Patients with thoracic outlet syndrome can be treated with osteopathic manipulative treatment (OMT) to alleviate dysfunction and restriction of the pectoralis minor muscle (PMM) and the resulting compression of the brachial plexus. Neuromuscular ultrasonography (US) can demonstrate abnormalities in the thoracic outlet that are amenable to OMT and can be used to monitor intervention. The present report identifies PMM deformation and brachial plexus compression in a 32-year-old woman with thoracic outlet syndrome who was treated successfully with OMT. Neuromuscular US results were used to measure the degree of PMM deformation with the pectoral bowing ratio and confirm the diagnosis. Osteopathic manipulative treatment was applied and monitored using neuromuscular US to confirm that the operator’s manipulating hand had direct contact with the PMM. Symptoms abated immediately after treatment. Results of a second neuromuscular US examination showed that the pectoral bowing ratio decreased into the normal range and thus confirmed that PMM deformation had resolved.

J Am Osteopath Assoc. 2011;111(9):543-547

This case report was presented as a poster during the midyear meeting of the American Osteopathic College of Physical Medicine and Rehabilitation in Phoenix, Arizona, on April 16, 2011.

Financial Disclosures: None reported.

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Submitted January 29, 2011; accepted June 20, 2011.

Ultrasonography-Guided Osteopathic Manipulative Treatment for a Patient With Thoracic Outlet Syndrome

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Thoracic outlet syndrome (TOS) frequently involves compression of the neurovascular bundle by the pectoralis minor muscle (PMM).1-6 In my clinical experience the past few years, I have documented such compression with neuromuscular ultrasonography (US) that correlates with patient symptoms (B.M.S., unpublished data, 2011). Neuromuscular US in patients with TOS can be used to identify deformation of the PMM; that deformation can be quantified by measuring the degree of angulation, or bowing, with the pectoral bowing ratio. A similar bowing ratio has been used in carpal tunnel syndrome to measure deformation of the transverse carpal ligament by the bulging contents of the carpal tunnel.7 In TOS, however, the neurovascular bundle is tethered under the tight and shortened PMM; this process causes deformation.

I previously demonstrated thermographic abnormalities that were used to confirm the diagnosis of TOS.1 I applied a specific osteopathic manipulative treatment (OMT) protocol with myofascial release of the PMM, which led to alleviation of upper limb symptoms and resolution of abnormalities on thermographic imaging.1,8

Neuromuscular US is a fairly novel imaging modality for examining nerve structures within the thoracic outlet area.9,10 Neuromuscular US has unique applications for TOS. As Caress and Walker11 noted, “evaluation of proximal nerves is often problematic using electrodiagnostic techniques and may be more easily studied with ultrasound.” In the current report, I present a case of TOS that was successfully managed with US–documented OMT. I also illustrate the manipulating hand (finger) as it contacted the offending dysfunctional structure and led to symptom resolution. Abnormalities found on repeated neuromuscular US images reverted to normal.

Report of Case

A 32-year-old woman presented with complaints of left upper limb pain, numbness, and weakness for the past 18 months. Numbness involved all of the digits of the left hand, especially the medial 3 fingers. Clinical examination revealed normal findings, except for palpatory restriction over the left PMM, left shoulder protraction, and a positive thoracic outlet stress test with hyperabduction. Results of an electrodiagnostic test were normal.

Neuromuscular US was performed the same day in the left infraclavicular region with an M-Turbo US unit, 6-13 MHz
Images revealed that the medial and lateral cords of the brachial plexus contacted the posterior edge of the PMM during arm abduction as upper limb symptoms developed and increased (Figure 1). Symptoms began at 70° of abduction and progressively increased. The pectoral bowing ratio was abnormally high (more than 10%; B.M.S., unpublished data, 2011) and measured 13.6% at 145° abduction, confirming a diagnosis of TOS.

Osteopathic manipulative treatment was provided using the myofascial release technique directed to the PMM (Figure 2) for 3 to 4 minutes. Immediately after treatment, neuromuscular US was repeated with the arm in abduction, and results revealed that the pectoral bowing ratio decreased into the normal range (3.6%; normal, <10%; B.M.S., unpublished data, 2011). Symptoms with the arm in abduction abated immediately, even at 120° of abduction (Figure 3).

