JOURNAL OF BASIC AND APPLIED SCIENCES

Comparative Assessment of Hepatic Parameters Among HIV and Hepatitis B Positive Patients Attending the University of Port Harcourt Teaching Hospital, Port Harcourt, Rivers State.

ARTICLE INFORMATION

Original Article

Received: January 28,2026
Revised: May 26,2026
Accepted: May 26,2026
Published online:

Citation: Lawrence C. Chuku· Harrison O. Eruotor Okoye, Mitchelle Chukwudumebi Chigbu Chinenye Blessing, **and** Anyanwu Annabel (2026) Chibundu Comparative Assessment of Hepatic Parameters Among HIV and Hepatitis B Positive Patients Attending the University of Port Harcourt Teaching Hospital, Port Harcourt, Rivers State.

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Abstract

This research compared liver function in HIV-positive patients and those diagnosed with Hepatitis B for better understanding of the physiological impact these conditions would have on liver health. Liver function markers, such as Bilirubin, Total protein, Albumin, Alanine transaminase (ALT), Aspartate transaminase (AST), Alkaline phosphatase (ALP) and Gamma-Glutamyl Transferase (GGT) of the two disease populations and negative control groups were evaluated using standard methods. Results showed elevated levels of Bilirubin (32.14 ± 20.72µmol/L), AST (47.07 ± 17.14 U/L), ALT (35.46 ± 12.00 U/L) and ALP (149.82 ± 65.93 U/L), and a decreased Albumin level (26.60 ± 6.79 g/L), consistent with liver damage observed in Hepatitis patients. There were also significant differences between the levels of Bilirubin (39.55 ± 7.28 µmol/L), Total Protein (80.2 ± 4.52 g/L), Albumin (19.95 ± 2.45 g/L), ALT (51.95 ± 14.04 U/L), ALP (219.35 ± 31.92 U/L) and GGT (48.85 ± 4.55 U/L) in the HIV positive and Hepatitis positive patients and the negative control groups (Control Albumin: 33.85 ± 1.26 g/L), indicating that the parameters studied are likely influenced by HIV and hepatitis B status.

Keywords: HIV; Hepatitis B; Liver enzymes; Patient.

1. Introduction

Hepatitis B is an infectious disease that is caused by Hepatitis B virus (HBV). It affects the liver and can be acute or chronic (WHO, 2025a). About 3.5% of the global population is chronically infected with Hepatitis B Virus (Yuen et al., 2018), and the burden of infection is highest in the WHO Western Pacific Region and the WHO African Region, where 97 million and 65 million people, respectively, are chronically infected (WHO, 2025a). Hepatitis B virus can be transmitted through needle-stick injury, tattooing, piercing and exposure to infected blood and body fluids, such as saliva and menstrual, vaginal and seminal fluids. The probability of Hepatitis B infection becoming chronic depends on when the infection was acquired. Hepatitis B acquired during childhood leads to Chronic Infection in 95% of cases, whereas it is less than 5% if infection is acquired in adulthood (WHO, 2025a).

Human Immunodeficiency Virus (HIV) is a virus that attacks the immune system. It is deadly because it weakens the body's defense mechanism and leaves the patient vulnerable to further infections that ultimately lead to AIDS (acquired immunodeficiency syndrome). HIV can be transmitted through the exchange of body fluids from infected people, through breastmilk, semen, vaginal secretions, and blood (WHO 2025b).

The liver is a vital organ responsible for detoxification, protein synthesis and bile production in the body (Ozougwu, 2017). Both Human Immune Deficiency Virus (HIV) and Hepatitis viruses can infect and impair the proper functioning of the liver (Alfano et al., 2019). HIV infection can increase the risk of developing Hepatitis B or C co-infections, which can further accelerate liver damage (Crane et al., 2012).

Previous studies have shown that HIV infection can lead to liver abnormalities, even in individuals with well-controlled viral loads (Feleke et al., 2020). Different strains of Hepatitis viruses (A, B and C) have varying effects on the liver, causing inflammation, fibrosis (scarring)**,** and potentially, cirrhosis (severe scarring) (Feleke et al., 2020). Research has been conducted on the liver function of both HIV and Hepatitis individually, but limited data exists on a direct comparison.

Understanding how HIV and different Hepatitis strains impact liver function differently can help the discovery of newer and more efficient treatment strategies (Sherman et al., 2017). Identifying potential interactions between HIV and Hepatitis co-infections on liver health is crucial for managing these patients effectively (Lacombe & Rockstroh, 2012). This comparative analysis can provide valuable insights to improve patient care and potentially reduce liver-related complications in these populations.

The aim of this study is generally to investigate, compare and understand the individual Hepatic function of HIV-positive and Hepatitis B-positive patients to identify potential differences in the severity of hepatic damage caused by each condition, or of one in relation to the other.

The significance of this study lies in its potential to improve our understanding of how each of the viruses directly or indirectly affects liver and kidney function.

2. Materials and Methods

In the cross analytical study, 100 adult males and females diagnosed with HIV and HbsAg were used as the subjects. 5mls of blood were collected into heparinized anti-coagulant bottles from the HIV and Hepatitis phlebotomy unit. Each batch of collected samples was taken to the chemical pathology laboratory for analysis. The biochemical parameters were analyzed using spectrophotometric method and the corresponding kits. The procedures were carried out using the manual step-by-step procedure.

Inclusion: The study aims at HIV and HBsAg-positive patients.

Exclusion: HIV and HbsAg-negative control patients.

Statistical analysis

Statistical Analysis of the data obtained was done using analysis of variance (ANOVA), followed by Tukey's post-hoc test to compare between the study groups, and the results were presented as mean ± SEM. P-values of <0.05 were considered statistically significant.

3. Results and Discussion

3.1. RESULTS

Hepatic Parameters for HIV groups

Table 1: Levels of Hepatic Parameters for the HIV-positive group

Total Bilirubin	39.55 ± 7.28 µmol/L
Conjugated Bilirubin	13.7 ± 3.64 µmol/L
Total Protein	80.2 ± 4.52 g/L
Albumin	19.95 ± 2.45 g/L
AST	57.15 ± 16.53 U/L
ALT	51.95 ± 14.04 U/L
ALP	219.35 ± 31.92 U/L
GGT	48.85 ± 4.55 U/L

Values are represented as means ±STD of triplicate determinations

Table 2: Levels of Hepatic Parameters for the HIV-negative group (control group).

