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Sudden Coronary Spastic Angina of a Patient with Spinal Cord Injury

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Abstract: A 61-year-old man could not move after heavily drinking alcohol and fell down. Upon arrival at our hospital, he showed abdominal breathing, paraplegia and bradycardia. Based on a high-intensity signal from C4 through C6 on magnetic resonance imaging (MRI), he was diagnosed with spinal cord injury (SCI). On the morning of day 5 of hospitalization, he suddenly entered sinus arrest after a short duration of bradycardia. He was resuscitated after immediate chest compression. ST- segment elevation on ECG and high levels of serum troponin I suggested acute coronary syndrome (ACS). There was no stenosis of any coronary arteries on coronary angiography. After the injection of acetylcholine chloride (50 μg) into the left coronary artery, excessive spasm and contrasting delay appeared. We diagnosed coronary spastic angina (CSA) and administered vasodilators. After rehabilitation, he recovered his ability to walk. He had neither cerebral hypoxia nor angina. Cardiovascular complications often occur with SCI above T6, due to hyperactivity of the parasympathetic nerve system. These complications are particularly likely during the first two to three weeks after injury. Careful management is necessary to avoid fatal cardiovascular complications of not only bradycardia, but also CSA during the acute phase of SCI.
Keywords: acute coronary syndrome (ACS), spinal cord injury (SCI), coronary spasm angina (CSA), cardiac arrest, autonomic nerves, cervical cord injury.

INTRODUCTION

Between 250,000 and 500,000 people suffer from spinal cord injury (SCI) every year worldwide [1]. Although thromboembolism and arrhythmia are common complications of SCI, acute coronary syndrome (ACS) is also often reported as a complication, and the risk of ACS is increased with SCI [2]. These complications of the cardiovascular system are particularly likely in the acute phase of SCI [3]. We herein report an SCI patient who suddenly entered cardiac arrest after undergoing surgery for SCI. After resuscitation, angiography revealed coronary spastic angina (CSA).

CASE PRESENTATION

A 61-year-man fell down after heavily drinking alcohol and was transferred to our emergency department because he could not move. On arrival, he showed alert consciousness, open airway, normal blood pressure (118/66 mmHg), slight bradycardia (47/minute) and normal SpO_2. He showed abdominal breathing, paraplegia, numbness to both arms and desensitization to both lower limbs. According to these findings, we diagnosed him with SCI, American Spinal Injury Association impairment scale grade C (ASIA C).

He had untreated diabetes mellitus and smoked 30 cigarettes per day. Computed tomography demonstrated no fracture of the spine, but magnetic resonance imaging (MRI) revealed a high-intensity signal from C4 through C6 (Fig. 1). His initial 12-lead electrocardiogram (ECG) showed sinus bradycardia (Fig. 2). His serum troponin I level was 0.015 ng/mL (normal range: 0-0.06 ng/mL).

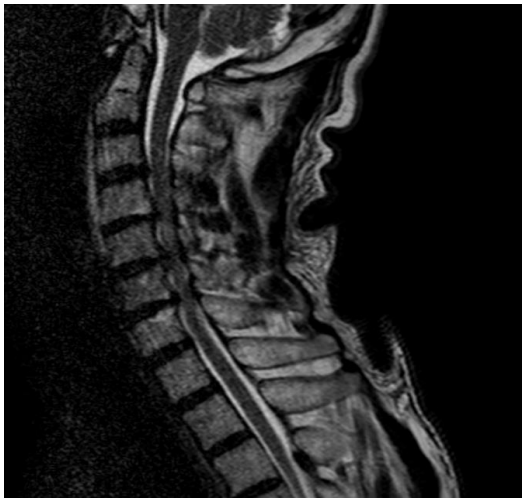

Fig-1: MRI: Sagittal T2-weighted MRI revealed a high-intensity signal from C4 through C6

