ABSTRACT Objective: This study aimed to look into the occurrence and aetiology of potassium abnormalities (hypokalemia and hyperkalemia) in hospitalised patients and treatment options. Subjects and Methods: All hospitalized patients at the tertiary care centre who had at least one measurement of serum potassium throughout their stay were included in the study. The hospital records included information on electrolyte levels in the blood, demographic characteristics, admission reasons, drugs, the aetiology of potassium abnormalities, and treatment techniques. Results: A blood potassium anomaly was found in 1,265 (14.0%) of the 9,045 hospitalised patients; 604 (6.7%) had hypokalemia, and 661 (7.3%) had hyperkalemia. The most important explanations for hypokalemia were gastrointestinal losses in 555 (91.8%) patients and renal losses in 252 (41.7%) patients. Correcting the underlying cause and replenishing the potassium shortage were the most common therapy techniques. At hospital release, 319 (52.8%) of the 604 hypokalemic patients were normokalemic. Treatment with renin-angiotensin-aldosterone system blockers was the most common cause of hyperkalemia in 228 (34.4%) patients, followed by renal failure in 191. (28.8 percent). Patients were observed for 298 days (45.0%) without receiving any specific treatment. At hospital discharge, 324 (49.0 percent) of the 661 hyperkalemic patients were normal. Conclusion: The prevalence of potassium imbalance was high among hospitalised patients in this study. Although most of the potassium abnormalities were mild/moderate, about half of the patients treated for hypokalemia or hyperkalemia were discharged with persistent dyskalemia.

KEYWORDS Hypokalemia, Hyperkalemia, Disease induced, Drug-induced, Potassium supplements

Introduction

Potassium is an important electrolyte for cellular processes, and its serum content should be kept between 3.5 and 5.5 mEq/l [1].
nalised patients, as well as the causes of potassium abnormalities and treatment options.

Material and Methods

Patients

During a 4-month study period, all hospitalised patients (n = 9,045) in tertiary care centres who underwent at least one serum potassium measurement were included. In addition, those with potassium deficiencies had their records taken. Throughout the hospitalisation, potassium levels were monitored.

Methods

During the trial, a total of 21,435 potassium measurements were taken in 9,045 participants. The hospital’s electronic health records system obtained serum potassium levels. As soon as the samples arrived, all experiments were carried out utilising an ion-selective electrode technique with a Roche modular system. Hemolysis-positive samples were eliminated. The findings were divided into three categories: normokalemia (serum K+ = 3.5–5.0 mEq/l), hypokalemia (serum K+ <3.5 mEq/l), and hyperkalemia (serum K+ >5.0 mEq/l). If patients had hypokalemia and hyperkalemic measures while in the hospital, the earliest abnormal potassium result was taken as the current abnormal potassium result. The patients’ demographic and laboratory characteristics, such as age, gender, the reason for hospitalisation, comorbidities, medications, serum creatinine, sodium, calcium, phosphorus, magnesium, and albumin levels, as well as the results of arterial or venous blood gas analysis, urinalysis, and 24-hour urine tests, were recorded. Impaired oral intake, redistribution, gastrointestinal loss, and renal loss were studied as causes of hypokalemia. Impaired oral intake is characterised as ingesting only clear liquids or nothing by mouth. Pseudohyperkalemia, redistribution, aldosterone shortage or resistance, and reduced renal excretion were all explored as possible causes in hyperkalemic patients. The therapy techniques for patients with potassium anomalies, as well as the treatment results, were documented. All therapies were carried out by the patients’ doctors, with no intervention from the researchers.

Statistical Methods

All statistical analyses were performed using the Statistical Package for Social Sciences version 18, (SPSS Inc., Chicago, Ill., USA). Descriptive statistics were used to define demographics and the frequency of dyskalemia. Categorical variables were compared by means of the $\chi^2$ test. $p < 0.05$ was considered statistically significant.

