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The Diabetic Shoulder – A Literature Review

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Abstract

Shoulder pain is one of the most common complaints of patients with diabetes that causes motion limitation, functional disability and decreased quality of life. There is higher prevalence of shoulder disorders in patients with diabetes, with adhesive capsulitis (AC) and rotator cuff (RC) tendinopathy being the most common disabling shoulder disorders. The pathophysiology that predisposes patients with diabetes for the development of AC or RC tendinopathy is not well-understood. However, the increased glycosylation of collagen fibers of the joint capsule, tendons and ligaments, and the diabetic microangiopathy might potentially explain the pathological process. Although some of the therapeutic interventions have been shown to be effective in managing shoulder disorders, several studies have reported higher shoulder pain, reduced mobility, poor functional outcomes, and a diminished response to treatment in patients with diabetes than patients without diabetes. In the current literature, there is lack of studies on the best treatment approach for managing shoulder disorders in patients with diabetes. Furthermore, the effect of diabetes on shoulder function after shoulder arthroplasty is not well investigated. Future research is required to examine the effectiveness of different surgical and non-surgical interventions on managing shoulder disorders in patients with diabetes. In addition, more research is required to investigate the impact of diabetes on shoulder recovery and factors predicting shoulder function following shoulder arthroplasty.

Keywords: Diabetes, Shoulder disorders, Therapeutic interventions

Functional Anatomy of the Shoulder Complex

The shoulder complex is composed of three bony structures: the clavicle, scapula, and humerus, which are connected to form three synovial (glenohumeral, acromioclavicular, and sternoclavicular) and two functional (scapulothoracic and subacromial) joints [1]. These articulations link the upper extremity to the thorax and allow for great mobility of the arm. As a result, the hand can be placed and moved through a large volume of space [2].

The combined mechanics of the articular joints and the surrounding soft tissue structures (muscles, capsules, and ligaments) interact to provide mobility and stability of the shoulder complex. In a normally functioning shoulder complex, both static and dynamic stabilizers result in a broad range of joint movements and provide adequate stability. However, the unique design of the shoulder complex that provides mobility with reduced stability also makes it highly susceptible to dysfunction and instability [1,2].

Glenohumeral joint (shoulder joint)

The glenohumeral joint is a triaxial joint that connects the head of the humerus with the glenoid fossa of the scapula. This joint has greater mobility than any other joint in the body [1]. Only 25% to 30% of the humeral

head is connected to the glenoid fossa at any given time. This anatomical configuration results in an extensive joint mobility but low stability [2]. However, the interplay between the static (capsule, labrum, ligaments) and the dynamic (muscle) forces provide a precise constraint of the center of rotation through a large arc of motion [3].

The glenoid labrum deepens the fossa to provide additional stability and serves as the attachment site for the joint capsule. The superior part of the joint capsule along with the superior glenohumeral and coracohumeral ligaments tighten to limit inferior joint translation and provide static stability to the shoulder. Further static stability is provided through the adhesive and cohesive forces of the synovial fluid and the negative joint pressure that hold joint surfaces together [1,2].

During arm elevation, the dynamic stability of the glenohumeral joint is provided mainly by the muscular forces of the rotator cuff and the deltoid. The rotator cuff consists of the subscapularis, supraspinatus, infraspinatus and teres minor muscles. This group of muscles inserts onto the facets of the greater and lesser tuberosities and provide a continual ring shape insertion from posterior-inferior to anterior-inferior on the proximal humerus [2]. The contraction of the supraspinatus, along with the deltoid, causes arm elevation. The contraction of the infraspinatus and the teres minor muscles provides an external rotation force while the internal rotation force results from the contraction of the subscapularis muscle [1].

The co-contraction of the rotator cuff produces a concavity-compression effect directed toward the glenoid center to promote glenohumeral joint stability, while asymmetric contraction causes humeral head rotation (steering mechanism) and depression during shoulder abduction motion. However, due to the small size of the rotator cuff and its proximity to the joint center of rotation, they generate lower muscle forces when compared to the larger and more superficial muscles (deltoid, latissimus dorsi, trapezius, and pectoralis major) [2].

The long head of the biceps muscle plays a role in stabilizing the head of the humerus. Along with the rotator cuff, it functions to depress the humeral head during shoulder abduction. In addition, the contraction of the long head of biceps during the late phase of throwing reduces anterior translation and resists external rotation [4]. Further static stabilization is promoted by the tension placed on the static restraints and the glenohumeral ligaments that limit excessive translations of humeral head [1].

Acromioclavicular and sternoclavicular joints

The acromioclavicular and the sternoclavicular joints are triaxial joints that connect the clavicle to the acromion process of the scapula and the sternum, respectively. The stability of the acromioclavicular joint is maintained through static stabilizers composed of a thick capsule, intra-articular disc, and the acromioclavicular and coracoclavicular ligaments. The acromioclavicular ligaments restraint the posterior translation of the acromioclavicular joint, while the coracoclavicular ligaments restraint the vertical displacement of the joint [5].

The small sternoclavicular joint is the only joint that connects the shoulder complex to the axial skeleton. The stability of the sternoclavicular joint is provided by the surrounding ligaments composed of the intra-articular disc-ligament, costoclavicular ligament and interclavicular ligament which act as a checkrein against medial displacement, excessive upward rotation and excessive downward rotation of the clavicle, respectively [1,2].

Scapulothoracic articulation and muscles

The scapulothoracic articulation is a functional joint (not a true joint) that represents a space between the thoracic cage and the anterior scapula. There is considerable soft tissue flexibility that allows a relatively smooth slide of the scapula along the underlying thorax. The scapulothoracic articulation synchronizes with the glenohumeral joint and allows for 150° to 180° of shoulder range of motion (ROM) into flexion or abduction with elevation. For every 2° of glenohumeral elevation, there is 1° of scapulothoracic elevation. However, this ratio can vary among individuals and for any part of the arc of movement [1,2].

Several muscles that originate from or insert into the scapula provide motion and dynamically stabilize the scapula. In the dependent position, the scapula is maintained in downward rotation, forward tilting, and protraction position. This position is stabilized by the balanced forces of the trapezius, serratus anterior, levator scapula, and rhomboids musculature. The dependent position of the scapula is further maintained by the static stabilization of the cohesive forces of the subscapular bursa, acromioclavicular and sternoclavicular joint ligaments, and the scapulothoracic facia [1,2].

