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A STUDY OF OUTCOME OF AUTOLOGOUS PLATELET RICH PLASMA INJECTION IN PATIENTS WITH CHRONIC PLANTAR FASCIITIS

UNDER THE GUIDANCE OF

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I hereby declare that this dissertation entitled "**A STUDY OF OUTCOME OF AUTOLOGOUS PLATELET RICH PLASMA INJECTION IN PATIENTS WITH CHRONIC PLANTAR FASCIITIS"** is a bonafied and genuine research work carried out by me under the guidance of **Dr. N. S. GUDI,** Professor & HOD of Orthopaedics, Sri Devraj Urs Academy of Higher Education and Research Centre(Deemed University), Tamaka, Kolar**.**

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ABSTRACT

INTRODUCTION AND OBJECTIVES:

Plantar fasciitis is one of the most common musculo skeletal problems in the orthopaedic practice. Any heel pain due to plantar fasciitis persisting will often distress the patient, so right intervention at the right time is needed Plantar fasciitis is also common in the rural folks.

Need of the study is to evaluate the response rate and outcome of autologous platelet rich plasma injections in patients with chronic plantar fasciitis. In studies elsewhere autologous platelet rich plasma injections reduced heel pain.

Objectives: To study the outcome of plantar fasciitis after Platelet rich plasma injections.

Materials and methods:

Patients between December 2011 to April 2013 a study group of Sixty Two patients with hundred painfull heels persisting for 4 to 6 months with age of 18yrs and above will be taken up for the study.

Patient's characteristics like history of heel pain, age, height, gender, weight, hours spent on standing, durations of symptoms and types of prior treatment will be noted. Any intervention procedure will be done after taking the written consent by the patient.

METHODS:

Patient's with heel pain of duration of 4 to 6 months will be included in the study.

PREPARATION OF PLATELET RICH PLASMA

Autologous blood obtained from the patient will be subjected to centrifugation methods to obtain platelet concentrate. Marx technique that uses double centrifugation technique will be used. The platelet rich plasma will be studied with regard to platelet number. This will be injected with full aseptic precautions in to the painfull area of the heel.

The entire procedure is done as an office procedure. The outcome study is analyzed by its pain relief and comfortable return to daily activities. Assessing the outcome is done in 2^{nd} week and 3^{rd}month of the study group and statistically evaluated. Patients who do not follow the regime of treatment and who do not come for follow up are excluded and a complete description of responses to the outcome will be presented in the statistical data.

Discussion:

100 painful heels were screened and evaluated in this study, with base line characteristics of sex 36 were females, 26 were males. Among this 38 received bilateral PRP injections and 24 received unilateral PRP injections. Site of injection showed 53% right, 47% left. The mean age groups of patients were 43.51 ± 12.5 years and mean duration of complaints were 12.95 ± 10.13 months. There was considerable improvement in visual analogue score before injection with P value of 0.878 to VAS score after 3 months with P value 0.947.

The mean VAS value before injection was 7.48± 0.948 and after 3 months post injection it was 4.97±1.35.And hence significant improvement was noted.

In this study the base line characteristics like age, sex, duration of symptoms, site of symptoms does not affect the outcome of the study statistically. In this study the outcome of PRP injection is as follows.

31 % had 60% improvement in VAS score, 18% had 50% improvement in VAS score, 6 % of subjects had 80% with highest improvement in VAS score, and the least improvement is 10%, was observed in 5% of subjects. These results were at the end of 3 months. Majority of the PRP treated patients had improved quality of life status. The results of the study were comparable to other short term follow up studies in the literature.

Conclusion:

PRP is a biological option for a common orthopaedic and recalcitrant orthopaedic problem like heel pain/plantar fascitis.

In our study a significant number of patients showed improvement in symptoms. There were no complications. Symptomatic and persistent relief enabled the patients to perform their daily activities.

PRP injection may prove to be a superior alternative to the existing treatments for chronic heel pain.

FIGURES

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LIST OF ABBREVIATIONS

1. PRP: Platelet rich plasma
2. PF: Plantar fascitis
3. VAS: Visual analogue scale
4. ACL: Anterior cruciate ligament

INTRODUCTION:

Plantar fasciitis is a common pathological condition affecting the hind foot, and can often be a challenge for clinicians to successfully treat[9]. It is an overuse injury causing inflammation at the origin of the plantar fascia and surrounding perifascial structures, such as the calcanealperiosteum. It is the most common clinical problem that causes infero medial heel pain in adults[40].Lapidus and Guidotti[81], in 1965, found that the number of patients in their foot clinicwith plantar fasciitis was greater than those with any other recorded foot lesion. Regarding the history of this disease plantar fasciitis is an overuse syndrome has been recognized for almost two hundred years ago by an author named wood,woods[82] and other authors stated that plantar fasciitis is called by different names they are heel pain syndrome, subcalcaneal pain syndrome, calcaneodynia, subcalcaneal bursitis, calcaneal periostitis, neuritis, heel spur syndrome, subcalcaneal spur syndrome, stone bruise, medial arch sprain, runner's heel, jogger's heel, and policeman's heel[82].

The different names given by different authors is due to the confusion about the etiology[6,83] of plantar fasciitismany authors in their study stated that Successful treatment of plantar fasciitis usually requires a combination of treatment modalities, than administering only one treatment at a time[85].It is stated that mechanical interventions in combination to any other treatment will relieve the heel pain to the maximum extent, but there are studies which shows that mechanical intervention is not of much significance, the mechanical treatment modalities like foot orthoses, foot taping, footwear, night splints, rest, and walking casts is stated that these will reduce the load and stress applied to the inflamed plantar fascia to a tolerable level[19].Plantar fasciitis is the degenerative problem of the plantar fascia resulting from repetitive trauma at its origin on the calcaneus. Plantar fasciitis causes heel pain in both active and sedentary adults of all ages.[15,16]

Few authors like(kaya2010,chen2010) stated that plantar fasciitis is an inflammatory disease at an acute stage due to mechanical overload will lead to chronic inflammation and degenerative changes[85].Plantar fasciitis accounts for 11 to 15% of all foot problems in adults, it peaks between 40 to 60 years of age group and in younger age group like runners, the predominance of this condition according to sex varies from one study to other.(cole et al.2005,Gill and kiebzak 1996,Neufeld and cerrato2008)The most common site of abnormality is near the origin of plantar aponeurosis at the medial plantar tubercle of the calcaneus.[23]

Due to multifactorial[6] causes of plantar fasciitis the treatment modalities are wide in number, to relieve pain nsaids, night splints, low dye taping, heel pads, cups, orthoses, steroid injection were used, extra corporeal shock wave therapy is used in the recent years to treat this disease with life style modifications only 5 to 10% of the people will need surgical intervention like removal of calcaneal spur, neurectomy, plantar fasciotomy.

The advent of platelet rich plasma for treatment of plantar fasciitis in recent times is due to its wide advantages with early recovery of pain levels and improved functional activities of the patient in comparison with above mentioned treatments.

OBJECTIVES OF STUDY

- To evaluate the response and outcome of autolougous platelet rich plasma injections in patients with chronic plantar fasciitis.

REVIEW OF LITERATURE

PLANTAR FASCIITIS

ETIOLOGY

The exact cause of plantar fasciitis is still unknown, there are wide number of causes and thus number of other names have come up for plantar fasciitis. The variety of treatments used to treat plantar fasciitis is due to the unknown etiology and pathogenesis of this disease. Many authors have stated that this disease with many different theories of etiology and treatment has no valid proof of any one single cause as said by snook and chrisman. Many factors which are involved in this disease are divided under 3 headings.[31,36,37,83]

ANATOMIC FACTORS: Pesplanus, pescavus, obesity, limb length discrepancy and shortened achillis tendon.

BIOMECHANICAL FACTORS: Weak plantar flexors, eqinus, weak intrinsic muscles of foot, excessive sub talar pronation, poor foot wear and limited dorsiflexion.

ENVIRONMENTAL FACTORS:

Trauma, hard surface prolonged weight bearing, inadequate stretching, limited ankle dorsiflexion. In most cases it is stated that combination of all these factors are involved in the development of plantar fasciitis. Many authors have noted that abnormal anatomic foot configurations will develop plantar fasciitis like pesplanus with excessive pronation is the most common mechanical cause of development of this disease in around 80 to 86% of patients, increased pronation will decreased stability of hind foot and it will produce further strain on the plantar facia which will eventually cause plantar fasciitis.[84] Mann and Inman confirmed this by noting that heel pronation increases the tension along the medial aspect of the heel will result in instability of foot to supinate from mid to terminal stance, due to little load supported by the bones and ligaments along the mid foot the excess load is laid on plantar fascia. Few authors have noted that cavus foot is also one of the cause for plantar fasciitis due shifting of vertical load from mid foot to forefoot will result in tight plantar fascia during stance phase.[2,37] A tight achillis tendon and external rotation deformity of the lower limb will cause increase in load on the foot mainly in the intrinsic muscles of the foot during stance phase will assume plantar fasciitis due restriction of supination in almost 78% of patients.

Overuse heel, obesity and improper foot wear are some of the established causes for plantar fasciitis, author named hill and cutting stated that obesity and plantar fasciitis will account in 40% males and 80% females, improper foot wear in low socio economic patients are some of the causes in our society.[6]

DIAGNOSIS OF PLANTAR FASCIITIS:

Even now the diagnosis of plantar fasciitis is mainly based on the patient history and physical examination.[23]X rays, blood tests and EMG studies are done to rule out other disorders that cause heel pain. Pain is the most common symptom in the inferior heel region for weight bearing individuals and the morning foot pain which lasts for about 30 to 40 minutes is due to the equinus position of the foot during sleep in the night will lead to plantar fascia under tension. The main characteristic feature of pain is sharp or knife like intermittent pain, but patients with chronic pain may be dull or achy or constant and the discomfort progresses distally to the entire central band of plantar fascia.[3,6],Pain is usually insidious in nature. This condition is usually not completely disabling, however patients frequently have limitations in their routine daily activities.

Using the[85] Physical Activity sub-scales of the Health Status Questionnaire or foot function index will give us the status of the limitation of the patients functional activities. Physical examination on deep palpation will reveal the extent of discomfort to the patient and the exact location of tenderness along the plantar fascia, localized swelling can be noted in the chronic cases of plantar fascia. Recent studies have come to differentiate plantar fasciitis with other causes by ultrasonography and bone scintigraphy.[14,38,83]

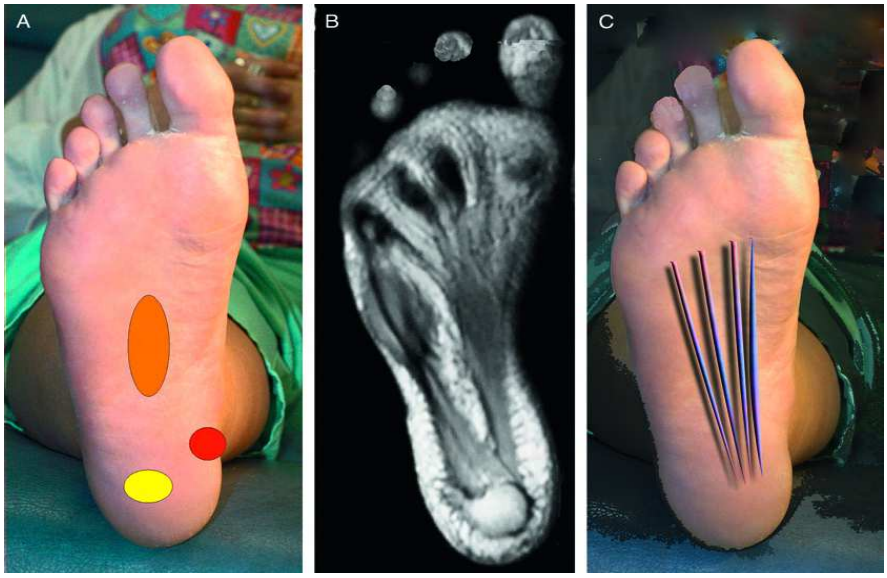


Figure-1: Yellow indicates central calcaneal tuberosity pain, red indicates along plantar medial tubercle, orange indicates localized along plantar fascia.

