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CASE REPORT

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

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Key Clinical Message

The high-risk "Shark Fin" electrocardiogram (ECG) pattern has been associated with transmural ischemia but can also result from electrolyte anomalies. Therefore, the decision for invasive coronary catheterization requires a detailed history and dedicated biochemical tests.

Abstract

Pseudo-infarction ECG pattern resembling "Shark Fin" was demonstrated in a 76-year-old lady with a previous total thyroidectomy who presented with unspecific symptoms. An incidental finding of hypokalemia and hypocalcemia was thought to be related to delayed onset hypoparathyroidism. Potential etiologies like coronary vasospasm and catecholamine-associated myocardial injury were suggested.

KEYWORDS

case report, delayed onset hypoparathyroidism, pseudo-infarction, ST elevation mimics

1 | INTRODUCTION

The identification of ST-segment elevation patterns on electrocardiogram (ECG) during clinical practice is vital to recognize myocardial ischemia, as timely myocardial reperfusion affects major adverse cardiovascular events. In clinical practice, the decision to proceed with reperfusion strategy is made before laboratory results are made available. To date, many etiologies have been associated with pseudo-infarction patterns 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.

The incidence of postoperative permanent hypoparathyroidism varies from 1.7% to 68%. Although it is 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.[5] We report a case of ST-segment elevation mimicking acute coronary syndrome in the setting of hypokalemia and hypocalcemia with delayed onset postsurgical hypoparathyroidism.

2 | 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 2-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 a generalized "triangular" QRS-ST-T waveform mimicking a "shark fin pattern" (Figure 1A). She was then transferred to the regional percutaneous coronary intervention center for further evaluation of

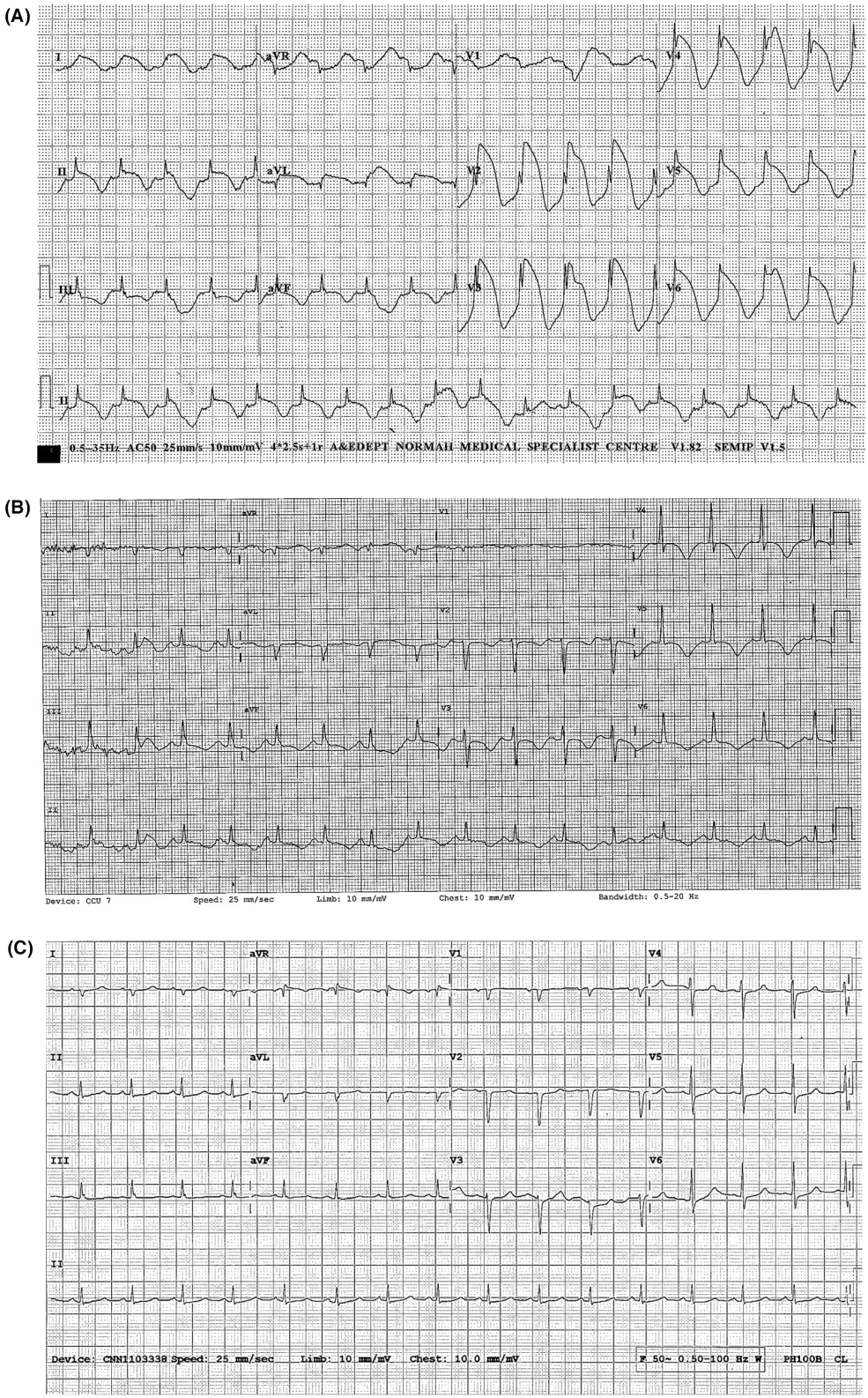

FIGURE 1 (A) Admission electrocardiogram (ECG) showing marked ST segment elevation. (B) Resolution of ST segment elevation after aggressive electrolyte correction. (C) ECG on discharge.

