"FUNCTIONAL OUTCOME OF PLATELET RICH PLASMA IN PARTIAL THICKNESS ROTATOR CUFF TEARS - A PROSPECTIVE STUDY."

By

DR. NIVETHAN RAVICHANDARAN

Thesis submitted to the

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Inpartial fulfillment of the requirements for the degree of

MASTER OF SURGERY IN ORTHOPAEDICS

Under the guidance of

DR. SANTOSH S. NANDI **M.S. Orthopedics**

PROFESSOR AND HOD

DEPARTMENT OF ORTHOPAEDICS

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B.M.PATIL MEDICAL COLLEGE

HOSPITAL & RESEARCH CENTRE

VIJAYAPURA, KARNATAKA

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Date: 30/06/2024

Place: Vijayapura

DR. NIVETHAN RAVICHANDRAN

B. L. D.E. (DEEMED TO BE UNIVERSITY)
SHRI B. M. PATIL MEDICAL COLLEGE,
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPURA, KARNATAKA

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"FUNCTIONAL OUTCOME OF PLATELET RICH PLASMA IN PARTIAL THICKNESS ROTATOR CUFF TEARS - A PROSPECTIVE STUDY."** a bonafide research work done by

Dr. NIVETHAN RAVICHANDRAN in partial fulfillment of the requirement for the degree of M.S. in ORTHOPAEDICS.

Date- 30/06/2024

Place-Vijayapura

DR. SANTOSH S NANDI M.S. ORTHO

Professor and HOD
Department Of Orthopaedics
B.L.D.E. (Deemed To Be University)
SHRI B.M. PATIL MEDICAL COLLEGE
HOSPITAL & RESEARCH CENTER,
VIJAYAPURA, KARNATAKA

B. L. D.E. (DEEMED TO BE UNIVERSITY)
SHRI B. M. PATIL MEDICAL COLLEGE,
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPURA, KARNATAKA

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Date: 30/06/2024

Place:Vijayapura

DR. SANTOSH S NANDI M.S. ORTHO
Professor and HOD,
Department Of Orthopaedics
B.L.D.E. (Deemed To Be University)
SHRI. B. M. PATIL MEDICAL COLLEGE
HOSPITAL & RESEARCH CENTRE
VIJAYAPURA, KARNATAKA

B. L. D.E. (DEEMED TO BE UNIVERSITY)
SHRI B. M. PATIL MEDICAL COLLEGE,
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPURA, KARNATAKA

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Date- 30/06/2024
Place-Vijayapura

DR. ARAVIND PATIL
PRINCIPAL
B. L. D. E. (DEEMED TO BE UNIVERSITY)
SHRI B.M.PATIL MEDICAL COLLEGE
HOSPITAL & RESEARCH CENTRE,
VIJAYAPURA, KARNATAKA

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DR.NIVETHAN RAVICHANDRAN

LIST OF ABBREVIATION

PRP - Platelet rich plasma

FGP - Fibroblast growth factor VAS-Visual analogue scale

NSAID - Nonsteroidal anti inflammatory drugs RMS -Roles Maudsley score PRF: Platelet-Rich Fibrin

PPP - Platelet-Poor Plasma

SGHL - Superior Glenohumeral Ligament

MGHL - Middle Glenohumeral Ligament

IGHL - Inferior Glenohumeral Ligament

PDGF - Platelet Derived Growth Factor

ACD - Acid citrate dextrose

VAS - Visual Analogue Scale

RBS - Random blood sugar

RBC - Red blood corpuscle

ABI - Autologous blood injection

WBC - White blood corpuscle

USG - Ultrasonography

MRI - Magnetic Resonance imaging PDGP-Platelet derived growth factor

TGP - Transforming growth factor VEGP-Vasoactive endothelial growth factor EGP- Epidermal growth factor

IGP - Insulin like growth factor

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ABSTRACT

INTRODUCTION

A partial thickness rotator cuff tear is defined as the disruption of tendon fibres and is characterized by pain and activity limitation , occurring due to degenerative factors and overhead movements. Have high prevalence in older people . The estimated prevalence of PTRCT is 13% to 37% and is expected to increase with the increasing of the aging population due to positive correlation between age and rotator cuff diseases.

MATERIALS AND METHOD

This study was carried out by the BLDE Hospital's orthopaedics department from August 1, 2022, until May 31, 2024. Forty patients were selected for the research. Our criteria were used to select the patients, and a diagnosis was only ascertained following a clinical assessment. The pain status was assessed using the VAS grading methods.

Patients were evaluated at four, six, and six-month intervals. Both the amount of activity and the pain after the injection were noted. Based on the level of activity and pain at the conclusion of the sixth month, the final result was rated in three categories: excellent, very good, and poor.

RESULTS

The result for PRP group for partial thickness rotator cuff tear at 4 weeks , 6 weeks and six months follow up.

CONCLUSION

We came to the conclusion that PRP injection in partial thickness rotator cuff tear provides pain relief and prolonged relief, at 4 weeks, 6 weeks and 6 months of follow-up.

KEYWORDS: Visual Analogue Scale, Platelet Rich Plasma, Partial thickness rotator cuff tear

INTRODUCTION

Twenty to forty percent of shoulder joint disorders are marked by rotator cuff tears (RCTs), which are characterised by discomfort and restrictions in activity. The pain is usually brought on by degeneration at the rotator cuff muscle. RCT can be separated into partial-thickness and full-thickness tears based on the size of the tear. There is considerable clinical evidence that most partial rips cannot heal on their own, notwithstanding the paucity of study on the genesis and natural history of partial-thickness rotator cuff tears (PTRCT).

The growth factors required for tissue regeneration are concentrated in PRP, which is derived from autologous blood. A relatively new and developing idea for treating partial thickness rotator cuff problems is PRP therapy. Our study examined the efficacy of PRP therapy for partial thickness rotator cuff injury.

Repetitive microtears of the supraspinatus muscle are the cause of degeneration. The most common symptom of a partial thickness rotator cuff tear is excruciating discomfort that worsens with shoulder joint range 0f motion. In more extreme situations, the discomfort will also get worse when going about normal tasks.

Conservative treatment methods, such as non-steroidal anti-inflammatory drugs, orthotics, physical therapy, stretching exercises, corticosteroid injections, and extracorporeal shockwave therapy, result in significant improvement for about 80% of patients.

OBJECTIVE OF THE STUDY

1. To study the functional outcome of partial thickness rotator cuff tear treated with platelet rich plasma therapy.
2. To study complications of platelet rich plasma therapy in partial thickness rotator cuff tear.

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22. Three different kinds of PRP preparation techniques were used by Augustus D. Mazzocca et al. In one way, an arthrex ACP syringe is utilised, and in another, a gps 3 platelet concentrating device. Both systems only needed one spin. In the third kind, twofold spin process was employed, with the first centrifugation occurring at 1500 rpm and the second at 6300 rpm (14).

ANATOMY OF SHOULDER JOINT

Although the glenohumeral joint is a ball-and-socket joint anatomically, it is regarded as a diarthrodial, multiaxial joint functionally. The primary articulation of the shoulder girdle is the glenohumeral articulation, which connects the humeral head to the glenoid cavity of the scapula. The scapulothoracic, acromioclavicular, and sternoclavicular joints' minor articulations are likewise included in the latter. The human body's most movable joint is the glenohumeral joint.The shoulder joint is composed of four articulations:

1. *The Sternoclavicular*: This joint connects the upper extremity to the axial skeleton.
2. *The Acromioclavicular*: The joint between the acromion process and the clavicle.
3. *The Scapulothoracic*: This joint is physiological. It is the joint that connects the dorsal thoracic wall to the underside of the scapula. It permits the scapula to pass easily across the ribs.
4. *The Glenohumeral*: It is a ball-and-socket synovial joint located in the scapula's shallow glenoid fossa. It is well-suited for movement because of the articulation (which makes up only 25–30% of the articulation at any given moment) between the scapula's small glenoid fossa and large humeral head. It also consists of the bone, ligament, tendon, and bursa structures that support joint function.

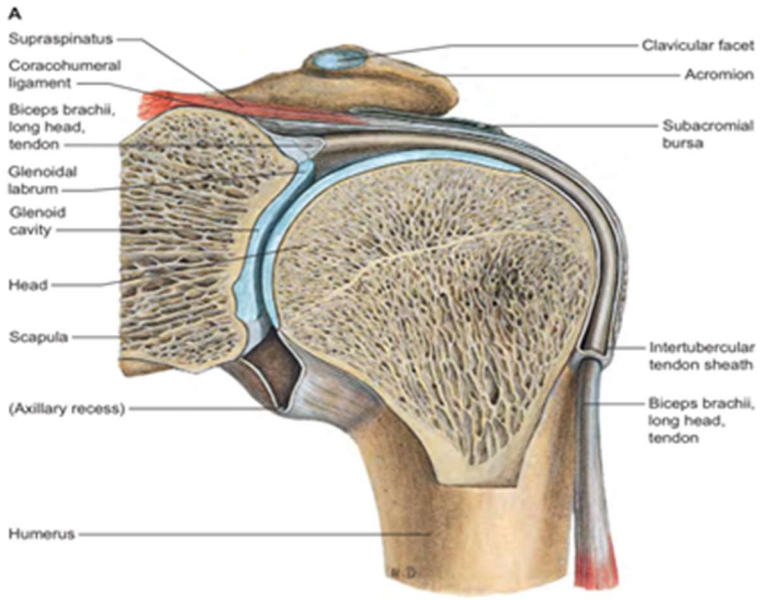


Fig. Coronal section of the left shoulder joint. Anteriorly placed coronal section to show tendon of biceps, long head.

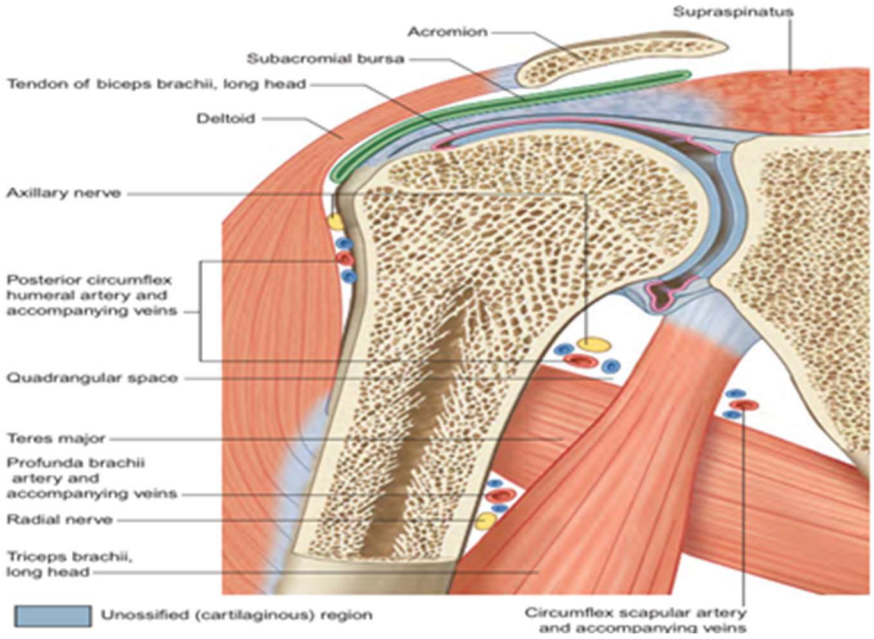


Fig. Coronal sections taken from the posterior portion of the left shoulder joint. Coronal slice positioned posteriorly to display the quadrangular space's contents and the subacromial bursa

Sternoclavicular Joint

The only bone link that connects the upper limb to the trunk is this joint. The synovial saddle-shaped sternoclavicular joint is made up of the costoclavicular ligament, the anterior and posterior sternoclavicular ligaments, the interclavicular ligament, the medial side of the clavicle, the sternoclavicular joint surface, an articular disc, and a capsule. The articular surfaces of the clavicle and sternochondral regions are connected to a fibrous capsule that encloses the joint.

Acromioclavicular Joint

The synovial plane joint known as the acromioclavicular joint is composed of a capsule, the lateral end of the clavicle, the medial border of the acromion, an articular disc, the acromioclavicular ligaments, the coracoclavicular ligaments, and the coracoacromial ligament. Rather than the joint's bony structure, the surrounding ligaments sustain the joint's stability. The acromion is more likely to go under the clavicle and downward because of the planar joint surfaces' medial and downward slope. Enclosing the joint, the articular capsule attaches at the articular borders. The capsule is supported by the deltoid and upper trapezius muscle fibers, the anterior, inferior, and posterior acromioclavicular ligaments, and the robust superior acromioclavicular ligament superiorly. The wedge-shaped articular disc, which descends into the joint from the superior region of the capsule, achieves more congruence between the articular surfaces. Despite their separation from the joint medially, the coracoclavicular ligaments are the primary stabilisers of the joint. The posteromedial conoid ligament and the anterolaterally located trapezoid ligament are its two components, which are named for their respective shapes. The acromioclavicular joint cannot be dislocated anteriorly or superiorly without the help of the coracoclavicular ligaments.

Scapulothoracic Joint

The scapulothoracic joint lacks the typical joint traits, making it an imperfect anatomic joint. The scapulothoracic joint moves freely without any ligament restrictions, with the exception of its attachment to the axial skeleton at the acromioclavicular joint and the coracoclavicular ligaments. The scapulothoracic joint, despite not being a real joint, is crucial to the biomechanics of the shoulder complex. The upper limb functions from a movable platform, the scapula.

Glenohumeral Joint

The most dynamic and least stable joint in the body is the glenohumeral, a multiaxial ball and socket joint. The head of the humerus, glenoid fossa, glenoid labrum, glenohumeral ligaments, coracohumeral ligament, and transverse humeral ligament make up this joint. Additionally, the long head of the biceps tendon and the rotator cuff muscles' tendons stabilise the glenohumeral joint externally.

The Humeral Head and Glenoid Fossa

Less than one-third of the humeral head's size, or the narrow and shallow glenoid fossa, articulates with it. The axis forms a 135° angle with the shaft and a 30° angle (retroversion angle) with the frontal plane. The glenoid points anteriorly, laterally, and slightly superiorly, while the head looks superiorly, medially, and posteriorly. Compared to the humeral head's convexity, the humeral head's concavity is less noticeable and uneven. The inconsistency of the minimal bony contact between the two joint surfaces accounts for the instability of the joint and the need for additional stabilising mechanisms. Hyaline cartilage covers the articular surfaces of the glenoid and humerus. On the humerus, it is thinner towards the periphery and thickest in the centre, whereas on the glenoid part of the joint, the opposite is true.

