

# Unrecognized Peripheral Nerve Lesions in a Traumatic Brain Injury Patient

## ABSTRACT

Patients with traumatic brain injury constitute a diagnostic challenge since peripheral nerve injuries may be overlooked due to cognitive dysfunction and priority given to life-sustaining measures. Electromyography may be helpful in the differential diagnosis of weakness and atrophy. Problems specific for the traumatic brain injury patients, namely heterotopic ossification, hypertrophic callus formation and myositis ossificans should be considered by the physician. We report a 15-year-old patient involved in a pedestrian motor vehicle accident with traumatic brain injury. He had weakness and atrophy of the left upper extremity. Electromyographic examination revealed axillary nerve injury and carpal tunnel syndrome. Differential diagnosis of atrophy and weakness in traumatic brain injury patients is discussed.

**KEY WORDS:** Axillary nerve, Carpal tunnel syndrome, Fracture, Humerus, Peripheral nerve, Traumatic brain injury.

## INTRODUCTION

Traumatic brain injuries (TBI) are often accompanied by additional trauma that can remain undiagnosed because of cognitive dysfunction of patients and priority given to life-sustaining measures. Extremity fractures are common in patients with TBI and upper extremity fractures are often associated with peripheral nerve injuries (6). Patients with TBI and skeletal injuries have increased rates of excessive bone healing (hypertrophic callus formation and/or heterotopic ossification) (1). There is ongoing research on the immunohistochemical basis of hypertrophic callus formation in patients with TBI (1,14).

Brachial plexopathies as a result of compression by hypertrophic callus have been reported (2,5) but other peripheral neuropathies associated with hypertrophic callus formation in patients with TBI are rarely encountered.

This case report describes a TBI patient with an axillary nerve injury associated with hypertrophic callus formation, and median nerve injury recognized with electromyographic examination.

## CASE REPORT

A 15-year-old boy was involved in a pedestrian motor vehicle accident. Soft tissue swelling of the scalp in right temporoparietal area and a linear fracture of the right temporal bone were detected in computed tomography of the brain. A humerus fracture was detected in an anteroposterior shoulder X-ray. Plate and screw fixation was used for fracture stabilization and removed after one month.

He was admitted to a rehabilitation ward 2.5 months after the trauma. On initial examination at the rehabilitation center, the patient

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had restricted active motion of left shoulder in all planes. Passive motion of the shoulder was painful but not limited. He had diffuse atrophy of the left upper extremity including the deltoid, periscapular muscles and intrinsic hand muscles. He had weakness of both proximal and distal muscles of the left upper extremity (Medical Research Council grade 3). His deep tendon reflexes were hyperactive on the left side. Plantar response was flexor on the right side and extensor on the left side. He had slight increase of tonus in the left upper extremity (Modified Ashworth Scale grade 2).

Anteroposterior X-ray of the shoulder demonstrated hypertrophic callus formation around the neck of the humerus (Figure 1).

Electromyography (EMG) was done for the differential diagnosis of the left upper extremity weakness. The left axillary nerve Erb latency could not be determined. Erb latency for radial, suprascapular and musculocutaneous nerves were within normal limits. Nerve conduction studies revealed prolonged distal motor latency and decreased compound muscle action potential amplitude of the left median nerve but both motor and sensory nerve conduction studies were otherwise normal. Needle examination of the left



**Figure 1:** Anteroposterior X-ray of the shoulder demonstrates hypertrophic callus formation on the site of humerus fracture.

deltoid muscle denervation potentials (fibrillations and positive sharp waves) was performed and Motor Unit Potentials (MUP) could not be obtained. Examination of the Abductor Pollicis Brevis by needle demonstrated MUPs with greater than normal amplitude and decreased recruitment on maximum contraction.

Rehabilitation efforts included a range of motion exercises, strengthening exercises and occupational therapy.

## DISCUSSION

Injuries are the leading cause of death in children, and brain injury is the most common cause of pediatric traumatic death (13). While the injury may be the cause of a patient's abnormal development, other factors should also be considered. Survival has increased in recent years with improvement in the medical management of TBI patient. Minimizing disability is especially important because most survivors are young males, with many years of productivity ahead (6). In a study with 74 pediatric polytrauma patients, 80% survived but 22% were disabled 1 year after injury, mainly due to severe brain injuries (12).

Schalomon and co-workers reported that injuries of the head/neck area were most frequent (87%) followed by extremity fractures (76%) in children with polytrauma (10). In one series of 328 patients with severe TBI; 58% had associated trauma, mostly in the skeletal system and humerus fractures constituted 10% of all fractures (4). There is increased risk of compression neuropathy occurrence due to excessive bone formation (heterotopic ossification and/or hypertrophic callus) in patients with TBI. Compression of the axillary nerve occurs in quadrilateral space syndrome, but injury is most commonly seen after glenohumeral joint dislocation, proximal humerus fracture, or a direct blow to the deltoid muscle (8). In our patient, we considered peripheral nerve injury in the differential diagnosis of unilateral upper extremity weakness and atrophy. Hypertrophic callus that was held responsible for the axillary nerve injury was detected on X-ray. Brachial plexopathy, suprascapular neuropathy and C5 radiculopathy were excluded according to EMG findings. Since many patients are able to compensate for the loss of the deltoid muscle with time, the clinical presentation of the axillary nerve injury may be quite variable (8). Additionally, our patient had carpal tunnel syndrome affecting the motor fibers of

the median nerve that could further adversely affect his upper extremity function. Flexed posture of the wrist due to increased tonus has been reported to cause carpal tunnel syndrome in patients with TBI (7,11).

In another series 50 patients with TBI the incidence of peripheral nerve injury was 34%; a variety of nerve injuries were seen, the most frequent of which were ulnar nerve entrapment at the elbow (10%) and brachial plexus injuries (10%); all neuropathies were missed prior to admission (11). Axillary nerve injury and carpal tunnel syndrome are rare in patients with TBI. The physician should keep in mind that any peripheral nerve injury can occur and remain undiagnosed in patients with TBI.

EMG is useful for the differential diagnosis of peripheral nerve injuries. Philip et al. reported that twelve patients met clinical diagnostic criteria for peripheral nerve injury in 157 children with TBI and an electrodiagnostic study confirmed the clinical diagnosis in 11 patients (9). Our patient had diffuse atrophy and weakness in left upper extremity that might be attributed to TBI or disuse. The differential diagnosis of a suspected axillary neuropathy includes a lesion of the posterior cord of the brachial plexus, suprascapular neuropathy, a C5 or C6 radiculopathy, and neuralgic amyotrophy (3).

In polytrauma patients, peripheral nerve lesions may be overlooked in the first weeks after trauma since weakness may be attributed to pain or the fracture. The physician should have enough knowledge about challenges specific to the TBI patient such as heterotopic ossification, hypertrophic callus formation and myositis ossificans. A high index of suspicion is required since the patient with TBI may be unable to define the problem due to cognitive impairments. Satisfactory functional achievements can only be obtained by careful differential diagnosis and treatment of accompanying disorders.

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