DIFFERENTIAL DIAGNOSIS:

This disease is an age related and treatment is time consuming. As it is a limiting condition with symptoms persist for 18 to 36months[apleys],In plantar fasciitis the pain is at the level of sub-calcaneal level, it should be differentiable from inflammatory conditions like local causes are sub calcaneal heel pain syndrome, periostitis, painfull heelpad, subcalcanealbursitis, tenosynovitis (Flexor hallucis longus, flexor digitorum longus)calcaneal apophysitis (severs disease).Systemic causes: seronegative spondyloarthropathies, ankylosing spondylitis, reiters syndrome, psoriatic arthritis, behcet syndrome, rheumatic arthritis, gouty and pseudo gouty arthritis. Other causes: Atrophy of fat pad , calcaneal spurs and other neurological conditions. Plantar fasciitis is often called "heel spur syndrome," although this terminology is confusing the clinicians because 15 to 25% of the general population without symptoms have heel spurs and half of patients with plantar fasciitis do not have heel spurs.[23,40,83] heel spur is a bony osteophyte located at the medial process of the calcaneal tubercle any greater pull of the plantar facsia will lead to periosteal hemorrhage and inflammatory reaction and will lay a new bone which will lead to spur formation which may be asymptomatic in nature[40],Literatutre says that heel spur is more often associated with flexor digitorum brevis muscle rather than plantar fascia. Differential diagnosis includes rupture of the plantar fascia, inflammatory rheumatologic conditions, tumors, nerve entrapment, tarsal tunnel syndrome, stress fracture of the calcaneus, fat pad atrophy, subcalcaneal bursitis and calcaneal periostitis. Acute heel or arch pain suggests rupture of the plantar fascia, especially following athletic activity. Bilateral symptoms could represent a manifestation of an inflammatory disorder. In younger patients with bilateral plantar fasciitis we should rule out inflammatory disorders like rheumatoid arthritis, spondylitis, Reiters syndrome[82 83]. Any older patient with bilateral plantar fasciitis may have gout or osteomalacia, these symptoms will raise the suspicion because they are not relieved by conservative means. Nocturnal pain should raise the suspicion of several causes of heel pain such as inflammatory disorders, tumors, and neuropathic pain including nerve entrapment and tarsal tunnel syndrome.

Heel pain was recently reported to involve the nerve to abductor digitiminimi, which supplies a motor branch to the abductor digitiminimi and sensory branches to the periosteum and plantar fascia. In 20% of the cases of inferior heel pain, the pain may be caused by this nerve being trapped, or affected by inflammation of the plantar fascia.[10]Tenderness on mediolateral compression of the heel (squeeze test) should lead to a suspicion of a stress

fracture of the calcaneus. Tenderness in the center of the posterior part of the heel may be due to atrophy of the heel pad, subcalcaneal bursitis or calcaneal periostitis .

To rule out other causes of plantar fasciitis many clinicians prefer stress views xrays with weight bearing antero posterior and lateral to rule out stress fractures of calcaneus, tumours, rheumatoid arthritis changes in calcaneum or erosions due to sub calcaneal bursitis. Positive percussion tinel sign on the medial side of the heel should raise suspicion about the nerve entrapment to abductor digitiminimi or a tarsal tunnel syndrome. Complete blood count with erythrocyte sedimentation rate is done in patients to rule out inflammatory disorders in atypical type of plantar fasciitis.[83]

The best method to diagnose plantar fasciitis is by clinical presentation with pain and palpable tenderness in the area of the medial tubercle of the calcaneus at the heel. (Neufold and Cerrato2008)[23].Plantar fasciitis is an self limiting disease for it to heel with conservative treatment like stretching exercise it may take 8 to 12 weeks duration for healing acute cases of plantar fasciitis.[APLEYS,83]

EVOLUTION OF TREATMENT FOR PLANTAR FACSIITIS:

In our present day daily practice we treat plantar fasciitis in a conservative approach, for the healing to occur it may take few weeks to months about 18months[40,80.],the used treatments are nsaids, low dye taping, heel pads, cups, orthoses, soft soled shoes and night splints, most of them have limited scientific evidence of their efficacy. (crawfod and thompson 2003[4])Stretching programmes are frequently used to reduce plantar fasciitis symptoms, it increases the flexibility of plantar fascia, it recreates the windlass mechanism so that the tissue tension is restored, other treatments used in combination with stretching are anti inflammatory medication, heel cushion, heel cups, injections and rest.

(DAVIS ET AL.1994)[7],he reported that plantar fasciitis pain will subside in 90% of the patients with in 18 months. Martin et al.(1998)[20] treated patients with a standardized protocol consisting of stretching of plantar fascia, a night splint and a heel cup,51% of patients reported complete pain relief. An author named (probe et al.1999)[26] said that with achillis stretching or night splint will reduce pain and will prevent contracture of plantar fascia. Orthoses such as heel pad and arch support decrease excessive pronation and reduce the biomechanical loading of the foot(Neufeld and cerrato 2008).The cushioning from orthoses reduce shock in walking by 42%.(DeMaio et al.1993).

(Lynch et al.1998)[8,19] reported that 80% success rate is noted in orthoses,30% in steroid injections,33% using NSAIDS. But the effectiveness of treating plantar fasciitis using orthoses is still inconclusive by (cole et al.2005)[3].

One clinical randomized study done by Landorf et al.(2005)[18] compared results using orthotic treatment, he noted no improvement of pain in 12 months duration, but their functional status improved.

Landorf in the year 2005 also stated that custom made shoe insoles may improve function but not pain in the span of 3months.Gudeman et al.1997 stated that other local physical therapies like ultrasound, lasertherapy, iontophoresis are also used for treating plantar fasciitis, he reported pain relief after 2 weeks of treatment of treatment but no significiant difference is noted after 1 month of treatment. Thompson et al.(2005)[32] has done a randomized control study on the effects extra corporeal schock wave therapy for plantar fasciitis patients, he stated that this treatment is widely used from the year 1990,he noted that this treatment has better pain relief but the duration of treatment is longer. At present day many doctors advise life style modifications like strengthening exercises (swimming, cycling), physical therapy as the best modes of treatment to prevent the disease to progress further.

Surgical management is advised only when all the conservative therapy fail, Only 5 to 10% cases progress to surgery(O'Malley et al.2000)[24] few surgical options are Removal of calcaneal spur which may be one of the cause of heel pain, this procedure was started from early 19th century by classical medial approach just above calcaneum'.Other procedures like neurectomy of the medial calcaneal nerve which is one of the cause of heel pain and release of its adhesions. Plantar fasciotomy was performed since 1930's. Many patients had pain relief in 3 weeks with the removal of degenerative plantar fascia and spurs but care to the medial calcaneal nerve, post operatively all the surgical procedures improve by 3 months within maximum period of 6 months as such many surgeons noted many complications like medial calcaneal nerve injury, heel pad numbness, neuroma formation, delayed wound healing, deep venous thrombosis, superficial and deep infections and iatrogenic calcaneal fracture after spur excision.

The advent of other treatment have started from late 19th century. the advent of new treatments have started after the failure of conservative treatments, NSAIDS are helpful only in acute cases, the prolonged use of NSAIDS will cause drug induced gastritis, For patients who do not tolerate NSAIDS, local steroid injection can be used for both theraputic and diagnostic intervention[5],

The complication which occurs after injection of 2 steriod injection are plantar fascial rupture, steroid induced fat necrosis, the injection of steroid into heel pad will cause mechanical disruption of fibrous septa and fat necrosis with loss of shock absorbing capabilities. Other treatments like taping and cups are designed to remove stress from plantar fascia and to restore its compressibility. Orthoses are used to control pesplanus and over pronation, as pronation is one of the causes of plantar fascia pain, it also help to decrease tension on plantar fascia during gait, but the use of orthosis is a time taking process of the disease to regress[23]. Other treatments recommended like stretching exercises to stretch plantar fascia and achillis tendon for 4 to 6 weeks, but it may be helpful in acute cases.

The cast treatment is only helpful in some chronic and resistant cases but the results are varied. Radiation therapy which is used now a days to relieve spasms, reduction of pain, which can increase blood circulation has good results but in along run it has disappointing results. with many advantages and disadvantages of both conservative and surgical treatment a new method of treatment with platelet rich plasma started from the year in the early 90's which shows promising results in the treatment of plantar fasciitis.

TREATMENT MODALITIES:

Conservative approach: Many studies stated that 90 to 95%of patients will be releived with their symptoms in the time bound manner without the need of any surgical intervention.

1.) To reduce pain and inflammation.

Anti inflammatory medications-to reduce pain and swelling ,they are Nsaids and steroids. but disadvantages of this treatment is that it does alter the structural changes of plantar fascia, advantage is that it has immediate pain relief. Many disadvantages with cortisone injections they are osteomyelitis of calcaneum, loss of cushioning, fat pad atrophy, collagen degeneration and calcification, weakness and rupture of plantar fascia, they are usually followed by recurrence of symptoms. In addition to medications, a variety of physical agents, including iontophoresis, phonophoresis, ultrasound, cryotherapy, and hydrotherapy, have been described as effective in the management of plantar fasciitis, but their effectiveness of treatment is not well understood, they have wide varied results. With the advent of many advantages and disadvantages in treating plantar fasciitis a new treatment in the year 1992 was started in Europe that is a shock waves to treat any musculoskeletal problems. Even though the treatment is safe the exact effectiveness of this treatment is not well understood.[3,7,19 83]

2.) To reduce tissue stress.

Few treatment modalities like foot orthoses, foot taping, change in foot wear, has been used to reduce the amount of stress to the inflamed tissue and correct the foot pronation which is associated with plantar fasciitis.

Other treatment modalities like night splints, rest and walking casts but there are doubts in their effective treatment. Orthotic devices are mainly custom orthotics, heel pad, the material used may be rigid, semi rigid and soft arch supports, many people prefer semi rigid material due to its softness and its application which will relieve the stress on the plantar fascia. In chronic plantar fasciitis with marked limitation of activity studies have shown that it is best treated with below knee or walking cast for the duration of 3 to 6 weeks, it provides rest to the plantar fascia, reduces pressure on the heel at heel strike, provides support for the arch and prevents tightening of the achillis tendon.[18,19]

3.) To restore muscle strength and flexibility.

Most patients have tightness of achills tendon with shortening of plantar fascia due to pain[1]this will increase stress on the inflamed fascia during gait. Many authors like boyd in 1992 and davis et al.stated that stretching is the most effective treatment toassume the foot in more midline or supinated foot in mid or terminal stance which will reduce strain on the fascia.[7]

SURGICAL OPTIONS:

Many surgeons do not prefer surgical treatment as it is best cured by conservative treatment, surgical options are only for patients within irreducible heel pain which is restricting their functional activities. Surgical treatment is the last resort for treating plantar fasciitis when the conservative treatment has failed ,as such many authors say that there is no time limit for any surgical intervention, few prefer 1year and above duration while others prefer more than 2 years duration[86] as said by Howell. There have been more than 30 surgical series reported on the treatment of plantar fasciitis in the literature. The operations have included drilling decompression of the calcaneus, Steindler stripping, plantar fasciotomy, excision of a heel spur, neurolysis of the nerve to abductor digitiminimi, neurolysis of the calcaneal nerve, and calcaneal neurectomy.is surprising is that almost all of these interventions have been associated with a high success rate.

Studies have shown that there is good success rate in the span of 8 months with rehabilitation. Post-surgical complication as noted by Huang et al.is that the plantar fascia is

an during plantar fasciotomy has got deleterious effects they are decrease the stiffness of the foot, less rigid and deformed arch.[24 83,86]

EVOLUTION OF PRP AS A MODALITY OF TREATMENT;

Autologous PRP was first used in cardiac surgery by Ferrari et al. in 1987 as an autologous transfusion component after an open heart operation to avoid homologous blood product transfusion . PRP has also been use in conditions like Soft tissue injuries treated with PRP include tendonopathy, tendonosis, acute and chronic muscle strain, muscle fibrosis, ligamentous sprains, and joint capsular laxity. PRP has also been utilized to treat intra-articular injuries. used in multiple specialties such as maxillofacial, cosmetic, spine, orthopedic, and for general wound healing.The use of platelet rich plasma in musculoskeletal problems was started from early 1990s,its wide application of: Examples include arthrofibrosis, articular cartilage, arthritisdefects, meniscal injury and chronic synovitis or joint inflammation its main application is due to its mechanism and effectiveness of treatment Robert marx in the year 2004 has described its use in oral maxillofascial surgeries with good results.

Author Allan Mishra evaluated 20 patients for musculoskeletal injuries all the patients had improvement in the pain levels,60% improvement is noted all prp patients with 1 injection had lower pain and good range of motion. Barrett noted results in plantar fasciitis patients he noted good results with complete relief of pain and good functional motion[88].

Aizasayedomar, in his study in Egypt noted that 73% improvement noted in musculoskeletal injuries, it was done in the year 2011.

Barrett and Erredge had studied the effectiveness of prp injection in plantar fasciitis patients 77.8%success rate is noted. Kiteret al.in the year 2006 had studied the results of prp injection in the study group of 54 patients showed 68% results[87].

Joost c peerbooms[75] in the year 2010 has done a study on plantar fasciitis which showed good results. Mark w.scoli in the year2011 had done a study on plantar fasciitis with 30 patients, good relief was noted in 28 patients in 6 months follow up[74].

Another study done by Akshanin et al in the year 2012 showed good results for injecting platelet rich plasma in patients with chronic plantar fasciitis.

FUNCTIONAL ANATOMY OF FOOT:

Human foot is composed of 28 bones and 33 joints,(Caillet et Al.1996)[79] organized these structures in to 4 segments, they are 1.rear foot (tarsus). 2.midfoot(lesser tarsus). 3. forefoot(metatarsus).4.phalanges.

