution of Resistance Percentages across Antibiotics in *Staphylococcus aureus* (Non-parametric Analysis). P, penicillin; BP, benzathine penicillin; C, chloramphenicol; S, streptomycin; CN, gentamicin; TE, tetracycline; E, erythromycin; CT, colistin; F, nitrofurantoin; FU, Fucidin; CL, clindamycin

The present study shoed a strong association between pairs of resistance to some antibiotics used, e.g. penicillin with tetracycline, other resistances appeared to stand out as being independent (Table 3). Mann–Whitney U analysis revealed significant differences in the degree of association among antibiotics. Erythromycin, tetracycline, and penicillin demonstrated significantly higher association levels compared to gentamicin, streptomycin, and fucidin ($p < 0.05$). These findings indicate a structured co-resistance network, suggesting shared resistance mechanisms and potential genetic linkage among specific antibiotic groups.

Table 3: Degree of association between pairs of antibiotics. The tabulated values are the contribution to X^2 of the doubly resistant cell in each 2 x 2 table (40 strains of *Staphylococcus aureus*)

Degree of association between resistance to indicated pairs of drugs*:											
	CT	**F**	**BP**	**FU**	**CN**	**CL**	**E**	**C**	**S**	**TMP**	**TE**
P	3.5	21	11.1	16	15	1.5	1.7	8.1	15	1.2	0
TE	1.8	22	12.1	17	16	1.7	1.2	9	16	1.2	-
TMP	4.2	20	10.1	15	12	0.5	3.2	7.1	11.1	-	-
S	16	8.1	1.5	0.5	0.25	9	17	3.2	-	-	-
C	11.1	13	0.5	4.9	3.1	4.1	10.5	-	-	-	-
E	0.5	27	17	21	19	6.7	-	-	-	-	-
CL	4.9	19	6.1	12	11	-	-	-	-	-	-
CN	16	8.1	1.1	0.4	-	-	-	-	-	-	-
FU	18.3	7.2	4.1	-	-	-	-	-	-	-	-
BP	12.1	12.1	-	-	-	-	-	-	-	-	-
F	25	-	-	-	-	-	-	-	-	-	-
CT	-	-	-	-	-	-	-	-	-	-	-

*= P, penicillin; BP, benzathine penicillin; C, chloramphenicol; S, streptomycin; CN, gentamicin; TE, tetracycline; E, erythromycin; CT, colistin; F, nitrofurantoin; FU, Fucidin; CL, clindamycin.

