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SENSITIVITY OF COMMERCIAL ANTIBIOTICS TO PATHOGENIC BACTERIA

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AUTHORS' CONTRIBUTIONS

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ABSTRACT

The sensitivity of human pathogens to some antibiotics used to treat infections and infections caused by these species in humans was tested. The sensitivity of human pathogens to some antibiotics used to treat infections and infections caused by these species in humans was tested. In this study, some pathogenic bacterial isolates, some Gram-negative and Gram-positive, were used. And that from different pathological samples (urine, wound secretions and ear secretions). Obtained from the laboratory of Tishreen University Hospital at Tishreen University in Lattakia Governorate. The sensitivity of pathogenic bacterial isolates to a number of antibiotics was tested using the disk diffusion method. The sensitivity of bacteria was determined by measuring the diameter of the inhibition halos, and its resistance to antibiotics was determined based on the measurement of the diameter of the inhibition zone. The results showed that S. aureus Sensitive only to (SXT) Trimethoprime-Sulfamethoxazole, (VA) Vancomycin, (CN) Gentamycin, and Streptococcus S. faecalis showed sensitivity only to (VA) Vancomycin, (CRO) Ceftriaxone, (AK) Amikacin, (SXT) (rimethoprime-sulfamethoxazole, As for the common P. vulgaris, it was only sensitive to each of the antibiotics (VA) Vancomycin, (AK) Amikacin and (CX) Cloxacillin. And E. coli was allergic only to Trimethoprime-Sulfamethoxa (SXT), (CN)Gentamycin, (LEV) Levofloxacin (SXT) and Amikacin (AK) And if the blue pus bacillus, all strains of P. aeruginosa showed sensitivity only to each of the antibiotics, namely (AK) Amikacin, (CIP) Ciprofloxacin K. pneumoniae showed sensitivity to (VA) Vancomycin, (AK) Amikacin, (LEV) Levofloxacin, (CXm) Cefuroxime, (CRO) Ceftriaxone.

Keywords: Bacillus; antibiotics; chemical compounds; pathogenic bacteria.

1. INTRODUCTION

Antibiotics are chemical compounds, whether manufactured by living organisms or produced industrially, that are capable of eliminating germs by stopping their reproduction and growth or killing them. Antibiotics are derived from three sources: fungi and germs and an industrial or semi-synthetic substance, used either internally or topically, and its function is to either inhibit the growth of bacteria or kill them. Thus, antibiotics can be divided into Bactriostatic antibiotics that inhibit the growth of bacteria only, and Bacteriocidal antibiotics that kill germ cells [1].

Antibiotics selectively affect the vital functions of bacteria with minimal or no effect on the functions of

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the host, either by inhibiting some vital processes or metabolic reactions important for microbial cells. Antibiotics are divided into four main groups according to the mechanism of their effect [2]:

1- Antibiotics that inhibit cell wall synthesis: such as penicillines and cephalosporins.
2- Antibiotics that inhibit DNA synthesis: such as fluoroquinolones.
3- antibiotics that inhibit protein synthesis: such as aminoglycosides.
4- Antibiotics that inhibit bacterial metabolic reactions: such as sulfamides and trimethoprime, or their combination [3,4].

The pathogens resistant to antibiotics are a public health problem spread around the world, as the lack of development of new antibacterial compounds in conjunction with their misuse has led to the emergence of microorganisms resistant to many antibiotics, to the extent that it has become a challenge facing the treatment of diseases, and a high increase in the cost of treatment As a result, many common infections become untreatable and even fatal [5,6].

Also, these chemical drugs are almost devoid of elements that have negative side effects on human health, when used as a treatment for bacterial infections and often lead to secondary complications in the long term, and the appropriate way to obtain new effective drugs against multi-resistant bacteria, and to eliminate common problems of side effects. Antibiotics are the use of those natural plant compounds and marine biological compounds that have anti-bacterial properties and are free from toxic chemicals. They have the best and effective therapeutic effect and fewer side effects, as it was found that more than 60% of the antibacterial agents used in the treatment are of natural origin [7].

Most studies confirm that the misuse of antibiotics, including their increased use in human and veterinary medicine and agriculture, represents the most important risk factor in the emergence and spread of bacterial resistance, but the main reason for the spread of antibiotic-resistant bacteria is the continued effect on the same cellular sites (because all antibiotics The biological used affects the germ cell by one of the four known mechanisms), which leads to mutations of the germ defense genes [8,3].

2. THE IMPORTANCE AND OBJECTIVES OF THE RESEARCH

2.1 Research Materials and Methods

2.1.1 Study locations

In this study, some pathogenic bacterial isolates, some Gram-negative and Gram-positive, from different pathological samples (urine, wound and ear secretions) were used. Obtained from the laboratory of Tishreen University Hospital at Tishreen University in Lattakia Governorate (Table 1).

