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Long-Term Effects of SARS-CoV-2 Exposure on Kidney and Thyroid Health in a Nigerian Urban Population

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Abstract: COVID-19, caused by the newly discovered coronavirus SARS-CoV-2, is a contagious disease that has affected millions worldwide. It often begins with symptoms related to the respiratory system, such as pneumonia that affects the small air sacs and tissues in the lungs. But the virus doesn't stop there; it can also impact other organs, including the kidneys and thyroid glands. These organs have a special receptor called ACE2, which the virus uses to enter cells, making them vulnerable. Currently, there's limited information on how the pandemic might change kidney and thyroid health in the long run. This study aims to shed light on how the COVID-19 pandemic has affected the thyroid and kidney health of residents in Oyo State, Nigeria. We conducted a cross-sectional study focusing on individuals in Ibadan, exploring their thyroid and renal health after exposure to SARS-CoV-2. Venous blood samples were randomly collected from 165 participants in different areas of Ibadan. We gathered socio-demographic information and divided the participants into two groups: 85 individuals who were unexposed, testing negative for both SARS-CoV-2 RNA and antibodies, and 80 individuals who were exposed, testing positive for both. The renal markers (cystatin C, urea, creatinine, electrolytes) and thyroid function markers (FT3, FT4, TSH) of both groups were analyzed using an independent sample t-test. There was no significant differences ($p > 0.05$) found in the mean ± SD values of the identified markers between individuals exposed to SARS-CoV-2 and those unexposed. The findings suggest that individuals infected with SARS-CoV-2 in this study experienced full recovery of renal and thyroid functions during the pandemic.

Keywords*: COVID-19, Renal Function Markers, SARS-CoV-2, Thyroid Function Markers.*

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I. INTRODUCTION

The Coronavirus Disease 2019 (COVID-19) has led to significant global health challenges (1). The pathogen causing this illness is a novel RNA coronavirus, designated as Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2). The pandemic originated in the city of Wuhan, located in the People's Republic of China (PRC), and subsequently spread rapidly to other cities and countries worldwide (1). Due to its 80% genetic similarity to SARS-CoV—the virus responsible for acute respiratory distress syndrome (ARDS) and high mortality during the 2002–2003 outbreak—the novel coronavirus was designated as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), also known as 2019-nCoV (2). SARS-CoV-2 was initially thought to have originated from zoonotic transmission, potentially linked to a seafood market in Wuhan, China. However, it soon became evident that human-to-human transmission played a significant role in the subsequent global outbreak (2).

Nigeria announced its initial confirmed case of COVID-19 on the 27th February 2020, in Ogun State, according to the Federal Ministry of Health. This made Nigeria the third African country, after Egypt and Algeria, to detect an imported case of the virus (3). There exists a significant likelihood that SARS-CoV-2 may adversely affect other essential organs, notably the kidneys and thyroid gland. The expression levels of the SARS-CoV-2 cell receptor, specifically the ACE2 receptor gene, differ across various human organs, with the highest expression observed in the small intestine, followed by the hypothalamus, pituitary gland, testes, heart, thyroid, kidneys, and lungs, each exhibiting varying levels of expression (4).

The mortality rate among patients suffering from severe Acute Kidney Injury (AKI) requiring dialysis has not shown a significant decrease over the past fifty years, despite advancements in supportive care (5). Given the report of SARS-CoV-2 inducing renal dysfunction and the associated economic and health repercussions, this issue warrants critical examination (6). Research shows that patients recovering from COVID-19 may also face long-term effects called "long COVID." These can involve symptoms affecting the kidneys, thyroid, and other organs beyond the lungs, aside from the acute illness. Even if patients had kidney issues before, acute kidney injury remains a notable non-respiratory complication of COVID-19 (7). A recent report has also described the development of thyroid dysfunction in patients diagnosed with COVID-19, although such information remains scarce within our environment (8). So far, we have seen that organ dysfunction is becoming more common both in Nigeria and around the world. We also know that there's a noticeable connection between the thyroid and kidneys (9,10).

