# PREVALENCE OF ROTATOR CUFF DISEASES IN TYPE II DIABETES MELLITUS- A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY


BY

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREVALENCE OF ROTATOR CUFF DISEASES IN TYPE II DIABETES MELLITUS – A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY",** is ethical and justifiable.

The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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# UNDERTAKING

"I, **REG. NO: BL0120004**, hereby declare that the information and the data mentioned in my dissertation entitled "PREVALENCE OF ROTATOR CUFF DISEASES IN TYPE II DIABETES MELLITUS- A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY" belongs to me and is original.

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# ABSTRACT

Background:

There is an increased risk of shoulder disorders in patients with Diabetes Mellitus Type II, as compared to general population. In patients with Type II Diabetes mellitus, the Non Enzymatic Glycosylation of Collagen with Advanced Glycation End products (A.G.E.s) which affect the physical and chemical properties of collagen proteins, result in reduction of solubility of collagen, which gets tougher, stiffer and weaker, thus loosing its elasticity and become more prone to Rotator Cuff tears. So, there is an increased need to study and find out prevalence of rotator cuff diseases in Type II Diabetes Mellitus, so as to restrict any further damage by achieving control of blood sugar, and if need be, achieve early surgical repair of the injury.

Methods:

Patients with shoulder pain or restricted shoulder movements who are a known case of Diabetes Mellitus type II presenting to the Out Patient Department of Orthopaedics, K.L.E.S. Dr. Prabhakar Kore Hospital and Medical Research Centre and Charitable Hospital, Belagavi in between 1st January 2021 to 31st December 2021, over a period of 1 year, were examined clinically and requested to undergo Ultrasonography of the affected shoulder, to correlate the pathology.

Results:

The mean age group of the study participants was found to be 54.12 $\pm$ 10.465 years. 65.3% of the study participants were males with females contributing to 34.7% of the study participants. 72% of the study participants were affected on the right shoulder and 28% of the study participants were affected on the left shoulder**.** The range of restriction of flexion of the study participants is seen in the following order: 0°> 20°, and 90° > 10°, and 30° > 80° > 70° > 40° > 60° > 50°. The range of restriction of extension of the study participants is seen in the following order: 0°> 10° > 20°. The range of restriction of abduction of the study participants is seen in the following order: 10°> 0° > 30° > 20° , 80° > 60°, 70° and 90° > 50° > 40°. The range of restriction of adduction of the study participants is seen in the following order: 0°> 10°. The range of restriction of internal rotation of the study participants is seen in the following order: 0°> 30° > 20° > 10° > 40°. The range of restriction of external rotation of the study participants is seen in the following order: 0°> 10° > 20°. 73.3% of the study participants were found to be negative on Jobe test and only 26.7% of the study participants were found to be

positive on Jobe test. 85.3% of the study participants were found to be negative on Drop arm test and only 14.7% of the study participants were found to be positive on Drop arm test. 38.7% of the study participants were found to have external rotation lag and 48.0% of the study participants were found to have Internal rotation lag. 61.3% of the study participants were found to be negative on Gerber's lift off test and only 6.7% of the study participants were found to be positive on Gerber's lift off test. Gerber's lift off test could not be elicited in 32% of the study participants. 97.3% of the study participants were found to be negative on Biceps tendon palpation test and only 2.7% of the study participants were found to be positive on Biceps tendon palpation test. 24% of the study participants were found to be normal on USG shoulder. 33.3% of the study participants were diagnosed with Adhesive Capsulitis followed by 12% with Partial Supraspinatus Tear on USG shoulder. 9.3%, 6.7% and 4% of the study participants were diagnosed with AC joint arthritis, Supraspinatus tendinitis and Full thickness Rotator cuff tear on USG shoulder respectively. 2.7% each of the study participants were diagnosed with Bicipital tendinitis, Subscapularis tendinosis and Supraspinatus tendinosis respectively. 1.3% each of the study participants were diagnosed with Bicipital tendinosis and Complete Supraspinatus tear respectively.

29.33% of the study participants with diabetes was found to have rotator cuff disease upon clinical examination and Ultrasonography of the affected shoulder joint, whereas 70.66% of the study participants were not found to have rotator cuff disease

Conclusion:

The results of our study depict worsening of degeneration of the Rotator Cuff Tendons in patients who suffer from Diabetes Mellitus. In addition to clinical screening, USG imaging is a helpful tool for identifying at-risk individuals who may develop shoulder disorders in the symptomatic stage. Careful metabolic control using appropriate food habits and anti-diabetic medications should be advised in these people, who represent an increasing section of the older population. The development of tear size should also be tracked over time, and appropriately managed by either surgery or medicines along with physiotherapy.

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# LIST OF ABBREVIATIONS

**ROM-** RANGE OF MOVEMENTS

**NSAID-** NON STEROIDAL ANTI-INFLAMMATORY DRUGS

**USG-** ULTRASONOGRAPHY

**MRI-** MAGNETIC RESONANCE IMAGING

**DM-** DIABETES MELLITUS

**NIDDM-** NON INSULIN DEPENDENT DIABETES MELLITUS

**IDDM-** INSULIN DEPENDENT DIABETES MELLITUS

**LIC-** LOCAL INJECTION OF CORTICOSTEROIDS

**AGE-** ADVANCED GLYCATION END PRODUCTS

**ALE-** ADVANCED LIPATION END PRODUCTS

**RAGE-** RECEPTOR FOR ADVANCED GLYCATION END PRODUCTS

**HbA1C-** GLYCOSYLATED HAEMOGLOBIN

**VEGF-** VASCULAR ENDOTHELIAL GROWTH FACTOR

**OA-** OSTEOARTHRITIS

**ACL-** ANTERIOR CRUCIATE LIGAMENT

**NFKB-** NUCLEAR FACTOR KAPPA BETA

**CML-** CARBOXY METHYL CELLULOSE

**RNAase-** RIBONUCLEASE

**ELISA-** ENZYME LINKED IMMUNO SORBENT ASSAY

**HMGB1-** HIGH MOBILITY GROUP BOX 1 PROTEIN

**p21 ras-** p21 RAT SARCOMA GENE

**TNF A-** TUMOUR NECROSIS FACTOR ALPHA

**ANOVA-** ANALYSIS OF VARIANCE

# LIST OF PHOTOGRAPHS

# INTRODUCTION

Musculoskeletal ailments are one of the most frequently encountered problems in diabetics, still receive less importance. Extent of problems of musculoskeletal complications may not be as notable as cardiac problems, but the linked diseases without a fail cause physical and psychological harm to patients with diabetes. Amongst musculoskeletal ailments, painful shoulders are a frequently encountered complaint, the symptoms of which are pain and restriction of movements. Shoulder pain disables the daily activities, which cause a decreased quality of life and can affect the metabolic control.

A large prevalent rate (27.55 %) of shoulder diseases in diabetics is noted in contrast to 5.0% prevalence rate seen with general individuals, according to previous studies. The two most prevailing shoulder ailments are frozen shoulder and cuff tendinopathies. Adhesive capsulitis is characterised by progressing pain, stiffness, restricted active and passive range of movement of the shoulder, specifically lateral rotation, with night pain. Even though etiology of Adhesive Capsulitis is less known, it is thought to be because of peri-vascular inflammation and fibroblastic growth, which leads to capsular fibrosis and contractures. Adhesive capsulitis can be diagnosed clinically by history and physical examination. Management of frozen shoulder comprises analgesics, such as N.S.A.I.D.s, paracetamol along with Local injection of corticosteroids (LIC). When symptoms of pain and inflammation alleviate, physiotherapy enhancing the movements is started. Surgical options include manipulation under anaesthesia or Arthroscopic capsular release, which is required in rare cases. [1]

## INTRODUCTION

The rotator cuff is made up of supraspinatus, infraspinatus, teres-minor and subscapularis. Originating from scapula, it makes tendons covering proximal humerus, giving shoulder joint motion control and stabilising it. At high risks of rotator-cuff problems are the elderly, abnormal shoulder structure, some athletics and work involving over-head activity. Ranging from mere inflammation to full thickness tendon-tears, mostly of supraspinatus tendon, Rotator cuff diseases comprise a spectrum of ailments affecting the shoulder joint, the symptoms of which are shoulder-pain, decreased muscular power and restricted ROM. Comprehensive history as well as physical examination aid the diagnosis, whereas Radiological imaging which include USG and MRI, confirms diagnosis providing more insight on magnitude and extending rotator-cuff ailment.

Conventional radiographs to negate bone pathology and tendon calcification also aids in coming to a conclusive diagnosis. Non operative treatment of rotator-cuff pathology comprise N.S.A.I.D.s, intra-lesion steroids, physiotherapy. Surgical modalities needed for extensile pathologies, like totally torn tendons include mini open as well as arthroscopic repair of tendons.[1]

Increased prevalence of Rotator-cuff disease as well as Adhesive capsulitis in diabetics is because of the biochemical changes in the rotator cuff tendons and the shoulder joint capsule, as well as certain intrinsic factors like repeated micro trauma and late tendinous-healing. There is abnormal storing collagen on tissues, which can be because of non-enzymatic glycosylation in collagen forming Advanced Glycation End product (A.G.E.s) , which impact physical and chemical nature of protein, enhancing number of inter-molecular collagen crosslinks, which result in reducing solubility in collagen which get tough, stiff and weak. It looses elastic strength thus becomes more prone-to tearing.[10-13]

## AIM AND OBJECTIVES

AIM:

Aim of the study is to evaluate prevalence of Rotator cuff diseases from people with Diabetes mellitus

OBJECTIVES:

There is increased need to study and find out the prevalence of Rotator cuff diseases in patients having Diabetes mellitus so that-

1) Optimum metabolic control of blood sugar with proper dietary modifications and /or medications can be achieved so as to restrict further damage to the affected shoulder joint.

2) And if need be, surgical repair of the rotator cuff injury, for example tendon tears can be achieved as early as possible after the diagnosis is made on clinical as well as radiological grounds.

# REVIEW OF LITERATURE

The most frequent cause of shoulder impairment in persons over 50 is rotator cuff tear, and as people become older, the frequency rises.[19] After 50 years, there was thought to be a statistically significant rise in frequency and loss-of muscle-tendon-unit with ageing. Prevalence of rotator cuff tear range 5 to 40%, and there were 30–70% cases of sore shoulders caused by rotator cuff diseases. Clinical diagnosis of rotator cuff tears is not certain because they can be asymptomatic.[20] As a result, different studies have found varying prevalent-rates and epidemiological characteristics of rotator-cuff injuries. Tempelhof et al. and Schibany et al., showed that complete thickness tears were more common in 411 and 212 participants, respectively, at 23% and 6%.[21]. According to a Japanese paper, 1366 shoulders had ultrasonographic exams, and the results showed that 20.47% of the shoulders had rotator cuff tears, and that prevalent increased as age.[22]

The pathophysiology of rotator cuff tears has historically been divided into intrinsic (hypo- perfusion, degeneration, micro-trauma, apoptotic theory, with extracellular matrix changes) and extrinsic (over-use, chronic-impingement syndrome, among multi-factorial) etiologies.[23] Pain alleviation, increased functionality, and patient satisfaction are all benefits of repairing a torn rotator cuff.[24] The common surgical techniques for rotator cuff repair are arthroscopic, mini open, and open. Surgery is required for people who have a complete rotator cuff tear to restore the tendon to the bone.[25] Therefore, in patients who have a substantial rotator cuff tear and markedly reduced shoulder functionality, hospitalisation with surgical intervention is advised.[16]

One of the most prevalent and disabling medical disorders is still diabetes mellitus (DM). A rotator cuff deterioration was found during an ultrasound evaluation in older DM patients who were asymptomatic.[1] Additionally, a favourable correlation between glycemia and rotator cuff tears was found.[26] Diabetics exhibited 2.1 time more risk-of rotator-cuff problems than individuals without

DM, according to a recent large population based Taiwan study.[27] The prevalence of adhesive capsulitis in DM patients was calculated to be 13.4%. [28]

In addition, latest systemic review and meta analysis indicated that patients diabetics suffered larger tendon tears compared to controls, and more than three times the likelihood of developing tendinopathy. [29] When comparing patients with DM to the general population, Zakaria. et al. looked into prevalence of tendon tear in diabetics in Australia. They found that adjusted odd's ratio 1.84 in over-all tendon rupture needing hospitalisation for diabetics in 2014 (Zakaria. et. al.). Although these studies identified a link between diabetes and rotator-cuff diseases, Zakaria. et al. discovered that DM patients have a high-risk of different type of tendon damage, there aren't any research that specifically examine the link of diabetes with prevalence of rotator cuff injury. [30]

There is no known mechanism through which DM affects onset of adhesive capsulitis and rotator-cuff ailment. It is possible that the two conditions have similar patho-mechanism linked with DM: Affected micro-circulation with non enzymatic-glycosylation process are two examples (Fig. 1). In actuality, hyperglycemia leads to the development of advanced glycosylation end-products and the subsequent production of non-enzymatic glycosylation products. These AGEs (Advance Glycation End Products) promote collagen, tendon, and ligament cross-linking, making these tissues stiffer and less flexible. Additionally, AGEs cause inflammatory alterations by interacting with receptors at the surface of tenocytes and fibroblasts. Furthermore, the shoulder joint also experiences the adverse microvascular environment brought upon by hyperglycemia. The reduced circulation causes tissue hypoxia, an excess of free radicals, and ultimately cell death. Joint tissue breakdown and the acceleration of degenerative processes may result from this cumulative injury. [14]

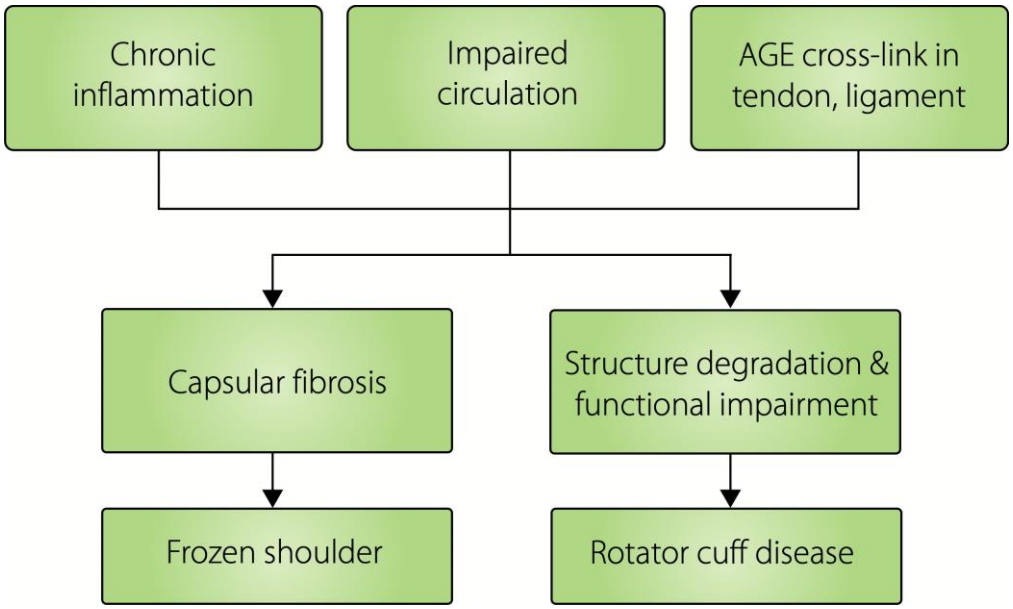



**FIG.1 – PATHOMECHANISM OF ADHESIVE CAPSULITIS AND ROTATOR CUFF DISEASES IN DIABETICS**

In the meantime, the cross linking collagen that builds up on shoulder capsule causes chronic inflammatory processes and joint stiffness due to hyperglycemia that may intensify inflammatory response at synovium. In the end, the shoulder joint's capsular fibrosis is a result of these findings. The process may shed light on the link between diabetes and frozen shoulder. Notably, the rotator cuff tendon experiences a similar pathogenetic pathway, which is accompanied with tendon degeneration, as well as morphological and functional abnormalities. AGEs cause the tendons to become weaker, and the rotator cuff tendon becomes more prone to damage due to poor circulation followed by an inflammatory response. As a result, the deteriorated rotator cuff tendon can be more susceptible to tears.

Numerous sonographic investigations conducted on general population demonstrate rotator-cuff lesions are more common as people get older. The prevalence of tears varies significantly over time, generally from 0 to 15 % during 1960s , till 30 to 50% during 1980s. The variations are elaborated by the features of the participants enrolled plus the sonographic criteria used to identify lesions.[1]

When it comes to identifying pathogenic changes in shoulders that are asymptomatic, MRI is a more accurate way compared to ultrasonography. However, its less availability and greater costs restrict its application for epidemiological objectives.

Moreover, research done on symptomatic people have demonstrated DM being a significant risk factor of rotator-cuff diseases. Additionally, diabetics exhibit a reduced ROM in shoulder with high incidence of re-tears following surgical repair.

Spontaneous condensation of glucose with metabolic intermediates ( triose phosphates, glyoxal, methylglyoxal) to amino-groups in lysine, hydroxylysine and arginine lead to covalent bond between sugar plus amino-acid ( Amadori compound), subsequent reaction gives rise to formation of A.G.E.s.

The amount of cross-links between collagen molecules increases as a result of AGEs' impact on the chemical and physical characteristics of proteins. Collagen becomes less soluble as a result, becoming hard, stiff and weak. It also looses elastic strength and is more susceptible tearing.

One more way that A.G.E.s might cause harm is by their interaction with certain receptors (R.A.G.E.), that are seen at membranes of chondrocytes, tenocytes and fibroblasts.

When a ligand engages RAGE, cell-specific signalling is triggered, which increases the production-of reactive oxygen specie then activates transcription of nuclear factor N.F.-k.B.[1] A defective cell phenotype results from continuous up regulation of pro-inflammatory mediators, adhesion molecules, with A.G.E.s crosslink formation at collagen fibres as a result of this.

Vascular endothelial growth-factor, cytokines, plus RAGE is over-expressed in diabetes, which may help to explain why people with diabetes have more lesions as well as inflammatory reactions.

In addition to A.G.E.-mediated pathogenetic pathway, micro-vascular illness can result in tissues hypoxia, which generates oxygen free radicals and causes an excess of growth factors and cytokines to be produced.

