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ORIGINAL ARTICLE

Knowledge of medical specialists on the emergency management of hyperkalaemia with a focus on insulin-based therapy

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ABSTRACT

Introduction: Hyperkalaemia is a common electrolyte disorder in hospitalised patients and may cause life-threatening cardiac arrythmias and death. There is a lack of consensus regarding its optimal management, which may result in wide variations in practice and the guidance provided to junior staff.

Methods: We conducted a survey on a Research Electronic Data Capture (REDCap) platform to evaluate the knowledge of medical specialists regarding the diagnosis and management of hyperkalaemia, with a focus on insulin-based therapy. A convenience sample of 70 specialists in nephrology, internal medicine, emergency medicine and critical-care medicine were invited to participate. Comparisons were also made between nephrologists and non-nephrologists.

Results: A total of 51 medical specialists responded, of whom 47% were nephrologists. They were more likely to initiate therapy at a potassium concentration ([K]) of 6 mmol/L, whereas non-nephrologists tended to start at a lower concentration ($P < 0.01$). Half the respondents regarded blood gas machine measurements as providing an accurate measure of [K]. Non-nephrologists were more likely to perform an ECG before starting treatment ($P = 0.02$). All respondents regarded insulin and dextrose as the most effective and reliable means for shifting K. Only 22% monitored the serum glucose concentration beyond 2 hours following insulin-based therapy, and 22% thought that hypoglycaemia was an uncommon complication if dextrose also was administered.

Conclusions: This is the first comprehensive survey to report on the knowledge of specialists regarding the emergency management of hyperkalaemia. There is a need to address knowledge gaps, particularly around the optimal and safe use of insulin-based therapies. Our findings and recommendations should be useful in informing the development of consensus guidelines and educational resources on hyperkalaemia.

Keywords: hypoglycaemia; insulin; dextrose; glucose; electrocardiogram.

INTRODUCTION

Hyperkalaemia is a common electrolyte disorder encountered in hospitalised patients, with a reported incidence rate of 2.8 cases per 100 patient-years and a prevalence rate of 6.3% [1,2]. Since potassium (K) is responsible for maintaining the resting membrane potential of skeletal muscle cells and the conducting system of the heart, hyperkalaemia may result in respiratory muscle weakness, cardiac arrhythmias, and death [3].

Principles of management include protecting the heart using calcium salts, shifting K intracellularly using insulin and dextrose, beta-2 agonist nebulisations and sodium bicarbonate when hyperkalaemia is accompanied by acidosis, eliminating K from the body via the kidneys and gastrointestinal tract, and reducing sources of K and stopping drugs that interfere with the renal elimination of potassium.

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Insulin-based therapy is the most favoured pharmacological method for treating hyperkalaemia [4-6], although few randomised controlled trials have been performed [7,8]. Dextrose is usually co-administered to prevent hypoglycaemia. Various recommendations exist regarding the dosing, sequence and rates of administration for insulin and dextrose [5,9]. As a result, there are wide variations in practice [10]. Hypoglycaemia, a serious complication of insulin-based therapy, has been reported to occur in as many as 75% of patients [5]. Factors that may influence the risk of this complication include the dose of insulin administered [11], the dose of dextrose [12], the baseline serum glucose concentration [12-15] and the presence of kidney failure [11].

There is a paucity of data on the knowledge and practice patterns of doctors regarding the emergency management of hyperkalaemia. A survey that assessed the knowledge of trainees reported a low overall score of 52% [16]. The lowest scores were achieved for knowledge regarding the normal [K] range, the threshold for treating hyperkalaemia and drugs that may result in hyperkalaemia. Another survey of paediatricians regarding the choice and sequence of administration of drugs during cardiac arrest associated with hyperkalaemia reported significant variability and recommended that a standardised approach be developed to improve management [17]. Lastly, a survey of the practice patterns of residents and specialists regarding the treatment of hyperkalaemia in the emergency department reported wide variation in practice. The authors speculated that this may have resulted from concerns regarding the risk of hypoglycaemia when insulin-based therapy was used in patients with kidney failure [10].

