Journal of Rural Medicine

Case report

A case of Takotsubo cardiomyopathy with apical hypertrophic cardiomyopathy-like morphological changes during recovery

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Abstract

Objective: Few cases of Takotsubo cardiomyopathy with apical hypertrophic cardiomyopathy (APH)-like morphological changes during the recovery process have been reported.

Patient: A 56-year-old woman diagnosed with Takotsubo cardiomyopathy showed a morphology similar to that of APH during recovery. We examined this patient using 2D speckle-tracking echocardiography based on the method used for hypertrophic cardiomyopathy, which suggested that the circumferential strain (CS) of the middle wall indicated myocardial function of the left ventricle, and the CS of the inner wall was associated with left ventricular chamber function.

Results: We measured the CS of the endocardial, middle, and epicardial layers and found that the apical inner layer CS (CSinner), middle layer CS, and outer layer CS were all decreased at the onset. CSinner showed a strong tendency to recover on echocardiography performed when APH-like morphology was observed.

Conclusion: The morphology of the apex in our case likely contributed to the maintenance of chamber function.

Key words: Takotsubo cardiomyopathy, apical hypertrophic cardiomyopathy, circumferential strain, echocardiography

(J Rural Med 2022; 17(3): 171–175)

Introduction

A few cases of Takotsubo cardiomyopathy with apical hypertrophic cardiomyopathy (APH)-like morphological changes during the recovery process have been reported[1–3]. Furthermore, a single-center prospective study that included patients with Takotsubo cardiomyopathy who showed APH-like morphological changes during the recovery process reported the incidence, characteristics, and clinical outcomes[4].

Received: October 11, 2021
Accepted: February 8, 2022
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We report a case in which the apical left ventricular thrombus was complicated in the acute phase of Takotsubo cardiomyopathy and showed an APH-like morphology during recovery. We examined this patient using 2D speckle-tracking echocardiography, based on the method used for hypertrophic cardiomyopathy[5,6]. We have obtained informed consent for this manuscript.

Case report

A 56-year-old woman, admitted to the psychiatric department for treatment of schizophrenia, had unstable dietary intake, received an intravenous drip from the peripheral vein, and was monitored via electrocardiography. On the 24th day after admission, despite the absence of any subjective symptoms, the nurse noticed an abnormal electrocardiogram and consulted with our cardiology department. The patient's vital signs were as follows: body temperature, 37.4°C; blood pressure, 98/70 mmHg; heart rate 69/min, and SPO_2, 95% in room air. The electrocardiogram showed gi-

ant negative T waves in the I, II, III, aVF, and V2-6 leads (Figure 1A), and the QT interval was extended to 490 ms. Furthermore, a positive T wave was seen in the aVR and V1 leads, which has been previously reported to strongly suggest Takotsubo cardiomyopathy rather than myocardial infarction[7]. Echocardiography showed extensive left ventricular asynergy, except in the basal part of the heart. The apex of the heart presented with ballooning, which showed a morphology consistent with that of Takotsubo cardiomyopathy (Figure 1A). The left ventricular ejection fraction (LVEF) was 48.9%, left ventricular end-diastolic diameter (LVDd) was 45 mm, left ventricular end-diastolic volume (LVEDV) was 78 mL, left ventricular end-systolic diameter (LVDs) was 27 mm, and left ventricular end-systolic volume (LVESV) was 37 mL. There was no congestion on a chest radiograph, with a cardiothoracic ratio of 46%. Blood test results showed a slight increase in creatine phosphokinase (585 U/L), creatine phosphokinase-MB (10 U/L), and troponin I (312 ng/mL). However, because the degree of elevation of these myocardial deviation enzymes and that of myocardial damage was inconsistent, Takotsubo cardiomyopathy was strongly suspected rather than myocardial infarction. No electrolyte abnormalities or antipsychotics causing myocardial damage were observed. Coronary computed tomography angiography revealed no significant stenosis in the coronary arteries. Unfortunately, because coronary angiography was infeasible in our case owing to the patient's psychiatric condition, it was difficult to determine if vasospastic angina was present. By comprehensively evaluating these test results, we diagnosed the patient with takotsubo cardiomyopathy. Apical thrombosis was documented, and anticoagulant therapy with heparin and warfarin was initiated. We did not administer any other drugs, and only followed the patient at rest. Echocardiography on the 18th day of hospitalization showed a morphology similar to that of APH (Figure 1B; LVEF, 73%; LVDd, 50 mm; LVDs, 26 mm; LVEDV, 60 mm; LVESV, 16 mm), and a giant negative T wave in the V3-4 lead was found on electrocardio-

