NAME: IKYER DOOYUM

MATRIC NUMBER: 17/MHS06/039

ONE PROTEIN SYNTHESIS INHIBITOR IS;

**Chloramphenicol**

Chloramphenicol is a bacteriostatic broad-spectrum antibiotic that is active against both aerobic andanaerobic gram-positive and gram-negativeorganisms**.** It is active also against rickettsia*. Hemophilic influenza, N. meningitides, and some strains of Bactericides are highly* susceptible, and for them chloramphenicol may be bactericidal. Clinically significant resistanceemerges and may be due to production of chloramphenicol acetyltransferase, an enzyme that inactivates the drug. This is by the transfer of R- factor by conjugation.

**Mechanisms of action:**

Chloramphenicol blocksproper binding of 50S site which, stops protein synthesis. It does inhibit mitochondrial ribosomal protein synthesis because these ribosomes are 70S, the same as those in bacteria. It hinders the transfer of the elongating peptide chain to the newly attached amino acyl tRNA at the ribosome mRNA complex .It specifically attaches to the 50S ribosome and therefore hinder the access of amino acyl-tRNA to the acceptor for amino acid incorporation, It prevents formation of peptide bond, This may be responsible for the dose related anemia caused by chloramphenicol

**Pharmacokinetics:** chloramphenicol is rapidly and completely absorbed. It is widely distributed to virtually all tissues and body fluids. The drug penetrates cell membranes readily. Excretion of active chloramphenicol and of inactive degradation products occurs by way of the urine. A small amount of active drug is excreted into bile or feces. Newborns less than a week old and premature infants clear chloramphenicol inadequately.

**Clinical Uses**:

Because of potential toxicity, bacterial resistance, and the availability of other effective drugs, chloramphenicol may be considered mainly for treatment of serious rickettsia infections, bacterial meningitis caused by a markedly penicillin-resistant strain of pneumococcus or meningococcal, and typhoid fever.

**Toxicity**

Newborn infants lack an effective glucuronic acid conjugation mechanism for the degradation and detoxification of chloramphenicol. Consequently, when infants are given dosages above 50 mg/kg/d, the drug may accumulate, resulting in the gray baby syndrome, with vomiting, flaccidity, hypothermia, gray color, shock, and collapse.

**Interaction with other drugs** Chloramphenicol inhibits hepatic microsomal enzymes the metabolize several drugs. Like other bacteriostatic inhibitors of microbial protein synthesis, chloramphenicol can antagonize bactericidal drugs such as penicillin’s or aminoglycosides

**Adverse Reactions**

**Gastrointestinal disturbances**: Adults occasionally develop nausea, vomiting, and diarrhea.

Oral or vaginal candidiasis may occur as a result of alteration of normal microbial flora.

**Bone marrow disturbances***: Chloramphenicol commonly causes a dose-related reversible* suppression of red cell production at dosages exceeding 50 mg/kg/d after 1-2 weeks. Aplastic anemia is a rare consequence of chloramphenicol administration by any route. It is an idiosyncratic reaction unrelated to dose, though it occurs more frequently with prolonged use. It tends to be irreversible and can be fatal.