# Electrolyte Abnormalities in Patients Presenting With Ventricular Arrhythmia (from the LYTE-VT Study)

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**Electrolyte abnormalities are a known trigger for ventricular arrhythmia, and patients with heart disease on diuretic therapy may be at higher risk for electrolyte depletion. Our aim was to determine the prevalence of electrolyte depletion in patients presenting to the hospital with sustained ventricular tachycardia or ventricular fibrillation (VT/VF) versus heart failure, and identify risk factors for electrolyte depletion. Consecutive admissions to a tertiary care hospital for VT/VF were identified between July 2016 and October 2018 using the electronic medical record and compared with an equal number of consecutive admissions for heart failure (CHF). The study included 280 patients (140 patients in each group; mean age 63, 60% male, 59% African American). Average EF in the VT/VF and CHF groups was 30% and 33%, respectively. Hypokalemia (K < 3.5 mmol/L) and severe hypokalemia (K < 3.0 mmol/L) were present in 35.7% and 13.6%, respectively, of patients with VT/VF, compared to 12.9% and 2.7% of patients with CHF ($p < 0.001$ and $p = 0.001$, respectively, between groups). Hypomagnesemia was found in 7.8% and 5.8% of VT/VF and CHF patients, respectively ($p = 0.46$). Gastrointestinal illness and recent increases in diuretic dose were strongly associated with severe hypokalemia in VT/VF patients (odds ratio: 11.1 and 21.9, respectively; $p < 0.001$). In conclusion, hypokalemia is extremely common in patients presenting with VT/VF, much more so than in patients with CHF alone. Preceding gastrointestinal illness and increase in diuretic dose were strongly associated with severe hypokalemia in the VT/VF population, revealing a potential opportunity for early intervention and arrhythmia risk reduction.**  **(Am J Cardiol 2020;129:36−41)**

Patients admitted with ventricular tachycardia and ventricular fibrillation (VT/VF) have a high inpatient mortality, especially those with heart failure.[1] Electrolyte disturbances, particularly hypokalemia and hypomagnesemia, have long been implicated in the development of VT/VF,[2−4] and patients with heart disease may be at increased risk of electrolyte disturbance for several reasons, including chronic diuretic therapy. Identifying electrolyte abnormalities and risk factors for the development of electrolyte abnormalities in these patients may represent a treatable target to reduce the incidence of VT/VF. We hypothesized that electrolyte disturbances would be associated with emergency department presentations for VT/VF. We determined the prevalence of and risk factors for electrolyte abnormalities in this patient population, compared with a control cohort of patients presenting with congestive heart failure (CHF) without VT/VF.

## Methods

The study was approved by our institution's IRB (Temple University). No informed consent was required. Retrospective chart review identified consecutive patients presenting to our inner-city emergency department with VT/VF between July 2016 and October 2018. VT/VF was defined as presenting with a primary diagnosis of sustained ventricular arrhythmia, cardiac arrest due to ventricular arrhythmia, or appropriate implanted cardioverter defibrillator shock for ventricular arrhythmia. A comparison group of consecutive patients admitted with CHF over the time period was identified. Each patient's chart was reviewed to confirm the clinical circumstances of the presentation and the admission diagnosis. Hypokalemia was defined as a serum potassium less than 3.5 mmol/L and subdivided into mild to moderate hypokalemia (3.0 to 3.5 mmol/L) and severe hypokalemia (<3.0 mmol/L). Hypomagnesemia was defined as a serum magnesium less than 1.6 mmol/L. Nausea, vomiting, and/or diarrhea (N/V/D) was considered present on admission if it was documented in the history of present illness or review of systems. The presence of a recent increase is diuretic dose was defined as an increase in a patient's loop or thiazide diuretic dose <2 weeks prior to admission. Study size was based on a power calculation to provide 80% power to detect an absolute difference in the prevalence of hypokalemia of 15% in the VT/VF group compared with the heart failure group, assuming a 20% baseline prevalence of hypokalemia.[5,6] In an unadjusted analysis, prevalence of potassium and magnesium abnormalities were compared using the Chi-squared method and mean electrolyte values were compared using a two sided *t* test. Univariate and multivariate analysis using linear and logistic regression was performed using the R statistical