Table 1. Distribution of potassium levels in all measurements

| Potassium level | Patients, n (%) |
|----------------|----------------|
| Hypokalemia    | 233 (38.6)     |
| Normokalemia   | 319 (52.8)     |
| Hyperkalemia   | 52 (8.6)       |

Results

Prevalence of Dyskalemia

Table 1 displays the distribution of serum potassium measurement findings. 5,216 (24.3 percent) of the 21,435 measurements were abnormal. There were 1,265 (14.0%) potassium abnormalities among the 9,045 hospitalised patients, with hypokalemia in 604 (47.7%) and hyperkalemia in 661 (52.3 percent). 451 of the 661 hyperkalemic patients (68.2%) were men, while 358 of the 604 hypokalemic patients were women (59.2 percent).

Hypokalemia

The hypokalemic patients had a mean age of 54.4 ± 16.3 years and a mean serum potassium level of 3.25 ± 0.24 mEq/l. 371 (61.4%) of the 604 hypokalemic patients were admitted to surgical wards. The severity of hypokalemia was as follows: severe (K+ <2.5 mEq/l) in 8 patients (1.3%), moderate (K+ 2.5–2.99 mEq/l) in 68 patients (11.3%), and mild (K+ 3.0–3.49 mEq/l) in 528 individuals (87.4 percent). In 555 individuals (91.8 percent), impaired oral intake or evidence of gastrointestinal potassium loss (primarily owing to vomiting and diarrhoea) or renal loss (mostly due to diuretic medication) were the causes of hypokalemia (41.7 percent). Catecholamine excess, insulin therapy, intravenous hydration without potassium replacement, and hypomagnesemia were among the less common causes. Correcting the underlying causes by ensuring enough oral intake and discontinuing diuretics in 401 patients (66.4%), and intravenous or oral potassium supplementation in 255 (42.2%) and 144 (23.8%) patients, respectively, were the main treatment choices for hypokalemia. Other less prevalent methods included taking aldosterone receptor blockers or taking magnesium supplements. 36 hypokalemic patients received no specific treatment (6.0 percent). Serum potassium levels at discharge after therapy were as follows: severe hypokalemia (K+ <2.5 mEq/l) in four patients (1.7%), severe hyperkalemia (K+ ≥ 6 mEq/l) in seven (13.5%), and normokalemia in 319 (52.8%). (table 2).

Hyperkalemia

The hyperkalemic patients’ average age was 56.8 ± 17.0 years, and their mean serum potassium level was 5.4 ± 0.4 mEq/l. 397 (60.1%) of the 661 hyperkalemic patients were admitted to surgical wards. The severity of hyperkalemia was distributed as follows: mild (K+ 5–5.49 mEq/l) in 483 (73.1%), moderate (K+ 5.5–5.99 mEq/l) in 124 (18.8%), and severe (K+ ≥6.0 mEq/l) in 54. (8.2 percent). In 225 (34.4%) patients, hyperkalemia was caused by the use of renin-angiotensin system (RAS) blockers or nonsteroidal anti-inflammatory medications (NSAIDs), while 178 had reduced renal function (27 percent). Diuretics, intravenous dextrose insulin, and inhaled salbutamol were the most commonly employed treatment methods. In 52 hyperkalemic patients, hemodialysis was conducted (7.9 percent). No specific
treatment was reported for 298 (45.0%) hyperkalemic individuals. The following were the serum potassium levels at the time of discharge after treatment for hyperkalemia: hypokalemia in 43 patients (6.5%), normokalemia in 324 (49.5%), and hyperkalemia in 294 patients (44.5 percent). As a result, 337 individuals (51.0%) had abnormal potassium levels upon discharge.

