Prevalence, associated factors, management and outcomes of hypokalaemia in hospitalised patients at a South African tertiary centre

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Background. Hypokalaemia is a common electrolyte disorder encountered in hospitalised patients and is associated with significant morbidity and mortality. There is a lack of data regarding its prevalence, associated factors, management and outcomes among hospitalised patients in South Africa (SA).
Objectives. To evaluate the prevalence, associated factors, management and risk factors for all-cause mortality in hospitalised adult patients with hypokalaemia at a tertiary centre in SA.
Methods. We conducted a retrospective cohort study of all adult patients admitted with, or who developed, hypokalaemia during hospitalisation in 2019. Hypokalaemia was defined as a serum potassium concentration (K^+) <3.5 mmol/L. Based on a sample size calculation, a computer-generated random sample of 245 patients was used.
Results. The period prevalence of hypokalaemia was 6.8% (3 539/52 243). The median (interquartile range) age was 46 (33 - 63) years, and 60% were female. Patients who died had a lower K^+ during hospitalisation (3.0 mmol/L v. 3.2 mmol/L, $p<0.01$). Half of the patients had hypokalaemia on admission. The most common causes were gastrointestinal (37%) and renal (36%) losses. More than half (56.7%) of the patients received no potassium replacement, and of those discharged, only 37.5% were normokalaemic. In-hospital mortality was 16.7%. Only blood pH was associated with in-hospital death (adjusted odds ratio 0.01, 95% confidence interval 0.00 - 0.92, $p=0.046$). On survival analysis, there was no difference regarding in-hospital death by K^+ category (log rank $p=0.786$).
Conclusion. Although the prevalence of hypokalaemia among adult patients who were hospitalised was found to be low, their in-hospital mortality rate was high. Moreover, the investigations and management of hypokalaemia were frequently found to be inadequate. Therefore, it is imperative for healthcare providers in the hospital setting to enhance their knowledge and management of hypokalaemia. The findings of this study have implications for developing evidence-based guidelines for managing hypokalaemia in SA.

Keywords: hypokalaemia, retrospective cohort study, mortality, Africa

S Afr Med J 2024;114(12):e2066. https://doi.org/10.7196/SAMJ.2024.v114i12.2066

Hypokalaemia, a condition characterised by low levels of serum potassium, is a common electrolyte disturbance observed in hospitalised patients, and is associated with significant morbidity and mortality.[1-7] The normal serum potassium concentration (K^+) is tightly regulated within a narrow range of 3.5 - 5.0 mmol/L.[8,9] Hypokalaemia is defined as a serum K^+ <3.5 mmol/L, with reported prevalence in hospitalised patients ranging from 3.5 to 23%.[2,5,6,10-12] It may be classified as mild, moderate, or severe when the K^+ is between 3.0 and 3.4 mmol/L, 2.6 - 2.9 mmol/L and ≤2.5 mmol/L, respectively.[9]

Hypokalaemia occurs when there is an imbalance in the body's homeostasis, and can result from abnormal losses of potassium, reduced potassium intake, or intracellular shifts.[1,9] The most commonly identified causes of hypokalaemia are gastrointestinal and renal losses, while decreased intake is often a contributing factor, but rarely the sole cause.[1,13,14] To determine the underlying cause, a comprehensive history and physical examination, along with an analysis of urinary K^+, is necessary, as it helps differentiate between renal and non-renal losses as well as intracellular K^+ shifts.[8,9] In most cases, patients with hypokalaemia experience mild symptoms, or may be asymptomatic.[1] However, clinical symptoms tend to manifest when hypokalaemia reaches a moderate to severe level, and the severity, duration and rate of evolution of the condition can affect the clinical presentation.[9] Muscle weakness and life-threatening arrhythmias are the most concerning manifestations associated with moderate to severe hypokalaemia.[9,15] Even mild or moderate hypokalaemia has been associated with an increased risk of mortality.[7] In a prospective cohort of acute medical admissions, first-time admissions with hypokalaemia were associated with an increased hazard of death at 0 - 7 days and 8 - 30 days.[11] An inability to identify and correct the cause, along with inadequate therapy or overcorrection, likely contributes to the increased mortality associated with hypokalaemia.[16,17]

Despite the clinical importance of hypokalaemia, there is a dearth of comprehensive data on its prevalence and underlying causes among hospitalised patients in Africa, particularly South Africa (SA).