In summary, since there is a lack of consensus and a paucity of data regarding the knowledge of medical specialists on the emergency management of hyperkalaemia and many medical specialists are involved in training medical students and junior colleagues, we conducted this survey to identify their current knowledge and practice patterns, with a focus on insulin-based therapy. The aim was to identify knowledge gaps and to inform the development of learning resources to guide the optimal management of this life-threatening condition.

METHODS

We performed a survey on a convenience sample of medical specialists who frequently encounter patients with hyperkalaemia in their clinical practice. Participants were asked to complete the survey according to their current practice.

Items that were included in the survey (Tables 1 and 2, supplementary Tables S1, S2 and S3) were selected based on published guidelines [18-20], protocols in major textbooks [21-23] and also drew on our own experience. They focused on the following aspects: diagnostic tests which would guide treatment such as blood tests and the electrocardiogram (ECG), pharmacological and non-pharmacological therapies with a focus on insulin-based therapies, and the treatment-related adverse event of hypoglycaemia (Tables 1 and 2).

Survey items on diagnostic tests included the potassium concentration ([K]) that should trigger therapy, and the utility of the ECG and point-of-care potassium measurements.

Items on pharmacological management included protecting the heart using calcium salts, shifting K into cells using insulin-based therapy, eliminating potassium from the body, and removing sources of potassium intake and stopping drugs that interfere with renal potassium excretion.

In relation to insulin, the survey items tested knowledge of the type of insulin to be used, and the doses and rates of administration. This included the time required for the [K] to reach its nadir, the expected magnitude of the drop, whether [K] was expected to return to pre-shift concentrations, and the definition of refractory hyperkalaemia.

Regarding treatment-related adverse events, we included items on the anticipation of hypoglycaemia when shifting potassium with insulin, monitoring of blood glucose concentrations, and the dose, route, and timing of dextrose administration.

The survey was developed and managed using the Research Electronic Data Capture (REDCap) platform [24]. After distribution, email reminders were sent weekly until a threshold response rate of at least 60% was achieved. Permission to conduct this study was granted by the Health Research Ethics Committee (HREC) of Stellenbosch University (HREC study number: 10988).

Data analysis

Data were exported from REDCap into Stata version 16.1 (StataCorp LLC, Texas, USA) and summarised using counts and percentages. Chi-squared or Fisher's exact tests were used to compare choices between nephrologists and other specialists. Statistical significance was regarded as a P value of less than 0.05 and 95% confidence intervals were used.

RESULTS

A total of 51 of 70 specialists responded, a rate of 73%. Of these, 24 (47%) were nephrologists and the remainder were specialist physicians (18, 35%), emergency medicine physicians (7, 14%) and intensivists (2, 4%).

[K] prompting therapy and diagnostic tests (Table 1 and Table S1)

Nearly half the respondents (43%) selected a [K] of 6 mmol/L as the threshold value that would prompt their treatment of hyperkalaemia; 63% of the nephrologists selected 6 mmol/L as compared with only 26% of the non-nephrologists ($P < 0.01$) (Figure 1).

Most respondents (96%) regarded laboratory K measurements as accurate whereas only half considered point-of-care measurements on blood gas samples as accurate.

Two-thirds of respondents routinely performed an ECG before deciding whether a patient required treatment for hyperkalaemia, with more non-nephrologists performing an ECG ($P = 0.02$). Nearly three quarters of respondents thought that there was poor correlation between [K] and the presence of ECG changes. The ECG change that would most frequently prompt the initiation of treatment was tall, tented T waves (94%), whereas broad QRS complexes, ventricular tachycardia/fibrillation, flattened or absent P waves, sine waves and sinus bradycardia were selected by 86%, 77%, 77%, 71% and 57% of respondents, respectively.

Pharmacological management of hyperkalaemia (Table 2 and Table S2)

Most of the respondents indicated that they would invariably administer calcium salts. Of those who reported that they would not use calcium salts routinely, 24% commented that they would use them only when hyperkalaemia was associated with ECG changes.

