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Hypocalcaemic cardiomyopathy presenting as heart failure exacerbation due to untreated primary hypoparathyroidism

Background

Calcium plays a central role in the sequence of myocardial excitation–contraction coupling.[1,2] Low plasma calcium levels may lead to decreased contractility, resulting in prolonged hypocalcaemia that can culminate in ventricular dysfunction, cardiac dilatation and, in some cases, myocardial fibrosis.[3] Although hypocalcaemia is a common clinical problem, hypocalcaemic cardiomyopathy (HC) is a very rare cause of congestive heart failure (HF), with only a few cases reported in the literature.[4] The most common cause of HC in adulthood is primary hypoparathyroidism, often due to accidental damage to the parathyroid glands during thyroid surgery.[5,6] Traditional therapy for HF is generally ineffective and may even exacerbate symptoms.[7] As restoration to normal serum calcium levels usually leads to a rapid improvement in cardiac function,[8] it is essential to assess the underlying reversible aetiology in each patient with HF. We present the case of a patient who presented to our hospital with congestive HF, in which a diagnosis of HC was established following a comprehensive evaluation and a multidisciplinary approach.

Case description

A 54-year-old man, affected by mild untreated arterial hypertension and hypothyroidism treated with levothyroxine replacement therapy, presented to our hospital with progressive dyspnoea and orthopnoea [New York Heart Association (NYHA) class IV], accompanied by chest pain and cough with non-purulent expectoration. He was a light smoker and had been a moderate alcohol consumer in the past, but he had completely abstained from alcohol intake in the last year. He had undergone a total thyroidectomy for thyroid cancer 20 years earlier.

On admission, the patient was conscious and tachypneic and had a normal body temperature. His blood pressure was 160/110 mmHg, and oxygen saturation was 98% in room air. A physical examination revealed signs of pulmonary congestion. Cardiac auscultation demonstrated muffled heart sounds without any murmurs. Abdominal examination findings were within normal limits. An electrocardiogram (ECG) showed sinus rhythm, a heart rate of 83 b.p.m., signs of biatrial enlargement, lateral and inferior repolarization abnormalities and a QTc interval of 470 ms.

The polymerase chain reaction test for COVID-19 was negative. The chest X-ray revealed bilateral hilar enlargement, bilateral blunting of the costophrenic angles and an increased cardiothoracic index. The main laboratory test results are shown in *Table* 1. In summary, the laboratory examination at admission revealed a normal haemoglobin level, leucocytosis with neutrophilia, elevated liver enzymes, normal sodium and potassium levels and elevated brain natriuretic peptide (BNP). Lactate dehydrogenase (LDH) and creatine phosphokinase (CPK) were elevated, but high-sensitivity troponin was within normal physiological limits. C-reactive protein was high, and procalcitonin was normal. Thyroid-stimulating hormone (TSH) was within the normal range. Importantly, blood calcium and phosphorus levels were not tested upon admission. Transthoracic echocardiography (*Figure* 1, upper panel) revealed a dilated left ventricle with global hypokinesia, severe left ventricular (LV) systolic and diastolic dysfunction, a reduced LV ejection fraction (EF 19%) and left atrial dilatation. Mild-to-moderate tricuspid valve regurgitation with mild right ventricular dysfunction was also noted. The estimated pulmonary artery pressure was 46 mmHg.

Due to the exacerbation of HF, intravenous furosemide was administered, and disease-modifying pharmacological therapy with bisoprolol, sacubitril/valsartan and eplerenone was initiated and gradually up-titrated. On the second day of hospitalization, levosimendan was administered as a continuous 24 h infusion. A positive clinical response to medical therapy and progressive improvement in BNP, neutrophilia, C-reactive protein and liver function tests were observed (*Figure* 2). Due to the elevated liver enzymes and the patient's

Table 1 Blood tests during hospitalization and follow-up.

	Admission	After a week (detection of hypocalcaemia)	Last day before discharge	After 2 months	After 8 months
Haemoglobin, mg/dL	14.9	17.5	15.8	16.2	15.5
Platelets, $10^3/\mu L$	341	305		307	
Sodium, mmol/L	141	140	141	144	140
Potassium, mmol/L	3.80	3.88	3.78	3.43	3.9
Calcium, mmol/L		1.57	2.26	2.54	2.3
Ionized calcium, mmol/L		0.61	1.01		
Phosphorus, mg/dL		7.2		5.4	5.7
Magnesium, mg/dL		1.68	1.68	1.95	
PTH, pg/mL		8.8			
1,25-OH vitamin D, ng/mL		17		25	
Albumin, g/dL		3.08	3.08	3.87	
Creatinine, mg/dL	1.21	1.20	0.95	0.97	0.89
CPK, IU/L	494	263		97	
CK-MB, ng/mL	1.96				
High-sensitivity troponin I, ng/mL	15.68	5.22			2.66
LDH, IU/L	523	380	209	214	
AST, IU/L	53.0	26.6	24	20.8	
ALT, IU/L	101	44.5	45	34.2	
BNP, pg/mL	1888	499	133	195	36
C-reactive protein, mg/dL	22.6	8.9			5.3
Procalcitonin, ng/mL	0.25				
TSH, μIU/mL	3.186			4.010	

