A Case-Based Approach to Shoulder Pain

A Pocket Guide to Pathology, Diagnosis and Management

Jasmin Harounian · Grant Cooper · Joseph E. Herrera · Scott Curtis *Editors*

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Dedicated in memory of my father, Nathan, for always being a pillar of strength. Dad—I will always love you to the moon and back.

Jasmin Harounian

For Ana, Mila, Lara, Luka, Twinkle, and Lili

Grant Cooper

Foreword

In Greek mythology, the Titan Atlas had to carry the weight of the heavens on his shoulders, a testament to the immense strength and durability these remarkable anatomical structures possess. The shoulder joint and its complex relationship of tendons, muscles, ligaments, nerves, and its interaction with the cervical spine, chest, scapula, and torso altogether allow it to carry out several important upper extremity activities in day-to-day life. At the same time, it is often prone to injury and a common reason to see a clinician in the office.

The group of expert authors in this book seek to help demystify the unique anatomy of the shoulder, while comprehensively discussing how to best identify, evaluate, manage, prevent, and treat the diverse array of shoulder pathology that exists.

I am confident that the book you hold in your hands will enlighten both new and experienced physician readers alike and will help serve as a valuable guide and tool toward the goal to alleviate and free the burden of pain, dysfunction, and suffering of millions of patients with shoulder disorders.

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Richard G. Chang

Acknowledgments

I would like to thank all of the contributing authors and my coeditors for their time and effort in compiling this text. Thank you to my family, Shahla, Ben, and Karla, for your unconditional love and support throughout my ongoing journey in medicine.

Jasmin Harounian

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Grant Cooper

I would like to thank my amazing family, Sandra, Alex, Mikhayla, and Andrew, for all of their continued love and support through this journey. Thank you of course to my long-time friend, colleague, and coeditor on this and so many other projects, Dr. Grant Cooper. Thank you to all of our fantastic authors and other editors who came together to make this book a success.

Joseph E. Herrera

I would like to extend my sincere gratitude and appreciation to all the authors and coeditors for their dedication to this book. I would also like to thank my family for their endless love and support. This book is dedicated to my wife Amanda, our two children Lily and Ryan, and most importantly our cockapoo, Charlie.

Scott Curtis

Contents

Shoulder Anatomy

1

Caroline Varlotta and Monica Gibilisco

General Shoulder

The majority of the motion is from the surrounding bones and not through the joint itself. The clavicle rotates 40–50° posteriorly with shoulder elevation. There are eight degrees of rotation through the acromioclavicular joint. The remainder is from scapular rotation and sternoclavicular motion. Of note, the scapular plane is located 30° anterior to the coronal plane (Fig. 1.1).

A total of 180° of abduction comes from the two joints in a 2:1 ratio—120° from the glenohumeral joint and 60° from the scapulothoracic joint. Abduction requires external rotation to clear the greater tuberosity from impinging on the acromion. Therefore, if someone has an internal rotation contracture or spasticity, they may not be able to abduct past 120° [2–4].

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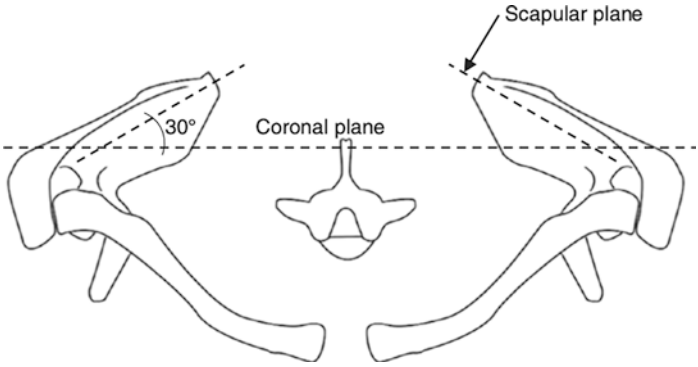

Fig. 1.1 Scapular plane in relation to the coronal plane [1]. Panayiotou Charalambous C. (2019) Shoulder Anatomy. In: The Shoulder Made Easy. Springer, Cham. https://doi.org/10.1007/978-3-319-98908-2_2

Bony Anatomy

The bones comprising the shoulder joint include the scapula, the clavicle, and the humerus. The scapula is the most posterior bone of the shoulder joint, located lateral to the vertebral column and on the posterior thorax. It is flat and triangular, with the glenoid projecting anteriorly. Other components of the scapula include the body, the scapular neck, and the coracoid process. The coracoid process projects anteriorly from the scapula. Medial to the coracoid process is the suprascapular notch, where the suprascapular neurovasculature passes (Fig. 1.2). From the posterior surface of the scapula emerges the scapular spine, which projects toward the lateral end of the clavicle and forms the acromion. The supraspinatus fossa lies superior to the scapular spine, while the infraspinatus fossa lies inferiorly. On the anterior aspect of the scapula is the subscapularis fossa (Fig. 1.3).

The humerus is the long bone of the upper arm with a proximal head that articulates with the glenoid at the glenohumeral

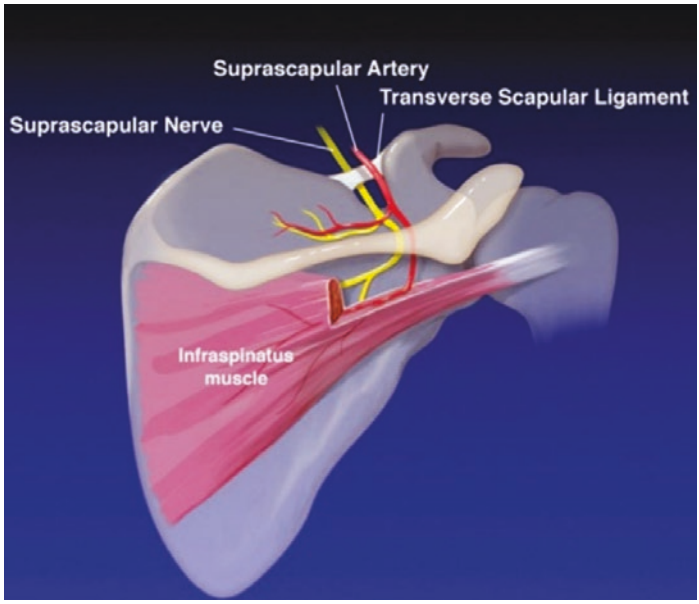

Fig. 1.2 Course of suprascapular neurovasculature through suprascapular notch [5]. Plancher et al. Suprascapular Nerve In: Normal and Pathological Anatomy of the Shoulder

joint. The medial surface of the articular head is covered by articular cartilage. The lesser and greater tuberosities are lateral to the articular cartilage. They are separated by the bicipital groove, through which the long head of the biceps runs. The humerus also has two junctions or "necks." The anatomical neck is the junction of the articular surface humeral head and the surrounding bone. The more inferior surgical neck is the junction of the head and shaft of the humerus. The clavicle is a long bone extending on the anterior trunk from the sternum medially to the shoulder joint laterally [1].

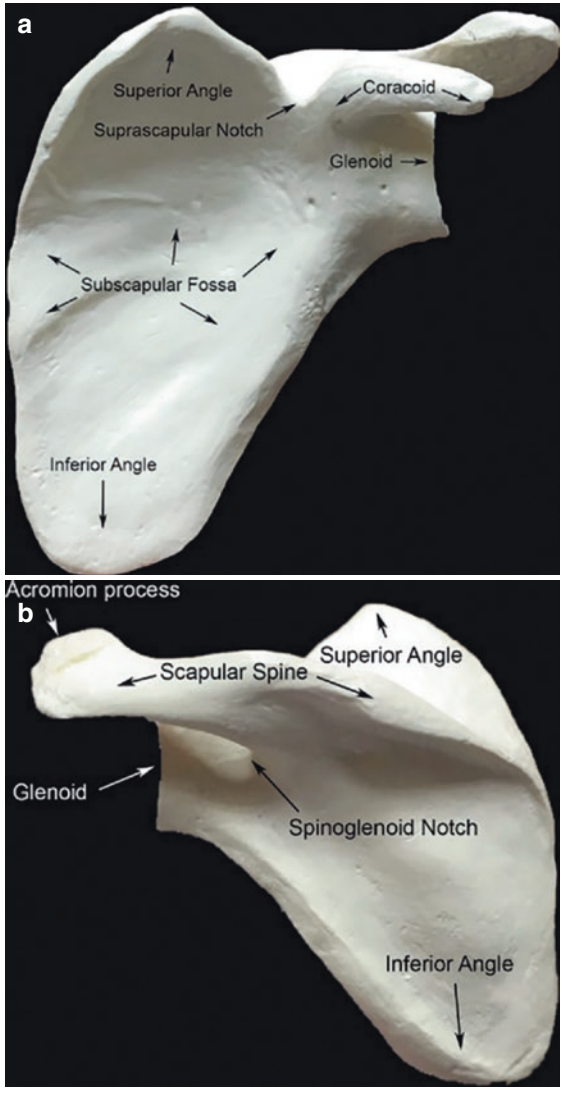

Fig. 1.3 Bony anatomy of the scapula. (**a**) Anterior view. (**b**) Posterior view [6]. Huri, G et al. (2017). Shoulder Anatomy. In: The Shoulder. Springer. https://doi-org.eresources.mssm.edu/10.1007/978-3-319-51,979-1

Joints

There are four smaller joints that comprise the shoulder joint complex: the glenohumeral (GH) joint, the acromioclavicular (AC) joint, the sternoclavicular (SC) joint, and the scapulothoracic (ST) joint [7].

Glenohumeral Joint

The GH joint is a "ball-and-socket" joint lined with hyaline cartilage, with the ball being the humeral head inside of the socket or the glenoid fossa. As only 25% of the humeral head articulates directly with the glenoid, the presence of the labrum, a ring of connective tissue surrounding the glenoid, allows for increased stabilization by providing more surface area. Surrounding the GH joint is a capsule lined with synovial membrane extending from the anatomic neck of the humerus to the border of the glenoid fossa. Synovial fluid inside the joint provides lubrication [2–4]. Superior to the GH joint and inferior to the acromion is a subacromial bursa to provide cushioning and facilitate motion.

Static restraints can be divided into GH ligaments, glenoid labrum, articular congruity and version, and negative intra-articular pressure. Most of the static stability of the shoulder is due to the GH ligaments. The GH ligaments can further be divided into anterior restraints, inferior restraints, and posterior restraints. The superior glenohumeral ligament (SGHL) is located from the anterosuperior labrum to the humerus. It will resist inferior translation when the arm is positioned in 0° of abduction or neutral rotation with the arm at the side. This prevents anteroinferior translation of the long head of the biceps. The middle glenohumeral ligament (MGHL) is located slightly inferior to the SGHL, stretching from the anterior labrum to the humerus. It resists anterior and posterior translation in the mid-range of abduction in external rotation and is considered an anterior and posterior restraint. The inferior glenohumeral ligament (IGHL) is a complex of anterior, superior, and posterior bands.

The posterior band of the IGHL is the most important restraint that prevents posterior subluxation when the arm is abducted. The anterior band is responsible for restraining anterior and inferior translation of the humeral head when the arm is abducted to 90 degrees with external rotation, such as in the late cocking phase of throwing. It also functions as an anchor to the anterior labrum. Bankart lesions are associated with the anterior band at the attachment along the anterior labrum, as this is the weakest band of the IGHL [9–14] (Fig. 1.4).

The coracohumeral ligaments are located between the coracoid and the humerus. They function to limit posterior translation when the shoulder is flexed and internally rotated and to resist inferior translation when the arm is adducted to neutral while externally rotated. The coracoacromial ligament is a triangular band of tissue attaching the coracoid process to the anterior acromion, preventing superior humeral head displacement from the GH joint [13, 15, 16].

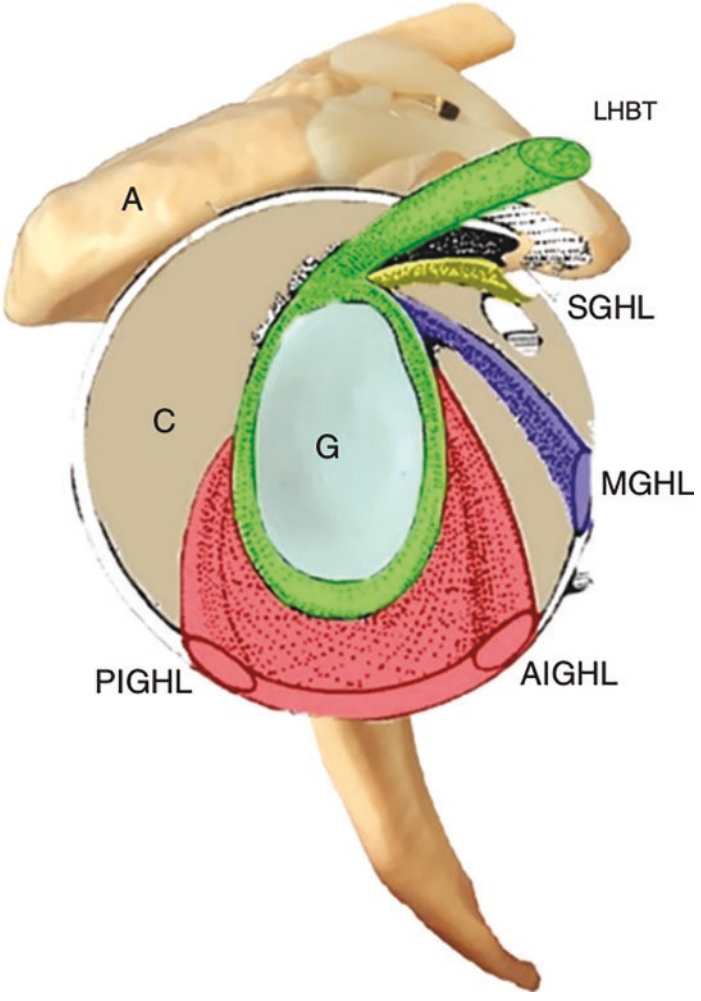

Fig. 1.4 Insertions around the glenoid. C: capsule. G: glenoid. A: Acromion. LHBT: long head of biceps tendon. SGHL: superior glenohumeral ligament. MGHL: middle glenohumeral ligament. AIGHL: anterior band of the inferior glenohumeral ligament. PIGHL: posterior band of the inferior glenohumeral ligament [8]. Rey, Alfonso Ricardo Barnechea. (2021). Anatomy and Kinematics of the Shoulder. In: Orthopedaedic Biomechanics in Sports Medicine. Springer. https://doi-org.eresources.mssm.edu/10.1007/978-3-030-81,549-3

Acromioclavicular Joint

The AC joint is a diarthrodial joint consisting of a fibrocartilaginous intra-articular disc located between osseous segments. The AC joint is surrounded by an independent joint capsule. The joint is limited to gliding motions only, and its primary function is to facilitate shoulder motion with the axial skeleton.

The AC ligament provides horizontal stability, while the coracoclavicular ligaments provide vertical stability. The AC ligament can be divided into superior, inferior, anterior, and posterior components. The superior is the strongest of these ligaments. The coracoclavicular ligaments consist of trapezoid and conoid ligaments. The trapezoid ligament inserts 2.5 cm from the end of the clavicle and functions as a stabilizer against horizontal and vertical loads. The stronger conoid ligament inserts 4.5 cm from the end of the clavicle in the posterior border.

In addition to these ligaments, the deltotrapezial fascia, the joint capsule, the deltoid muscle, and the trapezius muscle also act as stabilizers [7, 17–19].

Sternoclavicular Joint

The SC joint is a saddle joint with an independent joint capsule. There is approximately 50% articulation between the sternum and the clavicle. Because of the SC joint, the arm can be elevated without motion of the trunk or thorax [20, 21].

Scapulothoracic Joint

The ST joint is an articulation between the scapula and thorax or rib cage. There is no joint capsule, and thus this is not a true joint. Its main responsibility is to facilitate greater range of motion at the shoulder. When the ST joint elevates, depresses, protracts, retracts, or rotates, this provides the shoulder joint with a new plane of motion and greater flexibility. The ST joint is responsible for about one third of shoulder abduction [20, 22].

Muscular Anatomy

There are 17 muscular attachments along the shoulder [23, 24]. Key muscles of the shoulder motion include the rotator cuff muscle complex, deltoid, biceps brachii, teres major, coracobrachialis, pectoralis, and latissimus dorsi. The trapezius, triceps, rhomboids, serratus anterior, levator scapulae, and subclavius have a smaller role in shoulder motion and are considered supportive muscles (Fig. 1.5).

The rotator cuff muscles overly the shoulder capsule. The complex consists of four muscles – the supraspinatus, infraspinatus, subscapularis, and teres minor muscles. They originate from the scapula and insert onto the humerus. These muscles work in conjunction to maintain active shoulder range of motion while protecting the GH joint from extreme forces [25]. In addition, each muscle also has their own specific function.

The subscapularis is located most anteriorly and courses across the anterior aspect of the GH joint capsule from the subscapular fossa to the lesser tuberosity of the humerus. It is innervated by

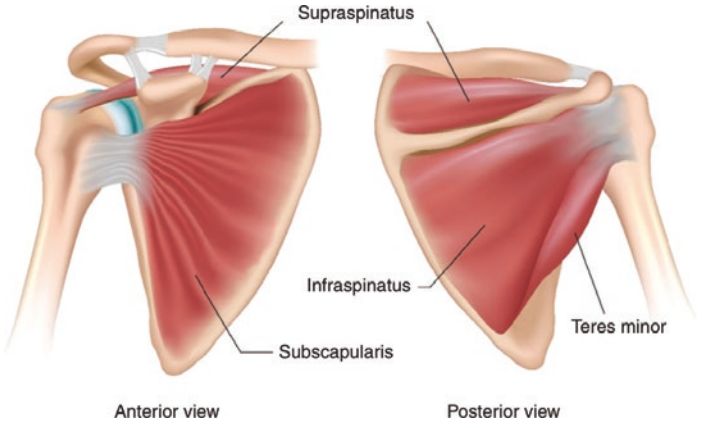

Fig. 1.5 Rotator cuff muscles [8]. Rey, Alfonso Ricardo Barnechea. (2021). Anatomy and Kinematics of the Shoulder. In: Orthopedaedic Biomechanics in Sports Medicine. Springer. https://doi-org.eresources.mssm.edu/10.1007/978-3-030-81,549-3

the upper and lower subscapular nerves. Due to its location, the subscapularis functions to provide the anteroinferior border of the rotator interval or the space between the rotator cuff tendons that acts as a pulley for the long head of the biceps brachii. Other borders of the rotator interval include the coracohumeral ligament, the SHGL, and the supraspinatus tendon. The subscapularis facilitates humerus adduction and internal rotation. It also functions to restrain anterior translation of the humeral head [26, 27].

Immediately posterior to the subscapularis muscle is the supraspinatus muscle. The supraspinatus originates on the superoposterior aspect of the scapular spine. The deltoid and the supraspinatus muscle together provide the ability for shoulder abduction, especially the first 15°. The infraspinatus muscle originates inferior to the spine of the scapula and is directly below the supraspinatus muscle. It contributes to external rotation of the arm, especially from neutral abduction. Both muscles are innervated by the suprascapular nerve. The most posterior muscle of the rotator cuff complex is the teres minor, which originates on the axillary border of the scapula. It is innervated by the axillary nerve and aids in external rotation of the arm when the arm is already abducted. The supraspinatus, infraspinatus, and teres minor muscles insert along the greater tuberosity of the humerus [27–29].

The most prominent superficial muscle of the shoulder is the deltoid which is innervated by the axillary nerve. Its main function is abduction of the arm. The deltoid can be divided into thirds. The posterior and middle deltoid originate on the scapula, while the anterior deltoid's proximal attachment is on the distal clavicle. The anterior and posterior segments assist with flexion and extension, respectively. The entire deltoid inserts onto the lateral humerus. The deltoid branch of the thoracoacromial artery and the posterior humeral circumflex artery provide blood supply to the deltoid muscle [7].

The biceps brachii has actions at the forearm, the elbow, and the shoulder. Its primary function is supination and flexion of the forearm and elbow, respectively. The biceps muscle also allows for arm flexion and adduction, as it crosses the shoulder joint. It is innervated by the musculocutaneous nerve. There are two heads of the biceps that insert onto the radius. The long head of the

biceps originates at the supraglenoid tubercle, crosses the rotator interval, and travels down the bicipital groove of the proximal humerus. The short head of the biceps forms a joint tendon with coracobrachialis at their origin on the coracoid. The coracobrachialis inserts onto the medial aspect of the humerus and allows for adduction and flexion of the arm. It is innervated by the musculocutaneous nerve [27, 30–33].

Also attaching to the coracoid is the pectoralis minor, which originates from the third, fourth, and fifth ribs, and is innervated by the medial pectoral nerve. The action of the pectoralis minor is depression of the shoulder by drawing the scapula inferiorly.

The pectoralis major is located ventrally relative to its minor counterpart. It is responsible for the chest contour in addition to flexion, adduction, and internal rotation of the arm. The pectoralis major is innervated by the medial and lateral pectoral nerves. The muscle originates from two heads—the sternal head at the level of the sixth and seventh rib and the clavicular head. The tendon inserts medial to the deltoid on the humeral shaft lateral to the bicipital groove [34, 35].

The latissimus dorsi originates from the mid back, courses across the inferior angle of the scapula, and inserts onto the humeral shaft between the pectoralis major and teres major. It is innervated by the thoracodorsal nerve and functions to extend, adduct, and internally rotate the arm [35, 36].

The teres major muscle originates on the inferolateral aspect of the scapula and inserts onto the medial aspect of the humeral shaft. It is innervated by the lower and middle subscapular nerves and functions to assist in humerus internal rotation and adduction. Identification of the teres major reveals an important anatomical area, the quadrilateral space. The quadrilateral space contains the posterior humeral circumflex vessels, the scapular circumflex vessels, and the axillary nerve. Its borders include the teres major inferiorly, the long head of the biceps medially, the subscapularis superiorly, and the surgical neck of the humerus laterally [37–39].

The trapezius originates at the upper spine and inserts onto the lateral scapula, the clavicle, and the acromion. It supports stabilization of the scapula and enables movement in the scapulothoracic plane. The serratus anterior originates from the first eight

ribs and inserts onto the scapula. It is innervated by the long thoracic nerve and functions to pull the inferior aspect of the scapula laterally and forward, allowing for shoulder abduction beyond 90 degrees in the ST plane. The rhomboid major and minor facilitate depression of the scapula and are antagonists to the serratus anterior muscle. These muscles originate from the spinous processes of the second to fifth thoracic vertebrae and insert onto the medial border of the scapula. The rhomboid muscles are innervated by the dorsal scapular nerve [34].

Although the triceps primarily functions as an extensor of the elbow, it proves minimal support for adduction and retroversion of the arm. It provides this assistance as it crosses the shoulder joint from its origin at the infraglenoid tubercle of the scapula to its insertion on the olecranon process [40]. The triceps is innervated by the radial nerve.

The levator scapulae enables elevation and medial rotation of the scapula in the scapular plane. It originates from the cervical spine and inserts onto the medial scapula. It is innervated by the dorsal scapular nerve. The subclavius muscle depresses the shoulder while pulling the clavicle inferiorly. It stretches from the first rib to the subclavian groove on the inferior surface of the middle one third of the clavicle and is innervated by the subclavian nerve [41].

Brachial Plexus

The brachial plexus originates from nerve roots C5-T1 and courses anteriorly and laterally toward the arm. It contains subdivisions of trunks, divisions, cords, and ultimately branches (Fig. 1.6). By understanding the brachial plexus, a careful clinical exam can localize the level of brachial plexus injury [43].

The terminal branches of the brachial plexus are the lateral pectoral nerve, the musculocutaneous nerve, the axillary nerve, the radial nerve, the median nerve, and the ulnar nerve. The lateral pectoral nerve provides motor innervation to the pectoralis major muscle. The musculocutaneous nerve supplies the elbow flexors and provides sensation to the radial (or lateral) side of the forearm

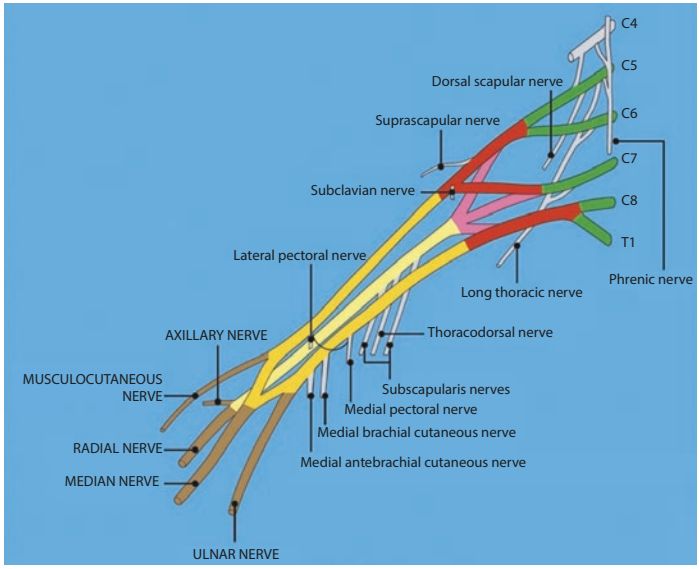

Fig. 1.6 Brachial plexus [42]. Shin A. et al. (2021). Surgical Anatomy of Brachial plexus Injuries. In: Operative Brachial Plexus Surgery. Springer. Pages 19–39. https://doi.org/10.1007/978-3-030-69,517-0

[44]. The radial nerve supplies all extensor muscles of the upper limb and sensation to the dorsum of the hand. The median nerve supplies all the forearm flexors except for the flexor carpi ulnaris, the ulnar half of the flexor digitorum profundus, muscles of the thenar eminence, and sensation to the radial three and a half digits of the hand. The ulnar nerve supplies the flexor carpi ulnaris, half of the flexor digitorum profundus, the small muscles of the hand excluding the thenar muscles, and sensation to the ulnar one and a half digits [1].

The axillary nerve arises from the posterior cord of the brachial plexus. It courses inferiorly along the border of the subscapularis, through the quadrilateral space, then around the surgical neck of the humerus, and anteriorly under the deltoid muscle. Anterior and posterior divisions are most commonly found in the quadrilateral space but can be located under the deltoid muscle. The ante-

rior branch of the axillary nerve usually courses adjacent to the posterior circumflex humeral artery. The posterior branch gives rise to the superior-lateral brachial cutaneous nerve [45].

Cutaneous sensation of the shoulder and upper limb can be described as a dermatome or a peripheral nerve sensory territory. A dermatome would refer to the area of the skin innervated by a single root. A peripheral nerve sensory territory is the area of the skin with contributions from multiple roots. The shoulder dermatomes are C4, C5, T2, and T3 (Fig. 1.7). The main peripheral nerves of the shoulder are the supraclavicular nerves and the posterior branch of the axillary nerve [1] (Fig. 1.8).

