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The Frequency and Contributing Factors of Fluoroquinolone Resistance among Significant Bacterial Pathogens

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Abstract

Pathogens from the Enterobacteriaceae family account for 84.3% of urinary tract infections (UTIs). Within the bacterial family Enterobacteriaceae, encompassing Proteus, Klebsiella, Enterobacter, and Citrobacter species, alongside Pseudomonas, Enterococcus, Streptococci, Staphylococci, and Candida albicans, E. coli is the predominant pathogen responsible for urinary tract infections (UTIs) more frequently than any of the aforementioned organisms. This has caused a lot of problems in clinical care, infection control, and antimicrobial management. The current study, called " The Frequency and Contributing Factors of Fluoroquinolone Resistance Among Significant Bacterial Pathogens," tried to find out where common clinical bacterial pathogens are found, how well they are at fighting off antibiotics, how common multidrug-resistant organisms are, and how resistance patterns differ between different types of bacteria and clinical specimens. The prevalence of multidrug resistance was considerable, with A. baumannii demonstrating the most prominent MDR characteristics. Notable resistance phenotypes, including as MRSA, ESBL production, carbapenem resistance, and VRE, were also recognized. Statistical study indicated that multidrug-resistant (MDR) status showed significant variability across bacterial species, whereas the relationship between specimen type and MDR status was very weak in the model assessment.

Keywords: Antimicrobial resistance (AMR), Antimicrobial susceptibility testing (AST), Clinical bacterial isolates, ESBL-producing bacteria

Introduction

UTIs are often brought on by the usual symbiosis that exists between the surrounding sites and the distal urethra. Climbing is becoming more popular as a means of infection. Pathogens that are found in the urinary tract are a component of the common faecal flora. These bacteria first colonize the perianal region, and then they spread to the female vaginal opening, which serves as a reservoir for a number of urinary tract infections after their transmission. According to Franz and Horl (1999), the colonization spreads in the periurethral area, which includes the bladder and the urethra, are significantly dependent on engaging in sexual activities. As a result of UPEC's capacity to bind the host tissues in the urinary system, bacteria are able to withstand the flow of enormous quantities of urine, and UPECs are able to stick to urothelial cells. This makes it feasible for UPEC to colonize the urinary tract. UPEC preferentially colonizes the bladder and causes cystitis after it has entered the urinary system. This increases the invasion of the condition.

Routes of Infection

Microorganisms might enter the urinary system by ascending, hematogenous, or lympatic pathways, depending on the method of transport.

- **Ascending route of infection**

In the majority of instances, pathogens infiltrate the urinary tract via the ascending route, commencing in the urethra. The upward progression of urinary system infections is a complex phenomenon linked to bacterial adherence, pathogenicity, motility characteristics, along with the host's anatomical structure, genetic predispositions, and humoral elements. The ascending route accounts for more than 95 percent of all instances of urinary tract infections (UTI). Concerning E. coli and other Enterobacteriaceae, this is a quite common phenomenon.

- **Hematogenous route of infection**

The only uropathogens that may induce endogenous urinary tract infections are a select handful that are quite uncommon. These uropathogens include yeast (often Candida albicans), Mycobacterium tuberculosis, Salmonella spp., and Staphylococcus aureus, all of which are responsible for primary infections in other parts of the body. Candia albicans is a common source of acute clinical urinary tract infections; however, it is also a rare cause of progressive infection that occurs after antibiotic treatment or when catheterization is performed. Less than five percent of urinary tract infections (UTIs) are caused by hematogenous spread, which often happens as a consequence of bacteremia.

Lymphatic route of infection

During the process of pathogenesis, the status of the Ur pathogen's lymphatic dissemination to the urinary system is uncertain.

- **Clinical presentation**

Urinary tract infections have generally been regarded as acute illnesses and are often able to resolve on their own. This idea, however, has been called into question by more recent research suggesting that acute bladder infections are the result of a complicated chain of interactions between the host and the pathogens that ultimately result in the entrance and survival of germs. the germs, which ultimately determines the progression of the illness over time. In most cases, a urinary tract infection (UTI) may also be categorized as asymptomatic bacteriuria, cystitis, or acute pyelonephritis. The majority of cases of cystitis are connected with bladder invasion.