After confirming its purity, the pathogenic isolates were kept in glycerol 25% at a temperature of -20°C until conducting the laboratory study and verifying their identity through direct examination, microscopic examination and conducting a number of biochemical tests, depending on the scientific references used to diagnose bacteria [9] in addition to using the API 20E profiling syntax (Bio Merieux, French.).

The sensitivity of pathogenic bacterial isolates to a number of antibiotics was tested using the disk diffusion method. The bacterial suspension was prepared for each bacterial type of pathogenic bacteria at a density of 0.5 according to McFarland, which is equivalent to 810 x 1.5 cells/ml in a physiological solution. Then spread 100 µl of the bacterial suspension on solid Muller-Hinton (MHA) medium.

For each bacterial species alone, the plates were kept at room temperature for a quarter of an hour, then commercial antibiotic tablets were distributed on the plates, and then incubated at 37 °C for 24 hours. After that, the amount of bacterial sensitivity was determined by measuring the diameter of the inhibition halos, and its resistance to antibiotics was determined based on measuring the diameter of the inhibition zone (in millimeters) according to what was mentioned in [10].

Table 1. Pathogenic bacterial isolates tested in this research and their sources

Sample source	Pathogenic bacteria
Ear secretions	Staphylococcus aureus
Wound exudate	Streptococcus faecalis
Wound exudate	Proteus vulgaris
Pee	Escherichia coli
Wound exudate	Pseudomonas aeruginosa
Pee	Klebsiella pneumonia

Some antibiotics from the penicillin family have been used, such as Oxacillin, Cloxacillin, (Ampicillin-Sulbactam, cephalosporins of the first generation (Cephradine) and the second generation Cefaclor), (Cefuroxime and the third generation Cefixime), (Ceftriaxone, and aminoglycosides such as Amikacinmethethe) and (Gentamyprime-Gentamycin) The sulfamides, Levofloxacin and Ciprofloxacin are from the fluoroquinolones family, These antibiotics were adopted in this study because they are used commonly and frequently, and they act according to the four known mechanisms of antibiotic effect [11] Table (2) shows the diameters of the rings of bacterial growth inhibition resulting from sensitivity to some of the antibiotics used in the study, estimated in millimeters on the medium of Muller Hinton agar [12].

3. RESULTS

The sensitivity of human pathogenic microbes to some antibiotics used to treat infections and infections caused by these species in humans was tested, in order to compare the effectiveness of the extracts with the effectiveness of the antibiotics. Table (3) shows the results of sensitivity and resistance of pathogenic microbes to some antibiotics.

Sensitive only to (SXT) Trimethoprime-Sulfamethoxazole, (VA) Vancomycin, (CN) Gentamycin, moderately sensitive to (CEC) Cefaclor, (CEC) Cefaclor, (LEV) Levofloxacin and insensitive to (OX) Oxacillin, (SAM) Ampicillin-Sulbactamin Table (3).

Sensitive only to (SXT) Trimethoprime-Sulfamethoxazole, (VA) Vancomycin, (CN) Gentamycin, moderately sensitive to (CEC) Cefaclor, (CEC) Cefaclor, (LEV) Levofloxacin and insensitive to (OX) Oxacillin, (SAM) Ampicillin- Sulbactamine Tablet (3).

It showed sensitivity only to two antibiotics: (VA) Vancomycin, (AK) Amikacin, (CX) Cloxacillin and moderately sensitive (CIP) Ciprofloxacin, (SAM) Ampicillin-Sulbacta and insensitive to (SXT) Trimethoprime-Sulfamethoxazole, (CN) Gentamycin, Table (3).

And ***Escherichia coli*** showed sensitivity only to two antibiotics, namely (SXT) Trimethoprime-Sulfamethoxa, (CN) Gentamycin, (LEV) Levofloxacin and (AK) Amikacin.

Moderate Sensitivity (CIP) Ciprofloxacin, (CEC) Cefaclor, (SAM) Ampicillin-Sulbactam Insensitive to (VA) Vancomycin, (CRO) Ceftriaxone, (CFM) Cefixime, (CEm) Cefuroxime, (CE) Cephradine, (Cx) Cloxacillin, (OX) Oxacillin Table (3).

And the blue pus bacilli were all strains of *Pseudomonas aeruginosa* Only showed sensitivity to two antibiotics, namely (AK) Amikacin and (CIP) Ciprofloxacin. Moderately sensitive (SXT) Trimethoprime-Sulfamethoxa, (CN) Gentamycin, (LEV) Levofloxacin, (CRO) Ceftriaxone, (CE) Cephradine, (Cx) Cloxacillin and insensitive to (VA) Vancomycin, (CXm) Cefixime, (CEC) Cefaclor, (SAM) Ampicillin-Sulbactam, (OX) Oxacillin, (CFM) Cefixime Table (3).