Glenoid Labrum

The glenoid fossa is encircled by the glenoid labrum, a rim of fibrous and fibrocartilaginous tissue. Some researchers claim that the majority of the labrum is made up of fibrous tissue, with fibrocartilage only being found in a tiny, narrow transitional zone where the glenoid fossa's hyaline cartilage, the periosteum of the scapular neck, and the capsule converge. The face of a clock is commonly used to characterize it: the anterior part is marked 3 o'clock, the posterior piece is marked 9 o'clock, the superior portion is labeled 12 o'clock, and the inferior portion is designated 6 o'clock. Consequently, it is referred to as anterosuperior, anteroinferior, postero-superior, and postero-inferior, as shown in fig.

The labrum performs a variety of crucial tasks for the glenohumeral joint, such as the following:

- Offers a 50% glenoid deepening and an extension to the glenoid fossa's concavity.
- Increased stability against translating pressures is achieved by providing a greater depth and, to a lesser extent, width.
- Acts as an attachment point for the long head of the biceps tendon, the ligaments, and the capsule.
- Provides an articular surface to the humeral head.

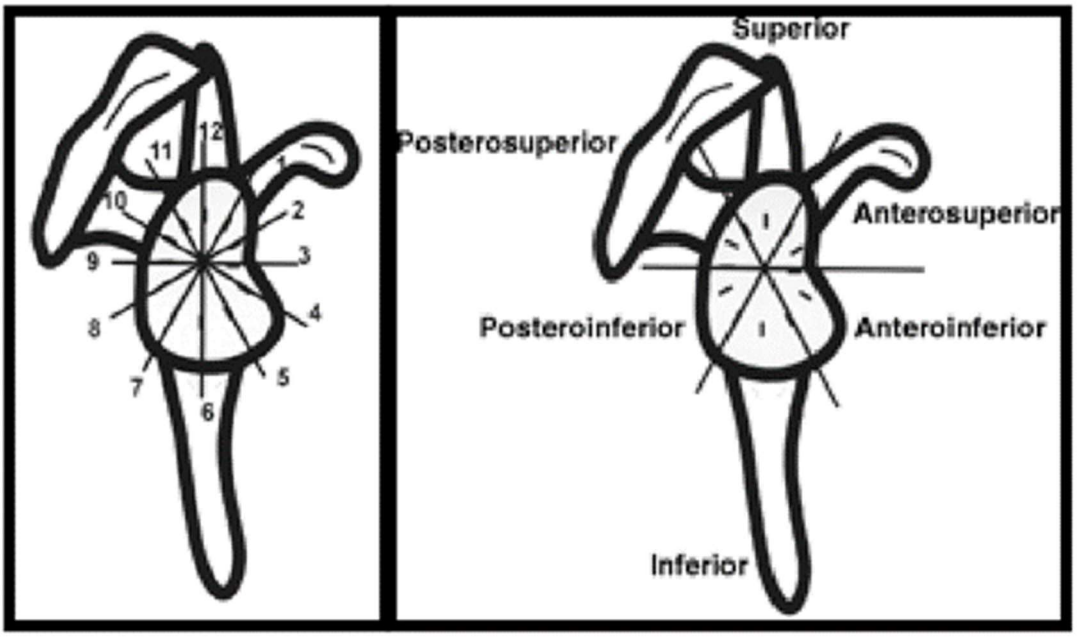


Fig. The glenoid labrum.

Joint Capsule

The flexible, thin, superfluous sheath that makes up the joint capsule both adds to the joint's mobility and instability. Just medial to the tuberosities, the humeral head capsule connects to the anatomic neck. It then continues onto the medial surface of the shaft, directly beneath the articular head. Its two entrances are on the capsule. The long head of the biceps tendon can pass through the upper end opening, whereas the subscapular bursa can communicate with the joint cavity through the anterior opening. The capsule affixes to the labrum and, less commonly, the scapular neck on the glenoid side. Along the whole posterior boundary of the glenoid cavity, the glenoid labrum and the capsule of the glenohumeral joint are tightly connected. However, differences in the glenoid labrum's connection to the joint capsule can be found anteriorly. Based on how close the insertion site is to the anterior part of the labrum, there are three different types of anterior capsular insertions.There are three different types of capsular insertions: type I, which happens near or into the labrum; type II, which happens further medially; and type III, which happens into the scapular neck. Generally speaking, type III insertions are thought to be less stable, possibly contributing to or originating from anterior glenohumeral instability. Most people believe that a type I capsular insertion is more stable. Assistance is required to stabilise the glenohumeral joint because of its laxity, which results in a joint capsule that is double the size of the humeral head. The coracohumeral ligaments and the glenohumeral ligaments contribute to provide support.

Ligaments

The main stabilisers at extremes of motion are the ligamentous restrictions. The base of the lateral coracoids is where the coracohumeral ligament, a thick band of capsular tissue, begins. The stability of the humeral head during adduction is provided by the coracohumeral ligament and the superior glenohumeral ligament, which prevent the humeral head from translating inferiorly during forward flexion, adduction, and

internal rotation. The superior glenohumeral ligament extends from the anterosuperior margin of the glenoid to the top of the smaller tuberosity. (Fig.). The middle glenohumeral ligament is the most variable ligament, missing in 8% to 30% of cases. The inferior glenohumeral ligament is the thickest and most reliable of the three glenohumeral ligaments. Recurrent instability is largely caused by damage to the inferior glenohumeral ligament, which can occur from a single traumatic event (dislocation) or from repetitive microtrauma (such as pitching).

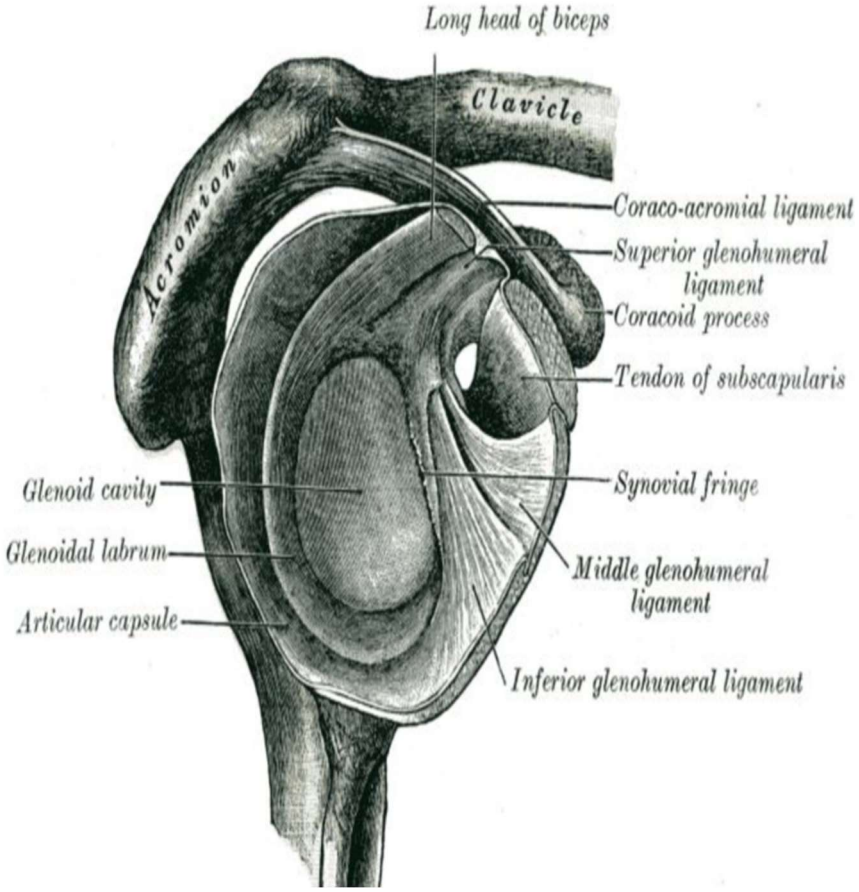


Fig. Interior of Shoulder Joint; lateral aspect.

The anterior stability of the joint is supplied by three unique and intrinsic capsular ligaments. In front of the joint capsule, the anterior, inferior, middle, and superior glenohumeral ligaments unite to form a Z. These ligaments tense up and limit the humerus's range of motion. Following the involvement of other static and dynamic stabilisers, they are the final structures to offer stability.

The superior glenohumeral ligament (SGHL)

The superior glenoid border and base of the coracoid process are where the superior glenohumeral ligament (SGHL) originates. These locations are just in front of the long head of the biceps tendon's origin. It continues until the coracohumeral ligament's merging point at the fovea capitis line, which is just anterior to the smaller tuberosity. Frequently, a broad band-like structure visible in the axial plane called the superior GHL originates from the superior glenoid tubercle, which runs parallel to the coracoid process. Situated between the

anterior margin of the supraspinatus tendon and the superior margin of the subscapularis tendon, the rotator interval is an important anatomical area. The long head of the biceps tendon is covered and supported by it.

The middle glenohumeral ligament (MGHL)

The middle glenohumeral ligament (MGHL) starts from the superior labrum, which is next to the superior ligament, and the supraglenoid tubercle. It is attached medially to the base of the lesser tuberosity, below the subscapularis tendon. From an arthroscopic perspective, the middle GHL is characterised as being medial to the articular edge and attached to the anterior surface of the scapula. After that, it mixes in with the anterior capsule and is positioned obliquely, posterior to the superior edge of the subscapularis muscle. It is connected distally, beneath the superior GHL's insertion, to the front portion of the proximal humerus. The inferior GHL or the superior GHL alone may be the source of the intermediate GHL. It can merge with the subscapularis tendon prior to reaching the tuberosity, although it usually inserts at the humerus near the base of the lesser tuberosity. A labral rip or misplaced fragment occurs as it passes in front of the glenoid labrum. The middle glenohumeral ligament's main function is to restrict external rotation at 45° of abduction. An additional secondary constraint against anterior dislocation is supplied by this ligament.

The inferior glenohumeral ligament (IGHL)

The inferior glenohumeral ligament (IGHL) complex originates from the anteroinferior labrum and the glenoid border. It connects to the smaller tuberosity directly below the middle ligament. The three components of this ligament are an anterior band, a posterior band, and the axillary pouch or recess.The ligament is fashioned like a hammock. While the posterior band of the inferior GHL attaches to the posterior inferior glenoid quadrant, the anterior band inserts in the anterior glenoid rim near the mid glenoid notch. As the humeral head rotates, the anterior and posterior bands reciprocally tighten. At 90° of abduction, the anterior band serves as the main barrier against anterior dislocation and external rotation. Anterior instability in throwing athletes is mostly caused by the degeneration of this ligament. Visualising the glenohumeral ligaments, particularly the inferior GHL, is much easier with MRI arthrography that allows for sufficient distention of the shoulder joint.

The coracohumeral ligament (CHL)

The transverse humeral ligament, the tops of the larger and lesser tuberosities, and the lateral border of the horizontal arm of the coracoid process are where the wide band known as the coracohumeral ligament (CHL) connects. Twenty percent of people may have pectoralis minor tendon fibres insert onto the coracohumeral ligament. Thus, the coracohumeral ligament is merely the pectoralis minor muscle's primitive insertion. This ligament's main function is to prevent the humeral head from inferior subluxing and to stabilise the adducted shoulder.

The transverse humeral ligament

The transverse humeral ligament extends from the greater to the smaller tuberosity. The primary purpose of this ligament is to maintain the stability of the long head of the biceps tendon within the bicipital groove.

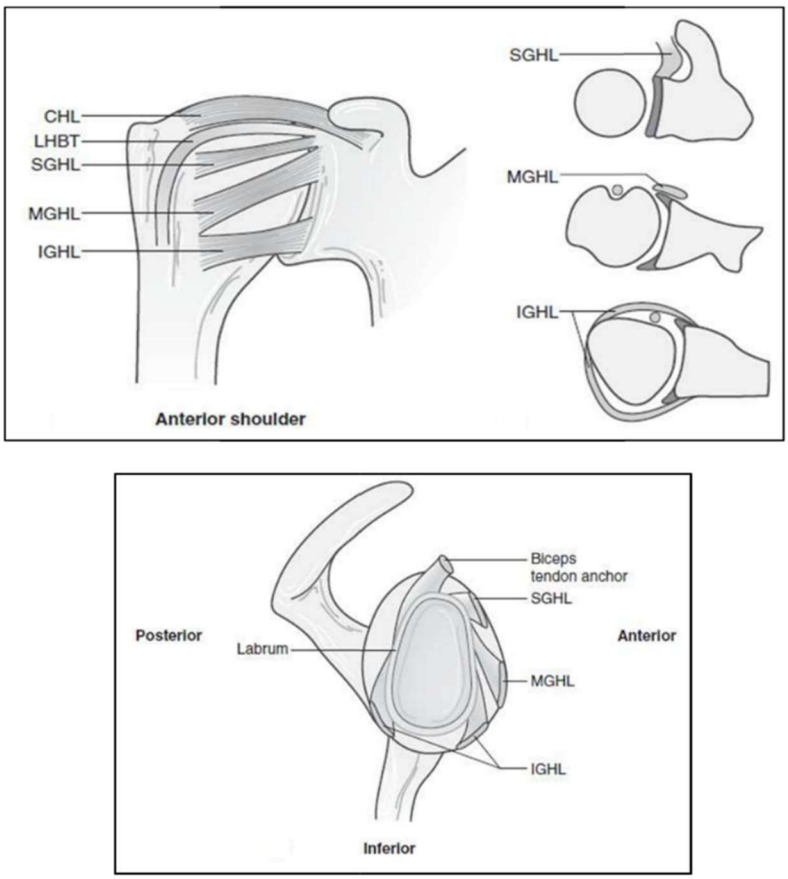


Fig. The ligaments of the shoulder joint.

Rotator cuff muscles and long head of the biceps tendon

Four connected muscles that originate from the scapula and attach to the tuberosities make up the rotator cuff. Their tendons form a continuous cuff around the head, providing the cuff muscles with many opportunities to rotate and realign the humeral head within the glenoid fossa. This creates the perfect muscular balance necessary for precisely timed movements. The medial two thirds of the supraspinous fossa on the scapula give rise to the supraspinatus muscle. This muscle inserts onto the superior aspect of the greater tuberosity and joint capsule after passing beneath the acromion and acromioclavicular joint. The suprascapular nerve (C4-C5-C6) innervates the supraspinatus muscle. Its main functions include abduction of the shoulder and stabilisation of the humeral head in the glenoid fossa.

