Review Article

Diagnosis and Management of Partial Thickness Rotator Cuff Tears: A Comprehensive Review

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ABSTRACT

Partial thickness rotator cuff tears (PRCTs) are a challenging disease entity. Optimal management of PRCTs continues to be controversial. Although advances in magnetic resonance imaging and ultrasonography have aided in early diagnosis, arthroscopic evaluation remains the benchmark for diagnosis. Conservative treatment is often the first line of management for most patients; however, evidence suggests that surgical intervention may limit tear progression and the long-term sequelae. Surgical decision making is driven by factors such as age, arm dominance, etiology, activity level, tear thickness, and tear location. Many surgical options have been described in the literature to treat PRCTs including arthroscopic débridement, transosseous, in situ repair techniques, and tear completion and repair. Biologic supplements have also become an attractive alternative to aid in healing; however, the long-term efficacy of these modalities is largely unknown. This article will provide a detailed review of the etiology and natural history of PRCTs, as well as diagnosis, and current management to guide clinical decision-making and formulate an algorithm for management of PRCTs for the orthopaedic surgeon.

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Rotator cuff (RC) pathology is a leading cause of shoulder-related disability with a continuum of disease from tendinosis to partial-thickness (PRCT), full-thickness (FTT), and massive tears. Although strong evidence supports treatment of small to medium FTT, the literature is limited for PRCTs. Conservative treatment and arthroscopic repair of high-grade PRCTs[1] yield good functional results in carefully selected patients. However, tear progression and long-term consequences are still a concern.[2,3] This article will provide a comprehensive review of the etiology, natural history, diagnostic methods, and current trends in nonsurgical and surgical treatment of PRCTs.

Etiology, Natural History, and Tear Progression

The reported prevalence of PRCTs varies by imaging modality: 15.87% in magnetic resonance imaging (MRI), 17.2% in ultrasonography, and 18.49%

in cadaveric studies.[4] Intratendinous tears are the most common tear type, and articular-sided tears are 2 to 3-times more common than bursal-sided tears.[5] A large insurance database study suggests that PRCTs occur most frequently in women and patients aged 65 to 69 years; however, others suggest that the incidence is higher in men.[6] Prevalence estimates may be underrepresented in younger and overhead athletes because not all PRCTs are symptomatic. Del Grande et al[7] reported the MRI prevalence of PRCTs to be 32% among asymptomatic baseball pitcher draft picks. Similar findings have also been reported in collegiate baseball pitchers and professional tennis players.[8]

The etiology of PRCTs is multifactorial and varies by tear location (eg, articular-sided, bursal-sided, and intratendinous). Extrinsic causes are the result of direct compression of the RC tendon against the undersurface of the acromion and coracoacromial ligament as well as mechanical overuse, greater tuberosity fractures, and glenohumeral dislocations. Bursal-sided tears are more commonly associated with subacromial impingement and occur at the tendon–bone interface.[9] Acute trauma, chronic microtrauma from overuse, instability, internal impingement, and intrinsic factors, including age-related histological changes and decreased tendon vascularity, contribute to increased tendon strain at its insertion and resultant degeneration. Articular-sided tears are more common in young athletes and the older patient population and occur more posteriorly at the supraspinatus–infraspinatus interval.[10,11] Intrasubstance tears occur from shear forces on a degenerated tendon and can occur in isolation with an outer bursal and inner articular surface that is intact or in conjunction with articular-sided or bursal-sided tears.

Animal models of partial-thickness supraspinatus and infraspinatus tears suggest that spontaneous healing results in fibrocartilage formation and weaker tendon-to-bone attachment.[12] This poor healing potential may contribute to tear progression, a primary concern when determining optimal treatment.

Keener et al[2] evaluated tear enlargement and progression in 224 subjects with asymptomatic RC tears. Forty-four percent of PRCTs progressed to FTTs at a median of 5.1 years. Forty-six percent developed new pain at a median of 2.6 years. Tear progression was a risk factor for new onset of symptoms with a 1.69-times higher prevalence of pain compared with stable PRCTs. Mall et al[13] reported that 33% of patients with asymptomatic PRCTs became symptomatic at 1.92-year follow-up; 40% of symptomatic PRCTs progressed to FTTs evidenced on ultrasonography.

Maman et al[14] followed up 26 patients with PRCTs treated nonsurgically. At 20-month follow-up, two patients (8%) had tear progression on MRI, 23 patients (89%) exhibited no change, and one patient had a >5 mm decrease in tear size. Kong et al[3] assessed the role of tear location and progression. Eighty-one patients (23 articular-sided and 58 bursal-sided) with high-grade PRCTs (eg, tear involvement >50% mediolateral footprint) underwent MRI evaluation at 19.9 ± 10.9-month follow-up. Thirteen patients (16%) exhibited tear progression (ie, >20% increase in size; two articular-sided, 8.7%; 11 bursal-sided, 19.0%), 48 patients (59%) experienced no change, and improvement (ie, >20% decrease in size) was noted in 20 patients (25%, 9 articular-sided and 11 bursal-sided). Matthewson et al[15] investigated the relationship between tear size and progression. Fifty-five percent of high-grade PRCTs exhibited progression compared with 14% of low-grade PRCTs (ie, <50% tendon thickness). In summary, tear progression can lead to new onset of symptoms and is more likely in tears >50% of tendon thickness.

