中文題目：冠狀動脈痙攣引起類似 Brugada 症候群心電圖和心室頻脈

英文題目：Coronary Artery Spasm Induced Brugada pattern ECGs and Ventricular Arrhythmias

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Background: The coronary artery vasospasm and inherited primary arrhythmia syndromes both induced ventricular arrhythmias and sudden cardiac death. These diseases could be coexistent. Here we reported a patient manifested with coronary artery spasm and Brugada-like ST-segment elevation ECG pattern. The differentiation between Brugada syndrome and coronary artery spasm would bring out significant differences of diagnosis and was critical for tailoring treatment strategies.

Methods: Not applicable

Results: A 56-year-old man presented to the clinics because of recurrent syncope without prodrome since one year ago. Upward gazing with myoclonus movement was associated. Since then, frequent hospital visits and tests did not come up with any diagnosis. Echocardiography showed grossly normal heart. An electrocardiogram reportedly showed only first degree AV block. Treadmill exercise test revealed dynamic ST segment depression and coronary angiogram showed only 50% stenosis of LAD-D2 (Fig.1). The 24-hour Holter one month ago did not reveal any significant finding. According to recent exacerbation of clinical symptoms, we repeated the examination of 24 Holter recording. There were occasional ventricular premature beats (VPBs) through the recording periods. The six episodes of ST segment elevation on all three leads were noted at night, which all recovered spontaneously. Maximal elevation of ST segment was not associated with arrhythmias (Fig. 2A and B). However, the recovery phase of ST elevation was accompanied by polymorphic ventricular tachycardias (VT) (Fig. 2C and D). The uneven recovery of ST elevation (heterogeneous recovery of cardiac ischemia) between three leads was associated with the manifestation of Brugada-pattern ECG on V1 (Fig. 2C). VPC triggers with shorter coupling intervals between sinus beats induced non-sustained polymorphic VTs and intriguingly improved ST segment elevation. There was no prolongation of QT intervals or QT alternation. Negative flecainide challenge test did not favor the diagnosis of Brugada syndrome. We did not perform spasm provocation test. The patient received an implantation of intra-cardiac defibrillator and were well responded to verapamil. No attack of VTs and spasm were noted during follow-up. The mechanism of VTs could probably be explained by heterogeneity conduction due to cardiac ischemia, decreased I_{to} current, and phase 2 reentry.

Conclusions: We presented a patient of coronary spasm-associated polymorphic VTs, manifested with Brugada-pattern ECG. The heterogeneous ischemia of ventricular substrate could be linked to phase 2 reentry.