During active arm motion, the scapulohumeral muscles maintain an effective length-tension relationship and function to stabilize and control the position of the scapula, allowing a smooth movement of the humerus. The serratus anterior maintains the medial angle of the scapula against the chest wall and along with the upper

and medial trapezius, upwardly rotates the scapula during arm elevation [1,2].

During flexion and pushing activities, the serratus anterior muscle protracts the scapula on the thorax. However, during arm extension or pulling activities, the rhomboids retract the scapula and cause downward rotation while the latissimus dorsi, teres major, and rotator cuff muscles function to exert rotational forces that cause the inferior scapula to move away from the midline (upward rotation). In addition, these muscles eccentrically contract to control the upward rotation and protraction of the scapula. The levator scapula elevates the superior angle, resulting in upward and medial rotation of the scapula, while the pectoralis minor protracts and rotates the scapula inferiorly [1,2].

Common Musculoskeletal Disorders of the Shoulder Joint

Musculoskeletal disorders affecting shoulder joint can either lead to hypomobility (restricted mobility) or hypermobility (excess mobility) of the joint. Common pathologies that limit shoulder movements include arthritis [rheumatoid arthritis (RA) or osteoarthritis (OA)], adhesive capsulitis (AC)/frozen shoulder, and rotator cuff tendinopathy (RC)/impingement syndrome [1].

Hypermobility of the shoulder joint causes joint instability and can be atraumatic or traumatic. Atraumatic joint hypermobility can be due to an inherent generalized connective tissue laxity or secondary to repeated microtrauma. However, traumatic instability is usually caused by high direct or indirect applied forces to the shoulder joint that often lead to joint dislocation (complete separation of the articular surfaces) and soft tissue damage. Further, inherent instability may be a predisposing factor to traumatic dislocation, especially with repetitive stressful overhead activities. A secondary effect of joint hypermobility is painful shoulder syndrome [1].

Shoulder arthritis

Arthritis can be defined as joint pain or joint disease. It can affect people of all ages, genders, and races. Overtime, arthritis can lead to impaired mobility and functional limitations. Many types of arthritis may affect shoulder joint including:

Osteoarthritis: It is a chronic degenerative disorder affecting the articular cartilage of shoulder joint leading to pain and stiffness. With degeneration, the capsule also becomes thickened causing further loss of rotational movements. Shoulder OA is not as common as OA of the knee or hip, however, it is reported to affect 32.8% of patients over the age of 60 years [6]. The etiology of the primary shoulder OA is unknown but is related to age (over the age of 65), genetics and sex; women are affected more frequently than men. Secondary OA may occur as a result of repeated micro or high impact trauma, chronic dislocation, or infection [6,7].

Rheumatoid arthritis: It is an autoimmune, chronic, progressive inflammatory, systematic disorder primarily affecting the synovial joint capsule and connective tissue. Shoulder RA results in pain, loss of ROM, stiffness, progressive deformity and functional disability [1,8]. The prevalence of shoulder RA is 1% worldwide and presents in about 5% of people over the age of 70 years. It affects women more frequently than men with a ratio of 3:1. Shoulder symptoms develop in about 91% of patients with long-standing RA (more than 5 years) [8].

Post-traumatic/ immobilization arthritis: It occurs in response to an injury or fracture to the shoulder; or from lack of movement, which causes rapid destruction of articular cartilage. Immobilization arthritis could also occur as a secondary effect of medical conditions such as stroke, diabetes, or heart disease [1].

Rotator cuff tendinopathy/impingement syndrome

Rotator cuff tendinopathy is a progressive disorder of the rotator cuff tendons. The condition begins with acute tendinitis of the muscle tendon (mainly the supraspinatus) and progress to tendinosis with degeneration and partial thickness tears. The condition may result in a full thickness rupture. Rotator cuff tendinopathy causes pain in the shoulder region, leading to a restricted and painful arc of motion, sleep disturbance, and shoulder dysfunction [1,9].

The etiology of RC tendinopathy is often multifactorial, and the symptoms are usually brought on by repetitive or excessive overhead activities. Both intrinsic and extrinsic mechanisms play a role in the pathology development and progression. Extrinsic factors are defined as those causing narrowing of the subacromial space during arm elevation, leading to mechanical compression/impingement and irritation of the soft tissues (rotator cuff and subacromial bursa). Extrinsic factors could be anatomical, such as the shape and angle of the acromion, or biomechanical (postural and muscular impairments) or a combination of both. The extrinsic mechanism was first described by Codman (1934) and the concept was popularized by Neer in the 1980s who coined the term subacromial impingement syndrome [9,10].

On the other hand, intrinsic factors affect the structural integrity of the musculotendinous structures, leading to RC tendon degeneration. These factors include vascular changes in the RC tendons, tissue tension overload, and collagen disorientation and degeneration. The condition

is observed most often in patients over 40 years old and disease prevalence increases with age and can affect more than 50% of the population greater than 60 years old [1,9].

Adhesive capsulitis/ frozen shoulder

Adhesive capsulitis (AC), also known as 'frozen shoulder', is characterized by the development of dense adhesions and capsular thickening leading to a progressive and painful restriction of shoulder ROM and functional disability [11]. The condition does not cause arthritic changes in the joint cartilage and bone as seen with OA and RA. The onset is gradual and usually occurs between the ages of 40 and 65 years [1]. Further, it is five times more common in people with diabetes and is more frequent in women [12].

Codman (1934) was the first to describe the condition, coin the term 'frozen shoulder' and define the common criteria shared by most frozen shoulder patients which include slow onset of pain, inability to sleep on the affected side, painful and restricted shoulder abduction and external rotation motions, and a normal radiological appearance [13].

The frozen shoulder was termed shoulder adhesive capsulitis by Neviaser (1945) who found thickening and contracture of the joint capsule and described peeling the capsule from humeral head as peeling adhesive plaster from skin. In 1969, Lundberg suggested to subdivide frozen shoulder, based on Codman criteria, into two groups: primary or idiopathic frozen shoulder, which has no clear underlying cause, and secondary frozen shoulder, in which the condition is secodary to soft tissue injury, OA, RA, trauma, or secondary to known systemic disease such as diabetes [13].

