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PHARMACOKINETICS AND PHARMACODYNAMICS OF COMMONLY USED ANTIBIOTICS: A COMPREHENSIVE REVIEW

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ABSTRACT

Antibiotics are common agents used in modern healthcare. This was not always the case. From ancient times, people sought ways to treat those with infections. Dyes, molds, and even heavy metals were thought to hold promise for healing. Various microorganisms have medical significance, including bacteria, viruses, fungi, and parasites. Antibiotics are compounds that target bacteria and, thus, are intended to treat and prevent bacterial infections. This activity will examine the various classes of antibiotics, their mechanisms of action, bacterial susceptibilities, and potential adverse events. Antibiotics are chemical substances that inhibit the growth of or destroy bacteria and are widely used in the treatment of bacterial infections. Since the discovery of penicillin by Alexander Fleming in 1928, antibiotics have revolutionized modern medicine, drastically reducing morbidity and mortality from infectious diseases. Antibiotics are classified based on their structure, mechanism of action, and spectrum of activity. Common classes include penicillins, cephalosporins, tetracyclines, aminoglycosides, and fluoroquinolones. However, the widespread and often inappropriate use of antibiotics has led to the emergence of antibiotic-resistant bacteria, posing a significant global health threat. This abstract provides a brief overview of the types, mechanisms, applications, and challenges related to antibiotics, emphasizing the need for responsible use and continued research to combat resistance and discover novel antimicrobial agents.

KEYWORDS: Antibiotic, Bacterial infections, Antibiotic resistance, antimicrobial agents.

1. INTRODUCTION

Antibiotics are chemical agents used to inhibit or kill bacteria. With the rise of antibiotic resistance, it's critical to understand the efficacy of different antibiotics against various pathogens.[1] This study compares the effectiveness of selected antibiotics using standard microbiological methods. Antibiotics are chemical substances produced by microorganisms or synthetically derived that inhibit the growth or kill other microbes. They have revolutionized modern medicine by effectively treating bacterial infections. However, the widespread use and misuse of antibiotics have led to the emergence of antibiotic-resistant strains, which is a growing global concern.[2]

The pharmacology behind antibiotics includes destroying the bacterial cell by either preventing cell reproduction or changing a necessary cellular function or process within the cell. Antimicrobial agents are classically grouped into two main categories based on their in vitro effect on bacteria: bactericidal and bacteriostatic. Common teaching often explains that bactericidal antibiotics "kill" bacteria and bacteriostatic antibiotics "prevent the growth" of bacteria. The true definition is not so simple. To accurately define each category, the minimum inhibitory concentration (MIC) and minimum bactericidal concentration (MBC) must be understood. The lowest concentration that inhibits visible bacterial growth at 24 hours is the MIC. The MBC is the concentration of an antibiotic that reduces bacterial density by 1000-fold at 24 hours.[3]

2. Objectives

- Identify the various classes of antibiotic medications.-

- Explain the various mechanisms of action of different classes of antibiotics.
- Review the potential adverse effects both of antibiotics in general and class-specific side effects.
- Summarize the monitoring requirements for patients receiving antibiotic therapy. To compare the antibacterial activity of different antibiotics.
- To determine the most effective antibiotic against selected bacterial strains.
- To evaluate resistance patterns among bacteria. To compare the efficacy of different antibiotics (e.g., Amoxicillin, Ciprofloxacin, Erythromycin, and Gentamicin).
- To test their effect on common pathogenic bacteria (e.g., E. coli, S. aureus, Pseudomonas aeruginosa, Salmonella typhi).
- To interpret the zone of inhibition using the disk diffusion method.[4]

3. Common antibiotics

A. Amoxicillin
B. Ciprofloxacin
C. Azithromycin
D. Clindamycin
E. Gentamycin

A. Amoxicillin

Amoxicillin is a β-lactam antibiotic that works by inhibiting bacterial cell wall synthesis. It binds to penicillin-binding proteins (PBPs), which are essential for peptidoglycan cross-linking in the bacterial cell wall.[5] Amoxicillin is bactericidal and is most effective against Gram-positive bacteria such as Streptococcus and Enterococcus, and some Gram-negative organisms like Escherichia coli and Haemophilus influenzae. It is widely used to treat respiratory infections, urinary tract infections (UTIs), and skin infections. However, resistance is common due to β-lactamase-producing bacteria, which can inactivate the drug. In such cases, it is often combined with clavulanic acid to inhibit β-lactamase activity. Common side effects include nausea, diarrhoea, and allergic reactions.[6,7,8]

Pharmacokinetics (PK)

- **Absorption:** Well, absorbed orally (bioavailability ~75%).
- **Distribution**: Widely distributed in body fluids and tissues; low protein binding
- **Metabolism:** Minimal hepatic metabolism.
- **Excretion:** Primarily excreted unchanged in urine through renal elimination.
- **Half-Life** : About 1–1.5 hours.[9]