Regarding therapies used during the emergency treatment, the two most frequently employed therapies were insulin and dextrose (100%) and calcium salts (96%). All respondents selected insulin and dextrose as the most effective and reliable means for shifting K into cells and used short-acting insulin. Nearly three quarters used a dose of 10 units of insulin. Insulin was administered as a push (bolus) by half, whereas a quarter administered it over 30 minutes. Less than half expected the [K] value to reach its nadir at 60 minutes. Most (63%) expected an average decrease in the serum [K] of 1 mmol/L. Most (61%) anticipated serum [K] to return to its pre-shift value and expected it to occur at 2–3 hours following treatment with insulin (63%). Just over half defined refractory hyperkalaemia as two or more shifts

Table 1. Potassium concentration prompting therapy, and diagnostic tests.

	All (n = 51)	Other (n = 27)	Nephrologist (n = 24)	P value
	Diagnostic			
Serum [K] prompting treatment (mmol/L)				
>5.0	1 (2)†	1 (4)	0 (0)	<0.01
>5.5	13 (26)	12 (44)	1 (4)	
>6.0*	22 (43)	7 (26)	15 (63)	
>6.5	13 (26)	5 (19)	8 (33)	
>7.0	2 (4)	2 (7)	0 (0)	
Accurate method of measurement (select all that apply)				
Blood gas	25 (49)	14 (53)	11 (46)	0.67
Laboratory*	49 (96)	26 (96)	23 (96)	1.00
Routine ECG?				
Yes*	34 (67)	22 (82)	12 (50)	0.02
No	17 (33)	5 (19)	12 (50)	
Correlation between serum [K] and the ECG				
Yes	11 (22)	6 (22)	5 (21)	0.86
No*	38 (75)	21 (78)	17 (71)	
Not sure	1 (2)	0 (0)	1 (4)	
ECG changes prompting treatment (select all that apply)				
Sinus bradycardia*	29 (57)	14 (52)	15 (63)	0.44
Flattened or absent P waves*	39 (77)	21 (78)	18 (75)	0.81
Broad QRS complexes*	44 (86)	24 (89)	20 (83)	0.69
Tall, tented T waves	48 (94)	26 (96)	22 (92)	0.59
Sine waves*	36 (71)	18 (67)	18 (75)	0.51
Ventricular tachycardia/fibrillation*	39 (77)	19 (70)	20 (83)	0.28

Abbreviation: ECG, electrocardiogram. [K], potassium concentration. *Preferred or correct option(s). †Percentage in brackets.

required within 24 hours. Most (94%) had safety concerns when using insulin therapy, mainly related to the risk of hypoglycaemia.

Nearly all respondents (82%) routinely checked blood glucose concentration before administering insulin treatment. Most used 50 mL (57%) or 100 mL (41%) of 50% dextrose. Approximately half administered the dextrose mixed with insulin whereas the remainder provided it before (37%) or after (11%) insulin administration. Nearly all (96%) administered this treatment via the intravenous route. One third expected hypoglycaemia to occur within one hour following the administration of insulin and dextrose, another third expected hypoglycaemia after 1–2 hours, and 22% thought that hypoglycaemia was uncommon if dextrose was administered. Two-thirds checked the blood glucose concentration before insulin administration whereas only 22% checked it at 3 hours after insulin administration (Figure 2).

K elimination

Although 80% of the study population used drugs to eliminate K from the body, nearly all the nephrologists made use of this treatment (96% vs. 67%, $P < 0.01$). Drug classes used included K-binding resins (85%), loop diuretics (76%), cathartics (42%) and intravenous sodium bicarbonate (42%). Most of the respondents (88%) were aware of serious gastrointestinal adverse effects, particularly colonic ulceration/colitis, colonic necrosis, and bowel obstruction associated with the use of K-binding resin.

Non-pharmacological management (Table S3)

Nearly all respondents routinely stopped foods with a high potassium content (88%) and drugs that interfere with the renal elimination of K (96%).

DISCUSSION

We identified important shortcomings regarding the knowledge and management of hyperkalaemia among medical specialists. These included tented T waves as the most common ECG change to trigger therapy, the time for [K] to reach its nadir following insulin administration, whether the potassium concentration would return to its pre-shift value and when this was expected to occur, defining refractory hyperkalaemia, and the expectation and surveillance of hypoglycaemia following insulin-based therapy.

