Original Article

# Derangements of Liver Enzymes in a Study of 201 COVID-19 Patients in Abuja, Nigeria's Federal Capital Territory

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## Abstract

**Background:** Several studies reveal abnormalities in liver function tests of COVID-19 patients. However, there are little data on African patients. **Aim:** This study aimed to evaluate liver function tests (LFT) in severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infected patients admitted in Asokoro COVID-19 Isolation and Treatment Centre in Abuja, Nigeria. **Patients, Materials and Methods:** This was a retrospective study of 201 laboratory-confirmed SARS-CoV-2-infected patients hospitalized in Asokoro District Hospital COVID-19 Isolation and Treatment Centre between April 10 and July 31, 2020. Demographic, clinical, and laboratory data were obtained, and the outcome measure was LFT abnormalities at presentation. Statistical analysis was done using IBM SPSS Version 24, with $P < 0.05$ considered statistically significant. **Results:** Patient median age was 39.3 years (IQR: 26–52); 65.7% were males and 33.8% were health workers. Approximately 49.2% of patients were overweight or obese. Hypertension (22.9%) and diabetes mellitus (7.5%) were the most common comorbidities and only 1% had a known history of liver disease. Abnormal LFTs were observed in 53% of patients ($n = 106$), most frequently elevated direct bilirubin (78.3%) and alanine aminotransferase (38.7%). Comorbidities were not found significantly associated with LFT abnormalities. Females (odds ratio [OR] = 0.367 $P = 0.004$ confidence interval [CI] 0.186–0.724) and patients aged 20–29 years (OR = 0.067 $P = 0.043$ [CI] 0.005–0.916) were found less likely to have abnormal LFTs. **Conclusion:** Regardless of clinical status at presentation, about half of SARS-CoV-2 patients admitted at the Asokoro Isolation and Treatment Centre in Abuja had abnormal LFT results. It is therefore recommended that LFT is included as a part of baseline investigations during the management of COVID-19 for improved outcomes.

**Keywords:** COVID-19, laboratories, liver diseases, liver function tests, severe acute respiratory syndrome coronavirus 2

## Introduction

The ongoing coronavirus disease 2019 (COVID-19) pandemic caused by the new severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was first reported in Hubei province, Wuhan, China, in December 2019.[1,2] As of June 2021, there have been over 180,000,000 confirmed cases spanning over 213 countries, 3,900,000 deaths, and more than 700,000,000 people fully vaccinated globally.[1] Nigeria's first case was identified on the February 27, 2020, and by June 2021 had increased to over 167,375 confirmed cases, 163,917 discharged, and 2118 deaths from the virus.[3]

At the time of writing, Nigeria's epicenter of the outbreak was Lagos State (35.4% of confirmed cases), with Abuja the Federal Capital Territory (FCT) recording the second highest number of infections, accounting for 12.1% of the country's confirmed cases.[3]

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Infection is commonly acquired through inhalation of infected droplets released into the air when an infected individual coughs, sneezes, or speaks. However, transmission can also occur when an individual encounters an infected surface and introduces the organism into the eyes, nose, or mouth. The organism then usually settles in the respiratory tract where it binds to angiotensin-converting enzyme-2 (ACE-2) receptors, leading to infection.[4] The most common presentations of COVID-19 include fever and cough, with approximately 14%

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of patients presenting with severe disease and 5% requiring admission into intensive care following substantial alveolar damage and respiratory failure. Gastrointestinal symptoms such as diarrhea, nausea, and vomiting reportedly present in 2%–10% of patients.[5,6]

Knowledge of the virus and the clinical and pathological patterns of the disease are also rapidly evolving, and this virus has also been linked to gastrointestinal system and liver complications.[7-9] Reported abnormalities in liver function tests (LFTs) of COVID-19 patients include elevated levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and bilirubin in up to one-third of patients.[10-12] Post-admission, LFT abnormalities may be related to lopinavir/ritonavir-based treatment regimens and prolonged hospital stay. In addition, ACE-2 receptors are found in hepatocytes and cholangiocytes, and the binding of SARS-CoV-2 virus to these receptors may cause cholangiocyte dysfunction, inducing a systemic inflammatory response and cytokine release leading to liver damage.[13] Other possible causes of coronavirus hepatic involvement are direct cytopathic injury and ischemia to the liver due to hypoxia.[8,14,15]

The link between liver impairment and coronaviruses is not a new phenomenon and has been reported previously with both SARS and MERS-CoV, with occurrence as high as 60% in SARS-CoV infection.[14,16,17] Studies have documented these findings in COVID-19 patients in China, Italy, and the United States.[8,15,18] However, not much data are available on hepatological enzyme findings in African patients.

