Non-Ischaemic Polymorphic Ventricular Tachycardia Case Report: Treatment Shift to Cardiac Ablation

Carina Lam[1] and Ebunoluwa Segun[1]

[1] School of Medicine, University College Cork, Cork, Ireland

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Abstract

Introduction: Long-term management of ischaemic polymorphic ventricular tachycardia (pVT) has traditionally relied on beta-blockers as first-line therapy, with cardiac ablation reserved as the alternative option. In non-ischaemic cases, ablation was historically less recommended due to higher procedural risk and lower success rates. Emerging evidence, however, suggests a shift toward cardiac ablation as a gold-standard treatment for both ischaemic and non-ischaemic pVT. This case describes a 41-year-old man presenting with a two-month history of exertional palpitations consistent with non-ischaemic right ventricular outflow tract (RVOT) tachycardia and the decision to pursue cardiac ablation.

Methods: Patient provided consent for the case to be used.

Results: The patient was commenced on bisoprolol as interim medical therapy and scheduled for cardiac ablation for long-term management.

Discussion: This report reviews historical treatment strategies for ischaemic and non-ischaemic pVT and examines the evolving role of cardiac ablation as first-line therapy in non-ischaemic cases.

Conclusion: Cardiac ablation was previously reserved for patients with recurrent pVT refractory to anti-arrhythmic drug therapy and for those who demonstrated limited pharmaceutical success in non-ischaemic cases. Advances in electrophysiological mapping and ablation techniques have improved cardiac ablation practices, posing it as a potential primary treatment for both ischaemic and non-ischaemic pVT. However, this shift in treatment management will depend on the results of ongoing trials such as PAUSE-SCD, IMPRESS, and LESS-VT.

Introduction

Untreated polymorphic ventricular tachycardia (pVT) carries a significant mortality risk, as it can progress to ventricular fibrillation, a cardiac emergency (1,2). Early intervention is therefore critical for long-term prognosis. pVT may be ischemia-induced or non-ischaemic, the latter most commonly due to an underlying genetic mutation (3). Both forms cause electrical abnormalities that lead to rapid, uncoordinated ventricular contractions (2).

Treatment depends on the underlying cause, with the main options being beta blockers or cardiac ablation (1,4). Beta blockers were originally first-line therapy, but following the first stage of the VANISH trials, cardiac ablation became the gold standard for ischemia-induced pVT due to superior outcomes (5). This was observed as a 13.6% absolute reduction in the risk of VT recurrence between those treated with cardiac ablation and those treated solely with beta-blockers in an ischaemia-induced VT cohort (5).

With continued innovations and fewer procedural complications, a similar shift is anticipated for non-Ischaemic pVT. Randomized controlled trials, including PAUSE-SCD, IMPRESS, and LESS-VT, are investigating whether ablation should become the default treatment for non-Ischaemic pVT (6,7).

This case report presents a 41-year-old Asian male with right ventricular outflow tract (RVOT) pVT and discusses how his management reflects the evolving approach to pVT treatment.

Case Background

41 year old male with Right Ventricular Outflow Tract Tachycardia treated at the Ottawa Heart Institute in Canada.

Presenting Complaint

T.C. is a 41-year-old Asian male presenting with a two-month history of reproducible exertional palpitations and near-syncopal episodes. He has experienced three transient episodes, each triggered by spiking a volleyball. The episodes have increased in frequency since returning from an overseas trip. During these events, he experiences blurred vision and dizziness that resolve only after extended rest. He denies associated chest pain, shortness of breath, orthopnea, or peripheral oedema.

Past Medical History

T.C. denies any illicit drug use, allergies, or known cardiac or endocrine conditions in his family.

Social History

He denies any significant stressors, or any alcohol consumption or previous smoking.

Clinical Findings

On examination, T.C. was alert and comfortable at rest. He was well perfused with normal S1 and S2 and had no additional heart sounds or stigmata of cardiac disease. At the time of assessment, his heart rate was 80 bpm with no appreciable or palpable palpitations. Head-to-toe examination was noncontributory for endocrine causes.

