NON-SUSTAINED, SYMPTOMATIC VENTRICULAR TACHYCARDIA: A CASE REPORT AND LITERATURES REVIEW

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Abstract

Non-sustained ventricular tachycardia (NSVT) is commonly seen but poorly understood. Patients with NSVT might be asymptomatic or encountered sudden cardiac death. The prognosis depends on what the concomitant heart disease is. Poor prognosis were found in patient with NSVT during or post exercise; acute myocardial infarction more than 24 hours; chronic ischemic heart disease with left ventricular ejection fraction (LVEF) below 40% with/without inducible sustained ventricular tachycardia in electrophysiology testing; and hypertrophic obstructive cardiomyopathy in the young. Medical therapy including beta-blockers and class III antiarrhythmic agents, while implantable cardiac defibrillators (ICD) and radiofrequency ablation are indicated in certain patients. Here, we report a case that had near syncope and palpitation with NSVT on 24-hours electrocardiography, finally received ablation and antiarrhythmic therapy. No more arrhythmia was found during out patient department follow up.

Key Words: Non-sustained ventricular tachycardia (NSVT), Left ventricular ejection fraction (LVEF), Right ventricular outflow tract (RVOT), Implantable cardioverter defibrillator (ICD)

Introduction

NSVT is usually a benign cause, but it may provoke malignant arrhythmia and then induce sudden cardiac death. The symptoms of NSVT vary from asymptomatic to heart failure. This report describes a 71-year-old woman with NSVT. The symptoms were stabilized after antiarrhythmic agents and electrophysiology study plus ablation.

Case Report

A 71-year-old woman presented with a 2-weeks history of epigastric pain, chest tightness, shortness of breath, near syncope, and palpitation. The electrocardiography (Fig. 1) revealed sinus tachycardia and complete right bundle branch block. Initial vital signs showed that the blood pressure was 202/73 mmHg, the pulse rate was 105 beats per minute, the respiratory rate was 18 breaths per minute and the body temperature was 37.2°C. Physical examination revealed mild pale conjunctivae, no jugular vein engorgement, rapidly regular heart beat, no heart murmur, clear breath sound, no legs edema. Laboratory data showed that microcytic anemia, normal cardiac
enzymes (Hgb: 10.4 gm/dl, Hct.: 30.5%, MCV: 78.2 fl, CK: 35 U/L, CK-MB: 13.4 U/L). Traced her past history, she has had hypertension with medical control (unknown drugs) for 4 years. She also received internal fixation for left femoral fracture 2 years ago. Under the tentative diagnosis of hypertensive emergency, conduction disorder, anemia, peptic ulcer, she was admitted for further management. The upper gastrointestinal panendoscopy revealed gastric ulcer, duodenal ulcers, and gastroesophageal reflux disease. The 24-hours electrocardiography showed 39 episodes of frequent NSVT (the longest duration was 4 seconds), occasional premature ventricular contractions, and no ischemic change (Fig. 2). The echocardiogram disclosed no hypertrophic or dilated cardiomyopathy and normal left ventricular systolic function. There is no prominent hemodynamic compromise.

Fig. 1. Electrocardiography revealed sinus tachycardia and complete right bundle branch block.

Fig. 2. One episode of NSVT during 24-hours electrocardiography.
and syncope, so the patient was monitored at intensive care unit without anti-arrhythmic medication initially. The magnetic resonance imaging of chest (Fig. 5) disclosed no evidence of arrhythmogenic right ventricular dysplasia (ARVD). The coronary angiography disclosed insignificant coronary artery disease (Fig. 3) and the electrophysiology study disclosed right ventricular outflow tract ventricular tachycardia. Radiofrequency catheter ablation with anatomically guided linear ablation of the anterior septum and posterior septum little moving to free wall was performed with a 4-mm-tip ablation catheter (Safire, St. Jude Medical, Minnetonka, MN, USA) with the temperature preset at 60°C and radiofrequency pulse duration of 120 seconds (Fig. 4). After discharged, the oral medications included amiodarone 200mg po daily after intravenous loading dose, plus atenolol 25mg po daily and adalat OROS 30mg po daily. Another 24-hour electrocardiographic recording that revealed no more ventricular tachycardia at outpatient department two months later.

