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IMPACT OF MALARIA AND HIV CO-INFECTION ON THE HEMATOLOGICAL AND IMMUNOLOGICAL PARAMETERS OF HIV-POSITIVE WOMEN IN ABIDJAN (CÔTE D'IVOIRE)

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ABSTRACT

Our study aimed to study the behavior of hematological and immunological parameters in women co-infected with malaria and HIV / AIDS. This was a comparative study over a period of one (1) year from January 2017 to December 2018. It was conducted in the Integrated Center for Bioclinical Research Abidjan (CIRBA). This study included 75 non-malarial HIV-positive women and 50 HIV-positive women with malaria.
The results indicated that erythrocyte level, anemia was very common in 70% of HIV-positive women compared to 57,3% in HIV-positive non-malarial women. Thrombocytosis was only present in HIV-infected women with 40% malaria. The prevalence of neutropenia was observed in 68% of HIV-infected women with malaria compared with 40% of women with non-malarial HIV. Eosinophilia was reported in 36% of HIV-positive women compared with 17,3% of women in non-malarial seropositive women. As for lymphopenia, it was revealed in 22% of HIV-positive women compared to 2,7% of women with non-malarial HIV. According to CD4 levels, a high prevalence of immunodeficiency was observed in 36% of HIV-positive women compared to 10,7% of women in non-malarial seropositive women.
This study shows that co-infection with HIV / AIDS has led to a very significant decrease in certain haematological and immunological parameters in co-infected women.

Keywords: Co-infection; malaria ; HIV; hematological and immunological parameters; Abidjan, Côte d'Ivoire).

1. INTRODUCTION

Malaria and AIDS are the major endemics of our century. According to WHO estimates in 2015[1], there were 214 million malaria cases and 438,000 deaths in the world, with a clear predominance in sub-Saharan Africa, where 88% of global malaria cases and 90% of deaths from malaria have occurred [2]. According to a UNAIDS report in 2017[3], 36,5 million people were living with HIV with 940000 deaths.
Their association remains a major public health problem with adverse consequences for the health of the world's population. They are the leading causes of mortality in the world [4]. The reciprocal interaction between malaria and HIV infection has been well documented since the 1990s. Most of the studies that have shown an interaction of these two pathologies have been performed in

pregnant women, especially African women ([5] ;[6]; [7];[8];[9] ; [10];[11];[12];[13]). According to some of these authors, malaria is more common and more severe in HIV-infected patients, in whom it increases HIV replication and immunosuppression.
In Côte d'Ivoire, like other Sahelian countries, is regularly confronted with these two major scourges. It remains one of the most affected countries in the West African subregion by HIV, with a prevalence of 2,7% [14]. For malaria, according to the most recent data from the World Health Organization [1], it had about 6,4 million suspected cases, 4,7 million suspected and confirmed cases, and 2069 reported deaths. Despite the high prevalence of these two major conditions, most studies focused on the prevalence of malaria and HIV co-infection ([15];[16];[17] ; [18]).
As the main targets of these two pathologies are certain hematological and immunological parameters, it seemed appropriate to study their impact on the hematological and immunological parameters in co infected women.
It is therefore important to evaluate and compare the hematological and immunological parameters between HIV-positive women with malaria and non-malarial HIV-infected women. It will study the impact of HIV and malaria co-infection on the erythrocyte, thrombocyte, leukocyte and immunological parameters in these two groups of women.

2. MATERIEL AND METHODS

2.1. Sites and study population

This is a comparative study over a 1-year period from January 2017 to December 2018. The willing study population was represented by 75 non-malarial HIV-positive women and 50 HIV-positive women with malaria. These women had an axillary temperature above 37 ° C and a thick positive or non-positive.
They were recruited after obtaining authorization from the authorities of the CIRBA (Integrated Center for Bioclinical Research Abidjan) and their consent. They were all followed in this di center.
These HIV-positive women were screened through three types of HIV serology tests (Determine Test, STAT-PAK and Bioline Test (HIV-1 / HIV-2).) The diagnosis of malaria was made by observation of the parasites haematozoa on the May-Grünewald and Giemsa stained blood smear and blood smear (MGG). The analysis of the blood count (NFS) and the counting of the $CD4^+$ lymphocytes were also done.

