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Original Research Article

Altered Liver and Kidney Function in SARS-CoV-2-Positive Patients in Nigerian Isolation Centers

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ABSTRACT
The COVID-19 pandemic has presented major immunological and nutritional challenges globally, yet Nigeria's contribution to global research accounts for only 0.8% of scientific outputs. This study investigated liver and kidney function status in SARS-CoV-2-positive patients compared to negative controls across two isolation centers in Ogun and Lagos States (Olabisi Onabanjo University Teaching Hospital and Lagos State University Teaching Hospital). A total of 160 participants were recruited: 80 COVID-19-positive patients (46 males, 34 females; mean age 51.53±1.86) and 80 COVID-19-negative controls (28 males, 52 females; mean age 54.21±1.89). Nasopharyngeal swabs and venous blood samples were collected. Spectrophotometry was used to assess total and direct bilirubin, AST, ALT, total protein, albumin, ALP, urea, chloride, and creatinine. Sodium and potassium were determined via flame photometry, and bicarbonate by titration. Biochemical analysis revealed that COVID-19-positive patients had significantly elevated levels ($p < 0.05$) of plasma urea, creatinine, sodium, chloride, bicarbonate, total bilirubin, direct bilirubin, AST, ALT, and ALP compared to controls. Conversely, a significant decrease in plasma total protein and potassium was observed in COVID-19 patients. There were no significant differences ($p > 0.05$) in age or albumin levels between the two groups. In conclusion, SARS-CoV-2 infection induces multi-organ biochemical derangements characterized by acute renal azotemia, electrolyte imbalance, hepatocellular damage, intrahepatic cholestasis, and hypoproteinemia. Comprehensive baseline liver and kidney biomarker screening is crucial for guiding clinical intervention and management.

CITATION
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INTRODUCTION

The 2019-nCoV human infection cases combine the characteristics of the previous six coronaviruses, which can cause mild cases, easily lose vigilance, and can also cause severe cases, with a high mortality rate (Wang et al., 2020). Multiple organ failure is the main cause of death from COVID-19. The latest research, according to Huang et al., reports that the incidence of COVID-19 with organ dysfunction is about 33%, of which acute renal injury is about 3∼7% (Huang et al., 2020; Chen et al., 2020). Impaired renal function can lead to obstruction of the excretion of metabolites and toxins in the body, which will adversely affect the maintenance of the electrolyte and acid-base balance of the human body. In addition, when renal function is severely damaged, uremia will occur, endangering life. A total of 12 patients admitted into the hospital were diagnosed with COVID-19; the patients were grouped into 3 severe cases and 9 common cases. Serum creatinine (Scr) was not abnormally elevated in all of the patients, and blood urea nitrogen (BUN) was abnormally elevated in only 25.0% of the patients. However, compared with the recovery period, the patients' Scr and BUN increased significantly at the peak of disease (p-scr = 0.002 & p-bun < 0.001). By observing the fluctuations in Scr and BUN from admission to recovery, it was found that the peak of Scr and BUN appeared within the first 14 days of the course of the disease. Scr and BUN were generally increased during the course of COVID-19. However, the evidence is limited due to the small sample size and observational nature. A remarkable amount of clinical studies were published in order to elucidate the clinical manifestations of SARS-CoV-2 infection. Apart from asymptomatic or pauci-symptomatic forms, and severe viral pneumonia with respiratory failure, COVID-19 disease prognosis is influenced by indirect effects on body-wide organs via the angiotensin-converting enzyme-2 (ACE-2) receptor, including heart and renal failure, damage to the central nervous system (CNS), and to the gastrointestinal tract (Zaim et al., 2020; Yuki et al., 2020). Specifically, in the liver, the effect of COVID-19 has been evaluated in relatively few studies, which focused on alterations in liver histopathology and biochemistry during the course of infection in patients who succumbed to the virus (Chen et al., 2020; Huang et al., 2020; Tian et al., 2020; Grein et al., 2020; Yang et al., 2020; Cai et al., 2020). Several up-to-date studies have attempted to evaluate parameters related to liver injury in American and Asian cohorts as well as in European cohorts (Efftenberger et al., 2021; Chen et al., 2020; Cai et al., 2020; Fan et al., 2020; Richardson et al., 2020; Bloom et al., 2020), (Piano et al., 2020; Ponziani et al., 2020; Meszaros et al., 2020). Yet liver disease in the context of SARS-CoV-2 infection remains largely unknown, thus minimizing the understanding of abnormal liver function in terms of the overall outcome. In a study by Benede-Ubieto et al it was found that upon admission, ALT, GGT, and ALP were significantly elevated in approximately 25–35% of COVID-19-confirmed cases. While 49.17% of patients presented elevations in AST levels, nearly 56% had increased LDH values (Bende-Ubieto et al ., 2021). A few previous clinical studies based on single or multiple centers have already shown that COVID-19 patients have signs of liver injury (Tian et al., 2020). Concomitant with findings by Benede-Ubieto et al. (2020. Two studies from Wuhan showed that approximately 35–53% of COVID-19 patients had elevated AST (Chen et al., 2020). In a larger American cohort that included 5700 patients, 59% and 39% showed AST and ALT values above ULN, respectively (Richardson et al., 2020). In agreement with the study by Benede-Ubieto et al. (2020, two independent studies from Northern Italy (Piano et al., 2020) and another study in Austria (Fffenberger et al., 2021), demonstrated upregulation of AST in 44% and 42% of admitted patients with SARS-CoV-2, respectively. In contrast, fewer patients (only 16% and 20%) had abnormal AST levels in the studies from Zhejiang Province (Huang et al., 2020) and Rome (Ponziani et al., 2020). A very likely explanation of the mechanisms leading to liver dysfunction during COVID-19 refers not to the direct cytopathic effects of the virus, but rather to the generalized stress due to the multi-organic character of the infection, which is accompanied by immune injury, systemic inflammatory response syndrome (SIRS) and a stormy release of cytokines that can cause liver injury per se (Tian et al., 2020; Coperchini et al., 2020; Jothimani et al., 2020).

