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Abstract: Long coronavirus disease 2019 (COVID-19) may occur after experiencing acute COVID-19 infection with many effects on patients´ kidneys. This study aimed to evaluate the clinical and laboratory aspects of kidney function in individuals with long COVID in the Amazon region of Brazil. This cross-sectional study selected participants from a 'long COVID' clinical care program. Clinical data and baseline demographics were obtained, and blood samples were collected to quantify kidney-related markers. Patients were evaluated after COVID-19 infection at several time points as follows: 0–3 months, 3–6 months, 6–12 months, and 12 months. Altogether, 246 patients were selected for this study, and only 79 were hospitalised (9 in the intensive care unit because of COVID-19. Of the patients, 38 were diagnosed with hypertension after COVID-19, 52 cases had to have their medications adjusted, and 83 patients presented with kidney function decline. The older age group and individuals with glycaemic alterations presented with a higher risk of developing kidney dysfunction. Therefore, health professionals should monitor long-term kidney markers in long COVID.

Keywords: SARS-CoV-2; COVID-19; chronic kidney disease; long COVID; Acute Kidney Injury; Amazon region; Kidney function tests

1. Introduction

In 2019, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was identified as the causative agent of coronavirus disease 2019 (COVID-19) [1,2]. In a short time, the disease spread to other countries, and in March 2020, the World Health Organization (WHO) officially declared it a pandemic [3]. COVID-19 has a major impact on the respiratory system, with symptoms of flu-like illness that can progress to progressive respiratory infection, acute respiratory distress syndrome, and death. However, the disease can compromise other organs and systems such as the kidneys, leading to different clinical behaviours, laboratory findings, and sequelae [4,5].

Kidney involvement in the acute phase is present in 0.5%–15% of this population, and 20%–50% of patients are admitted to the intensive care unit (ICU), with approximately 6% requiring renal substitutive therapy, such as haemodialysis [6,7,8,9]. The most common renal alterations were increased urea and creatinine levels, which tended to reflect a reduction in the glomerular filtration rate (GFR); alterations in the urinary sediment, such as proteinuria and haematuria; and in more severe cases, a reduction in the urinary volume, or even anuria. Acute kidney injury (AKI) is associated with poor clinical outcome and mortality [7,10].

However, part of the population affected by COVID-19 manifested persistent clinical symptoms several systems weeks after the diagnosis of the disease. According to the definition by the National Institute for Health and Care of Excellence, the persistence or appearance of symptoms 4 weeks after the diagnosis of the disease, which cannot be explained by other causes, is called post-COVID syndrome or long COVID [11,12,13,14,15]. Long COVID is considered a multisystemic condition, with >200 symptoms described, compromising the health and quality of life of patients [11,12].

Kidney injury occurs through multiple mechanisms that are influenced by individual factors such as age and comorbidities, and by factors such as direct viral injury, inflammatory state, hypercoagulability, states of shock and hypovolaemia, endothelial micro- and macrovascular injury, nephrotoxic drugs, and rhabdomyolysis. [16]. The importance of compromising the renin–angiotensin–aldosterone system has also been highlighted considering that angiotensin-converting enzyme 2 (ACE-2) is the main receptor for SARS-CoV-2 in human cells. Hence, a trend towards reduced availability of functioning ACE-2, with a consequent reduction in the production of angiotensin 1–7 and a decrease in its anti-inflammatory, vasodilatory, and anti-thrombotic effects has been observed [17,18].

Renal alterations resulting from these processes can cause sequelae of varying degrees in long COVID, causing lesions that can last for long periods and are potentially irreversible [19,20]. In the face of long COVID-19 conditions, surviving patients, after 30 days of infection, exhibited an increased risk of acute renal failure, a decline in the estimated GFR, and other kidney-related adverse events. The risk and kidney damage increase with the severity of acute infections [21]. The possibility of developing metabolic syndromes with the onset of diabetes mellitus (DM) and systemic arterial hypertension (SAH) has also been described, which may further contribute to the development of kidney disease considering the strong association of chronic kidney disease (CKD) with these conditions [22].