2.2. Blood samples and assays of biological parameters

In each of the women recruited, two types of blood samples collected in dry tubes and purple tubes of 5 ml each were made. Whole blood collected on purple tubes with anticoagulant (EDTA) allowed the determination of CD4 (by flow cytometry with Fascalibur®), NFS (Blood Formula Count) by an automaton Sysmex Xt 2000i and the thick drop (QBC). The recovered blood in the dry was centrifuged at 1107 Newton for five minutes. The serum obtained in the dry tubes was used to determine HIV serology using three tests : Determine, Stat Pak and Bioline HIV-1 / HIV-2.

2.3. Statistical analysis of biological parameters studied

The statistical analysis of the data was performed by the computer program Statsoft Statistica version Windows 7.1 [19]. It made it possible to compare the averages of the social characteristics and the hematological and immunological parameters by analysis of variances (Anova 1).
The different observed proportions of social characteristics, HIV types, and hematological and immunological parameters were compared by the G-likelihood test or log-like-ratio ratio with the Windows-based R.2.0.1 software [20]. This proportion comparison was conducted between non-malarial HIV-positive women and HIV-positive women with malaria. The difference was considered statistically significant if p was less than or equal to 0,05.

3. RESULTS

3.1. Social characteristics and types of HIV

The mean age in this study was 31,7 years for HIV-positive non-malarial women and 27,6 years for HIV-positive women.
Women aged 18 and 19 were fewer. However, they were significantly more numerous among malarias (22%) than non-malarias (9%).
The majority of women were adults. They accounted for 90,7% of the number of HIV-positive non-malnourished women compared to 88% for HIV-positive women.
The mean body mass index was approximately equal for the two groups of women (25,2 kg / m^2 for HIV-positive non-malarial women and 24,4 kg/m^2 for HIV-positive women). The abnormal body mass index was significantly higher ($p < 0,05$) in HIV-positive women with a prevalence of 18% compared to 6,7% among HIV-positive non-malarious women. On the other hand, no significant difference ($p > 0,05$) was observed for the prevalences of normal and above normal body mass index between these two groups of women. The women in this study had a good level of education with 11,2% of illiterates overall. In this study, HIV-1 was the most prevalent type in both HIV-positive non-malarial women (86,7%) and HIV-positive women (92%). Non-malarial women with HIV-2 (10,7%) outnumbered HIV-2 malarious women (6%) without any significant difference. Only 2,6% of non-malarial women and 2% of malarial women were co-infected with HIV-1 and 2 (Table I).

Table I : Social characteristics and HIV type of the study population.

Characteristics and type of HIV	Non-malarial women HIV+ (N=75)			Malarial women HIV+ (N=50)			p
	n	M±SEM	%	n	M±SEM	%	
Age (years)		**31,7 ± 0,9**			**27,6 ± 0,9**		**0,49(NS)**
18-19	7		**9,3**	6		**22**	0,02 (S)
20-45	68		**90,7**	44		**88**	0,84 (NS)
Body mass index (kg.m-2)		**25,2 ± 0,5**			**24,4 ± 0,7**		**0,98 (NS)**
< 19,8	5		**6,7**	9		**18**	0,02 (S)
19,8 – 26	43		**57,3**	26		**52**	0,61 (NS)
> 26	27		**36**	15		**30**	0,46 (NS)
Education attainment							
Illettrées	8		**10,7**	6		**12**	0,78 (NS)
Primaire	30		**40**	21		**42**	0,05 (NS)
Secondaire	24		**32**	15		**30**	0,80 (NS)
Supérieur	13		**17,3**	8		**16**	0,82 (NS)
Types of HIV							
HIV-1	65		**86,7**	46		**92**	0,69 (NS)
HIV -2	8		**10,7**	3		**6**	0,24 (NS)
HIV -1 et 2	2		**2,6**	1		**2**	0,08 (NS)

N: Total number of women; n: Number of observed in each woman's group; M: average; SEM: Standard error on average; %: Percentage; S: Difference Statistically significant for p <0,05; NS: Difference not statistically significant for p> 0,05.

