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Case Report

Hypokalemia Induced Ventricular Arrhytmia In Heart Failure Patient With Complete Revascularization: A Case Report

Icmi Dian Rochmawati[1*], Ardian Rizal[2], Mohammad Saifur Rohman[2], Indra Prasetya[2]

[1]Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.
[2]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.

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ABSTRACT

Background: Sudden mortality due to persistent VT or VF accounted for around half of all fatalities in these high-risk individuals. Myocardial ischemia, acute heart failure, electrolyte abnormalities, hypoxia, and drug-related arrhythmogenicity are all risk factors for electrical storms. The most common electrolyte imbalance is hypokalemia.
Case Illustration: A 54-year-old man was readmitted to ER with palpitations and chest pain. The patient's heart rate was recorded as sinus bradycardia however, shortly the patient developed ventricular tachycardia of approximately 300 beats per minute (bpm) and unstable. Although multiple synchronized cardioversion dosage was administered, the VT reoccurred again. Complete revascularization was demonstrated at his most recent catheterization three months ago. His potassium in the serum was 2.88 mmol/L and corrected with drip KCl. The patient's potassium levels were then normalized stable for the remainder of their hospital stay.
Conclusion: Careful medication reconciliation is critical for avoiding the potentially fatal cardiovascular effects of severe hypokalemia. Patients with CHF are more likely to have life-threatening hypokalemia and ventricular arrhythmias. The phenotypic expression of ventricular tachycardia in HF results from alterations in neurohormonal signaling, structural remodeling, and electrophysiology.

1. Introduction

Those who report ventricular arrhythmias in expansion to an intense MI have a much more prominent mortality rate. In expansion to changing the normal course of an infarct, essential percutaneous coronary intercession (PCI) and beta-blocker utilization have diminished the predominance of maintained ventricular tachycardia (VT) or ventricular fibrillation (VF) inside 48 hours after the graduation of an intense coronary disorder (ACS). Drugs counting antiplatelets, statins, pro inhibitors, and beta-blockers, at the side fast revascularization, have incredibly decreased the predominance of VA. In spite of this, post-MI survivors have a tall passing rate (approximately 25% at 2 a long time) within the months and a long time after clinic release, influencing over 10% of the populace. More than half of the fatalities in this high-risk gather were caused by tireless VT or VF. Patients with determined chest torment, those who have had fair fractional revascularization, and those who have an arrhythmogenic substrate stay at chance.[1,2]

Despite continued therapeutic advancements, the long-term prognosis for heart failure (HF) remains bleak, with a 5-year mortality rate that surpasses 50% overall and is much higher in more severe stages (NYHA III-IV). SCD accounts for 50% of all HF mortality and is often caused by ventricular tachyarrhythmias (VTs). It is well-recognized that hypokalemia increases the risk of ventricular arrhythmias and mortality after an acute myocardial infarction; it is also common and independently associated with worse clinical outcomes in HF patients.[3]

Hypokalemia is the condition that occurs when the plasma potassium level goes below 3.5 mmol/dL. Electrolyte imbalances are a typical problem in healthcare institutions and among outpatients. This disorder affects around 20 percent of hospitalized patients. Cardiovascular illnesses such as hypertension, congestive heart failure, and acute myocardial infarction increase the probability of hypokalemia. According to Savarese et al., 21% of CHF patients encountered hypokalemia at least once throughout the research period, with 3.7% suffering severe hypokalemia. Hypokalemia has catastrophic repercussions, including an increase in hospital mortality, as a result of its impact on cardiac rhythm and blood pressure. Hypokalemia raises the risk of sudden death in heart failure patients. Here, we report a case study illustrating how serious hypokalemia may

* Corresponding author at: Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.
E-mail address: icmidianr@hotmail.com (I.D. Rochmawati).

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be for individuals with HF. In addition, it underlines the need of medication reconciliation in averting such consequences. [4]

An electrical or arrhythmic storm is characterized by the rapid recurrence of ventricular arrhythmias. The most common kind of tachycardia is ventricular (VT), although atrial (AF) and polymorphic VT may also occur. Most patients with arrhythmias seek medical help to control their symptoms, although certain arrhythmias may go away on their own (defibrillation or anti-tachycardia pacing).[5]

Myocardial ischemia, electrolyte imbalances, acute heart failure, hypoxia, and drug-induced arrhythmogenicity are among situations that have been linked to electrical storms. Most commonly electrolyte imbalance that leads to lightning strikes is hypokalemia. Here, we provide a case of VTS that was successfully treated with intravenous potassium chloride after antiarrhythmic medications given intravenously had failed.[5]

