Open Access Library Journal
2017, Volume 4, e3697
ISSN Online: 2333-9721
ISSN Print: 2333-9705

Seroprevalence Virus in Human Immunodeficiency Sickle Cell Anemia Duffy-46C/C in Democratic Republic of Congo

—Case of the City of Kinshasa and Lubumbashi

P. Lungu Anzwal[1], A. Kambote Kalumba[1], J. Kasali Mwamba[2], Saragosti S. Sentob[3], H. Situakibanza Nani Tuma[4], Ekwalanga M. Balaka[1]

[1]Department of Medical Biology, Faculty of Medicine, University of Lubumbashi, Lubumbashi, Democratic Republic of Congo
[2]Provincial Laboratory for the Program to Fight HIV/AIDS, Lubumbashi, Democratic Republic of Congo
[3]INSERM U941, University of Paris Diderot, Paris, France
[4]Department of Internal Medicine, Department of Tropical Medicine, Infectious and Parasitic Diseases, Faculty of Medicine, University of Kinshasa, Kinshasa, Democratic Republic of Congo
Email: michelnzaji@yahoo.fr

How to cite this paper: Anzwal, P.L., Kalumba, A.K., Mwamba, J.K., Sentob, S.S., Tuma, H.S.N. and Balaka, E.M. (2017) Seroprevalence Virus in Human Immunodeficiency Sickle Cell Anemia Duffy-46C/C in Democratic Republic of Congo. *Open Access Library Journal*, **4**: e3697.
https://doi.org/10.4236/oalib.1103697

Received: May 31, 2017
Accepted: July 17, 2017
Published: July 20, 2017

Abstract

Homozygous sickle cell disease undergoes multiple blood transfusions during their life because of chronic hemolytic anemia associated with their condition and the risk of infection is most convincing, for example vis-à-vis certain viruses such as HIV, the virus hepatitis. This study aims to determine the HIV seroprevalence among sickle cell anemia Duffy-46C/C. This is a cross-sectional descriptive study to determine HIV seroprevalence in Duffy-46C/C sickle-cell anemia, conducted at the Yolo Sud SS Joint Medical Center in St. Crispin in Kinshasa and Jason Sendwe Hospital in Lubumbashi, from 2015 to 2016. HIV seroprevalence of 505 subjects homozygous sickle cell Duffy-46C/C was established by Immuno-chromatography tests determined HIV1/2 test Uni-Gold to determine the anti-HIV antibodies. CD4 + rate was also determined and plasma viral load. We collected 505 samples of blood from sickle cell anemia aged from 1 year to 65 years (mean age 15.6 years) with 235 subjects male and 270 female subjects. A positive serology was found in 6 cases (1.19%), including 5 female subjects of which 4 cases had received 1 - 5 transfusions and 2 cases were vaso-occlusive crisis (VOC). Viral load was determined, and 4 individuals were detectable and 2 were undetectable. Their CD4 + levels were raised to more than 500 c/μL, ranging between 543 to 893 c/μl. The blood transfusion remains a risk in the transmission of HIV/AIDS among people polytransfusees, due probably to the window door. We will have to strengthen blood safety by equipping laboratories and blood banks with reliable early tests that detect viral RNA of HIV.

Subject Areas

Public Health

Keywords

HIV, Sickle Cell Anemia Duffy-46C/C, Blood Transfusion

1. Introduction

Sickle cell disease or sickle cell anemia is a common genetic disorder, autosomal recessively. The first medical description was made 1904 by James Herrick. It is characterized by an alteration of hemoglobin, abnormal hemoglobin S whose structure was identified in 1949 by Linus PAULING.

In the Democratic Republic of Congo (including in Lubumbashi), the prevalence of sickle cell trait $\beta A/\beta S$ varies between 20% to 30% [1] and that of sickle cell disease $\beta S/\beta S$ is 1.4% [2].

Sickle cell disease is a major public health problem in the city of Lubumbashi and Kinshasa. In the Democratic Republic of Congo, HIV seroprevalence was 1.1% according to DHS 2013-2014.

Many studies have shown that HIV seroprevalence among sickle cell anemia was low despite multiple blood transfusions they undergo [3] [4] [5] [6].

In our previous work, we have demonstrated the existence of two mandatory mutations in homozygous sickle cell Africans mutation $\beta S/\beta S$ and mutation Duffy-46C/C). During HIV infection, the impact of the sickle cell mutation is predominant with respect to susceptibility to HIV (low susceptibility), and it is the result of the double mutation with respect to the progression of viral infection: slow progression [7].