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BNP, brain natriuretic peptide; CK-MB, creatine kinase-MB; CPK, creatine phosphokinase; LDH, lactate dehydrogenase; PTH, parathyroid hormone; TSH, thyroid-stimulating hormone.

Figure 1 Echocardiography findings at diagnosis and at follow-up. The figure shows the echocardiographic variables of the patient during hospitalization (upper panel) and at follow-up (lower panel), highlighting the left ventricular reverse remodelling together with haemodynamic improvement. 2D-LV, left ventricular volumes at a two-dimensional evaluation; 3D-LV, left ventricular volumes at a three-dimensional evaluation; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; GLS, global longitudinal strain; LA, left atrium; MR, mitral regurgitation; TAPSE, tricuspid annular plane systolic excursion.

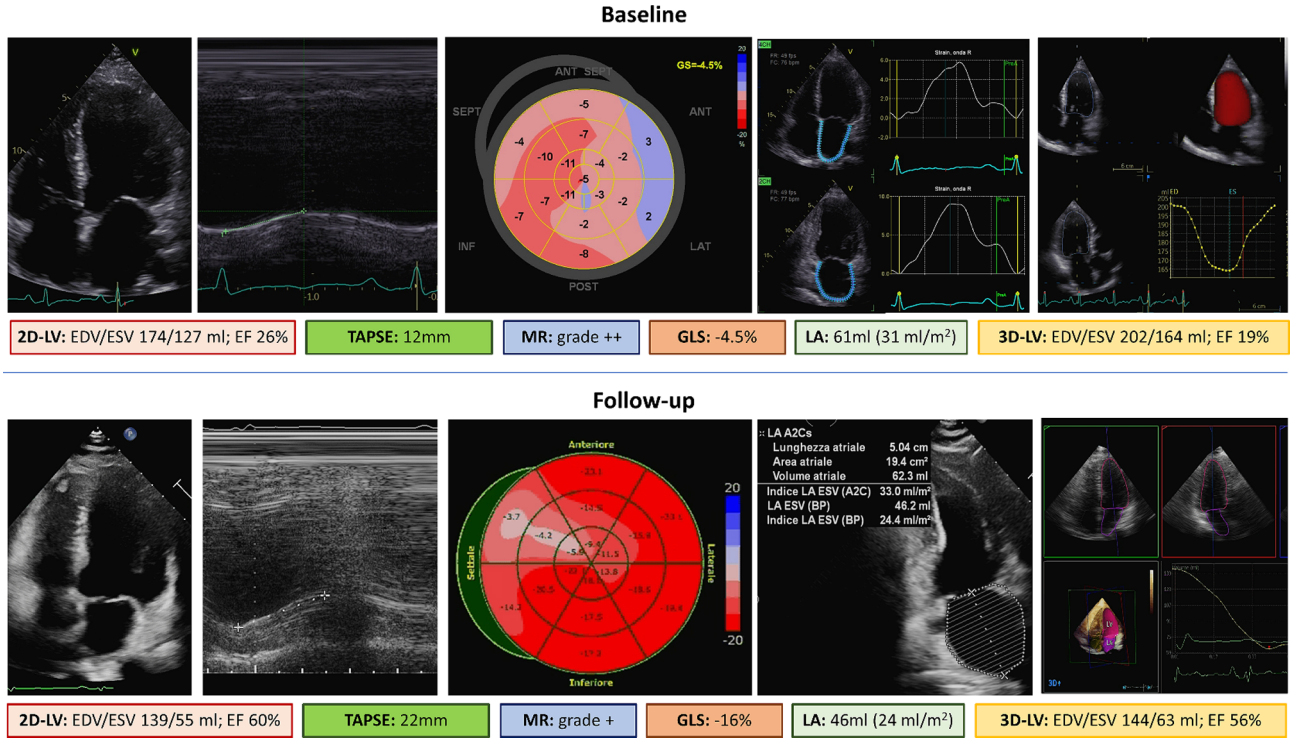

