[Athletic Training]

Rehabilitation Considerations of a Brachial Plexus Injury With Complete Avulsion of C5 and C6 Nerve Roots in a College Football Player: A Case Study

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Severe brachial plexus injuries are rare in sports, but they have catastrophic results with a significant loss of function in the involved upper extremity. Nerve root avulsions must be timely managed with prompt evaluation, accurate diagnosis, and surgical treatment to optimize the potential for a functional outcome. This case report describes the mechanism of injury, diagnostic evolution, surgical management, and rehabilitation of a college football player who sustained a traumatic complete nerve root avulsion of C5 and C6 (upper trunk of the brachial plexus). Diagnostics included clinical evaluation, magnetic resonance imaging, computed tomography myelogram, and electromyogram. Surgical planning included nerve grafting and neurotization (nerve transfer). Rehabilitation goals were to bring the hand to the face (active biceps function), to stabilize the shoulder for abduction and flexion, and to reduce neuropathic pain. Direct current stimulation, bracing, therapeutic exercise, and biofeedback were used to maximize the use of the athlete's upper extremity. Although the athlete could not return to sport or normal function by most standards, his results were satisfactory in that he regained an ability to perform many activities of daily living.

Keywords: nerve root avulsion; preganglionic; brachial plexus; nerve graft

An injury to the brachial plexus is typically referred to as a *burner* or *stinger*, and it is common in sports. The injury is usually classified as a transient neuropraxia, and it is reported to affect as many as 65% of college football players at some time in their career.[5] Symptoms generally include a burning pain that radiates down one of the arms, and it may include temporary weakness, especially if the injury is recurrent. Persistent pain or weakness may indicate a chronic syndrome and require further evaluation and treatment.[3] However, the majority of athletes who experience a burner are able to continue to participate in contact sports without consequence. High-velocity brachial plexus injuries rarely occur in sports, but they have catastrophic impact on function of the entire upper extremity. Appropriate diagnosis, surgical management, rehabilitation, and counseling are important to the overall outcome of the problem.

Severe brachial plexus injuries may include avulsion, stretch, or rupture of a cervical nerve root and so lead to significant disability. These traumatic injuries are increasing with the popularity of extreme sports and with the improvement in survival rates of motor vehicle accidents.[6] Most injuries are closed and often result as a combination of fractures and compression in high-energy collisions.[6] However, injuries to the supraclavicular region of the brachial plexus commonly have a traction-based cause and occur when the head and neck are forced away from the involved shoulder.[6] This mechanism is common in motorcycle accidents. Brachial plexopathies with nerve root avulsion in football are rare, with only 1 other case reported in the literature.[2] That case focused on the EMG findings during the diagnostic period and after the surgical repair, whereas this case will focus on the rehabilitative concerns and injury management.

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No potential conflict of interest declared.

DOI: 10.1177/1941738109343544

CASE PRESENTATION

The present case involves a 19-year-old right-hand-dominant male athlete. As a defensive back (safety) in collegiate football, the patient made an open-field tackle, striking the offensive player by leading with his left shoulder. From the sidelines, the impact appeared to be helmet to helmet. A later review of the game film noted that the offensive player ducked his head on impact, striking the patient in the upper left side of his chest and shoulder, causing his head to be pushed to the side. The patient had a history of 3 or 4 stingers, 2 to 3 years before this incident (ie, in high school), all of which quickly resolved. This athlete was playing as a true freshman and had no stingers during the preseason practices. Baseline cervical spine x-rays were taken as part of his preparticipation physical, the findings of which were negative; furthermore, he had no history of cervical spine injury.

