International Journal of Research in Medical Sciences
Sdegbindin R et al. Int J Res Med Sci. 2022 Dec;10(12):2737-2743
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20223074

Original Research Article

Innate immune cells and T CD4 cells profile during hepatitis B among HIV co-infection Beninese

Rafiou Sdegbindin[1], Gatien Adjimon Gbènakpon Lokossou[2], Paulin Kpodji[3], Prisca Gnancadja[3], Banarbé Lafia[4], Olga Rosine Quenum[5], Maximin Senou[1]*

[1]Experimental and Clinical Biology Laboratory, National School of Applied Biosciences and Biotechnologies, National University of Science, Technology, Engineering and Mathematics (UNSTIM), Dassa-Zoumé, Benin
[2]Research Laboratory in Applied Biology, Polytechnic School of Abomey-Calavi, University of Abomey-Calavi, Cotonou, R. Benin
[3]Regional Laboratory of the National Agency for Blood Transfusion, Atlantique Littoral, Health Ministry, Bénin
[4]National Reference Center for Research and Care of People Linving with HIV (NRCRCPL-HIV) of Cotonou Bénin
[5]Laboratory of Hemorrhagic and Viral Fevers (LFHV) of Cotonou Bénin

Received: 10 October 2022
Revised: 05 November 2022
Accepted: 07 November 2022

***Correspondence:**
Dr. Maximin Senou,
E-mail: senouxim@yahoo.fr

ABSTRACT

Background: Worldwide, human immunodeficiency virus (HIV) infection remains a real public health. Hepatitis B virus (HBV) and HIV have the same routes of transmission and shared risk factors and epidemiology similarities. The purpose of this study was to assess the impact of HBV and HIV co-infection on T CD4 cells and innate immune cells.
Methods: A cross-sectional and descriptive study was carried among 260 persons living with HIV (PLHIV) admitted and supported with antiviral tri therapy at the national reference center for research and Care of HIV infected person (NRCRC) of the national hospital and university center in Cotonou, Benin. After PLHIV peripheral blood collection, surface hepatitis B antigens (HBsAg) and HIV serology were tested using ELISA (Enzyme linked immuno sorbent assay). White blood cell count and leukocyte formula were performed using flow cytometry. After staining with anti CD4 antibodies, $TCD4^+$ lymphocytes frequency was determined using flow cytometry. Means were calculated using student T test.
Results: Of the 260 PLHIV, 10.77% (n=28) were co-infected with HBV. Our data has shown a significant decrease of lymphocytes among HIV and HBV co-infected persons and a very significant increase in immune innate cells including eosinophils, polynuclear basophils and monocytes, suggesting an important role of innate immune cells during HIV and HBV coinfection.
Conclusion: HIV and HBV coinfection results in hyperinflammatory response associated with viral clearance. How this hyperinflammatory response is mounted was still unclear. More data are needed for better management of HIV and HBV co-infection.

Keywords: Innate immune cells, HIV, HBV, Co-infection, Benin

INTRODUCTION

Worldwide, human immunodeficiency virus (HIV) infection remains a real public health concern.[1] Hepatitis B virus (HBV) and HIV have the same routes of transmission and shared risk factors and epidemiology similarities.[2] HBV is more transmitted in HBV/HIV co-infected patients, and chronic infection with HBV is not rare.[3] HBV/HIV co-infection results in virological and

immunological changes leading in important morbidity and mortality.[1]

Highly active antiretroviral therapy (HAART) introduction is associated to the decline of HIV/acquired immunodeficiency syndrome (AIDS) morbidity and mortality. However, liver diseases related to hepatitis B viral remain the second major cause of death in HBV/HIV co-infected individuals.[4] Almost 10% of all HIV-infected patients are chronically co- infected with HBV with differences depending in prevalence of HBV.[5] This proportion could reach 15% in Africa. Unfortunately, hepatitis flares occur in 20-25% of HBV/HIV co-infected after HAART.[6] In sub-Saharan Africa HIV/HBV frequency varied from 0% to more than 28.4%.[7]

