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Dynamic J-Point Elevation Associated with Epileptic Hemiplegia: The Osborn Wave of Todd's Paralysis

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Key Words
Todd's paralysis · Osborn wave · J-wave · Epilepsy · Stroke · STEMI · Hemiplegia · Postictal · ACS

Abstract
This case report exposes a phenomenon which, although proposed, has not been described in clinical literature: transient postictal hemiplegia (Todd's paralysis) with concomitant electrocardiographic J-point deflection (Osborn waves). Although typically associated with hypothermia, a prominent J-wave on the electrocardiogram (ECG) results from a transmyocardial voltage gradient during ventricular repolarization. Rarely, the Osborn wave may be observed in a non-hypothermic setting such as hypercalcemia or cerebral hemorrhage. Transient postictal hemiplegia has been attributed to localized cerebral hypoperfusion resulting from motor cortex exhaustion following an epileptic seizure. The same central nervous system autonomic dysfunction has been theorized to produce subendocardial hypoperfusion with electrocardiographic change and cardiac troponin T elevation. This is the first described ECG evidence of a dynamically displaced J-point in the setting of postictal hemiplegia.

Background

Typically described in association with hypothermia, a prominent J-wave on the electrocardiogram (ECG), referred to as the Osborn wave, results from a transmyocardial voltage gradient during ventricular repolarization [1]. Rarely, this phenomenon may be observed in a non-hypothermic setting including brain injury [2, 3]. Postictal epileptic hemiplegia, also known as 'Todd's paralysis', has been associated

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with localized transient cerebral hypoperfusion as a result of motor cortex exhaustion lasting an average of 15 h following a seizure [4]. The same central nervous system autonomic dysfunction can produce subendocardial hypoperfusion with electrocardiographic change and cardiac troponin T (cTnT) elevation [5].

Case Report

While shopping at his local market, a 75-year-old epileptic man had a witnessed loss of consciousness with urinary incontinence and subsequent postictal confusion. Paramedics noted left-sided upper and lower extremity hemiparesis with aphasia and he was taken to a local hospital. ECG performed within the first hour after the event revealed ST-segment abnormalities of V3–V6, with serum cardiac troponin I (cTnI) 0.01 (normal 0.00–0.40 ng/ml) and no symptoms of cardiac ischemia (**fig. 1**). Tympanic temperature was 37.2°C. Ionized calcium was 1.15 (1.13–1.32 mmol/l). The patient was transferred to a facility capable of emergent percutaneous coronary intervention for ST-elevation myocardial infarction (STEMI) and cardiology consultation was performed on arrival. Initial serum cTnT was 0.492 mg/ml (normal <0.100 ng/ml). Neuroimaging with non-contrast head CT and MRI did not reveal hemorrhage or focal ischemia. Signs of unilateral hemiplegia resolved within fifteen hours of seizure coinciding with resolved J-point displacement primarily in leads V3–V6 consistent with Osborne waves (**fig. 2**). A transthoracic echocardiogram (TTE) identified an akinetic cardiac apex with a normal base and reduced left ventricular systolic function. No segmental wall motion abnormality was identified to suggest ischemia of a single coronary territory. At its peak, cardiac biomarker elevation was attributed to CNS-mediated autonomic dysfunction due to flattened release of troponin [cTnT 0.687 (<0.100 ng/ml); CK 243 (30–259 U/l); MB fraction 24.1 (0–8.9 ng/ml); relative index 9.9 (0–3.9%)] (**table 1**). Appropriate medical therapy was initiated and follow-up TTE at one month revealed normalization of cardiac function. This finding suggested resolution of a stress-induced cardiomyopathy.

Discussion

In this clinical vignette, hypoperfusion following a postictal surge of autonomic tone produced evidence of both cerebral and cardiac ischemia. Osborn waves have been described in other instances of cerebral injury such as trauma or hemorrhage; however, ECG evidence of a dynamically displaced J-point has not been previously described in the setting of postictal hemiplegia.

Disclosure Statement

The authors have no conflict of interest.

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Table 1. Cardiac troponin serum concentrations following epileptic seizure

Elapsed time	30 min	2 h	9.5 h	18 h
cTnT		0.492	0.687	0.637
cTnI	0.01			

* The normal range for cTnT and cTnI is <0.010 and <0.40 ng/ml, respectively. Note the flattened release of cTnT as postictal hemiplegia resolves.