The infraspinatus muscle attaches to the middle facet of the larger tuberosity and joint capsule after emerging from the medial two thirds of the infraspinous fossa of the scapula. The suprascapular nerve, which runs from C4 to C5, innervates this muscle. Its main function is to externally rotate and stabilise the humerus' head.

The teres minor muscle emerges from the upper two thirds of the dorsal part of the lateral border of the scapula and joins to the joint capsule and lower facet of the bigger tuberosity. Its primary job is to stabilize and spin the head of the humerus externally.

The subscapularis muscle attaches to the joint capsule and lesser tuberosity after emerging from the scapula's subscapular fossa. The upper and lower subscapular nerves (C5-C6-C7) innervate this muscle. Its main function is to externally rotate and stabilise the humerus' head.

The supraglenoid tubercle of the scapula is the source of the tendon that runs the entire length of the biceps muscles. It leaves the shoulder and affixes itself to the radius tuberosity after passing via the bicipital groove beneath the transverse humeral ligament. The musculocutaneous nerve (C5–C6) innervates the long head of the biceps. Its primary purpose is to flex and stabilize the humeral head and elbow.

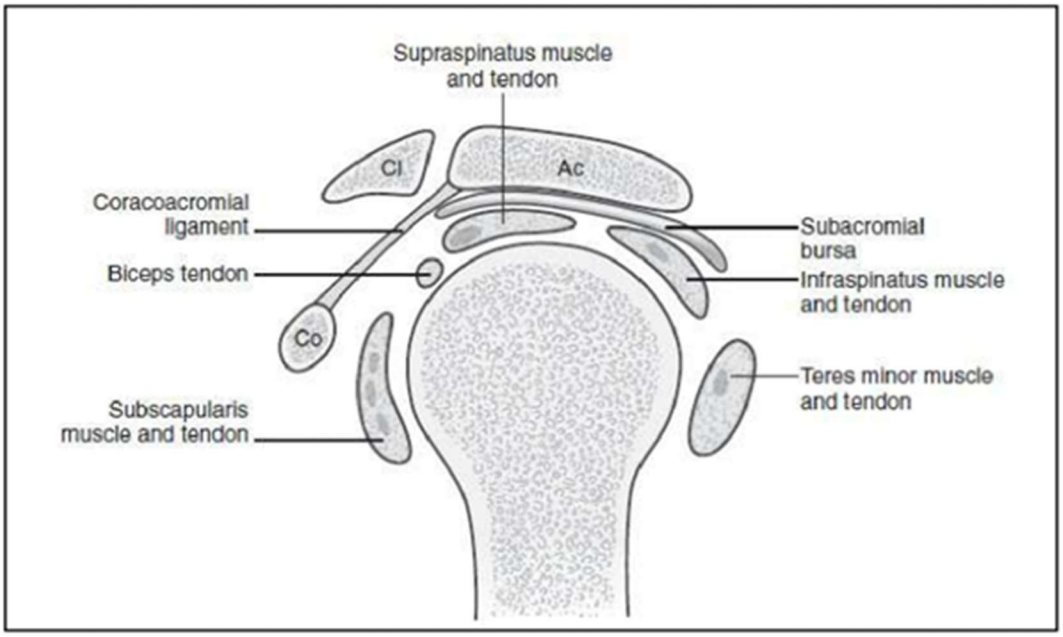


Fig. The Rotator Cuff.

The joint subdeltoid Like the scapulothoracic joint, the subdeltoid joint is anatomically nonexistent. The subdeltoid is made up of the coracoacromial ligament, the rotator cuff, the subacromiodeltoid bursa, the undersurface of the acromion, and the long head of the biceps tendon. Their concave structure complements the convex humeral head, just like the glenoid fossa does. This is the fifth joint in the shoulder, and numerous authors have noted its significance. Two primary functions of the subdeltoid joint are as follows:

- Gives the humerus's head a smooth surface to glide on, particularly during abduction and flexion.
- Superior stability is provided by ii. It resists the humeral head's upward pull during abduction and flexion.

The acromion's undersurface and the coracoacromial ligament's degenerative alterations tend to confirm that this physiologic joint is involved in shoulder mobility. The tendinous insertions of the rotator cuff muscles, articular capsule, coracohumeral ligament, and glenohumeral ligament complex combine form a confluent sheet prior to entering into the humeral tuberosities. Dividing the tendons of the infraspinatus and supraspinatus muscles by blunt dissection is a challenging task. They connect about 15 mm proximal to their

insertion. Close to their musculotendinous connections, the infraspinatus and teres minor merge. The biceps tendon is encased in a sheath formed by the union of the supraspinatus and subscapularis tendons at the opening of the bicipital groove. A section of the supraspinatus tendon makes up the sheath's roof, and the subscapularis tendon forms the sheath's floor. This connection is important because lesions of the long head of the biceps frequently coincide with tears in the subscapularis tendon. A thick band of fibrous tissue called the coracohumeral ligament runs from the coracoid process along the capsule's surface to the tuberosities that lie between the supraspinatus and subscapularis tendons. The ligament forms part of the roof of the biceps sheath, blending together with the capsule and supraspinatus tendon, deep to the tendinous insertion of the cuff. From the coracoid, where the coracohumeral ligament originates, posterior to the infraspinatus, there is a 1-cm-wide thickening of fibrous tissue. This band runs between the cuff tendons and the capsule as an extension of the coracohumeral ligament. Additionally, a strip of fibrous tissue originating from the coracohumeral ligament spreads posterolaterally, covering the superficial insertions of the supraspinatus and infraspinatus tendon. Five unique layers were found in histologic analyses of the supraspinatus and infraspinatus tendons. Large arterioles and coracohumeral ligament fibers are found in the outermost layer, or layer one. The fibers in this 1 mm thick layer are positioned obliquely to the long axis of the muscular bellies. The direct tendinous insertion into the tuberosities is represented by layer two, which is 3 to 5 mm thickThe huge bundles of closely spaced parallel tendon fibers, ranging in diameter from 1 to 2 mm, comprise layer two. Similar collagen fiber bundles paralleling the muscle's long axis and splaying before insertion can be seen in the anatomy of the subscapularis tendinous insertion. The floor of the biceps sheath is made up of bundles from the subscapularis that combine with supraspinatus fibers, whereas layer two supraspinatus fibers create the roof of the sheath. Layer three is composed of smaller collagen bundles with a less regular orientation than layer two, and it is around 3 mm thick. The interdigitating meshwork formed by the fibers in this layer traveling at 45-degree angles to one another aids in the fusing of the cuff tendon insertion. At the most anterior edge of the supraspinatus, layer four merges with the coracohumeral ligament, consisting of thick collagen bands and loose connective tissue. The interwoven collagen sheet that runs from the glenoid labrum to the humerus is part of layer five, which is 2 mm thick and forms the shoulder capsule.

The term "footprint" is frequently used to describe the location where the rotator cuff tendon inserts at the larger tuberosity. In 2002, Dugas and colleagues analyzed twenty typical cadaver rotator cuff specimens and used a three-space digitizer to map the footprint. The supraspinatus, infraspinatus, teres minor, and subscapularis tendons had mean medial-to-lateral insertion widths of 12.7 mm, 13.4 mm, 11.4 mm, and 17.9 mm, in that order. The midpoint of the supraspinatus was the site of the mean minimal medial-to-lateral insertion breadth of the whole rotator cuff insertion, measuring 14.7 mm. Along the anterior 2.1 cm of the supraspinatus-infraspinatus insertion, the distance from the articular surface to the tendon insertion was less than 1 mm. At the most inferior aspect of the teres minor insertion, this gap gradually grew to a mean of 13.9 mm. The supraspinatus, infraspinatus, teres minor, and subscapularis insertions were measured with mean anteroposterior distances of 1.63 cm, 1.64 cm, 2.07 cm, and 2.43 cm, respectively. The triangle region in the anterior and superior shoulder where there are no rotator cuff tendons is known as the rotator interval. Therefore, the coracoid medially, the subscapularis inferiorly, and the supraspinatus superiorly define the interval's boundaries. The transverse humeral ligament serves as a laterally defined apex of the triangle. The

rotator interval contains the superior glenohumeral ligament, biceps tendon, and coracohumeral ligament. In pathologic conditions, the rotator interval is changed; it has been observed to be enlarged in individuals with shoulder instability and constricted in those with adhesive capsulitis. The coracoid and the anterior acromion, which are separated by the coracoacromial ligament, make form the coracoacromial arch, which is located above the glenohumeral joint. Typically, the distal clavicle is also regarded as a component of the arch. This arch is crossed by the proximal humerus, the o/biceps tendon, the subacromial bursa, and the rotator cuff tendons. any visible and palpable location within the subcutaneous tissues, whether acquired or contracted.

PATHOPHYSIOLOGY

Even though the shoulder complex gives the arm its maximum range of motion, this improved mobility has a cost; it frequently causes instability or impingement of the shoulder's soft tissue or bony structures, which results in pain. Any of the four separate muscles and tendons that make up the rotator cuff can experience structural breakdown and/or tissue disruption, which is referred to as rotator cuff tears. Partial-thickness rotator cuff tears (PT RCTs) are tears that cause disruption to the rotator cuff but do not completely penetrate the tendon. PT RCTs do not cause the muscle-tendon unit to retract and are more common than full-thickness rips. A full-thickness tear is characterised by the total termination of rotator cuff fibres, leading to in contact between the articular and bursal spaces.

Among a wide range of patients, RCTs are among the most prevalent shoulder issues, and as the population ages, the prevalence of RCTs rises linearly. As many as 65% of people over 70 have a PT RCT, while as many as 28% of those over 60 have an FT RCT, according to data from cadaveric investigations. RCTs frequently exhibit no symptoms, which makes early detection and therapy difficult. Studies that indicate the size of a tear may have a major role in the development of symptoms point to a trend towards a link between tear size, progression, and the emergence of new symptoms.

Many factors might contribute to rotator cuff injuries, which can irritate or harm the muscles and tendons. The aetiology of RCTs has been the subject of numerous ideas over time, which have been divided into extrinsic and internal causes. One of the most well-known extrinsic pathogenic elements in RCTs is the chronic impingement idea, which was first put forth by Neer. One of the most common reasons people have shoulder pain is impingement, especially if they continue to be physically active well into their thirties and forties.

Impingement, or the mechanical compression and probable abrasion of the rotator cuff tendons, is a forerunner to RCTs if the condition progresses. (Fig.). The supraspinatus tendon is the most often impinged site and is caused by shoulder movement above the horizontal plane or repetitive overhead use. Pinching like this can result in inflammation, which thickens the tendon and damages it more with repeated motions. Muscle fraying results from swelling within the muscle tissue, which reduces its vascularity.

Dislocations, fractures of the greater tuberosity of the humeral head, and mechanical misuse of the shoulder are other significant extrinsic causes. RTCs may also be caused by elements that hinder tissues' innate ability to repair. For example, research has demonstrated that nicotine negatively affects tendon recovery. Compared to nonsmokers, smokers are less likely to recover from rotator cuff repair procedures and have lower post-operative function and satisfaction. Another possible risk factor for RCTs is diabetes. Researchers studying patients with asymptomatic rotator cuff illness discovered that diabetics had a greater incidence of histologic rotator cuff tendon alterations associated with ageing and a more limited range of motion in their shoulders.in rates of re-tear following surgical repair, as well as increased incidence of complications and infections following repair.

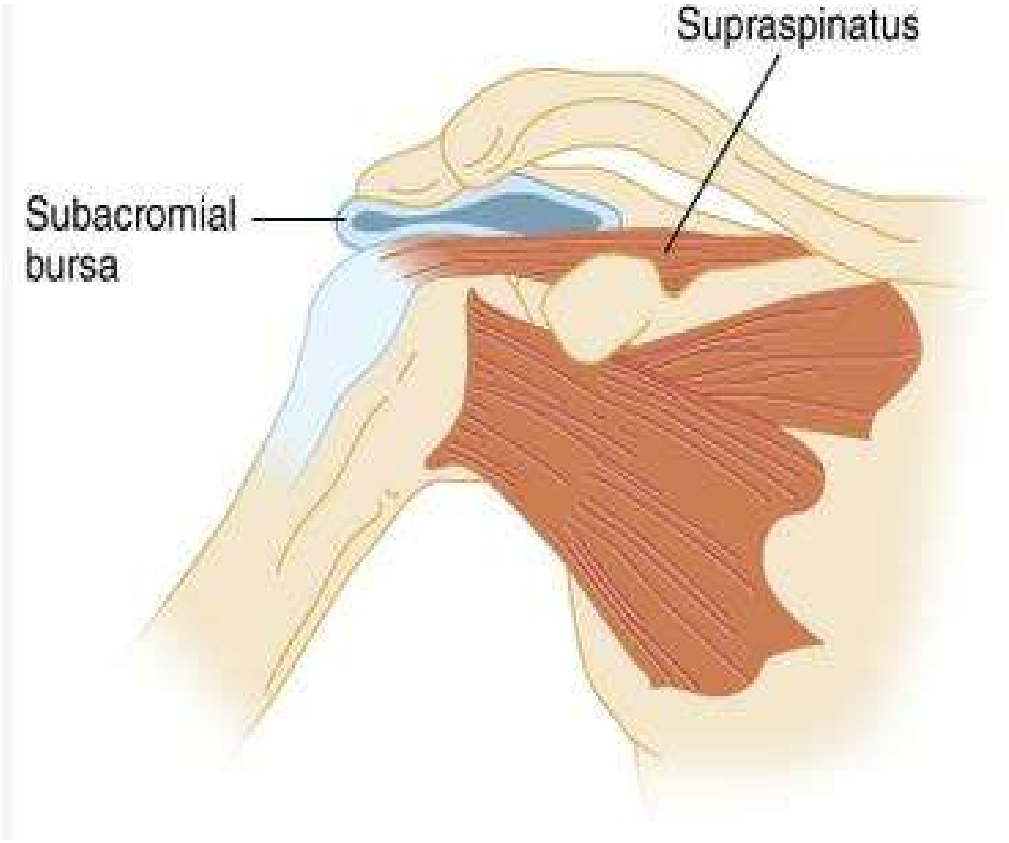


Fig.

The illustration depicting the impingement of the rotator cuff and subacromial bursa shows the possible abrasion and mechanical compression of the rotator cuff muscles. When an individual raises their arm above their head, the larger tuberosity of the humerus and the acromion and coracoacromial arch may squeeze the supraspinatus and subacromial bursa. This pinching may result in thickening of the tendon and inflammation that damages the tendon over time with repeated motions.