Clinical Presentation

Clinical diagnosis of PRCTs is based on a thorough history and physical and radiologic examination. Although many PRCTs are asymptomatic, pain and limited shoulder motion lead patients to seek medical advice. Symptoms can vary from complaints of chronic night pain, pain exacerbated with overhead activities, decreased throwing velocity, and deep posterior shoulder pain with fatigue. Bursal-sided tears are often more painful than articular-sided tears.[16] The clinical symptoms of intratendinous tears mimic a FTT or painful bursal-sided tear. Onset of pain is usually insidious; however, some patients may experience a pop in the shoulder, which could be indicative of an acute tear. New onset shoulder pain in an incidentally diagnosed asymptomatic PRCT or an increase in pain in an already symptomatic patient may indicate tear enlargement.[13]

Physical Examination

A comprehensive clinical examination must be conducted in a shoulder gown that allows for visual inspection of the shoulder girdle. The examiner should first rule out any radicular pain originating from the cervical spine. Full active and passive shoulder range of motion (ROM) is often exhibited although should be compared with the contralateral side. A painful arc of motion from approximately 90° to 120° in flexion and/or abduction may be present.[17] Adaptations of increased external rotation at 90° abduction and resultant decreased internal rotation with a normal arc of motion are frequently observed in throwing athletes; however, loss of ROM >20° compared with the contralateral shoulder, known as glenohumeral internal rotation deficit, may be present. Glenohumeral internal rotation deficit is an adaptive process in throwers with posterior capsule tightness and anterior capsule stretching, leading to microinstability and risk of PRCTs when accompanied with internal impingement. RC weakness is rare; therefore, strength testing of the RC and scapular muscles may prove to be normal. Pain with resisted external rotation with the arm at the side is more suggestive of infraspinatus pathology.

Provocative testing is often nonspecific. Positive Neer and Hawkins subacromial impingement signs are associated with PRCTs, although not diagnostic.[5] The internal rotation resistance strength test is done with the patient's shoulder in 90° abduction and 80° to 85° of external rotation. Apparent weakness with resisted internal rotation may suggest internal impingement.[13] Biceps or labral pathology may accompany RC changes and would be evidenced by a positive Speed, O'Brien, or Yergason test. Instability should be evaluated using anterior drawer, sulcus sign, and apprehension/relocation tests, especially in the young, throwing athlete.

Diagnostic Injections

Selective, diagnostic injections of an anesthetic (eg, lidocaine) into either the subacromial space or glenohumeral joint may help confirm suspicion of pathology if the patient experiences persistent or recurrent symptoms. Provocative examination maneuvers are repeated after the injection to assess for alleviation of symptoms. Pain relief with a subacromial injection is suggestive of subacromial impingement with or without a RC tear. If symptom relief is achieved with a subacromial injection, we caution against proceeding to the operating room. Symptom relief after a glenohumeral injection increases suspicion of intraarticular pathologies (eg, labral tears, internal impingement, osteoarthritis, and adhesive capsulitis).

Imaging

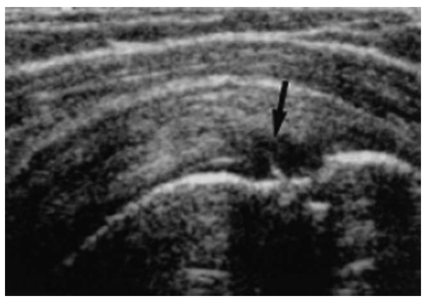
Initial evaluation should include plain radiographs, including true AP, axillary, and scapular-Y views. Although plain imaging is often nonspecific, it can assess for fracture, glenohumeral osteoarthritis, acromial spurs, or other sources of pain. A reduction in the acromiohumeral distance is usually indicative of a FTT (normal 9 to 14 mm).

Ultrasonography (US) has been increasingly used as an imaging modality due to its availability, portability, cost-effectiveness, and dynamic visualization. Confirmatory ultrasonography findings of PRCTs include a focal, contour tendon defect, a mixed hypohyperechoic linear band, or a linear band of anechoic appearance (Figure 1). Although ultrasonography is reliable in diagnosing FTTs, distinguishing PRCTs from tendon scarring and small FTTs can be difficult. The reported accuracy, sensitivity, and specificity of US in detecting PRCTs are 87%, 66.7%, and 93.5%, respectively.[18]

MRI provides valuable information on the anatomy and structural integrity of the RC, including tear location, size, and muscle atrophy, as well as other soft-tissue injuries (eg, labral tears and biceps tendon pathology). MRI findings indicative of a PRCT include increased signal intensity at the RC insertion onto the greater tuberosity on either the bursal or articular surfaces or within the tendon substance on T2-weighted, fat-suppressed images matching fluid signal (Figures 2–4). MRI is invaluable in diagnosing intratendinous tears that are not evident on arthroscopic evaluation because only the outside surface of the tendon can be visualized; however, they are often missed because of patient positioning. When placed in the MRI scanner with the arm positioned at the side, the layers of the RC are compressed and do not allow for visualization of interstitial tears.

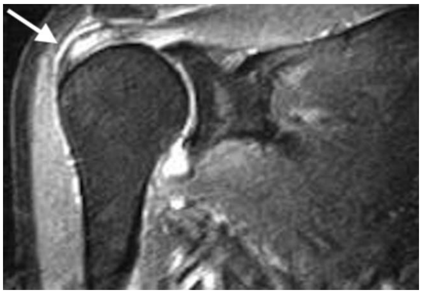
A systematic review of 44 studies reported a sensitivity and specificity of MRI in detecting PRCTs of 80% and 95%, respectively.[19] However, Brockmeyer et al[20] did not demonstrate the same degree of accuracy with MRI (sensitivity 51.6, specificity 77.2, positive predictive value 41.3%, and negative predictive value 83.7%). Although some suggest similar sensitivity and specificity

Figure 1



Ultrasound image showing a small articular-sided partial thickness cuff tear.