The aim of this study was therefore to describe the sociodemographic and clinical characteristics of a cohort of 201 laboratory (polymerase chain reaction [PCR]) positive COVID-19 patients admitted to one of six COVID-19 isolation and treatment centers in Abuja, Nigeria's FCT with specific regard to alteration in LFTs.

## Patients, Materials and Methods

### Study design

A retrospective study of 201 laboratory-confirmed SARS-Cov-2-infected patients admitted to the Asokoro COVID-19 Isolation and Treatment Centre in Asokoro District Hospital between April 10 and July 31, 2020. This study is included in the Asokoro District Hospital COVID-19 Clinicopathological Profile Project, which investigates the clinical, laboratory, radiologic, and pathologic presentation of COVID-19 patients managed at the hospital in Abuja, FCT, Nigeria. Ethical approval was obtained from the FCT Health Research Ethics Committee.

### Study setting

The study was conducted on SARS-CoV-2-infected patients treated at the COVID-19 Isolation and Treatment center in Asokoro District Hospital, Abuja FCT, North-Central Nigeria. According to the 2018 National Demographic and Health Survey, Abuja's population is over 3.5 million.[19,20] The Asokoro District Hospital was designated to isolate and treat COVID-19 patients, following the University of Abuja Teaching Hospital, Gwagwalada, as a foremost public infectious disease hospital in Nigeria's capital with funding support from the World Health Organization (WHO), the United States Centers for Disease Control and Prevention, and African Field Epidemiology Network. It has a 60-bed admission capacity, a female-to-male bed space ratio of 20:40, a dedicated biosafety laboratory, emergency operations center, and a biosecurity unit.

Admission into the facility involved identification of laboratory-confirmed SARS-CoV-2 individuals by a remote triage team, coordinated by the State Epidemiologist and Department of Public Health followed by their transportation to the isolation facility through a COVID-19 evacuation ambulance. On arrival, consent was obtained, and baseline clinical assessments were conducted after which a bedspace was allocated to inpatient care consenting individuals. WHO criteria for discharge from the isolation facilities during the period of the study were as follows: (a) three days after resolution of symptoms and (b) two negative reverse transcription PCR (RT-PCR) SARS-CoV-2 results, at least 24 h apart.[21,22]

### Study data

Clinical and laboratory records of hospitalized and outpatient laboratory-confirmed COVID-19 cases seen between April 10, 2020 and July 31, 2020 were obtained. Laboratory-confirmed SARS-CoV-2 infection defined as a positive result following high – throughput sequencing or real-time RT-PCR assay of a nasopharyngeal swab, and oropharyngeal specimens based on WHO guidelines were required for inclusion.[23] Those excluded from the study included patients on Lopinavir/Ritonavir therapy before admission, those whose blood samples could not be obtained by day 2 of admission, patients with known history of liver disease, or those for whom a hepatitis B and C screening could not be done.

### Data collection

A blood sample was collected from all patients on Day 2 of admission and placed in EDTA bottle for full blood count, lithium heparin for serum electrolytes, urea, and creatinine (E/U/Cr) and LFT. This was analyzed using the MIURA 200 fully automated clinical chemistry analyzer (ISE Srl, Albuccione Italy). The LFT included ALT with reference value of <40 IU/L, AST with reference value <40 IU/L, total bilirubin with reference value <17.1 μmol/l, direct bilirubin with reference value of <4.3 μmol/l, alkaline phosphatase (ALP) with a reference value of <270 IU/L, total protein with a reference value of 64–83 g/L, and albumin with reference interval of between 35 and 52 g/L. An abnormal LFT was defined as any values of the AST, ALT, ALP, total bilirubin, direct bilirubin, total protein, and albumin greater than the upper limit of normal. Age-specific variations in the reference values of some LFT variables were taken into consideration. All patients were tested for hepatitis B and C.