Only one study was found, which reported on the prevalence and risk factors of hypokalaemia specifically among pregnant women in the Eastern Cape Province of SA.[18] Therefore, owing to the limited availability of data from Africa, our objective was to determine the prevalence, causes, management and outcomes of hypokalaemia in adult patients admitted to a major tertiary centre in Cape Town, SA. Our study aims to address the knowledge gap of hypokalaemia in hospitalised patients in SA, and provide valuable information to improve the management and outcomes of this common electrolyte disturbance.

Methods

Study design

We conducted a retrospective descriptive study of all adult patients (≥18 years old) admitted with, or who developed, hypokalaemia during hospitalisation at Tygerberg Hospital (TBH) between 1 January 2019 and 31 December 2019. Hypokalaemia was defined as a K^+ concentration <3.5 mmol/L.

Study setting

TBH is a 1 340-bed government-funded tertiary hospital in Cape Town, SA. It provides secondary and tertiary level care to approximately 2.6 million people in the Western Cape Province. Laboratory services at TBH are provided by the National Health Laboratory Service (NHLS). The NHLS Chemical Pathology laboratory is accredited by the SA National Accreditation Services (SANAS), a regulatory body responsible for laboratory conformity to ISO15189 assessments in SA. Result quality is validated with internal quality control, and the laboratory participates in an external quality control scheme.

Study sample

All patients with a K^+ <3.5 mmol/L during 2019 were identified from the NHLS database. During the study period, K^+ was measured using indirect ion selective electrode potentiometry on the Roche Cobas 6000 (Roche Diagnostics, Switzerland) according to the manufacturer's recommendations. The NHLS reported the haemolysis index for all blood samples, therefore only validated K^+ concentrations were included. Outpatients, those with no clinical records available and patients receiving chronic dialysis were excluded.

The estimated sample size was 245, using a margin of error of 5%, a confidence interval (CI) of 95%, an estimated population size of 50 000 admissions per year, and a prevalence of hypokalaemia in hospitalised patients of 20%. A representative sample was obtained by means of computer-generated random sampling.

Data collection

For all patients in the sample, electronic patient records were examined, and a standardised data capture sheet was used to obtain information on demographics, clinical diagnosis, clinical manifestations of hypokalaemia, comorbid diseases including HIV infection, hypertension, diabetes mellitus, heart failure, acute or chronic kidney disease and malignancy. We also recorded information regarding mean arterial pressure (MAP), blood gas measurement and treatment received in hospital. The investigators inferred the causes of hypokalaemia by reviewing the patient's history, clinical notes and laboratory results. Chronic low intake was considered a diagnosis of exclusion. Laboratory data included the serum K^+ concentration at admission, lowest K^+, normalisation of K^+ and whether urinary K^+ measurements were performed. Outcomes data, including the length of hospital stay, whether hypokalaemia was corrected and in-hospital death, were also recorded.

Data analysis

Data analysis was performed using Stata version 17.0 (StataCorp, USA). We used the Shapiro-Wilk test for normality. Continuous variables were described using mean and standard deviation if normally distributed, or median and interquartile range (IQR) if they did not follow a normal distribution. Categorical variables were reported as frequencies and percentages of the number of cases. Chi-squared or Fisher's exact test was used to compare categorical variables, and for continuous variables, the Mann-Whitney *U* test was used for non-normally distributed data. Bivariate logistic regression analysis was performed to determine risk factors associated with in-hospital mortality. All factors that were significant ($p<0.1$) on univariate analysis were included in a backward multivariable logistic regression model. Kaplan-Meier survival analysis along with log-rank *p*-values were determined for in-hospital deaths. We considered $p<0.05$ to be statistically significant, and 95% CIs were reported.

Ethical considerations

Ethical approval was obtained from the Health Research Ethics Committee of Stellenbosch University (ref. no. S21/08/154), and we were granted a waiver of informed consent. All data were anonymised to ensure the privacy and confidentiality of participants' personal information, with each participant assigned a unique identifier.

Results

A total of 143 621 K^+ measurements were requested and processed at the TBH NHLS laboratory between 1 January 2019 and 31 December 2019. Of these, 10 915 had a K^+ <3.5 mmol/L. Samples not originating at TBH, duplicate records and patients <18 years old were excluded, leaving 3 539 records remaining. During 2019, there was a total of 52 243 admissions. The period prevalence of hypokalaemia was 6.8% (3 539 of 52 243 total admissions during 2019).

Computer-generated random sampling was performed on the remaining records to obtain a sample size of 245 patients, which were included in the final analysis.

