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Ventricular Tachycardia with ST Elevation in Lead aVR Associated with Normal Coronary Arteries: A Case Report and Review of Literature

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Abstract As per the 2013 guidelines of ACCF/AHA (American College of Cardiology Foundation/ American Heart Association), ST Elevation (STE) in lead augmented vector right (aVR), along with ST depression in multiple leads, is associated with critical stenosis of left main coronary artery (LMCA), left anterior descending artery (LAD) or a triple vessel disease (TVD). Early identification of ST-Elevation Myocardial Infarction (STEMI) is important as timely reperfusion with intervention can save myocardium and improve survival. We present a case of a 70 years old female, with cardiovascular risk factors, who presented to the emergency department with chest pain decompensating with ventricular tachycardia. On cardioversion, she was found to have ST elevation in aVR with ST depression in V4-V6, I, II, and aVL. However subsequent echocardiogram and coronary angiogram showed normal coronary arteries and left ventricular function.

Keywords: *coronary angiography, Augmented vector right (aVR), Electrocardiogram (ECG) criteria, ST elevation myocardial infarction (STEMI), left anterior descending artery (LAD), left main coronary artery (LMCA)*

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1. Introduction

An acute STEMI occurs when transmural myocardial ischemia results in myocardial injury or necrosis resulting in abnormal cardiac biomarkers [1,2]. As per the 2013 guidelines of ACCF/AHA (American College of Cardiology Foundation/ American Heart Association), STE in lead augmented vector right (aVR), along with ST depression in multiple leads, is associated with critical stenosis of left main coronary artery (LMCA), left anterior descending artery (LAD) or a triple vessel disease (TVD) [3]. The importance of Electrocardiogram (ECG) changes in predicting the critical occlusion of LMCA and LAD have been well proved and is found to be a strong predictor of 30-days mortality [4,5]. We are writing this unique case report of an elderly patient who was found to have STE in aVR on ECG with chest pain and ventricular tachycardia. However, the discovery of normal coronary arteries with no occlusion on angiography was an interesting finding emphasizing the fact that not all EKG changes correlate with expected myocardial injury and damage.

2. Case Report

A 70 years old female with a past medical history of obesity, hypertension and dyslipidemia presented with sudden onset 10/10 non-radiating that started suddenly while walking. She was alert, awake, and orientated on arrival at the emergency room. She was febrile, tachycardia, and blood pressure was 80/60 mm Hg. Electrocardiogram revealed ventricular tachycardia (Image 1) and in setting hypotension patient synchronized

cardioversion with 200 joules was performed (Image 2) which revealed Sinus rhythm with ST-segment elevation in leads aVR, V1-V2 and ST-segment depression in leads V4-V6, I, II and aVL. An emergent coronary angiogram was performed due to concerns of underlying left main stenosis and/or triple vessel coronary artery disease which revealed normal coronary angiogram. Investigations revealed hemoglobin of 13.7g/dL, white cell count of 4.59M/uL, and platelet count of 214K/uL, (all within normal limit). The patient did not have any electrolyte abnormality with potassium 4.2mmol/L and magnesium level at 2.1 mg/dL, eGFR was also normal. Troponin-I levels at presentation were 0.031 ng/ml and subsequently peaked at 1.89 six hours later. Transthoracic echocardiography showed a left ventricle ejection fraction of 55% without wall motion abnormality. Cardiac magnetic resonance imaging obtained to rule out infiltrative disease ruled out the same. The patient underwent cardiac electrophysiology study and successful ventricular tachycardia ablation.

