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Updates on Human Immunodeficiency Virus and Platelets

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Abstract

HIV refers to Human Immunodeficiency Virus. The virus transmits via certain body fluid to damage the body immune cells that flow around the body, detecting errors and disorders in cells as well as infections. HIV infects vital cells in the human immune system, such as helper T cells (specifically CD4+T cells), macrophages, and dendritic cells. Platelets are small, anucleate blood cells that originate as evaginations from bone marrow megakaryocytes. Platelets are essential for haemostasis, but also contribute to other fundamental biological processes, including inflammation and defense against invading pathogens. The paper was written to update the public on the relationship between HIV and platelets. Thrombocytopenia is a frequent complication in HIV infection, affecting 10-50% of the infected individuals. Different search engines like Pubmed, Google Scholar, Researchgate, Medline, etc were utilized to write this paper. In addition, platelets counts were found to be associated with viral load and disease progression, indicating that platelets might modulate viral spread in patients.

Keywords: HIV, CD4+T cells, platelets,

Introduction

HIV stands for Human Immunodeficiency Virus. It is a lentivirus (a member of retrovirus) that causes HIV infection and overtime Acquired Immunodeficiency Syndrome (AIDS).The virus spread through certain body fluid to attack the body immune cells that move around the body, detecting faults and anomalies in cells as well as infections. When HIV targets these cells, it allows life threatening opportunistic infection and cancer to thrive (Gopal, *et al.*, 2014).

HIV infects vital cells in the human immune system, such as helper T cells (specifically CD4+T cells), macrophages, and dendritic cells. HIV cannot replicate on its own, so in order to make new copies of itself, it must infect cells of the human immune system. CD4 cells are white blood cells that play a central role in responding to infections in the body. Over time, CD4 cells are killed by HIV and the body's ability to recognize and fight some types of infection begins to decline. If HIV is not controlled by treatment, the loss of CD4 cells leads to the development of serious illnesses, or 'opportunistic infections'. In people with normal CD4 cell levels, these infections would be recognized and cleared by the immune system (Brenchley *et al.*, 2012).

HIV causes low levels of $CD4^+$ T cells through a number of mechanisms, Pyroptosis of abortively infected T cells, apoptosis of uninfected bystander cells, and killing of infected $CD4^+$ T cells by $CD8^+$cytotoxic lymphocytes that recognize infected cells. When $CD4^+$ T cell numbers decline below a critical level, cell-mediated immunity is lost, and the body becomes progressively more susceptible to opportunistic infections, leading to the development of AIDS (Brencley *et al*., 2012).

HIV is called a retrovirus because it works in a back-to-front way. Unlike other viruses, retroviruses store their genetic information using RNA instead of DNA, meaning they need to 'make' DNA when they enter a human cell in order to make new copies of themselves.HIV is a spherical virus. The outer shell of the virus is called the envelope and this is covered in spikes of the glycoproteinsgp120 and gp41, which allow HIV to lock onto the CD4 receptor on CD4 T cells and enter the cell (Li *et al*., 2016).

Inside the virus envelope is a layer called the matrix. The core of the virus, or nucleus, is held in the capsid, a cone-shaped structure in the centre of the virion. The capsid contains two enzymes essential for HIV replication, the reverse transcriptase and integrase molecules. It also contains two strands of RNA – which hold HIV's genetic material (Lyumkis *et al*., 2013).

HIV can be transmitted through body fluids that include blood, semen, vaginal, rectal fluids and breast milk of the infected person to HIV free person. The major routes of transmission are unsafe sex, contaminated sharp objects, transfusion of contaminated blood. Also HIV can be transmitted from infected mother to her child during pregnancy, childbirth, and breastfeeding. Within this fluid HIV is present as both free virus particles and virus within infected immune cells (Lemussurier *et al*., 2018).

