Hepatitis B, HIV, and HBV-HIV Coinfection in a Nigerian Population and Gender-Specific Variations in Their Liver Function Parameters: Insights from a Study in Rivers State, Nigeria

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Abstract:

Background*: Liver dysfunction is a critical comorbidity in individuals infected with HIV and Hepatitis B Virus (HBV), particularly when coinfection occurs. This study aimed to evaluate liver function parameters across genders in patients with HIV, HBV, and HBV-HIV coinfection. This study was done to determine the liver function indicators in subjects who are human immunodeficiency (HIV) and Hepatitis B positive.* ***Methodology****: A total of 100 participants were categorized into control, HBV-positive, HIV-positive, and HBV-HIV coinfected groups. Liver function markers including total and direct bilirubin, AST, ALT, alkaline phosphatase, GGT, total protein, albumin, and globulin were measured and analyzed. Significant differences ($p < 0.05$) were observed in AST, alkaline phosphatase, and total protein across groups. One hundred (100) subjects made up of Twenty five (25) each for HIV Positive, Hepatitis B virus positive and HIV-Hepatitis B Virus coinfection Positive had their aspartate aminotransferase (AST), alanine aminotransferase (ALT),Gammma glutamyl transpeptidase (GGT), Alkaline Phosphatase (ALP), Bilirubin, and Proteins determined using VITROS 250/350/950/5,1 FS and 4600 Chemistry Systems and the VITROS 5600 Integrated System manufactured by Ortho-Clinical Diagnostics, Inc.* ***Results****: Females with HBV exhibited significantly higher GGT and alkaline phosphatase levels compared to males, suggesting gender-related hepatic vulnerability. The result showed that there was significant difference ($P<0.05$) in activities of AST, ALP, Total Protein and globulin concentrations in HIV Positive, Hepatitis B virus positive and HIV-Hepatitis B Virus coinfection Positive while there was no significant difference in bilirubin, albumin concentrations as well as GGT and ALT activities in HIV Positive, Hepatitis B virus positive and HIV-Hepatitis B Virus coinfection Positive. The result of the study has shown that there was liver dysfunction in HIV Positive, Hepatitis B virus positive and HIV-Hepatitis B Virus coinfection Positive.* ***Conclusion****: This study highlights the importance of gender-sensitive diagnostic and therapeutic strategies in managing liver-related complications in HIV and HBV-infected individuals.*

Keyword: *Liver function, HIV, Hepatitis B, aspartate aminotranferase (AST), alanine aminotranferase (ALT), Gammma glutamyl transpeptidase (GGT), Alkaline Phosphatase (ALP), Bilirubin, and Proteins.*

Introduction/Background: Hepatitis B Virus (HBV) and Human Immunodeficiency Virus (HIV) remain major global public health challenges, particularly in sub-Saharan Africa where the prevalence of both infections is high (WHO, 2022). Coinfection with HBV and HIV accelerates liver disease progression, complicating treatment and prognosis (Thio, 2009). Liver function tests (LFTs) are critical in monitoring hepatic injury and guiding therapeutic decisions. However, there is limited data on gender-based liver function variations in mono- and co-infected individuals in Nigeria. Previous studies have documented elevated liver enzymes among HIV and HBV-infected individuals (Nash et al., 2020; Otedo et al., 2018). Gender disparities in liver function outcomes have also been reported, with males typically exhibiting higher ALT and AST levels (Kim et al., 2021). Coinfection further complicates liver pathology due to synergistic cytopathic effects and antiretroviral drug hepatotoxicity (Chen et al., 2016). Coinfection exacerbates hepatic dysfunction, increasing morbidity (Soriano et al., 2017). However, there is limited data on gender-based liver function trends in coinfected populations within Nigeria. This study assessed liver function parameters across different gender groups among HBV-positive, HIV-positive, HBV-HIV coinfected, and healthy controls.

Methodology: A cross-sectional study was conducted involving 100 individuals categorized into control, HIV-positive, HBV-positive, and HBV-HIV coinfected groups. Blood samples were collected and analyzed for liver biomarkers: total bilirubin, conjugated bilirubin, AST, ALT, alkaline phosphatase, gamma-glutamyl transferase (GGT), total protein, albumin, and globulin. Statistical analyses were performed using ANOVA and post hoc tests to determine significance ($p<0.05$).

Materials and Method

Subjects: One hundred (100) subjects made up of Twenty five (25) each for HIV Positive, Hepatitis B virus positive and HIV-Hepatitis B Virus confection Positive while the control was made up of twenty five(25) HIV and Hepatitis B negative subjects.

