Journal of Cardiology Cases 10 (2014) 58–61

Contents lists available at www.sciencedirect.com

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

A case of massive pericardial effusion associated with hypocalcemic cardiomyopathy

Tetsuro Yokokawa (MD)[a,*], Keiji Sakamoto (MD)[a], Hiroyuki Mizuno (MD)[a], Yasuhiro Shimizu (MD)[a], Yuko Matsui (MD)[a], Hironori Kaneko (MD, PhD)[a], Yuichi Ujiie (MD, PhD)[a], Eisuke Miura (MD, FJCC)[a], Yoshitane Seino (MD, PhD)[a], Mikihiro Kijima (MD, PhD, FJCC)[a], Yukio Maruyama (MD, PhD, FJCC)[a], Yasuchika Takeishi (MD, PhD, FJCC)[b]

[a] Department of Cardiology, Hoshi General Hospital, Koriyama, Japan
[b] Department of Cardiology and Hematology, Fukushima Medical University, Fukushima, Japan

ARTICLE INFO

Article history:
Received 24 March 2014
Received in revised form 18 April 2014
Accepted 2 May 2014

Keywords:
Heart failure
Hypocalcemia
Hypocalcemic cardiomyopathy
Idiopathic hypoparathyroidism
Pericardial effusion

ABSTRACT

A 60-year-old woman with a 6-year history of numbness in her hands was admitted to hospital with dyspnea. Laboratory findings showed the elevation of creatine kinase (creatine kinase MB isoenzyme was less than 4 IU/l). Chest X-ray revealed cardiomegaly and pulmonary edema. Electrocardiogram showed a T wave inversion in V_{2-5} and a prolonged QT interval. Echocardiography demonstrated reduced left ventricular ejection fraction (LVEF) and massive pericardial effusion. The patient was diagnosed with heart failure. Further testing found hypocalcemia and idiopathic hypoparathyroidism. In addition to diuretics, calcium replacement therapy for hypocalcemia improved the LVEF and reduced pericardial effusion. Hypocalcemia rarely leads to heart failure and pericardial effusion. In our case, heart failure and the massive pericardial effusion were secondary to hypocalcemia due to idiopathic hypoparathyroidism.
<**Learning objective:** Hypocalcemia should be considered in patients with heart failure, reduced ejection fraction, and massive pericardial effusion. Supportive findings for diagnosis of heart failure caused by hypocalcemia are numbness, elevation of creatine kinase, T wave inversion, and prolonged QT interval.>

Introduction

Calcium is essential for the heart. Previous studies have revealed that hypocalcemia is one unusual cause of heart failure [1–7], and rarely leads to pericardial effusion [2,4,7,8]. Herein we present a case of heart failure and massive pericardial effusion, which were secondary to hypocalcemia due to idiopathic hypoparathyroidism.

Case report

A 60-year-old woman was admitted to a local hospital complaining of dyspnea, and upon admission she was suspected of having ischemic heart disease. Her electrocardiogram showed T wave inversion and her serum creatine kinase was elevated. She had suffered from numbness in her hands for 6 years, and had a documented history of cataract since 5 years earlier. She was referred to our hospital for further examination and treatment. On admission, her blood pressure was 115/82 mmHg, heart rate was 103 bpm, body temperature was 37.4 °C, and room air oxygen saturation was 93%. On physical examination, a grade 1 systolic murmur was heard at the left sternal border. Trousseau sign was present. Laboratory findings before treatment of diuretics were as follows: aspartate aminotransferase, 36 IU/l; lactate dehydrogenase, 670 IU/l; creatine kinase (CK), 1548 IU/l; CK-MB (MB isoenzyme of creatine kinase), less than 4 IU/l; calcium, 4.2 mg/dl; inorganic phosphorus, 11.9 mg/dl; magnesium, 1.4 mg/dl; albumin, 4.18 g/dl; brain natriuretic peptide, 466.6 pg/ml; thyroid-stimulating hormone, 0.475 U/ml; free thyroxine, 1.74 ng/dl; free triiodothyronine, 2.63 pg/ml (Table 1). A chest X-ray showed cardiomegaly, bilateral pleural effusion, and pulmonary edema. Electrocardiogram detected sinus tachycardia, T wave inversion in V_{2-5}, and a prolonged QT interval with QTc 623 ms (Fig. 1a). Echocardiography revealed left ventricular dysfunction with a left ventricular ejection fraction (LVEF) of 31.0% by a modified Simpson's method, a

