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Clinical Improvement in a Young Patient with Hypertrophic Obstructive Cardiomyopathy Treated with Combination Therapy Including Amiodarone

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Abstract

There has not been an evidence-based medication established for pediatric hypertrophic obstructive cardiomyopathy. We report the clinical details and treatment of a 14-year-old boy with heart failure caused by hypertrophic obstructive cardiomyopathy, which was first diagnosed when he was an infant. He complained of breathlessness while climbing stairs and chest pain. Cibenzoline, 450mg, and metoprolol, 60mg, were administered. Although his symptoms disappeared, the left ventricular outflow tract obstruction did not improve. He had a MYH7 (β-Cardiac Myosin Heavy Chain) mutation [c.2609G>T (p.Arg 870Leu)] and an interventricular wall thickness of more than 30 mm. When he was 17 years old, amiodarone, 100mg, was started for the prevention of ventricular arrhythmia and sudden death. After 7 years, the maximum peak flow at the left ventricular outflow tract decreased from 5.1m/s to 1.5m/s, and his brain natriuretic peptide level went from 310 pg/ml to 8 pg/ml. The New York Heart Association class improved from the second to the first grade. There were no side effects for each medication. The pressure gradient in the left ventricular outflow tract and heart failure in this patient with hypertrophic obstructive cardiomyopathy improved with combination therapy including amiodarone.

Keywords: Hypertrophic obstructive cardiomyopathy; Heart failure; MYH7 (β-cardiac myosin heavy chain); Amiodarone; Cibenzoline

Introduction

Child-onset hypertrophic cardiomyopathy is rare compared with adult-onset disease. When presenting during childhood, the disease affects mortality [1]. Sarcomeric variants were more common in childhood-onset hypertrophic cardiomyopathy and carried a worse prognosis. The survival rate for infant-onset idiopathic hypertrophic cardiomyopathy at 10 years was 85.3% [2]. Furthermore, hypertrophic obstructive cardiomyopathy is a form of hypertrophic cardiomyopathy, a genetic condition where the ventricular wall becomes abnormally thick. In hypertrophic obstructive cardiomyopathy specifically, the thickened septum causes dynamic obstruction of the left ventricle and leads to heart failure and sudden death [1,3]. However, there have been no reports of improvement in heart failure with medication, except mavacamtem in pediatric hypertrophic obstructive cardiomyopathy patients [4]. It is a reversible cardiac myosin ATPase inhibitor developed to target excessive myosin binding seen in hypertrophic cardiomyopathy [5].

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Although mavacamten has been used in patients with hypertrophic obstructive cardiomyopathy, its clinical use for Japanese pediatric hypertrophic obstructive cardiomyopathy has not yet been started. No evidence-based medication has been established for pediatric hypertrophic obstructive cardiomyopathy [6]. On the other hand, it is known that cibenzoline improves the pressure gradient of the left ventricular outflow tract in patients with hypertrophic obstructive cardiomyopathy [7-9]. Furthermore, amiodarone is reported to prevent ventricular arrhythmias in adult patients [10]. However, amiodarone has some severe side effects, and its use for hypertrophic obstructive cardiomyopathy in children is not so common. We report a 10-year clinical improvement with combination therapy including amiodarone in an adolescent hypertrophic obstructive cardiomyopathy patient.

Case Report

A boy was diagnosed with hypertrophic cardiomyopathy at 1 month, because of a heart murmur. Carteolol was administered at the age of 3 months. At 6 months, the pressure gradient of the left ventricular outflow tract by two-dimensional echocardiography was 53 mmHg, and he was diagnosed with hypertrophic obstructive cardiomyopathy. He had no familial history of cardiomyopathy. His brain natriuretic peptide levels at 5 years, 8 years, and 10 years were 8 pg/ml, 85 pg/ml, and 265 pg/ml, respectively. He complained of breathlessness when climbing stairs and chest pain. Verapamil, 120 mg, was taken in addition to carteolol, 10mg. He could not go to school because of fatigue when he was 12 years old. The maximum peak flow of the left ventricular outflow tract was 4.5 m/s, and his brain natriuretic peptide level was 310 pg/dl. Couplets were detected on his Holter electrocardiogram.

He was referred to our hospital at the age of 14 years. A systolic heart murmur and S3 gallop heart sound rhythm were auscultated on admission. His height and weight were 153 cm and 62.5kg, respectively. The troponin T level was 0.066 ng/ml. The cardiothoracic ratio was 62% on the chest X-ray (Figure 1).