On the other hand, most of the respondents were aware that the ECG correlated poorly with [K], that short-acting insulin and dextrose was the most reliable method for shifting K and that Kexelate use was associated with colonic/bowel necrosis.

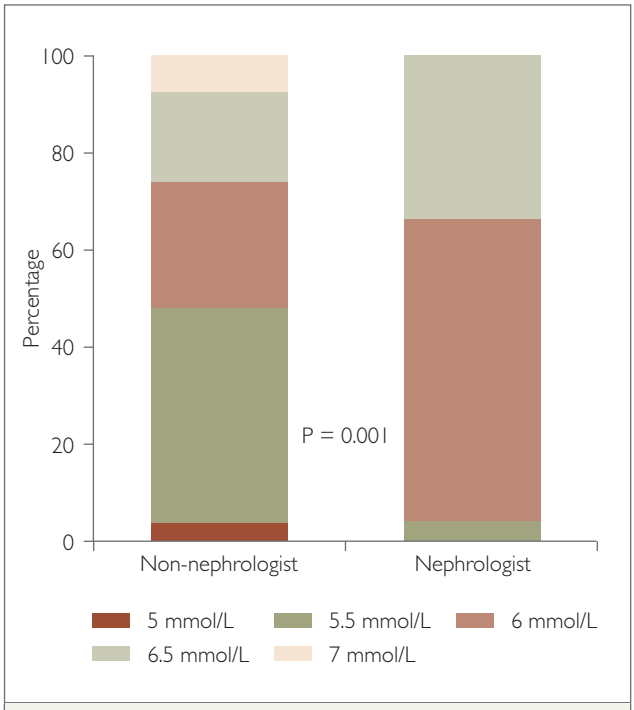

Figure 1. Threshold potassium concentrations prompting treatment of hyperkalaemia by nephrologists and non-nephrologists.

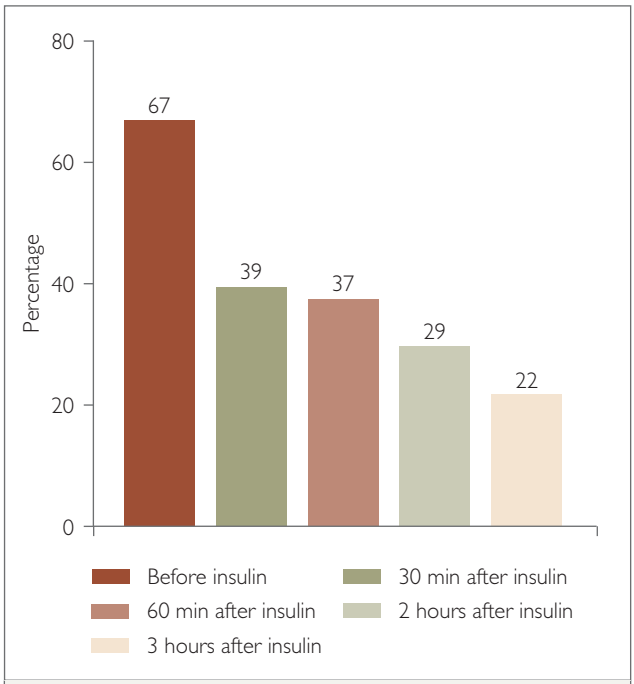

Figure 2. Proportion of medical specialists checking the capillary glucose concentrations before, as well as 30 minutes, 60 minutes, 2 hours and 3 hours after insulin administration.

Nephrologists tended to start therapy at a higher range of serum [K] (6.0–6.5 mmol/L), defined by many as moderate to severe hyperkalaemia [9,25]. We speculate that this is because of the high frequency with which they encounter patients with severe hyperkalaemia. A recent KDIGO con-

Table 2. Pharmacological management of hyperkalaemia.

