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Transient increase in wall thickness of the left ventricular apex during recovery from "ampulla" cardiomyopathy

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"Ampulla" cardiomyopathy is a syndrome characterized by transient abnormal left ventricular wall motion with hypokinesia around the apical area and hyperkinesia at the basal area, without any detectable coronary lesion. Two cases of transient wall thickening of the left ventricular apex during recovery from "ampulla" cardiomyopathy are described. Apical wall thickening was documented by left ventriculography, echocardiography, and thallium (^{201}Tl) single-photon emission computed tomography (SPECT) during the recovery phase. The thickness of the apical wall subsequently returned to normal. Both patients underwent provocation tests. Coronary spasms were positive. This transient increase in left ventricular apical volume may have been caused by myocardial inflammation secondary to "ampulla" cardiomyopathy.

Key words: "ampulla" cardiomyopathy, transient apical wall thickening, thallium single-photon emission computed tomography

INTRODUCTION

"AMPULLA" cardiomyopathy is a syndrome characterized by transient abnormal left ventricular wall motion with hypokinesia around the apical area and hyperkinesia at the basal area, without any detectable coronary lesion. Coronary spasm is sometimes positive on a provocation test. Abnormal wall motion usually resolves within 2 weeks.[1] We report two cases of transiently increased apical wall thickness of the left ventricle that developed during recovery from "ampulla" cardiomyopathy.

CASE REPORT

Case 1

An 83-year-old woman who was hospitalized for rehabilitation after cholecystectomy complained of chest discomfort at night. Serial electrocardiograms (ECGs) obtained 15 minutes after onset are shown in Figure 1; ST-segment elevation was seen in leads V_{2-6}. Two-dimensional echocardiography revealed hypokinesia of the entire left ventricle, except the basal area. Isosorbite dinitrate was administered, and the chest discomfort resolved 30 minutes later. There was no elevation of creatine-phosphokinase. Cardiac catheterization was performed 3 days later. Left ventriculography showed hypokinesia around the apical area and hyperkinesia at the basal area (Fig. 2). Coronary angiography revealed no abnormalities (Fig. 3). ^{201}Tl scintigraphy showed moderate to low perfusion at the apex 10 days after the attack. ^{201}Tl SPECT showed high accumulation at the apex 2 months after the attack. Six months later, ^{201}Tl SPECT showed normal accumulation (Fig. 4). Consistent findings were obtained by left ventriculography. One month after the attack, left ventriculography showed an "ace of spades" configuration, indicating thickening of the apex. Echocardiography also showed apical wall thickening of the left ventricle (Fig. 5). Thirty months later, the "ace of spades" configuration had completely disappeared (Fig. 6). She underwent provocation tests with acetylcholine. Coronary spasm was positive (Fig. 7).

Case 2

A 60-year-old woman was hospitalized for a fracture of

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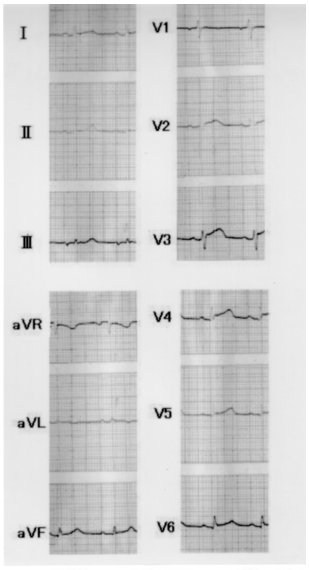

Fig. 1 Serial electrocardiograms of case 1. ST-segment elevation was seen in lead V_{2-6} on the day of onset.

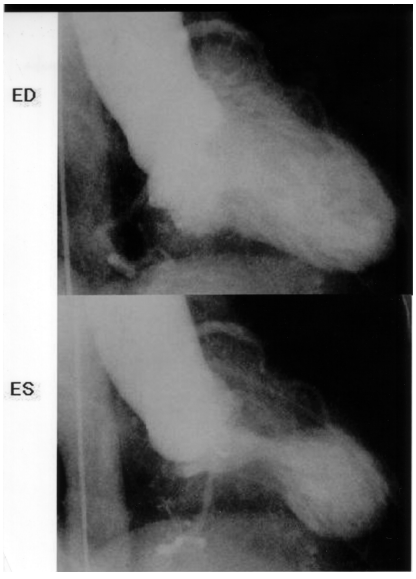

Fig. 2 Serial left ventriculograms of case 1. Upper panel is at end-diastole, and lower one is at end-systole. Left ventriculography on the 3rd day after onset showed hypokinesia around the apical area and hyperkinesia at the basal area.

