Evaluation of Patients with VT storm

Kyoung-Min Park, MD
Samsung Medical Center, Electrophysiology Program
Kyoungmin.park@samsung.com
Ventricular Electrical storms

- Definition; 
  ≥ 3 episodes of VT/VF or appropriate ICD shock/ 24-hrs

- Incidence; 10-28% of ICD patients – secondary prevention
  4% in MADIT II study – primary prevention

- Triggers and Risk factors;
  - electrolyte imbalance; K⁺, Mg²⁺↓
  - acute coronary syndrome
  - worsening heart failure
  - severely compromised ventricular function, chronic renal insufficiency and VT

From Bansch et al., Circulation 2003
MADIT II: Reduced Survival after Shocks

<table>
<thead>
<tr>
<th>Episode Type</th>
<th>Therapy Type</th>
<th>Hazard Ratio</th>
<th>Risk of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT Shocked</td>
<td></td>
<td>3.4 (1.9, 5.9), p&lt;0.001</td>
<td>≈3.4 times increase</td>
</tr>
<tr>
<td>VF Shocked</td>
<td></td>
<td>3.3 (1.3, 8.1), p=0.01</td>
<td>≈3.3 times increase</td>
</tr>
</tbody>
</table>

TABLE 2. Transient and Lingering Risk of Death: Multivariate Results

<table>
<thead>
<tr>
<th>Variable</th>
<th>RR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time-dependent</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transient (3-month) risk</td>
<td>5.4 (2.4 to 12.4)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Residual (lingering) risk</td>
<td>1.9 (1.0 to 3.5)</td>
<td>0.04</td>
</tr>
<tr>
<td>Antiarrhythmic drug use</td>
<td>1.2 (0.8 to 2.1)</td>
<td>0.4</td>
</tr>
<tr>
<td>Other VT/VF</td>
<td>1.0 (0.6 to 1.6)</td>
<td>0.9</td>
</tr>
<tr>
<td><strong>Baseline</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (per decade)</td>
<td>1.7 (1.4 to 2.2)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2.0 (1.3 to 3.0)</td>
<td>0.0009</td>
</tr>
<tr>
<td>Ejection fraction (per 0.1)</td>
<td>0.7 (0.6 to 0.9)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Male sex</td>
<td>0.8 (0.5 to 1.3)</td>
<td>0.3</td>
</tr>
<tr>
<td>History of heart failure</td>
<td>1.2 (0.8 to 1.8)</td>
<td>0.5</td>
</tr>
<tr>
<td>VF index arrhythmia</td>
<td>1.4 (0.9 to 2.1)</td>
<td>0.1</td>
</tr>
</tbody>
</table>

**Window for Intervention**

Because the risk of death is most prominent in the initial 3 months after electrical storm, prompt implementation or augmentation of therapies previously demonstrated to be efficacious in similar populations seems warranted. ACE inhibitors improve outcome in patients with left ventricular dysfunction, primarily via a reduction in heart failure progression. β-Blockers further reduce mortality in these patients through reductions in sudden death and heart failure progression. β-Blockers may be particularly effective in patients with electrical storm. Spironolactone has also been demonstrated to reduce mortality in patients with heart failure via a reduction heart failure progression. Coronary artery revascularization seems to be an important adjunct in patients with ischemic left ventricular dysfunction, both in terms of reducing the risk of future arrhythmias and in optimizing ventricular function. Finally, because electrical storm may be an inciting rather than a contributing factor for premature death, therapies specifically aimed at reducing recurrent arrhythmias may prove useful. Amiodarone and other antiarrhythmic drugs are often initiated in these patients, but there are insufficient prospective data to support or refute this approach.
Pathophysiology of VT storm

Clinical conditions that can lead to recurrent ventricular arrhythmias and electrical storm

Predisposing clinical conditions

Genetic conditions
- BrS / ERS
  - Early repolarization
  - Altered conduction
- LQTs
  - ↓ Repolarization reserve
  - Ca²⁺-handling abnormalities
  - EADs
- CPVT
  - Ca²⁺-handling abnormalities
  - DADs

Acquired conditions
- Hypertrophy / HF
  - ↓ Repolarization reserve
  - Ca²⁺-handling abnormalities
  - Altered excitability / conduction
  - EADs / DADs
- Animals
  - EADs / DADs

Arrhythmogenic Mechanisms
- Reentry

Recurrent Ventricular Arrhythmias

Defibrillation

Experimental models

Initiating factors
- Electrolyte imbalance
- Sympathetic tone
- Ischemia
Classification of VT storm patterns

- Reentrant, scar related sustained monomorphic VT (SMVT)

- Polymorphic VT (PMVT) triggered by PVC
Tachyarrhythmias – VT storm

• Monomorphic VT
  – Reentry around scar, typically from old myocardial infarction
  – Slow conduction around a fixed path, with recovery of myocytes before the signal reaches the start again
  – QRS morphology is determined by the location of the circuit and where the signal escapes to the rest of the ventricular tissue
Tachyarrhythmias – VT storm

