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ORIGINAL ARTICLE

Glenohumeral osteoarthritis: overview, therapy, and rehabilitation

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ABSTRACT

Purpose: Glenohumeral osteoarthritis (GHOA) is a common cause of pain and functional disability of the shoulder. Despite the limited evidence, there are several options for the treatment of this pathology. The aim of this article is to provide current information on the characteristics of the disease and the pathophysiology, evidence based on medical and surgical treatments with emphasis on the rehabilitation process.

Methods: It was performed with an extensive literature review, mainly clinical practice guidelines, randomized controlled trials, reviews, focusing on the rehabilitation management.

Results: There are few clinical practice guidelines that address GHOA as a pathology with unique characteristics. Evidence based treatment recommendations are mostly supported by low-quality evidence and experts' opinions, with few high levels of evidence studies guiding treatment decisions.

Conclusions: Despite the lack of good quality evidence, rehabilitation programs have proven to be efficient and reliable, and this revision provides information and recommendations in this field.

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➤ IMPLICATION OF REHABILITATION

- Glenohumeral osteoarthritis is a common cause of pain and functional disability of the shoulder
- There are few clinical practice guidelines that address Glenohumeral Osteoarthritis as a pathology with unique characteristics, and recommendations for rehabilitation and therapeutic exercise are poor
- The paper provides current information on the characteristics of the disease, its rehabilitation process, and could be of interest for rehabilitation professionals to direct their practices in this field

Introduction

The aim of this review is to provide current information on the Glenohumeral Osteoarthritis (GHOA) characteristics, also evidence based on conservative and surgical treatments, with emphasis on the rehabilitation process, and the exercise programs.

Osteoarthritis (OA) is a complex joint disease of multifactorial etiology, which represents a set of pathologies with clinical manifestations and morphological and radiological changes. These include damage to the cartilage structure, an increase in mechanical stress, subchondral bone changes, inflammation, synovial proliferation, and thickening and scarring of the joint capsule and ligaments.[1,2]

OA begins when there is disequilibrium in the state of the dynamic balance between cartilage catabolism and the repair mechanisms. As a result, secondary phenomena occur when the mechanical joint load increases;[3] however, it has recently been described how other tissues, such as subchondral bone and synovial membrane, participate actively in the progressive onset of the disorder, leading to a complex pathophysiological process that has not been fully explained till date.[4]

OA can arise in any diarthrodial joint, although it is more prevalent in femorotibial, patellofemoral, coxofemoral, interphalangial, or facet joints.[5] OA of the GH joint significantly affects quality of life and performance of activities of daily living (ADLs). This invests it with enormous clinical importance due to the potential disability that it can generate.

The GH joint possesses a small surface contact area between the humeral head and the glenoid; tendons, muscles, and ligaments maintain joint congruence and stability. These biomechanical characteristics confer not only upon the joint and its distinct position as the most mobile, but also upon the most unstable joint of the human body. Under normal conditions, the humeral head articulates with the glenoid cavity through a smooth and well-lubricated cartilaginous surface.[6] The GH joint has a near frictionless articulation and terminal end range of motion and is established by ligament constraint.[7]

Progressive degeneration of joint cartilage results in an abnormal distribution of loads in the GH joint and successive adaptive changes in the subchondral bone, namely the wearing down of the humeral head and glenoid socket that gradually creates deformity of the boney articulation and limits joint movement.[8]

Cartilage loss typically begins in the posterior section of the glenoid and at the center of the head, with the formation of osteophytes around the anatomical humerus neck. These osseous changes often give rise to the loss of the humeral head's central

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position with respect to the glenoid, which develops posterior subluxation that avoids concentric rotation. Osteophytes formed around the glenoid and the humeral head can mechanically block rotations and increase bone volume within the capsule.[9]

There are few articles that address the GHOA and recommend specific rehabilitation programs, this paper is addressed to rehabilitation professionals who can have essential information on the pathology and take it to their clinical practice.

