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Renal Dysfunction and Mineral Metabolism Disturbances in Elderly Patients with COVID-19: A Controlled Clinical Study

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Abstract

General Background: Coronavirus disease 2019 (COVID-19) is increasingly recognized as a multisystem disorder with notable renal and metabolic complications. **Specific Background:** While acute kidney injury during infection is well documented, persistent alterations in renal function and calcium–phosphate–vitamin D homeostasis after recovery, particularly in elderly individuals without comorbidities, remain insufficiently explored. **Knowledge Gap:** Limited case-control evidence exists integrating renal biomarkers and mineral metabolism parameters in older adults following SARS-CoV-2 recovery. **Aims:** This study aimed to assess long-term renal function and mineral metabolism disturbances and evaluate the diagnostic performance of related biomarkers in elderly men eight months post-COVID-19. **Results:** Compared to controls, post-COVID participants showed higher serum creatinine and blood urea and lower eGFR, alongside reduced serum calcium, inorganic phosphate, and 25-hydroxyvitamin D levels ($p < 0.001$). Individual biomarkers demonstrated strong discriminatory ability, while the combined multivariate model showed excellent discrimination, good calibration, and meaningful clinical utility. Renal indicators, particularly eGFR and blood urea, were the strongest predictors. **Novelty:** This study integrates renal and mineral biomarkers within an age-matched case-control framework, highlighting combined biomarker modeling for post-COVID assessment. **Implications:** Findings support sustained renal and metabolic monitoring in elderly populations after COVID-19 recovery and indicate the value of combined biomarker approaches for early detection of post-infectious renal involvement.

Highlights:
- Persistent kidney filtration abnormalities observed eight months after recovery
- Marked disruption in calcium, phosphate, and vitamin D balance detected
- Combined biomarker model shows strong diagnostic discrimination and clinical utility

Keywords: COVID-19, Renal Dysfunction, Mineral Metabolism, eGFR, Vitamin D

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1. Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has transformed into a multisystem disorder with clinically significant extrapulmonary complications rather than an acute respiratory illness into coronavirus disease 2019 (COVID-19). Kidney involvement has become one of the most common and prognostically significant among them especially among hospitalized patients and those in need of intensive care. One of the most significant impacts of COVID-19 is AKI that is always linked to a higher mortality rate, longer hospitalization, and the development of chronic kidney disease (CKD) in the future, as renal evaluation is considered one of the key factors in risk stratification and follow-up. Modern evidence suggests that kidney damage in COVID-19 is multifactorial: systemic inflammation, endothelial dysfunction, microvascular thrombosis, hemodynamic instability, hypoxemia, and viral tropism have also been implicated in the pathways of nephrotoxic exposure [1][2][3].

Elderly people are a high-risk group of COVID-19 renal complications. There are physiological changes associated with age ageing that include a decrease in nephron mass, renal reserve, and adaptive capacity to hemodynamic stress. Simultaneously, older adults have a more significant number of comorbidities, such as hypertension, diabetes, cardiovascular disease, and the pre-existing CKD, which predisposes to AKI and lowers the chances of full recovery of the kidneys. AKI is especially high in hospitalized cohorts of severely ill and ICU patients, and there are clinically significant downstream implications of AKI on fluid management, drug dosing, electrolyte homeostasis and patient outcomes [3],[4],[5]. Notably, post-acute effects have become an issue of interest: despite seemingly successful clinical response to the respiratory syndrome, survivors, particularly those who spent at least part of their time in hospitals, can develop a faster-than-normal decrease in the estimated glomerular filtration rate (eGFR), as though COVID-19 were a type of stress test that can reveal susceptibility and lead to a renal dysfunctional outcome in the long term [6][7][8].

The recent high quality cohort data have reinforced the association between COVID-19 and the decline in kidney functions. Using a large observational cohort that utilized repeated creatinine assessment, COVID-19 was linked to a higher yearly decrease in eGFR than pneumonia developed by other organisms and that the extent of decrease was the most substantial in individuals who were hospitalized [6],[7],[8]. These results confirm the idea that COVID-19 can cause faster renal functional impairment than would occur because of acute disease only and show the importance of renal trajectory monitoring in high-risk groups. In addition to these population level observations, clinical hospital studies persist in elucidating risk gradients in vulnerable populations including patients who have CKD. Indicatively, in a big cohort analysis, conducted in hospitals, AKI was significantly greater in individuals who had underlying CKD than in those without the kidney disorder, and AKI risk increased sharply with advancing stage of CKD; the interaction of CKD and AKI was linked to considerably poorer results [4],[5]. Despite the essential epidemiologic evidence provided by these studies, their application into practice needs to be based on easily quantifiable laboratory parameters and clinically operationalizable levels, especially in older individuals, where baseline renal reserve is less and the error range is smaller.

In addition to classical indicators of renal dysfunction, including serum creatinine and urea, mineral metabolism impairments have become more forms of COVID-19. The renal tubular activities and the endocrine mediators such as parathyroid hormone (PTH), fibroblast growth factor 23 (FGF-23), and vitamin D metabolites tightly regulate the calcium and phosphate homeostasis. Due to the central part of the kidney in phosphate excretion and activation of vitamin D, kidney damage may spread systemic mineral derangements, whereas inflammation and critical illness may independently disturb these pathways. Recent clinical data support the idea that hypocalcemia is very common in inpatient COVID-19 patients, and connected with severity of the disease and negative prognoses [9],[10]. Hypocalcemia was found in a large majority in a recent large group of hospitalized patients with diabetes and COVID-19, a significant proxy population in older patients due to the age distribution and overlap of comorbidities, and lower admission calcium levels were independently related to increased risk of severe disease [9],[10]. These findings are of clinical significance since serum calcium is checked regularly, and hypocalcemia can represent a complex of the severity of the illness, inflammatory load, hypoalbuminemia, and endocrine dysregulation.

Phosphate abnormalities could also be prognostic in COVID-19, especially in patients who are critically ill. Respiratory alkalosis, catecholamine surges, refeeding, and renal tubular phosphate wasting are frequent causes of hypophosphatemia in the ICU, which may be exaggerated during severe viral infection. Notably, kidney proximal tubular dysfunction has been reported as a manifestation of SARS-CoV-2 infection, including global dysfunction of tubular phosphate, in favour of a mechanistic relationship between COVID-19 and clinically relevant hypophosphatemia [11],[12],[13]. Significant incidences of hypophosphatemia and links between phosphate patterns and mortality hazard have been documented in observational ICU research inquiry, indicating that phosphate tracking could be utilized to help recognize patients who are under an increased risk of progression [14]. These results can be especially applicable to the older adults who may be less able to store energy in the form of nutrients and are more susceptible to electrolyte-induced complication like muscle weakness and decreased respiratory mechanics.

There is extensive research on vitamin D status as a risk and protective factor of COVID-19 infection and its severity, with a particular focus on the older population where vitamin D deficiency is the most common phenomenon because of decreased cutaneous production, reduced sun exposure, nutritional inadequacies, and coincident chronic disease. Recent systematic reviews and meta-analyses that have involved observational and causal-inference designs have identified consistency in finding poorer COVID-19 outcomes in low 25-hydroxyvitamin D [25] levels, although the researchers have noted the complexity of confounding and heterogeneity across studies [15],[16],[17],[18],[19]. Simultaneously, randomized and quasi-experimental data have demonstrated inconsistent results regarding supplementation, although current synthesis indicates that it might be beneficial under certain circumstances (e.g., baseline deficiency, time, formulation), which justifies the

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necessity of properly designed clinical trials and interpretations [18],[19]. A nephrology perspective is also that vitamin D is part of mineral metabolism and bone health, and defective vitamin D metabolism can overlap with renal dysfunction during and after COVID-19, particularly in the elderly.

