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Spinal Accessory Nerve Palsy: Evaluation of Four Atypical Cases

Spinal Aksesuar Sinir Felci: Dört Atipik Hastanın Değerlendirilmesi

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Summary

Spinal accessory nerve palsy commonly occurs as a result of iatrogenic injury, usually during lymph node excision in the posterior cervical triangle. Numerous alternative etiologies are described. Trauma is the most common cause of non-iatrogenic palsy. Sometimes, the cause of the palsy cannot be identified. Here, we report four atypical cases of isolated spinal accessory nerve palsy leading to trapezius muscle dysfunction. In one patient, accessory nerve palsy developed after reduction mammaplasty and abdominoplasty surgery (Case 1). One of them experienced a stretch injury due to weight lifting (Case 2). In one patient, a clear reason could not be identified (Case 3). In a 12-year-old girl, accessory nerve palsy was caused by her heavy school bag (Case 4). All three segments of trapezius muscle were involved in all cases, except for the case 1 who had only the upper segment involvement. The patients were followed clinically and electrophysiologically for 24-30 months. Both cases 1 and 2 showed complete recovery within six months, whereas cases 3 and 4 showed incomplete recovery. The clinical similarities and differences, and also the neurophysiological findings of cases are discussed in this paper. Our long-term follow-up findings may help us to understand the mechanism and the natural course of the accessory nerve palsies. *Turk J Phys Med Rehab 2011;57:248-52.*

Key Words: Accessory nerve palsy, trapezius muscle, stretch injury, nerve conduction, neuropathic pain

Özet

Spinal aksesuar sinir felci genellikle posterior servikal lenf nodu eksizyonu sırasında iatrojenik olarak ortaya çıkmaktadır. Birçok farklı etiyolojiler tanımlanmıştır. Travma, iatrojenik olmayan aksesuar sinir felçlerinin en sık sebebidir. Bazen de felcin nedeni belirlenemeyebilir. Bu çalışmada, trapez kası disfonksiyonu olan dört atipik izole aksesuar sinir felci olgusu sunulmaktadır. Bir olguda, aksesuar sinir felci göğüs küçültme ve abdominoplasti operasyonundan sonra ortaya çıkmıştı (Olgu 1). Bir olgu ise ağır kaldırmaya bağlı gerilme yaralanmasıydı (Olgu 2). Bir hastada ise net bir sebep ortaya konamadı (Olgu 3). On iki yaşında bir kız çocuğunda ise aksesuar sinir felci, ağır sırt çantasına bağlı olarak ortaya çıkmıştı (Olgu 4). Üç hastada trapez kasının her üç segmenti de etkilenmişken bir hastada sadece üst segment etkilenmişti. Hastalar klinik ve elektrofizyolojik olarak 24-30 ay süresince izlendiler. Birinci ve 2. olgular altı ay içinde tam düzelme gösterirken 3 ve 4. olgular kısmi düzelme gösterdi. Burada, olguların klinik benzerlikleri, farklılıkları ve ayrıca nörofizyolojik özellikleri tartışılmıştır. Uzun süreli takip bulgularımız, aksesuar sinir felcinin mekanizmasını ve doğal seyrini daha iyi anlamamıza yardım edebilecektir. *Türk Fiz Tıp Rehab Derg 2011;57:248-52.*

Anahtar Kelimeler: Aksesuar sinir felci, trapez kası, gerilme yaralanması, sinir iletimi, nöropatik ağrı

Introduction

Isolated accessory nerve palsy is a rare focal neuropathy. The majority of spinal accessory nerve lesions occur after lymph node biopsy and tumor resection in the posterior cervical triangle (1-7). Other etiologies include blunt trauma, irradiation, penetrating injury, vascular ectasia, carotid endarterectomy, cardiac surgery, laceration, compression, strenuous weight lifting, whiplash, spontaneous palsy (idiopathic) due to plexitis, and other more rare occasions (3-5,8-22).

The spinal accessory nerve innervates the sternocleidomastoid and trapezius muscles. In majority of cases with accessory nerve palsy, the trapezius is the only muscle involved, because most injuries occur below the level of supply to the sternocleidomastoid muscle (3,4,23).

Trapezius muscle consists of three segments. The upper segment is the scapular elevator and rotator, the middle segment is scapular

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adductor/retractor, and the lower segment is the scapular depressor. Paralysis of the trapezius muscle causes scapular instability, pain and loss of limb mobility.