By the end of 2022, >861,000 cases were recorded in Pará. According to estimates by the WHO, approximately 10%–20% of the infected population develops some type of sequela. Therefore, the number of affected people who may need care for long COVID is worrying and further overloads the weakened public health system in the Amazon region [23]. The lack of data and dissemination of the possibility of renal sequelae are worrying factors locally and worldwide [24]. According to studies by the National Kidney Foundation-Harris Poll, only 17% of Americans are aware of the risks of kidney damage; therefore, little information is available on renal sequelae in patients with long COVID, thus delaying proper diagnosis and treatment [20].

Therefore, this study aimed to evaluate renal function, alterations, and sequelae evidenced by clinical and laboratory findings. It also aimed to establish the incidence, clinical and laboratory factors, and types of kidney injuries in the affected population, allowing the identification of risk and predictive factors of chronic kidney injury and guiding public policy measures to avoid greater social and economic impacts on the health scenario in the Amazon region and Brazil.

2. Materials and Methods

This prospective, observational, cross-sectional, descriptive, and analytical study was conducted in accordance with the principles of the Declaration of Helsinki. This study was approved by the Research Ethics Committee of the State University of Pará (UEPA) (Opinion No. 4.252.664) and was conducted between 2020 and 2022, involving clinical and laboratory evaluation and investigation of renal function, as well as analysis of inflammatory markers, in patients with long COVID. All the participants signed an informed consent form (ICF).

The study was conducted on adult individuals (>18 years old) with a confirmed diagnosis of SARS-CoV-2 through a reverse transcription polymerase chain reaction (PCR) test of a throat swab and a history of a clinical picture compatible with COVID-19 not attributable to any other cause. Participants were selected according to the inclusion criteria proposed in the research (Figure 1) and the order of voluntary registration in a UEPA program aimed at caring for patients with long COVID (Post-COVID Program). The participants only started the follow-up protocol after prior telephone

contact 30 days after the first symptoms of infection. Patients with previous reports of CKD and pregnant women were excluded.
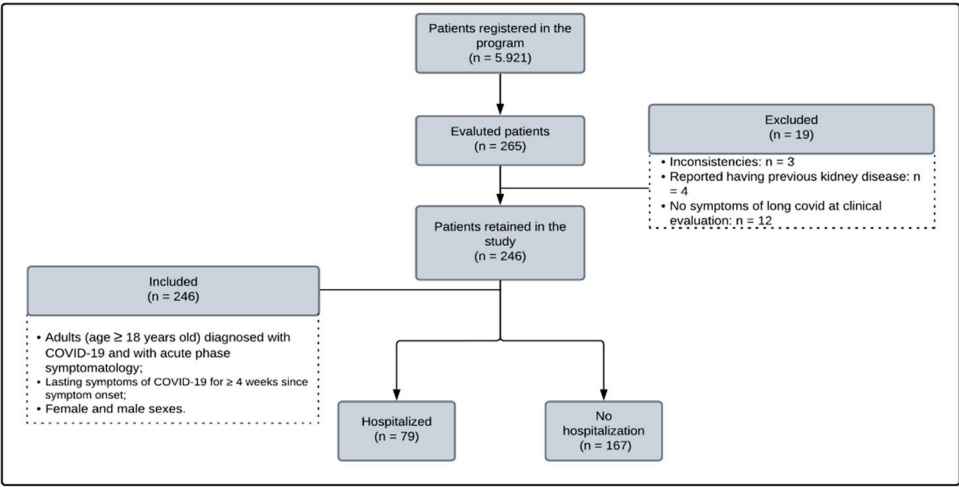

Figure 1. Flowchart of patient recruitment in the study. Created with Lucidhart.com (www.lucidchart.com. accessed June 4, 2023).

Information was extracted from interviews with each individual by completing a specific protocol form (Appendix A) from which general information, clinical data, anthropometric data, and information including past illnesses, smoking, age, need for hospitalisation, and use of medications such as angiotensin-converting enzyme inhibitors (ACE) or angiotensin receptor blockers (ARB) were collected. The collected laboratory data broadly evaluated the patient's health with haematimetric, coagulation, metabolic, hepatic, immunological profiles, inflammatory markers, and above all, urea and creatinine levels for better evaluation of renal function, as well as urine tests with active urinary sediment. The Clinical Analysis Laboratory of the State University of Pará was selected for performing the necessary clinical analyses.

