



Case Report

Hypocalcemia as a cause of reversible heart failure: A case report and review of the literature



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ABSTRACT

Introduction: In both pediatric and adult populations, hypocalcemia-induced heart failure is an extremely rare presentation. The aim of the current study is to report a case of reversible heart failure caused by severe hypocalcemia resulting from hypoparathyroidism, which was resolved by correcting the serum calcium level.

Case report: A 29-year-old female presented with orthopnea, dyspnea on mild exertion, and bilateral lower limb swelling. She had a positive Trousseau's sign. Vital signs were stable except for tachycardia. On chest auscultation, there were bilateral basal fine inspiratory crackles. She was immediately treated as a case of pulmonary edema with intravenous furosemide and oxygen therapy. Subsequent treatment for correcting the hypocalcemia was then initiated.

Discussion: Hypocalcemia has been proven to influence cardiac function, resulting in lower cardiac contractility as determined by decreased left ventricular work index, stroke index, and cardiac index. In rare circumstances, the clinical, biochemical (elevation of cardiac enzymes), electrocardiographic, and echocardiographic data may lead to an incorrect diagnosis of an acute ischemic attack.

Conclusion: Hypocalcemia as a possible factor leading to heart failure should be considered in the differential diagnosis of all individuals with congestive heart failure.

1. Introduction

Heart failure is a common clinical condition characterized by symptoms caused by an anatomical or functional heart abnormality that limits the ventricle's capacity to fill with or eject blood [1]. It is the most common cardiac cause of hospitalization in patients over the age of 60 years [2]. Regardless of significant improvements in therapeutic modalities, heart failure is associated with significant morbidity and mortality and creates a major burden on patients, caregivers, and healthcare systems [3]. Heart failure is becoming more common around the world, owing to an increase in incidence due to population aging, an increase in the prevalence of poorly controlled risk factors such as hypertension, diabetes, and obesity, and increased survival of patients with heart failure due to the implementation of more effective, evidence-based treatments [3].

Calcium is essential for cardiac contraction and relaxation [4]. Despite its well-established role, however, hypocalcemia-induced heart

failure is an extremely rare presentation in both pediatric and adult populations [5].

The current study's goal is to report a case of reversible heart failure induced by severe hypocalcemia resulting from hypoparathyroidism, which was resolved by correcting the calcium level concentration. The report is structured in accordance with SCARE guidelines and includes a brief literature review [6].

2. Case report

Patient's information: A 29-year-old female presented with orthopnea, dyspnea on mild exertion, bilateral lower limb swelling, and chest tightness. The patient had a history of epilepsy and hypocalcemia, and she was only given anticonvulsant therapy (levetiracetam) without calcium. She had no previous surgeries, no history of medication allergies, and a negative family history.

Clinical examination: Upon admission, the patient had carpopedal

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spasm, and this became more obvious during blood pressure measurement (Trousseau's sign). The patient's blood pressure was 140/90 mmHg, pulse rate was 110 beats per minute, respiratory rate was 25 cycles per minute, and her temperature was 37.1 °C. General physical examination revealed puffiness of the face, elevated jugular venous pressure, and bilateral pitting edema. On auscultation of the chest and precordium, there were bilateral basal fine inspiratory crackles with S3 gallop rhythm. She was immediately treated for pulmonary edema with intravenous furosemide and oxygen therapy.

Diagnostic assessment: Electrocardiogram (ECG) showed prolongation of the QT interval and T inversion in leads V1 to V4 (Fig. 1). Echocardiogram revealed global hypokinesia with severe left ventricular dysfunction (ejection fraction 20%). Blood investigations showed normal blood counts, renal function tests, liver function tests, and thyroid function tests. She had very low serum calcium (3.31 mg/dL), elevated phosphorous (9.34 mg/dL), low serum magnesium (1.13 mg/dL) and low parathyroid hormone (PTH) levels (2.33 pg/mL).

Therapeutic interventions: The patient was admitted to hospital. Intravenous calcium gluconate infusion (990 mg per 24 hours) was administered along with alfacalcidol tablet (1 mcg, three times daily) and magnesium sulfate infusion.