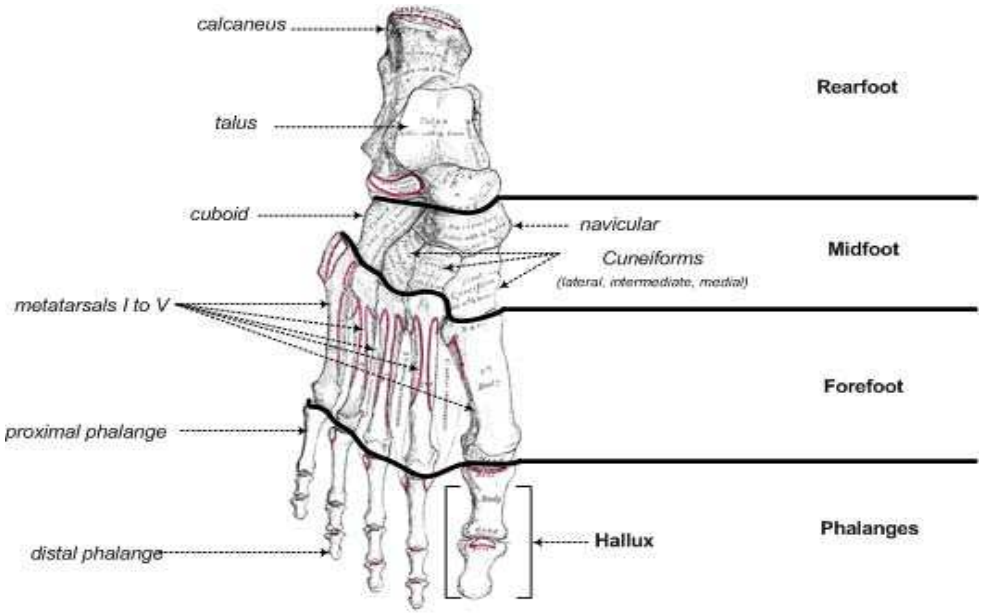
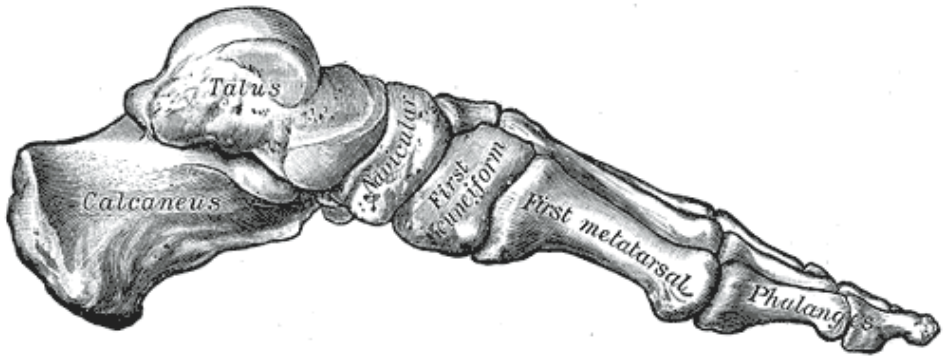


Figure 2. Bones (italicized) and segments (bolded) of the healthy human foot (adapted from Gray, 1918

For an functional aspect of foot three arches are important, they are medial longitudinal arch, lateral arch and transverse metatarsal arch, the medial longitudinal arch is the largest and most functionally important of all the three arches, bones that constitute the medial arch is talus, navicular, calcaneus, three cuneiforms and three metatarsals.



Fi-3:The bones of the medial longitudinal arch (Gray, 1918)

FUNCTIONALANATOMY OF PLANTAR FASCIA

The plantar fascia is a fibrous band of connective tissue, originating from the plantar medial tubercle of calcaneous, it is composed of 3 major bands Medial, Central ,Lateral of which the meta-tarsophalyngeal joints, the thinner medial and lateral segments run distally and coalesce with fibers of central segment to form the origins of intra muscular septa .Studies done by young et al.(2000)[40] has shown that plantar fascia is an passive contributor of medial longitudinal central is the strongest and the thickest of the three.(The central segment courses distally and at the level of metatarsal bases, separating in tho 5 slips that attach to the plantar plates of each of the arch, any surgical release will lower the medial longitudinal arch.(Hicks,1954.daly et al.1992)[13,

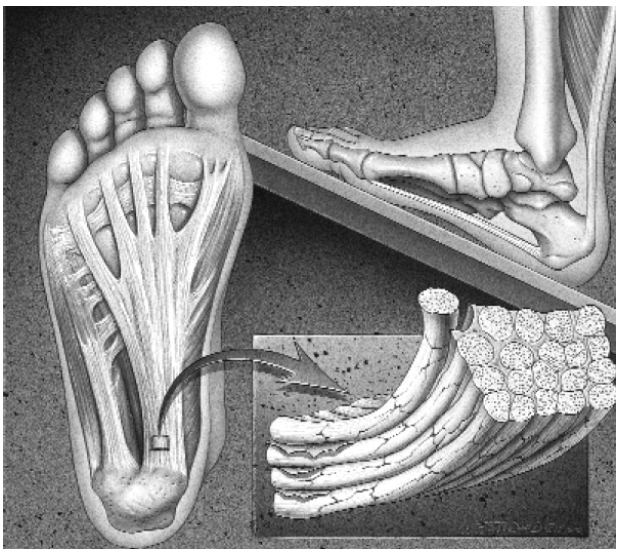


Fig-4: The plantar fascia (Young et al., 2001)

Its orientation of fibers mainly longitudinal and some transversly.(grey's anatomy)43 The medial and lateral borders over intrinsic muscles of hallux and fifth toe, while the central part overlies the long and short flexor's of digits.(grey's anatomy) 83 At the attachment of the medial process of calcaneal tuberosity it is narrow posteriorly.(grey's anatomy)

The fascia attachment is proximal to flexor digitorumbrevis attachment at the level of medial calcaneal tuberosity.(grey's anatomy)

The fascia while traced distally it becomes broader and thinner and at the level of meta tarsal heads it divides into five bands one for each toe.(grey's anatomy) .These five bands diverge below the metatarsal shafts and attaches to the proximal plantar and little distal to the metatarsal heads and the metatarsal joints, they are all united by transverse bands.(grey's anatomy)

The medial band of aponeurosis covers abductor hallucis is thin, it continues proximally with flexor retinaculum, medially with the fascia of dorsalispedis laterally with central part of the plantar aponeurosis, the lateral band covers abductor digitiminimi which is thin distally and thick proximally it continues medially with the central part of aponeurosis

MUSCLES OF THE ARCH OF FOOT:

INTRINSIC MUSCLES ARE:

There are four main Compartments of the foot by Jones 1949. Plantar fascia surrounds the medial and inferior portion of medial compartment the inferior portion of central compartment is surrounded by plantar fascia the lateral compartment is surrounded by fascia inferiorly and laterally,their important role is to maintain the medial arch.

The compartments are

Medial compartment:	Abductor hallucis
	Flexor hallucis brevis
Central compartment:	Flexor digitorum brevis
	Adductor halucis
Lateral compartment:	Abductor digiti minimi
	Flexor digiti minimi brevis
Fourth Interosseous compartment:	7 interossi
	Introsseous fascia
	Metetarsals.

Few studies by(Headle et al.2008) stated that when the intrinsic muscles are fatigued will lead to decrease in medial arch height.

EXTRINSIC MUSCLES:

Tibialisposteror is the most important muscle which support the medial arch,as stated by(Funk et al., 1986; Thordarson et al., 1995; Dyal et al., 1997;) medial arch will be effected in cases of dysfunctional or rupture of tibialis posterior muscle.

Innervation:

It is supplied by Posterior tibial nerve giving rise to medial calcaneal nerve at the site of medial malleoli most commonly piercing flexor retinaculum and innervate medial aspect of heel.Sural nerve gives rise to the lateral calcaneal nerve at the level of lateral malloli innervate lateral aspect of foot.

VASCULARITY OF PLANTAR FASCIA:

By its nature plantar fascia is an relatively hypovascularstructure,blood supply to plantar fascia from the the lateral plantar artery which is an branch of the posterior tibial artery.(moore et al.2010)[21,22]

ANATOMY AND PHYSIOLOGY OF FUNCTION OF PLATELETS:

PLATELET ANATOMY AND FUNCTION

Platelets are small discoid blood cells (approximately 1-3 μm). The average platelet count ranges from 1.5-3.0 x 10-5 per mL of circulating blood and the half-life of platelets is about seven days. Platelets are formed from megakaryocytes and are synthesized in bone marrow by pinching off pieces of cytoplasm. Thereafter, platelets are extruded into the circulation. Platelets have a ring of contractile microtubules (cytoskeleton) around their periphery, containing actin and myosin. Inside the platelet, a number of intracellular structures are present containing glycogen, lysosomes and two types of granules. These are known as dense granules, which contain ADP, ATP, serotonin, and calcium. The α-granules contain clotting factors, growth factors, and other proteins. Platelets are equipped with an extensively invaginated membrane with an intricate canalicular system, which is in contact with the extracellular fluid. Normally, in the resting state, platelets are nonthrombogenic and require a trigger before they become a potent and an active player in hemostasis and wound healing. Upon activation (e.g. by thrombin) they change shape and develop pseudopodia, which promotes platelet aggregation and the subsequent release of the granule content via the open canalicular system.[41]

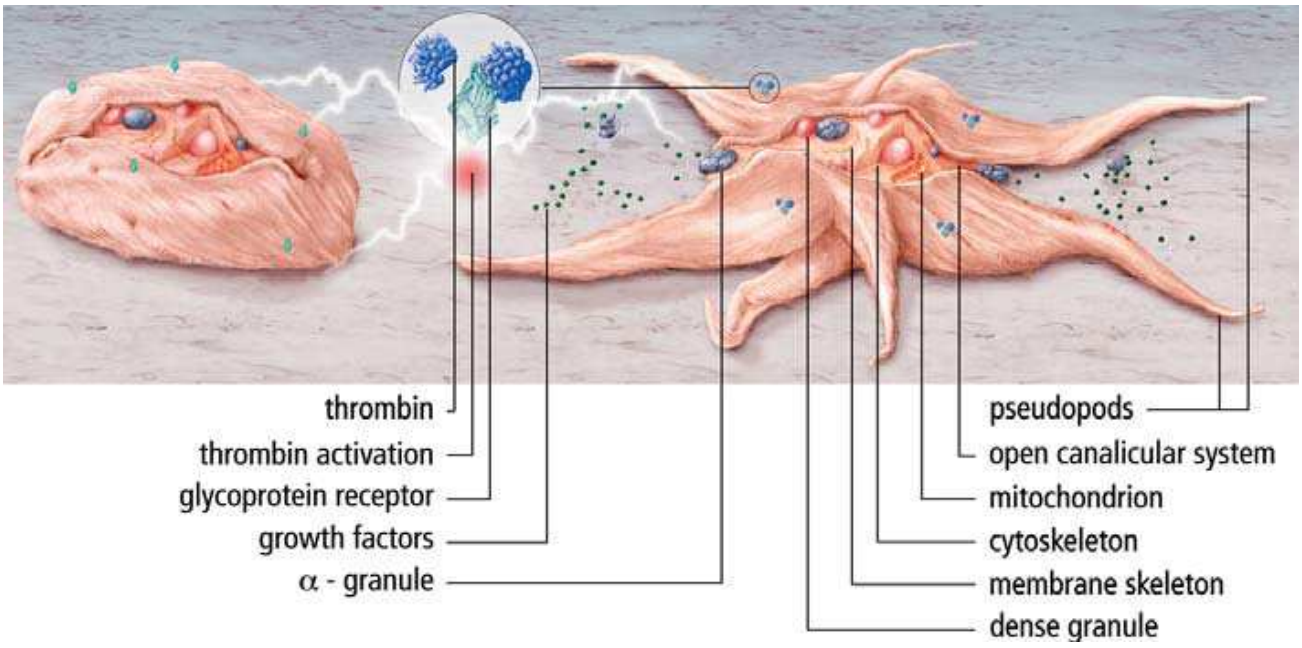


Fig-5: Structure of platelets

Platelets play a role in heamostasis with plasma proteins and low molecular weight substances, activation of platelets are done on adhesion to damaged blood vessel wall which in turn act via acts via the glycoprotein Ib and IIb/IIIa receptors, which are present in the platelet membrane and then they become activated upon activation the platelets change from discoid to spherical shape and aggregate to injured tissue.[42,43] Based on the fundamental role of platelets in hemostasis, as discussed above, it may be hypothesized that exogenously applied PRP would contribute to a more effective hemostatic condition of (surgical) wound surfaces, where it attaches to tissues as a solid platelet plug. Stover *et al.*[45]