ON-FIELD EVALUATION

The athlete had no loss of consciousness. As the medical staff approached, they observed him lying supine, lifting his head and calling for assistance. He was moving his right arm and both legs in distress. His left arm remained in an abducted position, with no movement. The athlete complained of left upper extremity numbness and claimed that his shoulder felt dislocated. He denied cervical spine pain, and there was no midline cervical spine tenderness to palpation. No motor function was elicited in the left shoulder or bicep, but motor function was rapidly developing in the hand and wrist, with finger movement observed on the field. After ruling out a cervical spine injury, he was able to walk off the field with standby assistance and support for the left upper extremity. The shoulder did not appear to be dislocated, as determined by palpation. The arm hung loosely at the athlete's side, and there was no voluntary control of the upper extremity. The helmet and shoulder pads were removed on the sideline so that the upper extremities could be palpated and visualized. Within 30 minutes, he began to have a severe burning, dysesthetic pain in the left upper extremity, especially in the C5-6 dermatome. The left upper trapezius was tender to palpation, with minimal acromioclavicular joint tenderness and no chest or sternoclavicular joint pain. Radiographs at the stadium were negative for fracture or dislocation of the shoulder, clavicle, or cervical spine. Active range of motion was absent in the left shoulder and biceps, whereas active motion at the hand and wrist improved quickly. Manual muscle testing revealed the following: 5/5, grip, finger extension, abduction, thumb extension, wrist flexion and extension; 4/5, triceps; 1/5, biceps, anterior deltoid, pectoralis major with forward flexion; 0/5, middle and posterior deltoid and rotator cuff musculature.

The athlete was placed in a sling, and full shoulder and cervical spine films were repeated with flexion and extension views the following day. An MRI of the neck and chest was ordered owing to the continued pain, dysesthesia, and weakness. MRI findings of the cervical spine were normal. The chest MRI revealed extensive brachial plexus injury, but the extent of the injury at each nerve root could not be determined. The MRI is considered useful in the diagnosis of a brachial plexus injury because the entire plexus can often be visualized.[6] A CT myelogram was ordered to further evaluate the nerve roots, given that this test has been shown to distinguish injury of the nerve roots, rootlets, and trunk.[1] The myelogram showed left C5 and C6 nerve root sleeve avulsions with no visualization of nerve rootlets at those levels. A meningocele was seen at C6, indicative of a preganglionic injury (Figures 1-3).[1] The oblique coronal image depicted nerve roots exiting the neural foramen below C6 but were absent in C5 and C6 (Figure 3). The athlete was prescribed gabapentin (Neurontin, Pfizer, New York, New York) for alleviation of neuropathic pain, but he discontinued all medications because they made him drowsy.

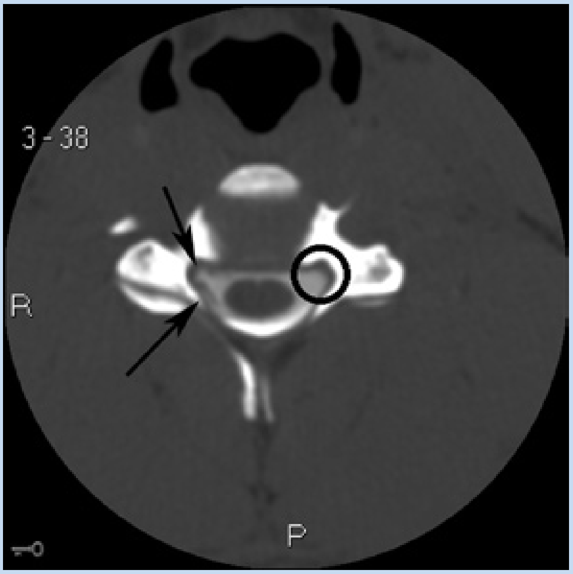

Figure 1. CT myelogram axial view at C5. Nerve roots and rootlets are visualized on the uninvolved side (arrow); no nerve roots are visualized on the involved side (right side of image, circled).

