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Isolated musculocutaneous nerve injury following a pedestrian-automobile collision: A case report

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Abstract

Isolated musculocutaneous nerve (MCN) lesion is rare and usually associated with direct trauma. Along with the rarity of this condition, other muscles involved in elbow flexion, such as brachioradialis and pronator teres, can mask the weakness induced by the MCN injury and make it difficult to identify it. Here, we report a 17-year-old patient with isolated MCN palsy following a single episode of anterior shoulder contusion. A lack of suspicion for this rare condition delayed diagnosis until 7 months post injury, when atrophy of muscles in the left upper arm became prominent and weakness of the elbow flexors persisted. After 6 months of rehabilitation therapy rather than undergoing surgical exploration, elbow flexor strength was nearly fully recovered but sensory symptoms remained. The mechanism of injury is speculated to be a sudden overloading of the anterior shoulder with extension and external rotation, which overstretched and compressed the MCN within the coracobrachialis muscle where the nerve is relatively fixed. Although isolated peripheral nerve injury is rare, it can be caused by a single episode of vigorous impact. Therefore, even in patients without any external wounds, careful physical examination with suspicion of peripheral nerve injury as one of the differential diagnoses is needed.

Keywords: Musculocutaneous nerve, lateral antebrachial cutaneous nerve, electrodiagnosis

INTROUDUCTION

Isolated musculocutaneous nerve (MCN) injury, i.e., MCN injury that does not involve the brachial plexus, is rare. The few cases of isolated MCN injuries reported have been caused by a variety of mechanisms including direct stab or crush injury, vigorous activity, and continuous activity.[1-5]

Here, we report a rare case of proximal MCN palsy that was derived from a single episode of shoulder contusion without any fracture or dislocation of the shoulder during a pedestrian-automobile collision. A lack of suspicion for this rare condition delayed diagnosis until 7 months after the injury and, as a result, the patient missed the critical window in which surgical exploration would have been beneficial. Fortunately, with conservative treatment there was distinct motor improvement clinically and electrophysiologically; however, sensory symptoms such as numbness and hypoesthesia remained.

CASE REPORT

A 17-year-old adolescent presented to the electromyography (EMG) laboratory reporting an inability to flex his elbow and numbness of his left forearm. Seven months prior to presentation, he had sustained a left ulnar-styloid fracture and multiple contusions during a pedestrian-automobile collision. The exact details of the accident were unknown because he lost consciousness for seconds following the collision. The staff in the emergency department recorded an external wound to the left forehead (2 cm depth) and bruising to multiple sites including the left anterior shoulder, left upper arm, and left knee. Radiologic examinations revealed only an ulnar-styloid fracture. His forehead laceration was sutured and a short-arm splint was placed to stabilize the ulnar fracture. After 4 weeks, when ulnar bone union was complete, the splint was removed and the patient returned to his everyday life.

The patient noticed prominent atrophy of muscles in the left upper arm and persistent

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Table 1: Results of nerve conduction studies

	Initial (7 months after onset)		Follow up (13 months after onset)	
	Latency (ms)	Amplitude (mV)	Latency (ms)	Amplitude (mV)
Musculocutaneous, motor (biceps brachii)				
Right	4.3	12.5	4.4	13.0
Left	5.9	6.1	4.6	11.8
Lateral antebrachial cutaneous, sensory				
Right	1.4	0.0243	1.5	0.0242
Left	1.6	0.0100	1.6	0.0201

weakness of the left elbow flexors over the following months. Seven months after the injury, he visited a family physician with the chief complaint of atrophy of muscles in the left upper arm and was referred to our EMG laboratory for further evaluation.

Physical examination revealed remarkable atrophy of the left biceps brachii, with elbow flexion and forearm supination weakness evident on manual muscle testing (fair and good, respectively). The left biceps jerk was 1+ grade and sensory examination revealed a dense impairment of light touch, pin-prick, and temperature sensations in the anterolateral aspect of the left forearm. Passive range of motion of the left upper limb, cranial nerve function, coordination, and gait were normal.