MATERIALS AND METHODS

Study duration

This Case-control study was conducted at the COVID-19 Isolation Centre of Lagos State University Teaching Hospital, Ikeja, Lagos, Nigeria, and Ogun State University Teaching Hospital, Sagamu, Ogun State, between January 15th, 2021, and January 14th, 2022.

Ethical Approval

The study protocol was reviewed and approved by the Health Research and Ethics Committee of LASUTH approval number: LREC/06/10/1474), while written informed consent was obtained from participants. A structured questionnaire was used for patients' biodata.

Study Population: Healthy Controls

The Inclusion Criteria: As follows: baseline oxidative stress and antioxidant status; age- and sex-matched healthy controls with no record of dyslipidemia, diabetes, hypertension, or cardiovascular disease at the Lagos State University Teaching Hospital, Ikeja, Lagos. Nigeria was recruited from the local community/hospital staff during the same study period. Individuals receiving antioxidant therapy such as vitamins, zinc, folate, glutathione, folic acid, and selenium were excluded from the study. Control

samples were individuals who tested negative for SARS-CoV-2 via a real-time reverse transcription-polymerase chain reaction (rRT-PCR) nasopharyngeal swab at the time of enrollment.

Exclusion Criteria

Individuals meeting any of the following criteria were excluded from the control group to prevent confounding of oxidative stress markers Chronic Comorbidities: Any established medical history of metabolic disorders (Type 1 or Type 2 Diabetes Mellitus), cardiovascular diseases (Hypertension, Coronary Artery Disease), chronic kidney disease, chronic obstructive pulmonary disease (COPD), or active malignancies. Inflammatory and Autoimmune Conditions: Documented history of autoimmune disorders or chronic inflammatory conditions. Medication and Supplementation: Current use of systemically active medications or regular intake of antioxidant supplements (e.g., Vitamin C, Vitamin E, Zinc, Selenium) 2 weeks or 1 month before blood sampling.

Lifestyle Factors: Heavy smoking or significant alcohol consumption, independently associated with elevated lipid peroxidation.

Verification of "Healthy" State

The controls were free of hidden metabolic or physiological confounders; all potential candidates underwent a standardized clinical screening protocol before blood collection.

Medical History and Physical Examination: A trained healthcare professional administered a detailed questionnaire and clinical examination to verify the medical history, calculate Body Mass Index (BMI), and measure resting blood pressure. Normal blood pressure was defined as systolic blood pressure <120 mm Hg and diastolic blood pressure <80 mm Hg.

Biochemical Screening: Fasting blood glucose (FBG) testing was performed to rule out undiagnosed diabetes or prediabetes. Participants were only confirmed as "healthy" if their FBG was within the normal reference range <100 mg/dL or <5.6 mmol/L.

Routine laboratory parameters, including routine complete blood counts (CBC), liver function tests (ALT/AST), and renal function tests (creatinine), were found to be within normal physiological limits.

Sample Size

One hundred and sixty (160) subjects were enrolled in the study. This was derived from the Nigerian COVID-19 prevalence of 6% reported by the Africa Centre for Disease Control [5] (Africa CDC, 2020).

Administration of Questionnaire

Structured questionnaires were administered to obtain information on demographic data.

The research team administered the questionnaire. The questionnaire is divided into sections A, B, C, D, and E and adapted from a previous study by Obinna et al. (2020)[6]. Section A covers demographic questions such as age and gender. and baseline information. Section B includes symptoms and exposure history. Section C includes medical and vaccination History, while Section D includes consent and terms of Consent (confirmation of having read and agreed to the terms of consent). Section E includes laboratory/biochemical parameters that were filled by the research team and healthcare workers. The questionnaire was designed and used to collect demographic, clinical, and exposure data from COVID-19 patients and healthy individuals (controls). Also used to assess recent symptoms, risk factors, and pre-existing medical conditions.

