Late-onset spinal accessory nerve palsy after traffic accident: case report

Trafik kazası sonrasında geç gelişen spinal aksesuvar sinir lezyonu: Olgu sunumu

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An injury to the spinal accessory nerve is mostly reported after surgical procedures performed in the posterior triangle of the neck. In addition, it may be caused by fractures in the jugular foramina, traumas or skull base tumors. Clinically, paralysis of the trapezius muscle leads to weakness, downward rotation of the scapulae and falling down of the shoulder girdle. A 38-year-old male with left shoulder pain, scapular deviation and weakness in the left upper extremity, whose symptoms developed over a two-year period following a traffic accident, is presented herein. In the electromyography (EMG) study, partial spinal accessory nerve palsy was detected. The patient was treated conservatively for the nerve palsy since the time elapsed rendered surgical intervention inappropriate. We report a case in which spinal accessory nerve palsy developed two years after a traffic accident. Accessory nerve injury following a traffic accident is very uncommon.

Key Words: Late onset; spinal accessory nerve palsy; traffic accident.

The spinal accessory nerve (SAN) may be injured at any point along its course.¹ Because of its superficial location in the posterior cervical triangle, it is especially susceptible to damage from penetrating injuries. It may also be injured during operations such as lymph node biopsy or radical neck dissection.¹ Woodhall² has given an accurate description of the symptoms and findings that follow surgical injury to this nerve: the patient complains of generalized weakness in the affected shoulder girdle and arm, inability to abduct the shoulder above 90°, and a sensory disturbance that may vary from a pulling sensation in the region of the scar to aching in the shoulder and arm. The aching may radiate to the medial margin of the scapula and down the arm to the fingers, and is sometimes incapacitating. The superior one-third of the trapezius muscle on the affected side always atrophies, the shoulder sags, and power to elevate it is weak. The scapula rotates distally and laterally and flares slightly; its inferior angle is closer to the midline than is its superior angle. This position is accentuated when the arm is abducted; the flaring of the inferior angle disappears when the arm is raised anteriorly, in contrast to the usual deformity caused by paralysis of the serratus anterior.³

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CASE REPORT

A 38-year-old male with symptoms of left shoulder pain and weakness in the left upper extremity was admitted to our clinic two years following a traffic accident. Upon presentation to our clinic, the patient complained of pain at the base of his neck and left upper back, without upper extremity radiation. The pain was increased by sitting with his arm unsupported, by prolonged standing and overhead activities. He also complained of asymmetry of the shoulders and decreased range of motion. The physical examination demonstrated marked wasting of the trapezius muscle, and the lateral border of the left scapula was noted to appear more prominent or to wing with abduction, but not with forward flexion (Fig. 1). Active movements were restricted to 90° abduction and flexion due to pain. He had a full range of passive movements, the rotator cuff was intact and neurological examination of motor function of the left trapezius muscle was 2/5. Plain radiographs were normal. In the electromyography (EMG) study, partial spinal accessory nerve palsy (SANP) was detected. The patient was treated conservatively. After one year, repeated EMG study showed a delay in the motor potentials of the left SAN compared to the right side and the reference values of normal. In view of the time elapsed, surgical intervention was not appropriate in this patient, and he was thus referred for physical therapy. The goal of the treatment was to decrease pain both at rest and with functional activities and to increase left upper extremity function. Passive range of motion of the left glenohumeral joint was performed to maintain glenohumeral range of motion. The patient reported a decrease in pain over the following one month.

DISCUSSION

The accessory nerve is a “purely” motor nerve; it consists of a cranial root and spinal root. The cranial root arises from the caudal segment of the nucleus ambiguous and runs laterally toward the jugular foramen, where it unites with the spinal root. The spinal root originates from the nucleus of the SAN, which extends from C1-C6. The spinal root runs between the dorsal roots of the spinal nerves and the dentate ligament, upwards through the foramen magnum, where it meets the cranial root.[4] After exiting the cranium, the accessory nerve passes deep to the sternocleidomastoid and then passes under the trapezius muscle. It innervates these two muscles; the sternocleidomastoid rotates the head to the opposite side, and the trapezius stabilizes the scapula, elevates (shrugs) the shoulder, and assists with scapular adduction and arm abduction at the shoulder.[5] The diagnosis of SANP usually relies on a pertinent history and examination. Weakness and atrophy that begins after a surgical procedure or penetrating trauma is often the case. On examination, the affected shoulder is in a lower position (drooping) compared to the normal shoulder, trapezoidal atrophy is present, and some winging of the scapula may be evident at rest.[5,6] These are some of the more useful examination findings; therefore, the diagnosis can often be made with careful observation alone. There is weakness of shoulder shrug; however, the patient can often still shrug the shoulder, and some patients even have a symmetrical shrug because this movement is also performed by the levator scapula, which is innervated by the dorsal scapular nerve.[5] The trapezius assists in arm abduction above 90°; therefore, patients have trouble with this movement. When the patient windmills their arms in abduction overhead, incoordination of the affected scapula can be seen. Scapular winging also occurs, which unlike a serratus anterior palsy, does not persist when the arm and shoulder are protracted forward.[5,6] When the sternocleidomastoid muscle is weak or atrophic, skull base and foramen magnum pathology should be excluded with magnetic resonance imaging (MRI). Electrodiagnostic testing confirms injury to the SAN, and can help evaluate partial injuries, or those with early reinnervation.[7] Nevertheless, some partial injuries without denervation may be difficult to diagnosis. Because the SAN is superficial and readily exposed, early surgical exploration and repair should be considered, especially with iatrogenic or sharp, lacerating injuries.[1,7] Alternatively, when nerve continuity is likely, or if partial function is present, it may be prudent to observe these patients for 3-6 months with serial electrodiagnostic tests, and explore those patients who fail to recover. Transected nerves should be repaired directly, or with an interposition nerve graft obtained from the greater auricular or sural nerve. If the nerve is not found transected and positive nerve action potentials are present, then an external neurolysis should be performed.[1,8,9] For patients who do not have a viable proximal accessory nerve stump to repair (e.g., after it was removed with a

Fig. 1. Image showing winging of the left scapula.
skull base tumor), a split thickness nerve transfer from the hypoglossal to the SAN should be considered. For chronic palsies (older than 1-2 years), where nerve surgery is no longer an option, one may undergo a tendon transfer where the levator scapulae and rhomboids are advanced from under the scapula to over the margin of the scapula.[7,10-12] Few cases of SANP secondary to stretch or traction injury have been reported in the literature. Two separate cases were reported to follow lifting heavy objects. To our knowledge, there is one case report in the English literature of SANP one year after a traffic accident.

In conclusion, there have been many causes of SANP reported in the literature. We found one case report about SANP following whiplash injury. We report the second case of SANP after a traffic accident, this case occurring two years later. Lesions of the SAN can be identified and differentiated from other clinical entities by careful history and physical examination. The treatment of SANP should be comprehensive and begin as early as possible. A high index of suspicion for SANP should be present following traction-type injuries to the cervical spine. Early diagnosis and treatment of SANP may lead to more effective pain relief and better functional outcome.

REFERENCES

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