2. Case Illustration

A 54-year-old male with previous acute coronary syndrome. The patient was brought to the hospital with complains of palpitations and chest pain due to a lifetime of cigarette smoking. Prior to his hospitalization, he had never had stomachache or diarrhea. On the electrocardiogram, leads II, III, and aVF had a reversed T wave. When the patient was first evaluated, their heart and lungs sounded normal. No more clinical symptoms of disease were seen. Two months before, the patient had comparable symptoms and was diagnosed with Ventricular tachycardia. His daily medicine regimen typically consisted of 80 mg aspirin, 2x90 mg ticagrelor, 20 mg atorvastatin, 2.5 mg bisoprolol, 5 mg ramipril, furosemide 40 mg and 25 mg spironolactone. He has previously had catheterization three times. His most recent catheterization, performed three months ago, demonstrated complete revascularization: the left main coronary artery was normal, the left anterior descending (LAD) was diffusely stenotic from proximal to mid-LA with maximal stenosis (90%) in proximal LAD, OM1 was diffusely stenotic with maximal stenotic 90% and the right coronary artery (RCA) showed insignificant lesions. A drug-eluting stent (DES) is implanted in the proximal and middle LAD. The patient reported to the emergency room (ER) with sinus bradycardia with PVC bigeminy, but soon afterwards developed ventricular tachycardia of around 300 beats per minute (bpm) and the individual became hemodynamically unstable. Three consecutive synchronized cardioversion with maximal dosage of 200 J was administered, however the VT reappeared nonetheless.

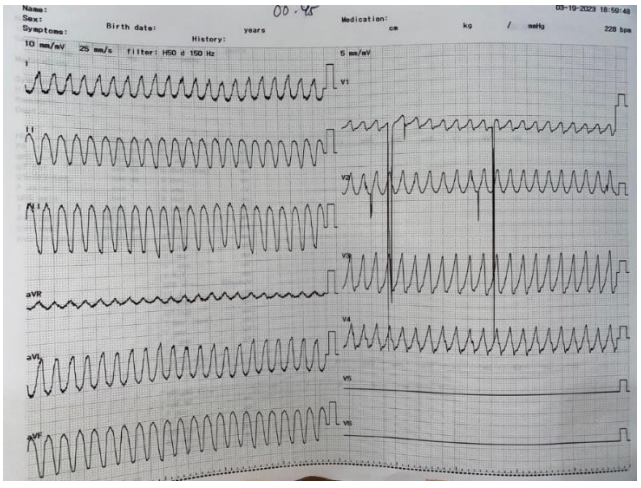

Figure 1. Electrocardiography Showed Monomorphic Ventricular Tachycardia

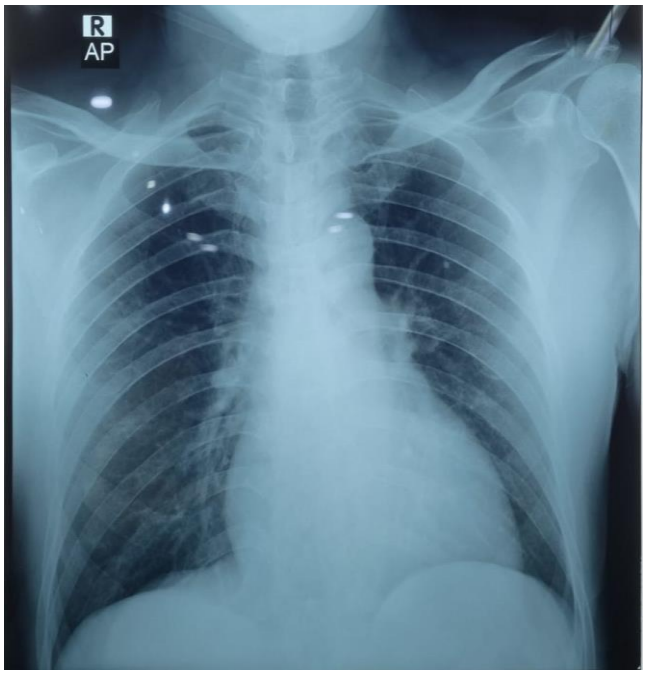

Figure 2. Chest X ray showed cardiomegaly with Left Ventricular Hypertrophy Configuration

The decision was made to implant a TPM capable of overdrive pacing in an effort to normalize the patient's heart rate. Serum potassium was measured to be 2.88 mmol/L. To control the patient's potassium levels, a central venous catheter (CVC) was placed. He was given a steady drip of KCl via her central line. The patient's hemodynamic stability was maintained throughout their hospital stay, and his potassium levels were restored to a satisfactory level (4.06 mmol/L).