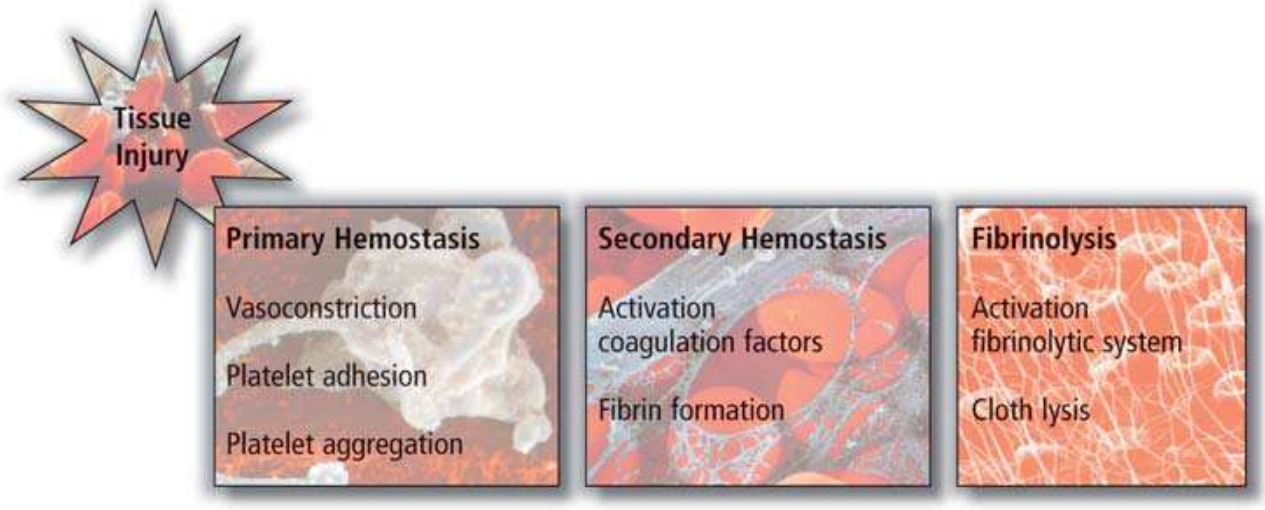


Fig-6: Stage of activation of platelets

Secondary hemostasis is initiated with the activation of coagulation factors and the formation of a fibrin network that stabilizes the platelet plug[45]. The final step is the activation of leukocytes invading the affected area with the release of cytokines which then activate the fibrinolytic system leading ultimately to clot lysis. The healing process in wound healing is a well orchestrated and a complex one, the process of events are first 2 days inflammatory process are initiated, during 3rd day angionesis and fibroplasias from 3rd to 5th day it is the beginning of collagen synthesis.[46,47]The process of binding of platelets to the injured tissue by platelet tyrokinase receptor which is present on cell membrane of tissue cells.[53,54]

GROWTH FACTORS IN PLATLETS:

PDGF is a glycoprotein with a molecular weight of approximately 30 kD, with two disulphide-bonded polypeptides, referred to as A and B chains. There are three isoforms, PDGF AA, -BB and AA-The most specific function of PDGF includes mitogenesis and angiogenis[55,56] and macrophage activation .Bowen-Pope[57]., studied the production of PDGF and concluded that there are approximately 0.06 nanograms of PDGF per 106 platelets, or

about 1200 molecules per platelet66. Therefore, one might assume that PG with a platelet count in excess of 3 to 5 fold the baseline level would have a profound effect on both wound healing and bone regeneration.[57] Transforming growth factor beta are the group of proteins with a molecular weight of 25kd, TGF-β is part of a super family to which BMP also belongs. In humans, three subtypes of TGF- β are present, but[58,59] TGF-β1 and TGF-β2 appear to be the most important with regard to general connective tissue repair and bone regeneration. TGF-β is found predominantly in platelets which account for 95% of the total, while some is also found in macrophages, the other functions of TGF-β are to promote chemotaxis and mitogenesis of fibroblasts and osteoblastic precursor cells.[60,61]Very recently, Kubota and others described a new PGF known as Connective tissue growth factor.[62]The ctgf component is 20 times more of pdgf ,its main functions are angiogenitic activity cartilage regeneration and fibrosis. According to literature studies done in animals which shows the source of platelets are elaborated in the following way[63,64]

Growth Factor Source

Transforming Growth Factor-beta, Platelets, extracellular matrix of bone, cartilage matrix, activated TH1cells and natural killer cells, macrophages/monocytes and neutrophils. Basic Fibroblast Growth Factor-Platelets, macrophages, mesenchymal cells, chondrocytes, osteoblasts. Platelet Derived Growth Factor- Platelets, osteoblasts, endothelial cells, macrophages, monocytes and smooth muscles. Epidermal Growth Factor- Platelets, macrophages, monocytes Vascular endothelial growth factor, Platelets, endothelial cells VEGF- Connective tissue growth factor, CTGF Platelets through endocytosis from extracellular environment in bone marrow.[62,66]Normal platelet activation leads to three necessary stages of healing: Inflammation, Proliferation, and Remodeling (9).

Inflammatory phase - Functions of platelets upon activation

- Anti-microbial
- Adhesion
- Aggregation
- Clot retraction
- Pro-coagulation
- Cytokine signaling
- Chemokine release
- Growth factor release

Following the initial inflammatory phase, which typically lasts for two to three days, fibroblasts enter the site and begin the proliferative phase . Low pH and low oxygen levels stimulate fibroblast proliferation in the injury site . Fibroblasts become the most abundant cell by day no. 7. The fibroblasts are then responsible for deposition of collagen and ground substance. This phase lasts for two to four weeks.

The Proliferative **Phase** – Fibroblast function

• Wound contraction
• Peaks day 5-15
• Can last for weeks
• Fibroblasts differentiate into myofibroblasts
• Actin contracts making wound smaller

Low pH and hypoxemia also stimulates neovascularization. Neovesselsbegin to form at approximately day 5 to 7 and this process proceeds until the neovessels disappear near completion of the remodeling phase.

The **Remodeling Phase** – Collagen maturation and strength. Biotensegrityrepair

• Starts when production and break down of collagen equalize
• Can last over a year
• Type III collagen is replaced by Type I collagen
• Reorganization occurs
• Blood vessels disappear

It has become apparent, then, that PRP function via a triad of interactions, known as the cell proliferation triangle . Each piece of this triangle must be present for effective tissue repair and pain relief.

Haynesworth*et al.*, studied the response of PRP on cellular mechanism of adult human mesenchymal stem cells (ahMSC) [66]. In soft tissue and bone healing, ahMSC are essential components for the repair process[67]. It was shown that release of PRP growth factors stimulates the migration and proliferation of ahMSCs, in a PRP concentration dependent manner. A significant cellular response occurred with a 4 to 5 fold increase of platelet count, when compared to the baseline platelet count.

In another study, Liu *et al.*, showed that the fibroblast proliferation and type I collagen production were augmented by a 4 to 5 fold increase in the PRP platelet count[68]

PLATELET RICH PLASMA FIGHT AGAINST INFECTION:

However, little attention has been given to the role of the WBC, despite the fact that PG is a buffy coat product, including both neutrophils and monocytes containing high levels of myeloperoxidase (MPO), which might contribute to bacterial killing[69]. Theoretically, PRP might be an ideal autologous prepared biological blood product, rich in growth factors with enhanced antimicro bacterial capabilities. The neutrophils and macrophages are agents which kill the bacterial pathogens when suspended with platelet rich plasma, the release of myeloperoxidase from neutrophils act as bacterial toxins.[70]The myeloperoxidase catalyzes the oxidation of chloride to generate hypochlorous acid and other reactive oxygen radicals, these substances act as potent bacterial oxidants and kill microorganisms and fungi.[70]There are recent studies which shows the release of antimicrobial properties from platelet rich plasma is also effective from staphylococcus aureus infection as noted by Hampton and klinger.[71,72]

In a study done by Robert marx after his study in microbiological laboratories which shows that PRP with an ph of 6.5 to 6.7 which is more acidic compared with an mature blood clot of 7.0 to7.2 does not promote infections.[39] The amount growth factors yield depends on the preparation method and human variability, according to study done by weibrich and kiles[73] suggested that there is no benefit when poor content of PRP is applied to patient.

PREPARATION OF PLATELET RICH PLASMA:

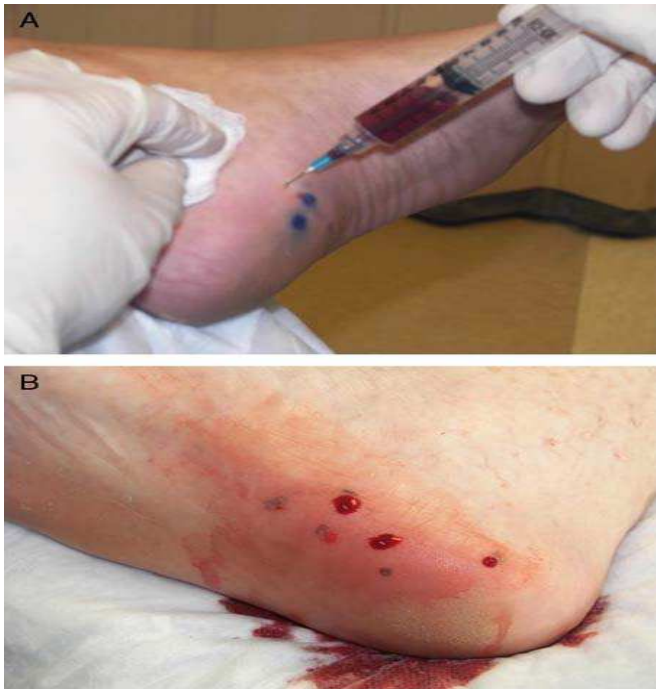
For collection of blood approximately 5% of body weight,the blood is drawn from the medial cubital vein,use of appropriate anticoagulant and refrigrated technique with discarding platelet poor plasma and red blood cells.[76]True PRP is always autologous and is not homologous. An example of this confusion is the use of lyophilized donor platelets. Homologous platelets are not viable and could not possibly secrete bioactive growth factors. Homologous platelets are also antigenicdue to their abundance of cell membranes. Certainly, antiplatelet antibodies could develop from this product and second set reactions would follow. Such substances offer no useful comparison to PRP.[77]PRP works via the degranulation of the _ granules in platelets, which contain the synthesized and prepackaged growth factors. The active secretion of these growth factors is initiated by the clotting process of blood and begins within 10 minutes after clotting. More than 95% of the presynthesized growth factors are secreted within 1 hour.[77,78]PRP has been shown to remain sterile and the concentrated platelets viable for up to 8 hours once developed in the anti coagulated state and placed on a sterile surgical table. .

The red layer indicates platelet rich plasma and the yellow indicate platelet poor [74]plasma.

Fig-7: Layers of platelet

This picture is taken from a study done by SCIOLI M. from the Journal general clinical orthopaedic society[74].

They site of injection of platelet rich plasma at the maximum tender point from medial to lateral side in an transverse plane,proximal to attachment of plantar fascia.[74.]



These pictures which showes site of application is taken from the study done by SCIOLI M. from the Journal general clinical orthopaedic society[74].

Fig-8: Site of injection

BIOMECHANICS OF PLANTAR FASCIA

Plantar aponeurosis maintains the longitudinal arch of the foot.

WINDLASS MECHANISM:

At toe off when the metatarsophalyngeal joints are extended the plantar fascia becomes taut, causing the height of the arch to increase, the rear foot to re-supinate and the foot to become a rigid lever then causing propulsion.[2,37] The *windlass* model is the theory on the relationship between the plantar fascia, toe dorsiflexion are medial arch kinematics (Hicks, 1954)[13]. In this model, the foot is represented by two rigid beam segments resembling the rear foot and forefoot Fig:) The plantar fascia, metatarsal head and proximal phalanx were modeled as a cable, a windlass drum and a drum handle. Hicks also stated that higher arch is always an stable foot, Sarrafian(1987) expanded on hicks idea that plantar fascia is strengthened and tensioned when the windlass mechanism is engaged that is when the toes are dorsi flexed which will cause push off.

Fig-9: Windlass mechanism (Hicks, 1954).

- The architecture of the foot resembles a roman arch, it describes the protection against lateral movements of both ends.[13]
- Shear load of flat joint surfaces is avoided in the architecture of arch of foot and it helps in gait when the heel is lifted from the floor at push off.[2,30]
- The pattern of arch can be well made at in stress x-ray's the pattern of trabaculae, the hydroxyappetite lines can be made out.
- According to mantor whole foot involves in weight bearing, so for the maintaining of arch both internal stress and external forces should maintain an equilibrium, therefore its necessary for fixation of metatarsal heads by Hick's[13]
- Maximum weight / strain exerted at plantar fascia during midstance and push off of stans phase by scott winter[48.]
- According to Thordarson and colleagues the dynamic support provided to longitudinal arch by muscles and plantar aponeurosis during the stance phase of gait.

The tendons posterior tibialis, flexor digitorumlongus, flexor hallucislongus, peroneus longusbrevis act as force transducers.

- Plantar loads up to 700 neutons, the significant arch is maintained in dorsi flexion of toes.
- According to Huang and colleagus the stiffness of arch depends on short and long plantar ligament.

  Plantar calcaneo-navicular ligament.

PATHOLOGY: Injury to medial border of the tissue at the origin of calcaneous.

