# "A RANDOMIZED CONTROLLED TRAIL TO EVALUATE THE EFFICACY OF AUTOLOGOUS BLOOD INJECTION VERSUS LOCAL CORTICOSTEROID INJECTION IN TREATMENT OF PLANTAR FASCIITIS"

**By**

REG NO. BL0110001

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
ORTHOPAEDICS

**DEPARTMENT OF ORTHOPAEDICS,**
**JAWAHARLAL NEHRU MEDICAL COLLEGE,**
**BELGAUM, KARNATAKA**

**APRIL – 2013**

## KLE UNIVERSITY, BELGAUM, KARNATAKA

# ENDORSEMENT

This is to certify that the dissertation entitled **"A RANDOMIZED CONTROLLED TRAIL TO EVALUATE THE EFFICACY OF AUTOLOGOUS BLOOD INJECTION VERSUS LOCAL CORTICOSTEROID INJECTION IN TREATMENT OF PLANTAR FASCIITIS"** is a bonafide research work done by **CANDIDATE (REG NO. BL0110001)**


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# ABSTRACT

**Background and objectives**

Recently an injection of autologous blood has been reported beneficial for both intermediate/long term outcome for treatment of plantar fasciitis and there was significant decrease in pain. The present study was aimed to evaluate the efficacy and role of autologous blood injection in plantar fasciitis by comparing with the local corticosteroid injection.

## Methodology

This one year randomized controlled trial was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2011 to December 2011 on a total 120 confirmed patients of plantar fasciitis. Based on the computer generated randomization the selected patients were randomized into two groups that is, group A (n=60; received autologus blood injection+ 1 ml 0.5% bupivacaine) and group B (n=60; received methyl prednisolone acetate 80mg+1 ml 0.5% bupivacaine).

**Results**

In this study slight male preponderance was seen in both the groups (53.33% and 55%). The mean in group A was 41.80±10.96 years and in group B the mean age was 40.68±10.47 years. Most of the patients in group A and B presented with right foot involvement (51.67% and 65.00%). The mean duration in group A was 10.88 compared to 8.62 weeks in group B. The mean VAS score at the beginning were comparable in both the groups (7.55±1.40 vs 7.70±1.14;

p=0.810). At fourth week the mean VAS scores in group A significantly reduced to 3.18±2.38 and at 12 weeks and six months to 0.3±1.37 suggesting significantly less pain in group A compared to group B. Similar trend of reduction among patients in groups A was observed with Nirschal staging scores. No patient in group A reported complications and recurrence was not observed in patients with group A.

**Conclusion and interpretation**

Overall, autologus blood injection significantly reduced the pain without complications with no recurrence and provided complete relief of pain for the period of six months.

**Keywords**

Autologus blood injection; Corticosteroid injection; Plantar fasciitis.

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# INTRODUCTION

Plantar fasciitis is the most common cause of heel pain for which professional care is sought. It is an inflammation of the fascia of the plantar surface of the foot, usually at the calcaneal attachment .[1] It is the most common cause of heel pain presenting to the outpatient clinic.[2] Although thought of as an inflammatory process, plantar fasciitis is a disorder of degenerative changes in the fascia, and may be more accurately termed plantar fasciosis.[3] Plantar fasciitis is diagnosed on the basis of a history of pain on taking the first few steps in the morning, worsening pain with weight bearing, and pain and tenderness to palpation over the medial calcaneal tubercle.[2-6]

Various terms have been used to describe plantar fasciitis, including jogger's heel, tennis heel, policeman's heel, painful heel syndrome, heel spur syndrome, subcalcaneal pain, calcaneodynia, calcancal periostitis[2] and even gonorrhoeal heel. Although a misnomer, this condition is sometimes referred to as heel spurs by the general public.

The typical presentation is sharp pain localized at the anterior aspect of the calcaneus. Plantar fasciitis is often associated with a heel spur (exostosis); however, many asymptomatic individuals have bony heel spurs, whereas many patients with plantar fasciitis do not have a spur.[7]

Approximately 10% of the United States population experiences bouts of heel pain, which results in 1 million visits per year to medical professionals for treatment of plantar fasciitis.[8] the annual cost of treatments for plantar fasciitis, are estimated to be between $192 and $376 million dollars.[9]

The etiology of this condition is multifactorial, and the condition can occur traumatically; however, most cases are from overuse stresses.

Plantar fasciitis can be a difficult problem to treat, with no panacea available. No evidence strongly supports the effectiveness of any treatment for plantar fasciitis.[10, 11] Fortunately, most patients with this condition eventually have satisfactory outcomes with nonsurgical treatment.[12] For patients who do not improve after initial treatment, corticosteroid injection or dexamethasone (Decadron) iontophoresis may provide short-term benefit. However, these therapies do not improve long-term outcomes[11] and may cause plantar fascia rupture.[13]

Recently an injection of autologous blood has been reported beneficial for both intermediate / long term outcome for treatment of plantar fasciitis and there was significant decrease in pain. Platelet derived growth factor induce fibroblastic mitosis. Transforming growth factor cause fibroblast to migrate and it has been found to cause angiogenesis. A specific humoral mediator may promote the healing cascade in the treatment of tendinosis as well. This growth factor triggers stem cell recruitment, increases local vascularity and directly stimulate the production of collagen of fascia.[14]

However, so far, few studies have evaluated injection of autologous blood for plantar fasciitis. Several studies have been conducted in lateral epicondylitis as treatment modality and reported that, its application is minimally traumatic; it has a reduced risk for immune-mediated rejection, devoid of potential complications such as hypoglycaemia, skin atrophy, tendon tears associated with corticosteroid injection,

simple to acquire and prepare, easy to carry out as outpatient procedure and inexpensive.[15-16]

Hence the present study was undertaken to evaluate the efficacy and role of autologous blood injection in plantar fasciitis by comparing with the local corticosteroid injection.

# OBJECTIVE

Objectives of the present study were to evaluate the efficacy and role of autologous blood injection versus local corticosteroid injection in the management of plantar fasciitis.

Chapter 3

# Review of Literature

# REVIEW OF LITERATURE

## Plantar fasciitis

Plantar fasciitis is a common pathological condition affecting the hindfoot, and can often be a challenge for clinicians to successfully treat.[17,18] it is an overuse injury causing inflammation at the origin of the plantar fascia and surrounding perifascial structures, such as the calcaneal periosteum.[19-22]

This overuse syndrome has been recognized for almost two hundred years.[23,24] In 1812, Wood described this condition, which has been referred to by various synonyms, including plantar fasciitis, 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.[18,25,26,27] This confusion in terminology reflects the poor understanding of the etiology of the plantar fasciitis.[28]

The plantar fascia is a thick, fibrous, relatively inelastic sheet of connective tissue originating from the medial heel, where it then passes over the superficial musculature of the foot and inserts onto the base of each toe. The plantar fascia is the main stabilizer of the medial longitudinal arch of the foot against ground reactive forces, and is instrumental in reconfiguring the foot into a rigid platform before toe-off.[29-31] under normal conditions; the plantar fascia performs this function appropriately without incurring injury.

**Anatomy**

The foot and ankle can be divided into the hindfoot, midfoot, and forefoot. The hindfoot consists of four bones: the distal aspect of the tibia and fibula, the calcaneus, and the talus. The midfoot consists of five bones: the cuboid, navicular, and three cuneiforms. The forefoot consists of nineteen bones: five metatarsal bones and fourteen phalanges (Figure 3). The plantar fascia originates from the medial calcaneal tuberosity, dividing into a medial, central, and lateral band that attaches to the superior surface of the abductor hallucis, flexor digitorum brevis, and abductor digiti minimi musculature, respectively. The fascia then splits into five slips that cross the metatarsophalangeal joints and inserts onto the phalanges of digits.

The foot has a visible medial longitudinal arch (MLA) that aids in distributing the force attributed to weight bearing. The MLA of the foot resembles two rods: a posterior rod consisting of the calcaneus and talus, and an anterior rod consisting of the navicular, three cuneiforms, and the first three metatarsals. These rods are connected at their base by the plantar fascia.

When force is applied to the apex of the MLA, the arch depresses, the two rods separate, and tension is distributed throughout the plantar fascia8,21 (Figures 4 and 5). The main ligaments that aids in supporting the MLA are the long and short plantar ligaments and the calcaneonavicular ligament (spring ligament) (figure 6).

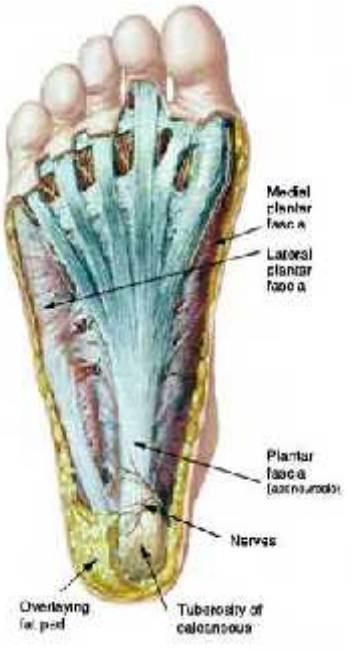



**Figure 1. Plantar Fascia**


**Figure 2. Plantar Fascia Superficial Plantar Muscles of the Foot**

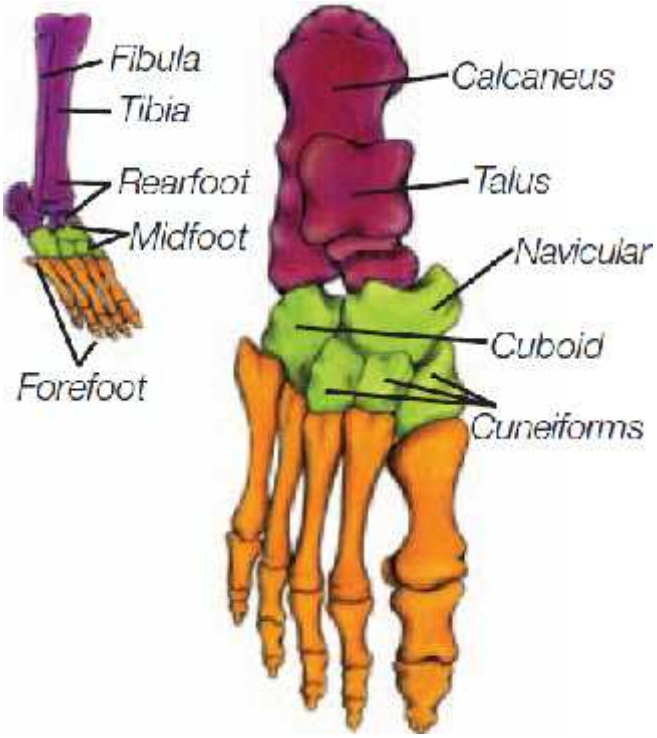



**Figure 3. Bones of the Foot and Ankle**

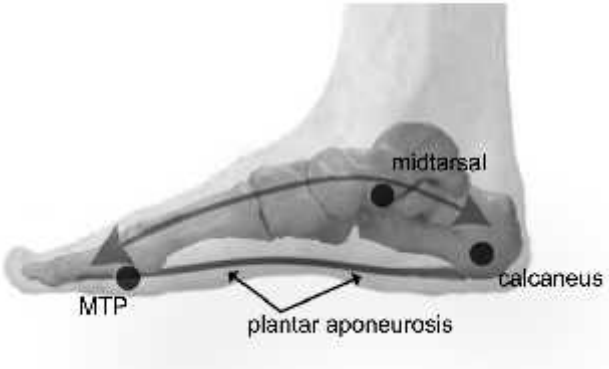



**Figure 4. Medial Longitudinal Arch. The Calcaneus and Talus represent the posterior rod; the Navicular, Cuneiforms, and the first three Metatarsals represent the anterior rod. The Plantar Fascia connects the bases of the two rods.**

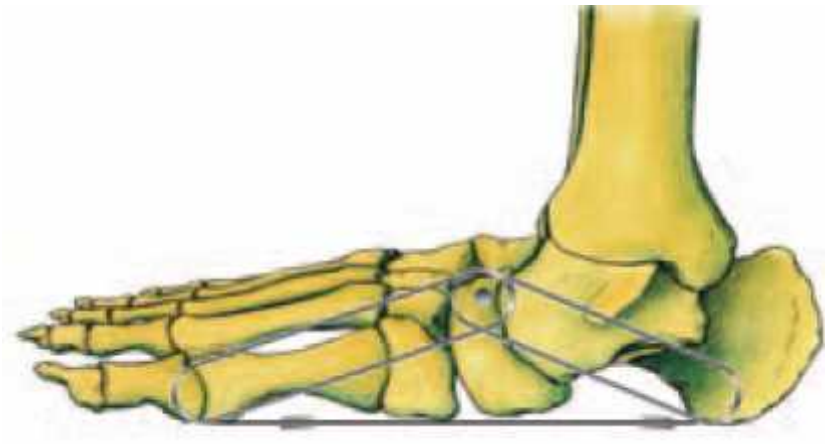


**Figure 5. Flattening of medial longitudinal arch, causing separation of the bases of anterior and posterior rods, placing an increased strain**

**Figure 6. Ligaments that aid in supporting the Medial Longitudinal Arch – Plantar View of the Foot**

The plantar fascia is an extremely strong structure composed of a thin multi-layered fibrous aponeurosis. The fascia divides into medial, central and lateral components. The central portion is the most dominant and the usual site of pathologic disorders. It originates on the plantar surface of the posteromedial calcaneal tuberosity and runs forward to form the medial longitudinal arch. Distally, five tracts are formed with superficial and deep components. The superficial portion anchors the skin, providing support from shear forces. The deep portion of the plantar fascia attaches to the plantar plates of the metatarsophalangeal joints and the bases of the proximal phalanges of the toes by connections to the flexor tendon sheaths. The medial component is the fascial covering of the abductor hallucis. The lateral component originates from the lateral margin of the medial calcaneal tubercle. It may be rudimentary or a fully developed fascial structure with distal bands to the plantar plates of the metatarsophalangeal joints of the fourth and fifth toes.[19]

The medial process of the calcaneal tubercle serves as the point of origin of the abductor hallucis, flexor digitorum brevis and abductor digiti minimi muscles.[19] The plantar fascia is innervated by the medial calcaneal nerve, a branch of the posterior tibial nerve.[34] The posterior tibial nerve bifurcates into the medial and lateral plantar nerves, which course deep to the abductor hallucis muscle. The lateral plantar nerve gives off the nerve to the abductor digiti minimi before coursing deep to the abductor hallucis muscle. The nerve to the abductor digiti minimi travels adjacent to the medial calcaneal tubercle in close proximity to the plantar fascia and the fascia of the abductor hallucis where it may be

compressed.[35] A variety of bursae are present in the foot. A subcutaneous plantar calcaneal bursa is a perifascial structure often involved with plantar fasciitis.[19]

Histologically, the extracellular matrix within the plantar fascia is comprised of collagenous and elastic fibers. The elastic fibers are present in longitudinal strands and in wavy, bundled networks. These elastic fibers may alter orientation from wavy to straight under increasing amount of acute and chronic loading, leading to stiffening of the fascia.[36]

**PATHOMECHANICS**

The function of the plantar fascia is to support the medial longitudinal arch during static and dynamic loading of the foot, and to provide midfoot stability. It also assists the heel pad in dynamic shock absorption.[37] Just after heel strike during the first half of the stance phase of the gait cycle, the tibia turns inward and the foot pronates to allow flattening of the foot. This stretches the plantar fascia. The flattening of the medial longitudinal arch allows the foot to accommodate to irregularities in the walking surface and also to absorb shock.[19]

The plantar fascia functions through the windlass mechanism to limit the flattening of the foot and to elevate and stabilize the medial longitudinal arch. This occurs when the toes are dorsiflexed, passively pulling the plantar fascia under the metatarsal heads. Thus, each time the foot passes from heel rise to toe off in the stance phase of the gait cycle, the plantar fascia is placed under increased tension.[19]

**Figure 7. Windlass mechanism**

Mechanistically, Hicks appears to be the first to describe the windlass mechanism by which passive dorsiflexion of the toes causes the medial longitudinal arch to raise, the hindfoot to supinate, the leg to externally rotate, and the plantar fascia to become more tense than when the foot and toes are in neutral. He stated that the plantar fascia acts as a cable that is wound around the metatarsal head, which acts as a drum, with the proximal phalanx acting as a handle to provide the winding.[38]

The plantar fascia is prone to repetitive injury at the posterior insertion due to its role in maintaining the medial longitudinal arch and through the stress placed on it by the shock absorbency function of the heel.[39] If there is a predisposing or aggravating factor, the repetitive traction placed on the plantar fascia during walking or running may lead to micro- and macro-tears, which induce a reparative inflammatory response.[27] The healing response is then interrupted by the continued stress produced by weight bearing, resulting in chronic degenerative changes.[27]

Histologically, these changes include collagen necrosis, angiofibroblastic hyperplasia, chondroid metaplasia and matrix calcification.[28] A single histologic study of specimens obtained from cases with inflamed plantar fascia revealed mucinoid degeneration or fibrous degeneration in 34 of 35 specimens.[18] Pathologically, prolonged inflammatory changes in the tissue are seen initially as edema, and are seen later as thickening of the plantar fascia. In one study,[37] the dorsoplantar thickness of the plantar fascia was 3 mm in normal subjects and 15 mm in patients with plantar fasciitis.