In order to put strategies in place to assist prevent shoulder pain from developing, it is vital to identify linked illnesses and hence potential risk factors because shoulder pain tends to be persistent and carries a heavy burden of disease. A correlation between shoulder pain and diabetes is supported by data from clinic research, and a number of investigations have found a link between musculoskeletal pain and the length of diabetes. The risk of developing microvascular and macro vascular problems from diabetes mellitus is well recognised to rise with poor glycemic control or chronic hyperglycemia. However, there is no proven link between poor diabetic management (as determined by HbA1c levels) and musculoskeletal problems in individuals with diabetes mellitus, according to clinical investigations. This may imply that the apparent link between shoulder discomfort and/or stiffness and diabetes is due to other reasons. (6)

Clinical samples from tertiary outpatient clinics and primary care settings have typically been used in studies on the relationships between shoulder discomfort and diabetes. According to these research, diabetes patients have a higher prevalence of shoulder complaints, ranging from 11% to 35%, as opposed to 2% to 17% in control groups. In a recent study, shoulder pain and impairment were found to be worse in people with diabetes compared to controls in a tertiary care setting. (6)

Data from population based research particularly examining the relationship between diabetes and shoulder symptoms are scarce. A relationship between diabetes mellitus and chronic rotator cuff tendinitis was found in a study comparing the factors that influence specific shoulder disorders against nonspecific shoulder pain, but it was not discovered in the same way for nonspecific shoulder pain. However, a study of Finnish workers found no evidence of a link between diabetes or elevated plasma glucose levels and chronic shoulder conditions. After adjusting for age and sex,

another study that included participants from the Mini Finland Health Survey discovered that diabetes was linked to shoulder disability, with an OR of 1.6 (95% CI 1.2-2.1). [6]

The first mention of joint mobility restriction in diabetes mellitus dates back to 1957. The condition, known as cheiroarthropathy, was particularly noticeable in the hands. The term "restricted joint mobility" more correctly defines the phenomenon because the pathology is likely in the periarticular collagen and since it is not specific to the hands.

Between 8% and 42% of people with diabetes mellitus have reported having restricted joint mobility. Both insulin-dependent diabetes (IDDM) and non-insulin-dependent diabetic mellitus (NIDDM) were the first conditions in which it was first described.[5] It has been established that restricted joint mobility is linked to other diabetes problems, particularly retinopathy and nephropathy. Although the connection between shoulder capsulitis, another well-known aspect of diabetes, and restricted joint mobility is less clear, Fisher et al. did discover one. [17]

Limited joint motion most likely has multiple contributing factors. Type II collagen can exhibit increased non-enzymatic glycosylation in vitro when incubated with high glucose concentrations. Collagen fibres become cross-linked as a result of glycosylation, making them more resistant to enzymatic deterioration. The same thing happens to hair, and it has been found that higher hair glycosylation levels in diabetes correspond with higher levels of glycosylated haemoglobin. This could simply be a result of the differing metabolic rates at which collagen and haemoglobin degrade. Microvascular disease and regional mechanical variables are two additional factors that may contribute to the development of restricted joint mobility. [7]

It is recognised that the buildup of AGEs plays a role in the pathologies connected to longterm consequences-of DM. The A.G.E.s have been linked to the aetiology of maladies such cartilage

degeneration (osteoarthritis [O.A.]), vascular stiffness, along with different amyloidoses, and they also play a significant part in the overall ageing process. The creation with detection of A.G.E.s, pathophysiological effects-of A.G.E. accumulation on extra-cellular matrix, with a focus over articular cartilage, along with emergence of anti aging therapies are all discussed in this review. (11)

**Chemistry of AGE (Advanced Glycation End Products) formation**

It has long been believed that non enzymatic glycation, common post translational modification of protein with reducing sugars, is solely caused because of spontaneous condensation of reducing sugar (like glucose) with metabolic inter-mediates (like triose phosphates, glyoxal, methyglyoxal) by aminogroups lysine, hydroxy-lysine, arginine residues. Subsequent reactions lead to the production of AGEs after the first establishing covalent link between sugar and amino acid
( Amadori compound). It is now known that in addition to lipid peroxidation, metal catalysed glucose auto-oxidation can also cause AGE formation. Lipids, not glucose, seem to be the main cause of AGE generation and the ensuing loss of renal function in Zucker rats with hypertriglyceridaemia. Advanced lipoxidation end products (ALEs) are the name given to these

AGEs formed from lipids to emphasise the alternative process underlying their synthesis. Recently, a thorough analysis of the expanding significance-of alteration-of protein with lipids on diabetics is published. Thus, the generation of AGE in vivo has a variety of origins and methods. Many of the contributing variables are still unclear, despite the increasing understanding of the variety of processes that go into the development of AGE in vivo. Other factors, in addition to environmental ones like nutrition and smoking, also contribute to the development of AGEs. Serum measurements of AGEs, such as carboxy-methyl-lysine, on group of healthy, non diabetic female monozygotic or di-zygotic twin showed the majority of the variance in circulating AGE levels is hereditary. The production of A.G.E.s by a range of chemical structures is another effect of these extremely different

reaction path-ways leading A.G.E. formation. CML and carboxyethyllysine are two examples of AGEs that act as protein adducts, while other AGEs have protein-protein crosslinks (pentosidine, glyoxal de-rived lysine dimer plus methylglyoxal de-rived lysine dimer). Most
A.G.E.s (more recently, ALEs) are discovered since Sell and Monnier's characterisation of the AGE crosslink pentosidine in the late 1980s. It is uncertain which A.G.E.s, if any, is to-be called significant, as more A.G.E.s continuously are found. (11)

It is becoming increasingly obvious that AGE quality that is, kind along with location—is contributing impaction-of A.G.E. accumulation along with sensitive quantification-of all A.G.E.s. The predominant mechanism of CML synthesis, as revealed by proteomic analysis utilising electrospray liquid chromatography mass spectrometry, by self oxidation of Amadori compound rather than glyoxal, that gets produced through self-oxidation of glucose. Additionally, particular preferred places within a protein are where Amadori compound first forms, along with sub-sequent processes that lead to the creation of CML ( RN.ase). Neighbouring amino acids with bound-ligands have an impact on each of these processes (like phosphate / phosphorylated compound). One research used matrix assisted laser desorption / ionization-time-of-flight mass spectroscopy identifying kind with location of A.G.E.s generated in vitro (by incubations along glucose) as well as in vivo. This method was slightly different from the one used in the previous study (in diabetic rats). The findings once again point to specific, preferential sites within proteins where AGE is formed, and they imply that even in cases where protein modifications are minimal overall, localising significant portion of modifications on small number of re-active site may show significant ramifications to understand change of protein functionality. (11)

A small amount-of A.G.E.s are found in vivo, comprising pentosidine, C.M.L., carboxy-ethyl-lysine, argpyrimidine, pyralline, methyl-glyoxal derived lysine dimer, with glyoxal derived lysine dimer, despite advances in our understanding chemicals and mechanisms which lead-to generation-

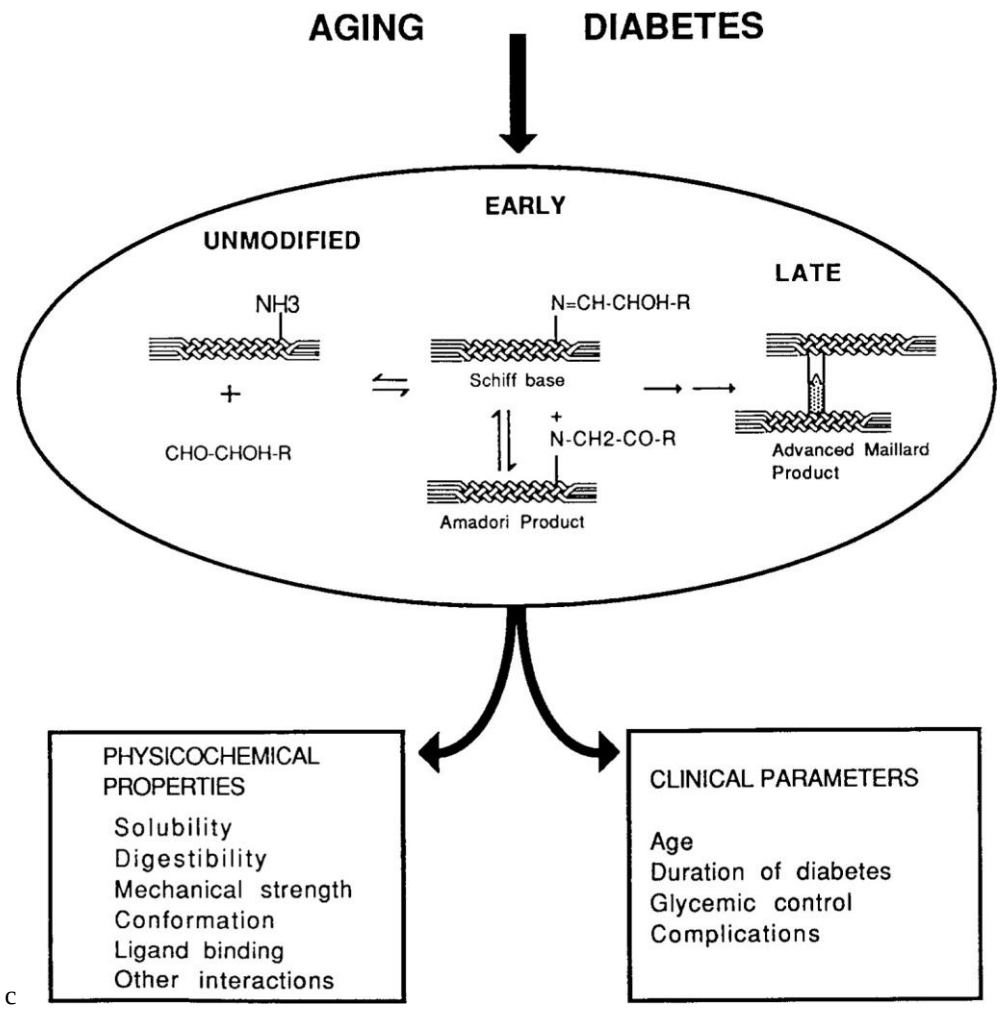
of AGEs. Immunoassays (E.L.I.S.A.) / chromatographic techniques like high- performance-liquid-chromatography ,gas-chromatography-mass-spectroscopy are typically used to detect these A.G.E.s. Latest invention-of liquid-chromatography-mass-spectroscopy approach which enables simultaneous quantitative measurement of Sixteen bio-markers indicating protein glycation, oxidation, along with nitrosation are key tool in light of the growing variety of AGEs.

These cutting-edge techniques are essential for understanding AGE formation mechanisms in greater detail and assessing cutting-edge treatments meant to reduce AGE accumulation. (11)

Only until the protein from which they were created is broken down do AGEs begin to break down. Significant accumulation of A.G.E.s therefore happen in tissue which contains long lived proteins, or-in proteins having slower turn-over, like crystallin of eye lens as well as collagen of extra- cellular matrix in connective tissues (like cartilage, osteon, tendon, or cuticle). This is because proteins as well as lipoproteins ( intra-cellular or extra-cellular) which have lysine, hydroxy-lysine, and arginine residual is susceptible to A.G.E. formation. AGEs are known to influence the chemical and physical characteristics of proteins as well as the tissues in which they are found. For instance, collagen cross linking between molecules affects the strength of connective tissue. Accumulation of AGE crosslinks in articular cartilage causes the tissue to become more rigid and brittle, making it more vulnerable to mechanical damage. Similar to this, AGE buildup is linked to a rise in tissue stiffness in arteries, lenses, skin, and tendons. In the early 1990s, studies on rat cortical bone and human lens capsules showed that AGE buildup increased tissue brittleness. However, a more recent study reveals that in vitro incubation with glucose has no effect on the mechanical properties of rat bones, although the same incubation has an impact on the mechanical properties of rabbit tendons. In addition to altering the mechanical characteristics of tissues, AGEs also prevent matrix proteins from being susceptible to proteolytic destruction. Comparatively to unmodified collagen, matrix metalloproteinases have a harder time degrading AGE-modified cartilage collagen. The pathogenic aggregates of amyloid fibrils of amyloidoses, like Alzheimer's disorder as well as familial amyloidosis, are frequently

changed via A.G.E.s. For-long there was no clarity if A.G.E.s caused plaque development by reducing protein solubility, or-if accumulated plaque proteins were more vulnerable to AGE accumulation due to their slower turnover. Starting function-of A.G.E.s of the process is now supported by the finding that AGE production causes protein aggregation by causing albumin, which was initially globular, to refold into amyloid fibrils with the distinctive cross-beta structure.[11] Additionally, AGEs disrupt biological functions like gene expression, cell proliferation, and adherence to the extracellular matrix. At higher AGE levels, articular cartilage chondrocytes produce less collagen and proteoglycan. Presence of A.G.E. receptor (R.A.G.E.) over chondrocytes suggest importance of the pleiotropic receptor, even if the exact method by which AGEs affect chondrocytes is yet unknown. RAGE was initially discovered to be a receptor for AGEs, but it also has key ligands in inflammatory mediators with high-mobility groupbox I with cal-granulin/S100, and the majority of research of R.A.G.E. focuses over inflammatory illnesses (like inflammatory arthritis). It can be partially due to the fact that it is yet unclear which AGE biochemical properties are responsible for AGE binding and activation. A paper examined A.G.E.s which attach with R.A.G.E. with its in vitro production kinetics using standardised protocols and a range of AGE measurements (such as fluoroscence, absorbance, carbonyl-content, reactive-free- amino-content, molecular weight, pentosidine levels, as well as C.M.L. levels). Only the free amine content of the AGE measurements had a strong correlation with RAGE binding affinity. RAGE initiates intracellular signal transduction pathway which is partially known upon interaction of these ligands. Nuclear factor-kB is activated as a result of AGE with albumin inducing R.A.G.E.- mediated-activation of p-21ras with mitogen-activated protein-kinase. The rheumatoid arthritis patients' synovial fluid-derived macrophages, HMGB-1 causes the release of interleukin-1b, interleukin-6, as well as tumour necrosis-factor-A via the RAGE pathway. These investigations may serve as the starting point for additional investigation of relationship bet-ween R.A.G.E. along with ligands with potential anti-A.G.E. treatments that target receptors.[11] The combination of cartilage's in-creased sensitivity to

mechanical-damage along with chondrocytes' diminished ability of remodelling (thereby repairing) the extra-cellular matrix around them makes cartilage vulnerable for destruction. This may give molecular mechanism from-which the age related buildup of A.G.E.s causes osteoarthritis. Moreover, it was recently shown that the buildup of A.G.E.s predisposes to Osteoarthritis utilising a in-vivo structure of the disease. Animals having increased A.G.E. limits of articular cartilage demonstrated statistically significant rise of OA severity when contrasted to dogs having non diseased cartilage A.G.E. limits utilising well-established dog ACL transection model in Osteoarthritis. This work is the first to offer a biological explanation for the rise in OA incidence associated with ageing.(11)




c

**FIG.2 - EFFECT OF AGEING WITH DM OVER NON ENZYMATIC GLYCATION OF COLLAGEN ALONG WITH ITS RELATIONSHIP WITH PHYSICO-CHEMICAL CHANGES IN PARAMETERS. UNMODIFIED COLLAGEN SCHEMATICALLY SHOWN REACTING WITH GLUCOSE FORMING EARLY AND LATE GLYCATION END PRODUCT**

## ANATOMY(31,32,33)

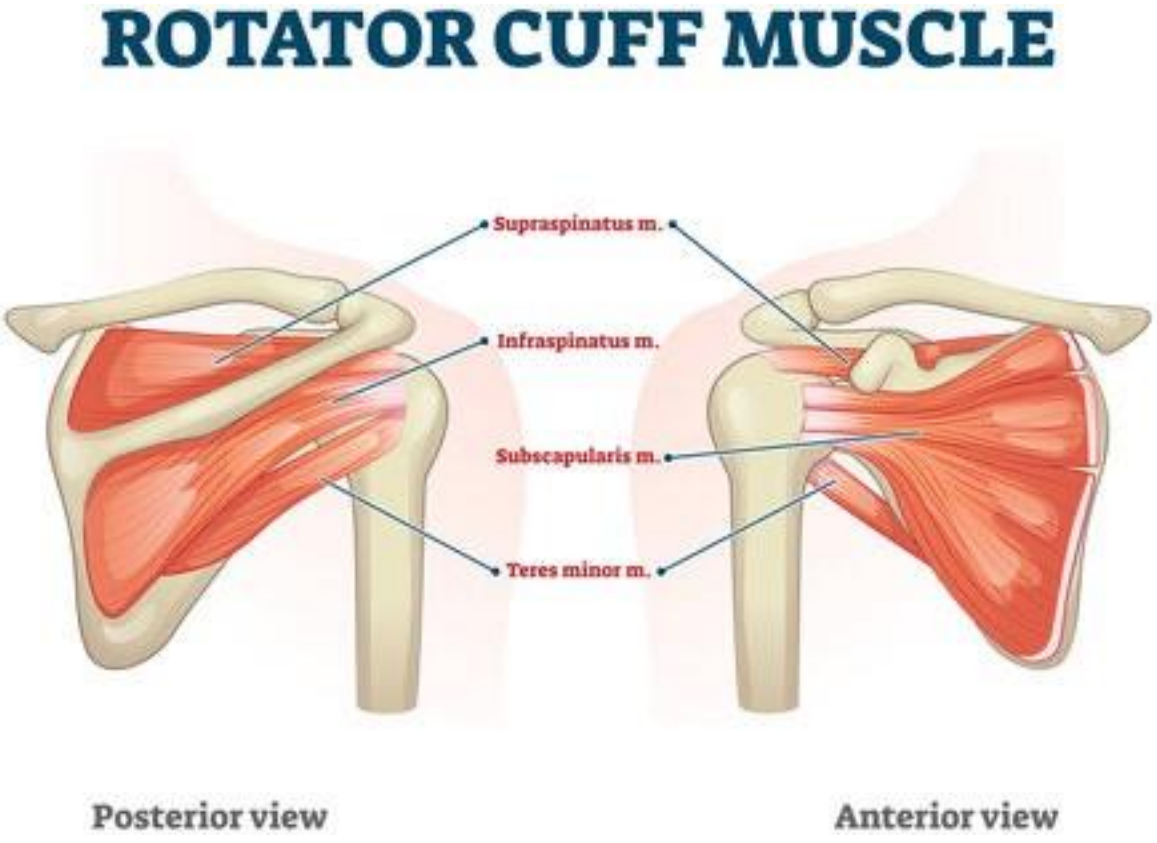



**FIG.3-ANTERIORANDPOSTERIORVIEWSOFROATORCUFFMUSCLES**

Muscles of shoulder called rotator-cuff allows for extensile ROM along with preserving a stable gleno-humeral-joint. Following muscles form rotator-cuff:

Subscapularis

Infraspinatus

Supraspinatus

Teres minor

The large spherical humerus head with timid glenoid form gleno-humeral-joint,“ball and socket

joint". This joint being extensively mobile due to its architecture, is highly unstable. Gleno-humeral joint's non-contracting tissues(static stabilizers), that are Glenoid labrum, joint-capsules, negative intra-articular pressures, along with gleno-humeral ligament, as well as contracting tissue (dynamic stabilizers), that are rotator-cuff musculo-tendinous unit along with long head of biceps-brachi, synchronize stabilizing gleno-humeral joint.

**Arrangement and Purpose**

Rotator-cuff's primary bio-mechanical activity is compressing humeral head on glenoid-cavity stabilizing gleno-humeral joint. All four muscles insert over humeral head originating at scapula. Inferior-part of joint remains unprotected as rotator cuff muscles' tendon merge with joint capsule forming musculo-tendinous-cuff over posterior, superior, as well as anterior part, which stands important as most subluxations occur if humeral head goes inferior via joint's exposed surface. Rotator-cuff muscle contracts at the time of shoulder ROM, stopping humeral head to slide, which allows complete ROM with a stable joint. Also, allowing the abduction, medial rotation, lateral rotation, rotator cuff muscle aids mobilizing shoulder .

