A DISSERTATION ON

"A PROSPECTIVE ANALYSIS ON THE EFFICACY OF PERCUTANEOUS FLEXOR TENOTOMY PROCEDURES IN RATE OF HEALING AND RECURRENCE OF DIABETIC NEUROPATHIC DISTAL TOE ULCER"

Submitted to

THE TAMIL NADU DR.MGR MEDICAL UNIVERSITY
CHENNAI – 600032

In partial fulfillment of the regulations
for the awards of the degree of

M.S. DEGREE - GENERAL SURGERY BRANCH – I

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE, SALEM

MAY 2020

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DECLARATION BY THE CANDIDATE

I solemnly declare that this dissertation **"A PROSPECTIVE ANALYSIS ON THE EFFICACY OF PERCUTANEOUS FLEXOR TENOTOMY PROCEDURES IN RATE OF HEALING AND RECURRENCE OF DIABETIC NEUROPATHIC DISTAL TOE ULCER"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem- 636030 under the guidance and supervision of **Prof.Dr.C.RAJASEKARAN,M.S** Professor and HOD of General Surgery, Govt .Mohan Kumaramangalam Medical College and Hospital, Salem. This dissertation is submitted to the TamilnaduDr.M.G.R Medical University, Chennai- 38 in fulfilment of the University regulations for the award of the degree of M.S. General Surgery (Branch I).

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AProspective Analysis on the efficacy of percutaneous flexor tenotomy proceduresin rate of healing and recurrence of diabetic neuropathic distaltoe ulcer

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Abstract

Introduction and Methods

One of the most devastating complications of diabetes is foot ulceration. One of the reasons why digital ulcers are very important is that they commonly precede limb amputations in diabetic patients upto 63.9% of the time. The early diagnosis, treatment and prevention is essential for prevention and management of diabetic ulcers and improve prognosis in the long term. In diabetes mellitus, the neuropathy is responsible for the decrease in the sensitivity which in turn lead to the loss of the reflexive protective sensation and in turn leads to toe deformities. The aim of the study is to study the outcome and efficacy of flexor tenotomy procedure in the recurrence of diabetic distal toe ulcer and in the rate of healing of distal toe ulcer.From July 2017 to June 2019, a Prospective Single Center Study was done among 50 patients admitted with with diabetic neuropathic ulcer in the distal end of toes and all patients with toe deformity in GMKMC hospital. The following data was collected using a structured questionnaire: age, demographic characteristics, socio economic status, patients complaints and duration of complaints. A detailed clinical examination was done. Systemic examination and basic investigations were done.

Results

Patients between the age groups of 18 years and 60 years were included who were affected with diabetic neuropathic ulcer in the distal end of toes, presented with toe deformity and classified as Wagner's classification 1 and 2. The mean age is 52 years.Among fifty patients, majority of them were males (n=33, 66%) while the rest were females

(n=17, 34%). All patients in the study group were diagnosed with diabetes mellitus. The mean duration of type II DM was 36.06 months.Out of fifty patients, 68% (n=34) of them were on regular treatment while the remaining 32% (n=16) were on irregular treatment. The mean duration of ulcer was 38.35 days. Infection was present in 40% (n=20) of the patients. Rest of them did not have any infection (n=30, 60%). All of the patients had deformity.The neuropathic symptoms was present in 88% (n=44) of them .The x-ray of the affected limb showed the presence of osteomyelitis in 8% (n=4) of the cases. ABI was normal in all the patients. Flexor tenotomy was done in all the fifty patients. 80% (n=40) of them had a therapeutic tenotomy while 20% (n=10) of them had a prophylactic Tenotomy. The mean percentage reduction of ulcer in first week is 28.5% The mean percentage reduction of ulcer in second week is 58.3%, in first month is 88.08% The mean percentage reduction of the ulcer in second month is 96.87% .In the group that had prophylactic tenotomy, all 10 patients did not have any ulcer during the study period.There were no complications at week one and week two. At the end of month one; there was one amputation and three transfer ulcers in the therapeutic group. Follow up 2-Month, 4-Month, 6-Month and One Year showed an increase in cure rates after flexor tenotomy in the therapeutic group and no ulcer in the prophylactic group. At the end of one year, the cure rate was 90% (n=36) with cured and transfer ulcers being 7.5% (n=3) and 2.5% (n=1) amputation

Discussion and Conclusion

Diabetic foot ulcers are caused by a number of factors related to the architecture of the bone of foot, peripheral neuropathy and atherosclerotic peripheral arterial disease.All these factors lead to the infection of the foot .The stiffness of the ligaments in the disease is due to the

nonenzymatic glycation. In diabetes mellitus, the neuropathy is responsible for the decrease in the sensitivity of the foot and thereby lead to the loss of the reflexive protective sensation which cause the mechanical stress to the otherwise normal foot during ambulation and lead to the complications .The insensate foot leads to poor reflexes during gait or weight bearing. Subsequently, there is more stress to the pressure points. Also, it results in callus formation. Long standing callus leads to tissue trauma and resultant ulcer[6].One of the main treatment goal is to off-load pressure from the sites of ulcer. This off-loading helps in the reduction of stress, pressure and callus formation enabling an environment for the healing of ulcer. Continual treatment helps in preventing the recurrence of the ulcer. The study shows that percutaneous tenotomy is very effective in the management of diabetic foot ulcers with better outcome and prognosis with better healing rates and lesser recurrence rates.

Introduction

One of the most devastating complications of diabetes is foot ulceration. The most common sites are;

a) Dorsum

b) Apices

c) Plantar aspects of toes

The ulcers that occur on the 1-5 toes comprise anywhere between 43 and 55.5% of all reported foot ulcers[1,2]. Other sites are also involved like forefoot, mid-foot and heel ulcers where the presentation is larger than the digital ulcers[3]. One of the reasons why digital ulcers are very important is that they commonly precede limb amputations in diabetic patients upto 63.9% of the time[4]. The early diagnosis, treatment and prevention is essential for prevention and management of diabetic ulcers and improve prognosis in the long term.

Diabetic foot ulcers are caused by a number of factors related to the architecture of the bone of foot, peripheral neuropathy and atherosclerotic peripheral arterial disease. These are the common manifestations of people affected by diabetes[17]. All these factors lead to the infection of the foot in advanced conditions and is known to be a common factor for diabetes related hospitalisations and amputation[18]. The stiffness of the

ligaments in the disease is due to the nonenzymatic glycation. In otherwise normal individuals, too much pressure and stress on the pressure points in the foot may lead to the reflexive change in position due to responsive nerve endings.

But, in diabetes mellitus, the neuropathy is responsible for the decrease in the sensitivity of the foot and thereby lead to the loss of the reflexive protective sensation and also to the coordination errors in the foot and leg muscles. All these factors together cause the mechanical stress to the otherwise normal foot during ambulation and lead to the discussed complications[19]. This is the reason why diabetes happens to be the most common non-traumatic cause of foot amputations with around 5% of diabetic population developing it and approximately 1% requiring amputation[20].

The assessment of diabetic foot ulcer requires a multipronged approach[21];

a) Physical examination of the extremity affected by the ulcer
    - Examination of ulcer
    - Condition of the extremity
b) Vascular insufficiency assessment

The depth of involvement of soft tissue and bone dictates the staging of the disease[22-24]. Then routine investigations of complete blood cell count,

serum creatinine, serum glucose and glycosylated haemoglobin. The management is very elaborate starting from the offloading of weight from the foot by using appropriate footwear[25,26]. Saline dressings are required to maintain a moist environment[27], do debridement and put on antibiotic therapy is there are any infections[28,29]. The diabetic control along with the monitoring and correction of the insufficiencies of the peripheral arteries are also important.

Toe deformities are also common in the long term like 'hammer' and 'claw' toes in the evolution of diabetic foot ulcers. These deformities are common when there are comorbities and complications like neuropathy and peripheral vascular disease[5]. The problem with diabetic foot is that the nerve endings in the affected foot becomes insensate. This leads to poor reflexes during gait or weight bearing. Subsequently, there is more stress to the pressure points. Also, it results in callus formation. Long standing callus leads to tissue trauma and resultant ulcer[6].

One of the main treatment goal is to off-load pressure from the sites of ulcer. This off-loading helps in the reduction of stress, pressure and callus formation enabling an environment for the healing of ulcer. Continual treatment helps in preventing the recurrence of the ulcer[7].

Traditional methods of treatment for off-loading has been conservative with[8-10];

- regular debridement of the corn and callus (superficial skin lesions)
- deflective padding
- insoles
- therapeutic foot wear

Studies are inadequate in suggesting the efficacy and effectiveness of such conservative treatments. Further, poor patient adherence confounds the actual effectivness of the methods suggested above. Studies have shown that pressure relieving footwear has poor adherence as with other wearing removable offloading devices[11].

Surgical interventions might reduce the risk and recurrence of diabetic foot ulcers especially in patients with peripheral neuropathy[12]. Infections and healing rates in diabetic foot ulcers may be altered by Minimally-invasive surgical procedures[13].

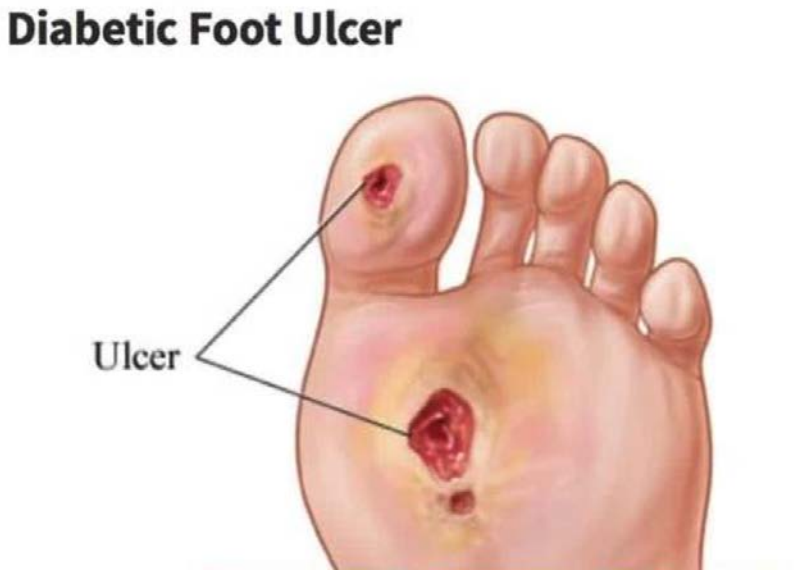
For flexible toe deformities, flexor tenotomy procedure can be advocated for hallux and the lesser toes[14]. Flexor tenotomy procedure is done under local anesthetic where a plantar incision is made to transect flexor digitorum/hallucis longus tendon[15].

There are surgeons who also prefer to release the tendon of flexor digitorum/hallucis brevis[16]. The aim of this procedure is to release the tendon of flexor digitorum brevis/longus to make the position of the toe straighter. This leads to the reduction in the amount of pressure, stress and subsequent callus and ulcer formation. Ulcer at the toe apices are the most benefitted. The procedure is usually done as an outpatient basis.

Review of Literature

An overview of the disease

Diabetic foot ulcers are caused by a number of factors related to the architecture of the bone of foot, peripheral neuropathy and atherosclerotic peripheral arterial disease. These are the common manifestations of people affected by diabetes[17]. All these factors lead to the infection of the foot in advanced conditions and is known to be a common factor for diabetes related hospitalisations and amputation[18]. The stiffness of the ligaments in the disease is due to the nonenzymatic glycation. In otherwise normal individuals, too much pressure and stress on the pressure points in the foot may lead to the reflexive change in position due to responsive nerve endings.



But, in diabetes mellitus, the neuropathy is responsible for the decrease in the sensitivity of the foot and thereby lead to the loss of the reflexive protective sensation and also to the coordination errors in the foot and leg muscles. All these factors together cause the mechanical stress to the otherwise normal foot during ambulation and lead to the discussed complications[19]. This is the reason why diabetes happens to be the most common non-traumatic cause of foot amputations with around 5% of diabetic population developing it and approximately 1% requiring amputation[20].

The assessment of diabetic foot ulcer requires a multipronged approach[21];

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monitoring and correction of the insufficiencies of the peripheral arteries are also important.

The systematic review for diabetic foot ulcers

The management and understanding of the foot infections has dramatically increased in the last three decades and has been concluded that a multidisciplinary approach is required. A systematic search in literature for diabetic foot infections from from January 1960 till June 2019 shows how the knowledge on the diabetic infections, their etiopathogenesis and management has progressed with improved insights in the field of microbiology, surgery, pathology and management. Yet, the application of this knowledge is not apt in clinical practice. Evidence-based guidelines with multidisciplinary teams are the need for the day. The treatment of DFI using antibiotics, conservative and surgical managements are rapidly seeing transformations due to the upcoming technological innovations. The increasing longevity of the patients with diabetes would bring in more incidence of DFI and thereby require more insight and understanding for the management of the patients. The following figure shows how the knowledge of DFI varies within a span of 30 years.

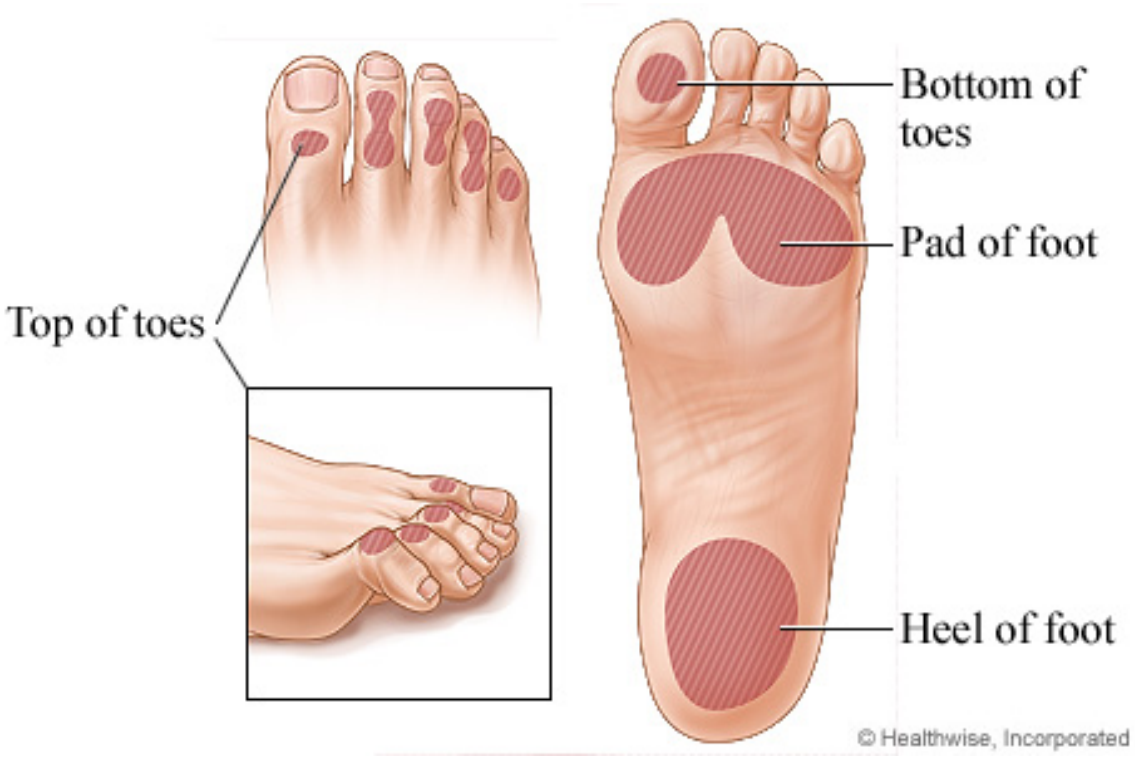
Table 1
Key changes in the knowledge and management of diabetic foot infections in the last 30 years–summary of the authors' views

Research field	1985	2015
Pathogens	Methicillin-susceptible *Staphylococcus aureus*, streptococci, *Enterobacteriaceae*	More multidrug-resistant organisms (MRSA, ESBLs) Predominance of Gram-negative pathogens in (sub)tropical climates
Microbiological diagnosis	Standard cultures, usually of swab specimens	Aerobic and anaerobic cultures of tissue specimens (soft tissue and bone) Molecular microbiology (e.g., PCR) Metagenomics
Imaging	Plain X-rays; scintigraphy (bone, leukocyte scans)	MRI; SPECT/CT; PET/CT
Antibiotic agents	Penicillins; 1st to 3rd generation cephalosporins; some 2nd generation fluoroquinolones	4th/5th generation cephalosporins; carbapenems; 3rd/4th generation fluoroquinolones; linezolid; daptomycin
Route of administration and site of treatment	Initial (sometimes prolonged) intravenous administration, usually in hospital	Mostly oral (sometimes after a brief intravenous course), even in the presence of vascular disease or osteomyelitis; some topical; outpatient except for severe infections or complex treatments
Spectrum of antibiotic therapy	Relatively broad (directed at Gram-positive and Gram-negative pathogens)	Very broad empiric therapy for severe infections; more targeted for mild/moderate infections and for definitive therapy
Duration of antibiotic therapy	Many weeks for soft tissue infections; ≥6–12 weeks for bone	1–2 weeks for soft tissue infections; 4–6 weeks for osteomyelitis
Surgical approach	Aggressive (ablative) therapeutic surgery; inpatient treatment	More conservative (tissue sparing) therapeutic (even for osteomyelitis) and preventive surgery; corrective surgery; often in outpatient facilities and specialized diabetic foot centres
Revascularization	Open vascular surgery	More percutaneous angioplasty and distal bypasses, including infragenicular
Management	Mostly individual, empirical approaches	Clinical guidelines based on systematic reviews; multidisciplinary teams, especially including podiatry; clinical pathways; some behavioural sciences
guidelines	Individual recommendations and practices on the hospital level	national guidelines; validation of guidelines
Adjunctive treatments	Stimulation with growth factors; platelet-rich products; larval biotherapy (maggots)	Hyperbaric oxygen therapy; granulocyte-stimulating factors; research in stem cell and bacteriophage therapies; microbiome concepts
Dressing	Simple dressings, with separate use of disinfection agents	More hydrofibre and silver-containing dressings; studies with topical antibiotics embedded in dressings
Scientific publications	Mostly case series	More prospective randomized trials, multicenter studies, and evidence-based (Cochrane) meta-analyses

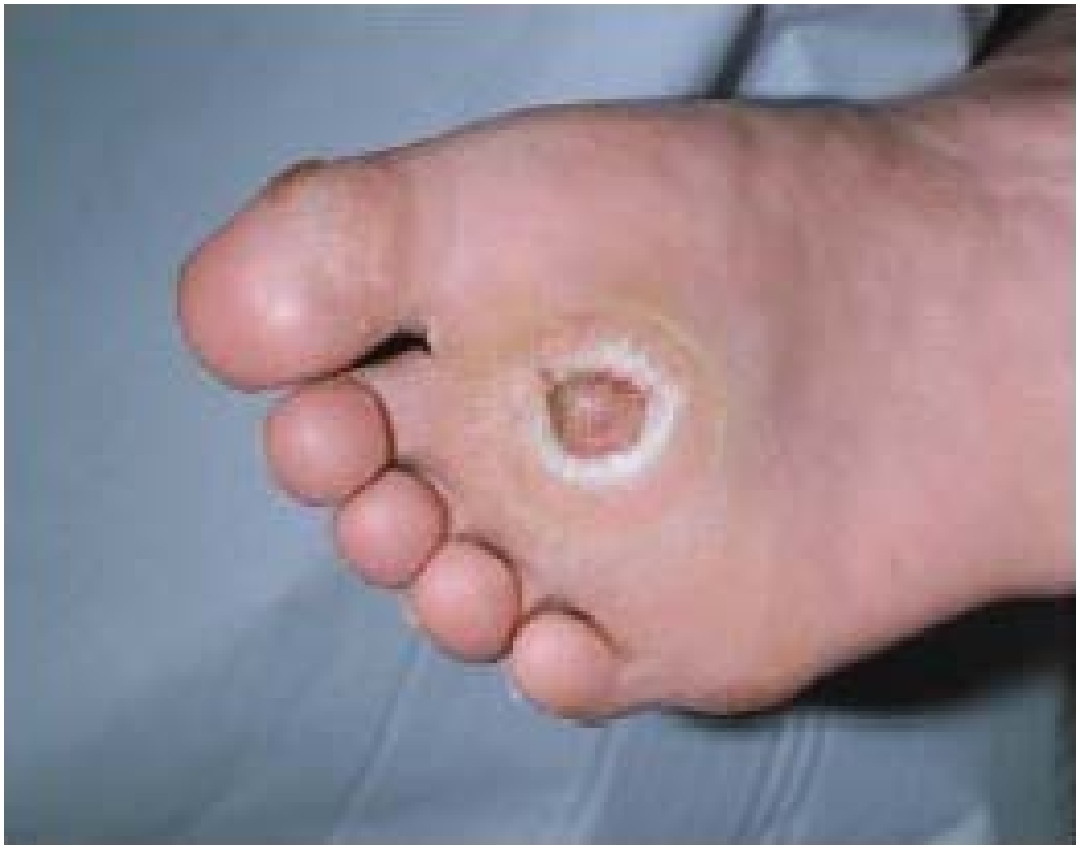
ESBL, extended-spectrum beta-lactamase; MRI, magnetic resonance imaging; MRSA, methicillin-resistant *Staphylococcus aureus*; PET/CT, positron emission tomography/computed tomography; SPECT/CT, single photon emission computed tomography/computed tomography.