Biochemical Studies: The biochemical analyses were done using VITROS 250/350/950/5,1 FS and 4600 Chemistry Systems and the VITROS 5600 Integrated System manufactured by Ortho-Clinical Diagnostics, Inc.

Principles of the Alkaline phosphatase Procedure: The VITROS ALKP Slide is a multilayered, analytical element coated on a polyester support. A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. The spreading layer contains the *p*-nitrophenyl phosphate substrate and other components needed for the reaction. The ALKP in the sample catalyzes the hydrolysis of the *p*-nitrophenyl phosphate to *p*-nitrophenol at alkaline pH. The *p* -nitrophenol diffuses into the underlying layer, and it is monitored by reflectance spectrophotometry. The rate of change in reflection density is converted to enzyme activity.

Principles of the AST Procedure: A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. In the assay for aspartate aminotransferase, the amino group of L-aspartate is transferred to α-ketoglutarate in the presence of pyridoxal-5-phosphate (P-5-P) to produce glutamate and oxaloacetate. The oxaloacetate formed in the deamination of the L-aspartate is converted to pyruvate and carbon dioxide by oxaloacetate decarboxylase. Pyruvate is oxidized to acetylphosphate and hydrogen peroxide by pyruvate oxidase. The final reaction step involves the peroxidase-catalyzed oxidation of a leuco dye to produce a colored dye. The rate of oxidation of the leuco dye is monitored by reflectance spectrophotometry. The rate of change in reflectance density is proportional to enzyme activity in the sample. The low wavelength cutoff filter on the slide support minimizes the blank rate effects of incident light during the dye development.

Principles of the ALT Procedure: The VITROS ALT Slide is a multilayered, analytical element coated on a polyester support. A drop of patient sample was deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. The spreading layer contains the ALT substrates L-alanine and sodium α-ketoglutarate. Alanine aminotransferase catalyzes the transfer of the amino group of L-alanine to α-ketoglutarate to produce pyruvate and glutamate. Lactate dehydrogenase (LDH) then catalyzes the conversion of pyruvate and NADH to lactate and NAD^+. The rate of oxidation of NADH is monitored by reflectance spectrophotometry. The rate of change in reflection density is proportional to enzyme activity.

Principles of the GGT Procedure
The VITROS GGT Slide is a multilayered, analytical element coated on a polyester support.
A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. GGT catalyzes the transfer of the γ-glutamyl portion of L-γ-glutamyl-*p*-nitroanilide to glycylglycine, simultaneously producing *p*-nitroaniline. The rate of change in reflection density is measured and is used to calculate the enzyme activity of GGT.

Principles of the Total Protein Procedure: The VITROS TP Slide is a multilayered, analytical element coated on a polyester support. The method of analysis is based on the biuret reaction,(Kingsley 1942) which produces a violet complex when protein reacts with cupric ion (Cu^{+2}) in an alkaline medium. A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. When the fluid penetrates the reagent layer, the reagent diffuses up to the spreading layer and reacts with protein. The reaction between protein and copper tartrate takes place largely in the spreading layer where the protein is confined because of its high molecular weight. The amount of colored complex formed is proportional to the amount of total protein in the sample and is measured by reflectance spectrophotometry.

Principles of the Albumin Procedure: The VITROS ALB Slide is a multilayered, analytical element coated on a polyester support.A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. When the fluid penetrates the reagent layer, the bromcresol green (BCG) dye diffuses to the spreading layer and binds to albumin from the sample. This binding results in a shift in wavelength of the reflectance maximum

of the free dye. The color complex that forms is measured by reflectance spectrophotometry. The amount of albumin-bound dye is proportional to the concentration of albumin in the sample.

