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Fourth Cranial Nerve Palsy in a Collegiate Lacrosse Player: A Case Report

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Objective: To present the case of a National Collegiate Athletic Association Division I men's lacrosse athlete with fourth cranial nerve injury as the result of a minor traumatic blow.

Background: The athlete was struck on the right side of his head during a lacrosse game. On-field evaluation revealed no cervical spine involvement or loss of consciousness. He complained of headache and dizziness, with delayed reports of visual disturbance. Sideline visual acuity and cranial nerve screenings appeared within normal limits. Consultation with the team physician indicated that immediate referral to the emergency department was unnecessary.

Differential Diagnosis: Concussion, third cranial nerve palsy, fourth cranial nerve palsy.

Treatment: The certified athletic trainer safely removed the athlete from the playing field and monitored him on the sideline. After being seen by the team physician, the patient was referred to a neurologist, ophthalmologist, and finally a neuro-ophthalmologist before a definitive diagnosis was made. The palsy did not necessitate surgical intervention, resolving with conservative treatment. The athlete was able to return to full athletic ability at his preinjury level by 8 months postinjury.

Uniqueness: Superior oblique palsy as the result of fourth cranial nerve injury is the most frequent isolated cranial nerve palsy; however, these palsies are often underdiagnosed by health professionals. Such palsies are uncommon within the athletic realm, making timely diagnosis even less likely.

Conclusions: Cranial nerve palsy may present very subtly in patients. Therefore, on-field health care providers should be aware of the descriptions and types of compensations that signal nerve injury.

Key Words: trochlear nerve, neurologic injuries, visual disturbances

Cranial nerve (CN) examination is a routine component of the evaluation for any head injury. Three cranial nerves control eye motion: CN III (oculomotor), CN IV (trochlear), and CN VI (abducens). Injury to any of these nerves is rare in sport-related head injury; however, the location of the fourth cranial nerve makes it especially susceptible to injury with severe head trauma.[1] Unfortunately, CN IV palsies are underdiagnosed by hospital services.[2] The purposes of this report are to present the unique case of a CN IV injury in a collegiate lacrosse athlete and to review relevant clinical evaluation components that may assist certified athletic trainers in timely diagnosis of this injury.

CASE REPORT

A 19-year-old collegiate male lacrosse player (National Collegiate Athletic Association Division I) with no history of concussion was struck on the head just above his right ear by an opponent's elbow during a game. The athlete was wearing a helmet at the time of the blow, but his mouthguard appeared to have been knocked out by the blow. When I arrived on the field, the athlete was not moving but was fully conscious. Teammates who arrived at his side seconds after the blow stated that he never lost consciousness. The athlete was lying supine, with his mouthguard hanging out of the side of the mouth and the helmet chin strap pushed up on his chin. The athlete was not coherent upon initial questioning and could not say what had happened to him. He also could not say whether he initially had any neck pain. After approximately 30 seconds, he began responding coherently to my questioning, and I eliminated the possibility of neck injury through palpation and demonstration of full sensory and motor ability of all extremities. I helped the athlete into a sitting position and then a standing position in increments and walked off the field with him. Sideline evaluation methods included a clinical examination, symptoms checklist, and standard assessment of concussion.

The athlete initially complained of headache and dizziness but denied nausea or tinnitus. His pupils were equal and reactive to light and accommodation. His pulse rate was normal (56 beats/min). Cranial nerve screening revealed no obvious deficit. Initially, the athlete was not oriented to place or time of game and could not answer basic questions about events that happened in weeks before that day, earlier that day, or immediately after the concussive event. After approximately 20 minutes of sideline monitoring, he became oriented to his surroundings and began to slowly regain his memory of the events leading up to the injury. At this time, he was able to repeat strings of numbers in reverse order, recite the months of the year in reverse order, and recall 5 of 5 words 5 minutes after hearing them and 4 of 5 words after 10 minutes. He was able to recall events from several weeks ago and all events before the hit but was unable to recall the hit, how he was taken off of the field, or what occurred during the first 20 minutes postinjury. The athlete was now following the game and was reacting appropriately to events on the field. At this time, he complained of blurry vision and thought he had lost a contact lens. Inspection of his eyes revealed that both contact lenses were in place; however, the athlete continued to complain of blurry vision. He was able to see clearly through each individual eye with the other eye closed but experienced a type of double vision with both eyes