Incidence

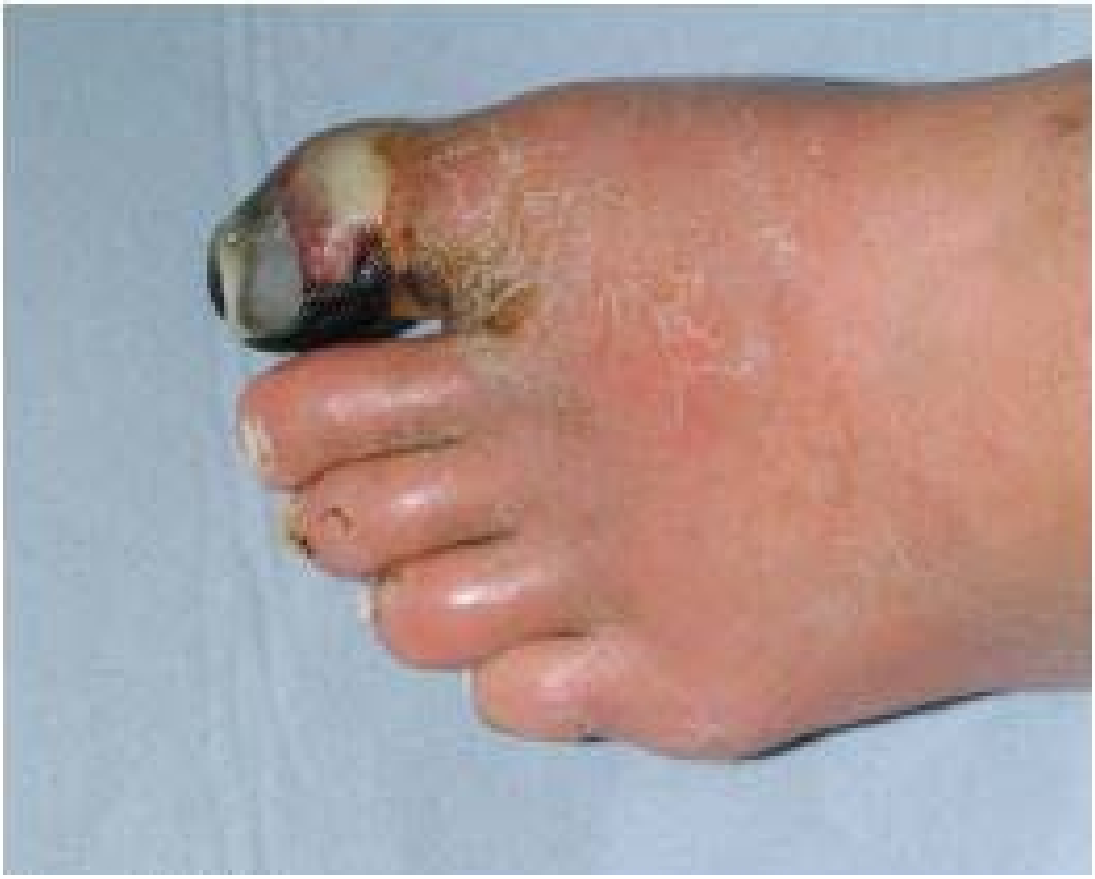
With a known case of Diabetes Mellitus, the life time risk of diabetic foot infection and ulcer is 25% while an annual risk is 4%[30]. The presentation is often cellulitis and post-traumatic[31]. It is often seen as a result of progressive peripheral neuropathy that leads to ulcerations[32].



The ulcers that occur on the 1-5 toes comprise anywhere between 43 and 55.5% of all reported foot ulcers[33,34]. Other sites are also involved like forefoot, mid-foot and heel ulcers where the presentation is larger than the digital ulcers[35]. One of the reasons why digital ulcers are very important is that they commonly precede limb amputations in diabetic patients upto 63.9% of the time[36]. The early diagnosis, treatment and prevention is essential for prevention and management of diabetic ulcers and improve prognosis in the long term[37].



Toe deformities are also common in the long term like 'hammer' and 'claw' toes in the evolution of diabetic foot ulcers. These deformities are common when there are comorbities and complications like neuropathy and peripheral vascular disease[38]. The problem with diabetic foot is that the nerve endings in the affected foot becomes insensate. This leads to poor reflexes during gait or weight bearing. Subsequently, there is more stress to the pressure points. Also, it results in callus formation. Long standing callus leads to tissue trauma and resultant ulcer[39,40].



Pathophysiology

The initiation, development and progression of the disease is multifactorial. From a clinician’s point of view, it is necessary to understand the etiopathogenesis and the pathophysiology for deciding the management. Uncontrolled diabetes leads to the thickening of the ligaments of the foot leading to deformities. This in addition to the neuropathy leads to excessive pressure and stress to the foot. Continual pressure leads to callus formation and subsequent ulceration. The following images show the pathophysiology of the disease. Toe deformities are also common in the long term like 'hammer' and 'claw'

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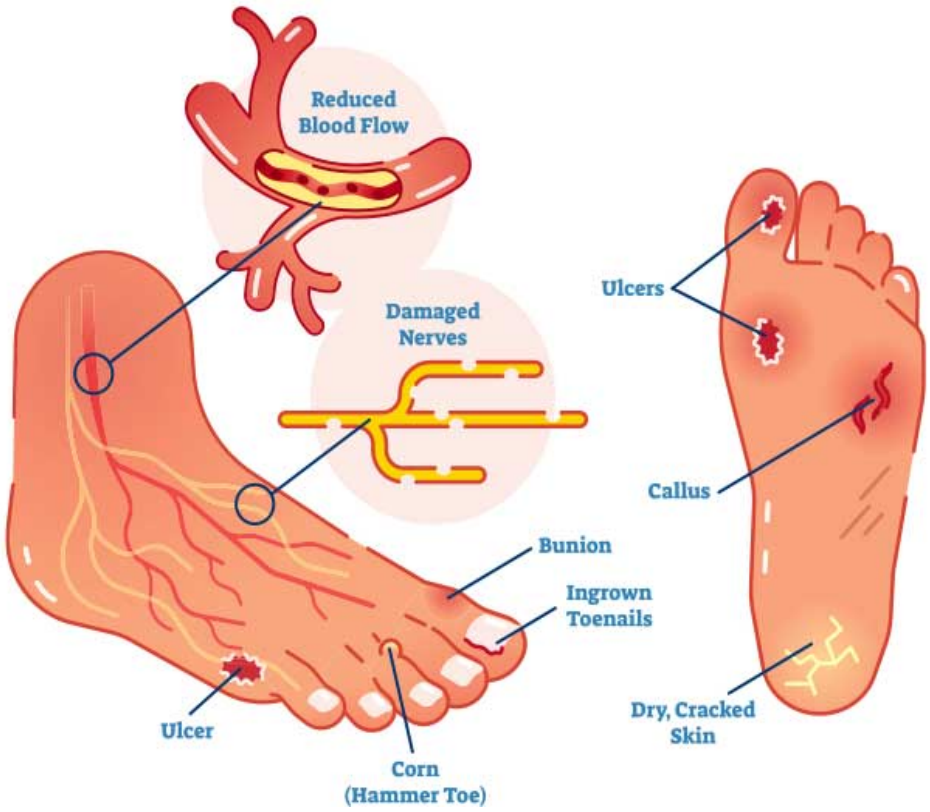

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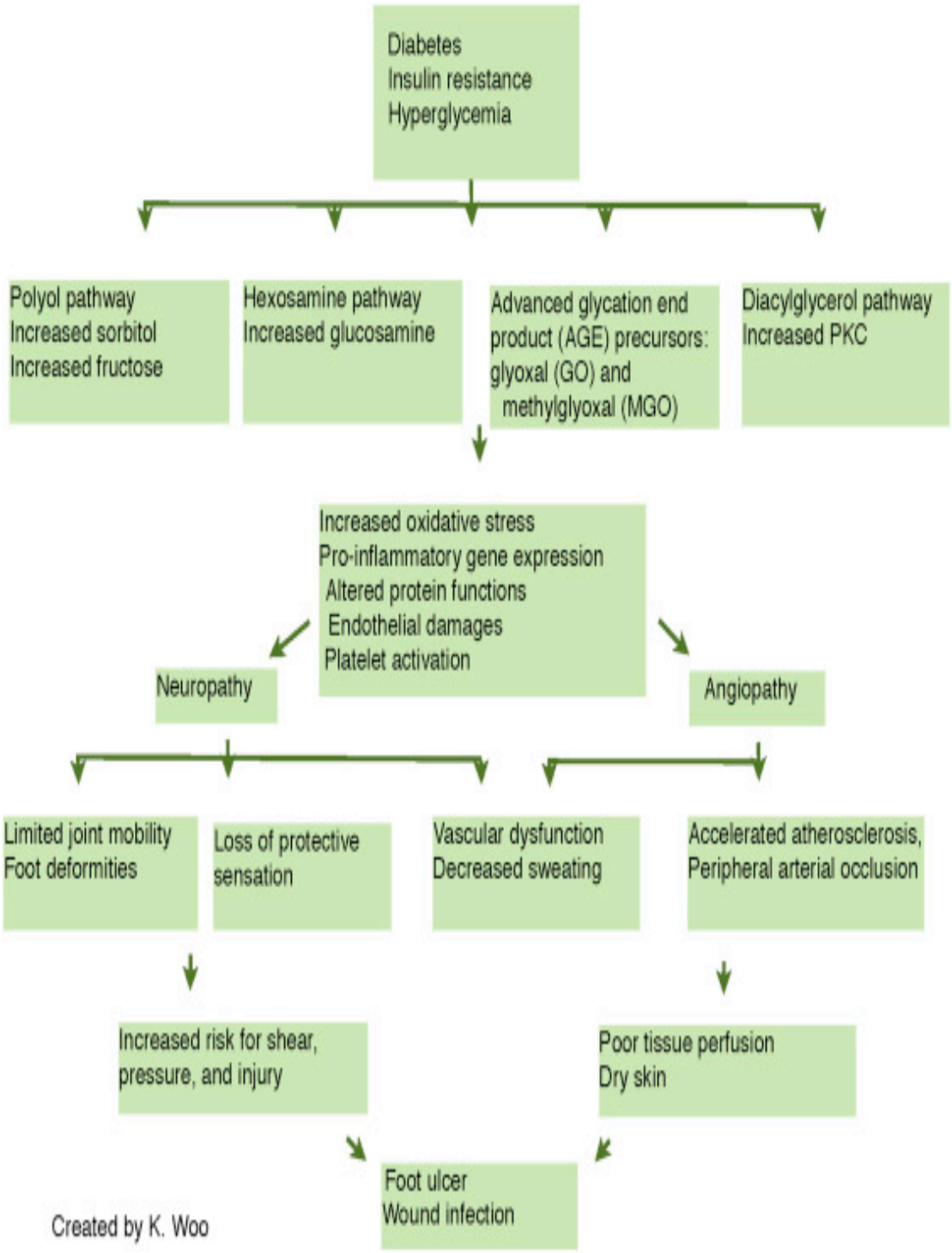
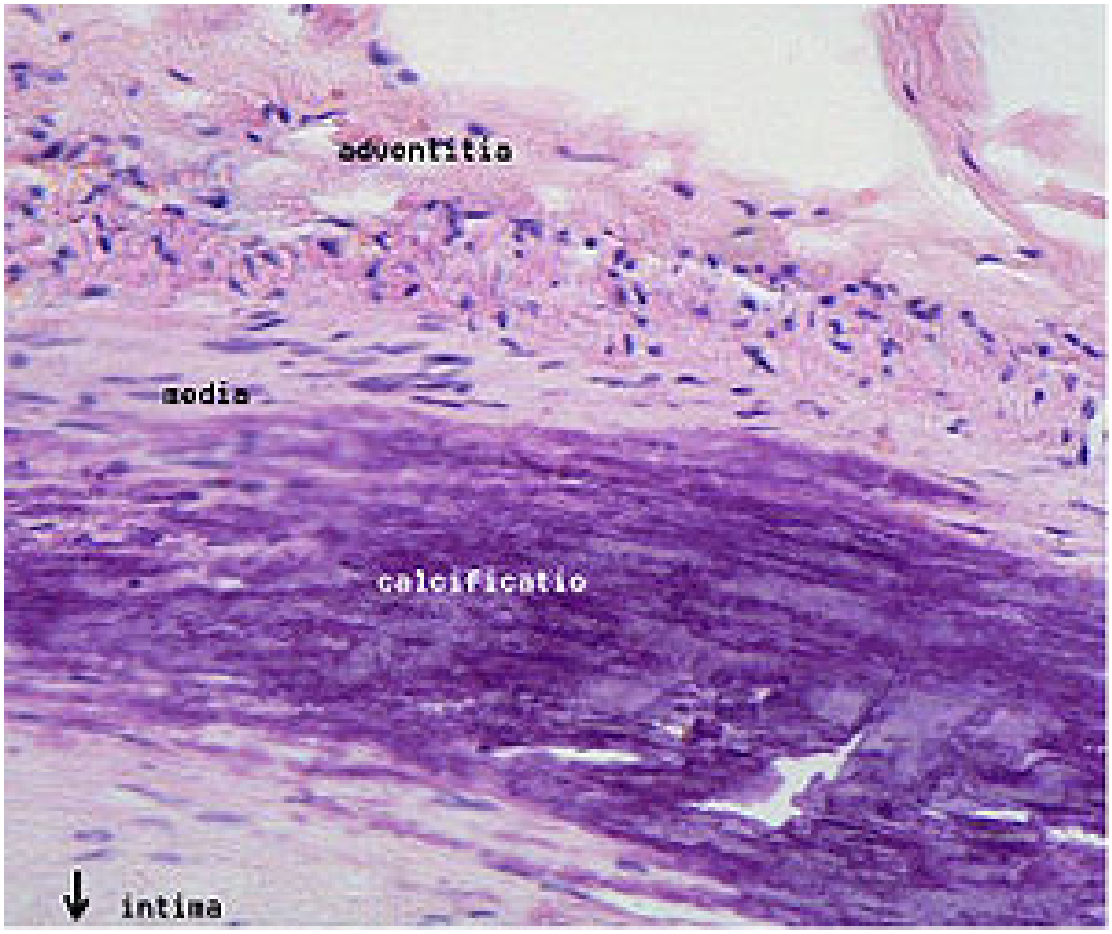


The image above shows how diabetes, insulin resistance and hyperglycemia initiate and maintain the foot ulcer and subsequent wound infection.

Atherosclerosis related to Diabetes

There is a thickening of the capillary basement membrane with endothelial proliferation and arteriolar hyalinosis in diabetes mellitus. The incidence of Mönckeberg sclerosis where there is thickening and calcification of the media of the arteries is high in diabetes.



How this calcification contributes to the progression of the disease is still unclear. The involvement ofinfrapopliteal segments of the arteries is common in the diabetic population. When there is an infected ulcer nearby, the collateral circulation is completely lost and therefore leads to

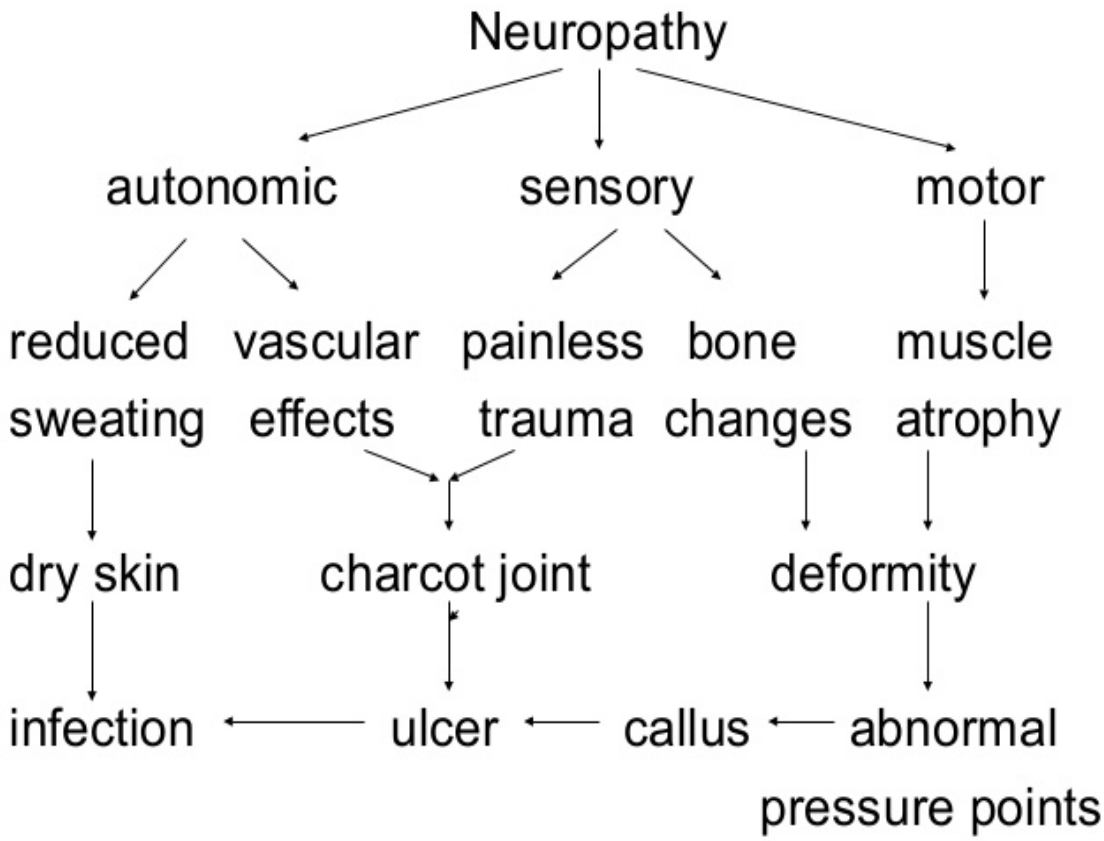
gangrene. A number of metabolic abnormalities are noted in patients with DM which are known to cause the disease;

a) High LDL and VLDL
b) Low HDL
c) Elevated plasma von Willebrand factor
d) Inhibition of prostacyclin synthesis
e) Elevated plasma fibrinogen levels
f) Increased platelet adhesiveness.

Diabetic peripheral neuropathy[41]

A multifactorial pathway is conceived in the pathophysiology of diabetic peripheral neuropathy. There is an occlusion of the arteries due to the following vascular disease;

a) Occlusion of vasa nervosum
b) Endothelial dysfunction
c) Deficiency of myoinositol-altering myelin synthesis
d) Decreasing Sodium-potassium adenine triphosphatase (ATPase) activity
e) Chronic hyperosmolarity (leading to edema of nerve trunks)
f) Increased sorbitol and fructose.