* Corresponding author at: Department of Cardiology, Hoshi General Hospital, 159-1 Mukaigawaramachi, Koriyama 963-8501, Japan. Tel.: +81 24 983 5511; fax: +81 24 983 5588.
E-mail address: yokotetu@fmu.ac.jp (T. Yokokawa).

http://dx.doi.org/10.1016/j.jccase.2014.05.003
1878-5409/

Table 1
Laboratory findings before treatment of diuretics.

TP	7.1 g/dl	Ca^{2+}	2.2 mg/dl	BNP	446.6 pg/ml
Alb	4.18 g/dl				
AST	36 U/l	CK	1548 IU/l	TSH	0.475 μIU/ml
ALT	20 U/l	CK-MB	Less than 4 IU/l	FT_4	1.74 ng/dl
LD	670 U/l			FT_3	2.63 pg/ml
ALP	201 U/l	CRP	0.17 mg/dl		
Γ-GTP	10 U/l			Intact PTH	5 pg/dl
T-Bil	1.05 mg/dl	FBS	91 mg/dl	25-(OH)-D	14.0 ng/ml
		HbA1c	5.9% (NGSP)		
BUN	18.7 mg/dl			*Blood gas analysis (room air)*	
Cre	0.97 mg/dl	PT	64%	pH	7.440
Na	144 meq./l	PT-INR	1.20	pCO_2	35.0 mmHg
K	3.9 meq./l	APTT	34.5 s	pO_2	47.8 mmHg
Cl	101 meq./l			HCO_3^-	23.4 meq./l
Ca	4.2 mg/dl	WBC	$6.6 \times 10^3/\mu l$		
IP	11.9 mg/dl	RBC	$3.75 \times 10^6/\mu l$		
Mg	1.4 mg/dl	Hb	11.5 g/dl		
		PLT	$19.7 \times 10^3/\mu l$		

TP, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LD, lactate dehydrogenase; ALP, alkaline phosphatase; γ-GTP, γ-glutamyl transpeptidase; T-Bil, total bilirubin; BUN, blood urea nitrogen; Cre, creatinine; IP, inorganic phosphate; CK, creatine kinase; CK-MB, MB isoenzyme of creatine kinase; CRP, C-reactive protein; FBS, fasting blood sugar; HbA1c, hemoglobin A1c; PT, prothrombin time; PT-INR, prothrombin time-international normalized ratio; APTT, activated partial thromboplastin time; WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; PLT, platelet; BNP, brain natriuretic peptide; TSH, thyroid-stimulating hormone; FT_4, free thyroxine; FT_3, free triiodothyronine; Intact PTH, intact parathyroid hormone; 25-(OH)-D, 25-hydroxyvitamin D.

suspected bicuspid aortic valve with a peak velocity of 2.5 m/s, moderate mitral regurgitation, and massive pericardial effusion (over 10 mm surrounding the entire heart with no sign of tamponade; Fig. 2a). Chest computed tomography to evaluate the amount of pericardial effusion also showed pleural effusion and massive pericardial effusion (Fig. 3a). Treatment was initiated for heart failure with diuretics including carperitide, azosemide, furosemide, and spironolactone. On day 6 after admission, although alfacalcidol administration had already been started, the patient experienced a sudden convulsion due to low serum calcium level. Head computed tomography revealed intracranial calcification in the basal ganglia. Intravenous calcicol and calcium aspartate were then added for treatment of hypocalcemia after head computed tomography. The intact parathyroid hormone was found to be 5 pg/ml (normal range 10–65 pg/ml), and 25-hydroxyvitamin D was 14.0 pg/ml (normal range 9–33.9 ng/ml). Thus, autoimmune syndrome, polyglandular syndrome, adrenal insufficiency, and cancer were excluded, and idiopathic hypoparathyroidism was diagnosed. Diuretics were discontinued on day 7. As the patient's serum calcium level was increased, the numbness in her hands disappeared and heart failure was improved. CK level was decreased to within normal range. Her LVEF was improved to 44.7% by a modified Simpson's method of echocardiography and pericardial effusion was reduced. Cardiac catheterization showed no significant coronary lesions. On day 29, laboratory data after administration of calcium showed intact parathyroid hormone was 1 pg/ml. On day 30, she was discharged. The patient was followed up on day 87 with some examinations performed to assess her conditions. Laboratory data showed almost normal levels of serum calcium and brain natriuretic peptide. Electrocardiogram showed nonspecific ST–T wave abnormalities and normal QT interval with QTc 363 ms (Fig. 1b). Echocardiography showed almost normal function of the left ventricle (Figs. 2b and 4). Chest computed tomography showed the improvement of cardiomegaly and pericardial effusion (Fig. 3b). Left ventricular dysfunction and massive pericardial effusion were secondary to low calcium level due to idiopathic hypoparathyroidism.

