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Postfixed Brachial Plexus Radiculopathy Due to Thoracic Disc Herniation in a Collegiate Wrestler: A Case Report

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Objective: To present the unique case of a collegiate wrestler with C7 neurologic symptoms due to T1–T2 disc herniation.

Background: A 23-year-old male collegiate wrestler injured his neck in a wrestling tournament match and experienced pain, weakness, and numbness in his left upper extremity. He completed that match and 1 additional match that day with mild symptoms. Evaluation by a certified athletic trainer 6 days postinjury showed radiculopathy in the C7 distribution of his left upper extremity. He was evaluated further by the team physician, a primary care physician, and a neurosurgeon.

Differential Diagnosis: Cervical spine injury, stinger/burner, peripheral nerve injury, spinal cord injury, thoracic outlet syndrome, brachial plexus radiculopathy.

Treatment: The patient initially underwent nonoperative management with ice, heat, massage, electrical stimulation, shortwave diathermy, and nonsteroidal anti-inflammatory drugs without symptom resolution. Cervical spine radiographs were negative for bony pathologic conditions. Magnetic resonance imaging showed evidence of T1–T2 disc herniation. The patient underwent surgery to resolve the symptoms and enable him to participate for the remainder of the wrestling season.

Uniqueness: Whereas brachial plexus radiculopathy commonly is seen in collision sports, a postfixed brachial plexus in which the T2 nerve root has substantial contribution to the innervation of the upper extremity is a rare anatomic variation with which many health care providers are unfamiliar.

Conclusions: The injury sustained by the wrestler appeared to be C7 radiculopathy due to a brachial plexus traction injury. However, it ultimately was diagnosed as radiculopathy due to a T1–T2 thoracic intervertebral disc herniation causing impingement of a postfixed brachial plexus and required surgical intervention. Athletic trainers and physicians need to be aware of the anatomic variations of the brachial plexus when evaluating and caring for patients with suspected brachial plexus radiculopathies.

Key Words: neuropathy, burner, stinger, cervical spine, thoracic spine

Injury to the brachial plexus, or brachial plexopathy, is one of the most common upper extremity injuries in athletics, especially collision sports, such as football, hockey, and wrestling. The incidence in football players has been reported to be as high as 49%,[1] but the true incidence may be higher because the symptoms typically are transient and many patients do not report their symptoms to health care providers. Patients with brachial plexus injuries typically present with unilateral numbness, muscle weakness, and burning or stinging pain radiating down the upper extremity.[2–6] Symptoms typically last only a few minutes but may persist for hours, days, or longer.

In the medical literature, researchers[7–12] commonly describe the brachial plexus as formed by the combination of spinal nerve roots from the C5–T1 spinal cord levels. Whereas the brachial plexus has numerous anatomic variations, 2 of the most common are known as prefixation and postfixation. *Prefixation* occurs when the C4 nerve root has an increased contribution to the plexus and the T1 nerve root has substantially less or no contribution. Conversely, *postfixation* occurs when the T2 nerve root has increased contribution and the C5 nerve root has diminished contribution. Prefixation is much more common than postfixation.[11,13–17]

Intervertebral disc herniation within the brachial plexus is a serious injury because it may impinge on the nerve roots, causing radiculopathy or partial paralysis. Intervertebral disc herniation that impinges on the brachial plexus occurs almost exclusively in the cervical spine. Thoracic intervertebral disc herniations are much less common than cervical and lumbar disc herniations. High-level thoracic disc herniations, which occur from T1–T4, are exceedingly uncommon.[18–26]

We present the case of a 23-year-old male collegiate wrestler who sustained what appeared to be a C7 brachial plexus traction injury but ultimately was diagnosed with a T1–T2 thoracic disc herniation impinging a postfixed brachial plexus that required surgical intervention. Given the rarity of T1–T2 disc herniation, we believe this case illustrates the importance of understanding the common anatomic variations of the brachial plexus to aid in proper diagnosis and appropriate treatment of these conditions, especially when the response to treatment does not progress as anticipated.

