Evaluation and Treatment of Apparent Reactive Tendinopathy of the Biceps Brachii

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Overuse injuries are prevalent in sports medicine, and while an exact prevalence is unknown, it has been estimated this presentation accounts for 30–50 % of the pathologies treated in sports medicine[1] and commonly result in disability in the general population.[2] Athletic trainers often treat patients with complaints of pain and dysfunction without a clear mechanism of injury and an insidious onset. A patient presenting with pain at the location of a tendon associated with an supposed overuse or overload mechanism has commonly been diagnosed with "tendonitis" or "tendinitis," implying an inflammatory process is occurring.[2,3] Literature evidence indicates tendons exposed to these mechanisms present with little or no active inflammation leading to concerns about the efficacy of traditional employed intervention aimed at treating the inflammatory process perceived to be present in these cases.[2–6]

Key Points

- Positional Release Therapy can produce clinically significant improvement without altering participation in physical activity.
- Clinicians must consider the role of spinal function and central sensitization in the diagnosis and treatment of tendinopathy.
- Outcomes measures used to assess tendinopathy should encompass a variety of factors that are patient-centered.

In recent years, the term *tendinosis* has gained traction as a more accurate diagnostic term for many patients.[2,7] Variability exists in the description of this term as well and debate continues over whether the tendon is in a state of true irreversible degeneration or if it is simply in a failed healing phase.[8] Clinically, however, the diagnosis of this type of pathology has now shifted to the term *tendinopathy*, which describes a variety of tendon conditions that may result from apparent overuse and/or have an insidious onset.[2,3]

Another term has also recently appeared in the literature in regards to treating tendon pathology at the lateral epicondyle of the humerus. The term—lateral epicondylalgia—is defined as any lateral epicondylar pain without reference to a direct underlying cause of tendon disruption as the source of pathology.[9] Using this premise, the term *tendinalgia* could be applied to the presentation of tendon pain throughout the body. Tendinalgia, while still fitting into the tendinopathy paradigm, differs from the other tendon terms in that the classification is made when the main complaint or finding during clinical exam is pain at a tendon without reference to a predicted stage of tissue pathology. The clinician acknowledges pain may be the cause or result of dysfunction and may be associated with edema, but the patient would not display other signs of inflammation or degeneration during the clinical exam. Other researchers have suggested a model of tendon pathology that acknowledges the

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complexity of tendinopathy presentation and recommend a continuum that is constantly adjusted to improve prevention, diagnosis, and treatment of this pathology. The proposed tendinopathy model focuses on three stages of injury progression: (a) reactive tendinopathy, (b) tendon disrepair, and (c) degenerative tendinopathy, which result from both mechanical and structural factors.[2,8]

Reactive tendinopathy is defined as a noninflammatory state that results from acute mechanisms involving tensile or compressive loads producing in thickened tendon, reduced stress tolerance, and increased stiffness. In this state, cell matrix remodeling precedes clinical symptom presentation. Local blood circulation becomes impaired and results in impaired metabolic activity, altered oxygen transport, inefficient molecular cross-linking, and tissue disrepair.[8] Recent theories support the model, as suggested pain mechanisms within this continuum may result from biochemical stimulants/irritants and impaired blood vessel regulation.[2,8] Reactive tendinopathy is more common in young patients, typically results from acute overload on a structure, and presents with tendon thickening, swelling, pain, and stiffness. When using this tendinopathy continuum, it is important for clinicians to have clinical techniques that address the chemical, neural, and metabolic factors associated with intratendinous modifications.[2,8,10] A potential therapeutic option in this stage is addressing somatic dysfunction. Somatic dysfunction is typically defined as an impaired or altered healing response in the skeletal, joint, or myofascial systems and their related elements. In acute cases, this dysfunction is thought to involve tissue trauma, microscopic hemorrhage, local tissue edema, nociceptive involvement, increased neuropeptide release, and diminished tissue pliability.[11,12]

Positional release therapy (PRT), also referred to as "strain-counterstrain" or "counterstrain," is an indirect therapeutic treatment designed to treat acute, subacute, and chronic somatic dysfunction that fits the continuum. PRT uses tender points (TPs) and a position of comfort (POC) to relax the muscle-spindle mechanism producing a sustained neuromuscular contraction by placing the strained tissue in a relaxed shortened state.[11,12] When using PRT, the gross POC is identified by positioning the patient's body/extremity to shorten/relax the strained tissue. The treatment POC for is then determined by fine-tuning (e.g., rotation, joint compression) the position until a change (e.g., softening, pulsing) in the TP is palpated. Once the POC is identified, this position is held for a period of time (e.g., 90 s) to facilitate restoration of normal tissue length and function.[11,12] The technique is typically performed by treating the most severe TP first, followed by the more proximal or medial TPs before distal or lateral TPs, the area of the greatest accumulation of TPs, and the middle TP in a row of equally sensitive TPs. PRT has relatively few contraindications and offers the potential of being an effective treatment for a variety of soft-tissue injuries.[11,12,13]

