VT epizodu olan AFaÜÇ xəstəsinin farmakolojik təqibə

Dr. Ceyhun Umudov
**AFaÜÇ (HFrEF)**

<table>
<thead>
<tr>
<th>Type of HF</th>
<th>HFrEF</th>
<th>HFmrEF</th>
<th>HFpEF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Symptoms ± Signs(^a)</td>
<td>Symptoms ± Signs(^a)</td>
<td>Symptoms ± Signs(^a)</td>
</tr>
<tr>
<td>2</td>
<td>LVEF (\leq 40)%</td>
<td>LVEF 41 – 49(^b)</td>
<td>LVEF (\geq 50)%</td>
</tr>
<tr>
<td>3</td>
<td>–</td>
<td>–</td>
<td>Objective evidence of cardiac structural and/or functional abnormalities consistent with the presence of LV diastolic</td>
</tr>
</tbody>
</table>

Before disease-modifying therapies became available, the incidence of SCD in patients with HFrEF was higher than 20% per year \(^{10}\), nevertheless with current pharmacologic and electric therapy, the incidence of SCD has decreased to about 3% per year \(^{11}\).

Currently, SCD accounts for about 40% to 45% of all deaths in HFrEF patients, and the proportion of SCD is higher in patients with milder symptoms (New York Heart Association (NYHA) class II-III) \(^{12}\), indeed two-thirds of patients with NYHA functional class II, experience SCD, compared with only one-third of those with NYHA functional class IV symptoms, who died preponderantly for advanced HF \(^{13}\).

<table>
<thead>
<tr>
<th>Signs of HFpEF</th>
<th>Signs of HFmrEF</th>
<th>Signs of HFrEF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced exercise tolerance</td>
<td>Laterally displaced apical impulse</td>
<td>Tachypnoea, Cheyne-Stokes respiration, Hepatomegaly, Ascites, Cold extremities, Oliguria, Narrow pulse pressure</td>
</tr>
<tr>
<td>Fatigue, tiredness, increased time to recover after exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankle swelling</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
VT patofiziologiyası:

1. **Enhanced normal automaticity**
   (Sinus tachycardia)

2. **Abnormal automaticity***
   (Most Ischemic VT, Catecholamine sensitive)

- **Early after depolarisation (EAD)***
  - VPDs/VT due to brady dependent/Hypoxia/Acidosis/hypokalemia/
  - & Ion channel defect

- **Delayed after depolarisation (DAD)**
  - Digoxin related

**Re-entry**

- **Macro-re-entry** (Commonest) Scar mediated VT /OTVTs/BBR/Fasc.VT/Atrial flutters)
- **Micro-re-entry** (Ischemic /Brugada)
Re-entry:

Excitable gap

Functional block
Intravenous verapamil is not recommended in broad QRS complex tachycardia of unknown mechanism.\textsuperscript{308,309}
Randomized comparison of intravenous procainamide vs. intravenous amiodarone for the acute treatment of tolerated wide QRS tachycardia: the PROCAMIO study

Mercedes Ortiz¹†, Alfonso Martín², Fernando Arribas³, Blanca Coll-Vinent⁴, Carmen del Arco⁵, Rafael Peinado⁶ and Jesús Almendral¹∗†, on Behalf of the PROCAMIO Study Investigators

In this randomized prospective study comparing intravenous procainamide and amiodarone for the treatment of the acute episode of sustained monomorphic well-tolerated wide QRS tachycardia (probably VT), procainamide therapy was associated with less major cardiac adverse events and a higher proportion of tachycardia termination within 40 min.
In patients with heart failure with reduced ejection fraction (HFrEF), the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure recommend angiotensin-converting enzyme inhibitor (ACE-I)/angiotensin receptor blocker (ARB)/angiotensin receptor neprilysin inhibitors (ARNIs), mineralocorticoid receptor antagonists (MRAs), beta-blockers, and sodium-glucose co-transporter 2 (SGLT2) inhibitors to reduce mortality due to heart failure and SCD.
**Conclusions:** In a nonrestricted STEMI population, early intravenous metoprolol before PCI was not associated with a reduction in infarct size. Metoprolol reduced the incidence of malignant arrhythmias in the acute phase and was not associated with an increase in adverse events.

Prophylactic treatment with AADs (other than beta-blockers) is not recommended in ACS.
Comparison of β-Blockers, Amiodarone Plus β-Blockers, or Sotalol for Prevention of Shocks From Implantable Cardioverter Defibrillators
The OPTIC Study: A Randomized Trial

Results  Shocks occurred in 41 patients (38.5%) assigned to β-blocker alone, 26 (24.3%) assigned to sotalol, and 12 (10.3%) assigned to amiodarone plus β-blocker. A reduction in shocks was noted in patients assigned to amiodarone plus β-blocker or sotalol compared to β-blocker alone. Adverse pulmonary and thyroid events and symptomatic bradycardia were more common among patients randomized to amiodarone.