Principles of the Bilirubin Procedure:A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. Aided by caffeine and sodium benzoate in the spreading layer, Bu dissociates from albumin and migrates with Bc through the masking layer to the reagent layer. Proteins (including the albumin-bound delta bilirubin and hemoglobin), as well as lipids and lipochromes, are retained in the spreading layer. The masking layer optically blocks potentially interfering compounds trapped in the spreading layer, thereby preventing them from being measured. In the reagent layer, Bu and Bc bind to a cationic mordant. As a result, the absorbance peaks of the bilirubin fractions are shifted, and molar extinction coefficients are significantly increased. Bilirubin mono- and di-glucuronides, when bound to the mordant, have identical spectra and are quantitated together as Bc. In the vicinity of 400 to 420 nm, Bu and Bc have similar molar absorptivities; at 460 nm, Bu has a higher molar absorptivity than Bc. Because of these unique spectral characteristics, the reflection densities at two wavelengths, 400 and 460 nm, are used to determine the concentrations of Bu and Bc. With VITROS Chemistry and Integrated Systems, TBIL, Bu, and Bc are directly measured.
Other, more conventional bilirubin methods, especially those employing diazo chemistry, directly measure both total bilirubin and the direct bilirubin fraction (approximately equivalent to Bc), but cannot directly measure unconjugated bilirubin (Bu). For these diazo chemistry methods, unconjugated bilirubin (Bu) is determined by using a calculation, once the total bilirubin and the direct bilirubin measurements are known (Indirect bilirubin = Total Bilirubin - Direct Bilirubin). While this approach is certainly a viable alternative, it is important to recognize that variation and errors in determination of either of the measured fractions may contribute significantly to errors in the estimation of the indirect bilirubin. Because unconjugated bilirubin (Bu) is directly measured on VITROS Chemistry and Integrated Systems, lower analytical variation in the determination of unconjugated bilirubin (Bu) is achievable when using the VITROS BuBc Slides. VITROS Chemistry and Integrated Systems use two types of VITROS Slides (TBIL and BuBc) to determine the concentration of bilirubin components in human serum.
Total bilirubin (TBIL) is measured by the TBIL Slide as the diazo derivatives of unconjugated Bu, conjugated, and covalently protein-bound “delta” bilirubins. Conjugated and unconjugated bilirubins are measured simultaneously on the BuBc Slide using a dual-wavelength spectral measurement. Direct bilirubin values are calculated on the systems using these measured results and the following equation: direct bilirubin =TBil-Bu

Results: Control males exhibited higher liver function values than control females in all parameters. In HBV-HIV coinfection, males showed markedly elevated bilirubin but lower AST and alkaline phosphatase compared to females. HIV-positive females had higher AST, GGT, and alkaline phosphatase than males. In HBV-positive individuals, females demonstrated the highest GGT (191 ± 73.44 U/L) and alkaline phosphatase (152.27 ± 44.49 U/L) levels. Significant group differences were found in AST ($F=4.962$, $p=0.000$), alkaline phosphatase ($F=6.042$, $p=0.000$), and total protein ($F=3.341$, $p=0.003$). Post hoc analysis revealed key differences between control and HBV-HIV coinfected groups.
The total bilirubin (Umol/l) of control male was 9.18+1.45 and 6.93+0.87 in female control. The total bilirubin (Umol/l) of 18.92+14.92, 5.23+1.00, 7+1.68, 5+0.53, 6.07+0.58 and 17.55+10.27 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV

positive female, HBV positive male and HBV positive female respectively. The Conjugated bilirubin (Umol/l) of control male was 0.00+0.00 and 0.00+0.00in female control. The Conjugated bilirubin (Umol/l) of 9.08+8.36, 0.46+0.21, 0.00+0.00, 0.00+0.00, 0.00+0.00 and 7.63+7.63 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively. The direct bilirubin (Umol/l) of control male was 5.72+1.32 and 3.43+0.75 in female control. The direct bilirubin (Umol/l) of 3.25+2.05, 0.70+0.51, 2.25+0.85, 1.09+0.28, 2.21+0.51 and 4.18+2.11 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively.

The AST (U/l) of control male was 33.18+1.34 and 26.71+2.28 in female control. The AST (U/l)) of 17.83+2.81, 22.84+5.93, 42.75+7.46, 48.23+5.11, 36.14+2.50 and 50.72+11.38 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively. The ALT (U/l) of control male was 27.36+2.54 and 22.29+2.06 in female control. The ALT (U/l)) of 28.33+4.59, 24.23+2.95, 29.75+16.32, 23.05+3.62, 29.57+2.68 and 26.64+4.19 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively. The Alkaline Phosphatase (U/l) of control male was 73.64+6.32 and 66.57+6.74 in female control. The Alkaline Phosphatase (U/l)) of 10+1.00, 19.38+6.23, 128.50+38.21, 105.29+18.18, 74.64+9.57 and 152.27+44.49 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively. The GGT (U/l) of control male was 43.55+7.58 and 29+4.04 in female control. The GGT (U/l)) of 100.42+33.77, 129.15+59.03, 47.50+9.28, 102.57+28.18, 55+6.82 and 191+73.44 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively.