The purpose for this case report was to assess the effectiveness of PRT in treating the clinical presentation of a patient that met the criteria for reactive tendinopathy. Questions included the following: (a) Will a patient who displays the clinical symptoms of a reactive tendinopathy present with tender points (TPs)? (b) Does PRT effectively decrease tenderness to palpation measured by the Numerical Rating Scale (NRS) at the TP site? (c) Does PRT decrease the level of disablement, as measured by the NRS and the Disablement in Physically Active (DPA) scale, in a patient with this clinical presentation?

Case Description

A 21-year-old female swimmer presented with complaints of general shoulder discomfort of approximately 10 days duration that she attributed to muscular soreness from beginning preseason swim training activities. However, as her symptoms had significantly worsened over the previous 3 days and had localized to the area of her biceps tendon, she sought out treatment due to a previous history of biceps tendinitis. The patient had been diagnosed with biceps tendinitis during each of the previous three swim seasons and conveyed that her current symptoms and presentation felt similar to those cases. In the previous cases, she was typically treated with bouts of rest, nonsteroidal anti-inflammatory medication, modalities (i.e., thermal ultrasound, stretching, electrical stimulation, friction massage), and strengthening exercises. She reported that it generally took several weeks for any noticeable change in her symptoms and that she experienced discomfort throughout her competitive season until she could rest for a few weeks at season completion.

In the current case, the patient did not report any acute trauma and she could not identify the exact onset of her symptoms in regard to a specific swim

activity. She did, however, describe an increase in the intensity and duration of her swim workouts, resistance training, and overhead activities over the past 3 days. The patient rated her current pain as a 6/10 and her worst pain during activity as an 8/10 on the NRS. She reported that her pain decreased during the first half of swim practice, but would then begin to worsen until it reached the point that she could not continue her activity. The patient did not report a recent history of illness, discomfort in her other shoulder, or previous cervical or thoracic spinal pathology.

During initial exam, the patient presented with mild forward shoulder posture bilaterally, but without obvious mal-positioning of the head, cervical spine, or scapulas. Palpation revealed pain and thickening of the long head of the bicep tendon in the bicipital groove. Using TP identification defined by D'Ambrogio and Roth,[11] the patient presented with "jump signs" at the biceps long head (BLH) and supraspinatus (SSL) PRT TPs. The patient also reported significant pain (8/10) at the pectoralis minor (PMI) TP, while palpation of other structures was otherwise unremarkable.[11] Palpation of the TPs indicated specific palpable areas of tenderness, did not produce a referred pain pattern, and were more than four times as sensitive to palpation (as rated by the patient) as surrounding and bilaterally corresponding tissues.[11,12]

Assessment of the patient's active and passive ROM indicated movements that were within normal limits in all directions at the cervical spine and shoulder. The patient reported experiencing pain (6/10) with glenohumeral flexion, extension, and internal rotation on the involved side which she perceived as altering her quality of motion. Manual muscle testing (MMT) was normal bilaterally at the forearm (i.e., supination and pronation), elbow (i.e., flexion, extension), and shoulder (i.e., flexion, extension, internal rotation, external rotation, abduction, adduction, horizontal adduction, and horizontal abduction), but pain (6/10) was reported with elbow flexion and shoulder flexion of the involved side. Speed's Test was positive for pain (6/10), but the remainder of the orthopedic special test (i.e., Hawkins-Kennedy, Neer's, O'Brien's, Apprehension, Empty/Full Can, Joint Play Assessment) and neurological examination were unremarkable. The Disablement in the Physically Active (DPA) Scale was given to the patient who reported an initial score of 42 (out of 64). The working patho-anatomic diagnosis was reactive tendinopathy of the biceps brachii tendon. The proposed underlying physiology of reactive tendinopathy, combined with the patient's presentation and "S.T.A.R." (**S**ensitivity, tissue **T**exture change, **A**symmetry, altered **R**ange of Motion) objective findings during physical exam led to the clinical decision to treat the patient with PRT. The S.T.A.R. mnemonic is used in osteopathic medicine to diagnose somatic dysfunction and guide its treatment.[12]