Figure 2



T2-weighted fat suppressed image showing a >50% thickness articular-sided partial thickness rotator cuff tear in a right shoulder of a 75-year-old woman. Copyright K. Plancher, MD, MPH

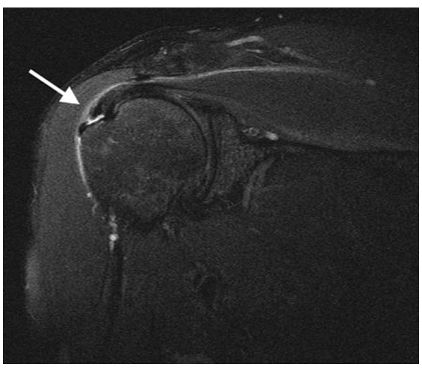
between MRI and ultrasonography, a meta-analysis of 144 studies showed MRI to have a higher sensitivity and superiority index compared with ultrasonography.[21]

Magnetic resonance arthrography uses the injection of gadolinium contrast into the joint for evaluation. Articular-sided tears are visualized as tendon discontinuity with contrast extruding from the articular side on T2-weighted images (Figure 5). Bursal tears are seen as focal hyperintense lesions on fat-saturated intermediate-weighted images, extending to the subacromial bursa.[22] Magnetic resonance arthrography has superior diagnostic value in terms of sensitivity and specificity for articular-sided PRCTs compared with ultrasonography and MRI, although similar diagnostic accuracy has not been found for bursal-sided tears.[23]

Classification

Several classification systems exist for categorizing PRCTs by tear location, tear size, and tendons involved.

Figure 3



T2-weighted fat-suppressed image showing a bursal-sided partial thickness rotator cuff tear involving >50% thickness in a right shoulder of a 56-year-old man. Copyright K. Plancher, MD, MPH

Figure 4

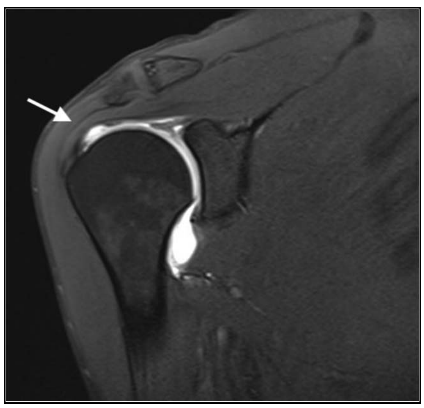
T-2 weighted image showing an intrasubstance tear of the supraspinatus tendon in a right shoulder. Copyright K. Plancher, MD, MPH

Harvard Ellman classified PRCTs according to tear location (eg, bursal surface, articular surface, and interstitial/intrasubstance) and tear depth (Table 1). Snyder et al proposed an alternate classification system defining PRCTs as bursal-sided or articular-sided further grading them from zero to 4 depending on tear size and severity. Although there is agreement between the Ellman and Snyder classification systems, the interobserver reliability of these systems is fair.[24]

Nonsurgical Treatment

Given a low risk of tear progression, fatty infiltration, and muscle atrophy with low-grade PRCTs, nonsurgical treatment is typically the first line management, although evidence to support its use is limited.[13,14] A formal physical therapy program and the use of NSAIDs are

Figure 5



MRA of the right shoulder showing an articular-sided partial thickness rotator cuff tear in a 47-year-old woman. Copyright K. Plancher, MD, MPH

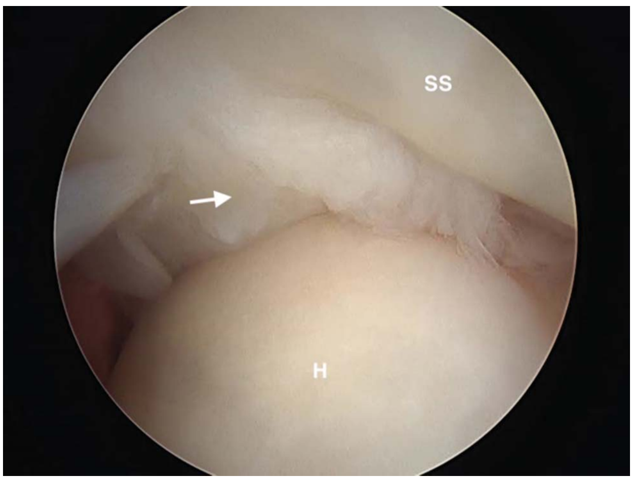
Table 1. **Ellman and Snyder Classification System for Partial Rotator Cuff Tears**

Location	Ellman Classification		Snyder Classification	
A Articular	1	<3 mm (<25%)	0	Normal cuff with smooth coverings of synovium and bursa
B Bursal	2	3-6 mm (25-50%)	I	Minimal superficial bursal or synovial irritation or slight capsular fraying in a small, localized area; usually <1 cm
C Intratendinous	3	>6 mm (>50%)	II	Actual fraying and failure of some RC fibers in addition to synovial, bursal, or capsular injury; usually 1-2 cm
			III	More severe RC injury, including fraying and fragmentation of tendon fibers, often involving the entire surface of a cuff tendon (most often the supraspinatus); usually 2-3 cm
			IV	Very severe partial rotator tear that usually contains a sizable flap tear in addition to fraying and fragmentation of tendon tissue and often encompasses more than a single tendon; usually >4 cm

RC = rotator cuff
Based on a normal supraspinatus tendon thickness of 12 mm.

advisable with an initial emphasis on pain control and activity modification. Internal rotation deficits and posterior capsule tightness should be addressed to facilitate restoration of shoulder active and passive ROM. Postural and periscapular strengthening exercises can improve scapulohumeral rhythm and muscle activation patterns to avoid a posture of protraction. Targeted RC strengthening should occur in a pain-free ROM and should be progressed to incorporate plyometric and functional, sport-specific or job-specific exercises to promote safe return to full activities. Subacromial corticosteroid injections can also be considered when other conservative options have failed. The 2019 American Academy of Orthopaedic Surgeons Clinical Practice Guidelines assert that there is moderate evidence (eg, low-quality evidence and benefits exceed potential harm) for improvement in pain and function in patients with shoulder pain.[25] Although short-term relief may be provided, multiple, repeated corticosteroid injections and injections administered within a year before arthroscopic RC repair carry a greater risk of complications and revision surgery.[26]