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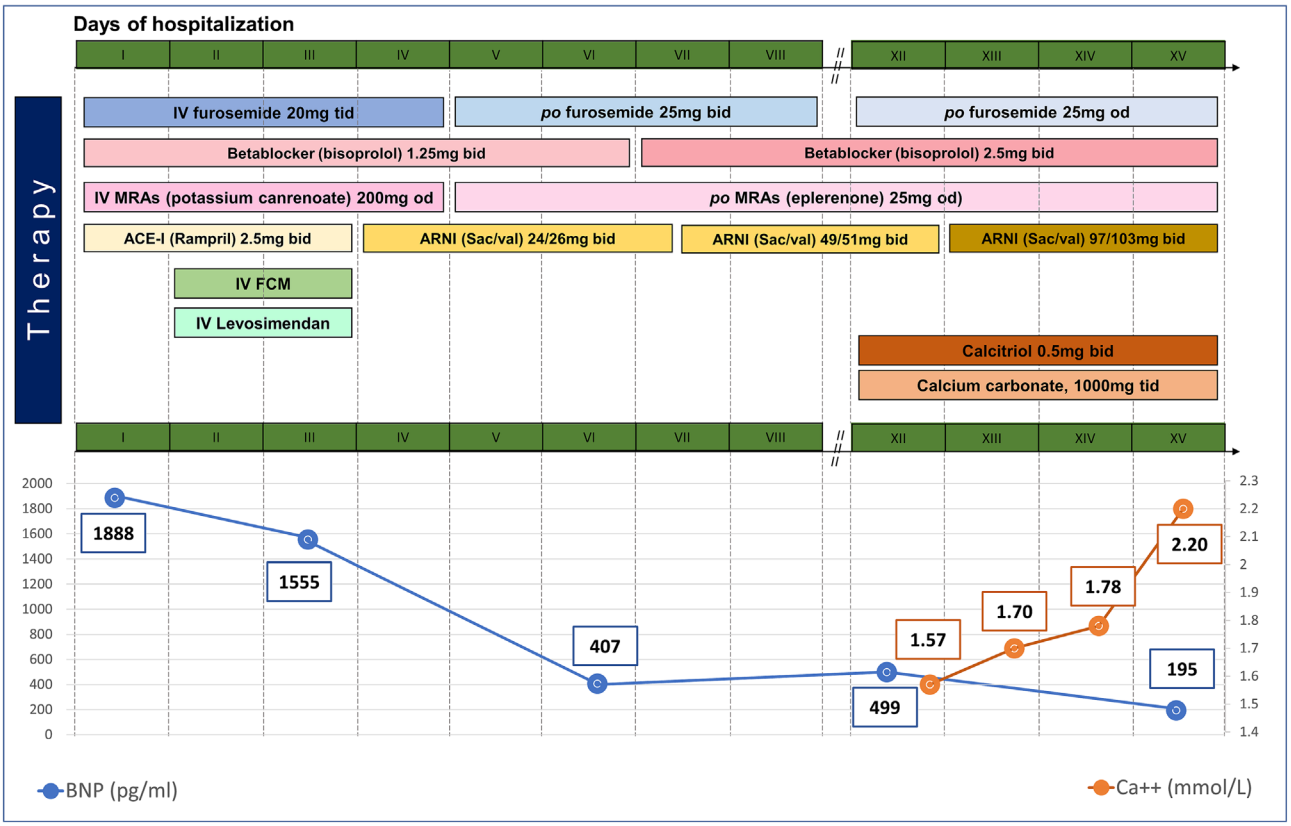
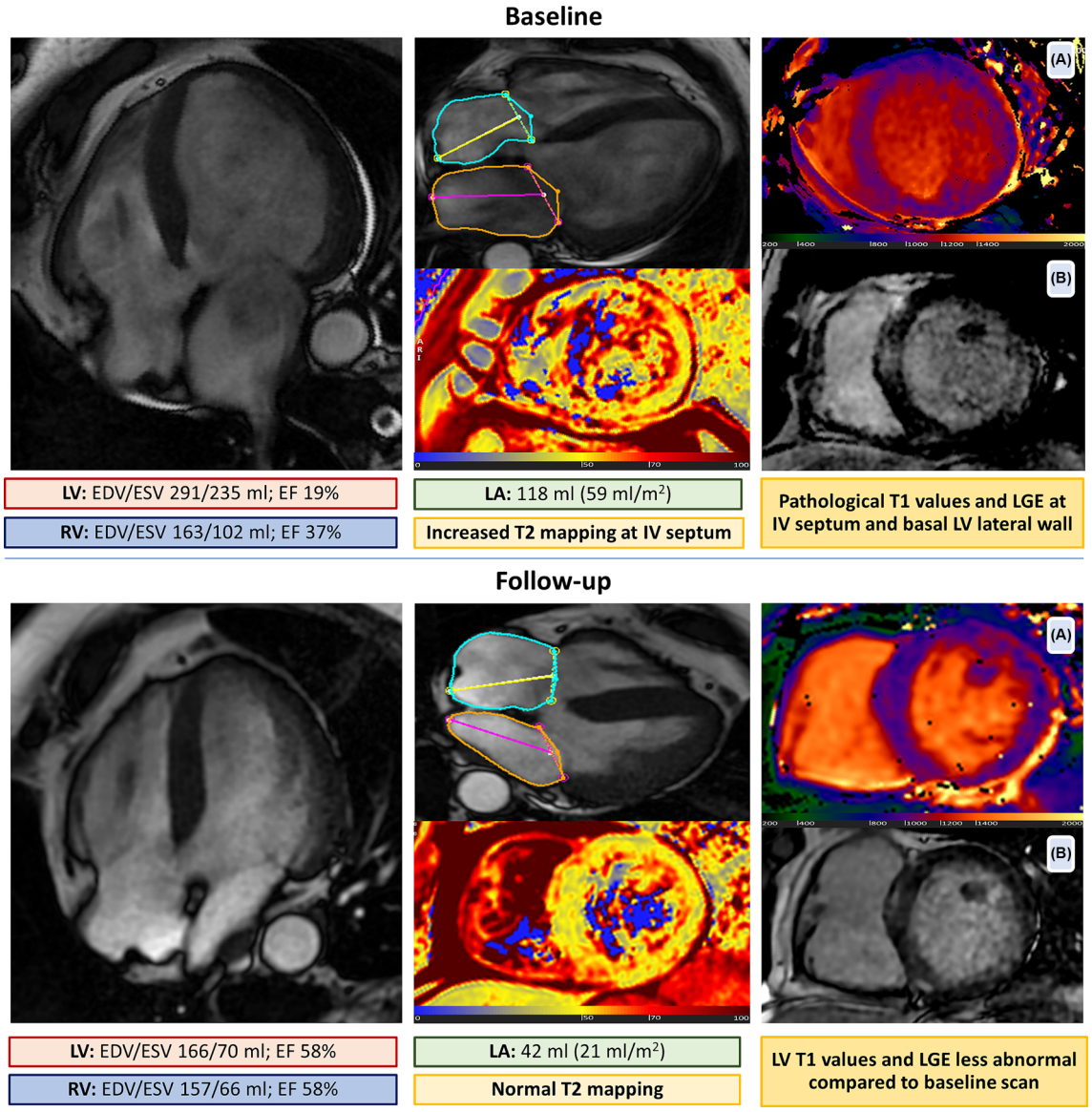
Figure 2 Therapy regimen and brain natriuretic peptide (BNP) values during hospitalization. The figure shows the therapy regimen during hospitalization (upper panel) and BNP and calcium values in blood tests (lower panel). After the acute phase, the heart failure therapy was up-titrated rapidly to the maximum tolerated dose, with a progressive reduction in BNP values. In the second phase of the hospitalization, a replacement therapy was started with calcium supplementation with normalization of the serum values. ACE-I, angiotensin-converting enzyme inhibitor; ARNI, angiotensin receptor neprilysin inhibitor; FCM, ferric carboxymaltose; IV, intravenous; MRAs, mineralocorticoid receptor antagonists; PO, per os.



