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ORIGINAL ARTICLE

Prevalence and outcome of hospitalised patients with unexplained hyperkalaemia at a tertiary centre in South Africa

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ABSTRACT

Background: Hyperkalaemia is frequently encountered in hospitalised patients and is associated with high morbidity and mortality. However, there is a lack of data on the prevalence and outcomes of hospitalised patients with unexplained hyperkalaemia in Africa. This study addresses this knowledge gap.

Methods: We conducted a retrospective cohort study of all adult patients hospitalised with hyperkalaemia, defined as a potassium concentration of ≥5.5 mmol/L, between 1 January and 31 December 2019. Patients with kidney disease, rhabdomyolysis, tumour lysis syndrome, or who were taking medications that interfere with renal potassium elimination, were excluded.

Results: Hyperkalaemia prevalence was 8.2% (184/2,256). The median age of the patients was 40 years (interquartile range: 30–56), and 67% were men. All-cause in-hospital deaths were 8%, with no difference in potassium concentration between patients who died or were discharged alive (5.8 mmol/L vs. 5.7 mmol/L, P = 0.11). Only older age (adjusted odds ratio: 1.04; 95% confidence interval: 1.01–1.08) was associated with in-hospital death. There was no association between all-cause in-hospital deaths and potassium concentration range on survival analysis (P = 0.85). Regression analysis revealed associations between potassium concentration and leukocyte counts in patients requiring emergency surgery and those with haematological disorders. Blood gas analysis was performed on 17%, with a mean laboratory-to-blood gas potassium concentration bias of +1.3 mmol/L. Insulin therapy was prescribed to 7% with one episode of hypoglycaemia.

Conclusion: Unexplained hyperkalaemia was associated with low all-cause in-hospital deaths. We speculate that the elevated potassium concentrations were spurious and likely due to pre-analytical errors.

Keywords: pseudohyperkalaemia; spurious hyperkalaemia; prevalence, South Africa.

INTRODUCTION

Hyperkalaemia is a common electrolyte disorder in hospitalised patients and is associated with high morbidity and mortality. Potassium maintains the resting membrane potential of cell membranes and therefore plays a crucial role in regulating neuromuscular and cardiac conduction [1]. Hospitalised patients with underlying comorbidities, such as acute and chronic kidney disease, cardiovascular disease, and hypertension, as well as kidney transplant recipients and diabetics, are frequently found to have hyperkalaemia. Additionally, patients taking medications that interfere with renal elimination of potassium, such as renin–angiotensin–aldosterone system inhibitors, non-steroidal anti-inflammatory drugs, and trimethoprim, are also at risk of developing hyperkalaemia [2,3]. Other causes include tumour lysis syndrome, and rhabdomyolysis [4,5]. Clinical guidelines focus on early identification and prompt treatment of hyperkalaemia to prevent severe complications such as cardiac arrhythmias and neuromuscular weakness [6].

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Less information is available in the medical literature on unexplained hyperkalaemia, which is often the result of spurious hyperkalaemia, or pseudohyperkalaemia. Pseudohyperkalaemia is suspected when the measured serum potassium value exceeds the measured plasma potassium value by more than 0.3–0.4 mmol/L [7]. Spurious hyperkalaemia may occur during pre-analytical or analytical phases of sample handling. Pre-analytical causes of pseudohyperkalaemia occur prior to sample testing and include fist clenching [8], which releases intracellular potassium due to muscle contraction, thus potentially causing a rise in serum potassium by approximately 1 mmol/L [8]. Tourniquet application, which decreases potassium clearance from the clenched fist, and other errors such as using a small needle gauge, excessive pressure on the syringe plunger during aspiration, pneumatic tube transport, and excessive mixing of tubes can increase turbulence and cause sample haemolysis [9].

Analytical causes of pseudohyperkalaemia include the movement of potassium from intracellular components into the serum in vitro. This has been well described in serum samples from patients with thrombocytosis as potassium is released during clotting [10], as well as in patients with hyperleukocytosis from haematological malignancies. As a result of cell fragility, potassium is released into serum during centrifugation [11].

The finding of hyperkalaemia in the absence of electrocardiographic changes consistent with hyperkalaemia and in the absence of typical causes such as kidney disease, haemolysis, thrombocytosis, hyperleukocytosis, rhabdomyolysis, tumour lysis syndrome, or drugs known to interfere with renal potassium elimination, should trigger the consideration of pseudohyperkalaemia [12].