3.2. Distribution of erythrocyte and thrombocyte parameters

The analysis of the mean values in Table II shows that a significant difference was observed only between the average thrombocyte values of the two groups of women. This rate was significantly elevated (p <0,0001) in HIV-positive women in women who were seropositive with non-malarial (558,8 10^3/mm^3 vs 272,2. 10^3/mm^3). Conversely, the mean values of erythrocyte parameters tested were statistically not different (p> 0,05) between the two groups of selected women. In this same table, for all the proportions of erythrocyte parameters and thrombocytes, with the exception of the values of the normal thrombocyte level (150-500 10^3/mm^3) and the abnormal higher values of the red blood cell count (>5,4. 10^{12}/L) and thrombocytes (>500. 10^3/mm^3), no statistically significant difference (p> 0,05) was observed between the proportions of the other parameters. However, the prevalence of abnormal values of these parameters was higher in HIV-positive women than in HIV-positive non-malarial women.

Table II : Mean values and proportions of erythrocyte and thrombocyte parameters

Erythrocyte and thrombocyte parameters	Non-malarial women HIV+ (N=75)			Malarial women HIV+ (N=50)			p
	n	M±SEM	%	n	M±SEM	%	
Red blood cell counts (10^{12}/l)		**4,06 ± 0,08**			**3,9 ± 0,07**		**0,95 (NS)**
< 4	34		**45,3**	33		**66**	0,05 (NS)
4-5,4	37		**49,3**	17		**34**	0,09 (NS)
> 5,4	4		**5,3**	-		**-**	0,007 (S)
Hemoglobin(g/dL)		**11,6 ± 0,2**			**11,1 ± 0,2**		**0,91 (NS)**
< 12	43		**57,3**	35		**70**	0,26 (NS)
12-16	32		**42,7**	15		**30**	0,14 (NS)

Hematocrit (%)		**36 ± 0,6**			**34,7 ± 0,6**	**0,88 (NS)**
< 35	23		**30,7**	22	**44**	0,12 (NS)
35-47	52		**69,3**	28	**56**	0,23 (NS)
MCV (fL)		**89,3 ± 1,2**			**90,3 ±1,8**	**0,94 (NS)**
< 85	25		**33,3**	13	**26**	0,34 (NS)
85 – 95	30		**40**	20	**40**	1 (NS)
> 95	20		**26,7**	17	**34**	0,35 (NS)
MCH(pg)		**28,9 ±0,4**			**28,9 ±0,7**	**1 (NS)**
< 27	20		**26,7**	16	**32**	0,35 (NS)
27-31	36		**48**	19	**38**	0,28 (NS)
> 31	19		**25,3**	15	**30**	0,53 (NS)
MCHC (g/dl)		**32,1 ± 0,2**			**31,9 ± 0,2**	**0,98 (NS)**
< 32	32		**42,7**	26	**52**	0,34 (NS)
32-36	41		**54,7**	23	**46**	0,39 (NS)
> 36	2		**2,7**	1	**2**	0,74 (NS)
Thrombocyte (10^3/mm^3)		**272,2 ± 9,9**			**558,8 ± 77,1**	**$2,2.10^{-16}$ (S)**
< 150	10		**7,5**	6	**12**	0,31 (NS)
150-500	65		**92,5**	24	**48**	0,0002 (S)
> 500	-		**-**	20	**40**	$9,6.10^{-14}$ (S)

N: Total number of women; n: Number of observed in each woman's group; M: average; SEM: Standard error on average; %: Percentage; MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; MCHC: Mean corpuscular concentration of hemoglobin concentration ;S: Difference Statistically significant for p < 0,05; NS: Difference not statistically significant for p > 0,05.

3.3. Distribution of leukocyte parameters and CD4 count

Mean values and proportions of leukocyte parameters and $CD4^+$ are shown in Table III. The analysis of the comparison of mean values showed that the mean value of $CD4^+$ in non-malarial women was significantly higher ($p = 3,55. 10^{-10}$) than the mean for malarial women (457 ± 30,4/ mm^3 vs 286,7 ± 25,8/mm^3). On the other hand, no significant difference (p> 0,05) was observed between the average values of leukocyte parameters between these two groups of women.