Pharmacodynamics of Amoxicillin

- **Mechanism of Action:** Amoxicillin is a β-lactam antibiotic that inhibits bacterial cell wall synthesis by binding to penicillin-binding proteins (PBPs), causing cell lysis and death.
- **Type of Killing**: Time-dependent bactericidal activity – effectiveness depends on how long the drug concentration stays above the minimum inhibitory concentration (MIC).
- **Spectrum**: Broad-spectrum against Gram-positive and some Gram-negative bacteria.[10]

B. Ciprofloxacin

Ciprofloxacin belongs to the fluoroquinolone class and functions by inhibiting bacterial DNA gyrase and topoisomerase IV, enzymes involved in DNA replication and transcription.[2] This results in the rapid death of susceptible bacteria, making ciprofloxacin a bactericidal agent. It is particularly effective against Gram-negative bacteria such as Pseudomonas aeruginosa, Klebsiella pneumoniae, and Salmonella species, and also works against some Gram-positive organisms. Ciprofloxacin is commonly prescribed for UTIs, gastrointestinal infections, and typhoid fever. Resistance can develop due to mutations in target enzymes, efflux pumps, or reduced membrane permeability. Major side effects include gastrointestinal upset, tendon rupture, CNS disturbances, and QT interval prolongation, especially in elderly or cardiac patients.[11,12]

Pharmacokinetics of Ciprofloxacin

- **Absorption:** Rapidly absorbed orally; bioavailability ~70%.
- **Distribution:** Widely distributed in tissues and body fluids; high concentrations in urine, lungs, and GI tract.
- **Metabolism:** Partially metabolized in the liver (~15%).
- **Excretion:** Mainly excreted unchanged through the kidneys (renal excretion).
- **Half-life:** About 4 hours.[13]

Pharmacodynamics of Ciprofloxacin

- **Mechanism of Action:** Inhibits DNA gyrase and topoisomerase IV, enzymes essential for bacterial DNA replication and repair.
- **Effect:** Bactericidal (kills bacteria).
- **Activity:** Concentration-dependent killing with post-antibiotic effect (PAE).
- **Spectrum:** Broad-spectrum antibiotic effective against many Gram-negative and some Gram-positive bacteria.[14]

C. Azithromycin

Azithromycin is a macrolide antibiotic widely used to treat various bacterial infections. It is widely used to treat various bacterial infections including pneumonia, bronchitis, sinusitis, skin and soft tissue infections, otitis, typhoid fever and sometime prescribed in diarrheal diseases. due to its broad-spectrum activity and long half-life. It works by inhibiting bacterial protein synthesis, stopping the growth and multiplication of bacteria. It produce nausea, vomiting, diarrhoea, abdominal pain, allergic reaction and rarely liver toxicity, Prolongation of QT.[15.16]

Pharmacokinetics of Azithromycin[17]

- **Absorption:** Oral bioavailability ~37%, peak plasma concentration in 2–3 hours.
- **Distribution:** Extensive tissue penetration (lungs, macrophages).
  Long tissue half-life → once-daily dosing.
- **Metabolism:** Minimal hepatic metabolism (CYP3A4), fewer drug interactions.
- **Excretion:** Mainly via bile/faces, small amount via urine (6–14%).
  Terminal half-life ~68 hours.

Pharmacodynamics of Azithromycin[18]

- **Mechanism of Action**
- Binding to Ribosome:

Azithromycin binds specifically to the 50S ribosomal subunit of susceptible bacteria.

- Blocking the Peptidyl Transferase Centre:

It interferes with the peptidyl transferase activity, preventing the proper alignment of tRNA.

D. Clindamycin

- Clindamycin is a lincosamide antibiotic derived from lincomycin. It is bacteriostatic but can be bactericidal at higher concentrations. It is commonly used for serious bacterial infections likely, Skin and soft tissue infections (e.g., cellulitis, abscesses), Bone and joint infections (e.g., osteomyelitis), Respiratory tract infections (e.g., pneumonia), Intra-abdominal infections (with anaerobes), Dental infections, Alternative to penicillin for patients with allergies caused by anaerobic bacteria and certain gram-positive organisms. In sometime unwanted effects such as, Nausea, vomiting, diarrhoea, Abdominal pain, Skin rashes, Severe allergic reactions (anaphylaxis, rash), Liver toxicity (rare).[19,20]

Pharmacokinetics of Clindamycin

- **Absorption**: well, absorbed orally (bioavailability ~90%).
- **Distribution**: Widely distributed, including bone and tissues.
- **Metabolism:** Extensively metabolized in the liver.
- **Excretion:** Mainly through bile and faces.
- **Half-life**: 2–3 hours.

