Can Ferritin and Interleukin-6 Levels Prognosticate Patients with COVID-19

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Background and study aim: During the coronavirus disease 2019 (COVID-19) pandemic efforts were focused on identifying biomarkers that predict disease severity and clinical outcomes. Biomarkers, including ferritin and interleukin-6 (IL-6), have been implicated in contributing to worsening prognosis. However, there is limited data on their prognostic utility in African populations. This study aimed to determine whether serial ferritin and IL-6 measurements could serve as prognostic biomarkers for COVID-19 outcomes in a South African quaternary referral hospital.

Patients and Methods: A retrospective analysis of 125 patients (≥12 years) with confirmed COVID-19 infection, admitted to Inkosi Albert Luthuli Central Hospital from January to December 2020, was conducted. Demographic, clinical, and laboratory data were extracted from electronic medical records. Patients were stratified based on outcome (alive vs. demised). Statistical analyses included binary logistic regression, the Kruskal-Wallis test, and correlation analyses to evaluate associations between serial measurements of ferritin and IL-6 levels, comorbidities, and mortality risk.

Results: The cohort was predominantly female (64%), with a median age of 45.1 years (IQR: 32.0–56.0). The most prevalent comorbidities were hypertension (48.0%), diabetes mellitus (23.2%), and chronic kidney disease (CKD) (31.2%). Hypertension was associated with reduced odds of mortality ($p = 0.031$, OR = 0.228), while CKD significantly increased mortality risk ($p = 0.001$, OR = 8.768). Ferritin and IL-6 levels were significantly elevated in the demised group, with ferritin increasing from 2,127.32 ng/mL (baseline) to 10 020.2 ng/mL (endpoint) ($p < 0.001$) and IL-6 rising from 443.2 pg/mL to 821.03 pg/mL ($p < 0.001$). In contrast, alive patients exhibited stable or decreasing levels of both markers at the endpoint (ferritin: 563.96 ng/mL to 336.29 ng/mL; IL-6: 97.24 pg/mL to 18.1 pg/mL, $p < 0.001$). Kruskal-Wallis analysis and Receiver Operator Curve analysis confirmed significant associations between ferritin, IL-6 levels, and outcomes.

Conclusion: This study provides evidence that serial ferritin and IL-6 measurements can serve as prognostic indicators in COVID-19 patients. Findings suggest that ferritin may serve as a surrogate marker for IL-6, potentially offering an alternative for risk stratification. Further prospective studies are warranted to explore the clinical applicability of these markers across diverse populations and conditions.

INTRODUCTION

Coronaviruses have previously caused severe outbreaks, most notably the 2002–2003 SARS epidemic, which exhibited high mortality due to severe acute respiratory syndrome coronavirus-1 (SARS-CoV-1) [1]. In December 2019, a novel coronavirus, severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), emerged in Wuhan, China, leading to a global pandemic officially declared by the World Health Organization (WHO) on March 11, 2020 [1,2,3]. The virus demonstrated genomic similarity to SARS-CoV-1, but with enhanced transmissibility, contributing to its rapid global spread [2]. South Africa confirmed its first cases on March 5, 2020 [4].

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Initially thought to be of zoonotic origin, SARS-CoV-2 was later found to be predominantly transmitted via respiratory droplets, aerosols, and contaminated surfaces [3]. Its high infectivity compared to SARS-CoV-1 is linked to structural differences in viral proteins and viral load kinetics [1,2]. The virus binds to the angiotensin-converting enzyme 2 (ACE2) receptor, which is expressed in respiratory, intestinal, renal, and vascular endothelial cells, facilitating widespread infection [1,2,3]. Clinical manifestations vary from mild symptoms such as fever, cough, and anosmia to severe complications including acute respiratory distress syndrome (ARDS) and multi-organ failure, with higher mortality in older individuals, males, and those with comorbidities [2,4].

Progression to severe disease is associated with viral migration to the lower respiratory tract, where SARS-CoV-2 infects type II alveolar epithelial cells, triggering an inflammatory cascade. This immune response is characterized by the release of various cytokines, including macrophage-derived inflammatory mediators [1]. A subset of patients exhibits immune dysregulation marked by sustained lymphocyte and macrophage activation, culminating in a hyperinflammatory state known as the "cytokine storm" [6]. Additionally, macrophage activation syndrome (MAS) has been documented in COVID-19 patients, as evidenced by a distinct cytokine profile and hyperferritinemia [7].

Hyperferritinemia and iron overload have been implicated in the exacerbation of the inflammatory response via the generation of reactive oxygen species (ROS) [8]. Multiple studies have demonstrated that elevated ferritin and interleukin-6 (IL-6) levels serve as prognostic biomarkers, aiding in risk stratification and clinical outcome prediction in COVID-19 patients. However, there remains a paucity of local data in South Africa to validate these findings, particularly given the high prevalence of comorbidities and demographic variations that may influence disease outcomes in this population.