Diagnosing AKI can be quite challenging because the standard methods we use to estimate urea and creatinine levels often lead to delays in recognizing the condition early on. It's important to be aware of this to ensure timely diagnosis and treatment (11). There is limited information about the long-term effects of SARS-CoV-2 infection on kidney and thyroid functions after the pandemic. It is also unclear whether people exposed to SARS-CoV during the pandemic might have sub-clinical renal and thyroid issues that could worsen over time. Therefore, this study aims to investigate the effects of SARS-CoV-2 on kidney and thyroid health in individuals exposed to the virus. The findings could be crucial for the early detection, ongoing monitoring, and management of post-pandemic health issues related to the virus.

II. MATERIALS AND METHOD

This was a cross-sectional study involving a total of 165 apparently healthy men and women between ages of 18 and 60 years. The participants were selected from various local government areas in Ibadan, Oyo State. All individuals agreed to take part in the study after providing informed consent. Ethical approval for the research was obtained from the Oyo State institutional review board prior to the study. The research focused on a period of up to eight months following the participants' exposure to SARS-CoV-2, and it included a comparison group of healthy individuals who had not been exposed to the virus.

The study involved an initial group of 120 individuals exposed to SARS-CoV-2 and a matched group of 150 healthy participants with no known exposure to the virus. To ensure accurate classification, all participants were tested for SARS-CoV-2 antibodies. Blood pressure, HbA1c, urea, and creatinine levels were also measured as part of the screening process. Ultimately, the final study included 85 unexposed individuals who tested negative for both the virus and antibodies, and 80 exposed individuals who tested positive for the virus and antibodies.

A semi-structured questionnaire was used to gather valuable insights from participants about their age, gender, background, socioeconomic status, and dietary habits. The study carefully excluded individuals with a history of kidney problems, high blood pressure, diabetes, thyroid issues, those who had received a COVID-19 vaccine, and anyone who chose not to give consent, ensuring respectful and precise selection.

Blood samples were collected from the participants for various tests. We used serum to analyze electrolytes such as chloride, sodium, potassium and bicarbonate, as well as urea, creatinine, cystatin-C, thyroid stimulating hormone (TSH), free T4, free T3, and SARS-CoV-2 spike protein antibodies. Additionally, we collected samples into EDTA tubes to measure glycosylated hemoglobin (HBA1C).

The serum levels of SARS-CoV-2 spike protein antibodies, FT3, FT4, and TSH were measured using a chemiluminescence immunoassay, following the standardized protocol provided with the Abbott Architect analyzer. Serum cystatin C levels were determined via spectrophotometry,

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adhering to the manufacturer's protocol for the Hitachi-Roche Cobas analyzers. Similarly, serum urea and creatinine were measured using spectrophotometry, using the protocols supplied with the Abbott Architect c4000. Electrolytes such as potassium, bicarbonate, sodium, and chloride were analyzed with the Erba Lyte analyzer, following the standard procedure included with the ERBA kit.

➢ *Statistical Analysis*

The data collected were analyzed using SPSS version 28. Results were presented as the mean value plus or minus the standard deviation for each parameter. To determine if there were significant differences between the groups, an independent Student's t-test was performed. We considered results with a p-value less than 0.05 as statistically significant.

III. RESULTS

Table 1: Socio-Demographic Data of Participants

Variable	Unexposed n (%)	Exposed n (%)
Gender		
Male	58 (68.2)	54 (67.5)
Female	27 (31.8)	26 (32.5)
Age		
19-25	18 (21.18)	19(23.8)
25-40	52(61.18)	38(47.5)
40 >	15 (17.65)	23 (28.8)
Marital Status		
Married	51 (60)	54 (67.5)
Single	32 (37.6)	25 (31.3)
Divorce	2 (2.4)	1 (1.2)
Local Government of Residence		
Ibadan North	49 (57.6)	47 (58.8)
Lagelu	24 (28.2)	25 (31.3)
Oluyole	2 (2.4)	3 (3.8)
Ibadan Northwest	10 (11.8)	5 (6.3)

