An update of human immunodeficiency virus infection: Bleeding disorders.

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Abstract

Human immunodeficiency virus (HIV) infection has been linked to a number of coagulation disorders. High plasma von Willebrand factor concentrations have been associated with HIV illness and may be a sign of active endothelium. Human Immunodeficiency Virus -related thrombocytopenia (Tr-HIV) is the most common haemostatic disorder with a high morbidity and affects patients from every risk group independently of age, sex, or stage of infection.

Keywords: Human immunodeficiency virus, Bleeding disorders, Risks factors.

Introduction

Human immunodeficiency virus (HIV) infection has been linked to a number of coagulation disorders. High plasma von Willebrand factor concentrations have been associated with HIV illness and may be a sign of active endothelium [1]. Important homeostatic mechanisms of non-thrombotic vascular surfaces, vascular tone control, and immunomodulation all include the endothelium. Injury to the endothelium triggers a localised inflammatory response, and as a direct result, occlusive thrombosis events occur [2, 3]. These events are mediated by leucocyte recruitment, platelet adhesion and aggregation, activation of the blood clotting system, and abnormal fibrinolysis. Endothelial dysfunction has been linked to HIV infection. HIV infection is linked to endothelial dysfunction, which might cause activation and consumption of coagulation factors, leading to coagulation malfunction [4].

Interestingly, another study [5] confirmed that certain abnormalities of the coagulation cascade, such as a lack of coagulation factors, should be taken into consideration in pregnant women since bleeding disorders might pose difficulties for pregnancy and childbirth in women. During a typical pregnancy, levels of factors VII, VIII, X, XII, von Willebrand, and fibrinogen considerably rise. Factors XI declines, while factors II, V, and IX just slightly change or stay unchanged [6,7].

Sometimes the bleeding happens on its own, for no apparent reason. Defects in blood components including platelets and/or clotting proteins, commonly known as clotting factors, can result in improper clotting. There are 13 clotting factors made by the body. Blood clotting is impacted if any of them are lacking or faulty, which can cause a mild, moderate, or severe bleeding condition. Hemophilia, for example, is a bleeding illness that can either be hereditary or acquired. Others can result from illnesses such anaemia, liver cirrhosis, HIV, leukaemia, and vitamin K insufficiency [8].

In a study on [4], bleeding disorder notably, Hemophiliacs with HIV disease are at risk for developing acquired immune deficiency syndrome (AIDS), which is typically a painful and debilitating sickness. They also are susceptible to of central nervous system (CNS) damage, extra invasive medical operations, and even death [9]. Young people with HIV often experience social isolation and have trouble becoming independent. The quality of life and prognosis of individuals with HIV infection have changed significantly as a result of the development of HIV protease inhibitor medications (PIs). However, a high incidence of a broad spectrum of negative effects has been linked to the medications in this family. The emergence of an elevated bleeding tendency is a specific issue in individuals with inherited bleeding disorders. The precise mechanism by which protease inhibitor medications cause greater bleeding has not yet been defined, despite the fact that this side-effect has grown to be a severe clinical issue [9].

Human Immunodeficiency Virus (HIV)

HIV's history is rife with both successes and setbacks. Death and life, the HIV timeline started at the start of 1981. An uncommon kind of cancer among homosexual men was found to be on the rise in July of that year, according to the New York Times. The Centers for Condition Control (CDC) frequently referred to the disease by its linked diseases in the beginning because it lacked an official name [1].

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The World Health Organization classifies human HIV infection as pandemic (WHO). It affects crucial immune system components such dendritic cells, macrophages, and helper T cells like CD4 + T cells. Similar to cancer, researchers are still working to try to develop a treatment for this illness [4].

Even in countries with limited resources, the number of AIDS-related fatalities has significantly dropped because to access to stronger antiviral therapies. The majority of HIV-positive individuals in the U.S. now don't progress to AIDS thanks to these life-saving medications. HIV usually progresses to AIDS if left untreated in 8 to 10 years [8]. Your immune system has been severely compromised when AIDS strikes. You'll therefore be susceptible to illnesses that a person with a strong immune system wouldn't typically get [10].

Bleeding disorders in human immunodeficiency virus

There are different factors that affect the normal hemostatic system, of which HIV infection is known to have been one of the main causes of hemostatic abnormality [11]. HIV infection causes serious haemostatic complication especially in the late stage of HIV infection, as immune suppression, and the presence of concurrent infection or neoplastic diseases exacerbates the condition. It is the major causes of the haematological disorder, as the virus deregulates haematopoiesis process and the coagulation system. The virus itself, virus-associated opportunistic infections, adverse effect of antiretroviral therapies (ART), and other associated complications were the possible cause of the abnormalities [12].