Total Bilirubin	6.85 ± 1.19 µmol/L
Conjugated Bilirubin	0.0 µmol/L
Total Protein	72.2 ± 1.68 g/L
Albumin	33.85 ± 1.26 g/L
AST	45.5 ± 60.52 U/L
ALT	21.0 ± 3.76 U/L
ALP	75.0 ± 6.18 U/L
GGT	16.35 ± 3.89 U/L

Values are represented as means ±STD of triplicate determinations

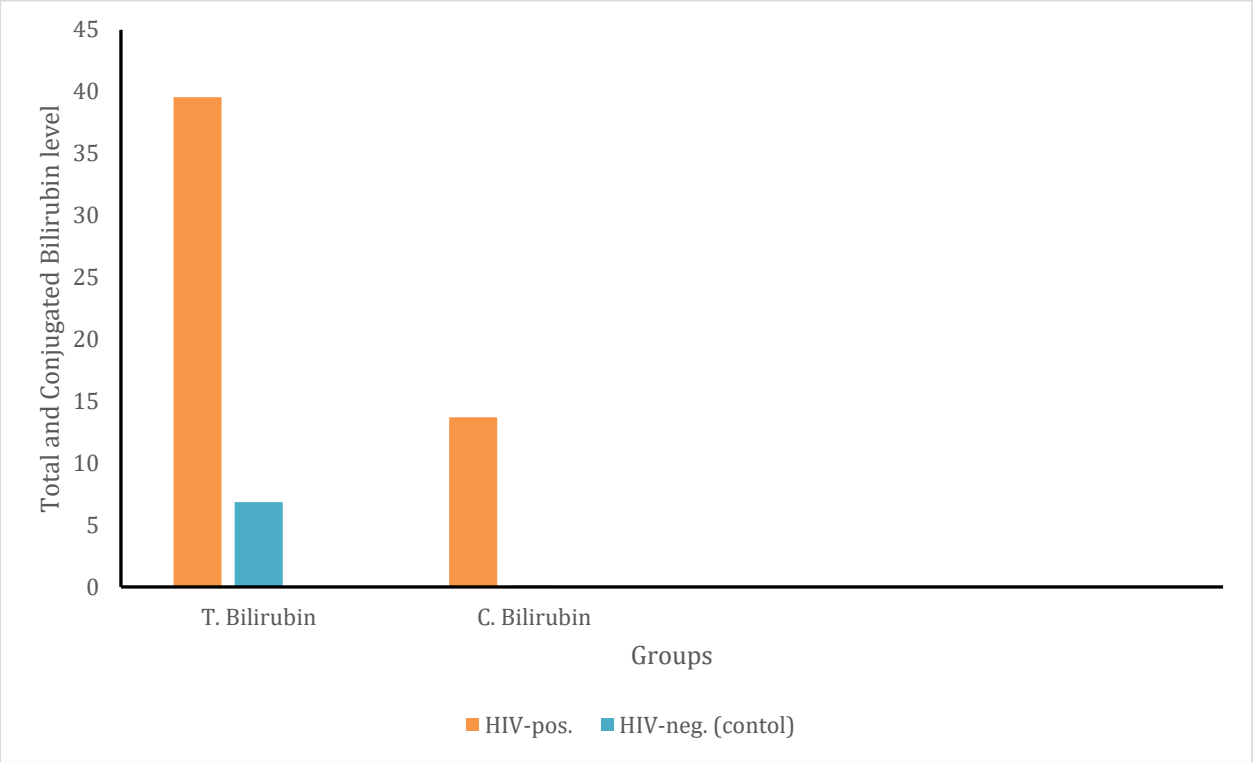

Figure 1: Total Bilirubin and Conjugate bilirubiin in HIV-positive compared to negative control group
Figure 1 shows a significant increase of total bilirubin and conjugate bilirubin in HIV patients when compared to negative control groups.

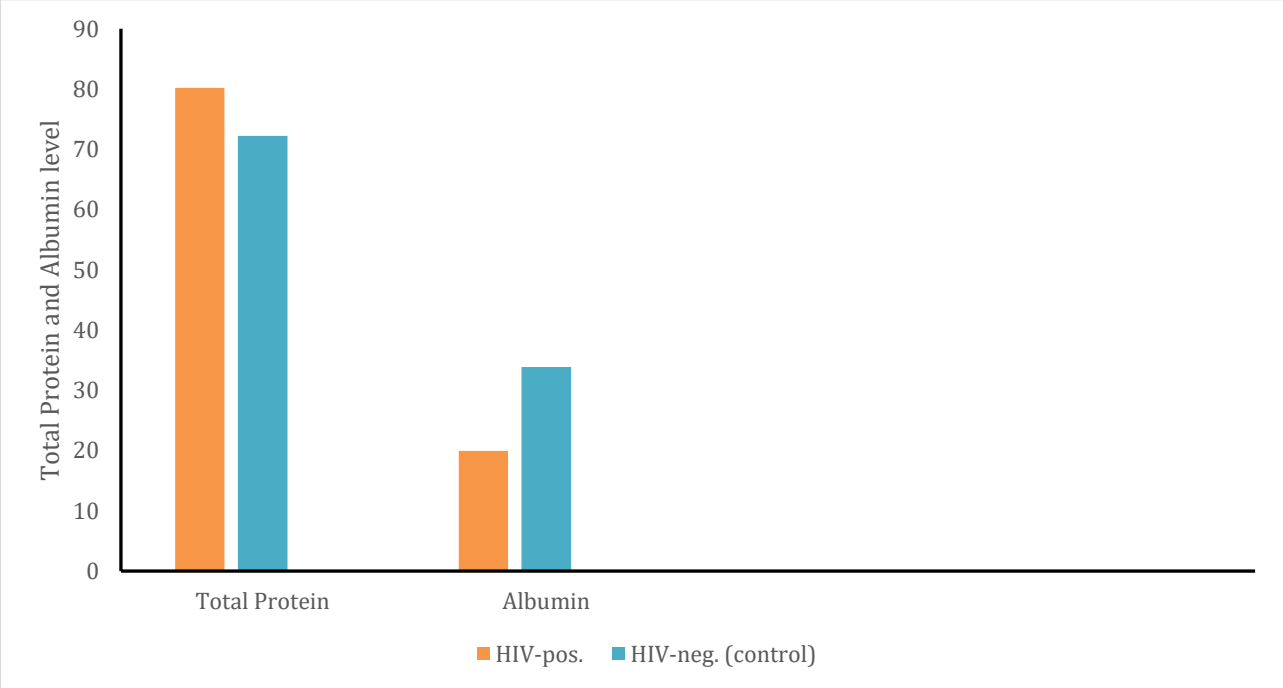

Figure 2: Total Protein and Albumin in HIV-positive compared to negative control group
Figure 2 shows a significant increase of total protein and decreased concentration of albumin in HIV patients when compared to negative control group.

