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# Biochemical Profile of Patients Hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020

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## Abstract

**Background:** COVID-19, an infectious viral disease, has caused a global health crisis. Most cases remain asymptomatic. The majority of patients have mild symptoms while about 15% develop a severe form. The clinical spectrum of SARS-CoV-2 infection appears broad, encompassing asymptomatic infection, upper respiratory tract symptoms, and severe viral pneumonia with respiratory failure that can lead to death. Laboratory tests play an important role in the management of COVID-19 patients. In addition to being essential for the diagnosis, several biological analyses make it possible to identify the inflammatory processes and the potential complications of this disease. This study attempted to identify biochemical assays that could help in the prognosis of the disease to ensure early management. **Methods:** This was a descriptive study. It focused on patients hospitalized for COVID-19 from March 19, 2020, to January 26, 2021, at the Infectious Disease Management Centre in Lomé (Togo). Medians were compared using the (Mann-Whitney and Wilcoxon) test and frequencies were compared using the Chi-square test or Fisher's exact test. **Results:** We included 782 patients. The median age was 41 years IQR from 32 to 55. We observed several biochemical abnormalities in

varying proportions for all biochemical parameters studied. Compared to non-serious patients, critically ill patients at admission had more frequently elevated urea, creatinine, transaminases, TG, GGT, CRP and blood glucose. Also, they had more frequent decreases in total cholesterol, HDL-c, blood chloride, and blood calcium. As for patients who died during hospitalization, compared with healed patients, they had more frequent elevations of urea, creatinine, AST, ALT, GGT. CRP and blood glucose. They also had a more frequent decrease in total cholesterol, HDL-c, blood chloride, blood calcium, and blood glucose ($p = 0.025$). **Conclusion:** This study shows that COVID-19 is a multi-organ systemic inflammatory viral disease that should be systematically investigated once the diagnosis is confirmed.

## Keywords

COVID-19, Biochemical Anomalies, Togo, 2022

## 1. Introduction

Coronaviruses (CoV) are viruses known to cause generally mild respiratory infections in humans and animals. Some strains may be more virulent to humans, such as those of severe acute respiratory syndrome (SARS) and MERS (*Middle East respiratory syndrome*). SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) is the official name of the new coronavirus identified in the city of Wuhan, the capital of Hubei province in China. It is the etiological agent of the infectious lung disease epidemic that spread around the world in late December 2019. This disease has been named COVID-19 (*Coronavirus Disease 2019*) by the World Health Organization (WHO) on February 11, 2020. On January 30, 2020, WHO declared the situation a public health emergency of international concern. On March 11, it declared a global pandemic [1]. As of November 20, 2020, the number of people worldwide who have contracted COVID-19 was 57,274,018, including 1,368,000 deaths [2]. In Togo, the first case of contamination was detected on March 5, 2020 [3], as of November 19, 2020, there were 2771 confirmed cases, 2101 of which were cured, and 63 deaths [4]. SARS-CoV-2 infection is still little known. The majority of patients have mild symptoms, while about 15% develop a severe form. The clinical spectrum of SARS-CoV-2 infection appears to be broad, encompassing asymptomatic infection, upper respiratory tract symptoms, and severe viral pneumonia with respiratory failure that can lead to death [5]. Indeed, some authors have reported that SARS-CoV-2 infection, far from being a respiratory disease, is above all a multisystem pathology responsible for multi-visceral lesions that can worsen the prognosis of the disease [6]. Thus, the evolution of COVID-19 remains unpredictable. The scientific community needs reliable biomarkers linked to the progression of COVID-19 to identify high-risk patients. The rapid spread of the disease requires immediate categorization of patients into risk groups after diagnosis to ensure optimal re-

source allocation. Biomarkers are needed to identify patients whose disease will rapidly progress to severe complications and death [7]. In this sense, biochemical examinations, which include a wide range of tests, from the exploration of some organ functions to the hydro electrolytic balance and the evaluation of inflammatory processes, are important. Also, several studies concerning biochemical parameters have been carried out throughout the world, with results that are often heterogeneous [7]-[12].

To date, to our knowledge, there is no data in Togo on the biochemical profile of patients hospitalized for COVID-19. However, since the creation of the Centre for the Management of Infectious Diseases, several patients have been admitted to this structure. Most of them have benefited from a biochemical assessment on admission, some as part of a systematic pre-therapeutic assessment, and others depending on the clinical picture. This study attempts to identify biochemical analyses whose measurement could help in the prognosis of the disease to ensure early management. The general objective is to describe the biochemical profile of the admission of adult patients hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020.

## 2. Methods

### 2.1. Scheme and Study Period

This was a descriptive study that focused on patients hospitalized from March 19, 2020, to January 26, 2021, at the Infectious Disease Management Centre in Lomé, Togo. In this facility, all symptomatic patients diagnosed as COVID-19 positive by RT-PCR on nasal or oropharyngeal swabs were admitted. On admission, most patients had routine blood sampling for blood glucose, renal, and liver function tests. Others, depending on the clinical picture, benefited from a lipid profile, an electrolyte panel, and a C-reactive protein (CRP) test.

We performed an exhaustive sampling of these patients with the inclusion criteria of being 18 years or older and having performed a biochemical test of interest.

### 2.2. Analysis of Blood Samples

The blood tests were performed at the biochemistry laboratory of the "Institut National d'Hygiène (INH)". These results were obtained from fasting blood samples taken once on admission only.

### 2.3. Material

The Roche-Hitachi Cobas C311 chemical analyzer was used to analyze the blood samples.

### 2.4. Reagents, Assay Principles, and Reference Values

#### 2.4.1. Renal Function Tests

**Uremia**

Reagent: Urea/BUN.

Principle: a kinetic test using urease and glutamate dehydrogenase.

Reference values: 0.15 - 0.45 g/L.

**Creatinine**

Reagents: creatinine plus ver.2 (CREP2).

Principle: enzymatic method with creatininase, creatinase, and sarcosine oxidase.

Reference values: Female: 5.1 - 9.5 mg/L, Male: 6.7 - 11.7 mg/L.

### 2.4.2. Hepatic (Liver) Function Panel

**Aspartate Aminotransferase/Alanine Aminotransferase (AST/ALT)**

Reagent: ASTLP/ALTLP: Aspartate/Alanine aminotransferase according to International Federation of Clinical Chemistry (IFCC).

Principle: according to IFCC/standard method 94 with pyridoxal phosphate activation, measured at 37˚C [13].

Reference values: Female: 10 - 35 IU/L; Male: 10 - 50 IU/L.

**Ɣ-Glutamyl Transferase (GGT)**

Reagent: GGT-2: ɣ-Glutamyl transferase ver.2 standardization IFCC/Szasz.

Principle: enzymatic colorimetric test.

Reference value: Male: <60 IU/L; Female: <40 IU/L.

**Alkaline Phosphatase (ALP)**

Reagent: Alkaline Phosphatase acc. to IFCC Gen.2 (ALP2).

Principle: colorimetric test according to a standardized method (measurement at 37˚C).

Reference values: Male: 40 - 129 U/L, female: 35 - 104 U/L.

### 2.4.3. Lipid Profile

**Total Cholesterol (TC)**

Reagent: Cholesterol Gen.2 (CHOL2).

Principle: enzymatic colorimetric method.

Reference value: <0.2 g/L .

**High-Density Lipoprotein (HDL-c)**

Reagent: HDL-c-cholesterol Gen.4 (HDL-C4).

Principle: enzymatic colorimetric test in homogeneous phase.

Reference value: female: 0.45 - 0.65 g/L.

**Low-Density Lipoprotein (LDL-c)**

Reagent: LDL-c-Cholesterol Gen.3 (LDL C3).

Principle: enzymatic colorimetric test in homogeneous phase.

Reference value: <0.1 g/L.

**Triglycerides (TG)**

Reagent: triglycerides (TRIGL).

Principle: enzymatic colorimetric test.

Reference value: <1.5 g/L.

### 2.4.4. Electrolyte Panel

**Natremia ($Na^+$), Kalemia ($K^+$) and Chloremia ($Cl^-$)**

Reagent: Indirect ion-selective electrode (ISE) Na-K-Cl for Gen.2.

Principle: Electromotive force (EMF) using an ISE.

Reference value: $Na^+$ 136 - 145 mmol/L; $K^+$ 3.5 - 5.1 mmol/L; $Cl^-$ 98 - 107 mmol/L.

#### 2.4.5. Calcium ($Ca^{2+}$)

Reagent: Calcium Gen.2.