	All (n = 51)	Other (n = 27)	Nephrologist (n = 24)	P value
K-lowering therapies				
Therapies frequently used				
Calcium salts	49 (96)†	26 (96)	23 (96)	1.00
Beta-2 agonist nebulisations	20 (39)	13 (48)	7 (29)	0.17
Intravenous sodium bicarbonate	18 (35)	8 (30)	10 (42)	0.37
Insulin and dextrose	51 (100)	27 (100)	24 (100)	-
Potassium-binding resins	28 (55)	13 (48)	15 (63)	0.30
Loop diuretics	18 (35)	8 (30)	10 (42)	0.37
Protect the heart				
Routine calcium salt administration				
Yes	36 (71)	18 (67)	18 (75)	0.51
No*	15 (29)	7 (33)	6 (25)	
Shift K into cells				
Most effective and reliable method for shifting potassium				
Intravenous insulin and dextrose*	51 (100)	27 (100)	24 (100)	-
Beta-2 agonist nebulisations	0 (0)	0 (0)	0 (0)	
Intravenous sodium bicarbonate	0 (0)	0 (0)	0 (0)	
Intravenous dextrose only	0 (0)	0 (0)	0 (0)	
Type of insulin used				
Short acting*	51 (100)	27 (100)	24 (100)	-
Intermediate acting	0 (0)	0 (0)	0 (0)	
Long acting	0 (0)	0 (0)	0 (0)	
Dose of insulin used (units)				
5	9 (18)	3 (11)	6 (25)	0.14
10*	38 (74)	22 (82)	16 (67)	
15	1 (2)	0 (0)	1 (4)	
20	2 (4)	2 (7)	0 (0)	
30	1 (2)	0 (0)	1 (4)	
Rate of insulin administration				
Push (Bolus)*	27 (53)	13 (48)	14 (58)	0.30
Over 5 min	7 (14)	4 (15)	3 (13)	
Over 30 min	13 (26)	6 (22)	7 (29)	
Over 1 h	4 (8)	4 (15)	0 (0)	
Over 2 h	0 (0)	0 (0)	0 (0)	
Expected time to reach its lowest concentration (min)				
15	10 (20)	6 (22)	4 (17)	0.20
30	10 (20)	6 (22)	4 (17)	
45	5 (10)	4 (15)	1 (4)	
60*	22 (43)	11 (41)	11 (46)	
120	4 (8)	0 (0)	4 (17)	
Expected average decrease in the serum potassium concentration (mmol/L)				
0.5	4 (8)	1 (4)	3 (13)	0.50
1.0*	32 (63)	17 (63)	15 (61)	
1.5	13 (26)	7 (26)	6 (25)	
2.0	2 (4)	2 (7)	0 (0)	
2.5	0 (0)	0 (0)	0 (0)	
Anticipation of serum [K] to return to its pre-shift value				
Yes*	31 (61)	15 (56)	16 (67)	0.61
No	13 (26)	7 (26)	6 (25)	
Unsure	7 (14)	5 (19)	2 (8)	
Timing of serum [K] to return to its pre-shift value (h)	N = 30	N = 14	N = 16	
2–3	19 (63)	7 (50)	12 (75)	0.26
4–6*	9 (30)	5 (36)	4 (25)	
≥7	2 (7)	2 (14)	0 (0)	

Table 2 continued. Pharmacological management of hyperkalaemia.

