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Case report

Takotsubo cardiomyopathy in a bedridden patient with dementia and communication difficulties due to Alzheimer's disease

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Abstract

Takotsubo cardiomyopathy is a transient wall motion abnormality of the left ventricular apex, accompanied by emotional or physical stress. Although Takotsubo cardiomyopathy is generally considered a benign disease, severe clinical complications may occur, and early detection of the disease is important. In this report, we present the case of an 86-year-old bedridden woman with a history of bronchial asthma who was transferred to our hospital because of wheezing. She was diagnosed with Alzheimer's disease and had communication difficulties. After an asthma attack and improvement, Takotsubo cardiomyopathy was identified via electrocardiography. She was unable to complain of any symptoms but showed serial electrocardiographic changes, elevated myocardial markers, and transient left ventricular apical ballooning. The prevalence of dementia increases dramatically with age. This case indicates that Takotsubo cardiomyopathy may occur even in patients with severe dementia, who are bedridden and show communication difficulties in a clinical setting.

Key words: Takotsubo cardiomyopathy, dementia, bedridden, communication difficulties, Alzheimer's disease

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Introduction

Takotsubo cardiomyopathy (TTC) is characterized by a reversible left ventricular wall motion abnormality that is observed as apical ballooning without significant coronary arterial stenosis[1]. It commonly occurs in postmenopausal women and is often precipitated by intense physical or emotional stress. Prognosis of TTC is generally favorable; however, it has been shown that severe clinical complications can occur, especially in the early stage of disease[2]. Recent studies have also shown that TTC and acute coronary syndrome show comparable morbidity and mortality rates[3, 4]. Therefore, early detection and diagnosis of the disease are important.

Due to the unprecedented increase in the proportion of elderly individuals, the occurrence of patients with cognitive impairment and dementia is rapidly becoming a significant burden for medical care[5]. Alzheimer's disease is the most common cause of dementia, accounting for approximately two-thirds of cases in the elderly population[6]. Dementia due to Alzheimer's disease is characterized by a continuous decline in thinking and behavioral and social skills that disrupt a person's ability to function independently. Difficulty or ineffectual communication with a patient suffering from Alzheimer's disease also complicates recording of clinical symptoms.

Here, we present a case in which TTC was discovered after an acute episode of an asthma attack in a bedridden patient with dementia and communication difficulties due to the progression of Alzheimer's disease.

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Case Report

An 86-year-old woman with a history of bronchial asthma was transferred to our hospital because of wheezing. She was diagnosed with Alzheimer's disease at the age of 76 years and had been hospitalized for cognitive deterioration since she was 79 years old. She was bedridden and had communication difficulties because of the advanced stage of Alzheimer's disease.

On admission, her body temperature was 36.8°C, blood pressure was 140/96 mmHg, heart rate was 102 beats/min, and oxygen saturation was 85% with room air. Physical examination revealed stridor upon chest auscultation, and her first and second heart sounds were normal. Laboratory data showed an elevation in her white blood cell count (12,500/μL). C-reactive protein, creatine kinase (67 IU/L, normal range <163 IU/L), and D-dimer levels were all within normal limits. An arterial blood gas test revealed hypoxemia (PaO_2 50.0 mmHg) without retention of carbon dioxide. An electrocardiogram (ECG) showed sinus tachycardia with a heart rate of 105 beats/min, and ST changes were not observed (Figure 1A).

An asthma attack was suspected, and treatment was initiated with intravenous steroids, nebulized short-acting beta-adrenergic agonist, transdermal long-acting beta-adrenergic agonist, and oxygen administration. Appropriate asthma treatment improved clinical signs and hypoxia on day 3. On day 4, electrocardiographic monitoring revealed a sudden onset of narrow QRS tachycardia. A 12-lead surface ECG demonstrated atrial flutter with 2:1 atrioventricular conduction and ST elevation in leads II, III, aVF, and V_1-V_4, as well as negative T waves in leads V_4-V_6 (Figure 1B). Atrial flutter continued for 20 min and spontaneously returned to a sinus rhythm with premature atrial stimulation (Figure 1C). Transthoracic cardiac echocardiography immediately after termination of the atrial flutter showed akinesis of the apical and mid-segments of the left ventricle with normal contraction of the basal segments, and the ejection fraction measured by the modified Simpson's method was 40% (Figure 2A). Blood examination exhibited a slight elevation in the levels of creatine kinase (226 IU/L) and of high-sensitivity cardiac troponin T (hs-cTnT) (0.10 ng/mL, normal range <0.10 ng/mL). On day 6, the level of creatine kinase decreased to 126 IU/L, hs-cTnT increased to 0.13 ng/mL, with a creatine kinase-muscle brain form level of 16.1 ng/mL (normal range <7.5 ng/mL). Because of her condition, neither cardiac catheterization nor coronary CT angiography was performed. Her left ventricular asynergy improved on day 10 without any specific treatment and normalized on day 15 (Figure 2B). Her ECG showed giant inverted T waves with QT prolongation on day 15 (Figure 1D). Thereafter, her clinical course was uneventful and she was transferred to her previous hospital on day 20.

