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Case Report

Safety Monitoring for Obstructive Hypertrophic Cardiomyopathy During Exercise

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ABSTRACT

Patients with hypertrophic cardiomyopathy (HCM) are prohibited from engaging in intensive exercise, to avoid sudden death. Given that patients with HCM, even those without left-ventricular outflow tract obstruction at rest, potentially have exercise-induced obstruction, reasonable monitoring methods during exercise are required. We present the case of a woman with HCM with exercise-induced obstruction whose hemodynamics during stress echocardiography were under observation using noninvasive cardiac output monitoring. Stroke volume declined during exercise before the manifest elevation of the left-ventricular outflow tract pressure gradient. As shown here, a noninvasive monitoring method can be useful in monitoring hemodynamics during exercise in HCM patients.

RÉSUMÉ

Les patients atteints d'une cardiomyopathie hypertrophique (CMH) se voient interdire la pratique d'activités physiques intenses, sous peine de mort subite. Étant donné le risque d'obstruction induite par l'effort auquel sont exposés les patients atteints de CMH, même ceux qui ne présentent pas d'obstruction de la voie d'éjection ventriculaire gauche au repos, des méthodes de surveillance raisonnables doivent être employées durant l'activité physique. Nous présentons le cas d'une femme atteinte de CMH ayant présenté une obstruction induite par l'effort et dont les paramètres hémodynamiques durant une échocardiographie à l'effort étaient surveillés au moyen d'un dispositif non invasif de mesure du débit cardiaque. Le volume d'éjection a diminué durant l'effort avant même que l'élévation du gradient de pression dans la voie d'éjection ventriculaire gauche puisse être observée. Comme nous l'expliquons ici, il peut être utile d'avoir recours à une méthode non invasive pour surveiller les paramètres hémodynamiques durant l'effort chez les patients atteints d'une CMH.

Hypertrophic cardiomyopathy (HCM) is one of the main causes of sudden death during and after exercise; therefore, guidelines prohibit competitive sports for patients with HCM.[1] However, some experts have recently raised the points that aerobic exercise is not contraindicated, and cardiac rehabilitation (CR), which is strongly recommended for patients with ischemic heart disease, might be similarly recommended for HCM patients. Some patients with HCM without left-ventricular outflow tract (LVOT) obstruction at rest have exercise-induced obstruction.[2] Establishing procedures for monitoring is an urgent need, to visualize the hemodynamic abnormalities during exercise, so that exercise can be safely undertaken by HCM patients.

Here, we propose a noninvasive cardiac output measurement method to monitor hemodynamics during exercise in HCM patients.

Case

A 60-year-old woman visited our hospital with a complaint of discomfort during exertion. She had no family history of cardiac diseases, no history of hypertension, and was asymptomatic at rest, with a resting blood pressure of 129/75 mm Hg and a pulse rate of 85 beats per minute. An electrocardiogram showed high voltage in the precordial leads (Fig. 1A), and a chest radiograph revealed cardiomegaly (Fig. 1B). The echocardiogram revealed normal systolic function, left-ventricular hypertrophy (Fig. 1C) with an LVOT pressure gradient (LVPG) of 15 mm Hg (Fig. 1D), and systolic anterior movement of the mitral valve. Neither sigmoid septum nor aortic stenosis was observed. B-type natriuretic peptide level at presentation was not elevated (21.6 pg/mL). There was no family history of Fabry disease, and no

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Ethics Statement: This study was approved by the Ethical Committee of the University of Tokyo (Approval No.2650-6) and conducted in accordance with the principles of the Declaration of Helsinki.

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Novel Teaching Points

- Exercise might induce sudden cardiac-related death for patients with HCM.
- Patients with HCM may have LVOT obstruction during exercise.
- In the present case of exercise-induced obstructive HCM, the decrease in SV during exercise was observed before the elevation of the LVPG.
- Monitoring the hemodynamics of patients with HCM during exercise is essential for obtaining the parameters of safe exercise.
- Noninvasive cardiac output measurement during exer cise can be useful for setting safe exercise intensity levels in CR for patients with HCM.

echocardiographic findings showing storage in the ventricular walls; given that the patient was a 60-year-old woman, it is unlikely that her cardiac hypertrophy is a manifestation of classic Fabry or cardiac Fabry diseases. Taking these findings together, the patient was diagnosed with HCM. At that time, no drug was administered to her.

In addition to monitoring blood pressure, pulse rate, and arrhythmias using a respiratory-cardiac monitoring device, a noninvasive measurement of cardiac output using an AESCULON mini (Osypka Medical, Berlin, Germany) was performed during ergometer exercise echocardiography. Cardiac output and stroke volume (SV) were continuously monitored during exercise. The LVPG was measured using the echocardiogram.