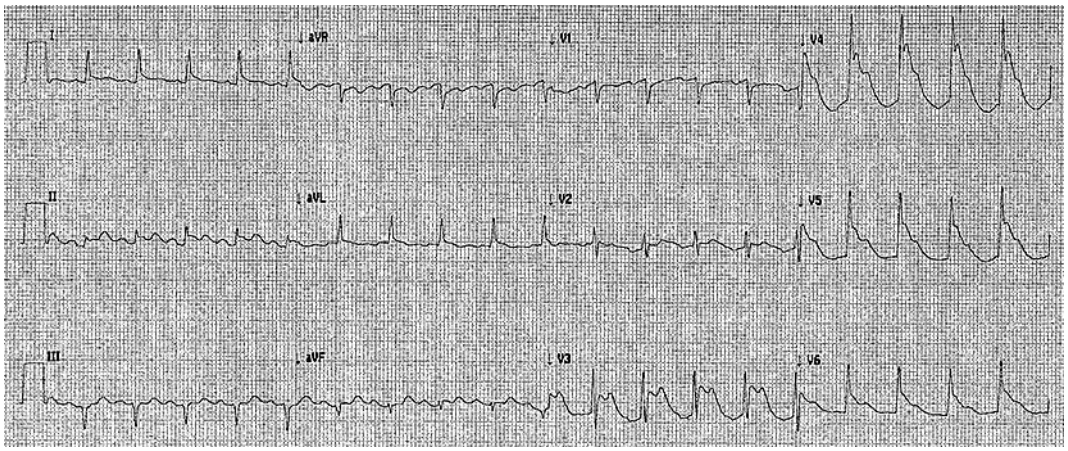

Fig. 1. ECG on presentation with J-point elevation in V3–V6.

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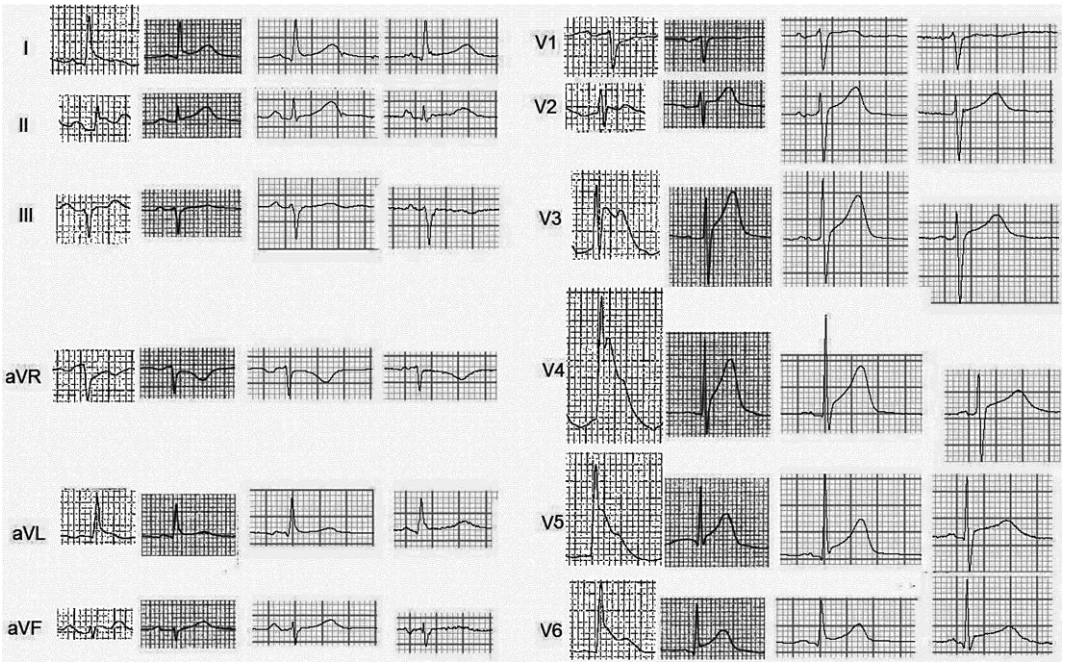

Fig. 2. J-point normalizes as hemiparesis resolves. ECG progression from presentation (approximately 45 min after seizure), prior to transfer (approximately 90 min after seizure), arrival (approximately 120 min after event), and the next morning (approximately 16 h after seizure).

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