Right ventricular wall motion abnormalities (RWMA) were seen in the anterior, anteroseptal, and anterolateral segments, and transthoracic echocardiography revealed a decrease function in left ventricular systolic with left ventricular ejection fraction of around 41% along with minimal mitral insufficiency dt leaflet tether and diastolic dysfunction gr I, according to the Transtorachal echocardiographic (TTE) findings. The doctor decided the patient's condition was stable enough to discharge him. An EP study was planned so that the possibility of implanting an ICD to prevent VT could be discussed with the patient.

The patient was discharged from critical care on day 7 after no longer experiencing bradycardia or palpitations after being treated by the hospital's cardiologist. The patient was then scheduled. The next step in preventing a recurrence of the VA was to have an ICD implanted in his chest.

3. Discussion

Those with a myocardial infarction who do not get treatment often die suddenly of vascular arrest (MI). Prior to the development of reperfusion therapies, beta-blockers, anti-thrombotic drugs, and statins, electrical cardioversion/defibrillation and acute volume overload therapy were among the most vital lifesaving operations in coronary care units. Numerous arrhythmias are induced by electrical changes in acutely ischemic myocardium, especially during the transitional period of a myocardial infarction (MI). In contemporary coronary care, the incidence of acute coronary events has been drastically reduced due to adequate revascularization therapy, which consisting of interventional reopening of occluded vessels and stent insertion of the culprit lesion, as well as secondary prevention drugs therapies drugs. When a patient has a history of myocardial infarction or a predisposition for electrophysiological instability (referred to as "pre-existing cardiac damage"), therapeutic options are more restricted. [2]