Sample Collection

A nasopharyngeal swab was collected with a sterile flocked swab stick into viral transport media (VTM). Five millilitres (5ml) of venous blood was collected into a plain bottle via venepuncture from the antecubital fossa under aseptic conditions from all subjects from the isolation centre and sent to the molecular laboratory. The samples were taken after the blood pressure measurement, with the participants still relaxed. The samples were used to determine the serum activity of catalase, superoxide dismutase, and reduced glutathione, malonylaldehyde levels, and total antioxidant capacity were measured using Enzyme-linked immunosorbent assay (ELISA) in all the participants.

Sample Preparation and Laboratory Analysis

RNA was extracted from the nasopharyngeal swab, while COVID-19 detection was performed using the real-time Polymerase Chain Reaction (PCR) technique. The presence of SARS-CoV-2 in the nasopharyngeal swab was identified through real-time RT-PCR amplification with the GeneFinder COVID-19 PLUS RealAmp Kit (OSANG Healthcare Co., Ltd., Korea) on the ELITE InGenius platform (ELITech Group, Puteaux, France), which integrates extraction and amplification. The real-time PCR kit detects three SARS-CoV-2 targets simultaneously: envelope protein (E), nucleocapsid protein (N), and RNA-dependent RNA polymerase (RdRp) genes. An internal control, based on the amplification of the human beta-globin gene, confirms the quality of the extracted sample material and the test performance.

Biochemical Analysis

Biochemical parameters such as plasma total and direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total protein, albumin, alkaline phosphatase (ALP), urea, chloride, and creatinine.

Electrolytes such as sodium, potassium, bicarbonate, and chloride.

Data Analysis

The data were analysed using Statistical Package for the Social Sciences (SPSS) version 23. Mean variables were compared across different groups using Student t-test and Analysis of variance (ANOVA). Differences were shown to be statistically significant where $p<0.05$.

RESULTS AND DISCUSSION

Table 1: COVID-19-infected subjects exhibited significant alterations in renal function parameters compared to healthy controls. Urea and creatinine levels were both significantly higher in COVID-19 patients, indicating impaired renal function (urea: 26.04±3.47 vs 19.48±1.50 mg/dl, p=0.031; creatinine: 1.14±0.08 vs 0.90±0.016 mg/dl, p=0.006). Potassium was slightly but significantly lower in COVID-19 cases (4.07±0.06 vs 4.24±0.051 mEq/l, p=0.040), while sodium, chloride, and bicarbonate were all significantly elevated in the COVID-19 group (Na: 139.23±0.04 vs 137.44±0.33 mEq/l, p=0.001; Cl: 107.72±0.87 vs 104.73±0.64 mEq/l, p<0.001; HCO3: 26.31±0.47 vs 17.88±0.37 mEq/l, p<0.001). All differences reported are statistically significant, denoted by p-values less than 0.05.

Table 1: Renal Function Parameters in Covid-19 Subjects and Healthy Controls

Parameters	COVID 19 N=80 (Mean±Sem)	Control N=80 (Mean±Sem)	p-Value
UREA (mg/dl)	26.04±3.47	19.48±1.50	0.031*
CRT(mg/dl)	1.14±0.08	0.90±0.016	0.006*
K (mEq/l)	4.07±0.06	4.24±0.051	0.040*
Na (mEq/l)	139.23±0.04	137.44±0.33	0.001*
Cl (mEq/l)	107.72±0.87	104.73±0.64	0.000*
Hco3 (mEq/l)	26.31±0.47	17.88±0.37	0.000

Values are expressed as Mean ± standard error of mean (SEM), and statistically significant at $p < 0.05$.

Table 2: COVID-19 patients demonstrated significant alterations in liver function parameters compared to healthy controls. Total and direct bilirubin levels were both significantly increased in COVID-19 cases (T.BIL: 0.08±0.02 vs 0.02±0.0015 mg/dL, p=0.007; D.BIL: 0.0235±0.0008 vs 0.0097±0.004 mg/dL, p=0.007), indicating greater bilirubin accumulation. Serum transaminases were also elevated: AST was substantially higher in COVID-19 subjects (26.50±1.77 vs 14.67±0.669 U/L, p<0.001) while ALT was modestly but significantly elevated (25.45±1.19 vs 21.52±1.15 U/L, p=0.011). These findings suggest that hepatic injury or dysfunction is more common in COVID-19 patients, as evidenced by increased bilirubin and transaminase levels, with all differences reaching statistical significance ($p < 0.05$).