- It is considered as serious injury due to long recovery period its causes are multifactorial.
- At muscular level its caused by overload of posterior tissue of calf and foot.
- Kibler and colleagues found deficiencies in muscle strength and flexibility in many patients with plantar fasciitis.
- Anatomic hyperpronation is one of the cause for plantar fasciitis, so the limb should be kept opposite to pronation to relive pressure and pain as told by Bojsen-moller[2].
- The main objective to treat plantar fasciitis is to relieve any tenderness along the medial plantar surface and to reduce the excess pressure in that area and to reduce any tendency towards pronation.[2]
- Perry reported that absence of triceps pull on calcaneus leads to plantar fascia contracture that will lead to loss of arch support.
- Cavus foot type is associated with high plantar pressure due to increased inclination of first metatarsal head and navicular.[56]
- According to Karr there is a correlation that exists between decrease first metatarsal joint movements in patients with plantar fasciitis.
- Plantar fasciitis is one of the cause for heel pain syndrome, it is the gradual onset pain described as burning sensation with maximum tenderness at just anterior to the plantar medial calcaneal tubercle.
- Plantar fascia pain is described as more intense pain in the morning first foot step of the bed, the pain follows the period of non-weight bearing and rest.

  The pain also recurs after prolonged weight bearing activity and continues until rest.[3,6]
- The occurrence of pain during early ambulation after a period of non-weight bearing and rest is the pathognomic feature for heel pain syndrome.

- Bilateral presentation of plantar fascities has been reported in 4 to 30% of the patients.
- We should rule out the cause of heel pain which cause the limp and referred pain from calf, knee or hip.
- There are multifactorial causes of heel pain may present secondary to local and systemic disorders.[6,25,31 83]
- The tenderness associated with plantar fasciitis heel pain is due to maximal strain on the plantar fascia ,which results in fascial and perifascial inflammation ,micro tears and fibrosis of the plantar fascia at the site of origin.[15,16]
- Few studies have reported that **changes** in plantar fascia are

  Collagen degeneration (Lemont et al.2003)[17]

  Angioplastic hyperplasia

  Chondroid metaplasia

  Matrix calcification

  The findings contribute to incomplete healing and repair, chronic inflammation and fatigue failure of plantar fascia.[17]
- With multifactorial cause of plantar heel pain [85]Snook and Chrisman stated that pathology with many causes does not have a valid proof of any one cause.
- According to study done by Rubin and Witten 125 of 461 patients were asymptomatic for heel pain had plantar calcaneal spurs and those with heel spurs only 10% experienced heel pain.
- Tanz also found that 50% of his patients with plantar heel pain had plantar calcaneal spur.
- An anatomic study done by Schepsis and co-workers found that plantar spur is located at the origin of short flexor tendon not at the origin of plantar fascia as initially thought by our elders.[31]
- So the study says that it is not the spur that causes heel pain but may be by inflammation or any nerve contrapment.
- Another study done in USA shows that 73% of people with heel pain had radio-graphic evidence of calcaneal hyperostosis.

  So the cause may be spur or may not be the spur alone.
- Routine x-rays anterioposterior, medial oblique are done for all patients to rule out the presence or absence of spur, but also assist in ruling out less frequent causes of heel

pain. Such as calcaneal cyst, foreign body, bony tumour, osteomyelitis or stress fracture.[38]

- The horizontal orientation of spur usually noted from the calcaneal tuberosity.[38]
- According to studies done by Borkowitz and colleagues the MRI study shows that the thickness of plantar fascia is increased to 7.40mm to 7.56mm in symptomatic patients, whereas the asymptomatic patients thickness vary from 3mm to 4mm.[1,12]
- In a study of Wall and associates the study is done with ultrasonography the thickness of symptomatic patients had significant increase in thickness of plantar fascia with inflammatory changes, thickness of greater than 4mm or more.[35]
- A routine lateral radiography of the foot may demonstrate distortion of the soft tissue planes or periostitis.[35]
- In chronic cases of plantar fasciitis the traction exostosis of the medial calcaneal tuberosity, a calcaneal spur may form.
- The soft tissues inferior to the medial tuberosity or calcaneal spur when thickened and inflamed are responsible for pain.

  The calcification of soft tissues inferior to spur are also responsible for pain.

MATERIALS AND METHODS:

SOURCE OF DATA:

Patients between December 2011 to October 2013 in the department of Orthopaedics in R L Jallapa hospital and research centre attached to Sri Devaraj Urs medical College, Tamaka Kolar, with heel pain due to chronic plantar fasciitis will be added to study group and followed up from the time of therapeutic intervention to a minimum period of 3 months.

MATERIALS:

Study population:

Patients between December 2011 to October 2013 a study group hundred painful heels who had heel pain persisting of 4 to 6 months with age of 18yrs and above will be taken up for the study.

Patients characteristics like history of heel pain, age, height, gender, weight, hours spent on standing, duration of symptoms and types of prior treatment should be noted.

Any intervention procedure will be done after taking the written consent by the patient.

METHODS: Patient's with heel pain of duration of 4 to 6 months will be included in the study.

PREPARATION OF PLATELET RICH PLASMA

Autologous blood obtained from the patient will be subjected to centrifugation methods to obtain platelet concentrate. Marx technique that uses double centrifugation technique will be used. The Platelet rich plasma will be studied in regard to platelet number, this plasma will be injected with full aseptic precautions into the pain full heel.

The entire procedure is done as an office procedure.[87]

Stage 1:

Precautions:

1. With draw anti platelet drugs 10 days before the procedure.
2. Approximately 5% of the body weight is the estimated volume of the blood to be withdrawn.

Stage 2:

Blood collection-Select suitable vein,collect the blood using standard procedure with double bag, complete collection with in 10 minutes.

Stage 3:

Approximately 75 ml of blood is withdrawn after adjusting the anticoagulant in the bag with 15 ml. Prp is separated by two step configuration at 1500 rpm for 15 minutes and then 2000 rpm for another 15 minutes and kept at incubator for 30 to 60 minutes.

Stage 4:

After incubation, keep the bag on platelet agitator for at least 10 minutes before issuing.

Accepted platelet rich plasma concentrate should have two times increase in platelet concentration.

The outcome study is analyzed by its pain relief and comfortable return to daily activities. Assessing the outcome is done in 2nd week. 3rd month of the study group and statistically evaluated.

Patients who do not follow the regime of treatment and who do not come for follow up are excluded and a complete description of responses to the outcome will be presented in the statistical data.

STATISTICAL ANALYSIS

This is a randomized study of patients who are the patients in RLJH and attached hospitals. This study will include 100 painful heels and will be statistically evaluated.

Inclusion Criteria:

1. Age more than 18 years.
2. Heel pain persisting for more than 4 to 6 months.

Exclusion criteria:

1. Recent trauma.
2. Any radicular pain.

3. Any previous surgeries around the foot ankle.
4. Any neurological abnormalities.
5. Pregnancy
6. Low platelet count (<1.05/uL)

Injection technique:

The procedure is explained to the patient, consent is obtained. The area to be injected is cleaned thoroughly.

22 gauge needle and 5 ml syringe is used to draw 3 to 5 ml of platelet rich concentrate and injected in to the area of maximal tenderness. A single skin poke with peppering technique is used.

Post injection protocol:

Immediately after the injection, the patient is kept in sitting position without moving the foot for 15 minutes. Patients will go to the physiotherapist to obtain stretching exercises. Patients are sent home with instructions to limit their use of the feet for approximately 48 hours and use hydrocodone or acetaminophen for pain. The use of nonsteroidal medication is prohibited. After 48 hours, patients are given a standardized stretching protocol to follow for 2 weeks. A formal strengthening program is initiated after this stretching. At 4 weeks after the procedure, patients are allowed to proceed with normal sporting or recreational activities as tolerated. Any type of foot orthoses will not be allowed.

Statistical Analysis:

Data was coded and entered into Microsoft excel data sheet and analysis was done by using SPSS 11 software. Descriptive statistics like mean, standard deviation was computed for continuous variables and frequency and proportions for categorical data. Independent t test was used to compare the mean difference between two groups, paired t test was used to compare the mean difference between before and after paired data. Correlation was done for continuous variables.

Table 1: Sex distribution among the subjects

Sex	Frequency	Percentage
Female	36	58.07
Male	26	41.93
Total	62	

In the study it was observed that 58.07% of cases were females and 41.93% were males.

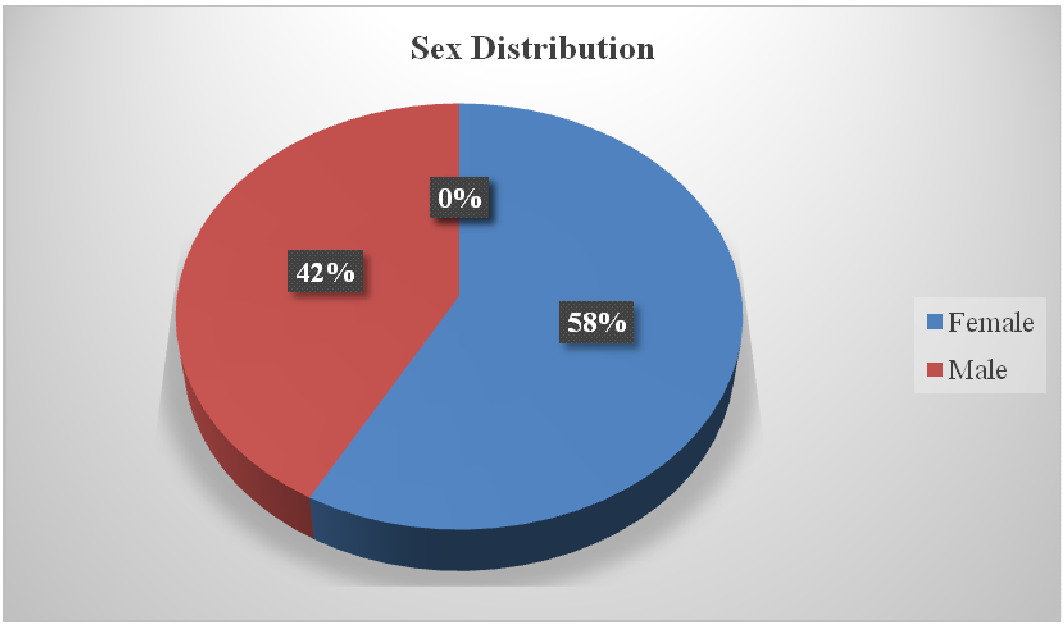


Figure 10: Pie diagram showing sex distribution of patients

Table 2: No. of sites used for platelet injection.

No of sides	Frequency	Percentage
Bilateral (both heels)	38	61.29
Unilateral (One heel)	24	38.71
Total	62	

In the study it was observed that 38 patients i.e. 61.29% both heels received prp injections and in 24 patients i.e. 38.71% only one heel was used for injection.

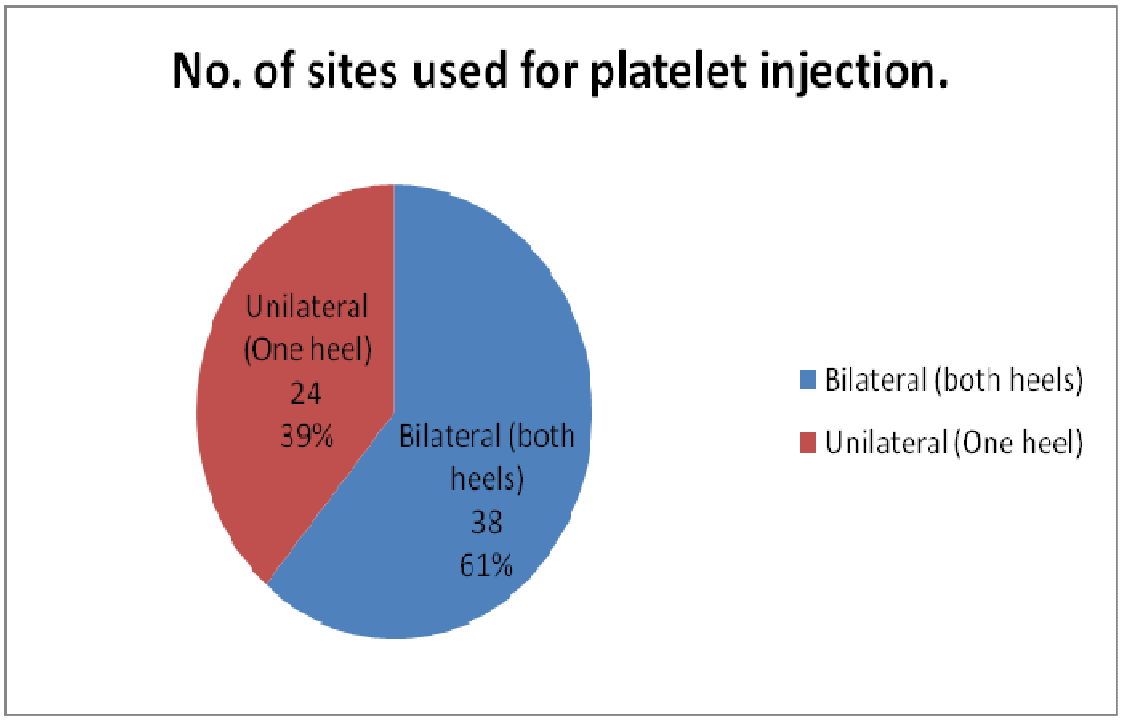


Figure 11: Pie diagram showing distribution of patients according to No of sites of injection

Table 3 : Showing Mean difference between Unilateral and Bilateral sites for continuous variables

	Unilateral (n=24)		Bilateral (n=38)		t value	p value
	Mean	**SD**	**Mean**	**SD**		
Age	42.50	13.465	43.03	12.526	-0.157	0.876
Duration of Complaints	10.21	6.953	13.68	10.928	-1.389	0.170
VAS Score Before Injection	7.58	1.018	7.39	0.855	0.785	0.435
VAS Score After Injection at 2 weeks	4.6667	1.34864	5.0132	1.29180	-1.011	0.316
VAS Score After Injection at 3 months	3.875	1.7084	4.132	1.7269	-0.572	0.569
Outcome %	52.92	20.319	48.68	18.184	0.853	0.397

In the study it was observed that the mean difference between Unilateral and Bilateral sites for Age, Duration of complaints, VAS Score before and after treatment and outcome % was not significant. Hence it can be inferred that number of sites of injection will not affect the outcome.

