Syringomyelia is a tubular cavitation in the spinal cord that contains fluid similar to cerebrospinal fluid. Although multiple causes are known, including spinal trauma, neoplasm, and inflammation associated with postoperative changes or meningitis, the most common cause is Arnold-Chiari malformation. Symptoms include sensory disturbances, gait ataxia, motor weakness, and sphincter disturbances that vary depending on size, spinal level, and syrinx location within the cord. Treatment includes inserting a subarachnoid drain to decompress the syrinx.

This article presents a patient with syringomyelia without Arnold-Chiari I malformation.

CASE REPORT

A 21-year-old Division I college football player presented with right arm paresthesia during a game after a direct hit to the right shoulder with lateral neck bending to the left. He reported immediate onset of complete numbness and paralysis in the entire right upper extremity, lasting only a few seconds.

Sideline evaluation exhibited no neck pain with normal cervical spine range of motion. The results of Spurling's testing were negative. Sensory examination revealed decreased sensation laterally in the right arm, forearm, and thumb. Strength was 5/5 to manual testing in all upper extremity muscle groups. The player was thought to have suffered a stinger.

Symptoms resolved and physical examination results returned to normal after approximately 15 minutes. He was cleared for team practice with no restrictions.

Four days later, the player was involved in a similar collision during practice, resulting in the subsequent return of symptoms. He did not report the injury until the end of practice. Right biceps and right deltoid strength was 4/5 to manual testing, and decreased sensation to light touch in the right thumb was noted. No cervical spine tenderness or motion loss was detected.

No other weakness or sensory abnormalities were present in the upper or lower extremities. Reflexes were symmetric, and no upper motor neuron findings were noted.

Plain radiographs of the cervical spine revealed no fracture or dislocation. Indomethacin was administered, and the patient was allowed to practice in non-contact drills only.

Despite these measures, physical examination results remained unchanged over the next 3 days. A solumedrol
dose pack was administered. Three days after receiving oral corticosteroids, some improvement in strength was noted. Thirteen days after initial injury, the patient continued to experience decreased sensation in the right thumb with 4+/5 right deltoid and biceps strength. He was subsequently referred to an orthopedic spine surgeon for further evaluation.

Magnetic resonance imaging (MRI) of the cervical spine revealed a large ovoid lesion in the spinal cord, extending from the C4-C5 interspace to the C5-C6 interspace. The lesion exhibited a signal similar to cerebrospinal fluid, with increased signal intensity on T2-weighted images and decreased signal intensity on T1-weighted images. The lesion was centrally located within the cord on both sagittal and axial images (Figures 1 and 2). No other remarkable findings were noted. Specifically, no evidence of an Arnold-Chiari malformation was noted.

The patient was referred for evaluation by a neurosurgeon. A thorough neurologic history was negative for any previous motor or sensory disturbances. Slowly improving brachial plexus neurapraxia with an incidental, asymptomatic syringomyelia was diagnosed. The neurosurgeon concluded that the stinger and syringomyelia were unrelated. In addition, the syrinx had been present for a long time.

Two additional neurosurgeons who work with professional football teams were consulted by telephone. The consensus from all three was that the syrinx posed a small but ultimately unknown risk and was not an absolute contraindication to playing football. If the patient accepted this risk, he would be allowed to play. He would require serial MRI examinations at 3-month intervals to evaluate the syrinx.

Seventeen days post-injury, the patient regained full strength and normal sensation, as revealed by physical examination. Due to the lack of symptoms, the patient resumed playing football.

**DISCUSSION**

In this case report, distinguishing the cause of the patient’s symptoms and determining the appropriate treatment were difficult. It is possible that a stinger and subsequent neurapraxia, commonly associated with high-impact collision sports, caused the symptoms. However, advanced imaging study results and syrinx location complicated matters. The patient experienced C5 and C6 weakness and C6 sensory changes after injury. As evident by MRI, C5-C6 was the exact location of the syrinx. It is unknown how long the syrinx had been present. Ultimately, it was decided that the player sustained a routine stinger, unrelated to an underlying, asymptomatic syrinx.

Few cases of syringomyelia in athletes have been reported in the literature. One case of syringomyelia in a college football player caused acute hemiparesis during weight lifting. The patient’s weakness resolved after 10 minutes, but left-sided paresthesias continued. Magnetic resonance imaging revealed a syrinx at C3-C6, and an Arnold-Chiari I malformation was also present. The authors hypothesized that a Valsalva maneuver during strenuous weight lifting caused the syrinx to become manifest by increasing pressure within the cerebrospinal fluid system.

In another case, MRI was used to diagnose cavitory dilation of the central canal at T4-T5 in a synchronized swimmer. The patient suffered from recurrent thoracic girdle pains; however, she also had an Arnold-Chiari I malformation. The patient avoided straining and holding her breath, and the pains resolved within 10 days. Follow-up MRI performed at 2 months revealed the cavitation had disappeared. The authors hypothesized that the combination of breath holding and hyper-extension of the spine while participating in the sport might have increased the patient’s intrathoracic and intracranial pressures, thereby causing the dilation.

The cause of syringomyelia in our patient remains unclear. Our case is unique because it does not include Arnold-Chiari I malformation, the most common cause of syringomyelia. Furthermore, the patient had no history of neoplasm, surgery, or meningitis. This raises the question of whether his participation in football could have played a role. It is thought that any trauma to the spinal arachnoid causing inflammation can impede the fluid flow from the spinal cord. Direct trauma to the cord parenchyma is not required. It is unknown whether the patient’s history of collisions produced multiple hyperflexion or hyper-extension episodes in the cervical spine and ultimately caused inflammation of the spinal arachnoid. The nature of football and the rigorous weight lifting that the sport requires can lead to severe straining. Valsalva maneuvers could also play a role in increasing intracranial pressure and fluid pressure within the cord.

One report presented three cases of central canal cavitation that was discovered on MRIs of patients without Arnold-Chiari malformations and were completely asymptomatic. The authors labeled this condition idiopathic localized hydromyelia. It is possible that our patient had a similar condition. Our patient elected to continue to play football and will be followed closely with serial MRIs.

**REFERENCES**