Cruciate paralysis: a clinical and radiographic analysis of injuries to the cervicomedullary junction

CURTIS A. DICKMAN, M.D., MARK N. HADLEY, M.D., CONRAD T. E. PAPPAS, M.D., PH.D., VOLKER K. H. SONNTAG, M.D., AND FRED H. GEISLER, M.D., PH.D.

Spinal Cord Injury Service, Division of Neurological Surgery, Barrow Neurological Institute, Phoenix, Arizona; the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems; and University of Maryland, Baltimore, Maryland

Fourteen patients with superior cervical spinal cord injuries and the clinical signs and symptoms of cruciate paralysis are presented. This rare injury pattern is characterized by weakness of the upper extremities with little or no compromise of lower-extremity function following trauma to the superior spinal cord. Anatomically, cruciate paralysis is thought to represent selective injury to descending corticospinal tracts as they decussate at the cervicomedullary junction. The clinical and radiographic findings of each patient are outlined and the incidence and natural history of the injury syndrome, including a review of the literature, are presented.

Key Words • cruciate paralysis • spinal cord injury • spine trauma • cervicomedullary junction • pyramidal decussation

Cruciate paralysis is an uncommon clinical manifestation of neurological compromise at the cervicomedullary junction. It has been reported to occur after traumatic injury to the superior cervical spine. The syndrome of cruciate paralysis is characterized by bilateral upper-extremity paresis or paralysis, with minimal or no lower-extremity involvement. Nine cases of cruciate paralysis have been described in the literature, the majority of which were the result of traumatic injuries.2,3,9,10,21,23-25

The neuroanatomical explanation proposed to account for this syndrome and the associated syndrome of hemiplegia cruciata was outlined in 1901 by Wallenberg.27 He suggested that a complex somatotopic and anatomical segregation of the corticospinal tracts in the decussation at the cervicomedullary junction results in distinct locations for the decussating fibers of the arms and legs. The relative paucity of reports of cruciate paralysis in the literature may be explained by uncertainty about the specific neuroarchitecture of the cervicomedullary junction at the level of the pyramidal decussations and by the fact that injuries to this level typically result in more severe neurological deficits or death rather than isolated bilateral upper-extremity paralysis. Even when suspected clinically, cruciate paralysis may be difficult to differentiate from findings associated with the cervical central-cord syndrome.9,23,24

In this report, we describe 14 patients with documented cruciate paralysis following trauma, and attempt to delineate the clinical and radiographic features of this unique neurological disorder.

Clinical Material and Methods

Fourteen patients with isolated upper cervical spinal cord injuries resulting in cruciate paralysis were identified between 1984 and 1989. Eleven cases were managed at the Barrow Neurological Institute; three other cases were treated at other facilities and included in this study. Only patients with the definitive clinical features of cruciate paralysis were considered in this review. We excluded from the study patients with severe head injury, brachial plexus or peripheral nerve injuries, severe preexisting diseases involving the upper extremities (such as neoplasm or severe arthritis), extensive fractures of the upper extremities, isolated cervical root injury, or deficits from spinal cord injury at another level.

The medical records and radiographic studies of each of the 14 patients were reviewed. Patients were characterized according to sex, age, mechanism of injury, diagnosis, neurological deficits, associated injuries, treatment, radiographic findings, and outcome. Follow-up assessment consisted of reexamining the patients,
Cruciate paralysis

Illustrative Cases

Case 10

This 10-year-old boy was an unrestrained back-seat passenger in a stationary automobile when it was struck in a "rear-end" collision. He was propelled forward and struck his forehead but did not lose consciousness. He experienced the sudden onset of pain in his upper neck, left arm, and trunk, as well as paresthesias.

At examination 1 hour later, he was alert and anxious, and had a left Horner's syndrome, left spinal accessory nerve dysfunction, and a flaccid paralysis of the entire left upper extremity. Left leg strength was 4/5 proximally and 5/5 distally. Motor function of the right arm and leg was normal. Sensory testing revealed hyperpathic pain involving the left C2-7 dermatomes. Pinprick, light touch, two-point discrimination, and proprioception were intact in all dermatomes. The biceps, brachioradialis, and triceps reflexes were absent on the left but were normal on the right. Hyperreflexia with ankle clonus and Babinski responses were present in both lower extremities, more pronounced on the left. Sphincteric function, bulbocavernous, cremasteric, and abdominal reflexes were preserved. Plain x-ray films of the cervical spine and computerized tomography (CT) of the head and cervical spine were unremarkable. The initial diagnosis was spinal cord injury without radiographic abnormality, and the patient was immobilized in a Philadelphia collar. An MR image demonstrated a Chiari I malformation. In addition, a focal contusion of the cord was identified at the cervicomedullary junction/C-1 level, localized to the left anterolateral cervical spinal cord (Fig. 1).

