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A comprehensive rehabilitation program for posterior instability of the shoulder

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ABSTRACT

Posterior shoulder instability is recognised as being less prevalent than anterior instability, however the diagnosis of this pathology is easily overlooked or missed and this may contribute to an underestimation of prevalence. Recently, there has been increasing recognition of this condition and consequently a greater requirement for knowledge of diagnostic procedures and treatment directions. Currently there is limited research into the conservative management of posterior instability, although it is recommended as first-line treatment prior to surgical review, particularly in those with an atraumatic instability mechanism. The aim of this paper is to outline a comprehensive rehabilitation program for the conservative management of posterior instability with a focus on scapular and humeral head control. The information provided includes extensive written information, flowcharts, figures and a table of management parameters that will provide therapists with adequate detail to replicate the program in the clinical setting.

Posterior instability: incidence and prevalence

Glenohumeral joint instability is estimated to affect 2% of the general population,[1] with anterior the most prominent direction of instability. The incidence of anterior shoulder dislocation has been reported to range from 8.2 to 56.3 dislocations per 100,000 people per year,[2-5] with a projected prevalence of 1.7%.[6] The prevalence of instability is likely underestimated when based on dislocation data alone because subluxations occur at a rate of 5 times higher than dislocations in young athletes.[7] Traditionally, posterior instability has been considered to represent less than 10% of all instability cases[8-10]; however, posterior subluxation and translational instability can be easily overlooked and misdiagnosed[11] and this may contribute to the low reported incidence of this condition. More recently, there has been increasing recognition of this pathology in active populations.[12,13]

Posterior instability: classification and pathogenesis

There is no consensus on classification systems for categorizing instability,[6,14] and the classification of posterior instability remains controversial.[15] The mechanism of injury (traumatic versus atraumatic), severity (subluxation vs dislocation), direction of instability, presence of ligamentous laxity, and presence of volitional instability have all been described as considerations for classifying glenohumeral instability.[14,16,17] Posterior instability has previously been described by the severity of instability (acute dislocation, chronic fixed/locked posterior dislocation, and recurrent subluxation)[18] and mechanism of injury.[15] Posterior instability can be associated with a single traumatic onset (such as a tackle in football or rugby), repetitive overuse and microtrauma (such as a backhand in tennis), or soft tissue disorders.[15]

In the presence of a history of trauma and resulting unilateral instability, structural lesions are more likely and can include posterior inferior glenohumeral ligament injury, reverse Bankart lesion, reverse humeral avulsion of the glenohumeral ligament (HAGL), posterior labral tears, or posterior capsuloligamentous complex stretch.[15] Bidirectional posterior instability is combined posteroinferior instability, which is typically posterior instability

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superimposed on an underlying hypermobile joint.[13] It can also be associated with a structural lesion and can be induced either through traumatic mechanisms or overuse. It is estimated that up to 30% of the normative population is hypermobile,[19,20] and there is some evidence that this predisposes an individual toward instability.[20,21] Posterior instability is often present in multidirectional instability (MDI) of the shoulder.

Posterior instability can also be associated with congenital anomalies such as posterior capsulolabral redundancy or deformation and glenoid retroversion (increase posterior angulation of the glenoid fossa).[22] These will result in a posteriorly centered humeral head (HH) which may predispose to posterior instability, and usually, if it is present, it occurs bilaterally.

Clinical examination

Posterior dislocation is notorious for being "missed" at examination.[23] It is difficult to recognize clinically (chronic fixed/locked posterior dislocation) as the HH can position itself posteriorly behind the glenoid being masked by the overlying musculature.[24,25] The clinical diagnosis is further hampered by the fact that many shoulders with significant posterior structural lesions (labral and ligament tears) do not present with a history of requiring reduction.[26]

Subjective assessment

In our experience, it is particularly important to listen for a "moment in time" (an incident or activity) when symptoms started. An incident may be indicative of a structural lesion of significance.[22] Often patients are still able to move their arm at the time and that they do not actually feel it dislocate or sublux, but they are aware something happened or "went" in their shoulder. The next day their arm is worse and more sore and restricted in motion, and it can take weeks to recover.

