ANNEXURE I

INFORMED CONSENT

TO COMPARE EFFICACY OF VACCUM-ASSISTED CLOSURE (VAC) THERAPY VERSUS CONVENTIONAL POVIDONE IODINE DRESSING IN THE MANAGEMENT OF DIABETIC FOOT ULCERS- A RANDOMISED CONTROL TRIAL.

Principal Investigator:-

Professor
Department Of General Surgery,
J. N. Medical College, Belgaum.

Co-investigator:-

Post Graduate Student
Department Of General Surgery,
J. N. Medical College, Belgaum.

INTRODUCTION AND PURPOSE:

The diabetic ulcers are a common complication of diabetes mellitus and treatment of which is a major challenge. Presently these ulcers are being managed by local dressings with agents like Povidone ,Eusol etc. using moist gauze and pads have their own limitations.VAC (VACCUM ASSISTED CLOSURE) Therapy may represent an alternative to the currently available method of dressing and may answer the quest for better control of wound infection in diabetic patients. So, the study has been undertaken to compare the efficacy of VAC Therapy in management of diabetic foot ulcers.

You are requested to participate in a study which is an attempt to find out the efficacy of Vaccum Assisted Closure (VAC) Therapy as compared to conventional

povidone iodine dressing in the healing of diabetic foot ulcers-A randomized control trial.

This study will be conducted by _____,Post Graduate in Department of Surgery, under the direct supervision and guidance of _____, Associate Professor , Department of Surgery, J. N. Medical College, Belgaum.

PROCEDURE:

You need to be eligible, meeting all the selection criteria to participate in this study. You should be willing to provide information about yourself. 30 subjects will be enrolled in this study that will then be randomized in either of 2 groups (details given below).

If you agree to participate in this study, you will be randomly allotted into a group (A or B) and accordingly receive either the standard management (dressing with povidone iodine) or the newer management (VAC Therapy). The ulcer will be assessed before start of dressing on day 0,day 7 and on day of discharge/day 14.

BENEFITS/RISKS:

It applies controlled, localized negative pressure to help uniformly draw wounds closed.Helps remove interstitial fluid allowing tissue decompression.Helps remove infectious material.Assists granulation.There is no observable risk associated with this study.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No financial incentives are being offered to enrolled subjects. It is purely being done with the idea of research purpose and all cost of the study will be borne by the investigator. In the event that you become injured as a result of taking part in this study, treatment will be offer ed to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.,or you will be given information about where to receive medical care in which case you/your insurance company will be responsible for the costs. However, no reimbursement, compensation or free medical care will be given.

AUTHORIZATION TO PUBLISH RESULTS:

The results of the study may be used to publish an article.When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

CONFIDENTIALITY:

Every effort will be made to protect the confidentiality of the information you provide. Only _____and _____ will have access to the information provided by you . Results of this study may be published but your identity will not be revealed.

VOLUNTARY PARTICIPATION / WITHDRAWING / REMOVAL FROM THE STUDY:

Taking part in this study is voluntary; you may choose not to enroll in this study. Your decision will not change the present or future health care services offered to you at KLES Dr. Prabhakar Hospital, Belgaum. The alternative that you have is to undergo the traditional procedure that is carried out in KLES Hospital.

ALTERNATIVES:

Even if you decline the participation in study, you will get the routine line of management.

CONTACT DETAILS:

If you have any queries about the study, you may contact Co-investigator, _____ , post graduate student, J.N.M.C., Belgaum / Principal investigator, _____Professor , Department of Surgery, J. N. Medical College, Belgaum ,without any hesitation.

In case you need any further information regarding your rights as a study participant, you may contact _____, Prof of Pathology and HOD and Chairman of College Ethical Dissertation and Research Committee, JNMC, Belgaum, phone no: _____.

ANNEXURE II

DATA COLLECTION INSTRUMENT

ID No.

Date of admission_____

Date of interview_____

1. Name:_____

2. Age: 1: <20 years 2: 20-40 years 3: >40 years

3. Sex: 1-Male 2-Female

4. Address: 1-Belgaum 2-Outside Belgaum

5. Occupation: 1-Unemplyoed
   2-Unskilled
   3-Semi-skilled
   4-Skilled
   5-Professional

6. Education: 1-Illiterate
   2-Primary (1st-7thstd)

3-High school (8^{th} -10^{th}std)