All these factors lead to the loss of sensation in the foot and concomitant cyclical stress. Most of the injuries does unnoticed and leads to structural deformities of the foot namely;

a) Hammertoes

b) Bunions

c) Metatarsal deformities

d) Charcot foot

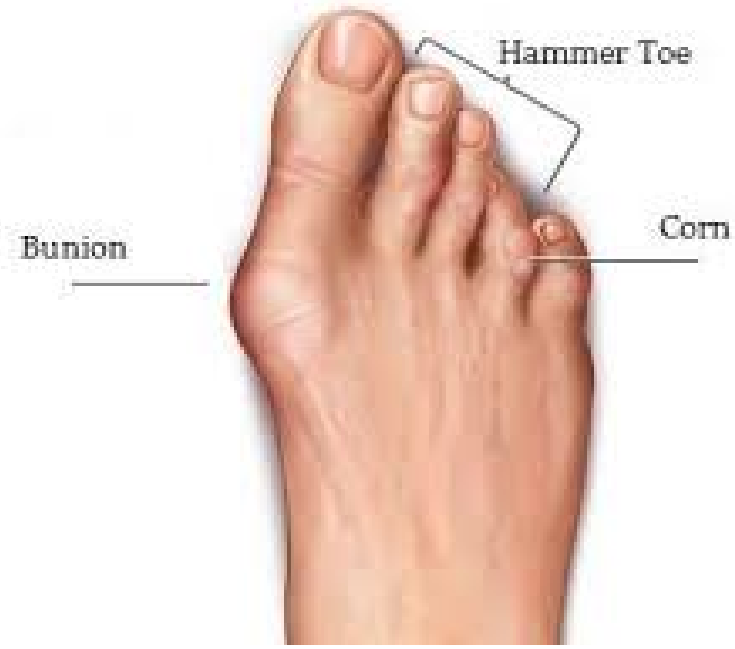


Image : Hammer Toe and Bunion

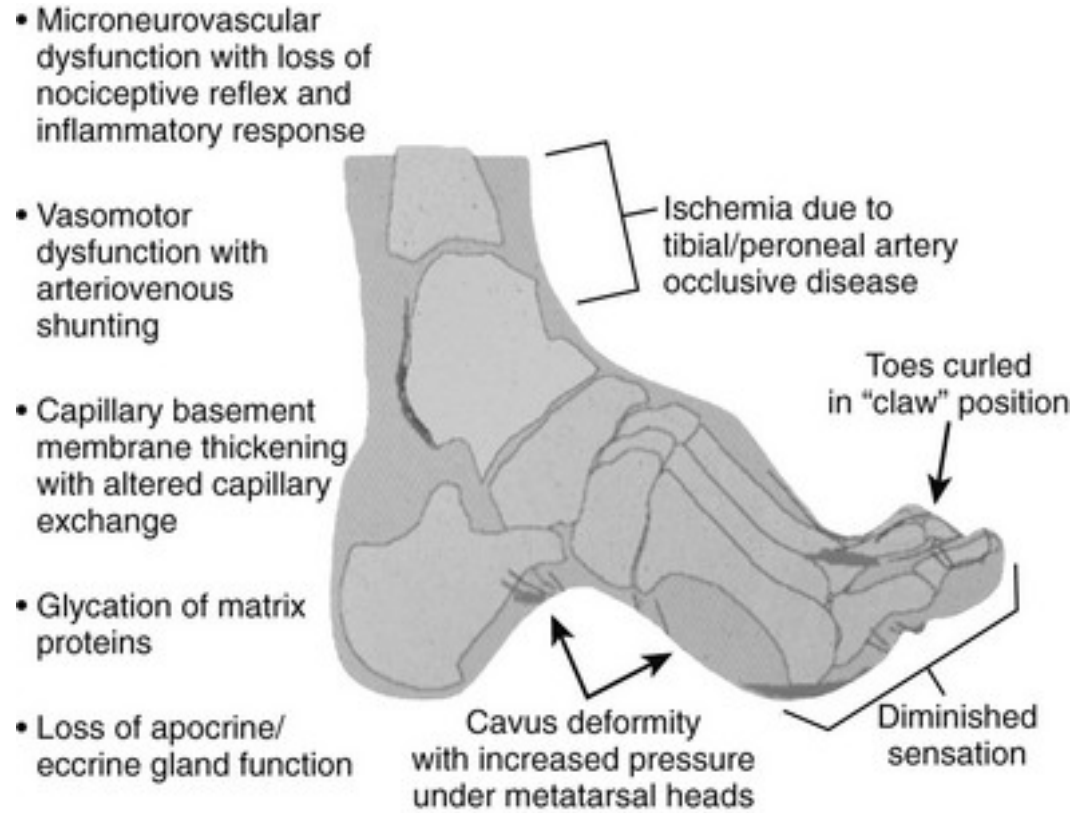


Image: Metatarsal Deformity

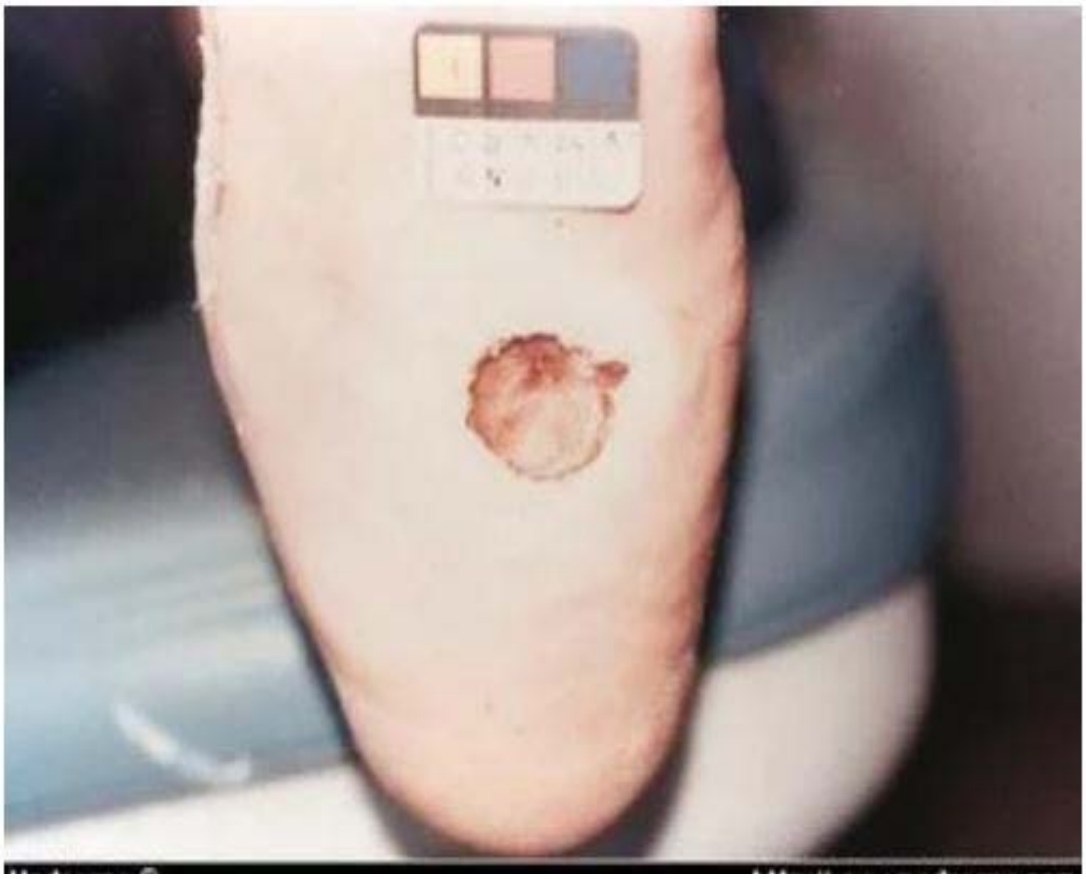


Image: Charcot Deformity with ulcer of plantar midfoot

These deformities lead to stress and breakdown of tissues. Sometimes unnoticed cold, heat or pressure from shoes, injuries from sharp objects may lead to blistering, wounds and ulcerations. When all these factors lead to poor arterial blood flow, the risk of losing limb and subsequent amputation is high.

Etiology

The following are known to cause diabetic ulcers;

a) Neuropathy[42]
b) Arterial disease[43]
c) Pressure[44]
d) Foot deformity[45]

The peripheral neuropathy in diabetes is seen in 60% of the patients with diabetes and in 80% of the patients with diabetic foot ulcers thus making it the one single most significant factor in the pathogenesis of the disease. Tissue mechanics is also studied to understand how it is associated with DFUs. The heal pad is known to be stiffer in patients with diabetes than those who are not having DM[46]. High thickness of plantar soft tissue with lower stiffness of the soft tissues in the first metatarsal head is known to contribute to the DFUs[47]. This indicates that the calculation of risk for diabetic foot ulcers should also focus on the anatomy of the foot. People with higher risk are those who have flat foot and disproportionate distribution of weight bearing across the surface of the foot.

Epidemiology

A study by Zhang et al in 2017 to understand the international epidemiology of diabetic foot ulceration shows the following features[48];

a) Global prevalence=6.3% (95%CI: 5.4–7.3%)
b) Gender distribution= higher in males(4.5%, 95%CI: 3.7–5.2%) than females (3.5%, 95%CI: 2.8–4.2%)
c) Incidence high in DM-II (6.4%, 95%CI: 4.6–8.1%) compared to DM-I (5.5%, 95%CI: 3.2–7.7%)
d) Region-wise prevalence and distribution
    1. North America=highest prevalence (13.0%, 95%CI: 10.0–15.9%)[49]
    2. Oceania= lowest (3.0%, 95% CI: 0.9–5.0%)[50]
    3. Asia= 5.5% (95%CI: 4.6–6.4%)[51]
    4. Europe= 5.1% (95%CI: 4.1–6.0%)[52]
    5. Africa=7.2% (95%CI: 5.1–9.3%)[53]
    6. Australia (lowest)= 1.5% (95%CI: 0.7–2.4%)[54]
    7. Belgium= highest prevalence (16.6%, 95%CI: 10.7–22.4%)[55]
    8. Canada=high prevalence (14.8%, 95%CI: 9.4–20.1%)[56]
    9. USA=high prevalence (13.0%, 95%CI: 8.3–17.7%)[57].
e) Age distribution: Older age group were more affected[58]
f) BMI: Lower BMI was correlated to DFU

g) Duration of diabetes: Longer duration was positively correlated with the incidence of the disease

h) Associated comorbid conditions;

   1) Hypertension

   2) diabetic retinopathy

   3) smoking history

Wound Classification and Staging[59, 60]

There are a number of classifications have been proposed and tested. The widely used classification system is the Wagner Diabetic Foot Ulcer classification.

Ulcer grading	Description
Grade 0	No ulcer but high-risk foot
Grade 1	Superficial ulcer
Grade 2	Deep ulcer, no bony involvement or abscess
Grade 3	Abscess with bony involvement (as shown by X-ray)
Grade 4	Localized gangrene e.g. toe, heel etc
Grade 5	Extensive gangrene involving the whole foot

Note: Grade 1–3 ulcers are termed *non-gangrenous ulcers* and Grade 4 and 5 ulcers are termed *gangrenous ulcers*

Image: Wagner Diabetic Foot Ulcer Classification

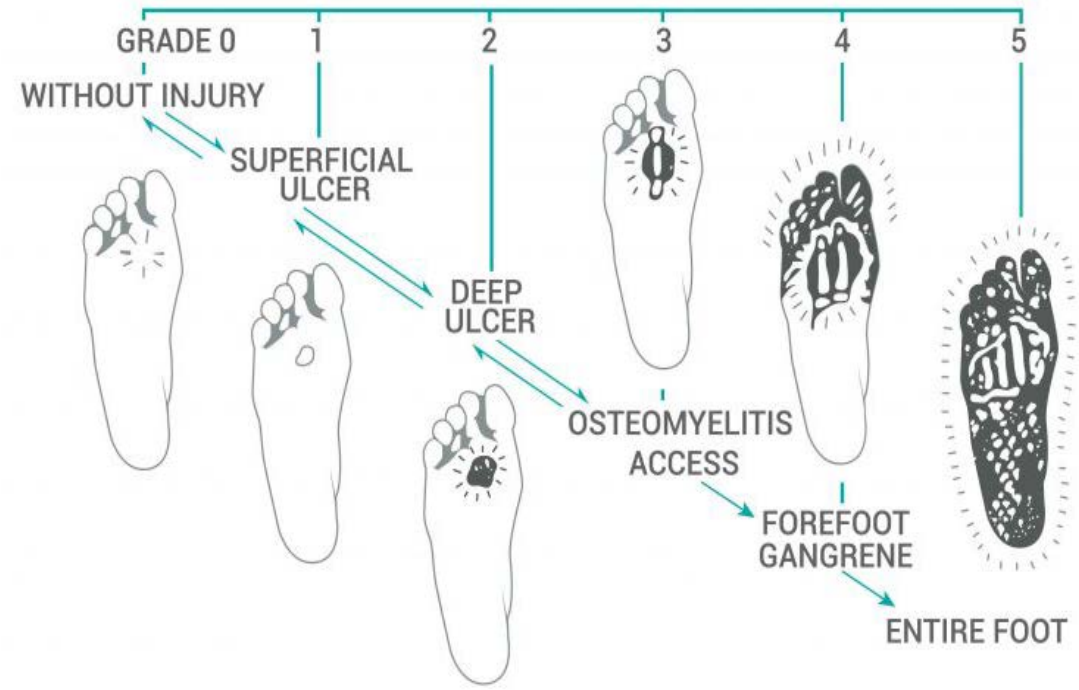


Image: Wagner Diabetic Foot Ulcer Classification

The Wagner classification system is mainly based on the depth of the wound. The University of Texas added one more dimension of ischemia and infection. The following image shows the University of Texas Classification system[61].

University of Texas Diabetic Wound Classification System				
Stage	Grade			
	0	I	II	III
A (no infection or ischemia)	Pre- or post-ulcerative lesion completely epithelialized	Superficial wound not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	Infection and ischemia	Infection and ischemia	Infection and ischemia	Infection and ischemia

Image: University of Texas Diabetic Wound Classification System

Prognosis

The mortality and morbidity associated with diabetic foot ulcers are more related to the comorbid conditions and the severity of the occlusion of the arteries mainly the large vessels. The health quality assessment showed the following risk factors[62,63];

1) existence of pain
2) CRP protein level >10 mg/L
3) ulcer size >5 cm^2
4) ankle-brachial index <0.9
5) high glycosylated hemoglobin level
6) body mass index >25 kg/m^2.

If the treatment is delayed, the risk of losing the limb to amputation is high[64].The 5-year risk of a contralateral amputation is around 50%[65]. The

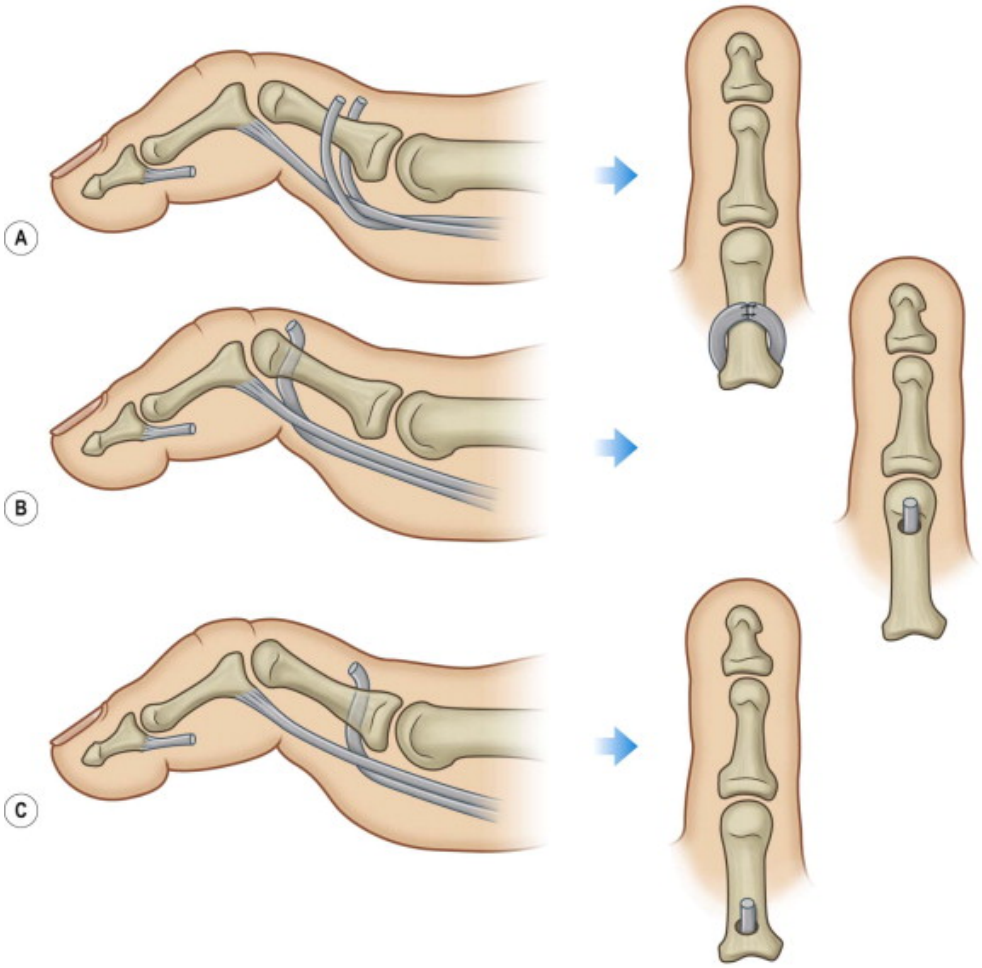
recurrence rate is around 66% even in case of successful treatment and recurrent amputation is 12% for patients with neuropathy[66].

The main reason for premature death in DFU is ischemic heart disease (62.5%) [67]. One of the late complications with significant morbidity is the DFU-ISI (invasive systemic infection associated with the ulcer). The most commonly associated causative organism is the methicillin-resistant *Staphylococcus aureus* (MRSA).

Flexor tenotomy

Background

Tenotomies are not rare in foot and ankle surgeries for many decades. They were performed either alone in cases of tendon contractures or in combination with bone surgeries when the bones were involved. Successful digital surgeries have always depended on tenotomies[68-71]. The minimally invasive technique of tenotomy was first reported by McGowan[72]. As the search for less invasive procedures began, surgeons started moving towards the percutaneous procedures for ankle and foot deformities especially for club foot deformities[73-75].



The same technique has found importance in treating Achilles tendonopathies[76,77]. Vertical talus deformity is the latest addition in this list[78,79,80]. The first person to describe this technique is Minkowitz[81]. For club foot repair, he modified the Ponsetti method by using a large gauge needle for the lengthening of the Achilles tendon percutaneously.

Method

This can be performed as an out-patient procedure using a local digital block.

a) Using a 15 blade, a small incision is done at the flexor crease of the distal or proximal interphalangeal joint

b) The tendon is then transected

c) The incision was then closed using simple interrupted sutures

d) Bandage the foot

e) Post-operatively, keep the surgical site clean and remove sutures at two weeks

The simplified technique uses an 18-gauge needle with no requirement for suturing.

a) At the desired tenotomy level, the 18-gauge needle is inserted

b) The sharp beveled edge is use to transect the longitudinal fibers of the tendon

c) For additional contracture release, incision of the joint capsule may be done

d) Bandage for splinting the toe in rectus position is applied

e) Steri-strip is used to keep the toe in position

f) This is continued for 1 to 2 weeks.

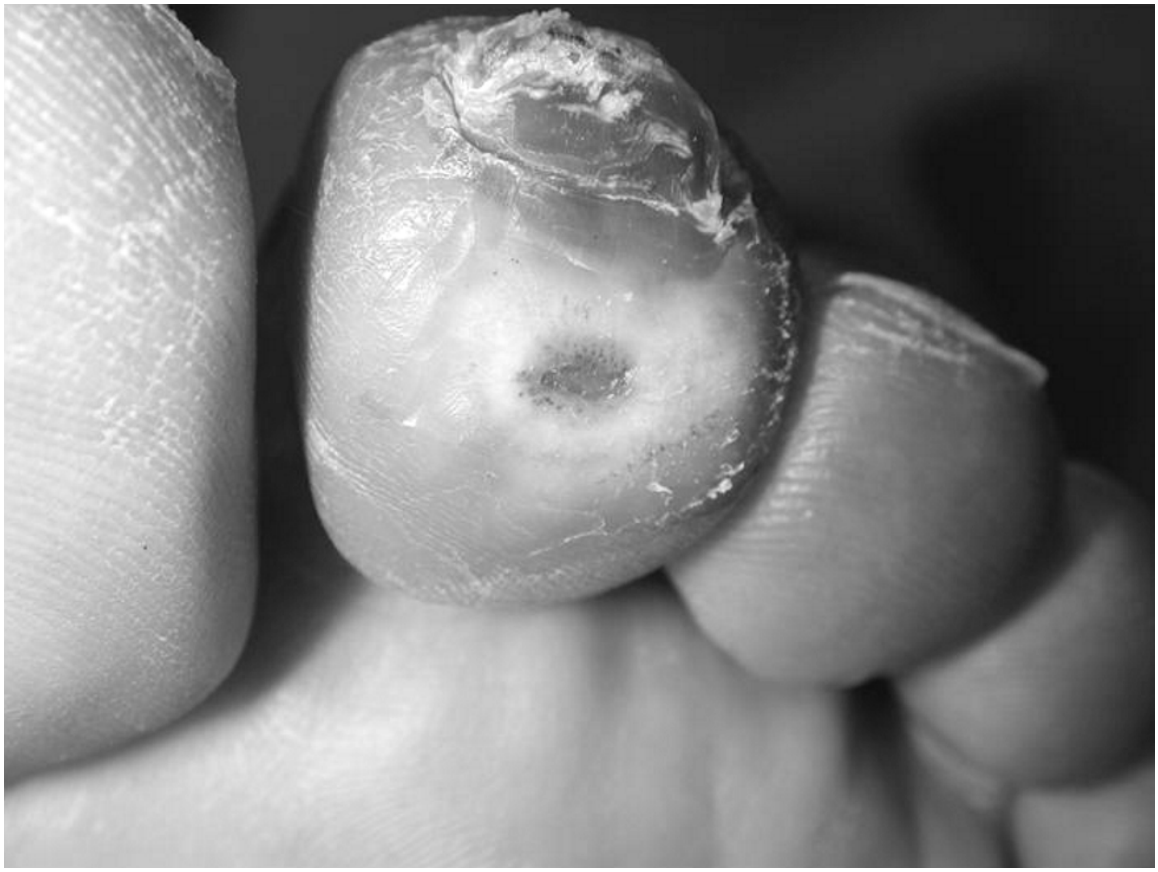


Image : Typical neuropathic ulcer at distal aspect of digit.