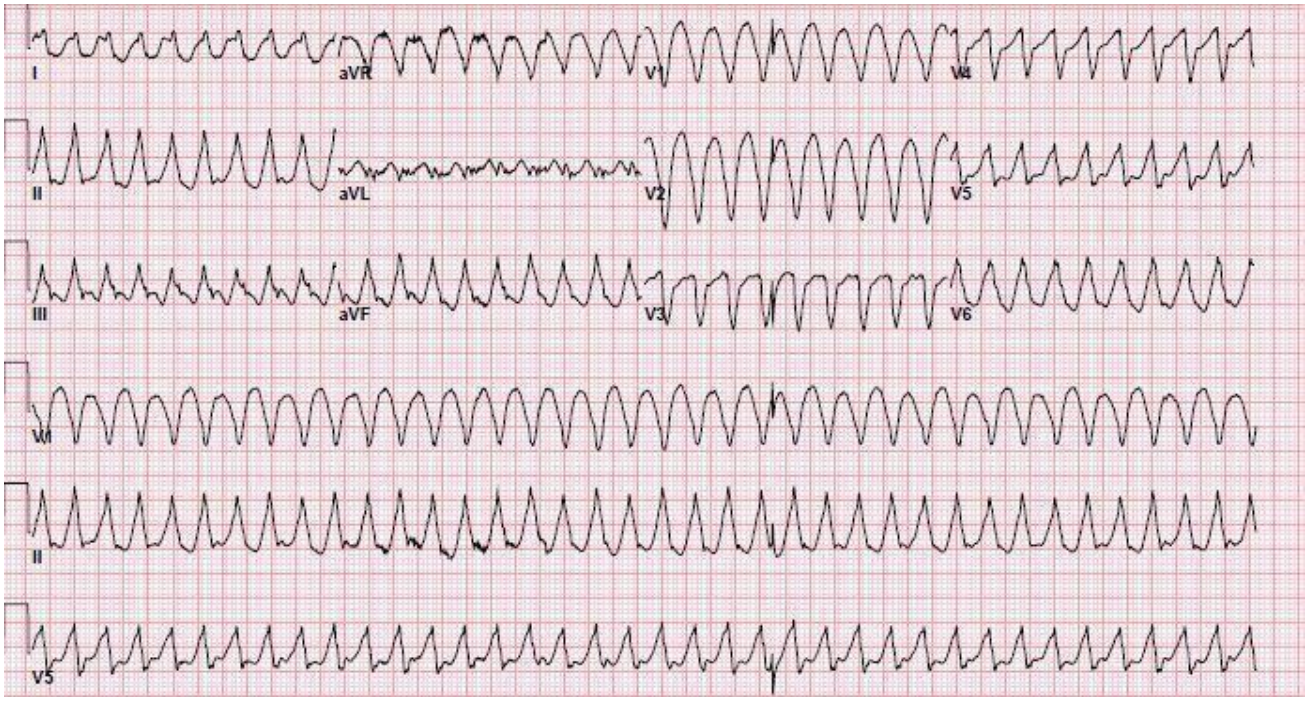

Image 1. Electrocardiogram showing ventricular tachycardia at presentation

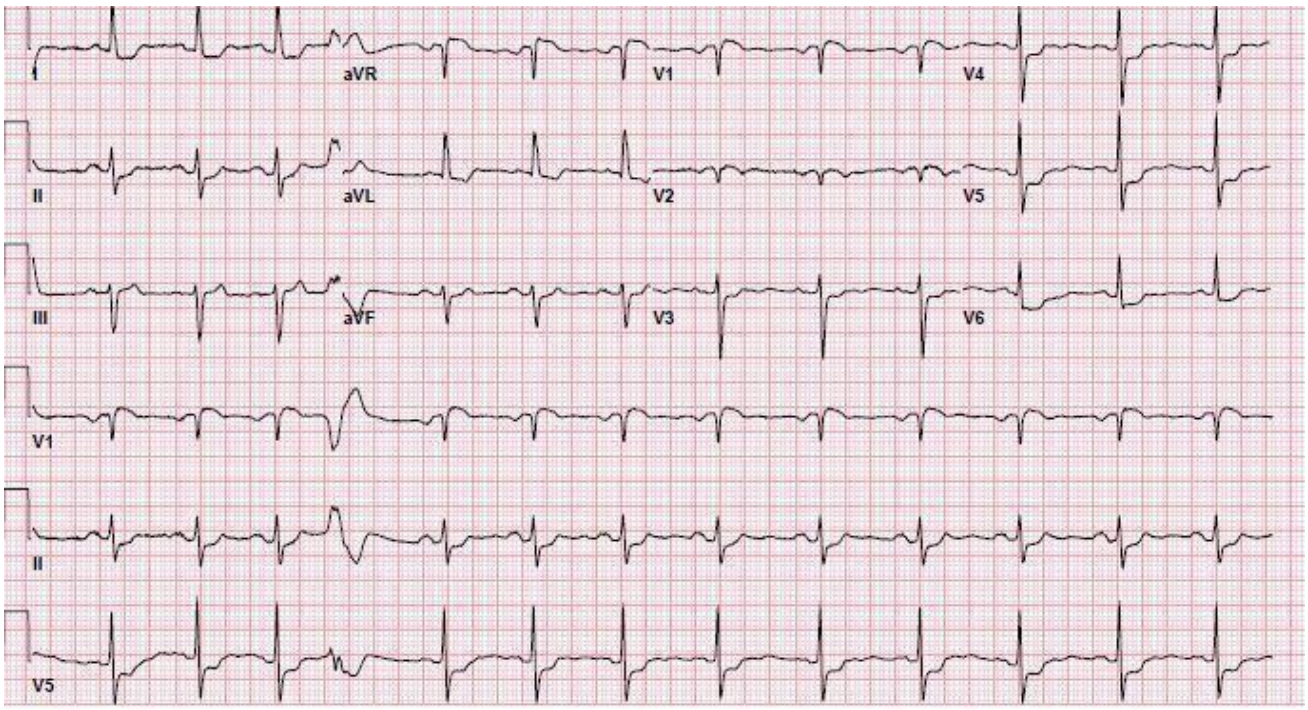

Image 2. Electrocardiogram showing normal sinus rhythm with ST-segment elevation in leads aVR, V1-V2, and ST-segment depression in leads V4-V6, I, aVL, and II