Discussion

Hypocalcemia is a rare cause of heart failure and heart failure induced by hypocalcemia is known as hypocalcemic cardiomyopathy [1–7]. Calcium, which plays a vital role in myocardial excitation–contraction coupling, enters the cell and triggers calcium release from the sarcoplasmic reticulum. Intracellular calcium binds to troponin, which leads to contraction, and calcium dissociates from troponin for relaxation [9]. The mechanism of hypocalcemic cardiomyopathy has not been fully investigated. Previously reported cases of patients with hypocalcemic cardiomyopathy suffered from hypocalcemia over several years and the duration of hypocalcemia was regarded as an important factor that caused hypocalcemic cardiomyopathy [1–3,6,7].

It has been reported that hypocalcemia associated with hypoparathyroidism leads to pericardial effusion in a patient

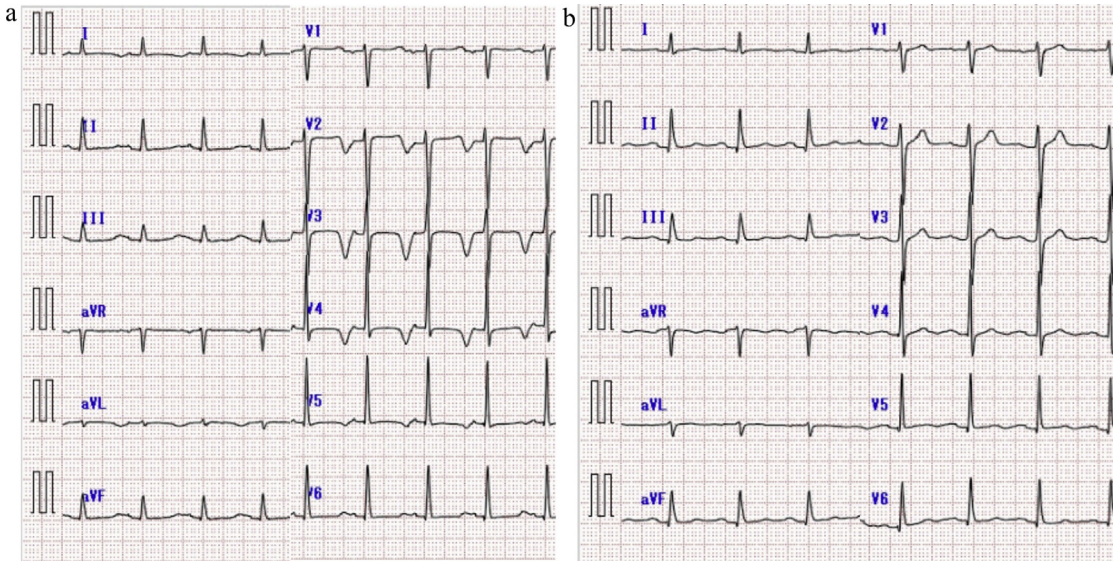

Fig. 1. Electrocardiograms. (a) On admission, electrocardiogram detected sinus tachycardia, T wave inversion in V_{2-5}, and prolonged QT interval with QTc 623 ms. (b) On day 87, electrocardiogram showed nonspecific ST–T wave abnormalities and normal QT interval with QTc 363 ms.