Among the 85 unexposed participants, 58 (68.2%) were male, while 27 (31.8%) were female. The age distribution of the unexposed subjects (see Table 1) ranges from 19 to 25 years, comprising 18 individuals (21.18%), 25 to 40 years, accounting for 52 individuals (51.18%), and over 40 years, representing 15 individuals (17.65%). The most prominent age group in this study is the 25-40 years range, which is typically the most active and productive demographic with the highest mobility; however, this group was partially restricted by the lockdown during the pandemic. From the 80 exposed subjects, 54 (67.5%) were male while 26 (32.5%) were female. The age range for the exposed participants (Table 1) is 19-25 years with 19 (23,8%), 25- 40 years with 38 (47,5%), and those above 40 years with 23 (28.8%) (Table 1).

Furthermore, most of the subjects in the unexposed and exposed groups were married and most of them reside at the Ibadan North Local Government area.

The study found no significant difference in cystatin C levels between unexposed and exposed subjects, with a p-value greater than 0.05. Similarly, the levels of electrolytes, urea, and creatinine did not differ significantly between the two groups (Table 2).

Table 2: Renal Markers (Cystatin C, Electrolytes, Creatinine and Urea)

Renal Markers	Unexposed Mean ± SD	Exposed Mean ± SD	p-value
Cystatin C (mg/dl)	0.76 ± 0.25	0.72 ± 0.18	0.202
Na^+ (mmol/L)	139.24 ± 2.728	139.68 ± 2.782	0.307
K^+ (mmol/L)	3.945 ± 0.3386	3.971 ± 0.2926	0.592
CL^- (mmol/L)	101.34 ± 3.045	102.02 ± 2.311	0.108
HCO_3^- (mmol/L)	23.53 ± 2.950	23.02 ± 1.903	0.197
Urea (mg/dl)	20.94 ± 5.838	22.59 ± 5.158	0.057

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All renal parameters analyzed were within normal reference ranges, except for 1.3% of the exposed population whose serum sodium levels slightly exceeded the normal limit (Table 3). Values are presented as mean ± SD. *Significance at $p < 0.05$

Table 3: Prevalence of Renal Dysfunction in Exposed

Biomarker	Exposed Mean ± SD	Reference Range	Low	Normal	Elevated	% with elevated level
Cys C (mg/L)	0.72± 0.181	0.6-1.5	17	63	0	0
Na^+ (mmol/L	139.68 ± 2.78	135 - 145	3	76	1	1.3
K^+ mmol/L	3.97 ± 0.29	3.5 -5.0	0	80	0	0
CL^- mmol/L	102.02 ± 2.3	95 - 110	0	80	0	0
HCO_3^- mmol/L	23.02 ± 1.90	20 - 30	2	0	0	0
Urea (mg/dl)	22.59 ± 5.158	15 - 45	24	56	0	0
Cr (mg/dl)	0.829 ± 0.21	0.5 - 1.5	2	78	0	0

There were no notable differences in thyroid function tests, such as FT3, FT4, and TSH—between the groups that were unexposed and those that were exposed, with all values falling within normal ranges. (See Table 4 for details).

Table 4: Markers of Thyroid Function

Biomarkers	Unexposed Mean ± SD	Exposed Mean ± SD	p-value
Thyroid function Markers			
Free Tri-iodothyronine (FT3) pmol/L	3.89 ± 0.439	3.86 ± 0.454	0.838
Free Thyroxine (FT4) pmol/L	15.2 ± 2.67	15.89 ± 2.48	0.098
Thyroid Stimulating Hormone (TSH) mIU/L	1.62 ± 1.04	1.41 ±0 .975	0.194

IV. DISCUSSION

Based on the results, out of a total of 165 participants, 80 tested positive for SARS-CoV-2 antibodies, indicating a seroprevalence of 29.63% in this study. This figure is lower than that reported in a previous study among healthcare workers at University College Hospital Ibadan, which found a seroprevalence of 45.1%. The elevated rate observed among healthcare workers may be ascribed to their frontline responsibilities during the pandemic, which subject them to an increased risk of infection. Throughout this period, medical and healthcare personnel have been notably vulnerable due to their direct involvement in sample collection, patient monitoring, treatment, and follow-up care.