Blood samples were collected from patients who had fasted for at least 8 h. This collection consisted of puncturing the peripheral vessel with a needle and collecting samples in 3-ml Vacuette tubes (Greiner Bio-One, Kremsmunster, Austria). Three blood tubes were collected from each patient as follows: (a) tubes containing the anticoagulant ethylenediaminetetraacetic acid for analysis of whole blood, (b) tubes containing separator gel and clot activator for serum analysis, and (c) tubes with citrate for performing coagulation tests. The respective reagent manufacturers adopted standard reference ranges for the tests mentioned above (Appendix B, Table A2). Urinalysis was performed using an isolated sample of morning urine and was analysed using reagent strips and microscopy.

GFR was measured to evaluate renal function. For its realisation, indirect markers can be used, such as the determination of creatinine and cystatin C in the blood or the performance of the glomerular filtration rate with indicators such as inulin or other substances [25]. In this study, serum creatinine dosage was the option for assessing GFR because it was performed in the UEPA reference laboratory and because of its low cost, although it has a low sensitivity for detecting less advanced degrees of renal function loss. To minimise possible difficulties, the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, which uses sex, age, and serum creatinine level as the main variables, was used.

The formula proposed by Levey et al. [26] is as follows:

CKD-EPI creatinine-based:

$$141 \times \min\left(\frac{\text{serum creatinine}}{k}, 1\right)^{a} \times \max\left(\frac{\text{serum creatinine}}{k}, 1\right)^{-1,209}$$
$$\times 0,993^{\text{age}}[\times 1,018 \text{ (if woman)}][\times 1,159 \text{ (if black)}] \ ,$$

where k is 0.7 for women and 0.9 for men, α is −0.329 for women and −0.411 for men, min is minimum serum creatinine/k or 1, and max is maximum serum creatinine/k or 1.

After data collection, an electronic spreadsheet was created to store the data using Microsoft Excel 360. For the descriptive representation of the data, tables and graphs were created using the Microsoft Word 360® Software, representing the means, standard deviation, median, interquartile ranges, and minimum and maximum of numerical variables. Categorical variables are presented as absolute and relative percentage frequencies. Data analysis was performed using the Jamovi 2.3.24 program. For all analyses, a significance level of 5% was used (p-value<0.05). To compare quantitative variables such as age (in years), erythrocyte sedimentation rate, ferritin, albumin, creatine phosphokinase, lactic dehydrogenase, pyocytes, haemoglobin, red blood cells, glucose, glycated haemoglobin, and bodymass index (BMI), the mean and median values were evaluated between groups of patients with and without renal alterations. For this analysis, the t-student test was used when the data exhibited a normal distribution, while the Mann-Whitney test was employed when the assumptions of normality were violated according to the Shapiro-Wilk test. The Yates-corrected chi-square test was used to compare the proportion of female sex, age (>60 years), DM, SAH, use of ACEI/ARB variables, hospitalisation, ICU or ward admission, long COVID symptoms, kidneys, protein C reagent positivity, time of long COVID evaluation, and proteinuria due to the presence or absence of renal alteration.

3. Results

Of the 246 selected patients, 155 (64.01%) were female, and 72 (29.63%) were aged >60 years. DM and SAH were the most common comorbidities, reported by 32 (13.01%) and 88 (35.77%) patients, respectively. Only 75 patients previously reported using ACE inhibitors or ARBs, and the mean BMI was 29.24 kg/m^2, indicating the prevalence of overweight. Only 79 patients required hospitalisation for the treatment of COVID-19, with only nine in the ICU.

Table 1. Clinical and laboratory profile of the studied patients.

