REVIEW ARTICLE

COMMON ORTHOPAEDIC SHOULDER DIAGNOSES ENCOUNTERED IN THE PRIMARY CARE SETTING

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KEYWORDS

Shoulder pain

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Frozen shoulder

ABSTRACT

Shoulder pain and shoulder disorders are commonly seen in the primary care setting. While many of these disorders can be managed by the primary care physician, some may pose a diagnostic dilemma. This article will review 10 common shoulder disorders, evaluating when conservative management is appropriate and when referral to a specialist is warranted.

INTRODUCTION

Shoulder pain and shoulder pathology are common chief complaints in the primary care setting. The shoulder consists of multiple joints, mainly the glenohumeral joint. The other joints that are considered in the shoulder include the acromioclavicular, sternoclavicular, and scapulothoracic joints. Each joint has associated pathology and should be considered in the workup of shoulder pain. Most common pathologies, and the majority of those further discussed, involve the glenohumeral joint. The glenohumeral joint is a ball-and-socket joint formed between the humeral head and the glenoid of the scapula. The joint exhibits significant freedom of motion in all planes, including flexion, extension, abduction, adduction, internal rotation, and external rotation. Motion also exists at the scapulothoracic joint, which also may present with pathology. The glenohumeral joint is stabilized both dynamically and statically. The major dynamic forces include the musculature of the shoulder, most importantly the rotator cuff musculature and biceps brachii, which comprise a large subset of pathology discussed in this article. This complex joint provides multiple areas for pathology to arise.

Shoulder pain and shoulder disorders are commonly seen in the primary care setting. While many of these disorders can be managed by the primary care physician, some may pose a diagnostic dilemma. This article will review 10 common shoulder disorders, evaluating when conservative management is appropriate and when referral to a specialist is warranted.

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SUBACROMIAL IMPINGEMENT SYNDROME

Subacromial impingement syndrome is a common cause of shoulder pain seen in the primary care setting. It represents 36% of all shoulder disorders.[1] Subacromial impingement syndrome is characterized by inflammation, which may be related to repetitive movement. The spectra of disorders related to subacromial impingement syndrome range from subacromial bursitis, to rotator cuff tendinopathy, to partial- or full-thickness rotator cuff tears. Typically, there is a compression of the rotator cuff, subacromial bursa, or other soft tissue between the humeral head and the acromion, acromioclavicular (AC) joint, or coracoacromial arch. Many patients who exhibit subacromial impingement syndrome also have abnormal scapular movement.[2] In addition, impingement syndrome may predispose the patient to rotator cuff tears.[3]

Subacromial impingement syndrome is believed to be a three-phase progression. Stage 1 involves younger patients (less than 25 years old) and is characterized by acute bursitis with subacromial edema and inflammation. Stage 2 is more common in patients 25–40 years of age and is mostly characterized by rotator cuff tendonitis and/or fraying of the anterior fibers of the supraspinatus. Stage 3 is characterized by partial- or full-thickness tearing of the rotator cuff.[4]

At presentation, the patient may complain of shoulder pain exacerbated by overhead activity. Exacerbation of symptoms is

common with elevation greater than 90°. Pain may also worsen at night.[5] A thorough history and physical exam are important, as many conditions can mimic impingement syndrome. A comprehensive physical exam should include range of motion, strength, and special testing. Special testing includes Neer and Hawkins tests, which are sensitive but not specific for impingement syndrome.[6] During the Neer test, the examiner passively flexes the patient's shoulder with the arm internally rotated; reproduction of pain is a positive exam. With the Hawkins test, which is performed at 90° of shoulder and elbow flexion, the examiner exerts an internal rotation force on the shoulder. If pain is reproduced with internal rotation of the shoulder, this is a positive Hawkins test. Calcific tendonitis is a common cause of active therapy failure, and there are multiple surgical options available for treatment. A lidocaine challenge injection, which involves injecting 5–10 mL of 1% or 2% lidocaine without epinephrine to the subacromial space, can support a diagnosis of impingement syndrome but is not frequently performed in clinical practice. Full pain relief upon reexamination postinjection supports a diagnosis of impingement syndrome.

The majority of patients with subacromial impingement syndrome improve with conservative treatments. Nonsurgical treatment typically includes home exercises, physical therapy (PT), and subacromial cortisone injections. Osteopathic manipulative treatment (OMT) may also be performed to help mobilize the ribs, stabilize the scapula (to prevent dyskinesis), and treat restrictions at the AC joint.[7] Studies have found that two-thirds of patients experience significant improvement with conservative management.8 If the patient fails conservative treatment, they may be a candidate for orthopedic surgical intervention.