The median (IQR) age was 46 (33 - 63) years, and 60% were female. There were no differences in age, sex, or comorbidities between patients who died in hospital and those who were alive at discharge. Most (40%) patients with hypokalaemia were admitted to medical wards. Patients admitted to the intensive care unit (ICU) were more likely to die in hospital (19.5% v. 7.4%, $p=0.01$), whereas patients admitted to obstetrics and gynaecology wards were less likely to die during admission (2.4% v. 18.1%, $p=0.01$) (Table 1).

There were 126 (51.4%) patients who had hypokalaemia on admission, and 119 (48.6%) developed hypokalaemia during their hospital admission. There was no significant difference in admission K^+ between patients who died in hospital and patients who were alive at discharge. Hypokalaemia was mild in 173 patients (70.6%), moderate in 50 (20.4%) and severe in 22 patients (9.0%), with more patients who had severe hypokalaemia dying during hospitalisation (19.5% v. 6.9%, $p=0.02$). Patients who died in hospital developed lower K^+ during hospitalisation (3.0 mmol/L v. 3.2 mmol/L, $p<0.01$) (Table 1).

The MAP was lower in patients who died in hospital (81.3 mmHg v. 91.3 mmHg, $p=0.02$). Patients who died were more likely to have had a blood gas determination during admission (73.2% v. 47.1%, $p<0.01$), with no difference in blood pH (7.45 v. 7.48, $p=0.42$). Only six (2.5%) patients had a urinary K^+ performed as part of the investigations to determine the cause of the hypokalaemia, all of whom were alive at discharge.

Regarding clinical manifestations of hypokalaemia, only three patients (1.2%) had documented muscle weakness, all of whom had severe hypokalaemia. No arrythmias were documented (Table 1).

There was no difference in length of hospital stay or time to correction between those who died in hospital and patients who were alive at discharge (Table 1).

More patients who died were thought to have chronic low intake (19.5% v. 5.9%, $p<0.01$). There were no differences between those who were alive at discharge and those who died in hospital for any of the other causes identified (Table 2).

More patients who died during hospitalisation received potassium replacement than those who were discharged alive (63.4% v. 39.2%, $p<0.01$) (Table 3). More than half (56.7%) of the study population received no potassium replacement during admission, and of the 204 patients who were discharged alive, only 77 (37.5%) had a normal K^+ at discharge.

On bivariate logistic regression, severe hypokalaemia (odds ratio (OR) 4.19, 95% CI 1.54 - 11.37, $p<0.01$), chronic low intake (OR 3.88, 95% CI 1.47 - 10.21, $p<0.01$), treatment with intravenous K^+ replacement only (OR 4.27, 95% CI 1.90 - 9.56, $p<0.01$) and both oral and intravenous K^+ replacement (OR 2.67, 95% CI 1.09 - 6.50, $p=0.03$) were associated with in-hospital death; however, only blood pH was associated with in-hospital death on a multivariable model (adjusted OR 0.01, 95% CI 0.00 - 0.92, $p=0.046$) (Table 4).

Forty-one patients (16.7%) died during hospitalisation (Fig. 1). Fig. 2 shows a regression analysis of the relationship between in-hospital death and K^+ concentration after adjusting for age, sex, hypertension, diabetes mellitus, heart failure, acute and chronic kidney disease, malignancy and HIV. On Kaplan-Meier survival analysis, there was no significant difference in survival associated with the severity of hypokalaemia (log rank $p=0.79$) (Fig. 3).

Table 1. Baseline characteristics of hypokalaemic patients and comparison of patients with in-hospital death v. patients discharged alive (*N*=245)