Patients typically present complaining of pain when performing certain activities, and instability of their shoulder is a secondary concern.[27] The most aggravating positions are loading the arm in flexion, horizontal flexion, and internal rotation (IR) or any combinations thereof.[22] Aggravation of pain can occur during pulling off a top, turning a steering wheel, lying on the side, bench press, push ups or overhead weights, and any use of the arm in long-lever flexion, particularly weighted flexion.[28] Patients can complain of pain in both the front and back of the joint.[28] The HH sliding excessively posteriorly can stress soft tissues on both sides of the joint in a similar way to what has been described as occurring with anterior instability and internal impingement but in reverse.[29] The pain in the front may be derived from the long head of biceps and/or the top edge of subscapularis as these structures can be compressed intraarticularly as the HH abuts the glenoid. In some more extreme or chronic cases, odema, compression, and flattening of the HH can occur or even Reverse Hill-Sachs lesions can be seen on magnetic resonance imaging and at surgery.[22,26]

Posterior joint pain is also common and this can correlate with either a posterior labral tear or posterior glenoid chondral surface damage.[22,26] In our experience, the posterior pain can be more generalized in some cases, and patients may report a gripping or tightening feeling in the back of the joint, particularly with movement into horizontal flexion. This may lead to a misdiagnosis of a stiff posterior capsule as horizontal flexion appears blocked or stiff when it is actually guarding.

If the joint is hypermobile, the patient may not always be aware that their shoulder has developed excessive translation as they have always had "clunking," "clicking" loose feeling shoulders.

Physical examination

The diagnosis of posterior instability, like most shoulder pathologies, cannot be made by one stand-alone diagnostic test. A recent systematic review by McIntyre et al[28] revealed that for diagnosis, clinicians must rely on a combination of a thorough history, including the mechanism of injury, symptoms and recognition of risk factors as well as clustering of the Jerk test, Kim test, and posterior impingement sign. In addition, examination for glenohumeral joint laxity is essential to determine which subgroup of posterior instability the patient may belong.[30]

The clinical diagnosis of posterior instability can be further complicated by the coexistence of acromioclavicular pathology. Whether this association is due to a direct biomechanical linkage between posterior translation of the HH and increased stress on the acromioclavicular joint or due to the fact that the provocation position for posterior instability (flexion, horizontal flexion, and IR) is similar to that for the acromioclavicular joint[31,32] is currently not known.

The excessive posterior translation of the HH can set up articular surface cuff changes around the posterior cuff (supraspinatus and infraspinatus) creating clinical signs of pain and weakness and sometimes wasting around the posterior cuff.[26] The secondary or coexisting rotator cuff signs that are often present can mean that the diagnosis is missed or delayed as treatment remains focused on the rotator cuff and not the underlying joint instability. This is particularly exacerbated in posterior instability due to the fact that frank instability or subluxation is rarely a primary feature of this pathology.

The range of motion presentation in posterior instability is dependent on the history, subgroup, and chronicity. If a patient presents acutely after a traumatic event, typically the range of motion will be painful and restricted, particularly in flexion, horizontal flexion, and IR. Excessive translation of the HH posteriorly can be seen and palpated, and motions are painful and guarded. Clinicians should keep in mind that chronic locked dislocations can occur.[25] The patient will present with an altered contour of the glenohumeral joint anteriorly where the HH normally sits. The joint will appear flattened, and there will be a prominent coracoid process. The glenohumeral joint is locked, and there is gross weakness in the external rotators (Fig. 1). The diagnosis is confirmed by radiological investigation. In most clinical practices, patients typically present nonacutely. In general, abduction and external rotation (ER) ranges are nonrestricted in range although there may some abduction arc pain coming from any secondary rotator cuff

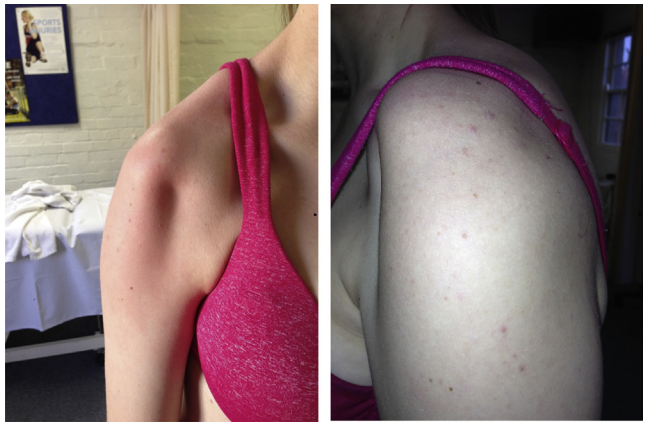

Fig. 1. Chronic locked dislocation. Altered contour of joint anteriorly, flattened joint with a prominent coracoid process.