Treatment Outcomes and Associated Clinical Laboratory Parameters among Patients Hospitalised with COVID-19 at a Private Hospital in Kiambu County, Kenya

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Abstract

Background: COVID-19 was a novel infection whose outcomes were not well established to be associated with any factors during the start of the pandemic that it caused globally. This research explored the relationship between clinical laboratory tests and mortality among patients hospitalized with a positive COVID-19 test at a tertiary hospital in Kiambu County, Kenya.

Methods: A retrospective analytical cross-sectional study was conducted among 154 COVID-19–positive patients admitted to a private tertiary hospital in Kiambu County, Kenya, to identify laboratory tests associated with mortality. Data was analysed using SPSS version 24, with descriptive statistics summarizing clinical and outcome variables. Binary logistic regression assessed associations between laboratory parameters and mortality, while significant factors were included in multivariate models to control confounders. Kaplan–Meier analysis evaluated time to death and discharge, with statistical significance set at $p < 0.05$.

Results: The mean age of patients admitted with a diagnosis of COVID-19 was 54.43(SD±17.1) years (54.43 years). Almost two-thirds of patients had comorbidities (n=93; 60.4%). In-hospital death was reported for 23.4% (n=36) of the investigated patients, and 76.6% (n=118) were discharged alive. Logistic regression revealed that the clinical laboratory parameter that predicted mortality was elevated C-reactive protein (adjusted odds ratio 1.067; 95% CI: 1.014–1.123; p=0.013).

Conclusion: Elevated C-reactive protein levels were identified as a significant predictor of mortality among hospitalised COVID-19 patients, highlighting the prognostic value of inflammatory markers in disease outcomes. Despite this, the majority of patients recovered and were discharged alive, suggesting generally favourable clinical outcomes in this setting.

Keywords: COVID-19, Clinical laboratory test, Mortality, Inflammatory markers, Critical care

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Introduction

In December 2019, pneumonia of unknown origin was discovered in 27 patients in Wuhan, China. Chinese authorities identified a new virus of the Coronaviridae family as the causative agent. The pathogen was a novel virus that was phylogenetically similar to the SARS-COV of 2003. Therefore, the new virus was named SARS-CoV-2 and the resulting disease COVID-19 [1]. Transmission occurs through respiratory droplets and direct contact. Clinical

symptoms of COVID-19 were fever, dry cough, fatigue, dyspnea, ageusia, and anosmia, or a combination of these symptoms [2]. COVID-19 could be asymptomatic, mild, moderate, or severe. A combination of respiratory failure, shock, multi-organ failure, and cytokine storm was a common complication in patients infected with SARS-CoV-2 [3]. Globally, clinical characteristics have been identified that determine the outcomes for patients with COVID-19. Research indicated that some laboratory findings (e.g., lymphopenia >40%, C-reactive protein >10 mg/L, procalcitonin >5 ng/mL, D-dimers >500 ng/mL, liver enzymes >35 U/L, and lactate dehydrogenase >280 U/L) were associated with poor outcomes [4]. In a Bellevue hospital in New York, 87 of 137 (59.9%) patients died after an average stay of 8 days in the critical care unit (CCU), 26 (19%) were discharged home, and 15 (10.9%) were discharged to a rehabilitation facility [5]. A South Korean study reported that the majority of patients admitted with asymptomatic or mild illness remained in status quo until discharge, 3.4% progressed to severe illness, and 1.1% died in hospital. The case fatality rate was 29.8% for patients with severe illness and 62.5% for patients in the intensive care unit [6]. In Africa, SARS-COV-2 arrived later than in most other parts of the globe. The case fatality rate was 2.1% which was against the prediction of the WHO's prediction that there would be higher mortality rates because of the less developed healthcare systems. However, this was not the case [7]. For instance, in the Democratic Republic of Congo, the overall in-hospital mortality rate was 13.2%. Independent risk factors for increased mortality included age below 20 years (compared with patients aged 20–39 years), obesity, and

[4]. In Kiambu County, as of March 2022, 19,778 COVID-19 cases and 120 deaths were reported, with Thika Sub-County being the epicenter [8]. Avenue Hospital Thika was one of the tertiary referral hospitals that admitted and treated these patients. However, data on clinical characteristics and outcomes among hospitalized patients in Kiambu County remained scanty. We therefore aimed to describe clinical characteristics and outcomes of hospitalized patients with COVID-19 in Avenue Hospital, Thika and to compare them with other African and non-African populations.