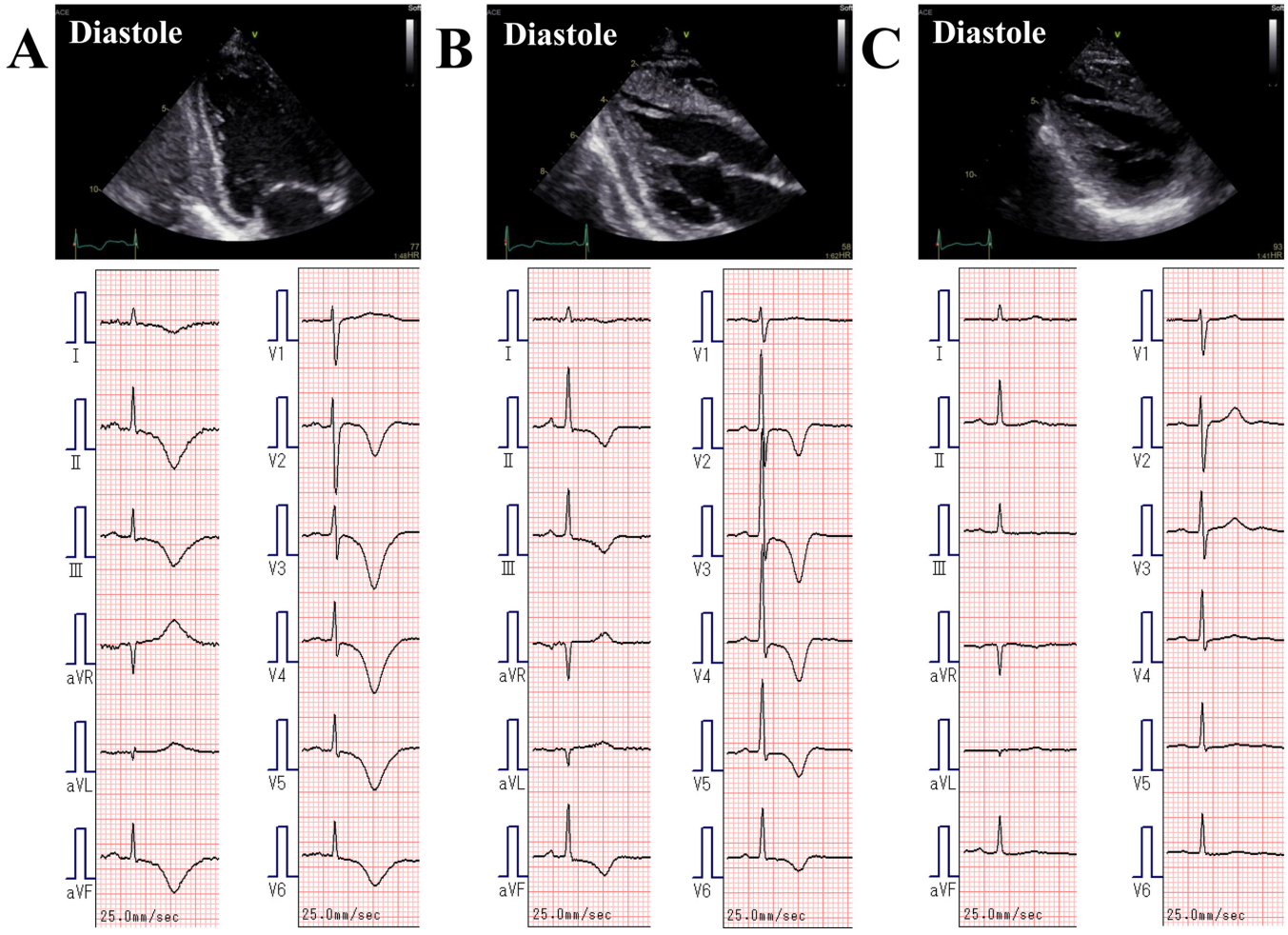


Figure 1 Electrocardiogram and echocardiographic images.
The electrocardiogram and echocardiographic images at the time of onset (A), at the time of APH-like morphological changes (B), and at the time when the morphology and wall motion were normalized (C) are shown.

