PO-637-08

A COMPARISON BETWEEN DECELERATION ZONES ON ISOCHRONAL LATE ACTIVATION MAPPING AND SUBSTRATE FOR VENTRICULAR TACHYCARDIA USING CT LATEIODINATED CONTRAST ENHANCEMENT AND WALL THINNING

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Background: Isochronal late activation mapping (ILAM) is an important tool for identification of targets for ablation without requiring induction of ventricular tachycardia. Computed tomography is also a promising non-invasive method of identification of scar. Late iodinated contrast enhancement and wall thinning with acquisition of sub-1mm slice thickness are used, which are surrogates for ablation targets. The relationship of deceleration zones observed on ILAM with substrate observed on CT imaging is unknown.

Objective: To assess the correlation between deceleration zones seen on ILAM with CT substrate.

Methods: Consecutive patients who underwent VT ablation during a 2-year period using the NAVX (St. Jude Medical, Minneapolis, MN) electroanatomic mapping system and underwent CT imaging (InHeart, Bordeaux, France) were retrospectively reviewed. Patients with incomplete maps, low density maps, or incomplete imaging were excluded (n=15). After CT image integration into the EA map, the presence of DZs was assessed and the presence of underlying substrate on imaging defined. All maps and images following integration were evaluated by 3 independent reviewers who were blinded to ablation lesion sets and clinical outcomes. The DZ was identified and demarcated and the superimposed substrate on imaging was evaluated.

Results: There were 20 patients in the final analysis. The cohort was 80% male with mean age of 68 years with a left ventricular ejection fraction of 34%. Ischemic heart disease was present in 55% of subjects. In 17 of the 20 patients (85%) wall thinning of 1-3mm superimposed on the DZ sites was observed. Dense scar was present in only 7 patients (35%) at DZ sites.

Conclusion: DZs observed on ILAM area are highly associated with areas of wall thinning from 1-3 mm on CT. Dense scar was less frequently noted with DZs. These findings may serve as a basis for imaging guided VT ablation in future studies.

Figure 1: An isochronal late activation map of the left ventricle in a left lateral view. An image of the iodinated CT has been overlaid with purple and yellow areas representing wall thinning.

PO-638: Posters: Clinical EP at Pod 10

Friday, April 29, 2022
3:00 PM - 5:00 PM

PO-638-01

THE FUTURE OF ACLS: IVABRADINE’S ROLE FOR THE TREATMENT OF REFRACTORY VENTRICULAR ARRHYTHMIAS

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Background: Ivabradine (IVA) is an attractive antiarrhythmic for the treatment of refractory ventricular arrhythmias (RVA) as it does not affect blood pressure or prolong the QT interval. IVA’s efficacy in ventricular tachycardia (VT)/ventricular fibrillation (VF) has been validated in multiple animal studies. We present a case series demonstrating complete resolution of RVA in humans with the addition of IVA.

Objective: Illustrate the outcomes of RVA with the treatment of IVA.

Methods: Retrospective case series analysis of 4 patients treated with IVA for RVA.

Results: Pt 1: 63M with ischemic cardiomyopathy was admitted for RVA despite amiodarone, quinidine, and a beta block. IVA 2.5mg BID was initiated with resolution of VF. Pt 2: 61F was admitted with cardiogenic shock secondary to giant cell myocarditis complicated with VT/VF. IVA 2.5mg BID was initiated for RVA despite amiodarone and quinidine. IVA was up titrated to 5mg BID with resolution of VT/VF. Pt 3: 58M without significant history was admitted with a VF arrest. The addition of IVA 2.5mg BID to amiodarone, quinidine, and procainamide resolved VF burden. Pt 4: 37M with cardiomyopathy with continued VT/VF despite amiodarone, and lidocaine, and Impella mechanical circulatory support. IVA 2.5mg BID was initiated, and lidocaine was weaned. These therapies stabilized his rhythm, allowing for successful left ventricular assist device implant. All four patients’ RVA (Images A, C, E, G) were converted to sinus rhythm (Images B, D, F, H) with the addition of IVA to standard antiarrhythmic drugs (Table 1/Figure 1).

Conclusion: IVA may be a beneficial additive treatment for RVA. However, randomized studies are required to determine the clinical success of RVA attenuation with IVA.

Table 1: Clinical characteristics of patients with refractory ventricular arrhythmias treated with ivabradine.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Baseline Parameters</th>
<th>IVA Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt 1</td>
<td>63</td>
<td>M</td>
<td>Ischemic</td>
<td>LVEF 36%</td>
<td>2.5mg BID</td>
<td>VF resolved</td>
</tr>
<tr>
<td>Pt 2</td>
<td>61</td>
<td>F</td>
<td>Myocarditis</td>
<td>LVEF 35%</td>
<td>2.5mg BID</td>
<td>VF resolved</td>
</tr>
<tr>
<td>Pt 3</td>
<td>58</td>
<td>M</td>
<td>No history</td>
<td>LVEF 34%</td>
<td>2.5mg BID</td>
<td>VF resolved</td>
</tr>
<tr>
<td>Pt 4</td>
<td>37</td>
<td>M</td>
<td>Cardiomyopathy</td>
<td>LVEF 32%</td>
<td>2.5mg BID</td>
<td>Sinus rhythm</td>
</tr>
</tbody>
</table>