Table 4: Showing Site of Injection

Site	Frequency	Percent
Left	47	47.0
Right	53	53.0
Total	100	100.0

In the study it was observed that the most common site of injection was right side i.e. in 53%.

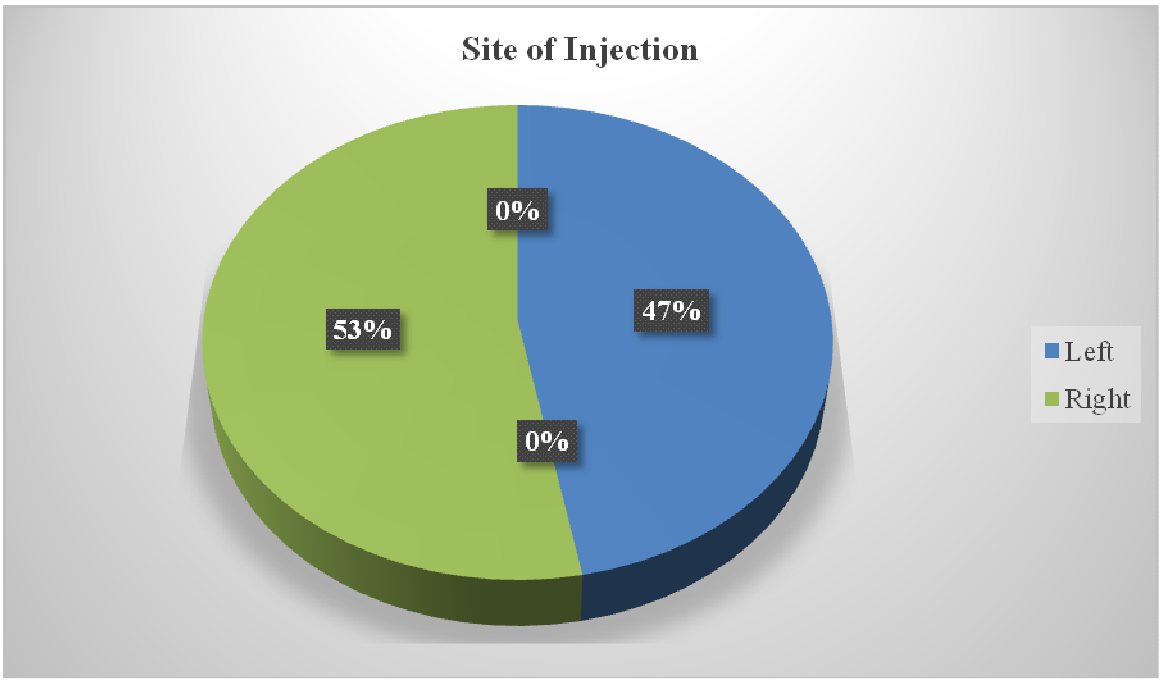


Figure 12: Pie diagram showing site of injection in study subjects

Table 5: Mean values of Age and Duration of Complaints

	Age (Years)	Duration of Complaints (Months)
Mean	43.51	12.95
Std. Deviation	12.530	10.132

In the study the Mean age of the subjects was 43.51 ± 12.5 years and mean duration of complaints is 12.95 ± 10.13 months

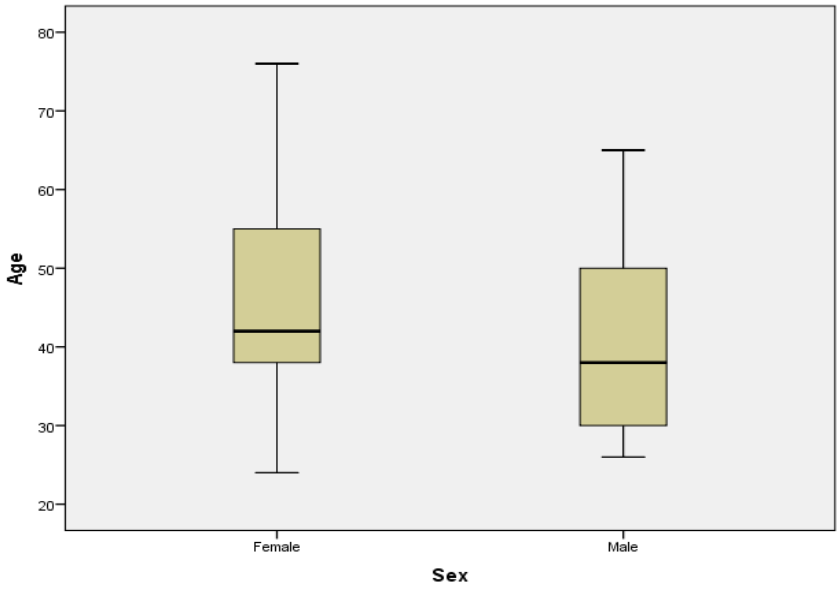
Table 6: Showing Mean difference between male and female for continuous variables

	Male (n=26)		Female (n=36)		t value	p value
	Mean	**SD**	**Mean**	**SD**		
Age in years	41.34	12.232	45.57	12.264	-1.680	0.096
Duration of Complaints in months	13.90	13.065	12.57	7.606	0.631	0.530
Outcome %	47.32	15.815	48.57	20.039	-0.332	0.741
VAS Score Before Injection	7.49	0.746	7.52	1.079	-0.153	0.878
VAS Score after Injection at 2weeks	4.89	1.1	5.03	1.50	-0.520	0.604
VAS Score After Injection at 3 months	4.256	1.4410	4.232	1.9282	0.067	0.947

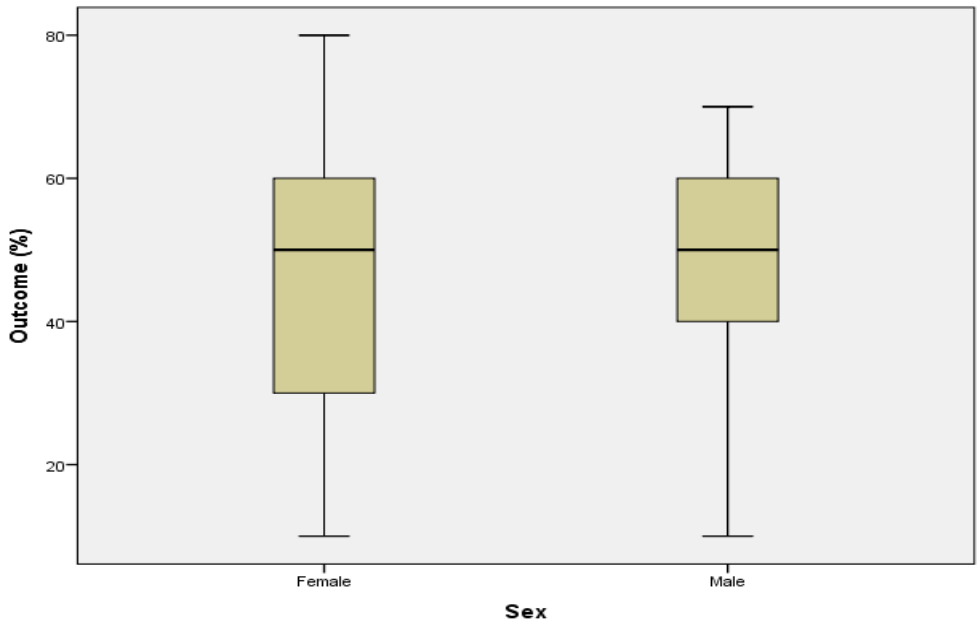
In the study it was observed that the mean difference between male and female for Age, Duration of complaints, VAS Score before and after treatment and outcome % was not significant. Hence it can be inferred that sex will not affect the outcome.

Parts of Box Plot:

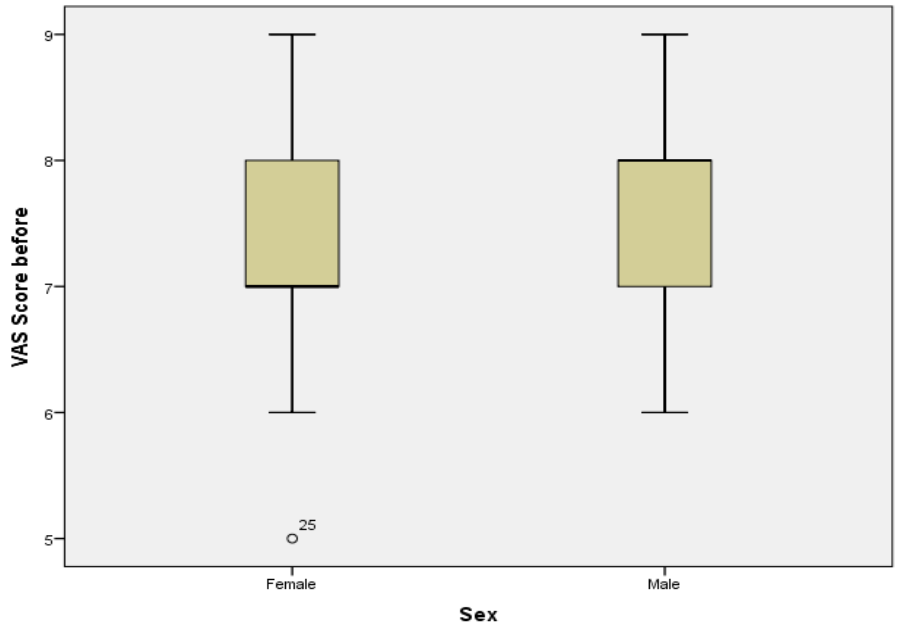
1. Centre of plot: Median
2. A box which surrounds the median with the top and bottom being the limits within which 50% of observations fall
3. Two whiskers stick out which extend to the highest and lowest extreme values



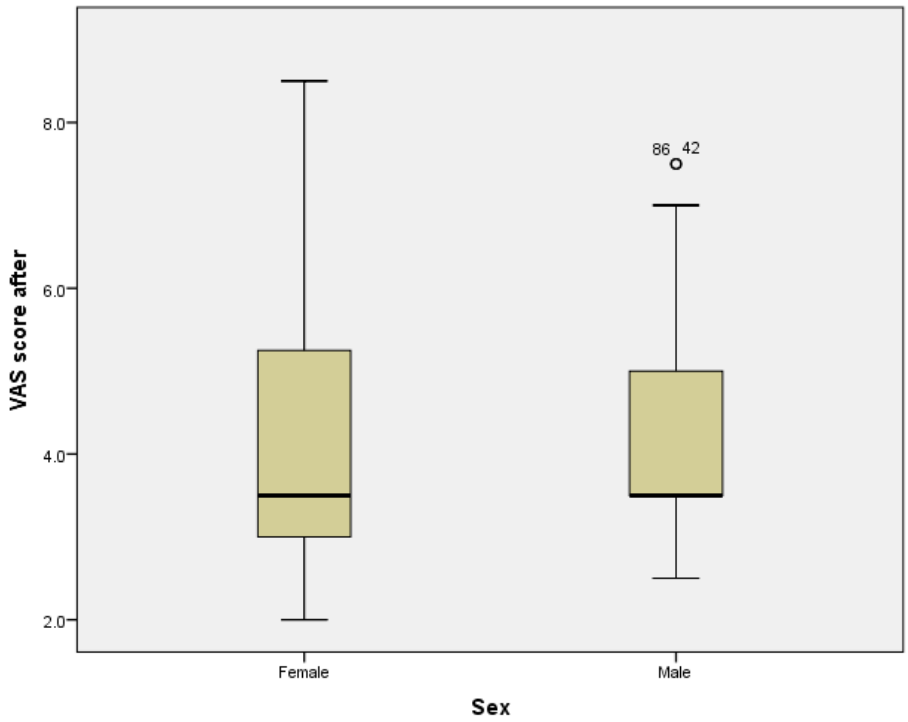
Graph1: Box plot showing Mean scores of Age for males and females



Graph 2 : Box plot showing Mean scores of outcome for males and females



Graph 3: Box plot showing Mean scores of VAS score before injection for males and females



Graph 4 : Box plot showing Mean scores of VAS Scores after injection for males and females

Table 7: Showing Mean difference between right side and left side for continuous variables

	Right (n=53)		Left (n=47)		t value	p value
	Mean	**SD**	**Mean**	**SD**		
Age	44.04	12.838	42.91	12.284	0.445	0.657
Duration of Complaints	12.60	9.753	13.34	10.635	-0.361	0.719
VAS Score Before Injection	7.36	0.879	7.62	1.012	-1.367	0.175
VAS Score After Injection at 2 weeks	4.92	1.35	5.03	1.36	-0.394	0.694
VAS Score After Injection at 3 months	4.132	1.7352	4.287	1.7344	-0.446	0.656
Outcome %	48.49	18.749	48.09	18.254	0.109	0.913

In the study it was observed that the mean difference between right and left side for Age, Duration of complaints, VAS Score before and after treatment and outcome % was not significant. Hence it can be inferred that site of injection will not affect the outcome.