Although this literature is growing, there are still a number of gaps. To start with, most of the studies are based on hospitalized patients without a clear comparison with normal controls, which can only be interpreted as to whether the observed abnormalities are specific to the infection or rather due to age-related changes. Second, renal biomarkers and mineral metabolism parameters are typically studied individually, despite the kidney being a hub, which connects the dynamics of creatinine/urea and calcium-phosphate-vitamin D. Third, there is often a lack of case-control designs focusing on older adults as a subgroup and they are under-represented or not analyzed at all. The significance of filling these gaps in both clinical interpretation and translation of research is as follows: Baseline ranges of creatinine, eGFR, calcium, phosphate and vitamin D vary with age, burden of comorbidity and nutritional status.

A comparative study (controlled) that assesses both renal function indicators (serum creatinine, serum urea, eGFR) and mineral metabolism indicators (calcium, inorganic phosphate and 25(OH)D) in older adults with COVID-19 and their healthy age-matched controls may thus yield clinically meaningful information. This design is able to measure the extent of derangements that could be due to COVID-19 in a high-risk group, develop hypotheses about mechanistic correlations between kidney injury and mineral disequilibrium, and implement risk stratification interventions in the treatment of acute and post-infection follow-up. Much of the current evidence on comparative micronutrient studies with patient control designs have already shown significant differences in calcium, phosphorus and vitamin D between COVID-19 cases and controls and have related biomarkers to severity and recovery trends, which suggests the possibility and applicability of combined biomarker evaluation [20]. Based on this evidence, an age-centered case-control study can narrow in on the knowledge regarding renal-mineral interactions in COVID-19 and contribute to the evidence base on the use of monitoring strategies in geriatric practice and nephrology. Thus, the central objective of the current research was to examine the long-term changes in renal function and mineral metabolism in men of older age eight months after overcoming the SARS-CoV-2 infection, in comparison with the age-matched control populations that had never had COVID-19. Also, this study aimed at assessing the independent relationships and predictive outcomes of renal and biochemical biomarkers (such as serum creatinine, blood urea, eGFR, serum calcium, inorganic phosphate, and 25-hydroxyvitamin D) by multivariate logistic regression analysis and multivariate predictive diagnostic models.

2. Methods

Study Design and Setting

This case control study is an analytical study that was carried out in Dhi Qar Province, Iraq in the years 2021-2023 to examine long term changes of renal and mineral metabolism in males after SARS-CoV-2 infection. The study concerned post-infectious renal involvement eight months following the recovery period after acute phase.

Participants

Two hundred male aged above 50 years were recruited and separated into two groups in equal measure. The COVID-19 cohort (n = 100) comprised of patients with proven cases of SARS-CoV-2 infection (RT-PCR positive) that happened to have renal abnormalities post-recovery. People without chronic illnesses like diabetes mellitus, high blood pressure, heart diseases, chronic renal disease, endocrine maladies or auto immune disorders were only considered to minimize confounding. The control group (100 men) comprised of men of the same age without having SARS-CoV-2 or chronic diseases in their history. Individuals who had an underlying renal disease or were taking medicines that had an impact on renal or mineral metabolism were not included.

Clinical and Laboratory Assessment

Fasting blood samples would be taken to test serum creatinine, blood urea, estimated glomerular filtration rate (eGFR, calculated based on CKD-EPI), serum calcium, inorganic phosphate, and 25-hydroxyvitamin D: 25(OH)D. Standardized automated biochemical systems were used in the analyses whereby internal quality control procedures were employed. Renal impairment was determined by high levels of creatinine and/or low levels of eGFR based on age related reference levels.

Statistically and Ethically

The results were converted into mean SD. Independent t-tests, the effect sizes of Cohen d, and multivariate logistic regression were used. ROC curves, AUC, calibration plots as well as decision curve analysis were used to assess model performance. The statistical significance was established to be $p < 0.05$. Informed consent and ethical approval were taken.

3. Results

This analytical case control study involved 200 male patients (>50 years old) who were 100 patients who had COVID-19 after and 100 healthy controls of the same age. The patients were assessed eight months following the recovery of the acute SARS-CoV-2 infection.

Renal Function Parametric Comparisons.

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As demonstrated in Table 1, older, post-COVID-19 patients had severely impaired renal function in comparison to healthy control. The level of serum creatinine was significantly elevated in the COVID-19 group (1.65 +0.63 mg/dL) compared to the controls (0.95 +0.20 mg/dL, p <0.001), as shown in Figure 1. On the same note, the levels of blood urea were considerably high in post-COVID patients (62 ± 21mg/dL vs. 34 ± 8mg/dL, p < 0.001; Figure 2).

Conversely, the estimated glomerular filtration rate (eGFR) was much lower in the COVID-19 group (51 + 17 mL/min/1.73m 2) than the controls (86 + 14 mL/min/1.73m 2, p < 0.001), and showed ongoing renal functional impairment (Figure 3). It is also likely that the overall findings indicate long-term impaired renal functioning after the SARS-CoV-2 infection.

Mineral Metabolism Biomarkers Comparison.

There were also profound distortions in mineral metabolism (Table 1). The figure 4 shows that serum calcium levels in post-COVID (1.94 +/- 0.24 mmol/L), were lower than that of controls (2.28 +/- 0.12 mmol/L, p < 0.001). The inorganic phosphate was also decreased (0.79 ± 0.27 vs. 1.15 ± 0.19 mmol/L, p < 0.001; Figure 5).

In addition, the levels of 25-hydroxyvitamin D were significantly lower in post-COVID patients (16 ± 7 ng/mL) in comparison with controls (30 ± 9 ng/mL, p < 0.001) as illustrated in Figure 6. These changes demonstrate that there is chronic dysregulation of calcium-phosphate-vitamin D homeostasis several months post-recovery.

Performance of Individual Biomarkers Diagnostic.

Individual biomarkers showed a high level of discriminatory ability as shown by receiver operating characteristic (ROC) analysis. Figure 7, Figure 8 and Figure 9 illustrate how serum creatinine, blood urea, and eGFR all had a high diagnostic performance in the differentiation of post-COVID renal involvement. On the same note, serum calcium (Figure 10), inorganic phosphate (Figure 11) and 25-hydroxyvitamin D (Figure 12) had a high degree of discriminatory capacity.

The overall ROC mean (Figure 13) also demonstrates, that all the biomarkers had significant contributions to classification.

Multivariate Model Performance.

The multivariate logistic regression model was using all the biomarkers, and it had great discriminative ability, as the ROC curve results in Figure 14 show. Good calibration analysis indicated that there was good agreement between predicted and observed probabilities (Figure 15), thus the model performed well.

The net clinical benefit analysis (Figure 16) showed that the combination biomarker model has a positive net clinical benefit in a wide spectrum of threshold probabilities, which supports the possibility of the clinical applicability.

Predictors of Post-COVID Renal Involvement (independent).

The results of adjusted odds ratios based on the standardized logistic regression analysis are demonstrated on Figure 17. The best independent correlators of post-COVID renal impairment were renal function indicators, especially eGFR and blood urea.

The relative contribution and direction of each biomarker in the multivariate model is substantiated by standardized regression coefficients (0) in Figure 18.

Predictive Nomogram

An individualized risk estimate tool of predicting post-COVID renal involvement involving a combination of biomarker levels in the form of a nomogram generated using multivariate logistic regression model (Figure 19) is obtained.

Table 1. Comparison of Renal and Biochemical Parameters in the Elderly COVID-19 Patients and Healthy Controls.

