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Assessment of liver fibrosis by transient elastography among human immunodeficiency virus/hepatitis B virus and hepatitis B virus-mono-infected patients on tenofovir therapy in Jos, Nigeria

Joseph Anejo-okopi[1], Oludare Oladipo Agboola[2], David Ochola Amanyi[3], Ocheme Julius Okojokwu[4], Chika Onwuamah[5], Bulus Jonathan[6], Chima Anyuku Azubuike[7], Akpa Samuel Tanko[8], Seljul Mamzhi Crown Ramyil[9], Otobo Innocent Ujah[10]

Departments of [1]Microbiology and [2]Biological Sciences, Federal University of Health Sciences, Otukpo, [3]Department of Family Medicine, Jos University Teaching Hospital, [4]Department of Microbiology, University of Jos, Jos, Benue, [5]Department of Microbiology, Human Virology Laboratory, Nigerian Institute of Medical Research, Yaba, Lagos, [6]Department of Family Medicine, Plateau State Specialist Hospital, [7]Department of Family Medicine, Bingham University Teaching Hospital, [8]Department of Internal Medicine, AIDS Prevention Initiative in Nigeria, Jos University Teaching Hospital, [9]Department of Medical Microbiology, Bingham University Teaching Hospital, Jos, Benue, [10]Department of Obstetrics and Gyneacology, Federal University of Health Sciences Otukpo, Nigeria.

ABSTRACT

Objectives: Chronic hepatitis B (CHB) infection, both in human immunodeficiency virus (HIV) coinfection and hepatitis B virus (HBV)-mono-infection, is associated with a risk of progression to chronic liver disease. In Nigeria, there is a paucity of data on transient elastography (TE) in HIV/HBV and HBV-mono-infected patients. This study aimed at assessing liver fibrosis using TE in relation to liver function biomarkers and HBV deoxyribonucleic acid (DNA) among HIV/HBV and HBV-mono-infected patients on long-term antiviral therapy.

Material and Methods: This was a cross-sectional study among HBV–HIV and HBV-mono-infected adult's patients receiving a tenofovir-containing antiretroviral and mono-tenofovir ≥12 months at three selected tertiary hospitals in Jos Metropolis from February 2018 to May 2019, after obtaining ethical approval from the Institutional Review Boards and informed consents. The patients' HBV DNA, platelet count, hematological, and biochemical parameters were assessed, and liver stiffness was measured by TE in kilopascals (kPa), and valid TE measurements were interpreted as: normal (F0–1 0–4), minimal fibrosis (F2 5–7.4), moderate (F3 7.5.9.4), and severe fibrosis and cirrhosis (F4 ≥9.5).

Results: A total of 101 (50 HIV/HBV and 51 HBV-mono-infected) were enrolled during the study period, comprising 42.6% males and 57.4% females. The median age interquartile range among HIV/HBV coinfected was 40.5 years (36.0–45.3) and HBV-mono-infected was 41.0 years (35.0–49.0). The median platelet count was low in the HBV-mono-infected group 195 × 10^9/L (168–257), $P = 0.034$. The overall prevalence of severe liver fibrosis (≥9.5 kPa) was 13/101 (13.0%), and among HIV/HBV-coinfected and HBV-mono-infected patients, the prevalence was 4/50 (8.0%) and 9/51 (17.6%), respectively. The plasma HBV DNA was <20 copies/mL in 38/50 (76.0%) HIV/HBV coinfected individuals and in 30/51 (58.8%) of HBV-mono-infected patients. In addition, 10/50 (20.0%) HIV/HBV coinfected and 19/50 (37.3%) HBV-mono-infected patients had plasma HBV DNA levels of 20–20,000 copies/mL. In the case of HIV/HBV coinfection, the prevalence of severe fibrosis (≥9.5) was 4/50 (8.0%), while in HBV-mono-infected patients, the prevalence was was 9/51 (17.6%). The overall prevalence of thrombocytopenia was observed in 4/101 cases (3.9%): 1/50 (2.0%) in HIV/HBV coinfected individuals and 3/51 (5.9%) in HBV-mono-infected patients.