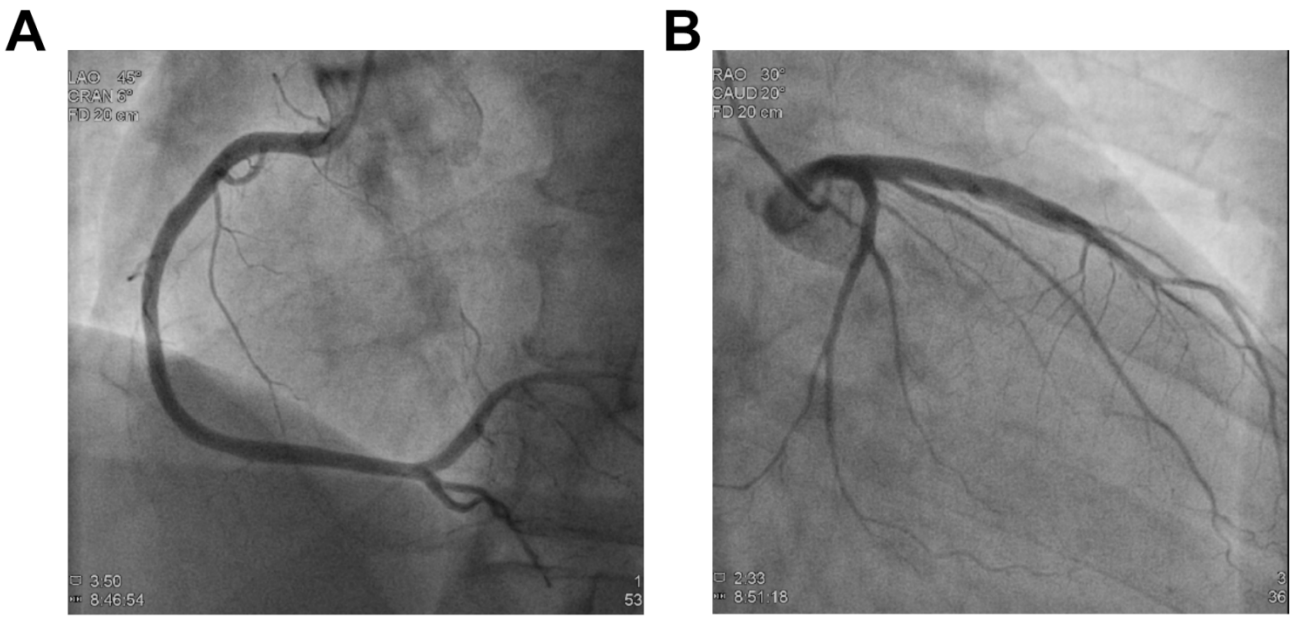

Figure 1. A, RCA: patent. **B,** LAD-D2: 50% stenosis; LCX: patent.

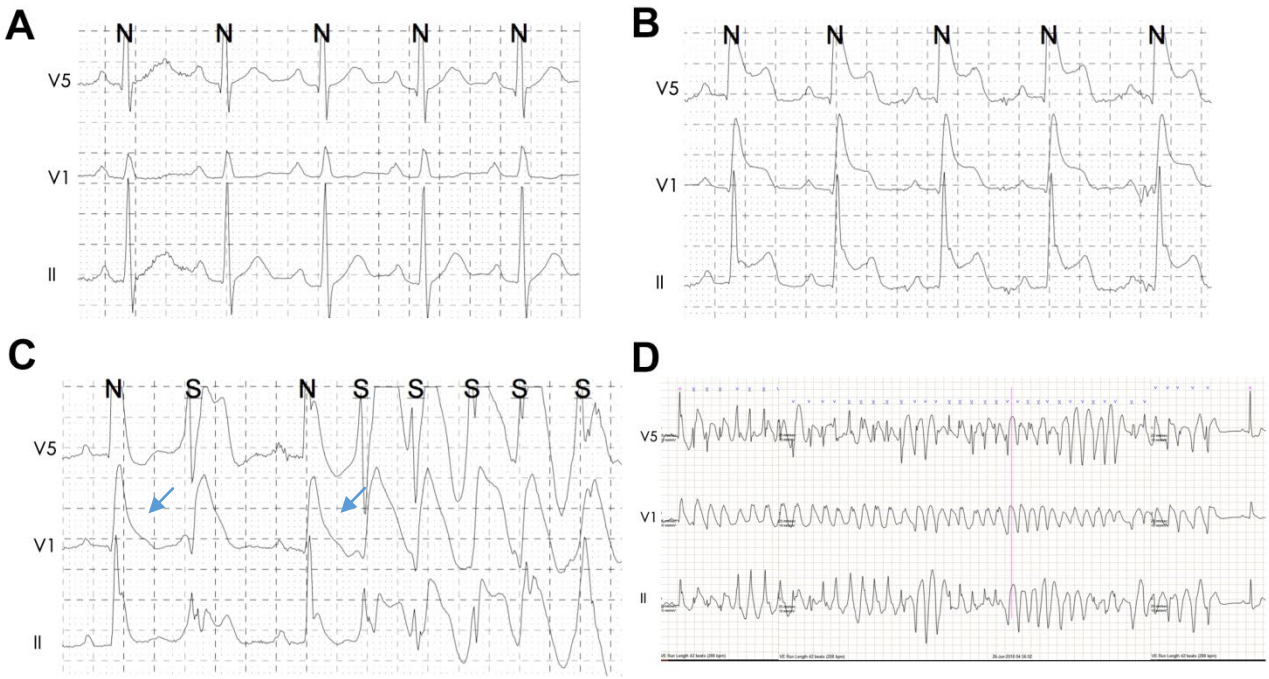

Figure 2. A, Baseline ECG, normal sinus rhythm. **B**, Maximal diffuse elevation of ST segment **C**, Manifestation of Brugada-pattern ECG on V1 during the recovery of ST segment elevation. Polymorphic VT was initiated by a shorter coupling interval of ventricular triggers. **D**. The episode polymorphic VT lasted for 15 seconds.