# Revisiting cruciate paralysis: A case report and systematic review

#### ABSTRACT

**Objective:** Cruciate paralysis is a rare, poorly understood condition of the upper craniovertebral junction that allows for selective paralysis of the upper extremities while sparing the lower extremities. Reported cases are few and best treatment practices remain up for debate. The purpose of this study was to conduct a systemic literature review in an attempt to identify prognostic predictors and outcome trends associated with cases previously reported in the literature.

**Materials and Methods:** We conducted a systematic literature review for all cases using the term "Cruciate Paralysis," reviewing a total of 37 reported cases. All outcomes were assigned a numerical value based on examination at the last follow-up. These numerical values were further analyzed and tested for statistical significance.

**Results:** Of the 37 cases, 78.4% were of traumatic causes. Of these, there were considerably worse outcomes associated with patients over the age of 65 years (P < 0.001). Those patients undergoing surgical treatment showed potentially worse outcomes, with a P value approaching significance at P = 0.08.

**Conclusion:** Numerous cases of trauma-associated cruciate paralysis have been reported in the literature; however, there remains a strong need for further study of the condition. While certain risk factors can be elicited from currently reported studies, insignificant data exist to make any sound conclusion concerning whether surgical intervention is always the best method of treatment.

Key words: Central cord syndrome; craniovertebral junction trauma; cruciate paralysis; paralysis.

#### Introduction

Termed first by Bell in 1970, "Cruciate Paralysis" is a rare neurological disease of the cervicomedullary junction.<sup>[1]</sup> Cruciate paralysis often presents with bilateral paresis of the upper extremities while sparing the lower extremities.<sup>[2]</sup> Patients may also present with difficulty breathing, cranial nerve deficits, or a comatose state.<sup>[3-5]</sup> While trauma is the most common cause of cruciate paralysis, the exact mechanism for these symptoms is not entirely understood.<sup>[3,6]</sup> The leading hypothesis involves disruption of the anatomy of the pyramidal decussation at the cervicomedullary junction.<sup>[3]</sup> The anatomical decussation extends longitudinally, spanning from the cervicomedullary junction to the C-2 level. Within this region, the motor tract fibers of the upper extremities

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cross both ventrally and superiorly to the fibers supplying the lower extremities. By crossing at a spatially different location, the independent upper extremity fibers provide a way for lesions to preferentially damage upper extremity fibers while sparing those of the lower extremities.<sup>[3]</sup> However, cruciate

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paralysis is a rare condition with few reported studies; hence, treatments have been variable and are often without supportive evidence.

# **Materials and Methods**

In this report, we conducted a systematic literature review from 1966 to the present of patients diagnosed with cruciate paralysis to identify potential prognostic predictors for the outcome. Using MEDLINE and PubMed Central, a comprehensive search for all papers under MeSH and keyword term "Cruciate Paralysis" was performed. Additional information and cases were obtained through Google and Google Scholar, and appropriate search of relevant sources was performed using the same keywords. A case was included if it met the following criteria: (1) The paper under review demonstrated appropriate signs and symptoms of cruciate paralysis as defined above; (2) a mechanism of injury was noted; (3) the type of intervention and treatment was noted; and (4) a follow-up examination was documented. Cases with patients presenting in a comatose state were excluded along with papers written in languages other than English. Our study focused on patients who were noncomatose and carried the diagnosis of cruciate paralysis. This is due to the fact that different states of coma may affect appropriate examination of the upper and lower extremities and hence the diagnosis. We were able to identify 38 cases from the literature. One additional case treated at our institution was also added. Of the 39 cases initially found, 37 of them met our criteria [Table 1]. Follow-up results were classified into three categories of recovery: Insignificant recovery, moderate recovery, or full recovery. A case was considered to have made a full recovery if, at the time of the last documented follow-up, upper extremity neurologic deficits had completely resolved. A case was considered to have made a moderate recovery if, at the time of the last follow-up, symptomatic improvement was documented, but residual upper extremity neurologic deficits still remained. Finally, a case was considered to have made an insignificant recovery if, at the time of the last follow-up, there was little to no change in upper extremity neurologic deficits since the time of initial presentation. Each category was assigned a numerical score of 1, 2, or 3, respectively, for simplicity of analysis. Treatments were further characterized into two groups: Those who underwent surgical intervention and those who did not. Each of the 36 cases was analyzed for outcome trends based on cause (trauma or nontrauma related), and an appropriate ANOVA test was run using the mean numerical scores of each category [Table 2]. The 28 cases associated with trauma were further analyzed, and respective ANOVA tests were run to determine trends and associations of outcomes categorized by age, gender, and type of intervention [Table 3].

