

**“A STUDY OF EFFICACY WITH LOCAL METHYL
PREDNISOLONE ACETATE INJECTION VERSUS AUTOLOGOUS
BLOOD INJECTION IN THE TREATMENT OF LATERAL
EPICONDYLITIS”**

By

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IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE DEGREE
OF
MASTER OF SURGERY
IN
ORTHOPAEDICS

Under the guidance of

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APRIL 2014

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ABSTRACT

Background & Objectives

Lateral epicondylitis, is a common problem encountered in the orthopaedic practice. It is a common practice to give local corticosteroid infiltration for tennis elbow. Histopathological reports have shown that lateral epicondylitis is not an inflammatory process but a degenerative condition termed 'tendinosis'. Beneficial effects of local corticosteroid infiltration have sound lack of scientific rationale, since surgical specimens show lack of any inflammatory process. In recent studies no statistically significant or clinically relevant results in favor of corticosteroid injections were found. Recently an injection of "autologous blood injection" has been reported to be effective for both intermediate and long term outcomes for the treatment of lateral epicondylitis. It is hypothesized that blood contains platelet derived growth factor induce fibroblastic mitosis and chemotactic polypeptides such as transforming growth factor cause fibroblasts to migrate and specialize and have been found to induce healing cascade. The objective of the study is to compare the functional outcome of local Methyl prednisolone acetate injection versus Autologous Blood injection in lateral epicondylitis.

Materials and Methods

A prospective, randomized study was done in R L Jalappa General Hospital. 60 patients were included in the study. 30 patients received 2 milliliter autologous blood and 30 patients received 2 milliliters local corticosteroid (Methyl prednisolone acetate 80 mg) at the lateral epicondyle.

Outcome is measured using 'Pain score'. Student t test is applied to calculate the significance of results.

Results

Follow-up done for total 6 months divided in to intervals at 2week,2 month and 6 month. At 2nd week the corticosteroid injection group showed a statistically significant decrease in pain compared to autologous blood injection group. At 2nd month and 6 months follow up autologous blood injection group showed statistically significant decrease in pain compared to corticosteroid injection group. At the end of 6 months 46.66% patients in Corticosteroid injection group and 90% patients in autologous blood injection group were completely relieved of pain.

In Corticosteroid injection group till 2nd month there was significant improvement with 63.3% of patients completely relieved of pain. Many of these patients reported recurrences at 6th month follow up. The rate of recurrence was 36.8% at the end of 6 months.

In autologous blood injection group at 2nd month follow up, 16.66% of patients were completely free of pain. At the end of 6 months follow up, 90% of patients were completely free of pain. There was no recurrence.

Interpretation & Conclusion

Autologous blood injection technique for lateral epicondylitis offers a better treatment with least side effects, cost effective and with minimum recurrence rate.

Key words

Lateral epicondylitis; Local corticosteroid; Autologous blood injection

LIST OF ABBREVIATIONS

cms	centimeters
D	Dominant side
ERCB	Extensor carpi radialis brevis
ERCL	Extensor carpi radialis longus
F	Female
L	Left side elbow
M	Male
m	Manual
MRI	Magnetic Resonance Imaging
ND	Non dominant side
Nm	Non manual
NS	Non significant
R	Right side elbow
S	Significant
SD	Standard deviation
VAS	Visual Analog Scale

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INTRODUCTION

Lateral epicondylitis, or tennis elbow, is a commonly encountered problem in orthopedic practice. It has been found to be the second most frequently diagnosed musculoskeletal disorder in the neck and upper extremity in a primary care setting.¹

It has an incidence of 4-7 per 1000 per year in general practice, with a peak between the ages of 35 and 54 years, with a mean age of approximately 42 years.^{2,3} Tennis elbow (Lateral epicondylitis) designates a pattern of pain at the origin either of the extensors of the fingers and wrist on the lateral epicondyle of humerus. It is a common problem in office orthopaedics, and is reported to be four times as common in the fourth decade of life as in any other decade. It is said to involve the lateral epicondyle seven times more frequent than the medial. Its name “Tennis elbow” is a misnomer because it occurs more commonly in non athletes than in tennis players . The most widely held theory is that there are macroscopic or microscopic tears in the common tendon as described by Cyriax⁴

Much controversy has been there over the pathophysiology and there is not enough scientific evidence to favour any particular type of treatment for acute lateral epicondylitis⁵. Currently degeneration of the origin of the extensor carpi radialis brevis (ECRB), repeated micro trauma and incomplete healing response has been accepted as the cause of lateral epicondylitis by most of the researchers.⁶

Histopathological reports have shown that lateral epicondylitis is not an inflammatory process but a degenerative condition termed ‘tendinosis’.^{5,6} There are numerous treatment modalities for lateral epicondylitis both conservative and operative. Most conservative modalities such as local corticosteroid injection have focused on suppressing inflammatory process that does not actually exist. A recent

review article concluded that for short term outcomes (6 weeks), statistically significant and clinically relevant differences were found on pain and global improvement with corticosteroid injection compared to placebo, local anaesthetic, or other conservative treatments.¹³ For intermediate (6 weeks to 6 months) and long term outcomes (more than 6 months), no statistically significant or clinically relevant results in favour of corticosteroid injections were found. So it is not possible to draw a firm conclusion on the effectiveness of corticosteroid injection.^{7,8}

Recently an injection of autologous blood has been reported to be effective for both intermediate and long term outcomes for the treatment of lateral epicondylitis. There was a significant decrease in pain.⁵ It is hypothesized that mitogens such as platelet derived growth factor induce fibroblastic mitosis and chemotactic polypeptides such as transforming growth factor cause fibroblasts to migrate and specialize and have been found to cause angiogenesis. A specific humoral mediator may promote the healing cascade in the treatment of tendinosis as well. These growth factors trigger stem cell recruitment, increase local vascularity and directly stimulate the production of collagen by tendon sheath fibroblasts.¹⁸

Autologous blood was selected as the medium for injection because (1) its application is minimally traumatic, (2) it has a reduced risk for immune-mediated rejection, devoid of potential complications such as hypoglycemia, skin atrophy, tendon tears associated with corticosteroid injection (3) it is simple to acquire and prepare, easy to carry out as outpatient procedure and (4) it is inexpensive⁵.

There are very few studies done to evaluate injection of autologous blood for lateral epicondylitis as treatment modality. Hence it is evaluated by comparing with

the corticosteroid injection which is a commonly practiced conservative treatment modality.⁵

The purpose of this dissertation is to evaluate the efficacy and role of autologous blood injection at lateral epicondylitis by comparing with local corticosteroid injection (methyl prednisolone acetate).

OBJECTIVE

To compare the functional outcome of local Methyl prednisolone acetate injection versus Autologous Blood injection in lateral epicondylitis.

REVIEW OF LITERATURE

Tennis elbow was first described in 1883 by Major as a condition causing lateral elbow pain in tennis players. Over the years, this term has become synonymous with all lateral elbow pain, despite the fact that the condition is most often work related and many patients who have this condition do not play tennis. The anatomic basis of the injury to extensor carpi radialis brevis origin appears to be multifaceted, involving hypovascular zones, eccentric tendon stresses and a macroscopic degenerative response¹⁰.

In 1936, a condition was described, the symptoms and signs of which are as constant as those of tennis elbow, may well be supposed to have but one pathology and, as a corollary, but one type of treatment. The pathology of tennis elbow (lateral epicondylitis) remains an enigma. 26 different pathological causes were documented and concluded that the evidence is overwhelmingly in favour of a typical tennis elbow being caused primarily by a tear between the tendinous origin of the extensor carpi radialis brevis and the periosteum of the anterior surface of the lateral epicondyle¹¹.

A study in 1955, concluded that the pathological changes in the annular ligament were causative of the symptoms of tennis elbow and suggested an ingenious operation (lengthening of the extensor carpi radialis brevis at the wrist) as a surgical cure.¹²

A study in 1961 reported that, with such clearly defined clinical and diagnostic features the unknown pathology of tennis elbow is surprising.¹³

In 1979, a study reporting on 88 surgical elbows noted the association in tennis and golf (in the non-dominant arm) with symptoms, but also pointed out that tennis elbow occurs in non-tennis players.¹⁴

In 1993 a study reporting on 63 surgical cases noted “The extensor origin was grossly normal in all but six patients and noted vascular proliferation in 46%, mucoid degeneration in 27% and no evidence of inflammatory reaction. Amorphous white steroid deposits were identified in five patients¹⁵.

Although originally described as inflammatory process much controversy has been there over the pathophysiology and treatment of this disorder. Different entities have been proposed as its etiology which included bursitis, perostitis, infection, aseptic necrosis and neuritis of branches of the radial nerve or of the dorsal antebrachial cutaneous nerve, radiohumeral synovitis with irritation of a synovial fringe, irritation of the collateral ligament or the annular ligament, and so forth. The most widely held theory is that there are macroscopic or microscopic tears in the common tendon.^{16,11.}

Now the consensus is that lateral epicondylitis is initiated as a micro tear most often within the origin of extensor carpi radialis brevis. Microscopic finding demonstrate immature reparative tissue that resemble angiofibroblastic hyperplasia.¹⁴

Relevant Anatomy:

Elbow joint is a synovial joint with ginglymus or hinge-joint type. Its complexity is increased by continuity with the superior radio-ulnar joint. It includes two articulations. These are the humero-ulnar, between trochlea and humerus and the ulnar trochlear notch, and the humero-radial, between the capitulum of the humerus and the radial head.¹⁷

Elbow Joint [Opened] Anterior and Posterior Views

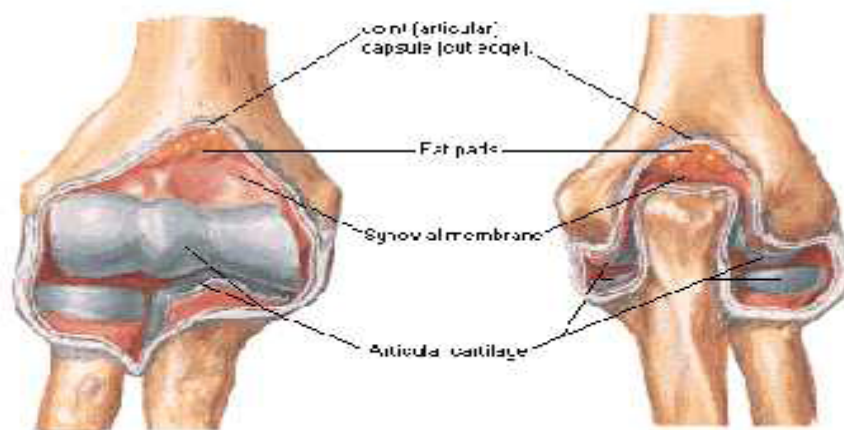


Fig. 1 : Elbow Joint [Opened]

Anterior and Posterior views

Articulating surfaces- The articulating surfaces are the humeral trochlea and capitulum, and the ulnar notch and radial head. The trochlea is not a simple pulley because its medial flange exceeds its lateral, thus projecting to a lower level. This means that the plane of the joint is 2 cm distal to the inter-epicondylar line, which is located inferomedially. The trochlea is also widest posteriorly and here its lateral edge is sharp. The trochlear notch is not wholly congruent with it. In full extension the medial part of its upper half is not in contact with the trochlea and a corresponding

lateral strip loses contact in flexion. The trochlea has an asymmetrical surface, largely concave transversely, convex anteroposteriorly.

Since the humero-ulnar and humero-radial articulations form a largely uniaxial joint, the ligaments are capsular ligaments, ulnar and radial collateral ligaments.¹⁷

Fig. 2 : Ligaments of Elbow Right Elbow – Anterior View

The fibrous capsule is broad and thin anteriorly. It is attached proximally to the front of the medial epicondyle and humerus above the coronoid and radial fossae.

and distally to the edge of the ulnar coronoid process and anular ligament, and is continuous at its sides with the ulnar and radial collateral ligament. Anteriorly it receives numerous fibers from brachialis.