Principle: Colorimetric test.

Reference value: 8.6 - 10.0 mg/dL.

#### 2.4.6. Magnesium ($Mg^{2+}$)

Reagent: Magnesium Gen.2.

Principle: Colorimetric test, endpoint method.

Reference value: 1.6 - 2.6 mg/dL.

#### 2.4.7. Blood Glucose

Reagent: Glucose HK (GLUC3).

Principle: Ultraviolet (UV) test, hexokinase enzymatic reference method.

Reference value: 0.7 - 1.09 g/L.

#### 2.4.8. C-Reactive Protein (CRP)

Reagent: C-reactive Protein Gen.3 (CRPL3).

Principle: Immunoturbidimetric test on latex particles.

Reference value: <5 mg/L.

### 2.5. Interpretation of Results

#### 2.5.1. Renal Function Tests

Hyperuremia of urea was considered when urea value > 0.45 g/L, a high level of creatinine was considered when creatinine level > 12 mg/L in women and >14 mg/L in men.

#### 2.5.2. Hepatic (Liver) Function Panel

Elevated transaminases were considered when AST values >1.5 N (>57 IU/L) in men (46.5 IU/L) in women, and ALT >1.5 N (60 IU/L) in men and 48 IU/L in women.

Alkaline phosphatase (ALP) was considered elevated when the level was >1.5 N (418.5 IU/L).

Gamma-glutamyl transferase (GGT) were considered elevated when > 1.5 N (78 IU/L).

#### 2.5.3. Electrolyte Panel

Kalemia ($K^+$): Hypo- and hyperkalemia were considered when kalemia levels were, respectively, < 3.3 mmol/L and > 5.1 mmol/L.

Natremia ($Na^+$): Hypo and hypernatremia were considered for natremia < 135 mmol/L and > 145 mmol/L, respectively.

#### 2.5.4. Blood Calcium ($Ca^{2+}$)

Hypo and hypercalcemia were defined for blood calcium < 90 mg/L and >110 mg/L respectively.

#### 2.5.5. Magnesemia ($Mg^{2+}$)

Hypo and hypermagnesemia were considered for magnesemia < 16 mg/L and >27 mg/L respectively.

#### 2.5.6. Lipid Profile

Total cholesterol (TC): Hypo and hypercholesterolemia were defined for cholesterol levels < 1.4 g/L and >2.2 g/L respectively.

HDL-c was considered decreased if <0.4 g/L in men and <0.35 g/L in women; increased if >0.60 g/L in men and >0.65 g/L in women.

LDL-c was considered increased if it was greater than 1.5 g/L.

Triglycerides (TG): Hypo and hypertriglyceridemia were defined for triglyceride levels < 0.60 g/L and >1.65 g/L respectively.

#### 2.5.7. Other Medical Check-Ups

Hypo and hyperglycemia were defined for blood glucose levels < 0.5 g/L and >1.10 g/L respectively.

CRP was considered increased if >6 mg/L.

D-dimer was considered increased if >500 µg/L.

HbA1c was considered increased if >6.5%.

### 2.6. Data Collection Technique

Patient data were entered into an electronic xlsform deployed through the KoboToolbox platform. Variables of interest were secondarily extracted and analyzed.

### 2.7. Variables of Interest

We collected:

Socio-demographic characteristics data: Age, gender.

Anamnestic data: Patient medical history or co-morbidities such as hypertension, diabetes, heart diseases, asthma, lung disease, allergies...

Clinical data: Symptoms on admission such as cough, fever, dyspnea, myalgia, anosmia, ageusia...

We used WHO clinical classification of COVID-19.

Biochemical data:

Renal function tests: urea (g/L), creatinine (mg/L).

Liver function tests: transaminases (IU/L), ALP (IU/L), GGT (IU/L).

Electrolyte panel ($Na^+$, $Cl^-$, $K^+$, $Ca^{2+}$, $Mg^{2+}$).

Lipid profile (TC, LDL-c, HDL-c, TG) in g/L.

Other biological parameters: CRP (mg/L), blood glucose (g/L), D-dimer, glycated hemoglobin (HbA1c).

## 2.8. Operational Definitions

### Clinical Features

#### WHO Clinical Classification

There are five stages:

Asymptomatic stage: No clinical signs with a positive SARS-CoV-2 PCR test.

Mild stage: Symptoms of acute upper respiratory infection, including fever, fatigue, myalgia, cough, sore throat, runny nose, and sneezing without pneumonia.

Moderate stage: With pneumonia, fever, and frequent coughing some may have wheezing, but no obvious hypoxemia such as shortness of breath.

Severe stage: Rapid progression within 1 week, dyspnea, with central cyanosis, oxygen saturation below 92% on room air, with other manifestations of hypoxemia.

Critical stage: Patients with acute respiratory distress syndrome (ARDS) or respiratory failure, shock, multi-organ dysfunction referral [14].

For this study we considered patients classified as severe and critical to be serious and asymptomatic, mild and moderate patients to be non-serious.

## 2.9. Data Analysis

### 2.9.1. Seizure and Discharge

The data of interest were extracted from the electronic xlsform deployed through the KoboToolbox platform. The data were then cleaned by removing duplicates and correcting outliers. The database was then analyzed using R 4.0.4 software (*R Core Team*, Vienna) in the RStudio1.4 environment.

### 2.9.2. Statistical Analysis

The qualitative variables were presented according to their respective numbers and percentages, the quantitative variables according to their medians and interquartile ranges. Medians were compared using non-parametric tests (Mann-Whitney, Wilcoxon) and frequencies using the Chi-square test or Fisher's Exact test when indicated. The threshold of significance was $p < 0.05$. Missing data were not included in the analysis.

### 2.9.3. Ethics and Data Confidentiality

A password-protected computer was used for data entry and analysis. The data were processed anonymously. We also got an authorization of the centre to collect these data.

# 3. Results

## 3.1. Socio-Demographic and Clinical Characteristics

The median age was 41 years with an IQR [32 - 55]. The elderly (≥60 years) accounted for 17.6% (n = 138). More than half, 63.6% (n = 497) were male, with a sex ratio of 1.7. Comorbidities were observed in 49.00% (n = 383). The main comorbidities were hypertension (HTN) and diabetes in 45.9% (n = 176), and

31.0% (n = 119) respectively. On admission, 64.8% (n = 502) had at least one symptom. Cough and fever accounted for 31.1% (n = 244) and 26.6% (n = 209) respectively. On admission, serious forms accounted for 14.2% (n = 111). We recorded 10.2% (n = 80) deaths during hospitalization, while the majority, 89.8% (n = 702) were cured. Demographic and clinical characteristics are summarized in Table 1.

Table 1. Demographic and clinical characteristics of patients hospitalized at the Infectious Disease Management Centre in Lomé in 2020.

| Characteristic | Size (N = 782) | Proportion (%) |
|---|---|---|
| **Age** | | |
| <60 | 138 | 17.6 |
| ≥60 | 644 | 82.4 |
| **Sex** | | |
| Men | 497 | 63.6 |
| Women | 285 | 36.4 |
| **Comorbidities** | **383** | **49.0** |
| HTN | 176 | 45.9 |
| Diabetes | 119 | 31.0 |
| HTN and Diabetes | 72 | 18.7 |
| Obesity | 40 | 5.1 |
| Asthma | 25 | 3.2 |
| Peptic ulcer | 25 | 3.2 |
| HIV | 21 | 2.7 |
| Atopic terrain | 21 | 2.7 |
| Heart disease | 6 | 0.7 |
| Tuberculosis | 6 | 0.7 |
| Veinous thromboembolic disease | 5 | 0.6 |
| Cancer | 5 | 0.6 |
| Chronic renal failure | 3 | 0.3 |
| **Symptoms** | **502** | **64.8** |
| Cough | 244 | 31.1 |
| Fever | 209 | 26.6 |
| Dyspnea | 179 | 22.8 |
| Headache | 156 | 19.9 |
| Asthenia | 105 | 13.4 |
| Myalgia | 70 | 8.9 |
| Rhinitis | 62 | 7.9 |
| Anosmia | 52 | 6.6 |

