CME Article

Electrocardiographical case. J wave and presyncope in a middle-aged woman

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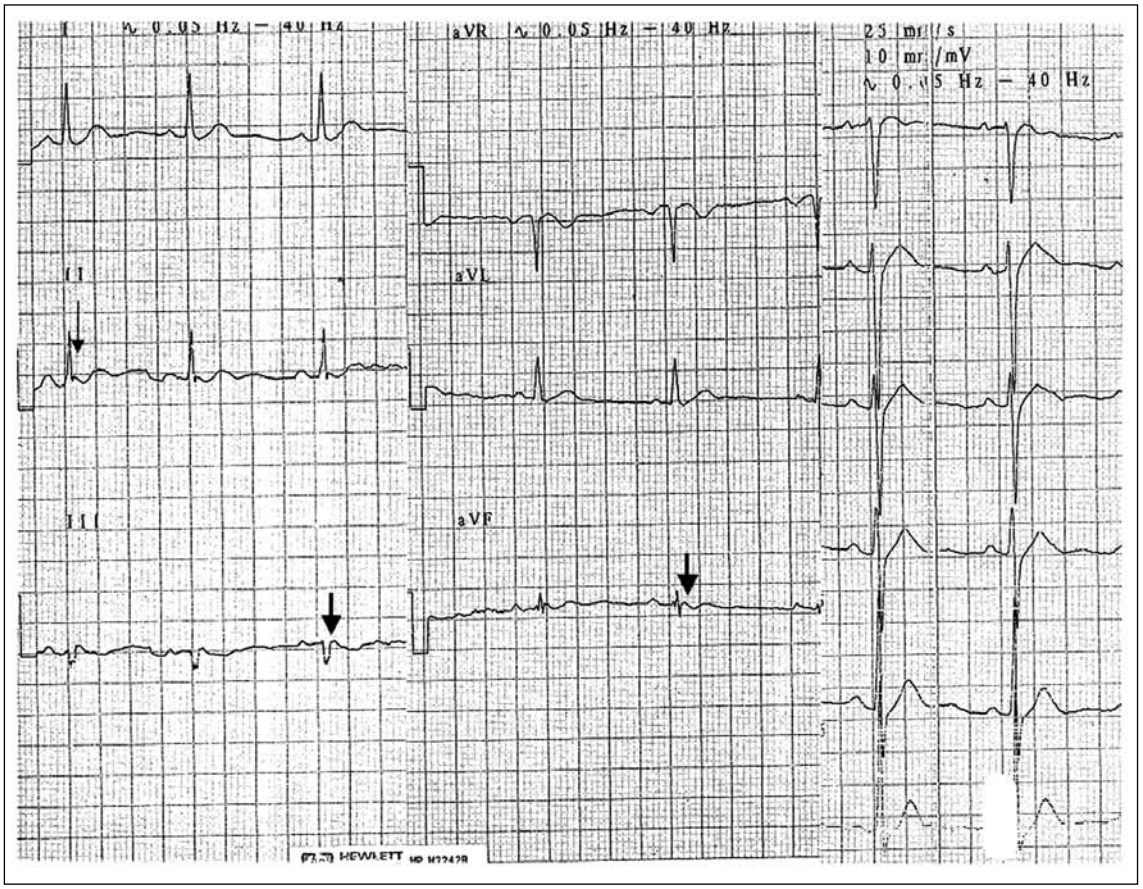

Fig. 1 12-lead ECG at admission.

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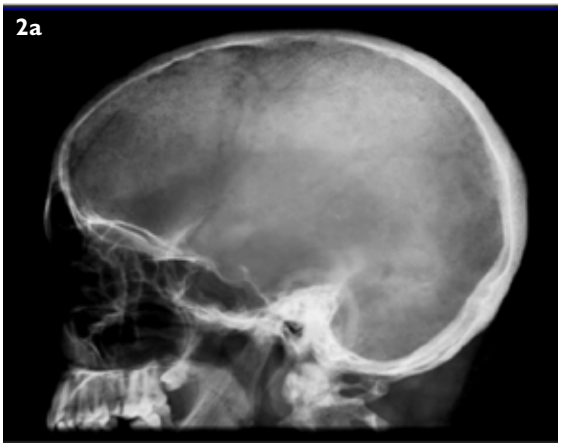
CLINICAL PRESENTATION

A 46-year-old woman with a history of hypertension for the last 12 years, presented to us with three episodes of presyncope in the previous two days. Clinical examination was normal. The 12-lead electrocardiogram (ECG) was done (Fig. 1). What does the initial ECG show? What is your diagnosis?