Inadvertent treatment of pseudohyperkalaemia with insulin-based therapy may result in potentially harmful adverse outcomes, including hypokalaemia and hypoglycaemia [9]. Data are lacking on the outcomes of unexplained hyperkalaemia in hospitalised patients from Africa. This study was thus conducted to identify the prevalence and outcomes of unexplained hyperkalaemia among hospitalised patients at our centre in Cape Town, South Africa.

METHODS

Ethical considerations

The Health Research Ethics Committee (HREC) of Stellenbosch University (HREC study number: 10988) approved the study protocol and a waiver of informed consent was granted. We conducted the study in accordance with the Declaration of Helsinki.

This was a retrospective cohort study conducted between 1 January and 31 December 2019 at Tygerberg Hospital, a 1,380-bed tertiary hospital in Cape Town, South Africa. Patients were identified by the primary investigator from the electronic database of the National Health Laboratory Service, a reference laboratory. Patients who were aged 18 years and older with a documented episode of hyperkalaemia, defined as serum potassium ≥5.5 mmol/L, were included. We excluded outpatients, patients on chronic kidney replacement therapy, and diabetic ketoacidosis.

Definition of unexplained hyperkalaemia

We defined unexplained hyperkalaemia as cases with serum potassium ≥5.5 mmol/L in the absence of symptoms or electrocardiographic changes ascribed to hyperkalaemia, absence of acute kidney injury and chronic kidney disease, absence of medications that increase potassium concentration such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, spironolactone, trimethoprim and non-steroidal anti-inflammatory drugs, and absence of other aetiologies that cause hyperkalaemia, including rhabdomyolysis, tumour lysis syndrome, or haemolysed blood samples.

An investigator reviewed all electronic medical records. Demographic and clinical data retrieved included age, sex, admitting diagnosis, and comorbidities, including hypertension, diabetes, cardiovascular disease, HIV status, and malignancy. Accompanying laboratory parameters reviewed from the electronic database included complete blood count, and results of kidney function tests and blood gas analysis.

Data analysis

Data were analysed using Stata version 16.1 (StataCorp LLC, Texas, USA). We used the Shapiro–Wilk test to evaluate normality. Numerical data with a normal distribution were described using means and standard deviations. Data that were not normally distributed were reported as medians and interquartile ranges (IQR). Student's t-test was used to compare the means of continuous variables with a normal distribution, and the Mann–Whitney U test was employed for variables that were not normally distributed. Chi-squared and Fisher's exact tests were used to compare categorical variables. Spearman's correlation coefficient was used to examine the relationships between potassium concentration and platelet and leukocyte counts, as well as the relationships between laboratory-to-blood gas difference in potassium concentration and platelet and leukocyte counts. We also determined the correlation between potassium concentration and leukocyte and platelet count by clinical diagnosis. Simple linear regression analysis was conducted when the strength of the correlation was at least moderate ($r > 0.4$). Agreement between the laboratory and blood gas potassium concentration was determined using the Bland–Altman analysis. Univariate and stepwise backward multivariable logistic regression analyses were performed to identify predictors of all-cause in-hospital death. Variables with a P value of <0.1 on univariate analysis were included in the multivariable model. Kaplan–Meier survival analysis was conducted and associated log-rank P value was determined for three potassium concentration categories, namely, 5.5–5.9 mmol/L, 6.0–6.9 mmol/L and ≥7.0 mmol/L.

RESULTS

We screened 3,183 inpatient records, of which 2,072 represented patients who had an identifiable cause of hyperkalaemia; 927 patients were excluded because they were either outpatients or receiving kidney replacement therapy. In total, 184 patients with unexplained hyperkalaemia were included, resulting in a period prevalence of 8.2%.

The median age was 40 (IQR: 30–56) years, and two-thirds of the patients were male (Table 1). Most patients (65%) were admitted to medical wards. There were no differences in HIV status, comorbidities, or clinical diagnosis between those who died in hospital and those who were discharged alive.

For the blood results, the median potassium concentration was 5.7 (IQR: 5.6–5.9) mmol/L with no difference between patients who died or were discharged alive (5.8 mmol/L vs. 5.7 mmol/L, P = 0.11). There were also no differences in the potassium concentration range. Blood gas analysis was performed in only 17% of patients, with a laboratory-to-blood gas potassium concentration mean bias of +1.3 mmol/L (Figure 1). There were no statistically significant correlations identified between potassium concentration and platelet count (r = 0.016, P = 0.83), potassium concentration and leukocyte count (r = 0.081,

Table 1. Clinical and demographic characteristics of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January and December 2019.