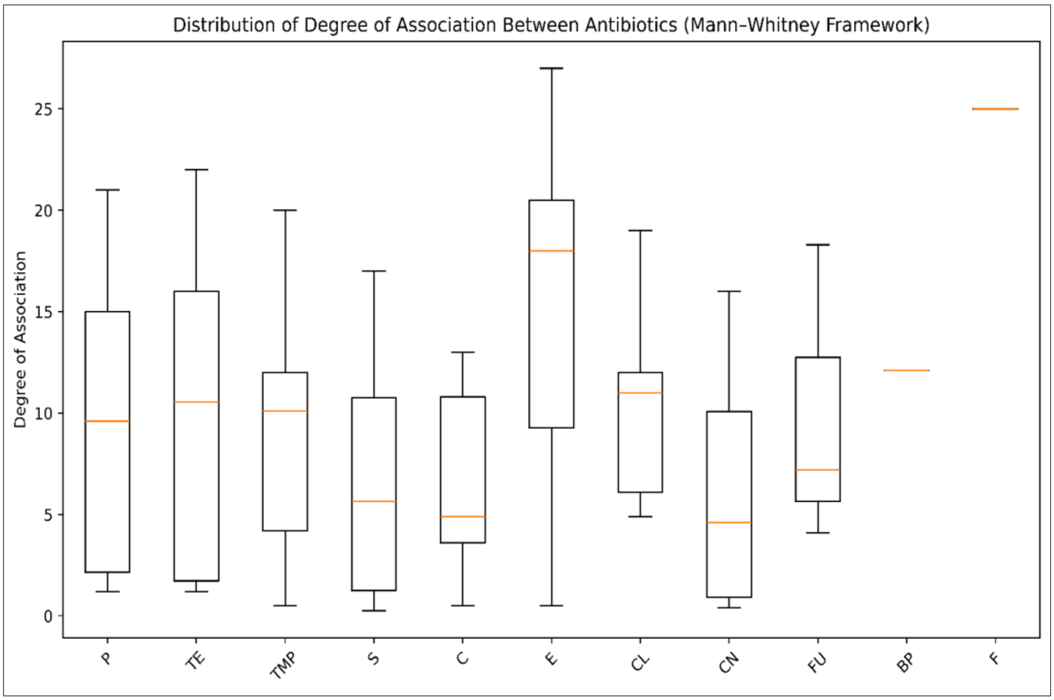

Figure 3: Distribution of Degree of Association among Antibiotic Resistance Patterns in *Staphylococcus aureus.* P, penicillin; BP, benzathine penicillin; C, chloramphenicol; S, streptomycin; CN, gentamicin; TE, tetracycline; E, erythromycin; CT, colistin; F, nitrofurantoin; FU, Fucidin; CL, clindamycin

DISCUSSION

The degree of association between the antibiotic resistance among the 40 strains of Staphylococcus aureus was studied. The present study showed a shared resistance between pairs of antibiotics tested. It was seen that there were large numbers of trains resistant to pairs of antibiotics at the same time even of different antibiotics groups, e,g. colistain sulfate and erythromycin, erythromycin and tetracycline, clindamycin and penicillin. Nitrofurantoin showed no shared resistance with any of the antibiotics used in the present study. Almost the same resistance association was found by Neil and his colleagues who found that strains of methicillin-resistant *S. aureus* isolated from Australian teaching hospitals frequently showed resistance to many other antibiotics [17]. The result of present study showed that resistance is frequently multiple and that cross-infection in the hospital plays an important role in the resistance of microorganisms to antibiotics. This is possibly due to absence of genetic markers to be combined with those responsible for resistance of other antibiotics [18]. The present study revealed that the percentage matrices were not symmetrical as determined by the considered resistance, e.g. 30% of strains resistant to tetracycline were resistant to streptomycin while 100% of the strains resistant to streptomycin were resistant to tetracycline. In general, other similar findings were found elsewhere [19-22]. Richardson and Marples found the same finding among isolates of *S. epidermidis* [23]. The common mechanism by which bacteria become resistant to antibiotics is the modification of the antibiotic's target like penicillin-binding proteins (PBPs) which leads to resistance to β-lactam drugs [24]. The mechanism of resistance reflects the amount of drug that can combine to the target is affected by changes in the number of PBPs [25]. A structural modification e.g. the development of the mecA gene in *S. aureus* will decrease or might completely prevent drug binding to the target [26, 27]. The methylase of the erythromycin ribosome (erm) gene family, will methylate 16S rRNA and changes the drug-binding site leads to resistance to macrolides, streptogramins, and lincosamines due binding prevention with them [28-30]. Resistance mediated by changes in DNA gyrase or topoisomerase IV leads to inactivation of fluoroquinolones by inhibit nucleic acid synthesis. These mutations lead to the composition change of gyrase and topoisomerase to reduce or exclude the drug's ability to link to these components [31-32].

