

Similarities between early repolarization Syndrome and Brugada syndrome - 2021

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1. **Sex:** Both are much more frequent in the male gender; consequence of testosterone modulation of ion currents underlying the epicardial AP notch;
2. **Age:** Both are predominantly observed in young adults (between 30-50 yo);
3. Both do not have **apparent or macroscopic** Structural Heart Disease (SHD)
4. Both have high electrocardiographic dynamicity of repolarization;
5. Both frequently present conduction disturbances in the His system;
6. Both may present discrete QRS interval widening.
7. Both may display saddleback appearance of repolarization;
8. Both may reverse ventricular repolarization pattern during stress test;
9. Both improve ventricular repolarization with IV isoproterenol, probably because the drug reduces repolarization dispersion which triggers VF events;

10. Both have a shortening of phase 2 action potential due to electrophysiological substrate, in the ventricular epicardium thickness by intensification of the notch in phase 1, mediated by the I_{to} channel;
11. Both may have modification in the I_{to} and $I_{Ca^{++-L}}$ channels by electrophysiological substrate, which explains the J point and ST segment elevation causing intensification of the notch in phase 1 and decrease in phase 2 duration in the ventricular epicardium thickness;
12. Both may affect in different degrees, ventricular repolarization in the right precordial leads as well as in the lateral wall (V4-V6) and inferior leads I, II, and III (atypical forms of BrS);
13. Both may have vagally mediated accentuation of ECG pattern;
14. Both have VF events often during sleep or at a low level of physical activity caused by higher level of vagal tone and higher levels of I_{to} at the slower heart rates;
16. Both have VT/VF triggered by short-coupled PVCs by phase 2 reentry mechanism;
17. Both have ameliorative response to quinidine and bepridil by inhibition of I_{to} and possible vagolytic effect;
18. Both have ameliorative response to isoproterenol, denopamine, milrinone and cilostazol by increased $I_{Ca^{++}}$ and faster heart rate;

20. Both have ameliorative response to pacing by reduced availability of I_{to} due to slow recovery from inactivation;
21. Both have vagally mediated accentuation of ECG pattern by direct effect to inhibit $I_{Ca^{++}}$ and indirect effect to increase I_{to} (due to slowing of heart rate);
22. Both have augmented J waves high by accelerated inactivation of I_{Na} and accelerated recovery of I_{to} from inactivation;
23. Both have augmented J waves by slowed activation of I_{Ca} , leaving I_{to} unopposed. Increased phase 2 reentry but reduced pVT due to prolongation of APD.