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software (R Foundation for Statistical Computing, Vienna, Austria). All multivariate models were adjusted for age, gender, race, and body mass index with additional adjustments specified in the Tables 3 to 5. Predictors of VT/VF and hypokalemia were identified a priori. Given the small prevalence of hypomagnesemia in our cohorts, we did not use hypomagnesemia as a dependent variable. Separate analyses were performed using serum potassium as both a continuous and a categorical variable. Multivariate subgroup analysis excluding patients with cardiac arrest prior to initial electrolyte measurement (Subgroup A), univariate analysis excluding all patients who suffered arrest or defibrillation (Subgroup B) prior to measurement of serum electrolytes, and subgroup analysis by type of VT/VF were also performed.

Table 1
Baseline characteristics at presentation

| | VT/VF (n = 140*) | CHF (n = 140) | p value |
|---|---|---|---|
| Age (years) | 61 ± 13 | 65 ± 14 | 0.02 |
| Men | 89 (63.6%) | 79 (56.4%) | 0.22 |
| Clinical Characteristics | | | |
| Non-ischemic Cardiomyopathy | 69 (50.4%) | 72 (51.4%) | 0.86 |
| Ischemic Cardiomyopathy | 39 (28.4%) | 33 (23.6%) | 0.35 |
| Left Ventricular Ejection Fraction | 30 ± 18 | 33 ± 19 | 0.15 |
| Coronary Artery Disease | 63 (45.7%) | 64 (45.7%) | 0.99 |
| Hypertension | 120 (87.6%) | 129 (92.1%) | 0.21 |
| Diabetes Mellitus | 53 (38.7%) | 58 (42.1%) | 0.56 |
| Chronic Kidney Disease | 53 (38.7%) | 91 (65%) | <.001 |
| Acute Kidney Injury | 32 (23.7%) | 33 (23.6%) | 0.98 |
| Additional Diagnoses at Admission | | | |
| Decompensated Heart Failure | 19 (14.2%) | – | |
| Acute Coronary Syndrome | 27 (20.3%) | – | |
| Prescribed Antiarrhythmic Drug | | | |
| Any Antiarrhythmic Drug | 50 (37.3%) | – | |
| Amiodarone | 34 (20.1%) | – | |
| Sotalol | 10 (7.5%) | – | |
| Mexilitene | 11 (8.2%) | – | |
| Dofetilide | 2 (1.5%) | – | |
| Flecainide | 1 (0.7%) | – | |
| Combination Therapy | 8 (6.0%) | – | |
| Ventricular Arrhythmia Type | | | |
| Monomorphic VT | 74 (55.2) | – | |
| Polymorphic VT/VF | 60 (44.8) | – | |
| Pre-existing ICD | 90 (67.2) | – | |
| Baseline Medications | | | |
| Loop Diuretic | 82 (60.7%) | 110 (78.6%) | 0.001 |
| Thiazide | 25 (18.5%) | 15 (10.7%) | 0.07 |
| Spironolactone | 30 (22.2%) | 19 (13.6%) | 0.06 |
| Beta-blocker | 107 (79.3%) | 119 (85%) | 0.21 |
| ACEi/ARB | 73 (54.1%) | 78 (55.7%) | 0.78 |
| Potassium supplement | 34 (25.2%) | 25 (17.9%) | 0.14 |
| Magnesium supplement | 24 (17.8%) | 17 (12.1%) | 0.19 |
| Digoxin | 24 (17.8%) | 12 (8.6%) | 0.02 |

Values are mean ± SD or n (%).

ACEi/ARB = angiotensin converting enzyme inhibitor/angiotensin receptor blocker; CHF = congestive heart failure; ICD = implantable cardioverter defibrillator; VT/VF = ventricular tachycardia/ventricular fibrillation.

* 6 cardiac arrest patients did not uniformly have patient comorbidity information available, therefore for each patient characteristic, n < 140.