Pyuria, fever, a burning feeling while peeing, stomach discomfort in the lower region, itching, ulcers and blisters developing in the genital area, and pain in the genitals and suprapubic bone are some of the symptoms of urinary tract infections (UTIs). The intensity of these symptoms often fluctuates based on the individual's age and the orientation of the urinary tracts.

- **Treatment**

The treatment for a urinary tract infection (UTI) might vary based on the patient's age, sex, associated conditions, the causative pathogen, and whether the illness affects the upper or lower urinary tract. The management of urinary tract infections (UTIs) often entails the administration of a broad-spectrum antibiotic rather than a narrow-spectrum one, due to apprehensions over infections from antibiotic-resistant organisms. The antibacterial agent including a combination of sulfamethoxazole and trimethoprim, along with beta-lactams, fluoroquinolones, nitrofurantoin, and fosfomycin, is often used to address uncomplicated urinary tract infections (UTIs). The acceptability of these medications, together their efficacy against the suspected uropathogens and their favorable pharmacokinetic characteristics, are the main factors contributing to their extensive use.

- **Beta lactams**

Historically, the predominant approach to treating urinary tract infections (UTIs) was beta-lactam antimicrobials, including aminopenicillins (like ampicillin and amoxicillin) and cephalosporins (such as cephalexin). Despite their ability to generate elevated urine concentrations, first-generation cephalosporins and aminopenicillins are no longer commonly prescribed as first-line treatments for urinary tract infections (UTIs) because of their resistance to other antibiotics and higher recurrence rates compared to alternative medications.

Trimethoprim-sulfamethoxazole

Due to the efficacy of trimethoprim-sulfamethoxazole against prevalent uropathogens and its tolerability, it has long been considered the standard treatment for severe and recurrent urinary tract infections. This is attributable to the prolonged usage of trimethoprim-sulfamethoxazole. This collaborative pairing of trimethoprim and sulfamethoxazole operates at the two distinct stages of the bacterial folate metabolism, which are disrupted by the inhibition of DNA synthesis. Currently, the use of trimethoprim-sulfamethoxazole is limited because of prevalent microbial resistance. This drug is advised for use in areas with a documented low resistance rate (below 20%) and subsequent to culture. Notwithstanding this, trimethoprim sulfamethoxazole remains a potent first-line antimicrobial therapy in regions with little resistance.

- **Nitrofurans**

Nitrofurans are a family of chemicals categorized by the existence of one or more nitro groups attached to a nitroaromatic or nitro-heterocyclic framework. Furazolidone, nitrofurazone, and nitrofurantoin are all compounds that are part of this category. All has the capability to suppress bacterial proliferation and are used in medical environments to address various illnesses (Sandegren *et al*. 2008). Nitrofurantoin is the first antibacterial agent that is both efficacious and secure for the treatment of urinary tract infections (UTIs), however its range of effectiveness is restricted.

- **Fluoroquinolones**

Nalidixic acid and fluorinated quinolones are included under the quinolone category, which is derived from synthetic antibacterial agent classes. The therapy of an ailment caused by gram-negative bacilli employs fluoroquinolones, potent antibiotics that are chemically related to nalidixic acid. Fluoroquinolones are used extensively in clinical environments because to their oral bioavailability, overall tolerability, and broad spectrum of activity. The use of fluoroquinolone in medical environments has been associated with a rise in cases of quinolone-resistant bacteria.

- **Prevention of UTI**

Patients who have to be carefully considered for preventative measures include women who have had recurrent urinary tract infections (UTIs), children who have any additional deviations from the normal pattern of the urinary system or who have experienced recurrent UTIs, patients who have spinal cord injuries or neurogenic bladders, and patients who have recently undergone a kidney transplant.

Antibiotic resistance

There exists a perpetual cycle in nature in which organisms develop resilience to fluctuating environmental conditions. This method is referred to as an infinite process. According to Manikandan *et al*. (2010), this may stem from pre-existing variables inside the organisms or result from acquired factors.