The aim of our study was to document the susceptibility of HIV in sickle cell anemia Duffy-46C/C in Kinshasa and Lubumbashi.

2. Materials and Methods

The research targeted topics attending care centers sickle cell in the city of Lubumbashi in the general referral hospital Jason Sendwe and in Kinshasa the Joint Medical Center and anemia SS (CMMASS) in Mabanga Yolo South and Polyclinic Saint Crispin in Ngaliema.

The study population consisted of 505 subjects homozygous sickle cell disease including 235 males and 270 females, aged 1 year to 65 years (age moyen 15, 6 years). SS anemia was determined by the Emmel test and confirmed by acid pH hemoglobin electrophoresis on cellulose acetate gel. Inclusion criteria were HIV/AIDS positive serology and exclusion criteria were the presence of other viral infections and children under 1 year. The statistical population in this study consisted of any sickle cell patient who had presented to the selected health facilities. This study was conducted over a period from November 5, 2015 to September 22, 2016.

The study was to determine the seroprevalence of HIV among sickle cell anemia Duffy-46C/C for anti HIV antibody tests Determine *HIV*1/2 *test Uni-Gold, confirmed by measurement of viral load technology Abbott Real Time HIV*1/2 *and determination of CD*4 + *levels. A data collection sheet was established for information on certain variables, including the number of blood transfusion, age, sex, antiretroviral treatment, vaso occlusive crisis (VOC) or not and the mode of transmission HIV.* 5 ml *of whole blood were collected in EDTA anticoagulant and plasma was obtained after centrifugation of* 3000 *revolutions/minute for* 10 *minutes at room temperature,* 25˚C. *The plasma was stored at* −20˚C *for determination of viral load.*

Statistical analyzes were performed with the following tests: Fischer exact test or chi-square, frequency and average. The software Epi Info, SPSS, Excel and Windows 7 *were used. The statistical significance threshold was calculated and defined at* $p < 0.05$.

The limitations of our study were to find homozygous sickle-cell anemia with a positive HIV serology. The authorization of the ethics committee has given us before conducting this study (No. approval: UNILU/EMC/071/2017.

3. Results

The results of HIV seroprevalence are shown in Table 1: six cases of 505 samples were found positive 1.19%.

The results of HIV distribution by age group are shown that the positive cases were found in the age group of 1 to 10 years (3 cases), 11 to 21 years (1 case) and that of 22 to 32 years (2 cases). After these ages no positivity was detected (Table 2).

Table 1. HIV Prevalence homozygous sickle cell Duffy-46C/C.

SEROLOGY	Frequency	Percent
Negative	499	98.81%
Positive	6	1.19%
Total	505	100.00%

Table 2. Distribution of sickle-cell anemia by age.

AGE (years)	Frequency	Percent
1 - 10	182	36.04%
11 - 21	199	39.41%
22 - 32	99	19.60%
33 - 43	15	2.97%
44 - 54	6	1.19%
Greater or equal to 55	4	0.79%
Total	505	100.00%

The results of this table are shown that the majorities were female, 5 out of 6 cases (Table 3).

The frequency distribution according to age groups shows that SCD 199 were in the age group from 11 to 21 years, 199 (39.41%) of 505 (Table 4).

The results in this table show that 70.39% received between 1 and 5 blood transfusions (Table 5).

The number of blood transfusion between 1 to 5 revealed more positive HIV cases, 4 cases of 6 and 466 sickle cell patients had previously received at least one blood transfusion and 39 others had never had a blood transfusion (Table 6).

The results according to the clinical profile indicate that 271 sickle cell patients were CVO crises, among them two positive cases of HIV and 234 others were without crisis four positive cases of HIV at the time of blood collection (Table 7).

The results of viral load shows detectable 4 cases, 3.14 Log, Log 2.43, 5.38 log, 3.60 Log and 2 cases undetectable. Among the detectable 4 cases, 3 were naive to ARV. And 2 cases were undetectable on ART. The determination of CD4 + levels shows high values over 500 c/.mu.l ranging from 543 to 893 c/µl in all sickle cell anemia HIV + (Table 8).

Table 3. Distribution of sickle-cell anemia by sex.

Sex	Frequency	Percent
Male	235	46.53%
Female	270	53.47%
Total	505	100%

Table 4. Positive serology according to age groups.

