J. Expt. & Clin. Anat. 4(2): 48-51 (2005).

Comparative Effects Of HIV And Malaria Infections On Certain Haematological Indices In Children - A Hospital Based Study In Enugu

*R O NNELI[1] AND J. EGENE[2]

[1]Department Of Physiology, College Of Medicine & Health Sciences,
Abia State University, Uturu, Nigeria.
[2]Department Of Medical Laboratory Services, Mother Of Christ Specialist Hospital, Ogui, Enugu. Nigeria.

*Author for correspondence

ABSTRACT

This one-year hospital based study investigated the effects of malaria and hiv infections on certain haematological indices in children aged 0 13 years old. HIV screening was carried out using the quick test kits for hiv-1 hiv-2 while malaria parasitaemia was determined by using both thin and thick peripheral blood films stained with Leishman's and Geimsa stains examined under microscope. Hb levels of PCV was also carried out. Total anddifferential white cell counts were determined. The results showed that the higher the malaria parasitaemia, the lower the PCV and total white cell count with a higher neutrophil levels. HIV infections caused a decrease in PCV and an increase in total white cell count with incrased lymphocyte levels. Both infections decreased PCV to levels indicative of moderately severe anaemia. In high malaria endemic regions like Sub-Saharan Africa, a co-infection with HIV would not only worsen anaemia because of its adverse effects on blood cells but increase the mortality rates among the paediatric age as we observed in only one patient with HIV co-morbid malarial infection.

Keywords: HIV, Malaria, Haematological indices, Infection, Children.

HIV/AIDS is an increasingly important cause of under 5 years old mortality, and this mortality cannot be explained alone by HIV infection because of other killer diseases of children in the developing world (Walker et al, 2002). In Sub-Saharan Africa, there is a high malarial endemicity and in areas with stable malaria, severe malaria is mainly a problem of children aged less than 5 years (O'Dempsey, 2002), and infections are less than 5 percent below the age of three months and approach 90 percent by the end of the first year. Anaemia is major health problem in endemic areas due to infection for young children and pregnant women (Ekvall, 2003). According to Xia et al (1998) and Jakobsen et al (1995), Plasmodium falciparum infection modulates HIV pathogenesis by activating lymphocytes and stimulating the viral replication through the production of cytokines. HIV infection has an adverse effect on malaria and makes its symptoms worse and increases the parasite density. Also, malaria infection by causing the release of proinflammatory cytokines, might be expected to encourage HIV replication which in turn would promote HIV transmission and disease progression (Kublin et al, 2005; Whitworth and Hewitt, 2005). In addition to malaria, other conditions like viral, malnutrition, sickle cell disease, poor hygiene cause anaemia in childen (Ekunwe, 1996; Sharma et al, 1985). At Jos University Teaching Hospital, Angyo et a,(2000) showed that infections were common in children with AIDS and that the range of infections appear similar to those observed in children with severe malnutrition. Hence, the objective of this was to elucidate the effects of HIV, co-morbid malaria infection on certain haematological indices of chidlen aged 0-13 years old.

MATERIALS AND METHODS

This one-year study was carried out between January and December 2002 at Mother of Christ Specialist Hospital, Ogui Enugu, Nigeria. The study was approved by the Ethical Committee and informed consent was sought and obtained from the parents of the children.

Selection criteria

Children aged 0 13 years wee screened for haemoglobin genotype (sickling test) to eliminate the effect of sickle cell anaemia on malaria infection Hb SAg screening was done to eliminate the consequences of hepatitis A,B,C

viruses. The control subjects tested negative to both HIV screening and malaria parasite count. A total of sixty (60) children aged 0 13 years old were enlisted for this stud.

Experimental procedures

HIV screening was done using the quick test kits for HIV-1/HIV-2with serum or whole blood (ACON Laboratory Inc., CA 92121, USA). Malaria parasite count, total white blood cell count and differential white blood cell (WBC) count respectively were done using both thin and thick peripheral blood films stained with Leishman's and Geimsa stains examined under the microscope at x 100 magnification (Charterjee, 1976). Haemoglobin (Hb) levels or Packed cell volume (PCV) was carried out by using the cynamethaemoglobin method or microhaematocrit centrifuge and reader (Dacie and Lewis, 1991).