Epidemiology

As in other forms of OA, GHOA is more common in women,[10] and its incidence rises with aging, with an exponential increase after the age of 50 years.[11] There are scarce studies that report the epidemiology of GHOA. One Asian study found a radiological prevalence of 16.1% in subjects aged over 65 years, lower than what had been reported in other parts of the body, yet still elevated.[12]

Risk factors

OA can be classified as primary when there is no single explanatory factor, and as secondary when it is a direct result of a previous alteration.[13] Around 90% of cases are primary and a result of the interaction of various risk factors, such as repetitive motion injuries due to shoulder overuse, occupations that involve excessive use of upper extremities, engaging in sports with overhead movements, such as baseball, tennis, or basketball, a history of trauma or luxation, obesity and, as in all forms of OA, an important genetic component that contributes to the pathogenesis.[12,14] This revision focuses on primary GHOA.

Rotator cuff tears are not commonly associated with OA, but they are frequent in cases of inflammatory arthritis, and the rule in cases of cuff tear arthropathy.[14]

Classification

Primary OA

Primary OA or GH degenerative joint disease is a set of pathologies with common pathophysiology, articular cartilage damage and dense subcondral bone, osteophytes, posterior glenoid erosion and posterior displacement of the humeral head; without prior injury or surgery.

Secondary OA

A. Post-traumatic – comes from a prior injury that causes imbalance of normal charges distributions in the articular surface, with an accelerated degeneration as a mechanic effect.
B. GH instability – arthritis caused by abnormal joint movement secondary to instability, or after surgical repair for GH instability.
C. Chondrolysis – could be idiopathic or caused after intra-articular infusion of local agents that can lead to chondrocytes and extracellular matrix destruction.
D. Dysplasia – developmental defect, resulting in deficient posterior inferior glenoid, retroversion and posterior instability.

This classification was modified by Matsen (2013).[15]

Other forms of inflammatory arthritis such as autoimmune arthritis (rheumatoid arthritis, lupus), septic arthritis, avascular necrosis of the humeral head, and rotator cuff tear arthropathy can cause severe articular destruction, but the pathophysiological processes are different from OA, so they must be considered as alternative diagnostics.

Clinical profile

Patients typically present shoulder pain characterized by gradual onset and chronic evolution. The pain progressively worsens and is referred as deep and oppressive. It can be reported as localized in the deep and posterior parts of the shoulder, although the location can be unspecified and associated with the movement and use of the joint. In more advanced stages, the pain can be sufficiently severe to wake the patient at night, and can be accompanied by crepitus, rigidity, and joint movement limitation. If pain and joint limitation progresses, could result in a loss in the capacity of the patient to perform basic ADLs, such as eating, dressing, and self-care.[16]

The clinical interview should include duration of symptoms, the mechanism that triggered the onset, surgical or traumatic history, and degree of functionality that is compromised.[17]

The physical examination should reveal pain upon palpation of the anterior and posterior regions of the shoulder that contain the swollen synovium of the GH joint and the protruding boney osteophytes. There exists a progressive loss of mobility in the majority of planes and variable degrees of hypotrophy due to disuse.[17,18] With respect to range of motion, the specific planes of limited movement can correspond to the areas affected by the capsule. The scapula movement is also altered because abnormal substitution patterns arise as a compensatory mechanism, with an increase in scapulo-thoracic rotation.[19] The contracture of the anterior capsule due to posterior subluxation can cause loss of external rotation, which is the first movement affected by GHOA.[20]

Clinical evaluation

It is important to identify the sites of pain; detect contractures or muscle spasms in the trapezius, deltoids, brachial biceps, pectoral muscle, or the shoulder girdle muscles. It is also necessary to evaluate passive and active shoulder movement. In OA, both of these will be diminished and painful; in patients with important joint limitation, it is convenient to evaluate the movement in both decubitus and seated positions. If pain is only felt during active movement; other diagnoses, such tendonitis of the long head of biceps, rotator cuff tears, subacromial or subdeltoid bursitis, should be considered. Another differential diagnoses includes subacromial impingement, adhesive capsulitis, and labral or cartilage injuries, acute or chronic cervicalgia with shoulder radiation pain, although all of these could possible present in conjunction with OAGH. Frequently, there is an association with acromio-clavicular OA that also produces localized pain.[17]

Evaluation of muscle strength should include all scapular girdle and deltoid muscles. The loss of scapular rhythm can be noted with the patient by elevating their extremities in flexion and abduction. This provides valuable information concerning the scapular girdle weakness, which requires specific rehabilitation focus. Stability is rarely compromised in primary GHOA, except in cases of cuff tear arthropathy.[21,22]

