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Images in Cardiology

Regression of Electrocardiographic Left Ventricular Hypertrophy and Strain Pattern Using Pharmacotherapy in a Patient With Dilated Cardiomyopathy

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A 41-year-old man was admitted to our hospital for acute decompensated heart failure. His blood pressure was 102/70 mm Hg, and heart rate 120 beats per minute. He showed pulsus alternans and cold extremities without edema. Chest x-ray showed cardiomegaly and pulmonary congestion. Electrocardiogram showed high QRS voltage in precordial leads and strain pattern in leads I, aVL, V_5, and V_6, and also showed partial interatrial block in leads I and II, and P-wave terminal force in leads V_1 and V_2 (Fig. 1). Echocardiography showed severe global hypokinesis (left ventricular [LV] ejection fraction, 11%), dilatation of the left ventricle (LV internal dimension in diastole, 72 mm), and wall thinning (7 mm) with mild functional mitral regurgitation due to tethering of the posterior leaflet. His plasma B-type natriuretic peptide level was 2412.3 pg/mL. He did not have a history of longstanding uncontrolled hypertension or diabetes mellitus. Coronary angiography was normal. He had no atrial tachyarrhythmias, frequent premature ventricular contractions, nor ventricular tachyarrhythmias. Cardiac magnetic resonance imaging showed late gadolinium enhancement in the interventricular septum (Supplemental Fig. S1), but endomyocardial biopsy showed no specific findings suggestive of myocarditis or secondary cardiomyopathies. He was diagnosed with idiopathic dilated cardiomyopathy (DCM), and standard care with β-blocker (carvedilol), angiotensin-converting enzyme inhibitor (enalapril), mineralocorticoid receptor antagonist (spironolactone), and sodium-glucose cotransporter 2 inhibitor (empagliflozin) was initiated after acute care with dobutamine, carperitide, furosemide, and tolvaptan. He was discharged and continued regular visits to our outpatient clinic without hospitalization for heart failure. As drugs were up-titrated (carvedilol 40 mg, enalapril 10 mg, spironolactone 25 mg), electrocardiographic (ECG) LV hypertrophy (ECG-LVH) and strain pattern disappeared over a year (Fig. 2). Echocardiography showed reverse remodelling (LV internal dimension in diastole, 72-51 mm; LV ejection fraction, 11%-44%). ECG findings were normalized at 6 months, whereas echocardiographic reverse remodelling continued to occur (Supplemental Fig. S2 and Supplemental Table S1).

ECG-LVH is generally considered as a marker of pathological LVH caused by hypertension, aortic stenosis, hypertrophic cardiomyopathy, and secondary cardiomyopathies such as mitochondrial cardiomyopathy and Fabry disease. ECG-LVH and strain pattern have been reported as independent predictors of mortality, adverse cardiovascular events, and heart failure in a subclinical population free of cardiovascular diseases, hypertensive patients, and patients after aortic valve replacement.[1-3] ECG-LVH and strain pattern were also observed without pathological LVH among DCM patients (approximately 30%).[4] In contrast to the general heart failure population, ECG-LVH

Novel Teaching Points

- ECG-LVH and strain pattern might be a predictor of better prognosis in patients with dilated cardiomyopathy unlike other cardiovascular diseases.
- Regression of ECG-LVH might be a sensitive marker of reverse remodelling by pharmacotherapy in patients with dilated cardiomyopathy.

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Ethics Statement: The case we reported here has adhered to the relevant ethical guidelines.

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See page 389 for disclosure information.

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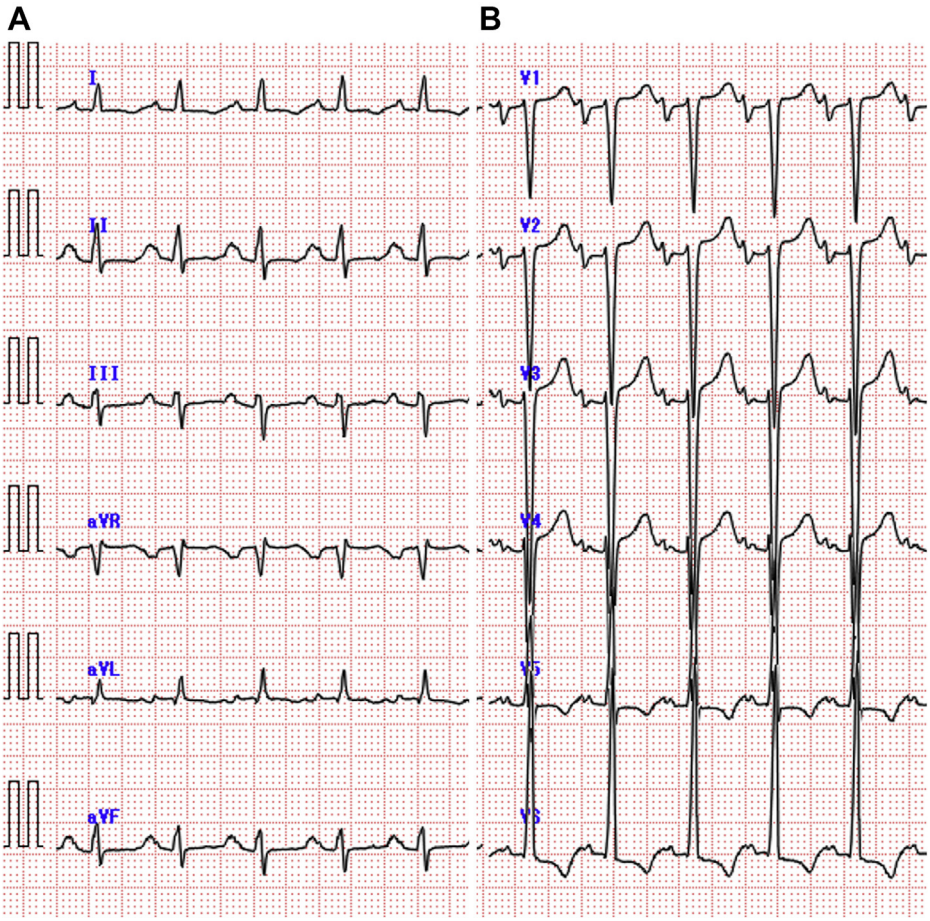