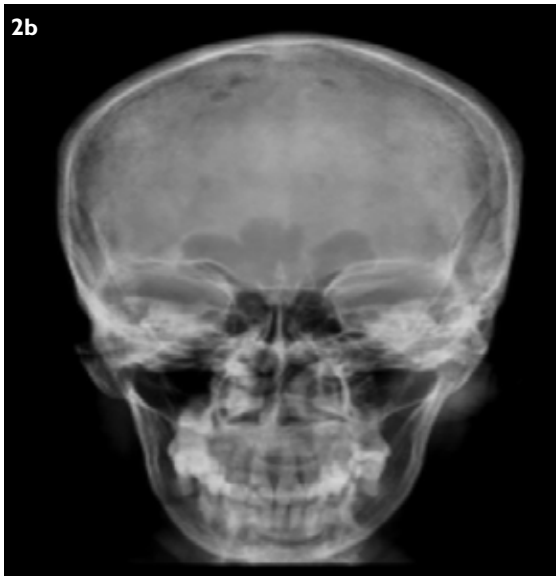


Fig. 2 (a) Lateral and (b) antero-posterior radiographs of the skull show tiny punched-out lesions, producing a so-called salt-and-pepper appearance.

ECG INTERPRETATION

The ECG showed sinus rhythm, relatively-short QT interval (QTc of 340 msec) with shortening predominantly involving the ST segment, and prominent J-waves in inferior leads (Fig.1). Decreased T-wave amplitude with prominent U-waves in lead II and midprecordial leads were also noted. Electrocardiographical J-waves have been described with hypothermia, hypercalcaemia, Brugada syndrome, subarachnoid haemorrhage and vasospastic angina.

DIAGNOSIS

The presence of relatively shortened QTc, J-waves and normal clinical condition made us to look for possible hypercalcaemia in this patient.

CLINICAL COURSE

There was no documented brady- or tachyarrhythmia. Biochemical evaluation revealed serum calcium of 17.4 mg/dL, phosphorus of 2.3 mg/dL and alkaline phosphatase of 533 IU/ml. She was evaluated for hypercalcaemia with a provisional diagnosis of primary hyperparathyroidism. Radiographical evaluation revealed a salt-and-pepper appearance of the skull (Figs. 2a–b) and subperiosteal resorption of phalanges (Fig. 3). Ultrasonography of the neck showed adenoma of the right upper parathyroid gland. Parathyroid scintigraphy with Tc-99m Tc-MIBI showed increased activity in the right upper parathyroid gland, which persisted in the late images. She was managed with saline diuresis followed by elective parathyroidectomy. Peri- and postoperative periods were uneventful. The ECG became normal with the disappearance of J-waves after treatment for hypercalcaemia (Fig. 4). She was asymptomatic at six months follow-up.