Table 8: Showing Mean difference of VAS score before and after the injection at 2 weeks

	Mean	Std. Deviation	t value	df	p value
VAS Score before (n=100)	7.48	0.948	21.960	99	0.0001**
VAS score after (n=100)	4.97	1.35			

In the study it was observed that there was significant reduction in VAS score before and after the injection. VAS mean score before the injection was 7.48 ± 0.948 and after the injection was 4.97 ± 1.35. The mean VAS score before the injection was high and after injection it decreased at a very significant level at weeks

Table 9: Showing Mean difference of VAS score before and after the injection at 3 months

	Mean	Std. Deviation	t value	df	p value
VAS Score before (n=100)	7.48	0.948	22.22	99	0.0001**
VAS score after (n=100)	4.20	1.7278			

In the study it was observed that there was significant reduction in VAS score before and after the injection. VAS mean score before the injection was 7.48 ± 0.948 and after the injection was 4.20 ± 1.72. The mean VAS score before the injection was high and after injection it decreased at a very significant level at 3 months.

Table 10: Showing improvement in the outcome after injection

Outcome %	Frequency	Percent
10	5	5.0
20	10	10.0
30	10	10.0
40	12	12.0
50	18	18.0
60	31	31.0
70	8	8.0
80	6	6.0
Total	100	100.0

In the study it was observed that majority i.e. 31% had 60% improvement after the injection, followed by 50% in 18% and highest improvement of 80% in 6% of patients and least improvement of 10% was observed among 5% of subjects.

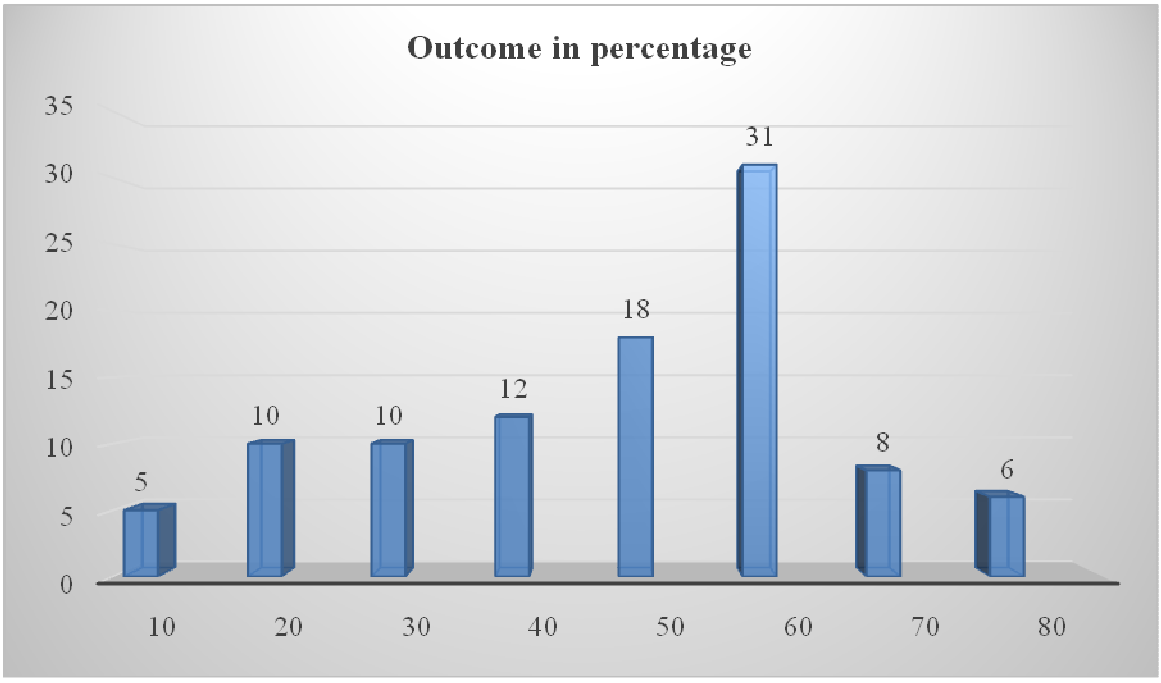
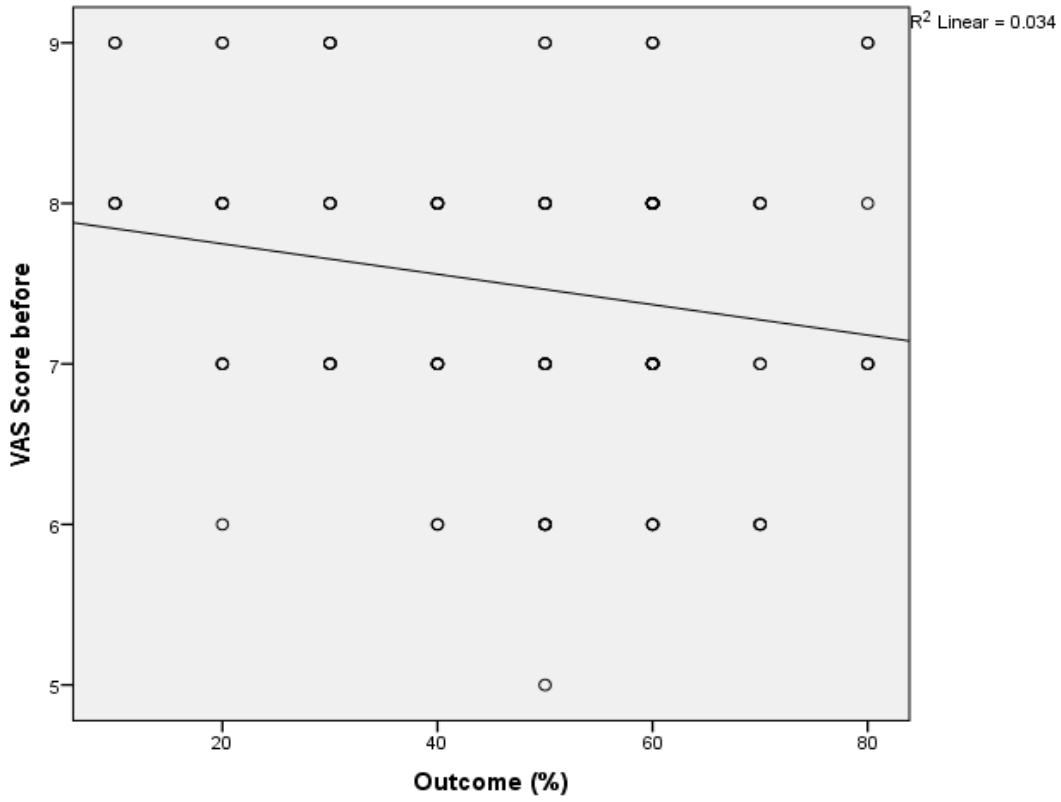


Figure 13: Bar Diagram showing improvement in the outcome after injection

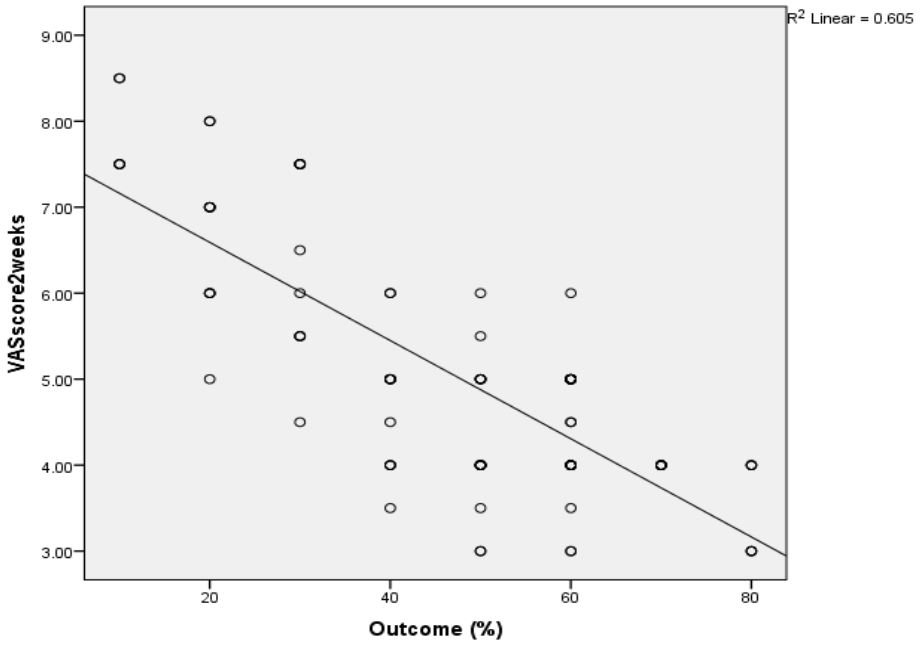
Table 11: Showing Correlation between improvement in outcome and VAS score before and after

		VAS score before	VAS score after 2 weeks	VAS score after 3months
Outcome (%)	Pearson Correlation	-0.184	-0.778**	-0.911**
	p value	0.067	0.0001	0.0001
	N	100	100	100

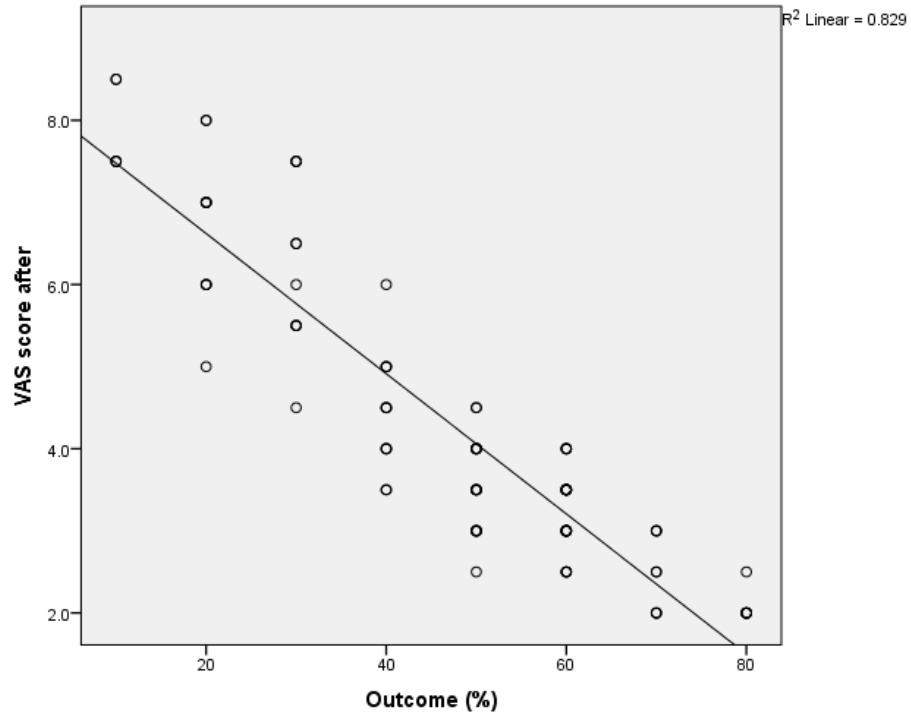
In the study it was observed that there is negative correlation between outcome and VAS score. I.e. as the outcome percentage increases there is decrease in VAS Score or as there is increase in VAS Score there is decrease in Outcome percentage. There was no significance for VAS score before the treatment and a highly significant negative correlation was observed in VAS scores after the treatment at 2 weeks and very high significance at 3 months.



Graph 5 : Scatter plot showing correlation between Outcome and VAS score before the injection



Graph 6 : Scatter plot showing correlation between Outcome and VAS score after the injection at 2 weeks



Graph 7 : Scatter plot showing correlation between Outcome and VAS score after the injection at 3 months

DISCUSSION

We designed this study to determine the outcome of platelet rich plasma injection in patients with plantar fascitis.Plantar fascitis is a very common musculo skeletal problem encountered in orthopaedic day today practice.Heel pain or plantar fascitis when acute or when chronic is quite a disabling condition. It certainly affects the day to day quality of life of patients.

Many treatment modalities have been in practice. Physiotherapy and bracing have been advised. Corticosteroid injections have been extensively used. Their efficacy is still conflicting. Corticosteroid injection can lead to local and permanent damage to structure of fascia.