Conclusion: Severe liver fibrosis as observed among HIV/HBV-coinfected and HBV-mono-infected patients in this study affirmed the necessity of routine HBV screening in clinics and it highlights the immense potentials of tenofovir therapy in the treatment of CHB patients.

Keywords: Liver fibrosis, Transient elastography, Tenofovir, Chronic hepatitis B virus, Nigeria

INTRODUCTION

Hepatitis B virus (HBV) infection remains a severe public health challenge worldwide, especially in Sub-Saharan Africa (SSA), despite the introduction of HBV vaccination and effective anti-viral therapy to treat HBV. Although the risk of horizontal and vertical mother–to–child transmission has significantly reduced,[1] it is estimated that over 248 million people are still chronically infected by HBV worldwide.[2] However, the social and economic burden remains a serious concern, and there have been few concerted efforts by policymakers and health-care providers to improve funding, awareness, and access to HBV infection management.[3] The

***Corresponding author:** Joseph Anejo-okopi, Department of Microbiology, Federal University of Health Sciences, Otukpo, Benue, Nigeria. josephokopi@yahoo.com

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clinical manifestation of chronic hepatitis B (CHB) vary significantly, ranging from the spontaneous resolution of the infection to severe consequences, including the asymptomatic phase, development of hepatic failure, cirrhosis, end-stage liver disease, and hepatocellular carcinoma.[4] The disease progression of HBV infection is a consequence of combined factors, including the host immune response viral factors, as well as age, sex, and environmental factors.[5] Individuals with CHB have a relatively high lifetime risk of severe adverse outcomes such as HBV reactivation prior to exposure to the virus, especially while undergoing immunosuppressive therapy.[6] Early diagnosis and initiation of antiviral treatment for those at risk are essential to prevent these clinical complications. In Nigeria, the prevalence of HBV infection is homogeneous across different populations and geopolitical zones. A recent national survey reported the prevalence of hepatitis B infection as 12.2%.[7] The pooled prevalence of HBV in Nigeria from different studies carried out among adults between 2000 and 2013 was 13.6%, and for children, it ranged from 1.2 to 15.5%.[8-10] The prevalence of HBV in Nigeria varied based on the screening methods used, with results ranging from 9% to 17.5%.[8] However, HBV infection is thus hyperendemic in Nigeria and maybe the highest in SSA. The prevalence among human immunodeficiency virus (HIV) infected and pregnant women ranged from 3% to 15%.[9-12]

Many studies have demonstrated that liver function markers such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), albumin (ALB), and bilirubin exhibit marked variation in different HBV-infected patients.[13] However, HBV deoxyribonucleic acid (DNA) replication and levels serve a central role in maintaining persistent infection and are associated with the extent of liver damage and severity.[14] It is also important to note that intervention through early detection of hepatic fibrosis, which is a pathological change caused by chronic liver damage, is critical to the management of HBV infection. The early stage of hepatic fibrosis is reversible, and therefore, the prevention and control of early liver fibrosis are of great significance. Transient elastography (TE) (Fibroscan, Echosens, Paris, France) is a non-invasive technique conceived to indirectly assess liver fibrosis for measuring liver stiffness (LS).[15,16] The scan is performed with an ultrasound transducer probe that produces vibrations of mild amplitude and low frequency, which induces an elastic shear wave that propagates through the liver tissue.[16] The velocity of the shear wave is directly related to liver tissue stiffness; the harder the tissue is, the faster the shear wave propagates.[16] The result is expressed as a pressure in kilopascals (kPa). The use of TE for the detection of early liver fibrosis remains a feasible strategy to identify and prevent disease progression in CHB patients.[17] The present study investigated liver fibrosis using TE in relation to liver function biomarkers and HBV DNA among HIV/HBV and HBV-mono-infected patients on long-term antiviral therapy.

MATERIAL AND METHODS

Study design

This was a cross-sectional study conducted at Faith Alive Foundation Jos, Bingham University Teaching Hospital Jos, Plateau State Specialist Hospital HIV Treatment Center, Jos, Nigeria, after obtaining Ethical approval from the Institutional Review Boards and written informed consent from the patients.

