Severe VT in ischaemic cardiomyopathy: Why current care isn't enough

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Managing sustained ventricular tachycardia (VT) in patients with ischaemic cardiomyopathy and severe left ventricular systolic dysfunction presents a significant clinical challenge. These patients face a high risk of sudden cardiac death, demanding a precise and often multi-modal therapeutic approach.
This case illustrates the critical decisions involved, from initial stabilisation to long-term rhythm management, in a patient population where myocardial scarring creates a fertile substrate for re-entrant arrhythmias.

KEY TAKEAWAYS

- The Pivot: Aggressive, multi-modal therapy is often necessary for sustained VT in severe ischaemic cardiomyopathy, moving beyond single-modality approaches.
- The Data: Patients with severe left ventricular systolic dysfunction (LVEF The Action: Clinicians should consider early referral for advanced electrophysiological assessment and intervention, including catheter ablation, in addition to optimal medical therapy and device implantation.

Ventricular tachycardia in the setting of ischaemic cardiomyopathy with severe left ventricular systolic dysfunction is a life-threatening arrhythmia. The underlying pathology, typically extensive myocardial scarring from prior myocardial infarction, creates a heterogeneous substrate that facilitates re-entry circuits. These circuits are often complex, making both pharmacological and interventional management challenging. The presence of severe left ventricular systolic dysfunction, commonly defined as an ejection fraction below 35%, further complicates matters, as these hearts have reduced physiological reserve and are more susceptible to haemodynamic compromise during episodes of VT.
The typical patient presenting with sustained VT in this context is often male, with a history of multiple myocardial infarctions, and frequently has co-morbidities such as diabetes, hypertension, and chronic kidney disease. These patients have usually undergone coronary revascularisation, either percutaneous coronary intervention or coronary artery bypass grafting, but residual ischaemia or extensive scar burden persists. The clinical presentation can range from palpitations and presyncope to syncope or sudden cardiac arrest, depending on the rate and duration of the VT and the patient's haemodynamic tolerance. A thorough diagnostic work-up is essential, including a detailed history, physical examination, 12-lead electrocardiogram, echocardiography to assess left ventricular function and wall motion abnormalities, and often cardiac magnetic resonance imaging (CMR) to precisely delineate scar burden and morphology. Electrophysiological studies (EPS) are crucial for mapping the VT circuit and guiding therapy. For a comprehensive overview of modern cardiological practice, clinicians may find the Oxford Handbook of Cardiology a useful reference.

Initial Stabilisation and Diagnostic Work-up

Acute management of sustained VT depends on the patient's haemodynamic stability. Unstable patients, presenting with hypotension, altered mental status, signs of shock, or acute heart failure, require immediate synchronised direct current (DC) cardioversion. Stable patients can initially receive antiarrhythmic medications. Intravenous amiodarone is a common first-line agent, typically administered as a bolus of 150 mg over 10 minutes, followed by a continuous infusion. Procainamide or sotalol are alternatives, but their use requires careful consideration of potential proarrhythmic effects and haemodynamic impact, particularly in patients with severe left ventricular dysfunction. Beta-blockers, while cornerstone therapy for chronic heart failure, are generally not effective for acute termination of sustained monomorphic VT, but they play a vital role in preventing recurrence.

Once the patient is stabilised, the diagnostic process intensifies. A 12-lead ECG during VT is critical for characterising the morphology and determining the likely origin. Ischaemic VT is often monomorphic, indicating a re-entrant circuit within a fixed scar. Serial troponin measurements are necessary to rule out acute myocardial ischaemia as a trigger. Electrolyte abnormalities, particularly hypokalaemia and hypomagnesaemia, must be corrected promptly, as they can exacerbate arrhythmias. Imaging studies, such as echocardiography, provide essential information on left ventricular ejection fraction (LVEF), chamber dimensions, and valvular function. CMR offers superior tissue characterisation, identifying the precise location and extent of myocardial scar, which is invaluable for guiding subsequent ablation procedures. Late gadolinium enhancement (LGE) on CMR accurately delineates the arrhythmogenic substrate.

Long-Term Management Strategies

Long-term management for patients with sustained VT in ischaemic cardiomyopathy is multi-faceted, typically involving optimal medical therapy, implantable cardioverter-defibrillators (ICDs), and often catheter ablation. Optimal medical therapy includes guideline-directed medical therapy (GDMT) for heart failure, such as ACE inhibitors or angiotensin receptor blockers, beta-blockers, mineralocorticoid receptor antagonists, and SGLT2 inhibitors. These therapies improve overall cardiac function and reduce mortality, indirectly impacting arrhythmia burden. However, GDMT alone is insufficient to prevent recurrent sustained VT in high-risk patients.

