Glenohumeral internal rotation deficit: insights into pathologic, clinical, diagnostic, and therapeutic characteristics

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INTRODUCTION

Among throwing sports, repetitive overhead motions of the arm can increase an athlete's risk of shoulder injuries, including rotator cuff tears, labral injuries, and impingement [1]. In addition to these conditions, glenohumeral internal rotation deficit (GIRD) is a common pathology that affects many overhead athletes and leads to declines in performance and quality of life. It is described as loss of internal rotation greater than the increase in external rotation of the throwing shoulder in comparison to the unaffected shoulder [2]. The increase in external rotation of the arm allows the athlete to achieve an optimal arm position in order to produce the greatest hand and ball velocity [3]. Although this adaptive change is beneficial, a decrease in the internal rotation of the arm often accompanies this change [3]. GIRD can be caused by a repetitive cocking motion due to the resulting posterior capsular and rotator-cuff tightness [4].

In a study by Tokish et al. [5], 35.0%–43.0% of asymptomatic baseball pitchers were observed to have GIRD, suggesting the prevalence of this condition among overhead athletes. Another
study by Wilk et al. [6] stated that the presence of GIRD nearly doubled the risk of shoulder injury in pitchers. In a study by Hel-lem et al. [7], the authors suggested consideration of GIRD along with other factors when calculating overhead athletes’ risks for injury, especially in younger throwers. Although some overhead athletes may be asymptomatic, it is important to understand the risks to these athletes to maintain their safety and performance.

Exploring current literature is necessary to provide a holistic perspective on the prevalence, symptoms, treatments, and risks of GIRD. To our knowledge, no recent studies have reviewed the current body of knowledge available regarding GIRD. As such, the purpose of this study was to explore the symptoms, etiology, risk factors, therapeutic management options, and relevant considerations based on reported clinical outcomes.

INCIDENCE AND PREVALENCE

Actions that involve throwing motions and overhead activities can predispose to the development of GIRD [4]. As such, the prevalence and incidence of this pathology are particularly high among sports like baseball, handball, and volleyball [4]. In one study, Ohuchi et al. [8] explored the risk factors for GIRD in adolescent athletes and noted only participation in overhead sports. Among 214 athletes in their study, 34 had GIRD, and 20 of them participated in overhead sports (P = 0.007). Another study by Sri-vastav et al. [9] explored the prevalence of GIRD in collegiate athletes who partake in overhead sports and determined that 29.1% of the participants had the pathology. These findings were corroborated by many other studies in the literature and have generated concerns regarding the effects and prevalence of GIRD among throwing athletes [9].

A study by Shanley et al. [10] showed that the prevalence of GIRD in baseball players is around 25.0%, while another study by Lee et al. [11] reported a prevalence of 21.4%. Pitchers have been reported to be particularly susceptible to GIRD due to the repetitive and high-force nature of their throwing mechanics [12]. Schmalzl et al. [13] explored the prevalence of GIRD in male handball and volleyball athletes of different training levels and studied its relationship with other pathologies. The authors [13] reported that, among 134 handball and volleyball players, 72.0% presented with GIRD. They also added that GIRD was more prevalent in those who have participated in the sport for a longer time, who train more frequently, and who participate in handball [13]. Another study by Cigercioglu et al. [14] explored the prevalence of GIRD among junior tennis players and found that 19 of 42 (45.0%) participating athletes had the pathology, while also noting the existence of significant differences in strength, range of motion, and functional performance between their non-dominant and dominant shoulders. Many other studies have explored the impact of GIRD on throwing athletes and delineated the prevalence and harm that can be generated by this pathology, generating much interest in interventions that can help limit its progression and alleviate its symptoms [15-19].

ETIOLOGY AND BIOMECHANICS

GIRD is primarily associated with stiffness of the posterior capsule in the glenohumeral joint. When the posterior capsule of the glenohumeral joint tightens, it causes translation in the opposite direction. Thus, tightness in the postero-inferior capsule results in postero-superior translation, whereas tightening of the posterior capsule leads to antero-superior translation [20]. Patients with GIRD commonly report new-onset posterior shoulder pain during late cocking, reproducible through palpation of the posterior and surrounding aspects of the affected joint [4,21]. This condition manifests as tightness, leading to a decrease in the total range of motion, particularly in internal rotation [22-24]. Clinically, GIRD is characterized as an asymmetrical difference in glenohumeral internal rotation exceeding 20° resulting from contracture of the posterior glenohumeral joint capsule and the posterior band of the inferior glenohumeral ligament [23,25].