Description of participants

The study recruited HIV coinfected with HBV (HIV/HBV) and HBV-mono-infected adults aged ≥18 years between February 2018 to May 2019, who had been on tenofovir-based antiretroviral combination (HIV/HBV), and tenofovir monotherapy (HBV-mono-infected) for at least 12 months. Patients are classified as HIV/HBV-coinfected or HBV-mono-infected if the patient tested positive for hepatitis B surface antigen. All patients are tested for liver fibrosis using TE (Fibroscan), HBV DNA, hemoglobin, neutrophil, creatinine, ALT, aspartate transaminase (AST) levels, platelet count, bilirubin, and ALB at enrollment. HBV DNA was determined using the Roche COBAS® TaqMan® HBV Test (Roche Diagnostics GmbH, Mannheim, Germany) with a lower limit of detection of 10 copies/mL. At the time of HBV DNA quantification and TE testing, the HIV/HBV was on tenofovir based-ART and the HBV-mono on tenofovir monotherapy. Plasma HIV type 1 (HIV-1) ribonucleic acid (RNA) was quantified by RealTime HIV-1 assay (Abbott Diagnostics, UK). All laboratory tests were performed according to the manufacturer's specifications.

Measurement of LS by TE

TE was performed using portable equipment (Fibroscan, Echosens, France). Valid TE measurements were interpreted as follows: normal (F0–1 0–4), minimal fibrosis (F2 5–7.4), moderate (F3 7.5.9.4), severe fibrosis, and cirrhosis (F4 ≥9.5).[18] Blood samples were collected at the time of TE, CD4 cell counts, full blood counts, and serum biochemistry performed in the APIN laboratory at Jos University Teaching Hospital. The LS measurement in kPa was performed by trained physicians provided by the manufacturer.

Statistical analysis

The data obtained were analyzed using the Statistical Package for the Social Sciences version 20.2 Inc. Chicago, Illinois-USA for descriptive statistics, and continuous variables were presented as mean (standard deviation) or median (interquartile range [IQR]), as appropriate. Categorical variables were presented in numbers (percentage). Student t-test was used in a comparison of Fibrosis score values

between groups. $P < 0.05$ for a 2-sided test was considered to be statistically significant.

RESULTS

A total of 50 HIV/HBV and 51 HBV-mono-infected were enrolled during the study period, comprising 42.6% males and 57.4% females. The summary of the characteristics of the study population is presented in Table 1. The median age (IQR) among the HIV/HBV group was 40.5 (36.0–45.3), and among HBV-mono-infected individuals, it was 41.0 (35.0–49.0).

The overall prevalence of severe liver fibrosis (≥9.5 kPa) was 13/101 (13.0%), and among HIV/HBV-coinfected and HBV-mono-infected patients, it was 4/50 (8.0%) and 9/51 (17.6%), respectively. The plasma HBV-DNA was <20 copies/mL in 38 of 50 (76.0%) HIV/HBV and in 30 of 51 (58.8%) HBV-mono patients, 10/50 (20.0%) HIV/HBV and 19/51 (37.3%) HBV-mono patients had plasma HBV-DNA of 20–20000 copies/mL. LS, measured by TE (Fibroscan) and quantified in kPa, was scored as follows: F0/F1–1 0–4.9 as normal, F2 5–7.4 as minimal fibrosis, F3 7.5–9.4 as moderate fibrosis, and F4 ≥9.5 as severe fibrosis.

The prevalence of severe fibrosis (≥9.5%) in HIV/HBV coinfected was 4 (8.0%), and in HBV-mono-infected, it was 9 (17.6%). Thrombocytopenia was defined as a platelet count of $<150 \times 10^9$/L. The overall prevalence of thrombocytopenia was 4/101 (3.9%): 1/50 (2.0%) in HIV/HBV coinfected, $P = 0.029$ and 3/51 (5.9%) in the HBV-mono-infected group. The median HIV-RNA among the HIV/HBV was 33 (10–283), and the CD4 cell count was 497 (314.8–659.3) in HIV/HBV-coinfected. The median lymphocyte (IQR) was low in HBV-mono 46 cells/μL and 48 cells/μL in HIV/HBV-coinfected. Furthermore, the neutrophil count was low: 41 (34–51) in the HBV-mono-infected group and 47.5 (38.8–56.5) in the HIV/HBV coinfected group. The median platelet count was low: 195×10^9/L (168–257) in the HBV-mono-infected group, $P = 0.034$.

