Cervical disc herniation producing acute Brown-Sequard syndrome: dynamic changes documented by intraoperative neuromonitoring

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Abstract

Introduction Brown-Sequard syndrome is an incomplete spinal cord lesion characterized by ipsilateral loss of motor function and contralateral loss of pain and temperature sensitivity, reflecting a hemi-compression or hemi-section of the spinal cord. Cervical disc herniation is an exceptional cause of this syndrome.

Material and methods We report a case of cervical disc herniation causing Brown-Sequard syndrome in a patient with an unusually rapid neurological deterioration associated to cervical extension, which was documented by neuromonitoring.

Conclusion A prompt diagnosis, followed by spinal cord decompression should be warranted. Intraoperative neuromonitoring is a useful tool in preservation of neurologic function in these cases.

Keywords Brown-Sequard syndrome · Cervical disc herniation · Motor-evoked potentials · Neuromonitoring

Introduction

Brown-Sequard syndrome (BSS) is an incomplete spinal cord lesion characterized by ipsilateral loss of motor

function and contralateral loss of pain and temperature sensitivity, reflecting a hemi-compression or hemi-section of the spinal cord. It occurs most often after traumatic injuries or tumor compression to the spinal cord. Cervical disc herniation as a cause of BSS was first described by Stookey [1], but few cases have been reported, representing an exceptional cause of this syndrome [2–13].

We report a case of cervical disc herniation causing BSS in a patient with an unusually rapid neurological deterioration associated with cervical extension, which was documented by motor-evoked potentials (MOPs).

Case report

Institutional review board approval was obtained to perform this report.

An otherwise healthy 51-year-old male physician with a history of 2 weeks of right-side neck and scapular pain developed suddenly and progressive weakness of his right arm and leg (while looking back over his right shoulder talking to another person seated two rows back) with no history of trauma. The patient arrived to the emergency room 2 h after symptoms started; on examination, he presented with M3 paresis in his right upper and lower extremities: long forearm extensors, triceps, flexor digitorum profundus (FDP), interosseous, iliopsoas, tibialis anterior, extensor hallucis longus (EHL) and gastrocnemius-soleus; only quadriceps was normal. He also had loss of vibratory sensation up to the superior iliac spine in the right-side and reduced pain and temperature sensation on the left hemi-trunk and left lower extremity; plantar responses were extensor at the right-side and flexor at the left. These findings were consistent with BSS due to compression of the right-side of the spinal cord.

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Fig. 1 T2-weighted image of MRI showing a large posterior right paramedian C6–C7 herniated disc with cephalad migration and a big disc fragment behind C6 vertebral body, associated with spinal stenosis

Magnetic resonance imaging (MRI) showed a large posterior right paramedian C6–C7 herniated disc with cephalad migration and a big disc fragment behind C6 vertebral body, associated with spinal stenosis (canal diameter = 11 mm at C5 level), causing right-sided compression of the spinal cord; no abnormal cord signal intensity on T2-weighted images was observed (Fig. 1).

While obtaining the MRI, the patient noticed an increase in his right-side weakness; a new examination after the MRI revealed plegic (M0) right FDP, interosseous, iliopsoas, tibialis anterioris, and EHL; right quadriceps remained M5 but gastrosoleus was M2. The patient had a diminished rectal tone and experienced difficulty to urinate.

The patient was prepared for surgery by keeping him in a Philadelphia collar in semi-flexion; a new neurological examination when the patient arrived to the operating room (3 h after finishing the MRI) showed that he recovered to the paresis as he presented upon arrival to the hospital.

The patient underwent awake intubation. Intraoperative neuromonitoring with somatosensory-evoked potentials (SSEP) and transcranial MOPs was initiated immediately after the patient was anesthetized; MOPs were present in both arms and left leg and absent in the right leg before patient positioning. The neck was gently extended to allow an easier Smith-Robinson approach; however, less than a minute later, the signal from both right upper extremity and left lower extremity were lost. After the patient's neck was placed in neutral position, MOPs reappeared (Fig. 2).

A C6 corpectomy and instrumented fusion were carried out using a fibular allograft (Fig. 3). A large amount of herniated disc material was found to be compressing the

spinal cord, and a complete thecal sac decompression was obtained. No deterioration in somatosensory-evoked potentials or motor-evoked potentials was observed during surgery.

Postoperatively, the patient improved rapidly, with a motor index score of 96 12 h after surgery, and without difficulties to urinate. Four days after surgery, he had a normal motor and sensory examination.