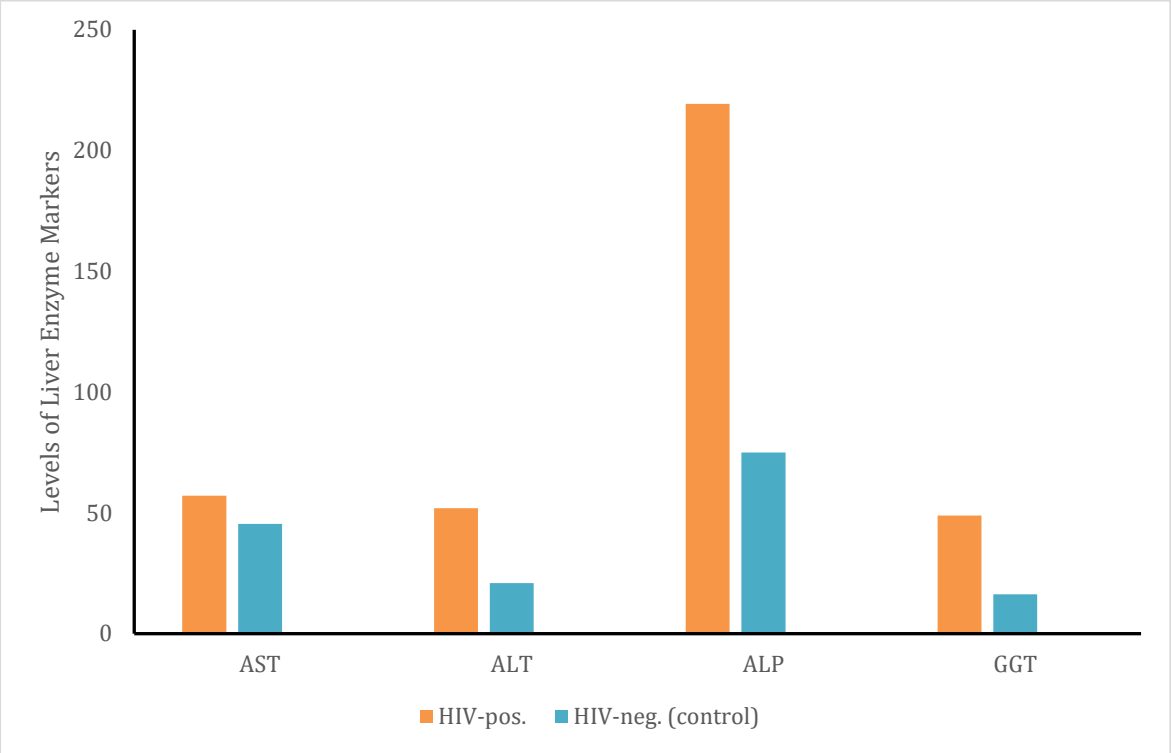

Figure 3: Liver enzyme markers in HIV-positive compared to negative control group

Figure 3 shows a significant increase of liver enzymes in HIV patients when compared to negative control groups.

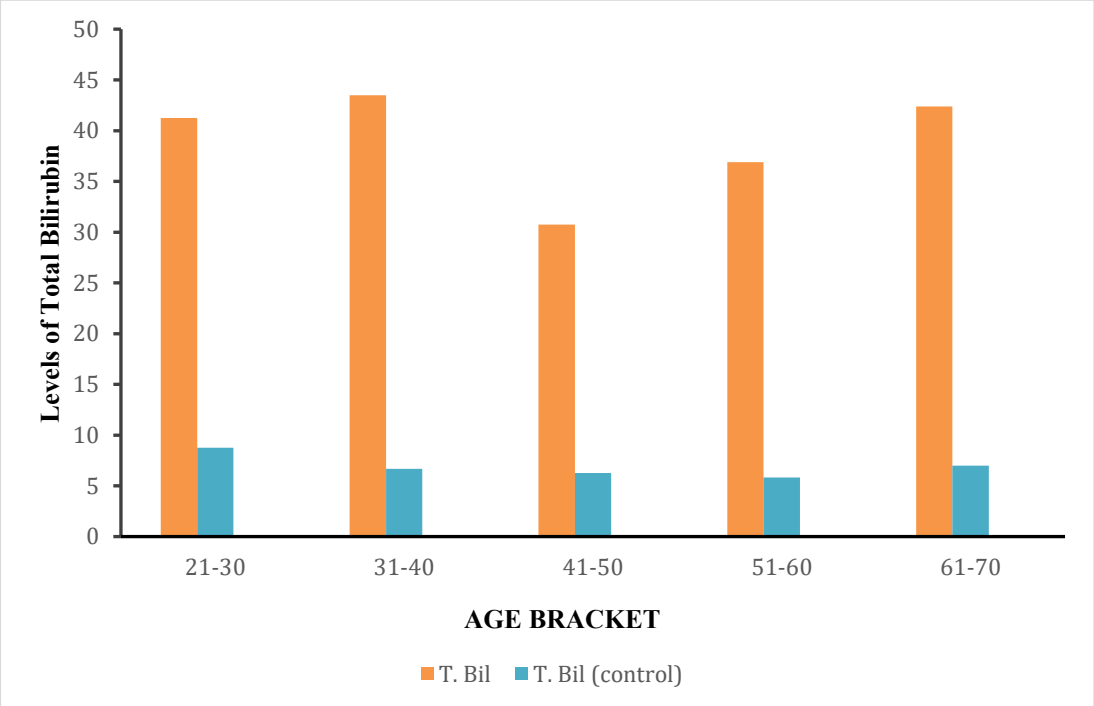

Figure 4: Total Bilirubin in HIV-positive compared to negative control group across different age brackets

Figure 4 shows a significant increase ($p<0.05$) of total bilirubin in HIV patients and within the age of 31-40 when compared to negative control groups.