#### Results

In patients who carried the diagnosis of cruciate paralysis and who were not comatose, the overall reported outcome was favorable with 54% of patients achieving full recovery and 29.7% of patients achieving moderate recovery.

The overall outcomes associated with cruciate paralysis secondary to trauma did not differ significantly from other nontraumatic causes, P = 0.5 [Table 2]. Since the majority of cases of cruciate paralysis were traumatic (29 patients, 78.4%), we analyzed factors that might impact outcomes of traumatic cruciate paralysis [Table 3]. Patients over the age of 60 years showed significantly worse outcomes as compared to those under the age of 60, P < 0.001. Similarly, patients in the both 0-20 and the 20-40 age ranges had statistically better outcomes when compared to the rest of the cohort, P = 0.02 and P = 0.02, respectively. Male patients also seemed to have slightly better outcomes on average than female patients, P = 0.08. Finally, patients treated without surgical intervention had better prognoses than those treated surgically but did not reach statistical significance, P = 0.08 [Table 3]. We included the details of the patient with traumatic cruciate paralysis that was treated at our institution in Figures 1-3.

## **Discussion**

Cruciate paralysis is a syndrome that results from cervicomedullary compression. It resembles central cord



Figure 1: A 59-year-old woman suffered a motor vehicle accident. She was intubated at the scene. Her neurological examination showed a motor strength of 1/5 in the upper extremities and 3/5 in the lower extremities. Sagittal T2-weighted sequence magnetic resonance imaging of the cervical spine demonstrating a Type III odontoid fracture with posterior subluxation causing compression of the cervicomedullary junction with upper cervical spine signal cord change

# Table 1: Clinical studies investigating the management of cruciate paralysis

Study	Age	Sex	Type of injury	Intervention	Initial exam presentation	Examination at the last follow-up	Duration of follow-up
Bell, 1970 <sup>[1]</sup>	53	Female	Large meningioma on the right side of posterior fossa	Suboccipital craniectomy and upper cervical laminectomy	Marked ataxia of all four extremities, unable to walk because of marked loss of balance, no weakness	Leg strength normal, 20% weakness in the left arm and 50% weakness in the right arm. Moderate improvement of ataxia	2 months
	72	Female	Fractured odontoid process completely dislocated posteriorly on C2 bringing C1 with it	Crutchfield tongs and maintained traction for 51 days	Unable to move arms voluntarily with only minimal flexion of biceps in response to painful stimuli. Movement in both legs (30% weakness of the left and 60% weakness of the right). Sensation is normal throughout except for patchy hypoesthesia in arms. Normal deep tendon reflexes in arms but hypoactive in legs. No pathological reflexes could be found	Normal gait, minimal widening of the base. 25%-30% weakness of the right biceps, triceps, deltoid muscles, and left biceps. 15° limitation of motion of the right shoulder. Brisk deep tendon reflex in upper extremities, hypoactive in lower extremities. Bilateral Hoffmann responses. Normal sensation	6 months
Bruni <i>et al</i> ., 1994 <sup>[7]</sup>	54	Male	Jefferson's fracture	SOMI brace and Philadelphia collar	No upper extremity movement	Complete recovery	Not mentioned
Current study	59	Female	Odontoid Type III fracture	Crown halo vest	Motor strength 1/5 in upper extremities and 3/5 in bilateral lower extremities, dysphagia, and dysarthria	Moderate recovery	6 months
Dai <i>et al.,</i> 1995 <sup>®]</sup>	26	Male	Odontoid fracture	Occipital traction and plaster cast	Upper limbs right: 2/5, left: 2/5. No lower limb deficiencies, sensory deficits, or cranial nerve deficits	Complete recovery	9 years
	26	Male	Atlantoaxial subluxation	Occipital traction and plaster cast	Upper limbs right: 4/5, left: 3/5, no lower limb deficiencies or cranial nerve deficits. Sensory deficits were present	Complete recovery	1 week
	32	Male	Odontoid fracture	Occipital traction and plaster cast	Upper limbs right: 4/5, left: 4/5, no lower limb deficiencies or cranial nerve deficits. Sensory deficits were present	Complete recovery	6 years
	38	Male	Odontoid fracture	Occipital traction and plaster cast	Upper limbs right: 3/5, left: 3/5; no lower limb deficiencies, sensory deficits were present, cranial nerve deficit (XI)	Complete recovery	8 years
	51	Male	Atlantoaxial dislocation, compression fracture of C6	Open reduction and occipitocervical fusion	Upper limbs right: 3/5, left: 2/5; Lower limbs right: 4/5, left: 4/5; no sensory deficits, no cranial nerve deficits	Upper limb weakness (4/5)	10 years
Dickman <i>et al.</i> , 1990 <sup>[3]</sup>	8	Male	Atlantoaxial instability, spastic torticollis	Open reduction and internal fixation	Upper extremity weakness (right: 2/5, left: 2/5), Respiratory insufficiency, Cranial nerve IX, X, XI, XII deficits, urinary dysfunction	Died of aspiration pneumonia	6 months
	10	Male	Chiari I malformation, focal contusion of the cord at the cervicomedullary junction/C1 level, localized to left anterolateral cervical spinal cord	Philadelphia collar, SOMI brace	Flaccid paralysis of upper left extremity, the left leg strength 4/5 proximally and 5/5 distally. The right arm and leg normal motor function. Absent bicep, brachioradialis, and tricep reflexes on the left. Babinski responses present in both lower extremities and hyperreflexia with ankle clonus (more pronounced on the left)	Mild spastic weakness (4/5) in the left arm with hyperreflexia and positive Hoffman's sign in this limb. Follow-up flexion and extension roentgenograms of cervical spine were normal	5 months