Surgical treatment options include open or arthroscopic subacromial decompression. Arthroscopic subacromial decompression is associated with decreased recovery and less pain in the immediate postoperative period.

FROZEN SHOULDER

Frozen shoulder, also known as adhesive capsulitis, refers to a global loss in range of motion in the shoulder. This is due to a soft tissue contracture that limits both passive and active range of motion. There are three stages of frozen shoulder: freezing, frozen, and thawing. Frozen shoulder can occur in combination with other conditions, including rotator cuff tears and degenerative joint disease. While there is no consensus on the biological cause of frozen shoulder, systemic disorders such as diabetes mellitus, thyroid disease, cardiovascular disease, and neurologic conditions may contribute to it. Patients with diabetes mellitus are at a greater risk for frozen shoulder than the general population and the condition tends to be more severe.[9] The prevalence of developing frozen shoulder is 2%–5% in a lifetime for all individuals but is most prevalent in those who are 50–60 years of age. Frozen shoulder is more common in females and more frequently located on the nondominant side.[10]

Shoulder motion should be assessed and documented diligently. Differential diagnosis should include frozen shoulder, rotator cuff pathology, and glenohumeral arthritis. Although a finding of global decrease in both passive and active range of motion is highly suspicious for frozen shoulder, these findings must be consistent to determine whether or not treatment is successful. The physician should record both passive and active range of motion. Passive motion should be evaluated with the patient supine to restrict scapulothoracic movement. Passive flexion, external rotation in abduction (arm away from the patient's body), external rotation and internal rotation in adduction (arm at the patient's side), and cross-chest adduction should be measured. Magnetic resonance imaging (MRI) findings may demonstrate thickening of the joint capsule and the coracohumeral ligament; however, frozen shoulder is a clinical diagnosis. An MRI is also useful for eliminating other sources of shoulder pain.

Frozen shoulder tends to resolve with nonsurgical treatment, but resolution may take as long as one to three years. For primary frozen shoulder, defined as insidious onset without inciting event, a supervised PT program is successful in the majority of patients. Secondary frozen shoulder, defined as the diminished global range of motion of the shoulder secondary to shoulder injury or surgery, does not have the same success rate with formal PT alone. Typically, 6 weeks of formal PT is recommended for both subsets of patients. If the patient continues to make progress, an additional 6 weeks of PT, followed by a home exercise program, is reasonable. Techniques using OMT can be applied to the upper thoracic area, upper ribs, and entire shoulder complex to improve motion. For example, Spencer techniques can be utilized to challenge the range of motion barriers in multiple planes of motion. In conjunction with formal PT, other conservative measures should be exhausted in patient treatment, including nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroid injections, and glenohumeral lidocaine injections.[11] In the freezing phase, where pain is the largest concern, corticosteroid injection and oral medications are most successful.[11] In the frozen phase, where restricted range of motion is most prevalent, formal PT is best used.[11] If, after 12 to 16 weeks, there is no improvement or worsening of symptoms, surgical intervention may be considered.

Surgical management includes manipulation under anesthesia (MUA). MUA is often performed in combination with an arthroscopic capsular release. A formal course of PT, typically for 6 weeks, is essential postoperatively to maintain range of motion and improve shoulder strength

SHOULDER OSTEOARTHRITIS

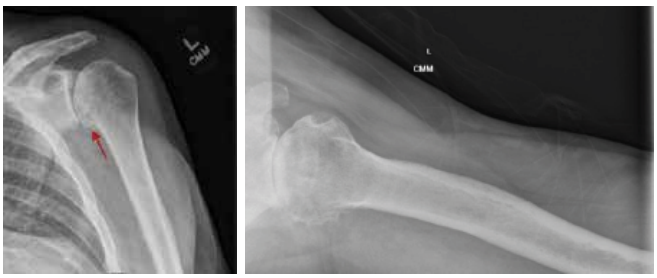
Osteoarthritis of the shoulder can occur at both the acromioclavicular joint and the glenohumeral joint; these pathologies present a common cause of shoulder pain. Shoulder osteoarthritis is commonly referred to as a degenerative joint disease. As a progressive disease, this results in the loss of articular cartilage over time, evidenced by radiographic changes, including osteophyte formation, subchondral sclerosis, and subchondral cyst formation, which leads to pain and functional impairment. The prevalence is higher among females and increases with age. Other risk factors include participating in overhead sports and occupations associated with physical labor, such as those related to construction. Primary osteoarthritis does not have a specific cause, while secondary osteoarthritis is related to a predisposing

factor. Such factors include previous trauma, dislocations, osteonecrosis, or chronic rotator cuff tears.