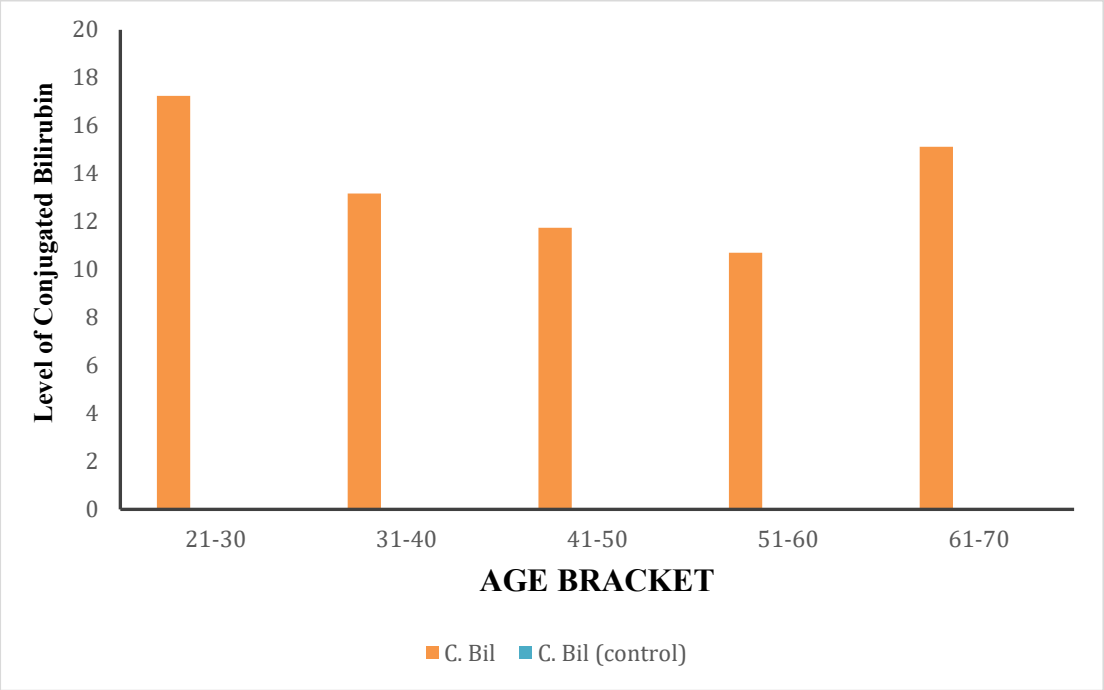

Figure 5: Total Bilirubin in HIV-positive compared to negative control group across different age brackets

Figure 5 shows a significant increase ($p<0.05$) of conjugate bilirubin in HIV patients and within the age of 21-30 when compared to negative control groups**.**

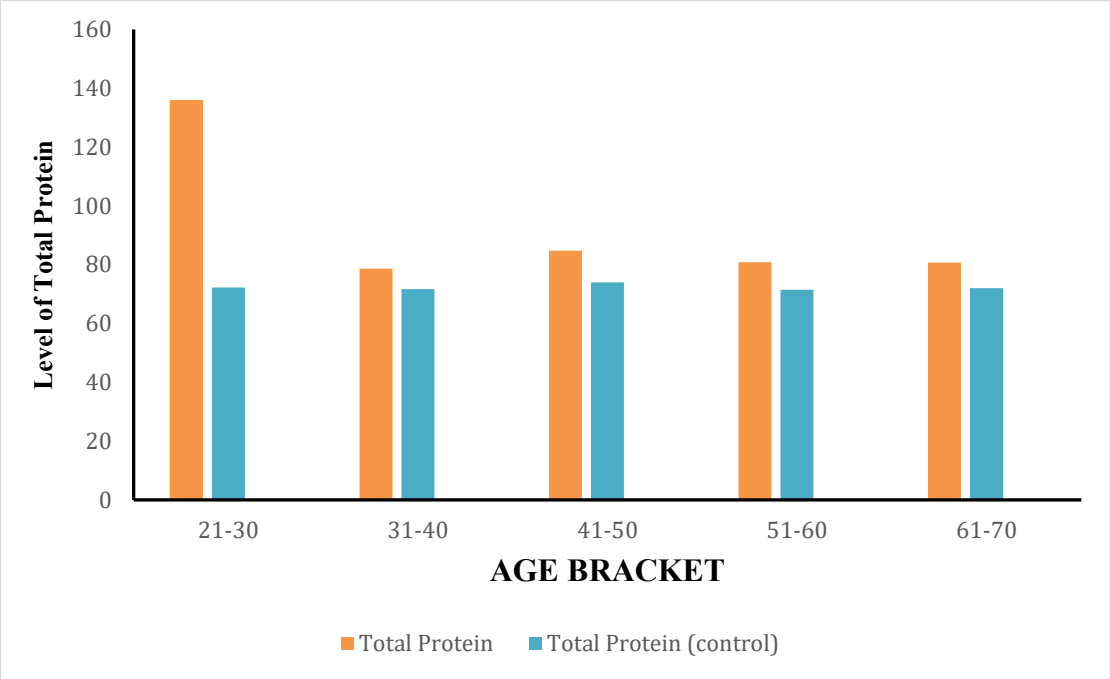

Figure 6: Total Protein in HIV-positive compared to negative control group across different age brackets

Figure 6 shows a significant increase of total protein in HIV patients and within the age of 21-30 when compared to negative control groups.