## Results

We identified 140 consecutive patients presenting to the emergency department with a primary diagnosis of VT/VF, and a comparison group of the 140 most recent patients presenting with heart failure during the same study period. Baseline characteristics of the VT/VF and heart failure groups are shown in Table 1. Comparison of electrolyte abnormalities between groups is demonstrated in Figure 1. Patients presenting with VT/VF more often had hypokalemia and severe hypokalemia compared with those presenting with CHF (35.7% vs 12.9% and 13.6% vs 2.7%, respectively, $p \leq 0.001$ for both). On average, serum potassium level on admission was lower among those with VT/VF (Table 2). The prevalence of hypomagnesemia was similar in each group (7.8% with VT/VF vs 5.8% with CHF, $p = 0.46$), and mean serum magnesium was also similar (Table 2). In multivariate regression analysis, decreasing serum potassium was strongly associated with VT/VF admissions ($p < 0.001$). Chronic kidney disease (CKD) and loop diuretic use were negatively associated with VT/VF admissions while digoxin use was positively associated (Table 3).

Among patients presenting to the hospital with VT/VF, N/V/D was markedly more common in patients with severe hypokalemia versus mild to moderate hypokalemia (33% vs 3.4%, $p < 0.005$) and versus those with normal serum potassium (4.6%, $p < 0.001$). Among patients with VT/VF and severe hypokalemia, 28% had their diuretic dose increased within 2 weeks prior to admission, compared to none of the patients with mild to moderate hypokalemia ($p < 0.003$), and 2.3% of patients with normal serum potassium ($p < 0.001$; Figures 2 and 3). Neither of these predictors (antecedent N/V/D and recent diuretic dose escalation) were associated with VT/VF admissions in our adjusted multivariate analysis.

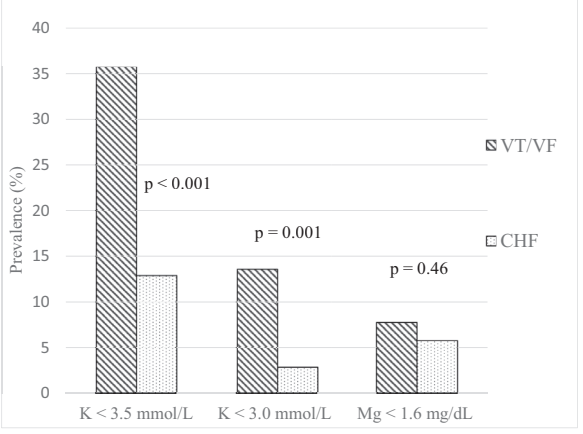


Figure 1. Prevalence of electrolyte abnormalities in patients with ventricular arrhythmia (VT/VF) versus congestive heart failure. CHF = congestive heart failure; K = potassium; Mg = magnesium; VT/VF = ventricular tachycardia/ventricular fibrillation.

Table 2
Serum electrolytes on admission

| | VT/VF | VT/VF subgroup A* | VT/VF subgroup B† | CHF | p value‡ |
|---|---|---|---|---|---|
| Potassium (mmol/L) | 3.7 ± 0.6 | 3.7 ± 0.6 | 3.7 ± 0.5 | 4.1 ± 0.7 | <0.001 |
| Magnesium (mmol/L) | 2.0 ± 0.4 | 2.0 ± 0.3 | 1.9 ± 0.3 | 2.0 ± 0.3 | 0.9 |

Values displayed as mean ± standard deviation.
* Subgroup A excludes patients with electrolytes measured post cardiac arrest.
† Subgroup B excludes patients with electrolytes measured post cardiac arrest and post ICD/external shock.
‡ Based on two sided *t* test, comparison for full VT/VF cohort. p values for VT/VF Subgroup A are: p < 0.001 (potassium) and p = 0.2 (magnesium). p values for Subgroup B are: p = 0.005 (potassium) and p = 0.07 (magnesium).
VT/VF = ventricular tachycardia/ventricular fibrillation.

