Polymorphic ventricular tachycardia in patients with vasospastic angina

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Introduction

*Development of polymorphic ventricular tachycardia (PVT) and sudden cardiac death (SCD) is frequently observed in the conditions of prolonged QT intervals.

*In the settings of normal QT intervals, PVT and SCD are mostly seen in acute myocardial infarction and catecholaminergic PVT (CPVT).

*Vasospastic angina is another condition to develop PVT and SCD with normal QT interval, although its incidences are relatively low, but should be kept in mind for clinical practice.
Case presentation-1; 57 yrs, male

[Clinical symptoms]; Syncope

[Past history]; Hypertension, AF, gout

[Family history]; np

[Present illness];

* 03/2001 During admission to other hospital because of embolism in superior iliac artery, he experienced frequent episodes of chest pain. His ECG monitor demonstrated ST elevation and development of VT/VF associated with chest pain.

* Coronary angiogram demonstrated multiple coronary arterial spasms.
* After administration of Ca antagonist and nitrate, he was free from chest pain and arrhythmias.

* On 07/2005, he was admitted to the other hospital for the treatment of gastric ulcer.

* On 07/19/2005, he was given medication for gastric ulcer and, then, he again experienced chest discomfort.

* At noon time of next day, he suddenly passed out with his monitor ECG showing VF.

* On 07/22/2005, he was referred to our hospital for re-evaluation and treatment of VF.
Baseline 12-lead ECG

Chest X-ray

UCG
Dd/Ds; 44/30, EF=60%, AR II°, MR I°
His baseline 12-lead ECG showed AF and flattened T waves in V5/V6. Chest X-ray demonstrated no remarkable changes. UCG showed mild to moderate mitral (MR) and aortic regurgitation (AR).
VF (11:37)

VT (17:54)

ST elevation (19:28), VT

* Continuous
His monitor ECG on the 2nd admission. PVT and VF were seen with ST elevation.
Findings of Coronary Angiogram; No organic stenosis at baseline

[RCA]
# 1: spontaneous spasm of 90% occlusion at baseline

[LCA]
# 6: 90% and # 7: 100% occlusion
#13: 99% occlusion by 20 \(\mu g\) Acetylcholine (Ach)
Findings of his coronary angiogram demonstrated multiple arterial spasm on both RCA and LCA during baseline and after intra-coronary administration of Ach.
Programmed ventricular stimulation at RVOT

1. Programmed ventricular stimulation at RVOT.
2. The diagram shows the electrocardiogram (ECG) waveforms from different leads.
3. Leads I, II, aVf, V1, V5, RVA1-2, RVA3-4, RVOT1-2, RVOT3-4, and Ao are displayed.
4. The time scales are marked at 500 ms.
5. The intervals between the S waves are indicated as 600, 270, 220, 220 ms.
Programmed ventricular stimulation (PVS) induced two ventricular responses but failed to induce PVT.
ICD implantation

DFT: 20J B>AX

VF
Because of frequent development of PVT/VF associated with attacks of vasospastic angina, he was implanted ICD.
Comments on PVT in vasospastic angina-1

• We studied Holter ECG recordings in 60 consecutive patients with vasospastic angina (Jpn Circ J 2001;65:519-525). Eight patients had at least one episode of PVT during Holter monitorings and remaining 52 were free of PVT.

• Ischemic ST segment elevation preceded the development of PVT in all 8 cases and 4 had silent coronary spasm. The onset of PVT was initiated with R-on-T, long-short sequence or ST wave alternans in 6 of 8 cases.
During a follow-up of 73 ± 17 months, a high incidence of sudden death (2/8 cases; 25 %) in PVT group was observed, while no death was found in 52 cases of non-PVT group.

Two cases of sudden death victims showed atrial fibrillation (AF) in baseline rhythm. Therefore, AF may carry a high risk in patients with vasospastic angina.

