MANAGEMENT OF CHRONIC PLANTER FASCIITIS BY PLATELET RICH PLASMA THERAPY-A PROSPECTIVE STUDY

By

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Dissertation submitted to

BLDE (Deemed to be University) Vijayapura, Karnataka

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

In

ORTHOPAEDICS

Under the guidance of

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HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

KARNATAKA

2020

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LIST OF ABBREVIATION

PF- Plantar fasciitis

PRP -Platelet rich plasma

FGP- Fibroblast growth factor

VAS-Visual analogue scale

NSAID- Nonsteroidal anti inflammatory drugs

RMS -Roles Maudsley score

ATP-Adenosine tri phosphate

ECM- Extra cellular membrane

AOFAS -American orthopaedic foot ankle society

ACD-Acid citrate dextrose

RBS – Random blood sugar

VISA-Victoria institute of sport assessment

RBC – Red blood corpuscle

ABI-Autologous blood injection

WBC- White blood corpuscle

PRTEE-Patient rated tennis elbow evaluation
HB -Haemoglobin

CAM- Controlled ankle motion walking shoes

USG- Ultrasonography

MRI- Magnetic Resonance imaging

PDGP-Platelet derived growth factor

TGP- Transforming growth factor

VEGP-Vasoactive endothelial growth factor

EGP- Epidermal growth factor

IGP- Insulin like growth factor

MCR-Micro Cellular Rubber footwear

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ABSTRACT

INTRODUCTION-

Plantar fasciitis is caused by repetitive overuse or over stretching gets inflamed.In plantar fasciitis, inflammation and degeneration occurs simultaneously. This is one of the commonest tendinopathies affecting in humans. It typically seen in both men and women of age group 40-70 years, with predominance in women.

MATERIALS AND METHOD-

The Orthopedics department of BLDE Hospital conducted this study from January 1 to May 31, 2022. With 30 patients were chosen for the study. The patients were chosen based on our criteria, and a diagnosis was only determined after a clinical examination. The VAS and AOFAS grading systems were used to evaluate the pain status. Patients were checked on at two, six, twelve, and twenty-four weeks. Pain following injection and activity level were reported. The final result was assessed in three categories—excellent, very good, and poor—based on the amount of activity and the pain status at the end of the 24-week period.

RESULTS-

The result for PRP group in mean VAS significantly decreased from 8.4 to3.3, at six months follow up, and AOFAS 27.66 to 50.93.

CONCLUSION

We came to the conclusion that intralesional PRP injection in plantar fasciitis provides pain relief and prolonged relief, at 3 months and 6 months of follow-up.

KEYWORDS: Heel spur, Visual Analogue Scale, Intralesional Injection, Platelet Rich Plasma, Corticosteroid, Plantar Fasciitis

INTRODUCTION

Plantar fasciitis is a condition where the plantar fascia becomes irritated from repetitive overuse or overstretching. Inflammation and degeneration coexist in plantar fasciitis. One of the most prevalent chronic tendinopathies affecting people in this condition. In the age range of 40 to 70 years, it commonly affects both men and women, but primarily women. 10% of the overall population has it, and 33% of occurrences are bilateral.

In the past ten years, PRP and other regenerative therapies have dramatically increased clinical use in musculoskeletal, spinal, and sports medicine. Various factors have come together throughout this time to support this development. Advances in musculoskeletal ultrasound to facilitate diagnosis and guide interventions, as well as translation of treatment paradigms from colleagues in orthopaedics and surgery, have all worked in this field for a better knowledge of tendinopathy as a degenerative cellular and connective tissue process.

Since the initial reports of PRP therapy's medical use from 1980-2000, with applications related to the fields of cardiac, dental, and maxillofacial surgery, it has grown in popularity in tissue regeneration and other specializations. PRP has been proven to be a successful autologous source for transfusion in cardiac surgery for correction of hematologic abnormalities and surgical blood loss following cardiopulmonary bypass. Anitua55 showed in the dental field that using PRP therapy at tooth extraction sites helped bone regeneration take place in sockets with compact, mature bone that had normal morphology. Marx and colleagues in maxillofacial surgery examined how PRP affected bone density and bone maturation rate in bone graft repairs of mandibular continuity deficits, showing that the addition of PRP to grafts boosted bone

development. Marx and associates in maxillofacial surgery examined how PRP affected bone density and bone maturation rate in bone graft repairs of mandibular continuity deficits, shows the addition of PRP to grafts boosted bony development.

As an intralesional biologic used to speed up the healing of cartilage, muscle, ligament, and , tendon PRP therapy has emerged as a highly sought-after treatment in today's musculoskeletal and sports medicine for its positive risk and impact on the healing of affected tissue, treating a variety of diseases, and speeding up the process of recovery.

This article offers the most recent, clinically useful information on PRP's basic science, practical considerations for using it, evidence supporting PRP and steroid use in musculoskeletal medicine, recommendations for PRP preparation and steroid, patient selection, and suggested post-procedure recovery and return-to-sport protocols. The authors will discuss the gaps in our understanding of this form of regenerative medicine and suggest important areas for further investigation.

OBJECTIVE OF THE STUDY:

1. To study the management of plantar fasciitis by platelet-rich plasma therapy
2. To find out the effectiveness of platelet-rich plasma chronic plantar fasciitis
3. To study complications of platelet-rich plasma therapy

REVIEW OF LITERATURE

1. The plantar fascia is a fibrous structure that arises from the calcaneal tuberosity and runs forward towards metatarsals, according to Enab Mohamed et al. in their research on PRP for plantar fasciitis. It serves as a supporting component for the foot's longitudinal arch (1). He added that the plantar fascia serves as a dynamic shock absorber in addition to offering static support (1).
2. The diagnosis of plantar fasciitis can be made based on the history, where symptoms were more prevalent in the morning, and the presence of discomfort when the medial calcaneal tubercle is palpated. The most common symptom is the severe pain, which is felt when taking the first few steps in the morning or when an activity begins and lessens as it warms up (2).
3. The pathogenesis of plantar fasciitis is thought to be degenerative rather than inflamed. Histological data showed that the plantar fascia was deteriorating, and cells indicative of chronic inflammation and fibroblast proliferation were also observed. The findings mentioned above was seen in surgical specimens and described in Ertgrul Aksahin et al's PRP literature (2).
4. Plantar fasciitis is a self-limiting condition that typically goes away in six to 18 months (1). Both parties will be frustrated by these symptoms' prolonged continuance. doctors who treat patients (1). In the beginning, conservative therapy is preferred (2). Changes in daily routine, orthoses, stretching, taping, the use of NSAIDs, extracorporeal shock wave therapy, targeted shock wave therapy, and other conservative treatment techniques are examples (1, 2, 4). In addition to other therapies, local injections are utilised to treat recalcitrant plantar fasciitis (2).
5. Platelet rich plasma is defined as a portion of the plasma fraction of autologous blood with a platelet value above baseline. Along with having more platelets, PRP also has all of the necessary clotting factors and secretory proteins (5).
6. Megakaryocytes produce platelets, which are then produced in bone marrow. Platelets lack a nucleus and are unable to reproduce. The five to nine-day platelet life span. Following tissue damage or surgery, platelets become trapped in damaged blood arteries, where they come into direct touch with

a variety of extracellular proteins . Blood typically has platelet counts between 150000 and 350000/L, averaging around 200000/L. (6). "M. Ferrari first advocated platelet-rich plasma as an autologous component following an open heart surgery to avoid homologous blood and blood components transfusion in 1987."

7. Platelets are necessary for tissue repair (7). The initial step in tissue healing is the creation of clots and the activation of platelets (7). The release of several growth and differentiation factors by platelets aids in tissue healing (7). These substances, which are bioactive proteins, draw macrophages, mesenchymal stem cells, and osteoblasts, promoting the clearance of necrotic tissue while simultaneously promoting tissue regeneration and healing (8).
8. Primary growth factors present in platelet alpha granules are TGEF(transforming growth factor beta), vascular endothelial growth factor (VEGF), platelet derived growth factor (PDGF), and endothelial growth factor (EGF) (9). Other growth factors include connective tissue growth factors, epidermal growth factor, and basic fibroblast growth factor (9).
9. In his article, Kenneth S. Lee mentioned the Cugat et al. research on PRP treatment for acute muscle injuries. The damaged muscle was evaluated clinically and ultrasonographically. Of his patients, 50% had successful clinical and functional outcomes (7). N According to Lindsay Harris' research on rabbit tissues, PRP injections cause tiny alterations in healthy muscle tissue. (10). 18 rabbits were used in his studies. He injected the tissues with 1/2 cc of PRP.
10. Additionally, PRP was applied to the STSG graft donor site. PRP helps to accelerate epithelialization and shorten the time between crusts (5). Crovetti et al. and McAleer et alstudies .'s on the effects of PRP on musculoskeletal injuries were mentioned by Steven Sampson in his writings (8). In the investigations by Crovetti et al. and McAleer et al., respectively, nine out of twenty-four and twenty out of twenty-four patients, respectively, had completely healed chronic ulcers.
11. For the preparation of PRP, various studies used diverse techniques. Cascade autologous platelet system, a commercially available kit, was used by Keith S. Hetchman et al. to manufacture PRP. They took 9 ml of the patient's blood and put it in a tube with 1 ml of thioxotropic separation gel and

trisodium citrate. The tube containing the blood was centrifuged at 1100g for 6 minutes (relative centrifugal force). Following first centrifugation, RBC and WBC are isolated from plasma. The plasma is put into a tube with 0.1ml of cacl2 (11).

12. For the preparation of PRP, Christos Thanasas et al made use of GPS 3 system. With 3-5 ml of anticoagulant, 27 to 55 ml of blood were drawn. They provide 3-6 ml of PRP after centrifuging the blood for 15-17 minutes at 3200-3500 rpm (12).
13. In an in vitro investigation, T M Bielecki et al. PRP was prepared using a gps 1 system after collecting 54 ml of blood in a tube with 6 ml of citrate solution. 6 ml of PRP were obtained after the whole blood was centrifuged over 12 minutes at 3200 rpm (38)
14. Three different kinds of PRP preparation techniques were used by Augustus D. Mazzocca et al. In one way, an arthrex ACP syringe is utilised, and in another, a gps 3 platelet concentrating device. Both systems only needed one spin. In the third kind, a twofold spin process was employed, with the first centrifugation occurring at 1500 rpm and the second at 6300 rpm (14).
15. 90% of the growth factors are released within 10 minutes of PRP activation. Since most growth factors have brief half lives, activating PRP will increase their effectiveness. Before injection, PRP can be activated exogenously by thrombin or cacl2, as well as endogenously by mechanical stress. A fibrin network develops after PRP is triggered, solidifying plasma and producing a fibrin clot or membrane. If PRP is overly stimulated, the fibrin network will become unstable. A more stable tetra molecular network is created by physiological activation, which improves the enmeshing of cells and growth factors (18).
16. In a 2012 study, Ertugral Aksahin et al. compared the effects of platelet-rich plasma and steroid injections on patients with plantar fasciitis (2). They chose 60 patients, and they received conservative care for three months. The injections of PRP and steroids were administered to 30 individuals each. They assessed the patients utilising "modified Roles and Maudsley scores, visual analogue scale, and visual analogue scale before injection, 3 weeks after injection, and 6 months after injection. "By the assessment of pain by visual score, roles, and mausey score, there were no

significant differences in the steroid and PRP group, and both had a significant decrease in pain at 6 months," they found " (16)

17. In a study done in 2014 by Ferhat SAY et al., the mean AFAS for the PRP group was 85.54.2 at six weeks and 90.62.6 at 6 months, while it was 75.34.8 and 80.34.7, respectively, for the steroid group (p0.001). At the sixth week and sixth month, there was a statistically significant difference in the average VAS here between PRP group and the steroid group (2.40.8 and 10.8, respectively), compared to 41.1 and 2.60.9, respectively. The PRP group experienced significantly greater changes in A0FAS and VAS ratings (p 0.001). Consequently, it was determined that PRP therapy was more successful than steroidal therapy. (17).
18. In a 2016 study by Pankaj Mahindra et al., three groups of participants were treated: one group received platelet-rich plasma therapy, the group 2 received steroid therapy, and the third group received a placebo. After a three-month follow-up, it was determined that both Prp and corticosteroids therapy are effective treatments for plantar fasciitis, with PRP therapy being the more successful option. (18)
19. A randomised control trial for the treatment of long-term plantar fasciitis was undertaken in 2019 by Sunil H. Shetty et al. They came to the conclusion that corticosteroid injection and platelet-rich plasma therapy both significantly affect and improve pain alleviation. However, compared to corticosteroid therapy, platelet-rich plasma therapy offers longer-lasting relief and requires fewer injection sessions (19).

ANATOMY OF PLANTAR FASCIA

From the medial tubercle of the calcaneum to the toes, the plantar fascia is made up of white-coloured fibres organised longitudinally.

ATTACHMENTS:

ln proximity to the calcaneum's medial tubercle.

Distal: Five slips that press against the metatarsophalangeal joints and are attached to the proximal phalanges of the corresponding toes make up the fascia's outer extension.

The principal components are: (a) Central (b) Medial (c) Lateral band Proximally compared to distally, the middle band is thicker and narrower. Its medial process is where it attaches to the calcaneal tuberosity. Distally, it separates into 5 processes, one for each toe, close to the metatarsal heads. The metatarsophalangeal joint of each toe is where every band splits into a two bands 1.superficial 2.deep strata. Transverse sulcus of the skin serves as the attachment point for the superficial stratum. Each of the two slips that make up the deep stratum encloses the sides of the flexor tendons. Four sheaths of their respective tendons and transverse metatarsal ligament are then linked to them, creating a band of arches through which the flexor tendons go to the toes.