Variable	n = 246	%
Female	155	64.01%
Male	91	35.99%
>60 years old	72	29.63%
>18 and <60 years old	174	70.37%
Diabetes mellitus	32	13.01%
SAH	88	35.77%
CVD	19	7.72%
Previous lung disease	16	6.50%
Non-smoker	182	73.98%
Current smoker	9	3.66%
Former smoker	55	22.36%
ACE inhibitors/ARB use	75	30.49%
Hospitalisation	79	32.11%
ICU	9	3.66%
Otorhinolaryngological	34	13.82%
Neurological	84	34.15%
Dermatological	12	4.88%
Psychiatric	11	4.47%
Cardiovascular	55	22.36%
Renal	25	10.16%
Post SAH	90	36.59%
Muscular	61	24.80%
Respiratory	110	44.72%
Osteoarticular	20	8.13%
Gastrointestinal	16	6.50%

SAH, systemic arterial hypertension; CVD, cardiovascular disease; ACE, angiotensin-converting enzyme; ARB: angiotensin 2 receptor blockers; ICU, intensive care unit; ENT, otorhinolaryngological.

Neurological (n = 84), muscular (n = 61), and respiratory (n = 110) symptoms were the most frequently reported. Renal symptoms were observed in 25 patients. Findings related to SAH stand out, in which 38 new cases emerged and 52 patients with previous hypertension demonstrated worsening blood pressure levels, requiring an increase in the dose of antihypertensive drugs.

Study participants were evaluated at various time points after COVID-19 at 0–3 months, 3–6 months, 6–12 months, and >12 months (Figure 2). Moreover, 83 (33.74%) patients presented changes in renal function measured through GFR using the CKD-EPI equation, suggesting a slightly lower incidence at 0–3 months.
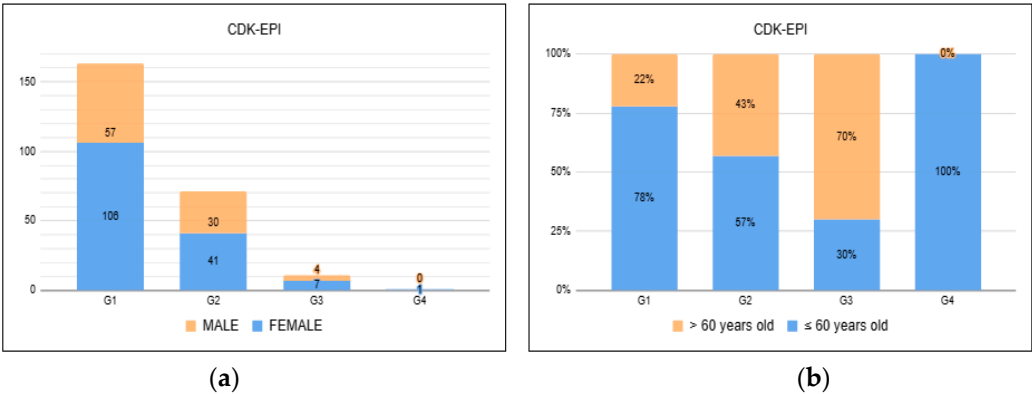

(a)   (b)

Figure 2. Identification in absolute numbers and percentage of glomerular filtration rate (GFR) stratification separated by sex (a) and age (b) at onset according to the Chronic Kidney Disease Epidemiology Collaboration equation (CKD-EPI). G1: GFR ≥ 90 ml/min; G2 GFR ≥60 and ≤89 ml/min; G3 ≥30 and ≤59 ml/min; G4 ≥15 and ≤29 ml/min; and G5 <15 ml/min.

Regarding metabolic issues, 103 (41.87%) patients had high fasting glycaemia, and of the 188 participants in whom glycated haemoglobin markers were analysed, 122 (64.89%) had alterations, of which 65 (34, 57%) were within the criteria for pre-diabetes and 57 (30.32%) were suggestive of DM according to the WHO criteria [27].

Table 2. Qualitative variables in patients evaluated with long COVID.