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## Table 1: Contd...

Study	Age	Sex	Type of injury	Intervention	Initial exam presentation	Examination at the last follow-up	Duration of follow-up
	12	Male	Axis fracture	Halo brace	Upper extremity weakness (right: 2/5), sensory deficits, respiratory insufficiency	Complete recovery	39 months
	16	Male	Axis fracture	Halo brace	Upper extremity weakness (right: 3/5, left: 2/5)	Complete recovery	24 months
	19	Male	Axis fracture	Open reduction and internal fixation	Upper extremity weakness (right: 2/5, left: 2/5), lower extremity weakness (right: 4/5, left: 4/5), sensory deficits	Complete recovery	14 months
	21	Male	C2 and C3 fractures	Halo brace	Upper extremity weakness (left: 2/5)	Complete recovery	8 months
	25	Male	Axis fracture	Halo brace	Upper extremity weakness (right: 4/5, 2/5), sensory deficits	Complete recovery	37 months
	26	Female	Spinal cord injury without radiographic abnormality	Skull occipital mandibular immobilization brace	Upper extremity weakness (right: 3/5, left: 3/5)	Complete recovery	18 months
	28	Male	Gunshot injury of atlas	Open reduction and internal fixation	Upper extremity weakness (right: 0/5, left: 0/5), lower extremity weakness (4/5 in both extremities)	Complete recovery	23 months
	43	Male	Odontoideum with atlantoaxial instability and a 9 mm horizontal subluxation of C1 on C2 from the flexed to the extended position	Atlantoaxial arthrodesis	Hyperreflexia in all extremities with bilateral Hoffmann's sign and equivocal plantar responses. Several days of upper extremity weakness and clumsiness	Neurologically intact without hyperreflexia. Patient had stable C1–C2 bone fusion	6 months
	62	Male	Fracture of odontoid process and posterior ring of C1, burst fracture of the atlas and fracture through the base of the dens	Halo orthosis immobilization	2/5 strength in left upper limb, 4/5 strength in other three limbs. Patchy-diminished pinprick sensation and associated burning dysesthesias involving C4–C6 dermatomes. Deep tendon reflexes hypoactive (1/4 in upper extremities, 3/4 in lower extremities). Bladder dysfunction with urinary retention	Mild weakness (4/5) of the left arm, but otherwise completely recovered. No sensory symptoms. Mild hyperreflexia in all four extremities	17 weeks
	62	Male	Gunshot injury of odontoid	Philadelphia collar	Upper extremity weakness (right: 0/5, left: 0/5), respiratory insufficiency	Bilateral upper extremity spastic paresis (4/5), spastic gait	58 months
	70	Male	Atlas and axis fractures	Halo brace	Upper extremity weakness (right: 2/5, left: 2/5), lower extremity weakness (right: 3/5, left: 3/5). Sensory deficits. Cranial right XI nerve deficit. Urinary dysfunction	Mild upper extremity spastic paraparesis (5—/5), normal gait, diffuse hyperreflexia	31 months
	72	Female	Atlas and axis fractures	Open reduction and internal fixation	Upper extremity weakness (right: 0/5, left: 0/5) sensory deficits	Bilateral spasticity, upper extremity weakness (4/5)	29 months
Dumitru and Lang 1986 <sup>[6]</sup>	39	Male	Small hypodense fluid collection in the right frontoparietal region	Stabilization	Flaccid bilateral upper extremity paralysis. Flexor withdrawal response of lower extremities. Bulbocavernosus, cremasteric, corneal, and abdominal reflexes present. Trace patellar and Achilles reflex noted. No Hoffmann's sign and upon plantar reflex stimulation toes were downgoing. Impairment of IX–XII cranial nerves	Fair grade strength in upper extremities. Positive bilateral Hoffman's signs, but toes still down going with plantar stimulation	5 months

