Pseudo-infarction electrocardiographic changes in delayed onset hypoparathyroidism: a case report

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Abstract

Pseudo-infarction electrocardiographic changes associated with hypokalemia and hypocalcemia is rare. A 76-year-old lady with previous total thyroidectomy presented with unspecific symptoms. Electrocardiogram demonstrated ST-elevation. She was found to have hypokalemia and hypocalcemia associated with delayed onset hypoparathyroidism. This may suggest possible etiologies like coronary vasospasm and catecholamine-associated myocardial injury.

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Keywords: Pseudo-infarction, Delayed Onset Hypoparathyroidism, ST Elevation Mimics, Case report

Introduction

The identification of ST-segment elevation pattern on ECG during clinical practise is vital to recognise myocardial ischemia, as timely myocardial reperfusion affects major adverse cardiovascular events (MACE). In clinical practice, the decision to proceed with reperfusion strategy is made before laboratory results are made available. To date, many aetiologies have been associated with pseudo-infarction pattern on ECG other than obstructive coronary disease. Causes such as coronary vasospasm, increased myocardial demand, and electrolyte abnormalities have been demonstrated [1-3]. Such knowledge is important to avoid unneeded invasive procedural risks in an unstable patient.

Incidence of post-operative permanent hypoparathyroidism varies from 1.7-68%. Although rare, delayed onset of hypoparathyroidism post thyroidectomy has been reported, and this has been demonstrated in the form of laryngeal spasm, parkinsonism, seizure, and cardiac arrhythmia. We report a case of ST-segment elevation mimicking acute coronary syndrome in the setting of hypokalemia and hypocalcemia with delayed onset post-surgical hypoparathyroidism.

Case Presentation

A 76 years-old lady with hypertension and previous history of total thyroidectomy in 2017 was brought to a local emergency department with a two-day history of vague generalized weakness and poor oral intake. Premorbidly, she stayed alone and was visited by her family members regularly. Her previous oral medications include Amlodipine/ Valsartan combination and L-Thyroxine. The initial 12-lead ECG demonstrated generalised "triangular" QRS-ST-T waveform mimicking a "shark fin pattern" (Figure 1A). She was then transferred to the regional percutaneous coronary intervention (PCI) centre for further evaluation of acute silent myocardial infarction. Assessment on arrival revealed no angina symptoms but a more detailed history could not be elicited due to delirium as well as lack of collateral history from her family. Physical examination showed a frail and dehydrated patient, with blood pressure of 79/51mmHg, with a heart rate of 112 beats per minute. Neurological examination was unremarkable. Bedside echocardiography revealed normal ejection fraction, with no evidence of pericardial effusion or regional wall motion anomalies. Laboratory investigations showed a raised Troponin T of 46ng/L (Roche Elecsys). Other routine biochemistry studies were notable for potassium concentration of 2.3mmol/l, corrected calcium concentration of 1.41mmol/l, phosphate of 1.39mmol/l, albumin of 36g/L as well as creatinine level of 116umol/L. pH was 7.58, pCO2 31mmHg, pO2 67mmHg, HCO3 29.1mmol/L. A preliminary diagnosis of electrolyte induced ECG changes was made, for which she was commenced on intravenous replacement infusion. Further investigation was remarkable for serum intact parathyroid hormone of <1.2 pmol/L (reference range 1.6-6.9 pmol/L). With careful electrolyte correction and hydration, as well as close interval monitoring, she improved clinically and the ECG changes resolved as potassium and calcium concentrations normalised (Figure 1B). She was subsequently able to provide a clearer history of diarrhoea episodes during the period of feeling unwell. No further invasive coronary study was performed as there was resolution of ECG changes following correction of electrolytes and dehydration. ECG repeated prior to discharge did not show any ST-T changes (Figure 1C). At follow-up about 3 months later, she remained asymptomatic of angina.