Figure 1. Electrocardiogram at the first presentation.

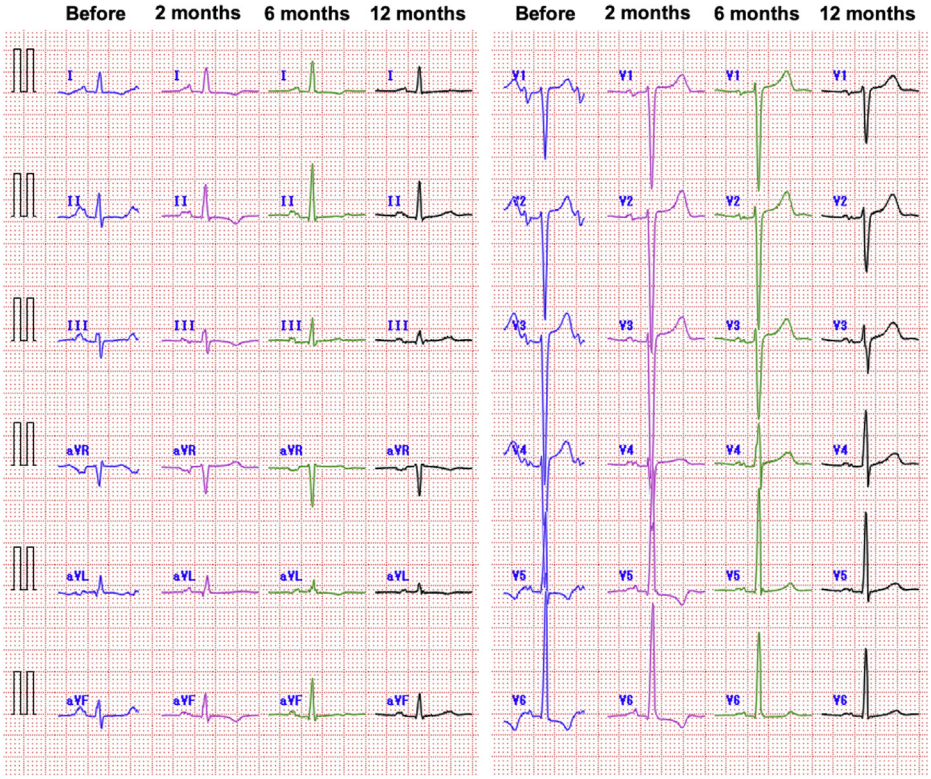

Figure 2. Serial electrocardiograms after initiation of pharmacotherapy.

in DCM might be a marker of better prognosis.[4,5] ECG-LVH likely indicates relative preservation of viable myocytes as well as LV volume overload in DCM. Regression of ECG-LVH by antihypertensive treatment or aortic valve replacement, and electrophysiological reverse remodelling with cardiac resynchronization therapy or LV assist device have been studied, however, ECG reverse remodelling by pharmacotherapy in DCM has not been well characterized. ECG-LVH and strain pattern clearly regressed by pharmacotherapy in the present case. In addition, the finding of ECG-LVH regression preceding echo-based reverse remodelling warrants further study in the prognostic significance of electrocardiography during follow-up. In conclusion, ECG-LVH and its regression can predict and monitor reverse remodelling in DCM.

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Disclosures

The authors have no conflicts of interest to disclose.

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Supplementary Material

To access the supplementary material accompanying this article, visit *CJC Open* at https://www.cjcopen.ca/ and at https://doi.org/10.1016/j.cjco.2020.11.009.