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Case Report

Cardiac arrest attributable to dysfunction of the autonomic nervous system after traumatic cervical spinal cord injury

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ABSTRACT

Bradycardia is the most common form of dysrhythmia developing after disruption of the sympathetic pathway by a spinal cord injury (SCI), and it can have fatal consequences, including cardiac arrest. Here, we report a case of cardiac arrest developing after cervical SCI attributable to sympathetic hypoactivity. A 26-year-old male pedestrian was admitted after a traffic accident. Radiologically, fractures were apparent at the C_{6-7} bilateral articular facets, and cord contusion with hemorrhage was evident at C_{4-7}. During his stay in ICU, intermittent bradycardia was noted, but the symptoms were not specific. On the 22nd postoperative day, the patient was taken to the computed tomography suite for further evaluation and experienced cardiac arrest during a positional change. After immediate cardiac massage, the patient was resuscitated. We scheduled Holter monitoring, which detected 26 pauses, the longest of which was 17.9 s. The patient underwent cardiac pacemaker insertion. No further cardiac events were noted.

Introduction

Spinal cord injury (SCI) is the most devastating form of neurological injury, disturbing the normal sensory, motor, and/or autonomic functions.[1] The annual incidence of SCI in the USA is about 40 cases per million of population.[2] SCI is caused principally by trauma (e.g. traffic injuries, falls, and violence). The risk of death is highest in the first year after SCI and varies by level and severity of injury.[3] Cardiovascular disease is one of the principal causes of death.[3,4] In the USA, diseases of the circulatory system are the most common causes of death.[4] In Norway, the main causes of death following the experience of a SCI are pneumonia/influenza, ischemic heart disease, and urogenital disease.[3]

In patients with acute cervical SCIs (rather than thoracolumbar SCIs), cardiovascular deficits, including severe bradycardia, asystole, and loss of peripheral vascular tone, are known complications.[5,6] Acute autonomic imbalances developing after disruption of the sympathetic pathway (located in the cervical cord) are thought to trigger such cardiovascular events.[5–7] Although autonomic dysfunction developing after SCI can have fatal consequences, including cardiac arrest, many clinicians do not recognize such risks, and the conditions are often misdiagnosed. Here, we report a case of cardiac arrest developing after cervical SCI. The immediate cause was autonomic nervous system dysfunction. The patient improved after pacemaker insertion.

Case report

A 26-year-old male pedestrian was admitted to hospital via the emergency room after a traffic accident. He was mentally alert and complained of weakness and numbness of both the upper and lower extremities. On physical and neurological examination, no external injuries were evident except for an abrasion on the left arm. However, paraplegia was apparent. The baseline electrocardiogram (ECG) revealed sinus bradycardia (57 beats/min, Fig. 1). Initial chest and abdominal computed tomography (CT) did not reveal any abnormality or trauma-related injury. A skull fracture, with a small subdural hemorrhage at the right frontal convexity was evident on brain and facial CT. Spinal CT revealed acute fractures at the C_{6-7} bilateral articular facets and the right-side lamina of C_6 (Fig. 2A). Magnetic resonance imaging revealed cord contusion with hemorrhage at C_{4-7} (Fig. 2B).

On the first day of admission, the patient underwent microscopy-assisted corpectomy and discectomy with anterior

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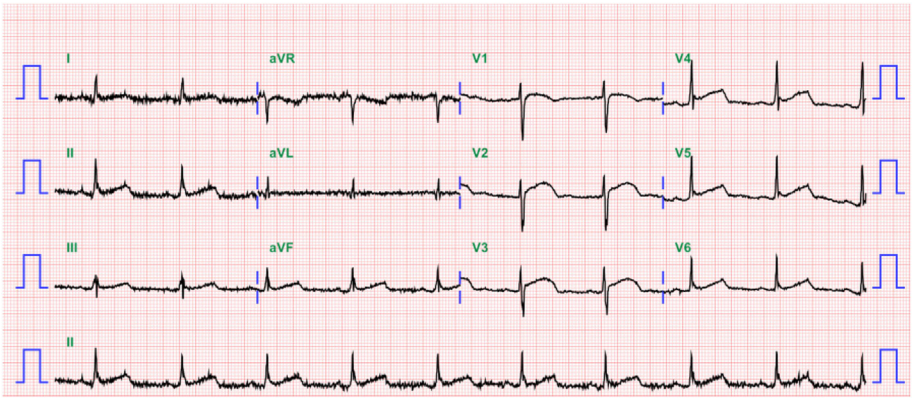

Fig. 1. The baseline electrocardiogram (ECG) revealed sinus bradycardia (57 beats/min).