The human gastrointestinal tract serves as a crucial reservoir of antimicrobial resistance characteristics. These attributes facilitate the preservation and dissemination of resistance throughout the ecosystem. The enteric microbes present in fecal matter are generally regarded as rather harmless, with E. coli identified as the primary vector of antibiotic resistance.

Following its first identification in E. coli in 1940, the resistance of antimicrobial organisms is now acknowledged as a growing global issue. The incidence of antibiotic resistance in urinary tract infections (UTIs) is increasing, and this pattern is evolving based on geographical and regional factors.

Antibiotic Resistance in Bacteria

There are hundreds of thousands of fatalities that occur annually as a direct result of widespread antibiotic resistance among microorganisms. The most significant issue is the ever-increasing number of germs that are resistant to antibiotics that are frequently used, even medications that are designated as a last option (vancomycin). The alarming increase in an issue that has an impact on public health on a worldwide scale and calls for international cooperation is confirmed by the rapidity with which resistance genes may spread throughout the globe (Supplementary Table S1). In 2014, the World Health Organization (WHO) called this a major global health problem because of the big increase in the number of types that are resistant to multiple drugs that was seen around the world. There have been a lot of laws passed to limit or get rid of the use of antibiotics. These laws include actions to stop using antibiotics as animal feed additives and as antibacterial agents in prevention and treatment, which was the main point of EU Regulation 2019/6.

Improving research in areas like animal genetic enhancement is important for finding markers that are linked to higher innate resistance to pathogens, looking into new antimicrobial substances, and figuring out what role bacteria play in the spread of antibiotic resistance in the microbiomes of humans and animals. This is one way that people are trying to lower the number of germs that are resistant to medicines. Right now, the main ways to fight drug resistance are to use bacteriophages or their enzymes and to make next-generation medicines. Introducing new ways to feed animals that include prebiotics, probiotics, bacterial byproducts, and phytobiotics is an important thing to think about. Scientists are also very interested in proteins and peptides that can kill bacteria.

Mechanisms of Acquisition of Drug Resistance Among Bacteria

Indigenous resistance is the most basic type of resistance. It means that something is naturally not vulnerable. There is something about a species, breed, or whole group of bacteria that is always there. Due to its "innate" resistance to certain classes of antibiotics, a certain type of bacteria can't be hurt by antibiotics. It might have something to do with the antibiotic not having a receptor, having less affinity, the cell wall not letting the antibiotic through, or enzymes being made.

Changes in a bacteria's susceptibility can happen in two ways: first, they can become more or less susceptible. Primary resistance is a type of resistance that happens naturally and can show up even if the person hasn't been exposed to any medications. The code for this type of resistance is stored on chromosomes, but it is not passed on to other types of bacteria. It is very rare for bacteria to become changed, but when a drug is present, mutant bacteria are better than the rest of the population. In this way, they are able to stay alive and spread to groups that are weak against the drug. Either they can be passed on to other microorganisms or they can move inside the same person and take up new homes. Bacteria have developed many ways to fight off antibiotic drugs over the course of their past. These defenses were made so that they can avoid being hurt by antibiotics and other antibacterial drugs. The acquisition of resistance genes by bacteria can make them partially or completely resistant to a certain antibiotic. Building up other control systems is how this resistance is achieved.

Mechanisms of Transfer of Resistance Based on Examples of Various Species of Bacteria *Campylobacter* spp.

Campylobacter, particularly C. jejuni, is well recognized as one of the most common pathogens responsible for food-related gastroenteritis in humans. Research indicates that poultry serves as the predominant reservoir for these pathogens, with the main route of Campylobacter transmission to humans being via the ingestion of tainted food, especially chicken meat. Infections in humans caused by C. jejuni and, to a lesser extent, C. coli are also attributed to ruminants, especially cattle and pigs. Ruminants account for the preponderance of these illnesses.

The overwhelming majority of campylobacteriosis cases are often self-resolving and do not need any antimicrobial treatment. Nevertheless, medical interventions are necessary for very young or old persons, pregnant women, or in cases where conditions such as Guillain-Barré syndrome arise. In circumstances such as these, macrolides, including erythromycin, are predominantly the preferred antimicrobials. Nonetheless, fluoroquinolones like ciprofloxacin or tetracyclines provide feasible options.

