Original Article

Incidence, Risk Factors and Prognosis of Hypokalaemia in Patients with Normokalaemia at Hospital Admission

Jakka Bhargava, Stalin Viswanathan

Department of General Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research, Dhanvantari Nagar, Pondicherry, India

Abstract

Background: Hypokalaemia (K+<3.5 mmol/L) is observed in 20% of hospitalised patients. Previous studies have often dealt with the symptoms, prevalence and risk factors in hospitalised patients. Very few studies have dealt with hospital-induced hypokalaemia. The aim was to determine the incidence, predisposing risk factors and prognosis of patients developing hypokalaemia after admission. **Materials and Methods:** A prospective observational study was performed for two months. Patients with at least two potassium values after admission and normal K values at admission were considered for inclusion. Clinical features, diagnoses, laboratory reports and treatment details, including antibiotics, were noted. **Results:** A total of 653 patients were studied; 138 (21.1%) developed hypokalaemia. Diabetes, ischaemic heart disease (IHD), heart failure, chronic kidney disease, hypertension, chronic liver disease and chronic obstructive pulmonary disease (COPD) were the most associated comorbidities. Urea, creatinine, transaminases and neutrophilia at admission differed significantly between those with and without hypokalaemia groups. Most patients developed mild hypokalaemia (78.2%). Hypokalaemia developed mostly on the second (22.4%) and third (24.6%) days of hospitalisation. Antibiotics were used in 60% of patients. The potassium values returned to normal within 2.5 ± 1.9 days. Three patients subsequently developed hyperkalaemia. **Conclusion:** Patients admitted under general medicine mostly developed mild hypokalaemia, even if they had multiple risk factors for developing hypokalaemia. Inpatient hypokalaemia had an incidence of 21%. An overwhelming majority (~88%) had at least one risk factor. Hypokalaemia was not attributed to causing mortality in any patient.

Keywords: Antibiotics, aetiology, hospital-induced hypokalaemia, hypokalaemia, inpatient hypokalaemia

Introduction

One of the most common electrolyte abnormalities in clinical practice is a low serum potassium (≤3.5 mmol/L) concentration.[1] Hypokalaemia is found in over 20% of hospitalised patients but is clinically significant only in 4–5%.[2] It is often well tolerated but can be life-threatening if severe. Even mild and moderate hypokalaemia increases the risk of morbidity and mortality in patients with cardiovascular disease. Hypokalaemia is either mild (3.0–3.5 mmol/L), moderate (2.5–3.0 mmol/L) or severe (<2.5 mmol/L).[3] The degree and duration of serum potassium reduction determine the severity of the clinical manifestations. Compared to other electrolytes, potassium is predominantly an intracellular ion, and extracellular fluid contains only 2% of all potassium in the body; therefore, a small decrease in serum potassium may demonstrate a significant decrease in intracellular potassium.[4] Patients with mild hypokalaemia usually have no symptoms. Moderate hypokalaemia causes minor complaints, such as fatigue, muscle weakness, muscle pain, cramps and constipation.[5] Severe hypokalaemia can cause potentially life-threatening complications such as cardiac dysrhythmias, respiratory failure, rhabdomyolysis, paralysis, urinary retention, paralytic ileus and diaphragmatic weakness.[5]

Hypokalaemia generally results from either increased potassium excretion or intracellular shifts and less frequently from reduced potassium intake.[6] Regulation of potassium homoeostasis is done by kidneys, gastrointestinal tract,

Access this article online

Quick Response Code:

Website: https://journals.lww.com/indjem/

DOI: 10.4103/ijem.ijem_159_23

Address for correspondence: Dr. Stalin Viswanathan, Department of General Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research, Dhanvantari Nagar, Pondicherry - 605 006, India.
E-mail: stalinviswanathan@ymail.com

Submitted: 07-Apr-2023 **Revised:** 11-May-2023
Accepted: 25-Jun-2023 **Published:** 11-Jan-2024

How to cite this article: Bhargava J, Viswanathan S. Incidence, risk factors and prognosis of hypokalaemia in patients with normokalaemia at hospital admission. Indian J Endocr Metab 2023;27:537-43.

hormones and cellular transports.[7] Hypokalaemia developing after hospitalisation is often due to ongoing newly initiated treatment or the disease process itself. Normokalaemia at presentation may lead to a delay in the diagnosis of newly developed hypokalaemia. A delay in diagnosis or correction can lead to significant morbidity and occasional mortality.[3] Most studies have investigated the aetiology and symptoms of hypokalaemia and its prevalence at the time of admission. Hypokalaemia in hospitalised patients who were normokalaemic at admission is not well established. To our knowledge, no such study has been performed among hospitalised patients in India. Therefore, we performed this study to determine the incidence of hypokalaemia in hospitalised patients following admission and its associated risk factors.