		All-cause in-hospital death, n (%)		
Characteristics	All, n (%)	Yes	No	P value
Total, n (%)	184 (100)	14 (8)	170 (92)	
Age (years), median (IQR)	40 (30–56)	53 (42–62)	39 (29–56)	0.01
Sex, n (%)				
Male	123 (67)	7 (50)	116 (68)	0.24
Ward, n (%)				
Medical	114 (62)	9 (64)	105 (62)	0.85
Surgical	70 (38)	5 (36)	65 (38)	
Comorbidities, n (%)				
HIV status, n (%)				
Negative	86 (47)	8 (57)	78 (46)	
Positive	28 (15)	3 (21)	25 (15)	0.44
Unknown	70 (38)	3 (21)	67 (39)	
Hypertension, n (%)	19 (10)	4 (29)	15 (9)	0.04
Diabetes, n (%)	14 (7)	1 (7)	13 (8)	1.00
CVD, n (%)	1 (0.5)	0 (0)	1 (0.6)	1.00
Clinical diagnosis				
NCD, n (%)	21 (11)	3 (21)	18 (10)	
ID, n (%)	26 (14)	2 (14)	24 (14)	
Malignancy, n (%)	26 (14)	1 (7)	25 (15)	
Trauma, n (%)	54 (29)	2 (14)	52 (31)	
Emergency surgery, n (%)	23 (13)	0 (0)	23 (13)	0.08
Elective surgery, n (%)	7 (4)	1 (7)	6 (4)	
Haematological disorder, n (%)	9 (5)	1 (7)	8 (5)	
Burns, n (%)	14 (8)	4 (30)	10 (6)	
Psychiatry, n (%)	1 (0.5)	0 (0)	1 (0.5)	
Unknown, n (%)	3 (1.5)	0 (0)	3 (1.5)	

Abbreviations: IQR, interquartile range; NCD, non-communicable disease; CVD, cardiovascular disease; ID, infectious disease. NCD included acute coronary syndrome, venous ulcers, hypertension, stroke, acute limb ischemia, diabetic foot, myocarditis, peripheral vascular disease, heart failure, valvular heart disease, diabetes, essential thrombocytosis, pneumothorax, anaemia, myelopathy, pancreatitis, hematuria, hemoptysis, psychosis, gout, cholelithiasis, chronic obstructive pulmonary disease, connective tissue disease.
ID included puerperal sepsis, pelvic inflammatory disease, pulmonary tuberculosis, undifferentiated sepsis, syphilis, viral hepatitis, spinal tuberculosis, liver abscess, pneumonia, intra-abdominal abscess, scrotal abscess, infective diarrhoea, disseminated tuberculosis.

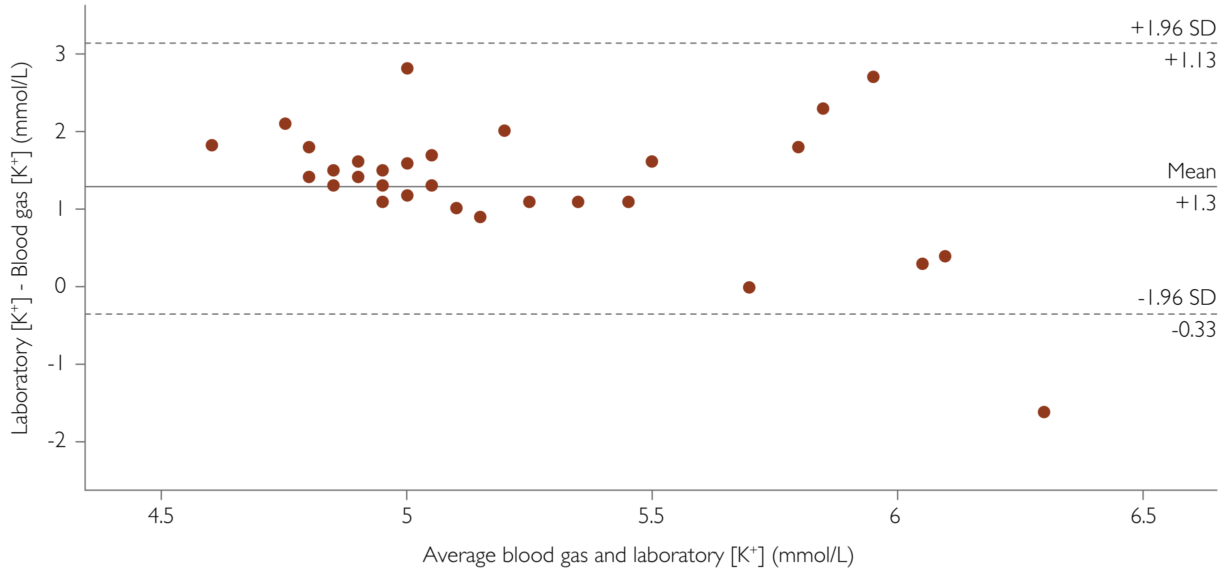


Figure 1. **Bland–Altman analysis of laboratory vs. blood gas potassium concentration of blood samples of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January and December 2019.**
Dashed lines represent the upper and lower limits of agreement, and the continuous line represents the mean difference.
Abbreviations: [K^+], potassium concentration; SD, standard deviation.

