Non-Invasive Ablation of Ventricular Tachycardia

Dr Shaemala Anpalakhan

Newcastle upon Tyne Hospitals NHS Foundation Trust
Freeman Road, Newcastle Upon Tyne, NE7 7DN

Contact: shaemala@doctors.org.uk

Introduction

Ventricular tachycardia (VT) can occur in both structurally normal and abnormal hearts. On the surface electrocardiogram (ECG) it is characterised as a regular broad complex tachycardia. Untreated these arrhythmias are associated with ICD interventions, including shocks, as well as haemodynamic disturbance, syncope and death. In the abnormal heart, VT occurs in the presence of scar tissue which disrupts the normal patterns of cellular activation, leading to the formation of micro and macro re-entry circuits.

Management of VT

In addition to optimal management of underlying cardiac disease, specific treatment options for VT include:

i) Anti-arrhythmic drugs

ii) Implantable cardiac defibrillator (ICD)

iii) Catheter ablation using radiofrequency energy.

Anti-arrhythmic agents can be associated with significant side effects (e.g. amiodarone) and may also worsen a given individual's risk of arrhythmia. ICDs have been demonstrated to improve survival in patients at risk of life threatening

dysrhythmia but can be associated with inappropriate shocks, which can be multiple during periods of VT storm.

Catheter ablation is an invasive procedure which involves the mapping of arrhythmic substrate using either voltage criteria (voltage mapping) or impulse propagation (activation mapping). Arrhythmic substrate is usually endocardial; requiring peripheral venous access and either peripheral arterial access or a trans-septal puncture to access the left sided circulation.

VT circuits are however not always endocardial. Epicardial substrate requires percutaneous puncture into the pericardial space. The further risks of an epicardial ablation therefore include cardiac tamponade, phrenic nerve injury and inadvertent damage to surrounding organs including the liver. Prior to catheter ablation scar tissue imaging is required from either echocardiography or cardiac magnetic resonance imaging (CMR). The use of CMR may be limited by the presence of an ICD, however can be undertaken in some centres with close supervision, in those with compatible ICD systems.

Patients with intractable VT often already have an ICD in situ; may have had previous ablations and can already be on more than one anti-arrhythmic agent. Treatment options are limited and these patients have a poorer prognosis[1]. Furthermore if the location of the circuit is difficult to access, radiofrequency energy may be technically challenging to apply.

Non Invasive Ablation

In December 2017, the New England Journal of Medicine published a study looking at the feasibility and impact of radiotherapy as a non-invasive method of ventricular tachycardia ablation[2]. The aim of the study was to investigate the role of radiotherapy in treating this group of patients.

The study included five patients with recurrent VT (three with non-ischemic and two with ischemic cardiomyopathy, mean age 66 years). In each patient the above three treatment strategies had already been applied and/or considered. Two patients had contraindications to catheter ablation, for example one with non-ischemic cardiomyopathy had a metallic mitral valve requiring uninterrupted anticoagulation. As per hospital protocol, they were all evaluated for possible heart transplantation and deemed not eligible. Patients with ventricular assist devices were also excluded from the study.

The study combined multi-electrode surface ECGs, indwelling ICDs to induce and terminate VT, computed tomography (CT) or CMR to plan the electro-anatomical site for the application of radiotherapy. Stereotactic body radiation therapy (SBRT), usually applied in the treatment of solid tumours, was used as a means of delivering radiation to the scar tissue of interest, as early preclinical studies had shown some feasibility using this technique. In this study a dose of 25Gy in a single fraction was used and the mean treatment time was fourteen minutes. Doses required in the treatment of solid tumours can be between 20Gy and 80Gy, usually delivered in multiple fractions.

Study follow up was twelve months. Where possible anti-arrhythmic agents were reduced or suspended (patients were on amiodarone and mexiletine on discharge). Beta-blockers were continued in the context of heart failure management. ICDs were used to monitor the burden of VT pre and post radiotherapy. Echocardiography and chest CT were used to assess for cardiac and extra-cardiac adverse events respectively.

Results

There was a significant reduction in VT episodes in all five patients. During the 3 months before treatment, the patients had a combined history of 6577 episodes of ventricular tachycardia. During a 6-week post-ablation "blanking period" (when arrhythmias may occur owing to post-ablation inflammation), there were 680 episodes of ventricular tachycardia. After the 6-week blanking period, there were 4 episodes of ventricular tachycardia over the next 46 patient-months, representing a reduction from baseline of 99.9%

One patient died three weeks post treatment due to a stroke (anticoagulation was not prescribed due frailty-associated bleeding risk). Three patients out of the remaining four wear successfully weaned off their anti-arrhythmic agents. There was no overall change in mean ejection fraction. Inflammatory changes detected in nearby lung tissue at three months, which had resolved at twelve months.

Discussion

The results of this small study are certainly exciting as it potentially provides another treatment strategy in patients with recurrent VT, especially where conventional

options have been exhausted. Although clearly this is a very small data set, and as already described in the study further refinements are required. For example the dose of radiotherapy applied would have to have no clinically significant sequelae for both the cardiac and extra-cardiac tissue. Although given the poor prognosis in recurrent VT the importance of long-term side effects may be diminished.

Imaging modalities are also limited by motion artefact and this will need to be accounted for when applying radiotherapy, especially when wanting to avoid healthy cardiac tissue, especially the coronary arteries. Furthermore this study looked at endocardial VT and excluded epicardial VT. Finally while this study suggests at a useful fourth long-term treatment strategy in VT management, it does not provide a fix for a patient acutely in VT.

References

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