		In-hospital death, *n* (%)*		
Demographic data	**Patients, n**	**Yes, 41 (16.7), *n* (%)**	**No, 204 (83.3), *n* (%)**	***p*-value**
Age, median (IQR)	46 (33 - 63)	54 (37 - 66)	45 (32 - 61)	0.09
Female	147 (60)	24 (58.5)	123 (60.3)	0.83
Department				0.01†
Medical	97 (39.6)	17 (41.5)	80 (39.2)	
Surgical	79 (32.2)	13 (31.7)	66 (32.4)	
ICU	23 (9.4)	8 (19.5)	15 (7.4)	
O&G	38 (15.5)	1 (2.4)	37 (18.1)	
Other	8 (3.3)	2 (4.9)	6 (2.9)	
Comorbidities†				
Hypertension	96 (39.2)	18 (43.9)	78 (38.2)	0.50
Diabetes mellitus	36 (14.7)	4 (9.8)	32 (15.7)	0.33
Heart failure	23 (9.4)	2 (4.9)	21 (10.3)	0.28
Acute kidney injury	60 (24.5)	14 (34.2)	46 (22.6)	0.12
CKD	8 (3.3)	3 (7.3)	5 (2.5)	0.13
HIV	45 (18.4)	6 (14.6)	39 (19.1)	0.50
Malignancy	36 (14.7)	9 (22.0)	27 (13.2)	0.15
Other	17 (6.9)	3 (7.3)	14 (6.9)	0.57
None	65 (26.5)	7 (17.1)	58 (28.4)	0.13
Clinical and laboratory data				
K^+ (mmol/L), median (IQR)				
Admission K^+	3.4 (3.2 - 4.0)	3.7 (3.1 - 4.3)	3.4 (3.2 - 4.0)	0.74
Lowest K^+	3.2 (2.9 - 3.3)	3.0 (2.7 - 3.2)	3.2 (2.9 - 3.3)	<0.01†
Discharge K^+	n/a	n/a	3.4 (3.2 - 3.9)	
Hypokalaemia severity category				
Mild (3.0 - 3.4 mmol/L)	173 (70.6)	23 (56.1)	150 (73.5)	0.02†
Moderate (2.6 - 2.9 mmol/L)	50 (20.4)	10 (24.4)	40 (19.6)	
Severe (≤2.5 mmol/L)	22 (9.0)	8 (19.5)	14 (6.9)	
MAP (mmHg), median (IQR)		81.3 (70 - 102)	91.3 (80.8 - 103.7)	0.02†
Blood gas done	126 (51.4)	30 (73.2)	96 (47.1)	<0.01†
pH, median (IQR)	7.47 (7.43 - 7.51)	7.45 (7.42 - 7.51)	7.48 (7.44 - 7.5)	0.42
HCO_3 (mmol/L), median (IQR)	27.0 (22.9 - 29.6)	26.1 (21.0 - 28.2)	27.2 (23.2 - 30.0)	0.12
Urine K^+	6 (2.5)	0	6 (2.5)	0.23
Clinical manifestations	3 (1.2)	2 (4.9)	1 (0.5)	0.07
Corrected in hospital	92 (37.6)	15 (36.6)	77 (37.8)	0.89
Time to correction (days)	2 (1 - 5)	2 (1 - 3)	2 (1 - 5)	0.44
LOHS (days), median (IQR)	9 (4 - 17)	7 (3 - 19)	9 (4 - 17)	0.56

IQR = interquartile range; ICU = intensive care unit; O&G = obstetrics and gynaecology; CKD = chronic kidney disease; K^+ = potassium concentration; MAP = mean arterial pressure; n/a = not applicable; HCO3 = bicarbonate; LOHS = length of hospital stay.
*Unless otherwise indicated.
†Patients may be counted more than once.

Discussion

To the best of our knowledge, this is the first study from Africa to report on the prevalence, causes, management and outcome of adult patients hospitalised at a tertiary centre with hypokalaemia.

We found a prevalence of 6.8%, which is within the 3.5 - 23% prevalence range previously reported.[3,5,6,10-13] The only other study performed in SA reported a prevalence of 5.3% among pregnant women.[18] The wide range in prevalence is largely due to differences in the definition of hypokalaemia, the population investigated, comorbid diseases and whether it was based on hypokalaemia at presentation or developed during hospital admission.

Females were mostly affected, which is similar to other reports.[11,19] Bardak *et al.*[20] found female sex to be an independent risk factor for hypokalaemia in patients aged >65 years. It is not clear whether