history of moderate alcohol consumption, abdominal ultrasonography was performed, revealing a mildly steatotic liver with regular size and margins; the remaining organs that could be explored by this exam were within normal limits. A coronary computed tomography (CT) scan ruled out significant coronary lesions. Cardiac magnetic resonance (CMR) imaging confirmed severe LV systolic dysfunction, demonstrated increased native T1–T2 values and revealed an intramyocardial 'patchy' distribution of late gadolinium enhancement (LGE) involving the interventricular septum and basal LV lateral wall (*Figure* 3, upper panel). To exclude active myocarditis, the patient underwent an electroanatomic mapping-guided endomyocardial biopsy (EMB) of the interventricular septum, which ruled out the presence of viral DNA/RNA and signs of active inflammation (*Figure* 4). Additionally, programmed electrical stimulation was conducted, revealing the absence of the inducibility of sustained ventricular arrhythmias. Cardiopulmonary exercise testing (CPET) showed severe functional capacity impairment and periodic breathing during incremental exercise, which is a marker of HF severity,[9] disappearing at the anaerobic threshold (*Figure* 5, upper panel). After 1 week, despite a progressive improvement in HF status, the patient reported an episode of dysarthria, confusion and muscle spasms. An urgent brain CT scan was performed, ruling out acute ischaemic or haemorrhagic events but revealing diffuse basal ganglia calcification (*Figure* 6A).

