[Orthopaedic Surgery]

Does a Syrinx Matter for Return to Play in Contact Sports? A Case Report and Evidence-Based Review of Return-to-Play Criteria After Transient Quadriplegia

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Transient quadriplegia is a rare injury that can change the course of an athlete's career if misdiagnosed or managed inappropriately. The clinician should be well versed in the return-to-play criteria for this type of injury. Unfortunately, when an unknown preexisting syrinx is present in the athlete, there is less guidance on their ability to return to play. This case report and review of the current literature illustrates a National Collegiate Athletic Association (NCAA) Division I football player who suffered a transient quadriplegic event during a kickoff return that subsequently was found to have an incidental cervical syrinx on magnetic resonance imaging. The player was able to have a full neurologic recovery, but ultimately he was withheld from football.

Keywords: transient quadriplegia; syrinx; return to play

Cervical spine injury in contact sports resulting in transient quadriparesis has been well described.[5,7-9,12,14,20,22-26] Transient motor or sensory loss results from a contusion, causing demyelination of the axons at the level of injury.[1,26] This rare injury is estimated to occur in 2 per 100,000 collegiate football players.[3] Cervical spine fracture, congenital stenosis, fusion, instability, or spondylosis are risk factors for this event. Return-to-play criteria after an episode of transient quadriplegia have been promulgated, following rigid radiologic guidelines.[7,8,12,22,23] To prevent the possibility of catastrophic recurrence, it is critical to understand risk factors for recurrence when deciding safe return to play versus recommendation of permanent cessation from contact sport.

The use of magnetic resonance imaging (MRI) has led to improved recognition of associated spinal cord and soft tissue injury after acute transient quadriplegic events.[5] However, this increased information has also added new levels of uncertainty to classic return-to-play decision-making algorithms, which are based solely on clinical and plain radiographic findings.[8,11,12,23] This case report highlights the return-to-play dilemma when an athlete has a single transient quadriplegic event during sport, with follow-up excellent range of motion (ROM) and strength, near normal radiographic parameters, but abnormal MRI findings of an asymptomatic congenital syrinx.

CASE REPORT

A 22-year-old man, Division I football athlete with no previous medical history was hit in the chest while on punt coverage and had an immediate loss of consciousness for approximately

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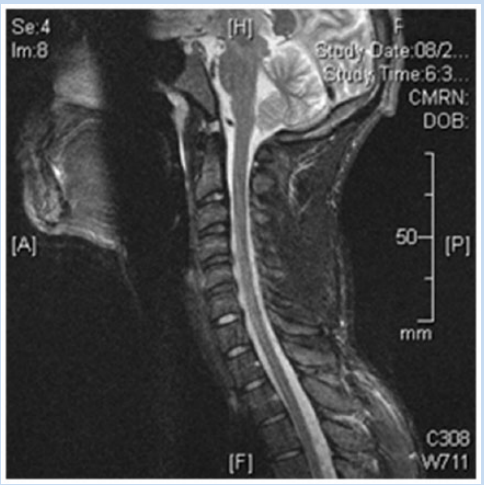

Figure 1. Immediate postinjury magnetic resonance image demonstrating intact spinal cord canal with cerebrospinal fluid signal surrounding the spinal cord at all levels.

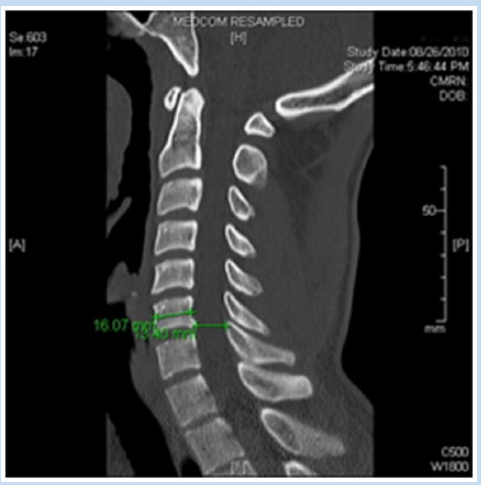

Figure 2. Immediate postinjury computed tomography scan demonstrating normal cervical spinal canal width and measurements for Torg ratio.