- Subscapularis: Medial (internal) rotation at shoulder
- Supraspinatus: Abduction at the arm
    - First Zero-Fifteen degree at shoulder abduction motion, it is necessary.
    - Beyond a 15-degree angle, Deltoid abducts shoulder.
- Infraspinatus: Lateral (external) rotation at shoulder
- Teres-Minor: Lateral (external) rotation at shoulder

These muscles could be independently evaluated during a physical examination based on their unique motions.

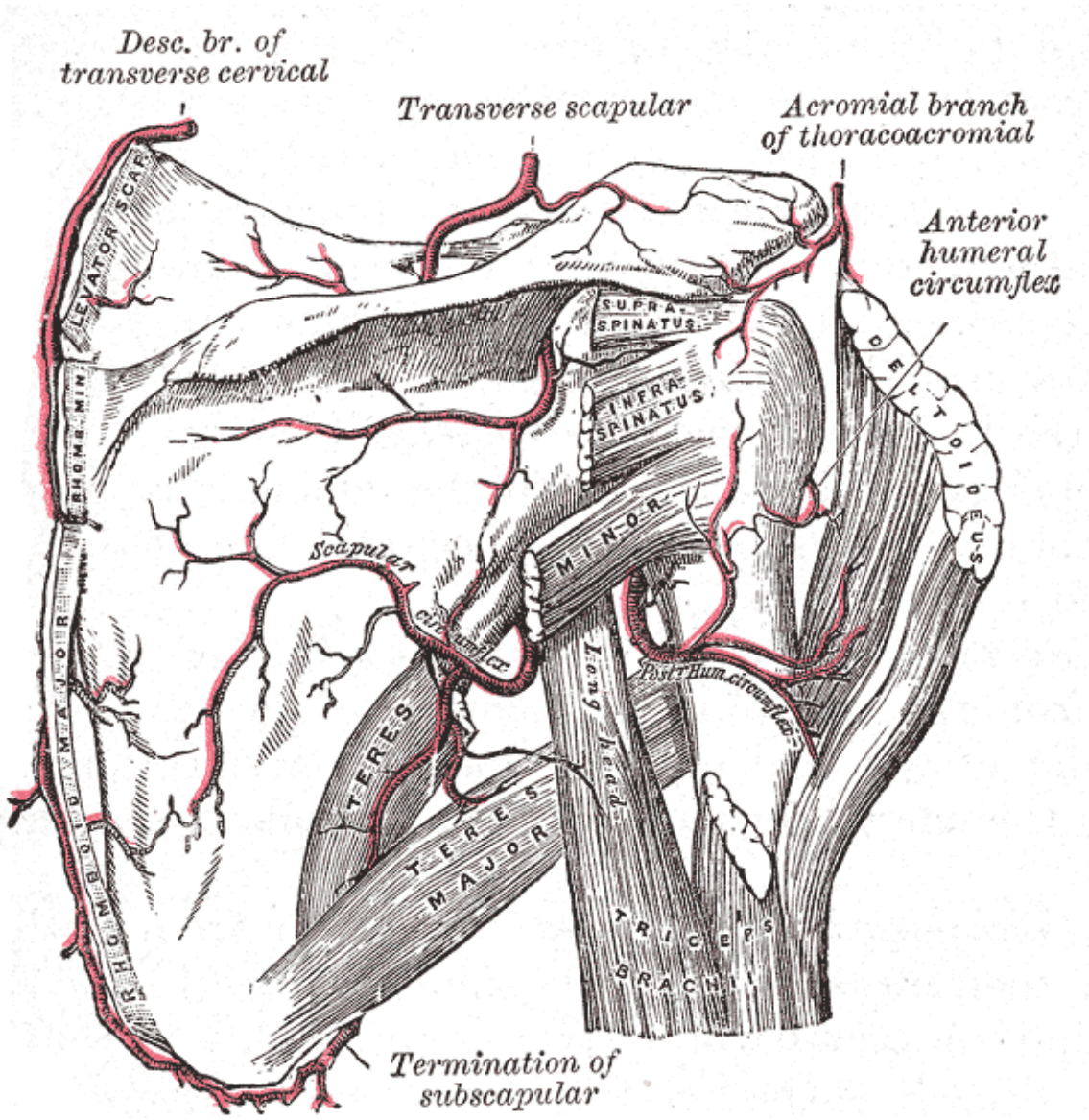



FIG.4-VASCULATUREOFTHEROTATOR CUFF

**Embryology**-The body's muscles and ligaments are derived from mesoderm.

**Circulation and Lymphatic drainage:** Supra-scapular, sub-scapular, along with posterior-circumflex-humeral arteries provide the majority of rotator-cuff's vascular supply. **S**upra-scapular artery starts near cervical base. It is branch of thyrocervical trunk, which is a significant branch of Subclavian artery. Supraspinatus and infraspinatus is supplied by it as it enters posterior scapular area superior of supra-scapular foramina (nerve passes from foramina).

Sub-scapular artery makes axillary-artery's biggest branch. Circumflex scapular artery with thoraco-dorsal artery emerge from 3$^{rd}$ segment of axillary artery after travelling along inferior subscapularis. Subscapularis muscle receives vascular supply from it.

3rd segment of axillary artery at axilla serves as origin of the posterior circumflex humeral-artery. Axillary nerve travels along with it as it reaches the quadrangular space in posterior scapular area for supplying the teres minor muscle.

Lymph nodes of axilla are final destination of all upper limb lymphatics.

**Neurology:**

Subscapularis gets innervated from sub-scapular nerve (proximal and distal branch).

- Originates at brachial plexus's posterior cord.
- C 5,6,7

Infraspinatus along with supraspinatus gets innervated from supra-scapular nerve.

- Originates at brachial plexus's superior trunk.
- Traverses supra-scapular foramina.
- C5and C6

Teres-minor gets innervated from axillary nerve.

- Originates at brachial plexus's posterior cord.
- Enters posterior scapular region via quadrangular space
- C5and C6

**Muscles-**Largest area at axilla's posterior wall comprises subscapularis, which medially rotates humerus and prevents anterior humeral dislocation during abduction. The muscle and scapular-neck are divided by a sizable bursa.

- Originates from scapula’s sub-scapular fossa.
- Inserting at Lesser humeral tubercle

Only rotator-cuff muscle that isn’t a rotator of the humerus is supraspinatus.

- Origin: the scapula's supraspinous fossa
- Overlies the gleno-humeral joint
- Greater humeral tuberosity insertion

An effective external-rotator at humerus is infraspinatus. A bursa may occasionally exist between the tendon of this muscle and the gleno-humeral joint capsule.

- Origin: Scapula’s infraspinous fossa
- Inserting at greater tubercle on humerus, immediately distal to supraspinatus.

Deltoid covers the entire teres minor muscle, which is long and slender and hardly distinguishable with infraspinatus.

- Originates from lateral scapular-surface.
- Inserting at humeral greater tuberosity

**Normal Range of Movements at Shoulder Joint-**

**Forward Flexion :** Zero to 150-180 degrees.

**Extension:** Zero to 45-60degrees.

**Abduction :** Zero to 150 degrees.

**Adduction :** Zero to 30 degrees.

**Internal (Medial) Rotation:** Zero to 90 degrees.

**External (Lateral) Rotation:** Zero to 90 degrees.

# METHODS AND METHODOLOGY

**Source of data collection :**

Patients with shoulder pain or restricted shoulder movements presenting to the Out Patient Department of Orthopaedics – K.L.E.S. Dr. Prabhakar Kore Hospital and Medical Research Centre And Charitable Hospital, Belagavi in between 1st January 2021 to 31st December 2021, over period of 1year.

**Study design :**

A hospital based one year Cross Sectional Study.

All patients who come to the orthopaedics OPD with symptoms of shoulder pain and restricted range of movement at affected shoulder joint, who are known case of Diabetes Mellitus Type II, will be clinically evaluated and will be suggested to get an Ultrasonography of the affected shoulder.

**SAMPLE SIZE: 75**

**Sampling method :**

Minimum sample size formula based over prevalence rate :

$$'n' = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

where P stands for percentage prevalence, 'd' percentage likely difference of prevalence. "$z_\alpha$" links to level-of significance. In 5percent level significance "$z_\alpha = 1.96$."

Ref. : With "P = 45%" "d = 25% P" = 11.25%, sample-size is 75.

**Study Requirements :**

Known case of type II diabetes mellitus presenting to the Out Patient Department of orthopaedics with shoulder pain , restricted shoulder movements or both.

**SELECTION CRITERIA:**

**INCLUSION CRITERIA:**

Known case of diabetes mellitus type II

Age > 18 years

No history of trauma

No history of surgery

**EXCLUSION CRITERIA:**

Rheumatic Disorders

Endocrinopathies

Malignancies

Systemic Diseases (Renal, Hepatic, Cardiac)

Treated by corticosteroids

**Methods:**

In this study we are assessing prevalence of rotator cuff diseases in type II diabetes mellitus

A one year hospital based cross sectional study.

**Procedure:**

All patients who come to the orthopaedics OPD with symptoms of shoulder pain and limited ROM at affected shoulder joint and who is a known case of Diabetes Mellitus, will be clinically evaluated and will be suggested to undergo Ultrasonography of the affected shoulder.

