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Case Report

A Case of Takotsubo Cardiomyopathy Following a Spinal Epidural Hematoma

Akira Tempaku, MD*

Department of Neurosurgery, Hokuto Hospital. 7-5 Inada-cho-kisen, Obihiro, Hokkaido, Japan.

*Corresponding Author: Akira Tempaku, Department of Neurosurgery, Hokuto Hospital, 7-5, Inada-cho-kisen, Obihiro, Hokkaido, 080-0833, Japan,

Abstract

Introduction: Takotsubo cardiomyopathy is known to be a cardiac dysfunction associated with neurological disorders. While it often occurs as a complication following a stroke or head injury, it rarely develops as a result of spinal cord disorders. The author has encountered a patient accompanied with takotsubo cardiomyopathy following surgical treatment for a spinal epidural hematoma from lower cervical to upper thoracic level.

Case: A 75-year-old woman was admitted by the emergency medical service presenting with sudden weakness in both lower limbs. Patient was found to have motor and sensory deficit below forth thoracic (Th4) level. Following magnetic resonance imaging (MRI) check, patient was diagnosed with an epidural hematoma of the spinal cord at the sixth cervical (C6) to seventh thoracic (Th7) level. Patient subsequently underwent emergency epidural hematoma evacuation and laminectomy. On the fifth day following surgery, patient developed secondary takotsubo cardiomyopathy. Her cardiac dysfunction improved with conservative medical treatment involving fluid volume replacement with anticoagulant infusion.

Discussion: It was believed that an epidural hematoma of the spinal cord extending from the lower cervical spine to the upper thoracic spine suppressed sympathetic nerve activity. Then, operative decompression of spinal cord led to the development of takotsubo cardiomyopathy. It was thought that rapid adrenergic nerve activity fluctuations resulting from the functional recovery from a dysfunction of the sympathetic nervous system were the cause of cardiac dysfunction.

Keywords: Takotsubo cardiomyopathy, Spinal Epidural Hematoma, Sympathetic Nerve.

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Introduction

Takotsubo cardiomyopathy was first reported in 1990 as a cardiac dysfunction associated with brain disorders [1, 2]. It can be triggered not only by stroke [3-7] and head trauma [8, 9] but also by psychological stress [10-12]. It is considered to be primarily caused by impaired regulation of cardiac function due to excessive catecholamines and is categorized as one of the neurocardiac disorders [13-17]. While plenty reports and reviews of central nervous system disorders causing takotsubo cardiomyopathy involve brain lesions [3-9, 18], cases associated with spinal cord disorders are rare [19-26].

The author reports a case of takotsubo cardiomyopathy associated with an epidural hematoma in the cervical to thoracic spinal cord. There have been three previously reported cases of takotsubo cardiomyopathy associated with thoracic spinal cord subdural hematomas [19-21]. In addition, there have been reports of takotsubo

cardiomyopathy caused by spinal epidural hematoma resulting from trauma or surgical complications [22-25]. However, based on the previous literature examined, only one case of takotsubo cardiomyopathy associated with a spontaneous spinal epidural hematoma was identified [26]. Spinal epidural hematoma is known to a low incidence rate of approximately 0.2 cases per 100,000 people [26-28]. Then, takotsubo cardiomyopathy associated with spinal epidural hematoma is thought to be even less common. As these are rare pathological conditions, the author discusses its pathophysiology.

Case Presentation

A 75-year-old woman was emergency transported to the hospital complaining of sudden paralysis in both lower limbs. She had no trauma, hard works including rift-up heavy objects or excessive body trunk movement. At the initial presentation, the patient was in clear consciousness, with blood pressure was 137/66 mmHg, heart rate was 52/minute, and body temperature was 36.3°C. Patient had no speech impairment and no paralysis of the upper limbs. The lower half of her body, including both lower limbs, was completely paralyzed—both sensory and motor functions—within the dermatome at and below the Th4 level. Bladder and rectal disturbance were also noted. A head MRI revealed no significant acute intracranial abnormalities. A spinal MRI revealed an epidural hematoma with spinal cord compression in the range from C6 to Th7 (Figure 1).

