Renal Replacement Therapy

CASE REPORT

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Lethal ventricular arrhythmia can be prevented by adjusting the dialysate potassium concentration and the use of anti-arrhythmic agents: a case report and literature review

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Abstract

Background: Hypokalemia is common in patients with malnutrition undergoing hemodialysis and is often involved in the development of lethal arrhythmia. Moreover, hemodialysis therapy decreases the serum potassium concentration due to potassium removal to the dialysate. However, it is difficult to adjust the dialysate potassium concentration owing to the use of the central dialysate delivery system in Japan. Here, we have presented a case undergoing hemodialysis with dialysate potassium concentration adjustment to prevent ventricular arrhythmia.

Case presentation: A 56-year-old man with Emery-Dreifuss muscular dystrophy and chronic heart failure was admitted to our hospital and needed subsequent hemodialysis therapy due to renal dysfunction. During hemodialysis, the cardiac resynchronization therapy defibrillator was activated to the treatment of his lethal ventricular arrhythmia. Decreases in serum potassium concentration after hemodialysis and changes in serum potassium concentration during HD were considered the causes of lethal ventricular arrythmia. Therefore, along with using anti-arrhythmic agents, the dialysate potassium concentration was increased from 2.0 to 3.5 mEq/L to minimize changes in the serum potassium concentration during hemodialysis. Post-dialysis hypokalemia disappeared and these changes during hemodialysis were minimized, and no lethal ventricular arrhythmia occurred thereafter.

Conclusions: In this case, we prevented lethal arrhythmia by maintaining the serum potassium concentration by increasing the dialysate potassium concentration, in addition to the use of anti-arrhythmic agents. In the acute phase of patients with frequent lethal arrhythmia undergoing hemodialysis, an increase in dialysate potassium concentration may be an effective method for preventing arrhythmogenic complications.

Keywords: Post-dialysis hypokalemia, Dialysate potassium concentration, Hemodialysis, Ventricular arrhythmia, Potassium gradient

Background

Hyperkalemia is observed in patients with chronic kidney disease (CKD) because of a gradual decrease in renal potassium excretion with CKD stages progression [1, 2]. Therefore, they require hemodialysis (HD) therapy to manage electrolyte disorders, especially potassium imbalance. The serum potassium concentration affects

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the excitation of the myocardial stimulatory conduction system, and changes in the concentrations may lead to severe complications such as lethal arrhythmia [3]. Hyperkalemia is a known cause of sudden death among patients undergoing HD [2, 4]. Conversely, the predialysis serum potassium concentration is often low in elderly adults with malnutrition undergoing HD. However, hypokalemia is also associated with an increased risk of mortality [5] and is a trigger for lethal arrhythmia [6]. Furthermore, a low potassium dialysate concentration and hypokalemia may increase the risk of sudden cardiac death [7]. Therefore, in patients undergoing HD, both hyperkalemia and hypokalemia may be associated with an increased risk of mortality [5, 8, 9]. In Japan, a dialysate potassium concentration of 2.0 mEq/L is mainly used; however, it is difficult to change this concentration because limited dialysates with different potassium concentrations are commercially available, and they are supplied through a central dialysate delivery system (CDDS) [10]. Here, we have reported a case wherein lethal arrhythmia was prevented by maintaining the serum potassium concentration by increasing the dialysate potassium concentration and administering anti-arrhythmic agents.