# PICTORIAL REPRESENTATION OF ULTRASONOGRAPHY OF SHOULDER [18]

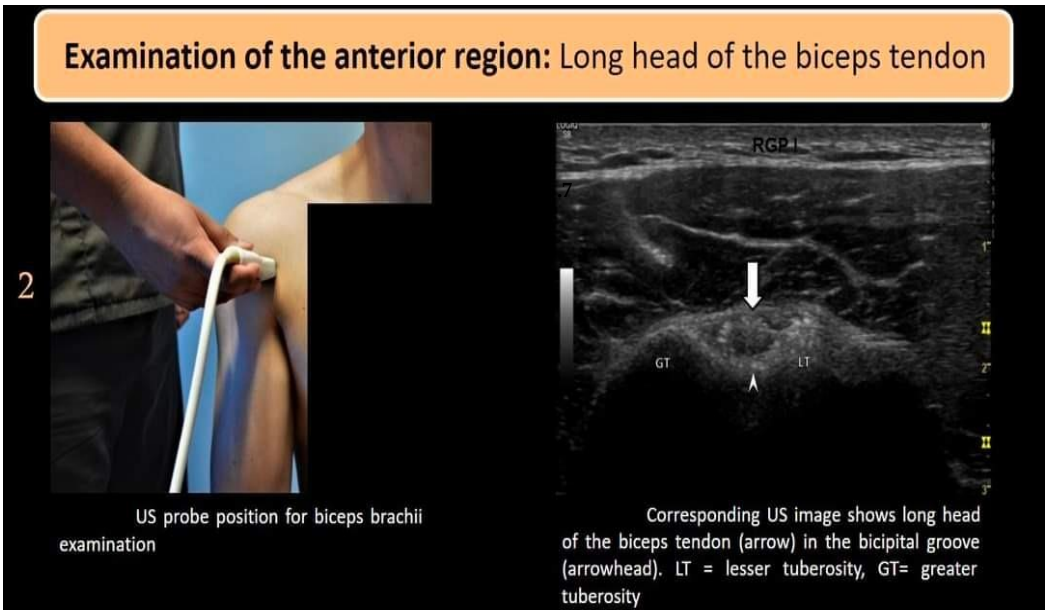



**FIG.5 USG EXAMINATION OF THE ANTERIOR REGION: LONG HEAD OF BICEPS TENDON**

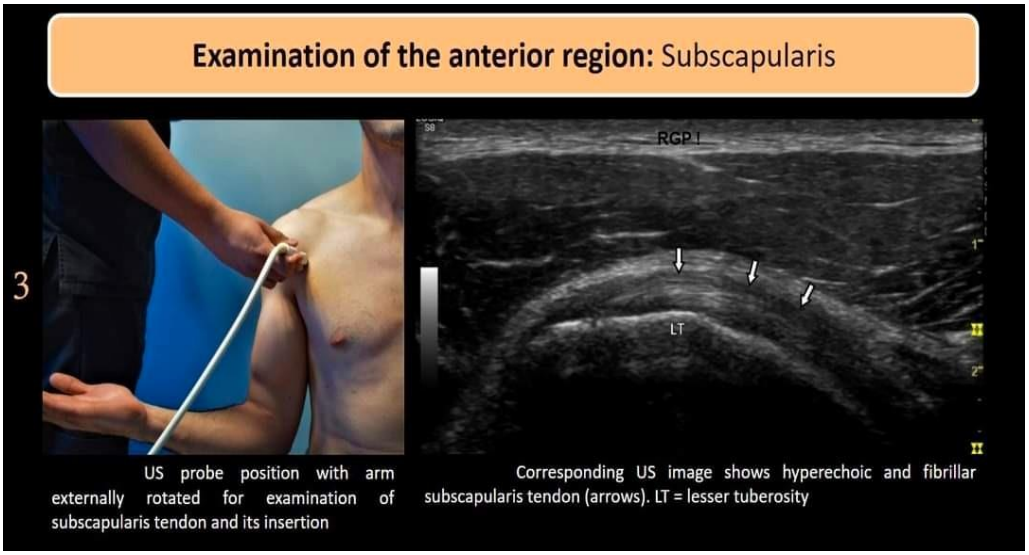



**FIG.6 USG EXAMINATION OF THE ANTERIOR REGION: SUBSCAPULARIS**

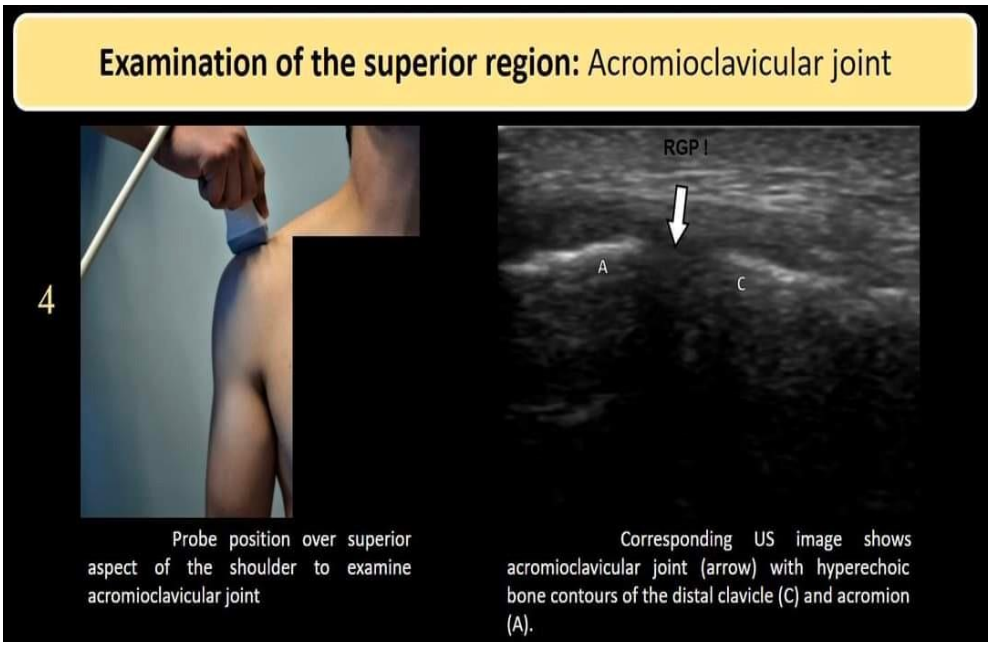



**FIG.7 USG EXAMINATION OF THE SUPERIOR**

**REGION: ACROMIO-CLAVICULAR JOINT**

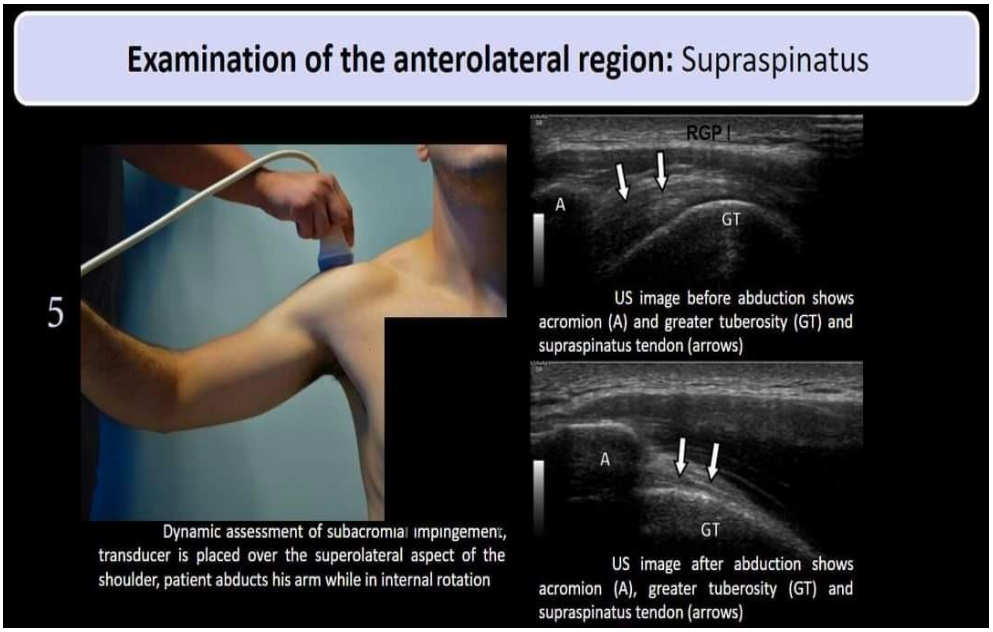



**FIG. 8 USG EXAMINATION OF THE ANTEROLATERAL**

**REGION: SUPRASPINATUS**

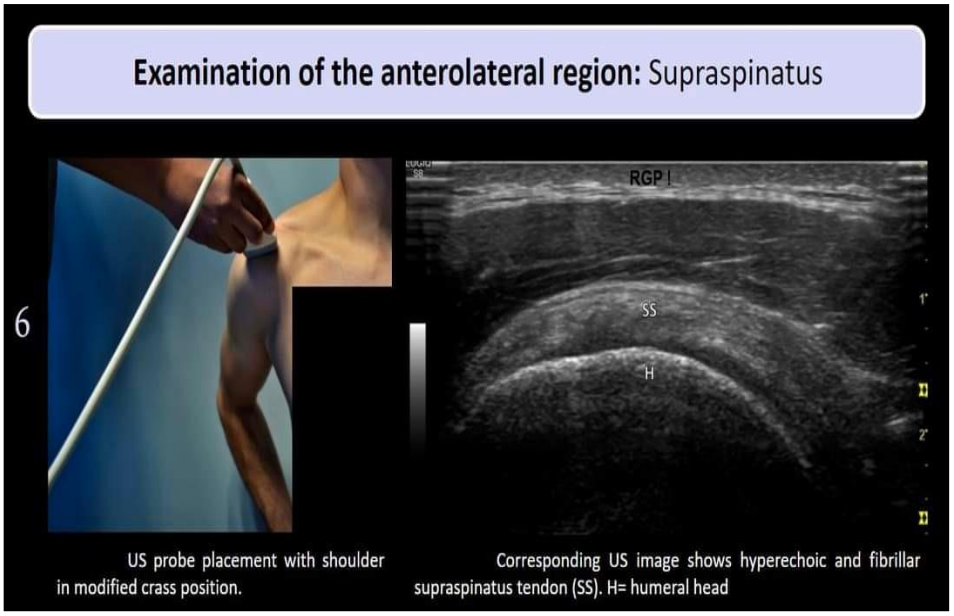



**FIG.9 USG EXAMINATION OF THE ANTEROLATERAL**

**REGION: SUPRASPINATUS**

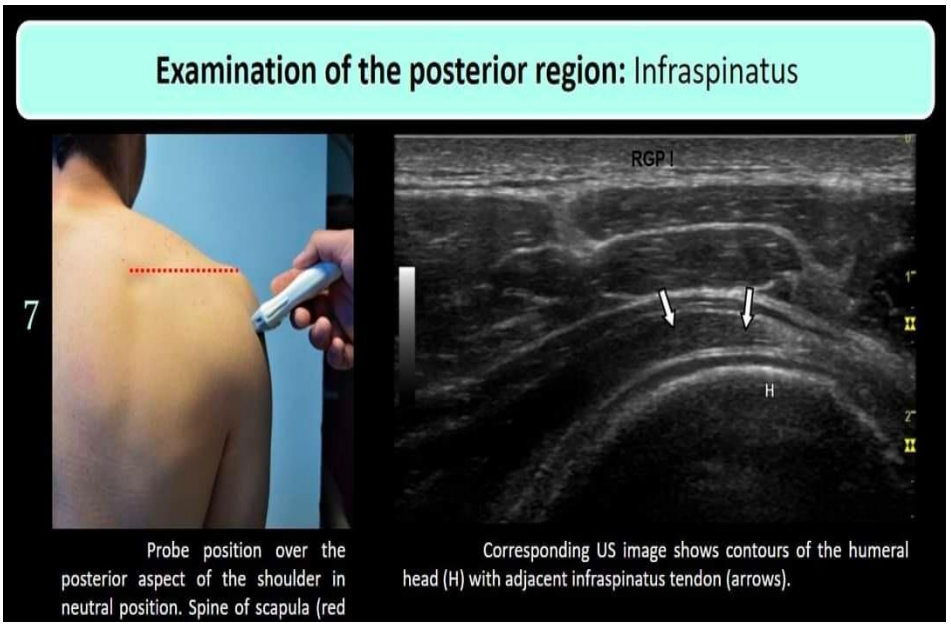



**FIG.10 USG EXAMINATION OF THE POSTERIOR**

**REGION: INFRASPINATUS**

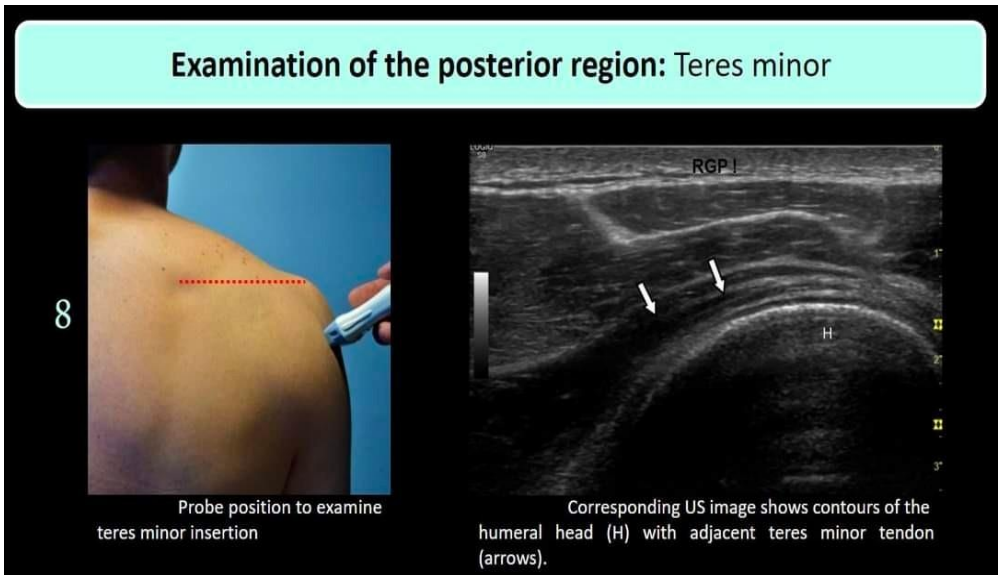



**FIG.11 USG EXAMINATION OF THE POSTERIOR**

**REGION: TERES MINOR**

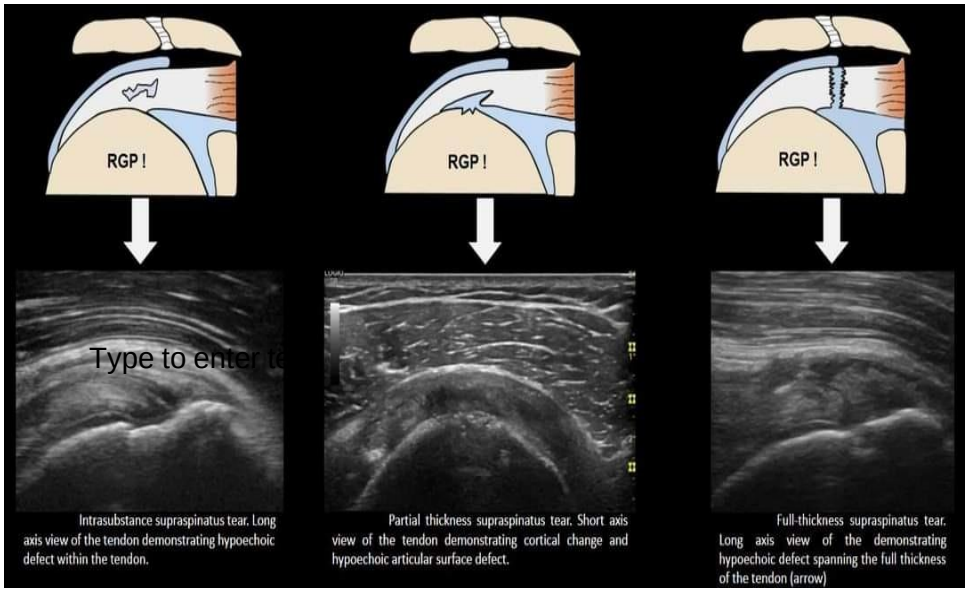



**FIG.12 USG SHOWING INTRA SUBSTANCE,**

**PARTIAL THICKNESS AND FULL THICKNESS SUPRASPINATUS TEARS**

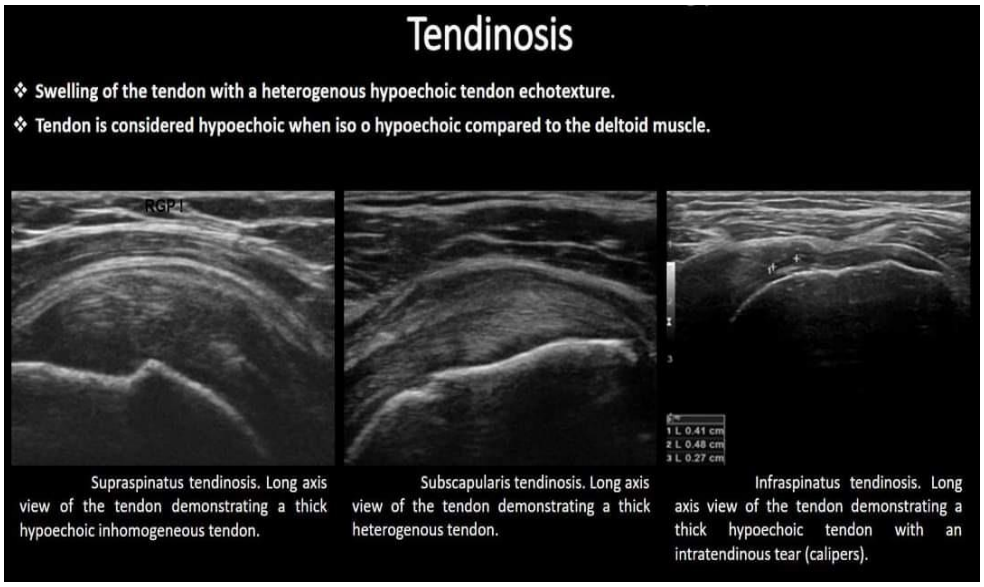



**FIG.13 USG SHOWING ROTATOR CUFF TENDINOSIS**

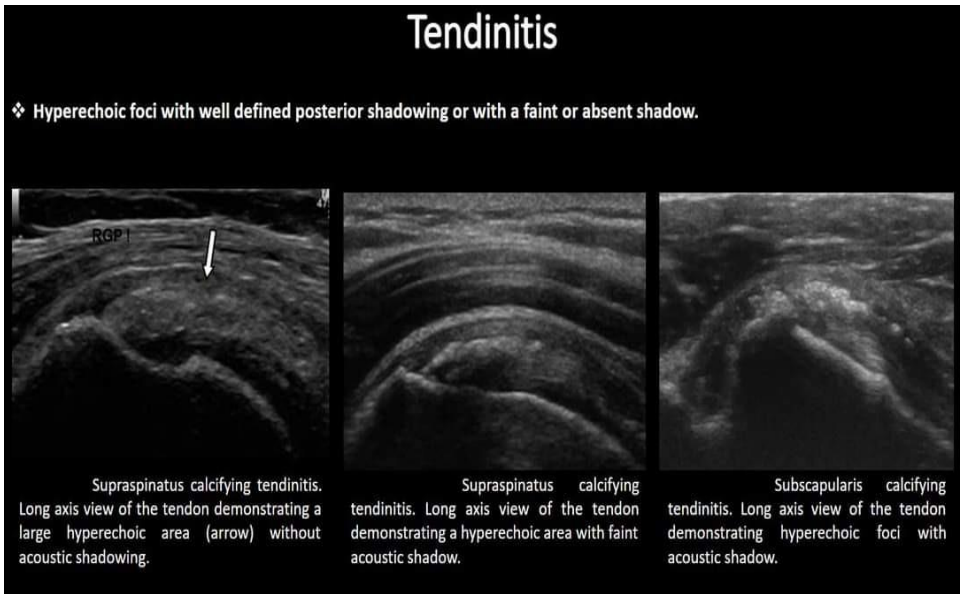



**FIG. 14 USG SHOWING ROTATOR CUFF TENDINITIS**

# STATISTICAL ANALYSIS

As this study being an observational-study, analytical plans are stated below.

For continuous quantitative variable mean and standard deviation is to be calculated. For comparison purpose, if data is divided in 2 groups pertaining a specific qualitative characteristic, continuous variables will be compared using suitable tool of statistics such as “student’s unpaired t test.” Pre as well as post treatment measures will be compared by “student’s paired t test”

Discrete variables will be represented by median.

The categorical data is expressed in terms of rate, ratio & percentage. Association between outcome, clinical & demographic characteristics is measured by “Chi-square test”, test of proportion or “Fisher’s exact test.”

In discrete variables nonparametric test will be used.

Along with stated tests, appropriate tools like ANOVA, correlation, regression, is utilized as per the requirement.

Appropriate graphs are utilized for depicting comparison. For all tests, value of “p” < 5 percent(0.05) is considered significant.

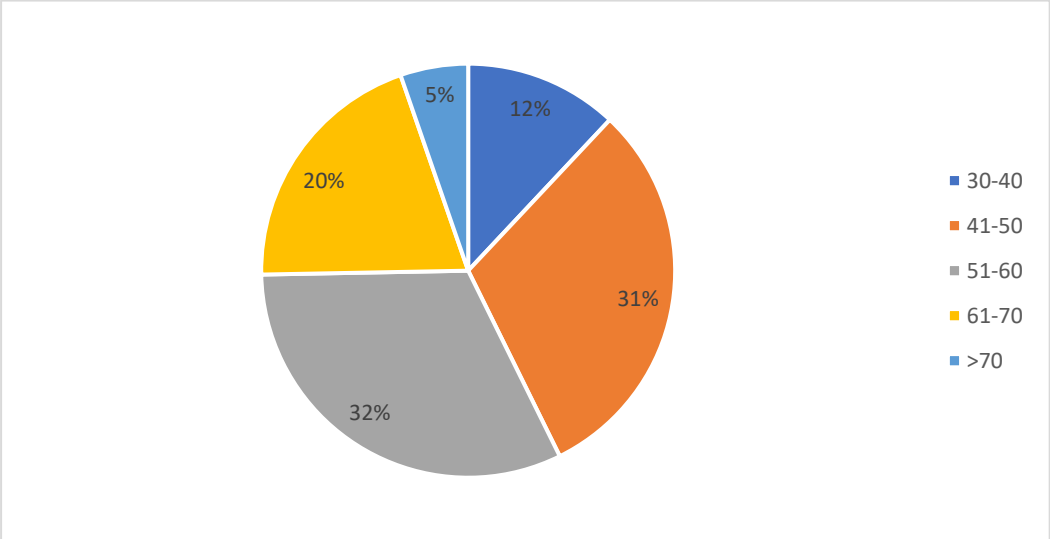
## RESULTS:

### AGE GROUP OF THE STUDY PARITICPANTS:

| AGE GROUP | FREQUENCY | PERCENT |
|---|---|---|
| **30-40** | 9 | 12.0 |
| **41-50** | 23 | 30.7 |
| **51-60** | 24 | 32.0 |
| **61-70** | 15 | 20.0 |
| **>70** | 4 | 5.3 |
| **MEAN ± SD** | 54.12 ± 10.465 | |

32% of the study participants were of the age group between 51-60 years of age followed by 30.7% in the age group between 41-50 years of age. The mean age of the study participants was found to be 54.12 ± 10.465 years.

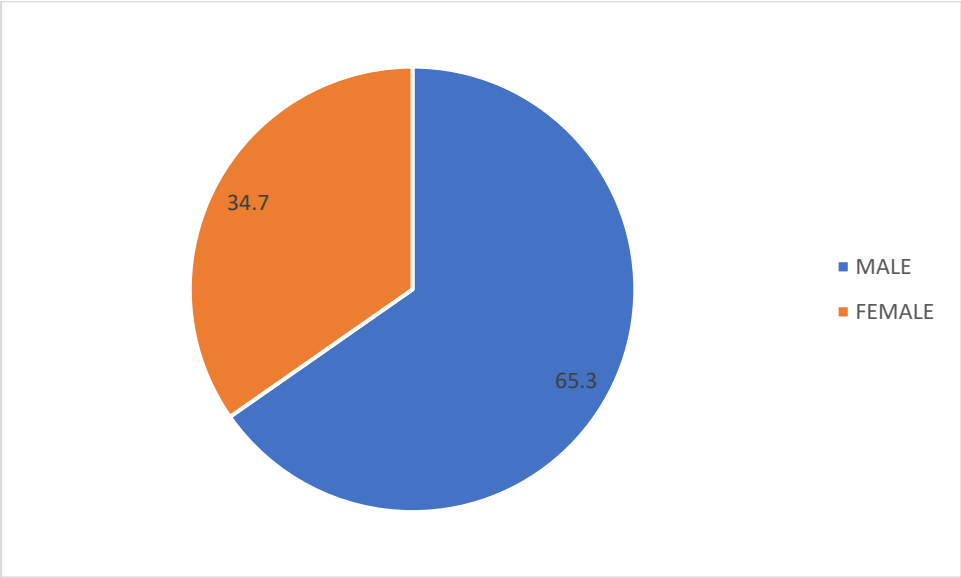
### AGE GROUP OF THE STUDY PARITICPANTS:



**GENDER OF THE STUDY PARITICPANTS:**

| GENDER | FREQUENCY | PERCENT |
|---|---|---|
| **MALE** | 49 | 65.3 |
| **FEMALE** | 26 | 34.7 |

**GENDER OF THE STUDY PARITICPANTS:**



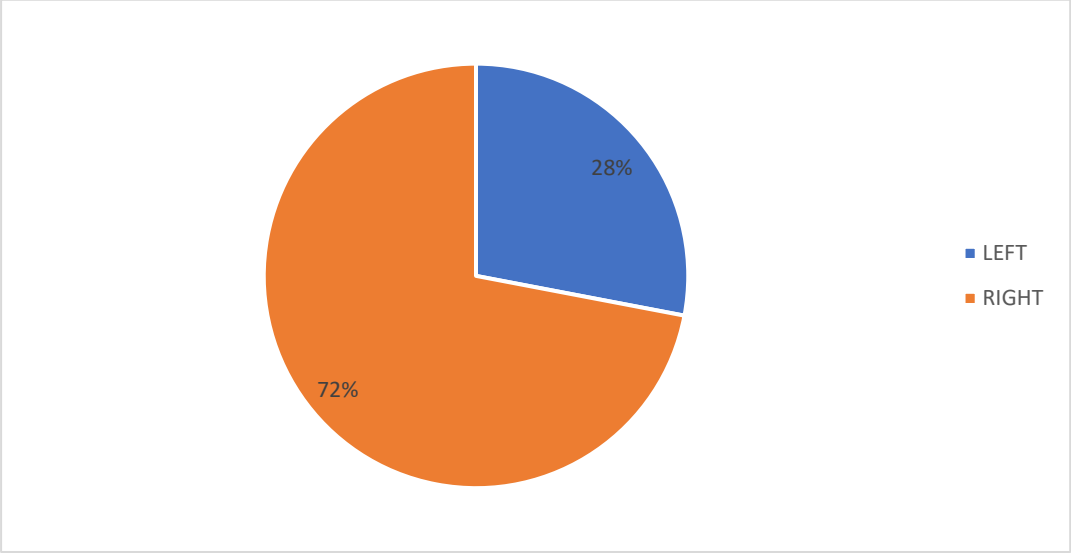

65.3% of the study participants were males with females contributing to 34.7% of the study participants.

