THE ETIOLOGY AND INCIDENCE OF HYPOKALEMIA IN INTENSIVE CARE UNIT

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ABSTRACT

Objective: Hypokalemia, a frequent electrolyte imbalance encountered in the Intensive Care Unit (ICU), is an important cause of morbidity and mortality in critically ill patients. In this study the etiology and incidence of hypokalemia in critically ill patients was investigated retrospectively.

Methods: The ICU records of 440 patients, followed up more than 2 days in surgical intensive care and reanimation unit between 01.01.1999 and 31.12.2000 were analysed retrospectively. When hypokalemia was first observed (K<3.5 meq/L) in patients, the coexisting pathologies such as hypothermia (axillary body temperature <36°C), dialysis, presence of diarrhea, vomiting and ketoacidosis, arterial pH values, type of nutrition, use of insulin, diuretics and beta adrenergic agents, presence of blood transfusions were recorded. The relationship between these data, age, type of surgery and hypokalemia was recorded. The data obtained were compared statistically with ANOVA, Fisher's exact and Chi-square tests ($p<0.05$).

Results: In 192 of 440 patients (40%) hypokalemia was found for the first time on 2.3±1.3th day of their ICU stay. The incidence of hypokalemia concerning the type of surgery was found as 59% in radical cystectomies, 51% in vascular craniotomies, 50% in radical prostatectomies and 47% in tumoral craniotomies. The relationship between hypokalemia and clinical findings, and the medications used are found significant ($p<0.05$). In the presence of metabolic alkalosis, the incidence of hypokalemia was 76%. Hypokalemia incidence in patients receiving enteral or parenteral nutrition was twice that of patients receiving oral nutrition.

Conclusion: Causes and incidence of hypokalemia must be established and follow up of plasma potassium levels should be done frequently in critically ill patients in the high risk group. This will decrease the mortality and morbidity with early replacement therapy.

Key words: Electrolyte imbalance, Hypokalemia, Intensive Care Unit.

INTRODUCTION

Disturbances in potassium homeostasis presenting especially as low serum potassium level, are common among critically ill patients (1). If hypokalemia, defined as serum potassium level less than 3.5 meq/L is left untreated, it will probably increase morbidity or mortality.

Marmara Medical Journal 2003;16(1):32-35

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Unfortunately, hypokalemia is often asymptomatic, especially for patients with mild hypokalemia (serum potassium between 2.5-3.5 meq/L), but severe hypokalemia (serum potassium less than 2.5 meq/L) is associated with profound signs (2). The management of hypokalemia is important in critically ill patients and requires close attention and orientation toward its etiology. On the other hand, the knowledge of its incidence among critically ill patients may help physicians to manage hypokalemia promptly and earlier in specific illnesses.

Another point of view is that health care has become increasingly expensive and attempts to decrease the costs of critical care medicine gain importance (3). So, unnecessary and frequent blood sampling for the determination of hypokalemia may be reduced just by knowing its etiology. To know the relationship of hypokalemia with illnesses, type of surgery or medications administered may lead us to create protocols according to the incidence of hypokalemia.

Accordingly, in this study the etiology and incidence of hypokalemia in patients at surgical intensive care and reanimation unit (ICU) was investigated.

METHODS

During the period from 1 January 1999 through 31 December 2000, the ICU records of 440 patients were analysed retrospectively. The patients older than 18 years and followed up for more than 2 days in ICU were included in the study.

Hypokalemia was defined as serum potassium level below 3.5 meq/L. When it was first observed in patients, the coexisting pathologies such as; hypotermia (axillary body temperature below 36°C), dialysis treatment, presence of diarrhea, vomiting, ketoacidosis, arterial pH values, type of nutrition, use of insulin, diuretics and beta-adrenergic agents, blood transfusions administered were recorded. The amount of diarrhea and vomiting, the degree of hypothermia, ketoacidosis and the doses of agents investigated were not taken into consideration, they were just recorded as binary data (yes or no). The relation of hypokalemia with these data, age of the patients and type of surgery was recorded.

The data obtained were compared statistically with ANOVA, Fisher's Exact and Chi-square tests. $p<0.05$ was considered significant.

RESULTS

The study population consisted of 440 patients and hypokalemia was found in 192 of these patients (40%).