Posteriorly the capsule is thin and attached to the humerus behind its capitulum and near its lateral trochlear margin, to all but the lower part of the edge of the olecranon fossa, and to the back of the medial epicondyle. Inferomedially it reaches the superior and lateral margins of the olecranon and is laterally continuous with the superior radio-ulnar capsule deep to the annular ligament. It is related posteriorly to the tendon of triceps and to the anconeus.¹⁷

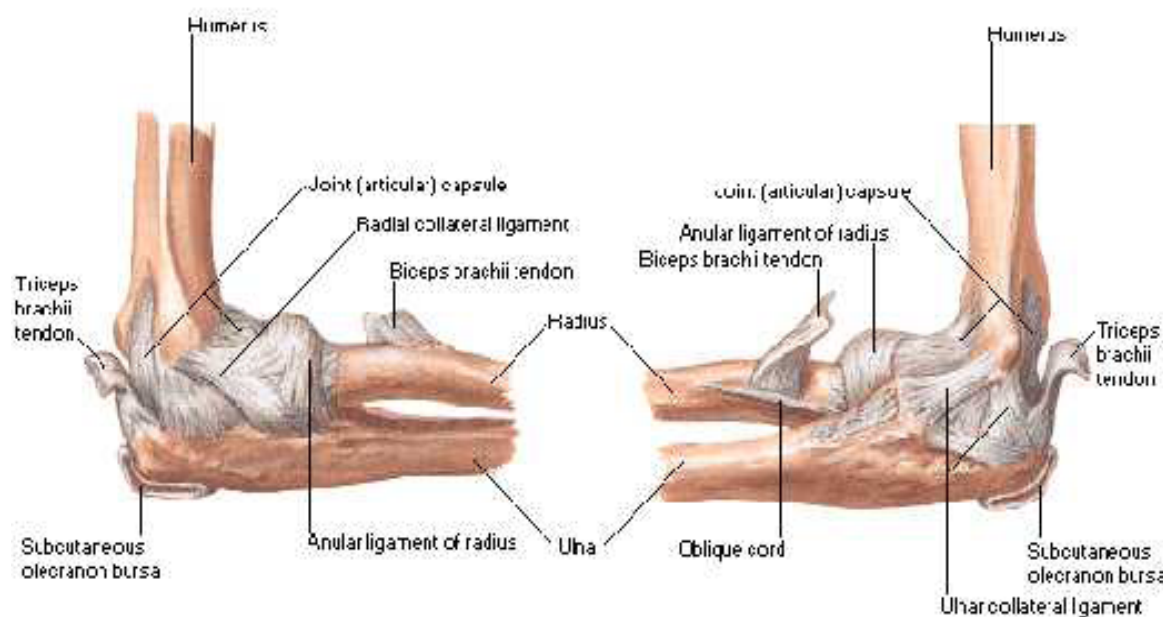


Fig. 3 : Ligaments of Elbow in 90°

Lateral and Medial Views

Ulnar collateral ligament (ligamentum collaterale ulnare; internal lateral ligament)

This ligament is a thick triangular band consisting of two portions, an anterior and posterior united by a thinner intermediate portion. The anterior portion directed obliquely forward is attached above by its apex to the front part of the medial epicondyle of the humerus and below by its broad base to the medial margin of the coronoid process. The posterior portion also of triangular form is attached above by its apex to the lower and back part of the medial epicondyle below to the medial margin of the olecranon. Between these two bands a few intermediate fibers descend from the medial epicondyle to blend with a transverse band which bridges across the notch between the olecranon and the coronoid process. This ligament is in relation with the Triceps brachii and Flexor carpi ulnaris and the Ulnar nerve, and gives origin to part of the Flexor digitorum sublimis.¹⁷

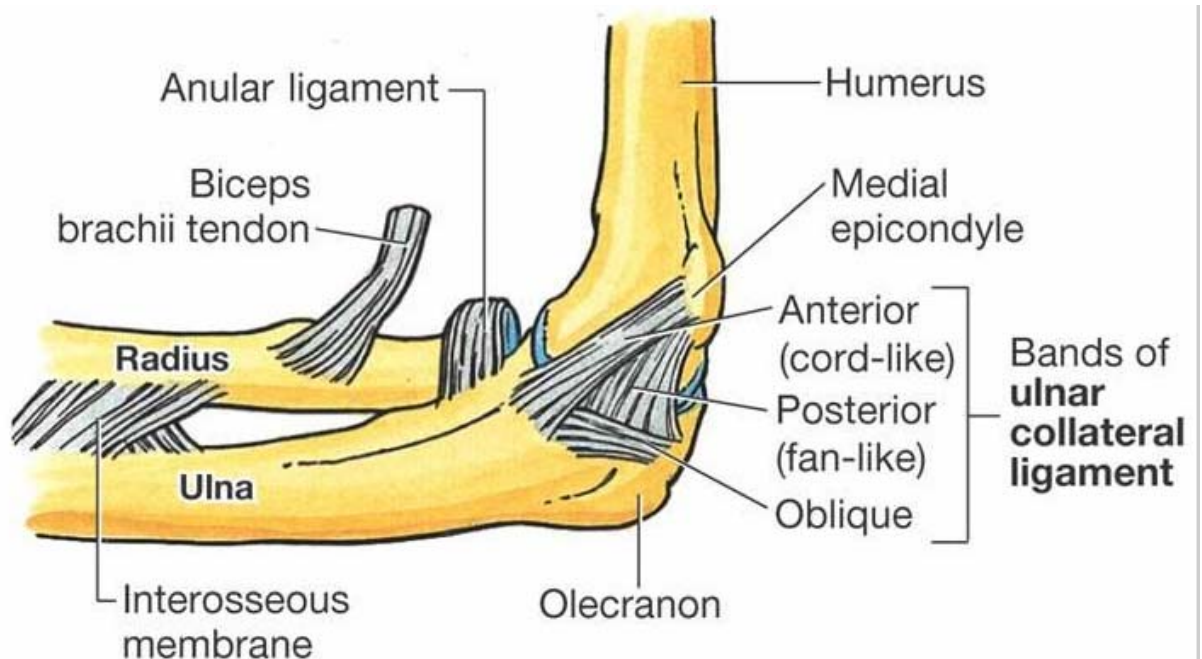


Fig (4) Ulnar collateral ligament

The lateral collateral ligament complex

It consists of the annular ligament, the radial collateral ligament, the lateral ulnar collateral ligament. The radial collateral ligament originates from the lateral epicondyle and terminates indistinguishably in the annular ligament, which stabilizes the proximal radioulnar joint.

The Annular Ligament

The annular ligament wraps around the head of the radius and attaches to the ulna. It stabilizes the radius in the radial notch and allows for movements that involve rotating the hand, such as turning a screwdriver, bowling or hurling.

The Radial Collateral Ligament (ligamentum collaterale radiale; external lateral ligament)

This ligament is a short and narrow fibrous band, less distinct than the ulnar collateral, attached above to a depression below the lateral epicondyle of the humerus, below to the annular ligament. Some of its most posterior fibers inserted into the lateral margin of the ulna. It is intimately blended with the tendon of origin of the Supinator.¹⁷

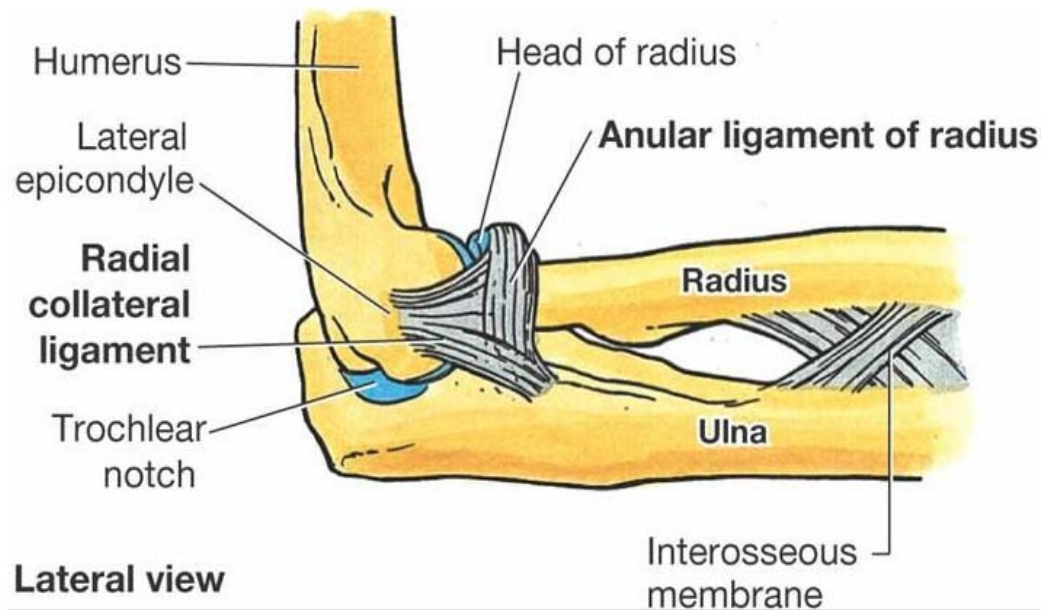


Fig (5) The lateral collateral ligament complex

The lateral ulnar collateral ligament

The lateral ulnar collateral ligament originates from the lateral epicondyle, blending with fibers from the annular ligament but arching superficial and distal to it. This ligament is the primary restraint to varus stress and is deficient in posterolateral rotatory instability of the joint.



Fig(6) Lateral Ulnar collateral ligament

Synovial Membrane

The synovial membrane is very extensive. It extends from the margin of the articular surface of the humerus, and lines the coronoid, radial and olecranon fossæ on that bone.

It is reflected over the deep surface of the capsule and forms a pouch between the radial notch, the deep surface of the annular ligament, and the circumference of the head of the radius. Projecting between the radius and ulna into the cavity is a crescentic fold of synovial membrane, suggesting the division of the joint into two; one the humeroradial, the other the humeroulnar.

Between the capsule and the synovial membrane are three masses of fat: the largest, over the olecranon fossa, is pressed into the fossa by the Triceps brachii during the flexion, the second over the coronoid fossa, and the third over the radial fossa, are pressed by the Brachialis into their respective fossæ during extension.

The muscles in relation with the joint are in front the Brachialis, behind the Triceps brachii and Anconeus, laterally the Supinator and the common tendon of origin of the Extensor muscles, medially the common tendon of origin of the Flexor muscles and the Flexor carpi ulnaris.

The arteries supplying the joint are derived from the anastomosis between the profunda and the superior and inferior ulnar collateral branches of the brachial, with the anterior, posterior, and interosseous recurrent branches of the ulnar, and the recurrent branch of the radial. These vessels form a complete anastomotic network around the joint.

The nerves of the joint are a twig from the Ulnar nerve, as it passes between the medial condyle and the olecranon; a filament from the Musculocutaneous nerve and two from the Median nerve.¹⁷

Movements

The elbow-joint comprises three different portions—viz., the joint between the ulna and humerus, that between the head of the radius and the humerus, and the proximal radioulnar articulation, described below. All these articular surfaces are enveloped by a common synovial membrane, and the movements of the whole joint should be studied together. The combination of the movements of flexion and extension of the forearm, pronation and supination of the hand, which are being performed at the same joint, is essential to the accuracy of the various minute movements of the hand.

The portion of the joint between the ulna and humerus is a simple hinge-joint, and allows movements of flexion and extension only. Owing to the obliquity of the trochlea of the humerus, this movement does not take place in the antero-posterior plane of the body of the humerus. When the forearm is extended and supinated, the axis of the arm and forearm are not in the same line; the arm forms an obtuse angle with the forearm, the hand and forearm being directed lateral-ward. During flexion, however, the forearm and the hand tend to approach the middle line of the body, and thus enable the hand to be easily carried to the face. The accurate adaptation of the trochlea of the humerus, with its prominences and depressions, to the semilunar notch of the ulna, prevents any lateral movement.

Flexion is produced by the action of the Biceps brachii and Brachialis, assisted by the Brachioradialis and the muscles arising from the medial condyle of the humerus; extension, by the Triceps brachii and Anconeus, assisted by the Extensors of the wrist, the Extensor digitorum communis, and the Extensor digiti quinti proprius.

