How Can I Know This Patient Has Normal Heart

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PVCs, When to worry!
M/61 Palpitation

24hr Holter monitoring

<table>
<thead>
<tr>
<th>General</th>
<th>Heart Rates</th>
</tr>
</thead>
<tbody>
<tr>
<td>99033 QRS complexes</td>
<td>46 Minimum at 22:51:04 08-May</td>
</tr>
<tr>
<td>8944 Ventricular beats (9%)</td>
<td>66 Average</td>
</tr>
<tr>
<td>39 Supraventricular beats (&lt; 1%)</td>
<td>132 Maximum at 11:17:17 08-May</td>
</tr>
<tr>
<td>&lt; 1% of total time classified as noise</td>
<td>599 Beats in tachycardia (&gt;=100 bpm), &lt; 1% total</td>
</tr>
<tr>
<td></td>
<td>19169 Beats in bradycardia (&lt;=60 bpm), 19% total</td>
</tr>
<tr>
<td></td>
<td>1.8 Seconds Max R-R at 23:33:29 08-May</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Supraventriculars (S, J, A)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>21 Isolated</td>
<td>2 Beats longest run 110 bpm 22:50:53 08-May</td>
</tr>
<tr>
<td>2 Couplets</td>
<td>3 Beats fastest run 137 bpm 18:48:11 08-May</td>
</tr>
<tr>
<td>3 Bigeminal cycles</td>
<td></td>
</tr>
<tr>
<td>82 Bigeminal cycles</td>
<td></td>
</tr>
<tr>
<td>0 Runs totaling 0 beats</td>
<td></td>
</tr>
</tbody>
</table>

12-lead ECG

2D EchoCG: no structural heart disease
F/50 palpitation

24hr Holter monitoring

12-lead ECG

2D EchoCG: no structural heart disease
PVCs incidence

• 1% to 4% in the general population
• Clinical normal or apparently healthy people
  • 1% by 12-lead ECG
  • 40-75% by 24-48 hours ambulatory (Holter) ECG recordings
• Commonly thought to be a benign entity
• Increase with age
  • More prevalence...but more heart diseases

Kennedy HL et al., N Engl J Med 1985
Diagnostic evaluation of PVCs

- Symptomatic PVCs: Initial evaluation: 12-lead ECG, 24 hr ambulatory monitor
  - Daily burden (number or %)
  - Different morphologies
- Intermittent symptoms: may need 30-day monitoring, rarely loop recorder
- TTE: to identify significant structural heart disease
- Cardiac MRI or cardiac PET scan
  - Multiple morphologies of PVC or abnormal surface ECG
  - ARVC, amyloidosis, sarcoidosis
- Exercise ECG
  - Response to ecotype to catecholamines
  - Provoked sustained or nonsustained VT
- EPS: not generally necessary
History Taking

• Symptomatic
  • Palpitations, fatigue, DOE, exertional intolerance, lightheadedness, presyncope, syncope, CHF symptoms, 헷기침, 낮에는 모르겠는데 자기 전에 심장이 두근거려요

• Asymptomatic
  • 맥을 잡아보면 심장이 안 뛰어요 (맥이 걸러요)
Symptoms

• Palpitation, dizziness
• Caused by subsequent compensatory pause
• The pause allows time for greater calcium uptake by the myocardium and a sinus beat following PVC is hypercontractile.

Post-extrasystolic potentiation
PVC, bigeminy

Nonperfused PVC producing a sudden decrease in HR and presyncope
Triggers

• Exercise/Rest
• Alcohol
• Caffeine
• Dehydration
• For women, menses
• Stress
• Fatigue, lack of sleep
• Eating a large meal
• Lying on my left side
Social and Family histories

• Is there any substance abuse (alcohol, drugs, stimulants)

• Is there a family of sudden death, arrhythmias cardiomyopathy
Many Types of PVC

