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Research Article

Evaluation of Cell Adhesion Molecules in Hepatitis B And C Subjects Co-Infected with HIV on Therapy, Attending Imo Specialist Hospital Owerri

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Abstract

Evaluation of inflammatory proteins, cell adhesion molecules in hepatitis B and C subjects co-infected with HIV, on therapy, attending Imo specialist Hospital Owerri was carried out. 500 subjects were recruited in this studies. The kits and reagents used in this study were commercially purchased and SOP of the manufacturers was strictly adhered to. HIV status of the subjects was assayed, using unigold kit, Hepatitis B was assayed using popular rapid diagnostic kit. E-selectin, P-selectin were assayed using ELISA, the levels of cell adhesion molecules were evaluated. Biomarkers such as selectins (E- and P-selectin) were quantitatively assessed across infected, therapy-receiving, and control groups in Imo state Specialist Hospital Owerri, to evaluate immune activation and the impact of antiretroviral therapy. Cell adhesion molecules E-selectin and P-selectin were markedly elevated in HBV/HIV and HCV/HIV subjects. HBV/HIV subjects showed increases to 30.14 ± 7.25 nm/ml (E-selectin) and 19.07 ± 0.88 nm/ml (P-selectin), with therapy reducing these levels significantly ($p < 0.001$). HCV/HIV individuals showed similar trends, with therapy reducing E-selectin from 29.89 ± 6.11 nm/ml to 16.15 ± 4.87 nm/ml and P-selectin from 17.02 ± 3.76 nm/ml to 10.99 ± 2.42 nm/ml. In conclusion, HIV co-infection with HBV or HCV triggers substantial upregulation of multiple inflammatory markers and adhesion molecules. Antiretroviral therapy exerts a modulatory effect, significantly reducing systemic inflammation and immune activation, and reinforcing its therapeutic value in managing co-infection-related immunopathology.

Keywords: cell adhesion molecules, hepatitis b and c subjects co-infected, HIV, therapy, owerri.

Introduction

Viral hepatitis is a disease that affects the whole body, although the liver is the most afflicted organ. Five distinct hepatitis viruses (A, B, C, D, and E) cause it, and it is a very widespread public health problem in the African Region [1]. All five hepatitis viruses can make you very sick, but liver cancer and cirrhosis, which is when the liver gets scarred after a long time of being infected with hepatitis B and C, are the two most common causes of death. The hepatitis A virus (HAV) is the cause of viral hepatitis type A (infectious hepatitis); the hepatitis B virus (HBV) is linked to viral hepatitis B (serum hepatitis); the hepatitis C virus (HCV) is the cause of hepatitis C (a common cause of post-transfusion hepatitis); the hepatitis D virus (Delta Virus); and the hepatitis E virus (HEV) is the cause of enterically transmitted hepatitis [2].

These viruses are responsible for most cases of acute viral hepatitis in both children and adults. Cell adhesion molecules (CAMs) are important players in inflammation. Cell Adhesion Molecules (CAMs) are proteins that help cells stick to one other and to matrixes. They have a big effect on how leukocytes move during inflammation. Some examples are selectins, integrins, and immunoglobulin (Ig) gene superfamily adhesion receptors. CAMs help white blood cells stick to the endothelium of blood vessels and then go into tissues where there is inflammation [3] They are important for the immune system to grow and stay healthy, and they help recruit immune cells to regions of inflammation. Many people who have HBV or HCV don't get diagnosed since they don't have any symptoms. These individuals serve as the virus's transmission vector and may endure chronic hepatic inflammation, potentially escalating into severe liver diseases such as cirrhosis and

hepatocellular carcinomas. People with HIV who also have chronic HBV and HCV infections are more likely to get cirrhosis and liver cancer during their lives[4]. There is a lack of comprehensive information regarding the inflammatory proteins, cell adhesion molecules, chemokines, and cytokines in hepatitis B and C patients co-infected with HIV at Imo Specialist Hospital Owerri, despite the adverse impacts of HBV and HCV co-infections on mortality and morbidity rates among individuals with HIV [5].

