

# Predictors of mortality in acute hospitalised COVID-19 pneumonia patients: A retrospective cohort study at two tertiary-level hospitals in Zambia

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**Background.** The global COVID-19 pandemic has resulted in increased acute hospitalisations, a high demand for intensive care and high in-hospital mortality, placing a huge burden on healthcare systems.

**Objectives.** To assess in-hospital mortality outcomes and associated factors in acute hospitalised COVID-19 pneumonia patients in Zambia.

**Methods.** We performed a retrospective cohort review of patients admitted to two tertiary-level hospitals in Zambia from 1 March 2020 to 28 February 2021. We examined the factors (demographic, clinical and laboratory) that were associated with in-hospital mortality using multivariate logistic analysis. Adjusted odds ratios with their 95% confidence intervals (CIs) are reported.

**Results.** Of 350 patients, 59.4% were aged  $\geq 55$  years and 52.6% were male. The commonest comorbidities were hypertension, diabetes mellitus (DM), HIV/AIDS and chronic kidney disease (49.6%, 28.5%, 22.0% and 8.1%, respectively). The overall in-hospital mortality rate was 42.6%, and mortality was significantly increased in patients aged  $\geq 55$  years (52.0% v. 48.0%) and in those with DM (52.1% v. 47.9%), cardiac disease (68.0% v. 32.0%), a Quick Sequential (Sepsis-Related) Organ Failure Assessment (q-SOFA) score  $\geq 2$  (75.4% v. 24.6%), and admission blood glucose levels  $\geq 7.0$  mmol/L (66.3% v. 33.7%). Compared with patients who survived, who spent a median (interquartile range) of 6 (3 - 10) days in hospital, the median time between admission and death in those who died was 2.5 (1 - 6) days. In multivariate logistic analysis, age  $\geq 55$  years, a q-SOFA score  $\geq 2$  and a random blood sugar level  $\geq 7.0$  mmol/L were predictors of in-hospital mortality, with adjusted odds ratios of 1.54 (95% CI 1.09 - 2.17), 2.17 (95% CI 1.40 - 3.38) and 1.65 (95% CI 1.18 - 2.30), respectively. Raised serum creatinine was not associated with in-hospital COVID-19 mortality after adjusting for other confounders.

**Conclusions.** This study highlights that high in-hospital COVID-19 mortality was associated with a high q-SOFA score, hyperglycaemia on admission and older age. The study reinforces the need to invest in emergency healthcare services for optimal management of COVID-19 patients presenting with high q-SOFA scores in resource-limited countries.

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COVID-19 has stretched healthcare systems globally<sup>[1]</sup> since its identification. It has resulted in increased acute hospitalisations, a high demand for intensive care and a high in-hospital mortality rate.<sup>[2]</sup>

Available data from around the globe show that SARS-CoV-2 infection is associated with increased but varied mortality rates in persons with underlying comorbidities.<sup>[3,4]</sup> Some studies in developed countries have reported increased mortality rates as high as 70%.<sup>[3,5]</sup> A meta-analysis that included 60 studies with 51 225 patients reported a 24% in-hospital mortality rate among COVID-19 patients, with older age, kidney disease and diabetes mellitus (DM) being listed as risk factors for increased mortality.<sup>[6]</sup> In a study of hospitalised elderly patients with COVID-19, Becerra-Muñoz *et al.*<sup>[5]</sup> found almost 40% in-hospital COVID-19 mortality, with kidney disease, lymphopenia and hypoxaemia as independent positive predictors of mortality. In this study, a Quick Sequential (Sepsis-Related) Organ Failure Assessment (q-SOFA) score  $>1$  was associated with an 8.3-fold increase in the mortality rate. Recently, studies have also reported hyperglycaemia and high C-reactive protein (CRP) to be poor prognostic markers in patients hospitalised with COVID-19.<sup>[7-9]</sup>

Despite a high COVID-19 disease burden, sub-Saharan Africa, including Zambia, has a dearth of information on poor prognostic

markers associated with in-hospital deaths. Mwananyanda *et al.*<sup>[10]</sup> recently reported a high prevalence of SARS-CoV-2 among bodies examined post mortem at the largest teaching hospital in Zambia. Sub-Saharan African countries have a poor supply of emergency commodities such as oxygen,<sup>[11]</sup> a critical first-line treatment in severe COVID-19 pneumonia.

