Lateral antebrachial cutaneous nerve compression after traumatic rupture of the long head of the biceps: a case series

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Background: Lateral antebrachial cutaneous (LABC) nerve compression is a rare but debilitating injury. There are limited data on the association of LABC nerve compression and proximal biceps rupture. We theorized that because of distal migration, the biceps muscle and tendon cause compression on the nerve. Methods: We present 2 cases in which patients had proximal biceps ruptures with tendon retraction and developed neurologic symptoms associated with the LABC nerve. To demonstrate our theory, we performed a cadaveric experiment. After making an incision to expose the entire biceps muscle and the musculocutaneous nerve and its branch into the LABC nerve, we marked each structure in 2-cm increments with a marking pen. The long head of the biceps was then cut to simulate a proximal biceps rupture. Results: The relationship between the 3 structures was then studied, showing no change in position of the musculocutaneous nerve or LABC nerve. The biceps muscle and tendon had migrated distally toward the LABC nerve, demonstrating compression of the nerve. Conclusion: Proximal biceps tears commonly occur from trauma as well as iatrogenically after a biceps tenotomy for treatment of biceps tendinopathy. However, it is unusual for neuropathy of the LABC nerve to occur. Patients who develop neuropathy associated with the LABC nerve after a proximal biceps rupture can be effectively treated with LABC nerve decompression and biceps plasty. We found that this was an effective surgical treatment of LABC neuropathy without the need for proximal biceps tenodesis. Level of evidence: Level IV, Case Series, Treatment Study. Copyright © 2014 Journal of Shoulder and Elbow Surgery Board of Trustees. Keywords: Lateral antebrachial cutaneous nerve; biceps; elbow pain

Lateral antebrachial cutaneous (LABC) nerve compression is a rare but debilitating injury. It most commonly is manifested as numbness or neuropathic pain over the elbow and lateral aspect of the forearm. Patients typically have a history of injury or overuse of the elbow involving extension and maximal pronation of the elbow with activities such as weightlifting and tennis. Narasanagi initially defined the diagnosis, and Bassett and Nunley later described the pathologic process in which the nerve is compressed by the biceps aponeurosis and tendon against the fascia of the brachialis muscle. There are limited data on the association of LABC nerve compression and proximal biceps rupture. Brogan et al described it as traction neuritis, rather than entrapment, as they believed that the biceps displaced the

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nerve laterally. Therefore, their treatment algorithm involved a proximal biceps tenodesis as the mainstay of treatment in addition to neurolysis of the LABC nerve. We present 2 cases of LABC neuropathy after traumatic proximal biceps tendon rupture. Both cases were treated with decompression of the LABC nerve distally, including a biceps plasty without proximal biceps tenodesis. We theorized that because of distal migration, the biceps muscle and tendon cause compression on the nerve (Fig. 1). To demonstrate, we simulated a traumatic biceps rupture in a cadaver specimen and investigated the relationship between the biceps muscle and tendon and the LABC nerve to provide more evidence to support this theory.

Case report 1

A 58-year-old, right-hand dominant male manual laborer, with no previous history of shoulder or elbow injury, presented to the clinic with a 2-month history of left shoulder pain after he felt a “pop” while cutting and hauling wood. Before his orthopedic presentation, he presented to his primary care physician and was given anti-inflammatory medication. He was referred for continued left shoulder pain. On examination, he had full range of motion and normal abduction/external rotation strength; the Hawkins impingement sign was present. The biceps muscle belly was prominent and had migrated distally. Because of the patient’s continued pain, magnetic resonance imaging (MRI) of the shoulder was ordered. The MRI showed rotator cuff tendinosis and an intra-articular rupture of the long head of the biceps tendon with retraction and an empty bicipital groove. He was then given a subacromial injection and was prescribed a structured physical therapy program. The patient returned 6 weeks after his initial presentation with resolution of shoulder pain but had developed point tenderness at the lateral aspect of the distal biceps with paresthesias at the lateral aspect of the forearm without numbness. He had decreased grip strength because of pain. At that time, he was diagnosed with compression of the LABC nerve, and an injection of corticosteroid was performed at the point of maximum tenderness. The patient reported complete relief for approximately 2 days; however, the pain returned. He tried rest, ice, a brace, anti-inflammatory medication, and a steroid injection without complete relief. A second MRI study was performed of the elbow that showed a mass consistent with a hematoma with enhanced granulation tissue at the proximal portion of the biceps muscle.