The joint between the head of the radius and the capitulum of the humerus is an arthrodial joint. The bony surfaces constitute an enarthrosis and allow of movement in all directions, were it not for the annular ligament, by which the head of the radius is bound to the radial notch of the ulna, and which prevents any separation of the two bones laterally. It is to the same ligament that the head of the radius owes its security from dislocation, which would otherwise tend to occur, from the shallowness of the cup-like surface on the head of the radius. In fact, but for this ligament, the tendon of the Biceps brachii would be liable to pull the head of the radius out of the joint. The head of the radius is not in complete contact with the capitulum of the humerus in all positions of the joint. The capitulum occupies only the anterior and inferior surfaces of the lower end of the humerus, so that in complete extension a part of the radial head can be plainly felt projecting at the back of the articulation. In full flexion the movement of the radial head is hampered by the compression of the surrounding soft parts, so that the free rotator movement of the radius on the humerus (pronation and supination) takes place in semiflexion, in which position the two articular surfaces are in most intimate contact.

Flexion and extension of the elbow-joint are limited by the tension of the structures on the front and back of the joint; the limitation of flexion is also aided by the soft structures of the arm and forearm coming into contact.

In any position of flexion or extension, the radius, carrying the hand with it, can be rotated in the proximal radioulnar joint. The hand is directly articulated to the lower surface of the radius only, and the ulnar notch on the lower end of the radius travels around the lower end of the ulna. The latter bone is excluded from the wrist-joint by the articular disk. Thus, rotation of the head of the radius around an axis

passing through the center of the radial head of the humerus imparts circular movement to the hand through a very considerable arc.¹⁷



Fig (7) Extensor carpi radialis brevis muscle

The extensor carpi radialis brevis (ECRB) muscle arises from the lateral epicondyle. The ECRB muscle lies deep to the extensor carpi radialis longus (ECRL) muscle and superficial to the joint capsule. The annular and collateral ligaments are located beneath and just distal to the origin of the ECRB muscle. The tendinous origin of the muscle is described, as an entheses.¹⁸

Enthesis is a specialized junction of a ligament, tendon and bone. Hence tennis elbow is termed as an enthesopathy affecting the common extensor origin from the lateral epicondyle of humerus.¹⁸

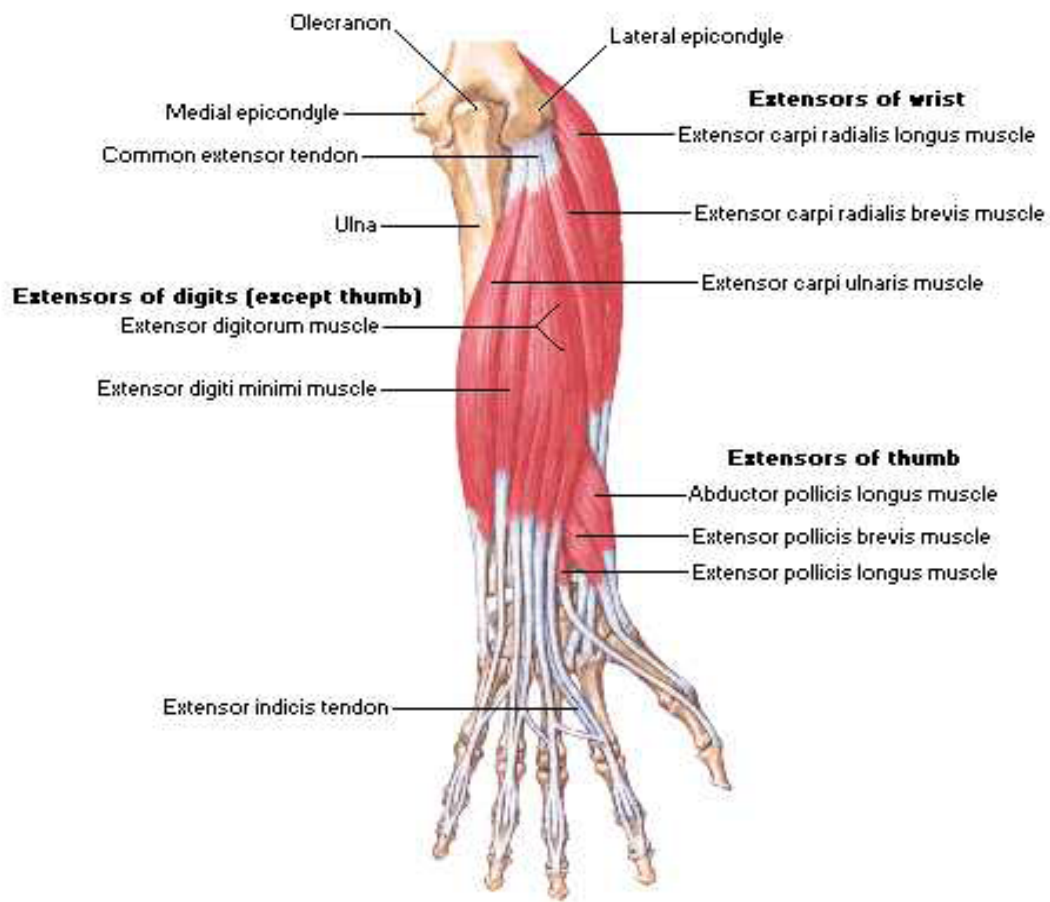


Fig (8) : Common extensor origin from lateral epicondyle

Extensor carpi radialis brevis is the most common muscle to be involved. Cadaveric studies proved that in majority of the population, there is an avascular area in the posterior aspect in the origin of extensor carpi radialis brevis. Although tennis elbow commonly involves the origin of the extensor carpi radialis brevis, the origin of

extensor digitorum communis, the extensor carpi radialis longus and the supinator can be involved in the process.⁶ A cadaveric study concluded that, it is clear that even under most controlled situation as in a cadaver dissection it was not possible to separate the origin of extensor carpi radialis brevis from the common extensor tendon. At times the tendon appear to interdigitate.¹⁶

Biomechanics

Tennis is the most common sport to cause lateral epicondylitis, but the condition can also be seen in those who play squash and badminton.

Symptoms can occur after an improper backhand hitting technique, which can occur when the athlete attempts to increase power by increasing forearm force rather than relying on core, rotator cuff, and scapular power. This results in snapping the wrist with supination and irritation of the extensor tendons. Symptoms can also occur when an athlete does not get his or her feet into position and hits the ball late or with a bent elbow. The power of the hit is again generated from the forearm instead of the core.

Other causes of extensor tendinopathy in tennis are using a new racquet, using a racquet that is strung too tightly, or using a racquet that is too heavy, as well as hitting wet or heavy balls or hitting into the wind. Another common racquet abnormality that causes lateral elbow extensor tendinosis is having a grip that is too large.

Industrial workers have certain occupational and leisure activities that lead to overuse injuries of the forearm wrist extensors, causing pain at the lateral epicondyle. These include carpenters, bricklayers, seamstresses and tailors, politicians (excessive handshaking), and musicians (e.g, pianists, drummers). Such injuries can also be seen

in individuals who perform a lot of computer work, a lot of typing, and a lot of mouse work for their occupations.

Pathophysiology:

Tendon injuries can be divided into several categories on the basis of the nature of their onset and the tissues involved.

- Acute tendon injuries, such as laceration of the flexor tendons of the fingers, are traumatic in nature.
- Chronic overuse injuries are the result of multiple micro traumatic events that cause disruption of the internal structure of the tendon and degeneration of the cells and matrix, which fail to mature into normal tendon; at times, such injuries result in 'tendinosis'.

Tendons involved in locomotion and ballistic performance, which transmit loads under elastic and eccentric conditions, are susceptible to injury. Some tendons, such as those that wrap around a convex surface or the apex of a concavity, those that cross two joints, those with areas of scant vascular supply, and those that are subjected to repetitive tension, are particularly vulnerable to overuse injuries.

Repetitive micro trauma results in further tendon degeneration. A chronic cycle of tendon degeneration and repair ensues with further weakening of the tendon with potential for rupture. Chronic overuse injuries are the result of multiple micro traumatic events that cause disruption of the internal structure of the tendon and degeneration of the cells and matrix, which fail to mature into normal tendon; at times, such injuries result in tendinosis.

It has been demonstrated that the early lesion is a hypoxic degeneration process rather than inflammatory, that can be normal part of aging or to response of

stress of overload or overuse. It has been also postulated that, an incomplete healing response characterized by vascular and fibrous proliferation occurs in the area of poor vascularity. A cyclically applied cumulative type of tendon injury is perceived by body's immune system as sub clinical, because of lack of the haemopoietic system; therefore the normal sequence of the inflammatory response is bypassed. Instead tendon intra-substance proliferates, leading to degeneration in the poorly vascularised area with histology showing cellular atrophy, diminished protein synthesis and cyst formation.⁴³ As the degenerated area enlarges the tendon weakens and eventually ruptures (micro rupture) which initiate the classic inflammatory response and healing cascade.

Tendinosis is incompletely understood. Although the term tendinitis is used frequently and often indiscriminately, histopathological studies have shown that specimens of tendon obtained from areas of chronic overuse do not contain large numbers of macrophages, lymphocytes, or neutrophils. Rather, tendinosis appears to be a degenerative process that is characterized by the presence of dense populations of fibroblasts, vascular hyperplasia, and disorganized collagen. Some authors have described tendinosis as a degenerative process and others have described it as a dysfunctional, immature tendon repair. This constellation of findings has been termed angiofibroblastic hyperplasia. Regardless of what it is called, tendinosis is the result of failed tendon-healing after repetitive microtrauma.⁶

Lateral epicondylitis, is a well-known example of an injury in such a region. It noted that the origin of the extensor carpi radialis brevis was the primary site of this injury, and pathological changes have been consistently documented at this location. One third of patients also have involvement of the origin of the extensor digitorum communis^{11,4,} Histopathological studies have demonstrated that tennis elbow is not

an inflammatory condition; rather, it is a fibroblastic and vascular response called angiofibroblastic degeneration, now more commonly known as tendinosis. Thus, the terms epicondylitis and tendinitis are misnomers⁶. Although it is commonly presumed that any painful structure is inflamed, connective-tissue pain can be perceived by the patient as the result of nociception and a noxious chemical environment.

The importance of distinguishing tendinosis from tendinitis is more than just a need for semantic accuracy. Proper treatment depends on a correct understanding of the nature of the injury and the goals of therapeutic intervention.

Gross examination of this region characteristically shows grayish, gelatinous, friable immature scar tissue that appears shiny and edematous⁶.

Microscopy-

The normal tendon shows parallel bundles of uniform-appearing collagen oriented along the long axis of the tendon. The matrix, which is composed primarily of proteoglycans, glycosaminoglycans, and water, is stained evenly. No vascular structures are apparent within the tendon.⁶

Tendinosis is characterized by hypertrophy of fibroblasts, abundant disorganized collagen, and vascular hyperplasia in what are, under normal circumstances, avascular tendon fascicles.

“Tendinosis” can be described as the disruption of normally orderly tendon fibers by a characteristic pattern of invasion by fibroblasts and atypical granulation tissue. It is also described as an immature reparative process set in a background of focal hyaline degeneration.⁶

Those authors termed the condition angiofibroblastic tendinosis because angiofibroblastic tissue was found to be insinuating itself through abnormal

hypercellular regions and extending focally into adjacent normal-appearing tendon fibers.

Electron microscopy

Electron microscopy of the fibroblasts revealed many vacuoles, open nuclear chromatin, abundant production of collagen along the periphery of the cells, and, interestingly, contractile elements within some of the fibroblasts as are seen with myofibroblasts, cells that are not native to tendon. Thus, we could identify two populations of fibroblasts: those with intracellular contractile elements and those without them, with the latter type having a more normal appearance. Both types of fibroblasts had lysosomes and fatty vacuolation, with abundant endoplasmic reticulum..⁶

The progressive stages with micro trauma in lateral epicondyle tendinosis are-

Stage-1 injury is probably inflammatory, is not associated with pathological alterations, and is likely to resolve

Stage-2 injury is associated with pathological alterations such as tendinosis, or angiofibroblastic degeneration

Stage-3 injury is associated with pathological changes (tendinosis) and complete structural failure (rupture)

Stage-4 injury exhibits the features of a stage-2 or 3 injury and is associated with other changes such as fibrosis, soft matrix calcification, and hard osseous calcification. The changes that are associated with a stage-4 injury also may be related to the use of cortisone. ^{5,6.}

In practice, it is the second stage (angiofibroblastic degeneration) that is most commonly associated with sports-related tendon injuries such as tennis elbow and with overuse injuries in general.