Case presentation

A 56-year-old man with Emery-Dreifuss muscular dystrophy and chronic heart failure was admitted to our hospital due to overhydration and diuretic resistance. His medical history included cardiac dysfunction with a low ejection fraction of 33%, ventricular tachycardia (VT), and advanced atrioventricular block complicated by muscular dystrophy. Therefore, a cardiac resynchronization therapy defibrillator (CRT-D) was implanted at the age of 50 years, and treatment with 1.25 mg of enalapril, 80 mg of furosemide, 1 mg of warfarin, 10 mg of rabeprazole, and 25 μg of levothyroxine was initiated. His height was 157 cm and body weight (BW) was 98 kg on admission; however, his BW was 61 kg a year and a half ago. The patient's vital signs on admission were as follows: blood pressure, 87/68 mmHg; pulse rate, 67 beats/min; and oxygen saturation of the peripheral artery (room air), 92%. Laboratory findings demonstrated the following: white blood cell count, 5460/μL; hemoglobin level, 10.5 g/dL; platelet count, 11.6×10^3/μL; total protein level, 6.5 g/dL; serum albumin level, 3.5 g/dL; blood urea nitrogen level, 36 mg/dL; serum creatinine level, 1.44 mg/dL; serum sodium level, 136 mEq/L; serum potassium level, 5.0 mEq/L; serum calcium level, 8.9 mg/dL; serum phosphate level, 3.8 mg/dL; brain natriuretic peptide level, 183 pg/mL; KL-6 level, 239 U/mL; thyroid stimulating hormone level, 31.1 μIU/mL; free T4 level, 1.01 ng/mL; and free T3 level, 1.47 pg/mL. On physical examination, his heart sounds were regular due to pacing rhythm without murmur; respiratory sounds were coarse crackles; and face, trunk, and bilateral lower extremities were edematous. His chest radiograph, electrocardiogram, and echocardiogram on admission are shown in Fig. 1. Echocardiography showed a decrease in the ejection fraction (35%) with moderate mitral regurgitation. After admission, although the furosemide dosage was increased and tolvaptan was initiated to reduce excess body fluid, his urine volume did not increase. Thereafter, intra-aortic balloon pumping and continuous renal replacement therapy were initiated in the intensive care unit because of acute deterioration in cardiac function and persistent excess body fluid. The excess body fluid was removed, reducing the BW from 97 to 73 kg, and his hemodynamic status gradually improved. However, after cessation of intra-aortic balloon pumping, his urine volume significantly decreased; therefore, HD was needed to manage his body fluid status and mineral and electrolytes imbalance. After initiation of HD therapy, CRT-D was activated twice (42nd and 44th days of hospitalization) to defibrillate the pulseless VT during HD. Despite an infusion of potassium chloride solution and initiation of amiodarone, the patient frequently presented with episodes of non-sustained VT with a decrease in the serum potassium concentration. Therefore, the decrease in the serum potassium concentration after HD and changes in the serum potassium concentration during HD were considered to influence the occurrence of VT. The dialysate potassium concentration was changed from 2.0 to 3.5 mEq/L to minimize changes in the serum potassium concentration during HD. Thereafter, his serum potassium concentration did not fluctuate, and the changes in the serum potassium concentration before and after HD disappeared (Fig. 2). The dialysate potassium concentration (3.5 mEq/L) was adjusted until the 133rd day of hospitalization. An increase in his food intake led to a consequent increase in potassium intake; therefore, the dialysate potassium concentration was decreased to 3.0 mEq/L, along with taking carvedilol, and 1 week later, to 2.0 mEq/L. During this period, defibrillation by the CRT-D did not occur probably because of the improvement of potassium intake and the effect of the anti-arrhythmic agents (amiodarone and carvedilol). The patient was discharged on the 162nd day of hospitalization. He continued HD therapy in another maintenance dialysis unit without CRT-D operation.

Discussion and conclusions

Many maintenance dialysis facilities in Japan use the CDDS [10]. This system is useful for the supply of the dialysate with the same electrolyte concentrations for multiple HD machines. Our patient was treated with