Medical imaging

Standardized radiological images used to evaluate GHOA include Anteroposterior (AP), true AP, and axillary and scapular in Y views. These projections display the joint space and permit the evaluation of the affliction's degree. Key elements include reduction in the GH space, irregularities in surfaces, the presence of

Figure 1. AP view of a normal and a OA GH joint, note the important reduction in space, irregularities in the surfaces, and the subcondral sclerosis in the humerus and glenoid.

osteophytes, wearing down of the humeral head, bony sclerosis in the humerus or glenoid, subluxation or bone malformation and subcondral cysts,[23] as seen in Figure 1.

True AP places the humeral head in its maximum profile and centers the humerus in the glenoid socket. This view shows joint space narrowing, surface's irregularities, and/or superior humeral migration, which are indicative of chronic massive rotator cuff tears.[9]

The axillary view is crucial for evaluating glenoid erosion, abnormal glenoid version, or humeral subluxation. Other characteristics that should be noted in the radiographic evaluation include the presence and localization of osteophytes, bone cysts, the degree of medial glenoid erosion, and the presence of cystic changes in the greater tuberosity, which can indicate rotator cuff tear,[23] as tuberosity cysts do not indicate cuff tear, but can be associated with cuff disease. Peripheral osteophytes in the glenoid and humeral head result in the wearing down of joint surfaces, and disuse osteopenia could also be observed around the greater tuberosity.[24,25]

Other studies are not necessary for the classification of the disease. However, Ultrasound (US), Magnetic resonance imaging (MRI), or Computed tomography (CT) scans should be obtained if dealing with certain cases such as the following: secondary OA; in the presence of any composition or aggregate inflammatory pathologies; to verify rotator cuff integrity, or in the case of considering the need for surgical management. CT scans are useful for evaluating anatomy and glenoid version, joint surfaces, or bone defects, or in cases where bone quality or quantity could alter surgical treatment. Also, there is the possibility of three-dimensional (3D) reconstruction in the CT for displaying 3D relationships that maintain the structures and the degree of joint involvement, as seen in Figure 2. MRI or US images can additionally be employed to confirm tendon integrity, ligaments, labrum, localized cartilaginous injuries, atrophies, or fatty degeneration of the rotator cuff or deltoid muscles. These are indicators of the chronicity of the pathology and surgical prognosis, taking into account that MRI possesses greater sensitivity for visualizing soft tissues than US, but the latter permits dynamic evaluation of the joint.[26–28]

Recently, morpho-functional studies have described the cartilage structure as the time of relaxation in T2 or the T1rho of the MRI. This provides information on very incipient disease stages, even though these images have been utilized principally in research studies.[29,30]

Figure 2. CT 3D reconstruction of OAGH. It is useful to observe glenoid wear pattern and to assess for posterior humeral subluxation. The humerus can be removed and the glenoid 3D visualized.

Classification criteria

In contrast with OA of the knees, hips, or hands, GHOA does not have specific classification criteria proposed by associations such as the European League Against Rheumatism (EULAR) or the American College of Rheumatology (ACR). For radiological stratification, as in other OA locations, the classification proposed by Kellgren-Lawrence of grades 0-4 is used as follows: grade 0 is the absence of radiographic signs of arthrosis; grade 1, uncertain joint-space narrowing and the possible presence of osteophytes; grade 2, definite osteophytes and narrowing of joint space; grade 3, osteophytes, important narrowing, subchondral sclerosis, and possible bony deformity, and grade 4, multiple osteophytes, marked narrowing of the space, severe sclerosis, and definitive bony deformity.[31]

Another radiological classification specific for dislocation arthropathy of the shoulder is that of Samilson-Prieto.[32] The latter takes into consideration osteophyte size, their location inferior to the humeral head or in the glenoid, presence of irregularities in

the joint surface observed in the true AP view in the glenohumeral joint and dislocation arthropathy of the shoulder.[33]

The classification is as follows:

Mild arthrosis: Osteophyte on the inferior region of the humeral head and/or glenoid, <3 mm

Moderate arthrosis: Osteophyte measuring from 3 to 7 mm in the inferior region, with slight irregularity on the joint surface

Severe arthrosis: Osteophyte in the inferior region, >7 mm, with a reduction in joint space and bone sclerosis.