Statistical analysis

Students' t-test and ANOVA were used to compare the means and different variables in the data. A p value of less than 0.05 was considered statistically significant. [POSITION FOR TABLES 1 7].

RESULTS AND DISCUSSIONS

From our result (table 1), malaria, a co-infection in HIV produced a decline in Hb level which agreed with the report f Usanga (1993), and a relative leucopenia with increased neutrophil count thus supporting the postulation of Zeigler et al (1989). Previous studies have reported that malaria caused red blood cells destruction during schizogony, increased red cell rigidity and decrease erythropoiesis (Young and Alters, 1994). Eriksson et al (1989) reported a lower white blood cell counts and marginal Hb levels in malarial patients when compared with the controls, and this agreed with out present result in children who had P. falciparium but without HIV infection (tables 1 and 2). This could have been due to both immuno haemolyticand destruction of parasitised red blood cells (Nand et al, 2001). Uko et al,(1996) reported no significant difference in the haematological indices between the non-infected and infected patients with P. falciparium, however, this present study observed a significant difference ($P<0.05$) tables 1,2,4, and 5). Also, anaemia was observed in the 0 5 years age but no in the 6 13 year range (table 4) thus indicating both the degree of parasitaemia was higher in the former age range hence the anaemia. This too, was consistent with the postulation of Uko et al (1996) that the degree of anaemia in P. falciparium is variable and depends on the degree of parasitaemia. Mc Gregor

Table: Effect of Malaria parasitaemia On Packed Cell Volume (PCV) In Children

	Control (non infected)	Test (infected)	P<0.05
Number	37	23	
Age range	0 – 13		
PCV % (SEM)	38.2 (1.5)	29.2 (5.2)	Yes
Unclassified	6	8	

Table 2: Effect of malaria parasitaemia on total white Blood and differential counts in children

	Control (non-Infected	Test (Infected)	P<0.05
No	37	23	
TWBC% (SEM)	9829.4 (871)	9677.8 (1027	
Neutrophil% (SEM)	47.9 (3.4)	53.2 (4.6)	Yes
Lymphocyte % (SEM)	45.(3.9)	43.2 (4.9)	No
Eosinophil % (SEM)	1.5 (0.7)	3.0 (0.8)	No
Basophil % (SEM)	3.0 (1.4)	1.67 (0.7)	No
Monocyte % (SEM)	23.(1.0)	4.75 (1.7)	No

Table 3: Effect Of HIVOn TWBC, Neutrophil and Lymphocyte counts

	Control (non-Infected	Test (Infected)	P<0.05
No	37	23	
Age range	0 – 13	0 – 13	
TWBC % (SEM)	9829.4 (871)	11374 (2429)	Yes
Neutrophil % (SEM)	47.9 (3.4)	43.0 (5.7)	No
Lymphocyte % (SEM)	45.7 (3.9)	57.0 (5.7)	Yes

Table 4: Age Related Effect Of HIV Infection

No	Age range	PCV Control	No	HIVONPCV	NO	MPONPCV	P<0.05
27	0–5	39.5(1.8)	7	21.4(3.9)	12	25.5(2.6)	Yes
9	6–13	34.3 (2.1)	2	34.0(0)	3	33.0 (1.0)	No

Table 5: Effect Of MP On Severity Of Anaemia In Children

No	Age range	PCV Control	No	Test PCV%	
23	0 – 5	39.5(1.8)	5	16.7(2.0)	Yes
7	6 – 13	34.3 (2.1)	-	-	No

MP = Malaria parasitaemia

Table 6: Effect Of HIV Co-Infection With Malaria On Blood Indices Of The Only Child

No	Sex	Age (yr)	PCV (%)	ESR (mm/hr)	TWBC(/cu mm)	Neut.(%)	Lymph.(%)
1	F	I ½ r	20	76	13400	23	77

PCV = packed cell volume; ESR = Erythrocyte sedimentation Rate; TWBC = Total white blood cell; Neut = neutrophils; Lymphocytes.

