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REVIEW ARTICLE

Potassium Profiling in Hemodialysis

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Abstract

Cardiac dysrhythmia and sudden death account for a large proportion of cardiac mortality in dialysis patients. Risk factors for sudden death that are specific to dialysis patients include fluid and electrolyte imbalances during hemodialysis, particularly those of potassium. The risk of arrhythmia may be related to changes in serum K+ concentration during dialysis, and thus close attention should be paid to the dialysate K+ concentration and the serum–dialysate concentration gradient. Potassium profiling is a technique where the dialysate K+ concentration is gradually reduced to keep the gradient between blood and dialysate at a non-fluctuating low level. We provide a review of studies that compare constant potassium concentration in dialysate to gradual reduction in dialysate potassium concentration. These studies illustrate that adequate and more gradual potassium removal can be achieved with potassium profiling techniques, while having lower cardiac irritability.

Keywords: arrhythmia; dialysate potassium; potassium profiling; potassium removal; sudden cardiac death

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Introduction

Mortality and morbidity in End Stage Renal Disease (ESRD) patients on hemodialysis (HD) remains high, and higher than non-dialysis patients with similar co-morbidity burden (1). Cardiac dysrhythmia and sudden death account for a large proportion of cardiac mortality in these patients, amounting to 26.9% of total deaths (1). Risk factors for sudden death that are specific to dialysis patients include fluid and electrolyte imbalances during HD, particularly those of potassium(K+) (2). Most of the evidence linking the risk of arrhythmia to dialysis is derived from Electrocardiogram (EKG) markers such as ventricular repolarization indices which include QT duration (QTc), QT dispersion (QTd), PCA-T (principal component analysis of T wave), and E1-T (first eigenvalue of T wave) (3–11). These indices are known to reflect increased risk of arrhythmia (11, 12) and one of the factors which has been shown to change these indices is the change in serum potassium, stemming from the critical role of the K+ ion in myocardial repolarization (6, 13–15). Both hypokalemia and hyperkalemia have been shown to have associations with higher mortality in HD patients (16).

Due to lack of renal function to handle potassium excretion, ESRD patients undergo potassium removal during dialysis. In HD, this is achieved by diffusion of potassium from a higher concentration in serum to lower concentration

in dialysate, and is thus directly proportional to the concentration gradient between blood and dialysate. Hyperkalemia is associated with higher mortality in dialysis patients (16) and high potassium concentration in dialysate might impair potassium removal. But at the same time, multiple studies have associated low dialysate potassium with higher risk of sudden cardiac death. It was noted in a study with the dialysate K+ concentration of 0 and 1 meq/L (17), and with dialysate K+ of less than 3 (18). Further, another study showed less arrhythmia when the K+ bath was 3.5 versus 2 (19). This may be related to rapid changes in serum potassium during dialysis, and the challenge is to balance adequate potassium removal with risk of arrhythmia while doing it.

Potassium profiling is a technique where the dialysate K+ concentration is gradually reduced to limit the gradient between blood and dialysate constant and low. It has the potential advantage of reducing rapid changes in serum potassium level during dialysis (and thus reducing cardiac irritability), while also allowing for adequate potassium removal.

Here we provide a review of the randomized control trials that have compared potassium profiling of dialysate to a fixed dialysate potassium concentration.

Methods

We searched the PubMed database using search terms "potassium profiling," "potassium profiling in hemodialysis," "potassium hemodialysis," and "dialysate potassium." We included only the randomized control trials done in the past 25 years. The first study in our literature review to employ this technique was published in 1990 by Ebel et al. (20). Using a computerized program, they removed K+ slowly at a rate of 15%/hour and showed that incidence of arrhythmia was reduced from 60 to 25% (20). Since then, multiple randomized control trials have looked at potassium profiling. Although they have used different profiling techniques, all these studies have looked at effects of potassium profiling on cardiac stability using electrocardiographic markers and also ability to effectively remove potassium during dialysis.

Results

A brief overview of these studies is provided in Table 1 (21–26). All studies included in our review used a cross over design to compare constant potassium concentration in dialysate to gradual reduction in dialysate potassium concentration. The number of subjects was small, ranging from 10 to 36 patients. Inclusion and exclusion criteria were variable; some studies excluded patients with significant cardiovascular disease (3, 4), while others included only those who were considered high risk for arrhythmias (1, 6, 7). Although none of these studies looked exclusively at hyperkalemic patients, the average serum K+ concentration in these studies was normal or slightly high (up to 6 meq/L) and two studies excluded patients with hypokalemia (2, 6).