Several factors influence the chronic stage of HBV infection. These factors include suppressed $CD4^+$ T-cell responsiveness owing to dendritic cell impairment or upregulation of PD-1 expression, down regulation of innate immune responses with decreased natural killer cell function, increased number of regulatory T cells (Treg) resulting in suppression of HBV-specific T-cell responses and therefore decreased viral clearance.[8-12] On the other hand, HBV infection in HIV-positive patients is associated with a lower number and a lower recovery of CD4 lymphocytes, resulting in lower virologic response in a course of HAART.[13]

Other factors such as induction of apoptosis of lymphocytes through activation of Fas/FasL pathways in Kupffer cells lead to immune tolerance to HBV.[14] Moreover, these immune dysregulations are thought to be a major factor leading to liver diseases and carcinogenesis. Even it is thought that the interactions between the different immunological pathways are complicated, it is clear that a strong Th1-like immune response, with high levels of functional cytolytic T lymphocytes (CTL), leads to virus control and better outcomes. Therefore, the reduction of T CD4 lymphocytes will impact the outcome of the disease.[15] Indeed, many studies have shown that HBV/HIV co-infected patients have reduced or impaired CD4 cell count compared to HIV mono-infected.[16] Moreover, higher HBV DNA is observed in HBV/HIV co-infected individuals with low CD4 cell count or advanced HIV/AIDS stages.[17,18] Even on HAART, in these co-infected patients, the immune response was impaired compared to HIV mono-infected patients. This immune-compromission increases the risk of AIDS and death.[22]

However, one large retrospective study has shown an increased CD4 cell count in HBV/HIV co-infected individuals compared to HBV mono-infected on HAART showing that further studies are needed on the consequences of HBV/HIV co-infection on innate and adaptive immunity. How HIV facilitates liver-related damage is not clear. HIV-induced immunodeficiency seems to enhance HBV-related hepatotoxicity.[19] Indeed, depletion of $CD4^+$T cells in HIV infection results in suppression of the antigen presentation of liver resident Kupffer cells and the cytokine secretion of lymphocytes, allowing the host immunosuppression. The inhibition of the host immune response therefore enhances HBV replication with more severe liver damage.[20]

Determining the impact of HBV/HIV co-infection on innate and adaptive immune responses may provide a means to determine the likelihood of resolving an HBV infection or the risk of developing hepatocellular carcinoma.

Even HBV prevalence is in regression in Benin, HBV co-infection in HIV positive patients remains a great concern and data on the impact of this co-infection on innate immune cells needs to be addressed.[21]

METHODS

Enrolled patients

A cross-sectional and descriptive study was carried out in the national reference center for research and care of HIV infected person (NRCRC) of the national hospital and university center in Cotonou (Benin) from September-2019 to January-2020. Intentional sampling was performed in all patients admitted to NRCRC during the study period and who gave their consent to participate in the study.

Were included in this study, persons living with HIV (PLHIV) treated during the study period, regardless of age, sex and origin and who gave their consent to participate in the study. Two hundred and sixty persons living with HIV (PLHIV) were admitted and supported with HAART at NRCRC. Blood samples were collected from patients in tubes adapted for analysis after their consent.

The exclusion criteria for the study were the non-consent of patients or patients treated for chronic pathologies.

Ethics statement

The study was approved by the national research ethic review boards of Benin.

Blood samples from patients after their consent. To ensure anonymity, an identification code was assigned to each participant.

Blood sampling, serological diagnoses and white blood cells analysis

The serological analyses were carried out at the regional site of the national agency for blood transfusion of Cotonou. After PLHIV peripheral blood were collected in dry tube without anti-coagulated, serological diagnoses of HBV infection (HBsAg) were detected by ELISA

(Monolisa™ HBs Ag ultra, METTRE LE PAYS). To diagnose HIV, serological diagnoses of HIV were performed by a sensitive test (Genscreen ultra HIVAg-Ab, compagnie et pays) and by a discriminatory test (Geenius™ HIV ½, compagnie et pays).