Variable	n = 246	%
HB>12	210	85.71%
GLYC>100	103	41.87%
Positive CRP	35	14.23%
COLT>200	120	48.78%
HDL>50	89	36.18%
TG>150	104	42.28%
PTNS URINE		
0	233	94.72%
1	5	2.03%
2	6	2.44%
3	2	0.81%
HB URINE		
0	229	93.09%
1	10	4.07%
2	4	1.63%
3	3	1.22%
GLYC URINE		

0	228	92.68%
1	2	0.81%
2	1	0.41%
3	12	4.88%
4	3	1.22%
GFR <90 ml/min	83	33.74%
Long COVID time (months)		
0 to 3m	59	23.98%
>3 and <6m	59	23.98%
>6 and <12m	60	24.39%
>12m	68	27.64%
Glycated Hb		
Normal	66	35.11%
Prediabetes	65	34.57%
Diabetes	57	30.32%

HB, haemoglobin; GLYC, glycaemia; CRP, C-Reactive Protein; COLT, total cholesterol; HDL, mean high density lipoproteins; TG, triglycerides; PTNS, percutaneous tibial nerve stimulation; GFR, glomerular filtration rate.

In the analysis of clinical and laboratory data related to renal function, 35 (14.23%) patients had a positive PCR test, and insignificant alterations in the urinary sediment occurred in <7% of the cases. A trend towards low albumin levels was also observed in the group that presented with alterations in GFR, as well as higher levels of LDH and glycated haemoglobin.

Table 3. Clinical data correlated with renal function.

Variable	No kidney Alteration, n (%)	With Kidney Alteration, n (%)	p-Value
Female sex	106 (65%)	49 (59%)	0.43*
Age (>60 years)	35 (21.60%)	37 (45.68%)	0.00019*
DM	21 (12.8%)	11 (13.2%)	0.99*
SAH	56 (34.4%)	34 (40.9%)	0.38*
CVD	12 (7.3%)	7 (8.4%)	0.96*
ACE inhibitors/ARB use	44 (26.9%)	31 (37.3%)	0.12*
Hospitalisation	48 (29.4%)	31 (37.3%)	0.26*
ICU	5 (3.1%)	4 (4.8%)	0.49**
Nursery	42 (25.7%)	27 (32.5%)	0.33*
Long COVID symptoms	163 (100%)	83 (100%)	–
Renal	14 (8.6%)	11 (13.2%)	0.35*
Positive long COVID	21 (12.8%)	14 (16.8%)	0.42*
Time long COVID			0.20**
0–3m	45 (27.6%)	14 (16.8%)	
>3 m and <6 m	37 (22.7%)	23 (27.7%)	
>6 m and <12 m	46 (28.2%)	22 (26.5%)	
>12 m	35 (21.5%)	24 28.9%)	
PTNS			0.55**
0	154 (94.5%)	79 (95.2%)	
1	4 (2.4%)	1 (1.2%)	
2	3 (1.8%)	3 (3.6%)	
3	2 (1.2%)	0 (0%)	

*Chi-square test. **Fisher's exact test. DM, diabetes mellitus; SAH, systemic arterial hypertension; CVD, cardiovascular disease; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; ICU, intensive care unit; COVID, coronavirus disease 2019; PTNS, percutaneous tibial nerve stimulation.

Table 4. Laboratory data correlated with renal function.

Variable	No Kidney Alteration		With Kidney Alteration		p-Value
	Median	AIQ	Median	AIQ	
Age (years)[a]	48.6	12,9	57.3	11.7	0.0000**
ESR	31	(32.500)	34	(32.000)	0.749*
ferritin	134	(192.250)	130	(124.000)	0.594*
albumin	4.2	(0,7)	3.9	(0,5)	0.053*
CPK	81.5	(63.500)	88.5	(84.000)	0.247*
DHL	309	(90.000)	321.5	(115.250)	0.036*
pYoc	3	92.000)	3	(2.500)	0.589*
Hb[a]	13.2	(1.23)	13.3	(1,49)	0.552**
hcs	0	(2.000)	0	(2.500)	0.762*
glyc	0	(0)	0	(0)	0.995*
HbGlyc	5.9	(1.300)	6.2	(1.000)	0.049*
BMI	28	(7.500)	28	(6.000)	0.149*

[a]Mean (standard deviation). *Mann–Whitney. **Student's t-test. AIQ, amplitude interquartile; ESR, erythrocyte sedimentation rate; CPK, creatine phosphokinase; DHL, lactic dehydrogenase; PYoc, pyoctes; Hb, haemoglobin; hcs, human chorionic somatomammotropin; glyc, glycaemia; HbGly, glycated haemoglobin; BMI, body mass index.