Materials and Methods

Study design and area

A retrospective analytical cross-sectional design was adopted, and data were abstracted from the patient files at Avenue Hospital, a private tertiary hospital in Thika, Kenya. This hospital serves a diverse population comprising patients from the urban middle to high socioeconomic classes. The hospital has general medical and surgical, critical, renal, oncology, pediatric, theatre, radiology, and pharmacy departments, and offers 24-hour outpatient, laboratory, and ambulance services.

The study participants consisted of adult patients admitted with COVID-19 between May 2020 and November 2021 at Avenue Hospital-Thika. Included in the study were medical records of patients aged 18 years and above with a confirmed diagnosis of COVID-19, as verified by polymerase chain reaction (PCR) testing performed at the hospital's laboratory accredited by the Kenya Accreditation Service (KENAS). Excluded were medical records of patients younger than 18 years and those without a PCR-confirmed diagnosis of COVID-19. The accessible population for this study consisted of 200 adult patients admitted with confirmed COVID-19 at Avenue Hospital, Thika, between May 2020 and November 2021. The minimum required sample size was computed using Yamane's (1967) simplified formula for proportions in finite populations, assuming a 95% confidence level ($\alpha = 0.05$) and a precision level (e) of 5%:

n=N/(1+N(e ^2)

Where:

N=200 (accessible population size),

e=0.05 (desired level of precision corresponding to a 95% confidence level).

n=200/1+200(0.05) 2

n=200/1+0.5

=133.3

Thus, the minimum required sample size was 133 participants.

To enhance precision and account for unusable or incomplete medical records, we incorporated an expected unusable rate (r) of 15%, consistent with prior retrospective hospital-based studies in Kenya and similar low and middle-income settings where missing or incomplete clinical data ranged from 10–20% [4, 23]. The adjusted sample size was therefore calculated as follows:

nadj=n/(1-r)

=133.3/(1-0.15)

=133.3/0.85

=156.8

Rounding down to the nearest feasible whole number, the target sample size was set at 154 participants.

This oversampling ensured adequate statistical power

$(1 - \beta = 0.80)$ to detect moderate effect sizes at a significance level of $\alpha = 0.05$, while maintaining subgroup representativeness (e.g., sex, presence of comorbidities). The finite population correction was inherently applied in the Yamane equation since the total sampling frame (N = 200) was known.

Accordingly, all 154 eligible participants with complete records were included in the final analysis to maximize precision, minimize sampling error, and allow subgroup analyses of clinical laboratory parameters and outcomes.

Study tools

The study employed a data abstraction tool for comprehensive data collection. The tool was developed by the authors to collect clinical characteristics of the patients and outcomes of care. The tool had three parts. Part one was to capture participants' demographic data. Part two captured laboratory results, including lymphocyte count, C-reactive protein, procalcitonin, D-dimer, liver function tests, serum ferritin levels, and radiological findings. While the third part captured patient outcomes, categorized as either death or discharge.

Quality assurance was ensured through both pretesting and expert evaluation of the data abstraction tool. The tool was pretested using 5% of patient files (n=10) from the same tertiary hospital to assess its clarity, completeness, and consistency, following methodological guidance on appropriate pilot study proportions. Face validity of the abstraction tool was ensured by structuring the instrument into three parts: demographics, clinical characteristics, and outcomes of care. Construct validity was ensured by checking items in the data collection instruments against study objectives to ensure all constructs under study were measured. An alpha Cronbach of 0.715 was achieved for the internal consistency of the tool. The reliability and internal consistency analysis were performed using SPSS version 24.