As a result, heart rate, cardiac index (CI), and SV increased rapidly after the onset of exercise (Fig. 2A). However, 3 minutes after the onset of exercise, SV began to decline in a stepwise fashion. Afterward, 7 minutes after the onset of exercise, the LVPG started to increase, reaching 45 mm Hg at the maximal exercise load, and mild chest symptoms appeared. Increased LVPG during exercise did not normalize but rather increased further, reaching 58 mm Hg during the recovery phase after exercise (Fig. 1E). The E/e' ratio (ratio between early mitral inflow velocity and mitral annular early diastolic velocity) of 19 at rest before exercise changed to 24 after exercise. She had no arrhythmias during or after exercise.

Discussion

In this patient suffering from HCM with exercise-induced LVOT obstruction, hemodynamics during exercise could be described successfully using a noninvasive monitoring system; CI and SV increased steeply after the onset of exercise, but SV started to decline during exercise, just before the manifest elevation of LVPG. Moreover, LVPG increased further during the recovery phase after exercise. Such findings were not observed in a patient with nonobstructive HCM (Fig. 2B).

In a previous report on the exercise hemodynamics of obstructive HCM, cardiac output response was impaired due to failure of SV augmentation. [3] In our case, SV decreased just before the manifest elevation of LVPG, providing a snapshot

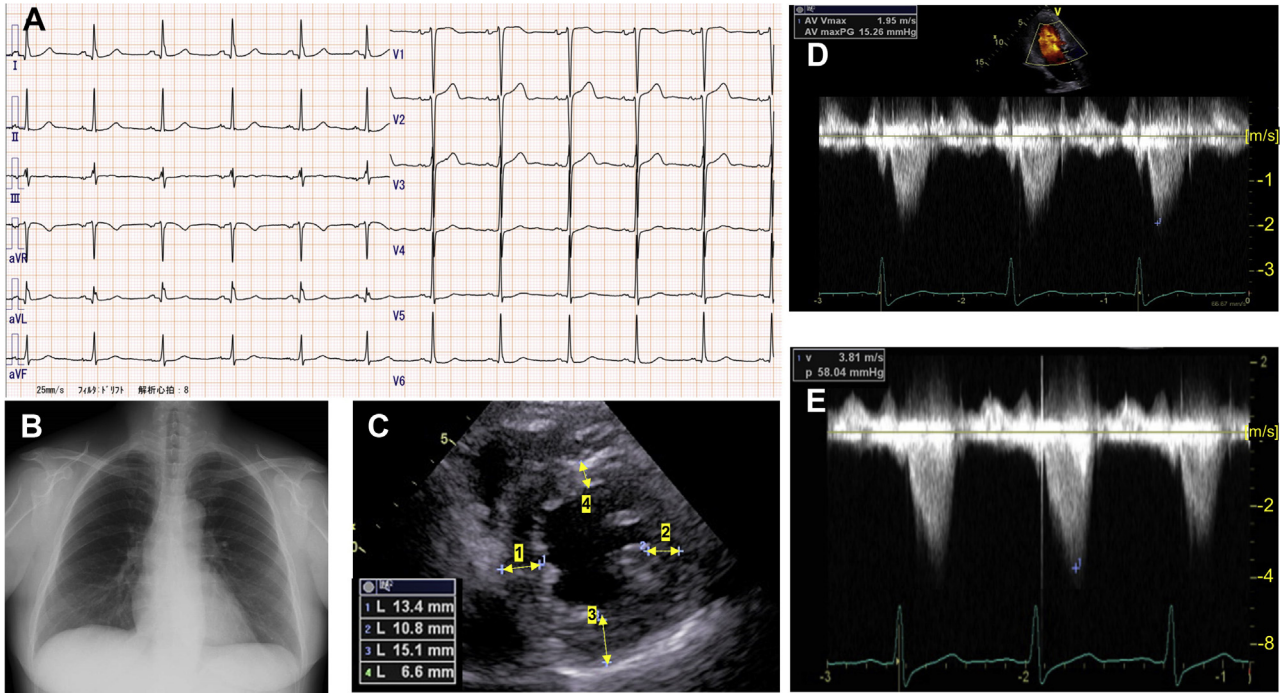

Figure 1. (**A**) Electrocardiogram showed high voltage in the precordial leads; (**B**) chest radiograph revealed cardiomegaly; (**C**) echocardiogram revealed left ventricular hypertrophy; (**D**) LVPG at rest was 15 mmHg; (**E**) maximum LVPG during the recovery phase after exercise was 58 mmHg. LVPG, left-ventricular outflow tract pressure gradient.