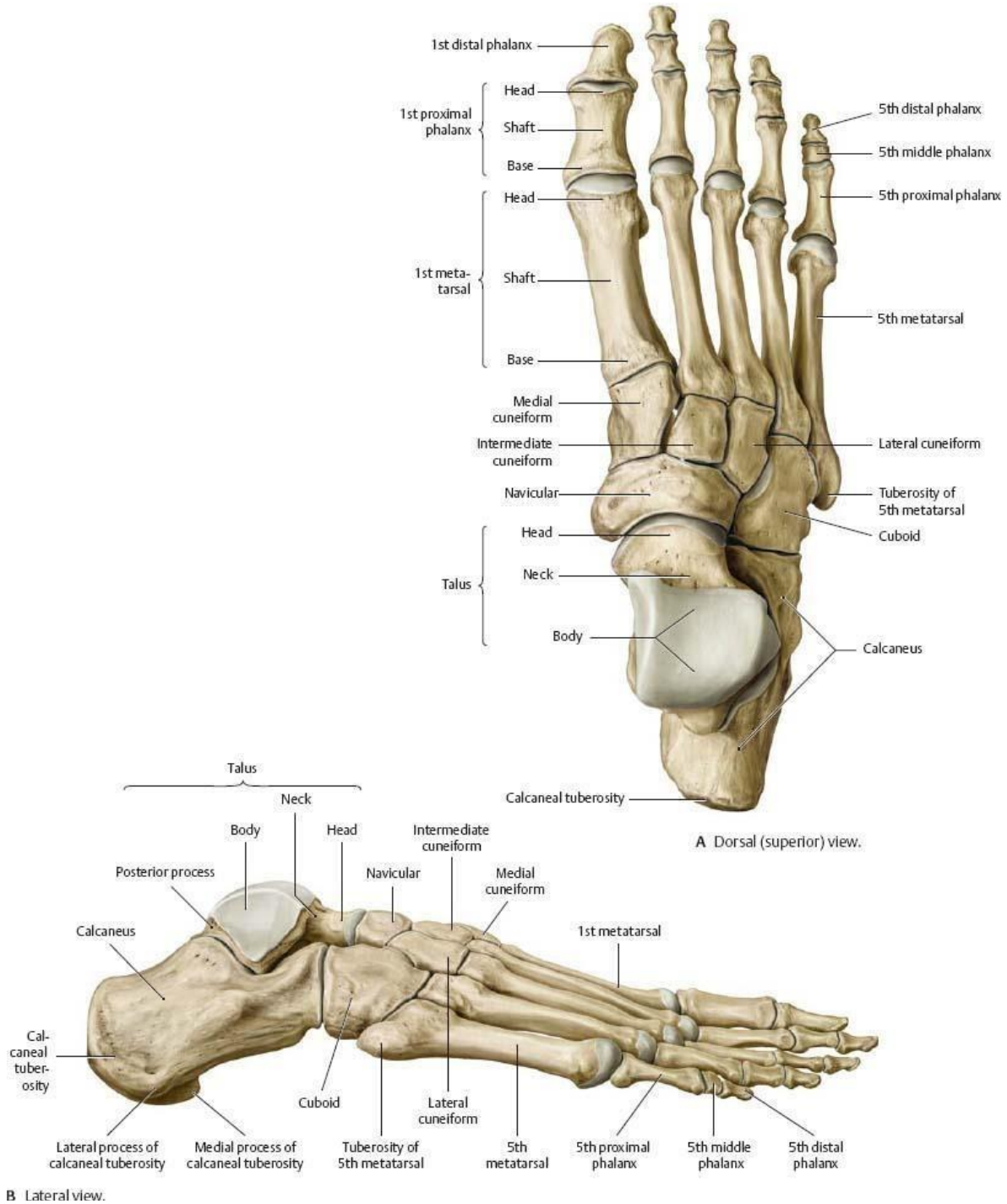
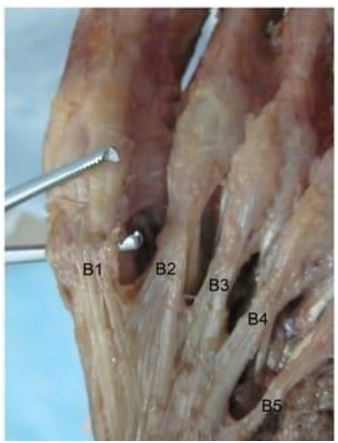
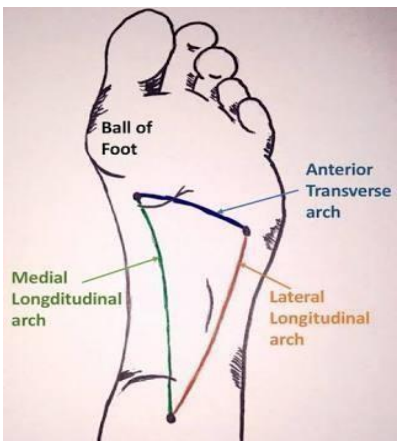


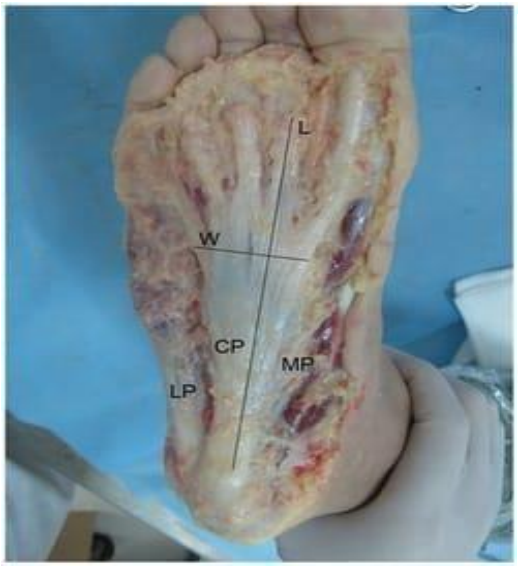
FIG 1 A&B: ANATOMY OF FOOT BONES

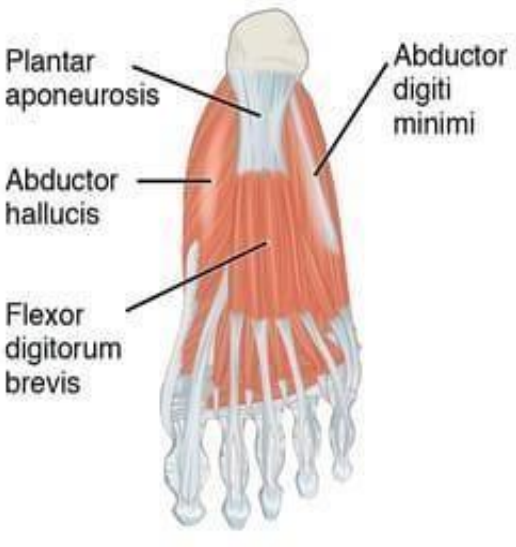
2A : SLIPS OF FASCIA



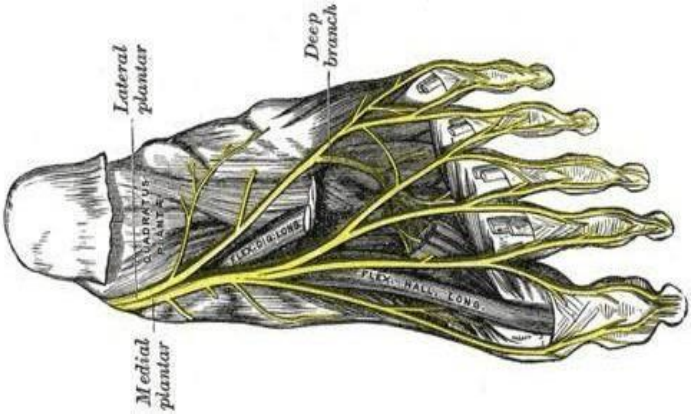
2B-SCHEMATIC REPRESENTATION



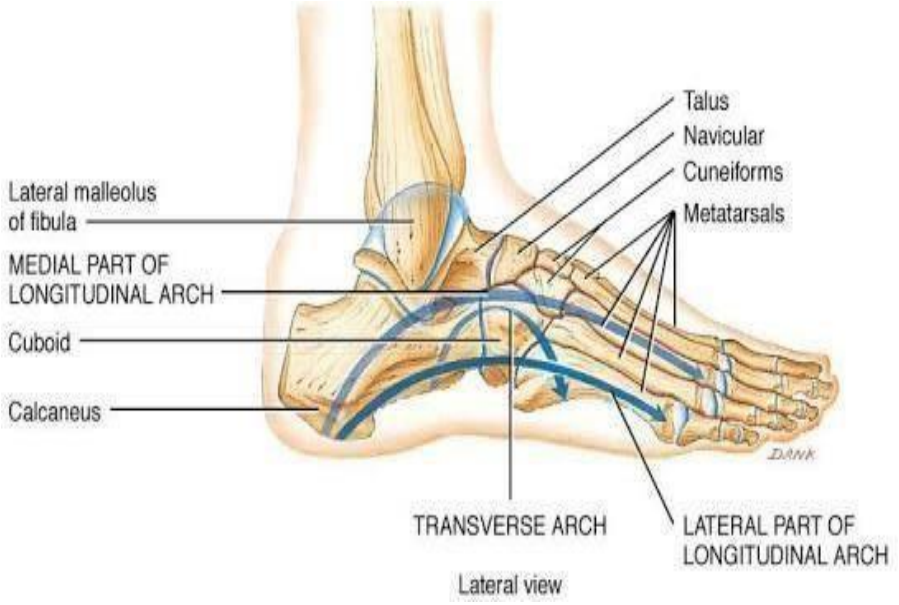




The lateral, medial, and central portions are all connected by the centre one. The point where the medial, lateral, and central bands converge is where two vertical intermuscular septae emerge. The three groups of plantar muscles are divided by these septa (central, medial and lateral). Laterally and medially, the medial section continues with the central portion; medially, it continues with the dorsal fascia. It is linked posteriorly to a laciniate ligament and is rather thin in comparison to the other two parts (flexor retinaculum). Both laterally and medially, the lateral section is connected with the dorsal fascia, and it is connected with central portion. It is thinner in the front and thicker in the back.

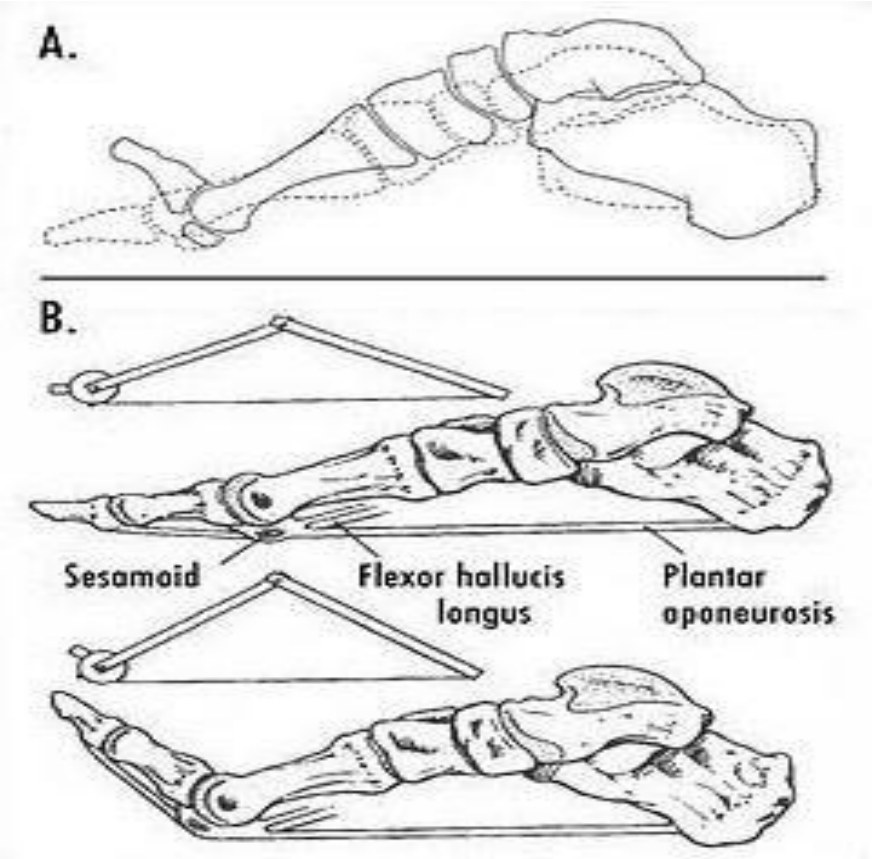


BIOMECHANICS



Plantar fascia helps to keep the foot's longitudinal arch in place. When the foot naturally bears weight, it is put under stress, maintaining the arch. The plantar fascia carries 14% of the force on the foot, per a biomechanical study model3. Surgery to remove it reduces the dynamic strain on the ankle by 10%. Plantar fascia only failed at stresses as high as 1189 newtons, below which it is sustaining the load, according to a different cadaver study. 3 The proximal attachment was the site of failure most of the time. This is consistent with the location of the calcaneum, which is the site of chronic plantar fasciitis. The longitudinal arch of the foot's rigidity was reduced after a full surgical release. The dynamic function of a typical gait is greatly influenced by the plantar fascia. Between mid-stance and the toe-off phase of gait, it lengthens to a degree of 9–12%, aiding in propulsion. The toes are dorsiflexed during the propulsive phase, which causes the fascia to tighten and elevate the longitudinal arch of the foot. This mechanism is comparable to the windlass.(20)

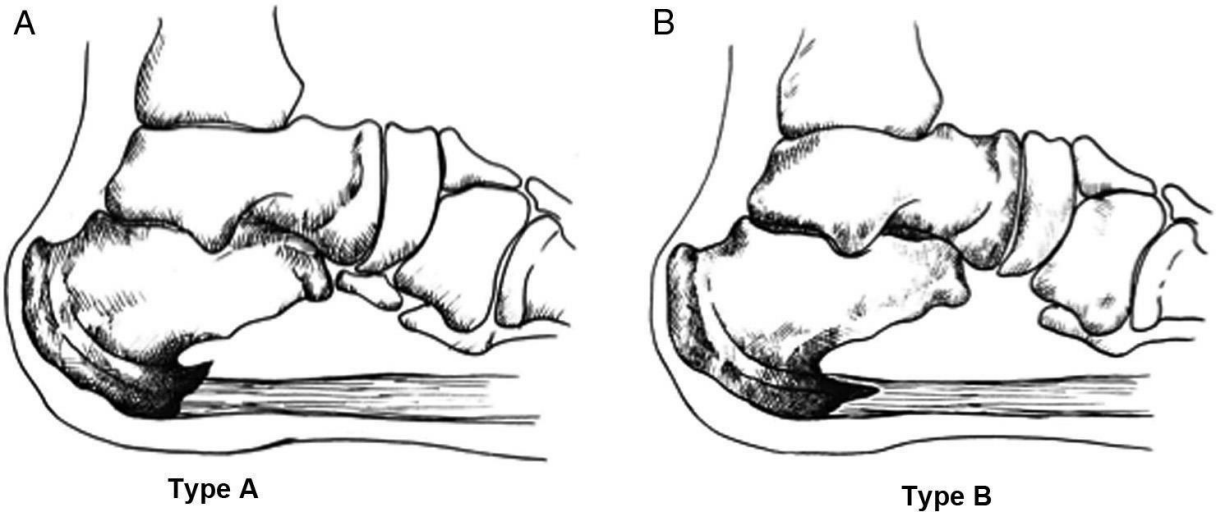
WINDLASS SYSTEM:



A windlass2, 3, is a tool for moving large weights. It was employed to raise anchors in boats and ships. The windlass mechanism is similar to the plantar fascia. During normal weight bearing, the tibia and the talus transfer the body's weight to the ground. The calcaneus and the metatarsals prefer to pass ground reaction force upward. The medial longitudinal foot arch is prone to collapse due to both of the aforementioned stresses. During dorsiflexion, the plantar fascia becomes taut and prevents the medial longitudinal arch from

collapsing, serving as a windlass or tie-rod.