Materials and Methods

This prospective cohort study was performed in the general medicine wards of a teaching government hospital under the Indian Council of Medical Research-Short term studentship (ICMR-STS) scheme (Reference ID: 2022-05095). This study was approved by the institutional ethics committee (No. JIP/IEC-OS/2022/241). We aimed to estimate the incidence of hypokalaemia in patients with normal potassium levels at admission and to determine the predisposing risk factors and outcomes in such patients. For two months, patients admitted to five wards with a total of 180 beds were monitored for serial potassium values that were available in the bedside charts. Patients with at least two potassium levels after admission (and admission values ≥3.5 mmol/L) were considered for inclusion in the study. Those with hypokalaemia (<3.5 mmol/L) observed in the second sample were included in the final analysis. Patients with K^+<3.5 mmol/L in the first sample were excluded. The diagnosis, demographics, comorbidities, symptoms and signs, investigations and treatment details of these patients were recorded in an Epicollect 5 form created for this purpose. Attention was paid to drugs or conditions that could affect potassium balance through reduced oral intake, loss through urine or the gastrointestinal tract and redistribution. Gastrointestinal loss was considered when the patient experienced vomiting or diarrhoea. The renal loss was considered when the patient was on diuretics, had polyuria or had a documented urinary potassium level >20 mmol/L. Reduced oral intake pertained to vomiting, abdominal pain, reduced appetite, altered sensorium and nasogastric feeding, which caused reduced intake compared with the usual intake by >50%.

The IBM Statistical Package for the Social Sciences (SPSS) for Windows v22 was used for statistical analysis. Frequencies of categorical variables, such as comorbid illness and treatment details, were calculated and analysed using the Chi-square test. Continuous variables were compared between those with and without hypokalaemia using Student's *t*-test. A paired *t*-test analysis was performed to compare potassium supplementation and treatment dosages of insulin and diuretics before and after the development of hypokalaemia. Statistical significance was set at $P < 0.05$.

The data set is available at 10.6084/m9.figshare. 21864507

Table 1: Baseline characteristics of the study population

Characteristics	Hypokalaemia (*n*=138)	No hypokalaemia (*n*=515)	*P*
Males	92 (66.7)	319 (61.9)	0.30
Age (y)	44.6±15.7	44.9±15.1	0.86
Hypothyroidism (*n*)	10 (7.2)	3 (0.6)	<0.001
Diabetes mellitus (*n*)	45 (32.6)	70 (13.6)	<0.001
Hypertension (*n*)	39 (28.3)	34 (6.6)	<0.001
Chronic kidney disease	27 (19.6)	71 (13.8)	0.09
Chronic liver disease (*n*)	7 (5.1)	12 (2.3)	0.08
Chronic obstructive pulmonary disease (*n*)	1 (0.7)	9 (1.7)	0.38
Ischaemic heart disease (*n*)	28 (20.3)	119 (23.2)	0.48
Stroke (*n*)	3 (2.2)	25 (4.9)	0.16
Admission potassium (mg/dL)	4.1±0.5	4.3±0.5	0.005
Second potassium (mg/dL)	3.8±0.6	4.3±0.5	<0.001
Serum sodium (mmol/L)	135.0±0.7	136.1±0.7	0.92
Urea (mg/dL)	59.3±59.0	47.1±51.8	0.01
Creatinine (mg/dL)	2.8±4.3	2.0±3.7	0.02
Corrected calcium admission (mg/dL)	7.4±4.9	7.9±10.3	0.56
Magnesium (mg/dL)	2.0±0.4	2.8±11.8	0.48
Aspartate transaminase (IU/L)	1592.9±1246.7	126.5±5.7	0.05
Alanine transaminase (IU/L)	422.5±36.2	144.1±6.5	<0.001
Alkaline phosphatase (IU/L)	94.0±8.15	186.5±8.5	0.35
Phosphate (mg/dL)	2.3±0.9	1.8±0.0	0.33
Haemoglobin (g/dL)	10.1±3.8	10.5±3.2	0.54
Total leucocyte count (×10^9/L)	16.1±45.3	11.3±11.2	0.03

Results

Our study included 653 patients, among whom 138 developed hypokalaemia, with an incidence of 21.1% [Table 1]. Diabetes mellitus (DM) was the most common comorbidity (n = 115), followed by ischaemic heart disease (IHD) and heart failure (n = 147) and chronic kidney disease (CKD) (n = 98). Ninety-two patients with hypokalaemia were male. Most patients had mild hypokalaemia (n = 108), while moderate (n = 27) and severe (n = 3) hypokalaemia were observed in smaller numbers. Potassium, urea, creatinine, transaminases and neutrophilia at admission also differed significantly between the two groups. Cardiovascular symptoms were the most common in patients who developed hypokalaemia, while pallor was the most common clinical finding [Table 2].