For fibroscan results

Three (6.0%) patients had F3, 7 (14.0%) in both HIV/HBV coinfected and HBV-mono-infected groups, and 4 (8.0%) patients had F4, while 9 (17.6%) in both HIV/HBV coinfected and HBV-mono-infected groups, respectively.

Table 1: Characteristics of HIV/HBV and HBV-mono-infected patients on long-term tenofovir therapy in.

Characteristics	HIV-HBV Coinfection (*n*=50)	HBV Mono-infection group (*n*=51)	*P*-value
Age, and, median (IQR)	40.5 (36.0–45.3)	41.0 (35.0–49.0)	
Sex	Male 19, Female 31	Male 24, Female 27	
HBV DNA, copies/median (IQR)	1 (1–44)	20 (1–121)	
<20	38 (76.0)	30 (58.8)	0.155
20–20000	10 (20.0)	19 (37.3)	
>20000	2 (4.0)	2 (3.9)	
Log_{10} HBV DNA copies/mL, median (IQR)	0 (0–1.5)	0 (0–2.1)	
Fibroscan score, kPa, categorized, No. (%)			
F0/F1 Normal (<5)	24 (48.0)	13 (25.5)	0.072
F2 Minimal fibrosis (5–7.4)	19 (38.0)	22 (43.1)	
F3 Moderate (7.5–9.4)	3 (6.0)	7 (13.7)	
F4 Severe fibrosis (≥9.5)	4 (8.0)	9 (17.7)	
Fibroscan score, kPa, categorized, No. (%)			
Not significant (<9.3)	46 (92.0)	42 (82.4)	0.234
Significant fibrosis (≥9.3)	4 (8.0)	9 (17.6)	
RNA viral load, median (IQR)	33 (10–283)	-	0.28
CD_4 count, cells/μL, median (IQR)	497.5 (314.8–659.3)	-	0.62
Hemoglobin, g/dL, median (IQR)	13.1 (12–14)	13 (13–14)	0.647
WBC, cells/μL, median (IQR)	6.0 (4.9–7.6)	5.9 (5–6.9)	0.067
Lymphocytes, cells/μL, median (IQR)	48 (38.0–54.5)	46 (35–50)	0.706
Neutrophil, (10^9/L) median (IQR)	47.5 (38.8–56.5)	41 (34–51)	0.645
Platelet (10^9/L), median (IQR)	259 (198.3–298.8)	195 (168–257)	0.034
Creatinine, μMol/L, median (IQR)	65.5 (55.5–76.3)	70 (57–85)	0.078
ALT, (μ/L), median (IQR)	26.1 (19.8–35.5)	27 (20–41)	0.538
AST, (μ/L), median (IQR)	28 (23.8–36.0)	27 (19–32)	0.334
Albumin, g/dL, median (IQR)	4.8 (4.5–5.0)	4.6 (4.0–4.9)	0.067
Bilirubin, mg/dL, median (IQR)	1.3 (1.2–1.5)	1.4 (1.3–1.6)	0.058
Urea, mg/dL, median (IQR)	21 (15.8–27.3)	20 (15–23)	0.235

IQR: Interquartile range, HIV: Human immunodeficiency virus, HBV: Hepatitis B virus, DNA: Deoxyribonucleic acid, WBC: White blood count, ALT: Alanine aminotransferase, kPa: Kilopascals, RNA: Ribonucleic Acid, AST: Aspartate Aminotransferase

In comparison to the age category, the result showed no significant difference, but severe liver fibrosis was found more in age 38–47 years in HIV/HBV coinfected group (2 [8.3%]) and HBV-mono-infected group (4 [25.0%]). For the sex category, severe liver fibrosis was observed in females: 4 (12.9%) in HIV/HBV coinfected group and 5 (18.5%) in HBV-mono-infected group. The use of alcohol was also compared between the two groups. Severe liver fibrosis was more prevalent in those who reported alcohol consumption in the HBV-mono-infected group (5 [19.2%]), showing a statistically non-significant difference [Table 2]. When comparing patients with different liver fibrosis stages and HBV DNA, although most patients in both groups had viral loads of 20–20000 copies/mL [Table 3] with no statistically significant difference, a significant difference was observed in relation to severe liver fibrosis and thrombocytopenia in HIV/HBV coinfected patients (P = 0.029) [Table 4].