The total protein (g/l) of control male was 78+2.25 and 73+1.25 in female control. The total protein (g/l) of 70.08+2.50, 76.69+2.36, 88.25+5.94, 81.61+3.02, 82.21+2.12 and 81.18+2.87 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively. The albumin (g/l) of control male was 43.55+1.21 and 41.21+1.06 in female control. The albumin (g/l) of 38.92+1.83, 41.16+1.19, 47+1.47, 43.09+2.02, 44.86+0.79 and 40+1.80 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively. The globulin (g/l) of control male was 34.46+1.23 and 31.79+0.89 in female control. The globulin (g/l) of 31.17+2.79, 35.54+2.80, 41.25+6.63, 38.47+2.25, 37.35+2.02 and 41.18+3.34 was obtained in HBV-HIV coinfected male, HBV-HIV coinfected female, HIV positive male, HIV positive female, HBV positive male and HBV positive female respectively.

The total bilirubin in control was 7.92+0.81 in control,while it was 11.80+7.16, 5.32+0.53 and 11.12+4.56 in HBV-HIV Coinfection, HIV Positive and HBV Positive respectively ($P>0.05$) as shown below.The conjugated bilirubin concentrations were 0.00+0.00, 4.60+4.02, 0.00+0.00 and 4.60+4.02 in control, HBV-HIV Coinfection, HIV Positive and HBV Positive respectively ($P>0.05$). Direct bilirubin concentrations were 4.44+0.74, 1.52+1.05, 1.28+0.28 and 3.08+0.97 in control, HBV-HIV Coinfection, HIV Positive and HBV Positive respectively ($P<0.05$). The enzyme AST(U/L) had activities of 29.56+1.53, 20.44+3.33, 47.36+4.43 and 42.56+5.27 in control, HBV-HIV Coinfection, HIV Positive and HBV Positive respectively ($P<0.05$) while the ALT(U/L) activities were 24.52+1.65, 26.20+2.66, 24.12+3.84 and 28.28+2.35 respectively for

control, HBV-HIV Coinfection, HIV Positive and HBV Positive (P>0.05). The enzyme GGT(U/L) had activities of 35.40+4.20, 115.36+34.14, 93.76+23.97 and 114.84+34.54 respectively for control, HBV-HIV Coinfection, HIV Positive and HBV Positive (P>0.05)while alkaline Phosphatase activities were 69.68+4.65, 14.88+3.32, 109+16.24 and 108.80+21.28 respectively for control, HBV-HIV Coinfection, HIV Positive and HBV Positive (P<0.05).The total Protein concentration (g/l) was 75.2+1.29, 73.52+1.81, 82.68+2.72 and 81.76+1.70 respectively for control, HBV-HIV Coinfection, HIV Positive and HBV Positive (P<0.05) while albumin(g/l) had concentrations of 42.24+0.82, 40.08+1.08, 43.72+1.73 and 42.72+1.01 respectively for control, HBV-HIV Coinfection, HIV Positive and HBV Positive (P>0.05).Globulin had concentrations of 32.96+0.77, 33.44+1.99, 38.92+2.11 and 39.04+1.86 respectively for control, HBV-HIV Coinfection, HIV Positive and HBV Positive (P<0.05) as shown in table 1 below.

Table 1: Liver Function Parameters in Hepatitis B and HIV Positive Individuals

Group	**TB (µmol/L)**	**CB (µmol/L)**	**DB (µmol/L)**	**AST (U/L)**	**ALT (U/L)**	**ALP (U/L)**	**GGT (U/L)**	**TP (g/L)**	**Albumin (g/L)**	**Globulin (g/L)**
Control	7.92±0.81	0.00±0.00	4.44±0.74	29.56±1.53	24.52±1.65	69.68±4.65	35.40±4.20	75.2±1.29	42.24±0.82	32.96±0.77
HBV-HIV Coinf.	11.80±7.16	4.60±4.02	1.52±1.05	20.44±3.33	26.20±2.66	14.88±3.32	115.36±34.14	73.52±1.81	40.08±1.08	33.44±1.99
HIV Positive	5.32±0.53	0.00±0.00	1.28±0.28	47.36±4.43	24.12±3.84	109.00±16.24	93.76±23.97	82.68±2.72	43.72±1.73	38.92±2.11
HBV Positive	11.12±4.56	3.36±3.36	3.08±0.97	42.56±5.27	28.28±2.35	108.80±21.28	114.84±34.54	81.76±1.70	42.72±1.01	39.04±1.86
F	0.494	0.807	3.251	9.880	0.477	10.574	1.923	5.579	1.619	3.589
Pvalue	0.687	0.493	0.025	0.000	0.699	0.000	0.131	0.001	0.190	0.016