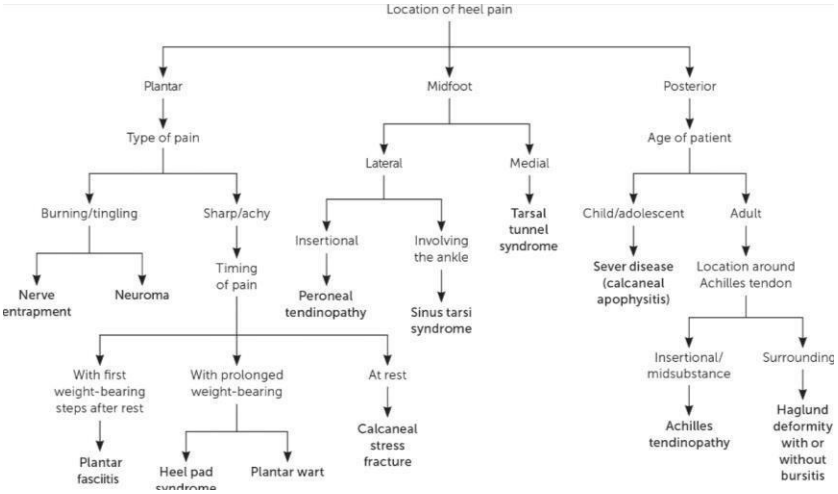
CHRONIC PLANTAR FASCIITIS PATHOLOGY:



Type A Type B

Pathological alterations similar to those seen in inflammation and degeneration are brought on by repetitive tensile loading of the plantar fascia at its connection to the calcaneum. Inflammation and degeneration are two of the events that are involved in the pathology's progression. An connection with heel cord contracture might exist. However, the actual reason of the discomfort in chronic plantar fasciitis remains unknown. Some writers contend that plantar fascia degeneration rather than the actual inflammation found in acute situations is the underlying pathophysiology in this syndrome.

Distally, the plantar fascia attaches to the base of the proximal phalanges of the toes, which fans out over a larger area. Proximally, the plantar fascia is linked to a relatively small area on the calcaneal tuberosity. This configuration pulls more strongly at the location of its proximal connection. As a result, the calcaneum is where pain is felt the most. The calcaneum's periosteum is pulled by this force. Plantar fasciitis pain is caused by the periosteum, a portion of the body that is sensitive to pain and is densely innervated by nerve fibres. A heel spur develops as a result of the plantar fascia's fibrous continuity into the calcaneum's bony matrix, which encourages the overgrowth of new bone. The pathological alterations include inflammation, collagen degeneration, a rise in ground material, and vascularity, and they are comparable to those seen in other chronic tendinopathies.(21)



CHRONIC PLANTAR FASCIITIS CAUSES:

The most frequent reason is having extremely tight calf muscles, which causes significant overpronation of the foot. This causes the plantar fascia to stretch too much and become inflamed and degenerated. Similarly, excessive supination can change the biomechanics of the foot, making it more likely for it to develop. Other factors include having feet with very high or very low arches and wearing arch-deficient footwear on a regular basis.

RISK FACTORS:

Foot anomalies like rigid feet, flat feet, high arches, and low arches.
Other risk factors include growing older, a family history of the condition, a poor walking pattern, inadequate arch support in shoes, tight calf muscles, and excessive body weight.

CLINICAL FEATURES:

Heel pain:

Tenderness over the heel and heel pain are typically at their worst first thing in the morning when the person

gets out of bed because the fascia tightens overnight and loosens up when the foot warms up. Later, the agony can last the entire day. 10

DIAGNOSIS:

The diagnosis is made only on the basis of a clinical examination11,12. Redness, discoloration, tenderness in the medial calcaneum, as well as below and under the heel, the midfoot, and the forefoot, are all symptoms of plantar fasciitis.

Investigations to determine the presence, type, and thickness of spurs as well as accompanying conditions;

1) X-RAY: A calcaneal spur and abnormal bone morphology are visible.

2)USG7: Shows thicker plantar fascia, but these findings are not diagnostic. According to ultrasound measurements, the thickness of the plantar fascia in symptomatic heels ranges from 5.7 mm to 6 mm, while it is 2.3 mm to 4.3 mm in asymptomatic heels (fig. 9a&c).

3) MRI:13 An MRI is only performed if pain continues despite all treatment plans and solely to rule out alternative causes of heel pain. Coronal and sagittal pictures are the best for displaying the plantar fascia. MR images show a consistent low signal intensity structure that is 3 to 4 mm thick and extends from the base of the proximal phalanges to the inferior calcaneal tuberosity. Plantar fascia thickening (>4 mm in the craniocaudal dimension), higher intrasubstance signal intensity on T1-weighted and T2-weighted images, and perifascial and bone

HEEL PAIN: DIFFERENTIAL DIAGNOSIS 1, 10,

1. Neurologic conditions such as entrapment syndromes Calcaneal stress fracture, second 3. Skeletal conditions such as Paget's disease. 4.Tumours. 5. Calcium-induced apophysitis. 6. Fat pad disorder. bursitis 7. Tendinitis, 8. 9.Rheumatoid arthritis, gout, and fibromyalgia. 10. Spondyloarthropathies with seronegatives. 11. Plantar warts, osteomyelitis, and diabetic ulcers 12. Lumbar radiculitis (L4-S2) 13.Trauma. 14.Rare tumours: Ewing sarcoma, neuromas, and 15.Vascular (rare) (rare).marrow oedema at the calcaneal insertional area are all MR imaging findings of plantar fasciitis. There is debate concerning the prevalence and relationship between an inferior calcaneal spur and plantar fasciitis.

TREATMENT OF CHRONIC PLANTAR FASCIITIS:

CHRONIC PLANTAR FASCIITIS IS TREATABLE BY A VARIETY OF MECHANISMS;

The first course of treatment typically involves conservative measures like rest, ice packs, NSAIDs, and footwear adjustments.

With this treatment plan, the majority of patients experience pain relief. Patients who don't respond to the aforementioned therapy procedure for their pain are given more aggressive treatments.

The various types of therapy options include;

CONSERVATIVE TREATMENT

REST & IMMOBILIZATION: For the majority of patients, rest is typically the first prescription. However, immobilisation and rest are only effective in severe conditions when inflammation is thought to be the main pathology and can only be maintained for brief periods of time. Rest has a relatively limited role in chronic instances where degeneration also exists, although it can still be explored.

NSAIDS: Non-steroidal anti-inflammatory medicines, or NSAIDs, work by preventing the production of prostaglandins by the enzyme cyclooxygenase. Prostaglandins are the main inflammatory mediators, and inflammation can be controlled by preventing their production. Therefore, it would seem logical to utilise it

in severe circumstances. Additionally, long-term analgesic use is not advised because to the risk of gastrointestinal bleeding, renal failure, and liver damage.

ICE THERAPY: Ice packing is a method of medicine that involves placing ice packs directly on the area that is inflamed. This aids in easing the discomfort brought on by inflammation.
Stretching the plantar fascia can be beneficial for chronic conditions. stretching the calf muscle and the fascia surrounding the knee, ankle, and metatarsal-phalyngeal joints.

NIGHT SPLINTS: The goal of using night splints is to keep the limb in a neutral position. Very tight calf muscles are a major risk factor for persistent plantar fasciitis. The goal of this treatment is to resolve it by splinting and keeping the limb in a neutral position, which causes the plantar fascia to expand and lengthen and may promote healing.

ORTHOTICS: Patients suffering from chronic plantar fasciitis are frequently advised to use heel pads and arch supports. Gel foam, silicone, or rubber are used to make heel pads. They lessen the strain on the plantar fascia by absorbing the impact of unequal weight bearing. For individuals with extremely high or extremely low arches and flat feet, arch supports are added to the shoes to maintain the longitudinal arch of the foot.

Ultrasonic; When a high frequency ultrasound wave is applied to a specific tissue, the tissue is heated, which breaks them down. This contributes to chronic plantar fasciitis by destroying the affected fascial tissues.
A cream containing corticosteroids and analgesics is applied to the target area before using the phonophoresis procedure, which uses ultrasonic waves to penetrate deeply into the tissues and break down heated tissue.

The procedure known as **EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY (ESWL)** is a more

recent one. It also goes by the name orthotripsy. According to the waves that are produced, orthotripsy basically comes in two flavours. Studies only indicate subpar outcomes while using it. 22,23 powerful waves:

Benefits: 1. They are more efficient than low-energy waves. 2. You might want to sit lower. Disadvantages: 1. Demand expensive equipment 2. Might not be offered in all centres 3. Extremely unpleasant when exposed 4. The patient must be sedated or under anaesthesia in order to withstand pain.

LASER THERAPY: One of the following 4 laser therapy modes, as listed below, is used to deliver laser waves to the plantar fascia: Low level laser therapy is number one. LEDs (light-emitting diodes) 2. Super luminous diodes (SLD) 4.MLS: Laser with a Class 4 multi-wave locking system. 9

The same idea underlies the operation of electrical stimulation and iontophoresis. Iontophoresis is the process of electrical stimulation followed by the application of a steroid cream to the affected area. Only a few reports are offered to support its use.

PROLOTHERAPY and **SCLEROTHERAPY** are two invasive treatments. Process of action: The inflammatory cascade is triggered by an irritating injection into the afflicted location, which enables the tissues to recover by leaving a scar. Its function in persistent plantar fasciitis is still being researched.

LOCAL INJECTIONS: To promote healing and the remission of the ailment, a number of medications or substances can be injected locally into the damaged plantar tissues. Accurate local or intralesional injections can be achieved by using ultrasound imaging to pinpoint the afflicted region. 24 Numerous studies show the relative benefits and drawbacks of various substances. 25,26,27 Following are a few of the drugs that are frequently injected:

1. Personalized blood
2. Botulinum toxin.

3. Corticosteroids

4. Plasma Rich in Platelets

1.AUTOLOGOUS BLOOD-

Small amounts of the patient's own blood are injected into the damaged tissue in this procedure. In recent years, this relatively newer method has taken the place of corticosteroid injections as a substitute. Edwards and Calandruccio performed the first successful autologous blood injection for tennis elbow in 2004. It was progressively expanded to include additional tendinopathies and persistent inflammatory diseases.

Process of action: When autologous blood is injected into an area experiencing inflammation and degeneration, it typically provides growth factors as well as cellular and humoral mediators. These growth hormones frequently draw in stem cells, which leads to collagen synthesis and tissue repair that promotes healing

Approximately 2 millilitres of the patient's venous blood are extracted, mixed with 1 millilitre of a local anaesthetic, and then injected into the most painful location.

Advantages: 1. As the patient's own blood is used, there is no possibility of an allergic reaction. 2. Budget-friendly.

Disadvantage: 1. May take a while to take effect 2. The patient might reject it.

2. INJECTION OF BOTULINUM TOXIN:

Injections of botulinum toxin have been utilised in the past to treat refractory plantar fasciitis. If the plantar fascia is painful, it can be injected there; if the calf muscles are tight, it is injected into the material found in the muscles. Studies suggest that this injection has certain advantages over a placebo.

Mechanism of action: In the fascia, it reduces pain by severing the nerve fibres that are sensitive to it. This works by promoting muscular relaxation and reducing muscle volume when injected into tight calf muscles, much like it does when used to treat cerebral palsy spasticity. Both of these sites have similar effects on central sensitization, sympathetic activity, and the buildup of pain-mediators including substance-p and glutamate.

3. INJECTION OF CORTICOSTEROIDS:

Chronic inflammatory musculoskeletal problems are treated with two different forms of corticosteroids: Methylprednisolone acetate (moderately insoluble, long-acting), and Fluorinated Hydrocortisone, such as Betamethasone, Dexamethasone, etc (highly soluble, short acting).

For local inflammation, methylprednisolone acetate is 5 to 7.5 times more potent than glucocorticoids.

Although 8–10 times more powerful than prednisolone, intralesion use of betamethasone is ineffective.

Methylprednisolone is primarily utilised for its anti-inflammatory properties, like most adrenocortical steroids. However, glucocorticoids have a variety of impacts, such as modifications to the immune system and metabolism. Like other corticosteroids like prednisolone, methylprednisolone has a lengthy list of medical disorders for which it is prescribed. Additionally, it is used to treat tendinopathies and fasciitis.

A METHOD OF ACTION

Unbound glucocorticoids breach cell membranes and bind to particular cytoplasmic receptors with high affinity, altering transcription and protein synthesis. By interfering with inflammatory mediators, suppressing humoral immune responses, and inhibiting leukocyte infiltration at the site of inflammation, glucocorticoids are able to control inflammation. The phospholipase A2 inhibitory proteins lipocortins, which regulate the manufacture of potent inflammatory mediators such prostaglandins and leukotrienes, are hypothesised to play a role in the anti-inflammatory effects of corticosteroids.

By lipocortin-1 production, corticosteroids29 produce their anti-inflammatory effects.

The major mediators of inflammation, prostaglandins and leukotreines, are not produced because lipocortin-1 inhibits phospholipase A2. Additionally, it prevents inflammatory processes such phagocytosis, chemo taxis, and white blood cell migration.

Steroids' role in persistent plantar fasciitis:

Except in severe situations, corticosteroids' anti-inflammatory effects could not be relevant in this situation. However, the curative effects of corticosteroids in this illness appear to have existed for a while. It is known that corticosteroids reduce the production of ground substances and limit fibroblast growth. 30,31 Instead of acting as an anti-inflammatory, the steroid injection's positive effects could be attributed to the aforementioned action.

Injection method:

Once the location of greatest soreness has been palpated, a steroid injection is administered. It is typically given in conjunction with a local anaesthetic to manage the pain that is felt right after the injection.

Advantages: 1. Quick action 2. The condition may be resolved with just one injection. 3. Being economical **4.** No need for costly equipment

3. **PLATELET-RICH PLASMA THERAPY**

MODE OF ACTION-

Similar to autologous blood, although in this case centrifuged platelet rich plasma is used to achieve the same results instead of giving the whole blood.

Advantages: No response to PRP 2. Budget-friendly. Disadvantages: 1. Requires a centrifugation device 2. More blood needs to be collected in larger amounts 3. You might require several injections. 4. The patient might have to limit their activity for a few weeks.

LOCAL INTRALESIONAL INJECTION COMPLICATIONS:

1. Septicemia

2. Cellulitic changes

3. Neurovascular injuy

4. Rupture of plantar fascia

5. Fat pad necrosis of heel.

6. Persistence and escalation of discomfort.

Indications:

1. For 6–9 months, plantar fasciitis did not improve with conservative treatment.

2. Reluctant patients for surgical operations.

Operative principle:

1. The puncturing of the fascia may cause local trauma and bleeding, which may result in a physiological reaction resembling that of platelet rich plasma injections.

2. Might mechanically dissolve the fascial calcifications.

Technique:

Multiple punctures can be made in a single or multiple sessions with a large needle that is introduced into the plantar fascia either blindly or with ultrasound guidance. The peppering technique refers to the combination of this with local anaesthetic infiltration.

SURGERY-

Surgery is only an option for resistant cases of persistent plantar fasciitis when all other forms of treatment have failed for at least 6 to 9 months. The success rate varies between 70 and 90%. 37,38

The several surgical options include:

1. Release of the plantar fascia (complete or partial)

2.Surgical removal of the heel spur.

Complete plantar fascial release:

It has its own drawbacks, entails releasing the entire plantar fascia, and has virtually been abandoned. If plantar fascia release is to be considered at all, the arch of the foot should not collapse and only a release of less than 40% should be performed. Minimal invasive surgery, Percutaneous partial plantar fasciotomy, Drilling and resection of total calcaneal spur can be done.

Complications:

1) The longitudinal foot arch collapses, altering biomechanics and causing mid tarsal pain. 2) Disease Calcaneal fracture 3 4) Damage to the posterior tibial nerve, etc.

