



## Triple Peripheral Nerve Injury Accompanying to Traumatic Brain Injury: A Case Report

### Travmatik Beyin Hasarına Eşlik Eden Üçlü Periferik Sinir Hasarı: Bir Vaka Sunumu

Peripheral Sinir Hasarı ve Beyin Hasarı / Peripheral Nerve Injury and Brain Injury

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#### Özet

Travmatik beyin hasarı (TBH) ile eşzamanlı olarak ikincil yaralanmalar özellikle ekstremitelerde fraktürleri görülebilir. Periferik sinir hasarları bu fraktürlere eşlik edebilir. Ancak bu durum özellikle akut evrede göz ardı edilebilir. Bu vaka sunumunda araç içi trafik kazasına (AİTK) bağlı TBH gelişen bir hastada akut evrede tespit edilemeyen humerus alt uç fraktürüne sekonder gelişen radial, ulnar ve median sinir hasarı sunulmuştur. 29 yaşında bir erkek hasta sağ üst ekstremitede güçsüzlük şikayetiyle kliniğimize başvurmuştur. Öyküsünde 9 ay önce AİTK'ye bağlı travmatik beyin hasarı geçirdiği ve takiplerinde humerus alt uç fraktürü saptandığı öğrenildi. Muayenesinde periferik sinir hasarından şüphelenilerek tanı elektronöromiyografi (ENMG) ile teyit edildi. Hasta, uygun rehabilitasyon programı uyguladık-tan sonra tedaviye olumlu yanıt verdi. TBH'lı bir hastada, ikincil bir travma olabileceği ve buna bağlı olarak periferik sinir lezyonlarının gelişebileceği akılda tutulmalı ve ayrıntılı tanıda dikkatli olunmalıdır.

#### Anahtar Kelimeler

Travmatik Beyin Hasarı; Periferik Sinir Hasarı; Rehabilitasyon

#### Abstract

Secondary injuries especially extremity fractures may be seen concurrently with traumatic brain injury (TBI). Peripheral nerve damages may accompany to these fractures and may be missed out, especially in acute stage. In this case report; damage of radial, ulnar and median nerves which was developed secondarily to distal humerus fracture that could not be detected in acute stage, in a patient who had motor vehicle accident (MVA). 29-year-old male patient was admitted with weakness in the right upper extremity. 9 months ago, he had traumatic brain injury because of MVA, and fracture of distal humerus was detected in follow-ups. Upon the suspect of the peripheral nerve injury, the diagnosis was confirmed with ENMG. The patient responded well to the rehabilitation program treatment. In a TBI patient, it must be kept in mind that there might be a secondary trauma and therefore peripheral nerve lesions may accompany to TBI.

#### Keywords

Traumatic Brain Injury; Peripheral Nerve Injury; Rehabilitation

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## Introduction

Traumas are the leading cause of mortality in children, young adults and adults (aged between 1 and 44), moreover traumatic injury is third most common mortality cause after cardiac diseases and cancer in all age groups [1]. In our country, according to a study conducted in a large center, 1,267 patients admitted to the emergency room, and 440 of them (34.7%) has been identified in head and neck injury [2]. Long treatment periods, care requirements and serious disabilities are the problems in question in head traumas and especially in motor vehicle accident (MVA), and these leads to socioeconomic problems.

In recent years, survival rates of these patients increased significantly with the developments in treatment management of TBI[3]. Elimination or minimization of the disabilities gained a special importance because the majority of the surviving patients are young males who can still have productivity for years. Traumatic brain injury (TBI) may be accompanied with secondary injuries simultaneously[3]. Especially extremity fractures may develop in TBI and it also increase the risk of peripheral nerve damage[3]. However this condition may be missed especially in acute stage, because the interventions is targeting the survival of the patient draw more attention and also because of the impairment of the cognitive state of the patient. Peripheral nerve damages affect the functional recovery and returning to work, dramatically. Furthermore, since peripheral nerve damages could increase the risk of falling, fracture or other secondary damages; early detection and treatment of these lesions become crucial in these patients [4].

Here, a case who had right-sided hemiplegia after a MVA, and then diagnosed with radial, median and ulnar nerve damages secondary to the fracture of distal humerus, in the clinical follow-up, was presented.

## Case Report

A 29-year-old male patient applied to our clinic with a complaint of the right-sided weakness. From the patient's medical history, we learned that he had MVA 9 months ago, and developed linear fracture in left parietotemporal bone and acute subdural hematoma in left parietal region after the accident. It was learned that hematoma was evacuated surgically. Fracture of the distal humerus was detected in the follow-up of the patient, and it was fixed with cast for one month and a half, and with splint after that (Figure 1). The patient was followed and treated in intensive care unit for 38 days, and then in neurology inpatient clinic for 16 days, in that period. The patient admitted to our clinic for rehabilitation, 9 months after the accident.