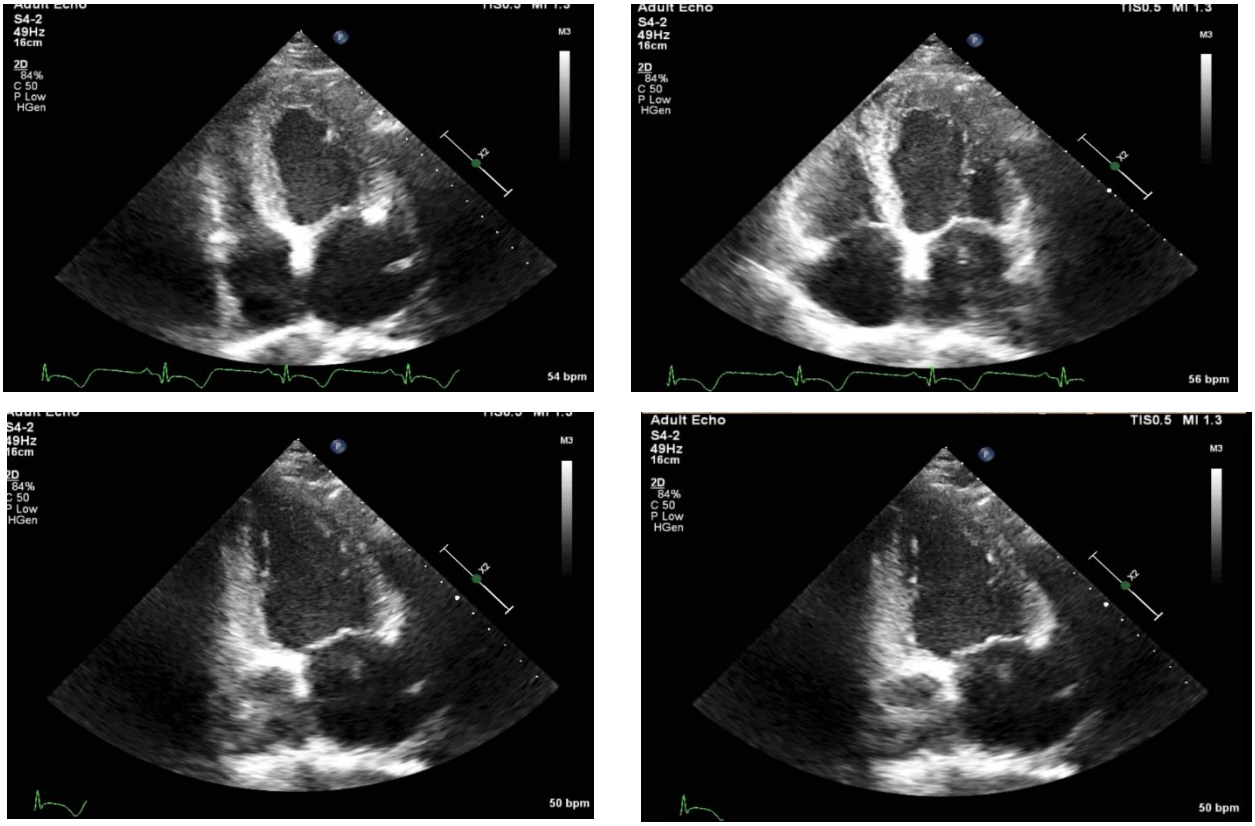

Figure 3. Echocardiography During Diastole And Systole Phase

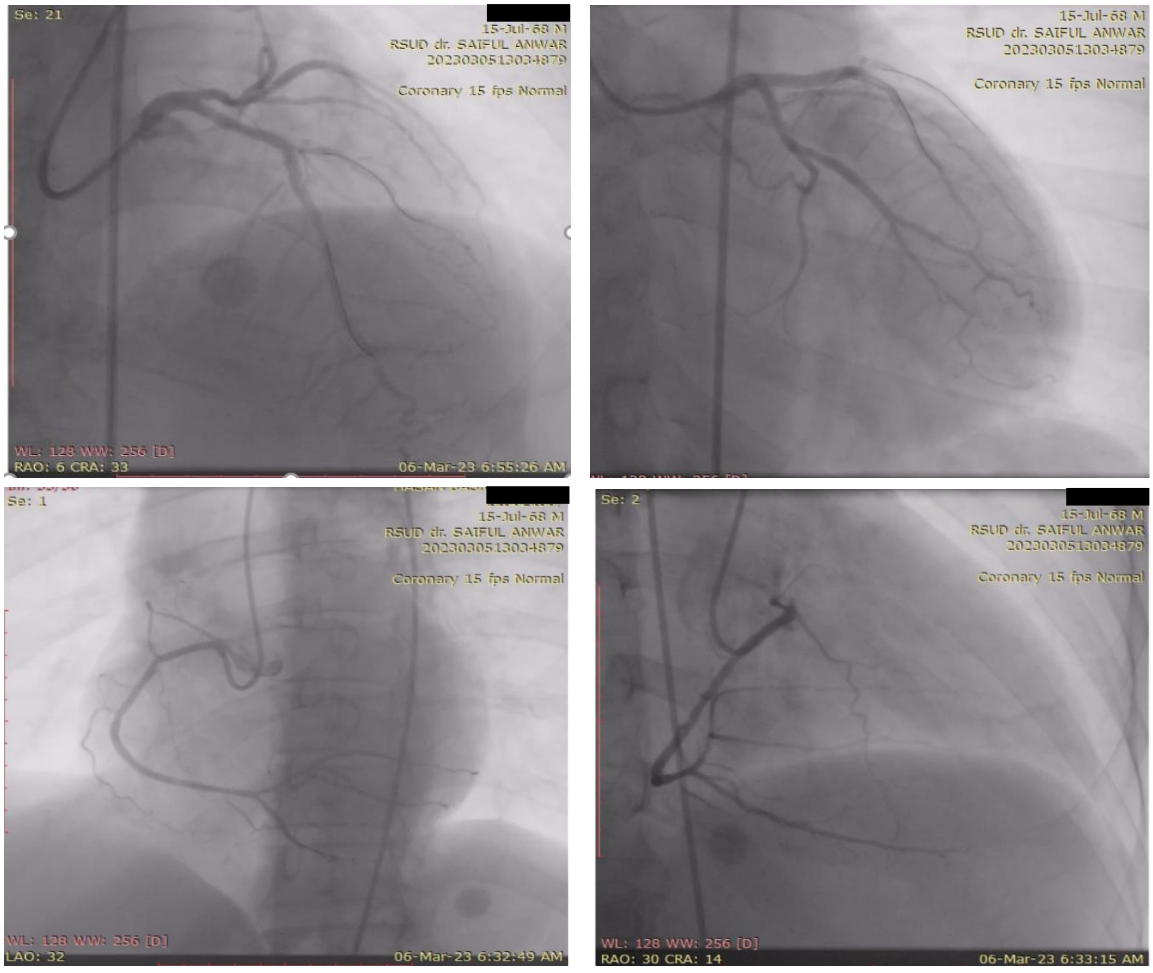

Figure 4. Angiography Showed Complete Revascularization In LAD And RCA With Non Significant Lesion In LCx
LAD Left Anterior Descending, RCA Right Coronary Artery, LCx Left Circumflex