Discussion

Cervical disc herniation presenting clinically as acute BSS is very unusual. In this patient, neurophysiological dynamic changes in response to neck position were documented by MOPs, which represent an unreported situation.

BSS involves ipsilateral loss of motor function due to corticospinal tract dysfunction, combined with ipsilateral loss of vibratory sensation and contralateral loss of pain and temperature sensation as a result of spinocerebellar and spinothalamic tract compromise, respectively, reflecting hemisection of the spinal cord in the cervical or thoracic region. The spinothalamic tract crosses the midline of the spinal cord one to two segments cephalad of the entry level; this explains the contralateral deficit in sensation of pain and temperature starting at a dermatome a few levels below the cord injury on the contralateral side, as it was the case in our patient. The symptoms can also be explained by a different degree of spinal cord compression and an individuality of distribution of anterior spinal artery [7].

In a review of the 25 cases of BSS secondary to cervical disc herniation reported in the English literature until 2008 [11], the average age at presentation was 48 years, and the vast majority of cases did not report cervical trauma, with 67% of the cases involving C5–C6 or C6–C7. The reported interval between the onset of symptoms and the diagnosis has ranged from 1 day to 18 months (mean 4.9 months); our patient was diagnosed 2 h after his symptoms started, and the early surgical treatment may explain the rapid and complete neurological recovery, while the literature has reported only about 50% of the cases reaching a normal motor and sensory function [8]. Classical radicular symptoms were absent in our patient; this has been noticed in previous reports, and it is explained since the compression probably affects the cord itself rather than the nerve roots [11].

A characteristic radiographic finding is a paramedian cervical disc herniation with cord compression; usually associated with cervical spinal stenosis [7], as in our patient. The most likely mechanism of neurological deficit was ischemia due to increased pressure secondary to the herniated disc in a narrow canal. We witnessed both clinical and electrophysiological fluctuation in the severity of

Fig. 2 Intraoperative monitoring samples. The recordings as soon the patient was anesthetized are shown in **a**. First dorsal interosseous (FDI), abductor pollicis brevis (APB), tibialis anterioris (AT) and abductor hallucis (AH) muscles were selected. No response was obtained from the right lower extremity muscles (*bottom*). The amplitude was small in both left AH (*upper*) and right FDI and APB (*bottom*). After the neck was extended, the signal from both right upper extremity and left lower extremity were lost (**b**). Those

responses reappeared after the patient's neck was placed in a neutral position (**c**). Cortical somatosensitive-evoked potentials were elicited from the posterior tibial and ulnar nerves. They did not change during the procedure (not shown). For motor-evoked potentials, multiple high-voltage electrical stimulations trains were delivered (250–450 V, pulse width 50 μ s, number of trains five, and interpulse interval 2 ms)

Fig. 3 Lateral radiograph showing C6 corpectomy and C5–C7 instrumented fusion with a fibular allograft

the deficit in response to position changes. The patient's spinal cord blood flow was at a critical threshold keeping different spinal cord volumes in a penumbra state. Surgical decompression, which was followed by a complete and fast clinical recovery, prevented a cord infarct.

A prompt decompression through an anterior approach is preferred in cases of BSS secondary to a cervical disc herniation, since it allows removal of the disc herniation without mobilizing the spinal cord (with better recoveries reported than those patients in whom a posterior approach is performed) [3, 4, 6], and minimizes intraoperative blood loss. A more limited approach in this case (without C6 corpectomy) was thought inadequate because the cephalad migration of the herniated disk reached the superior aspect of C6 vertebral body, compressing the cord at that level (Fig. 1).

Surgical treatment, however, could increase cord damage during patient positioning and decompression on an already injured spinal cord (with a variable extent of anterior spinal artery compromise), which is a threat that

spinal surgeons face. Intraoperative neuromonitoring is a valuable tool for optimization of outcome in complex spinal surgery, allowing changes of intraoperative strategies to minimize or reverse deficit [14]. In our case, MOPs not only allowed a safe, complete decompression of the spinal cord with real-time assessment of his neurologic condition, but also prevented preoperative neurological deterioration, identifying and recording a positioning-related injury. Previous reports have not documented any role of spinal cord monitoring in these cases.

Conclusion

Cervical disc herniation associated with spinal stenosis represents a very unusual cause of BSS. A prompt diagnosis by MRI, followed by spinal cord decompression using an anterior approach should be warranted. Intraoperative neuromonitoring is a useful tool in preservation of neurologic function in these cases, mainly since it can mandate a change in surgical strategy.

Conflict of interest None of the authors has any potential conflict of interest.

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