This study aims to evaluate the prognostic utility of ferritin and IL-6 levels in predicting disease severity and clinical outcomes in COVID-19 patients within the South African healthcare setting.

PATIENTS AND METHODS

Study design

This retrospective chart review was conducted following ethical approval from the University of KwaZulu-Natal Biomedical Research Ethics Committee (BREC) and the Provincial Health Research and Ethics Committee (PHREC), under project number KZ202412003. The study involved a systematic analysis of computerized medical records from a database of COVID-19 polymerase chain reaction (PCR) test-positive patients who were admitted to Inkosi Albert Luthuli Central Hospital between January 1 and December 31, 2020.

Data collection:

A total of 125 patients aged 12 years and older were included from an initial dataset of 1,215 patients. Patients were excluded if they lacked serial ferritin and interleukin-6 (IL-6) measurements from admission to either discharge or death or if they had active pulmonary Mycobacterium tuberculosis infection. A significant proportion of excluded cases were healthcare workers who had tested positive but were not admitted due to mild disease or isolation at an alternative facility.

Key data points—including demographics, laboratory results, clinical outcomes, symptom duration, comorbidities, and serial oxygen saturation measurements—were extracted directly from hospital electronic medical records. The data were securely transcribed onto a password-protected Microsoft® Excel document. To maintain patient anonymity, hospital numbers were used as unique identifiers. Patients were stratified into two groups based on their clinical outcomes.

Statistical Analysis

Data analysis was conducted using IBM SPSS Statistics version 29.0, with guidance from a biostatistician. Categorical data were summarized using graphs, cross-tabulations, and frequency distributions. To assess relationships between categorical variables, Chi-square tests were applied, while correlation analyses were performed to explore associations between clinical markers.

To identify predictors of clinical outcomes, binary logistic regression models were employed. The models incorporated significance

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testing, with a p-value of 0.05 used as the threshold for statistical significance. Further evaluation of predictive accuracy was conducted using Receiver Operating Characteristic (ROC) curve analysis.

Continuous data were visualized using bar plots with error bars, expressed as mean ± standard deviation. The Wilcoxon Signed Ranks test and two-tailed t-tests were used to compare related datasets that were not normally distributed. Additionally, to analyze the distribution of continuous variables across different clinical outcomes, the Independent-Samples Kruskal-Wallis Test was applied.

RESULTS

The demographic profile of the study population is summarized in Table 1. The majority of patients were of Black ethnicity (n = 100, 80%), followed by Indian (n = 21, 16.8%) and White (n = 4, 3.2%).

Patient age exhibited a relatively wide distribution, with an interquartile range (IQR) of 32.0 to 56.0 years and a median age of 45.1 years.

A statistically significant difference in sex distribution was observed (p = 0.002), with a predominance of female participants (n = 80, 64%) compared to males.

Table 1: Master Mix preparation for 100 reactions (1st batch) Table (1): Demographic characteristics

Ethnicity	**Count**	**Percent**	**P-values**
Black	100	80.0	
Indian	21	16.8	<0.001
White	4	3.2	
Age (years)			
Median (IQR)	45.1 (32.0-56.0)		
Sex			
Female	80	64.0	0.002
Male	45	36.0	

Table 2 summarizes the prevalence of comorbidities among the study cohort. Diabetes mellitus (23.2%), hypertension (48.0%), and chronic kidney disease (31.2%) were the most common conditions. Statistically significant associations (p < 0.001) were observed for most comorbidities, except for hypertension (p = 0.655). Retroviral disease was present in 23.4% of patients, with 10.7% having an unsuppressed viral load. No cases of active tuberculosis were recorded.

Table (2): Comorbid conditions

		Count	Percent	P-values
Diabetes Mellitus	No	96	76.8	< 0.001
	Yes	29	23.2	
Hypertension	No	65	52.0	0.655
	Yes	60	48.0	
Chronic Kidney Disease	No	86	68.8	<0.001
	Yes	39	31.2	
Chronic Obstructive Pulmonary Disease	No	119	95.2	<0.001
	Yes	6	4.8	
Coronary Artery Disease	No	122	97.6	<0.001
	Yes	3	2.4	
Valvular Heart Disease	No	119	95.2	<0.001
	Yes	6	4.8	
Pregnant	No	73	91.3	<0.001
	Yes	7	8.7	
Retroviral Disease	No	96	76.4	<0.001
	Yes	29	23.6	
Antiretroviral Therapy usage	No	96	76.8	<0.001
	Yes	29	23.2	
Viral Load	Suppressed	25	89.3	<0.001
	Unsuppressed	3	10.7	
Tuberculosis	No	125	100.0	<0.001
	Yes	0	0	