White blood cell count and leukocyte formula were performed from anti-coagulated EDTA tube using flow cytometry (sysmex XT 2000i, COMPAGNIE ET PAYS). After staining with anti CD4 antibodies, $TCD4^{+}$ lymphocytes frequency was characterized using flow cytometry.

Statistical analysis

Results were expressed as mean ± standard error on the mean (SEM). Statistical analysis between HIV and HBV co-infected patients and HIV mono-infected patients were performed using the student's t test (GraphPad, version 6.01).

RESULTS

Sociodemographic characteristics of enrolled patients

Demographics for the 260 PLHIV are listed in Table 1. Patients were predominantly female (n=184, 70.77%) among the whole group. Of the 260 PLHIV, 10.77% (n=28) were co-infected with HBV. The average age for mono-infected HIV patients was 41±1 years, 37±2 years for HBV/HIV co-infected patients. Among the 10% co-infected patients, 2.1% were male and 7.9% female.

Table 1: Ages, sexes and serological status of enrolled patients, (n=260).

Parameters	N (%)
Age (Years)	
19-25	12 (4.62)
26-35	62 (23.65)
36-45	118 (45.38)
46-55	46 (17.69)
56-65	18 (6.92)
Over 65	04 (1.54)
Gender	
Male	76 (29.23)
Female	184 (70.77)
PL HIV	
HBS negative	232 (89.23)
HBS positive	28 (10.77)

Leucocytes profile in HIV mono-infected patients and HBV/HIV co-infection patients

Figure 1 A shows whole blood leucocytes count among mono-infected HIV patients and HBV/HIV co-infected patients. The mean of leucocytes among mono-infected PLHIV was 4.9±0.11 G/L whereas it was 5.4±0.17 G/L among HBV/HIV co-infected patients, showing a significantly increase of leucocytes during HBV/HIV co-infection (p=0.03).

Mononuclear cell profile in HIV mono-infected patients and HBV/HIV co-infection patients

Total number of lymphocytes decreases in HBV/HIV co-infected patients compared to mono-infected PLHIV (1.8±0.08 G/L vs. 2.0±0.05 G/L), (p=0.04) (Figure 1 B). HBV/HIV co-infection was associated to non-statistically significant T CD4 lymphocytes decrease with a mean of 468±21 cells/µL in HBV/HIV co-infected individuals and a mean of 518±12 cells/µL in HIV mono-infected patients (Figure 1 C). However, the mean of the number of monocytes increases significantly during HBV/HIV co-infection (0.48±0.019 G/L) compared to HIV mono-infection (0.42±0.011 G/L) (p=0.009) (Figure 1 D).

Polynuclear cell profile in HIV mono-infected patients and HBV/HIV co-infection patients

The difference between polynuclear neutrophils numbers in HIV mono-infected patients (2.3±0.08 G/L) and in HBV/HIV co-infected individuals (2.1±0.16 G/L) was not statistically significant as shown in Figure 2 A. However, the number of eosinophilic polynuclear cells significantly (p=0.002) increases during HBV/HIV co-infection (0.22±0.015 G/L) compared to HIV mono-infected individual (0.15±0.009 G/L) (Figure 2 B). Figure 2 C also shows that HBV/HIV co-infection is associated with increased numbers of basophilic polynuclear cells (0.032±0.0021 G/L) compared to HIV mono-infected individual (0.026±0.001 G/L) (p=0.005).

