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Anterior shoulder instability in collision and contact athletes

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ABSTRACT

Glenohumeral instability is a common problem for the collision and contact athlete. An understanding of the pathoanatomy, aetiology and injury mechanisms can help guide the clinical examination and appropriate investigations. This in turn can help guide appropriate management of patients, with the aim to return them to pre-injury levels of sport.

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Contents

1. Introduction

Glenohumeral instability is a common disability for the collision and contact athlete. It accounts for 23% of shoulder injuries in American Collegiate Athletes,[1] and in Australian professional rugby union players was the soft tissue injury that led to the greatest time off.[2] Similar results were found in the UK professional rugby competition where glenohumeral instability results in the highest rate of absence from playing and training and has the highest recurrence of all shoulder injuries.[3] Rugby league, Australian Rules football, lacrosse and ice hockey all had similar high rates of shoulder injuries and instability.[4–7]

Collision and contact sports are often treated as the same patient group. Collision sports are best described as sports where the athletes purposely and repeatedly collide at high force with each other or inanimate objects, such as the ground. Sports such as rugby union, rugby league, lacrosse, American football and boxing are typical collision sports. In contrast, in contact sports whilst collisions still occur regularly during the game, such as soccer and basketball[8,9] they usually involve lower levels of force. It may be important to differentiate between these two groups, as we know that a sport such as rugby union has a specific and different high velocity injury pattern to many other sports.[10,11]

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2. Pathoanatomy of anterior instability

The glenoid labrum improves the stability and depth of the glenohumeral articulation[12] but also act as the insertion of the glenohumeral ligaments to the glenoid. Removal of the labrum in cadaveric specimens leads to easier dislocation in the anterior and inferior direction.[13] Avulsion of the labral attachment of the antero-inferior glenohumeral ligament (AIGHL), between the 3 and 5 o'clock position, in traumatic anterior shoulder instability, was described in detail by Blundell Bankart in 1938[14] (Fig. 1). This Bankart lesion is the most common lesion seen in first time traumatic shoulder dislocations.[15],[16] However, we now understand that the soft-tissue injury not only involves avulsion of the AIGHL from the glenoid but the ligament may also avulse from the humeral side (HAGL), mid-substance capsular injuries or in combination.

The soft tissue Bankart tear can occur with an associated avulsion fracture of the anterior glenoid rim (bony Bankart lesion), particularly with the higher energy dislocations seen in collision and contact sports. Further erosive glenoid bone loss can occur in recurrent shoulder instability and has been reported as affecting up to 90% of patients.[17] Higher rates of glenoid bone defects have been found following traumatic dislocations in younger aged patients.[18] Bone loss decreases the safe zone that the humerus can pass through before becoming reliant on the AIGHL for restraint.[19] Itoi et al in 2000 found in a cadaveric model, that in the presence of glenoid bone loss of greater than 21%, repair of a soft tissue Bankart lesion failed to provide sufficient stability in translation and external rotation.[20]

Bone defects can also occur on the humerus, as a pathological impaction fracture of the posterior humeral head as described by Hill and Sachs in 1940.[21] The arthroscopic appearance of the Hill Sachs lesion was well described in 1989 by Calandra et al, who found it present in a high proportion of their instability cases.[22] Burkhart and De Beer reported a 100% failure rate for arthroscopic stabilization procedures in patients who had an engaging Hill Sachs lesion, which they defined as a defect that engages with the glenoid in a functional position of abduction and external rotation.[23] However, it is the combined relationship of bone loss on both the humerus and glenoid that determines the implications on instability. Yamamoto quantified this bipolar loss via the "glenoid track" concept. The glenoid track was defined as the contact zone between the humerus and the glenoid during maximal external rotation and increasing degrees of abduction. In a cadaveric model they mapped out this contact zone and deemed this the glenoid track. When the Hill Sachs lesion fell medial to this, they were defined as "off track" and therefore more likely to engage during physiological range. "On-track" lesions were contained within the track and therefore should not engage in physiological movement. The measurement from the posterior rotator cuff footprint to the medial margin of the normal glenoid track measured 84% of the width of the glenoid, with glenoid bone loss leading to a narrower glenoid track and potentially more "off track" lesions[24] (Fig. 1). Kurokawa et al found that "off track"" humeral lesions were associated with glenoid bone loss of at least 12%, with more than half of the patients having glenoid bone loss of greater than 20%. [25]

