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Ergonovine induced ventricular fibrillation in a patient with Brugada syndrome

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Introduction: The Brugada syndrome and vasospastic angina are one of the etiology of sudden cardiac death and the two disease entities can coincide. This report describes a case of ventricular fibrillation (VF) induced by intracoronary ergonovine injection in a patient with Brugada syndrome. **Methods:** N/A **Results:** A 32-year-old male patient was brought to the Emergency Department because of aborted sudden cardiac collapse and his initial rhythm showed VF. VF was restored to sinus rhythm after defibrillation and the patient was successfully resuscitated without neurologic sequelae. Past history was denial and the patient has no history of chest discomfort, palpitation or syncope before admission. There was no familial history of ischemic heart disease, arrhythmia, or sudden death. Cardiac enzyme including Troponin I and CK-MB was not elevated. Electrocardiogram (ECG) revealed sinus rhythm without ST elevation or J wave after stabilization. Echocardiography revealed no structural heart disease with normal left ventricular ejection fraction (LVEF: 63 %). The patient underwent coronary angiography with provocation test to rule out coronary artery disease or vasospastic angina as the cause of sudden cardiac arrest. Coronary angiography revealed no significant stenosis at major epicardial coronary artery. After injection of 40 µg of ergonovine at right coronary artery, ECG showed dynamic ST segment change in right precordial leads with QRS widening and then VF was initiated. The patient was successfully resuscitated after cardiopulmonary resuscitation with a number of shocks from an external defibrillator. Flecaïnide challenging test was performed which revealed Brugada type I ECG and he was diagnosed as Brugada syndrome. He was decided to implant implantable cardioverter defibrillator. **Conclusions:** Brugada syndrome and vasospastic angina can coincide and ischemia might act additively with the substrate responsible for the Brugada syndrome to elevate the ST segment and precipitate VF. **Keywords:** Brugada syndrome; Ventricular fibrillation; Vasospastic angina

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A case of MELAS in MIDD patient

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Mitochondrial encephalomyopathy, lactic acidosis, stroke-like episodes syndrome (MELAS syndrome) and Maternally inherited diabetes and deafness (MIDD) are disease that occurs because of the same mutations in mitochondrial DNA (A3243G). The symptoms occur primarily in the skeletal muscle, central nervous system, heart, renal tubules, and pancreas, all of which have high energy demands. The onset of symptoms and clinical manifestation in patients with MELAS syndrome are diverse, depending on the affected tissues and organs, which delays the diagnosis in many cases. Common clinical features of MIDD are diabetes, neurosensory hearing loss, low BMI, short stature, and the macular dystrophy. In the present case, type 2 diabetes and hearing impairment occurred in the patient after the age of 40, and the patient had symptoms of heart failure after the age of 60. MIDD was suspected during differential diagnosis, and DNA testing revealed a 3243A > G mitochondrial DNA mutation, which confirmed MIDD. 3 months after diagnosis, the patient was hospitalized in the department of neurology after visiting the emergency department with a chief complaint of hallucination. MR brain imaging was performed and showed diffuse restricted lesions along the cortices of the right temporal and left temporoparietal lobes, indicating a high possibility that it was due to MELAS when compared with the previous test results.