A diagnosis of shoulder osteoarthritis is based on symptoms, physical examination, and radiographic findings. During the early phases of osteoarthritis, patients present with progressive pain exacerbated by activity. This is often described as a generalized dull ache, deep in the joint. Initially, the physical examination may be unremarkable. As the disease progresses, the symptoms may become more severe and can include night pain and crepitation. In advanced cases, decreased range of motion and pain may affect the patient's activities of daily living.

Imaging studies, including a full set of plain radiographs (AP [anteroposterior], Grashey, scapular Y, and axillary) are crucial to a diagnosis of shoulder osteoarthritis. In the majority of cases, a diagnosis of degenerative joint disease can be established with conventional radiographic imaging. Early on, radiographic findings (Figure 1) may be subtle, but as the disease progresses, they may show joint space narrowing, osteophytes, subchondral sclerosis, and cysts. Axillary views are particularly useful for evaluation of joint space narrowing.

The goals of treatment are to improve function and pain control. Initial conservative treatment consists of activity modification and acetaminophen. Typical acetaminophen dosing is 650 mg or 1,000 mg every 6 hours, with maximum dosing being 4,000 mg in a 24-hour period. Physical therapy is useful to maintain range of motion. Approximately 50%–67% of patients demonstrate improvement of symptoms with NSAIDs but with varying side effects.[12,13] The primary care physician must weigh the risks and benefits of NSAIDs. In advanced degenerative joint disease, intra-articular corticosteroid injections may be used to improve pain and swelling. If conservative therapy fails, there are multiple surgical options available and the patient should be referred to an orthopedic surgeon. Surgical options include arthroscopic debridement, resurfacing, and shoulder arthroplasty. The procedure of choice will vary based on the age of the patient, functional expectations, and integrity of the rotator cuff.



I II

FIGURE 1:
Anteroposterior radiograph of the glenohumeral joint (I) and axillary (II) radiograph of the shoulder demonstrate glenohumeral osteoarthrosis with joint space narrowing and inferior spur formation (red arrow) of the humeral head.

BICEPS TENDONITIS

Biceps tendon dysfunction can occur in isolation but is commonly seen in conjunction with other shoulder pathology. The biceps brachii is a muscle in the upper arm that acts to supinate the forearm and flex the elbow. This muscle has two heads: the short head and the long head. The origin of the short head is on the coracoid process, while the long head originates on the supraglenoid tubercle and the superior glenoid labrum. While there are various types of injuries to this complex, this section will solely cover biceps tendonitis.

Biceps tendonitis is a commonly encountered pathology associated with the long head of the biceps tendon. Patients typically present with anterior shoulder pain, which may radiate to the muscle belly of the biceps. The pain may be exacerbated with overhead activity or with resisted elbow flexion. A thorough physical exam is necessary to differentiate the cause of pain. There may be tenderness with palpation of the biceps in the bicipital groove. Muscle strength testing and special tests, including Speed's and Yergason's, are helpful in a diagnosis of biceps tendonitis. Speed's test is performed with forward flexion of the shoulder against resistance, with the elbow extended and forearm supinated. Speed's test is positive if pain is elicited at the bicipital groove. Yergason's test is performed with active supination, with the forearm pronated, elbow flexed to 90°, and the shoulder adducted. Yergason's test is also positive if pain is elicited at the bicipital groove.

Radiographs are not diagnostic but should be obtained to help evaluate for potential bony pathology. Patients with tendonitis of the long head of the biceps will typically have normal radiographs. An MRI and magnetic resonance arthrography may be useful to evaluate for labral pathology or intra-articular biceps pathology; however, they are not as reliable as arthroscopy in the diagnosis of biceps pathology.[14]

In a patient diagnosed with biceps tendonitis, first-line treatment is conservative. This often includes NSAIDs, activity modification, PT, and corticosteroid injections. Corticosteroid injection can be administered to the bicipital sheath and should not be directly injected into the tendon. If a patient fails nonoperative management, referral to an orthopedic surgeon for further evaluation is recommended, as biceps tenodesis or tenotomy can be performed for refractory cases.