The addition of oral amiodarone or beta-blocker replacement by sotalol should be considered in patients with CAD with recurrent, symptomatic SMVT, or ICD shocks for SMVT while on beta-blocker treatment.\textsuperscript{318,581}

Conclusions  Despite use of advanced ICD technology and treatment with a β-blocker, shocks occur commonly in the first year after ICD implant. Amiodarone plus β-blocker is effective for preventing these shocks and is more effective than sotalol but has an increased risk of drug-related adverse effects.
A PVC burden of at least 10% appears to be the minimal threshold for development of PVC-induced cardiomyopathy, and the risk increases further with a PVC burden >20%. In patients with a PVC burden <10%, other cardiomyopathy aetiologies should be suspected and further diagnostic work-up undertaken.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Ablation</th>
<th>Beta-blocker</th>
<th>CCB</th>
<th>Flecaïnide</th>
<th>Amiodarone</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVOT/fascicular PVC/VT: Symptomatic, normal LV function</td>
<td>Class I</td>
<td>Class IIa</td>
<td>Class IIa</td>
<td>Class IIa</td>
<td>Class III</td>
</tr>
<tr>
<td>PVC/VT other than RVOT/fascicular: Symptomatic, normal LV function</td>
<td>Class IIa</td>
<td>Class I</td>
<td>Class I</td>
<td>Class IIa</td>
<td>Class III</td>
</tr>
<tr>
<td>RVOT/fascicular PVC/VT: LV dysfunction</td>
<td>Class I</td>
<td>Class IIa</td>
<td>Class IIIa</td>
<td>Class IIa</td>
<td>Class IIa</td>
</tr>
<tr>
<td>PVC/VT other than RVOT/fascicular: LV dysfunction</td>
<td>Class I</td>
<td>Class IIa</td>
<td>Class IIIa</td>
<td>Class IIa</td>
<td>Class IIa</td>
</tr>
<tr>
<td>PVC: Burden &gt;20%, asymptomatic, normal LV function</td>
<td>Class IIb</td>
<td></td>
<td></td>
<td></td>
<td>Class III</td>
</tr>
</tbody>
</table>
Positive genetic testing for LMNA mutations has crucial clinical and prognostic implications. Mortality in patients with LMNA-CMP is estimated to be 40% at 5 years (Pasotti et al., 2008), whereas 45% suffered SCD or aborted SCD.
The addition of oral amiodarone or replacement of beta-blockers by sotalol should be considered in patients with DCM/HNDCM and an ICD who experience recurrent, symptomatic VA despite optimal device programming and beta-blocker treatment.
Among definite/probable ARVC patients considered at high risk for VA, 23–48% will experience appropriate ICD intervention during a mean follow-up of 4.7 years. In 16–19% of cases, ICD intervention is triggered by fast VT ≥250 b.p.m. or VF, which is considered as surrogate for a life-threatening event. In a large cohort of 864 ARVC patients (38.8% with a prior VA), 43% had VT/VF during a median follow-up of 5.75 years, but only 10.8% a potentially life-threatening event. Thus, in 3 out of 4 ARVC patients, ICD therapy is appropriate but may not be considered acutely life-saving.
Beta-blocker therapy may be considered in all patients with a definite diagnosis of ARVC.

Data on AADs to prevent VT recurrence are limited to small observational studies and registries. In general, AAD therapy has limited efficacy. Although sotalol was effective to prevent inducibility of VT, it did not suppress clinically relevant arrhythmias. Treatment with amiodarone or class 1 drugs was associated with a trend to lower VT recurrence as compared with sotalol. The addition of flecainide to beta-blockers/sotalol was beneficial in a small cohort.

In patients with ARVC and recurrent, symptomatic VT despite beta-blockers, AAD treatment should be considered.
Miokarditdə VT epizodunun müalicəsi:

Sustained VAs may occur in acute myocarditis. In a large series of patients, in-hospital VF or CA was reported in 2.5% of cases. In the majority of acute myocarditis, children younger than 1 year old.

AADs should be considered (preferably amiodarone and beta-blockers) in patients with symptomatic non-sustained or sustained VAs during the acute phase of myocarditis.