Pharmacodynamics of Clindamycin

- **Mechanism of Action:** Binds to the 50S ribosomal subunit of bacteria → inhibits protein synthesis.
- **Effect:** Mainly bacteriostatic (stops bacterial growth), but bactericidal at higher concentrations.
- **Activity:** Effective against Gram-positive bacteria and anaerobes.
- **Resistance:** May occur due to modification of ribosomal binding sites.[21]

E. Gentamicin

Gentamicin is a powerful aminoglycoside antibiotic that binds to the 30S ribosomal subunit, causing misreading of mRNA and inhibiting protein synthesis.[22] It is bactericidal and is most effective against aerobic Gram-negative bacteria, including Pseudomonas aeruginosa, Proteus, E. coli. Due to its potential toxicity, gentamicin is typically used in severe hospital-acquired infections or sepsis, often in combination with other antibiotics for synergistic effects. Its major drawback is toxicity nephrotoxicity (kidney damage) and ototoxicity (hearing loss) are serious side effects.[23,24]

Pharmacokinetics of Gentamicin

- **Absorption:** Poorly absorbed from the gastrointestinal tract, so it is given parenterally.
- **Distribution:** Distributes well in extracellular fluids (blood, urine, pleural fluid).
- **Metabolism:** Not metabolized in the body; remains in its active form.
- **Excretion:** Clearance is directly related to kidney function.
- **Half-life:** 2–3 hours in healthy adults.

Pharmacodynamics of Gentamicin

- Gentamicin is an aminoglycoside antibiotic.
- It exhibits bactericidal activity by inhibiting bacterial protein synthesis.
- Binds to the 30S ribosomal subunit, causing misreading of mRNA.
- Effective mainly against aerobic Gram-negative bacteria.[25]

4. Methods and Materials Used

METHOD

- Prepare nutrient agar plates and allow them to solidify.
- Inoculate each plate with one bacterial strain using a sterile swab.
- Place antibiotic disks on the surface of the agar.
- Incubate plates at 37°C for 24 hours.
- Measure the diameter of the zone of inhibition (in mm) around each disk.

MATERIALS

- Nutrient agar plates
- Bacterial strains: E. coli, S. aureus, Pseudomonas aeruginosa, Salmonella typhi
- Antibiotic disks: Amoxicillin, Ciprofloxacin, Azithromycin, Clindamycin, Gentamicin
- Sterile cotton swabs
- Petri dishes, Incubator.[26]

5. Pharmacokinetics and Pharmacodynamics

Pharmacokinetic (PK) and pharmacodynamic (PD) parameters are used together to maximize the efficacy of antimicrobial therapy through optimization of dosing in patients. Absorption, distribution, metabolism, and excretion are the PK components that affect the

antibiotic concentration over time. These processes describe how an antibiotic moves through the body from the time it enters the body until the parent drug or metabolites are removed.[27] PD of an antibiotic describes the drug effect within the body when it reaches the infection target. The main principles that guide PD are the percent of the time the free drug is over the MIC, the amount of free drug area under the concentration to MIC, and the maximum concentration to MIC.[28]

Bactericidal activity is either concentration-dependent or time-dependent. If an antibiotic displays concentration-dependent killing, for example, fluoroquinolones or daptomycin, the efficacy of bacterial killing increases as the concentration of the antibiotic increases. Penicillin and tetracyclines are time-dependent; therefore, the duration of the effective concentration of these antibiotics determines bactericidal activity.[29]

After an antibiotic is absorbed, the distribution influences the extent of antimicrobial activity. The total amount of drug in the body to serum concentration is the volume of distribution. The level of protein binding will affect the availability of the active drug at the site of infection. If an antibiotic is highly protein-bound, there will be less free drug available for an antimicrobial effect, as seen in patients with hypoalbuminemia. Increased adipose tissue in a patient will increase the volume of distribution if a drug has high lipophilicity properties.[30]

The location of infection is crucial to note because some antibiotics are inappropriate for treating certain infections. In the treatment of meningitis, for example, the penetration of the blood-brain barrier is critical if one wants to achieve therapeutic antibiotic levels at the site of infection to prevent treatment failure.[31]

6. Adverse Reactions

All medications have the potential for an adverse reaction, and antibiotics are no exception. One in five hospitalized patients has been shown to develop an adverse reaction to an antibiotic, and nearly the same proportion of drug-related Emergency Department visits are due to adverse antibiotic reactions. An immune-mediated reaction or hypersensitivity is classified as an allergy. This includes IgE -mediated anaphylaxis and angioedema. Medications often reach harmful levels in the body due to reduced metabolism and elimination, or high dosing regimens can cause toxicity due to supratherapeutic drug levels.[32] If a reaction occurs that is not mediated by the immune system and is unrelated to the drug level; then it is considered a side effect.[33]