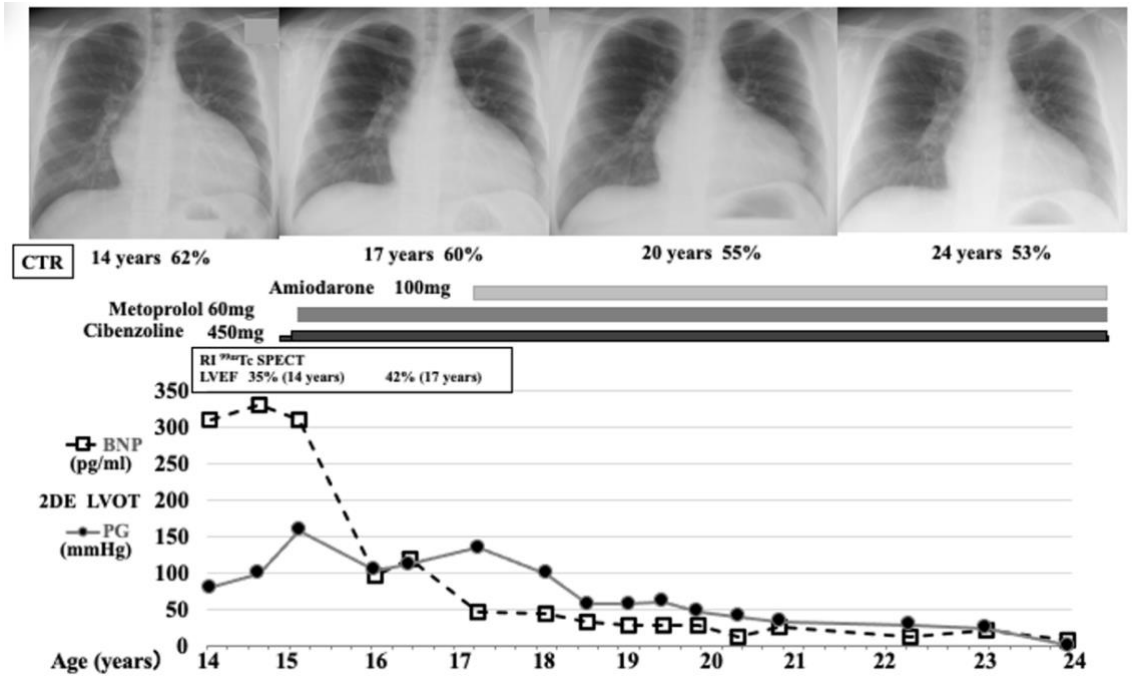

Figure 1: Clinical course in a pediatric hypertrophic obstructive cardiomyopathy patient who underwent combined medication therapy.

CTR: Cardiothoracic ratio; **BNP:** Brain natriuretic peptide; **LVOT:** Left ventricular outflow tract; **PG:** Pressure gradient; **SPECT:** Single photon emission computed tomography.

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Negative T waves in leads I, II, aV_L, aV_F, and V_5~V_6, wide QRS waves, and high voltage of R waves in V_5~V_6 were observed on the 12-lead electrocardiogram (Figure 2, left).