## Objectives

To examine clinical and laboratory markers linked with poor prognosis in acute hospitalised COVID-19 pneumonia patients, and thus to lay a foundation for planning during future SARS-CoV-2 waves.

## Methods

### Study design and setting

In a retrospective cohort study, we extracted and examined non-electronic medical records of all adult patients ( $\geq 18$  years) acutely hospitalised with severe COVID-19 pneumonia at two tertiary-level hospitals in Zambia (Ndola Teaching Hospital and Kitwe Teaching Hospital) from 1 March 2020 to 28 February 2021. The two hospitals were designated as the main referral isolation centres in the Copperbelt region. Ethical approval for the research

was obtained from the Tropical Diseases Research Centre Ethics Review Committee (ref. no. #TRC/C4/05/2021) and the National Health Research Authority (ref. no. #NHRA00004/8/06/2021). Excluded were 109 medical patients' files that were missing data or lacked clinical information.

### Study procedures

The following information was extracted from the non-electronic medical records: demographic data, comorbidity data on HIV/AIDS, hypertension (HTN) and DM, and laboratory data (CRP, D-dimers, haematology, kidney function, etc.). We also recorded admission vital signs (respiratory rate, blood pressure, etc.) and discharge outcomes. Confirmation of COVID-19 was based on a positive reverse transcription polymerase chain reaction test for SARS-CoV-2<sup>[12]</sup> or a positive antigen test, in line with the Ministry of Health (MoH) of Zambia guidelines. Both of the designated tertiary-level hospitals were equipped with a 5-bed COVID-19 intensive care unit (ICU) with 3 - 4 ventilators, and more wards were opened to accommodate increased numbers of patients.

However, the COVID-19 ICUs at both hospitals lacked a pressure swing adsorption oxygen plant, so the oxygen source was delivered via an F-cylinder manifold reticulation piping system to the COVID-19 ICUs and via F-cylinders (without an oxygen piping system) and oxygen concentrators to the COVID-19 main isolation wards. The major limitation was lack of oxygen delivery equipment in the face of increased oxygen consumption.

The two tertiary COVID-19 designated centres admitted moderate to severe/critical COVID-19 patients who were referred from primary health centres and from the other eight COVID-19 isolation units in the Copperbelt region. Patients who were triaged as severe/critical were expected to require more oxygen or intensive care management. The reason for triaging to the two facilities was to provide high-flow oxygen via C-circuits, high-flow nasal cannulas, and non-invasive and/or invasive ventilation that were not available in the lower COVID-19 facilities. After triage, patients were managed according to the MoH COVID-19 guidelines, which were in line with the World Health Organization (WHO) therapeutic guidelines.<sup>[13,14]</sup> All severe and critically ill patients were escalated on oxygen based on the MoH and WHO therapeutic guidelines.<sup>[13]</sup> In addition, all patients routinely received dexamethasone

or any available steroid, anticoagulation, and antibiotics when indicated and as outlined in the MoH guidelines. Remdesivir was also routinely ordered for all severely and critically ill patients with <10 days of symptoms or from the day of the SARS-CoV-2 result. However, owing to cost limitations, D-dimers and CRP were measured in 33.0% and 36.0% of patients, respectively, while remdesivir was administered in 21.8% of the eligible patients. Nurse-patient ratios were 1:3 in the COVID-19 ICU and 1:9 in the main COVID-19 wards during the period under review.

### Study definitions and outcomes

In this study, chronic kidney disease and new-onset DM definitions were based on the Kidney Disease Improving Global Outcomes (KDIGO)<sup>[15]</sup> and American Diabetes Association (ADA)<sup>[16]</sup> criteria, respectively. Patients with a clinical diagnosis of heart failure, hypertensive heart disease and ischaemic heart disease were defined as having cardiovascular disease. The outcome measures were in-hospital outcome (discharge or mortality), and exposures were markers associated with in-hospital COVID-19 mortality.