Nevertheless, investigations conducted between December 2020 and March 2021, before the deployment of the SARS-CoV-2 vaccine across Nigeria's six geopolitical zones, demonstrated a seroprevalence of 66.8%, indicating a notably significant level of herd immunity at the height of the pandemic (13). A cross-sectional study involving 4,904 participants across 12 states in Nigeria reported a high seroprevalence of 78.9%. The data showed consistent seropositivity, ranging from 69.8% in Lagos to 87.7% in Borno. These findings indicate an increase in seroprevalence following the rollout of the vaccine. Additionally, the study suggests that COVID-19 infection remained widespread in Nigeria, despite a relatively low hospitalization rate at the time of sampling (14).

Furthermore, the low rate of antibodies observed in this study isn't unexpected. This is because we specifically selected individuals who tested negative for SARS-CoV-2 using RT-PCR, meaning they were likely unexposed. Also, it's worth noting that natural immunity to the virus can last up to about 11 months after infection, but then it tends to fade over time (15).

The mean ± SD of the renal predictor cystatin C showed no significant difference ($p > 0.05$) between the unexposed and exposed groups, indicating no statistically meaningful difference. This implies that individuals who contracted SARS-CoV-2 during the pandemic likely fully recovered from any renal impairment incurred during infection. Alternatively, renal impairment might have been less common in Ibadan during the active phase of the disease.

Studies conducted outside Nigeria during the same period reported elevated serum cystatin C levels in many patients, which were associated with increased COVID-19 severity and higher mortality (16). Elevated levels of cystatin C in people with COVID-19 often suggest kidney problems, like acute kidney injury. But they might also be a sign of the widespread inflammation and increased oxidative stress that many COVID-19 patients experience (17).

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This research examines how SARS-CoV-2 infection affects kidney health after the pandemic. Our findings show that people who have been exposed to the virus generally do not experience ongoing kidney problems. However, this seems to differ from other reports that suggest some patients, even those who didn't suffer from acute kidney injury initially, may experience a decline in kidney function over the 6 to 12 months following infection (18).

The mean ± SD values for electrolytes (sodium, potassium, chloride, and bicarbonate) showed no significant difference between unexposed and exposed groups ($p > 0.05$). This indicates that there are no electrolyte imbalances following initial exposure to COVID-19. However, electrolyte disturbances such as hyponatremia, hypernatremia, and hypercalcemia have been commonly reported during active COVID-19 infection and are associated with poorer outcomes, independent of inflammatory marker levels. Additionally, other studies have observed electrolyte imbalances in COVID-19 patients admitted to emergency departments (20).

According to the study, individuals typically do not experience electrolyte imbalances following an initial COVID-19 infection. However, there has been a reported case of persistent hypokalemia in a patient for more than five months post-diagnosis. Additionally, this patient exhibited prolonged hypomagnesemia. If not monitored closely, these electrolyte disturbances could potentially result in life-threatening arrhythmias and seizures (21).

The mean ± SD values of urea and creatinine levels did not show significant differences between the unexposed and exposed groups ($p > 0.05$). This indicates that there is no evidence of kidney dysfunction following initial exposure to COVID-19. It is worth noting that kidney dysfunctions are commonly reported during active SARS-CoV-2 infection (22). This may be because the kidney, as one of the extra-pulmonary organs, expresses the ACE-2 receptor, which SARS-CoV-2 uses to enter cells. Additionally, hyponatremia, frequently observed in COVID-19 patients, may also impact kidney function (23).

