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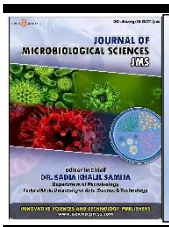
Research Article



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Impact Of Steroidal and Non- Steroidal Drugs on The Microflora of Gastrointestinal Tract

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Abstract

Antimicrobial agents are supposed to be the most important means for treating bacterial infections. The use of steroidal and non-steroidal drugs is leading the disturbance of the normal microflora of gastrointestinal tract. This interference may result in abnormality of natural defence mechanism which is due to the colonic microbial ecosystem, making the host resistant to infections caused by commensal microorganisms and nosocomial pathogens. The agar well diffusion assay method was performed to observe the zones of inhibition caused by antimicrobial agents. Up to 25 antimicrobial drugs were used and out of 25 drugs, five antimicrobial drugs gave huge zones of inhibition.

Keywords: antimicrobial agents, pathogens, zone of inhibition

Introduction

The normal microbial flora of human has its own dynamic ecosystem. It is very valuable in metabolic operations, and the colonization provides resistance and prevention from many infections (Sullivan et al., 2001). A variety of enzymes in this ecosystem are implicated in reduction, deconjugation, and other biochemical activities which result in altering the structure and activation sites of different molecules (Rowland, I. R., 1995). In vitro, there are many experiments that change in population composition of the microflora of human intestinal tract with reduced colonization resistance (Wagner et al., 2008). Furthermore, the microbial ecosystem may have an unidentified influence on the immune system (van der Waaij, D., & Nord, C. E., 2000) and that of intestine appears to be a stimulator of the host immune system which respond quickly to pathogen challenges (Berg, R. D., 1996). Although the colonic microbial community is very stable but it can be disturbed by intake of antimicrobial drugs which are used for the treatment of different bacterial infections (Peck, J. J., Fuchs, P. C., & Gustafson, M. E., 1984). These bacterias are exposed to antibiotics whether the drug is administered orally or intravenously (Edlund, C., & Nord, C. E., 1999) and this contact may occur due to the partial absorption of antibiotics that are administered orally . Antibiotics which are given orally are absorbed in the upper side of the small intestine (Nord, C. E., & Edlund, C. 1990) have different effect as compared to those that are not completely absorbed. Exposure of the microbial ecosystem to antibiotics may result changes in different components as a result causes the suppression of some microorganisms (Edlund, C., & Nord, C. E., 1999). Beta- lactam antibiotics were used for this study. Beta-lactam antibiotics are one of the most repeatedly prescribed drug (Thakuria, B., & Lahon, K. (2013). These drugs have 3-carbon and 1-nitrogen ring (beta-lactam ring) which is highly reactive. This class includes:

- Penicillins: These antibiotics contain a nucleus of 6-animopenicillanic acid ring and other ringside chains. The group includes natural penicillins, beta-lactamase-resistant agents, aminopenicillins, carboxypenicillins, and ureidopenicillins.
- Cephalosporins: They contain a 7-aminocephalosporanic acid nucleus and side-chain containing 3, 6-dihydro-2 H-1, 3- thiazane rings. Cephalosporins are further divided into five generations.
- Carbapenems: A carbapenem coupled to a beta-lactam ring that confers protection against most beta-lactamases, although resistance to these compounds is a significant issue and occurs mainly among gram-negative pathogens (e.g., *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii).*

- Monobactams: The beta-lactam ring stands alone and not fused to another ring.
- Beta-lactamase inhibitors: They work primarily by inactivating serine beta-lactamases, which are enzymes that hydrolyze and inactivate the beta-lactam ring. These agents include the first-generation beta-lactamase inhibitors.

Resistance to beta-lactams is an alarming a public health challenge as well. Although bacterial resistance to beta-lactams mostly expresses through the production of beta-lactamases, other mechanisms are involved. Following are the mechanisms of resistance (Ibrahim et al., 2019)

- Inactivation by the production of beta-lactamases
- Decreased penetration to the target site.
- Alteration of target site PBPs
- Efflux from the periplasmic space through specific pumping mechanisms

Materials and Methods

The antimicrobial activity was carried out against two organisms which are *Streptococcus aureus and Escherichia coli.* Agar well diffusion method was used to determine the antibacterial activity on nutrient agar plates. A 50 µL of respective microbial inoculum was taken using a micropipette in order to provide an even lawn of cells, and loaded onto the agar plates evenly. The agar plates were inoculated with the respective microorganisms by even swab over the entire surface of the plate three times rotating the Petri plates at 60° approximately after each applications. Finally, it was swabbed all around the periphery of the agar surface. Four wells of 7 mm size and 4 mm depth were made at an equal distance and 70 µL volume of each antibacterial drug was dispensed into the wells with the help of micropipettes. The plates were then incubated at 37°C for 24 hours in an aerobic environment. The Petri plates were observed for zone of inhibition, which were measured using a scale in millimeters. The tests were repeated three times to minimize errors (Seol et al., 2006; Nalawade et al., 2016)