### Statistical analysis

Data analysis was conducted using SPSS Statistics, version 20 (IBM Corp., USA). Pearson's  $\chi^2$  test was used to establish associations at the 5% significance level. Independent factors associated with mortality were established using binary logistic regression analysis considering the backward (likelihood ratio) variable selection method,

probability of stepwise entry of 0.050 and probability of removal of 0.051. Adjusted odds ratios and their 95% confidence intervals are reported, together with *p*-values.

## Results

In total, 350 COVID-19 medical files of hospitalised patients were included in the study. Almost 60% were aged  $\geq 55$  years and 52.6% were male. The main comorbidities were HTN (49.6%), DM (28.5%) and HIV/AIDS (22.0%) (Table 1). Compared with women, men were more likely to have had a stroke, to have chronic kidney disease and to have raised serum creatinine. The most common presenting symptoms were shortness of breath (75.9%), cough (74.5%) and fever (56.5%).

### In-hospital mortality

Clinical characteristics of patients who survived and those who died are presented in Table 2. The medical files reviewed showed that in-hospital mortality was 42.6%. Mortality was significantly increased in patients aged  $\geq 55$  years (52.0% v. 48.0%) and in those with DM (52.1% v. 47.9%) or cardiac disease (68.0% v. 32.0%). The patients who died were likely to have a raised q-SOFA score and raised admission blood glucose, D-dimers, CRP and serum creatinine. The median (interquartile range (IQR)) time between admission and death in the deceased group was shorter than the median time to discharge for the survivors (2.5 (1 - 6) days v. 6 (3 - 10) days;  $p < 0.001$ ) (Table 2 and Fig. 1). The rate of hyperglycaemia was nearly twice as high in

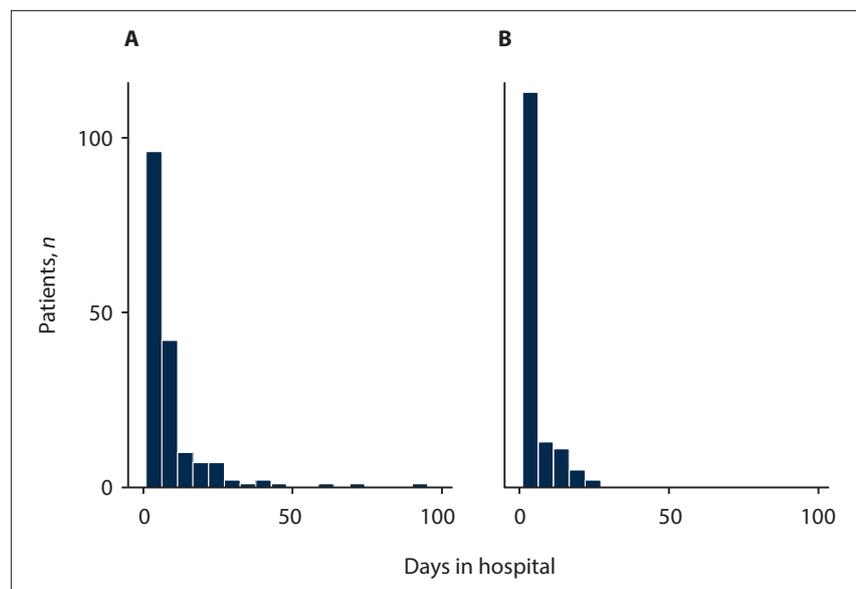


Fig. 1. Histogram frequency plot showing duration of stay in hospital in relation to outcome for patients who survived (A) v. those who died (B).