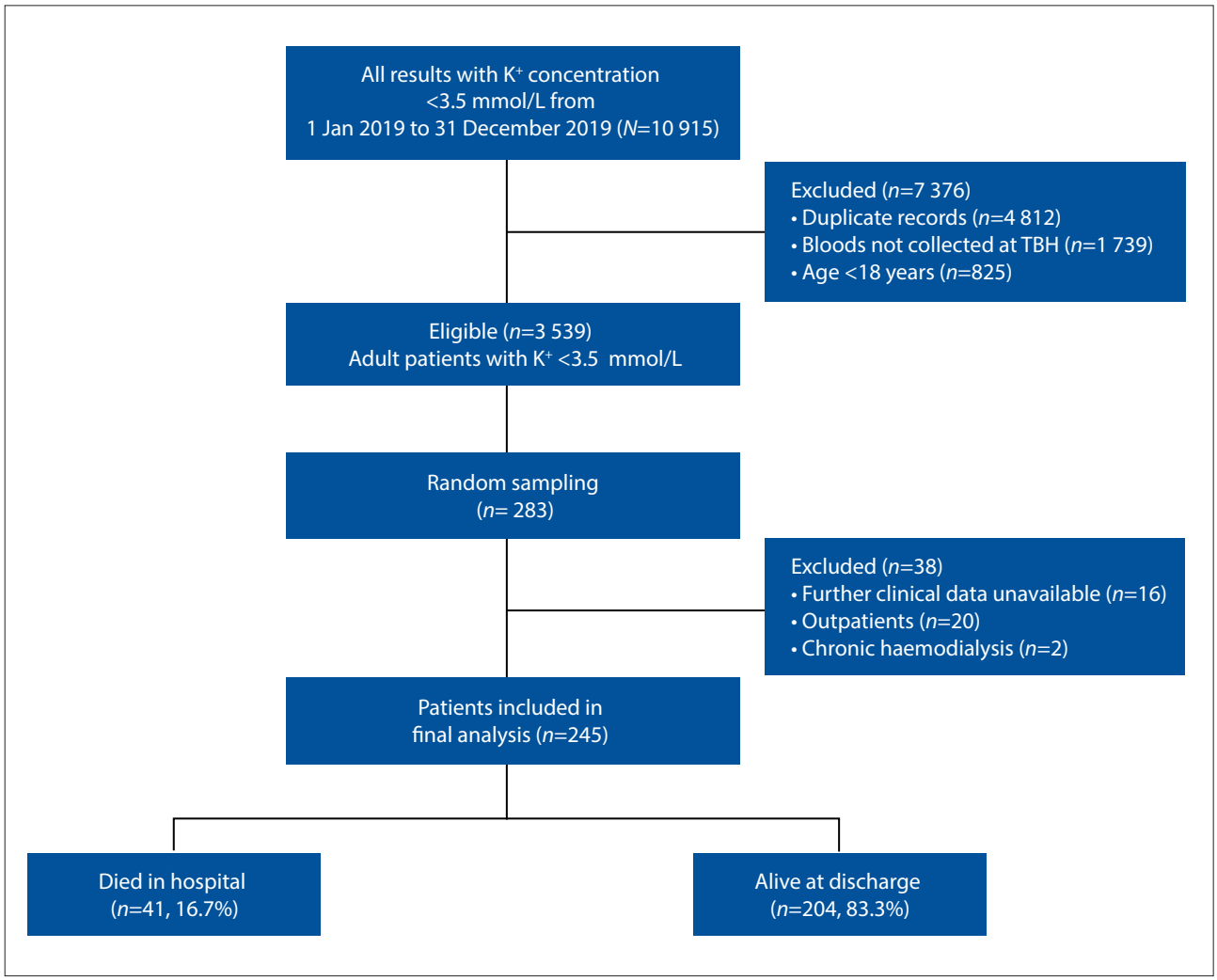

Fig. 1. Consort diagram of patients with hypokalaemia. (TBH = Tygerberg Hospital.)

Table 2. Associated factors of hypokalaemia (*N*=245)

	In-hospital death		
Associated factor, *n*%	**Yes, 41 (16.7), *n* (%)***	**No, 204 (83.3), *n* (%)***	***p*-value**
Chronic low intake	8 (19.5)	12 (5.9)	<0.01
Gastrointestinal losses	20 (48.8)	70 (34.3)	0.08
Shift into cells	6 (14.6)	19 (9.3)	0.22
Renal losses	14 (34.2)	74 (36.3)	0.80
Diuretics	8 (19.5)	59 (28.9)	0.22
Hyperaldosteronism	2 (4.9)	7 (3.4)	0.46
Antibiotics/other drugs	1 (2.4)	4 (2.0)	0.60
Metabolic acidosis	3 (7.3)	8 (3.9)	0.27
Other causes	13 (31.7)	78 (38.2)	0.43

*Patients may be counted more than once.

this higher prevalence is solely due to lower lean body mass and lower total body K^+ content, or if the difference is related to the more frequent comorbidities and diuretic use.

