Clinical Differences between Shoulder Impingement Syndrome and Frozen Shoulder

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Abstract: The prevalence of shoulder pain in the world is around 7-34%. Shoulder pain affects the clinical and functional aspects. This will decrease the ability to perform daily activities and quality of life thus it is important to be prevented and treated. One shoulder pain that occurs often is shoulder impingement syndrome. This syndrome should be distinguished from adhesive capsulitis so rehabilitation can be held properly and quickly. The prevalence of Shoulder Impingement Syndrome is 44-65% of all shoulder pain while adhesive capsulitis is 2-5%. This syndrome can be distinguished from adhesive capsulitis based on clinical and support examination. The clinical differences in this syndrome with adhesive capsulitis include the type of shoulder pain and limitation of ROM of the shoulder joint.

Keywords: impingement, adhesive capsulitis, frozen shoulder, symptom

1. Introduction

Most people have ever complained in the bones, joints, and muscles, especially the elderly. Elderly people generally begin to experience degeneration in organs, including joints, thus decrease the activity. A decrease in activity accompanied by a lack of exercise causes the joint to become stiff and painful therefore limit the movement. Joint stiffness in the elderly is often felt in the shoulder and knee area.

Shoulder pain is the third most common complaint in health services [1]-[4]. The prevalence of this pain ranges between 16-30 % every month [4]. The high morbidity due to shoulder pain causes a decrease in the ability to perform daily activities, work and quality of life as well as an increase in medical costs [1]-[3].

The most common cause of shoulder pain is a defect in the rotator cuff and shoulder impingement syndrome (SIS), which is 44-65% of all shoulder pain [4], [5] and frozen shoulder (adhesive capsulitis), by 2-5% of the general population [6], [7]. Shoulder impingement syndrome is defined as rotator cuff compression and subacromial bursae [3] while frozen shoulder is a state of global range of movement (ROM) shoulder restriction in a capsular pattern [8]. The characteristic of this pattern is a limitation of the movement, especially in passive lateral rotation and abduction [8].

Failure to diagnose, identify causes (40%) and technical errors (40%) in SIS can lead to complications, stretch capsules also improper mobilization techniques.[4], [9]. These improper techniques will worsen the stability of the shoulder and reduce the success of rehabilitation [9]. Pain in frozen shoulder can be associated with an increased risk of breast cancer, lung and non-Hodgkin's lymphoma or a complication or a symptom of cancer that can be used to predict or initial diagnosis of cancer [10] thus early diagnosis and rapid also appropriate treatment is needed [1].

There are several differences in treating shoulder pain, depending on the cause. The difference in clinical symptoms of each shoulder pain is important to diagnose and provide an appropriate treatment [11]. This paper discusses clinical differences between SIS and frozen shoulder, according to the large prevalence of SIS and the relationship of frozen shoulder with cancer.

2. Anatomy of the Shoulder Joint

This joint is included in ball and socket joint and constituted a load support joint that has a large range of movement (ROM). The shoulder joint is less stable than the hip joint due to the amount of ROM and inserted part of the humeral head in the glenoid cavity [4], [12]. The stability of the joints is maintained by the tone of the musculotendinous of the rotator cuff, namely: supraspinatus, infraspinatus, teres minor, and subscapularis [12].

3. Movement of the Shoulder Joint

The shoulder joint is the most moveable joint due to the weakness of the joint capsule and the size of the humeral head compared to the glenoid cavity. The movements of these joints include flexion, extension, abduction, adduction, rotation, and circumduction. Lateral rotation of the humerus increases the abduction range. When the arm abucted without rotation, the greater tubercle of humerus converges the coracoacromial arch, thus limiting further abduction. If the arm rotates laterally 180 degrees, the greater tubercle of humerus rotates posteriorly and does not converge the coracoacromial arch then abduction can be continued until the arm is lifted up. Abnormalities such as joint stiffness will limit the movement. These joint was moved by the axioappendicular and scapulohumeral muscles. Other muscles act to prevent dislocation and maintain the humeral head in the glenoid cavity without producing any movement [12].