The analysis of the proportions of these parameters showed very significant statistical differences (p>0,01), highly significant (p>0,0001) abnormal leucocyte values (<10.10^3/mm^3), polymorphonuclear neutrophils (<40%), eosinophilic polynuclear cells (<1%), lymphocytes (<15%) and CD4 + lymphocytes (<200/mm^3 and ≥ 499/mm^3) between these two groups of women. In addition, a highly significant difference was observed between the prevalences of neutrophil polymorphonuclear normal values (40% -70%) of these two groups of women. Overall, all of these prevalences of abnormal leukocyte parameters and CD4 counts were significantly higher in HIV-positive women than in non-malaria seropositive women.

Table III : Mean values and proportions of leukocyte parameters and CD4 +

Leukocyte and $CD4^+$ parameters	**Non-malarial women HIV+ (N=75)**			**Malarial women HIV+ (N=50)**			**p**
	n	**M±SEM**	%	n	**M±SEM**	%	
Leukocytes (10^3/mm^3)		**5,8 ± 0,4**			**4,8 ± 0,2**		**0,7 (NS)**
< 4	22		**29,3**	16		**32**	0,7 (NS)
4 – 10	48		**64**	34		**68**	0,7 (NS)
< 10	5		**6,7**	-		**-**	0,002 (S)
Polynuclear N (%)		**46, 2 ± 1,6**			**32,4 ± 2,7**		**0,1 (NS)**
< 40	30		**40**	34		**68**	0,007 (S)
40–70	42		**56**	14		**28**	0,002 (S)
> 70	3		**4**	2		**4**	1 (NS)

Polynuclear E (%)		**2,8 ± 0,3**			**3,1 ± 0,6**	**0,9 (NS)**
<1	13	**17,3**	18		**36**	0,01 (S)
1–5	52	**69,3**	25		**50**	0,08 (NS)
> 5	10	**13,3**	7		**14**	0,9 (NS)
Polynuclear B (%)		**0,4 ± 0,04**			**0,4 ± 0,04**	**1 (NS)**
0-1	71	**94,7**	49		**98**	0,8 (NS)
> 1	4	**5,3**	1		**2**	0,2 (NS)
Lymphocytes (%)		**41,7 ± 1,4**			**36,2 ± 3**	**0,5 (NS)**
< 15	2	**2,7**	11		**22**	$3,37\ 10^{-5}$ (S)
15–40	25	**33,3**	12		**24**	0,2 (NS)
> 40	48	**64**	27		**54**	0,4 (NS)
Monocytes (%)		**8,6 ± 0,4**			**8,9 ± 0,7**	**0,9 (NS)**
2 –10	52	**69,3**	28		**56**	0,2 (NS)
> 10	23	**30,7**	22		**44**	0,1 (NS)
Lymphocytes TCD4 (/mm^3)		**457± 30,4**			**286,7 +25,8**	**$3,55.10^{-10}$(S)**
< 200	8	**10,7**	18		**36**	0,0001(S)
200 – 499	44	**58,7**	26		**52**	0,5 (NS)
≥ 499	23	**30,6**	6		**12**	0,004 (S)

N: Total number of women; n: Number of observed in each woman's group; M: average; SEM: Standard error on average; %: Percentage; ; N : Neutrophils ; E : Eosinophils ; B : Basophils ; S: Difference Statistically significant for p < 0,05; NS: Difference not statistically significant for p > 0,05.

4. DISCUSSION

HIV-malaria co-infection is a public health problem. It is the leading cause of death in the world in general, and particularly in sub-Saharan Africa, which includes Côte d'Ivoire [1]. However, most studies in Côte d'Ivoire focus on the prevalence of HIV infection ([15] ; [16] ; [17] ; [18]). However, co-infection with HIV-malaria is thought to be responsible for some hematologic and immunological manifestations observed during follow-up of HIV-positive patients ([21] ; [12] ; [22]).