The present study showed a strong association between pairs of resistance to some antibiotics used, e.g. penicillin with tetracycline, other resistances appeared to stand out as being independent. Fidalgo *et al*., found that there was an association between erythromycin and clindamycin resistance among isolates of *S. aureus*, the so-called macrolises-lincosamides resistance but no relationship between methicillin and macrolide-lincosamide resistance was observed [33]. Erythromycin, tetracycline, and penicillin demonstrated significantly higher association levels compared to gentamicin, streptomycin, and fucidin ($p < 0.05$). These findings indicate a structured co-resistance network, suggesting shared resistance mechanisms and potential genetic linkage among specific antibiotic groups. In contrast, Al-Ani found a weak relationship between the resistance to the same antibiotics among strains of *S. aureus* in Mosul city [1]. Moreover, the association in antibiotic resistance in the present study is partially similar in some combinations to that found by others [1-35]. The association between pairs of resistance to many antibiotics might be explained by presence of resistance to one antibiotic induces the organism to resist another antibiotic from the same group even if not exposed to it, this is known as cross-resistance. In addition, presence of multiple antibiotic resistance at the same time might be due to the presence of multiple R-determinants on the same plasmids carried by the organism [36,37]. It is natural for bacteria to develop antibiotic resistance which is encoded by the antibiotic resistance genes (ARGs) which is not more than production of billions of years of evolution. It has been found that bacteria living in the environment already possess ARGs which are responsible for resistance to newly approved antibiotics before using of these drugs [17]. Inherited structural and / or physiological properties lead to intrinsic resistance to antibiotics. These functional properties including efflux to actively eliminate antibiotics from bacterial cells which entered through porin which is the mechanism by which the antibiotics unable to pass the outer membrane and by this mechanism cannot reach the target site [17-39]. The changing susceptibility of P. aeruginosa to antibiotics as a result of exposure to disinfectants has been demonstrated, and 32% of the antibiotic-resistant strains became sensitive to all the antibiotics tested. Apart from antibiograms, other typing procedures for similarity were not performed and the stability of the antibiograms was not tested in control strains similarity cultured but unexposed to disinfectants [5-44]. The growing concerns about the development of biocide resistance and cross-resistance with antibiotics among pseudomonads have been suggested. It is clear that clinical isolates particularly of P. aeruginosa should be under continuous surveillance and possible mechanisms associated with disinfectant-resistance should be further investigated particularly among hospitals where patients who are mostly immunocompromised are resident [30-47]. However, the present findings revealed that exposure of some hospital pseudomonads to disinfectants could change the antibiotic sensitivity pattern, and this might lead to the erroneous conclusion that strains are unrelated. In these circumstances, other typing methods are required. When a laser beam, or- a photon, alters the energy level of an atom through shifting between e2 and e3, this event establishes a change in physicochemical cellular function which acts as a trigger agent for beginning a mimicry cascade system [26-28] A second photon that will be in phase, propagate in the same direction as the excited photon. This phenomenon, that is called stimulated emission, is the basic of laser light generation. It has been mentioned that in most of the biological tissues, photons are preferably scattered in forward direction [29-50].

CONCLUSIONS

The present study showed a shared resistance between pairs of antibiotics tested. It was seen that there were large numbers of trains resistant to pairs of antibiotics at the same time even of different antibiotics groups, e,g. colistain sulfate and erythromycin, erythromycin and tetracycline, clindamycin and penicillin. Nitrofurantoin showed no shared resistance with any of the antibiotics used in the present study. The Mann–Whitney U test revealed significant differences in resistance distributions among antibiotics. Penicillin, tetracycline, trimethoprim, and clindamycin demonstrated significantly higher resistance levels compared to gentamicin, streptomycin, and fucidin ($p < 0.05$). Nitrofurantoin exhibited the lowest resistance, differing significantly from all other antibiotics ($p < 0.001$). These findings indicate a non-random, structured pattern of antimicrobial resistance, likely reflecting differential usage and underlying resistance mechanisms. Erythromycin, tetracycline, and penicillin demonstrated significantly higher association levels compared to gentamicin, streptomycin, and fucidin ($p < 0.05$). These findings indicate a structured co-resistance network, suggesting shared resistance mechanisms and potential genetic linkage among specific antibiotic groups.

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Statement of Ethics: All the procedures involving human participation were conducted in strict accordance with ethical standards of Institutional Research Committee, Department of Scientific Research, Tikrit University as well as the 1964 Helsinki Declaration and its subsequent amendments or equivalent ethical norms.

Conflict of Interest Statement: The author declares that he has no conflicts of interest, financial or otherwise.

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