	All (n = 51)	Other (n = 27)	Nephrologist (n = 24)	P value
	Shift K into cells			
Defining refractory hyperkalaemia				
Serum K does not decrease after insulin therapy*	20 (39)	9 (33)	11 (46)	0.58
Two or more shifts required within 24 h	27 (53)	15 (56)	12 (50)	
Other	4 (8)	3 (11)	1 (4)	
Safety concerns when using insulin				
Yes*	48 (94)	25 (93)	23 (96)	1.00
No	3 (6)	2 (7)	1 (4)	
Checking blood glucose concentration before administering insulin				
Yes*	42 (82)	23 (85)	19 (79)	0.72
No	9	4 (15)	5 (21)	
Routine administration of dextrose?				
Yes*	46 (90)	23 (85)	23 (96)	0.35
No	5 (10)	4 (15)	1 (4)	
Volume of 50% dextrose administered when using insulin (mL)	N = 46	N = 23	N = 23	
50	26 (57)	13 (57)	13 (57)	1.00
100*	19 (41)	10 (44)	9 (39)	
200	0 (0)	0 (0)	0 (0)	
300	1 (2)	0 (0)	1 (4)	
Timing of dextrose administration	N = 46	N = 23	N = 23	
Before insulin*	17 (37)	8 (35)	9 (39)	0.29
Mixed with insulin	24 (52)	14 (61)	10 (44)	
After insulin	5 (11)	1 (4)	4 (17)	
Route of dextrose administration				
Oral	1 (2)	1 (4)	0 (0)	1.00
Intravenous*	49 (96)	26 (96)	23 (96)	
Timing of hypoglycaemia after insulin-based therapy				
0–1 h after	17 (33)	10 (37)	7 (29)	0.85
1–2 h after	16 (31)	9 (33)	7 (29)	
2–3 h after*	7 (15)	3 (11)	4 (17)	
Hypoglycaemia is uncommon if dextrose is given	11 (22)	5 (19)	6 (25)	
Checking blood glucose concentration relative to insulin (select all that apply)				
Before	34 (67)	18 (67)	16 (67)	1.00
5 min after	7 (14)	5 (19)	2 (8)	0.42
30 min after	20 (39)	8 (30)	12 (50)	0.14
60 min after	19 (37)	8 (30)	11 (46)	0.23
2 h after*	15 (29)	5 (19)	10 (42)	0.07
3 h after*	11 (22)	6 (22)	5 (21)	0.90
I do not check the glucose concentration	0 (0)	0 (0)	0 (0)	-
	K elimination			
Routine use of drugs to eliminate K?				
Yes	41 (80)	18 (67)	23 (96)	<0.01
No	10 (20)	9 (33)	1 (4)	
Drug(s) used to eliminate K (select all that apply)	N = 41	N = 18	N = 23	
K-binding resins (Kexelate)	35 (85)	16 (89)	19 (83)	0.13
Loop diuretics	31 (76)	17 (94)	14 (20)	0.73
Cathartics	17 (42)	9 (50)	8 (35)	1.00
Intravenous sodium bicarbonate	17 (42)	8 (44)	9 (39)	0.55
Aware of serious adverse effect(s) of Kexelate?				
Yes*	45 (88)	24 (89)	21 (88)	1.00

Abbreviation: [K], potassium concentration. *Preferred or correct option(s). †Percentage in brackets.

troversies paper supports the selection made by nephrologists along with the accompaniment of ECG changes [9]. However, their recommendation was based on the currently available studies of stable, pre-dialysis patients. Thus, the [K] value recommended to prompt the emergency treatment has not been tested in acute, unstable patients.

Only half of the respondents considered blood gas [K] measurements as accurate. Since there are often delays in reporting the laboratory [K] value and the diagnostic accuracy of the ECG is poor, the blood gas machine is an important point-of-care tool in the emergency department. A recent study that compared the difference in measurement between a blood gas machine and laboratory samples found that the average blood gas measurements were 0.4 mmol/L lower than laboratory values, with this negative bias remaining constant across the hyperkalaemic range [26]. The authors suggested that clinicians should consider using blood gas K measurements but should make the adjustment for the 0.4 mmol/L difference in concentrations.

Most non-nephrologists indicated that they routinely performed an ECG before deciding whether treatment was needed. There is a poor correlation between the ECG findings of hyperkalaemia and serum [K] [27] and this was acknowledged by most respondents. However, many guidelines recommend that an ECG be performed when hyperkalaemia is a consideration [9,18], since it is easily performed in the emergency department and there are often delays in the reporting of laboratory K measurements.

Most respondents were prompted to treat hyperkalaemia when accompanied by classic changes involving P waves and QRS complexes, and ventricular tachycardia/fibrillation. However, although tall, tented T waves are the first classic ECG change to appear during the evolution of hyperkalaemia, previous studies have not demonstrated any association with adverse outcomes [28]. On the other hand, sinus bradycardia was the least common ECG finding to prompt therapy despite its high prevalence in patients with hyperkalaemia [29] and its strong association with short-term adverse outcomes [28].