Comment
As I noted in previous articles,1,8 I have successfully implemented and confirmed the use of OMT to treat patients with TOS and to alleviate symptoms that correlate with resolution of abnormalities found by means of thermography.1,8 Although thermography is no longer used as a diagnostic tool in most medical facilities, especially for disorders such as TOS, neuromuscular US imaging has recently gained acceptance as a diagnostic method for nerve compression syndromes.9-13 In addition, I have combined neuromuscular US with dynamic stress testing to assist in the diagnosis of TOS (B.M.S., unpublished data, 2011).

Furthermore, I previously performed limb manipulation during cadaver dissection of the thoracic outlet and observed sucher • case report

Figure 1. Infraclavicular region in the initial neutral adduction position (A) and progressive abduction at 120° (B) and 145° (C) in a 32-year-old woman who presented with left upper limb pain, numbness, and weakness. The upper portion (horizontal layer) of the image is the skin (brighter, white, or hyperechoic) and subcutaneous tissue (darker, hypoechoic, with white or hyperechoic streaks). The next layer is the pectoralis major muscle (grayish, relatively hyperechoic to the subcutaneous layer), which is wedge-shaped and tapering to the left. The third layer is the thinner pectoralis minor muscle, which is oriented transversely as it angles upward to the left of the image and is slightly darker (more hypoechoic) than the pectoralis major muscle. The hypoechoic circular structures below the pectoralis minor are the axillary artery (on the left) and vein (on the right). The medial cord of the plexus is the small hyperechoic circular structure to the right of the artery, and the lateral cord is the small hyperechoic circular structure to the left of the artery (B, white vertical arrow; C, without the arrow). The pectoralis minor muscle is initially very straight in the neutral position (A), but as abduction progresses, there is bowing or indentation from the lower edge (posterior) by the neurovascular bundle, moderate at both 120° (B) and 145° (C). In panel C, the pectoral bowing ratio is calculated by measuring a line transversely from medial to lateral across the posterior arc of the pectoralis minor muscle (line A-A), drawing a vertical line (B-B) perpendicularly from line A-A to the apex of the pectoralis minor muscle, and dividing B-B by A-A. In this patient, the ratio was 13.6% (0.23/1.69).
For the present case, however, video capture of the manipulation was obtained, and it confirmed that the manipulating hand (finger) was in contact with the PMM, which was the primary anatomic structure of interest (http://www.jaoa.org/cgi/content/full/111/9/543/DC1). It can be noted that the manipulating finger creates marked anterior deformation of the PMM, which verifies that firm pressure is being applied focally. I have previously discussed this type of direct pressure as a required component of successful OMT for patients with TOS—the pressure “releases or ‘frees up’ focal myofascial adhesions/restrictions, and ‘guides’ the myofascial unit back into more proper alignment and function.”

I have also previously discussed the pathologic mechanics in TOS, a key element of which involves the theory of progressive PMM shortening. Alterations of posture caused by tightening of the PMM lead to scapular protraction, forward “collapse” of the shoulder girdle, and relative closure of the thoracic outlet. Entrapment of the neurovascular bundle then leads to upper limb symptoms because these structures are stretched as they “hook” underneath the PMM just inferior to the coracoid attachment site. As noted by Simons et al, “stretch and torsion of the brachial plexus and axillary artery can occur as they hook beneath the pectoralis minor muscle where it attaches to the coracoid process.” (p850) The illustration they provide demonstrates the neurovascular bundle indenting the PMM from below (posterior) in the abducted arm position, resulting in nerve entrapment. Simons et al described how both the medial and lateral cords can be impinged or compressed either together, causing symptoms in the entire hand, or separately, resulting in only medial or lateral hand symptoms. I have also illustrated indentations that when the arm is abducted, external rotation causes the brachial plexus to contact the posterior edge of the PMM. Arm rotation internally causes the plexus to “drop away” from the PMM, thus allowing vigorous manipulation of the muscle without irritating the neurovascular structures. The usual position for most overhead activity (such as grooming) is achieved with external rotation, which puts the plexus at risk for irritation or impingement by the PMM. Close-up views of anatomy dissections confirm the proximity relationship of the brachial plexus and PMM and reveal mild “indentation” at even 90° of abduction. Regrettably, medical history regarding possible TOS symptoms on the cadaver is unavailable, and feedback for symptom provocation during stress maneuvers requires a living specimen.

I obtained digital video recordings for some patients with TOS and demonstrated that the brachial plexus (and axillary artery) appears to press into and indent the posterior edge of the PMM during arm abduction. Unfortunately, it can be challenging (and time consuming) to create a real-time dynamic recording because increasing abduction causes the anterior axillary fold to “push” the US transducer off the area of interest and the image is easily lost. Thus, video recordings are not routinely obtained during the typical neuromuscular US examination protocol.
tation of the PMM by the neurovascular bundle pulling against the posterior muscular edge during arm abduction and resulting in nerve entrapment.