Indeed, the specific pathologic features responsible for any patient's symptoms are not well understood. However, it is suggested that the normally resilient fascia becomes stiffened and prone to reinjury, thus setting up a vicious circle of persistent pain.[20] In addition, thickening of the plantar fascia, decreased vascularity, peritendinous inflammation, and alteration of nocioceptor physiology all may play roles in the onset and persistence of the heel pain.[36]

**Epidemiology**

Incidence

Plantar fasciitis is an important public health disorder as it is the most common cause of heel pain in the outpatient setting.[40] Ten percent of people in the United States may present with heel pain over the course of their lives, with 83% of these patients being active working adults between the ages of 25 and 65 years old.[41,42] Two large national data sets of ambulatory care data (excluding visits to podiatrists or federal, military, or Veterans Administrations facilities) from the Centers for Disease Control and Prevention's National Center for Health

Statistics found that plantar fasciitis accounts for an average of one million patient visits per year to medical doctors.[42] Sixty-two percent of these visits were made to general medicine clinics, while 31% of patients were evaluated by orthopaedic or general surgeons. Additionally, a recent survey of members of the American Podiatric Medical Association revealed that plantar fasciitis/heel pain was the most prevalent condition being treated in podiatric clinics.[43] Within the current literature, prevalence rates of plantar fasciitis among a population of runners have been shown to be between 4% and 22%.[44, 45]

Ambrosius and Kondracki (1992)[46] report that PF accounts for 8.5-10% of all sports injuries. This is further supported by Evans and Fairclough, [47] Calliet, [48] and Norkin and Levangie, [49] who state that PF is common in the athletic population, tallying 7-9% of total sports injuries.[50] Although PF is common in athletes, many patients are not athletic, as noted by Gill and Kiebzak.[24] In congruence with this, Davis, *et al.* (1994)[51] reported that 30 of their 105 patients participated in athletic activity at least three times per week. Nevertheless, and despite activity level, PF tends to occur mainly in an older age group (over 40).[24, 46, 49]

Discrepancies exist between findings of studies as to gender predominance as some authors report that PF affects both males and females equally, [52] while Ambrosius and Kondracki [46] report a male predominance. In contrast to this, Gill and Kiebzak[24] and the South African studies by Blake, [53] Du Plessis, [54] Hammond and Morris[55] report a slight female predominance. Thus the literature seems inconclusive in this respect. This lack of congruence with regard to some aspects of PF may stem from the assertion by Ryan[56], who notes that the

pathophysiology of this condition is often misunderstood and predisposing conditions are frequently not recognized making it an enigmatic clinical syndrome. Furthermore he feels that a lack of understanding, of especially the biomechanics of PF, may have resulted in an inadequate treatment plan and failure to relieve the patient's pain in clinical terms, making research in this field both problematic in terms of clinical definition as well as measurement outcomes.

Age-, sex-, and race-related demographics

The exact incidence and prevalence by age of plantar fasciitis is unknown, but the condition is seen in adults essentially of all ages. A peak incidence may occur in women aged 40-60 years. An increased incidence exists in patients with certain spondyloarthropathies (ankylosing spondylitis), which often present in patients aged 20-40 years.

Women are affected by plantar fasciitis twice as often as men. In young people, the condition occurs equally in both sexes. Race and ethnicity play no role in the incidence of plantar fasciitis.

Rano et al[57] found that the average age of the patients presenting to their facility with heel pain was almost 10 years higher than controls who presented for other reasons. Matheson et al's retrospective review of 1407 patients from an outpatient sports medicine clinic, found that younger athletes had a lower prevalence of plantar fasciitis (2.5%) than older athletes (6.6%).[58] The association of plantar fasciitis with increasing age is consistent with the histopathological findings of degenerative, rather than inflammatory, changes within the plantar fascia.[59] These degenerative findings support the hypothesis that plantar fasciitis

is secondary to repetitive microtrauma caused by prolonged weightbearing activities.[60] The constant overload inhibits the normal repair process, resulting in collagen degeneration, which causes both structural changes and perifascial edema.[61,62] These changes in turn lead to a thicker heel pad, which has been shown to be associated with pain in individuals with plantar fasciitis.[63,64] Increasing heel pad thickness leads to a loss of heel pad elasticity; both of these factors are associated with increasing age and increasing BMI.[65] The decrease in elasticity of the fascia seen with increasing age is associated with a decrease in shock absorbing capabilities,[65] which may be a result of the degenerative fascia's inability to resist normal tensile loads.[66] It is this decrease in shock absorbing capability that is believed to cause the pain associated with plantar fasciitis.

The current literature is inconsistent regarding the association between sex and plantar fasciitis, with some studies showing an increased prevalence in men, [67, 68] while others show an increased prevalence in women.[57] In a retrospective case-control study of running athletes, Taunton et al found a significant sex difference within their study population, as 54% of those affected were male and 46% were female. In contrast, a prospective study including athletes of varying skill levels by Rano et al[57] found a higher percentage of women in the heel pain group than in the control group (66.1% compared with 42.6%; p=0.015). There are no theories within the current literature hypothesizing the reason for a difference in the prevalence of plantar fasciitis between the two sexes, whether it is a function of different hormones or structural differences caused by genetic variations, as is suggested by the increased incidence of anterior cruciate ligament tears in women compared with men.

Risk factors

Increased body weight[69] and increased body mass index (BMI)[6,8,9,11] have been shown to be significant risk factors for plantar fasciitis, with a BMI of more than 30 kg/m$^2$ having an odds ratio of 5.6 (95% confidence interval, 1.9 to 16.6; p < 0.01) compared with a BMI of less than 25 kg/m$^2$. Frey and Zamora[70] demonstrated a 1.4-fold increased probability of plantar fasciitis being diagnosed in an overweight or obese patient. Rome et al[71] suggested that BMI is not related to plantar fasciitis pain in the athletic population, but other factors such as a low estrogen levels in female athletes leading to a reduction in the elasticity of collagen may predispose these patients to plantar fasciitis. Riddle et al[5] hypothesized that reduced ankle dorsiflexion is the most important risk factor for the development of plantar fasciitis, as the greater the limitation in ankle dorsiflexion, the greater the amount of compensatory foot pronation and therefore the higher level of loading on the plantar fascia.

A study by Scott et al[72] found that older patients (mean age 80.2) had reduced ankle range of motion compared with younger patients (mean age 20.9). An exponential relationship between decreasing ankle dorsiflexion and the risk of developing plantar fasciitis has been found, with individuals who have 0$^o$ of dorsiflexion or less having an odds ratio of 23.3 (95% confidence interval, 4.3 to 124.4).[5] Foot pronation alone, as measured by the Foot Posture Index, [73] has also been shown to be significantly greater in patients with chronic plantar heel pain.

In addition to these intrinsic factors, various extrinsic factors have been related to the development of plantar fasciitis. Several studies have shown an

association between work-related prolonged weightbearing and plantar fasciitis.[68] In their case series, Lapidus and Guidotti's patient population included a predominance of occupations that necessitate continual standing or walking, such as waiters, maids, and kitchen workers. In addition, each heel strike during running causes compression of the heel pad up to 200% of body weight.[74] Therefore, in individuals who may not have adequate muscle strength or flexibility, and therefore have decreased shock-absorbing capabilities, the initiation of a new training program can exacerbate overloading of the plantar fascia.[74] Increases in tensile loading, seen with new increases in running intensity or frequency and changes in general footwear have been associated with overloads of the plantar fascia leading to microtears. In particular, firm footwear may exacerbate the developing plantar fasciitis in these patients.[75] Additionally, plantar fasciitis has also been associated with young individuals engaging in sports involving jumping.[76]

Etiology

Several factors may contribute to the development of plantar fasciitis. The underlying factors that have been said to precipitate the condition can be divided into anatomical, biomechanical, and environmental factors.[19] Anatomical factors include low arch or pes planus, high arch or pes cavus, sudden gain in body weight or obesity, unequal leg length, and fat pad atrophy.[19]

Biomechanical factors include tight Achilles tendon or equinus, weak plantar flexor muscles, weak intrinsic musculature, excessive subtalar joint pronation, and externally rotated lower extremity.[19]

Environmental factors include trauma, an increase in activity, unyielding surfaces, going barefoot, improper or excessively worn footwear, occupation involving prolonged weight bearing, and inadequate stretching.[20] In most cases, a combination of these factors leads to the development of plantar fasciitis.[17]

Many authors have noted that specific anatomic foot configurations are associated with the development of plantar fasciitis.[19] Pes planus with excessive pronation is the most common mechanical cause of structural strain on the plantar fascia resulting in plantar fasciitis.[19] Between 81 and 86% of individuals with symptoms consistent with plantar fasciitis have been classified on examination as having pes planus with excessive pronation.[17] The theoretical basis for this finding is the increased tension placed on the plantar fascia as a result of a lower arch during standing and walking. In addition, increased pronation results in decreased stability of the hindfoot, which produces additional stress on the origin of the central band of the plantar fascia and may ultimately lead to plantar fasciitis.[17]

Excessive pronation results in an inability of the foot to supinate from mid to terminal stance.[19] Consequently, little load is conveyed through the lateral portion of the midfoot and normal loading forces are inadequately supported by the bones and ligaments. The vertical impulse is thus shifted away from the midfoot, and secondary structures, such as the plantar fascia, must assume a greater load.[22] Mann and Inman confirmed this by noting that heel pronation increased the tension along the medial aspect of the heel.

It has been reported that most cases of plantar fasciitis are the result of different factors that cause abnormal pronation. These include leg length discrepancy, ankle equinus, excessive tibial torsion, worn shoes, loose heel counters, inadequate arch support, and tight shoebox construction.[19] However, research studies have not demonstrated that foot pronation is a primary factor in the cause of plantar fasciitis.[17]

The cavus foot is also commonly associated with the occurrence of plantar fasciitis.[22] It has been suggested that the intrinsically tight plantar fascia develops fasciitis secondary to its inability to dissipate force during stance phase.[19] The result is similar to the stretching of a bowstring with increased tension generated within the fascia.[19] Notably, a cavus foot by itself, without concurrent fasciitis, has been shown to load the midfoot to a lesser extent, and the forefoot to a greater extent than in the normal foot. The shifting of the vertical impulse to the forefoot and, more particularly, away from the midfoot is certainly consistent with the theory of intrinsically tight fascia.[22] While some authors have noted an association between pes cavus and plantar fasciitis, another study of 323 patients (364 feet) with plantar fasciitis could find no causal relationship.[68]

A tight Achilles tendon is found in 78% of patients with plantar fasciitis.[20] It limits ankle joint dorsiflexion, which increases the load on the intrinsic muscles of the foot and results in abnormal compensatory pronation of the subtalar joint as ankle dorsiflexion progresses during the stance phase of gait.[19]

The externally rotated lower extremity resulting from excessive femoral or tibial torsion is another significant pathomechanical factor for plantar fasciitis.

The stance foot is not capable of supination from mid to terminal stance, and instead pronation occurs, because the medial portion of the midfoot assumes a greater load.[19]

Obesity occurs in 40% of men and 90% of women with plantar fasciitis, compared to 20% of both men and women without plantar fasciitis.[28] Hill and Cutting found a statistically significant correlation between plantar fasciitis and increased body weight, and concluded that increased body weight is an associated factor in many patients with plantar fasciitis. This finding is consistent with other studies reporting a strong correlation between obesity and the incidence and severity of plantar fasciitis.[77]

Overuse, rather than anatomy, is the most common cause of plantar fasciitis in athletes. A history of an increase in weight bearing activities is common, especially those involving running, which causes micro-trauma to the plantar fascia and exceeds the body's capacity to recover. One study found a significant correlation between activity level and plantar fasciitis. Specifically, the plantar fasciitis group was more active than the control group.[24]

Most patients with plantar fasciitis work on hard floors. Indeed, there is an association between plantar fasciitis and the type of floor on which individuals work.[24] Other associations have been proposed, such as occupations involving prolonged weight bearing, wearing shoes with poor cushioning or inadequate arch support, and walking barefoot. With the exception of prolonged weight bearing, these associations have not been substantiated.[37]

Prognosis

About 80% of plantar fasciitis cases resolve spontaneously by 12 months; 5% of patients end up undergoing surgery for plantar fascia release because all conservative measures have failed.

For athletes in particular, the slow resolution of plantar fasciitis can be a highly frustrating problem. These individuals should be cautioned not to expect overnight resolution, especially if they have more chronic pain or if they continue their activities.[78] Generally, the pain resolves with conservative treatment.[78-79]

Although no mortality is associated with this condition, significant morbidity may occur. Patients may experience progressive plantar pain, leading to limping (antalgic gait) and restriction of activities such as walking and running. In addition, changes in weight-bearing patterns resulting from the foot pain may lead to associated secondary injury to the hip and knee joints.

**Presentation**

History

The sine qua non of plantar fasciitis is a history of intense sharp heel pain with the first couple of steps in the morning or after other long periods without weight-bearing.[80] Pain is experienced primarily on the plantar surface of the foot at the anterior medial aspect of the calcaneus, but it may radiate proximally in more severe cases. A limp may be present, and patients may prefer to walk on their toes. Associated paresthesias, nocturnal pain, or systemic symptoms should

raise suspicion of other causes of heel pain (neoplastic, infectious, neurologic causes).

Initially, the pain decreases with ambulation or athletic warm up, but then increases throughout the day as activity increases. In more severe cases, patients complain of heel pain after periods of prolonged sitting. A dull ache may be felt in the heel at the end of the day, especially after extensive walking or standing. In addition to pain, patients may complain of stiffness in the foot and localized swelling in the heel.

An important element in the history is the period preceding the start of plantar fasciitis. Patients may report that before the onset of pain, they had increased the amount or intensity of activity including, but not limited to, running or walking. They may have also started exercising on a different type of surface or may have recently changed footwear (started a barefoot style running program). They may have sustained previous trauma to the foot (falls, motor vehicle accidents, work-related injuries).

Any precipitating factors should be identified if possible. Ask the patient what makes the pain worse and what makes it better.

- Most patients report that the pain usually is most severe during the first few steps after prolonged inactivity, such as sleeping or sitting.
- Patients may report that symptoms typically are relieved by unloading the affected foot (via sitting, elevation, or other means).
- Pain may be worsened by walking barefoot on hard surfaces or by walking up stairs.

- In athletes, the pain may be particularly aggravated by sprinting.
- Patients who are generally on their feet all day report that the symptoms may actually worsen by the end of the day.

Physical findings

The pain of plantar fasciitis can usually be reproduced by palpating the plantar antero-medial calcaneal tubercle at the site of plantar fascial insertion to the calcaneum.[81] Less frequently, the pain will localize directly below the calcaneum or even in the midsubstance of the plantar arch. In more severe cases, pain may be reproduced by palpation over the proximal portion of the plantar fascia.

A tight Achilles tendon (as in talipes equinus) is commonly a secondary finding and usually contributes to the pathology; [81, 82] ankle dorsiflexion may be limited as a result. Other findings may include various deformities, skin changes, and flat-foot or pes planus foot type, overpronation, pes cavus or high-arched foot type, leg-length discrepancy, excessive lateral tibial torsion, and excessive femoral anteversion.

Other maneuvers that may reproduce the pain of plantar fasciitis include passive dorsiflexion of the toes, which is sometimes called the windlass test, and having the patient stand on the tiptoes and toe-walk. In a study by De Garceau et al, having the patient bear weight during the windlass test increased the sensitivity of the test from 13.6% to 31.8%.[83]

**Diagnosis**

Even in this age of modern technology, the diagnosis of plantar fasciitis is based mainly on the patient history and physical examination.[21] A detailed history will often provide enough information to make the diagnosis of plantar fasciitis, and physical examination will confirm it. A complete description of the pain is essential.[20] Further investigations, such as radiographs, electrophysiological studies, and blood tests, are used only to rule out other disorders that cause inferior heel pain.