ICDs are the cornerstone of secondary prevention for sudden cardiac death in these patients. Guidelines strongly recommend ICD implantation for patients who have survived an episode of sustained VT or ventricular fibrillation (VF) not due to a reversible cause. The ICD provides immediate termination of life-threatening arrhythmias through antitachycardia pacing (ATP) or defibrillation shocks. While ICDs are highly effective at preventing sudden death, they do not prevent VT episodes themselves. Frequent ICD shocks can significantly impair quality of life and are associated with increased mortality. This highlights the need for therapies that reduce the burden of VT.

Catheter ablation has emerged as a critical adjunctive therapy for recurrent sustained VT. The goal of ablation is to identify and eliminate the arrhythmogenic substrate, typically by creating lesions that interrupt the re-entrant circuits within the myocardial scar. This procedure is complex and requires advanced electrophysiological mapping techniques, often involving 3D mapping systems. Success rates vary depending on the complexity of the VT and the extent of the scar, but studies show that ablation can significantly reduce VT recurrence and ICD shocks. For patients with multiple VT morphologies or extensive scar, repeat ablations may be necessary. The procedure carries risks, including cardiac perforation, tamponade, and stroke, but in experienced centres, these are generally low. Pre-procedural planning with CMR data is increasingly used to guide ablation strategies, improving efficacy and safety.

Pharmacological Adjuncts and Emerging Therapies

Antiarrhythmic drugs (AADs) play a role in reducing VT burden, either as monotherapy or in conjunction with ICDs and ablation. Amiodarone is the most effective AAD for VT suppression in patients with structural heart disease, but its long-term use is limited by significant extracardiac toxicities, including pulmonary fibrosis, thyroid dysfunction, and liver damage. Sotalol and mexiletine are other options, but their efficacy is generally lower than amiodarone, and they also carry proarrhythmic risks. Dofetilide can be considered in selected patients, particularly those with atrial fibrillation, but requires careful inpatient initiation and monitoring due to QT prolongation risk.

Newer approaches include stellate ganglion blockade or renal denervation for refractory VT, though these are typically reserved for highly selected cases where conventional therapies have failed. Sympathetic modulation can reduce arrhythmogenicity, but the evidence base for these interventions in ischaemic cardiomyopathy is still evolving. Gene therapy and stem cell therapy are investigational and not yet part of standard clinical practice. The focus remains on optimising existing therapies and integrating them effectively to manage this challenging patient population.

Where it Falls Short

Despite advances, managing sustained VT in severe ischaemic cardiomyopathy remains challenging. The open-label nature of many real-world treatment decisions is an obvious caveat; randomised controlled trials comparing different ablation strategies or specific antiarrhythmic drug regimens are often difficult to conduct in this critically ill population. The trial was not powered to detect differences in specific scar morphologies, and that gap matters for guiding individualised therapy. Furthermore, the long-term efficacy of ablation can be limited by disease progression, with new arrhythmogenic substrates developing over time. Patient adherence to complex medical regimens and lifestyle modifications also presents a persistent challenge, impacting overall outcomes. The financial burden of ICDs and repeated ablation procedures is also a significant consideration for healthcare systems.

The optimal timing and sequence of interventions, particularly the integration of ablation with ICD implantation, continue to be debated. While ablation can reduce ICD shocks, it does not eliminate the need for an ICD in most patients with severe left ventricular dysfunction. The decision to pursue ablation often comes after multiple ICD shocks, rather than proactively. This reactive approach may expose patients to unnecessary morbidity from shocks. Future research needs to focus on identifying patients who would benefit most from early, aggressive ablation strategies to prevent the first shock, and on developing more durable ablation techniques that account for the dynamic nature of the arrhythmogenic substrate in ischaemic cardiomyopathy.

> **CLINICAL IMPLICATIONS**
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> The management of sustained ventricular tachycardia in ischaemic cardiomyopathy with severe left ventricular systolic dysfunction demands a proactive, integrated approach. Relying solely on optimal medical therapy and an ICD is often insufficient to prevent recurrent, debilitating episodes and the associated morbidity from shocks.
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> Clinicians should consider early referral for electrophysiological assessment and catheter ablation in patients experiencing their first episode of sustained VT, especially if haemodynamically stable. This aggressive strategy may reduce the burden of ICD shocks, which are known to negatively impact patient quality of life and long-term prognosis.

> The role of advanced imaging, particularly cardiac MRI, in delineating the arrhythmogenic substrate cannot be overstated. Integrating this information into pre-procedural planning for ablation can significantly improve procedural success rates and reduce complications. We must move beyond a reactive approach to VT management in this high-risk group.

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