Data collection procedure

Data was collected by the principal investigator (PI) and trained research assistants through abstraction of data from the medical records using the data abstraction tool, in Google Forms format. The data captured included patients' demographic details, medical history, pre-existing conditions, clinical symptoms, laboratory findings, and patient outcomes. Data collection was conducted from 1st February 2022 to 29th July 2022. After completion, the questionnaires were checked for completeness.

Data analysis

Data were coded and thereafter entered into SPSS version 24, where descriptive statistics were implemented to evaluate clinical laboratory parameters and patient outcomes. The association between laboratory parameters and outcomes related to COVID-19 was modelled through binary logistic regression. Significant determinants were entered into a multivariate analysis to control for confounders and generate

adjusted odds ratios (AOR). Time to death and time to discharge were analysed using Kaplan-Meier. Statistical significance was determined at $p<0.05$.

Ethical considerations

Ethical approval was granted by the Nairobi Hospital Bioethics and Research Committee, reference number TNH-ERC/DMSR/RP/018/21. Permission to access patient records was also sought from the hospital administration. Confidentiality was maintained by adhering to the recommendations outlined in the Helsinki Declaration and the Belmont Report. Directives from the Kenyan government on the prevention of transmission of COVID-19 were followed during this study.

Results

Sociodemographic characteristics

As shown in Table 1, the mean age of the patients admitted to the facility with a diagnosis of COVID-19 was 54.43(SD±17.1) years.

Table 1:

Socio-demographic characteristics of patients admitted with COVID-19

Variables		Frequency (n)	Percent (%)
	Age	N=154 Mean=54.4±17.1	
Gender	Female	76	49.4
	Male	78	50.6
County of Residence	Nairobi	20	13
	Machakos	2	1.3
	Murang'a	15	9.7
	Kitui	1	0.6
	Nyeri	4	2.6
	Nakuru	1	0.6
	Kiambu	111	72.1
The main source of income	Others	84	54.5
	Agriculture	16	10.4
	Finance	24	15.6
	Healthcare	11	7.1
	Security	4	2.6
	Education	8	5.2
	Transport	5	3.2
	Hospitality	2	1.3
Co-morbid patient	Yes	58	37.7
	No	96	62.3
Specific Co-morbidity	None	61	39.6
	Diabetes	27	17.5
	Hypertension	31	20.2
	Diabetes & Hypertension	30	19.5
	Cancer	2	1.3
	Renal disease	3	1.9

Patients' outcomes

Of the 154 patients included in the study, 36 (23.4%) died, corresponding to a mortality rate of 234 per 1,000 patients admitted with COVID-19. The remaining 118 patients (76.6%) were discharged during the study period. Figure 1.

Time to death

Thirty-six patients (23.4%) died within 32 days of admission. The median survival time was 22 days (95% CI: 16.626 to 27.374), indicating that by the 22nd day of admission, half of the patients died due to the infection. Table 2 and Figure 2 summarize this information.

Clinical laboratory characteristics

All patients received several tests, and the lowest and highest measures for each patient were recorded. The highest individual lymphocyte count was 7.8, the highest mean level was 1.6 (95%CI: 1.4–1.8), and the lowest was 0.5 (95%CI: 0.41–0.59). Table 3 presents these results.

Bivariate analysis of clinical laboratory characteristics and mortality

The bivariate analysis presented in Table 4 indicates that a one-unit increase in C-reactive protein increased the odds of mortality by 0.7% (highest: crude odds ratio [COR] 1.007, 95% CI: 1.003–1.011, p=0.001; lowest: COR 1.007, 95% CI: 1.001–1.013, p=0.015).