Table 6:Effect of HIV co-infection with malaria

Sex	Contr.	HIV+	P.falci.	HIV+ & P.falci.	% Prev.	% Prev.	% Prev.
Male	20	2	7	-	9.09	25.93	
Female	20	7	3	1	31.82	13.04	4.76
Total	40	9	10	1	16.37	20.00	2.44

Contr. = control gp; HIV + HIV+ Children gp; P.falc. = P falciparum children gp; HIV + & P.falci = co-infected Malaria child.

(1984) and Weatherall (1982) reported that anaemia was an inevitable consequence of malarial infection hence the severe anaemia observed in the 0-years age range (tables 4, 5) and in the one case of HIV co-infection with malaria (table 6) having a PCV of 20% with the report that 90% of HIV/AIDS cases had anaemia of one form or another (Barry et al, 1994; Essien 1993). This pattern observed herein corroborated the hypothesis that chronic latent malarial infection prepares the niche for feeble HIV co-infection and both co-morbid infections are known to decrease natural immunity (Yaffe, 2001). The decreased PCV obtained in both the HIV infected children aged 0 5 years old (table 4) and one HIV co-infected child with malaria (table 6) was consistent with observation of Angyo et al (2000) among children suffering from AIDS at Jos University Teaching Hospital, Jos, Plateau State, Nigeria. The elevated total white blood cell counts (lymphocytosis) (table 3) observed in the HIV asymptomic children agreed with Angyo et al (2000) and confirmed the report that there could be an initial rise in leucocytes and lymphocytes in HIV infections (Bain, 1997). With a 10% prevalence of HIV infection observed, the effects of HIV seropositvity and malaria parasitaemia on packed cell volume (PCV) of these children, it would be of greater consequence to be co-infected with both HIV and malaria. Therefore, it confirms the suggestion that P.falciparium infection increase HIV related immunosuppression and could lead to a higher parasitaemia (Wenike et al, 1990). We observed that a female child aged one-and-half years old co-infected with HIV malaria had a PCV (20%) indicative of a severe anaemia; ESR (76mm/ hr) suggesting the presence of an active disease capable of the release of reactive proteins and, or gamma globulins (Bulls and Brailsford, 1975); a total WBC count (13,400cell/cubic mm) (lymphocytosis) with 23% of neutophils (neutropenia) and 77% of lymphocytes (lymphocytosis) (table 6) and hence agreed with agreed with the postulations of Bain (1997); Murphy et al, (1987); and Davis & Zauli, (1992). This is probably a condition in which malaria increased HIV load in this HIV infected child (Kublin et al, 2005; Whithworth and Hewitt, 2005; Mera/Africa Health 2005), and suggests that HIV co-infection with malaria in Sub-Saharan Africa endemic in both diseases may in for increased mortality and morbidity rates. We conclude that HIV and malaria in children given our results in the 0 5 years age range in particular could be deliberating and is capable of worsening their mortality rates.

REFERENCES

Angyo I.A, Okpeh ES, Mandong B and Onah, J (2000). Infections in Children with AIDS at the JUTH, Jos, Nigeria. Nig. Med. Pract. **38**(4-6): 15-17.

Bain BJ. (1997). Review: The Haematological features of HIV infection. Brit. J. of Haematol. **99**: 1-8.

Barry M, Howe JC, Black DJ, Swart AM, Breckenbridge AM, Weller IV, Beeching N and Nyc F. (1994). Zidovudine Pharmacokinetics on zidovudine Induced boen marrow toxicity. Brit. J. Clin. Pharmacol. **37**: 7-12.

Bull BS & Brailsford JD. (1972). Zeta sedimentation ratio. Blood **40**: 550 557. Charterjee KD. (1976). Parasitology, Helminthology and Protozoology. Eleventh Edition, Calcutta. 72-92pp.

