Pharmacological Approach for Symptomatic Nonsustained Ventricular Tachycardia

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ABSTRACT
Nonsustained ventricular tachycardia (NSVT) is a common arrhythmia associated with heart failure, cardiomyopathy, coronary artery disease, electrolyte imbalances, and congenital heart disorders (Foth et al., 2023). NSVT is often asymptomatic depending on its burden percentage. However, typical NSVT presentation in the emergency department (ED) includes palpitations, near-syncope, dizziness, skipped beats, chest pain, and/or dyspnea (Katritsis et al., 2012). In some instances, NSVT can present with elevated or slightly elevated troponin from demand ischemia. A definite diagnosis of NSVT is not of high complexity; nevertheless, it is not always identified on electrocardiogram (ECG) by the time the patient arrives to the ED. Identification of NSVT usually requires prolonged cardiac monitoring, mobile cardiac telemetry (MCT), and in some instances internal loop recorder placement. The purpose of this case is to discuss the typical presentation and pharmacological approach of patients with stable NSVT. Key words: antiarrhythmics, mobile telemetry, nonsustained ventricular tachycardia, palpitations, Vaughan-Williams classifications, ventricular arrhythmia

A 72-YEAR-OLD Hispanic man with a history of coronary artery disease (CAD), heart failure with preserved ejection fraction, hypertension, aortic stenosis, hypothyroidism, and benign prostatic hyperplasia presents to the emergency department (ED) complaining of intermittent episodes of palpitations, and mild chest discomfort for the past 2 hr. He was previously seen by cardiology due to dizziness. He had a 14-day mobile cardiac telemetry (MCT) monitor attached to his chest and was on day 4. He was planning on going to the cardiac center the following morning, but his symptoms were becoming too bothersome. He admitted for mild shortness of breath and dyspnea with exertion. There were no reports of near-syncope, fatigue, orthopnea, edema, or paroxysmal nocturnal dyspnea. No recent medications changes,
alcohol, drug, or tobacco use. The patient denied any other symptoms and his physical examination was unremarkable.

**VITAL SIGNS**

- Blood pressure 142/72 mmHg
- Heart rate 88 beats/min (bpm)
- Oxygen saturation 98%
- Respiratory rate 18

**MEDICATION RECONCILIATION LIST**

- Metoprolol succinate 100 mg daily
- Furosemide 20 mg daily
- Sacubitril/valsartan 24–26 mg twice a day
- Finasteride 5 mg daily
- Tamsulosin 0.4 mg daily
- Aspirin 81 mg daily
- Levothyroxine 100 mcg daily

The initial provider orders included complete blood count (CBC) with differential, comprehensive metabolic panel (CMP), troponin I, chest x-ray, 12-lead electrocardiogram (ECG), and continuous cardiac monitoring. The differential diagnosis included myocardial infarction, atrial fibrillation with rapid ventricular response, ventricular tachyarrhythmias, ectopy, supraventricular tachycardia (SVT), electrolyte imbalance, and anxiety. The patient had a total of 4 points on the history, electrocardiogram, age, risk factors, and initial troponin (HEART) score placing him at intermediate risk. According to Backus et al. (2013), a HEART score of 4–6 has a 16.6% risk of major adverse cardiac event (MACE). The thrombolysis in myocardial infarction (TIMI) score had a total of 2 points placing him at 8% risk for new or recurrent myocardial infarction. The TIMI scale is useful non-ST-elevation myocardial infarction (NSTEMI) risk stratification score for patients with high-risk mortality at 14 days. However, a recent study found that the HEART score was more effective than the TIMI risk score in predicting MACE at 30 days in Hispanic patients (Torralba et al., 2020). The patient also had a pulmonary embolism rule-out criteria (PERC) score of 1 given his age; nonetheless, he had no significant clinical signs and symptoms of an acute pulmonary embolism with a very low pretest probability (based on provider clinical judgment) requiring immediate imaging. Holding off on a chest computer tomography angiogram was deemed appropriate.