# Table 1: Contd...

Study	Age	Sex	Type of injury	Intervention	Initial exam presentation	Examination at the last follow-up	Duration of follow-up
Erlich <i>et al.</i> , 1989 <sup>(9)</sup>	3	Female	Undisplaced fracture of left lateral mass of C1 and Chiari malformation	None	Normal cranial nerves, flaccid plegia of both upper extremities, lack of reflexes in upper extremities	Great proximal recovery, child remains somewhat weak and clumsy distally. Return of motor stimuli to upper extremities and confirmed Chiari malformation	5 months
Georgiadis and Schulte-Mattler 2002 <sup>(5)</sup>	52	Male	Cortical atrophy without focal lesions	None	Upper extremity weakness (deltoid, triceps, biceps, brachioradialis muscles 3/5, hand flexion/extension, small finger muscles 0/5). More profound weakness on the right side. No sensory deficits	Full strength of both shoulder and arm muscles, mild weakness present in small finger muscles (3/5)	1 year
Gopalakrishnan <i>et al.,</i> 2013 <sup>[10]</sup>	63	Male	Giant fusi-saccular aneurysm	None	Moderate spasticity and weakness predominantly in the right arm and the left leg. Pain and thermal sensation disturbances in the right arm, touch and vibration senses markedly diminished in the lower extremities. Deep tendon reflexes exaggerated in the right arm and the left leg. Pathological reflexes, positive in the right arm	Increased right upper limb weakness, lower limb remained the same	Not mentioned
Inamasu <i>et al.,</i> 2001 <sup>[4]</sup>	85	Male	Type II odontoid fracture with posterior atlantoaxial dislocation	Brooks method to correct instability (C1/ C2 fusion)	Elective paralysis of the arms, only trace movement of the shoulders and arms, movement of both legs was well preserved	Little functional recovery of muscle strength of upper extremity	32 days
Ladouceur <i>et al.,</i> 1991 <sup>[11]</sup>	88	Female	Type III odontoid fracture	Cervical traction and halo vest	Bilateral flaccid paralysis in upper limbs, no weakness of lower limbs identified. No deep tendon reflexes in upper extremities, however, present and normal in lower extremities. Plantar reflexes flexor bilaterally	Drowsiness improved. No change in upper paralysis until she died of respiratory pneumonia 3 months later	3 months
Laubscher <i>et al.,</i> 2012 <sup>[12]</sup>	16	Female	Undisplaced type II odontoid peg fracture	Traction in Cone's calipers for 6 weeks, then a Philadelphia collar for another 6	Weakness in both arms (left weaker than right). The slight decrease in hip flexion on the right side. No sensory deficits. ASIA motor score=74	Weakness improved. ASIA motor score=92	12 weeks
	18	Male	Type III odontoid peg fracture (minimal displacement)	Traction in Cone's calipers for 6 weeks, then a Philadelphia collar for another 6	The weakness of upper limbs (right side weaker than left), Slight weakness in right leg. No sensory deficits. ASIA motor score=76	Neurological deficit improved. ASIA motor score=90	12 weeks
	26	Male	Subarachnoid hemorrhage, an interhemispheric subdural hematoma, and a right zygomatic arch fracture. Chiari 1 malformation and bulbous dens	Philadelphia collar	Decreased consciousness, no movement of upper limbs, movement of lower limbs on command. No obvious sensory deficits. ASIA motor score=68	Full neurologic recovery after 3 months. After 5 days ASIA motor score=92	3 months