P = 0.27), laboratory-to-blood gas difference in potassium concentration and platelet count (r = 0.328, P = 0.07), or laboratory-to-blood gas difference in potassium concentration and leukocyte count (r = –0.048, P = 0.79). There was a moderate positive correlation between potassium concentration and leukocyte count in patients who required emergency surgery (r = 0.442, P = 0.035) and a strong positive correlation between potassium concentration and leukocyte count in patients with haematological disorders (r = 0.644, P = 0.061) (Figure 2). On simple linear regression, there were statistically significant associations between potassium concentration and leukocyte counts in patients who required emergency surgery (β = 0.023, standard error (SE) = 0.009, t = 2.69, adjusted r^2 = 0.22, P = 0.01) and those who had a haematological disorder (β = 0.005, SE = 0.002, t = 2.97, adjusted r^2 = 0.49, P = 0.02). There were no correlations between potassium concentration and platelet count by clinical diagnosis (Figure 3 and Table 1). Patients with haematological disorders had high leukocyte counts and relatively low platelet counts (Figures 1 and 2).

An electrocardiogram (ECG) was performed in only 5% of patients. Seven percent of patients received insulin therapy, of whom only two-thirds had blood glucose levels monitored. One episode of hypoglycaemia was documented, whereas no episodes of hypokalaemia occurred. There was no difference in the length of hospital stay between patients who died and those who were discharged alive (18.5 days vs. 15 days, respectively) (Table 2).

All-cause in-hospital deaths were recorded for 8% of the patients. On univariable logistic regression, only age [crude odds ratio (cOR) 1.04, 95% CI 1.01–1.08, P = 0.02] and hypertension (cOR 4.12, 95% CI 1.15–14.70, P = 0.03) were associated with in-hospital death. However, only age (adjusted OR 1.04, 95% CI 1.01–1.08, P = 0.02) was associated with death on a multivariable model (Table 3). Multivariable regression plot analysis showed that all-cause in-hospital death ranged from almost 0% at low potassium levels to just over 0.5% at the higher end of the range (Figure 4). On Kaplan–Meier survival analysis, we did not find a difference in survival regarding potassium concentration categories, log-rank P = 0.851 (Figure 5).

DISCUSSION

To the best of our knowledge, this is the first description of the prevalence and outcome of unexplained hyperkalaemia in Africa. We found a modest prevalence of only 8.2%, which may have resulted from our strict exclusion criteria. However, patients who demonstrated a clear cause of hyperkalaemia may also have experienced misleadingly elevated potassium concentrations. This phenomenon was highlighted in a separate investigation, which indicated a noteworthy occurrence of unexplained hyperkalaemia at 16% within a urology outpatient setting. Hyperkalaemia was defined as a potassium concentration exceeding 5.0 mmol/L, with patients at stage 5 chronic kidney disease being omitted from the study. We have previously reported

