A Case of Takotsubo-Shaped Hypokinesis of the Left Ventricle Caused by a Lightning Strike

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SUMMARY

A 62-year-old woman was struck by lightning while on a mountain and fortunately did not suffer burns or unconsciousness. She stayed at a mountain lodge overnight and was taken to our hospital by helicopter the next day. Upon admission, electrocardiography showed ST segment elevation indicating acute lateral myocardial infarction, and echocardiography showed takotsubo-shaped hypokinesis of the left ventricle indicating an apical aneurysm. Her serum enzyme levels were increased, as is typical in cases of myocardial infarction, however, she did not complain of cardiac symptoms. Coronary arteriography performed 4 days after admission showed a normal coronary artery while left ventriculography showed apical akinesia. An echocardiogram obtained 2 days later showed resolution of the LV wall motion abnormality. This is the first reported case of takotsubo cardiomyopathy caused by lightning. Takotsubo-shaped hypokinesis is not described as a complication of lightning-induced cardiac injury and its pathogenesis remains controversial. (Int Heart J 2005; 46: 933-938)

Key words: Takotsubo-shaped hypokinesis, Lightning, Catecholamine-induced cardiomyopathy

CASE REPORT

A 62-year-old woman was admitted to our hospital in July 2004 after being struck by lightning. Before the event, she had been healthy, and the results of electrocardiography (ECG) performed at her annual health examinations were normal. She was climbing Mt. Hakuba (2932 meters) with two other people when her walking-stick was struck by lightning. All three stayed at a mountain lodge that night, and the next day they were brought to our hospital by helicopter. Upon admission, the woman complained of a slight headache, but there were no cardiac symptoms. However, one of her companions had sustained second-degree burns

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Figure 1. Patient electrocardiograms. A: Twelve-lead electrocardiogram obtained upon admission depicts ST segment elevation in leads V3 to V6. B: Twelve-lead electrocardiogram obtained three days later reveals negative T waves in leads II, III, aVF, and V2 to V6. C: Twelve-lead electrocardiogram obtained ten days later reveals regression of T wave inversion.

Figure 2. Echocardiographically depicted reversible apical wall motion abnormality. A: Echocardiogram obtained upon admission depicts takotsubo-shaped hypokinesis of the left ventricle. B: Echocardiograms taken 10 days after admission show improvement of the apical wall motion. RA indicates right atrium; RV, right ventricle; and LV, left ventricle.
on 20% of his body, and the other was admitted after cardiac and respiratory arrest.

The patient's temperature was 37.0 centigrade, her blood pressure was 114/68 mmHg, and her pulse was 88 beats per minute. Physical examination revealed no burns. There were no rales. Cardiac auscultation revealed a grade 1 systolic ejection murmur. No neurologic abnormalities were found. Blood tests revealed a white blood cell count of 11,100/µL. Serum enzyme levels were increased, as is typical in cases of myocardial infarction; the creatine phosphokinase (CPK) level was 1548 IU/L (normal range: 30-165), CPK-MB 35.5 ng/mL (normal: < 5.0), and troponin T was positive. Chest X-ray findings were normal.

ECG (Figure 1A) study detected ST segment elevation in leads V3 to V6 and echocardiography (Figure 2A) showed an apical aneurysm and so-called takotsubo-shaped hypokinesis of the left ventricle without apparent valvular abnormalities (ejection fraction; 65%). Both ventricles were of normal size, and there was no pericardial effusion. A diagnosis of acute myocardial infarction was made on the basis of these findings. Oral administration of aspirin at 81 mg/day and isosorbide mononitrate at 40 mg/day was started. Four days after admission,

Figure 3. Coronary arteriography and left ventriculography. A: Coronary arteriography showed normal coronary arteries. B: Left ventriculography showed apical akinesia. RCA indicates right coronary artery; LCA, left coronary artery; EDV, end-diastolic volume; ESV, end-systolic volume; and EF, ejection fraction.
coronary arteriography showed both arteries to be normal, and ventriculography showed an apical akinesia (Figure 3). In the course of ten days, ECG (Figure 1B and C) revealed negative T wave and regression of T wave inversion, and serum enzyme levels were normalized. Furthermore, repeat echocardiography (Figure 2B) showed normal left ventricular motion.

During the course of the illness, there was no sign of heart failure. Her serum norepinephrine level 3 days after admission was 602 pg/mL, which is significantly higher than the normal range (100-450 pg/mL) (Table). However, serum norepinephrine had decreased to a normal level 8 days after admission. The results of repeated tests for serum levels of epinephrine and dopamine were normal; however, these levels were decreased in the normal range. The results of repeated tests for urinary levels of epinephrine, norepinephrine, and dopamine were also normal. Therefore, a diagnosis of catecholamine-induced takotsubo-shaped cardiomyopathy was made.