Follow-up: The patient did very well, with resolution of her symptoms (shortness of breath and carpopedal spasm) within three days. She was discharged on calcium carbonate, alfacalcidol, bisoprolol, spironolactone, perindopril, and indapamide. After one month from the discharge, her echocardiography showed improvement in the left ventricular function with an ejection fraction of 40% and a normal serum calcium level (8.5 mg/dL).

3. Discussion

Primary hypoparathyroidism (PHPT) is defined by an abnormally low amount of PTH release [7]. The primary function of PTH is to keep the calcium concentration in extracellular fluid (ECF) within a narrow normal range [8]. Hypocalcemia can therefore result from PTH deficiency [7]. Hypocalcemia, along with hypokalemia and hypomagnesemia, is a significant component in stress-induced cardiac cytotoxicity [9]. In the literature, a few cases of heart failure induced by hypocalcemia accompanied by hypoparathyroidism have been reported [10].

Calcium is essential for excitation–contraction coupling in the

myocardium. Because the calcium confined within the sarcoplasmic reticulum is inadequate to begin contraction, extracellular calcium influx is primarily responsible for the initiation and extent of cardiac contraction [11]. These findings establish biologic plausibility and provide an approach to understanding the pathophysiologic relationship between extracellular hypocalcemia, namely low blood calcium levels, and reversible heart failure [11]. Hypocalcemia has been proven to influence cardiac function, resulting in lower cardiac contractility as determined by decreased left ventricular work index, stroke index, and cardiac index [12]. The prevalence of heart failure increases with age, rising from 1.9% in males and 1.4% in females between the ages of 40 and 59 to 14.7% and 12.8%, respectively, beyond the age of 80 [10]. The exact cause of the increased prevalence is unclear. Many risk factors for heart failure, such as diabetes and hypertension, are more common in the aged population and have been discussed elsewhere [10]. The current case was 29-year-old with no history of diabetes mellitus or hypertension.

In pediatric population, vitamin D deficiency is a common cause of hypocalcemia, whereas in adults, cardiac function impairment is more often multifactorial and is typically associated with either hypoparathyroidism or chronic kidney disease while it is less likely to be associated with vitamin D deficiency alone [5]. The hypocalcemia of the presenting case was caused by low PTH. Following thyroidectomy, hypocalcemia can be induced by the unintentional removal of one or more parathyroid glands, ischemia caused by disruption of their fragile blood supply, or by merely manipulating the glands. Hypocalcemia is now recognized as a somewhat common surgical consequence that is usually temporary and asymptomatic [13].

Severe manifestations of hypocalcemia include confusion, muscle spasms, numbness in the hands, feet, and face, depression, hallucinations, muscle cramps, brittle nails, and an increased risk of bone fractures [11]. Tingling is a common sign of hypocalcemia. The Chvostek and Trousseau's signs are associated with severe hypocalcemia or a sudden onset of hypocalcemia [14]. Trousseau's sign was positive in the current case. The occurrence of hypocalcaemic heart failure as the initial manifestation of primary hypoparathyroidism is an exceedingly rare condition [15]. This was the case in this study. Hypocalcemia has been associated with a prolonged QT interval, arrhythmias, and reduced left ventricular ejection fraction (LVEF) [16]. Hypocalcemia should be evaluated in individuals with heart failure and prolonged QT intervals.