Nearly 40% of the patients were admitted to medical wards, with the most common causes of hypokalaemia being renal and gastrointestinal losses. A significant number of these patients had hypertension and/or heart failure and were often on diuretic therapy. Additionally, diarrhoeal disease was frequently identified as a cause of gastrointestinal potassium losses, regardless of HIV status. Among surgical patients, renal losses due to vomiting were a frequent cause of hypokalaemia, typically resulting from gastrointestinal obstruction. In ICU patients, other identified causes included free-draining nasogastric tubes, diuretics, steroids, beta-adrenergic drugs and insulin therapy. Hypokalaemia due to K^+ shifting into cells was uncommon, primarily occurring during the treatment of diabetic ketoacidosis. These patients were often managed in the emergency department, where point-of-care blood gas analysers were readily available, which allowed clinicians to promptly correct potassium concentrations without the need for obtaining laboratory potassium samples.

In our study, the overall mortality rate was 16.7%, which is higher than that reported by others who only investigated acute medical admissions with hypokalaemia.[7] There was an increase in mortality rate as the nadir K^+ decreased (Fig. 2), even after adjustment for multiple confounders, indicating the independent risk of in-hospital death associated with progressive hypokalaemia. A previous study from a high-income country evaluating hypokalaemia in medical admissions found a two-fold greater risk of death at day 7 of hospitalisation when the K^+ was <2.9 mmol/L.[11]

While most patients were admitted to medical wards, differences in mortality between departments were significant, with patients admitted to the ICU more likely to die in hospital, which suggests that the severity of the underlying illness in association with hypokalaemia is an important predictor of in-hospital death. Similarly to our data, pH has been found to have a major impact on in-hospital mortality in ICU patients with hypokalaemia.[21] We have previously reported

Table 3. Distribution of patients treated, and type of potassium replacement received (*N*=245)

		In-hospital death		
Treatment, *n* (%)	**Patients, *n***	**Yes, 41 (16.7), *n* (%)**	**No, 204 (83.3), *n* (%)**	***p*-value**
Treated	106 (43.3)	26 (63.4)	80 (39.2)	<0.01
Oral therapy only	18 (7.4)	0	18 (8.8)	
Intravenous only	47 (19.2)	16 (39.0)	31 (15.2)	
Both	41 (16.7)	10 (24.4)	31 (15.2)	
No treatment	139 (56.7)	15 (36.6)	124 (60.8)	

Table 4. Bivariate and multivariable logistic regression for in-hospital death

	Bivariate			Multivariable		
Variable	**OR**	**95% CI**	***p*-value**	**OR**	**95% CI**	***p*-value**
Age	1.01	1.00 - 1.03	0.14	n/a	n/a	n/a
Female sex	0.93	0.47 - 1.83	0.83	n/a	n/a	n/a
Department (reference: medical)						
ICU	2.51	0.92 - 6.86	0.07	1.40	0.38 - 5.14	0.61
O&G	0.13	0.16 - 0.99	0.049	n/a	n/a	n/a
MAP, mmHg	0.99	0.97 - 1.00	0.15	n/a	n/a	n/a
Blood pH	0.28	0.00 - 1.83	0.09	0.01	0.00 - 0.92	0.046
Serum HCO_3, mmol/L	0.95	0.89 - 1.02	0.17			
Chronic low intake	3.88	1.47 - 10.21	<0.01	2.58	0.42 - 15.73	0.31
GI losses	1.82	0.93 - 3.59	0.08	n/a	n/a	n/a
Shift into cells	1.67	0.62 - 4.48	0.31	n/a	n/a	n/a
Renal losses	0.91	0.45 - 1.84	0.80	n/a	n/a	n/a
Hypertension	1.26	0.64 - 2.49	0.50	n/a	n/a	n/a
Diabetes	0.58	0.19 - 1.74	0.33	n/a	n/a	n/a
Heart failure	0.45	0.10 - 1.98	0.29	n/a	n/a	n/a
Acute kidney injury	1.78	0.86 - 3.67	0.12	n/a	n/a	n/a
CKD	3.14	0.72 - 13.70	0.13	n/a	n/a	n/a
HIV	0.73	0.29 - 1.85	0.50	n/a	n/a	n/a
Malignancy	1.84	0.79 - 4.3	0.16	n/a	n/a	n/a
K^+ range (reference 3.1 - 3.4 mmol/L)						
2.6 - 3.0 mmol/L	1.90	0.89 - 4.02	0.10	0.91	0.29 - 2.84	0.88
≤2.5 mmol/L	4.19	1.54 - 11.37	<0.01	2.83	0.44 - 18.01	0.44
IV treatment	4.27	1.90 - 9.56	<0.01	2.76	0.83 - 9.16	0.10
IV and oral treatment	2.67	1.09 - 6.50	0.03	1.05	0.20 - 5.38	0.96

OR = odds ratio; 95% CI = 95% confidence interval; n/a = not applicable; ICU = intensive care unit; O&G = obstetrics and gynaecology; K^+ = potassium concentration; MAP = mean arterial pressure; HCO_3 = bicarbonate; GI = gastrointestinal; CKD = chronic kidney disease; IV = intravenous.