Dacie JV, and Lewis SM. (1991). Practical Haematology. Seventh Edition. Churchill Livingstone, London. 33-72pp.

Davis BR and Zauli G. (1995). Effects of HIV infection on haematopoiesis. Ballier's Clin. Haematol. **8**: 113-117.

Ekunwe E.O.A (1996). Anaemia prevalence in selected population subgroups Nig. Quart. J. Hosp. Med. **6**(1): 23-26.

Ekvall H. (2003). Malaria and anaemia. Curr. Opin. Hematol. **10**(2): 108-114.

Eriksson B, Hellgren U and Rombo L.(1989). Changes in erythrcyte sedimentation rate, C reactive protein and hematological parameters in patients with acute malaria. J. Infect. Dis. **21**(4): 434-441.

Essien E.M. (1993). Infection Associated Haematological disorders. Haematological Trends. **93**: 170. Schattauer, New York.

Jakobsen PH, Bate CA, Taverne J. et al (1995). Malaria: Toxins, cytokines and disease. Parasite Immunol. **17**: 223-231.

Kublin JG et al. (2005). Effect of Plasmodium falciparum malaria on concentration of HIV 1 RNA in the blood of Adults in rural Malawi: a prospective cohort study. Lancet. **356**:233-240.

Mc Gregor IA. (1984). Epidemiology, malaria and pregnancy. Am J. Trop. Med. Hyg. **33**:517-525.

MERA/Africa Health. (2005). Malaria and HIV load In: Medicine Digest, Mera/Africa Health, **27**(4): 30(May).

Murphy MF, Metcalfe P, Waters AA, Carne CA, Weller IVD, Linch DC and Smith A. (1987). Incidence and mechanism of neutropenia on thrombocytopenia in patients with HIV. Brit. J. Haematol. **66**: 337 340. Nand N, Aggarwal H, Sharma M, ad Singh M. (2001). Systemic Manifestations of Malaria J. Ind. Acad. Of Clin. Med. **2**(3): 1-9.

O'Dempsey T. (2002). Malaria in Children. Africa Health (Suppl). 21 (Sept).

Sharma A, Sharma SK, Gover AK, Tewari AA, and Abriol P. (1985).

Anaemia in Protein Energy Malnutrition. Ind. J. Paediatr. **22**: 841-844.

Uko EK, Useh MF, and Ekere EF. (1996). The impact of asymptomatic Malaria on some haematological parameters in Calabar. J. Med. Lab. Sci. **5**: 17-20.

Usanga EA. (1983). Preliminary report on iron metabolism changes in P.falciparum malaria. East Afr. Med. J. **60**: 612 - 615.

Walker N, Schwartlander B, and Bryce J. (2002). Meeting International Goals inchild Survival and HIV/AIDS. Lancet **360**(9329): 284-289.

Weatherall M. (1982). The anaemia of P.falciparum malaria. Brit. Med. Bull. **38**: 147-151.

Weinke T, Schere W, and Pohle HD. (1990). Malaria tropica in HIV Infection Klin. Wochenschr. **68** (10): 533-536.

Withworth JAG and Hewitt K.A. (2005). Effect of malaria on HIV 1 Progression and transmission. Lancet 365: 196-197 (Comment). Xia L, Owen SM, Rudolph DL, Lal RB, and Lal AA. (1998).

Plasmodium falciparum Antigen induced Human Immunodeficiency Virus Type 1 Republication is mediated through induction of Tumour Necrosis Factor 2. J. Inf. Dis. 177:437-445.

Yaffe I. (2001). AIDS possibly caused HIV and latent malaria Coinfection. Med. Hypotheses 57(4): 419-422.

Young SW, Altes BP. (1994). Aplastic Anaemia, acquired and Inherited. Am. J. Trop. Hyg. 49(5): 574-580.

Zeigler JL (1989). Increased in Immunological competent cells, Lymphocytes, plasma cells, kuppfer cells and splenic histocytes in Malaria infection. Brit. Med. J. 5: 15-20.