None of the patients developed any cardiovascular complications or neuroparalysis. Most patients developed hypokalaemia on the second (n = 31) and third (n = 34) days of hospitalisation. Only 21 of 138 patients developed hypokalaemia beyond the 7th day after admission. The median pH and bicarbonate levels in arterial blood gas analysis were 7.34 and 17.6, respectively. Urinary electrolytes were measured in less than 10% of patients with hypokalaemia and revealed both potassium loss and sodium loss in urine [Table 3]. Antibiotics were used in 60% of patients with hypokalaemia, with 23% and 24.5% of patients receiving one and two antibiotics, respectively. The use of diuretics in two-fifth, laxatives in one-fifth, insulin in one-fourth and inhaled salbutamol in one-sixth of the study group was also observed [Tables 4 and 5]. Cephalosporins were administered to nearly half of the patients [Table 6]. Twenty-six per cent of the study population did not have traditional risk factors related to comorbid illnesses or treatment. The potassium values returned to normal with a mean of 2.5 ± 1.9 days. Three patients developed hyperkalaemia, one each with severe falciparum malaria, community-acquired pneumonia with diabetes and hypothyroidism and aplastic anaemia.

Table 2: Clinical characteristics of patients with hypokalaemia

Characteristics	Means±SD/*n* (%) (*n*=138)
Age (y)	44.6±15.7
Duration of symptoms (d)	22.8±37.6
Fever (n)	76 (55.1)
Gastrointestinal symptoms (*n*)	69 (50)
Gastrointestinal loss (*n*)	56 (40.6)
Renal symptoms (*n*)	53 (38.4)
Neurological symptoms (*n*)	42 (30.4)
Cardiovascular symptoms (*n*)	89 (64.5)
Respiratory symptoms (*n*)	42 (30.4)
Elevated JVP (*n*)	28 (20.3)
Pallor (*n*)	65 (47.1)
Pedal oedema (*n*)	54 (39.1)
Clubbing (*n*)	6 (4.3)
Icterus (*n*)	19 (13.8)
Reduced skin turgor (*n*)	2 (1.4)
Dry oral mucosa (*n*)	7 (5.1)
Pulse rate (*n*)	97.1±19.7
Systolic blood pressure (mmHg)	120.7±27.8
Diastolic blood pressure (mmHg)	75.6±15.9
Respiratory rate (breaths/min)	21.6±6.9
Ascites (*n*)	13 (9.4)
Focal neurological deficits (*n*)	12 (8.7)
Infections (*n*)	54 (39.1)
Haematological disorders (*n*)	12 (8.6)

Table 3: Other inpatient laboratory values in patients with hypokalaemia

Values	Means±SD/*n* (%) (*n*=138)
First hypokalaemia value (mmol/L)	3.1±0.2
Hyponatraemia (*n*)	60 (43.4)
Hypomagnesaemia at admission (n)	5 (3.6)
Corrected calcium after hypokalaemia (mg/dL)	9.0±0.7
Magnesium after hypokalaemia (mg/dL)	1.8±0.3
Hypomagnesaemia after hypokalaemia (n)	9 (6.5)
PTH (pg/mL)	262.2±257.1
TSH (mIU/mL)	6.5±16.3
Urine potassium (mmol/L)	25.2±16.8
Urine sodium (mmol/L)	86.8±40.1
Urine Ca/creatinine ratio	2.68±3.7
Urine pH	5.5±0.5
Urine osmolality (mOsm/kg)	320.3±112.4
ECG heart rate (per minute)	101.0±23.9
ECG PR interval (ms)	147.6±29.1
ECG QRS duration (ms)	91.7±19.6
ECG QTc interval (ms)	454.3±70.0

Discussion

One hundred and twenty-one (87.6%) patients had at least one risk factor, while 81 (58.6%) had ≥2 risk factors. Only 2% of our study population had K+ less than 3 mmol/L, which is similar to other studies.[4,6] Hypokalaemia is seen in 40% of patients taking diuretics and 17% of patients with cardiovascular conditions.[8] Elderly patients may be predisposed to the development of hypokalaemia as an independent risk factor.[9] All patients >60 years in this study had either mild (n = 19) or moderate (n = 3) hypokalaemia. Age was not correlated with the development of hypokalaemia (r = -0.163). Half of the elderly patients (7/15) were treated with diuretics.