The patient was treated using only PRT after the conclusion of swim practice on the day of the initial exam. The corresponding PRT technique was applied for 90 s at each of the TPs: BLH (Figure 1), SSL (Figure 2), and PMI (Figure 3). After treatment, the patient was allowed to return to normal activity without participation restriction. The patient was instructed not to perform any additional treatments or take any pain medications. Treatment was applied again the next day and the patient was discharged on the fourth day, following an asymptomatic reexamination that matched the initial exam. Pain scores using the NRS were taken before and following each treatment, while DPA scale scores were recorded at initial exam and discharge.

Outcomes

Initial treatment produced a reduction in pain of 5 points at the BLH TP and 4 points at the SSL and PMI TPs (Figure 4). Pain with ROM and MMT was also reduced by 2 points (Figure 5). In addition, the apparent thickening

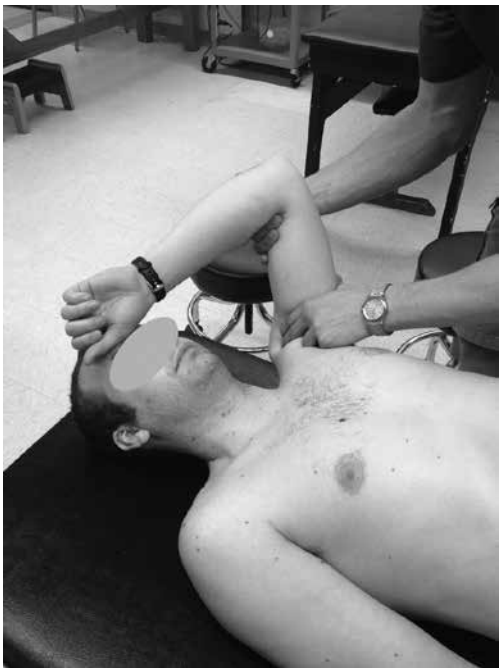


Figure 1 General POC position for the BLH tender point.

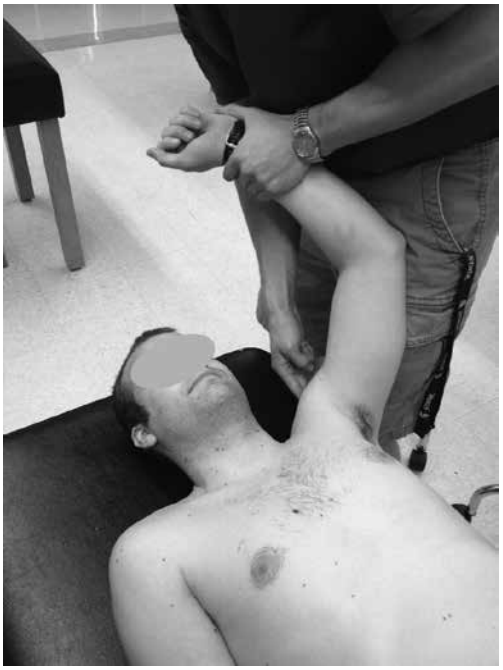


Figure 2 General POC position for the SSL tender point.

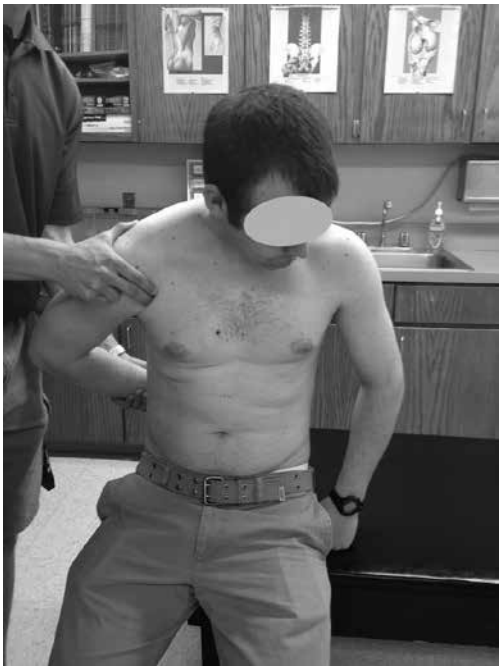
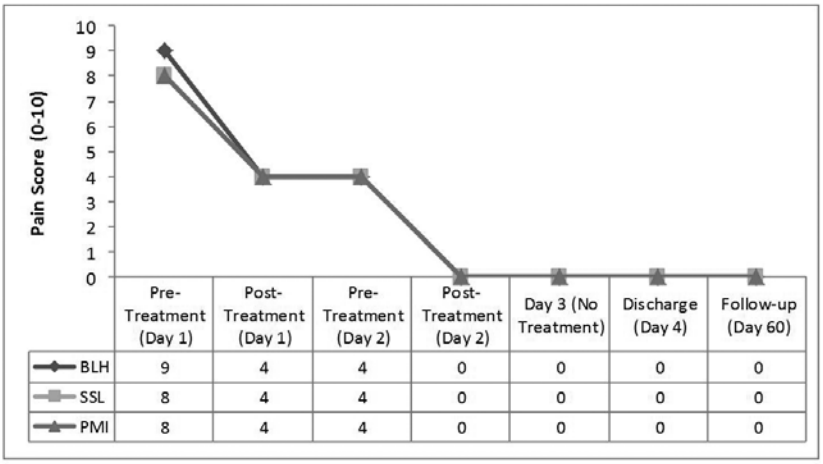