The pathological progression of GIRD in throwing athletes can involve the intra-articular shoulder, extra-articular shoulder, and pathology related to the kinetic chain. These structures include the labrum, joint capsule, articular-sided rotator cuff (intra-articular), bursal-sided rotator cuff, and acromion (extra-articular) as well as components of the kinetic chain such as the lower extremity, core, scapula, and elbow [4,21-24]. GIRD primarily affects the dominant arm of overhead athletes and contributes to a disabled throwing shoulder [22,25,26]. In throwing athletes, alterations in the kinetic chain are characterized by increased upper trunk rotation toward the non-dominant side and decreased total rotation of the pivot leg to alleviate limitations in shoulder internal rotation and to uphold optimal functional performance during upper extremity activities [23-25,27]. Nonetheless, current evidence regarding the asymmetric deficiency angle of GIRD is conflicting, with some studies suggesting that adaptive GIRD can mitigate the likelihood of injury occurrence and others suggesting that it can lead to different shoulder and elbow pathologies [1,28,29].

Tightening of the posterior capsule and rotator cuff results from the formation of scar tissue from repetitive movements leading to capsular injury and subsequent involvement of neighboring structures in the affected arm [26,29-31]. Alterations in

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supraspinatus tendon thickness correlate with deficits resembling GIRD, whereby increased tendon thickness restricts glenohumeral internal rotation while concurrently facilitating external rotation [31]. Moreover, during the early stages of childhood, the humerus exhibits a relatively retroverted position. In response to the glenohumeral joint response to stress, the humerus begins rotation toward antetorsion as maturation progresses. In overhead athletes who began their training during childhood, the throwing arm tends to display retroversion compared to the contralateral side. This process can strain the epiphysis, potentially leading to epiphyseal changes that restrict the natural progression towards antetorsion during maturation. Consequently, modifications in humeral retrotorsion and glenoid retroversion are observed among competitive throwers at collegiate and major league levels [7,32-35]. Humeral retrotorsion can cause tightness in the posterior capsule and posterior shoulder musculature as the anterior capsule and anterior shoulder musculature may become stretched or weakened, resulting in a loss of internal rotation [1,4,30]. These alterations not only contribute to the advancement of GIRD, but also modify the center of the glenohumeral rotation axis, affecting joint kinematics [4,30].

The risk of capsular injury–induced GIRD is greatest in pitchers, particularly with shoulder hyperextension in abduction during the late cocking and early acceleration phase. During an overhead throw, the scapula undergoes specific movements. In the late-cocking phase, the scapula retracts, posteriorly tilts, and upwardly rotates. Then, during acceleration and follow-through, it protracts [36]. These coordinated actions optimize shoulder positioning and movement throughout the throw [20]. The elevated risk of GIRD in overhead athletes warrants a high degree of suspicion. Patients commonly exhibit restricted internal rotation mobility at 90° relative to the unaffected shoulder. When assessing the shoulder in patients suspected to have or at risk for GIRD, it is crucial to ensure scapular stabilization during the evaluation of supine abduction and external rotation and supine abduction internal rotation measurements.

However, symptoms of GIRD can be mistaken for chronic scapular adaptations, making it critical to differentiate pathological from non-pathological conditions. Athletes may experience scapular adaptations that lead to alterations in position, dynamics, orientation, and motion, which are collectively known as scapular dyskinesia [20]. In non-pathological GIRD, the arc of motion is the same bilaterally (internal rotation deficit is equal to external rotation gain in the affected arm). Therefore, we recommend periodic motion profile testing to establish a baseline total range of motion to compare results when an injury is suspected [20]. Moreover, clinical decisions should not be made based on the results of a single test and should instead incorporate the patient’s entire shoulder-motion profile.

**DIAGNOSIS**

**History and Physical Exam**

Throwing athletes who exhibit shoulder symptoms must be evaluated with a high index of suspicion of GIRD. Therefore, passive internal rotation and total rotational motion (external rotation + internal rotation) of the shoulder must be evaluated in all throwers who are experiencing shoulder pain. The most typical symptoms that patients report are discomfort that radiates to the posterior shoulder [37], shoulder stiffness, and a few athlete-specific signs such as a prolonged warm-up and a decrease in arm velocity (Fig. 1) [38].

On physical examination, reproduction of the pain can be achieved with palpation of the posterior joint line and surrounding soft tissue of the affected shoulder [21]. Shoulder internal rotation assessment can be conducted in several ways. When performing the first technique, the patient should be placed in a supine position on the examination table while the examiner is flexing both elbows and abducting both shoulders to 90° [4]. Measurements of external and internal rotations are compared to those of the contralateral shoulder, and variations can be detected using a goniometer [4]. The point at which the scapula starts to rise from the examination surface is known as maximum passive internal rotation [39]. A discrepancy ≥20° compared to the contralateral side is typically regarded as indicative of GIRD [4]. Another simple method, which is more reliable and not affected by elbow or scapulothoracic motion, is to assess the achieved vertebral level during internal rotation [40,41]. Moreover, internal rotation assessment can be done with the patient lying in a lateral decubitus position with the affected shoulder up and in a 90° abduction, neutral rotation, and the arm in maximum abduction [42]. Then, the distance traveled by the medial epicondyle is measured in centimeters, the difference between shoulders is calculated, and a correlation between loss of abduction and internal rotation loss is made, with every 1 cm corresponding to a 5° loss, respectively [24,42]. A discrepancy ≥20° compared to the contralateral side is also indicative of GIRD (Fig. 1).