For many years, AC has been described as a self-limiting condition that progresses through a natural history of painful, frozen and thawing phases, leading to full recovery without treatment. However, a recent systematic review assessed the quality of the evidence that describes the theory of AC phases and the theory of full recovery without treatment [14]. The authors reported a lack of evidence to support the theortical phases of AC. In addition, this review found that moderate-quality evidence supported an early improvement in shoulder ROM and function that slows over time and leads to long-term limitations [14].

The pathophysiology of idiopathic AC was studied in a recent systematic review that included 13 observational studies. There was consistent agreement among studies that the pathological changes in the anterior shoulder joint capsule originated from the subscapularis bursa, at the base of the origin of the long head of the biceps (rotator interval) [15]. These pathological changes were described as a proliferation of fibroblasts arranged alongside layers of dense collagen tissue, leading to capsular contracture. This fibrous tissue was noted to become tight if the arm was placed in external rotation, forming a checkrein to further movement. The systematic review suggested that immune, inflammatory and fibrotic changes where associated with primary frozen shoulder [15].

Association between Diabetes and Shoulder Disorders

Diabetes is a metabolic condition that is characterized by persistent hyperglycemia due to insulin deficiency, impaired effectiveness of insulin action, or both. Diabetes is considered one of the most challenging health problems in the 21st century. It is one of the most disabling diseases and the fifth leading cause of death in most developed countries [16,17].

Based on the etiology, diabetes can be classified into two main types: type 1 diabetes, which results from cellular-mediated autoimmune destruction of pancreatic islet beta cells causing the loss of insulin production; and type 2 diabetes, which occurs due to insulin deficiency and/ or insulin resistance. However, other types of diabetes do exist such as gestational diabetes (occurs during pregnancy), type 3 diabetes (resistance to insulin in the brain), secondary diabetes (as a consequence of other medical condition), neonatal diabetes (affect babies under 6 months old), and many others [16].

Type 1 diabetes occurs more commonly in children, while type 2 diabetes is seen more frequently among adults and older adults and constitutes about 85% to 95% of all diabetes in developed countries. Diabetes can be found in almost every population in the world. The global burden of diabetes is estimated to be 10.4% for persons aged 20-79 years by the year 2040. In Canada, there is large increase in the number of people with diabetes from 9% in 2003 to 11.2% in 2025. In addition, the prevalence of diabetes is more than four times higher among First Nations women than non-First Nations women and more than 2.5 times higher among First Nations men as compared to non-First Nations men [16-18].

Diabetes has many well-described complications including neuropathy, cardiovascular diseases, retinopathy, stroke, peripheral vascular disease (amputation), and renal failure that are resulting in increased disability, reduced life span, and increased health cost [16]. Complications involving the musculoskeletal system are generally less well-described. Shoulder pain is one of the most common complaints of patients with diabetes that causes motion limitation, functional disability and decreased quality of life. There is

higher prevalence of shoulder disorders in patients with diabetes, with AC and RC tendinopathy being the most common disabling shoulder disorders [11,12].

The association between diabetes and AC was first recognized by Bridgman (1972) who found that 10.8% of diabetic patients had AC as compared to 2.3% nondiabetic patients [19]. Subsequent studies have supported this association and reported a prevalence of 10-76% in type 1 and 7-30% in type 2 diabetes as compared to 0-10% in the general population [20-23]. Adhesive capsulitis was also reported to be associated with age in both types of diabetes [20] and with the duration in type 1 diabetes [21,22,24].

Diabetic patients, with or without the use of insulin, have a high risk for developing RC tendinopathy, with a hazard ratio (HR) of 2.11 as compared with those without diabetes [11]. In addition, chronic RC tendinopathy and shoulder pain have been associated with diabetes [25]. Further, patients with diabetes have been reported to have a concurrent diagnosis of AC and RC tendinopathy, leading to shoulder pain and contracture [26]. Furthermore, diabetes has been associated with postoperative stiffness after rotator cuff repair [26,27].

The pathophysiology that predisposes diabetics for the development of AC or RC disease is not well-understood. However, the two diseases might share similar diabetes-related mechanisms [11]. Indeed, several potential mechanisms have been suggested that explain the pathological process including the increased glycosylation of collagen fibers of the joint capsule and the diabetic microangiopathy [11,28,29].

Collagen is a protein fiber that makes up the extracellular matrix of tissues including muscles, tendons, ligaments, capsules, skin, and bones. Normally, collagen fibers are glycosylated meaning that collagen protein molecules have sugar molecules covalently bonded to them through a specific enzymatic process. However, in diabetic tissue, hyperglycemia can cause a non-enzymatic covalent bonding of sugar molecules to the collagen fibers. Over time, the glycating sugar reacts further leading to the formation of advanced glycation end-products (AGEs). These AGEs increase crosslinking in the collagen fibers of shoulder capsule and change the mechanical properties of tissue by making these structures stiffer and weaker [11,28-30].

Further, arthroscopic biopsies of joint synovium in diabetics showed greater amounts of endothelial growth factors and reduced amounts of inflammatory growth factors. These findings explained the prolonged course and severity of AC in patients with diabetes [30]. However, other studies reported that AGEs interact with the receptors on the surface of tenocytes and fibroblasts, leading to chronic inflammatory changes in the joint synovium, which contribute to capsular fibrosis of the shoulder joint [11,29].

The impaired microcirculation (diabetic microangiopathy) is another pathological process that may contribute to the development of AC and RC tendinopathy in patients with diabetes. There is a consensus among studies that uncontrolled hyperglycemia leads to macrovascular and microvascular complications in patients with diabetes [11,29].

It is documented that AGEs are prevalent in the diabetic vasculature and contribute to the development of atherosclerosis. AGE cross-linking of collagen fibers in the basement membrane of the extracellular matrix leads to thickening of the basement membrane, stiffness of blood vessels and alterations in vascular contractility. As a result, tissue hypoxia occurs causing joint tissue destruction and degenerative changes [11,29]. Moreover, AGEs induce vascular endothelial growth factor which causes synovial cell proliferation in the subacromial bursa synovium, leading to the development of shoulder joint contracture in diabetic patients with AC and RC tendinopathy [27].