Liver Function Parameters in Different Gender in Hepatitis B and HIV positive individuals

Group	Gender	Total Bilirubin (umol/l)	Conjugated Bilirubin (umol/l)	Direct Bilirubin (umol/l)	AST (U/L)	ALT (U/L)	Alkaline Phosphatase (U/L)	GGT (U/L)	Total Protein (g/l)	Albumin (g/L)	Globulin (g/l)
Control	Male (a)	9.18+1.45	0.00+0.00	5.72+1.32	33.18+1.34	27.36+2.54	73.64+6.32	43.55+7.58	78+2.25	43.55+1.21	34.46+1.23
	Female(b)	6.93+0.87	0.00+0.00	3.43+0.75	26.71+2.28	22.29+2.06	66.57+6.74	29+4.04	73+1.25	41.21+1.06	31.79+0.89

HBV-HIV Coinfection	male (c)	18.92+14.92	9.08+8.36	3.25+2.05	17.83+2.81	28.33+4.59	10+1.00	100.42+33.77	70.08+2.50	38.92+1.83	31.17+2.79
	Female(d)	5.23+1.00	0.46+0.21	0.70+0.51	22.84+5.93	24.23+2.95	19.38+6.23	129.15+59.03	76.69+2.36	41.16+1.19	35.54+2.80
HIV Positive	Male(e)	7+1.68	0.00+0.00	2.25+0.85	42.75+7.46	29.75+16.32	128.50+38.21	47.50+9.28	88.25+5.94	47+1.47	41.25+6.63
	Female(f)	5+0.53	0.00+0.00	1.09+0.28	48.23+5.11	23.05+3.62	105.29+18.18	102.57+28.18	81.61+3.02	43.09+2.02	38.47+2.25
HBV Positive	male (g)	6.07+0.58	0.00+0.00	2.21+0.51	36.14+2.50	29.57+2.68	74.64+9.57	55+6.82	82.21+2.12	44.86+0.79	37.35+2.02
	Female(h)	17.55+10.27	7.63+7.63	4.18+2.11	50.72+11.38	26.64+4.19	152.27+44.49	191+73.44	81.18+2.87	40+1.80	41.18+3.34
	F	0.850	2.610	1.040	4.962	0.551	6.042	1.899	3.341	1.780	2.041
	P	0.549	0.017	0.409	0.000	0.793	0.000	0.078	0.003	0.101	0.058
Post Hoc											
	A vs b	.985	.	.952	.411	.945	1.000	.884	.750	.972	.860
	A vs c	1.000	.997	1.000	.004	1.000	.000	.897	.464	.636	.999
	A vs d	.542	.617	.030	.876	1.000	.000	.962	1.000	.979	1.000
	A vs e	.999	.	.579	.957	1.000	.919	1.000	.863	.803	.991
	A vs f	.295	.	.110	.196	1.000	.918	.698	1.000	1.000	.947
	A vs g	.712	.	.415	.999	1.000	1.000	.999	.985	1.000	.995
	A vs h	1.000	.999	1.000	.931	1.000	.844	.707	1.000	.915	.777
	b vs c	1.000	.997	1.000	.400	.995	.000	.645	.999	.999	1.000
	b vs d	.993	.617	.020	1.000	1.000	.001	.876	.980	1.000	.989
	b vs e	1.000	.	.998	.680	1.000	.861	.779	.507	.217	.918
	b vs f	.786	.	.191	.018	1.000	.717	.322	.283	1.000	.219

