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Brown-Séquard Syndrome of the Cervical Spinal Cord after Chiropractic Manipulation

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Summary: We report a case of increased signal in the left hemicord at the C4 level on T2-weighted MR images after chiropractic manipulation, consistent with contusion. The patient displayed clinical features of Brown-Séquard syndrome, which stabilized with immobilization and steroids. Follow-up imaging showed decreased cord swelling with persistent increased signal. After physical therapy, the patient regained strength on the left side, with residual decreased sensation to pain involving the right arm.

Brown-Séquard syndrome results from cord hemisection or damage, most frequently caused by penetrating trauma; injuries caused by blunt trauma are much less common. Reports of complications from chiropractic manipulation of the cervical spine have included vertebral artery dissection, disk herniation, and fracture. We report a case of Brown-Séquard syndrome after chiropractic treatment, and discuss the imaging findings and the relationship between the chiropractic technique and the possible mechanism of injury.

Case Report

A 58-year-old right-handed woman went to her chiropractor because of midthoracic and posterior cervical pain, which she reported to be greater on the left side. The pain was sharp in character and did not radiate into her arms. There was no history of trauma or underlying myelopathic process, and no imaging studies had been obtained previously. The patient underwent three sessions of neck manipulations without relief. In each of these sessions, the chiropractor used a "thrust" technique (a high-velocity, low-amplitude thrusting movement of the hand upon the neck). This thrust technique was administered with the patient lying supine, head first in extension then flexion, with rotation both to the left and right. Immediately after her last visit, the patient began to experience new symptoms, consisting of weakness on her left side, left upper extremity spasticity, and a decrease in pain and temperature sensation on her right side extending from her foot up to her arm.

The patient sought medical care from a neurologist, who prescribed an exercise regimen, steroids, and a muscle relaxant. Her symptoms persisted, however, resulting in a visit to the emergency department 3 days later. Neurologic examination at this time revealed a strength of 4+/5 in the left upper extremity, 3/5 in the left lower extremity, and 5/5 in the right upper and lower extremities. Hyperreflexia was present in all muscle tendon groups on the left with increased clonus in the left ankle. Finger-to-nose, heel-to-shin and fine motor movements were intact, but slowed on the left because of weakness. Sensory examination revealed decreased pain and temperature sensation on the right from T4 down, and hyperalgesia with pain to cold temperature on the right side of the trunk. Pinprick sensation was decreased on the right from the C4–C5 level down, and proprioception and vibration were intact throughout. Plain film radiographs of the cervical spine revealed disk space narrowing, anterior and posterior osteophyte formation, and facet degenerative changes involving C4 through C7 (Fig 1A). MR imaging of the cervical spine, performed on an outpatient basis 2 days later, showed an area of increased signal on sagittal and axial T2-weighted images with enlargement involving the left hemicord at the C4 level (Fig 1B and C). No enhancement was present on the contrast-enhanced images, and there was no evidence of ligamentous or soft tissue injury. The radiologic differential diagnosis included nonhemorrhagic contusion, ischemia, a demyelinating process, and neoplasm. Given the patient's history, cord contusion was thought to be the most likely diagnosis.

Although her symptoms persisted, the patient discontinued her prescribed medications, and returned to the emergency department 12 days after the initial MR examination, at which time she was admitted to the hospital. She was placed in a soft cervical collar, and given methylprednisolone 250 mg every 6 hours. This treatment was continued for 5 days without any symptomatic change. At this point, the patient was examined by the physical therapy service, and she was discharged to be followed up with outpatient physical therapy. A follow-up T2-weighted MR imaging study obtained before discharge (17 days after the first MR examination) showed a decrease in hemicord swelling and in extent of abnormally increased signal intensity (Fig 1E–G). After 12 weeks of physical therapy, the patient had regained a significant amount of strength on the left, was ambulating with a cane, and had some residual decreased sensation to pain and temperature involving her right arm.

