RA Journal of Applied Research
||Volume||3||Issue||04||Pages-895-897||April-2017|| ISSN (e): 2394-6709
www.rajournals.in

Blood Cells Positive Increase Regulated By Haemopoietic Stimulating Plasma Antimalarials Concentration

DR. Jimmy E. O

Department Of Physiology, Faculty of Basic Medical Sciences, University of Uyo, Akwa Ibom State - Nigeria

ABSTRACT: Effects of varied plasma concentrations of amalar, chloroquine, cotecxin, fansidar and lapdap on blood cells and its parameters were investigated in twenty four albino rats for 28 days. There was significant difference in the plasma concentration of various antimalarials, amalar; 5.9611mg/L, chloroquine; 3.0140mg/L, cotecxin,; 1.6571mg/L, fansidar; 10.704mg/L and lapdap; 0.9601mg/L, ($P<0.05$). Fansidar showed the highest plasma concentration amongst all the drugs. Despite these plasma variations, chloroquine increased red blood cells, haemoglobin and packed cell volume than other drugs, ($P<0.05$) lapdap increased white blood cells count than others, ($P<0.05$) whereas, cotecxin increased platelet counts than other drugs ($P<0.05$). Chloroquine is found in the study to be very beneficial to blood cells development. It is also shown that the increase in blood cells and its parameters is not a function of the various increase concentration of the antimalarials but it varied haemopoietic stimulating potentials.

Key words: Antimalarials, concentration, haematopoietic , potentials

INTRODUCTION

Antimalaria drugs are used in the treatment of malaria infection. The right choice of the drugs is a function of its requirement for the cure or prevention of the disease. There are many antimalaria drugs the old ones; chloroquine, camoquine, fansidar which are mainly curative. The new ones are amalar, Lumenfantrine/Artemater/cotecxin, lapdap to mention a few which are also preventive and curative. However, drug resistance to almost all these antimalarials has developed (WHO2014). But cotecxin, a dihydro artemisinin and lumenfantine/Artemeter are potent drugs against the resistant malaria species and now as first line treatment drugs. Antimalaria drugs have been observed of recent to ameliorate or directly affect glucose levels (Jimmy, 2014). It is on this note that investigation of selected antimalarials effects on blood cells was carried out and particularly the plasma concentration of these drugs and any related effects in the blood cells development. This is majorly aimed at finding other drugs that could enhance blood cells development and perhaps compete favourably with the old blood haematinics or syrup with high availability and affordability. Blood cells are very essential in the body for oxygen transport e.g. Red cells, and white cells for protection, and for arrest of bleeding, platelets (Hoffman 1981). In malaria disease situation there is reduction in red blood of cells and platelet; anaemia and thrombocytopenia (Jimmy 1993, Essien, 1979) if the antimalarial drugs have haemopoietic stimulating potentials it means malaria patients will benefit maximally and such will reduce the morbidity and mortality malaria related effects associated with blood cells.

Malaria remains a scourge in Africa as it ranks first as the most potent killer disease than HIV/AIDS (Nortey 2007)

About 300,000 children die of malaria years in Nigeria (Unicef 2010). The severest form of the disease is caused by Plasmodium falciparum and it is the most malignant and drug resistant other species are P. Vivax, P. Ovale and P. Malarial (Lucie 2016).

MATERIALS AND METHODS

Experimental: A total of twenty four (24) male and female albino rats weighing 0.7 to 0.147kg were used for the study. They were kept in a well ventilated Department of Pharmacology, animal house, University of Uyo. All the rats were cared for according to the rules and regulations of the instillute of animal ethics committee (IAEC) and all the ethical standard laid down in 1964 declarations of Helsinki were strictly adhered to.

PREPARATION OF DRUGS AND ADMINISTRATION

The drugs; Amalar, chloroquine, cotecxin, fansidar and lapdap were purchased from a Licensed Pharmacy shop in Uyo, Akwa Ibom State, Nigeria. The standard preparation of the drugs was based on the average weight of man 70kg in the preventive dosages of amalar and fansidar and in curative dosages of chloroquine, cotecxin and lapdap. The drugs were administered orally by -passing oesaphagus and delivered into the stomach, Jimmy et al 2014, Bertram 2004 and Robert, et al 1979. The drugs effects were observed for 7, 14, 21 and 28 days.