Discussion

Definition of NSVT

NSVT is defined as three or more consecutive premature ventricular contractions, which may be monomorphic or polymorphic, occurred with rate above 120 beats per minute, lasting less than 30 seconds.

Etiology of NSVT

The mechanisms of NSVT included: (1) increased automaticity, (2) reentry, and (3) triggered automaticity. Increased automaticity (such as right ventricular outflow tract ventricular tachycardia) is often associated with autonomic tone, ischemia, reperfusion, acidosis, hypokalemia, hypomagnesemia, or cardiotoxic factors. It may not have sudden onset or termination. Reentry is usually scar related. NSVT occur after acute myocardial infarction is believed to result from the formation of scar tissue. Monomorphic ventricular tachycardia typically occurs due to reentry or triggered automaticity, while polymorphic ventricular tachycardia is caused by increased automaticity. The most common form of right ventricular outflow tract (RVOT) ventricular tachycardia is related to triggered automaticity arising from delayed after-depolarizations. It is thought to be dependent on intracellular calcium overload and cyclic adenosine monophosphate. Other causes of triggered automaticity are described in Table 1.
Non-sustained, symptomatic ventricular tachycardia

Taiwan Crit. Care Med. 2011; 12: 182-192

Prevalence of NSVT

Using 24 to 48 hours electrocardiographic recording, the prevalence in people without symptoms or history of cardiac disease ranged from 0 to 3%.\(^7\)\(^-\)\(^11\) approximately 20% of patients with hypertrophic cardiomyopathy,\(^12\)\(^,\)\(^13\) 40 to 50% in patients with dilated cardiomyopathy.\(^14\)\(^-\)\(^17\) The prevalence of NSVT in patients with coronary artery disease depend on the stage of disease. Within the first 24 hours of acute MI, the prevalence ranged from 45 to 75%.\(^18\)\(^-\)\(^20\) and declined as time went on after the event. The SPRINT study showed the incidence of NSVT 48 hours post MI was 3%.\(^21\) In the 24-48 hours post infarction, the NSVT is usually due to automaticity or triggered activity; however NSVT is often due to reentry while more than 48 hours post infarction.\(^20\)\(^,\)\(^21\) The symptoms of NSVT include palpitation, chest distress, pre-syncope, syncope, and heart failure. It is usually asymptomatic but a risk factor for sustained ventricular tachycardia, ventricular fibrillation, and sudden cardiac death, especially in patients with high frequency or left ventricular systolic dysfunction. NSVT also may lead to tachycardia-induced cardiomyopathy,
which is reversible. Ventricular tachycardia is the most common manifestation of arrhythmogenic right ventricular dysplasia that is a familial disease causing sudden cardiac death. Idiopathic right ventricular tachycardia commonly presents as non-sustained or repetitive monomorphic ventricular tachycardia. Ventricular tachycardia arising from the RVOT in the absence of structural heart disease is common, accounting up to 10% of all ventricular tachycardias. The symptoms of idiopathic RVOT ventricular tachycardia occurred typically between the age of 20 and 40 years, more commonly in women. Idiopathic right ventricular tachycardia that is most often arising from the septal region of the right ventricular outflow tract is a benign course.

### Diagnosis of NSVT

Diagnosis of NSVT is typically based on the finding of ambulatory 24-hour Holter electrocardiography. It can establish the frequency, duration, and morphology of the NSVT. Echocardiography can provide the left ventricular function and identify other underlying cardiomyopathy. Patients with suspected coronary heart disease may require coronary angiography. Besides, the MUSTT study suggested inducibility of tachyarrhythmia-identified patients whose death was significantly more likely to be arrhythmic. The utility of electrophysiology study in risk stratification for arrhythmic death is influenced by the LVEF. Patients with LVEF between 30% and 40% were suggested for electrophysiology study. In ischemic heart disease with LVEF >40%, the role of electrophysiology study is not yet established. In patients with coronary artery disease, the two and five years rate of sudden death were 18 and 32 percent if not treated in NSVT and induced sustained monomorphic VT. Without induced sustained monomorphic VT, the two & five years rate of sudden death were 12 and 24 percent in patients with coronary artery disease and NSVT. The ACC/AHA/ESC guidelines suggested electrophysiology testing is reasonable for risk stratification in patients with remote myocardial infarction, NSVT, and LVEF equal to or less than 40% (Class IIa; Level of Evidence: B).