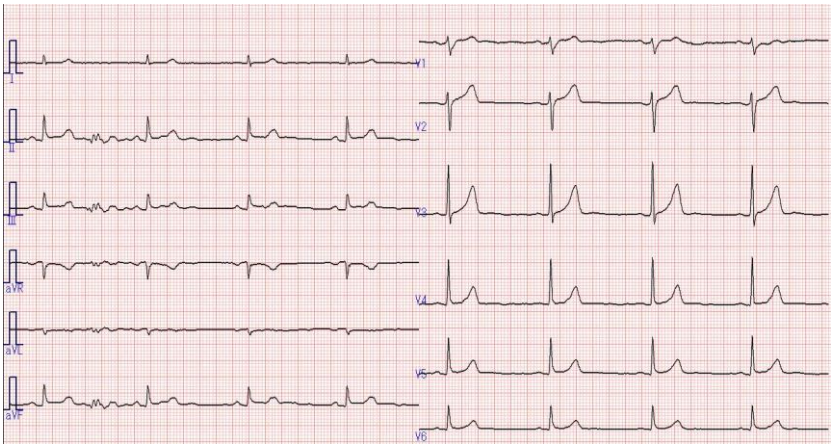

Fig-2: The ECG is only bradycardia on admission

Because he was not elderly and had no other injuries, he underwent laminectomy of C3-6 for decompression and nerve recovery on the day of admission. His vital signs were stable during the operation. He was extubated the day after the operation, and his paralysis recovered gradually. His cardiac rate became normal.

On day 5 after the operation, he suddenly experienced bradycardia (30/minute) without any symptoms. Intravenous atropine (0.5 mg) was given, but he entered sinus arrest (Fig. 3). After chest compression for two minutes, he was resuscitated. ECG revealed ST-segment elevation in leads II, III, aVF, V5 and V6 on the contrary ST-segment depression in leads V2-4 (Fig. 4). His serum troponin I level was 1.9 ng/mL. Because of ECG alterations and elevated troponin, we diagnosed him with ACS, for which we performed emergency coronary angiography.

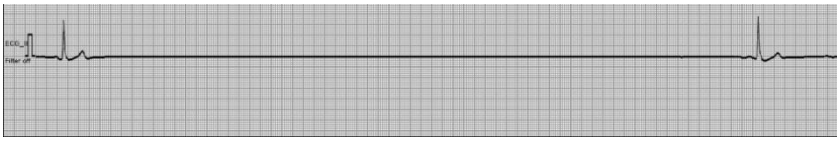

Fig-3:The holter ECG after bradycardia revealed sinus arrest

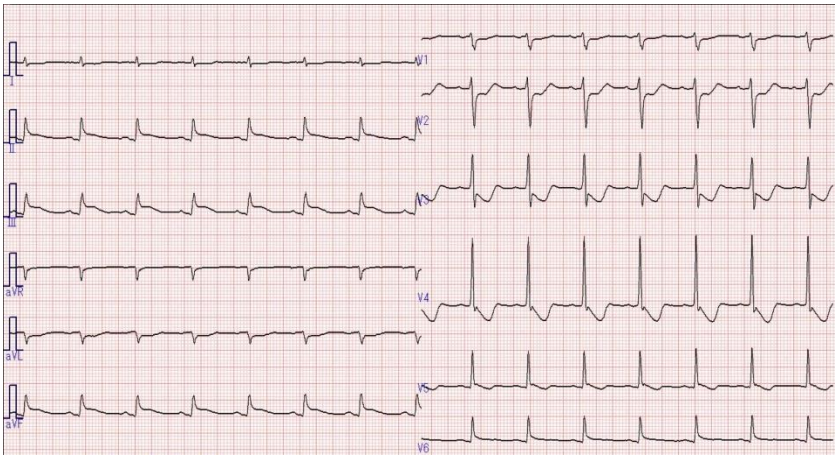

Fig-4:The ECG after resuscitation revealed ST-segment elevation in leads II, III, aVF, V5 and V6 on the contrary ST-segment depression in leads V2-4

There was no stenosis of any coronary arteries on coronary angiography (Fig. 5). Acetylcholine chloride (50 μg) into the left coronary artery induced excessive spasm and contrasting delay (Fig. 6). The intracoronary administration of nitroglycerin (5 mg) relieved the severe spasm of the left coronary. As we had diagnosed him with CSA, we started benidipine (2 mg/day), nicorandil (15 mg/day) and aspirin (100 mg/day) by oral administration.