4. Bursa around the Shoulder Joint

Bursaries that located around the shoulder joint includes subscapular bursa and subacromial bursa. The subscapular bursa is located between the subscapular tendon and neck of the scapula and is associated with the articular cavity of the glenohumeral joint. This bursa protects part of the tendon
that passes through the coracoids process and neck of the scapula. Subacromial bursa is often referred to as subdeltoid bursa. This bursa is located between the acromion, coracoacromial ligament and deltoid superiorly with supraspinatus tendon and the joint capsule of the glenohumeral joint inferiorly. This bursa will facilitate the movement of the supraspinatus tendon [12].

Subacromial space is bounded caudally by the head of the humerus and rotator cuff, cranially is bounded by the osteofibrous roof of the shoulder that consists of acromion, coracoacromial ligament, and coracoid processes. Subacromial space contains subacromial bursa and rotator cuff. Subacromial space has a helper joint between the rotator cuff and the roof of the structure [4].

5. Shoulder Impingement Syndrome (SIS)

Impingement syndrome is a group of symptoms and signs on the shoulder due to inflammation of the rotator cuff tendon and subacromial bursa [5]. The syndrome is a result of a mechanical collision of the rotator cuff (when walking under the coracoacromial ligament) with acromion [5], [13]. This impingement can tear the rotator cuff tendon partially or entirely [1]. Impingement can also occur between the acromion and humeral head. Etiology of SIS is multifactorial thus it is difficult to determine the location of the lesions and effective treatment [5]. Shoulder impingement is classified into primary and secondary. Structural changes in the primary type occur due to intrinsic, extrinsic or combination factors. Thus the subacromial space becomes narrow and presses the rotator cuff mechanically [1], [4].

Secondary impingement occurs due to malfunction (such as muscle imbalance) in positioning humeral head. This condition causes an abnormal placement of the center of rotation at elevation and clamping of the connective tissue [4]. Secondary type underlies the unstable glenohumeral joint and collision between biceps tendon with rotator cuff until rotator cuff tears occur [9]. Structural alteration of the rotator cuff tendon due to acute or chronic processes, such as degenerative changes by age, overuse, repetitive, trauma or tension overload are intrinsic impingement [1], [5]. Smoking is one of the intrinsic predispositions of rotator cuff damage [4]. Clinical symptoms of this syndrome depend on the size of the tear on the rotator cuff muscles so monitoring is needed to view the progress of the tear [5].

Morphological variations and degenerative acromion alteration, abnormal rotator cuffs, attachment or thickening of the coracoacromial ligament, acromioclavicular joint changes, osteophytes, inflammation, and poor scapular muscles or posture can suppress the supraspinatus tendon mechanically [1], [5]. Impingement due to abnormalities of the structures outside the tendon of the rotator cuff is called extrinsic impingement [5].

The extrinsic abnormality is divided into 3 stages. The first stage is characterized by acute inflammation, edema, and hemorrhage at the subacromial bursa and rotator cuff. Usually, this phase was seen in patients less than 25 years old, unilateral and non-traumatic [1], [5], [13]. Pain is felt around acromion and deteriorates when lifting the upper arm [13]. This stage disorder can be cured only by conservative treatment [1]. The second stage is around 25 to 40 years old and histomorphological alterations (fibrosis, swelling and rotator cuff tendinitis) begin irreversibly [1], [3], [5]. The third stage occurs over 40 years old [1], [5]. Chronic alteration begin to increase, such as partial or complete tear of the rotator cuff, tendon rupture of biceps brachii, changes in bone, degeneration of the tendon and osteophyte formation at the coracoacromial arch, therefore narrows the subacromial space [1], [3], [5].