An electrodiagnostic examination was performed during a second visit. Motor and sensory nerve conduction studies of both upper extremities showed no evidence of abnormality except in the left MCN and lateral antebrachial cutaneous nerve (51.2% and 58.8% reduction in amplitude compare to the contralateral side, respectively; Table 1). Needle EMG of the left biceps brachii and brachialis muscles revealed abundant abnormal spontaneous activities with several long-duration polyphasic motor units active on volition with a markedly reduced recruitment pattern. Needle EMG of paracervical muscles and other muscles of the left upper limb, including the coracobrachialis, did not show any abnormal findings.

With the suspicion of left MCN palsy distal to the innervation of the coracobrachialis muscle, a shoulder magnetic resonance imaging (MRI) study was performed. This did not reveal any remarkable findings except fluid collection below the coracoid process (Figure 1). The rotator cuff and the other soft and bony tissues were sound.

The physician and patient agreed to forgo surgical exploration at this time, and conservative management such as strengthening exercises was started. At 6 months after initial diagnosis (13 months after the injury), there was clinical improvement in muscle power (elbow flexion good, forearm supination good to normal) and atrophy. Electrophysiologic study at 13 months after injury revealed improvement compared to the initial study (Table 1). However, the patient still experienced sensory discomfort and numbness on the anterolateral forearm.

DISCUSSION

The MCN arises from the lateral cord of the brachial plexus. At the level of the midshaft of the clavicle and coracoid process, the lateral cord separates into the lateral root of the median nerve and the MCN. After penetrating the coracobrachialis muscle 31–82 mm distal to the coracoid process, the MCN passes between the biceps brachii and the brachialis muscles to the lateral side of the arm.[6] Before piercing the coracobrachialis muscle, the MCN has a short course and is deep. After this point, it is relatively fixed by branches along its course between the biceps brachii and brachialis muscles. As such, MCN injury that does not involve brachial plexus injury is rare. An isolated MCN injury was reported by Sunderland[7], with an incidence of 1/1,008 injuries. The few cases of isolated MCN injuries reported have a variety of mechanisms, including direct stab or crush injury, vigorous activity, and continuous activity.[1-5] This is the first reported case of MCN injury caused by a single episode of contusion to the anterior shoulder.

Injury to the MCN can cause weakness of elbow flexors and forearm supinators.[8] Along with the rarity of this condition, the presence of other muscles involved in these motions, such as

the brachioradialis and pronator teres, may mask the weakness, making it difficult to diagnose an isolated musculocutaneous neuropathy. Even after complete loss of motor function of the biceps brachii and brachialis muscles, which are innervated by MCN, functional elbow flexion strength can be obtained by contraction of the brachioradialis and pronator teres muscles. In the case reported here, neither the patient nor the physician had any suspicion of nerve injury or elbow flexor weakness at the time of splint removal. Instead, they regarded it as a result of disuse, until prominent atrophy of the biceps brachii muscle became evident. As a result, the patient missed the critical window in which surgical exploration would have been beneficial. Fortunately, with conservative treatment, there was distinct motor improvement clinically and electrophysiologically. Although sensory symptoms such as numbness and hypoesthesia remained, they were tolerable.

In this case, involvement of the biceps brachii with sparing of the coracobrachialis indicates that the MCN lesion was located between the offshoots of the branches to the coracobrachialis and biceps brachii. We speculate that the mechanism of this injury was a sudden overloading on the anterior shoulder while it was in an extended and externally rotated position, which overstretched and compressed the MCN within the coracobrachialis muscle where the nerve is relatively fixed. MRI revealed fluid below the coracoid process (Figure 1). Similar to previous reports[5,9], this may point to the level of nerve injury being below the coracoid process, after the MCN branches into the coracobrachialis muscle. However, it is not obvious if the fluid below the coracoid process was related directly to the nerve injury or not, because the MRI study was performed 7 months after the initial injury occurred.