4-Intermediate

5-Degree and above

7. Socio-economic status:

1-Low 2-Middle 3-High

Screening

8.H/o Diabetes: 1-Yes 2-No

9. If answer to 8 is yes, current status: 1-Under control 2-Uncontrolled

3-Not applicable

10.On medication for diabetes: 1-Yes 2-No 3-Not applicable

11. H/o Hypertension? 1-Yes 2-No

12. If Yes to Q11, current status: 1-Controlled 2-Uncontrolled

3-Not applicable

13.H/o recent intake of steroids : 1-Yes 2-No

14. If answer to 13 is yes, duration of intake:

1. less than 6 months 2. 6 to12 months 3. >12 months

4. Not applicable

15. H/o serious systemic illness: 1-Yes 2-No

16. If answer to 15 is yes, which system involvement:

1-GIT 2-Renal and genitourinary 3-Respiratory 4-Cardiac

5-CNS 6-Immune system related 7-Others 8-Not applicable

17. Level of hemoglobin: 1: Less than 10gm% 2: 10 gm% 3: >10gm%

18. Applicant willing to give consent? 1-Yes 2-No

19. Final result

1-Ineligible 2-Eligible but refused 3-Eligible and participating

20. Chief complaint: 1. History of trauma to foot 2.Wound 3.ulcer over leg

4. Fever5. Others 6. More than 1 complaint

21. 1. Mode of onset

1-Traumatic

2-Spontaneous

3-Pressure

4-Others

22. Duration of ulcer: 1: <1 week 2: 1-4 weeks 3: >4 weeks

23. Received any treatment elsewhere for same complaint(s):

1-Yes 2-No

24. If yes to Q.23, Surgical or medical treatment? 1-Surgical 2-Medical

3-Not applicable

25. Outcome of treatment? 1-Not improved 2-Improved

3-Improved with recurrence of symptoms

4-Not applicable

26. Method of dressing A or B? 1-A 2-B

27. WOUND OBSERVATIONS :

	Before dressing Day 0	Day 5	Day of discharge(if the >95% red granulation is present) /day 14
1. Site			
2. Size (greatest dimension) 1- <5cms. 2- 6-10cms. 3- 11-15cms.			
3. Shape 1- Oval 2- Circular 3- Irregular			
4. Edge 1.Indistinct, diffuse = unable to clearly distinguish wound outline. 2.Attached = even or flush with wound base, no sides or walls present; flat. 3.Not attached = sides or walls are present; floor or base of wound is deeper than edge. 4.Rolled under, thickened = soft to firm and flexible to touch 5.Hyperkeratosis = callous-like tissue formation around wound and at edges 6.Fibrotic, scarred = hard, rigid to touch			
5. Margin 1-Regular 2-Irregular			
6. Floor 1-Red granulation tissue 2-Pale yellow granulation tissue 3.Other than ½			
7. Base 1-Fascia 2-Soft tissue 3-Bone			
8. Discharge 1.Bloody = thin, bright red 2.Serosanguineous = thin, watery, pale red to pink			

3.Serous = thin, water, clear 4.Purulent = thin or thick, opaque tan to yellow 5.Foul purulent = thick, opaque yellow to green with offensive odor			
9. Surrounding Skin 1-Oedema 2-Eczema 3-Pigmentation 4.None			
10. Slough /necrotic tissue 1.White/gray non-viable = may appear prior to wound opening; skin surface is white or gray. 2.Non-adherent yellow slough = thin, mucinous substance; scattered throughout wound bed; easily seperated from wound tissue. 3.Loosely adherent yellow slough = thick, stringy clumps of debris; attached to wound tissue. 4.Adherent, soft black eschar = soggy tissue; strongly attached to tissue in center or base of wound. 5.Firmly adherent, hard black = firm, crusty tissue; strongly attached to wound base and edges (like a hard scab). 11. Area of the ulcer			

12.Culture and sensitivity report	DAY 0 Before dressing	DAY 14/DAY OF DISCHARGE
1-No growth 2-Growth present (If 2,mention name of the isolated organism)		

29.ANY FOOT DEFORMITY PRESENT

Toe deformity : ☐

Charcot's foot: ☐

Foot drop: ☐

1-Present

2-Absent

30.IFDEBRIDEMENT HAS BEEN DONE

Specify, Date :

Type of anaesthesia:

No of debridements:

INVESTIGATIONS :

CBC :Hb- ( ) TLC- ( ) DC- (N- , L- , M- ,E-)

FBS : ( ) Date Time

Blood Urea ( )

Sr. Creatinine ( )

Urine :

Routine

Microscopy

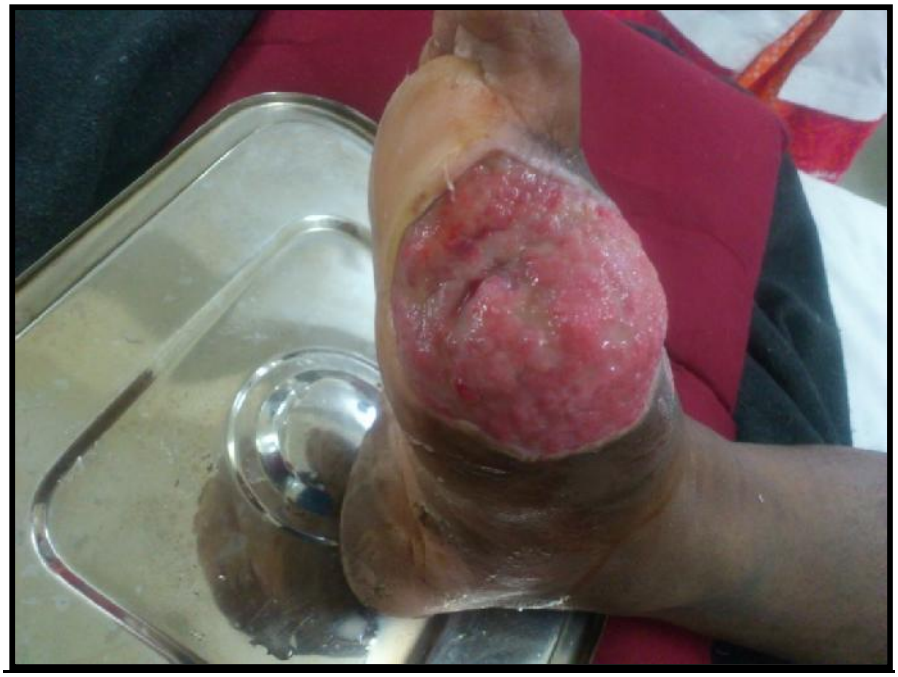
X-ray

AP view :