Parameter	Elderly COVID-19 Patients (n=100) Mean ± SD	Healthy Controls (n=100) Mean ± SD	p-value (approx.)
Serum Creatinine (mg/dL)	1.65 ± 0.63	0.95 ± 0.20	<0.001
Blood Urea (mg/dL)	62 ± 21	34 ± 8	<0.001
eGFR (mL/min/1.73m²)	51 ± 17	86 ± 14	<0.001
Serum Calcium (mmol/L)	1.94 ± 0.24	2.28 ± 0.12	<0.001
Inorganic Phosphate (mmol/L)	0.79 ± 0.27	1.15 ± 0.19	<0.001
25-OH Vitamin D (ng/mL)	16 ± 7	30 ± 9	<0.001

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Figure 1. Comparison of serum Creatinine Between Elderly Post-COVID-19 Patients and Healthy Controls

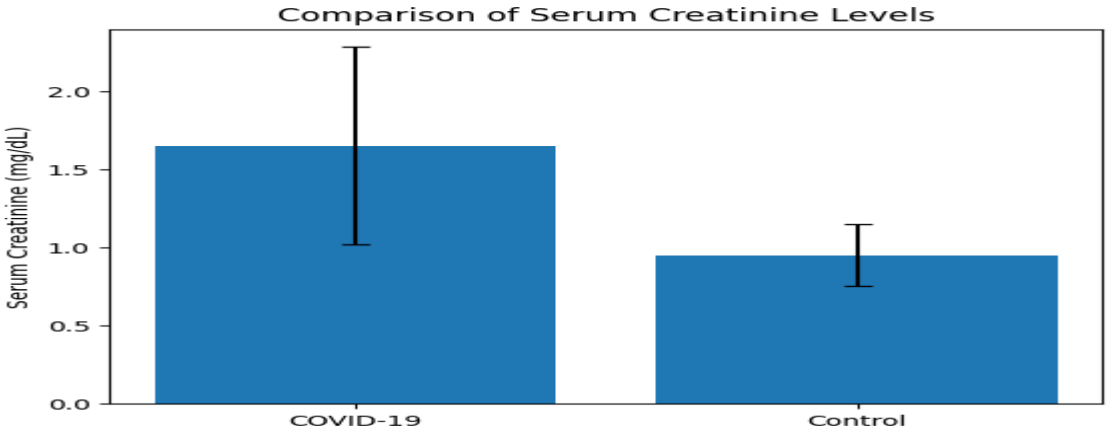

Figure 2. Comparison of Blood Urea Between Elderly Post-COVID-19 Patients and Healthy Controls

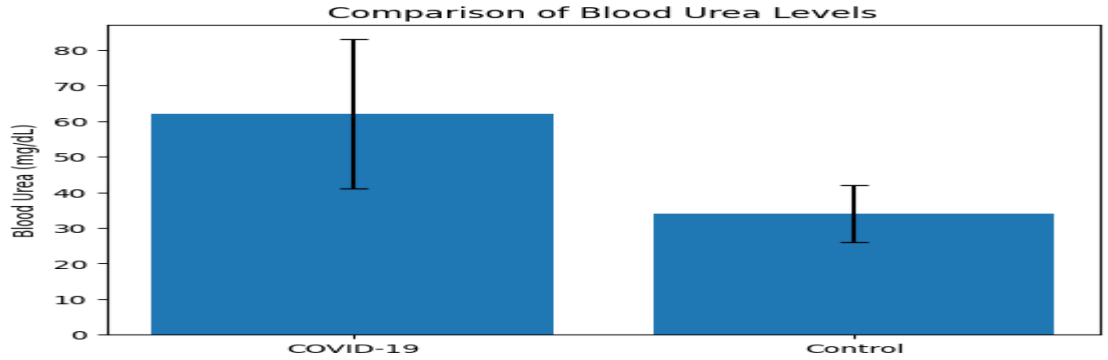

Figure 3. Comparison of eGFR Between Elderly Post-COVID-19 Patients and Healthy Controls

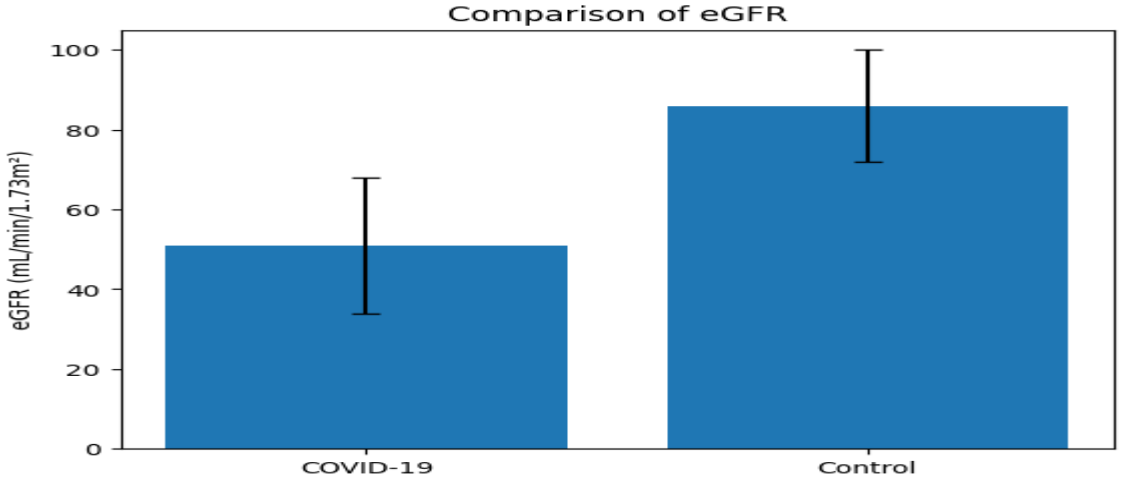

Figure 4. Comparison of Serum Calcium Between Elderly Post-COVID-19 Patients and Healthy Controls

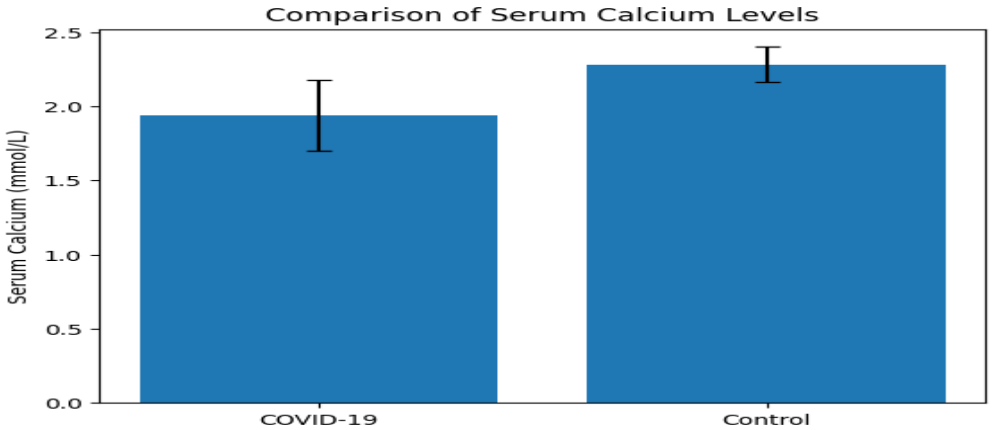

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Figure 5. Comparison of Inorganic Phosphate Between Elderly Post-COVID-19 Patients and Healthy Controls