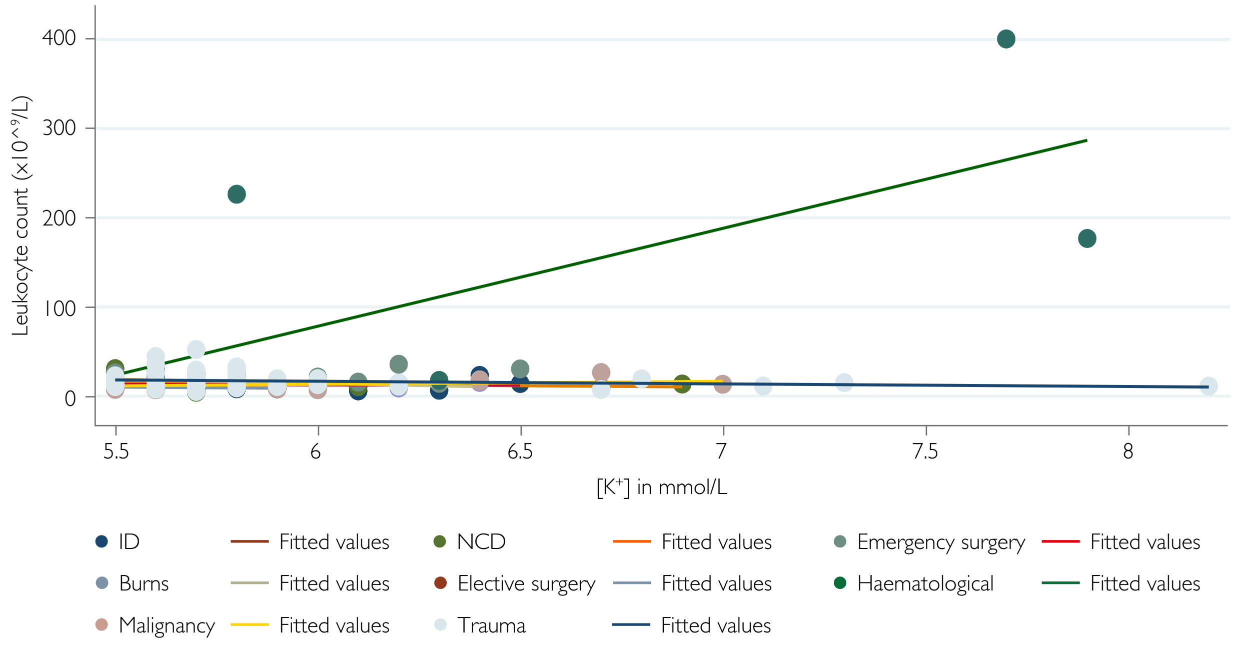


Figure 2. **Graph indicating the correlation between potassium concentration and leukocyte count by clinical diagnosis of patients with unexplained hyperkalaemia attending Tygerberg Hospital, South Africa, between January 2019 and December 2019.**
Abbreviations: ID, infectious disease; NCD, non-communicable disease.

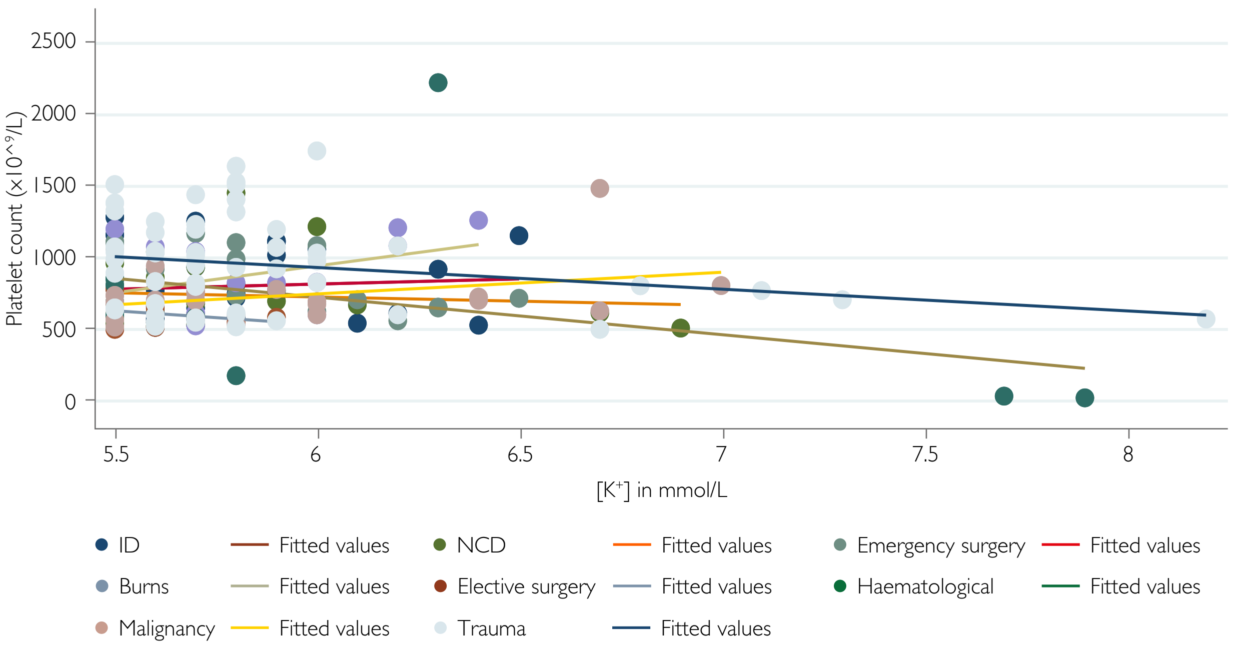


Figure 3. **Graph indicating the correlation between potassium concentration and platelet count by clinical diagnosis of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January 2019 and December 2019.**
Abbreviations: ID, infectious disease; NCD, non-communicable disease.

a hyperkalaemia prevalence of 3.7% in hospitalised patients at Tygerberg Hospital with an identifiable aetiology [13]. A systematic review in 2022 reported a much higher prevalence (8.6%) of hyperkalaemia (defined as a potassium concentration ≥5.5 mmol/L) in hospitalised patients [14]. However, the latter study included kidney transplant recipients, which may have contributed to the observed higher prevalence.