**Continued**

| | | |
|---|---|---|
| Chest pain | 48 | 6.1 |
| Arthralgia | 42 | 5.3 |
| Aguesia | 36 | 4.6 |
| Diarrhoea | 20 | 2.5 |
| Abdominal pain | 20 | 2.5 |
| Vomiting | 20 | 2.5 |
| Sore throat | 15 | 1.9 |
| **Severity stage** | | |
| **Not serious** | **671** | **85.8** |
| Asymptomatic | 274 | 35.0 |
| Slight | 296 | 37.9 |
| Moderate | 101 | 12.9 |
| **Serious** | **111** | **14.2** |
| Severe | 84 | 10.7 |
| Critical | 27 | 3.5 |
| **Outcome of the disease** | | |
| Healed | 702 | 89.8 |
| Deceased | 80 | 10.2 |

### 3.2. Biochemical Parameters

Table 2 describes the biochemical characteristics of patients hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020. As shown in Table 2, the most common tests performed were transaminases in 72.1% (n = 565) followed by uremia in 71.6% (n = 561). CRP was performed in 17.3% (n = 134). Five hundred and forty-eight patients (70.3%) had venous blood glucose. D-dimer testing was the least performed test in 0.8% (n = 07). The medians of the renal, liver, lipid and electrolyte panel parameters were within the reference range. The medians for CRP with 12 mg/L IQR [6 - 48], glycated hemoglobin in diabetics 7.3% IQR [5.8 - 9.4], and D-dimer 1700 µg/L IQR [1330.9 - 3246.1] were increased. In the renal function tests, hyperuremia and high levels of serum creatinine were observed in 13.7% (n = 77) and 11.3% (n = 57) respectively. Regarding the liver function tests, AST, ALT and GGT were increased by 25.1% (n = 142), 16.1% (n = 91), and 24.3% (n = 95) respectively. As for the lipid profile, hypocholesterolemia was observed in 21.1% (n = 79), hypo-HDL-c in 20.5% (n = 41), and hypertriglyceridemia in 16.5% (n = 79). For the electrolyte panel, hypocalcemia was found in 41.2% (n = 47), hypochloremia in 24.0% (n = 46), hyponatremia in 12.2% (n = 22). Eighty patients (59.7%) had an elevated CRP. Hyperglycemia was observed in 33.0% (n = 183). Of the diabetic patients, 57.7% (n = 41) had a glycated hemoglobin greater than 6.5%.

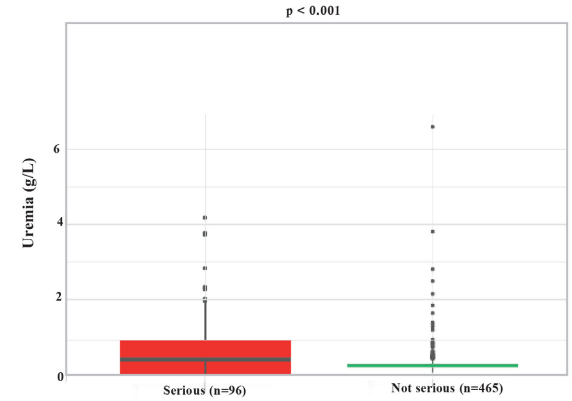
**Table 2.** Biochemical characteristics of patients hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020.

| Characteristic | Size n (%) | Reference value | Median [IQR] | Low n (%) | Normal n (%) | High n (%) |
|---|---|---|---|---|---|---|
| **Renal function tests** | | | | | | |
| Uremia (g/L) | 561 (71.6) | <0.45 | 0.2 [0.2 - 0.3] | NA | 484 (86.3) | 77 (13.7) |
| Serum creatinine (mg/L) | 506 (64.8) | <14 | 10.0 [9.0 - 12.0] | NA | 449 (88.7) | 57 (11.3) |
| **Hepatic function panel** | | | | | | |
| AST (IU/L) | 565 (72.1) | <57 | 38.0 [28.0 - 56.7] | NA | 423 (74.9) | 142 (25.1) |
| ALT (IU/L) | 565 (72.1) | <60 | 30.0 [20.2 - 45.0] | NA | 474 (83.9) | 91 (16.1) |
| GGT (IU/L) | 391 (49.9) | <78 | 39.0 [24.0 - 76.0] | NA | 296 (75.7) | 95 (24.3) |
| ALP (IU/L) | 74 (9.5) | <418.5 | 91.0 [69.0 - 163.0] | NA | 71 (95.9) | 03 (4.1) |
| **Lipid profile** | | | | | | |
| Total Cholesterol (g/L) | 374 (47.8) | 1.4 - 2.2 | 1.7 [1.4 - 2.0] | 79 (21.1) | 243 (65) | 52 (13.9) |
| HDL-cholesterol (g/L) | 205 (26.2) | 0.40 - 0.65 | 0.5 [0.4 - 0.6] | 42 (20.5) | 163 (79.5) | 12 (5.9) |
| LDL-cholesterol (g/L) | 208 (26.6) | ≤1.5 | 1.1 [0.7 - 1.3] | NA | 181 (87) | 27 (13.0) |
| Triglycerides (g/L) | 352 (44.9) | 0.6 - 1.65 | 0.9 [0.7 - 1.4] | 29 (8.2) | 265 (75.3) | 58 (16.5) |
| **Electrolyte panel** | | | | | | |
| Natremia (mmol/L) | 180 (23.1) | 135 - 155 | 140.0 [136.2 - 143.6] | 22 (12.2) | 158 (87.8) | 0 |
| Chloremia (mmol/L) | 192 (24.7) | 98 - 107 | 102.0 [98.0 - 105.0] | 46 (24.0) | 111 (57.8) | 35 (18.2) |
| Kalemia (mmol/L) | 192 (24.7) | 3.3 - 5.3 | 4.4 [4.0 - 4.8] | 10 (5.2) | 164 (85.4) | 18 (9.4) |
| Calcemia (mg/L) | 114 (14.6) | 90 - 110 | 91.0 [86.0 - 98.5] | 47 (41.2) | 63 (55.3) | 04 (3.5) |
| Magnesemia (mg/L) | 98 (12.6) | 16 - 27 | 20.0 [18.0 - 22.0] | 15 (15.3) | 77 (78.6) | 01 (01.1) |
| **Other** | | | | | | |
| CRP (mg/L) | 134 (17.3) | <6 | 12.0 [6.0 - 48.0] | NA | 54 (40.3) | 80 (59.7) |
| Blood glucose (g/L) | 548 (70.3) | 0.5 - 1.1 | 0.9 [0.8 - 1.2] | 05 (0.9) | 360 (65.7) | 183 (33.4) |
| HbA1c (%) | 71 (9.0) | <6.5 | 7.3 [5.8 - 9.4] | NA | 30 (42.3) | 41 (57.7) |
| D-dimer (µg/L) | 7 (0.9) | <500 | 1700.0 [1330.9 - 3246.1] | NA | 00 | 07 (100) |

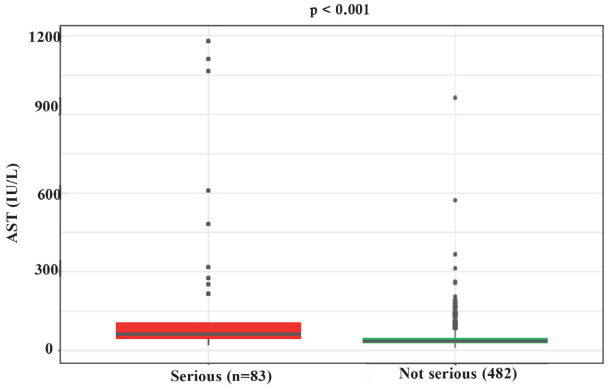
IQR= interquartile range; g = gram; mg = milligram; µg = microgram; L = litre; IU = international unit; mmol = millimole; n= size; (%) proportion estimated as a percentage; The percentages reported are percentages in rows; NA = not applicable. AST = aspartate aminotransferase; ALT = alanine aminotransferase; GGT = gamma-glutamyl transferase; ALP = alkaline phosphatase.