CALCIFIC TENDONITIS

Calcific tendonitis of the shoulder typically affects patients between the ages of 30 and 60 years of age. Females are more frequently affected than males. Bilateral shoulder involvement is common. Calcific tendonitis is a painful condition that involves the deposition of calcium hydroxyapatite within the rotator cuff tendons, but many patients with radiographic findings may be asymptomatic. The supraspinatus is the most commonly involved tendon, with an incidence of 51.5%–90% of cases.[15] The cause of calcific tendonitis is still unclear, but there is an association with endocrine disorders such as diabetes and hypothyroidism.[15]

There are four stages of calcific tendonitis. The precalcific stage is usually pain free and includes fibrocartilaginous transformation within the rotator cuff tendon. The formative stage is the stage in which calcium is deposited in the rotator cuff tendon. This stage may or may not be painful. During the resting phase, the calcium deposition is terminated and there is no inflammation or vascular infiltration. Similarly, this phase may or may not be painful. The resorptive phase is considered the most debilitating for patients. Calcium crystals may extravasate into the subacromial bursa, which is a process commonly associated with severe pain and loss of range of shoulder motion. This phase can last for up to 2 weeks.

Physical examination findings are similar to subacromial impingement syndrome. Pain is worse at night and limited range of motion with overhead activities may be present. Imaging is necessary to distinguish between calcific tendinosis and other sources of shoulder pain. Plain radiographic imaging is usually diagnostic for calcific tendonitis (Figure 2). Other modalities such as ultrasound and MRI are not usually required for diagnosis but assist in evaluating for other associated pathology.

Calcific tendinosis is, in general, self-limited. Treatment is usually supportive with NSAIDs, acetaminophen, steroid injections, and PT. Steroid injections tend to be particularly effective in the acute setting.[16,17] If calcific tendonitis fails to respond to conservative treatment, referral to an orthopedic specialist is recommended, as a patient may be a candidate for arthroscopic calcific debridement.

FIGURE 2:

Anteroposterior (I) and scapular Y (II) radiographs of the shoulder demonstrate calcific tendonitis (arrow).

ROTATOR CUFF TEARS

The rotator cuff provides dynamic stability to the glenohumeral joint. It is composed of four muscles: infraspinatus, teres minor, supraspinatus, and subscapularis. These muscles work together to balance the glenohumeral joint in the coronal and transverse planes.

A common cause of shoulder pain in patients over 40 years of age, rotator cuff tears can be the result of an acute injury or progressive degeneration due to impingement. Acute avulsion injuries typically occur as a result of trauma or, in the case of older patients, may result from sustaining a fall or shoulder dislocation. In patients over 60 years of age, rotator cuff tears tend to be caused by chronic degeneration or chronic impingement. Patients over the age of 70 are more likely to have full-thickness rotator cuff tears.[18,19]

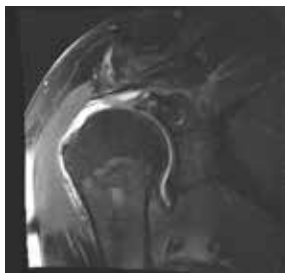
Patients with rotator cuff tears may present with progressive weakness, worsened with overhead motion of the affected arm. Patients who sustained an acute traumatic tear may present with acute pain and weakness of the affected arm or pseudoparalysis. In addition, night pain is commonly associated with rotator cuff tears. A thorough physical exam should be performed on all patients, and all four rotator cuff muscles should be tested individually with muscle strength testing and associated special tests. Supraspinatus primarily functions in abduction of the shoulder and is best tested with resisted abduction. The drop arm test and the Jobe test (also known as the empty can test) are two special tests to evaluate the supraspinatus. Infraspinatus and teres minor both function to externally rotate the shoulder. Infraspinatus is best examined by testing muscle strength in external rotation at 0° of abduction, while the teres minor is best tested in external rotation at 90° of abduction. The teres minor can be examined with the Hornblower's test, in which the patient's arm is abducted to 90° with the elbow flexed to 90°. The patient is then asked to externally rotate the arm to 90° against resistance. If the arm drops back to a neutral position, the test is positive. The subscapularis functions to internally rotate the shoulder and is best tested with resisted internal rotation at 0° of abduction. Special tests for the subscapularis include the belly press, bear hug, or lift-off sign.