3. Aetiology and injury mechanism

The mechanism of injury pattern can guide the clinician to the injury and commonly occurring associated lesions. Within rugby, the injury mechanisms can be broken down into an injury involving the tackler, try-scoring injury, direct impact injury and flexed fall injury[10],[26] (Fig. 2). However, these positions are frequently replicated in all collision and contact sports.

The tackler has a posteriorly directed force applied to the abducted, externally rotated arm, usually leading to an anterior shoulder dislocation. This will often lead to the standard Bankart lesion, with SLAP tears and HAGL injuries also common.

The try-scorer has a posterior force applied with the arm in flexion rather than abduction. Whilst both Bankart tears and SLAP tears are common, rotator cuff tears are more common with this mechanism (Fig. 3).

The direct injury mechanism is when the athlete falls directly onto the lateral side of the shoulder with the arm held by the side, often in internal rotation. This exerts a large compressive force across the glenohumeral joint leading to a higher rate of bony glenoid lesions as well as complex labral tears. Fractures around the shoulder girdle as well as acromio-clavicular injuries are also common.

Flexed fall injury is the last common mechanism, where the athlete falls onto the elbow with the arm held in a flexed posture. This results in a posteriorly directed force across the shoulder joint causing higher rates of injury to the posterior shoulder, including posterior labral and glenoid damage, posterior HAGL tears and posterior rotator cuff injuries.

4. History & clinical examination

A detailed description of the mechanism of injury is crucial, as this will frequently provide much of the information required. However, shoulder instability can occur as subluxation events

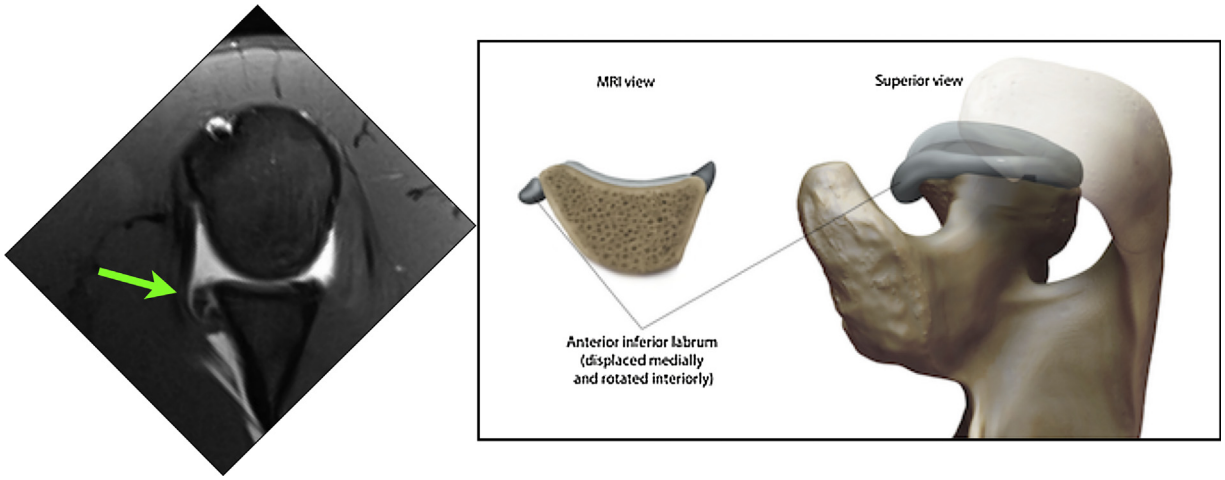

Fig. 1. Labral tear.

Antero-inferior labral tear as described by Bankart.

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