CASE REPOSITORY

Treatment of Cervical Spondylotic Amyotrophy With Nerve Transfers

Charmaine Baxter, BEng, MD,* Thomas A. Miller, BSc, MD,† Douglas C. Ross, MEd, MD,‡ Christopher Doherty, MPH, MD‡

Cervical spondylotic amyotrophy is characterized by severe, proximal upper extremity weakness including an inability to abduct the shoulder and flex the elbow. Treatment using both medical and surgical decompression approaches has produced variable results. This paper reports the use of nerve transfers (spinal accessory to suprascapular, flexor carpi ulnaris fascicle of ulnar to biceps motor branch, radial nerve branch to triceps to axillary) to restore shoulder and elbow function in a case of unilateral cervical spondylotic amyotrophy involving C5 and C6 myotomes. Evidence of regeneration was observed on electromyography as well as clinically at 5 months postoperatively. At 3 years after surgery, recovery of elbow flexion and shoulder abduction was Medical Research Council grade 4/5 with improved external rotation and considerably improved patient-rated Disabilities of the Arm, Shoulder and Hand scores. We propose that nerve transfers be considered along with other reconstruction modalities in the treatment of cervical spondylotic amyotrophy. *(J Hand Surg Am. 2018;■(■):1.e1-e4.*

Online Exclusive

CERVICAL SPONDYLOSIS CAUSING upper extremity muscle atrophy with minimal sensory disturbance is a rare disorder known as cervical spondylotic amyotrophy (CSA).[1−3] CSA is typically a unilateral condition predominantly affecting males, with age of onset ranging from the third to sixth decade. It is classified according to the most predominantly affected muscle groups.[2,3] "Proximal CSA" presents with atrophy in the C5−C6 myotomes, whereas "distal CSA" involves the C7−C8 and T1 myotomes. The pathophysiology is thought to be mechanical impingement or ischemic injury to the ventral root or anterior horn cell.[2,3] CSA is a diagnosis of exclusion with amyotrophic lateral sclerosis, Parsonage-Turner syndrome, and Hirayama's disease to be considered in the differential diagnosis. Whereas cervical radiculopathy is characterized by pain and marked sensory disturbances in a dermatomal distribution, CSA is painless with few, if any, sensory symptoms.

CSA generally follows a self-limited course with weakness not progressing beyond a few myotomes. After initial onset, the symptoms typically stabilize and treatment includes both nonsurgical and surgical management. Nonsurgical treatment modalities include physical therapy, immobilization of the neck, cervical traction, and vitamins B12 and E.[1,2]

Surgical treatments have also been used to restore function in eligible patients. Different types of decompressive procedures have been reported, including anterior cervical decompression and fusion or laminoplasty, with or without foraminotomy. Case

*From the *Division of Plastic Surgery, McMaster University, Hamilton; the †Department of Physical Medicine and Rehabilitation; and the ‡Division of Plastic Surgery, Roth-McFarlane Hand and Upper Limb Centre, St Joseph's Health Care, Western University, London, ON, Canada.*

Received for publication March 19, 2017; accepted in revised form December 20, 2017.

No benefits in any form have been received or will be received related directly or indirectly to the subject of this article.

Corresponding author: Douglas C. Ross, MEd, MD, Roth-McFarlane Hand and Upper Limb Centre, St Joseph's Health Care, Room D0-210, 268 Grosvenor Street, London, ON, N6A 4L6, Canada; e-mail: dross@uwo.ca.