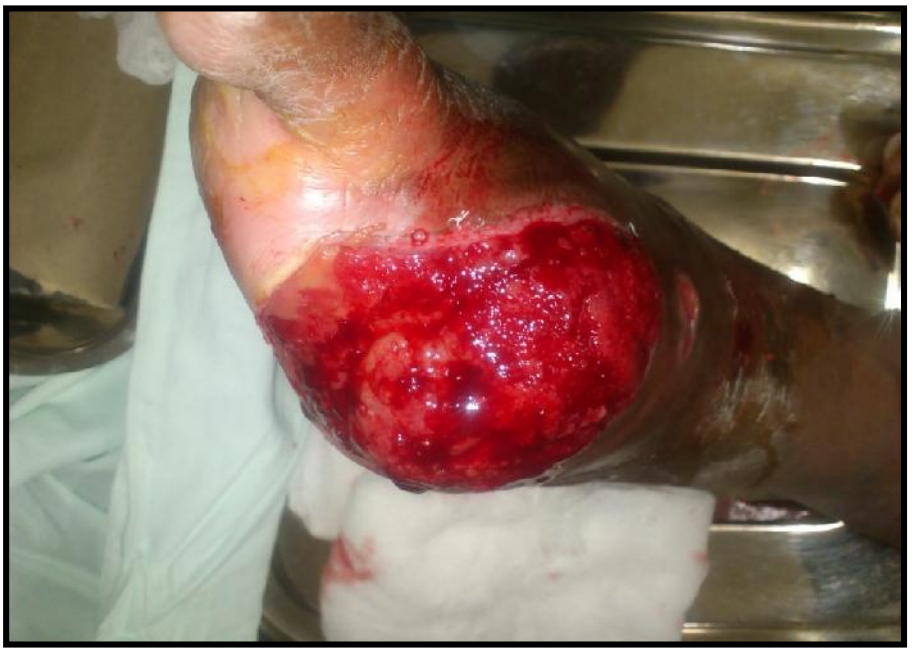
Lat. View :

COLOUR DOPPLER (if ischemia/PVD is suspected) :

ANNEXURE III



PHOTOGRAPH 3: DIABETIC FOOT ULCER ON DAY 0 BEFORE APPLICATION OF VACCUM ASSISTED CLOSURE THERAPY.



PHOTOGRAPH 4:DIABETIC FOOT ULCER ON DAY 14 AFTER APPLICATION VACCUM ASSISTED CLOSURE THERAPY.

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By

REG NO. BH0112003

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
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DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

APRIL – 2015

KLE UNIVERSITY, BELGAUM,

KARNATAKA

ENDORSEMENT

This is to certify that the dissertation entitled **"TO COMPARE EFFICACY OF VACCUM-ASSISTED CLOSURE (VAC) THERAPY VERSUS CONVENTIONAL POVIDONE IODINE DRESSING IN THE MANAGEMENT OF DIABETIC FOOT ULCERS- A RANDOMISED CONTROL TRIAL"** is a bonafide research work done by **(REG NO. BH0112003)**

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LIST OF ABBREVIATIONS USED

VAC	-	Vaccum Assisted Closure
VACT	-	Vaccum Assisted Closure Therapy
CTPID	-	Conventional Topical Povidone Iodine Dressing
PDGF	-	Platelet-derived growth factor
TFGB	-	Transforming growth factor beta
PMNs	-	Polymorphonuclear leukocytes
IL-1	-	Interleukin-1
TNF-α	-	Tumor necrosis factor alpha
VEGF	-	Vascular endothelial growth factor
IGF-1	-	Insulin-like growth factor 1
TGF-α	-	Transforming growth factor α
TGF-β	-	Transforming growth factor β
IL-8	-	Interleukin-8
FGF	-	Fibroblast Growth Factor
KGF	-	Keratinocyte growth factor
TIMP	-	Tissue inhibitors of metalloproteinase
HEMA	-	Hydroxyethyl methacrylate
PHEMA	-	Poly Hydroxyethyl methacrylate
RGD	-	Arginine-glycine aspartic acid peptide
PZT	-	Lead zicornate titanate
LILT	-	Low Intensity Laser Therapy
GIT	-	Gastro-intestinal system
CNS	-	Central Nervous System

H/O	-	History of
CBC	-	Comple blood count
TLC	-	Total Leukocyte Count
DC	-	Differential count
N	-	Neutrophils
L	-	Lymphocyte
M	-	Monocytes
B	-	Basophils
E	-	Eosinophils
Sr.	-	Serum
PVD	-	Peripheral Vascular Disease
FBS	-	Fasting blood sugar
Hb	-	Haemoglobin
AP view	-	Antero-posterior view
cm	-	centimeter
YRS	-	Years
SAB	-	Sub arachnoid block

ABSTRACT

Background and objectives

Diabetes mellitus is a group of metabolic disorders characterized by elevated levels of glucose in the blood (hyperglycemia) resulting from defects in insulin secretion, insulin action or both. Diabetic foot ulcers are the single biggest risk factor for non traumatic foot amputations in persons with diabetes.The study was carried out to assess efficacy of vaccum assisted closure therapy (VACT) versus conventional topical povidone iodine dressing in diabetic foot ulcers.