Hypokalemia was observed for the first time on 2.3±1.3th day of patients' ICU stay. The surgical operation at which hypokalemia was the most frequently seen was radical cystectomy (59%). Cratiotomies done for vascular pathologies (intracranial aneurysms, hematomas, arteriovenous malformations) were in the second order (51%). Radical prostatectomy followed these with an incidence of 50%. The incidence of hypokalemia following craniotomies done for resection of mass lesions was 47% (Table I).

Table I: Frequency of hypokalemia according to the type of surgery

Type of surgery	%
Radical Cystectomy	59
Craniotomy (vascular)	51
Radical Prostatectomy	50
Craniotomy (tumoral)	47
Hip Prothesis	29
Thoracotomy	19
Vertebra Surgery	18

No correlation was found between age and hypokalemia (Table II).

Hypotermio (axillary body temperature lower than 36°C) ($p<0.01$) as well as polyuria ($p<0.001$), vomiting ($p<0.01$) and diarrhea ($p<0.01$) were found to be related with increased incidence of hypokalemia ($p<0.01$) (Table II).

The relationship between hypokalemia and the application of dialysis, administration of insulin ($p<0.01$), diuretics ($p<0.001$) and beta-adrenergic agents ($p<0.01$) was found significantly correlated with higher incidence of hypokalemia ($p<0.01$) (Table II). Blood

transfusions also were found to be correlated with hypokalemia ($p<0.01$) (Table II).

Table II: Relationship between hypokalemia and age, clinical findings, medications

Age	$p>0.05$
Hypothermia	$p<0.01$
Polyuria	$p<0.001$
Vomiting	$p<0.01$
Diarrhea	$p<0.01$
Insulin treatment	$p<0.01$
Diuretic treatment	$p<0.001$
Beta-adrenergic agent treatment	$p<0.01$
Blood transfusion	$p<0.01$

In the presence of metabolic alkalosis, the incidence of hypokalemia was 76%. A significant statistical correlation was found between type of nutrition and hypokalemia ($p<0.001$). The incidence of hypokalemia in patients receiving enteral or parenteral nutrition was twice that of patients receiving oral nutrition.

DISCUSSION

In this study, the incidence and causes of hypokalemia in critically ill patients were evaluated.

Hypokalemia is defined as a serum potassium of less than 3.5 meq/L (2). It results from increased loss, transcellular shift or decreased intake of potassium (4).

Increased potassium loss which is the most common cause of hypokalemia occurs mostly in patients who are on diuretics or in patients with gastrointestinal diseases such as diarrhea (4). In our critically ill patients, diuretic treatment and diarrhea were also highly related with hypokalemia. Thiazide and loop diuretics, by increasing the delivery of sodium to the collecting ducts, create a stimulus for potassium secretion. Furthermore, when a volume depletion occurs, through the activation of renin-angiotensin-aldosterone pathway, aldosterone secretion increases. This can cause metabolic alkalosis by increasing hydrogen mediated bicarbonate reabsorbtion in the collecting duct worsening diuretic induced hypokalemia (4). This explains the high incidence (76%) of hypokalemia in the presence of metabolic alkalosis, in our ICU. On the other hand, vomiting resulting in metabolic alkalosis may contribute to hypokalemia. Hypokalemia due to potassium shift into cells is caused by medication, hypothermia, hormonal disregulation, or metabolic alkalosis (5-8). Among the medication, we investigated in this study, beta-adrenergic drugs and insulin lead to hypokalemia. We did not take into consideration the relationship between the dosages of these medications and hypokalemia. Not surprisingly, we also found a correlation between hypothermia and hypokalemia.

Decreased potassium intake (i.e. less than 1 g/day) is rare but leads to hypokalemia because the kidneys lose 10-15 meq potassium per day (4). We also found a significant relationship between type of nutrition and hypokalemia. The incidence of hypokalemia in patients receiving enteral or parenteral nutrition was twice that of patients receiving oral nutrition. In fact, enteral and parenteral solutions contain sufficient amounts of potassium to meet the nutritional needs of the patients. So the association of enteral nutrition to hypokalemia might be a consequence of decreased transit time in the gut due to a high osmolar load. Hypokalemia associated with parenteral alimentation was probably related to the stimulation of insulin secretion.

Magnesium depletion is a common electrolyte disorder in hospitalised patients causing hypokalemia by increasing potassium loss (9). Neverthless, we did not investigate this electrolyte disorder in our study, since we did not routinely follow up magnesium levels in all patients. This would not give an appropriate correlation between hypokalemia and magnesium level because we lack the serum magnesium level of many patients.