Photo courtesy of Dr. William Stevenson
Tachyarrhythmias – VT storm

• Monomorphic VT
  – Reentry around scar, typically from old myocardial infarction
  – Slow conduction around a fixed path, with recovery of myocytes before the signal reaches the start again
  – QRS morphology is determined by the location of the circuit and where the signal escapes to the rest of the ventricular tissue
Tachyarrhythmias – VT storm

• Polymorphic VT
  – Changing wavefront, typically seen in ischemic myocardium
  – Electrical properties of substrate may be changing
  – More urgent than monomorphic VT both because less tolerated hemodynamically, and likely active problem with myocardium that needs to be immediately addressed
Tachyarrhythmias – VT storm

• What is the cause? (Why now?)
  – Ischemia
  – Heart failure (with increased catechols)
  – Metabolic (low K\(^+\), low Mg\(^{++}\), thyroid storm)
  – Proarrhythmic effect of new medication
  – New cardiac substrate (ectopy or more favorable path for reentry)
Tachyarrhythmias – VT storm

• Management - 1
  – IV Antiarrhythmics
    • Lidocaine
    • Amiodarone
    • Procainamide
  – Electrolyte repletion
    • Can give empiric magnesium
  – Beta blockade
    • IV esmolol, metoprolol, propranolol
    • Sympathetic ganglion block

Nademaneec, Circ 2000
Tachyarrhythmias – VT storm

• Management - 2
  – Sedation (works by reducing catechols)
    • Benzodiazepines, propofol Burjorjee, Can J Anesth 2002
    • General anesthesia with intubation
  – Fix ischemia (? Revascularization)
  – Heart failure management
  – Stop offending medication
  – Emergency VT ablation (rarely needed)
    • Target reentrant substrate
    • Target ectopic beats that initiate VT/VF

Bansch, Circ 2003; Marrouche JACC 2004
Case 1. M/65 Electrical storm

A

B

C

D

Na⁺ 134
K⁺ 4.1
VF storm in patients with ICM

- Triggered by monomorphic PB, driven by the purkinje-like potential from scar border zone
- Ablation this trigger point, prevent recurrence of future VF

Natale et al. JACC 2004;43:1715-20
Emergency?
Tachyarrhythmias – VT storm

- Bidirectional VT
  - Digoxin toxicity
  - Enhanced automaticity or triggered activity of Purkinje fibers
  - Treatment is digoxin antibody (Digibind)
Tachyarrhythmias – Torsades
Tachyarrhythmias – Torsades de Pointes

• Mechanism of Torsades de Pointes
  – “R on T” phenomenon from early afterdepolarization
  – Twisting reentry related to slowed conduction from new depolarization prior to previous completed repolarization (during QT interval)
Tachyarrhythmias – Torsades

• Seen with long QT interval
  – Hypokalemia, hypomagnesemia
  – Bradycardia

• Drug-related
  • Antiarrhythmics – Class III, Class Ia
  • Phenothiazines – e.g. Haldol, Droperidol
  • Others – e.g. Erythromycin, Cisapride, Seldane

• Intracranial disease, myocardial infarction
• Inherited syndromes
Tachyarrhythmias – Torsades

- Treatment
  - Replete potassium, magnesium
  - Empiric magnesium
  - Stop offending agent(s)
  - Pacing or isoproterenol to increase heart rate and shorten QT interval
Case 2. 48/F dilated cardiomyopathy

- Dec 2017  Acute heart failure → CPR due to VF arrest
- 17.12.20  IABP insertion, but recurrent VT; reduced LVEF (20%) → refer to SMC
- 17.12.22  IABP removal after stabilization
Baseline ECG

Recurrent VT

Long QT interval with R on T phenomenon
Management

17.12.23  CAG: no significant stenosis

ECMO insertion

Norepinephrine, amiodarone → lidocaine infusion

K and Mg replacement to upper normal range

17.12.26  isoproterenol infusion due to bradycardia
TPM insertion
ECMO insertion & CAG

Recurrent VT

Potassium & Mg replacement
Amiodarone $\rightarrow$ lidocaine continuous infusion
Isoproterenol infusion

TPM insertion

Elevated basal HR
Proposed algorithm of the management of patients presenting with VT storm

1. Haemodynamically stable
   - ICD reprogramming
   - Sedation

2. Haemodynamically unstable
   - Deep sedation/intubation
   - ICD reprogramming

3. Remain unstable
   - Search and treat triggers:
     - i.e. electrolyte imbalances, ischaemia, heart failure etc.

4. Anti-arrhythmic agents:
   - B-blockers, Amiodarone, Sotalol
   - Class 1 anti-arrhythmic agents

5. Ongoing ventricular arrhythmia storm
   - Catheter ablation
   - Temporarily consider discontinuing CRT
   - Consider novel treatment-
     - isoproterenol in VA storm associated with
     - Brugada syndrome & idiopathic VF

Ventricular electrical storm is an important independent predictor of poor prognosis.

Try to find and correct triggers and risk factors like hypokalemia, acute coronary syndrome, worsening heart failure, chronic renal insufficiency to reverse the storm.

RF ablation is the best method to prevent recurrence of VT storm which is unresponsive to correction of reversible causes.

Take Home Messages