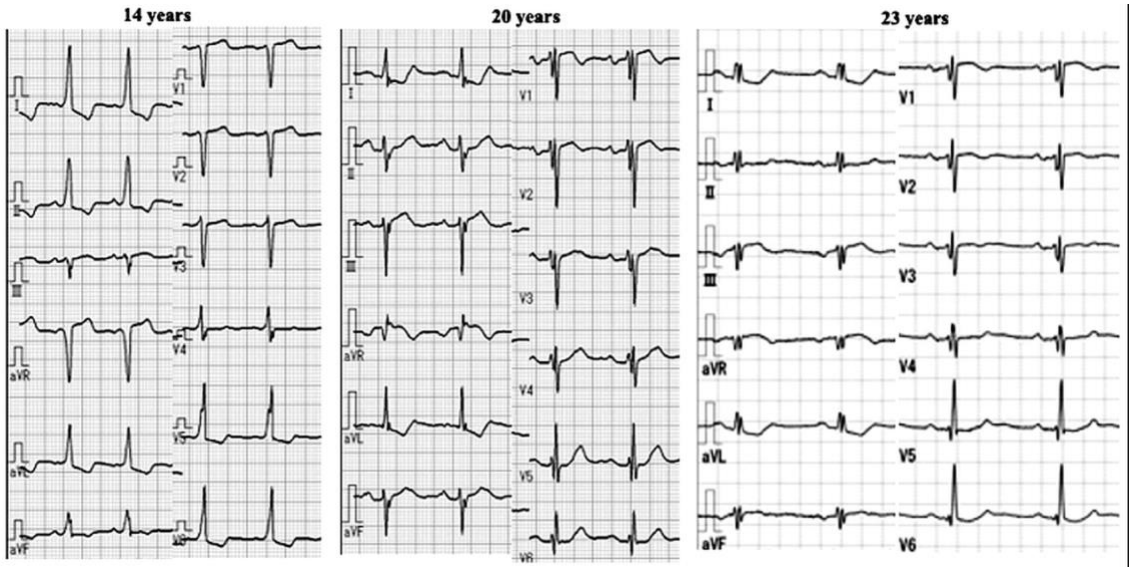

Figure 2: Changes in the 12-lead electrocardiogram. **Left**, 14 years, heart rate 89/ min, QRS 0.148 msec, QTc 0.440, R V_5 55.7 mV; **Middle**, 20 years, heart rate 63/ min, QRS 0.130 msec, QTc 0.445, R V_5 11.8 mV; **Right**, 23 years, heart rate 58/ min, QRS 0.120 msec, QTc 0.449, R V_5 12.0 mV. A decrease of in the heart rate, duration of QRS waves, and R waves in V_5 was found. The QTc was slightly elongated.

On two-dimensional echocardiography, the left ventricular end-diastolic diameter and end-systolic diameter were 46 mm and 27mm, respectively, and the thickness of the interventricular septum and posterior wall of the left ventricle were 23mm and 12mm, respectively (Figure 3, upper). The maximum mid-ventricular wall thickness of the left ventricle was 37mm on two-dimensional echocardiography. Systolic anterior movement of the mitral valve was detected, and the maximum peak flow of the left ventricular outflow tract was 5.1 m/s upon admission. Regurgitation of the mitral valve was moderate, and that of the aortic valve was slight. Cardiac catheterization was performed. The end-systolic and end-diastolic pressures in the left ventricle were 186 mmHg and 16 mmHg, respectively. The mean pulmonary artery pressure was 25 mmHg. The cardiac index by thermodilution was 2.4 l/min/m^2. After intravenous administration of cibenzoline 1.4mg/kg for 5 minutes, the pressure gradient between the left ventricle and ascending aorta decreased from 70 mmHg to 10 mmHg. The left ventricular ejection fraction was 35% as measured by ^{99m}Tc single-photon emission computed tomography (SPECT) myocardial perfusion imaging. A biopsy of the right ventricular myocardium revealed moderate hypertrophy, disarray, and moderate interstitial fibrosis. Although he had no history of familial cardiomyopathy, he had a MYH7 (β-Cardiac Myosin Heavy Chain) mutation [c.2609G>T (p.Arg 870Leu)].

Cibenzoline, 300 mg, was administered, and verapamil was stopped. The dose of cibenzoline was increased to 450 mg after 1 month (Fig. 1). The blood level of cibenzoline was within an effective range, and there were no side effects. Carteolol was stopped, and metoprolol, 60mg, was started. At the age of 17 years, his symptoms disappeared. His brain natriuretic peptide level decreased, and his left ventricular ejection fraction improved to 42% detected by 99mTc single-photon emission computed tomography myocardial perfusion imaging. However, the pressure gradient of the left ventricle on the two-dimensional echocardiography did not decrease. In the magnetic resonance findings, late gadolinium enhancement was found in the septal and inferior wall of the left ventricle.

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Because the maximum mid-ventricular wall thickness of the left ventricle was 37 mm, amiodarone was administered for the prevention of ventricular arrhythmias and sudden death. The level of amiodarone ranged from 1.0 to 1.4 μg/ml, which was within an effective range. Subsequently, the pressure gradient of the left ventricle gradually improved on two-dimensional echocardiography, and the cardiothoracic ratio also gradually decreased (Fig 1). The New York Heart Association class improved from the second to the first grade. His wide QRS and negative T waves in leads V_5 and V_6 improved at 20 years old (Fig 2, middle).

On the two-dimensional echocardiography at 23 years old, as compared with that at 14 years old, the maximum peak flow in the left ventricular outflow tract decreased from 5.1m/s to 1.5m/s. The ratio of the E/A in the inflow of the mitral valve decreased from 2.2 to 1.8, and the average E/e' also decreased from 21.0 to 7.6. The diameter of the left atrium decreased from 50 mm to 40 mm. Both regurgitation of the mitral valve and the aortic valve become trivial. The left ventricular end-diastolic diameter and end-systolic diameter and the thickness of the left ventricle did not remarkably change (Figure 3. Lower). Furthermore, his brain natriuretic peptide and cardiothoracic ratio decreased to 10 pg/ml and 53%, respectively. There were no side effects for each medication. Several premature ventricular contractions were detected on his Holter electrocardiogram.