In post-myocarditis patients with recurrent, symptomatic VT, AAD treatment should be considered.

patients with sustained VAs during the acute phase of myocarditis (LVEF 53 ± 10%) had a high risk (45% at 3 years) of VT/VF recurrences during follow-up.
Unexpected SCD is an important characteristic and outcome of CS (*Take home figure*). It accounts for 14% of the presenting manifestations and as many as 80% of all fatalities in CS. Furthermore, nearly two-thirds of all deaths caused by CS occur suddenly in individuals with undiagnosed sarcoid granulomas in the heart. Of patients in whom CS causes symptoms during life, 85% can be expected to live beyond 5 years and 76% beyond 10 years from symptom onset. For patients receiving immunosuppressive and device therapy, the 5- and 10-year survival estimates are 93% and 87%, respectively.
Effect of Corticosteroid Therapy on Ventricular Arrhythmias in Patients with Cardiac Sarcoidosis

Kenji Yodogawa, M.D.,* Yoshihiko Seino, M.D.,* Toshihiko Ohara, M.D.,† Hideo Takayama, M.D.,† Takao Katoh, M.D.,† and Kyoichi Mizuno, M.D.†

From the *Division of Cardiology, Department of Internal Medicine, Nippon Medical School Chiba Hokusho Hospital, Chiba, Japan; †Division of Cardiology, Hepatology, Geriatrics, and Integrated Medicine, Department of Internal Medicine, Nippon Medical School, Tokyo, Japan

Results: As a whole, there were no significant differences in the number of PVCs and in the prevalence of NSVT before and after steroid therapy. However, the less advanced LV dysfunction patients (EF ≥ 35%, n = 17) showed significant reduction in the number of PVCs (from 1820 ± 2969 to 742 ± 1425, P = 0.048) and in the prevalence of NSVT (from 41 to 6%, p = 0.039). Late potentials on SAECG were abolished in 3 patients. The less advanced LV dysfunction group showed a significantly higher prevalence of gallium-67 uptake compared with the advanced LV dysfunction group (EF < 35%, n = 14). In the advanced LV dysfunction patients, there were no significant differences in these parameters.

Conclusions: Corticosteroid therapy may be effective for ventricular arrhythmias in the early stage, but less effective in the late stage.
Anti-aritmik dərmanlar:

<table>
<thead>
<tr>
<th>Anti-arrhythmic drug</th>
<th>Effects on ECG</th>
<th>Indications (specific indication)</th>
<th>Oral dose per day (i.v. dose)</th>
<th>Side effects</th>
<th>Contraindications, precautions, other considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Decreases sinus node frequency, prolongs QT interval(a)</td>
<td>PVC, VT, VF</td>
<td><strong>200–400 mg</strong>&lt;br&gt;Loading dose: 600–1200 mg/24 h 8–10 days.&lt;br&gt;(Loading dose: 5 mg/kg in 20 min–2 h. 2–3)</td>
<td><strong>Cardiac:</strong> Bradycardia, TdP (infrequent)&lt;br&gt;<strong>Extracardiac:</strong> Photosensitivity, corneal deposits, hypothryoidism, hyperthyroidism,</td>
<td><strong>Precautions:</strong> Sinus node dysfunction, severe AV conduction disturbances, hyperthyroidism&lt;br&gt;<strong>Other considerations:</strong> Can be used in patients with...</td>
</tr>
</tbody>
</table>

Until now, no AAD except for beta-blockers has demonstrated reduction in all-cause mortality. Each drug has a significant potential for causing adverse events, including pro-arrhythmia.

| Sotalol | Decreases sinus node frequency, prolongs QT interval\(a\) | VT | **160–640 mg**<br>(0.5–1.5 mg/kg in 10 min. If necessary, can be repeated after 6 h) | See beta-blockers, TdP\(^d\) (<2% of patients, close monitoring of QT interval and CrCl) | **Contraindications:** Severe sinus node dysfunction, severe AV conduction disturbances, severe heart failure with reduced LVEF, significant LVH, CrCl <30 ml/min, coronary vasospasm, LQTS<br>**Precautions:** Concomitant treatments associated with QT interval prolongation, hypokalaemia<br>**Precautions:** Discontinue if QRS widening >25% or bundle branch block |
Patient requiring a drug associated with QT prolongation

Assessment of patient risk profile before initiation

Long QT syndrome or baseline QTc >500 ms

- Y: Contraindicated: Do not initiate treatment
- N: Hypokalaemia/hypomagnesaemia/Renal failure/liver failure/LV hypertrophy/Heart failure/Additional QT prolonging drug

- Y: Start drug if strong indication with close follow-up
- N: Initiate treatment

ECG at baseline, after 1 day and after 1–2 weeks of initiation or increase in dosage

- Y: QTc >500 ms
  - Y: Consider to change to different drug
    - If strong indication, consider to reduce dose
    - Consider to assess drug serum level
  - N: Continue treatment
    - Inform patient about warning symptoms (syncope)
    - Control changes in renal and hepatic function
    - Be aware of potential drug interactions
- N: Continue treatment
Reference:

- A Companion to Braunwald Textbook of Cardiovascular Medicine-2018 (11th) Douglas P. Zipes MD, Peter Libby MD PhD, Robert O. Bonow MD MS, Douglas L. Mann MD
- Manual of Cardiovascular Medicine-2019 (5th) Brian P. Griffin
- Mayo Clinic Cardiology-Oxford University Press (2012)
- 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death
- 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy
- Companion to Braunwald’s Heart Disease-G. Michael Felker, Douglas Mann - Heart Failure-Elsevier (2019)
Rhythm of Life