J Shoulder Elbow Surg (2009) 18, e15-e17

ELSEVIER

Journal of Shoulder and Elbow Surgery

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Spinal accessory nerve injury after rhytidectomy (face lift): A case report

Peter J. Millett, MD, MSc[a,*], Alex Romero, MD[b], Sepp Braun, MD[a]

[a]*Steadman-Hawkins Research Foundation, Vail, CO*
[b]*University of Illinois at Chicago, Chicago, IL*

Injury to the spinal accessory nerve can be a disabling disorder.[6,7] Patients with a spinal accessory nerve injury may have pain, paresthesias, and motor weakness that may result in scapular winging. The nerve can be injured by open, penetrating, or closed trauma; however, the most common cause is iatrogenic.[6,7] This is most commonly encountered after procedures in the posterior triangle of the neck such as radical neck dissection, lymph node dissection, or biopsy.[2] It has been reported on a less frequent basis with procedures such as carotid endarterectomy and abscess drainage.[1,7] We report the case of a 66-year-old woman who developed iatrogenic, postsurgical injury to the spinal accessory nerve after rhytidectomy (face-lift), which subsequently resulted in a painful weak shoulder girdle. We believe that it is important for upper extremity surgeons to be aware of this as a potential cause for scapular winging.

Case report

The patient is a 66-year-old female who presented to our clinic with a 3-month history of left shoulder pain and weakness. The patient noted that the symptoms began immediately after having a face-lift surgery performed at an outside facility. She denied any locking, catching, or instability. She did have occasional paresthesias radiating down the involved extremity consisting of a tingling sensation, but denied any significant numbness. She had started physical therapy 2 weeks prior to presentation, but had not yet noticed any improvement. Her past medical history was only significant for the face lift, noted above. There were no other confounding factors such as cervical spine disease, neuropathy, or diabetes.

On physical examination, the patient had atrophy of the trapezius muscle with shoulder ptosis (Figure 1) and profound scapular winging with dyskinesis. She did have limited active but full passive range of motion. Her active forward flexion was only to 100°, and she had only 80° of total abduction (Figure 2). Provocative maneuvers for intra-articular/subacromial pathology were normal. She did have global weakness of her upper extremity that was secondary to pain. Most notably was the patient's weakness to abduction and forward flexion. The initial working diagnosis was a spinal accessory nerve injury. She was asked to continue physical therapy and to obtain electromyography (EMG) for further evaluation of the severity of nerve injury.

The patient returned to the clinic after 6 weeks of therapy and having obtained an EMG. Subjectively, her pain and weakness had improved, and on physical examination, she had improvement in glenohumeral range of motion. Strength was near normal in all muscle groups; however, the patient continued to have scapular winging. EMG demonstrated spinal accessory nerve neuropathy with severe chronic degeneration, with reinnervation of the superior pole of the trapezius. The findings were consistent with a neurapraxia. She was encouraged to continue with her physical therapy regimen with expectant observation. The patient's symptoms completely resolved and she had full function of the shoulder girdle within 12 months of the injury.

Discussion

The trapezius originates along the ligamentum nuchae above C7 and from the spinous processes from C7–T12.[7] It has multiple insertion sites with the upper portion inserting into the posterior border of the lateral 3rd of the clavicle, the

*Reprint requests: Peter J. Millett, MD, MSc, Steadman Hawkins Research Foundation, Attn: Clinical Research, 181 West Meadow Drive, Suite 1000, Vail, CO 81657.

E-mail address: drmillett@steadman-hawkins.com (P.J. Millett).

1058-2746/2009/$36.00 - see front matter
doi:10.1016/j.jse.2009.01.012

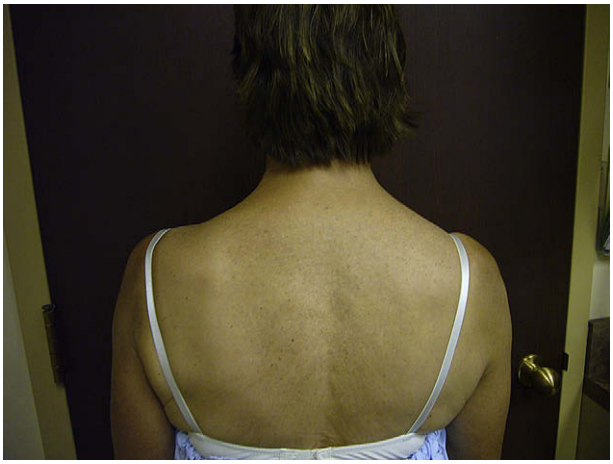

Figure 1 Posterior view of patient at rest demonstrating trapezius atrophy and shoulder ptosis but no scapular winging.

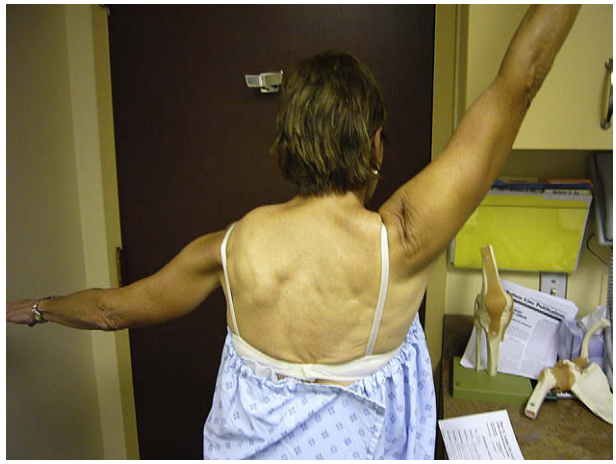

Figure 2 Patient attempting active abduction and, consequently, scapular winging.