- Idiopathic
- PVCs due to structural heart disease
  - ARVC
  - Cardiomyopathy (ischemic and non ischemic)
- PVCs due to inherited channelopathies
  - Catecholaminergic Polymorphic VT
PVCs (RVOT origin)

- LBBB morphology
- Inferior axis
- Transition >V3
- Negative in aVR and aVL
RVOT VT

M/20

GXT도중 VT발생 → carotid sinus massage 후 termination

3-4년 전부터 운동 시 불규칙한 맥박 있었으나 경과관찰 중에 증상이 자주 발생하여 내원
지속시간: 길면 2-3분, 짧으면 10-15초 정도. 증상 있을 때 헤미.field 지시가 흐려진다 함.
Outflow Tract PVC’s

- Most common idiopathic PVC origin
- Clinical course is typically benign – no increase in mortality compared to control population
- Bimodal age distribution (30s/40s and 70s-80s)
- Female predominance
- Mechanism is “triggered activity” (focal)
- Can originate from the RVOT or Aortic cusps/LVOT
- Exacerbated during exertion
- Hormonal triggers in women
Treatment Options for Idiopathic PVCs

- Reassurance (if asymptomatic, normal EF, PVC burden low)
- β-blockers
- Class Ic AAD (flecainide and propafenone) if preserved LVEF
- Class III AAD (soltalol, amiodarone) if LVEF reduced
- Catheter ablation about 90% effective
Three major concerns in PVCs

1. Risk for sustained VT
2. Ventricular beat may be coupled closely with the preceding QRS complex and produce VF
3. Tachycardia-induced cardiomyopathy
PVCs, When to Worry!

- History of syncope
- Frequent ectopy
- Abnormal LVEF (PVC induced cardiomyopathy)
- Right precordial T wave inversions in SR-abnormal RV function
- Fast sustained RVOT VT/200 bpm
- Short coupled PVCs or Torsades
- Multiple VT/PVC morphologies or unusual morphology
PVC-induced CMP
Pathophysiology

- Alterations in intracellular calcium and membrane ionic currents
- Hemodynamic impairment
- Alterations in heart rate dynamics
- Tachycardia-induced cardiomyopathy
- PVC-induced cardiomyopathy
- Ventricular dyssynchrony
- Increased oxygen consumption
- Myocardial and peripheral vascular autonomic dysregulation

Cha YM et al. Circ Arrhythm Electrophysiol. 2012;5:229-36
PVCs induced CMP: PVC burden cut off value (24h Holter)

16%

24%

26%

Baman TS et al. Heart Rhythm 2010;7:865-869
PVC burden and cardiomyopathy

- PVC >10% start seeing reduced LVEF
- Even more LV dysfunction when PVC burden approaches >4%

N=174 pts with frequent PVCs = PVC burden 57/174 (33%) with decreased EF

Baman TS et al. Heart Rhythm 2010;7:865-869
Reversibility of PVC-induced CMP

• After successful ablation (80% reduction in initial PVC burden) most patients demonstrate recovery in EF within 4 months.

• RFCA vs Medical Tx

Common PVC site of origin

• RVOT
• LVOT
• RCC, LCC, AMC
• Left posterior fascicle
• Posteromedial papillary muscle

Prystowsky, En et al, J Am Coll Cardiol 2012;59:1733-44
ECG patterns associated with selected outflow tract locations
ECG RVOT vs LVOT

LVOT

RVOT

Early transition \(\leq V2\)

Late transition \(\geq V3\)
LVOT PVC/VT

• 10-15% of idiopathic VT and can be mapped to the aortic cup with other LV location (AMC).
• LBBB, inferior axis morphology
• Early transition< V₃
  • PVC from RCC: transition V₂-V₃
  • PVC from LCC: transition V₁-V₂
• Broader R-wave in V₁ (duration>40% of QRS and amplitude>30%)
LVOT PVC
$V_2$ transition ratio

$\frac{B}{(B+C)}_{PVC} \geq 0.6 \rightarrow \text{LVOT origin}$

Batensky BP, et al. JACC 2011;57:2255-62
Algorithm for OTVT with Lead V₃ PVC/VT R/S Transition

Lead V₃ PVC/VT R/S transition

PVC/VT R/S transition later than SR?