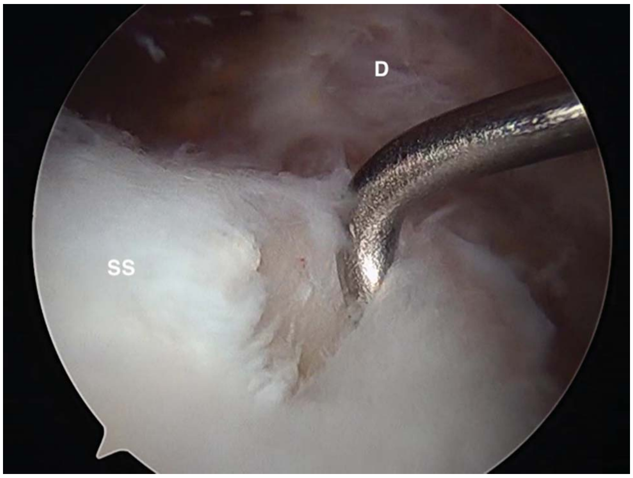
Figure 6



Arthroscopic image showing an articular-sided partial thickness rotator cuff tear in a 47-year-old woman. Copyright K. Plancher, MD, MPH. H = humeral head, SS = supraspinatus

Lo et al[27] presented the results of 37 patients with PRCTs (age 52.9 ± 9.3 years, six acute, 31 chronic) treated with conservative treatment. Functional scores markedly improved at 46 ± 7-month follow-up. Seventy-six percentage did not demonstrate tear progression. Nondominant involved side, atraumatic onset, and tear thickness <50% demonstrated better outcomes. Kim et al[28] conducted a randomized controlled trial (RCT) between immediate repair and delayed repair after 6 months of nonsurgical treatment. Both immediate and delayed repair had notable improvements in pain and functional scores at 31.9 and 37 months, respectively, and similar re-tear rates. Twenty-one percent of the delayed repair group voluntarily withdrew at 6 months after nonsurgical treatment because of improved symptoms. These data

Figure 7



Arthroscopic image showing an bursal-sided partial thickness rotator cuff tear in a 55-year-old man. Copyright K. Plancher, MD, MPH. D = deltoid, SS = supraspinatus

support the use of nonsurgical management of PRCTs and suggest that delayed surgical intervention does not compromise outcomes at up to 3 years.

Arthroscopic Diagnosis

Arthroscopy remains the best method for diagnosing and surgically treating PRCTs (Figures 6 and 7). Field and Lindeman[29] suggest a 30 to 30 position (ie, 30° forward flexion, 30° abduction, and gentle downward traction) in a beach chair position to increase the space below the superior capsule and enhance visualization of the supraspinatus and infraspinatus footprint. Intratendinous tears are often missed on arthroscopic investigation because the outer surface of the tendon is not visualized. Bellows sign, a ballooning of capsular tissue attached to the RC, is indicative of an intrasubstance tear (Figure 8, A and B).[29] A dimple sign is a small clue that near the rotator cable and articular surface there may be an interstitial tear (Figure 9).[29] Palpation is our preferred way of detecting the fall off or hollow feeling between the bursal and articular surfaces. Finally, the most common way to detect this type of PRCT is to push the arthroscopic probe into the defect and watch it fall right in.

Débridement and a subacromial decompression can improve visualization and diagnostic accuracy of bursal-sided tears.[29] Identifying the location of the bursal side of an articular-sided tear can be aided with the placement of a spinal needle inserted from outside (bursal side) through the tear (articular side). A suture may be passed through the needle and retrieved through the anterior portal. The suture is left in place as a marker to aid in definitive treatment of an articular-sided tear.

Surgical Treatment

Surgical intervention should be considered when patients have failed 3 to 6 months of conservative management and in younger patients with acute, traumatic injury and is often directed by patient age, activity level, arm dominance, tear thickness, and location. Biomechanical studies support tear thickness as a major determinant for surgical decisions with tears >50% of tendon thickness yielding increased strain on the remaining portion of the intact tendon.[1] Professional athletes and especially overhead throwers may require alternative treatment in a different timeline.

Figure 8

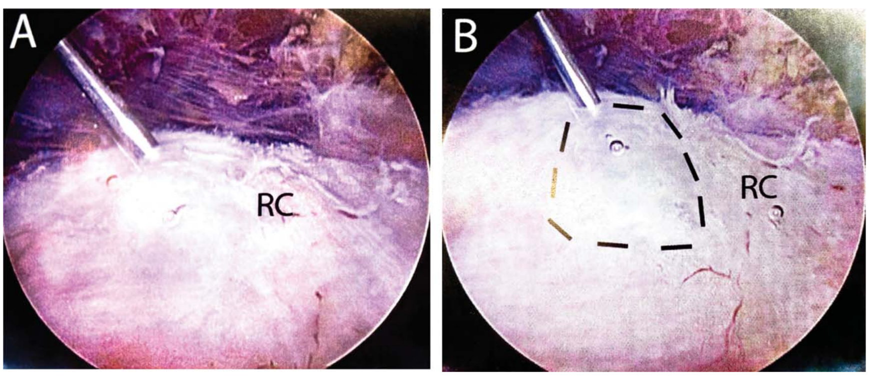


Image showing a Bellows sign in a left shoulder. **A**, A Spinal needle is inserted into the RC at the site of a suspected interstitial RC tear. **B**, As normal saline is injected, the interstitial defect fills and creates a visible bubble (dashed black lines), confirming an interstitial tear of the RC. RC, rotator cuff. (Reproduced from Burkhart SS, Lo IK, Brady PC, Denard PJ. *The Cowboy's Companion: A Trail Guide for the Arthroscopic Shoulder Surgeon.* (Wolters Kluwer/Lippincott Williams & Wilkins: Philadelphia, PA, USA, 2012): Figure 5.43.