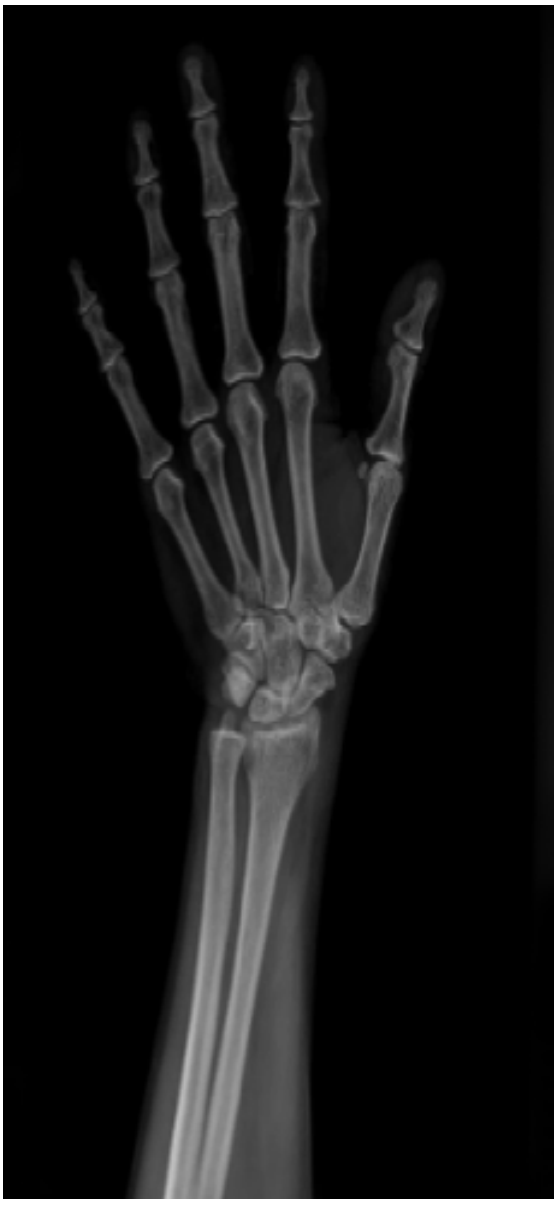


Fig. 3 Antero-posterior radiograph of the hand shows subperiosteal resorption of digital tufts and phalanges.

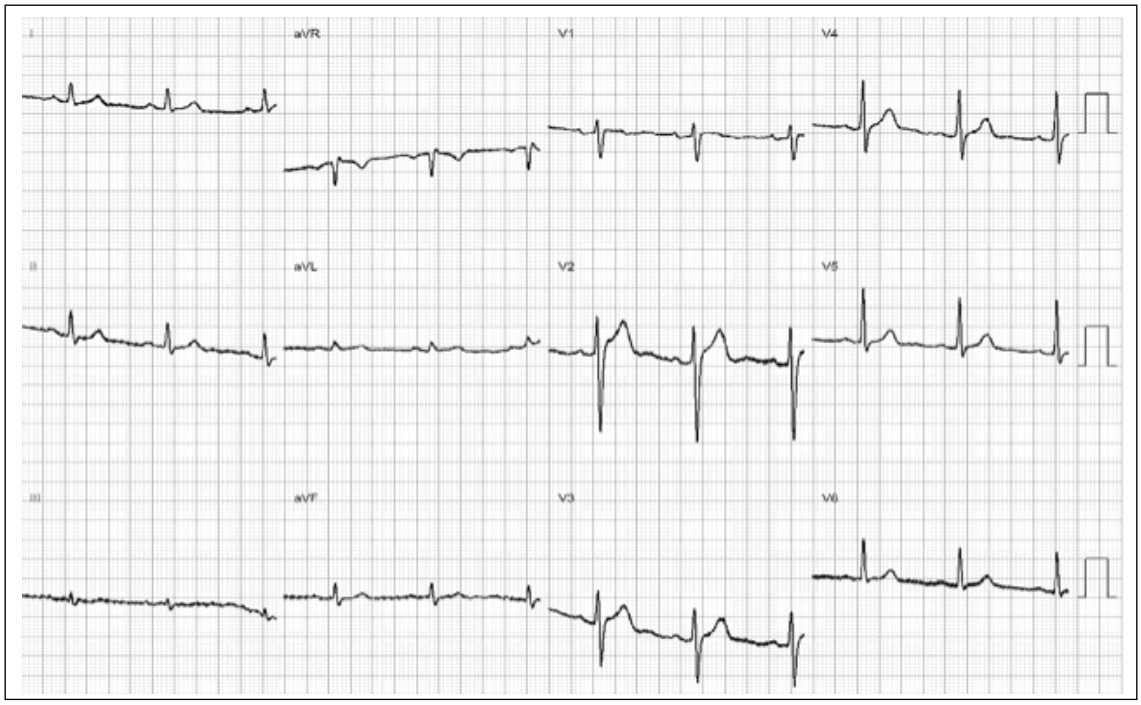


Fig. 4 ECG, after correction of hypercalcaemia (Ca^{2+} 8.2 mg/dL), shows normal QTc and no J-waves.

DISCUSSION

The presence of J-waves (Osborne waves) in hypercalcaemia has rarely been reported.[1-6] J-waves were described with hypothermia, Brugada syndrome and, more uncommonly, with subarachnoid haemorrhage and vasospastic angina. Our case was unique in that the presenting symptom was presyncope, rather than systemic symptoms often seen with primary hyperparathyroidism. We assume the cause of presyncope in our case to be transient ventricular arrhythmias. However, no tachy- or bradyarrhythmia could be documented in the hospital. Calcium levels came down to 10.4 mg% within 24 hours of admission and the patient did not have recurrence of symptoms afterwards.