**DIAGNOSTIC RESULTS**

- CBC, CMP: unremarkable
- First troponin I: 0.003 ng/ml (normal)
- Second troponin I: 0.003 ng/ml (normal)
- Chest X-ray: mild cardiomegaly
- 12-lead ECG: Abnormal (see Figure 1)

Cardiology consultation was advisable despite a mostly normal evaluation with no acute findings. The patient had a live MCT monitor that would help to identify any arrhythmia prior to ED arrival. The live MCT monitor identified the following: sinus rhythm 64–120 bpm (average rate of 92 bpm); 45 episodes of atrial tachycardia (fastest 160 bpm); and 220 episodes of monomorphic ventricular tachycardia (longest 22 beats; fastest 197 bpm). The nonsustained ventricular tachycardia (NSVT) identified on MCT can be appreciated on Figure 2. The cardiologist recommended a transthoracic echocardiogram (TTE), magnesium level, and sotalol 80 mg in hospital with cardiac monitor. It is worth noting that the patient had a coronary angiogram 2 years prior identifying nonobstructive CAD (left anterior descending [LAD] 40% stenosis; left circumflex [LCx] 30% stenosis).

**TRANSTHORACIC ECHOCARDIOGRAM RESULTS**

- **TTE**: Ejection fraction 50%–55% with no wall motion abnormality; mild concentric left ventricular hypertrophy; right ventricular systolic pressure of 23 mmHg; moderate aortic stenosis with a pressure gradient 36.97/16.23 mmHg; and an aortic valve area of 1.8 cm².
VENTRICULAR TACHYCARDIA

Ventricular tachycardia (VT) is a potential and common life-threatening arrhythmia resulting in cardiac death in the United States (Foth et al., 2023). The American Heart Association (AHA, 2020) has defined VT as a cardiac arrhythmia of more than three consecutive complexes that originate in the ventricle at a rate greater than more than 100 bpm. The different types of VT include sustained VT (≥30 s) requiring termination due to hemodynamic compromise, and nonsustained VT (more than three beats) with spontaneous termination. Monomorphic VT is a
stable single QRS morphology with each ventricular beat. Polymorphic or bidirectional VT has alterations in the QRS frontal plane axis, and it is often seen in the setting of digitalis toxicity or catecholaminergic VT (Al-Khattib et al., 2017).

According to Havakuk et al. (2020), characteristics and presentation for unstable VT are palpitations, chest pain, chest discomfort, dyspnea, dizziness, syncope, low oxygen saturation, and abnormal vital signs. Fluctuations in blood pressures may arise due to variability in the degree of left ventricle filling, stroke volume, and cardiac output. Variability in heart sounds may be present, resulting in the intensity of heart sound S1. Cannon “A” waves (intermittent and irregular jugular venous pulsations of great amplitude) can be present during certain tachyarrhythmias including VT. This is the result of contraction of right atrium against closed tricuspid valve (Al-Khattib et al., 2017). These ECG findings can be appreciated in Figure 3.

VT is considered most common wide complex tachycardia with more than 120 bpm that originates in the distal bundle of his ventricular myocardium. A wide QRS interval of more than 120 ms was observed with no regularly occurring P wave to QRS association (Long & Koyfman, 2017). Polymorphic VT can result in a malignant tachyarrhythmia changing QRS pattern. Characteristics of polymorphic VT vary in the QRS complex shape with multiple morphologies. It can terminate spontaneously or progress to ventricular fibrillation (VF). Diagnosis is of utmost importance due to different forms of therapy for polymorphic VT (Rosso et al., 2021). Typical ECG findings of polymorphic VT can be appreciated in Figure 4. Monomorphic VT is characterized as a rapid heartbeat ranging from 150+ bpm with the same QRS morphology. It is also life-threatening and may lead to VF and cardiac arrest (Kanagasundram et al., 2019). Additionally, various tachyarrhythmias may elevate troponin I levels due to shortening of diastole causing subendocardial ischemia; however, the exact cause is not well understood (Kanjwal et al., 2008).

PHARMACOLOGICAL MANAGEMENT

The pharmacological management of VT varies based on symptoms and underlying causes. Some variables that must be considered when selecting appropriate antiarrhythmic drugs (AADs) include ejection...
fraction, obstructive CAD, electrolytes (i.e., potassium, magnesium, and calcium), renal/liver function, blood pressure, and resting heart rate. Low serum magnesium, calcium, and potassium levels should be corrected in all patients presenting with VT (Foth et al., 2023). The following AADs can be used for VT. The purpose of this pharmacological review is to provide basic insight into their clinical indications and appropriateness following the Vaughan-Williams classification. Table 1 provides a reference for the use of AAD based on VT acuity.