Assessing the impact of comorbidities on COVID-19 outcomes is critical for risk stratification and clinical decision-making. Several conditions were found to significantly influence mortality risk. Chronic kidney disease (p = 0.001, OR = 8.768) was strongly associated with increased mortality, while each unit increase in BMI correlated with an 8% increase in mortality risk (p = 0.011, OR = 1.079). Interestingly, hypertension was associated with reduced odds of mortality (p = 0.031, OR = 0.228), suggesting a possible protective effect.

Although diabetes mellitus demonstrated an elevated odds ratio (OR = 2.881) for mortality, the association did not reach statistical significance (p = 0.086), indicating a potential but inconclusive risk. Neither age (p = 0.377, OR = 0.984) nor

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sex (p = 0.147, OR = 2.309) emerged as significant predictors of mortality. However, ethnicity showed a strong association with mortality (p < 0.001, OR = 8.919), underscoring the importance of demographic factors in disease progression. (Table 3)

Table (3): Relationship between comorbidities and outcomes

Variable	Unadjusted Exp(B)	Unadjusted 95% CI (Lower-Upper)	Unadjusted P-value	Adjusted Exp(B)	Adjusted 95% CI (Lower-Upper)	Adjusted P-Value
BMI	1.051	0.997-1.109	0.066	1.079	1.018-1.144	0.011
Diabetes Mellitus	2.304	0.815-6.517	0.116	2.881	0.862-9.626	0.086
Hypertension	0.457	0.153-1.364	0.16	0.228	0.059-0.872	0.031
Chronic Kidney Disease	6.148	1.939-19.489	0.002	8.768	2.308-33.303	0.001
Chronic obstructive Pulmonary disease	5.535	0.880-34.809	0.068	2.981	0.376-23.626	0.301
Valvular Heart Disease	1.051	0.107-10.321	0.966	1.757	0.170-18.189	0.636
Ethnicity Group	-	-	-	8.919	2.484-323.024	<0.001
Age	-	-	-	0.984	0.951-1.019	0.377
Sex	-	-	-	2.309	0.745-7.155	0.147

Table 4 & Figures 1 and 2 illustrate the temporal changes in ferritin and interleukin-6 (IL-6) levels from baseline to endpoint in both survivors and non-survivors. In the demised group, both ferritin and IL-6 levels increased significantly over time (p < 0.001), peaking at endpoint (ferritin: 10,020.2; IL-6: 821.03). Conversely, in the alive group, ferritin levels remained stable or declined (p < 0.001), while IL-6 levels significantly decreased over time (p < 0.001).

Baseline ferritin and IL-6 levels were significantly higher in the demised group compared to survivors (p < 0.001), with a substantial rise in inflammatory markers observed in those who did not survive. These findings suggest a strong correlation between elevated ferritin and IL-6 levels and poor prognosis in COVID-19 patients.

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Figure (1). Longitudinal Changes in Interleukin-6 Levels Stratified by Outcomes (Alive vs. Demised)