Results

S.No	Name of antibiotic	Potency (mg)	Sensitive(mm)	Observed(mm)	Resistant(mm)
1	amoxicillin	30	≥18	17	≤13
2	Cloxacillin	5	≥25	22	≤21
3	Cephalothin	30	≥18	10	≤14
4	cephalin	25	≥18	21	≤12
5	cefuroxime	30	≥23	20	≤14
6	Cefixime	5	≥19	22	≤15
7	kanamycin	30	≥18	13	≤13
8	streptomycin	10	≥15	15	≤11
9	neomycin	30	≥17	14	≤12
10	vancomycin	30	≥12	10	≤9
11	erythromycin	15	≥23	13	≤13
12	azithromycin	15	≥18	13	≤13
13	ciprofloxacin	15	≥21	28	≤15
14	levofloxacin	5	≥17	11	≤13
15	tetracycline	30	≥15	14	≤11
16	doxycycline	30	≥14	16	≤10
17	cotrimoxazole	25	≥16	16	≤10
18	chloramphenicol	30	≥18	12	≤12
19	penicillin	10	≥28	28	≤19
20	Gentamycin	10	≥15	18	≤12
21	moxifloxacin	5	≥24	22	≤20
22	nitrofurantoin	30	≥17	15	≤14
23	rifampin	5	≥20	20	≤16
24	minocycline	30	≥19	22	≤14
25	clindamycin	2	≥21	20	≤14

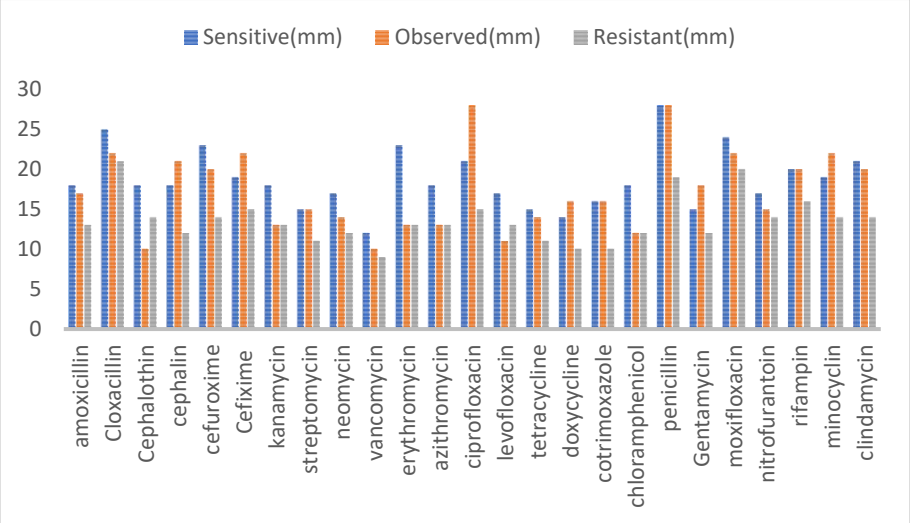

Figure 1: shows the comparison among resistant. Sensitive and observed zone of inhibition

Up to 25 different antimicrobial drugs were used in this research. Each antibiotic has different mode of action. The amoxicillin 30mg gave 17mm zone of inhibition, cloxacillin 5mg, cephalothin 30mg, kanamycin 30mg, streptomycin 10mg, neomycin 30mg, vancomycin 30mg, azithromycin 15mg, ciprofloxacin 15mg, levofloxacin 5mg, tetracycline 30mg, cotrimoxazole 25mg chloramphenicol 30mg, penicillin 10mg, cefuroxime 30mg, The moxifloxacin 5mg, nitrofurantoin 30mg, rifampin 5mg and clindamycin 2mg gave intermediate zones of inhibition i.e., they have no harmful effect on gut microbial flora and are considered as safe drugs. Whereas Cephalin 25mg gave a quite huge zone of inhibition of 22mm which indicates that it may cause disturbance in the colon microflora. Cefixime 5mg, gentamycin 10mg and minocycline 30mg gave large zones of inhibition so these cannot be considered as a safe drugs. Meanwhile doxycycline 30mg gave a little bit greater zone of inhibition.

Discussion

This study describes that *S. aureus* and *E. coli* are sensitive to cephalin 25mg, cefixime 5mg, gentamycin 10mg and minocycline 30mg, doxycycline 30mg. While shows resistance against other 20 antibiotics. Penicillin-binding protein 2a of *Staphylococcus aureus* is refractory to inhibition by available beta-lactam antibiotics, resulting in resistance to these antibiotics. The results are interpreted on the basis of zones of inhibition. The zone of inhibition is a parameter measured in the Calibrated Sensitivity test, to determine if microorganisms are resistant or susceptible to antibiotics at required concentrations. The zone of inhibition actually refers to an area on an agar plate around the well of an antibiotic in which bacteria should not grow if they are susceptible to the antibiotic. This is because the antibiotic interferes with one or more components that the cells need to survive. So while a lawn of bacteria grow elsewhere on the plate, there is a circle around that disc where the concentration of antibiotics is high enough that these organisms can't grow (Hudzicki, J., 2009).

Conclusion

There is a complex bidirectional interaction between antimicrobial agents and the microflora of intestinal tract. Physicians must be aware that antitherapeutic agents can also change the microflora and can result in impaired health outcome. The antibiotics cause the alteration in the microbes, harm the host body and cause different enteric infections which are not supposed to be good for the human health.

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