The spur can be removed using an open procedure or an endoscopic procedure.

RECENT ADVANCES:

Radiofrequency micro tenotomy: This procedure involves inserting a probe into the plantar fascia and burning the damaged tissue with a high-frequency radio wave. Additionally, it negatively impacts the sensory nerve fibres that transmit pain, making the discomfort worse rather than better. The co-ablation therapy is the name of the procedure.

TREATMENT OF HEEL SPURS:

Conservative treatments for heel spurs often work well, include Analgesics, orthotic, heel stretch, etc. In cases where heel spurs are not responsive to treatment, excision of the spur either using an open procedure or an endoscopic method may be taken into consideration.

PLATELETS AND PRP BIOACTIVE COMPONENTS IN GENERAL-

In the clotting cascade, platelets play a significant role. They are megakaryocyte-derived, colourless, non-nucleated fragments of cells that are found in the bone marrow. They comprise cytokines and granules, the most crucial of which, called, contains more than 30 proteins and is essential for the healing and hemostasis

of soft tissues. Within minutes of the platelets aggregating, the granules begin to produce and secrete a number of proteins. Among the secretions produced by the granules are osteocalcin, fibrinogen, fibronectin, platelet-derived growth factor, insulin-like growth factor, vascular endothelial growth factor, epidermal growth factor, and epithelial cell growth factor. These growth factors start the growth, morphogenesis, and differentiation of their target cells.(22,23)

Inflammation, proliferation, and remodelling are the typical three stages of wound healing. The platelets become active during the time of tissue injury and the inflammatory phase. Through the granules, they start to produce their proteins, such as cytokines and growth factors. They also create bioactive substances like calcium, adenosine, histamine, serotonin, and dopamine. Serotonin and histamine will increase the capillaries' permeability before they are transferred to the wound site. Normal platelet aggregation does not occur until a stimulant is present. A biological mixture of proteins enables the platelets to start clotting and the thrombus process as soon as there is tissue damage.
Fibronectin, laminins, collagen, von Willebrand factor, among other proteins, stimulate the platelets. Platelet aggregation and activation are brought on by even the secretions produced by the platelet itself, including serotonin and adenosine diphosphate. The platelets will start to produce the fibrin clot after they have been activated.

It is challenging to determine the proper PRP concentration for clinical application. Between 150,000 and 350,000 platelets per litre of blood constitute the normal platelet concentration. To improve healing, a level of at least 1,000,000/L is required. Most PRP has a level that is 3- to 5-fold higher than the baseline. Other trials, however, have indicated efficacy at 2.0- to 8.5-fold doses. (23)

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

1. After activation, P-PRP(pure platelet rich plasma) or leucocyte-poor platelet-rich plasma (PRP) products are preparations with a low-density fibrin network but no leucocytes.

2. Leucocyte- and PRP (L-PRP) products are leucocyte- and fibrin-network-containing preparations that have been activated. The most commercial or experimental systems can be found in this family. In particular, numerous automated methods have been created in recent years, necessitating the use of particular kits that permit the least amount of handling of the blood samples and the highest level of preparation standardisation.

3. Leucocyte-poor platelet-rich fibrin preparations, also known as pure platelet-rich fibrin (P-PRF), are platelet-only and have a high-density fibrin network. These products cannot be injected or used in the same way as conventional fibrin glues; they only come in the form of a substantially activated gel.

4. Second-generation PRP products, also known as leucocyte- and platelet-rich fibrin (L-PRF) or L-PRF, are leucocyte- and fibrin-network-containing preparations. A multidisciplinary consensus meeting that was published in 2012 heavily referenced, supported, and validated this classification system.

PRP is essentially used to raise the concentration of platelets at an injured region, according to the current theory. Normally, platelets are activated during the inflammatory phase of an acute injury to start the healing process. The addition of PRP to an acute injury raises the baseline level of platelet concentration in the local tissue. Chronic injuries that have not responded to conservative treatments are likely to have reached the end of the inflammatory phase, have a deficiency in platelets, and have less capacity to recover. In these circumstances, PRP would result in two positive outcomes. PRP injections are a straightforward way to treat tendon, ligament, or muscle injuries since they merely stimulate the affected tissue and start an inflammatory reaction, which transforms the chronic damage into a brand-new acute injury.Second, adding autologous platelet concentrations should presumably speed up the healing process. Now that the origin of this new injury is recognised, it can be treated in a carefully monitored post-injection setting (Eg; immobilization, bracing, or non weight bearing). Anti-inflammatory drugs and therapies must be avoided throughout this period in order to prevent the desired effect from being reversed.

Preparation PRP(23)

Principles of PRP preparation include: Differential centrifugation is the method used to create PRP. Distinct cellular components are sedimented using different acceleration forces in differential centrifugation, depending on their varying specific gravities. PRP can be prepared in several different methods. Both the PRP approach and the buffy-coat method can be used to prepare it. In the PRP procedure, red blood cells are first separated by centrifugation, which is then followed by a second centrifugation to concentrate platelets.

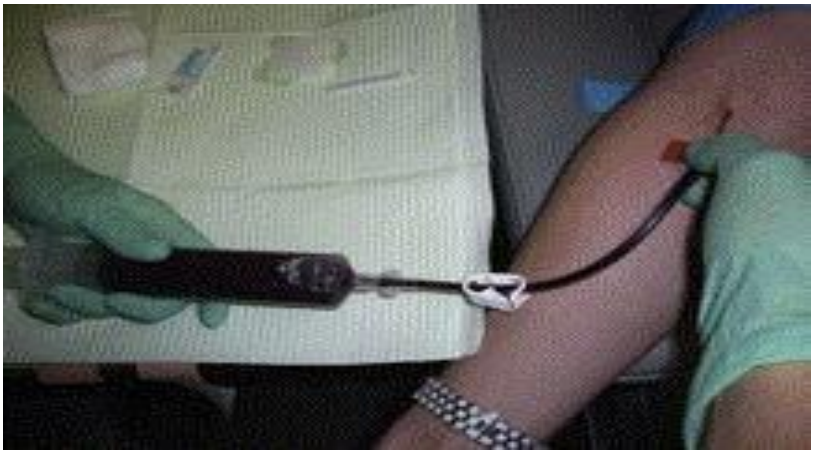
PRP is produced using a double centrifugation process in which whole blood is initially drawn into tubes containing anticoagulants. To separate RBCs from the remaining volume of whole blood, the first spin step (soft spin) is carried out at constant acceleration. The whole blood separates into three layers after the first spin step: an upper layer that is primarily made up of platelets and leucocytes, an intermediate layer known as the buffy coat that is rich in WBCs, and a bottom layer that is primarily made up of RBCs.Upper layer and superficial buffy coat are transferred to an empty, sterile tube in order to create pure PRP (P-PRP). In order to create leucocyte-rich PRP (L-PRP),

Almost no RBCs and the complete buffy coat layer are transported. The next stage is the second spin, or hard spin. The second spin of the centrifuge should be just enough to help produce the soft pellets (erythrocyte-platelet) at the tube's bottom.

The upper portion of the volume that is composed mostly of PPP (platelet-poor plasma) is removed.

The buffy coat is collected after whole blood is centrifuged at a "high speed" in the buffy coat method. A buffy coat can be created by drawing a very little amount of whole blood that has a high concentration of leucocytes. The challenge is distinguishing the RBC layer from this thin buffy coat layer, which primarily consists of white blood cells and platelets.

The cubital vein is used to collect blood for the creation of autologous PRP. The therapeutic use and desired concentration dictate how much blood is acquired. Centrifugation is then used to separate the platelets from the plasma. The PRP can be obtained using a wide variety of technologies that are already on the market.



ACQUIRING BLOOD FROM CUBITAL VEIN

It is possible to use a different technique for an automated centrifugation process that separates platelets from whole blood and then automatically transfers the product to a different syringe by using an infrared microprocessing sensor to distinguish between red blood cells and platelet-rich plasma. The precision and reproducibility of concentrations seem to increase with this kind of equipment. Because the blood product is separated automatically, there should be less chance for error. "Magellan Autologous Platelet Separator System" is an example of such a gadget. Regardless of the approach, the blood collection tube needs to have an anticoagulant. Anticoagulant tubes are frequently included in the kits that are included with the items, or they can be purchased separately**.**

The idea of activating the platelets before usage appears to be controversial in the literature. While some studies expressly identify the substance used to activate the PRP, others do not make any reference of whether or not the PRP was activated. Without discussing activation, de Vos and colleagues published a study on the effects of PRP on Achilles tendinopathy. Foster and colleagues45 propose activation with bovine thrombin in their review article.(25). Thoms and colleagues mention using calcium and thrombin together (bovine, human, or recombinant).

Using thrombin and calcium, PRP can be converted into a platelet gel, which can both disperse growth factors to promote wound healing and constrict blood vessels to lessen bleeding. Additionally, the activation will improve platelet performance. The gel's concentration of leukocytes can prevent infection and enhance tissue adhesion when used as a scaffold. It has also been demonstrated to lessen pain after surgery. The material called platelet gel is

typically applied intraoperatively to seal wounds and encourage bone repair. (27)

Cascade autologous platelet system, a commercially available kit, was used by Keith S. Hetchman et al. to manufacture PRP. 9 ml of the patient's blood were drawn and placed in a tube with 1 ml of trisodium citrate and Thioxotropic separation gel. The blood collection tube was centrifuged at 1100 rpm for 6 minutes.Following first centrifugation, RBC and WBC are isolated from plasma. The plasma is put into a tube with 0.1ml of cacl2.(28)

For the preparation of PRP, Christos Thanasas et al used a "Gravitational platelet separator system 3 (GPS)". With 3-5ml of anticoagulant, 27 to 55ml of blood were drawn. After 15 minutes of centrifuging whole blood at 3200 rpm, they eventually administer 3-6 cc of PRP. (29) In an in vitro investigation, T M Bielecki et al. created PRP using a GPS 1 system after collecting 54 ml of whole blood in a tube with 6 ml of citrate solution. Six millilitres of PRP were obtained after centrifuging the entire blood for 12 minutes at 3200 rpm.(30)

Three different kinds of PRP preparation techniques were used by Augustus D. Mazzocca et al. Autologous conditioned plasma (ACP) from Arthrex This approach uses a double syringe, while the alternative method uses a device that concentrates platelets separately by gravity. Both systems only needed one spin. The third type employed a double spin technique, with the first spin occurring at 1500 rpm and the second spin occurring at 6300 rpm. (31)

PREPARATION SIMPLIFICATION:

1. Before centrifugation, whole blood should be kept between 20 and 24 degrees Celsius.

2. Centrifuge whole blood.

3. As a result of its density, three layers are created: a bottom layer made up of RBCs, a middle layer made up of platelets and leucocytes, and a top layer made up of platelet-poor plasma.

4. Remove the tube's supernatant plasma (top layer).

5. Insert another sterile tube with the buffy-coat layer in it.

6. To separate leucocytes, centrifuge at a low speed or use a leucocyte filtering filter.

STEPS-

1. Venipuncture whole blood into acid citrate dextrose (ACD) tubes.

2. Don't cool the blood before or while the platelets are separating.

3. Spin the blood gently in a centrifuge.

4. Place the platelet-containing supernatant plasma in a different sterile tube (without anticoagulant).

5. To obtain a platelet concentrate, spin the tube in a centrifuge at a higher speed.

6. The upper 2/3rd is platelet-poor plasma, and the lower 1/3rd is PRP (PPP). Platelet pellets develop at the tube's bottom.

7. Remove the platelet-poor plasma and, using a gentle tube buffy coat shake, suspend the platelet pellets in a minimum amount of plasma (2 to 4 ml).

Therapeutic USES:

Numerous studies on animals and people have looked into the advantages and safety of PRP. The safety and effectiveness of PRP in the clinical and surgical environment have been satisfactorily demonstrated by several of these investigations. The inconsistent results, small sample sizes, and absence of controls in the human research are their main drawbacks. Due to varying PRP concentrations and post-procedure technique, it is challenging to compare research. Studies that support the benefits of PRP appear to be as numerous as those that are inconclusive. The question of whether PRP is as helpful during the acute stages of tissue healing as it might be during chronic disease must be addressed as the usage of PRP rises.(32)

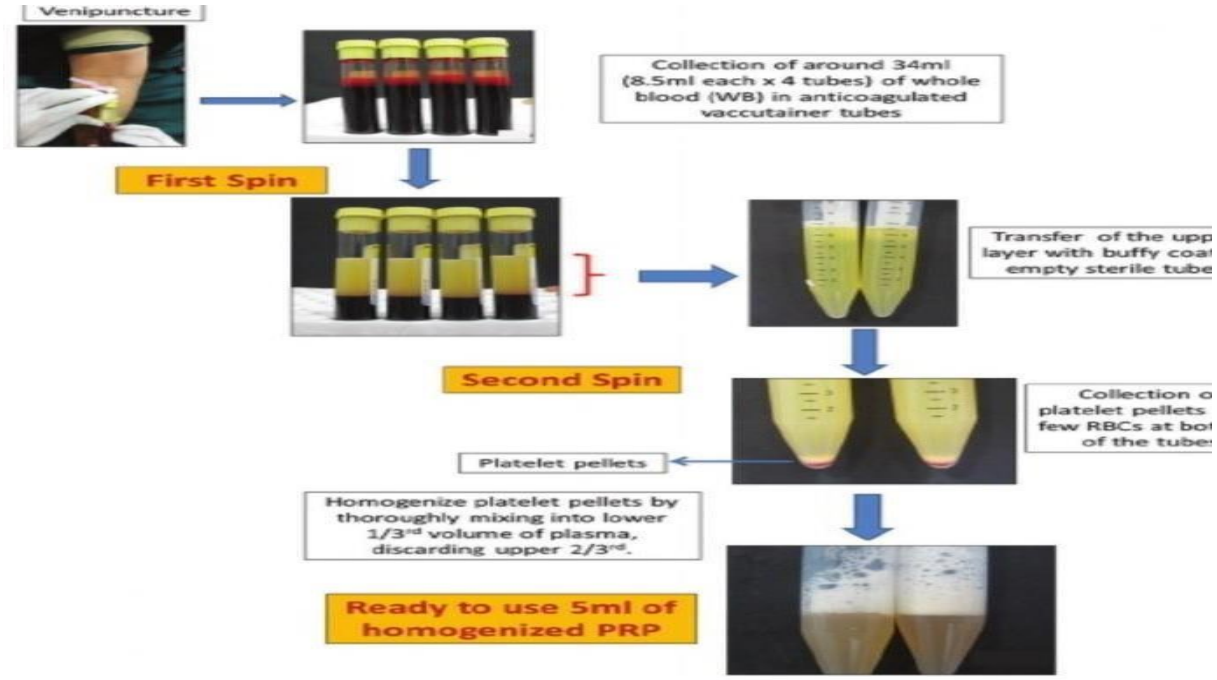


FIG 13 A,B&C : Steps of preparation (A), PRP after first centrifuge (B) and aftersecond centrifuge (C)

Early Use of Platelet-Rich Plasma:

Since the first reports of PRP therapy's clinical use in the 1980s and 1990s, with applications traceable to the fields of cardiac, dental, and maxillofacial surgery, it has grown in popularity in regenerative medicine and other disciplines. PRP has been proven to be a successful autologous source for transfusion in cardiac surgery to correct surgical blood loss and hematologic abnormalities after cardiopulmonary bypass. Anitua(33) showed that using PRP to treat tooth extraction sites enhanced bone regeneration in sockets with compact mature bone and normal morphology. Marx and colleagues in maxillofacial surgery examined how PRP affected bone density and bone maturation rate in bone graft repairs of mandibular continuity deficits, showing that the addition of PRP to grafts boosted bone development.