The dialysate potassium in control patients was mostly 2 meq/L. The potassium profiling techniques used in study population varied in different studies and are listed in Table 1.

A limitation of these studies is that arrhythmic risk using indirect EKG and Holter markers was assessed rather than actual arrhythmia events. This relates to the rather small number of study subjects and therefore the low overall incidence of arrhythmia. In the absence of a unanimous EKG marker for predicting arrhythmia with changes in K+, indirect measures of cardiac excitability and ventricular repolarization including Premature Ventricular Contractions (PVCs), changes in QTc/QTd/PCA-T/E1-T were used as surrogate markers of arrhythmic risk. Lower cardiac irritability with potassium profiling was shown in all studies. The effects were generally more pronounced in patients who are at higher risk of arrhythmia or who are dialysis sensitive. Comparison of potassium removal with standard versus potassium profiled dialysate techniques was done by measuring post-HD or pre-HD (prior to next session) serum potassium concentrations. There was no significant difference in these levels between the two techniques. Further, the training and technical requirements to implement this technique in a HD unit did not increase the work load of the nephrologist or the nurses, nor did it increase the technical complexity (27).

Conclusion

There are no standard practices for dialysate potassium concentrations and no recommendation has been provided in the NKF-KDOQI (National Kidney Foundation–Kidney Disease Outcomes Quality Initiative) cardiovascular disease guideline. In a large international cohort of HD patients, dialysate potassium concentration varied among clinical practices and countries and ranged anywhere from 1 meq/L to 3 meq/L (28). There are no large studies showing a difference in clinically significant arrhythmic events when gradually removing potassium in dialysis patients via potassium profiling of dialysate. However, there are multiple small studies suggesting a lower risk of arrhythmia with potassium profiling by using indirect markers of cardiac excitability and ventricular repolarization. Also, these studies suggest that an adequate amount of potassium could be removed without having a high gradient, at no extra financial cost. In our opinion, it would thus make sense to consider potassium profiling of dialysate in HD patients to reduce cardiac irritability, especially in patients who are at higher risk.

Table 1. Randomized controlled trials on potassium profiling.

Study no.	No. of patients/type of study	Control dialysate bath[a] K conc in mEq/L	Study dialysate bath[b] K conc in mEq/L	Measurement of outcomes	Difference in potassium removal	Result
Redaelli (21)	36, RCT, Crossover	<3 mean 2.3	Serum K-1.5,[b] Reduce to K-2.5	PVC	No change in pre-HD serum K level	Reduced PVC significantly
Santoro (22)	10, RCT, Crossover	2	Serum K-1, Reduce to K-1.5	PVC, QTd, PCA-T, E1-T	No change in post-HD serum K levels	Lesser PVC in pro-arrhythmic patients, Lesser QT-prolongation
Severi (23)	10, RCT, Crossover	2	Serum K-0.5, Reduce to K-1.5	QTd, QRS duration, St depression, PCA-T, E1-T	No change in post-HD serum K levels	Lesser variation in PCA-T, E1-T
Santoro (24)	12, RCT, Crossover	2	Serum K-3.2-4, Reduce to K-1-1.3	QTc, QTd, PVCs	No change in post-HD K levels	Lesser change in QTc, reduce high grade PVCs
Santoro (25)	15, RCT, Crossover	2	Serum K-1, Reduce to K-1.5	Ectopic beats	No change in post-HD K levels	Less ectopic beats significantly in pro-arrhythmic patients
Buemi (26)	24, RCT, Crossover	2	Serum K-1, Reduce to K-2	RBC REMP,[c] QTc, QTd	No change in post-HD serum K levels	Lesser change in QTc, QTd, REMP
Muñoz (27)	30, RCT, Crossover	2.5	Serum K-0.5, Reduce to K-1.5	PVC	No change in post-HD K levels	Lesser PVCs, Lesser QT-prolongation

[a]Control bath = dialysate potassium fixed during the entire dialysis session (not profiled).
[b]Study bath = potassium profiled bath. In this case, dialysate K concentration Kd = serum K – 1.5 meq/L.
[c]Red blood cell electrical membrane potential at rest (RBC REMP).
Premature ventricular contraction (PVC), QT duration (QTc), QT dispersion (QTd), principal component analysis of T wave (PCA-T), first eigenvalue of T wave (E1-T).

Conflict of Interest

The authors declare no potential conflicts of interest with respect to research, authorship, and/or publication of this article.

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