Regarding pharmacological therapy, all the respondents indicated that they regarded insulin and dextrose therapy as the most effective and reliable method for shifting K into cells. This was not surprising because insulin-based therapy is regarded by most authorities as the cornerstone of treatment [4]. However, several knowledge gaps of concern were identified. Fewer than half of the respondents were aware that serum [K] would reach its nadir at 60 minutes after insulin was administered [5,30] and less than two-thirds indicated that they anticipated the [K] value to return to its pre-shift value. Only a third expected this to occur at 4–6 hours following insulin therapy. This may explain why more than half the respondents regarded refractory hyperkalaemia as two or more shifts required within 24 hours. Since the intracellular shifting effect lasts only up to 6 hours, several shifts may be required within a 24-hour period. We recommend that the term "refractory hyperkalaemia" be used when serum [K] does not decrease following a single attempt using insulin.

Hypoglycaemia is the most common and serious complication of insulin-based therapy, occurring three to six hours following therapy [30]. Of concern was the low expectation of hypoglycaemia by respondents, with only 14% anticipating hypoglycaemia between 2–3 hours after insulin administration and 22% indicating that hypoglycaemia was uncommon if dextrose was co-administered. Only 30% checked serum glucose concentration at 2 hours, and only 22% at 3 hours. A systematic review of the management of hyperkalaemia reported that hypoglycaemia occurred in up to 75% of patients [5]. Most of these episodes occurred when 25 g of dextrose (50 mL of 50% dextrose) were used. Lower baseline serum glucose concentrations have been associated with a higher risk of hypoglycaemia following treatment with insulin [12-15,31]. We recommend that, in patients with a serum glucose concentration of less than 10 mmol/L, 50 g of dextrose (100 mL of 50% dextrose) be used, and that patient symptoms and serum glucose concentrations be monitored for at least three hours, and up to six hours, following insulin-based therapy.

Nearly all nephrologists indicated that they routinely used drugs to eliminate K. Some of these therapies, such as K-binding resins, are of questionable efficacy during the emergency treatment [32]. We speculate that because nephrologists were more likely to be involved in the chronic care of patients prone to develop hyperkalaemia, they are more likely to prescribe therapies that would reduce total body K.

Based on the knowledge gaps identified and the appraisal of the current literature, we recommend the following:

1. A [K] ≥ 6 mmol/L should prompt the start of therapy, or any degree of hyperkalaemia which is accompanied by symptoms or ECG changes.
2. Point-of-care blood gas [K] measurements may be used in the emergency setting provided adjustment for the 0.4 mmol/L negative bias is made.
3. An ECG should be performed on all patients.
4. Calcium salts should be administered only when there are ECG changes.

5. All ECG changes, except tented T waves, should trigger therapy. This includes sinus bradycardia.
6. Short-acting insulin should be administered as a push (bolus) of 10 units, intravenously. Lower doses (5 units) should be considered in patients with chronic kidney disease and kidney failure [33].
7. In patients with a serum glucose concentration <10 mmol/L, 50 g of dextrose (100 mL of 50% dextrose) should be infused, before administering insulin.
8. Patients should be monitored for symptoms of hypoglycaemia and serum glucose concentrations measured hourly for at least three hours, and up to six hours, following insulin-based therapy.
9. The [K] should be checked one hour following insulin-based therapy as this is when the nadir is expected.
10. The term "refractory hyperkalaemia" should be used when serum [K] does not decrease at the one-hour time point following a single attempt using insulin-based therapy.

CONCLUSIONS

This is the first comprehensive survey to report on the knowledge of specialists regarding the emergency management of hyperkalaemia. There is a need to address knowledge gaps, particularly around the optimal and safe use of insulin-based therapies. Our findings and recommendations should be useful in informing the development of consensus guidelines and educational resources on hyperkalaemia.

Supplementary materials

Questions from the REDCap survey are provided as Tables S1, S2 and S3 in Appendix 1.

Conflicts of interest

No conflicts of interest to declare.

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APPENDIX 1: SUPPLEMENTARY TABLES

Table S1. Potassium concentration prompting therapy, and diagnostic tests.