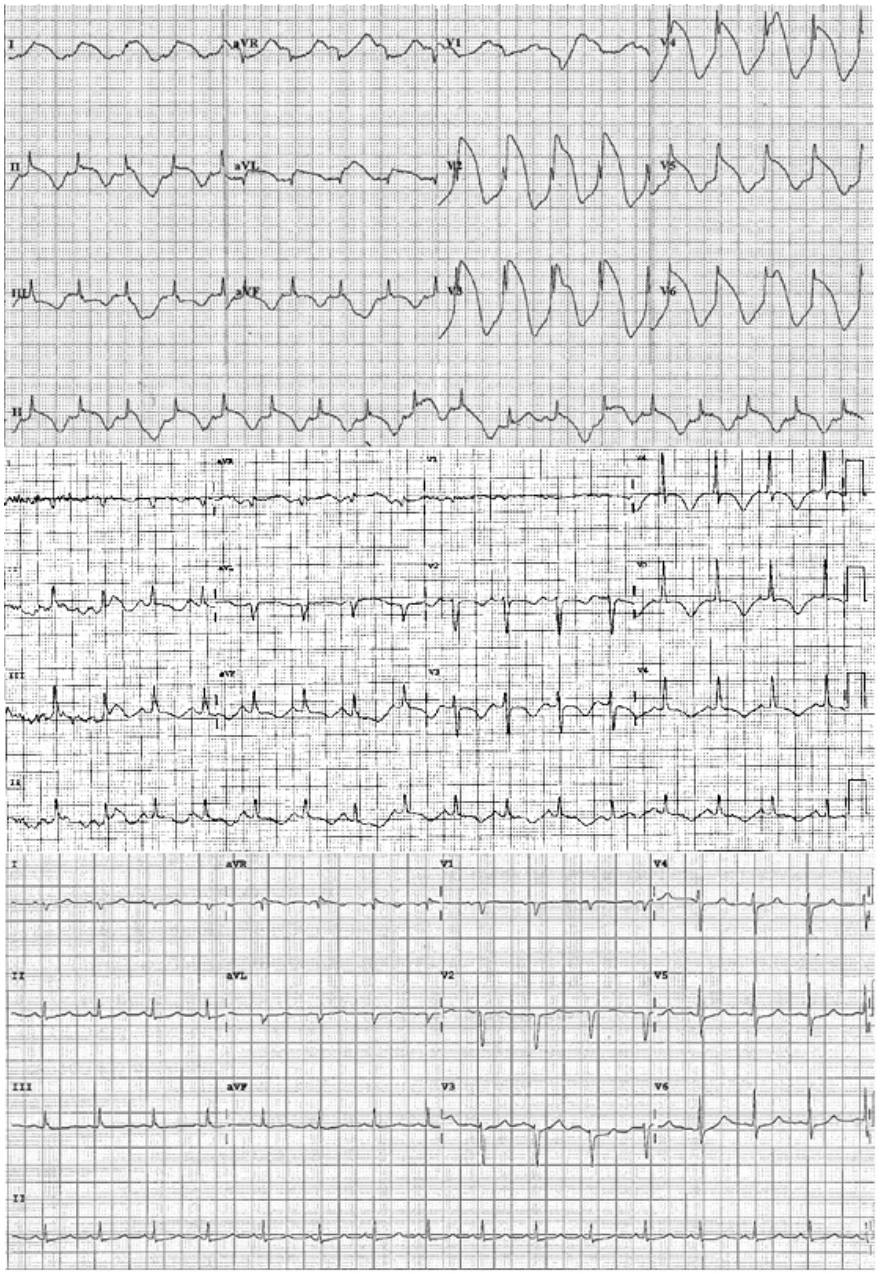

Figure 1: (A) Admission electrocardiogram showing marked ST segment elevation**. (B)**

Resolution of ST segment elevation after aggressive electrolyte correction. **(C)**

Electrocardiogram on discharge.

Discussion

This case illustrated an unusual ECG pattern of diffuse J-point elevation which is non-cardiac in aetiology. The authors would like to specifically mention that there is no ST-segment upsloping, but rather 'J-point elevation' with an elevated cardiac troponin level. Such an ECG pattern can be alarming, as it may suggest early changes of acute myocardial infarction. Of note, her deterioration during presentation was without

symptoms typical of myocardial infarction and there was a resolution of ST-segment changes following electrolyte correction and hydration. These findings strengthen the proposition that electrolyte imbalance secondary to gastrointestinal loss and the newly diagnosed hypoparathyroidism was the cause of the global ST-segment elevation. ECG changes of global ST- segment elevation is well recognized in hyperkalemia. However, reported cases associated with hypokalemia are rare. A pseudoinfarction ECG pattern as seen in hyperkalemia was noted previously in a patient with severe hypokalemia undergoing correction, and was postulated to be associated with rapid changes in intracellular/extracellular [K+] ratio[6]. Hypocalcemia has been shown to simulate ECG patterns of myocardial injury with subsequent investigations showing no evidence of infarction[8-9] and it has also been suspected to provoke coronary vasospasm[9]. Dehydration, severe hypotension, congestive heart failure, coronary vasospasm, myocardial bridging, and hypertensive emergencies have all been linked to provoking conditions that can cause a mismatch in myocardial oxygen supply (type II myocardial infarction). An elevated cardiac troponin has been found in these situations[10]. Taken together, these facts fit well in the case of our patient but the exact mechanism and contribution of these factors to the ECG manifestation remain unknown. According to earlier case studies, metabolic acidosis associated with DKA may also result in an elevation in ST-segment[2-4]. However, our patient's pH, serum bicarbonate, or other serum electrolytes did not notice any noteworthy alterations when the ECG was normalized.

To date, several authors have reported cases of hypoparathyroidism diagnosed many years after surgery. This phenomenon of delayed onset hypoparathyroidism has been postulated to be associated with scar formation as well as progressive atrophy of parathyroid glands. We report this rare case with the aim of creating awareness about this potential complication in post thyroidectomy patients. This case adds to the literature as well the association of hypokalemia and hypocalcemia with pseudo-ischemic electrographic changes that clinicians should be aware of.

Conclusion

The finding of ST-elevation pattern in ECG can be non-specific and has been reported to be associated with electrolyte imbalance, pericarditis and coronary vasospasm[1]. Taking into account ischemic time as well as circulatory failure, the consideration of other differential diagnoses in clinical practice might be difficult before laboratory results are made available. The present case reinforces the importance to explore the possibility of reversible electrolyte-induced ECG changes even if an ischemic cause is suspected. We propose further research in coronary investigations to demonstrate the underlying mechanism. Late-onset hypoparathyroidism has been shown to give rise to hypocalcemia several years after thyroidectomy and can be missed due to the non-specific nature of hypocalcemia symptoms as well as the lack of consistency in regular follow up. This is an important phenomenon to consider when assessing patients with history of previous neck surgery even if they appear to be well controlled in symptoms or given history of taking thyroxine diligently. While this case illustrates the rarity of the combination of etiologies leading to the patient's presentation, it reminds practitioners to remain comprehensive when assessing patients in acute setting, especially the geriatric patient population.

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Declaration

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Ethical Approval

This study was registered via National Medical Research Register Malaysia (NMRR) with a Research ID of NMRR ID-22-02447-HBF

Consent for Publication

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Abbreviations

ECG : Electrocardiogram

MACE : Major Adverse Cardiovascular Events

PCI : Percutaneous Coronary Intervention

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