

# Electrographic Osborn wave in severe hypercalcaemia

Abdul Aziz A. Asbeutah <sup>1,2\*</sup> and Majed H. Salem<sup>2</sup>

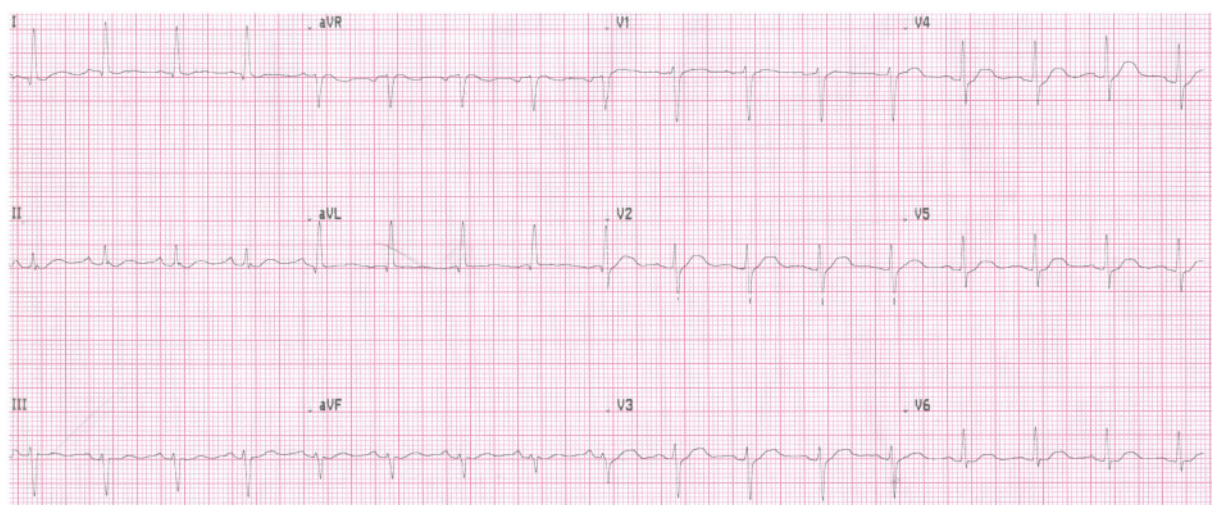
<sup>1</sup>Division of Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Avenue, Boston 02215, MA, USA; and <sup>2</sup>Division of Internal Medicine, Al-Amiri Hospital, Bin Misbah Street, Capital Governorate, Sharq, Kuwait City 15300, Kuwait

Received 19 August 2019; first decision 28 August 2019; accepted 12 September 2019; online publish-ahead-of-print 26 October 2019

A 62-year-old woman was referred to our emergency room for generalized weakness, weight loss, and recent change in her mental status over the past week. On arrival, the patient was vitally stable, with a body temperature of 37.2°C and had a score of 10 on the Glasgow Coma Scale; however, nor her or her relatives could recall her pertinent past medical history. On initial investigation, she was found to have a serum creatinine of 105 µmol/L, serum potassium of 4.3 mmol/L, and normal serum cardiac biomarkers. However, serum calcium was 3.55 mmol/L (reference 2.1–2.6 mmol/L), serum phosphate was 0.71 mmol/L (reference 1.1–1.9 mmol/L), and serum parathyroid hormone level was elevated to 525 pg/mL (reference 10–65 pg/mL). A routine electrocardiogram exhibited 0.1 mV positive

deflections following the QRS complex in lead II consistent with Osborn waves, or J-waves (Figures 1 and 2).

Osborn, initially described Osborn waves, or J-waves, as the ‘current of injury’ in 1953 as a response to hypothermia in dogs and noted that the amplitude of the J-wave was inversely related to body temperature.<sup>1</sup> Since its description it has been associated with several other conditions, even at normothermia. The differential for ‘normothermic Osborn waves’ has evolved over the past several decades and has come to include entities such as benign early repolarization, severe hypercalcaemia, myocardial injury following cardiopulmonary resuscitation, illicit drug overdose, channelopathies, and with subarachnoid haemorrhage.<sup>2,3</sup>



**Figure 1** Routine 12-lead electrocardiogram.

\* Corresponding author. Tel: +1(617)-667-6010, Email: [asbeutah@hotmail.co.uk](mailto:asbeutah@hotmail.co.uk)

Handling Editor: Borislav Dinov

Peer-reviewer: John Camm

© The Author(s) 2019. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact [journals.permissions@oup.com](mailto:journals.permissions@oup.com)



**Figure 2** Osborn waves (J-waves) of 0.1 mV amplitude in lead II.

Our patient was immediately treated with intravenous fluids, calcitonin, and placed on an infusion of zoledronic acid to reduce serum calcium. On further investigation, the patient was diagnosed with a parathyroid adenoma as part of Multiple Endocrine Neoplasia type 1 (MEN-1) syndrome. Repeat electrocardiogram at follow-up showed disappearance of the J-wave and return back to baseline.

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and

associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

## References

1. Osborn JJ. Experimental hypothermia: respiratory and blood pH changes in relation to cardiac function. *Am J Physiol* 1953;**175**:389–398.
2. Patel A, Getsos JP, Moussa G, Damato AN. The Osborn wave of hypothermia in normothermic patients. *Clin Cardiol* 1994;**17**:273–276.
3. Sridharan MR, Horan LG. Electrocardiographic J wave of hypercalcemia. *Am J Cardiol* 1984;**54**:672–673.