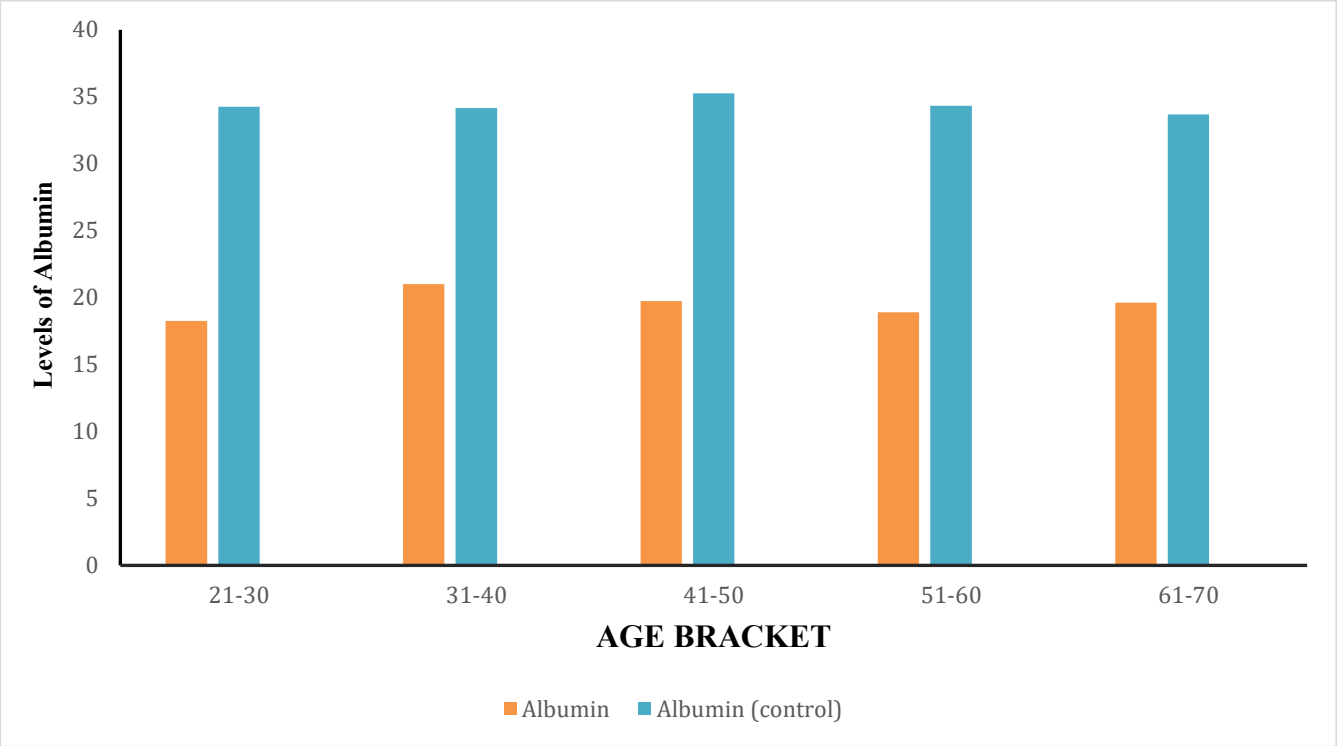

Figure 7: Total Bilirubin in HIV-positive compared to negative control group across different age brackets

Figure 7 shows a significant decrease ($p<0.05$) of albumin concentration in HIV patients within the age of 21-30 when compared to negative control group.

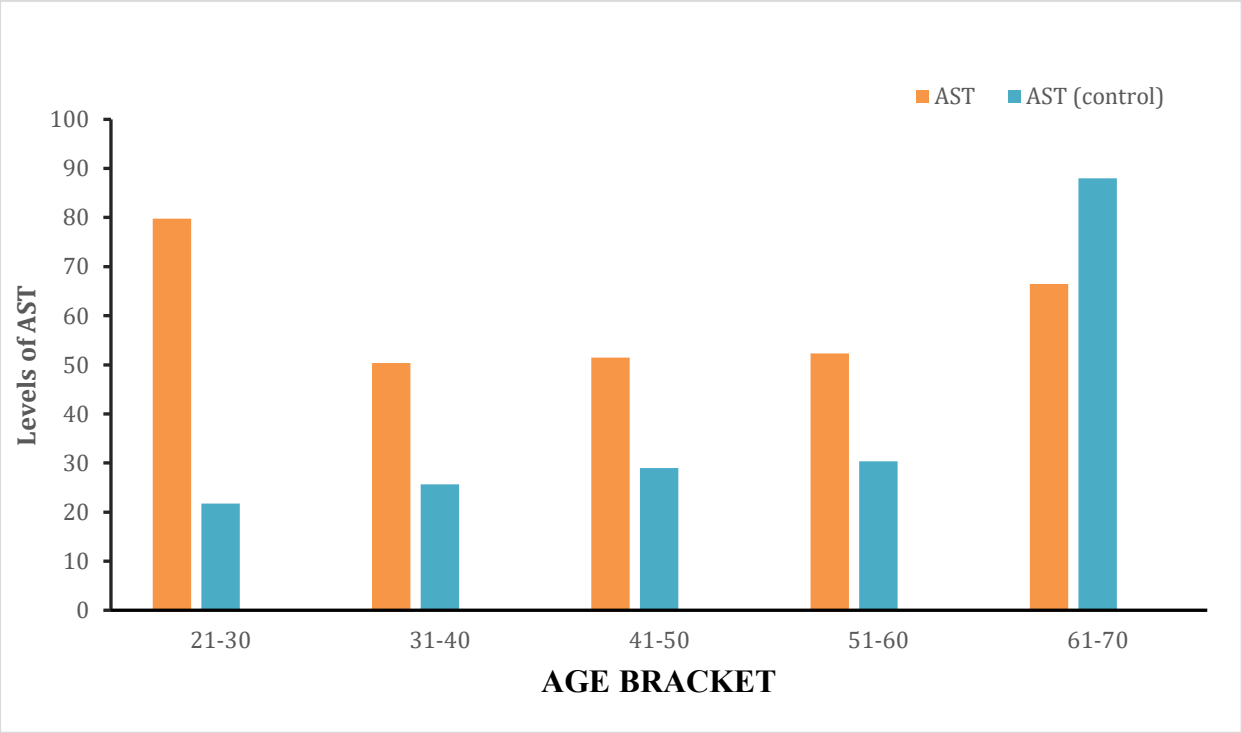

Figure 8: AST levels in HIV-positive compared to negative control group across different age brackets

Figure 8 shows a significant increase of AST activity in HIV patients and within the age of 21-30 when compared to negative control group.