The suprascapular nerve arises from the brachial plexus. It courses deep to the trapezius muscle until it emerges through the suprascapular notch where it supplies the supraspinatus muscle (Fig. 1.2). The suprascapular artery will pass superficial to the transverse scapular ligament, while the suprascapular veins and nerve will pass deep to the ligament within the notch. The nerve will course under the spinoglenoid ligament to supply the infraspinatus muscle. In addition to innervating the supraspinatus and infraspinatus muscle, the suprascapular nerve will provide sensory branches to the GH joint, the coracohumeral and coracoacromial ligaments. The suprascapular nerve may also provide variable sensation to the posterior aspect of the shoulder [46–48].

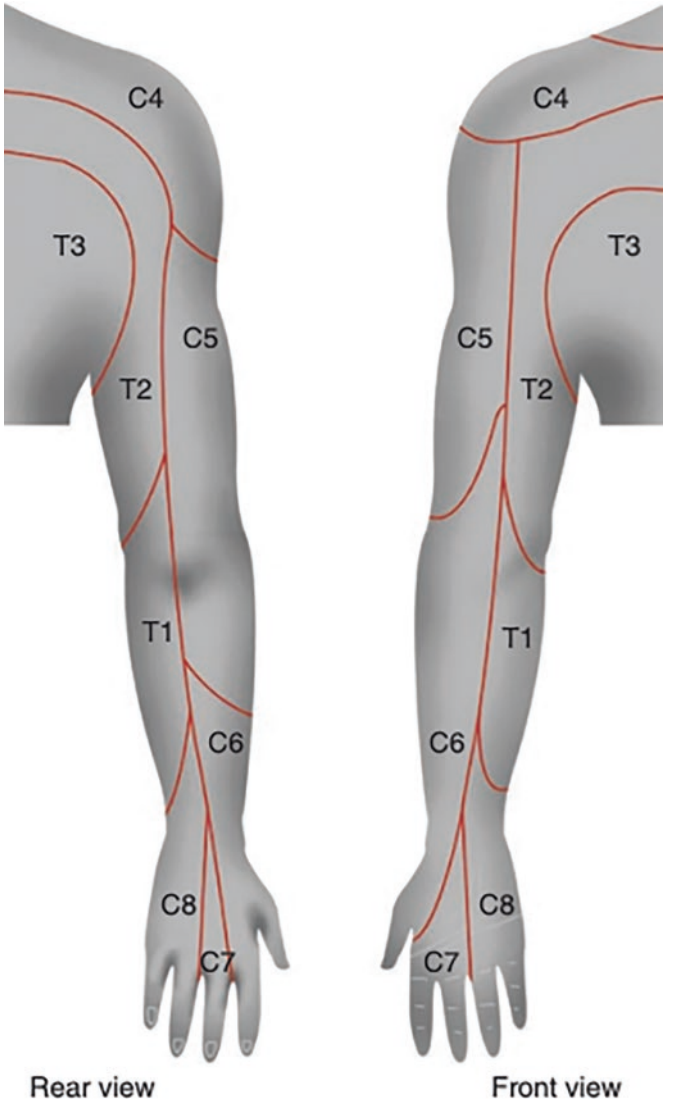

Fig. 1.7 Dermatomal sensory innervation of the upper limb [1]. Panayiotou Charalambous C. (2019) Shoulder Anatomy. In: The Shoulder Made Easy. Springer, Cham. https://doi.org/10.1007/978-3-319-98908-2_2

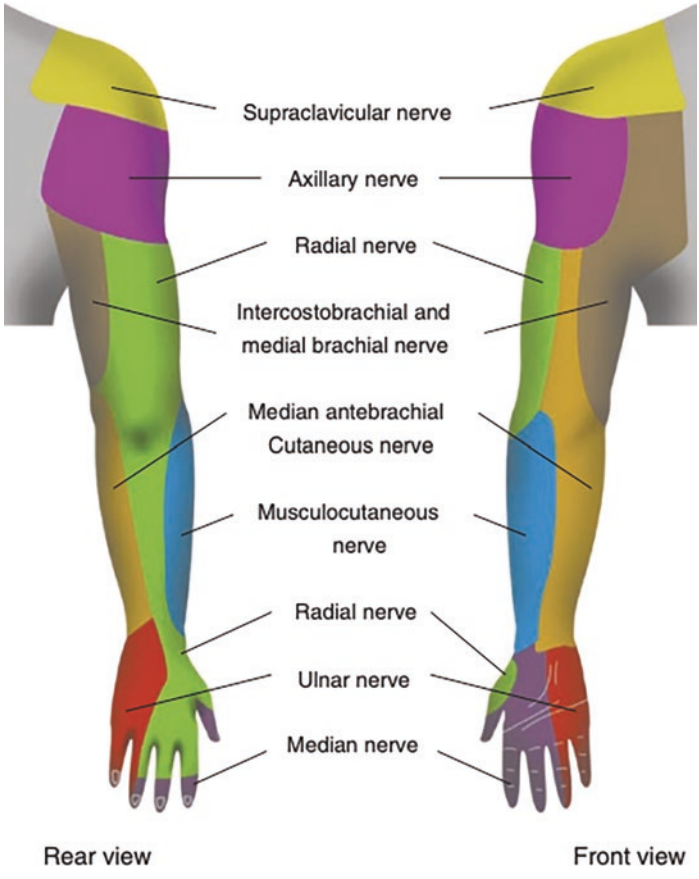

Fig. 1.8 Distribution of peripheral sensory nerves of the upper limb [1]. Panayiotou Charalambous C. (2019) Shoulder Anatomy. In: The Shoulder Made Easy. Springer, Cham. https://doi.org/10.1007/978-3-319-98908-2_2

Blood Supply

The main vascular supply of the shoulder structures is the axillary artery. Proximally, it travels anterior to the pectoralis minor and then dives posteriorly where the thoracoacromial trunk branches

off. The axillary artery courses lateral to the pectoralis minor, where it then gives rise to the subscapular artery and the humeral circumflex arteries (anterior and posterior). Four centimeters from its origin, the subscapular artery gives rise to the scapular circumflex artery and thoracodorsal artery [49, 50]. The scapular circumflex vessels travel through the triangular space to the posterior aspect of the shoulder, while the thoracodorsal artery supplies the latissimus dorsi. The posterior humeral circumflex artery, thought to be larger than the anterior counterpart, arises below the lower border of the subscapularis muscle. It then travels through the quadrangular space with the axillary nerve, supplying the inferior deltoid, shoulder capsule, and, most importantly, the humeral head. The posterior branch has recently been established as the majority vascular supply to the humeral head. The anterior humeral circumflex artery runs beneath the coracobrachialis muscle anteriorly and wraps around the anterior humeral neck. At the intertubercular sulcus, it gives rise to the arcuate artery, which is responsible for vascular supply to the humeral epiphysis. The two humeral circumflex vessels then anastomose at the lateral humerus [51–53].

Critical Shoulder Angle

The CSA is calculated by measuring a line from the inferior edge of the glenoid to the superior edge of the glenoid and a line from the inferior edge of the glenoid to the lateral edge of the acromion on a true anterior-posterior shoulder radiograph. Normal CSA is between 30 and 35°. Increased CSA is thought to increase superior shear forces on the rotator cuff muscles, increasing the load on these muscles and predisposing to rotator cuff tears. Decreased CSA may lead to abnormally elevated compressive forces, potentially leading to GH arthritis. Research has supported and refuted the association between the critical shoulder angle (CSA), shoulder disease, and clinical treatment outcomes. Therefore, more studies are needed to determine its true clinical significance [54].

Thoracic Outlet

The thoracic outlet is located from the cervical spine to the inferior border of the pectoralis minor muscle and includes essential neurovasculature for the upper limb (Fig. 1.9). Contents include the C5 to T1 nerve roots, the brachial plexus, the subclavian artery, and the subclavian vein. The thoracic outlet can be divided into three spaces—the interscalene triangle, the costoclavicular space, and the retropectoralis minor space. The interscalene triangle is located in the lateral part of the neck behind the sternocleidomastoid muscle. It is bounded by the anterior scalene muscle anteriorly, the middle scalene muscle posteriorly, and the first rib inferiorly. This is the most common site for compression in a true neurogenic thoracic outlet syndrome. The costoclavicular space is bound by the upper aspect of the scapula posterolaterally, the middle one third of the clavicle anteriorly, the subclavius muscle medially, and the site of the insertions

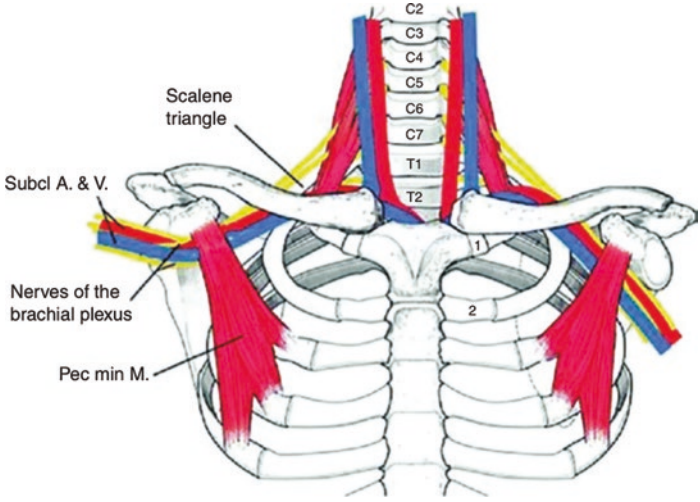

Fig. 1.9 Thoracic outlet [55]. Illig et al. (2021). Anatomy of the Thoracic Outlet and Related Structures In: Thoracic Outlet Syndrome, Springer. https://doi.org/10.1007/978-3-030-55,073-8

of the anterior and middle scalenes on the first rib posteromedially. The retropectoralis minor space is bound by the coracoid process superiorly, the pectoralis minor muscle anteriorly, and the rib cage posteriorly [56–58].

Normal Anatomic Structures that May Mimic Pathology

Individuals may have normal variants of shoulder anatomy that can mimic pathology on imaging. Variants are important to identify and integrate into the clinical presentation to avoid unnecessary workup.

Labral variants include sublabral recess, sublabral foramen, and the Buford complex. Sublabral recess, or sulcus, is a physiologic recess and represents a variation in the configuration of the biceps labral complex. On imaging, the sublabral sulcus will extend medially toward the glenoid, while a type II superior labrum anterior to posterior (SLAP) tear will extend laterally or posteriorly [14, 59]. The sublabral foramen is less common and provides a communication between the GH joint and the subscapularis recess [60]. It is commonly confused with an anterosuperior labral tear, which will propagate a greater distance. The Buford complex is a thickening of the MGHL with a cord-like appearance. It is associated with congenital absence of the anterosuperior labrum and may be mistaken for a displaced labral fragment. Sagittal oblique images will distinguish between the two.

One of the most common muscular variants is an accessory head of the biceps muscle, which can be mistaken for a longitudinal split tear of the long head of the biceps [61]. The rotator cable is a collection of fibers perpendicular to the axis of tendons that run from the intertubercular groove to the posterior aspect of the greater tubercle and should not be mistaken for a rotator cuff tear [14].

The anatomy of the shoulder is essential in the diagnosis and treatment of shoulder pain and disability. The following chapters will describe injuries to structures mentioned in this introduction.

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Rotator Cuff Tendonitis and Bursitis

2

Charles Kent and Eric Xu

Pathology

The rotator cuff is made up of four muscles—the supraspinatus, infraspinatus, subscapularis, and teres minor. Together, these rotator cuff muscles contribute significantly to the stability and mobility of the shoulder joint. A primary function of the rotator cuff is to stabilize the glenohumeral joint. In a healthy individual, the resting tension of the rotator cuff muscles compress the humeral head into the glenoid fossa, balancing it in both the coronal and the axial plane. During initiation of movement around the shoulder, electromyography (EMG) activity is seen in the rotator cuff muscles before other larger muscles, such as the pectoralis major and the deltoid [1]. This suggests that the rotator cuff muscles also help prepare the shoulder joint for motion. Individually, the rotator cuff muscles also allow the glenohumeral joint to move in shoulder abduction, internal rotation, and external rotation.

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A bursa is an enclosed sac of fluid designed to reduce friction and function as a cushion between tissues of the body. The subacromial bursa of the shoulder is surrounded by multiple structures including fibers from the supraspinatus and deltoid, the coracoacromial ligament, the acromion, and the coracoid. Subacromial bursitis is caused by inflammation of the bursa anywhere in this region. The most common cause of subacromial bursitis is overuse of the glenohumeral joint, but it can also be caused by impingement, trauma, infection, and autoimmune diseases. Subacromial bursitis is closely associated with rotator cuff tendinopathy. Due to the proximity of the subacromial bursa to the rotator cuff tendons, worsening bursitis decreases lubrication of the rotator cuff tendons, reducing their movement, and contributing to rotator cuff tendinopathy [2].

Injuries to the rotator cuff can range from acute contusions and tendonitis to chronic calcific tendinosis as well as partial- or full-thickness tears; however, the focus of this chapter is to explore rotator cuff tendonitis. Historically, "tendonitis" was a term that corresponded to a histopathological description of tendon impairment with intratendon inflammation. This would be clinically associated with pain located at the muscle tendon insertion with muscle activation or eccentric stretching. Over time, tendonitis has been replaced by the term "tendinopathy," which describes any overuse condition that causes pain around the tendon. Overall, tendinopathy occurs when injury to the tendon is followed by an impaired healing cascade leaving the tendon with histological disorganization and thickening. These changes can alter the physical properties of the tendon, which can then perpetuate the original insult and perpetuate a patient's clinical symptoms [3, 4].

Rotator cuff tendinopathy is a very common cause of shoulder pain and is often associated with subacromial impingement syndrome (SIS), the most common cause of shoulder pain. The most significant risk factor for rotator cuff tendinopathy is repetitive overhead motion; however, other risk factors include scapular instability, old age, and obesity [5]. Acute rotator cuff tendinitis can occur from direct trauma, a fall, participating in sports involv-

ing repetitive overhead motions, or through exacerbation of a chronically injured rotator cuff. Proposed mechanisms of tendinopathy can be organized into extrinsic and intrinsic mechanisms, although rotator cuff tendinopathy can commonly result from a combination of two or more of these [3].

Extrinsic Mechanisms

Extrinsic mechanisms causing rotator cuff tendinopathy are due to impingement of the tendon and can be related to a multitude of anatomical variables such as acromion shape, spurring of the acromion or acromioclavicular joint, and degenerative subacromial bursa [2]. Impingement stresses the tendon through direct repetitive forces, which causes inflammation in the acute setting and contributes to chronic changes of the tendon over time [4]. Impingement usually occurs due to changes in the anterior portion of the shoulder; however, it is proposed that changes in the posterior shoulder can contribute as well [3, 6].

Subacromial impingement, which is a narrowing of the subacromial space in the anterior shoulder, is the most common type of shoulder impingement. The acromion can differ in size and shape, varying from flat to curved or hooked. Furthermore, the acromioclavicular (AC) joint can affect the rotator cuff tendon as it degenerates and potentially develops osteophytes that increase the risk of impingement [3, 6].

In the posterior shoulder, glenohumeral movement can be limited by a tight posterior shoulder capsule. This, in turn, increases the risk for impingement in the anterior shoulder due to excessive anterior-superior humeral head translation in shoulder flexion [3]. Glenoid impingement can occur in the posterior superior shoulder and is due to repetitive overhead activity. These motions can compress the rotator cuff between the greater tuberosity and the glenoid rim and incidentally can also injure the superior and inferior labrums as well [1, 6].

Internal impingement, also known as “thrower’s shoulder,” can occur in young athletes with overhead activity, particularly throw-

ing athletes. This form of impingement occurs during the cocking phase of throwing where the shoulder is put into abduction, external rotation, and extension. This position can cause compression and impingement of the rotator cuff tendons between the humeral head and the posterior superior labrum [1, 6].

Intrinsic Mechanisms

Common intrinsic factors that can influence rotator cuff tendinopathy include age, gender, and the presence of either inherited or acquired systemic disease, such as Ehlers-Danlos syndrome, rheumatoid arthritis, or diabetes mellitus [7]. These factors may or may not be modifiable. Increasing age has been related to degeneration of the tendon over time in addition to decreased elasticity, fibrovascular proliferation, and development of calcifications [6].

Hypovascularity may also play a role in rotator cuff tendinopathy [3]. Vascular adaptation occurs secondary to age-related changes and repetitive forces to the tendon. Extrinsic mechanisms may lead to blood vessel damage and subsequent tendinopathy in the setting of ischemia [6].

Clinical Presentation

Rotator cuff tendinopathy presents as shoulder pain that can be accompanied by mild weakness. Patients with acute rotator cuff tendinitis typically have lateral shoulder pain, which is exacerbated by overhead activities and direct palpation. A common presentation is a patient who has pain while sleeping on their shoulder. These patients may have a history of fall, trauma, or overuse. Athletes in sports that involve repeated overhead activities, such as swimming, javelin throwing, baseball, and rugby, are also at greater risk.

Weakness may be present in rotator cuff tendonitis, and patients may have limitations to the strength of the rotator cuff muscles due to pain. Weakness is more likely to be present with

partial- or full-thickness tears of the rotator cuff. Atrophy is more commonly found in chronic etiologies, such as rotator cuff tendinosis. If weakness is present in the area, other possible etiologies of muscle weakness should be considered and ruled out (i.e., cervical radiculopathy, brachial plexopathy, or various myopathies.)

In subacromial bursitis, patients typically present with anterior or lateral shoulder pain. The pain is usually worse at night and can interfere with the ability to sleep comfortably. Impingement and subacromial bursitis are closely related due to the narrowed space between the humerus and the acromion, which can cause inflammation of the surrounding tendons and bursa.

Physical Exam [8]

A thorough physical exam of any joint should always include inspection, palpation, range of motion, muscle strength, and sensory and any additional special tests. It is important to remember that pain or other symptoms may be a result of pathology from the joint above or below the symptomatic joint. Therefore, for any shoulder complaints, an evaluation should also include the cervical spine, contralateral shoulder, elbow, trunk, and upper limb neurovascular structures [8]. A consistent approach to evaluation of the shoulder will help organize the physical exam and will provide useful information to help narrow the differential diagnosis.

The first step to evaluation is inspection. Both shoulders should be examined and compared with different angles. Any changes in posture, neck position, scarring, atrophy, swelling, ecchymosis, erythema, rashes, deformities, shoulder height, and scapular positioning should be noted. In rotator cuff tendinitis without a history of recent trauma, previous injuries, or chronic rotator cuff tendinopathy, the shoulders should appear symmetrical. For any rotator cuff tendinitis with traumatic etiologies, there may be skin changes, mild deformity, and swelling around the shoulder. In chronic rotator cuff tendinopathy, there may be mild atrophy present in the affected shoulder.

Palpation of the shoulder should take notice of all superficial structures including the sternoclavicular joint, clavicle, AC joint,

subacromial bursa, bicipital groove, coracoid process, supraclavicular fossa, and spine of the scapula. In rotator cuff tendinitis, the shoulder will be painful to palpation. Classically, rotator cuff tendonitis correlates with anterolateral tenderness to palpation below the lateral margin of the acromion.

Range of motion testing should be done both actively and passively. Active range of motion promotes patient comfort as patients will avoid painful positions, while passive range of motion can be performed to isolate particular joint motions. The rotator cuff muscles are particularly responsible for shoulder abduction, as well as glenohumeral internal and external rotation. Normal shoulder abduction active range of motion is between 0 and 180 degrees. Normal internal rotation is 0–70 degrees, and external rotation is 0–90 degrees. Patients with rotator cuff tendinitis will have a full range of motion in active movements; however, particular movements in abduction, external rotation, and internal rotation may be painful.

Special tests to isolate for rotator cuff pathology and for signs of shoulder impingement are best done in combination with one another as individual tests alone lack the sensitivity and specificity to consistently diagnose pathologies. There are a variety of tests that can be used to isolate the individual muscles of the rotator cuff, which can be helpful if rotator cuff tendinitis is suspected. In general, the special tests will be positive if they reproduce pain or weakness. In rotator cuff tendinitis, the patient will have pain with movement but should be able to fully resist the force applied, demonstrating close to full muscle strength. Contrastingly in rotator cuff tears, weakness in the shoulder may be appreciated more regularly.

Empty Can Test

The empty can test specifically evaluates the supraspinatus muscle. To perform this test, abduct the shoulder to 90 degrees in the scapular plane—the scapular plane is approximately 45 degrees between the planes of shoulder forward flexion and lateral extension—with the elbow extended and the arm pronated, pointing the

thumb down. The practitioner will then apply downward force on the patient's arm, while the patient actively opposes the force. A positive test will elicit either pain or weakness. For detecting supraspinatus tendonitis, this test has a sensitivity of ~77% and a specificity of ~38% [8].

Full Can Test

The full can test can test the supraspinatus muscle in a similar fashion as the empty can test, except with the thumb pointing up. While this test can be used for tendinitis, it has been studied to a larger extent for evaluation of supraspinatus tears. Again, the practitioner will apply downward force on the arm and ask the patient to push against the force. A positive test will elicit either pain or weakness.

Drop Arm Test

The drop arm test is another test that can be used for evaluation of rotator cuff tendinopathy, most commonly for rotator cuff tears around the supraspinatus. In this test, the practitioner passively abducts the arm to 90 degrees. The patient is then asked to slowly lower their arms in the same arc. A positive test occurs when the patient is unable to slowly return their arm to their side or the test elicits significant shoulder pain.

Patte Test

The Patte test is helpful for diagnosing rotator cuff tendinopathy in either the infraspinatus or the teres minor. In this test, the practitioner elevates and supports the patient's arm anteriorly in the scapular plane with the elbow flexed to 90 degrees. The patient will externally rotate the shoulder against resistance. A positive test will elicit pain or weakness in the area. For detecting infraspi-

natus tendonitis, this test has a sensitivity of ~57% and a specificity of ~70% [8].

Lift-off Test

The lift-off test assesses the muscle strength of the subscapularis. In this test, the patient internally rotates their arm to place the dorsum of their hand on the small of their back. The patient is asked to "lift off" their hand away from their back. A positive test will elicit pain or weakness in the area.

Bear Hug Test

The bear hug test also assesses the integrity of the subscapularis. The patient is asked to use the arm with the affected shoulder to grab the opposite shoulder across their own body. The practitioner will attempt to remove the patient's hand from the shoulder, and the patient should resist this force. A positive test is when the patient cannot keep their hand on their opposite shoulder.

Neer Sign

The Neer sign tests for impingement of the shoulder. The test is performed with the practitioner passively forward flexing the arm while stabilizing the scapula posteriorly. A Neer sign is positive if it elicits pain in the deltoid or the anterior shoulder. Typically, the pain will be produced between 90 and 120 degrees of forward flexion.

Hawkins Test

The Hawkins test can assess for signs of subacromial bursitis or rotator cuff pathology. The test is performed by forward flexing

the arm with the elbow in 90 degrees of flexion. Internal rotation of the arm will produce pain in the shoulder or deltoid area.

The Painful Arc

The Painful arc alone cannot make a diagnosis of rotator cuff tendinopathy, but it can be useful in combination with other tests. This test can also be used for assessing subacromial bursitis. Patients with rotator cuff tendinopathy can have pain in active abduction between 70 and 120 degrees of the arc. Pain will often be in the deltoid or the anterior shoulder region.

Yocum's Test

Yocum's test assesses for shoulder impingement. Using the affected arm, the patient places their hand on the opposite shoulder. The practitioner applies a downward force to the elbow, and the patient is asked to resist the force. A positive test will elicit pain or weakness in the area.

Patients with bursitis will have point tenderness on the anterolateral portion of the shoulder just below the acromion. The pain typically does not radiate and is exacerbated with shoulder abduction past 75 degrees. Patients may have a positive Neer or Hawkins test. In order to differentiate between rotator cuff tendonitis, bursitis, and rotator cuff tears, muscle strength must be tested as it is usually normal in tendonitis and bursitis.

Diagnostic Studies

Rotator cuff tendinitis and bursitis are considered a clinical diagnosis; however, imaging can be used to further evaluate the extent of any other rotator cuff tendinopathy. Baseline imaging is recommended for any acute or chronic shoulder pain.

Radiographs (X-Ray)

Radiographs to fully evaluate the shoulder should include internal and external rotation, anteroposterior, and supraspinatus outlet views. Anteroposterior images of the glenohumeral joint are taken with the patient slightly rotated from the coronal plane and can be helpful in evaluating the space between acromion and humeral head. In rotator cuff arthropathy, the space is reduced due to compromise of the rotator cuff muscles that serve to depress the humeral head [9]. The supraspinatus outlet view is able to evaluate the morphology and angle of the acromion, as variations in size and shape can be responsible for subacromial impingement and rotator cuff tendinopathy.

Radiographs are also helpful in identifying osteophyte complexes and other degenerative changes, particularly on the acromion, the acromioclavicular joint, or the humeral head. Furthermore, calcifications of the rotator cuff tendons and the coracoacromial ligament can be identified as well.

Ultrasound (US)

Ultrasound is excellent at visualizing soft tissue pathology and allows for a relatively quick look that can supplement the physical exam. Ultrasound technology is cost-effective and can be done quickly in the office setting. This modality is particularly good at picking up other etiologies of rotator cuff tendinopathy, such as partial- and full-thickness tears. Even though ultrasound is an excellent tool to evaluate the soft tissues, it is highly operator-dependent. Sensitivity and specificity of identifying rotator cuff tears can both exceed 90% with well-trained operators.

Magnetic Resonance Imaging (MRI)

MRI can be very helpful in diagnosing rotator cuff pathology, particularly in identifying potential rotator cuff tears. Furthermore, it provides excellent evaluation of the glenoid labrum, subcortical

bone, and deep soft tissues when compared to other modalities, which can be helpful in narrowing a diagnosis. It also can be helpful in identifying potential sites of subacromial impingement. MRI can directly evaluate the bursa, where findings of increased bursal thickness and fluid surrounding the bursa or AC joint may indicate bursitis.

Treatment

Treatment of rotator cuff tendonitis and bursitis should always be centered on the pathology and severity of the patient's symptoms. Conservative treatments have been shown to be very effective tools in successfully treating patients, particularly when strategically combined. They include activity modification, physical therapy, medications (both anti-inflammatory medication and local injections), and alternative modalities such as ultrasound and electrical stimulation.

Physical therapy should focus on working toward painless range of motion and maximization of strength. Rotator cuff stretching and strengthening can help correct any contributing muscle imbalances. Range of motion exercises should be utilized to prevent stiffness over time. Physical therapy sessions should also include patient education with a home exercise program that the patient can consistently do on their own.