Other findings include deep posterior shoulder pain when the shoulder is in maximum passive abduction and external rotation and is indicative of a posterosuperior labral tear and partial articular-sided supraspinatus tear lesion [43]. This is called the posterior impingement sign [10,43]. Furthermore, the presence of a sulcus sign can be seen in patients with GIRD [44]. Additionally, the so-called “SICK” scapula—which consists of scapular malpo-
sition, inferior medial border prominence, coracoid discomfort and malposition, and dyskinesis of scapular movement—is another characteristic pathologic finding in the evaluation of GIRD [22]. The easiest way to determine the scapula’s static position is from behind. It can be useful to draw the bony prominences bilaterally for comparison. The patient’s maximal forward elevation can then be evaluated for active motion, and any dyskinesia can be identified (Fig. 1) [22].

**Imaging**

On imaging, plain radiographs are often non-diagnostic of GIRD. Nevertheless, a rare finding is Bennett’s lesion—an osteophyte in the posterior glenoid that can be seen on the axillary view of shoulder radiography [4]. Furthermore, computed tomography imaging could be contributive when it reveals sclerosis of the posterior glenoid rim [45].

However, magnetic resonance imaging (MRI) remains the imaging modality of choice in the diagnosis of GIRD, and it can be useful in ruling out other existing pathologies [4]. This imaging modality can show posterior glenoid internal impingement [4]. Other features include partial undersurface tears of the supraspinatus and anterior infraspinatus, wearing of the chondral glenoid, bony cystic changes at the humeral head in its posterosuperior part, thickening of the posterior band of the inferior glenohumeral ligament, superoposterior humeral head subluxation, and labral pathologies such as type II superior labrum anterior-to-posterior (SLAP) tears and posterosuperior labral tears (Fig. 1) [46,47].

Another modality that can be considered is magnetic resonance arthrography, which can detect SLAP lesions as well as partial rotator cuff tears, especially when the shoulder is placed in abduction and external rotation [48-50].

**TREATMENT**

**Conservative Treatment**

Nonoperative management of GIRD is typically preferred over surgical management. Treatment begins with prolonged physical therapy, where stretching and strengthening will be the core of the treatment plan. The goal of physical therapy is to increase both internal rotation and the total rotational range of motion. Common stretches used to relieve tightness of the posterior shoulder that contributes to GIRD include the following: sleeper stretch, cross-body adduction stretch, prone-passive stretch, all-fours posterior stretch, and doorway stretch [4]. Stretching of the posterior shoulder and postero-inferior capsular muscles can relieve symptoms associated with GIRD in approximately 90.0% of throwers [25]. Among these stretches, the sleeper stretch has also been found to increase the acromiohumeral distance in overhead athletes, whose acromiohumeral distance has shortened, causing external impingement of the bursal side of the rotator cuff [51]. Several studies support a daily stretching protocol to increase internal rotation and to prevent GIRD [52,53].

Instrument-assisted soft tissue mobilization (IASTM) is also used to treat GIRD in overhead athletes. IASTM uses an ergonomically designed tool that induces microtrauma to affected soft tissue, mobilizes scar tissue, and stimulates a local inflammatory response. Gohil et al. [54] reported that IASTM significantly

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**Fig. 1.** Summary of the clinical investigation conducted to confirm glenohumeral internal rotation deficit (GIRD) diagnosis. CT: computed tomography, MRI: magnetic resonance imaging.
improved the range of motion and flexibility in overhead athletes with GIRD. When IASTM is combined with stretching, it provides greater internal rotation improvement than that achieved with stretching alone [55].

Muscle-energy techniques (METs) and myofascial release (MFR) have been shown to improve shoulder function in overhead athletes with GIRD. METs use post-isometric relaxation to lengthen muscles and fascia that lack flexibility, while MFR is comprised of manual pressure to release tension within the fascia. Single application of METs in overhead athletes with GIRD can increase their internal rotation and horizontal adduction [56]. Immediate improvement of internal rotation has also been observed after a session of MFR [57]. Although both techniques improve shoulder function in overhead athletes with GIRD, MET is more effective at improving internal rotation [58].