Assessment of Shoulder Joint

The assessment of the shoulder joint is essential for the diagnosis and formulation of an appropriate management for patients with shoulder disorders. The assessment usually starts with obtaining a detailed history about patient's demographics, medical history, and the onset of the condition symptoms, followed by inspection and palpation. The assessor inspects shoulders for symmetry and deformity and palpates joints for any tenderness, swelling, or anatomic abnormalities. It is also essential to examine neck area to rule out cervical spine pathology and referred neck pain [31,32].

One of the next steps in diagnosing shoulder pathology is to measure shoulder active and passive ROM including forward flexion, abduction, external and internal rotation. The assessment of shoulder ROM can be performed by visual estimation or by using goniometer. Goniometric measurements are extensively used in physical therapy for measuring any joint motion in degrees. Measuring shoulder joint active and passive ROM can provide useful information in differentiating some shoulder disorders such as AC and RC tendinopathy. For example, AC is reported to be associated with limitation in both active and passive ROM, while RC tendinopathy is usually associated with limitations in only active ROM [31].

There are a number of special and strength tests with high clinical values that are often used to diagnose shoulder pathology. However, one test is not interpreted in isolation

but is clustered with additional clinical findings when establishing a clear diagnosis for shoulder problems. Examples of these tests include Hawkins' test and Neer's sign to help in diagnosing shoulder impingement syndromes; Drop-arm test, Lift-off test and Empty Can test are strength tests that indicate a defect in the RC; Apprehension and Relocation tests to diagnose anterior shoulder instability; and Yergason's test and Speed's maneuver to assist in the diagnosis of biceps tendon instability or tendonitis [31,32].

Several self- and examiner-reported outcome measures tools have been validated to assist in the examination of shoulder pain and function. These measures include the Visual Analogue Scale (VAS) for pain assessment, and measures of shoulder function and disability such as the Shoulder Pain and Disability Index (SPADI), Constant Shoulder Score (CSS), American Shoulder and Elbow Surgeons (ASES), Disabilities of the Arm, Shoulder, and Hand (DASH; Quick DASH), and Simple Shoulder Test (SST) questionnaires. All of these questionnaires have been shown to be valid and reliable for the assessment of shoulder function in various clinical situations [33].

Lastly, there are several imaging tests to confirm the diagnosis of shoulder joint pathology including plain x-ray to diagnose bone abnormalities such as osteoarthritis, ultrasound which may be used to diagnose rotator cuff tears, and magnetic resonance imaging (MRI) and computerized tomography scan (CT), which are used to diagnose soft tissue abnormalities [32].

Management of Shoulder Disorders

As mentioned earlier, obtaining a complete patient history and performing a thorough physical examination are essential in determining proper means of treatment for different shoulder disorders. Several studies have examined the effectiveness of surgical and non-surgical treatment interventions for managing shoulder disorders such as AC, RC tendinopathy, arthritis, and shoulder instability. The non-surgical interventions may include physiotherapeutic interventions, pain-control medications, and steroid injection. Examples of surgical approaches include shoulder arthroscopy and shoulder arthroplasty.

Non-surgical interventions

The most common shoulder disorders that might be treated by non-surgical interventions are AC, RC tendinopathy, mild to moderate shoulder OA, and shoulder instability. It is generally recommended to start with a non-surgical treatment for managing these disorders when pain and functional limitation are modest. Surgical interventions may be considered for severely limited conditions [33,34].

Physical therapy interventions have been shown to benefit patients with different shoulder conditions. For example, active ROM exercises, self-stretching and joint mobilization techniques have been reported by several systematic reviews to reduce pain and restore shoulder ROM and function in patients with AC [35-38]. Further, gentle ROM and isometric strengthening of the rotator cuff and scapulothoracic muscles are effective in reducing pain and improving shoulder ROM in patients with mild to moderate shoulder OA with no evidence of atrophy or contracture [6].

Physical therapy programs that emphasize progressive strengthening of the rotator cuff, deltoid, and scapulothoracic muscles combined with functional exercises that require coordination among multiple muscle groups have been commonly recommended for treating patients with shoulder instability. These exercises are reported to control glenohumeral joint translation, improve shoulder joint stability, and reduce anterior glenohumeral ligamentous strain especially during arm elevation [39]. In contrast, shoulder immobilization followed by ROM and stability exercises for the treatment of post-traumatic shoulder instability is only supported by weak evidence (on a Critical Appraisal Form) [34].

The effectiveness of exercise therapy for the treatment of RC tendinopathy/impingement has been investigated in several systematic reviews [40-43]. Data from these reviews strongly suggest that therapeutic exercises combined with manual therapy produce statistically and clinically significant reductions in pain and improvement in shoulder ROM. In addition, exercise therapy may be effective at improving shoulder function. This effect may be augmented with acromioplasty and joint mobilization techniques [40-43]. However, a moderate-quality evidence (on GRADE scale) indicates that subacromial decompression provides no improvement in shoulder pain and function for the treatment of RC tendinopathy [44].

A typical physical therapy program may include pendulum exercise, active assisted and active ROM exercises, postural exercises, scapular stabilization exercises, stretching exercises, joint mobilization techniques, and soft tissue mobilization techniques. However, none of these systematic reviews described the specific components of the exercise protocols (type, intensity, duration and frequency) that are associated with best outcomes [40-43].

Intra-articular steroid injections are commonly used for patients with shoulder pain. There is moderate evidence

that supports small short-term pain reduction in patients with AC following steroid injection [35-37,45]. Further, steroid injections provide superior improvements in shoulder pain for patients with RC impingement when compared to no injection controls, and ultrasound guided injections are superior to non-guided injections [43]. Furthermore, corticosteroid injections seem to relieve shoulder pain in patients with shoulder OA [6,46].

Surgical interventions

In patients with persistent shoulder pain and dysfunction despite conservative treatments, surgery may be indicated to relieve pain and restore joint function. A number of different surgical approaches to manage shoulder disorders have been reported in the orthopedic literature including shoulder arthroscopy and shoulder arthroplasty.

