CASE REPORT

Diaphragmatic Paralysis in a Patient with Spinal Cord Infarction

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Abstract

This report describes the rare case of a 72-year-old woman with spinal cord infarction who presented with persistent diaphragmatic paralysis. Her neurological examination showed tetraplegia, sensory loss to pain and thermal stimulations, and paradoxical abdominal movement. Chest X-ray and diaphragmatic fluoroscopy revealed absent diaphragmatic movement. A cervical magnetic resonance image showed bilateral anterior spinal cord lesions from the level of the second to the fifth cervical vertebrae. Diaphragmatic paralysis should be recognized as a clinical sign of cervical spinal cord infarction. Particular attention must be given to paradoxical abdominal movement during respiration in this disorder.

Key words: diaphragmatic paralysis, phrenoplegia, spinal cord infarction, anterior spinal artery syndrome, stroke, respiratory failure

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Introduction

Patients with high cervical spinal cord infarction often present sudden respiratory failure (1, 2). Although various mechanisms can contribute to respiratory failure, any mechanism engenders a poor outcome for the patient. Therefore, respiratory failure should be detected immediately.

Diaphragmatic paralysis leading to respiratory failure is usually caused by compression, a tumor, or surgical complications to phrenic nerves (3-5). Commonly, the respiration of patients with diaphragmatic paralysis exhibits a paradoxical movement of the abdomen: the abdominal wall retracts during inspiration and protrudes during the expiration phase (6). Diaphragmatic paralysis is an uncommon clinical finding in patients with spinal cord infarction. Here, we describe diaphragmatic paralysis in a patient with spinal cord infarction.

Case Report

The patient was 72-year-old woman. When she was sitting at the table for breakfast, she complained suddenly of dyspnea and incapability of moving the limbs, although she had no prior symptom such as neck pain. Minutes later, she fell and presented labored breathing. She later became unconscious during oxygen administration in an ambulance and entered the emergency room of our hospital with a bag valve mask.

She had taken antihypertensive medication for hypertension from the age of 50 years. Her family and life histories were unremarkable. She was a non-smoker.

On admission, her blood pressure was 220/70 Torr, with a pulse of 60 beats/min, and body temperature of 36.9°C. The oxygen saturation monitor showed about 70% during maximal oxygen administration (10 L/min). Therefore, she was intubated and ventilated. After ventilation, she regained consciousness. Neurological examination showed tetraplegia (manual muscle test gave the following findings: proximal arm 0, distal arm 2-3, and leg 3-4), areflexia in the extremities, and loss of sensation of bilateral arms to pain and temperature (dissociated sensory loss). Cervical magnetic resonance image (MRI) demonstrated high-intensity lesions in the symmetrical anterior part of the C2-C5 levels of the spinal cord on T2-weighted image (Fig. 1). In contrast, brain MRI, brain magnetic resonance angiography (MRA), and...
cervical MRA yielded no abnormal findings. Based on sudden tetraplegia with dissociated sensory loss and the anterior cervical spinal cord lesions, a diagnosis of spinal cord infarction (anterior spinal artery syndrome) was made. Aspirin (100 mg/day) and heparin (10,000 U/day) were administered for antithrombotic therapy, although the cause of the ischemic stroke remained unidentified.

By the seventh day after admission, tetraplegia had recovered slightly except for the proximal arm muscles (manual muscle test gave the following results: proximal arm 0, distal arm 3-4, and leg 4). The biceps and triceps tendon reflexes remained diminished, but the patellar and Achilles tendon reflexes became exaggerated. Positive Babinski sign appeared bilaterally. With the recovery of tetraplegia, respiratory thoracic movement was also recovered. Our careful observation revealed characteristic paradoxical abdominal movement under both voluntary and spontaneous breathing (Fig. 2). A chest X-ray revealed that the right diaphragm was fixed at the eighth intercostal space and the left diaphragm, at the ninth intercostal space in both inspiration and expiration phases (Fig. 3). Diaphragmatic fluoroscopy revealed no diaphragmatic movement. Polysomnography revealed no sleep apnea.

On the tenth day, tracheotomy was performed prior to removal of the ventilator. Tracheostomy positive pressure ventilation (TPPV) was begun because of difficulty in expectoration. Two months later, she was discharged from our hospital with nocturnal TPPV and elderly care services including rehabilitation.

After four months from onset, she became able to maintain a standing position. After two years, she was able to walk inside her home with a cane or a handrail. However, the nocturnal TPPV could not be discontinued.

**Discussion**

We reported a 72-year-old woman with spinal cord infarction who presented with persistent diaphragmatic paralysis. Diaphragmatic paralysis was confirmed by chest X-ray and diaphragmatic fluoroscopy. Respiratory problems caused by diaphragmatic paralysis were managed well using the TPPV. Based on the following discussion, we speculate that impairment of the phrenic nuclei contributed to the diaphragmatic paralysis.

Respiratory failure in patients with cervical spinal cord infarction might result from several lesions in the respiratory system: (i) supranuclear lesions (descending motor tract lesions), (ii) nuclear lesions (spinal motoneuron lesions), and (iii) infranuclear lesions (peripheral nerve and muscle lesions) (6).

Howard et al (1998) reported cases of four patients with cervical spinal cord infarction showing respiratory failure.
Figure 2. Paradoxical abdominal movement under respiration. The abdominal wall is retracted during the inspiration phase under voluntary breathing (A) and relaxation (C). It is protruded during the expiration phase under voluntary breathing (B) and relaxation (D).

Figure 3. Chest X-rays in a supine position. Chest X-rays show that diaphragmatic movement is absent in the deep inspiration phase (A) and deep expiration phase (B).

(1) One patient was unable to breathe volitionally due to disturbed voluntary breathing. Another patient was unable to breathe during sleep because of disturbed spontaneous breathing. The others presented apnea caused by the disturbance of both voluntary and spontaneous breathing. The cause of respiratory failure in these patients is considered to be involvement of the corticospinal tract and/or reticulospinal tract because the descending motor pathway of voluntary breathing is considered as the bilateral corticospinal tracts to respiratory muscles (7); the pathway of spontaneous breathing is considered to be the bilateral reticulospinal tracts (8-10).

Unfortunately, the mechanism of diaphragmatic paralysis is not definite in the patient described herein. She became able to breathe volitionally and automatically using thoracic respiratory muscles. Therefore, the mechanism of her respiratory failure apparently differs from that described in earlier reports. Furthermore, she presented paradoxical abdominal movement, which is usually observed in patients who have diaphragmatic paralysis resulting from impairment of phrenic nerves or phrenic nuclei (6). Based on these findings, we consider that the most probable explanation for her condition is bilateral impairment of phrenic nuclei. Nevertheless, it remains unknown whether the patients described in earlier reports suffered from diaphragmatic paralysis in the process of recovery as well as the present patient. Therefore, we cannot deny the possibility that the present patient has the same mechanism as those reported in previous papers, i.e., the bilateral involvements of both corticospinal tract and reticulospinal tract to diaphragm. To confirm the mechanism of diaphragmatic paralysis, electrophysiological studies including diaphragm electromyography and phrenic nerve conduction study might have been useful.

Whatever the mechanism, diaphragmatic paralysis should be detected immediately and managed carefully because patients suffer from respiratory failure and require emergency

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treatment including assisted respiration in the acute stage; they suffer from expectoration and frequently need suction in the chronic stage. Therefore, for early detection, diaphragmatic paralysis should be recognized as a clinical sign of cervical spinal cord infarction. Particular attention must be given to paradoxical abdominal movement during respiration in this disorder.

References