A RANDOMIZED CONTROL TRIAL TO EVALUATE THE EFFICACY OF AUTOLOGOUS BLOOD INJECTION VERSUS LOCAL CORTICOSTEROID INJECTION FOR TREATMENT OF LATERAL EPICONDYLITIS

Submitted by:

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DISSERTATION

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In partial fulfillment
of the requirements for the award of the degree of

MASTER OF SURGERY
in
ORTHOPAEDICS

Under the Guidance of:

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LIST OF ABBREVIATIONS USED

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

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 evaluate the efficacy and role of autologous blood injection versus local corticosteroid injection in the management of lateral epicondylitis of humerus.

Materials and Methods

A prospective, randomized study was done in K.L.E’s Dr. Prabhakar Kore hospital. 60 patients were included in the study. 30 patients received 2 milliliter autologous blood drown from contra lateral cubital vein + 1 milliliter 0.5% Bupivacaine, and 30 patients received 2 milliliters local corticosteroid (Methyl prednisolone acetate 80 mg) + 1 milliliter 0.5% Bupivacaine at the lateral epicondyle.

Outcome is measured using 'Pain score' and 'Nirschl staging of lateral epicondylitis'. Mann-Whitney U test (non parametric test) is applied to calculate the significance of results.

Results

Follow-up done for total 6 months divided in to intervals at 1week, 4week, 12 week and 6 month. At 1st week and 4th week the corticosteroid injection group showed a statistically significant decrease in pain compared to autologous blood injection group. At 12th week 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 4 weeks there was significant improvement with 63.3% of patients completely relieved of pain. Many of these patients reported recurrences at 12 weeks and 6 month follow up. The rate of recurrence was 36.8% at the end of 6 months.

In autologous blood injection group at 4th week 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

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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.[2,3,4] Various findings have been reported in the literature with respect to gender prevalence, however no distinct prevalence is evident.[4,5] The dominant arm has been found to be predisposed to lateral epicondylitis. An epidemiologic study reported an actual rate - 87% of his cases involved the dominant arm.[6]

Its most characteristic findings are pain and tenderness over the lateral epicondyle. Lateral epicondylitis is extremely common in today's active society and have been reported to be the result of overuse from many activities. Even though it has been termed tennis elbow and called same routinely, it is seen to affect non-athletes rather than athletes.[7,8]

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.[9,10] 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.[11]

Histopathological reports have shown that lateral epicondylitis is not an inflammatory process but a degenerative condition termed 'tendinosis'.[10,11,12] 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.[13,14,15,16]

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.[10,12,17] 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 inexpensive.

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.[8,16,19,20,21]

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.

OBJECTIVE

To evaluate the efficacy and role of autologous blood injection versus local corticosteroid injection in the management of lateral epicondylitis of humerus.

REVIEW OF LITERATURE

Lateral epicondylitis as a clinical entity was first described in 1873 and was associated with extended use of arm in writing.[22] Later, symptoms of lateral epicondylitis in workers in a variety of occupations including carpenters, plumbers, violinists etc. were documented.

In 1982, tennis elbow was attributed to sprain of the pronator radii caused by forcible backstroke whereby the forearm is brought into rapid and forcible pronation.[23]

In 1936, it was described as a condition, 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 epicondyle.[24] This view was later supported by other study in 1979.[27]

A study in 1955, concluded that the pathological changes in the orbicular 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.[25]

A study in 1961 reported that, with such clearly defined clinical and diagnostic features – the continued obscurity of the pathology of tennis elbow is surprising.[26]

A study in 1973 gave specific examples of force overload (due to an ineffective lever system the elbow has a mechanical predisposition to force overload) at the elbow causing lateral epicondylitis. They were: 1) intrinsic overload could result from inadequate forearm extensor strength and endurance to withstand the moments of force placed on the forearm 2) extrinsic overload from insufficient forearm extensor flexibility; and 3) intrinsic and extrinsic overload due to overwhelming moments of force or repetition in an individual with reasonable muscle flexibility, strength and endurance. [22]

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.[27]

"Overuse" was defined in 1990 as the level of repetitive microtrauma sufficient to overwhelm the tissues' ability to adapt, therefore leading to injury.[28]

In 1990, a report addressed to the Industrial Injuries Advisory Council echoed these views and stated "The cause and nature of tennis elbow are still subjects for speculation".[29]

In 1993, a study reported that the ultimate cause of lateral epicondylitis is a combination of the duration and overall intensity of arm use. It identified the common extensor origin, specifically the extensor carpi radialis brevis, as the most frequently implicated structure responsible for lateral epicondylitis.[30]

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 patients.[31]

More recently in 2002, a study observed "Lateral epicondylitis or tennis elbow is a common condition that has been widely researched but not yet fully understood. It is difficult to confidently determine the location of pathology causing pain". It identified that the muscle to the middle finger of the extensor digitorum communis is the only segment of that muscle to take origin from the lateral epicondyle and postulated that "disease within this muscle" may be the cause of symptoms in patients with a positive Maudsley's test.[32]

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 orbicular ligament, and so forth. The most widely held theory is that there are macroscopic or microscopic tears in the common tendon.[33,24]

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.[27]

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.[34]

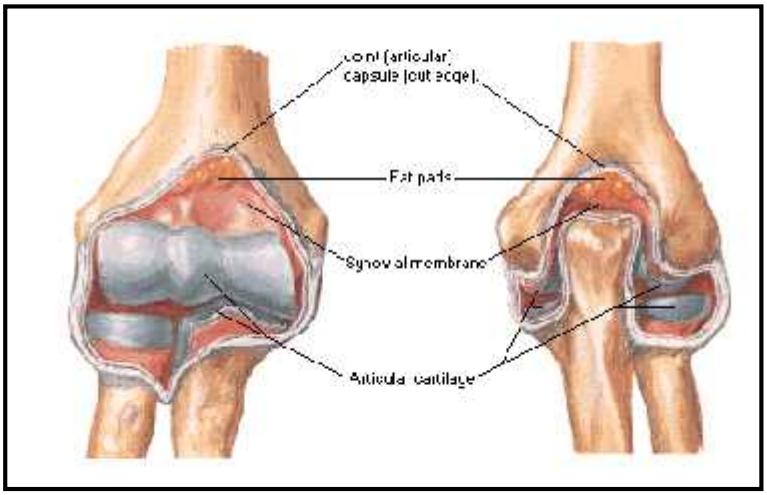


Fig. 1 : Elbow Joint [Opened]
Anterior and Posterior views

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, is tilted 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 sellar surface, largely concave transversely, convex anteroposteriorly.

Swing is therefore accompanied by screwing and conjunct rotation. The olecrenon and coronoid parts of the trochlear notch are usually separated by rough srip, devoid of articular cartilage and covered by fibroadipose tissue and synovial membrane. The capitullum and the radial head are reciprocally curved, closest contact occurs with a semiflexed radius in midpronation. The rim of the head, which is more prominent medially, fits the grove between humeral capitulum and trochlea.

Since the humero-ulnar and humero-radial articulations form a largely uniaxial joint, the ligaments are capsular, ulnar and radial collateral.[34]

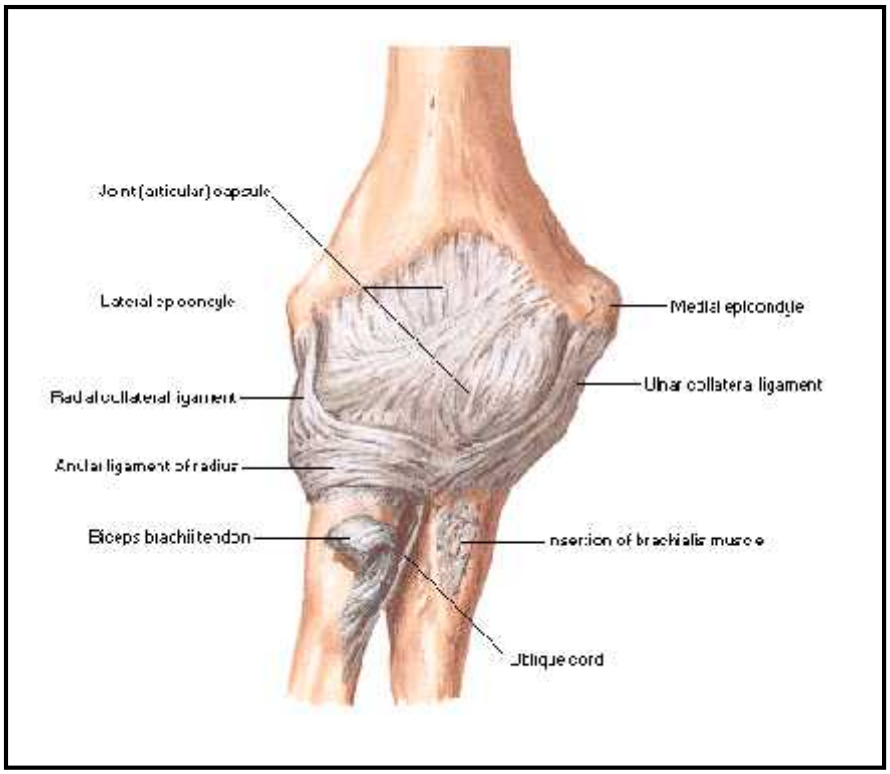


Fig. 2 : Ligaments of Elbow Right Elbow – Anterior View

Fibrous capsule

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 continues 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 humerous behind its capitullum and near its lateral trochlear margin, to all but the lower part of the edge of the olecrenon fossa, and to the back of the medial epicondyle. Inferomedially it reaches the superior and lateral margins of the olecrenon 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.[34]

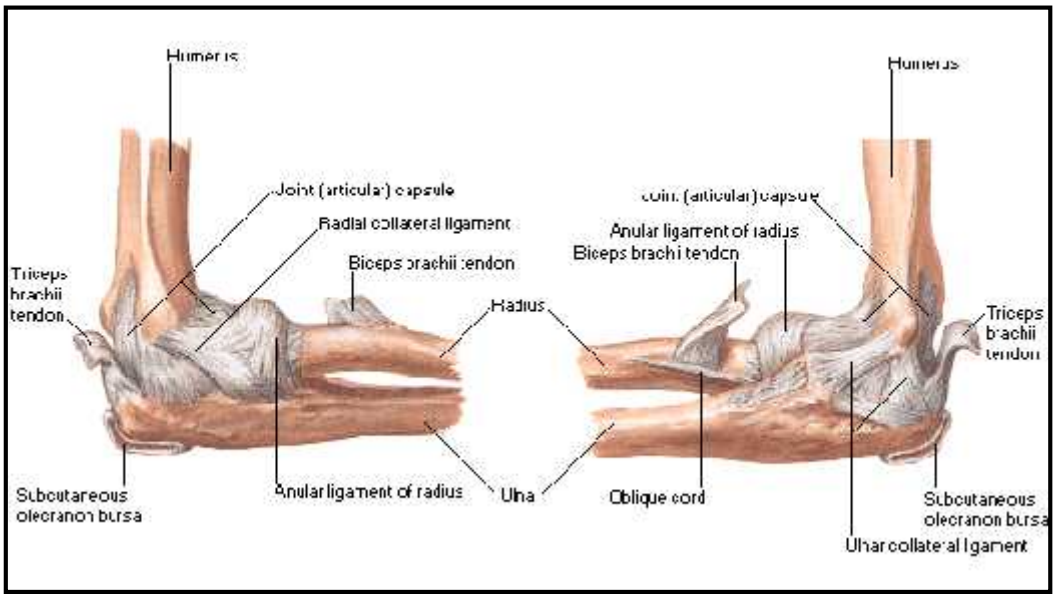


Fig. 3 : Ligaments of Elbow in 90°
Lateral and Medial Views

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.[34]

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.[34]

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 Anconæus, 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.[34]

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 with those of pronation and supination of the hand, which is ensured by the two 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 of 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 axes 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 Anconæus, 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 freest rotatory 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.[34]

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 enthesis.[35]

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. [35]