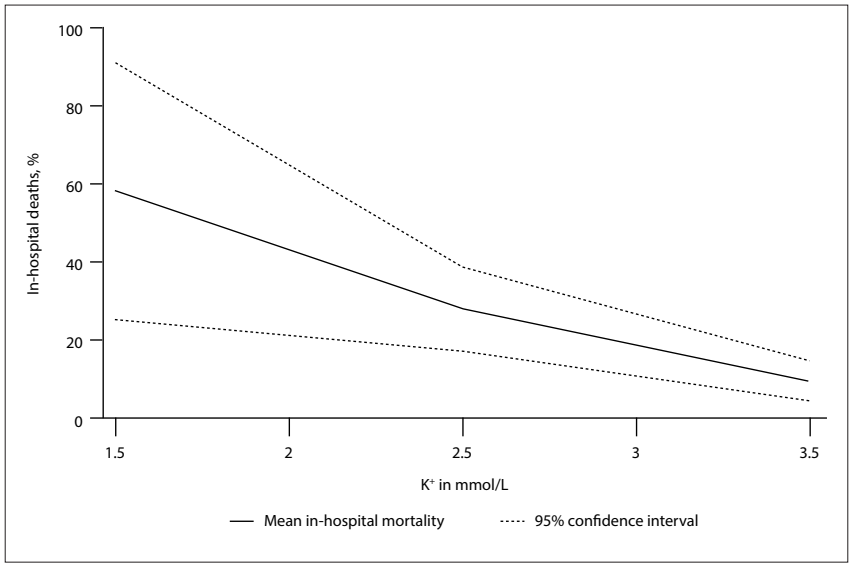

Fig. 2. Multivariable regression plot of the association between in-hospital death and lowest potassium concentration after adjustment for age, sex, hypertension, diabetes mellitus, heart failure, acute or chronic kidney disease, HIV and malignancy. The solid black line represents the mean in-hospital mortality, and the dotted lines represent 95% confidence intervals.

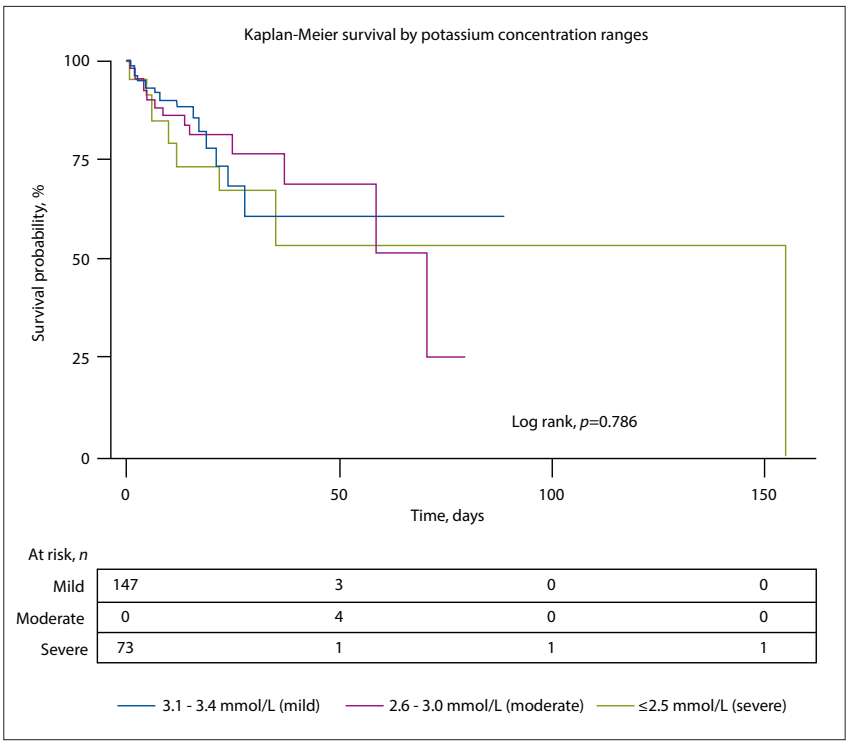

Fig. 3. Kaplan-Meier survival analysis showing the association between potassium concentration and in-hospital death.

that three of four patients had an acid-base disorder at the time of admission to the ICU, with no difference in ICU or in-hospital death due to the acid-base disorder.[22] Predictors of in-hospital death included male sex, the APACHE II score, and the corrected anion gap.