Table 3
Risk factors for presentation with ventricular arrhythmia

| Risk Factor | Full VT/VF cohort (N = 134)* | | VT/VF Subgroup A (N = 110)*,‡ | | VT/VF subgroup B (N = 31)†,§ | |
|---|---|---|---|---|---|---|
| | OR (95% CI) | p value | OR (95% CI) | p value | OR (95% CI) | p value |
| Electrolytes | | | | | | |
| Lower Serum Potassium¶ | 2.39 (1.47−4.01) | <0.001 | 2.84 (1.65−5.13) | <0.001 | 2.75 (1.38−5.89) | 0.006 |
| Lower Serum Magnesium¶ | 0.56 (0.22−1.34) | 0.21 | 0.61 (0.20−1.81) | 0.38 | 3.22 (0.94−12.32) | 0.07 |
| Patient Characteristics | | | | | | |
| Non-Ischemic Cardiomyopathy | 0.85 (0.25−2.84) | 0.79 | 0.9 (0.24−3.29) | 0.87 | 0.30 (0.11−0.79) | 0.02 |
| Ischemic Cardiomyopathy | 1.53 (0.43−5.47) | 0.51 | 2.01 (0.50-8.14) | 0.32 | 0.82 (0.31−2.11) | 0.68 |
| Lower Ejection Fraction¶ | 1.02 (1.00−1.05) | 0.10 | 1.02 (0.99−1.05) | 0.19 | 0.99 (0.97−1.01) | 0.24 |
| Coronary Artery Disease | 0.82 (0.36−1.85) | 0.64 | 0.6 (0.24-1.44) | 0.26 | 0.85 (0.38-1.85) | 0.68 |
| Hypertension | 1.48 (0.50−4.37) | 0.48 | 2.15 (0.68−7.22) | 0.2 | 1.25 (0.31−8.33) | 0.78 |
| Diabetes Mellitus | 1.24 (0.64−2.41) | 0.53 | 1.21 (0.60−2.46) | 0.59 | 0.98 (0.44−2.14) | 0.96 |
| Chronic Kidney Disease | 0.4 (0.20−0.79) | 0.01 | 0.44 (0.21−0.89) | 0.02 | 0.25 (0.10-0.57) | 0.001 |
| Acute Kidney Injury | 1.25 (0.61−2.57) | 0.54 | 1.04 (0.48−2.26) | 0.91 | 0.62 (0.20−1.62) | 0.36 |
| Medication Use | | | | | | |
| Loop Diuretic use | 0.22 (0.10−0.48) | <0.001 | 0.24 (0.11−0.55) | <0.001 | 0.17 (0.07−0.39) | <0.001 |
| Thiazide diuretic use | 1.22 (0.51−2.93) | 0.66 | 1.1 (0.43−2.78) | 0.83 | 1.59 (0.48−4.52) | 0.41 |
| Aldactone use | 1.37 (0.59−3.17) | 0.46 | 1.61 (0.68−3.83) | 0.28 | 1.29 (0.40-−3.59) | 0.64 |
| Beta Blocker use | 1.35 (0.58−3.17) | 0.49 | 1.62 (0.66−4.07) | 0.3 | 0.37 (0.16−0.93) | 0.03 |
| ACEi/ARB use | 0.66 0.34−1.26) | 0.21 | 0.74 (0.37−1.45) | 0.38 | 0.66 (0.30−1.45) | 0.30 |
| Potassium supplement use | 1.33 (0.60−2.92) | 0.48 | 1.59 (0.70−3.62) | 0.26 | 1.33 (0.49−3.31) | 0.56 |
| Magnesium supplement use | 1.52 (0.62−3.72) | 0.36 | 1.57 (0.62−3.99) | 0.34 | 0.82 (0.18−2.69) | 0.77 |
| Digoxin use | 3.03 (1.17−8.16) | 0.02 | 3.12 (1.17−8.67) | 0.03 | 1.25 (0.27−4.31) | 0.75 |

* Multivariate logistic regression, adjusted for age, gender, race, body mass index.
† Univariate logistic regression.
‡ Subgroup A excludes patients with cardiac arrest prior to initial serum electrolyte measurement.
§ Subgroup B excludes patients who suffered arrest or defibrillator shock prior to initial serum electrolyte measurement.
¶ Continuous variable, odds ratio expressed for one unit decrease (mmol/L potassium, mg/dl magnesium, % ejection fraction).
ACEi/ARB = angiotensin converting enzyme inhibitor/angiotensin receptor blocker; VT/VF = ventricular tachycardia/ventricular fibrillation.