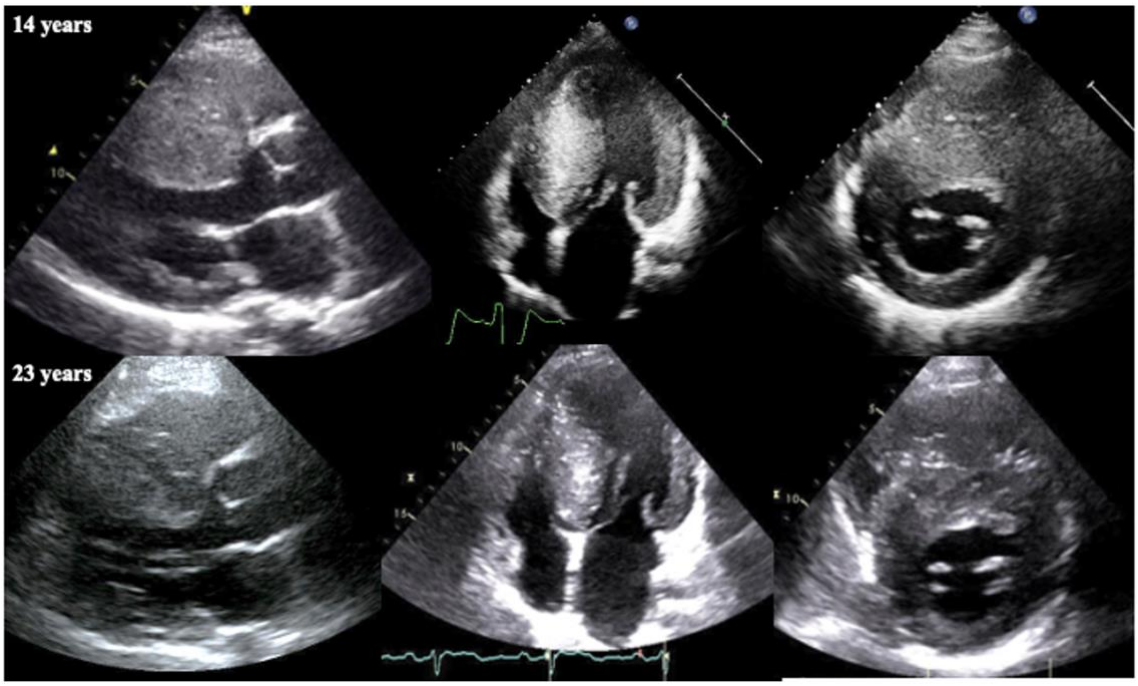

Figure 3: Cardiac echocardiogram by 2-dimensional echocardiography at 14 years and 23 years.

Upper, 14 years, Lower, 23 years.

Left, long-axis view, middle, 4-chamber view, right, short-axis view.

He had asymmetrical hypertrophy of the left ventricular septum. The mid-ventricular septum was severely thickened. He was obese, and it was challenging to obtain good echo images.

14 years, the left ventricular end-diastolic diameter and end-systolic diameter were 46 mm and 27mm, respectively, and the thickness of the interventricular septum and posterior wall of the left ventricle were 23mm and 12mm, respectively.

23 years, the left ventricular end-diastolic diameter and end-systolic diameter were 46 mm and 27 mm, respectively, and the thickness of the interventricular septum and posterior wall of the left ventricle were 21 mm and 12 mm, respectively.

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Discussion

Hypertrophic obstructive cardiomyopathy is usually genetic and inherited in an autosomal dominant pattern. However, sporadic cases can occur, especially in patients without a family history. He had a point mutation of β-Cardiac Myosin Heavy Chain 7 (c.2609G>T (p.Arg 870Leu). Usually, hypertrophic cardiomyopathy with a mutation of β-Cardiac Myosin Heavy Chain 7 can have ventricular hypertrophy and adverse events, and there is a high risk of an implantable Cardioverter Defibrillator and myectomy [11]. The median age at which patients suffer a first major cardiac event is 14.3 years [12]. The evidence that characterizes these genotypes and phenotypes in pediatric literature is limited. The biomolecular mechanisms leading to the hypertrophic cardiomyopathy phenotype are not well-known.

