Partial Thickness Rotator Cuff Tears: What Do We Know?

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Shoulder & Elbow 2010 2: 77
DOI: 10.1111/j.1758-5740.2010.00054.x

Shoulder & Elbow. ISSN 1758-5732

REVIEW ARTICLE

Partial thickness rotator cuff tears: what do we know?

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Received
Received 9 November 2009;
accepted 5 February 2010
Keywords
Partial thickness, rotator cuff tears, articular-sided, bursal-sided
Conflicts of Interest
None declared
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DOI:10.1111/j.1758-5740.2010.00054.x

ABSTRACT

Partial thickness rotator cuff tears are not an uncommon pathology encountered within the sporting community. However, the pathology associated with them has not been well documented. This has lead to difficulty in the diagnosis and classification of these injuries and their optimal treatment is yet to be decided. This review presents the current literature on partial thickness rotator cuff tears, including their incidence, aetiology and pathology. The available literature on diagnosis and treatment is also presented. It is hoped that this review will provide the clinician with a concise summary of the current knowledge of partial tears and help to direct the clinician in the management of these injuries. The review also highlights those areas where further evidence is required.

INTRODUCTION

There exists an abundance of publications on partial thickness rotator cuff tears (PTRCTs); however, the optimal treatment of these lesions is still not known. The aim of this review is to critically assess the current literature and present the best evidence-based knowledge in this area.

TERMINOLOGY AND CLASSIFICATION

PTRCTs can be described by their position as either articular, bursal or intratendinous. Multiple terms have also been used for articular-sided tears at the point of insertion of the supraspinatus into the bone. These include 'Rim rents' [1], the 'PASTA' lesion (partial articular supraspinatus tendon avulsion) [2] and 'articular-sided footprint' lesions [3]. The 'Rim rent' is described as a partial tear of the articular-sided supraspinatus tendon at its insertion on to the humerus and is likely to be identical to a PASTA lesion. A 'PAINT' (partial articular tear with intratendinous extension) appears to be a PASTA lesion with an associated proximal delaminated flap [4].

Multiple classifications exist for full thickness rotator cuff tears (FTRCTs), some of which include partial thickness tears within them. The Snyder classification [5] (Table 1) identifies the location and grades the cuff damage.

Ellman initially classified tears based on their position into articular, bursal and intratendinous tears [6] and then further graded PTRCTs into grade I (<3 mm), grade II (3 mm to 6 mm), or grade (>6 mm) [7]. This is the most commonly used classification within the literature, probably as a result of its simplicity. However, this classification is based on an assumption that the average thickness of the rotator cuff is 12 mm. This was suggested by Ellman [7] to be the normal thickness of the rotator cuff, although no study has documented the normal superior to inferior thickness of the supraspinatus tendon. Anatomical studies of the supraspinatus tendon at its insertion on to the humerus have reported mean medial to lateral widths ranging from 9.6 mm to 16 mm [8–12]. This would suggest that a 6-mm defect or avulsion from the insertion point represented between 37.5% and 62.5% of the tendon substance. Scanning electron microscopy studies have shown that the medial edge of the supraspinatus tendon insertion is adherent to the articular cartilage [8]. Thus, although the footprint of the supraspinatus has been well mapped, there may be some normal variation in thickness of the tendon. Moreover, the inter-observer agreement of tear depth using an arthroscopic probe is poor (observed agreement = 0.49, kappa value = 0.19) [13].

A further classification has been described for articular-sided footprint lesions [3], which describes the amount of peel-off of the footprint from the undersurface of the supraspinatus footprint and the extent of the tear in the sagittal plane. This classification is limited to footprint avulsion injuries and has not been widely used within the literature. It is apparent that the ideal classification has yet to be established.

INCIDENCE

A systematic review [14] selected 55 publications relating to the incidence of PTRCTs and found their accumulated incidence in the cadaveric population to be 18.5% (mean age 70 years). This compared to an incidence of 11.8% for FTRCTs. The incidence in asymptomatic patients detected by ultrasound was 17.2%, whereas the incidence in the symptomatic shoulder was 6.7%

Table 1 The Snyder classification [5]

		Location of tear		
		A — articular	B — bursal	C — full thickness
Grade	0	Normal cuff		
	1	Inflamed synovium and superficial fraying <1 cm		
	2	Moderate tear with actual fibre disruption 1 cm to 2 cm		
	3	Disruption and fragmentation of tissues 2 cm to 3 cm		
	4	Complex PTRCT (flap formation and retraction) >3 cm		

PTRCT, partial thickness rotator cuff tear.

(mean age 54 years). From magnetic resonance imaging (MRI) assessment in the asymptomatic patient, the incidence was 15.9% (mean age 44.3 years), whereas the value for the symptomatic patient was 8.57% (mean age 43.6 years). This would suggest that approximately 5% to 10% of patients presenting with shoulder pain have a symptomatic PTRCTs. The incidence of FTRCTs was higher in the symptomatic group compared to the asymptomatic group, which may account for the smaller numbers of PTRCTs in the symptomatic group compared to the asymptomatic group. This would also suggest that more FTRCTs are symptomatic than is the case with PTRCTs.