Clinical Presentation:

Patients present with complain of lateral elbow and forearm pain exacerbated by use. The typical patient is a man or woman aged 35-55 years who either is a recreational athlete or one who engages in rigorous daily activities.

Typically, the patient with lateral epicondylitis reports pain centered over the lateral epicondyle. The pain often extends into the dorsal forearm, it may extend proximally, and it is exacerbated by lifting, gripping, or repetitive wrist activity. The patient gives a history of pain that can be elicited with simple activities of daily living, such as lifting pots and pans or gripping a container of milk.

The single most important diagnostic finding is the location and reproducibility of pain.. Upon examination, the patient has a point of maximal tenderness just distal (5-10 mm) to the lateral epicondyle in the area of the ECRB muscle.²⁰

A number of clinical tests have been suggested for the evaluation of Tennis elbow, of which **Cozen's test** and **Mill's maneuver** are the commonest to be practiced.

Cozen's test: patient is made to make a firm fist. While the patient maintains the position, try to passively flex the wrist. Patient will feel pain at the lateral epicondylar region.

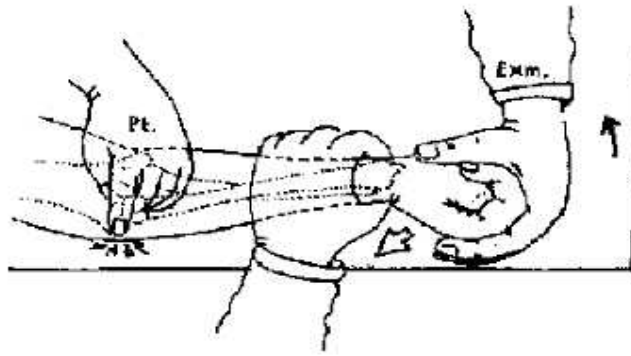


Fig. 9 : Cozen's test

Mill's maneuver: While the patient keeps his /her elbow firmly straight and wrist flexed pronation of the forearm initiates pain at the lateral epicondylar region.



Fig. 10 : Mill's maneuver

Other helpful test is the **chair raise test**. The patient stands behind their chair and attempts to raise it by putting their hands on the top of the chair back and lifting. In patients with lateral epicondylitis, pain results over the lateral elbow.

Chair test: The patient is asked to get up from a chair with both hands firmly gripping and pressings the arms of the chair. In a positive test pain is felt at the lateral epicondylar region of affected side.

Jug Test: Patient is asked to lift a jug full of water, holding the mouth from above, in a positive test pain will be felt at the lateral epicondylar region.

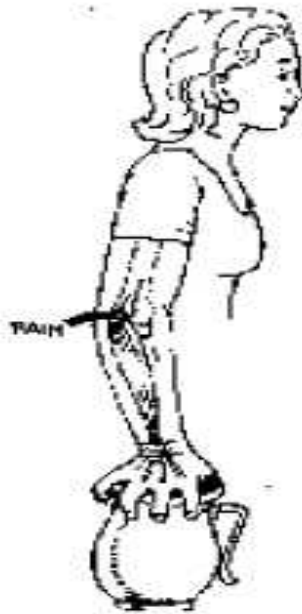


Fig. 11 : Jug test

Wringing Test: Patient is asked to wring a towel; pain is felt at the lateral epicondylar region in a positive test.

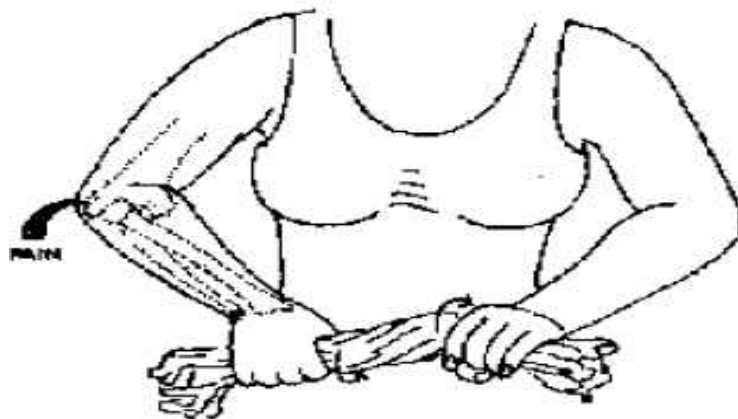


Fig. 12: Wringing Test

Diagnosis:

Diagnosis is based on clinical tests and further investigations are usually done only to rule out when other pathologies are suspected.

Imaging Studies: whenever necessary

- **Radiographs** can be helpful in ruling out other disorders or concomitant intraarticular pathology (e.g.: osteochondral loose body, posterior osteophytes).

Calcification in the degenerative tissue of the ECRB muscle origin can be seen in chronic cases.

- **Magnetic resonance imaging (MRI)** can help confirm the presence of degenerative tissue in the ECRB muscle origin and can help diagnose concomitant pathology; however, it is very rarely needed. On MRI the normal common extensor tendons are seen as smooth well-defined black structures of uniform thickness on all sequences. Tendinosis manifest by thickening and signal change. In the early stages, the tendon demonstrates poorly defined low to intermediate signal change on T1weighted images, with a relative increase in signal on T2 weighted images. On T2 weighted sequences with fat suppression or STIR imaging, the affected tendon returns high signal. In later stages, cystic change may occur, with focal areas of high signal seen within the tendon on T2 weighted images. This may be complicated by partial or complete tears of the tendon and be associated with collateral ligament derangement.



Fig. 13 : X-ray antero-posterior and lateral view of elbow

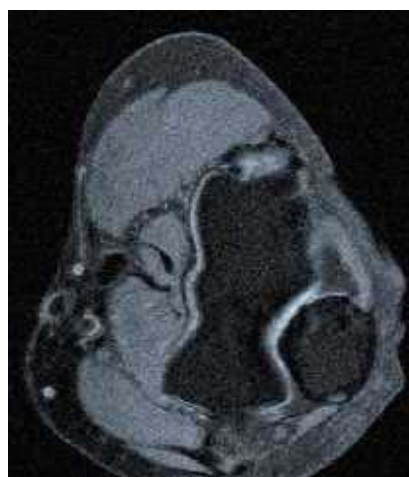


Fig. 14 : Axial T1 MRI Elbow



Fig 15 : Coronal



Fig 16: Coronal T2 fat suppression MRI Elbow

- **Ultrasonography** —namely calcification within the common extensor tendon, tendon thickening, adjacent bone irregularity, focal hypoechoic regions in the tendon, and diffuse tendon heterogeneity

Other Tests:

- If the clinical examination indicates a possible neural etiology for the patient's symptoms, **electromyography (EMG)** can be helpful in excluding posterior interosseous nerve compression syndrome as the diagnosis.
- Anesthetic injections into the origin of the ECRB muscle can help confirm the diagnosis, as the patient should experience relief from symptoms.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of this condition include other conditions that can produce pain in this general vicinity like, commonly radial tunnel syndrome, osteochondritis dessicans of the capitellum(Panner's disease), lateral compartment arthrosis, varus instability and cervical radiculopathy.

Treatment

There are numerous treatment modalities for lateral epicondylitis both conservative and operative. Many methods of treatment are available but benefits of most are unclear. The choice of treatment for individual cases remains controversial because it is empirical and based on personal experience of the physician treating the patient²¹.

Conservative treatment has been the method of choice in the literature for the individual with lateral epicondylitis.²¹

Principles of conservative treatment include,

- Cold application,
- Rest,
- Control of inflammation
- Rehabilitation- graded physiotherapy stretching and strengthening exercises, avoidance of causative activity (correct technique or address equipment concerns in athletes who participate in racquet sports, modify jobs or activities in patients who are not athletes).²²

It includes non steroidal anti-inflammatory drugs, physiotherapy (including ionophores and laser), cryotherapy, acupuncture, local anaesthetics, local autologous blood injection, local corticosteroid injection, extracorporeal shock wave, laser treatment, and Botulinum toxin injection, orthoses (splints, straps, braces), counter force braces.^{7,8,20,23.}

Corticosteroid injection is usually preferred. Yet no definitive type of treatment is universally endorsed.²¹

In operative treatment is are considered only when failure of conservative methods for 6-12 months²⁰. Most commonly used techniques are-

- Intra articular- Boyd, McLeod procedure and Almquist procedure (anconeus interposition).
- Extra articular- Debridement, excision, Lengthening of Extensor corpi radialis brevis and reattachment.¹⁷
- Open fasciotomy and release of extensor aponuerosis.
- Percutaneous lateral release of extensor origin.
- Lateral epicondylectomy.
- Arthroscopic release.

Most treatment modalities in tennis elbow are focused on suppressing inflammatory process, which is absent in tennis elbow. With the use of local injections, fenestration of the area may be beneficial because of the bleeding that occurs in the new channels that are created through the degenerated myxoid tissue.

The mechanical disruption may transform a failed intrinsic healing response into an extrinsic response may initiate healing response in the tendon.^{6,24} This may be the reason that injections of local steroid have fortuitous lasting benefits.

A systematic review in 1992, evaluated the effectiveness of various treatments for lateral epicondylitis. The review included five (randomized) clinical trials on corticosteroid injections published between 1966 and 1990 in French or English. Because of the poor quality of methods and the contradictory results, concluded that there was insufficient scientific evidence for any particular type of treatment for lateral epicondylitis.²¹

In 1996 systematic review of validity and outcome of randomised controlled trials of corticosteroid injections for lateral epicondylitis was performed.

Effectiveness of treatment in these studies was assessed by the change in pain score or a global assessment by the patient or an assessor. Pooled analysis indicated short-term effectiveness only (2-6 weeks). At longer term follow up (>6 weeks) no difference between corticosteroid injection and other treatments including placebo. No conclusions could be made about the most suitable corticosteroid, dose, injection interval, or injection volume.⁸

In 1999 a multicentre pragmatic randomised controlled trial in 164 subjects presenting with a new episode of lateral epicondylitis, comparing local corticosteroid injection, oral non-steroidal anti-inflammatory drugs, and simple analgesics was done. After four weeks, 82% of patients were “better” (pain 3 on patient's 10 point Likert scale) in the corticosteroid group compared with 48% in the non-steroidal anti-inflammatory group and 50% in the analgesic groups. However, at 1 year, outcome was similar in all groups (84% v 85% v 82%).²⁵

A study in 2002 concluded that for short-term outcomes (>6 weeks), statistically significant and clinically relevant differences were found on pain, global improvement and grip strength for corticosteroid injection compared to placebo, local anaesthetic and conservative treatments. For intermediate (6 weeks–6 months) and long term outcomes (>6 months), no statistically significant or clinically relevant results in favour of corticosteroid injections were found. Although the available evidence shows superior short-term effects of corticosteroid injections for lateral epicondylitis, it is not possible to draw firm conclusions on the effectiveness of injections, due to the lack of high quality studies. No beneficial effects were found for intermediate or long-term follow-up. More, better designed, conducted and reported RCTs with intermediate and long-term follow-up are needed.⁷

A study in 2006 concluded that corticosteroid injection showed significantly better effects at six weeks. The significant short-term benefits of corticosteroid injection are paradoxically reversed after six weeks, with high recurrence rates, implying that this treatment should be used with caution in the management of tennis elbow. Significantly poorer outcomes in the long term were seen compared to physiotherapy or wait and see policy, which were not significantly different from each other.²⁶

Studies on animal models have shown that intratendinous corticosteroid adversely affect the biomechanical properties of tendons. Corticosteroids can inhibit formation of adhesions, granulation, and connective tissue; reduce tendon mass; and decrease biomechanical integrity and the amount of load that can be taken before failure.²⁷ The biomechanical effects of peritendinous corticosteroid on human tendons are unestablished. However, case reports of rupture of tendons after injection are common.²⁸

Corticosteroid injection is associated with side effects. Sepsis is reported in up to 1 in 17 intra-articular or soft tissue injections.⁷⁶ Other side effects are post injection pain(11-58%), local skin atrophy(17-40%), facial flushing, post injection flare, hyperglycemia and hypersensitivity reactions²⁸ Resuscitation facilities should be available in case patients have a rare severe reaction.

