



## Brachial plexus neuropathy (stinger syndrome) occurring in a patient with shoulder laxity

### *Omuz laksitesi zemininde gelişen brakial pleksus nöropatisi (stinger sendromu): Olgu sunumu*

Mehmet Can UNLU,<sup>1</sup> Hayrettin KESMEZACAR,<sup>2</sup> Isik AKGUN<sup>2</sup>

<sup>1</sup>Private Duygu Hospital, Department of Orthopedics and Traumatology;

<sup>2</sup>Istanbul University, Cerrahpasa Faculty of Medicine, Department of Orthopedics and Traumatology

Brakial pleksusun traksiyon veya kompresyon yaralanmasına bağlı gelişen sık bir nöropati olan stinger sendromu genellikle sporcu genç erişkinlerde görülür ve etyolojisinde majör bir kontak travma bulunur. İki taraflı glenohumeral eklem laksitesi olan 11 yaşındaki erkek hasta, koşarken sol omuz ekstansiyonda iken, boynun karşı tarafa minimal lateral fleksiyonu ile duvara çarptıktan sonra sol omuzda ağrı, sol kolda uyuşukluk ve kuvvet kaybı yakınmalarıyla başvurdu. Hastanın sol omzunda, nötral rotasyonda humerus başının anteroinferior doğrultuda ciddi derecede pasif translokasyonu ve sulkus bulgusu vardı. Hastaya omuz laksitesi zemininde traksiyona bağlı brakial pleksus nöropatisi (stinger sendromu) tanısıyla omuz kol askısı verildi. İkinci haftadaki muayenesinde sağ deltoid, supraspinatus ve infraspinatus kaslarında atrofi saptanması üzerine aktif ve pasif harekete yönelik omuz egzersizlerine başlandı. Üç ay sonra yapılan kontrolde aktif eklem hareketleri açık ve kas kuvvetleri normal bulundu.

**Anahtar sözcükler:** Brakial pleksus/yaralanma; eklem instabilitesi/komplikasyon; omuz eklemi/yaralanma.

The stinger syndrome is a common neuropathy caused by traction or compression of the brachial plexus. In general, it is seen in young adults involved in sport activities and a major contact trauma is the rule. An 11-year-old boy with bilateral glenohumeral joint laxity had pain in the left shoulder, numbness and decreased strength in the left arm that developed after striking against a wall while running, with the left shoulder in extension and the neck in minimal lateral flexion to the contralateral side. Physical examination showed extreme anteroinferior passive translocation of the humeral head in neutral rotation and a positive sulcus sign in the left shoulder. The diagnosis was made as brachial plexus neuropathy (stinger syndrome) resulting from traction trauma and shoulder joint laxity and a shoulder-arm brace was applied. After two weeks, atrophy was detected in the right deltoid, supraspinatus, and infraspinatus muscles, and active and passive motion exercises of the shoulder were initiated. At the end of three months, he achieved normal range of motion of the shoulder and muscle strength.

**Key words:** Brachial plexus/injuries; joint instability/complications; shoulder joint/injuries.

Traction injuries, postirradiation injuries, local and metastatic tumours, some hereditary and idiopathic disorders can result in brachial plexus neuropathy. Stinger syndrome is seen mostly in young adults who participate in sport activities; traction or compression of the brachial plexus caused by contact trauma is the etiologic factor.<sup>[1]</sup> Traction injuries due to joint laxity without a major trauma are relatively rare and can be

seen in patients with Ehlers-Danlos syndrome.<sup>[2]</sup> Joint laxity is not mentioned as a risk factor. In this paper we reported a child who had bilateral glenohumeral joint laxity and who was referred with brachial plexus neuropathy due to traction injury which occurred with the minimal lateral flexion of the neck to the opposite site when the shoulder is inferiorly depressed in extension.