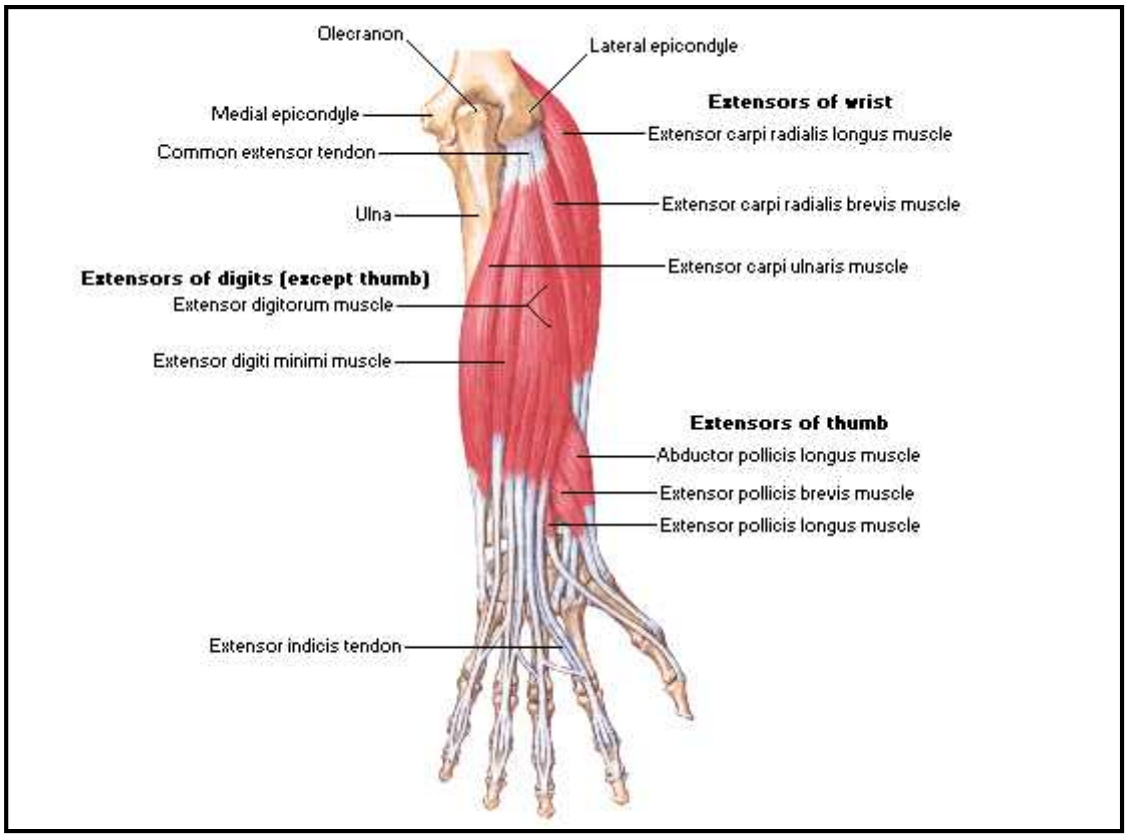


Fig. 4 : Common extensor origin from lateral epicondyle

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.[3] 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. [33]

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.[36]

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.[37,38,39,40,41]

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.[37]

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.[42] 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 haemopoitic 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.[37,42,44] 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.[11,31,37,44,45,46,47,48,49] This constellation of findings has been termed

angiofibroblastic hyperplasia.[18] Regardless of what it is called, tendinosis is the result of failed tendon-healing after repetitive microtrauma.[11]

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.[24,42,50,51,52] 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.[53] Thus, the terms epicondylitis and tendinitis are misnomers.[7,11,15,50] 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.[37]

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 edematous.

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.[11]

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

A study described an invasion of round cells, fibroblasts, and vessel tufts as being pathognomonic of epicondylitis.[52]

"Tendinosis" can be described as the disruption of normally orderly tendon fibers by a characteristic pattern of invasion by fibroblasts and atypical granulation tissue.[27,50,53] It is also described as an immature reparative process set in a background of focal hyaline degeneration.[42]

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.[54,44]

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.[10,11,55]

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.[56,57] 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.[8]

Fig 5 : Tenderness just distal to lateral epicondyle

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.

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.

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.

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.

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.

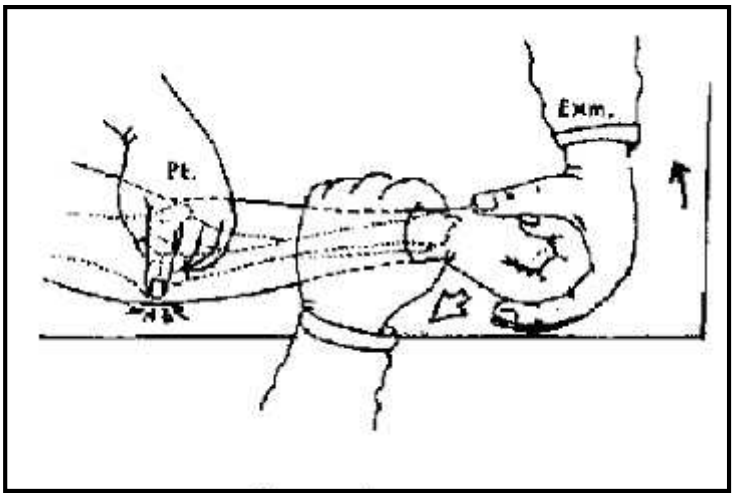
Jug Test: Patient is asked to lift a jug full of water, holding the mouth from above, in a positive test pain will be felt at the lateral epicondylar region.

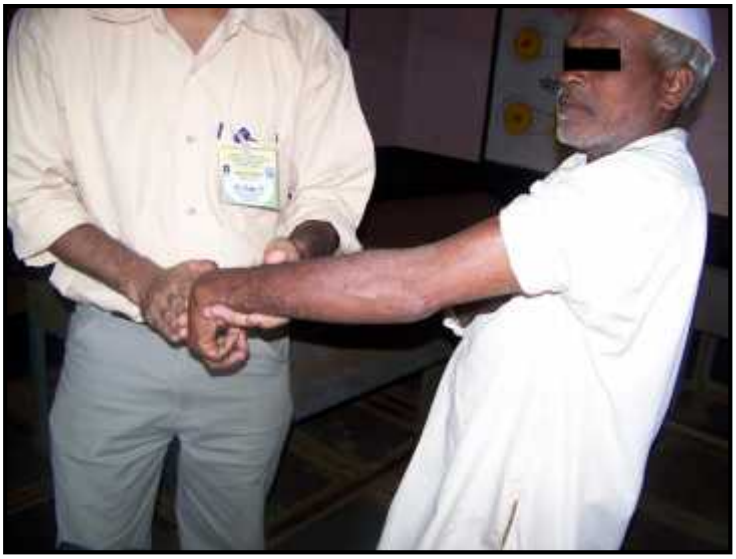
Wringing Test: Patient is asked to wring a towel; pain is felt at the lateral epicondylar region in a positive test.

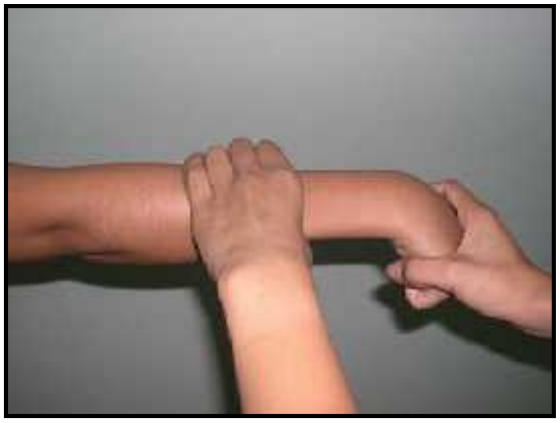
Diagnosis:

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

Fig. 6 : Cozen's test







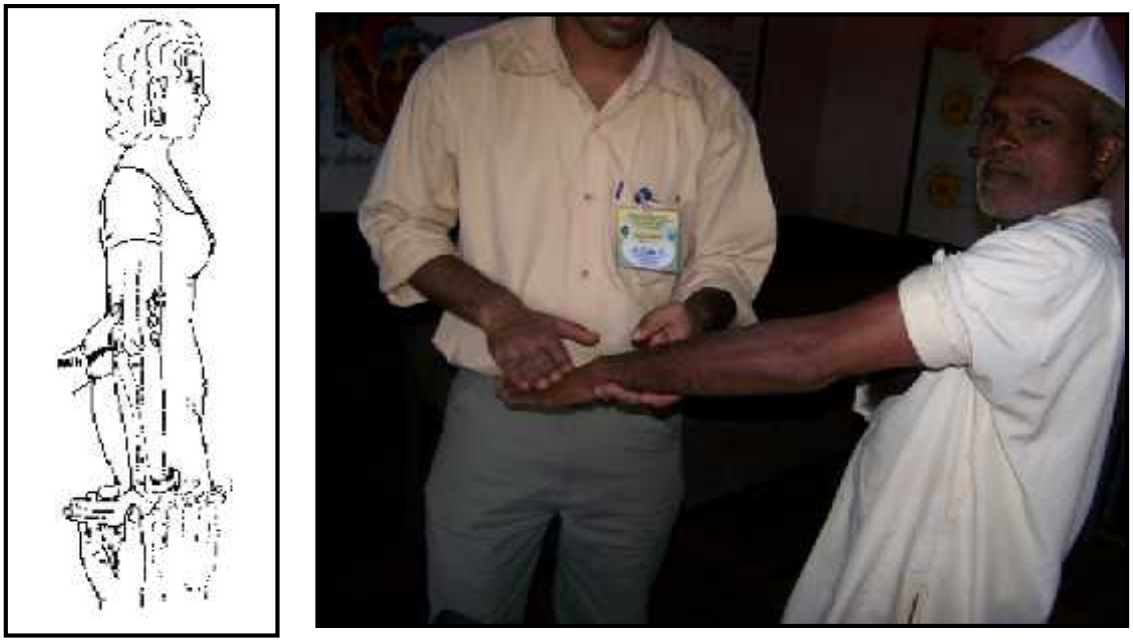


Fig. 7 : Mill's maneuver

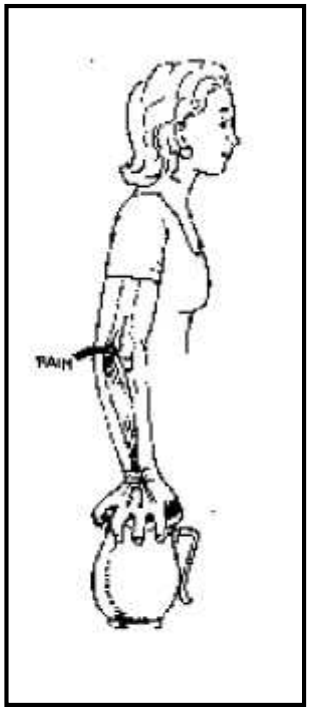


Fig. 8 : Jug test

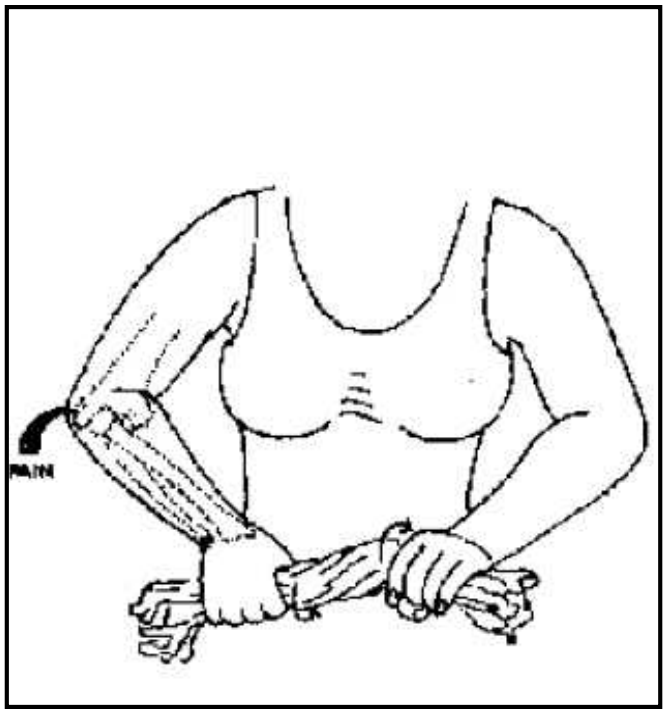


Fig. 9 : Wringing Test

Imaging Studies: whenever necessary

- **Radiographs** can be helpful in ruling out other disorders or concomitant intra-articular 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.