Shoulder arthroscopy is performed by inserting an endoscope into the joint through a small incision. This minimally invasive surgical procedure allows for an examination and treatment of various joint pathologies. For patients with AC, a diagnostic arthroscopy is performed to confirm the diagnosis followed by release of fibrotic structures such as the rotator interval, the middle glenohumeral ligament and the axillary pouch. Two systemic reviews have reported that this procedure improves shoulder ROM and function and is an effective treatment for AC [45,47]. However, confidence in these findings is a concern due to poor methodological quality [45,47].

Further, arthroscopic treatment of partial-thickness RC has been shown to significantly improve shoulder symptoms and function treated using different arthroscopic approaches such as debridement of the tear with or without acromioplasty, trans-tendon repair, or conversion of the lesion to full thickness tear followed by repair [48].

Shoulder arthroplasty is a common surgical procedure in which all or part of the shoulder joint is replaced by a prosthetic implant to alleviate shoulder pain and restore joint function [49]. Shoulder arthroplasty was initially introduced by Gluck and Péan in the 1800s for debriding tuberculous arthritis of the shoulder which failed miserably, and the procedure was eventually revisited by Neer in the 1950s for the treatment of proximal humeral fractures. However, advancements in prosthesis design have resulted in expanded indications and a concomitant increase in the rates for shoulder arthroplasty [50,51].

Currently, end-stage primary glenohumeral osteoarthritis is the primary diagnosis for 77% of shoulder arthroplasty and often occurs more frequently among adults aged 65 years or older [52]. This surgical procedure is usually indicated when conservative treatments such as therapeutic exercises and manual therapy techniques fail [52].

There are three main types of shoulder arthroplasty: total shoulder arthroplasty (TSA), hemiarthroplasty (HA), and reverse total shoulder arthroplasty (rTSA). TSA involves replacing both the humeral head and the glenoid fossa, while HA involves replacing only the humeral head with metal implants. Patients with OA and an intact or reparable rotator cuff typically undergo TSA, while patients with OA and an irreparable rotator cuff tear traditionally undergo a HA [53]. However, TSA has been reported to result in greater improvement in shoulder ROM and pain, and in less need for surgery revision when compared to HA [51].

The main indication for rTSA is shoulder OA with irreparable rotator cuff tear when conventional surgery fails. However, the advancement of the prosthetic design has led to expansion of the indications to include any condition about the shoulder where rotator cuff function is deficient including RA and proximal humeral fractures [54].

Despite the improvements of shoulder function and pain after shoulder arthroplasty, shoulder arthroplasty is not without complications. Shoulder subluxation or dislocation, humeral fracture, and joint infection are the most commonly reported postoperative complications [49,55]. Additional complications that are specific to rTSA may include glenoid loosening, musculocutaneous nerve palsy, and acromial fractures [54].

Impact of Diabetes on Shoulder Recovery

All the above-mentioned interventions have been evaluated in generic populations and have been shown to be effective in reducing shoulder pain and improving ROM and function. However, patients with shoulder disorders and concurrent comorbidities such as diabetes have been reported to respond less favorably to these interventions. Indeed, a recent systematic review evaluated the effectiveness of non-surgical interventions for managing AC in patients with diabetes [56]. The authors reported that low quality evidence suggests large effects of joint mobilization plus exercises on AC in people with diabetes and even weaker support was available for corticosteroid and manipulation under anesthesia (MUA) [56].

Vastamaki et al. followed-up patients with and without diabetes who have AC for the duration of 10 years. Although shoulder ROM improved over time in patients with diabetes, this improvement was inferior to patients without diabetes and remained below normal ROM [57]. Further, Juel et al. and Larkin et al. have also shown that patients with type 1 diabetes develop long-lasting shoulder stiffness, functional disability and reduced ROM than patients without diabetes. These studies suggested

that early shoulder assessment and treatment may be needed to reduce disability and improve quality of life of patients with diabetes [21,22].

Furthermore, Cole et al. and Rill et al. have reported higher shoulder pain, reduced mobility, poor functional outcomes, reduced quality of life, and a diminished response to treatment in diabetic patients with AC than patients without diabetes [58].

Studies that evaluated the impact of diabetes on shoulder recovery following arthroscopic rotator cuff repair have reported an inferior improvement in shoulder pain and function in patients with diabetes, in addition to an increased risk of anatomic failure of the repaired rotator cuff tendon especially in patients with uncontrolled hyperglycemia [60,61].

Diabetes has been shown to be an independent risk factor for increased risk of non-home discharge (Odds ratio (OR): 1.3), and longer hospital stays (OR: 1.4) following shoulder arthroplasty [62,63]. Further, diabetes, along with hypertension and obesity, are associated with postoperative complications such as humeral fracture and joint infection [64]. However, the impact of diabetes on functional outcomes and motion after shoulder arthroplasty has not yet been investigated.

Summary of Limitations in Current Knowledge

Studies consistently report that diabetics are more frequently affected by AC, have long lasting symptoms and a poorer prognosis than non-diabetics [11,20-23,65]. Further, although some of the therapeutic interventions have been shown to be effective in managing primary AC, several studies have reported higher shoulder pain, reduced mobility, poor functional outcomes, reduced quality of life, and a diminished response to treatment in patients with diabetes than patients without diabetes [21,22, 58,59]. There is lack of systematic reviews to assess the effectiveness of therapeutic interventions in managing AC in patients with diabetes and to show whether the current recommendations for treatment of AC can be equally applied to patients with diabetes.

There have been efforts to define an optimal physical therapy protocol for managing AC in patients with diabetes. The usual approach to AC includes mobilization of soft tissues and implementation of shoulder exercises to restore function. However, recovery is slow and often incomplete, especially for people with diabetes. Aerobic exercises can improve hyperglycemia and insulin sensitivity in skeletal musculature [66], which may have a greater impact on the AC pathophysiology. To the author's knowledge, an optimal physical therapy protocol for managing AC in patients with diabetes is not defined.

Given the fact that hyperglycemia has a negative impact on body tissue [28], and the adverse effect of diabetes on postoperative complications and hospital stays [62,64], there is a need to investigate whether diabetes affects functional outcomes after shoulder arthroplasty. To the author's knowledge, no study has evaluated the impact of diabetes on shoulder pain and function following shoulder arthroplasty.