The patient was placed in a skull occiput mandibular immobilization (SOMI) brace. He remained stable clinically and began to recover motor function in the left arm on the 4th day of hospitalization. At the end of the 2nd week postinjury, the sensory and cranial nerve findings had resolved, and antigravity motor function was present in the left upper extremity. Five months after the injury, the patient had a mild spastic weakness (4/5) of the left arm, with hyperreflexia, and a positive Hoffmann's sign in this limb. Follow-up flexion and extension roentgenograms of the cervical spine were without evidence of subluxation.

Case 12

This 62-year-old man lost control of his all-terrain vehicle in the desert. He fell from the vehicle, striking his cranial vertex, but did not lose consciousness. He experienced the sudden onset of quadriplegia and respiratory arrest. He reported remaining fully alert but "trapped inside [his] body, unable to breath or move." Bystanders immediately instituted cardiopulmonary resuscitation. After stabilization at an outlying hospital, he was transferred to our facility. Radiographs, including thin-section CT scans of the cervical spine, demonstrated multiple fractures of the atlas and the axis (Fig. 2). Neurologically, the patient was quadriparetic. Motor examination revealed 2/5 strength in the left upper limb and 4/5 strength in the other three limbs. Sensory examination revealed patchy diminished pinprick sensation and associated burning dyesthesias involving the right C4–T6 dermatomes. Deep-tendon reflexes were hypoactive (1/4) in both upper extremities and brisk (3/4) in the lower extremities. The patient had bladder dysfunction with urinary retention.
After immobilization of the patient in a halo orthosis, MR studies of the head, cervical spine, and cervicomedullary junction were obtained. There was no evidence of neural injury on these studies. A cervical myelogram and postmyelogram CT scan showed no sign of cord compression, hematoma, or intervertebral disc herniation. The patient demonstrated progressive improvement in his motor, sensory, and bladder dysfunction. Within 2 weeks, full strength returned to his lower extremities and his sensory and bladder symptoms resolved. Weakness (4/5) of both upper extremities persisted.

On follow-up examination, 17 weeks postinjury, the patient had mild weakness (4/5) of the left arm but had otherwise completely recovered. No sensory symptoms recurred. Mild hyperreflexia was observed in all four extremities.

Case 14

This 43-year-old man presented after he had been assaulted, during which he was forcefully shaken, beaten, and struck about the head. Upon regaining consciousness, he was unable to move his arms or legs. It was reported that his quadriplegia lasted for 5 to 10 minutes; within an hour he had regained full motor function of his lower extremities. Weakness and clumsiness of both upper extremities persisted for several days.

Examination 4 days after the injury revealed only hyperreflexia in all extremities with a bilateral Hoffmann's sign and equivocal plantar responses. He had no residual weakness, sensory changes, or sphincteric dysfunction. Conventional radiographs and CT demonstrated the presence of an os odontoideum with atlantoaxial instability and a 9-mm horizontal subluxation of C-1 on C-2 from the flexed to the extended position (Fig. 3). Magnetic resonance (MR) imaging displayed a small area of increased signal intensity in the cervical spinal cord at the level of C-1 (Fig. 4). The remainder of the vertebral column and spinal cord was normal, as was a CT scan of the head.

Treatment consisted of an atlantoaxial arthrodesis, which was performed without complication. Six months postinjury, the patient was neurologically intact without hyperreflexia and had a stable C1–2 bone fusion.
Results

The 14 patients with cruciate paralysis reported here represented approximately 2% of all cervical spine injuries and 4% of all cervical spinal cord-injured patients treated during the 5-year study period. There were 12 men and two women, ranging in age from 8 to 72 years (mean age 34 years). The location of traumatic injury common to all patients was the cervicomedullary junction and superior spinal cord. Injuries and underlying pathology associated with cruciate paralysis in the 14 patients are summarized in Table 1.

