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In Practice

Dialysate and Serum Potassium in Hemodialysis

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Most patients with end-stage renal disease depend on intermittent hemodialysis to maintain levels of serum potassium and other electrolytes within a normal range. However, one of the challenges has been the safety of using a low-potassium dialysate to achieve that goal, given the concern about the effects that rapid and/or large changes in serum potassium concentrations may have on cardiac electrophysiology and arrhythmia. Additionally, in this patient population, there is a high prevalence of structural cardiac changes and ischemic heart disease, making them even more susceptible to acute arrhythmogenic triggers. This concern is highlighted by the knowledge that about two-thirds of all cardiac deaths in dialysis are due to sudden cardiac death and that sudden cardiac death accounts for 25% of the overall death for end-stage renal disease. Developing new approaches and practice standards for potassium removal during dialysis, as well as understanding other modifiable triggers of sudden cardiac death, such as other electrolyte components of the dialysate (magnesium and calcium), rapid ultrafiltration rates, and safety of a number of medications (ie, drugs that prolong the QT interval or use of digoxin), are critical in order to decrease the unacceptably high cardiac mortality experienced by hemodialysis-dependent patients.

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Note from Editors: This article is part 1 of a 4-part series of invited In Practice reviews highlighting issues related to the composition of dialysate.

CASE PRESENTATION

A 68-year-old African American man with end-stage renal disease secondary to long-standing hypertension had been on dialysis therapy for almost 2 years. His dialysis treatment prescription was dialyzer F-200 (Fresenius Medical Care); treatment length, 3.5 hours; dialysate potassium concentration, 2 mEq/L (K2); calcium concentration, 2.5 mEq/L; and bicarbonate concentration, 39 mEq/L (the bicarbonate bath was from a concentrate mix that also had 8 mEq/L of acetate, resulting in a total buffer of 47 mEq/L). His interdialytic weight gain was an average of 4 to 4.2 kg, necessitating an ultrafiltration rate of ∼15 mL/kg/h to achieve his dry weight of 77 kg. His physical examination findings were unremarkable. His medical history included left ventricular hypertrophy and diastolic heart failure, which had been controlled since the initiation of dialysis therapy. More recently, he had significant dental pain, had been eating less over the last 2 to 3 weeks, and was scheduled for an extensive dental workup. His most recent monthly laboratory tests showed Kt/V of 1.5, hemoglobin level of 10.7 g/dL, and predialysis serum potassium level of 4.2 mEq/L, bicarbonate level of 27 mEq/L, and calcium level of 8.9 mEq/L. He was undergoing his usual dialysis treatment on a Monday when he experienced sudden cardiac arrest during the second half of the dialysis treatment. Unfortunately, the patient did not respond to cardiac resuscitation maneuvers and died. Patient medications included cinacalcet, 30 mg, at bedtime; calcium acetate, 3 capsules with meals; nifedipine, 90 mg, at bedtime; and omeprazole, 20 mg daily. He also recently had started azithromycin prescribed by his dentist.

INTRODUCTION

Sudden cardiac death due to arrhythmic mechanisms continues to be the leading cause of attributable mortality in both the incident and prevalent dialysis populations. In the prevalent hemodialysis (HD) population, sudden cardiac death accounts for ∼67% of all cardiac deaths and 26.9% of overall deaths, as reported by the Centers for Medicare & Medicaid Services death form 2746.[1] These estimates are consistent with those reported by trials and observational studies.[1,2] Although rates of sudden cardiac death in prevalent dialysis patients have been improving over time, this reduction in sudden cardiac death–related mortality has been particularly evident since 2001.[1] Rates of sudden cardiac death in the HD population decreased from 72.1 per 1,000 person-years in 2001 to 49.2 per 1,000 person-years in 2011; in part, these changes could depend on the use of a more accurate definition, as well as in better coding in the data sources. Nevertheless, the rate of these tragic sudden cardiac death events is more than 25- to 49-fold higher in dialysis patients compared to the general population (1-2 sudden cardiac deaths/1,000 person-years).[3]