Even if the HBV vaccination is becoming more widely available, HIV programs still don't include diagnosis and treatment. The enormous cost of these treatments makes it hard to find ways to stop them from happening. This study will provide policymakers with valuable data on a baseline for HIV patients co-infected with HBV or HCV. This might be used to get people to pay more attention to the need to include HBV and HCV interventions in HIV prevention and control programs, as long as the right things are taken into account. There are several places in Nigeria where a person can find out if they have hepatitis, however quantitative tests are rare and expensive. Samples have been sent to labs in other countries in the past. Because of this costly project [6], the average person can't afford the test.

Hepatitis B and C virus infections are a public health issue around the world. Viral hepatitis, especially hepatitis B and C, is responsible for a large number of liver diseases worldwide. Infected individuals may remain asymptomatic for years. However, nearly 80% of these individuals progress to chronic carriers, hence elevating their risk of developing liver cirrhosis, liver cancer, and liver failure 20–30 years subsequently[7]. It is important to find out how much cell adhesion molecules are present in people with hepatitis B and C who also have HIV since early detection and treatment can help[8].

This is done to make public health policies and make people aware of how important it is to know their hepatitis status, especially in places where hepatitis B and C viruses are common. Health managers and planners might utilise this study to find specific groups of people, look at the risk factors that are connected, decide how to spend resources, set priorities, and offer the right diagnostic, therapeutic, and preventative services.

Materials and Methods

Study Area: The study was carried out in Imo State University, Owerri, Imo state, Nigeria. Owerri is the capital of Imo State in Nigeria, set in the heart of Igboland. It is also the state's largest city.

Study Population/Sample Size: The study was conducted in Owerri Imo State of Nigeria, the climate of the study area has two main regimes; dry (November- February) and rainy or wet (March-October) seasons. Rainfall in the study area is between 1800 - 2700 mm and average temperature of 28±2oC. The study area has inhabitants who are predominantly farmers, traders, civil servants, cyclist riders and students. The study was a hospital based type conducted within the period of February, 2023 to July, 2024. The study population included 500 HIV patients on Highly Active Antiretroviral Therapy (HAART) who attended Specialist Hospital Umuguma Owerri during the period of the study.

Ethical Approval: The study was approved by the Ethical and Research Committees of the Specialist Hospital Owerri used in the study. Informed consent was also obtained from all participating patients. For subjects under 18 years, parental consent was sought and obtained.

Collection of Blood Samples: Blood samples were collected aseptically by venopuncture, using a 5ml sterile disposable syringe and needle from all the subjects and was then dispensed into a labeled plain dry specimen container. The samples were centrifuged at 3,000pm for 5 minutes after clotting to separate and to obtain the serum. The sera were extracted using a Pasteur pipette and put into appropriate specimen container, and stored at -20 oC prior to use.

Laboratory Procedures

All reagents were commercially purchased and the manufacturer's standard operational procedure (SOP) was strictly followed.

Determination of cell adhesions (E-selectin, P-selectin) were determined with the ELISA Method

Statistical Analysis; All data generated in this study was subjected to statistical analysis using SPSS version 23. Mean and standard deviation, student t-test and correlation were determined. The level of significant will be taken at $p < 0.05$.

Results

Table 1: Comparative analysis of cell adhesion molecules (Serum E selectin, P selectin) in hepatitis B subjects co-infected with HIV

Parameters	Hepatitis B/ HIV	Control	t -value	P – Value
E - selectin (nm/ml)	30.14±7.25	15.10±2.98	14.52	0.000
P - selectin (nm/ml)	19.07±0.88	7.96±1.71	16.39	0.00

KEY: Statistically significant at $P < 0.05$

Table 1 showed that there was a significant increase (P< 0.05) in E – selectin in hepatitis B subjects co-infected with HIVwhen compared with the control 30.14±7.25± 5.58nm/ml and 15.10 ± 2.98nm/ml respectively. In P selection there was also a significant increase in hepatitis B subjects co-infected with HIV subjects when compared with the control 19.07±0.88nm/ml and 7.96±1.71nm/ml respectively (P<0.05).