Here, we report four unusual cases of isolated accessory nerve palsy and paralysis of the trapezius muscle. The clinical and electrophysiological features in these rare cases are discussed.

Case 1

A 37-year-old woman complained of weakness in her right shoulder. The patient had reduction mammaplasty and abdominoplasty surgery which lasted 5.5 hrs. Following the surgery, she could not lift her shoulder above the horizontal plane. Physical examination revealed drooping and anterior shifting of her right shoulder. Active shoulder abduction was limited to 80 degrees. Muscle strength tests showed moderate weakness of all three segments of the right trapezius muscle (Figure 1a, b). There was no weakness of the sternocleidomastoid, shoulder girdle or extremity muscles.

Electromyography (EMG) study was performed three weeks after the onset of the symptoms. The accessory nerve was stimulated with a bipolar surface stimulator 1-2 cm posterior to the posterior border of the sternocleidomastoid muscle, mid-way between the mastoid process and the suprasternal notch. Recordings were made from all three segments of the trapezius muscle with a bar electrode with surface discs 2.5 cm apart (24). Compound muscle action potentials (CMAP) were recorded from the upper, middle and lower segments of the right trapezius muscle. All segments had normal latencies, but decreased amplitudes compared to the left side (Table 1). Axillary nerve, suprascapular nerve, long thoracic nerve and extremity peripheral nerve conduction studies were normal.

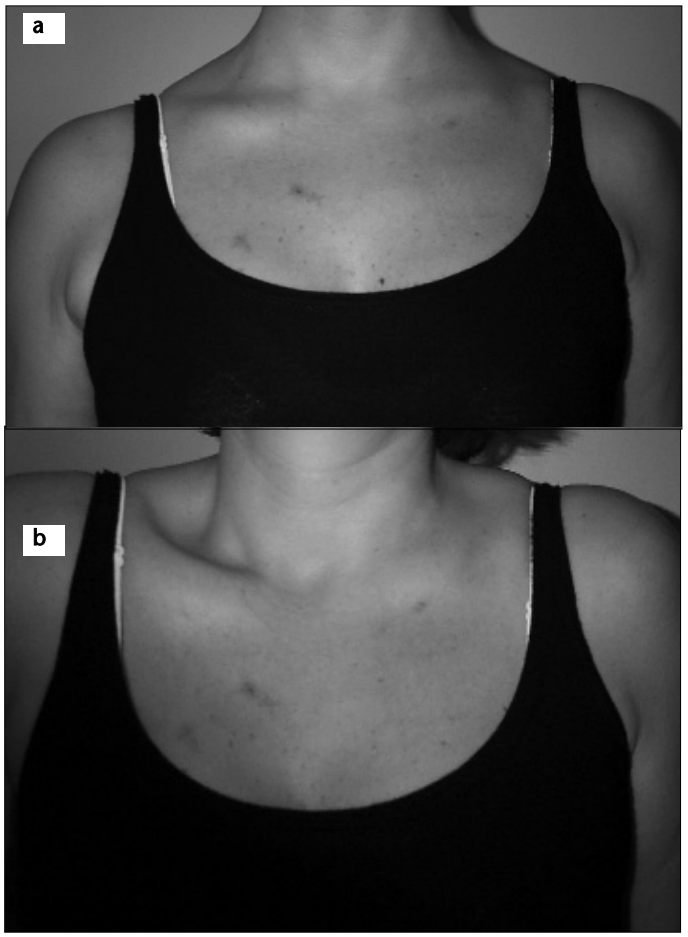

Figure 1. **a)** The right shoulder is slightly drooped due to weakness of the upper segment of the right trapezius muscle. **b)** When the patient attempted to elevate her shoulders, the supraclavicular notch was visible on the right side. Weakness of the middle and posterior segments of the trapezius muscle resulted in inability to perform sufficient scapula retraction.

Needle EMG revealed abnormal spontaneous activity (positive sharp waves-PSW, fibrillation potentials) and markedly reduced recruitment of motor units from all segments of the trapezius muscle. The needle EMG was normal for the sternocleidomastoid, rhomboid, infraspinatus, supraspinatus, serratus anterior and extremity muscles.

A home exercise program was given to improve muscle strength. Trapezius muscle atrophy did not recur. A rapid improvement was observed in the first three months. Four months later, muscle strength returned to normal and the patient was able to easily do full shoulder abduction. Repeat accessory nerve conduction studies and needle EMG of the trapezius muscle were performed and no pathology was detected. There were no further problems reported during the 28-month follow-up period.