Contraindications to corticosteroid injection in soft tissue lesions ²⁹

- Local or systemic infection
- Coagulopathy
- Tendon tear
- Young patients

Drugs have different potency and solubility, and solubility is inversely correlated with the duration of action. Short or moderate acting, more soluble preparations (such as hydrocortisone and methyl prednisolone) are recommended for soft tissue injections because in theory they cause fewer side effects. Drugs with low solubility should not be used for soft tissue injections.

In 2003, a study was conducted on 28 people in whom conservative therapy had failed to resolve symptoms from their lateral epicondylitis. The study demonstrated 79% (22 of 28) of the patients had a reduction in pain over 9.5 months after autologous blood injection therapy. Most often, this occurred after only one injection.¹⁰ It is hypothesized that mitogens such as platelet derived growth factor induce fibroblastic mitosis and chemotactic polypeptides such as transforming growth factor cause fibroblasts to migrate and specialize and have been found to cause angiogenesis. A specific humoral mediator may promote the healing cascade in the treatment of tendinosis as well.²⁴

Another study demonstrated significantly reduced pain when treating chronic elbow tendinosis with buffered platelet rich plasma. 140 patients with elbow epicondylar pain were evaluated. 20 patients continued to consider surgical intervention after conservative therapy failed to resolve their symptoms. These patients were then administered either a single percutaneous injection of platelet-rich plasma or bupivacaine (control group). At 8 weeks after therapy, the authors demonstrated a 60% pain improvement in the group who received the platelet-rich plasma compared with a 16% pain improvement in the control group. At 6 months and final follow-up (mean, 25.6 months; range, 12-38 months), the patients who had received the platelet-rich plasma continued to report significant pain reduction.³⁰

Sonographic-guided blood injection has been reported to improve clinical outcome. It can also be used to monitor the changes to the common extensor origin.

A study in 2012 demonstrated the autologous blood injection technique has been used successfully in the treatment of lateral epicondylitis³¹

METHODOLOGY

Source of Data

All confirmed patients of lateral epicondylitis willing for the treatment attending R L Jalappa General Hospital, Tamaka, Kolar, from July 2012– April 2013.

Method of Collection of Data

1. By interview & examination
2. By follow-up of total 6 months. It is divided in to intervals at 2week, 2nd month and 6 th month
3. **Sample size:** The randomized control trial is a pilot study, so 30 cases and 30 controls were selected.
4. **Study design:** Randomized control trial comparing the efficacy of autologous blood injection with local corticosteroid injection.
5. Consent was taken from the participants.

No blinding procedure can be followed because it was difficult to blind either patient or investigator in regard to drawing and injecting autologous blood

Cases are injected with intralesional autologous blood injection and controls are injected with local Corticosteroid injection at lateral epicondyle.

Randomization

A randomization coding system derived from a computer generated randomization table was followed. After a proper clinical diagnosis patients were selected into two groups according to randomization table.

Inclusion Criteria

1. Duration of lateral epicondylitis(tennis elbow) > 6 weeks and age of patients more than 18 years.
2. Lateral elbow pain that is maximal over the lateral epicondyle, increases with pressure on the lateral epicondyle and resisted dorsiflexion of wrist.

Exclusion criteria

1. Trauma to the affected elbow in the preceding 6 weeks
2. Patients with a past history of elbow instability
3. Previous elbow surgery
4. Steroid injection for the presenting condition within the previous 6 months

Procedure

Group A / autologous blood injection group:

Patients were infiltrated with a injection of 2 milliliters autologous blood at the lateral epicondyle according to the below mentioned technique.

Group B / Local Steroid with local anesthetic injection group:

Patients were infiltrated with 2 milliliters of local corticosteroid (Methyl prednisolone acetate 80mg), at the lateral epicondyle according to the below mentioned technique.

Injection technique: The elbow is flexed to 90° with the palm facing down. Procedure: With patient in supine or sitting posture, elbow will be painted and draped. The bony anatomical landmarks are identified. Two milliliters of autologous blood

drown from the contralateral upper extremity vein. The elbow is flexed to 90° with the palm facing down. The needle introduced proximal to the lateral epicondyle along the supracondylar ridge and gently advanced in to the undersurface of the extensor corpi radialis brevis while infusing the blood intralesionally. Then after two minutes Mill's manipulations were done. With forearm in maximum pronation and wrist in maximum palmar flexion the elbow was repeatedly extended and stretched six to seven times. And then a small adhesive sterile dressing was given at the injection site, which was advised to be removed after 2 days. Patients were advised to give rest to the upper limb for 3 days. And after that no restriction of activity is advised.

Controls were injected with 2 millileters local corticosteroid (Methyl prednisolone acetate 80mg) in the same technique as described above.



Fig 17 Methyl prednisolone acetate



**Fig 18 Methyl prednisolone acetate
Injection at lateral epicondyle**



**Fig 19 Autologous blood injection at
lateral epicondyle**

Outcome evaluation

Outcome is measured using ‘Pain score’

Outcome measures:

PAIN SCORE; VISUAL ANALOGUE SCALE:

Pain of the participants will be assessed by most widely used and accepted “visual analogue scale”. It consists of a 10 centimeter line marked at one end with “no pain” and at other end with “worst pain ever”. Participant is asked to indicate where on the line he or she rates the pain on the day of presentation, 2,6weeks and 6 month of follow-ups. Numerical value is then given to it simply by measuring length between “no pain” to patients mark.

No pain ____ 1 ____ 2 ____ 3 ____ 4 ____ 5 ____ 6 ____ 7 ____ 8 ____ 9 ____ 10 worst pain ever.

Statistical test : Student t test is applied to calculate the significance of results.

OBSERVATIONS AND RESULTS

Procedure was done in 60 patients under the present study. Participants were clinically evaluated. A baseline VAS scores of the pain at lateral epicondyle was recorded. Cases were treated with autologous blood injection and controls with local corticosteroid injection. After the procedure patients were asked to report immediately if any increase in pain was there and were asked to follow up at 2 weeks, 6 weeks and 6 months interval after the intervention. If pain persisted analgesics were given and was advised to be taken only if there is unbearable pain. Some patients were given just placebos like calcium tablets or B-complex capsules for one to three weeks, if they had vague complaints which were not corresponding to the clinical findings.

Table No. 1 : Age Distribution in both the groups

	Local steroid injection	Autologous blood injection
Mean age of participants	42.27	42.9
S.D	9.51	12.8

Age group encountered in the study ranged from 17 years to 67 years, with a mean age of 42.6. Peak incidence at fourth decade of life was seen. The mean age of patients in autologous blood injection group was 42.9 and in corticosteroid injection group was 42.2

p value= 0.8283 which was non significant. Thus age of patients in both the groups was comparable.

Graph 1 : Mean Age of Patients in both the groups

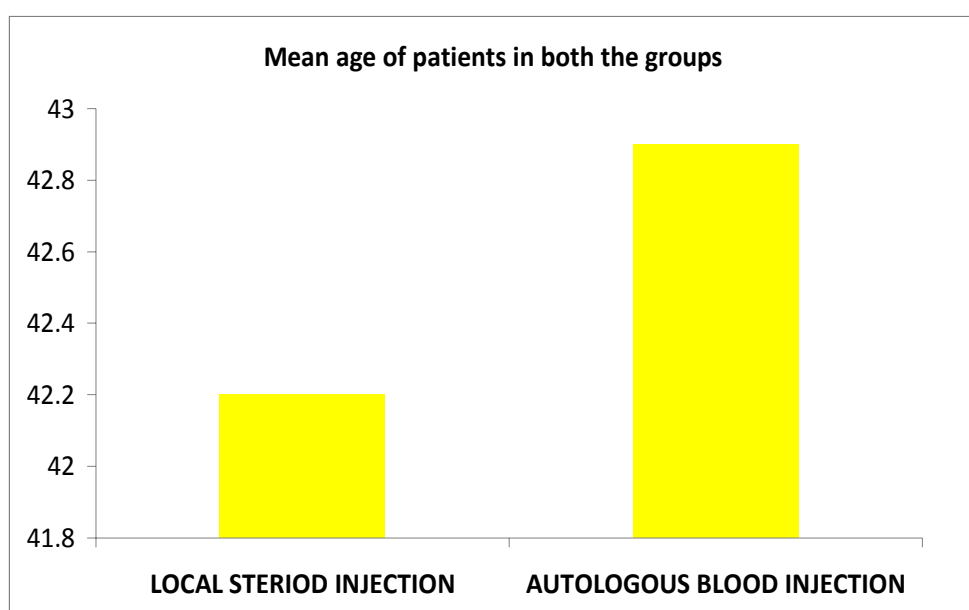


Table 2 : Sex distribution in both the groups

Sex	Local steroid injection	Autologous blood injection	Total
Male	13	13	26
Female	17	17	34
Total	30	30	

Out of the 60 participants, 26 were males and 34 were females.

In corticosteroid injection group 13 were males and 17 were females.

In autologous blood injection group 13 were males and 17 were females.

p value= 1 which is non significant. Thus both the groups were comparable in terms of number of males and females in each group.

Graph 2 : Sex distribution in both the groups

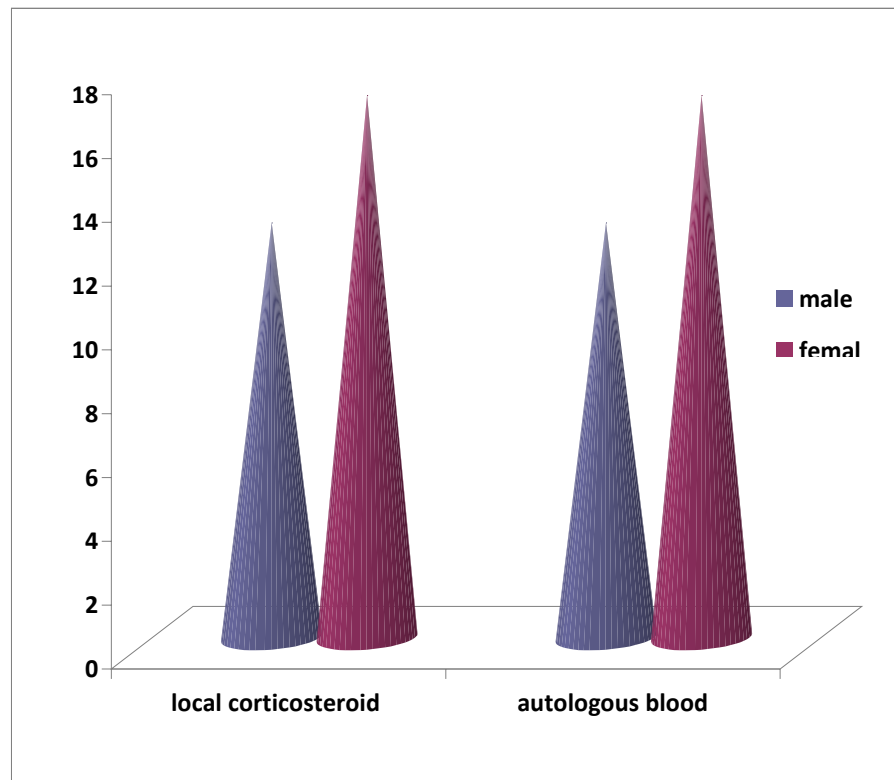


Table No. 3 : Elbow side involved in both groups

	Type		
Side	Blood	Steroid	Total
Left	6	5	11
Right	24	25	49
Total	30	30	60

Out of the 60 participants, 49 participants had their right side elbow affected and 11 had their left side affected.

p value= 1 which is non significant. Thus both the groups were comparable in terms of side of elbow involvement.