Table 2. Comparison of the baseline blood results and effect of emergency treatment of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January 2019 and December 2019.

		All-cause in-hospital death		
Laboratory results and clinical management	All patients	Yes	No	P value
Total, n (%)	184 (100)	14 (8)	190 (92)	
Potassium concentration				
Laboratory (mmol/L), median (IQR)	5.7 (5.6–5.9)	5.8 (5.6–6.5)	5.7 (5.6–5.9)	0.11
Category:				
5.5–5.9 mmol/L, n (%)	140 (76)	9 (64)	131 (77)	
6.0–6.9 mmol/L, n (%)	38 (21)	4 (29)	34 (20)	0.27
7 mmol/L or more, n (%)	6 (3)	1 (7)	5 (3)	
No. of blood gas analyses performed, n (%)	32 (17)	4 (29)	28 (16)	0.27
Blood gas (mmol/L), median (IQR)	4.4 (4.2–4.7)	4.7 (4.3–4.7)	4.4 (4.2–4.8)	0.87
Laboratory-blood gas difference (mmol/L), mean bias ± SD	1.3 ± 0.8	1.5 ± 0.7	1.3 ± 0.8	0.63
Creatinine (μmol/L), mean ± SD	62.0 ± 17.8	56 ± 19.3	63.0 ± 17.6	0.17
Platelets ($\times 10^9$/L), median (IQR)	721 (602–1035)	702 (611–1045)	726 (602–1034)	0.72
Leukocyte count ($\times 10^9$/L), median (IQR)	13.2 (10.2–17.4)	14.7 (12.5–22.9)	13.0 (9.9–16.8)	0.03
CD4 count (cells/mm^3), mean ± SD	321 ± 173	547 (422–664)	321 (149–380)	0.01
Emergency treatment, n (%)	16 (9)	3 (21)	13 (8)	0.11
Laboratory potassium concentration (mmol/L), median (IQR)	6.1 (5.8–6.7)	6.9 (5.6–7.0)	6.1 (5.8–6.4)	0.46
Insulin therapy, n (%)	12 (7)	2 (14)	10 (6)	0.23
Glucose monitored if treated with insulin, n (%)	8 (67)	1 (7)	7 (4)	1.00
Hypoglycaemia, n (%)	1 (0.5)	0 (0)	1 (0.6)	1.00
Length of hospital stay (days), median (IQR)	15.5 (6–42.5)	18.5 (1–59)	15 (6–42)	0.73

Abbreviations: IQR, interquartile range; SD, standard deviation.

Table 3. Univariate and stepwise backward multivariable logistic regression for all-cause in-hospital death of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January 2019 and December 2019.

	Univariate			Stepwise backward multivariable		
Predictor variables	Odds ratio	95% CI	P value	Odds ratio	95% CI	P value
Age	1.04	1.01–1.08	0.02	1.04	1.01–1.08	0.02
Male sex	0.47	0.16–1.39	0.17			
HIV positive	1.17	0.29–4.75	0.83			
Hypertension	4.12	1.15–14.70	0.03			
Diabetes	0.92	0.11–7.62	0.94			
Emergency treatment	3.29	0.82–13.31	0.09			
Potassium concentration, mmol/L	2.28	0.91–5.66	0.08	2.40	0.93–6.21	0.07
Surgical ward	1.02	0.33–3.20	0.97			

Abbreviation: CI, confidence interval.

A lower mortality rate of only 8% was found in the study reported here compared to an all-cause in-hospital mortality rate of nearly 30% for patients with an identifiable cause of hyperkalaemia reported by our centre during the same study period [13]. We found that mortality was attributable to age, as there was no significant difference in potassium concentration levels between those who survived or died, and potassium concentration was not identified as a predictor of death in the regression analysis conducted. The regression plot showed that all-cause in-hospital death was relatively very low across the entire potassium concentration range. In contrast, our previous study revealed that when hyperkalaemia was linked to an identifiable cause, the lowest potassium concentration on the regression plot was associated with all-cause in-hospital death rates of approximately 25% and that potassium concentration was an independent predictor of death on multivariable regression [13].