Discussion

Brown-Séquard syndrome, first described in 1849 (1), results from injury to the anatomic hemicord. This injury causes disruption of the descending lateral corticospinal tracts, the ascending dorsal columns (both of which decussate in the medulla), and the ascending lateral spinothalamic tracts, which cross within one or two levels of the dorsal root entrance (2–5). Hence, the clinical presentation of a patient with this syndrome would include ipsilateral spastic paralysis and loss of position, touch, and vibratory

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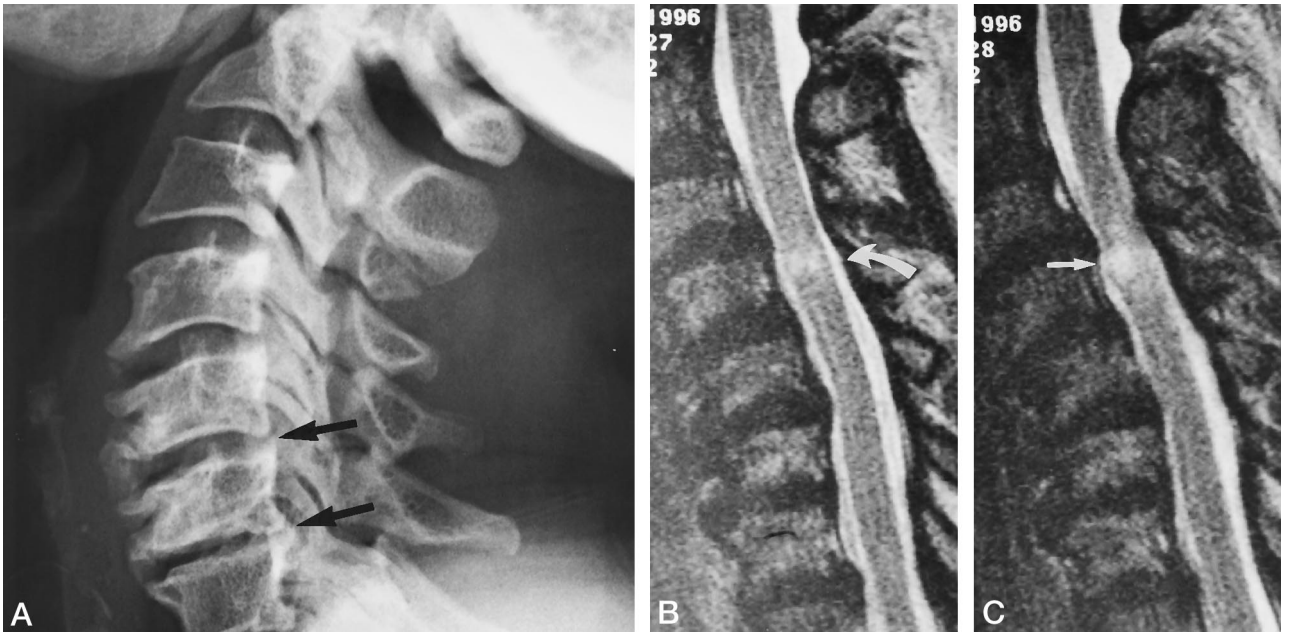


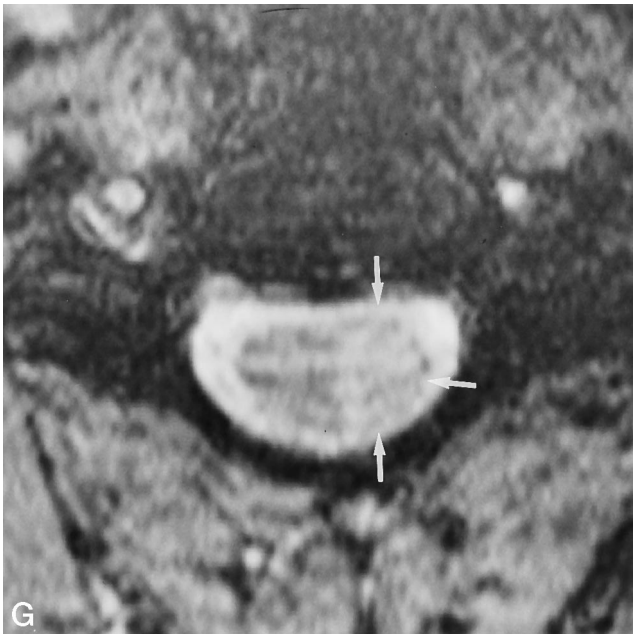


FIG 1. 58-year-old woman with Brown-Séquard syndrome of the cervical spinal cord after chiropractic manipulation.