In his examination, there was a restriction of the right shoulder joint movements (shoulder flexion 120°, abduction 90° and internal-external rotation was open). In his motor examination, muscle strength was assessed as 5/5 in right shoulder area, 4/5 in elbow flexor and extensors, 4/5 in wrist extensors, 3/5 in finger abduction and adduction movements, and also there was atrophies in thenar and hypothenar area and claw hand developed in the right hand (Figure 2). There was no increase in tonus, the deep tendon reflexes were increased in right upper and lower extremities. Plantar responses were flexor in both sides. Electroneuromyography (ENMG) was performed after being suspected for peripheral nerve lesion in right of the examina-



Fig 1. Fragmented distal humerus fracture



Fig 2. Claw hand and atrophies in the right hand

Table 1. ENMG findings of patient before the treatment and 3 months after the treatment

	Pre-treatment	Post-treatment
Median nerve MCV (m/s)	50.6	51.3
Median nerve SCV (m/s)	52.2	73.9
Ulnar nerve MCV(m/s)	57.9	59.4
Ulnar nerve SCV(m/s)	51.9	51.9
Radial nerve MCV(m/s)	43.8	68.4
Radial nerve SCV(m/s)	59.5	59.5

MCV: motor conduction velocity; SCV: sensory conduction velocity

tion findings of the patient. It was considered as partial axonal damage at chronic stage in median, radial and ulnar nerves in the right side. An outpatient physical therapy program was administered an hour a day for 30 days, under the supervision of an experienced physiotherapist (YK).The rehabilitation program including electrical stimulation which performed to weakened muscles, and upper extremity strengthening, and also fine motor skills exercises, was applied to the patient as treatment pro-

gram. Clinical improvement was observed in the patient after the treatment. After three months of PT, in his control examination, the restrictions in shoulder and elbow movements was detected to be reduced (flexion 140°, abduction 100°) and in motor examination the muscle strengths of elbow flexion and extension muscles, wrist extension muscles were detected to be 5/5. And also significant improvement of conduction velocities were detected in the ENMG performed in the 3rd month follow-up visit (Table 1).

## Discussion

Extremity injuries and peripheral nerve damages may be seen concurrently with TBI. In addition to the difficulties in the diagnosis of peripheral nerve damage, it can also worsen the clinical presentation by increasing the disability of the patient. Here, a case was presented who had fracture of the distal humerus concurrently with TBI, and then developed radial, median and ulnar nerve damages secondary to this, and diagnosed approximately 9 months after the trauma.

Groswasser et al., [5] showed in their study that 58% of the 328 severe TBI patients had skeletal system fractures related to primary trauma concurrently, and the humerus fractures may be seen at 10% frequency. In a review by Garland et al. including 254 adult TBI patients, extremity fractures or dislocations were present in 72 of them, and neuropathy was present in 29 of them [6]. It was detected that 60% of the patients who had peripheral nerve lesion, also had head trauma [4]. Peripheral nerve damages may also develop secondarily to late-term complications such as hypertrophic callus, myositis ossificans, and heterotopic ossification [3]. Similarly, in our patient, it can be observed that pseudoarthrosis was developed as complication (Figure 1). And, this condition can also increase the risk of peripheral nerve damage. Pseudoarthrosis seen after humerus injury, may especially associated with radial nerve damage [7]. This risk is even more prominent, particularly in patients who are being followed-up conservatively [7]. It was learned from our patient's history, that humerus fracture was detected while he was staying in intensive care unit, but since his clinical condition then was not suitable for surgery, humerus was stabilized with plaster cast. Afterwards, the patient was consulted to orthopedics, and no surgical intervention was recommended.

In a study performed by Stone et al., it was detected that 10-34% of the TBI patients had peripheral nerve damage concurrently, and the most common developed pathologies were entrapment of the ulnar nerve at the wrist (10%) and brachial plexus damage, however none of these damages were diagnosed in the first application [6,8-9]. This delay was reported as 60 days averagely by authors, while this delay period was determined as 140.4 days in the Stone's study [9]. Our case was also diagnosed 9 months after the trauma.