Figure 9

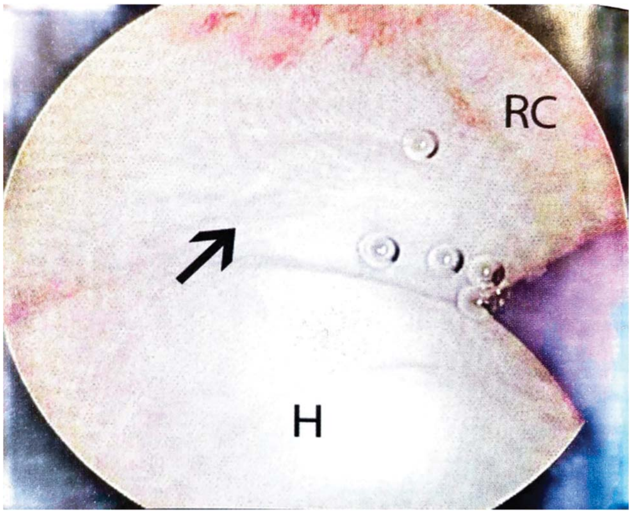


Image showing a dimple sign in a left shoulder. A medial bulge (black arrow) of the rotator cuff is seen from the posterior glenohumeral viewing portal indicative of an interstitial rotator cuff tear. (Reproduced from Burkhart SS, Lo IK, Brady PC, Denard PJ. *The Cowboy's Companion: A Trail Guide for the Arthroscopic Shoulder Surgeon.* (Wolters Kluwer/Lippincott Williams & Wilkins: Philadelphia, PA, USA, 2012): Figure 5.41.

Surgical options include arthroscopic débridement with or without subacromial decompression and acromioplasty, tear completion and repair (ie, conversion repair), and partial repair using various repair techniques. The American Academy of Orthopaedic Surgeons Management of Rotator Cuff Injuries Clinical Practice Guidelines report strong evidence for the use of either conversion repair or transtendinous (TT)/in situ repair for high-grade (eg, tear involvement >50% mediolateral footprint) PRCTs.[25]

Arthroscopic Débridement

Arthroscopic débridement without subacromial decompression and acromioplasty has been shown to be effective in tears <50% of tendon thickness.[30] A full-radius shaver is used to remove the frayed edges to achieve a healthy rim to promote healing. Reynolds et al[31] evaluated return to sport in 82 professional baseball pitchers who underwent arthroscopic débridement of small PRCTs. Sixty-six percent returned to competitive pitching at the professional level, and 55% returned to the same or higher level of competition. These data suggest arthroscopic débridement with or without acromioplasty is a viable option to return athletes with both bursal-sided and articular-sided tears to sport. Although early studies demonstrated added benefit of subacromial decompression, more recent studies do not suggest superior outcomes.[32]

Jaeger et al[33] published a 20-year follow-up in 22 patients with PRCTs who underwent arthroscopic débridement and acromioplasty. Two patients (9%) had revision surgery at 20 years, and 91% were satisfied. Similar findings were reported by Ranebo et al[30] in 45 patients with PRCTs (44 articular-sided and one bursal-sided) undergoing débridement and acromioplasty without RC repair at a 22-year follow-up. Seven percent (3/45) had radiological evidence of cuff tear arthropathy. The authors concluded that most PRCTs remained unchanged with good functional scores at the final follow-up.

Tear location also plays a crucial role. Cordasco et al[34] reported a notable failure rate (38%) in Ellman type 2B (<50% thickness bursal-sided) compared with articular-sided tears (failure rate 5%) treated with arthroscopic débridement and acromioplasty, suggesting that bursal-sided PRCTs involving <50% tendon thickness treated with débridement and acromioplasty have poorer outcomes.

Arthroscopic Repairs

Tear progression is the primary concern when patients present with asymptomatic PRCTs. Repair techniques include in situ repair with TT all-inside, or transosseous techniques and conversion repairs.

In Situ Repairs

In situ repairs preserve the intact tendon and repair the delaminated medial tendon. Although the remaining intact tendon is preserved, repair techniques are technically challenging. TT repair is the most commonly used in situ repair technique (Figure 10).

Ranalletta et al[35] reported excellent outcomes in 80 patients (age 51 ± 5.4 years) with articular-sided PRCTs (Ellman grade 3A, >50% thickness) and TT repair. Improvements in function and pain were evident at 62-month follow-up with 92.5% being satisfied. Five patients developed adhesive capsulitis resolved with rehabilitation. Rossi et al[36] presented a 10.4-year follow-up in 62 patients with PRCTs (53% bursal-sided and 47% articular-sided). Eighty-seven percent of athletes returned to their preoperative sport and 80% returned to the same level with no notable differences between articular-sided and bursal-sided tears.

Good clinical outcomes have also been reported with TT repair in intratendinous PRCTs.[37] Park et al[37] reported clinical and radiographic outcomes in 33 patients (age 53.4 ± 9.1 years) with arthroscopically