Case 2

A 26-year-old man presented with complaints of weakness and atrophy of his right shoulder. One month before, he was bringing down a heavy object (70 kg). The object was suspended for 10 sec. One day later, the patient felt pain in his right shoulder. The patient noticed that he got tired quickly and had difficulty in using his right arm. This condition was exacerbated by his job, which required 10 hr right-handed ironing every day. One week later, he noted that he could not execute some right shoulder movements.

Inspection at our clinic revealed mild atrophy of the upper fibers of the trapezius. His shoulder drooped and he could not abduct his shoulder higher than the horizontal plane. We did not find weakness or atrophy of the sternocleidomastoid muscle. The amplitude of recorded CMAPs from stimulation of the accessory nerve was reduced in the upper segment of the right trapezius compared to the left side. CMAPs with normal amplitude and latency were recorded from the middle and lower segments of the trapezius (Table 1).

EMG examination of the upper segment of the trapezius muscle revealed reduced recruitment of motor units, increased polyphasia, and occurrence of mild PSWs and fibrillation potentials. The middle and the lower segments of the trapezius muscle were normal. A home-based daily exercise program was recommended in order to increase shoulder strength and to restore function. At one-month follow-up, he was able to do complete abduction of the right shoulder with a little difficulty. At the end of the fifth month, his shoulder strength improved to nearly normal levels. The patient was satisfied with his condition during the two-year follow-up period.

Case 3

A 27-year-old man complained of left shoulder pain for two months and wasting of his left shoulder girdle muscles. The patient noticed difficulty in shoulder movement one month after the onset of pain. Medical examination revealed left shoulder drooping and atrophy of all segments of the trapezius muscle. Atrophy was most visible in the upper segment (Figure 2). Shoulder elevation and retraction strength were moderately reduced. Neurological examination was otherwise normal.

The amplitudes of CMAPs recorded from the upper and the lower segments of the right trapezius muscle were decreased. A CMAP was not obtained from the lower segment. There were no significant

differences in latencies. EMG examination of all three segments of the trapezius muscle showed reduced motor unit recruitment, increased polyphasia, and mild PSWs and fibrillation potentials. Needle EMG of the sternocleidomastoid and other shoulder girdle muscles were normal. Magnetic resonance imaging (MRI) was used to detect lesions that compress or otherwise affect the accessory nerve. MRI revealed no abnormality.

When interviewed, the patient reported riding a crowded public bus for a 4-hour commute to his job. During the standing commute, he hung on to the ceiling handle. The patient worked in a textile factory and used a sewing machine for 8-hour every day. We advised him not to hold the ceiling handle, but rather hold the side handles, alternating arms frequently. A home exercise program was designed to increase strength of the trapezius and other shoulder girdle muscles. Changing the job was not an option for the patient.

Figure 2. Atrophy of the upper segment of the left trapezius muscle and asymmetry of the scapulae.

The patient was examined every three months. There was no improvement in his clinical situation till the ninth month. The patient could elevate his arm above the horizontal plane after nine months. In one year, a slight regression of atrophy was observed in the upper segment of the trapezius muscle. After 16 months of the onset of symptoms, shoulder elevation strength had significantly increased. Atrophy still remained but to a lesser degree. Electrophysiological tests showed that the amplitudes of CMAPs from both upper and middle segments of the trapezius muscle were increased, but still lower than those in the right side. CMAP could not be obtained from the lower segment. Needle EMG of the trapezius muscle showed that there were no PSWs or fibrillation potentials for the upper and middle segments. In addition, there were abundant nascent potentials (reinnervation potentials) with reduced amplitude and increased polyphasia. Reduced recruitment of motor units was observed in the lower segment. There was not any denervation or reinnervation detected. Ultrasonographic examination of the neck region showed no abnormality. After 24 months of the onset of palsy, the patient had nearly normal muscle strength, but the atrophy of the upper trapezius muscle was still visible.

Case 4

A 12-year-old girl was admitted to our clinic because of weakness and atrophy of the left shoulder. Her school bag (3-5 kg) crossed over her left shoulder when she was 6-8 years old. When she experienced pain in her left shoulder, she started to carry a bag with two straps, which evenly distributed the weight to the both shoulders. One month after changing the bag, the pain disappeared. Six months after the onset of pain, her mother noticed wasting of her daughter's shoulder muscles.