Fractures

Fractures of the upper cervical spine (C1-3) were found in eight patients (57%). Gunshot injuries, ligamentous atlantoaxial instability, Chiari malformation, and spinal cord injury without radiographic abnormality were also identified. In each of the eight patients with vertebral fractures, the C-2 vertebra was involved (Table 2); four patients suffered isolated axis fractures; among the remaining cases, C-2 was fractured in combination with fractures of the atlas in three and C-3 in one. The types of axis fractures included five miscellaneous C-2 fractures (non-odontoid/non-hangman’s fractures), two odontoid Type II fractures, and one odontoid Type III fracture. The three patients with combination atlas-axis fractures each sustained multiple-ring (burst) fractures of the atlas in addition to the C-2 injury.

In all patients, trauma precipitated the sudden onset of the neurological deficits (Table 3). In addition to the cruciate paralysis, nine patients (71%) sustained minor head injuries, five (36%) had associated thoracic or abdominal injuries, and two (14%) had fractures of the thoracic or lumbar spine without associated neurological injury.

Clinical Presentation

Table 4 summarizes the clinical presentation, treatment, and outcome among the 14 patients with cruciate paralysis. All patients displayed profound upper-extremity weakness with less severe or no lower-extremity involvement. Four of these patients had plegia of one or both arms while the other 10 demonstrated marked paresis of the upper extremities. Upper-extremity plegia/paresis was symmetrical in eight patients (57%), bilateral and asymmetrical in three (21.5%), and unilateral in three (21.5%). Of the three individuals with unilateral arm plegia/paresis, discrepancy between the distribution of the sensory findings and motor deficits

---

**FIG. 3.** Case 14. Left: Three-dimensional computerized tomography reconstruction of the os odontoideum. Center and Right: Significant atlantoaxial instability is present on the lateral plain x-ray films in flexion (center) and extension (right).

**FIG. 4.** Case 14. T2-weighted magnetic resonance image demonstrating a lesion of the cervicomedullary junction and upper cervical spinal cord (large arrow) in association with an os odontoideum (small arrow).
### TABLE 3
Mechanisms of injury associated with cruciate paralysis in 14 patients

<table>
<thead>
<tr>
<th>Mechanism of Injury</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>motor-vehicle accident</td>
<td>6</td>
</tr>
<tr>
<td>all-terrain vehicle</td>
<td>2</td>
</tr>
<tr>
<td>gunshot injuries</td>
<td>2</td>
</tr>
<tr>
<td>fall</td>
<td>1</td>
</tr>
<tr>
<td>bicycle accident</td>
<td>1</td>
</tr>
<tr>
<td>assault</td>
<td>1</td>
</tr>
<tr>
<td>water-skiing accident</td>
<td>1</td>
</tr>
</tbody>
</table>

### TABLE 5
Transient signs and symptoms in 14 patients with cruciate paralysis

<table>
<thead>
<tr>
<th>Signs &amp; Symptoms</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>lower-extremity paresis</td>
<td>5</td>
</tr>
<tr>
<td>sensory deficits</td>
<td>8</td>
</tr>
<tr>
<td>cranial nerve deficits</td>
<td>3</td>
</tr>
<tr>
<td>respiratory insufficiency</td>
<td>4</td>
</tr>
<tr>
<td>urinary dysfunction</td>
<td>3</td>
</tr>
</tbody>
</table>