Figure 3 General POC position for the PMI tender point.

of the tendon of the long head of the bicep in the bicipital groove was no longer present after the treatment of the TPs on day 1 and remained normal on all subsequent follow-up examinations. Examination the next day revealed maintenance of these improvements and a current reported pain of 2/10. On day 2, the treatment was repeated and resulted in a complete resolution of the patient's reported pain at rest, during palpation of TPs, ROM assessment, and all MMTs. Examination on day 3 revealed a maintained resolution of signs and symptoms and the patient was instructed to continue normal sport participation without treatment to determine if signs and symptoms would return without treatment. At final examination, on day 4, the patient was asymptomatic during physical exam. At this time, she did not display any tenderness to palpation, was able to complete all ROM and functional tests asymptomatically, displayed a negative special test finding for Speed's Test, and reported a DPA scale score of 6 (Figures 4–6). As a result, the patient was discharged at this time. Follow-up examination one month later, 60 days later, and upon season completion (approximately 5 months later) indicated maintenance of the final measurements and a continued resolution of symptoms.



	Pre-Treatment (Day 1)	Post-Treatment (Day 1)	Pre-Treatment (Day 2)	Post-Treatment (Day 2)	Day 3 (No Treatment)	Discharge (Day 4)	Follow-up (Day 60)
BLH	9	4	4	0	0	0	0
SSL	8	4	4	0	0	0	0
PMI	8	4	4	0	0	0	0

Figure 4 Patient reported pain during tender point palpation.

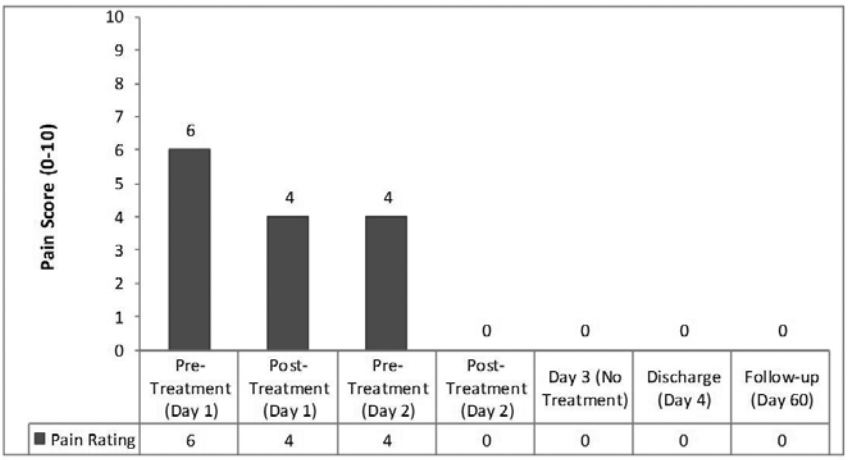


Figure 5 Patient reported pain during ROM testing.