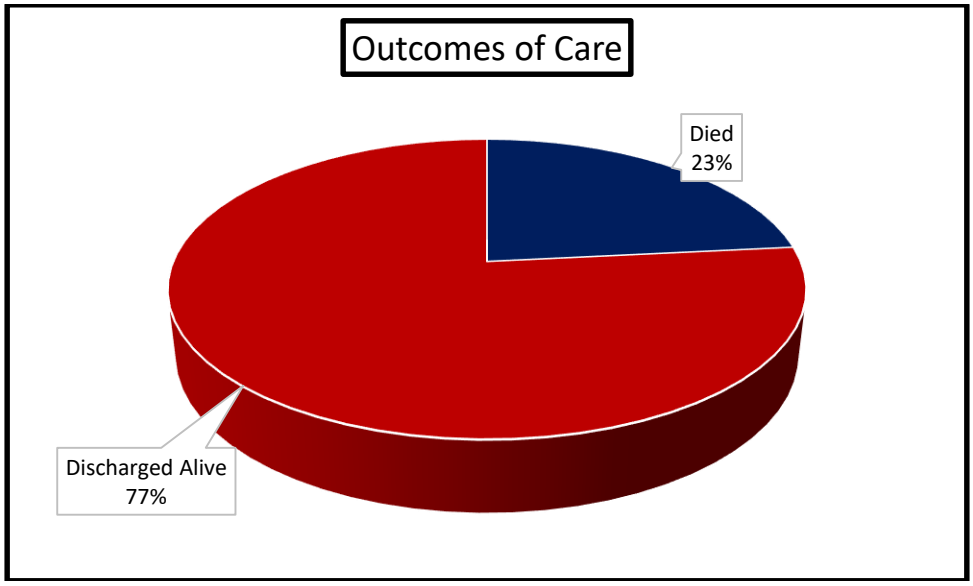

Figure 1:
Outcomes of Care among the Participants

Table 2:
Mean and Median Survival Time

Mean				Median			
Estimate	Std. Error	95% CI		Estimate	Std. Error	95% CI	
		Lower Bound	Upper Bound			Lower Bound	Upper Bound
20.96	1.486	18.049	23.872	22	2.742	16.626	27.374

Estimation is limited to the largest survival time if it is censored.