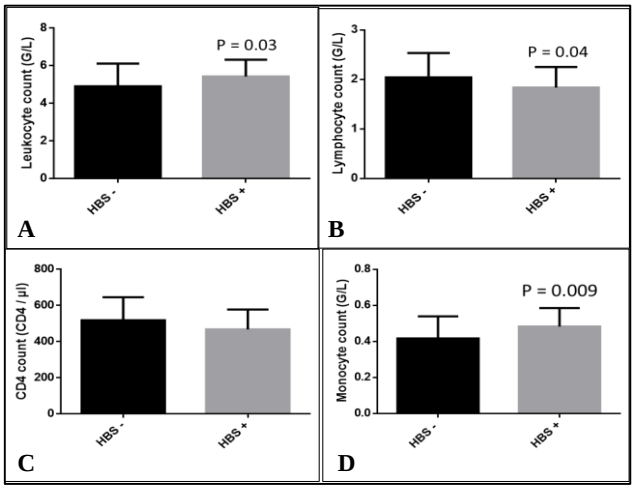

Figure 1 (A-D): Leucocytes profile in HIV mono-infected patients and HBV/HIV co-infection patients. Whole blood leucocytes were counted among mono-infected HIV patients and HBV/HIV co-infected patients mononuclear cell profile in HIV mono-infected patients and HBV/HIV co-infection patients were evaluated. T CD4 lymphocytes decrease in HBV/HIV co-infection. Monocytes increases significantly during HBV/HIV co-infection.

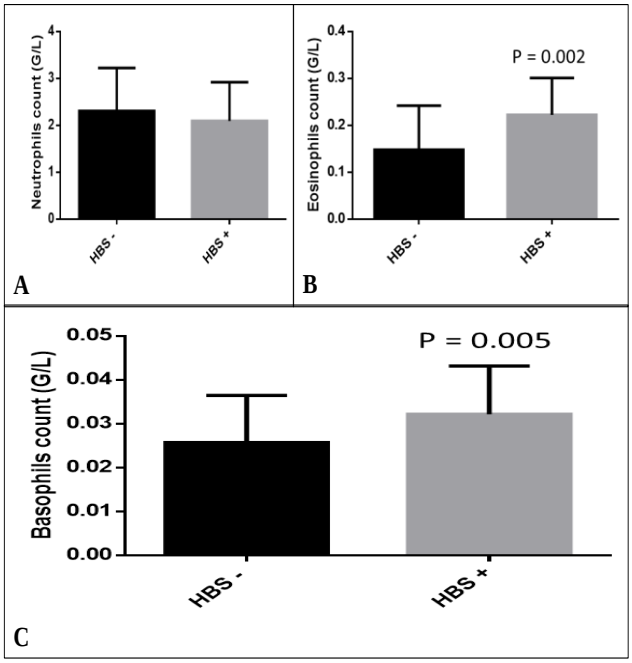

Figure 2 (A-C): Polyonuclear cell profile in HIV mono-infected patients and HBV/HIV co-infection patients. Polynuclear neutrophils number did not significantly change between mono-infected HIV patients and HBV/HIV co-infected patients. Eosinophil and basophil number significantly increases during HBV/HIV co-infection.

DISCUSSION

This descriptive and cross-sectional study aimed to assess the impact of co-infection with HBV and HIV on the number of certain immune cells including monocytes, macrophages, polymorphonuclear cells and T lymphocytes. Our data showed that female patients were more numerous (70.77%) than male. The female predominance observed in this study and in others countries including in Benin, Bostwana, Ghana, Burkina Faso, Cote d'Ivoire, Uganda, could be explained by the ease women have in getting to the hospital and thus get better care. This vulnerability is favored by economic, educational, psychological and socio-cultural factors.[22-27]

The prevalence of HIV and HBV co-infection in the present study is 10.77%. This prevalence is below that observed in other studies such as in Benin and elsewhere in Africa, showing the importance of preventive measures and awareness campaigns, which must be continued. Moreover, these measures explain the low prevalence observed in Guinea, in Senegal, in Bostwana, and in Uganda.[23,27-31] These preventive measures limit the circulation of the virus according to geographical areas, the types of groups at risk and the modes of exposure.