Based on clinical evidence, it appears that intrinsic causes such microtrauma or degeneration are the primary cause of most RCTs. A single traumatic injury can cause rotator cuff tears, but more frequently, misuse of the muscles and tendons over an extended period of time causes intrinsic tendon degradation, which eventually results in a tear. These degenerations appear to be frequent modifications implicated in the early degeneration of rotator cuff tendons prior to tearing, based on their frequency and distribution.

The cells receive (GFs). The primary intrinsic causes in tendinopathies and tendon ruptures appear to be pathologic and physiological changes to the extracellular matrix. The development of hard tissues, matrix maturation, and mineralization have all been linked to transglutaminases (TGs). Since they have a crosslinking activity under normal circumstances, they are crucial for preserving the structural integrity of tendons. Reductions in the expression of the TG2 protein, one of the nine distinct TGs present in mammals, have been observed in injured supraspinatus tendons. This decline in TG2 expression may indicate that the tendon's capacity for repair has been reached.

Diagnosis of Rotator Cuff Tears

A physical examination and a comprehensive assessment of the patient's medical history are required for the diagnosis of rotator cuff damage. Actually, distinguishing in this manner between an FT RCT and PT RCT, or even between rotator cuff injuries and other inflammatory rotator cuff diseases, may prove to be quite difficult.

When a patient is examined and has a history of acute shoulder pain following substantial trauma or chronic shoulder discomfort, the diagnosis is mostly based on the discovery of rotator cuff weakening. Regular shoulder radiographs are helpful in assessing RCTs and related intra-articular disease; two of the most often employed imaging modalities are magnetic resonance imaging (MRI) and ultrasound.

For the diagnosis of rotator cuff disease, ultrasound has shown to be a very useful technique, especially in full-thickness RCTs. It can be used to assess patients with unstable shoulders and post-operative patients with metallic artifacts that would mask MRI findings. However, for the most precise results, the ultrasonic technique depends heavily on a skilled operator and the right equipment, making it less useful in clinical settings. Conversely, when evaluating individuals who have shoulder pain, conventional MRI is a very helpful imaging modality (Fig.). Research indicates that MRI can diagnose FT RCTs with 100% sensitivity and 95% specificity, and PT RCTs with 82% sensitivity and 85% specificity.

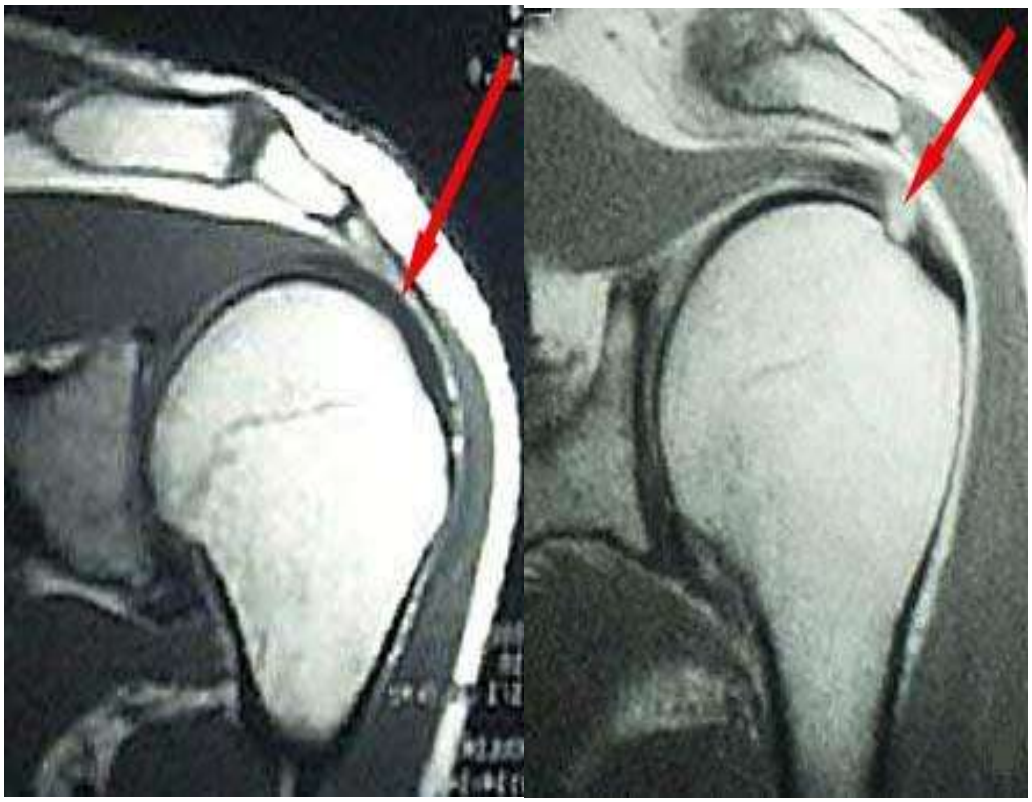


Fig. MRI Images showing rotator cuff tears and normal scans The radiographic differences between a normal rotator cuff (left) and a torn rotator cuff (right) are shown in the figures. A tear in the rotator cuff fibers is shown by the white signal that interrupts the black band on the right, in contrast to the image of a normal rotator cuff, which shows a totally biack signal band below the tip of the red arrow.

Treatment Options

It can be difficult to decide whether to treat rotator cuff tears surgically or non-operatively. There is now no clear agreement on the optimal course of care for patients, and novel treatment approaches are constantly being investigated and proven to be effective, frequently with conflicting outcomes. There is a large range in the published success rate of non-operative management (33%–92%). While there isn't enough data at this time to draw firm conclusions about the conservative management of RCTs, some authors advise against surgery for patients who are in pain but don't show any obvious or growing weakening. Additionally, positive relationships have been discovered with other prognostic markers, including tear size, duration of symptoms, and clinical presentation. The length of the symptoms (less than a year) and the size of the tears. Conservative treatment outcomes are positively correlated with symptoms lasting less than a year and tears less than 1 cm, for example.

Surgical therapy carries a larger risk of morbidity than conservative approaches, particularly in the older population with many co-morbidities. Nevertheless, in certain circumstances, it can provide more immediate pain relief and increased function. The majority of people who suffer from rotator cuff dysfunction are elderly, and their limitations are severe enough to impair their ability to function. Conservative treatments utilized for partial RCTs presently include corticosteroid injection, concentrated physical therapy, oral medication such as non-steroidal anti-inflammatory drugs (NSAIDs), modification of daily activities, and/or medication. The goal of conservative treatment is to lessen shoulder pain and return function while lowering the possibility of further rotator cuff tendon injury.

A. Physical Therapy

Stretching and strengthening exercises are the basic components of physical therapy (PT), which patients carry out at home either in accordance with a prearranged programme or under a physical therapist's supervision [28]. Nonetheless, there is no set standard for a workout program's attributes. To reduce stiffness and improve function and range of motion, exercise routines should be tailored to the particular region of the tear.

B. Medications

NSAIDs are among the systemic medications used by individuals with shoulder pain. Selective inhibitors of cyclo-oxygenase-2 (COX-2) have also been released recently for the treatment of shoulder pain [28]. Both COX-2 inhibitors and NSAIDs have negative side effects and show short-term effectiveness in controlled clinical trials. Up to 76% of patients experience unfavorable gastrointestinal effects after taking conventional NSAIDs [28]. In rare cases, NSAIDs have also shown negative effects on the neurological system, dermatology, and kidneys [28]. Potential cardiovascular risk has been raised in cases where patients have been using COX-2 inhibitors or NSAIDs for longer than three months [28, 46]. It is therefore advised to use such medications for brief periods of time. Angiotensin-converting enzyme (ACE) inhibitors, which are primarily

used to treat hypertension and congestive heart failure, interference with diuretics, beta-blockers, and angiotensin type-2 receptor antagonists are other common adverse effects that can affect a patient's ability to control their blood pressure.

C. Corticosteroid injections

In clinical settings, a variety of intra-articular injections are frequently utilised to relieve pain. Treatment for shoulder pain typically involves injections into the glenohumeral and acromioclavicular joints, or into the subacromial space. The injections are usually given in a solution containing a corticosteroid, such as cortisone, a longer-acting anaesthetic, and a short-acting anaesthetic. In order to enable patients to complete PT programmes with less discomfort, corticosteroids are typically prescribed in addition to oral NSAIDs and physical therapy. The literature on corticosteroid injection in patients with randomised controlled trials is scarce, and reported outcome metrics are not always consistent. Patients with partial RCTs who had failed PT and had symptoms that lasted longer than six months did not show any appreciable improvements.

The precision with which the drug is delivered to the intended target is a major worry. Certain investigations have found a variation in the efficacy of the intervention carried out under radiographic control, typically under ultrasound guidance, for example. Corticosteroid injections into tendon structures can be a useful way to reduce pain temporarily, but they can also restrict or prevent their use for a while. They may also result in collagen fiber necrosis, weakened tissue, and a higher chance of rupture. Because of this, it is not recommended to have more than two injections year, once every six months.

Therefore, sodium hyaluronate injection may be more advantageous for patients experiencing chronic pain than corticosteroids. Due to its natural viscosity, sodium hyaluronate, a regular constituent of synovial fluid, helps to preserve physiological joint friction.

Randomized controlled trials (RCTs) have demonstrated its efficacy in a limited number of patients by demonstrating pain reduction, decreased need for oral analgesics, and improved range of motion. It seems to be an effective therapeutic choice with little to no side effects. Further study is required to confirm its efficacy.

PLATELETS AND PRP BIOACTIVE COMPONENTS IN GENERAL

Platelets are important components of the clotting cascade. These are colourless, non-nucleated, megakaryocyte-derived cell fragments that are present in bone marrow. They consist of granules and cytokines, the most important of which is termed. It has about thirty proteins and is necessary for soft tissue hemostasis and healing.

The granules generate and secrete many proteins within minutes of the platelets aggregating. Numerous chemicals, such as osteocalcin, fibrinogen, fibronectin, insulin-like growth factor, platelet-derived growth factor, vascular endothelial growth factor, epidermal growth factor, and epithelial cell growth factor, are secreted by the granules. These growth factors trigger the morphogenesis, differentiation, and proliferation of their target cells. The three normal stages of wound healing are inflammation, proliferation, and remodeling. During the period of tissue damage and inflammation, platelets become active. They begin producing their proteins, including growth factors and cytokines, through the granules. Bioactive compounds like as calcium, adenosine, histamine, serotonin, and dopamine are also produced by them.

Serotonin and histamine will increase the capillaries' permeability before they are transferred to the wound site. Normal platelet aggregation does not occur until a stimulant is present. A biological mixture of proteins enables the platelets to start clotting and the thrombus process as soon as there is tissue damage. Fibronectin, laminins, collagen, von Willebrand factor, among other proteins, stimulate the platelets. Platelet aggregation and activation are brought on by even the secretions produced by the platelet itself, including serotonin and adenosine diphosphate. The platelets will start to produce the fibrin clot after they have been activated.

It is challenging to determine the proper PRP concentration for clinical application. Between 150,000 and 350,000 platelets per litre of blood constitute the normal platelet concentration. To improve healing, a level of at least 1,000,000/L is required. Most PRP has a level that is 3- to 5-fold higher than the baseline. Other trials, however, have indicated efficacy at 2.0- to 8.5-fold doses.

According to the CLASSIFCATION Ehrenfest et al. (2009) presented, four primary groups of Preparations may be identified based on their fibrin architecture and cell composition.

1. 0p–11. Following activation, preparations known as leucocyte-poor platelet-rich plasma (PRP) or pure platelet-rich plasma (PRP) have no leucocytes but a low-density fibrin network.
2. Activated leucocyte and fibrin network preparations are known as leucocyte-and PRP (L-PRP) products. The most commercial or experimental systems can be found in this family. In particular, numerous automated methods have been created in recent years, necessitating the use of particular kits that permit the least amount of handling of the blood samples and the highest level of preparation standardisation.

3. Leucocyte-poor platelet-rich fibrin preparations, also known as pure platelet-rich fibrin (P-PRF), are platelet-only and have a high-density fibrin network. These products cannot be injected or used in the same way as conventional fibrin glues; they only come in the form of a substantially activated gel.
4. Second-generation PRP products, also known as leucocyte- and platelet-rich fibrin (L-PRF) or L-PRF, are leucocyte- and fibrin-network-containing preparations. A multidisciplinary consensus meeting that was published in 2012 heavily referenced, supported, and validated this classification system.

The current thinking states that PRP is primarily used to increase the concentration of platelets at a damaged site. In order to initiate the healing process following an acute injury, platelets are often stimulated during the inflammatory phase. When platelets are added to an acute injury, the local tissue's baseline concentration of platelets is increased. When conservative treatments fail to relieve chronic injuries, it's likely that the inflammatory phase has ended, platelets are low, and the injury's ability to heal is diminished. PRP would provide two advantageous results in these conditions.

PRP injections simply stimulate the injured tissue and initiate an inflammatory response, turning the chronic damage into a fresh acute injury. This makes them an easy way to treat tendon, ligament, or muscle problems.Second, it stands to reason that including autologous platelet concentrations will expedite the healing process. Now that the cause of this recent injury has been identified, it can be managed in a post-injection setting under close observation (e.g., bracing, immobilisation, or non-weight bearing). During this time, anti-inflammatory medications and treatments must be avoided to avoid the desired effect from being undone.

Preparation PRP

Principles of PRP preparation include: Differential centrifugation is the method used to create PRP. Distinct cellular components are sedimented using different acceleration forces in differential centrifugation, depending on their varying specific gravities. PRP can be prepared in several different methods. Both the PRP approach and the buffy-coat method can be used to prepare it. In the PRP procedure, red blood cells are first separated by centrifugation, which is then followed by a second centrifugation to concentrate platelets. PRP is produced using a double centrifugation process in which whole blood is initially drawn into tubes containing anticoagulants. To separate RBCs from the remaining volume of whole blood, the first spin step (soft spin) is carried out at constant acceleration. After the first spin stage, the total blood separates into three layers: an upper layer mainly composed of leucocytes and platelets, an intermediate layer rich in WBCs called the buffy coat, and a bottom layer mainly composed of red blood cells.To make pure PRP (P-PRP), the upper layer and superficial buffy coat are transferred to an empty, sterile tube. To produce PRP that is rich in leucocytes (L-PRP), The whole buffy coat layer and almost no RBCs are transferred. The second spin, sometimes known as the hard spin, comes next. The centrifuge's second spin ought to be just sufficient to aid in the production of the erythrocyte-platelet soft pellets at the tube's bottom. The top part of the volume, which is primarily made up of platelet-poor plasma (PPP), is eliminated. The buffy coat is collected after whole blood is centrifuged at a "high speed" in the buffy coat method. A buffy coat can be created by drawing a very little amount of whole blood that has a high concentration of leucocytes. The challenge is distinguishing the RBC layer from this thin buffy coat layer, which primarily consists of white blood cells and platelets. The cubital vein is used to collect blood for the creation of autologous PRP. The therapeutic use and desired concentration dictate how much blood is

acquired. Centrifugation is then used to separate the platelets from the plasma. The PRP can be obtained using a wide variety of technologies that are already on the market.