EMG examination was performed at 3 weeks postinjury and included nerve conduction studies. Early electrodiagnostic studies are often unreliable because of the ongoing Wallerian degeneration, which is typically complete at 3 to 4 weeks.[6] There were abnormal sensory responses indicating involvement at or proximal to the dorsal root ganglion in both C5 and C6. There was no evidence of C5-6 upper trunk innervation, but normal function of the rhomboids suggested either (1) the lesion was distal to the takeoff of the dorsal scapular nerve and therefore not a true nerve root avulsion or preganglionic injury at C5 or (2) there was C4 contribution to the dorsal scapular nerve. The EMG study revealed a complete preganglionic lesion at C6. The serratus anterior was also denervated, and there was no functional supination. Table 1 presents a summary of the preoperative EMG findings and physical exam.

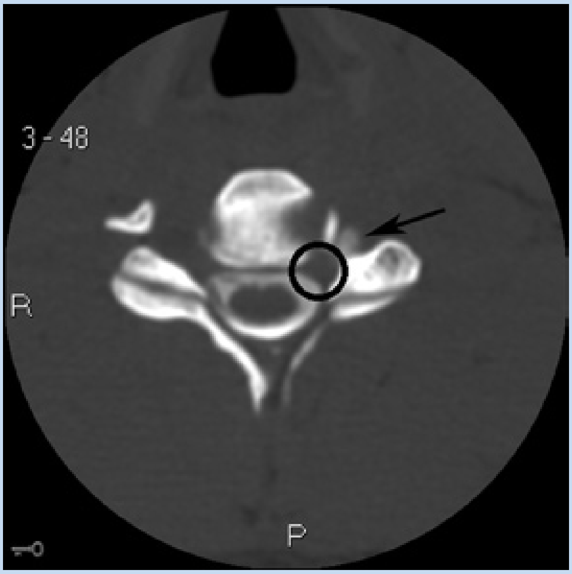

Figure 2. CT myelogram axial view at C6. No nerve root or rootlets are visualized on the injured side (right side of the image, circled); meningocele is shown (arrow).

Figure 3. CT myelogram oblique coronal image. The nerve roots are absent in the neural foramena at C5 and C6 (circled) yet visible in the levels below the lesion (arrows).

Table 1. Summary of Electromyogram and Physical Examination Preoperatively.

Muscle	Nerve Root	Insert Activity[a]	Spontaneous Fibrillation[a]	Spontaneous Fasciculation[a]	Physical Exam[b]
Biceps brachii	C5-7	Increased	++[c]	0	0
Deltoid	C5-6	Increased	+++	0	0
First dorsal interosseus	C7-8, T1	Normal	0	0	5
Flexor pollicus longus	C8, T1	Normal	0	0	5
Infraspinatus	C5-6	Increased	++++	0	0
Pronator teres	C5-8, T1	Increased	+++	0	0
Rhomboid major	C4-5	Normal	0	0	4+
Serrratus anterior	C5-7	Increased	++	0	0
Supraspinatus	C5-6	Increased	+++	0	0
Triceps brachii	C5-8, T1	Increased	+	0	3+
Trapezius	CNX1, C3-4	Normal	0	0	4+

[a]Electromyogram.
[b]Manual muscle test.
[c]No motor unit potential.