Supraspinatus tendons are the most susceptible to shoulder impingement syndrome due to the avascular area which is often the site of the collision. This area is broader as age increases. The proximity of the supraspinatus muscle with the anterior inferior margins of the acromion in 60-120° of abducted position with 45° internal rotation (lifting the arm above the head, reaching the top of the cupboard, swimming, lifting, throwing or using racquet sports) will narrow the subacromial space. Therefore collision occurs between the major tuberculum humeri with the inferior part of the acromion resulting in pain in the anterolateral shoulder so that external rotation reduces pain [1], [5]. This pain which happens when lying down at night on the anterolateral acromion is a specific sign of SIS. Pain can radiate to the middle from the lateral humerus. There is also the loss of shoulder muscle strength, weakness and loss of upper arm function [3]-[5]. This indicate a rotator cuff injury or supraspinatus tendinitis. Based on physical examination, edema, muscle deformity, and stiffness can be obtained [5]. Diagnosis of this disease can be made through history, physical examination (inspection, palpation, ROM and muscle strength), ultrasound and magnetic resonance imaging [1], [2]. Treatment of this syndrome depends on the stage of the disease and the patient itself. Conservative treatment is used at the initial stage and the next phase is treated by surgery, due to tears of the rotator cuff or acromion or both. Surgery also considered if conservative treatment fails, especially in young sufferers. Medications such as analgesia, steroids, shock-wave therapy, non-steroidal anti-inflammatory drugs, and physical therapy were given for 7-14 days to reduce pain. Steroid use should be limited and given for 3-12 months. Long-term steroid use can result in rotator cuff damage [1]. The prognosis of this disease is bad if the duration of pain is long. Shoulder impingement syndrome can be prevented by doing regular exercise for more than 3 hours per week, at least 10 months a year [13].

6. Adhesive capsulitis on the glenohumeral joint

The attachment of the inflamed glenohumeral joint capsule with the rotator cuff, subacromial bursa and deltoid are called adhesive capsulitis or frozen shoulder [14]. There is a deficiency of synovial fluid, thickening, swelling and attachment of capsule to bone which is easily removed [8], [15]. These capsules will reduce joint space for the humerus thus movement of the shoulder becomes stiff and painful [8]. This disease can recover by itself within 2-3 years but the symptoms persist in 40% patients accompanied by ROM limitations while the disease persisted in 15% patients [16].
Incident of the frozen shoulder is about 3% of the general population. This event occurs mainly in women aged around 40 to 70 years old, in the non-dominant arms, diabetics, and prediabetics. The existence history of frozen shoulder increases the risk of recurrence of this disease both on the opposite and same side, especially in diabetics [17]. The risk of frozen shoulder is also increased in patients with Dupuytren disease, shoulder trauma, cardiac, endocrine and neurological disorders [16]. The disease is difficult to define, cured and explained based on its pathology [14], [15]. Some researchers divide frozen shoulder into 2 types, namely primary and secondary. The main cause of primary frozen shoulder is unclear (idiopathic). While the secondary is associated with trauma to the capsule due to injury or surgery, diabetes, rotator cuff injury, cerebrovascular accident (CVA) and heart disease. This condition will slow healing time and limit the success of healing [11], [15]. Phage and Labbe (2010), in their article, mention that primary frozen shoulder is the result of fibroblast proliferation as an abnormal immune system response to chronic inflammation [11]. Inflammation is suspected from supraspinatus tendinitis but can also spread from other components of the rotator cuff, subacromial bursa, capsule, and extra-capsular ligament. The pathophysiology of frozen shoulder is unclear, it is thought to be a fibromatosis [14]. This characterized by progressive loss of active and fascial movement in the glenohumeral joint due to contractures of the joint [18]. Characteristics of this disease is consisted of 3 phases. The first phase is the pain phase. The shoulder is very painful even in a resting state. This phase lasts for 3 to 6 months. The next phase is the adhesive phase. Pain has decreased but active and passive movements are limited. The last phase is the phase of resolution. Healing begins and the functions return in this phase. These three phases are experienced for approximately 30 months or longer. About 50% patients fail to get complete recovery so ROM is limited [14].