Hold–relax proprioceptive neuromuscular facilitation (PNF) has also been reported as a non-surgical treatment option for overhead athletes with GIRD. Hold–relax PNF places the joint at the maximum range of motion with isometric contraction against resistance. When hold–relax PNF is combined with vibration therapy, it can increase internal rotation in overhead athletes with GIRD [59].

**Surgical Treatment**

When conservative treatments have been exhausted, some surgical interventions can be considered to provide symptomatic relief for the presenting patient. As stated previously, overhead athletes commonly present with SLAP tears, and these may require arthroscopic shoulder surgery. There are multiple SLAP surgical repairs and implants that may be used, and the choice between them is dependent on surgeon preference; hence, there exists an inconsistency in the literature as to how well overhead athletes perform after their procedure. Thayaparan et al. [60] reported that 69.6% of overhead athletes who underwent arthroscopic repair for SLAP tears returned to play at their prior level of performance, following an average of 9 months of recovery. Type II SLAP repairs are most commonly surgically repaired, with a high number (> 70.0%) of overhead athletes returning to play at their pre-injury level of competition [61,62]. If SLAP repair fails, biceps tenodesis has been shown to help alleviate pain and to lead to good return-to-sports rates among overhead athletes [63-65]. Arthroscopic posterior-inferior capsular release has also been shown to improve GIRD. To achieve capsular release, the muscle bellies of the posterior rotator cuff are completely exposed. Coddington et al. [66] reported that 77.0% of overhead athletes returned to their same or a greater level of play following this procedure.

**SEQUELAE AND RELATED PATHOLOGIES**

If a patient with GIRD fails to seek treatment or if treatment options fail to relieve the presenting symptoms, GIRD can cause debilitating impacts beyond soft tissue structures [4]. According to Lee et al. [67], patients with GIRD sustain an imbalance in shoulder rotator muscle strength. In their study, 24 players (10 pitchers and 14 field players) were evaluated, including 10 with GIRD and 14 without GIRD. The external rotation/internal rotation ratio was significantly lower (35.7% ± 5.0% vs. 55.5% ± 6.2%; 95% confidence interval, –24.7 to –14.7; effect size: –3.515; P < 0.001), and the muscle strength of the internal rotation muscles was significantly greater (75.0 ± 7.6 Nm/kg × 100 vs. 55.7 ± 16.4 Nm/kg × 100; 95% confidence interval, 7.7–30.9; effect size: 1.510; P = 0.002) in the throwing shoulders of the group who had GIRD than in the group who did not [67].

Studies on collegiate and professional baseball pitchers have revealed bone alterations, including greater humeral retroversion, despite the historical belief that the adaptive changes seen in GIRD primarily affect soft tissue [68]. Crockett et al. [68] reported that throwing athletes’ dominant shoulders showed more humeral and glenoid retroversion than their non-dominant shoulders as well as greater external rotation at 90° and 45° of abduction and less internal rotation at 90°. In the study, the subject groups were 25 male professional baseball pitchers, all of whom began pitching before the age of 10 years, and 25 male control participants, all of whom did not participate in any overhead throwing activities [68].

According to the results of Shaffer and Huttman [69], tears of the rotator cuff are common in the throwing athlete. The rotator cuff experiences extreme stress when performing repetitive overhead activities. These supraphysiological strains have the potential to damage the cuff fiber, most frequently on the underside where tensile overload takes place. The cuff becomes increasingly compromised as a result of intrinsic shear stresses and undersurface fiber failure, which are exacerbated by a tight posterior capsule, anterior instability, and internal impingement.

Multiple studies have demonstrated that the peel-back mechanism, involving the biceps anchor and the posterosuperior labrum, retracts during the late cocking motion, increasing the risk of SLAP lesions in throwers with GIRD by 25.0% [4,45,70]. However, the hallmark of GIRD is a posterosuperior labral tear [4,70]. Lesions to the posterosuperior labrum and articular side of the supraspinatus tendon result from the greater tuberosity impinging against the glenoid rim during late cocking.

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CONCLUSIONS

GIRD is a shoulder pathology common in young athletes participating in overhead sports. Diagnosis can be confirmed through detailed history-taking and a comprehensive physical examination. Imaging studies, particularly MRI, can be helpful in evaluating the presence of possible concomitant pathologies like SLAP tears, rotator cuff tears, and tightness of surrounding ligaments. Treatment options are mainly conservative and revolve around physical therapy regimens that improve internal rotation and relieve symptoms of posterior tightness. Surgical options are reserved for when symptoms persist despite trials of physical therapy and when concomitant pathologies, like SLAP tears, are severe enough to warrant intervention. Throughout the entire management process, proper education on the different facets of the disease to the patient is pivotal to ensure patient satisfaction, engagement, and adherence to therapeutic instructions.

NOTES

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