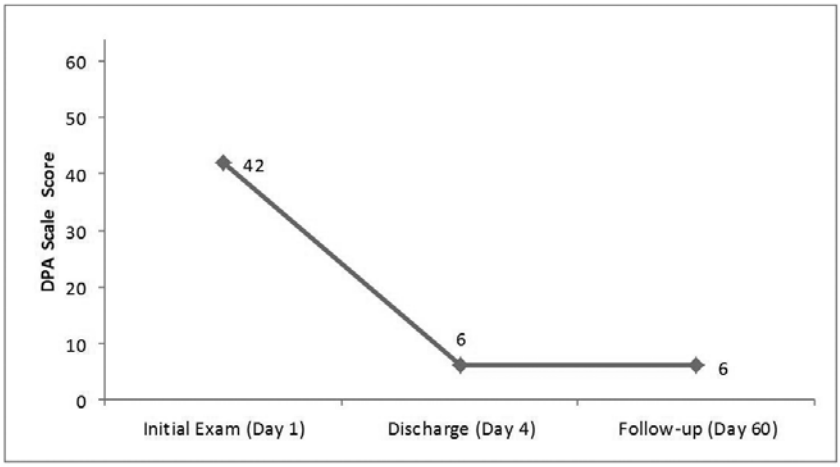


Figure 6 Patient outcome reported on the Disablement in the Physically Active Scale.

Discussion

Currently, a gold standard for treating tendinopathies has not been established, and high-level evidence supporting the use of many traditional interventions used in sports medicine is lacking.[2,3,10,14–16] Recent evidence indicates the application of mechanical therapies (e.g., eccentric exercise, pulsed ultrasound, massage) can be effective treatments when applied over several weeks for reducing pain and dysfunction. Although there is no consensus on the mechanism that is responsible for improvement, it seems likely that treatments that encourage mechanical remodeling and reorganization are likely the source of improvements.[17–20] Eccentric exercises appear to provide the most consistent outcomes,[2–5] but typically requires the patient to experience pain and discomfort for several weeks through the course of treatment. In addition, current application is based on tendon dysfunction necessitating the reorganization and restructuring mechanism to produce its effects.[2–5,8,16,18,21] Recent literature evidence, however, does not correlate structural change with outcomes and certain evidence refutes the structural change model as the explanation of the outcomes experienced with eccentric loading.[10]

Nonsteroidal anti-inflammatories and other modalities (e.g., iontophoresis, phonophoresis, continuous ultrasound, low-level laser) continue to be used with limited success, while corticosteroid use appears to be limited to temporary pain relief without long-term resolution.[2,3,8] Given the complexity of the pathology presentation, it is unlikely that a single intervention used to treat all presentations of tendinopathy will be regularly successful.[10] Additionally, it is difficult to

suggest traditional interventions, regularly employed to treat any tendinopathy presentation, will be successful without a clinician's ability to appropriately classify a patient's exact stage in the continuum of tendon healing when using this model.[2,3,6,8,10,14] Appropriate loading of the tendon, which includes a proper functional loading progression and preparation of both muscle and tendon components for activity, appears to be the most important rehabilitative modality at this time. It is also necessary, however, for clinicians to consider the role of spinal function and central sensitization in the diagnosis and treatment of tendinopathy.[10]

A potential option to improve tendinopathy classification and treatment outcomes is the use of advanced diagnostic imaging; however, this is not current clinical practice in most healthcare settings and is not recommended at this time due to the limitations of imaging methods.[2,10] As such, it is likely that tendinopathy treatments will continue to vary significantly based on clinician factors (e.g., use of movement exam, functional assessments, perceived tendinopathy stage) and patient presentation (e.g., site of pathology, activity level of patient, movement dysfunctions).[10] Generic treatment protocols, even if based on the most recent evidence, are unlikely to provide the optimal treatment of tendinopathy, especially in athletes.[2,3,8,10] It is necessary for sound clinical reasoning, guided by the current clinical presentation, to be applied by the clinician to identify the most effective treatment options.[10]

Thus, it has been recommended for clinicians to classify patients and design a treatment plan along a tendinopathy continuum, focusing on patient presentation as opposed to the generic term *tendinopathy*, which implies the source of the patient's pain is a disordered tendon resulting from a physiological tendon state that must be addressed through reorganization and remodeling.[2,8,10] The use of the continuum provides logical basis for clinicians to acknowledge sources of tendon pain in addition to tendon disruption and allows for the use of targeted treatment paradigms designed to address the current clinical presentation of each individual patient. Other sources for a clinician to consider in tendinopathy cases include referred pain from another source,[22] articular causes or neuroprocessing issues,[23] and other pain philosophies (e.g., pain neuromatrix)[24,25] and regional interdependence issues.[26] The adoption of this model may aid in the selection of an intervention that addresses the associated dysfunction at a given stage for each individual patient case. Clinicians may then assess treatment efficacy with outcome measures with the idea that apparent tendinopathy cases may resolve quickly as opposed to the current patho-anatomical tissue model suggesting weeks to months for recovery.[2,5,8,10,16,27,28]