4. Discussion

SARS-CoV-2 infection is now recognised not only as a respiratory tract condition but also as a multiorgan syndrome that commonly leads to AKI [28]. The persistence of symptoms after the acute phase of the disease can lead to potential sequelae in different organs and systems, which refers to the so-called long COVID [29,12]. Data on renal dysfunction in patients with long COVID remain scarce and require further study [24].

Our study included 246 patients (155 women, 174 aged 18–59 years). Of the patients, 79 required hospitalization during the acute phase of COVID-19; however, they demonstrated no kidney injury outcomes. The most frequently reported symptoms in long COVID include neurological, muscular, and respiratory symptoms. Furthermore, regarding the presence of comorbidities, we identified 32 patients with a previous diagnosis of DM and 88 patients with SAH; 100 patients had a BMI of >30 kg/m². Alterations in renal function through GFR were identified in 83 patients, with a statistically significant relationship observed in patients aged >60 years. However, the findings related to changes in urinary sediment proved to be of little relevance in the sample, where proteinuria, haematuria, and glycosuria were present in <7% of the patients and indicated no relationship with GFR changes.

Evidence in the literature demonstrates that kidney injury occurs in patients with >12 months of long COVID [11,30]. This perspective corroborates the findings of this study, in which kidney injury was present at all evaluated periods. These data demonstrate the potential of renal sequelae developed by patients with long COVID, known to have a multifactorial cause related to the maintenance of inflammatory factors, intrinsic tubular damage, and incomplete resolution of the condition, causing an impact on morbidity and the health system, considering the increase in number of patients with chronic non-communicable diseases such as CKD [31,32,33].

A statistically relevant relationship between changes in renal function and patients aged >60 years was demonstrated using GFR, which is in line with studies on long COVID [34]. Of the 83 patients who had impaired renal function, 71 had a GFR between <90 ml/min and ≥60 ml/min. What the literature brings so far regarding long COVID is the perpetuation of changes compatible with a decline in GFR, with the most frequent range being G2 [30] consistent with our findings.

In the literature, DM and SAH were also the most reported comorbidities in individuals who developed long COVID, and the development of these conditions was also identified in some previously healthy patients with post-COVID syndrome [13,32,35,36]. This perspective is worrisome because hyperglycaemia is known to cause kidney damage and complications, with genetic,

haemodynamic, and metabolic factors related to its pathophysiology [37,38]. Regarding BMI, the association between obesity and severe cases of COVID-19 in the acute phase is well known; therefore, obesity has a greater potential for sequelae and the development of long COVID-19 [39].

