

Successful Treatment of Electrical Storm with Isoproterenol in a Patient of Myocarditis with Early Repolarization in Hypothermia

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A 19-year-old man without any past medical history visited a local hospital complaining of fever which had developed 3 days prior. He had been treated with antibiotics and evaluated for a fever focus. However, there was no definite fever focus. During admission, the fever persisted and vital signs became unstable. As a result, he was transferred to the emergency department of our center. On examination, his blood pressure was 80/50 mmHg with pulse rate 80/min, respiratory rate 26/min and body temperature was 36.1°C. Troponin I was 11.904 ng/mL (0-0.05 ng/mL), CK-MB was 64.90 ng/mL (0-5 ng/mL) and proBNP was 27277 pg/mL (0-278 pg/mL). A chest X-ray demonstrated pulmonary edema on both lung fields with normal cardiac shadow. Electrocardiography (ECG) demonstrated sinus tachycardia with ST depression of inferior leads and ST elevation

of lead I and aVL (Fig. 1A). Transthoracic echocardiography showed severely decreased systolic function with normal cardiac chamber size. Coronary angiography showed no critical stenosis in both coronary arteries (Fig. 2A, B). An endomyocardial biopsy was performed. During the procedure, the patient collapsed and we began cardiopulmonary resuscitation. Then, Extracorporeal Membrane Oxygenation (ECMO) was established for hemodynamic support. During the intensive care unit (ICU) care, the patient's body temperature decreased to below 35.0°C because of a large volume of fluid circulation through ECMO. At that time, the ECG demonstrated early repolarization with J point elevation in most of the leads and notching of the terminal portion of QRS complex known as Osborn wave (Fig. 1B). The QTc interval was a prolonged 513 ms and K was 3.8 mEq/dL.

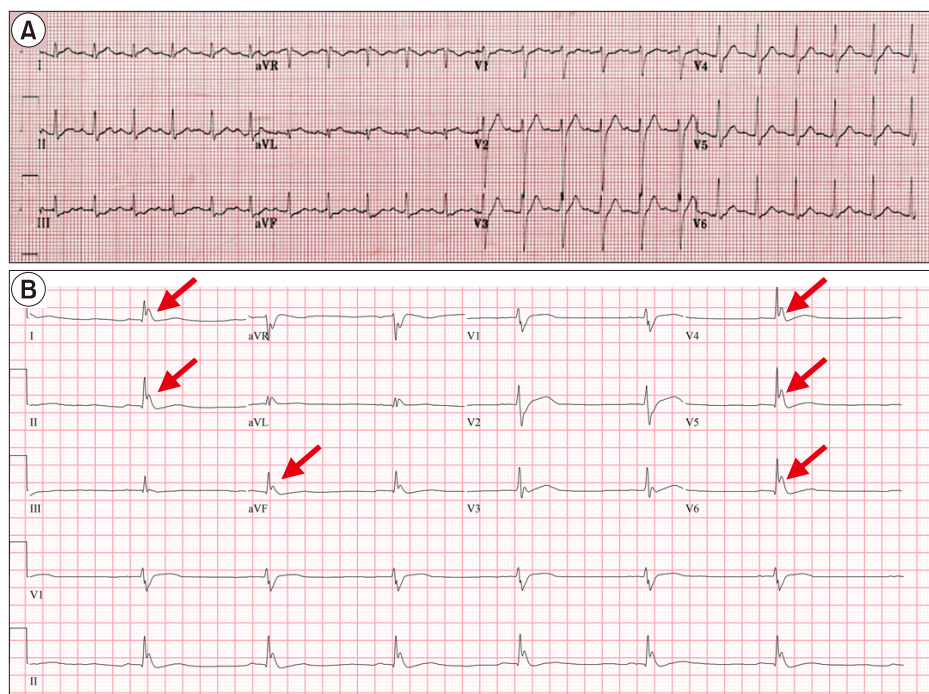


FIG. 1. (A) A twelve lead electrocardiogram (ECG). Sinus tachycardia with ST depression of inferior leads and ST elevation in I, aVL were observed. (B) A twelve lead ECG. J point elevation in the most of leads and notching of terminal portion of QRS complex were observed. It was prominent, especially in the lateral leads (red arrow). (C) Ventricular fibrillation occurred and it was terminated by defibrillation. After defibrillation, Osborn wave still remained. (D) A twelve lead ECG after infusion of isoproterenol. Early repolarization was nearly normalized. The notch in the lateral leads was noticeably decreased. Heart rate was accelerated by the chronotropic effect of isoproterenol.