### 3.3. Biochemical Parameters According to the Stage of Severity

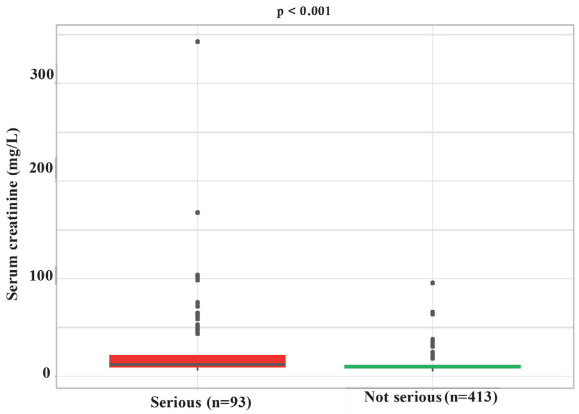
The Biochemical characteristics according to the stage of severity are summarized in Table 3 and Figure 1. In terms of renal function, patients with serious disease on admission had significantly higher blood urea and creatinine levels than non-serious patients ($p < 0.001$). Moreover, hyperuremia and high levels of serum creatinine predominated in severely affected patients, with respectively (43.8% vs. 7.5%: $p < 0.001$) and (36.6% vs. 5.6%: $p < 0.001$). As for hepatic function tests, the medians of AST, ALT, and GGT were statistically higher in the



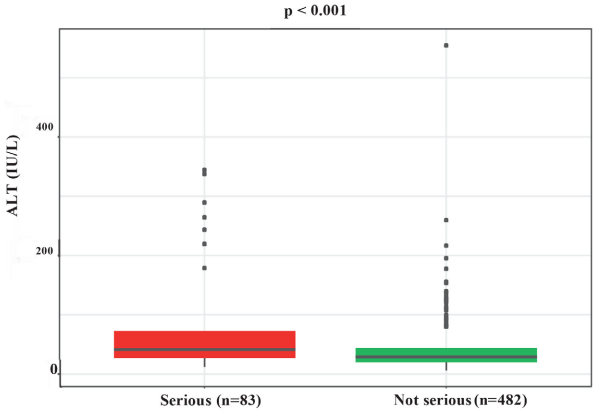

Distribution of uremia by severity stage.



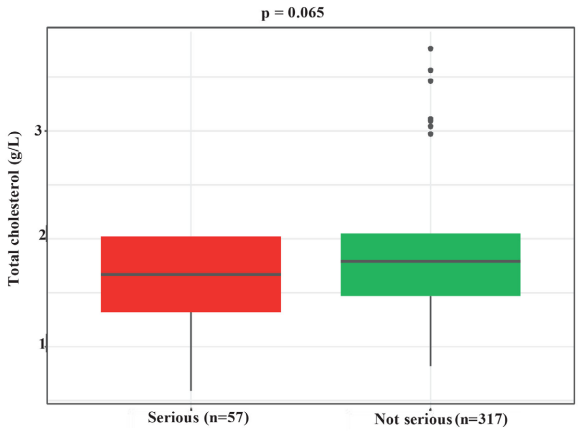

Distribution of AST by severity stage.



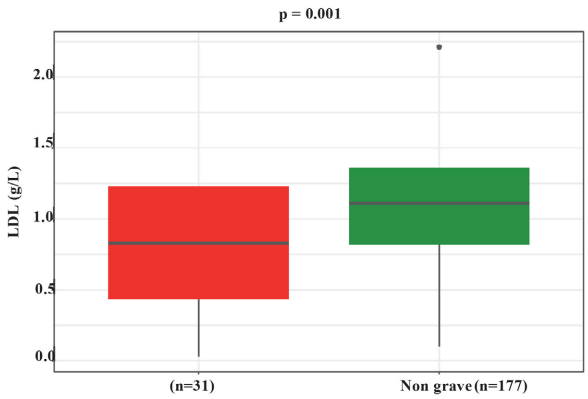

Distribution of serum creatinine by severity stage.



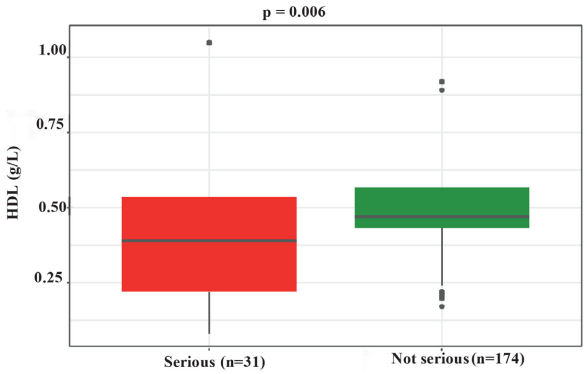

Distribution of ALT by severity stage.



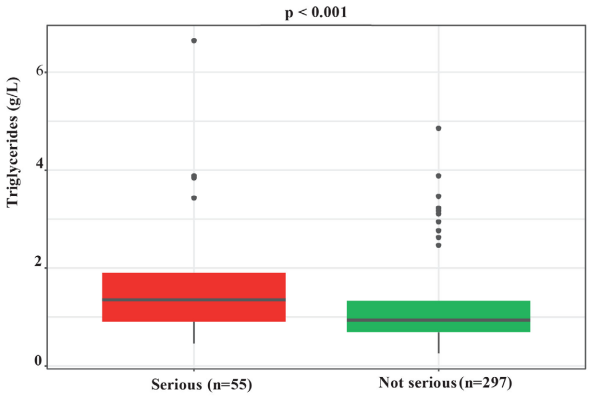

Distribution of total cholesterol by severity stage.




Distribution of LDL by severity stage.




Distribution of HDL by severity stage.




Distribution of triglycerides by severity stage.

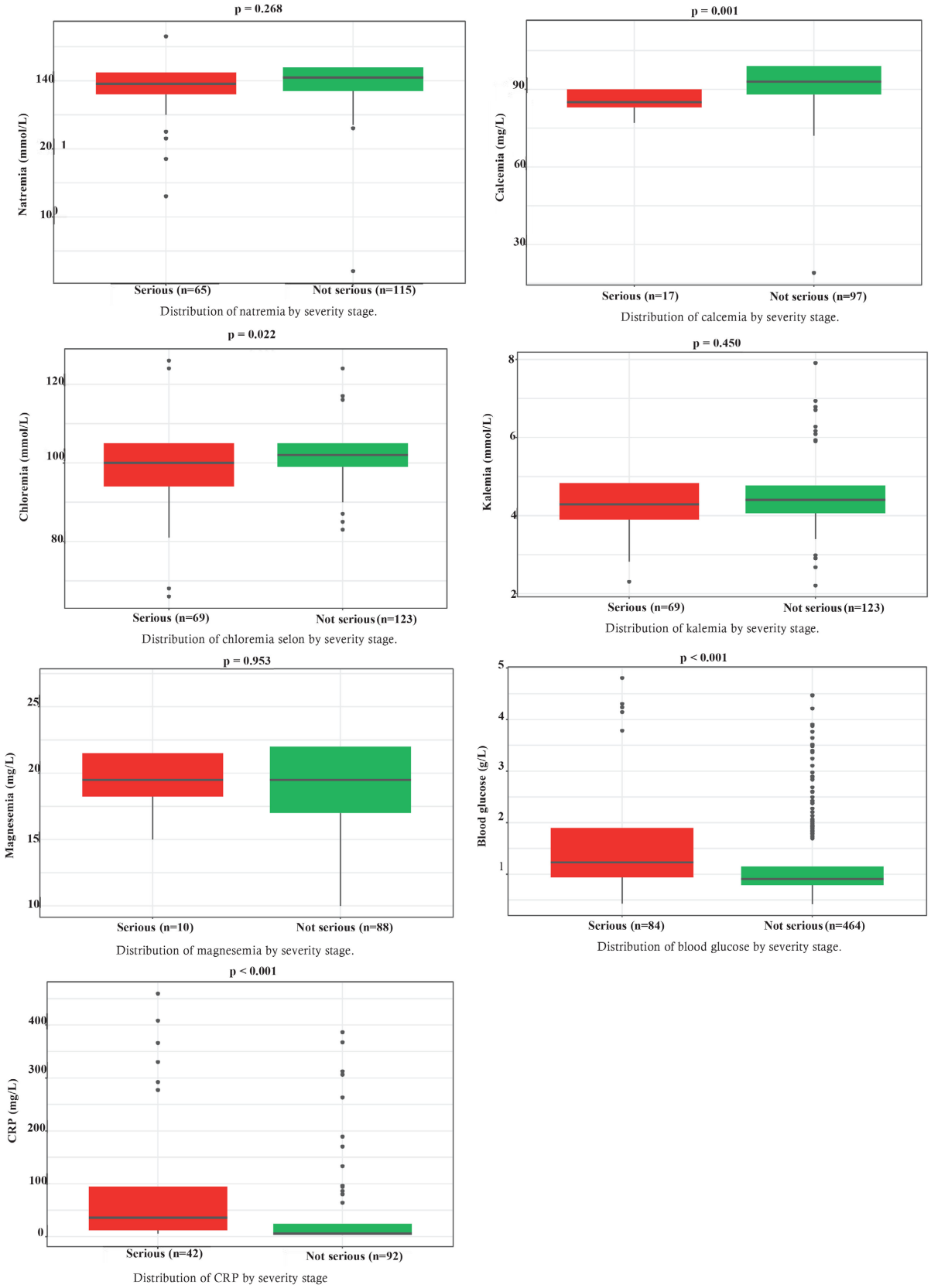



**Figure 1.** Biochemical characteristics according to the stage of severity of patients hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020.