Discussion

This study found a high prevalence (24.3 percent) of serum potassium abnormalities in hospitalized patients, within the range of 13.7–48.0 percent previously reported [3, 7, 9, 10]. The wide range could be due to the different cut-off points and patient characteristics used in these studies. The prevalence of hypokalemia in hospitalised patients in this study was 6.7 percent, compared to 12–20 percent in earlier studies [9, 11], which could be attributable to the varying cut-off points utilized in the studies patient characteristics. Hypokalemia was caused by a decrease in oral intake as well as gastrointestinal and renal loss. Another prominent cause was intravenous saline treatment without potassium supplementation in patients without oral intake; this is a preventable iatrogenic cause of hypokalemia. This study found that diuretic use was the primary source of potassium loss in the kidneys (41.7 percent of the cases). Diuretics were found to be a significant cause of hypokalemia (39.0 percent) in a prior investigation of a geriatric population [12]. Although our study did not include any follow-up data, it is widely recognised that diuretic-induced hypokalemia increases the incidence of arrhythmia. In diuretic-treated individuals, the frequency of malignant ventricular arrhythmias increases 2-fold when serum potassium levels are less than 3 mEq/l [13]. Hypokalemia also lengthens the stay in the hospital [14]. In this investigation, oral or intravenous potassium replacement was the most common hypokalemia therapy. Total parenteral feeding was provided to some individuals who had insufficient oral intake. Iatrogenic hyperkalemia has previously been associated with total parenteral feeding [9]. More than half of the hyperkalemia patients in this study were released with aberrant potassium levels, and 8.6% were discharged with iatrogenic, overshoot hyperkalemia. These findings highlight the importance of carefully scrutinising hospital practices in evaluating and managing hypokalemia (e.g., the excessive use of diuretics or intravenous fluid replacement without any additional potassium as potential causes of hypokalemia or the inadvertent use of total parenteral nutrition as a cause of overshoot hyperkalemia).

Hyperkalemia was present in 7.3 percent of the hospitalised patients in this investigation. Severe hyperkalemia was found in 8.2 percent of the patients, which is consistent with earlier observations [15, 16]. Medication (NSAIDs, RAS blockers, beta-blockers, spironolactone, and heparin), chronic kidney disease, insulin insufficiency, and hemolysis were the main causes of hyperkalemia in these patients. As in earlier investigations, RAS blockers were the most common cause of hyperkalemia. RAS blockers enhance the likelihood of hyperkalemia in patients with a blood creatinine level >1.5 mg/dl [17]. After one year of treatment, RAS blockers cause hyperkalemia in 10–38 percent of hospitalised patients and 10% outpatients. Patients with diabetes and chronic renal disease are at a higher risk of hyperkalemia [10, 18–21]. According to previous research, 22–25 percent of hyperkalemic hospitalised patients got iatrogenic potassium replacement [22], with most of these patients also receiving diuretic treatment to prevent hypokalemia [16]. Because there were more patients with congestive heart failure in this trial than in previous ones, diuretics and beta-blockers were used more frequently. Patients in the medical and surgical wards had diverse causes of hyperkalemia, with the main causes being RAS blockers and postoperative NSAIDs, respectively. In appropriate clinical conditions, some unusual causes of hyperkalemia should be explored, such as accidental potassium consumption [23]. In this study, over 8% of the patients required hemodialysis to manage hyperkalemia. The burden of hyperkalemia is increased by this treatment, both for the patient and for hospital resources. In a prior study, patients with hyperkalemia had a 17.0 percent mortality rate. However, the exact relationship between hyperkalemia and mortality remained unclear [24]. The increased use of RAS blockers in individuals with congestive heart failure and chronic renal disease may cause high mortality. In such circumstances, more research into the role of hyperkalemia in mortality is needed.

Almost 45.0 percent of hyperkalemic individuals were not treated and were followed up on. Potassium levels in most of these patients were between 5.0 and 5.5 mEq/l. We found that 6.5 percent of patients with hyperkalemia were discharged with a serum potassium level less than 3.5 mEq/l, while 44.5 percent were discharged with a level greater than 5 mEq/l. Although most of the blood potassium anomalies at the time of discharge were minor, there were a few cases where the potassium levels were >6.0 mEq/l—discharging more than half of the patients with a potassium level beyond the normal range signals that hyperkalemia treatment was insufficient and raises the risk of increased morbidity and mortality. This study has significant limitations, including a lack of data on long-term morbidity, follow-up, and utilisation of dyskalemia-related hospital resources, as well as a lack of data on the specific cause of potassium abnormalities treatment techniques.

Conclusion

Potassium abnormalities were found to be common among hospitalised individuals in this investigation. Although the majority of the potassium anomalies were mild to moderate, there were a number of severe cases where basic hospital surveillance could prevent serious morbidity and mortality. Furthermore, nearly half of patients treated for hypokalemia or hyperkalemia were discharged from the hospital with persistent dyskalemia. More research is needed to evaluate morbidity and mortality and the hazards associated with a dyskalemia-related duration of stay in the hospital and discharge with potassium abnormalities.

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Conflict of interest

There are no conflicts of interest to declare by any of the authors of this study.

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