Three months later, the patient opted for surgical intervention that included exploration and decompression of the LABC nerve at the distal musculotendinous junction with biceps plasty.

At the time of surgery, a longitudinal incision was made over the lateral aspect of the distal biceps tendon. The LABC nerve was identified as it exited just lateral to the biceps musculotendinous junction (Fig. 2). The nerve was decompressed both proximally and distally, and a biceps plasty was performed by taking the lateral side of the proximal biceps tendon and sewing it over to the medial aspect of the tendon (Fig. 3). Of note, an additional longitudinal incision was made at the proximal biceps muscle to decompress the hematoma.

The patient had complete resolution of symptoms immediately after surgery. He was released to work without restrictions 4 weeks later. He has had no reported return of symptoms during the 13-month follow-up period.

Case report 2

A 50-year-old male, right-hand dominant machine operator, with no previous history of shoulder or elbow injury, presented to the orthopedic clinic with a 6-month history of right shoulder pain. He was lifting a heavy bag of fertilizer and felt a painful “pop” in his anterior shoulder. The patient reported a change in contour and intermittent pain in his biceps muscle; Tinel sign was absent at the LABC nerve lateral to the biceps tendon. A nerve conduction velocity test and MRI of the elbow were ordered. The nerve conduction velocity test confirmed compromise of the LABC nerve at the distal aspect of the arm/elbow, and MRI revealed moderate distal biceps plasty without proximal biceps tenodesis. We theorized that because of distal migration, the biceps muscle and tendon cause compression on the nerve (Fig. 1). To demonstrate, we simulated a traumatic biceps rupture in a cadaver specimen and investigated the relationship between the biceps muscle and tendon and the LABC nerve to provide more evidence to support this theory.
tendinosis without a tear. At that point, a local anesthetic injection was performed at the origin of the paresthesias; however, no significant relief was obtained with the injection. Because of the long-standing symptoms, the patient decided to undergo operative intervention including exploration and decompression of the LABC nerve at the distal musculotendinous junction with biceps plasty.

Once again, a longitudinal incision was made over the lateral aspect of the distal biceps tendon. The LABC nerve was identified as it exited just lateral to the biceps musculotendinous junction. The nerve was decompressed both proximally and distally, and a biceps plasty was performed by taking 7 to 8 mm of the lateral aspect of the proximal biceps tendon and sewing it over to the medial aspect of the tendon.

Postoperatively, the patient had immediate resolution of symptoms. He had no numbness or tingling in his right arm. He returned to work 4 weeks after surgery with complete resolution of pain and full range of motion. He was examined 3 months after surgery with no return of numbness or tingling in the right forearm.

**Cadaveric experiment**

A fresh right upper extremity cadaveric specimen was mounted by clamps to a tower (Fig. 4). An anterior incision was made to expose the entire biceps muscle and the musculocutaneous nerve and its branch point into the LABC nerve. Special attention was paid to the relationship distally between the LABC nerve and the biceps tendon. Once the anterior structures were adequately exposed, the nerve, biceps muscle and tendon, and soft tissue around the structures were marked in 2-cm increments with a marking pen. This was done to study the relationships between these 3 structures. The long head of the biceps muscle was then sharply cut to simulate a long head of the biceps rupture. The relationship between these 3 structures was then studied, showing
The LABC nerve is the sensory terminal branch of the musculocutaneous nerve that runs in the upper arm, where it is protected by the biceps muscle. It then emerges from beneath the lateral margin of the biceps about 2 to 5 cm proximal to the elbow flexion crease, where it pierces the brachial fascia and becomes subcutaneous. At this point, it travels behind the cephalic vein and divides into volar and dorsal branches, providing sensation to both the radial volar and radial dorsal aspects of the forearm. Anatomic variations in the course of the nerve have been reported. Wongkerdsook et al., in a study of 96 upper extremities in 48 adult cadavers, found that the LABC nerve pierced the deep fascia distal to the interepicondylar line in 84.4%, with mean distances of 1.8 cm and 1.2 cm (male and female, respectively). Rosen et al. found that in 2 cases, the nerve passed through the biceps muscle directly before piercing the deep fascia. However, even with these anatomic variations, the most commonly reported area of entrapment is at the point where the nerve emerges from beneath the biceps tendon and pierces the deep fascia, proximally or at the elbow crease. The lateral margin of the biceps aponeurosis exerts a compression force on the LABC nerve as the elbow extends, and the nerve is caught between the biceps tendon and the brachialis fascia.