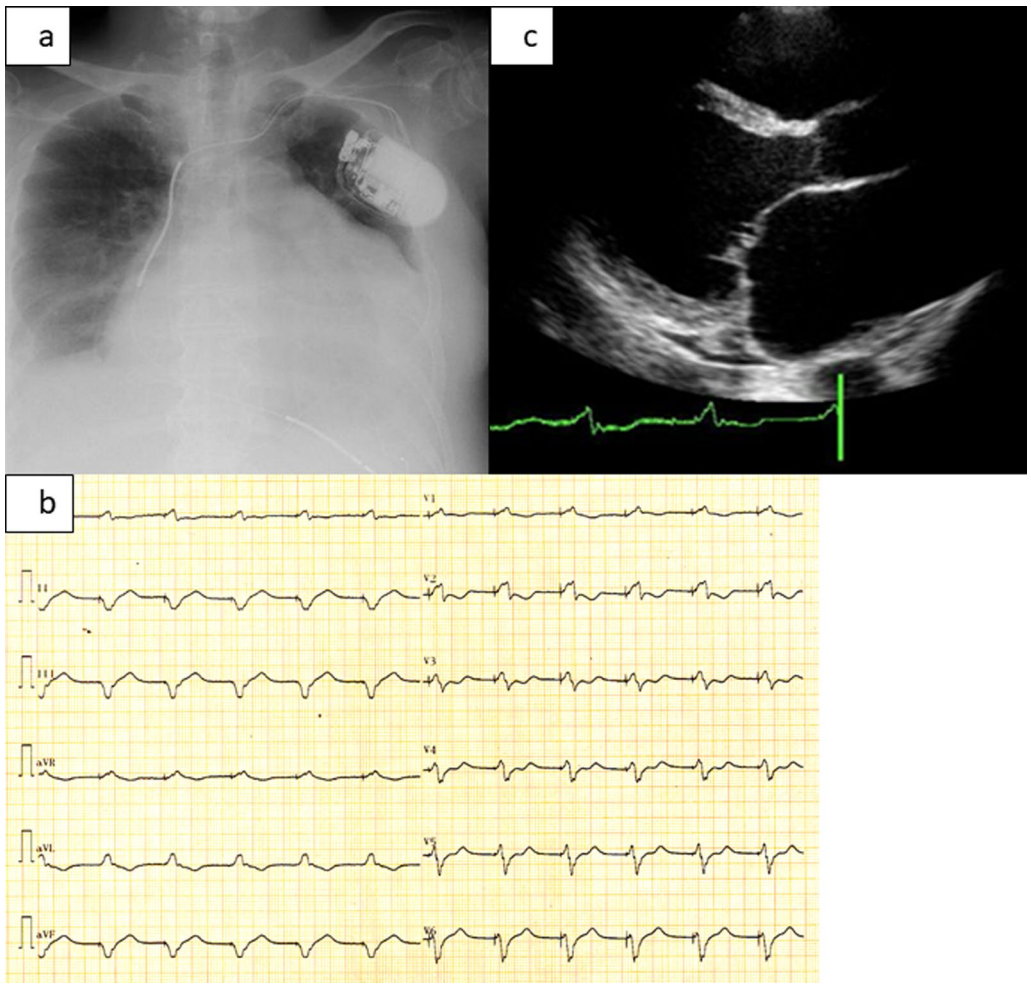

Fig. 1 **a** Chest radiograph on admission. **b** Electrocardiogram on admission. **c** Echocardiography on admission

a standard dialysate used in Japan composed of Na^+, 140 mEq/L; K^+, 2.0 mEq/L; Cl^-, 110 mEq/L; Ca^{2+}, 3.0 mEq/L; Mg^{2+}, 1.0 mEq/L; HCO_3^-, 30 mEq/L; and glucose, 100 mg/dL. A dialysate potassium concentration of 2.0 mEq/L is suitable for patients with hyperkalemia undergoing HD; however, a dialysate potassium concentration < 2.0 mEq/L is associated with an increased risk of sudden cardiac arrest due to hypokalemia [3]. In this case, decreases in serum potassium concentration during HD were suspected to be the cause of lethal arrhythmia. The increase in the dialysate potassium concentration to 3.5 mEq/L using a personal dialysate delivery system contributed to the disappearance of lethal arrhythmia. According to the Dialysis Outcomes and Practice Patterns Study, a dialysate potassium concentration of 2.0 mEq/L is used for > 99% patients in the clinical setting of HD therapy in Japan [2]. However, other countries use various dialysate potassium concentrations that can be as low as 1.0 mEq/L and higher than 3.0 mEq/L [2]. Furthermore, the dialysate is usually supplied using a personal dialysate delivery system in many countries except in Japan; therefore, dialysate potassium concentrations can easily be changed using a personal dialysate delivery system compared with those using the CDDS. According to a survey report by the Japanese Society for Dialysis Therapy on the dialysate prescription, 46% of the surveyed facilities reported modifications in their dialysate [11]. Since the CDDS cannot supply individualized dialysate composition [12], the composition can be modified using a personal dialysate delivery system.