The most common symptom associated with plantar fasciitis is pain and discomfort in the inferior heel region, which is aggravated on weight bearing after a period of non-weight bearing.[17] Patients will often note that they have excruciating pain when arising from bed in the morning. This is typical of plantar fasciitis because the foot tends to remain in an equinus position during the night and the fascial tissues contract. In the morning, putting weight on the foot puts the plantar fascia under tension, aggravating the pain. The pain may become so incapacitating that the patient limps to the bathroom or hobbles around with the heel off the ground. However, the acute discomfort will slowly subside during the next 30 to 45 minutes.[17] If the patient has a long commute to work, patient can also report that heel was not painful during the commute but that the pain commenced immediately as he/she attempted to weight bear again on the involved extremity.[17]

The duration of activity before the onset of heel pain can serve as an excellent indicator of the degree of irritability of the involved tissues.[17] In

general, the pain is brought on by weight bearing activities, such as standing, walking, jogging, or running, and relieved with rest.[17]

The source of pain is believed to be inflammation of the plantar fascia that results from excessive tension.[19] In its acute stage, the discomfort most often is localized to the origin of the medial and central bands of the plantar fascia at the medial tubercle of the calcaneus and is characterized as a sharp or knife-like intermittent pain. However, patients who present with chronic complaints indicate that the pain may become dull or achy and constant, and the discomfort may progress distally along the entire course of the central band in the region of the medial longitudinal arch.[17]

The pain is usually insidious.[20] It is not unusual for a patient to endure the symptoms and try to relive them with home remedies for many years before seeking medical treatment. Acute trauma is not common; however, further questioning may indicate a recent increase in either the amount or intensity of physical activity or a change of shoe wear before the onset of the symptoms.[17]

The condition is usually not completely disabling; however, patients frequently report limitations in their routine daily activities.[77] Using the Physical Activity sub-scales of the Health Status Questionnaire Short Form 36, a recent study showed that, on average, physical activity of patients with plantar fasciitis was inferior to that of patients with diabetes and equivalent to that of patients with acute sciatica.[84]

Physical examination of patients with plantar fasciitis most often yields few objective findings.[84] Careful palpation is required in the physical examination

to determine the exact location of the patient's discomfort and to ensure a correct diagnosis of plantar fasciitis.[17] On deep palpation, the patient usually has localized tenderness at the anteromedial aspect of the heel with no significant pain on compression of the calcaneus from a medial to a lateral direction; firm finger pressure is often necessary to localize the point of maximum tenderness.[20] The patient may also have tenderness along the entire plantar fascia. Passive dorsiflexion of the toes or ankle stretches the fascia, reproducing the pain of weight bearing, and facilitates palpation of the plantar fascia.[20] The pain may also be exacerbated by having the patient stand on the tips of the toes.[80] Tightness of the Achilles tendon, as noted by limited ankle dorsiflexion with the knee in extension, is usually found in patients with this condition.[20] Although localized swelling is usually absent, nodules or thickening of the plantar fascia may be noted when the condition is chronic.[19]

The clinical diagnosis of plantar fasciitis is relatively easy; however, when patients present with atypical or chronic symptoms, differential diagnostic testing may provide useful information.[20] In a recent study, both ultrasonography and bone scintigraphy confirmed the clinical diagnosis in a total of 25 of 27 heels, highlighting the accuracy of clinical diagnosis. This suggests that clinical examination is sufficient to establish the initial diagnosis of plantar fasciitis and that the diagnostic role of ultrasonography and scintigraphy should be limited to the evaluation of persistent heel pain in order to rule out rare, alternative pathologies.[85]

Ultrasonography and bone scintigraphy are equally effective in the diagnosis of plantar fasciitis.[85] Ultrasound examination may show increased

thickness of the plantar fascia and appearance of inflammatory changes.[22] On the other hand, bone scintigraphy confirms plantar fasciitis by uptake at the origin of the fascia.[20] MRI is rarely indicated but may show thickening and inflammation of the medial bundle of the plantar fascia. Radiographically, a heel spur on the inferior surface of the calcaneus frequently is evident but is not considered pathognomonic of the disorder. In addition, standard weight bearing radiographs demonstrate the biomechanical character of the hindfoot and forefoot; however, they usually serve only as an aid to confirm the clinical diagnosis.[86]

**Treatment**

Plantar fasciitis can be a difficult problem to treat, with no panacea available. No evidence strongly supports the effectiveness of any treatment for plantar fasciitis.[68] Fortunately, most patients with this condition eventually have satisfactory outcomes with nonsurgical treatment.[27] However, patients have differing degrees of pathology and varying types of body habitus and lifestyle and will therefore respond differently to various treatments. Even with individualized care, some patients respond quickly, and others exhaust all conservative measures before relief is achieved.

General measures

Treatment protocols in most studies include the use of ice and nonsteroidal anti-inflammatory drugs (NSAIDs). However, no studies have specifically examined the effectiveness of these treatments alone.

Taping

No studies have adequately evaluated the effectiveness of taping or strapping for managing plantar fasciitis.

Shoe inserts

Many types of shoe inserts have been used to manage plantar fasciitis. One randomized controlled trial[87] showed that magnet-embedded insoles were no more effective than placebo insoles in alleviating pain.

Another study[88] that compared custom orthotics and prefabricated shoe inserts (silicone heel pad, felt pad, rubber heel cup) combined with stretching found that the use of prefabricated insoles plus stretching was significantly more effective than custom orthotics plus stretching. Only five patients would need to be treated with prefabricated insoles to benefit one.

Night splints

Posterior-tension night splints maintain ankle dorsiflexion and toe extension, creating a constant mild stretch of the plantar fascia that allows it to heal at a functional length. Physicians can make custom splints in the office[89] or purchase prefabricated splints. One Cochrane review[90] found limited evidence to support the use of night splints to treat patients with pain lasting more than six months. Patients treated with custom-made night splints improved, but patients treated with prefabricated night splints did not.[90]

Stretching

Stretching protocols often focus on the calf muscles and Achilles tendon or on the plantar fascia itself.[28] In a prospective RCT[28] that compared these two approaches, researchers found that patients who stretched the plantar fascia showed a greater decrease in "pain at its worst" and a decrease in pain with first steps in the morning. Both groups, however, experienced an overall decrease in pain. The benefits of stretching both the plantar fascia and the Achilles tendon are unknown.

Corticosteroid injections

Limited evidence supports the use of corticosteroid injections to manage plantar fasciitis. Results of a Cochrane review[90] showed that corticosteroid injections improved plantar fasciitis symptoms at one month but not at six months when compared with control groups. The same review showed that steroid iontophoresis also improved short-term outcomes. However, physicians should be cautious about administering this treatment, because corticosteroid injection is associated with plantar fascia rupture, which may cause long-term discomfort.[91]

Extracorporeal shock wave therapy

Recent systematic reviews[90, 92] have evaluated RCTs that studied the effectiveness of extra-corporeal shock wave therapy (ESWT) in the management of heel pain. In general, the reviewers found that the quality of the studies was poor and that no conclusive evidence supported the effectiveness of ESWT in

reducing night pain, resting pain, and pressure pain in the short term (i.e., within six and 12 weeks).

Since the release of these systematic reviews, three groups have published RCTs[93, 94] that studied ESWT. Two well-designed RCTs[93, 94] compared ESWT with a placebo procedure in patients with chronic plantar fasciitis. Neither study found a significant difference between the treatment and control groups three months after treatment. One RCT[95] included 45 runners who had chronic heel pain for more than 12 months. According to the study, three weekly treatments of ESWT significantly reduced morning pain in the treatment group at six and 12 months when compared with the control group.

Casting

In one case series, [23] investigators studied 32 patients with chronic heel pain who had not responded to multiple treatments. For six months, the patients wore well-padded fiberglass walking casts with the ankle in neutral to slight dorsiflexion and the toe plate in extension. At long-term follow-up, 25 percent of patients had complete resolution of pain, and an additional 61 percent had some improvement. However, case series and other uncontrolled studies typically overestimate the benefits of treatment.

Surgery

No RCTs have evaluated the effectiveness of surgery in the management of plantar fasciitis. Five retrospective case series, [23, 96-99] which included 278 patients who had experienced pain for an average of 14 months before surgery,

showed that 75 to 95 percent of patients had long-term improvement as measured by various criteria. Up to 27 percent of patients still had significant pain, up to 20 percent had some activity restriction, and up to 12 percent had moderate pain that impaired function. The recovery time ranged from four to eight months. No studies have directly compared open procedures with endoscopic procedures.

**Literature review comparing autologous blood injection versus local corticosteroid injection in treatment of plantar fasciitis**

When conservative treatment results in a non-satisfactory outcome, the patient is often interested in treatment options other than surgery.

Corticosteroids

Corticosteroids can be administered either orally or via injections. Oral preparations, such as a methylprednisolone dose pack, are distributed systemically and can be used in the acute phase in conjunction with, or in place of, NSAIDS. Corticosteroid injections, on the other hand, involve local, concentrated administration and are generally reserved as a tertiary level of treatment after failure of other primary conservative measures (eg, stretching, shoe inserts, or orthoses) in severe recalcitrant cases.[100] Whether or not injected corticosteroids alter the long-term pathology of chronic inflammation, many patients experience acute symptomatic improvement.[101]

Before steroids are injected, potential causes of heel pain other than plantar fasciitis should also be considered, and a plain radiograph of the foot or calcaneus should always be obtained.

A corticosteroid injection may be given through a plantar or a medial approach, with or without ultrasound guidance, typically in conjunction with a local anesthetic. The basic technique may be briefly summarized as follows:

- Use a 22-gauge, 1.5-in. (3.8-cm) needle containing a mixture of 1 mL of local anesthetic (eg, lidocaine) and 2mL (80 mg) of corticosteroid (eg, methylprednisolone)
- Palpate the most anterior aspect of the medial plantar calcaneal tubercle, and insert the needle at this site
- Advance the needle until it reaches the most anterior (distal) aspect of the plantar medial calcaneal tuberosity
- When the proximal (anterior) edge of the calcaneum has been identified, advance the needle immediately anterior to this spot
- Avoid injecting within the superficial layers of the subcutaneous tissue, because corticosteroid injection into the superficial fat pad can cause fat necrosis and atrophy, which reduce the shock-absorbing capacity of the plantar heel.

Studies have reported success rates of 70% or better.[102] Corticosteroid injections have been shown to improve symptoms at 1 month but not at 6 months. It is recommended not to give more than 3 steroid injections within a year.

A randomized, controlled study demonstrated that intralesional corticosteroid injection is more efficacious and more cost-effective than low-

energy ESWT in the treatment of plantar fasciitis that has persisted for more than 6 weeks.[103]

In a preliminary report, a posterior tibial nerve block prior to steroid injection was shown to decrease the pain from injection and to improve compliance with treatment, without any complications.[104]

Trials of ultrasound-guided steroid injection have shown its potential efficacy. This approach has been shown to produce a good clinical response when palpation-guided injection is unsuccessful.[101] Accurate injection under ultrasonographic guidance may also minimize adverse effects from the injection.[105]

A study of 25 patients who received corticosteroid injections for plantar fasciitis showed that patients received symptomatic relief as measured by tenderness threshold and a visual analog scale (VAS).[105] Although this benefit was obtained whether the injection was performed with imaging (ultrasound) guidance or with palpation alone, patients receiving image-guided injections had a lower rate of recurrence of heel pain. Thus, although injection is helpful with or without imaging guidance, the use of imaging may provide additional benefit.

The general risks involved with the use of corticosteroids include skin atrophy, skin hypopigmentation, soft-tissue atrophy, infection, bleeding, and failure to work. A steroid flareup, which consists of increased pain for up to several days, may occur in up to 2% of individuals who use corticosteroids.[106]

Potential risks of corticosteroid injection include plantar fascia rupture, which was found in almost 10% of patients after plantar fascia injection in one case series, and fat pad atrophy.[107, 108] Long-term sequelae were found in approximately 50% of patients with plantar fascia rupture.[107]

Improper placement of a corticosteroid injection for plantar fasciitis can result in necrosis and atrophy of the plantar fat pad at the heel. This complication may result in significant pain and a decreased activity level for the patient. Bleeding or bruising generally is expected only in patients who have bleeding disorders or are taking anticoagulants. Infection at the injection site is rare, but possible. In addition to the sterile technique for the procedure itself, patients need to maintain good foot hygiene after the injection. Allergic reactions to the injected medications are rare, but possible.

Intravascular injection could potentially cause cardiac dysfunction as a consequence of the inherent toxicity of local anesthetic agents. Peripheral nerve dysfunction is possible if the local anesthetic is injected either close to or within the medial plantar nerve or the calcaneal branch of the tibial nerve.

In diabetic patients, transient elevation of blood glucose levels may occur after corticosteroid injection. Corticosteroid injection can be performed during pregnancy, although safety for use during pregnancy has not been established. With pediatric patients, obtain informed consent from the parent or legal guardian before proceeding with examination or any injection.

Patients should be informed that the symptomatic improvement from the corticosteroid usually does not begin to take effect until a few days after the

injection. They may experience a transient, mild increase in symptoms when the effect of the short-term local anesthetic has ended, but the long-term corticosteroid effect has not yet begun.

Steroid injections are a popular method of treating the condition but only seem to be useful in the short term and only to a small degree.[109]

Treatment with corticosteroids has a high frequency of relapse and recurrence, probably because intra fascial injection may lead to permanent adverse changes within the structure of the fascia and because patients tend to overuse the foot after injection as a result of direct pain relief.[109]

The use of corticosteroids is particularly troubling as several studies have linked plantar fascia rupture to repeated local injections of a corticosteroid.[109] When neither rest and neither activity restriction nor conservative treatments result in a satisfactory outcome, the patient is often interested in treatment options other than surgery.

In an animal model the addition of growth factors to the ruptured tendon has been shown to increase the healing of the tendon.[109] In humans it has been shown that the injection of whole blood into the tendon decreases the pain.[109]

Autologous blood and plasma

There is insufficient evidence in the published peer-reviewed medical literature to support the use of autologous blood injection for the treatment of plantar fasciitis.

There is some evidence to suggest that platelet-rich plasma may be beneficial in the treatment of chronic plantar fasciitis. Further research is under way to elucidate how PRP injections compare with corticosteroid injections in this setting.[110] Although both autologous blood and PRP injections appear to cause resolution of the symptoms of plantar fasciitis, these studies have shown results that are not significantly different when compared corticosteroid injections.

PRP is promoted as an ideal autologous biological blood-derived product, which can be exogenously applied to various tissues where it releases high concentrations of platelet derived growth factors that enhance wound healing, bone healing and also tendon healing. In addition PRP possesses antimicrobial properties that may contribute to the prevention of infections.[109] When platelets become activated, growth factors are released and initiate the body's natural healing response.

Injection of autologous blood into the plantar fascia origin is thought to stimulate an acute inflammatory reaction, providing factors that stimulate fibroblast activity and vascular growth and there by lead to reinitiation of the healing process. This treatment has been shown to be effective in limited studies of chronic inflammatory musculotendinous conditions.[111]

In a recent study of Peerbooms et al.[112] a positive effect of injection of PRP in the common extension origin for lateral epicondylitis was seen. This report describes the first comparison of an autologous platelet concentrate with corticosteroid injection as a treatment for lateral epicondylitis in patients who

have failed non-operative treatment. It demonstrates that a single injection of concentrated autologous platelets improves pain and function more than corticosteroid injection. These improvements were sustained over time with no reported complications.

The injection of platelet-rich-plasma (PRP) into the effected tissue addresses the healing stages necessary to reverse the degenerative process which are going on in the base of the plantar fascia. The individual cytokines present in the platelet α-granules have been shown to enhance fibroblast migration and proliferation, up-regulate vascularization, and increases collagen deposition in a variety of in vitro and in vivo settings. The cytokines present in platelet α-granules have been shown to affect the healing stages necessary to reverse a chronic plantar fasciitis condition. Additionally, many of these cytokines have been seen to work in a dose dependent manner.[109]

Lee and Ahmad (2007)[113] conducted a prospective, randomized, controlled, observer-blinded study (n=64) to compare the efficacy of intralesional autologous blood with corticosteroid injection for plantar fasciitis. Data were complete for 61 patients, 30 patients in the autologous blood group and 31 patients in the corticosteroid group. Over the six-month follow-up period, a significant reduction in pain levels was noted in both groups ($p<0.0001$). At six months after treatment, patients who had received the corticosteroid injection had lower average levels of pain than those who had received the autologous blood injection, but the difference was not significant ($p= 0.094$). Acknowledged limitations of this study include its short-term follow-up and the lack of a control group that would show the natural history of the disease without intervention.