As an injectable biologic used to speed up the healing of tendon, ligament, muscle, and cartilage, PRP therapy has emerged as a highly sought-after treatment in today's musculoskeletal and sports medicine for its potential benefit and influence on repairing injured tissue, treating a variety of degenerative disorders, and accelerating return to sport.

Fundamentals of Wound Healing

The efficiency of PRP in promoting healing is especially important for tendons, ligaments, and cartilage since these tissues' restricted blood supply and slow cell turnover make repair processes there particularly sluggish and ineffectual. There are three stages of wound healing that can typically be distinguished: inflammation, proliferation, and remodelling. The early stages of inflammation are characterised by hemostasis, in which platelets aid establish clot formation, and the release of growth factors that help activate and attract inflammatory cells like neutrophils and macrophages to the site of injury. The proliferation phase is characterised by granulation, contraction, and epithelialization along with the formation of an extracellular matrix. Scar tissue and collagen also develop during the remodelling process.

COMPONENTS OF PLATELET-RICH PLASMA:

PLATELETS;

Platelets serve a critical role in mediating the anabolic effects of PRP by releasing growth factors from their alpha granules, in addition to being vital for hemostasis. During the initial stages of wound healing, activated platelets clump together and produce a fibrin matrix that draws and encourages cell migration into the wound. The continuous production of cytokines and platelet growth factors, which support cell adhesion, differentiation, and communication, then uses this matrix as a tissue scaffold. Despite storing both angiogenic and antiangiogenic chemicals, platelets release distinct molecules at different times. Notable growth factors secreted by platelets that are crucial in the healing process include platelet-derived growth factor (PDGF), transforming growth factor, vascular endothelial growth factor, basic fibroblast growth factor, and insulin-like growth factor. (34)

LEUKOCYTES:

Key functions mediated by leukocytes include the inflammatory response, host defence against pathogenic pathogens, and wound healing. Neutrophils are involved in the inflammatory phase of a lesion's recovery. Monocytes and macrophages initiate tissue repair by debriding and phagocytosing damaged tissue and cell debris. The growth factors secreted by macrophages, like those by platelets, are essential for tissue healing and have been associated with the regeneration of subchondral bone. Leukocytes play a crucial part in tissue repair and serve as an important line of defence against infectious agents, but their pro-inflammatory and immunologic effects can potentially unintentionally damage local cells and tissues, delaying the healing effects of PRP therapyIn vitro studies have shown that PRP with high quantities of leukocytes might produce an inflammatory milieu that inhibits the healing process. Boswell and colleagues'(36) experiments on tendon models also showed that boosting platelet concentrations was more successful at boosting PRP efficacy than lowering leukocyte concentrations, which lowered the inflammatory response. More research is needed to establish the optimal leukocyte concentration that will maximise benefit and minimise harm for each target tissue type.

BIOACTIVE COMPONENTS OF PRP[56]:

	Origin	**Function**
PDGF	Alpha granule of platelets	Cell differentiation, neovascularization
PDGF	Alpha granule of platelets	Cell differentiation, fibroblast migration, extracellular membrane synthesis
PDGF	Alpha granule of platelets	Cell proliferation and differentiation, collagen remodeling
TGF	Alpha granule of platelets	Stimulation of collagen formation
TGF	Alpha granule of platelets	Tendon differentiation
VEGF	Alpha granule of platelets	Neovascularization, prevention of apoptosis
EGF	Alpha granule of platelets	Fibroblast proliferation
Stromal-derived factor	Alpha granules of platelets	Promotes catabolism of degenerative issue; recruitment of mesenchymal stem cells and fibroblasts

Fibrin	Plasma	Component of ECM; stimulation of phagocytosis
Fibronectin	Plasma	Component of ECM; stimulation of phagocytosis
Vitronectin	Plasma	Coordination of cell migration
Interleukin	Macrophage	Increases leukocyte maturation and FGF activity
FGF	Alpha granule of platelets	Neovascularization, stimulation of ECM production and cell migration
IGF-1	Alpha granule of platelets	ECM synthesis, fibroblast proliferation

BLOOD CELLS:

PRP's red blood cell content is diminished or missing as a result of the centrifugation procedure. As part of their primary function, RBCs transport and provide nutrients, oxygen, other metabolic gases, and regulatory substances like nitric oxide to the tissues. Despite the fact that it is known to produce vasodilation, nitric oxide has been related to mediating insensitivity in diseased cartilage to the anabolic actions of insulin like gowth factor. During the oxidative process, iron included in heme molecules can generate toxic oxygen free radicals, which causes the host cells to go through apoptosis. It is hypothesised that this harmful process occurs in human synovial cells treated with RBC concentrates, drastically accelerating cartilage degradation and cell death.

Guidelines for Using Platelet-Rich Plasma:

There is still no widespread consensus on the best methods to prepare PRP or the best concentrations of blood components to add in, as each PRP formulation has unique biologic features and consequences. Results have been

erratic as a result. PRP preparation protocols have been used by researchers in a variety of ways, with variations in preparation methods, centrifugation systems, number of centrifugation steps, activation techniques with or without thrombin and calcium, and final PRP component concentrations. It is challenging to draw trustworthy inferences from the literature to guide PRP manufacture and establish indications for usage, which is what led to the creation of PRP due to the large variety of PRP formulations in use.

PRP: ACTIVATED vs. NON-ACTIVATED

Roh and associates(37) It was shown that growth factor release from PRP activated with a low dosage mixture of thrombin and calcium over a 7-day period was significantly boosted compared to non-activated PRP. It is still unclear whether quick, bolus administration of growth factors is the best method for tissue repair. Studies on PRP activation have produced conflicting results; compared to non-activated preparations, it promotes good bone regeneration but less effective fibroblast differentiation and wound healing. 90% of the growth factors are released within 10 minutes of PRP activation. Since most growth factors have brief half lives, activating PRP will increase their effectiveness. Before injection, PRP can be triggered exogenously by thrombin, endogenously by mechanical stress, and endogenously by calcium chloride. A fibrin network develops after PRP is triggered, solidifying plasma and producing a fibrin clot or membrane. If PRP is overly stimulated, the fibrin network will become unstable. A more stable tetra molecular network is created during physiological activation, which improves cell and growth factor enmeshing.

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

Autologous thrombin was employed by Stefano Gumino et al. to activate platelets. 10% calcium chloride was employed by Juan Ramon Valenti Nin et al(39) to activate platelets intraoperatively. According to research by Kenneth S. Lee et al, puncturing the skin with a needle during an injection will cause bleeding, which will provide the thrombin needed to activate platelets. (38)

DRUGS AND INTERACTIONS;

PRP is contraindicated for those who are receiving antiplatelet medication because it may inhibit platelet degranulation and the release of growth factors and bioactive compounds. This reduces the biologic tissue's capacity for regeneration. Examples of such antiplatelet drugs with various mechanisms of action include adenosine reuptake inhibitors, phosphodiesterase inhibitors, and glycoprotein inhibitors. It was demonstrated that autologous PRP significantly delayed platelet aggregation in participants receiving reversible cyclo-oxygenase inhibitors, such as nonsteroidal

anti-inflammatory medications (NSAIDs), which are frequently used to treat pain. Anti-hyperglycemic drug pioglitazone was found to increase aspirin's suppression of platelet aggregation and ATP release as well as directly prevent the release of thromboxane, which is a factor in platelet aggregation.(40)

EFFECTS OF PRP IN DIFFERENT TISSUES

MUSCLE: According to Kenneth S. Lee's paper(38), PRP research has been done on acute muscle injuries. The damaged muscle was evaluated clinically and using ultrasound technology. 50% of his patients experienced positive clinical and functional outcomes.

Following PRP injections, normal muscle tissue underwent microscopic alterations, according to Lindsay Harris in a study on rabbit tissues. rabbits were used in his research. He gave the tissues a 0.5 cc injection of PRP.

He discovered signs of irritation two weeks after the injection. In the tissue, he discovered inflammatory cells and noticed calcium deposits.

He observed the inflammation's persistence six weeks after the injection. There were cells that suggested inflammation. Muscle necrosis is present, along with fibrosis and calcification.

He noticed no signs of irritation after receiving injections for 12 weeks.

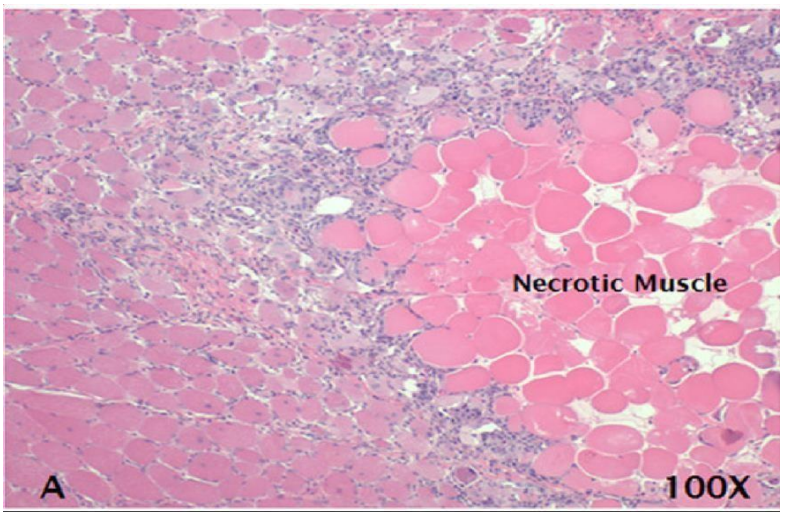


FIG 14 A

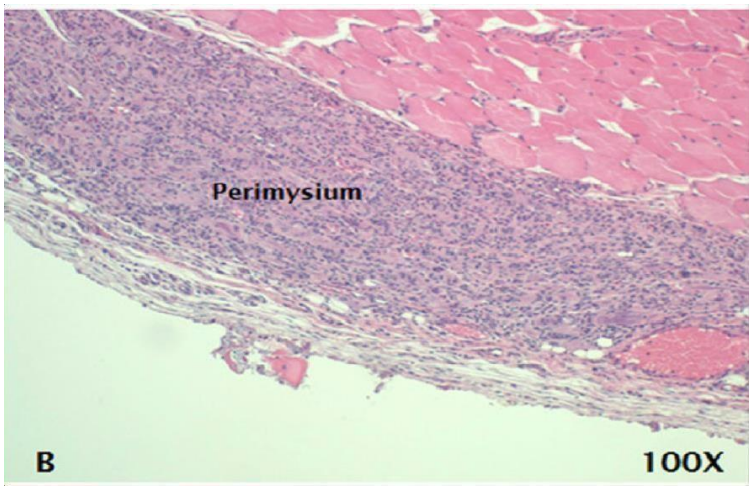


FIG 14 B

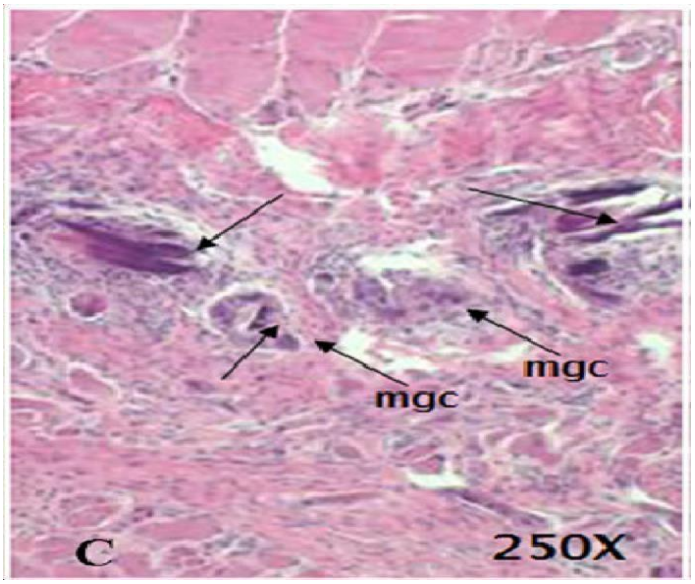


FIG 14 C

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

SUBCUTANEOUS TISSUE:

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

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

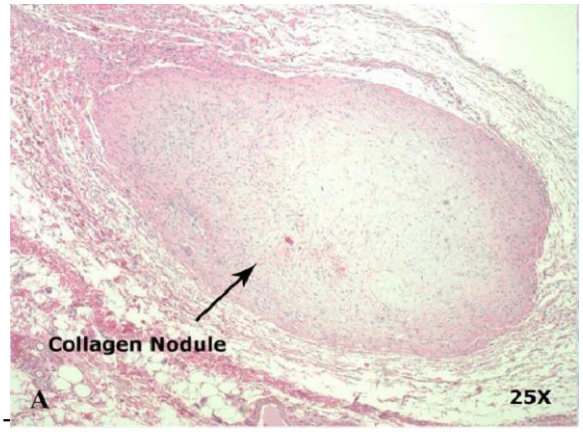


FIG 15 A

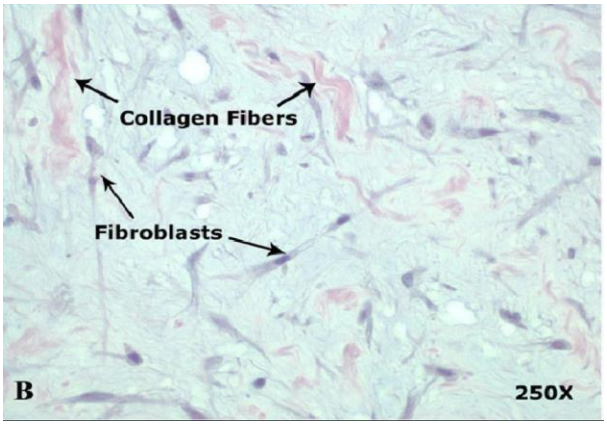


FIG 15 B

At 12 weeks, the images in 15A&15B show collagen nodules and collagen

TENDON:

In his article, Samir Mehta discussed the use of PRP for tendon injuries and tendinitis.

The use of PRP in tendinopathies was also discussed by Steven Sampson in his article.The use of PRP in tendon injuries is supported by numerous articles and research. N According to Lindsay Harris, PRP has an impact on rabbit tendon(41). At two weeks, cells of inflammation and a thick peritenon were observed. In tendon tissue, vacuoles and inflammatory cells can also be detected. Also seen are collagen bundles. Peritenon displays inflammation after six weeks. Twelve weeks later, the inflammation subsided.