The acid–base status influences the intracellular and extracellular potassium homeostasis. In particular, the bicarbonate can affect the potassium redistribution between the intracellular and extracellular fluid, and hypokalemia is induced by the increase in serum bicarbonate concentration, which leads to the increased cell

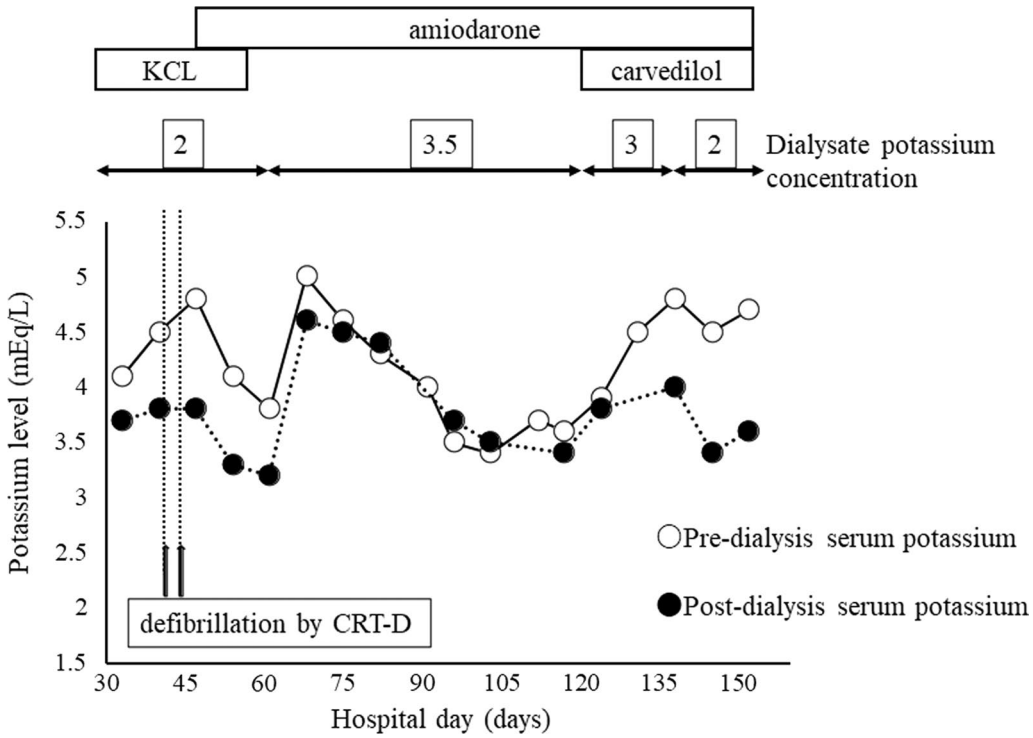

Fig. 2 Trends in dialysate potassium concentrations and pre- and post-dialysis serum potassium levels during hospitalization. Abbreviations: *KCL* potassium chloride, *CRT-D* cardiac resynchronization therapy defibrillator

sodium uptake, stimulation of Na^+, K^+-ATPase activity, and net cellular potassium uptake [13]. In this patient, the bicarbonate concentration before HD was similar during his clinical course (20.5 mEq/L, 21.4 mEq/L, and 20.0 mEq/L at 47th day, 82nd day, and 117th day, respectively). Therefore, there was no association between the serum potassium concentration and bicarbonate concentration before HD in this case. However, we cannot comment on the association between them after HD because the serum bicarbonate after HD was not examined. Calcium and magnesium contribute to the arrythmia development during HD since these cations play an important role in the development of the ventricular action potential and propagation of the electrical impulse [14]. Furthermore, low calcium dialysate (< 2.5 mEq/L), higher corrected serum calcium, and increasing serum dialysate calcium gradient have been associated with an increased risk of sudden cardiac death [15]. According to the laboratory findings obtained on the 40th day of hospitalization, which was closer to the day when CRT-D was activated to defibrillate the pulseless VT during HD, serum corrected calcium concentration before and after HD was 9.0 mg/dL and 9.3 mg/dL, respectively, and serum magnesium concentration before HD was 3.0 mg/dL, with the dialysate calcium concentration of 3.0 mEq/L. Thereafter, no hypocalcemia and hypomagnesemia were confirmed in his clinical course; therefore, in this case, serum calcium and magnesium concentration were considered to have no association with ventricular arrhythmias. In addition, high systolic blood pressure [16], intradialytic hypotension [17], and fluid overload [18] have been associated with the development of cardiac arrhythmias. In both the HD sessions with CRT-D activation in response to the pulseless VT, systolic blood pressure was maintained at around 100 mmHg and no intradialytic hypotension occurred during HD. However, when this HD session was initiated, the body fluid status was noted to be excessive because of the rapid decrease in urine volume; therefore, excessive body fluid might be associated with the occurrence of ventricular arrhythmia.