This case report was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent was obtained from the patient's family for publication of this case report and any accompanying images.

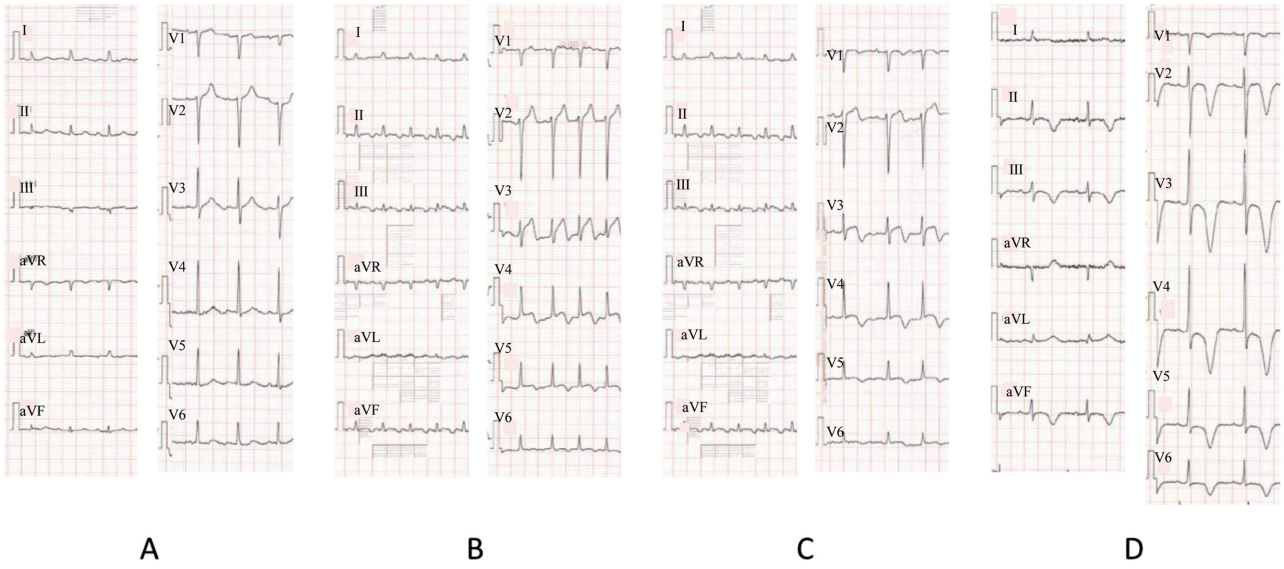

Figure 1 Electrocardiograms of the presented patient. On admission (A), sinus tachycardia with a heart rate of 105 beats/min was shown, and ST change was not present. On day 4, atrial flutter with 2:1 atrioventricular conduction, and ST elevation in leads II, III, aVF, and V_1-V_4, as well as negative T waves in leads V_4-V_6 were observed (B), and returned spontaneously to sinus rhythm with premature atrial stimulation (C). On day 15, giant inverted T waves with QT prolongation were found (D).