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#### Table 1: Contd...

Study	Age	Sex	Type of injury	Intervention	Initial exam presentation	Examination at the last follow-up	Duration of follow-up
Marano <i>et al.</i> , 1986 <sup>[13]</sup>	62	Male	Bullet wound in the C1 arch slightly to the right of midline	None	Complete bilateral upper extremity paralysis, lower extremity strength normal. Reflexes 2/4 in both upper and lower extremities. After 48 h development of lower extremity weakness, hyperactive upper extremity reflex and Babinski's reflexes in lower extremities	Spastic paraparesis in upper extremities and spastic gait, normal strength in all major muscle groups of lower extremities	6 months
Sweet <i>et al.</i> , 2010 <sup>[2]</sup>	39	Male	21 mm of atlanto-occipital dislocation and ligamentous disruption	Instrumented occiput-C4 fusion for stabilization	Bilateral abducens nerve palsies, flaccid paralysis of the bilateral upper extremities, antigravity strength in the bilateral lower extremities. Diffuse hyperreflexia and bilateral Babinski signs present. Lower extremity (3/5) upper extremity (1/5)	Significant bilateral lower extremity improvement (4+/5). Moderate improvement to upper extremities (3/5)	2 months
Yayama <i>et al.,</i> 2006 <sup>[14]</sup>	49	Female	Shifted odontoid process, compressing the spinal cord anteriorly	Suboccipital craniotomy for posterior decompression and posterior fusion (of occiput and C3)	Marked spasticity in right arm/left leg. Unsteady ataxic gait. Muscle weakness in right arm/both legs. Pain and thermal sensation disturbances in the right arm and leg. Touch and vibration sensation diminished in all extremities except left leg. Exaggerated deep tendon in both arms, no pathological reflexes	Ambulatory, very occasional use of a cane and doing well. Sensory recovery was insignificant	7 years after intervention
	67	Female	Displaced spinal cord from foramen magnum to C3 and healed odontoid base fracture	Lateral decompression and bone grafting	Wasted left side of tongue/ deviation of the tongue to the left. Marked spasticity in the left arm and right leg. Pain and thermal sensation deficits more evident on the right arm/right leg. Deficits in touch and vibration in left arm and leg. Exaggerated Deep tendon reflex in the left arm and right leg. Positive pathological reflexes in the left arm and right leg. Reported difficulty in voluntary micturition	No neurological improvement	2.3 years after intervention
	67	Female	Lesion suggestive of synovial hypertrophy	Posterior atlantoaxial fusion	Spasticity in right arm/left leg, muscle weakness in both legs (mainly left side), pain/temperature sensation diminished in left upper and lower extremities. Symmetric decreased sense of touch. Insignificantly positive deep tendon reflexes in the right arm/left leg. Hoffman and Wartenberg reflexes positive. Babinski's and Chaddock's reflexes positive on contralateral side	Bony union successful, patient attained almost full neurologic recovery	6 months

ASIA - American Spinal Injury Association; SOMI - Sternal occipital mandibular immobilizer

syndrome of the subaxial cervical spine, in that it usually affects the upper more than the lower extremities; however, since it is localized to the upper cervical spine, it is also associated with various degrees of lower cranial nerve palsies and at times states of coma.<sup>[15]</sup> Our review demonstrated that most cases are traumatic in nature with 78.4% of the cases reported. Overall, in the absence of coma, the outcome following this injury is favorable with 54% of patients achieving full recovery and 29.7% of patients achieving moderate recovery. Patients who were older than 60 years had a worse outcome than younger patients suffering from traumatic cruciate paralysis. While no concrete treatment recommendations