In addition to physical examination, imaging also plays an important role in the diagnosis and management of rotator cuff tears. Plain radiographs are useful in assessing associated calcific deposits in the tendons or ligaments or to evaluate for superior migration of the proximal humerus. Superior migration is a sign of long-standing rotator cuff arthropathy. Because MRI is the gold standard for diagnoses of rotator cuff injuries, it should be ordered when there is a high clinical suspicion (Figure 3). Ultrasound can also be useful in providing static or dynamic examination.

Treatment of rotator cuff tears consists of nonoperative management and operative management. The conservative approach is typically first-line treatment for most tears, especially partial tears. A crucial component of conservative measures is PT, with focus on regaining lost range of motion, followed by rotator cuff strengthening and scapular stabilization. Administration of NSAIDs and subacromial corticosteroid injections can be useful in symptom management. Indications for surgery and referral to orthopedics include acute full-thickness tears, pseudoparalysis, massive rotator cuff tears, and tears greater than 50% that have failed conservative treatment. Open or arthroscopic rotator cuff repair can be performed.

FIGURE 3:

MRI T2-weighted coronal sequence of the shoulder demonstrates a full-thickness supraspinatus tear with retraction.



GLENOHUMERAL JOINT DISLOCATIONS

Glenohumeral joint dislocations are common and represent 50% of all joint dislocations, with 97% being anterior. Posterior and inferior dislocations may also occur; however, inferior dislocations (luxatio erecta) are rare, accounting for less than 1% of shoulder dislocations.[20] Shoulder dislocations most commonly occur in younger males or older females. Shoulder dislocations in younger patients tend to be traumatic or sports related. Shoulder dislocations in older patients are more likely to occur from falls or be associated with fractures. The mechanism of injury for anterior dislocations is forced abduction and external rotation of the arm.

A patient with an anterior glenohumeral dislocation will typically present with their arm held in internal rotation and abduction with reluctance to move the arm. The presumptive diagnosis of glenohumeral dislocation is often apparent based on history and physical examination. A prominence can be palpated in the anterior shoulder with an emptiness below the acromion. Radiographic imaging is used for definitive diagnosis and to evaluate for associated fractures. These views should be obtained: AP, lateral, and axillary or modified axillary (Valpeau). A Valpeau view is performed with the affected arm adducted and internally rotated onto the patient's chest with the patient leaning backward at a 30- to 45-degree angle. Radiographic imaging should be obtained before attempting close reduction to evaluate the direction of dislocation and presence of associated fractures. In an anterior dislocation, the radiograph will show an anterior, inferior, and medially located humeral head. Postreduction radiographic imaging should also be obtained to confirm successful reduction.

Once a glenohumeral dislocation is confirmed, it is important to reduce the dislocation to help avoid muscle spasms and potential neurovascular injury.[21] There are multiple reduction techniques. An intra-articular injection of lidocaine, 20 mL of 1% lidocaine utilizing either the anterior or posterior portal, may first be attempted for pain control.[22] If adequate pain control is not achieved or the reduction is difficult, conscious sedation should be used. There are multiple different reduction maneuvers. Traction-countertraction technique is a commonly used reduction maneuver and involves wrapping a sheet under the axilla. Traction is applied at the wrist and elbow while an assistant applies countertraction from the opposite side. Other reduction maneuvers include the Stimson technique; Fast, reliable, and safe (FARES) technique; scapular manipulation; external rotation; and the Milch technique.[23] Following a successful reduction, the patient should be immobilized for 3 to 4 weeks with gradual return to full active range of motion.

There is a high incidence of recurrence in younger patients, with a 90% risk of repeat dislocation in patients less than 20 years old.[24] Young patients should be referred to an orthopedist because early intervention decreases the risk of recurrent instability. In cases of recurrent glenohumeral dislocation and associated injuries, additional imaging and orthopedic referral should be considered. Commonly associated injuries include Bankart lesions, Hill-Sachs defects, tuberosity fractures (greater or lesser), rotator cuff tears, and other labral tears. All of these associated injuries should be referred to an orthopedic surgeon promptly, as they may require surgical intervention.