Figure 10

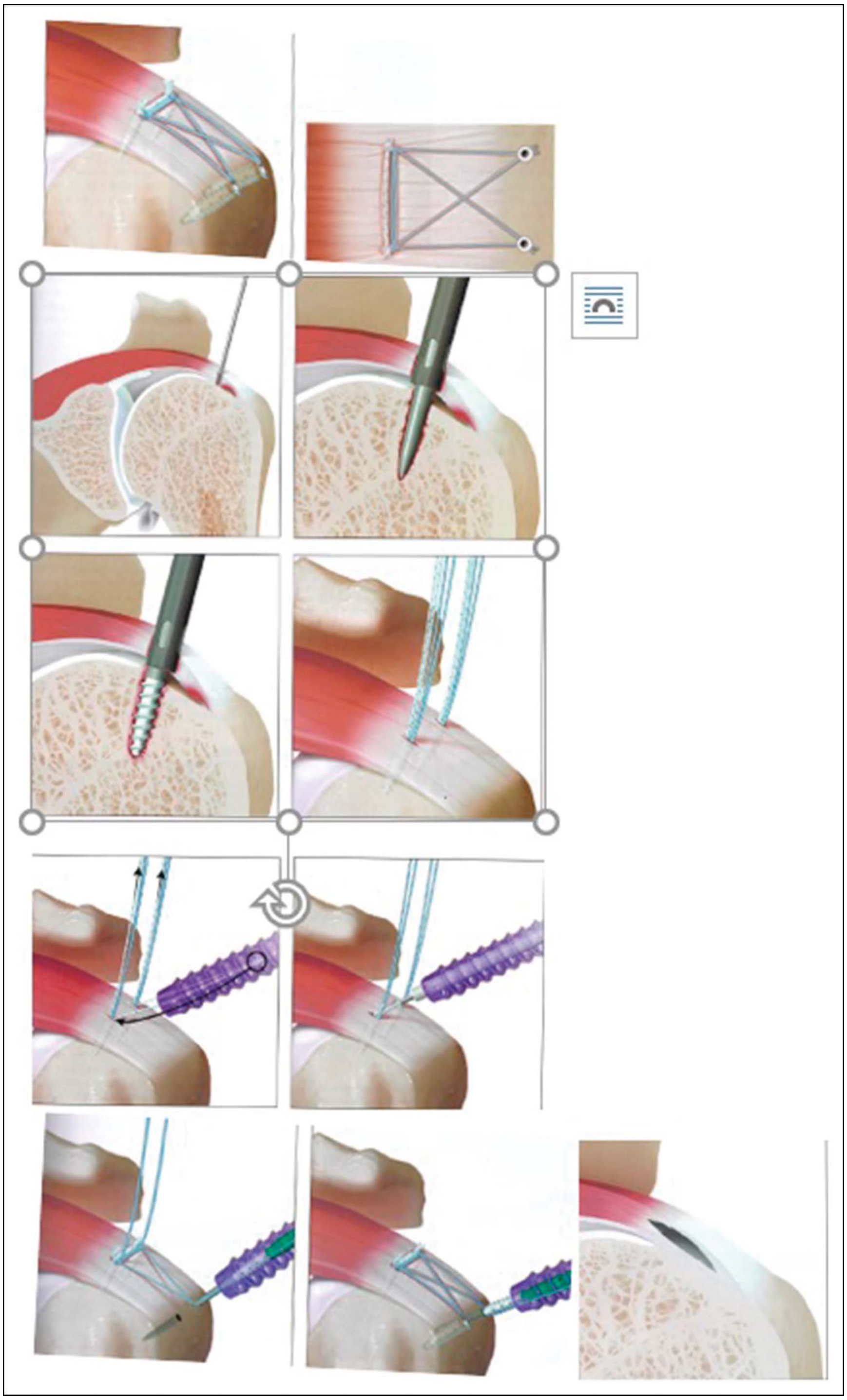
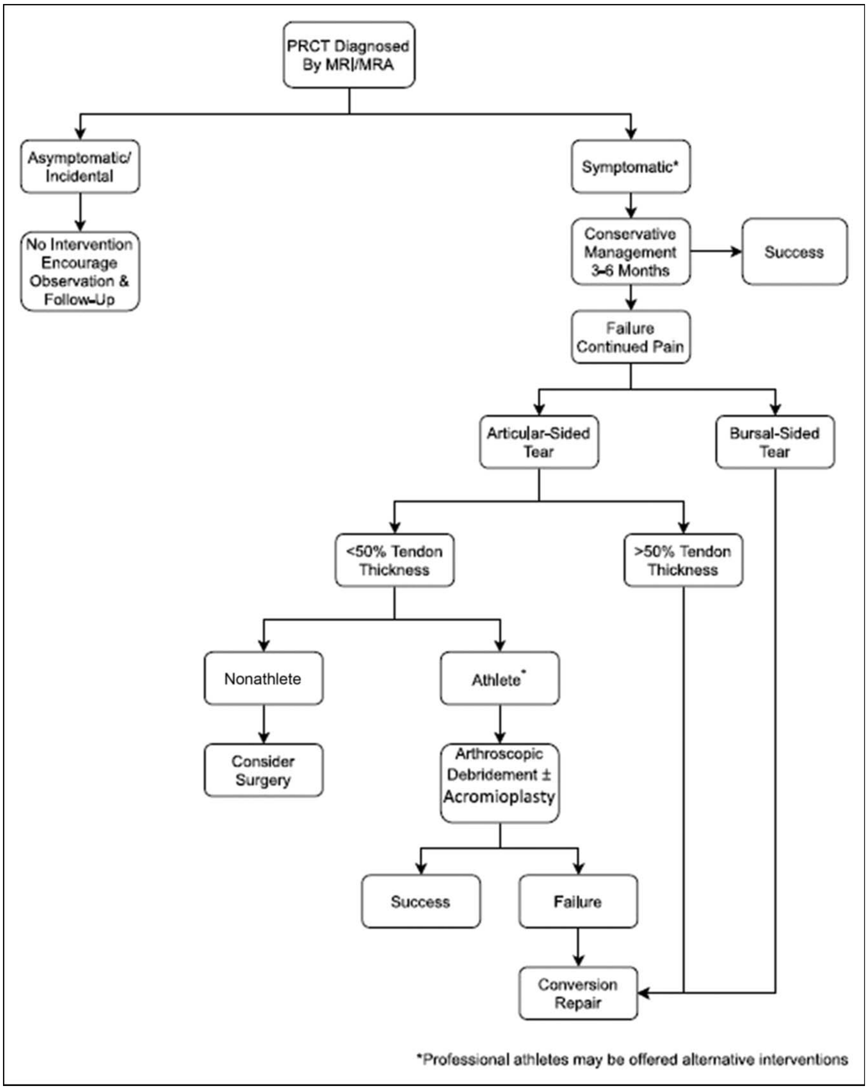