intermediate portion inserting into the medial acromion and scapular spine and the inferior portion into the base of the scapular spine.[7] The trapezius receives motor innervation primarily from the spinal accessory nerve, but variations in innervation have been described, most commonly being motor contributions from the 3rd and 4th cervical nerve roots.[5] The trapezius muscle is the main suspensory muscle of the shoulder girdle and integral component of scapulohumeral motion.[6,7] It acts in conjunction with other muscles of the upper extremity and scapula to provide the coordinated motion of the glenohumeral joint.[6,7] The trapezius is the largest of the scapulothoracic muscles and acts to elevate, retract, and rotate the scapula. Each of these motions has contributions from different parts of the muscle.[7] The upper portion acts to elevate, intermediate fibers act to retract, and inferior fibers rotate the inferior angle of the scapula medially.[7]

The spinal accessory nerve (11th cranial nerve) is formed from a cranial and a spinal root.[2,5] The accessory nerve exits the jugular foramen with the vagus nerve and the internal jugular vein. It descends obliquely through the anterior triangle, medial to the styloid process, stylohyoid, and digastrics muscles. It exits the anterior triangle by perforating through the sternocleidomastoid, for which it is the main innervation.[2,5] The spinal accessory nerve then exits the posterior border of the sternocleidomastoid usually 7–9 cm from the clavicle and passes posteriorly and inferiorly across the posterior triangle. It exits deep to the investing layer of the deep cervical fascia on the levator scapulae but separated from it by the prevertebral layer of deep cervical fascia and adipose tissue. It is relatively superficial in this position, especially more proximally, and is easily damaged during neck surgery.[2] Considerable variations of its course within the posterior cervical triangle and of its cervical root contributions have been documented.

Patients with injury to the spinal accessory nerve present with shoulder pain and trapezius muscle palsy that subsequently results in drooping of the shoulder girdle inferior and laterally along with scapular winging.[7] Loss of abduction of the arm is commonly due to the malposition of the scapula, and it is not uncommon for patients to have multiple painful sequelae as a result, including impingement symptoms and adhesive capsulitis.[7] Diagnosis is confirmed and the level of injury assessed with the use of EMG and nerve conduction studies.

Iatrogenic injury is the most common cause of spinal accessory nerve dysfunction.[1,7] Because of its superficial position in the posterior triangle of the neck, it is very susceptible to injury.[2,5] Injury to the nerve is seen most commonly following radical neck dissections, cervical lymph node biopsy, and excision of masses.[2] Injury to the spinal accessory nerve after a face-lift procedure has only been described in 4 patients (none of which are in the orthopaedic literature).[1] Blackwell et al[1] presented 2 cases, 1 in whom shoulder pain was present immediately after surgery and weakness developed early. That patient did not seek orthopaedic treatment until after 1 month of persistent symptoms. The patient was managed nonoperatively, and although nerve function improved over serial EMG's, no clinical improvement was noted. The second patient failed conservative management and elected for surgical exploration, which revealed a 75% transection with neuroma formation. The neuroma was resected and the nerve was repaired. The patient had return of function, but pain persisted.[1] Our patient was followed conservatively and had return of full function and resolution of pain.

Loss of spinal accessory motor nerve function due to neurapraxia should be managed conservatively. Patients often have pain secondary to loss of the ability to suspend the shoulder girdle appropriately. Therapy should be guided toward relieving the weight of the arm from the shoulder girdle. Strengthening of the remaining scapular stabilizers, prevention of trapezius stretch/lengthening, and maintaining full range of motion of the shoulder girdle are important to good function after the nerve recovers.[4] Trapezius function has not improved and the patient still has severe

symptoms after 12 months, it seems unlikely that there will be any further recovery. Furthermore, active patients with long-standing trapezius palsy typically do poorly with nonoperative management alone.[7] For those nerves that do not recover with conservative management alone, one can consider surgical management to relieve pain and restore some function.[7]

Surgical management of trapezius palsy depends on multiple factors. Historically, static soft tissue stabilizing procedures were attempted; however, these routinely failed secondary to stretching of the soft tissue sling construct. Today, dynamic stabilization, as described by Eden and Lange, is the most common palliative procedure.[6,7] This is accomplished by triple transfer of the levator scapulae, rhomboideus major, and rhomboideus minor muscles laterally on the scapula.[6,7] Intermediate results of this procedure, with mean follow-up of 7.4 years, has shown satisfactory to excellent outcomes in 19 of 22 patients.[7] Teboul et al[6] offered an algorithm for the management of trapezius palsy based largely upon the time since and the type of injury. Patients with symptoms less than 12 months and no sign of clinical or electrical recovery by 3 months should undergo nerve exploration with neurolysis, repair, or grafting. Those with symptoms for 12-20 months should have the nerve explored. If at the time of exploration the muscle has a contractile response to intra-operative stimulation, the surgeon should attempt neurolysis or grafting. If there is no response and the patient is less than 50 years of age, nerve surgery is still recommended; others however, only recommend nerve surgery up to 12 months.[3] The Eden-Lange procedure is recommended for those patients who have symptoms for greater than 20 months in a spontaneous palsy that remains symptomatic, iatrogenic palsy after radical neck dissection, or a failed microsurgical repair.[6,7] In Teboul's series, patients with spontaneous nerve palsy who underwent nerve surgery all did poorly. Teboul et al recommend proceeding to palliative surgery in the case of an unresolved spontaneous nerve palsy.[6]

Conclusion

Spinal accessory nerve palsy is a rare but potentially debilitating complication after rhytidectomy. It most commonly results in pain, scapular winging, and shoulder dysfunction, and the diagnosis is confirmed with EMG/NCV studies. Treatment includes physical therapy, possible nerve exploration with neurolysis, repair or grafting, or palliative surgical management (Eden-Lange muscle transfers).

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