Kiter et al. (2006)[114] evaluated the efficacy of autologous platelet injection for plantar fasciitis in an RCT (n=45). The 45 patients were treated for heel pain using either the peppering technique (n=15), autologous blood injection (n=15) or corticosteroid injection (n=15). In the peppering technique group, after infiltration of one milliliter (ml) of 2% prilocaine, the needle was inserted, withdrawn and redirected 10–15 times without emerging from the skin. At six-month follow-up, clinical improvement was evaluated using a VAS. Improvements in VAS scores were reported to be 68%, 68% and 65% for the peppering technique, autologous blood injection and corticosteroid injection groups, respectively. Larger, well-designed RCTs are needed to further define the role of autologous blood injection in the treatment for plantar fasciitis.

Chapter 4

# Methodology

# METHODOLOGY

The present study was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2011 to December 2011.

**Study design**

The study design one year randomized controlled trial.

**Study period**

Present study was conducted from January 2011 to December 2011.

**Place**

This study was carried out at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum attached to Jawaharlal Nehru Medical College, KLE University, and Belgaum

**Source of Data**

Confirmed patients of plantar fasciitis attending KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

**Sample Size**

A total sample size of 60 cases divided into two groups.

**Sample size calculation**

Based on the prevalence available from the literature, using a desired power of 0.8 and a type I error of 0.05 the sample size was derived from the formula as mentioned below.

$$n = \frac{2(Z_\alpha + Z_\beta)^2 \text{ x Standard Deviation (S.D.)}^2}{(X_1-X_2)^2}$$

Where,

$Z_\alpha$ = 1.65 for $\alpha$=0.05

$Z_\beta$ = 0.84 for $\beta$=0.2

S.D. = 24

$X_1$ = 30

$X_2$ = 15

Using this formula the sample size was determined as 30 in each group. However during the study period more than 120 patients presented with plantar fasciitis and the sample size was determined as 60 in each group.

**Selection Criteria**

Inclusion criteria

- Clinically confirmed cases of plantar fasciitis with a pain in the antero-medial border of calcaneum
- Either sex
- Age above 15 years

Exclusion criteria

- Patients receiving steroid injections within three months before blood injection.
- A history of substantial trauma.
- Previously treated by surgery for plantar fasciitis.
- Other causes of heel pain such as;
    - Calcaneal stress fracture
    - Retrocalcaneal bursitis
    - Peroneal ,posterior tibial ,Flexor hallucis longus tendonitis
    - Tarsal tunnel syndrome
    - Lumbar radiculopathy
- Other causes like
    - Rheumatoid arthritis
    - Ankylosing spondylitis
    - Reiters syndrome
    - Osteoarthritis

**Randomization**

Based on the computer generated randomization the selected patients were randomized into two groups namely;

- Group A (n=60) – Autologus blood injection group.
- Group B (n=60) – Corticosteroid injection group.

**Ethical clearance**

The study was approved by the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belgaum.

**Informed Consent**

Patients fulfilling the selection criteria were briefed about the nature of the study and a written informed consent was obtained from the selected patients (Annexure I).

**Investigations**

Investigations such as X-ray foot AP and lateral,

random blood sugar, bleeding time and clotting time were done.

**Data collection**

After obtaining written informed consent from the selected patients, demographic data, chief complaints at presentation and history was taken and clinical examination was done for all patients and findings were recorded on predesigned and pretested proforma (Annexure II).

**Procedure**

Group A/ Autologous blood injection group

Under aseptic precautions patients were infiltrated with an injection of 2 ml autologous blood drawn from cubital vein and 1 ml 0.5% bupivacaine at the medial side of the calcaneum.

Group B/ Local corticosteroid injection group

Patients were infiltrated with 2 ml of local corticosteroid (mixed with methyl prednisolone acetate 80 mg) mixed with 1ml 0.5% bupivacaine at medial border of according to below mentioned technique.

Technique

*Patient position:* supine position

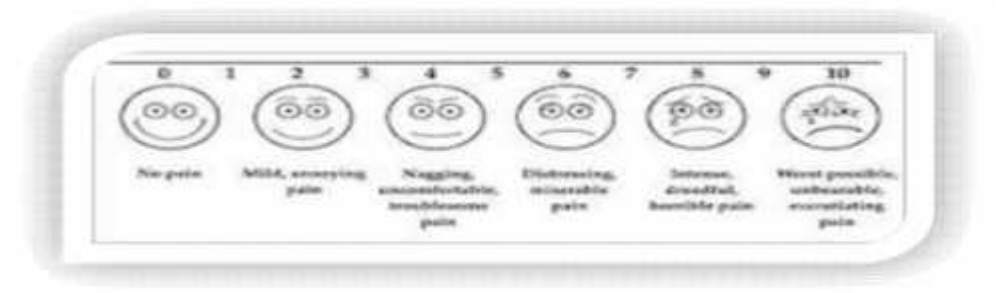
*Land marks:*

- Distal longitudinal crease at medial sole
- Proximal base of medial longitudinal arch.
- Level of antero-medial process of calcaneal tuberosity.
- Identify point of maximum tenderness.

Mark needle insertion site based on landmark.

**Outcome variables**

Pain of the participants will be assessed by most widely used and accepted "visual analogue scale". It consists of a 10 centimeter line marked at one end with "no pain" and at other end with "worst pain ever". Participant is asked to indicate where on the line he or she rates the pain on the day of presentation, 1, 4, 12weeks and 6 month of follow-ups. Numerical valve is then given to it simply by measuring length between "no pain" to patients mark.




**Visual analog scale**

**2. NIRSCHL STAGING:**

phase1: mild pain with exercise; resolves within 24 hours

phase2: pain after exercise; exceeds 48 hours

phase3: pain with exercise; does not alter activity

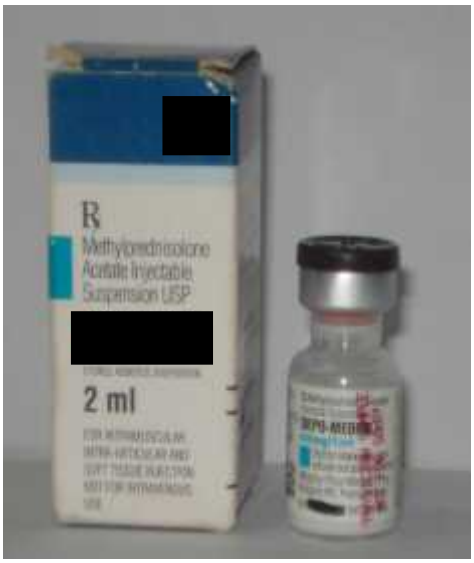
phase4: pain with exercise; alters activity

phase5: pain with heavy activities of daily living

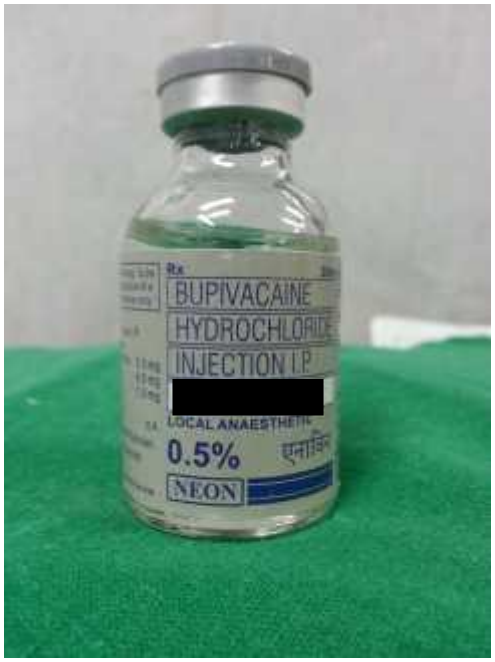
phase6: pain with light activities of daily living; Intermittent pain at rest

phase7: constant pain at rest; disrupts sleeps

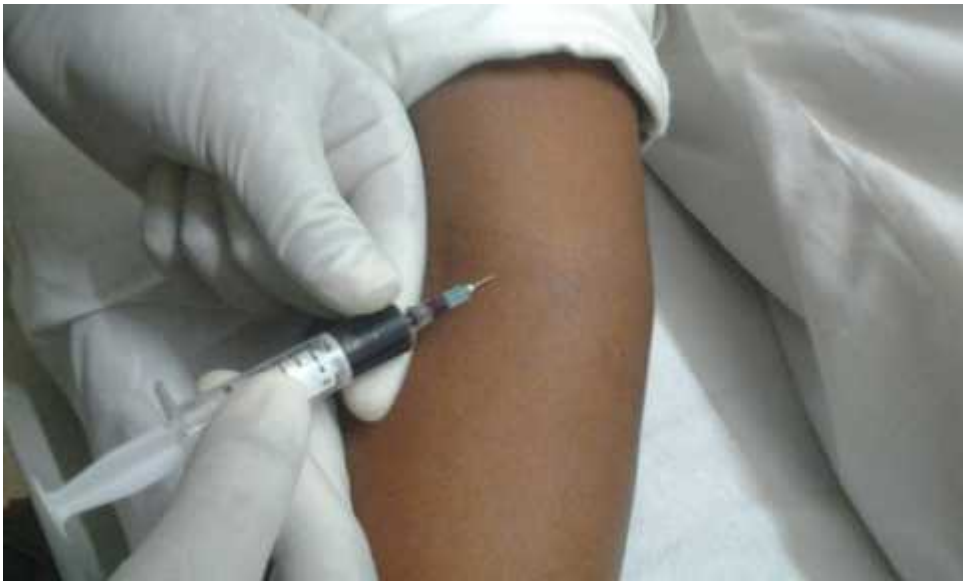
No pain_____1 _____ 2_____ 3_____4_____ 5_____ 6 _____ 7 worst pain



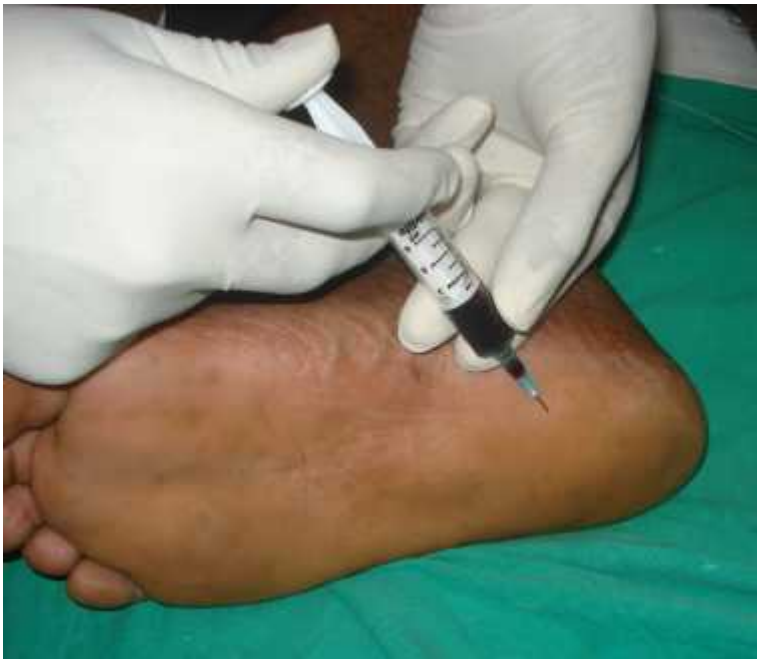

**Photograph 1. Methyl prednisolone acetate**



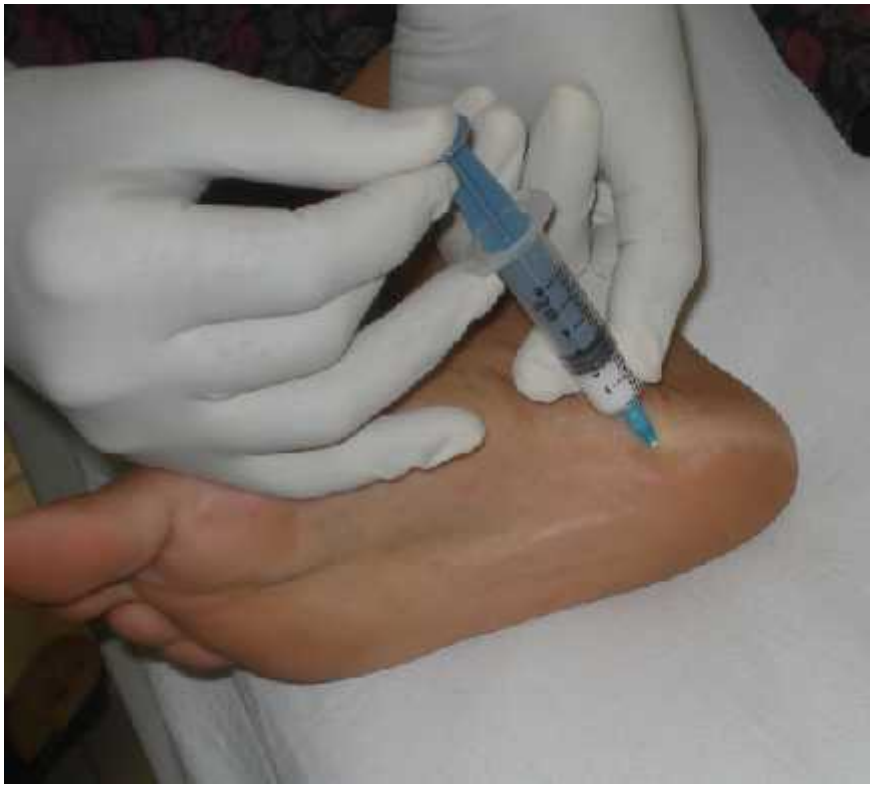

**Photograph 2. 0.5% Bupivacaine is added in both groups**



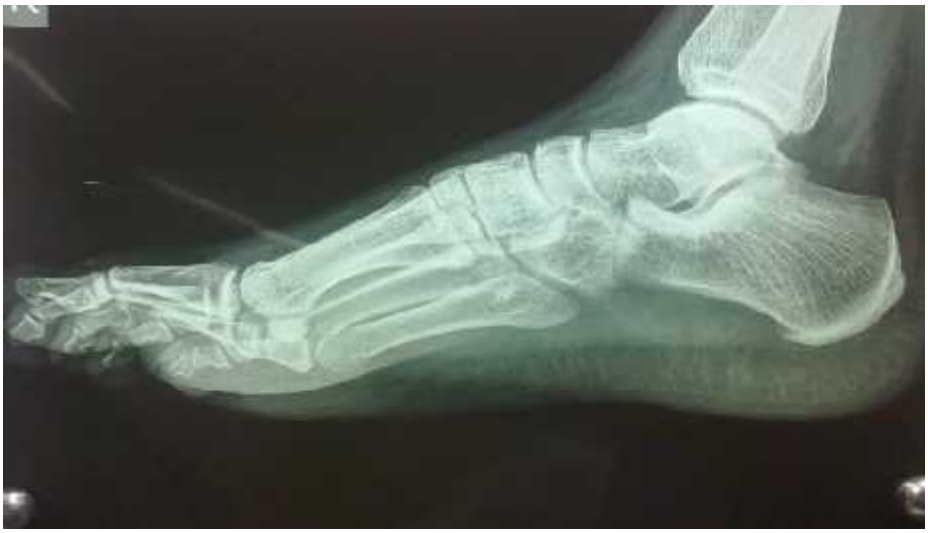
**Photograph 2. : Autologous blood (2 ml) is drawn from the upper limb vein**



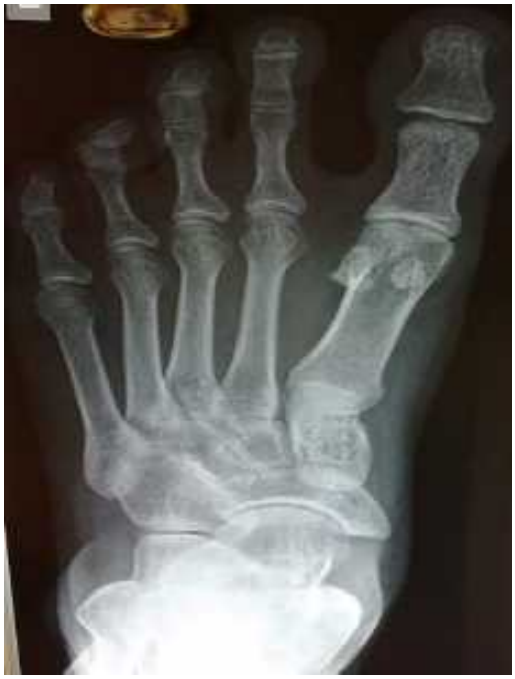
**Photograph 3. Group A – Autologus blood injection at anteromedial aspect of affected foot**



**Photograph 4. Group B Corticosteroid injection at anteromedial aspect of affected foot**



**Photograph 5. X-ray foot lateral view**



**Photograph 6. X-ray foot AP view.**

**Statistical analysis**

Data obtained was coded and entered into Microsoft Excel spreadsheet. The categorical data was expressed as rate, ratio and percentage. The continuous data was expressed as mean ± S.D. A 'p' value of less than or equal to 0.05 was considered as statistically significant.