ACQUIRING BLOOD FROM CUBITAL VEIN

An alternative method for an automated centrifugation process that uses an infrared microprocessing sensor to discern between red blood cells and platelet-rich plasma can be used to separate platelets from whole blood and then automatically transfer the product to a different syringe. With this kind of equipment, concentrations appear to be more accurate and repeatable. Error should be reduced because the blood product is automatically separated. An illustration of one of these devices is the "Magellan Autologous Platelet Separator System". The blood collection tube must have an anticoagulant, regardless of method. Anticoagulant tubes can be purchased individually or are often included in the kits that come with the products.

The idea of activating the platelets before usage appears to be controversial in the literature. While some studies expressly identify the substance used to activate the PRP, others do not make any reference of whether or not the PRP was activated. De Vos and colleagues published a study on the effects of PRP on Achilles tendinopathy without addressing activation.

Foster and colleagues 45 propose activation with bovine thrombin in their review article. Thoms and colleagues mention using calcium and thrombin together (bovine, human, or recombinant).

PRP can be transformed into a platelet gel with the help of thrombin and calcium. This gel has the ability to constrict blood vessels to reduce bleeding and distribute growth factors to aid in wound healing. The activation will also enhance platelet function. When utilised as a scaffold, the concentration of leukocytes in the gel can improve tissue attachment and prevent infection. It has also been shown to reduce post-operative discomfort. Usually used intraoperatively, platelet gel is a substance that seals wounds and promotes bone healing.

Keith S. Hetchman et al. generated PRP by utilizing the Cascade autologous platelet system, a commercially available kit. Nine milliliters of the patient's blood were drawn out and placed in a tube with one milliliter of trisodium citrate and thioxotropic separation gel. The tube containing the blood was centrifuged at

1100 rpm for six minutes.Following the first centrifugation, plasma is separated from RBC and WBC. The plasma is put into a tube that holds 0.1 ml of $CaCl_2$.

For the preparation of PRP, Christos Thanasas et al used a "Gravitational platelet separator system 3 (GPS)". With 3- 5ml of anticoagulant, 27 to 55ml of blood were drawn. After 15 minutes of centrifuging whole blood at 3200 rpm, they eventually administer 3-6 cc of PRP. Using a GPS 1 system, T M Bielecki et al. generated PRP in an in vitro study after obtaining 54 milliliters of whole blood in a tube containing 6 milliliters of citrate solution. After centrifuging the entire blood for 12 minutes at 3200 rpm, six milliliters of PRP were recovered.

Three different kinds of PRP preparation techniques were used by Augustus D. Mazzocca et al. Autologous conditioned plasma (ACP) from Arthrex This approach uses a double syringe, while the alternative method uses a device that concentrates platelets separately by gravity. Both systems only needed one spin. The third type employed a double spin technique, with the first spin occurring at 1500 rpm and the second spin occurring at 6300 rpm.

PREPARATION SIMPLIFICATION

1. Before centrifugation, whole blood should be kept between 20 and 24 degrees Celsius.
2. Centrifuge whole blood.
3. Its density causes three layers to form: platelets and leucocytes in the middle layer, RBCs in the bottom layer, and platelet-poor plasma in the top layer.
4. Remove the tube's supernatant plasma (top layer).
5. Insert another sterile tube with the buffy-coat layer in it.
6. Use a leucocyte filtration filter or centrifuge at a low speed to separate leucocytes.

STEPS

1. Whole blood should be venipunctured into acid citrate dextrose (ACD) tubes.
2. Avoid cooling the blood either prior to or during the platelets' separation.
3. Spin the blood gently in a centrifuge.
4. Transfer the platelet-containing supernatant plasma (without anticoagulant) into an additional sterile tube.
5. Increase the centrifuge speed of the tube to obtain a platelet concentration.
6. Platelet-poor plasma (PPP) makes up the lower 1/3 and platelet-rich plasma (PRP) the upper 2/3. At the bottom of the tube, platelet pellets form.
7. Gently shake the tube buffy coat to suspend the platelet pellets in at least 2 to 4 ml of plasma after removing the platelet-poor plasma.

THERAPEUTIC USES

The benefits and safety of platelet-rich plasma have been extensively studied in both human and animal trials. Many of these studies have provided convincing evidence of the safety and efficacy of PRP in the surgical and therapeutic settings. Their key shortcomings are the small sample sizes, lack of controls in the human studies, and contradictory results. Because PRP doses and post-procedure procedures vary, it is challenging to compare research. There appear to be as many research outlining PRP's benefits as there are contradicting findings. As PRP usage increases, it becomes necessary to address the question of whether PRP is as beneficial during the acute stages of tissue healing as it might be during chronic disease.

FIG. A,B&C : Steps of preparation (A), PRP after first centrifuge (B) and after second centrifuge (C)

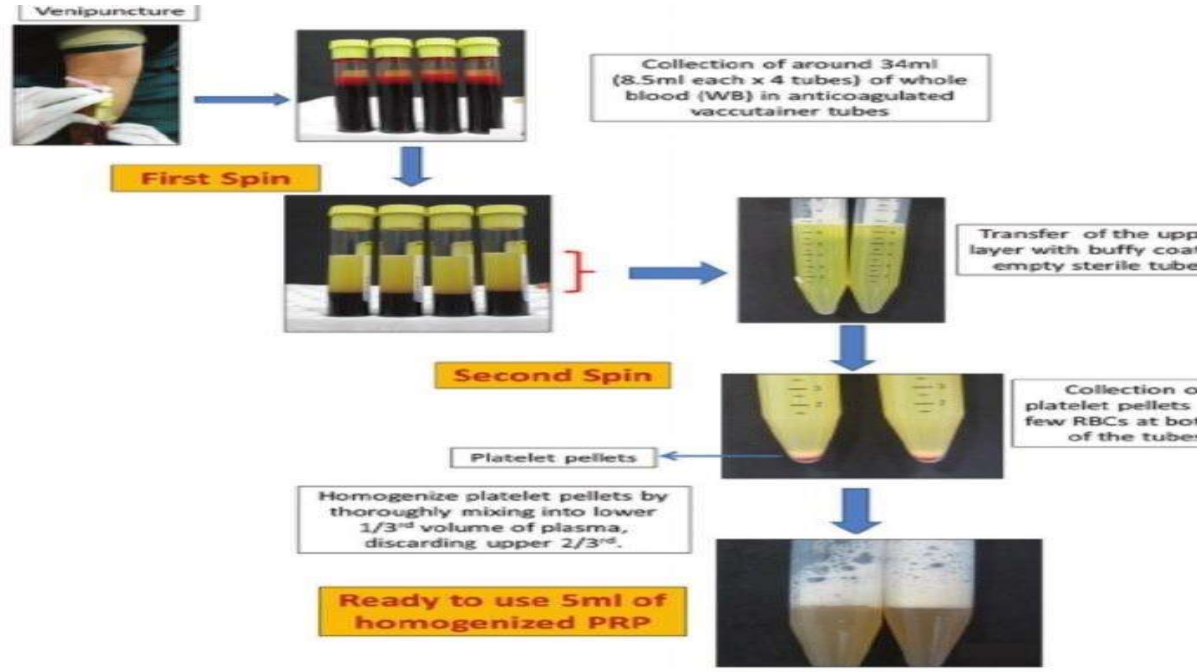


Fig A

Fig B

Fig C

Early Use of Platelet-Rich Plasma

Since the first reports of PRP therapy's clinical use in the 1980s and 1990s, its applications in the domains of cardiac, dental, and maxillofacial surgery have gained wider recognition in regenerative medicine and other fields. Hemorrhage and hematologic abnormalities following cardiopulmonary bypass have been demonstrated to be effectively addressed by platelets, an autologous source of transfusion used in heart surgery. PRP treatment of tooth extraction sites improved bone regeneration in sockets with compact mature bone and normal morphology, as demonstrated by Anitua. In their investigation of the effects of PRP on bone density and bone maturation rate in bone graft restorations of mandibular continuity deficiencies, Marx and colleagues in maxillofacial surgery demonstrated that adding PRP to grafts enhanced bone development. PRP therapy is an injectable biologic that has gained popularity in modern sports and musculoskeletal medicine because of its potential to cure a range of degenerative conditions, mend injured tissue, and hasten the return to sports. It is applied to hasten the healing of cartilage, muscles, ligaments, and tendons.

Fundamentals of Wound Healing

Because of their limited blood supply and sluggish cell turnover, tendons, ligaments, and cartilage repair processes are extremely slow and ineffective. For this reason, the effectiveness of PRP in encouraging healing is very significant. Generally, wound healing can be divided into three phases: remodelling, proliferation, and inflammation. Early stages of inflammation are characterized by hemostasis, in which platelets aid in the formation of clots, and the release of growth factors, which help activate and attract inflammatory cells like neutrophils and macrophages to the site of injury. Granulation, contraction, and epithelialization, as well as the creation of an extracellular matrix, are characteristics of the proliferation phase. Collagen and scar tissue are also produced during the remodelling process.

COMPONENTS OF PLATELET-RICH PLASMA

PLATELETS

Platelets serve a critical role in mediating the anabolic effects of PRP by releasing growth factors from their alpha granules, in addition to being vital for hemostasis. During the initial stages of wound healing, activated platelets clump together and produce a fibrin matrix that draws and encourages cell migration into the wound. The continuous production of cytokines and platelet growth factors, which support cell adhesion, differentiation, and communication, then uses this matrix as a tissue scaffold. Despite storing both angiogenic and antiangiogenic chemicals, platelets release distinct molecules at different times. Prominent growth factors secreted by platelets that are critical to the healing process include platelet-derived growth factor (PDGF), transforming growth factor, vascular endothelial growth factor, basic fibroblast growth factor, and insulin-like growth factor.

LEUKOCYTES

Leukocytes play a key role in wound healing, host defense against pathogenic microorganisms, and the inflammatory response. Neutrophils play a role in the inflammatory stage of the healing process of a lesion. Debriding and phagocytosing injured tissue and cell debris, monocytes and macrophages start the process of tissue healing. The growth factors secreted by macrophages, like those by platelets, are essential for tissue healing and have been associated with the regeneration of subchondral bone. Leukocytes are essential for tissue repair and are a vital first line of defense against pathogenic agents. However, because of their pro-inflammatory and immunologic properties, leukocytes may inadvertently harm nearby cells and tissues, which could postpone the benefits of platelet-rich plasma therapy.Studies conducted in vitro have demonstrated that PRP with large amounts of leukocytes may create an inflammatory environment that impedes the healing process. Boswell and colleagues' experiments on tendon models also showed that boosting platelet concentrations was more successful at boosting PRP efficacy than lowering leukocyte concentrations, which lowered the inflammatory response. More research is needed to establish the optimal leukocyte concentration that will maximise benefit and minimise harm for each target tissue type.

BLOOD CELLS

The centrifugation process reduces or eliminates the red blood cell content of PRP. RBCs' major job is to carry and provide the tissues with nutrients, oxygen, other metabolic gases, and regulatory chemicals like nitric oxide. Nitric oxide has been linked to mediating insensitivity in diseased cartilage to the anabolic activities of insulin like growth factor, even though it is known to cause vasodilation. Iron included in heme molecules can produce harmful oxygen free radicals during the oxidative process, which results in the host cells dying. According to theory, this detrimental process takes place in human synovial cells treated with RBC concentrates, sharply quickening the degeneration of cartilage and the death of cells.

BIOACTIVE COMPONENTS OF PRP[56]

	Origin	Function
PDGF	Alpha granule of platelets	Cell differentiation, neovascularization
PDGF	Alpha granule of platelets	Cell differentiation, fibroblast migration, extracellular membrane synthesis
PDGF	Alpha granule of platelets	Cell proliferation and differentiation, collagen remodeling
TGF	Alpha granule of platelets	Stimulation of collagen formation
TGF	Alpha granule of platelets	Tendon differentiation
VEGF	Alpha granule of platelets	Neovascularization, prevention of apoptosis
EGF	Alpha granule of platelets	Fibroblast proliferation
Stromal- derived factor	Alpha granules of platelets	Promotes catabolism of degenerative issue; recruitment of mesenchymal stem cells and fibroblasts
Fibrin	Plasma	Component of ECM; stimulation of phagocytosis
Fibronectin	Plasma	Component of ECM; stimulation of phagocytosis
Vitronectin	Plasma	Coordination of cell migration
Interleukin	Macrophage	Increases leukocyte maturation and FGF activity
FGF	Alpha granule of platelets	Neovascularization, stimulation of ECM production and cell migration
IGF-1	Alpha granule of platelets	ECM synthesis, fibroblast proliferation

GUIDELINES FOR USING PLATELET-RICH PLASMA

Since every PRP formulation has different biologic properties and effects, there is still no general agreement on the optimal ways to prepare PRP or the appropriate amounts of blood components to add. This has led to inconsistent results. Researchers have employed different PRP preparation strategies, including differences in final PRP component concentrations, number of centrifugation steps, centrifugation systems, activation approaches with and without thrombin and calcium, and preparation methods. Because there are so many different PRP formulations in use, it can be difficult to draw reliable conclusions from the literature to direct PRP manufacture and define recommendations for usage. This challenge is what led to the formation of PRP.