gram, showing changes characteristic of APH (Figure 1B). A pressure gradient was not observed in the left ventricle. The patient had no subjective symptoms, vital signs were stable, and there were no signs of heart failure. Despite similar case reports in the literature[1–3] no specific treatment has been reported; thus, we continued with the follow-up. The APH-like findings disappeared after approximately 5 months (Figure 1C; LVEF: 74%; LVDd: 47 mm; LVDs: 27 mm; LVEDV, 63.2 mL; LVESV: 20.1 mL). We examined the patient using 2D speckle-tracking echocardiography, based on the method used for hypertrophic cardiomyopathy[6]. We measured the circumferential strain (CS) and longitudinal strain (LS) of endocardial, middle, and epicardial layers. The images of the CS in each layer at the apex are shown in Figure 2, and the CS and LS values in each layer are shown in Figure 3. Previously reported normal values for each layer of the CS and LS were used as a reference[8]. At the early stage of onset, the inner layer CS (CSinner), middle layer CS (CSmid), and outer layer CS (CSouter) at the apex showed a marked decrease. Echocardiography performed when an APH-like morphology was observed during the recovery process of Takotsubo cardiomyopathy showed a sharp improvement in the CSinner. Echocardiography performed at the time of normalization of myocardial morphology and wall motion showed a normalized CSinner. LSinner, LSmid, and LSouter at the apex were not normalized despite the disappearance of the APH-like morphology and normalization of wall motion.

Discussion

Aurigemma *et al.*[5] showed that in patients with hypertensive left ventricular hypertrophy with preserved left ventricular ejection fraction, midwall fractional shortening (FS) decreased, and endocardial FS increased compared with those in the controls. Moreover, the difference between increased endocardial FS and decreased midwall FS in individual cases correlated with wall thickness, suggesting that