The anticipation of adverse events is warranted when initiating antimicrobial therapy. Certain patients are at higher risk, for example, the elderly, patients with multiple co-morbidities, and hospitalized patients. It is important to monitor patients for reactions as many develop over time. Some antibiotics necessitate monitoring drug levels to guide therapy for efficacy and prevention of adverse effects such as vancomycin and aminoglycosides. Renal toxicities may develop if these antimicrobials maintain high trough levels; therefore, monitoring renal function is necessary and measuring drug levels.[34]

Common Adverse Reactions Associated[35]

Renal

Acute tubular necrosis, Interstitial nephritis, Renal failure, Crystallization in renal tubules.

Cardiac

QT prolongation.

Hematologic

Thrombocytopenia, Leukopenia, Agranulocytosis, Abnormal platelet aggregation, INR increase (often due to drug interactions)

Dermatologic

Rash, Erythema multiforme, Stevens-Johnson syndrome, Toxic epidermal necrolysis.

Neurologic

Ototoxicity, Vestibular dysfunction, Seizure, Peripheral neuropathy.

Other

Hepatotoxicity, Myopathy, Drug-induced fever, Drug-induced diarrhoea.

7. Antibiotic Resistance

The increased use of antimicrobial agents in clinical practice and other industries such as livestock farming has led to bacterial resistance to antibiotic agents. Bacteria have developed mechanisms to promote this resistance to survive.[36]

The MIC of a bacterial isolate can serve as a metric for bacterial susceptibility to certain antibiotics. A high MIC above the susceptibility threshold to an antibiotic will report as a resistant infection. Bacteria may possess resistance to an antimicrobial agent due to intrinsic or acquired properties. Not all antibiotics are effective against all types of bacteria.

If a bacterium does not contain the target for a particular antibiotic, it is to have intrinsic resistance. Vancomycin, an antibiotic known to target work against gram-positive bacteria, cannot cross the cell wall of gram-negative bacteria. Also, beta-lactam antibiotics require a cell wall to function and, therefore, will not be effective against bacteria such as Mycoplasma species that lack this cellular component.

Bacteria also have the capability to gain resistance through attaining resistance genes from other bacteria or developing a mutation resulting in reduced or elimination

of antibiotic efficacy. This type of resistance is known as acquired resistance.

More than one type of bacterial resistance may be present in a bacterial organism. Common resistance strategies are listed here.[37,38]

9. Mechanisms of Resistance[39]

Reducing Intracellular Antibiotic Concentrations

- Increased efflux
- Decreased influx

Antibiotic Inactivation

- Enzymatic modification
- Chemical degradation

Target Site Alteration

- Mutation of the target site
- Antibiotic modification
- Target site protection
- Elimination of the target site.

10. Clinical Significance

Approach to Antimicrobial Therapy

The causative organisms and infection source are not always known when a patient first presents. Antibiotic therapy is often initiated before an exact infectious disease diagnosis, and microbiological results are available. Antibiotics used in this manner are referred to as empiric therapy. This approach attempts to cover all potential pathogens. When microbiology tests result and antibiotic susceptibilities are known, definitive antibiotic treatment can be tailored to the specific infection etiology.[40]

Prophylactic therapy is used to prevent infections in patients who do not have an active infection. Immunocompromised patients may receive prophylaxis against specific opportunistic pathogens. Prophylactic antibiotics are also used before surgical procedures and traumatic injuries such as open fractures and animal bites.

The severity of potential bacterial infection will determine the level of aggressiveness in antibiotic therapy. For example, in a life-threatening infectious disease such as sepsis, empiric broad-spectrum parenteral antibiotics should be administered quickly after sepsis identification and continued until more information is gathered regarding the etiology and causative bacteria. Empiric antibiotics are used to cover all potential bacteria before culture results. After bacterial cultures are available and have resulted, antibiotics can be deescalated to only what is necessary. This approach is termed directed antibiotic therapy. Often, empiric antibiotics are broad-spectrum, which refers to medications targeting many different bacterial classes (i.e., gram-positive, gram-negative, and anaerobic bacteria). In a simple skin and soft tissue infection that does not require hospitalization, narrower spectrum antibiotics may be given orally.[41]

In addition to the possible source(s) of infection, likely pathogens, and situation urgency, different patient factors merit consideration. Patient age, medication allergies, renal and hepatic function, past medical history, the presence of an immunocompromised state, and recent antibiotic usage need to be evaluated before an antibiotic selection. Many of these patient factors contribute to the pharmacodynamics and pharmacokinetics of antibiotics that will influence dosing to optimize efficacy.[42]

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