30 seconds. The hit was described as a direct lateral blow causing his head to at first hyperflex ventrolaterally then rebound in hyperextension. He described dense quadriplegia once he regained consciousness approximately 30 seconds after the hit. His cervical spine was immediately immobilized, and initial on-field evaluation by the team physician revealed 0/5 motor strength in all extremities while conscious, along with absent sensation to touch in all extremities (Figures 1 and 2). He was then taken to a level 1 trauma center, and in the ambulance, regained slight sensation to touch with his left upper and lower extremities, but no motor function. His sensation progressively improved with his primary complaint being weakness in all 4 extremities. He had good rectal tone, anal sensation, and a bulbocavernosus reflex. Using the standard neurologic classification of spinal cord injury, the ASIA (American Spinal Injury Association) scale,[13] he scored 20 out of 50 for bilateral upper and lower extremity motors and was 112 out of 112 for his sensation, giving him an ASIA impairment scale as "C" (incomplete), as he had motor function with muscles below neurologic level with a grade of less than 3 out of 5. A computed tomography (CT) scan of his head and cervical spine revealed no bony abnormalities. An MRI of his cervical spine revealed no congenital narrowing of his canal and no bulging disc, with a syringomyelia of the cervical spinal cord extending from the level of C5 to C7.

Examination after imaging showed improvement bilaterally of his upper extremity motor function to 3 out of 5. His strength gradually improved through the night to 4 out of 5 in his upper extremities, and 5 out of 5 in his lower extremities. His motor strength further improved to 5 out of 5 in upper and lower extremities on postinjury day 2. His cervical spine was not tender or dynamically radiographically unstable. He was discharged on postinjury day 2. On follow-up examination, he had full pain-free cervical ROM, intact preinjury neck strength, and radiographic evidence of normal lordotic curvature, normal anteroposterior diameter C3 to C7, abnormal C3 to C4 Torg ratio (C3, 0.69; C4, 0.79), and no radiographic instability (Figure 3). Follow-up MRI demonstrated no evidence of disc disease, spinal cord bleeding, or contusion. Postinjury MRI showed increased T2 signaling in the central cord consistent with either benign central cord dilation or congenital syringomyelia (Figure 4). Given the abnormal MRI findings and history of 48 hours of neurologic deficit, the decision was made to disqualify him from further participation in contact sports. At final 2-year follow-up, he remains neurologically intact without subsequent episodes during daily activities. All patient confidentiality is protected in this report according to the US Health Insurance Portability and Accountability Act (HIPAA).

DISCUSSION

Current evidence-based guidelines suggest that a player who experiences a single episode of transient quadriplegia may return to play with no increased risk of spinal cord injury resulting in paralysis if they have had no previous episodes, achieve complete neurologic recovery and full cervical ROM, have a spinal canal anterior-posterior diameter of >13 mm, and have no CT or MRI evidence of functional stenosis, ligament injury, intrinsic cord abnormality, or neural compression (Tables 1 and 2).[8,12]

Torg and colleagues[22-24] are credited with determining the association between transient neuropraxia and stenosis of the cervical canal. Specifically, sagittal plane cervical spinal canal narrowing is associated with the risk of transient quadriparesis.[1,7] Historically, cervical stenosis has been measured by 2 methods: the anterior to posterior diameter and a ratio method.[11,24] A normal spinal canal, from C3 to C7, has an anterior-posterior

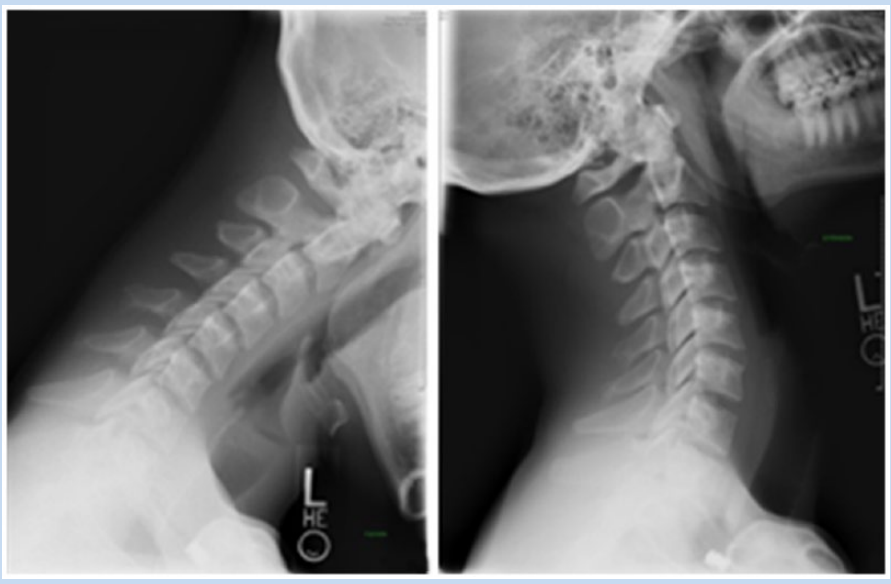

Figure 3. Flexion-extension radiographs at follow-up examination. Torg ratios: C3, 0.69; C4, 0.79; C5, 0.93; C6, 0.90; C7, 0.80.

