Alcohol-induced Ventricular Fibrillation in Brugada Syndrome

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A 37-year-old man lost consciousness suddenly due to ventricular fibrillation (VF). After cardioversion, twelve-lead ECG showed a pattern characteristic of type 1 Brugada. An implantable cardioverter defibrillator (ICD) was implanted for Brugada syndrome. In the following three years, VF occurred eight times after consumption of alcohol. Association between the Brugada syndrome and alcohol consumption has rarely been reported. Recently, it was reported that alcohol has inhibitory effect on single cardiac sodium channel gating and it may be that alcohol acted as a sodium channel blocker in this patient. Here we report a case of alcohol-induced VF in Brugada syndrome.


Key words: Alcohol, Ventricular Fibrillation, Sodium channel blocker, Diagnostic criteria

Introduction

The Brugada syndrome is associated with a high risk for sudden cardiac death in healthy adults. The ECG manifestations of Brugada syndrome are often changed and may be modulated by sodium channel blockers, body temperature, autonomic nervous activity, antidepressants, and blood electrolyte concentration. Sometimes these conditions and agents induce VF in Brugada syndrome.

Alcohol has been reported to affect the heart in certain ways, especially the proarrythmic effects of acute alcohol consumption.1) But, to our knowledge, a clear relationship between Brugada syndrome and alcohol has not been reported.

Case report

The patient was a 37-year-old man with no family history of sudden death and no previous abnormal ECG findings on routine medical examination. He suddenly lost consciousness for 10 minutes, and was transported to a hospital. He lost consciousness again in the emergency room of the hospital. The ECG monitor showed VF. After electric defibrillation was performed, his cardiac rhythm returned to sinus rhythm. Twelve-lead ECG showed ST-segment elevation of ≥2 mm followed by a negative T wave in the V1 lead (Figure 1). This is called Type 1 Brugada ECG. Brugada syndrome is definitively diagnosed when a type 1 ST-segment elevation is
observed in more than 1 right precordial lead (V1 to V3), in conjunction with one of the following: documented VF, polymorphic ventricular tachycardia (VT), a family history of sudden cardiac death before reaching age 45, inducibility of VT with programmed electrical stimulation, syncope, or nocturnal agonal respiration (2). Therefore, he was diagnosed as Brugada syndrome. In patients with Brugada syndrome who have spontaneous Type 1 ECG and history of VF, an ICD prevents cardiac sudden death (class I evidence level).2) The patient was referred to our hospital since he met the indications for ICD implantation. Echocardiography, treadmill test, and enhanced cardiac MRI were normal. We then performed an electrophysiological study (EPS). Triple extra stimuli from the right ventricular outflow tract induced polymorphic VT developing to VF (Figure 2). ECG did not change after the administration of pilsicainide (50 mg). After administration of propranolol (2 mg), triple extra stimuli from right ventricular apex induced polymorphic VT developing to VF. An ICD is necessary to prevent cardiac sudden death; therefore, he underwent implantation of an ICD. After he was discharged, he did not drink alcohol for 6 months. However, he began drinking after 6 months and VF occurred after drinking. He has had 8 episodes of VF in the last three years (Figure 3). All events occurred when he drank more than 50 g of ethanol, and VF occurred after at least two hours had passed after his drinking. We have counseled him to stop drinking, but he is unable to stop his drinking habit. Thus, we are considering other treatments such as drugs and ablation.

Discussion

In Brugada syndrome most arrhythmic events occur at rest and at night.3) Thus, the Brugada syndrome is inseparably connected with autonomic nervous activity, especially of the parasympathetic nervous system. In this respect Brugada syndrome differs greatly from other cardiac diseases, such as ischemic heart disease and cardiomyopathy. In these diseases enhanced activity of the sympathetic nervous system induce arrhythmic events, but in the reported case VF occurred at night after drinking, hence alcohol-induced enhanced activity of the parasympathetic nervous system may be related to triggering of VF. Hypokalemia and insulin secretion cause ST segment changes in patients with Brugada syndrome.2,4) Humoral regulation from alcohol intake may be related to the induction of VF in this case. In addition, it was recently reported that alcohol has an inhibitory effect on single cardiac sodium channel gating.5) Because sodium channel blockers induce VF in patients with Brugada syndrome, alcohol may act as a sodium channel blocker in this patient. However, his ECG was unchanged when pilsicainide was administered.

A variety of drugs have been reported to produce a Brugada-like ST-segment elevation including antiarrhythmic,6) antianginal,7) psychotropic,8) and other drugs.9) The diagnosis of Brugada syndrome is also made when a type 2 (saddleback pattern) or type 3 ST-segment elevation is observed in more than 1 right precordial lead under baseline conditions and conversion to the diagnostic type 1 pattern occurs after sodium channel blocker administration. This patient usually showed normal ECG, and only once
showed type 1 ECG after electric defibrillation for VF. Pilsicainide administration could not change his ECG. Treadmill testing also did not elevate the ST segment. This case does not show characteristic findings of the Brugada syndrome. A Brugada-like ECG can occasionally appear for a brief period or several hours after direct current cardioversion.\textsuperscript{10,11} However, this patient did fulfill the diagnostic criteria. It is an open question whether the diagnostic criteria are sufficient to make a definite diagnosis of Brugada syndrome. However, QT interval, enhanced cardiac MRI, echocardiography, coronary angiography, right ventriculography and cardiac biopsy findings were normal. There were no findings to suggest arrhythmogenic right ventricular cardiomyopathy (ARVC), long QT syndrome, and other structural heart disease. We therefore think that this case shows an atypical Brugada syndrome. It is of utmost importance for the prevention of VF that he abstains from drinking; however he is unable to stop his drinking habit. Therefore, we are considering other treatments such as drugs or ablation.\textsuperscript{12} A number of reports have shown the effectiveness of quinidine in treatment of Brugada syndrome.\textsuperscript{13–19} Because the typical features of Brugada syndrome were not observed in this case, it is unclear whether quinidine would have been effective. If VF storms occur despite the administration of quinidine, we are considering EPS and ablation under alcohol administration.

**Conclusion**

Here we reported a rare case of alcohol-induced VF in Brugada syndrome. Alcohol has a marked influence on the autonomic nervous system and functions as a sodium channel blocker. However the relation between alcohol and Brugada syndrome is unclear. Future study is necessary in order to manage Brugada syndrome patients. This case fulfilled the diagnostic criteria, but it did not show findings characteristic of Brugada syndrome. It is an open question whether this case was correctly diagnosed as Brugada syndrome. Depending on the diagnosis, the method for the treatment of VF is different. Therefore, the diagnostic criteria of Brugada syndrome should be reconsidered.
References