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 a lack of collateral history from her family. Physical examination showed a frail and dehydrated patient, with blood pressure of a 79/51 mmHg, 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 46 ng/L (Roche Elecsys). Other routine biochemistry studies were notable for potassium concentration of 2.3 mmol/L, corrected calcium concentration of 1.41 mmol/L, phosphate of 1.39 mmol/L, albumin of 36 g/L as well as creatinine level of 116umol/L. pH was 7.58, pCO_2 31 mmHg, pO_2 67 mmHg, HCO3 29.1 mmol/L (refer to Table 1). 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 normalized (Figure 1B). She was subsequently able to provide a clearer history of diarrhea episodes during the period of feeling unwell. No further invasive coronary study was performed as there was a resolution of ECG changes following correction of electrolytes and dehydration. ECG repeated before discharge did not show any ST-T changes (Figure 1C). At follow-up about 3 months later, she remained asymptomatic of angina.

TABLE 1 Laboratory test results on the day of presentation.

Blood test	Results	Reference range
Troponin T	46	ng/L (<40)
Potassium	2.3	mmol/L (3.3–5.1)
Corrected calcium	1.41	mmol/L (2.20–2.60)
Phosphate	1.36	mmol/L (0.74–1.52)
Albumin	36	g/L (34–54)
Creatinine	116	μmol/L (50–98)
pH	7.58	(7.35–7.45)
pCO_2	31	mmHg (35–45)
pO_2	67	mmHg (75–100)
HCO_3	29.1	mmol/L (22–26)
Intact parathyroid hormone	<1.2	pmol/L (1.6–6.9)

3 | DISCUSSION

This case illustrated an unusual ECG pattern of diffuse J-point elevation which is noncardiac in etiology. 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 of 46 ng/L. Such an ECG pattern can be alarming, as it may suggest early changes of acute myocardial infarction. Of note, her deterioration during the 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 are well recognized in hyperkalemia. However, reported cases associated with hypokalemia are rare. A pseudo-infarction 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[7–9] 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 to create awareness about this potential complication in post-thyroidectomy patients. This case adds to the literature as well as the association of hypokalemia and hypocalcemia with pseudo-ischemic electrographic changes that clinicians should be aware of.

4 | CONCLUSION

The present case reminds practitioners to consider etiologies of reversible electrolyte-induced ECG anomalies even when an ischemia cause is suspected, particularly in the unusual presentations of the aging population with numerous comorbidities.

AUTHOR CONTRIBUTIONS

Han Bing Chow: Conceptualization; funding acquisition; investigation; methodology; project administration; resources; validation; visualization; writing – original draft; writing – review and editing. **Caryn Tsujean Lim:** Conceptualization; methodology; project administration; resources; supervision; validation; visualization; writing – review and editing. **Yik Hon Ho:** Conceptualization; methodology; project administration; resources; supervision; validation; visualization; writing – review and editing. **Yee Ling Cham:** Project administration; supervision; validation; writing – review and editing. **Alan Yean Yip Fong:** Project administration; supervision; validation; writing – review and editing. **Asri Said:** Funding acquisition; project administration; supervision; validation; writing – review and editing. **Tiong Kiam Ong:** Project administration; supervision; validation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

ETHICS STATEMENT

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

CONSENT

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

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

1. Hanna EB, Glancy DL. ST-segment elevation: differential diagnosis, caveats. *Cleve Clin J Med*. 2015;82(6):373-384. doi:10.3949/ccjm.82a.14026
2. Adeel MY, Clarke JD, Shetty S, Arora A, Buscher MG. Severe hypocalcemia mimicking acute inferior ST-segment elevation myocardial infarction. *Oxf Med Case Reports*. 2018;2018(12):omy103. doi:10.1093/omcr/omy103
3. Lehmann G, Deisenhofer I, Ndrepepa G, Schmitt C. ECG changes in a 25-year-old woman with hypocalcemia due to hypoparathyroidism. Hypocalcemia mimicking acute myocardial infarction. *Chest*. 2000;118(1):260-262. doi:10.1378/chest.118.1.260
4. Dalzell JR, Jackson CE, Petrie MC. Profound hypokalaemia mimicking acute myocardial infarction. *QJM*. 2009;102(11):822-824. doi:10.1093/qjmed/hcp102
5. Kamath SD, Rao BS. Delayed post-surgical hypoparathyroidism: the forgotten chameleon! *J Clin Diagn Res*. 2017;11(2):OD07-OD09. doi:10.7860/JCDR/2017/23609.9260
6. Madias JE, Madias NE. Hyperkalemia-like ECG changes simulating acute myocardial infarction in a patient with hypokalemia undergoing potassium replacement. *J Electrocardiol*. 1989;22(1):93-97. doi:10.1016/0022-0736(89)90027-7
7. Khardori R, Cohen B, Taylor D, Soler NG. Electrocardiographic finding simulating acute myocardial infarction in a compound metabolic aberration. *Am J Med*. 1985;78(3):529-532. doi:10.1016/0002-9343(85)90351-1
8. Reddy CV, Gould L, Gomprecht RF. Unusual electrocardiographic manifestations of hypocalcemia. *Angiology*. 1974;25(11):764-768. doi:10.1177/000331977402501105
9. Popow M, Kochanowski J, Krakowian M. Coronary spasm secondary to hypocalcaemia and hypomagnesaemia. *Kardiol Pol*. 2015;73(1):57. doi:10.5603/KP.2015.0005
10. Mahajan N, Mehta Y, Rose M, Shani J, Lichstein E. Elevated troponin level is not synonymous with myocardial infarction. *Int J Cardiol*. 2006;111(3):442-449. doi:10.1016/j.ijcard.2005.08.029

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