PROXIMAL HUMERUS FRACTURES

Proximal humerus fractures are a common fracture type and are most frequently seen in older individuals, particularly females greater than 65 years of age.[25–27] In younger patients less than 50 years old, high-energy trauma, such as motor vehicle accidents or sports injuries, are common causes.[27] In older patients, the mechanism of injury will often involve a fall from standing height onto an outstretched hand. Osteoporosis is a risk factor for older patients secondary to diminished bone quality leading to increased fragility of the bone, which increases the likelihood of proximal humerus fracture.[28]

Patient presentation and physical exam are important steps in the diagnosis of proximal humerus fractures. Physical exam findings include pain and swelling of the shoulder and upper arm, with decreased range of motion of the shoulder. It is always important to perform a thorough neurovascular exam as concomitant injury can occur to the axillary nerve. It may not be possible to assess motor function in the acute setting due to pain. Sensory testing of the lateral shoulder should be performed. In addition to the physical exam, diagnosis of a proximal humerus fracture requires radiographic imaging. A true AP or Grashey view, a scapular Y view, and axillary views should be obtained. Additional studies, including computed tomography (CT), may be useful for preoperative planning, especially if there is concern for intra-articular comminution or there is an unclear view of the fracture fragments on plain radiographs, but are not necessary in the primary care setting.

Management of proximal humerus fractures is based on the fracture pattern and extent of displacement. The Neer classification is used to classify proximal humerus fractures. This classification system is based on the anatomy of the potential fracture segments.[29] The four potential segments include the greater tuberosity, lesser tuberosity, humeral head, and humeral shaft. The distinction of a part or segment is important to the system. A fracture fragment is classified as a distinct part or segment if it is displaced greater than one centimeter or if there is more than 45° of angulation.

Most fractures of the proximal humerus can be treated conservatively. Indications for a nonsurgical treatment include a minimally displaced surgical or anatomical neck fracture, or greater tuberosity fractures with less than 5-mL displacement.30 Nonoperative management consists of sling immobilization followed by PT for rehabilitation.[30]

Displaced fractures should be referred to an orthopedic surgeon for consideration of surgical intervention. There are various operative fixation methods, including open reduction with internal fixation, percutaneous fixation, intramedullary nailing, and arthroplasty.

CLAVICLE SHAFT FRACTURES

Clavicle shaft fractures are a common injury seen secondary to trauma. Clavicle fractures account for 2.5%–5% of all fractures, and midshaft clavicular fractures account for 69%–82% of all clavicle fractures.[30–32] These injuries are more often seen in children and

young adults, and most commonly in males less than 30 years old.[33] Fractures of the clavicle are often displaced secondary to deforming forces. The sternocleidomastoid muscle will pull the medial fragment superiorly and posteriorly, while the pectoralis will move the lateral fragment inferiorly and medially.

A patient with a clavicular fracture will often present with anterior shoulder pain following trauma. Ecchymosis or skin breakdown around the clavicle may occur. In some instances, there may be a visible deformity of the bone, but this may not be obvious due to swelling of the soft tissues. Physical exam of these patients may reveal a palpable deformity, tenderness, crepitus, and/or motion around the site of the clavicular fracture. In addition to examination of the injured area, a complete physical exam should be completed. A neurovascular exam of the upper extremity is important as these injuries can be associated with concomitant brachial plexus injury. If a clavicle fracture is suspected, in addition to standard shoulder imaging, dedicated clavicle views should be obtained, including a serendipity view and a Zanca view, which is performed at 15° of cephalic tilt. The Zanca view can be helpful in determining the extent of fracture displacement. Treatment is determined by the extent of displacement and shortening of the fracture fragments.

The goal of clavicle fracture management is to restore shoulder function while avoiding nonunion or symptomatic malunion. Conservative treatment consists of either sling immobilization or figure-of-eight bracing. If nonsurgical treatment will be pursued, there should be less than two centimeters of shortening and no neurovascular injury. Operative treatment of midshaft clavicle fractures is often used for fractures with associated neurovascular injury, open fractures, significantly displaced fractures with skin tenting, floating shoulder, or fractures with more than two centimeters of shortening with 100% displacement.[33] Patients with these injury patterns should be referred to an orthopedic surgeon for operative fixation, which will allow patients an earlier return to normal activity.

CERVICAL CAUSES

When evaluating a patient presenting with complaints of shoulder pain, it is important to consider cervical causes. It may be difficult at times to distinguish between shoulder pain referred from cervical pathology or primary shoulder pathology; however, identifying the correct pain generator allows for appropriate treatment. A patient with cervical radiculopathy may have shoulder pain as the major complaint. In patients presenting to a shoulder specialist, 3.6% were found to have primary cervical pathology.[34] Radicular symptoms may occur after trauma or develop insidiously. The most common nerve roots affected are C-6 and C-7 roots.