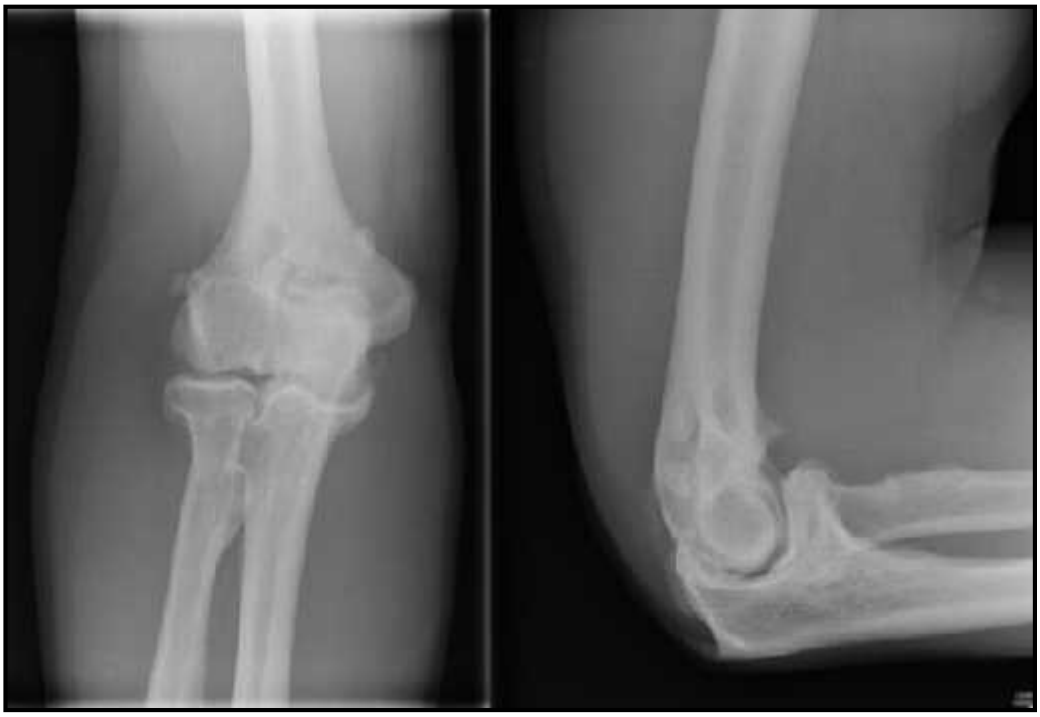


Fig. 10 : X-ray anterio-posterior and lateral view of elbow

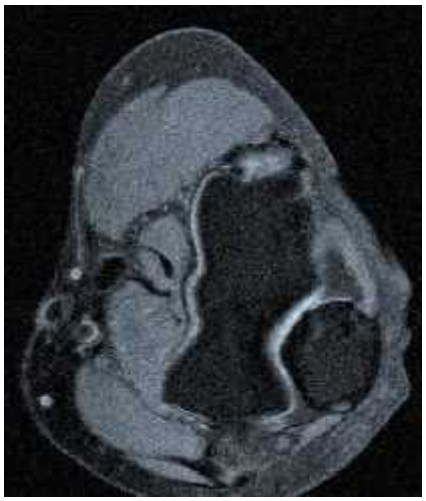


Fig. 11 : Axial T1 MRI Elbow

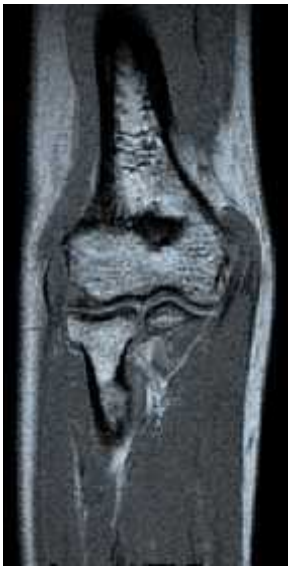


Fig 12 : Coronal T1 MRI elbow

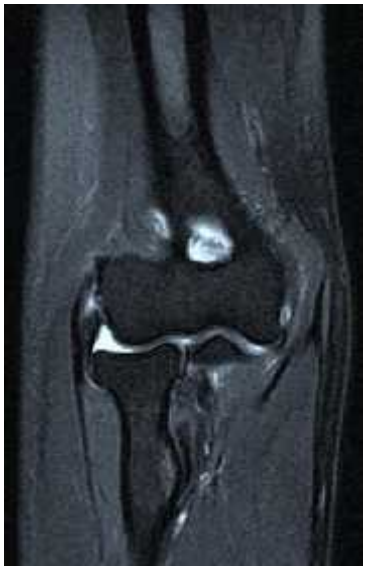


Fig 13 : Coronal T2 fat suppression MRI Elbow

- **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 patient.[15]

Conservative treatment has been the method of choice in the literature for the individual with lateral epicondylitis.[19,58,59]

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).[45]

It includes non steroidal anti-inflammatory drugs, physiotherapy (including inophores 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.[15,16,60,10,17,61,13,14,21,62,63,64,65]

Corticosteroid injection is usually preferred. Yet no definitive type of treatment is universally endorsed.[15]

In operative treatment is are considered only when failure of conservative methods for 6-12 months.[35,61] 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.[66]
- 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.[10,11,18,67,68]

This may be the reason that injections of local steroid have fortuitous lasting benefits.

A study in 1991 concluded "if corticosteroid has any effect on patients with lateral tennis elbow, it is of a short duration".[69]

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.[15]

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.[14]

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%).[70]

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.[13]

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.[71]

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.[72,73] The biomechanical effects of peritendinous corticosteroid on human tendons are unestablished. However, case reports of rupture of tendons after injection are common.[74,75]

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 reactions. [20,62,70,75,76,77] Resuscitation facilities should be available in case patients have a rare severe reaction.[77]

Contraindications to corticosteroid injection in soft tissue lesions[77,78]

- 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.[77]

Local anaesthetic is usually mixed with the corticosteroid in soft tissue injections to make the procedure more comfortable for patients and increase the volume of the injection for wider dispersion.[60,76,77] However, some manufacturers advise against mixing drugs because of the theoretical risk of clumping and precipitation of steroid crystals. There is no evidence that injection of local anaesthetic before the corticosteroid is beneficial, nor of a difference in outcome between long acting and short acting anaesthetics.[60]

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. [10,11,18,67,68]

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.[79]

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.[17]

A study in 2006 demonstrated the autologous blood injection technique has been used successfully in the treatment of medial epicondylitis. [80]

METHODOLOGY

Injection of autologous blood is independent variable and pain at lateral epicondyle is dependent variable.

Source of Data

All confirmed patients of lateral epicondylitis willing for the treatment attending KLES Dr. Prabhakar Kore Hospital & MRC, Belgaum, from January 1st, 2007–December 31st, 2007.

Method of Collection of Data

1. By interview & examination
2. By follow-up of total 6 months. It is divided in to intervals at 1week, 4week, 12 week and 6 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.

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

Cases of lateral epicondylitis, men and women above fifteen years of age with a pain in the lateral aspect of elbow, tenderness over the common extensor origin, and a positive Mills' sign, were included in the study.

Exclusion Criteria

1. Patients receiving steroid injections within three months before blood injection.
2. A history of substantial trauma.
3. Previously treated by surgery for lateral epicondylitis.
4. Other causes of elbow pain such as osteochondritis dessicans of capitellum, lateral compartment arthosis, varus instability, radial head arthritis, posterior interosseous nerve syndrome, cervical disc syndrome, synovitis of radiohumeral joint, cervical radiculopathy, fibromyalgia,Osteoarthritis of elbow, Carpel tunnel syndrome.

Procedure

Group A / autologous blood injection group:

Patients were infiltrated with a injection of 2 milliliters autologous blood drawn from contralateral cubital vain and 1 milliliters 0.5% Bupivacaine, 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) mixed with 1 milliliters 0.5% Bupivacaine, 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 and mixed with 1 milliliter of 0.5% bupivacaine. 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-anaesthetis mixture intraarticularly. 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 milileter local corticosteroid (Methyl prednisolone acetate 80mg) mixed with 1milliliter 0.5% bupivacaine in the same technique as described above.

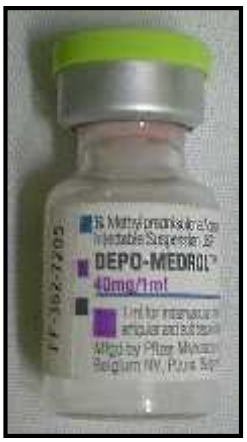


Fig. 14 : Methyl prednisolone acetate

Fig. 15 : 0.5% bupivacaine

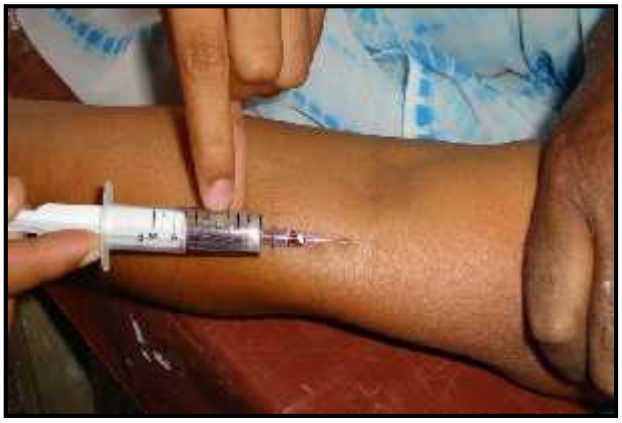


Fig. 16 : Autologous blood (2 ml) is drawn from contra lateral upper limb vein

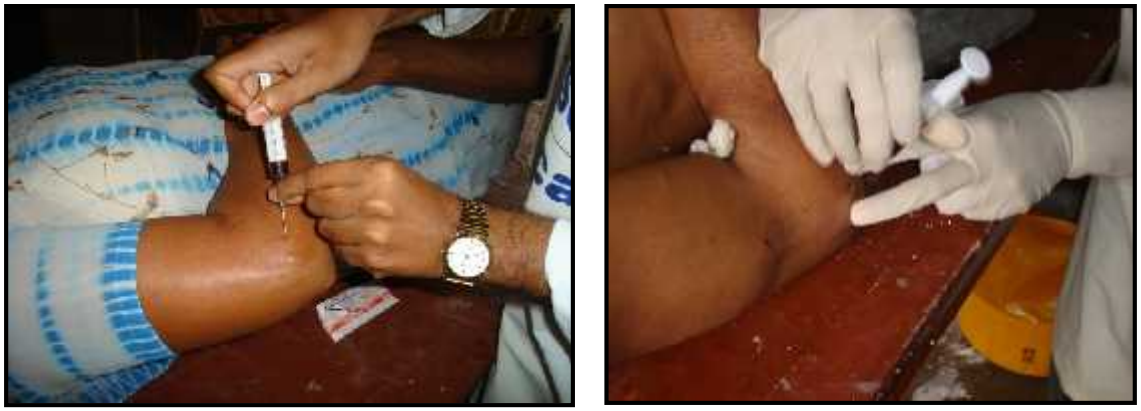


Fig. 17 : Autologous blood injection at lateral epicondyle

Outcome evaluation

Outcome is measured using 'Pain score' and 'Nirschl staging of lateral epicondylitis'. [6,10]

Outcome measures:

1. 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, 1, 4, 12weeks and 6 month of follow-ups. Numerical valve 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.

2. NIRSCHL STAGING:

phase1: mild pain with exercise; resolves within 24 hours

phase2: pain after exercise; exceeds 48 hours

phase3: pain with exercise; does not alter activity

phase4: pain with exercise; alters activity

phase5: pain with heavy activities of daily living

phase6: pain with light activities of daily living; Intermittent pain at rest

phase7: constant pain at rest; disrupts sleeps

No pain_____1 _____ 2_____ 3_____4_____ 5_____ 6 _____ 7 worst pain

Statistical test: **Mann-Whitney U test (non parametric test)** is applied to calculate the significance of results.

OBSRVATIONS AND RESULTS

Procedure was done in 60 patients under the present study. Participants were clinically evaluated. A baseline VAS scores and Nirschl staging 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 1 week, 4 weeks, 12 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 corticosteroid 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.

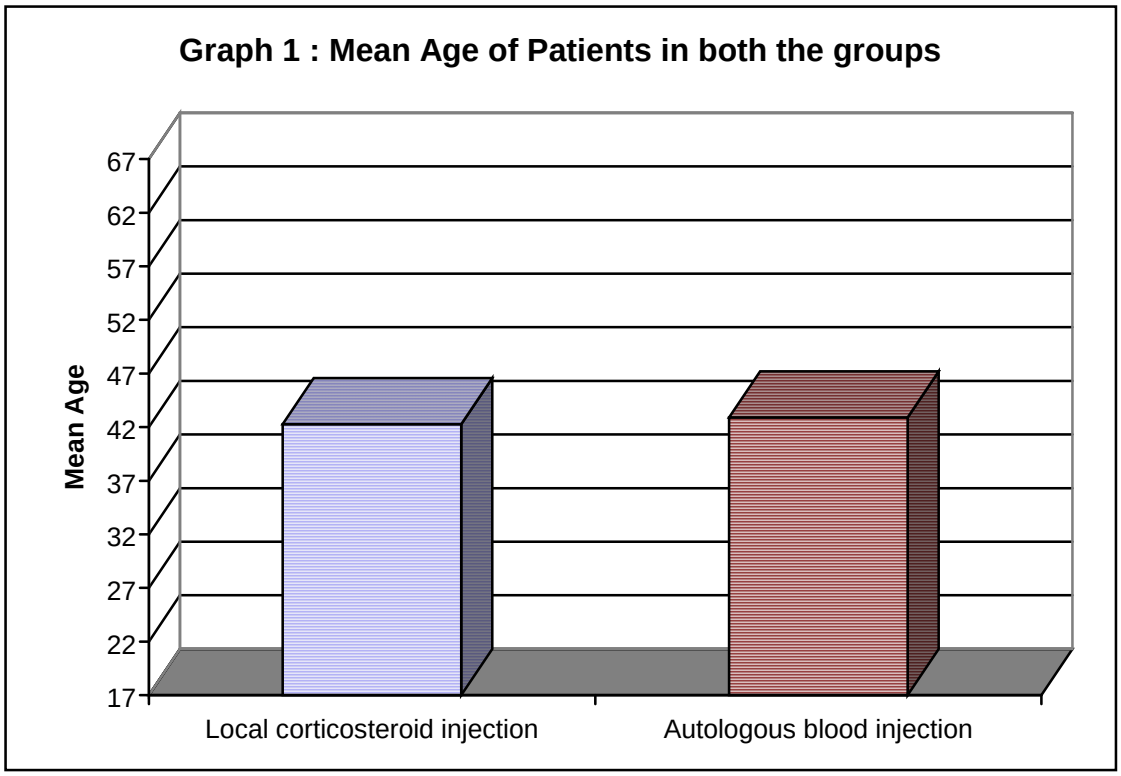

Graph 1 : Mean Age of Patients in both the groups
Mean Age
67
62
57
52
47
42
37
32
27
22
17
Local corticosteroid injection
Autologous blood injection

Table 2 : Sex distribution in both the groups

		Treatment Type		Total
		Local corticosteroid injection	**Autologous blood injection**	
Sex	Male	12	13	25
	Female	18	17	35
Total		**30**	**30**	

Out of the 60 participants, 25 were males and 35 were females. In corticosteroid injection group 12 were males and 18 were females. In autologous blood injection group 13were 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