### TABLE 4
Clinical presentation, treatment, and outcome in 14 patients with cruciate paralysis injuries

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis</th>
<th>Motor Deficits</th>
<th>Sensory Deficits</th>
<th>Respiratory Insufficiency</th>
<th>Cranial Nerve Deficits</th>
<th>Urinary Dysfunction</th>
<th>MR Findings</th>
<th>Treatment</th>
<th>Follow-Up Period</th>
<th>Clinical Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25, M</td>
<td>axis fracture</td>
<td>4/5 2/5</td>
<td>n n</td>
<td>+ n n</td>
<td>n n</td>
<td>ND halo brace</td>
<td>ND ORIF</td>
<td>37 mos</td>
<td>complete recovery</td>
<td>bilat spasticity, upper-extremity weakness (4/5)</td>
</tr>
<tr>
<td>2</td>
<td>72, F</td>
<td>atlas &amp; axis fractures</td>
<td>0/5 0/5</td>
<td>n n</td>
<td>+ n n</td>
<td>n n</td>
<td>ND ORIF</td>
<td>ND ORIF</td>
<td>29 mos</td>
<td></td>
<td>died of aspiration pneumonia</td>
</tr>
<tr>
<td>3</td>
<td>8, M</td>
<td>spastic torticollis, atlantoaxial instability</td>
<td>2/5 2/5</td>
<td>n n</td>
<td>+ lt IX, X, XI, XII</td>
<td>+ ND ORIF</td>
<td>ND supportive measures, Philadelphia collar</td>
<td>ND ORIF</td>
<td>6 mos</td>
<td>complete recovery</td>
<td>bilat upper-extremity spastic paresis (4/5), spastic gait</td>
</tr>
<tr>
<td>4</td>
<td>62, M</td>
<td>gunshot injury of odontoid</td>
<td>0/5 0/5</td>
<td>n n</td>
<td>+ n n</td>
<td>n n</td>
<td>ND halo brace</td>
<td>ND ORIF</td>
<td>58 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>16, M</td>
<td>axis fracture</td>
<td>3/5 2/5</td>
<td>n n</td>
<td>n n n</td>
<td>n n</td>
<td>ND halo brace</td>
<td>ND ORIF</td>
<td>24 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>28, M</td>
<td>gunshot injury of atlas</td>
<td>0/5 0/5</td>
<td>4/5 4/5</td>
<td>n n n</td>
<td>n n</td>
<td>ND ORIF</td>
<td>ND ORIF</td>
<td>23 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>12, M</td>
<td>axis fracture</td>
<td>2/5</td>
<td>n n</td>
<td>+ + n n</td>
<td>n n</td>
<td>nd halo brace</td>
<td>ND halo brace</td>
<td>39 mos</td>
<td>complete recovery</td>
<td>mild upper-extremity spastic paraparesis (5-/5), normal gait, diffuse hyperreflexia</td>
</tr>
<tr>
<td>8</td>
<td>70, M</td>
<td>atlas &amp; axis fracture</td>
<td>2/5 2/5</td>
<td>3/5 3/5</td>
<td>+ n rt XI</td>
<td>+ ND halo brace</td>
<td>ND halo brace</td>
<td>ND ORIF</td>
<td>31 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>19, M</td>
<td>axis fracture</td>
<td>2/5 2/5</td>
<td>4/5 4/5</td>
<td>+ n n n</td>
<td>+ ORIF</td>
<td>ND ORIF</td>
<td>ND ORIF</td>
<td>14 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>10, M</td>
<td>Chiari I</td>
<td>n 0/5</td>
<td>n 4/5</td>
<td>+ n lt XI &amp; Horner’s</td>
<td>n + SOMI brace</td>
<td>ND halo brace</td>
<td>ND ORIF</td>
<td>5 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>26, F</td>
<td>SCIWORA</td>
<td>3/5 3/5</td>
<td>n n</td>
<td>n n n</td>
<td>n SOMI brace</td>
<td>ND halo brace</td>
<td>ND SCIWORA</td>
<td>18 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>62, M</td>
<td>atlas &amp; axis fractures</td>
<td>4/5 2/5</td>
<td>5-/5 4/5</td>
<td>+ + n +</td>
<td>n halo brace</td>
<td>ND ORIF</td>
<td>ND ORIF</td>
<td>4 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>21, M</td>
<td>C-2 &amp; C-3 fractures</td>
<td>n 2/5</td>
<td>n n</td>
<td>n n n</td>
<td>n halo brace</td>
<td>ND ORIF</td>
<td>ND ORIF</td>
<td>8 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>43, M</td>
<td>os odontodeum, atlantoaxial instability</td>
<td>4/5 4/5</td>
<td>n n</td>
<td>+ n n n</td>
<td>+ ORIF</td>
<td>ND ORIF</td>
<td>ND SCIWORA</td>
<td>6 mos</td>
<td>complete recovery</td>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations: MR = magnetic resonance; n = normal function/findings; + = positive/abnormal findings; ND = not done; ORIF = open reduction and internal fixation; SOMI = skull occiput mandibular immobilization; SCIWORA = spinal cord injury without radiographic abnormality. Roman numerals denote cranial nerves.*

---

**J. Neurosurg. / Volume 73 / December, 1990**

---

C. A. Dickman, et al.
excluded injury to the brachial plexus, roots, or peripheral nerves. The distribution of plegia/paresis of the upper extremities initially tended to involve all muscle groups diffusely.