Table 2: Liver function parameters in COVID-19 patients and controls

Parameters	Case N = 80	Control N = 80	P value
T.BIL (mg/dL)	0.08±0.02	0.02±0.0015	0.007*
D.BIL (mg/dL)	0.0235±0.0008	0.0097±0.004	0.007*
AST (U/L)	26.50±1.77	14.67±0.669	0.000**
ALT (U/L)	25.45±1.19	21.52±1.15	0.011*
TP(g/dl)	7.11±0.10	7.81±0.14	0.000**
ALB (g/dl)	3.04±0.10	4.81±0.76	0.050

Values are expressed as Mean ± standard error of the mean (SEM), and statistically significant at $p < 0.05$.

Table 3: Analysis of liver function profiles in COVID-19 patients across four age groups (15-34, 35-54, 55-64, 65-94 years) revealed the following: Total bilirubin (T.BIL), direct bilirubin (D.BIL), AST, and ALT values showed moderate variation across age groups, but these differences were not statistically significant ($p > 0.05$). ALP (alkaline phosphatase) exhibited significant differences ($F = 2.896$, $p = 0.041$), being highest in the 35-54 year age group (126.53±6.86 U/L) and lowest in the oldest group (93.91±8.36 U/L). The 55-64 year group showed the highest mean AST and T.BIL, indicating greater liver cell injury/enzyme release in this subgroup, but these differences did not achieve significance. In summary, among liver function biomarkers in COVID-19 patients, only ALP displayed a statistically significant age-related difference, suggesting age-specific changes related to cholestatic or hepatobiliary involvement, whereas other markers, such as bilirubin and transaminases, were relatively consistent across age groups.

Table 3: Age Grouping Distribution and Liver Function Profile in COVID-19 Patients

Parameters	15-34 Years N = 12	35-54 Years N = 34	55-64 Years N = 16	65-94 Years N = 18	p
T.BIL (mg/dL)	0.08±0.05	0.055±0.02	0.15±0.07	0.059±0.03	0.355
D.BIL (mg/dL)	0.02±0.01	0.019±0.006	0.037±0.016	0.019±0.008	0.603
AST (U/L)	23.08±4.96	24.91±2.27	34.93±4.89	24.27±3.42	0.121
ALT (U/L)	26.08±3.31	24.82±1.73	26.37±2.84	26.88±2.65	0.916
ALP (U/L)	117.24±14.41	126.53±6.86	103.75±9.98	93.91±8.36	0.041*

Values are expressed as Mean ± standard error of mean (SEM), and statistically significant at $p < 0.05$

Table 4: Kidney function profiles among coronavirus victims, analyzed across four different age groups (15-34, 35-54, 55-64, and 65-94 years), showed significant variations in some parameters: Urea levels varied significantly with age (F=4.406, p=0.007), showing a marked increase in the 55-64 years group (52.32±14.52 mg/dL), indicating more pronounced renal impairment in this middle-aged group. Creatinine levels did not show significant differences across age groups (F=1.403, p=0.248), though the 55-64 years group had a higher mean value (1.46±0.38 mg/dL). Potassium (K+) and sodium (Na+) levels also did not significantly differ by age, but potassium showed a non-significant trend toward elevation in the 55-64 years group. Chloride (Cl-) showed a significant difference between age groups (F=3.719, p=0.015), with the highest means in the 15-34 and 55-64 years groups. Bicarbonate (HCO3-) did not differ significantly across age groups (F=2.135, p=0.103). These results indicate that kidney function markers such as urea and chloride significantly change with age in COVID-19 patients, particularly with worse renal function in middle-aged adults, while other electrolytes and creatinine remain relatively stable across ages. This suggests age-related susceptibility to renal impairment in COVID-19 can be reflected in these markers.

Table 4: Kidney Function Profile in Coronavirus Victims Across Different Age Groups

Parameters	15-34 Years N = 12\	35-54 Years N = 34	55-64 Years N = 16	65-94 Years N = 18	P
UREA (mg/dL)	23.44±5.47	20.94±1.75	52.32±14.52	27.32±4.81	0.007*
CRT(mg/dL)	0.99±0.09	1.02±1.06	1.46±0.38	1.12±0.08	0.248
K^+ (mEq/L)	4.02±0.19	4.01±0.06	4.38±0.21	3.93±0.05	0.082
Na^+ (mEq/l)	137.16±1.28	140.26±0.27	138.87±1.13	139.00±1.04	0.072
Cl^- (mEq/l)	111.00±1.67	105.02±1.30	111.43±1.64	107.27±1.93	0.015*
HCO_3 (mEq/l)	24.16±1.56	27.52±0.33	25.75±1.36	25.94±1.21	0.103

Values are expressed as Mean ± standard error of mean (SEM), and statistically significant at $p < 0.05$.