**SIDE OF AFFECTED SHOULDER OF THE STUDY PARITICPANTS:**

| AFFECTED SHOULDER | FREQUENCY | PERCENT |
|---|---|---|
| **LEFT** | 21 | 28.0 |
| **RIGHT** | 54 | 72.0 |

72% of the study participants were affected on the right shoulder and 28% of the study participants were affected on the left shoulder**.**

**SIDE OF AFFECTED SHOULDER OF THE STUDY PARITICPANTS:**

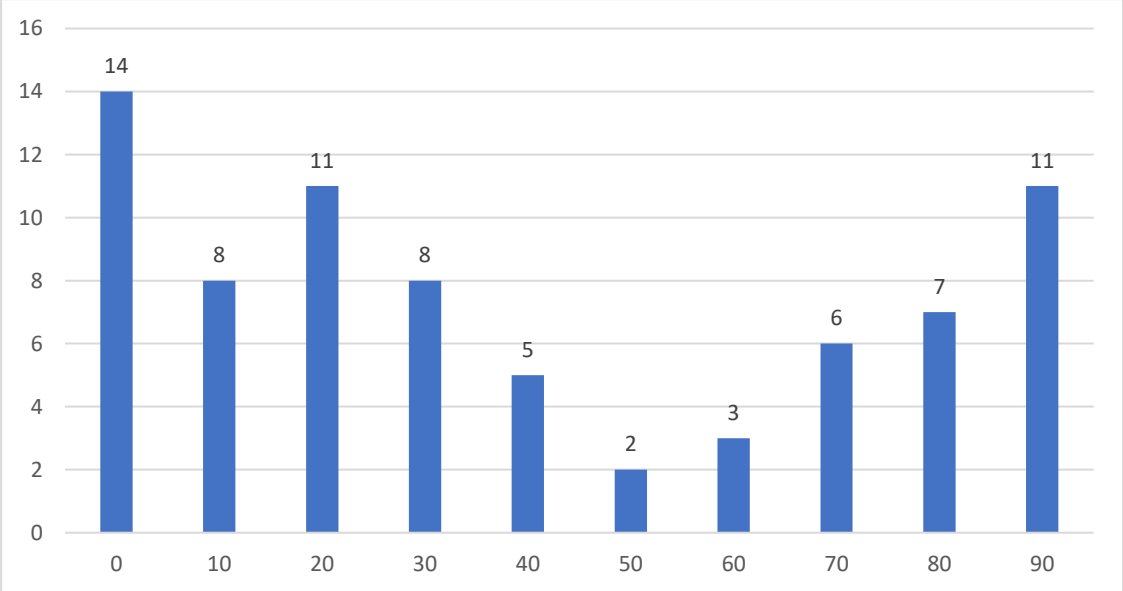


**RESTRICTION OF FLEXION OF THE STUDY PARITICPANTS:**

| RESTRICTION OF FLEXION (DEGREES) | FREQUENCY | PERCENT |
|---|---|---|
| 0 | 14 | 18.7 |
| 10 | 8 | 10.7 |
| 20 | 11 | 14.7 |
| 30 | 8 | 10.7 |
| 40 | 5 | 6.7 |
| 50 | 2 | 2.7 |
| 60 | 3 | 4.0 |
| 70 | 6 | 8.0 |
| 80 | 7 | 9.3 |
| 90 | 11 | 14.7 |

The range of restriction of flexion of the study participants is seen in the following order: 0°> 20° and 90° > 10° and 30° > 80° > 70° > 40° > 60° > 50°.

**RESTRICTION OF FLEXION OF THE STUDY PARITICPANTS:**

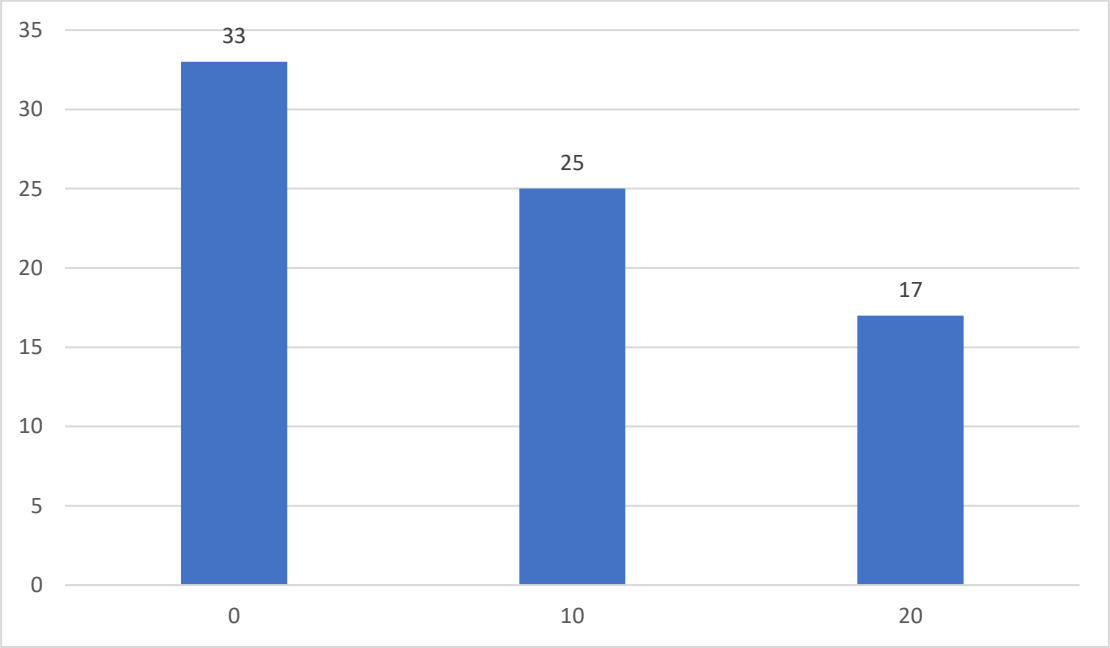


**RESTRICTION OF EXTENSION OF THE STUDY PARITICPANTS:**

| RESTRICTION OF EXTENSION (DEGREES) | FREQUENCY | PERCENT |
|---|---|---|
| 0 | 33 | 44.0 |
| 10 | 25 | 33.3 |
| 20 | 17 | 22.7 |

The range of restriction of extension of the study participants is seen in the following order: 0°> 10° > 20°.

**RESTRICTION OF EXTENSION OF THE STUDY PARITICPANTS:**

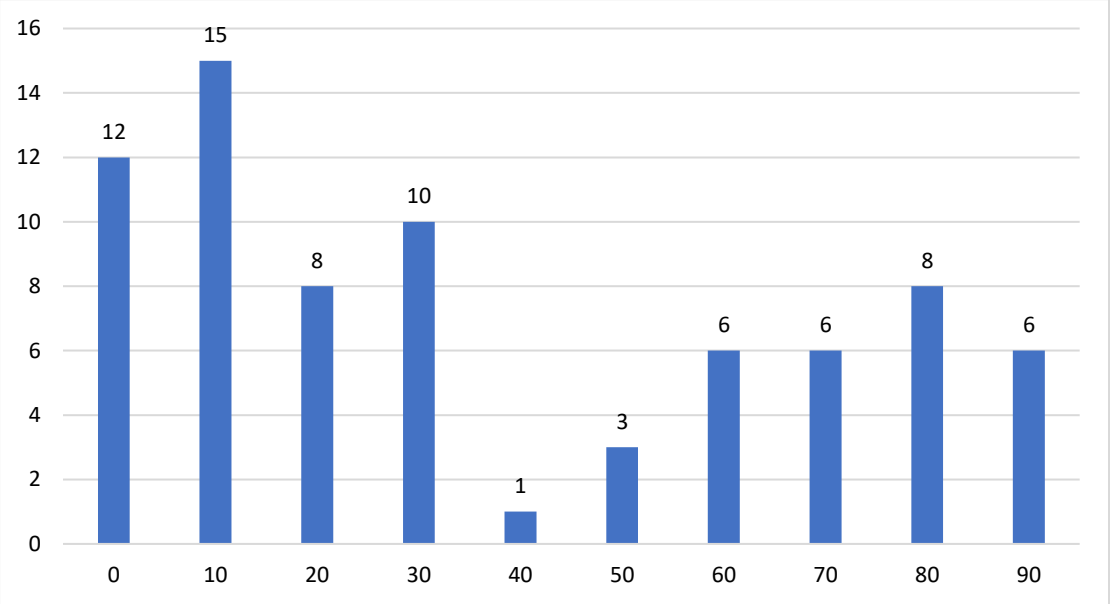


**RESTRICTION OF ABDUCTION OF THE STUDY PARITICPANTS:**

| RESTRICTION OF ABDUCTION (DEGREES) | FREQUENCY | PERCENT |
|---|---|---|
| 0 | 12 | 16.0 |
| 10 | 15 | 20.0 |
| 20 | 8 | 10.7 |
| 30 | 10 | 13.3 |
| 40 | 1 | 1.3 |
| 50 | 3 | 4.0 |
| 60 | 6 | 8.0 |
| 70 | 6 | 8.0 |
| 80 | 8 | 10.7 |
| 90 | 6 | 8.0 |

The range of restriction of abduction of the study participants is seen in the following order: 10°> 0° > 30° > 20° and 80° > 60°, 70° and 90°, > 50° > 40°.

**RESTRICTION OF ABDUCTION OF THE STUDY PARITICPANTS:**

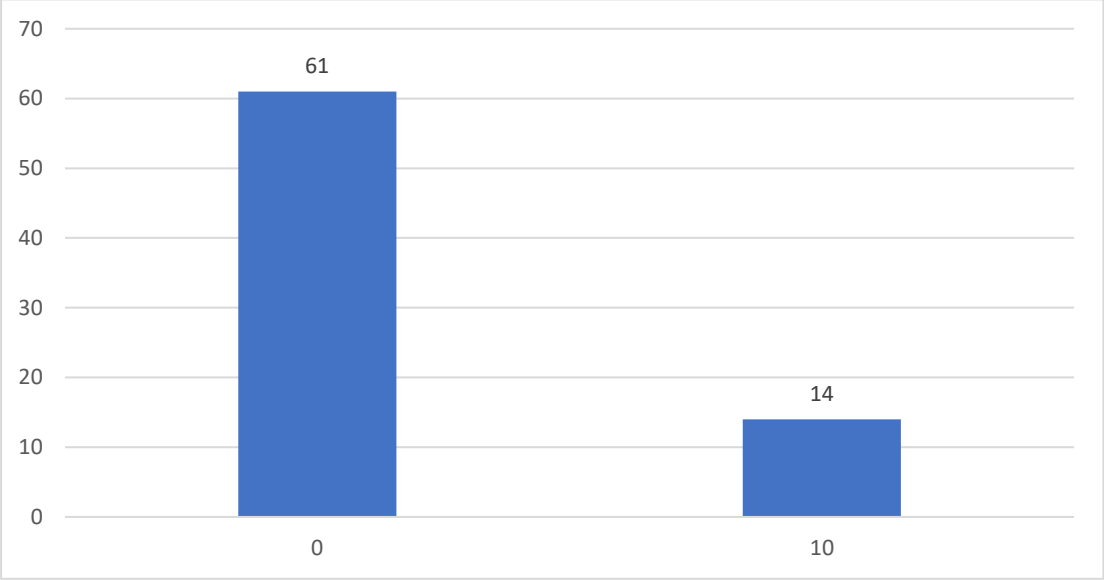


**RESTRICTION OF ADDUCTION OF THE STUDY PARITICPANTS:**

| RESTRICTION OF ADDUCTION (DEGREES) | FREQUENCY | PERCENT |
|---|---|---|
| 0 | 61 | 81.3 |
| 10 | 14 | 18.7 |

The range of restriction of adduction of the study participants is seen in the following order: 0°> 10°.

**RESTRICTION OF ADDUCTION OF THE STUDY PARITICPANTS:**

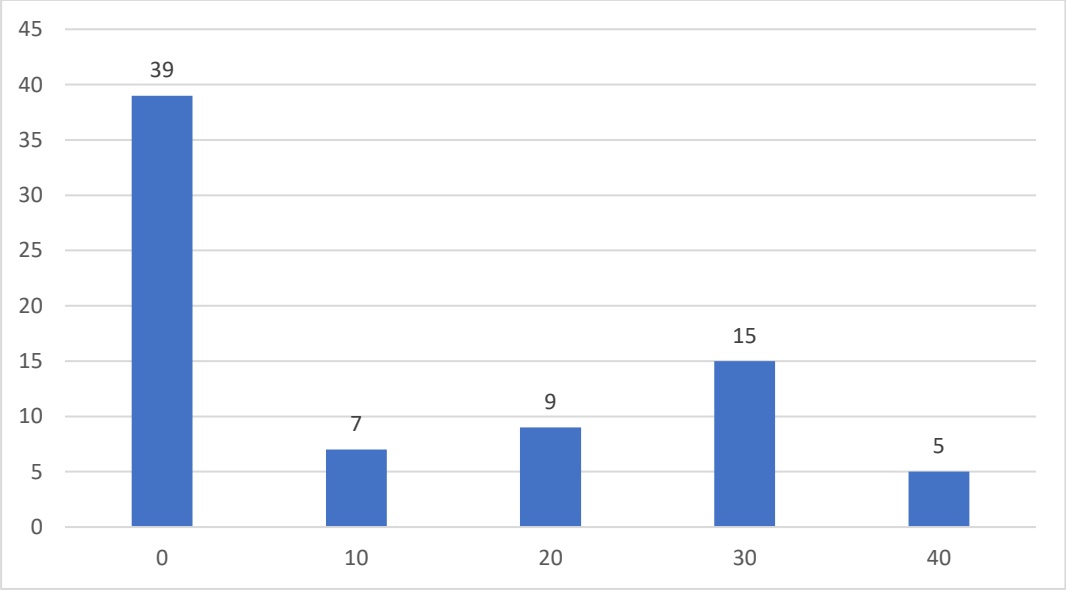


**RESTRICTION OF INTERNAL ROTATION OF THE STUDY PARITICPANTS:**

| RESTRICTION OF INTERNAL ROTATION (DEGREES) | FREQUENCY | PERCENT |
|---|---|---|
| 0 | 39 | 52.0 |
| 10 | 7 | 9.3 |
| 20 | 9 | 12.0 |
| 30 | 15 | 20.0 |
| 40 | 5 | 6.7 |

The range of restriction of internal rotation of the study participants is seen in the following order: 0°> 30° > 20° > 10° > 40°.

**RESTRICTION OF INTERNAL ROTATION OF THE STUDY PARITICPANTS:**

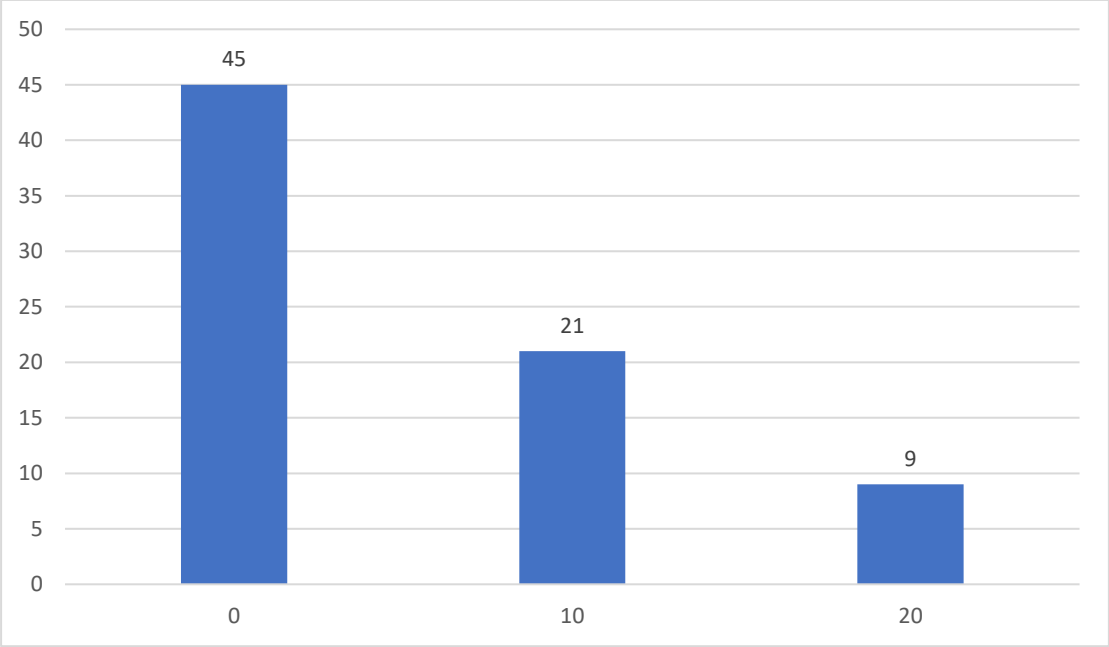


**RESTRICTION OF EXTERNAL ROTATION OF THE STUDY PARITICPANTS:**

| RESTRICTION OF EXTERNAL ROTATION (DEGREES) | FREQUENCY | PERCENT |
|---|---|---|
| 0 | 45 | 60.0 |
| 10 | 21 | 28.0 |
| 20 | 9 | 12.0 |

The range of restriction of external rotation of the study participants is seen in the following order: 0°> 10° > 20°.

**RESTRICTION OF EXTERNAL ROTATION OF THE STUDY PARITICPANTS:**

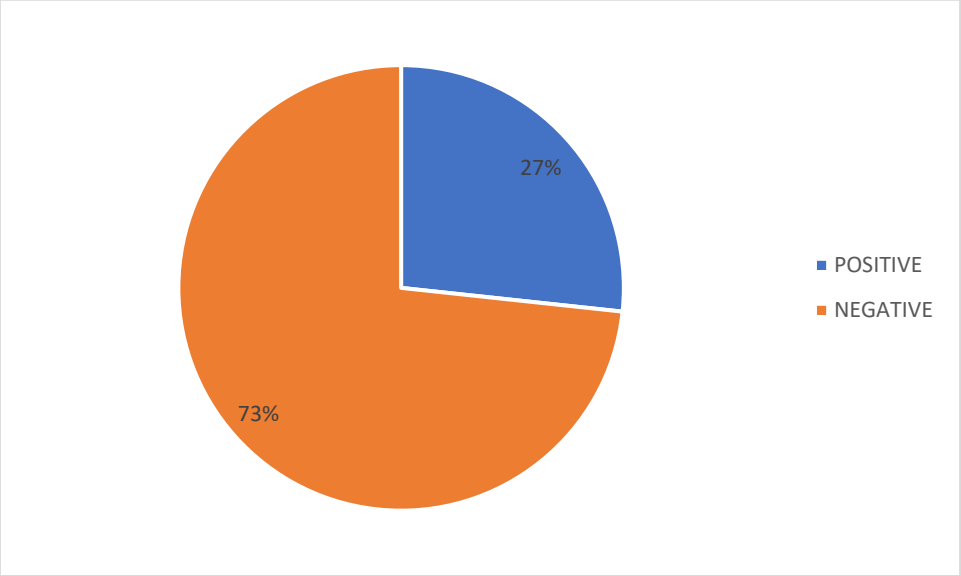


**JOBE (EMPTY CAN TEST) OF THE STUDY PARITICPANTS:**

| JOBE (EMPTY CAN TEST) | FREQUENCY | PERCENT |
|---|---|---|
| **POSITIVE** | 20 | 26.7 |
| **NEGATIVE** | 55 | 73.3 |

73.3% of the study participants were found to be negative on jobe test and only 26.7% of the study participants were found to be positive on jobe test.