In multivariate regression analysis, the presence of N/V/D was associated with lower serum potassium while beta blocker use was associated with higher serum potassium. Lower serum magnesium and preceding diuretic dose augmentation were associated with hypokalemia (Table 4). Univariate logistic regression showed that N/V/D and a recent increase in diuretic dose were both strong predictors of severe hypokalemia in patients with VT/VF. Thiazide diuretic use and beta blocker nonuse were additional predictors of severe hypokalemia (Table 5**).**

Similarly, subgroup analyses of Subgroup A and Subgroup B demonstrated a strong association of lower serum potassium with VT/VF (Table 2 and 3). Subgroup analysis of the subtypes of ventricular arrhythmia also demonstrated a consistent association of lower serum potassium with presentation for both monomorphic VT and polymorphic VT/VF (Supplementary Tables 1 and 2).

## Discussion

In a retrospective review of patients presenting to the hospital with VT/VF, we found that more than a third of patients with VT/VF had hypokalemia on admission, a prevalence that was three times that found in heart failure patients. Nearly 1 in 7 patients with VT/VF had severe hypokalemia, far more common than what was observed in heart failure patients. Similarly compelling, in VT/VF patients with severe hypokalemia, one third had preceding gastrointestinal symptoms and one third had a recent increase in their diuretic dose, highlighting the importance of recognizing these risk factors for electrolyte depletion. It is important to note that a similar proportion of both groups were on potassium sparing agents (ACEi/ARB and aldosterone antagonist), and a higher proportion of patients in the heart failure group were on diuretics,

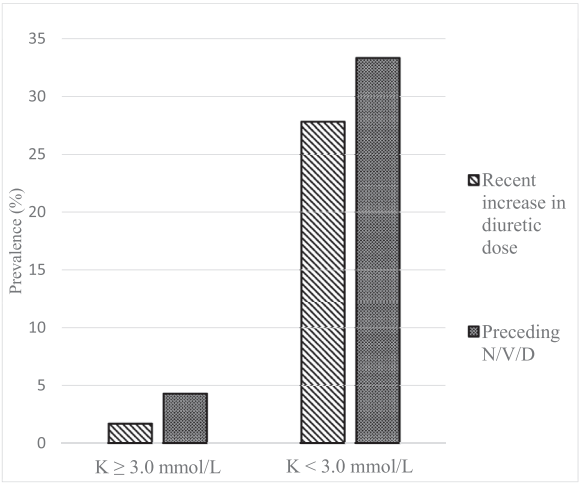


Figure 2. Prevalence of preceding gastrointestinal illness and diuretic augmentation in patients presenting with ventricular arrhythmia, stratified by serum potassium. K = potassium, N/V/D = nausea, vomiting, and/or diarrhea.

yet the rate of hypokalemia was still three-fold higher in the VT/VF group.

