EP CASE REPORT

# J-wave syndrome with giant negative T-wave in severely activated arrhythmogenicity on 12-lead electrocardiography

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J-wave syndrome is one of the causes for idiopathic ventricular fibrillation (VF). A 42-year-old man with DC shock-resistant VF had giant negative T-wave with J-wave augmentation on electrocardiography (ECG). Twenty-seven hours after the onset, DC shock successfully recovered sinus rhythm. Just after the recovery, these ECG abnormal findings were disappeared, which might be associated with the severe arrhythmogenic activity.

A 42-year-old man with focal segmental glomerulosclerosis, who was admitted to our hospital, had sudden cardiopulmonary arrest. During cardiopulmonary resuscitation, electrocardiography (ECG) monitor indicated ventricular fibrillation (VF), which was resistant to DC shock, lidocaine, magnesium sulphate, and amiodarone. After he was intubated and transferred to intensive care unit, ECG showed giant negative T-wave with J-wave augmentation (*Figure 1A*). Intravenous administration of propranolol, amiodarone, and nifekalant was not able to maintain sinus rhythm. While coronary angiography showed normal epicardial coronary arteries, he was treated by intra-aortic balloon pumping (IABP) and percutaneous cardiopulmonary support system (PCPS) due to severely activated arrhythmogenicity. Even after IABP and PCPS support, DC shock-resistant VF was still observed (*Figure 1B*). Twenty-seven hours after the onset, DC shock successfully recovered sinus rhythm. Just after the recovery, ECG showed normalized giant negative T-wave and improved J-wave augmentation (*Figure 1C*).

In this report, we were able to document giant negative T-wave with J-wave augmentation on 12-lead ECG. J-wave syndrome (early repolarization syndrome) is one of the causes for idiopathic VF, which is characterized by early repolarization at the end of QRS wave, especially in the inferior leads. Early repolarization has been accepted as a benign ECG variant for decades; however, ST-segment elevation, which also includes J-wave augmentation as in the present study, is usually observed as a result of transmural voltage gradient, caused by the

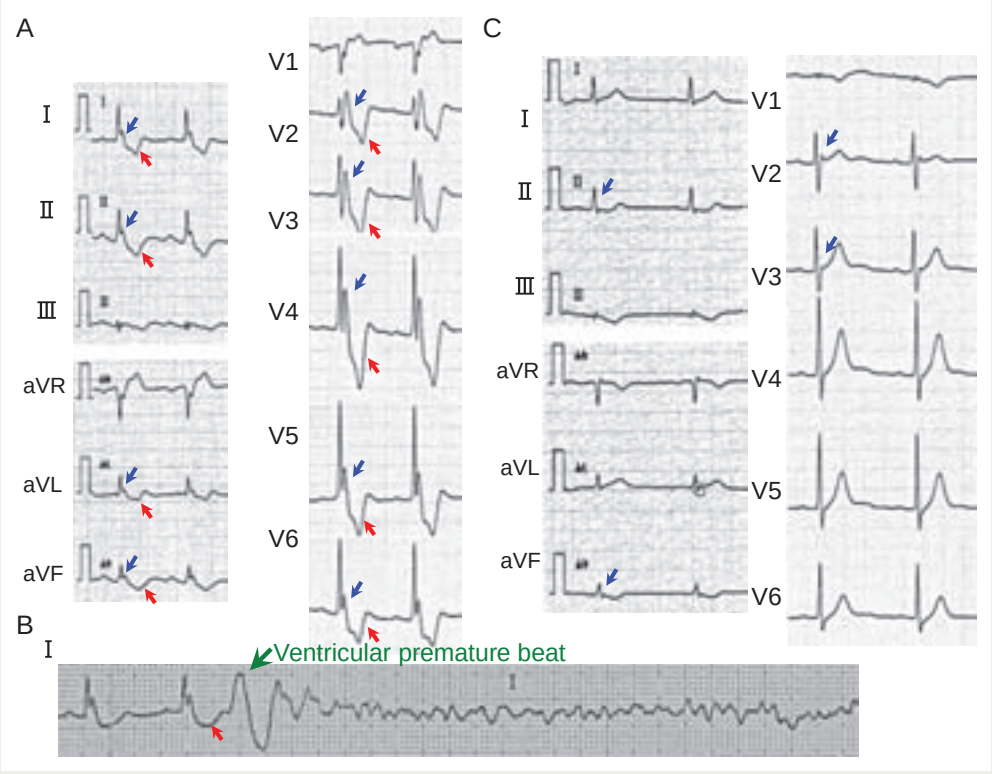


**Figure 1** (*A*) Giant negative T-wave (red arrows) with J-wave augmentation (turqvoise arrows), (*B*) VF initiated with giant negative T-wave (red arrow), and (*C*) attenuated J-wave just after recovery (turqvoise arrows).

differences in the level of the action potential plateau among cells spanning the ventricular wall.[1,2] Furthermore, it has been reported that VF storms in patients with idiopathic VF are highly associated with J-waves, which showed augmentation prior to the VF onset.[3]

We consider that the giant negative T-wave suggested the excitation delay in the whole left ventricle, which can be caused by persistent VF and the administration of antiarrhythmic drugs.[4] In this pathological condition, $Ca^{2+}$ overload in the myocardium exacerbate the arrhythmogenicity. In addition, activated $IK_{ATP}$ caused by cellular ischaemia due to VF storm might cause the progression of loss-of-dome, which is prone to Phase 2 reentry and VF storm. Accordingly, the augmented J-wave combined with giant negative T-wave is associated with the severe arrhythmogenic activity, as observed in this case.

**Conflict of interest:** none declared.

## References

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