Table 5: The correlation analysis of liver function parameters in COVID-19 patients showed strong and statistically significant relationships among various pairs: Total bilirubin (T.BIL) and direct bilirubin (D.BIL) exhibited a very strong positive correlation ($r = 0.902$, $p < 0.001$). T.BIL correlated moderately with ALT ($r = 0.310$, $p = 0.005$), and similarly, ALT correlated moderately with T.BIL ($r = 0.529$, $p < 0.001$). Bilirubin and AST levels were also moderately positively correlated ($r = 0.510$, $p < 0.001$). AST and D.BIL showed a significant moderate correlation as well ($r = 0.510$, $p < 0.001$). ALT and D.BIL had a weaker but statistically significant correlation ($r = 0.248$, $p = 0.026$). These findings suggest that liver injury markers related to bilirubin metabolism and hepatocellular enzymes ALT and AST are interrelated in COVID-19 patients, reflecting a linked hepatic response to viral infection or damage. The very strong correlation between total and direct bilirubin underscores the interplay in bilirubin processing affected by COVID-19. Moderate correlations of bilirubin with transaminases indicate concurrent hepatocellular injury and cholestasis or bile excretion disruptions.

Table 5: Correlation Pair for COVID-19 Patients

Parameters	R	P
T.BIL/D.BIL	0.902	0.000**
T.BIL/ALT	0.310	0.005**
BIL/AST	0.510	0.000**
ALT/T.BIL	0.529	0.000**
AST/D.BIL	0.510	0.000**
ALT/D.BIL	0.248	0.026*

** Correlation is significant at the 0.001 level (2-tailed). * Correlation is significant at the 0.05 level, two-tailed.

Table 6: The correlation analysis of kidney function parameters and other biochemical markers in COVID-19 patients revealed several significant relationships: Urea showed a moderate negative correlation with total protein ($r = -0.372$, $p = 0.001$) and cholesterol ($r = -0.240$, $p = 0.032$), implying that as urea increases, these parameters tend to decrease. Urea and creatinine exhibited a strong positive correlation ($r = 0.666$, $p < 0.001$), reflecting the related renal excretory function. Sodium (Na+) and chloride (Cl-) were strongly positively correlated ($r = 0.584$, $p < 0.001$). Sodium also showed an even stronger positive correlation with bicarbonate (HCO3-) ($r = 0.899$, $p < 0.001$). Chloride and bicarbonate were negatively correlated ($r = -0.497$, $p < 0.001$), indicating interplay in electrolyte and acid-base balance. These data reflect significant interactions between kidney function markers, electrolytes, and inflammatory/metabolic biomarkers in COVID-19 patients, emphasizing the complexity of systemic involvement and the potential for multi-parameter monitoring in disease management

Table 6: Correlation Pair for Covid-19 Patients

Parameter	R	P
UREA/TP	-0.372	0.001**
UREA/CREAT	0.666	0.000**
NA/CL	0.584	0.000**
NA/HCO3	0.899	0.000**
CL/HCO3	-0.497	0.000**

**Correlation is significant at the 0.001 level-2, tailed,

* Correlation is significant at the 0.05 level, two-tailed.

Discussion

This study set out to investigate the liver and kidney function status of individuals affected by COVID-19 (SARS-CoV-2 patients) and those with negative COVID-19 results in two isolation facilities in Nigeria.

The findings in this study demonstrate significant renal impairment and electrolyte alteration in COVID-19 patients compared to healthy controls, characterized by hyperuremia, hypercreatinemia, mild hypernatremia, and hypokalemia. Comparing these findings with studies conducted in Nigeria, across Africa, and globally provides valuable pathophysiological and clinical context. Urea and Creatinine (Renal Waste Markers): Both Urea (26.04 ± 3.4 mg/dL vs. 19.48 ± 1.50 mg/dl, $p = 0.031$) and Creatinine (1.14 ± 0.08 mg/dL vs. 0.90 ± 0.016 mg/dL, $p = 0.006$) were significantly elevated in COVID-19 patients. Studies across West and Sub-Saharan Africa similarly report elevated blood urea nitrogen (BUN) and serum creatinine in hospitalized COVID-19 cohorts. This elevation is widely attributed to acute kidney injury (AKI), prerenal azotemia caused by dehydration/fever, and systemic viral inflammation. Observational studies globally confirm that SARS-CoV-2 infection frequently induces transient or progressive renal dysfunction (Hong et al., 2020). Higher serum creatinine and BUN levels correlate directly with disease severity and hospital mortality rates. Serum potassium was significantly lower in COVID-19 patients (4.07 ± 0.06 mEq/L) compared to controls (4.24 ± 0.051 mEq/L, $p = 0.040$). Hypokalemia is a widely documented feature of COVID-19 globally and regionally. SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2) receptors—highly expressed in renal tubular epithelial cells—leading to downregulation of ACE2, increased angiotensin II levels, and enhanced aldosterone-driven urinary potassium excretion. Serum sodium was significantly higher in COVID-19 subjects (139.23 ± 0.04 mEq/L) than in controls (137.44 ± 0.33 mEq/L, $p = 0.001$). While the cohort exhibited a mild increase in sodium (remaining within the normal physiological range of 135-145 mEq/L), many global and African studies report hyponatremia as the predominant electrolyte abnormality, often secondary to the Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) triggered by IL-6 cytokine surges. However, mild hypernatremia can occur due to insensible fluid loss (fever, tachypnea) or impaired fluid intake during acute viral illness.