The electrophysiological basis of the J-wave is controversial. Yan and Antzelevitch suggested a heterogeneous distribution of a transient outward current-mediated spike-and-dome morphology of the action potential across the ventricular wall as the cellular mechanism of the electrocardiographical J-wave.[7] An accentuation of the spike-and-dome morphology of the epicardial, but not endocardial action potential, was shown to provide a voltage gradient that manifests as a J-wave or elevated J-point in the ECG. In hypercalcaemia, this is thought to be due to an increased net outward current during the early phases of the action potential, possibly due to an increase in calcium-activated chloride current and a decrease in I_{Ca}.[8] The presence of a prominent transient outward current (Ito) in the epicardium has been shown to sensitise tissue to the effects of high calcium.[8] Apart from this phase 2 re-entry, delayed after-depolarisation-induced triggered activity in hypercalcaemia also contribute to the development of electrical inhomogeneity in the ventricle, and thus leading to the genesis of ventricular arrhythmias.

Apart from the Osborne wave, electrocardiographical changes associated with hypercalcaemia include a relatively shortened QT interval, mainly shortening the ST segment component of it. Decreased T-wave amplitude, T-wave notching and T-wave inversion are also observed in severe hypercalcaemia. With increasing levels of serum calcium, a high take-off of the ST segment in leads V1 and V2, simulating acute myocardial ischaemia, has also been described.[9-11] After it was described initially by Kraus in 1920,[1] J-wave in hypercalcaemia was reported in a few cases in humans.[2-6] Proper recognition of this abnormal ECG finding and evaluation eliminated further cardiac evaluation to investigate the cause of presyncope in our patient. This case is being reported to highlight the association of J-waves with hypercalcaemia and its uncommon presentation as presyncope.

ABSTRACT

A 46-year-old woman presented with three episodes of presyncope in the previous two days. Electrocardiogram (ECG) showed sinus rhythm, relatively short QT interval (QTc of 340 msec) and prominent J-waves in the inferior leads. Biochemical evaluation revealed serum calcium of 17.4 mg/dL, phosphorus of 2.3 mg/dL and alkaline phosphatase of 533 IU/ml. She was managed with saline diuresis followed by elective

parathyroidectomy. ECG became normal with the disappearance of J-waves after correction of hypercalcaemia. She was asymptomatic at six months of follow-up. The presence of J-waves (Osborne waves) in hypercalcaemia has rarely been reported.

Keywords: hypercalcaemia, hyperparathyroidism, J-wave

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SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 20082A)

	True	False
Question 1. Electocardiographical J-waves are seen in:		
(a) Subarachnoid haemorrhage.	☐	☐
(b) Vasospastic angina.	☐	☐
(c) Long QT syndrome.	☐	☐
(d) Brugada syndrome.	☐	☐
Question 2. Electrocardiographical changes associated with hypercalcaemia include:		
(a) "high take off of ST segment".	☐	☐
(b) Shortened QTc.	☐	☐
(c) Osborne wave .	☐	☐
(d) T-wave alternans.	☐	☐
Question 3. The mechanism by which hypercalcaemia result in ventricular arrhythmias include:		
(a) Phase 2 re-entry.	☐	☐
(b) Delayed after repolarisation.	☐	☐
(c) Triggered activity.	☐	☐
(d) Increased automaticity.	☐	☐
Question 4. Radiological features of hyperparathyroidism include:		
(a) Subperiosteal resorption.	☐	☐
(b) Osteosclerosis.	☐	☐
(c) "Salt and pepper" appearance of the skull.	☐	☐
(d) Delayed epiphyseal fusion	☐	☐
Question 5. The true statement on diagnosis and management of hyperparathyroidism include:		
(a) Saline diuresis is used in acute emergency.	☐	☐
(b) Emergency parathyroidectomy is life-saving.	☐	☐
(c) ECG changes revert after a few days of correction of hypercalcaemia.	☐	☐
(d) Parathyroid scintigraphic scan is useful in localisation of the adenoma.	☐	☐

Doctor's particulars:

Name in full: _____

MCR number: _____ Specialty: _____

Email address: _____

SUBMISSION INSTRUCTIONS:
(1) Log on at the SMJ website: **www.sma.org.sg/cme/smj** and select the appropriate set of questions. (2) Select your answers and provide your name, email address and MCR number. Click on "Submit answers" to submit.

RESULTS:
(1) Answers will be published in the SMJ April 2008 issue. (2) The MCR numbers of successful candidates will be posted online at **www.sma.org.sg/cme/smj** by 15 April 2008. (3) All online submissions will receive an automatic email acknowledgment. (4) Passing mark is 60%. No mark will be deducted for incorrect answers. (5) The SMJ editorial office will submit the list of successful candidates to the Singapore Medical Council.

Deadline for submission: (February 2008 SMJ 3B CME programme): 12 noon, 25 March 2008