The patient was discharged on the 11th day without administration of aspirin and isosorbide mononitrate.

**DISCUSSION**

The Japanese Ministry of Health, Labour, and Welfare reported that approximately 22 people are killed each year in Japan by lightning strikes (5 to 58 patients per year from 1954 to 1989). Lightning ranges from 2 million to 200 million volts and from 10 to 500 kilowatt hours of energy. The important feature of lightning-induced injury is the high mortality rate; 80% of reported patients died immediately. Cardiac involvement is the most important aspect of injury by a lightning strike. Lightning strike produces arrhythmia with the potential to cause cardiac arrest. Some mechanisms have been proposed to account for the cardiac damage seen in lightning-induced injury. These include the induction of coronary artery spasm, catecholamine-mediated injuries, direct thermal

### Table. Catecholamine Levels in Serum and Urine

<table>
<thead>
<tr>
<th>Catecholamine</th>
<th>Normal range</th>
<th>Time after admission</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>3 days</td>
</tr>
<tr>
<td>Serum (pg/mL)</td>
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<tr>
<td>Epinephrine</td>
<td>&lt; 100</td>
<td>51</td>
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<tr>
<td>Norepinephrine</td>
<td>100-450</td>
<td>602</td>
</tr>
<tr>
<td>Dopamine</td>
<td>&lt; 20</td>
<td>11</td>
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<tr>
<td>Urine (µg/day)</td>
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</tr>
<tr>
<td>Epinephrine</td>
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<td>10.7</td>
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<tr>
<td>Norepinephrine</td>
<td>48.6-168.4</td>
<td>138.6</td>
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<tr>
<td>Dopamine</td>
<td>365-961</td>
<td>589.3</td>
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injury, ischemia secondary to arrhythmia-induced hypotension, and coronary artery ischemia as part of a generalized vascular injury.

Takotsubo-shaped hypokinesis of the left ventricle has been documented in patients in Japan; it is characterized by an apical wall motion abnormality, sometimes in the absence of coronary artery disease. Akashi, et al reported takotsubo-shaped hypokinesis following an episode of polymorphic ventricular tachycardia especially with a short coupling interval, and following an episode of pneumothorax of the left lung. Our patient did not complain of palpitations, there were no signs of arrhythmia, and the chest X-ray findings were normal. Some reports have stated that this type of cardiomyopathy developed because of catecholaminergic or adrenoceptor-hyperactivation due to stress. Wittstein, et al reported that emotional stress could precipitate severe, reversible left ventricular dysfunction in patients without coronary disease. Also, it has been reported that a takotsubo-shaped left ventricle appeared to be induced by exposure to high-levels of plasma catecholamines. Singal, et al reported that catecholamines were a potential source of oxygen-derived free radicals and cause myocyte injury. Kurisu, et al suggested that changes in myocardial perfusion and metabolism due to catecholaminergic or adrenoceptor-hyperactivation could also cause takotsubo-shaped left ventricular dysfunction.

Lightning strikes may be the cause of catecholamine production. Our patient’s urinary catecholamine levels were examined repeatedly, and they remained normal. However, the serum norepinephrine level 3 days after admission was above the normal range and later decreased, which suggests that transient catecholaminergic or adrenoceptor hyperactivation was present. Furthermore, the decreasing serum norepinephrine level was accompanied by an improvement in left ventricular motion. This catecholamine-induced cardiomyopathy by a lightning strike appears to be the most probable mechanism in our case.

Other mechanisms have been proposed. Although electrical currents can damage the walls of the coronary arteries, there may also be a direct thrombogenic effect. Ekoe, et al reported a case of cardiogenic shock complicated by disseminated intravascular coagulation caused by lightning. Furthermore, it has been reported that direct current shocks cause cardiac dysfunction because of circulatory disturbance in endothelial and epicardial microvessels. The energy level of a lightning strike is much higher than that of a direct current shock, thus, it is also possible that lightning induced myocyte necrosis and circulatory disturbance in her microvessels.

Takotsubo-shaped hypokinesis of the left ventricle is not described as a complication of lightning-induced cardiac injury, and its pathogenesis remains controversial. The prognosis of patients with no symptoms after a lightning strike...
is reportedly good without any form of treatment; however, we will continue careful clinical and laboratory monitoring of our patient.

REFERENCES

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