There are limited studies which addressed the factors that influence postoperative functional outcomes following shoulder arthroplasty. Identifying preoperative factors that are predictive of one-year outcomes could assist surgeons and health care providers in providing patients with more realistic expectations on outcomes and may help plan postoperative pain management and rehabilitation. To the author's knowledge, factors that predict the clinical benefits following shoulder arthroplasty have not been thoroughly investigated.

Future Directions

There is a need to conduct more systematic reviews to determine the effectiveness of surgical (arthroscopy and arthroplasty) and non-surgical interventions (steroid injections, physiotherapeutic interventions, and MUA) on shoulder pain, function, and ROM for managing AC in people with diabetes. Further, clinical researchers need to run more robust randomized trials to examine the impact of these therapeutic interventions on shoulder function in patients with diabetes as compared to patients without diabetes. Furthermore, more studies are required to investigate the impact of diabetes on pain, patient-reported function, and impairments in shoulder ROM and muscle strength in patients who underwent shoulder arthroplasty. Lastly, further research is recommended to investigate whether factors such as comorbidities and demographics predict patient-reported outcomes including shoulder pain and function and clinical benefits following shoulder arthroplasty.

In summary, shoulder disorders, such as frozen shoulder and rotator cuff disease, are commonly seen in patients with diabetes. Future research is needed to examine the effectiveness of different surgical and non-surgical interventions on managing shoulder disorders in patients with diabetes. In addition, more research is required to investigate the impact of diabetes on shoulder recovery, and factors predicting shoulder function following shoulder arthroplasty.

References

1. Kisner, C., Colby, L. A., & Borstad, J. (2018). The shoulder and Shoulder Girdle. In & J. B. C. Kisner, L. A. Colby (Ed.), Therapeutic Exercsie: Foundations and Techniques (pp. 546–622). Philadelphia, PA: Fa Davis.

2. Terry, G. C., & Chopp, T. M. (2000). Functional anatomy of the shoulder. Journal of Athletic Training, 35(3), 248.

3. Lippitt, S. B., Vanderhooft, J. E., Harris, S. L., Sidles, J. A., Harryman II, D. T., & Matsen III, F. A. (1993). Glenohumeral stability from concavity-compression: a quantitative analysis. Journal of Shoulder and Elbow Surgery, 2(1), 27–35.

4. Rodosky, M. W., Harner, C. D., & Fu, F. H. (1994). The role of the long head of the biceps muscle and superior glenoid labrum in anterior stability of the shoulder. The American Journal of Sports Medicine, 22(1), 121–130.

5. Beim, G. M. (2000). Acromioclavicular joint injuries. Journal of Athletic Training, 35(3), 261.

6. Chillemi, C., & Franceschini, V. (2013). Shoulder osteoarthritis. Arthritis, 2013, 7.

7. Chen, A. L., Joseph, T. N., & Zuckerman, J. D. (2003). Rheumatoid arthritis of the shoulder. JAAOS-Journal of the American Academy of Orthopaedic Surgeons, 11(1), 12–24.

8. Millett, P. J., Gobezie, R., & Boykin, R. E. (2008). Shoulder osteoarthritis: diagnosis and management. Am Fam Physician, 78(5), 605–611.

9. Seitz, A. L., McClure, P. W., Finucane, S., Boardman III, N. D., & Michener, L. A. (2011). Mechanisms of rotator cuff tendinopathy: intrinsic, extrinsic, or both? Clinical Biomechanics, 26(1), 1–12.

10. Neer, C. S. (1983). Impingement lesions. Clinical Orthopaedics and Related Research (1976-2007), 173, 70–77.

11. Hsu, C. L., & Sheu, W. H. H. (2016). Diabetes and shoulder disorders. Journal of Diabetes Investigation, 7(5), 649–651. https://doi.org/10.1111/jdi.12491

12. Huang, Y., Fann, C., Chiu, Y., Yen, M., Chen, L., Chen, H., & Pan, S. (2013). Association of diabetes mellitus with the risk of developing adhesive capsulitis of the shoulder: a longitudinal population-based followup study. Arthritis Care & Research, 65(7), 1197–1202.

13. Bunker, T. D. (1997). Frozen shoulder: unravelling the enigma. Annals of the Royal College of Surgeons of England, 79(3), 210.

14. Wong, C. K., Levine, W. N., Deo, K., Kesting, R. S., Mercer, E. A., Schram, G. A., & Strang, B. L. (2017). Natural history of frozen shoulder: fact or fiction? A systematic review. Physiotherapy, 103(1), 40–47.

15. Ryan, V., Brown, H., Lowe, C. J. M., & Lewis, J. S. (2016). The pathophysiology associated with primary (idiopathic) frozen shoulder: A systematic review. BMC Musculoskeletal Disorders, 17(1), 340.

16. International diabetes federation. (2003). IDF Diabetes Atlas, 2th ed. Retrieved from https://www.idf.org/e-library/epidemiology-research/diabetes-atlas/23-atlas-2nd-edition-year.html

17. Ogurtsova, K., da Rocha Fernandes, J. D., Huang, Y., Linnenkamp, U., Guariguata, L., Cho, N. H., ... Makaroff, L. E. (2017). IDF Diabetes Atlas: Global estimates for the prevalence of diabetes for 2015 and 2040. Diabetes Research and Clinical Practice, 128, 40–50.

18. Dyck, R., Osgood, N., Lin, T. H., Gao, A., & Stang, M. R. (2010). Epidemiology of diabetes mellitus among First Nations and non-First Nations adults. Cmaj, 182(3), 249–256.

19. Bridgman, J. F. (1972). Periarthritis of the shoulder and diabetes mellitus. Annals of the Rheumatic Diseases, 31(1), 69.

20. Arkkila, P. E. T., & Gautier, J.-F. (2003). Musculoskeletal disorders in diabetes mellitus: an update. Best Practice & Research Clinical Rheumatology, 17(6), 945–970.

21. Juel, N. G., Brox, J. I., Brunborg, C., Holte, K. B., & Berg, T. J. (2017). Very high prevalence of frozen shoulder in patients with type 1 diabetes of≥ 45 years' duration: the Dialong Shoulder Study. Archives of Physical Medicine and Rehabilitation, 98(8), 1551–1559.