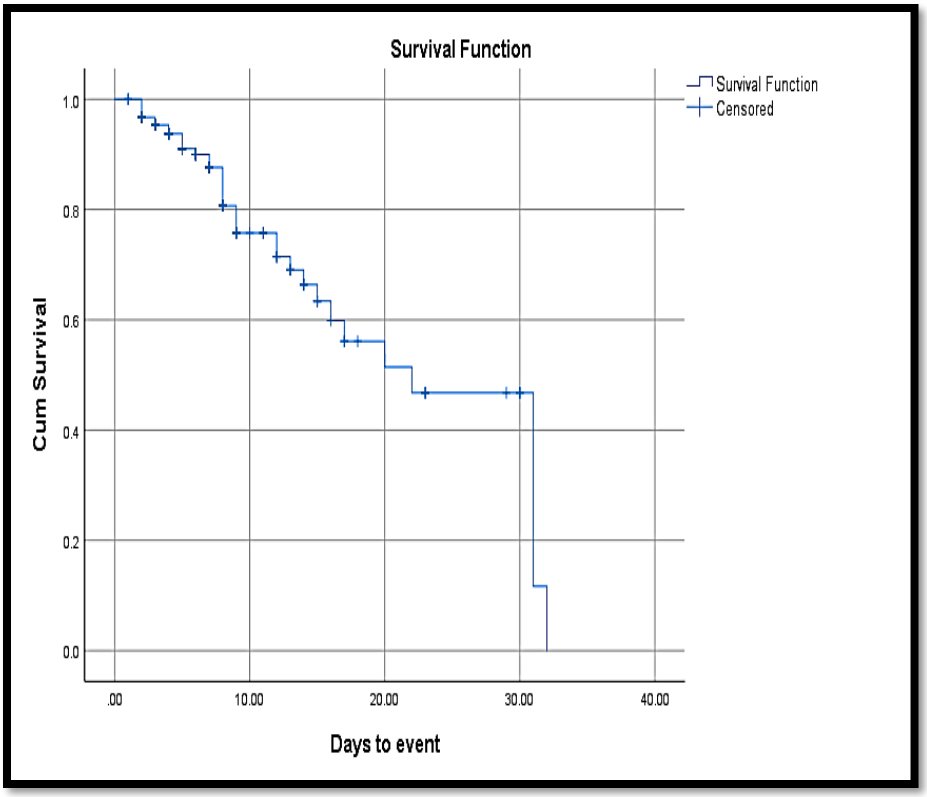

Figure 2:
Survival Function Time Based on Kaplan Meier Statistics

Table 3:
Patients' Clinical Laboratory Characteristics

		N	Maximum	Mean	95% CI	
					Lower	Upper
Lymphocyte (1.2–4 × 10^9/L)	Highest	154	7.800	1.588	1.411	1.7634
	Lowest	154	2.47	0.502	0.410	0.593
C-reactive protein (mg/L)	Highest	145	300	137.370	123.360	150.780
	Lowest	152	300	31.652	22.290	41.020
Pro-calcitonin (ng/mL)	Highest	150	100	4.8747	1.980	7.646
	Lowest	148	8.67	0.250	0.102	0.397
D-dimer (ng/mL)	Highest	145	10000	2497.980	1963.720	3032.340
	Lowest	154	9384	436.307	253.790	618.830
Alanine transaminase (U/L)	Highest	154	754	51.829	37.470	66.190
	Lowest	154	412.7	17.514	10.550	24.470
Aspartate transaminase (U/L)	Highest	154	854	51.958	38.160	65.750
	Lowest	154	139.200	14.397	10.550	18.240
Alkaline phosphatase (U/L)	Highest	154	377	74.371	62.310	85.430
	Lowest	154	246.600	28.281	20.850	35.710
Glutamyl transferase (U/L)	Highest	154	1018.600	106.836	80.550	133.120
	Lowest	154	618.500	40.375	26.350	54.340
Lactate dehydrogenase (ng/mL)	Highest	154	1901	227.984	178.900	277.070
	Lowest	154	919	42.028	19.540	64.518
Serum ferritin (ng/mL)	Highest	151	2000	359.183	272.480	445.880
	Lowest	154	960	19.461	1.340	37.580

Key: ng/mL=nanogram per milliliter, U/L=Units per liter, mg/L=milligrams per liter

Multivariate analysis of clinical laboratory characteristics and mortality

After accounting for confounders, the only factor that predicted mortality was C-reactive protein (AOR 1.067, 95%CI: 1.014–1.123; p=0.013). This implies that patients who had a decrease in C-reactive protein were 6.7% more likely to survive as compared to those with higher C-reactive protein levels. Table 5

Table 4:
Bivariate Analysis of Clinical Characteristics and Mortality

		Sig.	COR	95% CI	
				Lower	Upper
Lymphocyte	Highest	0.649	0.922	0.648	1.31
	Lowest	0.803	0.919	0.475	1.778
C-reactive Protein	Highest	0.001	1.007	1.003	1.011
	Lowest	0.015	1.007	1.001	1.013
Pro-calcitonin	Highest	0.143	1.014	0.995	1.034
D-dimer	Highest	0.004	1.000	1.000	1.000
	Lowest	0.099	1	1	1.001
Alanine transaminase	Highest	0.218	1.002	0.999	1.006
	Lowest	0.336	1.004	0.996	1.011
Aspartate transaminase	Highest	0.13	1.003	0.999	1.008
	Lowest	0.003	1.023	1.007	1.038
Alkaline phosphatase	Highest	<0.001	1.016	1.009	1.023
	Lowest	<0.001	1.019	1.010	1.027
Glutamyl transferase	Highest	0.002	1.004	1.001	1.006
	Lowest	0.072	1.004	1	1.007
Lactate dehydrogenase	Highest	0.001	1.002	1.001	1.003
	Lowest	0.015	1.003	1.001	1.005
Serum ferritin	Highest	0.044	1.001	1.000	1.001
	Lowest	0.053	1.003	1	1.007

Note: Sig. = significance, COR=crude odds ratio.