Blood tests revealed severe hypocalcaemia in the setting of primary hypoparathyroidism [total calcium 1.57 mmol/L, ionized calcium 0.61 mmol/L, phosphorus 7.2 mg/dL and parathyroid hormone (PTH) 8.8 pg/mL]. Further investigation showed serum albumin within the normal range and low vitamin D levels. Trousseau's sign (also known as 'obstetrician's hand') was positive, suggesting the presence of latent tetany (*Figure* 6B). The QTc interval on the ECG was 522 ms, which was subsequently normalized. Based on the low serum calcium level and cardiac imaging findings, a diagnosis of HF due to hypocalcaemia was strongly suspected. The patient

Figure 3 Cardiac magnetic resonance (CMR) findings at diagnosis and at follow-up. The figure shows the CMR variables of the patient during hospitalization (upper panel) and at follow-up (lower panel). A favourable reverse remodelling of both the left and right ventricles (LV and RV) is shown, together with an improvement in tissue characterization. EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; IV, interventricular; LA, left atrium; LGE, late gadolinium enhancement.



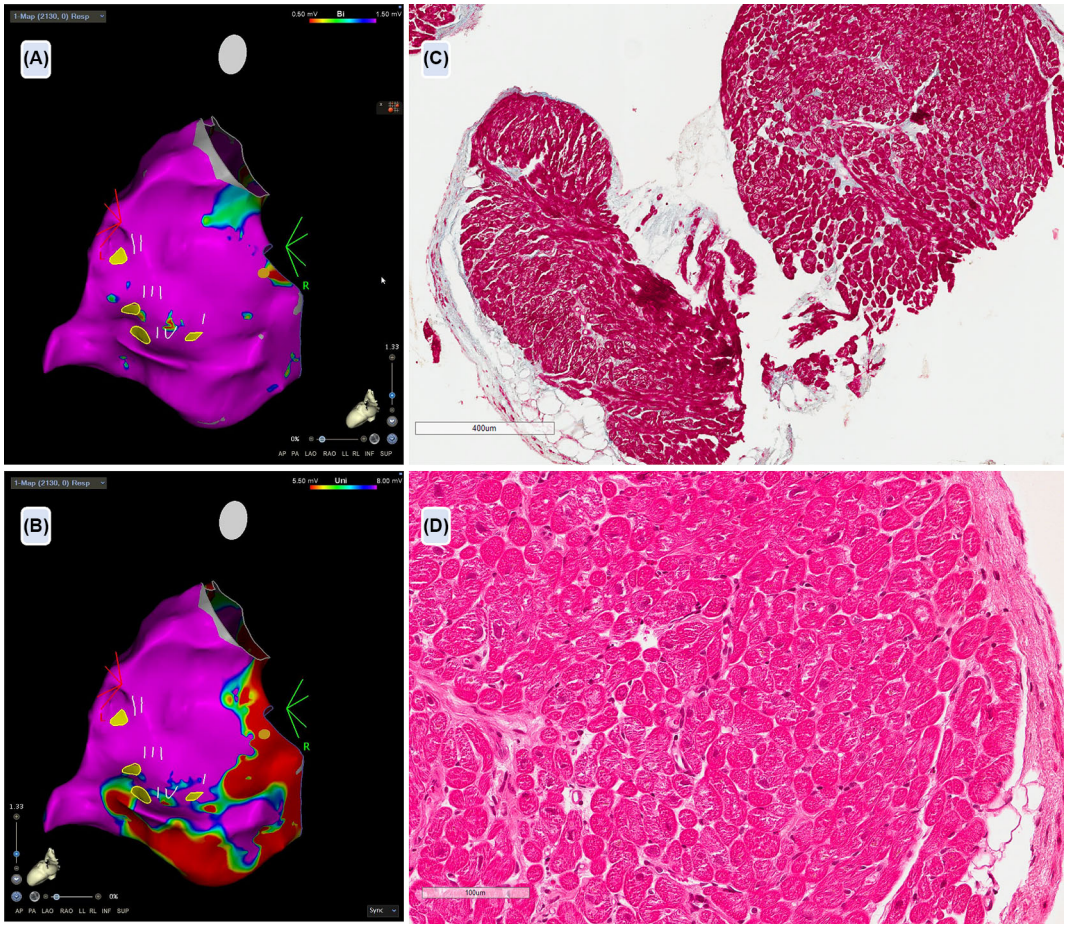
was promptly treated with intravenous calcium gluconate, followed by oral calcium carbonate and calcitriol. Neuromuscular symptoms completely resolved after normalization of calcium levels. After 5 days, the patient was discharged in good clinical condition.

