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