Hypokalemia is a known trigger of VT/VF,[2–4] and it exerts its proarrhythmic effects via a reduction in repolarization reserve leading to early and delayed after depolarizations.[7] Hypokalemia-mediated triggered PVCs can induce monomorphic reentrant VT if substrate exists, and early after depolarizations are known to initiate VF. In prior small studies, the rate of hypokalemia has ranged from 14% in patients presenting with electrical storm,[8] and as high as 41% in patients resuscitated after receiving defibrillation for out-of-hospital cardiac arrest.[9] These studies of course represent subgroups of all comers with VT/VF, and they are limited by the fact that post-arrest patients or patients with electrical storm requiring defibrillation may have hypokalemia as a result of their arrests and shocks, due to a high catecholamine state, or catecholamine therapy during the arrest, all leading to transcellular shift of potassium into cells.[9] In a study of patients with acute myocardial infarction and VF, hypokalemia remained a risk factor for VF after excluding patients with potassium measured after VF.[10] After we excluded patients with serum electrolytes initially drawn after cardiac arrest or any defibrillation, lower serum potassium remained strongly associated with VT/VF. Hypomagnesemia is also a known trigger of VT/VF, especially in cases of Torsades de Pointes, and may also have a direct potassium lowering effect through augmentation of potassium wasting in the urine.[11] We found that hypokalemia was significantly more common than hypomagnesemia in patients presenting with VT/VF, and hypomagnesemia was no more common in VT/VF patients than in patients admitted with heart failure. These results may be limited by an underrepresentation of Torsades patients, but suggest that hypokalemia is the more relevant electrolyte abnormality.

In our adjusted analysis, loop diuretic use was strongly associated with admission for CHF, which is not unexpected given the high rate of diuretic use in this population. This suggests that simply being on a loop diuretic is not a risk factor in isolation for developing hypokalemia or VT/VF. In fact, our multivariate analysis showed that thiazide diuretics, and not loop diuretics, were most important with regard to the risk of developing hypokalemia. It may be that particular attention should therefore be given to patients on thiazide diuretics, and prior work has demonstrated that even low dose thiazide diuretics used to treat hypertension result in a significant rate of hypokalemia.[12]

Furthermore, the presence of N/V/D and a recent increase in diuretic dose both emerged as strong univariate predictors of severe hypokalemia. Our study suggests that it may be prudent to be aggressive about laboratory monitoring and potassium supplementation during anticipated potassium loss

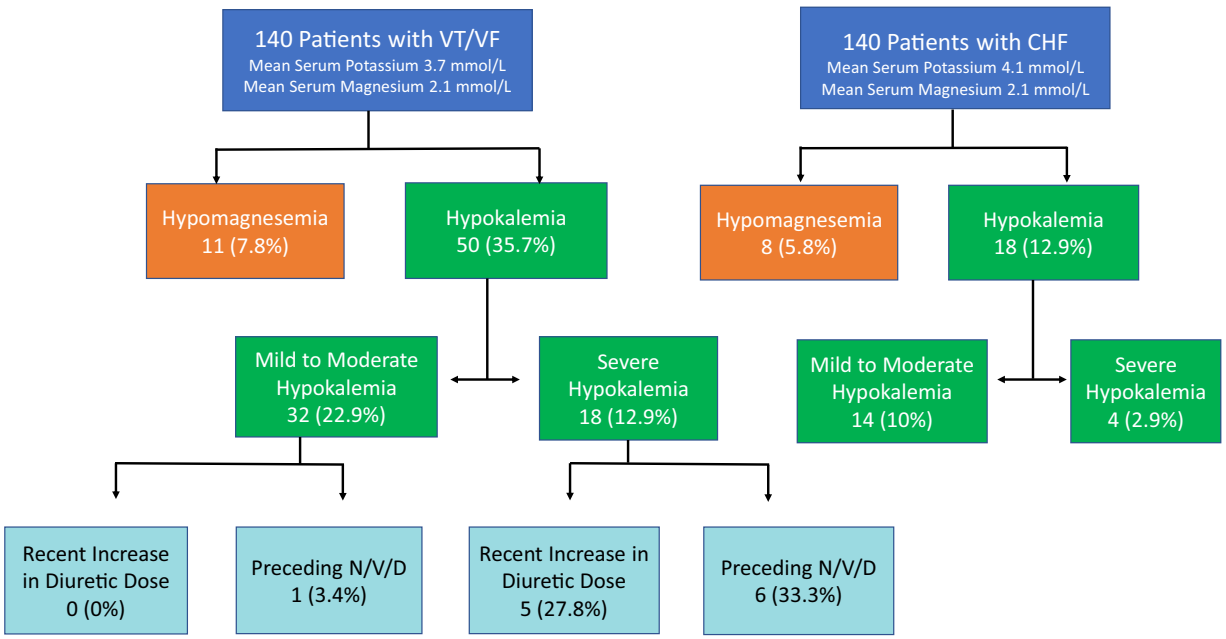


Figure 3. Flowchart of patient electrolyte abnormalities by ventricular arrhythmia versus heart failure presentation. CHF = congestive heart failure, N/V/D = nausea, vomiting, and/or diarrhea; VT/VF = ventricular tachycardia/ventricular fibrillation.