Medications can be helpful in reducing pain and increasing activity. Nonsteroidal anti-inflammatory medications are typically the first-line pain medications used. Alternatively, corticosteroid injections can be utilized if there is minimal relief with other pain medications; however, intra-articular steroid injections should be limited due to potential negative long-term effects on joint cartilage. Platelet-rich plasma can be also used and may be successful in patients with chronic rotator cuff tendinopathy.

Surgery should be considered in special populations. Patients with full-thickness tears should be referred to an orthopedic surgeon. Surgical referral should be made within a few months to maximize results. Partial-thickness tears can undergo a trial of conservative therapy; however, progression should be based on a

patient's symptoms and clinical progress. Surgery can be considered if progress stagnates or gets worse with conservative therapy. Chronic rotator cuff tears should be managed conservatively as there tends to be less healing potential.

Return to Activities

Patients with rotator cuff tendinopathy should be gradually returned to play after successfully achieving full ROM and return of strength in the shoulder.

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Biceps Tendonitis

3

Jonathan Lee and Carley Trentman

Pathology

Biceps tendonitis is defined as tenosynovitis of the long head of the biceps tendon. Biceps pathology has a wide range from acute tendonitis all the way to degenerative tendinopathy. The origin of the long head is the supraglenoid tubercle and superior labrum. From the origin, the tendon courses laterally into the intertubercular groove of the humerus and then courses down to the biceps muscle. The tendon is housed in the intertubercular or bicipital groove and is stabilized by the coracohumeral ligament, superior glenohumeral ligament, and rotator cuff muscles. These surrounding structures create a pulley-like course. The proximal portion of the tendon is at higher risk of abrasive wear and tear and injury. Overuse, anterior shoulder trauma, and concomitant rotator cuff injury can all contribute to biceps tendinopathy.

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What Is it?

The primary function of the biceps is elbow flexion and forearm supination. When the arm is externally rotated, it can also have a role in shoulder abduction.

Primary biceps tendonitis is less common compared to it being a secondary finding in shoulder pathology due to its close approximation with other structures around the shoulder joint. The biceps tendon sheath is associated with the glenohumeral synovium and, therefore, is found to be commonly affected by inflammation caused by shoulder impingement syndrome, rotator cuff pathology, superior labrum anterior-posterior (SLAP) lesion, osteoarthritis, subacromial bursitis, and acromioclavicular joint pathology [1]. A systematic review with a sample size of 599 within 5 included studies showed the percentage of associated lesions of long head of biceps tendon and supraspinatus tendon between 22% and 78.5% [2].

The innervation to the biceps tendon has not been classified; however, preliminary studies have shown that there is a network of sensory and sympathetic nerves that supply the tendon. These nerves disproportionally innervate the proximal part of the tendon toward the origin as well. It has also been proposed that substance P and calcitonin gene-related peptide are involved as neuromodulators for not only inflammation and pain but also tendon healing and regrowth [3].

The biceps tendon has blood supply from the anterior humeral circumflex artery. Cadaveric studies have proposed that the undersurface of the tendon within the bicipital groove of the humerus is a critical area of avascularity, which may contribute to increased risk of injury [4].

Clinical Presentation

Biceps tendonitis often can be preliminarily diagnosed with thorough history and physical exam. Patients with this condition will typically present with anterior shoulder pain which is exacerbated with shoulder flexion and overhead activity. The course is typi-

cally a progressive pain that is atraumatic in nature. Some patients may have radiating pain from the shoulder down the front of the arm. In addition, it is helpful to inquire about any patient history involving sports (baseball, volleyball, or other overhead activities) and physical labor in occupation.

As mentioned earlier, it is important to keep a broad differential diagnosis when suspecting biceps tendonitis given its common presentation as a secondary injury. For example, trauma and instability are typically not a primary cause of biceps pathology; however, trauma causing a SLAP tear with shoulder instability could result in biceps tendonitis. In addition, the practitioner should inquire if the patient has any associated neck pain as cervical radiculopathy should be ruled out.

There should be a high clinical suspicion for biceps tendonitis if the nature of the injury is traumatic and includes pain with abduction. The pain can be dull at rest, sharp with exacerbation with shoulder flexion, and typically not associated with neck pain.

Physical Exam

Due to a wide differential of shoulder pathologies, physical exam alone is difficult to diagnose biceps tendonitis in anterior shoulder pain. Typically, biceps tendonitis will have point tenderness at the anterior shoulder in the bicipital groove where the shoulder lies. It is important to find out which direction of shoulder movement exacerbates the pain and what range of motion the patient can do actively and passively. Establishing a baseline for range of motion evaluation is a good way to follow the progress of the patient as they undergo treatment. The unaffected shoulder should also be tested for comparison. On inspection, the gross deformity of budging of the biceps muscle is called a “Popeye sign” which is a finding in proximal biceps tendon rupture.

The special tests for biceps tendonitis are Yergason’s test and Speed’s test. Yergason’s test is when the patient’s shoulder is placed at their side, the elbow is flexed to 90 degrees, and the patient is instructed to supinate their forearm while resisted by the physician. Speed’s test is when the patient supinates their fore-

arm, extends their arm, and flexes the shoulder against resistance. Pain in the bicipital groove or radiating down the anterior arm is considered a positive test for biceps tendonitis. However, each test is not reliable if performed alone. In a study of 125 patients with ultrasound testing for confirmation, the sensitivity and specificity of Yergason's test were 32% and 78% and of Speed's test were 63% and 58%. The study did also show that combined Yergason's test and bicipital groove tenderness did show higher specificity (over 70%) compared to single tests [5].

Other special tests conducted were the empty can test which tests for rotator cuff injury and the Hook's test which tests for the integrity of the distal biceps tendon in the antecubital fossa. The Hook's test involves placing the patient's shoulder into abduction to 90 degrees, with flexion of the elbow and supination of the forearm. The examiner then tries to "hook" the distal biceps tendon. A positive test would be if the examiner is unable to palpate and hook the tendon, indicative of a distal biceps tendon tear.

Diagnostic Studies

Plain radiograph films are helpful for observing arthritic changes within the acromioclavicular and glenohumeral joint but are not diagnostic or revealing for biceps tendon pathology. The modalities for diagnosing biceps tendonitis include high-quality ultrasound, MRI, and magnetic resonance arthrography (MRA). MRI without contrast is often ordered; however, studies have shown that compared to the arthroscopic exam (the gold standard of biceps tendinopathy diagnosis), MRI is a poor imaging modality for ruling out tendinopathy. A study with 66 participants comparing non-contrast MRI to arthroscopic exam showed that the MRI had a sensitivity of 27.7% and specificity of 84.2% for partial long head biceps tears. In addition, although a non-contrast MRI can be helpful for surgical planning in shoulder pathology, a surgeon may be unaware of the biceps tendon pathology until directly visualized in surgery [6].

The superior imaging modality is MRA. MRA involves injection of contrast within the joint capsule, which helps to better visualize structures within the joint. A study with 42 patients with

confirmed surgical diagnosis of long head biceps pathology had MRAs reviewed by two separate radiologists and found overall high sensitivity and specificity for detecting abnormalities (tendinopathy and rupture). The reported sensitivity and specificity for observer 1 was 92% and 56% and observer 2 was 89% and 81% [7].

Ultrasound is a cost-effective imaging modality that can be quickly completed by an experienced musculoskeletal operator. Ultrasound is best utilized to visualize the extra-articular portion of the tendon, as it cannot visualize the tendon within the joint. The ultrasound is conducted to show the biceps tendon sitting within the bicipital groove (Figs. 3.1 and 3.2). In ultrasound diag-

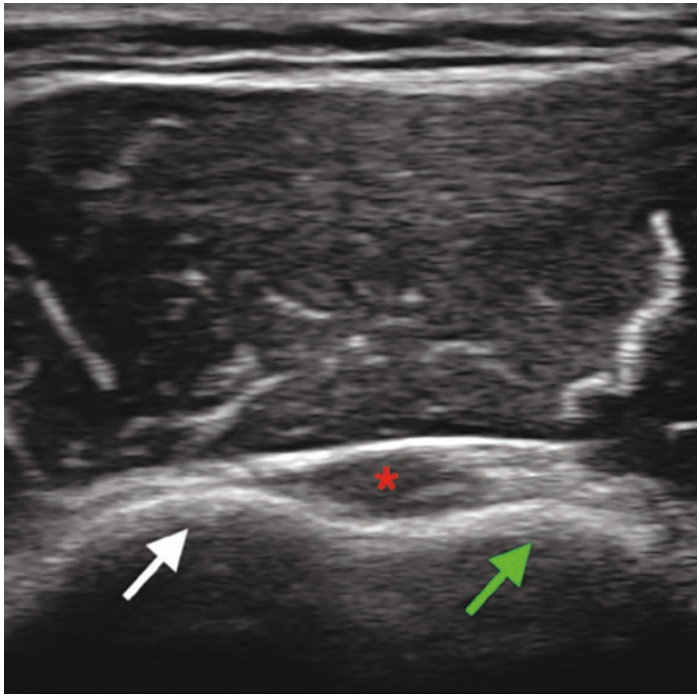

Fig. 3.1 Short-axis ultrasound image showing a normal biceps tendon (red *) sitting within the bicipital groove, which lies between the greater (green arrow) and the lesser tubercle (white arrow) of the humerus

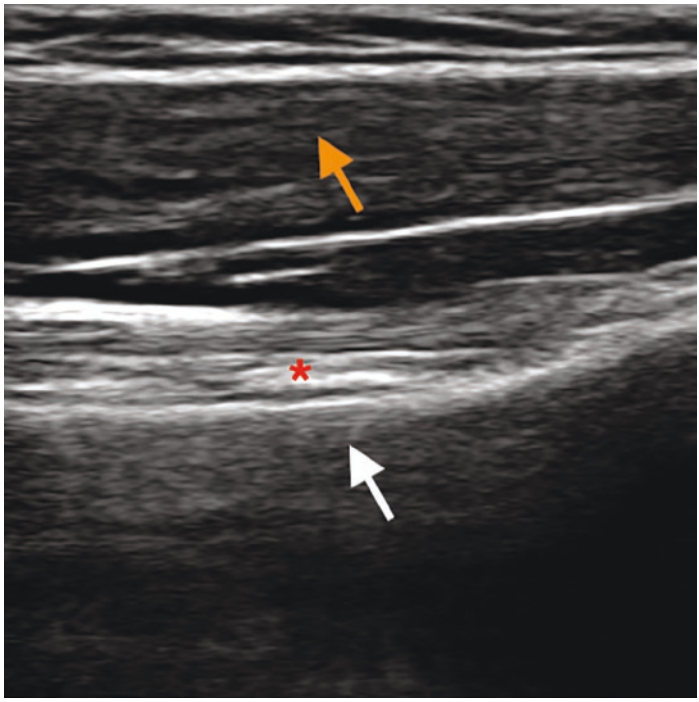

Fig. 3.2 Long-axis ultrasound image showing a normal biceps tendon (red *) running along the humerus (white arrow) and underneath the deltoid muscle (orange arrow)

nosis of tendinopathy, the structure will appear abnormally hypoechoic, and possibly partially thickened, and may have an effusion around the tent (Fig. 3.3). Doppler can be used to visualize active inflammation in the tendon sheath. Moderate tenosynovitis can be visualized as hyperechoic tissue around the tendon [8]. In a study comparing ultrasound to arthroscopy showed that ultrasound detected nearly all cases of tendon subluxation, dislocation, and complete ruptures but was poor at detecting partial ruptures [9]. Regarding tendinopathy specifically, ultrasound had a low sensitivity (0.22–1.00) and a high specificity

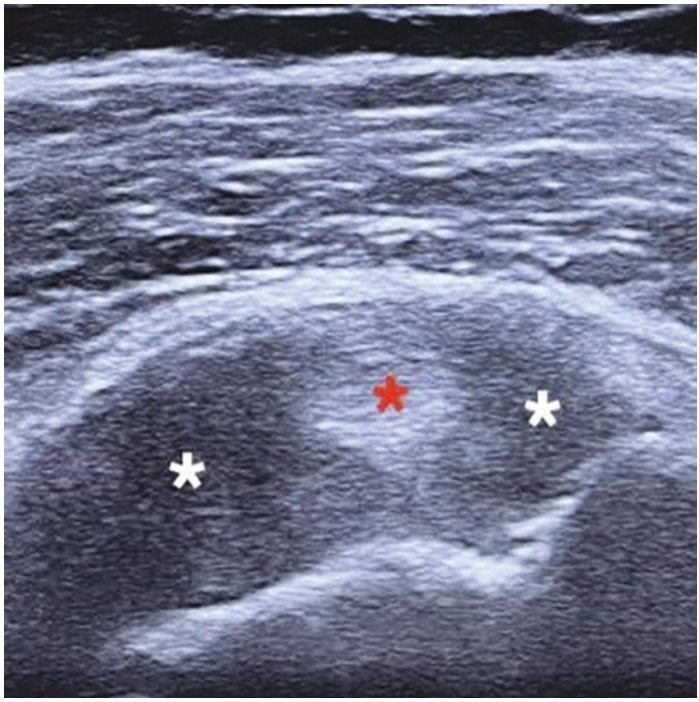

Fig. 3.3 Short-axis ultrasound image showing a large effusion (white *) around a biceps tendon (red *)

(0.88–1.00), which suggests that ultrasound may be a poor screening tool but a useful diagnostic tool [10].

In a nontraumatic presentation, without signs of contributing shoulder pathology, there typically is enough evidence clinically to diagnose biceps tendonitis from history and physical exam alone. If conservative treatment fails, to further diagnose or plan for injections, ultrasound can be considered after plain radiologic films to look for tears or effusion. MRI would be useful to determine the extent of his injury for possible surgical considerations and planning.

Treatment

Treatment is categorized as nonsurgical or surgical management. Once biceps tendonitis is clinically suspected or diagnosed with imaging, and conservative treatment should be started in the acute phase. This includes rest, specifically avoiding any exacerbating sports or activities, and nonsteroidal anti-inflammatory medications. Ice can be helpful to reduce inflammation. If the patient has improvement in pain with anti-inflammatory measures and rest, physical therapy should then be initiated to assist with ROM, strengthening of shoulder rotator cuff and girdle muscles surrounding the scapula.

If the pain severity persists despite oral medications, rest, or after 6–8 weeks of physical therapy, glenohumeral intra-articular or biceps tendon sheath injection can be administered [11]. Typically, a mixture of lidocaine and steroid is prepared as the lidocaine will provide an immediate pain-relieving component while the steroid will provide longer-lasting anti-inflammatory effects. Since the biceps tendon synovium is associated with the glenohumeral joint, an injection into the glenohumeral joint space can provide relief at the proximal biceps tendon (Nho 2010). Regarding accuracy, ultrasound-guided injections have been shown to have much higher successful injection rates (87%) compared to blind injections (27%) [4]. Injection of the biceps tendon involves the patient being supine with their arm externally rotated and hand supinated. The US transducer is placed perpendicular to the biceps tendon as it courses through the bicipital groove. The approach is then usually done in place from lateral to medial so that the needle can be visualized. Ideally, the best position for the needle is beneath the tendon to avoid injecting the subdeltoid bursa [12].

If tendonitis pain continues or worsens despite 6–8 weeks of conservative measures and steroid injections, the patient should be referred to orthopedics for surgical evaluation. Patients should also be referred for surgical evaluation if there are additional shoulder injuries, including tendon dislocation or subluxation, rotator cuff tears, or labral tears.

This chapter will not go into great depth of the surgical procedures offered; however, these options include biceps tenodesis and biceps tenotomy, which are considered to be simple and safe procedures, with high rates of patient satisfaction. Tenodesis involves cutting of the biceps tendon and reattaching it to the bicipital groove or transverse humeral ligament with sutures or screws. A tenotomy involves cutting the biceps tendon without reattaching it, allowing it to retract out of the shoulder joint [1, 13–15].

Return to Activities

Return to activity following a diagnosis of biceps tendonitis depends on a variety of factors. It is useful to consider whether the patient has undergone operative management or not. For a patient who does not undergo operative treatment, the patient is advised to return to the prior level of functional activity when s/he has full ROM and no pain or tenderness with a satisfactory clinical examination. Maintaining and continuing to improve upper extremity and core strengthening, flexibility, and neuromuscular drills are encouraged [16].

If the patient is an overhead athlete, another option for maintaining shoulder strength and flexibility is initiation of the Thrower's Ten program. This is a ten-step program that exercises all the major muscles necessary for throwing [16]. This includes the use of dumbbells, a chair, a table for support, a light resistance band tube, and a towel. There are various exercises performed including external and internal rotation at the waist and shoulders, abduction at the shoulder, press-ups, push-ups, and other exercises to strengthen and improve the overall power and endurance of the muscles surrounding the shoulder.

For patients undergoing operative management such as distal biceps repair, the average return to work timeline is just beyond 14 weeks. Averaged across all types of distal biceps repairs, a recent systematic review found that 89% of patients fully returned to work. This can be determined by a variety of meth-

ods by the clinician, such as achieving a passing score on the ASES (American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form) and SANE (Single Assessment Numeric Evaluation). The ASES assesses a patient's pain levels during a variety of tasks, and the SANE evaluates the patient's perception of his or her extremity functionality compared to pre-injury status [17].

Conclusion

Biceps tendonitis is overall a tenosynovitis of the shoulder that is observed less often as a primary injury but can commonly occur secondary injury with other shoulder pathology. History and physical exam alone can diagnose this condition, and MRA is the superior imaging choice, while ultrasound is the most cost-effective. Treatment includes conservative measures such as rest in the acute phase, anti-inflammatories, and ice. Physical therapy can be initiated following the acute phase, and if pain persists, a mixture of lidocaine and steroids can be administered. Orthopedic surgery referral can be recommended if these above interventions fail. Patients can generally return to activity when their pain has resolved, strength has been regained, and range of motion has improved.

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Labral Tears of the Shoulder

4

Elena Nehrbass

This chapter describes the normal anatomy and function of the labrum, pathologic conditions, clinical presentation, exam findings, diagnostic studies, treatment options, and return-to-activities protocols.

Normal Labral Anatomy and Function

The labrum is the fibrocartilaginous fold that lines the edge of the glenoid. The inner surface of the labrum is covered with synovium, and the outer surface attaches to the joint's capsule [1, 2].

The superior and anterosuperior portions of the labrum are loosely attached to the glenoid, and the macroanatomy of those portions is similar to that of the meniscus of the knee. The superior portion of the labrum inserts directly into the biceps tendon, while its inferior portion is firmly attached to the glenoid rim and appears as a fibrous, immobile extension of the articular cartilage.

The vascular supply of the periphery of the glenoid labrum comes from the suprascapular, circumflex scapular, and posterior circumflex humeral arteries. In general, the superior and anterosu-

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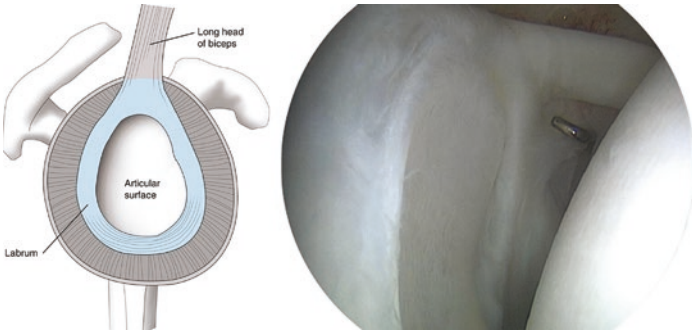

Fig. 4.1 Pictorial drawing (left) with face view of the glenoid, intact labrum, and long head of biceps insertion. Arthroscopic view (right) of the normal glenoid labrum [3]

perior parts of the labrum have less vascularity than the posterosuperior and inferior parts, and the vascularity is limited to the periphery of the labrum (Fig. 4.1). Vessels supplying the labrum originate from either capsular or periosteal vessels and not from the underlying bone [4].

The labrum has several functions which include increasing the depth of the glenoid, increasing the articulating surface of the glenoid, and providing attachment for the glenohumeral ligaments as well as attachment to the long head of the biceps tendon [3].

Lesions of the glenoid labrum can cause symptoms of shoulder instability through both anatomical and functional means. Anatomic instability occurs by allowing the shoulder to dislocate or subluxate recurrently, while functional instability occurs by allowing the shoulder to click, catch, and lock secondary to partially attached fragments becoming interposed between the articular surfaces [5].

Etiologies of labral tears include trauma, such as fall on the outstretched arm that forces the humeral head upward against the superior labrum, arm traction injury, direct force on the abducted shoulder, and chronic degeneration as seen with repetitive loading, overhead work-related activities, and throwing sports.

Types of Labral Tears

Labral tears can typically be subcategorized into the predominant location of pathology as delineated below:

- Superior labrum:
  - SLAP lesion
- Anteroinferior labrum:
  - Bankart lesion
  - Perthes lesion
  - GLAD
  - ALPSA
- Posteroinferior labrum:
  - reverse Bankart lesion
  - posterior GLAD
  - POLPSA
  - Kim lesion
- Circumferential labral lesion

Superior Labral Tears

SLAP tears occur when the superior labrum and its associated long head of the biceps (LHB) tendon insertion detach from the glenoid. Such lesions of the superior part of the labrum may extend anterior and posterior to the biceps tendon insertion, hence their designation as SLAP (superior labrum anterior and posterior) tears. The symptoms of SLAP lesions can mimic those of impingement syndrome, pathologic conditions of the rotator cuff or the acromioclavicular joint, or other shoulder disorders [6].

Anteroinferior Labral Tears

Bankart lesions occur at the anteroinferior aspect of the labrum, often as a result of an anterior shoulder dislocation. They are usually seen in association with a Hill-Sachs deformity of the glenoid.

Perthes lesions are a variant of Bankart lesions. They also present as an anterior shoulder injury where the anterior labrum is avulsed from the glenoid by remains partially attached to the scapula via the periosteum. It is best seen on MR arthrography.

Glenolabral articular disruption (GLAD) lesions are associated with injury to the anteroinferior articular cartilage and anteroinferior labrum.

Anterior labroligamentous periosteal sleeve avulsion (ALPSA) lesions occur when the anteroinferior labrum remains attached to the scapular periosteum, but the periosteum is often widely lifted or stripped. It is often associated with inferomedial displacement of the inferior glenohumeral ligament complex on MRI.

Posteroinferior Labral Tears

A reverse Bankart lesion occurs when there is detachment of the posteroinferior labrum and avulsion of the posterior capsular periosteum. Similar to its anterior counterpart, such lesions can lead to laxity and subsequent posterior displacement of the humeral head.

Posterior glenolabral articular disruption (posterior GLAD) lesions are also similar to their anterior variant. It is associated with injury to the posteroinferior articular cartilage and posteroinferior labrum.

Posterior labrocapsular periosteal sleeve avulsion (POLPSA) lesions occur when the posterior periosteum and posterior labrum strip off the glenohumeral joint. This is usually in the setting of trauma.

Kim lesions are superficial labral tears that occur between the posterior labrum and the glenoid articular cartilage. There is no associated labral detachment, though failure to identify and treat this type of lesion may lead to posterior instability.

Circumferential Labral Tears

Pan-labral or circumferential tears of the glenoid labrum are an uncommon injury, comprising roughly 2.4% of all labral lesions [7]. These 360-degree lesions can cause significant and recurrent instability and pain.

Clinical Presentation

Labral tears often present as deep-seated pain within the glenohumeral joint or anterior shoulder pain radiation to the biceps muscle. The pain can either be constant or activity-related. It is often associated with glenohumeral instability, as well as mechanical symptoms such as clicking, catching, popping, or locking. Neurological symptoms including paresthesias, numbness, and tingling can be present in the setting of paralabral cysts causing nerve compression [8]. Patients may also endorse a "dead arm" sensation.

Physical Exam: *Labral Tear Pain-Provoking Tests*

O'Brien Test for Superior Labrum Anterior Posterior (SLAP) Tear

With the patient standing, the arm is elevated forward to 90° and placed at 10–15° of adduction and full internal rotation (thumb pointing down). The patient is asked to hold the arm in that position and resist a downward force applied by the examiner over the distal forearm. This is repeated with the arm in the same position but in full external rotation (palm facing upward). The test is positive if the first maneuver causes or aggravates pain which improves with the latter maneuver. Pain felt deeply in the glenohumeral joint is suggestive of labral tear, while pain felt over the AC Joint is suggestive of AC joint arthropathy.

Jerk Test for Posterior Labrum Tear

With the patient sitting, the examiner stabilizes the scapula with one hand. With the other hand, the examiner abducts the patient's arm to 90° and internally rotates it to 90°. A posterior-directed axial force is then applied while bringing the arm into adduction. This aims to displace the humeral head posteriorly. Sharp glenohumeral pain is suggestive of a posterior labrum lesion.

Kim's Test for Posterior-Inferior Labrum Tear

The patient is sitting against the back of a chair. The arm is placed in 90° of abduction and internal rotation with the elbow flexed to 90°. The examiner holds the patient's elbow and proximal arm and flexes the arm forward by 45° while applying an axial posterior and inferior force on the proximal arm. The arm is then taken into adduction. The test is positive if this causes posterior shoulder pain with or without a posterior clunk of the humeral head. Essentially, Kim's test is a variation of the Jerk test that assesses the posterior-inferior part of the labrum (rather than the posterior labrum) due to the application of an inferior force. During the test, the humeral head is also compressed onto the glenoid.

Crank Test Vs O'Brien Test

According to Stetson et al. in a study of 65 patients with initial symptoms of shoulder pain, the Crank test was positive in 45% of patients, and the O'Brien test was positive in 63%. The Crank test had a positive predictive value of 41%, was 56% specific, was 46% sensitive, and had a negative predictive value of 61%. The O'Brien test had a positive predictive value of 34%, was 31% specific, was 54% sensitive, and had a negative predictive value of 50%. Shoulder MRI had a positive predictive value of 63%, was 92% specific, was 42% sensitive, and had a negative predictive value of 83% [9].

Diagnostic Studies [10, 13–15]

MRI and MR Arthrography

On conventional MRI, labral tears are best seen on fat-saturated (FS) fluid-sensitive sequences. However, in MR arthrography (MRA), the T1, T1 FS, and T2 FS sequences are typically combined for further assessment. Imaging in three orthogonal planes is recommended, and additional planes may be included for a more detailed assessment. There are differing viewpoints as to whether MRA is superior to conventional MRI in detecting labral tears. A 2012 meta-analysis demonstrated that the accuracy of MRA was slightly superior, with a sensitivity of 88% and a specificity of 93%, compared to a sensitivity of 76% and a specificity of 87% for conventional MRI [10].

The biggest advantage of MR arthrography comes from the joint distension, which can help spot otherwise occult tears. However, patients with acute lesions often have a joint effusion which also distends the joint space, making the contrast administration unnecessary. MR arthrography has excellent accuracy in differentiating between SLAP lesions and its anatomic variants [11, 12].