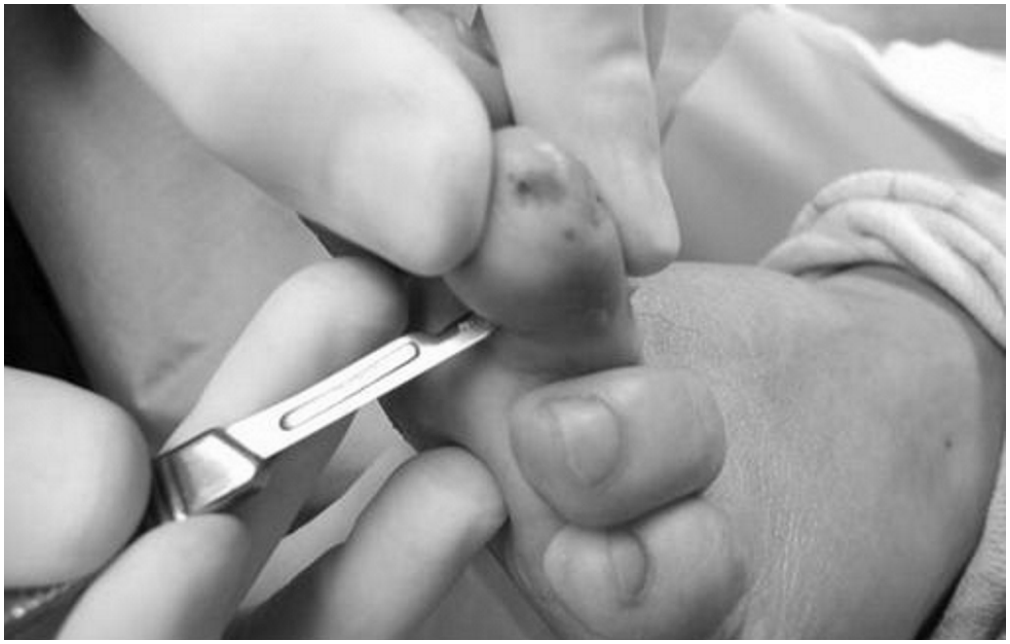


Image: Traditional tenotomy technique.

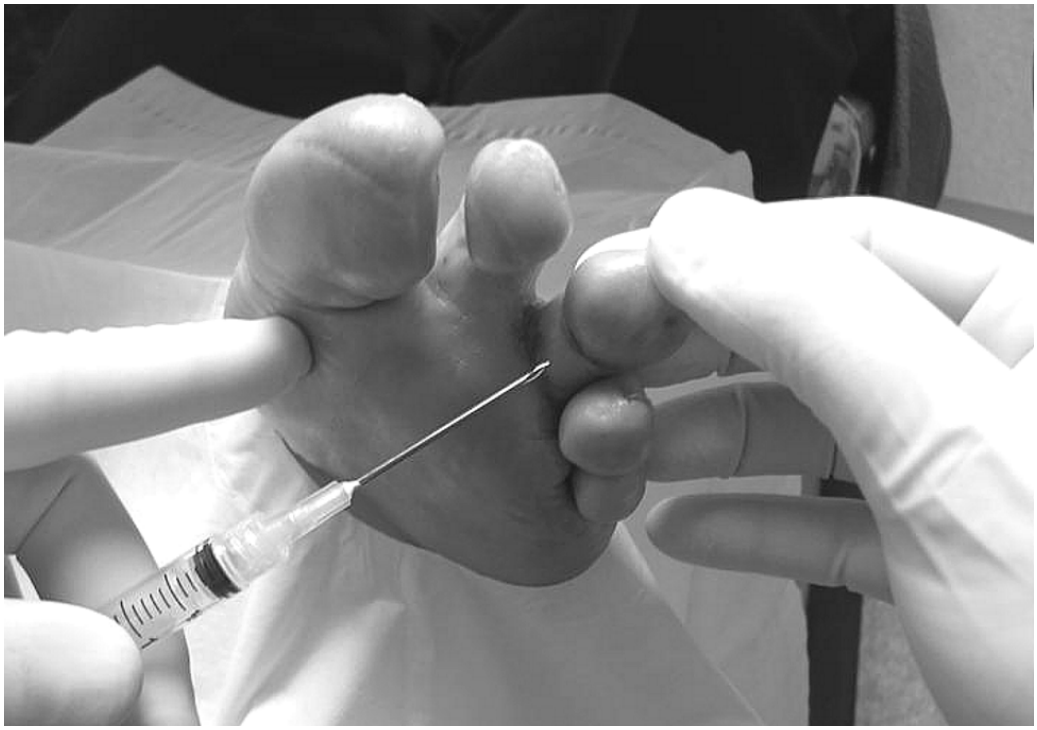


Image: 18-gauge needle percutaneous tenotomy technique.

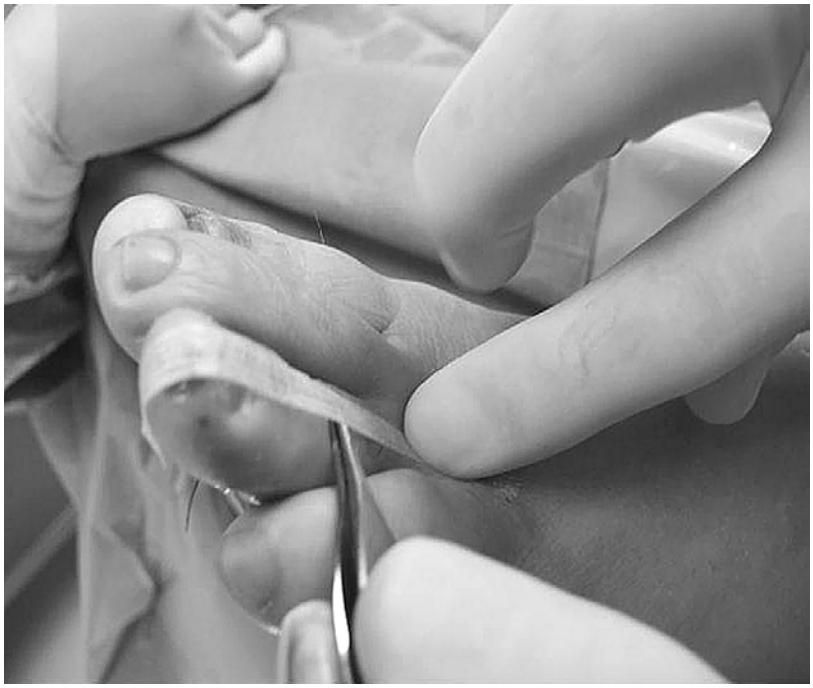


Image: Steri-strip splint to hold digit in rectus position.

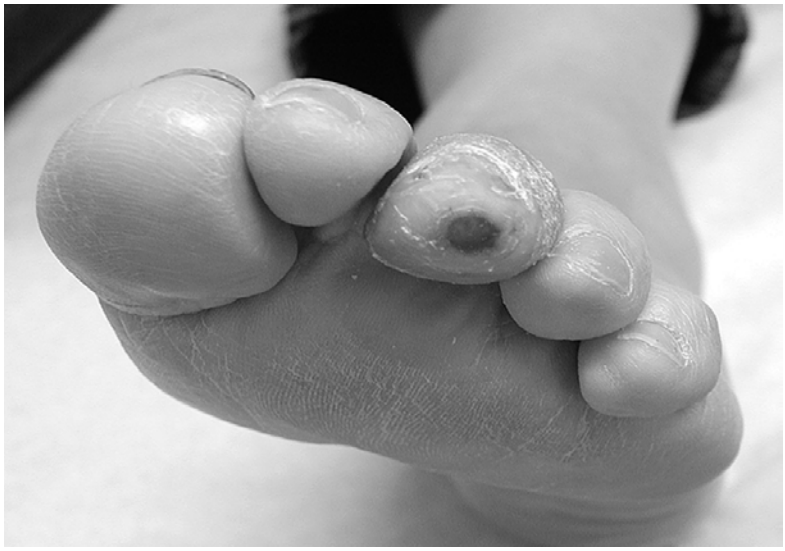


Image : Preoperative appearance of distal digital ulcer

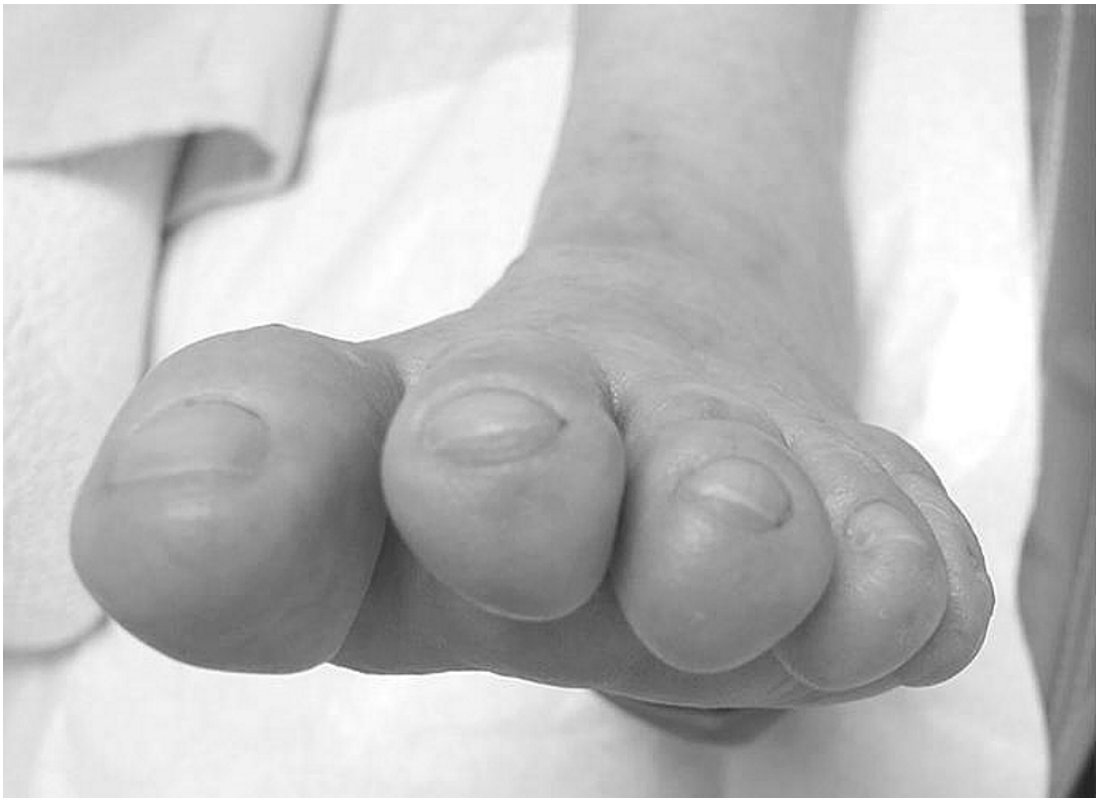


Image: Two year postoperative appearance with resolution of distal digital ulcer

Relevant Literature

The following studies have been chosen for comparison because of the similarity of sample size and the sample characteristics including methodology. The findings have been summarised in the following section.

	Kearney et al.[82]	Laborde[83]	Rasmussen et al.[84]	Tamir et al.[85]	Van Netten et al.[86]
Patients (No.)	48	14	16	55	30
Procedures (No.)	58	24	27	103	38
Age range (Years)	· Mean 68.1 ±2.3	· 40–81	· 37–91	· 48–89	· 42-93
		· Mean55	· Mean62.8	· Mean65	· Mean 69 ±12
Gender	· M:11,F: 37	· M: 7 F:11	· Insufficie ntly reported: prophylac tic and ulcerated patients reportedt ogether	· Notreporte d	· M: 17,F:16
Diabetes duration	· Notrep orted	· Notrepor ted	· Insufficie ntly reported: prophylac tic and ulcerated patients reportedt ogether	· Insufficient ly reported – patients receiving different interventio ns reportedtog ether	· Notrep orted
Incision location	Distal phalanx	Proximal portion of proximal phalanx	1 cm proximal to the web fold	Mid-portion of proximal phalanx	Mid - port ion of prox imal phal anx
Tendons transected	· FDL – 58toes	· FDL & FDB– · 10toe · FHL – 14Toes	· FDL & FDB– · 12Toes · FHL & FHB – 15Toes	· FDL – 87Toes · FHL – 16Toes	· FDL – 26Toes · FHL – 12Toes
Post-op offloading	• Immediat e weightbe	· Fullweight bearing · Post-op	· 2–3 days post-op hosp. immobiliza	Notreported	24 h offloadi ngplus

	aring	shoes/sandals/ extra depthshoe	tion Rocker bottom sandals + softinsoles		Pressure bandage
Returnappointment	Notreported	3–5daysthenweekly	1 weeks then as requireduntilhealed	1 week then regularlyuntilhealed	1 week thenregularly
Follow-up period (months)	Mean:28	•20–64 Average:36	2–48 Median:31	Minimum:5 Interquartilerange:16–29 Median 22	11-60 Mean=23 (S.D=11)

Need for Study

Studies are inadequate in suggesting the efficacy and effectiveness of such conservative treatments. Further, poor patient adherence confounds the actual effectivness of the methods suggested above. Studies have shown that pressure relieving footwear has poor adherence as with other wearing removable offloading devices[11]. Surgical interventions might reduce the risk and recurrence of diabetic foot ulcers especially in patients with peripheral neuropathy[12]. Infections and healing rates in diabetic foot ulcers may be altered by Minimally-invasive surgical procedures[13].

For flexible toe deformities, flexor tenotomy procedure can be advocated for hallux and the lesser toes[14]. Flexor tenotomy procedure is done under local anesthetic where a plantar incision is made to transect flexor digitorum/hallucis longus tendon[15].

There are surgeons who also prefer to release the tendon of flexor digitorum/hallucis brevis[16]. The aim of this procedure is to release the tendon of flexor digitorum brevis/longus to make the position of the toe straighter. This leads to the reduction in the amount of pressure, stress and subsequent callus and ulcer formation. Ulcer at the toe apices are the most benefitted. The procedure is usually done as an outpatient basis.

This study aims to fill the gap in the literature mainly from the perspective of the Indian population.

Materials and Methods

Aims and objectives of the study:

1. To study the outcome and efficacy of flexor tenotomy procedure in the recurrence of diabetic distal toe ulcer and in the rate of healing of distal toe ulcer
2. To describe the outcomes of prophylactic flexor tenotomy procedure in diabetic patients with toe deformity

Study design

Prospective Single Center Study

Place of study

GMKMC hospital

Study period

July 2017 to June 2019

Study population & Sampling Methodology

- Patients with diabetic distal neuropathic toe ulcers with Wagner's classification 1 and 2 and patients with claw or hammer toe deformity were selected
- Sample size=50

Inclusion criteria:

1. Age >18 yrs&< 60 yrs
2. All patients with diabetic neuropathic ulcer in the distal end of toes
3. All patients with toe deformity
4. All patients with Wagner's classification 1 and 2

Exclusion criteria:

1. Pregnant patients.
2. Age <15 years &>60 years
3. known cardiac, renal, respiratory disorders
4. Patients with Charcot's neuropathic joint
5. Patients with Wagner's classification 3,4 and 5

Methodology

The material for the study is taken from the cases admitted in the surgical ward of the Department of General Surgery, GMKMC Hospital who are with diabetic distal neuropathic toe ulcers with Wagner's classification 1 and 2.

1. A detailed history was taken
2. Examination was done
3. Systemic examination and basic investigations were done

The following data was extracted from the patient's history, clinical examination and during follow up:

1. Selection of the patient (Patients with diabetic distal neuropathic toe ulcers with Wagner's classification 1 and 2 and patients with toe deformity selected)
2. Diabetic status of the patient
3. Treatment compliance
4. Footwear examination
5. Infected / not infected
6. Flexor tenotomy and wound debridement done
7. Rate of healing of ulcers measured weekly until the ulcer is healed
8. Follow up of patients continued for one year after healing of ulcer monthly to look for ulcer recurrence

Analysis

Data were analyzed according to history, clinical examination and investigation. Data were entered in excel sheet and analyzed using SPSS v23. Frequencies and percentage analysis were done. Cross tabulation and Chi-square analyses were done to find the relationship and association between various variables.

Results

A Prospective Analysis was done on the efficacy of percutaneous flexor tenotomy procedures in rate of healing and recurrence of diabetic neuropathic distal toe ulcer. This study focused on studying the outcome and efficacy of flexor tenotomy procedure in the recurrence of diabetic distal toe ulcer and in the rate of healing of distal toe ulcer and to describe the outcomes of prophylactic flexor tenotomy procedure in diabetic patients with toe deformity. Patients between the age groups of 18 years and 60 years were included who were affected with diabetic neuropathic ulcer in the distal end of toes, presented with toe deformity and classified as Wagner's classification 1 and 2. The mean age is 52 years with the standard deviation of 9.85 years. The median age was 54.5 years within the range of 33 years to 70 years. Among fifty patients, majority of them were males (n=33, 66%) while the rest were females (n=17, 34%). All patients in the study group were diagnosed with diabetes mellitus. The mean duration of type II DM among the fifty patients was 36.06 months with a standard deviation of 10.1 months. The median duration was 35 years ranging between 16-58 months.Out of fifty patients, 68% (n=34) of them were on regular treatment while the remaining 32% (n=16) were on irregular treatment.

Age distribution

The mean age is 52 years with the standard deviation of 9.85 years. The median age was 54.5 years within the range of 33 years to 70 years. The following figure and table shows the age distribution of the patients.

Age distribution	In years
Mean	52.600
Median	54.500
Mode	59.0
Std. Deviation	9.8520
Minimum	33.0
Maximum	70.0

Table 1: Age distribution

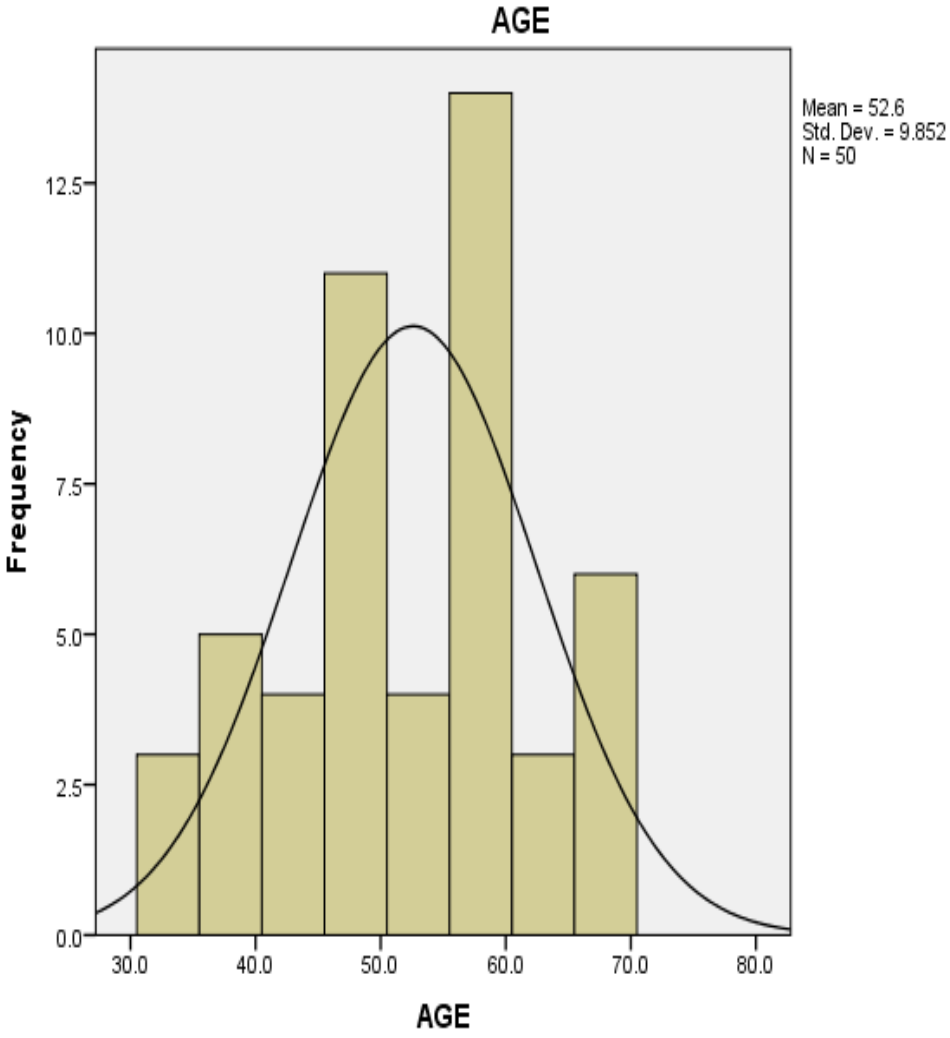


Figure 1: Age distribution

Gender distribution

Among fifty patients, majority of them were males (n=33, 66%) while the rest were females (n=17, 34%). The following figure and table shows the gender distribution of the participants.