CASE REPORT

A 23-year-old male collegiate wrestler was participating in a wrestling tournament when he reported that, while he was shooting in for a takedown, his opponent grabbed his left upper extremity and pulled it down and away from his body. He reported feeling immediate pain in his neck and shooting pain down his left upper extremity with lasting numbness and weakness of the posterior aspect of his extremity. The patient did not seek medical attention and completed the match in which he was injured and 1 additional match with no change in his symptoms. The patient presented to the athletic training department for evaluation 6 days postinjury because his symptoms had not resolved. He reported numbness and tingling in his posterior left upper extremity continuing distally to his fingers, as well as weakness with shoulder and elbow movement. The patient had no substantial previous neck or shoulder injuries but had undergone discectomy to treat a traumatic L5–S1 disc herniation secondary to an axial-load injury 2 years before this injury. Evaluation by a certified athletic trainer (AT) revealed mild tenderness only over the triceps brachii muscle belly. The remainder of the patient's left upper extremity, neck, and cervical spine were not tender. Cervical spine active and passive range of motion, including flexion, extension, lateral flexion bilaterally, and rotation bilaterally, were limited secondary to pain. The patient had full, nonpainful active and passive shoulder and elbow range of motion bilaterally. Manual muscle testing of the biceps brachii and triceps brachii revealed 4/5 and 3/5 strength, respectively, in the left upper extremity when compared bilaterally. All other manual muscle testing was 5/5. Brachial plexus traction testing was positive for pain and reproduction of symptoms. The Spurling test and cervical compression test were negative. No other pertinent findings were present on examination. Initial assessment indicated a brachial plexus neuropathy. The patient was not allowed to participate in practice, and nonoperative therapy was initiated with ice and therapeutic modalities.

Four days after the initial evaluation by the AT, the team physician evaluated the patient because his symptoms had not improved with nonoperative management. Examination revealed no new findings and also indicated brachial plexus traction injury. Cervical spine radiographs were negative for bony abnormalities. Over the next week, the patient did not participate in practice and was limited to isolated lower extremity strengthening and conditioning. Nonoperative management with ice, hot packs, massage, electrical stimulation, shortwave diathermy, and over-the-counter nonsteroidal anti-inflammatory drugs provided minimal improvement of his symptoms.

Ten days after initial presentation to the athletic training department and 16 days postinjury, the patient's symptoms continued to persist with nonoperative management. Reexamination by the AT now revealed a positive Spurling test, increasing concern for a nerve root pathologic lesion. The patient was referred to his primary physician for further evaluation. The physician diagnosed C7 radiculopathy of the left upper extremity because of the radicular symptoms, weakness, and sensory loss. He arranged both a magnetic resonance imaging (MRI) scan and follow-up appointment with a neurosurgeon for the next day. The MRI showed evidence of a left paracentral disc herniation at the T1–T2 level with resultant mild to moderate central narrowing and moderate left lateral recess narrowing of the vertebral canal (Figure 1). Immediately after the MRI, the patient met with the neurosurgeon to review these findings and for further evaluation. Based on his own examination of the patient, the neurosurgeon provided an initial diagnosis consistent with that of the referring physician: C7 radiculopathy. However, after reviewing the MRI, the neurosurgeon diagnosed a postfixed brachial plexus with substantially increased contribution from T1 to the posterior cord and radial nerve. He determined the posterolateral herniation of the T1–T2 disc impinging on the T1 nerve root combined with the postfixed brachial plexus resulted in symptoms typical of a C7 radiculopathy due to the increased contribution from T1 to the posterior cord. The patient desired aggressive intervention because he was an elite wrestler in his final season and the upcoming national tournament was only 6 weeks away. Given the reproducible radicular symptoms with weakness and sensory loss secondary to an acute injury, the neurosurgeon believed surgical intervention was warranted to relieve symptoms and allow faster return to activity. The next day, the patient underwent surgery for excision of the T1–T2 intervertebral disc herniated nucleus pulposus and T1–T2 laminoforaminotomy to decompress the T1 nerve root.