Information recorded included patient demographic data, medical history, clinical features, laboratory, and radiographic findings. A team of experienced clinicians then reviewed and abstracted the relevant data which were collected from electronic medical records and entered in a Microsoft Excel spreadsheet (2019). The Research Unit of the FCT Health and Human Services Secretariat coordinated this process. All variables required double entry, with reconciliation by a third party. Where missing data were noted, clarification requests were sent to the Center Case Manager who either contacted the attending clinicians or verified from the source records. As at July 31, 2020 (NCDC update week 31), 206 cases were discharged,[24] of which five cases had incomplete or missing records on demographic and/or clinical information ($n$ = 5) and had to be excluded. No patient deaths were recorded over the period under study.

### Study variables

The study outcome of interest was at least one abnormal LFT result obtained from samples collected at admission. Individuals with any of the various signs and symptoms of COVID-19 (such as fever, cough, sore throat, malaise, headache, muscle pain, nausea, vomiting, diarrhoea, loss of taste, and smell) but who did not have shortness of breath, dyspnoea, or abnormal chest imaging were described as having mild disease. Those with evidence of lower respiratory disease during clinical or radiological assessment and who had an oxygen saturation (SpO2) ≥94% on room air at sea level were categorized as moderate COVID-19. Severe illness, however, described individuals with SpO2 <94% on room air at sea level, a ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (PaO2/FiO2) <300 mm Hg, respiratory frequency >30 breaths/min, or lung infiltrates >50%.[25]

### Statistical analysis

Statistical analysis was carried out with IBM SPSS Statistics for Windows, Version 24.0. Armonk, NY, USA: IBM Corp. Descriptive statistics were used to analyze demographic data, medical history, COVID-19 features, and laboratory findings. The association between the categorical data was determined using Pearson's Chi-square test, while the association between demographic information, medical history, and abnormalities in LFTs was investigated using bivariate and multivariate logistic regression models. To assess continuous variables, tests of normality were used. $P < 0.05$ was considered statistically significant, and tests were 2 tailed with confidence levels of 95%.

## Results

### Sociodemographic and clinical characteristics of patients

Analysis was done on a total of 201 cases (mean age of 39.3 years [standard deviation ± 13.1], range 1–70; 65.7% [$n$ = 132 males]; 198 Nigerians, 1 Arab, 1 Caucasian and 1 Asian patient). Forty-nine-point two percent ($n$ = 99) of patients were overweight or obese, with 30.8% ($n$ = 62) overweight and 18.4% ($n$ = 37) obese. Majority of patients 95.5% ($n$ = 192) were non-smokers and 73.6% ($n$ = 148) did not consume alcohol [Table 1].

Mild COVID-19 was present in about 68.2% of patients, and 26.8% were asymptomatic at presentation, only 3% presented in severe disease with respiratory distress, and no patients were jaundiced. Approximately 33.3% of patients had one or more comorbidities, of which the most frequent were hypertension (22.9%) and diabetes mellitus (7.5%) [Table 2]. Six patients (3.0%) screened positive for hepatitis B,

**Table 1: Sociodemographic and lifestyle characteristics of coronavirus disease-2019 patients**

| Variables | Frequency ($n$=201), $n$ (%) |
|---|---|
| Age Group (years) | |
| <20 | 11 (5.5) |
| 20-29 | 35 (17.4) |
| 30-39 | 54 (26.9) |
| 40-49 | 47 (23.3) |
| 50-59 | 43 (21.4) |
| ≥60 | 11 (5.5) |
| Gender | |
| Female | 69 (34.3) |
| Male | 132 (65.7) |
| Occupation | |
| Healthcare workers | 68 (33.8) |
| Nonhealthcare workers | 133 (66.2) |
| Employment status | |
| Employed | 188 (93.5) |
| Unemployed | 13 (6.5) |
| Ethnicity | |
| Hausa | 41 (20.4) |
| Igbo | 40 (19.9) |
| Yoruba | 20 (10.0) |
| Others* | 100 (49.7) |
| Educational level | |
| No formal education | 15 (7.5) |
| Primary | 11 (5.5) |
| Secondary | 39 (19.4) |
| Tertiary | 136 (67.6) |
| BMI | |
| <18.5 | 5 (2.5) |
| 18.5-24.9 | 97 (48.3) |
| 25.0-29.9 | 62 (30.8) |
| 30.0-34.9 | 36 (17.9) |
| ≥35.0 | 1 (0.5) |
| Alcohol intake | |
| No alcohol intake | 148 (73.6) |
| <3 alcohol units/day | 42 (20.9) |
| ≥3 alcohol units/day | 11 (5.5) |
| Smoking status | |
| No smoker/past smoker (≥10 years) | 192 (95.5) |
| Past smoker (<10 years) | 2 (1.0) |
| Current smoker (<20 cigarettes/day) | 5 (2.5) |
| Current smoker (≥20 cigarettes/day) | 2 (1.0) |

