EFFECTS OF CILOSTAZOL ON THE CANINE RV WEDGE MODEL OF BRUGADA SYNDROME

Poster Contributions
Hall C
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Background: To date there is no recommendation of oral drugs that can be used for effective prevention of ventricular fibrillation (VF) in patients with Brugada syndrome (BS). Cilostazol, an oral phosphodiesterase type III inhibitor, is reported in cases to prevent VF in patients with BS, however, the underlying mechanism is poorly understood. We used arterially-perfused RV wedge preparations to investigate the electrophysiological effect of Cilostazol in a BS model.

Methods: BS model was created on 16 arterially-perfused canine RV wedge preparations. Pinacidil at 2ummol/L, Terfenadine at 5ummol/L and Pilsicainide at 5ummol/L were simultaneously perfused. Once BS model stabilized, the 16 preparations were divided into 2 groups: in one group, perfusion was continued with the aforementioned drugs (Control group); in another group, Cilostazol at 10ummol/L was added (Cilostazol group). Transmembrane action potential was recorded from epicardium and endocardium of the wedge preparation, as well as pseudo-ECG. Program stimulation was performed. Spontaneous or induced arrhythmia events were registered.

Results: In arterially-perfused canine RV wedge BS model, Cilostazol corrected J point elevation, decreased transmural and epicardial regional dispersion of repolarization, and was associated with less arrhythmia events (Table).

Conclusion: Cilostazol may prevent VF in BS by offsetting the electrophysiological abnormalities, especially the widened transmural dispersion of repolarization.

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Cilostazol Group</th>
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<tbody>
<tr>
<td>No. of preparations</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>QT (ms)</td>
<td>228±12</td>
<td>274±18</td>
</tr>
<tr>
<td>J point elevation (mv)</td>
<td>0.48±0.12</td>
<td>0.18±0.08</td>
</tr>
<tr>
<td>Transmural dispersion of repolarization (ms)</td>
<td>70±16</td>
<td>46±18</td>
</tr>
<tr>
<td>Epicardial regional dispersion of repolarization (ms)</td>
<td>76±18</td>
<td>32±16</td>
</tr>
<tr>
<td>Endocardial regional dispersion of repolarization (ms)</td>
<td>44±16</td>
<td>30±16</td>
</tr>
<tr>
<td>VT/VF incidence (episode)</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td>Endocardial ERP (ms)</td>
<td>155±10</td>
<td>175±10</td>
</tr>
<tr>
<td>Epicardial ERP (ms)</td>
<td>150±10</td>
<td>170±10</td>
</tr>
<tr>
<td>Dispersion of ERP (ms)</td>
<td>5±10</td>
<td>5±10</td>
</tr>
</tbody>
</table>

①: P<0.05; ERP: effective refractory period