Obscured Meshwork Structure after Treatment for Heart Failure: A Case Study

Taku Rokutanda¹, Ikuo Misumi¹, Yousuke Hanaoka¹, Ryuichiro Akahoshi¹, Mitsuhiro Matsumoto¹, Nahoko Takeda¹, Hiroyuki Obayashi², Koichi Kaikita³, Megumi Yamamuro³, Seigo Sugiyama³ and Hisao Ogawa³

Abstract

An 87-year-old man with heart failure was admitted to our hospital. A transthoracic echocardiography showed diffuse mild left ventricular (LV) hypokinesis and LV noncompaction at the apex. A three-dimensional transthoracic echocardiography confirmed a trabecular meshwork. After treatment for heart failure, LV end-systolic dimension decreased and trabeculae seemed to converge and became obscure in end-systole. This is a rare case suggesting mechanism of obscured LV noncompaction after treatment for heart failure.

Key words: left ventricular noncompaction, three-dimensional echocardiography

(DOI: 10.2169/internalmedicine.51.7029)

Introduction

Left ventricular (LV) noncompaction is a rare congenital cardiomyopathy characterized by prominent trabeculations with interlaced recesses. We report a case of local noncompaction that disappeared after heart failure treatment.

Case Report

An 87-year-old man who experienced increasing exertional dyspnea was admitted to our hospital. He had a blood pressure of 186/101 mm Hg and a pulse rate of 81 beats/min. Auscultation of the heart provided normal results, but a fine crackle was heard in the bilateral lower lung fields. Blood sample analysis showed mild anemia, mild liver injury, and a high level of plasma brain natriuretic peptide (BNP) (1,587.5 pg/mL). The electrocardiogram showed sinus rhythm and a high R wave in lead V5. Chest radiography showed a cardiothoracic ratio of 58% with bilateral pleural effusion. A two-dimensional transthoracic echocardiography showed mild LV hypertrophy (interventricular septum thickness of 11.7 mm, left ventricular posterior wall thickness of 12.0 mm, and LV weight of 241 g) and diffuse mild LV hypokinesis (end-diastolic dimension of 50.2 mm, end-systolic dimension of 39.4 mm, and ejection fraction of 43.4%) (Table 1 and Fig. 1, upper panels). A local thin lesion with mildly developed trabeculae was located at the apex where the ratio of the distances from the epicardial surface to the recesses and to the peak of trabeculation was 0.27 (Fig. 2, upper, left panel). A three-dimensional transthoracic echocardiography revealed that the endocardial noncompacted layer consisted of a trabecular meshwork (Fig. 3, upper panels). After treatment with furosemide, spironolactone, valsartan, and azelnidipine, blood pressure decreased to 140/60 mmHg and the plasma BNP level decreased to 487.6 pg/mL. There was no change in electrocardiographic findings. A subsequent two-dimensional transthoracic echocardiography showed improved LV ejection fraction without significant change in LV end-diastolic dimension. (Table 1 and Fig. 1, lower panels) (LV end-diastolic dimension of 51.4 mm, end-systolic dimension of 32.1 mm, and ejection fraction of 67.3%). The features of noncompaction seemed to converge in systole and became obscure in

¹Internal Medicine, Kumamoto Saisyunsou Hospital, Japan, ²Cardiology, Obayashi Shin-chi Clinic, Japan and ³Department of Cardiovascular Medicine, Kumamoto University School of Medicine, Japan

Received for publication December 1, 2011; Accepted for publication January 18, 2012
Correspondence to Dr. Ikuo Misumi, misumi@saisyunsou1.hosp.go.jp
end-systole in both two-dimensional and three-dimensional echocardiography (Fig. 2, lower panels; and Fig. 3, lower panels). Color flow Doppler imaging during diastole showed the direct blood flow from the ventricular cavity into intertrabecular recesses that may support the diagnosis of non-compaction (Fig. 4). The patient then underwent cardiac catheterization. His coronary arteriogram and hemodynamic data were normal (pulmonary capillary wedge pressure, 10 mm Hg; cardiac output, 3.2 L/min; cardiac index, 2.3 L/min-m²), and he was discharged without symptoms.

**Discussion**

**Diagnosis of LV noncompaction**

LV noncompaction is a rare anomaly characterized by excessive prominent trabecular meshwork and deep intertrabecular recesses. The etiology of this disease is believed to be
an arrest of myocardial morphogenesis (1). The diagnostic criteria include (i) a ratio of the distances from the epicardial surface to the recesses and to the peak of trabeculation of ≤ 0.5 and (ii) the presence of numerous excessively prominent trabeculations and deep intertrabecular recesses (2). Because three-dimensional echocardiography can reveal the structure of the network of trabecular meshwork, it is useful in differentiating noncompaction from false tendon or other cardiac disorders (3). Song et al. reported that three-dimensional echocardiography might provide an accurate diagnosis of noncompaction (4). In our case, the ratio of the distances from the epicardial surface to the recesses and to the peak of trabeculation was 0.27, and three-dimensional echocardiography revealed that trabeculation formed a meshwork appearance that was diagnostic of noncompaction. Moreover, color flow Doppler imaging showed the direct blood flow from the ventricular cavity into intertrabecular recesses (Fig. 4) that is helpful for the diagnosis of noncompaction (4).

Afterload mismatch as a possible mechanism of heart failure

It is known that the area of LV noncompaction is usually hypokinetic and may become a cause heart failure. In the present case, this theory is unlikely, because LV wall motion was diffusely hypokinetic on admission and became almost normal after treatment. Leite-Moreira et al. (5) reported about afterload mismatch by narrowing the ascending aorta of open-chest rabbits. They found that acute rise in systolic blood pressure caused decrease in LV fractional shortening without significant change in LV end-diastolic dimension. In this case, as LV ejection fraction improved without significant change in LV end-diastolic dimension after decrease in blood pressure, high-blood pressure might have caused afterload mismatch and caused heart failure.

Obscured LV noncompaction in end-systole after treatment

Few reports exist about the resolution of noncompaction with concomitant improvement in LV wall motion. Stöllberger et al. (6) reported a case of dilated cardiomyopathy with hypertrabeculation that seemed to disappear after improved LV wall motion by biventricular pacing and hypothesized that the trabeculations might either disappear altogether or regress such that they are no longer visible. Luckie et al. (7) also reported a case of resolution of noncompaction after treatment for heart failure and speculated that noncompaction may occur as a consequence of increased intracavity pressure in predisposed individuals, or perhaps as an adaptive mechanism aimed at increasing endocardial surface area and optimizing stroke volume.

In the present case, noncompaction was observed throughout cardiac cycle on admission and obscured in end-systole after treatment. The trabeculae seemed to converge during
systole in both two-dimensional and three-dimensional echocardiography. As LV end-systolic dimension decreased after treatment, we hypothesized that trabeculation might have closed like bellows of accordion in end-systole. Under the condition of impaired cardiac function and a dilated left ventricle, the noncompacted tissue might have appeared like “opened bellows” throughout cardiac cycle.

Although the clinical implication of this reversible non-compaction is unknown, this condition may increase the susceptibility of the heart to pressure overload, which may lead to heart failure. On the other hand, recovery from heart failure may be rapid due to mild tissue remodeling.

The prevalence of LV noncompaction is rare and reported to be only 0.05% in the general population (8). This case suggests the significance of detection and clinical evaluation of localized and reversible LV noncompaction.

The authors state that they have no Conflict of Interest (COI).

References

6. Stöllberger C, Keller H, Finsterer J. Disappearance of left ven-