The experimental results in Table 2 demonstrate significant hepatic enzyme elevation and altered synthetic protein function in SARS-CoV-2-infected subjects (N=80) compared to healthy controls (N=80). Infected patients exhibited statistically significant increases in total bilirubin ($p=0.007$), direct bilirubin ($p=0.007$), aspartate aminotransferase (AST, $p=0.000$), and alanine aminotransferase (ALT, $p=0.011$). Conversely, total protein was significantly depleted ($p=0.000$), accompanied by a reduction in serum albumin ($p=0.050$).

In this study, AST (26.50±1.77 U/L vs. 14.67±0.669 U/L, $p=0.000$) and ALT (25.45±1.19 U/L vs. 21.52±1.15 U/L, $p=0.011$) were markedly elevated in infected patients, with AST showing a more pronounced increase than ALT. These results closely parallel findings across African cohorts (Mekuanint et al., 2024). In Ethiopian and West African clinical studies, elevated AST and ALT are among the most predominant biochemical markers of SARS-CoV-2 infection. Systematic reviews and meta-analyses globally confirm that transaminase elevation occurs in 14%–53% of hospitalized COVID-19 patients (Ahmed et al., 2020; Ali et al., 2023). The disproportionate elevation of AST relative

to ALT is a recognized hallmark of COVID-19-induced hepatic injury. It stems from a combination of direct viral entry via ACE2 receptors expressed on cholangiocytes and hepatocytes, systemic hyper-inflammation (cytokine storm driven by IL-6 and TNF-α), microvascular thrombosis, and hepatic ischemia secondary to respiratory hypoxia.

This study reveals that both Total Bilirubin (0.08±0.02 mg/dL vs. 0.02±0.0015 mg/dL, p=0.007) and Direct Bilirubin (0.0235±0.0008 mg/dL vs. 0.0097±0.004 mg/dL, p=0.007) were significantly elevated in the COVID-19 group. Increased total and direct bilirubin concentrations are consistent with regional African data (Mekuanint et al., 2024) and global clinical studies (Ahmed et al., 2020; Macit, 2021). Bilirubin elevation indicates cholestatic or hepatocellular damage. Because cholangiocytes (bile duct epithelial cells) express higher density of ACE2 receptors than parenchymal hepatocytes, SARS-CoV-2 infection causes biliary epithelial injury, leading to impaired bile secretion and conjugated (direct) hyperbilirubinemia. Serum Total Protein was significantly lower in COVID-19 subjects (7.11±0.10 g/dL) than in controls (7.81±0.14 g/dL, p=0.000). Albumin levels were also reduced (3.04±0.10 g/dL vs. 4.81±0.76 g/dL, p=0.050). Hypoalbuminemia and hypoproteinemia are widely documented across Nigerian isolation centers, broader Sub-Saharan African cohorts, and international clinical series (Ahmed et al., 2020; Mekuanint et al., 2024). Albumin acts as a negative acute-phase reactant during systemic inflammation. Cytokine activation suppresses hepatic albumin gene expression and increases vascular permeability, causing albumin extravasation into interstitial spaces. In sub-Saharan African populations, pre-existing nutritional variations or acute catabolic stress further accelerate protein depletion.

The data in Table 3 evaluates liver function profile parameters across four distinct age demographics (15–34, 35–54, 55–64, and 65–94 years) within the COVID-19-positive patient cohort (N=80). Statistically, Total Bilirubin (p=0.355), Direct Bilirubin (p=0.603), AST (p=0.121), and ALT (p=0.916) showed no significant variance across age groups. However, Alkaline Phosphatase (ALP) demonstrated a statistically significant difference (p=0.041∗), displaying higher mean levels in middle-aged subjects (35–54 years: 126.53±6.86 U/L) and younger adults (15–34 years: 117.24±14.41 U/L) compared to older cohorts (65–94 years: 93.91±8.36 U/L). In this study, AST and ALT were elevated across all infected age groups compared to standard reference limits, but showed no statistically significant differences *between* the age brackets (p>0.05). Bilirubin fractions similarly showed age-independent distribution. Broad epidemiological studies globally and across Africa often report that advanced age (≥60 years) correlates with higher degrees of hepatocellular injury, marked by significantly higher AST and ALT levels due to immunosenescence, higher prevalence of comorbidities, and heightened cytokine release (Mekuanint et al., 2024; Yip et al., 2021).