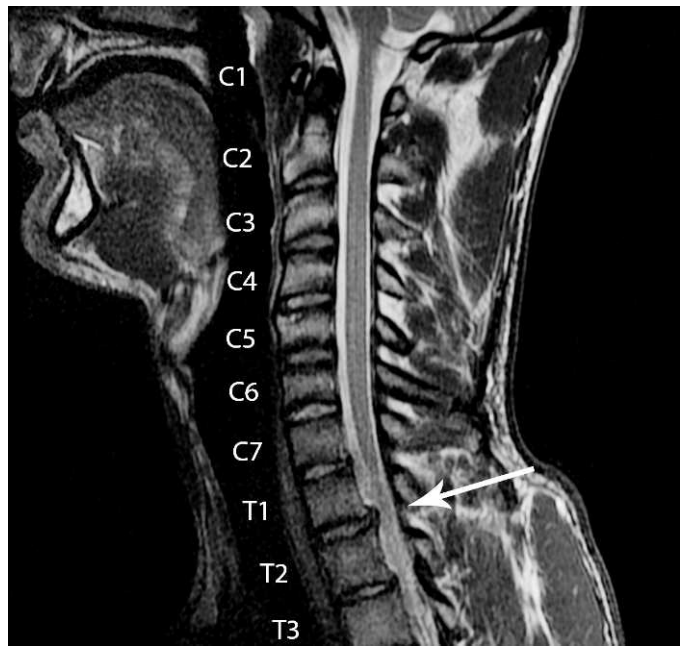

Figure 1. T2-weighted sagittal magnetic resonance image shows an intervertebral disc herniation at the T1–T2 level (arrow).

After surgery, the patient was restricted from participating in all activity for 10 days until his sutures were removed and his wound had healed. At that time, the patient resumed cardiovascular conditioning and initiated upper extremity strengthening exercises under AT supervision. Rehabilitation began with rotator cuff strengthening, triceps extensions, and lateral raises using resistance bands. The patient quickly progressed over the next week and added push-ups, chin-ups, dips, seated and bent rows, and dumbbell chest presses with increasing repetitions and weight daily. Eighteen days after surgery, the patient was evaluated by his neurosurgeon. The examination showed the surgical wound was healed fully; a negative Spurling test; no pain,

paresthesia, or reproducible symptoms; and normal strength. Based on the examination, the neurosurgeon released the patient to participate fully without restrictions in all wrestling activities. The patient resumed practicing and experienced only minimal, transient discomfort with left upper extremity traction or axial loading of his neck. Thirty-one days after surgery, he participated in the conference championships without difficulty, winning first place in his weight class. The patient competed at the national tournament without symptoms just over 6 weeks after surgery, earning National Collegiate Athletic Association All-American honors.

DISCUSSION

The brachial plexus comprises the ventral rami of the C5–T1 spinal nerve roots, which join together to form the roots, trunks, divisions, cords, and terminal branches of the brachial plexus (Figure 2).[5,7–12] This distribution is the most common organization of the brachial plexus, but it has many variations, 2 of which are known as prefixation and postfixation. The prefixed brachial plexus occurs when the C4 nerve root provides substantial contribution to the brachial plexus and the T1 nerve root provides little to no contribution, effectively creating a C4–C8 brachial plexus innervation. Conversely, a postfixed brachial plexus receives little or no innervation from C5 and substantial innervation from T2, resulting in essentially a C6–T2 brachial plexus distribution. Prefixation is much more common, occurring in 26% to 48% of the population, whereas postfixation is found in up to 4% of the population.[11,13–17] In most people, these variations are asymptomatic and have little clinical relevance. However, our case demonstrates that in the setting of intervertebral disc herniation or other pathologic condition, these variations can cause symptoms that do not match expected injuries, making accurate diagnosis challenging and affecting the management strategy.