22. Larkin, M. E., Barnie, A., Braffett, B. H., Cleary, P. A., Diminick, L., Harth, J., ... Lorenzi, G. (2014). Musculoskeletal complications in type 1 diabetes. Diabetes Care, 37(7), 1863–1869.

23. Zreik, N. H., Malik, R. A., & Charalambous, C. P. (2016). Adhesive capsulitis of the shoulder and diabetes: a meta-analysis of prevalence. Muscles, Ligaments and Tendons Journal, 6(1), 26.

24. Cagliero, E., Apruzzese, W., Perlmutter, G. S., & Nathan, D. M. (2002). Musculoskeletal disorders of the hand and shoulder in patients with diabetes mellitus. The American Journal of Medicine, 112(6), 487–490.

25. Rechardt, M., Shiri, R., Karppinen, J., Jula, A., Heliövaara, M., & Viikari-Juntura, E. (2010). Lifestyle and metabolic factors in relation to shoulder pain and rotator cuff tendinitis: a population-based study. BMC Musculoskeletal Disorders, 11(1), 165.

26. Tauro, J. C. (2006). Stiffness and rotator cuff tears: incidence, arthroscopic findings, and treatment results. Arthroscopy: The Journal of Arthroscopic & Related Surgery, 22(6), 581–586.

27. Handa, A., Gotoh, M., Hamada, K., Yanagisawa, K., Yamazaki, H., Nakamura, M., ... Fukuda, H. (2003). Vascular endothelial growth factor 121 and 165 in the subacromial bursa are involved in shoulder joint contracture in type II diabetics with rotator cuff disease. Journal of Orthopaedic Research, 21(6), 1138–1144.

28. Abate, M., Schiavone, C., Pelotti, P., & Salini, V. (2010). Limited joint mobility in diabetes and ageing: recent advances in pathogenesis and therapy. International Journal of Immunopathology and Pharmacology, 23(4), 997–1003. https://doi.org/4 [pii]

29. Goldin, A., Beckman, J. A., Schmidt, A. M., & Creager, M. A. (2006). Advanced glycation end products: Sparking the development of diabetic vascular injury. Circulation, 114(6), 597–605. https://doi.org/10.1161/CIRCULATIONAHA.106.621854

30. Whelton, C., & Peach, C. A. (2018). Review of diabetic frozen shoulder. European Journal of Orthopaedic Surgery & Traumatology, 28(3), 363–371.

31. Andrews, J. R. (2005). Diagnosis and treatment of chronic painful shoulder: review of nonsurgical interventions. Arthroscopy: The Journal of Arthroscopic & Related Surgery, 21(3), 333–347.

32. Stevenson, J. H., & Trojian, T. (2002). Evaluation of shoulder pain. Journal of Family Practice, 51(7), 605–611.

33. Angst, F., Schwyzer, H., Aeschlimann, A., Simmen, B. R., & Goldhahn, J. (2011). Measures of adult shoulder function: Disabilities of the arm, shoulder, and hand questionnaire (DASH) and its short version (QuickDASH), shoulder pain and disability index (SPADI), American shoulder and elbow surgeons (ASES) society standardized shoulder. Arthritis Care & Research, 63(S11), S174–S188.

34. Gibson, K., Growse, A., Korda, L., Wray, E., & MacDermid, J. C. (2004). The effectiveness of rehabilitation for nonoperative management of shoulder instability: a systematic review. Journal of Hand Therapy, 17(2), 229–242.

35. Blanchard, V., Barr, S., & Cerisola, F. L. (2010). The effectiveness of corticosteroid injections compared with physiotherapeutic interventions for adhesive capsulitis: a systematic review. Physiotherapy, 96(2), 95–107.

36. Favejee, M. M., Huisstede, B. M. A., & Koes, B. W. (2011). Frozen shoulder: the effectiveness of conservative and surgical interventions—systematic review. British Journal of Sports Medicine, 45(1), 49–56.

37. Jain, T. K., & Sharma, N. K. (2014). The effectiveness of physiotherapeutic interventions in treatment of frozen shoulder / adhesive capsulitis : A systematic review. Journal of Back and Musculoskeletal Rehabilitation, 27, 247–273. https://doi.org/10.3233/BMR-130443

38. Maund, E., Craig, D., Suekarran, S., Neilson, A. R., Wright, K., Brealey, S., ... Rangan, A. (2012). Management of frozen shoulder: a systematic review and cost-effectiveness analysis. Health Technology Assessment (Winchester, England), 16(11), 1.

39. Hayes, K., Callanan, M., Walton, J., Paxinos, A., & Murrell, G. A. C. (2002). Shoulder instability: management and rehabilitation. Journal of Orthopaedic & Sports Physical Therapy, 32(10), 497–509.

40. Desmeules, F., Boudreault, J., Dionne, C. E., Frémont, P., Lowry, V., MacDermid, J. C., & Roy, J.-S. (2016). Efficacy of exercise therapy in workers with rotator cuff tendinopathy: a systematic review. Journal of Occupational Health, 15–103.

41. Hanratty, C. E., McVeigh, J. G., Kerr, D. P., Basford, J. R., Finch, M. B., Pendleton, A., & Sim, J. (2012). The effectiveness of physiotherapy exercises in subacromial impingement syndrome: a systematic review and meta-analysis. In Seminars in arthritis and rheumatism (Vol. 42, pp. 297–316). Elsevier.

42. Kuhn, J. E. (2009). Exercise in the treatment of rotator cuff impingement: A systematic review and a synthesized evidence-based rehabilitation protocol. Journal of Shoulder and Elbow Surgery, 18(1), 138–160. https://doi.org/10.1016/j.jse.2008.06.004

43. Steuri, R., Sattelmayer, M., Elsig, S., Kolly, C., Tal, A., Taeymans, J., & Hilfiker, R. (2017). Effectiveness of

conservative interventions including exercise, manual therapy and medical management in adults with shoulder impingement: a systematic review and meta-analysis of RCTs. British Journal of Sports Medicine, 51(18), 1340–1347.

44. Karjalainen, T. V, Jain, N. B., Page, C. M., Lähdeoja, T. A., Johnston, R. V, Salamh, P., … Vandvik, P. O. (2019). Subacromial decompression surgery for rotator cuff disease. Cochrane Database of Systematic Reviews, (1).