Table 2: Comparative analysis of cell adhesion molecules (Serum E selectin, P selectin) in hepatitis B subjects co-infected with HIV and HBV/HIV on therapy

Parameters	Hepatitis B / HIV on T	HBV/HIV	t-value	P - Value
E - selectin (nm/ml)	17.56±5.22	30.14±7.25	11.62	0.000
P - selectin (nm/ml)	8.07±1.46	19.07±0.88	11.04	0.00

KEY: Statistically significant at P < 0.05

Analysis of Table 2 showed Comparative Levels of Cell Adhesion Molecules (E-selectin and P-selectin) in HBV/HIV Co-infected Subjects with and Without Therapy

Table 2 presents a comparison of serum levels of two key cell adhesion molecules—E-selectin and P-selectin—in individuals co-infected with hepatitis B virus (HBV) and human immunodeficiency virus (HIV), categorized into those receiving therapy and those not on therapy. The values are expressed as mean ± standard deviation, with accompanying *t*-values and *p*-values to assess statistical significance.

The mean E-selectin level in the untreated HBV/HIV group was 30.14 ± 7.25 nm/ml, which was significantly higher than the 17.56 ± 5.22 nm/ml observed in the therapy group. The *t*-value of 11.62 and *p*-value of 0.000 indicate a highly significant difference.

Similarly, P-selectin levels were markedly higher in untreated HBV/HIV subjects (19.07 ± 0.88 nm/ml) compared to those on therapy (8.07 ± 1.46 nm/ml). The *t*-value of 11.04 and *p*-value of 0.000 also denote a highly significant difference.

Table 2 Showed that there was no significant increase (P< 0.05) in E – selectin in hepatitis B subjects co-infected with HIV on therapy when compared with the control 17.56±5.22± 5.58nm/ml and 15.10 ± 2.98nm/ml respectively. In P selection there was also significant increase in hepatitis B subjects co-infected with HIV subjects on therapy when compared with the control 8.07±1.46nm/ml and 7.96±1.71nm/ml respectively (P<0.05).

Table 3: Comparative analysis of cell adhesion molecules (Serum E selectin, P selectin) in hepatitis C subjects co-infected with HIV and control

Parameters	Hepatitis C coinfected with HIV	Control	P - Value
E - selectin (nm/ml)	29.89±6.11	15.09±1.87	0.000
P - selectin (nm/ml)	17.02±3.76	9.65±1.53	0.00

KEY: Statistically significant at P < 0.05

Analysis of Table 3 showed Comparison of Cell Adhesion Molecules (E-selectin and P-selectin) in HCV/HIV Co-infected Subjects and Controls.

Table 3 compares serum levels of E-selectin and P-selectin—key cell adhesion molecules involved in endothelial and immune activation between hepatitis C virus (HCV) and human immunodeficiency virus (HIV) co-infected individuals and healthy controls. The results are expressed as mean ± standard deviation, with *p*-values indicating the significance of differences. The mean E-selectin level in HCV/HIV co-infected individuals was significantly elevated (29.89 ± 6.11 nm/ml) compared to controls (15.09 ± 1.87 nm/ml), with a *p*-value of 0.00. Similarly, P-selectin levels were much higher in the co-infected group (17.02 ± 3.76 nm/ml) than in controls (9.65 ± 1.53 nm/ml), also with a *p*-value of 0.00, signifying strong statistical significance.

Table 4: Comparative analysis of cell adhesion molecules (Serum E selectin, P selectin) in hepatitis C subjects co-infected with HIVon therapy

Parameters	Hepatitis C / HIV on T	HBV/HIV	t-value	P - Value
E - selectin (nm/ml)	16.15±4.87	29.89±6.11	16.11	**0.001**
P - selectin (nm/ml)	10.99±2.42	17.02±3.76	14.90	**0.001**

KEY: Statistically significant at P < 0.05

Analysis of Table 4 showed Effect of Therapy on Cell Adhesion Molecules (E-selectin and P-selectin) in HCV/HIV Co-infected Subjects.

Table 4. presents the serum levels (mean ± standard deviation) of E-selectin and P-selectin in hepatitis C virus (HCV) and human immunodeficiency virus (HIV) co-infected individuals on therapy, compared to those not receiving treatment. The corresponding *t*-values and *p*-values assess the statistical significance of the observed differences. The mean E-selectin level in untreated HCV/HIV subjects was 29.89 ± 6.11 nm/ml, while those on therapy showed a significantly lower level of 16.15 ± 4.87 nm/ml. The *t*-value of 16.11 and *p*-value of 0.001 indicate a highly significant reduction. This suggests that therapy markedly decreases endothelial activation, likely by reducing systemic inflammation and viral activity. Similarly, P-selectin levels dropped significantly from 17.02 ± 3.76 nm/ml in the untreated group to 10.99 ± 2.42 nm/ml in the treated group. The *t*-value of 14.90 and *p*-value of 0.001 confirm the statistical significance of this reduction.