Image showing the transtendinous knotless repair technique. Copyright book. Permission for reproduction pending.

confirmed intratendinous PRCTs who underwent TT suture-bridge repair. At 4.6-year follow-up, 6.1% (N = 2) demonstrated Sugaya type III healing (ie, <50% normal tendon thickness without discontinuity). Notable improvement was seen in functional outcomes and shoulder ROM with 91% demonstrating good/excellent outcomes. Similarly, Xiao and Cui[38] reported good/excellent shoulder function and ROM in 33 patients who underwent bursal side débridement and single-row repair for intratendinous PRCT. Eighteen and a half percent of patients with an MRI follow-up of 15.2 months postoperatively demonstrated Sugaya type III healing.

Figure 11



Flowchart showing an algorithm for treating partial thickness rotator cuff tears.

Conversion Repair

Conversion repair involves incising the portion of the RC from the bursal side or articular side that is morphologically intact. The tendon edges are débrided, and the footprint is prepared in a routine fashion. The FTT is then repaired using the surgeon's preferred technique. Conversion repair allows for the removal of degenerative tissue and better access to the RC footprint for repair. Although good outcomes have been reported with in situ repairs for intrasubstance and articular-sided tears, our preferred method of treatment is conversion repair. Good functional outcomes and retear rates between 9.5% and 35.3% have been reported in the literature.[1]

Conversion Repair Versus In Situ Repair for PRCTs

Aydin et al[39] reported clinical outcomes of conversion repair in 29 patients (age 55.2 ± 7.6 years) with high-grade, bursal-sided tears. Constant score improved from 38.9 preoperatively to 89.2 and 87.8 at 2 years and 5 years postoperatively, respectively. Chung et al[40] analyzed outcomes among 34 consecutive patients with high-grade PRCTs (17 articular-sided, 16 bursal-sided, and one combined, age 57.9 ± 7.2 years) treated with conversion repair. Failure rate was 35.3% (12 patients); failures were attributed to higher tendinosis grade. All functional scores improved markedly at minimum 2-year follow-up.

Table 2. Summary of Studies for Treatment of Partial Thickness Rotator Cuff Tears

Author	Treatment	Number of Patients	Tear Type	Follow-up	Results
Maman et al[14] (2009)	Conservative	26	7-58 mo (mean 20 mo)		MRI Tear progression - 2 (8%) Stable - 23 (89%) Decrease - 1
Lo et al[27] (2018)	Conservative	37	46 ± 7 mo		Notable improvement in ASES and SST MRI tear progression - 9 (24%)
Jaeger et al[33] (2016)	Débridement and acromioplasty	22	19.5-20.5 yr (mean 19.9 yr)		Successful outcomes—90.9%
Ranebo et al[30] (2017)	Débridement and acromioplasty	45	Articular—44 Bursal—1	21-25 yr (mean 22 yr)	Ultrasonography Retear—16 (42%) Mean relative CM—101 WORC—81%
Aydin and Karaismailoglu[39] (2017)	Conversion repair	29	Bursal	5 yr	CSS 38.9/87.8 (pre-op/post-op) ($P < 0.001$) VAS 7.9/1.31 (pre-op/post-op) ($P < 0.001$)
Chung et al[40] (2015)	Conversion repair	34	Articular—17 Bursal—16 Both—1	Minimum 2 yr	Retear—12 (35.3%) CTA VAS, ASES, and UCLA scores improved ($P < 0.001$)
Ranalletta et al[35] (2016)	TT	80	Articular	2 yr	ASES 44.4/76.1 (pre-op/post-op) ($P < 0.001$) VAS 6.3/1.3 (pre-op/post-op) ($P < 0.001$) 92.5% patient satisfaction
Rossi et al[36] (2019)	TT	62	Articular—29 Bursal—33	8-12 yr (mean 10.4 yr)	ASES 45.6/85.1 (preop/postop) ($P < 0.001$) VAS 6.4/1.6 (pre-op/post-op) ($P < 0.001$) No notable difference articular vs. bursal
Castricini et al[41] (2019)	Conversion repair vs. TT	94—conversion repair 59—transtendinous	PASTA lesions	24-142 mo (mean 72.9 mo)	Overall retear rate 13.7% No notable difference in CMS, SST, satisfaction, and retear

ASES = American Shoulder and Elbow Surgeons score, CM = Constant Murley Score, CSS = Constant Shoulder Score, CTA = Computerized Tomography Arthrography, SST = Simple Shoulder Test, TT = transtendinous, UCLA = University of California Los Angeles, VAS = visual analog pain scale

Katthagen et al[1] conducted a systematic review comparing conversion and in situ repair. Six studies (277 patients) analyzed outcomes of conversion repair at 33.7-month follow-up. Retear rate among 146 patients with MRI scans was 15.1%. Complication rate was 4.5% including adhesive capsulitis, scapular bursitis and subcoracoid and subacromial impingement. Higher retear rates were found in bursal-sided compared with articular-sided tears (9.5% and 21.5%, respectively). Outcomes of in situ repair were reported in six studies (236 patients). Retear rate was 12.5% in 152 patients, and complication rate was 4.7% (eg, adhesive capsulitis, bursal-side anchor pullout) among 106 patients at 40.1 months. No difference in outcomes were reported between conversion and in situ repair for PRCTs involving >50% of tendon thickness. Castricini et al[41] compared outcomes of 94 patients with conversion repair and 59 patients with TT repair for PASTA (partial articular supraspinatus tendon avulsion) lesions. No notable difference in Constant and SST scores or satisfaction rates were reported between groups. The retear rate was 13.5% and 13.9% in the conversion repair and TT groups, respectively.