	b vs g	1.000	.	.987	.213	.605	1.000	.083	.030	.228	.371
	B vs h	0.998	.999	1.000	.664	1.000	.762	.584	.339	1.000	.307
	c vs d	1.000	.999	.924	1.000	1.000	.944	1.000	.765	1.000	.999
	c vs e	1.000	.997	1.000	.308	1.000	.354	.944	.381	.098	.934
	c vs f	1.000	.997	.999	.000	1.000	.001	1.000	.144	.957	.686
	c vs g	1.000	.997	1.000	.002	1.000	.000	.982	.030	.180	.842
	C vs h	1.000	1.000	1.000	.270	1.000	.162	.997	.179	1.000	.503
	d vs e	0.999	.617	.539	.655	1.000	.417	.976	.785	.226	1.000
	d vs f	1.000	.617	.711	.076	1.000	.004	1.000	.994	1.000	1.000
	d vs g	1.000	.617	.091	.665	.988	.002	.990	.875	.319	1.000
	d vs h	0.992	1.000	.734	.594	1.000	.224	1.000	.996	1.000	.991
	e vs f	0.981	.	.958	1.000	1.000	1.000	.809	.996	.936	1.000
	e vs g	1.000	.	1.000	.999	1.000	.935	1.000	.996	.967	1.000
	e vs h	0.999	0.999	1.000	1.000	1.000	1.000	.742	.991	.201	1.000
	f vs g	0.989	.	.758	.624	.977	.968	.917	1.000	1.000	1.000
	f vs h	0.990	.999	.956	1.000	1.000	1.000	.997	1.000	.999	1.000
	g vs h	.996	.999	1.000	.988	1.000	.866	.794	1.000	.414	1.000

Table 3: Post-Hoc Analysis of the Study

Group	Total Bilirubin (umol/l)	Conjugated Bilirubin (umol/l)	Direct Bilirubin (umol/l)	AST (U/L)	ALT (U/L)	Alkaline Phosphatase (U/L)	GGT (U/L)	Total Protein (g/l)	Albumin (g/L)	Globulin (g/l)
Control	7.92+0.81	0.00+0.00	4.44+0.74	29.56+1.53	24.52+1.65	69.68+4.65	35.40+4.20	75.2+1.29	42.24+0.82	32.96+0.77
Post Hoc										

Control vs HBV-HIV Coinfection	0.994	0.820	0.155	0.101	0.995	0.000	0.153	0.970	0.512	1.000
Control vs HIV	0.061	1.000	0.002	0.004	1.000	0.147	0.130	0.099	0.966	0.071
Control vs HBV	0.980	0.892	0.839	0.136	0.718	0.390	0.166	0.021	0.999	0.029
HBV-HIV Coinfection Vs HIV Positive	0.930	0.820	1.000	0.000	0.998	0.000	0.996	0.044	0.385	0.323
HBV-HIV Coinfection Vs HBV Positive	1.000	1.000	0.853	0.006	0.992	0.001	1.000	0.010	0.385	0.236
HIV Positive Vs HBV Positive	0.748	0.892	0.396	0.980	0.925	1.000	0.996	1.000	0.996	1.000

Discussion: Our findings corroborate earlier research linking HIV/HBV infection to hepatic impairment, with AST and alkaline phosphatase being particularly elevated in co-infected and HIV-positive individuals (Muriuki et al., 2019). Gender disparities, especially the elevated GGT and alkaline phosphatase in females, suggest possible hormonal or metabolic influences on hepatic response to infection (Marrone et al., 2020). These findings underscore the necessity for tailored treatment approaches in liver disease management among infected populations. Significant elevations in AST and ALP among HIV and HBV-positive individuals align with previous reports of hepatocellular damage and cholestasis in viral infections (Konopnicki et al., 2005). The high GGT levels in coinfected and HBV-positive individuals may reflect increased hepatic oxidative stress. Though total bilirubin and ALT did not differ significantly, variations suggest underlying hepatic dysfunction. Globulin was elevated among HIV and HBV patients, possibly due to chronic immune activation (Walker et al., 2020). No significant differences in albumin suggest

compensated hepatic synthetic function. Liver function alterations in HIV and HBV infections have been well documented. Studies show elevated liver enzymes in HIV patients due to antiretroviral therapy (ART) and HIV-associated hepatotoxicity (Mocroft et al., 2015). HBV infection leads to chronic hepatitis and cirrhosis, with elevated ALT and AST reflecting hepatocellular injury (Lemoine et al., 2016).

Conclusion: This study reveals significant gender-specific differences in liver function among HIV, HBV, and co-infected individuals. Females, especially those with HBV, are more prone to elevated liver enzymes, indicating a higher hepatic burden. The study highlights significant derangements in liver enzymes and protein profiles among HIV and HBV patients compared to controls. AST, ALP, and globulin were particularly affected, underscoring the need for hepatic surveillance. Liver function abnormalities were more pronounced in infected individuals, especially among HIV and HBV-positive subjects. Routine liver function monitoring should be incorporated in the management of HBV and HIV patients to guide therapeutic decisions.

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