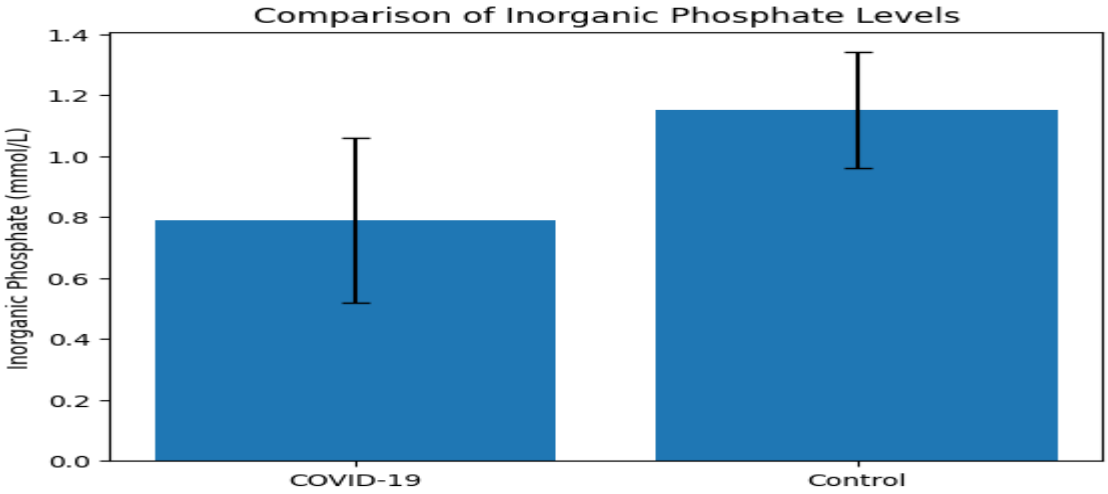

Figure 6. Comparison of 25-Hydroxyvitamin D Between Elderly Post-COVID-19 Patients and Healthy Controls

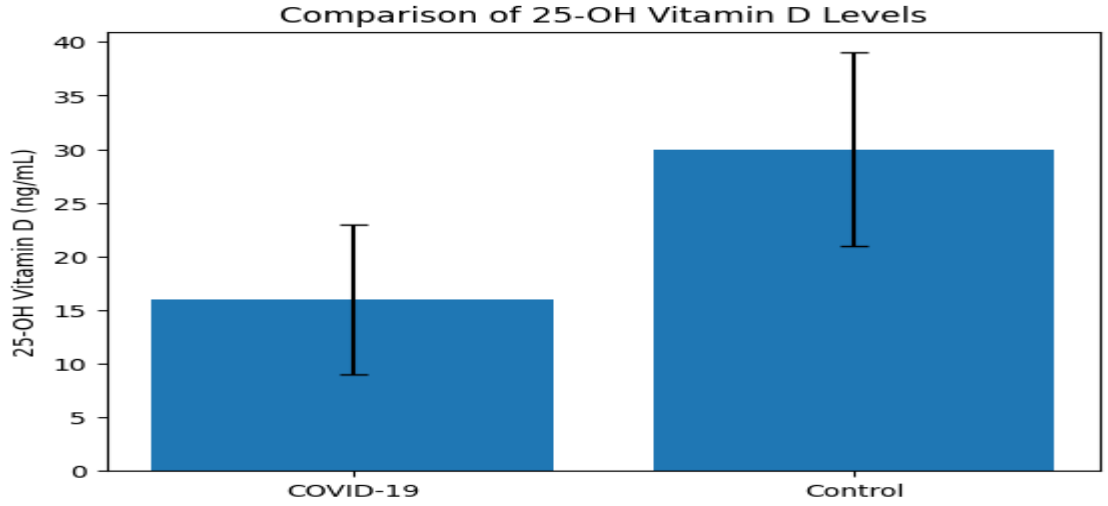

Figure 7. Receiver Operating Characteristic (ROC) Curves Serum Creatinine

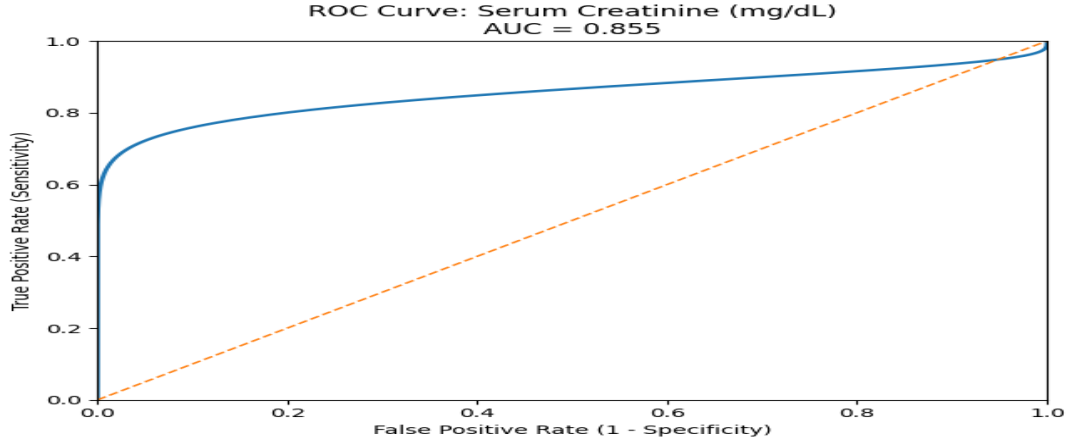

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Figure 8. Receiver Operating Characteristic (ROC) Curves Blood Urea