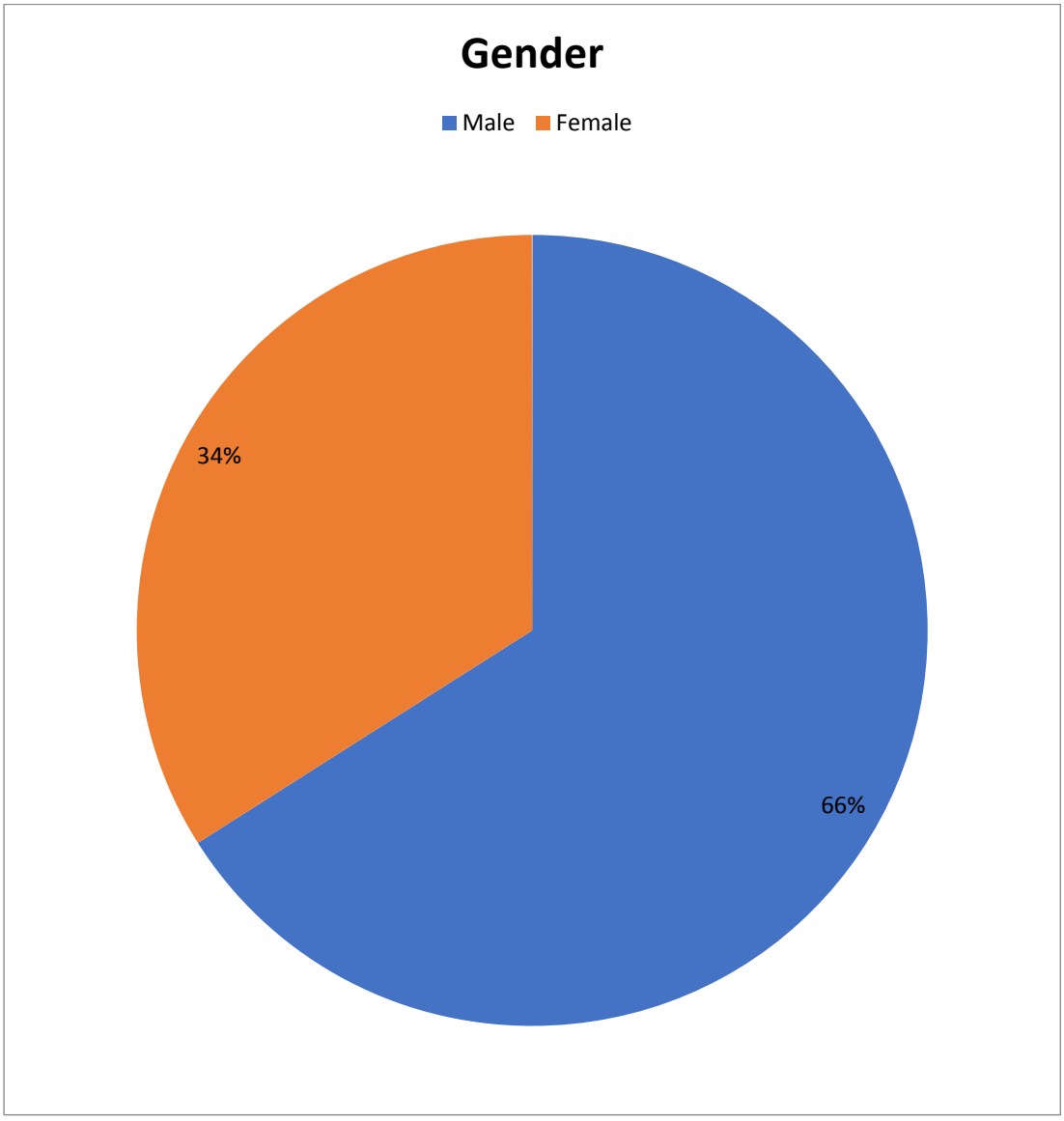


Figure 2: Gender Distribution

Gender Distribution	Frequency	Percent
Female	17	34.0
males	33	66.0
Total	50	100.0

Table 2: Gender Distribution

Type of Diabetic Mellitus

All patients in the study group were diagnosed with Type II diabetes mellitus.

Duration of Diabetic Mellitus

The mean duration of type II DM among the fifty patients was 36.06 months with a standard deviation of 10.1 months. The median duration was 35 years ranging between 16-58 months. The following table and figure shows the duration of diabetic mellitus among the patients.

Duration of DM	In months
Mean	36.060
Median	35.000
Mode	34.0
Std. Deviation	10.1003
Minimum	16.0
Maximum	58.0

Table 3: Duration of Diabetes Mellitus

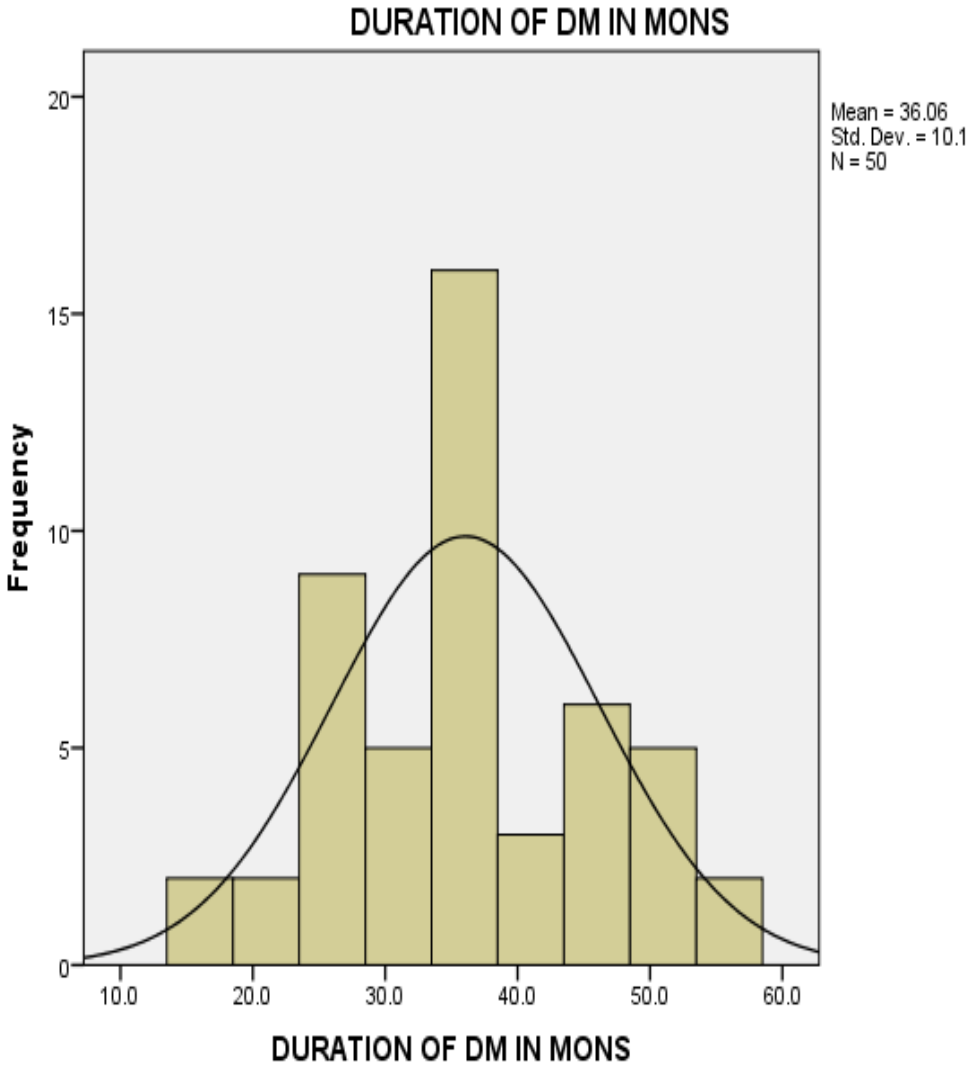


Figure 3: Duration of DM

Treatment Regularity

Out of fifty patients, 68% (n=34) of them were on regular treatment while the remaining 32% (n=16) were on irregular treatment. The following figure and treatment shows the treatment irregularity of the patients of the study.

Treatment(Regular/Irregular)	Frequency	Percent
Irregular	16	32.0
Regular	34	68.0
Total	50	100.0

Table 4: Treatment Regularity

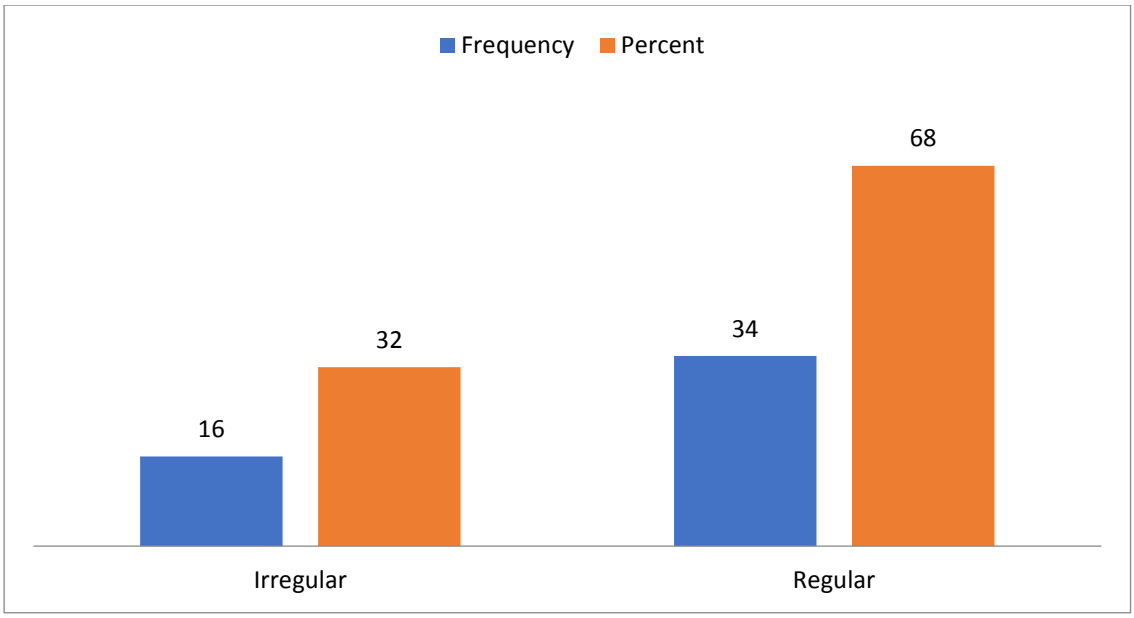


Figure 4: Treatment Regularity

Ulcer characteristics

The following section deals with the characteristics of the ulcer namely; duration of ulcer, site of ulcer, and presence of infection.

Duration of ulcer

The mean duration of ulcer was 38.35 days with a standard deviation of 11.03 days. The median number of days is 38.5 days ranging between 14 days and 60 days. The following table and figure shows the duration of ulcer in days.

Duration of Ulcer	In days
Mean	**38.350**
Median	**38.500**
Mode	**37.0**
Std. Deviation	**11.0397**
Minimum	**14.0**
Maximum	**60.0**

Table 5: Duration of ulcer (in days)

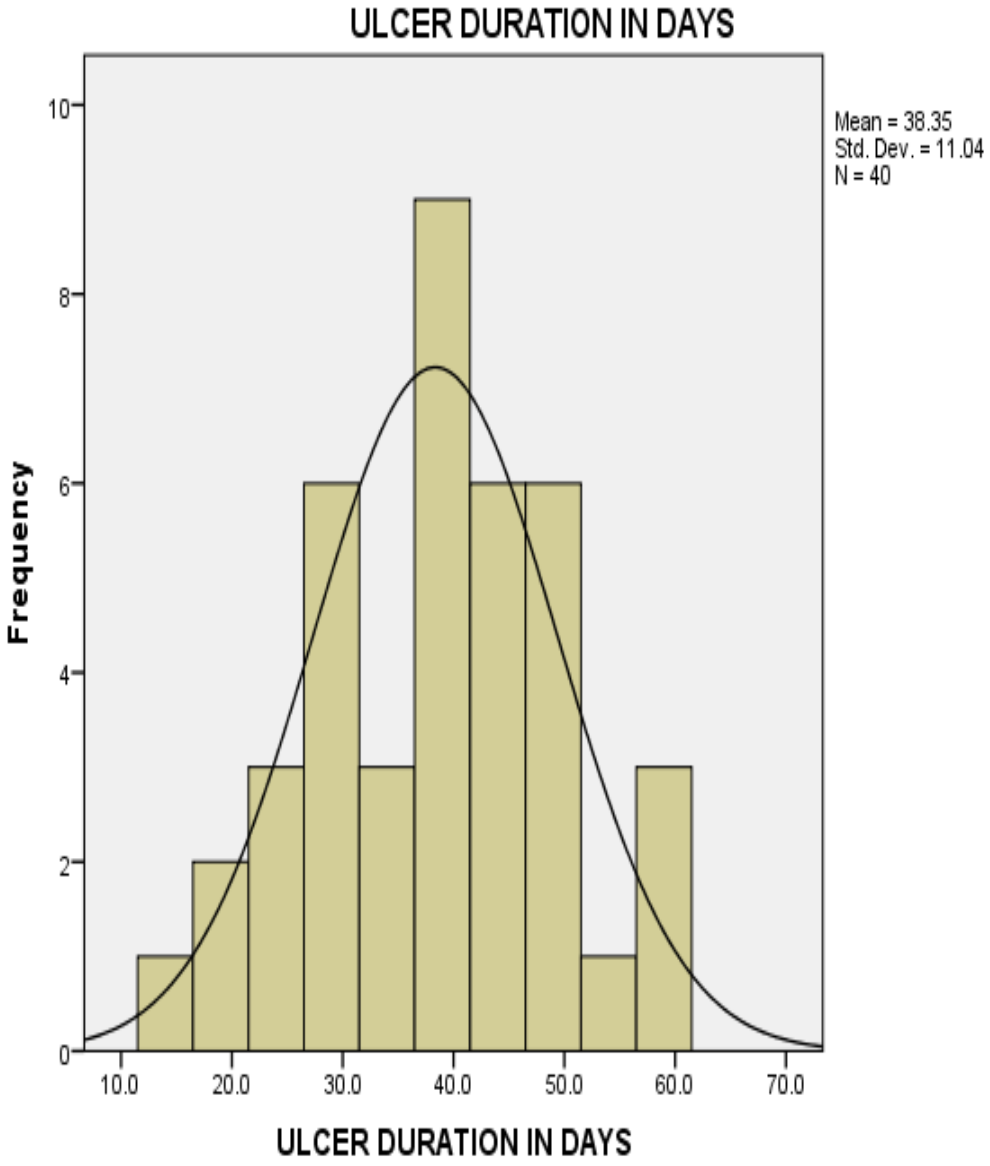


Figure 5: Duration of ulcer (in days)

Ulcer site

The following table shows the frequency of the ulcers in different sites. The subsequent figure shows the same.

Ulcer	Frequency	Percent
1	15	30.0
2	5	10.0
3	7	14.0
4	7	14.0
5	6	12.0
N	10	20.0
Total	50	100.0

Table 6: Ulcer Site

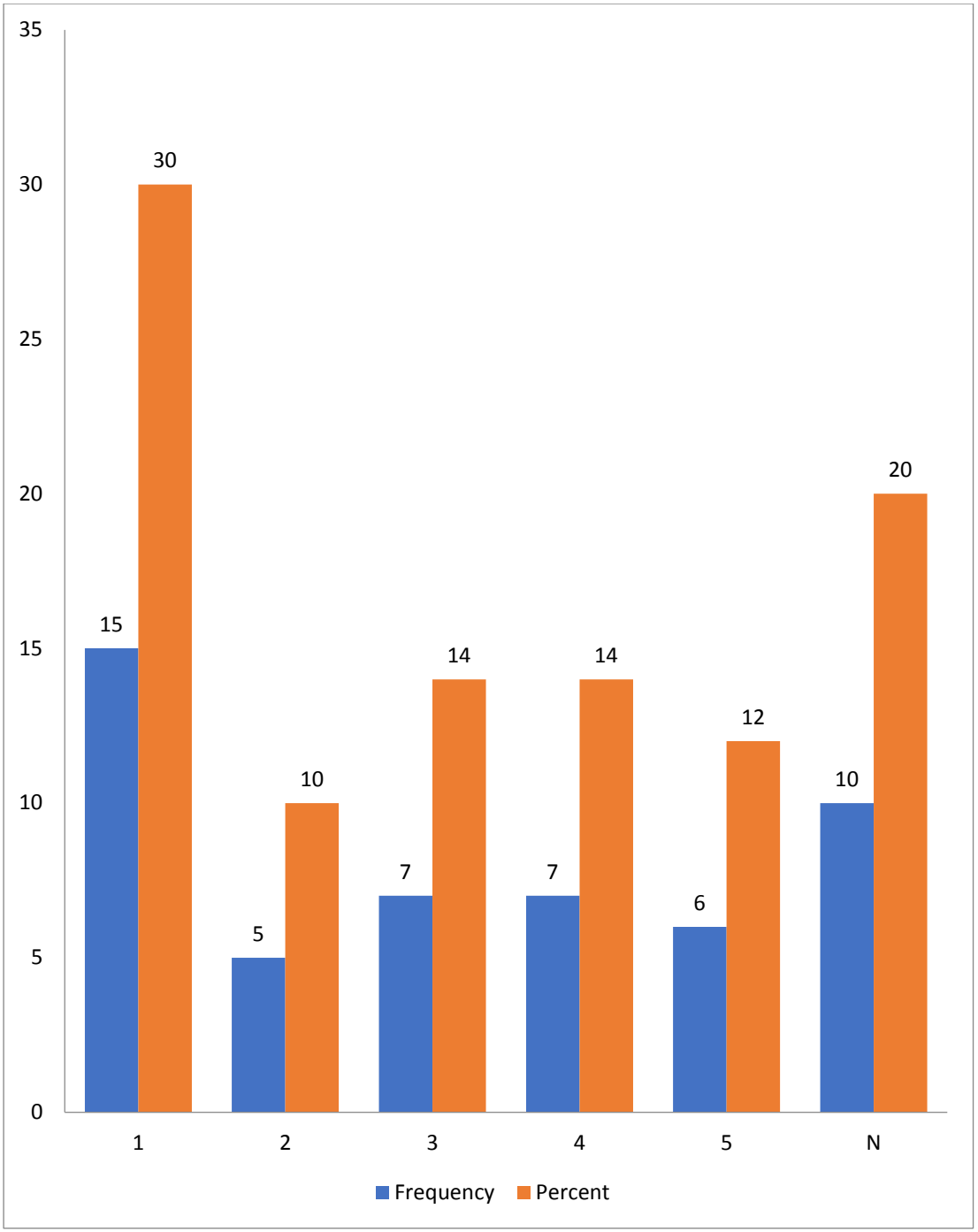


Figure 6: Ulcer site

Presence of infection

Infection was present in 40% (n=20) of the patients. Rest of them did not have any infection (n=30, 60%). The following table and figure shows the presence of infection.