Methodology

This one year randomized controlled trial was done from January 2013 to December 2013 at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 60 patients with diabetic foot ulcers were enrolled.

- Group A – receive Vaccum Assisted Closure (VAC) Therapy.
- Group B –receive Conventional Topical Povidone Iodine Dressing (CTPID).

Results

The age and sex distribution between both the groups were statistically not significant. The mean surface area of an ulcer on day 14 was 11.30cm^2 in CTPID and 8.6cm^2 in VACT group. This decrease in surface area on day 14^{th} was statistically significant (P value of 0.029). Repeat culture on day 14^{th} ,22 patients showed growth in CTPID group whereas in VACT group only 2 patients had positive growth. When compared,there was statistically significant difference

between CTPID and VACT group on day 14th.Thus application of VAC helps to reduce infection status of an ulcer.

Conclusion and interpretation

Thus we conclude that mean surface area of the ulcer is reduced and infection is controlled better in the VAC Therapy, making VAC a superior option in the management of patients with Diabetic foot Ulcers.

Keywords

Diabetic Foot Ulcers, Vaccum Assisted Closure Therapy, Conventional Topical Povidone Iodine Dressing , Wound healing

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INTRODUCTION

Diabetes mellitus is a group of metabolic disorders characterized by elevated levels of glucose in the blood (hyperglycemia) resulting from defects in insulin secretion, insulin action or both.[1]

Diabetes is an ice berg disease, according to recent estimates the prevalence of diabetes mellitus in adult was around 4th world wide and this means that over 150 million persons now affected.[2]

Diabetes is a serious chronic disease. In 2003 the global prevalence of diabetes was estimated at 194 million. This figure is predicted to reach 333 million by 2025 as a consequence of longer life expectancy, sedentary lifestyle and changing dietary patterns. This rise is likely to bring a proportional increase in the numbers of people with diabetes complications, including problems of the foot. Extensive epidemiological surveys have indicated that between 40% and 70% of all lower extremity amputations are related to diabetes. This means that every 30 seconds a lower limb is lost to diabetes. The vast majority (85%) of all diabetes-related amputations are preceded by foot ulcers. India is home to some 50 million diabetics and 15% of this population suffer or are likely to suffer from the dreadful complication of diabetes including the diabetic foot. Up to 85% of all amputations in people with diabetes are preceded by foot ulcers[3,4].

Diabetic foot ulcers are the single biggest risk factor for non traumatic foot amputations in persons with diabetes. The peculiar characteristic is the refusal of the diabetic foot ulcer to heal despite the best wound care management given. The false notion that the wounds heal better if kept dry has given way to the newer concept of wound healing which allows chronic wounds to re-epithealize

much faster or develop granulation tissue much faster when treated with moist wound dressings.

Diabetic foot ulcer is the most frequent reason for hospitalization in patients with diabetes and is a major cause of morbidity and excess hospital care cost for the patients. Though there are many modalities of wound care, the problem of treating diabetic wounds is still enormous.

Many techniques have been tried over the centuries to heal chronic leg ulcers. Although there exists no ideal wound dressing, the management of chronic wounds especially diabetic foot ulcers has seen many new developments. The traditional moist dressings were initially supplemented with hydrocolloid dressings, gels, foams, and other measures like hyperbaric oxygen, laser stimulation, vaccum assisted closure therapy, growth factors, and various offloading therapies.

OBJECTIVE:

To compare efficacy of vaccum-assisted closure (vac) therapy versus conventional povidone iodine dressing by measuring surface area and infection status of an ulcer.

REVIEW OF LITERARTURE

HISTORY:

The Ebers Papyrus, circa 1500 BC, details the use of lint, animal grease, and honey as topical treatments for wounds. The lint provided a fibrous base that promoted wound site closure, the animal grease provided a barrier to environmental pathogens, and the honey served as an antibiotic agent[5].

Hippocrates, the legendary father of medicine himself had a leg ulcer. That was the age when anaesthesia, anatomy and physiology were never heard of. He was against treating various ulcerations by surgical means. He treated multiple varicose veins by puncturing them at different levels to avoid non-healing of ulcers and about 400 years B.C. He wrote - “In case of an ulcer, it is not expedient to stand, especially if the ulcer be situated on the leg"[6]

From the Classical Period to the Medieval Period, the body and the soul were believed to be intimately connected, based on several theories put forth by the philosopher Plato. Wounds on the body were believed to correlate with wounds to the soul and vice versa; wounds were seen as an outward sign of an inward illness[7].

From the 10th to 18th centuries various physicians including Halu, Abbas, Avicenna, Falopio and Pare attributed ulceration of the leg to accumulation of black bile or bad tumours and believed that ulceration of the leg served useful purpose in getting rid of these live Substances[8].

The work of Joseph Lister and Louis Pasteur established a sound basis for the management of infection by identifying the cause and developing methods for preventing it. Joseph Lister advocated cleanliness in the hospital, the frequent use of soap and water on wounds and carbolic acid dressings of contaminated wounds. Later Semmelweis, Ehrlich, Fleming, and Florey also realized that bacteria were pathogens.