Age (years)	Total		HIV serology	
			Positive	Negative
1 - 10	182	(36.04%)	3	179
11 - 21	199	(39.41%)	1	198
22 - 32	99	(19.60%)	2	97
33 - 43	15	(2.97%)	0	15
44 - 54	6	(1.19%)	0	6
≥55	4	(0.79%)	0	4
Total	505	(100%)	6	499

Table 5. Frequency allocation according to the number of slices blood transfusion.

SLICE NUMBER TRANSFUSION	Frequency	Percent
1 - 5 transfusions	328	70.39%
6 - 10 transfusions	98	21.03%
sup 10 transfusions	40	8.58%
Total	466	100.00%

Table 6. Positive serology as number of blood transfusions slices.

SLICE NUMBER TRANSFUSION	serology		
	Negative	Positive	Total
1 - 5 transfusions	324 (98.78%)	4 (1.22%)	328 (100%)
6 - 10 transfusions	97 (98.98%)	1 (1.02%)	98 (100%)
sup 10 transfusions	39 (97.50%)	1 (2.50%)	40 (100%)
TOTAL	460 (98.71%)	6 (1.29%)	466 (100%)

Table 7. Distribution of frequencies according to the clinical profile.

CLINICAL PROFILE	Frequency	Percent
In crisis	271	53.66%
without crisis	234	46.34%
Total	505	100.00%

Table 8. Results of viral load of sickle cell anemia Duffy-46C/C.

No. ECH	ARVs	Start / Ttt	CD4 +	PL CV
1	Yes	2006	700 c/.mu.l	Log 3.14
2	Yes	2010	893 c/.mu.l	IND
3	Naive	2015	882 c/.mu.l	Log 2.43
4	Naive	03/2016	543 c/.mu.l	Log 5.38
5	Naive	03/1016	645 c/.mu.l	3.60 Log
6	Yes	2015	734 c/.mu.l	IND

4. Discussion

Our study aimed to document the susceptibility of HIV in sickle cell anemia Duffy-46C/C, show that HIV prevalence is low. These results confirm those found by NGALY *et al.* 1987 [3], or 5.4% versus > 10% by Lukuni *et al.* 1989 [8], or 5.5% vs. > 10%, and Neto *et al.* 2010 [9], (or 0.8% compared to other infections (13.4% HCV, HTLV-1 4.7%, HBV 3.1% and 2.8% Chagga disease) and Tshilolo *et al.* 2010, 2012 [4] [10], which years larger multicenter trials including countries in Central Africa (DRC, Gabon and Cameroon), the risk of viral infections (HIV, HBV, HCV) in sickle cell anemia $\beta S/\beta S$ showed firstly that the prevalence of HIV infection was significantly reduced compared to that of the normal population (16.5% versus 22%) and secondly that they (sickle cell anemia βS / βS) became slow progressors to viral infection. These results corroborate those found by Mehdi *et al.* 2012 [5], 1.5% versus 3.3% and maintain that sickle cell anemia is associated with a lower risk of coinfection with HIV, and this protection against HIV infection may be related to defense immune reinforced in SCD against HIV. These results also similar to those found by Stephen *et al.* 2012 [11] confirming the study Mehdi *et al.* 2012 [5] on the low HIV seroprevalence among African Americans with sickle cell disease, and attest that the low preva-

lence could be related to the inhibition of viral replication of HIV, due to the persistence of inflammation, metabolism Fe immunological changes in sickle cell disease. Our results also confirm those found by Diarra *et al.* 2013 [6], 1% versus 3% 1% HBV and HCV, a study in Mali on the prevalence of HIV, HBV and HCV in sickle cell before and after blood transfusion. Among the positive sickle HIV, 5 of 6 were women and most of the positive cases (4/6) were in the range of transfusion number from 1 to 5. These results corroborate those found by Ngo Sack [12] who showed that all positive patients have already been transfused HIV status increased with the number of blood transfusion.

We also determined the rate of CD4 + and our results reveal CD4 + high rates over the 500 c/µl in all sickle cell anemia Duffy βS/βS HIV +. These results confirm those found by Bagasra *et al.* [13] who pioneered the study of pathophysiological mechanisms in HIV infection in sickle cell anemia βS/βS. Their works based on viral load during infection have shown that AIDS progresses much more slowly in the βS/βS patients.

In tracking a small cohort, the authors showed that 44% of βS/βS patients (8 of 18) were long-term asymptomatic with a high CD4 count and a low viral load. In the control group, only 13.9% (5 of 36) were asymptomatic long-term, although in our study, we noticed that some homozygous sickle cell patients had a viral load a little high. These sickles were naive to antiretroviral treatment and had a recent viral infection. This study opens the door to other opportunities, for example, conduct a study of the pathophysiological mechanisms of the double mutation in homozygous sickle cell βS/βS Duffy-46C/C in order to understand the low susceptibility to HIV in sickle cell or the protective effects vis-à-vis HIV in sickle cell.