**JOBE (EMPTY CAN TEST) OF THE STUDY PARITICPANTS:**

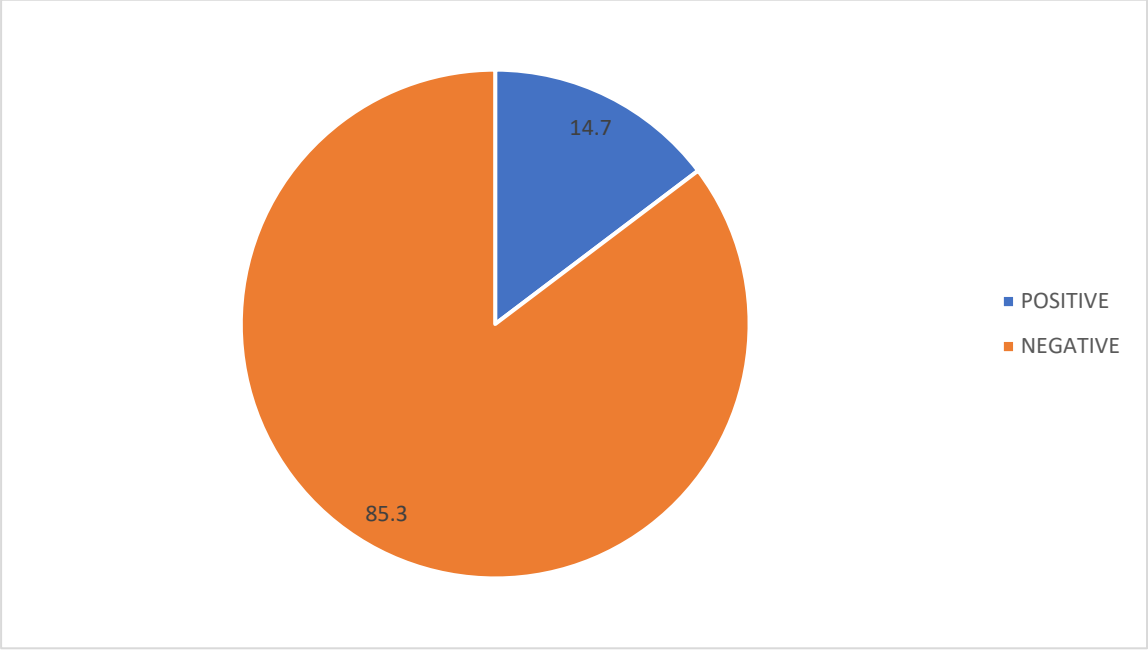


## DROP ARM TEST OF THE STUDY PARITICPANTS:

| DROP ARM TEST | FREQUENCY | PERCENT |
|---|---|---|
| **POSITIVE** | 11 | 14.7 |
| **NEGATIVE** | 64 | 85.3 |

85.3% of the study participants were found to be negative on drop arm test and only 14.7% of the study participants were found to be positive on drop arm test.

## DROP ARM TEST OF THE STUDY PARITICPANTS:

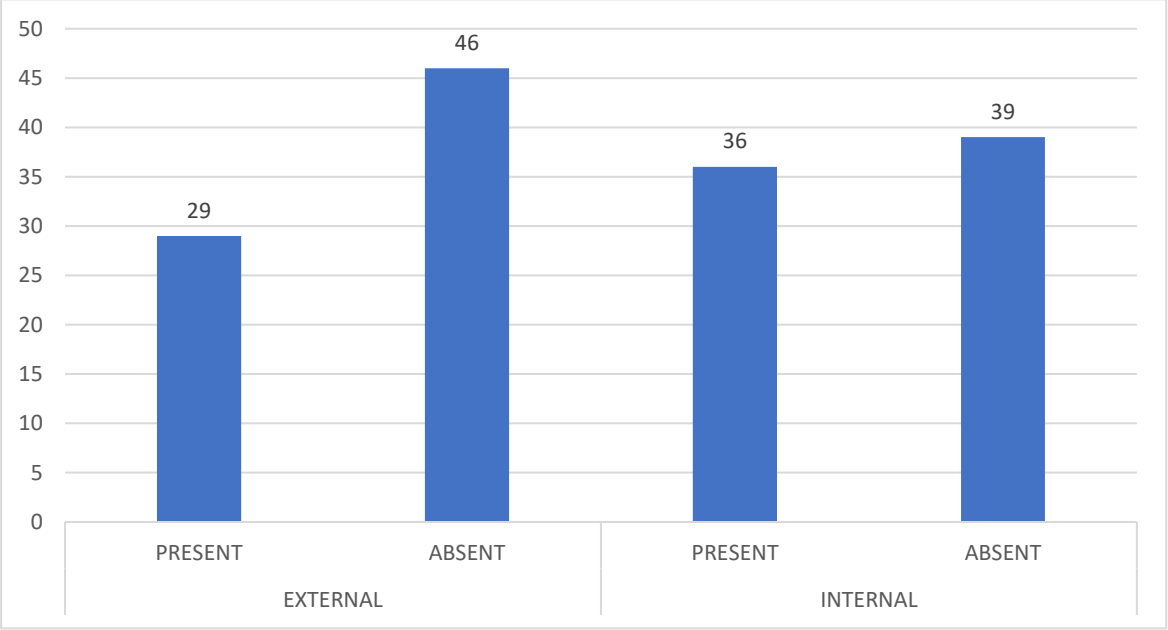


## ROTATION LAGS OF THE STUDY PARITICPANTS:

| ROTATION LAG | | FREQUENCY | PERCENT |
|---|---|---|---|
| **EXTERNAL** | **PRESENT** | 29 | 38.7 |
| | **ABSENT** | 46 | 61.3 |
| **INTERNAL** | **PRESENT** | 36 | 48.0 |
| | **ABSENT** | 39 | 52.0 |

38.7% of the study participants were found to have external rotation lag and 48.0% of the study participants were found to have internal rotation lag.

## ROTATION LAGS OF THE STUDY PARITICPANTS:

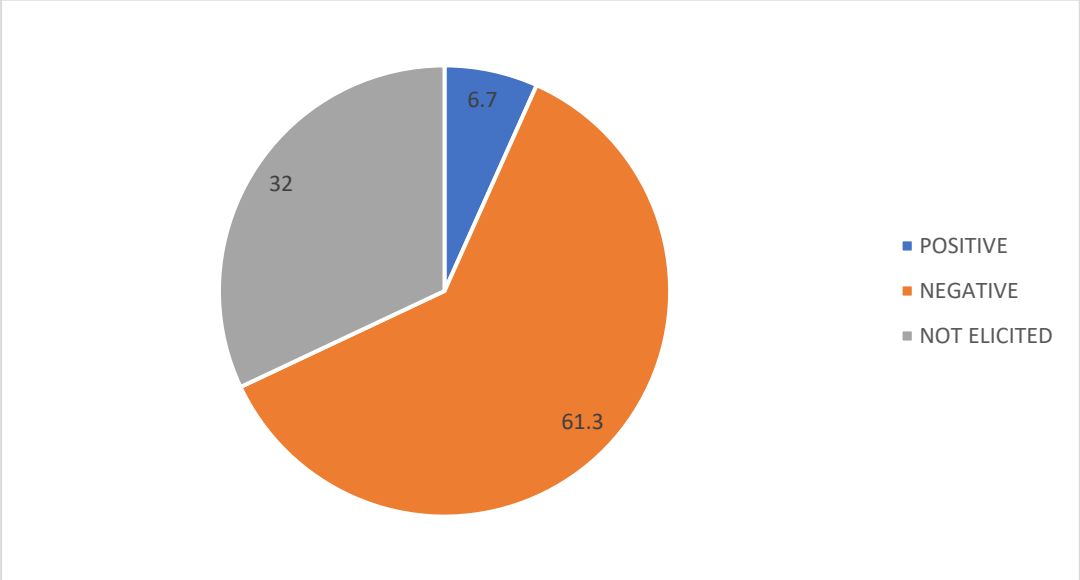


**GERBER'S LIFT OFF TEST OF THE STUDY PARITICPANTS:**

| GERBER'S LIFT OFF TEST | FREQUENCY | PERCENT |
|---|---|---|
| **POSITIVE** | 5 | 6.7 |
| **NEGATIVE** | 46 | 61.3 |
| **NOT ELICITED** | 24 | 32.0 |

61.3% of the study participants were found to be negative on gerber's lift off test and only 6.7% of the study participants were found to be positive on gerber's lift off test. Gerber's lift off test could not be elicited in 32% of the study participants.

**GERBER'S LIFT OFF TEST OF THE STUDY PARITICPANTS:**

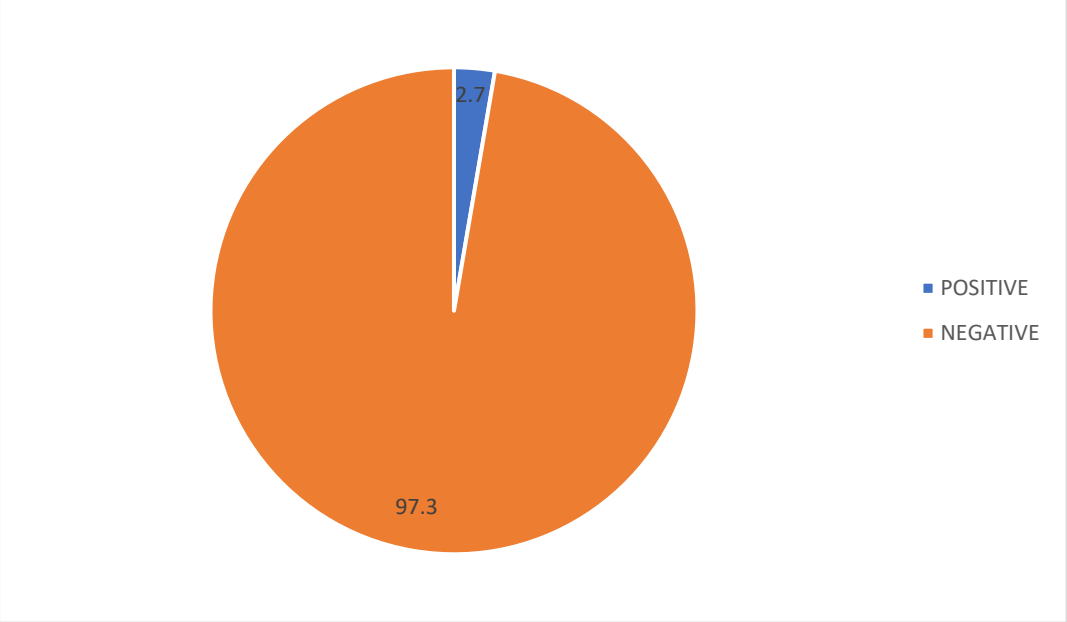


**PALPATION OF BICEPS TENDON TEST OF THE STUDY PARITICPANTS:**

| PALPATION OF BICEPS TENDON TEST | FREQUENCY | PERCENT |
|---|---|---|
| **POSITIVE** | 2 | 2.7 |
| **NEGATIVE** | 73 | 97.3 |

97.3% of the study participants were found to be negative on biceps tendon palpation test and only 2.7% of the study participants were found to be positive on biceps tendon palpation test.

**PALPATION OF BICEPS TENDON TEST OF THE STUDY PARITICPANTS:**

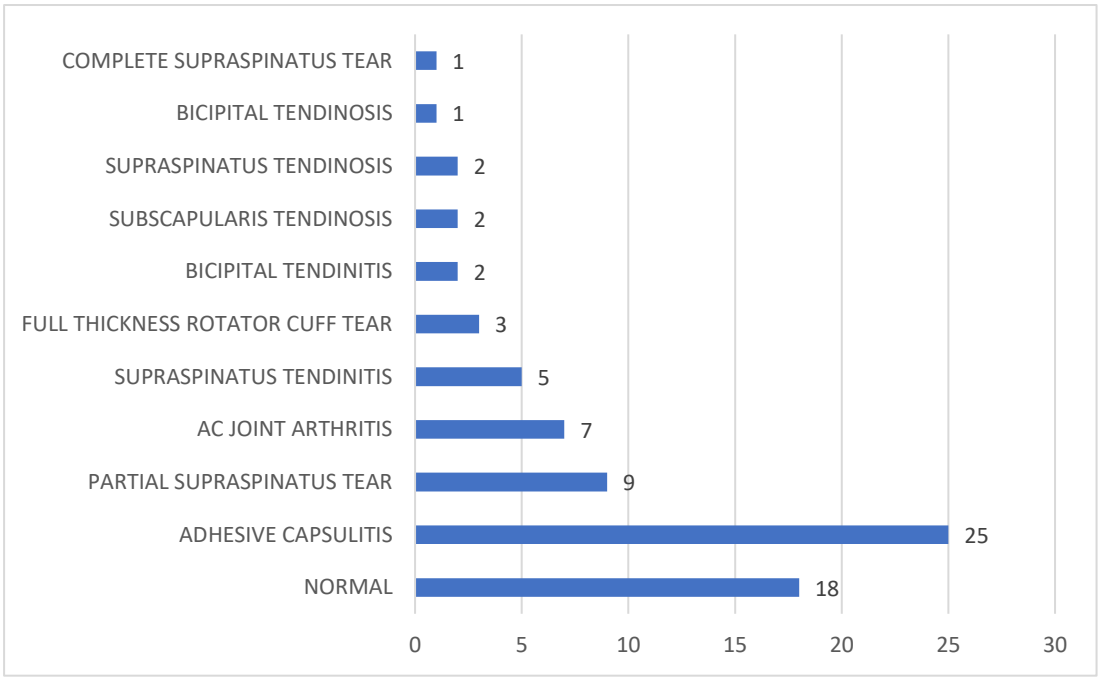


**DIAGNOSIS BASED ON USG SHOULDER:**

| DIAGNOSIS BASED ON USG SHOULDER | FREQUENCY | PERCENT |
|---|---|---|
| **NORMAL** | 18 | 24.0 |
| **ADHESIVE CAPSULITIS** | 25 | 33.3 |
| **PARTIAL SUPRASPINATUS TEAR** | 9 | 12.0 |
| **AC JOINT ARTHRITIS** | 7 | 9.3 |
| **SUPRASPINATUS TENDINITIS** | 5 | 6.7 |
| **FULL THICKNESS ROTATOR CUFF TEAR** | 3 | 4.0 |
| **BICIPITAL TENDINITIS** | 2 | 2.7 |
| **SUBSCAPULARIS TENDINOSIS** | 2 | 2.7 |
| **SUPRASPINATUS TENDINOSIS** | 2 | 2.7 |
| **BICIPITAL TENDINOSIS** | 1 | 1.3 |
| **COMPLETE SUPRASPINATUS TEAR** | 1 | 1.3 |

24% of the study participants were found to be normal on USG shoulder. 33.3% of the study participants were diagnosed with adhesive capsulitis followed by 12% with partial supraspinatus tear on USG shoulder. 9.3%, 6.7% and 4% of the study participants were diagnosed with AC joint arthritis, supraspinatus tendinitis and full thickness rotator cuff tear on USG shoulder respectively. 2.7% each of the study participants were diagnosed with Bicipital tendinitis, subscapularis tendinosis and supraspinatus tendinosis respectively. 1.3% each of the study participants were diagnosed with bicipital tendinosis and complete supraspinatus tear respectively.

## DIAGNOSIS BASED ON USG SHOULDER:

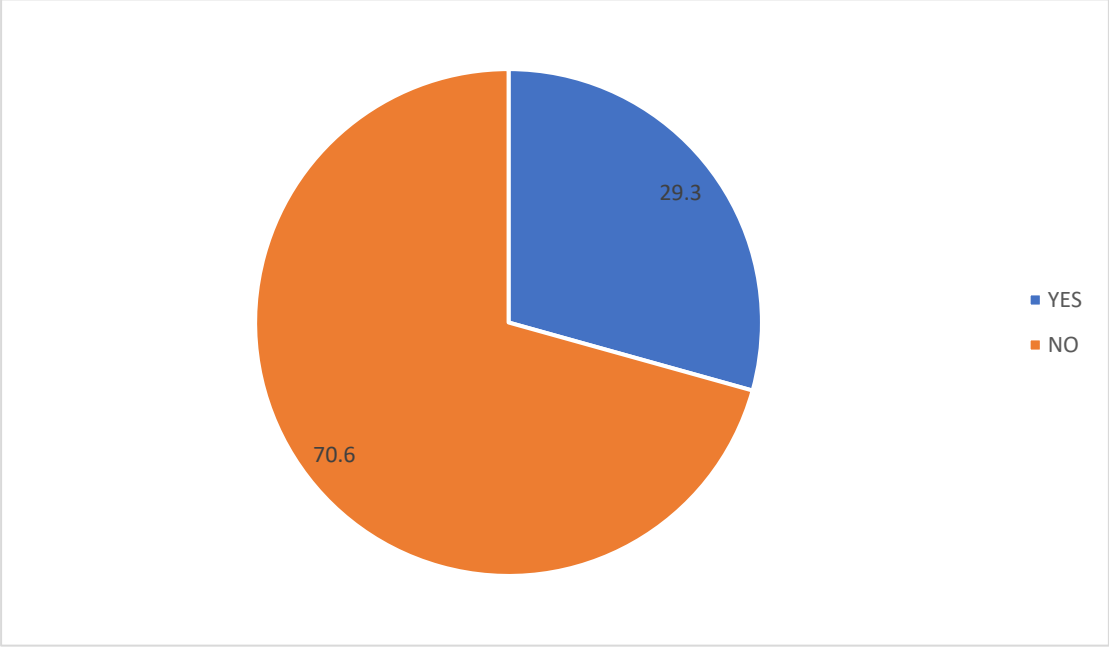


**PREVALANCE OF ROTATOR CUFF DISEASE:**

| ROTATOR CUFF DISEASE | FREQUENCY | PERCENT |
|---|---|---|
| **YES** | 22 | 29.33 |
| **NO** | 53 | 70.66 |

29.33% of the study participants with diabetes was found to have rotator cuff disease during clinical examination. 70.66% of the study participants were not found to have rotator cuff disease

**PREVALANCE OF ROTATOR CUFF DISEASE:**



# DISCUSSION

Numerous studies show that Rotator Cuff diseases are more prevalent in Diabetics, and such patients suffer from pain and/or limitation of Range of Movements at the affected shoulder.
This is a well known fact that Diabetics have increased risk of developing shoulder diseases, such as Adhesive Capsulitis or Rotator Cuff Tendinopathies. In addition to this, such patients tend to show a higher incidence of Re-tears and limitations of movements at shoulder, even after undergoing surgical intervention for the same. Thus, it can be linked to the suboptimal quality of the repaired tissues.

In comparison with healthy individuals, patients with Diabetes have shown a generalised restriction in Range of Movements at the shoulder joint. The exact etiology remains unclear, though there are several adverse biochemical changes which are associated with Rotator Cuff Diseases in Diabetics. Increased glycosylation of collagen proteins which leads to altered synthesis of collagen, along with forming Advanced-Glycation-End-products (A.G.E.s), that deposits, and over a period of time accumulates in the tissues of the shoulder joint, such as in the joint capsule or the tendinous insertions in its proximity. These AGEs show a detrimental effect in a number of cellular and extra cellular activities of tissues, especially those of the shoulder joint and its proximity.

The result of this study indicates that diabetics are more prone to developing Rotator Cuff Diseases than healthy people. A high Prevalence rate of 29.33 % of Rotator Cuff Diseases in Diabetics, as observed in our study, supports this inference.