The brachial plexus typically is injured by acute trauma via traction or compression of the plexus or of the spinal nerve roots forming the brachial plexus. Traction injuries occur when the shoulder is depressed and the neck is forcefully flexed laterally to the contralateral side. Compression injuries may occur either from ipsilateral lateral flexion of the neck with shoulder elevation or from stenosis of the spinal canal, such as with axial loading of the spinal column while the neck is extended.[2–5] Both types of injuries can occur during collision sports, such as football, hockey, or wrestling. Patients with brachial plexopathy most commonly present with symptoms such as radicular pain, tingling, numbness, or weakness of the upper extremity. Patients with cervical radiculopathy, which may be caused by cervical spondylosis, foraminal or spinal stenosis, or intervertebral disc herniation, often present with symptoms similar to a brachial plexopathy, making it difficult to differentiate clinically.[6] Brachial plexopathies are classified as peripheral nerve injuries, whereas cervical radiculopathies are considered spinal nerve or spinal nerve root injuries.

Intervertebral disc herniation most commonly occurs in the lumbar spine and less frequently in the cervical spine secondary to chronic degenerative changes over time. Comparatively, thoracic intervertebral disc herniation is very rare, responsible for less than 1% of all herniated discs reported in most studies.[18–22] Within the thoracic spine, most disc herniations occur at or below T8 and account for approximately 75% of all thoracic disc herniations, with most occurring at T11–T12.[18–20] High-level thoracic disc herniation (T1–T4) is very uncommon, with T1–T2 disc herniations accounting for less than 6% of all thoracic disc herniations.[18–26] In a review of 280 thoracic disc herniations, Arce and Dohrmann[20] found only 25% of patients had histories of trauma but noted that symptoms lasting more than 24 hours but less than 1 month and including signs of spinal cord compression or radicular symptoms were almost always associated with a history of trauma. Similarly, Abbed and Coumans[3] reported acute cervical radiculopathy in younger patients is more likely due to intervertebral disc injury. In retrospect, the mechanism of injury for our patient was likely more complex than upper extremity traction alone. Our patient was shooting on his opponent, making the possibility of axial loading of the neck fairly high. This mechanism coupled with the extremity traction and acute onset of symptoms (radicular pain, weakness, and numbness) supports the ultimate diagnosis of an acute intervertebral disc herniation. However, as noted, cervical radiculopathy due to disc herniation can present very similarly to brachial plexopathy, and given his report of the injury, it is easy to see the challenge in identifying the true pathophysiology of his symptoms. Another item to consider is that our patient had a history of traumatic lumbar disc herniation secondary to an axial-load injury 2 years earlier. How much this factored into his current injury cannot be quantified and is beyond the scope of this paper, but it raises questions for future discussion.

A T1–T2 disc herniation commonly compresses the T2 nerve root, but lateral protrusion may impinge on the T1 nerve root, as occurred in our case. A patient with T1 impingement typically presents with radicular pain radiating down the medial forearm to the medial hand and digits 4 and 5; weakness of the intrinsic hand muscles; and anterior chest, shoulder, and axillary pain. Rarely, a patient with T1–T2 disc herniation may present with Horner syndrome (ipsilateral ptosis, miosis, anhydrosis, and erythematosis) because the herniation compresses the sympathetic outflow from the spinal cord.[3,27–32] The C8 radiculopathy has a similar presentation, but it does not include anterior chest pain and typically also includes wrist and finger flexion-extension deficits.[27,28,33,34] Presentation of a "typical" C7 radiculopathy includes weakness of elbow and wrist extension, sensory loss over the posterior upper extremity, and decreased triceps reflex.[2,3,5] Radicular pain in acute radiculopathy is often the primary concern and typically is distributed in a myotomal rather than dermatomal distribution (ie, triceps pain in a C7 radiculopathy).[3] The radial nerve, which is the termination of the posterior cord, innervates much of the distribution of the C7 nerve root, making injury to either structure present similarly. The posterior cord typically receives innervation from all nerve roots but is highly variable. This variation is important to understand because injury to any nerve root potentially can cause C7 radiculopathy symptom patterns. Our case demonstrates how the combination of multiple factors can make the diagnosis challenging. In our patient, the postfixed brachial plexus meant that his posterior cord