Experiencing a collection of these infections is the most advanced stage of HIV, which is when a person is also said to have AIDS (Acquired Immune Deficiency Syndrome). Effective testing and treatment of HIV means that the large majority of people living with HIV do not reach this stage (Maartens *et al*., 2014).

Antiretroviral therapy (ART) is the use of HIV medicines to treat HIV infection. People on ART take a combination of HIV medicines (called an HIV treatment regimen) every day. ART is recommended for everyone who has HIV. People with HIV should start ART as soon as possible. ART can't cure HIV, but HIV medicines help people with HIV live longer, healthier lives. ART also reduces the risk of HIV transmission. The main goal of ART is to reduce a person's viral load to an undetectable level. An undetectable viral load means that the level of HIV in the blood is too low to be detected by a viral load test. People with HIV who maintain an undetectable viral load have effectively no risk of transmitting HIV to their HIV-negative partner through sex. Although HIV can be controlled by antiretroviral therapy, it cannot be eliminated from the body (Kibaru *et al*., 2015). This is because HIV evades the normal immune system mechanisms for getting rid of cells infected by viruses. HIV integrates itself into the DNA of human immune system cells and only replicates when the cell is stimulated to respond to an infection. These cells are called latently-infected cells. These cells are not recognized as infected by the immune system and killed off, allowing them to persist for as long as the cell lives (Ifeanyichukwu *et al*., 2012).

Signs and symptoms

Lopex *et al*. (2012) reported that the symptoms of HIV vary depending on the stage of infection. Though people living with HIV tend to be more infectious in the first few months after being infected, many are unaware of their status until the later stages. In the first few months after initial infection people may experience no symptoms or an influenza-like illness including fever, headache, rash or sore throat. As the infection progressively weakened the immune system, they can develop other signs and symptoms, such as swollen lymph nodes, weight loss, fever, diarrhea and cough. Without treatment, they could also develop severe illness such as tuberculosis, cryptococcal meningitis, severe bacterial infection and cancer such as lymphomas and kaposi's sarcoma.

Transmission

HIV is transmitted via the exchange of variety of body fluids from infected person, such as blood, breast milk, semen and vaginal secretions. HIV and also be transmitted from mother her child during pregnancy or delivery. Individual cannot become infected through ordinary day to day contact such as kissing, hugging, shaking hands, or sharing personal objects, food and water (Patel *et al.*, 2014).

The life cycle of HIV

1. Attachment and Entry- The process of producing new viruses begins when HIV gains entry to a cell. This process happens in two stages, attachment and fusion. When HIV makes contact with a CD4 cell, the gp120 spikes on the surface of HIV lock onto the CD4 receptor and another co-receptor, either CCR5 or CXCR4.The gp41 protein is used to fuse the HIV envelope with the cell wall. This process of fusion allows the HIV capsid to enter the CD4 cell.

Several types of antiretroviral drug have been developed to block different stages of the processes of attachment and entry:

- CCR5 inhibitor
- Attachment inhibitor
- Fusion inhibitor

The gp41 and gp120 proteins on the surface of the virus are also targets for vaccines that are designed to produce antibody responses. (Protein Data Bank, 2014).

2. Reverse transcription - When HIV RNA enters the cell it must be `reverse transcribed` into proviral DNA before it can be integrated into the DNA of the host cell. HIV uses its reverse transcriptase enzyme to convert RNA into proviral DNA inside the cell.

Two types of antiretroviral drug have been developed to stop the action of reverse transcriptase and the creation of proviral DNA:

Nucleoside and nucleotide reverse transcriptase inhibitors (NRTIs and NtRTIs) block HIV production by inserting a nucleoside or nucleotide into the chain of HIV DNA as it is created, terminating the chain.

Non-nucleoside reverse transcriptase inhibitors (NNRTIs) block HIV production by binding directly to the reverse transcriptase enzyme (Protein Data Bank, 2014).