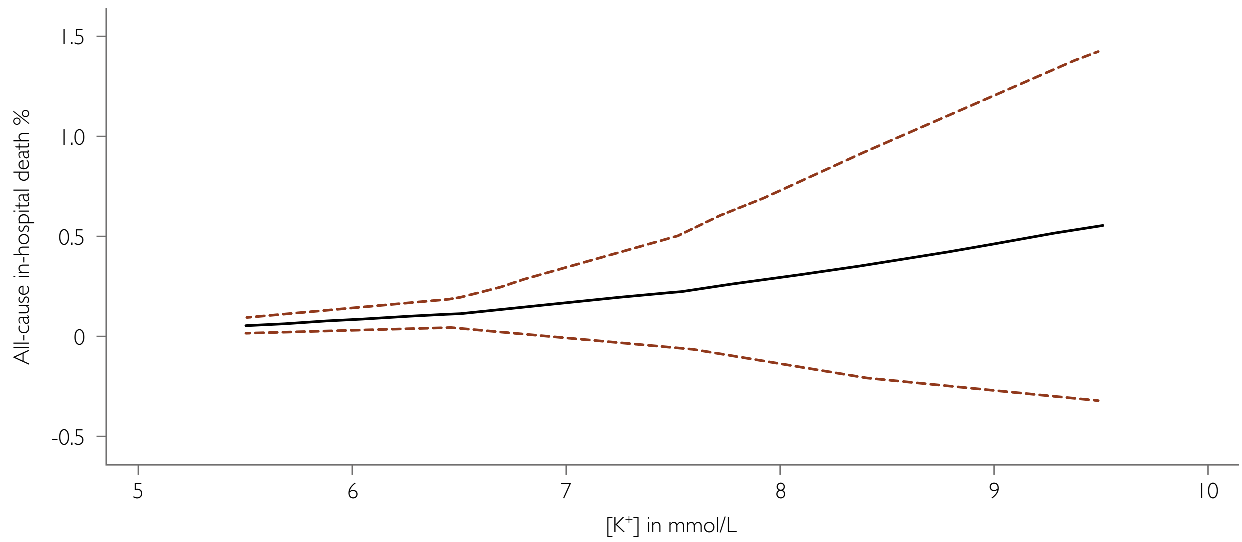
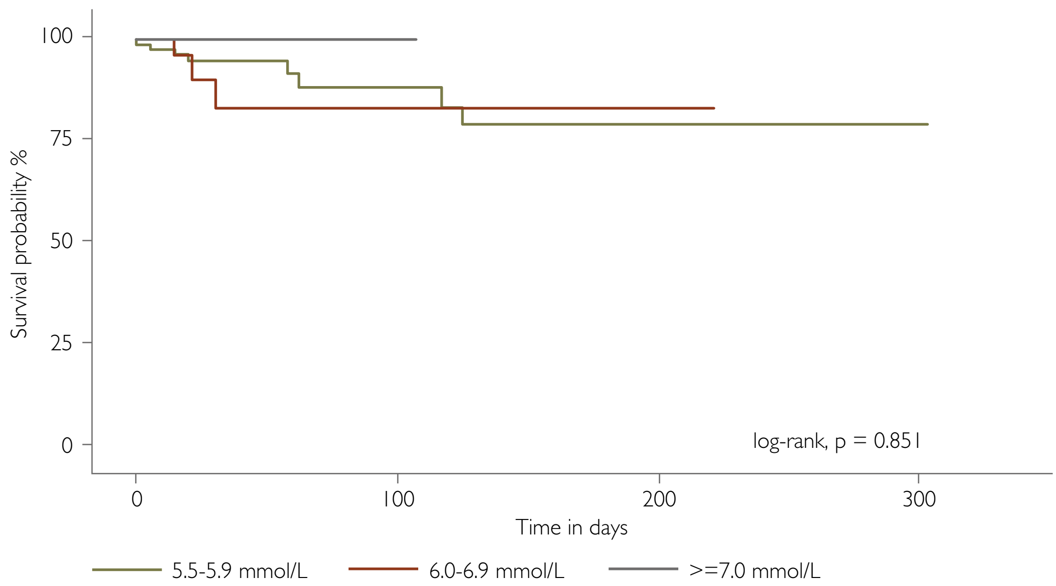


Figure 4. **Multivariable regression plot of all-cause in-hospital deaths by potassium concentration of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January 2019 and December 2019.**
Regression plot adjusted for age, sex, diabetes, hypertension, heart disease, and emergency treatment. The continuous black line represents the main all-cause in-hospital deaths. Dashed lines represent 95% confidence intervals.
Abbreviation: [K^+], potassium concentration.