Treatment

Therapeutic management should be started with conservative methods in order to reduce pain and improve range-of-motion independently of the severity of the disease or the initial indication of surgery, because pre-surgical therapeutic management can improve postoperative prognosis.[10]

Lifestyle changes, activity modification, and adoption of joint-hygiene measures comprise the first actions which the patients have to take. If possible, activities that involve weight bearing or impact on the joint should be avoided. Information on the disease, potential treatments, and prognosis should be provided. The patient should also be sensitized to the need for adherence to an exercise program, prescribed in an individualized manner.[34,35]

The use of pharmacological analgesics and Nonsteroidal anti-inflammatory drugs (NSAID) is indicated in GHOA. As with other forms of OA, the recommendation is to begin with Acetaminophen, adapting the treatment to the response. Patients without relevant comorbidities can opt for treatment with nonselective NSAID + Proton pump inhibitor (PPI) or COX-2 selective inhibitor NSAID.[10,34,36]

If pain control continues at a sub-optimal level, oral opiates can be employed concomitantly for short time periods in a symptomatic manner, although there are no established recommendations for their use for GHOA.[35,36]

There are no recommendations for the use of glucosamine, chondroitin sulfate, or a combination of both, as well as of vitamin supplements, soybean and avocado unsaponifiables, or diacerein due to the lack of good quality evidence. There is scarce evidence concerning their use at this site, although evidence exists of their use in other sites (knees and hips) as an adjuvant for pain management, but not as cartilage structure modifiers.[34,36]

The use of other medications such as bisphosphonates is not advised because they have not been demonstrated as efficacious in this condition and at this location.[34]

Intra-articular steroid administration can be performed in cases of persistent pain for patients who were unresponsive to oral therapy; it can be useful at the beginning of exercise and physiotherapy programs, although the recommendation is not conclusive.[35] The American Academy of Orthopedic Surgeons (AAOS) recommends the intra-articular use of High-molecular-weight (HMW) hyaluronates for pain relief in a symptomatic and temporal manner. Reports exist regarding the usefulness of botulinum toxin or intra-articular hydrolyzed collagen, although recommendations have not been established for their use.[36]

Due to the lack of evidence on its usefulness, currently there are no recommendations for the intra-articular administration of other substances such as platelet-rich plasma, growth factors, stem cells, or others that are still in experimental phases.

Rehabilitation

Therapeutic exercise programs are recommended to be performed along with articular care recommendations, and concomitant use of oral or articular medications.[36,37] All should emphasize the patients' individual needs, their clinical status, and their physical capabilities at the time of evaluation. If possible, the program should begin within an institution in cases of patients who are in contact with therapeutic exercise for the first time, because these patients need to learn correct techniques, or with patients with painful symptomatology who would benefit from pain control through physical agents (surface heat, low-level laser therapy, therapeutic ultrasound, electrotherapy, among others).[38]

There are no recommendations concerning the superiority of a physical agent for the treatment of GHOA, which means that prescriptions are based on the Attending Physician, who must always take the symptomology into account. Superficial or deep heat, electrotherapy, or phototherapy is recommended as an adjuvant in pain management, relaxing soft tissues and to reduce inflammatory process.[36]

Exercise programs should include joint mobilization techniques (active-assisted, auto-assisted, or passive). It is preferable to start with passive mobilization techniques with the patient in supine decubitus. Movements should include shoulder flexion, abduction, adduction, internal and external rotation and extension. It should be performed to patient's tolerance and slowly progressed to gain mobility. Patients with better earnings mobility exercises may progress to semi-sitting and sitting and start with mobility active assisted or self-assisted, namely the patient performs the active movement and physical therapist assists the movement, trying to improve mobility. Patients may also be aided with the contralateral arm to increase the range of movement or devices such as canes or sticks assisting the movement with the contralateral arm.[39–41] Mobility exercises are illustrated in Figures 3 and 4.

At the same time, it should start a stretching program assisted by the therapist with the aim of achieving major elasticity of tissues (muscles, tendons, and ligaments) and decrease contractures. Must perform stretching exercises for the shoulder joint capsule, the deltoids, rotators, trapezius, latissimus dorsi, the brachial biceps and neck muscles such as the sternocleidomastoid, scalene and cervical spine extensors;[42] examples are provided in Figure 5.