Which serum potassium concentration would prompt your initiation of the emergency treatment for hyperkalaemia?
>5.0 mmol/L
>5.5 mmol/L
>6.0 mmol/L
>6.5 mmol/L
>7.0 mmol/L
Which method do you consider an accurate measure of the potassium concentration? (Select all that apply)
Blood gas – Yes/No
Laboratory – Yes/No
Is it your current practice to routinely do an electrocardiogram (ECG) before deciding if your patient requires treatment for hyperkalaemia?
Yes
No
Is there a good correlation between the serum potassium concentration and the presence of ECG changes during hyperkalaemia?
Yes
No
Not sure
Which ECG change would prompt your initiation of treatment for hyperkalaemia? (Select all that apply)
Sinus bradycardia
Flattened or absent P waves
Broad QRS complex
Tall, tented T waves
Sine waves
Ventricular tachycardia/fibrillation

Abbreviation: ECG, electrocardiogram; K+, potassium.

Table S2. Pharmacological management of hyperkalaemia.

Do you always administer calcium salts (e.g., calcium gluconate) to patients with hyperkalaemia?
- Yes
- No

Which pharmacological therapies do you frequently use during the emergency treatment of hyperkalaemia?
- Calcium salts
- Beta-2 agonist nebulisations
- Intravenous sodium bicarbonate
- Insulin and glucose
- Potassium-binding resins
- Loop diuretics

What do you consider to be the most effective and reliable method for shifting potassium into cells during hyperkalaemia?
- Insulin and glucose
- Beta-2 agonist nebulisations
- Intravenous sodium bicarbonate
- Intravenous glucose only

When using insulin to shift potassium into cells, which type do you use?
- Short acting
- Intermediate acting
- Long acting

When using insulin to shift potassium into cells, what dose do you routinely administer?
- 5 units
- 10 units
- 15 units
- 20 units
- 30 units

At what rate do you usually administer the insulin?
- Push (Bolus)
- Over 5 min
- Over 30 min
- Over 1 hour
- Over 2 hours

How long after administering insulin therapy do you expect the serum potassium to reach its lowest concentration?
- 15 min
- 30 min
- 45 min
- 60 min
- 120 min

What do you think is the average decrease in the serum potassium concentration during the peak effect of insulin therapy?
- 0.5 mmol/L
- 1.0 mmol/L
- 1.5 mmol/L
- 2.0 mmol/L
- 2.5 mmol/L

Do you anticipate the serum potassium concentration to return to its pre-shift value?
- Yes
- No
- Not sure

When do expect the serum potassium concentration to return to its pre-shift concentration following treatment with insulin?
- 2-3 hours
- 4-6 hours
- ≥ 7 hours

What do you regard as refractory hyperkalaemia?
- Serum K does not decrease after insulin therapy
- Two or more shifts required within 24-hrs
- Other

Do you have any safety concerns when using insulin to treat hyperkalaemia?
- Yes
- No

Do you routinely check the blood glucose concentration before administering insulin for the treatment of hyperkalaemia?
- Yes
- No

Before administering insulin for the treatment of hyperkalaemia, do you routinely administer glucose?
- Yes
- No

How much glucose do you usually administer when using insulin?
- 50 mL of 50% dextrose
- 100 mL of 50% dextrose
- 200 mL of 50% dextrose
- 300 mL of 50% dextrose

When do you administer the glucose in relation to insulin therapy?
- Before the insulin
- Mixed with insulin
- After insulin

How do you administer the glucose?
- Oral
- Intravenous

When do you expect hypoglycaemia after the administration of insulin and glucose for the treatment of hyperkalaemia?
- 0-1 hour after
- 1-2 hours after
- 2-3 hours after
- Hypoglycaemia is uncommon if glucose is given

When do you check the blood glucose concentration relative to insulin administration? (Select all that apply)
- Before
- 5 minutes after
- 30 minutes after
- 60 minutes after
- 2 hours after
- 3 hours after
- I do not check the glucose concentration

Do you routinely use drugs to increase the elimination of potassium during your management of hyperkalaemia?
- Yes
- No

Which drug(s) do you use to increase the elimination of potassium? (Select all that apply)
- K-binding resins (Kexelate)
- Loop diuretics
- Cathartics
- Intravenous sodium bicarbonate

Are you aware of any serious adverse effect(s) of Kexelate?
- Yes
- No

Table S3. Non-pharmacological management of hyperkalaemia.

Do you routinely stop foods with high potassium content?
Yes
No
Do you routinely stop drugs that may interfere with the renal elimination of potassium?
Yes
No