Number at risk				
5.5-5.9 mmol/L	137	20	8	3
6.0-6.9 mmol/L	37	6	2	0
>=7.0 mmol/L	4	1	0	0

Figure 5. **Kaplan–Meier survival analysis by potassium concentration category of patients with unexplained hyperkalaemia attending Tygerberg Hospital, between January 2019 and December 2019.**

A small proportion of patients (17%) were subject to simultaneous blood gas potassium concentration testing. These patients met the criteria for pseudohyperkalaemia, which was characterised by a mean bias of +1.3 mmol/L between the laboratory and blood gas potassium concentration [7]. Pseudohyperkalaemia is frequently caused by thrombocytosis that exceeds 500 x 10^9/L [15]. Although the median platelet count was high in our cohort, there was no correlation between potassium concentration and platelet count, but there was a weak positive correlation between the laboratory-to-blood gas difference in potassium concentration and platelet count. The absence of statistical significance may have resulted from the limited number of paired samples recorded. Nijsten et al. compared the difference between the serum and plasma potassium concentration to the entire platelet count range and reported a statistically significant correlation of moderate strength across the entire potassium concentration range [16].

Given the absence of correlations between potassium concentration and age, platelet, and leukocyte counts, we postulate that pre-analytical factors related to phlebotomy techniques like fist clenching and tourniquet use may have contributed to this phenomenon. Phlebotomy errors are important contributors to spurious hyperkalaemia. An intervention study that involved educating phlebotomy staff on optimal blood draw practices and avoiding fist clenching, demonstrated a significant reduction in spurious hyperkalaemia rates from 16% to 3.8% [17]. Our study revealed an association between leukocyte count and potassium concentration in patients who underwent emergency surgery. Given that these patients were managed in the emergency department, it is probable that suboptimal phlebotomy techniques may have played a role. We also found an association between potassium concentration and haematological disorders. Three individuals were diagnosed with malignancies, including acute myeloid leukaemia, acute lymphocytic leukaemia, and chronic lymphocytic leukaemia, and their leukocyte counts ranged from 177–400 × 10^9/L. Leukaemia is widely acknowledged to be a potential cause of pseudohyperkalaemia as leukaemic cells are fragile and can release potassium in vitro during centrifugation and pneumatic tube transportation [15].

Although a recent survey revealed that up to two-thirds of medical specialists perform an ECG on patients with hyperkalaemia before management [18], only a small proportion of our study population underwent additional evaluation with an ECG and blood gas analysis. This is particularly crucial when dealing with unexplained hyperkalaemia, as clinicians must first exclude pseudohyperkalaemia before implementing any intervention. Despite recommendations from many hyperkalaemia treatment algorithms to exclude pseudohyperkalaemia first [19], there may be a lack of awareness among healthcare providers. A study reported that clinicians considered pseudohyperkalaemia in only 8% of patients [20].

It is of concern that 7% of patients in our study population received treatment with insulin therapy. A quarter of patients treated with insulin had their blood gas analysed, when the median laboratory-to-blood gas potassium concentration bias was +1.2 mmol/L. Of note, the timing of the blood gas sample was remote from insulin therapy and therefore the bias in potassium concentration was unlikely to reflect the effect of insulin. In addition, the bias remained high (+1.4 mmol/L) in patients who did not receive insulin therapy. These patients were at risk of developing cardiac arrhythmias and muscle weakness because of hypokalaemia. Another study revealed that among patients treated for pseudohyperkalaemia, one-third experienced severe hypokalaemia [20]. Additionally, one patient in our study manifested hypoglycaemia. The possibility of overlooking patients with hypoglycaemia cannot be excluded, given that the risk may be as high as 17% [21].

This was a small, single-centre, retrospective study. The possibility of bias exists due to missing data. Patients using atenolol were not excluded despite its potential to cause hyperkalaemia; however, the number of patients with cardiovascular disease was minimal and unlikely to have had a significant influence on our results. As only a small number of patients had paired laboratory and blood gas samples available, the possibility that they experienced pseudohyperkalaemia remains speculative.

CONCLUSION

We believe this is the first description of the outcome of unexplained hyperkalaemia in hospitalised patients in sub-Saharan Africa. The prevalence of unexplained hyperkalaemia was remarkably low. Moreover, unexplained hyperkalaemia had low all-cause in-hospital cases of death in this study. We suspect that the spurious elevation in potassium concentration may have occurred due to pre-analytical errors. Our results require validation through a prospective study that includes paired laboratory and blood gas samples performed concurrently on patients with unexplained hyperkalaemia.

Conflict of interest

The authors have no conflicts of interest to declare.

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