Surgical intervention was performed at a nationally recognized brachial plexus treatment center, with the goal of restoring elbow flexion and shoulder abduction for the purpose of activities of daily living. The brachial plexus was explored, and the C5 nerve root was found to the salvageable, although entrapped in scarring consistent with a significant traction injury. Intraoperative EMG included somatosensory-evoked potentials and motor-evoked potentials of both C5 and C6. These intraoperative studies are used to assess whether injuries are pre- or postganglionic, to evaluate the viability of the injured nerve roots, and to assist in surgical planning. There was no viable nerve root at C6 with dissection to the foramen, and stimulation of C5 resulted in no motor function. The diagnosis was consistent with the preoperative physical examination, radiographic, and electrodiagnostic studies. The surgical plan (based on intraoperative findings and monitoring) was to use the C5 nerve root to graft an autologous sural nerve cable to the axillary nerve, with the hope of recovering deltoid and teres minor function. The descending branch of the spinal accessory nerve was transferred to the suprascapular nerve, with the objective of regaining internal and external rotation of the shoulder. The transfer of a functioning nerve fascicle to the distal portion of a nonfunctioning nerve is termed a *nerve transfer* or *neurotization*. Trapezial function is not compromised, because the ascending branch of the spinal accessory nerve is not violated. The Oberlin procedure was used for biceps reanimation.[4] This involves using a fascicle of the flexor carpi ulnaris (carefully separated from other fascicles of the ulnar nerve) to reinnervate the biceps branch of the musculocutaneous nerve, with the goal of restoring elbow flexion. Elbow flexion is the first priority in brachial plexus reconstruction because this allows for placement of the hand in space. Two fascicles of the median nerve were transferred to the brachialis branch of the musculocutaneous nerve to dual innervate for elbow flexion. The brachioradialis was reanimated using a transfer of a fascicle of the radial nerve. All nerve fascicle transfers were done with the assistance of neuromonitoring so that specific fascicles could be isolated before transfer. Fascicles with redundancy in motor function were used. For instance, wrist flexion is provided by flexor carpi ulnaris and flexor carpi radialis; as such, the flexor carpi ulnaris fascicle can be used as a nerve transfer without compromising wrist flexion because the flexor carpi radialis still provides this function. Follow-up evaluations, including EMG and nerve conduction studies, were scheduled every 4 months. The outcome was satisfactory, determined at 2 years postinjury. The patient was able to raise his hand to his face and abduct the shoulder to approximately 60°. He was pain-free but moderately disabled by the limited use of his left upper extremity.

DISCUSSION

In sports medicine, it is important to rule out injury to the spinal cord on the field, for appropriate immobilization and transfer to the sidelines or medical center. Brachial plexus injuries will cause paresthesia, pain, and paralysis to upper extremity. Any indication of a neurological compromise in any other extremity or cervical spine tenderness should dictate spine boarding until further cervical spine injury can be ruled out. Furthermore, fractures to the clavicle, coracoid process, humerus, or upper rib should be included in the initial differential diagnosis to prevent further complications associated with muscle and sensory testing.

Diagnosing whether a brachial plexus injury is a pre- or postganglionic lesion is critical when considering the possibility of spontaneous recovery, given that there is little potential for recovery without surgical reconstruction for preganglionic injuries.[6] Postganglionic lesions have potential for recovery, depending on the severity of the injury. Observation with close serial monitoring of functional return, with physical exams and EMG (including nerve conduction studies), is indicated for many postganglionic lesions. However, preganglionic lesions and nerve root avulsion have no potential for spontaneous recovery; thus, early surgical intervention is warranted to maximize the functional recovery of the extremity. Accurate early diagnosis of the injury, to determine whether it is pre- or postganglionic, is critical in deciding whether early surgical intervention could restore function. The physical examination was consistent with an absence of both C5 and C6 nerve roots, although the rhomboid function caused some speculation about the full avulsion at C5. The electrodiagnostic studies and imaging both indicated avulsions at C5 and C6, with a strong implication of a preganglionic injury at C6; therefore, the decision to operate within a 2-month time frame was made.

The athlete was seen preoperatively almost daily for ice to the supraclavicular area, passive range of motion for the elbow and shoulder (within pain limits), and for light hand and wrist functional tasks. Within several days of the injury, atrophy of the deltoid was already apparent in this well-developed athlete. The left shoulder also began to sublux owing to lack of muscle tone, causing more pain. There was a visual sulcus sign; namely, gravity pulled the humerus inferior without the influence of the supraspinatus. A hemi-sling (Sammons Preston Rolyan, Inc, Bolingbrook, Illinois) was used to prevent the painful subluxation and to support the upper extremity (Figure 4). The sling was effective in pain management, and it acted to support the weight of the arm, with a band around the proximal humerus and a figure-of-8 brace. The medical staff consulted neurosurgical specialists with experience in managing nerve root avulsions. The consensus was to protect the upper extremity and to wait until EMG examination could further delineate the nerve function. Somewhat encouraging was the good function in the triceps, wrist, and hand despite the diagnostic evidence of injury to C7 and below. However, the athlete was prepared to anticipate what might be a realistic outcome; he was prepared for the psychosocial implications of permanent disability.