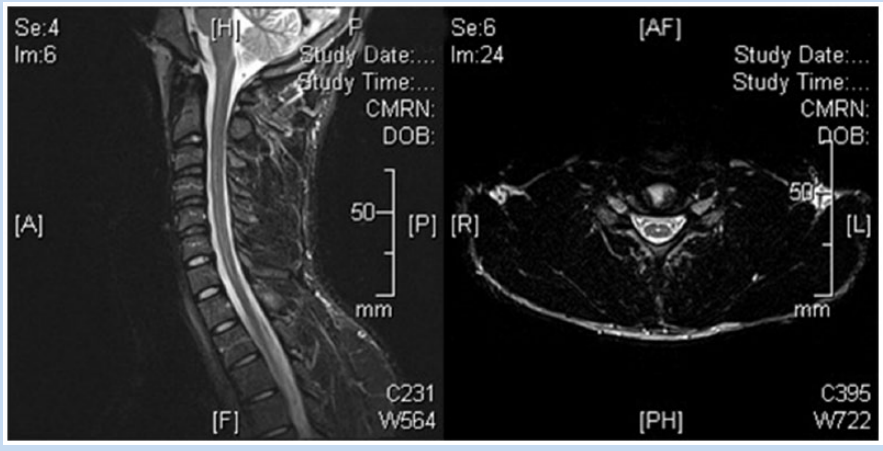

Figure 4. Follow-up magnetic resonance image demonstrating a syrinx in the central canal of the spinal cord.

diameter of 15 mm or greater when measured by plain radiographs. A stenotic spinal canal has a diameter of less than 13 mm.[11,24] The ratio method compares the anterior-posterior diameter with the diameter of the cervical body at the measured level when measured on lateral radiographs.[24] A normal ratio is defined as 1:1, with a ratio of less than 0.8 indicative of a stenotic canal.[17,23,24] Using Torg's definition, he has stenosis at C3 and C4. Patients without spinal stenosis defined by sagittal diameter >15 mm often have stenosis by the ratio method.[1,23,24]

In the evaluation by Torg et al[24] of 45 athletes with transient neuropraxia, the duration of motor weakness ranged from 2 minutes to 48 hours and sensory deficits from 2 minutes to 48 hours. One patient had weakness with dorsiflexion of his foot for 48 hours, and 1 had quadriplegia initially with weakness in his upper extremities for 2 weeks and lower extremities for 1 month after the injury. The central syringomyelia in our patient's cervical spine may have contributed to the delayed recovery of his upper motor strength. The upper extremity motor axons are more centrally located in the spinal cord compared with the lower extremity, placing these neurons closer to the syrinx.

A case series of cervical spine injury in rugby found that buckling of the cervical column via hyperextension was the major cause of injury while tackling.[14] It is argued that for football players suffering cervical spine contusion via hyperextension, the Torg ratio, which measures the space available to the spinal cord in a region that does not change with ROM, should not be used to predict spinal cord injury.[5] Instead, dynamic stenosis, measured from the posterior/inferior

Table 1. Recommendations for return to play after transient quadriparesis[8,12,18,21]

No previous episode of transient quadriparesis
Complete neurologic recovery
Return of preinjury strength
Full cervical range of motion
Spinal canal AP diameter of >13 mm
No CT/MRI evidence of functional stenosis or ligament injury

AP, anterior-posterior; CT, computed tomography; MRI, magnetic resonance imaging.

Table 2. Contraindications to return to play after transient quadriparesis[8,12,18,21]

Spinal canal AP diameter <13 mm
Functional stenosis on CT/MRI
Spinal cord compression
Multiple occurrences

AP, anterior-posterior; CT, computed tomography; MRI, magnetic resonance imaging.

border of the superior vertebra to the cephalad/anterior-most edge of the lamina of the inferior vertebra, should be used. This space narrows as the neck is extended and posterior translation of the superior vertebral segment occurs. More anterior-posterior translation is seen at the C3 to C4 level, as are more injuries.[5]

Torg et al[23] suggested that anyone with a spinal canal diameter of <13 mm is more at risk for spinal cord injury, but not permanent injury, and thus not an absolute contraindication to return to play in the absence of residual symptoms. There must be no (1) ligamentous instability, (2) disc involvement, (3) degenerative changes, (4) MRI evidence of cord defect or swelling, (5) positive neurologic findings lasting more than 36 hours, and (6) more than 1 reoccurrence.[22]

Cantu expanded that theory of increased risk to "functional stenosis" (ie, decreasing spinal canal diameter and cerebral spinal fluid space in flexion and extension). Cantu et al[8] suggest that any MRI abnormality, even with a normal neurologic examination, is a relative contraindication to return to play, as is transient quadriparesis lasting >24 hours or a second episode of transient quadriparesis. A stenotic canal (diameter <13 mm), functional stenosis on CT or MRI, cord compression, or edema are all absolute contraindications to return to play.[8] Mild to moderate stenosis in the absence of spinal cord parenchymal injury with full cervical ROM and no residual symptoms is a relative contraindication.[12] Evidence of intrinsic cord abnormality or other MRI finding should be a relative contraindication to play until evaluated further.[12]