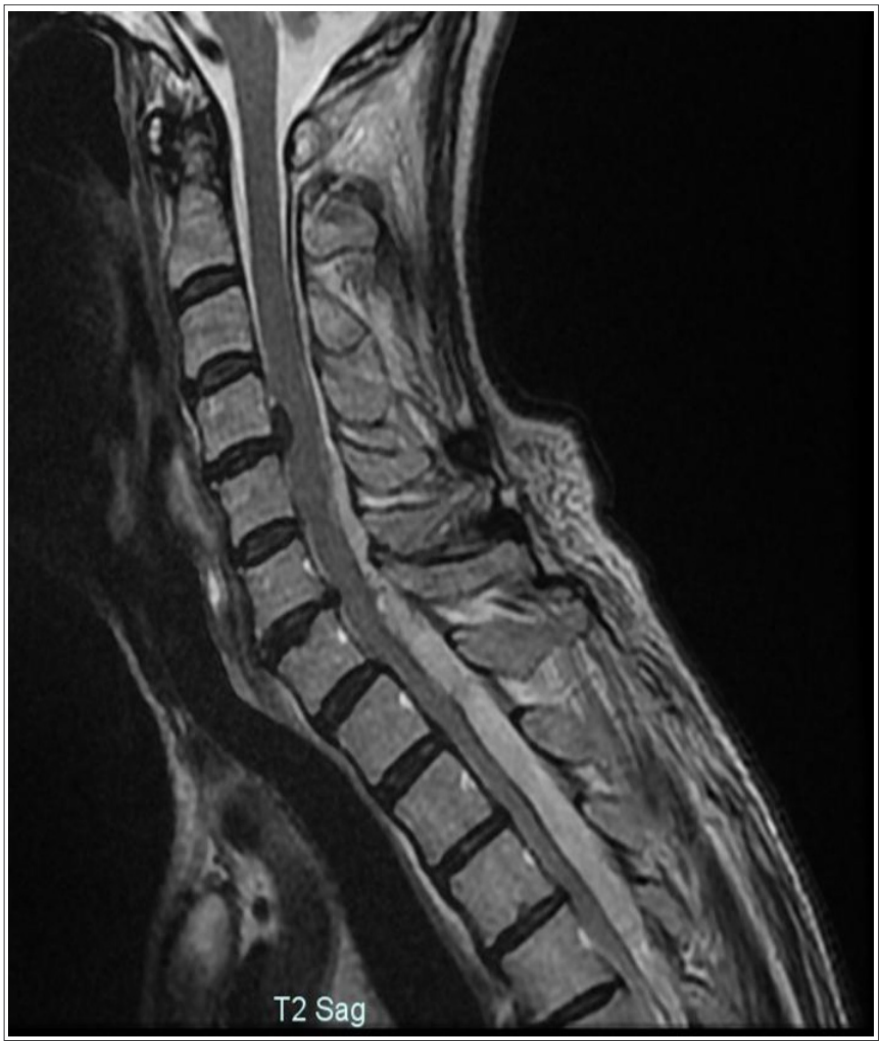

Figure 1. *Sagittal T2-weighted MRI images of the cervical and thoracic spine. From C6 to Th7, a diffuse, well-defined occupying lesion with slight lower intensity than the cerebrospinal fluid is noted on the dorsal side of the spinal cord.*

Following emergency admission, the patient underwent urgent thoracic laminectomy and epidural hematoma evacuation under general anesthesia in the prone position. Postsurgical decompression of the cervical and thoracic spinal cord was achieved satisfactorily. However, improvement in motor and sensory deficits in the lower body was limited.

Subsequently, on the fifth day after admission and surgery, cardiac dysfunction was observed. The patient presented with wheezing accompanied by hypotension (blood pressure in the 70th) rather than chest pain. An electrocardiogram (ECG) revealed ST-segment elevation in leads III, aVF, and V2–V4. Echocardiography revealed apical left ventricular akinesis and hypercontractility

of the left ventricular base. These results consistent with takotsubo-like wall motion abnormalities. Cardiac catheterization revealed moderate coronary artery stenosis (LAD #7, 50%). Additionally, left ventricular ejection fraction showed poor apical wall motion (Figure 2), leading to a diagnosis of takotsubo cardiomyopathy. Blood tests revealed elevated levels of brain natriuretic peptide (BNP) (948.8 pg/mL), creatine phosphokinase-MB (CPK-MB) (26.8 U/L), norepinephrine (0.53 ng/mL), cortisol (54.1 μg/dL), and adrenocorticotropic hormone (ACTH) (111.0 pg/mL). Renin activity and thyroid hormone levels were within normal ranges.