So, to verify this situation in Côte d'Ivoire, a study on the variation of the biological blood parameters in 125 women living with HIV at the Abidjan Integrated Center for Bioclinical Research (CIRBA) was conducted. These women were composed of 75 non-malarial HIV-positive women versus 50 HIV-positive women. The distribution of these women by age showed that age averages were 31,7±0,9 years for non-malarial seropositive women versus 27,6±0,9 years for those with malaria. These results are below the average age reported by Aba *et al.*[23]. The average age observed by these authors in the women in their study was 38±8,3 years. Indeed, women who participated in the studies of Aba *et al*. [23]were all adults with an age between 30 and 40 years old. Unlike their studies, although dominated by the presence of adult women (90,7% of non-malarial seropositive women versus 88% of malaria-positive women) in our study, there was the presence of some adolescent girls. The prevalence of these HIV-positive young women with malaria was significantly higher than that of non-malarial HIV-positive women (22% versus 9,3%).

The educational attainment of women in this study was generally acceptable. Only 10,7% of non-malarial HIV-positive women compared with 12% of HIV-positive women were illiterate.

Regarding HIV serology, HIV-1 was the most prevalent type with 86% in non-malarial seropositive women compared to 92% in those withmalaria. These high prevalences are similar to those observed by Aba *et al*. [23]who observed 86% of the type of HIV-1. This predominance of HIV type 1 is explained by the fact that HIV type 1 is the predominant type in sub-Saharan Africa [24].

In this study, the variation in blood biological parameters was evaluated between these two groups of women. Our results show that the blood parameters of HIV-positive women have undergone significant alteration compared to non-malarious women living with HIV. In fact, anemia was observed in 70% of HIV-positive women compared to 57,3% of women with HIV who were not malaria-positive. Given these results, there was no statistically significant difference between these two groups of women.These same findings were reported by Yitayih *et al*. [12]who observed respectively 65,8% of cases of anemia. On the other hand, those observed by Baldé *et al*. [9]at Donka National Hospital (Conakry) and Sanyaolu *et al*. [22]in Nigeria were lower than ours. These authors found respectively in their study 56,1% and 25,8% of cases of anemia.

Co-infection is thought to be the cause of this high prevalence of anemia among co-infected women in our study. Indeed, anemia is caused by several factors. In malarial women, intravascular haemolysis of parasitized red blood cells would be responsible for this. Furthermore, in the context of HIV infection, chronic inflammation, micronutrient deficiency, and opportunistic infections are also thought to be the cause of anemia in our patients ([25] ; [26] ; [27] ; [28] ; [29]).

In this study, the prevalence of thrombocytopenia was high in malaria-positive women compared to non-malarial seropositive women. This thrombocytopenia could be explained according to Delabesse *et al.* [30]by a peripheral hyperdestruction of platelets during viral infections including HIV. In addition, Wachtman *et al.*[31]showed that thrombocytopenia may be caused by a decline in thrombocyte levels related to atrophy of different brain regions in some HIV-positive people (neurosida).

Among the leukocyte parameters, the prevalence of neutropenia, eosinophilia, and lymphopenia were significantly higher in HIV-positive women than in non-malarial HIV-infected women. In fact, with regard to CD4 + levels, immunodeficiency was observed in 36% of HIV-positive women compared to 10,7% in non-malarial HIV-positive women. Our results corroborate those of Yitayih *et al*. [12]in Ethiopia with 34,3% of cases of immunodeficiency.

According to some authors, this immunodeficiency is due to malaria that would activate CD4 cells. This environment would promote the entry of the virus into CD4 cells and stimulate viral replication [32]. This activation would accelerate the destruction of CD4 cells and lead to more rapid disease progression [33].

5. CONCLUSION

The study conducted in women co-infected with malaria and HIV and those who were HIV-positive, evaluated changes in this co-infection on hematological and immunological parameters. These hemato-immunological changes mainly concerned hemoglobin, thrombocyte, leucocyte and CD4 + lymphocyte levels. This study found high prevalence of anemia, neutropenia, eosinophilia, lymphopenia and immune deficiency in co-infected women compared to non-malarial HIV-positive women.
This work should be continued by characterizing the erythrocyte and leukocyte parameters according to the different stages of CD4 +. And also, assess the prevalence of different types of anemia depending on the progression of HIV infection.