We observed a progressive improvement in biventricular systolic function, with complete normalization of LVEF, ventricular volumes, exercise capacity and cardiac biomarkers at 9 months (*Figures* 1, 3 and 5, lower panels), as also confirmed by follow-up CMR. After 2 years, the patient is still symptomatic (NYHA I) with stable echocardiographic data and persistently low BNP levels. No drug therapy has been changed. A genetic test was performed, excluding pathogenic/likely pathogenic genetic variants.

Discussion

This case report describes a reversible form of HF and dilated cardiomyopathy (DCM), highlighting the importance of establishing the correct aetiology to promptly initiate appro-

Figure 4 Electroanatomic mapping-guided endomyocardial biopsy (EMB). The left part of the figure shows the electroanatomic mapping of the right ventricle. No scar areas were detected at endocardial (Panel A) and epicardial (Panel B) voltage mapping. Yellow dots indicate the localization of biopsies in the interventricular septum. The right part of the figure shows, on Azan–Mallory (Panel C) and haematoxylin–eosin (Panel D, higher magnification) staining, a histological image of right ventricular tissue that shows enlargement of myocyte diameter and absence of inflammation. EMB also ruled out the presence of viral DNA/RNA.



priate therapy and alter the natural course of the disease. Hypocalcaemia is a rare but reversible cause of HF and cardiomyopathy.[7,8,10] Calcium plays a crucial role in normal ventricular systolic and diastolic function by binding to the troponin–tropomyosin complex, facilitating actin–myosin sliding and jointing.[11] Additionally, recent evidence suggests that PTH may have an independent role. PTH has been shown to exert a positive chronotropic effect on cardiomyocytes. Several case reports have demonstrated that isolated hypoparathyroidism is associated with significant, reversible LV systolic dysfunction.[12,13] Investigation of hypocalcaemia in our patient led to a diagnosis of primary hypoparathyroidism, characterized by low or absent circulating PTH levels, resulting in hypocalcaemia and hyperphosphataemia. The most common cause of primary hypoparathyroidism is inadvertent damage to the parathyroid glands during thyroid surgery.[14] In the present case, the patient's medical history, laboratory findings and complete recovery of contractile function following treatment suggested that long-term hypocalcaemia was the cause of cardiomyopathy (HC). However, several other aetiologies were considered and subsequently ruled out. Ischaemic cardiomyopathy was excluded due to patent coronary arteries and the absence of CMR findings indicative of previous myocardial infarction. Although the patient had a history of moderate alcohol consumption and elevated liver enzymes upon admission, an alcoholic aetiology was considered unlikely as the patient reported abstaining from alcohol for over a year. Furthermore, normalization of liver tests after medical therapy and the absence of significant liver disease on abdominal ultrasonography argued against this possibility. While CMR revealed myocardial oedema, myocardial biopsy did not show signs of active inflammation, thus excluding DCM secondary to myocarditis. Although a specific secondary cause was identified, it is important to note that an idiopathic origin of DCM cannot be definitively ruled out. Therefore, we opted to continue

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Figure 5 Cardiopulmonary exercise testing (CPET) findings at diagnosis and at follow-up. The figure shows the CPET variables of the patient during hospitalization (upper panel) and at follow-up (lower panel). Remarkably, an amelioration of all the CPET strong prognostic parameters is shown with a periodic breathing (PB) disappearance. AT, anaerobic threshold; PVO_2, peak oxygen intake; RER, respiratory exchange ratio; VE/VCO_2, minute ventilation-to-carbon dioxide output.