The literature demonstrates that articular-sided tears are at least twice as common as bursal-sided tears [7,15–22] and that most partial thickness tears involve the supraspinatus tendon [22–24].

Progression of symptomatic PTRCTs to FTRCTs with non-operative treatment has been seen in 18% of patients followed up for over 1 year, with a further 34% exhibiting increase in partial tear size [25].

There is no evidence to suggest why some partial thickness tears are asymptomatic and no documentation of the progression from asymptomatic PTRCTs to symptomatic ones.

AETIOLOGY

The extrinsic theory of rotator cuff pathology [26,27] describes the abrasion of the cuff by an abnormal acromion, which could result in a bursal-sided partial cuff tear. Neer originally described three stages of rotator cuff pathology caused by external abrasion [27] (Stage I — haemorrhage and oedema, Stage II — fibrosis and tendonitis and Stage III — tearing). Bursal-sided tears have demonstrated histological changes on the undersurface of the acromion, whereas articular-sided tears did not [20], suggesting that the pathologies of the two types of tears are different. Internal impingement has been described whereby the undersurface of the supraspinatus impinges on the glenoid leading to articular-sided PTRCTs, particularly in overhead throwing athletes [28,29].

The intrinsic theory of cuff pathology is based on an internal degeneration of the tendon. Degeneration from histological examination was noted to be more prominent on the articular side in the intact supraspinatus [30]. The articular side of the supraspinatus is also known to have a reduced tensile strength compared to the bursal side [31]. These factors may contribute to the fact that articular-sided tears are more common.

Other described etiological factors for PTRCTs include trauma, repetitive microtrauma, instability and insidious onset associated with age-related degenerative change [16,23,32–35]. A number of studies of PTRCTs treated with decompression have demonstrated progression to FTRCTs [22,36,37].

PATHOLOGY

The inability of PTRCTs to heal has been attributed partly to the poor microvascular supply within the supraspinatus tendon [38–40]. The vascular supply has also been noted to be predominantly on the bursal side, with virtually no vessels on the articular side [1,38]. However, some caution most be taken when interpreting results from histological studies using injected suspensions because this has the drawback of creating micro-emboli [39], which may reduce the recorded vascularity.

In vivo measurements of the microvasculature have shown an increase in blood flow on the edge of a full thickness tear [41] and hypervascularity in a small sample of partial thickness tears [42], thus contradicting the histological findings. *In vivo* histological samples have also suggested that the increase in vascularity decreases as the size of the tear increases [43].

DIAGNOSIS AND IMAGING

Both the clinical and radiological diagnosis of PTRCTs is difficult. There is no consensus in the literature regarding any particular clinical test that can reveal a partial tear. Some studies have suggested that PTRCTs are more painful than FTRCTs [44,45], although both clinical examination and pain have been found to be a poor indicator of the size of full thickness tears [46] and not useful for differentiating between partial and full thickness tears [47].

MRI, magnetic resonance angiography (MRA) and ultrasound remain the predominant modes of imaging, although some plain film radiographical signs have been reported to be associated with articular-sided PTRCTs [48].

Two meta-analyses have been performed on the imaging of PTRCTs. The first analysis, published in 2003 [49], included studies with nonsurgical findings (e.g. arthrography) as the gold standard. The second study in 2009 [50] only included studies with operative findings as the gold standard. For ultrasound, the sensitivity was 67% in both studies, with a specificity of 94% in both studies. MRI originally had a sensitivity of 44%, rising to 64% in the most recent study, and a specificity of 90% and 82%, respectively. For MRA, the sensitivity was originally 62%, increasing to 86% in the most recent study, with a specificity of 92% and 96%, respectively. The findings of the latest study [50] concluded that MRA was statistically more sensitive and specific than either ultrasound or MRI ($p < 0.001$), with no statistical difference between ultrasound and MRI.

TREATMENT

A systematic review of the use of exercise therapy in FTRCTs concluded that all studies showed an improvement in outcome scores, although the evidence was limited [51]. For PTRCTs, most studies advocate a period of non-operative management,

Table 2 Summary of surgical outcomes for partial thickness rotator cuff tears within the literature