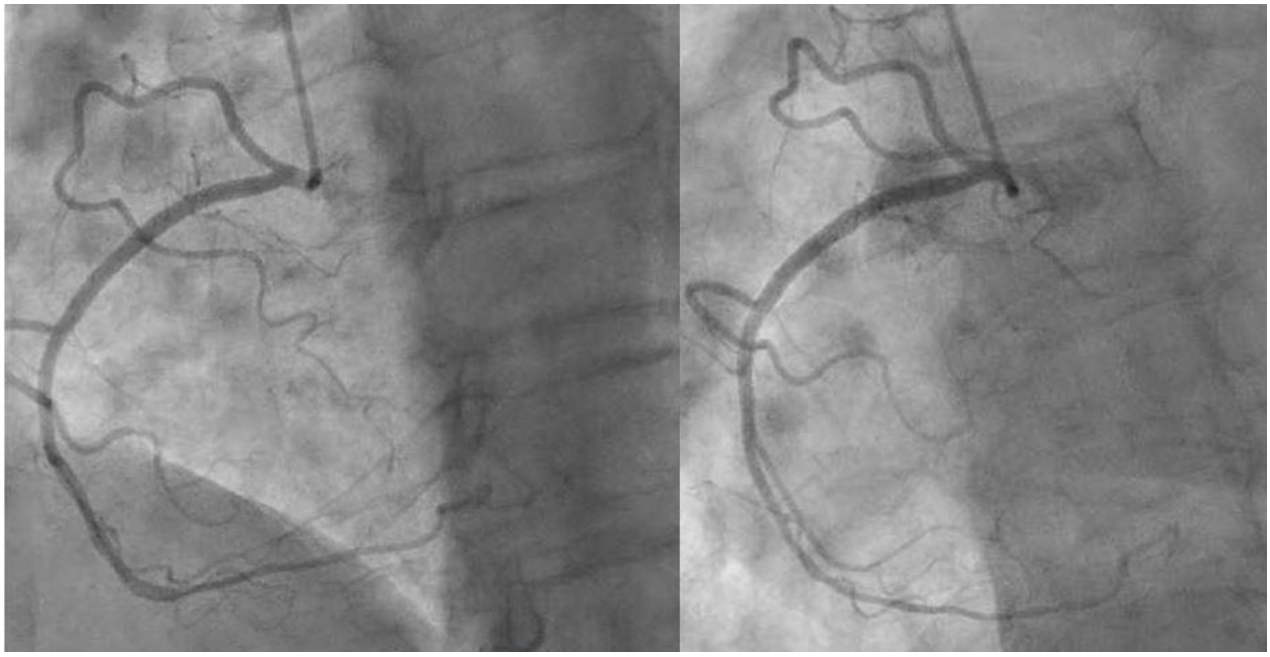

Image 3. Normal coronary angiogram of right coronary artery

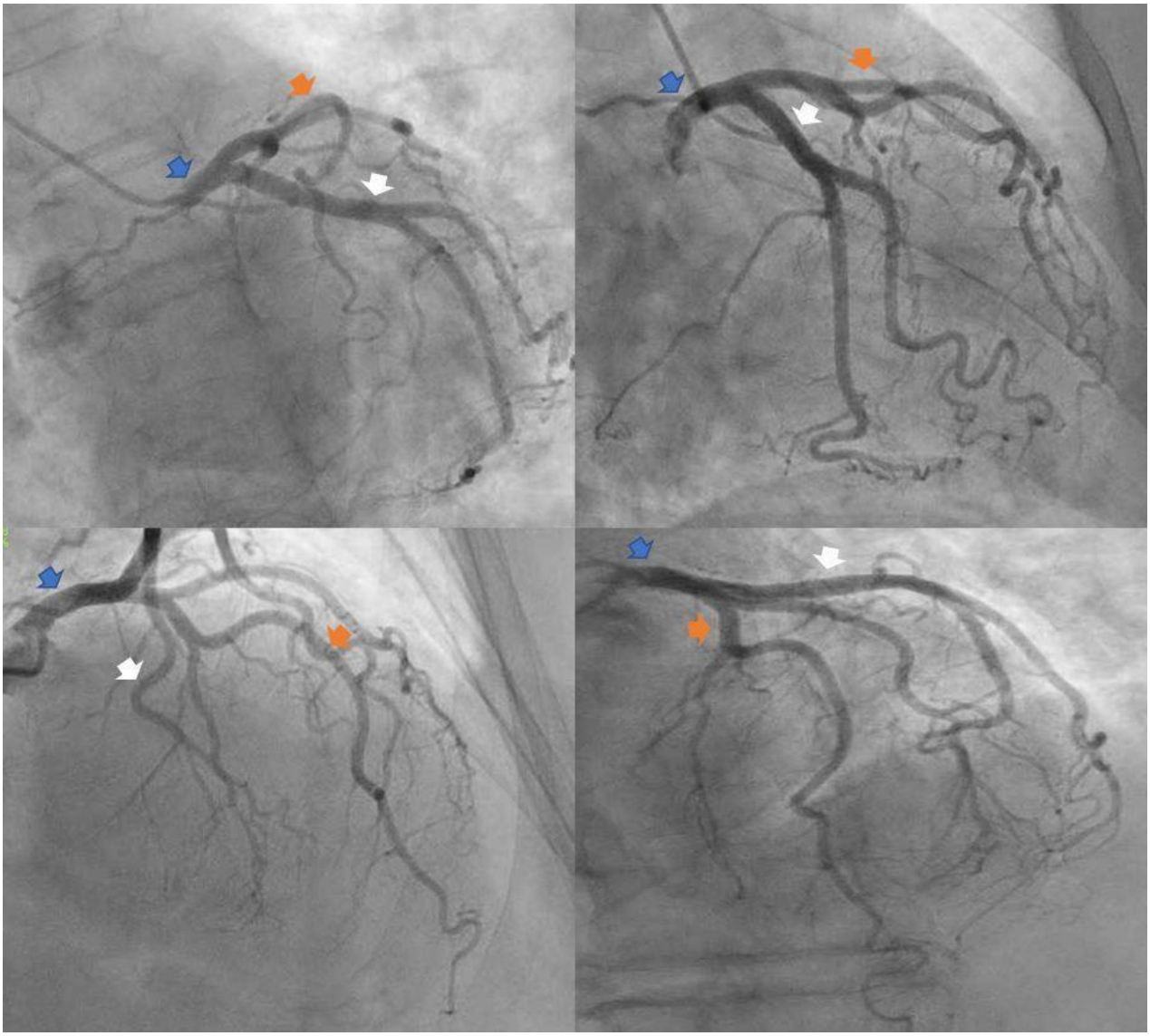

Image 4. Normal coronary angiogram of the left coronary artery. Blue arrow indicating left main artery, a saffron arrow indicating the left anterior descending artery, and a white arrow indicating left circumflex artery