PRP: ACTIVATED vs. NON-ACTIVATED

Roh and associates, It was shown that growth factor release from PRP activated with a low dosage mixture of thrombin and calcium over a 7-day period was significantly boosted compared to non-activated PRP. It is still unclear whether quick, bolus administration of growth factors is the best method for tissue repair. Studies on PRP activation have produced conflicting results; compared to non-activated preparations, it promotes good bone regeneration but less effective fibroblast differentiation and wound healing. 90% of the growth factors are released within 10 minutes of PRP activation. Since most growth factors have brief half lives, activating PRP will increase their effectiveness. Before injection, PRP can be triggered exogenously by thrombin, endogenously by mechanical stress, and endogenously by calcium chloride. A fibrin network develops after PRP is triggered, solidifying plasma and producing a fibrin clot or membrane. If PRP is overly stimulated, the fibrin network will become unstable. A more stable tetra molecular network is created during physiological activation, which improves cell and growth factor enmeshing.

The majority of commercially available PRP kits do not activate PRP. When drawing blood and reinjecting it, big bore needles are utilised to prevent inadvertent activation by harming cells. Similar to how centrifuge braking mechanisms contribute to unintended activation.

Autologous thrombin was employed by Stefano Gumino et al. to activate platelets. 10% calcium chloride was employed by Juan Ramon Valenti Nin et al(39) to activate platelets intraoperatively. According to research by Kenneth S. Lee et al, puncturing the skin with a needle during an injection will cause bleeding, which will provide the thrombin needed to activate platelets.

DRUGS AND INTERACTIONS

Patients on antiplatelet medication should not receive platelet-derived platelets (PRP) as this may impede platelet degranulation and the release of growth factors and bioactive substances. As a result, the biologic tissue's potential for regeneration is diminished. These antiplatelet medications come in a variety of forms, such as glycoprotein inhibitors, phosphodiesterase inhibitors, and adenosine reuptake inhibitors. In patients taking reversible cyclo-oxygenase inhibitors, such as nonsteroidal anti-inflammatory drugs (NSAIDs),

which are commonly used to manage pain, it was shown that autologous PRP significantly delayed platelet aggregation. It has been demonstrated that the anti-hyperglycemic drug pioglitazone both directly inhibits the synthesis of thromboxane, a factor in platelet aggregation, and amplifies the effects of aspirin in suppressing platelet aggregation and ATP release.

EFFECTS OF PRP IN DIFFERENT TISSUES MUSCLE

According to Kenneth S Lee's paper, PRP research has been done on acute muscle injuries. The damaged muscle was evaluated clinically and using ultrasound technology. 50% of his patients experienced positive clinical and functional outcomes.Following PRP injections, normal muscle tissue underwent microscopic alterations, according to Lindsay Harris in a study on rabbit tissues. rabbits were used in his research. He gave the tissues a 0.5 cc injection of PRP.

Two weeks following the injection, he started to experience irritation. He found calcium deposits and inflammatory cells in the tissue.

Six weeks following the injection, he noted the continued presence of inflammation. Certain cells gave the impression of inflammation. There is fibrosis, calcification, and muscle necrosis.

After 12 weeks of injections, he showed no symptoms of discomfort.

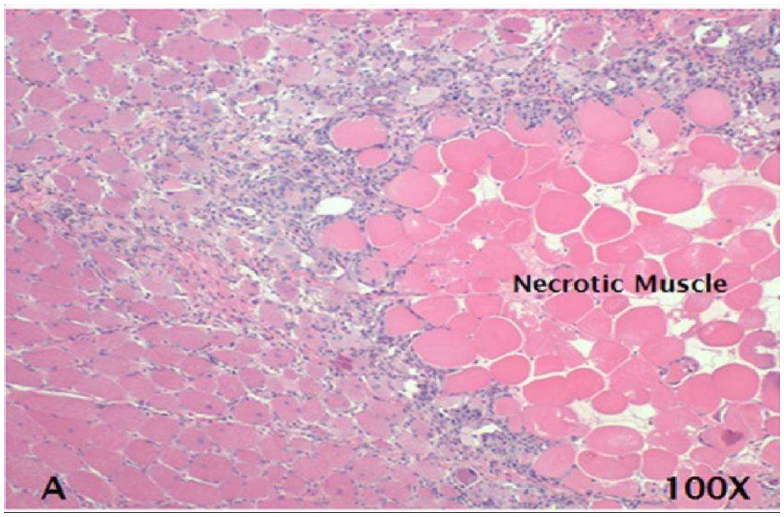


Fig. A

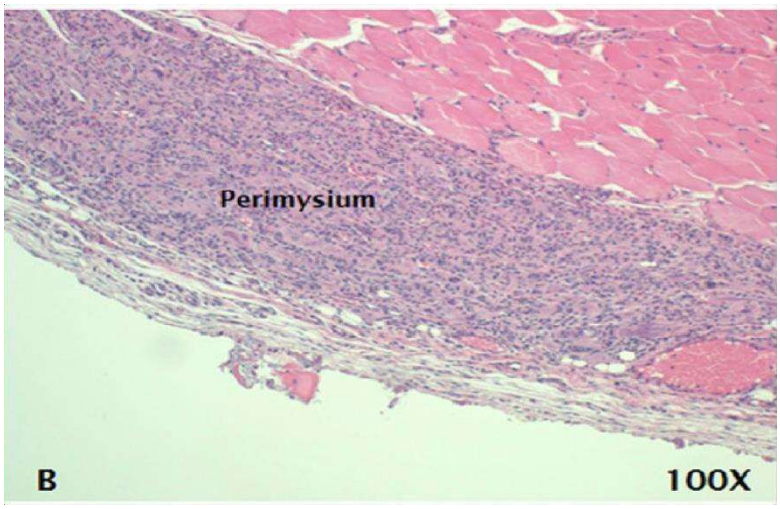


Fig. B

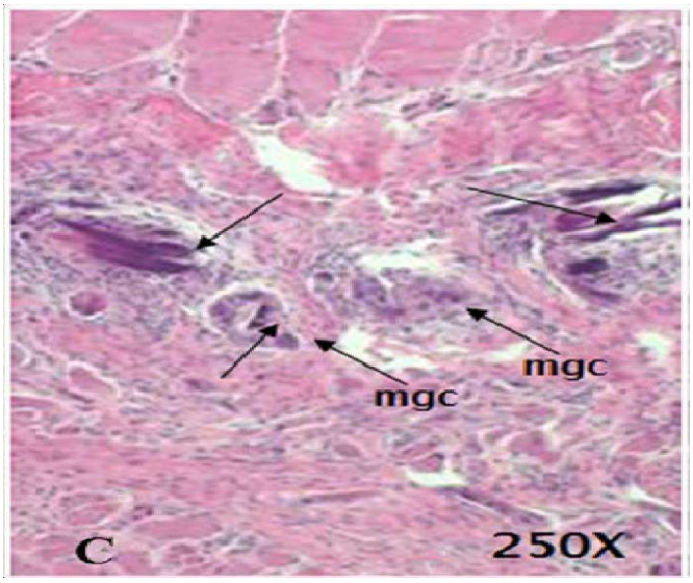


Fig. C

A and B show muscle at two weeks, and C shows muscle at six weeks. Figures A and B show signs of inflammation, and Figure C, where calcium deposition was noticed by arrows, shows signs of inflammation.

SUBCUTANEOUS TISSUE

Harris, N Lindsay(41) He discussed the impact of PRP on subcutaneous tissues in his study. Collagen nodules and fibrous tissue were observed at the two-week mark. Subcutaneous fat is replaced by fibrous tissue and inflammation-related cells.

Microcalcification was discovered at six weeks together with nearby cells of persistent inflammation. Small calcification that had previously been seen and inflammatory cells were not present at 12 weeks.

At 12 weeks, the images in A&B show collagen nodules and collagen

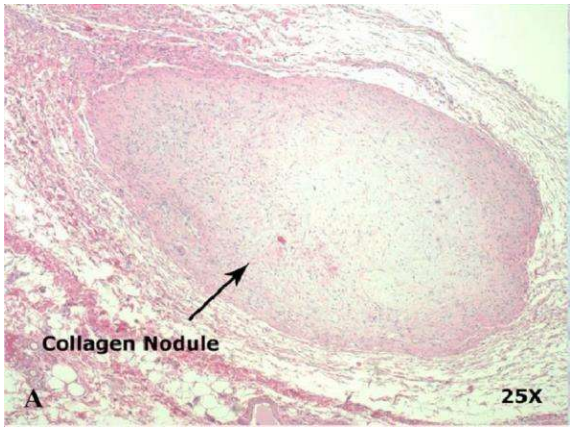


Fig.A

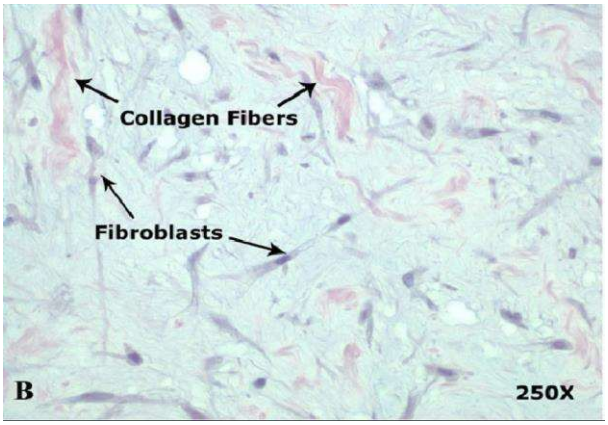


Fig.B

TENDON

Samir Mehta covered the use of PRP for tendinitis and tendon problems in his article. The use of PRP in tendinopathies was also discussed by Steven Sampson in his article.The use of Numerous studies and papers support the use of PRP in tendon injuries. N Lindsay Harris claims that PRP affects rabbit tendon. Two weeks later, dense peritenon and inflammatory cells were seen. Vacuoles and inflammatory cells are also identifiable in tendon tissue. Collagen bundles can also be visible. Inflammation is evident in the pattenon after six weeks. After twelve weeks, the swelling decreased.

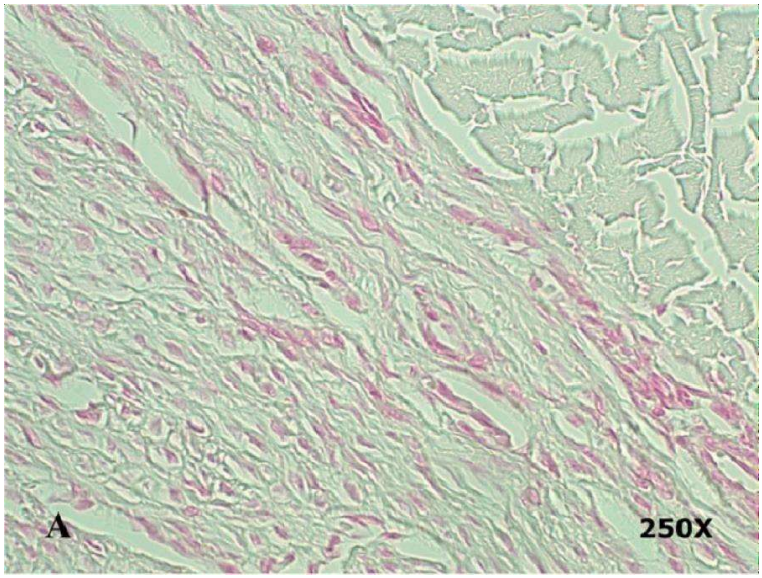


Fig. A shows no calcification at 2 weeks

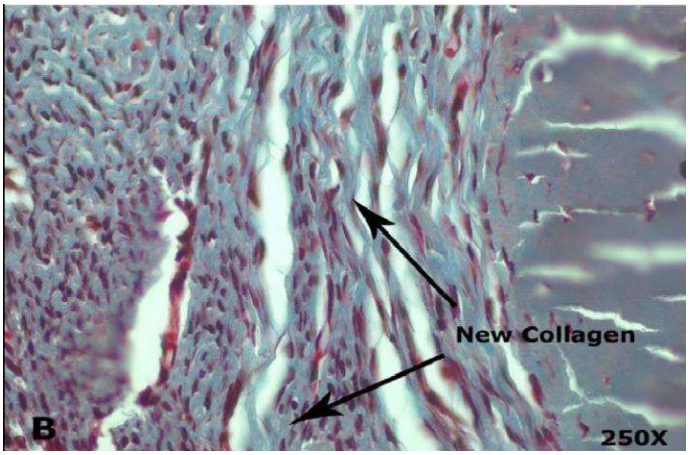


Fig. B shows collagen formation at 2 weeks

LIGAMENTS

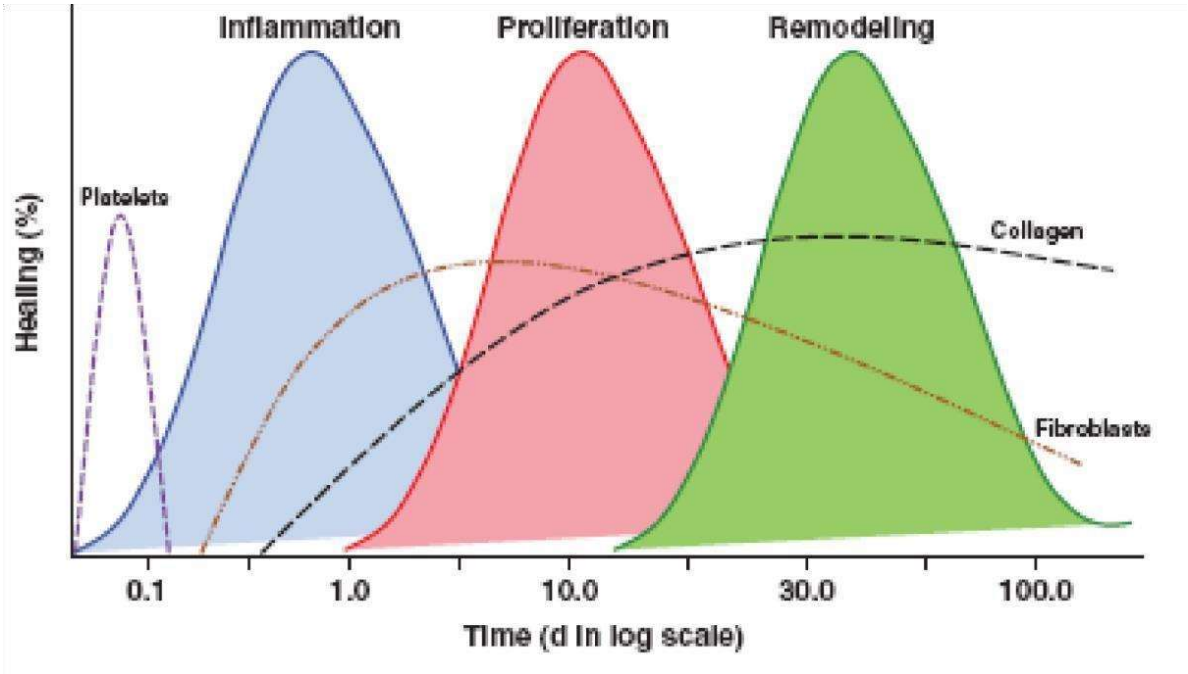
Foster et al.'s and Frie et al.'s research on acute ligament injuries were discussed in Kenneth S. Lee's assessment of the literature. They discovered that patients' return to regular sports and activities was accelerated. Citing the previously cited studies, he asserted that PRP would decrease instability caused by ligament damage and speed up ligament regeneration. N Lindsay Harris discovered that tissues that were thick and inflammatory after two weeks following PRP injection exhibited very little inflammation at six and twelve weeks. This was shown in a study on normal rabbit tissue.