Similarly, Kim et al[42] investigated outcomes in patients with intratendinous tears who underwent bursal débridement plus either single-row repair or in situ suture-bridge repair, arthroscopic débridement and TT repair, or conversion repair with a single-row, double-row, suture-bridge, or side-to-side repair technique. At 2-year follow-up, similar functional outcomes, ROM, and retear rates were reported regardless of repair technique. Three patients (10.7%) demonstrated PRCT retear on MRI or ultrasonography (two articular-sided and one bursal-sided). Kanatli et al[43] compared arthroscopic repair in articular-sided, bursal-sided, and intratendinous tears. Articular-sided tears underwent conversion repair with double-row repair, and bursal-sided and intratendinous tears were treated with repair using a lateral tension band technique. Notable improvements in functional outcomes and ROM were achieved in all patients regardless of tear type and repair technique.

Although there may be a biomechanical advantage of in situ techniques, clinical outcomes do not demonstrate superiority over conversion repair for articular-sided, bursal-sided, or intratendinous tears. Figure 11 proposes a suggested treatment algorithm with a summary of relevant studies in Table 2.

Postoperative Rehabilitation

After surgical repair, the initial phase of physical therapy should focus on protecting the repair site and pain control. Shoulder immobilization with a brace and abduction pillow for approximately 3 to 4 weeks minimizes muscle activation and strain on the repaired RC. Although some have suggested early immobilization is not necessary, others report increased retear rates with early motion.[44] Supine forward flexion to 90° and external rotation to 10° to 20° can be initiated on postoperative day one. Forward elevation greater than 30° and external rotation in the scapular plane minimize strain on the repaired tissue. Internal rotation stretching behind the back should be avoided for a limited time.

The goals of the intermediate phase of healing are to restore shoulder ROM and functional strength. Progression from active-assisted to active ROM supine forward flexion and external rotation exercises can be commenced at 4 to 6 weeks with discontinuation of the abduction pillow splint. The patient should achieve pain-free passive supine elevation to 120° by 6 weeks. Strengthening and resistance exercises can be instituted at 8 to 12 weeks. Sport-specific exercises for overheard throwers may begin at 5 to 6 months with progression through a throwing program to promote return to sports and proper mechanics. Return to sports and manual labor with the exception of professional athletes is usually permitted after 6 months.

Biologic Augments and the New Frontier

Despite the ability to alleviate pain, restore function, and prevent tear progression, high failure rates of surgical repair are still reported within 1 year. Biologic augments, including platelet rich plasma (PRP), platelet rich fibrin, platelet-derived growth factor, anabolic growth factors, bone marrow aspirate concentrate, stem cells, and proteinase inhibitors, have become an attractive adjunct to current repair techniques to improve tendon healing.

Early animal models suggest augmentation with PRP increases failure strain with more linear collagen fibers at 21 days. However, studies have not demonstrated similar clinical benefit with the PRP and meta-analyses demonstrate mixed results.[45-47] Randelli et al[47] delivered PRP at the tendon–bone interface during repair of FTTs in a RCT. Notable improvement was seen at 3 months with PRP; however, there were no differences in outcomes at 6 months. In another RCT, Castricini et al[45] showed notable differences in Constant score with a PRP matrix. A more recent meta-analysis suggested that the addition of PRP to repair of PRCTs and FTTs decreases retear rate and improves healing and clinical outcomes.[46] The lack of PRP standardization compounded by the variety of repair techniques and tear types makes it difficult to achieve consensus on the use of PRP for PRCT repair.

Mesenchymal stem cells are another option to improve healing rates although clinical evidence is limited in PRCT repair. Hurd et al[48] compared an injection of uncultured, unmodified, autologous adipose-derived regenerative cells with a combination of methylprednisolone and bupivacaine injection in patients with symptomatic PRCTs who failed conservative management. The uncultured, unmodified, autologous adipose-derived regenerative cell group showed markedly higher ASES scores at weeks 24 and 52. More evidence is needed to support the clinical use of stem cells in PRCTs healing both with and without surgical repair.

Augmentation with biologic scaffolds has also gained popularity. Augments include synthetic, xenografts, autografts, or allografts patches. A recent meta-analysis of multiple graft types suggests that augmentation results in lower retear rate, with autograft augments achieving superior graft integrity although no difference in clinical outcome scores was evidenced.[49]

Mechanical properties and biologic incorporation of different graft types varies greatly. A bioinductive collagen patch has been shown to yield cellular incorporation, new tissue formation, maturation, implant resorption, and biocompatibility.[50] However, similar to other biologic adjuvants, there is limited evidence for use in PRCTs. Schlegel et al[51] used a bioinductive collagen patch in 33 patients with PRCTs (11 articular-sided, 10 bursal-sided, four intrasubstance, and eight hybrid tears). All patients underwent arthroscopic subacromial decompression without repair, and the patch was "stapled" on the bursal side. Notable improvement in clinical scores was reported at 1 year with MRI evidence of a markedly increased mean tendon thickness of 2 mm. MRI showed complete healing in eight patients (24%), reduction in defect size in 23 patients (70%), one tear remained stable (3%), and one patient had progression to FTT (3%). Long-term research is still needed to support the use of these biologic adjuvants and other patch augmentation alternatives in PRCTs.

Summary

PRCTs continue to be a challenging disease entity. There is good evidence to support a trial of conservative management as the first line of treatment of most patients with PRCTs. When conservative measures fail, factors including patient age, activity level, tear location, and tear size can help guide surgical decision making. Arthroscopic débridement with or without acromioplasty is a reasonable option to treat articular-sided tears that involve <50% of tendon thickness, although the effectiveness in bursal-sided tears remains questionable. Tears that involve >50% thickness should be repaired with conversion repair or in situ repair techniques, which have been shown to be equally effective in the literature. The use of biologic adjuncts in treating PRCTs warrants continued investigation and hopefully will show promise to increase healing rates.

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