3. Integration- After HIV RNA is converted into DNA, HIV's integrase enzyme attaches itself to the end of the proviral DNA strands and it is passed through the wall of the cell nucleus. Once the proviral DNA enters the cell nucleus, it binds to the host DNA and then the HIV DNA strand is inserted into the host cell DNA.HIV integrase inhibitors have been developed to block the transfer of the HIV DNA strand into the host cell DNA. After the proviral DNA is integrated into the DNA of the host cell, HIV remains dormant within the cellular DNA. This stage is called latency and the cell is described as 'latently infected'. It can be difficult to detect these latently infected cells even when using the most sensitive tests (Protein Data Bank, 2014).

4. Transcription and Translation- The cell will produce HIV RNA if it receives a signal to become active. CD4 cells become activated if they encounter an infectious agent. When the cell becomes active, HIV uses the host enzyme RNA polymerase to make messenger RNA. This messenger RNA provides the instructions for making new viral proteins in long chains. The long chains of HIV proteins are cut into smaller chains by HIV's protease enzyme (Protein Data Bank, 2014).

5. Assembly and budding-These protein chains begin to assemble into new viruses at the cell wall.

- HIV protease inhibitors are designed to block the activity of HIV's protease enzyme.

As the virus buds from the cell wall, its genome becomes enclosed in a capsid produced from HIV's *gag* protein. After the new virus is assembled, it must leave the cell by pushing through the cell wall. To leave the cell completely and become infectious, the virus must take lipids (fats) from the cell wall to make the surface glycoproteins.

- Maturation inhibitors are being developed to block the cutting of the *gag* protein that is needed to produce a mature virus (Protein Data Bank, 2014).

Platelets

Platelets are also called thrombocytes, they are a component of blood whose function is to react to bleeding from blood vessel injury by clumping, thereby initiating a blood clot. Platelets are small, anucleate blood cells that originate as evaginations from bone marrow megakaryocytes. Platelets are essential for haemostasis, but also contribute to other fundamental biological processes, including inflammation and defense against invading pathogens. For instance, platelets can selectively kill erythrocyte harboring the malaria parasite *Plasmodium falciparum* and can protect against spread of the parasite.

Haemostasis

Haemostasis is the cessation or arrest of blood from a damaged vessel followed by repair. It is a process to prevent and stop bleeding; meaning to keep blood within a damaged blood vessel the opposite of haemostasis is haemorrhage (Shen *et al*., 2017). It is the first stage of wound healing. This involves coagulation, blood changing from a liquid to a gel. Intact blood vessels are central to moderating blood's tendency to form clots. The endothelial cells of intact vessels prevent blood clotting with a heparin-like molecule and thrombomodulin and prevent platelet aggregation with nitric oxide and prostacyclin. When endothelial injury occurs, the endothelial cells stop

secretion of coagulation and aggregation inhibitors and instead secrete von Willebrand factor (factor VIII) which initiate the maintenance of haemostasis after injury. Haemostasis has three major steps:

1) Vasoconstriction,
2) Temporary blockage of a break by a platelet plug,
3) Blood coagulation, or formation of a fibrin clot.

These processes seal the hole until tissues are repaired (Grover *et al*., 2019).

Haemostasis occurs when blood is present outside of the body or blood vessels. It is the innate response for the body to stop bleeding and loss of blood. During haemostasis three steps occur in a rapid sequence. Vascular spasm is the first response as the blood vessels constrict to allow less blood to be lost. In the second step, platelets plug formation, platelets stick together to form a temporary seal to cover the break in the vessel wall. The third and last step is called coagulation or blood clotting. Coagulation reinforces the platelet plug with fibrin threads that act as a molecular glue. Platelets are a large factor in the haemostatic process. They allow for the creation of the "platelet plug" that forms almost directly after a blood vessel has been ruptured. Within seconds of a blood vessel's epithelial wall being disrupted platelets begin to adhere to the sub-endothelium surface. It takes approximately sixty seconds until the first fibrin strands begin to intersperse among the wound. After several minutes the platelet plug is completely formed by fibrin (Andrew *et al*., 2013).