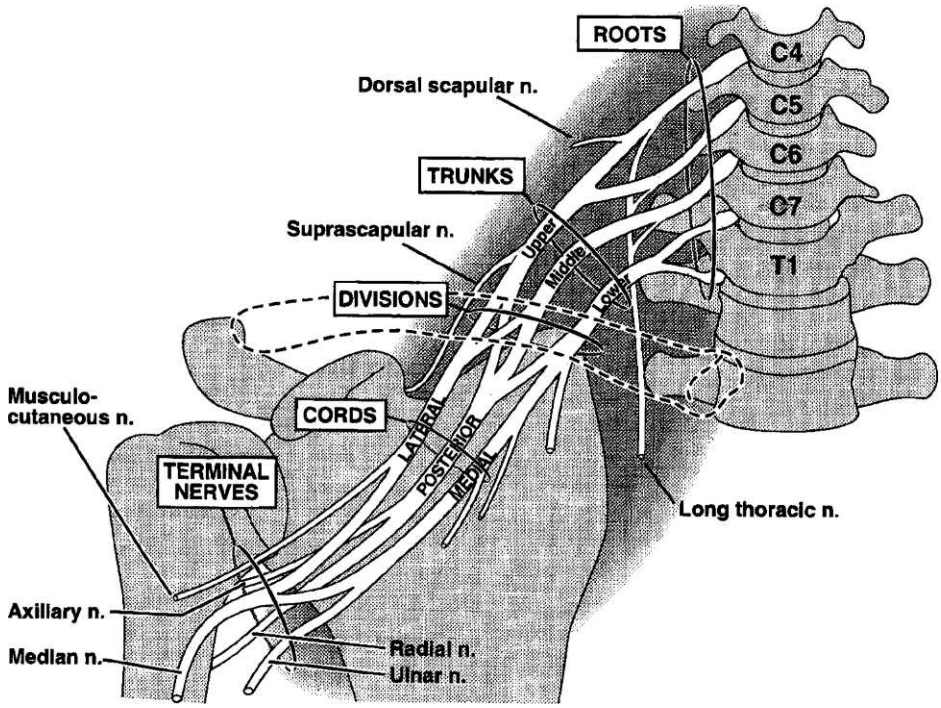

Figure 2. Illustration of the brachial plexus. Reprinted from Ferrante MA. Brachial plexopathies: classification, causes, and consequences. *Muscle Nerve.* 2004;30(5):547–568 with permission from John Wiley and Sons.

likely was receiving substantially more contribution from T1 than normal. The lateral T1–T2 disc herniation causing impingement of the T1 nerve root produced symptoms within the posterior cord distribution more commonly associated with C7 nerve root injuries.

Evaluation of brachial plexopathies commonly first occurs on the field with noting the mechanism of injury; most brachial plexus injuries occur after a direct blow to the head, shoulder, or upper extremity. Initial physical examination should focus on ruling out any cervical or brachial neurovascular injury or other trauma, such as fractures. When serious injury has been excluded, further examination for orthopaedic and nonorthopaedic injuries, including provocative testing of the neck, shoulder, and upper extremity, can be completed.