### Treatment of NSVT

The goals of treatment are prevention of malignant arrhythmia, sudden cardiac death and elimination of symptoms. Treatments included antiarrhythmic medications, implantable cardioverter defibrillator (ICD) and catheter ablation.

1. **Anti-arrhythmic medications**

   According to the CAST trial, class I antiarrhythmic drugs are not advised due to associated...
with increased mortality in patients with history of myocardial infarction. The safe & effective antiarrhythmic medications are commonly beta-blocker and amiodarone. Beta-blocker can reduce the mortality of patients with ischemic heart disease who both have LVEF below 40% and NSVT. Amiodarone is effective in suppressing ventricular arrhythmias and has a trend toward reduces mortality among those with nonischemic cardiomyopathy, but it cannot reduce the incidence of sudden death or prolong survival among patients with heart failure. Another study showed amiodarone is associated with a reduction in the incidence of sudden death in adult HCM patients (including young adults) with NSVT. The Canadian Myocardial Infarction Amiodarone Trial (CAMIAT) disclosed that amiodarone reduces the incidence of ventricular fibrillation or arrhythmic death among survivors of acute myocardial infarction with frequent VPCs or NSVT, but no significant reduction in overall mortality was found. Amiodarone in combination with beta blockers significantly reduced both arrhythmic and nonarrhythmic cardiac death.

2. Implantable cardioverter defibrillator (ICD)

ICD is first-line therapy in patients with ventricular tachycardia who also have structural heart disease with impaired LVEF. It is also superior to medical therapy in primary and secondary prevention of sudden cardiac arrest. In the patients who received ICD implantation as primary prevention of sudden cardiac death, 20% of them would experience at least one ventricular tachycardia episode within 3-5 years after ICD implantation. The MUSTT trial disclosed ICD reduced arrhythmic death in patients with asymptomatic NSVT, LVEF≤40%, and induced sustained ventricular tachycardia, comparing with no therapy or antiarrhythmic drugs. The MADIT I study proved the benefit of ICD for improved survival in patient with asymptomatic NSVT and symptomatic ischemic heart disease with LVEF<35%. According to the MUSTT and MADIT I results, patients with LVEF below 40%, NSVT, and inducible ventricular tachycardia in electrophysiology study should received ICD implantation. In patients without diminished ejection fraction, further studies will be needed to determine whether those patients would benefit from ICD therapy. The ACC/AHA/HRS 2008 guidelines suggested ICD therapy is indicated in patients with NSVT due to prior myocardial infarction, LVEF less than or equal to 40%, and inducible ventricular fibrillation or sustained ventricular tachycardia at electrophysiological study (Class I; Level of Evidence: B).

3. Catheter ablation

ICD can terminate ventricular tachycardia episodes but does not prevent them. Catheter ablation offers a cure for ventricular tachycardia, which is resistant to antiarrhythmic medications. It can also reduce ICD shock and reversing tachycardia-induced cardiomyopathy. Ablation was recommended in the ACC/AHA/ESC guidelines as useful therapy in patients who are otherwise at low risk for sudden cardiac death and have symptomatic nonsustained monomorphic VT that is drug resistant, who are drug intolerant or who do not wish long-term anti-arrhythmic drugs therapy(Class IIa; Level of Evidence: C). The success rate of catheter ablation for ventricular tachycardia ranges from 83%-100%, while recurrence rates from 0%-31% on a variable follow-up period. Ablation for RVOT ectopy was more likely to fail as opposed to RVOT ventricular tachycardia (sustained or non-sustained). The predictors of failed ablation included presence of QRS morphologic variation, wider mean QRS, and taller R wave amplitude in lead II. Ablation of ventricular tachycardia is often challenging and lower success rates than ablation of supraventricular tachycardias. The reasons included: (1) large infarct region. (2) Hemodynamically unstable and poorly tolerated. (3) Epicardial or intramural in location. (4) Several reentrant circuits. It is controversial that anti-arrhythmic medications needed after successful catheter ablation. Overall, catheter ablation of ventricular tachycardia in the setting of structural heart disease can only be considered an adjunctive therapy, which in general, will require ICD therapy.
**Prognosis of NSVT**