45. Uppal, H. S., Evans, J. P., & Smith, C. (2015). Frozen shoulder: A systematic review of therapeutic options. World Journal of Orthopedics, 6(2), 263.

46. Colen, S., Geervliet, P., Haverkamp, D., & Van Den Bekerom, M. P. J. (2014). Intra-articular infiltration therapy for patients with glenohumeral osteoarthritis: A systematic review of the literature. International Journal of Shoulder Surgery, 8(4), 114–121. https://doi.org/10.4103/0973-6042.145252

47. Grant, J. A., Schroeder, N., Miller, B. S., & Carpenter, J. E. (2013). Comparison of manipulation and arthroscopic capsular release for adhesive capsulitis: a systematic review. Journal of Shoulder and Elbow Surgery, 22(8), 1135–1145.

48. Strauss, E. J., Salata, M. J., Kercher, J., Barker, J. U., McGill, K., Bach Jr., B. R., … Verma, N. N. (2011). The Arthroscopic Management of Partial-Thickness Rotator Cuff Tears: A Systematic Review of the Literature. Arthroscopy, 27(4), 568–580. https://doi.org/10.1016/j.arthro.2010.09.019

49. Deshmukh, A. V, Koris, M., Zurakowski, D., & Thornhill, T. S. (2005). Total shoulder arthroplasty: long-term survivorship, functional outcome, and quality of life. Journal of Shoulder and Elbow Surgery, 14(5), 471–479.

50. Bankes, M. J. K., & Emery, R. J. H. (1995). Pioneers of shoulder replacement: themistocles Gluck and Jules Emile Péan. Journal of Shoulder and Elbow Surgery, 4(4), 259–262.

51. van den Bekerom, M. P. J., Geervliet, P. C., Somford, M. P., van den Borne, M. P. J., & Boer, R. (2013). Total shoulder arthroplasty versus hemiarthroplasty for glenohumeral arthritis: A systematic review of the literature at long-term follow-up. International Journal of Shoulder Surgery, 7(3), 110–115. https://doi.org/10.4103/0973-6042.118915

52. Kim, S. H., Wise, B. L., Zhang, Y., & Szabo, R. M. (2011). Increasing incidence of shoulder arthroplasty in the United States. JBJS, 93(24), 2249–2254.

53. Singh, J. A., Sperling, J., Buchbinder, R., & McMaken, K. (2011). Surgery for Shoulder Osteoarthritis: A Cochrane Systematic Review. The Journal of Rheumatology, 38(4), 598–605. https://doi.org/10.3899/jrheum.101008

54. Smith, C. D., Guyver, P., & Bunker, T. D. (2012). Indications for reverse shoulder replacement. The Journal of Bone and Joint Surgery. British Volume, 94–B(5), 577–583. https://doi.org/10.1302/0301-620X.94B5.27596

55. Norris, T. R., & Iannotti, J. P. (2002). Functional outcome after shoulder arthroplasty for primary osteoarthritis: a multicenter study. Journal of Shoulder and Elbow Surgery, 11(2), 130–135.

56. Alsubheen, S. A., Nazari, G., Bobos, P., MacDermid, J. C., Overend, T. J., & Faber, K. (2019). Effectiveness of Nonsurgical Interventions for Managing Adhesive Capsulitis in Patients With Diabetes: A Systematic Review. Archives of Physical Medicine and Rehabilitation, 100(2), 350–365. https://doi.org/10.1016/j.apmr.2018.08.181

57. Vastamäki, H., Ristolainen, L., & Vastamäki, M. (2016). Range of motion of diabetic frozen shoulder recovers to the contralateral level. Journal of International Medical Research, 44(6), 1191–1199.

58. Cole, A., Gill, T. K., Shanahan, E. M., Phillips, P., Taylor, A. W., & Hill, C. L. (2009). Is diabetes associated with shoulder pain or stiffness? Results from a population based study. The Journal of Rheumatology, 36(2), 371–377.

59. Rill, B. K., Fleckenstein, C. M., Levy, M. S., Nagesh, V., & Hasan, S. S. (2011). Predictors of outcome after nonoperative and operative treatment of adhesive capsulitis. The American Journal of Sports Medicine, 39(3), 567–574.

60. Cho, N. S., Moon, S. C., Jeon, J. W., & Rhee, Y. G. (2015). The Influence of Diabetes Mellitus on Clinical and Structural Outcomes After Arthroscopic Rotator Cuff Repair. The American Journal of Sports Medicine, 43(4), 991–997. https://doi.org/10.1177/0363546514565097

61. Clement, N. D., Hallett, A., MacDonald, D., Howie, C., & McBirnie, J. (2010). Does diabetes affect outcome after arthroscopic repair of the rotator cuff? The Journal of Bone and Joint Surgery. British Volume, 92–B(8), 1112–1117. https://doi.org/10.1302/0301-620X.92B8.23571

62. Mahure, S. A., Quien, M. M., Karia, R., Zuckerman, J. D., & Kwon, Y. W. (2017). Impact of diabetes on perioperative complications in patients undergoing elective total shoulder arthroplasty. Bulletin of the NYU Hospital for Joint Diseases, 75(3), 173.

63. Ponce, B. A., Menendez, M. E., Oladeji, L. O., & Soldado, F. (2014). Diabetes as a risk factor for poorer early postoperative outcomes after shoulder arthroplasty. Journal of Shoulder and Elbow Surgery, 23(5), 671–678.

64. Jain, Guller, U., Pietrobon, R., Bond, T. K., & Higgins, L. D. (2005). Comorbidities increase complication rates in patients having arthroplasty. Clinical Orthopaedics and Related Research®, 435, 232–238.

65. Hand, C., Clipsham, K., Rees, J. L., & Carr, A. J. (2008). Long-term outcome of frozen shoulder. Journal of Shoulder and Elbow Surgery, 17(2), 231–236.

66. Marwick, T. H., Hordern, M. D., Miller, T., Chyun, D. A., Bertoni, A. G., Blumenthal, R. S., … Rocchini, A. (2009). Exercise Training for Type 2 Diabetes Mellitus. Circulation, 119(25), 3244–3262. https://doi.org/10.1161/CIRCULATIONAHA.109.192521