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Scientific/Clinical Article

Rehabilitation of symptomatic atraumatic degenerative rotator cuff tears: A clinical commentary on assessment and management



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ABSTRACT

Study Design: Clinical Commentary.

Introduction: Atraumatic rotator cuff (RC) disease, is one of the most common cause of shoulder pain, which encompasses a continuum from tendinopathy to full thickness cuff tears. Extrinsic, intrinsic and environmental factors have been implicated in the pathophysiology of this disorder, affecting the clinical presentation of symptoms including pain and irritability. Successful rehabilitation of symptomatic atraumatic degenerative rotator cuff (SADRC) tears must address the underlying mechanisms causing dysfunction and correct modifiable factors.

Purpose of the Study: The purpose of this paper is to review the shoulder complex anatomy, introduce atraumatic degenerative RC pathology, differentiate between symptomatic and asymptomatic degenerative RC tears, propose an assessment and introduce the Rotator Cuff Protocol 1 (RCP1) designed by the clinical reasoning of one of the lead authors (LW) as a rehabilitation management approach for those clients who present with SADRC tears.

Methods/Results/Discussion: N/A for clinical commentary.

Conclusions: The ability to identify SADRC tears should consider shoulder anatomy, extrinsic, intrinsic and environmental factors, and the consideration for the natural history of atraumatic partial and full thickness tears in the general population. A thorough clinical history and examination, which includes shoulder symptom modification tests, allows the examiner to determine at what phase the patient may start their exercise program. The RCP1 is a program that has been used clinically by many therapists and clients over the years and research is underway to test this protocol in atraumatic rotator cuff disease including SADRC tears.

Level of Evidence: 5.

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Introduction

Shoulder pain can be attributed to rotator cuff (RC) disease, being either traumatic or atraumatic in nature.^{1–3} Its continuum encompasses tendinopathy without gross changes in tendon morphology or structure, through to partial thickness tear (PTT), and ending with full thickness tear (FTT) and arthropathy.^{1,4,5} The incidence of degenerative tendon changes increasing with age^{6,7} and symptomatic atraumatic degenerative rotator cuff (SADRC)

tears is one of the many causes of shoulder pain in general practice.^{3,8,9} Reported prevalence of asymptomatic and symptomatic atraumatic PTT and FTT RC tears ranges from 6% to 34% and increase with age in the general population.^{9,10}

Nonsurgical management of atraumatic PTT and FTT is well supported in the literature, and although exercise is the most consistently supported intervention,^{3,6,11} there remains a lack of evidence to assist therapists with the customization of assessment and treatment options for specific patients. The purpose of this article was to review the shoulder complex anatomy, introduce atraumatic degenerative RC pathology, differentiate between symptomatic and asymptomatic degenerative RC tears, propose an assessment, and introduce the Rotator Cuff Protocol 1 (RCP1) designed by the clinical reasoning of

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one of the lead authors (L.W.) as a rehabilitation management approach for those clients who present with SADRC tears.

Anatomy and kinetics

There are 4 joints that make up the shoulder complex: the glenohumeral (GH) joint, acromioclavicular joint, sternoclavicular joint, and the functional scapulothoracic articulation. Effective movement and function of the shoulder is achieved through combined effort from scapula stabilizers, deltoid, the RC (supraspinatus, infraspinatus, teres minor, and subscapularis) as well as the axiohumeral muscles (latissimus dorsi and pectoralis major).¹²⁻¹⁶

Depending on the movement direction and joint position of the shoulder, the specific contribution of the RC muscles will vary. We know that translation of the humeral head (HH) is constrained within normal limits by the coordinated activity of the RC.^{7,12} It is unclear whether the RC produces specific translational movement of the HH or stabilizes and/or compresses the HH in the glenoid fossa. Wickham et al¹⁵ demonstrates that supraspinatus, middle deltoid, and middle trapezius all consistently show anticipatory contraction before abduction of the shoulder, presumably to stabilize the scapula and HH. The RC muscles are also prime movers, with the infraspinatus, teres minor and supraspinatus contributing to external rotation (ER),^{17,18} and the subscapularis contributing to internal rotation (IR) and horizontal flexion.^{16,19} The RC does not function in isolation, with the scapula stabilizers (all components of trapezius and serratus anterior [SA]) and deltoid contributing to the actions of the shoulder^{12,13,20} through varying recruitment patterns, depending on the position of the shoulder and motion being performed.^{15,16,21}