Graph 3 : Elbow side involved in both the groups

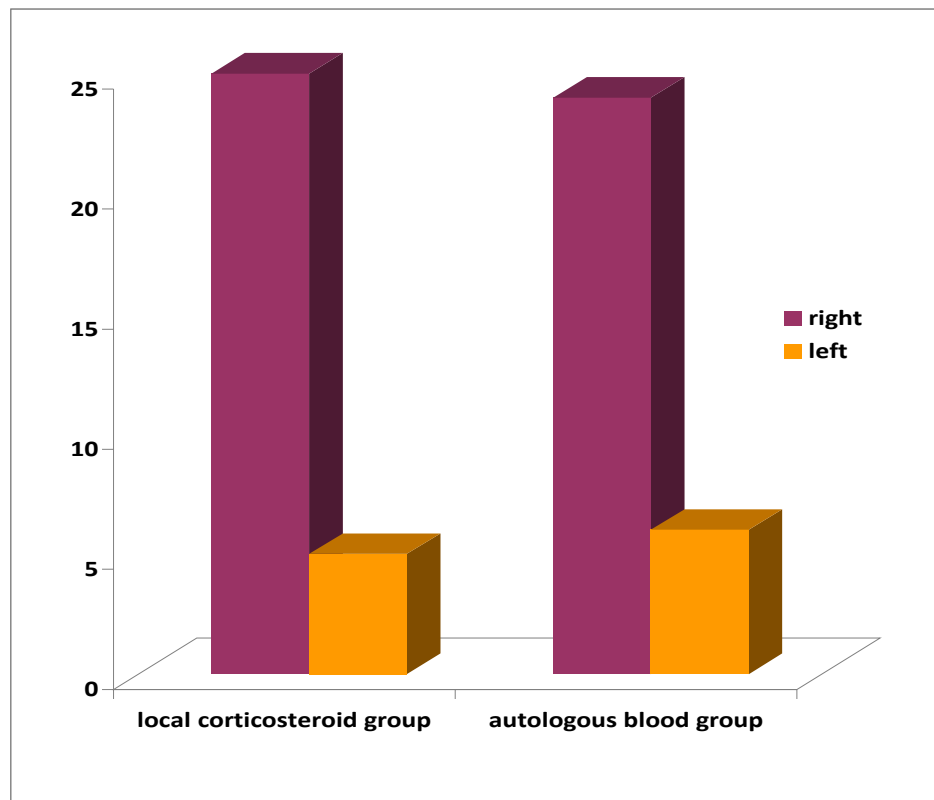


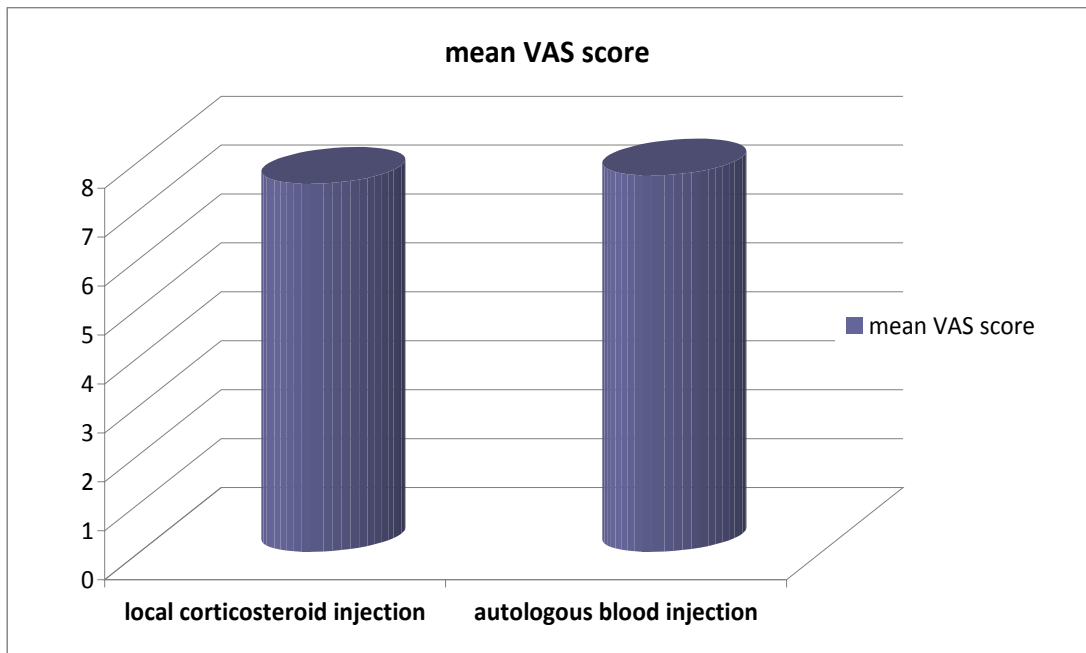
Table No. 4 : Outcome values at the time of Presentation (before injection)

	Local corticosteroid injection	Autologous blood injection	P Value	Inference
Mean VAS score	7.53	7.70	0.53945	N S
S D	1.2794	1.3429		

p value for VAS Score is 0.5395 which is statistically not significant.

Hence the outcome values before the injection are comparable

Graph 4 : Outcome values at the time of Presentation
(before injection)



First follow up at 2nd week

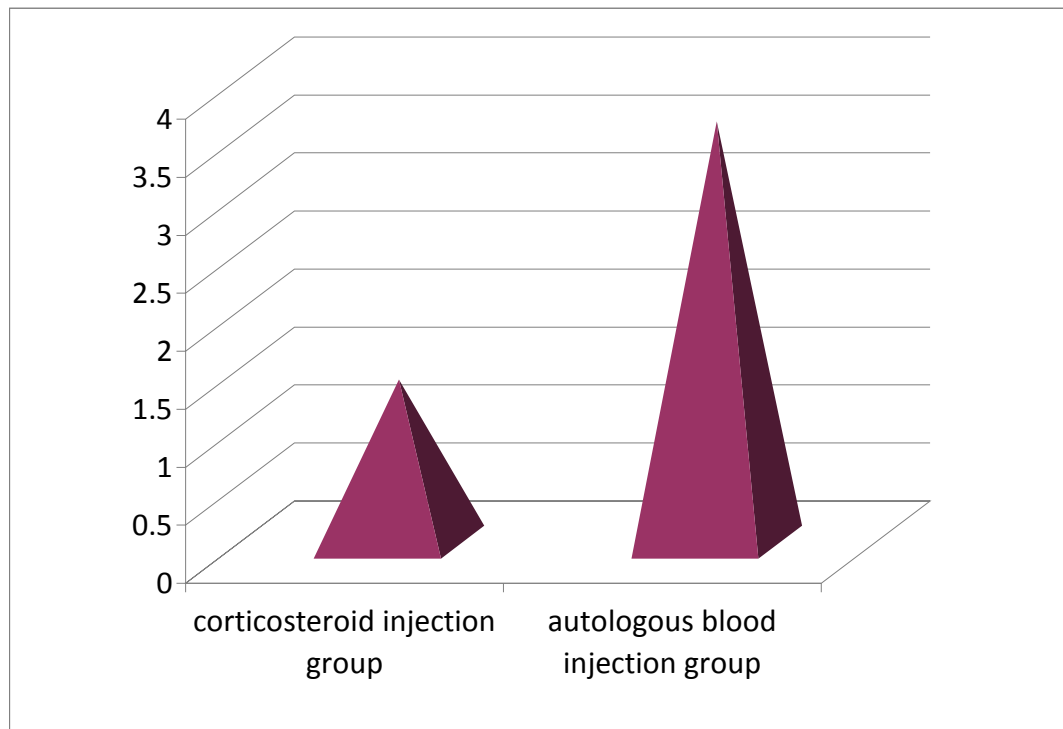
Table no 5: outcome value at 2nd week

	Corticosteroid injection group	Autologous blood group	P value	inference
Mean VAS	1.40	3.63	0.001	S
S D	0.62	0.61		

p value for VAS Score is < 0.001 which is statistically significant

Hence the decrease in pain at 2nd week is statistically significant in corticosteroid injection compared to autologous blood injection group.

Graph 5 : Outcome values at 2nd week



Follow up at 2nd month

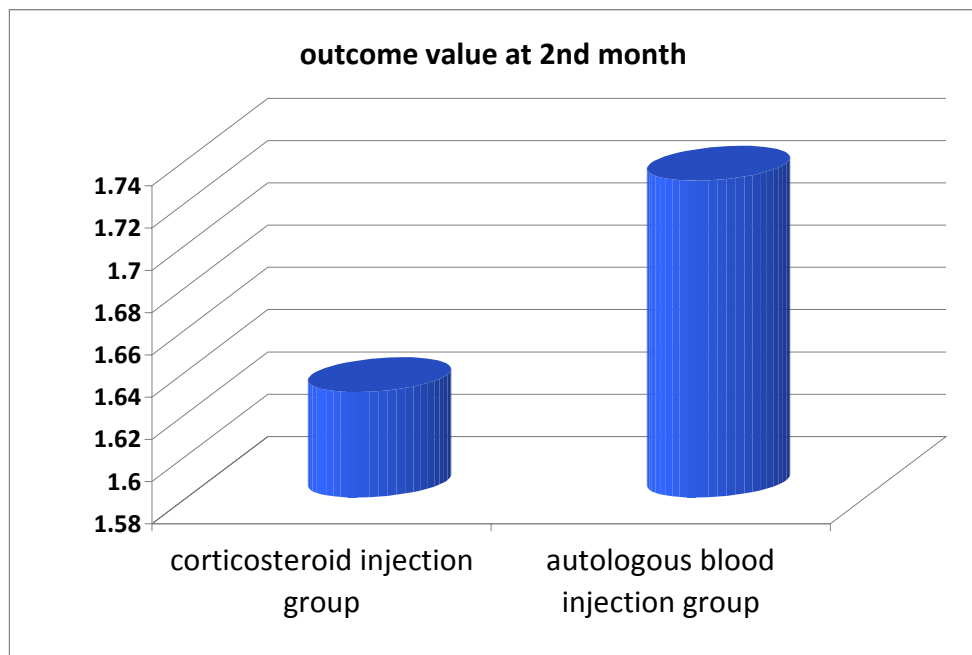
Table no 6: outcome value at 2nd month

	Corticosteroid injection group	Autologous blood group	P value	Inference
Mean VAS score	1.6	1.73	0.001	S
S D	0.25	0.63		

p value for VAS Score is 0.001 which is statistically significant

Hence the decrease in pain at 2nd Month is statistically significant in corticosteroid injection compared to autologous blood injection group.

Graph 6 : Outcome values at 2nd month



Follow up at 6 month

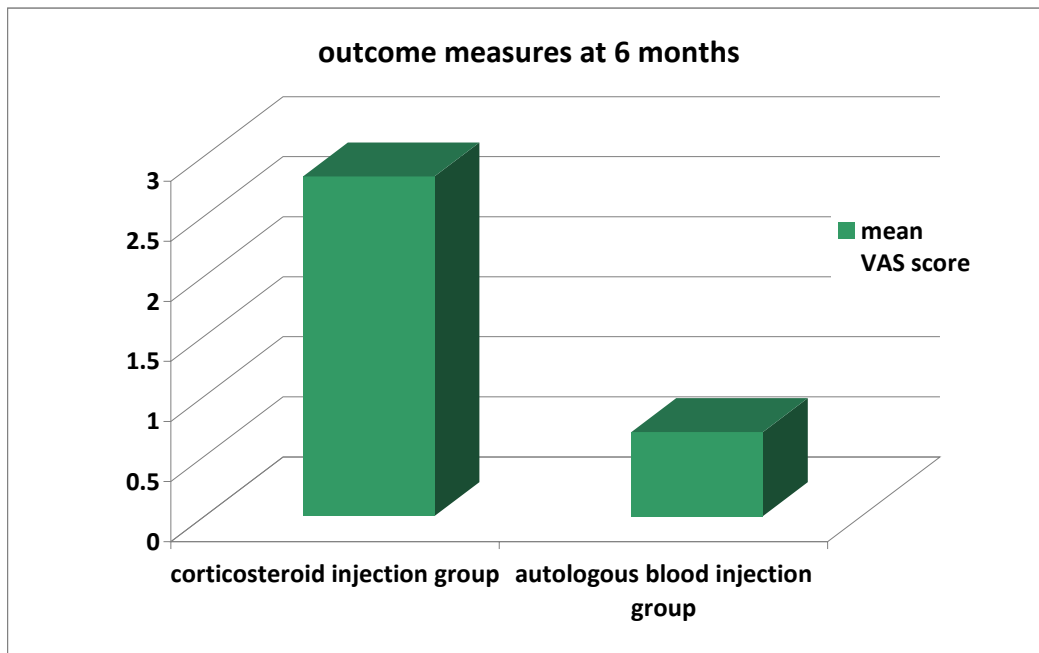
Table no 7 : Outcome value at 6th month

	Corticosteroid injection group	Autologous blood group	P value	Inference
Mean VAS score	2.83	0.7	0.001	S
S D	1.83	0.79		

p value for VAS Score is 0.001 which is statistically significant

Hence the decrease in pain at 6th Month is statistically significant in autologous blood injection group compared to corticosteroid injection group