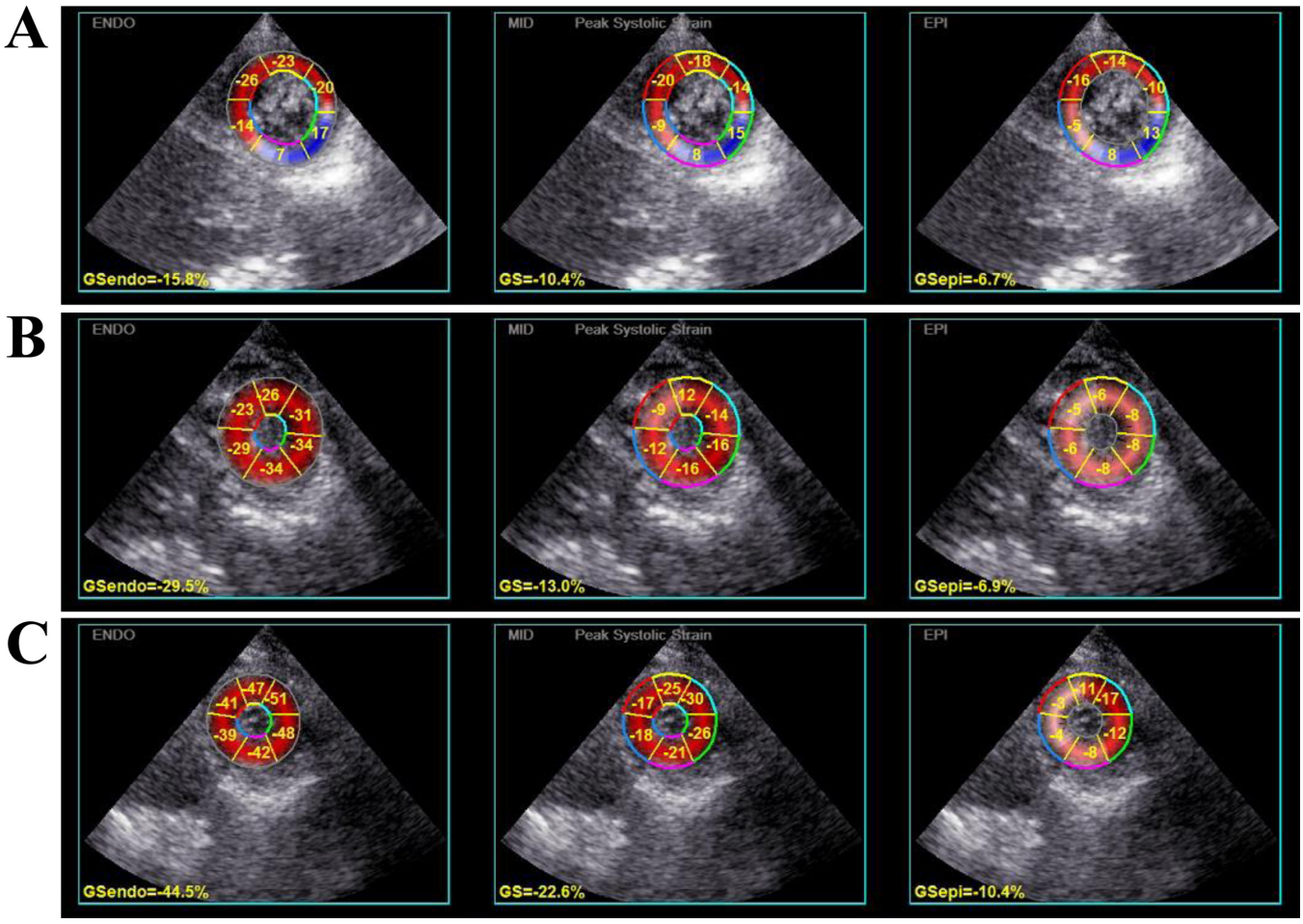
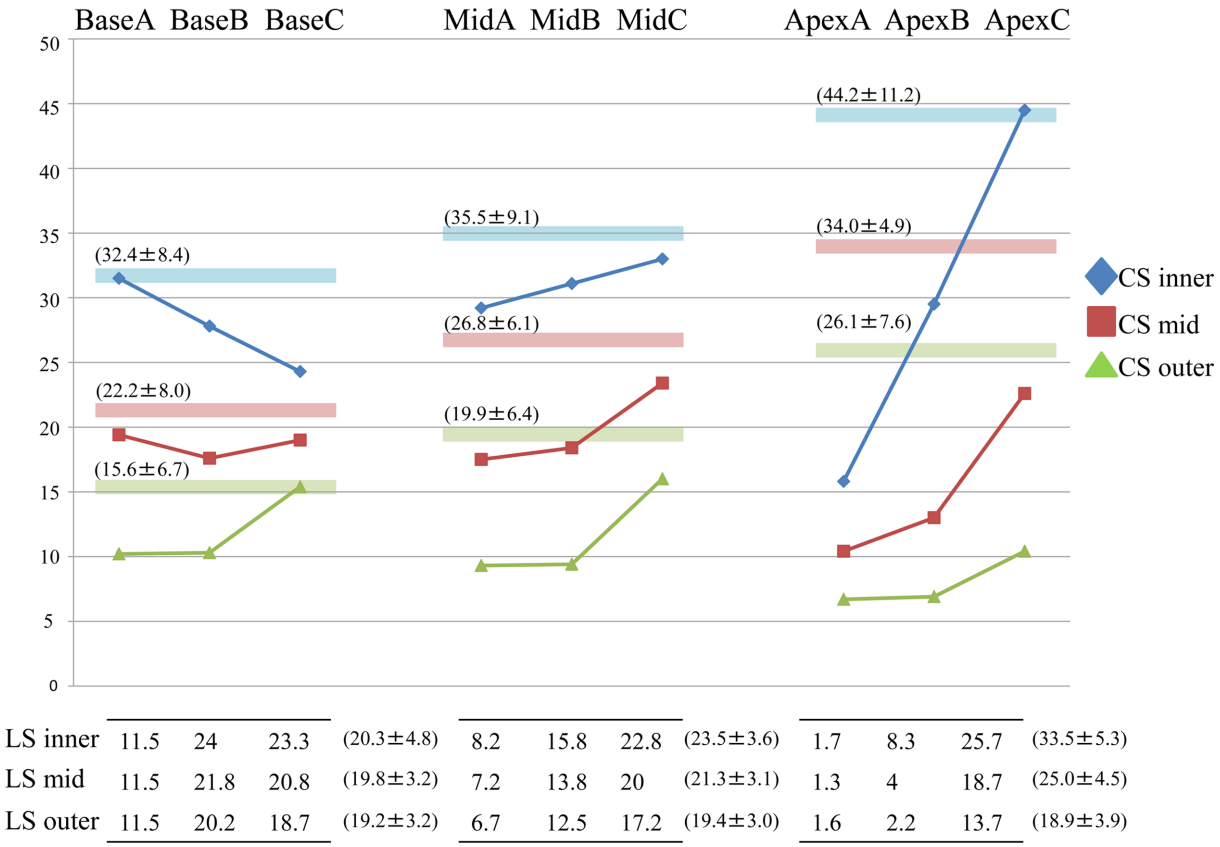