A, Lateral plain radiograph of the cervical spine shows spondylitic changes at C4–C5, C5–C6, and C6–C7. Note the osteophytes projecting posteriorly at C5–C6 and C6–C7 (*arrows*).

B and *C*, Sagittal T2-weighted (4700/112/3) MR images of the cervical spine (*B*, left of midline; *C*, midline) show a poorly defined area of abnormally increased signal within the spinal cord at the C4 level beginning at the midline (*curved arrow*) and extending to the left hemicord (*straight arrow*).

D, Axial T2-weighted image at the C4 level shows abnormally increased signal with enlargement of the left hemicord (*arrows*).

E and *F*, Follow-up MR study of the cervical spine 17 days after the first examination (after a 5-day course of methylprednisolone). Sagittal T2-weighted images (*E*, left of midline; *F*, midline) continue to show abnormally increased signal (*arrows*), which appears to have decreased in extent but has become more well defined.

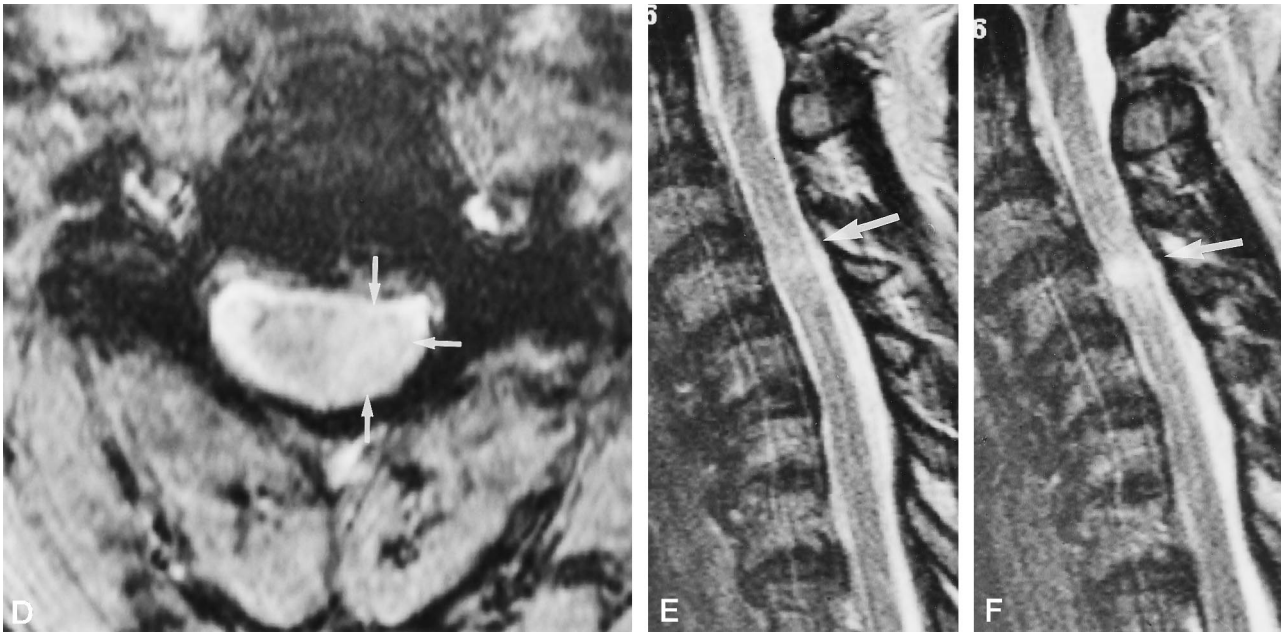
G, Axial T2-weighted image at C4 also shows residual abnormally increased signal, with a decrease in the left hemicord swelling (*arrows*).

sense, with contralateral loss of pain and temperature sensation below the level of the lesion.