Graph 7: Outcome value at 6th month

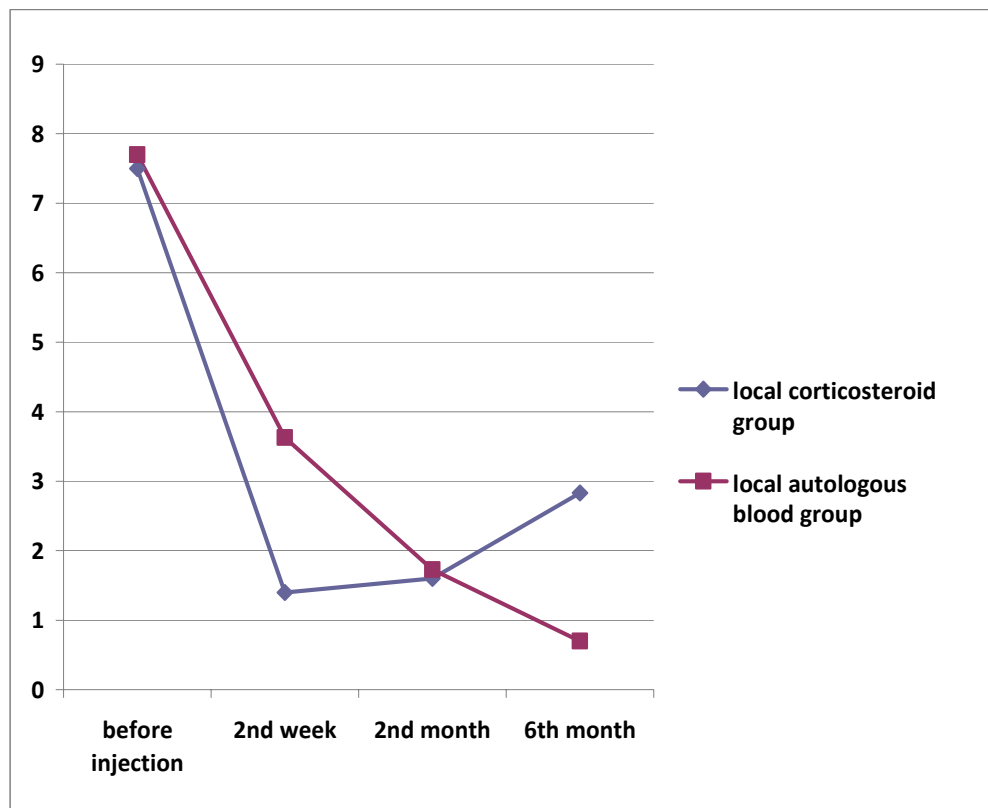


**The severity of pain during the day at baseline and during follow-up at 2week,
2nd month and 6th month**

Table no 8: Mean VAS score for the two groups

	Corticosteroid injection group		Autologous blood injection group			
Follow up period	Mean VAS Score	S D	Mean VAS Score	S D	p value	inference
Before injection	7.533	1.279	7.7	1.342	0.5395	N S
2 nd week	1.40	0.62	3.63	0.61	0.001	S
2 nd month	1.6	0.25	1.73	0.63	0.001	S
6 th month	2.83	1.83	0.7	0.79	0.001	S

Graph 8 : Mean VAS score pre and post procedure at 2nd week , 2nd month and 6th month



Complications

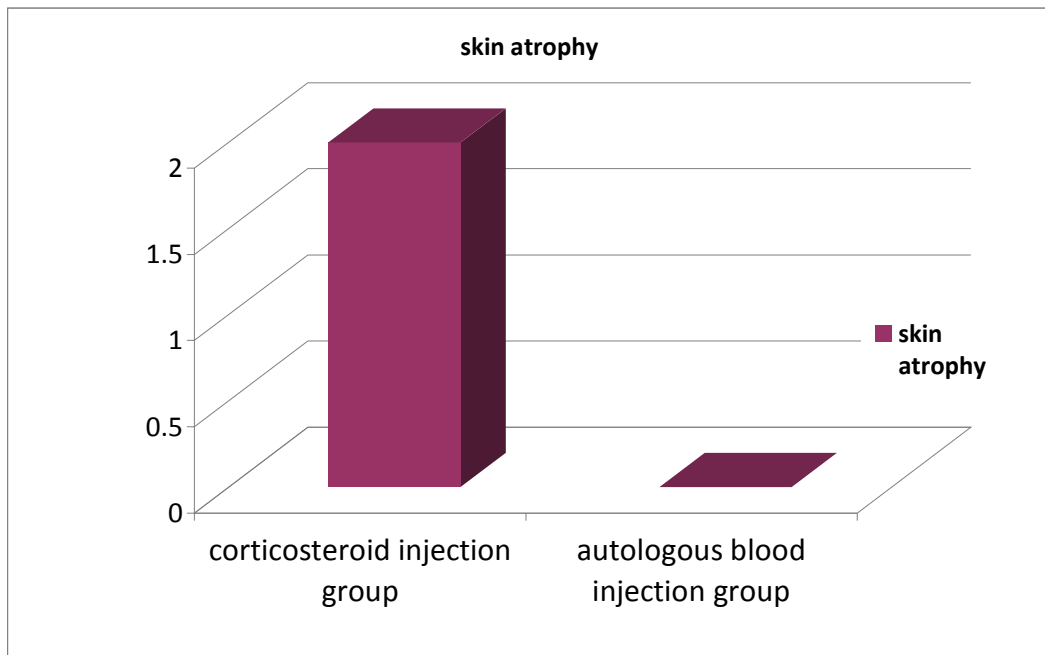
Table 9 : Local skin atrophy

		Corticosteroid injection	Autologous blood injection	Total
Local skin atrophy	yes	2	0	2
	no	28	30	58
Total		30	30	

Local skin atrophy: Only two patients (6.6%) had local skin atrophy in corticosteroid injection group while no patient in autologous blood injection group had this problem.

p value= .0150 which is non-significant. There was no statistical significance related to post intervention local skin atrophy.

Graph 9 : Complication- Skin atrophy



DISCUSSION

Tennis Elbow is a common problem encountered in Orthopaedic practice and general Practice. Majority of the treatment modalities used for its management lack scientific rationale. The role of local steroid is debatable²¹.

Recently an injection of autologous blood has been reported to be effective for both intermediate and long term outcomes for the treatment of lateral epicondylitis. There was a significant decrease in pain.⁵ It is hypothesized that mitogens such as platelet derived growth factor induce fibroblastic mitosis and chemotactic polypeptides such as transforming growth factor cause fibroblasts to migrate and specialize and have been found to cause angiogenesis. A specific humoral mediator may promote the healing cascade in the treatment of tendinosis as well. These growth factors trigger stem cell recruitment, increase local vascularity and directly stimulate the production of collagen by tendon sheath fibroblasts.⁹

In this current study, the mean age encountered was 42.7 years (Range: 17 to 67 years); the peak incidence was seen from 35 to 50 years. This was seen similar in two separate studies which observed mean age of 45 and 43 years.^{31,81} Another study observed the mean age to be 46.5 years.⁵

In this current study, out of the 60 participants, 25 (41.6%) were male patients and 35(58.3%) were female patients. Two other studies had more number of male patients.²⁵ one more study had equal number of males and female patients.⁵ Contrary to other studies more number of female patients in this current study may be due to that, females at this study area are more involved with household work which causes repetitive stress at the extensor carpi radialis brevis origin causing micro trauma, a relevant etiology for the initiation of the disease.

In this current study, out of the 60 participants, 46(76.6%) participants had their right side elbow affected and 14 (23.4%) had their left side affected. Out of the 60 participants, 51(85%) participants had their Dominant elbow affected and 9 (15%) had their Nondominant elbow affected. In other two studies, one had 84% of the patients with their dominant elbow affected, while in another 78.6% of the patients with their dominant side affected.⁵

Parameters like age, sex, side of elbow involved, dominance of upper limb involved, duration of symptom and type of occupation of the patients were comparable. The mean VAS score before injection in both the groups was comparable. Mean VAS score for steroid injection group was 7.533, mean VAS score for autologous blood injection group was 7.7, P value was 0.5395.

Till 2 weeks follow up, statistically significant difference between the two groups with VAS scoring was seen. Corticosteroid injection group showed statistically significant decrease in VAS score at 2nd week, 2nd month compared to autologous blood injection group. One study showed similar results with local corticosteroid injection group, when compared with oral naproxen.²⁵

At 2nd month and 6th month follow up autologous blood injection group showed statistically significant decrease in VAS score compared to corticosteroid group. At 6 months follow up, mean VAS score for steroid injection group=2.83, mean VAS score for autologous blood injection group was 0.7, P value was 0.001;

At the end of 6 months 46.66% patients in corticosteroid injection group and 90% patients in autologous blood injection group were completely relieved of pain.

This was highly statistically significant with a P value of <0.001.

One study reported that 22/28 patients (79%) responded to autologous blood injections with average Nirschl Scores decreasing from 6.5 to 2.0 with a mean follow up of 9.5 months.⁵

In Corticosteroid injection group till 2 weeks there was significant improvement with 63.3% of patients completely relieved of pain. Many of these patients reported recurrences at 2nd month and 6month follow up. The rate of recurrence was 36.8% in corticosteroid injection group. Similar recurrence rate was seen in one study where 14% patients worsened in their symptoms with corticosteroid injection.²⁵

In autologous blood injection group at 2nd month follow up, 16.66% of patients were completely free of pain. At the end of 6 months there was no recurrence. This was statistically significant with a P value of <0.001.

Maximum benefit reached at an average of 6.1 weeks in corticosteroid injection group. Maximum benefit reached at an average of 11.43 weeks in autologous blood injection group. This was statistically significant with a P value of 0.0003.

This study cannot prove conclusively whether the blood itself induced an inflammatory cascade or whether the injury created by the injection was responsible. It is theorized that the beneficial effects of steroid injection result from the bleeding caused by forcing fluid through tissue planes at high pressures.³²

It was seen that there was a significant increase in post intervention pain for few days in autologous blood injection group. In corticosteroid injection group 8 participants (26%) patients complained of post-intervention exacerbation of pain while in autologous injection group 18 participants (60%) complained of increase of pain after local injection. This was statistically significant with a p value of 0.009.

And these patients had to be managed with oral analgesics for varying period of days (2to7days) for pain relief.

Only two patients (6.6%) had local skin atrophy in corticosteroid injection group while no patient in autologous injection group had this problem. Between two groups there was no statistical significance related to post intervention local skin atrophy. ($p = 0.150$) showing that the local steroid infiltration done with proper investigations and care gives rise to negligible complication.

To conclude, autologous blood injection is beneficial both in short term and longterm for the treatment of lateral epicondylitis. Advantages of autologous blood injection are-highly acceptable, efficacious, economic, easy to carry out as outpatient procedure, devoid of potential complications such as hypoglycemia, skin atrophy, tendon tears associated with corticosteroid injection and low recurrence rate.

Clinical findings such as those presented should be correlated with histologic specimens showing evidence of healing such as organization of collagen bundles and return to normal cellular activity after injections of autologous blood into areas of tendinosis. The subject bias inherent in the design of our study was unavoidable because it was difficult to blind either patient or investigator in regard to drawing and injecting autologous blood. Furthermore most patients are reluctant to donate blood that may be discarded and not used for their benefit. Nonetheless this study offers encouraging results of an alternative treatment that addresses the pathophysiology of lateral epicondylitis that has failed traditional nonsurgical modalities. Further clinical studies may prompt other investigators to further define substances that may enhance tendon healing for lateral epicondylitis and other disabling tendinoses.