Very few studies have been performed on hospitalised patients to detect incident hypokalaemia after admission.[10–13] In 1979, Lawson *et al*.[10] studied 58167 patients in a teaching hospital, with 292 patients with severe hypokalaemia (<2.5 mmol/L), giving an incidence of 0.5%. Patients were from multiple medical and surgical specialties, were more likely to be women and had malignancies such as acute myeloid leukaemia. In 1986, the same cohort showed 21% with hypokalaemia and 56% attributable to drug therapy or intravenous fluids.[14] In the current study, 84 (60.8%) patients received antimicrobials,

Table 4: Drugs affecting potassium levels in patients with hypokalaemia

Treatment	*n* (%)	Admission potassium		*P*
		With drug	Without drug	
Frusemide (*n*)	42 (30.3)	4.0±0.5	4.3±0.5	0.002
Frusemide + spironolactone (*n*)	6 (4.3)	4.3±0.6	4.1±0.5	0.7
Mannitol (*n*)	7 (5.1)	3.7±0.3	4.1±0.5	0.02
Laxatives (*n*)	30 (21.7)	4.2±0.6	4.1±0.5	0.56
Salbutamol (*n*)	11 (7.9)	4.4±0.7	4.1±0.5	0.10
Insulin (*n*)	32 (23.1)	4.1±0.5	4.1±0.5	0.64
Steroids (*n*)	23 (16.7)	4.1±0.6	4.1±0.5	0.98
Thyroxine (*n*)	10 (7.2)	4.0±0.4	4.1±0.5	0.32
Digoxin (*n*)	5 (3.6)	4.1±0.3	4.1±0.5	0.95
Beta-blockers (*n*)	8 (5.7)	4.3±0.5	4.1±0.5	0.27
Enalapril (*n*)	5 (3.6)	3.8±0.3	4.1±0.6	0.21

Table 5: Treatment before and after the development of hypokalaemia

Treatment	Pre-hypokalaemia	Post-hypokalaemia	*P*
Frusemide dosage (mg)	358.6±6	156.3±26.4	<0.001
Frusemide duration (d)	2.6±0.2	1.4±0.1	<0.001
Spironolactone dosage (mg)	250.0±273.8	212.5±103.1	0.88
Spironolactone duration (d)	1.0 ± ± 0.2	0.7±0.1	0.70
Mannitol (mL)	678.5±339.6	411.2±205.6	0.18
Insulin dose (u)	20.0±71.7	14.7±48.2	0.39
Oral KCl supplementation (*n*)	4	83	0.001
Oral KCl (mEq)	165.0±161	60.0±30.0	0.55
Oral KCl duration (d)	0.6±0.0	1.4±0.1	0.01
IV KCl supplementation (*n*)	0	20	0.05
Intravenous KCl (mEq)	0	97.43±25.9	0.004
Intravenous KCl duration (d)	0.1±0.0.0	0.4±0.1	0.08
Juice (mL)	46.7±234.2	504.3±559.2	0.001

followed by diuretics with 42, insulin with 32 and laxatives with 30, none of them being mutually exclusive. Seventeen did not have a single risk factor.

Janko *et al*.[11] studied 1177 patients with hypokalaemia; 592 developed hypokalaemia after hospitalisation (cut-off, 3 mmol/L). DM, polytrauma and gastrointestinal and urogenital diseases were associated with hypokalaemia. This study did not include mild hypokalaemia. In an Indonesian study (2006) of 105 patients with infectious diseases (dengue = 82), DM, hypertension or hepatobiliary disorder receiving replacement solutions such as normal saline, ringer lactate and ringer's acetate, 39% (37/105) developed hypokalaemia. Most patients (91.4%) received Ringer's lactate.[13] Patients with renal and cardiac failure, diarrhoea and diuretic use were excluded from the study. In Saudi Arabia, hypokalaemia during hospitalisation (17.2%) increased as compared to the time of admission in patients with infectious diseases.[15] In the Netherlands, there was an 8.4% (100/1178) incidence of hypokalaemia in the general hospital population. Sixteen patients developed hyperkalaemia.[12] The study population included patients from internal medicine and medical subspecialties, surgery and surgical subspecialties, and gynaecology.[12] Our study focused only on patients admitted to general internal medicine care.