In individuals with HFrEF, ventricular tachyarrhythmias (VA) are prevalent, and their incidence rises with increasing NYHA class. CHF's increased arrhythmogenicity is caused by a number of underlying pathophysiologic causes. These structures may or may not be found in the cardiovascular system. Important pathophysiologic events in CHF that contribute to VA include cardiac hypertrophy, fibrosis, and ischemia, as well as electrophysiologic abnormalities such as calcium homeostasis disturbances, repolarization, and gap junction remodeling. Substance dependence disorders, such as cocaine use, and adverse drug reactions are among the leading causes of VA in patients with CHF. [4]

Despite having had complete revascularization for her coronary problem as per the guidelines 3 months ago owing to a history of inferior STEMI, this patient has been suffering recurring bouts of ventricular tachycardia for the last 2 months. Since HF provides a substrate for patches of scar, even if the scar is not immediately apparent on echocardiogram, it is an anatomic substrate for reentrant VT. Fibrosis patterns cause electrical changes that might lead to potentially fatal ventricular arrhythmias. After an infarct, the scar tissue remodels, and the fibrotic areas become less localized. Fibrotic regions are associated with slow conduction because to their complex interweaving with the surviving myocardium, and these regions are responsible for electrogram fractionation and the development of re-entrant ventricular tachycardia. Re-entry requires slow conduction regions, a unidirectional conduction block, and areas of conduction block that generally mark the re-entry path. Slow conduction in patchy fibrotic areas around healed infarcts is linked to re-entry-induced VT, according to Pogwizd et al. Delayed conduction and poor cellular coupling perpendicular to the fiber route hinder impulse propagation and generate reentry circuits, leading to VT in infarcted heart tissue.[6] The hypokalemia exacerbated the problem greatly. Arrhythmia might result from hypokalemia's effect on the resting membrane potential, the threshold potential, and the sporadic automaticity. [5]

Patients with congestive heart failure often have hypokalemia. The heart problem itself, the medications used to treat heart failure (particularly diuretics), and a variety of other conditions may all have a role. Multiple components of the electrical stability of the heart have been discovered to be affected by hypokalemia. Our patient developed recurrent ventricular arrhythmia due to hypokalemia caused by diuretics and prior structural heart disease.[4]

Several physiological consequences may be brought on by hypokalemia, a frequent electrolyte imbalance that can be brought on by a wide range of situations. The normal range for blood potassium according to clinical laboratories is 3.6–5.0 mmol/L. Nonetheless, the findings of this research indicated, via the use of a high potassium dosage and a sluggish serum response, that serum levels are simply a representation of total body storage. Ninety percent of the body's potassium is located inside the cells. The resting membrane potential of heart muscle cells relies primarily on the gradient of intracellular potassium to extracellular potassium. [7]

A cardiac VT storm may be pharmacologically reset by antiarrhythmic drugs such as beta blockers, Amiodarone, and sodium channel blockers, or the correction of an unbalanced ion level, as in hypokalemia. Unfortunately, the patient's hemodynamic condition may have deteriorated after a transvenous pacemaker (TPM) was installed to prevent VT. In addition, the therapy of ventricular arrhythmia often involves reestablishing the correct ion balance. A potassium deficiency may be treated using intravenous solutions of varied concentrations.[5]

Antiarrhythmic medications or the correction of an imbalanced ion level, as in hypokalemia, may be able to pharmacologically reset a cardiac VT storm. Unfortunately, the patient's hemodynamic status may have worsened after we implanted a transvenous pacemaker (TPM) to avoid VT. In addition, ventricular arrhythmia treatment often includes reestablishing proper ion balance. Treatment for potassium shortage may include administering an intravenous solution of varying doses.[8]

