ΚΑΤΑΛΥΣΗ ΚΟΙΛΙΑΚΗΣ ΤΑΧΥΚΑΡΔΙΑΣ ΑΠΟ ΤΟΝ ΧΩΡΟ ΕΞΩΘΗΣΗΣ ΤΗΣ ΔΕΞΙΑΣ / ΑΡΙΣΤΕΡΗΣ ΚΟΙΛΙΑΣ ΚΑΙ ΘΗΛΟΕΙΔΩΝ ΜΥΩΝ

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ΙΔΙΟΠΑΘΕΙΣ ΚΟΙΛΙΑΚΕΣ ΑΡΡΥΘΜΙΕΣ

Outflow tract VT

Perivalvular VT

Intracavity VT

Epicardial VT

Fascicular VT
ΦΑΣΜΑ ΤΩΝ ΑΡΡΥΘΜΙΩΝ ΑΠΟ RVOT/LVOT

• right ventricular outflow tract (RVOT-VT)
• left ventricular outflow tract (LVOT-VT)
• aortic cusps (Cusp VT)
• from the pulmonary artery (PA VT)
OT-VT tend to occur in the absence of structural heart disease and are focal in origin. The 12-lead ECG recorded during VT is a precise localizing tool.
Η ιδιοπαθής είναι οντωσί ιδιοπαθής;
Importance Of Delayed Enhanced Cardiac MRI In Idiopathic RVOT-VT: Differentiating Mimics Including Early Stage ARVC And Cardiac Sarcoidosis
Malignant Entity of Idiopathic Ventricular Fibrillation and Polymorphic Ventricular Tachycardia Initiated by Premature Extrasystoles Originating From the Right Ventricular Outflow Tract

- 101 pts with RVOT VT
- 16 pts with spontaneous VF/PVT initiated by LBBB/inferior axis VE
- Free of syncope/VF/CA post RFCA of the RVOT origin

Table 2. Comparison of the Clinical Parameters Between the VF/PVT Group and the RVOT-VT Group

<table>
<thead>
<tr>
<th></th>
<th>VF/PVT Group (n = 16)</th>
<th>RVOT-VT Group (n = 85)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>7/16 (44%)</td>
<td>25/85 (29%)</td>
<td>0.26</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>39 ± 10</td>
<td>43 ± 14</td>
<td>0.19</td>
</tr>
<tr>
<td>FH</td>
<td>1/16</td>
<td>1/85</td>
<td>0.29</td>
</tr>
<tr>
<td>Duration from onset of symptom to RFCA (months)</td>
<td>80 ± 103</td>
<td>69 ± 79</td>
<td>0.71</td>
</tr>
<tr>
<td>History of syncope</td>
<td>11/16 (69%)</td>
<td>15/85 (18%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Holter ECG findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isolated PVC (/day)</td>
<td>17,554 ± 11,338</td>
<td>15,506 ± 16,053</td>
<td>0.58</td>
</tr>
<tr>
<td>CI of VE (ms)</td>
<td>409 ± 62</td>
<td>428 ± 65</td>
<td>0.27</td>
</tr>
<tr>
<td>QRS duration of VE (ms)</td>
<td>148 ± 8</td>
<td>142 ± 12</td>
<td>0.03</td>
</tr>
<tr>
<td>CI of VT (ms)</td>
<td>245 ± 28</td>
<td>328 ± 65</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Noda et al. JACC 2005
RVOT VT constitutes 75% of all patients with outflow tract VT.

RVOT VT is more common in females, 30-50 years old.

Symptoms include palpitations, dizziness, atypical chest pain, and syncope.

Exercise testing reproduces the patient’s clinical VT 25 to 50% of the time.
MAPPING TOOL FOR OT-VT ECG

- ECG morphology
  - Could be non-inducible

- Pace mapping
  - Could be large area 2 cm²: different chamber, scar, or epicardium,

- Activation map
  - More accurate: more mapping sites, epicardium, different energy sources,
RVOT region can be divided into nine regions

Anterior sites demonstrate Q wave (Q or qR) in lead I and QS in lead aVL

Posterior sites demonstrate R wave in lead I and early precordial transition (R > S in V3)

Between anterior and posterior locations typically demonstrate a multiphasic QRS morphology in lead I.
Differentiation of septal from free wall RVOT VT

RVOT VTs originating from septum - taller, narrower monophasic R waves in inferior leads

Free wall RVOT VT - typically broader QRS (>140ms) and R wave notching in inferior leads

Later transition in precordial leads (>V4)
Monomorphic ventricular tachycardia with LBBB morphology and an inferior axis.

: DDx of RVOT and ASC origin

A $V_2$ transition ratio >0.6 predicted an LVOT origin with a sensitivity of 95% and specificity of 100%

Betensky et al, JACC 2011
Coupling Interval Variability Differentiates Ventricular Ectopic Complexes Arising in the Aortic Sinus of Valsalva and Great Cardiac Vein From Other Sources
Mechanistic And Arrhythmic Risk Implications
LVOT VT

- S wave in L1, R-wave transition in V1 or V2
- S wave in V5 or V6 absent
- S wave in V5 or V6 present

Supravalvular LVOT VT
Infravalvular LVOT VT

ECG waveform with arrows indicating potential LVOT VT.
LVOT VT MORPHOLOGY
Ventricular Tachycardia Originating From the Posterior Papillary Muscle in the Left Ventricle
A Distinct Clinical Syndrome

Harish Doppalapudi, MD; Takumi Yamada, MD; H. Thomas McElderry, MD; Vance J. Plumb, MD; Andrew E. Epstein, MD; G. Neal Kay, MD

Background—Several distinct forms of focal ventricular tachycardia (VT) from the left ventricle (LV) have been described. We report a new syndrome of VT arising from the base of the posterior papillary muscle in the LV.