In the case of central nerve system damage, the peripheral nerve system damage may easily be missed. Especially in acute stage, the emphasis is put on the interventions targeting the survival of the patient. Performing a neurological examination and taking a medical history is very difficult because of the impairment in the cognitive state of the patient, moreover the early clues of the peripheral nerve lesion accompanying to brain injury may only be looseness, areflexia and weakness in

the affected extremity. These symptoms may be attributed to traumatic brain injury of the patient in early stage. Because of all these reasons, the peripheral nerve damages may be missed out in this group of patients. TBI is usually courses with findings of upper motor neuron damage such as muscle weakness, spasticity, hyperactive deep tendon reflexes and development of pathological reflexes. Accompanying secondary motor neuron disorders such as secondary peripheral nerve damage or plexus damage should be considered in a TBI patient with findings of secondary motor neuron disorder such as hypotonia, hypoactive or absent deep tendon reflexes, significant muscle atrophy, fasciculation and sensation disorders; particularly in multi trauma patients [3]. Therefore, after stabilizing the patient clinically, the examinations must be repeated and the physicians must be careful in terms of possible peripheral lesion and nerve damage. ENMG takes an important place in diagnosis. Electrodiagnostic studies provide important benefits for the evaluation and management of nerve injury by giving information about the localization and severity of nerve damage [10,11,12]. Moreover, they give information on the estimation of prognosis and recovery period [12]. Optimal timing of these studies however, change based on the clinical condition. Following muscle denervation, ENMG stays occult for 5-7 days. Fibrillation potentials are seen frequently in denervated muscle, and these potentials come in sight in 3 weeks after the injury. For the situations in which it is important to define an early lesion, first evaluation may be performed in between 7th and 10th days to determine the localization and distinguish the axonotmesis. On the other hand, if the clinical conditions allow to wait, much more diagnostic information will be obtained from the studies performed 3-4 weeks after the trauma, because the fibrillation will be more apparent in needle ENMG [12]. First evidences of reinnervation can be detected with ENMG before the voluntary contraction becomes observable [13]. Low amplitude, short period and polyphasic motor unit potentials are the signs of repair and reinnervation. While regeneration continues; fibrillation potentials decrease and poliphasic motor unit frequency increases. Fibrillation potentials persist for 2-3 years, while this period may increase up to 4-5 years in serious nerve damages [14]. In our case the diagnosis was also confirmed with ENMG after clinical suspicion, and the rehabilitation program was reshaped according to nerve damages.

In conclusion, since the weakness in the multiple trauma patients is attributed to neurological condition, the peripheral nerve lesions may be missed out within the first weeks after trauma. In TBI patients, it must always be kept in mind that there might be peripheral nerve damage, and it may be remained as undiagnosed. This is extremely important in unraveling the health problem entirely, and in the minimization of the disability rate.

## Competing interests

The authors declare that they have no competing interests.

## References

1. Mac Kenzie EJ, Fowler CJ. Epidemiology. In: Mattox KL, Feliciano DV, Moore EE, editors. Trauma. 4th ed. New York: McGraw-Hill;2000.p.21-36.
2. Durdu T, Kavalcı C, Yılmaz F, Yılmaz MS, Karakılıç ME, Arslan ED. Analysis of trauma cases admitted to the emergency department. J Clin Anal Med 2014;5(3):182-5.
3. Kushwaha VP, Garland DG. Extremity fractures in the patient with a traumatic

brain injury. J Am Acta Orthop Surg 1998;6(5):298-307.

4. Noble J, Munro CA, Prasad VSSV, Midha R. Analysis of upper and lower extremity peripheral nerve injuries in a population of patients with multiple injuries. J Trauma 1998;45(1):116-22.
5. Groswasser Z, Cohen M, Blankstein E. Polytrauma associated with traumatic brain injury: incidence, nature and impact on rehabilitation outcome. Brain Inj 1990;4(2):161-6.
6. Garland DE, Douglas E, Bailey S. Undetected injuries in head-injured adults. Clin Orthop Relat Res 1981;(155):162-5.
7. Blum J1, Rommens PM. Surgical approaches to the humeral shaft. Acta Chir Belg 1997;97(5):237-43.
8. Cosgrove JL, James L, Vargo M, Reidy ME. A prospective study of peripheral nerve lesions occurring in traumatic brain-injured patients. Am J Phys Med Rehabil 1989;68(1):15-7.
9. Stone L, Keenan MAE. Peripheral nerve injuries in the adult with traumatic brain injury. Clin Orthop Relat Res 1988;(233):136-44.
10. Aminoff MJ. Electrophysiologic testing for the diagnosis of peripheral nerve injuries. Anesthesiology 2004;100(5):1298-303.
11. Chaudhry V, Glass JD, Griffin JW. Wallerian degeneration in peripheral nerve disease. Neurol Clin 1992;10(3):613-27.
12. Robinson LR. Traumatic injury to peripheral nerves. Muscle Nerve 2000;23:863-73.
13. Kline DG. Clinical and electrical evaluation. In: Kim DH, Midha R, Murovic JA, Spinner RJ, editors. Kline & Hudson's nerve injuries. 2nd ed. Philadelphia: Elsevier, Saunders; 2008.p.43-63.
14. Archibald KC. Clinical usefulness of EMG and nerve conduction tests in nerve injury and repair. In: Jewitt DL, McCarrol HR, editors. Nerve Repair and Regeneration:its clinical and experimental basis. St Louis: Ran Press;1980.p.308-20.

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