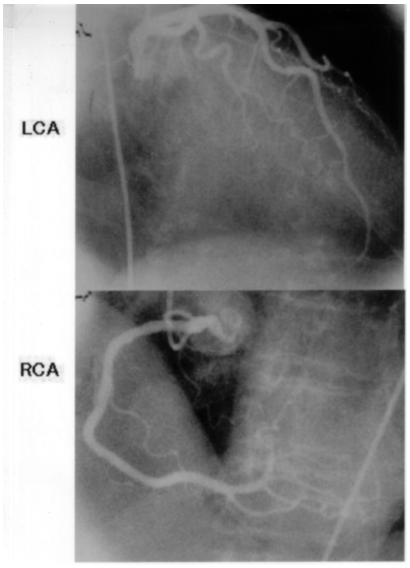

Fig. 3 Serial coronary angiograms of case 1. Upper panel is of the left coronary artery, and lower one is of the right coronary artery. No abnormalities were found on the 3rd day after onset. RCA, right coronary artery; LCA, left coronary artery.

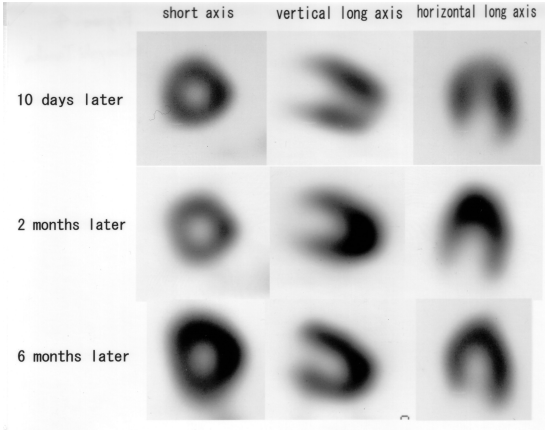

Fig. 4 Serial cardiac ^{201}Tl SPECT image of case 1. ^{201}Tl scintigraphy showed moderate to low perfusion at the apex on the 10th day after onset and high accumulation at the apex 2 months after onset. Six months later, normal accumulation was found.

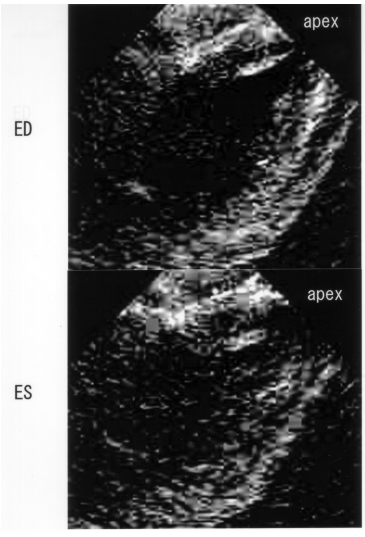

Fig. 5 Two-dimensional echocardiogram showing apical wall thickening of the left ventricle.

the right femur. She had chest pain on the day after the operation. The ECGs are shown in Figure 8. T-wave inversion was seen in leads I, aV_L, and V_{2-6}. Two-dimensional echocardiography revealed hypokinesia of the entire left ventricle, excluding the basal area. Cardiac catheterization was performed immediately. Left ventriculography showed hypokinesia around the apical area and hyperkinesia at the basal area (Fig. 9). Coronary angiography revealed no abnormalities (Fig. 10). Symptoms resolved during the examination. The maximum creatine-phosphokinase level was 580 IU/*l*. ^{201}Tl SPECT showed no abnormalities 3 days after the attack. ^{201}Tl SPECT on the 17th day showed increased Tl uptake at the apex. Nine months later, ^{201}Tl SPECT showed normal accumulation (Fig. 11). The left ventriculographic findings were similar to those of case 1. One month after the attack, left ventriculography showed an "ace of spades" configuration. Nine months later, the "ace of spades"