Presence of Infection	Frequency	Percent
Yes	20	40.0
No	30	60.0
Total	50	100.0

Table 7: Presence of Infection

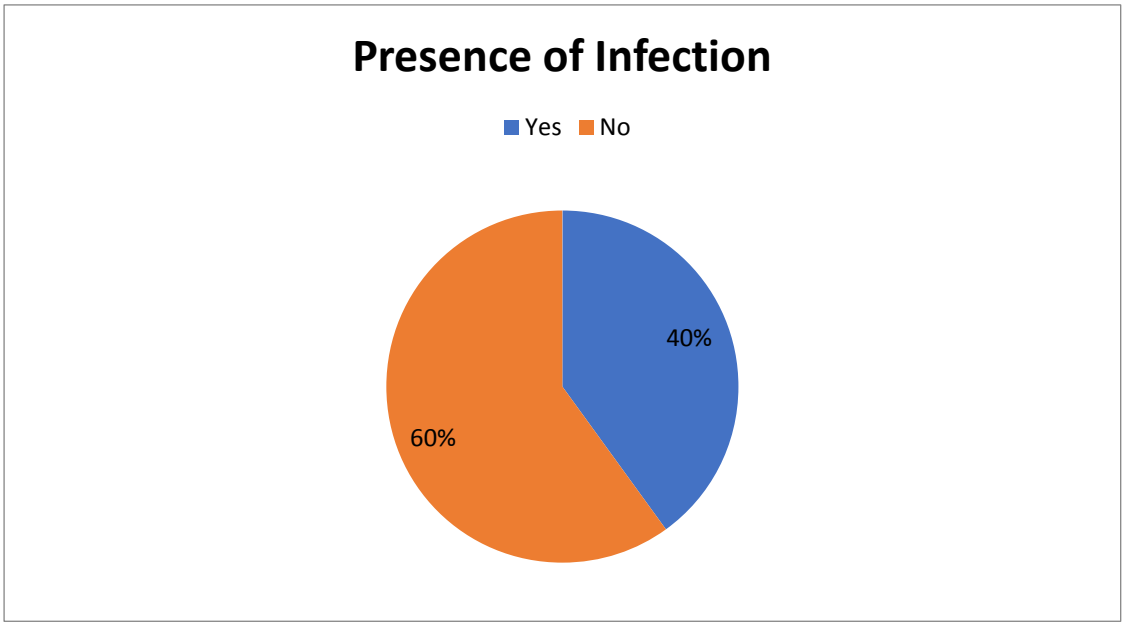


Figure 7: Presence of Infection

History of Callus Formation

Out of fifty patients, 39 (78%) of them had history of callus formation. Rest of them (n=11, 22%) had no history of callus formation. The following table and figure shows the history of callus formation.

History of Callus Formation	Frequency	Percent
Yes	20	40.0
No	30	60.0
Total	50	100.0

Table 8: History of Callus Formation

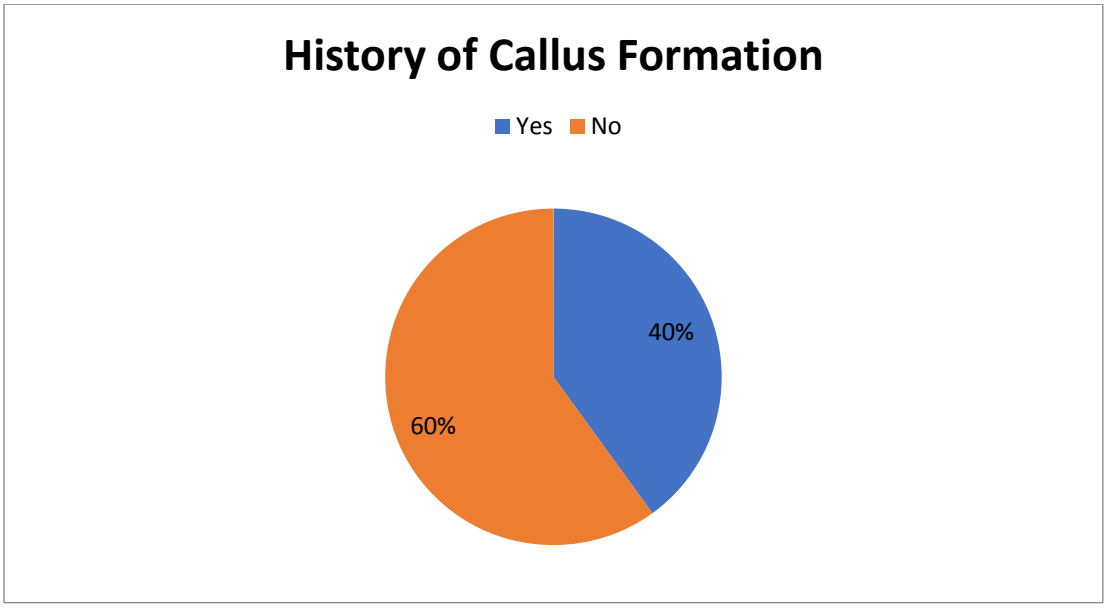


Figure 8: History of Callus Formation

Deformity

All of the patients had deformity.

Neuropathic Symptoms

The neuropathic symptoms was present in 88% (n=44) of them while it was absent in 12% (n=6) of the patients. The following table and figure shows the neuropathic symptoms.

Presence of Neuropathic Symptoms	Frequency	Percent
Yes	44	88
No	6	12
Total	50	100.0

Table 9: Neuropathic Symptoms

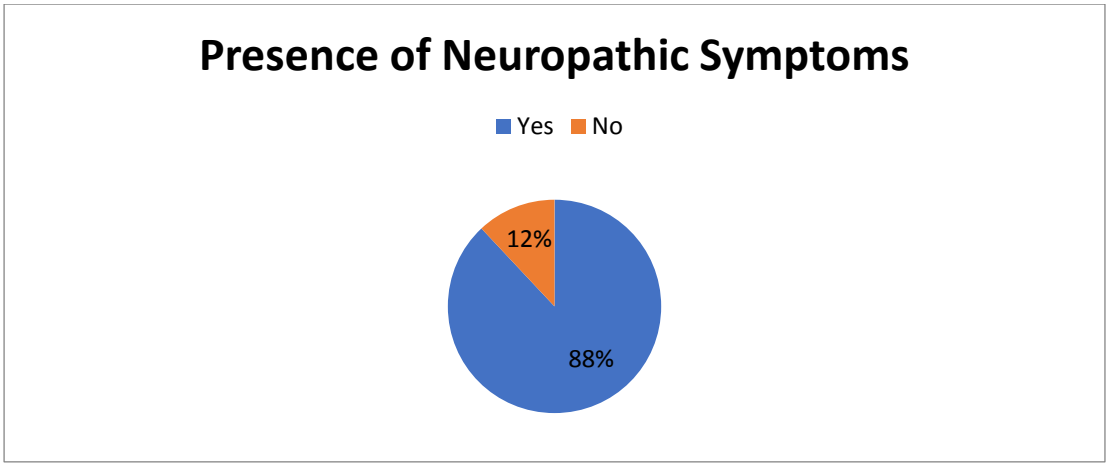


Figure 9: Neuropathic Symptoms

X-ray of the affected limb

The x-ray of the affected limb showed the presence of osteomyelitis in 8% (n=4) of the cases. Rest of the cases (92%, n=46) had a normal x-ray. The following figure shows the incidence of findings in the x-ray of the affected limb.

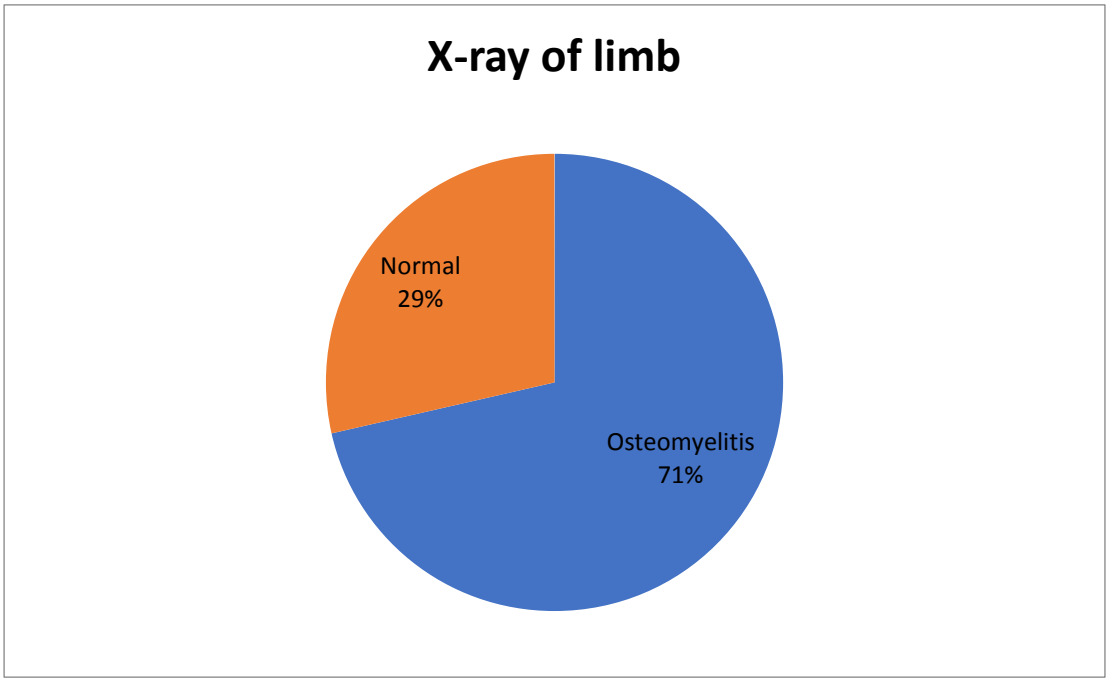


Figure 10: X-ray of the limb

ABI

ABI was normal in all the patients.

Flexor Tenotomy

Flexor tenotomy was done in all the fifty patients. 80% (n=40) of them had a therapeutic tenotomy while 20% (n=10) of them had a prophylactic Tenotomy. The following table and figure shows flexor tenotomy done for the patients.

Flexor Tenotomy	Frequency	Percent
Prophylactic	40	80
Therapeutic	10	20
Total	50	100.0

Table 10: Flexor Tenotomy

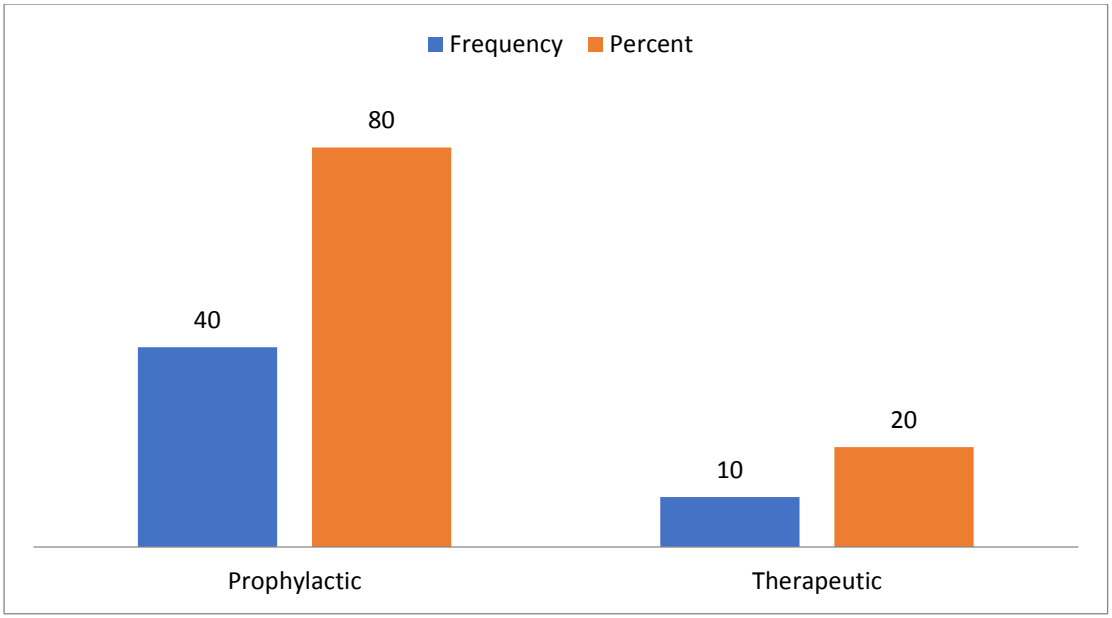


Figure 11: Flexor Tenotomy

History of Previous Surgery or Infection

Around 20% of the patients had a history of previous surgery or infection. Rest of them (n=40, 80%) had no history of previous surgery or infection. The following table and figure shows the history of previous surgery or infection.

History of previous surgery/infection	Frequency	Percent
No	40	80
Yes	10	20
Total	50	100.0

Table 11: History of Previous Surgery or Infection

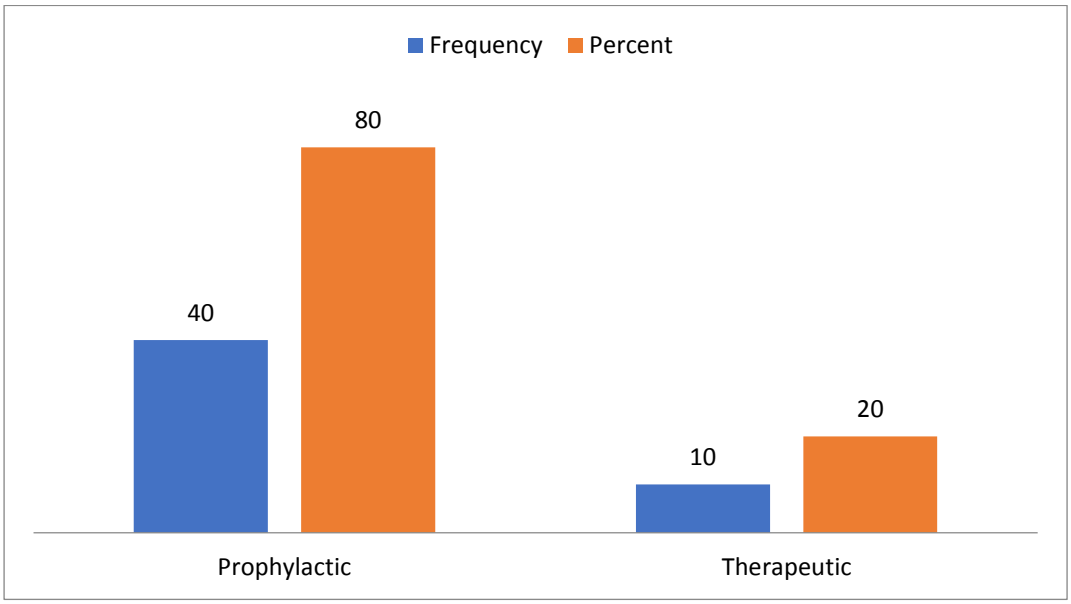


Figure 12: History of Previous Surgery or Infection

Pus culture and sensitivity

Out of the 50 patients, 36% (n=18) of them were positive for pus culture and sensitivity. Following figure shows the pus culture and sensitivity.

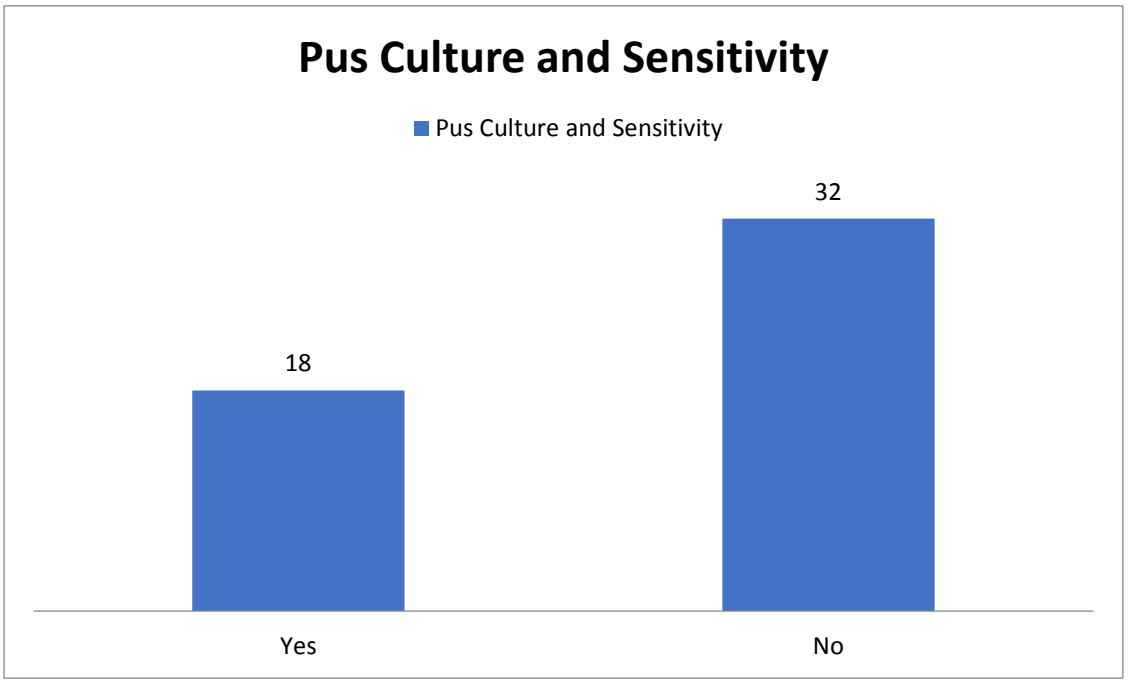


Figure 13: Pus Culture and Sensitivity

Presence of Neuropathy

All the patients present in the study were affected by neuropathy.

Presence of distal pulse

Distal pulse were not felt in 6% (n=3) of the cases. The following figure shows the presence of distal pulse.

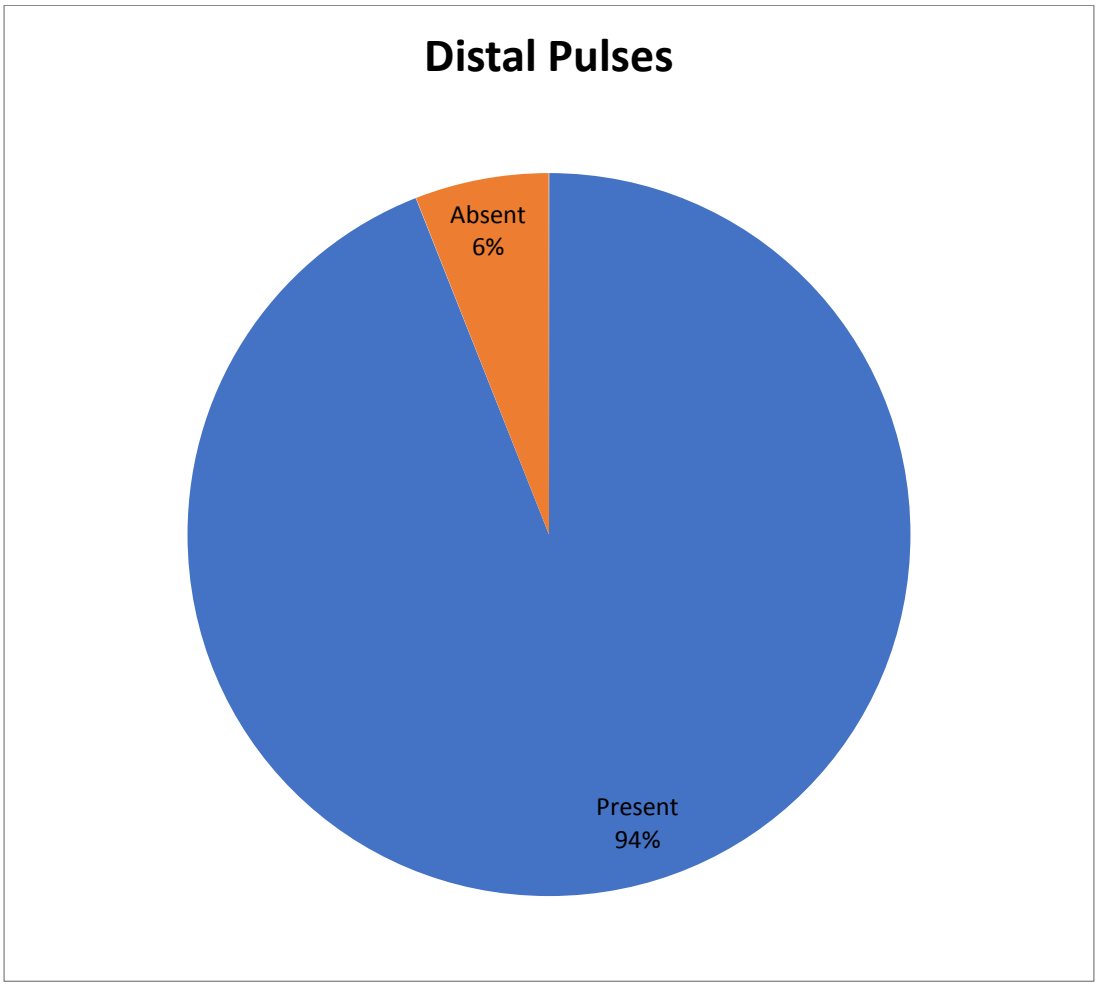


Figure 14: Distal pulse

Treatment and Follow-up

Flexor tenotomy was done in all the fifty patients. 80% (n=40) of them had a therapeutic tenotomy while 20% (n=10) of them had a prophylactic Tenotomy. The following table and figure shows flexor tenotomy done for the patients.