Table 5:
Multivariate Analysis of Clinical Laboratory Characteristics and Mortality

	B	SE	Wald	df	Sig.	AOR	95%CI	
							Lower	Upper
C-reactive protein								
Highest	−0.004	0.007	0.287	1	0.592	0.996	0.984	1.010
Lowest	0.065	0.026	6.124	1	0.013	1.067	1.014	1.123
Aspartate transaminase								
Lowest	0.054	0.047	1.316	1	0.251	1.055	0.963	1.157
Alkaline phosphatase								
Highest	0.028	0.018	2.227	1	0.136	1.028	0.991	1.066
Lowest	−0.034	0.022	2.278	1	0.131	0.967	0.926	1.010
Glutamyl transferase								
Highest	0.003	0.004	0.567	1	0.451	1.003	0.995	1.011
Lactate dehydrogenase								
Highest	0.005	0.004	1.283	1	0.257	1.005	0.997	1.013
Lowest	−0.011	0.011	0.884	1	0.347	0.989	0.968	1.012
Serum ferritin								
Highest	−0.001	0.001	0.712	1	0.399	0.999	0.996	1.002

Key: df=degrees of freedom, Sig.=significance, AOR=adjusted odds ratio, SE=standard error.

Discussion

The mortality rate in this study was 234/1000 patients, as 23.4% of the analysed patients died in the hospital. This was comparable to a study carried out in a tertiary teaching hospital in Seoul, South Korea, where the fatality rate was 29.8% [6]. However, the mortality rate in this study was low when compared with findings from Colombia (mortality rate, 51%) [8]. This could be attributable to the larger proportions of patients with COVID-19 with advanced age and comorbid conditions in that study, compared with our study. When compared to data from African settings, the mortality rate in the current study was slightly higher than that reported in Nigeria (16.3%) [9] and Ethiopia (19.4%) [10], but lower than in South Africa (37%) [11]. These variations may be explained by differences in patient demographics, access to critical care resources, and the timing of the pandemic waves in the respective countries. Furthermore, 118 (76.6%) patients were discharged alive in our inquiry, which differed from the results from Saudi Arabia, where 85% of admitted patients were discharged alive [12]. The difference between the two studies may be attributable to the severity levels of COVID-19 infection among patients.

We found various elevated laboratory parameters, including associated lymphocytosis, transaminitis, and elevated C-reactive protein. Additionally, the elevated CRP was associated with an increased risk of death. In the current study, the mean lymphocyte level was 1.6×10^9/L, higher than the mean levels reported in two studies from China (0.31×10^9/L and 1.36×10^9/L) [13,14]. These differences could be attributable to later discoveries of effective therapies compared with the onset of the pandemic, as reflected in other studies [13,14]. Nonetheless, the findings of this study are in congruence with findings by Ombajo in Kenya, who reported increased incidences of lymphocytosis that were associated with increased odds of death from COVID-19 disease [4]. In the current study, the mean C-reactive protein level was 137.37 mg/L, which differed from a study conducted at a university hospital in the US, where the mean was 130 mg/L [15]. Differences in the application of C-reactive protein as a treatment-guiding inflammatory marker may explain these discrepancies. Conversely, these findings are in agreement with findings by Loice, who found that most of the patients admitted in Kenya had elevated CRP levels that were then associated with increased risk of mortality. In addition, the mean measure of aspartate transaminase in this study was 52 U/L, whereas a study from China reported a lower mean of 29 U/L [16]. The average alkaline phosphatase level was 377 U/L, and that of gamma-glutamyl transferase was 106.84 U/L. These results resonate with findings by Nachega in Congo, where patients admitted due to COVID-19 had elevated levels of alkaline phosphatase [8]. However, a study conducted in Italy reported lower levels (mean alkaline phosphatase 47.9 U/L, mean gamma-glutamyl transferase 104.8 U/L) [16]. These differences suggest the possibility of liver injury caused by aggressive new therapies among patients with COVID-19. The mean level of lactate dehydrogenase in our study was 1901 U/L, which was higher than that reported in a study conducted in China (426 U/L) [17]. These results indicate that different types of comorbidities may lead to different complications.

