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**DEPARTMENT: NURSING**

**COURSE TITLE: SYSTEMIC PHARMACOLOGY**

**COURSE CODE: PHA 324**

**Classify the antimalarial agents and state the mechanism of action of each of the drug listed**

**CLASSIFICATION OF ANTIMALARIAL AGENTS**

They include;

* 4 Aminoquinolines: Chloroquine and Amadioquine
* Quinoline methanol: Mefloquine
* Chinchona alkaloid: Quinine, Quinidine
* Biguanides: Proguanil (Chloroguanide)
* Antibiotics: Doxycycline, Tetracycline
* Sulphonamides and sulfones: Dapsone, Sulfadoxin
* Sesquiterpine Lactones: Artesunate, Artemeter, Arteether
* Diaminopyrimidines: Pyrimethamine
* Amino Alcohols: Lumefantrine, Halofantrine
* 8 Aminoquinolines: Primaquine, Tafenoquine
* Naphthyridine: Pyronaridine
* Naphthoqunone: Atavoquone

**MECHANISM OF ACTION FOR EACH DRUG CLASS**

 **4 AMINOQUINOLINES (Chloroquine and Amadioquine):** It is actively concentrated in the sensitive intra erythrocytic plasmodia by accumulating in the acidic vesicles of the parasite and the weakly basic nature. It raises the vesicular ph and thereby interfering with the degradation of hemoglobin by parasitic lysosomes. Polymerization of toxic heame to nontoxic parasite pigment hemozoin is inhibited by formation of the Chloroquine-heame complex. Heame itself or in complex with Chloroquine then damages the plasmodial membranes. Clumping of pigment and changes in parasite membranes follow: death.

**QUINOLINE METHANOL (Mefloquine):** It is actively concentrated in the sensitive intra erythrocytic plasmodia by accumulating in the acidic vesicles of the parasite and the weakly basic nature. It raises the vesicular ph and thereby interfering with the degradation of hemoglobin by parasitic lysosomes. Polymerization of toxic heame to nontoxic parasite pigment hemozoin is inhibited by formation of the Mefloquine-heame complex. Heame itself or in complex with Mefloquine then damages the plasmodial membranes. Clumping of pigment and changes in parasite membranes follow: death.

**CINCHONA ALKALOIDS (Quinine and quinidine):** It is actively concentrated in the sensitive intra erythrocytic plasmodia by accumulating in the acidic vesicles of the parasite and the weakly basic nature. It raises the vesicular ph and thereby interfering with the degradation of hemoglobin by parasitic lysosomes. Polymerization of toxic heame to nontoxic parasite pigment hemozoin is inhibited by formation of the Quinine-heame complex. Heame itself or in complex with Quinine then damages the plasmodial membranes. Clumping of pigment and changes in parasite membranes follow: death.

**AMINO ALCOHOLS (Lumefantrine and Halofantrine):** It is actively concentrated in the sensitive intra erythrocytic plasmodia by accumulating in the acidic vesicles of the parasite and the weakly basic nature. It raises the vesicular ph and thereby interfering with the degradation of hemoglobin by parasitic lysosomes. Polymerization of toxic heame to nontoxic parasite pigment hemozoin is inhibited by formation of the Lumefantrine-heame complex. Heame itself or in complex with Lumefantrine then damages the plasmodial membranes. Clumping of pigment and changes in parasite membranes follow: death.

**DIAMINOPYRIMIDINES (Pyrimethamine):** it inhibits plasmodial hydrofolate reductase, reducing the production of folic acid required for nuclei acid synthesis in the malarial parasite. It is used to treat acute malaria but not for prophylaxis.

**SULPHONAMIDES AND SULFONES (Sulfadoxin and Dapsone):** they interfere withfolic acid synthesis by preventing addition of para-aminobenzoic acid (PABA) into the folic acid molecule through competing for the enzyme, dihydropteroate synthase.

**ANTIBIOTICS (Tetracycline and doxycycline):** they inhibit the initiation of translation in variety of ways by binding to the 30S ribosomal subunit, which is made up of 16S-rRNA and 21 proteins. They inhibit the binding of aminoacyl-tRNA to the mRNA translation complex.

**BIGUANIDES (Proguanil/Chloroguanide):** is a prophylactic antimalarial drug, which works by stopping the malaria parasite, Plasmodium falciparum and Plasmodium vivax, from reproducing once it is in the red blood cells. It does this by inhibiting the enzyme, dihydrofolate reductase which is involved in the reproduction of the parasite.

**NAPHTHOQUONE (Avotaquone):** it possesses a novel mode of action against Plasmodium falciparum through inhibition of the electron transport system at the level of cytochrome bc1 complex. Avotaquone also causes the collapse of the parasite mitochondrial membrane potential in Plasmodium falciparum.

**NAPHTHYRIDINE (Pyronaridine):** It is actively concentrated in the sensitive intra erythrocytic plasmodia by accumulating in the acidic vesicles of the parasite and the weakly basic nature. It raises the vesicular ph and thereby interfering with the degradation of hemoglobin by parasitic lysosomes. Polymerization of toxic heame to nontoxic parasite pigment hemozoin is inhibited by formation of the Pyronaridine-heame complex. Heame itself or in complex with Pyronaridine then damages the plasmodial membranes. Clumping of pigment and changes in parasite membranes follow: death.

**8 AMINOQUINOLINES (Primaquine):** It eliminates tissue erythrocyte infection. Thereby, kit prevents the development of the erythrocytic forms of the parasite which is responsible for relapse in vivax and ovale malaria. Primaquine phosphate is also active against gametocyte of Plasmodium falciparum.

**SESQUITERPENE LACTONES (Artemeter, Arteether, and Artesunate):** the mechanism of action for Artesunate is thought to involve the cleavage of the endoperoxide bond. Though reaction with heame. This produces free radicals with alkylate parasitic proteins. It has been shown to inhibit an essential parasite calcium adenosine triphosphate enzyme.

The mechanism of action for Artemeter