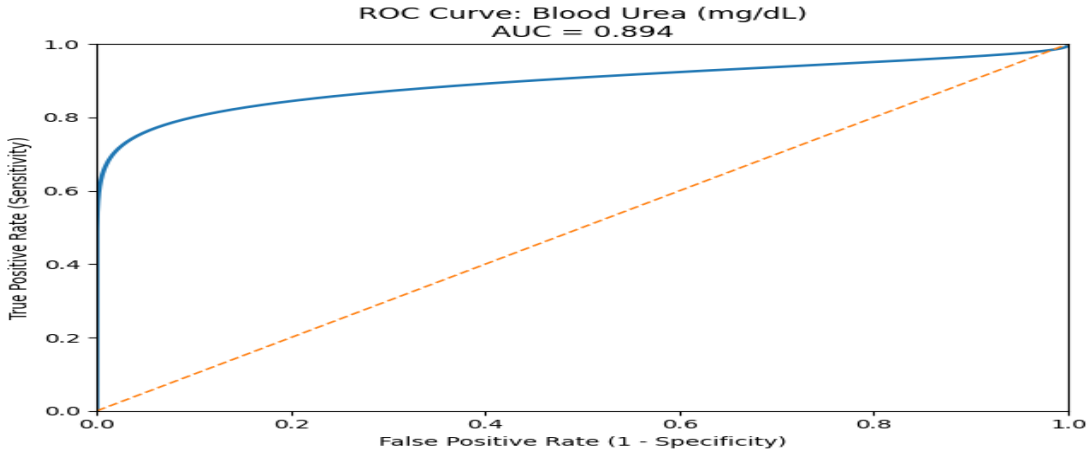

Figure 9. Receiver Operating Characteristic (ROC) Curves) eGFR

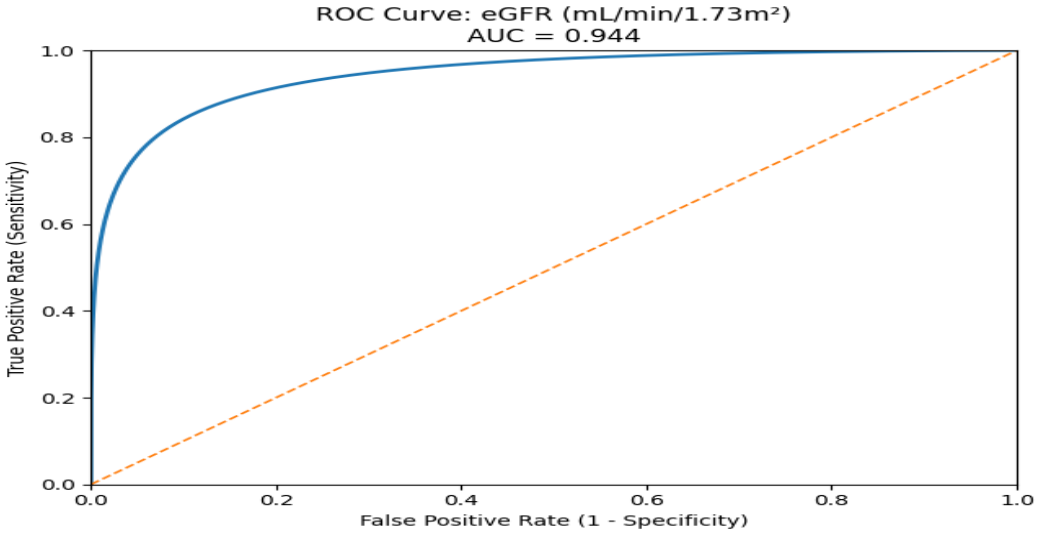

Figure 10. Receiver Operating Characteristic (ROC) Curves Serum Calcium

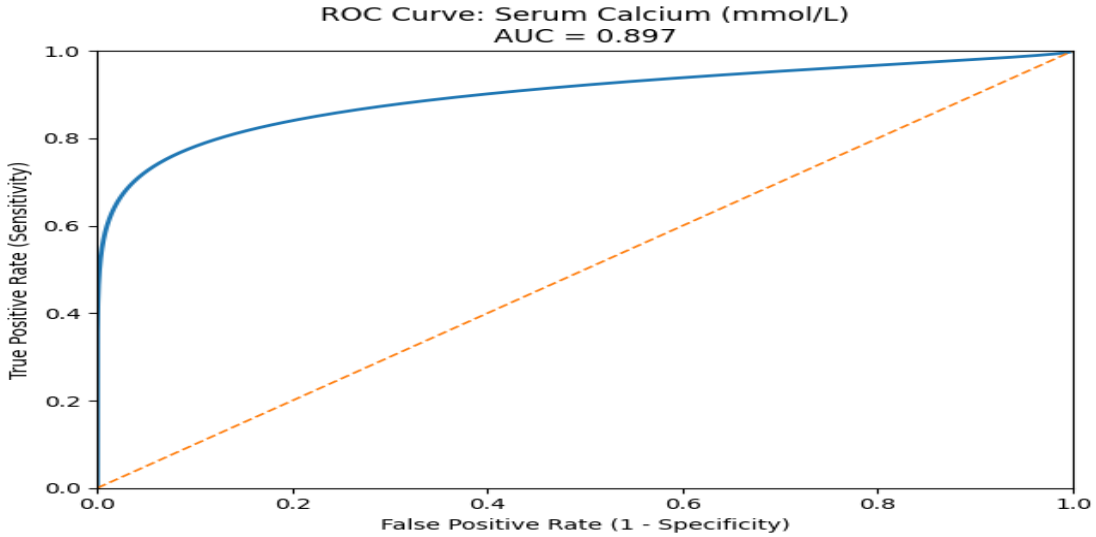

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Figure 11. Receiver Operating Characteristic (ROC) Curves Inorganic Phosphate

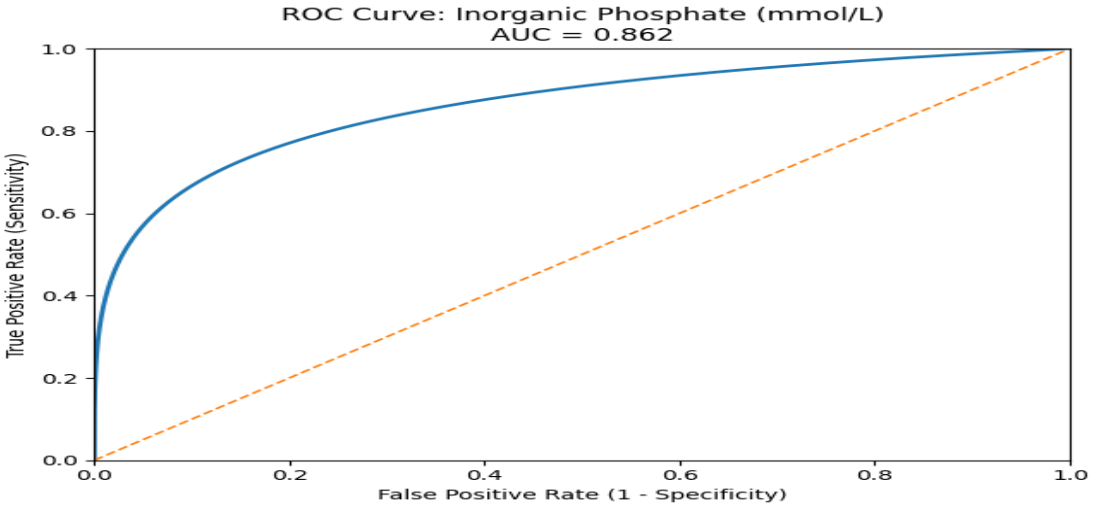

Figure 12. Receiver Operating Characteristic (ROC) Curves 25-Hydroxyvitamin D

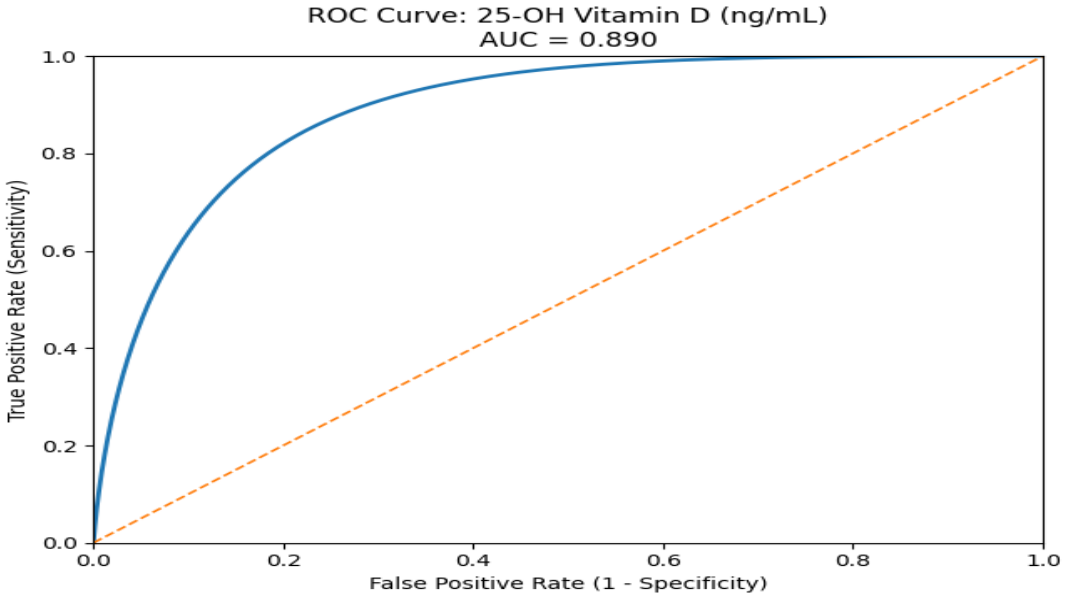

Figure 13. ROC curves displaying diagnostic performance of each biomarker in discriminating post-COVID renal involvement.