The differential diagnosis of MCN injury includes a rupture of the biceps brachii muscle, C5 or C6 radiculopathy, and a brachial plexus injury with or without involvement of other peripheral nerves. In the case reported here, there was no characteristic lump on the anterior surface of the middle or distal part of the arm, which is a typical feature of a biceps rupture. The possibility of cervical radiculopathy was low because less than fair muscle strength and profound muscle atrophy, especially isolated to the biceps brachii muscle, are rare in cervical radiculopathy. The distribution of sensory change did not indicate a cervical origin, and the patient did not have posterior neck pain. In this case, most important clues to diagnose MCN injury were derived from the previous trauma history and the physical exam. This diagnosis was confirmed by electrodiagnostic study. Nerve conduction studies did not reveal any evidence of abnormality in other peripheral nerves such as median, ulnar, radial, or axillary nerves. Needle EMG also supported the conclusion that muscles supplied by the C5-C6 roots such as deltoid, supraspinatus, infraspinatus, and teres minor were not involved.

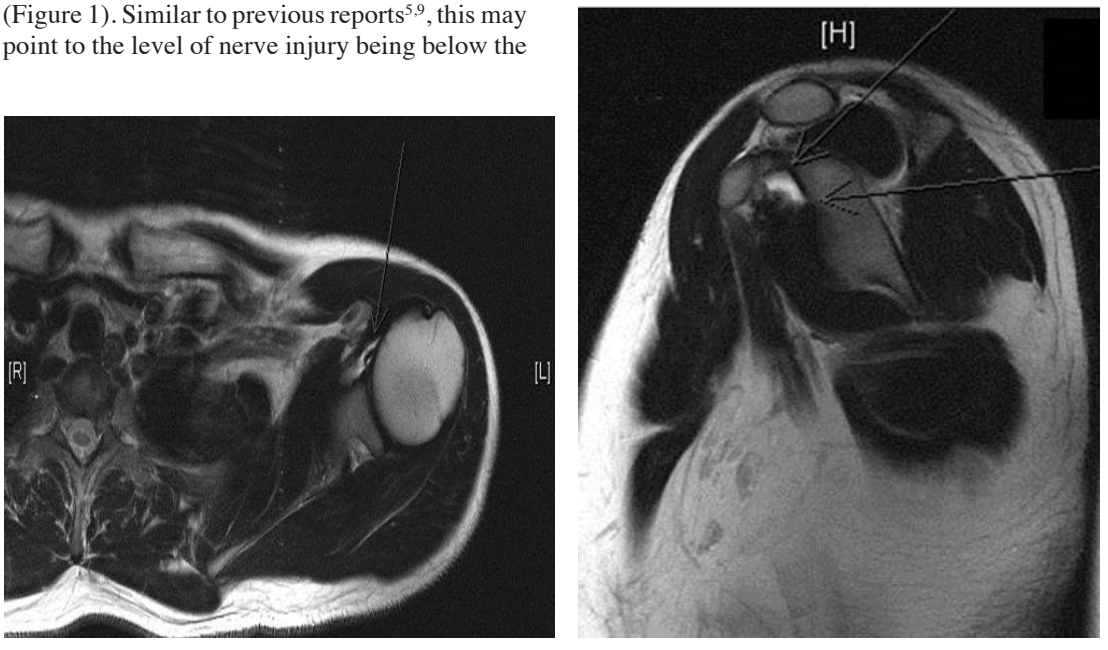

Figure 1. Axial (A) and sagittal (B) sections of magnetic resonance images of the left shoulder, demonstrating fluid collection below the coracoid process.

In summary, we have reported a rare case of peripheral nerve injury in which diagnosis was delayed beyond the critical period for surgical exploration. With closed nerve injuries, many authors agree that surgical exploration is useful in a time window of 3–6 months after the injury if no signs of recovery have occurred, but that a delay of more than 6 months will diminish the chance of recovery.[10-12] According to these recommendations, 7 months after nerve injury is too late for surgical exploration, leading to a poor prognosis.[12] Detection of peripheral nerve injuries is very important for facilitating proper diagnosis and management within the critical time period. Therefore, even in patients without any external wounds, physicians need to consider peripheral nerve injury as one of the differential diagnoses. In the management of the patients with pain from trauma, careful and serial physical exams are required to identify whether the motor weakness is from nerve injury or pain.

DISCLOSURE

Ethical approval: The ethics committee of CHA Bundang medical center granted an exemption of consent for publication of images, because the images are entirely unidentifiable and there are no details on individuals reported within the manuscript. The reference number is BD2015-215.

Informed consent: The patient has given his consent to participate and for the case report to be published.

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