Our observations showed an increase in the number of blood leukocytes (p=0.03) which could be due to the therapeutic success. This increase results in an increase in the number of polymorphonuclear eosinophils, basophils and blood monocytes. However, this increase had not been observed in other African regions, suggesting immunosuppression or treatment failure in these populations.[32] It would certainly be essential to evaluate the number of natural killer cells and other innate lymphocytes such as ILC-1 and cytotoxic T lymphocytes and Th17 lymphocytes which have significant antiviral activity.[33]

We have also shown that coinfection with HIV and HBV is associated with a drop in the number of lymphocytes (p=0.04). This decrease is linked to a high replication of HIV and HBV, further leading to the subversion of the immune system of these patients.[34] However, this relationship had not been observed in Mozambique in a population of co-infected or mono-infected people.[32]

Co-infection with HIV and HBV in our study does not seem to affect the mean number of $CD4^+$ T lymphocytes in both groups. This observation has been confirmed in other African studies.[25,29] Therefore, it will be relevant to access T cell subclasses to find the specific class of CD4 T cells that are affected. Recently, it was shown that pre-HAART, HIV-HBV co-infected individuals *vs.* HIV mono-infected individuals had reduce CD4+ T-cell compare to person infected by HIV alone, suggesting further immune depletion in co-infection than in HIV infection alone.[31] However, unlike to HBV mono-infection, it was shown that HIV and HBV co-infected patients without HAART are characterized by impaired HBV specific T-cell response, with significantly increase in liver-infiltrating IL-10-producing CD8+ T cells.[35] This leads in accelerated liver disease progression owing to impaired innate immunity and immune activation.[36] Indeed, studies have demonstrated a diminished HBV-specific CD8+ T-cell response in HIV-HBV co-infected patients on long term HAART, showing that other T cell class may be affected during HIV/HBV co-infection. This diminution was attributed to significant CD4+ T-cell depletion in HIV-HBV co-infected patients.[37] The average number of neutrophils does not vary as significantly during co-infection with HIV and HBV. Unlike neutrophils, co-infection with HIV and HBV is statistically associated with an increase in the number of eosinophils (p=0.002). The impact of HIV on HBV was demonstrated and showed an accelerated HBV-related liver disease progression and significantly higher liver-related mortality compared to infection with either HIV or HBV alone.[38] The increase number of eosineophils may be the consequence of the promotion of hepatocellular carcinoma in co-infected patients associated with hypersensitivity.[39]

Similarly, the number of basophils (p=0.005) and monocytes (p=0.009) increases significantly during co-infection with HIV and HBV. This increase in the average number of blood monocytes in this study suggests an important role for monocytes in antiviral responses and viral clearance against viral hepatitis B infection.[40] Mechanism involved in how HIV and or

HBV evade the immune system remains unclear. However, it was demonstrated that viruses used a large range of mechanisms to evade the host immune system. Recent data have shown that many of these, mechanisms target dendritic cells (DC) function.[41] Indeed, DC are susceptible to HBV and intracellular HBV particles leads to impair allostimulatory function of monocytes-derived dendritic cells (MoDC).[42] This may results in a generalized altered function of antigen-presenting capacity during HBV infection. Moreover, the impaired MoDC capacities to stimulate T cells are associated to low IL-12 production in HBV-MoDC cultures, resulting in Th1 impaired functions. Th1 role in virus clearance has been demonstrated in patients with HBV infection. Therefore, the increase of monocytes observed in our study will allow a well response during HIV and HBV co-infection. However, the role of IL-12 in viral infections remains controversial and some earlier reports indicate that IL-12 may not be necessary for induction of Th1 response in viral infection.[43]

It will also be relevant to investigate HLA molecules expression. Indeed, it was demonstrated that a lower expression of HLA-DR may additionally contribute to reduced allostimulatory activity of MoDC (Article discussion).

This study had some limitations. HBV viral load detection was not done. This detection of the viral load by PCR could allow us to take into account patients despite the negativity of their AgHBs. Furthermore, the leukocyte count was not determined before antiretroviral treatment in patients co-infected with HIV and HBV. This count could allow us to assess the impact of antiretroviral treatment on the quality and quantity of the innate immune response in these patients during treatment.

CONCLUSION

The impact of HIV on HBV is known. Our study showed different changes in the proportions of polymorphonuclear eosinophils, basophils and monocytes during co-infection between HIV and HBV. We have also observed a significant decrease of lymphocytes.

