Nigerian Journal of Clinical Practice
December 2002, Vol. 5 (2); 106–108

MONOCYTOSIS IN ACUTE MALARIA INFECTION

N. K. D. Halim, O. I. Ajayi, Fasheemo Oluwafemi*
*Department of Haematology, University of Benin, Benin City and *(Immunology) Medical Laboratory Scientist, Chevron Nigeria Limited, Medical Division, Warri.*

ABSTRACT

The haematocrit (Hct); the white blood cell (WBC) count (total and differential), platelet count and reticulocyte count were determined prior to administration of antimalaria therapy and one week after. A sysmex 2000, auto-analyzer was used to determine all the parameters excluding reticulocyte count which was determined manually. Tests were performed on the blood samples of sixty malaria patients (45 males and 15 females) with uncomplicated malaria, age range 2-15 years. The differential white blood cell counts revealed monocytosis pre-therapy and post therapy. Monocytosis observed was significantly higher in patients post-therapy compared to the pre-therapy patients (P-0.05). There was no significant change in the Hct, total WBC count platelet count and reticulocyte count in both the pre-therapy and post-therapy patients.

The monocytosis observed in patients, especially those on antimalaria therapy, may be indicative of an anti-malaria effect by monocytes. Thus monocytosis, may enhance the predisposition to a favourable clinical outcome.

INTRODUCTION

Haematological changes occurring in malaria include anaemia[1,2] thrombocytopenia[5], monocytosis, atypical lymphocytosis and disseminated intravasculation coagulation[3].

Previous studies by Zukerman et al[1] and Perrin et al[2], have revealed the destruction of red blood cells containing parasites at merogony, as a cause of the anaemia in malaria. Also included in the pathogenesis of anaemia is the accelerated destruction of non-parasitized red blood cells, which has been shown to be directly parallel to the level of malaria severity as reported by Liooareesuwan et al[4] and Davis et al[5]. The WBC change that occurs in acute malaria includes monocytosis[3] and neutropenia[6].

Thrombocytopenia in malaria has been ascribed to platelet activation[8]. Although it may also occur in the absence of this mechanism.

This study was undertaken in order to ascertain the effects of acute falciparum malaria on the Hct, WBC, platelet and reticulocyte counts.

METHOD

A descriptive study was performed on sixty malaria patients (45 males and 15 females) with clinical features of uncomplicated malaria (absence of features of cerebral malaria), within the age range 2-15 years. A verbal consent was obtained from the parents of each patient. The patients were Chevron Staff children, recruited from the sick clinic of Chevron Medical Centre Warri. A diagnosis of malaria was made based on the presence of fever, chills and the presence of trophozoites in the blood firm.

The inclusion criteria for the study, included the absence of previous anti-malaria treatment in any of our study patients, prior to the study. Patients with fever but without evidence of trophozoites in their blood film, were excluded from the study.

The blood samples and investigations were performed before noon and within one hour of collection, at the Chevron Medical Clinic Laboratory, Warri, Nigeria. The patients were investigated for the presence of malaria by using the thick and thin blood film technique[9]. The parasite density was determined by counting the number of parasites in the presence of 1000 white blood cells and multiplying by 8000, (the approximate normal while blood cell count) in a giemsa stained thick blood film.

Blood samples were collected, for malaria parasite check prior to the commencement of therapy (pre-therapy) and one week after the commencement of therapy (post-therapy).

The Hct, WBC count and platelet count were determined using the sysmex 2000 auto-analyzer. The reticulocyte count was determined manually. The t-distribution test was to test for significance between means for both tests. P-value less that 0.05 was considered significant.

RESULT

Table 1 shows the mean ± standard deviation (SD) for the Hct, WBC Count (total and differential), platelet count and reticulocyte count in 60 malaria patients. The mean ± SD for Hct was lower in the pre-therapy patients 34.1±1.4%, compared to the post therapy patients 35.5 ± 1.3%. The difference in the mean value was not statistically significant (P>0.05). The mean ± SD for WBC count was slightly higher in the pre-therapy patients 6.1 ± 1.2 x $10^3/\mu L$ compared to 6.0 ± 0.8x$103^3/\mu L$, for the post-therapy patient. The difference in the mean value was not statistically significant (P>0.05). The mean ± SD for differential neutrophil count was 48.5 ± 4.8% for the pre-therapy patients compared

*Correspondence: Dr. N. K. D. Halim

Table 1: **Mean ± SD values for Hct, WBC count (total and differential), platelet count and reticulocyte count, post-therapy and post anti-malaria therapy, in 60 malaria patients.**