Chapter 5

# Results

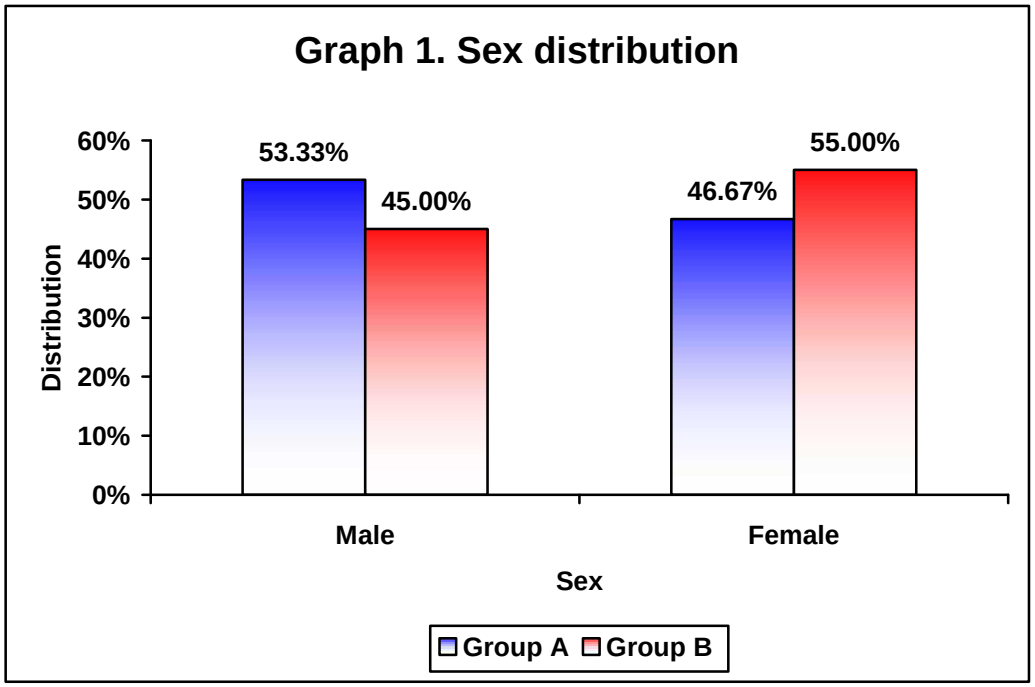
# RESULTS

Procedure was done in 120 patients under the present study. Participants were clinically evaluated. A baseline VAS scores and Nirschl staging of the pain at heel region was recorded. Cases were treated with autologous blood injection and controls with local corticosteroid injection. After the procedure patients were asked to report immediately if any increase in pain was there and were asked to follow up at 1 week, 4 weeks, 12 weeks and 6 months interval after the intervention. Some patients were given just placebos like calcium tablets or B-complex capsules for one to three weeks.

**Table 1. Sex distribution**

| Sex | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| Male | 32 | 53.33 | 27 | 45.00 |
| Female | 28 | 46.67 | 33 | 55.00 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**$x^2_1$=0.832** **p=0.361**

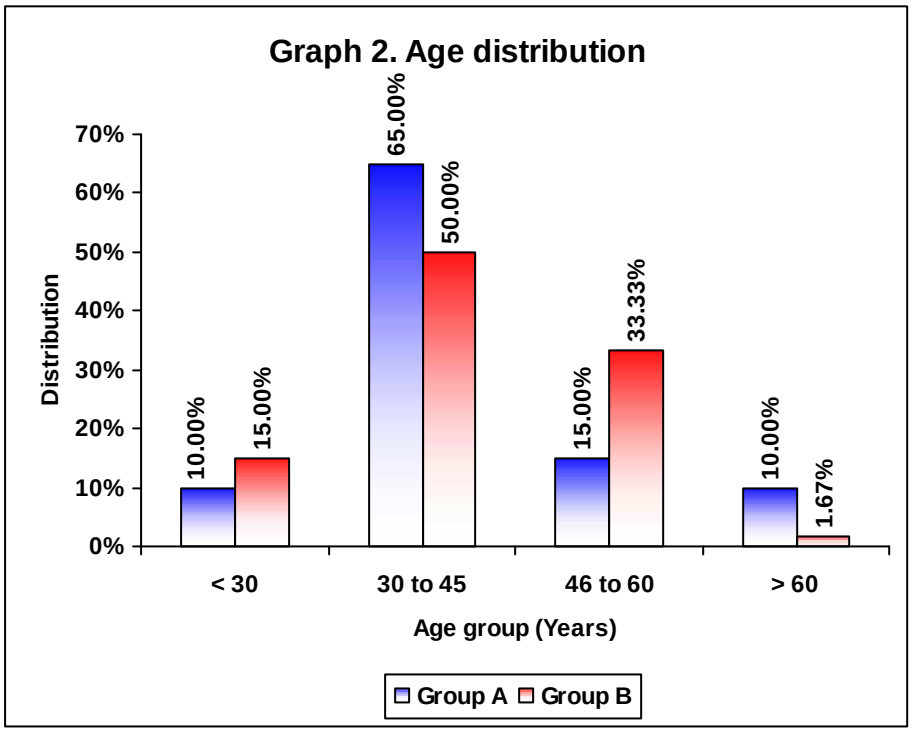



Out of the 120 participants, 59 were males and 61 were females. In autologous blood injection group 32 were males and 28 were females. In corticosteroid injection group 27 were males and 33 were females p=0.361 which is non significant. Thus both the groups were comparable in terms of number of males and females in each group.

**Table 2. Age distribution**

| Age group (Years) | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| < 30 | 6 | 10.00 | 9 | 15.00 |
| 30 to 45 | 39 | 65.00 | 30 | 50.00 |
| 46 to 60 | 9 | 15.00 | 20 | 33.33 |
| > 60 | 6 | 10.00 | 1 | 1.67 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**$x^2_3$=9.524** **p=0.023**

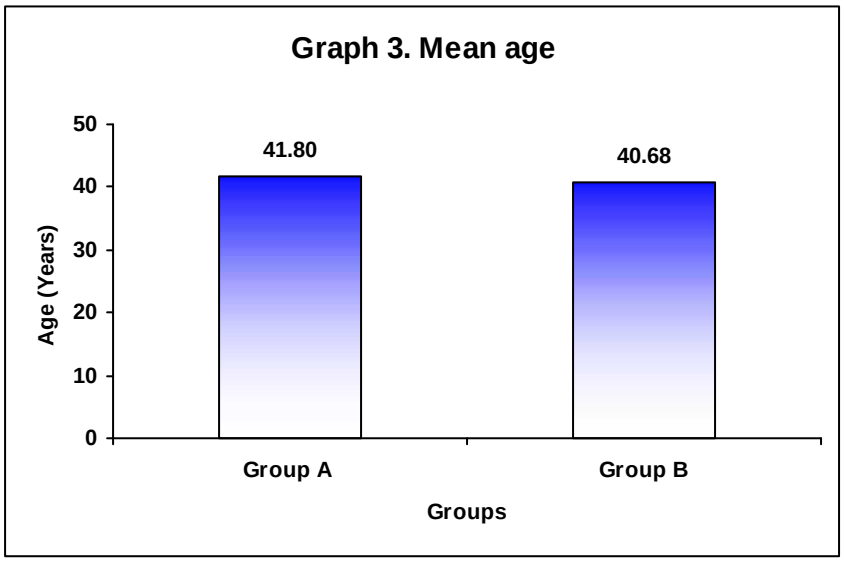



Most of the patients in group A (65%) and in group B (50%) were aged between 30 to 45 years.

**Table 3. Mean age**

| Age (Years) | Group A (n=60) | Group B (n=60) |
|---|---|---|
| Mean | 41.8 | 40.68 |
| SD | 10.96 | 10.47 |
| Median | 40 | 42 |
| Minimum | 22 | 17 |
| Maximum | 67 | 62 |

**t=0.572** **DF=118** **p=0.568**

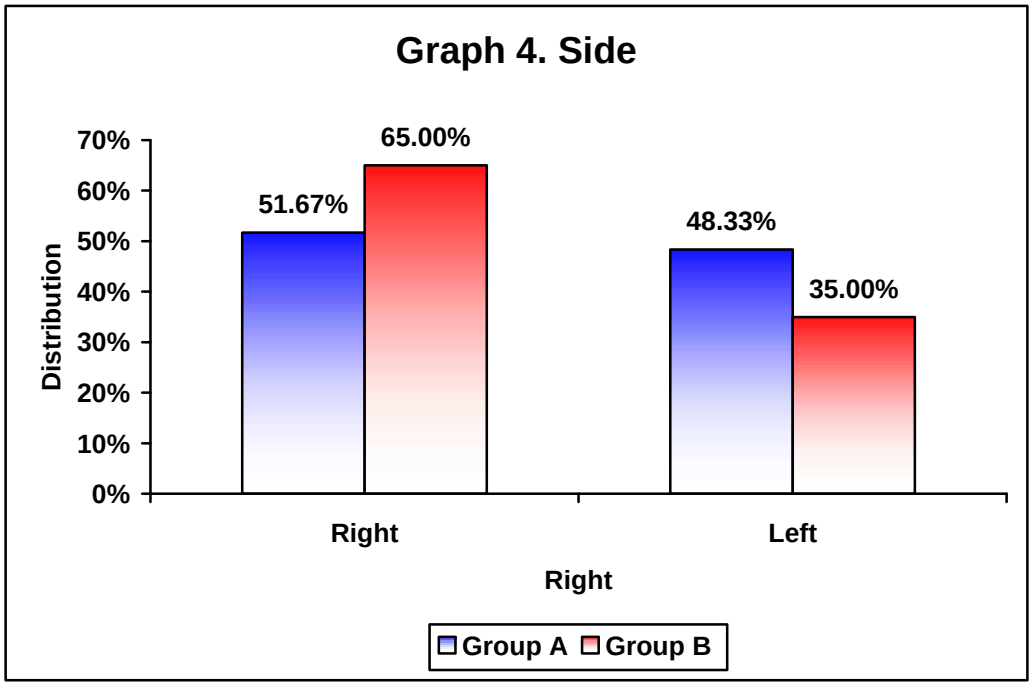



The mean in group A was 41.80 ± 10.96 years and in group B the mean age was 40.68 ± 10.47 years suggesting both the groups were comparable with respect to age (p=0.568).

**Table 4. Side**

| Side | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| Right | 31 | 51.67 | 39 | 65.00 |
| Left | 29 | 48.33 | 21 | 35.00 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**$x^2_1$=2.193** **p=0.138**




Out of the 120 participants, 70 participants had their right side elbow affected and 50 had their left side affected. p=0.138 which is not significant. Thus both the groups were comparable in terms of side of elbow involved.

**Table 5. Duration**

| Duration | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| < 5 | 21 | 35.00 | 30 | 50.00 |
| 5 to 10 | 15 | 25.00 | 5 | 8.33 |
| 11 to 15 | 7 | 11.67 | 10 | 16.67 |
| 16 to 20 | 5 | 8.33 | 12 | 20.00 |
| > 20 | 12 | 20.00 | 3 | 5.00 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**$x^2_4$=15.406** **p=0.004**

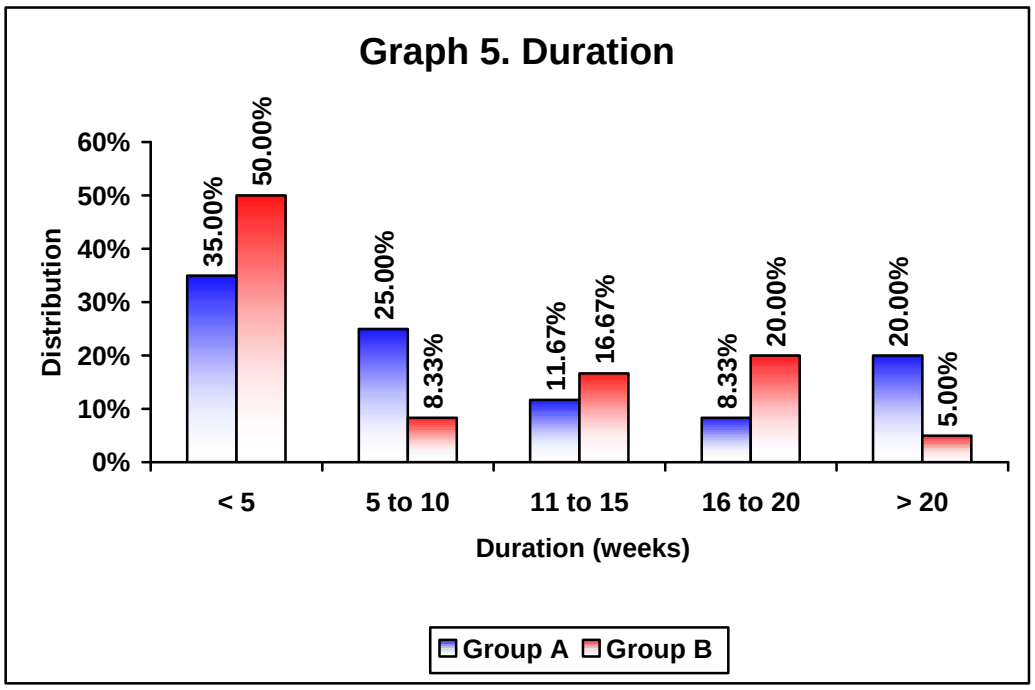
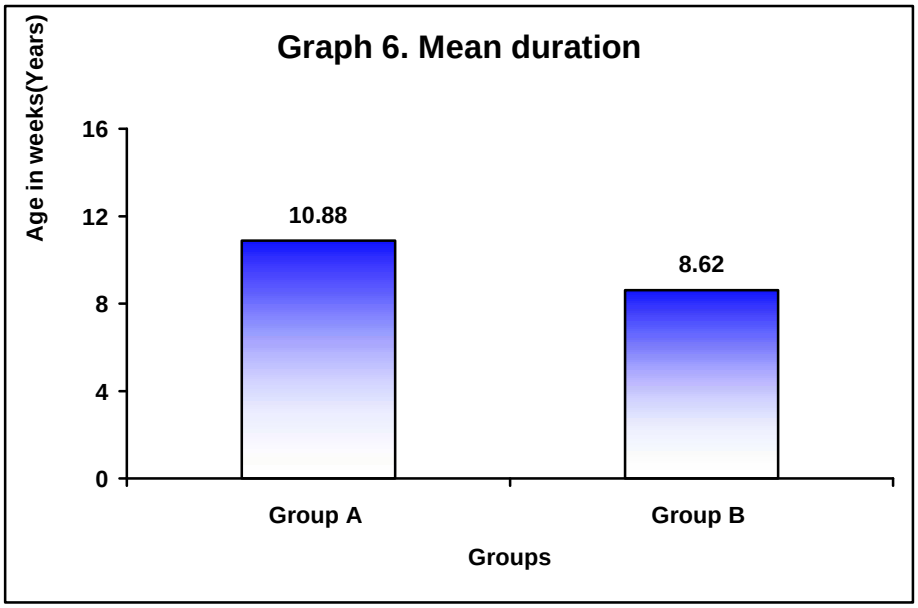


**Table 6. Mean duration**

| Duration (Weeks) | Group A (n=60) | Group B (n=60) |
|---|---|---|
| Mean | 10.88 | 8.62 |
| SD | 8.35 | 6.4 |
| Median | 8 | 5 |
| Minimum | 2 | 1 |
| Maximum | 26 | 24 |

**t=1.664** **p=0.101**



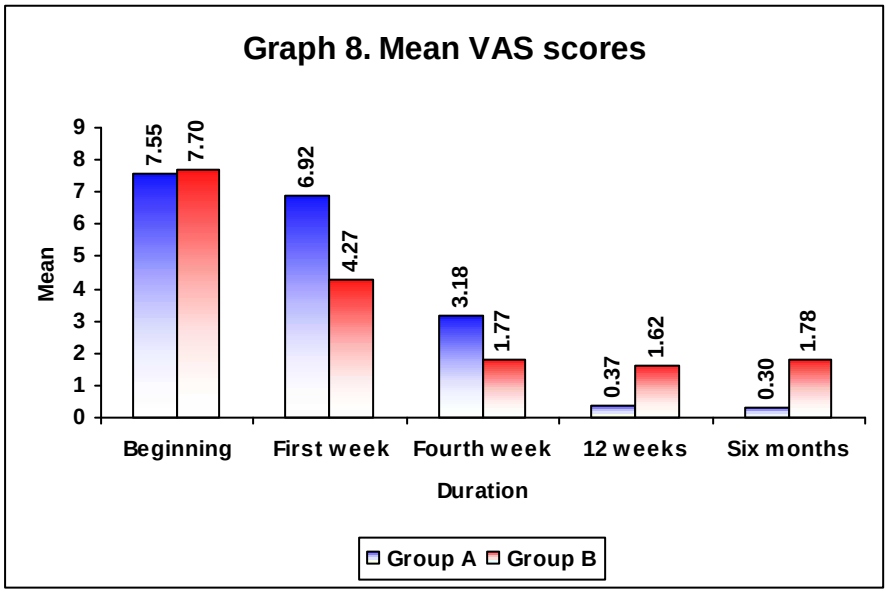

The mean duration of the condition in all 120 patients suffering from plantar fasciitis was 9.75 weeks. The mean duration of the condition in autologous blood injection group was 10.88 weeks. The mean duration of the condition in corticosteroid injection group was 8.62 weeks. P Value= 0.121 which is not significant. Thus both the groups were comparable in terms of duration of the condition in each group.