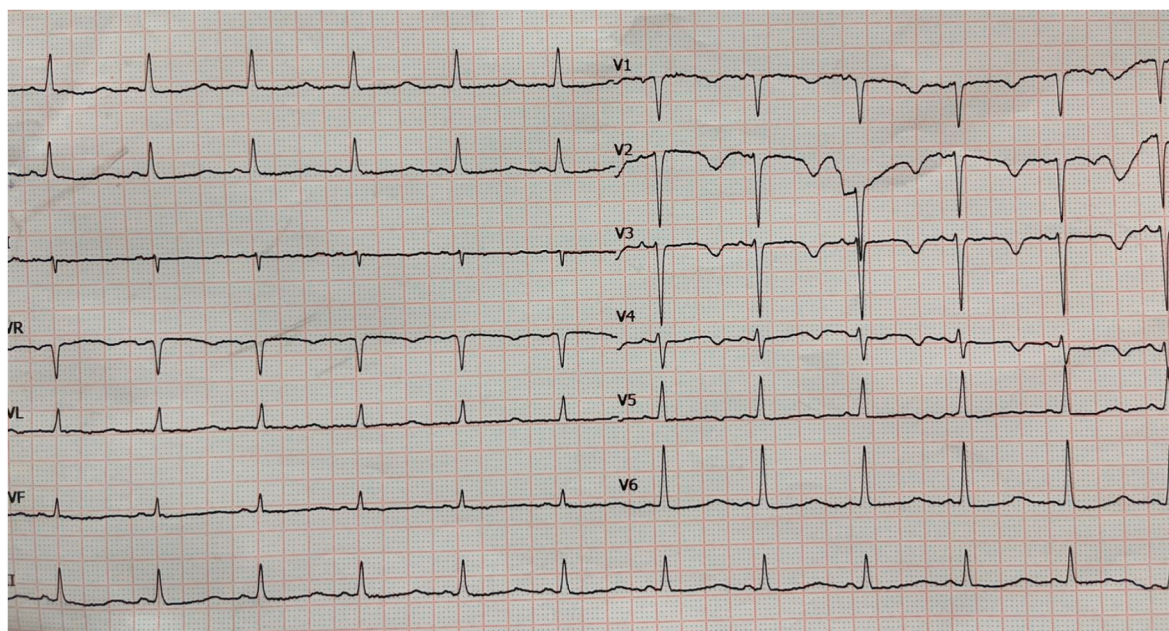


Fig. 1. The electrocardiogram (ECG) showing prolongation of the QT interval and T inversion in leads V1 to V4.

Myocardial dysfunction occurs in both chronic and acute hypocalcemia and has been reported in individuals ranging in age from infancy to old age [17]. The ECG of the current case showed a prolonged QT interval with T wave inversion.

In rare circumstances, the clinical, biochemical (elevation of cardiac enzymes), electrocardiographic, and echocardiographic data may lead to an incorrect diagnosis of an acute ischemic attack [18]. Heart failure of unknown cause and abrupt onset in a young patient offers a wide range of possible diagnoses. The most common cause is myocarditis, although drugs, drug abuse, and other hazardous factors should be thoroughly investigated, with a long list of compounds implicated [19]. Among the many causes of heart failure, hypocalcemia should not be ignored, and serum calcium should always be evaluated while examining plasma electrolytes, particularly in elderly patients on loop diuretics as these can cause calcium loss in the urine [10].

Hypocalcemia-induced heart failure is resistant to diuretics and digitalis [8]. Surprisingly, once serum calcium concentration is normalized, cardiac function and pulmonary artery pressure are completely restored to normal [20]. In almost all cases of hypocalcemic-induced heart failure, reversibility is a significant component of the cardiac dysfunction, including reversal of QT prolongation and restoration of left ventricular systolic function [11]. Cardiac function may take up to 6 months to return to normal [19]. Mikhael et al. reported a case where LVEF more than doubled in a seven-day period [21]. However, in rare situations, sustained severe hypocalcemia can cause structural damage and irreversibility, as in the case of Altunbaş et al. [22]. The pulmonary edema of the current case was treated with intravenous furosemide and oxygen. The hypocalcemia was treated with intravenous calcium gluconate infusion, alfacalcidol tablets, and magnesium sulfate infusion. Correction of serum calcium level was insufficient to restore myocardial function. It is believed that restoring intracellular calcium levels is more crucial than restoring myocardial function. Normalization of tissue calcium levels might take many months [14].

In conclusion, hypocalcemia as a possible cause of heart failure should be considered in the differential diagnosis of all individuals with congestive heart failure. The correction of serum calcium level results in the improvement of heart function and electrical activity.

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Author contributions

Abdulwahid M. Salh: major contribution of the idea, literature review, final approval of the manuscript. Dana H. Baqi, Shaho F. Ahmed: physicians managing the case, final approval of the manuscript. Fahmi H. Kakamad, Marwan N. Hassan: Writing the manuscript, literature review, final approval of the manuscript. Rawa M. Ali, Darko H. Amin H. Saed, Hiwa O. Baba, Fattah H. Fattah: literature review, final approval of the manuscript.

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Consent

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Declaration of competing interest

None to be declared.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.amsu.2022.103572>.

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