

Traumatic injury to the axillary nerve associated with paralysis of triceps brachii: A case report

Leila Sadat Mohamadi Jahromi¹, Mohamadreza Emad^{1,2}, Amin Niakan^{3,4}, Hamid Reza Farpour^{5,6}

¹Department of Physical Medicine and Rehabilitation, Shiraz University of Medical Sciences, Shiraz, Iran

²Department of Physical Medicine and Rehabilitation, Shiraz University of Medical Sciences, Faghihi Hospital, Shiraz, Iran

³Department of Neurosurgery, Shiraz University of Medical Sciences, Shiraz, Iran

⁴Trauma Research Center, Shiraz University of Medical Sciences, Shiraz, Iran

⁵Department of Physical Medicine and Rehabilitation, Bone and Joint Diseases Research Center, Shiraz University of Medical Sciences, Shiraz, Iran

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ABSTRACT

Although many textbooks reported the innervation of three heads of the triceps muscle with the radial nerve, some studies showed the axillary nerve supply to this muscle. Herein, we report a 21-year-old male patients with a traumatic injury to the axillary nerve associated with paralysis of his triceps muscle. Based on the electrophysiological findings, it could be reasonable to conclude that the main branch innervating all heads of the triceps muscle originated from the axillary nerve. It is vital to look for concomitant paralysis of the triceps in patients with an axillary nerve injury in terms of surgical management, prognosis, and nerve repair.

Keywords: Axillary nerve, radial nerve, paralysis, triceps brachii.

The axillary and radial nerves are both the terminal branches of the posterior cord of the brachial plexus.^[1] The axillary nerve arises at the level of the axillary area and carries fibers from the C5-C6 spinal nerves. The deltoid and teres minor muscles are usually supplied by this nerve. Its sensory fibers also innervate the skin covering the proximal part of the shoulder.^[2] The axillary nerve has an essential role in shoulder abduction and external rotation.^[3] The radial nerve carries nerve fibers from the C5-T1 spinal nerves.^[1] It is the motor nerve of all extensor muscles of the arm and forearm. The triceps brachii muscle is located in the posterior part of the arm and acts as a powerful extensor of the forearm at the elbow.^[2,4]

It has three heads (medial, long, and lateral) that are inserted into the olecranon process with a single tendon.^[5] Although many anatomy textbooks reported the innervation of all three heads with the radial nerve, some studies showed different results.^[1,4] They reported the axillary nerve supply to the long head of the triceps brachii. This variation could be important in traumatic nerve injuries for the evaluation of the shoulder weakness and the pre- and postoperative surgical planning of both axillary and radial nerves.^[1,6] Herein, we report a patient who was involved in a car accident that resulted in traumatic injury to the axillary nerve associated with significant paralysis of all three heads of the triceps brachii muscle. It could be

Corresponding author: Hamid Reza Farpour, MD. Shiraz University of Medical Science, Bone and Joint Diseases Research Center, Department of Physical Medicine and Rehabilitation, 7187719446 Shiraz, Iran.

e-mail: farporh@gmail.com

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possible that the main branch innervating all three heads of the triceps muscle originated from the axillary nerve in this patient.

CASE REPORT

The patient was a 21-year-old Iranian male without any previous diseases who had a car accident almost 40 days before referral to our clinic for electrodiagnosis in August 2021 due to the weakness

of the left upper limb. The patient was admitted to the hospital immediately after the accident due to transient loss of consciousness owing to a developing subdural hematoma (SDH) for close monitoring and supportive management. He also developed a fracture of the C2 vertebra. In the first days of the hospital admission, the patient felt paresthesia and numbness of the lateral part of his left shoulder and could not move his arm well. The weakness gradually

TABLE 1
Nerve conduction studies

Nerve/site	Rec/site	Latency (ms)	Amp (mv)	Velocity (m/s)
Sensory		Peak latency		
Left median	Digit III			
Palm		1.9	34.6	46.6
Wrist		3.4	32.4	
Right median	Digit III			
Palm		1.85	31.2	48.2
Wrist		3.3	30.2	
Left ulnar-wrist	Digit IV	2.9	35.5	
Right ulnar-wrist	Digit IV	2.8	38.2	
Left LAC-elbow	Forearm	3.9	10.3	
				1 point stimulation (Distance/onset latency-0.1)
Right LAC-elbow	Forearm	3.6	9.8	120/2.1-0.1=60.0
Left SRN-forearm	Thumb	2.3	38.6	120/1.9-0.1=66.0
Right SRN-forearm	Thumb	2.5	41.5	
Left MAC-above elbow	Medial forearm	3.8	10.2	
Right MAC-above elbow	Medial forearm	3.9	11.6	
Motor		Onset latency		
Left median-wrist/elbow	APB	3.2/6.6	5.8/5.4	56.0
Right median-wrist/elbow	APB	3.4/6.8	5.9/5.7	56.0
Left ulnar-wrist/elbow	ADM	3.5/7.4	8.2/8.0	60.0
Right ulnar-wrist/elbow	ADM	3.3/6.9	6.9/6.6	55.0
Left radial-forearm	EIP	4.4	3.1	
Right radial-forearm	EIP	4.1	3.6	
Left Erb's point	Deltoid/triceps	Absent	-	
Right Erb's point	Deltoid/triceps	4.4 /6.3	4.1/4.2	
				1point stimulation (Distance/onset latency-1.1)
Left Erb's point (Musculocut)	Biceps brachii	6.1	5.2	250/6.1-1.1=50.0
Right Erb's point (Musculocut)	Biceps brachii	6.3	4.9	240/6.3-1.1=46.1
F-Wave		Minimal latency		
Left median		27.2		