Investigations

All blood tests were normal. Point-of-care ultrasound showed normal left ventricular function with no pericardial effusion. ECG demonstrated sinus rhythm with a wide QRS secondary to the left bundle branch block and no evidence of prior ischaemia. Cardiac MRI showed no inflammation, infiltration, or prior infarction. Cardiac CT demonstrated normal coronary arteries with no stenosis and an Agatston score of 0. Stress testing and electrophysiological studies induced polymorphic ventricular tachycardia at a heart rate of 200 bpm, with the arrhythmic focus localized to the RVOT

Plan

Under the guidance of a cardiac electrophysiologist. T.C. commenced on 2.5mg interim course of bisoprolol and scheduled for an cardiac ablation for definitive treatment in a month's time.

Discussion

Treatment

Treatment of pVT is crucial, as this condition presents with significant complications, such as an increased risk of cardiac arrest and sudden cardiac death (SCD) (8). Studies have noted that up to 13% of patients with untreated pVT will develop SCD in their lifetime (8). Depending on the cause for the arrhythmia, treatment varies and can be subdivided into pharmacological management or procedures which can be invasive or non-invasive (8).

For long-term management of pVT, the first-line therapy traditionally consists of anti-arrhythmic drugs (AADs), particularly with non-selective beta blockers such as propranolol (8). It is recommended due to its safety profile and efficacy in reducing mortality in patients after myocardial infarction or with Heart failure with preserved ejection fraction (8). However, beta-blocker therapy has been associated with an increased risk of mortality, especially with patients who are greater than 70 years old, have low systolic blood pressure, or are tachycardic (9). In severe cases of ischaemic heart disease causing VT, cardiac ablation may be deferred in favor of implantable cardioverter defibrillators (ICD) for the prevention of sudden cardiac death (10).

Where AADs are poorly tolerated or there is a recurrence of pVT, cardiac ablation is recommended as second-line treatment and has been shown to successfully prevent VT recurrence. In Ischaemic-induced pVT, cardiac ablation is particularly effective in eliminating recurrence due to more precise mapping of arrhythmogenic lesions (3). In contrast, cardiac ablation for non-Ischaemic induced pVT have been less successful either due to poor mapping or anatomical barriers in accessing arrhythmogenic lesions.

Innovations in Treatment

Early studies showed higher recurrence rates in non-Ischaemic VT compared to Ischaemic VT, with overall recurrence after ablation reported at 43-72% within 2 years (11). Failure of cardiac ablation was due to technological and imaging limitations at the time, which made it difficult to visualize and target the lesion. While ischaemia-inducing lesions are commonly found in areas where myocardial infarctions occur, in the left ventricle, there is less consensus about where non-ischaemia induced pVT lesions predominate (12). Weinand et al. 's study (2025) determined that location depended on the type of genetic mutation. For instance, TTN mutants showed lesions in the basal anterior and septal left ventricular regions, whereas those with PKP2 mutations showed a stronger correlation with Inferolateral right ventricular lesions (13). Additionally, it was difficult to localize a specific region as non-ischemia induced pVT lesions are often distributed diffusely (13).

Advances in cardiac imaging and electrophysiologic mapping have reduced recurrence rates to 15 to 30 percent, prompting investigation into cardiac ablation as a primary treatment rather than reliance on beta blockers (7). Studies have shown that novel techniques like stereotactic ablation radiotherapy (STAR) has demonstrated a significant reduction of VT recurrence and ICD shocks by 81.5% (14), however there is still limited evidence of long term effects and potency.

Case Relevance

In this patient, the absence of comorbidities, strong physical health, and precise localization of the arrhythmogenic focus support cardiac ablation as an appropriate first-line therapy. As mapping accuracy continues to improve, cardiac ablation is expected to become the gold standard treatment. Although it is already the first-line treatment for ischemia-induced VT, the application as the primary treatment for non-ischemia-induced cases remains limited due to a lack of research and definitive data (6,15).

Conclusions

With advances in cardiac electrophysiology and improved mapping that reduce VT recurrence rates, cardiac ablation is expected to become the first line treatment for pVT, particularly because it can be performed minimally invasively. As a result, the benefits of ablation outweigh reliance on beta blockers. Unlike beta blockers, cardiac ablation offers definitive treatment rather than temporary suppression of electrical abnormalities, leading to improved long term outcomes. Therefore, the management in this case aligns with future practice, in which beta blockers serve as an alternative rather than primary therapy. Awaiting the results of current clinical trials investigating the impact of cardiac ablation on non-ischaemia induced VT, it is expected that in the next decade a change will occur, favoring ablation over pharmacological management

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