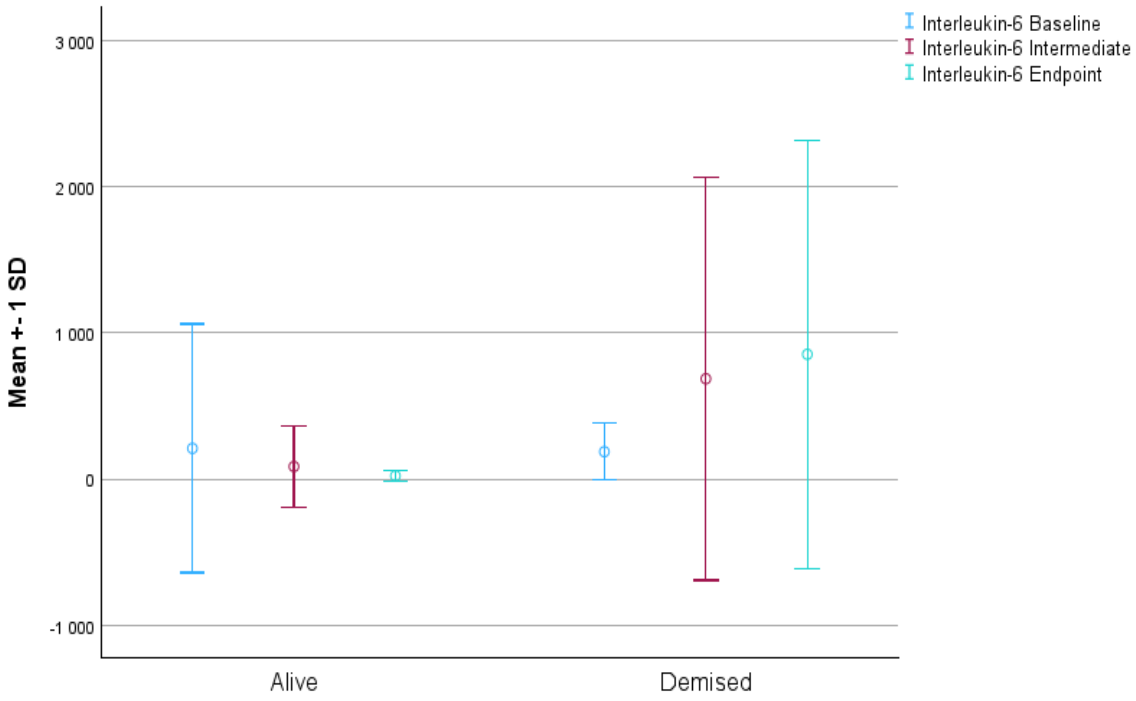

Figure (2). Longitudinal Changes in Ferritin Levels at Three Time Points Stratified by Outcomes (Alive vs. Demised)

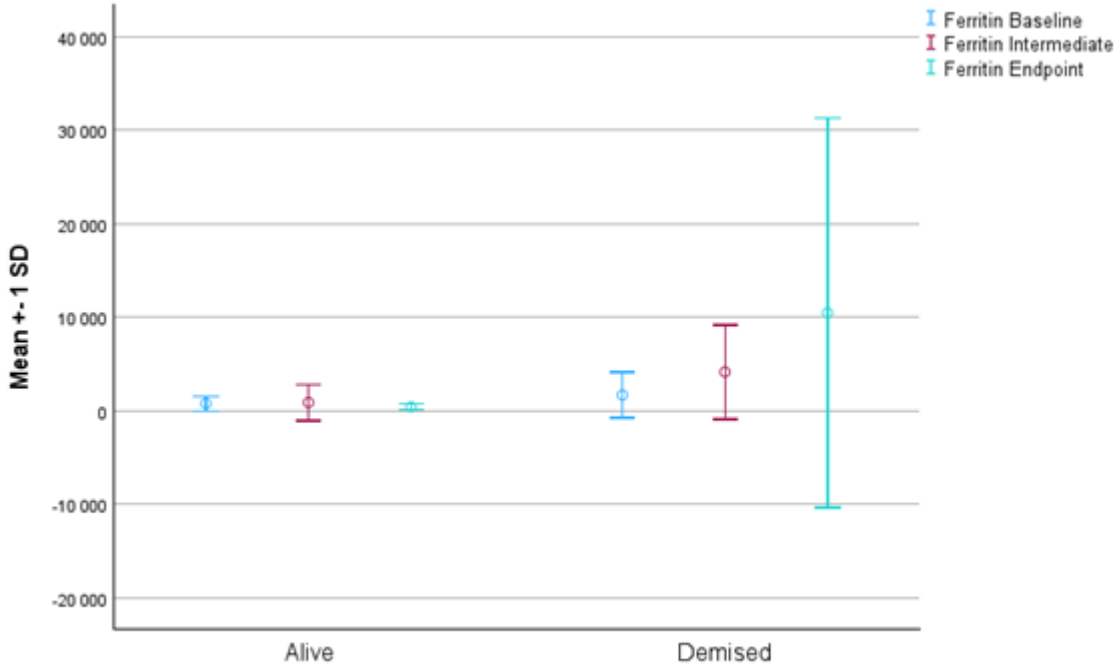

Table (4): Mean Interleukin-6 and Ferritin Levels at Baseline, Intermediate, and Endpoint Stratified by Outcomes (Alive vs. Demised)

	Demised	**Alive**
Interleukin -6 Baseline	443.26	97.24
Interleukin -6 Intermediate	737.64	85.24
Interleukin -6 Endpoint	821.03	18.1
Ferritin Baseline	2127.32	563.96
Ferritin Intermediate	4054.92	889.9
Ferritin Endpoint	10020.2	336.29