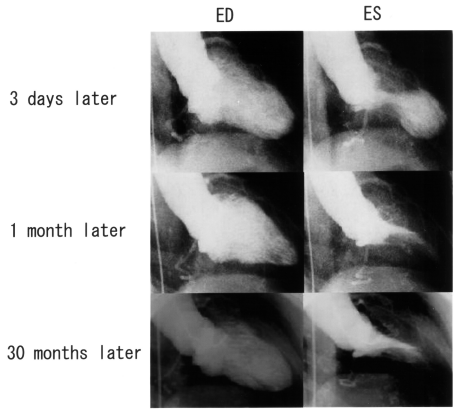

Fig. 6 Time course of the left ventriculograms of case 1. Left panels are at end-diastole (ED), and right ones are at end-systole (ES). One month after the onset, left ventriculography showed an "ace of spades" configuration, which means thickening of the apex. Thirty months later, the "ace of spades" configuration had completely resolved.

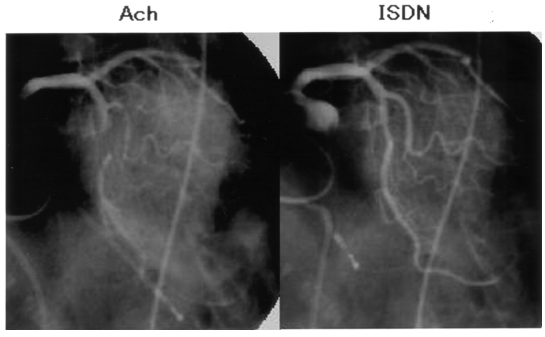

Fig. 7 Serial coronary angiograms of case 1. Injection of acetylcholine (Ach) into the left coronary artery induced spasm with 99% narrowing. After intracoronary administration of isosorbide dinitrate (ISDN), coronary angiograms returned to normal.

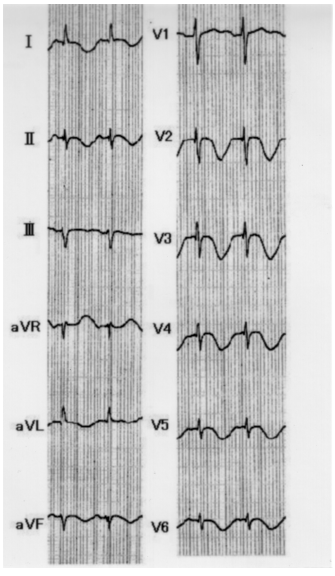

Fig. 8 Serial electrocardiograms of case 2. T-wave inversion was seen in leads I, aV_L, and V_{2-6} on the day of onset.

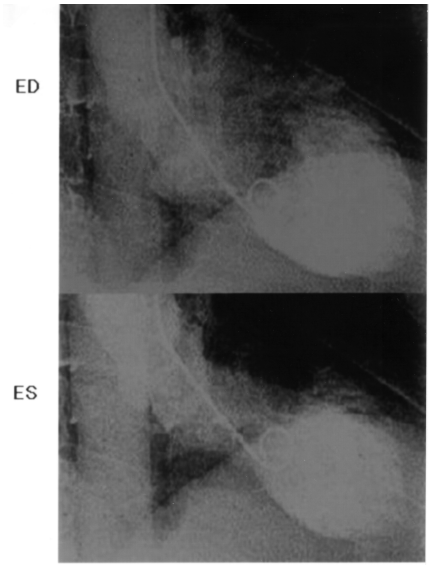

Fig. 9 Serial left ventriculograms of case 2. Upper panel is at end-diastole (ED), and lower one is at end-systole (ES). Left ventriculography on the day of onset showed hypokinesia around the apical area and hyperkinesia at the basal area.

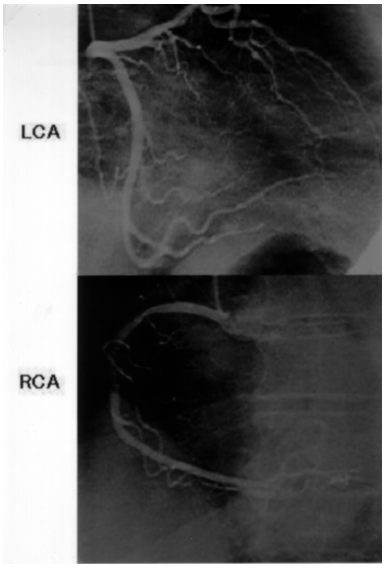

Fig. 10 Serial coronary angiograms of case 2. The upper panel is of the left coronary artery, and lower one is the right coronary artery. No abnormalities were found on the day of onset.

configuration had completely resolved (Fig. 12). Coronary spasm was positive on a provocation test (Fig. 13).