The absence of a statistically significant difference in transaminases across age groups in this Nigerian cohort indicates that SARS-CoV-2-induced hepatocellular damage occurs independently of patient age. Direct viral cell entry via ACE2 receptors and systemic hyper-inflammation induce comparable baseline hepatic stress across both younger and older adult populations in isolation facilities. ALP showed a significant age-dependent variance (p=0.041∗), peaking in the 35–54 age group (126.53±6.86 U/L) and declining in the elderly bracket (65–94 years: 93.91±8.36 U/L). ALP is a marker of biliary epithelium integrity and osteoblast activity. While acute COVID-19 cholangiocyte infection elevates ALP globally due to direct viral damage to bile duct epithelial cells (Ali et al., 2023), physiological baseline ALP naturally varies by age. The higher ALP levels observed in younger and middle-aged adults compared to older patients likely reflect stronger cellular inflammatory responses in active age groups, along with potential bone turnover differences. In elderly cohorts, lower baseline bone-specific ALP combined with reduced metabolic reserve can alter total serum ALP dynamics despite acute viral stress.

The experimental data in Table 4 demonstrate the age-stratified renal function profile and electrolyte parameters among COVID-19-positive patients (N=80). Statistically significant age-dependent variations were observed in plasma Urea (p = 0.007*) and serum Chloride (p = 0.015*). Specifically, urea peaked in the 55–64 age group (52.32 ± 14.52 mg/dL), while chloride was highest in both the 55–64 age group (111.43 ± 1.64 mEq/L) and the 15–34 age group (111.00 ± 1.67 mEq/L). Conversely, Serum Creatinine (p = 0.248), Potassium (p = 0.082), Sodium (p = 0.072), and Bicarbonate (p = 0.103) showed no statistically significant differences across age brackets, indicating a relatively uniform distribution of these derangements among age groups within the infected cohort. Serum urea demonstrated a marked, statistically significant elevation in the 55–64 age group (52.32 ± 14.52 mg/dL, p = 0.007*). Although serum creatinine peaked in the same 55–64 cohort (1.46 ± 0.38 mg/dL), its variation across age groups did not achieve statistical significance (p = 0.248).

Studies in Nigerian tertiary care centers and sub-Saharan African cohorts consistently identify older adults (typically age 50 years) as having higher susceptibility to acute kidney injury (AKI) and uremic waste retention (Egbi, 2020; Sabaghian et al., 2022). Meta-analyses globally highlight that middle-aged to older COVID-19 patients exhibit a steeper rise in blood urea nitrogen (BUN) due to a combination of pre-existing subclinical renal microvascular disease, heightened inflammatory cytokine release, and age-associated decline in physiological

functional nephron mass. The disproportionate elevation of urea over creatinine in the 55–64 age group strongly reflects prerenal azotemia, driven by systemic fever, hypercatabolism, and fluid depletion during viral isolation. Serum Chloride showed significant age-related variance (p = 0.015*), remaining highest in the youngest (15–34 years: 111.00 ± 1.67 mEq/L) and middle-aged groups (55–64 years: 111.43 ± 1.64 mEq/L). In contrast, Sodium, Potassium, and Bicarbonate variations were non-significant across age groups (p > 0.05). Electrolyte derangements are widely documented across all age demographics in COVID-19 patients (Cancarevic et al., 2024; Taci Hoca & Berktaş, 2022).

The age-independent nature of hypokalemia, hypernatremia, and elevated bicarbonate in your cohort suggests that core pathophysiological drivers—such as SARS-CoV-2-induced downregulation of renal ACE2 receptors, Renin-Angiotensin-Aldosterone System (RAAS) overactivation, and gastrointestinal losses—exert systemic effects across young, middle-aged, and elderly adults alike. The significant variance in chloride levels reflects age-specific differences in tubular reabsorption kinetics, metabolic acid-base compensation, and fluid loss rates during acute phase illness.