Resistance to Macrolides

The occurrence of mutations in the genes that encode macrolide resistance in Campylobacter is much less

common than that of mutations in the genes that encode fluoroquinolone resistance in the same organism. Moreover, the establishment of high-level resistance may include many phases of mutation. Consequently, macrolide-resistant Campylobacter mutants tend to emerge more slowly under selective antibiotic pressure than when fluoroquinolones are included. Therefore, they need long-term exposure to macrolide antibacterial treatments. Campylobacter resistance to this family of antibiotics is usually the consequence of a change in the binding site of the ribosome target. This alteration is usually due to a mutation of 23S rRNA at position 2074 (A2074C, A2074G or A2074T), 2075 (A2075G or A2075C) or both of the adenine residues in all three copies of this gene (rrnB operon). However, the change at A2075G in domain V of 23S rRNA is the major link between high-level macrolide resistance and this modification. These mechanisms of resistance to erythromycin have also been found to confer cross-resistance to other macrolides and related drugs in the lincosamide and streptogramin families.

Resistance to Tetracyclines

The tet (O) gene, responsible for encoding the ribosomal protective protein tet (O), mostly imparts tetracycline resistance to Campylobacter via the pTet plasmid. Alongside the tet (O) gene expressed by the plasmid, the CmeABC efflux pumps contribute to the bacteria's elevated tetracycline resistance. A self-conveying plasmid, measuring between 45 and 58 kb, harbors the tet (O) gene. Reports indicate that the chromosomal region is present even in tetracycline-resistant C. jejuni strains lacking the tet (O)-positive plasmid. It has been hypothesized that mobile genetic entities other than transmissible plasmids might contribute to the acquisition and spread of tet (O) tetracycline resistance genes in several bacterial species. This is corroborated by the finding that the tet (O)-bearing plasmid in Campylobacter had an insertion element IS607, similar to the IS607 present in the chromosome of Helicobacter pylori. Additional data from G-C sequence examination suggests that horizontal gene transfer from Streptomyces, Streptococcus, or Enterococcus species is the probable origin of this gene's incorporation into Campylobacter. Conjugation could have facilitated the spread of tetracycline resistance genes across Campylobacter and other bacterial species owing to the widespread presence of the conjugative tet (O)-bearing plasmid in this microorganism.

***Enterococcus* spp.**

The natural microbiota that resides in the intestine of both humans and animals is composed of bacteria from the Enterococcus genus.

In humans, Enterococcus species are often connected to intra-abdominal infections (IAI), which are the second most common type of infection. It has been found that people with endocarditis, bloodstream infections, wound and surgery site infections, urinary tract infections, and bloodstream infections all have bacteria that belong to the Enterococcus group. This bacterium is also linked to dental diseases that don't go away and recurrent periodontitis. Most of the time, a single antibiotic that kills enterococci is enough to treat simple wound infections, infections of the urinary tract, and most infections that happen inside the abdomen. Enterococcus species diseases are very dangerous to both people and animals' health because they are hard to treat. These bacteria possess the ability to transmit antimicrobial resistance genes more readily than other strains, resulting in their frequent resistance to several antimicrobials.

Resistance to β-Lactam Antibiotics

Due to their inadequate interaction with penicillin-binding proteins PBP5 in E. faecium and PBP4 in E. faecalis, enterococci have an intrinsic resistance to β-lactam antibiotics. Penicillin has the most efficacy against enterococci, whereas carbapenems have somewhat reduced efficacy, and cephalosporins show the least efficacy. This resistance fluctuates depending on the specific kind of β-lactams used. An alternative resistance mechanism associated with penicillin-binding proteins is sometimes seen in bacteria that develop a markedly heightened resistance to β-lactam antibiotics relative to wild-type strains.

Resistance to Aminoglycosides

Bacterial resistance to aminoglycoside antibiotics can develop for a variety of reasons, including changes in drug permeability, inactivation of antibiotics by enzymes, changes in 16S rRNA due to enzymatic alteration, or antibiotic modification by enzymes. Monotherapy with aminoglycosides is not an option because enterococci naturally develop resistance to low doses of these antibiotics via a process that involves decreased permeability of bacterial cell membranes for antibiotic molecules. However, if the bacteria show sensitivity to penicillin or glycopeptides in laboratory testing, then using an aminoglycoside in combination with these antibiotics will be effective. A characteristic that shows tolerance to large dosages of aminoglycosides is known as high-level aminoglycoside resistance (HLAR). It is an acquired resistance.