**Table 1. Clinical and laboratory characteristics of the study population**

Characteristics	Total (N=350)	Female (n=166)	Male (n=184)	p-value
Age ≥55 years, n (%)	206/347 (59.4)	106/206 (51.5)	100/206 (48.5)	0.103
Comorbidities, n (%)				
DM	96/337 (28.5)	47/96 (49.0)	49/96 (51.0)	0.582
HTN	169/341 (49.6)	91/169 (53.8)	78/169 (46.2)	0.027
HIV	53/241 (22.0)	30/53 (56.6)	23/53 (43.3)	0.324
History of CKD	14/173 (8.1)	3/14 (21.4)	11/14 (78.6)	0.038
Cardiac disease	25/325 (7.7)	12/25 (48.0)	13/25 (52.0)	0.974
Stroke	14/315 (4.4)	3/14 (21.4)	11/14 (78.6)	0.047
Presenting symptoms, n (%)				
Fever	182/322 (56.5)	84/182 (46.2)	98/182 (53.8)	0.667
Cough	248/333 (74.5)	117/248 (47.2)	131/248 (52.8)	0.836
SoB	255/336 (75.9)	119/255 (46.7)	136/255 (53.3)	0.670
Loss of smell	10/311 (3.2)	6/10 (60.0)	4/10 (40.0)	0.437
Died, n (%)	146/347 (42.6)	62/146 (42.5)	84/146 (57.5)	0.106
Time in hospital (days), median (IQR)	4 (2 - 9)	5 (2 - 10)	4 (2 - 7)	0.092
Vital signs				
SBP (mmHg), median (IQR)	130 (113 - 146)	130 (114 - 146)	132 (112 - 149)	0.583
DBP (mmHg), mean (SD)	82 (17.0)	81 (17.0)	82 (17.0)	0.769
RR (/min), median (IQR)	22 (20 - 26)	21 (20 - 26)	22 (20 - 26)	0.434
Pulse rate (bpm), median (IQR)	99 (86 - 115)	96 (85 - 112)	101 (87 - 117)	0.067
GCS score, median (IQR)	15 (15 - 15)	15 (15 - 15)	15 (14 - 15)	0.087
q-SOFA score, median (IQR)	1 (0 - 1)	1 (0 - 1)	1 (1 - 2)	0.096
SpO <sub>2</sub> (%), median (IQR)	86 (70 - 93)	86 (70 - 94)	87 (71 - 92)	0.814
Laboratory data, median (IQR)				
WCC (× 10 <sup>9</sup> /L)	7.7 (5.2 - 11.1)	7.5 (5.1 - 10.8)	7.8 (5.4 - 11.5)	0.357
Haemoglobin (g/dL)	13.3 (11.9 - 14.7)	12.6 (11.6 - 13.9)	14.0 (12.9 - 15.3)	<0.009
Platelets (× 10 <sup>9</sup> /L)	228 (164 - 315)	226 (164 - 330)	233 (159 - 309)	0.897
BS (mmol/L)	7.7 (5.7 - 12.2)	7.4 (5.8 - 12.7)	7.8 (5.7 - 12.2)	0.813
D-dimers (µg/mL)	1.6 (0.9 - 5.6)	2.0 (1.1 - 6.3)	1.3 (0.7 - 5.1)	0.162
CRP (mg/L)	105 (30.6 - 195.2)	105 (35.9 - 183.1)	104.1 (30.2 - 199.5)	0.924
Sodium (mmol/L)	141 (137 - 144)	140 (136 - 143)	142 (138 - 144)	0.086
Potassium (mmol/L)	4.2 (3.7 - 4.6)	3.9 (3.6 - 4.6)	4.3 (3.8 - 4.8)	0.101
Chloride (mmol/L)	100.5 (95.5 - 106.8)	101 (93 - 107)	100 (96.4 - 106.7)	0.652
Urea (mmol/L)	6.7 (4.2 - 9.5)	6.7 (3.7 - 8.8)	7.0 (4.9 - 10.0)	0.091
Creatinine (mmol/L)	113.2 (91.2 - 146.0)	106.1 (85.0 - 140.8)	122.8 (102.0 - 147.9)	0.018

DM = diabetes mellitus; HTN = hypertension; CKD = chronic kidney disease; SoB = shortness of breath; IQR = interquartile range; SBP = systolic blood pressure; DBP = diastolic blood pressure; RR = respiratory rate; bpm = beats per minute; GCS = Glasgow Coma Scale; q-SOFA = Quick Sequential (Sepsis-Related) Organ Failure Assessment; SpO<sub>2</sub> = oxygen saturation; WCC = white cell count; BS = random blood sugar; CRP = C-reactive protein.

patients with DM compared with those without (81.4% v. 44.9%;  $p=0.000$ ). Of the 44.9% of patients with hyperglycaemia who did not have DM, 38.7% were subsequently diagnosed with DM. Remdesivir was ordered for 294 patients, of whom 64 (21.8%) accessed the drug, with no significant survival benefit. The median (IQR) duration of admission in the remdesivir group v. the non-remdesivir group was 6 (2 - 10) days v. 4 (2 - 8) days ( $p=0.070$ ). In multivariate logistic analysis, a q-SOFA score  $\geq 2$ , a high admission blood sugar level and age  $\geq 55$  years were associated with in-hospital mortality. In multivariate analysis, raised serum creatinine was not associated with in-hospital mortality despite showing significance in unadjusted analysis.