Physical examination is critical to distinguishing cervical versus shoulder pathology. The Spurling maneuver may be done to elicit radicular symptoms. It is performed by extending the patient's neck, rotating the patient's head to the side of the pain, and then applying downward pressure on the head. A positive test is indicated by reproduction of the patient's symptoms with provocative maneuver. Initial radiographic workup consists of plain radiographs including AP, lateral, and lateral flexion and extension views. Plain radiographs can identify abnormalities in cervical spine alignment or arthritic changes.[34] Advanced imaging, including CT and MRI, can further evaluate the bony and soft tissue anatomy of the cervical spine.[34] A recent epidemiologic survey of cervical radiculopathy indicates that symptoms resolve in 75% of patients with conservative treatment.[35]

CONCLUSION

The 10 shoulder disorders reviewed in this article represent some of the most common shoulder diagnoses encountered in the primary care setting but is not all inclusive. Knowledge of these disorders is important because most of them can be managed without referral to a subspecialist. It is also imperative to identify when referral to a specialist is warranted.

REFERENCES

1. Juel NG, Natvig B. Shoulder diagnoses in secondary care, a one year cohort. *BMC Musculoskelet Disord*. 2014;18;15:89. doi:10.1186/1471-2474-15-89
2. Moura KF, Monteiro RL, Lucareli PRG, Fukuda TY. Rehabilitation of subacromial pain syndrome emphasizing scapular dyskinesis in amateur athletes: a case series. *Int J Sports Phys Ther*. 2016;11(4):552–563.
3. Dela Rosa TL, Wang AW, Zheng MH. Tendinosis of the rotator cuff: a review. *J Musculoskel Res*. 2001;5(3):143–158. doi:10.1142/S0218957701000544
4. Neer CS II. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. *J Bone Joint Surg*. 1972;54(1):41.
5. Armstrong AD, Hubbard MC. *Essentials of Musculoskeletal Care*. 5th ed. AAOS; 2016.
6. Lange T, Matthijs O, Jain NB, Schmitt J, Lützner J, Kopkow C. Reliability of specific physical examination tests for the diagnosis of shoulder pathologies: a systematic review and meta-analysis. *Br J Sports Med*. 2017;51(6):511–518. doi:10.1136/bjsports-2016-096558
7. Hunter DJ, Rivett DA, McKiernan S, Luton R, Snodgrass SJ. Thoracic manual therapy improves pain and disability in individuals with shoulder impingement syndrome compared with placebo: a randomized controlled trial with 1-year follow-up. *Arch Phys Med Rehabil*. 2022;103(8):1533–1543. doi:10.1016/j.apmr.2022.03.003
8. Morrison DS, Frogameni AD, Woodworth P. Non-operative treatment of subacromial impingement syndrome. *J Bone Joint Surg Am*. 1997;79(5):732–737. doi:10.2106/00004623-199705000-00013
9. Warner JJP. Frozen shoulder: diagnosis and management. *J Am Acad Orthop Surg*. 1997;5(3):130–140. doi:10.5435/00124635-199705000-00002
10. Zreik NH, Malik RA, Charalambous CP. Adhesive capsulitis of the shoulder and diabetes: a meta-analysis of prevalence. *Muscles Ligaments Tendons J*. 2016;6(1):26–34.
11. Cho CH, Bae KC, Kim DH. Treatment strategy for frozen shoulder. *Clin Orthop Surg*. 2019;11(3):249–257. doi:10.4055/cios.2019.11.3.249
12. Millett PJ, Gobezie R, Boykin RE. Shoulder osteoarthritis: diagnosis and management. *Am Fam Physician*. 2008;78(5):605–611.
13. Ansok CB, Muh SJ. Optimal management of glenohumeral osteoarthritis. *Orthop Res Rev*. 2018;10:9–18. doi:10.2147/ORR.S134732
14. Nho SJ, Strauss EJ, Lenart BA, et al. Long head of the biceps tendinopathy: diagnosis and management. *J Am Acad Orthop Surg*. 2010;18(11):645–656. doi:10.5435/00124635-201011000-00002

15. Sansone V, Maiorano E, Galluzzo A, Pascale V. Calcific tendinopathy of the shoulder: clinical perspectives into the mechanisms, pathogenesis, and treatment. *Orthop Res Rev*. 2018;10:63–72. doi:10.2147/ORR.S138225

16. Uhthoff HK, Loehr JW. Calcific tendinopathy of the rotator cuff: pathogenesis, diagnosis, and management. *J Am Acad Orthop Surg*. 1997;5(4):183–191. doi:10.5435/00124635-199707000-00001

17. Noël E. Treatment of calcific tendinitis and adhesive capsulitis of the shoulder. *Rev Rhum Engl Ed*. 1997;64(11):619–628.