Figures 3 and 4 illustrate the predictive value of ferritin and IL-6 levels (baseline, intermediate, and endpoint) in determining the outcome, "Demised". The Area Under the ROC Curve (AUC) indicated that for Ferritin Baseline: AUC = 0.660, p = 0.018 (95% CI: 0.527 - 0.793). The AUC of 0.660 suggests that the Ferritin Baseline has a modest discriminative ability in predicting mortality. It shows a fair predictive capability, though not very strong. Whilst Ferritin Intermediate: AUC = 0.814, p = 0.000 (95% CI: 0.696 - 0.931), the AUC of 0.814 indicates that Ferritin Intermediate levels have good discriminative ability for predicting mortality. Values between 0.8 and 0.9 suggest strong predictive power. At the Ferritin Endpoint: AUC = 0.918, p = 0.000 (95% CI: 0.832 - 1.004). An AUC of 0.918 signifies excellent discriminative ability. Ferritin Endpoint is a highly reliable predictor for mortality, making it a significant biomarker for severe outcomes in patients. Overall, elevated Ferritin levels, especially at intermediate and endpoint stages, are highly indicative of poor outcomes, such as mortality, in patients.

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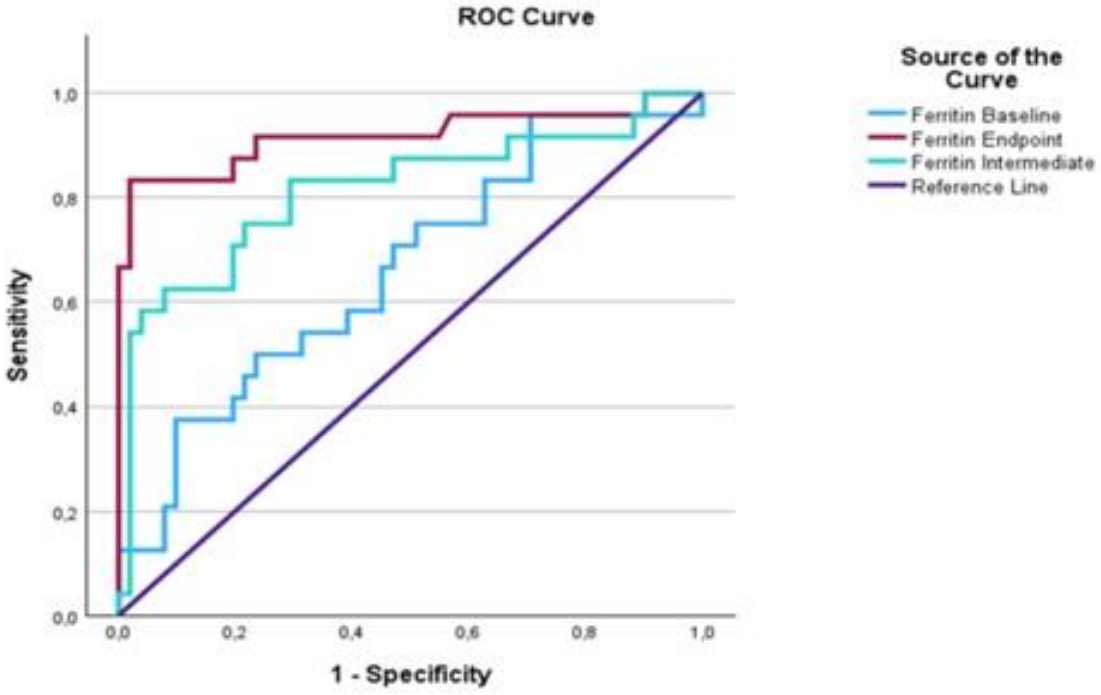
Figure (3). ROC analysis of Ferritin Levels at Baseline, Intermediate, and Endpoint Stratified by Outcome (Demised)



Similarly the predictive value of Interleukin-6 Baseline: AUC = 0.757, p = 0.001 (95% CI: 0.611 - 0.904), which indicates that IL-6 Baseline has moderate discriminative ability for predicting mortality. AUC values between 0.7 and 0.8 suggest fair predictive power. Interleukin-6 Intermediate: AUC = 0.914, p = 0.000 (95% CI: 0.826 - 1.002), and the AUC of 0.914 indicates strong discriminative ability, meaning that IL-6 Intermediate is a very good predictor of mortality. Interleukin-6 Endpoint: AUC = 0.946, p = 0.000 (95% CI: 0.885 - 1.007), showed that with an AUC of 0.946, IL-6 Endpoint shows excellent predictive performance, strongly indicating that higher IL-6 Endpoint levels are associated with mortality. Thus IL-6 levels, particularly Intermediate and Endpoint, can be used as significant indicators for predicting mortality in COVID-19 patients.