In the group that had therapeutic tenotomy, following were the findings;

- Out of 40 patients, the initial mean size of the ulcer is 2.37 cm (S.D=0.83); Range=1-4cm; Median=2 cm
- The mean size of ulcer in first week is 1.76 cm (S.D=0.78); Range=0.4-3.4 cm; Median=1.4 cm
- The mean size of ulcer in second week is 1.15 cm (S.D=0.84); Range=0-3 cm; Median=1 cm
- The mean size of ulcer in first month is 0.4 cm (S.D=0.68); Range=0-2.5 cm; Median= 0
- The mean size of the ulcer in second month is 0.11 cm (S.D=0.28); Range=0-1; Median=0

In the group that had prophylactic tenotomy, all 10 patients did not have any ulcer during the study period.

The following table and figure shows the reduction in the sizes of the ulcer in the 40 patients;

Statistics	Initial size (cm)	At 1 Week (cm)	At 2 Week (cm)	At 1 Month (cm)	At 2 Months (cm)
Mean	2.37	1.76	1.155	.395	.113
Median	2	1.6	1.000	.000	.000
Mode	2	1.4	.0	.0	.0
Standard Deviation	0.83	0.78	.84	.68	.28
Minimum	1	0.4	.0	.0	.0
Maximum	4	3.4	3.0	2.5	1.0

Table 12: Reduction in the sizes of the ulcer

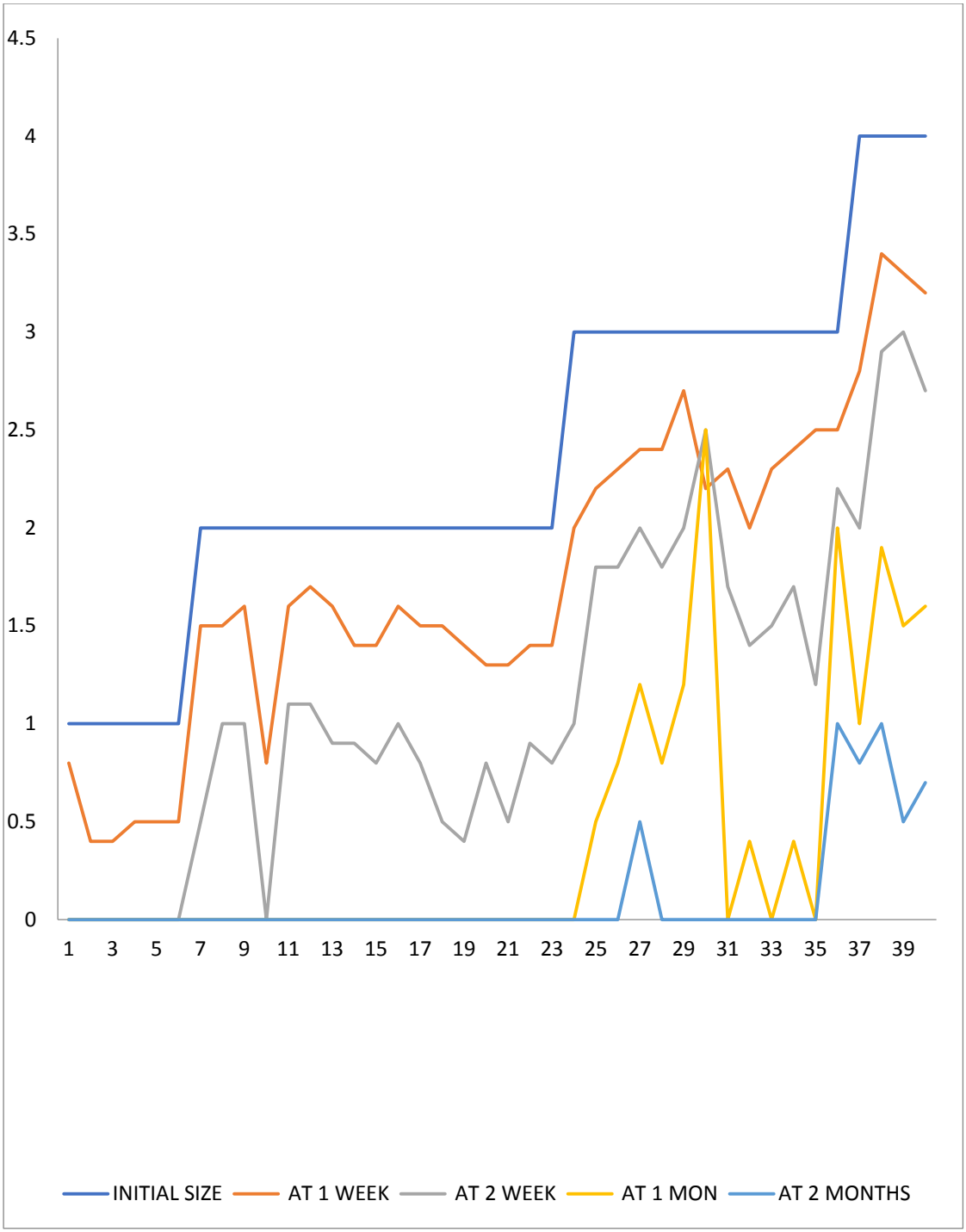


Figure 15:Reduction in the sizes of the ulcer

The following table and figure shows the percentage reduction in the sizes of the ulcer in the 40 patients;

- The mean percentage reduction of ulcer in first week is 28.5% (S.D=12.8%); Range=10%-60%; Median=25%
- The mean percentage reduction of ulcer in second week is 58.3% (S.D=24%); Range=16-100%; Median=54.17%
- The mean percentage reduction of ulcer in first month is 88.08% (S.D=0.68); Range=16.67%-100%; Median= 100%
- The mean percentage reduction of the ulcer in second month is 96.87% (S.D=7.98%); Range=66%-100%; Median=100%

In the group that had prophylactic tenotomy, all 10 patients did not have any ulcer during the study period.

	Percentage Reduction Week 1	**Percentage Reduction Week 2**	**Percentage Reduction Month 1**	**Percentage Reduction Month 2**
Mean	28.5208	58.2917	88.0833	96.8750
Median	25.0000	54.1667	100.0000	100.0000
Mode	20.00	100.00	100.00	100.00
Std. Deviation	12.81232	24.00658	20.78687	7.98358
Minimum	10.00	16.67	16.67	66.67
Maximum	60.00	100.00	100.00	100.00

Table 13: Percentage reduction in the size of the ulcer

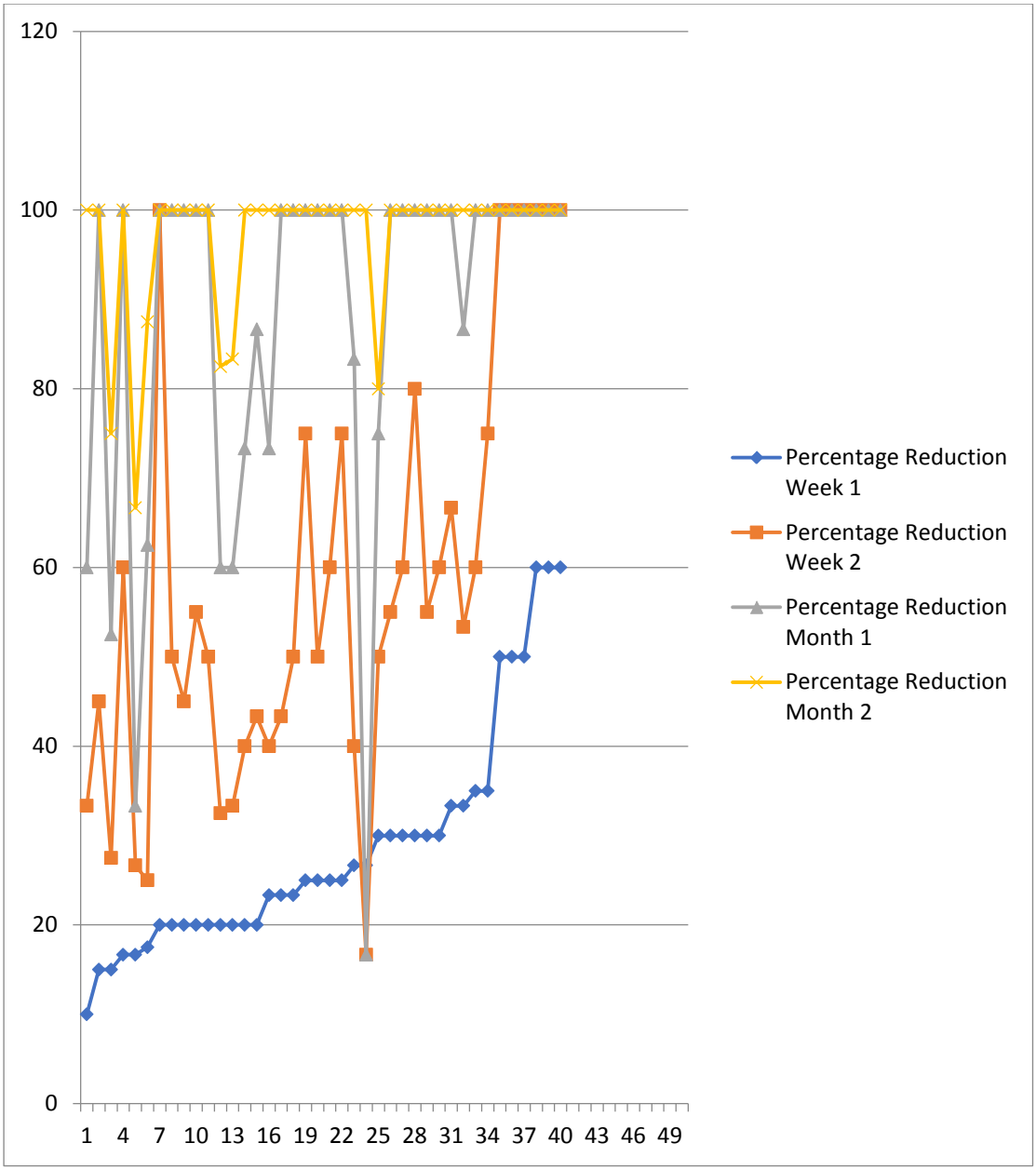


Figure 16: Percentage reduction in the size of the ulcer

Complications at week 1, week 2 and end of month one

There were no complications at week one and week two. At the end of month one; there was one amputation and three transfer ulcers in the therapeutic group. The following table and figure shows the complications at week 1, week 2 and end of month one.

	Week One	**Week Two**	**At the end of Month one (All patients with complications were in the therapeutic Group)**
Complications	Nil (n=50, 100%)	Nil (n=50, 100%)	Nil (n=46, 92%)
Amputation			N=1, 2%
Transfer Ulcers			N=3, 6%

Table 14:Complications at week 1, week 2 and end of month one

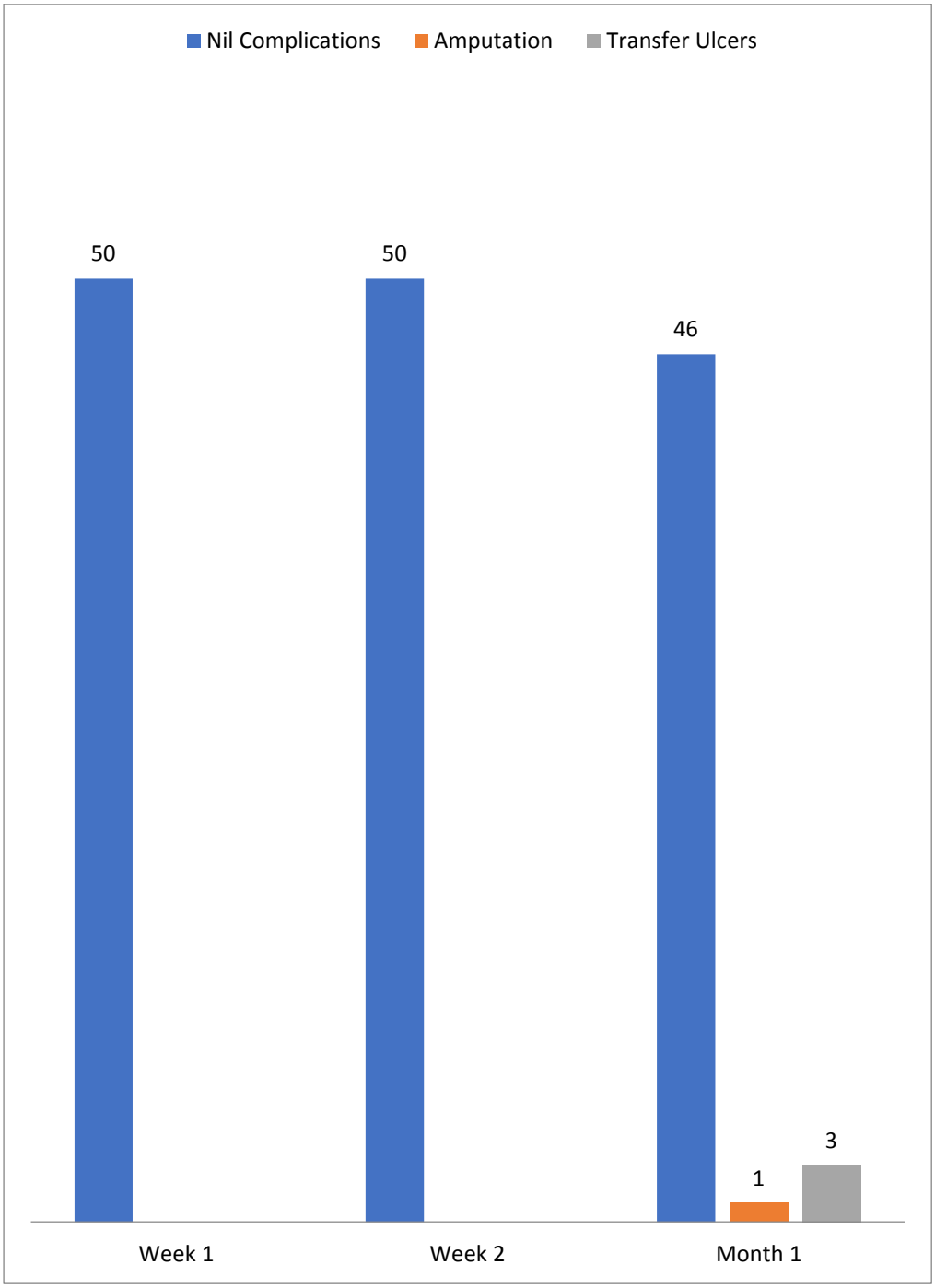


Figure 17:Complications at week 1, week 2 and end of month one

Follow up at 2-Month, 4-Month, 6-Month and One Year

Follow up 2-Month, 4-Month, 6-Month and One Year showed an increase in cure rates after flexor tenotomy in the therapeutic group and no ulcer in the prophylactic group. At the end of one year, the cure rate was 90% (n=36) with cured and transfer ulcers being 7.5% (n=3) and 2.5% (n=1) amputation.

	2-Month	**4-Month**	**6-Month**	**One Year**
Prophylactic (n=10, 20%)	**No ulcer**	**No ulcer**	**No ulcer**	**No ulcer**
Therapeutic (n=40, 80%)				
Cured	**31 (62%)**	**31 (62%)**	**31 (62%)**	**31 (62%)**
Cured with Transfer Ulcers	**2 (4%)**	**2 (4%)**	**2 (4%)**	**2 (4%)**
Amputation	**1 (2%)**	**1 (2%)**	**1 (2%)**	**1 (2%)**
0.5cm	**2 (4%)**	**Cured**	**Cured**	**Cured**
0.7 cm	**1 (2%)**	**Cured**	**Cured**	**Cured**
0.8 and Transfer Ulcer	**1 (2%)**	**Cured + Transfer Ulcer**	**Cured + Transfer Ulcer**	**Cured + Transfer Ulcer**
1 cm	**2 (4%)**	**Cured**	**Cured**	**Cured**

Table 15: Follow up at 2-Month, 4-Month, 6-Month and One Year

Discussion

A Prospective Analysis was done on the efficacy of percutaneous flexor tenotomy procedures in rate of healing and recurrence of diabetic neuropathic distal toe ulcer. This study focused on studying the outcome and efficacy of flexor tenotomy procedure in the recurrence of diabetic distal toe ulcer and in the rate of healing of distal toe ulcer and to describe the outcomes of prophylactic flexor tenotomy procedure in diabetic patients with toe deformity. Patients between the age groups of 18 years and 60 years were included who were affected with diabetic neuropathic ulcer in the distal end of toes, presented with toe deformity and classified as Wagner's classification 1 and 2. The mean age is 52 years with the standard deviation of 9.85 years. The median age was 54.5 years within the range of 33 years to 70 years. Among fifty patients, majority of them were males (n=33, 66%) while the rest were females (n=17, 34%). All patients in the study group were diagnosed with diabetes mellitus. The mean duration of type II DM among the fifty patients was 36.06 months with a standard deviation of 10.1 months. The median duration was 35 years ranging between 16-58 months.Out of fifty patients, 68% (n=34) of them were on regular treatment while the remaining 32% (n=16) were on irregular treatment.

Diabetic foot ulcers are caused by a number of factors related to the architecture of the bone of foot, peripheral neuropathy and atherosclerotic peripheral arterial disease. These are the common manifestations of people affected by diabetes[17]. All these factors lead to the infection of the foot in advanced conditions and is known to be a common factor for diabetes related hospitalisations and amputation[18]. The stiffness of the ligaments in the disease is due to the nonenzymatic glycation. In otherwise normal individuals, too much pressure and stress on the pressure points in the foot may lead to the reflexive change in position due to responsive nerve endings.

One of the most devastating complications of diabetes is foot ulceration. The most common sites are;

a) Dorsum

b) Apices

c) Plantar aspects of toes

The ulcers that occur on the 1-5 toes comprise anywhere between 43 and 55.5% of all reported foot ulcers[1,2]. Other sites are also involved like forefoot, mid-foot and heel ulcers where the presentation is larger than the digital ulcers[3]. One of the reasons why digital ulcers are very important is that they commonly precede limb amputations in diabetic

patients upto 63.9% of the time[4]. The early diagnosis, treatment and prevention is essential for prevention and management of diabetic ulcers and improve prognosis in the long term.

Diabetic foot ulcers are caused by a number of factors related to the architecture of the bone of foot, peripheral neuropathy and atherosclerotic peripheral arterial disease. These are the common manifestations of people affected by diabetes[17]. All these factors lead to the infection of the foot in advanced conditions and is known to be a common factor for diabetes related hospitalisations and amputation[18]. The stiffness of the ligaments in the disease is due to the nonenzymatic glycation. In otherwise normal individuals, too much pressure and stress on the pressure points in the foot may lead to the reflexive change in position due to responsive nerve endings.

But, in diabetes mellitus, the neuropathy is responsible for the decrease in the sensitivity of the foot and thereby lead to the loss of the reflexive protective sensation and also to the coordination errors in the foot and leg muscles. All these factors together cause the mechanical stress to the otherwise normal foot during ambulation and lead to the discussed complications[19]. This is the reason why diabetes happens to be the most common non-traumatic cause of foot amputations with around 5% of

diabetic population developing it and approximately 1% requiring amputation[20].