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Sudden death is defined as an unexpected nontraumatic death that occurs within 1 hour of the onset of symptoms. Sudden cardiac death usually occurs due to an acute stressor (trigger), generally in the context of a damaged myocardium. Both these circumstances are required for sudden cardiac death to occur. Dialysis patients have a high prevalence of structural heart disease, including a high prevalence of coronary artery disease and coronary artery calcification.[4] However, 2 case series of dialysis patients who experienced sudden cardiac death reported that ~70% of these patients had a normal ejection fraction.[5] Additionally, left ventricular hypertrophy, which is present in close to 75% of HD patients, is associated with a high component of fibrosis and dilated cardiomyopathy that also may play a role in the rate of sudden cardiac death.[6]

CARDIAC ELECTROPHYSIOLOGIC ABNORMALITIES IN HD PATIENTS

Another important contributing factor that places HD patients at increased risk of sudden cardiac death is QT dispersion, a measurement of repolarization of the ventricle.[7] QT dispersion is increased in the dialysis population, with more than half the patients having a prolonged corrected QT (QTc) interval.[8] Beaubien et al[8] confirmed the prognostic value of QTc dispersion and the significantly higher total and cardiovascular mortality risk associated with it in a cohort of 147 patients initiating dialysis therapy. Prolonged QT interval reflects unstable repolarization and greater vulnerability to left ventricular arrhythmias and re-entry arrhythmias (ie, torsade de pointes and ventricular fibrillation) and sudden cardiac death. Accordingly, a 50-ms difference in QTc was associated with a 1.53-time increase in cardiovascular and all-cause mortality.[8]

Transmembrane electrolyte shifts that occur during each HD session in conjunction with the structural changes (left ventricular hypertrophy and congestive heart failure) and subclinical ischemia that are highly prevalent in HD patients probably are the most important factors in the association between dialysis and prolongation of the QTc interval, although there are other contributing factors.[9] Different ionic countercurrents such as potassium and calcium control the different phases of ventricular depolarization and repolarization of the action potential.[10]

POTENTIAL MODIFIABLE FACTORS INVOLVED IN SUDDEN CARDIAC DEATH

In addition to the structural cardiac changes present in advanced chronic kidney disease (CKD), there are important and unique triggers of sudden cardiac death in the HD population. Some of the most important factors include rapid electrolyte shift (particularly potassium and calcium), fluid shift (ultrafiltration rates), and the use of certain medications that have important interactions with these electrolyte shifts. Understanding these HD-related triggers is critical because they potentially are modifiable and may be targeted for interventions to decrease the alarmingly high rates of sudden cardiac death in the HD population.

There are several observational studies that highlight that the HD procedure itself may be arrhythmogenic and increase the risk of sudden cardiac death.[7,11,12] A recent nationally representative study that included 32,065 patients receiving HD between 2004 and 2007 found that sudden cardiac death had a higher incidence on the first dialysis treatment of the week after the long interdialytic interval.[11] A limitation of these studies is that the time of death is not specified with respect to the dialysis procedure time. However, a prospective study showed there is a 3-fold increase in risk of sudden cardiac death in the interval of the 12 hours prior to dialysis after the long weekend interval and a 1.7-fold increase in the 12 hours including and following this dialysis procedure.[5] These observations highlight some of the risk factors predisposing to sudden cardiac death, such as large weight gain and hyperkalemia, both of which are more likely to be present after a long interdialytic interval and mandate rapid ultrafiltration rates during the dialysis, as well as a rapid reduction in serum potassium levels during the dialysis procedure. We discuss these factors separately next. Additionally, it is important to highlight that the associated rhythm disturbances differ in these circumstances, being more commonly asystole for hyperkalemia observed predialysis and ventricular fibrillation for hypokalemia observed following dialysis; this difference may have impact on treatment approaches.

IMPACT OF LOW POTASSIUM DIALYSATE AND PREDIALYSIS SERUM POTASSIUM ON SUDDEN CARDIAC DEATH IN HD PATIENTS

The role of predialysis serum potassium level as a contributing factor to sudden cardiac death has been documented in several studies.[13,14] A case-control by Pun et al[14] that matched 502 patients who experienced a witnessed cardiac arrest during dialysis treatment with 1,632 controls concluded that the increased risk was linked to both low and high predialysis serum potassium values. The association was of a U-shaped curve, with the lowest risk at a predialysis serum potassium value of 5 mEq/L and higher risk with both higher and lower serum potassium levels. This finding is consistent with the study by Kovesdy et al[13] using data from a large dialysis provider in which the values for predialysis potassium with the lower risk of death were 4.6 to 5.3 mEq/L and also followed a U-shaped association. However, the Kovesdy et al[13] study looked at all-cause

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