Lower-extremity weakness occurred in five patients, was most often bilateral (four of five patients), and involved the proximal musculature more than the distal groups. When lower-extremity involvement occurred it was mild, transient, and accompanied by long-tract signs including hyperreflexia, Babinski signs, and/or clonus. Three patients demonstrated a delayed deterioration of lower-extremity motor function between 24 and 72 hours after injury.

Sensory symptoms were the most common transient deficits to accompany cruciate paralysis injuries (Table 5). Two patients presented with an “onion-skin” pattern of facial hypalgesia and unilateral truncal and limb hypalgesia (Fig. 5). The other sensory findings included hyperpathic pain and diffuse patchy hypesthesia. Other transient neurological deficits included brief episodes of respiratory insufficiency in four patients, urinary retention in three, and transient cranial nerve dysfunction in three.

Radiographic Studies

Diagnostic radiographic studies included plain cervical radiographs (anteroposterior, lateral, and openmouth odontoid views) and CT scans that demonstrated fractures and/or subluxation in 12 (86%) of the 14 patients. The CT studies provided additional information regarding the details of the fractures and vertebral alignment compared with plain radiographic studies. In the two patients with normal conventional x-ray films, CT studies helped to exclude the presence of fractures.

In seven patients, MR studies of the craniovertebral junction, cervical spine, and spinal cord were obtained using a Signa GE 1.5-tesla MR unit. These studies were performed within 48 hours of injury in six patients and within 5 days of injury in one patient. In three patients, the MR images demonstrated contusions or edema localized to the anterior or anterolateral segments of the cervicomedullary junction and superior cervical spinal cord (Fig. 6); one of the three was identified as having a Chiari I malformation that had not been apparent on plain radiographs or CT studies. Four of the seven patients had normal MR studies.

Treatment and Outcome

The treatment of the 14 patients included immobilization, supportive therapy, and (in unstable injuries) surgical arthrodesis of the cervical spine. Six of the
eight patients with cervical spine fractures were treated with halo immobilization. Patients without fractures were treated with less rigid forms of external cervical immobilization: a Philadelphia collar in one case and a SOMI brace in two cases.

Surgical therapy was reserved for patients with ligamentous instability of the atlantoaxial complex (three cases) or unstable fractures (two cases), and consisted of posterior atlantoaxial arthrodesis in four patients or occipitocervical fusion in one. Of the two patients with fractures who were treated with early surgical therapy, one had a combination atlas-axis fracture with 8-mm posterior displacement of an odontoid Type II fracture and the other had an isolated Type II odontoid fracture of the axis with 9-mm posterior displacement of the dens.

Follow-up data were obtained for all 14 patients (mean 21.6 months, range 4 to 58 months). All patients had progressive improvement in neurological function, and eight patients recovered completely. The five patients with incomplete recovery had mild residual spastic weakness involving one or both upper extremities; only one patient (Case 4) has residual gait spasticity. All nine patients treated with external immobilization demonstrated successful union without instability. Of the five surgical patients, one (Case 3) died 6 months postoperatively from pneumonia; the remaining four showed evidence of solid fusion without instability.

**Discussion**

**Historical Perspective**

Cruciate paralysis, an unusual clinical entity, is "frequently undiagnosed or misunderstood," and can simulate the acute cervical central-cord injury syndrome. Nielsen first reported cruciate paralysis in two individuals: one with a tuberculoma of the odontoid process and the other with an odontoid fracture associated with posterior angulation. Bell reported three patients with cruciate paralysis caused by an odontoid fracture, a Chiari malformation with syringomyelia and basilar invagination from the dens, and an acute hydrocephalic crisis following posterior fossa surgery for tumor. Subsequently, Schneider, et al., Marano, et al., Dumitru and Lang, and Erlich, et al., described cases of cruciate paralysis due to traumatic atlantoaxial instability, a gunshot injury to the odontoid, a deceleration motor-vehicle injury without radiographic abnormalities, and an atlas fracture in a child with a Chiari I malformation. The case reported by Marano, et al., is included in the present clinical series. An analysis of the physical findings, clinical course, and outcome among the previously reported cases reveals similarities to the present series.

