Athletic Cervical Spine Injury in the Setting of Fusion Failure of the Anterior and Posterior Atlas

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abstract

This article describes a rare congenital abnormality of anterior and posterior C1 fusion failure presenting after an acute athletic injury to the fibrous nonunion. C1 congenital malformations are rare, occurring in approximately 2% of patients; even rarer are combined anterior and posterior arch malformations in the same patient. Posterior ring abnormalities are more common than anterior ring injuries (4.5:1, respectively). To the authors’ knowledge, combined anterior and posterior ring congenital malformations with subsequent injury have not been previously described.

In the current patient, a congenital failure of fusion of the anterior and posterior arches of C1 was identified. The anterior fibrous nonunion was injured while the patient played football, leading to transient neurologic injury and dysphagia from soft tissue swelling. The patient was initially diagnosed with an acute fracture at another facility; however, given advanced imaging, flexion and extension views, and a normal neurologic examination, the authors diagnosed a traumatized congenital defect. The injury healed with a short period of cervical collar immobilization and supportive measures. Such malformations are usually found incidentally, but they can be symptomatic after trauma. Images to distinguish these deficits can be difficult because the differences between chronic nonunions and congenital malformations are subtle. Surgery is rarely indicated for congenital malformations because they are often stable even after injury; however, they may predispose patients to neurologic injury in the future with high-risk activities. Because the current patient had an increased chance of future injury secondary to the lack of bone formation in the C1 vertebrae, he was restricted from participating in contact sports.

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CONGENITAL DEFECTS OF THE FIRST CERVICAL VERTEBRA ARE UNCOMMON ANATOMIC VARIANTS. THEY ARE SECONDARY TO FAILURE OF FUSION OF THE SYNCHONDROSSES NORMALLY PRESENT IN DEVELOPING SPIKES. ALTHOUGH RARELY SYMPTOMATIC OR CONSEQUENTIAL, THEY ARE OFTEN MISTAKEN FOR TRAUMATIC LESIONS. THIS ARTICLE DESCRIBES A CASE OF AN INJURY TO THE FIBROUS NONUNION OF THE ANTERIOR ATLAS IN A PATIENT WITH AN INCOMPLETELY OSSIFIED ANTERIOR AND POSTERIOR ATLAS.

CASE REPORT
A 16-YEAR-OLD MALE HIGH SCHOOL FOOTBALL PLAYER SUSTAINED A FLEXION COMPRESSION INJURY DURING A GAME BUT IMMEDIATELY RETURNED TO PLAY. HE SUBSEQUENTLY SUSTAINED A FLEXION/LEFT LATERAL BENDING INJURY DURING A COLLISION AND EXPERIENCED IMMEDIATE NUMBNESS AND TINGLING IN THE UPPER EXTREMITIES LASTING 5 MINUTES. HE DID NOT RETURN TO PLAY OR SEEK MEDICAL ATTENTION. HE PRESENTED TO ANOTHER HOSPITAL’S EMERGENCY ROOM 2 DAYS LATER, REPORTING CONTINUED NECK PAIN AND DYSPHAGIA. HE REPORTED NO NUMBNESS, TINGLING, OR WEAKNESS IN HIS EXTREMITIES. HE WAS DIAGNOSED WITH A C1 FRACTURE ON COMPUTED TOMOGRAPHY (CT) AND TREATED WITH A RIGID CERVICAL COLLAR.

ON PRESENTATION TO THE CURRENT AUTHORS’ CLINIC 2 WEEKS POSTINJURY, HE REPORTED CONSTANT, DIFFUSE, DULL NECK PAIN AND PERSISTENT DYSPHAGIA WITH NO OTHER SYMPTOMS. HE HAD CONTINUED TO WEAR HIS COLLAR AND AVOID CONTACT SPORTS AND HAD BEEN TAKING 1 TABLET OF OXYCODONE HYDROCHLORIDE/ACETAMINOPHEN DAILY AND 600 MG OF IBUPROFEN EVERY 6 HOURS FOR PAIN.