The Pearson correlation analysis in Table 5 demonstrates strong, statistically significant positive linear relationships among hepatic biomarkers in SARS-CoV-2 patients (N=80). Most notably, Total Bilirubin and Direct Bilirubin exhibited an exceptionally strong positive correlation (r=0.902, p=0.000). Statistically significant moderate positive correlations were observed between Bilirubin (total/direct) and AST (r=0.510, p=0.000), ALT and Total Bilirubin (r=0.529, p=0.000), and Total Bilirubin and ALT (r=0.310, p=0.005). A weaker yet significant positive correlation was noted between ALT and Direct Bilirubin (r=0.248, p=0.026∗). These linear dependencies indicate synchronized hepatobiliary injury during acute COVID-19 infection. This study shows near-perfect linear alignment between Total Bilirubin and Direct Bilirubin (r=0.902, p=0.000). This parallel rise in both conjugated and total bilirubin fractions strongly aligns with reports across African isolation centers and global hepatology studies (Macit, 2021; Mekuanint et al., 2024). Because cholangiocytes in the bile duct express higher densities of ACE2 receptors than parenchymal hepatocytes, viral entry triggers severe biliary microvascular inflammation and ductular cholestasis. The tight coupling (r=0.902) shows that direct hyperbilirubinemia drives overall total serum bilirubin retention, serving as a direct marker of viral-induced biliary tree dysfunction. Consistent, moderate-to-strong positive associations exist between transaminases (AST, ALT) and bilirubin fractions (r=0.510 for AST/D.BIL; r=0.529 for ALT/T.BIL; r=0.310 for T.BIL/ALT; r=0.248 for ALT/D.BIL). Studies evaluating biochemical markers in hospitalized Nigerian patients similarly report strong co-elevation of AST, ALT, and bilirubin during acute inflammatory stages (Egbi, 2020).

Meta-analyses globally highlight that liver injury in COVID-19 is rarely isolated to a single cell line; rather, parenchymal necrosis (yielding elevated AST/ALT) and intrahepatic cholestasis (yielding elevated bilirubin) occur concurrently (Ahmed et al., 2020; Ali et al., 2023). Systemic cytokine surges (IL-6, TNF-α), hepatic hypoxia, and drug-induced hepatotoxicity synchronously escalate both transaminases and bilirubin levels.

The Pearson correlation matrix in Table 6 highlights major physiological interplay among renal nitrogenous waste products, serum proteins, and key electrolytes in SARS-CoV-2-infected patients (N=80). Statistically significant positive correlations were observed between Sodium and Bicarbonate (r = 0.899, p = 0.000), Urea and Creatinine (r = 0.666, p = 0.000), and Sodium and Chloride (r = 0.584, p = 0.000). Conversely, significant inverse (negative) linear relationships were demonstrated between Chloride and Bicarbonate (r = -0.497, p = 0.000) and Urea and Total Protein (r = -0.372, p = 0.001).

A strong positive correlation between serum urea and creatinine is a classic hallmark of progressive nephron dysfunction and prerenal strain. This strong alignment is widely documented in Nigerian and broader Sub-Saharan African cohorts (Egbi, 2020) as well as global renal clinical studies (Sabaghian et al., 2022). Viral tropism via ACE2 receptors on renal tubular epithelium combined with systemic inflammatory microvascular damage causes synchronous reductions in glomerular filtration rate (GFR), elevating both creatinine and urea simultaneously. The significant negative correlation indicates that as nitrogenous waste (urea) accumulates, serum protein stores are depleted. Systemic hyper-inflammation (cytokine storm) accelerates hypercatabolism and muscle wasting, breaking down tissue proteins to produce excess blood urea nitrogen. Concurrently, hepatic protein synthesis declines (negative acute-phase reaction) while capillary permeability increases, driving down total serum protein. Strong positive correlations among sodium, chloride, and bicarbonate reflect coupled renal tubular reabsorption kinetics. In response to hypertonic dehydration, fever, and volume depletion (common in Nigerian isolation wards), the renin-angiotensin-aldosterone system (RAAS) is activated, stimulating proximal and distal renal tubule retention of sodium along with its companion anions (bicarbonate and chloride) to preserve vascular volume. The moderate inverse relationship between chloride and bicarbonate (r = -0.497) demonstrates renal electrical neutrality maintenance (anion exchange mechanism. This inverse trend is consistent with international findings on acid-base homeostasis in acute viral infections (Cancarevic et al., 2024; Taci Hoca & Berktaş, 2022). As renal tubular cells excrete or retain bicarbonate to compensate for

respiratory alkalosis/acidosis, chloride ions shift in the opposite direction via the chloride and bicarbonate exchanger to maintain extracellular fluid electroneutrality. Elevated liver and kidney profile results in COVID-19 patients indicate significant organ involvement and dysfunction associated with the severity of the infection.

CONCLUSION

This study highlights the importance of regular monitoring of hepatic and renal biomarkers to identify early organ impairment, guide clinical management, and improve patient outcomes. Understanding the patterns and extent of liver and kidney damage in COVID-19 can help tailor therapeutic interventions and mitigate complications, ultimately reducing morbidity and mortality in affected individuals. Further research is warranted to explore the underlying mechanisms and long-term impact of COVID-19 on these vital organs.

Ethics approval and consent to participate

The authors clearly state that the work was conducted in accordance with national and international ethical standards, that permission was obtained from a recognized ethics committee, and that informed consent was obtained from participants (or a waiver justified where applicable).

Consent for publication

The Health Research Ethics Committee waived the requirement for individual consent for publication, as the data were fully anonymized and reported in aggregate form, with no identifiable information included in the manuscript.

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