New literature evidence and changes in the clinical application of PRT advanced the proposed mechanism for the benefits of PRT in treating somatic dysfunction beyond the singular use of the muscle spindle theory. While the literature suggest PRT alters muscle spindle activity,[12,29] others propose the technique may address nociceptive dysfunction, produce anti-inflammatory effects, and positively alter the tissue environment.[12,30,31] It has been suggested that the maintenance of the POC allows for the restoration of the local environment through increased oxygen transport, improved ATP production, decreased inflammatory metabolites, and efficient coupling of actin and myosin.[12,31] Thus, it appears possible for PRT to be an effective treatment during the early stages of the tendinopathy continuum.

Research also indicates PRT is effective at decreasing pain,[30,32–34] while also improving ROM,[32] strength,[33] and function;[12,34] however, the technique may be more effective in acute or subacute cases.[12,34] An important component of this case was the patient was not required to rest or abstain from activity while receiving PRT, which is often recommended for a 24–48hr period due to increase muscular soreness.[11] While further research is needed, a published case series supports the concept that abstaining from activity following PRT treatment may not be necessary in acute/subacute cases.[32] The proposed clinical applications, theorized benefits, and literature evidence support PRT as an intervention to treat the underlying physiological components of a patient presenting with apparent reactive tendinopathy.

Based on the current literature recommendations, clinicians should incorporate the use of outcome measure instruments to assess the effectiveness of chosen interventions when treating tendinopathies. Currently core measures have not been established across all tendinopathies, but it is recommended that measures assess a variety of factors including, but not limited to pain, physical functioning, emotional functioning, and well-being.[10] In this case, the NRS was selected to assess pain improvement across multiple measures, while the DPA scale was used because it was designed to assess the majority of the recommended factors in a physically active population. Minimal clinically

important differences (MCIDs) were then assessed for each instrument to determine whether the patient experienced a clinically significant change after treatment.[35–37] In acute cases, the DPA scale has an MCID value of 9 points,[35] while the MCID value for the NRS is a 30% change or a reduction from baseline of 2 points.[36,37] In this case, the patient experienced a clinically significant change on the NRS when reporting general pain, pain with palpation, pain with motion, and pain during MMT following each treatment session. A clinically significant change was also experienced by the patient from initial exam to discharge as measured by the DPA scale. The use of PRT and outcomes measures guided the clinician in using an intervention that addressed the local pathological problems and resolved patient dysfunction so that normal tendon loading could occur during activity. The initial improvements, combined with the long-lasting benefit at discharge and at follow-up examination post-discharge, suggest PRT was effective a treating this clinical presentation without needing to alter the patient's level of activity.

Clinicians, however, must also carefully consider the clinical signs used to diagnose, treat, and assess treatment effectiveness.[10] In this case, palpation of a "thickened" biceps tendon was noted during the initial exam and was used to help classify the patient with a reactive tendinopathy. The apparent thickening during the initial exam may have been due to mild contraction of the muscle which tensions the tendon giving the appearance of "thickening" or low level contraction of the deltoid. Since the PRT treatment also puts the deltoid in a relaxed position, it is possible the reason the clinician felt an apparent reduction in biceps tendon thickness was due to relaxation of the deltoid muscle following treatment.[11] The resulting rapid change noted during palpation and the potential related causes may indicate palpation was an unreliable diagnostic indicator in this case, which supports previous literature indicating the unreliability of palpation for diagnosis.[38,39]

Conclusion

In the present case, PRT produced an immediate clinically significant improvement and resolved the symptoms in two treatment sessions. The patient was able to complete the entire competitive swim season without a continuation of shoulder pain or dysfunction, which had not occurred in previous seasons. In using the continuum approach to tendinopathy, the patient was treated using an appropriate intervention (i.e., PRT) for her case and was discharged on her third visit compared with the standard 12-week duration of commonly used approaches to treating tendinopathies.[5,16,27] Additionally, her recovery was quicker than the weeks of rest or treatment rat models have suggested necessary for tendon recovery following induced injury.[28,40,41] Further research is needed to determine the effectiveness of PRT in the treatment of reactive tendinopathy in other joints and different stages of the continuum. Additional research is also necessary to further elucidate the mechanism of the effects of PRT and to help create clinical decision rules to guide clinicians in the application of the technique.

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