Hypokalaemia was associated with both hyponatraemia (24%) and hypomagnesaemia (61%).[12] The corresponding figures for hyponatraemia and hypomagnesaemia in the current study were 62% and 9%, respectively. A triad consisting of hypomagnesaemia, hypokalaemia and hypophosphataemia has also been reported, which was observed in 8% of patients with hypomagnesaemia and 17% of patients with severe hypomagnesaemia (plasma magnesium < or = 0.50 mmol/L).[16] In the current study, six patients received oral magnesium supplements before hypokalaemia development. After the development of hypokalaemia, nine (6.5%) patients were found to have hypomagnesaemia. Hypophosphataemia was not associated with hypokalaemia.

Severe hypokalaemia in hospitalised patients is commonly the result of multiple iatrogenic factors, and medications such as insulin, beta-2 agonists, laxatives, diuretics, antibiotics and methylxanthines are generally known to induce hypokalaemia.[17] Most studies involving inpatient hypokalaemia have had shortcomings in treatment histories since they were retrospective in nature, or a computerised database was used instead of directly perusing patient files. Krogager *et al*.[18] studied a Danish hypertensive registry on combination antihypertensives containing diuretics. Thiazide combinations were associated with a high risk of hypokalaemia. Our study population had diuretic use (furosemide, spironolactone and mannitol) because of CKD, chronic liver disease (CLD), heart failure and stroke. Zhu *et al*.[19] studied 108 patients scheduled for laparoscopic colorectal resection and found hypokalaemia in two-thirds and a longer time to first faeces.[19] The use of ≥2 cathartics, restricted oral intake and hypertension were risk factors for hypokalaemia. The incidence of hypokalaemia was not reported. Falcone *et al*.[20] also utilised a single-study population of patients with bone and joint infections. Zhou *et al*.[21] retrospectively studied 4445 patients with traumatic brain injury and predicted the development of inpatient hypokalaemia using machine-based algorithms. An incidence of 46.5% was found that was associated with higher mortality. All these studies had a homogeneous population in contrast to the current study.

Hypokalaemia had a strong association in patients with severe coronavirus disease 2019 (COVID-19) infection and prolonged hospital stay in a study from China.[22] Antimicrobials commonly associated with hypokalaemia include piperacillin–tazobactam,[23] amphotericin B,[24] aminoglycosides,[24] ureidopenicillins,[24] vancomycin[25] and flucloxacillin.[26] In piperacillin–tazobactam-administered patients, the incidence

rate of hypokalaemia was as high as 20%, and patients aged >80.5 years were considered a high-risk group.[23] Falcone *et al*.[20] reviewed 150 patients with recurrent admissions who used a few combinations of ciprofloxacin, vancomycin and rifampicin. Severe hypokalaemia (39%) was common, particularly in the elderly; this was associated with the simultaneous use of loop diuretics and thiazides.[20] We had a similar number of patients using 15 different antibiotics, three antivirals, two antifungals, three antiparasitic agents and one antituberculous drug along with a miscellaneous group, many of which have been associated with hypokalaemia. Other antimicrobials that have been reported to cause or be associated with hypokalaemia are cyclosporine,[27] fluconazole,[28] acyclovir,[29] azithromycin,[30] ceftriaxone,[30] ampicillin,[17] rifampicin,[20] colistin,[31] clindamycin,[32] meropenem,[32] ciprofloxacin[20] and entecavir.[33] Other drugs in hospitalised patients include thyroxine,[34] thiamine,[35] vitamin B12,[36] imatinib,[37] hydroxychloroquine,[38] valproate[39] and noradrenaline.[40] [Table 7].

ECGs were available for only 113 patients after the development of hypokalaemia. Many of the changes, such as ST or T waves, could be explained by co-existing illnesses, such as IHD, heart failure, strokes, hypothyroidism, uraemic pericarditis and hypertension. As hypokalaemia was also mostly mild, electrocardiogram (ECG) was probably not very useful in this population.

Some studies have shown that the treatment of dyselectrolytaemia is often suboptimal. In a multiphasic ambispective Swiss educational programme in five hospitals over 10 years, only 60% of patients had normokalaemia before discharge and only 20% had serum magnesium levels checked and 1% developed hyperkalaemia.[41] The average length of stay, as well as the incidence of falls and mortality, is almost double in hypokalaemic patients compared with normokalaemic patients.[41] Magnesium levels were tested in 112 (81.1%) before hypokalaemia, and 108 (78.2%) had a repeat test after hypokalaemia. An Israeli study retrospectively observed the quality of care of patients with hypokalaemia from a computerised database.[42] Failure to stop drugs that led to hypokalaemia was considered inappropriate. Among patients admitted with severe hypokalaemia, about 30% were discharged from the hospital with a subnormal potassium level, and 6.4% had no subsequent serum K+ levels measured.[42] In our study, in fluid overload states such as CKD, CLD, heart failure and strokes, which necessitated diuretics, only the dose could be reduced but not discontinued. No in-hospital deaths were attributable to low potassium levels.