CONCLUSION

- Lateral epicondylitis, is a common problem encountered in the orthopaedic practice.
- Even though it has been termed tennis elbow and called same routinely, it is seen to affect non-athletes rather than athletes.
- Much controversy has been there over the pathophysiology and there is not enough scientific evidence to favour any particular type of treatment for acute lateral epicondylitis.
- Currently degeneration of the origin of the extensor carpi radialis brevis (ECRB), repeated micro trauma and incomplete healing response has been accepted as the cause of lateral epicondylitis by most of the researchers.
- Histopathological reports have shown that lateral epicondylitis is not an inflammatory process but a degenerative condition termed 'tendinosis'
- Most conservative modalities such as local corticosteroid injection have focused on suppressing inflammatory process that does not actually exist.
- Corticosteroid injection is associated with high recurrence on long term follow-ups.
- In this study autologous blood injection demonstrated a statistically significant decrease in pain compared to corticosteroid injection group even on long term follow up (6 months).
- At the end of 6 months 46.66% patients in Corticosteroid injection group and 90% patients in autologous blood injection group were completely relieved of pain.
- The duration for maximum benefit to reach is longer in autologous blood injection (11.43weeks) compared to corticosteroid injection (6.1 weeks).

- Autologous blood injection is associated with more post injection pain compared to corticosteroid injection.
- Autologous blood injection technique for lateral epicondylitis offers a better treatment with (1) its application is minimally traumatic, (2) it has a reduced risk for immunemediated rejection, devoid of potential complications such as hypoglycemia, skin atrophy, tendon tears associated with corticosteroid injection, (3) it is simple to acquire and prepare, easy to carry out as outpatient procedure and (4) it is inexpensive, (5) better relief of pain, (6) low recurrence rate.
- This study offers encouraging results of an alternative treatment that addresses the pathophysiology of lateral epicondylitis that has failed traditional nonsurgical modalities.

SUMMARY

- It is a prospective randomized control study comparing the efficacy of autologous blood injection with local corticosteroid injection.
- 30 cases and 30 controls were selected. Randomization is done using randomization table.
- All patients were clinically evaluated and the severity of pain is recorded using VASscoring
- Cases are injected with intralesional autologous blood injection and controls are injected with local Corticosteroid injection at lateral epicondyle.
- Participants were followed-up for total of 6 months. Follow up period was divided in to intervals of 2nd week, 2nd month and 6 months.
- Outcome is measured using ‘Pain score’ of lateral epicondylitis’.
- Student t test is applied to calculate the significance of results.
- At 2nd week the corticosteroid injection group showed a statistically significant decrease in pain compared to autologous blood injection group.
- At 2nd month and 6 months follow up autologous blood injection group showed statistically significant decrease in pain compared to corticosteroid injection group.
- At the end of 6 months 46.66% patients in corticosteroid injection group and 90% patients in autologous blood injection group were completely relieved of pain.
- Maximum benefit reached at an average of 6.1 weeks in corticosteroid injection group. Maximum benefit reached at an average of 11.43 weeks in autologous blood injection group.

- It was seen that there was a significant increase in post intervention pain for few days in autologous blood injection group. In corticosteroid injection group 8 participants (26%) patients complained of post-intervention exacerbation of pain while in autologous injection group 18 participants (60%) complained of increase of pain after local injection.
- Recurrence rate of 36.8% was noted in corticosteroid injection group and 0% in autologous blood injection group at the end of 6 months.
- Autologous blood injection technique for lateral epicondylitis offers a better treatment with least side effects, cost effective and with minimum recurrence rate.

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ANNEXURES

CLINICAL PROFORMA FOR LOCAL STEROID OR AUTOLOGOUS BLOOD INJECTION FOR LATERAL EPICONDYLITIS

PATIENT PARTICULARS:

Name	Age	Sex
Height	Weight	
Address		
Occupations		

CHIEF COMPLAINTS:

Anatomical location of pain

Onset of pain

Duration period of pain

Does pain increase on activity

Any morning pain

Any numbness of Hand

Is patient able to bear weight

Any referred pain

PAST HISTORY:

Any co-morbidities

Any previous local injuries around elbow

Any previous surgeries

Any treatment taken in the past

Is Patient pregnant if female

LOCAL EXAMINATION:

Any inflammation present

Sensation of hand

Movements of elbow - flexion
extension

Any deformity present

Any neurological deficit

SPECIAL TESTS

- 1) Max tenderness over lateral epicondyle -
- 2) Restricted middle finger extension test -
- 3) Cozen's test -
- 4) Mill's test -

INVESTIGATION:

General Blood investigations

CBC	HB	TC	DC	ESR	PS	BT	CT	BLOOD.GROUP	PLT
SAMPLE		HIV		HBV		HCV		MP	

Patients BP and GRBS status

X-ray – elbow, AP, Lateral, USG/MRI :- (If necessary)

Injection protocol	Date	Pain levels reduced
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1 st injection		
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POST INJECTION CARE:

1st 48hrs non weight bearing

NSAIDS to be avoided, pain killers like Acetaminophen a given

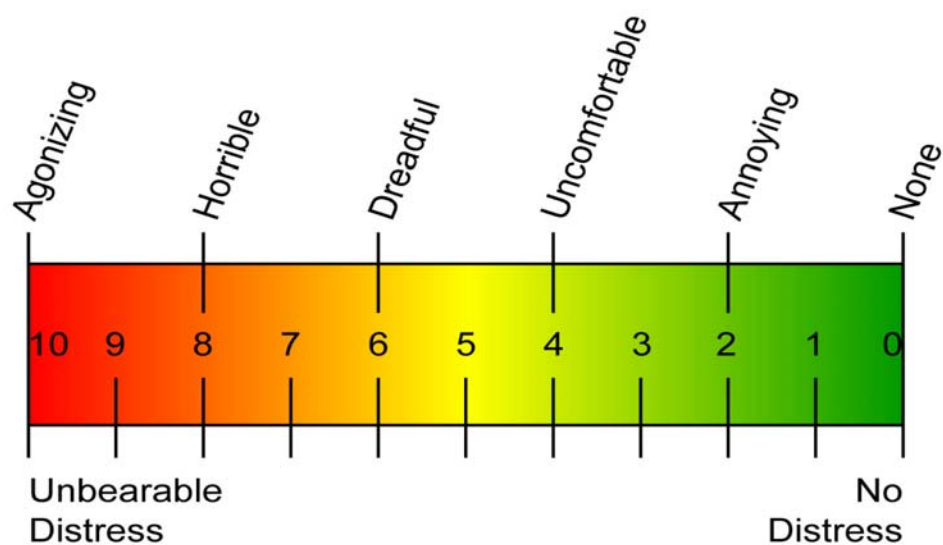
After 48hrs to 1week limited weight bearing

Assessment for pain	2 nd week
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(By visual analog scale)	2 nd month
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O- No pain	6 th month
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10- Worst pain



CONSENT OF THE PATIENT

PATIENT SIGNATURE

DOCTOR SIGNATURE

MASTER CHART : LOCAL METHYL PREDNISOLONE ACETATE INJECTION GROUP

SI NO	NAME	Hosp No	Age	Sex	Side	Date of Injection	VAS Score	2nd Week	2nd Month	6th Month
1	INDRAMMA	822125	45	Female	Right	18.07.12	6	2	1	1
2	NARAYANAPPA	822419	60	Male	Right	19.07.12	7	1	1	1
3	REDDAPPA	650245	65	Male	Left	10.07.12	6	1	1	6
4	NAGARATHNAMMA	SNR	32	Female	Right	05.10.12	7	1	1	5
5	NAGARATHNAMMA	829016	35	Female	Right	03.09.12	6	2	1	4
6	RATHNAMMA	835470	35	Female	Right	30.08.12	7	1	1	5
7	KOMALA	835495	28	Female	Right	31.08.12	6	3	1	1
8	CHAMUNDAMMA	SNR	65	Female	Left	12.06.12	7	1	1	4
9	MALLEESHAPPA	614813	45	Male	Right	12.09.12	8	2	1	3
10	AKHILA	762836	25	Female	Right	16.10.12	6	1	1	1
11	RAMANA	678657	46	Male	Right	24.11.12	7	1	1	4
12	DANALAKSHMI	869755	43	Male	Right	22.12.12	6	1	1	5
13	ANJANEYAPPA	895632	46	Male	Left	23.12.12	8	2	1	5
14	NARAYANASWAMY	870161	43	Male	Right	24.12.12	6	1	1	3
15	NANAMMA	871275	58	Female	Left	28.12.12	6	3	1	1
16	GOUSE PASHA	678681	43	Male	Right	05.01.13	7	2	1	1
17	SURAPPA	876512	43	Male	Right	16.01.13	6	1	1	5
18	KAMALAMMA	878071	36	Female	Right	21.01.13	6	2	1	1
19	BALARAJ	762116	36	Male	Right	15.02.12	7	1	1	4
20	VIJAY KUMAR	633410	37	Male	Right	18.02.13	7	1	1	1
21	LAKSHMAMMA	901345	44	Female	Right	21.02.13	7	1	1	5
22	SUMITHRAMMA	746754	45	Female	Right	24.02.13	7	1	1	1
23	SUMITHARA	888083	27	Female	Right	25.02.13	8	1	2	1
24	SHARADA	888616	35	Female	Right	26.02.13	7	1	2	1
25	KRISHNA REDDY	897564	38	Male	Left	26.02.13	7	1	1	4
26	BASAMMA	894893	56	Female	Right	20.03.13	8	1	1	5
27	DEVAMMA	898714	47	Female	Right	03.04.13	7	2	1	1
28	DEV PRASAD	913246	33	Male	Right	03.04.13	7	2	1	1
29	CHAMUNDAMMA	901690	46	Female	Right	15.04.13	7	1	1	1
30	PAPAMMA	907436	54	Female	Right	04.05.13	7	1	1	4

MASTER CHART : AUTOLOGOUS BLOOD INJECTION GROUP

SI NO	NAME	Hosp No	Age	Sex	Side	Date of Injection	VAS Score	2nd Week	2nd Month	6th Month
1	SONNA PALAIAH	813490	50	Male	Right	19.06.12	6	3	2	0
2	MUNI SWAMI	SNR	40	Male	Left	26.06.12	8	4	3	1
3	NARAYANA	783616	53	Male	Left	10.07.12	8	4	2	0
4	LAKSHMAMMA	825858	43	Female	Right	31.07.12	6	3	1	1
5	JOYSE	SNR	30	Female	Right	17.08.12	7	4	1	1
6	KANTHAMMA	SNR	35	Female	Right	23.08.12	6	3	2	1
7	RATHNAMMA	838668	52	Female	Right	09.09.12	6	3	2	1
8	LAKSHMAN REDDY	887654	36	Male	Right	21.09.12	8	3	1	0
9	JAYALAKSHMI	849625	25	Female	Right	12.10.12	7	3	1	1
10	RANGARAJU	786068	38	Male	Right	15.10.12	6	4	2	1
11	MALAVIKA	687849	43	Female	Right	22.10.12	7	3	1	0
12	MANGAMMA	678668	44	Female	Right	17.11.12	7	5	3	0
13	CHANNA REDDY	869661	48	Male	Right	22.12.12	6	4	2	0
14	NAYEEMUNNISSA	870129	48	Female	Right	24.12.12	8	4	3	1
15	KRISHNA MURTHY	871305	48	Male	Right	28.12.12	7	4	2	1
16	CHANNARAJ	871702	39	Male	Right	29.12.12	6	3	2	1
17	CHOWDAMMA	876562	44	Female	Right	16.01.13	7	3	1	1
18	KONAMMA	876432	45	Female	Right	17.01.13	7	4	2	1
19	KATHYAYINI	915463	35	Female	Left	17.01.13	7	4	2	0
20	KARYAPPA	878102	56	Male	Right	21.01.13	6	4	2	1
21	NOON JAN	930444	30	Female	Left	23.01.13	8	4	2	0
22	CHAITHANYA	571532	33	Male	Right	09.02.13	7	3	1	1
23	THAMMANNA	542102	44	Male	Right	10.02.13	8	4	2	1
24	ROY	863631	46	Male	Left	12.02.13	7	3	1	0
25	GANGAMMA	894793	56	Female	Right	20.03.13	6	4	2	0
26	KRISHNA MURTHY	894910	49	Female	Right	20.03.13	7	4	2	1
27	KAVYA	894846	26	Female	Left	20.03.13	8	5	2	4
28	SUBBARAYAPPA	898826	43	Male	Right	03.04.13	6	3	1	1
29	ASHWATHAMMA	901721	48	Female	Right	15.04.13	7	4	1	0
30	MANGAMMA	907535	52	Female	Right	04.05.13	8	3	1	0