DISCUSSION

Because of the typical clinical course and examination results, both cases were diagnosed as "ampulla" cardiomyopathy. To our knowledge, a transient increase in left ventricular mass at the apex during recovery from "ampulla" cardiomyopathy has not been reported previously. Left ventriculography, two-dimensional echocardiography, and ^{201}Tl SPECT showed findings characteristic of apical hypertrophic cardiomyopathy only for a brief period during recovery. Subsequently, these findings resolved.

A transient increase in left ventricular mass has been observed during recovery from active myocarditis and stunned myocardium.[2–4] This phenomenon is attributed

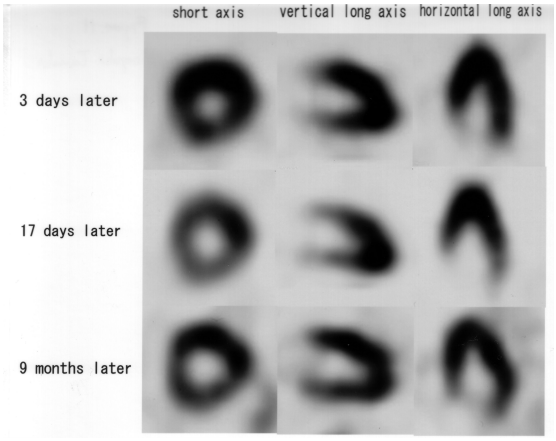

Fig. 11 Serial cardiac ^{201}Tl SPECT image of case 2. ^{201}Tl scintigraphy showed no abnormalities on the 3rd day after onset and high accumulation on the 17th day after onset. Nine months later, normal accumulation was found.

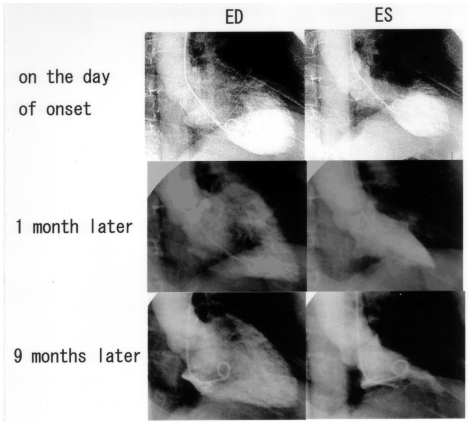

Fig. 12 Time course of the left ventriculograms of case 2. Left panels are at end-diastole (ED), and right ones are at end-systole (ES). One month after onset, left ventriculography showed an "ace of spades" configuration, indicating thickening of the apex. Nine months later, the "ace of spades" configuration had completely disappeared.

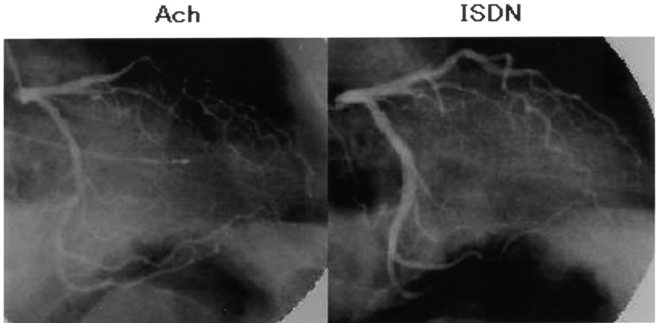

Fig. 13 Serial coronary angiograms of case 2. Injection of acetylcholine (Ach) into the left coronary artery induced diffuse spasm with 90% narrowing. After intracoronary administration of isosorbide dinitrate (ISDN), coronary angiograms returned to normal.

to the development of edema after the severe myocardial damage. Histopathologically, "ampulla" cardiomyopathy is sometimes associated with focal myocyte injury, myocardial depletion with cell infiltration (including polymorphonuclear leukocytes), or focal fibrosis. A transient increase in wall thickness of the left ventricular apex after "ampulla" cardiomyopathy is most likely caused by myocardial edema after the severe myocardial damage.

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