Osteoarthritis

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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

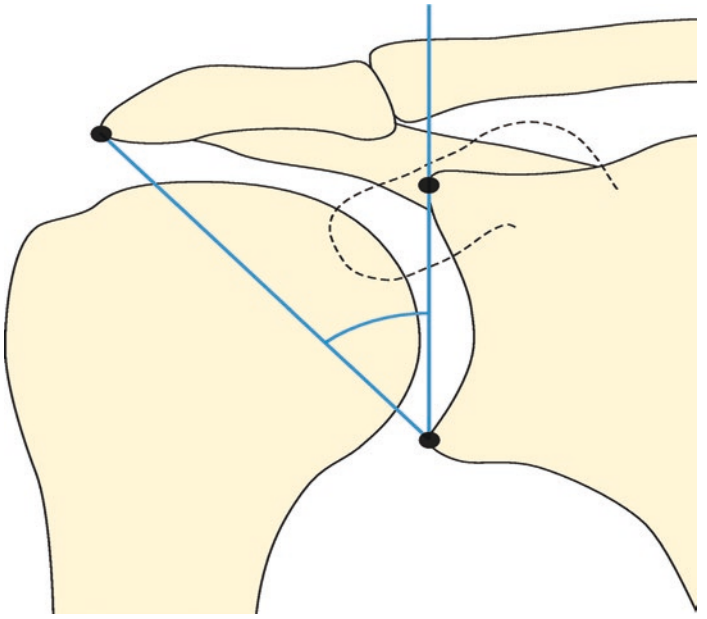

Fig. 6.1 Showing measurement of critical shoulder angle [6]

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.

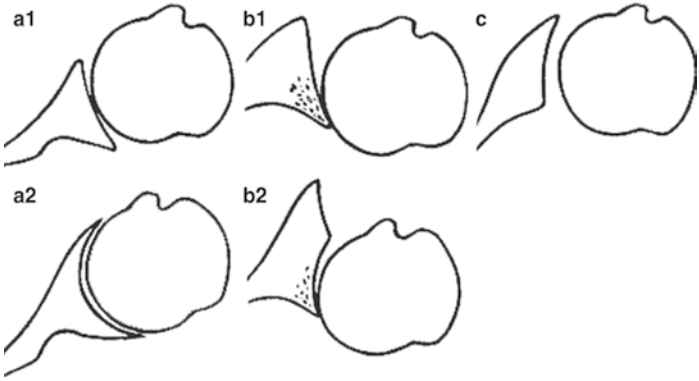

Fig. 6.2 Walch classification for glenoid wear patterns and glenoid bone loss [7]. (**a1**) mild central erosion, (**a2**) moderate central erosion with humeral head protrusion into glenoid, (**b1**) posterior joint space narrowing with subchondral sclerosis, (**b2**) biconcave morphology of glenoid,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

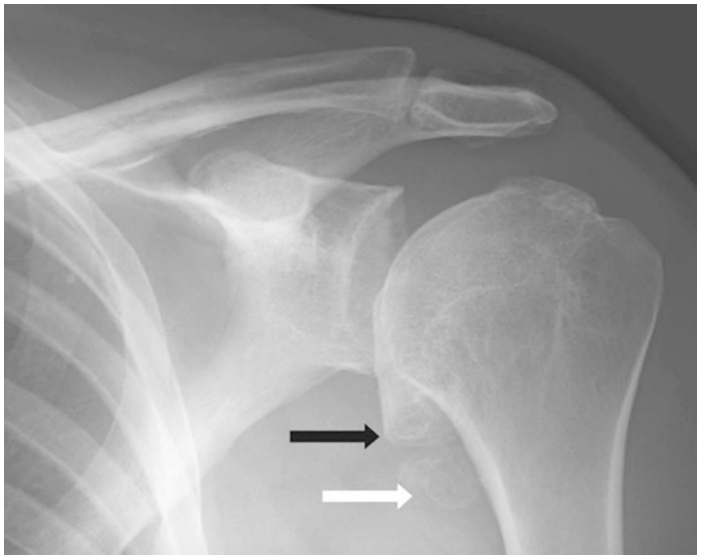

Fig. 6.3 Degenerative changes of the glenohumeral joint, including osteophytes medially (black arrow) and inferiorly (white arrow) shown in the shoulder X-ray [9]

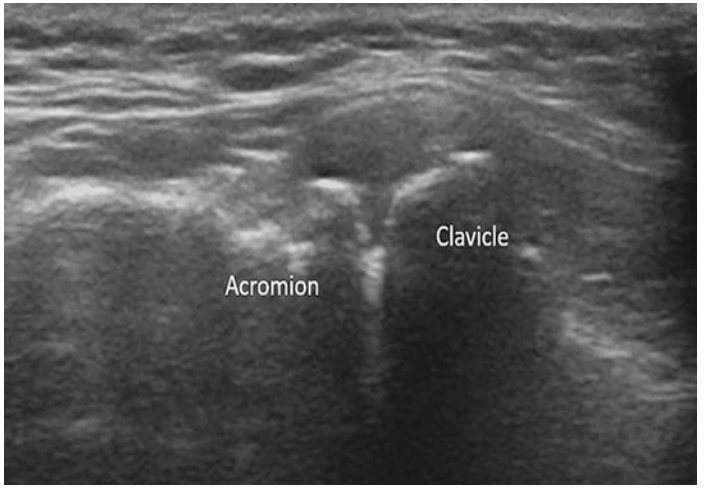

Fig. 6.4 Ultrasound scan revealing superficial osteophytes, joint-space narrowing, and capsular hypertrophy [10]

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

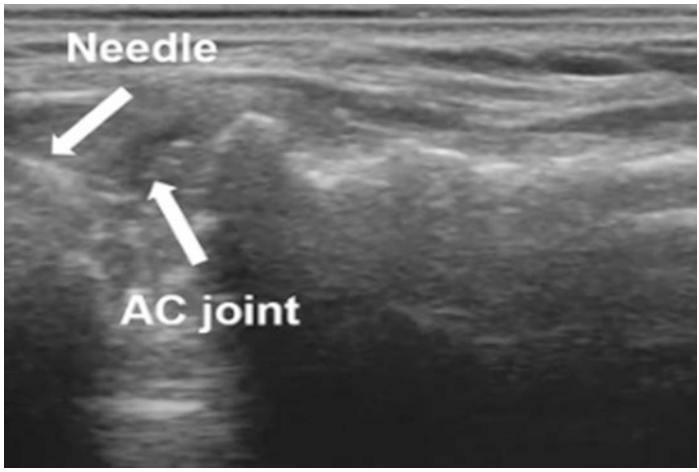

Fig. 6.5 Ultrasound-guided, AC joint injection with needle tip being visualized using the in-plane technique [10]

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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