A report on thrombocytopenia [13] is a common complication of human immunodeficiency virus (HIV) infection. Its pathogenesis has not yet been established. An increased platelet destruction either due to the nonspecific deposition of circulating immune complexes on platelets or to the presence of specific antiplatelet antibodies as well as direct infection of megakaryocytes by HIV with a resulting decrease in platelet production have been reported as possible mechanisms. About 30-50% of patients with moderate thrombocytopenia may show spontaneous remission. Patients with either severe thrombocytopenia (platelet count < 20 x 10(9)/l) or bleeding are usually first treated with corticosteroids or azidothymidine. If improvement does not occur, further therapeutic approaches are the same as for chronic idiopathic thrombocytopenic purpura.

Also, a study [14], revealed that normal levels of protein S, protein C and antithrombin activities are necessary for coagulation process. In HIV patients, protein S, protein C and antithrombin activities decrease with an increase in plasma D-dimer. Binding of viral and bacterial components to Toll-like receptors (TLRs) stimulate the procoagulant tissue factor to initiate the coagulation cascade [15]. This leads to thrombin activation which then cleaves the fibrinogen to fibrin. Plasmin cleaves the fibrin to produce fibrin degradation products. An increase in monocyte tissue factor expression leads to increase in D-dimer in HIV infection. Decrease protein S, protein C and antithrombin activities as well as increase in plasma D-dimer are the predisposing factors which will increase the risk HIV patients to thrombosis [14].

Conclusion

Human Immunodeficiency Virus -related thrombocytopenia (Tr-HIV) is the most common haemostatic disorder with a high morbidity and affects patients from every risk group independently of age, sex, or stage of infection.

References

1. Obeagu E, Nwovu A, Ochei K, et al. Coagulation system and HIV infection: A Review. Int J Adv Multidiscip Res. 2015;2:109-34.
2. Obeagu EI, Onyenweaku FC, Nwobodo HA, et al. Impact of HIV and Hepatitis Vvirus Coinfection on selected Haematological Markers of the patients in Umuahia, Abia State, Nigeria. Ann Clin Lab Res. 2017;5:2.
3. Obeagu EI, Okwuanaso CB, Edoho SH, et al. Under-nutrition among HIV-exposed Uninfected Children: A Review of African Perspective. J Med Health Sci. 2022;2(3):120-7.
4. Acharya SS. Rare Bleeding Disorders in Children: Identification and Primary Care Management. Pediatrics. 2013;132(5):882-92.
5. Shirzadi M, Radfar AH, Dehghani M. Recurrent miscarriage in a woman with congenital factor V deficiency: A case report. BMC Pregnancy Childbirth. 2022;22(1):915.
6. Obeagu El, Obeagu GU. Effect of CD4 counts on coagulation parameters among HIV positive patients in federal medical centre, Owcrri, Nigeria. Int J Curr Res Biosci Plant Biol. 2015;2:45-9.
7. Obeagu EI, Scott GY, Amekpor F et al. Update on the Roles Human Immunodeficiency Virus Infection and Malnutrition on Immunity. Int J Innov Appl Res. 2022;10(12):1-6.
8. Hoots K. Comprehensive care for hemophilia and related inherited bleeding disorders: Why it matters. Curr Hematol Rep. 2003;2:395-401.
9. Wilde JT, Lee CA, Collins P, et al. Increased bleeding associated with protease inhibitor therapy in HIV-positive patients with bleeding disorders. Br J Haematol. 1999;107(3):556-9.
10. Obeagu EI, Scott GY, Amekpor F, et al. Prevention of New Cases of Human Immunodeficiency Virus: Pragmatic Approaches of Saving Life in Developing Countries. J Med Health Sci. 2022;2(3):128-34.
11. Jackson BS, Nunes Goncalves J, Pretorius E. Comparison of pathological clotting using haematological, functional and morphological investigations in HIV-positive and HIV-negative patients with deep vein thrombosis. Retrovirology. 2020;17(1):14.
12. Seyoum M, Enawgaw B, Getaneh Z, et al. Basic Coagulation Parameters among Human Immunodeficiency Virus-Infected Adults in Gondar, Northwest Ethiopia: A Comparative Cross-Sectional Study. Biomed Res Int. 2018;2018:e5320827.

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13. Ephraim RKD, Ahadzie JE, Adu P, et al. Abnormal coagulation profile in people living with HIV-AIDS on combined Antiretroviral Therapy: Findings from a case-control study in the Ho municipality, Ghana. Pan Afr Med J. 2018;29(121).

14. DAD Study Group. Class of antiretroviral drugs and the risk of myocardial infarction. New Eng J Med. 2007;356(17):1723-35.

15. Majluf-Cruz A. Changes in blood coagulation in HIV infection. Rev Invest Clin. 1997;49(1):51-66.