

Unveiling an atypical response to HIV-1 infection by the patient carrier of the beta-s globin gene and Duffy antigen gene double mutation



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Biography

Philomène Lungu Anzwal is a PhD in biomedical sciences & formerly the faculty of medicine and associate professor at the University of Lubumbashi, DRC. She is also the director of the Center of Excellence HIV / UNILU. She is the co-author of the BELA UNILU.20 protocol, holder of a patent n ° 2020/0029 of July 27, 2020 for "BELA UNILU.20 Immunotherapeutic treatment: Combination of type 1 and 2 interferons boosted by chloroquine and antioxidants". She has also obtained the diploma of merit on 21 November 2020 awarded by UNILU for the same work.



Abstract

The selective pressures exerted by plasmodiums falciparum, vivax, and knowlesi on red blood cells led to the modification of membrane antigens, hemoglobin and enzymes thus leading to the mutation of the Duffy antigen, sickle cell disease, thalassemia and the G6PD deficiency. In this study, it was to determine the impact of the Duffy -46C/C mutation on the low susceptibility and slow progression of HIV infection observed in homozygous sickle-cell anemia. This is a descriptive cross-sectional study conducted on 274 subjects over a period of 4 years, from december 2013 to november 2017, subjects of both sexes whose age ranged between 6 months and 40 years of age and resorted to the detection of HIV antibodies by the Determine test, the viral load by Abbott reel Time, the rate of CD4+ by flow cytometry, electrophoresis of hemoglobin on cellulose acetate at pH 8.5 alkaline and identification of Duffy antigen using indirect Coombs test. The research targeted subjects attending sickle cell care centers in the city of Lubumbashi. The Epi Info 7 software, khi- square, mean and frequency were used. The significance level was set at p 0.05. The study found that HIV seroprevalence in $\beta S/\beta S$ is 1.03%. The mean viral load of $\beta S/\beta S$ naive antiretroviral is 14,185 copies/ml compared to that of $\beta A/\beta S$ 56,088 copies/ml. and $\beta A/\beta A$ 7,401 copies/ml. The mean CD4 + is 450 CD4/mm³ compared to $\beta A/\beta S$: 244 and $\beta A/\beta A$: 431 \pm 4. The prevalence of Duffy-46C/C in $\beta S/\beta S$ is 100%. The homozygous sickle Africans have a double mutation, the $\beta S/\beta S$ mutation and the Duffy - 46C/C mutation, which are adaptation mutations. Regarding susceptibility to HIV, the $\beta S/\beta S$ mutation dominates the Duffy -46C/C mutation. And both mutations contribute to the slow progression of HIV which is a synergistic and identical effect.

Publications

The $\beta S/\beta S$ homozygous sickle-cell anemia has a low susceptibility to HIV. All subjects in our study have the Duffy-46C/C mutation (the seroprevalence of Duffy negative antigen is high). The African $\beta S / \beta S$ homozygous sickle- cell subject has a second mandatory mutation : the Duffy-46C/C mutation. The sickle cell mutation βS (low susceptibility to HIV) dominates that of Duffy -46C/C (high susceptibility to HIV) and the two antagonist mutations concur with the slow progression of HIV infection which is a synergistic effect. In studies on the $\beta S / \beta S$ -HIV homozygous sickle cell interaction, the Duffy-46C/C mutation must be automatically associated