Given that most brachial plexopathies resolve spontaneously, initial management should entail nonoperative therapies and modalities, such as rest, ice, physical therapy, and nonsteroidal anti-inflammatory drugs or oral corticosteroids.[6,35–37] Patients whose symptoms do not begin to improve within a few weeks or worsen with nonoperative management may need further evaluation and radiographic imaging. Plain radiographs of the cervical spine should be obtained to rule out a bony fracture or dislocation that may be impinging on the nerve root. If plain radiographs are negative, computed tomography may help identify occult bony injury or hemorrhage. Magnetic resonance imaging is most effective for evaluating the patient for a soft tissue injury, disc herniation, foraminal pathologic lesion, or direct neural injury.[3,5,35,36,38]

Surgical intervention is warranted when (1) nonoperative therapy has been unsuccessful and symptoms have persisted for at least 3 to 4 weeks, (2) symptoms are worsening despite nonoperative management, or (3) the patient has weakness or sensory loss with associated radicular pain.[35,36,38,39] Although the potential loss of time from training and competition in competitive athletes is not normally an indication for surgical intervention, this factor may be considered in the decision to undergo surgical intervention.[36] Our patient's symptoms had persisted for less than 3 weeks, but the neurosurgeon believed surgical intervention was warranted given the patient's reports of acute-onset radicular pain with weakness and sensory loss after a specific trauma coupled with no improvement after nonoperative management. Surgical intervention may be performed through an anterior or posterior approach and is determined by the surgeon so as to provide the best possible outcome. In patients with reproducible radiculopathy or lateral disc herniations, laminoforaminotomy with concomitant discectomy as indicated via a posterior approach is ideal.[35,36,38–43] Laminoforaminotomy widens the intervertebral foramen of adjacent vertebrae to allow decompression of the impinged nerve root without decreasing stability.[36,40–43] Posterior laminoforaminotomy and discectomy is a well-tolerated procedure with positive outcomes. Most patients achieve pain relief and improvement of preoperative symptoms and muscle weakness secondary to impingement.[40–42]

Assessing return-to-participation status is conducted similarly for patients managed nonoperatively or operatively. In general, the patients should be pain free, have full range of motion, and have normal strength before returning to competition.[35–38,44] In addition, patients should not have reproducible symptoms with the Spurling maneuver or brachial plexus traction testing.[35,36] After posterior laminoforaminotomy, many patients can return to work and activity within 2 to 3 weeks and most return within 4 weeks.[38] Heavy laborers and athletes may take longer to return due to the increased demands on the shoulder and cervical musculature but typically return to full activity and

competition within 6 to 8 weeks.[36–38] Ultimately, return to participation depends on the patient being pain free and asymptomatic and regaining full strength.[35,36] Adamson[36] reported that competitive athletes returned to contact sports between 2.5 and 6 weeks after posterior laminoforaminotomy when these criteria were met. He attributed this finding to better-than-average healing and increased motivation. Our patient mirrored the patients Adamson[36] described: his preoperative conditioning level was very high, he was highly motivated, and the procedure relieved him of symptoms, allowing him to return to preinjury strength and conditioning levels quickly and receive clearance at 2.5 weeks. Whereas our patient had a successful outcome, care must be taken to ensure each patient returns to activity only when appropriate based on the physician's physical examination and clinical judgment.

CONCLUSIONS

Brachial plexopathy is a common athletic injury, and all health care professionals should be familiar with the presenting signs and symptoms. As our case showed, more serious conditions may mimic brachial plexopathy. Given that upper-level thoracic disc herniation and postfixed brachial plexus are uncommon conditions, health care providers, especially those caring for athletes in collision sports, should be aware of these potentially confounding disorders. Clinicians need to be receptive to all possible causes of a patient's symptoms because the injury may be worse than initially thought or the original diagnosis may be incorrect. They must be willing to re-examine or refer a patient for further evaluation if symptoms do not respond as expected. Collaboration and good communication within the entire medical team is essential to facilitate accurate diagnosis and management, as this case demonstrated.

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