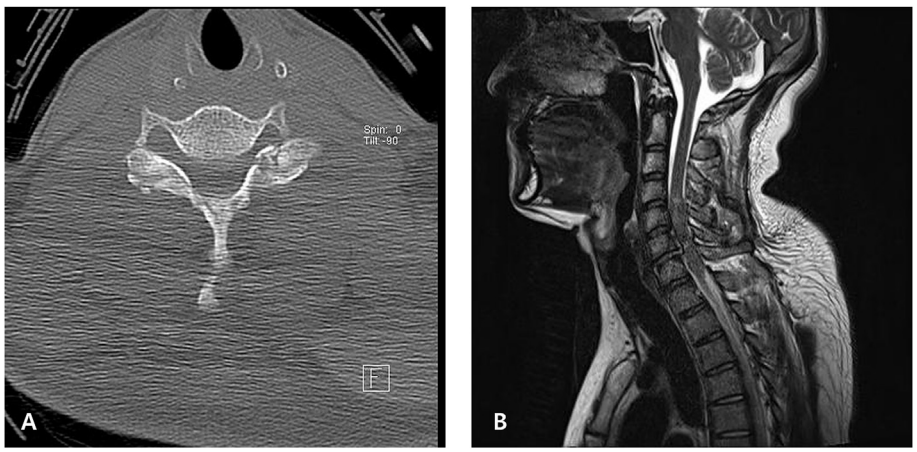

Fig. 2. A: A spinal computed tomography revealed acute fractures at the C_{6-7} bilateral articular facets and the right-side lamina of C_6. B: The magnetic resonance image showed contusion with hemorrhage at C_{4-7}.

interbody fusion. Postoperatively, the patient remained in the ICU for monitoring. On the fifth postoperative day (POD), a pneumonic infiltration with pleural effusion was evident on a follow-up chest X-ray (Fig. 3). Fever developed, and self-expectoration was impossible because respiration was shallow. Bronchoscopy was performed, and a large volume of purulent secretion was evident in the dependent portion. The patient was transferred to the Division of Pulmonology for further respiratory treatment in an ICU. Antibiotics were commenced, ventilator support put in place, and a tracheostomy performed.

During the ICU stay, intermittent bradycardia (30–55 beats/min) was evident, without specific symptoms; his vital signs were stable. Intravenous atropine (0.25 mg) was given twice to treat severe bradycardia; the heart rate was recovered in a few seconds. Echocardiography revealed no specific abnormality, and the left ventricle exhibited normal systolic function (ejection fraction 60%). On the 22nd POD, chest CT was scheduled for differential diagnosis of a pulmonary thromboembolism. During a positional change prior to imaging in the CT suite, the heart rate fell to 23 beats/min, and blood pressure was not palpable at the carotid artery. After immediate cardiac massage over 1 min, the patient was resuscitated and exhibited stable vital signs. Holter monitoring was performed on the day after cardiac arrest (Fig. 4). The average heart rate was 67 beats/min. However, 26 pauses were evident, the longest of which was 17.9 s. A temporary cardiac pacemaker was initially placed because of the patient's poor general condition and

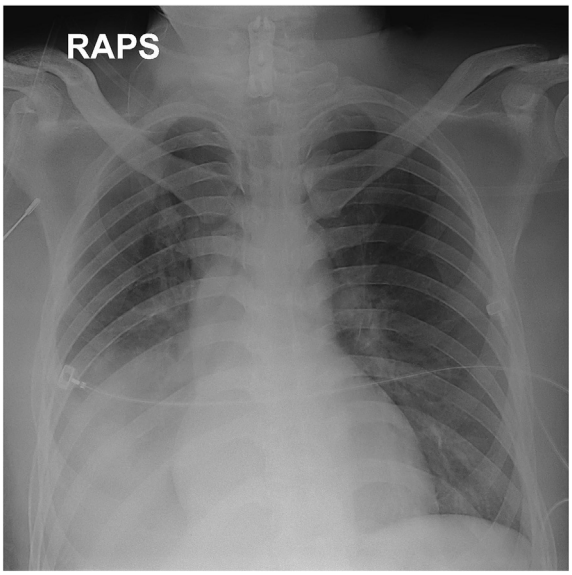

Fig. 3. On the 5th postoperative day, a pneumonic infiltration with pleural effusion was evident on a follow-up chest X-ray.

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