**Table 7. VAS scores**

| Duration | Pain | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|---|
| | | **Number** | **Percent** | **Number** | **Percent** |
| Beginning | Mild | 0 | 0.00 | 0 | 0.00 |
| | Moderate | 17 | 28.33 | 9 | 15.00 |
| | Severe | 43 | 71.67 | 51 | 85.00 |
| | Total | **60** | **100.00** | **60** | **100.00** |
| | | **$x^2_1$=1.162** | | | **p=0.204** |
| First week | Mild | 8 | 13.33 | 29 | 48.33 |
| | Moderate | 15 | 25.00 | 24 | 40.00 |
| | Severe | 37 | 61.67 | 7 | 11.67 |
| | Total | **60** | **271.67** | **60** | **285.00** |
| | | **$x^2_2$=34.451** | | | **p<0.001** |
| Fourth week | Mild | 34 | 56.67 | 42 | 70.00 |
| | Moderate | 24 | 40.00 | 15 | 25.00 |
| | Severe | 2 | 3.33 | 3 | 5.00 |
| | Total | **60** | **433.33** | **60** | **396.67** |
| | | **$x^2_1$=3.124** | | | **P=0.210** |
| 12 weeks | Mild | 58 | 96.67 | 46 | 76.67 |
| | Moderate | 0 | 0.00 | 14 | 23.33 |
| | Severe | 2 | 3.33 | 0 | 0.00 |
| | Total | **60** | **536.67** | **60** | **501.67** |
| | | **$x^2_1$=10.381** | | | **p=0.001** |
| Six months | Mild | 58 | 96.67 | 44 | 73.33 |
| | Moderate | 0 | 0.00 | 16 | 26.67 |
| | Severe | 2 | 3.33 | 0 | 0.00 |
| | Total | **60** | **640.00** | **60** | **601.67** |
| | | **$x^2_1$=12.814** | | | **p=0.0003** |

**Table 8. Mean VAS scores**

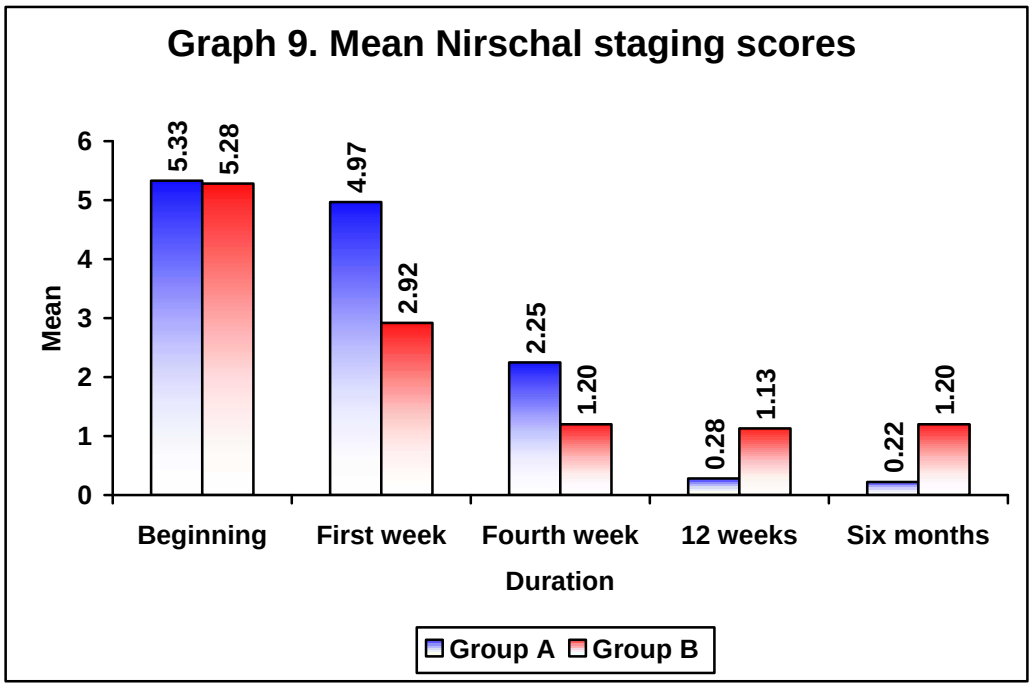
| Duration | Group A (n=60) | | Group B (n=60) | | 'z' value | 'p' value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| Beginning | 7.55 | 1.4 | 7.7 | 1.14 | 0.240 | 0.810 |
| First week | 6.92 | 2.04 | 4.27 | 1.76 | 6.229 | <0.001 |
| Fourth week | 3.18 | 2.38 | 1.77 | 2.49 | 3.969 | <0.001 |
| 12 weeks | 0.37 | 1.38 | 1.62 | 2.03 | 4.215 | <0.001 |
| Six months | 0.3 | 1.37 | 1.78 | 2.14 | 4.843 | <0.001 |




In this study, the mean VAS score at the beginning were comparable in both the groups (7.55 ± 1.40 vs 7.70 ± 1.14; p=0.810). At first week these scores reduced significantly in group B (4.27 ± 1.76) compared to group A (6.92 ± 2.04; p<0.001). Further, at fourth week the mean VAS scores in group A significantly reduced to 3.18 ± 2.38 and at 12 weeks and six months to 0.3 ± 1.37 (p<0.001).

**Table 9. Mean Nirschal staging scores**

| Duration | Group A (n=60) | | Group B (n=60) | | 'z' value | 'p' value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | | |
| Beginning | 5.33 | 1.23 | 5.28 | 0.98 | 0.508 | 0.611 |
| First week | 4.97 | 1.55 | 2.92 | 1.29 | 6.361 | <0.001 |
| Fourth week | 2.25 | 1.61 | 1.2 | 1.71 | 4.108 | <0.001 |
| 12 weeks | 0.28 | 0.94 | 1.13 | 1.5 | 4.003 | <0.001 |
| Six months | 0.22 | 0.92 | 1.2 | 1.52 | 4.758 | <0.001 |




At the beginning P value for Nirschal staging is 0.611 which are statistically not significant. Hence the outcome values before the injection are comparable.

At 1st week P value for Nirschal staging is p<0.001 which are statistically significant. Hence the decrease in pain at 1st week is statistically significant in corticosteroid injection group compared to autologous blood injection group.

At 4th week P value for Nirschal staging is p<0.001 Hence the decrease in pain at 4th week is statistically significant in corticosteroid injection group compared to autologous blood injection group.

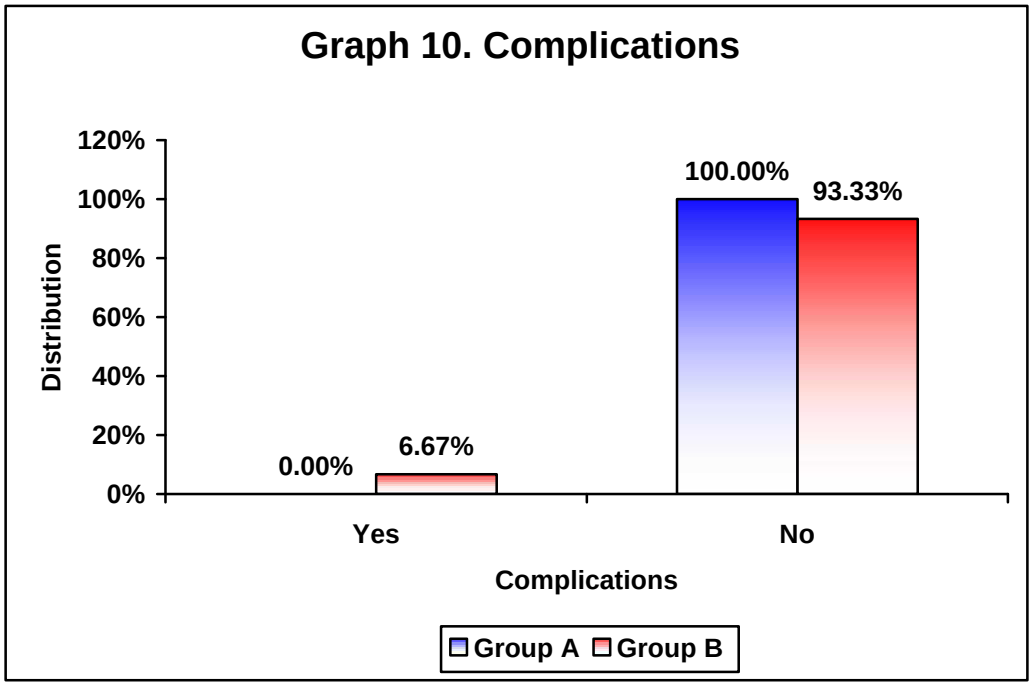
At 12th week P value for Nirschal staging is p<0.001 which are statistically significant. Hence at 12th week the decrease in pain is statistically significant in autologous blood injection group compared to corticosteroid injection group

At 6 month P value for Nirschal staging is p<0.001 which are statistically significant. Hence at 6 month the decrease in pain is statistically significant in autologous blood injection group compared to corticosteroid injection group.

**Table 10. Complications**

| Complications | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| Yes | 0 | 0.00 | 4 | 6.67 |
| No | 60 | 100.00 | 56 | 93.33 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**Fisher exact test p=0.118**

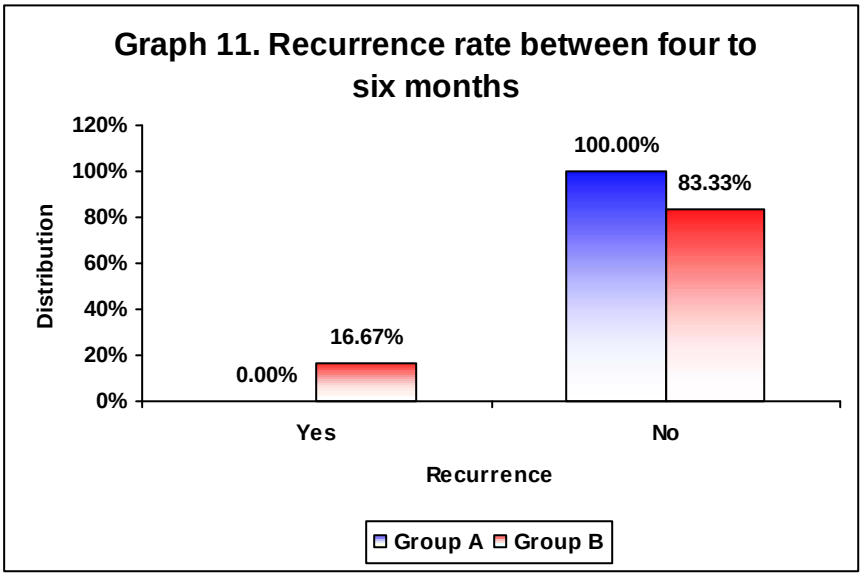



Only four patients (6.6%) had local skin atrophy in corticosteroid injection group while no patient in autologous blood injection group had this problem. P value=0.118 which is non-significant. There was no statistical significance related to post intervention local skin atrophy.

**Table 11. Recurrence rate between four months to six months**

| Recurrence | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| Yes | 0 | 0.00 | 10 | 16.67 |
| No | 60 | 100.00 | 50 | 83.33 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**$x^2_{YC}$=8.841** **p=0.003**

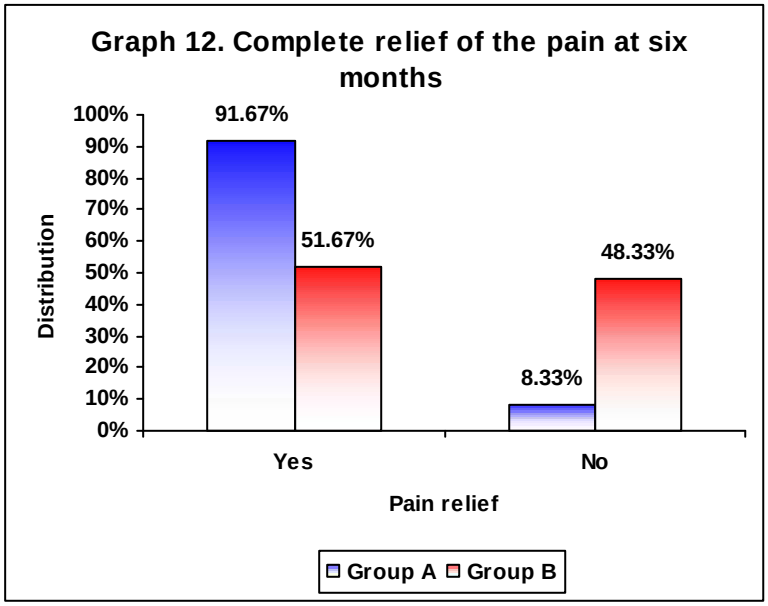



Patients reported recurrences rate between 4 to 6month follow up. The rate of recurrence was 16.67% in corticosteroid injection group. The rate of recurrence was 0% in autologous blood injection group. P= 0.003 which is significant. Thus corticosteroid injection group showed statistically significant high recurrence rate compared to autologous blood injection group.

**Table 12. Complete relief of the pain at six months**

| Pain relief | Group A (n=60) | | Group B (n=60) | |
|---|---|---|---|---|
| | **Number** | **Percent** | **Number** | **Percent** |
| Yes | 55 | 91.67 | 31 | 51.67 |
| No | 5 | 8.33 | 29 | 48.33 |
| **Total** | **60** | **100.00** | **60** | **100.00** |

**$x^2_1$=23.642** **p<0.001**




After 6 months of follow up, 55(91.67%) patients in autologous blood injection group were completely relieved of pain whereas 31 (51.67%) participants in corticosteroid injection were completely relieved of pain. P value= <0.001 which is significant. Thus autologous blood injection group had statistically significant more number of patients completely relieved of pain.

Chapter 6

# Discussion

# DISCUSSION

Plantar fasciitis is the most common cause of heel pain for which professional care is sought.[1] Although thought of as an inflammatory process, plantar fasciitis is a disorder of degenerative changes in the fascia, and may be more accurately termed plantar fasciosis.[3]

The major component contributing to discomfort is the irritation occurring secondary to the disease process, rather than a spur or other mechanical factor. Traditional therapeutic efforts have been directed at decreasing the presumed inflammation. These treatments include icing, nonsteroidal anti-inflammatory drugs (NSAIDs), rest and activity modification, corticosteroids, botulinum toxin type A, splinting, shoe modifications, and orthoses.

Other treatment techniques have been directed at resolving the degeneration caused by the disease process. In general, these techniques are designed to create an acute inflammatory reaction with the goal of restarting the healing process. These techniques include autologous blood injection, platelet-rich plasma (PRP) injection, nitroglycerin patches, extracorporeal shock-wave therapy (ESWT), and surgical procedures. Formal physical therapy can include components that target both goals.

It is important to note that these treatment modalities are to be used in combination, as components of a multimodal therapeutic approach. Such an approach can be challenging, in that it places high expectations on the patient with respect to responsibility, consistency, and compliance. If these expectations are met, the chances of success are good.