CT Arthrography

CT arthrography (CTA) has been reported to have 97.3% accuracy for detecting Bankart lesions and 86.3% for SLAP lesions. This makes CTA comparable to MRA and provides an alternative for patients with MRI contraindications [10]. CT is also superior to MR in assessing bony structures, so this modality is helpful in detecting coexisting small glenoid rim fractures.

Management

Treatments for pain and instability include analgesia, activity modification, corticosteroid injections into glenohumeral joint, as well as physical therapy guided toward posterior capsular stretch-

ing to address contractures and regain motion loss, improve scapular muscle strength and neuromuscular control, and increase extremity and core strengthening. Platelet-rich plasma, in conjunction with appropriate rehabilitation, can also assist in the clinical recovery of glenoid labral tears [16]. Nonsurgical treatment of SLAP tears can be quite successful, as some studies report up to 70% of patients with nonsurgical SLAP tears returning to sports with improvement in pain and function [17]. As such, a trial of nonsurgical treatment may be appropriate for some patients.

Surgical options can include *arthroscopic debridement* of the degenerate labrum tear if biceps anchor is stable, *resection of an unstable bucket handle fragment*, partial removal of a damaged LHB tendon segment/flap, and *arthroscopic labrum reattachment*. Alternatively, open or arthroscopic *LHB tendon tenotomy or tenodesis* is another option that is preferable in patients over 35–40 years old, as the outcomes of arthroscopic labrum repair in this population are inferior to those seen in younger patients [18, 19].

Outcomes of arthroscopic resection of glenoid labral tears in athletes vary depending on whether or not there is shoulder instability. In a 29 patient case study with greater than 2 year follow-up, there was a statistically significant difference in the functional outcome between patients with stable versus unstable glenohumeral joints. In those with stable joints, there was a 91% good or excellent functional outcome. In those with unstable joints, there was a 25% good functional outcome and a 75% fair or poor functional outcome. Arthroscopic resection of a longitudinal labral tear in a stable shoulder can relieve the patient's discomfort and allow him or her to return to athletic competition. In patients with anterior instability and labral tears, labral debridement was not a successful alternative to formal stabilization [20].

Outcomes of SLAP repair can also vary significantly. Roughly 88% of professional athletes return to pre-injury levels within a year [21], whereas 26% experience resolution of pain and only 13% have complete restoration of normal function [22].

Postoperative Rehabilitation and Return to Activities after SLAP Debridement or Repair

For the first 6 weeks after surgery, any position which may create tension on the biceps should be avoided and approached with caution thereafter. These positions include shoulder extension, internal rotation behind the back, and using the arm to carry or lift objects with the elbow extended. External rotation with the arm at 90° of abduction should also be approached with caution. When a biceps tenodesis is performed, any resistive active motion of the elbow, either in flexion or supination, is avoided [23, 24].

0–6 Weeks Postop: Protective and Restrictive Phase

A sling is used for comfort during the first 7 to 10 days after surgery. Phase I ROM exercises are then initiated and performed as tolerated. External rotation ROM may need to be limited to 45° in patients who have evidence of a peel-back tear. Patients are expected to achieve full passive forward elevation 6 weeks after surgery. Goals and treatments examples are listed below:

Pain and swelling control: Cryotherapy, electrical stimulation, grade I/II mobilizations.

Mobilization **(safe ROM, 10 to 25 repetitions, 2 to 3 times per day):**

- Sling for comfort for up to 3 weeks.
- Passive forward elevation in plane of the scapula by 2 days with physician-set limitations.
- Passive external rotation (ER) in plane of the scapula (POS) and abduction/external rotation with physician-set limitations.
- Pendulum.
- Progress to active ROM in all motions.

Strength **(safe ROM, 3 to 5 x 10 repetitions, 2 times per day, 0 to 5 lbs):**

- Begin with isometrics for flexion, adduction, abduction, extension, internal/external rotation, and grip strengthening.
- Wrist curls and extensions.
- Elbow curls and extensions.
- Shoulder shrugs with scapular adduction (retraction).
- Bent row.
- Scaption.
- Non-weight-bearing push-up.
- Seated press-up.
- Modified prone horizontal abduction.
- Side-lying external rotation.
- Modified prone 90–90° external rotation.
- Arm at side in internal rotation.

Proprioception **(safe ROM, 10 to 25 repetitions, once per day):**

- Rhythmic stabilization.
- Weight shifts (progress wall to table).
- Oscillations (Boing [Boing Ltd., Bristol, UK], Bodyblade [Fitter International Inc., Calgary, Alberta, Canada] or tubing.

Cardiovascular fitness **(30–60 min, 3–5 times per week):**

- Bicycle.
- Stairmaster/elliptical.
- Walking.

Mobilization **(active ROM against suture line in all directions):**

- Passive ROM and active ROM
- 60°–90° external rotation
- 45°–60° internal rotation
- 135°–155° abduction
- 135°–165° scaption

Strength**:**

- 3+ to 4/5 on manual muscle testing
- Progress exercises through available ROM.
- Add weight as tolerated.

6–12 Weeks Postop

At 6 weeks postop, active ROM, passive ROM, and rotator cuff and deltoid strength are assessed. Patients are instructed to begin phase II ROM exercises, which include extension, internal rotation, cross-body adduction, and phase I strengthening exercises, which include external rotation, internal rotation, and extension. Scapular retraction exercises with elastic resistance can also be performed at this time. Goals and treatments examples are listed below:

***Mobilization*:**
- Passive ROM and active ROM
- 90° external rotation
- Full internal rotation
- 160°–180° abduction
- Gradually increase passive ROM stretching
- Grades III–IV mobilization techniques
- Wand
- Overhead pulley

Strength
- 4 to 4+/5 on manual muscle testing
- Progress above exercise weights to 5 lbs. Progress to weight machines.
- 15% or less differences isokinetically:
  - Bench press
  - Military press
  - Seated row
  - Latissimus dorsi pull-down
  - Biceps
  - Triceps

***Proprioception*:**
- Progress to full weight-bearing on closed chain proprioceptive activities.
- Progress open and closed chain proprioceptive exercises closer to end range.

***Function*:**
- Light, nonrepetitive overhead activity, light lifting.
- Activities of daily living as tolerated.
- No sports activities.

12–24 Weeks Postop

Patients who are able to perform the phase I strengthening exercises with the green band are instructed in phase II strengthening (abduction, forward elevation, external rotation supported at 45°). Advanced scapular strengthening exercises may be used at this time. In addition, biceps strengthening with light weights may begin.

For the overhead athlete, sport-specific training can begin using the Bodyblade and plyometrics to enhance neuromuscular control, strength, and proprioception. Recommendations and instruction for proper use of gym equipment should also be done at this time. Patients should be encouraged to avoid exercises with the arm behind the plane of the body. Latissimus pull-downs should be performed to the chest, not behind the head. Caution should be used when performing any type of "pushing" exercise, such as chest press or shoulder press. It is safer to perform these exercises with a machine to allow for greater safety.

***Mobilization*:**
- Progressive passive and active ROM exercises
- Full or sufficient ROM to perform sport

***Strength* (continue weight machines):**
- 5/5 manual muscle testing: Progress to free weights
- <10% isokinetic strength difference:
  - Military press
  - Bench press
  - Incline press
  - Rows
  - Flies

***Proprioception* (weight-bearing on unstable surfaces):**
- <10% proprioception difference: Bodyblade, Plyoback.

***Function* (gradually progress to functional activities):**
- Begin return to football, wrestling, overhead activities.

4 Months Postop

The player or athlete must be ready to begin throwing. The role of the pitcher, number of throws, distance, intensity, and frequency of the program need to be considered. It is recommended to allow at least 6 weeks of strengthening before considering a throwing program. Scapular positioning, shoulder ROM, spinal mobility, and lower extremity ROM should also be evaluated at this time. When considering a return to throwing, it is important to evaluate the entire body mechanics in the process of throwing. Asymmetries are bound to exist, and it is the goal and the art of rehabilitation to conclude what is an acceptable difference. Leggin et al. recommend performing the following tests and measures prior to initiating a throwing program:

- Shoulder ROM (internal rotation at 90°, external rotation at 90°, total ROM in comparison with uninvolved side, flexion, abduction).
- Scapular evaluation—static and dynamic evaluation and any concerns for scapular dyskinesis (digital photo or video).
- Spinal ROM—lumbar flexion, sidebending, rotation (digital photos).
- Lower extremity ROM (hip internal rotation and external rotation in prone and seated positions, hip flexion, Thomas test, knee flexion).
- Functional movement screen performed (looking for asymmetry to be addressed with appropriate corrective exercises) [25, 26].

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Adhesive Capsulitis

5

Craig Silverberg and Habib Zahir

Introduction

Adhesive capsulitis, also known as arthrofibrosis or frozen shoulder, describes a condition where the shoulder is significantly limited in its range of motion leading to significant pain. This condition is a pathological process in which the connective tissue surrounding the shoulder joint capsule becomes thick, stiff, and inflamed. The shoulder joint capsule contains ligaments that attach the top of the humeral head to the glenoid (socket), also referred to as the "ball-and-socket" joint [1].

Adhesive capsulitis can be a primary or secondary finding. Primary, or idiopathic, adhesive capsulitis occurs without any inciting event or trauma. Females between the ages of 40 and 60 years old are most prone to this condition, which often involves the nondominant extremity. Secondary adhesive capsulitis is often observed after injury to the shoulder, such as a periarticular dislocation of the glenohumeral joint. It can also be a severe complica-

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tion after open or arthroscopic shoulder surgery, including rotator cuff repair and shoulder arthroplasty [2]. The incidence of adhesive capsulitis in the general population is approximately 3–5%. The incidence is significantly higher, approximately 20–25%, when individuals have other comorbidities, such as diabetes, thyroid disease, stroke, and Parkinson disease, and when people are taking antiretroviral medications [3].

Pathology

The pathology of adhesive capsulitis is poorly understood, and it is often idiopathic. The leading hypothesis is based upon arthroscopic observations that suggest an initial stage of inflammation which then progresses to fibrotic contracture of the shoulder capsule. Neviaser and Neviaser classified adhesive capsulitis into four stages based on the arthroscopic appearance of the joint capsule: [4].

- Stage 1 is the "pre-adhesive stage." It entails proliferation of fibroblasts without adhesion formation. Patients typically have full range of motion (FROM) and report pain, often at night. Symptoms are nonspecific and misdiagnosis is common.
- Stage 2 is the "acute adhesive synovitis." It is characterized by hypertrophy of the synovium and early adhesion formation, often in the inferior capsular fold. Patients begin to experience mild loss of range of motion (ROM) with pain.
- Stage 3 is the "maturation stage." It involves the transition of synovitis to fibrosis, in which the axillary fold is obliterated and is often adhered to the capsule. Pain is less severe in this stage than in the earlier stages, but ROM is significantly restricted.
- Stage 4 is the "chronic stage." It is the last stage in which ROM is severely reduced and there is an increase in dense fibrotic adhesions. This makes it very difficult to identify intra-articular structures. Patients at this stage have minimal pain, unless their ROM is forcefully moved beyond the restraints of their scarred capsule.

The initial inflammatory reaction involves the recruitment of inflammatory cells and cytokines, such as macrophages, B and T lymphocytes, TGF-b, TNF-a, and interleukins [5]. The presence of cytokines is evidence of a possible autoimmune process, but the relationship is also not well established [6]. One study suggests inflammation is seen in the axillary fold, the anterosuperior joint capsule, the coracohumeral ligament, and the rotator cuff interval [7]. Another study indicates that there is a pathophysiological difference in protein expression between the upper (rotator interval and middle glenohumeral ligament) and lower (anterior-inferior glenohumeral ligament) parts of the shoulder in adhesive capsulitis [8]. These inflammatory changes eventually progress to reactive capsular fibrosis, which is also driven by increased levels of TGF-B and other profibrotic cytokines [3, 6]. This process overlaps and corresponds to the different arthroscopic and clinical phases (Fig. 5.1).

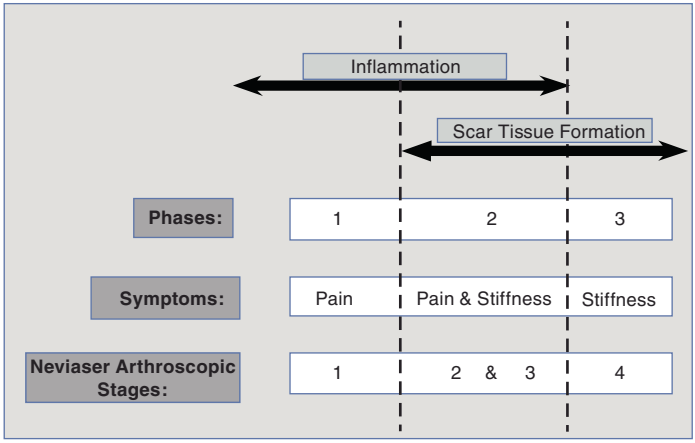

Fig. 5.1 Pathologic phases of adhesive capsulitis. Depiction of the overlapping nature of inflammation and scar tissue formation creating the breakdown of the three clinical phases of adhesive capsulitis. This is compared with four phases described by Neviaser AS, and Hannafin JA (2010)

In summary, inflammatory and fibrotic pathogenesis contributes to the thickening and contraction of the glenohumeral joint capsule and the tissue surrounding the joint, thereby diminishing the joint volume. The normal shoulder joint volumetric capacity is 28–35 mL of injected fluid, whereas in adhesive capsulitis, the joint is limited to only about 5–10 mL [4].

Clinical Presentation

Symptoms of adhesive capsulitis are divided into three stages:

- In the initial "freezing" stage, the shoulder becomes stiff and painful to move. Pain is usually worse at night, and the inability to move the shoulder steadily increases. This stage lasts 6 weeks to 9 months [4, 9].
- The "frozen" stage is the period in which the pain starts to decline, but the shoulder remains just as stiff. This makes it difficult to complete daily tasks and lasts about 2–6 months [4, 9].
- The "thawing" or recovery stage entails a further decrease in pain and slow improvement in shoulder mobility. This stage usually lasts 6 months to 2 years, with an eventual return to full motion and strength [4, 9].

Physical Exam/Diagnostic Imaging

This condition is usually diagnosed clinically, based on a thorough history and physical exam. Adhesive capsulitis causes significant decrease in both active and passive shoulder range of motion [10]. Subtle clues in the history and physical exam can help differentiate adhesive capsulitis from a variety of other conditions that cause a painful, stiff shoulder [11]. The physical exam findings are tenderness to touch at the deltoid insertion and over the anterior and posterior capsule [10]. There can be pain around the medial scapula due to scapulothoracic motion.

The normal shoulder range of motion in degrees best measured by a goniometer includes abduction 180, adduction 45,

extension 45, forward flexion 90, internal rotation 55, and external rotation 40–45. In individuals with adhesive capsulitis, typically, the range of motion is lost in the following order: external rotation, abduction, internal rotation, and then forward flexion [10]. When evaluating patients using special tests of the shoulder, the Neer and Hawkins tests for impingement and Speed's test for biceps tendinopathy are typically positive in patients with frozen shoulder. There is no laboratory testing indicated for diagnosis.

Additionally, X-rays are routinely obtained to make sure the symptoms are not due to another cause such as arthritis. Further imaging such as MRI and ultrasound are usually not needed to make the diagnosis; however, features of the shoulder to look out for using these modalities are described below, per Radiopaedia [12].

Ultrasound

- Limitation of movement of the supraspinatus is considered a sensitive feature.
- Limited external rotation, identified when positioning for subscapularis tendon assessment.
- Thickened coracohumeral ligament (CHL) can be suggestive.
- Thickened inferior glenohumeral capsule echogenic material around the long head of biceps at rotator interval.
- Increased vascularity of long head of biceps at rotator interval.

MRI/MR Arthrography

The signs of adhesive capsulitis are variable with some but rarely all of the following expected to be present:

- T2 hyperintensity of the inferior glenohumeral ligament on T2 fat-saturated sequences.
- Coracohumeral ligament thickening >4–7 mm.
- Subcoracoid triangle sign

- Joint capsule thickening:
  - Anterior capsule thickness > 3.5 mm and abnormal hyperintensity.
  - Axillary pouch thickening >3–4 mm.
- Abnormal soft tissue thickening within the rotator interval with signal alteration.
- Abnormal soft tissue encasing the biceps anchor.
- Variable capsular and synovial enhancement within the axillary recess and rotator interval

Management/Return to Activities

Management of adhesive capsulitis is based on the etiology and severity of the fibrosis. For a majority of patients, the symptoms are self-limiting and will resolve over a 1–3-year period with conservative management. Physical therapy combined with a home exercise program is the initial mainstay of treatment. It is recommended to initially start with a gentle range of motion of the joint combined with progressive stretching exercises. Strengthening is not the focus in therapy for this condition [4].

Pharmacological treatment is often used as an adjunct with physical therapy. Nonsteroidal anti-inflammatory drugs are first-line adjuncts to help patients with their pain to make therapy more tolerable; however, Neviaser AS et al. showed that these medications don't influence overall recovery [4]. Oral and intra-articular steroid injections also have transient effects on pain, but studies show no significant change in motion of the joint or overall recovery. Other agents such as collagenase injections and anti-TNF agents are being studied to target the physiologic markers thought to play a role in the development of the pathological process.

Patients who do not improve with physical therapy are referred to an orthopedic surgeon for surgical options. Manipulation under anesthesia used to be the mainstay surgical approach prior to the advancement of arthroscopic procedures. Arthroscopic capsular release is the most frequently used approach by many institutions

today. Arthroscopic interventions allow the entire joint to be visualized by the surgeon, which allows the surgeon to confirm the diagnosis and to perform a more precise capsulotomy. Studies have shown that the arthroscopic approach have better outcomes for patients in alleviating pain and restoring the function of the shoulder within a 2–5-year period postsurgery [4]. Le HV et al. showed that patients who tend to have the worst outcomes with the arthroscopic capsular approach are females over the age of 50 years old with type 2 diabetes [2].

Proper postoperative management is very important to prevent postoperative inflammation which can lead to recurrence of adhesive capsulitis. In order to prevent this common and dreaded complication following surgery, it is important that the shoulder is kept immobilized in 90 degrees abduction and external rotation, with the head of the bed elevated to 30 degrees. Regardless of the surgical approach performed, directed physical therapy should be started as early as postoperative day 1. Patients are encouraged to maintain the arm in 90 degrees abduction and to reach the arm across the top of the head to touch the opposite ear with emphasis on internally and externally rotating the arm. It is recommended for patients to sleep with their arm abducted for at least 2 weeks after surgery. Therapy should be continued outpatient in a supervised setting with the same emphasis on the initial preoperative therapy of stretching and ranging the joint, with minimal emphasis on strengthening or vigorous movement [4].

It is best for patient's to slowly and progressively ease back into vigorous activities. The length of time to return back to normal activities greatly varies based on the individual, but at the minimum, a few weeks of continuous physical therapy is essential to return back to normal activities.

Conclusion

Adhesive capsulitis is a very common shoulder condition, typically found in middle-aged females with comorbid conditions. It shares many clinical signs as other shoulder pathologies, so a

thorough history and clinical examination is key to the diagnosis. If managed efficiently and in a timely manner with a combination of pharmacological, rehabilitative, and/or surgical treatment, patients can return back to their baseline functioning and quality of life.

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Osteoarthritis

6

Aaron Bolds

Introduction and Pathology

Chronic shoulder pain is a common chief complaint among patients in musculoskeletal clinics. Roughly 30% of the general population is affected by persistent shoulder pain, with 12.7–24% of cases being secondary to acromioclavicular pathology [1]. Osteoarthritis (OA) is a common etiology for chronic shoulder pain. The anatomical locations affected are the acromioclavicular joint (ACJ) and glenohumeral (GH) joint, with ACJ being the most common site affected by OA. Glenohumeral osteoarthritis makes up approximately 5–17% of complaints in patients presenting with shoulder issues, including pain and decreased range of motion [2]. In general, osteoarthritis of the shoulder joint is less common than in weight-bearing joints such as the knee and hip. However, the disabling impact is similar because of the functional dependence of the shoulder joint. The rate and degree of disease progression are difficult to predict. A 14-year population-based cohort study showed that knee OA disease progression occurred at an annual rate of 2.8% [3]. However, there has not been any

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progression rate predicted for shoulder osteoarthritis. As the aging population increases, we can expect to see higher prevalence of shoulder OA.

Shoulder OA can be either primary or secondary, meaning either idiopathic or provoked by a preceding event such as trauma, dislocations, infections, or chronic rotator cuff tear [4]. The pathophysiology of OA is similar regardless of the anatomical location and joint affected. OA is a disease process that primarily affects the cartilage of the joint. The role of articular cartilage is to allow for smooth and lubricated surfaces for articulation. Cartilage is made up of collagen, proteoglycans, chondrocytes, and water. Collagen and proteoglycans are the building blocks of the extracellular matrix and represent 20–30% of cartilage. Chondrocytes represent roughly 1–2% with the remaining 70–80% being fluid, primarily water [5]. A hallmark of OA is the loss of balance between degradative and non-degradative enzyme activities, leading to progressive cartilage destruction.

Traditionally, OA was thought to be primarily a wear-and-tear process and sequela of aging. However, studies have shown variations between aging cartilage and OA cartilage. There appears to be more denatured type II collagen found in OA cartilage as compared to normal aging cartilage, as well as a difference in the water content [5]. Matrix metalloproteinases are key players involved in articular cartilage degradation. These enzymes are secreted by chondrocytes, which are induced by the inflammatory cascade secondary to repeated stimulation.

Some of the risk factors for developing shoulder OA include previous shoulder joint pathology, prior surgery, obesity, aging, mechanical forces related to exercise and occupation, and genetics. Numerous studies have established that shoulder dislocations are associated with increased risk of development or progression of shoulder OA. The prevalence of radiographic changes in glenohumeral OA following dislocations and instability surgery is reported to be as high as 56–68% [2]. Proximal humeral and glenoid fractures can cause damage to the articular cartilage and trigger joint degradation. Scapular morphology is also important to consider as a risk factor for shoulder OA. The critical shoulder angle (CSA) has implications in shoulder OA. It is measured by

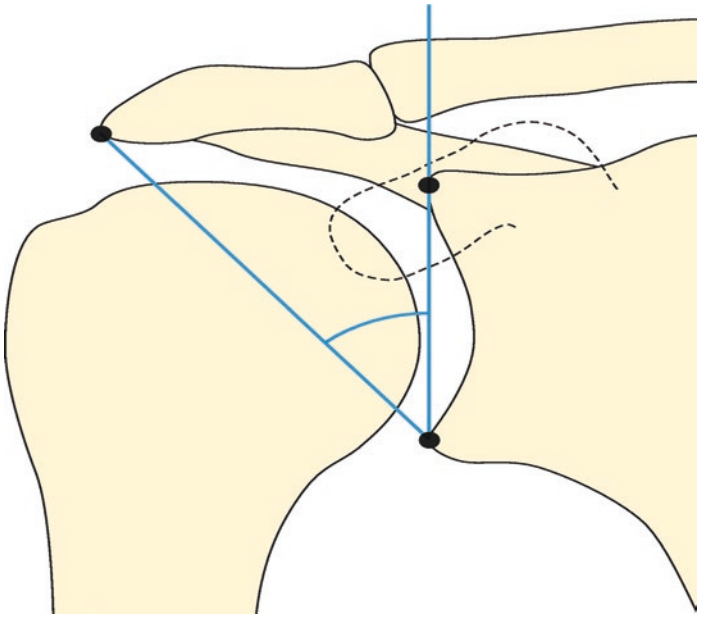

Fig. 6.1 Showing measurement of critical shoulder angle [6]

drawing a line from the inferior edge to the superior edge of the glenoid and an additional line from the inferior edge of the glenoid to the lateral edge of the acromion (Fig. 6.1). The mean CSA in individuals without shoulder pathology is 33.1 degrees [6]. In shoulder OA, the CSA is decreased, resulting in increased medially directed compressive forces across the glenohumeral joint [2].

Certain occupations are prone to developing shoulder OA, such as construction workers.

Athletes who participate in overhead sports, such as tennis, weight lifting, and baseball, are at increased risk of developing shoulder OA. While excessive mechanical loading can be detrimental to joint surfaces, some degree of loading is necessary for maintenance and improvement of biomechanical integrity of cartilage. Thus, both sedentary and excessively active lifestyles can predispose an individual to OA. Active lifestyle and manual work

lead to increased overhead and upper extremity use. Obesity is a proven risk factor for hip and knee OA secondary to increased weight loading on the joints. Obesity is also a risk factor for shoulder OA, but through a different mechanism. Prior studies show that adipose tissue can initiate systemic inflammation through the release of cytokines. Thus, in addition to dyslipidemia, obesity potentially can influence the progression of shoulder OA [2].

Clinical Presentation

Patients will typically present with chronic, deep shoulder pain, exacerbated by activity. Crepitus and locking can occur in later stages of disease. The pain associated with shoulder OA usually leads to restricted range of motion and difficulty with activities of daily living (ADLs). There can also be a nighttime temporal pattern associated with symptoms. Pain can be referred to different aspects of the shoulder, but patients will often complain of pain located posteriorly and/or superiorly if the glenohumeral joint is the site of pathology. Acromioclavicular joint arthritis will typically localize to the anterior and/or superior aspect of the shoulder. There is a psychological component to shoulder OA that should be considered. Patients can sometimes present with symptoms concerning depression. Pain that limits function and prevents participation in occupation, exercise or sporting activities, and ADLs can trigger or exacerbate existing depressive symptoms. A study on psychological status and quality of life of a cohort of patients with glenohumeral OA showed rates of anxiety and depression, at 19.5% and 15.2%, respectively [3].

Physical Exam

The evaluation of the patient should begin with appropriate history taking. Symptom onset, exacerbating factors, temporal pattern, pain characterization, and location should be addressed. The

clinician should also obtain information related to prior and current activity level, comorbidities, occupation, and functional expectations. These aspects of the medical history can help guide management and prognosticate outcomes. Prior studies indicate that most cases of OA in younger population (50 years and younger) were usually secondary to posttraumatic, osteonecrosis, and rheumatoid arthritis (RA) [7].

When a patient presents with shoulder pain, it is important to consider other etiologies. Effusions and any signs of infection should be ruled out on inspection. Palpating for point tenderness at the acromioclavicular and sternoclavicular joint should be followed by checking the range of motion. Insertion points of rotator cuff muscles should also be palpated. Cervical spine and shoulder range of motion should be examined while listening and feeling for crepitus. Manual muscle testing may show deficits, but they may be secondary to pain and not true weakness. Rotator cuff pathology and shoulder OA may have some signs and symptoms which overlap, specifically pain and decreased range of motion.