WOUND CLOSURE

Application of platelet-rich plasma helps hasten the healing of wounds. The phases of wound healing and the timing of platelet action are explained in diagram below. The high concentration of platelets in PRP will cause the release of growth factors essential for wound healing. Numerous research describing the impact of PRP on wound healing have been carried out in both human trials and animal models.

Samir Mehta used studies conducted by D R Knighton et al and C Gaino et al study in his work on platelet rich concentrate. In these trials, 78% of patients had their limbs spared, and 17 out of 21 patients achieved reepithelialization.

According to Steven Sampson, nine out of twenty-four patients and twenty out of twenty-four patients, respectively, in the investigations by Crovetti et al. and McAleer et al. experienced full healing of their chronic ulcers. This was stated in his paper about using PRP to treat musculoskeletal conditions.



BONE

There is disagreement over how platelet-rich plasma affects bone mending. The majority of animal research produced positive results, although others did not support the use of PRP. When combined with bone grafting, growth factors like TGF- and PDGF enhance bone healing.

Citing a study by Bielecki et al., J. Alsousou et al. discussed the application of platelet-rich plasma in orthopaedic surgery and showed that 13 out of 20 non-unions showed complete union after PRP injection. 45 The same publication states that no growth factors were found in non-union in a study looking at the levels of growth factors in fracture hematomas. In the same issue, Kitoh et al. published a study on distraction osteogenesis. It was found that the formation of calluses occurs between 34 and 47 days.

In his study on platelet rich concentrate, Samir Mehta made an observation about the application of PRP in nonunion. He claims that if there is adequate bone approach and the gap is not nonunion, PRP can help with bone healing. In his study on platelet-richrich concentrate, Samir Mehta made a remark about the application of PRP in nonunion. He claims that if there is adequate bone approach and the gap is not nonunion, PRP can help with bone healing.

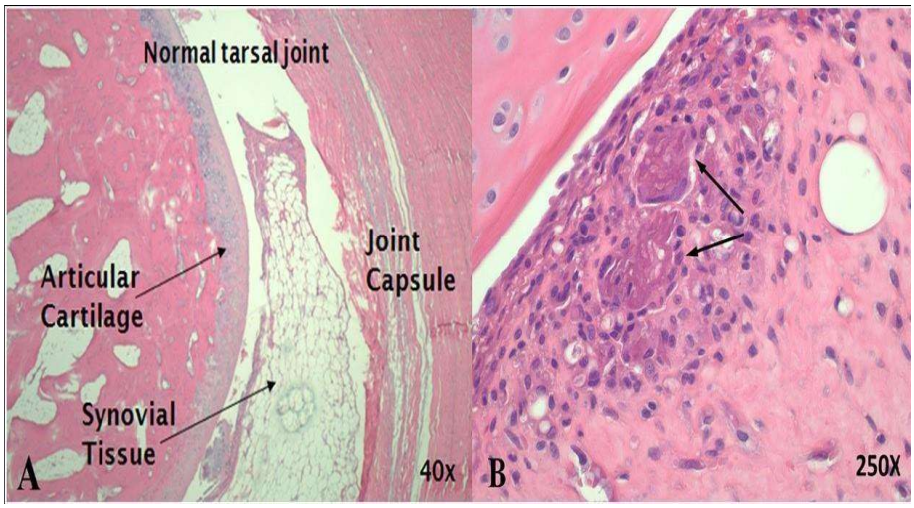
JOINT

N Lindsay Harris used a normal saline injection as a control while administering PRP to a healthy tarsal joint in rabbits for his investigation. Histological alterations that appeared to be calcification in subcutaneous fat at four weeks' time were among the synovitis symptoms he found in all of the samples, along with nodules in one sample at two weeks, as shown in the figure below. Every specimen displays normal outcomes identical to those of a saline specimen at six and twelve weeks.

SAFETY OF PLATELET-RICH PLASMA

In his study on platelet-rich concentrate, Samir Mehta talked about the safety of autologous concentrate. He asserted that there is no possibility of infectious diseases spreading because it is manufactured from the patient's own blood. He also talked about how people with coagulopathy and sensitivity to substances like bovine thrombin cannot benefit from platelet-rich plasma..

In his study comparing the effectiveness of PRP and steroid injections for treating plantar fasciitis, Ertugrul Aksahin discovered that PRP was recommended over steroid injection since it can avoid problems like fat necrosis. Joost C. Peer booms et al.'s study on PRP injection for tennis elbow reported no systemic or local side effects, with the exception of increased discomfort in the first few days after injection due to an inflammatory process. The effects of autologous PRP on the growth of methicillin-sensitive Staph aureus and pseudomonas aeruginosa were investigated by Bielecki et al.



METHODS

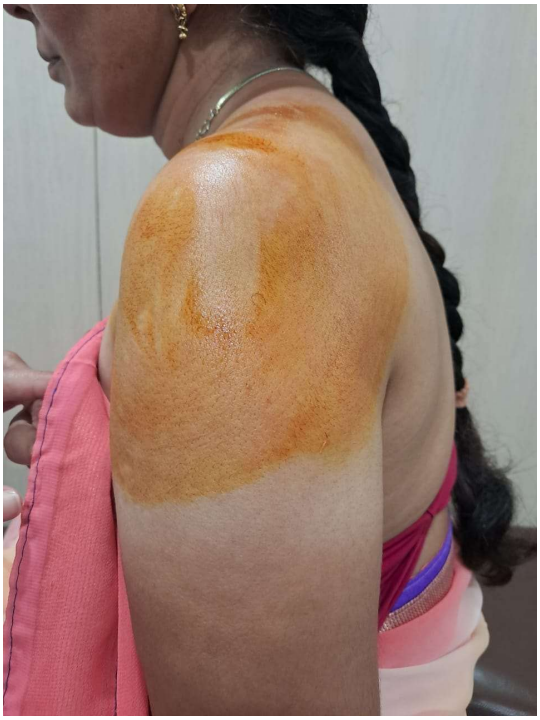
Freshly produced autologous PRP was used to treat 40 patients with partial thickness rotator cuff tear. The study's informed consent was provided by every patient, and it received institutional ethical committee approval.

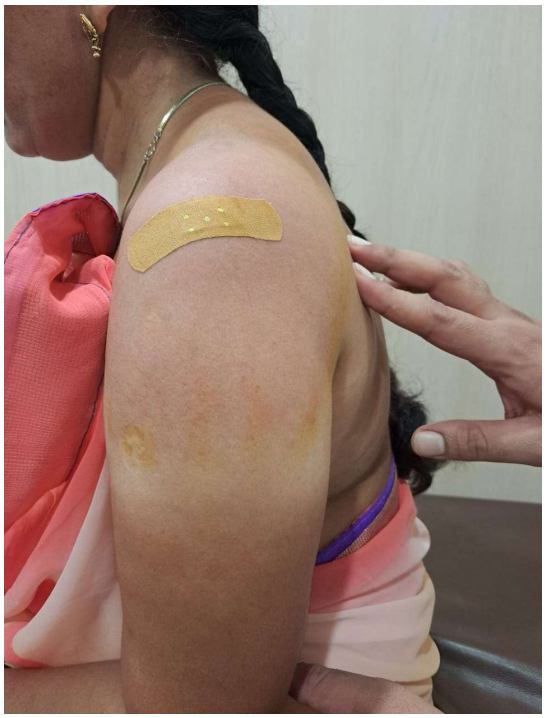
In order to meet the inclusion criteria, all patients underwent a plain x-ray of the shoulder joint and MRI shoulder joint from the side as well as basic investigations such haemoglobin, random blood sugar, lipid profile, and renal profile.In order to avoid sample clotting and platelet activation before usage, a sample of 18 cc venous blood was drawn from the patient's cubital vein and mixed with 2 cc of the anticoagulant Acid citrate dextrose solution (ACD). Soft and strong spins were used in the double spin approach here. To minimise mechanical harm to the platelets, this sample was next centrifuged at rev / min for 12 minutes using a soft spin technique. Additionally, the upper layer and intermediate layer, which contain few RBCs, are moved to a sterile container before being centrifuged hard for 10 minutes at 3000 rpm. The lower third of the plasma was removed, along with platelet pellets, and transferred to an injection syringe with an 18 gauge needle. The platelet-poor plasma was discarded. About 2.5 to 3cc of PRP are available for usage. This PRP is not activated or buffered. Nonsteroidal anti-inflammatory drugs use was not permitted during the first 2 weeks after treatment and was discouraged throughout the entire study period. No other treatment modalities were used during the study except exercises and footwear.

Physical examinations were performed, clinical symptoms and pain state were measured, and the results were compared to the pre-injection condition using interval VAS scoring. Assessment of pre- and post-injection state. periodically at the four weeks, six weeks and six months following therapy with indicated scores.

INJECTION TECHNIQUE

After confirming the diagnosis as partial thickness rotator cuff (clinically or radiologically or both), the patient is made to sit on the table with shoulder exposed. The afflicted shoulder is cleaned using a betadine scrub, which contains 7.5% povidone-iodine. After that, it was washed with spirit and painted with a 10% povidone-iodine betadine solution. While the patient was seated, PRP was injected into the bursal area and the crucial zone where the supraspinatus tendon inserts. Following therapy, patients were instructed to reduce their level of activity for a week and to take acetaminophenon and topical ice as needed. Patients were advised to resume regular activities after a week, but to keep changing their activities as prescribed before to therapy.



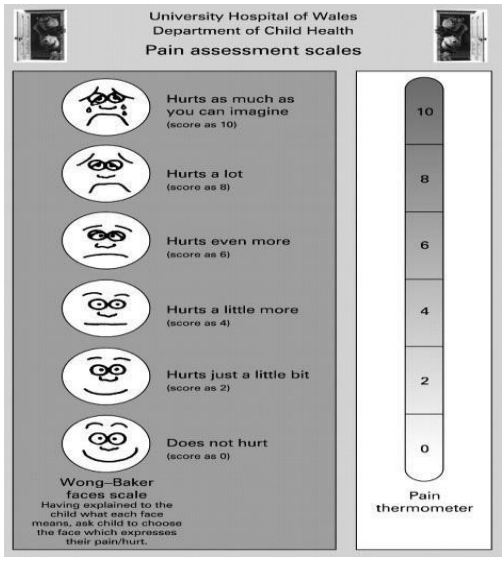


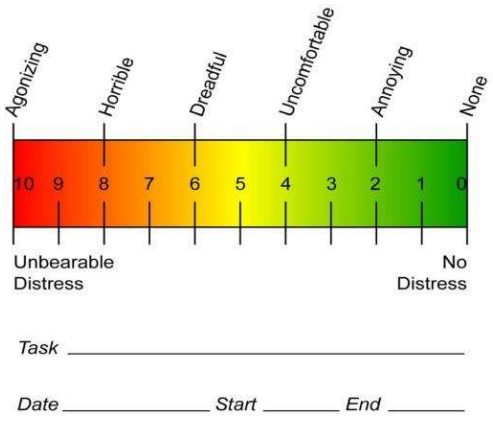
MAIN OUTCOME MEASUREMENTS

The study's results were computed using a visual analog scale scoring system. After the injection, the values are added at the fourth and sixth weeks. At six months of follow-up, the pain and activity level were used to calculate the final results.

VISUAL ANALOGUE SCALE

A patient's subjective rating of the perceived level of pain is called a numerical pain score. A score of 0 indicates no pain. The highest possible pain score is 10.





MATERIALS AND METHODS

1. SOURCE OF DATA

Individuals diagnosed with partial thickness rotational cuff tears are admitted to the orthopedic department at BLDE (DEEMED TO BE UNIVERSITY) Shri B.M. Patil's Medical College, Hospital and Research Centre, Vijayapura.

Complete information about the study will be given to the patients, and informed written consent will be sought.

The study will run from August 1, 2022, to May 31, 2024. There will be four, six, and eight weeks of follow-up.

2. METHOD OF COLLECTION OF DATA

Patients diagnosed with plantar fasciitis and admitted to the orthopedic department of BLDE (DEEMED TO BE UNIVERSITY) Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapura
By clinical examination.
History taking.

By Radiological examination – MRI OF SHOULDER JOINT

INCLUSION CRITERIA

1. Patients diagnosed with rotator cuff tear clinically, radiologically or both

2.. Patients with no history of any local steroids injections in past 2 months

3. Age of more than 18 years.

EXCLUSION CRITERIA

1.. Patients without any trial of conservative treatment

2. Infection or ulcer at the injection site

3. Pregnant ladies

4. Uncontrolled diabetes mellitus

SAMPLING

The study would need a sample size of 40 patients with a 95% level of confidence and 10% absolute precision due to the expected proportion of moderate limitation of activity (post surgery) among patients with partial thickness rotator cuff tears, which is 0.08% (ref).

Formula used

n= z2 p*q d2

Where,

Z = Z statistic at α level of significance

d2 = Absolute error

P = Proportion rate

q = 100-p

Statistical analysis:

The data obtained will be entered in a Microsoft Excel sheet, and statistical analysis will be performed using statistical package for the social sciences (Verson 20).

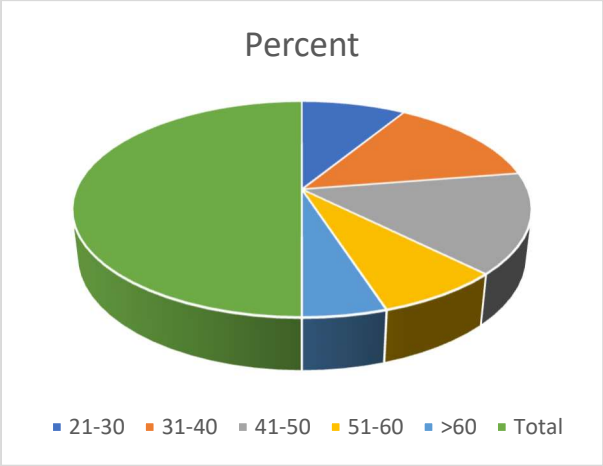
Results will be presented as Mean (Median) ±SD, counts and percentages and diagrams.