DISCUSSION

CHB infection mostly leads to liver disease, and the prognosis and management depend greatly on the amount and progression of liver fibrosis. The assessment of liver fibrosis by TE is considered an important factor to reliably rule out cirrhosis.[19] Liver-related decompensation and mortality are expected to rise over time due to the incidence of advanced liver fibrosis in sub-Saharan population.[20] Therefore, the correct and early evaluation of liver fibrosis is fundamental to the management of chronic liver disease and associated complications. Liver biopsy, an invasive method, is the gold standard in the assessment of liver fibrosis. However, non-invasive methods such as fibroscan have now been

Table 2: Liver stiffness measurement associated with age categories among HIV/HBV and HBV-mono-infected patients on long-term tenofovir therapy in Jos.

Age category	No. of sample (%)	HIV-HBV (%)				HBV (%)			
		18–27 years	28–37 years	38–47 years	>47 years	18–27 years	28–37 years	38–47 years	>47 years
F0/F1 Normal (<5)	60 (59.4)	0 (0.0)	13 (68.4)	17 (70.8)	6 (85.7)	1 (50.0)	6 (40.0)	11 (68.8)	6 (40.0)
F2 Minimal fibrosis (5–7.4)	18 (17.8)	3 (100.0)	3 (15.8)	4 (16.7)	0 (0.0)	0 (0.0)	4 (26.7)	1 (6.2)	3 (20.0)
F3 Moderate (7.5–9.4)	10 (9.9)	0 (0.0)	2 (10.5)	1 (4.2)	0 (0.0)	0 (0.0)	4 (26.7)	0 (0.0)	3 (20.0)
F4 Severe fibrosis (≥9.5)	13 (12.9)	0 (0.0)	1 (5.3)	2 (8.3)	1 (14.3)	1 (50.0)	1 (6.6)	4 (25.0)	3 (20.0)
P-value			0.797				0.166		
Sex		HIV-HBV (%)				HBV-mono (%)			
		Male		Female		Male		Female	
F0/F1 Normal (<5)	60 (59.4)	13 (68.4)		23 (74.2)		11 (45.8)		13 (48.2)	
F2 Minimal fibrosis (5–7.4)	18 (17.8)	3 (15.8)		4 (12.9)		6 (25.0)		5 (18.5)	
F3 Moderate (7.5–9.4)	10 (9.9)	3 (15.8)		0 (0.0)		3 (12.5)		4 (14.8)	
F4 Severe fibrosis (≥9.5)	13 (12.9)	0		4 (12.9)		4 (16.7)		5 (18.5)	
P-value			0.058				0.953		
Alcohol Use		Yes (%)		No (%)		Yes (%)		No (%)	
F0/F1 Normal (<5)	60 (59.4)	7 (58.3)		29 (76.3)		12 (46.2)		12 (48.0)	
F2 Minimal fibrosis (5–7.4)	18 (17.8)	1 (8.3)		6 (15.8)		4 (15.4)		7 (28.0)	
F3 Moderate (7.5–9.4)	10 (9.9)	2 (16.7)		1 (2.6)		5 (19.2)		2 (8.0)	
F4 Severe fibrosis (≥9.5)	13 (12.9)	2 (16.7)		2 (5.3)		5 (19.2)		4 (16.0)	
P-value			0.154				0.533		

HIV: Human immunodeficiency virus, HBV: Hepatitis B virus

Table 3: Liver stiffness measurement associated with HIV/HBV and HBV-mono-infected patients on long-term tenofovir therapy in Jos.