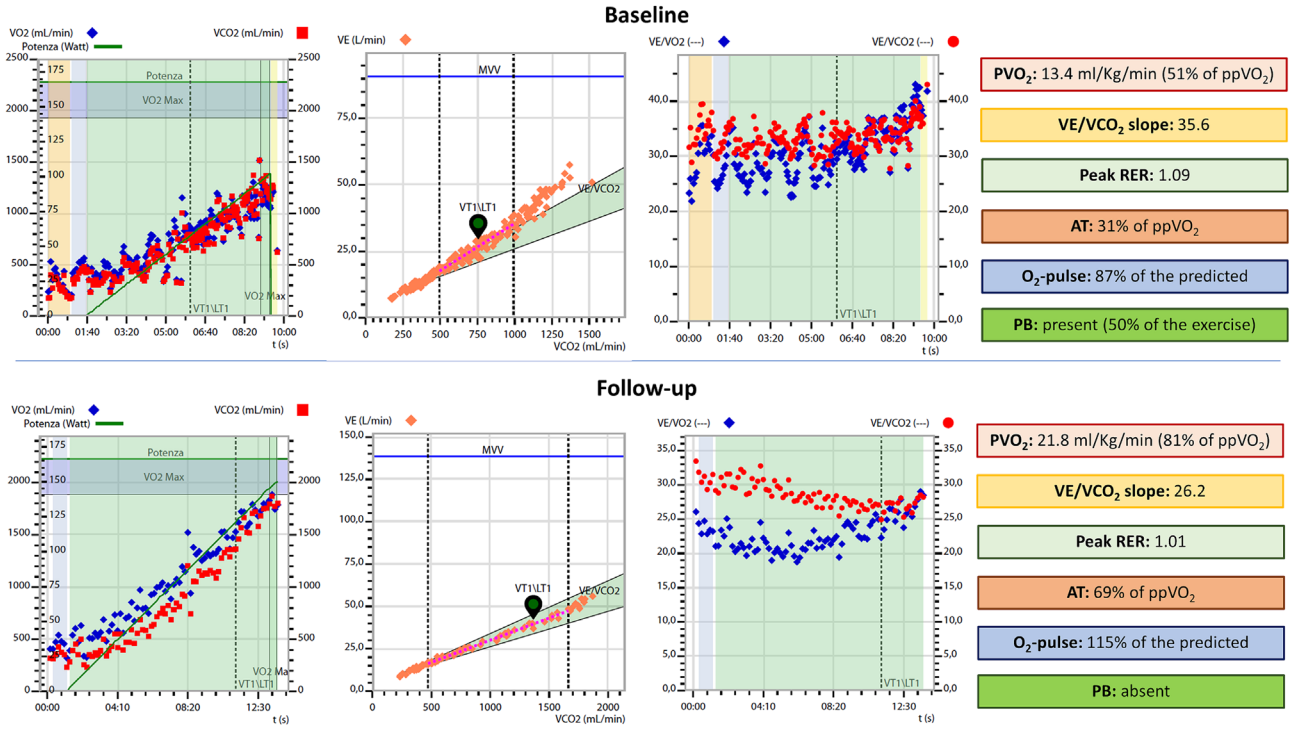
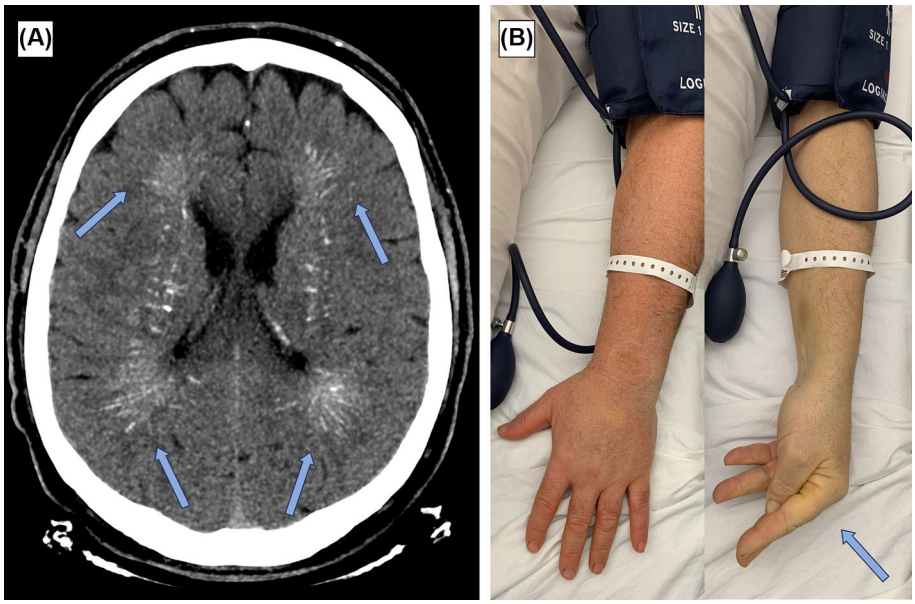


Figure 6 Brain computed tomography (CT) and Trousseau's sign. The figure shows the brain CT findings consistent with Fahr's syndrome (Panel A) and Trousseau's sign (Panel B).