Methods and Results—Among 290 consecutive patients who underwent ablation for VT or symptomatic premature ventricular complexes (PVCs) based on a focal mechanism, 7 patients were found to have an ablation site at the base of the posterior papillary muscle in the LV. All patients had normal LV systolic function and a normal baseline electrocardiogram. The electrocardiogram during VT or PVCs demonstrated a right bundle-branch block and superior-axis QRS morphology in all patients. VT was not inducible by programmed atrial or ventricular stimulation. In 2 patients with sustained VT, overdrive pacing neither terminated VT nor demonstrated any criterion for transient entrainment. Activation mapping localized the earliest site of activation to the base of the posterior papillary muscle in all patients. When Purkinje potentials were recorded at the site of successful ablation, these potentials preceded local ventricular muscle potentials during sinus rhythm. During VT or PVCs, however, the ventricular muscle potential always preceded the Purkinje potentials. After recurrence of VT or PVCs with standard radiofrequency ablation, irrigated ablation was successful in eliminating the arrhythmia in all patients. Over a mean follow-up period of 9 months, all patients have been free of PVCs and VT.

Conclusion—We present a distinct syndrome of VT arising from the base of the posterior papillary muscle in the LV by a nonreentrant mechanism. Ablation can be challenging, and irrigated ablation may be necessary for long-term success. (Circ Arrhythmia Electrophysiol. 2008;1:23-29.)

Key Words: tachycardia, ventricular, papillary muscles, posterior, catheter ablation
CONSIDERATION FOR ABLATION

1. Symptomatic when drug therapy is ineffective, not tolerated or not preferred

2. PVC- or tachy-mediated cardiomyopathy

3. High risk profile
Radiofrequency Ablation Versus Antiarrhythmic Medication for Treatment of Ventricular Premature Beats From the Right Ventricular Outflow Tract
Prospective Randomized Study

Table 1. Baseline Characteristics* of Study Patients

<table>
<thead>
<tr>
<th></th>
<th>AADs Group (n=165)</th>
<th>RFCA Group (n=165)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>50.54±11.52</td>
<td>52.68±10.37</td>
</tr>
<tr>
<td>Women n (%)</td>
<td>125 (75.8%)</td>
<td>118 (71.5%)</td>
</tr>
<tr>
<td>VPB burden, %</td>
<td>14 (IQR: 12, 21)</td>
<td>14 (IQR: 12, 21)</td>
</tr>
<tr>
<td>VPB numbers</td>
<td>13823 (IQR: 11948, 19892)</td>
<td>14049 (IQR: 11882, 19535)</td>
</tr>
<tr>
<td>Hypertension (n)</td>
<td>11 (165)</td>
<td>10 (165)</td>
</tr>
<tr>
<td>LAD, mm</td>
<td>34.39±2.65</td>
<td>34.78±2.76</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>64.48±4.89</td>
<td>64.07±5.21</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.38±2.53</td>
<td>23.80±2.36</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>130.56±7.52</td>
<td>128.15±7.23</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>80.47±7.61</td>
<td>77.84±5.56</td>
</tr>
</tbody>
</table>
Beneficial effects of catheter ablation of frequent premature ventricular complexes on left ventricular function

Study Name | N | Confidence Interval
---|---|---
Sekiguchi Y (2005) | 38 | -2.0 (-0.6 , 4.8) |
Takemoto M (2005) | 14 | -7.0 (-7.2 , -6.8) |
Leiakowski J (2009) | 22 | -4.6 (-5.8 , -3.4) |
Sarrazin (2009) | 15 | -5.0 (-7.1 , -2.9) |
Kim YH-Subgroup (2010) | 16 | -4.0 (-5.2 , -2.8) |
Wijnmaalen AP (2010) | 34 | -7.0 (-7.6 , -6.4) |
Del Carpio Munoz F (2011) | 30 | -2.8 (-3.6 , -2.0) |
Mountantonakis SE (2011) | 69 | -3.2 (-3.8 , -2.6) |
Ban JE (2012) | 28 | -7.0 (-8.2 , -5.8) |
Kuroki K (2012) | 31 | -3.0 (-3.5 , -2.5) |
Lakireddy D (2012) | 65 | -4.6 (-5.1 , -4.1) |
Lu F (2012) | 24 | -4.6 (-6.0 , -3.1) |
Yokokawa M (2012) | 249 | |
Penella D (2013) | 53 | |
Overall | | |

Change in LVEF post ablation

Change in LVEDd post ablation

Zang M et al. Heart 2014
ΈΦΗΒΟΣ 15 ΕΤΩΝ - ΠΡΩΤΑΘΛΗΤΗΣ ΣΤΙΒΟΥ

35000 PVCs/24hr

Last fu 3 years Holter <300/24hr
RIGHT CORONARY CUSP PVC

44000 PVCs and NSVTs/24hr in Holter
2.5 years asymptomatic with total recovery of LV function and size
ΣΥΜΠΕΡΑΣΜΑ

1. Η λέξη ιδιοπαθής συνήθως χρησιμοποιείται για να καλύψει τα κενά γνώσης στην ιατρική

2. Η ιδιοπαθής ΚΤ δεν χρειάζεται ΗΦ εκτίμηση ή θεραπεία σε κάθε περίπτωση........αλλά

3. Συνεκτίμηση με στοιχεία όπως ιστορικό (ΑΚΘ), ΗΚΓ (μορφολογία), απεικόνιση (MRI)......

«Ο μεγαλύτερος εχθρός της γνώσης δεν είναι η άγνοια, αλλά η ψευδαίσθηση της γνώσης»

Stephen Hawking
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