Imaging changes noted in the PMM during stress testing can be explained by deformation created from stretching force as the neurovascular bundle is pulled against the posterior edge of the muscle during arm abduction. Because the PMM is shortened in patients with TOS, there is no laxity to allow the motion without some indentation from the pressure of the neurovascular bundle impinging against the muscle. Neuromuscular US reveals an upward (anterior) bowing of the center of the PMM while the sides appear to angle downward (posterior), especially on the left side (proximal or superior) near the coracoid attachment (Figure 1B and Figure 1C).

The amount of bowing can be objectively measured as a pectoral bowing ratio (B.M.S., unpublished data, 2011), which is typically greater in patients with TOS (more than 10%) than in healthy individuals (less than 10%). This effect is similar to the palmar bowing ratio of the flexor retinaculum measured with magnetic resonance imaging in patients with carpal tunnel syndrome, who have been shown to have palmar bowing ratios greater than 10% compared with ratios less than 10% in healthy controls. The palmar bowing ratio of the flexor retinaculum was considered a useful parameter that correlated significantly with symptoms in patients with carpal tunnel syndrome, and it appeared related to enlargement of structures within the carpal tunnel that caused the retinaculum to be “pushed in a volar direction.” In TOS, the bowing is like a sling under the PMM that creates a pulley effect from the neurovascular bundle that is tethered at each end, and this tethering effect has also been demonstrated—but not objectively measured—in previous publications.

An understanding of the pathomechanics that contribute to development of TOS can be applied to treatment. In patients or individuals without TOS, the plexus simply “glides” under the PMM, avoiding impingement or indentation of the muscle. This observation supports the nerve gliding techniques of Totten and Hunter, who restored normal posture and mobility of the brachial plexus in patients with TOS as a method of alleviating symptoms. Case studies have demonstrated that OMT applied to the thoracic outlet can improve posture, relieve symptoms, and resolve thermographic abnormalities. A vital OMT technique for management of TOS is myofascial release of the PMM, which can restore the scapula to its normal position. These prior studies were not able to include neuromuscular US of the thoracic outlet to monitor changes in the neurovascular bundle and PMM before and after treatment.

Supplemental treatment of patients with TOS with stretching exercise for the PMM can improve results but is difficult because the PMM is a deep muscle and there is no simple method to separate the origin from the insertion. I developed a method that involves hanging by the arm, but not all patients can tolerate full abduction and partial body weight distraction on the shoulder joint simultaneously. In addition, simply separating the ends of the muscle does not ensure that the trigger points will release because the muscle fibers on either side of the trigger point may elongate as the trigger itself remains unchanged. Therefore, use of myofascial release applied directly to the PMM is a rapidly effective method to achieve lengthening and release. Unlike longitudinal stretch, application of vigorous direct force is perpendicular to the muscle fibers, and a “back and forth stripping” maneuver essentially “milks” out focal restrictions in the triggers, as shown in the video recording (http://www.jaoa.org/cgi/content/full/111/9/543/DC1).

Conclusion

Neuromuscular US of the thoracic outlet provides a new modality for clinicians to directly observe and monitor the effects of the manipulating hand at work. The PMM is a thin, flat muscle that is not always easy to identify, yet it is a primary OMT target for patients with TOS. Enhanced accuracy of treatment can lead to improved alleviation of somatic dysfunction and a better outcome in difficult cases. Neuromuscular US also allows the clinician to re-evaluate the effects of OMT in a noninvasive manner and without discomfort that accompanies electrodagnostic testing. Confirming a good clinical outcome objectively can also improve long-term management.

Such new understandings of the pathomechanics involved with TOS lend confirmation to previous studies that advocate aggressive OMT and stretching as essential components of management. It may be through application of neuromuscular US during the treatment process that a new clinical window to more effective management of TOS will be opened.

References

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**CASE REPORT**

**JAOA Peer Reviewer Seminar**

On Tuesday, November 1, 2011, JAOA—The Journal of the American Osteopathic Association will host a peer reviewer seminar during the American Osteopathic Association’s 116th Annual Osteopathic Medical Conference and Exposition in Orlando, Florida. Osteopathic physicians, researchers, and others interested in best practices in peer review are invited to attend this event, which will be held from 1:15 PM to 3:15 PM. The room will be announced at a later date. Contact JAOA staff at jaoa@osteopathic.org for more information.