There have been few reports to describe this compressive neuropathy. Bassett and Nunley described compression of the LABC nerve in a series of 11 patients. Their patients presented with anterolateral elbow pain and were separated into those with acute presentation, who had burning dysesthesias along the radial aspect of the volar forearm, and those with chronic presentation, who had vague aching discomfort in the anterior region of the elbow joint. All patients presented with direct tenderness over the area where the LABC nerve exits from beneath the biceps tendon, and 10 of the 11 patients had a decrease in sensation along the radial aspect of the volar forearm.

Naam and Massoud also described 23 patients with compression of the LABC nerve. In their series, all patients complained of pain along the anterolateral aspect of the involved elbow, with 6 patients complaining of numbness along the radial volar aspect of the distal forearm. Patients had localized tenderness at the anterolateral aspect of the elbow about 3 to 5 cm proximal to the elbow flexion crease along the lateral margin of the biceps muscle. Grip strength averaged 78% of the contralateral side, and pinch strength averaged 83% of the contralateral side. Of these patients, 7 were treated nonoperatively, whereas 16 were treated with surgical exploration and decompression of the LABC nerve. All patients recovered functionally, with 2 of the 16 patients reporting continued mild pain.

Other injuries to the LABC nerve that have been described include injury by phlebotomy, windsurfing, compression by a handbag, slam-dunking a basketball, and positioning while under general anesthesia. Also, there have been reports of compression distal to the elbow crease, as the nerve travels through the superficial antebrachial fascia. Treatment consisted of both operative and nonoperative management. Nonoperative management included rest, elbow pads, physical therapy, and nonsteroidal anti-inflammatory medications. Diagnostic as well as therapeutic injections, including local anesthetic with or without steroids, were also attempted. Surgical interventions included surgical decompression and exploration with some variation. This included resection of the lateral edge of the biceps versus creation of a triangular flap that was reflected medially and sutured to the remaining tendon.

A specific instance in which compression of the LABC nerve occurred after a proximal biceps rupture was described by Brogan et al. In their case report, the patient suffered a traumatic proximal biceps rupture and then developed paresthesias and burning pain in the lateral forearm and palm. The patient initially underwent nonoperative treatment, but symptoms did not resolve. Surgical intervention was then performed with a medial arm incision along the posterior border of the biceps, where they identified the musculocutaneous nerve displaced laterally and observed that the shortened biceps was causing a mass effect on the nerve. They then performed a proximal biceps tenodesis and a neurolysis of the LABC nerve. The patient had improvement in his allodynia within hours of the surgery and showed continued improvement during 10 months.

In our study, we also observed a distal migration of the biceps muscle, causing a compressive neuropathy of the LABC nerve at the musculotendinous junction. By performance of a neurolysis and biceps plasty, the symptoms of both patients resolved, and pain relief was maintained at 3
to 13 months. We believe that a biceps tenodesis is not crucial for symptom relief. We did perform a cadaveric experiment and observed results similar to our intraoperative findings, in which compression of the LABC nerve occurred as the biceps muscle migrated distally.

**Conclusion**

Proximal biceps tears commonly occur from trauma as well as iatrogenically after a biceps tenotomy for treatment of biceps tendinopathy; however, it is unusual for neuropathy of the LABC nerve to occur. What causes these neuropathic symptoms in some patients and not in others? Further study is needed to delineate if there is a crucial distance of migration of the biceps muscle that leads to these symptoms. Once these symptoms do occur, however, we think that on the basis of our 2 case reports, LABC nerve decompression with biceps plasty is an effective surgical treatment of LABC neuropathy without the need for proximal biceps tenodesis.

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