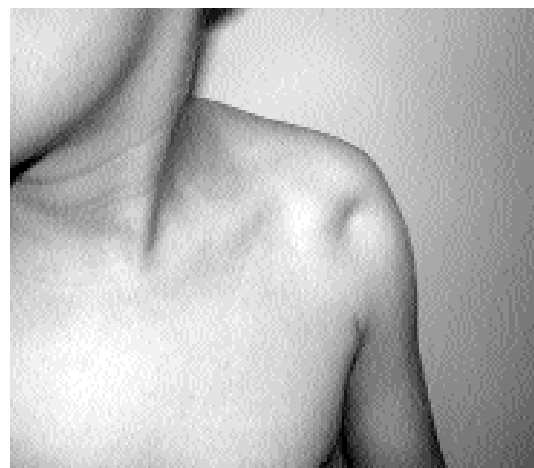
## Case report

An eleven years old boy without previously known disorder attained to hospital with pain in the left shoulder, numbness and weakness in the left arm that developed after striking against a wall while running, with the left shoulder in extension and the neck in minimal lateral flexion to the contralateral side (figure 1). Upper extremity muscle strength weakness was noted according to the MRC scale (Medical Research Council scale)<sup>[3]</sup>: deltoid (3/5), supraspinatus (3/5), infraspinatus (3/5), biceps brachii (3/5), triceps (4/5), brachioradialis (4+/5), wrist extensors (4+/5), wrist flexors (4+/5), abductor pollicis brevis (4+/5), first dorsal interossei (4+/5), and abductor digiti minimi (4+/5). Diminished biceps and brachioradialis reflexes and winging of the scapula were also noted with no sensorial abnormalities except of hypoesthesia and decreased two point discrimination in the lateral side of the shoulder. Sulcus sign as well as significant passive translocation of the humeral head to anteroinferior direction (until the point of dislocation) in neutral rotation were observed in the left shoulder (figure 2). The range of motion of the contralateral shoulder was normal with active elevation of 185 degrees, external rotation of 72 degrees and internal rotation



**Figure 1.** Mechanism of injury: Shoulder in extension and the neck in minimal lateral flexion to the opposite site.

to the level of T<sub>7</sub> were noted. Moderate passive translocation of the humeral head to anteroinferior direction in neutral rotation was also observed in the right shoulder. Systemic examination and laboratory findings (C-reactive protein, erythrocyte sedimentation rate, and blood count) were normal; no osseous pathology in the roentgenograms and no generalized joint laxity were found. The diagnosis was made as brachial plexus neuropathy (stinger syndrome) resulting from traction trauma and shoulder joint laxity. A shoulder-arm brace was applied and further evaluation was ordered. In magnetic resonance imaging (MRI) of the brachial plexus taken three days after the initial trauma, a minimal enlargement of the left 7th and 8th cervical root sheaths compared to the right side (an anatomic variation), fluid collection in the left glenohumeral joint and the subcoracoid bursa, and pathological signal changes consistent with trauma in T<sub>2</sub> weighted images in left supraspinatus and infraspinatus muscles were found (figure 3a-c). Nerve conduction study and electromyography (EMG) that were performed 11 days after the initial trauma were normal in both upper extremities except lower amplitudes for the combined action potential of the left musculocutaneous nerve than the right one. Sensorial conduction and needle EMG studies were also found as normal. The control examination in the second week revealed atrophy of the left deltoid, supraspinatus and infraspinatus muscles while the passive joint range of motion was normal. Then active and passive ROM exercises of the shoulder were initiated. After this



**Figure 2.** The sulcus sign found during the examination.

time, the patient was followed with 2 weeks' intervals. At the end of third month from the initial trauma the control nerve conduction study and EMG were normal, no atrophy was found, winging of the scapula related to weakness of serratus anterior muscle was largely improved and the sulcus sign was absent (figure 4a,b). Active joint motion and the muscle strength were normal (figure 5a,b).