Study	Number of patients	Age, years (range)	Surgical intervention	Tear size treated (% articular)	Mean follow up (months)	Conclusion
Andrews et al. [34]	36	22 (16–29)	Debridement only	All (N/R)	13	Good or excellent result in 85%.
Fukuda et al. [26]	66	54 (20–75)	Debridement, decompression and repair	All (33% articular)	32	Satisfactory in 94%
Wright et al. [2]	39	52 (21–73)	Debridement, bursectomy, decompression and repair	N/R (54% articular)	55	Excellent result in 59%
Weber et al. [57]	32	49 (N/R)	Debridement and decompression	>6 mm (89% Articular)	47	Good outcome in 44%
	33	46 (N/R)	Debridement, decompression and open repair		38	Good or excellent outcome in 94%.
Cordasco et al. [52]	25	57 (36–90)	Debridement and decompression	>3 mm (Grade I)	53	Mean L'Insalata score of 89 postoperatively
	52	53 (33–72)		3–6 mm (Grade II) (83% articular)		
Park et al. [21]	37	52 (34–70)	Debridement and decompression	<50% (<6 mm) (65% Articular)	42	83% Satisfactory results for articular-sided 92% Satisfactory results for bursal-sided
Park et al. [39]	22	55 (N/R)	Debridement, decompression and repair	>50% (>6 mm) (27% articular)	34	Good or excellent result in 93%
Ide et al. [53]	17	42 (17–51)	Debridement, bursectomy and repair	>6 mm (100% Articular)	39	Good or excellent outcome in 94%.
Kartus et al. [36]	26	51 (26–66)	Debridement and decompression	3–6 mm (50% articular)	101	35% Progress to FTRCT 38% remain PTRCT
Deutsch et al. [1]	41	49 (23–70)	Debridement, completion and repair	>50% (80% Articular)	38	Good or excellent outcome in 90%.
Liem et al. [13]	46	59 (33–76)	Debridement, bursectomy and decompression	<6 mm (100% articular)	50	Patient satisfaction good or excellent in 87%
Kamath et al. [40]	42	53 (34–72)	Debridement, bursectomy, decompression	>50% (79% articular)	39	Good or excellent result in 68%
			Completion and repair			88% healing

N/R, not reported; FTRCT, full thickness rotator cuff tear; PTRCT, partial thickness rotator cuff tear.

including physiotherapy, modification of activities, nonsteroidal anti-inflammatory drugs and steroid injections prior to the consideration of surgery [7,52–56]. However, there is no evidence to support or refute these treatments. There is also no guidance for the time that should be allowed for non-operative treatment to work before considering surgical intervention.

Many surgical treatments have been described, with different permutations of four procedures: debridement, sub-acromial decompression, repair *in situ* and completion and repair. Good outcomes have been described with debridement only [5,32,37,57,58], debridement and decompression [15,37,59], repair *in situ* [52,60–64], and conversion to full thickness tears with repair [16,65,66]. However, the definition of a 'good' or 'excellent' outcome depends on the scoring system used and has not always been defined. This makes direct comparison between outcomes difficult. A summary of the key studies is presented in Table 2.

Although there appears to be a consensus that PTRCTs greater than 50% should be repaired [7,10,16,24,53,61,65,67,68], there is no strong evidence to support this hypothesis. Other studies recommend that surgery is targeted at the underlying pathology associated with aetiology [23,24]. However, no strong comparative studies have been published to direct the clinician as to which patients would perform best with a particular procedure. One study comparing mini-open repair against arthroscopic debridement with acromioplasty for PTRCTs greater than 50% favoured open repair [22]. However, this was a retrospective study and compared an open procedure with an arthroscopic procedure. Also, one biomechanical study has shown an advantage to *in situ* repair compared to completion and repair [69], although this has yet to be replicated in a clinical scenario.

OUTCOME MEASUREMENT

The outcome measures used in studies relating to PTRCTs include Neers criteria [27], the UCLA score (University of California at Los Angeles) [70], the simple shoulder test [71], the American Shoulder and elbow society score [72], the L'Insalata score [73], the shoulder rating score [57], the Constant and Murley score [57,74], the JOA score (Japanese Orthopaedic Association) [10,75], and the self assessment activity score [36,76]. The diversity of scoring systems used demonstrates that no scoring system is ideal and makes direct comparisons between publications difficult.

CONCLUSIONS

This review has presented the current knowledge of PTRCTs. It has shown that many questions relating to this pathology still remain unanswered. What can be extrapolated from the literature is that it is not an uncommon condition to encounter within a shoulder or sports clinic. It is not easy to diagnose a partial cuff tear clinically and an MRA should be the investigation of choice if there is a suspicion of a PTRCT. However, the diagnosis may only be encountered at arthroscopy and should be considered in any patient who has been identified with rotator cuff tendinopathy or a full thickness tear on pre-operative imaging. There is evidence to suggest that, if left untreated, a significant number of PTRCTs will progress in size and may become FTRCTs. An ideal surgical intervention at this stage cannot be proposed based on the currently available evidence.

Articular, bursal and intratendinous tears are likely to differ in aetiology or combinations of aetiological factors. Consequently, they may respond differently to different treatments.

Many questions remain unanswered, including the need to determine the true microvascularity of the rotator cuff in normal and partially torn tendons; to be able to classify these lesions in order to aid in prognosis and compare outcomes; to determine the best nonsurgical and surgical treatments; to identify any factors that may predict outcome; and to identify at what stage surgical intervention should be considered.

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