Furthermore, a decrease in the serum potassium concentration is likely to be associated with a decrease in potassium intake, as seen in patients with malnutrition undergoing HD, in addition to the loss of potassium from the blood to the dialysate. In particular, severe hypokalemia after HD is occasionally observed in hospitalized patients. Therefore, we investigated and compared the serum potassium concentrations between hospitalized patients and outpatients undergoing HD from January 2018 to December 2019. As shown in Fig. 3a, the serum

potassium concentration before HD was distributed to the lower side in hospitalized patients compared to that in outpatients, and the proportion of the patients with a serum potassium concentration < 3.5 mEq/L was significantly higher among hospitalized patients (385 of 2593 patients) than among outpatients (43 of 3286 patients; $p < 0.0001$). Furthermore, after HD, the frequency of serum potassium concentration < 3.5 mEq/L was 47% (Fig. 3b). The incidence of hypokalemia after HD in Japanese patients was previously reported to be 44.8% [9], which is similar to our results.

Table 1 presents a summary of the literature review regarding the relationship between the dialysate potassium concentration and arrhythmia for this particular patient population. Jadoul et al. [19] reported that patients undergoing HD with pre-dialysis serum potassium concentrations of < 5.0 mEq/L could be at risk of sudden death due to the effects of post-dialysis hypokalemia, and prescribing a dialysate potassium concentration of > 3.0 mEq/L might reduce this risk. Ohnishi et al. [20] also reported an increase in the mortality rate associated with a post-dialysis serum potassium concentration of < 3.0 mEq/L. In this case, VT occurred even when the post-HD serum potassium concentration exceeded 3.0 mEq/L because of the high susceptibility of VT to recur as a primary disease. Therefore, along with administering amiodarone, the dialysate potassium concentration was adjusted from 2.0 to 3.5 mEq/L to prevent further decrease in serum potassium concentration after HD and minimize changes in potassium concentration after HD. Thereafter, the lethal arrhythmia disappeared, and this would be associated with the adjustment of dialysate potassium concentration and amiodarone usage. A reduction in the serum-to-dialysate gradient of potassium concentration decreases arrhythmogenic complications or the number of emergency department visits [21, 22]. Moreover, the risk of death was reportedly higher