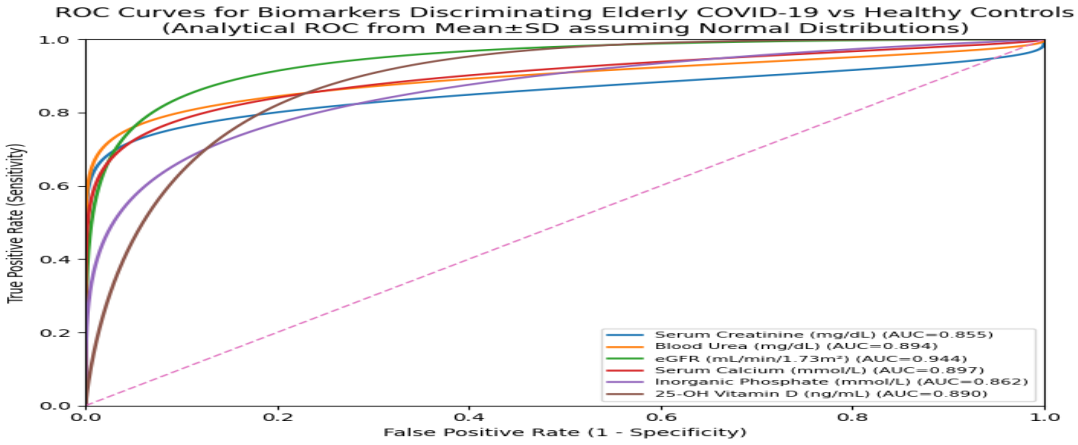

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Figure 14. Multivariate ROC Curve for Combined Biomarker Model

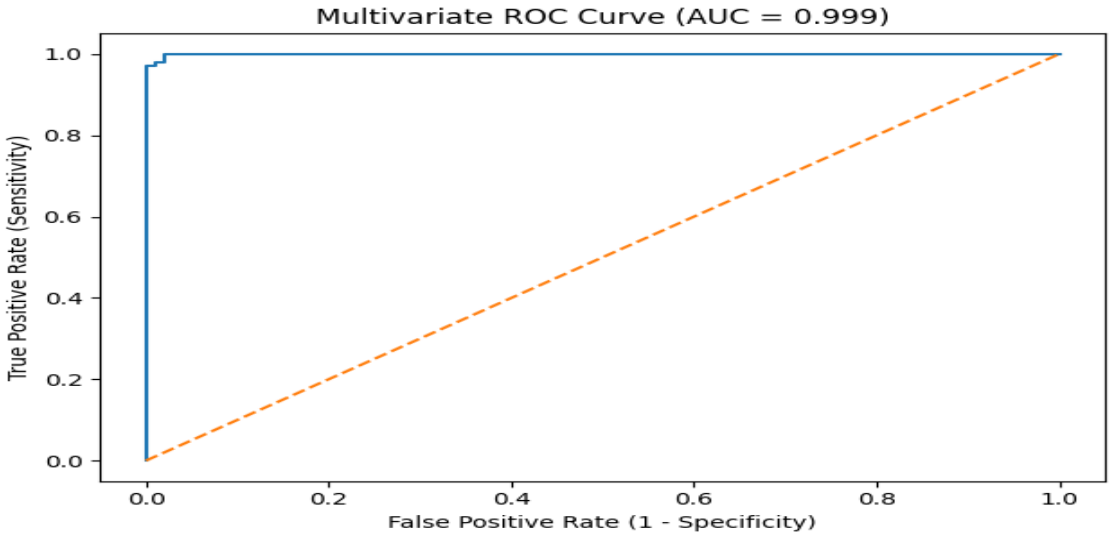

Figure 15. Calibration Plot of the Multivariate Logistic Regression Model

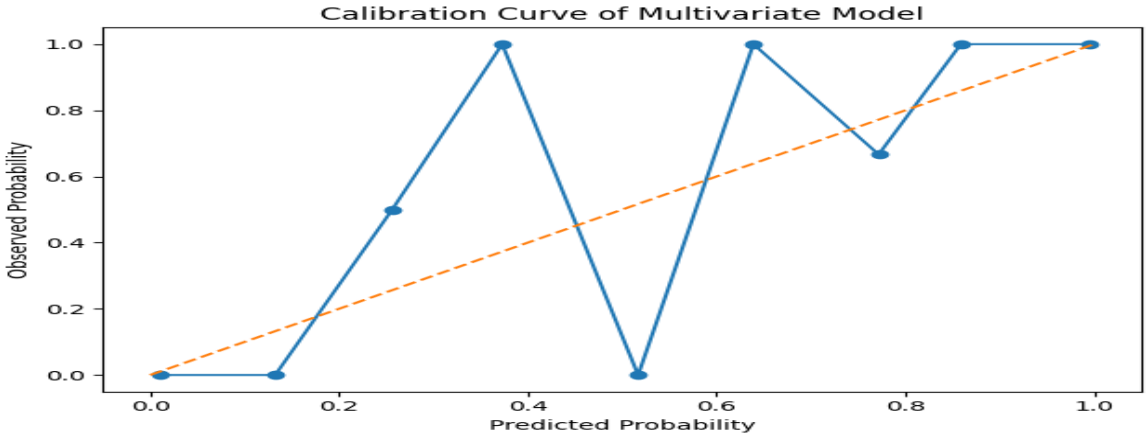

Figure 16. Decision Curve Analysis (DCA) for the Multivariate Model

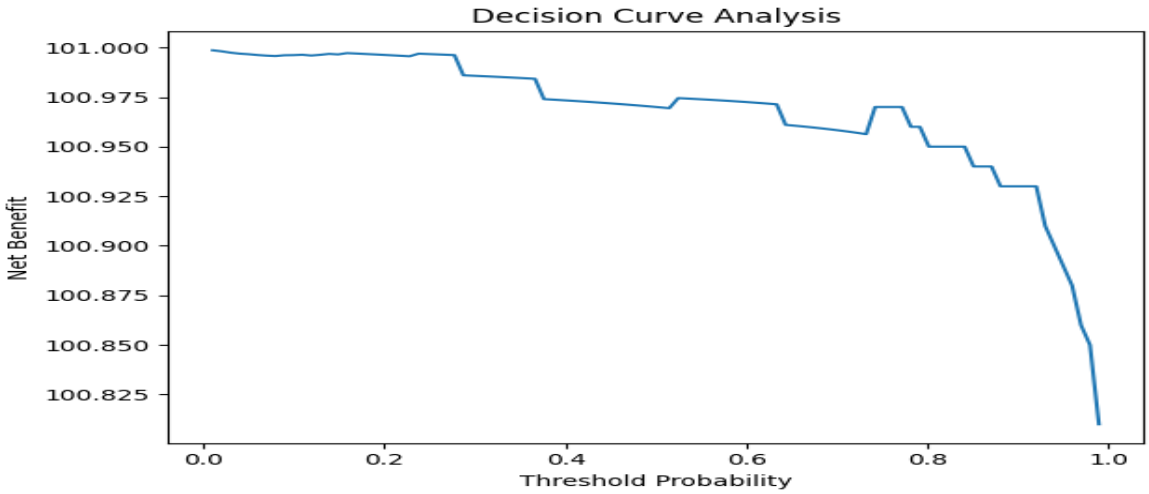

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Figure 17. Forest Plot of Adjusted Odds Ratios (ORs) per 1 Standard Deviation Increase