The common causes of Brown-Séquard syndrome include penetrating trauma, syringomyelia, hematomyelia, tumor, and blunt trauma, including atypical disk herniation (2). The most common cause by far is penetrating trauma (2, 3). Brown-Séquard syndrome due to blunt injury is much less common, with the largest reported series consisting of only three cases (2). The pathogenesis of cord injury in cases of blunt trauma may include vascular compromise, compression by bone or disk, or longitudinal tension on the cord (2, 3).

The imaging technique of choice to determine both the origin and nature of a Brown-Séquard syndrome is MR imaging (2, 6, 7). MR studies can accurately depict the extent of injury to the spinal cord, the surrounding soft tissues, the overall spinal alignment, and the status of the intervertebral disks (7). Additional important findings may include the presence of a hematoma or other space-occupying process that results in persistent cord compression (2). Plain films and CT scans are useful for detecting bony abnormalities, such as a fracture or osteophytic formation, which may predispose to such an injury.

Patients with a Brown-Séquard syndrome may recover almost full motor function within 6 months of the injury (2–4). The prognosis for recovery is better for patients who have sustained a blunt rather than a penetrating trauma (2). Recent hypotheses regarding the mechanism of recovery include neuronal plasticity mechanisms (denervation supersensitivity), collateral



sprouting (spared axons taking on the functions of the damaged axons), and spared motor axons in the contralateral cord assuming the functions of the damaged axons (4).

Chiropractic manipulation continues to be one of the most popular forms of alternative health therapy in the United States (8). Contraindications to chiropractic therapy should include bony abnormality, acute arthropathy, myelopathy, hypermobility, infection, malignant processes, severe diabetes, and anticoagulation therapy (8). Complications of this form of therapy involving the cervical region have been largely documented in case reports. The frequency of occurrence of complications reported in a recent comprehensive review of the literature is one per million cervical manipulations (9). The authors of this review concluded, however, that this figure is probably too low. A survey of 177 neurologists practicing in California revealed 44 strokes, 16 myelopathies, and 60 radiculopathies resulting from chiropractic manipulation during a 2-year period (8). Again, this may be an underrepresentation, as only a small subset of physicians (neurologists) was surveyed.

The mechanisms for vertebrobasilar injury with resultant stroke after chiropractic manipulation have been well documented in cadaveric studies, with occlusions of the vertebral artery occurring in four of 31 cases with full cervical extension and 50° rotation (10). This number increased to 18 of 31 with the addition of traction to the head equal to half the body weight (10), as could occur with the often-used chiropractic high-velocity thrust technique (11).

To our knowledge, no one has previously reported a case of Brown-Séquard syndrome resulting from chiropractic manipulation. In general, the thrust technique is classified as a mobilizing force (that is, an extrinsic force applied by the chiropractor), which has been a common chiropractic technique for over 100 years (12). Thrust has been defined as a localized force applied with high velocity and low amplitude (12). In treating the cervical spine, the chiropractor places the patient in a supine position, with the chiropractor standing by the patient's head. The chiropractor then places the lateral aspect of the second metacarpophalangeal joint of his index finger against a lateral mass of a cervical vertebra, and extends and rotates the neck above this point to the contralateral side. It is then that a high-velocity, low-amplitude thrust is applied by the hand against the lateral mass in a caudal direction. This is repeated with the head rotated in the opposite direction, and then the whole procedure is repeated with the head in flexion, and so on at each level and side from C2 through C7 (12). We hypothesize that our patient's underlying spondylosis may have predisposed her to blunt injury during this chiropractic manipulation. However, it is noteworthy that the level of cord injury (C4) did not correspond to the level showing the largest posteriorly projecting osteophytes (C5–C6) on plain films. This may be due to the fact that the amount and level of force were unevenly distributed, the greatest magnitude exerted at the C4 level on the left. The combination of extension and flexion coupled with rotation and the thrust technique may have produced enough focused force to cause a blunt injury affecting only one side of the cord, as demonstrated on the MR studies. Our patient's symptoms improved with physical therapy over a period consistent with that reported in the literature (2–4), and an additional MR examination after this point was not performed.

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