The impact of HIV and HIV-HBV co-infection complicated the mechanistic studies of HI and-HBV co-infection. Carefully matched controls groups, with patients infected with HIV alone and HIV-HBV co-infection alone, are needed to carefully adress the outcome of this co-infection and mechanism underlying it control. The widespread of HAART allow the reduction of the clinical burden of most HIV and HBV co-infections and this will continuous in coming years. However, in low-income countries HAART is still limited. Therefore, individuals often initiate HAART sometime too late. The prevalence of certain co-infections such as tuberculosis and malaria is very high, making HIV and HBVco-infection an important concern.

Finally, a better understanding of the immune-pathogenesis of this co-infection and the complexities of HAART will be helpful in the future.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. OMS. Organisation Mondiale de la Santé (OMS) Principaux repères sur le VIH/sida. 2020. Avalable at : https://www.who.int/fr/news-room/fact-sheets/detail/hiv-aids. Accessed on 17 July, 2020.
2. Matthews PC, Geretti AM, Goulder PJR., Klenerman P.. Epidemiology and impact of HIV coinfection with hepatitis B and hepatitis C viruses in Sub-Saharan Africa. J Clin Virol Off Publ Pan Am Soc Clin Virol. 2014;61:20-33.
3. Singh KP, Crane M, Audsley J, Avihingsanon A, Sasadeusz J, Lewin SR. HIV-hepatitis B virus coinfection: epidemiology, pathogenesis, and treatment. AIDS Lond Engl. 2017;31:2035-52.
4. Dharel N, Sterling RK. Hepatitis B Virus–HIV Coinfection: Forgotten but Not Gone. Hepat. B VIRUS. 2014;10:9.
5. Ganesan M, Poluektova LY, Kharbanda KK, Osna NA. Human immunodeficiency virus and hepatotropic viruses co-morbidities as the inducers of liver injury progression. World J Gastroenterol. 2019;25:398-410.
6. Rowley MW, Patel A, Zhou W, Wong M, Seetharam AB. Immune Reconstitution Syndrome with Initiation of Treatment of HBV/HIV Co-infection: Activity Flare associated with E antigen Seroconversion. Ann Hepatol. 2019;18:220-24.
7. Stabinski L, O'Connor S, Barnhart M, Kahn RJ, Hamm TE. Prevalence of HIV and hepatitis B virus co-infection in sub-Saharan Africa and the potential impact and program feasibility of hepatitis B surface antigen screening in resource-limited settings. J Acquir Immune Defic Syndr. 1999;68(3):S274-85.
8. Xu D, Fu J, Jin L, Zhang H, Zhou C, Zou Z, Zhao J-M, Zhang B, Shi M, Ding X, Tang Z, Fu Y-X, Wang F-S. Circulating and Liver Resident CD4+CD25+ Regulatory T Cells Actively Influence the Antiviral Immune Response and Disease Progression in Patients with Hepatitis B. J Immunol. 2006;177:739-47.
9. Li M, Sun X-H, Zhu X-J, Jin S-G, Zeng Z-J, Zhou Z-H et al. HBcAg induces PD-1 upregulation on CD4+T cells through activation of JNK, ERK and PI3K/AKT pathways in chronic hepatitis-B-infected patients. Lab Invest. 2012;92:295-304.
10. Liang X-S, Zhou Y, Li C-Z, Wan M-B. Natural course of chronic hepatitis B is characterized by changing patterns of programmed death type-1 of CD8-positive T cells. World J Gastroenterol. WJG. 2010;16:618-24.