Meanwhile, this study indicates that no post-exposure kidney dysfunction was observed in Ibadan. This finding contrasts with a multinational cohort study involving the United States, Singapore, France, Spain, and Italy, which reported that COVID-19-associated acute kidney injury is linked to poorer long-term recovery of kidney function after acute kidney injury (24).

A retrospective study conducted at a single center in Turkey found that patients who survived mild, moderate, or severe COVID-19 did not experience adverse kidney outcomes following the acute phase of the illness. The study further suggests that the kidneys may have a capacity for long-term self-protection. These findings are consistent with observations reported earlier in Ibadan, Nigeria.

This study found no statistically significant difference ($p>0.05$) in the mean ± SD values of FT3, FT4, and TSH between the unexposed and exposed groups. This suggests that there have been no cases of thyroid dysfunction among exposed individuals in Ibadan, Nigeria, post-pandemic. However, it has been reported that during the active phase of SARS-CoV-2 infection, the thyroid gland and the virus interact intricately via hormonal and immunomodulatory signaling pathways (26). Consequently, various thyroid dysfunctions such as thyroiditis, thyrotoxicosis, non-thyroidal illness syndrome, hypothyroidism, and hyperthyroidism have been observed. This is perhaps unsurprising, given that the thyroid gland is an extra-pulmonary organ that expresses the ACE-2 receptor for SARS-CoV-2 (4).

During the pandemic, numerous investigations identified cases of primary hypothyroidism associated with COVID-19. In a particular study, 5.2% (15/287) of patients experienced primary hypothyroidism, which was subclinical in 90% of cases (with FT3 and FT4 levels within reference limits), while in the remaining 10%, levels exceeded the upper limit of the reference range (27). The authors also observed that the in-hospital mortality rate among COVID-19 patients with hypothyroidism was higher compared to those with euthyroidism (27). Hypothyroidism may negatively impact COVID-19 outcomes, similar to thyrotoxicosis, but likely to a lesser degree. A study of individuals with COVID-19 who were admitted to intensive care units reported two cases of primary hypothyroidism caused by chronic autoimmune thyroiditis (CAT). In both cases, primary hypothyroidism manifested during the COVID-19 infection and persisted even after the patients were discharged. Additionally, a case report described an instance of overt primary hypothyroidism that developed seven days after the resolution of mild COVID-19 symptoms. There is growing evidence suggesting that primary hypothyroidism may arise during or after a COVID-19 infection (29).

Nevertheless, the findings of this study indicate the absence of post-pandemic thyroid dysfunction among exposed individuals in Ibada. This contrasts with previously cited reports and other studies that document the manifestation of thyroid diseases, such as Hashimoto's thyroiditis, Graves' disease, and subacute thyroiditis, subsequent to COVID-19 infection. (30).

V. CONCLUSION

There is an increasing global interest in understanding the impact of SARS-CoV-2 infection on both morbidity and mortality rates. This has spurred numerous research efforts and innovations aimed at combating the pandemic. Despite the virus claiming lives worldwide and many individuals recovering from the infection, there remains limited information about its long-term effects on vital organs beyond the lungs, particularly in Nigeria. This study shows no significant differences in electrolyte levels such as sodium, potassium, chloride, and bicarbonate between those exposed to the virus and those

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unexposed. Likewise, there were no significant differences in renal markers like urea and creatinine when comparing the control group with the exposed individuals. The predictive marker for kidney injury was analyzed, revealing no significant difference in the levels of the predictive marker (cystatin-C) between the exposed and unexposed groups. Additionally, thyroid function tests (FT3, FT4, TSH) were conducted in both groups, and no significant differences were observed in thyroid function between the exposed and unexposed groups.

Further investigations should be conducted to understand the post-pandemic effects of SARS-CoV-2 infection on other vital organs that express the virus's receptor. The results would provide valuable insights for clinicians in managing the long-term impacts of SARS-CoV-2 on these organs in exposed individuals, helping to reduce morbidity and mortality associated with the disease.

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