Resistance to Macrolides, Lincosamides and Streptogramins

The initial mechanism of resistance to macrolides that was reported was attributed to the activity of adenine-N6-methyltransferase. This process necessitated the post-transcriptional modification of 23S rRNA. Enzymes from this category are responsible for the transfer of one or two methyl residues to an adenine molecule in 23S rRNA, resulting in the formation of N6-methyladenine or N6, N6-dimethyladenine. Consequently, this not only reduces the binding of erythromycin in the ribosome, but it also reduces the binding of other macrolides (such as azithromycin and clarithromycin), lincosamides, and streptogramin B. The phenotype that indicates the presence of this mechanism is MLSB, which is expressed by the gene erm(B) or, less frequently, by erm(A). Another factor that may be employed to determine whether or not bacteria are resistant to macrolides and lincosamides is their ability to produce specific enzymes that render antibiotics inactive.

Resistance to Antimetabolites-Sulphonamides and Trimethoprim

Sulphonamides are able to block the formation of dihydrofolic acid because they function as competitive

inhibitors of the enzyme known as dihydropteroate synthase (DHPS). The creation of dihydropteroate synthase by the bacteria results in a decrease in the bacterium's affinity for the drug. This decrease in affinity is brought about by the bacteria. P-aminobenzoic acid, when produced in excessive quantities, engages in direct competition with other chemicals for access to the active center of DHPS. This is an additional consequence of excessive synthesis of p-aminobenzoic acid. As a consequence of the antibiotic's activity, some bacteria are able to produce an alternate metabolic pathway because of the antibiotic's presence. The drug has the ability to block a pathway, but this pathway has the ability to replace that pathway. This phenomenon is known as a "bypass mechanism," and it is recognized as a means of acquiring resistance to sulphonamides and trimethoprim. It is also known as a "bypass mechanism." It is necessary for bacteria to produce folacin in order for them to be able to produce nucleic acids. This is because the majority of bacteria are unable to accept folacin from their environment. Additionally, the combination of trimethoprim and sulfamethoxazole blocks two subsequent steps in the pathway that leads to the production of tetrahydrofolate. Consequently, this hinders the formation of folic acid and removes a broad range of germs in a way that is synergistic.

Resistance to Fluoroquinolones

Quinolones inhibit the growth of bacteria by interacting with DNA gyrase, topoisomerase IV, and type II topoisomerase, all of which are essential for bacteria to generate copies of their DNA. DNA gyrase is composed of two components, which are referred to as gyrA and gyrB respectively. It is mostly gram-positive bacteria that are affected by quinolones' topoisomerase IV activity. The two components that make up this structure are referred to as parC and par E, and they are analogous to gyrA and gyrB. A few minor alterations in the chromosomes of enterococci are responsible for their resistance to fluoroquinolone infections. It is thus impossible for genes that confer resistance to fluoroquinolones on bacteria to be transferred to other bacteria via the process of genetic material transfer. There is a correlation between the effectiveness of antibiotic therapy with medications belonging to this category and the frequency with which this mutation is found in the genome of bacteria. The genes that code for topoisomerase II (gyrase) and topoisomerase IV are the ones that undergo the majority of the modifications.

Resistance to β-Lactams

β-lactamases may be produced by bacteria either on their own or when they are instructed to do so. Cells are constantly producing constitutive enzymes, and the quantity of these enzymes that they produce does not alter regardless of whether or not antibiotics are present in the environment. It is a characteristic that comes with the strain or type of bacteria. Bacteria have the potential to develop resistance to antibiotics by natural processes, provided that they produce a enough amount of the enzyme known as constitutive β-lactamase. Furthermore, the presence of a medication known as β-lactam in the environment has the potential to induce the production of triggered β-lactamases by bacteria. The process of producing these enzymes continues until the activator is removed from the surrounding environment. The temporary resistance is established in this manner. There are potential alterations that might occur during the process of induction, which implies that the bacteria continue to produce the induced β-lactamase even after the inducing agent is removed. Because of this, the bacteria will retain their resistance to the antibiotic indefinitely.