Medical examination found that all segments of the left trapezius muscle were atrophic, especially the upper segment (Figure 3). Shoulder elevation, scapular retraction and depression strength were

Table 1. The latencies and amplitudes of elicited CMAPs from bilateral segments of the trapezius muscle.

* Unobtainable	Latency (ms) Normal side	Latency (ms) Involved side	Amplitude (mV) Normal side	Amplitude (mV) Involved side	Amplitude Reduction (%)
CASE 1					
Upper	1.79	1.86	8.3	4.4	47
Middle	3.1	3.0	7.0	3.4	51
Lower	4.0	4.10	3.1	1.8	42
CASE 2					
Upper	1.83	1.87	9.3	2.9	69
Middle	3.50	3.70	6.1	5.2	14
Lower	5.10	5.00	2.6	3.0	15
CASE 3					
Upper	1.71	1.74	8.8	2.0	77
Middle	4.10	4.30	4.5	2.5	44
Lower	4.80	*	1.2	*	100
CASE 4					
Upper	1.38	3.10	7.2	2.2	69
Middle	2.90	8.10	6.0	0.6	90
Lower	3.70	8.50	1.8	0.4	77

moderately decreased. The patient could abduct her shoulder with some difficulty. The sternocleidomastoid muscle was normal. In addition, the patient could rotate her forearm 360 degrees and touch her thumb to her forearm easily. A mild dorsal kyphosis and scoliosis were evident.

The amplitudes of the recorded CMAPs with accessory nerve stimulation were reduced. Latencies were longer on the left side compared to the right side (Table 1). Needle EMG of the trapezius muscle showed no abnormal spontaneous activity or motor unit abnormality. MRI of her neck region demonstrated no abnormalities.

A home exercise program was recommended. The patient was examined after 30 months. Muscle mass of the left trapezius muscle was slightly increased. We found a small increase in scoliosis curvature of the spine. Except for the atrophic appearance of the trapezius muscle, there was no detectable pathology on ultrasonography (USG) of the neck region.

Discussion

The majority of accessory nerve palsies occur after minor cervical surgery or trauma. There was no difficulty in diagnosing these patients. Although different etiologic factors have been reported, sometimes, a clear reason for non-iatrogenic accessory nerve palsy cannot be found. Donner and Kline (4) evaluated 83 cases of accessory nerve palsy. Fifty nine palsy cases were caused by iatrogenic lesion and the other cases were classified as non-iatrogenic (trauma, etc.). In another series of 23 cases with accessory nerve palsies, Berry et al. (3) reported 11 iatrogenic cases and 10 traumatic cases and two spontaneous cases.

Sternocleidomastoid muscle involvement is seen more frequently in non-iatrogenic accessory nerve palsy compared to iatrogenic palsy. The sternocleidomastoid muscle was normal in all four patients. All segments of the trapezius muscle were affected in the three cases (1, 2, 4). Only the upper segment was affected in one case (3). A similar case of a patient with only upper segment paresis due to accessory nerve palsy has been reported (13). That case was similar to our case because both patients carried heavy weight and the sternocleidomastoid muscle was not affected.

Carrying heavy weight mostly works the upper segment of the trapezius muscle. Excessive weight pulled the shoulder down and presumably produced a rapid increase in tissue tension along the course of accessory nerve in the neck and shoulder. This stretch force may have caused accessory nerve injury by compromising the intraneural microvascular flow and subsequently by axon rupture. Similar cases have been reported in the literature (13,14,19).

Figure 3. Severe atrophy of the trapezius was most visible when the patient tried to abduct her left shoulder.

In the intraoperative electrophysiological study of Kierner et al. (25), it was found that the upper segment of the trapezius muscle is innervated by a fine single branch arising from the spinal accessory nerve in the posterior triangle of the neck. Our case with only upper segment pathology supports their finding that cases with only upper segment involvement are possible.

Spinal accessory nerve palsy can occur if the nerve is injured or cut during an operation. The only reported case is case 1: spinal accessory nerve palsy caused by abdominoplasty and reduction mammaplasty. Surgeons reported that they had never seen such a case. They had not performed any action that would lead to accessory nerve damage. The accessory nerve palsy developed post-operatively; therefore, this case was not idiopathic. The palsy might be due to compression of the accessory nerve from lying down for a long time during the operation. Stretch injury due to prolonged positioning during surgery has also been proposed as another mechanism of spinal accessory nerve injuries (26).