Figure 2: The patient was placed in crown halo traction and her fracture fragment was reduced as demonstrated by the lateral cervical spine X-ray (a). The patient then was placed in crown halo vest, and a magnetic resonance imaging of the cervical spine was done revealing reduction and realignment with decompression at the cervicomedullary junction as demonstrated with a sagittal T2-weighted sequence (b)

#### Table 2: Percentage of cases making a full recovery, moderate recovery, or insignificant recovery by cause of symptoms

	Full recovery, %	Moderate recovery, %	Insignificant recovery, %	Р
Overall	54.0 (20/37)	29.7 (11/37)	13.5 (5/37)	-
Nontrauma causes	50.0 (4/8)	25.0 (2/8)	25.0 (2/8)	0.50
Trauma causes	55.2 (16/29)	34.4 (10/29)	10.3 (3/29)	

# Table 3: Percentage of trauma cases (29 patients) making a full recovery, moderate recovery, or insignificant recovery by age, gender, and type of correctional intervention

	Full recovery, %	Moderate recovery, %	Insignificant recovery, %	Р
Age				
0–20	100 (6/6)	0 (0/6)	0 (0/6)	0.02*
20-40	88.9 (8/9)	11.1 (1/9)	0 (0/9)	0.02*
40-60	40.0 (2/5)	60.0 (3/5)	0 (0/5)	0.87* 6.8E-07*
60+	0 (0/9)	66.6 (6/9)	33.3 (3/9)	0.0E-07
Male	65.0 (13/20)	30.0 (6/20)	5.0 (1/20)	0.08
Female	33.3 (3/9)	44.4 (4/9)	22.2 (2/9)	
Surgical intervention	33.3 (3/9)	44.9 (4/9)	22.2 (2/9)	0.08
No surgical intervention	65.0 (13/20)	25.0 (5/20)	5.0 (1/20)	

\*P value for given age range tested against all remaining age groups combined, \*P value for combined ANOVA test between all grouped age ranges

have been suggested in the literature, due to the presence of a neurological deficit, severe cruciate paralysis has traditionally warranted surgical intervention.<sup>[3,12]</sup> Our review, however, showed that patients who were treated nonsurgically may have better outcomes with a *P* value of 0.08. This may reflect that patients who warrant surgical intervention may be sicker due



Figure 3: The patient underwent tracheostomy and percutaneous endoscopic gastrostomy tube placement. Her neurological examination continued to improve. Ultimately, the tracheostomy and the percutaneous endoscopic gastrostomy tubes were removed. She was kept in a crown halo vest for 6 weeks, followed by 6 weeks of rigid collar placement. During her 6-month follow-up visit, she was ambulating with a walker. Her motor strength in her proximal upper extremities improved to + 4/5. Her intrinsic hand function was 3/5 and she her speech was dysarthric. Sagittal computed tomography of the cervical spine revealed complete healing of the fracture

to other associated injuries or suffer from a biomechanically unstable fracture requiring surgical intervention. Similarly, Dickman *et al.* recommended surgical intervention only be used for patients having more severe fractures with associated ligamentous instability of the atlantoaxial complex. Recommended procedures include posterior atlantoaxial arthrodesis and occipital-cervical fusion.<sup>[3,4,8,14]</sup>

Our study has a few limitations; the cohort size is small in size since our search focused only on papers that included cruciate paralysis as a keyword and hence some papers that may have included patients with cruciate paralysis secondary to atlantooccipital dissociation and combination atlas and axis fractures were not included. Moreover, patients who were in a comatose state were excluded as well since it would be hard to ascribe coma due to an intracranial or upper cervical spine injury.

#### Conclusion

While numerous cases of trauma-associated cruciate paralysis have been reported in the literature, there remain insignificant data to make any sound conclusion concerning whether or not surgical intervention is always the best method of treatment.

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#### **Conflicts of interest**

There are no conflicts of interest.

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