**Cruciate Paralysis**

Cruciate paralysis is characterized by isolated injury to the cervicomedullary junction resulting in upper-extremity paralysis with minimal or absent lower-extremity involvement. A spectrum of severity and patterns of involvement exists. Most commonly, the upper-extremity deficits are bilateral and symmetrical, but it is not unusual for the plegia/paresis to be asymmetrical or even unilateral (that is, monoparesis or hemiparesis) as identified in six patients in this series. The patterns of injury, clinical findings, and recovery support the concept that this injury involves selective damage to the corticospinal tracts or upper motor neurons subserving upper-limb function within the decussation. Initially, these patients present with a diffuse flaccid paralysis of the arms, with proximal function generally recovering before distal arm function. As recovery progresses, long-tract signs typically appear in the involved limb(s), including hyperactive reflexes, Hoffmann's signs, and hypertonus, indicative of upper motor neuron injury. When lower-extremity involvement occurred, it was transient, worse proximally than distally, and associated with long-tract signs.

The transient neurological deficits that accompany cruciate paralysis also support localization of the injury level to the cervicomedullary junction and upper cervical spinal cord. Transient respiratory insufficiency, urinary retention, hyperreflexia, lower cranial nerve palsies, and sensory deficits occur in up to 70% of cases. The cranial nerve most commonly involved is the spinal accessory nerve. Infrequently, hypoglossal, vagal, and glossopharyngeal dysfunction can complicate the clinical picture.

Sensory deficits in most patients have consisted of vague diffuse, patchy regions of hypesthesia, not confined to a discrete dermatomal distribution. Dysesthesias and hyperpathic pain are common, occurring in 30% of cases. Two patients displayed evidence of a trigeminal sensory onion-skin pattern (Fig. 5) resulting from involvement of the spinal tract of the fifth cranial nerve. These findings are also supportive of an injury localized to the upper cervical spinal cord. The pattern of sensory involvement is critical in the evaluation of patients with cruciate paralysis injuries and must be used to rule out possible root, brachial plexus, peripheral nerve, and acute cervical central cord injuries.

**Central Cord Syndrome**

The syndrome of acute central cervical cord injury may simulate the sensory and motor findings seen in cruciate paralysis. There are important differences between the two syndromes. With a central cord syndrome, the injury is localized to the middle or lower segments of the cervical spinal cord and involves damage to the anterior horns and lower motor neurons subserving arm function in the anterior horns and the lateral corticospinal tract. In a central cord injury the medial aspect of the lateral corticospinal tract, consisting of axons involved with upper extremity function, is usually injured more than the lateral aspect of the lateral corticospinal tract. This contrasts with cruciate paralysis which involves injury to the cervicomedullary junction and leads to selective upper motor
Cruciate paralysis

neuron dysfunction due to damage of the corticospinal decussation. The absence of cranial nerve dysfunction, trigeminal sensory deficits, and plain radiographic, CT, and MR findings may help to differentiate central cord injuries from cruciate paralysis injuries. However, based on these results, it would be impossible to differentiate lesions involving upper cervical levels with injury only to the lateral corticospinal tract and anterior horns from injuries involving only the corticospinal decussation. Both types of lesions have the same clinical presentation, including flaccidity and greater upper-extremity dysfunction than lower-extremity dysfunction.

Magnetic Resonance Findings and Correlations

Previous reports postulate that lesions causing cruciate paralysis must be superficial and minimal because extensive injuries to the lower medulla or superior spinal cord would produce massive neurological deficits and death.3,9,21 Magnetic resonance studies indicate that these injuries represent a spectrum of severity. The three patients in our series with positive MR findings had abnormal signal intensity changes indicating contusions and/or edema confined to the anterior or anterolateral portion of the cervicomedullary junction and superior cervical spinal cord. Four individuals had normal MR studies. All seven MR examinations support the concept that the spinal cord injury of cruciate paralysis must be mild to moderate, so as to damage motor function yet preserve life.