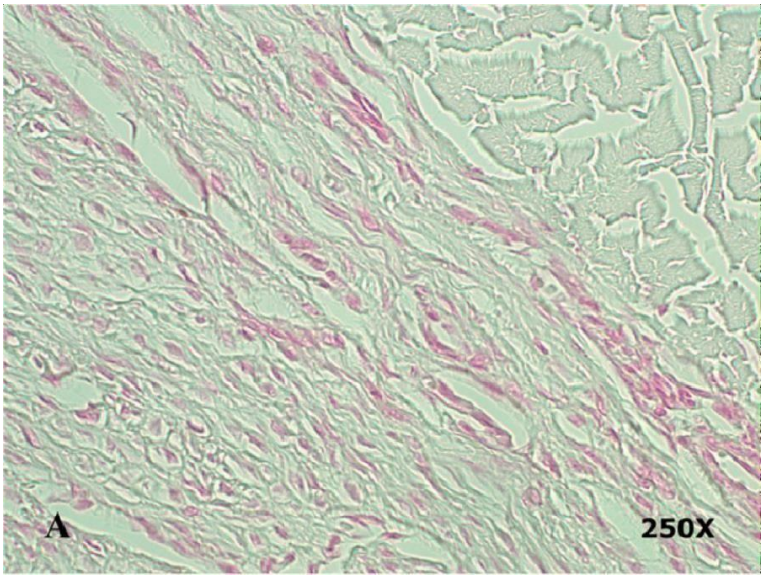


FIG16 A: shows no calcification at 2 weeks

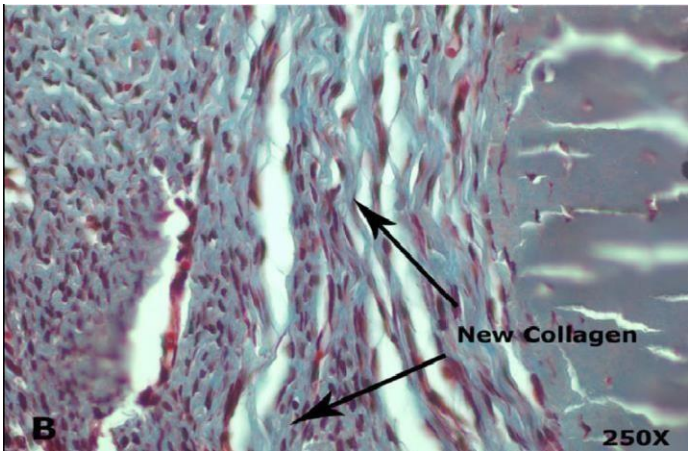


FIG16 B : shows collagen formation at 2 weeks

LIGAMENTS:

Kenneth S. Lee's evaluation of the literature included a discussion of the studies on acute ligament injuries conducted by Foster et al. and Frie et al. Patients' resumption to regular sports and activities were hastened, they found. He claimed that PRP would hasten ligament recovery and minimise instability brought on by ligament damage by citing the aforementioned studies. (42) N In a study on normal rabbit tissue, Lindsay Harris found that tissues that were thick and inflammatory at two weeks showed very minimal inflammation at six and twelve weeks after PRP injection.

WOUND CLOSURE:

Application of platelet-rich plasma helps hasten the healing of wounds. The phases of wound healing and the timing of platelet action are explained in diagram 17 below. The high concentration of platelets in PRP will cause the release of growth factors essential for wound healing. Numerous research describing the impact of PRP on wound healing have been carried out in both human trials and animal models.

In his work on platelet rich concentrate, Samir Mehta mentioned the studies carried out by D R Knighton et al and C Gaino et alstudy(43), .'s in which 78% of patients had their limbs saved and 17 out of 21 patients experienced reepithelialization

Steven Sampson pointed out that in the studies by Crovetti et al. and McAleer et al.(44), nine out of twenty-four and twenty out of twenty-four patients, respectively, had complete healing of their chronic ulcers. This was in his article on the application of PRP to musculoskeletal ailments.

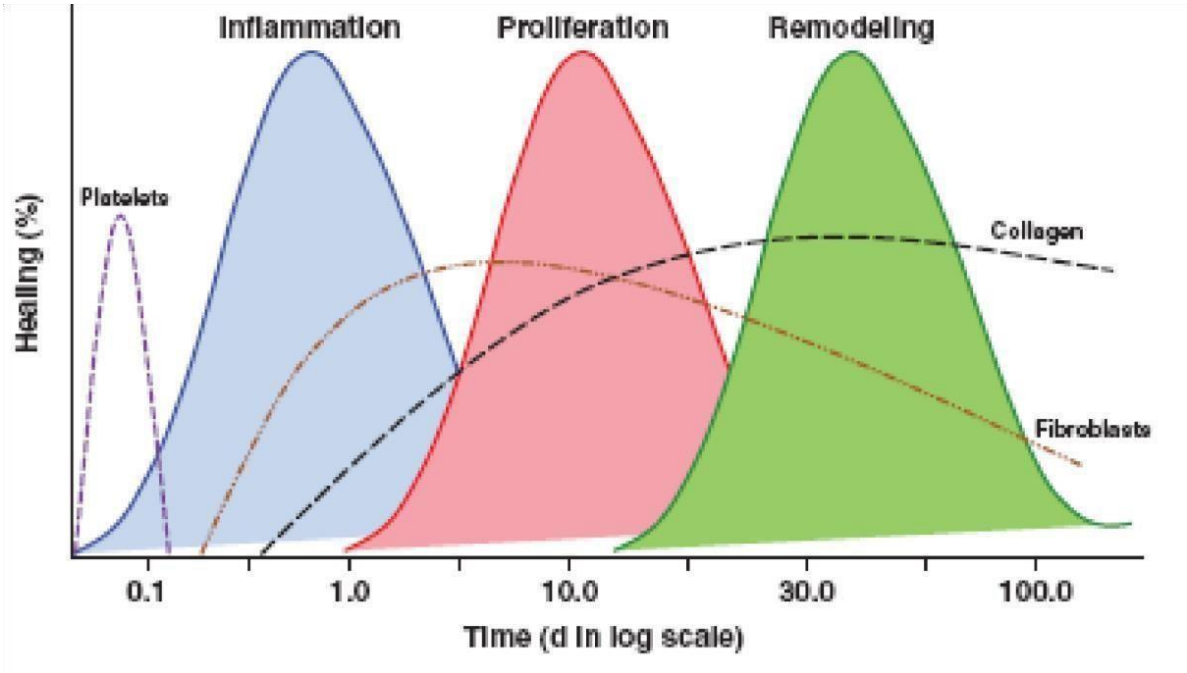


FIG 17

BONE:

There is disagreement over platelet-rich plasma's impact on bone repair. Even though some animal research did not support the usage of PRP, the majority of the investigations produced encouraging findings. When combined with bone grafts, growth factors like PDGF and TGF- enhance bone repair.(43).

J. Alsousou et al. referenced a study by Bielecki et al. in their article on the use of platelet-rich plasma in orthopedic surgery that found complete union in 13 out of 20 non-unions after PRP administration. (45) The same article reports that no growth factors were found in non-union in a study looking at the levels of growth factors in fracture hematoma. The same issue featured a study on distraction osteogenesis by Kitoh et al. It was found that callus formation takes place between 34 and 47 days.

In his study on platelet rich concentrate, Samir Mehta made an observation about the application of PRP in nonunion. (43) He claims that if there is adequate bone approach and the gap is not nonunion, PRP can help with bone healing.

In his study on platelet-richrich concentrate, Samir Mehta made a remark about the application of PRP in nonunion. (43) He claims that if there is adequate bone approach and the gap is not nonunion, PRP can help with bone healing.

JOINT:

N Lindsay Harris used a normal saline injection as a control while administering PRP to a healthy tarsal joint in

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

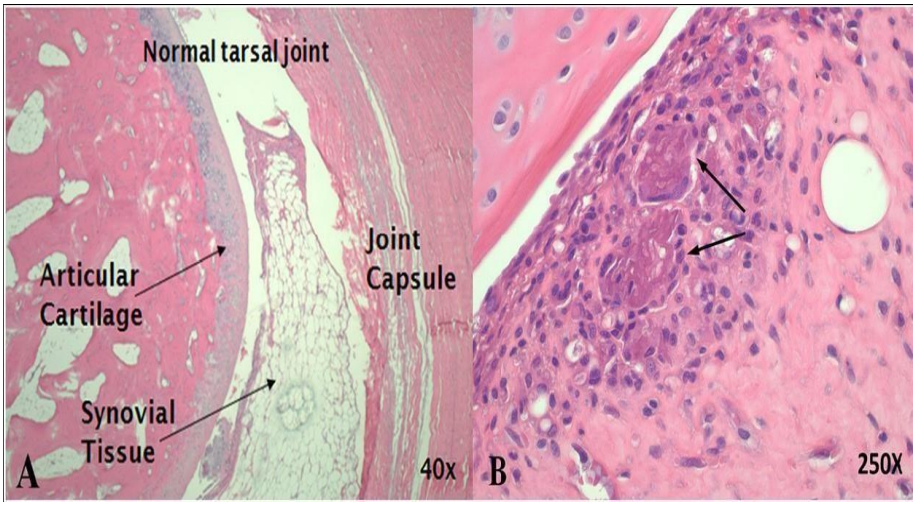


FIG 18A and 18B

SAFETY OF PLATELET-RICH PLASMA:

In his study on platelet-rich concentrate, Samir Mehta covered the safety of autologous concentrate (43). He said that there is no risk of the spread of contagious diseases because it is manufactured from the patient's own blood. He also talked about the limitations of PRP in individuals with coagulation problems and allergies to substances like bovine thrombin.

Ertugrul Aksahin found that PRP was preferred to steroid injection because it can avoid issues such fat necrosis in his study comparing the efficacy of PRP and steroid injections for treating plantar fasciitis. (46). Joost C. Peer booms et alstudy .'s on tennis elbow with PRP found no local or systemic adverse effects other than an increase in discomfort in the initial days following PRP injection because of an inflammatory process. (47) Bielecki et al. conducted a study to see how autologous PRP affected the growth of pseudomonas aeruginosa and methicillin-sensitive staph aureus (30).

MATERIALS AND METHODS

1. SOURCE OF DATA:

- Patients admitted in Department of Orthopedics in BLDE(DEEMED TO BE UNIVERSITY) Shri B.M.Patil's Medical College, Hospital and Research Centre, Vijayapura with the diagnosis of CHRONIC PLANTAR FASCIITIS.

- The patients will be informed about the study in all respects and informed written consent would be obtained.

- The period of study will be from. 1st November 2020 to 31st May 2022

  Follow up period will be 2 weeks,6 weeks, 3 months, 6 months.

2. METHOD OF COLLECTION OF DATA:

- Patients admitted to Department of Orthopedics in BLDE(DEEMED TO BE UNIVERSITY)

  Shri B.M.Patil Medical College, Hospital and Research Centre, Vijayapura with the diagnosis of plantar fasciitis

- By clinical examination.
- History taking.
- By Radiological examination – X-ray

INCLUSION CRITERIA

1. Patients diagnosed with chronic plantar fasciitis clinically,radiologically or both

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

3. Age of more than 18 years.

EXCLUSION CRITERIA

1.. Patients without any trial of conservative treatment

2. Infection or ulcer at the injection site

3. Pregnant ladies

4.uncontrolled diabetes mellitus

METHODS:

Freshly produced autologous PRP was used to treat 30 patients with severe plantar fasciitis who had failed lengthy standard non-operative treatment.

The study's informed consent was provided by every patient, and it received institutional ethical committee approval.

In order to meet the inclusion criteria, all patients underwent a plain x-ray of the ankle joint from the side as well as basic investigations such haemoglobin, random blood sugar, lipid profile, and renal profile.In order to avoid sample clotting and platelet activation before usage, a sample of 18 cc venous blood was drawn from the patient's cubital vein and mixed with 2 cc of the anticoagulant Acid citrate dextrose solution (ACD). Soft and strong spins were used in the double spin approach here. To minimise mechanical harm to the platelets, this sample was next centrifuged at rev / min for 12 minutes using a soft spin technique. Additionally, the upper layer and intermediate layer, which contain few RBCs, are moved to a sterile container before being centrifuged hard for 10 minutes at 3000 rpm. The lower third of the plasma was removed, along with platelet pellets, and transferred to an injection syringe with an 18 gauge needle. The platelet-poor plasma was discarded. About 2.5 to 3cc of PRP are available for usage. This PRP is not activated or buffered.

Nonsteroidal anti-inflammatory drugs use was not permitted during the first 2 weeks after treatment and was discouraged throughout the entire study period. No other treatment modalities were used during the study except exercises and footwear.

Physical examinations were performed, clinical symptoms and pain state were measured, and the results were compared to the pre-injection condition using interval AOFAS hindfoot score data and VAS scoring. Assessment of pre- and post-injection state. periodically at the second, fourth, twelve, and twenty-four weeks following therapy with indicated scores.

INJECTION TECHNIQUE

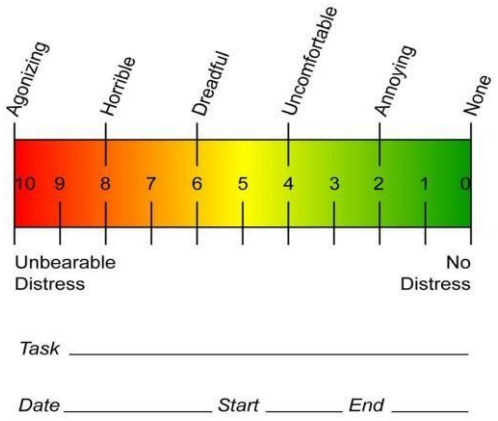
After confirming the diagnosis as plantar fasciitis(clinically or radiologically or both), the patient is made to lie in a lateral position on the table with knee flexed up to 90 degrees. The affected foot is scrubbed with betadine scrub(7.5% of povidone-iodine). Then cleaned with spirit and painted with betadine solution(10% povidone-iodine). The most tender point of the foot is palpated and marked. Then with 18G intramuscular needle the 3ml of PRP is injected with the peppering technique (fanning of PRP in all directions once u breach the subcutaneous layer).

MAIN OUTCOME MEASUREMENTS:

The outcomes of the study was calculated by Visual analogue scale scoring and AOFAS scoring . the values are added at pre-injection, 2^{th}, 4^{th}, 12^{th} and 24^{th} weeks post injection. Final results were measured based on the pain and activity level at 6 Months of follow-up.

VISUAL ANALOGUE SCALE

Numerical pain score is a subjective assessment of pain where the patient rates the intensity of the pain perceived. Score 0 refers to no pain. Score 10 refers to the worst pain possible.



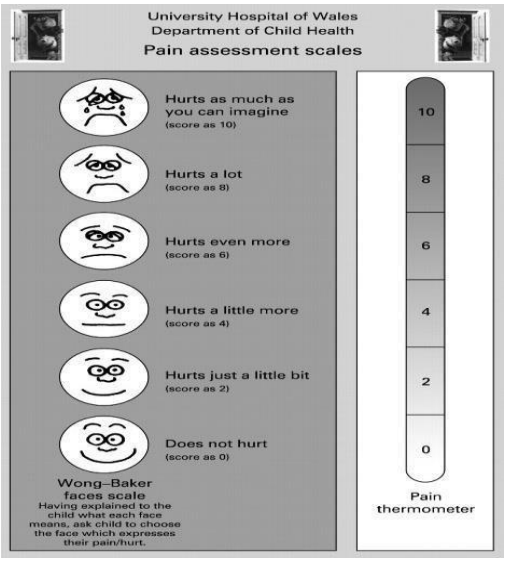


FIG 19A&B

American Orthopedic foot and ankle society ankle hindfoot score(AOFAS)

AOFAS rating system published in 1994, to rate the clinical status of ankle-hind foot, ranging from 0 to 100, with healthy ankle receiving 100.