*Others - Arab, Bini, Efik, Esan, Filipino, Gbagyi, Ibibio, Igala, Igbira, Ijaw, Ikwerre, Kalabari, Pakistani, Turkish. BMI: Body mass index

while four patients (2.0%) screened positive for hepatitis C. Cough (38.3%), fever (33.8%), fatigue (26.9%), poor appetite (26.9%) and headache (25.4%) were the most common symptoms at presentation, while the least common were insomnia (3.1%) and nausea (0.5%) [Table 3]. The median length of hospital stay was 13 days and most patients (99%) were discharged home following management and 1% ($n = 2$) referred for intensive care.

Approximately 93.5% ($n = 188$) of patients had normal chest radiological findings, with pulmonary infiltrates observed in 4% [Table 3].

Approximately 33.3% of the patients with hepatitis B positive test results had altered liver enzymes, while 75% of those with hepatitis C positive test results recorded alterations in their liver enzymes.

Altered renal function tests (derangements in urea and/or creatinine levels) were observed in about 7.5% ($n = 15$) of patients, whereas alterations in liver enzymes were observed in 52.7% ($n = 106$) of patients [Figure 1]. The most common changes in liver enzymes were observed in direct bilirubin (41.3%; $n = 83$), ALT (20.4%; $n = 41$) and ALP (18.4%, $n = 37$) [Figure 2]. The median values of patient LFTs were 4.0 for direct bilirubin, 26.0 for ALT, and 170.0 for ALP [Table 4].

### Association between COVID-19 patient characteristics and abnormal liver function tests

Table 5 illustrates the association between patient sociodemographic characteristics and abnormal LFTs. Age ($P < 0.015$), gender ($P = 0.001$), occupation ($P = 0.019$), and educational level ($P = 0.017$) were significantly associated with abnormal LFTs. No association was demonstrated between comorbidity, BMI, or alcohol intake and abnormal LFTs in this study. Following logistic regression analysis [Table 6], patients aged 20–29 years (odds ratio [OR]: 0.067; with 95% confidence interval [CI]: 0.005–0.916; $P = 0.043$), and female patients (OR: 0.367; with 95% CI: 0.186–0.724; $P = 0.004$) showed significantly lower odds of having abnormal LFTs.

## Discussion

This study aimed to describe the sociodemographic, clinical characteristics, and alterations in LFTs of 201 laboratory (PCR) positive COVID-19 patients managed in Abuja, Nigeria's FCT. Findings revealed a mean age of 39 years with a male predominance. Most patients were symptomatic, approximately one-third had one or more comorbidities, about one-quarter of patients regularly consumed alcohol, and the median duration of hospitalization recorded was about 13 days. The symptomatic presentation is similar to findings reported from Tokyo, Japan, during the first wave of the pandemic, where the majority of patients were male and presented in mild forms of COVID-19.[26] Globally, reports of the COVID-19 pandemic have highlighted a male preponderance in line with our study findings.