There were no deaths attributable to hypokalaemia in our study. The mean duration of hospital stay was 15.4 ± 8.4 days.

Strengths and limitations

The study had a large number of participants (n = 653) providing for the incidence rate. All patients were discharged only after the normalisation of potassium values. The study duration was two months, which precluded a longer follow-up of many patients who developed hypokalaemia beyond two weeks. Therefore, the number of patients with hypokalaemia may have been slightly higher. The time to repeat potassium testing (in hours) by the treating unit even after the admission K^+ was found to be normal, and the reason for doing so was not documented. None of our patients underwent ECG after the development of hypokalaemia. Most patients also did not get completely worked up for the aetiology of hypokalaemia in the form of arterial blood gases, urine electrolytes, pH, osmolality and thyroid profiles,

Table 6: Antimicrobials and other miscellaneous drugs affecting potassium

Admission potassium	*n* (%)	With drug	Without drug	*P*
Antimicrobials (*n*)	84 (60.8)	4.1±0.5	4.1±0.6	0.88
Linezolid (*n*)	2 (1.4)	3.5±0.1	4.1±0.5	0.01
Amikacin (*n*)	12 (8.6)	4.2±0.7	4.1±0.5	0.86
Acyclovir (*n*)	4 (2.8)	4.2±0.9	4.1±0.5	0.84
Vancomycin (*n*)	4 (2.8)	4.4±0.7	4.1±0.5	0.34
Piperacillin–tazobactam (*n*)	20 (14.4)	4.2±0.6	4.1±0.5	0.57
Cephalosporins (*n*)	61 (44.2)	4.2±0.6	4.0±0.4	0.07
Azithromycin (*n*)	16 (11.5)	4.4±0.6	4.1±0.5	0.06
Doxycycline (*n*)	17 (12.3)	4.1±0.7	4.1±0.5	0.93
Metronidazole (*n*)	10 (7.2)	4.0±0.4	4.1±0.5	0.34
Amoxicillin/ampicillin (*n*)	9 (6.5)	4.1±0.9	4.1±0.5	0.95
Cloxacillin (*n*)	8 (5.7)	4.3±0.9	4.3±0.5	0.35
Phenoxymethyl penicillin (*n*)	5 (3.6)	4.3±0.8	4.1±0.5	0.54
Rifampicin (*n*)	8 (5.7)	4.3±0.6	4.1±0.6	0.52
Heparin (*n*)*	24 (17.3)	4.0±0.3	4.2±0.5	0.09
Thiamine (*n*)#	24 (17.3)	4.0±0.6	4.1±0.5	0.40
Vitamin B12 (*n*)#	14 (10.1)	4.1±0.4	4.1±0.5	0.86
Second potassium				
Metronidazole (*n*)	10 (7.2)	3.3±0.3	3.8±0.7	0.02
Azithromycin (*n*)	16 (11.5)	4.2±0.9	3.7±0.6	0.007
Linezolid (*n*)	2 (1.4)	3.4±0.1	3.8±0.7	0.01
Cyclosporine (*n*)*	2 (1.4)	3.8±0.7	4.2±0.1	<0.001

Table 7: Other antimicrobials and miscellaneous drugs that were administered

Drug	No. of patients	Drug	No. of patients
Clindamycin (*n*)#	1	Imatinib (*n*)#	1
Colistin (*n*)#	1	Azathioprine (*n*)	1
Tigecycline (*n*)	1	Hydroxyurea (*n*)*	2
Meropenem (*n*)#	1	Allopurinol (*n*)	2
Amphotericin (*n*)#	1	Febuxostat (*n*)	1
Fluconazole (*n*)#	2	Hydroxychloroquine (*n*)#	1
Albendazole (*n*)	1	Noradrenaline (*n*)#	2
Ivermectin (*n*)	1	Diltiazem (*n*)#	1
Artesunate (*n*)	1	Valproate (*n*)#	2
Entecavir (*n*)#	1	Octreotide (*n*)*	1
Oseltamivir (*n*)	1		

#Studies report drug causation or association with hypokalaemia; *Drug usually causes hyperkalaemia

which could have helped in the diagnosis of other underlying disorders.