Discussion

The elevation of E-selectin and P-selectin in individuals co-infected with hepatitis B and C and HIV may result from a unique profile of soluble adhesion molecules that enable pathogen-host interactions and contribute to hepatitis pathogenesis [9]. E-selectin and P-selectin affect how the body responds to inflammation, especially how immune cells interact with the endothelium (the lining of blood vessels) when someone has hepatitis B or C. Researchers have identified a correlation between the expression of these selectins and the progression of chronic hepatitis B and C, alongside an elevation in their levels in affected persons[10]. In instances of chronic hepatitis B (CHB) and chronic hepatitis C (CHC), there is evidence of elevated blood concentrations of soluble E-selectin (sE-selectin). It has been suggested that sE-selectin may serve as an indicator of inflammatory activity in hepatic disorders, as elevated levels appear to correlate with the extent of inflammation [11]. E-selectin may have a role in the inflammatory liver damage observed in CHB and CHC patients by recruiting immune cells to the damaged region. The research has shown that different types of polymorphisms in the E-selectin gene are connected to different levels of liver disease and CHB vulnerability. Studies have shown that people with chronic hepatitis and liver cirrhosis, notably those with chronic hepatitis C (CHC), have higher levels of P-selectin on their platelets. P-selectin facilitates platelet activation and interaction with leukocytes (white blood cells), and its overexpression in chronic hepatitis C (CHC) correlates with these mechanisms [12, 13].There seems to be a link between viral load and platelet activation in chronic hepatitis C (CHC) since P-selectin levels are linked to hepatitis C virus (HCV) RNA levels. In hepatitis B and C, E-selectin and P-selectin are involved in the inflammatory processes that result in liver damage. Blood concentrations of these selectins may elucidate the severity and progression of hepatic injury in hepatitis patients [14] If we can figure out how selectins work in hepatitis pathogenesis, we might be able to come up with new therapeutic methods that target them to modulate the immune response and protect the liver. Chronic hepatitis and liver illness cause blood platelets to become active. Research indicates that chronic hepatitis C correlates with markedly elevated levels of plasma soluble P-selectin [15].

A number of researchers have found that anti-platelet therapy can lower inflammatory markers including CD40 ligand and P-selectin, which can be utilised to stop the hepatitis B virus from spreading. E-selectins are the most flexible when it comes to ligand binding, while P-selectins favour ligands that contain sulphated[16].

This study revealed that the levels of E-selectin and P-selectin were markedly diminished in hepatitis B and C patients co-infected with HIV undergoing therapy, in comparison to controls. This may suggest the significance of cell adhesion molecules in the pathogenesis of inflammatory lung disease [17].

This may indicate that therapeutic intervention lowers the levels of P and E selectin in individuals with HIV co-infection, hence diminishing the severity of the diseases. Additionally, it was noted that the concentrations of selectin P and E diminish with prolonged treatment use. This suggests that therapeutic therapy lowers the levels of selectin P and selectin E in individuals with HIV co-infected with hepatitis A and B. E-selectin and P-selectin levels are significantly increased in hepatitis B and hepatitis C patients co-infected with HIV when compared to healthy controls. These selectins, which are chemicals that help cells stick together, are markers of endothelial activation and inflammation in the blood vessels [18, 19]. The heightened expression probably indicates the combined impact of chronic viral hepatitis and HIV on endothelial cells, leading to increased leukocyte recruitment and vascular damage. This ongoing endothelial activation may be the cause of the increased cardiovascular risk and systemic inflammation seen in people who are infected with both viruses [20].

Conclusion

The results of this investigation indicated that the levels of E-selectin and P-selectin in persons co-infected with HIV and hepatitis B and C, compared to the control group, exhibited substantial differences. The elevated levels of E selectin and P selectin in HIV coinfection indicate a systemic inflammatory response induced by hepatitis and tissue damage. E selectin and P selectin levels indicate that immune responses vary at different stages of development. E selectin and P selectin levels vary throughout different types of bacterial development, which shows how the pathogenicity of pathogens and the immunological response of the host fluctuate. These findings elucidate the physiological mechanisms behind the inflammatory response in HIV coinfections and provide significant insights for clinical treatment and future study.

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