Recently, research has focused on regenerative therapies with high expectations of success. The use of autologous growth factors is thought to heal through collagen regeneration and the stimulation of a well-ordered angiogenesis. These growth factors are administered in the form of autologous whole blood or platelet-rich plasma (PRP). Platelets can be isolated using simple cell-separating systems. The degranulation of the α-granules in the platelets releases many different growth factors that play a role in tissue regeneration processes. Platelet-derived growth factor, transforming growth factor-β, vascular-derived endothelial growth factor, epithelial growth factor, hepatocyte growth factor and insulin-like growth factor are examples of such growth factors. Injections with autologous growth factors are becoming common in clinical practice.[115]

Hence, the present study was an attempt to evaluate the efficacy and role of autologous blood injection in plantar fasciitis by comparing with the local corticosteroid injection.

This one year randomized controlled trial was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2011 to December 2011 on 120 confirmed patients of plantar fasciitis.

In this study slight male preponderance was seen in both the groups (53.33% and 55%) with male female ratio of 1.14:1 in group A and 1:1.22 in group B. However this difference was statistically not significant (p=0.361). Most of the patients in group A (65%) and in group B (50%) were aged between 30 to 45 years. The mean in group A was 41.80 ± 10.96 years and in group B the mean

age was 40.68 ± 10.47 years suggesting both the groups were comparable with respect to age (p=0.568).

In the present study, most of the patients in group A and group B presented with right foot involvement (51.67% and 65.00% respectively; p=0.138). In group A, among 35% and in group B among 50% of patients duration of symptoms was within five weeks (p=0.004). The mean duration in group A was 10.88 compared to 8.62 weeks in group B (p=0.121). At the beginning of treatment severe pain was recorded among 71.67% in group A and 85% in group B. However this difference was statistically not significant. Hence, all the demographic and clinical variables were comparable in both the groups.

In this study at the first week of treatment, in group A 61.67% patients were still having sever grade of pain but in group B only 11.67% patients having pain. P value for VAS Score is p<0.001 which are statistically significant. Hence the decrease in pain at $1^{st}$ week is statistically significant in corticosteroid injection group compared to autologous blood injection group.

In the present study during the first week follow up significantly less number of patients had mild (13.33%), moderate (25%) and severe (61.67%) compared to group B (48.33%, 40% and 11.67% respectively) (p<0.001). Though at fourth week follow up most of the patients (70%) reported mild pain in group B compared to group C this difference was statistically not significant (p=0.210). At 12 weeks and six months follow up almost all the patients (96.67%) reported mild pain in group A compared 76.67% at 12 week follow up and 73.33% patients at six months follow up reported mild pain (p<0.001) suggesting overall

better pain control in group A at first week, 12 weeks and six months followup period.

In this study, the mean VAS score at the beginning were comparable in both the groups (7.55 ± 1.40 vs 7.70 ± 1.14; p=0.810). At first week these scores reduced significantly in group B (4.27 ± 1.76) compared to group A (6.92 ± 2.04; p<0.001). Further, at fourth week the mean VAS scores in group A significantly reduced to 3.18 ± 2.38 and at 12 weeks and six months to 0.3 ± 1.37 (p<0.001) suggesting significantly less pain in group A compared to group B. Similar trend of reduction among patients in groups A was observed with Nirschal staging scores (p<0.001).

In the present study, 6.6% patients had local skin atrophy in group B whereas no patient in group B had this problem (p=0.118) However, no statistically significant difference was observed between the groups.

In the present study patients recurrence was not observed in patients with group A whereas 16.67% patients reported recurrences between four to six months follow up suggesting significantly less recurrence rates with the treatment of autologus blood injection (p=0.003).

At six months of follow up, significantly more number of patients (91.67%) patients in group A were completely relieved of pain whereas more than half (51.67%) patients in group B were not relieved of pain (p=<0.001)

There is limited data showing comparison between autologous blood injection and corticosteroid injection. A prospective, randomized, controlled,

observer-blinded study[113] was done over a period of 6 months in Kuala Lumpur, Malaysia. Sixty-four patients were randomly allocated to either the autologous blood or corticosteroid treatment group. All patients were assessed for the worst pain daily on visual analogue scale (VAS) and tenderness threshold (TT) at the plantar fascia origin using a pressure algometer before treatment, and at 6 weeks, 3 months, and 6 months after treatment. A p value of 0.05 was considered significant. Data were complete for 61 patients. The reduction in VAS and increase in TT for both groups was significant over time ($p < 0.0001$). At 6 weeks and 3 months, the corticosteroid group had significantly lower VAS than the autologous blood group ($p < 0.011$ and $p < 0.005$, respectively), but the difference was not significant at 6 months. The corticosteroid group had significantly higher TT than the autologous blood group at 6 weeks, 3 months and 6 months ($p < 0.003$, $p < 0.003$, $p < 0.008$, respectively).

The limitation of the study was lack of comparison with other studies as there is minimal availability of the similar trials.

Chapter 7

# Conclusion

# CONCLUSION

Based on the results of our present study it may be concluded that, autologus blood injection significantly reduced the pain based on VAS and Nirschal staging without complications there by lowering the recurrence rate upto six months in patients with plantar fascitiis. It also provided complete relief of pain for the period of six months without any complication. Autologous blood is simple to acquire and prepare, easy to carry out.

Hence autologous blood provides intermediate and long term results in term of pain relief in compared to corticosteroid injection which gives short term relief.

Chapter 8

# Summary

# SUMMARY

Plantar fasciitis is an inflammation of the fascia of the plantar surface of the foot at the attachment to the calcaneum .Injection of autologous blood has been reported beneficial for both intermediate / long term outcome for treatment of plantar fasciitis and there was significant decrease in pain. The present study was undertaken to evaluate the efficacy and role of autologous blood injection in plantar fasciitis by comparing with the local corticosteroid injection.

This one year randomized controlled trial was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2011 to December 2011 on 120 confirmed patients of plantar fasciitis. Based on the computer generated randomization the selected patients were randomized into two groups namely; Group A (Autologus blood injection group) and Group B (Corticosteroid injection group).

In this study slight male preponderance was seen in both the groups (53.33% and 55%). The mean in group A was 41.80 ± 10.96 years and in group B the mean age was 40.68 ± 10.47 years. Most of the patients in group A and B presented with right foot involvement (51.67% and 65.00%). The mean duration in group A was 10.88 compared to 8.62 weeks in group B. The mean VAS score at the beginning were comparable in both the groups (7.55 ± 1.40 vs 7.70 ± 1.14; p=0.810). At fourth week the mean VAS scores in group A significantly reduced to 3.18 ± 2.38 and at 12 weeks and six months to 0.3 ± 1.37 suggesting significantly less pain in group A compared to group B. Similar trend of reduction among patients in groups A was observed with Nirschal staging scores.

No patient in group A reported complications and recurrence was not observed in patients with group A. At six months of follow up 91.67% patients in group A were completely relieved of pain whereas more than half (51.67%) patients in group B were not relieved of pain.

Overall, the present study showed that, autologus blood injection significantly reduced the pain without complications with no recurrence. It also provided complete relief of pain for the period of six months.

Chapter 9

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# Annexure I

# ANNEXURE I

## INFORMED CONSENT FORM FOR PARTICIPATION IN RESEARCH STUDY

## "A RANDOMIZED CONTROLLED TRAIL TO EVALUATE THE EFFICACY OF AUTOLOGOUS BLOOD INJECTION VERSUS LOCAL CORTICOSTEROID INJECTION IN TREATMENT OF PLANTAR FASCIITIS"

**Purpose of study:**

Since you have pain at medial tubercle of the calcaneum and been diagnosed as plantar fasciitis, you are eligible to participate in the study.

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

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

This study is done by Dr. Sharat Balemane under the guidance of Dr.S.H.Motimath, Professor, Dept. of orthopedics, J N Medical College, Belgaum**.**

**Procedure involved in the study**:

Technique of Autologous blood injection. You being in supine position affected foot will be painted and draped. The bony anatomical landmarks are identified. Two milliliters of autologous blood drown from the contra lateral upper extremity and mixed with 1 milliliter of 0.5% Bupivacaine. The needle introduced around medial tubercle of calcaneum.

Technique of Local corticosteroid injection: 2 milliliters of Methyl prednisolone acetate 80mg mixed with 1milliliter of 0.5% Bupivacaine. This is injected around medial tubercle of calcaneum.

Investigations may require whenever necessary for the study:

Anteroposterior/lateral view of Radiographs of foot.

Random blood sugar/ fasting blood sugar.

**Benefits of the study:**

The benefits are early decrease of pain. There will be no extra benefits to patients otherwise. No financial incentives will be given to the participants.

**Potential risk factors and discomforts:**

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

**Alternatives:**

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

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

**Privacy and confidentiality:**

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

I. If necessary to protect your rights or welfare.

II. If required by law.

**Institutional policy:**

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

**Financial incentives for participants:**

No financial incentives will be given to the participants.

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

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

**Authorization to publish results:**

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

**Consent statement:**

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

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

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

Dr. SHARAT BALEMANE 9916206204,

Dr. S.H.MOTIMATH. 9448110522

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

Participants name: signature:

Witness's name: signature:

Experimenter's name: signature:

Date:

# Annexure II

## ANNEXURE II – PROFORMA

# PROFORMA

S.NO :

I.P. NO:

O.P.NO:

NAME :

AGE :

SEX :

RELIGION:

OCCUPATION:

INCOME:

ADDRESS:

**CHIEF COMPLAINTS:**

**1. Pain at right / left side of the heel**

**2. Inability to dorsiflexion the ankle.**

**3. Duration**

**4. Any other associated illness**

## HISTORY OF PRESENT ILLNESS :

**PAIN Site :** **right side /left side of heel**

**Onset :** **sudden / gradual**

**Nature:** **continuous/intermittent**

**Relation with movement:** **present / absent**

**Character :** **dull aching /localized pain**

**Mode of onset:**

**Gradual onset/sudden onset**

**After prolonged walking,**

**Severity :** **mild/moderate/sever**

**Relation with movements:** **Pain more during resisted Dorsiflexion**

**Any other systemic complaints:**

**PAST HISTORY**

**H/O Previous episode:** **Y/ N**

**Any associated illness:**

**Diabetes mellitus**

**Bleeding disorders**

**Any anticoagulant intake**

**Rheumatoid arthritis**

**Osteo arthritis**

**Trauma**

**PERSONAL HISTORY**

**Smoker / non smoker**

**Alcoholic/non alcoholic**

**Nature of work**

**Footwear used**

**Bare foot walker: Y/N**

**FAMILY HISTORY**

**H/O Similar complaints in family**

**GENERAL PHYSICAL EXAMINATION**

**Built – poor/ moderate/well**

**Lymphadenopathy- significant / Non significant**

**Clubbing- Present / absent**

**Body mass index; kg/m2**

**Pulse: /min**

**B P: mm hg**

**R.R: /min**

**Temperature: febrile/ non febrile**

**LOCAL EXAMINATION:**

**EXAMINATION OF FOOT:**

**Attitude of the limb:**

**Overlying skin:**

**Deformity:**

**Local swelling: present/ absent**

**PALPATION:**

**Local temperature: Increased /Normal**

**Tenderness over the anterior medial aspect of the calcaneum**

**: Present / absent**

**Bony irregularity: Present / Absent**

**Swelling : Present / Absent**

**MOVEMENTS:**

**Range of movements: Active Passive**

**Dorsiflexion:**

**Plantar flexion:**

**Inversion:**

**Eversion:**

**Abduction:**

**Adduction:**

**INVESTIGATION:**

**X ray: foot AP / lateral view**
**Random blood disorder**

**TREATEMENT:**

**Local Autologous blood injection**

**Local corticosteroid injection**

**OUTCOME PARAMETERS AT THE TIME OF PRESENTATION:**

1. **PAIN SCORE: VISUAL ANALOGUE SCALE**

   **No pain _1_2_3_4_5_6_7_8_9_10_ worst pain ever**

2. **NIRSCHL STAGING ;**

   **No pain _1_2_3_4_5_6_7_worst pain**

**Name:**

**Age:**

**Sex:**

**Date of injection:**

| Weeks | VAS score | Nirschal staging |
|---|---|---|
| **At time of presentation (before injection)** | | |
| **1st week** | | |
| **4th week** | | |
| **12th week** | | |
| **6th month** | | |

**COMPLICATION:**

**Local skin atrophy:** **Y/N**

**Post injection exacerbation of pain:** **Y / N**

**(If yes how many days …….)**

**Infection :** **Y/N**

**Foot pad atrophy:** **Y/N**

**Fascia rupture:** **Y/N**

## MASTER CHART - GROUP A (AUTOLOGUS BLOOD INJECTION)

| Serial Number | Out patient number | Sex | Age (Years) | History | | Date of injection | Assessment of pain scores | | | | | | | | | | Maximum benefit (Weeks) | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | VAS Score | | | | | Nirschal staging | | | | | | |
| | | | | Side | Duration (weeks) | | Before injection | 1st Week | 4th week | 12th week | 6 months | Before injection | 1st Week | 4th week | 12th week | 6 months | | |
| 1 | 393240 | F | 56 | R | 3 | 2.1.2011 | 8 | 7 | 1 | 0 | 0 | 6 | 5 | 1 | 0 | 0 | 12 | N |
| 2 | 1361885 | F | 43 | R | 4 | 2.1.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 3 | 1434206 | M | 42 | L | 12 | 5.1.2011 | 6 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 4 | 1426858 | F | 52 | R | 8 | 7.1.2011 | 8 | 8 | 5 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 5 | 1451955 | M | 52 | L | 8 | 9.1.2011 | 7 | 3 | 2 | 0 | 0 | 5 | 3 | 1 | 0 | 0 | 12 | N |
| 6 | 1382600 | M | 62 | R | 6 | 10.1.2011 | 7 | 7 | 6 | 0 | 0 | 5 | 5 | 4 | 0 | 0 | 12 | N |
| 7 | 1511239 | F | 33 | L | 2 | 12.1.2011 | 6 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 8 | 1373484 | F | 34 | R | 12 | 14.1.2011 | 5 | 5 | 1 | 1 | 1 | 3 | 3 | 1 | 1 | 1 | 4 | N |
| 9 | 1364810 | M | 35 | L | 24 | 16.1.2011 | 9 | 9 | 6 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 10 | 1564113 | M | 35 | R | 6 | 18.1.2011 | 8 | 8 | 1 | 0 | 0 | 6 | 6 | 1 | 0 | 0 | 12 | N |
| 11 | 1522413 | M | 42 | R | 4 | 19.1.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 12 | 1582312 | F | 35 | R | 8 | 19.1.2011 | 8 | 8 | 5 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |
| 13 | 1367128 | M | 42 | L | 26 | 22.1.2011 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 4 | 0 | 0 | 12 | N |
| 14 | 1371828 | F | 38 | L | 8 | 27.1.2011 | 8 | 8 | 5 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 15 | 1318721 | F | 40 | R | 4 | 2.2.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 16 | 1511271 | M | 35 | R | 4 | 13.2.2011 | 5 | 5 | 3 | 1 | 0 | 3 | 3 | 2 | 1 | 0 | 25 | N |
| 17 | 1564781 | M | 42 | L | 8 | 23.2.2011 | 7 | 3 | 2 | 0 | 0 | 5 | 3 | 1 | 0 | 0 | 12 | N |
| 18 | 1572812 | F | 32 | R | 26 | 27.2.2O11 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 4 | 0 | 0 | 12 | N |
| 19 | 1571888 | M | 46 | L | 24 | 1.3.2011 | 9 | 9 | 6 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 20 | 1372818 | F | 42 | R | 4 | 5.3.2011 | 9 | 7 | 3 | 0 | 0 | 6 | 5 | 2 | 0 | 0 | 12 | N |
| 21 | 1565132 | F | 35 | L | 18 | 7.3.2011 | 8 | 8 | 7 | 7 | 7 | 6 | 6 | 5 | 5 | 5 | 4 | N |
| 22 | 1560182 | M | 40 | L | 26 | 12.3.2011 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 4 | 0 | 0 | 12 | N |
| 23 | 1374656 | F | 45 | R | 3 | 21.3.2011 | 7 | 6 | 3 | 0 | 0 | 5 | 4 | 2 | 0 | 0 | 12 | N |
| 24 | 1606675 | M | 40 | L | 2 | 24.3.2011 | 9 | 8 | 0 | 0 | 0 | 7 | 7 | 0 | 0 | 0 | 4 | N |
| 25 | 1608981 | F | 35 | L | 6 | 2.4.2011 | 8 | 8 | 1 | 0 | 0 | 6 | 6 | 1 | 0 | 0 | 12 | N |
| 26 | 606965 | M | 34 | L | 12 | 5.4.2011 | 5 | 5 | 1 | 1 | 1 | 3 | 3 | 1 | 1 | 1 | 4 | N |
| 27 | 854687 | F | 38 | R | 6 | 13.4.2011 | 8 | 8 | 6 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 28 | 1744132 | M | 40 | L | 26 | 22.4.2011 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 4 | 0 | 0 | 12 | N |
| 29 | 1711753 | F | 22 | L | 4 | 24.4.2011 | 5 | 5 | 3 | 1 | 0 | 3 | 3 | 2 | 1 | 0 | 25 | N |
| 30 | 851276 | F | 35 | R | 24 | 2.5.2011 | 9 | 9 | 6 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 31 | 1638720 | M | 64 | L | 12 | 9.5.2011 | 8 | 9 | 8 | 8 | 8 | 5 | 6 | 5 | 5 | 5 | 25 | N |
| 32 | 1647510 | F | 60 | L | 16 | 26.5.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 33 | 1637180 | F | 40 | R | 4 | 2.6.2011 | 9 | 7 | 3 | 0 | 0 | 6 | 5 | 2 | 0 | 0 | 12 | N |
| 34 | 1681360 | M | 23 | R | 3 | 5.6.2011 | 8 | 7 | 0 | 0 | 0 | 6 | 5 | 0 | 0 | 0 | 4 | N |
| 35 | 1671210 | F | 38 | L | 2 | 9.6.2011 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 5 | 0 | 0 | 12 | N |