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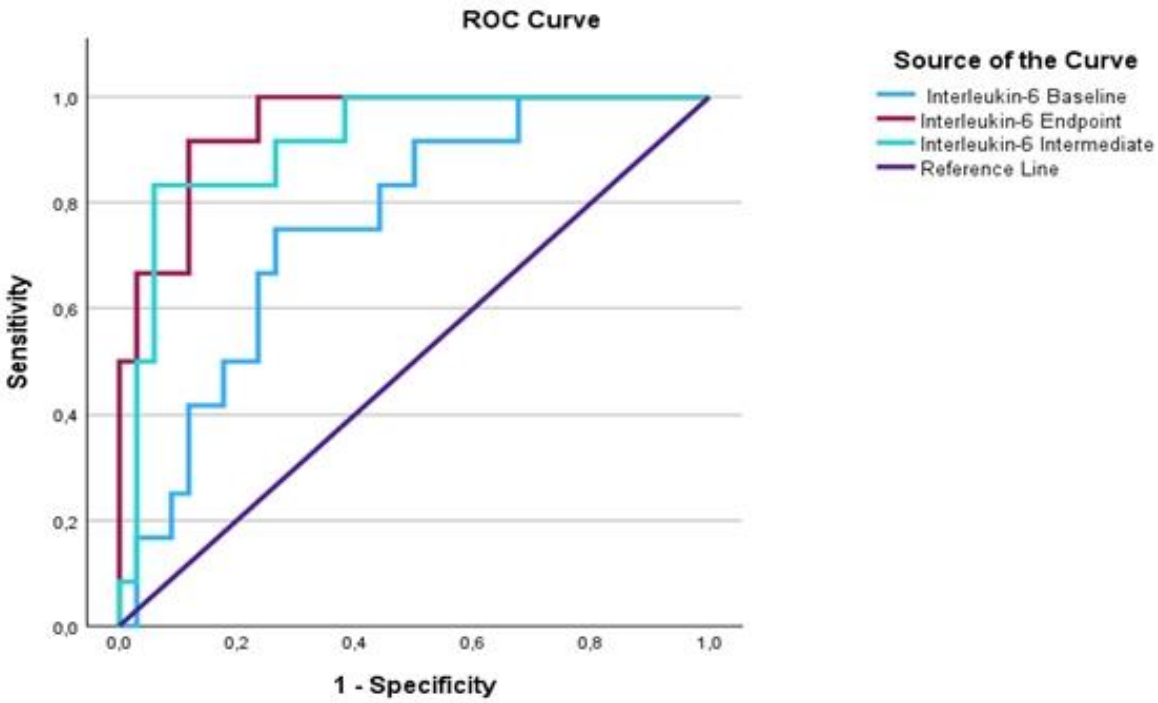
Figure (3). ROC analysis of Interleukin-6 levels at Baseline, Intermediate, and Endpoint Stratified by Outcome (Demised)



DISCUSSION

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the causative agent of COVID-19, has led to millions of deaths worldwide since its emergence in Wuhan, China, in December 2019 [9]. Several clinical and biochemical markers have been studied for their prognostic value in determining disease severity and outcomes [9,10,11,12,13]. This study specifically evaluated the association of ferritin and interleukin-6 (IL-6) levels with mortality risk in a South African cohort, while also considering key comorbidities such as hypertension, diabetes mellitus, and chronic kidney disease (CKD), along with other biochemical markers such as the neutrophil-to-lymphocyte ratio and HbA1C. While global studies have extensively investigated these factors, there remains limited data from African settings, necessitating further research to validate findings across different populations.

In this cohort of 125 patients, the median age was 45 years (IQR 32.0–56.0), and 64% were female. Neither age nor sex were significant predictors of mortality, which contrasts with studies from Europe and Asia, where older age and male sex were associated with poorer outcomes [9,12,14,15]. A South African study by Jalavu et al. reported similar findings, where more females than males were admitted with COVID-19, but higher mortality was observed in those older than 60 years [4].

Several studies have highlighted the role of comorbidities—including hypertension, diabetes mellitus, cardiovascular disease, and CKD—in worsening COVID-19 severity and outcomes [12,14,15]. In this study, BMI and CKD demonstrated strong associations with disease severity. Notably, for each unit increase in BMI, the odds of mortality increased by 8% (OR = 1.079). Li et al. previously reported that hypertension and diabetes contributed to increased COVID-19 severity [12,14,17].

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However, in this cohort, hypertension (prevalence = 48.0%) was associated with reduced odds of mortality (OR = 0.228, p = 0.031), suggesting potential protective effects of antihypertensive treatments or other unmeasured confounding factors, particularly when considering interactions with CKD and ethnicity. Meanwhile, diabetes mellitus (23.2%) was associated with elevated odds of mortality (OR = 2.881), but this did not reach statistical significance (p = 0.086), indicating that the risk of death may be confounded by other factors.