**Table 2: Associated comorbidities in the cohort of coronavirus disease-19 patients**

| Variables | Frequency ($n=201$), *n* (%) |
|---|---|
| Hypertension | 46 (22.9) |
| Diabetes | 15 (7.5) |
| Peptic ulcer disease | 11 (5.5) |
| Asthma | 8 (4.0) |
| Chronic liver disease | 2 (1.0) |
| Retroviral disease | 2 (1.0) |
| Stroke | 2 (1.0) |
| Anxiety disorder | 4 (2.0) |
| Bipolar affective disorder | 2 (1.0) |

**Table 3: Clinical features of the 201 coronavirus disease-19 patients**

| Features | Frequency ($n=201$), *n* (%) |
|---|---|
| General | |
| Fever and chills | 68 (33.8) |
| Easy fatiguability | 54 (26.9) |
| Body aches | 44 (21.9) |
| Respiratory | |
| Cough | 77 (38.3) |
| Chest pain | 34 (16.9) |
| Nasal congestion | 45 (22.4) |
| Shortness of breath | 25 (12.4) |
| Gastrointestinal | |
| Loss of appetite | 54 (26.9) |
| Painful swallowing | 28 (13.9) |
| Diarrhoea | 26 (12.9) |
| Abdominal discomfort | 24 (11.9) |
| Vomiting | 7 (3.5) |
| Nausea | 1 (0.5) |
| Neurological | |
| Headache | 51 (25.4) |
| Anosmia | 45 (22.4) |
| Dysgeusia | 44 (21.9) |
| Insomnia | 6 (3.1) |
| Chest radiological findings | |
| Normal | 188 (93.5) |
| Unilateral infiltrates | 3 (1.5) |
| Bilateral infiltrates | 5 (2.5) |
| Hypertensive features | 5 (2.5) |

**Table 4: Measures of distribution of coronavirus disease-19 patient liver function test results**

| Variables | Mean | Median | Mode | SD | Minimum | Maximum |
|---|---|---|---|---|---|---|
| Total bilirubin | 12.1 | 10.2 | 8.0 | 8.5 | 2.1 | 89.0 |
| Direct bilirubin | 4.5 | 4.0 | 3.2 | 2.1 | 1.3 | 13.4 |
| AST | 30.7 | 23.0 | 21.0 | 24.8 | 12.0 | 245.0 |
| ALT | 36.5 | 26.0 | 15.0 | 33.8 | 7.0 | 307.0 |
| ALP | 212.7 | 170.0 | 143.0 | 141.8 | 5.0 | 1169.0 |
| Total protein | 73.5 | 73.9 | 70.0 | 6.5 | 52.8 | 96.0 |
| Albumin | 46.7 | 47.0 | 47.0 | 4.9 | 4.3 | 54.1 |

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, SD: Standard deviation

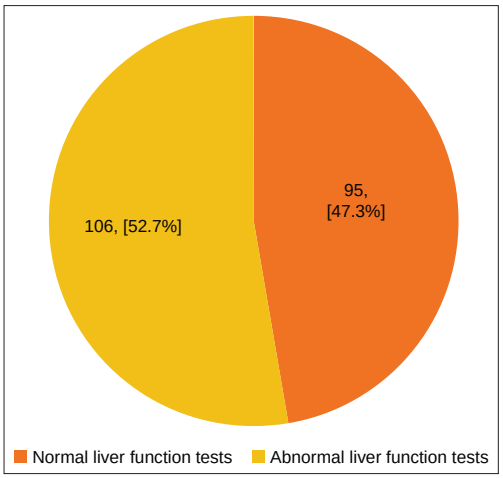


**Figure 1:** Distribution of abnormal liver function tests in COVID-19 patients in Abuja Nigeria

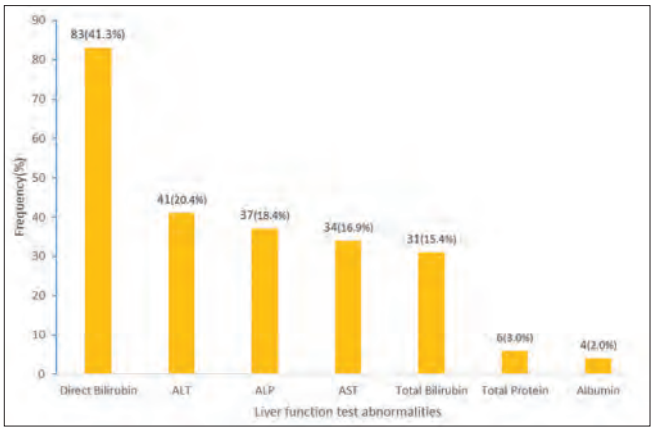


**Figure 2:** Frequencies of different liver function test abnormalities. ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, AST: Aspartate aminotransferase