The bivariate analysis showed that a one-unit increase in C-reactive protein increased the odds of mortality by 0.7%, which was consistent with findings from Turkey that indicated an association between C-reactive protein and a severe form of the disease; furthermore, C-reactive protein was an important predictor of mortality [18]. Additionally, these findings align with those of Ombajo in Kenya, where elevated CRP and transaminases predicted mortality. These results could suggest damage to multiple organs in such patients due to severe systemic inflammation. In this study, an increase in the

aspartate transaminase level increased the odds of dying by 2.3%. This corroborated the findings of a study from China [19]. Other liver enzymes that were significantly associated with mortality in this study were alkaline phosphatase and gamma-glutamyl transferase, which supported results from the US and Turkey [20,21]. The consistent findings linking elevated liver enzyme levels with increased mortality suggest that progressive deterioration of liver function may precipitate complications such as hepatic encephalopathy and hepatorenal syndrome, which can ultimately result in death.

We observed an association between elevated lactate dehydrogenase levels and mortality. A pooled analysis study of hospitalized patients with COVID-19 from the US reported a similar result [22]. This suggests that viral infections may cause elevated serum lactate dehydrogenase (LDH) levels, likely as a result of tissue damage in organs such as the lungs, which are affected by the virus. However, these findings are dissimilar to those of Loice, who found that elevated lactate dehydrogenase was not a positive predictor of death among patients admitted with COVID-19 in Kenya.

Increased serum ferritin levels were also associated with a higher risk of mortality, which contrasts with a study conducted in the United States of America among patients with Sickle-cell anemia that showed no link between mortality and ferritin levels [23]. However, a study from Pakistan also revealed that high ferritin levels increased the odds of mortality rates [24]. These results suggest that sickle cell disease could have protective features in the pathology of COVID-19; however, in its absence, ferritin could be indicative of liver damage or high inflammation caused by the virus. Conversely, in patients without sickle cell disease, elevated ferritin could serve as a marker for significant liver damage triggered by the virus, reflecting a heightened risk of adverse outcomes.

Limitations

This study has several important limitations.

- First, the retrospective design meant reliance on existing medical records, which may have been incomplete, inconsistently documented, or missing key clinical details, introducing possible information bias.
- Second, as the study was conducted in a single private tertiary hospital serving predominantly urban and middle-to-high socioeconomic populations, the findings may not be fully generalizable to patients managed in public hospitals or rural settings.
- Third, although the sample size was adequate for analysis, it was relatively small compared to multi-center studies, limiting the ability to detect weaker associations and perform more granular subgroup analyses.
- Fourth, some potentially relevant variables, such as treatment regimens, illness severity at admission, vaccination status, and oxygen or ventilatory support, were not consistently available and therefore could not be included in the analysis.
- Finally, the study period coincided with a rapidly evolving pandemic in which both treatment protocols and viral variants changed over time, which may have influenced patient outcomes and laboratory characteristics.

Despite these limitations, the study provides valuable insights into the clinical characteristics and outcomes of hospitalized COVID-19 patients in Kiambu County and contributes to the broader understanding of the disease in African settings.

Conclusion

The COVID-19 mortality rate in this study was 23.4%. Systemic inflammation was observed in patients with elevated levels of C-reactive protein. After controlling for confounders, CRP was associated with mortality among patients with COVID-19.

Recommendations

Based on the study findings, routine measurement of C-reactive protein (CRP) should be incorporated into the initial assessment of patients admitted with COVID-19, as elevated CRP levels are a significant predictor of mortality. Patients presenting with markedly elevated CRP levels should be prioritised for closer monitoring and early intervention to improve survival outcomes. Furthermore, integrating CRP as an inflammatory marker into hospital treatment protocols may enhance risk stratification and guide clinical decision-making. Furthermore, multicenter studies should be conducted to validate these findings and explore the predictive value of CRP alongside other inflammatory markers across diverse healthcare settings in Kenya.

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Author contribution

- Gabriel N, Newton O, Christopher O, & Robert N: Conceptualisation, methodology, investigation, data collection, original draft, review & editing writing, project administration & manuscript writing.
- Dr Elijah M: methodology, formal analysis, review & editing of writing.
- Dr Grace W & Dr Albanus M: Manuscript review, review & editing of writing

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