No correlation has been found between hypokalemia and increasing age. This is interesting, because elderly people have usually comorbid diseases and they are under insulin, B adrenergic or diuretic therapy (10). Furthermore, they have poor oral intake and they are more prone to hypothermia (10). These factors might predispose elderly people to develop hypokalemia but we could not find any correlation in our study.

Among the types of surgery investigated, the incidence of hypokalemia in radical cyctectomy was found to be 59%. This is not surprising since we usually overhydrate patients together with the administration of diuretics in order to supply high urine output through newly oriented ureters.

Vascular craniotomies were also highly correlated with hypokalemia (51%). This may not be due to polyuria caused by cerebral salt wasting syndrome encountered in patients with intracranial aneurysms or arteriovenous malformations, because hypokalemia is typically absent in this syndrome (11). Diabetes insipitus on the other hand, may increase urinary potassium excretion (12) and cause hypokalemia after vascular craniotomies. Furthermore, the reason for high hypokalemia incidence in patients undergoing craniotomies, may be explained by the pharmaceutic administration of hydrocortisone, since these patients are always under steroid therapy. Hydrocortisone, prednisolone or methylprednisolone administration often lead to decreased potassium levels (2). Also, the frequent administration of mannitol, which will produce a significant diuresis and kaliuresis can lead to hypokalemia. Interestingly, in the study of Fukui et al (13) the female gender has been found as a risk factor for hypokalemia after subarachnoid hemorrhages. In fact, we did not investigate the gender differences concerning hypokalemia. It is obvious that further studies are needed for the possible involvement of female gender in a high hypokalemia risk group.

Radical prostatectomies had again a high incidence of developing hypokalemia. There is always blood loss in these patients and the number of blood transfusions are high. So hypokalemia may be the result of these transfusions.

In conclusion, the causes and incidence of hypokalemia must be established and the follow up serum potassium levels should be done frequently in critically ill patients in a high hypokalemia risk group in the first four days of ICU stay. This will decrease the morbidity and mortality with early replacement therapy. Hypothermia, polyuria, vomiting, diarrhea, metabolic alkalosis, dialysis treatment, the use of insulin, diuretics or beta-adrenergic agents, blood transfusions, enteral or parenteral nutrition are considered as risk factors. Again, patients who have had craniotomies, radical cystectomies and radical prostatectomies are under the risk of developing hypokalemia.

On the other hand, decreasing the frequency of blood sampling for serum potassium level evaluation in a low risk group will help to decrease the costs of critical care medicine.

REFERENCES

1. *Frederic SB, Darryl YS. Critical care diagnosis and treatment. First Edition. Philadelphia, USA: Appleton&Lange, 1994: 313.*
2. *Marino PL. The ICU Book. Second Edition. Pennsylvania USA: Williams and Wilkins, 1998: 650.*
3. *Shorr AF. An update on cost-effectiveness analysis in critical care. Curr Opin Crit Care 2002; 8: 337-343.*
4. *Rastergar A, Soleimani M. Hypokalemia and hyperkalemia. Postrgrad Med J 2001; 77: 759-771.*
5. *Brown MJ, Brown DC, Murphy MB. Hypokalemia from beta-2 receptor stimulation by circulating epinephrine. N Engl Med 1983; 309: 1414-1419.*
6. *Androgue HJ, Madias NE. Changes in plasma potassium concentration during acute acid-base disturbances. Am J Med 1981;71:456-467.*
7. *Dubose TD Jr. Hyperkalemic hyperchloremic metabolic acidosis: pathophysiologic insight. Kidney Int 1997; 51: 591-602.*
8. *Gennari FJ. Hypokalemia. N Engl Med 1998; 339: 451-457.*
9. *Salem M, Munoz R, Chernow B. Hypomagnesemia in critical illness. Crit Care Clin 1991; 7: 225-252.*
10. *Wongsuravat N, David B, Morley J. Thermoregulatory failure in the elderly. J Am Geriatr Soc 1990; 38: 900-906.*
11. *Oh MS, Carroll HJ. Cerebral salt-wasting syndrome: We need better proof of its existence. Nephron 1999; 82: 110-119.*
12. *Fernandez RE, Martinez MM, Opava SS. Role of water balance in the enhanced potassium excretion and hypokalemia of rats with diabetes insipidus. J Physiol 1980; 305: 97-108.*
13. *Fukui S, Otani N, Katoh H, et al. Female gender as a risk factor for hypokalemia and QT prolongation after subarachnoid hemorrhage. Neurology 2002; 59: 134-136.*