Postoperative rehabilitation began as the swelling and pain subsided. Therapeutic goals were as follows: achieve 90° of active shoulder flexion and abduction, touch the opposite shoulder (and hand to mouth), and be pain-free. The anticipated recovery time was 1 to 2 years given that nerve regeneration and reinnervation occur at a rate of approximately 1-4 mm per day.[7] The recovery of nervous tissue can occur either by axonal regeneration from proximal to distal or by reinnervation through terminal collateral sprouting. The patient

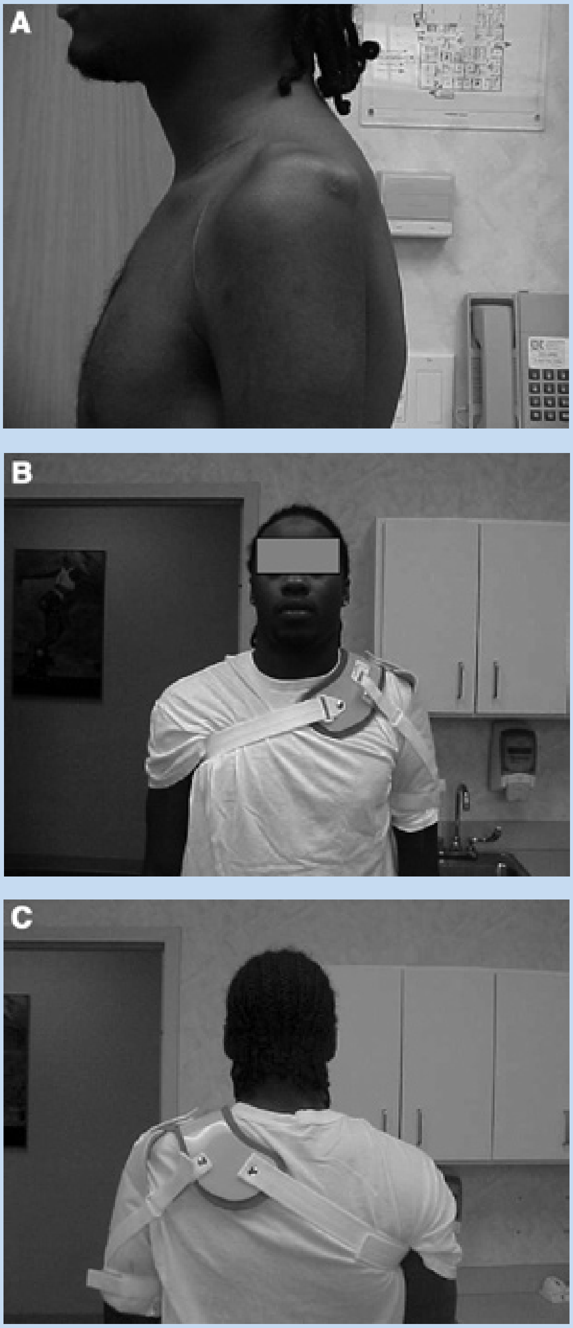

Figure 4. Hemi-sling used to help control shoulder subluxation. The sulcus sign is apparent on the lateral view.

Figure 5. Direct current stimulation of motor points of affected musculature. A twitch response was noted and repeated 8 times at each point.

understood the expectations and followed all postoperative procedures.

The upper extremity was protected against subluxation with the hemi-sling. Therapy was scheduled 3 times per week, and the patient understood that the recovery would be a prolonged process. His condition was comprehensively managed to incorporate physical fitness, wellness, continuation of his academic pursuits, and acceptance of his disability. Short breaks in therapy of less than 1 week were intermittently encouraged because of the length of time that he would need to recover.

