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Nonsustained Ventricular Tachycardia After Acute Coronary Syndromes: Recognizing High-Risk Patients

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The Case

A 51-year-old woman with coronary artery disease, diabetes, and hypertension was admitted with a non-ST elevation myocardial infarction (NSTEMI) and underwent percutaneous coronary intervention (PCI). After uncomplicated placement of two stents, she was transferred to the telemetry unit. About 12 hours after the procedure, the patient's nurse noted a five-beat run of ventricular tachycardia (VT). The patient was asymptomatic and other vital signs were stable. The nurse called the responsible physician who responded that "this was expected" (given her underlying heart disease and perhaps the possibility of reperfusion arrhythmias). The patient had several more short runs of non-sustained ventricular tachycardia (NSVT). However, given the physician's initial response, the nurse did not notify the physician about these additional runs. About 2 hours after the initial run of VT, the patient experienced a cardiac arrest secondary to sustained VT. She underwent cardiopulmonary resuscitation including chest compressions and defibrillation. Though the patient survived the initial arrest, she remained hypotensive and unfortunately did not regain any meaningful neurological function. Care was ultimately withdrawn 4 days after the event.

The Commentary

Patients with acute coronary syndromes (ACS) are at increased risk for multiple heart rhythm disorders, including conduction disease, bradycardia, atrial arrhythmias, and ventricular arrhythmias. In the case described above, a patient who underwent percutaneous coronary intervention developed a brief run of non-sustained ventricular tachycardia (NSVT) 12 hours after revascularization. NSVT is defined as any ventricular tachycardia (VT) that does not lead to hemodynamic compromise and is less than 30 seconds in duration. Most episodes of NSVT, like the one described above, are just a few beats in duration. NSVT is very common following ACS, affecting more than 50% of patients.(1) NSVT can have many causes following ACS, including ischemia, reperfusion, electrolyte imbalances, underlying myocardial scar,

enhanced automaticity in the infarct border zone (the myocardium immediately adjacent to the infarcted tissue), and increased sympathetic activity. Oftentimes NSVT is caused by a combination of these factors.

Research conducted in the 1970s and 1980s suggested that NSVT after ACS is benign and is not associated with increased risk of sudden death or all-cause mortality, particularly when it occurs within 12 hours of ACS.(2,3) However, more contemporary research challenges this view. Recent findings from a large clinical trial of patients with non-ST elevation myocardial infarction (NSTEMI) showed that 25% of patients had NSVT for a duration longer than 3 beats within 7 days of their ACS event and that these patients had a twofold increase in the risk of sudden cardiac death in the next year (incidence 2.9%–4.3%).(1) This finding creates a clinical conundrum: notwithstanding the increased risk, if NSVT is common after ACS and the absolute risks remain relatively low, what are clinicians to do when they encounter NSVT after ACS?

Although NSVT is common and can be expected in most of our patients with ACS, it still requires attention. Patients who have NSVT after an ACS should be queried for symptoms of ischemia and should have an evaluation of their serum electrolytes, particularly if they are on a diuretic or have had hypokalemia or hypomagnesemia. In the case of new-onset NSVT, NSVT that is increasing in frequency or duration, or NSVT that occurs in conjunction with symptoms concerning for ischemia, an electrocardiogram should be obtained. Close inspection of the electrocardiographic QT interval is also warranted, given the frequency of acquired prolonged QT, either due to ischemia and/or pharmacotherapy. Following identification of an NSVT event, further management is largely determined by the severity of the NSVT. In most cases of NSVT, each beat has a similar appearance and is termed monomorphic VT, which is usually due to scar or increased automaticity in the injured myocardium. However, polymorphic VT is ominous and is usually due to ischemia or QT prolongation. Polymorphic VT in the setting of a prolonged QT raises concern for torsade de pointes. Patients with complete heart block or severe bradycardia are also at risk for pause-dependent torsades, another serious arrhythmia.

While the 5-beat run of NSVT in this case may have been expected, the increasing frequency should have raised concern and more in-depth evaluation, including a focused interview and physical examination, 12-lead electrocardiogram, and electrolyte and biomarker determination (e.g., CK-MB and troponin). Any evidence of ischemia should be aggressively treated in a patient with increasing ventricular arrhythmia.

The Table details a list of factors that point to high-risk NSVT. Clinicians taking care of patients with NSVT and these accompanying features should consider a more intensive monitoring environment, such as a step-down or critical care unit. While all patients with NSVT should be optimally treated with beta-blockers and revascularization whenever possible (4,5), patients with high-risk NSVT should be treated with intensified beta-blockade, provided there are no contraindications such as cardiogenic shock or profound bradycardia. In the absence of prior sustained ventricular arrhythmias, antiarrhythmic therapy is not indicated for the treatment of NSVT. In fact, prophylactic antiarrhythmic therapy following ACS has been associated with harm.(6)

Revisiting the present case, while the patient was relatively young and underwent revascularization, her NSVT had several high-risk features including increasing frequency and duration. The cause of this escalation could have been due to recurrent ischemia and certainly merited investigation. It is possible that

beta-blockade could have averted the ultimate development of sustained VT. However, escalation of the patient's level of care would likely have led the patient to a care environment that was more vigilant and better equipped for management of cardiac arrest. One wonders what was the interval between the recognition of sustained VT arrest and defibrillation? This patient's outcome is surprising because rapid cardioversion/defibrillation has a very high success rate. Thus, the importance of time to defibrillation in VT/VF (ventricular fibrillation) arrest cannot be overemphasized.(7) Nursing units and health care teams that care for high-risk patients (such as the one described above) need to be proficient in responding to sustained VT/VF, including rapid and facile execution of advanced cardiac life support (ACLS), particularly defibrillation and cardiopulmonary resuscitation. Different institutions take different approaches in order to ensure quality resuscitation. Frequent ACLS drills and the use of rapid response teams have value and are used in many hospitals. Unfortunately, large studies have failed to show improved survival following in-hospital cardiac arrest despite several interventions, including use of automated external defibrillators and rapid response teams.(8,9)

In summary, NSVT is common following ACS. It can have many causes, several of which are modifiable. A key step in the management of NSVT is risk stratification. Patients with high-risk NSVT require prompt recognition and escalation of care. Investigation of ischemia and beta-blockade are the most important interventions for patients with NSVT after ACS.

Take-Home Points

- Nonsustained ventricular tachycardia (NSVT) is common after acute coronary syndromes.
- Increasing frequency of NSVT following acute coronary syndromes is concerning and merits aggressive evaluation.
- Recurrent coronary ischemia is often a cause of escalating NSVT following acute coronary syndrome.
- Patients with frequent ventricular arrhythmia and electrical instability require intensified care.
- In the event of sustained ventricular tachycardia, rapid defibrillation is of paramount importance.

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Table

Table. High-risk features in patients with nonsustained ventricular tachycardia.

High-risk features in patients with nonsustained ventricular tachycardia.

- Long duration (greater than 7 beats) (1)
- Increasing frequency
- Polymorphic appearance
- Electrocardiographic changes / biomarker elevation
- Recurrent ischemia
- Low left ventricular ejection fraction
- Heart failure (Killip II-IV)
- Prior ventricular arrhythmia
- Occurrence more than 12–24 hours after ACS
- Bundle-branch block

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