It is known that the pathophysiology of hypertrophic cardiomyopathy changes morphologically and functionally over the long-term period [13]. In this patient's 12-lead electrocardiogram, some remarkable changes with the improvement in the left ventricular outflow tract obstruction were detected over 10 years. The left axis deviation changed to an undetermined axis. The short PQ interval and wide QRS waves improved. The wide QRS waves changed to fragmented QRS waves, and the voltage of the QRS waves in the left precordial leads decreased remarkably. Further, the negative T waves in leads II, III, aV_F, V_5, and V_6 became positive T waves. Remarkable changes in the 12-lead electrocardiogram over 10 years were found, which revealed the functional changes in the left ventricle and the electrical remodeling. The relation between the findings of 12-lead electrocardiogram and late gadolinium enhancement in magnetic resonance imaging was reported [6,14]. The echo density in the thickened ventricular wall at the age of 23 years is mottled compared with that at 14 years. That might indicate the changes in the characteristics of the ventricular wall tissue.

Patients with hypertrophic cardiomyopathy often exhibit hypercontractions of the left ventricle, and this would deteriorate the left ventricular pressure gradient. The narrowing of the left ventricular outflow tract in hypertrophic obstructive cardiomyopathy can cause clinical symptoms and cardiac events. Hypertrophic obstructive cardiomyopathy is associated with ventricular arrhythmias, which can lead to sudden death, especially in individuals with high-risk features [15]. Therefore, medical treatment with beta-blockers and anti-arrhythmic agents is needed in patients after the diagnosis of hypertrophic obstructive cardiomyopathy [6,16,17]. The negative inotropic action of beta-blockers may result in a decrease in the pressure gradient of the left ventricular outflow tract and an improvement in heart failure [18,19]. Cibenzoline decreases the left ventricular pressure gradient in patients with hypertrophic obstructive cardiomyopathy by suppressing the myocardial contractility and improving the left ventricular diastolic function. Strong Na^+ channel-blocking agents provoke a decrease in the intracellular Ca^{2+} concentration [10-12]. His heart failure worsened at 14 years old. The induction of cibenzoline and metoprolol decreased his BNP levels. However, the pressure gradient of the left ventricular outflow tract did not resolve for 3 years after that. Seven years after the induction of amiodarone in addition to cibenzoline and metoprolol, the pressure gradient of the left ventricular outflow tract improved remarkably.

Amiodarone can prevent ventricular arrhythmia in adult patients with severe heart failure [10]. It is also helpful in children with severe heart failure, although it has some side effects, so it should be used with caution [20]. Although amiodarone was administered for the prevention of ventricular arrhythmias and sudden death, the pressure gradient of the left ventricle improved as a result in our case [21]. The effect of amiodarone on the pressure gradient of left ventricular outflow tract in patients with hypertrophic obstructive cardiomyopathy has been reported in previous reports [22]. Branzi A et al indicated the decrease in the pressure gradient of the left ventricular outflow tract with the short-term intravenous infusion of amiodarone in 2 patients with hypertrophic obstructive cardiomyopathy [23]. Imai T et al. reported a decrease in the left ventriclular pressure gradient in 5 of 6 patients with hypertrophic obstructive cardiomyopathy with amiodarone [24].

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Further, three case reports about the decrease in the pressure gradient of the left ventricular outflow tract were also found [25-27]. Although amiodarone was used as an antiarrhythmic agent in them, the pressure gradient of the left ventricle improved. However, the details about the cause of the improvement remain unknown. Because amiodarone has a negative inotropic effect and decreases the propagation velocity of a muscle fiber, it might affect the resolution of the left ventriclular pressure gradient. It exerts a sympatholytic effect and decreases heart rate in chronic amiodarone therapy [11,12]. Further, there were similarities among the previous successful cases, and they had a mid-ventricular obstruction of the left ventricle. A severe mid-ventricular hypertrophy was also found in our case. An improvement of left ventriclular pressure gradient due to amiodarone may be more effective in a mid-ventricular obstruction. The effect of amiodarone was more effective than that of cibenzoline in our case, in terms of his clinical course. The chronic effects of cibenzoline cannot be denied, and the synergy of these medicines with combined therapy can be considered [28]. The effectiveness of the combined therapy can exert a more beneficial effect than each alone. A careful, continuous follow-up is needed in the future. As the limitation, further studies are required to support the combination therapy and the findings of this clinical course.

Conclusions

The pressure gradient of the left ventricular outflow tract and heart failure with combination therapy, including amiodarone, improved in a young hypertrophic obstructive cardiomyopathy patient with a MYH7 (β-Cardiac Myosin Heavy Chain) mutation.

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Conflict of Interest: The authors declare no conflict of interests.

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