Spontaneous NSVT in apparently healthy individuals does not imply an adverse prognosis, but long-term follow up is advisable. NSVT in the presence of structural heart disease carries a more serious prognosis than NSVT in the absence of structural heart disease. It is adverse prognostic significance for NSVT attacks during or post exercise, in patient with acute myocardial infarction more than 24 hours, chronic ischemic heart disease with LVEF below 40%, and hypertrophic obstructive cardiomyopathy in the young.\(^{43,44}\) In patients with congestive heart failure, NSVT is an independent marker for increased overall mortality rate and sudden death.\(^{45}\) In adult patients with hypertrophic obstructive cardiomyopathy, NSVT is a sensitive marker for increased risk of sudden death.\(^{46}\) NSVT is an insensitive and nonspecific predictor of sudden death in idiopathic dilated cardiomyopathy patients.\(^{47}\) However, NSVT in patients with primary ventricular fibrillation, congenital long-QT, Brugada syndrome, arrhythmogenic right ventricular dysplasia, repaired congenital abnormalities, valvular heart disease, hypertension were unknown prognostic significance for sudden death\(^{49}\) (see Table 2). In post-myocardial infarction patients with LVEF less than 40%, the risk of sudden death is increased more than five-fold.\(^{48,49}\) The MUSTT study also disclosed patients with coronary artery disease, left ventricular dysfunction and asymptomatic NSVT in whom sustained ventricular tachycardia can be induced in electrophysiology testing have a higher risk of sudden death or cardiac arrest and higher overall mortality than similar patients without inducible sustained ventricular tachycardia.\(^{26}\)

**Conclusion**

NSVT is frequently encountered in clinical practice. It may increase the risk for malignant arrhythmia and sudden cardiac death. The prognosis depends on the etiologies. Clinical approach to the patient with NSVT should always be considered what the coexisted heart disease is. In the absence of heart disease, spontaneous NSVT does not carry any adverse prognostic significance. NSVT with adverse prognostic significance were found in patients with some conditions. Treatment to the NSVT included antiarrhythmic medications,

| Table 2. Prognostic significance of non-sustained ventricular tachycardia with different underlying heart condition.\(^{43-49}\) |
|---|---|
| **Adverse prognostic significance** | 1. NSVT during or post exercise  
2. Acute myocardial infarction more than 24 hours  
3. Chronic ischemic heart disease with LVEF below 40% with/without inducible sustained ventricular tachycardia in electrophysiology testing  
4. Hypertrophic obstructive cardiomyopathy in the young |
| **No Adverse prognostic significance** | Idiopathic dilated cardiomyopathy |
| **Unknown prognostic significance** | 1. Primary ventricular fibrillation  
2. Congenital long-QT  
3. Brugada syndrome  
4. Arrhythmogenic right ventricular dysplasia  
5. Repaired congenital abnormalities  
6. Valvular heart disease  
7. Hypertension |
ICD implantation and catheter ablation. Beta-blocker and amiodarone are safe and effective anti-arrhythmic medications. ICD implantation is considered as first line therapy to terminate ventricular tachycardia episode. In patients who have structural heart disease, catheter ablation is recommended as adjunctive therapy if frequent episodes of VT and ICD shocks. Anti-arrhythmic medications therapy after successful catheter ablation is controversial.

References

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非持續性具症状之心室心搏過速: 病例報告及文献回顧

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摘要

非持續性心室心搏過速臨床上很常見，但了解的人卻不多。具有非持續性心室心搏過速的病人輕微者可以無症狀，嚴重者卻有猝死的可能。預後跟合併的心臟疾病有關，不良的預後常見於運動誘發之非持續性心室心搏過速、急性心肌梗塞24小時過後、慢性缺血性心臟病及左心室射出分率小於40%合併有或無於心臟電生理檢查誘發出非持續性心室心搏過速、年輕人之肥厚性阻塞性心肌病變。藥物治療包含乙型交感神經阻斷劑及amiodarone，而植入式心臟整流去顫器及射頻燒灼術適用於某些病人。我們報告一個71歲女性合併有暈厥及心悸的症狀，24小時心電圖診斷非持續性心室心搏過速。經射頻燒灼術及藥物治療後病情獲得穩定控制。希望藉由這個病例讓大家更加認識這個疾病。

關鍵詞：非持續性心室心搏過速，左心室射出分率

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