The technique should include proper positioning of the arm and should be aided by the physiotherapist; to achieve the greatest stimulus stretching should be done three to five repetitions, holding between 20 and 30 s. Self-assisted stretching techniques must be taught so that the patients are able to reproduce at home.

Once worked mobility and elasticity and the pain has subsided, it must begin with strength training with progressive resistance according to the tolerance for the deltoid and scapular girdle, and scapular balance work.

Initial exercises can be performed with isometric or isotonic contractions unloaded, and then progress to patient tolerance weight exercises. Weight exercises can be carried out using dumbbells, pulleys, balls, or rubber bands, and supervised by the therapist, with concentric contraction mainly in 1–2 sets of 10 repetitions, to tolerance. Weight can be increased every 2–4 weeks if the patient tolerated.[41] Strengthening training examples are provided in Figure 6.

Although there is insufficient evidence to recommend the use of eccentric contraction exercises, they theoretically could also be useful in subjects with OAGH.[43]

Programs that have proven beneficial for patients with OAGH last at least 12 weeks. It is recommendable that the patient incorporates therapeutic exercise as part of their lifestyle.[44–46]

Open and closed kinetic chain exercise techniques can be combined with strengthening. In patients with muscle weakness or important atrophy, strengthening can be performed in decubitus

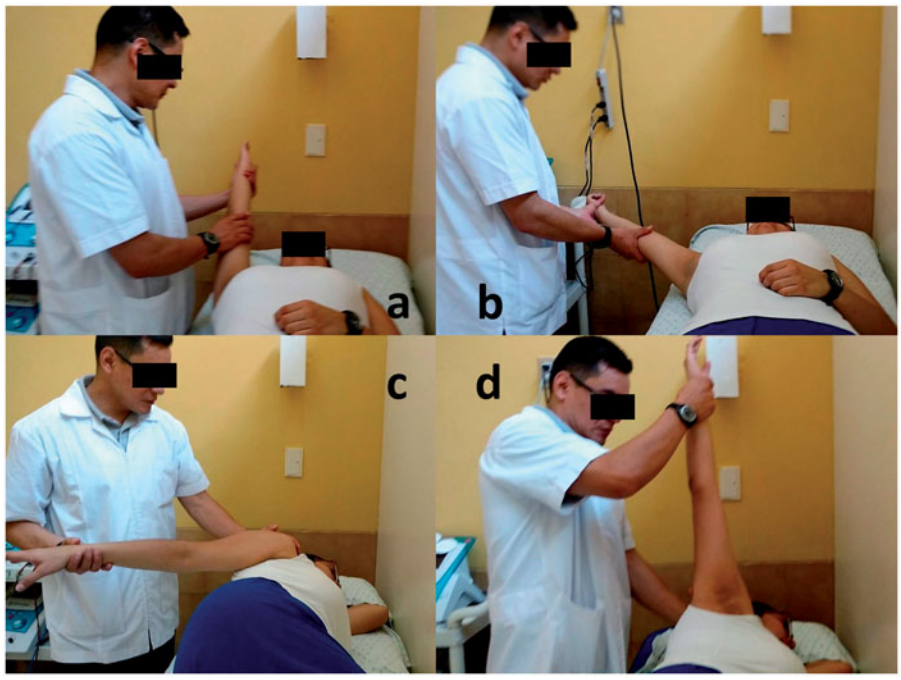

Figure 3. Joint mobilization techniques (active-assisted or passive), in dorsal and lateral decubitus, with progressive gaining mobility, in shoulder (a) flexion, (b) abduction in dorsal decubitus, (c) extension, and (d) abduction in lateral decubitus.