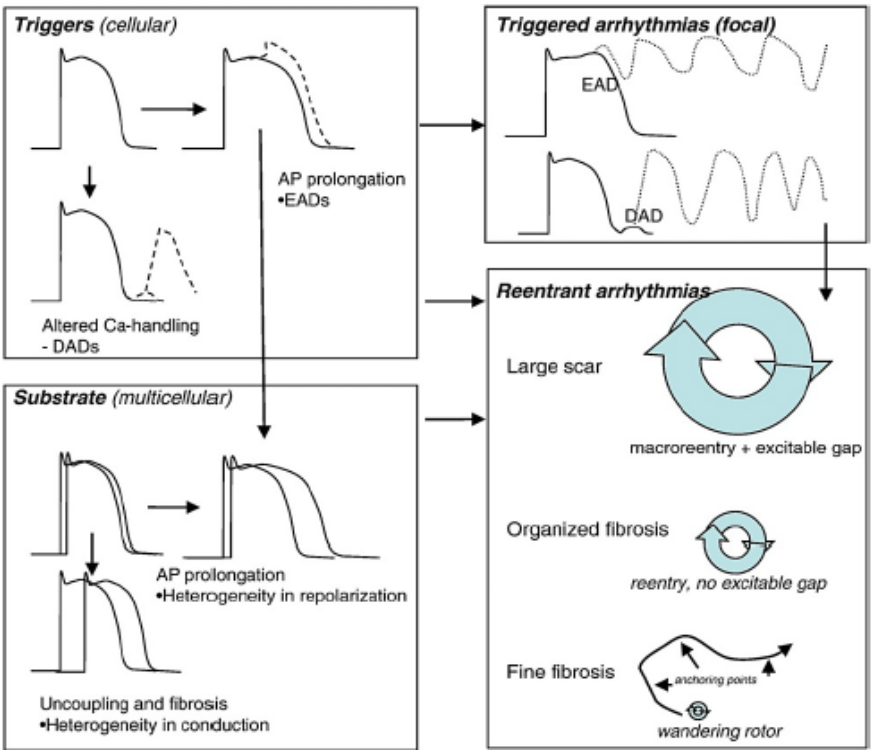

Figure 5. Diagram Demonstrating The Interaction Between The Cellular And Multicellular Arrhythmogenic Mechanisms Resulting In Arrhythmogenesis In Heart Failure[6]
AP Action Potential, EAD Early After Depolarization, EAD, DAD Delayed After Depolarization

4. Conclusion

Because of developments in medicine and research, the kind of patient we describe here is more reflective of the general public. The prevalence of congestive heart failure and related complications is growing as the average human lifespan increases. This population has an increased risk of sudden cardiac mortality due to underlying structural and electrophysiologic abnormalities, metabolic abnormalities, and the use of many medicines. Due to the effectiveness of preventative ICD implantation in lowering mortality in patients with moderate to severe cardiomyopathy, the number of patients presenting with these devices has grown significantly over the last several years. Antidysrhythmic medication is used in conjunction with other treatments by many of these individuals. In certain people, these medications might trigger polyrhythmias, necessitating more frequent ICD treatment cycles. Therefore, it is crucial to monitor their renal function, electrolyte balance, and medication intake. Chronically keeping blood potassium levels at or above 4.0 mEq/L may reduce the occurrence of ventricular dysrhythmias, especially torsades de pointes. However, cardiac and renal failure, along with other systemic disorders and pharmaceutical alterations, may lead to an increase in the number of individuals reporting VA to doctors. [9]

Careful medication reconciliation is critical for avoiding the potentially fatal cardiovascular effects of severe hypokalemia. Patients with CHF are more likely to have life-threatening hypokalemia and ventricular arrhythmias. ICDs are essential for individuals with CHF because they may avoid abrupt cardiac death from ventricular arrhythmia.[4]

Neurohormonal signaling modifications, structural remodeling, and electrophysiology all contribute to the phenotypic presentation of ventricular tachycardia in HF. New technologies may help us better understand the biology of heart failure and its role as a trigger for ventricular tachycardia by enabling us to target and change some of the processes that contribute to the formation of the arrhythmogenic substrate. [4]

5. Declaration

5.1 Ethics Approval and Consent to participate
Not applicable.

5.2. Consent for publication
Not applicable.

5.3 Availibility of data and materials
Data used in our study were presented in the main text.

5.4 Competing interests
Not applicable.

5.5 Funding Source
Not applicable.

5.6 Authors contributions
Idea/concept: ICR. Design: ICR. Control/supervision: AR, MSR, IP. Data collection/processing: ICR. Analysis/interpretation: ICR. Literature review: ICR. Writing the article: ICR. Critical review: AR, MSR, IP. All authors have critically reviewed and approved the final draft and are possible for the content and similarity index of the manuscript.

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