Physical exam maneuvers should be performed to evaluate rotator cuff muscles and impingement signs. Some of the tests that can be used are Hawkins test (impingement), empty can test (supraspinatus), resisted external rotation (infraspinatus and teres minor), and lift-off test (subscapularis). Bicep tendinopathy can be evaluated with Speed's test (forward flexion against resistance) and Yergason's test (supination against resistance). The AC joint can be evaluated with scarf test (cross-body adduction). The painful arc test (pain with shoulder abduction between 60 and 120 degrees) can be used to evaluate subacromial impingement.

Diagnosis

History taking and a thorough physical exam are essential for diagnosing shoulder OA. However, although not required, imaging is often used to confirm diagnosis. Radiographic characteristics of shoulder OA are joint-space narrowing, osteophytes,

subchondral sclerosis, cysts, and articular cartilage loss [4]. As the rest of the imaging findings are discussed, it is important to understand that imaging findings do not always align with a patient's symptoms. It is important that the patient is the focus of the treatment plan, and not imaging.

Studies show that MRIs depicted asymptomatic OA in 68% of volunteers aged 19–30 years old and in 93% of those older than 30 years of age [8]. Imaging features, such as capsular hypertrophy and effusion, do not correlate with symptoms, while subchondral marrow edema often suggests symptomatic OA [8]. In glenohumeral OA, the amount of posterior glenoid bone loss is significant with regard to indications for shoulder arthroplasty [7]. The Walch classification is used to grade the degree of glenoid bone loss and type of glenoid wear pattern (concentric vs eccentric (Fig. 6.2) [7].

Ultrasound is another diagnostic imaging tool that can be used to evaluate for shoulder OA (Fig. 6.3). Diagnostic scanning for shoulder OA can reveal capsular hypertrophy, joint-space narrowing, osteophytes, bony irregularities, and synovial hypertrophy [10] (Fig. 6.4). Ultrasound has advantages which include no radiation exposure for patient and practitioner, high sensitivity, and low cost. However, ultrasound is user-dependent, which can be a disadvantage compared to other imaging modalities.

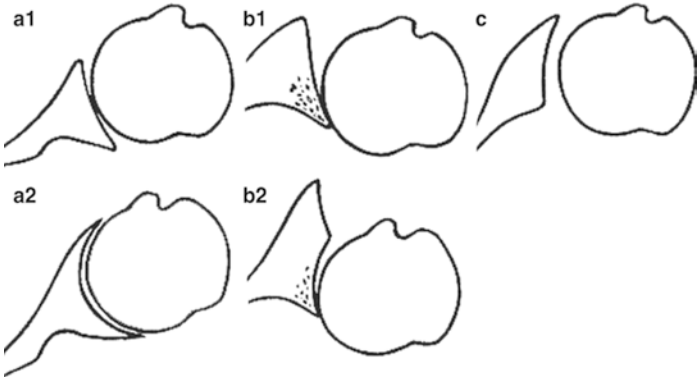

Fig. 6.2 Walch classification for glenoid wear patterns and glenoid bone loss [7]. (**a1**) mild central erosion, (**a2**) moderate central erosion with humeral head protrusion into glenoid, (**b1**) posterior joint space narrowing with subchondral sclerosis, (**b2**) biconcave morphology of glenoid, (**c**) glenoid retroversion

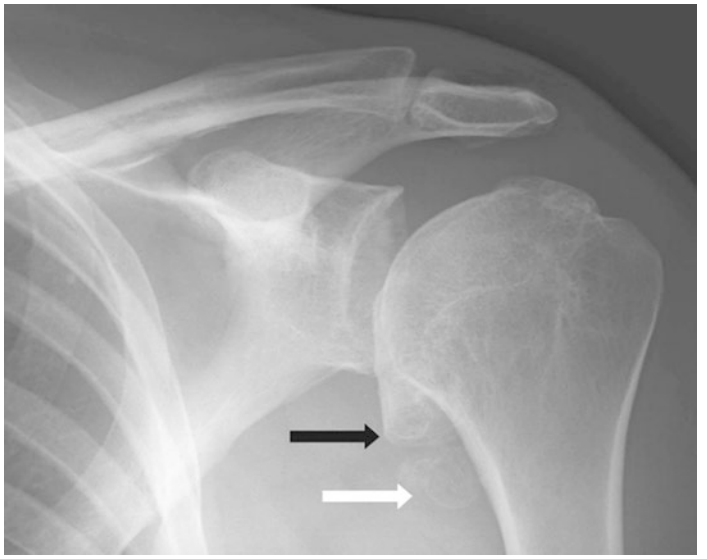

Fig. 6.3 Degenerative changes of the glenohumeral joint, including osteophytes medially (black arrow) and inferiorly (white arrow) shown in the shoulder X-ray [9]

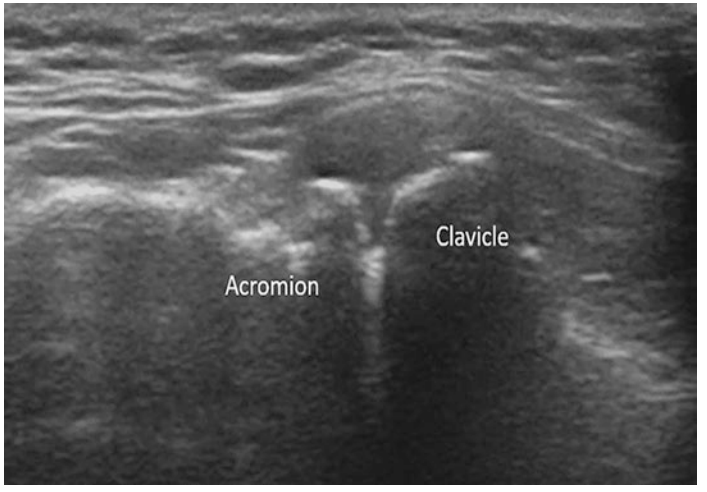

Fig. 6.4 Ultrasound scan revealing superficial osteophytes, joint-space narrowing, and capsular hypertrophy [10]

Treatment

Conservative, nonoperative management is a first-tier treatment for shoulder OA. Both oral and topical medications can be trialed. Physical therapy and modalities used during physical and occupational therapy also play an important role in the management of shoulder OA. Injections such as viscosupplementation, corticosteroids, and orthobiologics are considered second-tier treatment. Surgery is usually indicated for patients that have failed conservative treatment.

Age, activity level, symptoms, and radiographic signs are factors that should be considered when deciding on a treatment plan [11]. Conservative management typically begins with activity modification, physical therapy, and medications (oral and topical). NSAIDs should be used with caution, given the side effect profile. Patients with a history of gastrointestinal bleeding or renal disease should avoid long-term use of NSAIDs. Voltaren is a topical NSAID that has less systemic effects compared to its oral formulation, but it still should be used with caution in those with significant comorbidities. Tylenol has a better side effect profile and can be used at a maximum dose of 3000 mg per day. Topical capsaicin works on transient receptor potential cation channel subfamily V member 1 (TrpV1), also known as the capsaicin receptor and the vanilloid receptor 1, which is found on key sensory afferents. Capsaicin has been found to be effective in treating pain in OA [3].

Physical therapy and modalities (such as heat, ice, compression, myofascial release, and electrical stimulation) play an important role in both injury prevention and rehabilitation. Electrical stimulation with a TENS unit can stimulate nerves to decrease pain, increase blood flow to expedite healing, and provide an anti-inflammatory effect [1]. Exercises should focus on strengthening adjacent muscles, improving shoulder range of motion, and focusing on the ability to perform ADLs.

Intra-articular corticosteroid injections are part of a second-tier treatment plan. Studies have shown that ultrasound-guided acromioclavicular joint injections resulted in greater improvements in

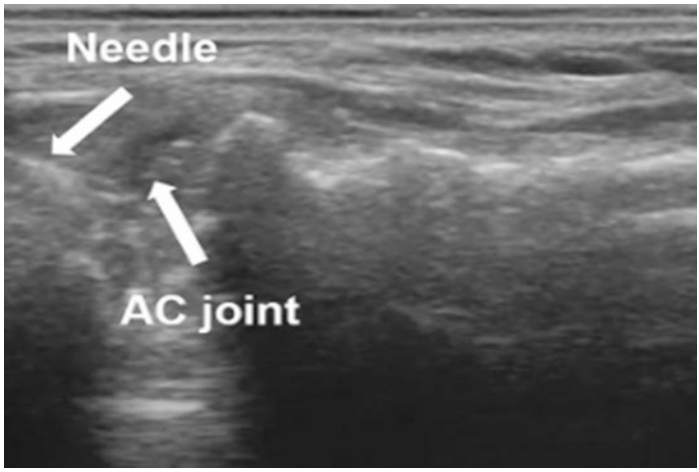

Fig. 6.5 Ultrasound-guided, AC joint injection with needle tip being visualized using the in-plane technique [10]

pain and functional status when compared to palpation-guided injections at 6-month follow-up [12] (Fig. 6.5). Triamcinolone and methylprednisolone are commonly used corticosteroids for these intra-articular injections. Viscosupplementation with hyaluronic acid has also been found to be beneficial, likely due to decreased levels of synovial hyaluronic acid in patients with advanced OA.

Orthobiologics including platelet-rich plasma (PRP) have demonstrated favorable clinical outcomes in knee OA. However, more studies are needed to evaluate their efficacy for the treatment of shoulder OA. The promise in PRP appears to be its role in hindering the catabolic process involved in articular cartilage degradation [13].

Distal clavicle excision is the surgical option for AC joint OA, after failed conservative management. Interestingly, a scoping review study showed that both conservative and surgical treatments were both effective in AC joint management, with neither appearing superior to the other [14]. Treating glenohumeral joint OA surgically is usually reserved for patients who have failed first- and second-tier treatment. Total shoulder arthroplasty (TSA)

is typically the treatment of choice for geriatric and middle-aged patients who live more sedentary lifestyles. However, for younger or more active patients, joint-preserving procedures (such as extensive capsular release, osteoplasty of the humeral head, and axillary nerve neurolysis) tend to have good early outcomes [3].

Total shoulder arthroplasty (TSA) utilizes a prosthetic humeral and glenoid fossa component, while hemiarthroplasty only utilizes a prosthetic humeral component. In reverse TSA, the normal ball-and-socket anatomy is reversed, placing the ball component at the glenoid fossa and socket component at the humeral head. TSA has been shown to be superior to hemiarthroplasty, allowing for better function and less pain [3]. Reverse TSA is indicated when patients have superimposed severe rotator cuff disease, such as full-thickness tears.

Staying active is encouraged in order to preserve quality of life. Aging athletes who were diagnosed with severe shoulder OA and have failed conservative management may turn to surgery. Studies have shown high successful return to sport rates after arthroplasty, with mean range of return to play being 3.6–8.4 months for swimming, tennis, and golf [7]. Of note, most studies had a mean patient age of 65.5–71 years old [7]. Shoulder OA in younger athletes is usually secondary to another pathology. Studies show success rates for arthroplasty in younger patient populations are worse than in older populations [7]. Therefore, return-to-play protocols after shoulder arthroplasty in younger patients are controversial.

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Sports Trauma

7

Elinor Naor

Noncontact Athletes

Athletes that participate in sports involving overhead include baseball, cricket, javelin, and tennis apply repetitive stress to their upper extremities. Within noncontact sports, overhead athletes are most likely to suffer an injury to the shoulder. In order to execute a throwing motion successfully, athletes must exercise coordinated muscle action at a velocity that is more rapid than the rest of the body. The biomechanical chain involved in overhead throwing includes the hip joint, trunk, scapula, and shoulder joint (Fig. 7.1). The shoulder maintains its stability during these actions via the glenohumeral capsular ligaments. A single kinetic chain failure can result in an overexertion injury to the muscles, ligaments, and tendons surrounding the shoulder [1].

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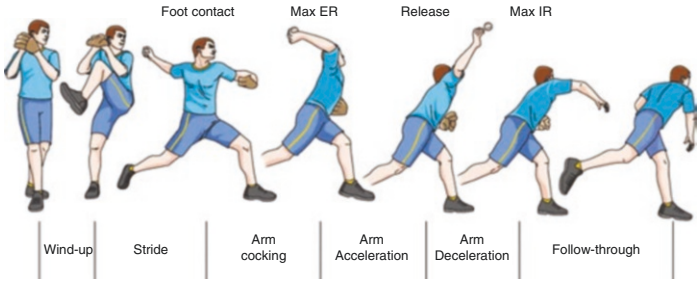

Fig. 7.1 Schematic of the phases of throwing motion from windup to follow-through [1]

Anterior Capsular Ligament Dysfunction

Pathology

When athletes perform repetitive throwing motions, the anterior capsule may be stretched to the point of creating excessive ligamentous laxity. During active abduction, extension, and external rotation, the anterior portion of the inferior glenohumeral ligament (IGHL) is loaded, thus preventing anterior humeral movement. This can sometimes result in traumatic subluxation as the arm moves through space. Subluxation differs from dislocation in that it is an incomplete, transient displacement of the glenohumeral joint. Anterior capsular ligament dysfunction includes any pathology involving the anterior capsule of the glenohumeral ligament.

Clinical Presentation

Athletes suffering from anterior shoulder instability present with shoulder pain elicited by end-range external rotation of an abducted shoulder. Anterior shoulder instability is typically seen in male athletes between the ages of 15 and 29 years old. This pathology is also associated with SLAP lesions as described later in this chapter.

Physical Exam

During the physical exam, inspection of athletes suffering from anterior capsular ligament dysfunction, there typically are no noticeable masses, deformities, or asymmetries. Special tests are used to isolate the anterior capsule as the culprit. The anterior load-and-shift test applies anterior strain on a flexed and abducted shoulder, while the scapula is stabilized to assess for glenohumeral translation. To examine for inferior sulcus sign, the examiner applies inferior traction to the arm while observing for a space between the acromion and the humeral head. Another useful test is the Gagey hyperabduction test, which assesses the IGHL by abducting the shoulder to 105 degrees. If athletes have severe injuries which may need surgical intervention, the physical exam can be performed under anesthesia.

Diagnostic Studies

To diagnose an athlete with anterior capsular ligament disorders, an X-ray (AP and axillary view) is initially obtained to rule out any bony deformities. The gold standard for labral and ligamentous evaluation is MR arthrography (Fig. 7.2). Ultrasound has not proven to be a useful modality for evaluation of deep ligaments of the shoulder. Direct visualization of ligamentous structures can be accomplished using surgical arthroscopy as well [3].

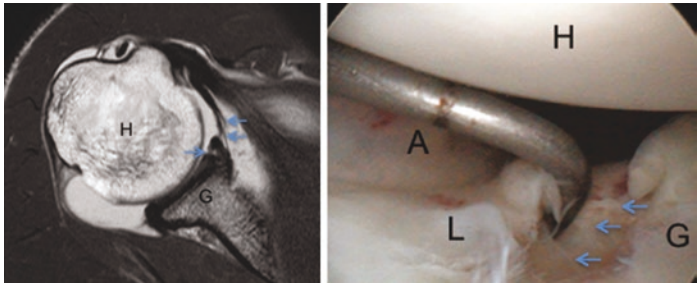

Fig. 7.2 (Left) Anterior labral tear and scapular ligament elongation on MR arthrography. (Right) Arthroscopic view. A, anterior band of the inferior glenohumeral ligament; G, glenoid; H, humeral head; L, anterior labrum [2]

Treatment

Injuries due to repetitive microtrauma, such as anterior capsular ligament dysfunction, typically resolve with conservative management. This includes pain management with oral anti-inflammatories (NSAIDs or acetaminophen) and a home exercise program. Therapeutic exercises should focus on strengthening the rotator cuff along with the core, trunk, and legs. These muscles are all involved in the kinetic chain and will be targeted in the rehabilitation process for other shoulder pathologies as well.

Return to Play

Athletes may return to play once they can execute throwing motions without pain.

Superior Labrum Anterior-Posterior (SLAP) Lesion

Overhead athletes stress their shoulders by straining the anterior capsule structures during the late cocking phase of throwing. This motion causes the posterior capsular structures to shorten and the anterior capsule structures to stretch, resulting in posterior-superior displacement of the humeral head. With repetitive irritation, this can progress into a posterior-superior impingement at the glenoid rim and an injury to the labral-biceps tendon complex. Labral pathologies are covered in detail in Chap. 4 (labral tears).

Pathology

A superior labrum anterior-posterior (SLAP) lesion is localized to the superior labrum of the glenoid and travels from anterior to posterior in a circular manner. There are a total of ten different types of SLAP lesions documented; in this chapter, we will note the most common. Type 1 is the fraying and degeneration of the superior labrum and intact biceps tendon. Type 2 is avulsion of the superior labrum and biceps tendon from the supraglenoid tubercle. Type 3 is a superior labral tear (bucket handle) with intact biceps tendon anchor. Type 4 is a superior labral tear extending to the biceps tendon [4].

Clinical Presentation

Typically, patients suffering a SLAP lesion will present with deep shoulder pain that is incited by throwing motions. They may also mention feeling popping, catching, or clicking of the shoulder. Athletes could also complain of "dead arm syndrome," stating that as they initiate the throw, they experience acute pain followed by a decrease in velocity with associated shoulder instability [5].

Physical Exam

When assessing range of motion in patients with SLAP lesions, asymmetry may be noted when comparing both shoulders. The throwing arm may show evidence of muscular hypertrophy and present in a "rolled forward" position. If palpation along the biceps' tendon elicits pain, a superior labral injury is likely present. When moving the affected shoulder passively in abduction and flexion, apprehension or a catch can be appreciated. In addition, restricted passive internal rotation and extreme external rotation can be observed [6]. A number of special tests are positive in SLAP lesions – these include but are not limited to the anterior glide test, compression rotation, active compression (Obrien), modified dynamic labral shear test, and Crank test [7].

Diagnostic Studies

While soft tissue injuries are not well visualized on plain films, X-rays (AP, scapular Y, and axillary views) are indicated in patients with shoulder pain to rule out associated fractures. MRI arthrography with the patient's arm in abduction and external rotation can be used to identify a SLAP lesion. If an MRI is contraindicated, a CT arthrogram can be done instead. For athletes under the age of 35, high complexity imaging modalities should only be ordered if it appears that surgical repair is needed, along with a referral to orthopedics [8].

Treatment

Initial treatment for SLAP tears should include avoiding exacerbating activities and taking oral anti-inflammatory medications as needed. High-level athletes involved in sports with overhead

throwing are initially evaluated for SLAP lesions; after this, they can begin a home exercise program that includes rotator cuff strengthening exercises within a pain-free range of motion. Recreational athletes who have failed physical therapy may need to be re-evaluated for surgical repair. The therapy script should include graded eccentric and concentric rotational strengthening shoulder exercises along with range-of-motion and strengthening exercises of the spine. Surgery is indicated in patients who fail conservative therapy. There is a large variety of surgical options which include arthroscopic labral refixation and biceps tenodesis. However, patients over the age of 60 years old rarely benefit from labral repair surgery [4].

Return to Play

One study showed that with physical therapy alone, 67% of athletes diagnosed with SLAP lesions were able to return to play, with an average timeline of 53 days for return to play. Studies have shown variable success rates in patients who have failed conservative therapy and have instead opted for arthroscopic SLAP repair [9]. Generally speaking, postoperative return to throwing has been shown to take as long as 6–12 months.

Proximal Humeral Epiphysiolysis (Little League Shoulder)

Pathology

Proximal humeral epiphysiolysis, also known as little league shoulder, is an injury to the growth plate (stress fracture) as a result of overuse [10]. In adolescents, the epiphyseal plate of the humerus is weaker than the surrounding tendons of the rotator cuff and is, therefore, at higher risk of injury with repetitive throwing motions. This recurring instance of microtrauma then does not get an opportunity to heal, ultimately leading to a proximal humeral stress fracture [1].

Clinical Presentation

Proximal humeral stress fractures are typically seen in patients between the ages of 11 and 16 who play overhead throwing sports such as baseball. Athletes will complain of progressively worsening shoulder pain over the span of several weeks that is localized to the proximal humerus and worse with throwing.

Physical Exam

When evaluating a patient with proximal humeral epiphysiolysis, a deformity may be noted along the surface of the proximal humerus. On physical exam, patients will have tenderness to palpation along the proximal lateral humerus. Patients may also complain of pain with active range of motion of the shoulder [11].

Diagnostic Studies

Little league shoulder is diagnosed by visualizing widening of the proximal humeral epiphysis on ultrasound or X-ray (AP view, shoulder externally rotated). An MRI of the shoulder can also be used to confirm the diagnosis. Of note, widening is commonly found in asymptomatic athletes as well; so a clinical correlation is necessary [12] (Fig. 7.3).

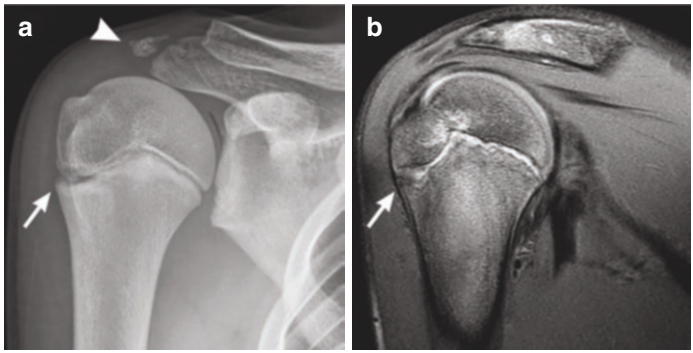

Fig. 7.3 (**a**) AP radiograph: physeal widening and irregularity in the lateral proximal humeral physis are visualized (arrow). Note the well-corticated acromial ossification center (arrow head). (**b**) On MR arthrogram: Physeal widening and irregularity, bone marrow edema and effacement (arrow) [13]

Treatment

When treating little league shoulder, anti-inflammatory medications and ice can be used to assist tolerance of motion and pain control. If diagnosed early, this condition can heal with physical therapy alone. Physical therapy exercises include core and both upper and lower extremity strengthening exercises to improve the kinetic chain. Patients should refrain from performing activities that exacerbate their symptoms. With relative rest, symptoms will resolve within 2–3 months.

Return to Play

Athletes may return to play after completing a "return to throwing" therapy program. During this program, therapists review proper biomechanics of throwing and provide 3–5-min rest breaks between throwing activities. Once full strength has been regained and the athlete is asymptomatic, they may return to full activity [12].

Contact Athletes

Contact and collision sports, such as ice hockey, football, basketball, and soccer, put players at high risk of musculoskeletal injuries. The shoulder is the most common musculoskeletal injury seen. More specifically, acromioclavicular (AC) joint trauma is the most common injury (32–41%), and shoulder dislocation is the most common cause of playtime loss [13].

Acromioclavicular Joint Trauma

Pathology

The AC joint is formed by the acromion (of the scapula) and the clavicle. This joint is stabilized by the AC ligament in the AP plane and the coracoclavicular (CC) ligaments (in the vertical plane [13]. Typically, traumatic injury to the superior lateral

shoulder while the arm is adducted can cause an AC joint sprain or, in more severe cases, AC and CC rupture. AC separation secondary to trauma will typically cause the acromion and scapula to move inferior to the clavicle, increasing AC ligamentous strain. This pressure causes the AC ligament to stretch and can eventually lead to CC ligament sprain [14].

The Rockwood classification system is used to classify AC injuries: Type 1 is a sprain isolated to the AC ligament. Type 2 is a torn AC ligament and a sprain of the CC ligament [13]. Types 1 and 2 are considered incomplete AC joint injuries and are twice as likely to occur than the more severe AC joint injuries [15]. Type 3 involves complete injury to both AC and CC ligaments, causing the scapular end of the clavicle to displace superiorly. Type 4 results from posterior displacement of the clavicle through the trapezius along with AC and CC ligament disruption. This level of injury is typically the result of a high-energy collision as in snowboarding or direct trauma as from a baseball bat. Type 5 occurs when both AC and CC ligaments are torn along with muscular and fascial damage.

Clinical Presentation

Patients with an AC joint injury will typically present after sustaining a trauma directly to the area. AC joint pain is localized to the top of the shoulder, but it can also refer to the base of the neck and trapezius [16].

Physical Exam

On inspection of the shoulder, bony deformity may imply AC joint separation or clavicular fracture. Tenderness to palpation along the AC joint is expected. Athletes with AC joint injuries will have limited shoulder flexion, adduction, and internal rotation. Touching the unaffected shoulder will elicit the pain due to pressure placed on the AC joint. There are several special tests to be performed on physical examination to confirm AC joint dysfunction. These include the cross-body adduction test, the AC shear test, and the active compression test [17].

In type 1 injuries, there may be mild swelling on exam, and moving the affected arm across the body will elicit pain. Type 2 injuries will show substantial swelling of the acromion. A visible AC joint deformity along with CC tenderness will be seen in type 3 injuries. In type 4 injuries, the posteriorly displaced clavicle is palpable and may affect a patient's breathing. Type 5 AC injuries cause a severe disruption to the supportive structures of the shoulder which cause the shoulder to translate inferiorly. The superior displacement of the clavicle may even break the skin or cause ischemia [15].

Diagnostic Studies

When AC joint pathology is suspected, shoulder radiographs are ordered and should include AP shoulder, AP glenoid, scapular Y, and Zanca views. The Zanca view is specific for the AC joint. It is taken with the arm in an abducted position, and the beam is directed at a 30-degree cephalic tilt (pointing down the AC joint) [18]. Another diagnostic tool that can be used is a bone scan, on which a painful joint will show increased metabolic activity. If surgical treatment is indicated, an MRI of the shoulder is also obtained to rule out other pathologies. Finally, a diagnostic ultrasound-guided lidocaine injection can be used. While an injection can be done by palpation of bony landmarks, ultrasound-guided injections have shown improvements in symptoms and range of motion for up to 6 months longer than palpation-guided injections [19].

Treatment

Type 1 and type 2 injuries are treated conservatively with immobilization, oral anti-inflammatories, ice, and physical therapy. Physical therapy will include range-of-motion exercises and strengthening of the muscles of the rotator cuff. If conservative treatment initially fails in type 3, surgery is indicated. In types 4 and 5 injuries, surgery is always indicated. Of note, glucocorticoid injections can be used for chronic pain management.

Return to Play

Physical therapy alone should lead to complete healing within 6–10 weeks in type 1 and type 2 injuries and 6–12 weeks in type 3 injuries. Exercise is increased gradually over the weeks, initially starting with gentle range-of-motion exercises and ending with full-strength training without pain. Return to play is limited by pain [20].

Shoulder Dislocation

Pathology

Even though only 25% of the humeral head articulates with the glenoid, the glenohumeral joint provides the shoulder with a large range of mobility. The shallow joint, along with the small point of contact, increases the chances of shoulder dislocation. A dislocation may occur in the anterior, posterior, or inferior direction. Anterior dislocation results from a trauma sustained to an abducted, externally rotated, and extended shoulder. Posterior dislocation results from a blow from the anterior to the posterior direction or from strong muscle contraction (electrocution or seizure). Finally, an inferior dislocation is typically caused by axial loading of an abducted shoulder or hyperabduction [21]. Sports that involve the action of arm tackling a player that is running, like in football or blocking a basketball shot, may cause an inferior dislocation [22].