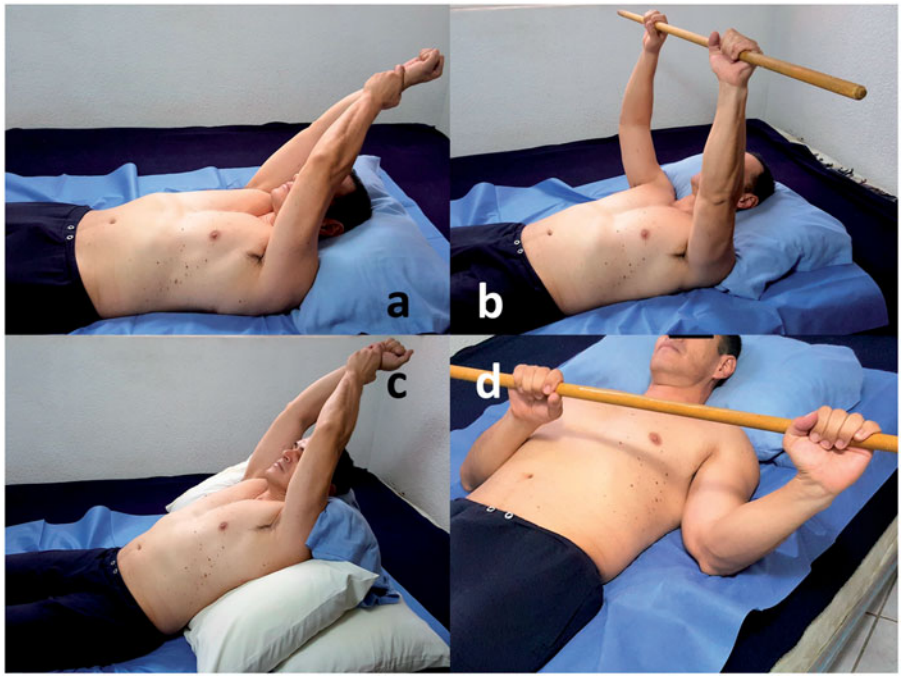

Figure 4. Self-assisted home mobility program; (a) contralateral arm assists injured shoulder in flexion in dorsal decubitus, (b) cane or stick assisted shoulder flexion, (c) self-assisted shoulder flexion with progression to semi seated position, (d) rotations assisted by a stick.

and slowly progress to a seated position. These programs will be progressive and adapted to the needs, expectations, and objectives of the patient's rehabilitation.[47]

The patients must be able to continue their training at home for an indefinite time. Programs that include a combination of exercise techniques are preferred to those that only include solely one. In addition, a low-to-moderate-intensity aerobic exercise program without joint impact is recommended.[34,48] When these methods fail, good quality evidence has shown that blockage of the suprascapular nerve could be useful.[48]

Surgical management

Arthroscopic procedures are frequently used prior to an arthroplasty. These include arthroscopic exposure, capsule release, or removal of intra-articular fragments, as well as cartilage repair techniques such as microfracture, chondroplasty, or labral repair.[49,50] Chondrocyte transplants are used for isolated injuries but not for GHOA.

Although the AAOS has not found conclusive evidence on their usefulness, these joint preserving techniques without the use of

Figure 5. Stretching program assisted by a therapist, (a) for deltoid and biceps, (b) latissimus dorsi and triceps, (c) joint capsule, dorsal and deltoid, and (d) pectorals.

Figure 6. Strengthening program using a ½ kg ball, for (a) shoulder flexors, (b) internal rotators, (c) external rotators, and (d) abductors.

arthroplasty are commonly employed in young subjects or in those with incipient stages of the pathology.[35] Total arthroplasty or hemiarthroplasty provides significant pain relief and functional improvement, especially in senior populations. These joint replacements are indicated in patients with temporary or inadequate response to conservative treatment, with important functional limitation, and with severe lesions such as massive rotator cuff tears or osteonecrosis.[7]

A total shoulder arthroplasty is indicated in patients with moderate to severe GHOA with complete loss of humeral and glenoid cartilage and an intact rotator cuff or reparable rotator cuff tears. Patients with irreparable tears or with rotator cuff tear arthropathy may be treated with reverse shoulder replacement. In younger adult patients, active in heavy physical labor or high impact sporting activities, hemiarthroplasty may be indicated over total shoulder replacement to prevent early loosening of the glenoid component replacement. In patients with intact glenoid cartilage, a significant humeral head cartilage loss or collapse from avascular necrosis a hemiartroplasty is also preferred.