Conclusion

The incidence of hypokalaemia among hospitalised patients admitted under general medicine was comparable to other studies. DM, hypertension, hypothyroidism and treatment, such as diuretics and antibiotics, contributed to the development of hypokalaemia, which was mostly mild in nature without causing cardiovascular or neurological adverse events. Nearly 88% of the patients had at least one risk factor for developing hypokalaemia. The workup for hypokalaemia was less than optimal considering the obvious risk factors, rapid recovery and enhanced costs.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Lippi G, Favaloro EJ, Montagnana M, Guidi GC. Prevalence of hypokalaemia: The experience of a large academic hospital: Letters to the Editor. Intern Med J 2010;40:315–6.
2. Udensi U, Tchounwou P. Potassium homeostasis, oxidative stress, and human disease. Int J Clin Exp Physiol 2017;4:111.
3. El-Sherif N, Turitto G. Electrolyte disorders and arrhythmogenesis. Cardiol J 2011;18:233–45.
4. Weir MR, Espaillat R. Clinical perspectives on the rationale for potassium supplementation. Postgrad Med 2015;127:539–48.
5. Gennari FJ. Disorders of potassium homeostasis. Crit Care Clin 2002;18:273–88.
6. Kardalas E, Paschou SA, Anagnostis P, Muscogiuri G, Siasos G, Vryonidou A. Hypokalemia: A clinical update. Endocr. Connect 2018;7:R135–46.
7. Rose BD, Post TW. Clinical Physiology of Acid-Base and Electrolyte Disorders. 5th ed. New York: McGraw-Hill; 2001. p. 836–57.
8. Elliott TL, Braun M. Electrolytes: Potassium disorders. Fam Pract Essent 2017;459:1–8.
9. Zuccala G, Pedone C, Cocchi A, Pahor M, Carosella L, Carbonin P, *et al.* Older age and in-hospital development of hypokalemia from loop diuretics: Results from a multicenter survey. J Gerontol A Biol Sci Med Sci 2000;55:M232–8.
10. Lawson DH, Henry DA, Lowe JM, Gray JM, Morgan HG. Severe hypokalemia in hospitalized patients. Arch Intern Med 1979;139:978–80.
11. Janko O, Seier J, Zazgornik J. [Hypokalemia--incidence and severity in a general hospital]. Wien Med Wochenschr 1992;142:78–81.
12. Crop MJ, Hoorn EJ, Lindemans J, Zietse R. Hypokalaemia and subsequent hyperkalaemia in hospitalized patients. Nephrol Dial Transplant 2007;22:3471–7.
13. Widodo D, Setiawan B, Chen K, Nainggolan L, Santoso WD. The prevalence of hypokalemia in hospitalized patients with infectious diseases problem at Cipto Mangunkusumo Hospital, Jakarta. Acta Med Indones 2006;38:202–5.
14. Paice BJ, Paterson KR, Onyanga-Omara F, Donnelly T, Gray JM, Lawson DH. Record linkage study of hypokalaemia in hospitalized patients. Postgrad Med J 1986;62:187–91.
15. Albeladi FI, Wahby Salem IM, Albandar AA, Almusaylim HA, Albandar AS. Electrolyte imbalance in infectious disease patients at King Abdulaziz Hospital, Jeddah. J Taibah Univ Med Sci 2022;17:256–63.
16. Crook MA. Hypophosphataemia and hypokalaemia in patients with hypomagnesaemia. Br J Biomed Sci 1994;51:24–7.
17. Veltri KT, Mason C. Medication-induced hypokalemia. *P* T 2015;40:185–90.
18. Krogager ML, Mortensen RN, Lund PE, Bøggild H, Hansen SM, Kragholm K, *et al.* Risk of developing hypokalemia in patients with hypertension treated with combination antihypertensive therapy. Hypertension 2020;75:966–72.
19. Zhu Q, Li X, Tan F, Deng Y, Gong C, Hu J, *et al.* Prevalence and risk factors for hypokalemia in patients scheduled for laparoscopic colorectal resection and its association with post-operative recovery. BMC Gastroenterol 2018;18:152.
20. Falcone C, Compostella L, Camardo A, Truong LVS, Centofanti F. Hypokalemia during antibiotic treatment for bone and joint infections. Eur J Orthop Surg Traumatol 2018;28:389–95.