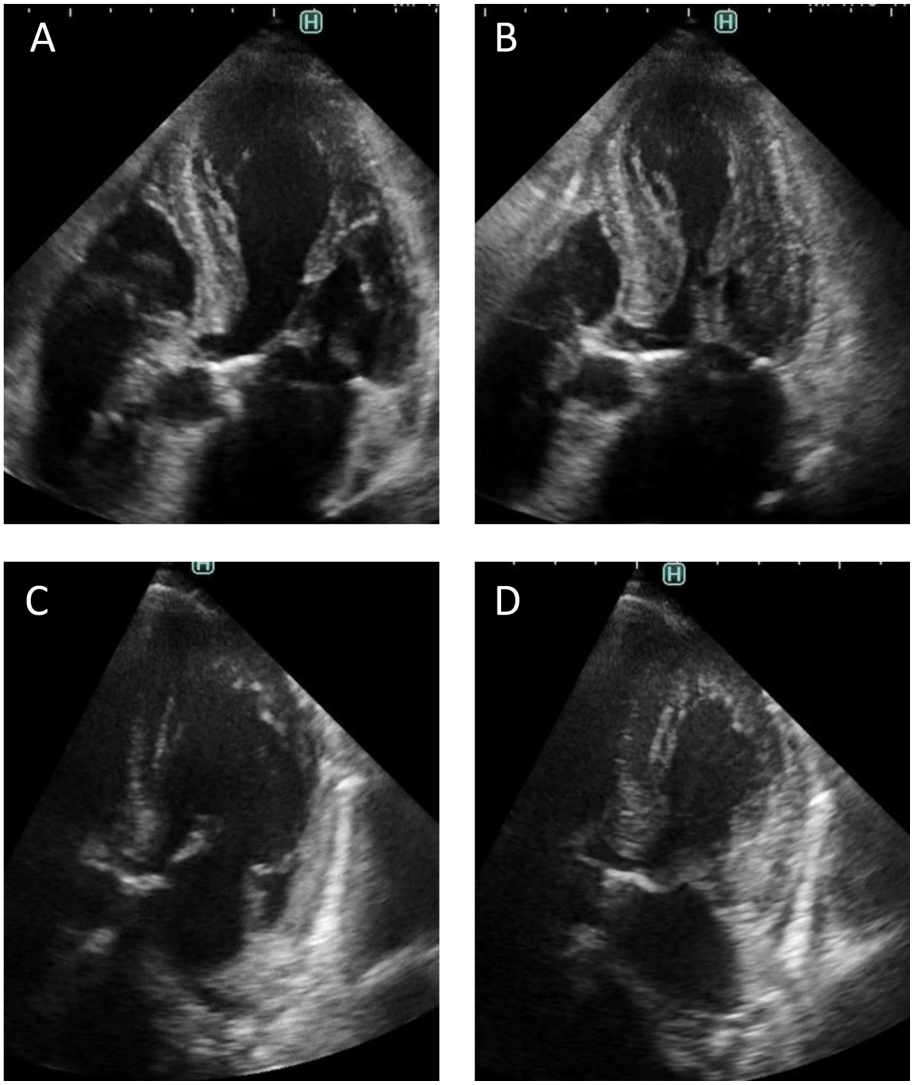

Figure 2 Echocardiogram of end diastole (A) and end systole (B) of the presented patient just after termination of atrial flutter. Akinesis of the left ventricular apex and mid segments was found on the apical 4-chamber view. Echocardiogram of end diastole (C) and end systole (D) on day 15 was performed. The wall motion of the left ventricle normalized. LV, left ventricle.

Discussion

The emanating point of interest in this case is that TTC may occur even in a patient with a severe stage of dementia who is bedridden and has communication difficulties. Clinical physicians should be aware of the possibility that patients with dementia who cannot complain of any symptoms may experience TTC.

Diagnosis of TTC is becoming common owing to heightened awareness[7]. Currently, there is no single universally accepted diagnostic definition of TTC. The Mayo Clinic diagnostic criteria are widely used in clinical practice and research[8, 9]. Our patient demonstrated transient left ventricular apical-mid dysfunction, electrocardiographic abnormalities, such as ST-segment elevation and T-wave inversion, and modest elevation in cardiac troponin levels. The absence of obstructive coronary disease, angiographic evidence of acute plaque rupture, or coronary artery spasm could not be excluded in this case. However, the ventricular wall motion abnormalities in this case seemed typical of TTC and could not be explained by a single epicardial coronary artery obstruction. Spasms of multiple epicardial vessels may contribute to TTC in a subset of patients. However, the causal role of coronary spasm has been questioned by many authors and is still controversial[10, 11].

Although prognosis of TTC is generally favorable and left ventricular wall motion abnormalities can rapidly improve, increasing evidence reveals that serious complica-

tions occur occasionally[12, 13]. Congestive heart failure and left ventricular apical thrombosis are common complications of TTC, while cardiogenic shock, arrhythmias, and ventricular rupture have also been described[2, 14]. Because these serious complications occur, especially in the early stages of disease, timely diagnosis is important. Chest pain and dyspnea are the most common presenting symptoms in TTC patients[1, 9]. Our patient, however, was unable to complain of any symptoms because of cognitive deterioration and communication difficulties due to the advanced stage of Alzheimer's disease. After finding evidence of tachyarrhythmia on the ECG monitor, a 12-lead surface ECG was performed, showing the presence of TTC. The ECG features of TTC are not diagnostic, but new ECG changes are useful for the early detection of TTC[15], especially in patients with communication difficulties, as in our case.

The precise pathophysiological mechanisms of TTC remain unclear, but sympathetic stimulation has been proposed as a central pathogenesis[10]. TTC has been associated with conditions of catecholamine excess and clinical features of TTC and wall motion abnormality can be caused by intravenous administration of catecholamines and beta-agonists[16]. Previous studies have reported an association between TTC and asthma attacks. Ind *et al.* demonstrated an increase in catecholamine levels in patients with acute asthma relative to healthy individuals[17]. Moreover, intravenous epinephrine and inhaled beta-adrenoceptor agonists may increase the risk of TTC[18, 19]. Thus, the asthma attack itself and/or administration of beta-adrenoceptor agonists could trigger TTC in this case. Some reports showed an association of TTC with Alzheimer's disease[20–23]. One possible explanation for the etiopathogenesis of TTC in individuals with Alzheimer's disease could be the increase in catecholamine concentration in the blood[24]. Thus, careful attention to the occurrence of TTC is required for patients suffering from an asthma attack, especially for patients with Alzheimer's disease.

Conclusion

Takotsubo cardiomyopathy may occur even in patients with severe dementia, who are bedridden and show communication difficulties. Future clinical studies investigating the prevalence of TTC in these patients are required.

Conflict of interest: The authors declare no conflicts of interest associated with this study.

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