Control of bacteria by asepsis, antiseptics and antimicrobials heralded a new era in wound management. World War I resulted in rapid discoveries surrounding the care of wounds, the foremost among these being the use of extensive debridement .[9]

Antibiotics, understanding fluid and blood replacement, and control of pain were the major contributions to wound management during the first quarter of the twentieth century. During the past two decades, wound care has made more advances than over the past two thousand years. This is because of five major factors :

1. The biologic mechanisms of repair are now being defined on an anatomic, a biochemical and a molecular level.
2. The social and financial impacts of chronic wounds is finally being appreciated by health care providers and governmental agencies.
3. There is more extensive research in wound healing as the discovery of effective treatment modalities are commercially profitable.
4. The development of new pharmacologic agents that enhance the healing of both acute and chronic wounds through the breakthroughs in molecula biology , and
5. Better reconstructive surgical techniques with the advent of muscular an musculocutaneous flaps as well as microvascular free-tissue transfers.[10]

It was not until the 1960's that the concept of moist wound environment for healing was considered. In addition to protecting the wound from infection, the moist environment would help to facilitate debridement, minimize inflammation, reduce pain and diminish scarring [11].

ANATOMY OF SKIN:

The skin covers the entire external surface of the human body and is the principal site of interaction with the surrounding world. It serves as a protective barrier that prevents internal tissues from exposure to trauma, ultraviolet (UV) radiation, temperature extremes, toxins, and bacteria. Other important functions include sensory perception, immunologic surveillance, thermoregulation, and control of insensible fluid loss.

The integument consists of 2 mutually dependent layers, the epidermis and dermis, which rest on a fatty subcutaneous layer, the panniculus adiposus. The epidermis is derived primarily from surface ectoderm but is colonized by pigment-containing melanocytes of neural crest origin, antigen-processing Langerhans cells of bone marrow origin, and pressure-sensing Merkel cells of neural crest origin. The dermis is derived primarily from mesoderm and contains collagen, elastic fibers, blood vessels, sensory structures, and fibroblasts.[12]

ANATOMY OF FOOT:

The human foot combines mechanical complexity and structural strength. The ankle serves as foundation, shock absorber and propulsion engine. The foot can sustain enormous pressure (several tons over the course of a one-mile run) and provides flexibility and resilience.

The foot and ankle contain: [13]

- 26 bones (one-quarter of the bones in the human body);
- 33 joints;
- More than 100 muscles, tendons, and ligaments.
- A network of blood vessels, nerves, skin and soft tissue.

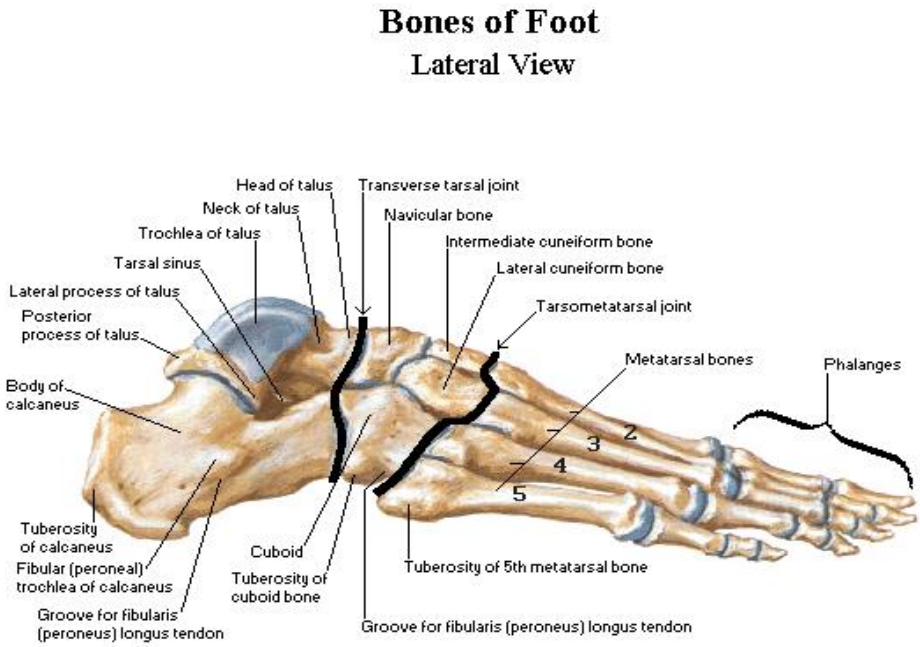


FIGURE 1 : BONES OF FOOT

The foot is connected to the leg by the ankle (talocrural) joint, which is an articulation between the tibia, fibula, and talus. The foot consists of 7 tarsal bones, 5 metatarsal bones, and 14 phalanges. Motion at the digits for abduction and adduction is defined by an imaginary line along the long axis of the second digit, unlike the hand

in which the long axis runs along the third digit. Each digit, with the exception of the great toe, consists of three phalanges (proximal, middle, and distal); the great toe has two phalanges (proximal and distal). The articulations between the bones of the foot create multiple joints. The muscles that move these joints are divided into two groups, intrinsic and extrinsic foot muscles. The intrinsic muscles originate and attach in the foot, whereas the extrinsic muscles originate in the leg and insert in the foot, creating motion at multiple joints.