## MASTER CHART - GROUP A (AUTOLOGUS BLOOD INJECTION)

| Serial Number | Out patient number | Sex | Age (Years) | History | | Date of injection | Assessment of pain scores | | | | | | | | | | Maximum benefit (Weeks) | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | VAS Score | | | | | Nirschal staging | | | | | | |
| | | | | Side | Duration (weeks) | | Before injection | 1st Week | 4th week | 12th week | 6 months | Before injection | 1st Week | 4th week | 12th week | 6 months | | |
| 36 | 1718820 | M | 48 | R | 16 | 14.6.2011 | 8 | 8 | 5 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |
| 37 | 1871222 | M | 27 | R | 8 | 18.6.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 38 | 1872110 | F | 45 | R | 6 | 22.6.2011 | 10 | 10 | 5 | 0 | 0 | 7 | 7 | 3 | 0 | 0 | 12 | N |
| 39 | 1887828 | M | 38 | R | 8 | 3.7.2011 | 7 | 3 | 2 | 0 | 0 | 5 | 3 | 1 | 0 | 0 | 12 | N |
| 40 | 1882315 | M | 32 | R | 26 | 7.7.2011 | 7 | 7 | 6 | 0 | 0 | 5 | 5 | 4 | 0 | 0 | 12 | N |
| 41 | 1889215 | F | 54 | R | 24 | 10.7.2011 | 6 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 42 | 1892175 | F | 32 | L | 4 | 14.7.2011 | 5 | 5 | 1 | 1 | 1 | 3 | 3 | 1 | 1 | 1 | 4 | N |
| 43 | 1877212 | M | 65 | L | 18 | 24.07.2011 | 9 | 9 | 6 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 44 | 1877234 | F | 45 | L | 26 | 30.7.2011 | 8 | 8 | 1 | 0 | 0 | 6 | 6 | 1 | 0 | 0 | 12 | N |
| 45 | 1750289 | F | 57 | L | 3 | 6.8.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 46 | 1749222 | M | 28 | R | 2 | 9.8.2011 | 8 | 8 | 5 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |
| 47 | 1733329 | M | 33 | L | 6 | 13.8.2011 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 4 | 0 | 0 | 12 | N |
| 48 | 1521491 | F | 26 | R | 12 | 18.8.2011 | 8 | 8 | 5 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 49 | 1537558 | M | 33 | L | 6 | 28.8.2011 | 6 | 6 | 1 | 0 | 0 | 4 | 4 | 1 | 0 | 0 | 12 | N |
| 50 | 1546559 | F | 44 | R | 26 | 4.9.2011 | 5 | 5 | 3 | 1 | 0 | 3 | 3 | 2 | 1 | 0 | 25 | N |
| 51 | 1546559 | M | 52 | R | 4 | 15.9.2011 | 7 | 3 | 2 | 0 | 0 | 5 | 3 | 1 | 0 | 0 | 12 | N |
| 52 | 1924153 | M | 31 | L | 24 | 22.9.2011 | 8 | 7 | 0 | 0 | 0 | 6 | 5 | 0 | 0 | 0 | 4 | N |
| 53 | 1546557 | M | 40 | L | 12 | 6.10.2011 | 9 | 9 | 6 | 0 | 0 | 7 | 7 | 5 | 0 | 0 | 12 | N |
| 54 | 1546556 | F | 62 | L | 4 | 17.10.2011 | 9 | 9 | 4 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |
| 55 | 1879301 | F | 30 | L | 2 | 20.11.2011 | 9 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 56 | 1879287 | M | 67 | R | 2 | 29.11.2012 | 6 | 6 | 1 | 1 | 0 | 4 | 4 | 1 | 1 | 0 | 25 | N |
| 57 | 1878935 | M | 45 | R | 8 | 3.12.2011 | 9 | 9 | 0 | 0 | 0 | 6 | 6 | 0 | 0 | 0 | 4 | N |
| 58 | 1884822 | M | 37 | R | 12 | 23.12.2011 | 8 | 9 | 3 | 0 | 0 | 5 | 6 | 2 | 0 | 0 | 12 | N |
| 59 | 188489 | M | 45 | L | 16 | 1.1.2012 | 9 | 9 | 5 | 0 | 0 | 6 | 6 | 4 | 0 | 0 | 12 | N |
| 60 | 1884817 | M | 65 | R | 4 | 6.1.2011 | 8 | 8 | 4 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |

# MASTER CHART - GROUP B (CORTICOSTEROID INJECTION)

| Serial Number | Out patient number | Sex | Age (Years) | History | | Date of injection | Assessment of pain scores | | | | | | | | | | Maximum benefit (Weeks) | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | VAS Score | | | | | Nirschal staging | | | | | | |
| | | | | Side | Duration (weeks) | | Before injection | 1st Week | 4th week | 12th week | 6 months | Before injection | 1st Week | 4th week | 12th week | 6 months | | |
| 1 | 393240 | M | 50 | R | 8 | 2.1.2011 | 10 | 9 | 5 | 3 | 5 | 7 | 6 | 3 | 2 | 3 | 12 | N |
| 2 | 1369139 | M | 21 | R | 16 | 5.1.2011 | 8 | 5 | 0 | 0 | 0 | 5 | 3 | 0 | 0 | 0 | 4 | N |
| 3 | 1401132 | M | 53 | R | 16 | 7.1.2011 | 8 | 5 | 5 | 5 | 6 | 6 | 4 | 4 | 4 | 4 | 1 | N |
| 4 | 1426858 | M | 22 | R | 8 | 8.1.2011 | 7 | 3 | 0 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 4 | N |
| 5 | 1451955 | F | 23 | L | 4 | 9.1.2011 | 9 | 4 | 4 | 0 | 0 | 6 | 3 | 3 | 0 | 0 | 12 | N |
| 6 | 1382600 | F | 46 | L | 4 | 12.1.2011 | 7 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 7 | 1364810 | F | 37 | R | 24 | 13.1.2011 | 5 | 5 | 0 | 0 | 0 | 4 | 4 | 0 | 0 | 0 | 4 | N |
| 8 | 1564113 | M | 23 | L | 4 | 14.1.2011 | 6 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 9 | 1487182 | M | 45 | L | 4 | 16.1.2011 | 7 | 5 | 0 | 0 | 0 | 5 | 4 | 0 | 0 | 0 | 4 | N |
| 10 | 443810 | F | 46 | R | 4 | 17.1.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | Y |
| 11 | 1373500 | F | 34 | L | 1 | 21.1.2011 | 8 | 3 | 0 | 2 | 2 | 5 | 2 | 0 | 1 | 1 | 4 | N |
| 12 | 1371281 | M | 23 | 46 | 2 | 26.1.2011 | 9 | 2 | 2 | 2 | 2 | 6 | 1 | 1 | 1 | 1 | 1 | N |
| 13 | 1372816 | F | 34 | R | 12 | 2.2.2011 | 8 | 6 | 6 | 6 | 5 | 6 | 4 | 4 | 4 | 3 | 25 | N |
| 14 | 1561821 | M | 34 | L | 16 | 12.2.2011 | 8 | 3 | 2 | 4 | 4 | 6 | 2 | 1 | 3 | 3 | 4 | N |
| 15 | 1562818 | M | 56 | R | 4 | 16.2.2011 | 9 | 8 | 8 | 5 | 5 | 6 | 5 | 5 | 4 | 4 | 12 | N |
| 16 | 1580118 | F | 34 | L | 16 | 18.2.2011 | 8 | 5 | 0 | 0 | 0 | 5 | 3 | 0 | 0 | 0 | 4 | N |
| 17 | 1432181 | F | 56 | L | 16 | 20.2.2011 | 8 | 5 | 5 | 5 | 6 | 6 | 4 | 4 | 4 | 4 | 1 | N |
| 18 | 1432143 | F | 22 | R | 8 | 22.2.2011 | 7 | 3 | 0 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 4 | N |
| 19 | 1568141 | M | 56 | R | 4 | 28.2.2011 | 9 | 4 | 4 | 0 | 0 | 6 | 3 | 3 | 0 | 0 | 12 | N |
| 20 | 1628613 | F | 45 | L | 6 | 3.3.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 21 | 1674708 | M | 48 | L | 12 | 8.3.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 22 | 1666853 | M | 46 | R | 16 | 14.3.2011 | 9 | 4 | 4 | 2 | 2 | 6 | 3 | 3 | 1 | 1 | 12 | N |
| 23 | 443810 | F | 37 | R | 4 | 18.3.2011 | 6 | 2 | 0 | 0 | 0 | 4 | 1 | 0 | 0 | 0 | 4 | N |
| 24 | 1723313 | F | 37 | L | 4 | 24.3.2011 | 8 | 5 | 0 | 4 | 5 | 6 | 4 | 0 | 3 | 4 | 4 | N |
| 25 | 1856175 | M | 32 | L | 16 | 28.3.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 26 | 1876135 | M | 48 | R | 12 | 4.4.2011 | 8 | 3 | 0 | 2 | 2 | 5 | 2 | 0 | 1 | 1 | 4 | N |
| 27 | 1911167 | F | 50 | R | 4 | 13.04.2011 | 9 | 2 | 2 | 2 | 2 | 6 | 1 | 1 | 1 | 1 | 1 | N |
| 28 | 1778162 | F | 40 | L | 16 | 18.4.2011 | 8 | 6 | 6 | 6 | 5 | 6 | 4 | 4 | 4 | 3 | 25 | N |
| 29 | 1887826 | M | 37 | R | 16 | 24.04.2011 | 8 | 3 | 2 | 4 | 4 | 6 | 2 | 1 | 3 | 3 | 4 | Y |
| 30 | 1887651 | F | 33 | L | 4 | 28.04.2011 | 9 | 8 | 8 | 5 | 5 | 6 | 5 | 5 | 4 | 4 | 12 | Y |
| 31 | 1887825 | M | 55 | R | 4 | 30.04.2011 | 7 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 32 | 1887625 | M | 30 | R | 12 | 6.5.2011 | 8 | 8 | 4 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |
| 33 | 1887125 | M | 45 | R | 1 | 16.5.2011 | 9 | 5 | 0 | 1 | 3 | 6 | 4 | 0 | 1 | 2 | 4 | N |
| 34 | 1887182 | F | 37 | R | 12 | 22.5.2011 | 7 | 6 | 4 | 1 | 4 | 5 | 4 | 3 | 1 | 3 | 12 | N |
| 35 | 1991712 | M | 35 | R | 3 | 27.05.2011 | 8 | 4 | 0 | 2 | 3 | 6 | 2 | 0 | 1 | 2 | 4 | N |

# MASTER CHART - GROUP B (CORTICOSTEROID INJECTION)

| Serial Number | Out patient number | Sex | Age (Years) | History | | Date of injection | Assessment of pain scores | | | | | | | | | | Maximum benefit (Weeks) | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | VAS Score | | | | | Nirschal staging | | | | | | |
| | | | | Side | Duration (weeks) | | Before injection | 1st Week | 4th week | 12th week | 6 months | Before injection | 1st Week | 4th week | 12th week | 6 months | | |
| 36 | 1518674 | F | 48 | L | 12 | 29.05.2011. | 8 | 5 | 0 | 0 | 0 | 5 | 3 | 0 | 0 | 0 | 4 | N |
| 37 | 1534766 | F | 37 | R | 2 | 10.06.2011 | 5 | 3 | 0 | 3 | 3 | 4 | 2 | 0 | 2 | 2 | 4 | N |
| 38 | 1811112 | F | 52 | R | 24 | 11.06.2011 | 6 | 3 | 0 | 1 | 1 | 4 | 2 | 0 | 1 | 1 | 4 | N |
| 39 | 1811057 | M | 37 | R | 2 | 15.6.2011 | 5 | 3 | 0 | 0 | 0 | 5 | 2 | 0 | 0 | 0 | 4 | N |
| 40 | 1811141 | F | 45 | R | 3 | 22.06.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 41 | 1811035 | F | 47 | L | 2 | 28.6.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 42 | 1817223 | M | 49 | R | 12 | 4.7.2011 | 9 | 4 | 4 | 2 | 2 | 6 | 3 | 3 | 1 | 1 | 12 | N |
| 43 | 1817115 | F | 42 | L | 2 | 7.7.2011 | 6 | 2 | 0 | 0 | 0 | 4 | 1 | 0 | 0 | 0 | 4 | N |
| 44 | 1816912 | F | 17 | R | 12 | 11.7.2011 | 8 | 5 | 0 | 4 | 5 | 6 | 4 | 0 | 3 | 4 | 4 | N |
| 45 | 1820585 | F | 40 | R | 4 | 18.7.2011 | 8 | 3 | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 0 | 4 | N |
| 46 | 1820357 | M | 45 | L | 1 | 22.07.2011 | 8 | 3 | 0 | 2 | 2 | 5 | 2 | 0 | 1 | 1 | 4 | Y |
| 47 | 1828818 | F | 60 | L | 2 | 26.07.2011 | 9 | 2 | 2 | 2 | 2 | 6 | 1 | 1 | 1 | 1 | 1 | N |
| 48 | 1824314 | M | 42 | L | 12 | 11.08.2011 | 8 | 6 | 6 | 6 | 5 | 6 | 4 | 4 | 4 | 3 | 25 | N |
| 49 | 1926032 | M | 50 | R | 16 | 13.08.2011 | 8 | 3 | 2 | 4 | 4 | 6 | 2 | 1 | 3 | 3 | 4 | N |
| 50 | 1654494 | F | 50 | R | 4 | 22.08.2011 | 9 | 8 | 8 | 5 | 5 | 6 | 5 | 5 | 4 | 4 | 12 | N |
| 51 | 1896861 | F | 43 | R | 16 | 24.08.2011 | 8 | 5 | 0 | 0 | 0 | 5 | 3 | 0 | 0 | 0 | 4 | N |
| 52 | 1879332 | F | 33 | R | 16 | 17.9.2011 | 8 | 5 | 5 | 5 | 6 | 6 | 4 | 4 | 4 | 4 | 1 | N |
| 53 | 1897003 | F | 39 | R | 8 | 28.09.2011 | 7 | 3 | 0 | 0 | 0 | 3 | 2 | 0 | 0 | 0 | 4 | N |
| 54 | 1848676 | F | 42 | R | 4 | 2.10.2011 | 9 | 4 | 4 | 0 | 0 | 6 | 3 | 3 | 0 | 0 | 12 | N |
| 55 | 1940595 | M | 32 | R | 4 | 10.10.2010 | 7 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 56 | 1848698 | F | 27 | R | 24 | 17.11.2011 | 5 | 5 | 0 | 0 | 0 | 4 | 4 | 0 | 0 | 0 | 4 | N |
| 57 | 1956424 | M | 62 | R | 4 | 2.12.2011 | 6 | 3 | 0 | 0 | 0 | 4 | 2 | 0 | 0 | 0 | 4 | N |
| 58 | 1848689 | F | 35 | R | 4 | 24.12.2011 | 7 | 5 | 0 | 0 | 0 | 5 | 4 | 0 | 0 | 0 | 4 | N |
| 59 | 1719257 | M | 52 | R | 2 | 2.1.2012 | 7 | 7 | 0 | 2 | 2 | 4 | 4 | 0 | 1 | 1 | 4 | N |
| 60 | 1982253 | F | 45 | L | 12 | 7.1.2012 | 8 | 8 | 4 | 0 | 0 | 6 | 6 | 3 | 0 | 0 | 12 | N |