The assessment of diabetic foot ulcer requires a multipronged approach[21];

e) Physical examination of the extremity affected by the ulcer
   - Examination of ulcer
   - Condition of the extremity
f) Vascular insufficiency assessment

The depth of involvement of soft tissue and bone dictates the staging of the disease[22-24]. Then routine investigations of complete blood cell count, serum creatinine, serum glucose and glycosylated haemoglobin. The management is very elaborate starting from the offloading of weight from the foot by using appropriate footwear[25,26]. Saline dressings are required to maintain a moist environment[27], do debridement and put on antibiotic therapy is there are any infections[28,29]. The diabetic control along with the monitoring and correction of the insufficiencies of the peripheral arteries are also important.

Toe deformities are also common in the long term like 'hammer' and 'claw' toes in the evolution of diabetic foot ulcers. These deformities are common when there are comorbities and complications like neuropathy and peripheral vascular disease[5]. The problem with diabetic foot is that

the nerve endings in the affected foot becomes insensate. This leads to poor reflexes during gait or weight bearing. Subsequently, there is more stress to the pressure points. Also, it results in callus formation. Long standing callus leads to tissue trauma and resultant ulcer[6].

One of the main treatment goal is to off-load pressure from the sites of ulcer. This off-loading helps in the reduction of stress, pressure and callus formation enabling an environment for the healing of ulcer. Continual treatment helps in preventing the recurrence of the ulcer[7]. Traditional methods of treatment for off-loading has been conservative with[8-10];

- regular debridement of the corn and callus (superficial skin lesions)
- deflective padding
- insoles
- therapeutic foot wear

Studies are inadequate in suggesting the efficacy and effectiveness of such conservative treatments. Further, poor patient adherence confounds the actual effectivness of the methods suggested above. Studies have shown that pressure relieving footwear has poor adherence as with other wearing removable offloading devices[11].

Surgical interventions might reduce the risk and recurrence of diabetic foot ulcers especially in patients with peripheral neuropathy[12]. Infections

and healing rates in diabetic foot ulcers may be altered by minimally invasive surgical procedures[13].

For flexible toe deformities, flexor tenotomy procedure can be advocated for hallux and the lesser toes[14]. Flexor tenotomy procedure is done under local anesthetic where a plantar incision is made to transect flexor digitorum/hallucis longus tendon[15].

There are surgeons who also prefer to release the tendon of flexor digitorum/hallucis brevis[16]. The aim of this procedure is to release the tendon of flexor digitorum brevis/longus to make the position of the toe straighter. This leads to the reduction in the amount of pressure, stress and subsequent callus and ulcer formation. Ulcer at the toe apices are the most benefitted. The procedure is usually done as an outpatient basis.

The peripheral neuropathy in diabetes is seen in 60% of the patients with diabetes and in 80% of the patients with diabetic foot ulcers thus making it the one single most significant factor in the pathogenesis of the disease. Tissue mechanics is also studied to understand how it is associated with DFUs. The heal pad is known to be stiffer in patients with diabetes than those who are not having DM[46]. High thickness of plantar soft tissue with lower stiffness of the soft tissues in the first metatarsal head is known to contribute to the DFUs[47]. This indicates that the calculation of risk for diabetic foot ulcers should also focus on the anatomy of the foot.

People with higher risk are those who have flat foot and disproportionate distribution of weight bearing across the surface of the foot. Tenotomies are not rare in foot and ankle surgeries for many decades. They were performed either alone in cases of tendon contractures or in combination with bone surgeries when the bones were involved. Successful digital surgeries have always depended on tenotomies[68-71]. The minimally invasive technique of tenotomy was first reported by McGowan[72]. As the search for less invasive procedures began, surgeons started moving towards the percutaneous procedures for ankle and foot deformities especially for club foot deformities[73-75].

The same technique has found importance in treating Achilles tendonopathies[76,77]. Vertical talus deformity is the latest addition in this list[78,79,80]. The first person to describe this technique is Minkowitz[81]. For club foot repair, he modified the Ponsetti method by using a large gauge needle for the lengthening of the Achilles tendon percutaneously.

The study shows that percutaneous tenotomy is very effective in the management of diabetic foot ulcers with better outcome and prognosis with better healing rates and lesser recurrence rates.

Summary and Conclusions

A Prospective Analysis was done on the efficacy of percutaneous flexor tenotomy procedures in rate of healing and recurrence of diabetic neuropathic distal toe ulcer. This study focused on studying the outcome and efficacy of flexor tenotomy procedure in the recurrence of diabetic distal toe ulcer and in the rate of healing of distal toe ulcer and to describe the outcomes of prophylactic flexor tenotomy procedure in diabetic patients with toe deformity.

a) Patients between the age groups of 18 years and 60 years were included who were affected with diabetic neuropathic ulcer in the distal end of toes, presented with toe deformity and classified as Wagner's classification 1 and 2.
b) The mean age is 52 years with the standard deviation of 9.85 years. The median age was 54.5 years within the range of 33 years to 70 years.
c) Among fifty patients, majority of them were males (n=33, 66%) while the rest were females (n=17, 34%).
d) The mean duration of type II DM among the fifty patients was 36.06 months with a standard deviation of 10.1 months.

e) The study shows that percutaneous tenotomy is very effective in the management of diabetic foot ulcers with better outcome and prognosis with better healing rates and lesser recurrence rates.

Limitations

The study had the following limitations;

a) The sample size was small

b) Samples were selected from only a single catchment area

c) Generalisability of the results are not possible

d) Long term follow-up and outcome analysis is not possible logistically

Recommendations

Future studies should focus on the following;

a) Establishing a cohort for long term follow up
b) Choose a number of catchment areas
c) Do randomised control trials for comparison with other methods
d) Long term follow ups with respect with morbidity and mortality is required to understand the true impact of these procedures.

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A Prospective Analysis on the efficacy of percutaneous flexor tenotomy procedures in rate of healing and recurrence of diabetic neuropathic foot ulcer

PROFORMA

NAME:

ADDRESS:

AGE/SEX:

OCCUPATION:

I.P No:

D.O.A:

CHIEF COMPLAINTS:

H/o trauma/blister/ulcer

-Onset

-Duration

-Progress

H/O PRESENTING COMPLAINTS:

DIABETIC HISTORY:

H/o diabetes - duration and treatment

H/o glycemic control

H/o footwear

H/o daily activities including work

H/o callus formation

H/o foot deformity

H/o neuropathic symptoms – numbness/loss of sensation/weakness of limbs

H/o claudication or rest pain

H/o previous infection/surgery

H/o Charcot foot – previous or active

PAST HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

TREATMENT HISTORY:

EXAMINATION

GENERAL EXAMINATION:

Built/Nourishment

Level of consciousness/Orientation

Facial puffiness/Pallor/Icterus

Clubbing/Cyanosis

Febrile/Afebrile

Pedal oedema/anasarca

VITALS:

BP: TEMP:

HR: RR:

EXAMINATION OF VASCULAR SYSTEM

PERIPHERAL PULSE CHART

SITE	**RIGHT**	**LEFT**
UPPER LIMB		
Axillary artery		
Brachial artery		
Radial artery		
Ulnar artery		
LOWER LIMB		
Femoral artery		
Popliteal artery		
Posterial tibial artery		
Dorsalis pedis artery		

CAPILLARY REFILLING TIME: ELEVATION PALLOR:

EDEMA: DEPENDENT RUBOR:

LOCAL EXAMINATION:

INSPECTION:

Ulcer site/size/extent

Colour of wound/smell of wound

Discharge from wound

Margins

Edge

Floor- Slough/Bone/Muscle/Maggots

Surrounding skin – Colour/Hair/texture/Oedematous/Dry

Movement of joints/limbs

Gait –

Any deformity

Nail – brittle/ingrowing/overgrowing/avulsion

Web spaces – maceration/fungal infection

Inguinal region –

PALPATION:

Inspectory findings confirmed

Warm/tenderness

Bleeds/does not bleed on touch

Base

Mobility of ulcer

Nerve thickening

Sensation – increased/decreased/loss/no change

Movement of major joints

Surrounding skin – nodules/pitting/induration

Inguinal region – nodes

WAGNERS CLASSIFICATION:

ULCER FOOT DIAGRAM:

NEUROLOGICAL EXAMINATION:

EXAMINATION OF OTHER SYSTEMS:

CVS -

RS –

ABDOMEN -

CNS -

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS

A. HB% ,PCV

B. URINE:

Albumin

Sugar

Acetone

C. BLOOD:

RBS

BLOOD UREA

SERUM CREATININE

D. TLC,DC,PLATELETS

E. FASTING/POSTPRANDIAL BLOOD SUGAR

F. BLOOD GROUPING & TYPING

G. BT/CT

H. HIV

I. ECG

J. CHEST X RAY PA VIEW

K. X RAY OF AFFECTED LIMBS

L. PUS CULTURE AND SENSITIVITY

FLEXOR TENOTOMY PROCEDURE

POST-OPERATIVE PERIOD / COMPLICATIONS:

ULCER HEALING FOLLOW UP:

FIRST WEEK	
SECOND WEEK	
ONE MONTH	
SECOND MONTH FOURTH MONTH	
SIXTH MONTH	
NINTH MONTH	
ONE YEAR	

PATIENT CONSENT FORM

STUDY TITLE:

"A PROSPECTIVE ANALYSIS ON THE EFFICACY OF PERCUTANEOUS FLEXOR TENOTOMY PROCEDURES IN RATE OF HEALING AND RECURRENCE OF DIABETIC NEUROPATHIC DISTAL TOE ULCER"

Department of General surgery, GMKMCH

PARTICIPANT NAME : AGE : SEX:

I.P. NO :

I confirm that I have understood the purpose of surgical/invasive procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.
I have been explained about the possible complications that may occur during and after medical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.
I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study. I hereby consent to participate in this study.

Date : Signature / Thumb Impression Of Patient

Place :

Signature of the investigator: _____

Name of the investigator : _____

KEY TO MASTER CHART

T- THERAPEUTIC FLEXOR TENOTOMY

P- PROPHYLACTIC FLEXOR TENOTOMY

M-MALE

F-FEMALE

R-REGULAR TREATMENT FOR DM

I-IRREGULAR TREATMENT FOR DM

1-GREAT TOE

2-SECOND TOE

3-THIRD TOE

4-FOURTH TOE

5-FIFTH TOE

Y-YES

N-NO

F-DISTAL PULSES FELT

MASTER CHART

S.NO.	AGE	SEX	TYPE OF DM(All are Type 2 DM ONLY)	DURATION OF DM IN MONS	TREATMENT(REGULAR/IRREGULAR)	ULCER SITE	ULCER DURATION IN DAYS	PRESENCE OF INFECTION	H/O CALLUS FORMATION	DEFORMITY	NEUROPATHIC SYMPTOMS	X RAY OF AFFECTED LIMBS(OSTEOMYELITIS PRESENT = Y,NO OSTEOMYELITIS =N)	ABI	FLEXOR TENOTOMY THERAPEUTIC	FLEXOR TENOTOMY PROPHYLACTIC	H/O PREVIOUS SURGERY/INFECTION	PUS C/S	PRESENCE OF NEUROPATHY(YES FOR ALL PATIENTS)	DISTAL PULSES(FELT FOR THE REST OF THE CASES)
1	55	m	2	52	R	1	60	N	Y	Y	Y	N	NORMAL	Y		N	N	Y	F
2	50	m	2	58	R	3	57	N	Y	Y	Y	N	N	Y		N	N	Y	F
3	45	f	2	54	R	5	59	Y	Y	Y	Y	Y	N	Y		N	Y	Y	F
4	35	f	2	53	R	4	54	Y	Y	Y	Y	N	N	Y		N	Y	Y	F
5	60	m	2	51	R	2	50	N	Y	Y	Y	N	N	Y		N	N	Y	F
6	38	m	2	48	I	3	48	N	N	Y	Y	N	N	Y		N	N	Y	F
7	45	m	2	49	I	5	47	N	Y	Y	Y	N	N	Y		N	N	Y	F
8	49	m	2	47	R	1	24	Y	Y	Y	Y	N	N	Y		N	Y	Y	F
9	56	f	2	48	R	3	14	Y	Y	Y	Y	N	N	Y		Y	Y	Y	F
10	59	m	2	35	R	4	19	Y	Y	Y	Y	N	N	Y		Y	Y	Y	NOT FELT
11	62	m	2	29	R	1	20	N	Y	Y	N	N	N	Y		N	N	Y	F
12	67	f	2	27	I	1	28	N	Y	Y	Y	N	N	Y		Y	N	Y	F
13	58	m	2	35	I	1	27	N	Y	Y	Y	N	N	Y		Y	N	Y	NOT FELT
14	54	m	2	34	R	1	30	N	Y	Y	Y	N	N	Y		N	N	Y	F
15	39	m	2	16	R	1	39	Y	Y	Y	Y	N	N	Y		N	Y	Y	F
16	43	f	2	18	R	4	38	Y	N	Y	Y	N	N	Y		N	Y	Y	F

17	46	m	2	19	R	3	37	Y	N	Y	Y	N	N	Y		N	Y	Y	F
18	49	f	2	20	R	5	40	Y	N	Y	N	N	N	Y		N	Y	Y	F
19	48	m	2	28	R	2	42	Y	N	Y	Y	N	N	Y		Y	Y	Y	F
20	52	m	2	24	R	1	44	N	Y	Y	N	N	N	Y		N	N	Y	F
21	57	m	2	29	R	1	48	Y	Y	Y	N	Y	N	Y		N	Y	Y	F
22	58	m	2	27	I	1	46	Y	Y	Y	Y	N	N	Y		N	Y	Y	F
23	46	m	2	26	I	4	45	Y	Y	Y	Y	N	N	Y		N	Y	Y	F
24	70	m	2	35	I	2	47	Y	Y	Y	Y	N	N	Y		N	Y	Y	NOT FELT
25	65	f	2	38	R	4	49	Y	Y	Y	Y	N	N	Y		N	Y	Y	F
26	59	f	2	45	R	3	46	N	Y	Y	Y	N	N	Y		Y	N	Y	F
27	67	f	2	46	R	5	42	N	Y	Y	Y	N	N	Y		N	N	Y	F
28	59	m	2	49	R	1	41	N	Y	Y	Y	N	N	Y		N	N	Y	F
29	68	m	2	35	R	5	25	Y	Y	Y	Y	N	N	Y		N	N	Y	F
30	64	m	2	31	I	4	26	Y	Y	Y	Y	Y	N	Y		N	N	Y	F
31	57	m	2	37	I	1	28	N	Y	Y	Y	N	N	Y		Y	N	Y	F
32	35	m	2	32	I	1	29	N	Y	Y	Y	N	N	Y		N	N	Y	F
33	40	m	2	34	I	1	34	N	Y	Y	Y	N	N	Y		N	N	Y	F
34	45	f	2	38	I	1	38	N	Y	Y	Y	N	N	Y		N	N	Y	F
35	48	f	2	39	R	3	35	N	Y	Y	Y	N	N	Y		N	N	Y	F
36	49	m	2	34	R	2	37	N	Y	Y	Y	N	N	Y		Y	N	Y	F
37	55	f	2	36	R	5	31	N	Y	Y	Y	N	N	Y		N	N	Y	F
38	59	m	2	38	R	3	39	Y	N	Y	Y	N	N	Y		N	Y	Y	F
39	57	m	2	37	R	4	37	Y	N	Y	Y	N	N	Y		N	Y	Y	F
40	33	m	2	39	I	2	34	Y	Y	Y	Y	Y	N	Y		N	Y	Y	F
41	37	m	2	47	I	N		N	N	Y	Y	N	N		Y	N		Y	F
42	39	f	2	42	R	N		N	N	Y	Y	N	N		Y	Y		Y	F
43	47	f	2	28	R	N		N	Y	Y	N	N	N		Y	N		Y	F
44	58	m	2	29	R	N		N	Y	Y	Y	N	N		Y	Y		Y	F
45	59	m	2	27	R	N		N	N	Y	Y	N	N		Y	N		Y	F
46	68	m	2	28	R	N		N	Y	Y	N	N	N		Y	N		Y	F

47	47	m	2	24	R	N		N	Y	Y	Y	N	N		Y	N		Y	F
48	49	f	2	36	R	N		N	Y	Y	Y	N	N		Y	N		Y	F
49	68	f	2	34	I	N		N	Y	Y	Y	N	N		Y	N		Y	F
50	57	f	2	38	I	N		N	N	Y	Y	N	N		Y	N		Y	F

S.NO	INITIAL SIZE	FOLLOW UP AT 1 WEEK(SIZE)	COMPLICATIONS	FOLLOW UP AT 2 WEEK(SIZE)	COMPLICATIONS	FOLLOW UP AT 1 MON(SIZE)	COMPLICATIONS	FOLLOW UP AT 2 MON	FOLLOW UP AT 4 MON	6MON	1 YEAR
1	2	1.5	N	0.5	N	0	N	CURED	CURED	CURED	CURED
2	3	2	N	1	N	0	N	CURED	CURED	CURED	CURED
3	1	0.8	N	0	N	0	N	CURED	CURED	CURED	CURED
4	2	1.5	N	1	N	0	TRANSFER ULCERS	CURED+TRANSFER ULCERS	CURED+TRANSFER ULCERS	CURED+TRANSFER ULCERS	CURED+TRANSFER ULCERS
5	3	2.2	N	1.8	N	0.5	N	CURED	CURED	CURED	CURED
6	2	1.6	N	1	N	0	N	CURED	CURED	CURED	CURED
7	3	2.3	N	1.8	N	0.8	N	CURED	CURED	CURED	CURED
8	2	0.8	N	0	N	0	N	CURED	CURED	CURED	CURED
9	1	0.4	N	0	N	0	N	CURED	CURED	CURED	CURED
10	2	1.6	N	1.1	N	0	N	CURED	CURED	CURED	CURED

11	2	1.7	N	1.1	N	0	N	CURED	CURED	CURED	CURED
12	3	2.4	N	2	N	1.2	N	0.5	CURED	CURED	CURED
13	2	1.6	N	0.9	N	0	N	CURED	CURED	CURED	CURED
14	4	2.8	N	2	N	1	TRANSFER ULCERS	0.8+TRANSFER ULCER	CURED+TRANSFER ULCER	CURED+TRANSFER ULCER	CURED+TRANSFER ULCER
15	2	1.4	N	0.9	N	0	N	CURED	CURED	CURED	CURED
16	1	0.4	N	0	N	0	N	CURED	CURED	CURED	CURED
17	2	1.4	N	0.8	N	0	N	CURED	CURED	CURED	CURED
18	2	1.6	N	1	N	0	N	CURED	CURED	CURED	CURED
19	3	2.4	N	1.8	N	0.8	N	CURED	CURED	CURED	CURED
20	3	2.7	N	2	N	1.2	N	CURED	CURED	CURED	CURED
21	3	2.2	N	2.5	N	2.5	AMPUTATION	AMPUTATION	AMPUTATION	AMPUTATION	AMPUTATION
22	4	3.4	N	2.9	N	1.9	N	1	CURED	CURED	CURED
23	1	0.5	N	0	N	0	N	CURED	CURED	CURED	CURED
24	3	2.3	N	1.7	N	0	N	CURED	CURED	CURED	CURED
25	2	1.5	N	0.8	N	0	N	CURED	CURED	CURED	CURED
26	2	1.5	N	0.5	N	0	N	CURED	CURED	CURED	CURED
27	3	2	N	1.4	N	0.4	N	CURED	CURED	CURED	CURED
28	2	1.4	N	0.4	N	0	N	CURED	CURED	CURED	CURED
29	1	0.5	N	0	N	0	N	CURED	CURED	CURED	CURED
30	2	1.3	N	0.8	N	0	N	CURED	CURED	CURED	CURED
31	3	2.3	N	1.5	N	0	N	CURED	CURED	CURED	CURED
32	4	3.3	N	3	N	1.5	N	0.5	CURED	CURED	CURED
33	2	1.3	N	0.5	N	0	N	CURED	CURED	CURED	CURED
34	4	3.2	N	2.7	N	1.6	N	0.7	CURED	CURED	CURED
35	2	1.4	N	0.9	N	0	N	CURED	CURED	CURED	CURED

36	1	0.5	N	0	N	0	TRANSFER ULCERS	CURED+TRANSFER ULCERS	CURED+TRANSFER ULCERS	CURED+TRANSFER ULCERS	CURED+TRANSFER ULCERS
37	2	1.4	N	0.8	N	0	N	CURED	CURED	CURED	CURED
38	3	2.4	N	1.7	N	0.4	N	CURED	CURED	CURED	CURED
39	3	2.5	N	1.2	N	0	N	CURED	CURED	CURED	CURED
40	3	2.5	N	2.2	N	2	N	1	CURED	CURED	CURED
41		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
42		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
43		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
44		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
45		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
46		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
47		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
48		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
49		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER
50		NOULCER	N		N		N	NO ULCER	NO ULCER	NO ULCER	NO ULCER