Table 4
Predictors of lower serum potassium* and hypokalemia† in VT/VF patients

| Outcome | Risk factor | Odds ratio | 95% CI | p Value |
|---|---|---|---|---|
| Lower Serum Potassium | Lower Serum Magnesium‡ | 1.22 | 0.92−1.62 | 0.16 |
| | Chronic Kidney Disease | 0.77 | 0.56−1.05 | 0.10 |
| | Acute Kidney Injury | 1.00 | 0.76−1.33 | 0.96 |
| | Loop Diuretic use | 0.94 | 0.67−1.33 | 0.73 |
| | Thiazide diuretic use | 1.25 | 0.93−1.69 | 0.15 |
| | Aldactone use | 1.01 | 0.74−1.39 | 0.94 |
| | Beta Blocker use | 0.69 | 0.50−0.96 | 0.03 |
| | ACEi/ARB use | 0.79 | 0.60−1.03 | 0.08 |
| | Potassium supplement use | 0.99 | 0.74−1.32 | 0.92 |
| | Magnesium supplement use | 1.06 | 0.76−1.49 | 0.69 |
| | Digoxin use | 0.90 | 0.65−1.25 | 0.53 |
| | N/V/D | 1.72 | 1.14−2.63 | 0.01 |
| | Recent Diuretic Increase | 1.59 | 0.94−2.63 | 0.08 |
| Hypokalemia (K < 3.5 mmol/L) | Lower Serum Magnesium‡ | 4.81 | 1.54−19.15 | 0.01 |
| | Chronic Kidney Disease | 0.92 | 0.30−2.74 | 0.87 |
| | Loop Diuretic use | 0.80 | 0.28−2.41 | 0.69 |
| | Beta Blocker use | 0.28 | 0.08−0.89 | 0.03 |
| | ACEi/ARB use | 0.70 | 0.27−1.81 | 0.45 |
| | N/V/D | 2.16 | 0.44−10.87 | 0.33 |
| | Recent Diuretic Increase | 11.44 | 1.51−188.81 | 0.03 |

* Multivariate linear regression, adjusted for age, gender, race, body mass index, and baseline comorbidities (coronary artery disease, cardiomyopathy, hypertension, and diabetes) with potassium measured as a continuous variable and the outcome displayed as a one unit decrease in potassium (mmol/L).
† Multivariate logistic regression, choosing predictors with $p < 0.1$ during univariate analysis.
‡ Continuous variable, Odds Ratio expressed for one unit decrease (mg/dl).
ACEi/ARB = angiotensin converting enzyme inhibitor/ Angiotensin receptor blocker; N/V/D = nausea, vomiting, and/or diarrhea.

associated with increases in diuretic doses and gastrointestinal illnesses. Interestingly, increased outpatient electrolyte monitoring with changes in diuretic dose or during gastrointestinal illness is not addressed in the heart failure guidelines.[13,14]

The finding that CKD was associated with CHF admissions and not VT/VF or hypokalemia may be explained by the mitigating effect of CKD regarding urinary loss of potassium from diuretics. Digoxin use was associated with admission for VT/VF, and this is not surprising given the known pro-arrhythmic effects of digoxin, especially in combination with hypokalemia or renal failure.[15] Lower serum magnesium levels were associated with hypokalemia in our study, which is expected given hypomagnesemia itself can lead to increased urinary losses of potassium, and most causes of gastrointestinal and urinary loss of potassium also lead to loss of magnesium. The fact that beta blocker use is associated with higher serum potassium could be explained by the downstream reduction in Na+/K+ ATPase activity from beta blockade, which results in decreased active transport of potassium intracellularly.[16]