Table 2. The diameters of the bacterial growth inhibiting rings according to the 2005 NCCLS standards resulting from sensitivity to antibiotics, estimated in mm on Muller Hinton agar medium

Sensitive (S)≥	Average sensitivity I	Resistant (R)≤	Disc focus	code	Biofouling
13	11-12	10	1µg	OX	Oxacillin
13	11-12	10	5µg	Cx	Cloxacillin
15	12-14	11	20µg	SAM	Ampicillin-Sulbactam
18	15-17	14	30µg	CE	Cephradine
18	15-17	14	30µg	CEC	Cefaclor
18	15-17	14	30µg		Cefuroxime
19	16-18	15	5µg	CFM	Cefixime
21	14-20	13	30µg	CRO	Ceftriaxone
21	16-20	15	5µg	CIP	Ciprofloxacin
17	14-16	13	5µg	LEV	Levofloxacin
17	15-16	14	30µg	AK	Amikacin
15	13-14	12	10µg	CN	Gentamycin
11	-	11	30µg	VA	Vancomycin
16	11-15	10	25µg	SXT	Trimethoprime-Sulfamethoxazole

Resistor R: Resistant, Sensitive I: Intermediate, Sensitive S: Sensitive

Table 3. Sensitivity and resistance of pathogenic bacteria to some antibiotics

P. aeruginosa	***K. pneumoniae***	***E. coli***	***P. vulgaris***	***Strep. Faecalis***	***Staph. Aureus***	
0	11	0	7	19	0	AX
R	I	R	R	S	R	
0	9	0	0	10	0	Cx
R	R	R	R	R	R	
0	0	0	13	16	0	SAM
R	R	R	I	S	R	
0	12	0	0	24	0	CE
R	R	R	R	S	R	
0	9	0	0	22	12	CEC
R	R	R	R	S	R	
10	0	9	0	27	0	CXM
R	R	R	R	S	R	
12	0	0	9	18	0	CFM
R	R	R	R	S	R	
10	11	0	10	22	9	CRO
R	R	R	R	S	R	
23	8	18	12	12	11	CIP
S	R	I	R	R	R	
19	18	24	10	10	15	LEV
S	S	S	R	R	I	
18	18	17	10	14	9	AK
S	S	S	R	R	R	
14	7	8	10	13	0	CN
I	R	R	R	I	R	
9	0	10	0	26	17	VA
R	R	R	R	S	S	
13	8	0	0	16	22	SXT
I	R	R	R	S	S	

Klebsiella pneumoniae showed Allergic to (VA) Vancomycin, (AK) Amikacin, (LEV) Levofloxacin, (CXm) Cefuroxime, (CRO) Ceftriaxone, Moderate Sensitive (CEC) Cefaclor, (Cx) Cloxacillin, (SXT) Trimethoprime-Sulfamethoxazole, (CN) Table (3): Gentamycin, (CIP) Ciprofloxacin (CFM) Cefixime, (CE) Cephradine, (SAM) ampicillin-Sulbactam.

4. DISCUSSION AND CONCLUSION

The results showed sensitivity to sulfonamides antibacterials, which include Trimethoprim. As the antimacrolides are broad-spectrum antibiotics, and they are considered to be deadly antigens for many Gram-negative and Gram-positive bacteria, and they work by inhibiting the synthesis of proteins by binding to the subunits of the ribosome and thus preventing the process of building the peptide chain [13].

Aminoglycosides have the ability to kill bacterial growth through their ability to penetrate the cell wall and membrane and bind to the ribosome, causing disruption in its function and then inhibiting the process of protein synthesis, which leads to bacterial cell death [14].

It is worth noting that the antibiotic Vancomycin is from a group of antibiotics belonging to aminoglycoside antibiotics that work to impede the construction of the cell well by binding it to the peptide chain as well [15].

Amikacin is a bacteriocidal antagonist that binds to the 30S ribosomal subunit, blocks translation initiation, does not read mRNA, and affects facultative and aerobic bacteria, as the group of aminoglycosides and Lincosamides becomes stronger when β-lactam-containing antagonists are added. The latter hinders the building of the cell wall of bacteria, facilitates the entry of aminoglycoside into the cell, and prevents resistance [16].

Studies in the last ten years have shown that the resistance of some pathogenic bacteria has increased to a very large extent to broad-spectrum beta-lactam antibiotics, as well as the third generation of

cephalosporins in addition to fluoroquinolones and penicillins. Multi-resistant bacteria [17].

Most studies confirm that the misuse of antibiotics, including their increased use in the field of human and veterinary medicine and agriculture, is a risk factor because it increases the resistance of pathogenic bacteria to the antibiotics used. The spread of antibiotic-resistant bacteria is mainly due to the fact that they continue to affect the same cellular sites (because all the antibiotics used affect the bacterial cell by one of the four known mechanisms: inhibition of cell wall synthesis and metabolic processes, inhibition of bacterial protein synthesis, and finally inhibition of nucleic acid synthesis) , which leads to mutations of bacterial genes [18].

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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