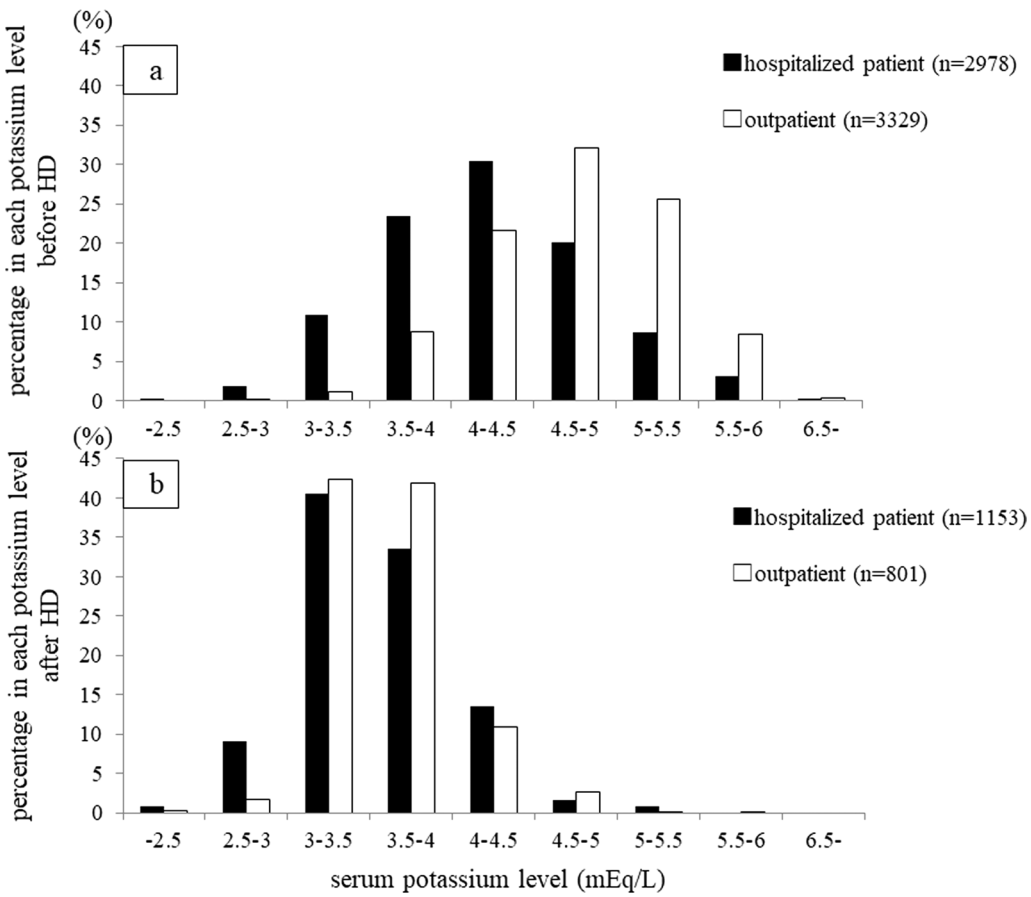

Fig. 3 **a** Distribution of pre-dialysis serum potassium levels in patients undergoing hemodialysis at our hospital. Black square represents hospitalized patients and white square represents outpatients. **b** Distribution of post-dialysis serum potassium levels in patients undergoing hemodialysis at our hospital. Black square represents hospitalized patients, and white square represents outpatients. Abbreviations: *HD* hemodialysis

Table 1 Literature review of the relationship between the dialysate potassium concentration and arrhythmia in patients undergoing HD

Authors	Publication year	Country	Dialysate potassium concentration	Patients	Results and findings	References
Morrison et al.	1980	The USA	2, 3.5	23	A significant reduction in the frequency of ventricular ectopy was confirmed with the use of 3.5 mEq/L potassium dialysates	[23]
Redaelli et al.	1996	Italy	2.1–4.1 (mean potassium levels)	36	A reduction in the plasma-dialysate potassium concentration gradient significantly led to the decrease in HD-induced arrhythmia in patients undergoing regular HD treatment	[21]
Karnik et al.	2001	North America	0, 1, 2, 2.5, 3, 4	> 77,000	The cardiac arrest rate was a 7 per 100,000 hemodialysis sessions (400 events of 5,744,708 HD session). Case patients were twice as likely to have been dialyzed against a 0 or 1.0 mEq/L potassium dialysate on the day of cardiac arrest	[24]
Kovesdy et al.	2007	The USA	< 1.0, 2.0, 3.0, > 3.0	81,013	In total, 8679 all-cause deaths were observed. The highest risk of death was observed among patients with a serum potassium concentration of > 5 mEq/L exposed to a dialysate potassium concentration of > 3; however, no increased risk of mortality was associated with a dialysate potassium concentration of < 2 mEq/L among patients with a serum potassium concentration of > 5 mEq/L	[7]
Al-Ghamdi et al.	2010	Canada	0 or 1, 2, 3 4	1267	In total, 515 all-cause deaths were observed. There were no independent associations between lower dialysate potassium concentrations and the risk of mortality compared to a dialysate potassium concentration of 2 mmol/L	[25]
Pun et al.	2011	The USA	1, 2, 3 4	2134	In total, 502 SCDs were observed, and the cardiac arrest rate 4.5 per 100,000 HD treatments. For pre-dialysis serum potassium levels (< 5 mEq/L), the difference in risk between those treated with a low (< 2 mEq/L) and high (> 2 mEq/L) potassium dialysate concentrations increases, but among subjects with higher pre-dialysis serum potassium levels (≥ 5 mEq/L), there was no significant difference in the risk based on the potassium dialysate level	[26]

Table 1 (continued)