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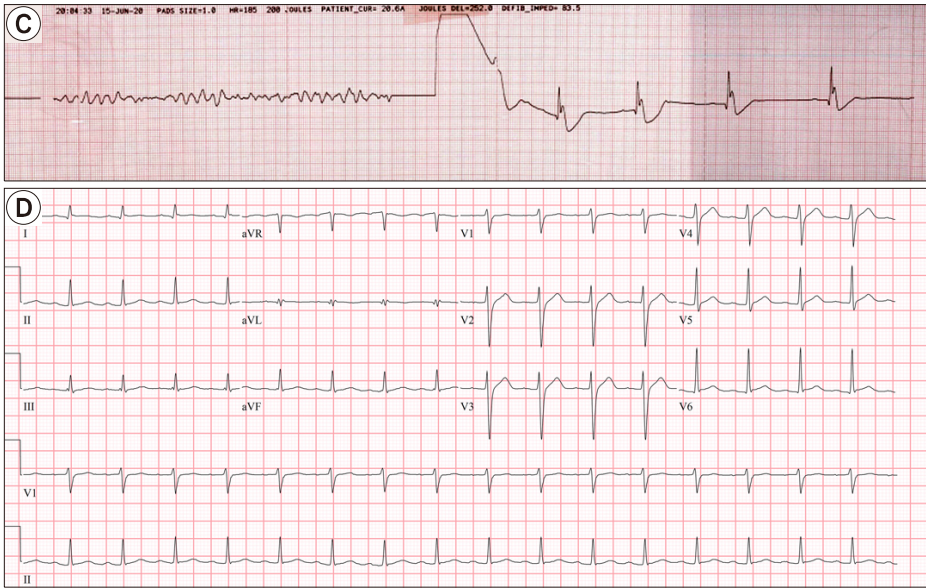


FIG. 1. Continued.

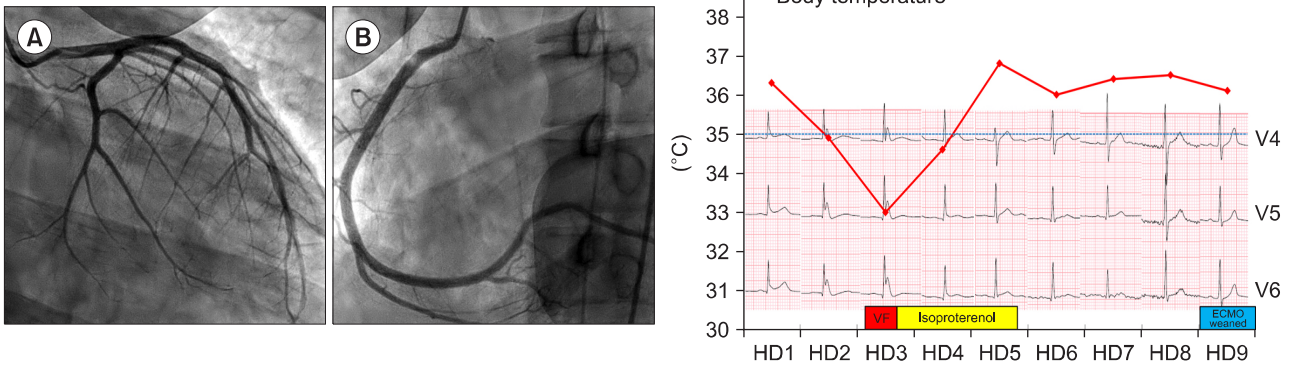


FIG. 2. (A) Right anterior oblique view of left coronary artery angiogram. There was no significant stenosis in left coronary arteries. (B) Left anterior oblique view of right coronary artery angiogram. There was no significant stenosis in right coronary artery. (C) Serial change of body temperature and QRS wave. QRS waves are V4, V5, V6 leads. As the body temperature decreased, the Osborn wave became prominent. At hospital day 3, ventricular fibrillation occurred. After infusion of isoproterenol, the Osborn wave disappeared. HD, hospital day; VF, ventricular fibrillation; ECMO, extracorporeal membrane oxygenation.

(3.5-5.0 mEq/dL) Then, a few hours later, recurrent ventricular fibrillation (VF) occurred (Fig. 1C). The patient was defibrillated for each VF episodes. However, short run of VF persisted repeatedly. Finally, the patient was stabilized by an infusion of isoproterenol which successfully suppressed early repolarization (Fig. 1D, 2C). Endomyocardial biopsy demonstrated inflammatory cells including lymphocyte infiltrated near myocytes injury which suggested myocarditis. At 9 days of hospitalization, ECMO was weaned and finally he was discharged on hospital day 37 after recovery.

Nowadays, ECMO is widely conducted in various shock patients for hemodynamic support. Since the large amount of fluid circulated through ECMO, patients' body temperature tends to decrease. ECG findings of hypothermia include the Osborn wave, bradycardia, interval (PR/QRS,

QT) prolongations.¹ Among them, the Osborn wave is a positive deflection in the terminal portion of QRS complex. It is attributed to transmembrane voltage gradient due to transient outward K current by I_{to} channel, mainly in the epicardium.² Previous studies suggested several mechanisms of the arrhythmogenic potential of Osborn wave. One of them was action potential notch changed to loss of dome, which could induce heterogeneity between epicardium and endocardium. That might eventually induce phase 2 reentry and VF.³ Isoproterenol can decrease those electrical voltage gradients and reduce J wave by enhancing the action of I_{Ca} channel which accelerated inward current of Ca. In addition, it could inhibit the I_{to} channel by acceleration of heart rate. Those effects could result in normalization of repolarization abnormalities.

Recently hypothermia commonly occurs in ECMO, con-

tinuous renal replacement therapy or therapeutic hypothermia. It is vulnerable for ventricular arrhythmia. In those cases, isoproterenol might be effective therapeutic option to early repolarization patients for preventing ventricular arrhythmia.

CONFLICT OF INTEREST STATEMENT

None declared.

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