Symptoms of hypokalaemia often appear at a K^+ <3.0 mmol/L.[9] Of the 72 patients in our cohort with a K^+ <3.0 mmol/L, only 3 had documented muscle weakness. In a study conducted by Marti *et al.*,[14] it was observed that in half of patients with a K^+ <2.6 mmol/L, symptoms could not be attributed to any pathology other than hypokalaemia. In a separate prospective study, symptoms were found in a quarter of patients with hypokalaemia.[23] It is unlikely that so many patients with severe hypokalaemia in our study experienced no symptoms. This is probably attributed to inadequate documentation.

Only six patients had a urinary potassium-to-creatinine ratio (K-Cr) performed as part of the investigations. In some cases, the cause of hypokalaemia may be suggested by a history of diuretic use or gastrointestinal losses. If the cause is not evident, a measure of urine K^+ excretion is recommended.[9,24] The urinary K:Cr ratio aids in establishing whether hypokalaemia is due to renal or non-renal losses. The limited number of times this test was performed suggests that hospitalised patients are not being adequately investigated for the underlying cause of hypokalaemia, resulting in recurrence.

Interestingly, only chronic low potassium intake was associated with in-hospital death. These patients were frequently diagnosed with advanced malignancies or HIV and were malnourished with poor caloric intake. It has been frequently identified as a contributing cause in other studies;[13,14,23-25] however, chronic low intake is seldom the sole cause of hypokalaemia. We are unable to say, from our data, whether patients with chronic low intake have more severe hypokalaemia, which directly causes the increased mortality in this group, or if malnutrition is a marker of increased frailty and more severe illness. This needs to be investigated further in a larger study.

The finding that a higher proportion of patients who died during hospitalisation received potassium replacement may reflect a higher proportion of patients with severe hypokalaemia who died in hospital. Notably, more than half of the study population received no potassium replacement during admission, and only 37.5% of patients who were discharged alive had normokalaemia prior to discharge. According to a controlled before-and-after study conducted in multiple centres in Switzerland, only half of hospitalised patients with hypokalaemia received treatment; however, after guidelines were revised and targeted education was provided, this percentage increased to 75%. Two other studies have reproduced this finding, indicating that clinicians may be insufficiently aware of and insufficiently investigating and treating hospitalised patients with hypokalaemia.[14,15]

Study strengths and limitations

To the best of our knowledge, this is the first study from the African continent to report on the prevalence, causes, management and outcomes of adult patients at a tertiary centre with hypokalaemia. We used computer-generated random sampling and had good data on the treatment and

in-hospital outcomes. There were a few limitations. This was a small, single-centre, retrospective study; therefore, the results may not be generalisable. Bias may have been introduced with the sampling method, and the retrospective design also results in missing data. Additionally, there may be undocumented confounding. The use of point-of-care blood gas analysers in admission areas may have contributed to the lower prevalence observed in this study. While factors associated with hypokalaemia could be identified, the exact causes could only be inferred. Electrocardiographic data were not specifically reviewed during data collection, and therefore cardiac manifestations of hypokalaemia may have been missed.

Conclusion

Overall, this study highlights the high in-hospital mortality among adult patients with hypokalaemia. At our centre, there is a need to enhance clinicians' understanding regarding the diagnosis and treatment of hypokalaemia. Future studies should focus on identifying the underlying causes of hypokalaemia in hospitalised patients, and developing strategies and standardised evidence-based protocols to prevent and manage this common electrolyte abnormality.

Data availability. The data for this study will be made available on reasonable request to the corresponding author, and following approval by the Health Research Ethics Committee of Stellenbosch University.

Declaration. The research for this study was done in partial fulfilment of the requirements for CB's MMed (Int Med) degree at Stellenbosch University.

Acknowledgments. None.

Authors contributions. SL, MYC and CB conceptualised the study and developed the protocol. AZ assisted with procurement of the raw data from the NHLS. CB performed data collection. MYC performed the statistical analysis. CB wrote the first manuscript draft and interpreted the data. All authors contributed to and approved the final manuscript.

Funding. None.

Conflict of interest. None.

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Received 27 March 2024; accepted 24 September 2024.