Haemostasis is maintained in the body via three mechanisms:

Vascular spasm (Vasoconstriction) - Vasoconstriction is produced by vascular smooth muscle cells, and is the blood vessel's first response to injury. The smooth muscle cells are controlled by vascular endothelium, which releases intravascular signals to control the contracting properties. When a blood vessel is damaged, there is an immediate reflex, initiated by local sympathetic pain receptors, which helps promote vasoconstriction. The damaged vessels will constrict (vasoconstrict) which reduces the amount of blood flow through the area and limits the amount of blood loss. Collagen is exposed at the site of injury; the collagen promotes platelets to adhere to the injury site. Platelets release cytoplasmic granules which contain serotonin, ADP and thromboxane A2, all of which increase the effect of vasoconstriction. The spasm response becomes more effective as the amount of damage is increased. Vascular spasm is much more effective in smaller blood vessels (Panova-Noeva *et al*., 2019).

Primary haemostasis- Platelets adhere to damaged endothelium to form a platelet plug (*Platelet plug formation*) and then degranulate. This process is regulated through thromboregulation. Plug formation is activated by a glycoprotein called Von Willebrandfactor (vWF), which is found in plasma. Platelets play one of major roles in the haemostatics process. When platelets come across the injured endothelium cells, they change shape, release granules and ultimately become ‘sticky’. Platelets express certain receptors, some of which are used for the adhesion of platelets to collagen. When platelets are activated, they express glycoprotein receptors that interact with other platelets, producing aggregation and adhesion. Platelets release cytoplasmic granules such as adenosine diphosphate (ADP), serotonin and thromboxane A2. Adenosine diphosphate (ADP) attracts more platelets to the affected area, serotonin is a vasoconstrictor and thromboxane A2 assists in platelet aggregation, vasoconstriction and degranulation. As more chemicals are released more platelets stick and release their chemicals; creating a platelet plug and continuing the process in a positive feedback loop. Platelets alone are responsible for stopping the bleeding of unnoticed wear and tear of our skin on a daily basis. This is referred to as primary haemostasis (Andrew *et al*., 2013).

Secondary Haemostasis - Once the platelet plug has been formed by the platelets, the clotting factors (a dozen proteins that travel along the blood plasma in an inactive state) are activated in a sequence of events known as 'coagulation cascade' which leads to the formation of Fibrin from inactive fibrinogen plasma protein. Thus, a Fibrin mesh is produced all around the platelet plug to hold it in place; this step is called Clot formation. During this process some red and white blood cells are trapped in the mesh which causes the primary haemostasis plug to become harder: the resultant plug is called as thrombus or Clot. Therefore, 'blood clot' contains secondary haemostasis plug with blood cells trapped in it. Though this is often a good step for wound healing, it has the ability to cause severe health problems if the thrombus becomes detached from the vessel wall and travels through the circulatory system. If it reaches the brain, heart or lungs it could lead to stroke, heart attack, or pulmonary embolism respectively. However, without

this process the healing of a wound would not be possible (Luyendyk *et al*., 2019).

Effects of HIV on platelets

Several disjointed observations suggest that platelets might also play a role in HIV spread. Thus, thrombocytopenia is a frequent complication in HIV infection, affecting 10-50% of the infected individuals. In addition, platelets counts were found to be associated with viral load and disease progression, indicating that platelets might modulate viral spread in patients.

HIV infection is known to cause coagulation abnormalities by various mechanisms, especially during its late course (Akinbami *et al*., 2010). Hepatic damage is caused by virus itself or by the anti-retroviral (ART) drugs that may also contribute to coagulation defects in HIV patients (Lopez *et al*., 2012).almost all of the coagulation factors are produced in the liver (fibrinogen, prothrombin, labile factor, stable factor, Christmas factor, Stuart power factor e.t.c. therefore leads to their reduction.