Class IA

Procainamide is an intravenous (IV) AAD typically used for acute unstable or sustained VT, atrial arrhythmias, Brugada syndrome, and Wolff-Parkinson-White syndrome. It should be noted that procainamide may cause heart failure exacerbation in patients with reduced ejection fraction (Al-Khatib et al., 2017). Mechanism of action (MOA): Class IA antiarrhythmics moderately bind to fast sodium channels preventing repolarization. They prolong the cardiac action potential, effective refractory period, and slow the speed of impulse conduction (Pritchard & Thompson, 2023). The use of procainamide for VT has declined over the years due to the development of newer and more effective AADs. However, according to a study by Ortiz et al. (2017), the PROCAMIO study suggests the use of procainamide for stable VT due to faster onset of VT resolution with less adverse effects compared with amiodarone.

Class IB

Lidocaine is an IV AAD typically used for acute episodes of unstable VT, VF, or cardiac arrest. Lidocaine has been included in the AHA (2020) cardiac arrest guideline diagram along with amiodarone. A recent study comparing the neurological outcomes and efficacy of amiodarone and lidocaine revealed that amiodarone was superior to lidocaine for patients in cardiac arrest; nonetheless, lidocaine is a relatively inexpensive drug with similar VT resolution (Wang et al., 2023). Mexiletine on the other hand is an oral AAD typically reserved for subacute cases of NSVT. Mexiletine is not a first-line AAD for NSVT but may be used for patients with a low resting heart rate unable to tolerate amiodarone, beta blockers, or flecainide (Al-Khatib et al., 2017). MOA: Class IB antiarrhythmics mildly bind to fast sodium channels during phase 0 of the cardiac action potential prolonging the effective refractory period. It has no significant effects on PR intervals and can slightly

Figure 4. Polymorphic ventricular tachycardia. Note. Author’s own work.
shorten the QTc (Singh et al., 2023). Therefore, mexiletine can be a good option for patients with NSVT and borderline bradycardia with or without reduced ejection fraction. Functional cardiac evaluation is recommended (e.g., stress testing, echocardiogram, and coronary computer tomography) before initiating flecainide. MOA: Class IC antiarrhythmics markedly bind to fast sodium channels leading to prolonged depolarization while increasing refractoriness (Arunachalam & Alzahrani, 2023).

**Class IC**

Flecainide is an oral AAD typically used for subacute NSVT, SVT, and high-burden premature ventricular contractions. Flecainide should not be used in patients with refractory VT. According to Al-Khatib et al. (2017), flecainide may induce monomorphic VT, Brugada syndrome, sinus node dysfunction, and atrial ventricular blocks in patients with obstructive CAD or heart failure with reduced ejection fraction. Functional cardiac evaluation is recommended (e.g., stress testing, echocardiogram, and coronary computer tomography) before initiating flecainide. MOA: Class IC antiarrhythmics markedly bind to fast sodium channels leading to prolonged depolarization while increasing refractoriness (Arunachalam & Alzahrani, 2023).

**Class II**

Selective and nonselective beta blockers (e.g., metoprolol, carvedilol, bisoprolol, and propranolol) are the most widely used AADs for VT, NSVT, SVT, atrial arrhythmias (atrial fibrillation/futter), and high-burden ectopy due to their significant efficacy and safety.
profile in patients with either preserved or reduced ejection fraction (Al-Khatib et al., 2017). The adrenergic effects of beta blockers have shown antiarrhythmic, antianginal, and reduced sudden cardiac death by approximately 35% (McMurray et al., 2012). Beta blockers can serve as first-line therapy for patients with NSVT with CAD and heart failure. A study by Kontos et al. (2011) advised to use beta blockers with caution in patients with acute coronary syndrome with two or more shock risk factors: older than 70 years, systolic blood pressure less than 120, symptomatic ST-elevation myocardial infarction (STEMI) for more than 12 hr. MOA: Beta blockers have chronotropic, inotropic, adrenergic, and renin effects that vary based on selectivity and specificity of receptors. Beta-1 selective blockers have potent antiarrhythmic properties and can decrease myocardial oxygen demand and heart rate. Nonselective beta blockers also have antiarrhythmic effects and decrease myocardial oxygen demand but may have a more potent outcome on blood pressure by attaching to both beta and alpha receptors (Khashayar & Arif, 2023).