Rec: Recording; ms: Millisecond; Amp: Amplitude; mv: Millivolt; LAC: Lateral ante-brachial cutaneous nerve; SRN: Superficial radial nerve; MAC: Medial ante-brachial cutaneous nerve; APB: Abductor pollicis brevis; ADM: Abductor digiti minimi; EIP: Extensor indicis pollicis; Musculocut: Musculocutaneous.

TABLE 2
Needle electromyography

Muscles	Insertional activity	Positive sharp wave	Fibrillation	Amplitude	Duration	Polyphasia	Recruitment
Left FDI	Normal	-	-	Normal	Normal	Normal	Normal
Left biceps	Normal	-	-	Normal	Normal	Normal	Normal
Left FCR	Normal	-	-	Normal	Normal	Normal	Normal
Left APB	Normal	-	-	Normal	Normal	Normal	Normal
Left ADM	Normal	-	-	Normal	Normal	Normal	Normal
Left teres minor	↑	4+	4+	-	-	-	No MUAP
Left deltoid	↑	4+	4+	-	-	-	No MUAP
Left triceps	↑	3+	3+	-	-	-	No MUAP
Left EIP	Normal	-	-	Normal	Normal	Normal	Normal
Right deltoid	Normal	-	-	Normal	Normal	Normal	Normal
Right triceps	Normal	-	-	Normal	Normal	Normal	Normal
Right FCR	Normal	-	-	Normal	Normal	Normal	Normal
Right APB	Normal	-	-	Normal	Normal	Normal	Normal

FDI: First dorsal interosseous; FCR: Flexor carpi radialis; APB: Abductor pollicis brevis; ADM: Abductor digiti minimi; EIP: Extensor indicis pollicis; MUAP: Motor unit action potential.

progressed after he was discharged; therefore, the patient was referred by the neurosurgeon to our clinic for electrodiagnosis. Marked findings in the physical examination were the weakness of the left deltoid muscle and the inability to abduct the left arm, particularly at more than 90° and external rotation, and the patient could not extend his elbow. All activities of the other muscles, including shoulder flexion, elbow flexion, wrist and fingers flexion, and extension, were intact. In the sensory examination, the light touch and pinprick sensation of the left lateral part of the proximal shoulder were decreased. However, other areas, specifically posterior parts of the arm and forearm innervated by the branches from the radial nerve, were normal. The deep tendon reflex of the left triceps brachii was decreased, whereas the others, including biceps and brachioradialis reflexes, were normal and symmetric. Mild atrophy of the left deltoid and triceps brachii muscles was detected. Although the patient had a history of upper motor neuron (UMN) injury, the neurological examination did not show any signs of hypertonicity, increased deep tendon reflexes, or pathologic reflexes, such as Babinski and Hoffman. Nerve conduction studies showed an absent left compound muscle action potential (CMAP) recording from the deltoid and triceps brachii muscles while stimulating from the supraclavicular area (Erb's point) even by the near nerve recording electrode with the concentric needle. However, the other CMAP recordings from the

ipsilateral median, ulnar, musculocutaneous, and the radial (from extensor indicis) nerves were normal and did not show any significant axonal loss compared to the contralateral side. Sensory nerve conduction studies, including median, ulnar, superficial radial, medial, and lateral antebrachial cutaneous nerves, were also normal. Nerve conduction velocities of several motor and sensory nerves, such as median, ulnar, radial, and musculocutaneous nerves, were in normal ranges as well (Table 1). The temperature for the data reported exceeded 31°C. The needle electromyography (EMG) showed frank denervation (fibrillation and positive sharp waves) as well as an absent motor unit action potential (MUAP) in the deltoid, teres minor, and three heads of triceps brachii muscles. However, the needle EMG of other muscles, including latissimus dorsi, supra- and infraspinatus, biceps brachii, brachioradialis, abductor pollicis brevis, abductor digiti minimi, and wrist and finger flexors and extensors (like extensor indicis) showed normal MUAPs with full recruitment without active denervation (Table 2). Based on the above findings, electrophysiological evidence was present for the severe axonal involvement of the left axillary nerve and also the nerve branch to the ipsilateral triceps brachii muscle. The patient underwent physical and occupational therapy to maintain the range of motion of the shoulder and the elbow as well as prevent further atrophy of the involved muscles.