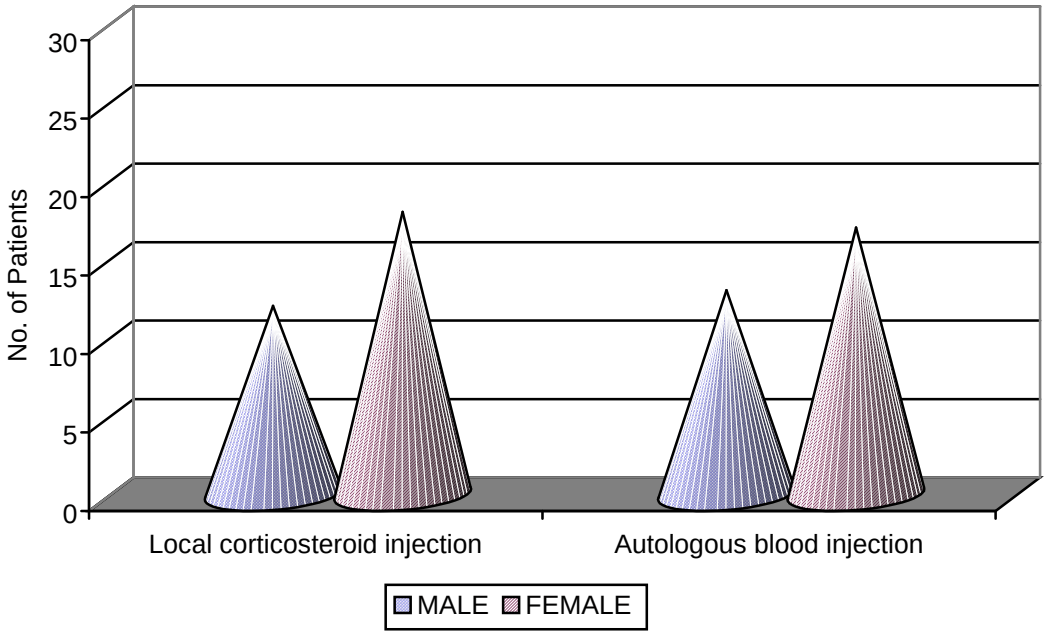


Table No. 3 : Elbow side involved in both groups

		Treatment Type		Total
		Local corticosteroid injection	**Autologous blood injection**	
Side Affected	Right	23	23	46
	Left	7	7	14
Total		**30**	**30**	

Out of the 60 participants, 46 participants had their right side elbow affected and 14 had their left side affected.

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

Graph 3 : Elbow side involved in both the groups

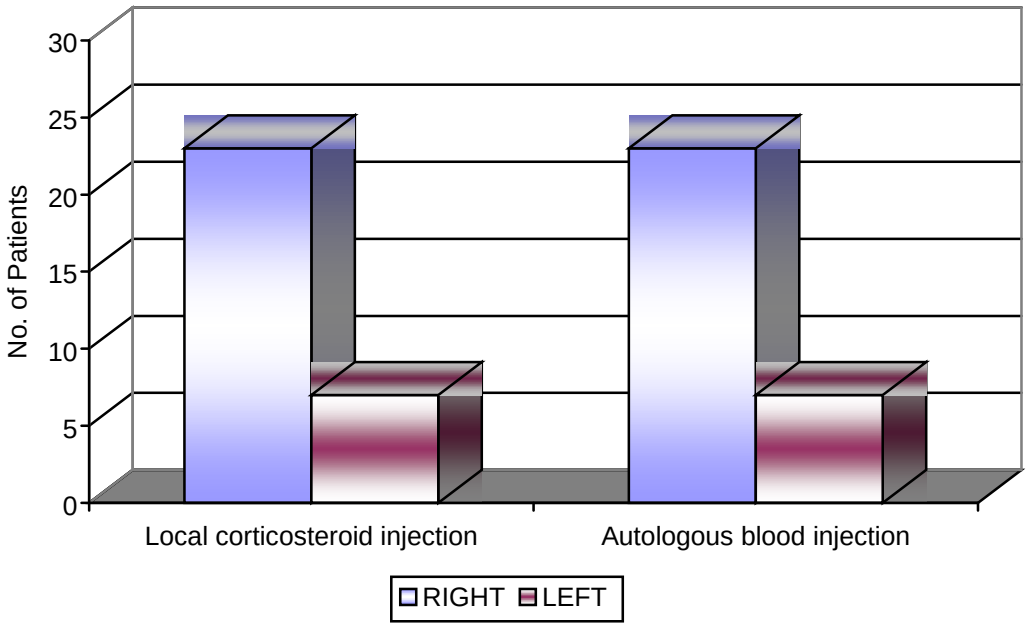


Table No. 4 : Dominance of elbow involved in both groups

		Treatment Type		Total
		Local corticosteroid injection	**Autologous blood injection**	
Dominance	Dominant side involved	26	25	51
	Non-dominant side involved	4	5	9
Total		**30**	**30**	

Out of the 60 participants, 51 participants had their Dominant elbow affected and 9 had their Nondominant elbow affected.

P value= 1 which is non significant. Thus both the groups were comparable in terms of dominance of elbow involved.

Graph 4 : Dominance of elbow involved in both groups

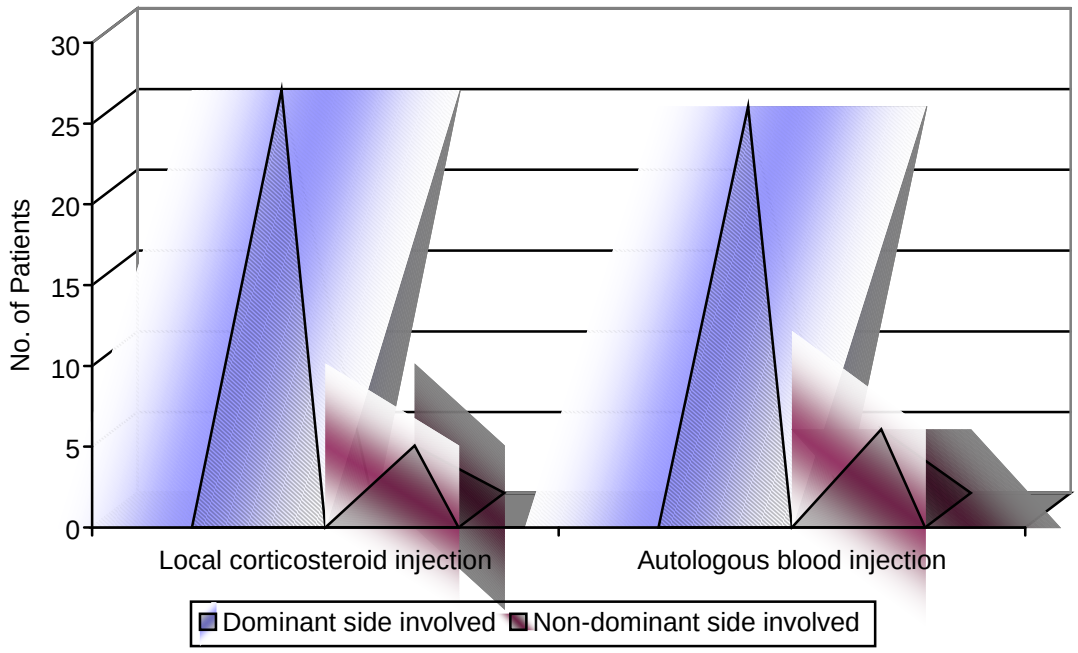


Table no. 5 : Duration of condition in both the groups

	Treatment Type	
	Local corticosteroid injection	**Autologous blood injection**
Mean duration of the condition	7.73	9.5
S.D	6.63	11

The mean duration of the condition in all 60 patients suffering from lateral epicondylitis was 8.62 weeks.

The mean duration of the condition in corticosteroid injection group was 7.73 weeks.

The mean duration of the condition in corticosteroid injection group was 9.5 weeks.

P Value= 0.8283 which is non significant. Thus both the groups were comparable in terms of duration of the condition in each group.

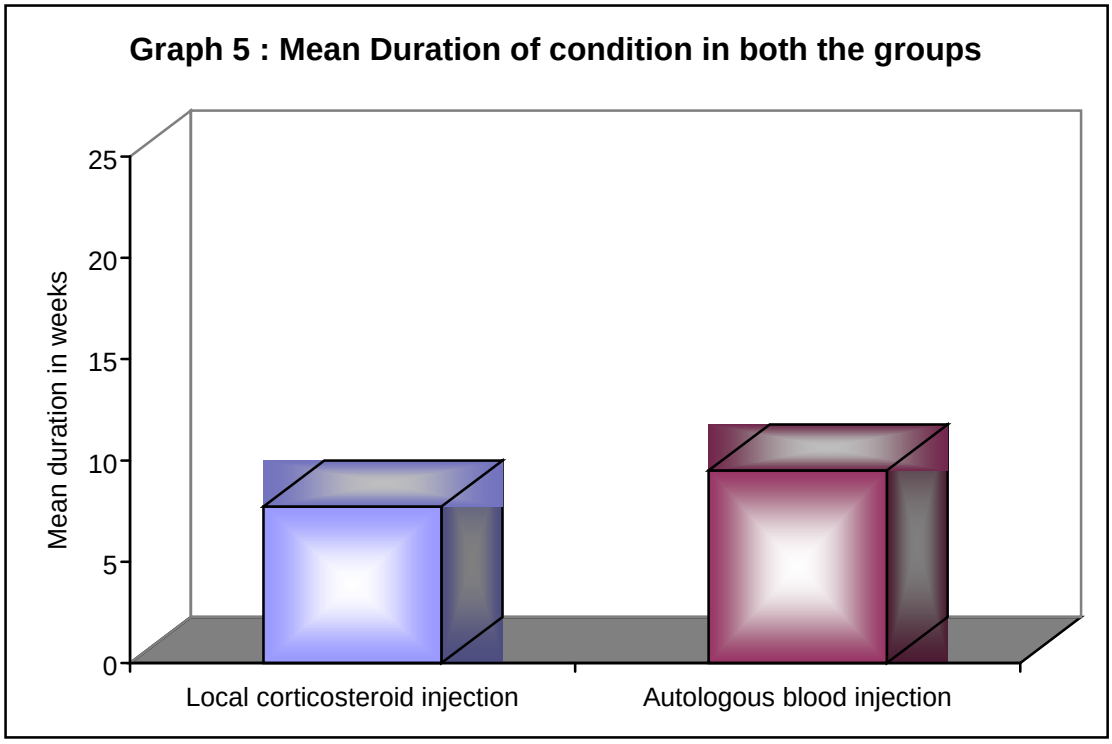

Graph 5 : Mean Duration of condition in both the groups
Mean duration in weeks
25
20
15
10
5
0
Local corticosteroid injection
Autologous blood injection

Table No. 6 : Occupation in both the groups

		Treatment Type		Total
		Local corticosteroid injection	**Autologous blood injection**	
Type of Occupation	Manual work	9	13	22
	Non manual work	21	17	38
	Total	**30**	**30**	

Based on the type of work the patient does at work place, the occupation is categorized as either manual work or non-manual work**.**

p value= 0.3 which is non significant. Thus both the groups were comparable in terms of type work they do at workplace.

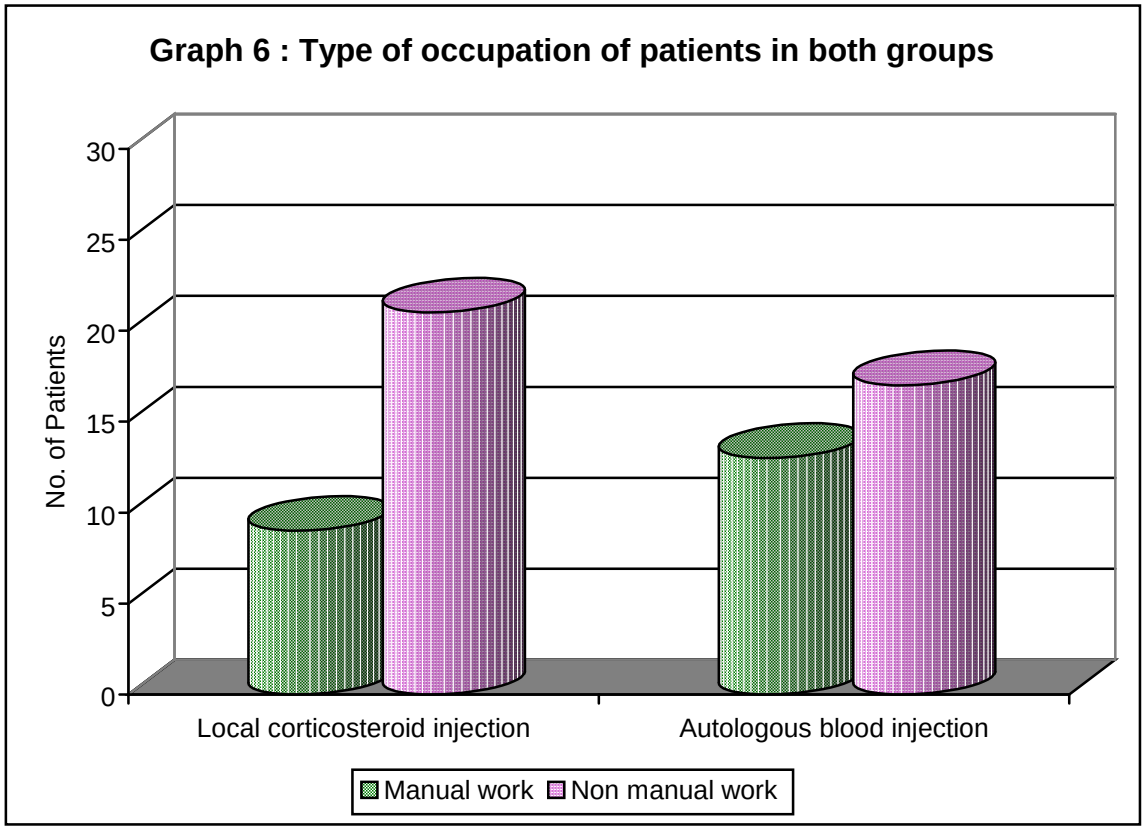

Graph 6 : Type of occupation of patients in both groups
No. of Patients
30
25
20
15
10
5
0
Local corticosteroid injection
Autologous blood injection
Manual work
Non manual work