## Discussion

Stinger syndrome is a peripheral nerve injury-brachial plexus neuropathy following neck and shoulder trauma. This injury is generally self limited, of short duration, but improvement may take several months in severe cases.<sup>[4]</sup> The primary symptom is burning and pain radiating from shoulder to the upper extremity accompanied with numbness, paresthesia and weakness. The syndrome is mostly seen in athletes following a major trauma.<sup>[1]</sup> Cervical fractures, dislocations and spinal cord contusion should first be excluded. When symptoms are bilateral; tenderness over the cervical vertebrae and lower extremity find-

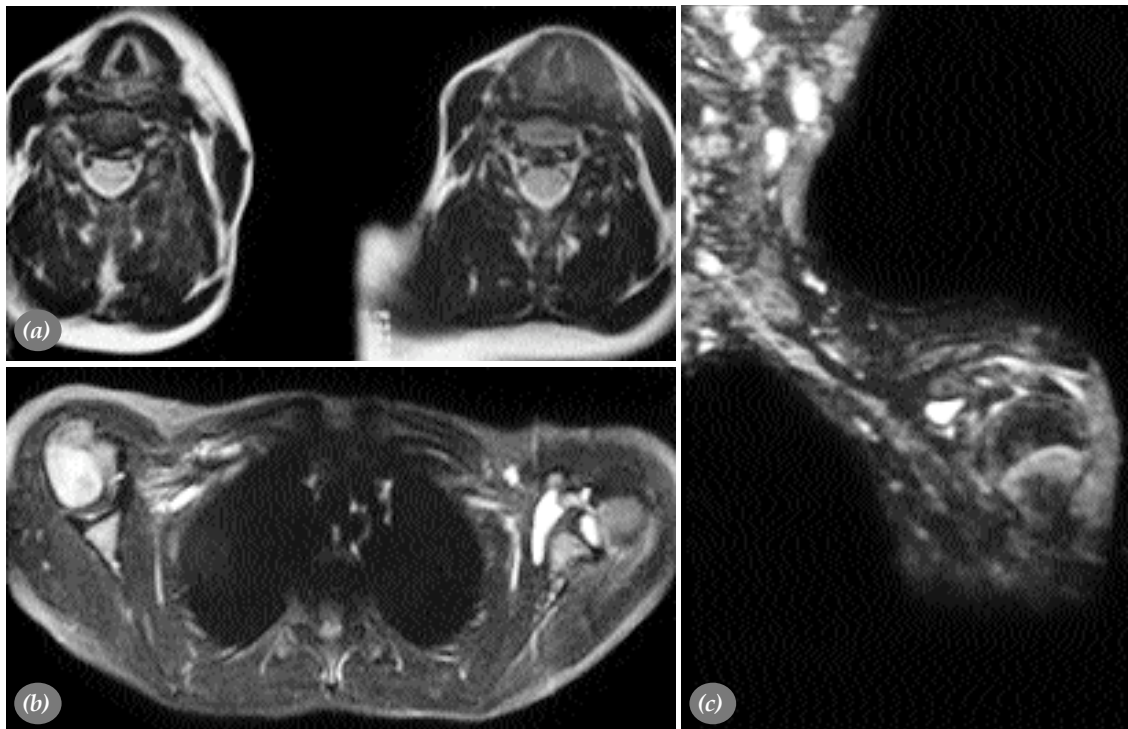
ings are present, it should be considered as cervical fracture or spinal cord contusion until proved otherwise. Clavicular fracture, shoulder dislocation and acromioclavicular joint separation should be included in the differential diagnosis.<sup>[4]</sup> There are 3 different mechanisms of trauma leading to this syndrome<sup>[5,6]</sup> :

1. The first mechanism is the traction injury of the brachial plexus; in this case, when shoulder is depressed, neck is forced to lateral flexion to the opposite side.