Endothelial dysfunction acts as a substrate for initiation of coagulation, which causes consumption of coagulation factors by the increase in von Willebrand factor. HIV increases coagulation via the alterations in extrinsic pathway factors (Mocroft *et al*., 2010).

Platelets play an important role in haemostasis, by forming the primary haemostatic plug following endothelial injury. Platelets decrease in HIV infection due to autoimmune destruction, direct infection of megakaryocytes by virus and ART causing thrombocytopenia (Idris *et al*., 2016). Platelets also decrease due to consumption coagulopathies occurring in Acquired Immune Deficiency Syndrome (AIDS) (Bibas *et al*., 2011). HIV infection has been associated with endothelial dysfunction which may result in activation and consumption of coagulation factors and ultimately coagulation defect most especially von Willebrand disease.

HIV infection is associated with endothelial dysfunction and liver damage. Both endothelial dysfunction and liver damage can result in coagulation defect (Jiang *et al.*, 2010). It is therefore expected that as HIV progresses coagulation abnormalities increases. Impairment of liver function during HIV infection by reducing coagulation factors adds to compromised coagulation state (Choi *et al*., 2011).

Since ART is known to induce hepatotoxicity, coagulation (especially vitamin k dependent) factors are also affected and this ultimately leads to impaired synthesis of these factors. The primary test routinely used for the assessment of coagulopathy are prothrombin time (PT), platelet count and partial thromboplastin time with kaolin (PTTK) (Lopez *et al*., 2012).

The human immunodeficiency virus (HIV) infection is becoming more complex. Haemostatic abnormalities occur frequently in the patient with HIV. HIV-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. Two mechanisms are responsible for the Tr-HIV: bone marrow failure and immunological disorders, namely, circulating immune complex deposited on the platelet membrane and the production of autoantibodies directed against platelet (Jansen *et al*., 2015).

Thrombocytopenia can occur in people with HIV for many reasons. First, HIV can infect the bone marrow cells that create platelets. Second, some drugs used to treat HIV and opportunistic infections can damage bone marrow, resulting in fewer platelets. Third, our own antibodies sometimes target healthy platelets and results in a condition called Immune thrombocytopenic purpura, or ITP. These antibodies are known as autoantibodies, as they are attacking the self and signal the spleen to destroy and remove platelets from the body.

A normal platelet count is between 150,000-400,000 platelets per cubic mililitre of blood. In mild cases of thrombocytopenia, the count is between 100,000-150,000. In severe cases, the count can be close to zero. If the platelet count falls below 30,000, the risk of uncontrolled bleeding is high and could be life-threatening.

Thrombocytopenia is a frequent accompaniment of infection with HIV and is considered as an AIDS-related manifestation. Isolated thrombocytopenia (IT) is frequently observed, occurring in the absence of any other clinical manifestation of AIDS and representing the majority of cases. In a minority of cases, thrombocytopenia closely resembles idiopathic thrombocytopenic purpura. However, the etiology of thrombocytopenia in HIV-infected patients remains unknown and may be multifactorial. Immune mechanism related to platelet antibodies and /or

immune complexes, impaired megakaryocytosis or direct infection of megakaryocytes have been evoked.

Conclusion

HIV stands for Human Immunodeficiency Virus. The virus spread through certain body fluid to attack the body immune cells that move around the body, detecting faults and anomalies in cells as well as infections. Platelets are small, anucleate blood cells that originate as evaginations from bone marrow megakaryocytes. Platelets are essential for haemostasis, but also contribute to other fundamental biological processes, including inflammation and defense against invading pathogens. Thrombocytopenia is a frequent complication in HIV infection, affecting 10-50% of the infected individuals. Also, platelets counts were found to be associated with viral load and disease progression, indicating that platelets might modulate viral spread in patients.

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