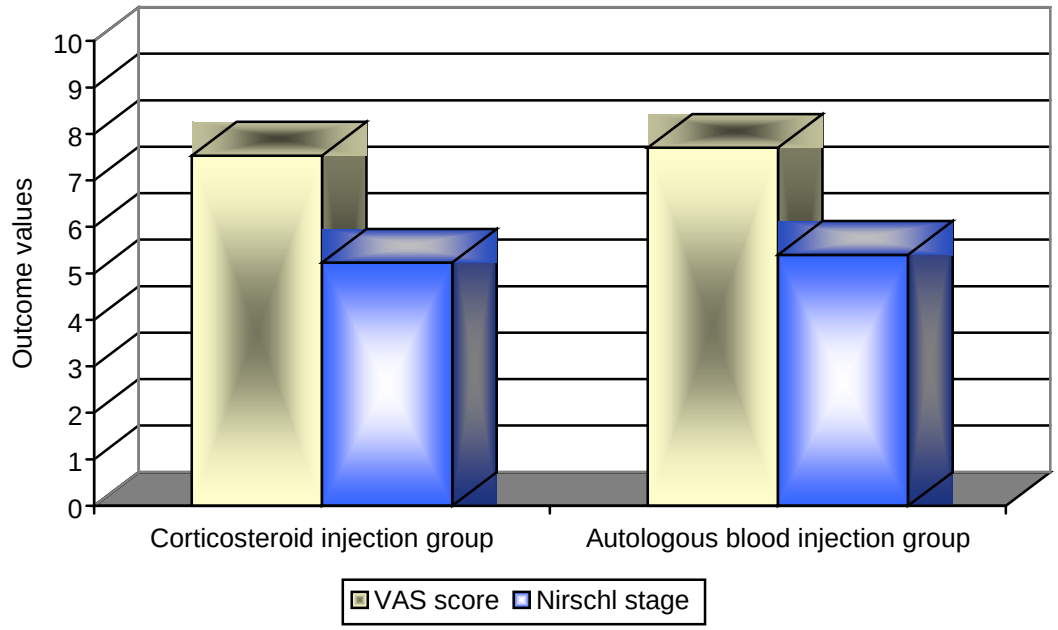
Table No. 7 : Outcome values at the time of Presentation (before injection)

	Local corticosteroid injection	Autologous blood injection	P Value	Inference
Mean VAS Score	7.533	7.7	0.5395	N.S
S.D	1.2794	1.3429		
Mean Nirschl stage	5.23	5.4	0.4918	N.S
S.D	0.9714	1.132589		

P value for VAS Score is 0.5395 and P value for Nirschl score is 0.4918 which are statistically not significant.

Hence the outcome values before the injection are comparable.

Graph 7 : Outcome values at the time of Presentation (before injection)



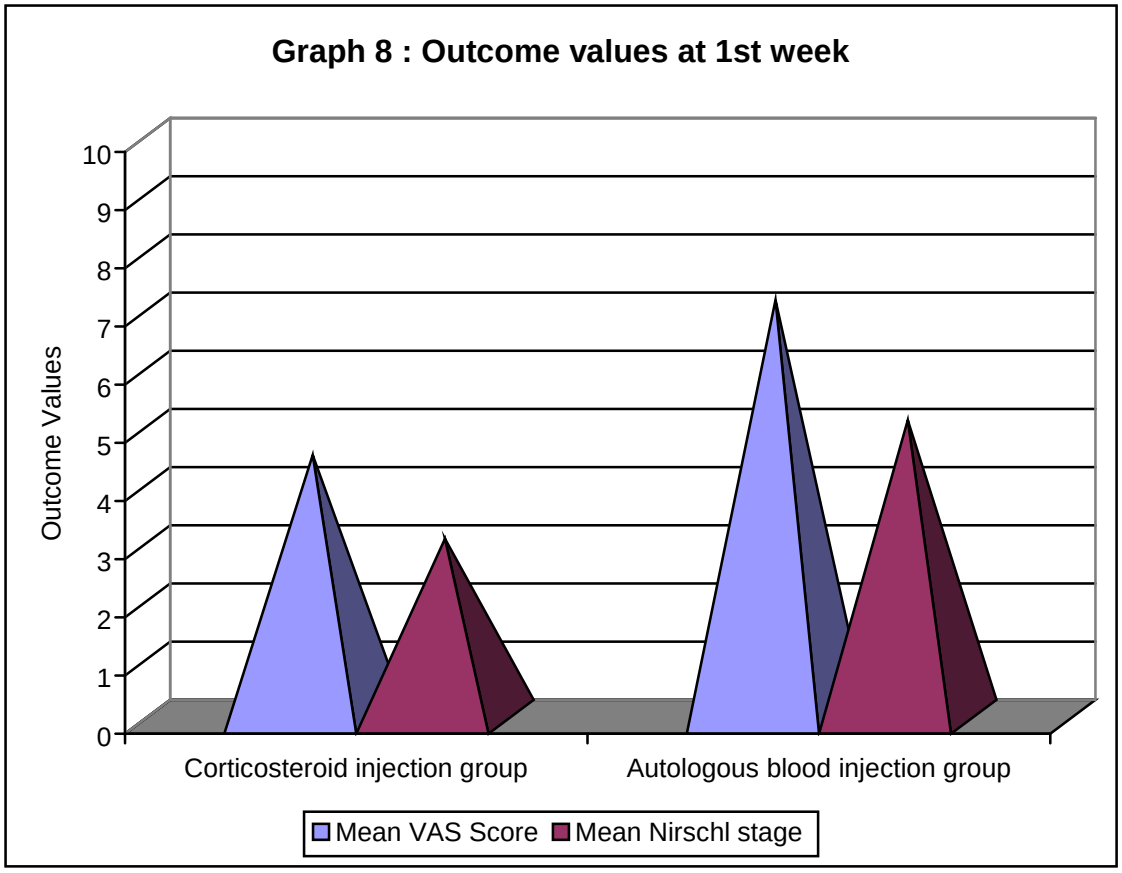
First follow up at 1st week

Table No. 8 : Outcome values at 1st week

	Corticosteroid injection group	Autologous blood injection group	P Value	Inference
Mean VAS Score	4.5	7.166	<0.0001	S
S.D	1.8708	1.931291		
Mean Nirschl stage	3.06	5.1	<0.0001	S
S.D	1.3629	1.470398		

P value for VAS Score is <0.0001 and P value for Nirschl score is <0.0001 which are statistically significant.

Hence the decrease in pain at 1st week is statistically significant in corticosteroid injection group compared to autologous blood injection group.


Graph 8 : Outcome values at 1st week
Outcome Values
10
9
8
7
6
5
4
3
2
1
0
Corticosteroid injection group
Autologous blood injection group
Mean VAS Score
Mean Nirschl stage

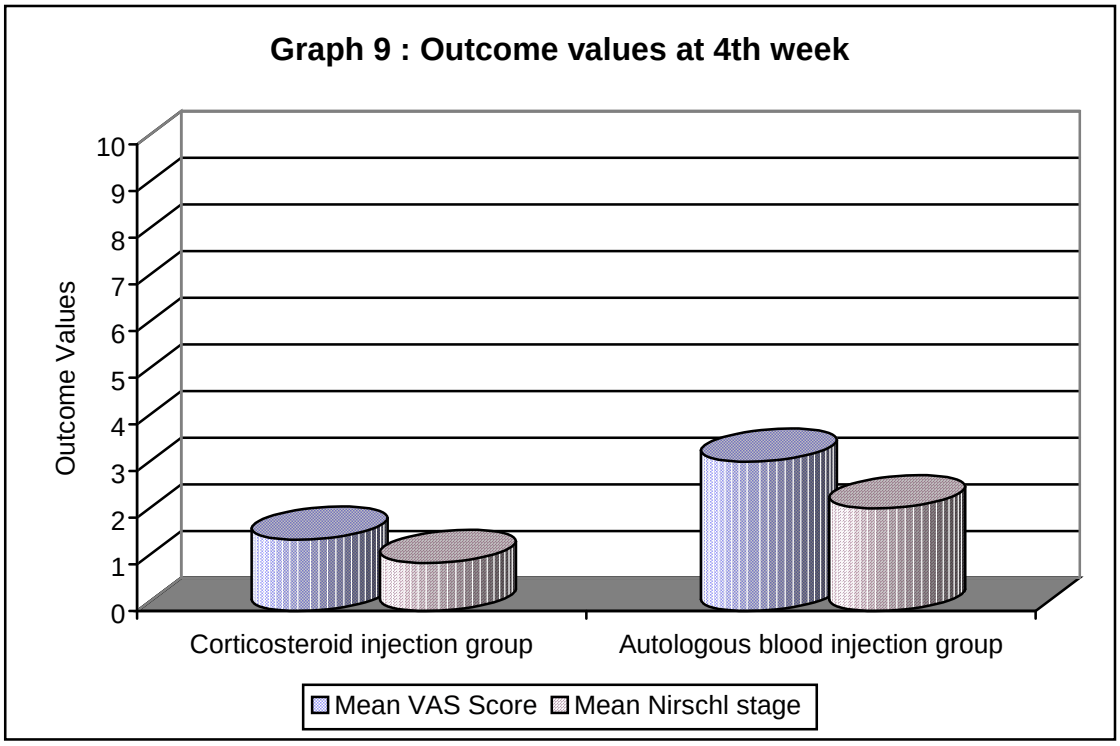
Follow up at 4th week

Table No. 9 : Outcome measures at 4th week

	Corticosteroid injection group	Autologous blood injection group	P Value	Inference
Mean VAS Score	1.53	3.2	0.0022	S
S.D	2.3154	2.369344		
Mean Nirschl stage	1.03	2.2	0.003	S
S.D	1.5862	1.648406		

P value for VAS Score is 0.0022 and P value for Nirschl score is 0.003 which are statistically significant.

Hence the decrease in pain at 4th week is statistically significant in corticosteroid injection group compared to autologous blood injection group.


Graph 9 : Outcome values at 4th week
Outcome Values
10
9
8
7
6
5
4
3
2
1
0
Corticosteroid injection group
Autologous blood injection group
Mean VAS Score
Mean Nirschl stage

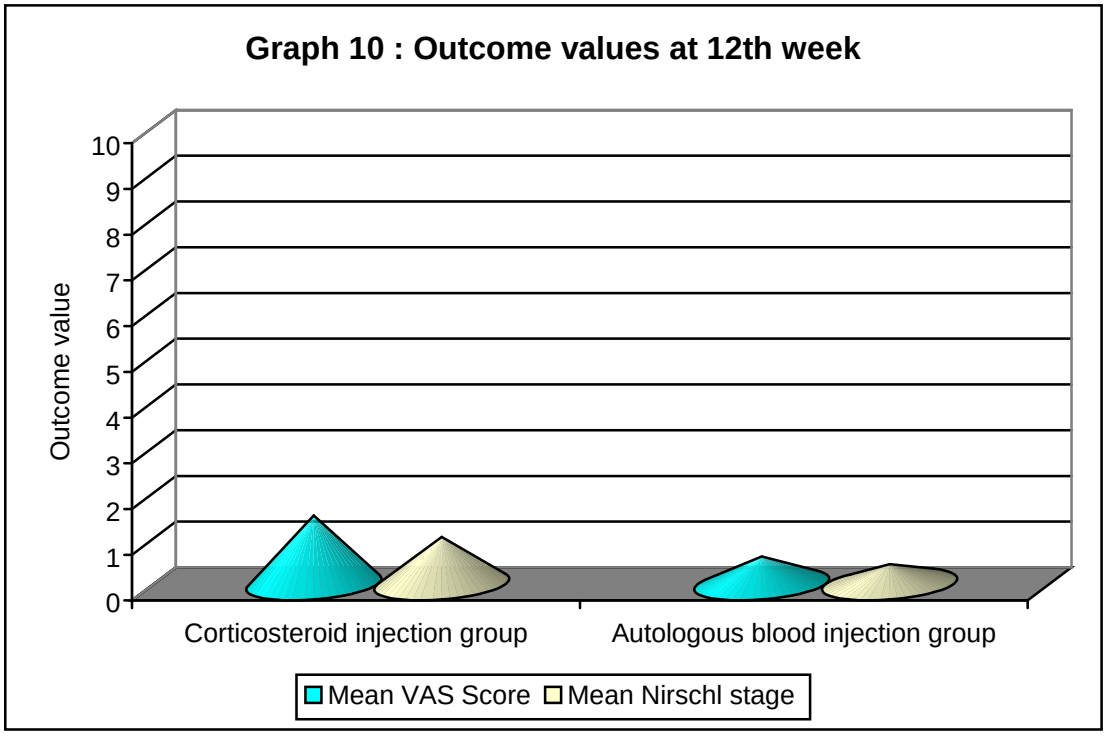
Follow up at 12th week

Table No. 10 : Outcome measures at 12th week

	Corticosteroid injection group	Autologous blood injection group	P Value	Inference
Mean VAS Score	1.5	0.6	0.0127	S
S.D	1.8147	1.904622		
Mean Nirschl stage	1.03	0.433	0.0184	S
S.D	1.3257	1.278019		

P value for VAS Score is 0.0127 and P value for Nirschl score is 0.0184 which are statistically significant.