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Authors contributions

All authors contributed equally in the study. They made substantial contributions to the design of the study, the collection of the data as well as the preparation and analysis of the data. They also drafted the manuscript and gave final approval for its submission to the journal for consideration of publication.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

REFERENCES

[1] OMS, 2015. Rapport sur le paludisme dans le monde. www.who.int/malaria/publications/world-malaria...2015/.../fr...Consulté le 26/11/2018.
[2] OMS, 2016. Paludisme. www.who.int/mediacentre/factsheets/fs094/fr. Consulté le 15/11/2018
[3] UNAIDS, 2017. Fiche d'information-Dernières statistiques sur l'état de l'épidémie de sida. www.unaids.org. Consulté le 28 /10/2018.
[4] Good M.F., Doolan D.L., 1999. Immune effector mechanisms in malaria. *Current Opinion Immunology* , 11: 412-9.
[5] Butcher G.A., 1992. HIV and malaria : a lesson in immunology ? Parasitol Today ;8:3071
[6] Verhoeff F.H., Brabin B.J., Hart C.A., Chimsuku L., Kazembe P., Broadhead R.L., 1999. Increased prevalence of malaria in HIV-infected pregnant women and its implications for malaria control. *Tropical Medecine and International Health*, 4: 5-12.
[7] Ayisi J.G., van Eijk A.M., ter Kuile F.O., 2003. The effect of dual infection with HIV and malaria on pregnancy outcome in western Kenya. *AIDS* ; 17: 585-94.
[8] Van Geertruyden J.P., Mulenga M., Kasongo W., Polman K., Colebunders R., Kestens L., D'Alessandro U.,2006. CD4 T-cell count and HIV-1 infection in adults with uncomplicated malaria. *Journal of Acquired Immune Deficiency Syndromes*, 43: 363-7.
[9] Baldé I., Camara A., Baldé O., Magassouba N'F., Bah M.S., Makanéra A., Gamy E.P., 2010. Paludisme et infection à VIH : aspects cliniques et biologiques à Conakry (Guinée)**.** *Medecine Tropicale*, 70: 349-352
[10] Martin-Blondel G., Soumah M., Camara B., *et al.*, 2010. Impact of malaria on HIV infection. *Medecine et Maladies Infectieuses*, 40: 256-67.
[11] Longdoh A.N., Henri-Lucien F.K., Dickson S.N., Jules-Clement N.A., Tebit E.K., 2012. Low Malaria Prevalence in HIV-Positive Patients in Bamenda, Cameroon. *Journal of Microbiology Research*, 2(3): 56-59.
[12] Yitayih W., Getachew F., Asmamaw A. and Dagnachew M., 2013. HIV-Malaria Co-infection and their immunohematological profiles.*European Journal of Experimental Biology*, 3(1):497-502.
[13] Tebit E Kwenti, 2018. Malaria and HIV. Coinfection in sub-Saharan Africa: prevalence, impact, and treatment strategies. *Research and Reports in Tropical Medicine*, 9 123–136.
[14] ONUSIDA, 2016. Stratégie de coopération *http://* www.who.int/countries/en/. Consulté le 20/11/2018.
[15] Eholié S.P., Ehui E., Adou-Bryn K., Kouamé K.E., Tanon A., Kakou A., Bissagnené E., Kadio A., 2004. Paludisme grave de l'adulte autochtone à Abidjan(Côte d'Ivoire). *Bulletin de la Société de Pathologie Exotique*, 97 (5): 340-344
[16] Aoussi E.F., Tanon K.A., Ehui E., Ouattara S. I., Inwoley K.A., Adoubryn K.D., Eholie S.P., Bissagnené E.,2011. Malaria and HIV infection in sub Saharan Africa : Another match made in Hell ? *Sante*, 21 (3): 174-177.
[17] Kra O., Ouattara B., Aba T., Kadjane´ N.J., Kadjo K., Bissagnéné E., Kadio A., 2012. Morbimortalité des pathologies infectieuses à l'hôpital militaire d'Abidjan, Côte d'Ivoire. *Médecine et Santé Tropicales* ; 22: 75-78.
[18] Harouna A. M., Amorissani-Folquet M., Eboua F.T., Desmonde S., N'Gbeche, S., Aka E.A., Kouadio K., Kouacou B., Malateste K., Bosse-Amani C., Ahuatchi C.P., Leroy V., 2015. Effect of cotrimoxazole prophylaxis on the incidence of malaria in HIV-infected children in 2012, in Abidjan, Côte d'Ivoire. *BMC Infectious Diseases*, 15:317.

