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HIV resistance in patients with Sickle Cell Anaemia

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ABSTRACT

These interactions affect the diagnosis, treatment, prognosis, and general healthcare of the patients with both conditions. Although HIV and SCD coexist, few properly designed studies show systematic investigation into their interaction. The evidence that is available and is presented in this review shows that SCD slows the progression of HIV into AIDS but that, on the other hand, HIV worsens SCD; drugs for treating both diseases interact with each other and the respective diseases; and both diseases are risk factors for certain diseases such as stroke, avascular necrosis, severe splenic dysfunction, PAH, and sepsis, which could synergistically increase the odds of getting those diseases. Properly documented information on this has the potential to inform national health policies in these areas, many of which are developing countries. There is therefore the need for further research on the impact of this contemporaneity on the health and well-being of individuals and their families, as well as society as a whole.

Keywords: Sickle cell anaemia, HIV, AIDS, resistance of HIV in sickle cell anaemia

INTRODUCTION

Sickle cell disease (SCD) is a set of genetic disorders caused by mutations in the HBB gene, which encodes the beta globin chain of haemoglobin [1-7]. Population estimates propose that every year nearly 300,000–400,000 neonates are born with SCD worldwide, around 75% in sub-Saharan Africa. In Brazil, 3500 newborns are born with SCD annually, though the incidence varies considerably between the states. SCD is a multisystem disorder characterized by a wide spectrum of clinical manifestations and severity. Genetic and environmental factors play a role in the phenotype diversity of SCD, including the occurrence of infections [8-12]. The World Health Organization data estimates that in 2018 approximately 36.9 million people were infected with human immunodeficiency virus (HIV)

worldwide, the majority in the African continent. In Brazil, approximately 860,000 (630,000 – 1,100,000) individuals were living with HIV in 2017. Human immunodeficiency virus (HIV) and sickle cell disease (SCD) are regarded as endemic in overlapping geographic areas; however, for most countries only scarce data on the interaction between HIV and SCD and disease burden exist. HIV prevalence in SCD patients varies between 0% and 11.5% in published studies. SCD has been suggested to reduce disease progression of HIV into AIDS. Various interactions of antiretroviral therapy with SCD exist. Both SCD and HIV act as common risk factors for stroke, avascular necrosis, severe splenic dysfunction, pulmonary arterial hypertension, and sepsis, which may result in synergistic increase in risk of developing these diseases [13].

There is limited literature focused on HIV infection in sickle cell disease (SCD), but published reports suggest HIV is relatively rare and disease penetrance is low in this population. Most studies comparing HIV prevalence in SCD to a non-SCD population have shown lower rates of HIV in SCD. Comparatively low HIV-associated mortality and progression to AIDS in SCD patients have also been reported. Speculation on the underlying mechanisms for lower HIV prevalence and/or progression in SCD has included behavioral factors reducing exposure and an inhibition of HIV replication due to changes in iron metabolism or the pro-inflammatory component of SCD pathophysiology [14].

SCD and HIV interaction

Sickle cell disease (SCD) is an inherited severe congenital disorder characterized by the presence of structurally abnormal hemoglobin S. Vaso-occlusive disease and hemolytic crisis are the clinical hallmarks of SCD. Vaso-occlusion results in painful episodes, known as sickle cell crisis, and several organ system complications that can cause long-term disabilities and early death. However, the clinical features of SCD vary markedly among the major genotypes [15].

Concomitantly, the human immunodeficiency virus (HIV) epidemic has reached every country where SCD is prevalent, and the spread has been particularly alarming in developing countries, especially sub-Saharan Africa where most patients with SCD live. It is therefore to be expected that some degree of interaction will occur between the 2 diseases—SCD and HIV [16].

Immunosuppression from HIV may affect the natural history of infection with other pathogens by expediting infection, modifying the disease presentation or its course. On the other hand, pathogens and pathogen-derived products may upregulate HIV replication, which can alter the progression of HIV. This includes malaria resulting from infection with *Plasmodium* species, which is a major cause of sickle cell crisis in SCD patients. Therefore, a plausible hypothesis is that HIV may exacerbate SCD, while SCD speeds up progression of HIV to AIDS [17].

SCD is characterized by vascular occlusions leading to anemia, ischemia damage to tissues, increased rate of hemolysis, and increased susceptibility to infection due to immune compromise. HIV, on the other hand, exploits white cell surface proteins, leading to lytic destruction, $CD4^+$ T cell lymphopenia, and immunosuppression. HIV-infected individuals frequently suffer from a wide range of health complications, some of which are directly related to the disease condition, while others are due to aging, antiretroviral therapy, or other patient factors. Life expectancy for patients with HIVand SCD has improved significantly over the past three decades because of new diagnostic methods as well as improved treatment options [18].

SCD protection Against HIV progression

SCD is hypothesized to have a protective effect against HIV infection by an enhanced immunological defense in which increased inflammation, iron metabolism, and immunologic alterations create an unfavorable environment for HIV replication. CCR5 is a chemokine receptor that, in addition to acting as an HIV co-receptor, modulates the action of inflammatory cells. An allele of the CCR5 gene with a 32-base pair deletion in the coding region is labeled CCR532 which causes the absence of cell surface expression of CCR5 in homozygotes, which has been linked to immunity to HIV infection. CCR5 expression is usually lower in people who are CCR532 heterozygous. In a case-control Brazilian study, 1.3% of healthy controls carried the CCR532 allele, while 5.1% of SCD patients had the allele, thus highlighting the association between CCR5Δ32 and SCD. Macrophage-tropic HIV-1 infections can be suppressed by this CCR532 allele [19].

HIV characteristics and outcomes in HIV positive participants with SCD

All HIV positive participants except one (14 of 15) received antiretroviral therapy (ART). Efavirenz, Tenofovir, and Stavudine were the most common drugs that had been prescribed. At the time of the HIV diagnosis, 11(73.3%) participants were asymptomatic, two (13.3%) had acute clinical symptoms (fever and skin rash), one (6.6%) received the diagnosis of acquired immunodeficiency syndrome (AIDS), and for one (6.6%) participant the information was unknown. The history of opportunistic infections among the 15 participants with HIV [16].

CONCLUSION

SCD appears to confer some protection against HIV infection as well as reduce HIV virulence/ Sickle cell disease (SCD), an inherited hemoglobinopathy, affects primarily African Americans in the U.S.A. In addition, about 15% African Americans carry sickle cell trait (SCT). Despite the risk associated with blood transfusions, SCD patients have lower risk of acquiring HIV-1 infection. SCT individuals might also have some protection from HIV-1 infection.

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