11. Chen Y, Sun R., Wu X, Cheng M, Wei H, Tian Z. CD4+CD25+ Regulatory T Cells Inhibit Natural Killer Cell Hepatocytotoxicity of Hepatitis B Virus Transgenic Mice via Membrane-Bound TGF-β and OX40. J. Innate Immun. 2016;8:30-42.
12. Trehanpati N, Vyas AK. Immune Regulation by T Regulatory Cells in Hepatitis B Virus-Related Inflammation and Cancer. Scand J Immunol. 2017;85:175-81.
13. Sun H-Y, Sheng W-H, Tsai M-S, Lee K-Y, Chang S-Y, Hung C-C. Hepatitis B virus coinfection in human immunodeficiency virus-infected patients: A review. World J Gastroenterol. WJG. 2014;20:14598-614.
14. Yuan F, Zhang W, Mu D, Gong J. Kupffer cells in immune activation and tolerance toward HBV/HCV infection. Adv Clin Exp Med Off Organ Wroclaw Med. Univ. 2017;26:739-45.
15. Cao H, Zhang R, Zhang W. CTLA-4 interferes with the HBV-specific T cell immune response (Review). Int J Mol Med. 2018;42:703-12.
16. Pappoe F, Hagan CKO, Obiri-Yeboah D, Nsiah P. Sero-prevalence of hepatitis B and C viral infections in Ghanaian HIV positive cohort: a consideration for their health care. BMC Infect. Dis. 2019;19:380.
17. Chun HM, Mesner O, Thio CL, Bebu I, Macalino G, Agan BK et al. Infectious Disease Clinical Research Program HIV Working Group. HIV outcomes in Hepatitis B virus coinfected individuals on HAART. J. Acquir. Immune Defic. Syndr. 2014;1999(66):197-205.
18. Thio CL, Smeaton L, Saulynas M, Hwang H, Saravanan S, Saravan S et al. Characterization of HIV-HBV coinfection in a multinational HIV-infected cohort. AIDS Lond Engl. 2013;27:191-201.
19. Thio CL. Hepatitis B and human immunodeficiency virus coinfection. Hepatol Baltim M. 2009;49:S138-45.
20. Gürtler LG. Effect of antiretroviral HIV therapy on hepatitis B virus replication and pathogenicity. Intervirology. 2014;57:212-7.
21. Lozes E, Dougnon V, Bankole H, Houngbegnon P, Anato P, Gahou A et al. Prévalence de l'antigène HBs à l'Hôpital de Zone d'Abomey-Calavi/Sô-Ava au Bénin de 2009 à 2014. Int J Biol Chem Sci. 2017.
22. Dovonou CA, Amidou SA, Kpangon AA, Traoré YA, Godjedo TPM, Satondji AJ et al. Prevalence of hepatitis B in people infected with HIV in Parakou in Benin] Pan Afr Med J. 2015;20:125.
23. Mandiwana A, Tshitenge S. Prevalence of human immunodeficiency virus-hepatitis B virus co-infection amongst adult patients in Mahalapye, Ngami, Serowe, Botswana: a descriptive cross-sectional study. South Afr Fam Pract. 2017;59:94-7.
24. Kye-Duodu G, Nortey P, Malm K, Nyarko KM, Sackey SO, Ofori S et al. Prevalence of hepatitis B virus co-infection among HIV-seropositive persons attending antiretroviral clinics in the Eastern Region of Ghana. Pan Afr Med J. 2016;25.
25. Ilboudo P. Aspect épidémiologiques, cliniques, paracliniques et évolutives de l'hépatite virale B chez les patients infectés par le VIH à l'hôpital de jour de Bobo-Dioulasso. Thèse, Med, Bobo-Dioulasso. 2013;05.
26. Attia KA. Prevalence and virological profiles of hepatitis B infection in human immunodeficiency virus patients. World J Hepatol. 2012;4:218.
27. Ocama P, Seremba E, Apica B, Opio K. Hepatitis B and HIV co-infection is still treated using lamivudine-only antiretroviral therapy combination in Uganda. Afr Health Sci. 2015;15:328-33.
28. Sehonou J, Fiogbe AA, Zannou DM, Bashi J, Houngbe F, Kodjoh N. Coinfection VIH-VHB chez des patients sous traitement antirétroviral au Centre National Hospitalier et Universitaire de Cotonou (République du Bénin). Rev CAMES. 2010;85-9.
29. Otegbayo JA, Taiwo BO, Akingbola TS, Odaibo GN, Adedapo KS, Penugonda S et al. Prevalence of hepatitis B and C seropositivity in a Nigerian cohort of HIV-infected patients. Ann Hepatol. 2008;7:152-6.
30. Kaba D, Diallo MS, Barry R, Diakite M, Diane BF, Keita F et al. Prévalence de l'Antigène HBS chez les Personnes Infectées par le VIH à l'Hôpital de Jour de Donka (Guinée). Health Sci Dis. 2020;21.
31. Lô G, Sow-Sall A, Diop-Ndiaye H, Mandiouba NCID, Thiam M, Diop F, Ndiaye O et al. Prevalence of hepatitis B markers in Senegalese HIV-1-infected patients: HBV/HIV Coinfection in Senegal. J Med Virol. 2016;88:461-5.
32. Chambal LM, Gudo ES, Carimo A, Corte Real R, Mabunda N, Maueia C et al. HBV infection in untreated HIV-infected adults in Maputo, Mozambique. PloS One 2017;12:e0181836.
33. Lujan RA, Vrba SM, Hickman HD. Antiviral Activities of Group I Innate Lymphoid Cells. J Mol Biol. 2022;434:167266.
34. Nikolopoulos GK, Paraskevis D, Hatzitheodorou E, Moschidis Z, Sypsa V, Zavitsanos X et al. Impact of hepatitis B virus infection on the progression of AIDS and mortality in HIV-infected individuals: a cohort study and meta-analysis. Clin Infect Dis Off Publ Infect Dis Soc Am. 2009;48:1763-71.
35. Iser DM, Avihingsanon A, Wisedopas N, Thompson AJ, Boyd A, Matthews GV et al. Increased intrahepatic apoptosis but reduced immune activation in HIV-HBV co-infected patients with advanced immunosuppression. AIDS Lond Engl. 2011;25:197-205.
36. Chang CC, Crane M, Zhou J, Mina M, Post JJ, Cameron BA et al. HIV and co-infections. Immunol Rev. 2013;254:114-42.
37. Sherman AC, Trehanpati N, Daucher M, Davey RT, Masur H, Sarin SK, Kottilil S, Kohli A. Augmentation of Hepatitis B Virus-Specific Cellular Immunity with Programmed Death Receptor-1/Programmed Death Receptor-L1 Blockade in Hepatitis B Virus and HIV/Hepatitis B Virus Coinfected Patients Treated with Adefovir. AIDS Res. Hum. Retroviruses 2013;29, 665–672. https://doi.org/10.1089/aid.2012.0320