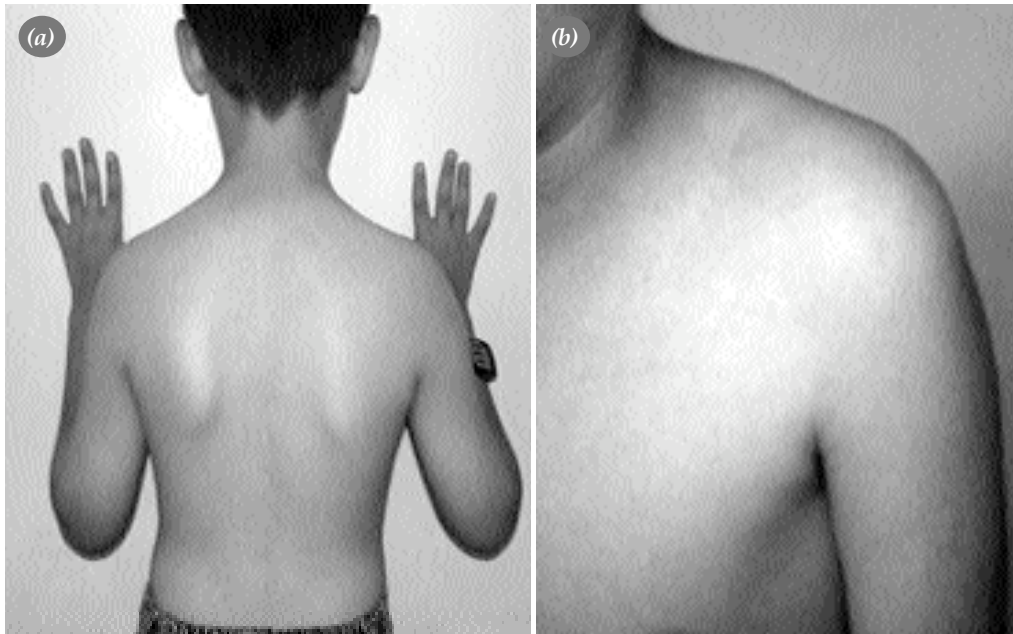
2. The second mechanism is direct blow to the supraclavicular fossa.

3. The third mechanism is neck hyperextension combined with a lateral flexion to the same side.

Cervical nerve root lesions are also reported along with upper trunk involvement in most cases.<sup>[5,7]</sup> Typically first or second degree of peripheral nerve injuries is present in this situation.<sup>[1]</sup> Neuropraxia is a first degree injury where axonal integrity is spared, but nerve functions are disrupted due to demyelination.



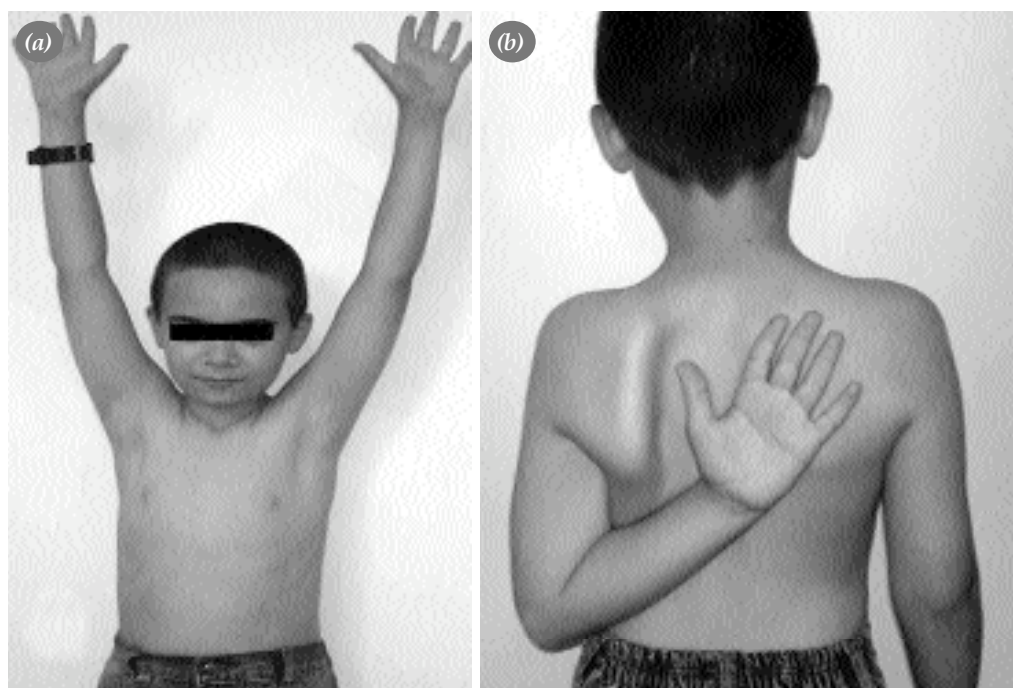
**Figure 3.** (a) Minimal enlargement of the left 7<sup>th</sup> and 8<sup>th</sup> cervical root sheaths in the cervical MRI, (b) fluid collection in the glenohumeral joint and the subcoracoid bursa in the axial view, (c) pathologic signal changes consisted with trauma are seen in the frontal images in supraspinatus and infraspinatus muscles.