Hence at 12th week the decrease in pain is statistically significant in autologous blood injection group compared to corticosteroid injection group.


Graph 10 : Outcome values at 12th week
Outcome value
10
9
8
7
6
5
4
3
2
1
0
Corticosteroid injection group
Autologous blood injection group
Mean VAS Score
Mean Nirschl stage

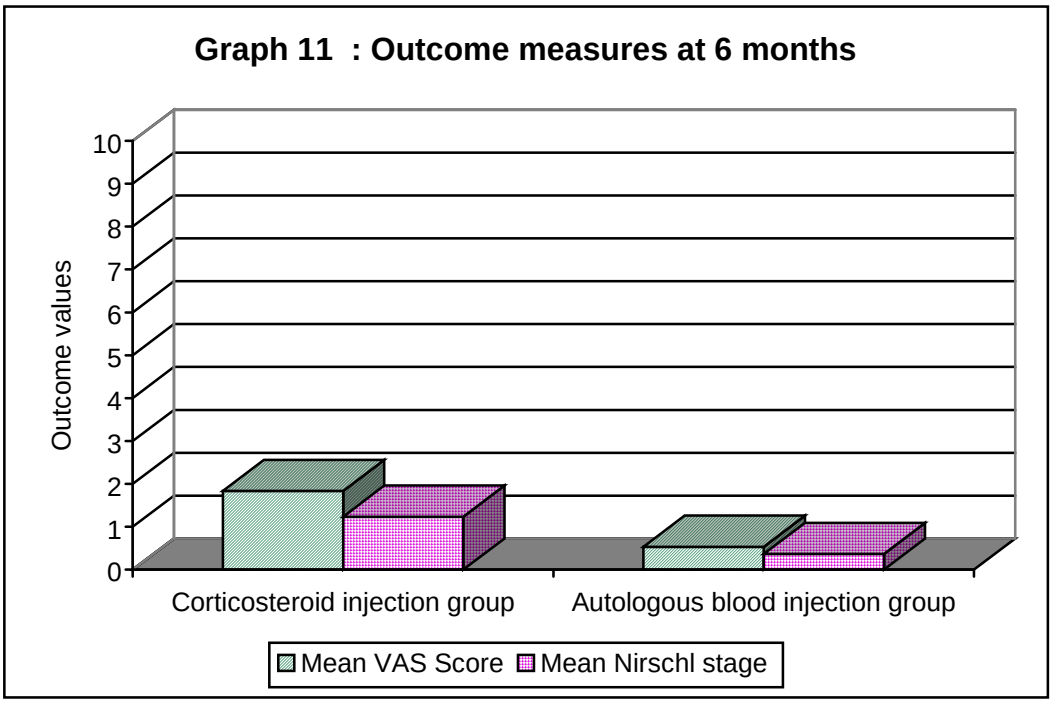
Follow up at 6 month

Table No. 11 : Outcome measures at 6 months.

	Corticosteroid injection group	**Autologous blood injection group**	**p Value**	**Inference**
Mean VAS Score	1.833	0.533	0.0058	S
S.D	2.0356	1.907035		
Mean Nirschl stage	1.233	0.366	0.0064	S
S.D	1.4308	1.272612		

P value for VAS Score is 0.0058 and P value for Nirschl score is 0.0064 which are statistically significant.

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


Graph 11 : Outcome measures at 6 months
Outcome values
10
9
8
7
6
5
4
3
2
1
0
Corticosteroid injection group
Autologous blood injection group
Mean VAS Score
Mean Nirschl stage

The severity of pain during the day at baseline and during followup at 1week, 4weeks, 12weeks and 6 months.

Table No. 12 : Mean VAS score for the two groups

	Corticosteroid injection group		**Autologous blood injection group**			
Followup 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
1st week	4.5	1.87	7.166	1.931	<0.0001	S
4 weeks	1.533	2.315	3.2	2.369	0.0022	S
12 weeks	1.5	1.814	0.6	1.904	0.0127	S
6 month	1.833	2.035	0.533	1.907	0.0058	S

Graph 12 : Mean VAS score pre- and post-procedure at 1week, 4weeks, 12weeks and 6 months

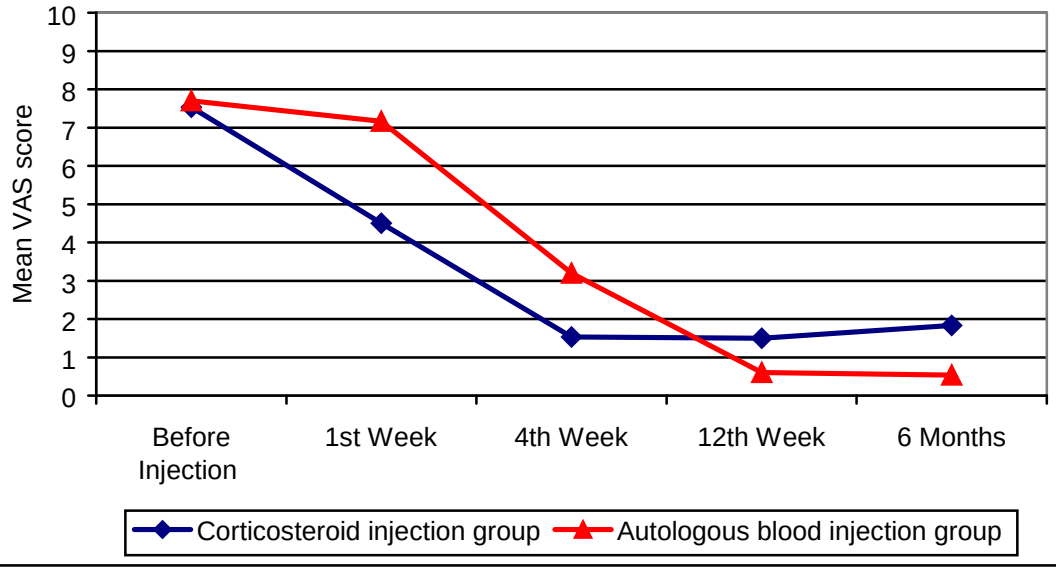


Table No. 13 : Mean Nirschl staging for the two groups

Followup period	Corticosteroid injection group		Autologous blood injection group		P Value	Inference
	Mean Nirschl Staging	S.D	Mean Nirschl Staging	S.D		
Before injection	5.2333	0.9714	5.4	1.132589	0.4918	N.S
1st week	3.0667	1.3629	5.1	1.470398	<0.0001	S
4 weeks	1.0333	1.5862	2.2	1.648406	0.003	S
12 weeks	1.0333	1.3257	0.433333	1.278019	0.0184	S
6 month	1.2333	1.4308	0.366667	1.272612	0.0064	S

Graph 13 : Mean Nirschl stages pre- and post-procedure at 1week, 4weeks, 12weeks and 6 months

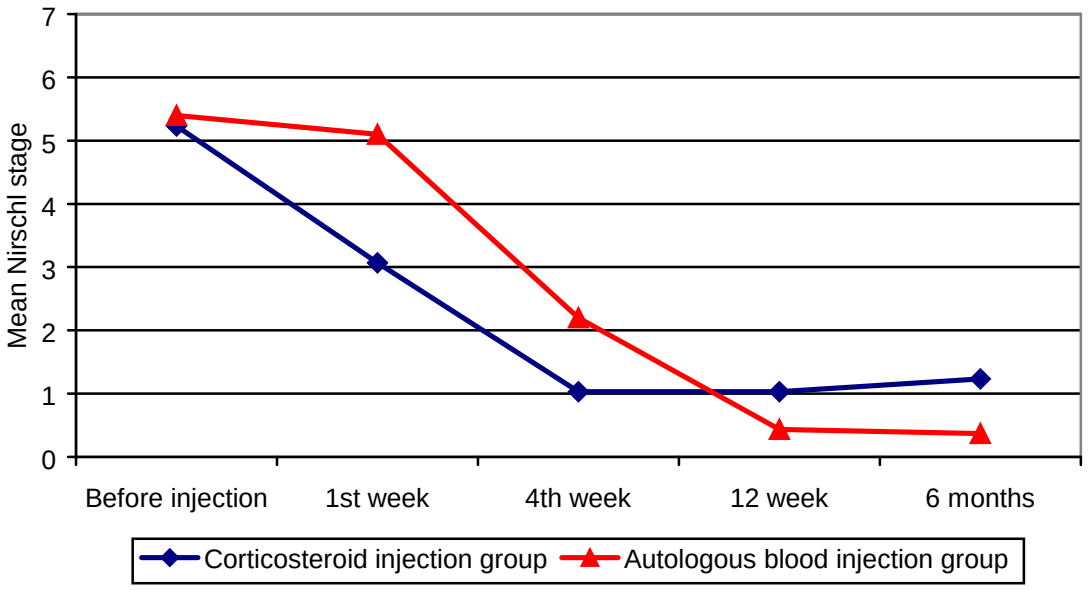


Table No. 14 : Maximum benefit reached in weeks

	Corticosteroid injection group	**Autologous blood injection group**
Maximum benefit reached at an average weeks	6.1	11.43
S.D	4.95	5.709

Maximum benefit reached at an average of 6.1weeks in corticosteroid injection group.

Maximum benefit reached at an average of 11.43weeks in autologous blood injection group.

P value= 0.0003 which is significant. Thus corticosteroid injection group shows statistically significant early maximum benefit.

Graph 14 : Maximum benefit reached in weeks

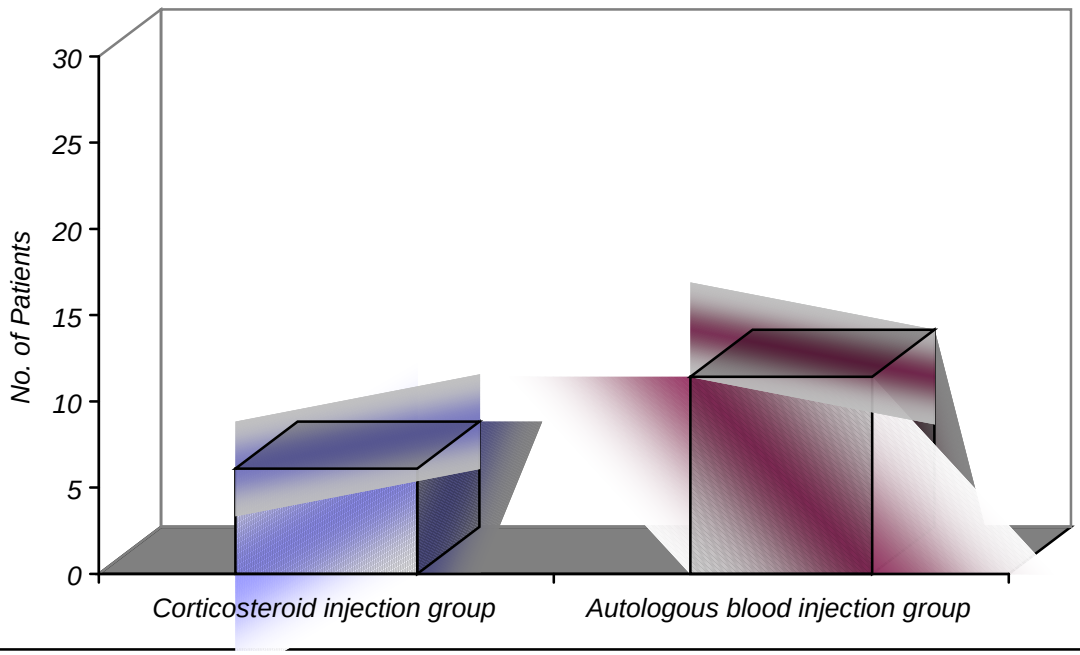


Table No. 15 : Recurrence rate

	Corticosteroid injection group	Autologous blood injection group
Number of patients completely relieved of pain at 4 week	19	5
Number of recurrences	7	0
Recurrence rate	36.8	0

Up to 4 weeks there was significant improvement with 63.3% of patients completely relieved of pain in patients treated with a corticosteroid injection and 16.66% in autologous blood injection group.

Many of these patients reported recurrences at 6month follow up. The rate of recurrence was 36.8% in corticosteroid injection group.

The rate of recurrence was 0% in autologous blood injection group.

P= <0.001 which is significant. Thus corticosteroid injection group showed statistically significant high recurrence rate compared to autologous blood injection group.