Clinical symptoms of frozen shoulder are important to know thus distinguish it from other shoulder pain [11]. There are progressive pain and limitations of passive movement in capsular patterns (especially abduction and lateral rotation) [15]-[17]. The pain is deep, blunt and cannot localize properly, generally in the anterior or posterior capsule, near the insertion of deltoid muscle [8], [17], [18]. Pain can radiate to the biceps area, become worsen and stiffer when the patient attempts to reach the head and back area. There are also weaknesses, crepitate and sleep disturbances due to pain. Neuropathic symptoms of the forearm and hands occur due to other conditions, such as cervical radiculopathy and non-frozen shoulder [17].

One limitation of the ROM in the adhesive phase is difficult to abduct arm more than 45°. Abduction more than 45° can be done by holding the elevation and rotation of the scapula. There is also a limitation of external rotation, about 50% exceeding abduction and internal rotation [12]. The average of ROM that can be performed on frozen shoulder patients is the abduction of 98°, flexion of 117°, external rotation of 33° and internal rotation of 18° with shoulder abduction of 90°. The limitation of movement is also due to an imbalance of the shoulder muscles, contractures of the shoulder ligament, fascial restriction, muscle tension and trigger points in the muscle. The superior part of the trapezius muscle tends to be more active than the inferior so the scapula becomes unbalanced. There are an increased elevation and upward rotation of the scapula when the glenohumeral joint undergoes elevation in the frontal and sagittal plane (scapula moves upward to the abduction of 60°). This sign called shrug sign. This occurs to compensate for the lack of capsular stretches such as changes in the motor nerve pathways due to lack of adaptation. Patients may experience postural deviation, such as shoulder become more anterior or increased of kyphosis due to pain and limited function of the shoulder muscles [11].

Diagnosis of adhesive capsulitis based on the clinical symptoms, including persistent, severe and prolonged pain (more than 4 weeks) with limited active and passive movements on the shoulder. There is shoulder elevation to anterior of 130°, external rotation of up to 50° and internal rotation of more than L5 [6]. There are several important signs in, namely loss of passive elevation and external rotation, thickening of the joint capsule and synovium up to 4 mm [18]. Ultrasound, MRI, and ultrasound arthography are used to support the diagnosis [16].

The principle of treating adhesive capsulitis is to relieve pain, maintain ROM and restore function. Pain can be relieved by massage, heating, ice, ultrasound, transcutaneous electrical nerve stimulation (TENS) and light amplification by stimulated emission of radiations (LASER). The other method which can be added to the above treatment is immobilization or exercise techniques. Generally, frozen shoulder can heal itself but to regain normal function, physical therapy is needed. This exercise is in the form of stretching shoulder capsules passively in every movement. The Maitland concept has been recognized as a process of examination, assessment, and treatment of neuromusculoskeletal disorders. This concept manipulates physiotherapy. Stage I and II Maitland mobilization techniques are used primarily in treating the limitations of joint movements due to pain. Oscillation can inhibit the perception of pain stimuli by repeatedly stimulating mechanoreceptors that inhibit the nociceptive pathway in the spinal cord or brain stem. This movement helps synovial fluid increasing nutrients to the cartilage. Stage III and IV are mainly used for stretching. Appropriate mobilization techniques used after a thorough examination and assessment [15].

7. Conclusion

It can be concluded that SIS can be differentiated from adhesive capsulitis from their clinical symptoms. In SIS, there are specific pain on the anterolateral shoulder which occur when lying down at night and lifting arm above the head, loss of shoulder muscle strength, weaknesses and loss of upper arm function. The pain can be reduced by external rotation.

The specific signs in adhesive capsulitis are the shrug sign, a persistent, severe and prolonged pain (more than 4 weeks) with limited active and passive movements on the shoulder, loss of passive elevation and external rotation, thickening of
the joint capsule and synovium up to 4 mm, weaknesses, crepitate and sleep disturbances due to pain.

References