Table. Results of Tests of Planes of Vision

Time Postinjury, wk	Findings in Gaze Positions, Diopters				
	Primary[a]	Left	Right	Down	Right Head Tilt
5	10 (hypertropia[b])	8 (exotropia); 20 (hypertropia)	WNL	NA	25 (hypertropia)
15	6 (exotropia[c]); 4 (hypertropia)	4 (exotropia); 15 (hypertropia)	WNL	15 (hypertropia)	NA
22	2 (hyperphoria[d])	18 (hypertropia)	WNL	4 (hyperphoria)	NA

Abbreviations: WNL, within normal limits; NA, not available.

[a] Some measurements were taken more than once.

[b] Hypertropia is a misalignment of the eyes in which the visual axis of one eye is higher than that of the other. In this case, hypertropia was on the right.

[c] Exotropia is a misalignment of the eyes in which the eyes are deviated outward.

[d] Hyperphoria is the tendency of the eye to deviate vertically.

open. The athlete continued to complain of headache and dizziness. No immediate concussion grading scale was used in this case, as both the team physician and I use the Vienna concussion definitions.[3] I spoke with 2 team general medicine physicians on the phone to determine if the athlete should be referred to the emergency department that night, but neither physician felt that this case warranted immediate referral. However, recommendations were made for continued observation overnight by a roommate. At 10:00 PM on the day of the injury, the athlete checked in with me over the phone: he reported no increase in symptoms, but headache and visual disturbance continued.

COURSE OF TREATMENT

The patient was seen in the athletic training facility the following day by the team general medicine physician. He continued to complain of headache and blurry vision as well as some dizziness while walking that he attributed to the visual disturbance. He reported that walking down stairs and watching television were particularly difficult. The patient had no problems falling asleep or staying asleep and had been awakened twice during the night by his roommates without problems. He had also been able to eat 2 meals without difficulty. Single-eye visual acuity remained unaffected, but vision remained abnormal when the patient was looking straight ahead with both eyes open. The patient stated that tilting his head down, tucking his chin, and looking upward seemed to help. Upon reevaluation, the pupils initially dilated in response to light and then constricted. Cranial nerves and upper and lower dermatome, myotome, and reflex screening all appeared within normal limits. Because of the pupil abnormality and continued vision disturbances, a brain magnetic resonance imaging (MRI) scan was ordered. The test was interpreted as normal.

By day 2 postconcussion, the patient's headache had resolved completely, but his vision had not improved. He reported double vision, with one image slanted relative to the other. He had also developed ptosis of the right eyelid. Single-eye vision remained unaffected. At this point, the decision was made to refer the patient to both a neurologist and an ophthalmologist. The ophthalmology examination on day 5 postinjury revealed no problems with single-eye visual acuity. An MRI of the orbits was ordered at this time and was read as normal (ie, globes were within normal limits). Upon examination by the neurologist on day 6, mild ptosis and decreased adduction of the right eye were noted. Diplopia was confirmed by the red glass test (the patient was able to see the red light in the lower and more lateral positions when the left lower visual field was examined but not when the right lower field was examined). He had mild nystagmus on extreme lateral gaze in either direction but no evidence of abducens muscle weakness in either eye. The patient was diagnosed with a contusion or stretch injury to CN III (oculomotor nerve) as the result of the blow to the head. The neurologist's impression was that the injury was so microscopic that it would not have appeared on MRI. At this time, the patient was given a prognosis of full resolution of symptoms within 3 to 4 weeks and was advised to continue to use the eye normally. The neurologist stated that the concussion was resolved, and the patient was cleared to bike and resume noncontact lacrosse activities. The patient was also referred to a neuro-ophthalmologist to see if there were any exercises to retrain and reeducate deficient eye muscles to facilitate nerve healing. Unfortunately, the patient could not be seen by the neuro-ophthalmologist in our area until 5 weeks postinjury.

