



MY APPROACH

MY APPROACH to the patient with ventricular arrhythmia and no structural heart disease[☆]

When a patient presents with presumed “idiopathic” ventricular arrhythmias (VAs), my initial approach is to address the following issues:

1. Confirm the diagnosis of “idiopathic” VAs. The diagnosis of idiopathic VAs is one of exclusion; structural heart disease, cardiomyopathy, and coronary artery disease should be excluded, usually by cardiac stress testing and echocardiography. In selected patients (especially those with polymorphic ventricular tachycardia [VT], multiple monomorphic VT morphologies, a history of cardiac arrest or recurrent syncope, or a family history of sudden cardiac death), more comprehensive evaluation is required to exclude arrhythmogenic right ventricular cardiomyopathy, Brugada syndrome, and other cardiac channelopathies.
2. Evaluate the burden of the arrhythmia (typically using ambulatory cardiac monitoring).
3. Assess the impact of the arrhythmia on the patient's quality of life. Symptoms can be absent in some patients but disabling in others.
4. Assess the impact of the arrhythmia on the cardiac function. Frequent idiopathic nonsustained VT or premature ventricular complexes (PVCs) can precipitate a reversible form of cardiomyopathy and can also exacerbate preexisting left ventricular (LV) dysfunction or hinder the effectiveness of biventricular pacing in heart failure patients treated with cardiac resynchronization therapy.
5. Formulate a management strategy.

Given the generally benign long-term prognosis, no pharmacological or invasive therapy is recommended in patients with idiopathic VAs who are asymptomatic or only mildly symptomatic and have normal LV function. In this group of patients, reassurance and counseling are the preferred management approach. Annual follow-up with ambulatory ECG monitoring and echocardiography is recommended in those with frequent PVCs (>10,000 PVCs per 24 h) to monitor for possible development of cardiomyopathy. Although frequent PVCs can precipitate cardiomyopathy in some

patients who are otherwise asymptomatic, there is currently no risk-stratification model to reliably identify patients at risk; hence, prophylactic PVC elimination for the sole purpose of preventing PVC-induced cardiomyopathy is not recommended.

Pharmacological therapy can be considered in symptomatic patients. Beta blockers, verapamil, and diltiazem are the drugs of choice, but they have limited efficacy and often are not well-tolerated by the generally young patient population. Although class I and III antiarrhythmic drugs (sotalol, flecainide, mexiletine, propafenone, amiodarone) are more effective in reducing the burden of PVCs, they are not optimal as first-line therapy because of a greater side-effect profile.

Catheter ablation offers cure rates of over 90% and is the treatment of choice for significantly symptomatic patients in whom drug therapy is unsuccessful, not tolerated, or not preferred. Additionally, we offer catheter ablation to patients with “malignant” forms of idiopathic VAs, such as those with short-coupled PVCs and syncope or cardiac arrest, or in whom the PVCs were found to trigger polymorphic VT or ventricular fibrillation (VF). Catheter ablation is also recommended for patients with frequent PVCs or nonsustained VT when they are presumed to be contributing to a cardiomyopathy (with suspected PVC-induced cardiomyopathy) or worsening of preexisting LV dysfunction, even in otherwise asymptomatic patients.

ICD implantation is not recommended in patients with idiopathic VAs. An exception is patients with “malignant” idiopathic VAs that trigger polymorphic VT or VF, especially those presenting with syncope or cardiac arrest and in whom the PVC trigger cannot be completely eliminated by catheter ablation.

Importantly, for patients presenting with frequent PVCs and LV systolic dysfunction who have a primary prevention indication for ICD implantation, our approach is to consider therapeutic measures to reduce the PVC burden (such as catheter ablation) before implanting a prophylactic ICD. Elimination of PVCs with ablation has been shown to improve LV systolic function within a few months in the majority of these patients such that the patients no longer qualify for an ICD.

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