**Figure 4.** (a)Winging of the scapula was largely improved; (b) appearance of the left shoulder with sulcus test.

Axonotmesis is a second degree injury with axonal damage and Wallerian degeneration. Neurotmesis is a third degree injury referring permanent nerve injury and usually is not seen in the stinger syndrome. With the evaluation of muscle strength, specific nerve involvement can be identified. Loss of motor strength

generally can be seen in the muscles innervated by the upper trunks (5<sup>th</sup> and 6<sup>th</sup>) of the brachial plexus. The diagnosis can be confirmed, localization and the severity of the lesion can be determined with electrodiagnostic studies. The time for occurrence of electromyographic changes was reported to be minimum of 3



**Figure 5.** Active elevation (a) and internal rotation (b) of the patient.

weeks following the trauma, although electromyographic studies performed 7-10 days after the trauma can show slower combined muscle action potentials in the neuropraxic lesions.<sup>[1,8,9]</sup> The goal of the treatment is first to restore the painless range of motion. Then, muscle strength could be increased with concentric and eccentric loading to different directions with different speed. Intravenous steroids are not helpful in the treatment of stinger syndrome.<sup>[10]</sup> Although it is a self limiting disorder, risk of recurrence is high and using protective equipment can be necessary during sports activities<sup>[11]</sup> and in patients whose symptoms and pathologic electrodiagnostic studies persists return to sport activities should not be allowed.<sup>[10]</sup> In our case, the mechanism of the brachial plexus injury was the traction with minimal lateral flexion of the neck to the opposite side while the shoulder was depressed. Muscle weakness that was found with the examination of the deltoid (axillary nerve; cervical 5-6), supraspinatus (suprascapular nerve; cervical 5-6), infraspinatus (suprascapular nerve; cervical 5-6); and biceps brachii (musculocutaneous nerve; cervical 5-6) muscles shows that the injury is to the upper trunks of the brachial plexus. Winging of the scapula was related to the weakness of the serratus anterior (long thoracic nerve; C5-7) and rhomboid muscles (dorsal scapular nerve; cervical 4-5). The low magnitude of the combined muscle potential in the first electrodiagnostic study and the normal results in control study suggests that the brachial plexopathy is of neuropraxia type. Clinical course also suggests this diagnosis. Brachial neuritis can be considered as an alternative diagnosis in our case.<sup>[1]</sup> Brachial neuritis is associated with motor loss and atrophy and sensorial involvement is not usual in children in contrast to adults.<sup>[12]</sup> Besides, most of pediatric brachial neuritis cases follow a systemic infection or vaccination and resolve at an average period of 6 months.<sup>[13]</sup> Absence of sensorial involvement, with no history of recent systemic infection, normal laboratory findings, and the fast improving clinical course which was noted retrospectively made us away from this diagnosis. Our case is interesting regarding with brachial plexus injury based on glenohumeral joint laxity. Generalized joint laxity is reported as a risk factor for anterior cruciate ligament injury; however, such a risk is not accounted for brachial plexus and other peripheral nerves.<sup>[14]</sup> Vaccaro et al.<sup>[15]</sup> reported that asymptomatic generalized ligamentous laxity is an absolute contraindication to return