Magnetic resonance imaging is the radiographic modality of choice for patients with cruciate paralysis. It allows examination of the intracranial compartment, the cervicomedullary junction, the vertebral column, and the spinal cord, and it depicts congenital anomalies and neural element injuries associated with high cervical spine injuries. Among all reported cases and the cases in our series, the injuries of cruciate paralysis were confined to the superior cervical spinal cord and cervicomedullary junction. Only two of these 22 total cases were without abnormalities on plain radiographic studies. The majority of patients with cruciate paralysis have fractures of the upper cervical spine or ligamentous atlantoaxial instability. Plain radiographic studies and thin-section CT scans followed by dynamic flexion-extension cervical radiographs (when indicated) are the diagnostic methods of choice for assessing the extent and relationships of bone and/or ligamentous injuries.4,5,8,12,15,18,26

Fractures

All eight patients presenting with cruciate paralysis and fractures of the cervical spine had fractures of the axis. Half of the fractures involved multiple vertebrae; C-2 was fractured in combination with the atlas or C-3 in 50% of cases. Neurological injuries are infrequent with isolated C-2 fractures but increase when fractures involving both C-1 and C-2 occur.8,11,13,14,17,20,26 The higher rate of neurological injury associated with multiple-level fractures probably reflects the greater force necessary to injure multiple contiguous vertebral segments.

Neuroanatomical Mechanism

The neuroanatomical theory that has been used to explain cruciate paralysis injuries2,3,7,9,16,21,24,25 was outlined by Wallenberg27 in 1901. He described a case of hemiplegia cruciata with crossed arm and leg paralysis and proposed an anatomical and somatotopic segregation of the corticospinal tracts and decussations of the motor fibers to the upper and lower extremities.5,16,21,27 A lesion of the lateral aspect of the pyramidal decussation, he argued, would involve uncrossed fibers to the lower extremity and crossed fibers to the upper extremity, thereby resulting in paresis of the ipsilateral arm and contralateral leg. He suggested that the decussation of the fibers to the upper limbs lay in a more rostral, medial, and ventral location in the superior spinal cord-cervicomedullary junction compared to a more caudal and lateral location of the decussating fibers to the lower limbs. This explanation has subsequently been employed by several authors to account for cruciate paralysis injuries in their patients.2,3,7,9,16,21,24,25,27 Neuropathological and neuroanatomical evidence to support the hypothesis of tract and decussation segregation has not been found in humans or animals.6,19,27 Using Marchi degeneration studies, Coxe and Landau6 found a diffuse distribution of corticospinal tract fibers from the upper and lower extremities at the pyramidal decussations in the Cynomolgus monkey. Using anterograde HRP-WGA tracing techniques, Pappas, et al., examined the upper-extremity and lower-extremity corticospinal tracts in the squirrel and rhesus monkey (CTE Pappas, personal communication, 1989). They identified a diffuse distribution of corticospinal fibers to the upper and lower extremities without evidence of anatomical segregation. Interestingly, these investigators demonstrated that the corticospinal tracts have synaptic connections at the cervicomedullary junction. The synapses serving the upper and lower extremities are spatially separate in this region. They proposed that these synaptic connections may account for the selective vulnerability to injury.

Prognosis

The prognosis for individuals with cruciate paralysis is good. Most patients with this injury demonstrate complete recovery, and residual deficits are usually mild. Excluding the one death, all patients in our series now function independently.

Respiratory insufficiency occurs in approximately 25% of cases and is frequently mild. Four patients in our series required temporary ventilatory assistance and none required tracheostomy. Improvements in emergency medical care, rapid patient transport, early spinal stabilization, and an increased awareness of these injuries may account for the increased recognition of this syndrome. In the past, many patients with cruciate paralysis and profound respiratory insufficiency may
the cruciate paralysis injury syndrome, several important clinical features about the entity are clear. Most reported cases are the result of trauma to the superior cervical spine (typically of C-2). Computed tomography and MR imaging are essential in assessing spine and spinal cord pathology among affected patients. In general, the prognosis for recovery after cruciate paralysis injury is good.

Conclusions

Despite the lack of knowledge about the exact neuroanatomical substrate which, when injured, defines the cruciate paralysis injury syndrome, several important clinical features about the entity are clear. Most reported cases are the result of trauma to the superior cervical spine (typically of C-2). Computed tomography and MR imaging are essential in assessing spine and spinal cord pathology among affected patients. In general, the prognosis for recovery after cruciate paralysis injury is good.

Acknowledgments

The authors wish to acknowledge Stephen Papadopoulos, M.D., Stephen Ondra, M.D., and Sharon L. Bradshaw, R.N., for their assistance in the preparation of this project.

References