DISCUSSION

The posterior cord of the brachial plexus consists of two terminal branches: axillary and radial nerves.^[1] Trauma to the axillary nerve is not a rare event, which can be due to a tractional insult, blunt trauma, or shoulder dislocation.^[3,7,8] Inability to abduct the shoulder is a major debilitating result of this nerve injury.^[3,9] The radial nerve, as the largest branch of the posterior cord, supplies all extensors of the arm and forearm.^[2,4] According to many anatomy textbooks, the triceps brachii muscle, as a strong extensor of the elbow, has three heads that are all innervated by the radial nerve with a complex branching pattern.^[3] However, some studies showed different supplying patterns of the long head of the triceps muscle by the posterior cord, axillary nerve, or combination of both the axillary and the radial nerves.^[6] In our study, we reported a 21-year-old man who was involved in a car accident leading to severe axillary nerve injury accompanied by paralysis of the triceps brachii muscle. There were some clues to conclude that the injury could not be from the posterior cord or the entire branch of the radial nerve. For example, we detected normal needle EMG of all other muscles innervating by the radial nerve or the proximal branches of the posterior cord (before the bifurcation of the axillary and radial nerves) such as the latissimus dorsi muscle. In addition, recording normal CMAP from the extensor indicis muscle as well as normal superficial radial sensory nerve action potential were other clues leading to this result. Erhardt and Futterman^[1] showed the classic innervation pattern of the radial nerve for the long head of the triceps brachii in only one of the ten cadavers that were dissected. De Sèze et al.^[6] also evaluated the posterior cord and the exact origin of the motor branch of the long head of the triceps brachii in 20 cadaver specimens. They showed the axillary nerve supply in most cases. As in the studies mentioned above, no study showed different innervation of the medial and lateral heads of the triceps brachii muscle, whereas we detected significant denervation without any MUAP in all three heads of this muscle. Furthermore, the nerve branches innervating three heads of triceps brachii muscle are multiple, and they are separated from the main branch at different distances.^[2] Thus, it was very unlikely that all of them could be damaged at the same time. Nevertheless, one study by Liveson^[10] that evaluated types of nerve lesions associated with shoulder dislocation in 11 cases showed the axillary nerve involvement concomitant with the damage of

branches innervating the triceps only in one case, while more distal nerve fibers of the radial nerve were spared.

Based on electrophysiological findings and reported variations in nerve supplying pattern of the triceps brachii muscle, it could be more reasonable to conclude that the possible origin of the nerve to all three heads of the triceps brachii muscle in our case was mostly from the axillary nerve. However, it was indeed debatable whether the main trunk of the nerve branch to all three heads of the triceps brachii muscle came from the axillary nerve or the trauma separately caused the injury to the axillary nerve and nerve branches of all heads of the triceps from the radial nerve. Dissection of the injured brachial plexus and its branches in the patient had to be performed to understand the exact mechanism. In addition, according to the history of SDH in the patient, one of the other differential diagnoses that were proposed could be the weakness of the relevant muscles due to the UMN lesion. In our patient, the SDH was small and stable without any pressure effects on the underlying tissues during the patient's follow-up by brain imaging as well as periodic physical examination, which revealed no signs and symptoms of UMN lesions, such as hyperreflexia, limb hypertonicity, or pathologic reflexes. Furthermore, according to Souayah et al.'s^[11] study, denervation potentials occurring after a UMN injury such as hemiplegia were more intense in distal muscles of the upper limbs. However, our patient developed frank fibrillation potentials and positive sharp waves in a very selective and localized pattern involving only a few proximal muscles, whereas the needle EMG of the distal muscles did not show any spontaneous activities. Hence, in contrast to the normal peripheral nerve conduction studies in UMN lesions, we detected absent recordings of CMAPs from the triceps and deltoid muscles with both surface and needle electrodes while stimulating from the supraclavicular area.

The main limitation of this study is that the axillary nerve innervation has not been anatomically visualized by imaging techniques such as magnetic resonance neurography or neuromuscular ultrasound due to the lack of accessibility to this method. Thus, further studies with more evaluations should be done in the future to confirm our conclusion. According to a study by Rezzouk et al.,^[12] injury to the axillary nerve associated with the paralysis of the long head of the triceps is a sign of a proximal and severe lesion of the axillary nerve with a poor prognosis. Therefore, it is crucial to

consider concomitant paralysis of the triceps muscle in patients with axillary nerve injury.

In conclusion, although many textbooks reported the innervation of all three heads of the triceps brachii muscle by the radial nerve, some studies show the axillary nerve supply to the long head of this muscle. This study revealed it was possible to claim that the main branch innervating all three heads of the triceps brachii muscle could originate from the axillary nerve. Thus, in patients with traumatic axillary nerve injury, it is imperative to consider the paralysis of the triceps brachii muscle, which can be important in surgical management, prognosis, and nerve repair.

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