**Table 5: Association between sociodemographic factors and alterations in liver function tests**

| Variables | Alteration in liver function | | $\chi^2$ | *P* |
|---|---|---|---|---|
| | Yes (*n*=106), *n* (%) | No (*n*=95), *n* (%) | | |
| Age group (years) | | | | |
| <20 | 10 (9.4) | 1 (1.1) | 14.113 | 0.015* |
| 20-29 | 18 (17.0) | 17 (17.9) | | |
| 30-39 | 22 (20.8) | 32 (33.7) | | |
| 40-49 | 29 (27.4) | 18 (18.9) | | |
| 50-59 | 24 (22.6) | 19 (20.0) | | |
| ≥60 | 3 (2.8) | 8 (8.4) | | |
| Gender | | | | |
| Male | 81 (76.4) | 51 (53.7) | 11.482 | 0.001* |
| Female | 25 (23.6) | 44 (46.3) | | |
| Occupation | | | | |
| Healthcare workers | 28 (26.4) | 40 (42.1) | 5.510 | 0.019* |
| Nonhealthcare workers | 78 (73.6) | 55 (57.9) | | |
| Educational level | | | | |
| No formal education | 12 (11.4) | 3 (3.2) | 10.237 | 0.017* |
| Primary | 8 (7.5) | 3 (3.2) | | |
| Secondary | 24 (22.6) | 15 (15.7) | | |
| Tertiary | 62 (58.5) | 74 (77.9) | | |

*Significant at 95%

More than half (52.7%) of the patients analyzed had altered liver function tests as indicated by elevations in one or more of the liver enzymes, namely, ALT, ALP, AST, and bilirubin. This finding correlates with observations from Italy's Lombardi region, where approximately half of the patients studied exhibited elevations in liver enzymes at admission.[15] Study reports from California, USA, however, cite slightly less at around 40%, while another US study reported elevated liver enzymes in as high as 69% of patients.[27,28] In the Bloom *et al*. study in the US, however, AST elevations were more frequent than ALT.[28] The case was the reverse in our study. In the United States, among the first twelve patients managed for COVID-19, all those who were hospitalized recorded elevated aminotransferase levels at admission.[29] Findings from an evaluation of hepatic enzyme changes in COVID-19 patients in Iran showed similarities to our study in ALP and direct bilirubin levels. However, the proportion of this study's patients with elevations in AST and ALT were lower than those recorded in Iran (20.4% and 16.9% compared to 30.3% and 29.2% in Iran).[30] The Iran study and several other studies from China and the United States have linked elevated liver enzymes in COVID-19 to severity of disease,[30-33] however, given that only two of our study's cases developed complications that necessitated intensive care transfer with all other patients recovering without complication, this difference could not be illustrated.

Liver dysfunction in COVID-19 has been linked with risk factors such as the administration of lopinavir/ritonavir-based treatment regimens, direct viral-initiated inflammatory response, cytopathic injury, and ischemia.[8,13-15] Following admission, the use of medications such as hydroxychloroquine, antibiotics, and antivirals such as lopinavir and ritonavir, is a critical risk factor for liver damage.[34-38] In this study, however, all patients studied had their blood samples collected before the initiation of treatment regimens, and a medication history that included lopinavir/ritonavir was a condition for exclusion from participating in the study.

A significant proportion (69.6%) of patients experienced gastrointestinal symptoms at the time of presentation. These symptoms included mostly loss of appetite, dysphagia, diarrhea, and abdominal discomfort, with vomiting and nausea experienced the least. Loss of appetite and diarrhea were commonly reported in California, USA patients, in addition to higher frequencies of nausea and vomiting observed.[33] However, for the patients in the US cohort, the gastrointestinal symptoms developed during admission, unlike our findings which were at presentation. Multimorbidity, medication regimen, polypharmacy, and dietary differences may account for these dissimilarities in presentation.[15,39]