Laboratory data revealed a significant correlation between LDH levels and kidney damage (p = 0.036) and glycated haemoglobin levels (p = 0.049). A trend towards an association between albumin levels (p = 0.053) was also observed. These aspects suggest possible associations already identified in other studies, indicating that LDH levels are closely related to inflammation and hypercoagulability states [32,40]. Evidence points to a tendency towards lower levels of serum albumin in long COVID owing to cellular hypercatabolism, which leads to a reduction in circulating albumin levels [40]. The higher levels of glycated haemoglobin in this study are consistent with data in the literature, as several studies have demonstrated an increased incidence of DM in the long COVID phase [41,42,43]. This is concerning because of the high incidence of this disease worldwide and its impact on healthcare systems [44,45].
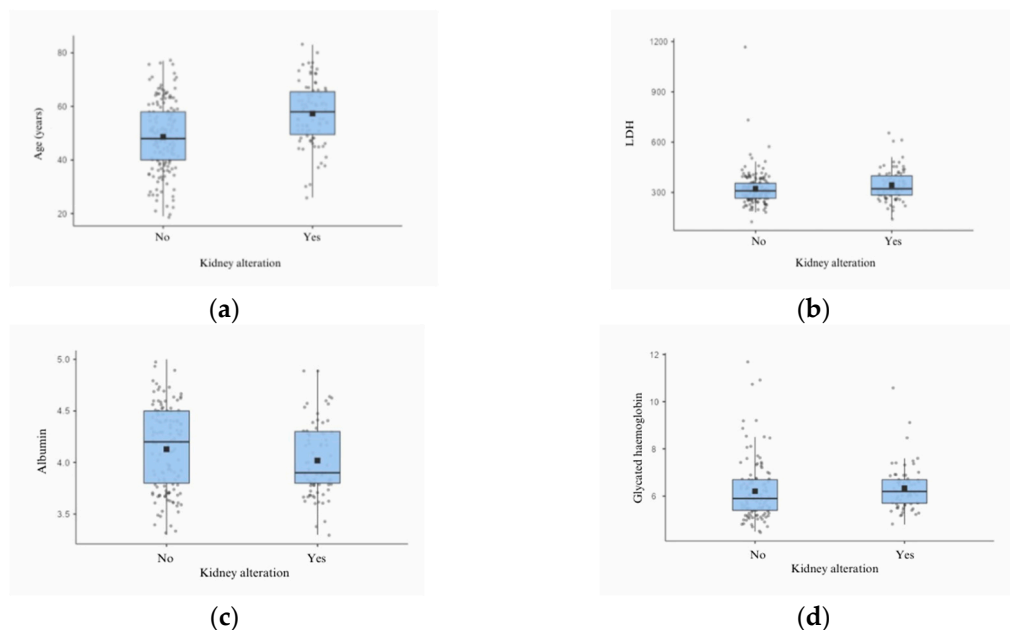

Figure 3. Boxplot presenting the list of variables that correlated with changes in glomerular filtration rate.

As for the use of medications, 75 patients used medications such as ACE inhibitors and ARBs. The possibility of worse clinical and renal outcomes in patients using these medications has been described in the literature because of the suspicion of positive regulation of ACE2 and the consequent increase in viral load [31,46]. This would also influence the production of angiotensin 1-7, which has antagonistic actions on angiotensin II, such as vasodilation and antiproliferative actions [47]. However, in our study, this association was not observed, which is in line with recent findings in the literature [48,49,50].

This study has some limitations. The availability of information such as the previous renal function of these patients and details regarding hospitalisation, considering the need for ventilatory support, drugs used, and even the development of AKI, makes it difficult to understand the process of renal illness. The follow-up of these patients for a longer period could provide greater support for assessing the evolution of renal function in patients with long COVID. However, our data are greatly valuable to the medical and scientific community, as they allowed us to identify which patients with long COVID are at risk of developing kidney damage for a period of >12 months after the acute phase of the disease. The elderly population and those with glycaemic alterations have been identified as being at a greater risk of developing such alterations, and monitoring of renal function should be more comprehensive in such individuals. Therefore, further studies are warranted.

5. Conclusions

Kidney injury in patients with long COVID in this study was observed in one-third of the population studied, with no significant differences between groups with comorbidities, use of medications such as ACE inhibitors and ARBs, need for hospitalisation, and even evaluation time after the acute phase, indicating that kidney damage can persist for >12 months after contact with SARS-CoV-2. Moreover, the elderly population and those who developed glycaemic alterations were at a greater risk of renal dysfunction with long COVID. Therefore, we must be attentive to laboratory findings related to the outcomes of renal injury, such as LDH, glycated haemoglobin, and serum albumin levels.

The lack of similar investigations in the Amazon region emphasises the importance of this study. These findings reinforce the need to establish programs that include renal evaluation in the follow-up of patients with long COVID, in a more comprehensive way, since even young people without comorbidities present alterations suggestive of renal disease. Thus, the implementation of therapeutic measures and health strategies aimed at reducing the medium- and long-term impacts of the COVID-19 pandemic on the health of the population in the Amazon region and worldwide may be related to better prognoses.