Fibrosis profile	No. of sample (%)	HIV-HBV (n=50) (%)			HBV (n=51) (%)		
		<20	20–20000	>20000	<20	20–20000	>20000
F0/F1 normal (<5)	60 (59.4)	30 (78.9)	5 (50.0)	1 (50.0)	14 (46.7)	9 (47.4)	1 (50.0)
F2 minimal fibrosis (5–7.4)	18 (17.8)	5 (13.2)	2 (20.0)	0 (0.0)	7 (23.3)	3 (15.8)	1 (50.0)
F3 moderate (7.5–9.4)	10 (9.9)	1 (2.6)	2 (20.0)	0 (0.0)	4 (13.3)	3 (15.8)	0 (0.0)
F4 severe fibrosis (≥9.5)	13 (12.9)	2 (5.3)	1 (10.0)	1 (50.0)	5 (16.7)	4 (21.1)	0 (0.0)
P-value			0.104			0.927	

HIV: Human immunodeficiency virus, HBV: Hepatitis B virus

Table 4: Liver stiffness measurement associated with platelet counts among HIV/HBV and HBV-mono-infected patients on long-term tenofovir therapy in Jos.

Fibrosis score	No. of sample	HIV-HBV			HBV		
		Thrombo-cytopenia	Normal	Thrombo-cytosis	Thrombo-cytopenia	Normal	Thrombo-cytosis
F0/F1 normal (<5)	60 (59.4)	0 (0.0)	35 (74.5)	1 (50.0)	1 (33.3)	23 (47.9)	0 (0.0)
F2 minimal fibrosis (5–7.4)	18 (17.8)	0 (0.0)	6 (12.8)	1 (50.0)	1 (33.3)	10 (20.8)	0 (0.0)
F3 moderate (7.5–9.4)	10 (9.9)	0 (0.0)	3 (6.4)	0 (0.0)	0 (0.0)	7 (14.6)	0 (0.0)
F4 severe fibrosis (≥9.5)	13 (12.9)	1 (100.0)	3 (6.4)	0 (0.0)	1 (33.3)	8 (16.7)	0 (0.0)
Total	101	1	47	2	3	48	0
P-value			0.029			0.750	

HIV: Human immunodeficiency virus, HBV: Hepatitis B virus

developed. Higher levels of LS measures and biochemical scores predict these events during treatment in HIV/HBV and HBV-mono-infected patients.[21]

This study presents the first analysis of liver fibrosis by TE (Fibroscan), and associated biomarkers of liver disease and HBV DNA among HIV/HBV-coinfected and HBV-mono-infected patients on long-term tenofovir therapy in Nigeria. However, few studies from Nigeria have assessed liver fibrosis using TE in HIV and HBV-coinfected patients,[22] as well as in Ghana,[23] Egypt,[24] Zambia,[25] and Canada.[26] In this study, the prevalence of liver fibrosis among HIV/HBV-coinfected (8.0%) and HBV-mono-infected patients (17.6%) was higher than a recently conducted study in Nigeria (3.0%) and lower than studies from other African countries[22-25] among HIV/HBV-coinfected patients. We observed more severe fibrosis in HIV/HBV and HBV-mono-infected patients aged 38–47 years; this agrees with an earlier study that clarified that severe fibrosis increases with age.[25] In the sex factor, the distribution showed female predominance among those with more severe liver fibrosis and no statistically significant association, $P = 0.58$ [Table 2]. This was in agreement with earlier findings that sex was not associated with a higher risk for fibrosis.[27] However, it does not corroborate that the male sex has a higher risk of fibrosis than the female. We also observed no significant difference between the groups regarding serum ALT, AST, hemoglobin, white blood count, lymphocytes, neutrophils, creatinine, ALB, and bilirubin. However, we found a statistically significant association with platelet count, which was lower in HBV-mono-infected patients. Serum ALB was lower among HBV-mono-infected patients, whereas total bilirubin was lower in HIV/HBV-coinfected patients, though these parameters were not analyzed in relation to liver fibrosis [Table 1]. This finding was not in agreement with an earlier study that showed a positive correlation of serum enzymes [28], but it corroborates with an earlier study[29] which does not show any significant association with serum enzymes. Furthermore observed, was alcohol intake, there was no significant difference between the HIV/HBV and HBV-mono-infected patients, and more patients had <5 kpa. We did not measure the quantity of alcohol intake, nor establish the impact of alcohol and liver fibrosis since the patients were not followed up. However, chronic and high alcohol consumption may lead to cirrhosis and is associated with a higher risk of liver disease progression to cirrhosis.[30]