Functional results are adequate in the majority of patients. While due to access and costs, surgery should be considered completely elective, the decision should be based on an informed

estimation of the benefits of the surgical treatment relative to the impact of the OA on the well-being, functionality, and general health of the patient and the risks associated with the surgical procedure. The surgical treatment should be directed, as much as possible, toward joint preservation.[51]

Post-surgical rehabilitation

The degree to which surgical restoration of shoulder biomechanics can be translated into clinical restoration of shoulder movement and functionality depends critically on the rehabilitation process.

An appropriate exercise technique is key to avoid injuries and achieve progressive advancements.

Rehabilitation protocols and programs must depend on the type of surgery performed and its objective. Rehabilitation following total shoulder arthroplasty or hemioarthroplasty should include an early passive movement program within the first week to prevent the stiffness. Patients recovering from reverse shoulder arthroplasty could be immobilized for 2–6 weeks post-operatively to prevent reverse shoulder dislocation. During post-arthroplasty recuperation, patients should immediately be placed in a passive movement program during the first days. This facilitates early movement during the initial recovery when patients are still incapable of actively participating in rehabilitation. Special care should be taken regarding the surgical technique utilized and the approaches and repairs performed, with special attention to surgeon guided limits to passive external rotation to prevent tearing of the surgical repair of the subscapularis tendon. The subscapularis is the structure that is cut to enter the shoulder during replacement and later repaired at the closure of the case. Excessive passive external rotation pulls apart this subscapularis repair and excessive force of active internal rotation also may jeopardize the subscapularis repair due the subscapularis muscle pulling apart the repair.[52]

It is always recommended to start exercises in a supine position, thereby eliminating the effect of gravity, and stabilization of the scapula to prevent compensation by the scapulo-thoracic joint. Gentle stretching in crossed adduction above the body and internal rotation begin within a functional range. To avoid contractures, it is necessary to always include movement of the elbow, wrist, and fingers. These exercises are performed several times a day during the first 6 weeks, time when movement restrictions are generally eliminated. Patients continue progressing their program until their movement is symmetrical with the unaffected side. In addition, isometric exercises should be begun and patients should be encouraged to reintegrate themselves gradually into their habitual self-care activities.

Strength training with progressively resistant isotonic exercises is recommended three months after the surgery, and should be focused on the rotator cuff, deltoids, and the scapular muscles. Progressive advancements can be carried out for to 6–9 months, despite their having been reported up to more than 1-year post-surgery. These timeframes can vary according to the individual case of each patient and the protocol utilized.[53–55]

Rehabilitation protocols can also be focused on preventing muscle atrophy and abnormal substitution patterns, which can be traced to chronic maladaptation to the OA. More emphasis should be placed on the resistance than on the strength.[56]

Continuing with the program at home is essential because it allows more frequent executions of the exercises, stimulates the patients to take a more active role in their recovery, and permits exercise during years following the surgical procedure.[49]

It is essential to inform patients about the care that they should take with the use of hemi- or total arthroplasty prosthesis in order to avoid replacement after a short time.

Oral antibiotic treatment prior to dental work or colonoscopy is recommended for at least the first few years after arthroplasty to prevent seeding an infection. Also, patients with total shoulder arthroplasty should avoid high impact sports like boxing, heavy weight lifting, shotgun or high powered rifle shooting as these activities likely cause early loosening of the glenoid component replacement.

Outcomes

The effectiveness of treatment should be evaluated in an objective manner. The outcome measures that can be reported are pain using a visual analog scale (VAS), mobility through electronic or manual goniometry, muscle strength using an isokinetic, isometric, or isotonic dynamometer and/or functionality and patient satisfaction with multi-modal scales as the Constant-Murley Scale, UCLA Shoulder rating scale, the Shoulder Pain and Disability Index (SPADI), The Disabilities of the Arm, Shoulder and Hand (DASH) Score, and/or the American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form (ASES).[57]

Conclusions

GHOA is a highly disabling pathology with elevated prevalence and incidence. There are few studies with high level of evidence to support treatment decisions as most clinical practice guidelines rely on empiric evidence and expert opinion. GHOA classification criteria is lacking from societies such as the OARSI, EULAR, and AAOS. It is necessary to understand the pathophysiological mechanisms of this disease, as well as its therapy and rehabilitation, in order to offer patients, the best care and to limit disability.

Disclosure statement

All the authors declare not having any conflict of interest. All figures have signed an informed consent by the participants to be used in this publication.

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