THE MEDICAL, SURGICAL, AND FAMILY HISTORY AND REVIEW OF SYMPTOMS WERE NONCONTRIBUTORY. PHYSICAL EXAMINATION SHOWED A WELL-DEVELOPED BOY IN NO APPARENT DISTRESS. DIFFUSE TENDERNESS TO PALPATION EXISTED IN THE POSTERIOR ASPECT OF CERVICAL SPINE WITHOUT POINT TENDERNESS OR STEPOFFS. HE HAD MILD PARASPINAL MUSCLE SPASMS. EXTREMITY STRENGTH WAS 5/5 THROUGHOUT. SENSATION WAS INTACT TO LIGHT TOUCH AND PINPRICK IN ALL DERMATOMES. ALL EXTREMITY REFLEXES WERE 1+ THROUGHOUT. HOFFMANN AND BABINSKI SIGNS WERE NEGATIVE, AND NO ANKLE CLONUS OCCURRED. GAIT WAS NORMAL.


ORIGINAL ASSESSMENT WAS NECK STRAIN WITH INCOMPLETE OSSIFICATION OF THE ANTERIOR AND POSTERIOR ATLAS AND TRAUMATIC INJURY TO THE SYNOSTOSIS OF THE C1 ARCH, WITH THE ANTERIOR HEMATOMA AS THE LIKELY CAUSE OF THE DYSPHAGIA. THE PATIENT WAS INSTRUCTED TO INCREASE THE IBUPROFEN TO 800 MG 3 TIMES DAILY AS NEEDED FOR PAIN, CONTINUE USING THE CERVICAL COLLAR, AVOID CONTACT SPORTS, OBTAIN ADDITIONAL CT AND MRI SCANS OF THE CERVICAL SPINE, AND RETURN TO THE CLINIC IN 2 WEEKS.

FIVE WEEKS POSTINJURY, HE REPORTED GRADUAL NECK PAIN IMPROVEMENT AND RESOLUTION OF HIS DYSPHAGIA. HE REPORTED NO NUMBNESS, TINGLING, OR WEAKNESS IN HIS EXTREMITIES OR CHANGES IN BOWEL OR BLADDER FUNCTION. HE WORE HIS CERVICAL COLLAR AT ALL TIMES. PHYSICAL EXAMINATION WAS UNCHANGED. COMPUTED TOMOGRAPHY SCANS OBTAINED AFTER THE 2-WEEK POSTINJURY VISIT SHOWED A 4-MM COMPLETE DEFECT THROUGH THE ANTERIOR ARCH OF C1, SLIGHTLY TO THE LEFT OF THE MIDLINE, WITH CORTICATED BUT SLIGHTLY ILL-DEFINED OSSUS MARGINS (FIGURES 2, 3). A SMALLER COMPLETE DEFECT ALSO EXISTED THROUGH THE POSTERIOR ARCH OF C1 WITH CORTICATED AND WELL-DEFINED OSSUS MARGINS (FIGURES 2, 3). MAGNETIC RESONANCE IMAGING SCANS SHOWED RESOLUTION OF THE RETROPHARYNGEAL HEMATOMA, CONGENITAL NONFUSION OF THE POSTERIOR ARCH OF C1, AND CONGENITAL NONFUSION/FIBROUS NONUNION OF THE ANTERIOR ARCH OF C1, BUT NO TRAUMATIC ABNORMALITIES.

FINAL DIAGNOSIS WAS CONGENITAL NONUNION OF THE ANTERIOR AND POSTERIOR ARCHES.
of C1 with fibrous nonunion of the anterior arch. The fibrous nonunion was likely torn during the football injury, causing edema and a hematoma that resulted in dysphagia and neck pain. The patient was gradually weaned from the cervical collar and started on neck stabilization and a strengthening physical therapy regimen. He was restricted from contact sports.

**Discussion**

Ossification of the first cervical vertebra normally begins during the seventh week of fetal development. Three primary ossification centers exist: 1 for each neural arch and 1 for the body. Posterior arch fusion generally occurs by age 3, and anterior arch or neural central synchondrosis generally occurs by age 7. Three main developmental anomalies have been described: (1) the body may be formed by 2 centers that fuse first with each other and then with the neural arches; (2) the body’s ossification center may fail to appear, and forward extension and fusion of the anterior portions of each lateral mass may occur; and (3) the body’s center may fail to appear, and the lateral masses may fail to fuse, resulting in anterior spondyloschisis. Failure of development and fusion of the posterior arch is more common than that of the anterior arch (4.5:1, respectively); both are rarely clinically significant, except when associated with trauma or other cervical defects that render the spine unstable. The incidence of combined defects is similar to that of anterior defects alone.

