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EXTRACORPOREAL MEMBRANE OXYGENATION SUPPORT FOR HYPOKALEMIA-INDUCED CARDIAC ARREST: A CASE REPORT AND REVIEW OF THE LITERATURE

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☐ **Abstract—Background: Hypokalemia is a reversible cause of cardiac arrest in patients presenting to the emergency department (ED). Extracorporeal membrane oxygenation (ECMO) is an established technology for cardiopulmonary support with emerging roles in resuscitation. Here, we review the literature of hypokalemic-induced cardiac arrests and discuss one such case successfully managed with ECMO. Case Report: A 23-year-old Central American man who presented to a community ED under federal custody with several days of nausea and vomiting was found to have a serum potassium level of 1.5 mEq/L. Repeat serum potassium level was 1.1 mEq/L upon arrival to our facility. Within 2 h of arrival, despite electrolyte repletion, he suffered cardiac arrest. Advanced cardiac life support was performed for 45 min. ECMO was initiated while active chest compressions were performed. After aggressive potassium repletion, return of spontaneous circulation was achieved and ECMO was eventually discontinued. Further investigation ultimately confirmed the presence of a potassium-wasting nephropathy, for which the patient had been treated with chronic potassium supplementation prior to entering federal custody. Why Should an Emergency Physician be Aware of This?: ECMO is a well-established modality for cardiopulmonary support, with an emerging role for patients in undifferentiated cardiac arrest presenting to the ED. There is a growing interest in the utility of ECMO in these circumstances. This report highlights hypokalemia as an important cause of cardiac arrest, reviews the treatment and causes of hypokalemia, and demonstrates a potential role for ECMO as a critical temporizing measure to provide time for potassium repletion.** © 2015 Elsevier Inc.

☐ **Keywords—hypokalemia; cardiac arrest; extracorporeal membrane oxygenation**

INTRODUCTION

Emergency physicians must simultaneously assess and treat patients presenting in cardiac arrest. Prioritizing the fundamentals of algorithm-guided acute resuscitation is critical, while simultaneously considering potentially reversible underlying etiologies. In this case report, we describe an unusual cause of cardiac arrest and a successful but rarely used intervention to stabilize a patient in acute cardiac arrest, both of which are applicable to emergency physicians' clinical practice.

Hypokalemia is a well-known etiology of cardiac dysrhythmia and a potentially reversible cause of cardiac arrest. By altering cardiac myocytes' action potential duration and resting membrane potential, hypokalemia can prolong membrane repolarization and promote

Written informed consent was obtained from the patient for the publication of this case report and any accompanying data. A copy of the written consent is available for the Editor-in-Chief of this journal.

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dysrhythmogenesis (1). The American Heart Association Advanced Cardiac Life Support algorithm states that hypokalemia is an etiology to be considered during cardiopulmonary resuscitation (CPR), though no specific intervention apart from electrolyte repletion is advised (2). Although postcardiac arrest patients demonstrate, on average, lower-than-normal serum potassium, the incidence of cardiac arrest directly attributed to hypokalemia is low (3). In one tertiary care center's experience, 5% of cardiac arrests were associated with a serum potassium level under 2.5 mEq/L, frequently in the context of an underlying eating disorder or diuretic abuse (3). Iatrogenic hypokalemia has been reported secondary to diuretics, antibiotics, insulin, and other medications (4–12). However, the incidence of hypokalemic cardiac arrest is rare, and Medline search using the keywords "hypokalemia," "hypopotassemia," and "cardiac arrest" identified fewer than 30 reports from 1967 to 2014 of cardiac arrest directly attributed to hypokalemia. A comprehensive listing of these studies is presented in Table 1. Of these

Table 1. Studies Reporting Hypokalemia-associated Cardiac Arrests, with the Suspected Cause of Hypokalemia

Citation	Case Report	Nadir K (mEq/L)	Proposed Cause of Hypokalemia	Cardiac Rhythm
Paulley (22)	Yes	3.0	Unknown	Asystole
Ohmae et al. (23)	Yes	2.3	Unknown	A Fib
Bannister et al. (15)	Yes	1.3	Licorice	V fib
Bashir & Tomson (10)	Yes	2	Mithramycin	Unknown, pulseless for 60 seconds without a cardiac monitor
Binder et al. (8)	Yes	2.6	Insulin tolerance test	V fib
Notarstefano et al. (24)	Yes	2.9	Chron's disease and Brugada syndrome	A fib to V fib
Aravena et al. (4)	Yes	2	Furosemide abuse	Sinus (no arrest)
Maeder et al. (5)	Yes (series of 4 patients)	2.9	Torsemide and metolazone	V tach
		2.6	Furosemide, metolazone, and spironolactone discontinuation	V tach
		3.2	Butizide	V tach
		2.6	Furosemide/hydrochlorothiazide	V tach
Scognamiglio et al. (25)	Yes (series of 2 patients)	1.7	Barters syndrome	V tach
		2.1	Gitelman syndrome	V tach
Schwartz et al. (26)	Yes (series of 2 patients)	1	Amantadine ingestion	V tach
		3	Amantadine ingestion	Sinus tachycardia
Chakravarty et al. (7)	Yes	3.1	Incidental hypokalemia with cardiac arrest due to fluconazole	V tach
Crean et al. (12)	Yes	1.6	Licorice	V fib
Jung et al. (27)	Yes	1	Thiopental	Bradycardia
Siau (6)	Yes	1.7	Vancomycin	Pulseless V tach
Bansal et al. (28)	Yes	2.4	Gittelman syndrome with superimposed febrile illness	V fib
Facciorusso et al. (29)	Yes	2.4	Stress cardiac arrest not attributed to hypokalemia	Not captured but pulseless
Heaps & Gormley (30)	Yes	2.2	Paracetamol toxicity	V tach
Mirzoyev et al. (31)	No	3.2 ± 0.7	Hypothermia	Polymorphic V tach
Wu et al. (32)	Yes	2.6	Hydrofluoric acid burn	Asystole
Ohashi et al. (33)	Yes	1.7	Poor oral intake and indapamide	V fib arrest
Sanei-Moghaddam et al. (34)	Yes	1.4	Emesis superimposed on Pendred syndrome	V fib arrest
Seidler et al. (3)	No	1.1	Dysmorphophobic eating disorders and furosemide abuse	Unknown
Struck & Nowak (11)	Yes	2.8	Orthopedic surgery	V fib arrest
Abdulaziz et al. (9)	Yes	Unknown	Insulin and bicarbonate treatment for diabetic ketoacidosis	Asystole
Chen et al. (35)	Yes	1.15	Thyrotoxic periodic paralysis treated with high carbohydrate load	Unknown (deceased)
Ewan & Moynihan (36)	Yes	1.8	Restrictive purging-type anorexia nervosa	Out of hospital (unknown)
Ruisz et al. (37)	Yes	1.1	Furosemide abuse	No cardiac arrest observed

V fib = ventricular fibrillation; v tach = ventricular tachycardia; A fib = atrial fibrillation.