Several biochemical markers have been used to guide ICU admission, risk of intubation, treatment decisions, and disease progression [13,16,18]. Hyperferritinemia, iron overload, and IL-6 have been implicated in the cytokine storm and macrophage activation syndrome, which are key drivers of severe COVID-19 [6,8,10,13,18]. Multiple studies have demonstrated that elevated ferritin and IL-6 levels predict both disease severity and mortality risk [9,10,12,14,15,16,19]. Ferritin levels above 400 ng/mL have been associated with severe disease, while values exceeding 3000 ng/mL are linked to higher mortality [16]. Cheng et al. found that baseline ferritin levels above 500 ng/mL predicted mortality in 58% of cases [9]. Similarly, IL-6 levels have been shown to correlate with disease progression and imaging severity on chest CT scans [18]. Luke et al. reported that IL-6 levels above 80 pg/mL significantly increased the risk of respiratory failure and mortality [6.[

In this study, baseline ferritin levels were significantly higher in the demised group (mean = 2127.32, p < 0.001) compared to the alive group (mean = 563.96). Interestingly, baseline ferritin levels in the alive group were higher than those reported in other international studies but were consistent with findings from Jalavu et al. in Cape Town. Ferritin levels in the demised group increased markedly at the endpoint (mean = 10,020.2), while in the alive group, levels peaked and subsequently declined (mean = 336.29). Similarly, IL-6 levels were significantly elevated in the demised group at baseline (mean = 443.26, p < 0.001) and continued to rise at the endpoint (mean = 821.03). In contrast, IL-6 levels in the alive group declined significantly from baseline (97.24) to endpoint (18.1). These findings corroborate previous studies by Tau et al., demonstrating that elevated IL-6 levels correlate with poor outcomes. However, it is notable that baseline IL-6 levels in the live group were higher than those reported in other global studies, yet did not predict poor outcomes in this cohort.

While both ferritin and IL-6 levels have been validated globally as biomarkers of COVID-19 severity and mortality, an important unresolved question is whether ferritin levels can serve as a surrogate marker for IL-6. Given the strong correlation between these two inflammatory markers, further research is warranted to explore their interdependence and predictive accuracy in guiding the clinical management of COVID-19.

Study strengths and limitations

One of the primary limitations of this study is its small sample size, which may impact the generalizability of the findings. Additionally, the sample does not fully represent the ethnic diversity of South Africa, limiting its applicability to the broader population.

However, the study has several notable strengths. The setting in a quaternary hospital, which serves patients from various geographical areas across KwaZulu-Natal, enhances the diversity of clinical presentations and disease severity observed in the cohort. Furthermore, the inclusion of both admitted patients and previously healthy healthcare workers ensures a more unbiased sample, allowing for a comprehensive assessment of the impact of premorbid functioning and comorbidities on COVID-19 outcomes.

Another strength is the exclusion of ICU patients transferred directly from other facilities, which minimizes potential selection bias. This prevents the dataset from being skewed towards severe disease and poor outcomes, thereby ensuring a more balanced representation of disease progression and prognostic factors.

Despite its limitations, the study provides valuable insights into the prognostic role of ferritin and IL-6 in COVID-19 and highlights the need for larger, more representative studies to confirm these findings in diverse populations.

CONCLUSION

This study demonstrated that serial measurement of ferritin and interleukin-6 (IL-6) levels can serve as valuable prognostic markers in COVID-19 disease, with both biomarkers showing a strong correlation with disease severity and

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clinical outcomes. Elevated levels of ferritin and IL-6 at baseline were associated with increased mortality risk, and their progressive rise in non-survivors further emphasized their role in disease progression. These findings suggest that early identification of high-risk patients using these markers could facilitate timely therapeutic interventions, potentially altering clinical outcomes.

Moreover, this study found that ferritin levels are comparable to IL-6 in predicting disease severity and mortality, raising the question of whether ferritin could serve as a surrogate marker for IL-6 in inflammatory-driven disease states. Given ferritin's ease of measurement and accessibility, its potential role as an alternative biomarker for guiding risk stratification and treatment escalation warrants further investigation.

Future research should explore the generalizability of these findings to other inflammatory and infectious diseases, including their role in cytokine storm syndromes and hyperinflammatory states beyond COVID-19. Additionally, understanding the mechanistic link between ferritin, IL-6, and immune dysregulation could further optimize biomarker-driven therapeutic strategies. Ultimately, integrating these markers into clinical decision-making algorithms may improve early risk assessment, resource allocation, and patient outcomes in both pandemic and non-pandemic settings.

Ethical considerations: This study was conducted following ethical approval from the University of KwaZulu-Natal Biomedical Research Ethics Committee (BREC) and the Provincial Health Research and Ethics Committee (PHREC), under project number KZ202412003.