RA Journal of Applied Research
||Volume||3||Issue||04||Pages-895-897||April-2017|| ISSN (e): 2394-6709
www.rajournals.in

Sample Collection: The rats were anaesthetized with chloroform and sacrificed 7, 14, 21 and 28 days. 5ml of blood was collected into EDTA bottle by cardiac puncture Dacie 2007. Analysis were made for RBC, WBC, Hb platelets and plasma concentration of the antimalaria drugs.

Plasma Drug Analysis: The serial dilution solution of the drugs was prepared from each tablet of the antimalaria. The calibration curve was prepared and the maximum absorbance determined using spectrophometer. This was done for control. Plasma samples were assessed same in the various drugs cumulatively for the period of 28 days.

RESULTS

Effects of the different concentration of antimalaria drugs on the blood cells and its parameters are shown in this study. Amalar with the plasma concentration of 5.9611±0.168 with RBC of 4.1, WBC, 8.6, platelet, 479.3, Hb; 10.43 and PCV of 38.7. Table 1 chloroquine with plasma concentration; 3.0144±0.118 has RBC; 5.1, WBC; 6.3, platelet; 388.8 Hb; 15.45, and PCV; 44.25 table 1. Cotecxin with plasma concentration of 1.6571±0.154 has RBC; 4.6, WBC; 6.7 platelets; 597.5 Hb; 13.90 and PCV of 41.75. Table 1 Fansidar with plasma concentration of 10.704±0.160 has RBC; 4.5, WBC, 4.6, platelet; 380.8, Hb; 13.65 and PCV; 43.3 Table 1. Lapdap with plasma concentration of 0.9704±0.060 has RBC; 4.7, WBC; 9.7, platelet; 409.5, Hb; 14.13 and PCV 42.5, Table 1.

TABLE 1 Effects of antimalarials for 28 days

Drugs	Drugs Concentrations mg/L	RBC x10^{12}/L	WBC No9/L	Platelets x 109/L	Hb g/dL	PCV%
Amalar	5.9611±0.168	4.1	8.6	479.3	10.43	38.75
Chloroquine	3.0144	5.1	6.3	388.8	15.45	44.25
Cotecxin	1.6571±0.154	4.6	6.7	13.90	41.75	41.15
Fansidar	10.70±0.154	4.5	4.6	380.8	13.65	43.3
Lapdap	0.9704+0.060	4.7	9.7x1	409.5	14.13	42.5

DISCUSSION

The effects of the various antimalaria drugs on the blood cells and its parameters are here based on their plasma concentrations. Amalar has been found in this study to affect red blood cells and pack cells volume by reducing their values. It also had the lowest haemoglobin value amongst the antimalaria drugs. This is an indication that amalar is not blood tissue friendly. Such effects are observed with fansidar, these drugs are preventive antimalarials. But very strikingly is that there is no significant difference in their actions on the blood cells and its parameters. This could not be argued otherwise after all both drugs have same pharmacologic contents of pyrimethamine and sulfadoxine. However, their plasma concentrations showed significant variations which would have been thought to impact on the blood cells particularly such high concentration in fansidar.

One would have loved to see such effects on the malaria parasite counts after administration though the drugs are for preventive treatment. But fansidar have been found to have antidiabetic potential which may be the result of its high plasma concentration (Jimmy, 2012). Malaria affects all blood cells parameters i.e. reducing their values, (Jimmy 1993) (Essien, 1979). It is therefore very risky to take any drug that negatively affects the blood cells and their parameters. This is indirectly introducing complications in such treatment e.g. anaemia.

Leukocytosis, thromhocytopemia as seen in the treatment with amalar but it has boosted platelet count. Review in the use of this drug for the management of malaria is therefore very imperative.

However, chloroquine has been shown in the study to increase red blood, cells, platelets, haemoglobin and packed cell volume. This is a very significant development as the negative effects of malaria parasites on the red blood cells e.g destruction of the membrane, feeding on the haemoglobin for its DNA protection and thromhocytopenia is countered by the positive effect of chloroquine.