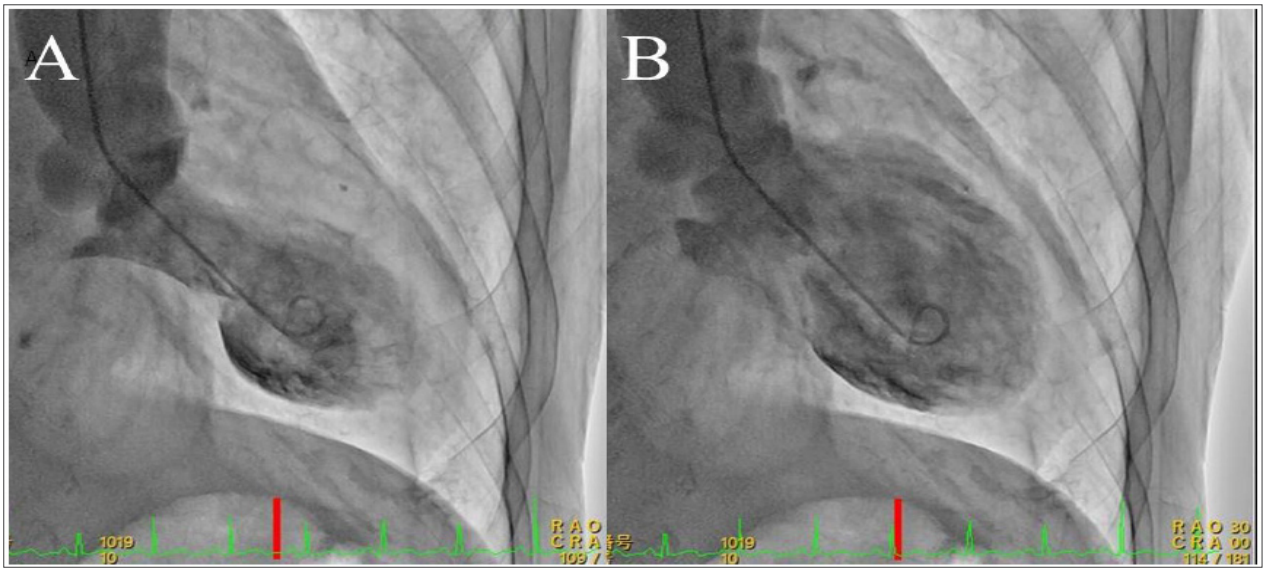

Figure 2. *Cardiographic findings regarding left ventricular wall motion. The morphology of the left ventricle was assessed under radiographic guidance using an iodine-based contrast agent. (A) It shows the morphology of the left ventricle during systolic phase. (B) It shows the morphology of the left ventricle during diastolic phase.*

Conservative treatment was initiated to maintain cardiac output through fluid volume replacement. Anticoagulant of heparin was also infused to avoid thrombosis. Cardiac dysfunction gradually improved.

Following spinal surgery and cardiac treatment, the patient continued rehabilitation with the goal of restoring lower limb function. Neurological function recovered gradually, and the patient acquired independent walking ability. One year later, the patient's modified Rankin Scale (mRS) score was 2.

Discussion

Takotsubo cardiomyopathy is characterized by impaired wall motion of the apex, causing the heart to assume a "takotsubo" shape (a configuration in which the apex remains dilated) during ventricular systole phase. The apex has a high density of β-receptors and exhibits high myocardial responsiveness to sympathetic stimulation. From these conditions, abnormal cardiac wall motion is triggered by excessive stimulation resulting from a sudden increase in endogenous catecholamine levels [29-32].

Sympathetic nerve outflow is transmitted via descending pathways from the brainstem through presynaptic sympathetic neurons in the thoracic and lumbar spinal cord. Consequently, functional impairment becomes markedly apparent following injury to the upper thoracic spinal cord [33-35].

Sympathetic ganglia are distributed from the lower cervical spinal cord to the upper thoracic spinal cord and control the heart rate [36]. The first hypothesis in this case is the sympathetic nervous system was suppressed due to spinal cord compression caused by an epidural hematoma. Surgical decompression of the spinal cord relieved this suppression. It might bring to excessive activity of the adrenergic nervous system, which in turn triggered takotsubo cardiomyopathy.

These sympathetic ganglion blocks have been reported to be effective in treating cardiac arrhythmias associated with takotsubo cardiomyopathy [37, 38]. This appears to provide indirect support for the hypothesis, that decompressed sympathetic nerve brought heart wall motion abnormality.

Another point of view, the adrenal medulla is controlled by the mid-thoracic spinal cord via the celiac ganglion [39]. The second hypothesis is catecholamine production was reduced due to inhibition caused by compression of the thoracic spinal cord from an epidural hematoma. Subsequently, it is conceivable that the disinhibition resulting from decompression of the thoracic spinal cord following surgical treatment caused excessive catecholamine production. Those hormonal upregulations might trigger takotsubo cardiomyopathy. However, endocrine tests conducted after the onset of dilated cardiomyopathy revealed only a slight increase in

catecholamines. In contrast, cortisol and ACTH levels were elevated. Following to these findings, since no sharp rise in catecholamines was observed, it seemed unlikely that there was a causal relationship with takotsubo cardiomyopathy. Based on those findings and knowledge, it was considered more likely that the induction of wall motion abnormalities was due to sympathetic nervous system activation rather than a catecholamine surge.

It was difficult to certain a direct causal relationship. However, based on clinical symptoms and medical course, it was inferred that the excessive sympathetic nerve activities caused takotsubo cardiomyopathy in pathophysiological process.

However, there are still discussion points that need to be clarified regarding the current hypothesis. In particular, the mechanism behind the time lag—specifically, the fact that the patient developed takotsubo cardiomyopathy five days after the surgical resolution of compressed cervical and thoracic spinal cord caused by an epidural hematoma—remains unclear. Research is needed to determine whether this delay was due to incomplete decompression achieved by surgery or whether recovery from spinal shock and the subsequent reactivation of the nervous system required several days or more. Further examination of similar cases in the future is considered necessary.

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Author's Contribution

A Tempaku: Conception of the work, design of the work, acquisition of data, analysis of data and interpretation of data. Drafting the work and revising the work critically for important intellectual content. Final approval of the version to be published. Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Ethical Statements

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and later versions.

Conflict of Interest: The author declares no conflict of interest.

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