## Discussion

This study revealed a high in-hospital COVID-19 mortality rate at nearly 43%, and admission hyperglycaemia, a high q-SOFA score and older age were predictors of in-hospital mortality. The in-hospital overall mortality in this study was higher than reported in

previous studies in Europe, China and the USA.<sup>[2,17,18]</sup> A meta-analysis consisting of 33 studies drawn from Europe, Asia and USA found a 17% overall in-hospital mortality rate.<sup>[17]</sup> In other studies carried out in tertiary-level hospitals, the in-hospital COVID-19 mortality rates were 26%, 30.1% and 29.7% in Saudi Arabia, Mexico and Italy, respectively.<sup>[19-21]</sup> A study from the SEMI-COVID-19 registry in Spain reported 21% overall in-hospital mortality.<sup>[22]</sup> In a retrospective examination of 127 inpatients with COVID-19 in China, Liu *et al.*<sup>[23]</sup> found a 16% hospital death rate. In-hospital mortality in a study using the Premier Healthcare Database in the USA was found to be 20.3% after analysis of 35 302 inpatients.<sup>[18]</sup>

It should be noted that the high in-hospital COVID-19 mortality rate reported in our study confirms the recent findings from the African COVID-19 Critical Care Outcomes Study (ACCCOS).<sup>[2]</sup> In this multicentre observational study of COVID-19 patients admitted to intensive care and high-care units in 10 African countries, an almost 50% in-hospital mortality was observed, translating to almost 23% more deaths than in developed countries.<sup>[2]</sup> In the ACCCOS study,

**Table 2. Clinical and laboratory characteristics of the patients who died v. those who survived**

Characteristics	Died (n=146)	Survived (n=197)	p-value
Gender male, n (%)	62/163 (38.0)	101/163 (62.0)	0.106
Age ≥55 years, n (%)	106/204 (52.0)	98/204 (48.0)	<0.001
Comorbidities, n (%)			
DM	49/94 (52.1)	45/94 (47.8)	0.033
HTN	77/166 (46.4)	89/166 (53.6)	0.142
HIV	19/53 (35.8)	34/53 (64.2)	0.771
CKD	6/14 (42.9)	8/14 (57.1)	0.256
Cardiac disease	17/25 (68.0)	8/25 (32.0)	0.004
Stroke	11/14 (78.6)	3/14 (21.4)	0.004
Presenting symptoms, n (%)			
Fever	61/179 (34.1)	118/179 (65.9)	0.003
Cough	102/244 (41.8)	142/244 (58.2)	0.954
SoB	112/249 (45.0)	137/249 (55.0)	0.077
Time in hospital (days), median (IQR)	2.5 (1 - 6)	6 (3 - 10)	<0.001
Vital signs			
SBP (mmHg), median (IQR)	133 (114 - 149)	129 (112 - 144)	0.242
DBP (mmHg), mean (SD)	81 (17.0)	82 (15.0)	0.399
RR (/min), median (IQR)	24 (20 - 30)	20 (20 - 24)	<0.001
Pulse (bpm), median (IQR)	101 (89 - 120)	96 (83 - 110)	0.004
GCS score, median (IQR)	15 (13 - 15)	15 (15 - 15)	<0.001
q-SOFA score			
Median (IQR)	1 (1 - 2)	1 (0 - 1)	<0.001
≥2, n (%)	49/65 (75.4)	16/65 (24.6)	<0.001
SpO <sub>2</sub> (%), median (IQR)	72 (50 - 87)	89 (83 - 95)	<0.001
Laboratory data			
WCC (× 10 <sup>9</sup> /L), median (IQR)	10 (6.1 - 13.6)	7.0 (4.8 - 9.8)	<0.001
HB (g/dL), median (IQR)	13.0 (11.6 - 14.4)	13.4 (12.3 - 14.9)	0.022
Platelets (× 10 <sup>9</sup> /L), median (IQR)	218 (159 - 283)	238 (169 - 329)	0.140
Admission blood sugar (mmol/L)			
Median (IQR)	8.5 (6.4 - 13.2)	6.3 (5.1 - 9.1)	<0.001
≥7, n (%)	69/104 (66.3)	35/104 (33.7)	<0.001
D-dimers (µg/mL), median (IQR)	2.9 (1.0 - 10.0)	1.3 (0.9 - 3.8)	0.038
CRP (mg/L), median (IQR)	160.5 (98.9 - 300.0)	79.5 (28.6 - 171.0)	0.005
Sodium (mmol/L), median (IQR)	142 (137 - 144)	140 (136 - 143)	0.459
Potassium (mmol/L), median (IQR)	4.2 (3.6 - 4.6)	4.2 (3.8 - 4.7)	0.698
Chloride (mmol/L), median (IQR)	102 (96 - 108)	98 (93 - 107)	0.234
Urea (mmol), median (IQR)	7.8 (5.3 - 10.8)	5.9 (3.7 - 8.5)	0.002
Creatinine (mmol/L), median (IQR)	124.6 (96.8 - 166.0)	108.6 (89.3 - 134.6)	0.014
Remdesivir, n (%)	29/64 (45.3)	35/64 (54.7)	0.658