Appendix A

POST-COVID NEPHROLOGICAL PROTOCOL

1. AGE: RACE:
2. GENDER   ( ) FEM   ( ) MAL
3. CONFIRMATORY EXAMINATION: DATE:
4. COMORBIDITIES:

(   )DIABETES MELLITUS

(   )SYSTEMIC ARTERIAL HYPERTENSION

(   )CORONARY ARTERY DISEASE

(   )OVERWHEIGHT/OBESITY   BMI: _____

(   ) COPD /ASTHMA/PNEUMOPATHY

(   )VASCULOPATHY

(   ) SMOKING

(   ) OTHERS : _____

5.   USED ACEI OR ARB DURING ILLNESS: ( ) YES   ( ) NO
6.   NEED FOR HOSPITALIZATION   ( ) YES   ( ) NO
7.   NEED FOR HEMODIALYSIS DURING HOSPITALIZATION   ( ) YES   ( ) NO
7.   INFORMATIONS ABOUT MEDICAL TREATMENT DURING COVID-19
_____
8.   CURRENT SYMPTOMS

( ) SAH / ARTERIAL PRESSURE: _____

( ) HEMATURIA

( ) EDEMA

( ) PROTEINURIA

( ) OTHERS _____

9.   CURRENT TREATMENTS: _____
10. NEED FOR FOLLOW-UP WITH NEPHROLOGIST   ( ) YES   ( ) NO
11. LABORATORY

Table A1. Laboratory parameters

	DATE
HEMOGRAM	
TGP/TG0	
ALKALINE PHOSPHATASE	
GAMMA GT	
UREA	
CREATININE	
ESR	
RCP	
ColT/HDL/LDL	
TRIGLYCERIDES	
GLUCOSE	
FERRITIN	
CPK	
SODIUM	
POTASSIUM	

LDH
aPTT/PT
ALBUMIN
URINE TEST
Protein URINE
RedBloodCels URINE
Gluc URINE
GLYCATEDHEMOG

TGO, mean oxoloacetic transaminase; TGP, mean pyruvic transaminase; ESR, erythrocyte sedimentation rate; RCP, mean C-reactive protein; ColT, mean total cholesterol; HDL, mean high density lipoproteins; LDL, mean low density lipoproteins; CPK, mean creatine phosphokinase; LDH, mean lactate dehydrogenase; APTT, activated partial thromboplastin time; PT, prothrombin time.

OBSERVATIONS:
REFERRAL:

Appendix B

Table A2. Reference range (adult men and women) of laboratory parameters.

Exam	Min	Max	Measure
Haemoglobin	13,5	18,0	g/dl
Haematocrit	40,0	54,0	%
Leukocytes	5,000	10,000	mm
Erythrocyte Sedimentation rate (ESR)	0	20	mm
Prothrombin Time (PT)	70	180	%
Activated Partial Thromboplastin (APTT)	24	39	seconds
Glycated Haemoglobin	4,8	5,9	%
C-Reactive Protein *	Negative	Positive	
Glucose	65	99	mg/dl
Total Cholesterol	-	200	mg/dl
HDL	60	-	mg/dl
LDL	-	100	mg/dl
Triglycerides	-	150	mg/dl
TGO	-	38	U/L
TGP	-	41	U/L
Alkaline Phosphatase	65	300	U/L
Gamma GT	11	50	U/L
Urea	15	45	mg/dl
Creatinine	0,4	1,3	mg/dl
Calcium	8,5	10,5	mg/dl
Iron	65	175	ug/dL
Ferritin	50	300	mg
Potassium	3,5	5,5	mEq/L
Sodium	134	149	mEq/L
Creatine phosphokinase (CPK)	-	195	U/L
Creatine phosphokinase MB	-	25	U/L
Lactic dehydrogenase(LDH)	230	460	U/L
Albumin	3,5	4,8	g/dL
Leukocytes urine	-	4	field
Red blood cells urine	-	2	field
Proteins urine	-	-	field

Min, minimum; Max, maximum; HDL, mean high density lipoproteins; LDL, mean low density lipoproteins; TGO, mean oxaloacetic transaminase; TGP, mean pyruvic transaminase. *Qualitative sample; negative referene value *Laboratory test values are influenced by physiological states and medication use, including diet or fasting time.

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