A 44-year-old man was hospitalized for meningitis, with increased cell count and protein level in the cerebrospinal fluid (CSF). Despite treatment with antibiotics, he developed disturbed consciousness after 8 days. A diagnosis of acute disseminated encephalomyelitis (ADEM) was made based on elevated oligoclonal IgG bands, myelin basic protein level, and 80% monocytes in the CSF with absence of serum antibodies against commonly causal viruses. Magnetic resonance imaging (MRI) indicated extensive damage to the brainstem and cerebellum.

Figure 1. (A, B) Sagittal T2-weighted magnetic resonance imaging (MRI) showing a broad high-intensity area in the (A) spinal cord and (B) pons (white arrows). (C) The high-intensity lesion was also observed at the edge and near the hypothalamus on fluid-attenuated inversion recovery MRI, coronal view (white arrows).
inflammatory response to virus, vaccine, or other infectious agent. It predominantly affects the white matter of the brain and spinal cord, occasionally involving the brainstem, diencephalon, or hypothalamus. Such systemic neural damage is reported to lead to functional disorders.

In this case, a thermoregulatory disorder was suspected because the patient complained of heat and sudoresis. Together with the T2-weighted MRI findings, ADEM-mediated hypothalamus damage would cause thermoregulatory disorder.

Early repolarization, characterized by J wave or slur after the QRS complex on ECG, is often seen in young healthy people. Some patients with idiopathic VF associated with ERS also have an augmented J wave before VF develops. Both repolarization and depolarization abnormalities can cause ERS, and the augmented cardiac transmural dispersion and accompanying phase-2 re-entry can provoke VF, based on genetic abnormality related to cardiac ion currents. Unusual physiological conditions such as hypothermia can also cause the early repolarization known as Osborn J wave, which is augmented with the severity of hypothermia. Interestingly, hypothermia affects including the edges of and regions near the hypothalamus (Figure 1). Three months later, his body temperature decreased to 34°C, but he felt hot and had sudoresis. Suddenly, repetitive ventricular fibrillation (VF) after prominent J wave developed (Figure 2A). On emergency cardiac catheterization and transthoracic echocardiography, no signs of structural heart disease were seen. Notably, his temperature decreased to 32.9°C while the electrocardiogram (ECG) showed prominent J wave in II, III, aVF, and V4-V6 (Figure 2B). He was rewarmed during deep sedation to inhibit repetitive VF, and no J wave was observed after the temperature normalized to 36.5°C (Figure 2C). Next-generation gene sequencing (HiSeq2000™; Illumina, San Diego, CA, USA) screening for 580 mutations and 20 microRNAs detected no mutation except the NDRG4 variant seen in 0.05% of the general population. VF did not recur for 3 years by avoiding hypothermia.

This case demonstrates that ADEM with hypothermia due to thermoregulatory disorder can cause VF associated with early repolarization syndrome (ERS). ADEM is an immune-mediated demyelinating condition triggered by an inflammatory response to virus, vaccine, or other infectious agent. It predominantly affects the white matter of the brain and spinal cord, occasionally involving the brainstem, diencephalon, or hypothalamus. Such systemic neural damage is reported to lead to functional disorders. In this case, a thermoregulatory disorder was suspected because the patient complained of heat and sudoresis. Together with the T2-weighted MRI findings, ADEM-mediated hypothalamus damage would cause thermoregulatory disorder.

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the transient outward potassium current, resulting in an accelerated epicardial action potential notch. Although no evidence for correlation was seen on genetic analysis, hypothermia would have played at least some role in this case.

To our knowledge, this is the first report of hypothermia triggering ERS and subsequent VF after ADEM-mediated hypothalamus disorder.

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Disclosures
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References