DM = diabetes mellitus; HTN = hypertension; CKD = chronic kidney disease; SoB = shortness of breath; IQR = interquartile range; SBP = systolic blood pressure; DBP = diastolic blood pressure; RR = respiratory rate; bpm = beats per minute; GCS = Glasgow Coma Scale; q-SOFA = Quick Sequential (Sepsis-Related) Organ Failure Assessment; SpO<sub>2</sub> = oxygen saturation; WCC = white cell count; CRP = C-reactive protein.

**Table 3. Independent factors associated with mortality**

Factor	aOR (95% CI)	p-value
Age group (years)		
<55	1	
≥55	1.54 (1.09 - 2.17)	0.014
q-SOFA score		
<2	1	
≥2	2.17 (1.40 - 3.38)	0.001
Admission blood sugar (mmol/L)		
<7	1	
≥7	1.65 (1.18 - 2.30)	0.003

aOR = adjusted odds ratio; CI = confidence interval; q-SOFA = Quick Sequential (Sepsis-Related) Organ Failure Assessment.

delayed admission due to shortages of human resources, equipment, etc. was associated with 2-fold odds of in-hospital mortality.<sup>[2]</sup> The high death rate observed in the present study may therefore also be explained by lack of regional oxygen supply, and limited human resources for an optimal clinician to acute patient care ratio.

Older age was associated with almost 2-fold odds of COVID-19 in-hospital death in the present study, a finding consistent with previously observed epidemiological and clinical data.<sup>[24-26]</sup> In previous studies, older age was associated with 6-fold, 1-fold and 1-fold odds of in-hospital death in the Italian, Spanish and African ACCCOS studies, respectively.<sup>[2,22,24]</sup> Various reasons have been put forward to explain the association of age and COVID-19 death, including underlying comorbidities in the elderly, and impaired lung function and muscle weakness as age progresses.<sup>[24]</sup>

In the present study, admission hyperglycaemia was associated with almost 2-fold odds of in-hospital mortality, a finding consistent with previous studies.<sup>[9,27-28]</sup> A retrospective analysis conducted in Boston among patients aged  $\geq 18$  years with and without DM observed that hyperglycaemia in this study was associated with almost 4.5-fold, 4.5-fold, 3.7-fold, 1.9-fold and 4.4-fold odds of in-hospital mortality, intubation, ICU admission, adult respiratory distress syndrome (ARDS) and septic shock, respectively.<sup>[27]</sup> In the Spanish SEMI-COVID-19 registry, Carrasco-Sánchez *et al.*<sup>[9]</sup> reported a 15.7% in-hospital death rate in patients with blood glucose levels  $< 140$  mg/dL compared with 41% in those with levels  $> 180$  mg/dL. In a meta-analysis of 16 studies examining the association between hyperglycaemia and outcomes, hyperglycaemia was linked with 3.5-fold and 2-fold odds of in-hospital death and severe disease, respectively.<sup>[28]</sup>