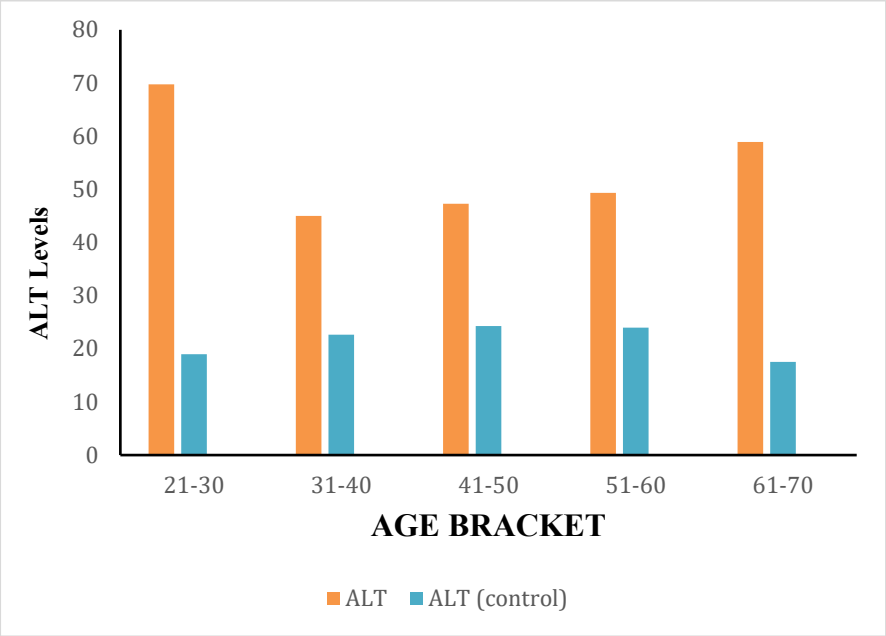

Figure 9: ALT levels in HIV-positive compared to negative control group across different age brackets

Figure 9 shows a significant increase of ALT activity in HIV patients and within the age of 21-30 when compared to negative control groups.

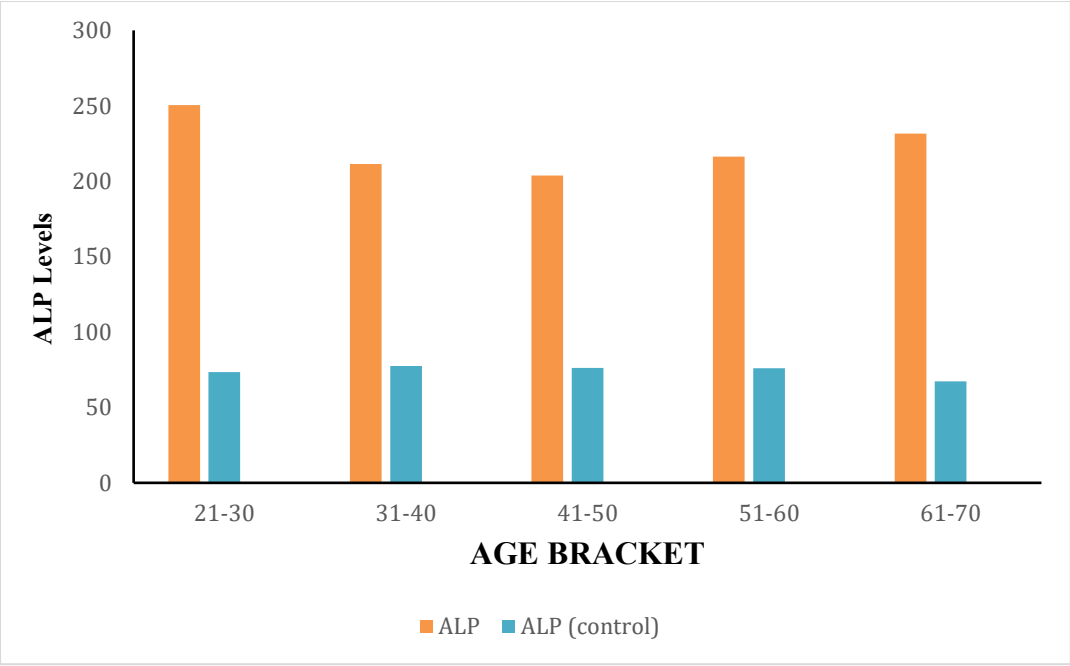

Figure 10: ALP levels in HIV-positive compared to negative control group across different age brackets

Figure 10 shows a significant increase of ALP activity in HIV patients and within the age of 21-30 when compared to negative control groups.

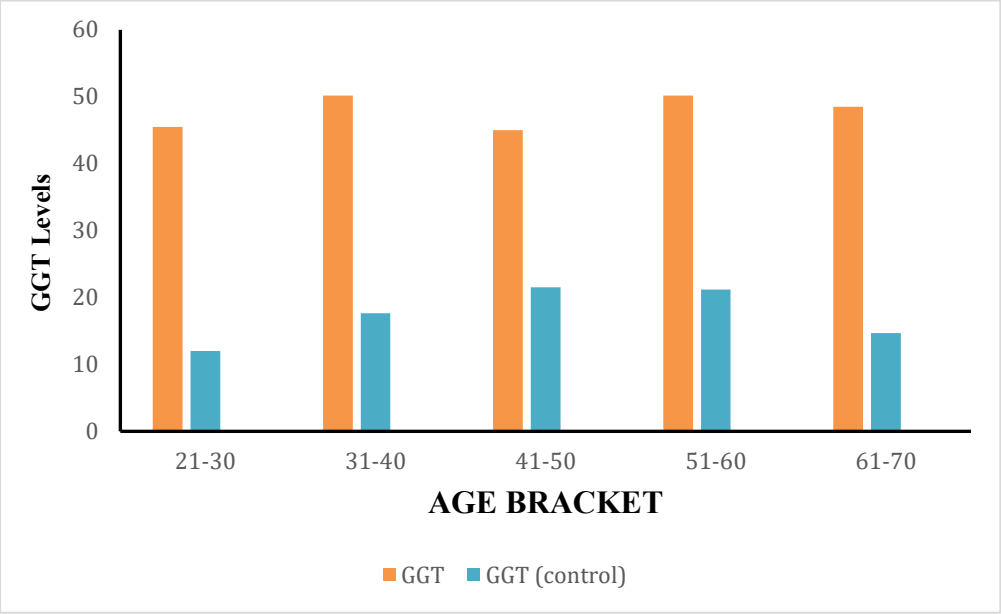

Figure 11: GGT level in HIV-positive compared to negative control group across different age brackets

Figure 11 shows a significant increase of GGT activity in HIV patients across all ages when compared to negative control groups.