**Table 3.** Biochemical characteristics according to the stage of severity and outcome of the disease of patients hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020.

| Characteristic | Total | Severity stage | | p | Outcome of the disease | | p |
|---|---|---|---|---|---|---|---|
| | | Not serious | Serious | | Healed | Deceased | |
| **Uremia (n)** | **561** | **465** | **96** | | **499** | **62** | |
| Median (g/L) [IQR] | 0.2 [0.2 - 0.3] | 0.2 [0.2 - 0.3] | 0.4 [0.3 - 0.9] | **<0.001** | 0.2 [0.2 - 0.3] | 0.6 [0.3 - 1.5] | **<0.001** |
| High n (%) | 77 (13.7) | 35 (7.5) | 42 (43.8) | **<0.001** | 42 (8.4) | 35 (56.5) | **<0.001** |
| **Serum creatinine (n)** | **506** | **413** | **93** | | **447** | **59** | |
| Median (mg/L) [IQR] | 10 [9 - 12] | 10 [8 - 12] | 12.2 [9 - 22] | **<0.001** | 10 [8 - 12] | 14 [9 - 48] | **<0.001** |
| High n (%) | 57 (11.3) | 23 (5.6) | 34 (36.6) | **<0.001** | 28 (6.3) | 29 (49.2) | **<0.001** |
| **AST** | **565** | **482** | **83** | | **514** | **51** | |
| Median (IU/L) [IQR] | 38 [28.0 - 56.7] | 36 [27 - 50] | 62 [44 - 108] | **<0.001** | 36 [28 - 52] | 65 [43 - 173] | **<0.001** |
| High n (%) | 142 (25.1) | 96 (19.9) | 46 (55.4) | **<0.001** | 112 (21.8) | 30 (58.8) | **<0.001** |
| **ALT (n)** | **565** | **482** | **83** | | **514** | **51** | |
| Median (IU/L) [IQR] | 30 [20.2 - 45.0] | 28.5 [20 - 44] | 41 [26 - 74] | **<0.001** | 29 [20 - 45] | 41 [24 - 71] | **<0.001** |
| High n (%) | 91 (16.1) | 62 (12.9) | 29 (34.9) | **<0.001** | 74 (14.4) | 17 (33.3) | **0.040** |
| **GGT (n)** | **391** | **340** | **51** | | **359** | **32** | |
| Median (IU/L) [IQR] | 39 [24 - 76] | 35 [23 - 62.5] | 79 [49 - 177] | **<0.001** | 36 [23 - 66] | 80.5 [58.5 - 195] | **<0.001** |
| High n (%) | 95 (24.3) | 69 (20.3) | 26 (51.0) | **<0.001** | 77 (21.4) | 18 (56.3) | **<0.001** |
| **ALP (n)** | **74** | **48** | **26** | | **61** | **13** | |
| Median (IU/L) [IQR] | 91 [69 - 163] | 91 [67.5 - 159.5] | 95.5 [75 - 164] | 0.575 | 91 [73 - 163] | 100 [65 - 163] | 0.847 |
| High n (%) | 3 (4.1) | 2 (4.2) | 1 (3.8) | 0.946 | 1 (1.6) | 2 (15.4) | 0.052 |
| **TC (n)** | **374** | **317** | **57** | | **342** | **32** | |
| Median (g/L) [IQR] | 1.7 [1.4 - 2.0] | 1.8 [1.5 - 2.1] | 1.7 [1.3 - 2.0] | 0.065 | 1.8 [1.5 - 2.1] | 1.5 [1.2 - 2.0] | **0.016** |
| Hypo-TC n (%) | 79 (21.1) | 61 (19.2) | 18 (31.6) | **0.035** | 64 (18.7) | 15 (46.9) | **0.020** |
| Hyper-TC n (%) | 52 (13.9) | 47 (14.8) | 5 (8.8) | 0.224 | 48 (14) | 4 (12.5) | 0.810 |
| **HDL-c (n)** | **205** | **174** | **31** | | **190** | **18** | |
| Median (g/L) [IQR] | 0.5 [0.4 - 0.6] | 0.5 [0.4 - 0.6] | 0.4 [0.2 - 0.6] | **0.006** | 0.5 [0.4 - 0.6] | 0.3 [0.2 - 0.4] | **<0.001** |
| Hypo-HDL-c n (%) | 42 (20.5) | 26 (14.9) | 16 (51.6) | **<0.001** | 1.1 [0.8 - 1.4] | 0.7 [0.4 - 1.0] | **<0.001** |
| **LDL-c (n)** | **208** | **177** | **31** | | **190** | **18** | |
| Median (g/L) [IQR] | 1.07 [0.7 - 1.3] | 1.1 [0.7 - 1.3] | 0.8 [0.4 - 1.2] | **0.001** | 1.1 [0.8 - 1.4] | 0.7 [0.4 - 1.0] | **<0.001** |
| Hyper-LDL-c n (%) | 27 (13) | 25 (14.1) | 2 (6.5) | 0.241 | 25 (14.1) | 2 (6.5) | 0.241 |
| **TG (n)** | **352** | **297** | **55** | | **320** | **32** | |
| Median (g/L) [IQR] | 0.9 [0.7 - 1.4] | 0.9 [0.7 - 1.3] | 1.4 [0.9 - 2] | **<0.001** | 1 [0.7 - 1.4] | 1.3 [0.9 - 2.3] | **<0.001** |
| Hypo-TG n (%) | 29 (8.2) | 25 (8.4) | 4 (7.3) | 0.776 | 27 (8.4) | 2 (6.3) | 0.667 |
| High n (%) | 58 (16.5) | 37 (12.5) | 21 (38.2) | **<0.001** | 43 (13.4) | 15 (46.9) | **<0.001** |
| **Natremia (n)** | **180** | **115** | **65** | | **140** | **40** | |
| Median (mmol/L) [IQR] | 140 [136.2 - 143.6] | 141 [137 - 144] | 139 [136 - 142.4] | 0.268 | 140 [137 - 143] | 140 [135 - 146] | 0.558 |
| Hyponatremia n (%) | 22 (12.2) | 13 (11.3) | 9 (13.8) | 0.617 | 13 (9.3) | 9 (22.5) | **0.024** |