Authors	Publication year	Country	Dialysate potassium concentration	Patients	Results and findings	References
Jadoul et al.	2012	12 countries in the DOPPS	≤ 1.5, 2–2.5, ≥ 3	37,765	In total, 1734 sudden deaths were observed, and dialysate potassium concentrations of ≤ 1.5 mEq/L and 2–2.5 mEq/L were associated with a higher risk of SCD compared to concentrations of ≥ 3.0 mEq/L	[19]
Flythe et al.	2014	North America	1, 2, > 2	76,462	In total, 924 cardiopulmonary arrests were observed, and the use of 1 mEq/L potassium dialysate was associated with an increased risk of cardiopulmonary arrests	[27]
Buiten et al.	2014	The Netherlands	1.6, 2 (mean potassium levels)	40	In patients with an implantable cardioverter defibrillator undergoing dialysis, 428 episodes of AF were observed. In patients with AF episodes, the potassium dialysate concentration was lower than that in patients without AF (1.6 mEq/L vs. 2 mEq/L)	[28]
Huang et al.	2015	Taiwan	1, 2, 2.5, 3	312	In total, 31 SCDs were observed. Using 1.0 mEq/L potassium dialysate in patients with hyperkalemia undergoing HD (> 5.5 mEq/L) and adequate adjustment of dialysate composition in patients with normo- or hypokalemia undergoing HD from 1.0 mEq/L potassium dialysate could reduce the rate of SCD	[29]
Karaboyas et al.	2017	20 countries in the DOPPS	1–1.5, 2–2.5, 3–4	55,183	In total, 13,114 deaths and 3300 composite events related arrythmias were observed. No meaningful difference in clinical outcomes was observed for patients treated with a dialysate potassium concentration of 3 and 2 mEq/L for both mortality and composite arrhythmia events	[2]
Ferrey et al.	2018	The USA	1, 2, 3	624	A dialysate potassium concentration of 1 mEq/L was associated with a higher risk of mortality in patients with higher serum potassium concentrations (≥ 5 mEq/L), but not in those with lower serum potassium concentrations (< 5 mEq/L)	[30]
Brunelli et al.	2018	The USA	1, 2, 3, 4	830,741	There was a dose–response relationship between higher K+ gradient [(serum K^+) – (dialysate K^+)] and greater risks of all-cause hospitalization and emergency department visit	[22]

Table 1 (continued)

Authors	Publication year	Country	Dialysate potassium concentration	Patients	Results and findings	References
Delanaye et al.	2021	Belgium	1, 3	27	An increase of 1 mEq/L in the dialysate potassium level was associated with a lower risk of hypokalemia and a lower QT dispersion on electrocardiogram after HD session	[31]

DOPPS dialysis outcomes and practice patterns study, *HD* hemodialysis, *SCD* sudden cardiac death, *AF* atrial fibrillation, K^+ potassium

Table 2 Adjustment of the dialysate potassium concentration in our dialysis unit

Targeted potassium concentration (mEq/L)	KCL volumes added to the dialysate (mL)
2.5	44
3.0	89
3.5	133
4.0	178

The concentration of added KCL solution was 2.36 mEq/mL, and the total amount of dialysate before dilution was 6 L

KCL potassium chloride

in patients with large differences in serum potassium concentrations before and after HD [20]. Therefore, also in this case, the adjustment of the dialysate potassium concentration might have contributed to the disappearance of lethal arrythmia. However, there have been no specific recommendations for the adjustment of dialysate potassium concentrations in Japan. At our dialysis center, adjustment of dialysate potassium concentration was performed, as shown in Table 2. An adjusted 2.36 mEq/mL sterilized potassium-chloride solution was prepared by our pharmaceutical department. The adjusted solution was added to the undiluted dialysate solution. To ensure the safety and reliability of the adjusted dialysate compositions, we routinely checked the dialysate potassium concentration supplied to the HD machine before HD.

In conclusion, we encountered a case in which lethal arrhythmia was prevented by maintaining serum potassium concentration by increasing the dialysate potassium concentration, in addition to the use of anti-arrhythmic agents. In the acute phase of patients with frequent lethal arrhythmia undergoing HD, an increase in the dialysate potassium concentration may be an effective method for preventing arrhythmogenic complications.

Abbreviations

HD: Hemodialysis; VT: Ventricular tachycardia; CRT-D: Cardiac resynchronization therapy-defibrillator; BW: Body weight; CDDS: Central dialysate delivery system.

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Author contributions

TU, SO, KI, HO, HH, MK, MK, YU, TH, NM and YM were responsible for taking care of the patient and decided the treatment regimen. TU, SO and KI drafted the manuscript and were responsible for the final version of the manuscript. All authors have read and approved the final manuscript.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Declarations

Ethics approval and consent to participate

This case report was written in compliance with the Declaration of Helsinki. Data collection was performed with the approval of the Ethics Committee of Jichi Saitama Medical Center (S19-006).

Consent for publication

Informed consent was obtained for the publication of this case report.

Competing interests

The authors declare that they have no competing interests.

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