Table 3: Hepatitis B Negative patients

Parameters	Mean ± SD
Age	51.5±8.46
T. BIL	16.2±14.96 µmol/L
C. BIL	2.4±3.70 µmol/L
TP	70±10.81g/L
ALB	28.67±7.12 g/L
AST	40.46±19.99 U/L
ALT	28.96±11.60 U/L
ALP	118.67±62.15 U/L
GGT	27.35±13.99 U/L

Values are presented in Mean ± Standard Deviation.

Table 4: Hepatitis B Positive

Parameters	Mean ± SD
Age	44.79 ± 11.61
T. BIL	32.14 ± 20.72 µmol/L
C. BIL	8.98 ± 7.61µmol/L
TP	77.81 ± 10.71 g/L
ALB	26.60 ± 6.79 g/L
AST	47.07 ± 17.14 U/L
ALT	35.46 ± 12.00 U/L
ALP	149.82 ± 65.93 U/L
GGT	34.34 ± 15.00 U/L

Values are presented in Mean ± Standard Deviation.

DISCUSSION

The present study demonstrated significant alterations in liver function parameters among HIV positive and Hepatitis B positive subjects compared with apparently healthy control subjects. The observed

elevations in serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and gamma glutamyl transferase (GGT) among infected subjects strongly suggest hepatic injury, hepatocellular inflammation, and impaired liver integrity associated with viral infections. These enzymes are important biochemical markers routinely used in the assessment of hepatic function because they are released into circulation following hepatocyte membrane damage or cholestatic injury.

The significantly elevated AST and ALT activities observed among HIV-positive subjects may indicate active hepatocellular damage and leakage of intracellular enzymes into the bloodstream. ALT is considered more liver-specific, whereas AST elevation may also reflect systemic tissue injury and metabolic stress associated with chronic HIV infection. The findings of this study are consistent with previous reports by Iroezindu et al. (2013), who documented abnormal liver enzyme activities among HIV-infected individuals receiving antiretroviral therapy. Similarly, studies by Anyanwu et al., (2020) reported significant elevations in transaminases among HIV-positive patients, suggesting progressive hepatic involvement during infection.

Several mechanisms may account for the hepatic dysfunction observed among HIV-positive subjects. Chronic immune activation and persistent systemic inflammation associated with HIV infection may contribute significantly to hepatocyte injury. Continuous viral replication stimulates inflammatory cytokine production, oxidative stress, and mitochondrial dysfunction, which may collectively impair hepatic cellular metabolism and structural integrity. In addition, antiretroviral therapy (ART), although beneficial in suppressing viral replication, has been implicated in drug-induced hepatotoxicity. Certain antiretroviral agents may induce oxidative damage, mitochondrial toxicity, hepatic steatosis, and cholestatic liver injury, thereby contributing to elevated liver enzyme levels observed among infected individuals.

Furthermore, opportunistic infections commonly associated with advanced HIV disease may further aggravate liver dysfunction. Coexisting infections such as tuberculosis, fungal infections, and viral hepatitis may increase inflammatory burden and worsen hepatic injury. Nutritional deficiencies, altered lipid metabolism, and immune dysregulation in HIV patients may also contribute to progressive liver impairment. Therefore, the elevated liver enzymes observed in this study may reflect a multifactorial process involving direct viral effects, immune-mediated injury, medication-associated toxicity, and secondary infectious complications.

The significantly elevated ALP and GGT activities observed among HIV-positive subjects may indicate cholestatic injury and biliary tract involvement. GGT elevation is particularly associated with hepatobiliary dysfunction and oxidative stress. Increased ALP and GGT activities may therefore suggest impaired bile flow, hepatic inflammation, or possible ART-induced cholestasis. Previous studies have shown that persistent elevation of cholestatic enzymes among HIV patients may predispose affected individuals to chronic liver disease and hepatic fibrosis if not adequately monitored and managed.

The present study also demonstrated significantly elevated total bilirubin and conjugated bilirubin concentrations among HIV-positive subjects. Bilirubin is an important indicator of hepatic excretory function, and elevated serum bilirubin levels may suggest impaired conjugation, reduced hepatic uptake, or obstruction of biliary excretion pathways. Increased bilirubin levels may also reflect increased hepatocyte destruction resulting from inflammatory injury. Hyperbilirubinemia among HIV-positive individuals has been associated with hepatic inflammation, antiretroviral drug toxicity, and hemolytic processes. Persistent elevation of bilirubin may predispose patients to jaundice and progressive hepatic dysfunction.

In contrast to bilirubin elevation, albumin concentration was significantly reduced among HIV positive subjects compared with controls. Albumin is synthesized exclusively by hepatocytes and serves as a major indicator of hepatic synthetic capacity. Reduced albumin levels may therefore indicate impaired protein synthesis secondary to chronic liver injury. Hypoalbuminemia may also be influenced by chronic inflammation, malnutrition, reduced nutrient absorption, and increased catabolic activity commonly observed among HIV infected patients. Similar findings were reported by Anyanwu et al. (2021) who observed decreased albumin concentrations among HIV-positive individuals. Reduced serum albumin is clinically important because it may contribute to edema formation, reduced drug-binding capacity, impaired immune response, and poor clinical prognosis.

Interestingly, total protein concentration was significantly increased among HIV-positive subjects despite the reduction in albumin concentration. This increase may be attributed to elevated globulin fractions resulting from chronic immune stimulation and persistent antibody production associated with HIV infection. Hyperglobulinemia is common in chronic inflammatory and infectious diseases and may reflect prolonged activation of B lymphocytes and immunoglobulin synthesis.