With advent of biological treatments in orthopaedics platelet rich plasma has been used in many clinical problems viz., wound hemostasis/healing, augmentation of bone grafts, ACL injuries and treatment of tendinosis.

In a study by Barett et al 6 of the 9 patients achieved complete symptomatic relief after 2 months. Additional PRP injection successfully relieved the symptoms in another patient.[87]In a study by Mishra et al of 20 patients 60% showed improvement in 8 weeks, 81% at 6 months, and 93% at 1½ year of follow up.[88]In a study done by Ajit P.chitre of 8 patients 100% showed improvement in 3 months[76].

We chose to study patients with plantar fascitis as it is a very common clinical problem involving the weight bearing portion of the limb.

100 painful heels were screened and evaluated in this study, with base line characteristics of sex 36 were females, 26 were males. Among this 38 received bilateral PRP injections and 24 received unilateral PRP injections. Site of injection showed 53% right, 47% left. The mean age groups of patients were 43.51 ± 12.5 years and mean duration of complaints were 12.95 ± 10.13 months. There was considerable improvement in visual analogue score before injection with P value of 0.878 to VAS score after 3 months with P value 0.947.

The mean VAS value before injection was 7.48± 0.948 and after 3 months post injection it was 4.97±1.35.And hence significant improvement was noted.

In this study the base line characteristics like age, sex, duration of symptoms, site of symptoms does not affect the outcome of the study statistically. In this study the outcome of PRP injection is as follows.

31 % had 60% improvement in VAS score, 18% had 50% improvement in VAS score, 6%had 80% with highest improvement in VAS score, and the least improvement is 10%, was observed in 5% of subjects. These results were at the end of 3 months.Majority of the PRP

treated patients had improved quality of life status.The results of the study were comparable to other short term follow up studies in the literature.

Limitations of this study could be short follow up and platelet concentrate was variable in nature. However PRP injection/intervention as a biological modality of treatment in orthopaedic condition is encouraging.

CONCLUSION

PRP is a biological option for a common orthopaedic and recalcitrant orthopaedic problem like heel pain/plantar fascitis.

In our study a significant number of patients showed improvement in symptoms. There were no complications. Symptomatic and persistant relief enabled the patients to perform their daily activities.

PRP injection may prove to be a superior alternative to the existing treatments for chronic heel pain.

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CLINICAL PROFORMA FOR PLATELATE RICH PLASMA INJECTION FOR CHRONIC PLANTAR FASCIITIS.

Patient particulars

Name

Age

Sex

Height

Weight

Address

Occupations

CHIEF COMPLAINTS:

Anatomical location of pain

Onset of pain

Duration period of pain

Does pain increase on activity

Any morning pain

Any numbness of foot

Is patient able to bear weight

Any referred pain

PAST HISTORY:

Any co-morbidities

Any previous foot injuries

Any previous surgeries

Any treatment taken in the past

Is Patient pregnant if female

LOCAL EXAMINATION:

Any inflammation present

Sensation of foot

Movements of foot	-	Plantar flexion,
		Dorsi flexion
		Rotationary movements
Arches of foot-		Supple foot
		Cavus foot

Any deformity present

Gait

Any neurological deficit

INVESTIGATION:

General Blood investigations

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

Patients BP and GRBS status

X-ray – AP lateral and 45^0 medial oblique :-

(In standing)

Injection protocol **Date** **Pain levels reduced**

1st injection

POST INJECTION CARE:

1st 48hrs non weight bearing

NSAIDS to be avoided, pain killers like Acetaminophen a given

After 48hrs to 1week limited weight baring

Assessment for pain – 2nd week

(By visual analog scale) 3 rd month

O- No pain

10- Worst pain

EXCLUSION CRITERIA:

Any intermediate treatment taken 24-48hrs before PRP injections

Pt who don't turn up for regular follow up

Pt with side effects of PRP injections- skin infection

Local anesthetic allergy

Toxicity

Medial trauma

CONSENT OF THE PATIENT

PATIENT SIGNATURE

DOCTOR SIGNATURE

INFORMED CONSENT TRANSFUSION OF BLOOD PRODUCTS

My/the patient's doctor_____has advised me that due to my/the patient's medical condition, the chances for my/the patient's improvement or recovery will be significantly helped by receiving blood products by transfusion such as platelets.

The doctor has explained the benefits that are expected from my/the patient being transfused and, as well, the risk. I understand that although the blood products to be administered have been prepared and tested in accordance with strict scientific rules established by the American Association of Blood Banks, there is still a very small (one in a thousand) chance the blood products will be incompatible with my/the patient's body and a transfusion reaction (Hemolytic Transfusion Reaction) can occur. Although transfusion reactions can be treated successfully. I understand that on very rare occasions they can be fatal (one in two hundred fifty thousand transfusion). I understand that allergic reactions to blood products with bives, itching and fever are more common but can be treated and may not even require the transfusion to be stopped. I understand that even with testing by the most up-to-date methods, there is a small chance the blood products may contain a virus that will enter my system and may not be recognized as an infection for many months or years. Even with proper testing, my chances for contracting viral hepatitis may be approximately thirty in every one million transfusions or of contracting HIV in three of one million transfusions.

I have had an opportunity to ask questions regarding transfusion of blood products for myself or for the patient and with my signature I give consent to administering blood products for myself or for the patient. I agree this informed consent may serve for consent to give additional necessary blood products for a time certain to end with this hospitalization or for the complete course of this illness if I have been advised that the future need for transfusion of blood products is quite likely and possibly on a recurrent basis still related to this same illness.

Witness: Patient's Name:

Time: Patient's signature:

Date:

MASTER CHART

Name	IP No	Place	Sex	Age	Duration of Complaints	Occu-pation	Site	Out come (%)	VAS Score before	2wk	VAS 3mths
Parvathamma	809234	Kolar	F	55	1Yr	H.W	R	40	8	6	5
Sarojamma	851131	Kolar	F	44	8Months	Teacher	L	80	9	4	2
Sudha	850913	Kolar	F	40	6Months	H.W	L	80	9	3	2
Girijamma	850884	Kolar	F	44	6Months	H.W	R	60	8	5	3.5
Lakshmi Devamma	843063	Malur	F	55	2Yrs	H.W	R	30	8	6	6
			F				L	20	9	8	8
Nachamma	811018	Malur	F	76	1Yr	H.w	R	20	9	8	8
			F				L	30	8	7.5	6.5
Shikar Singh	797680	Banglor e	M	26	1Yr	Student	R	60	8	5	3.5
			M				L	60	8	4	3.5
Jayamma	721438	Banglor e	F	55	1-1/2Yrs	H.W	L	30	9	7.5	7.5
Syed Allah	803394	Kolar	M	34	6Months	Buisness	R	20	8	7	7
Vishnu	805034	Kolar	M	37	1Yr	Cheff	R	30	9	7.5	7.5
			M				L	40	8	5	4.5
Manjunath.B. S	797466	Chinta mani	M	28	6Months	Barber	R	60	7	4	3
Najma	764130	Kolar	F	35	6months	H.W	L	20	7	6	6
Narayanappa	752453	Kolar	M	50	1Yr	Welder	R	70	7	4	2.5
Vijaya Lakshmi	772859	Kolar	F	65	1Yr	H.W	R	80	8	3	2.5
Manjula	722841	Srinivas apura	F	38	1-1/2Yr	Agricultu rist	R	60	7	5	3
			F				L	60	8	5	3.5
Sujatha	815492	Kolar	F	58	6Months	Agricultu rist	R	60	7	4	3
			F				L	70	6	4	2
Latha Venkatesh	816034	Kolar	F	58	6Months	H.W	R	60	7	4	3
			F				L	70	8	4	3
Chenna Basappa	813121	Kolar	M	55	3Yrs	Agricultu rist	L	50	9	5.5	4.5
Subramany	811985	Kolar	M	62	5yrs	Powerloo m	R	30	7	5.5	5.5
pruthviraj	961802	kolar	M	39	2yrs	bussiness	L	40	8	5	4.5
Maheswari	824705	kolar	F	32	6Months	H.W	R	60	6	4	2.5
Lalitha	791576	Kolar	F	75	1Yr 4 Mths	H.W	R	30	7	5.5	5.5
Raghunath	659710	Mulbag al	M	45	1Yr	Agricultu rist	R	60	8	3.5	3.5
Udaya	806546	Kolar	F	40	4Months	H.W	R	60	9	5	4
Manjula	801074	Kolar	F	35	6Months	H.W	R	80	7	3	2
			F				L	50	7	4	3.5

Mangamma	805993	Kolar	F	48	1Yr	H.W	R	60	8	5	3.5
			F				L	50	9	6	4.5
Sarojamma	802055	Kolar	F	38	2Yrs	H.W	R	70	8	4	3
			F				L	60	9	5	4
Jayamma	768413	Kolar	F	38	1Yr	Attendar	R	20	7	6	6
			F				L	20	8	7	7
Venkateshappa	792335	Kolar	M	45	2Yrs	Agriculturist	R	40	7	4	4
Lakshmamma	832633	Kolar	F	58	6Months	H.W	R	10	8	7.5	7.5
Zareena Bhegam	704453	Kolar	F	40	3Yrs	H.W	R	40	7	4	4
			F				L	30	8	6.5	6.5
Amjah Khan	820173	Kolar	M	45	1Yr	Buisness	R	50	7	5	3.5
mahadevappa	740085	kolar	M	46	1yr	farmer	L	60	8	5	3.5
Venkatachlapathi	816037	kgf	M	50	1Yr	Buisness	R	30	7	5.5	5.5
			M				L	40	8	4.5	4.5
Wazira Ahmed	822388	mulbagal	M	55	1Yr	Buisness	R	40	7	5	4
			M				L	50	7	3.5	3.5
lakshamma	874488	kgf	F	60	6M0nths	H.W	R	60	9	6	4
			F				L	50	6	3	3
Sridhar Murty	864708	kolar	M	57	8Months	Agriculturist	R	60	8	5	3.5
			M				L	50	8	4	4
srinivasulu	762786	kgf	M	38	7Months	Agriculturist	R	60	7	4	3
			M				L	50	6	4	3
Sanjay	863212	Malur	M	30	1Yr	Construction	R	60	7	5	3
			M				L	20	7	6	6
Vijay Kumar	961964	Malur	M	55	6Months	Shopkeeper	R	50	8	4	4
			M				L	40	8	6	6
Shankar Reddy	961962	Kolar	M	65yrs	5Yrs	Post master	R	60	7	5	3
			M				L	60	8	4.5	3.5
Geetha	961963	Malur	F	40	2Yrs	H.W	R	70	6	4	2
			F				L	60	7	5	3
Shashikala	902784	K.G.F	F	42yrs	8Months	H.W	R	20	8	7	7
			F				L	30	9	7.5	7.5
Rajathi	902236	Chintamani	F	26Yrs	1Yr	Teacher	R	60	8	5	3.5
			F				L	50	6	4	3
Shozaj Begam	919794	Kolar	F	41Yrs	6Months	H.W	R	60	7	5	3
			F				L	50	8	5	4
Rathnamma	923004	Kolar	F	42Yrs	6Months	H.W	R	70	7	4	2.5

			F				L	50	8	5	4
Pratap	930992	Kolar	M	43Yrs	8Months	Labourer	R	60	8	4	3.5
			M				L	40	7	5	5
Papamma	920195	Devarahalli	F	51yrs	1Yr	Labourer	R	10	8	7.5	7.5
			F				L	10	9	8.5	8.5
Manjula	913857	Kolar	F	31Yrs	1Yr	Asha Worker	R	70	6	4	2
			F				L	80	7	4	2
Madhu	929161	Mulbagal	M	38Yrs	7Months	Buisness	R	40	7	5	5
			M				L	50	7	4	3.5
Lakshmi Devamma	902887	Chintamani	F	55Yrs	2Yrs	H.W	R	10	9	8.5	8.5
			F				L	20	6	5	5
Muniraju	841380	Kolar	M	32Yrs	6Months	Diner	R	20	8	7	7
			M				L	10	8	7.5	7.5
Prakash	906291	Srinivasapura	M	28Yrs	8Months	Labourer	R	50	6	4	3
			M				L	60	7	4	3
Sridevi	914273	Kolar	F	36	1Yr	H.W	R	60	6	3	2.5
			F				L	50	5	3	2.5
roja	961847	kgf	female	42	8Months	h.w	R	40	6	4	3.5
			F				L	50	7	5	3.5
mamata	625588	kolar	F	24	6mon	h.w	R	60	6	3	2.5
			F				L	50	8	5	4
muniyamma	720605	kolar	F	55	8mon	h,.w	R	30	7	4.5	4.5
			F				L	50	6	4	3
jagadish	794060	kgf	M	28	10	driver	R	60	8	4	3.5
			M				L	60	8	4.5	3.5
vijayamma	961786	kolar	M	42	8	farmer	R	40	6	3.5	3.5
Anitha	879344	bangalore	F	25	7	student	left	80	7	4	2
PRAKASH BABU	906299	KOLAR	Male	28	10m	bussiness	R	60	6	4	2.5
			M				L	70	8	4	3