(Following two slides summarize the results of this study)
Plymorphic ventricular tachycardia in patients with vasospastic angina

-Clinical and electrocardiographic characteristics and long-term outcome-

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (years)</th>
<th>Date</th>
<th>Dominant rhythm</th>
<th>QT (ms)</th>
<th>QTC (ms)</th>
<th>Result of coronary arteriography</th>
<th>Site of coronary spasm</th>
<th>Angina</th>
<th>ST elevation</th>
<th>Interval from ST elevation to onset of PVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36/M</td>
<td>Jul. 91</td>
<td>Sinus</td>
<td>320</td>
<td>410</td>
<td>Normal</td>
<td>LAD(6,8), LCx(12)</td>
<td>+</td>
<td>+</td>
<td>60 s</td>
</tr>
<tr>
<td>2</td>
<td>73/M</td>
<td>Nov. 92</td>
<td>AF</td>
<td>340</td>
<td></td>
<td>Normal</td>
<td>LAD(6)</td>
<td></td>
<td>+</td>
<td>1 s</td>
</tr>
<tr>
<td>3</td>
<td>62/M</td>
<td>Apr. 92</td>
<td>Sinus</td>
<td>360</td>
<td>440</td>
<td>Normal</td>
<td>LAD(6)</td>
<td>-</td>
<td>+</td>
<td>105 s</td>
</tr>
<tr>
<td>4</td>
<td>60/M</td>
<td>Oct. 93</td>
<td>Sinus</td>
<td>380</td>
<td>414</td>
<td>Normal</td>
<td>LAD(6)</td>
<td>+</td>
<td>+</td>
<td>135 s</td>
</tr>
<tr>
<td>5</td>
<td>67/M</td>
<td>Apr. 93</td>
<td>Sinus</td>
<td>380</td>
<td>380</td>
<td>Normal</td>
<td>LAD(6)</td>
<td>-</td>
<td>+</td>
<td>180 s</td>
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<tr>
<td>6</td>
<td>52/M</td>
<td>Apr. 93</td>
<td>Sinus</td>
<td>420</td>
<td>400</td>
<td>Normal</td>
<td>LAD(8), LCx(13), RCA(2)</td>
<td>+</td>
<td>+</td>
<td>165 s</td>
</tr>
<tr>
<td>7</td>
<td>60/M</td>
<td>Nov. 94</td>
<td>AF</td>
<td>400</td>
<td></td>
<td>Normal</td>
<td>LAD(6)</td>
<td>-</td>
<td>+</td>
<td>75 s</td>
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<tr>
<td>8</td>
<td>44/M</td>
<td>Sep. 95</td>
<td>Sinus</td>
<td>400</td>
<td>417</td>
<td>Normal</td>
<td>LAD(7)</td>
<td>-</td>
<td>+</td>
<td>105 s</td>
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Jpn Cir J, 2001; 65: 519-525
## PVT in vasospastic angina -2

<table>
<thead>
<tr>
<th>R on T</th>
<th>Long–short sequence</th>
<th>T wave alternans</th>
<th>Tdp</th>
<th>Follow-up period (months)</th>
<th>Medication</th>
<th>Outcome</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>95</td>
<td>Diltiazem 90 mg, nicorandil 15 mg</td>
<td>Survived</td>
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<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Nifedipine 40 mg, ISDN 60 mg</td>
<td>Died at 5 months</td>
<td>VF</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>86</td>
<td>Nifedipine 40 mg, ISDN 60 mg</td>
<td>Survived</td>
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<tr>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>68</td>
<td>Diltiazem 90 mg, ISDN 60 mg</td>
<td>Survived</td>
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<tr>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>74</td>
<td>Diltiazem 120 mg, ISDN 60 mg, nicorandil 15 mg</td>
<td>Survived</td>
<td></td>
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<tr>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>74</td>
<td>Diltiazem 90 mg, nifedipine 40 mg, ISDN 80 mg</td>
<td>Survived</td>
<td></td>
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<tr>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>45</td>
<td>Diltiazem 90 mg, ISDN 60 mg</td>
<td>Died at 18 months</td>
<td>VF</td>
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<tr>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>45</td>
<td>Diltiazem 120 mg</td>
<td>Survived</td>
<td></td>
</tr>
</tbody>
</table>
Increased ventricular vulnerability

Hyperinsulinemia or insulin resistance

Increased inhomogeneity of ventricular repolarization (QT dispersion)

Coronary vasoconstriction (Subclinical ischemia)

Myocardial ischemia (Silent ischemia)

Sudden death
Our proposed schema contributing to PVT/VF (Fatal ventricular arrhythmia) and SCD.
Numbers indicate references cited in next slide.
References

- ① J Am Coll Cardiol, 1996; 27:1458-63
- ③ Am J Cardiol, 1999; 84:807-810
- ④ Am J Cardiol, 1996;77:355-360
- ⑤ Jpn Cir J, 2001; 65: 519-525