The Degenerative changes range from partial/ complete thickness tendon tears at rotator cuff to

increased thickness of these tendon, because of abnormal storage of collagen over tissues.
Yamaguchi et. Al. in a Two year Eight Month follow up study, state the clinical relevance of our observation. According to this study, pain along with limitation of functionality may develop in massive percentage (50percent) population having asymptomatic tear at base-line.(34)

Observation of bursal and peri-tendinous effusions, which are an early expression of reactive inflammation to insignificant tendon tears, following unrecognised trivial traumatic events, besides tendinopathic degeneration, was also observed among some patients in our study.
One observation made in our study is that there is increased prevalence of Rotator Cuff pathologies of the dominant hand of the patients, which confirms the theory that significant overuse plays a pathogenetic role, though our study could not differentiate overuse injuries from intrinsic injuries of Rotator-cuff seen in people having diabetes, as we faced difficulties in getting appropriate information about work related pathologies.

Also, our study could not find a significant correlation of Rotator Cuff Diseases with duration of Diabetes Mellitus, as we faced difficulties establishing age of diabetes on-set in the patients.
Moreover, these patients could have mild Non-Insulin-Dependent-Diabetes-Mellitus (N.I.D.D.M.) or Glucose Intolerance for some time before they were clinically established as Diabetics.

In Diabetic patient having no history of any significant trauma, it is likely that the Rotator cuff Disease is primarily due to intrinsic change of degeneration, related to ageing, mechanical impingement, or vascular abnormalities. Age related degeneration of rotator cuff tendons share similar biochemical mechanisms as that of tendinous degeneration seen in diabetics.

Non enzymatic glycosylation of collagen which gives rise to Advanced Glycation End products (AGEs), is considered the primary causative biochemical change.

The voluntary breakdown of glucose and its metabolites such as triose phosphates, glyoxal as well as methylglyoxal, alongside free amino acids such as lysine, xylysine and arginine gives rise to a chemical bond of amino acid with sugar, which is called as Amadori compound. The AGEs are formed by the chemical reactions that follow in this chain.

Advanced-Glycation-End-products exhibit debilitating effect on the physical as well as chemical properties of the proteins. As a result, the intermolecular collagen cross links are enhanced, which reduces the collagen solubility. Thus, the collagen undergoes irreversible changes, making it more tough, stiff and weak. As a result, it looses its elastic strength and becomes prone to degenerative pathologies.

The activity if certain receptors (R.A.G.E.) that are found over membrane of chondrocytes, tenocytes and fibroblasts, is another way the AGEs may have harmful consequences.(1) RAGE's ligand interaction sets off cell specific signalling that increases the production of reactive oxygen species, thus activates the transcription of Nuclear Factor Kappa Beta.(1)

As a result of continuous up regulation of proinflammatory mediators, adhesion molecules as well as A.G.E. crosslink formation in collagen fibres, a defective cell phenotype is made.(1) V.E.G.F. (Vascular Endothelial Growth Factor), cytokines as well as R.A.G.E. is over expressed in Diabetics, which may help to explain why people with diabetes have more lesions and inflammatory reactions of the Rotator Cuff.(1)

Along with the A.G.E. mediated pathogenetic pathway, microvascular changes of diabetes can also lead to tissue hypoxia and Oxygen Free Radical generation, which in turn results in an excess of growth factors and cytokines, which lead to further damage to the tissues if the Rotator Cuff.

It is important to recognise some of this study's shortcomings. The evaluation of pain and functional limitations was based on self reports, which is rather arbitrary. This study, which primarily focussed on tendon evaluation, did not consider spurs or other bone abnormalities. Therefore, we are unable to say if diabetes was associated with higher prevalence of pathologies which can be surgically removed avoiding impingement-type injuries. More-over, our study only evaluates the prevalence of Rotator cuff Diseases in people having Diabetes-Mellitus, it does not take into consideration the age related degenerative changes in the rotator cuff in healthy non diabetic population. Additionally, the examiner wasn't blinded to whether the subjects have diabetes or not. Lastly, it should be acknowledged that our study does not depict the modalities of intervention, conservative or surgical, for the various Rotator Cuff Diseases prevalent in patients with Diabetes Mellitus.

# CONCLUSION

The results of our study depict worsening of degeneration of the Rotator Cuff Tendons in patients who suffer from Diabetes Mellitus. In addition to clinical screening, USG imaging is a helpful tool for identifying at risk individuals who may develop shoulder disorders in the symptomatic stage.

Careful metabolic control using appropriate food habits and anti-diabetic medications should be advised in these people, who represent an increasing section of the older population. The development of tear size should also be tracked over time, and appropriately managed by either surgery or medicines along with physiotherapy.

# SUMMARY

A Hospital based cross sectional study was conducted among 75 patients who presented to the orthopaedics OPD of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre and Charitable Hospital, Belagavi, with Shoulder pain and limitation of ROM at shoulder joint and were a known case of Diabetes Mellitus type II, to find out the Prevalence of Rotator Cuff Diseases in Type II Diabetes Mellitus. The study was conducted for a period of one year from 1st January 2021 to 31st December 2021.

The following observation were made in the study -

1. The mean age of the study participants was found to be 54.12 $\pm$ 10.465 years.
2. 65.3% of the study participants were males with females contributing to 34.7% of the study participants.
3. 2% of the study participants were affected on the right shoulder and 28% of the study participants were affected on the left shoulder
4. The range of restriction of flexion of the study participants is seen in the following order: 0°> 20°, and 90° > 10°, and 30° > 80° > 70° > 40° > 60° > 50°.
5. The range of restriction of extension of the study participants is seen in the following order: 0°> 10° > 20°.
6. The range of restriction of abduction of the study participants is seen in the following order: 10°> 0° > 30° > 20° , 80° > 60°, 70° and 90° > 50° > 40°.
7. The range of restriction of adduction of the study participants is seen in the following order: 0°> 10°.
8. The range of restriction of internal rotation of the study participants is seen in the following order: 0°> 30° > 20° > 10° > 40°.
9. The range of restriction of external rotation of the study participants is seen in the following order: 0°> 10° > 20°.
10. 73.3% of the study participants were found to be negative on jobe test and only 26.7% of the study participants were found to be positive on jobe test.
11. 85.3% of the study participants were found to be negative on drop arm test and only 14.7% of

the study participants were found to be positive on drop arm test.

12. 38.7% of the study participants were found to have external rotation lag and 48.0% of the study participants were found to have internal rotation lag.
13. 61.3% of the study participants were found to be negative on gerber's lift off test and only 6.7% of the study participants were found to be positive on gerber's lift off test. Gerber's lift off test could not be elicited in 32% of the study participants.
14. 97.3% of the study participants were found to be negative on biceps tendon palpation test and only 2.7% of the study participants were found to be positive on biceps tendon palpation test.
15. 24% of the study participants were found to be normal on USG shoulder. 33.3% of the study participants were diagnosed with adhesive capsulitis followed by 12% with partial supraspinatus tear on USG shoulder. 9.3%, 6.7% and 4% of the study participants were diagnosed with AC joint arthritis, supraspinatus tendinitis and full thickness rotator cuff tear on USG shoulder respectively. 2.7% each of the study participants were diagnosed with Bicipital tendinitis, subscapularis tendinosis and supraspinatus tendinosis respectively. 1.3% each of the study participants were diagnosed with bicipital tendinosis and complete supraspinatus tear respectively.
16. 29.33% of the study participants with diabetes was found to have rotator cuff disease upon clinical examination and Ultrasonography of the affected shoulder joint

# BIBLIOGRAPHY

**1.** Michele Abate, Cosima Schiavone, Vincenzo Salini - Sonographic evaluation of the shoulder in asymptomatic elderly subjects with diabetes.Abate et al. BMC Musculoskeletal Disorders 2010, 11:278 http://www.biomedcentral.com/1471-2474/11/278

**2.** Arlan L. Rosenbloom, MD, and Janet H. Silverstein, MD - Connective tissue and joint disease indiabetes mellitus

**3.** Fukuda H, Hamada K, Yamanaka K. Pathology and pathogenesis of bursal side rotator cuff tearsviewed from en bloc histologic sections. Clin Orthop Relat Res. 1990;254:75–80.

**4.** Perttu ETArkkila, Ilkka M Kantola, Jorma SA Viikari, Tapani Ronnemaa - Shoulder capsulitis intype I and II diabetic patients : association with diabetic complications and related diseases.

**5.** Niluʹfer Balci, Mustafa Kemal Balci, Serdar Tuʹzuʹner - Shoulder Adhesive Capsulitis and Shoulder Range of Motion in Type II Diabetes Mellitus: Association withDiabetic Complications. Journal of Diabetes and Its Complications 1999; 13:135–140 - 1999 Elsevier Science Inc.

**6.** Antonia Cole, Tiffany K. Gill, E. Michael Shanahan, Patrick Phillips, Anne W. Taylor, and Catherine L. Hill - Is Diabetes Associated with Shoulder Pain or Stiffness? Results from a Population Based Study. J Rheumatol 2009;36;371-377 http://www.jrheum.org/content/36/2/371.

**7.** B. Pal , J. Anderson , W. C. Dick and I. D. Griffiths Departments of Rheumatology and Medicine, Royal Victoria Infirmary,Newcastle upon Tyne NE1 4LP, UK - Limitation of Joint mobility and Shoulder Capsulitis in Insulin and Non- Insulin-Dependent Diabetes Mellitus. British Journal of Rheumatology 1986;25:147-151

**8.** Arlan L. Rosenbloom, MD, and Janet H. Silverstein, MD - Connective Tissue and Joint Disease in Diabetes Mellitus. Chronic Complications of Diabetes 0889-8529/96

**9.** A Aydeniz, S Gurney and E Guney - Which Musculoskeletal Complications Are Most Frequently Seen in Type 2 Diabetes Mellitus? The Journal of International Medical Research 2008; 36: 505 –511.

**10.** Worland RL, Lee D, Orozco CG, SozaRex F, Keenan J. - Correlation of age, acromial morphology, and rotator cuff tear pathology diagnosed by ultrasound in asymptomatic patients.JSouth Orthop Assoc. 2003;12(1):23–26. [PubMed] []Google Scholar

**11.** Jeroen DeGroot - The AGE of the matrix: chemistry, consequence and cure. Current Opinion in Pharmacology 2004, 4:301–305 www.sciencedirect.com.

**12.** Karen M. Reiser - Non Enzymatic Glycation of Collagen in Ageing and Diabetes .(43158C).Dept. of Medicine, School of Medicine, University of California, Davis

**13.** Udayan Dutta, Menashi A. Cohenford , Madhumita Guha , Joel A. Dain - Non-enzymatic interactions of glyoxylate with lysine, arginine, and glucosamine: A study of advanced non- enzymatic glycation like compounds. Bio organic Chemistry 35 (2007) 11–24 www.elsevier.com/locate/bioorg .

**14.** Diabetes and shoulder disorders - Journal of Diabetes Investigation JDiabetes Investig Vol. No. 2016 .

**15.** Simon J. Thomas, Claire McDougall, Iain D. M. Brown, Marie-Claire Jaberoo, Adam Stearns, Raihan Ashraf, Miles Fisher, and Ian G. Kelly, Glasgow, Scotland - Prevalence of symptoms and signs of shoulder problems in people with diabetes mellitus .

**16.** Shih-Wei Huang, Wei-Te Wang, Lin-Chuan Chou, Tsan-Hon Liou, Yi-Wen Chen, Hui-Wen Lin- Diabetic Mellitus Increases the Risk of Rotator Cuff Tear Repair Surgery: A Population-Based Cohort Study. doi: 10.1016/j.jdiacomp.2016.07.015 Journal of Diabetes and Its Complications

**17.** Lorna Fisher, A. Kurtz Andm. Shipley - Association Between Cheiroarthropathy and Frozen Shoulder in Patients with Insulin Dependent Diabetes Mellitus. British Journal of Rheumatology 1986;25!l41-146 .

**18.** Gaillard F, Roberts D, Patel M, et al. Ultrasound of the Shoulder,Reference article, Radiopaedia.org (Accessed on 28 Dec 2022), https://doi.org/10.53347/rID-2238

**19.** Tashjian, RZ (2012) Epidemiology, natural history, and indications for treatment of rotator cuff tears. Clin Sports Med 31:589-604. DOI: 10.1016/j.csm.2012.07.001.

**20.** Oliva, F, Piccirilli, E, Bossa, M, Via, AG, Colombo, A, Chillemi, C, Gasparre, G, Pellicciari, L, Franceschetti, E, Rugiero, C, Scialdoni, A, Vittadini, F, Brancaccio, P, Creta, D, Buono, AD, Garofalo, R, Franceschi, F, Frizziero, A, Mahmoud, A, Merolla, G, Nicoletti, S, Spoliti, M, Osti, L, Padulo, J, Portinaro, N, Tajana, G, Castagna, A, Foti, C, Masiero, S, Porcellini, G, Tarantino, U & Maffulli, N (2015) I.S.Mu.L.T - Rotator Cuff Tears Guidelines. Muscles Ligaments Tendons J 5:227-63. DOI: 10.11138/mltj/2015.5.4.227.

**21.** Tempelhof, S, Rupp, S & Seil, R (1999) Age-related prevalence of rotator cuff tears in asymptomatic shoulders. J Shoulder Elbow Surg 8:296-9.

**22.** Yamamoto, A, Takagishi, K, Osawa, T, Yanagawa, T, Nakajima, D, Shitara, H & Kobayashi, T (2010) Prevalence and risk factors of a rotator cuff tear in the general population. J Shoulder Elbow Surg 19:116-20. DOI: 10.1016/j.jse.2009.04.006.

**23.** Via, AG, De Cupis, M, Spoliti, M & Oliva, F (2013) Clinical and biological aspects of rotator cuff tears. Muscles Ligaments Tendons J 3:70-9. DOI: 10.11138/mltj/2013.3.2.070.

**24.** Gartsman, GM, Khan, M & Hammerman, SM (1998) Arthroscopic repair of full-thickness tears of the rotator cuff. J Bone Joint Surg Am 80:832-40.

**25.** Mathew et al., A combination approach using manual therapy and exercise in the treatment of shoulder impingement syndrome. January 2013 Indian Journal of Physiotherapy and Occupational Therapy - An International Journal 7

**26.** Longo et al., Conservative versus surgical management for patients with rotator cuff tears: a systematic review and META-analysis. doi: 10.1186/s12891-020-03872-4. BMC Musculoskeletal Disorders.

**27.** Lin, TT, Lin, CH, Chang, CL, Chi, CH, Chang, ST & Sheu, WH (2015) The Effect of Diabetes, Hyperlipidemia, and Statins on the Development of Rotator Cuff Disease: A Nationwide, 11-Year, Longitudinal, Population-Based Follow-up Study. Am J Sports Med 43:2126-32. DOI: 10.1177/0363546515588173.

**28.** Zreik, NH, Malik, RA & Charalambous, CP (2016) Adhesive capsulitis of the shoulder and diabetes: a meta-analysis of prevalence. Muscles Ligaments Tendons J 6:26-34. DOI: 10.11138/ mltj/2016.6.1.026.

**29.** Ranger, TA, Wong, AM, Cook, JL & Gaida, JE (2015) Is there an association between tendinopathy and diabetes mellitus? A systematic review with meta-analysis. Br J Sports Med. DOI: 10.1136/bjsports-2015-094735.

**30.** Zakaria, MH, Davis, WA & Davis, TM (2014) Incidence and predictors of hospitalization for tendon rupture in type 2 diabetes: the Fremantle diabetes study. Diabet Med 31:425-30. DOI: 10.1111/dme.12344.

**31.** Varacallo M, El Bitar Y, Mair SD. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Sep 4, 2022. Rotator Cuff Syndrome. - Anatomy, Rotator cuff. Pubmed

**32.** Rotator Cuff Tendonitis - Matthew Varacallo , Youssef El Bitar , Scott D. MairIn: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Sep 4.Affiliations expand PMID: 30335303 Bookshelf ID: NBK532270

**33.** Anatomy, Back, Scapula - Paul T. Cowan, Andrew Mudreac, Matthew Varacallo In:StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan.2022 Aug 8.Affiliations expand PMID: 30285370 Bookshelf ID: NBK531475

**34.** Yamaguchi et al., The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders PMID: 16882890 DOI: 10.2106/ JBJS.E.00835

**35.** Colvin, AC, Egorova, N, Harrison, AK, Moskowitz, A & Flatow, EL (2012) National trends in rotator cuff repair. J Bone Joint Surg Am 94:227-33. DOI: 10.2106/JBJS.J.00739.

**36.** Bedi, A, Fox, AJ, Harris, PE, Deng, XH, Ying, L, Warren, RF & Rodeo, SA (2010) Diabetes mellitus impairs tendon-bone healing after rotator cuff repair. J Shoulder Elbow Surg 19:978-88.DOI: 10.1016/j.jse.2009.11.045.

**37.** Franke, S, Sommer, M, Ruster, C, Bondeva, T, Marticke, J, Hofmann, G, Hein, G & Wolf, G (2009) Advanced glycation end products induce cell cycle arrest and proinflammatory changes in osteoarthritic fibroblast-like synovial cells. Arthritis Res Ther 11:R136. DOI: 10.1186/ar2807.

**38.** Handa, A, Gotoh, M, Hamada, K, Yanagisawa, K, Yamazaki, H, Nakamura, M, Ueyama, Y, Mochida, J & 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. J Orthop Res 21:1138-44. DOI: 10.1016/S0736-0266(03)00102-5.

**39.** Schibany, N, Zehetgruber, H, Kainberger, F, Wurnig, C, Ba-Ssalamah, A, Herneth, AM, Lang, T, Gruber, D & Breitenseher, MJ (2004) Rotator cuff tears in asymptomatic individuals: a clinical and ultrasonographic screening study. Eur J Radiol 51:263-8. DOI: 10.1016/S0720-048X(03)00159-1.

**40.** Dutta, U, Cohenford, MA, Guha, M & Dain, JA (2007) Non-enzymatic interactions of glyoxylate with lysine, arginine, and glucosamine: a study of advanced non-enzymatic glycationlike compounds. Bioorg Chem 35:11-24. DOI: 10.1016/j.bioorg.2006.07.005.

**41.** Smith LL, Burnet SP, McNeil JD: Musculoskeletal manifestations of diabetes mellitus. Br JSports Med 2003, 37(1):30-35.

**42.** Valencia JV, Weldon SC, Quinn D, Kiers GH, DeGroot J, TeKoppele JM, Hughes TE: Advanced glycation end product ligands for the receptor for advanced glycation end products: biochemical characterization and formation kinetics. Anal Biochem 2004, 324(1):68-78.

**43.** Steenvoorden MM, Toes RE, Ronday HK, Huizinga TW, DeGroot J: RAGE activation induces invasiveness of RA fibroblast-like synoviocytes in vitro. Clin Exp Rheumatol 2007, 25(5):740-742.

**ANNEXURE – I PROFORMA**

## PROFORMA

"PREVALENCE OF ROTATOR CUFF DISEASES IN TYPE II DIABETES MELLITUS – A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY"

PATIENT NO. IP NO.