Passive range of motion and active-assistive range of motion were used to prevent adhesive capsulitis of the shoulder and elbow. There was an initial limitation in elbow extension. Overstretching was avoided and the stretch reflex was used to facilitate motor function of the biceps. Active range of motion and manual resistance were used at the wrist, hand, and triceps. Desensitization techniques were used in the paresthetic and dysesthetic regions of the upper extremity, and the skin was mobilized to prevent adherence at the incision sites. Light neural mobilizations that included positional release were directed at improving mobility and decreasing pain. A vigorous shower with water pressure directed at the well-healed incision sites was helpful in reducing neuropathic pain.

Interrupted direct current of the motor points was used to stimulate the deltoid, biceps, brachialis, supraspinatus, infraspinatus, and pectoralis muscles (Figure 5); specifically, each motor point was stimulated to elicit a twitch, 8 times at each site, 3 or 4 times per week. This was done to maintain some muscle viability until the time of reinnervation. Galvanic burns appeared at the stimulation sites when the treatment was performed daily. Exercises were modified for gravity elimination, and the use of his hand and wrist was exploited for active-assisted exercises using pulleys. He was able to raise the left hand by grasping it with the right, and he could hold onto a bar for an exercise, such as a lat pulldown. The eccentric component facilitated range of motion at the shoulder while promoting independence with exercise. The patient was also encouraged to use the exercise bike, on which he would grasp the handle with his left hand for modified weightbearing through the upper extremity. Pulleys were also used for triceps, shoulder adduction, and internal rotation. Likewise, cuff weights added to a weighted bar were used for supine shoulder flexion and a modified bench press. The bar enabled him to assist with the right side for stability. Elbow flexion and shoulder abduction required gravity-eliminated positions, sitting or supine, to allow the limb to slide across a table to activate those muscles. There was an emphasis on encouraging activation but not fatiguing these muscles.

At 4 months, the direct current was discontinued, and neuromuscular stimulation was used for the biceps and deltoid. Active contraction was encouraged for muscle reeducation. Duty cycles were set to 15 seconds on and 45 seconds off, for 15 contractions each session. Joint protection was continued with the hemi-sling. An EMG (with nerve conduction studies) was performed at 4 months, and it showed nascent potentials in the bicep and deltoid, indicating early reinnervation but no activity in the suprascapular nerve. There was improvement in elbow flexion to approximately 50° (muscle grade, 2+/5) and in his deltoid with shoulder abduction approximately 10° (trace motor grade). Hypesthesia remained in the thumb but had resolved in the other digits. He was pain-free.

Eight- and 12-month follow-ups showed similar progression of elbow flexion activity. He was able to bring the hand to the face and could perform biceps curls with 7 pounds (3 kg), with assistance at the end range of flexion. He could lift 30 pounds (14 kg) with bench press and could flex the shoulder in a supine position with the same weight. Shoulder abduction against gravity was 60°. Psychosocial considerations remained important given that the asymmetry of the chest and left arm created a disabled appearance that the patient tried to hide by wearing heavy clothing, such as sweat shirts, even during the summer. His outcome was that of moderate disability of the left upper extremity that affected all normal activities. The injury precluded any sports participation, even jogging because of the inability to stabilize the left shoulder. However, there were satisfactory results for most activities of daily living; that is, he could dress himself, eat, and drive independently. He remained unable to perform most bilateral tasks, including carrying large items. The decision to refer to surgery was based on the diagnosis of a preganglionic lesion that would have little chance of spontaneous recovery. Any additional improvement in muscle activity would translate into improved function of the upper extremity.

CONCLUSION

Brachial plexus injuries with this severity require a mechanism of high velocity that is rare in football but common with motor vehicle accidents. Sports medicine professionals should be aware of the clinical evaluation, treatment, modifications in exercises, and expected outcome of these injuries that require nerve grafting or nerve transfer procedures.

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