Beginning about day 7 postinjury, the patient developed a head posture that involved tucking the chin and tilting the head to the right. He felt this position helped him to focus on only one of the 2 planes of vision that he continued to see. Over the next 3 weeks, the patient reported subtle improvements in vision, but ptosis of the right eye continued, as did his report of seeing 2 distinct visual planes that remained tilted with respect to one another. Despite the neurologist's prognosis regarding symptom resolution, visual disturbances were not resolved by the 4-week mark. At this time, and when adopting the chin-tucked, head-tilted posture, he could focus when looking ahead and to the left for extended periods, but quick head motions or panning caused him to lose focus. Based on a battery of functional tests performed during the neuro-ophthalmologist appointment in week 5, the original diagnosis of CN III injury was rejected. The patient was now diagnosed with palsy of the right superior oblique muscle secondary to right CN IV palsy. This diagnosis was based on a positive Bielschowsky head-tilt test (a test to detect superior oblique muscle palsy), the double Maddox rod test (a test to detect and measure ocular torsion), and a test of planes of vision (using prisms). Cover testing was performed in various gaze positions (Table). The patient was given a prognosis of full resolution of symptoms within 3 to 4 months postinjury. The patient continued to be disqualified from contact lacrosse activities until his vision returned to normal. At this point, with vision still altered and only 4 games left in the lacrosse season, the patient chose to apply for a medical hardship waiver.

After returning to his home state for the summer, the patient had a follow-up appointment with a different

neuro-ophthalmologist 15 weeks postinjury. Based on prism diopters, the patient's double vision had resolved 50% from his previous appointment (Table). He continued to have no deviation when using the head-tilted, chin-tucked positioning. It was the opinion of the neuro-ophthalmologist that the full amount of vision that will return is normally achieved by 6 months postinjury. Because the patient's vision was continuing to improve, a decision as to whether surgery was warranted was deferred pending another follow-up visit in 6 weeks. By 5 months postinjury, the patient reported that he had no diplopia about 90% of the time (with most double vision occurring in extreme left gaze). Although he was still symptomatic in left gaze (Table), this diplopia did not have any functional implications. The neuro-ophthalmologist informed the patient and his family of the risk of returning to high-velocity sports with even intermittent diplopia, but the patient received medical clearance to return to competitive lacrosse. By 6 months postinjury he reported subjective normal vision. At that time, his risk of reinjury was equivalent to his preinjury risk. As an extra precaution, the patient was referred to a dentist for a custom-molded mouthguard. He was able to return to full competition during the fall 2008 season and competed fully in both the fall 2008 and spring 2009 competitive seasons with no recurrence of symptoms. At this time, the patient is 16 months postinjury and is back to his preinjury level of competition.

DISCUSSION

The fourth CN nucleus is located in the dorsal mesencephalon. The nerve exits the brain stem dorsally into the subarachnoid space, then courses around the brain to enter the cavernous sinus, superior orbital fissure, and orbit and to innervate the superior oblique muscle.[4,5] This muscle's actions include rotation in the vertical plane (depression and elevation of the eyeball) and rotation in the plane of the face (intorsion and extorsion of the eyeball). The force of the tendon's pull, therefore, has 2 components: a forward component that tends to pull the eyeball downward (depression) and a medial component that tends to rotate the top of the eyeball toward the nose (intorsion).[1,4,6–8]