AGE: SEX:

ADDRESS:

CHIEF COMPLAINTS:

HISTORY AND DEMOGRAPHY

AGE:

SEX:

OCCUPATION:

WEIGHT:

HEIGHT:

DIABETES MELLITUS STATUS:

**ANNEXURE – I PROFORMA**

TREATMENT HISTORY FOR DIABETES MELLITUS:

PAST HISTORY:

HISTORY OF SHOULDER TRAUMA:

HISTORY OF PRIOR SHOULDER SURGERY:

HISTORY OF SHOULDER PAIN RELATED TREATMENT:

HISTORY OF SYSTEMIC DISEASES:

PERSONAL HISTORY:

SLEEP:

BOWEL:

BLADDER:

APPETITE:

ADDICTIONS:

CLINICAL EVALUATION

1.) AFFECTED SHOULDER:

| RANGE OF MOTION | | ACTIVE | PASSIVE | RESTRICTED |
|---|---|---|---|---|
| FLEXION | | | | |
| EXTENSION | | | | |
| ABDUCTION | | | | |
| ADDUCTION | | | | |
| INTERNAL ROTATION | | | | |
| EXTERNAL ROTATION | | | | |

2.) UNAFFECTED SHOULDER:

| RANGE OF MOTION | ACTIVE | PASSIVE | RESTRICTED |
|---|---|---|---|
| FLEXION | | | |
| EXTENSION | | | |
| ABDUCTION | | | |
| ADDUCTION | | | |
| INTERNAL ROTATION | | | |
| EXTERNAL ROTATION | | | |

SPECIAL TESTS:

| NAME OF THE TEST | RESULT | INFERENCE |
|---|---|---|
| JOBE (EMPTY CAN) TEST | | |
| DROP ARM TEST | | |
| EXTERNAL ROTATION LAG TEST | | |
| INTERNAL ROTATION LAG TEST | | |
| GERBER'S LIFT OFF TEST | | |
| PALPATION OF BICEPS TENDON TEST | | |

RADIOLOGICAL INVESTIGATION:

USG OF THE AFFECTED SHOULDER JOINT:

FINAL DIAGNOSIS:-

# INFORMED CONSENT

**Purpose of the study**

I have been informed by REG NO : BL0120004, Post Graduate in M.S. Orthopaedics under the guidance of Professor and Head, Department of Orthopaedics, J.N. Medical College, KLE University, Belagavi is conducting a study to find out The Prevalence of Rotator Cuff Pathology in patients with Diabetes Mellitus Type II at KLE'S DR.PRABHAKAR CHARITABLE HOSPITAL AND MEDICAL RESEARCH CENTRE,BELAGAVI.

Musculoskeletal disease is one of the most common complication in patients with diabetes, and yet it receives relatively less attention. The severity and risks of musculoskeletal complications might not be well recognized as cardiovascular complications, however the associated ailments certainly inflict both physical and physiological harm on people with diabetes. Among the various musculoskeletal diseases, shoulder pain is one of the most common complaints. In general, it is characterized by pain and limited range of motion of one or both shoulders. Shoulder pain not only causes decreased quality of life, but also leads to disability in daily activities, and might interfere directly or indirectly with control of metabolic process.[16]

There is increased prevalence rate (27.5%) of shoulder disorders in patients with diabetes as compared with the rate of 5% found in general medical patients.[17]

Thus, there is an increased need to study and find out the prevalence of rotator cuff diseases in type II diabetes mellitus so that:-

Optimum metabolic control of blood sugar with proper dietary modifications and/or medications can be achieved so as to restrict further damage to the affected shoulder joint.

And if need be, surgical repair of the rotator cuff injury, for example tendon tears can be achieved as early as possible after the diagnosis is made on clinical as well as radiological grounds.

## STUDY PROCEDURE

Once you have signed the informed consent, necessary personal information and detailed medicalhistory will be taken by the investigator. After this based on the clinical evaluation of pain and restrictions of movements at the shoulder joint, you will be suggested to do USG of the affected shoulder joint to correlate clinically and confirm the rotator cuff pathology at the affected joint, if there is any.

## Potential risks

None.

## Benefits

Thorough clinical evaluation of the affected shoulder joint and confirmation of the pathology byradiological evaluation (USG of Shoulder Joint) will benefit the patient as follows:-

Optimum metabolic control of blood sugar with proper dietary modifications and/or medications canbe achieved so as to restrict further damage to the affected shoulder joint.

And if need be, surgical repair of the rotator cuff injury, for example tendon tears can be achieved as early as possible after thorough clinical and radiological evaluation.

## Financial incentive for participation

You will not receive any payment for taking part in this study.

## Alternatives

Your participation in this study is entirely voluntary. You are free to refuse to participate or withdrawfrom the study at any time. You will still receive standard medical care from the hospital. The investigator holds the right to terminate the study at any time

## Privacy

To protect my privacy, all the collected information will be given a number rather than using my name. Any information collected during the study will remain confidential. My medical files will be reviewed only at the hospital (or study doctor's office) to check the information and verify the resultwithout breaking my confidentiality.

## Authorization to publish results

The information about me will be analyzed together with other study participants. Results of this study will be published and presented to scientific groups for scientific purposes, but Iwill never be individually identified in the presentation of the study results.

## Institutional policy

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact Reg No : BL0120004, Department of Orthopaedics, KLE University's J.N Medical College, Professor and Head, Dept. Of Orthopaedics, KLE University's J.NMedical College, Belagavi

## Voluntary participation

Your participation in the study is voluntary. In case you need any further information regarding yourrights as study participant, you may contact Dr. Roopa M Bellad, Professor of Paediatrics, as Chairman of J. N. Medical College Institutional Ethics Committee on Human Subjects Research, Phone No.0831 2473777 ext-1350 at J. N. Medical College, Belagavi. You are free to stop participation in this study at any time and for any reason

Post-Graduate, Department ofOrthopaedics,
J.N.Medical College,Belagavi-590010.

Guide ,

Professor and Head,
Department of OrthopaedicsJ.N.Medical College, Belagavi-590010.

## CONSENT TO PARTICIPATE IN RESEARCH STUDY:

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicatedthat I have read this entire consent form or it has been read to me, and has been explained to me inmy vernacular language and had all my questions answered. I will be given a copy of this consent form.

I understand that I am participating in the study "Prevalence of Rotator Cuff Diseases in Diabetes Mellitus Type II, A one year hospital based cross sectional study."

I confirm that I have read and understood the information in the patient information sheet. Procedure is explained to me in detail along with information about the advantages and disadvantages of taking part in the study. I have been given the opportunity to discuss all aspects of the trial, to ask questions and hereby consent to participation in the trial outlined above.

I understand that the decision to take part in this study is completely voluntary and I am aware that I can choose to withdraw from the study at any point of time.

I consent to the photographing or recording of the procedure to be performed including appropriate portions of my body, for medical, scientific or educational purposes provided my identity is not revealed in the pictures or by the descriptive texts accompanying them.

I understand that there is no significant risk involved in the test that would be done in thisstudy.

No guarantee or assurance has given by anyone as to the results that may be obtained.

My signature on this form signifies that I have willingly decided to participate after understanding the above information.

Participant's Name/ legally authorized representative _____

Signature_____

Name and signature of witness_____

Name and signature of interviewer_____

Date:

Place:

(If a patient has limited ability to read and write, an impartial witness should be present during

the entire informed consent discussion and patient's legally acceptable representative should sign on thepatient's behalf.) In these instances the patient his/her thumb impression taken in place of signature.

**Patient's legally acceptable representative's statement: NA**

I, as the patient's legally acceptable representative was present during the consenting procedure andunderstand the preceding information describing this study. All of the questions regarding the studyand the patient's participation in it have been answered to my satisfaction. I state that all aspects of the study were clearly presented during the consent procedure. The patient is willing to participate in this study and I sign below on his/her behalf testifying to this effect.

**Name of the patient:**

**Name of representative:**

**Relationship to the patient:**

**Signature of representative:**

Date:

**Impartial witness declaration:**

By signing the consent form I attest that the information was accurately explained to and apparentlyunderstood by the patient and the representative (if applicable) and that the informed consent wasfreely given by the patient.

**Name of impartial witness**:

**Signature:**
Date –

# CLINICAL PHOTOGRAPHS

## PHOTOGRAPH 1

## PHOTOGRAPH 2

**PHOTOGRAPHS 1 AND 2 SHOWING CLINICAL DEMONSTRATION OF DROP ARM TEST FOR TESTING SUPRASPINATUS (LEFT SHOULDER)**

## ANNEXURE – III CLINICAL PHOTOGRAPHS

**PHOTOGRAPH 3: CLINICAL DEMONSTRATION OF EMPTY CAN TEST FOR TESTING SUPRAPSINATUS (LEFT SHOULDER)**

**PHOTOGRAPH 4: CLINICAL DEMONSTRATION OF GERBER'S LIFT OFF TEST FOR TESTING SUBSCAPULARIS (LEFT SHOULDER)**

## ANNEXURE – III CLINICAL PHOTOGRAPHS

**PHOTOGRAPH 5: CLINICAL DEMONSTRATION SHOWING EXTERNAL ROTATION LAG OF LEFT SHOULDER**

**PHOTOGRAPH 6: CLINICAL DEMONSTRATION SHOWING INTERNAL ROTATION LAG OF LEFT SHOULDER**

**PHOTOGRAPH 7**

**PHOTOGRAPH 8**

**PHOTOGRAPH 9**

**PHOTOGRAPHS 7, 8 AND 9 DEPICTS USG EXAMINATION OF THE SHOULDER JOINT IN ANTERIOR, POSTERIOR AND SUPERIOR REGIONS.**

# MASTER CHART

| AGE/ SEX | HOSPITAL REGISTRATION. NUMBER | AFFECTED SHOULDER | RESTRICTION OF FLEXION (DEGREES) | RESTRICTION OF EXTENSION (DEGREES) | RESTRICTION OF ABDUCTION (DEGREES) | RESTRICTION OF ADDUCTION (DEGREES) | RESTRICTION OF INTERNAL ROTATION (DEGREES) | RESTRICTION OF EXTERNAL ROTATION (DEGREES) | JOBE (EMPTY CAN TEST) | DROP ARM TEST | EXTERNAL ROTATION LAG | INTERNAL ROTATION LAG | GERBER'S LIFT OFF TEST | PALPATION OF BICEPS TENDON TEST | DIAGNOSIS BASED ON USG SHOULDER |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 39/M | 6212335 | RIGHT | 0 | 0 | 20 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 44/M | 6522334 | RIGHT | 0 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 40/M | 6114451 | RIGHT | 30 | 0 | 30 | 0 | 0 | 0 | POSITIVE | POSITIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 41/M | 6223344 | LEFT | 20 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 38/M | 6156239 | RIGHT | 10 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | POSITIVE | BICIPITAL TENDINITIS |
| 70/M | 5900223 | RIGHT | 40 | 10 | 30 | 0 | 0 | 0 | POSITIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 58/M | 6000023 | RIGHT | 90 | 0 | 50 | 10 | 10 | 10 | POSITIVE | POSITIVE | PRESENT | PRESENT | NEGATIVE | NEGATIVE | COMPLETE SUPRASPINATUS TEAR |
| 58/M | 6334521 | RIGHT | 90 | 10 | 90 | 10 | 20 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 90/M | 5903303 | RIGHT | 30 | 0 | 10 | 0 | 0 | 0 | POSITIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINITIS |
| 57/M | 6333456 | LEFT | 40 | 10 | 20 | 0 | 0 | 0 | POSITIVE | POSITIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 57/F | 6455667 | RIGHT | 20 | 10 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 43/M | 5989126 | LEFT | 40 | 0 | 30 | 0 | 0 | 0 | POSITIVE | POSITIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 48/M | 6234310 | LEFT | 40 | 0 | 20 | 0 | 0 | 0 | POSITIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINOSIS |
| 60/M | 6547789 | RIGHT | 0 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 46/F | 6134226 | RIGHT | 20 | 10 | 30 | 0 | 10 | 0 | POSITIVE | NEGATIVE | ABSENT | PRESENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 44/F | 5890126 | RIGHT | 30 | 10 | 30 | 0 | 10 | 0 | POSITIVE | NEGATIVE | ABSENT | PRESENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINITIS |
| 57/M | 6000433 | RIGHT | 0 | 10 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | POSITIVE | BICIPITAL TENDINOSIS |
| 39/M | 6433562 | LEFT | 20 | 20 | 10 | 0 | 0 | 0 | POSITIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINITIS |
| 63/F | 6001258 | LEFT | 0 | 10 | 30 | 0 | 30 | 0 | NEGATIVE | NEGATIVE | ABSENT | PRESENT | POSITIVE | NEGATIVE | SUBSCAPULARIS TENDINOSIS |
| 50/M | 5962310 | LEFT | 20 | 0 | 30 | 0 | 10 | 0 | NEGATIVE | NEGATIVE | ABSENT | PRESENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 66/M | 6003426 | RIGHT | 70 | 20 | 70 | 0 | 10 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 55/F | 6533278 | RIGHT | 10 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | BICIPITAL TENDINITIS |
| 47/F | 6785543 | RIGHT | 0 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 59/F | 5877689 | RIGHT | 0 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 50/M | 6111287 | RIGHT | 60 | 20 | 60 | 0 | 20 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 49/M | 5933201 | RIGHT | 20 | 10 | 20 | 0 | 0 | 0 | POSITIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINITIS |
| 40/M | 5960342 | RIGHT | 0 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |

# MASTER CHART

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67/M | 6432556 | RIGHT | 90 | 10 | 90 | 10 | 20 | 0 | NEGATIVE | NEGATIVE | ABSENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 53/M | 6001210 | RIGHT | 0 | 0 | 20 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 61/F | 6467634 | RIGHT | 60 | 20 | 70 | 0 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 42/M | 5922356 | RIGHT | 30 | 10 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 39/F | 6177828 | LEFT | 0 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 48/M | 6822476 | RIGHT | 90 | 0 | 90 | 10 | 30 | 20 | POSITIVE | POSITIVE | PRESENT | PRESENT | POSITIVE | NEGATIVE | FULL THICKNESS ROTATOR CUFF TEAR. |
| 54/M | 5923400 | RIGHT | 0 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 59/F | 6578665 | RIGHT | 20 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 60/M | 6755654 | LEFT | 70 | 10 | 80 | 10 | 20 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 37/M | 6321445 | RIGHT | 80 | 10 | 80 | 10 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NEGATIVE | NEGATIVE | ADHESIVE CAPSULITIS |
| 47/F | 6098334 | RIGHT | 30 | 10 | 30 | 0 | 0 | 0 | POSITIVE | POSITIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 56/F | 5878667 | RIGHT | 20 | 10 | 20 | 0 | 0 | 10 | NEGATIVE | NEGATIVE | PRESENT | ABSENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 45/M | 6000982 | RIGHT | 90 | 20 | 60 | 0 | 20 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 61/F | 6234122 | RIGHT | 60 | 20 | 70 | 10 | 30 | 20 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 68/M | 5988784 | RIGHT | 10 | 10 | 10 | 0 | 0 | 10 | NEGATIVE | NEGATIVE | PRESENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 49/F | 6212133 | LEFT | 20 | 10 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 38/F | 5900214 | RIGHT | 0 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 53/F | 6220354 | LEFT | 70 | 0 | 90 | 0 | 40 | 20 | POSITIVE | POSITIVE | PRESENT | PRESENT | POSITIVE | NEGATIVE | FULL THICKNESS ROTATOR CUFF TEAR. |
| 48/M | 5789675 | RIGHT | 10 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 60/M | 6098765 | LEFT | 20 | 10 | 40 | 0 | 10 | 0 | POSITIVE | POSITIVE | ABSENT | PRESENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 47/M | 5982231 | RIGHT | 70 | 10 | 80 | 10 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 40/M | 6001226 | RIGHT | 70 | 20 | 80 | 10 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 43/F | 6221215 | RIGHT | 90 | 20 | 90 | 0 | 20 | 20 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 53/M | 5912146 | RIGHT | 0 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 61/F | 6026785 | LEFT | 50 | 20 | 50 | 0 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 46/M | 6511545 | RIGHT | 80 | 10 | 70 | 0 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 65/M | 5888549 | LEFT | 10 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 67/M | 6165478 | RIGHT | 50 | 20 | 60 | 0 | 20 | 20 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 49/M | 6434556 | LEFT | 80 | 20 | 70 | 0 | 40 | 20 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 51/M | 5997887 | RIGHT | 90 | 10 | 80 | 0 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 59/M | 6006787 | RIGHT | 0 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 71/M | 6343561 | LEFT | 30 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | AC JOINT ARTHRITIS |
| 55/F | 6676854 | RIGHT | 30 | 0 | 20 | 0 | 0 | 0 | POSITIVE | POSITIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 68/F | 6223245 | RIGHT | 80 | 20 | 60 | 10 | 30 | 0 | NEGATIVE | NEGATIVE | ABSENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 57/M | 6134543 | LEFT | 80 | 20 | 80 | 0 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 49/M | 5908787 | LEFT | 10 | 0 | 0 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 58/F | 6231128 | RIGHT | 90 | 20 | 70 | 10 | 40 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 56/F | 6086887 | RIGHT | 80 | 0 | 80 | 10 | 30 | 20 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 50/F | 6112556 | LEFT | 10 | 0 | 10 | 0 | 0 | 0 | NEGATIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | NORMAL |
| 61/M | 5980983 | RIGHT | 80 | 20 | 50 | 0 | 20 | 20 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 54/M | 5902911 | RIGHT | 10 | 0 | 0 | 0 | 40 | 0 | NEGATIVE | NEGATIVE | ABSENT | PRESENT | POSITIVE | NEGATIVE | SUBSCAPULARIS TENDINOSIS |
| 51/F | 6143786 | RIGHT | 90 | 20 | 60 | 0 | 30 | 10 | NEGATIVE | NEGATIVE | ABSENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 69/F | 5911012 | LEFT | 40 | 10 | 30 | 0 | 0 | 10 | POSITIVE | NEGATIVE | PRESENT | ABSENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINOSIS |
| 74/M | 6001237 | RIGHT | 70 | 20 | 60 | 10 | 20 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |
| 62/F | 6233438 | RIGHT | 90 | 10 | 90 | 0 | 40 | 20 | POSITIVE | POSITIVE | PRESENT | PRESENT | POSITIVE | NEGATIVE | FULL THICKNESS ROTATOR CUFF TEAR |
| 49/M | 5987223 | LEFT | 20 | 10 | 20 | 0 | 0 | 0 | POSITIVE | NEGATIVE | ABSENT | ABSENT | NEGATIVE | NEGATIVE | SUPRASPINATUS TENDINITIS |
| 69/M | 6188923 | RIGHT | 30 | 0 | 30 | 0 | 10 | 0 | POSITIVE | POSITIVE | ABSENT | PRESENT | NEGATIVE | NEGATIVE | PARTIAL SUPRASPINATUS TEAR |
| 72/M | 6378665 | RIGHT | 90 | 10 | 80 | 10 | 30 | 10 | NEGATIVE | NEGATIVE | PRESENT | PRESENT | NOT ELICITED | NEGATIVE | ADHESIVE CAPSULITIS |