Several studies have examined the association between COVID-19, hyperglycaemia and patient outcomes. The hyperglycaemia in patients with COVID-19 results from both direct and indirect mechanisms. Hyperglycaemia has been found to be positively associated with increased levels of inflammatory markers.<sup>[27-29]</sup> The increased levels of tumour necrosis factor, interleukin-6 and CRP have been observed to increase peripheral insulin resistance,<sup>[9,28,29]</sup> leading to the release of cortisol by stimulation of the paraventricular nucleus and epinephrine/norepinephrine excretion post stimulation of the locus coeruleus norepinephrine.<sup>[29]</sup> SARS-CoV-2 also directly infects the pancreatic beta cells via the angiotensin-converting enzyme 2 (ACE2) receptors,<sup>[28]</sup> leading to apoptosis and hyperglycaemia.

Hyperglycaemia has been observed to be a poor prognostic marker in COVID-19 patients.<sup>[30]</sup> Hyperglycaemia up-regulates levels of ACE2 receptors and enhances its glycosylation, thereby speeding up internalisation of the virus itself.<sup>[30,31]</sup> The increased ACE2 expression in various tissues promotes endothelial dysfunction, thromboembolic events, hyperviscosity, coagulation, ARDS, interstitial fibrosis and multisystem failure.<sup>[30,31]</sup> Furthermore, hyperglycaemia has been shown to impair both adaptive and innate immunity.<sup>[31,32]</sup>

Consistent with previous studies,<sup>[2,23]</sup> q-SOFA scores in our study were higher in the patients who died than in those who survived, and high scores predicted 2-fold odds of in-hospital mortality. In the ACCCOS multicentre study, a q-SOFA score consisting of two and three factors was associated with respective 2-fold and 4-fold odds of in-hospital COVID-19 death.<sup>[2]</sup> In resource-limited countries, q-SOFA remains an optimal tool for early identification of patients with severe COVID-19 disease.<sup>[23]</sup>

We observed a non-significant association between use of remdesivir and patient mortality outcomes, a finding that was observed in previous studies.<sup>[33]</sup> However, unlike previous studies that showed a quick recovery time for patients in the remdesivir cohort,<sup>[34-36]</sup> patients on remdesivir in our study showed a trend towards a longer hospital stay. In the presence of severe COVID-19 pneumonia, the clinicians could opt to prescribe remdesivir for longer than 5 days, as was observed in a US veterans study.<sup>[37]</sup> We also observed no association between HIV and patient outcomes, unlike the ACCCOS study,<sup>[2]</sup> in which HIV was associated with 1.9-fold odds of in-hospital death. The patients in our study may have been immune competent; however, owing to the retrospective nature of this study, the CD4 cell counts were lacking.

## Study strengths and limitations

To our knowledge, the present study is the first to address factors associated with poor in-hospital COVID-19 patient outcomes in Zambia. The study had an adequate sample size, and it has laid a critical foundation in planning for incoming COVID-19 waves in

resource-limited countries. However, the study had limitations; it was retrospective in nature, and laboratory markers of renal disease (proteinuria, haematuria) and acute kidney injury (due to non-availability of baseline serum creatinine), and the impact of inadequate delivery of oxygen equipment, were not assessed. Our two hospital COVID-19 sites are tertiary hospitals located in urban areas, and the outcomes may not be representative of outcomes from other hospitals owing to differences in resource capacity, patient characteristics and treatment protocols. It is possible that other confounders that we did not investigate could have contributed to the number of deaths, though this effect is expected to be minimal.

## Conclusions

This study has highlighted a high in-hospital COVID-19 mortality rate and the importance of early identification of COVID-19 patients presenting with a high q-SOFA score and hyperglycaemia.

**Declaration.** None.

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**Conflicts of interest.** None.

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