Table 5
Predictors of severe hypokalemia, univariate analysis*

| Risk factor | Odds ratio | 95% CI | p value |
|---|---|---|---|
| Lower Serum Magnesium† | 2.85 | 0.71−14.45 | 0.18 |
| Chronic Kidney Disease | 0.46 | 0.12−1.37 | 0.19 |
| Acute Kidney Injury | 0.90 | 0.24−2.74 | 0.86 |
| Loop Diuretic use | 1.00 | 0.36−2.88 | 0.99 |
| Thiazide diuretic use | 3.46 | 1.14−10.06 | 0.02 |
| Aldactone use | 1.40 | 0.42−4.11 | 0.56 |
| Beta Blocker use | 0.15 | 0.05−0.42 | <.001 |
| ACEi/ARB use | 0.50 | 0.17−1.36 | 0.18 |
| Magnesium supplement use | 1.37 | 0.36−4.30 | 0.61 |
| Potassium supplement use | 1.15 | 0.35−3.35 | 0.80 |
| Digoxin use | 1.37 | 0.36−4.30 | 0.61 |
| Preceding N/V/D | 11.10 | 2.94−44.15 | <.001 |
| Recent Diuretic Dose Increase | 21.92 | 4.27−164.41 | <.001 |

* Univariate Logistic Regression.
† Continuous Variable, Odds Ratio expressed for one unit decrease (mg/dl).
Severe Hypokalemia = Serum Potassium < 3.0 mmol/L.
N/V/D = Nausea, vomiting, and/or diarrhea.

The present study had several limitations. As a retrospective prevalence study, causative relationships cannot be concluded. Although preceding N/V/D and diuretic dose augmentation were both associated with severe hypokalemia, the study was not powered to assess if they are independently associated with VT/VF. Using a comparison group of heart failure patients may limit this assessment as GI symptoms can be caused by heart failure, and outpatient diuretic dose adjustments are common in patients with CHF, especially in those requiring hospital admission. We did not stratify patients by the dosage of diuretics that were prescribed, or quantify the increase in diuretic dose. Higher digoxin serum levels are associated with increased mortality in patients with atrial fibrillation,[17] and it would have been ideal to incorporate digoxin levels in our study, however the total number of patients on digoxin was low, and serum levels were only drawn on a subset of these patients. A notable minority of patients in the VT/VF group had an acute coronary syndrome (n = 27), and this subgroup of patients was not compared to acute coronary syndrome patients without VT/VF as has been done in prior studies. Finally, although subgroup analysis revealed a strong association of lower serum potassium with both monomorphic VT and VF, additional research is needed

to characterize differences in risk factors for each ventricular arrhythmia subtype.

## Conclusion

In conclusion, among patients presenting to the emergency department for VT/VF versus heart failure, lower serum potassium was strongly associated with VT/VF, and hypokalemia and severe hypokalemia were markedly more common in the VT/VF group compared with the heart failure group. Among patients with VT/VF, a recent increase in diuretic dose and the presence of nausea/vomiting/diarrhea on admission were strongly associated with severe hypokalemia. These findings suggest that more aggressive electrolyte management at times of predictable potassium losses could represent an underappreciated antiarrhythmic strategy for patients with VT/VF.

## Author contribution

David Laslett: Conceptualization, Methodology, Visualization, Data Curation, Investigation, Formal Analysis, Software, Writing – Original Draft preparation. Joshua Cooper: Conceptualization, Methodology, Supervision, Project Administration, Writing – Review and Editing. Richard Greenberg: Writing – Review and Editing. George Yesenosky: Writing – Review and Editing. Anuj Basil: Writing – Review and Editing. Chethan Gangireddy: Methodology, Conceptualization. Isaac Whitman: Conceptualization, Methodology, Visualization, Supervision, Project Administration, Formal Analysis, Writing – Review and Editing.

## Disclosures

None to report.

## Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.amjcard.2020.04.051.


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