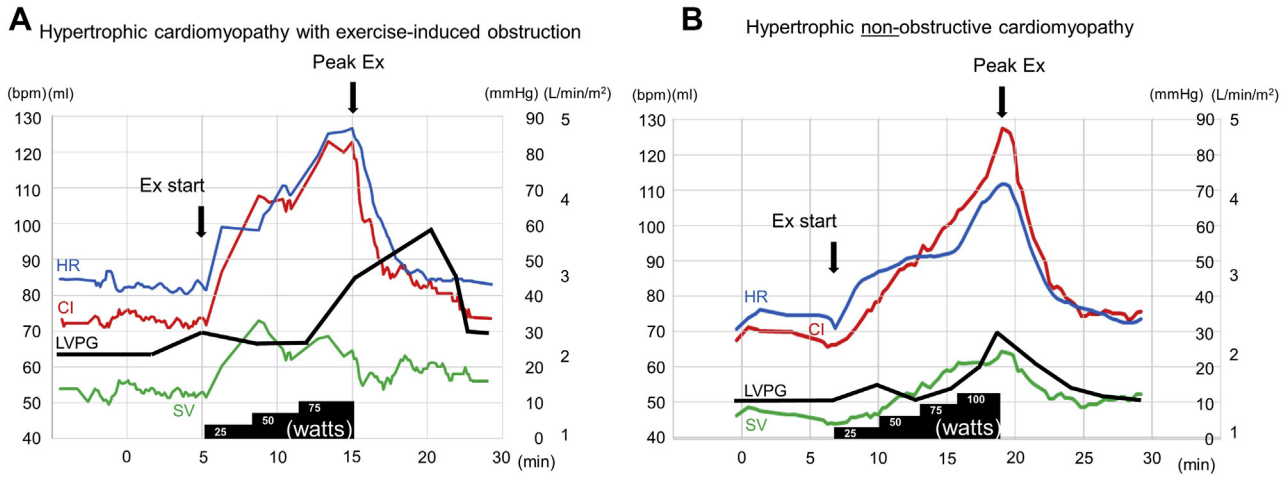

Figure 2. (**A**) Hemodynamics of the present case with hypertrophic cardiomyopathy with exercise-induced left-ventricular outflow tract obstruction. (**B**) Hemodynamics of a representative case with nonobstructive hypertrophic cardiomyopathy, a 49-year-old man with normal systolic function. bpm, beats per minute; CI, cardiac index; Ex, exercise; HR, heart rate; LVPG, left-ventricular outflow tract pressure gradient; SV, stroke volume.

of the exact timing of SV decrease during exercise in a patient with exercise-induced obstructive HCM. Some reports showed that diastolic dysfunction was induced soon after the start of exercise in the patients with heart failure with preserved ejection fraction and HCM. [4-6] In the presented case, the E/e' ratio was mildly elevated after exercise, although this index might not be accurately measured under tachycardia. Taken together, these findings indicate that left-ventricular diastolic dysfunction might be related to a decrease in SV during exercise. As the present case was not suffering from the typical HCM with asymmetric septal hypertrophy, further investigation is required to generalize our findings to the hemodynamic abnormalities that occur during exercise in HCM patients.

As shown here, we can easily observe CI and SV during exercise using this method of noninvasive monitoring, with which CI can be calculated by the electrical velocimetry method based on the change in the erythrocyte orientation monitored from the thoracic skin surface. [7] Measurement errors of the AESCULON mini may occur owing to displacement of the electrode patch by sweat, a low R-wave voltage, concurrent use of other electronic devices, and use of a deteriorated adhesive electrode patch. [8] Although the accuracy of the absolute value of the AESCULON mini measurements was inferior to that of measurements made with the Swan-Ganz catheter (Edwards Lifesciences, Irvine, CA), the device was shown previously to be very useful for monitoring trends in the CI. [7,8] Using this method to monitor hemodynamics during exercise, we can safely detect the patients who have exercise-induced LVOT obstruction, and thus obtain a safety margin for exercise. We can carefully set the intensity of exercise for CR, avoiding an SV decrease during exercise. Using this information to try to set safe exercise parameters in CR for HCM patients should be further investigated. This noninvasive monitoring method can be useful in monitoring hemodynamics during exercise in HCM patients, which will be helpful in ensuring the safety of exercise in CR for HCM patients.

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Disclosures

The authors have no conflicts of interest to disclose.

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