The demographic analysis revealed that subjects within the age range of 21–30 years demonstrated the highest degree of liver enzyme alterations and protein abnormalities. This finding may be associated with

increased exposure to behavioral, occupational, and environmental risk factors that predispose younger adults to viral transmission. Factors such as unsafe sexual practices, alcohol consumption, substance abuse, poor health-seeking behavior, and increased social mobility may contribute to higher infection rates and greater disease burden within this age category. Similar age-related trends have been reported in previous epidemiological studies involving HIV and Hepatitis B infections.

Among Hepatitis B-positive subjects, significant elevations in AST, ALT, ALP, and GGT activities were also observed when compared with apparently healthy controls. These findings strongly indicate active hepatocellular inflammation and hepatic injury resulting from Hepatitis B virus (HBV) infection. HBV primarily targets hepatocytes where viral replication induces inflammatory reactions, cellular necrosis, membrane damage, and progressive hepatic dysfunction. Leakage of intracellular liver enzymes into circulation therefore reflects the severity of hepatocyte injury. The findings of this study are in agreement with reports by Olawunmi & Olatunji (2006) and Sarhat et al., (2018), who documented elevated liver enzyme activities among HBV-infected patients.

The elevated AST and ALT activities among Hepatitis B-positive subjects may specifically indicate ongoing hepatocellular necrosis and inflammatory activity within the liver tissue. Persistent hepatic inflammation in chronic HBV infection may eventually progress to fibrosis, cirrhosis, portal hypertension, and hepatocellular carcinoma if early intervention is not instituted. Elevated ALP and GGT activities may additionally indicate cholestatic involvement and disruption of bile canaliculi caused by inflammatory injury.

The significant increase in bilirubin concentrations observed among HBV-positive subjects may reflect impaired bilirubin conjugation and reduced hepatic excretory function resulting from hepatocellular damage. As liver injury progresses, bilirubin clearance becomes compromised, leading to accumulation of both unconjugated and conjugated bilirubin in circulation. Clinically, persistent hyperbilirubinemia may manifest as jaundice, dark urine, pruritus, and generalized weakness.

Similarly, the reduced albumin concentration observed among Hepatitis B-positive subjects suggests impairment of hepatic synthetic function. Since albumin synthesis occurs exclusively in hepatocytes, chronic HBV infection may significantly reduce protein synthesis due to progressive destruction of functional liver cells. Hypoalbuminemia in chronic liver disease is associated with poor prognosis and may contribute to edema, ascites, and reduced plasma oncotic pressure.

The elevated total protein concentration observed among HBV-positive subjects may result from increased production of immunoglobulins during chronic viral infection and inflammatory immune responses. Chronic antigenic stimulation associated with HBV infection activates humoral immune mechanisms, leading to increased globulin synthesis and consequently elevated total serum protein levels.

The findings of this study further emphasize the substantial impact of HIV and Hepatitis B infections on liver function and biochemical homeostasis. Hepatic dysfunction associated with these infections may significantly increase morbidity and reduce quality of life if not properly managed. Regular monitoring of liver function parameters among infected individuals is therefore essential for early detection of hepatic complications, evaluation of disease progression, and assessment of therapeutic safety.

Routine biochemical monitoring of AST, ALT, ALP, GGT, bilirubin, albumin, and total protein levels may assist clinicians in identifying early hepatic abnormalities before the development of irreversible liver damage. Early intervention strategies, including prompt antiviral therapy, careful monitoring of antiretroviral drug toxicity, nutritional support, and lifestyle modification, may help reduce the progression of liver disease among affected patients.

Overall, the present study provides important evidence that HIV and Hepatitis B infections are associated with significant hepatic dysfunction. The study therefore underscores the need for comprehensive clinical management, routine liver function assessment, and continuous monitoring of infected patients in order to reduce hepatic complications and improve long-term clinical outcomes.

The present study demonstrated significant alterations in hepatic parameters among HIV-positive and Hepatitis B-positive patients when compared with the control groups. Elevated levels of AST, ALT, ALP, and GGT observed among HIV-positive subjects suggest hepatocellular injury and hepatic dysfunction associated with HIV infection. These liver enzymes are widely recognized biomarkers of liver cell damage and cholestatic injury. The observed increase agrees with the findings of Iroezindu et al., (2013) who reported abnormal liver enzyme activities among HIV-infected individuals. The increase may be attributed to chronic inflammation, immune activation, oxidative stress, and possible hepatotoxic effects of antiretroviral therapy.

Furthermore, total bilirubin and conjugated bilirubin concentrations were significantly elevated among HIV positive patients compared to the control group. Elevated bilirubin levels may indicate impaired hepatic excretory function and increased destruction of hepatocytes. Similarly, the significant increase in total protein concentration observed in HIV-positive subjects may be associated with increased globulin

synthesis secondary to chronic immune stimulation. In contrast, albumin concentration was significantly reduced in HIV-positive patients, suggesting impaired synthetic function of the liver. This finding is consistent with the report of Anyanwu et al., (2020) who demonstrated reduced albumin synthesis among HIV patients

4. Conclusion

The findings of this study suggest that HIV and Hepatitis causes a significant increase in liver enzymes, total protein, total bilirubin and conjugate bilirubin. Our finding also shows that HIV and Hepatitis decrease albumin concentration.

The findings emphasize the need for routine biochemical monitoring to detect early organ dysfunction in retroviral and hepatitis infections, enabling timely intervention and improved clinical outcomes.

Authors Contribution: Conceptualization and Supervision (Lead); Data Curation and Methodology (equal); writing – review and editing (equal); Investigation (equal). L.C.C, Data Curation and Methodology (equal); writing – original draft (lead); formal analysis (lead); writing – review and editing (equal); Conceptualisation (supporting); Investigation (equal). H.O. E, Formal Analysis (supporting); writing – review and editing (equal)., C.C. B, Writing – review and editing (equal). O.M.C Writing – original draft (supporting); Writing – review and editing (equal); Investigation (supporting).A.A.C

Funding: Authors declare that there was no external funding for this research.

Data Availability: The data presented in this study are available on request from the corresponding author.

Conflict of Interest: The authors declare no conflict of interest.

Ethical Approval: Ethical approval for this study was obtained from the University of Port Harcourt Teaching Hospital Ethical Research Committee with Protocol Number: **UPTH/ADM/90/S.11/VOL.XI/17465**.

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