Figure 2 Representative images of the circumferential strain.
Representative images of the circumferential strain for each layer in the apex at the time of onset (A), APH-like morphological changes (B), and when the morphology and wall motion were normalized (C) are shown. GS stands for global circumferential strain.



LS inner	11.5	24	23.3	(20.3±4.8)	8.2	15.8	22.8	(23.5±3.6)	1.7	8.3	25.7	(33.5±5.3)
LS mid	11.5	21.8	20.8	(19.8±3.2)	7.2	13.8	20	(21.3±3.1)	1.3	4	18.7	(25.0±4.5)
LS outer	11.5	20.2	18.7	(19.2±3.2)	6.7	12.5	17.2	(19.4±3.0)	1.6	2.2	13.7	(18.9±3.9)

Figure 3 The values of the circumferential strain and the longitudinal strain.
The values of the circumferential strain (CS) and longitudinal strain (LS) for the endocardial, middle, and epicardial layers at the time of onset (A), at the time of APH-like morphological changes (B), and at the time when the morphology and wall motion were normalized (C) are shown. The colored bars in the graph show the normal values of CS in each region. The normal values of LS in each region are shown in parentheses. In the apex, CS at the onset was severely reduced in all layers, but CSinner showed rapid improvement thereafter. CSouter: outer layer CS; CSmid: middle layer CS; CSinner: inner layer CS; LSouter: outer layer LS; LSmid: middle layer LS; LSinner: inner layer LS.

midwall FS indicates "myocardial function, endocardial FS indicates "chamber function, and left ventricular hypertrophy compensated for reduced "myocardial function" and maintained heart "chamber function". Okada *et al.*[6], who studied CS and LS for each layer in patients with hypertrophic cardiomyopathy, demonstrated reduced CSmid and maintained CSinner in those patients, indicating that lower CSmid reflected impaired myocardial function and maintained CSinner associated with the maintenance of chamber function. We were interested in these reports and performed layer-specific CS and LS in this case. As a result, CSouter, CSmid, and CSinner severely decreased in the apex at early onset. However, subsequently, the apex showed a sharp tendency of improvement in CSinner when an APH-like morphology was exhibited. The lack of improvement in CSouter and CSmid might indicate that the myocardial function had not recovered. In the apex, even when the APH-like morphology disappeared, LS was not normalized in all layers, indicating that myocardial function was not restored, although heart chamber function was maintained. We believe that the apex morphology of our patient contributed to the maintenance of chamber function and cardiac output.

Conclusion

We believe that LS and CS mid, which indicate myocardial function, contribute to the evaluation of myocardial function in Takotsubo cardiomyopathy patients with APH-like morphological changes during the recovery process. Moreover, the CSinner, which indicates the chamber function, shows an improvement in apparent wall motion.

Acknowledgment

We thank the staff of the Department of Cardiology at Imamura General Hospital.

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