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Functional Anatomy and Pathology of Traumatic Shoulder Instability

The increasing popularity of arthroscopic shoulder procedures in combination with advanced imaging techniques has rekindled interest in anatomy. Although one is looking deep into the shoulder, arthroscopic anatomy still is more like surface anatomy. Unfortunately, what one sees is not always what one gets. In imaging, higher resolution ultrasound and MRI have increased detail to such a level that it sometimes becomes difficult to differentiate normal anatomic structure from pathology or artefact, for example partial volume effects in MRI. Therefore, being able to correlate what one sees - be it indirectly through imaging or directly during arthroscopy - with the underlying anatomy is of paramount importance. A sound understanding of the related anatomy allows for an adequate management plan when dealing with instability.

In this lecture we will focus on the anatomy and pathology of the structures that are involved in traumatic (anteroinferior) shoulder instability. The glenohumeral capsule is reinforced by capsular ligaments that form a fibrous skeleton, dynamically allowing a very broad range motion while at the same time affording static stability of the joint at the extremes of motion. The ligaments remain more or less lax as long as they are not fully deployed for the direction of motion for which a specific ligament acts as a checkrein. This results in a tendency for “instability” of the humeral head on the glenoid fossa in the lower and midranges of motion. Although the limited congruity of head and glenoid is increased by the glenoid labrum, these static stabilisers only partially counteract this tendency. Nevertheless, the glenohumeral joint does not normally dislocate easily as a complex interplay of ligaments, capsule and muscles afford dynamic stability. The rotator muscles are the mainstay in maintaining control. Optimising the efficiency of these muscles in association with the periscapular stabilisers forms the mainstay of rehab, be it for conservative management or postoperatively.

The term inferior GHL complex (IGHLC) includes an anterior band (ABIGHL), a posterior band (PBIGHL), and an axillary recess. The fasciculus obliquus (FO) contributes to the IGHLC as well. Viewed from anterior, the ABIGHL (deep) and the FO (superficial) cross each other. The MGHL additionally reinforces the anterior capsule, running from the glenoid labrum medially to the FO and subscapularis laterally. Histologically, fibers of all ligaments contribute to the periarticular system that continues around the glenoid labrum.

In most patients with recurrent dislocations or instability, an avulsion of the anteroinferior labrum (Bankart lesion) is present. It could be considered as the quintessential lesion. However, clinical experience and studies have shown that a substantial group of patients has associated or different pathology. The lesion of the capsuloligamentous complex can also occur on the humeral side (HAGL) or midsubstance, as elongation or even tears. Variants of the typical Bankart tear, such as an ALPSA-lesion or bony Bankart due to fracture or attrition, must be taken into consideration. Underestimating these variants or additional pathology may lead to failure of a surgical stabilization procedure.