Conus Branch Ischemia Provokes Brugada-Type ST-Segment Changes in Patients With Coronary Artery Disease

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Recently, the intriguing coexistence of vasospastic angina and Brugada syndrome have been reported in some cases. However, the mechanistic relationship between Brugada type ECG changes and ischemia of the right ventricular outflow tract has yet to be fully elucidated. Here we present four cases with coronary artery disease who presented with ECG abnormalities induced by ischemia of the conus branch (CB) of the right coronary artery. The twelve-lead ECGs at rest showed normal sinus rhythm in all cases. Surprisingly, the salla-back ST segment elevation in leads V1-V3 was observed only during the percutaneous transluminal angioplasty procedure of the proximal right coronary artery or with an intracoronary ergonovine injection into the right coronary artery. The coronary angiography exhibited a total occlusion or 99% stenosis with a delay in the CB and significant stenosis of the proximal right coronary artery in three cases and coronary vasospasms of the CB and right coronary artery in the remaining case. These Brugada-type ECG changes reversibly disappeared after cessation of the ischemia from the CB lesion. In the fourth patient who had vasospasms of the right coronary artery, remarkable coved-type ST segment elevation was also observed during the ST elevation in right precordial leads with class I antiarrhythmic drug administration: pilsicainide, suggesting a concealed form of Brugada syndrome. These results suggest that the free wall of the right ventricular outflow tract supplied by the CB plays a pivotal role in the genesis of the Brugada-type ECG changes, and the ischemia from CB lesions, at least in part, contributes to the Brugada-type ST segment changes.

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Background: Brugada syndrome is characterized by a right bundle branch block pattern and ST-segment elevation in the right precordial leads. The Brugada-type electrocardiographic (ECG) pattern can be observed in asymptomatic healthy patients (pts) or conditions other than true Brugada syndrome. The mechanisms of Brugada-type ECG pattern are not fully understood.

Methods: We performed percutaneous coronary intervention (PCI) of proximal right coronary artery (RCA) for ischemic heart disease in 12 pts. No significant stenosis was observed in left coronary arteries in all pts. The ST changes were evaluated during PCI. Angiographical changes were carefully observed from major branches of RCA to small branches during PCI. We measured ST elevation in right precordial leads with class I antiarrhythmic drug administration: pilsicainide 50 mg (pure sodium channel blocker) after PCI. ST changes were compared with true Brugada syndrome (n=5).

Results: Brugada-type ECG was observed in 9 pts (42%) but not in 7 pts (32%) during PCI of proximal RCA. All 8 pts who had Brugada-type ECG demonstrated ST segment alternans from coved shape to saddle back shape during PCI. These ECG changes returned to normal after PCI. These pts who had Brugada-type ECG with ST alternans demonstrated selective small RV branch occlusion or vasospasm during PCI, which per- fured RV anterior or RV outflow. However, pts without Brugada-type ECG did not show RV branch occlusion. The class I antiarrhythmic drug administration showed no significant ST elevation both in pts with Brugada-type ECG and in pts without Brugada-type ECG (0.70±0.54mm vs 0.46±0.21mm, p=NS). There was a significant difference in ST elevation between pts with Brugada-type ECG during PCI and pts with true Brugada syn- drome. (0.70±0.54mm vs 2.86±0.61mm, p<0.05)

Conclusions: The pts who had RV branch occlusion during PCI showed Brugada-type ECG and ST alternans. Sodium channel impairment was not associated with these ECG changes. ST alternans demonstrated as prerequisites before developing ventricu- lar arrhythmia. These data suggest that merely ischemia of small RV branch could be one of the different entities showing Brugada-type ECG from true Brugada syndrome.