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anti-remodelling cardiac therapy indefinitely, as recommended by international HF guidelines.[15] HC presents as an acute and transient form of DCM with reduced EF and diffuse LV hypokinesia.[7] Despite its rarity in clinical practice, HC may result from early recognition and appropriate treatment of hypocalcaemia. Patients with HC typically exhibit symptoms of hypocalcaemia such as neuromuscular irritability, perioral paresthesias, tingling in the extremities, neuropsychiatric symptoms and spontaneous or latent tetany.[16] In the present case, we observed the presence of Trousseau's sign as a marker of latent tetany, which manifests as a carpopedal spasm induced by ischaemia secondary to the inflation of a sphygmomanometer cuff. Carpopedal spasm is characterized by flexion of the thumb and metacarpophalangeal joints with hyperextension of the fingers.[17] We believe that the elevated levels of CPK and LDH during hypocalcaemia originated from the skeletal muscle, as there was no elevation in creatine kinase-MB (CK-MB) or troponin. The ECG hallmark of hypocalcaemia is QT interval prolongation secondary to prolonged ST segment, which predisposes to ventricular arrhythmias.[18] This is a consequence of the increased duration of phase 2 of the action potential in cardiac muscle.[19] Calcification of the basal ganglia typically indicates a chronic hypocalcaemic state. The association between bilateral basal ganglia calcification and neuromuscular symptoms defines the clinical entity of Fahr's syndrome or Fahr's disease. Fahr's disease mainly refers to idiopathic forms in which no metabolic or other underlying causes are identified. Recently, genetic mutations have been identified. Fahr's syndrome refers to secondary forms such as infections, toxic exposures or, as in our case, endocrine abnormalities involving PTH.[20,21] The underlying pathophysiology appears to be related to abnormal calcium/phosphorus homeostasis.[22] A CT scan is considered the gold standard for diagnosis, identifying calcifications as hyperdense lesions that are typically bilateral and symmetrical.[20] Conventional therapy for acute HF, including vasodilators and diuretics, is not adequately effective in HC.[7,23] Furosemide can exacerbate hypocalcaemia by increasing urinary calcium excretion.[24] Regarding our case, we believe that the patient might have had mild or moderate hypocalcaemia because he had no symptoms of hypocalcaemia before hospitalization, and we were unaware of the initial calcium concentration. Severe hypocalcaemia might have been induced by intravenous furosemide during the initial HF treatment. However, unlike many cases reported in the literature, we observed clinical improvement in symptoms related to pulmonary congestion and BNP values, suggesting that treatment with furosemide could be considered after restoring the correct serum calcium values. Early correction of hypocalcaemia has a remarkable effect on the therapy of HC, and cardiac function generally improves after normalization of calcium levels. Supplementation with calcium and vitamin D remains the standard treatment to ensure normocalcaemia and heart function recovery.[4,7,25,26] Restoration of ventricular function and chamber size may take a few months to normalize.[25] However, prolonged and profound hypocalcaemia may sometimes cause irreversible myocardial damage and extensive interstitial fibrosis, making complete recovery of cardiac function impossible.[3,27] In the present case, we observed a complete recovery of biventricular systolic function, but on CMR, LGE in the interventricular septum persisted after several months, suggesting a chronic hypocalcaemic state. CMR provides additional information on tissue characterization in patients with DCM, including the presence of myocardial oedema and LGE.[28,29] When CMR reveals oedema and LGE in HC, an EMB may be considered for a definitive diagnosis.[30] A biopsy would further help differentiate CMR changes that are due to coexisting myocarditis or are secondary to HC. In this complicated context, a negative genetic test, although not totally diriment, can be a supportive element in ruling out a genetically determined form of DCM, making an acquired condition (i.e., an HC) more likely, also given the clinical context.

Conclusions

We presented a case that exemplifies a multidisciplinary approach, highlighting the role of hypocalcaemia as a rare but reversible cause of DCM. HC should be suspected when a patient presents with myocardial dysfunction, neuromuscular signs and QT prolongation on the ECG. Normalization of calcium concentration could lead to a complete recovery of cardiac function. Administration of diuretics, such as furosemide, may exacerbate hypocalcaemia by increasing urinary calcium excretion. It is important to assess plasma calcium and phosphorus levels in every patient with DCM and unexplained HF. A multiparametric approach to the evaluation of patients with suspected cardiomyopathy is recommended to characterize the cardiomyopathy phenotype, identify the aetiological diagnosis, establish the prognosis and guide treatment.

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Conflict of interest statement

The authors declare no conflicts of interest.

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