In this study, although the HBV viral load showed no statistical significance, more HIV/HBV-coinfected patients had the lowest levels of HBV viral load compared to HBV-mono-infected patients, which had higher levels (20–20000 cps/mL), suggesting that the coinfected patients usually start tenofovir combination therapy earlier compared to HIV-mono-infected patients.[23] This also means that HIV-mono-infected patients should be encouraged to initiate tenofovir monotherapy earlier at designated treatment hospitals. Although there was no significant association with severe liver fibrosis, observed higher HBV-DNA levels in HBV-mono-infected patients with severe liver fibrosis than in those with mild–moderate liver fibrosis score. This was contrary to earlier reports that severe liver fibrosis was strongly correlated with a higher viral load.[31] However, recent findings in Ghana showed that HBV DNA load was strongly associated with TE measurements,[23] including another study in antiretroviral therapy (ART)-naïve HIV/HBV-cinfected subjects in Nigeria.[22] Similarly, a study in HBV-infected patients from Taiwan reported HBV DNA load was the strongest predictor of liver disease progression to cirrhosis over time.[31] Our results support the evidence of the benefits of tenofovir combination therapy on virological and clinical outcomes in CHB patients on ART.

In both HIV-coinfected with HBV and HBV-mono-infected patients, the prevalence of thrombocytopenia and liver fibrosis was more among HBV-mono, with no statistically significance association, except for HIV/HBV-coinfected patients. It is interesting to note, that thrombocytopenia is a common hematological disorder in patients with chronic liver disease, and the cause is multifactorial, including hypersplenism, alcohol consumption, medications, and nutritional deficiencies.[32] However, in some African

populations, the association of TE score and platelet count can be influenced by inflammatory conditions due to endemic parasite diseases such as malaria.[33] Furthermore, other possible causes include suppression of platelet production in the bone marrow, splenic sequestration of platelets, and decreased hematopoietic growth factor thrombopoietin activity.[34] Although mild-to-moderate thrombocytopenia is common among chronic hepatitis patients, it usually does not interfere with the management, may require a therapeutic approach to replace deficient factors to avoid further complications.[34,35] The study did not establish the impact of these associations, but previous reports have shown that decreased thrombopoietin production plays an important role in patients with advanced liver disease patients.[36] The significant difference among the HIV/HBV group may be due to HIV-induced thrombocytopenia, hepatitis, or other drug-related factors. However, this suggestion could be unlikely in the group, as most of the patients had normal platelet levels, with a high median value of 259 × 10^9/L and normal ranges of AST and ALT values. This study had some limitations, such as the inability to measure HBeAg as a surrogate marker for active HBV viral replication. The other limitations include a lack of data on smoking, alcohol consumption measurement including locally brewed alcohol (Burukutu), which are source of aflatoxin, and hepatotoxicity due to herbal formulations. Furthermore, the diagnostic inaccuracy of TE measures the shear wave speed through the liver indicating stiffness, but not exact amount of fibrosis, and waist circumference interference due to overweight.[37]

CONCLUSION

In this study, sex and thrombocytopenia were significantly associated with severe liver fibrosis among HIV/HBV-coinfected patients. The tenofovir regimen is a predictor of good clinical outcomes in HIV/HBV-coinfected HBV-mono-infected patients. With the observed substantial proportion (13.0%) of severe liver fibrosis among HIV/HBV-coinfected and HBV-mono-infected patients, which support the need for continuous routine HBV screening in the HIV clinics, and prioritize tenofovir use in the treatment of CHB patients. Therefore, further larger studies are needed to monitor CHB patients using TE and investigate the impact of long-term antiviral therapy on the liver.

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Ethical approval

The research / study approved by the Institutional Review Board at Jos University, Teaching Hospital, Jos, Nigeria, number JUTH/DCS/ADM/127/XXV/142, dated 02-05-2017.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors affirmed that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing and editing of the manuscript and no images were manipulated using AI.

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