3. Discussion

The lead aVR is an augmented vector right, placed on the right shoulder, providing information from the right upper portion of the heart including the right ventricular outflow tract and the basal portion of the interventricular septum [6]. ST Elevation in lead aVR with ST-segment depression in lead V5, and ST-segment elevation in V1>2.5 mm predicts left main coronary artery (LMCA) disease and the proximal branch of LAD [7]. Several other clinical conditions are associated with STE in aVR such as aortic dissection involving left main coronary artery, acute pulmonary embolism, critical aortic stenosis, Brugada syndrome, left ventricular hypertrophy with repolarization abnormality, tricyclic antidepressant toxicity, tachyarrhythmias including Wolff Parkinson White (WPW) syndrome [8]. However, since lead aVR is electrically opposite to leads I, II, aVL, and V3-V6 hence an ST depression in the above-mentioned leads can produce reciprocal STE in aVR [9].

The underlying pathophysiology of STE in aVR can be explained by either diffuse anterolateral subendocardial ischemia with a reciprocal change in aVR or transmural infarction of the basal portion of the heart [9,10]. Tachyarrhythmias by itself can cause elevated troponin [11]. Several conditions other than myocardial infarction can have elevated troponin without significant coronary artery diseases such as congestive heart disease, sepsis, pulmonary embolism, and thoracic injuries [12]. In our patient, no evidence of coronary artery disease can well be attributed to either endothelial dysfunction and demand ischemia or global ischemia in small intracardiac vessels that cannot be evaluated by angiography [13].

As per a recent meta-analysis done by Lee et al, the STE in aVR and the degree of STE are independent predictors in diagnosing LMD or Myocardial infarction [8]. Recently a new term INOCA (Ischemia with no obstructive coronary artery disease) has come into play defined as patients with chest pain, evidence of ischemia but no obstructive CAD at coronary angiography [14]. Limited coronary flow reserve (CFR) or objective myocardial ischemia consistent with coronary microvascular dysfunction (CMD) could be the probable underlying etiologies [14]. STE in aVR coupled with ST depression in other leads represents a critical clinical condition suggestive of severe LMAD or proximal LAD disease [10]. Nevertheless, the prognostic value of STE-aVR in acute coronary syndrome is still debatable. Szymański et al study claimed STE in aVR as a strong and independent predictor of 30-day mortality in Non-STE myocardial infarction (NSTEMI) [5]. GRACE investigators did not consider it as an independent predictor of in-hospital or 6-month mortality [15]. INOCA group of patients are a heterogeneous population who are stable concerning coronary circulation but carry an elevated major adverse cardiovascular events (MACE) including death, nonfatal MI, nonfatal stroke, and hospitalization for heart failure or angina [14].

As far as treatment modalities are concerned medical management effective for ischemic heart diseases such as angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker, β-blocker, calcium channel blocker, or statin therapies are used [16]. Evidence-based primary and secondary treatment guidelines are needed to address the importance of the clinical burden of these myocardial diseases. The diagnostic and therapeutic uncertainty in such patients with documented atherosclerotic and ischemic burden poses a great risk for the cardiovascular outcome.

4. Conclusion

STE in aVR in the setting of the acute coronary syndrome (ACS) is often associated with severe disease of the LMAD or proximal LAD artery disease and clinicians are well aware of this fact. However, based on our case report, our patient did not have any signs of coronary artery disease as evidenced by the normal angiographic study. When the signs of myocardial ischemia are present and there is a distinct absence of obstructive coronary artery disease, physicians must be able to derive an alternative cause for such a finding based on the available clinical signs and symptoms. In this case report we can infer that the cause of STE in the aVR leads were most likely due to the direct effects of ventricular tachycardia causing demand related ischemia of the myocardium.

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