Joints of the Digits

The several bony articulations within the foot assist in accommodating uneven surfaces during weight-bearing activities. These motions of the foot are accomplished via the following joints (Figure 2)

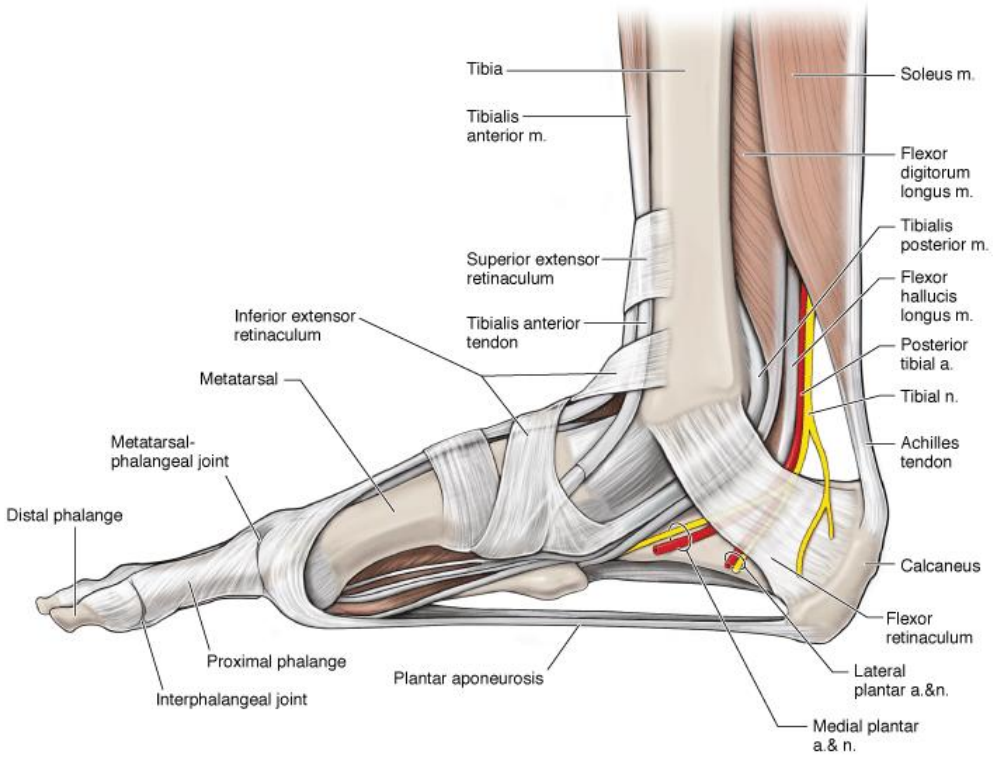


Figure 2: Medial view of the fascia of the right foot.

Fascial Structures of the Foot :

- **Plantar aponeurosis.** Radiates from the **calcaneus bone** toward the digits. The plantar aponeurosis is a very thick fascia that invests the muscles of the plantar surface of the foot.
- **Superior extensor retinaculum.** Attaches from the anterior border of the fibula to the tibia, proximal to the ankle joint. The superior extensor retinaculum holds the tendons of the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and fibularis (peroneus) tertius muscles next to the structures of the anterior ankle during contraction.
- **Inferior extensor retinaculum.** A "Y-shaped" structure that attaches laterally to the superior surface of the calcaneus bone and courses medially to attach to the medial malleolus and the medial side of the plantar aponeurosis. The inferior extensor retinaculum serves to tether the tendons of the fibularis (peroneus) tertius, extensor digitorum longus, extensor hallucis longus, and anterior tibialis muscles.
- **Flexor retinaculum.** Attaches between the medial malleolus and calcaneus bones, forming the roof of the **tarsal tunnel**. The tendons of the tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles as well as tibial nerve and posterior tibial artery pass through the tarsal tunnel to the enter into the plantar surface of the foot.
- **Fibular retinacula.** Tethers the tendons of the fibularis (peroneus) longus and brevis muscles on the lateral side of the ankle as they course inferior to the lateral malleolus bone.
- **Dorsal digital expansions.** An aponeurosis covering the dorsum of the digits that attaches proximally to the middle phalanx (digits 2–5) or proximal

phalanx (digit 1), via the central band, and distally to the distal phalanx, via the lateral bands. The extensor digitorum longus and brevis muscles and the extensor hallucis longus and brevis muscles attach proximally and centrally to the dorsal digital expansion. The lumbricals and the dorsal and plantar interossei attach on the free edges. Because of the attachment of the muscles and the location of the dorsal digital expansion, the small intrinsic muscles produce flexion at the metatarsophalangeal joint while extending the interphalangeal joints. [14]

Parts of the Foot:

Structurally, the foot has three main parts:

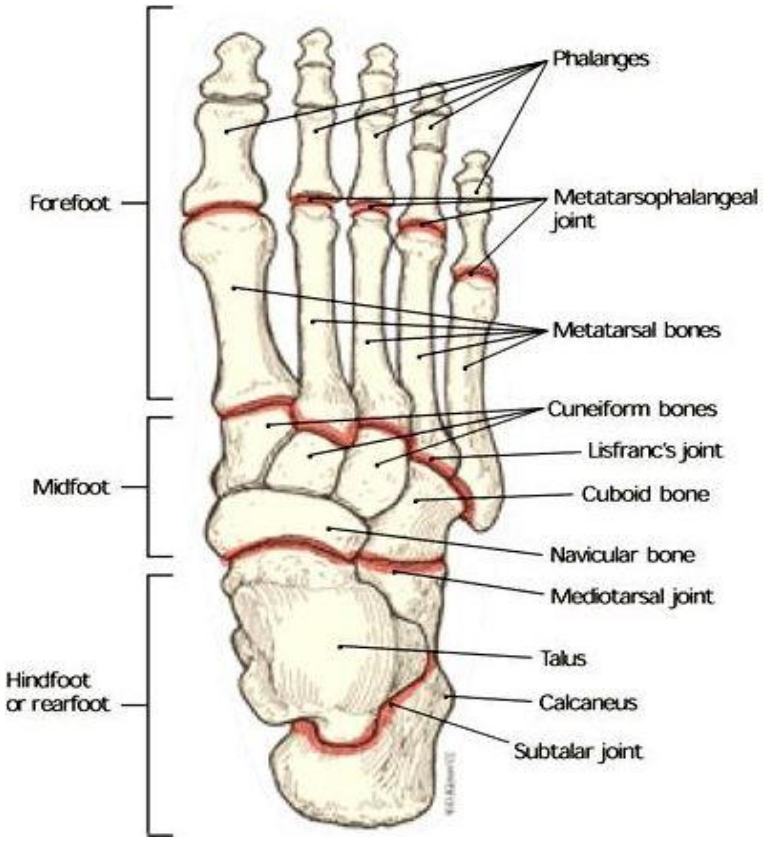


FIGURE 3 : PARTS OF THE FOOT

The forefoot: Is composed of the five toes (called phalanges) and their connecting long bones (metatarsals). Each toe (phalanx) is made up of several small bones. The big toe (hallux) has two phalanges, two joints (interphalangeal joints), and two tiny, round sesamoid bones that enable it to move up and down. The other four toes each have three bones and two joints. The phalanges are connected to the metatarsals by five metatarsal phalangeal joints at the ball of the foot. The forefoot bears half the body's weight and balances pressure on the ball of the foot.

The midfoot: forms the foot's arch, and serves as a shock absorber. The bones of the midfoot are cuboid, first, second, third cuneiform and navicular connected to the fore foot and the hind foot by muscles and the plantar fascia.

The hind foot: Is composed of three joints and links the midfoot to the ankle (talus). The top of the talus is connected to the two long bones of the lower leg (tibia and fibula), forming a hinge that allows the foot to move up and down. The heel bone (calcaneus) is the largest bone in the foot. It joins the talus to form the subtalar joint, which enables the foot to rotate at the ankle. The bottom of the heel bone is cushioned by a layer of fat.

The Arches: The foot has three arches. The medial longitudinal arch is composed of the calcaneus, talus, navicular, cuneiforms, and the first three metatarsals. The lateral longitudinal arch is composed of the calcaneus, cuboid and the fourth and fifth metatarsals. The transverse arch is composed of the cuneiforms, the cuboid and the five metatarsal bases. The arches of the foot are maintained not only by the shapes of the bones as well as by ligaments. In addition, muscles and tendons play an important role in supporting the arches[15].