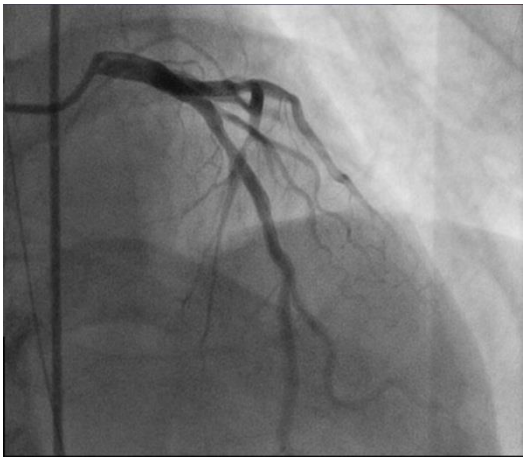

Fig-5: Coronary angiography and the acetylcholine provocation test. There was no stenosis on the left coronary arteries

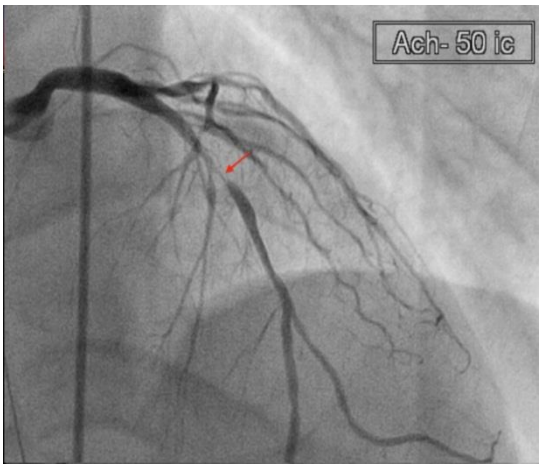

Fig-6: Coronary angiography and the acetylcholine provocation test. Acetylcholine chloride (50 μg) into the left coronary artery induced excessive spasm (arrow)

He was transferred to a rehabilitation hospital for his spinal cord injury on day 41 after surgery without any complications of cardiovascular symptoms nor cerebral hypoxia. After rehabilitation, he was ambulatory and never again experienced angina without any complications of cardiac arrest.

DISCUSSION

It is well known that there are few serious cardiovascular disease complications associated with SCI less than T6. On the other hand, such complications often occur with SCI greater than T6.

This is because sympathetic innervation to the heart is from T3-4 [3, 4]. ACS is the third-leading cause (11%) of death with SCI after sepsis (26%) and influenza/pneumonia (13%) [5]. The risk of ACS in SCI patients was shown to be 17.33 per 10,000 person-years and 1.4 times higher than others in a cohort study [2]. Cardiovascular complications occur in SCI patients due to atrophy of spinal sympathetic preganglionic neurons, instability of autonomic nerves or hyperactivity of the parasympathetic nerve system. This hyperactivity reduces the vessel tone and causes bradycardia, hypotension, coronary slow flow and ACS [3,4,6,7].

On admission, the present patient had no cardiovascular findings except for bradycardia. On day 5 after the operation, he suddenly experienced bradycardia and ACS. Abnormal ECG findings, especially ST elevation, are frequently observed in the acute phase of SCI [8, 9]. Patients with high quadriplegia are particularly prone to symptomatic bradycardia (29%) and primary unprovoked cardiac arrest (16%) during the first 2-3 weeks after injury [3], as the sympathetic nerve is imbalanced in the acute phase [6]. Primary rhythm abnormality is said often to require CPR [10]. In the acute phase, it is crucial to avoid any provocation that may lead to further vagal activation or enhancement of the vasovagal reflexes. Preparing for atropine and catecholamine administration at bedside is recommended.

In the present patient, we diagnosed CSA by the administration of acetylcholine chloride. Since acetylcholine chloride induces spasm of the coronary vessels and bradycardia, involvement of the parasympathetic nervous system is indicated [11]. CSA may occur because of parasympathetic nerve system abnormalities induced by SCI. Onuki *et al.* reported such a case [12], but theirs was the only report at the time. Our report is the second such case and therefore important for study.

We generally administer β-stimulant via catecholamine intravenous injection for sustained bradycardia after SCI and maintain the pulse and blood pressure. The β-stimulant also carries a risk of inducing CSA, but in this patient, CSA occurred without β-stimulant. We need to recognize the risk of CSA with SCI even in catecholamine-free patients, and the preparation for coronary expansion medicine is crucial for the early management of SCI. A pacemaker may be more useful than catecholamine and xanthines, which may activate the autonomic nervous system [7].

CONCLUSION

Serious cardiovascular diseases often occur after the acute phase of SCI with quadriplegia. Prompt, aggressive management is necessary in order to avoid fatal cardiovascular complications, even if there is no arrhythmia. We should bear in mind that these complications may be caused by abnormalities of the autonomic nervous system.

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