Anterior shoulder dislocations are associated with fractures known as Hill-Sachs deformities, Bankart lesions, and greater tuberosity fractures. The Hill-Sachs lesion appears on X-ray as a cortical depression deformity along the humeral head [23]. Bankart lesions occur due to bony fragment avulsion within IGHL complex [24]. Posterior shoulder dislocations are associated with tuberosity and surgical neck fractures, reverse Hill-Sachs lesions, and labral and rotator cuff pathologies [25].

Physical Exam

The appearance of a dislocated shoulder on physical examination varies depending on the direction in which the humerus is avulsed. If the shoulder is dislocated anteriorly, the arm at rest will be in an abducted and externally rotated position. On examination, the AC joint may be more prominent in thinner individuals. Shoulder dislocation in any direction may affect the axillary nerve, which can manifest as abnormal sensation along the superior lateral surface of the shoulder and may progress to deltoid weakness [26]. When the shoulder is posteriorly dislocated, exam findings will include a notable prominence posteriorly with a flattened anterior surface. At rest, the patient will hold the arm in the adducted and internally rotated position. If the shoulder is dislocated inferiorly, the athlete will have difficulties with shoulder adduction; therefore, at rest, the forearm is usually draped over the head [27].

Diagnostic Studies

When a shoulder dislocation is suspected, an initial X-ray (AP, scapular Y, and axial views) should be ordered to confirm the diagnosis and rule out fractures. An anteriorly dislocated shoulder will show a humeral head in the subcoracoid position on the AP view and positioned medial to the "Y" in the scapular view. The axillary view is useful in distinguishing a true dislocation from a false dislocation (when the humerus is fractured) [27]. In a posteriorly dislocated shoulder, AP X-ray will show the humeral head internally rotated, hiding the tuberosities resulting in a circular appearance known as "light bulb sign." In addition, the distance between the medial humeral head and the anterior glenoid rim may be widened in posterior dislocations. Of note, an inferior dislocation will show the humeral head below the coracoid as well; however, the shoulder will be in an abducted position [28]. If the location of the humeral head cannot be identified on X-ray, a CT scan can be used. In addition, a CT scan can be used to visualize associated fractures. If there is a suspicion for axillary artery injury, a CT angiogram is indicated. MRI arthrograms can be used to visualize associated lesions, such as the Bankart tear (Fig. 7.4),

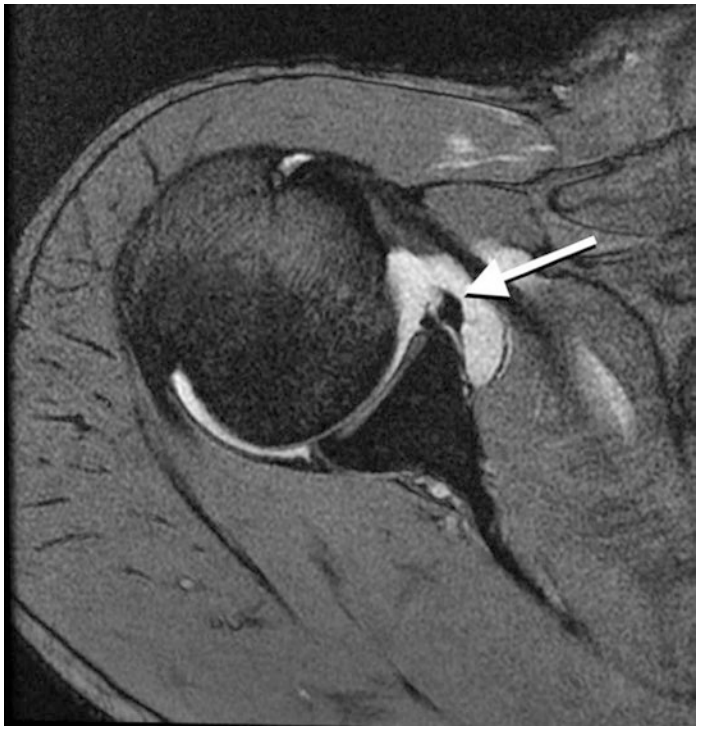

Fig. 7.4 Bankart lesion (a tear that happens in the lower rim of the labrum) on MRI arthrogram (white arrow) [13]

the reverse Bankart lesion, and the reverse Hill-Sachs lesions (Fig. 7.5) [29].

Treatment

Shoulder reduction is the first-line treatment for a dislocation. The risks are rare, but they can include humeral fracture, rotator cuff injury, or axillary neurovascular injury. In elderly patients, the risks are higher, and therefore, an orthopedic consultation may be

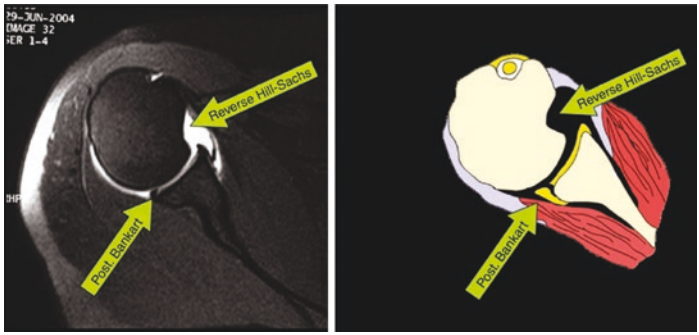

Fig. 7.5 Reverse Bankart lesion (detachment of the posteroinferior labrum with avulsion of the posterior capsular periosteum) and reverse Hill-Sachs lesion (fracture of the anteromedial aspect of the humeral head) on MRI arthrogram [13]

indicated. Acute, anterior, recurrent, or nontraumatic shoulder dislocations can be reduced without anesthetic. Following the reduction, X-rays are done to determine success [30]. Once the shoulder is in place, it is immobilized using a sling, and the patient is instructed to follow up within 1 week. Length of immobilization depends on age; if they are under the age of 30 years old, immobilization should last 3 weeks. If the patient is over the age of 30 years old, early mobilization at 1 week is recommended to avoid stiffness. Irreducible dislocations and Bankart fractures are indications for surgery.

Return to Play

During the immobilization phase, gentle range-of-motion exercises are advised while avoiding abduction and external rotation. Eventually, active-assisted range-of-motion and isometric exercises are initiated by therapists. Barring any complications, athletes may return to play with limitations at 12 weeks and may return to full activity at 16 weeks [31].

Acute Brachial Plexus Injury

Pathology

In contact sports, injury to the shoulder can result in neurovascular injury. There are three known mechanisms of upper trunk (C5, C6) brachial plexopathies in relation to the shoulder. One is a traction injury, where the neck is forced laterally away from a depressed shoulder. The second is a direct trauma to the supraclavicular fossa. The third is neck hyperextension and side bending, resulting in nerve compression [32]. This last brachial plexus injury, also known as a "burner" or "stinger," is a result of neck or shoulder trauma.

Clinical Presentation

Athletes with burner syndrome will typically present following neck and shoulder trauma with pain radiating down the affected arm. The pain is typically described as burning and surrounding the entire arm. Patients may also complain of numbness, heaviness, or sensitivity in the limb. Most often, these symptoms resolve within minutes.

Physical Exam

Patients suffering from an acute brachial plexus injury on inspection may hold their arm adducted at rest or may shake the affected limb. Over several weeks, deltoid atrophy is observed due to denervation. The cervical spine should be examined to rule out injury. Special tests such as the Spurling compression test can be used to rule out cervical nerve root compression. Multiple muscles can be affected by an injury to the upper trunk of the brachial plexus leading to weakness in shoulder abduction (deltoid, supraspinatus) and external rotation (infraspinatus), elbow flexion and supination (biceps brachii), and forearm pronation (pronator teres). Of note, the timeline of weakness development can vary from minutes to days [33].

Diagnostic Studies

Patients suspected of having nerve injury affecting the upper extremity should initially have a cervical spine X-ray (AP; lateral, in flexion and extension; and oblique views) to rule out spinal stenosis or cervical instability. An MRI should be done as well to rule out spinal cord dysfunction or nerve root compression. The gold standard for diagnosing peripheral nerve injury is an electrodiagnostic study. In an upper trunk brachial plexopathy, the electromyography findings can include fibrillation potentials, prolonged latencies, and positive sharp waves in C5 and C6 innervated muscles [34].

Treatment

Once diagnosed, burners are initially treated with physical therapy. Exercises include neck, shoulder, and upper extremity active range of motion and stretching. Stiffness and weakness predispose patients to burners, thus emphasizing the value of strength and mobility training. In addition, "chest-out" posturing is recommended to decrease nerve compression [34]. In contact sports, brachial plexus injuries can be prevented by using protective equipment such as shoulder pads.

Return to Play

Before an athlete can return to play, steps should be taken to educate them on proper biomechanics to avoid repeat injuries. Once symptoms have resolved and the range of motion of the neck and shoulder are back to baseline, they may return to contact sport. However, players cannot participate if neurological symptoms persist. Of note, recovery can take months and may even become a chronic issue [35].

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Shoulder Fractures

8

Rebecca Freedman and Jasmin Harounian

Clavicle Fractures

Introduction

Clavicle fractures are common injuries that are most frequently seen in both active males under the age of 30 and in the elderly population [1]. These fractures account for approximately 2.6–4% of all adult fractures and 44% of all shoulder fractures [1, 2]. Clavicle fractures also account for 10% of all sport-related fractures [3].

Mechanisms of injury include direct trauma to the clavicle or a fall onto the shoulder, such as from collisions in sports or motor vehicle accidents. Less commonly, clavicle fractures can result from a fall onto an outstretched hand. Young and active individuals typically sustain clavicle fractures by participation in contact sports, such as football and hockey, or sports with a risk of high-speed falls, such as skiing or bicycling. In comparison, the elderly population typically sustain clavicle fractures from simple falls directly onto their shoulder [3–6].

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The majority of clavicle fractures (about 80–85%) occur at the midshaft and are more likely to be displaced as compared with medial and distal third fractures [2, 4, 7]. The narrow cross section of the middle third of the clavicle makes it the most vulnerable to trauma [4]. It also lacks the strong ligaments and muscles which secure the medial and distal third of the clavicle [1]. Distal third fractures are the next most common type, seen in approximately 10–30% of clavicle fractures, and they tend to occur more in the elderly or osteoporotic individuals from falls [2, 4, 7, 8]. Medial third clavicle fractures are the least common.

There are several classification systems to assist in describing clavicle fractures, including Allman, Neer, Robinson, and Craig. These systems divide the clavicle into three segments and further divide into subgroups based upon various fracture factors including if the segment is displaced or comminuted.

The modified Neer classification system is widely used for distal clavicle fractures. It characterizes the fractures based upon the integrity of the coracoclavicular ligaments, consisting of the conoid and trapezoid ligaments:

Type I: Minimally displaced fracture lateral to the intact coracoclavicular ligaments, sparing the acromioclavicular joint.

Type IIA: Fracture is medial to the intact coracoclavicular ligaments with significant displacement of medial part of the clavicle.

Type IIB: Fracture is in between the torn conoid ligament and the intact trapezoid ligament with displacement of the medial clavicle.

Type III: Lateral to the intact coracoclavicular ligaments extending intra-articularly into the acromioclavicular joint.

Type IV: Physeal fracture in which the medial part of the clavicle is displaced superiorly as the periosteal sleeve alvuses from the inferior cortex and the coracoclavicular ligaments remain intact.

Type V: Comminuted fracture with medial clavicle displacement and a small inferior fragment attached to the intact coracoclavicular ligaments.

Clinical Presentation and Physical Exam

An individual may present with anterior shoulder pain after a known trauma. There may be an abrasion, swelling or ecchymosis, and both crepitus and tenderness upon palpation. Clavicle fractures present as an apparent deformity upon visualization, including skin tenting, which can indicate an impending open fracture. In midshaft clavicle fractures, the muscle attachments often cause the deformity seen after a fracture with subsequent clavicle shortening. The sternocleidomastoid muscle pulls the medial fragment posterosuperiorly, while the pectoralis major muscle and weight of the arm pull the lateral fragment inferomedially. Associated conditions, although rare, can include ipsilateral scapular fracture, rib fracture, and pneumothorax. A careful neurovascular exam is imperative to rule out any injury to the subclavian vessels or brachial plexus.

Diagnostic Studies

Diagnostic evaluation should consist of an anteroposterior (AP) radiograph ideally in the upright position and an AP radiograph with 15–20 degrees of cephalic tilt to help visualize the displacement of the clavicle. Occasionally, a stress view may be obtained, in which a 5–10 lb. weight is suspended on the ipsilateral wrist to determine the integrity of the coracoclavicular ligaments [4]. A CT scan can help further evaluate displacement, fracture type, articular extension, and clavicular shortening. CT scanning may be useful in identifying fractures of the medial clavicle.

Treatment

Treatment of clavicle fractures depends on both the location and the type of the fracture. Nonoperative care has traditionally been the treatment of choice for nondisplaced midshaft fractures. Sling immobilization for 2–4 weeks is recommended until there is evidence of clinical healing with an improvement in pain and

radiographic evidence of bony healing [3, 9, 10]. A figure-of-eight brace has fallen out of favor due to patient dissatisfaction, brachial plexopathies, and upper limb thrombosis [4, 10]. Fracture reduction is not recommended as no current evidence supports that fracture reduction is maintained or clinical outcomes are improved. After the period of immobilization, a rehabilitation program beginning with range of motion and progressing to shoulder strengthening at 6 weeks should be implemented.

Surgical intervention, either with plate fixation or intramedullary nailing, is indicated for skin tenting, open fractures, the presence of neurovascular compromise, Z-type fracture pattern, and a floating shoulder [7, 11]. Surgical treatment is now commonly preferred for displaced midshaft fractures and fractures with greater than 1.5–2 cm of shortening due to the high nonunion rate and functional deficits reported with nonsurgical treatment [7, 11]. Surgical treatment has been found to decrease the nonunion rate, shorten the time to union, lead to better short- and long-term functional outcomes, and improve return-to-play rates [9, 12]. Operative treatment in children and adolescents remains controversial, with recent evidence in favor of surgical care for active individuals who would benefit from quick restoration of normal anatomy and fixation [4].

Nondisplaced lateral fractures are generally managed with conservative care including sling immobilization for 2–4 weeks. Type IIA, IIB, and V distal clavicle fractures are considered unstable fractures requiring operative treatment. Operative intervention is routinely performed for displaced lateral fractures, especially in the athletic population, as high rates of nonunion and subsequent shoulder function impairment have been reported [13]. Medial fractures are mostly treated nonoperatively with a sling for immobilization followed by a rehabilitation program.

Return to Play

Most individuals with a clavicle fracture will return to sports activity at their pre-injury level of sport. The decision to return to sport includes clinical and radiographic evidence of fracture heal-

ing and full pain-free range of motion with full strength. However, return-to-play timelines vary. With nonoperative treatment, it is generally recommended to wait 6–12 weeks from the time of injury to return to activity [3]. Others recommend avoiding contact sports for a minimum of 4–5 months to allow clavicle healing [7]. Postoperatively, patients should remain in a sling for immobilization for 2–4 weeks with immediate gentle range of motion exercises followed by a rehabilitation program between 4 and 6 weeks [9]. Return to sport has been recommended anywhere from 6 to 12 weeks to 4 to 6 months [11, 14].

Scapula Fractures

Introduction

Scapula fractures are uncommon injuries to the shoulder girdle. These fractures account for less than 1% of all fractures and about 3–5% of all fractures of the shoulder girdle [15]. Scapula fracture locations include the coracoid, acromion, glenoid, scapular neck, and scapular body, with about 50% involving the body and spine of the scapula [16]. They typically occur with high-energy traumas, such as motor vehicle collisions, and are often associated with rib fractures, ipsilateral clavicle fracture, spine fracture, brachial plexus injury, lung injuries, and head injury [15, 17].

Clinical Presentation and Physical Exam

The most common symptoms of fractures of the scapula are severe pain, abrasions near the affected area, ecchymosis, swelling, and limited range of motion, particularly with abduction. Individuals with suspected scapula fractures should be examined standing or sitting. Inspect for obvious deformity or marked asymmetry. As shoulder girdle injuries are often associated with neurovascular injuries, a full and thorough assessment of the brachial plexus and distal perfusion must be performed. Particularly, the suprascapular and axillary nerves are at

increased risk for injury. If the time of presentation is more than 2 weeks after injury, an EMG can be performed to assess severity of injury and localize pathology to these nerves and the brachial plexus. Further, examination should include assessing for other associated injuries previously mentioned, especially those which are emergencies.

Diagnostic Studies

A true AP (Grashey view), scapular Y, and axillary lateral view radiographs are recommended. The axillary view is used to assess acromion and coracoid fractures. The Stryker notch view may be helpful to evaluate for coracoid fractures, too. A West Point lateral view can evaluate for glenoid rim fractures such as a bony Bankart lesion (fracture of the anterior-inferior glenoid cavity) or a reverse bony Bankart lesion (fracture of the posterior capsular periosteum).

A CT scan can assess for an intra-articular fracture or significant displacement. It allows for the evaluation of the size, location, degree of displacement, and humeral head position in relation to the glenoid fossa [16]. It can also be used for 3D reconstruction to better visualize displacement and assist in planning for surgical intervention.

Treatment

Treatment is dependent upon the location of the fracture within the scapula. The majority of scapular fractures are nondisplaced or minimally displaced and can be managed effectively with conservative treatment. Fractures of the scapular spine and body generally can be managed nonoperatively with excellent or good functional results [18]. This is largely due to the extensive muscular envelope which limits displacement of the scapular and facilitates healing. Conservative treatment typically consists of

pain control, immobilization with a sling for 2 weeks, and an early rehabilitation program. However, indications for surgical intervention vary.

Fractures of the glenoid fossa that result in articular displacement greater than 5 mm can increase the risk for developing posttraumatic degenerative joint disease [19]. As such, surgical treatment with open reduction and internal fixation is favored by some surgeons. Further, operative intervention is indicated if the glenoid fracture is associated with persistent or recurrent glenohumeral instability. Glenoid fossa fractures managed operatively have been found to have excellent or good results in the majority of the cases [18].

Surgical intervention should also be considered for significantly displaced scapular fractures. Displaced scapular neck fractures treated nonoperatively were found to have poorer functional outcomes, increased pain, decreased range of motion, and weakness [20, 21]. Operative treatment is recommended for all glenoid neck fractures with at least 1 cm of translation or 40 degrees of angulation in the AP plane of the scapula [16, 20, 21]. Isolated glenoid neck fractures with no involvement of the glenoid fossa can be managed nonsurgically with excellent results [18].

Another indication for surgical treatment is disruption of the superior shoulder suspensory complex in two different locations. This is made up of the glenoid, coracoid, acromion, distal clavicle, coracoclavicular ligaments, and acromioclavicular ligaments, which secure the upper extremity to the axial skeleton [19]. Without an open reduction and internal fixation, there may be malunion or nonunion and long-term functional impairments [19].

Return to Play

Conservative management should consist of immobilization with a sling for 2 weeks followed by early rehabilitation and range of motion with union expected at 6 weeks. Postoperatively, strength and endurance training can begin after 8 weeks, and return-to-normal activities can happen after 12 weeks [15].

Proximal Humerus Fractures

Introduction

Proximal humerus fractures are common osteoporotic fractures seen in the elderly after a low-energy fall. These fractures account for about 6% of all fracture types with a female-to-male ratio of 70:30 [22]. About 87% of proximal humerus fractures occur from falls from a standing height [6]. Less commonly, these fractures can happen in the younger population after a high-energy impact.

The most widely used classification system is the Neer classification. It is based upon four segments consisting of the greater tuberosity, the lesser tuberosity, the humeral head, and the humeral shaft. It characterizes proximal humerus fractures by the number of displaced segments with further categorization for articular fractures and dislocation. A fragment is considered displaced if it is separated more than 1 cm or angulated more than 45 degrees [23].

One-Part Fractures

These are fractures with no displaced segments regardless of the number of fracture lines or their location.

Two-Part Fractures

There is displacement of one segment which may be the greater or lesser tuberosity or the humeral head at the level of the anatomic or surgical neck.

Three-Part Fractures

The greater or lesser tuberosity is displaced as well as the surgical neck fracture.

Four-Part Fractures

In these severe fractures, all four segments meet the criteria for displacement. Of note, the articular segment is typically laterally displaced and no longer in contact with the glenoid. This carries a high risk of avascular necrosis.

Another separate category was added for valgus-impacted four-part fractures. The humeral head is rotated into a valgus position and forced downward between the greater and lesser tuberosities, which will splay outward. The articular surface maintains contact with the glenoid.

Additionally, the AO/OTA classification system is commonly used, which focuses on the progressive severity of the fracture pattern. There are three main fracture types which are then categorized based upon the degree of displacement, impaction, and dislocation.

An additional fracture type of the proximal humerus includes a Hill-Sachs lesion, defined as a cortical depression in the posterolateral head of the humerus, resulting from the forceful impact of the humeral head against the anteroinferior glenoid rim. This lesion is typically associated with an anterior shoulder dislocation.

Clinical Presentation and Physical Exam

On examination, there may be ecchymosis, abrasions, or edema near the affected area. Gross deformity or the presence of a subacromial sulcus sign may suggest dislocation of the humeral head. Evidence of an open fracture or skin tenting should be quickly identified. A thorough neurovascular exam should be conducted with particular attention to the axillary nerve. Examination should also assess the function of the elbow, wrist, and fingers.

Diagnostic Studies

A true AP view of the glenohumeral joint, scapular Y view, and axillary view radiographs of the glenohumeral joint should be obtained ideally with the patient in an upright position. A CT scan is recommended for complex fractures or when fracture lines are not well visualized. MRI can be helpful in assessing rotator cuff

integrity and other soft tissue injuries that may accompany a fracture. Rotator cuff pathology is frequently associated with proximal humerus fractures, both at the time of injury and 1 year later [24]. Additionally, a Stryker Notch view can assess for a Hill-Sachs lesion.

Treatment

Recommendations for the treatment of proximal humerus fractures vary and are still evolving. In general, minimally displaced fractures are treated conservatively. Displaced, comminuted, or angulated fractures are treated operatively; however, the type of intervention can be challenging. Operative interventions include percutaneous fixation, nailing, plating, and arthroplasty. Physiologic age, determined by bone quality and social independence, is more important than chronological age when determining treatment options and outcomes [25, 26].

About 50% to 66% of all proximal humerus fractures are minimally displaced, with the majority of all proximal humerus fractures involving the greater tuberosity or surgical neck [6, 27]. These fractures typically respond well to conservative treatment consisting of a sling for 4–6 weeks followed by early rehabilitation and physical therapy. Isometric, pendulum, and passive range of motion exercises should be initiated within a few days of injury [25]. Active strengthening exercises can begin once healing is evident, usually by 4–6 weeks. Minimally displaced injuries are at low risk for further displacement, nonunion, or avascular necrosis; however, patients are at risk for loss of range of motion and posttraumatic arthritis [28]. Isolated, minimally displaced greater tuberosity fractures can take up to a year for full recovery [25]. If the greater tuberosity is displaced more than 5 mm, surgery is indicated as it can result in impingement with loss of abduction and external rotation.

Surgery can be considered for two-part surgical neck fractures with significant displacement in patients with adequate bone quality. Surgical techniques include percutaneous wiring, nailing, and plating. Of the three, percutaneous wiring has been associated

with superior outcomes [29]. Displaced two-part tuberosity fractures may benefit from surgical fixation. Fractures of the lesser tuberosity more commonly occur in conjunction with a posterior dislocation. If fragments are large and displaced or involve the articular surface, fixation is warranted.

Three- and four-part proximal humerus fractures can be treated nonoperatively with closed reduction. In general, this leads to poor functional results, although patients have reported limited pain and overall satisfaction [26]. Despite current evidence suggesting satisfactory outcomes with conservative management, operative care has been increasingly performed with locking plate reduction or arthroplasty in patients with severe fractures [30]. However, outcomes between operative and nonoperative treatments in displaced two-, three-, or four-part fractures have not been found to be significantly different after 5 years [31]. Ultimately, treatment for displaced fractures should consider a patient's bone quality, surgical candidacy, level of independence, lifestyle, and expectations.

Return to Play

Treatment options and modalities should place emphasis on the patient's characteristics and goals. For those who are active or participate in athletics, sports activities after surgical treatment are close to pre-injury level, with some avoiding overhead sports [32].

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Medical Causes of Shoulder Pain

9

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Cervical Spine Causes of Shoulder Pain

One of the most common causes of shoulder pain is referred pain from the cervical spine. In fact, several studies have shown anywhere from 3.6% to 52% of patients who presented with shoulder pain were found to have significant cervical pathology [1, 2]. Further complicating the diagnostic process is that shoulder and neck pains are often concurrent complaints. As such, cervical pathology is important to consider when assessing shoulder pain.

Pathology

The pathophysiology of referred pain is in part due to the diffuse termination of spinothalamic pain fibers. The fibers involved in pain sensation first terminate in the dorsal root ganglia, and then the second-order pain fibers travel up the spinal cord through the paleospinothalamic pathway and terminate diffusely in the brain.

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Due to the diffuse termination of these fibers, nerve fibers from different areas of the body can synapse to the same second-order neurons. This process can create referred pain as neuroconnections from different parts of the body overlap [3].

Understanding the anatomy of the cervical spine and shoulder is important for understanding referred pain in these regions. Cervical intervertebral discs are innervated by the sinuvertebral nerve and have both autonomic and somatic innervations. The cervical facets contain nociceptive fibers from the medial branches of the dorsal rami. Meanwhile, the shoulder region is innervated by the suprascapular and axillary nerves, which originate from the superior trunk of the brachial plexus (C5, C6). Branches of the suprascapular nerve provide sensation to the shoulder joint, acromioclavicular joint (AC joint), subacromial bursa, and posterior capsule of the shoulder. The axillary nerve provides sensation to the inferior capsule and contributes to the sensation of the anterior capsule along with the lateral pectoral (C5, C6) and subscapular (C5, C6) nerves [3].

Classic pain referral patterns have been described through previous landmark studies. Stimulation of the paravertebral tissue from C3 to C6 referred to the shoulder, while stimulation of the cervical facets at joints of segments C2–C3 to C6–C7 referred pain to the posterior shoulder and trapezius. C5 and C6 radiculopathy has been found to refer to the suprascapular shoulder region with C8 radiculopathy referring to the scapular region [3]. The figure below illustrates the classic pain referral patterns described in this paragraph (Fig. 9.1) [4].