18. Yamamoto A, Takagishi K, Osawa T, et al. Prevalence and risk factors of a rotator cuff tear in the general population. *J Shoulder Elbow Surg*. 2010;19(1):116–120. doi:10.1016/j.jse.2009.04.006

19. Weber S, Chahal J. Management of rotator cuff injuries. *J Am Acad Orthop Surg*. 2020;28(5):e193–e201. doi:10.5435/JAAOS-D-19-00463

20. Farrar NG, Malal JJG, Fischer J, Waseem M. An overview of shoulder instability and its management. *Open Orthop J*. 2013;7:338–346. doi:10.2174/1874325001307010338

21. Gutkowska O, Martynkiewicz J, Urban M, Gosk J. Brachial plexus injury after shoulder dislocation: a literature review. *Neurosurg Rev*. 2020;43(2):407–423. doi:10.1007/s10143-018-1001-x

22. Orlinsky M, Shon S, Chiang C, Chan L, Carter P. Comparative study of intra-articular lidocaine and intravenous meperidine/diazepam for shoulder dislocations. *J Emerg Med*. 2002;22(3):241–245. doi:10.1016/s0736-4679(01)00475-9

23. Alkaduhimi H, van der Linde JA, Willigenburg NW, van Deurzen DFP, van den Bekerom MPJ. A systematic comparison of the closed shoulder reduction techniques. *Arch Orthop Trauma Surg*. 2017;137(5):589–599. doi:10.1007/s00402-017-2648-4

24. Polyzois I, Dattani R, Gupta R, Levy O, Narvani AA. Traumatic first time shoulder dislocation: surgery vs non-operative treatment. *Arch Bone Jt Surg*. 2016;4(2):104–108.

25. Kancherla VK, Singh A, Anakwenze OA. Management of acute proximal humerus fractures. *J Am Acad Orthop Surg*. 2017;25(1):42–52. doi:10.5435/JAAOS-D-15-00240

26. Gupta AK, Harris JD, Erickson BJ, et al: Surgical management of complex proximal humerus fractures–a systematic review of 92 studies including 4500 patients. *J Orthop Trauma*. 2015;29(1):54–59. doi:10.1097/BOT.0000000000000229

27. Kim SH, Szabo RM, Marder RA. Epidemiology of humerus fractures in the United States: nationwide emergency department sample, 2008. *Arthritis Care Res* (Hoboken). 2012;64(3):407–414. doi:10.1002/acr.21563

28. Osterhoff G, Morgan EF, Shefelbine SJ, Karim L, McNamara LM, Augat P. Bone mechanical properties and changes with osteoporosis. *Injury*. 2016;47 Suppl 2(suppl 2):S11–S20. doi:10.1016/S0020-1383(16)47003-8

29. Neer CS II. Displaced proximal humeral fractures. I. Classification and evaluation. *J Bone Joint Surg Am*. 1970;52(6):1077–1089.

30. Postacchini F, Gumina S, DeSantis P, Albo F. Epidemiology of clavicle fractures. *J Shoulder Elbow Surg*. 2002;11(5):452–456.

31. Gutiérrez S. CORR Insights®: what regions of the distal clavicle have the greatest bone mineral density and cortical thickness? A cadaveric study. *Clin Orthop Relat Res*. 2019; 477(12):2733–2734. doi:10.1097/CORR.0000000000001021

32. Kim W, McKee MD. Management of acute clavicle fractures. *Orthop Clin North Am*. 2008;39(4):491–505. doi:10.1016/j.ocl.2008.05.006

33. Jeray KJ. Acute midshaft clavicular fracture. *J Am Acad Orthop Surg*. 2007:15(4):239–248. doi:10.5435/00124635-200704000-00007

34. Sembrano JN, Yson SC, Kanu OC, et al. Neck-shoulder crossover: how often do neck and shoulder pathology masquerade as each other? *Am J Orthop (Belle Mead NJ)*. 2013;42(9):E76–E80.

35. Caridi JM, Pumberger M, Hughes AP. Cervical radiculopathy: a review. *HSS J*. 2011;7(3):265–272. doi:10.1007/s11420-011-9218-z