Yes → RVOT

No → Measure V₂ Transition Ratio

<0.6 → RVOT

≥0.6 → LVOT
Frequent PVCs

**Diagnosis and quantification of frequent PVCs**
- Symptoms: palpitations, presyncope, or decreased effort tolerance
- Physical exam: often normal, premature beats may be appreciated
- ECG: to determine PVC morphology
- Holter monitoring: to quantify PVC burden

**Echocardiography**
- To assess for LVEF and LV structural abnormalities

**Further cardiac evaluation**
- MRI, stress imaging and CAG if indicated
- Workup to exclude cardiomyopathies d/t other causes, such as drugs and endocrinologies

**Suppression of PVCs**
- If PVC-induced CMP is presumed, proceed with medical therapy or catheter ablation

**Follow-up**
- FU of between 3 and 12 months with repeat Holter and echocardiography
PVCs, When to Worry!

- History of syncope
- Frequent ectopy
- Abnormal LVEF (PVC induced cardiomyopathy)
- Right precordial T wave inversions in SR—abnormal RV function
- Fast sustained RVOT VT/200 bpm
- Short coupled PVCs or Torsades
- Multiple VT/PVC morphologies or unusual morphology
43-year old male with palpitation
2D EchoCG
Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

- Fibrous or fibrofatty replacement of the RV
- ECG depolarization/repolarization changes
- Ventricular tachycardia
- Sudden cardiac death
- Familial
- Male to female 2.8:1
- Prevalence 0.02-0.1%
PVCs, When to Worry!

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The "Short-Coupled" Variant of Right Ventricular Outflow Ventricular Tachycardia: A Not-So-Benign Form of Benign Ventricular Tachycardia?

SAMII VISKin, M.D., RAphaEL ROSSo, M.D., ORI roGOWSKI, M.D., and BERNArd BElHASEn, M.D.

Malignant Entity of Idiopathic Ventricular Fibrillation and Polymorphic Ventricular Tachycardia Initiated by Premature Extrasystoles Originating From the Right Ventricular Outflow Tract

Takashi Noda, MD, PhD,* Wataru Shimizu, MD, PhD,* Atsushi Taguchi, MD,* Takeshi Aiba, MD, PhD,† Kazuhiro Satomi, MD,* Kazuhiro Suyama, MD, PhD,* Takashi Kurita, MD, PhD,* Naohiko Aihara, MD,* Shiro Kamakura, MD, PhD*

Suita, Japan
## PVCs, When to worry

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

<table>
<thead>
<tr>
<th>Parameter</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>CL of VT (ms)</td>
<td>245 ± 28</td>
<td>328 ± 65</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>
Morphologies of PVCs

• In individuals without SHD:
  • High voltages, unnotched QRS complexes
  • ST segment depression when QRS positive and vice versa
  • T wave has asymmetrical branches

• Individuals with SHD:
  • QRS complexes present notched and slurring and are of low voltage
  • Symmetrical T waves
PVCs in underlying structural heart disease
GISSI-2 Study

6-month survival of patient by PVCs

Maggioni AP et al. Circulation 1993;87:312 –22
Treatment of PVCs in SHD

• Baseline optimal medical therapy

• If PVC burden > 10K – PVC suppression with AAD or ablation

• If EF < 35% ICD

• If LBBB CRT

• Ablation is also an option
Summary

• Careful history as well as review of diagnostic studies
  • will guide you to treat your patient in the most effective manner

• Knowing the origin of the PVC
  • In deciding on treatment options and in discussion potential complications for ablation (right vs left/epicardial)

• Knowing when to worry
  • syncope, frequent ectopy>20K daily PVCs, decreased LVEF or RVEF, fast sustained RVOT VT>230 bpm, short coupled PVCs, multiple PVC morphologies, unusual PVC morphology)