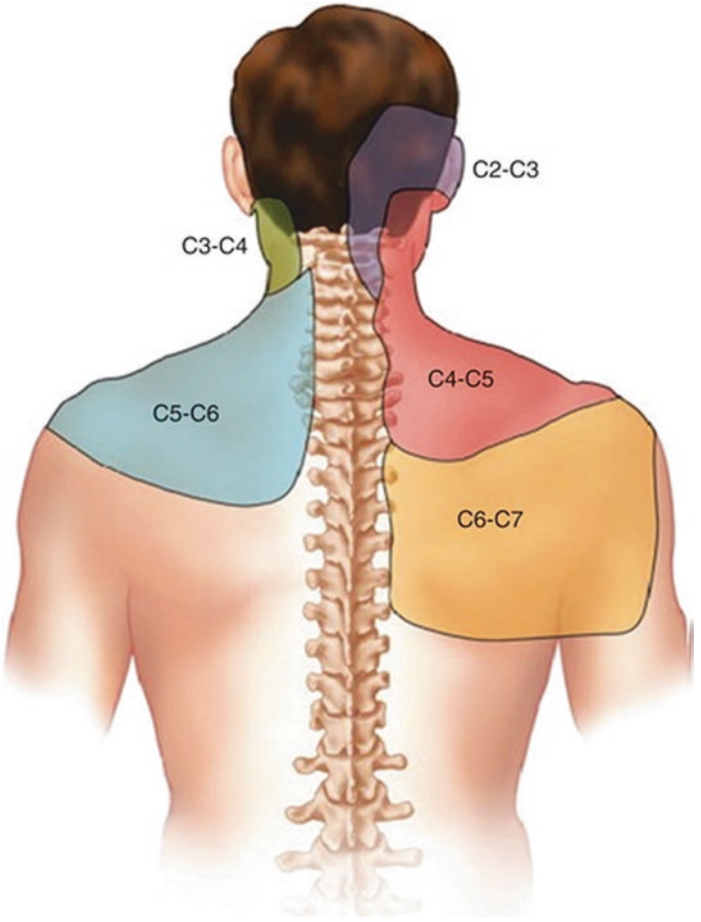

Fig. 9.1 This image demonstrates class pain referral patterns associated with cervical facet pain at different spinal levels [4]

History and Clinical Exam

Obtaining the proper clinical history and exam is important for differentiating between shoulder and cervical causes of pain. Findings that can increase suspicion for cervical pathology include concurrent neck pain, pain with cervical range of motion, and radicular pain originating from the neck. Common cervical conditions that lead to shoulder pain are identified and discussed below in Table 9.1.

Cervical radiculitis and radiculopathy are a common cause of shoulder pain. These patients typically present with neck pain that radiates to the upper extremity in a dermatomal pattern that depends on the affected level. Weakness can also be present with or without pain or sensory changes. Physical exam should include inspection for posture and signs of muscle atrophy; palpation for deformities, tenderness, or muscle spasms; and cervical range of motion to assess for concordant reproduction of symptoms and

Table 9.1 Common presentations of shoulder vs. cervical pathology are compared above [3]

	Shoulder pain	Cervical/referred pain
Pain pattern	Localized to shoulder, worse at night	Radiates from the neck/may radiate distal to the elbow
Weakness	Rotator cuff muscles	Myotomal distribution based on the affected levels
Sensory changes	Not normally seen	Follows dermatomal patterns, often radiates past the shoulder
Reflexes	Normal reflexes	Possibly abnormal reflexes
Special tests	Positive drop arm test, positive Neer's/ Hawkins tests (impingement signs), and positive shoulder abduction test (Bakody's test)	Positive Spurling's and arm squeeze tests, negative shoulder abduction sign (relief of pain)
Electrodiagnostic test	No abnormalities	EMG and nerve conduction study changes based on affected nerves

restriction of movement. Neurological assessment may reveal muscle weakness, sensory changes, and diminished reflexes, which can help localize the nerve roots involved. Special testing, such as Spurling's test, may help confirm cervical spine nerve irritation or compression. This test is performed by rotating the patient's head to the affected side and then laterally extending the cervical spine to place pressure on the exiting nerve roots of that side. A positive test occurs when the patient's radicular symptoms are reproduced. This test has a specificity of 93% and a sensitivity of 30% [5]. Other special tests include the shoulder abduction test, the neck distraction test, and the Valsalva maneuver test.

Cervical facet or uncinate arthropathy can also present with neck pain that radiates to the arm or back in a referred pain pattern based on the affected cervical joint/s involved [6]. Patients may present with a history of hyperextension injury to the neck or some form of trauma, although this is not always the case. Physical exam may reveal decreased cervical range of motion associated with axial and referred pain patterns as illustrated in Fig. 9.1.

Patients with cervical stenosis may also present with shoulder pain. Neurogenic symptoms can manifest as numbness, weakness, and discomfort radiating from the neck down to the shoulder and arm. Other pain patterns to be aware of include: shock-like paresthesias, clumsiness of fine movements, frequent falls, wide-based stance, and intermittent gait incoordination. Physical exam again involves neurologic evaluation of the upper extremities. Motor weakness, sensory changes, and abnormal reflexes can all be present. Patients can have both upper and lower motor neuron signs. Bowel and bladder retention is more likely to occur rather than incontinece with bladder involvement being more common [7].

Cervical myelopathy occurs when there is compression of the cervical spinal cord. This can be caused by a variety of conditions. The most common causes are cervical spondylosis, and degeneration of the cervical discs. On the same continuum, cervical central canal stenosis can also lead to myelopathy and presents similarly with progressive weakness, sensory changes, and radicular-type pain. Cervical myelopathy often presents with upper motor neuron signs, decreased fine motor skills, poor balance, and/or bowel

and bladder incontinence [5]. The key physical exam findings are hyper-reflexia, upper motor neuron signs and difficulty with tandem gait; a full neurologic exam should be performed.

Brachial plexus injuries can also result in shoulder pain. The history for a brachial plexus injury may include trauma, most commonly vehicular accident, that results in damage to the nerves of the brachial plexus [8]. Other causes such as Parsonage Turner syndrome, tumor, history of radiation therapy, and anatomic variants in the region shoulder also be considered. The clinical exam here should include detailed assessment of muscle strength, sensation, reflexes, and tandem gait as these can help identify which nerve(s) are potentially affected.

Syringomyelia is a condition in which a syrinx, or a fluid-filled cavity in the spinal cord, develops which results in neurologic changes. Symptoms of the syrinx depend on the levels affected. Patients commonly present with non-radicular paresthesia or hyperesthesias and loss of pain and temperature sensation with preserved touch and vibratory sense. There can also be significant non-radicular neuropathic pain in the affected dermatomes [9]. These symptoms generally present in a "cape-like" pattern over both extremities, and if the cervical spinal cord levels are affected, patients can have pain in the shoulders that can mimic shoulder pathology. This condition should especially be considered in those with prior spinal cord injury or prior cervical spine surgery. Of note, Charcot joint of the shoulder or neuropathic arthropathy can develop due to the lack of sensation if the syrinx involves cervical levels affecting sensation to the shoulder joints. The lack of sensation prevents normal guarding mechanisms resulting in repetitive trauma and ultimately severe osteoarthritis of the joint which can be painful if sensation is restored [10].

Diagnostic Steps and Treatment

The workup for the above conditions is similar and an X-ray is usually the best place to start. An AP, lateral, and possibly oblique cervical spine views can detect fractures, cervical stenosis, cervical facet arthropathy, osteophyte formation, ossified posterior lon-

gitudinal ligament, and instability if flexion/extension views are also ordered. It is also beneficial to get an MRI if suspecting the above conditions. MRI is the primary imaging modality for cervical radiculopathy as it is the best imaging study for observing potential nerve impingements and other soft tissue involvements. MRI is also useful in cases of cervical myelopathy, syringomyelia, and brachial plexus injury. EMG and nerve conduction studies can assist in diagnosis by providing objective data of which muscles or nerves are affected. Please note, however, that an electrodiagnostic study may be read as "normal" if there is a disc herniation that compresses a spinal nerve proximal to the dorsal root ganglion.

The treatment options for the above conditions often vary based on severity. Conservative management is preferred and includes nonsteroidal anti-inflammatory drugs (NSAIDs) as first-line management with gabapentin, pregabalin, selective serotonin reuptake inhibitors (SSRIs), serotonin and norepinephrine reuptake inhibitors (SNRIs), and tricyclic antidepressants (TCAs) being other options due to their neuropathic pain management properties. Physical therapy and occupational therapy are also indicated for patients with cervical radiculopathy, cervical myelopathy, cervical facet arthropathy, brachial plexus injury, and syringomyelia. If conservative measures are not successful, injections can be performed. Fluoroscopy-guided epidural injections can be performed for cervical radiculopathy patients, whereas medial branch blocks, intra-articular facet joint injections, and radiofrequency ablations can be performed for facet-mediated pain. For severe cases that do not respond to conservative measures or for those that require urgent intervention, surgical options can be explored.

Rheumatologic Causes of Shoulder Pain

Rheumatologic cases are often known to cause various presentations of joint pain, with the shoulder being a common place of manifestation. Table 9.2 summarizes the common rheumatologic causes of shoulder pain, their presentations, diagnostic workups, and treatments.

Table 9.2 This table summarizes common rheumatologic conditions that can present with shoulder pain

	Pathology	History	Physical exam	Diagnostic workup	Treatment
Gout	Gout is due to hyperuricemia, which is the increased deposition of monosodium urate in tissues. Monosodium urate is the end product of purine metabolism. Gout is associated with a diet high in meat, seafood, and alcohol. Certain medications, hypertension, kidney disease, obesity, diabetes mellitus, hyperlipidemia, and cardiovascular disease are also associated with gout [11]	Typically presents as severe monoarthritis at first onset with severe pain, warmth, swelling, and erythema within 12 h. 50% of first flares present in the first metatarsophalangeal joint. Other common sites include the feet, ankles, knees, elbows, wrists, and fingers Chronic gout, however, can become additive and progress in an ascending pattern which can include the shoulders. Destructive arthritis with degenerative changes, bony erosions, and tophi can occur in chronic gout [11]	Physical exam should include inspection for erythema and swelling. Palpation should also be performed to assess warmth and tenderness. Range of motion should also be assessed	The gold standard of diagnosis for gout includes the presence of negative birefringent, needle-shaped crystals in aspirated synovial fluid of an affected joint. Ultrasounds can also show crystal deposition in the affected joint [11]	Referral to a rheumatologist should be initiated. Rheumatologists use nonsteroidal anti-inflammatory drugs (NSAIDs) and various anti-gout medications such as allopurinol, colchicine, and probenecid [11]

Calcium pyrophosphate deposition disease (CPPD)	CPPD, commonly referred to as pseudogout, is caused by the deposition of calcium pyrophosphate crystals in articular and periarticular tissues [12]	Typically seen in patients older than 65 years of age, CPPD often presents as an acute monoarticular or oligoarticular arthritis with abrupt pain, warmth, and swelling. CPPD is often triggered by a concurrent medical or surgical condition. Polyarticular and bilateral arthritis can also be present in acute CPPD In chronic CPPD, there is a pattern of bilateral, symmetric, and deforming inflammatory polyarthritis. While frequently affecting the wrists and metacarpophalangeal joints, any joint can be affected, including the shoulders [12]	Similar to the exam for gout and should include inspection, palpation, and range of motion	Aspiration of synovial fluid in an affected joint yields rhomboid-shaped crystals with a weak positive birefringence On X-ray, crystal deposition can be seen in the fibrocartilage of the joints. There can also be calcifications of the hyaline cartilage and tendons. Ultrasound can reveal crystal deposition in peripheral joints which appear as thin hyperechoic bands within hyaline cartilage [12]	Once again referral to rheumatologist is recommended. Commonly used medications for CPPD include NSAIDs, glucocorticosteroids, colchicine, methotrexate, hydroxychloroquine, and anakinra [12]
Systemic lupus erythematosus	Lupus erythematosus is an autoimmune disease with an unclear exact etiology. There is an abnormal production of antibodies by B cells, but it is unclear if the B cells themselves are abnormal [13]	Lupus erythematosus is well known for its diverse presentations that are often patient-dependent. Generalized fatigue is one of the most commonly seen symptoms. Patients can also present with pulmonary, dermatologic, musculoskeletal, renal, central nervous system, or cardiovascular symptoms. The condition is more common in women and minority populations. Lupus often presents in early adulthood, but it can be diagnosed as late as 65 years of age Over 90% of patients with lupus erythematosus develop polyarthralgia or polyarthritis. If severe pain is present in an individual joint in these patients, there should be concern for avascular necrosis, which is most common in the hip [13]	Full musculoskeletal exam of the affected joints should be performed. Focal exams of other pertinent systems should also be performed	Laboratory testing, including a complete blood count, chemistry panel, and urinalysis, should be performed. Serologic testing should include antinuclear antibodies, anti-Rh_o, anti-La, anti-RNP, anti-Sm, anti-dsDNA, and antiphospholipid antibodies to help confirm the diagnosis. If avascular necrosis is suspected, an MRI can be obtained for early detection [13]	Lupus erythematosus is a complicated condition, and a rheumatology referral should be placed. Commonly used medications for lupus erythematosus include NSAIDs, hydroxychloroquine, glucocorticoids, methotrexate, and azathioprine [13]

(continued)

Table 9.2 (continued)

	Pathology	History	Physical exam	Diagnostic workup	Treatment
Rheumatoid arthritis	Rheumatoid arthritis is an inflammatory disease that is not fully understood. It is believed that genetics and environmental factors play a role in its development. Rheumatoid arthritis causes joint damage through the proliferation of synovial macrophages and fibroblasts. The proliferation occurs from a triggering event, which is thought to be due to autoimmune or infectious processes. There is then new blood vessel formation in the joint. These vessels then become occluded by small clots or inflammatory cells, which results in inflamed synovial tissue. Risk factors include female sex, family history, older age, silicate exposure, smoking, and high coffee intake [14]	Patients with rheumatoid arthritis typically present with pain and stiffness in multiple joints, but up to one third can present with monoarticular or oligoarticular symptoms. Typical progression is generally weeks to months and begins monoarticularly with a prodrome of anorexia, weakness, or fatigue. Typical joints affected are the wrists and metacarpal phalangeal joints, but symptoms can develop in any joint. Subcutaneous nodules, or rheumatoid nodules, are often present surrounding the affected joints or on extensor surfaces or bony prominence. Stiffness is worse in the mornings and usually lasts at least 45 minutes upon trying to initiate movement. Low-grade fever, malaise, or other systemic complaints can also arise [14]	Inspection and palpation should be performed. On inspection, swelling can be present and subcutaneous nodules could be observed. Atrophy may be observed around inflamed joints. Patients often hold joints in flexion to minimize pain. Palpation of the affected joints typically feels warm and boggy with associated tenderness in the joint. Epitrochlear, axillary, and cervical lymph nodes may be palpated. Weakness is often out of proportion to pain on exam [14]	Laboratory workup includes complete blood cell count with differential, rheumatoid factor, erythrocyte sedimentation rate, and C-reactive protein. Baseline renal and hepatic functional panels are also recommended for medication management guidance X-rays and other imaging studies may be normal initially, but often progress to show erosions along the bones of the affected joint [14]	Joint destruction can begin within a few weeks of symptom onset so initiation of treatment should begin quickly. Rheumatology referral is recommended with the above labs ordered to assist with medication management. Treatment usually consists of NSAIDs for pain, selective use of a glucocorticoid, and the initiation of a disease-modifying antirheumatic drug (DMARD) [14]

Fibromyalgia	Fibromyalgia is an idiopathic condition of unknown pathology that is generally considered to be a diagnosis of exclusion. The disorder often clusters in families, suggesting a genetic component. Environmental and psychological factors are also thought to play a role in its development. Some theories of etiology include central sensitization or hypothalamic-pituitary-adrenal axis dysregulation [15]	Fibromyalgia is more common in women 20–50 years old. However, it does occur in males and in patients of all ages, including children and adolescents. Patients often have comorbid somatization disorder, anxiety, and/or depression Symptoms of fibromyalgia include pain at multiple sites, fatigue, poor sleep, low back pain with radiation to buttocks and legs, and tightness in the neck or shoulders. The pain is often described as burning or gnawing soreness, stiffness, or aching. The symptom of stiffness is often worse in the morning and improves throughout the day. Reports of paresthesia without significant neurologic findings and subjective joint swelling without objective swelling are common features in fibromyalgia Patients often complain of cognitive issues such as memory problems, difficulty with word recall, and poor vocabulary. Headaches, light-headedness, dizziness, anxiety, and depression are other common complaints. Poor sleep, stress, and weather changes often aggravate symptoms, while moderate physical activity, adequate sleep, and relaxation often improve symptoms. Relevant social, personal, and family history is important in making the diagnosis [15]	Inspection and palpation of the areas of pain should be performed. Focused neurologic and musculoskeletal exams should also be performed. There are specific examination recommendations by the American College of Rheumatology that can be used to examine for fibromyalgia	Diagnosis of fibromyalgia is a clinical diagnosis. If in doubt, workup to rule out other rheumatologic disorders, like polymyalgia rheumatica, can be performed. Hypothyroidism can also mimic fibromyalgia, and the diagnosing clinician should consider a thyroid panel if there is any suspicion of hypothyroidism [15]	Treatment of fibromyalgia can be difficult. Non-pharmacologic treatments include exercise, which has been shown to be one of the best strategies for symptom relief in fibromyalgia patients. Other non-pharmacologic options include cognitive behavior strategies, patient education, and alternative therapies like acupuncture and biofeedback Pharmacologic management includes antidepressants, specifically TCAs. TCAs can help improve pain, sleep quality, and overall mood. SNRIs, like duloxetine, may also be beneficial in improving symptom. Pregabalin and cyclobenzaprine have also shown potential for symptomatic improvement in this patient population [15]

(continued)

Table 9.2 (continued)

	Pathology	History	Physical exam	Diagnostic workup	Treatment
Polymyalgia rheumatica	Polymyalgia rheumatica is an inflammatory condition of unknown cause that affects soft tissues around the joints [16]	Patients are typically elderly and present with profound pain and stiffness in the neck, bilateral shoulders, and hips, but without weakness. The painful effects of polymyalgia rheumatica on the proximal muscles of the extremities are classic for the conditions. The symptoms are worse in the morning and improve throughout the day. The severity pain and stiffness can sometimes significantly limit mobility and transfers [16]	Inspection and palpation should be performed, with palpation likely resulting in pain in the affected areas. Range of motion should also be tested, taking the likelihood of significant pain into account during the exam	Laboratory testing should include complete blood count, chemistry panel, erythrocyte sedimentation rate (ESR), and C-reactive protein. ESR and CRP are typically elevated. Other general inflammatory markers, such as plasma viscosity (PV), will also typically be elevated. Although not commonly used for diagnosis, ultrasound and MRI studies can reveal inflammation including synovitis and bursitis [16]	Urgent treatment is generally recommended as polymyalgia rheumatica can become debilitating quickly. A course of 15 mg of prednisolone daily is typically used at the onset. Patients should show a marked improvement within a couple of days. If the patient's symptoms do not improve within a few days, alternative diagnosis should be considered After initial treatment/ response, a slow taper is initiated. A reduction of prednisolone by 2.5 mg every 2–4 weeks is first done until a dose of 10 mg daily is reached. Then, the dose is decreased by 1 mg every month until cessation. If there is a flare, the patient can return to the previous effective dose at preventing flares. There is currently not a reliable alternative to glucocorticoid management of polymyalgia rheumatica [16]

Axial spondyloarthritis	Ankylosing spondylitis is the most common spondyloarthropathy. The exact mechanisms behind its development are unknown, but it is related to the prevalence of HLA-B27 [17]	Ankylosing spondylitis is most commonly seen in white males ages 15–40 and has an insidious onset. It typically presents as dull low back pain that radiates to the gluteal regions. Pain is worse in the morning and improves with activity. With time, the axial arthritis can progress, ascending from the sacroiliac joints to the cervical spine. Poor spinal mobility results in flattening of lumbar lordosis, increased thoracic kyphosis, and hyperextension of cervical spine. Early in the course of the disease, patients can develop arthritis in the hips and shoulders Patient often have extra-articular symptoms which can include fatigue, anorexia, mild fever, anterior uveitis, aortic and mitral root dilation, lung fibrosis, enthesitis, prostatitis, amyloidosis, IgA nephropathy, and oral ulcers [17]	Physical exam should include a complete musculoskeletal exam with a focus on the joints involved in the symptoms. Schober's test assesses spinal mobility. In this test, the patient's back is marked over the L5 spinous process and 10 cm above this point. When bending forward, the distance between the two marks should increase by 5 cm or more. An increase of less than 5 cm is indicative of decreased range of motion of the lumbar spine As ankylosing spondylitis can have many extra-articular symptoms, auscultation of the heart and lungs should be performed. The patient's eyes should also be inspected. If the patient has any pain at possible tendon sites, such as the ankle, feet, or patella, these areas should be examined as well	While the presence of the HLA-B27 gene is not diagnostic, as it can be present in patients without ankylosing spondylitis, gene assay for the HLA-B27 gene can be done. Erythrocyte sedimentation rate and C-reactive protein are elevated in up to 50–70% of patients Radiologic features on X-ray include bilateral symmetric sacroiliitis with progression from sclerosis to erosion to total ankylosis or fusion of the sacroiliac joints. Squaring of vertebral bodies can occur due to enthesitis with eventual progression to the ascending formation of marginal syndesmophytes. The ascending progression of syndesmophytes is due to the ossification of the annulus fibrosus, which may bridge the intervertebral space. This results in the classic "bamboo spine" pattern seen on imaging [17]	Patients should be referred to a rheumatologist for ongoing management. NSAIDS are used to provide quick symptomatic relief. A positive response to NSAID therapy can be used to help diagnose ankylosing spondylitis. Other medications used in the management of ankylosing spondylitis include sulfasalazine, methotrexate, corticosteroids, pamidronate, etanercept, and infliximab Non-pharmacologic management includes patient education, outpatient physical therapy, home exercise program, and proper posture training [17]

Cardiac Causes of Shoulder Pain

Pathology

Cardiac conditions can present with shoulder pain as a primary symptom due to referred pain. As discussed above, referred pain occurs when the brain perceives pain to be at a location that is different from the pain-generating source. The theory behind referred cardiac pain to the shoulder is that cardiac visceral sensory pain fibers follow the sympathetic nerves back to the spinal cord where their cell bodies are located in thoracic dorsal root ganglia from T1 to T4. The dermatomes of the upper body wall and upper limb have neuronal cell bodies in the T1–T5 dorsal root ganglia and, therefore, synapse on the same second-order neurons as the general visceral sensory fibers from the heart. The result is that the central nervous system cannot differentiate whether the pain is coming from the body wall or viscera. Pain originating from the heart can then be perceived as substernal pain, left shoulder, arm, hand, or jaw pain [18].

Table 9.3 includes the most common cardiac causes of shoulder pain and describes their presentation, physical exam signs, and the next diagnostic/treatment steps.

Table 9.3 This table summarizes common cardiac conditions that can present with shoulder pain as a primary symptom

	Clinical presentation	Physical exam	Next diagnostic or treatment steps
Myocardial infarction (MI)	Myocardial infarction typically presents with sudden onset substernal pain. There is often referred pain to the jaw/teeth, neck interscapular region, or the left arm/shoulder. Of note, the pain can also radiate down the ulnar surface of the bilateral arms. There is often a description of pressure or heaviness. This discomfort can sometimes be constricting or burning. In the elderly, pain can actually be absent during an MI. Instead, patients feel fatigue, dyspnea, or experience syncope [18]	Physicians should auscultate the heart in these scenarios to evaluate for any irregularities if there is any suspicion for cardiac cause of shoulder pain. The physician should also inspect for signs of heart dysfunction, such as lower extremity edema and weak peripheral pulses. If there is a suspicion for an acute myocardial infarction, the physical exam should not delay transport to an acute hospital setting	If an MI is suspected, the patient should be transported to the closest emergency room/hospital immediately Once at the hospital, laboratory studies, such as troponins and creatinine phosphokinase (CPK), an electrocardiogram (ECG), and an echocardiogram can be obtained [18]

(continued)

Table 9.3 (continued)

	Clinical presentation	Physical exam	Next diagnostic or treatment steps
Angina pectoris: Stable vs unstable	Patients with stable angina pectoris present with brief chest pain that can radiate to the jaw, shoulder, or arm. The pain usually lasts 5–10 min and rarely longer than 20 min. It is associated with exercise or stress. Stable angina pain responds to rest and sublingual nitroglycerin In unstable angina pectoris, the pain arises at rest and typically persists for longer than 20 minutes. The pain does not fully respond to nitroglycerin. There also tends to be associated pallor, sweating, nausea, vomiting, and/or a sense of impending doom [18]	If there is a suspicion for cardiac pathology leading to shoulder pain, the physician should auscultate the heart. Palpation for peripheral pulses and examination for lower extremity edema can also be performed	If the patient presentation is consistent with stable angina pectoris, referral to a cardiologist is warranted. Sublingual nitroglycerin can be given in the office for symptomatic relief, if indicated In cases of suspected unstable angina pectoris, it is critical to get the patient to a cardiologist immediately. An ECG, echocardiogram, and laboratory studies will then be obtained [18]
Pericarditis	In patients with pericarditis, the chest pain is worse with inspiration and changes in posture. The cause of pericarditis is often unclear, although it may be associated with viral illnesses [18]	Auscultation of the heart is the key exam maneuver for pericarditis as it is associated with a stereotypical friction rub heart sound	If pericarditis is suspected, urgent transport to an acute hospital setting is recommended
Aortic aneurysm	In an aortic aneurysm, there is an acute onset of sharp, tearing pain. The pain can be retrosternal in proximal dissection or interscapular in distal dissections. The pain can also change locations if there is further extension of the dissections [18]	If there is clinical suspicion for an aortic dissection, one can auscultate the heart; however, this should not delay transport to a hospital facility	Patients should be transported to the nearest hospital immediately for further workup and management

Gastrointestinal Causes of Shoulder Pain

Pathology

Pain is referred from intra-abdominal processes to the shoulder through irritation of the diaphragm. The diaphragm is innervated by the phrenic nerve. When a process irritates the diaphragm, the signals are sent up the trunk of the phrenic nerve to the brachial or cervical plexus. Thus, the suprascapular nerve, axillary nerve, and others can be affected leading to pain that is felt in the shoulder or arm [18].

Table 9.4 describes intra-abdominal processes that can result in shoulder pain. The clinical presentation, physical exam signs, and next diagnostic and treatment steps will be discussed.