RESULTS

The orthopedics department at BLDE Hospital and Research Center monitored all 40 patients who underwent PRP injection for partial thickness rotator cuff tears between August 1, 2022, and May 31, 2024.

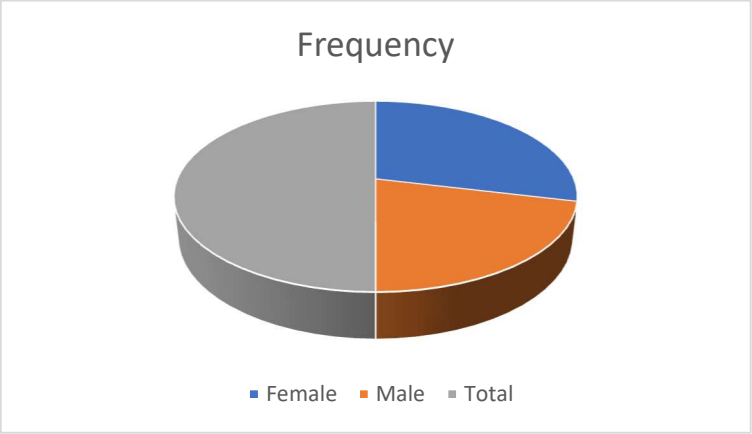
AGE DISTRIBUTION

Age	Percent
21-30	17.5
31-40	27.5
41-50	30
51-60	15
>60	10
Total	100



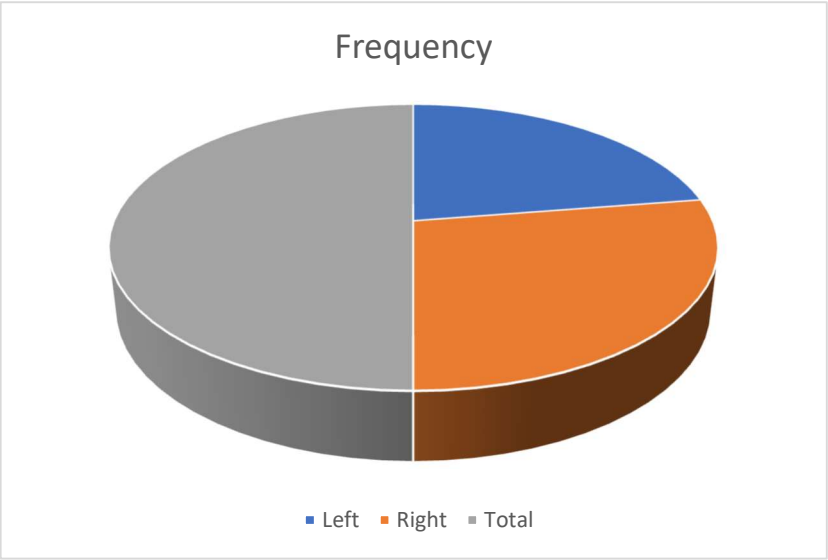
SEX DISTRIBUTION

Gender	Frequency	Percent
Female	23	57.5
Male	17	42.5
Total	40	100



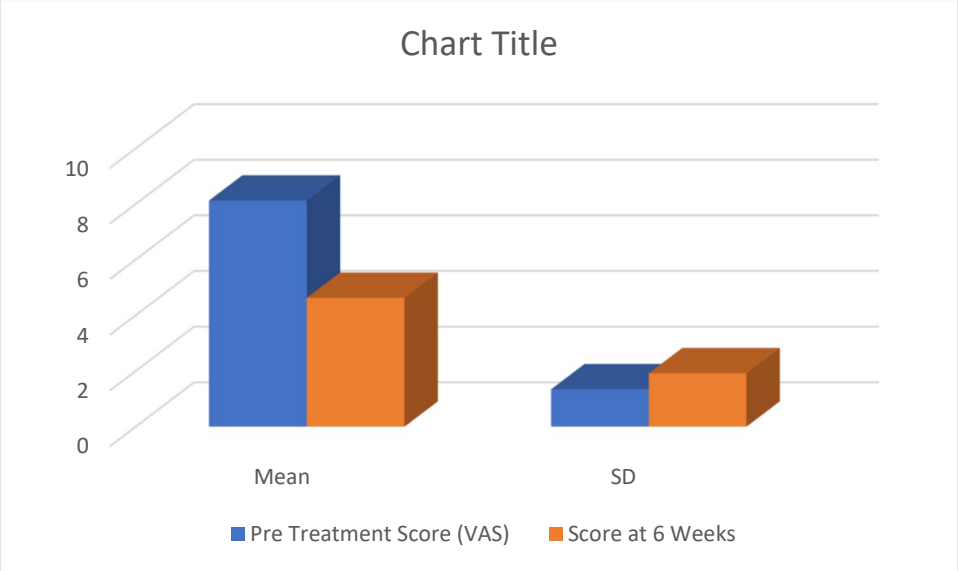
SIDE DISTRIBUTION

side affected	Frequency	Percent
Left	18	45
Right	22	55
Total	40	100

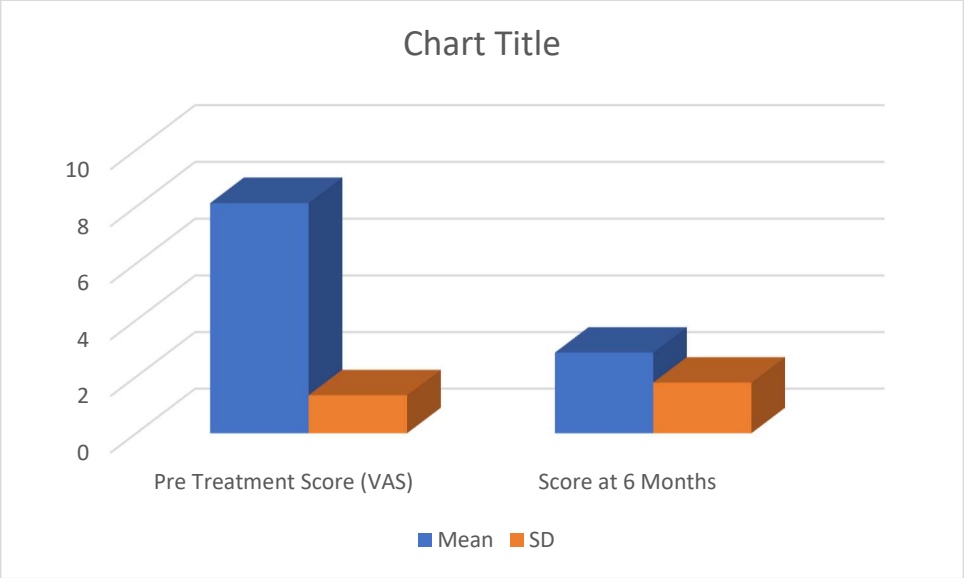


STATISTICS FINAL OUTCOME

	N	Mean	Mean Difference	%	SD	Wilcoxon signed-rank test	P
Pre Treatment Score (VAS)	40	8.125			1.343	820	< .001
Score at 6 Weeks	40	4.625	3.5	43	1.917		



	N	Mean	Mean Difference	%	SD	Wilcoxon signed-rank test	P
Pre Treatment Score (VAS)	40	8.125			1.343	0.953	0.098
Score at 6 Months	40	2.85	5.2	64	1.791		



DISCUSSION

A frequent illness of the shoulder, rotator cuff disease includes a wide range of issues, including partial-thickness rotator cuff tears (PTRCT). Because there is a significant link between rotator cuff illness and age, the estimated prevalence of PTRCTs is between 13% and 37%, and it is anticipated that this prevalence will rise while the population ages.

Many factors, such as the patient's age, symptoms, functional loss, tear location and size, etiology, daily activities, and the kind of origin (traumatic versus degenerative), all affect the best course of treatment for PTRCTs. A conservative treatment trial, such as activity modification with avoidance of overhead or pain-provoking activities, non-steroidal inflammatory drugs (NSAIDs), pain medications, physiotherapy, and steroid injection, is reasonable in most cases because the risk of fatty infiltration, muscular atrophy, and significant tear progression is relatively low compared to FTRCTs.

In a research by Denkers et al., 38 out of 76 consecutive PTRCT patients received conservative care and were monitored. Ninety-one percent of the patients were happy with the conservative care after an average of four years. Individuals with non-dominant hand atraumatic tears involving less than 50% of the tendon thickness were more likely to get conservative care.

Because PRP formulations were so effective at treating rotator cuff tear and they were also used to treat severe cases of tendinopathies.

CONCLUSION

The results of this study indicate that improvements in VAS scores for non operative partial thickness rotator cuff tear showed medically and statistically significant improvements with both shoulder functions and pain relief , especially at later stages 6 months compared to 4 weeks after treatment. The study found that a safe and efficient treatment for partial thickness rotator cuff tears is local PRP injection.

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ANNEXURE I

Ethical committee certificate

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 729/2022-23 30/8/2022

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "Functional outcome of platelet rich plasma in partial thickness rotator cuff tear" -a prospective study".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR NIVETHAN R

NAME OF THE GUIDE Dr.S.S.Nandi, Dept. of Orthopedics. .

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE II

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned,_____ , S/O D/O W/O _____, aged ____years, ordinarily resident of _____ do hereby state/declare that Nivethan.R of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on _____ at _____ (place) and it has been explained to me in my own language that I am suffering from _____ disease (condition) and this disease/condition mimic following diseases. Further Nivethan.R informed me that he/she is conducting dissertation/research titled **"FUNCTIONAL OUTCOME OF PLATELET RICH PLASMA IN PARTIAL THICKNESS ROTATOR CUFF TEARS - A PROSPECTIVE STUDY."** under the guidance of Dr. S.S. Nandi requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative, post-operative and follow-up observations will be utilized for the study as reference data.

The doctor has also informed me that during the conduct of this procedure, adverse results may be encountered. Most of them are treatable but are not anticipated hence there is a chance of aggravation of my condition and in rare circumstances, it may prove fatal despite anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study help in the evaluation of the results of the study which is a useful reference to the treatment of other similar cases in the near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made/ photographs/ video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during the course of treatment/study related to diagnosis, the procedure of treatment, result of treatment, or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and followup unless I request to be discharged. After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Shri/Smt _____ under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of the patient:

Signature of doctor:

Witness: 1.

2.

Date :

Place :

sANNEXURE – III

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

PROFORMA

CASE NO. :
FOLLOWUP NO. :
NAME :
AGE/SEX :
OPD NO :
DATE OF PROCEDURE :
DATE OF REVIEW :
OCCUPATION :
RESIDENCE :

Presenting complaints:

a. History of pain:
b. Duration of pain
c. Nature of pain
d. History of past medical disease
    - Diabetes mellitus
    - Hypertension /IHD
    - COPD
    - Pulmonary tuberculosis
    - Epilepsy
e. Personal and family history.

Systemic Examination

CVS :
RS :
CNS:
P/A :

General physical examination

1. Built and nourishment
2. Signs
3. Pallor, Icterus, clubbing, cyanosis, oedema, lymphadenopathy

Vital parameters:

4. Pulse :
5. BP:
6. Respiratory rate:

Pupillary reaction and Glasgow coma scale(GCS)

Local Examination

- Inspection
- Palpation
- Temperature
- Tendernes
- Abnormal mobility

(a) Measurements

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MASTER CHART

S.NO	Pt Name	Age	Gender	OPD No	Side affected	Pre Treatment Score (VAS)	Score at 4 weeks	Score at 6 Weeks	Score at 6 Months
1	Wadiyar	56	Male	48825	Right	8	6	5	2
2	Mohantabai	45	Female	230360	Right	6	5	5	2
3	Anusaya	34	Female	390096	Left	8	6	4	3
4	Ashok	42	Male	176982	Right	10	8	6	5
5	Pavadeppa	47	Male	192093	Left	8	6	5	3
6	Rajesh sharma	50	Male	275345	Left	6	5	3	1
7	Neelamma	58	Female	244037	Right	10	8	7	4
8	Sagar	38	Male	208735	Right	8	7	5	4
9	Ravindran ghadhar	46	Male	230387	Right	7	5	3	1
10	Rathabai	60	Female	162501	Left	9	7	6	4
11	Seetabai chavan	72	Female	387820	Left	6	5	2	1
12	Laxmibai	30	Female	171339	Right	10	8	7	3
13	Bajarao	70	Male	95597	Left	10	10	7	6
14	Karan	35	Male	140890	Left	10	8	7	5
15	Bhuvaneshwari	21	Female	135780	Right	9	7	4	2
16	Basangouda	63	Male	106382	Right	7	6	3	2
17	Sunitabai	29	Female	185790	Right	7	7	5	3
18	Meentab	48	Male	202775	Left	9	8	7	5
19	Pikalabai	46	Female	143721	Right	10	7	6	4
20	Arawind	25	Male	123888	Right	8	7	5	3
21	Maya	28	Female	132010	Left	9	6	4	2
22	Nayamma	35	Female	179703	Left	5	5	2	1
23	Tarabai	35	Female	32321	Left	9	7	7	5
24	Gouramma	31	Female	402909	Right	9	7	5	2
25	Srinidhi	28	Female	136370	Right	8	5	3	2
26	Heerappa	48	Male	146763	Left	9	8	7	7
27	Siddangouda	50	Male	131412	Right	8	8	7	6
28	Rukhmabai	72	Female	176743	Left	9	-	-	-
29	Heena	47	Female	176853	Right	7	5	3	1
30	Prathiba	33	Female	104595	Left	6	5	2	0
31	Murugappa	58	Male	49786	Left	9	8	6	4
32	Veena	50	Female	177301	Right	9	8	6	5
33	Dinesh	39	Female	185786	Right	6	7	4	2
34	Jawa	31	Female	129643	Left	8	6	-	-
35	Mallangouda	35	Male	176432	Right	8	6	4	2
36	Ambika	29	Female	59499	Right	7	6	5	4
37	Kamalabai	42	Female	157635	Right	9	7	6	2
38	Fathima	60	Female	151886	Right	9	7	6	4
39	Siddharay	57	Male	104932	Left	8	5	3	1
40	Santhosh	37	Male	166431	Left	7	6	3	1