to active sports following stinger syndrome for athletes, but they did not provide any information in the literature. Galan and Kousseff<sup>[2]</sup> reported a case of non-traumatic bilateral brachial plexus neuropathy in a patient who has a generalized joint laxity with Ehlers-Danlos syndrome. There was blockade in the nerve conduction speed study of the patient and the resolution was not complete. There are no reported risk factors for the brachial plexus traction injuries. The brachial plexus injury following a minor trauma and the absence of any other risk factor, suggests that the glenohumeral joint laxity is responsible for this situation in our case. Although relatively rare, glenohumeral laxity should be regard as a risk factor for brachial plexus injury.

## References

1. Hershman EB. Brachial plexus injuries. *Clin Sports Med* 1990;9:311-29.
2. Galan E, Kousseff BG. Peripheral neuropathy in Ehlers-Danlos syndrome. *Pediatr Neurol* 1995;12:242-5.
3. Medical Research Council of the UK. Aids to the investigation of peripheral nerve injuries. Memorandum No. 45. London: Pendragon House; 1976. p. 6-7.
4. Hershman EB. Injuries to the brachial plexus. In: Torg JS, editor. *Athletic injuries to the head, neck, and face*. 2nd ed. St. Louis: Mosby; 1991. p. 345-50.
5. Di Benedetto M, Markey K. Electrodiagnostic localization of traumatic upper trunk brachial plexopathy. *Arch Phys Med Rehabil* 1984;65:15-7.
6. Nicholas SA, Hershman EB, Posner MA. *The upper extremity in sports medicine*. 2nd ed. St. Louis: Mosby; 1995. p. 675-7.
7. Markey KL, Di Benedetto M, Curl WW. Upper trunk brachial plexopathy. The stinger syndrome. *Am J Sports Med* 1993; 21:650-5.
8. Warren RF. Neurologic injuries in football. In: Jordan BD, Tsairis P, Warren RF, editors. *Sports neurology*. Rockville: Aspen; 1989. p. 235-7.
9. Robinson LR. Traumatic injury to peripheral nerves. *Muscle Nerve* 2000;23:863-73.
10. Castro FP Jr. Stingers, cervical cord neurapraxia, and stenosis. *Clin Sports Med* 2003;22:483-92.
11. Weinberg J, Rokito S, Silber JS. Etiology, treatment, and prevention of athletic "stingers". *Clin Sports Med* 2003; 22:493-500, viii.
12. van Alfen N, Schuurin J, van Engelen BG, Rotteveel JJ, Gabreels FJ. Idiopathic neuralgic amyotrophy in children. A distinct phenotype compared to the adult form. *Neuropediatrics* 2000;31:328-32.
13. Tsairis P, Dyck PJ, Mulder DW. Natural history of brachial plexus neuropathy. Report on 99 patients. *Arch Neurol* 1972; 27:109-17.
14. Uhorchak JM, Scoville CR, Williams GN, Arciero RA, St Pierre P, Taylor DC. Risk factors associated with noncontact injury of the anterior cruciate ligament: a prospective four-year evaluation of 859 West Point cadets. *Am J Sports Med* 2003;31:831-42.

15. Vaccaro AR, Klein GR, Ciccoti M, Pfaff WL, Moulton MJ, Hilibrand AJ, et al. Return to play criteria for the athlete with

cervical spine injuries resulting in stinger and transient quadriplegia/paresis. *Spine J* 2002;2:351-6.