1. Pain 40 points

None	40
Mild occasional	30
Moderate	20
Severe	0

2. Function(50 points)

Activity limitations, support requirement	
No limitations, no support	10
No limitation of daily activities, limitation of recreational activities, no support	7
Limited daily and recreational activities, cane	4
Severe limitation of daily and recreational activities, walker, crutches, wheel chair, brace	0
Maximum walking distance, block	
Greater than 6	5
4-6	4
1-3	2
<1	0

Walking surface	
No difficulty on any surface	5
Some difficulty on uneven terrain ,stairs ,inclines, ladders	3
Severe difficulty on uneven terrain ,stairs ,inclines, ladders	0

Gait abnormality	
None, slight	8
Obvious	4
Marked	0

Sagittal motion	
Normal or mild restriction (30 degrees or more)	8
Moderate restriction (15-29 degrees)	4
Severe restriction (<15 degrees)	0

Hind foot motion	
Normal to mild restriction (75-100 %normal)	6
Moderate restriction (25-74% normal)	3
Marked restriction (<25% normal)	0

Ankle-hind foot stability	
Stable	8
Definitely unstable	0

3. Alignment (10 points)

Good, plantigrade foot, midfoot well aligned	15
Fair, plantigrade foot, some degree of midfoot malalignment, no symptoms	8
Poor, non plantigrade foot, severe malalignment, symptoms	0

SAMPLING:

1 With anticipated Proportion of moderate limitation of activity (post operative)among plantar fasciitis patients 0.08% (ref) , the study would require a sample size of 30 patients with 95% level of confidence and 10% absolute precision.

Formula used

$$n=\frac{z2\ p*q}{d2}$$

Where Z= Z statistic at α level of significance

d2= Absolute error

P= Proportion rate

q= 100-p

Statistical analysis:

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

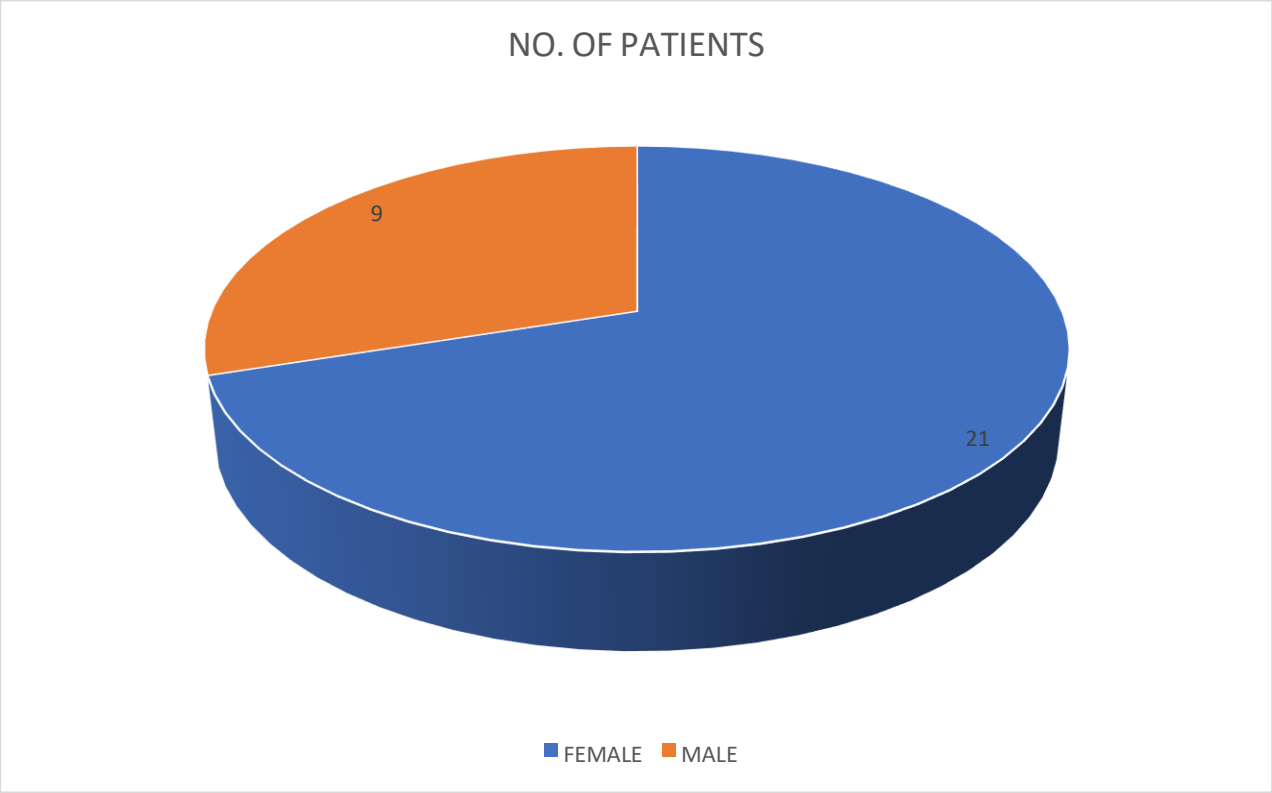
 Results will be presented as Mean (Median) ±SD, counts and percentages and diagrams.

RESULTS

In BLDE Hospital and research center, between 1st November to 31st May 2022, all 30 patients with plantar fasciitis were followed by dept of orthopedics.

SEX DISTRIBUTION-

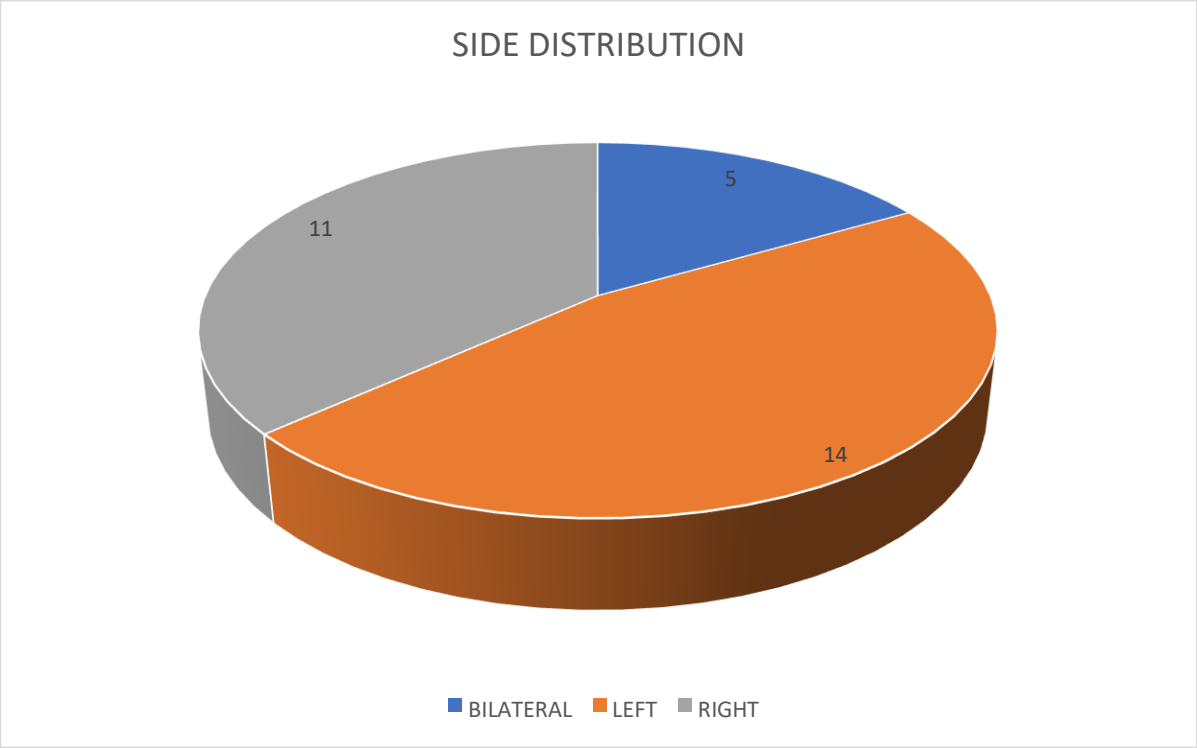
SEX	NO. OF PATIENTS	PERCENTAGE
FEMALE	21	70
MALE	9	30
TOTAL	30	100



In our study the total number of patients are 30, of which 21(70%), are female patients and 9(30%) patients are male. This study shows female predominance in plantar fasciitis.

SIDE DISTRIBUTION

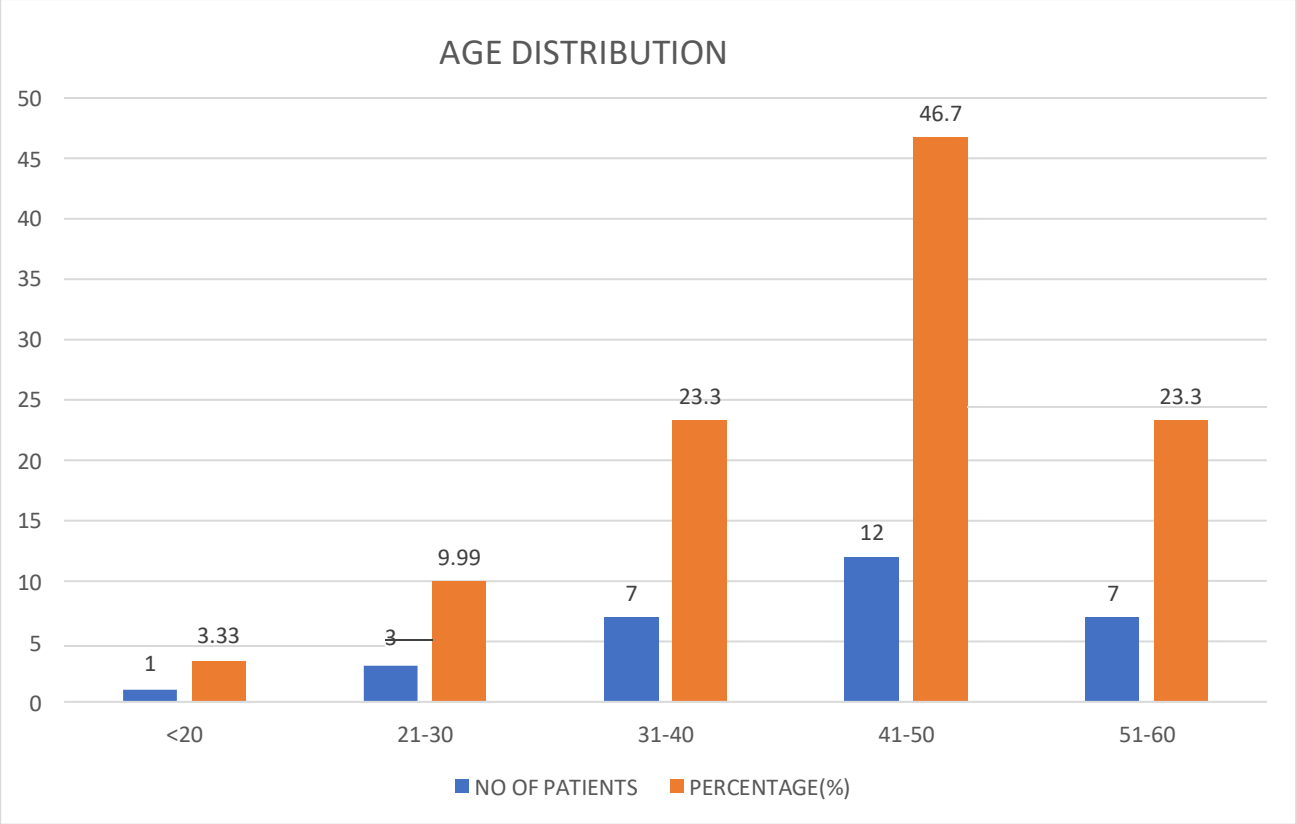
SIDE	NO. OF PATIENTS	PERCENTAGE
BILATERAL	5	16.7
LEFT	14	46.7
RIGHT	11	36.7
TOTAL	30	100



In the study conducted by dept of orthopaedics in BLDE hospital, 30 patients were treated with pl;atelet rich plasma therapy. Out of 30 patients, 14 showed symptoms on left foot and 11 showed symptoms on right foot and 5 showed symptoms on bilateral foot suggests that plantar fasciitis is seen commonly on left foot.

AGE DISTRIBUTION

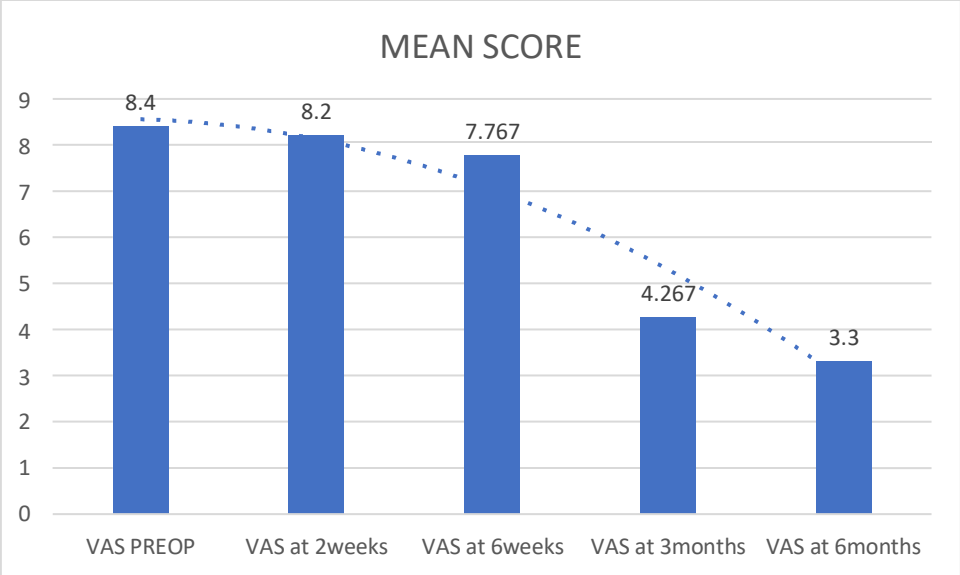
AGE(YEARS)	NO OF PATIENTS	PERCENTAGE(%)
<20	1	3.33
21-30	3	9.99
31-40	7	23.3
41-50	12	46.7
51-60	7	23.3
TOTAL	30	100



In this study which includes 30 patient, the mean age of patients presenting with plantar fasciitis is 41.30 years. The mean age of females patient included in our study is 43.3 years where mean age of male patients is 37.5. Majority of patients falling in the range of 40-50 years of age..