**Continued**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Chloremia (n)** | **192** | **123** | **69** | | **151** | **41** | |
| Median (mmol/L) [IQR] | 102 [98 - 105] | 102 [99 - 105] | 100 [94 - 105] | **0.022** | 102 [98 - 105] | 101.5 [95 - 106] | 0.773 |
| Hypochloremia n (%) | 46 (24 %) | 22 (17.9) | 24 (34.8) | **0.008** | 33 (21.9) | 13 (31.7) | 0.189 |
| Hyperchloremia n (%) | 35 (18.2) | 23 (18.7) | 12 (17.4) | 0.821 | 25 (16.6) | 10 (24.4) | 0.249 |
| **Kalemia (n)** | **192** | **123** | **69** | | **151** | **41** | |
| Median (mmol/L) [IQR] | 4.4 [3.99 - 4.8] | 4.4 [4 - 4.8] | 4.3 [3.9 - 4.8] | 0.450 | 4.4 [3.9 - 4.7] | 4.6 [4 - 5.1] | 0.094 |
| Hypokalemia n (%) | 10 (5.2) | 4 (3.3) | 6 (8.7) | 0.103 | 8 (5.3) | 2 (4.9) | 0.914 |
| Hyperkalemia n (%) | 18 (9.4) | 12 (9.8) | 6 (8.7) | 0.808 | 11 (7.3) | 7 (17.1) | 0.056 |
| **Calcemia (n)** | **114** | **97** | **17** | | **104** | **10** | |
| Median (mg/L) [IQR] | 91 [86 - 98.5] | 93 [88 - 99] | 85 [83 - 90] | **0.001** | 92 [87 - 99] | 82.5 [81 - 89] | **<0.001** |
| Hypocalcemia n (%) | 47 (41.2) | 35 (36.1) | 12 (70.6) | **0.007** | 39 (37.5) | 8 (80) | **0.009** |
| Hypercalcemia n (%) | 4 (3.5) | 4 (4.1) | 0 (0) | 0.394 | 4 (3.8) | 0 (0) | 0.527 |
| **Magnesemia (n)** | **98** | **88** | **10** | | **96** | **02** | |
| Median (mg/L) [IQR] | 20 [18 - 22] | 19.5 [17 - 22] | 19.5 [18 - 22] | 0.953 | 19 [17 - 22] | 20.5 [20 - 21] | 0.659 |
| Hypomagnesemia n (%) | 15 (15.3) | 13 (14.8) | 8 (80) | 0.663 | 15 (15.6) | 0 (0) | 0.543 |
| Hypermagnesemia n (%) | 1 (1) | 1 (1.1) | 0 (0) | 0.734 | 1(1) | 0 (0) | 0.884 |
| **CRP (n)** | **134** | **92** | **42** | | **113** | **21** | |
| Median (mg/L) [IQR] | 12 [6 - 48] | 6 [6 - 25.5] | 36 [12 - 96] | **<0.001** | | | |
| High n (%) | 80 (59.7) | 44 (47.8) | 36 (85.7) | **<0.001** | 63 (55.8) | 17 (81.0) | **0.036** |
| **Blood glucose (n)** | **548** | **464** | **84** | | **493** | **55** | |
| Median (g/L) [IQR] | 0.9 [0.8 - 1.2] | 0.9 [0.8 - 1.2] | 1.2 [0.9 - 1.9] | **<0.001** | | | |
| Hypoglycemia n (%) | 5 (0.9) | 3 (0.3) | 2 (2.4) | 0.124 | 3 (0.6) | 2 (3.6) | **0.025** |
| Hyperglycemia n (%) | 183 (33.4) | 133 (28.7) | 50 (59.7) | **<0.001** | 149 (30.2) | 34 (61.8) | **<0.001** |
| **HbA1c (n)** | **71** | **52** | **19** | | **59** | **12** | |
| Median (mg/L) [IQR] | 7.3 [5.8 - 9.4] | 7.4 [5.8 - 9.6] | 6.4 [5.9 - 9.1] | 0.550 | 7.4 [6 - 9.6] | 6.3 [5.2 - 9.2] | 0.211 |
| High n (%) | 41 (57.7) | 32 (61.5) | 9 (47.4) | 0.284 | 36 (61.0) | 5 (41.7) | 0.216 |

n = size; (%) proportion estimated as a percentage. The percentages reported are percentages in columns; IQR = interquartile range; AST = aspartate aminotransferase; ALT = alanine aminotransferase; GGT = gamma-glutamyl transferase; ALP = alkaline phosphatase. TC = total cholesterol; HDL-c = high density lipoprotein cholesterol; LDL-c = low density lipoprotein cholesterol; TG = triglycerides; CRP = C-reactive protein; HbA1c = glycated hemoglobin.

serious forms with ($p < 0.001$). A significant increase in transaminases (ASAT, ALAT) and GGT was observed in patients with serious forms compared with non-serious forms, in the respective proportions of (55.4% vs. 19.9%: $p < 0.001$) (34.9% vs. 12.9%: $p < 0.001$) and (51% vs. 20.3%: $p < 0.001$). As for the lipid profile, a decrease in median HDL-c and LDL-c was observed in patients with serious forms with ($p = 0.006$) and ($p = 0.001$) respectively, while median triglycerides were elevated ($p < 0.001$). Compared to non-serious cases, the seriously ill had hypocholesterolemia (31.6% vs. 19.2%: $p = 0.035$), hypo-HDL-c (51.6% vs.

14.9%: $p < 0.001$), and hypertriglyceridemia (38.2% vs. 12.5%: $p < 0.001$). For the electrolyte panel, the medians for chloremia and calcemia were lower in patients with serious forms, with ($p = 0.022$) and ($p = 0.001$) respectively. Hypochloremia and hypocalcemia predominated in the serious forms, with (34.8% vs. 17.9%: $p = 0.008$) and (70.6% vs. 36.1%: $p = 0.007$) respectively. CRP and blood glucose levels were also elevated in the serious form, with (85.7% vs. 47.8%: $p < 0.001$) and (59.7% vs. 28.7%: $p < 0.001$) respectively.

### 3.4. Biochemical Parameters by Disease Outcome

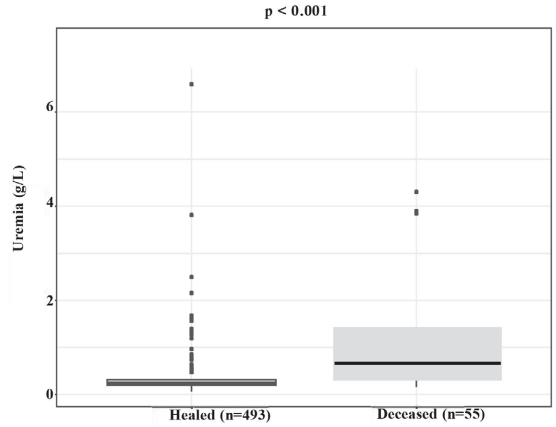
Figure 2 and Table 3 describe in detail the biochemical characteristics according to disease outcomes in the study population. Median blood urea and creatinine were higher in patients who died ($p < 0.001$). Also, deceased patients presented hyperuremia and high levels of serum creatinine with (56.5% vs. 8.4%: $p < 0.001$) (49.2% vs. 6.3%: $p < 0.001$). Medians of AST, ALT, and GGT were elevated in deceased patients ($p < 0.001$). Compared with those who had recovered, deceased patients had a significant increase in ASAT, ALAT and GGT, respectively (58.8% vs. 21.8%: $p < 0.001$) (33.3% vs. 14.4%: $p < 0.001$) and (56.3 vs. 21.4: $p < 0.001$). For lipid profile, medians for total cholesterol, HDL-c and LDL-c were reduced in deceased patients, with ($p = 0.016$), ($p < 0.001$) and ($p < 0.001$) respectively, while triglycerides were higher in deceased patients ($p < 0.001$). Compared with cured patients, hypocholesterolemia, hypo-HDL-c and hypertriglyceridemia were more frequent in deceased patients, respectively (46.9% vs. 18.7%: $p = 0.020$), (70.6% vs. 16%: $p < 0.001$) and (46.9% vs. 13.4%: $p < 0.001$). Median calcemia was lower in deceased patients ($p < 0.001$). Deceased patients had more hyponatremia and hypocalcemia than cured patients, with respectively (22.5% vs. 9.3%: $p = 0.024$) and (80% vs. 37.5%: $p = 0.009$). Also, elevated CRP and blood glucose levels were observed in patients who died with respectively (81% vs. 55.8%: $p = 0.036$) and (61.8% vs. 30.2%: $p < 0.001$), while hypoglycemia was noted in patients who did not survive (3.6% vs. 0.6%: $p < 0.025$).

## 4. Discussion

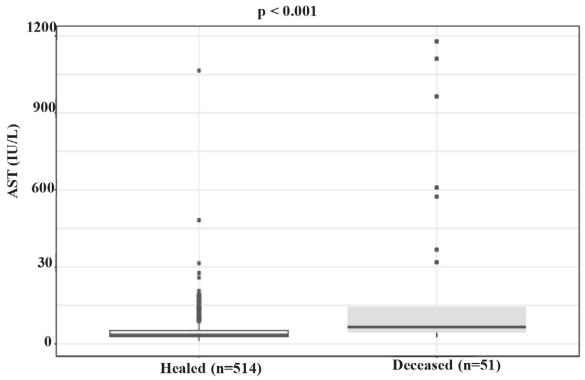
### 4.1. Summary of the Main Results

We included 782 patients, hospitalized at the Infectious Disease Management Centre in Lomé between March 19, 2020, and January 26, 2021. The median age was 41 years IQR [32 - 55]. More than half (63.5%) were male. Nearly half (48.5%) had comorbidities. More than half (64.8 %) had at least one symptom. One hundred and eleven patients (14.2%) had a serious form on admission. Eighty patients (10.2%) died during hospitalization.