Among the CNs, the trochlear nerve is the smallest nerve in terms of the number of axons it contains, has the greatest intracranial length, is the only CN to decussate before innervating its target, and is the only CN to exit from the dorsal aspect of the brain stem. The fourth CN enters the cavernous sinus, where it is joined by the other 2 extraocular nerves (CN III and CN VI), the internal carotid artery, and portions of the trigeminal nerve (CN V).[1,4,6–8] Contrecoup forces can compress the nerve against the rigid tentorium, which lies adjacent to the nerve for much of its course. Injury to the nerve can occur anywhere along its course from midbrain to orbit. A peripheral lesion reflects damage to the bundle of nerves, whereas a central lesion indicates damage to the trochlear nucleus or its fascicles within the brain stem. Peripheral lesions result in ipsilateral symptoms, and central lesions result in contralateral symptoms.[1,4,6–8] Our patient had a peripheral lesion, presenting with right-sided symptoms after right-sided head trauma.

The 2 most common causes of CN IV palsy are head trauma and vascular infarct.[2,9,10] In adults, approximately 40% of all isolated fourth nerve palsies are traumatic, 30% are idiopathic, 20% are due to vascular infarct, and 10% are due to tumor or aneurysm. Because of the large number of other neural structures that accompany CN IV as it travels through the cavernous sinus and superior orbital fissure, damage to this area rarely results in isolated fourth nerve palsy; more likely, associated palsy of CNs III and VI occurs.[1,4,6–8] In this case, however, superior oblique nerve palsy as the result of CN IV injury occurred without additional CN injury. Isolated injury to CN IV can be caused by any process that stretches or compresses the nerve.[1,4,6–9] Even relatively minor trauma can transiently stretch CN IV (by displacing the brain stem relative to the posterior clinoid process), but generally the trauma must be severe, with resultant loss of consciousness.[4] With trauma, patients usually report symptoms immediately after regaining consciousness. Patients with minor damage to CN IV complain of blurry vision; patients with more extensive damage notice frank diplopia and rotational (torsional) disturbances of the visual fields. Visual acuity is unaffected and pain is atypical.[2,4,10] In this case, the athlete had severe damage to CN IV (including vertical and torsional diplopia) as the result of relatively minor head trauma. The patient never lost consciousness and demonstrated no structural abnormalities on MRI of the brain or orbit.

Cranial nerve screenings by the athletic trainer on the sideline and by the team general medicine physician the day after the concussive blow both appeared within normal limits, although later diagnosis by a neuro-ophthalmologist revealed paralysis of the superior oblique muscle, innervated by CN IV. The patient did not report any visual disturbances until a full 20 minutes after initial injury, which coincided with the time that the athlete began to regain familiarity with and awareness of his surroundings. This is an important take-home point, as it may imply that visual acuity and ocular CN testing should be repeated in such athletes, the focus being on subtle noncongruence in eyes during various gazes. One should note, however, that paralysis of the superior oblique muscle is often not evident on screening; torsional deviation and diplopia can often only be measured by the double Maddox rod test.[10]

Visual disturbances are the result of paralysis of the superior oblique muscle, which prevents normal eye movement, and, specifically, weakness of downward eye movement with consequent vertical diplopia. The affected eye drifts upward relative to the normal eye as a result of the unopposed actions of the remaining extraocular muscles. The patient sees 2 visual fields (one from each eye) separated vertically. Diplopia is usually worse on downward gaze and gaze away from the side of the affected muscle and is especially problematic as the patient tries to read. To compensate for this, patients learn to tilt the head forward and tuck the chin in order to bring the fields back together.[4] Less commonly, trochlear nerve palsy also affects torsion (rotation) of the eyeball in the plane of the face. When onset is acute, hypertropia is usually most prominent in the field of gaze of the involved superior oblique muscle. In this patient, the right hypertropia that worsened with left gaze and right head tilt is consistent with right superior oblique palsy. Weakness of intorsion results in torsional diplopia, in which 2 different visual fields, tilted with respect to each other, are seen at the same time. To compensate for this, the patient typically presents with