RA Journal of Applied Research
||Volume||3||Issue||04||Pages-895-897||April-2017|| ISSN (e): 2394-6709
www.rajournals.in

Unfortunately chloroquine is not effectively utilized as antimalaria due to resistant p.falciparium malaria against it. But such showed be reviewed now with the observed haemopoietic stimulating potentials. The parasite clearance inefficacy of this drug may be attributed to the fact that some may be fake and many people take this drug on self prescription or medication (Jimmy 2000). Cotecxin drug with less plasma concentration than chloroquine, amalar and fansidar boosted platelet counts than any of the antimalaria drugs. Platelet function e.g. adhesion, aggregation, and release reaction are dependent also on its count though its viability is associated. Cotecxin could therefore be used in association with thrompoietin in stimulating platelet development and production particularly in bleeding diathesis. Lapdap has the least plasma concentration but boosted red blood cells second to chloroquine and with higher platelet counts. It also increases haemoglobin concentration second to chloroquine. This is encouraging as one of the new antimalaria drugs against the resistant strain. Its high white blood cells count is quite negative to body physiology as such may be a sign of hyper immunine splenomegaly associated with malaria, induced Leukaemia (Jimmy, 1996).

CONCLUSION

The study has revealed a non related plasma concentration of the antimalaria drugs on the blood cells and its parameter. But rather the haemopoietic related potentials of the drugs have been observed.

RECOMMENDATIONS

Chloroquine though is facing resistant from P.falciparum malaria is recommended as effective booster of haematinic therapy, alongside others in the management of malaria disease.

REFERENCES

Bertram, G. (2004): In Basic and Clinical Pharmacology 9th

ed. New York, Chicago, Sanfrancisco, London.

Dacie, J. V., Lewis S. M., Brain J., Bates, I. (2007): In Practical Haematology Churchill, London.

Essien, E. M. Ebhota, M. I. (1983). Platelet Secretary activities in acute malarial plasmodium falciparum Infection. Acta Haematol. 70(3) 183-8.

Hoffbrand, A. V. Moss, P. A. H., & Petil, J. E. (1981): In Essential Haematology, 5th ed. Black Well Publishing.

http://www.google.com.ng/search?9=malaria+mortality.

http://scientist against malaria.net/parasite/ plasmodium. falciparum-(2016).

Jimmy, E. O. and Udofia, A. J. (2014). Yoyo bitters. A potent

Alternative Herbal drug in the treatment of diabetes. International Journal of Innovative Medicine and Health Science 23:1-5.

Jimmy E. O., Sahli. I., Ademowo, O. (2003). Fibrinopeptide

A (FPA) and Fibrinogen interactions in acute Plasmodium falciparum malaria infection Annals of Tropical Medicine and Parasitology 9, (8) 879-881.

Jimmy, E. O., Okon, M. A. (2012). Periodic Validation of high anti-diabetic potentials of unripe plantain in comparison with glibenclamide and fansidar. American Journal of Pharmacology and Toxicology, 7(1): 15-18.

Jimmy E. O., George, A. Bates, I. Bevan, D., Rutherford, I.

(1996). Immunoglobulin Gene PCR to Distinguish Hyper-Reactive Malaria Splenomegaly from African Chronic lymphocytic Leukaemia and Splenic Lymphoma Trans. of Royal Society of Trop. Med. & Hygiene, 90 37-39.

Jimmy, E. O., Achelonu, E. Orji, S. (2000). Antimalaria Dispensing pattern by patent medicine dealers. *Journal of Public Health, England.* 114, 282-285.

Lucie P, Aba P, R, Odile M P, Jean, M A, Francoise, B V, (2016). Plasmodium falciparum: multifaceted resistance to artemisinins. Malaria Journal 15:149

Nortey D (2007) Malaria kills more than HIV/AIDS. https://www.modernghana.com/news Robert, G. (1979). Gastric Cytoprotective property of prostaglandin. Gastroenterol. 77:762-769.

Unicef (2010) Unicef- Nigeria media centre, https://www.unicef.org/nigeria/media

WHO, (2014). Antimalaria drug resistance. http://.www.

who.int/malaria/areas/drug-resistance/overview.en.

Ursos, L. M., Roepe, P. D. (2002). Chloroquine Resistance in the Malaria Parasite, Plasmodium Falciparum. Med.Res.Rev. 22(5) 465-91.