Although case 3 may be accepted as an idiopathic case, shoulder overuse was also evident, as in case 2. Most days of the week, the patient in case 3 commuted 4 hr on a bus. Crowded bus rides resulted in his prolonged shoulder abduction from holding a ceiling handle. In addition, this patient sewed 8-hour day at his job. Existing similar shoulder activities in cases 2 and 3 suggested that repetitive overuse of the shoulder may have prompted injury of the spinal accessory nerve. Given the unusually prolonged shoulder abduction in case 3, it may be considered idiopathic.

Case 4 is the youngest reported case of a patient with isolated accessory nerve palsy not caused by iatrogenic reasons. Her shoulder bag put all the weight on her left shoulder. A combination of head tilting to the opposite side and weight pulling the shoulder down may have resulted in a stretch injury to the accessory nerve. A similar accessory nerve palsy case was reported in a mail carrier (4). Another possible explanation may be development of a compression neuropathy caused by shoulder strap of the bag.

Our young patient had marked atrophy of the trapezius and prolonged distal latencies in conduction studies. Stable motor units were detected by needle EMG. Muscle mass did not significantly increase after 30 months of a regimented home exercise program. Especially prolonged latencies on nerve conduction studies suggest that newly formed nerve fibers emerging from injured site via axonal regrowth have slower conduction velocities than normal nerve fibers. All these findings demonstrated that the patient had severe accessory nerve palsy and recovered incompletely. There is no literature on hyperlaxity and accessory nerve palsy that was observed in our patient.

Pathological changes in spinal accessory nerve tissue can be visualized by USG (27). USG examination of cases 3 and 4 did not show soft tissue pathology, although healing was incomplete in the two cases.

Electrophysiology is used to characterize the nature of accessory nerve palsy. Electrophysiological analysis of cases 1 and 2 was compatible with a conduction block and mild degree of axonal involvement. Case 3 had mainly axonal involvement. The physical improvement in these cases was correlated with the electrophysiological findings. Accessory nerve palsy is generally associated with a good prognosis irrespective of needle EMG findings (5). Nerve conduction studies may be more important for the prognosis than needle EMG analysis and should be

performed separately on each segment of the trapezius muscle (24). Studies have shown that amplitude analysis of CMAPs recorded from the middle and inferior segments of the trapezius muscle increases the diagnostic sensitivity of electrophysiological testings (28). Even if recorded CMAPs are low in amplitude for each segment of the trapezius muscle, this indicates good prognosis due to preserved nerve continuity. Although accessory nerve conduction has been tested with magnetic stimulation (29), this procedure is not used in clinical practice.

Accessory nerve palsies usually have good prognosis. In 56 cases, Friedenberg et al. (5) found that 45% of patients had a good prognosis. Donner and Kline (4) also showed a high rate of recovery with conservative and surgical therapy. Impaired shoulder abduction is the most deleterious handicap in cases with accessory nerve palsy. This mostly originates from the palsy of the upper segment which externally rotates the scapulae and enables shoulder abduction over horizontal level. However, the upper segment is the closest segment to the injured nerve site and reinnervation is more likely due to a nearby nerve segment. Reinnervation of the lower trapezius may not occur because of the long distance between the injured site of accessory nerve and the muscle. In these cases, shoulder function may be recovered mainly by reinnervation of the upper segment of the trapezius muscle, as in case 3. To recover shoulder abduction, an exercise program should be designed to strengthen the other shoulder girdle muscles. Literature data also support our findings. High recovery potential has been shown after accessory nerve injuries in the recent literature (30,31). Akgun et al. (30), showed that conservative management is effective even in late diagnosed spinal accessory nerve injuries.

Conclusion

In patients with painful shoulder and slow trapezius muscle paralysis, accessory nerve pathology can remain unnoticed or misdiagnosed as shoulder joint or cervical root pathology. Electrophysiological studies should be performed on questionable cases in order to avoid misdiagnosis. The upper segment of the trapezius muscle can be affected independent of the middle and lower segments. Nerve conduction studies and needle EMG should be performed on the three individual segments of the trapezius muscle. Follow-up is important in accessory nerve injuries. In cases with preserved nerve continuity a good prognosis is important in order to avoid unnecessary surgery. There are some unique palsy cases, even in younger patients. Our cases showed the importance of patient history and daily routine for the diagnosis, identification of aggravating movements and design of a treatment plan that caters to the needs of the individual.

Conflict of Interest:

Authors reported no conflicts of interest.

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