0363-5023/18/■ ■-0001$36.00/0
https://doi.org/10.1016/j.jhsa.2017.12.020

series reporting outcomes of these treatments are small but document improved function in a substantial number of patients, particularly those with proximal type CSA.[1,2,4,5] However, surgery may be complex and time from surgery to evidence of recovery is typically prolonged, likely due to the distance from the site of decompression to motor endplates. Factors predicting poor outcomes from anterior cervical decompression and fusion and other cervical decompressive strategies include <30% of compound motor action potential in the deltoid and biceps relative to the contralateral, unaffected side[6] or Medical Research Council power of <2 and prolonged duration of symptoms.[7] More recently, reconstructive muscle transfers have been reported to restore elbow and shoulder function in those who were ineligible for, or had continuing dysfunction with, traditional surgical management.[3]

We propose that nerve transfers may also be considered as a potential therapy to restore function in patients with CSA, especially when denervation times are prolonged and the weakness causes a significant impact on function. This case report highlights the novel approach of using established nerve transfers as a modality to restore function in these patients.

CASE REPORT

A 70-year-old, right-handed, retired social worker was referred with a 6-month history of left shoulder and arm weakness. He denied any history of numbness, paresthesiae, pain, trauma, stretch injuries, or constitutional symptoms. His medical history included chronic neck and lower back pain, with no recent exacerbations, controlled hypertension, and benign prostatic hyperplasia.

Examination of his left shoulder showed marked atrophy of the C5−C6 musculature, specifically the deltoid, biceps, supraspinatus, and infraspinatus muscles. Passive range of motion was unrestricted in all planes with no evidence of capsular tightness. Medical Research Council grade strength of supra- and infraspinatus was 1/5, deltoid 0/5, biceps 2/5, brachioradialis 0/5, triceps 4/5, and trapezius 5/5. Other neurologic function was normal.

Investigations at the time of his presentation included plain radiographs, magnetic resonance imaging (MRI) of the cervical spine and left shoulder, and serial electromyogram (EMG) tests and nerve conduction studies. Plain radiographs were normal. MRI of the shoulder showed moderate-to-severe fatty atrophy and intramuscular edema of supraspinatus, deltoid, and medial aspect of subscapularis with a mild change in teres minor. There was no evidence of a rotator cuff tear. MRI of the cervical spine indicated multilevel spondylotic changes most severe at the C4−C5 and C5−C6 levels with bilateral osteophyte narrowing of the neural foramina and exiting root involvement most severely affecting the left nerve root at C5−C6 (Fig. 1). Mild flattening of the cord due to the disc protrusion was noted at these levels, however, more so on the left at C5−C6.

Although during his initial course of nonsurgical therapy, EMG revealed profuse denervation of the C5−C6 musculature with single motor units present in the deltoid, biceps, and brachioradialis, there were some nascent units present in the deltoid and infraspinatus supporting a nonsurgical approach (including physiotherapy and cervical traction). However, his motor function continued to decline. Neurosurgical consultation for possible spine surgery concluded that there was a limited possibility for return of motor function with foraminal decompressive surgery given the prolonged period of atrophy.

Eighteen months after the onset of the symptoms, follow-up EMG demonstrated no motor units in either the deltoid or infraspinatus and a single motor unit in the biceps with no nascent potentials. There was no denervation and normal recruitment in the potential donor nerve distributions (trapezius, triceps, and first dorsal interosseous muscles). The patient underwent nerve transfers typically used to reconstruct traumatic C5−C6 brachial plexus injuries. At surgery, all 3 recipient nerves (axillary, suprascapular, motor branch to brachialis) were exposed and stimulated with a hand-held nerve stimulator at 2 mA; no contraction was produced in any of the affected muscles. All recipient muscles within the surgical fields demonstrated the typical “washed out” appearance of chronically denervated muscle; however, none appeared fibrotic. The branch of the radial nerve to the long head of triceps was chosen as the donor for the axillary nerve based on diameter and length. This nerve was transected and transferred end-to-end to the anterior branch of the axillary nerve (posterior branch and branch to teres minor were left intact but not reconstructed). The spinal accessory nerve was delineated including at least 2 descending branches serving the medial scapular border, which were left intact. The distal spinal accessory nerve was transected and repaired end-to-end to the suprascapular nerve without the use of nerve grafts. No distal decompressions, such as at the suprascapular notch, were performed. Finally, an internal neurolysis of the ulnar nerve adjacent to the brachialis