Atraumatic degenerative RC pathology

The anatomic and pathogenesis of atraumatic RC degeneration is thought to occur naturally with aging.²² Extrinsic, intrinsic, and environmental factors (ie, repeated tensile loading) can contribute to the multifactorial etiology of atraumatic degeneration of the RC.²³⁻²⁷ Cook et al⁴ defines 3 stages of tendinopathy that can be applied to the continuum of degeneration of the RC which continues to be supported by the literature²⁸: (1) reactive tendinopathy, (2) tendon disrepair, and (3) degenerative tendinopathy. Once a tendon is in the degenerative stage, it is unable to return to its normal morphology; however, there is capacity for the tendon to provide functional motion for the shoulder.²⁹ The natural history of degenerative RC tears remains unclear; however, both PTT and FTT RC tears can present in asymptomatic and symptomatic shoulders.^{9,10} It is hypothesized that when excessive load and strain are repeatedly applied to a degenerative tendon through contractile overload or mechanical compression may convert an asymptomatic RC to a symptomatic RC^{30,31} (Fig. 1).

Extrinsic factors

External impingement is the term used to describe compression of structures within the subacromial and subcoracoid spaces, whereas internal impingement is understood to be compression of any structure within the GH joint.³²⁻³⁶ In a population with atraumatic degenerative RC tears, both the posterosuperior and anterosuperior structures can be compressed and develop pathology.^{33,37} Altered scapular and GH kinetics affect the congruency of joint surfaces, the GH fulcrum, and the ability for the RC to provide adequate stability and motor control, which predisposes the shoulder complex to mechanical compression.^{17,38} Numerous

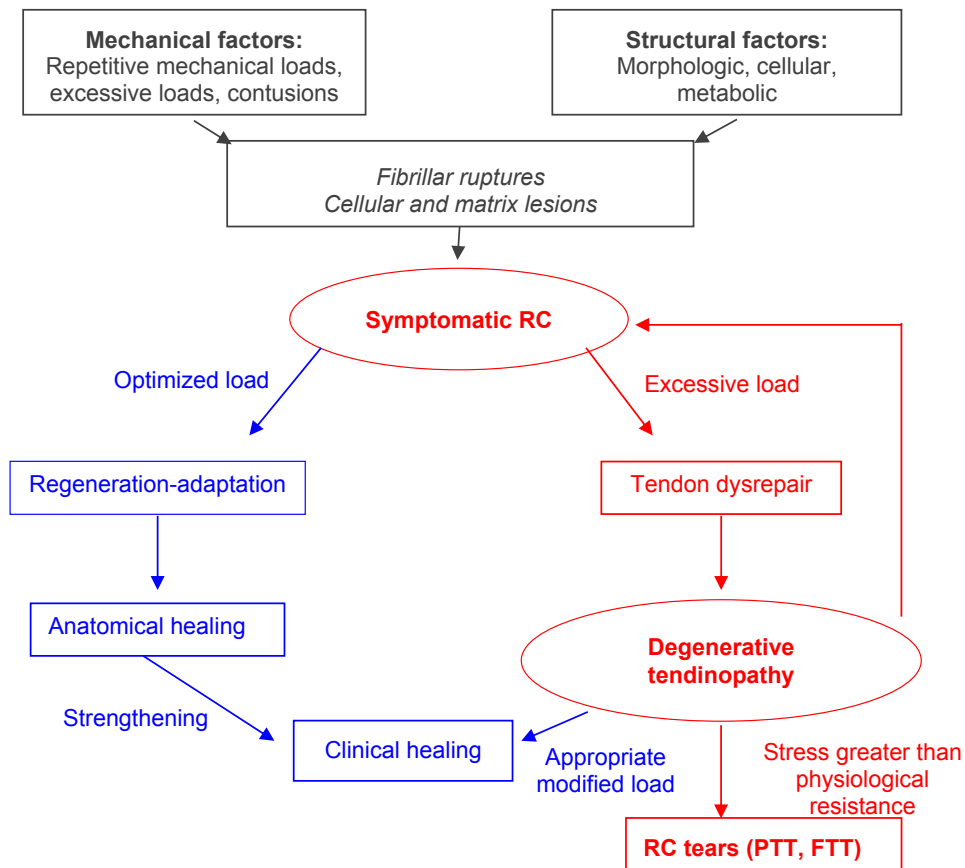


Fig. 1. Tendon loading and tendinopathy continuum. FTT = full thickness tear; PTT = partial thickness tear; RC = rotator cuff. Adapted with permission from Kaux et al.³⁰

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