21. Zhou Z, Huang C, Fu P, Huang H, Zhang Q, Wu X, *et al.* Prediction of in-hospital hypokalemia using machine learning and first hospitalization day records in patients with traumatic brain injury. CNS Neurosci Ther 2023;29:181-91.
22. Yin J, Yuan N, Huang Z, Hu Z, Bao Q, Shao Z, *et al*. Assessment of hypokalemia and clinical prognosis in Patients with COVID-19 in Yangzhou, China. PLoS One 20228;17:e0271132.
23. Kuramoto H, Masago S, Kashiwagi Y, Maeda M. Incidence and risk factors of hypokalemia in tazobactam/piperacillin-administered patients. Yakugaku Zasshi 2019;139:1591–600.
24. Schlaeffer F. Oxacillin-associated hypokalemia. Drug Intell Clin Pharm 1988;22:695–6.
25. Driemeyer C, Poloni JAT, Ulysséa LMT, Pasqualotto AC. Vancomycin-induced hypokalemia: A proof-of-concept case report. Clin Chimica Acta 2020;510:232–4.
26. Heijden CDCC, Duizer ML, Fleuren HWHA, Veldman BA, Sprong T, Dofferhoff ATSM, *et al.* Intravenous flucloxacillin treatment is associated with a high incidence of hypokalaemia. Br J Clin Pharmacol 2019;85:2886–90.
27. Karimzadeh I, Jafari M, Davani-Davari D, Ramzi M. The pattern of cyclosporine nephrotoxicity and urinary kidney injury molecule 1 in allogenic hematopoietic stem cell transplant patients. Exp Clin Transplant 2021;19:553–62.
28. Kidd D, Ranaghan EA, Morris TCM. Hypokalaemia in patients with acute myeloid leukaemia after treatment with fluconazole. Lancet 1989;333:1017.
29. Chávez-Iñiguez JS, Medina-Gonzalez R, Aguilar-Parra L, Torres-Vázquez EJ, Maggiani-Aguilera P, Cervantes-Pérez E, *et al.* Oral acyclovir induced hypokalemia and acute tubular necrosis a case report. BMC Nephrol 2018;19:324.
30. Rehan HS, Hotha P. Antimicrobial agents-induced hypokalemia: A possible causality association. Indian J Crit Care Med 2019;23:175–7.
31. Scoglio M, Bronz G, Rinoldi PO, Faré PB, Betti C, Bianchetti MG, *et al.* Electrolyte and Acid-Base Disorders Triggered by Aminoglycoside or Colistin Therapy: A Systematic Review. Antibiotics 2021;10:140.
32. Anuhya TV. Meropenem induced hypokalemia. J Clin Diagn Res 2017;11:OD05-06.
33. Fujii T, Kawasoe K, Ohta A, Nitta K. A case of entecavir-induced Fanconi syndrome. CEN Case Rep 2019;8:256–60.
34. Cheema MA, Zain MA, Cheema K, Ullah W. Thyroxine-induced periodic paralysis: A rare complication of nutritional supplements. BMJ Case Rep 2018;11:e227946.
35. Hershkowitz E, Reshef A, Munich O, Yosefi B, Markel A. Thiamine deficiency in self-induced refeeding syndrome, an undetected and potentially lethal condition. Case Rep Med 2014;2014:605707.
36. Vasavada A, Sanghavi D. Cyanocobalamin. *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2022. Available from: http://www.ncbi.nlm.nih.gov/books/NBK555964/. [Last accessed on 2022 Oct 29].
37. de Oliveira RA, Marques IDB, Seguro AC, Andrade L. Electrolyte disturbances and acute kidney injury induced by imatinib therapy. Clin Kidney J 2009;2:27–9.
38. Radke JB, Kingery JM, Maakestad J, Krasowski MD. Diagnostic pitfalls and laboratory test interference after hydroxychloroquine intoxication: A case report. Toxicol Rep 2019;6:1040–6.

39. Wang C, Zhou Y, Song L, Deng Z, Fang W. Valproic-induced Fanconi syndrome: Clinical features, risk factors, diagnosis and management. Front Med 2022;9:945244.
40. Moratinos J, Reverte M. Effects of catecholamines on plasma potassium: The role of alpha- and beta-adrenoceptors. Fundament Clin Pharmacol 1993;7:143–53.
41. Greco A, Rabito G, Pironi M, Bissig M, Parlato S, Andreocchi L, *et al.* Hypokalaemia in hospitalised patients. Swiss Med Wkly 2016;146:w14320.
42. Paltiel O, Salakhov E, Ronen I, Berg D, Israeli A. Management of severe hypokalemia in hospitalized patients: A study of quality of care based on computerized databases. Arch Intern Med 2001;161:1089.