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Conflicts of interest: The Authors declare no conflict of interest

No other relationships or activities that could appear to have influenced the submitted work

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HIGHLIGHTS

- Serial ferritin and IL-6 measurements can serve as prognostic indicators in COVID-19 patients.
- Findings suggest that ferritin may serve as a surrogate marker for IL-6, potentially offering an alternative for risk stratification.

REFERENCES

1. Parasher. A COVID-19: current understanding of its pathophysiology, clinical presentation and treatment. *Postgrad Med J* 2021; 97:312-320.
2. Cevik M, Kuppalli K, Kindrachuk J, Peiris M. Virology, transmission and pathogenesis of SARS-COV-2. *BMJ* 2020; 371:1-6 .
3. Yuki K, Fujiogi M, Koutsogiannaki S. COVID-19 pathophysiology: A review. *Clin haem* 2020; 215:1-6 .
4. Jalavu T, Chapanduka Z, Abdullah I, Cornelissen H, Musekwa E, Mashigo N, et al. Biochemical laboratory findings in adult patients with coronavirus disease 2019 (COVID-19) at a university hospital in Cape Town, South Africa. *Jou of Med LTSA* 2022; 4(1):10-14.
5. Mason RJ. Pathogenesis of COVID-19 from a cell biologic perspective. *Eur Respir J.* 2020; 55(4):1-3.
6. Chen L, Hoiland RL, Stukas S, Wellington C, Sekhon MS. Confronting the controversy: interleukin-6 and the COVID-19 cytokine storm syndrome. *Eur Respir* 2020; 56: 1-6 .
7. McGonagle D, Sharif K, O'Regan A, Bridgewood C. The role of cytokines including Interleukin-6 in COVID-19-induced pneumonia and macrophage activation syndrome-like disease. *Autoimm Rev* 2020; 1-6 .
8. Habib HM, Ibrahim S, Zaim A, Ibrahim WH. The role of iron in the pathogenesis of COVID-19 and the possible treatment with lactoferrin and other iron chelators. *J.biomedpha* 2020; 136:1-8 .
9. Cheng L, Li H, Li L, Liu C, Yan S, Chen H, et al. Ferritin in the coronavirus disease 2019(COVID-19): A systematic review and meta-analysis. *J Clin Lab Anal* 2020; 00:1-18.
10. Kaushal K, Kaur H, Sarma P, Bhattacharyya A, Sharma DJ, Prajapati M, et al . Serum ferritin as a predictive biomarker in COVID-19. A systematic review, meta-analysis, and

meta-regression analysis. J Crit *Care* 2021; 67:172-181 .
11. Kappert K, Jahic A, Tauber R. Assessment of serum ferritin as a biomarker in COVID-19:bystander or participant? Insights by comparison with other infectious and non-infectious diseases. *Biomarkers* 2020; 25(8):616-625 .
12. Sun Y, Dong Y, Wang L, Xie H, Li B, Chang C, Wang FS. Characteristics and prognostic factors of disease severity in patients with COVID-19: The Beijing experience. *J of Autoimmunity* 2020; 1-6 .
13. Karimi A, Shobeiri P, Kulasinghe A, Rezaei N. Novel systemic inflammation markers to predict COVID-19 prognosis. *Front. Immunol* 2021; 12:1-11.
14. Bozkurt FT, Tercan M, Patmano G, Tannverdi TB, Avni D, Yureki UF. Can ferritin levels predict the severity of illness in patients with COVID-19? *Cureus*.2021; 13(1):1-8.
15. Alroomi M, Rajan R, Omar AA, Alsaber A, Pan J, Fatemi M, et al. Ferritin level: A predictor of severity and mortality in hospitalized COVID-19 patients. *Immun Inflamm Dis* 2021; 9:1648-1655 .
16. Para O, Caruso L, Pestelli G, Tangianu F, Carrara D, Maddaluni L, Tamburello F. Ferritin as a prognostic marker in COVID-19: the fervid study. *Postgrad Med* 2021; 1-6 .
17. Li B, Yang J, Zhao F, Zhi L, Wang X, Liu L, et al. Prevalence and impact of cardiovascular metabolic diseases on COVID-19 in China.*Clin Res Cardiol* 2020; 109:531-538 .
18. Liu T, Zhang J, Yang Y, Ma H, Li Z, Zhang J, et al. The role of interleukin-6 in monitoring severe cases of coronavirus disease 2019. *EMBO Mol Med* 2020; 12:1-12 .
19. Onur ST, Altin S, Sokucu SN, Fikri BI, Barca T, Bolat E, et al. Could ferritin level be an indicator of COVID-19 disease mortality? *J Med Virol* 2020; 1-6.

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