There is disagreement over the relevance of abnormal MRI findings in an otherwise healthy athlete or an athlete who has returned to normal health and activity after injury. T2 changes in the cord on MRI or syrinx may be grounds to not return to play.[21] The conservative nature of these recommendations is understandable given the potential implications and the limited baseline MRI data from which to judge the importance of these abnormalities. In 10 athletes with transient spinal cord injury, 4 demonstrated cerebral spinal fluid (CSF) signal preservation with MRI, indicating lack of spinal cord compression. None of the 4 athletes suffered recurrent symptoms after return to sport. It does not appear that a single episode of temporary spinal cord dysfunction in an athlete with spinal stenosis or prodrome or recurrent symptoms will substantially increase the risk of future catastrophic spinal cord injury.[2] MRI of CSF signal may not be "protective," rather, it may correlate with the lack of irreversible spinal cord compression in high-velocity injuries.[2] In a series of football players with evidence of spinal cord contusion, a correlation between MRI of a spinal cord contusion (without evidence of other abnormality) and neurologic symptoms was not shown.[5] For this reason, MRI of a spinal cord contusion should not be used as an absolute contraindication to return to play without considering symptomatic correlation.[5]

According to the neurosurgical return-to-play criteria,[18] the central cord dilatation is benign and should not preclude a healthy player returning to play. Other rare entities in competitive athletes include Chiari malformations. If the player has an asymptomatic minor malformation, (<5 mm cerebellar tonsillar descent) without neurologic findings, there is no contraindication to play.[10] Also, if players must undergo surgical decompression for a symptomatic malformation, they may return to play as soon as they are neurologically intact.[18] An asymptomatic, severe malformation (>5 mm) is a relative contraindication to play and should be evaluated more closely by a neurosurgeon. A symptomatic malformation is an absolute contraindication to return to play (Table 3).[10,18]

The exact cause and pathophysiology of syringomyelia due to congenital malformation such as Chiari malformation has yet to be ascertained.[19] Posttraumatic syringomyelia is another variant.[4,6] Its onset is usually within a few months of the spinal cord trauma.[6] Pain, swelling, or headache can be the presenting symptoms of a previously asymptomatic syrinx.[19] Often, this abnormality is discovered after an acute injury, with the symptoms initially attributed to the event and the syrinx found on further investigation.[15]

It is not known whether the syrinx contributes to functional stenosis of the cervical spine. It is possible that a syrinx may cause intrinsic narrowing of the CSF surrounding the spinal cord, effectively pressuring the cord outward toward the walls of the canal or rendering the cord less compliant on bending. A

Table 3. Spinal cord abnormalities and return-to-play (RTP) criteria[10,18]

Syringomyelia
RTP:
Benign dilatation of central canal, no other findings on imaging or examination
Relative RTP:
Symptomatic syringomyelia (base return on treatment decided by spine surgeon)
Spinal cysts
RTP:
After referral to a spine surgeon for evaluation; most are benign, allowing for immediate return
Tethered spinal cord
RTP:
Asymptomatic radiographic finding; follow until skeletally mature
Neurologically intact after surgery
Relative RTP:
Symptomatic with athletic activity (may be candidate for prophylactic surgery)
Spinal cord tumor
RTP depends on:
Extent and success of surgical intervention
Length of bony exposure required
Subsequent stability of spine
Neurologic status
Chiari malformation
RTP:
Asymptomatic minor malformation (<5 mm cerebellar tonsillar descent)
Neurologically intact after surgical decompression for symptomatic malformation
Relative RTP:
Asymptomatic severe malformation (>5 mm)
No RTP:
Symptomatic malformation

syrinx that is asymptomatic may become symptomatic transiently as the canal is further narrowed by extended or traumatic hyperextension of the cervical spine, as in a football 3-point stance.[15] One case report presents an otherwise healthy male defensive end who was found to have left-side weakness and subsequent mild upper extremity atrophy that initially presented when getting into a 3-point stance and was noted only during football practices. He had a syrinx extending from C5 to T1. Conservative measures resolved his symptoms over 3 months. His syrinx remained stable on MRI, but he did not return to collision sports.[15]

Return to play after a spinal cord injury has become an important topic as we further our understanding of the long-term consequences of this type of trauma.[16] MRI has expanded our ability to examine the spinal cord after injury, but has made us aware of other pathology that may affect the player's neurologic status or risk for future injury. It will be important to continue to monitor these players for future sequelae and report the findings to improve our understanding of this potentially critical injury.

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