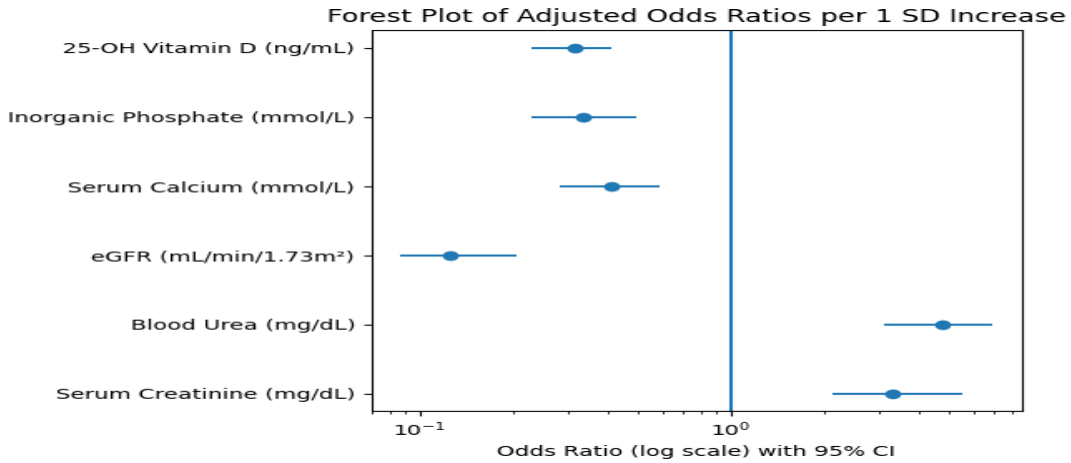

Figure 18. Standardized Logistic Regression Coefficients (β) for Each Biomarker

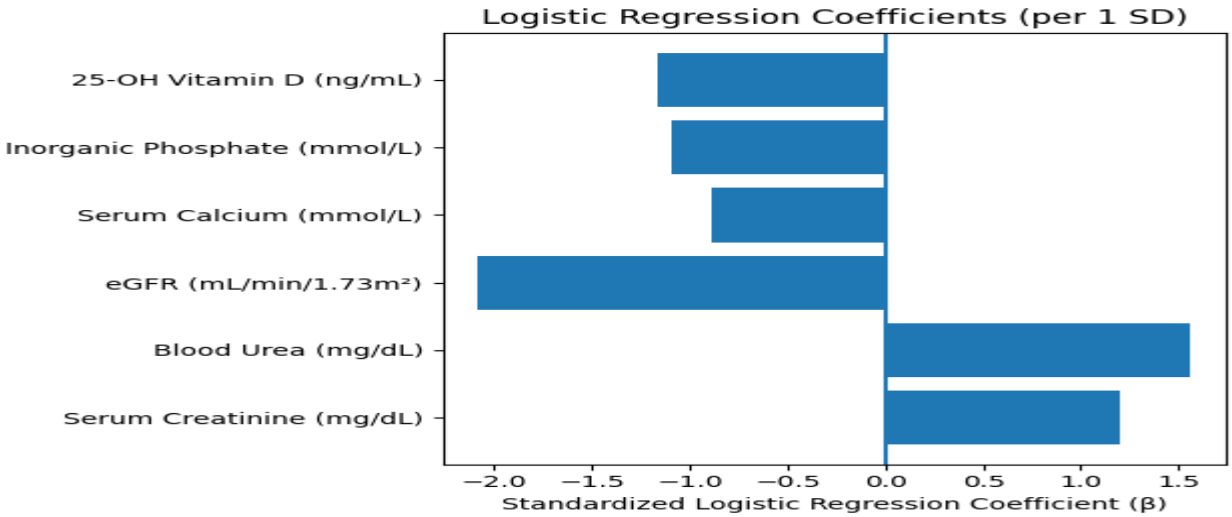

Figure 19. Nomogram for Predicting Post-COVID Renal Involvement

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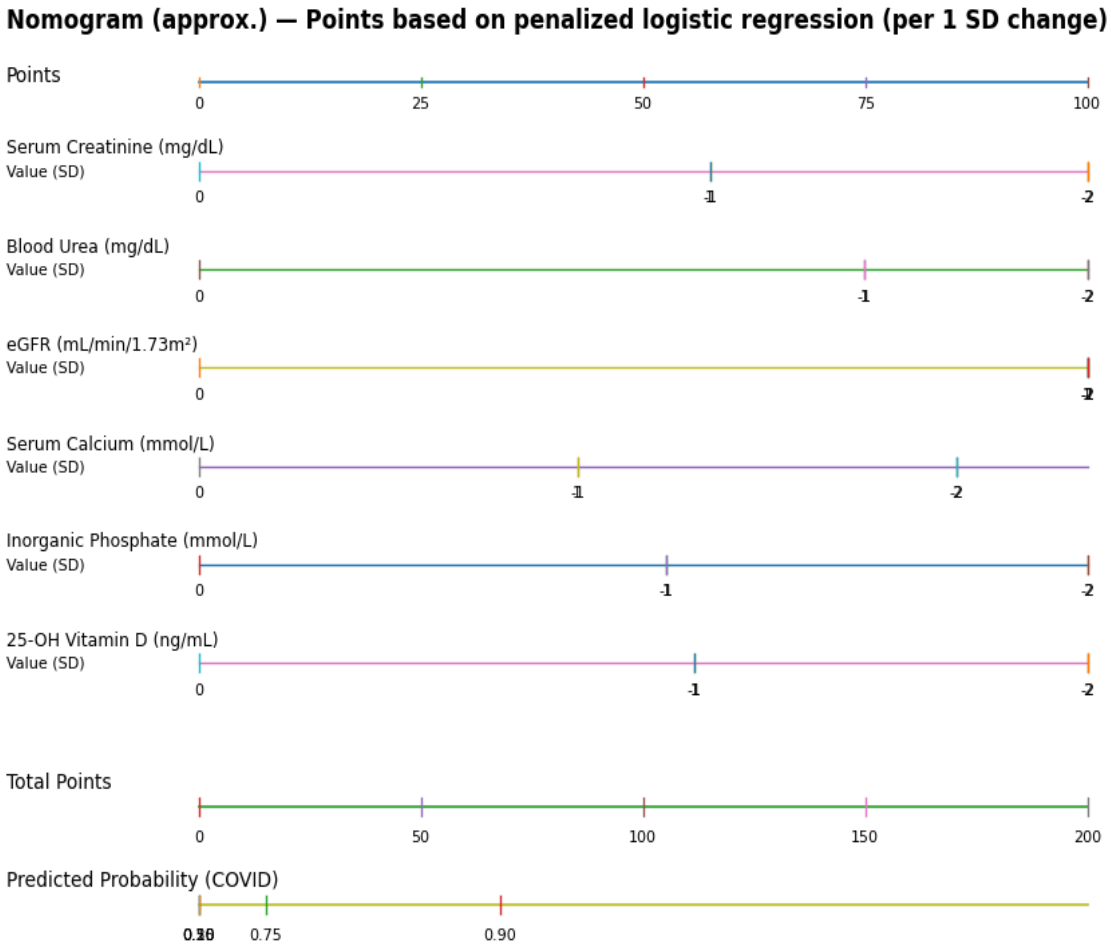

4. Discussion

It was a case -control study that assessed the parameters of renal function and mineral-metabolism biomarkers among 100 elderly patients with COVID-19 versus 100 healthy age-matched controls. These findings show a steady trend of renal dysfunction in the COVID-19 group, with much higher serum creatinine and urea concentrations and lower estimated glomerular filtration rate (eGFR). These anomalies were also seen to be accompanied by the reduced levels of serum calcium, the reduced level of inorganic phosphate, and the significantly decreased levels of 25-hydroxyvitamin D. Combined with other findings, these results confirm the existing evidence that SARS-CoV-2 infection constitutes a systemic disease where renal dysfunction and mineral metabolism disturbances are frequent, clinically significant, and especially abundant in older people.

The increase of creatinine and urea accompanied by a subsequent decrease of eGFR among older individuals with COVID-19 is biologically plausible and corresponds to recent data showing that acute kidney injury (AKI) and clinically significant renal dysfunction are high in hospitalized groups of people. Old age is always listed as a leading risk factor of AKI in COVID-19, probably because of the diminished renal reserve, stiffer vascularity, a greater number of comorbidities, and a greater tendency to hemodynamic unsteadiness in acute disease [21]. Recent critical-care research highlighted the issue of optimum fluid balance in patients with COVID-19, with both hypovolemia and hypervolemia potentially aggravating the situation of renal perfusion and tubular damage, especially in ICU facilities [21]. Even though we did not stage AKI specifically, the biochemical profile of the elderly patients implies impaired renal filtration even in non-critical-care settings.