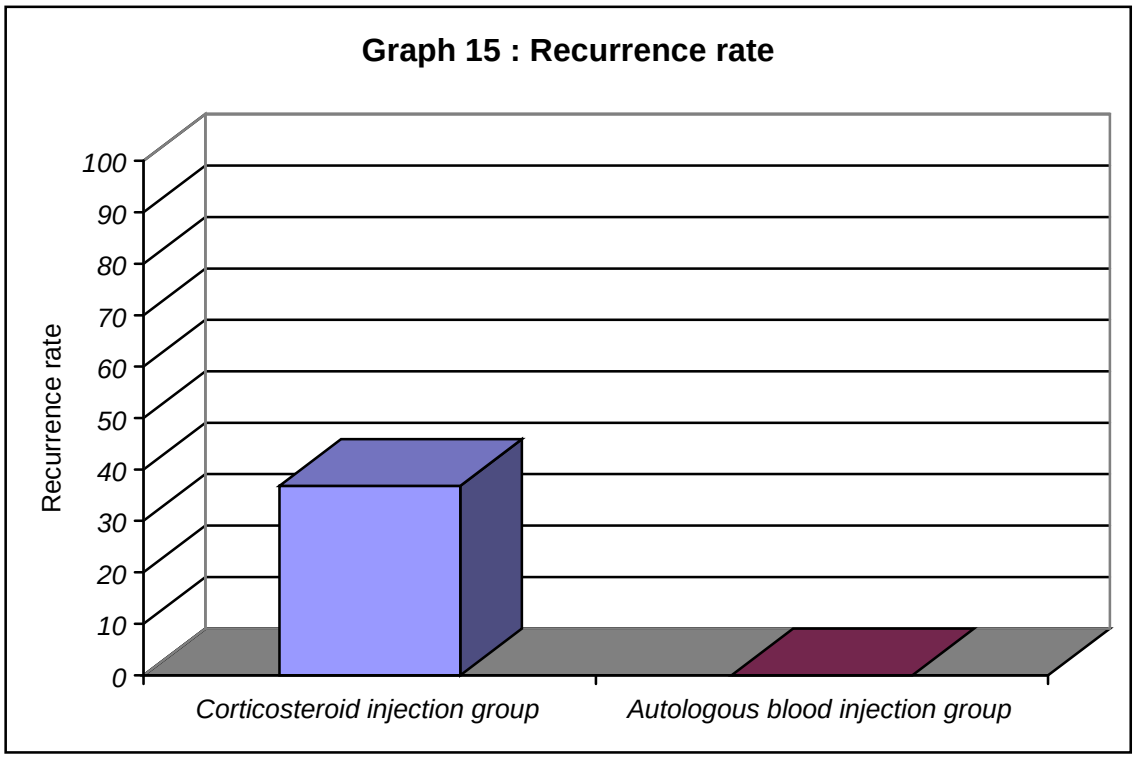

Graph 15 : Recurrence rate
Recurrence rate
100
90
80
70
60
50
40
30
20
10
0
Corticosteroid injection group
Autologous blood injection group

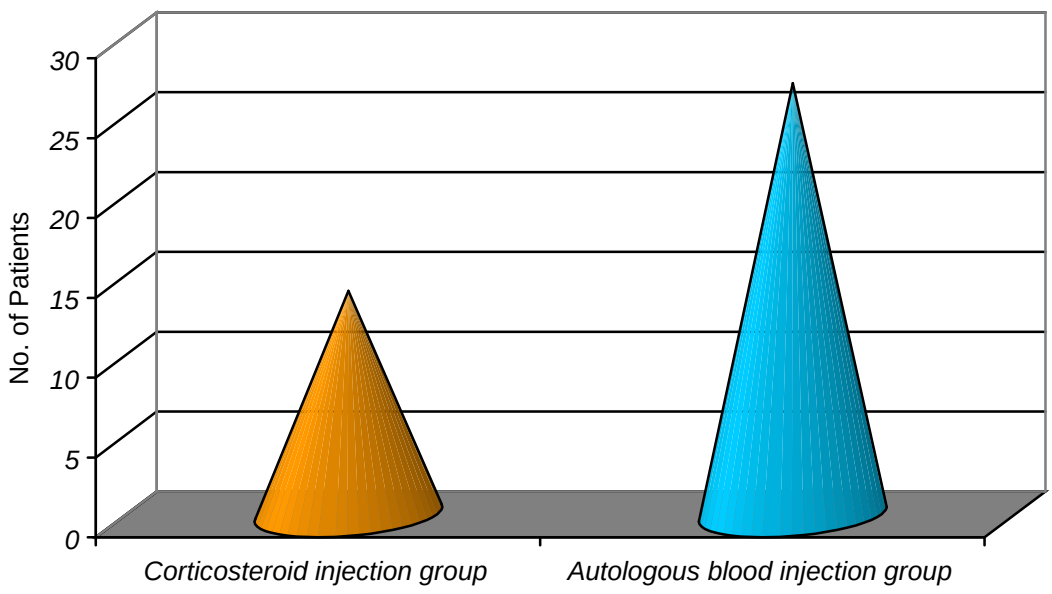
Table No. 16 : Number of patients completely relieved of pain at the end of follow up.

	Corticosteroid injection group	**Autologous blood injection group**
Number of patients completely relieved of pain	14	27

After 6 months of follow up, 14 (46.66%) patients in corticosteroid injection group were completely relieved of pain whereas 27 (90%) participants in autologous blood injection group were completely relieved of pain.

P value= <0.001 which is significant. Thus autologous blood injection group had statistically significant more number of patients completely relieved of pain.

Graph 16 : Number of patients completely relieved of pain



Complications

Table No. 17 : Local skin atrophy

		Treatment type		Total
		Corticosteroid injection	**Autologous blood injection**	
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 17 : Number of patients with post injection skin atrophy

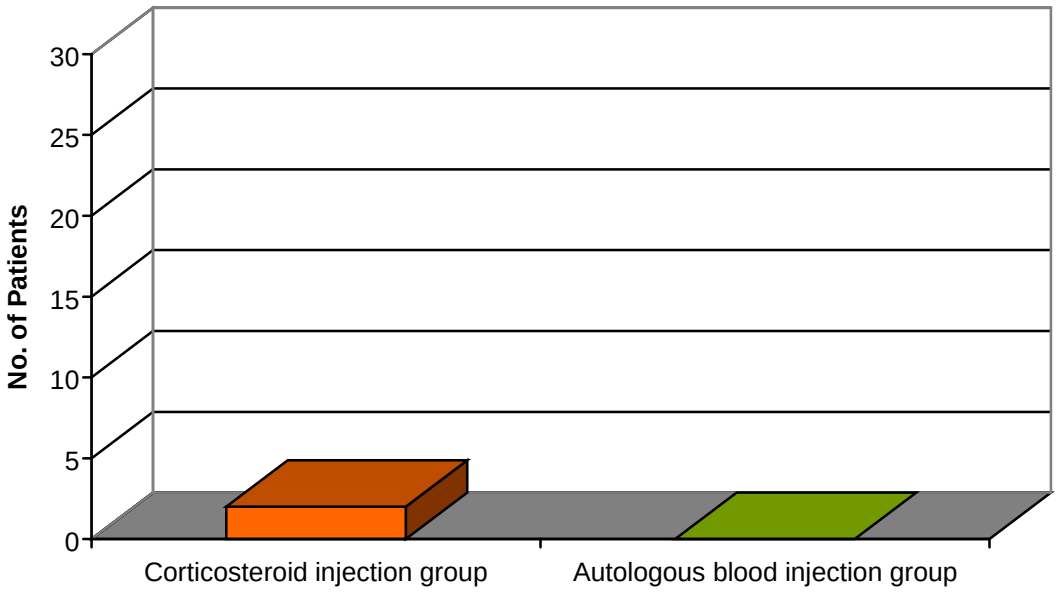


Table No. 18 : Post-intervention exacerbation of pain

		Treatment type		Total
		Corticosteroid injection	Autologous blood injection	
Post-intervention exacerbation of pain	Yes	8	18	36
	No	22	12	34
Total		**30**	**30**	

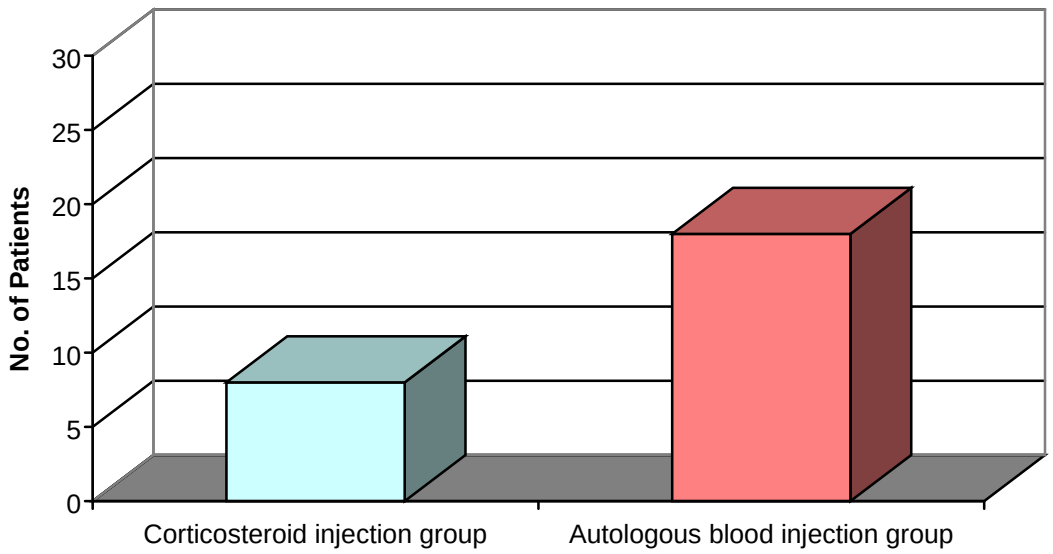
In corticosteroid injection group 8 participants (26%) patients complained of post-intervention exacerbation of pain while in autologous blood injection group 18 participants (60%) complained of increase of pain after local injection. And these patients with this increase of pain after the procedure had to be given analgesics for pain relief as a co-intervention.

P value=0.009 which is significant. Thus autologous blood injection group had statistically significant more post injection exacerbation of pain compared to corticosteroid injection group.

No patients reported elbow stiffness, infection, reflex sympathetic dystrophy, post injection flare, facial flushing, neurovascular damage or tendon rupture or other untoward complications.

.

Graph 18 : Number of patients with post injection exacerbation of pain



DISSCUSION

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.[15] The role of local steroid is debatable.

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.[10,17,79] 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]

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.[10]

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.[70,31,81] one more study had equal number of males and female patients.[10] 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.[81,10]

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 and Nirschl staging before injection in both the groups were 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; mean Nirschl staging for steroid injection group was 5.2333, mean Nirschl staging for autologous blood injection group was 5.4, P value was 0.4918.

Till 4 weeks follow up, statistically significant difference between the two groups with VAS scoring and Nirschl staging was seen. Corticosteroid injection group showed statistically significant decrease in VAS score and Nirschl stage at 1st week, 4th week compared to autologous blood injection group. One study showed similar results with local corticosteroid injection group, when compared with oral naproxen.[70]

At 12th week and at 6 month follow up autologous blood injection group showed statistically significant decrease in VAS score and Nirschl staging compared to

corticosteroid group. At 6 months follow up, mean VAS score for steroid injection group=1.833, mean VAS score for autologous blood injection group was 0.533, P value was 0.0058; mean Nirschl staging for steroid injection group was 1.2333, mean Nirschl staging for autologous blood injection group was 0.366667, P value was 0.0064.

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.[10]

In Corticosteroid injection group till 4 weeks there was significant improvement with 63.3% of patients completely relieved of pain. Many of these patients reported recurrences at 12 weeks 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.[70]

In autologous blood injection group at 4^{th} week 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.[67]

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 atophy. ($p = 0.150$) 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 long term 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 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 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 VAS scoring and Nirschl staging.

- 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 1st week, 4th week, 12th week and 6 months.

- Outcome is measured using 'Pain score' and 'Nirschl staging of lateral epicondylitis'.

- 'Mann-Whitney U test' (non parametric test) is applied to calculate the significance of results.

- At 1st week and 4th week the corticosteroid injection group showed a statistically significant decrease in pain compared to autologous blood injection group.

- At 12th week 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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INFORMED CONSENT FORM FOR PARTICIPATION IN RESEARCH STUDY

A randomized control trial to evaluate the efficacy of autologous blood injection versus local corticosteroid injection for treatment of lateral epicondylitis.

Purpose of study:

Since you have pain at lateral epicondyle and been diagnosed as lateral epicondylitis, you are eligible to participate in the study.

The objective of the study is to evaluate the efficacy of autologous blood injection versus local corticosteroid injection for treatment of lateral epicondylitis.

Sixty patients with lateral epicondylitis are being selected for the study over a period of 1 year. If you agree to participate, you will be treated for your lateral epicondylitis with either autologous blood injection or local corticosteroid injection.

This study is done by Dr. Chetan M.D. under the guidance of Prof Dr.V.G.Murakibhavi, Dept of orthopedics, J N Medical College, Belgaum**.**

Procedure involved in the study:

Technique of Autologous blood injection: You being 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 contra lateral upper extremity and mixed with 1 milliliter of 0.5% Bupivacaine. 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-anaesthetis mixture intraarticularly. Restriction of heavy or repetitive activity for minimum of 48-72 hours after injection is adviced.

Technique of Local corticosteroid injection: 2 milliliters of Methyl prednisolone acetate 80mg mixed with 1milliliter of 0.5% Bupivacaine. This is injected in lateral epicondyle in same technique as described above.