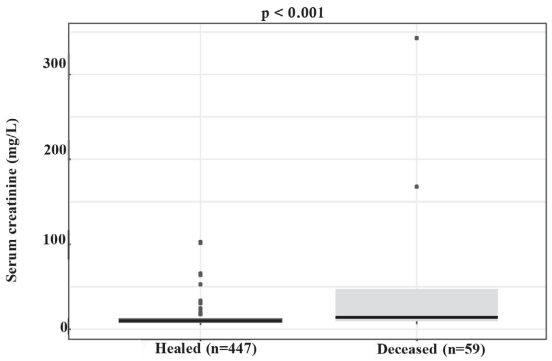
We observed biochemical abnormalities in varying proportions. These abnormalities concerned all the biochemical parameters studied. Compared to non-serious patients, seriously affected patients on admission had more frequently elevated urea, creatinine, transaminases, TG, GGT, CRP and blood glucose. There was also a decrease in TC, HDL-c, blood chloride, blood calcium. As for patients



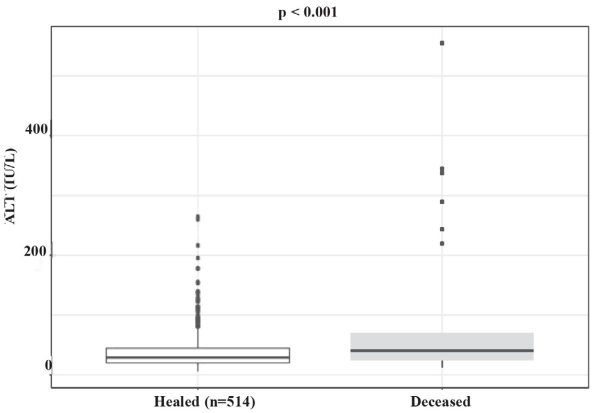

Distribution of uremia by disease outcome.



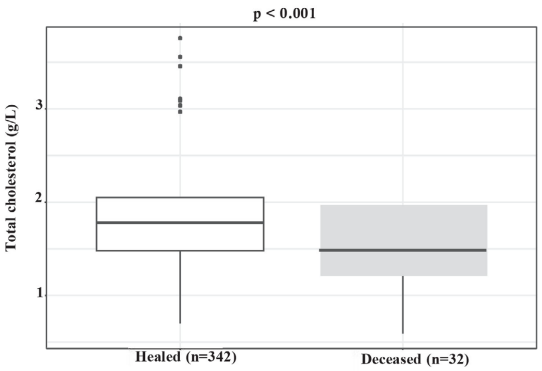

Distribution of AST by disease outcome.



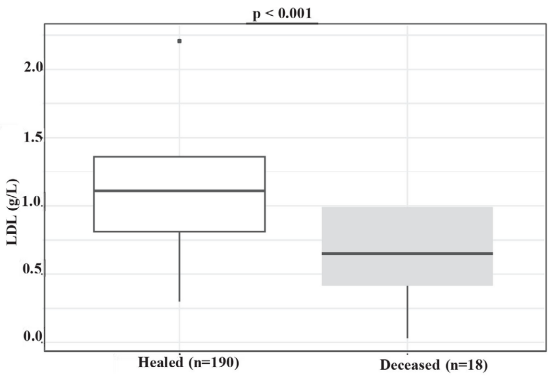

Distribution of serum creatinine by disease outcome.



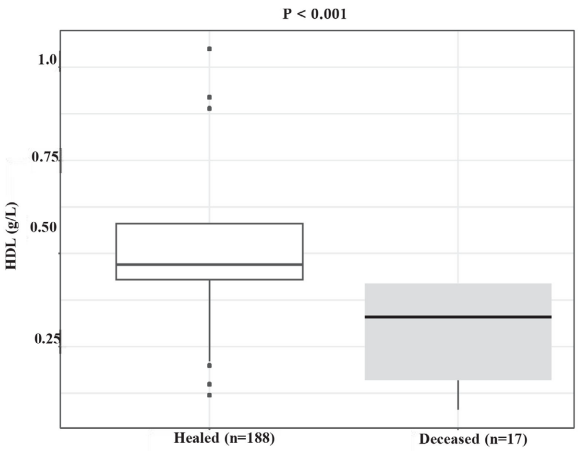

Distribution ALT by disease outcome.



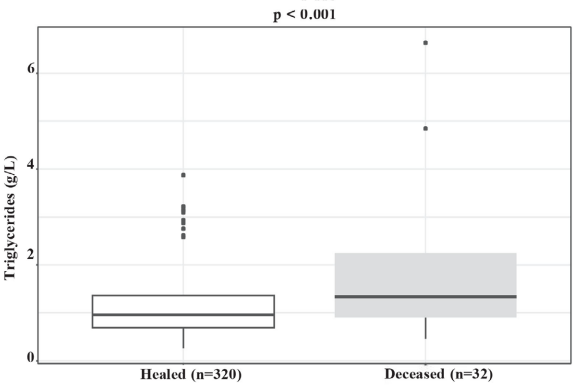

Distribution of total cholesterol by disease outcome.




Distribution of LDL by disease outcome.




Distribution of HDL by disease outcome




Distribution of triglycerides by disease outcome.

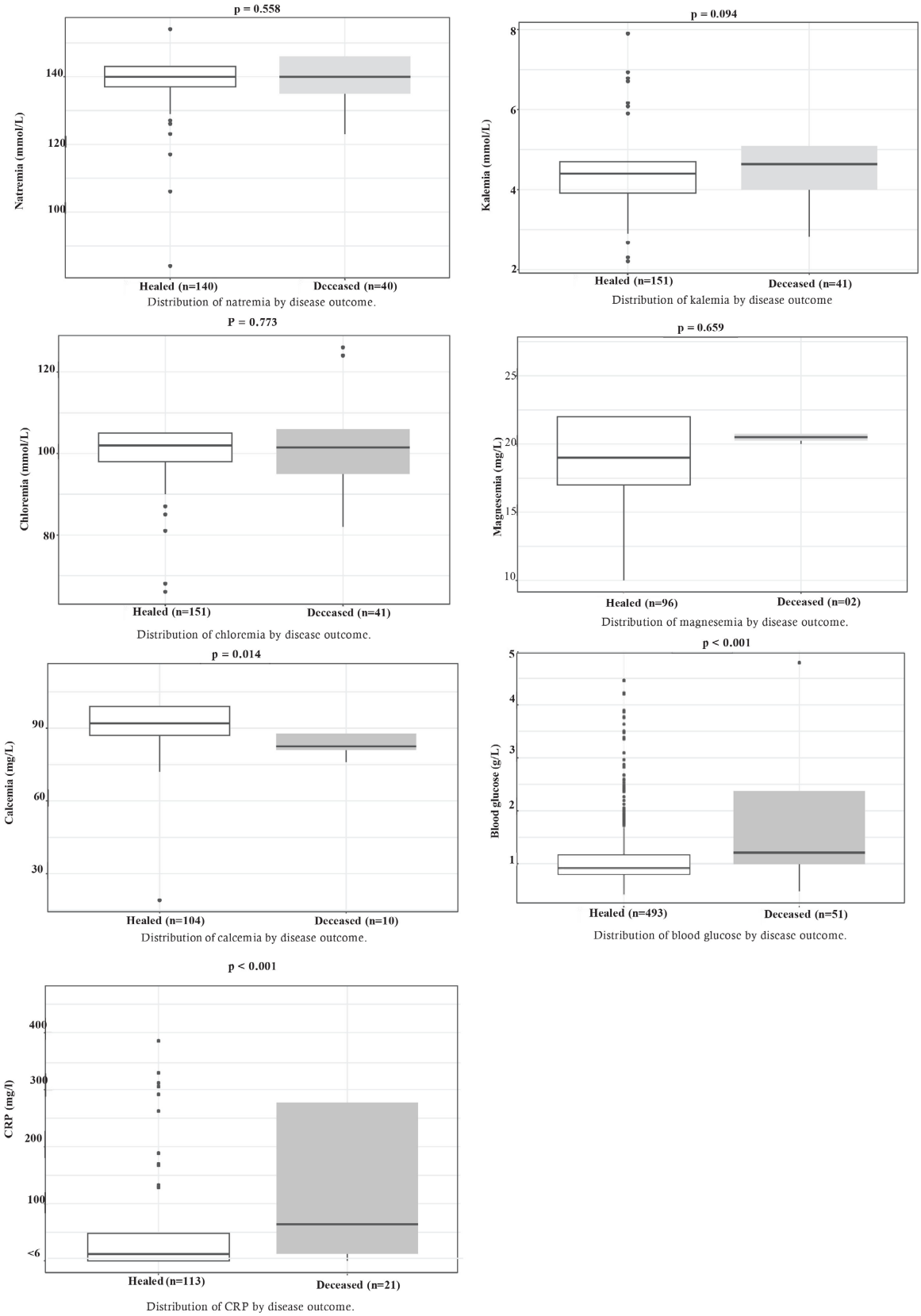



**Figure 2.** Biochemical characteristics according to the disease outcome of patients hospitalized for COVID-19 at the Infectious Disease Management Centre in Lomé in 2020.

who died during hospitalization, compared with cured patients, they showed an increase in urea, creatinine, ASAT, GGT, ALAT, CRP and blood glucose. They also had a decrease in TC, HDL-c, blood chloride, blood calcium and blood glucose.