Classification is based on CT findings: type A, failure of posterior midline fusion (most common; 97% of posterior defects; population incidence, 2.6%); type B, unilateral lateral cleft (second most common; population incidence, 0.54%); type C, bilateral lateral cleft (0 of 1354 cases studied); type D, absence of posterior arch with persistent posterior tubercle (0 of 1354 cases studied); and type E, complete absence of posterior arch (0.18% of cases studied). Because types A and B are less clinically significant and often need no intervention, accurate diagnosis is required when confounded by trauma. However, types C and D may lead to neurologic symptoms and require surgical intervention.

Lipson and Mazur reported a patient who presented with transient neck pain after a football injury. Initial radiographs were interpreted as a C1 burst fracture, and the patient was placed in cervical halter traction. However, additional imaging showed that the patient had anteroposterior spondyloschisis. Galindo and Francis described a 5-year-old boy who sustained a vertical compression injury of the occiput; initial findings were consistent with those of a cervical spine injury. Conventional radiographs and CT scans confirmed a C1 fracture through congenital anterior and posterior arch defects. Because flexion and extension films showed that the lesion was stable, no surgical intervention was undertaken.

Diagnosis in such settings is the most important clinical step. The treatment pathway for a Jefferson fracture differs radically from that of a C1 formation failure. Defects with smooth, contoured edges on imaging suggest a spondyloschisis, whereas sharp, irregular edges suggest a fracture. In one study, 44% of patients with congenital malformations had bilateral atlantoaxial lateral offset, a finding generally thought to result from a fracture. However, diagnosis of congenital malformations of the atlas may be made from conventional radiographs because they produce atlantoaxial lateral offsets of 1 to 2 mm, whereas Jefferson fractures produce offsets of more than 3 mm. C1 fracture and congenital anomalies are not mutually exclusive; CT scans and conventional radiographs are important for diagnosis. Other criteria to differentiate a C1 fracture from a congenital anomaly in the trauma setting have been suggested, including soft tissue swelling on CT scans, intact cortical margins, and the location of signal abnormality on MRI (ie, intrasosseous signal indicates fracture, and no signal or extrasosseous signal indicates anomaly). Differentiating between a chronic nonunion and a fibrous union or a failure of formation is best achieved with the clinical history. Radiographs may not show much difference. For a true chronic nonunion with ongoing motion and instability, bone can be remodeled about the nonunion. A fibrous union with relatively more stability may have less bone remodeling.

Congenital anomalies have been treated with symptomatic supportive measures alone, including analgesics and soft or rigid cervical collars. However, Jefferson fractures require external or internal rigid fixation for up to 16 weeks. Treatment algorithms should be modified to treat the most threatening of the patient’s injuries. Surgery for congenital anomalies should be considered only if they render the spine unstable.

The current patient illustrates the diagnostic challenge presented by congenital anomalies of the C1 vertebrae found incidentally after trauma. Radiographic findings and symptoms, such as transient paresis, transient parathesias, persistent pain, and dysphagia, could indicate fracture or congenital anomaly. Cervical instability is suggested by history, symptoms, and radiographic evidence of non-
physiologic motion between C1 and C2 and 3 mm or more lateral displacement of the lateral masses compared with the odontoid. However, the imaging findings in the current patient indicated that no acute fracture or instability existed and confirmed the presence of a congenital nonunion of the anterior and posterior arches of C1 with fibrous nonunion of the anterior arch.

Conclusion

Because treatment options for cervical spine injury range from invasive fusion to supportive measures based on the cause and extent of injury, determining the correct diagnosis is the most important step in management. Computed tomography and MRI are useful in assessing the post-trauma cervical spine because they help describe the defect’s character and distinguish acute and chronic lesions (ie, traumatic injury vs congenital anomaly). Although congenital anomalies are less common than fractures in the trauma setting, it is important to be cognizant of such possibilities.

References