**Table 6: Logistic regression for alterations in liver function tests**

| Variables | *B* | *P* | OR | 95% CI for OR | |
|---|---|---|---|---|---|
| | | | | Lower | Upper |
| Age | | | | | |
| <20[RC] | | | | | |
| 20-29 | −2.708 | 0.043* | 0.067 | 0.005 | 0.916 |
| 30-39 | −1.102 | 0.166 | 0.332 | 0.070 | 1.578 |
| 40-49 | −0.548 | 0.469 | 0.578 | 0.131 | 2.547 |
| 50-59 | −1.284 | 0.094 | 0.277 | 0.062 | 1.243 |
| ≥60 | −1.271 | 0.101 | 0.281 | 0.061 | 1.283 |
| Sex (female) | −1.002 | 0.004* | 0.367 | 0.186 | 0.724 |
| Occupation (nonhealth worker) | 0.321 | 0.380 | 1.378 | 0.674 | 2.820 |
| No formal education RC | | | | | |
| Primary | −0.991 | 0.199 | 0.371 | 0.082 | 1.682 |
| Secondary | −0.609 | 0.409 | 0.544 | 0.128 | 2.306 |
| Tertiary | −0.239 | 0.581 | 0.787 | 0.337 | 1.839 |

*Significant at 95%. RC: Reference category, OR: Odds ratio, CI: Confidence interval

Age groups of 20–29 years and female gender reduced the likelihood of altered LFTs in this study. Studies have referred to a gender-specific discrepancy in the expression levels and patterns of the angiotensin-converting enzyme 2, Type II transmembrane Serine Protease (TMPRSS2), and the androgen receptor genes.[40-42] These may therefore be responsible for the different susceptibility or responses to COVID-19 expressed in male patients as revealed in the outcomes of our study. The SARS-CoV-2 virus binding to these ACE-2 receptors in hepatocytes and cholangiocytes may provoke a systemic inflammatory response and liver damage that is observed more frequently in male COVID-19 patients. Similarly, ACE-2 has been found to increase with age, possibly explaining the lower odds of alterations in hepatic enzyme found in the age group 20–29 years in this study (age group <20 years was our reference category).

## Conclusion

Given the reported frequency of occurrence of altered liver function and elevated liver enzymes in COVID-19, our research serves to highlight the importance of conducting LFTs in hospitalized COVID-19 patients in low- and middle-income settings. Our study findings of the protective effects of younger age and female gender would support optimal management and strict monitoring of patients to limit the risk of developing acute liver failure and aid in stratifying patients for early access and where necessary prompt referral to intensive care. Given the enormous pressure the COVID-19 pandemic has laid on health systems globally, this is especially important as efficiencies in allocation and utilization of health-care resources are critical.[43-45]

Future longitudinal studies focusing on the occurrence and impact of liver impairment in COVID-19 patients and further exploration of the outcomes of COVID-19 in patients with chronic liver diseases are thus important.

## Study limitations

Limitations of this study included the fact that we were unable to present a direct causal link between COVID-19 and altered LFTs. As we did not obtain liver specimens from these patients, a direct SARS-CoV-2 cytopathic effect could not be ascertained. In addition, given the retrospective nature of the study, we could not establish whether confounding factors such as prior intake of certain hepatotoxic medications or other undiagnosed liver diseases may have contributed to liver function alterations observed. We thus endeavored to conduct laboratory evaluations before COVID-19 therapy was administered. As serial laboratory evaluations were not done, we cannot speak to a direct association between changes in these variables and patient outcomes. Although further management of patients with hepatitis B and C seropositivity and excessive alcohol intake was done, the outcomes were unavailable for presentation among our findings. Furthermore, as baseline LFT values were not known for all patients before COVID-19 infection, we cannot categorically state that the abnormal LFTs were due to COVID-19; and other causes of liver disease were not investigated, neither were patient clotting profiles such as international normalized ratio, prothrombin time, and partial thromboplastin time (PTTK) assessed. Furthermore, the small sample size of the study may limit the generalizability of our findings.

However, we have herein presented the clinical features, laboratory data, and health outcomes of a cohort of predominantly Black African COVID-19 patients, focusing on alterations in their LFTs. Furthermore, a major strength of our study is that we were able to ascertain the prevalence of HBV and HCV in the patients, and our findings were disaggregated by age and gender. We noted overt obesity in a small proportion of these patients, and few were active alcohol drinkers and smokers. This setting of our study also allowed us the opportunity to present data on liver enzyme derangements in patients with a considerably low prevalence of unhealthy lifestyle habits.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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