5. Conclusions

Most HIV positive sickle cell diseases were infected by blood transfusion, with the exception of a single case, contaminated by mother to child transmission.

The blood transfusion remains a risk in the transmission of HIV/AIDS among people polytransfusees, due probably to the window door. We will have to strengthen blood safety by equipping laboratories and blood banks with reliable early tests that detect viral RNA of HIV.

References

[1] Wembonyama, S., Mpaka, S. and Tshilolo, L. (2007) Medicine and Health in the DRC: From Independence to the 3rd Republic. *Med Trop*, **67**, 447-457.

[2] Tshilolo, L., Aissi, L.M., Lukusa, D., Kinsiama, C., Wembonyama, S., Gulbis, B. and Vertongen, F. (2009) Neonatal Screening for Sickle Cell Anemia in the Democratic Republic of Congo: Experience from a Pioneer Project Is 31204 Newborns. *Journal of Clinical Pathology*, **62**, 35-38.

[3] Ngaly, B., Kayembe, K., Mann, J., Ryder, R., Mbensa, M. and Francis, H. (1987) The Infection among Children with Sickle Cell Anemia, Abstract III. *International Conference on AIDS*, Washington DC, 1-5 June.

[4] Tshilolo, L., Wembonyama, S., Ekwalanga, M., Ngasia, B., Gonzalez, J.P., Wheat

Ondo, I. and Wamba, G. (2010) Sickle Cell Disease in DRC Congo and Central Africa a Century after-James B. Herrick. India.

[5] Nouraie, M., Nekhai, S. and Gordeuk, V.R. (2012) Sickle Cell Disease Is Associated with Decreased HIV HBV and HCV Goal Higher Co Morbidities in US Hospital Discharge Records: A Cross-Sectional Study. *Sexually Transmitted Infections*, **88**, 528-533. https://doi.org/10.1136/sextrans-2011-050459

[6] Diarra, A.B., Guindo, A., Kouriba, B., Dorie, A., Diabaté, D.T., Diawara, I.F., Fan, É. B., Toure, B.A., Traoré, A., Gulbis, B. and Diallo, D.A. (2013) Sickle Cell Anemia and Transfusion Safety in Bamako, Mali. Seroprevalence of HIV, HBV and HCV Infections and Alloimmunization Belonged to Rh and Kell Systems in Sickle Cell Anemia Patients. Elsevier Masson SAS.

[7] Lungu, A., Kalumba, A. and Saragosti Ekwalanga, Mr.S. (2013) HIV Infection Study in Sickle Cell Anemia *βS/βS Duffy*-46*C/C*(46*C-or DARC/C*) Lubumbashi.

[8] Lukuni, L., Binda, P., Mbensa, L. and Binda, K.M. (1989) Seroprevalence and Clinical and Biological Manifestation of AIDS in the Sickle Cell Child Department of Pediatrics, UNIKIN.

[9] Neto, J.P., Lyra, I.M., Reis, M.G., *et al.* (2011) The Combination of Infection and Clinical Severity in Sickle Cell Anemia Patients. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **105**, 121-126. https://doi.org/10.1016/j.trstmh.2010.11.007

[10] Tshilolo, L., Wembonyama, S., Ngasia, B., Kazadi, G., Ekwalanga, Mr., Sica, L., Wamba, G. and Gonzalez, J.P. (2012) Burden of Sickle Cell Disease in Central Africa: Morbidity and Causes of Mortality. Atlanta.

[11] Obaro, S. (2012) Does Sickle Cell Disease Protect against HIV/AIDS? Correspondence to Dr. Stephen Obaro, Department of Pediatrics and Human Development, Michigan State University, East Lansing, MI 48824, USA; Published Online First 20 June.

[12] Ngo, S., Eboumbou, C., Ngouadjeu, E., Zouhaïratou, H. and Mbanya, D. (2013) Prevalence of HIV Seropositivity among Sickle Cell Disease Patients at the Yaounde Central Hospital. *Health Science*, **14**.

[13] Bagasra, O., Steiner, R.M., Castro, S.K.B.O., Dornadula, G., Embury, S., Jungkind, D., Kutlar, L.B.A. and Burchott, S. (1998) Viral Burden and Disease Progression in HIV-1-Infected Patients with Sickle Cell Anemia. *American Journal of Hematology*, **59**, 199-207.