The presence of chronic kidney disease (CKD) prior to COVID-related renal injury also makes the interpretation more complicated in the elderly populations. Hospital-based studies show that the AKI prevalence rate is much greater among patients with underlying CKD and higher with the progression of the CKD stage, and the combination of CKD and AKI is related to worse clinical outcomes [22],[23]. Although our study did not longitudinally evaluate baseline kidney functioning, the extent to which eGFR declined indicates that some subjects might have suffered an undiagnosed CKD which surfaced in acute infection. This interpretation is in line with literature that the COVID-19 could reveal latent renal vulnerability due to inflammatory injury, endothelial dysfunction, microvascular alterations, hemodynamic instability, and nephrotoxic exposures [24][25][21].

Multinational analyses of ICU across SARS-CoV-2 variant periods still indicate high AKI prevalence rates, and renal injury has been a common complication in severe COVID-19 irrespective of viral wave [24],[25]. Other cases also were not restricted to ICU admissions, however, the recurrent increase of creatinine and urea adds weight to the idea that the kidney

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is one of the key organ targets in the case of high-risk and older COVID-19 patients.

A significant clinical issue is related to renal risk in the long-term after COVID-19. There is now some emerging evidence that AKI and decreased kidney functioning during acute infection may be a predictor of negative renal outcomes, but the strength of these predictors is variable by population [26];[29]. According to follow-up studies, survivors of COVID-related AKI can have persistent renal impairment, which would lead to the necessity of continuous monitoring [26]. Reviews of long COVID and renal outcomes identify endothelial dysfunction, inflammation, microthrombi, and tubular injury as the mechanisms with the potential to cause future CKD, particularly in patients with hypertension or diabetes [27],[28]. In this regard, our results justify post-acute renal follow-up of elderly patients who have survived the COVID-19 infection, especially when the creatinine or eGFR abnormalities were recorded during the disease.

In addition to renal indices, this research found that serum calcium in older patients with COVID-19 was significantly lower. Hypocalcemia has been described as common among hospitalized COVID-19 and linked with more severity of the disease and increased respiratory support [30]. Despite the difference in the study design compared to severity-stratified hospital cohorts, the direction of association remains the same: the poorer the calcium levels the more characteristic the elderly patient with COVID-19. Hypocalcemia can be a sign of inflammatory cytokine release, changes in parathyroid hormone, vitamin D deficiencies, hypoalbuminemia, and renal tubule dysfunction in electrolyte regulation, in a mechanistic fashion. Since the aged are likely to be malnourished, have low albumin, and already be deficient in vitamin D, calcium should ideally be interpreted with albumin correction or ionized calcium, with magnesium, phosphate, and vitamin D being evaluated.

Other interesting results were lower inorganic phosphate. Phosphate plays an important role in ATP generation, diaphragm activity, and oxygen supply through the regulation of 2,3-diphosphoglycerate. Critically ill COVID-19 patients have been reported to experience hypophosphatemia and such has been associated with clinical outcomes [31]. Even though our subjects did not have a time limit set to work in the ICU setting, lower phosphate concentration in the elderly COVID-19 group may indicate that phosphate imbalance could be a component of the metabolic profile of acute infection. They might be caused by respiratory alkalosis, catecholamine stress, nutritional deficiency, and renal tubular losses. To prevent such complications as calcium-phosphate precipitation, phosphate replacement is needed when working with elderly patients with COVID-19, and clinical electrolyte monitoring is necessary.

This significantly reduced level of 25-OH vitamin D in COVID-19 group is consistent with the growing body of literature that indicates that vitamin D deficiency is associated with risk and poor outcomes of COVID-19. A meta-analysis of meta-analyses with reported associations between low vitamin D status and more severe infections and mortality was provided, and it recognized heterogeneity and the necessity of higher-quality trials [32]. A recent meta-analysis of supplementation recommended that vitamin D treatment has the potential to decrease mortality, and possibly more so in the older population [33]. Even though observational comparisons cannot be used to determine causality, our results have shown that vitamin D deficiency is prevalent in elderly COVID-19 patients and is associated with hypocalcemia and phosphate imbalances.

Such deficiency in vitamin D in older people could be an indicator of decreased sunlight, chronic illness, malabsorption, as well as acute inflammatory impact on binding proteins. Significantly, secondary hyperparathyroidism and bone-mineral disorders can be further aggravated by impaired renal function which further reduces the activation of vitamin D metabolites. Therefore, combined measurement of renal activity along with calcium, phosphate, and vitamin D gives more clinically relevant images as compared to the analysis of any type of biomarker.

One of the key advantages of the evaluation of multiple biomarkers is their discriminatory and prognostic ability. The current kidney outcome studies are favorable of combining renal indices with inflammatory and metabolic biomarkers to enhance risk stratification in COVID-19 [26]. Findings show that our study has shown invariably differentiated values of renal functioning parameters and mineral-metabolism markers which supports the notion of using a combination of biomarker panel in order to improve clinical evaluation and post-acute follow-up in old age populations where multimorbidity and baseline variability is the rule. The key strength of this study is the age-matched case–control design, which improves interpretability by anchoring biomarker differences against an elderly healthy reference rather than relying solely on within-hospital comparisons. The study also evaluates a clinically practical panel of tests widely available in routine care, facilitating translation to real-world monitoring.

The interpretation of the findings should be subject to limitations. First, the design is cross-sectional and cannot establish the presence of a temporal and causal relationship; there were no pre-infection baseline kidney function and vitamin D status. Second, there is residual confounding (e.g., undiagnosed CKD, dietary factors, seasonal variation in vitamin D, consumption of drugs and acute hydration condition). Third, albumin can affect calcium interpretation and ionized calcium measure was not stated. Fourth, the applicability of the findings to other areas could be restricted because of the practices of healthcare in the area and period of variants, however, multi-wave studies indicate that the risk of kidney is significant across variants [24],[25]. Long-term follow-up should be considered in future studies to identify whether the found abnormalities can predict long-term eGFR decrease or mineral-metabolism sequelae as it was indicated in long-term kidney outcome studies and long-COVID nephrology reviews [27][28][29].

Our clinical findings suggest that the attention to renal and mineral-metabolism checks should be carried out as the first priority in elderly patients with COVID-19, particularly those who have to be hospitalized. Early signs of high creatinine/urea and low eGFR must trigger close monitoring of fluids and medication, which is in line with critical-care advice on the renal danger of hypovolemia and fluid overload [21]. Simultaneous detection of hypocalcemia, hypophosphatemia, and vitamin D deficiency can provide information to support care and nutrition, as well as be used as indicators of systemic disease load. The research-wise, future studies need to test integrated biomarker models and test whether the correction of the modifiable factors like vitamin D deficiency influences the outcomes in unbiased randomized trials, and respect current mixed trial literature and heterogeneity highlighted in recent syntheses [32][33] .

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5. Conclusions

In this analytical case-control study, the elderly men who were evaluated eight months post-COVID-19 recovery had persistent renal dysfunction and severe disruptions in mineral metabolism in the absence of any chronic disease history. In post-COVID patients, the level of serum creatinine, blood urea, and eGFR were increased, and the result was an indication of continuous renal filtration impairment. Also, low serum calcium, phosphate and 25-hydroxyvitamin D levels indicated long-term mineral homeostasis disturbance. Post-COVID renal involvement was strongly predicted by the renal markers, particularly, eGFR and blood urea. These results demonstrate the significance of renal and metabolic follow-up during the long-term in elderly patients who have recovered following COVID-19.

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