[19] Statsoft, (2005).Statistica (Data Analysis Software System). Version 7.1 available at www. Statsoft.com
[20] Ihaka R., Gentleman R., (1996). R: a language for data analysis and graphics. *Journal computational and graphical statistics*, 5: 299-314.
[21] Saracino A., Nacarapa E.A., Massinga É.A.C., Martinelli D., Scacchetti M., Oliveira C., Antonich A., Galloni D., Ferro J.J. and Macome C.A., 2012. Prevalence and clinical features of HIV and malaria co-infection in hospitalized adults in Beira, Mozambique. *Malaria Journal*, 11 :241
[22] Sanyaolu A.O., Fagbenro-Beyioku A.F., Oyibo W.A, Badaru O.S., Onyeabor O.S., Nnaemeka C.I., 2013. Malaria and HIV co-infection and their effect on haemoglobin levels from three healthcare institutions in Lagos, southwest Nigeria. *African Health Sciences;* 13(2).
[23] Aba T.Y., Moh R., Cissé L., Yapo-Kouadio G. C., Ello F. N., Mossou C., Diallo Z., Kra O., Bissagnené E., 2016. Plasmodium falciparum malaria in northern Côte d'Ivoire : prevalence in thegeneral hospital of Tanda sanitary district. *Malaria World Journal*, 7 (1): 2214-4374.
[24] Amat-Roze J.M., 1989. L'infection à V.I.H. et le SIDA en Afrique noire : facteurs d'épidémisation et de régionalisation. In : Cahiers d'outre-mer. N° 168 - 42e année, Octobre-décembre. pp. 333-355.
[25] Katodriitou E., Christakis J., 2006. Recent advances in the the pathogenesis and management of anaemia of chronic disease. *Haematology*. 9 (1):45-55.
[26] Drain P.K., Kupka R., Mugusi F., Fawzi W.W., 2007. Micronutrients in HIV-positive persons receiving highly active antiretroviral therapy. *American Journal of Clinical Nutrition*, 85: 333-45.
[27] Tamez P.A., Liu H., Fernandez-Pol S., Haldar K., Wickrema A., 2009. Stage-specific susceptibility of human erythroblasts to Plasmodium falciparum malaria infection. *Blood*, 114(17) :3652-5.
[28] Rayner JC (2009). The merozoite has landed: reticulocyte-binding-like ligands and the specificity of erythrocyte recognition. *Trends Parasitology*. 25 (3):104-6.
[29] Kazadi M., Kabongo K., Kassamba I., Bilonda M., Mundongo T., Mwembo T., Balaka E.M., Kazadi M.C., Kasamba I.E., Djouma J., Watu W.C., Mujing A.M.F., Balaka E.M., Kabongo J., Mundongo H., Kalombo M., Wembonyama S., Kalenga M.P., Mwembo-Tambwe A.N.A., 2014. Etude de l'anémie chez les enfants séropositifs au VIH naïfs au traitement antirétroviral à Lubumbashi, République Démocratique du Congo. *Pan African Medical Journal*, 17: 46.
[30] Delabesse E., Corre J., Ysebaert L., Laharrague P., Laurent G., 2010. Sémiologie Hématologique.51-53. htt://www.medecine.upstlse.fr/dcem1/semio_hemato/semio_hemato.pdf. Consulté le 23 Aout 2017.
[31] Wachtman L., Skolasky R., Tarwater P., 2007. Platelet decline : an avenue for investigation into the pathologenesis of human immunodeficiency virus-associated dementia. *Archives of Neurology*, 64(9):1264-72.
[32] Worku S., Björkman A., Troye-Blomberg M., *et al*.,1997. Lymphocyte activation and subset redistribution in theperipheral blood in acute malaria illness: DisctinctgammadeltaгT cell patterns in Plasmodium falciparumand P. vivax infections. *Clinicaland Experimental Immunology*, 108:34-41.
[33] Whitworth J.A., Hewitt K.A., 2005 Effect of malaria onHIV-1 progression and transmission. *Lancet*;365:196-7.