Investigations may require whenever necessary for the study:

Anteropostrior Radiographs of elbow joints

Random blood sugar, fasting blood sugar, Thyroid stimulating hormone estimation , T3, T4, Chest x-ray, X-ray cervical spine, Magnetic resonance imaging, ultrasonography of common extensor origin, Electromyography studies.

Benefits of the study:

The benefits are early decrease of pain at lateral epicondyle, increased grip strength early return to work. There will be no extra benefits to patients otherwise. No financial incentives will be given to the participants.

Potential risk factors and discomforts:

Mild pain after injection, rarely change of pigmentation after injection.

Alternatives:

The patients have the option to decline participation in this study without any discrimination, and the patient will be treated as per the existing protocol for the condition.

All information collected during the study from patient will be told as and when necessary.

Privacy and confidentiality:

The only people who will know that you area research subject are members of the research team. No information about you or provided by you during the research will be disclosed to others without your written permission except:

I. If necessary to protect your rights or welfare.

II. If required by law.

Institutional policy:

The study does not have any damaging aspects and there no chance of injury. There is no extra cost incurred by you. But in the event of injury related to this research study, treatment will be made available at the KLE'S Dr. Prabhakar Kore Hospital & MRC, Belgaum. However, you or your third party payers will be responsible for the payment of this treatment.

Financial incentives for participants:

No financial incentives will be given to the participants.

All the investigation will be done in your interest and you will be paying for the concerned investigation**.**

There will be no reimbursed for any expenses for participation in this research.

Authorization to publish results:

J N Medical College Belgaum have the rights to publish the results of the study without your consent, however your identity is kept confidential.

Consent statement:

We ask you to read this form and ask any question you may have before agreeing to be in the research. Your participation in the research is voluntary. Your decision whether or not to participate will not affect your current or future relation with KLE'S Dr. Prabhakar Kore Hospital & MRC, Belgaum. If you decide to participate you are free to withdraw at any time without affecting that relationship.

You may withdraw at any time without consequences of any kind and you may also refuse to answer any question you don't want to answer and still remain in the study.

You can contact if you have any question about the study and about your rights as a study participant at any time to-

Dr. CHETAN. M.D. 9844592532,

Dr. V. G. MURAKIBHAVI 9845274391.

Signature or left thumb print of participants or legally authorized representative.

Participants name: signature:

Witness's name: signature:

Experimenter's name: signature:

Date:

PROFORMA

S.NO :

I.P.NO :

O.P.NO :

Name :

Age :

Sex :

Religion :

Occupation :

Income :

Address :

CHIEF COMPLAINTS :

1. Pain at lateral aspect of right/left elbow.
2. Inability to grip articles
3. Inability turn keys
4. Any other complaints
5. Duration

HISTORY OF PRESENT ILLNESS :

PAIN Site Right side/ Left side lateral epicondyle

Dominent or Nondominent side.

Onset Sudden / Gradual

Nature Continuous/ Intermittent

Radiation Present/ Absent

Relation with movements Present/ Absent

Character: -Dull aching / Localized pain

Mode of onset:

- Gradual onset.
- After prolonged repetitive of provoking movements such as grasping objects, turning screw driver, turning keys.

Severity: Ability to grip

- Not able to grip
- Able to grip with pain

Relation to movements: pain more during resisted wrist dorsiflexion and forearm supination

Any Other systemic complaints

PAST HISTORY : H/o previous episode: Y/N

Any associated diseases

Diabetes Mellitus

Respiratory Diseases

Hypothyroidism

Hyperthyroidism

Cervical spondylosis, radiculopathy.

Rheumatoid syndrome.

Trauma

PERSONAL HISTORY: Smoker / Non smoker

Alcoholic / Non alcoholic

Vegetarian / Nonvegetarian / Mixed

Nature of work-housewife, tennis player, carpenter,plumber,violonists

FAMILY HISTORY: H/O similar c/o in family

GENERAL PHYSICAL EXAMINATION :

Built - Well / Moderate / Poor.

Lymphadenopathy-Significant/Non significant

Anaemic / Non anaemic

Clubbing-Present /Absent

Weight -

Pulse –

B.P-

R.R-

Temperature - Febrile / Afebrile

LOCAL EXAMINATION :

EXAMINATION OF ELBOW;

Attitude of limb.

Overlying skin.

Deformity

Bony relationships.

Local Swelling –present / absent. (Muscle hypertrophy/ Wasting)

PALPATION:

Local Temperature Increased / Normal

Tenderness over lateral epicondyle approximately

5 milimeter distal and anterior to midpoint of the condyle:

Present/ Absent

Bony Irregularity Present / Absent

Swelling Present / Absent

Hypertrophy Yes / No

MOVEMENTS : Range of movement Active Passive

Flexion

Extension

Supination

Pronation

COZEN'S TEST: Positive/ Negative

MILL'S TEST: Positive/ Negative

INVESTIGATIONS :

X-Ray ELBOW JOINT -Antero posterior view

-Lateral view

Random blood sugar.

Fasting blood sugar.

TREATMENT :

LOCAL AUTOLOGOUS BLOOD INJECTION

LOCAL CORTICOSTEROID INJECTION IN TO LATERAL EPICONDYLE.

OUTCOME PARAMETERS AT THE TIME OF PRESENTATION :

1. PAIN SCORE; VISUAL ANALOGUE SCALE

No pain_1 _ 2 _ 3_ 4 _ 5_ 6_ 7_ 8_ 9_ 10_worst pain ever

2. NIRSCHL STAGING

No pain_1 _ 2 _ 3 _ 4 _ 5 _ 6 _ 7_worst pain

Name:

Age:

Sex:

Date of injection:

Weeks	VAS score	Nirschl staging
At time of presentation (Before injection)		
1st week		
4th week		
12th week		
6th month		

Complications:

Local skin atropy: Y / N

Post intervention exacerbation of pain: Y / N

(If yes till how many days……)

Post injection stiffness of elbow joint: Y / N

Infection: Y / N

Tendon rupture: Y / N

KEY TO MASTER CHART

D	Dominant side
F	Female
L	Left side elbow
M	Male
m	Manual
Max	Maximum
ND	Non dominant side
Nirschl	Nirschl staging
nm	Non manual
R	Right side elbow
Sl no	Serial number
VAS	Visual Analogue Scale

Corticosteroid Injection Group

Sl. No.	Age (In Years)	Sex	Side Affected	Dominance	Duration (In Weeks)	Occupation	Date of Injection	Scores										Max benefit in weeks
								Before injection		1st Week		4th Week		12th Week		6th Month		
								VAS	NIRSCHL	VAS	NIRSCHL	VAS	NIRSCHL	VAS	NIRSCHL	VAS	NIRSCHL	
1	50	M	R	D	8	m	18.10.2006	10	7	9	6	5	3	3	2	5	3	12
2	30	M	R	D	4	nm	04.01.2007	7	5	7	5	2	1	2	1	3	2	4
3	45	M	R	D	1	nm	22.02.2007	9	6	5	4	0	0	1	1	3	2	4
4	37	F	R	D	12	nm	26.03.2007	7	5	6	4	4	3	1	1	4	3	12
5	35	M	R	D	3	m	16.05.2007	8	6	4	2	0	0	2	1	3	2	4
6	48	F	L	ND	12	nm	20.05.2007	8	5	5	3	0	0	0	0	0	0	4
7	37	F	R	D	2	nm	10.06.2007	5	4	3	2	0	0	3	2	3	2	4
8	52	F	R	D	24	m	11.06.2007	6	4	3	2	0	0	1	1	1	1	4
9	37	M	R	D	2	m	24.07.2007	5	5	3	2	0	0	0	0	0	0	4
10	45	F	R	D	3	m	28.07.2007	8	6	3	2	0	0	0	0	0	0	4
11	47	F	L	ND	2	nm	11.08.2007	8	6	3	2	0	0	0	0	0	0	4
12	49	M	R	D	12	m	17.09.2007	9	6	4	3	4	3	2	1	2	1	12
13	42	F	L	D	2	nm	23.09.2007	6	4	2	1	0	0	0	0	0	0	4
14	17	F	R	D	12	nm	11.10.2007	8	6	5	4	0	0	4	3	5	4	4
15	40	F	R	D	4	nm	17.10.2007	8	6	3	2	0	0	0	0	0	0	4
16	45	M	L	ND	1	nm	17.10.2007	8	5	3	2	0	0	2	1	2	1	4
17	60	F	L	D	2	nm	22.10.2007	9	6	2	1	2	1	2	1	2	1	1
18	42	M	L	ND	12	m	12.11.2007	8	6	6	4	6	4	6	4	5	3	25
19	50	M	R	D	16	nm	12.11.2007	8	6	3	2	2	1	4	3	4	3	4
20	50	F	R	D	4	nm	14.11.2007	9	6	8	5	8	5	5	4	5	4	12
21	43	F	R	D	16	nm	19.11.2007	8	5	5	3	0	0	0	0	0	0	4
22	33	F	R	D	16	nm	20.11.2007	8	6	5	4	5	4	5	4	6	4	1
23	39	F	R	D	8	m	20.11.2007	7	3	3	2	0	0	0	0	0	0	4
24	42	F	R	D	4	nm	24.11.2007	9	6	4	3	4	3	0	0	0	0	12
25	32	M	R	D	4	nm	26.11.2007	7	4	3	2	0	0	0	0	0	0	4
26	27	F	R	D	24	nm	27.11.2007	5	4	5	4	0	0	0	0	0	0	4
27	62	M	R	D	4	nm	31.11.2007	6	4	3	2	0	0	0	0	0	0	4
28	35	F	R	D	4	nm	13.12.2007	7	5	5	4	0	0	0	0	0	0	4
29	52	M	R	D	2	nm	18.12.2007	7	4	7	4	0	0	2	1	2	1	4
30	45	F	L	D	12	m	23.12.2007	8	6	8	6	4	3	0	0	0	0	12

Autologus Blood Injection Group

Sl. No.	Age (In Years)	Sex	Side Affected	Dominance	Duration (In Weeks)	Occupation	Date of Injection	Scores										Max benefit in weeks
								Before injection		1st Week		4th Week		12th Week		6th Month		
								VAS	NIRSCHL	VAS	NIRSCHL	VAS	NIRSCHL	VAS	NIRSCHL	VAS	NIRSCHL	
1	28	F	R	D	3	nm	18.10.2006	7	5	7	5	1	1	0	0	0	0	12
2	43	F	R	D	4	nm	21.12.2006	6	4	6	4	1	1	0	0	0	0	12
3	52	F	R	D	8	nm	27.12.2006	8	6	8	6	5	4	0	0	0	0	12
4	52	M	R	D	8	m	14.01.2007	7	5	3	3	2	1	0	0	0	0	12
5	62	M	R	D	6	m	15.01.2007	7	5	7	5	6	4	0	0	0	0	12
6	33	F	R	D	2	m	18.01.2007	6	4	3	2	0	0	0	0	0	0	4
7	34	F	R	D	12	nm	25.01.2007	5	3	5	3	1	1	1	1	1	1	4
8	35	F	R	D	8	nm	17.02.2007	8	6	8	6	5	3	0	0	0	0	12
9	35	F	R	D	54	nm	12.03.2007	8	6	8	6	7	5	7	5	7	5	4
10	40	M	L	D	26	m	26.04.2007	9	7	9	7	6	4	0	0	0	0	12
11	45	F	R	D	3	nm	14.05.2007	7	5	6	4	3	2	0	0	0	0	12
12	40	M	R	D	2	m	26.05.2007	9	7	8	7	0	0	0	0	0	0	4
13	35	F	L	D	6	nm	19.06.2007	8	6	8	6	1	1	0	0	0	0	12
14	22	F	L	ND	4	nm	19.06.2007	5	3	5	3	3	2	1	1	0	0	25
15	35	F	R	D	24	nm	19.06.2007	9	6	9	6	6	4	0	0	0	0	12
16	64	M	L	ND	12	nm	25.06.2007	8	5	9	6	8	5	8	5	8	5	25
17	60	F	L	ND	16	nm	08.07.2007	6	4	6	4	1	1	0	0	0	0	12
18	40	F	R	D	4	m	15.07.2007	9	6	7	5	3	2	0	0	0	0	12
19	23	M	R	D	3	nm	24.07.2007	8	6	7	5	0	0	0	0	0	0	4
20	38	F	R	D	2	nm	06.09.2007	9	7	9	7	6	5	0	0	0	0	12
21	48	M	R	D	16	m	04.11.2007	8	6	8	6	5	3	0	0	0	0	12
22	27	M	R	D	8	m	26.10.2007	6	4	6	4	1	1	0	0	0	0	12
23	45	F	R	D	6	nm	06.11.2007	10	7	10	7	5	3	0	0	0	0	12
24	62	F	R	D	4	nm	13.11.2007	9	6	9	6	4	3	0	0	0	0	12
25	30	F	L	ND	2	nm	20.11.2007	9	6	3	2	0	0	0	0	0	0	4
26	67	M	R	D	2	m	29.11.2007	6	4	6	4	1	1	1	1	0	0	25
27	45	M	R	D	8	m	30.11.2007	9	6	9	6	0	0	0	0	0	0	4
28	37	M	R	D	12	m	23.12.2007	8	5	9	6	3	2	0	0	0	0	12
29	45	M	L	ND	16	m	23.12.2007	9	6	9	6	5	4	0	0	0	0	12
30	65	M	R	D	4	m	29.12.2007	8	6	8	6	4	3	0	0	0	0	12

TABLES ,

GRAPHS