Class III
Amiodarone is both an oral and IV potassium channel blocker AAD that is widely used for sustained VT and high-burden NSVT. Amiodarone (IV) is a widely studied AAD that is supported by the 2020 AHA cardiac arrest guideline diagram due to its significant efficacy in the management of VT. Recent studies have shown significant reduction in sudden cardiac death utilizing amiodarone in patients with acute coronary syndrome with or without reduced ejection fraction. Oral amiodarone is typically reserved for subacute cases of NSVT (asymptomatic, low-to-intermediate burden NSVT, and stable vital signs). Several studies have shown that the long-term survival effects of amiodarone are controversial (Al-Khatib et al., 2017). Additionally, long-term use of amiodarone may lead to thyroiditis, optic neuritis, and pulmonary toxicity. MOA: Potassium ions are responsible for repolarizing the heart during phase 3 of the cardiac action potential. Amiodarone prolongs the cardiac action potential and effective refractory period by reducing the excitability of the myocytes (Florek et al., 2023).

Sotalol is an oral potassium channel blocker AAD that is not uncommonly used for subacute NSVT. According to Al-Khatib et al. (2017), sotalol has suitable efficacy in suppressing ventricular arrhythmias (VT/VF), but it must be used with caution due to its significant proarrhythmic effects. Sotalol should be started in hospital settings under cardiac monitoring. Sotalol may lead to heart failure exacerbation and should be avoided in patients with reduced ejection fraction. MOA: Sotalol has nonselective beta-blocker properties at lower doses, but it is used to prolong the cardiac action potential through its effects on potassium channels (DeMarco et al., 2021).

Class IV
Non-dihydropyridine (DHP) calcium channel blockers (CCBs) do not usually have a role in the management of ventricular arrhythmias. It has been reported that IV non-DHP CCBs should not be given during unstable sustained VT or in patients with acute heart failure. Non-DHP CCBs can suppress the cardiac outflow tract and lead to hemodynamic collapse. Nevertheless, oral non-DHP CCBs (i.e., verapamil and diltiazem) can help treat low-burden CCB-sensitive NSVT or bundle branch reentrant VT that failed treatment to conventional therapies (Al-Khatib et al., 2017).

DISCUSSION
The ED medical management of this patient was clinically appropriate given the circumstances, availability of records, and outside cardiac monitor tracings. The patient was admitted to the hospital telemetry floor for observation and discharged home within 48 hr. Under different circumstances the patient may have been discharged home.
with urgent cardiology follow-up. The patient did not show any evidence of NSVT while in the ED. The NSVT tracings were obtained by the cardiologist from the live MCT monitor, which would have otherwise not been available to the emergency provider. The use of sotalol was found to be clinically appropriate for this patient. The patient had stable and subacute NSVT with known preserved ejection fraction and absence of obstructive CAD. Oral amiodarone or flecainide would have also been clinically appropriate in this situation. The patient was discharged home with sotalol 80 mg twice a day with a 2-day cardiology follow-up for further evaluation and 12-lead-ECG.

CONCLUSION

Paroxysmal and NSVT should always be considered as part of the differential diagnoses for patients presenting with palpitations, dizziness, chest pain, dyspnea, or near syncope. A definite diagnosis of VT or NSVT is not of high complexity; however, it may require prolonged cardiac monitoring. Emergency providers must be aware that low-burden NSVT can be missed during short emergency admissions. The role of emergency providers is to suspect NSVT based on cardiovascular risk factors and consult with cardiology when clinically appropriate. The pharmacological management of NSVT varies based on patient symptoms and underlying causes. Providers must first determine whether the patient has acute or subacute NSVT. Variables that must be considered when selecting AADs include electrolytes (potassium, calcium, and magnesium), heart rate, blood pressure, ejection fraction, and CAD. Commonly used AADs for subacute NSVT include oral amiodarone, sotalol, metoprolol, and flecainide. Lastly, AAD risks versus benefits must be noted when selecting an appropriate AAD given their proarrhythmic properties. Cardiology consultation is highly advisable for all oral AAD therapy.

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