The various biochemical abnormalities observed allowed us to identify potential organ damage such as impaired renal and hepatic function in patients infected with SARS-CoV-2. The increase in serum urea and creatinine, relatively frequent in this study, could suggest disturbances of renal function in these patients. Indeed, the kidney expresses 100 times more ACE2 than the lungs. Therefore, it could be an ideal target for viral replication during infection. However, the cytokine storm would be more responsible for these lesions than the cytopathic effects of the virus [15].

The elevation of liver enzymes (transaminases and GGT) as well as a decrease in certain substances synthesized by the liver, notably lipoproteins (HDL-c and LDL-c), suggest an impairment of liver function, which is all the more marked in the serious forms of the disease. In contrast to the kidney, ACE2 is weakly expressed in hepatocytes, except in the bile ducts. Thus, liver damage, especially in serious forms, seems to be related less to viral cytotoxicity than to sepsis. It has been reported that pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β modulate lipid metabolism by modifying liver function and decreasing cholesterol efflux and transport as observed in some human immunodeficiency virus (HIV) infected patients [16]. In addition, hemodynamic changes and/or drug interactions have also been reported [17].

Hypocalcemia, hyponatremia, and hypochloremia are the electrolyte panel abnormalities frequently observed in this study with a predominance in serious forms and deceased patients. Hypocalcemia could be related to hypoalbuminemia which has been frequently reported in COVID-19 [18]. Albumin is largely synthesized by the liver as are lipoproteins. The mechanisms of hyponatremia and hypochloremia are not yet understood. Several hypotheses have been put forward to explain hyponatremia, in particular a syndrome of inappropriate secretion of antidiuretic hormone (SIADH) [19].

CRP was elevated in more than half of patients in whom it was performed and was even more marked in the serious forms. This indicates the inflammatory nature of the disease. This inflammation would be responsible for the multisystemic lesions observed in several studies such as this one [6].

Hyperglycemia was common in serious forms of the disease. This suggests disturbances in glycemic regulation that are more marked in serious forms. However, it would have seemed useful to correlate the patients with hyperglycemia with the existence or not of diabetes mellitus.

Some of the results deserve attention, however. Indeed, renal, liver functions, and blood sugar tests were performed by about seven out of ten patients. This could be explained by the fact that these tests are part of the systematic pre-therapeutic check-up performed on all hospitalized patients. However, in about 20% of pa-

tients, at least one of these tests was missing. Some patients transferred from other health facilities had in their possession the results of paraclinical examinations including the biochemical tests of interest. In addition, the more expensive lipid profile and serum electrolyte tests were particularly prescribed for patients with serious forms of the disease. Only 17.3% (n = 134) had a CRP test, of which 37.8% (n = 42) had a serious form. Also, the deceased patients who had a CRP test on admission represented only 26.2% (n = 21). These low proportions of CRP testing could be explained by the fact that patients routinely have a blood count and sedimentation rate (ESR) on admission, the results of which are readily available compared to CRP. However, the CRP remains more sensitive and more specific in assessing inflammatory phenomena than the ESR [20].

It should be noted that in the absence of pre-disease findings and post-cure monitoring it would be presumptuous to associate these abnormalities exclusively with COVID-19. Also, a significant proportion of patients were being treated for comorbidities such as diabetes and hypertension. Self-medication, a common practice in Togo [21] especially in the context of COVID-19, could influence these results.

### 4.2. Limitations of the Study

The first limitation of this study is that several patients admitted to the centre were transferred from hospital facilities. Some had benefited from therapeutic protocols including rehydration, use of potentially hepatotoxic and nephrotoxic drugs. This may have altered some biochemical parameters before admission. Also, some patients were admitted with pre-admission tests, therefore they did not benefit from certain tests as the focus was on checking these results a few days after admission.

Another limitation concerns missing data, affecting the completeness of the data collection. Indeed, the biochemical results of some patients were not found.

The third limitation concerns the application of different thresholds for classifying certain biological parameters as abnormal. Several factors, including the different assay techniques used, could explain this.

In the literature, there is the heterogeneity of data regarding biochemical abnormalities in COVID-19 positive patients.

### 4.3. Biochemical Parameters

#### 4.3.1. Renal Function Tests

The frequencies of hyperuremia (13.7%) and high levels of serum creatinine (11.3%) are similar to those of Kefti *et al.* [22] in Algeria (2020) and Cheng *et al.* [23] in China (2020), who reported hyperuremia in 20% and 14.1% respectively, and high levels of serum creatinine in 10.7% and 15.5% respectively. The predominance of these abnormalities in patients with serious forms and those who died in this series was similar to the studies of Tao *et al.* [24] Chen *et al.* [25], Li *et al.* [26] in China (2020) who reported a statistically significant increase in serum urea and creatinine in patients with serious forms and in those who did not

survive. The results were in line with the data in the literature.

#### 4.3.2. Hepatic (Liver) Function Panel

The frequency of liver function abnormalities, particularly elevated transaminases (AST: 25.1%) (ALT: 16.1%), was similar to those of Guan *et al.* [27] in China who reported elevated AST and ALT in 22.2% and 21.3% respectively. However, the thresholds used by the Guan *et al.* study were lower (AST/ALT > 40 IU/L) than ours (AST > 57 IU/L; ALT > 60 IU/L). The predominance of liver abnormalities in patients with serious forms and those who died was similar to the studies of El Adaoui *et al.* in Morocco [28], Zhou *et al.* [5], Tao *et al.* [24] in China. Our results were consistent with the literature.

#### 4.3.3. Lipid Profile

Our results were similar to those of Wei *et al.* [29] in China who reported a decrease in total cholesterol, LDL-c, and HDL-c levels in patients with SARS-COV-2 infections. The decrease in LDL-c and/or HDL-c was more profound the more serious the disease. Peng *et al.* [30] in Wuhan, China, reported that total cholesterol, HDL-c, and LDL-c levels were significantly lower in COVID patients than in the reference population. Masana *et al.* reported that patients with serious disease had lower HDL-c cholesterol and higher triglyceride levels than those with less serious disease ($p < 0.001$) [31]. Our results were consistent with the literature.

#### 4.3.4. Electrolyte Panel

The electrolyte panel abnormalities observed in this study were similar to those of Teczan *et al.* in [32] Türkiye (2020) who reported hyponatremia in 35.8%, hypocalcemia in 9.5%, and no cases of hypernatremia or hypercalcemia. The predominance of hyponatremia in severe forms was reported by Lippi *et al.* in Italy [10]. Our results were consistent with the literature.

#### 4.3.5. Other Biochemical Parameters

**CRP**

The frequency of CRP elevation (59.7%) in this study was similar to those of Kefti *et al.* [22] in Algeria and Guan *et al.* [27] in China who reported an elevation in 53.5% and 60.7% respectively. This marked elevation in serious forms and deceased patients was similar to the study by El Adaoui *et al.* [28] in Morocco, Yu *et al.* [33], and Zheng *et al.* [34] in China. Our results were consistent with the literature.

**Blood Glucose**

Very few data have been reported on the admission blood glucose profile of COVID-19 patients. The frequency of hyperglycemia in the serious forms and among the deceased patients is similar to the study by Wu *et al.* who reported that patients with hyperglycemia on admission had a higher risk of developing the serious forms of the disease and even of having an unfavorable outcome [35]. Our results were consistent with the literature.

## 5. Conclusions

This study aimed to identify biochemical assays which could help to predict the prognosis of the disease, to ensure early and appropriate management and to promote optimal resource allocation in developing countries.

Several abnormalities were observed in varying proportions in all the biochemical parameters studied. These abnormalities were the most predominant in patients who presented serious forms of COVID-19 and those who died. However, a non-negligible proportion of patients admitted a priori with no clinical signs of severity had biochemical abnormalities, notably elevated CRP. This suggests that, like the renal and liver function tests, CRP should be systematically monitored on admission, to optimize management. Biochemical tests thus play an important role in the management of SARS-COV-2 infection, through early identification and monitoring of disease complications.

In addition, a prospective study over the entire hospitalization period would enable changes in biochemical parameters to be monitored and would help to better identify biochemical abnormalities predictive of severity or death.

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## Conflicts of Interest

The authors declare that they have no competing interests.

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