AmpC β-Lactamases

With the exception of cefepime, ampC cephalosporins are capable of breaking down all penicillins and the majority of cephalosporins, the majority of which are of the first and second generation. A few of them are able to bind to aztreonam, but they do not break down the medication overall. It is not feasible for these enzymes to break down carbapenems, and clavulanic acid does not prevent them from doing so. The Ambler categorization places them in class C, which is the highest possible level. When it comes to E. coli, this particular kind of β-lactamase is often capable of being activated or deactivated. However, it is always present at a very low level and does not confer β-lactam resistance on wild strains from the bacteria. AmpC β-lactamases that are encoded on chromosomes have been discovered in E. coli strains that originate from both human and animal sources.

Resistance to Sulphonamides and Trimethoprim

To be resistant to sulphonamides, some bacteria possess sul genes that produce dihydropteroate synthase, an enzyme that does not react well with sulphonamides. This is the reason why certain bacteria are resistant to sulphonamides. Sulphonamides are present in the environment, which indicates that the bacteria are able to develop properly in that setting. At this point in time, there have been discovered four genes on plasmids that confer resistance to sulphonamides on plants. sul1, sul2, sul3, and sul4 are the names of these genes. Plasmids known as IncFII and IncI1 are responsible for the transmission of genes that confer resistance to suplhonamides in cells. When a cell has the dfr gene, which is responsible for the production of dihydrofolate reductases, the cell is resistant to the antibiotic trimethoprim. This antibiotic does not have the ability to delete these enzymes. Within the gene cassette that is located in the variable section of class 1 integrons is where the sul1 gene is located. Commonly, it coexists with other genes that confer resistance.

Resistance to Methicillin Macrolides Lincosamides and Streptogramin B

Any species of Staphylococcus that is resistant to streptogramin B, *lincosamides*, or macrolides will also be resistant to MLSB. These antibiotics inhibit protein synthesis by attaching to adenine at positions 2058 or 2059 in domain V of the 23S rRNA subunit. The erm genes encode ribosomal methylase, an enzyme that changes adenine at the antibiotics' target site in the 23S rRNA subunit. This process prevents the antibiotics from attaching to bacterial cells. There is MLSB resistance that is both constitutive and inducible. Constitutively resistant bacteria may still produce methylase when an inductor is not present, all because of their active messenger RNA. When an inductor is present, it activates the generated dormant mRNA, which results in the induction of MLSB resistance.

The majority of methicillin-resistant Staphylococcus aureus (MRSA) strains carry two or more erm genes, with erm(A) and erm(C) being the most common combinations (B) Common antibiotics such as gentamicin, sulphonamides, kanamycin, streptomycin macrolides, fluoroquinolones, and tetracyclines are not only ineffective against methicillin, but also against multidrug-resistant bacteria. As a typical defense mechanism, S. aureus bacteria often produce transferases known as aminoglycoside-modifying enzymes (AME). Acetyltransferase, phosphotransferase, nucleotidyltransferase, and ANT are all enzymes that fall into this category (9-I). In order to make antibiotics useless against bacteria, enzymes change the molecule in a certain manner.

Conclusion

The current investigation was carried out with the purpose of investigating the patterns of antibiotic resistance experienced by common clinical bacterial pathogens that were isolated from a variety of clinical specimens. The investigation focused on identifying the predominant bacterial species, determining their antimicrobial susceptibility profiles, assessing the prevalence of multidrug-resistant organisms, comparing resistance patterns across bacterial species and specimen types, and developing evidence-based recommendations for empirical therapy, antimicrobial stewardship, and infection-control practices. The study concludes that antibiotic resistance represents a significant problem among common clinical bacterial isolates. Resistance was not confined to a single organism or antibiotic class but was distributed across multiple pathogens and antimicrobial agents. E. coli was the most often isolated pathogen, while gram-negative organisms were the most common overall. It may not be safe to depend on ampicillin and third-generation cephalosporins as empirical treatments in the absence of evidence of local susceptibility due to the high resistance to these drugs.

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