VISUAL ANALOGUE SCORING-

VAS SCORING	MEAN SCORE
VAS PREOP	8.4
VAS at 2weeks	8.2
VAS at 6weeks	7.767
VAS at 3months	4.267
VAS at 6months	3.3



In the study conducted by department of orthopedics, 30 patients selected for the study were analyzed using visual analogue scoring system at 2 weeks, 6 weeks , 3months and 6 months. The results show that there is no much difference in the relieve of pain at 2 week of follow-up(mean VAS score-8.2), whereas at 6 weeks of follow-up there is slight improvement(mean VAS score 7.7)

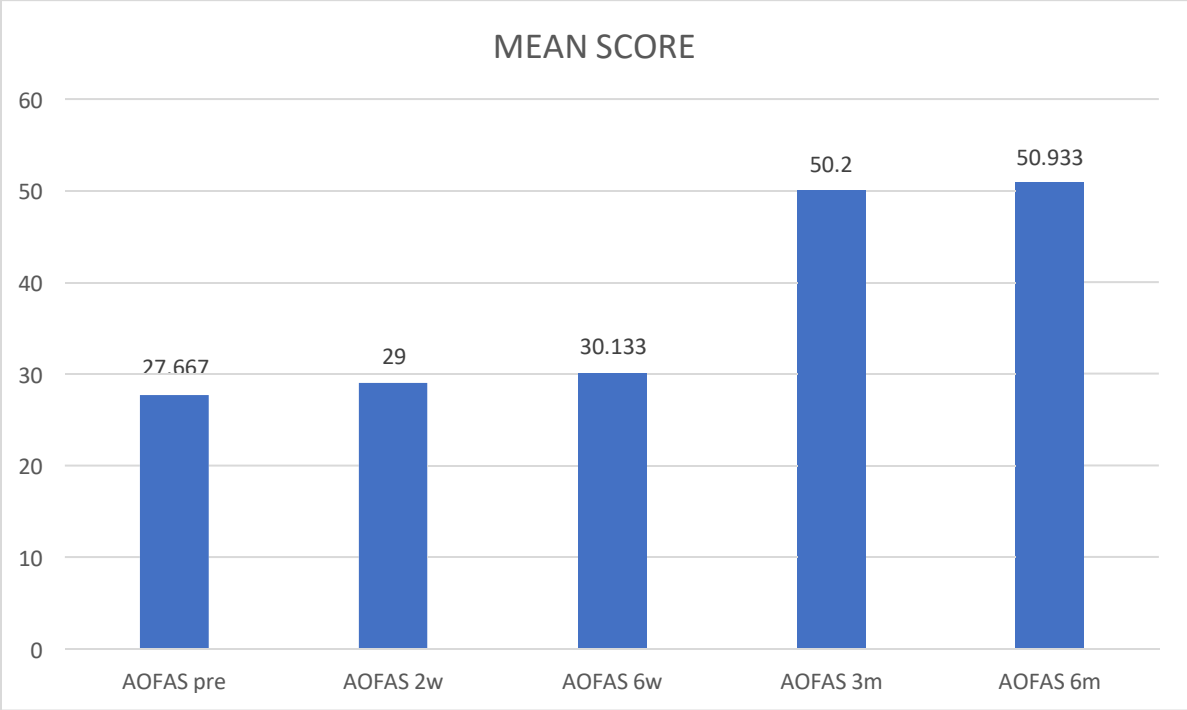
The patients when examined at 3month follow-up and 6 month follow-up there is significant relieve in pain with mean being 4.26 and 3.3 respectively.

This suggests that there is significant pain relief after 6 weeks of platelet rich plasma therapy, with more significant relief shown after at 3 months and 6 months follow up.

This signifies that the platelet rich plasma therapy shows relief in pain after 6 weeks of platelet rich plasma therapy and further there is significant reduction in pain relief at 3 months of follow-up and 6 months of follow-up.

AMERICAN ORTHOPAEDIC FOOT AND ANKLE SCORE

MEAN AOFAS SCORE	MEAN SCORE
AOFAS pre	27.667
AOFAS 2w	29
AOFAS 6w-	30.133
AOFAS 3m	50.2
AOFAS 6m	50.933



In the study conducted by department of orthopedics, 30 patients selected for the study were analyzed using AOFAS SCORING along with visual analogue scoring system at 2 weeks, 6 weeks , 3months and 6 months.

The results show that there is no much difference in the functional outcome at 2 week of

follow-up(mean AOFAS score mean-29), whereas at 6 weeks of follow-up there is slight improvement(mean AOFAS score mean- 30.13)

The patients when examined at 3month follow-up and 6 month follow-up there is significant increase in the function with mean being 50.3 and 50.9 respectively.

This suggests that there is slight increase in function after 6 weeks of platelet rich plasma therapy, with more significant function and moveent shown after at 3 months and 6 months follow up.

DISCUSSION-

In its most basic definition, plantar fasciitis refers to inflammation of the plantar fascia at the point where it attaches to the calcaneum. Recent research, however, suggests that it is more likely plantar fascia deterioration than actual inflammation. In 2004, Dr. Barrett proposed that the condition is actually a degenerative changes of the plantar fascia and was commonly known as plantar fasciosis. It was also confirmed by pathologist results that samples from chronic plantar fasciitis sufferers contained very few inflammatory cells.(48). Inflammation and degeneration are two of the events that are involved in the pathology's progression.

The initial course of treatment consists of a combination of conservative techniques, such as rest, the use of cold packs, non-steroidal anti-inflammatory medicines, and footwear changes, such as arch supports. Before the condition may be resolved, it may typically take several repeated session of the techniques like ultrasonic waves, electrical stimulation, and phonophoresis. Local intra-lesional injection or invasive plantar fascial release may be possibilities when the condition is unresponsive to the aforementioned conservative therapy approaches. Corticosteroid, Botulinum toxin, autologous blood, and platelet-rich plasma injections can be tested locally and intralesional. Numerous research highlight the benefits and drawbacks of various treatment options.

Because PRP formulations were so effective at treating chronic tendinopathies, they were also used to treat severe cases of plantar fasciitis.

CONCLUSION-

The results of this study indicate that short-term improvements in VAS scores for heel pain, functional outcome scores, and restoration of plantar fascia thickness in patients with chronic plantar fasciitis showed medically and statistically significant improvements. According to the study, local PRP injection is an effective and secure treatment for chronic plantar fasciitis.

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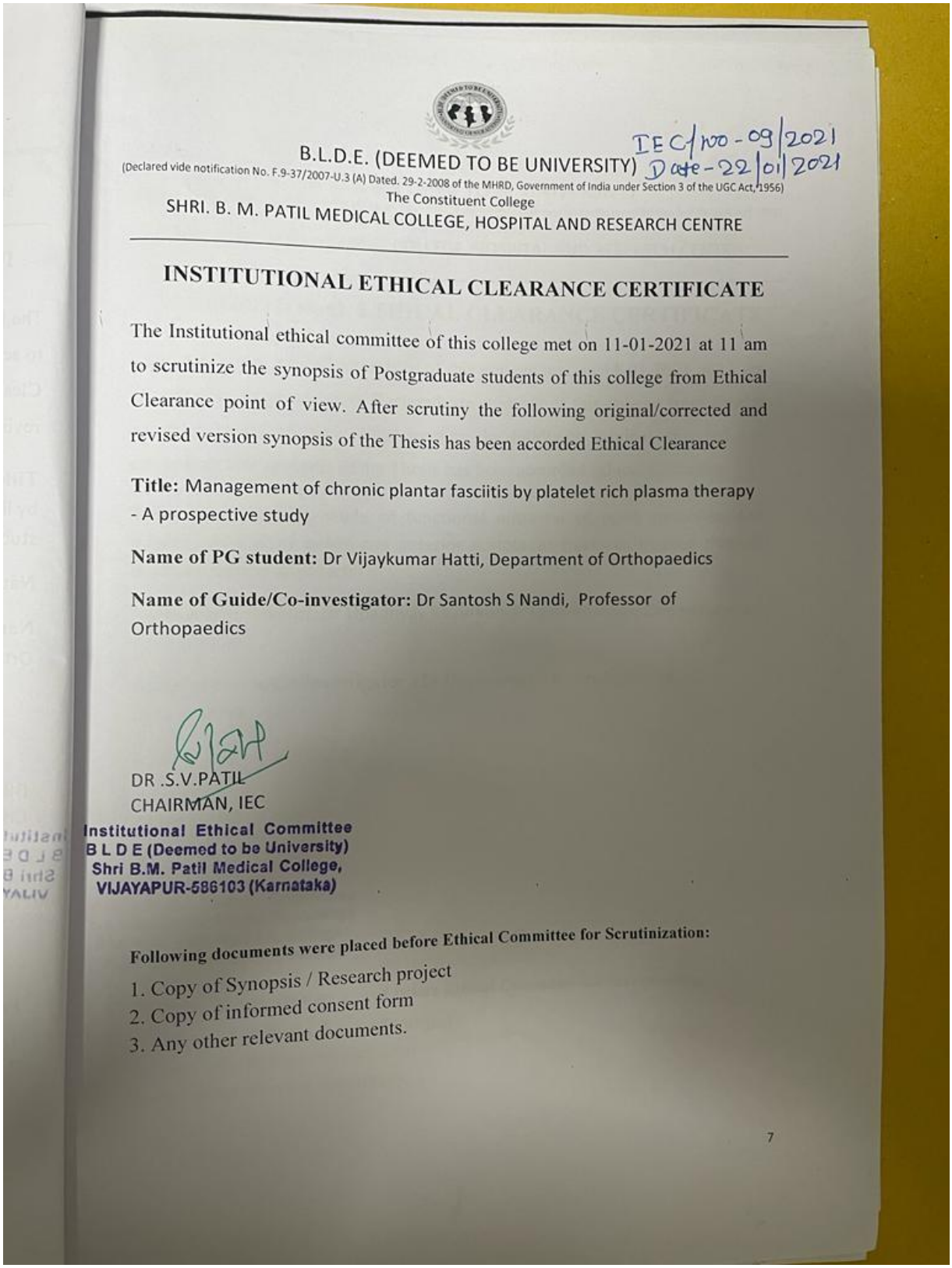
ANNEXURE I

Ethical committee certificate



B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/100-09/2021 Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act,1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Management of chronic plantar fasciitis by platelet rich plasma therapy - A prospective study

Name of PG student: Dr Vijaykumar Hatti, Department of Orthopaedics

Name of Guide/Co-investigator: Dr Santosh S Nandi, Professor of Orthopaedics

DR .S.V.PATIL
CHAIRMAN, IEC

Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

7

ANNEXURE II

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

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned,_____, S/O D/O W/O_____, aged_____years, ordinarily resident of_____do hereby state/declare that Dr. vijaykumar hatti of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on_____at_____(place) and it has been explained to me in my own language that I am suffering from_____disease (condition) and this disease/condition mimic following diseases. Further Dr. vijaykumar hatti informed me that he/she is conducting dissertation/research titled **"MANAGEMENT OF CHRONIC PLANTAR FASCIITIS BY PLATELET RICH PLASMA THERAPY- A PROSPECTIVE STUDY."** under the guidance of Dr. S.S. Nandi requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative, post-operative and follow-up observations will be utilized for the study as reference data.

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

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

The Doctor did inform me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during the course of treatment/study related to diagnosis, the procedure of treatment,

result of treatment, or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and followup unless I request to be discharged.

After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Shri/Smt_____under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of the patient:

Signature of doctor:

Witness: 1.

2.

Date:

Place :

ANNEXURE – III

SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA–586103

PERFORMA

CASE NO. :

FOLLOWUP NO. :

NAME :

AGE/SEX :

I P NO :

DATE OF ADMISSION :

DATE OF SURGERY :

DATE OF DISCHARGE :

OCCUPATION :

RESIDENCE :

Presenting complaints:

History of pain:

a. Duration of pain

b. Nature of pain

c. History of past medical disease

- Diabetes mellitus
- Hypertension /IHD
- COPD
- Pulmonary tuberculosis
- Epilepsy

g. Personal and family history.

MASTERCHART.

Sl. No.	Patient name	Age(YRS)	Sex	OPD no.	Side Effected	Pre treatment scores		Scores at 2 weeks		Scores at 6 weeks		Scores at 3 months		Scores at 6 months	
					Right/Left	VAS	AOFAS	VAS	AOFAS	VAS	AOFAS	VAS	AOFAS	VAS	AOFAS
1	APPASAHEB	42	M	14587	LEFT	6	44	5	50	4	48	3	50	2	48
2	PREMRAM	37	M	16852	LEFT	9	28	8	34	8	32	5	44	4	46
3	LOKNATH	42	M	19022	RIGHT	7	34	7	36	6	38	5	40	4	38
4	SAMALAL	32	M	18966	LEFT	9	26	8	30	8	30	5	44	4	42
5	BASAVARAJ	38	M	20279	BILATERAL	7	38	7	36	6	38	5	42	4	40
6	LOKESH	20	M	20988	LEFT	10	12	10	16	9	18	5	40	4	42
7	SHIVAMMA	42	F	22940	LEFT	8	28	8	30	8	30	4	56	3	58
8	KISHORI	51	F	26692	RIGHT	9	20	9	22	9	22	4	56	3	58
9	SUREKHA	24	F	26040	BILATERAL	8	28	7	36	6	40	5	42	4	44
10	PADMAVATHI	57	F	3739	LEFT	9	26	9	28	9	28	5	44	4	42
11	UMABAI	53	F	43533	BILATERAL	7	36	7	40	6	42	5	44	4	42
12	PRIYA	21	F	47985	RIGHT	8	30	8	32	7	34	3	60	2	64
13	KULUSUMA	43	F	83933	BILATERAL	9	20	9	24	9	24	5	42	4	44
14	VAISHNAVI	23	F	99591	BILATERAL	10	12	10	14	9	18	4	56	3	58
15	BHIMANNA	33	M	139513	RIGHT	8	30	8	32	8	34	4	58	3	60
16	ARAVIND	51	M	139591	LEFT	9	22	9	24	8	28	3	60	2	58
17	MAHADEVI	51	F	130289	LEFT	10	12	10	14	10	16	4	56	3	54
18	SUDHA	36	F	1302284	RIGHT	7	38	7	40	7	40	3	60	2	58
19	KASTURI	51	F	88264	LEFT	10	60	9	16	8	20	3	60	2	60
20	KAVERI	50	F	6373	RIGHT	10	12	10	14	10	14	5	42	5	40
21	GIRIJA	39	F	19171	LEFT	9	20	9	24	9	24	4	56	3	58
22	CHINAMMA	51	F	51700	RIGHT	10	16	10	18	10	18	5	42	4	46
23	LEELAVATHI	49	F	52013	RIGHT	8	26	8	28	8	28	4	56	3	58
24	BORAVVA	42	F	57837	LEFT	7	36	7	38	6	42	5	40	4	44
25	KALAVATHI	43	F	59887	LEFT	8	28	8	30	8	30	4	58	3	60
26	RADHA	51	F	72318	LEFT	7	34	7	36	7	36	3	60	2	58
27	SAVITRI	46	F	74230	RIGHT	10	12	10	14	10	14	5	44	4	46
28	KALAVATHI	48	F	78287	RIGHT	7	36	6	44	5	46	4	56	3	58
29	RADHABHAI	37	F	77293	RIGHT	8	34	8	36	8	36	5	44	4	48
30	NAGARAJ	43	M	82875	LEFT	8	32	8	34	7	36	4	54	3	56