Table 9.4 This table summarizes intra-abdominal processes that can present with shoulder pain as a primary symptom

	Clinical presentation	Physical exam	Next diagnostic or treatment steps
Gallstone disease (cholelithiasis)	Patients often present with right upper quadrant and epigastric abdominal pain. Patients can also have concurrent right shoulder pain. The pain is often sharp and severe and can be intermittent [18]	Physicians should perform a full abdominal exam including inspection, superficial and deep palpation, and auscultation. Rebound tenderness/guarding with deep palpation of the right upper quadrant is often seen	Depending on the severity of symptoms and whether there is concern for an acute abdomen, referral to a gastrointestinal specialist or transport to the nearest acute hospital is advised Further workup would include serum bilirubin, alkaline phosphatase, gamma-glutamyl transpeptidase, aspartate aminotransferase, and alanine aminotransferase. These studies are expected to be elevated in gallstone disease. Ultrasonography of the gallbladder and biliary tract is the preferred imaging method [18]
Conditions of the liver	Liver cancer or abscess could both present with right shoulder pain aggravated by breathing in patients with signs of clinical worsening, such as significant weight loss or fever [18]	In these cases, the physicians should pay close attention to the vital signs and perform a full abdominal exam including palpation of the liver	If an acute infection due to an abscess is suspected, the patient should be advised to go to an acute hospital. If liver cancer is suspected, recommend referral to a gastroenterologist or oncologist Ultrasound or a CT of the abdomen could be performed to evaluate the liver. A liver panel could also be obtained

Iatrogenic/ referred pain from intra-abdominal surgeries	Laparoscopic procedures, particularly laparoscopic cholecystectomies, can result in referred shoulder pain. Patients typically have nausea, vomiting, and pain in the abdomen and back. The pain may be due to pneumoperitoneum postoperatively. This is theorized to be due to carbon dioxide (CO_2) being converted to carbonic acid (H_2CO_3), which then irritates the diaphragm leading to referred pain Right shoulder pain is also common after liver biopsies and radiofrequency ablations for hepatocellular carcinoma. Severe pain shortly after a liver biopsy could represent bleeding or a subcapsular hematoma [18]	The physician should perform a full abdominal exam including inspection, palpation, and auscultation	An abdominal ultrasound or CT can be used to assess for bleeding, hematoma, or pneumoperitoneum. A CBC can also be obtained if there is concern for bleeding [18] If there are signs of an acute bleed or a worsening pneumoperitoneum, the patient should go to an acute hospital. Other conditions should be referred to a gastroenterologist or oncologist, as appropriate
Splenic rupture	Splenic rupture can lead to left shoulder pain and should be suspected in patients with recent abdominal trauma, though splenic rupture can occasionally happen spontaneously. Patient will present with left upper quadrant abdominal pain that refers to the left shoulder [18]	In splenic rupture cases, a full abdominal exam should be performed including inspection, palpation, and auscultation. Left upper quadrant pain radiating to the left shoulder is called Kehr's sign. While normally difficult to palpate, an enlarged spleen that has recently ruptured may be readily palpated [18]	An abdominal ultrasound or CT can be used to assess for splenic rupture. A CBC could also be obtained for signs of acute blood loss. Splenic rupture can be a serious condition, and referral to an acute hospital setting for further management and pain control is recommended [18]

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Shoulder Case Studies

10

Jasmin Harounian

Case 1 (Subacromial Bursitis)

Presentation

A 57-year-old right-hand-dominant male presents to the clinic with right shoulder pain that has been progressively worsening over the past 2 months without inciting event. He works as a construction worker and was a former tennis collegiate athlete. The pain is usually worse at night with laying on his right side, and it is exacerbated by reaching overhead and lifting or carrying objects. The patient denies any neck pain, weakness, numbness, or tingling in his upper extremities. He has tried acetaminophen, ibuprofen, and heating pads for his pain with only minimal relief. He has not started a formal physical therapy program yet but states he has a personal trainer at the gym. He started training about 2 weeks ago but noticed his shoulder pain has been preventing him from completing his workouts with his trainer.

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Physical Exam

On physical exam, inspection of the right shoulder reveals no atrophy, ecchymosis, or deformity. There is point tenderness on the superolateral aspect of the shoulder, distal to the AC joint. Strength testing is full in both upper extremities except for shoulder abduction, which is limited secondary to pain on the right. There are no sensory deficits. The patient has positive Neer's, Hawkin's, and a painful arc from 70 to 100° of abduction.

Imaging

X-rays of the right shoulder demonstrate mild osteoarthritis of the acromioclavicular and glenohumeral joints (Fig. 10.1). A diagnostic ultrasound of the right shoulder is performed, and a bursal effusion is visualized with image below.

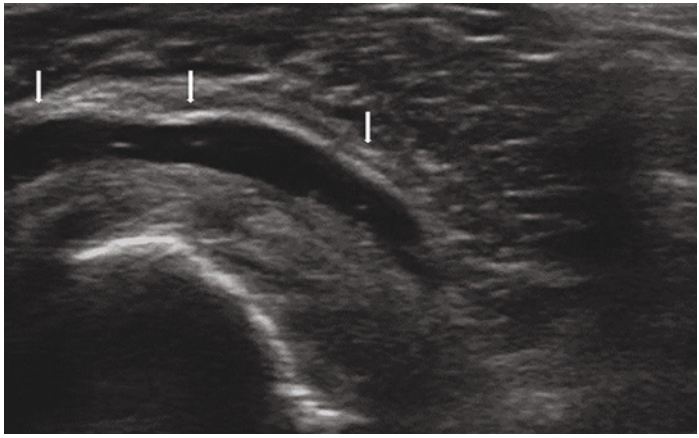

Fig. 10.1 Ultrasound diagnostic scan showing subacromial bursa effusion (white arrows)

Management

Given the patient's history, positive impingement signs of physical exam, and visualization of a bursal effusion on ultrasound, the patient's symptoms are likely due to a subacromial bursitis. The patient undergoes a subacromial subdeltoid bursa injection with corticosteroid under ultrasound guidance. He also receives a physical therapy prescription to begin a formal program. He returns to clinic 4 weeks later for follow-up and reports significant improvement in pain. He states physical therapy is going well, and his pain has decreased by more than 50%, allowing him to return to playing tennis.

Case 2 (Biceps Tendonitis)

Presentation

A 37-year-old right-hand-dominant female presents to clinic with right shoulder pain. The pain began without inciting event several months ago and has been progressively worsening. It is located along the anterior shoulder and occasionally radiates down to the elbow. Aggravating factors include lifting heavy objects and overhead movements. Alleviating factors include rest, NSAIDs, and heat. The pain ranges from 4/10 to 8/10. The patient reports she had previously trialed physical therapy without success. She denies any numbness, tingling, or radicular symptoms in the upper extremity. Medical, surgical, and family history are noncontributory. She is not taking any prescription medications.

Physical Exam

Inspection of the right shoulder reveals no atrophy, ecchymosis, or deformity. There is tenderness to palpation of the bicipital groove proximally. Range of motion is full and manual muscle testing is 5/5 in bilateral upper extremities. Provocative testing included a positive Speed's, Yergason's, and O'Brien's test. Hawkin's, Neer's, and Spurling's were negative.

Imaging

Shoulder XR shows no fracture or dislocation with well-maintained joint spaces (Fig. 10.2). An MRI of the right shoulder revealed a thickened intracapsular portion of the long head biceps tendon with hyperintense signal indicating tendinosis. The tendon remains located within the bicipital groove.

Axial views of the right shoulder MRI (Fig. 10.3) showing hyperintense signal around the long head of biceps tendon.

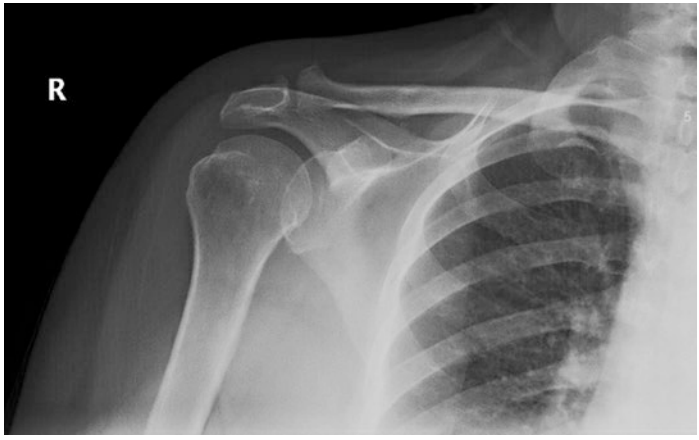

Fig. 10.2 Normal right shoulder XR

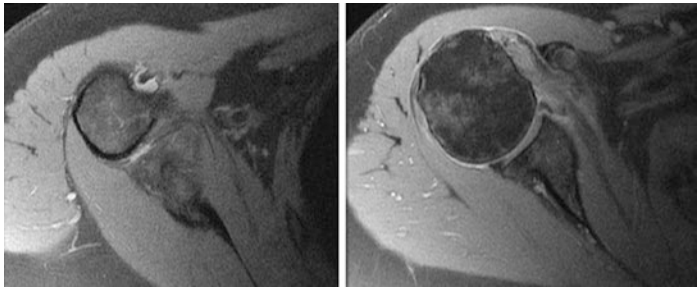

Fig. 10.3 Axial views of the right shoulder

Management

Given the patient's history and physical exam findings, her symptoms were most likely due to bicipital tendonitis. Patient underwent a biceps tendon sheath corticosteroid injection with ultrasound guidance. She reported 80% relief in the weeks that followed and was then given a new prescription for physical therapy for further rehabilitation.

Case 3 (Labral Tear)

Presentation

A 17-year-old right-handed baseball player presents to a primary care sports medicine clinic with complaints of right shoulder pain. The pain began 1 month ago after falling onto his shoulder while pitching. He has a history of rotator cuff tendinitis, but he states his current pain differs from what he has experienced in the past. He describes his current pain as a dull ache located deep in the shoulder. Occasionally, he will feel it "catch" but only with certain movements. It is not associated with any numbness or tingling in the arm, but the patient does notice his shoulder feeling more lax. He has tried taking over-the-counter medications without improvement of his symptoms.

Physical Exam

On exam, there is no obvious atrophy, ecchymosis, or deformity. Mild tenderness to palpation is elicited anteriorly at the subacromial space. Range of motion is full at the shoulder, elbow, wrist, and hand. Resisted shoulder abduction and shoulder flexion are limited due to pain. Sensation is intact throughout the upper

extremity. Provocative testing revealed a positive Crank and O'Brien's test and positive apprehension.

Imaging

X-rays of the right shoulder showed no evidence of acute displaced fracture or dislocation.

An MR shoulder arthrogram was ordered to evaluate for and better characterize any labral pathology (Figs. 10.4 and 10.5). It demonstrated an attenuated appearance of the anterior glenoid labrum. There was also a large nondisplaced tear of the posterior glenoid labrum, extending from the 10:00 to 6:00 position, involving the posterior inferior quadrant. There is also glenoid articular

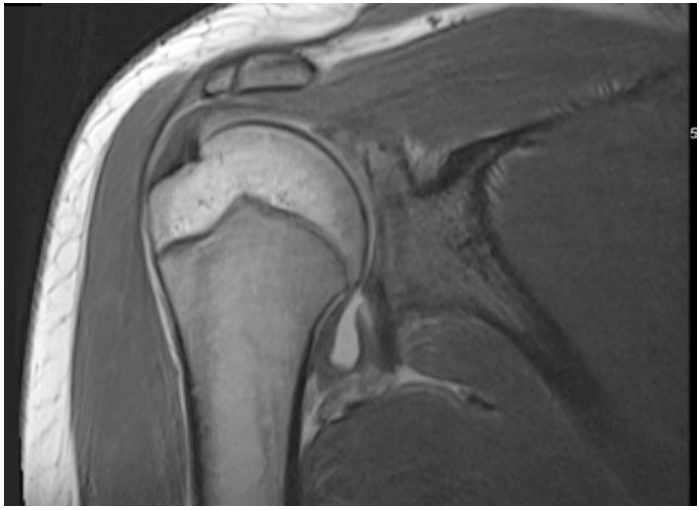

Fig. 10.4 MR arthrogram of the right shoulder, coronal view demonstrating posteroinferior labral tear

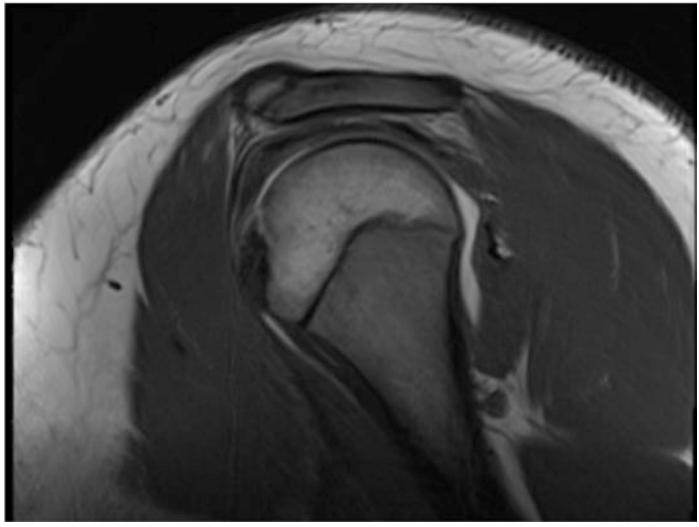

Fig. 10.5 Sagittal view demonstrating reverse Bankart lesion on the humeral head at 9:00 position

disruption around the 9:00 position and overlying disruption of the periosteum, which is concerning for a reverse Bankart lesion. There is slight flattening of the anterior humeral head measuring 0.9 × 0.6 × m at the level of the bicipital groove, raising concern for reverse Hill-Sachs lesion.

Management

Given the patient's pitching history, recent traumatic injury, and mechanical symptoms of "catching," the MR arthrogram was ordered as above. Once the results were obtained, a diagnosis of shoulder labral tear was confirmed. The patient was then referred to an orthopedic surgeon for surgical planning and repair.

Case 4 (Adhesive Capsulitis)

Presentation

A 47-year-old left-hand-dominant male presents with 1 month of left shoulder pain. He states the pain started 1 month after injuring his left wrist. He had flipped over the handlebars of his bike 2 months ago and suffered an intra-articular distal radius fracture. He underwent ORIF for this fracture and was casted until 1 month ago. After the cast was removed, the patient noticed left shoulder pain and stiffness with certain motions such as forward elevation, shoulder abduction, and internal rotation. He did not have any shoulder pain while wearing the cast and noted that he had full motion of the shoulder. He has used heat and done some physical therapy with his therapist that he sees for his wrist, both of which seem to improve the pain. He has not tried any medications. The pain is primarily located in the anterior and lateral shoulder.

Physical Exam

On exam, there is no obvious swelling, ecchymosis, or atrophy about either shoulder. Cervical spine and scapulothoracic range of motion are normal with no evidence of winging or crepitus. No tenderness to palpation over the AC joint, clavicle, or bicipital groove. He has a fairly limited arc of motion with some moderate capsular stiffness in all planes of shoulder abduction to 140 degrees, abduction to 50 degrees, and internal rotation to T10. Rotator cuff-related impingement signs are negative. Biceps arc signs are also negative. Motor and sensory function in the hand and arm is otherwise intact.

Imaging

XR left shoulder (Fig. 10.6) demonstrated no fracture or dislocation and no arthritic changes.

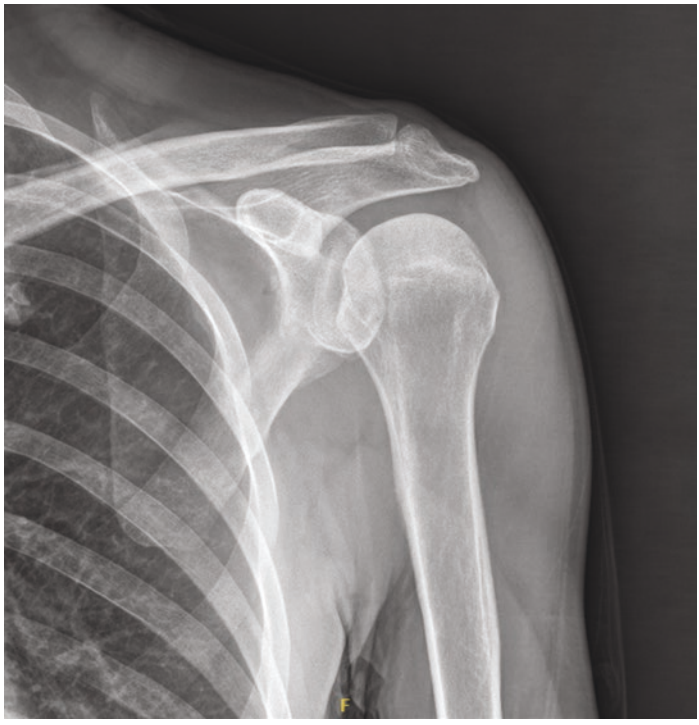

Fig. 10.6 X-ray of the left shoulder

Management

Given the patient's history and physical exam findings as well as the unremarkable X-ray, his symptoms are likely in the setting of adhesive capsulitis following trauma to the left arm. The patient was given a prescription for targeted physical therapy, focusing on shoulder capsule stretching and mobilization. If pain or limited range of motion persisted despite therapy, additional imaging could be obtained to further delineate any other concomitant pathology. The patient ultimately reported improvement at 3 month follow-up.

Case 5 (Shoulder Osteoarthritis)

Presentation

A 74-year-old right-hand-dominant male presents to clinic for bilateral (L > R) shoulder pain for the past few years. He is an avid yogi and wishes to continue his practice with less discomfort. The pain is described as a dull, ache deep in the shoulder joint. It is aggravated by carrying heavy objects and is also worse after exercise, particular after push-ups or planks. It is better with rest. The pain is also associated with stiffness in the mornings which improves as the day progresses. Currently, he is not taking any pain medications and has not tried going to physical therapy in the past. He denies any rashes or any stiffness or swelling of other joints.

Physical Exam

On exam, there is no obvious swelling, ecchymosis, or atrophy. Bilateral shoulders have good muscle build. Active range of motion was limited in forward flexion (95° on the left, 150° on the right) and shoulder abduction to 120° bilaterally. Passive range of motion demonstrated clunking in posterior shoulder joints bilaterally. Provocative testing reveals positive Hawkin's test bilaterally and negative Speed's, O'Brien's, and Yergason's tests.

Imaging

X-rays of bilateral shoulders were obtained. The left shoulder X-ray demonstrated advanced glenohumeral joint arthrosis with joint space narrowing, subchondral osseous remodeling, subchondral sclerosis, and marginal osteophytes, most prominently at the inferomedial margin of the humeral head. Cyst-like lucencies are present within the greater tuberosity. There is no acute fracture.

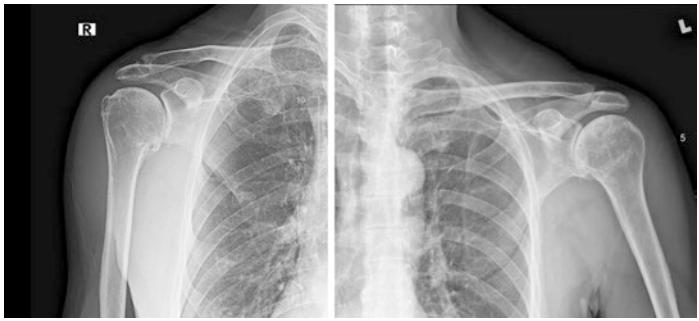

Fig. 10.7 X-rays of the bilateral shoulders

The right shoulder X-ray (Fig. 10.7) demonstrated moderate osteoarthritis with degenerative cysts throughout the humeral head. There is mild narrowing of the humeral acromial space. There is no acute fracture.

Management

The patient's symptoms were likely sequelae of his moderate-advanced glenohumeral osteoarthritis. He was given a prescription for a formal physical therapy program to work on rotator cuff and scapular stabilization and strengthening. He was also instructed to take acetaminophen 1000 mg up to three times a day as needed for pain control. He returned to clinic after 8 weeks with a 40% improvement in his symptoms. At this time, it was determined the patient would benefit from second-tier treatments. He then underwent a series of hyaluronic acid gel injections to bilateral glenohumeral joints under ultrasound guidance. At a 3-month follow-up visit, the patient reported he is now able to tolerate his current yoga practice with significantly less discomfort and is happy with his progress. He was to continue with his exercises and return for follow-up if his symptoms worsened.

Case 6 (Sports Trauma)

Presentation

A 58-year-old left-hand-dominant male presents with left shoulder and clavicular pain after falling off of his bike last week. The pain is primarily located at the left acromioclavicular joint and radiates upward toward the neck and shoulder blade. It is described as dull, achy, and stiff in nature. Pain is aggravated by any shoulder motion and alleviated by ice and rest. He has tried taking acetaminophen and ibuprofen with moderate relief. He uses a sling when he leaves his home to prevent additional motion. He denies any numbness, tingling, or weakness into the left hand.

Physical Exam

Inspection of the shoulder reveals an obvious bony deformity of the left acromioclavicular joint with an overlying healed abrasion. There was tenderness to palpation over the area of deformity. During active range of motion, there was limited left shoulder flexion to 90° due to pain. Strength testing was full except for pain-limited shoulder abduction.

Imaging

X-ray of the left clavicle (Fig. 10.8) demonstrated superior displacement of the distal clavicle compared to the acromion process, with possibly increased coracoclavicular distance.

Management

Given the patient's mechanism of injury and imaging findings, the patient's symptoms are consistent with a type III AC joint separation. After discussion with the patient regarding treatment options,

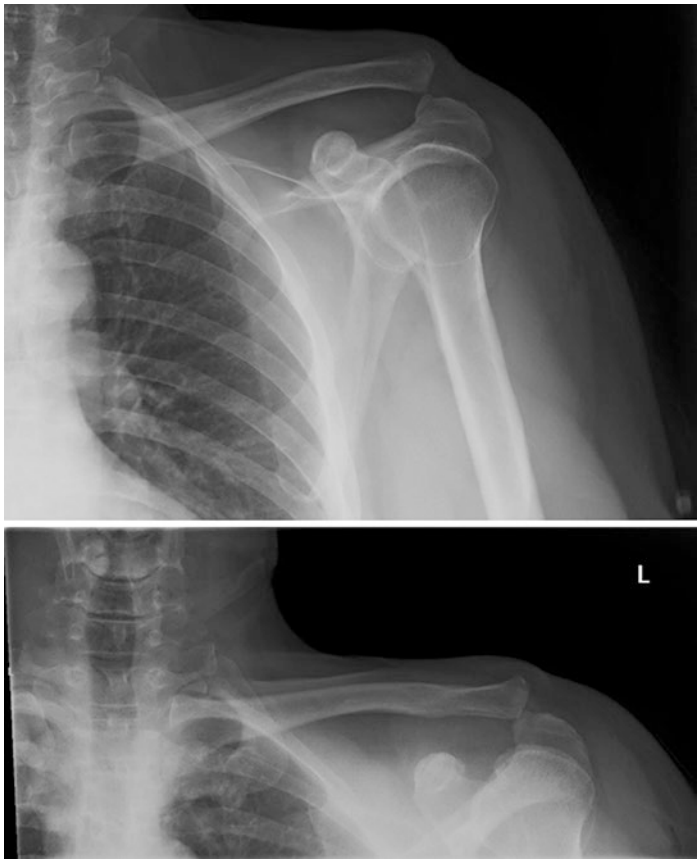

Fig. 10.8 X-rays of the left clavicle

the decision was made to manage his AC joint separation nonoperatively. He was given a prescription to initiate a physical therapy program and began gentle range-of-motion exercises at home. On 6-week follow-up, the patient reports return of full painless ROM and 5/5 strength in the left arm. He continued with a home exercise program.

Case 7 (Clavicular Fracture)

Presentation

A 34-year-old female presents to the Emergency Department after an ATV accident that occurred 3 days ago while she was abroad. The patient reports she flipped over the ATV with the handlebars hitting her bilaterally in the chest. The pain is rated as moderate and described as throbbing in nature. She denied any head trauma or loss of consciousness but noted immediate left shoulder pain and deformity. Initially, she did have tenting but reports a physician at the hospital abroad was able to reduce the shoulder. Currently, she denies any numbness, tingling, or weakness in the left arm.

Physical Exam

On inspection, there are mild abrasions on bilateral clavicles with some skin dimpling and inward caving of the left clavicle. Scattered ecchymoses are present across the chest with tenderness to palpation across the left>right clavicle. The fingers are warm and well-perfused. Radial pulses are 2+ and palpable bilaterally. Sensation is intact to light touch in the radial, medial, and ulnar nerve distributions. Patient is able to fire the anterior interosseous, posterior interosseous, and ulnar nerves. No tenderness to the cervical, thoracic, or lumbar spine.

Imaging

Left clavicle X-ray (Fig. 10.9) demonstrates an acute traumatic comminuted displaced fracture of the midclavicular shaft with approximately 1 cm of displacement of the distal component. No pneumothorax.

Management

Due to skin puckering over the fracture site, the patient was admitted for preoperative planning of the left clavicular fracture and was

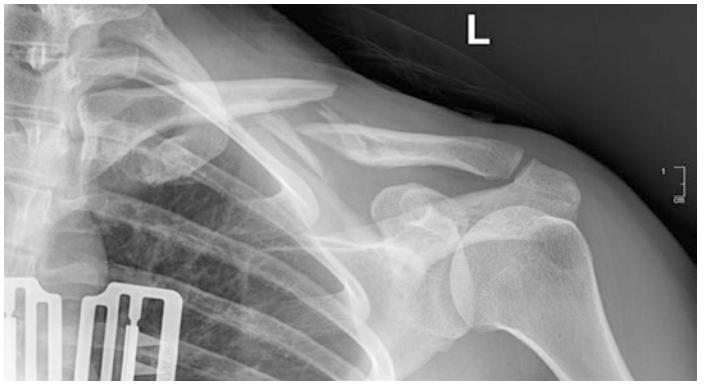

Fig. 10.9 X-ray of the left clavicle

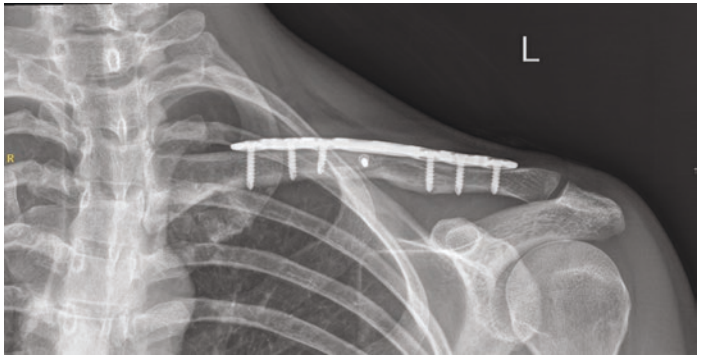

Fig. 10.10 Postoperative X-ray of the left clavicle

non-weight-bearing to the left upper extremity. She underwent left clavicle open reduction and internal fixation. At the 2-week follow-up visit, patient began gentle range of motion exercises, including shoulder pendulums, but continued non-weight-bearing status. She was allowed to perform activities below the waist, such as typing and texting. By the 4-month follow-up visit, patient reports she is doing well postoperatively and is back at work. She denies any pain and has been able to use her arm normally. Repeat X-rays showed maintained fracture reduction and implant position with no evidence of complication (Fig. 10.10).

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