38. Thio CL, Seaberg EC, Skolasky R, Phair J, Visscher B, Muñoz A et al. Multicenter AIDS Cohort Study. HIV-1, hepatitis B virus, and risk of liver-related mortality in the Multicenter Cohort Study (MACS). Lancet Lond Engl. 2002;360:1921-6.
39. Roufosse F, Weller PF. Practical approach to the patient with hypereosinophilia. J Allergy Clin Immunol. 2010;126:39-44.
40. Brempelis KJ, Crispe IN. Infiltrating monocytes in liver injury and repair. Clin Transl Immunol. 2016;5:e113.
41. Beckebaum S, Cicinnati VR, Zhang X, Ferencik S, Frilling A, Grosse-Wilde H et al. Hepatitis B virus-induced defect of monocyte-derived dendritic cells leads to impaired T helper type 1 response in vitro: mechanisms for viral immune escape. Immunology. 2003;109:487-95.
42. Beckebaum S, Cicinnati VR, Dworacki G, Müller-Berghaus J, Stolz D, Harnaha J et al. Reduction in the Circulating pDC1/pDC2 Ratio and Impaired Function of Ex Vivo-Generated DC1 In Chronic Hepatitis B Infection. Clin Immunol. 2002;104:138-50.
43. Xing Z. Breach of IL-12 monopoly in the initiation of type 1 immunity to intracellular infections: IL-12 is not required. Cell Mol Biol Noisy Gd Fr. 2001;47:689-94.

Cite this article as: Sdegbindin R, Lokossou GAG, Kpodji P, Gnancadja P, Lafia B, Quenum OR et al. Innate immune cells and T CD4 cells profile during hepatitis B among HIV co-infection Beninese. Int J Res Med Sci 2022;10:2737-43.