Parameters Data	Pre-therapy n = 60	Post-therapy	P value Level of (Significant)
Hct (%)	34.1 ± 1.4	35.5 ± 1.3	P>0.05
WBC (x 10^3/mL)	6.1 ± 1.2	6.0 ± 0.8	P>0.05
Differential Neutrophil Count (%)	48.5 ± 4.8	48.1 ± 3.5	P>0.05
Differential Iympholyte count (%)	15.2 ± 0.2	15.8 ± 0.6	P>0.05
Differential Eosinophil count (%)	2.2 ± 0.2	1.8 ± 0.1	P>0.05
Differential Basophil count (%)	0.8 ± 0.3	0.6 ± 0.2	P>0.05
Differential monocyte court (%)	19.9 ± 0.8	34.4 ± 1.2	P>0.05
Platelet count x 10^3/μL	1.36 ± 22	140 ± 31	P<0.05
Reticulocyte count (%)	1.3 ± 0.2	1.4 ± 0.3	P>0.05

to 48.1 ± 3.5% for the post-therapy patients. The difference in the mean value was not statistically significant (P>0.05). The mean ± SD for the differential lymphocyte count was 15.2 ± 0.2% in the pre-therapy patients, compared to 15.8 ± 0.6% for the post-therapy patient. The difference in the mean value was not statistically significant (P>0.05). The mean ± SD observed for the differential eosinophil count was 2.2 ± 0.2%, for the pre-therapy patients, compared to 1.8 ± 1%, for the post-therapy patient. The difference in mean value was not statistically significant (P>0.05). The mean ± SD observed for the differential basophil count pre-therapy was 0.8 ± 0.3%, compared to 0.6 ± 0.2% for the post-therapy patient. The difference in the mean value was not statistically significant (P>0.05). The mean ± SD observed for the differential monocyte count was 19.9 ± 0.8%, for the pre-therapy patients compared to 34.3 ± 1.2% for the post-therapy patients. The difference in the mean value was statistically significant (P<0.05). The mean ± SD for the platelet count was 1.36 ± 22 x 10^3/μL for the pre-therapy patient, compared to 140 ± 31 x 10^3/μL for the post-therapy patient. The difference in the mean value was statistically significant (P<0.05). The mean ± SD for the reticulocyte count in the pre-therapy patients was 1.3 ± 0.2% and 1.4 ± 0.3%, for the post-therapy patients. The difference in the mean value was not statistically significant (P>0.05).

Table 2: **Parasite density compared for pre and post mean PCV values (n = 60)**

Parameter	Parasite Density (μI)
Mean PCV (Pre-therapy) 34.1 ± 1.4	28 parasites/μl
Mean PCV (post-therapy) 35.5 ± 1.3	3 parasites/μl

n = number of patients

Table 3: **Correlation coefficient (r) for parasite density and mean differential monocyte count (pre and post-therapy)**

Parasite	r	P-value
Pre-therapy Monocyte count (n = 60)19.9 ± 0.8	-0.62	Not significant
Post-therapy Monocyte count (n = 60) 34.3 ±1.2	3.53	

n = number of patients

Table 2 reveals a decrease in parasite density, from 28 parasites/μl pretherapy with a mean PCV of 34.1 ± 1. 4 to a parasite density of 3 parasites /μI, when the PCV slightly improved to 35.5 ± 1.3. The difference in parasite densities pre and post therapy was statistically significant (P<0.05).

The Table 3 reveals the correlation co-efficient of the parasite density and the differential monocyte count. The r value was -0.62 pre-therapy compared to 3.53 post-therapy. The post-therapy r value was statistically significant (P<0.05).

DISCUSSION

This study reveals a marked increase in monocyte count in patients being treated for malaria, compared to the pre-therapy group of patients. This result is similar to that obtained by Mohan et al[10]. The mechanism responsible for the increase in monocytes in this study, cannot be readily explained. It may be as a result of increased chemotaxis (cell mobilization and migration), as monocytes tend to become phagocytic in the presence of protozoa, bacteria and fungi. We also wish to postulate that the longer the lifespan of the malaria parasite in the red blood cell, the greater the likelihood of a higher monocyte count. There may

be some cellular factors, which may account for the increase in monocytes particularly in those patients on anti-malaria therapy, compared to the pretherapy group. The results of this study revealed mild anaemia both pre and post-therapy with values of 34.1 ± 1.4 and 35.5 ± 1.3 respectively. The low degree of parasitaemia, would account for the mild anaemia, as against a severe anaemia. There was evidence of thrombo-cytopenia in the pre-therapy patients, with a mean platelet count of 1.36 ± 22 x 103/μl . Thrombocytopenia is a recognized complication of malaria infection[11]. This is explained on the basis of increased splenic sequestration of platelets, with resultant thrombocytopenia. The result of this study showed a positive correlation coefficient r, for post-therapy mean differential monocyte count, of 3.53 as against the parasite density. This cannot readily be explained. However it is postulated that with a decrease in the parasite density, during the recovery phase in malaria, the monocyte count tends to increase. It is also postulated that an increase in monocyte count is associated with a favorable outcome for these study patients. The reasons could be that monocytosis enhances the phagocytic index in malaria patients, as shown in previous studies[12]. A more favourable outcome could be due to the association between a high monocyte count and the presence of Cytokines, which enhance the inflammatory response. The latter has been highlighted in previous studies[12,13]. It is recommended that malaria patients should have a routine full blood count, in order to ascertain the presence of monocytosis, as a means of predicting a favourable clinical outcome.

ACKNOWLEDGEMENT

We wish to acknowledge the support of the Laboratory Science Staff of Chevron Medical Clinic Warri, for their immense assistance as regards obtaining relevant laboratory data.

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