a characteristic head tilt away from the affected side in order to fuse the 2 images into a single visual plane.[4] Interestingly, some patients develop a head tilt toward the side of the lesion, called a paradoxic head tilt, to create a wider separation of images, which allows the patient to suppress or ignore one image.[2,4,10,11] The head tilt occurs in approximately 70% of all patients, whereas the paradoxic tilt affects only 3% of patients.[5] A unique feature of this case is that the patient developed a same-sided (paradoxic) head tilt rather than the more common contralateral head tilt. Certified athletic trainers and other sports medicine professionals should be aware that these types of compensations can signal a CN injury.

Trochlear nerve palsy is traditionally diagnosed according to cause as acquired, congenital, or idiopathic. Diagnosis is confirmed by evaluating eye movements in all directions. As the patient's gaze is directed to specific areas, one eye appears slightly higher than the other (evaluation by the Bielschowsky head-tilt test). This misalignment improves or becomes worse with certain head positions.[12] The characteristic appearance of patients with CN IV palsies (head titled to one side, chin tucked in) indicates the diagnosis.[1,4,10–12] The magnitude of visual disturbance can also be evaluated more objectively using prisms. In one study[13] the mean vertical deviation of 20 patients (aged 19–70 years) with congenital or traumatic fourth CN palsy was 11 prism diopters. In this patient, the diplopia was both vertical and torsional, with left gaze being off by 20 prism diopters at 5 weeks postinjury. Unfortunately, as a result of the late diagnosis, the extent to which his gaze was affected during the first 5 weeks of symptoms is unknown. He had difficulty with reading, watching television, playing video games, and descending stairs. These difficulties decreased once he adopted the characteristic chin tuck and head tilt seen in patients with CN IV palsy.

Although most CN IV palsies are benign, patients with trauma-related palsy should undergo an MRI or computed tomography scan of the head to dismiss the possibility of a concurrent subarachnoid (or other traumatic) hemorrhage or brain lesion.[5] Once structural damage to the brain has been ruled out, few treatment options exist for CN IV palsy. When encountering sudden-onset isolated CN IV palsy, one should delay prescribing permanent prism glasses for at least 3 months in order to allow the palsy to recover. Prescribing temporary prisms may help to adjust the patient's visual field as the nerve heals, but the strength of these prisms will need to be changed as the condition resolves. The prisms do not aid in recovery but rather help treat the diplopia. However, while prisms may help to correct vertical diplopia, they cannot aid with torsional diplopia.[14] An alternative treatment is to patch one eye until the condition resolves. Most cases resolve within weeks to months, with nearly all cases completely recovering by 6 months. If double vision persists longer than 6 months, surgery to resect the inferior oblique muscle may be necessary to realign the eyes. When necessary, surgery generally produces excellent results.[15]

CONCLUSIONS

Persistent double vision in athletes who have suffered a concussion or head injury should be considered a red flag for ocular CN injury. Although CN IV palsy is certainly not a common occurrence in athletes, it is a potential result of any head injury or concussion because of the nerve's location in the brain and its susceptibility to trauma. The symptoms are fairly obvious if one is familiar with the condition, and early diagnosis by a certified athletic trainer or other sports medicine professional can prevent unnecessary diagnostic testing and psychological stress on the part of the patient and the family. Also, in addition to the implications of injury to the nerve itself, an undiagnosed defect in extraocular muscle function may markedly affect the individual's athletic performance. The characteristic appearance of patients with fourth nerve palsies (head titled to one side, chin tucked in) indicates the diagnosis, as does the location of the eye during gazes into various planes of vision. When a CN IV injury is suspected, the patient should be immediately referred to a neurologist or neuro-ophthalmologist, with documentation of pertinent evaluation findings.

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