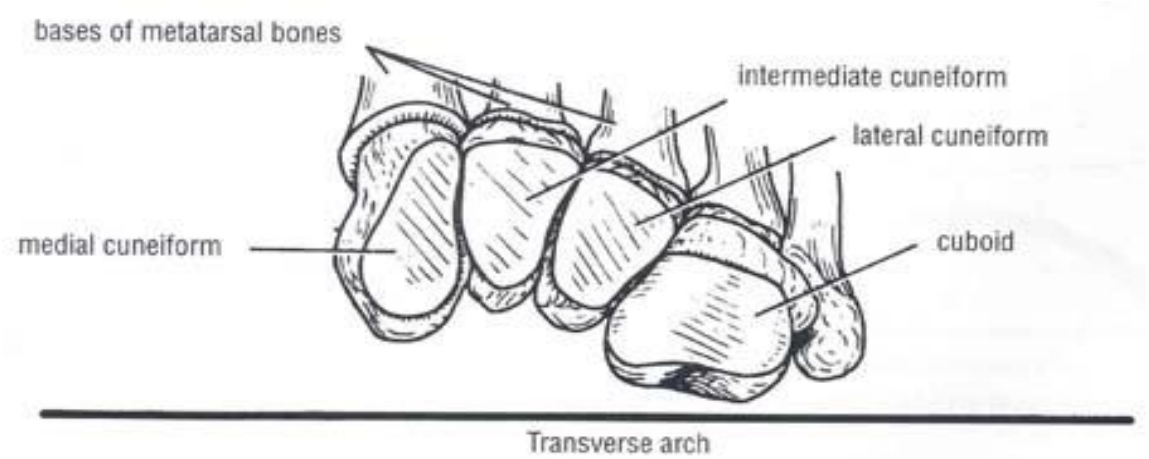


FIGURE 4 : TRANSVERSE ARCH

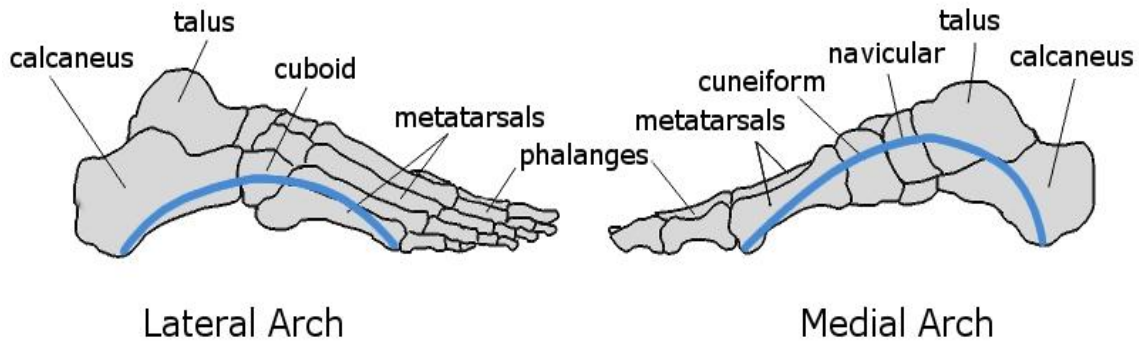


FIGURE 5 : LONGITUDINAL ARCH: LATERAL AAND MEDIAL

The muscles that control the major functions of the foot have their origin in the leg. The posterior muscles include the tibialis posterior, flexor digitorum longus, flexor hallucis longus, plantaris, and calcaneal or Achilles tendon (formed by the gastrocnemius and soleus muscles). The lateral muscles include the peroneus longus and peroneus brevis. The anterior muscles include the tibialis anterior, extensor hallucis longus, and extensor digitorum longus. As individuals age and chronic disease becomes evident, there is a decrease in muscle strength that can result in ambulatory dysfunction, imbalance, foot drop, and a higher risk for falls.

The muscle on the dorsum of the foot is the extensor digitorum brevis. The muscles on the plantar surface of the foot are covered by the plantar fascia and are organized in four layers from superficial to deep. The muscles of the deepest fourth layer include the dorsal interossei (four), plantar interossei (four), and the tendons of the peroneus longus and tibialis posterior muscles. It is the atrophy of the interossei muscles that is a major factor in the development of hammertoes in the aging patient.

LAYERS OF THE SOLE					
MUSCLE	**ORIGIN**	**INSERTION**	**NERVE SUPPLY**	**ACTION**	**COMMENTS**
FIRST LAYER					
Abductor Hallucis	Calcaneal tuberosity medial process	Proximal Phalanx of Great Toe	Medial Plantar Nerve	Abducts the Great Toe	Supports longitudinal arch medially
Flexor digitorum brevis [FDB]	Calcaneal tuberosity medial process	Sides of middle Phalanx of lateral 4 toes	Medial Plantar Nerve	Flex lateral 4 toes	Supports longitudinal arch
Abductor digiti minimi [ADM]	Calcaneal tuberosity med. & lat. process	Lateral base of proximal phalanx: 5th toe	Lateral Plantar Nerve	Abducts small toe	Supports longitudinal arch laterally
SECOND LAYER					
Quadratus plantae	Medial and lateral plantar calcaneus	Lateral FDL tendon	Lateral Plantar Nerve	Assists FDL with toe flexion	Two heads/bellies join on FDL tendon
Lumbricals	Separate FDL tendons	Proximal phalanges, extensor expansion	1. Medial plantar 2-4. Lateral plantar	Flex MTP joint, extend IP joint	1st lumbrical attaches to 1 FDL tendon
THIRD LAYER					
Flexor hallucis brevis [FHB	Cuboid, lateral cuneiform	Proximal Phalanx of Great Toe	Medial plantar	Assist great toe flexion	Sesamoid bones attach to each tendon
Adductor hallucis	Oblique: base 2-4 MT Transverse: Lateral 4MT	Proximal Phalanx of Great Toe	Lateral Plantar Nerve	Adducts great toe	Supports transverse arch
Flexor digiti minimi brevis [FDMB]	Base of 5th metatarsal	Base of proximal phalanx small toe	Lateral Plantar Nerve	Flex small toe	Small, relatively insignificant muscle
FOURTH LAYER					
Plantar interossei (3)	Med. 3, 4, 5th MTs	Medial proximal phalanges: toes 3-5	Lateral Plantar Nerve	Adduct toes (*PAD*)	Attachment to MT is medial for all 3
Dorsal interossei (4)	Adjacent MT shafts	Proximal phalanges toes 2-5	Lateral Plantar Nerve	Abduct toes (*DAB*)	Larger than the plantar interossei muscles

LAYERS OF SOLE OF A FOOT [16]

The nerves that control the foot include the lateral sural cutaneous, superficial and deep fibular (peroneal), medial dorsal cutaneous, dorsal digital, tibial, lateral calcaneal, medial plantar, lateral plantar, and saphenous nerves.

The arterial supply to the foot follows the femoral artery, to the popliteal artery, and divides to become the anterior and posterior tibial arteries. The anterior tibial artery becomes the dorsalis pedis artery on the dorsum of the foot, whose terminal branches are the dorsal metatarsal and dorsal digital arteries. The posterior tibial artery becomes the medial and lateral plantar arteries in the foot. The lateral calcaneal artery supplies the lateral segment of the calcaneal area of the foot. The

venous return includes the dorsal venous arch, plantar cutaneous venous plexus, medial and lateral plantar veins, and the greater and lesser saphenous veins.

The skin of the foot is made up of fitted, flexible, elastic inner dermis, covered by a much less sensitive outer epidermis. The skin varies in thickness from one-half a millimeter (as in the eyelid) to 4 or 5 mm, in the sole of the foot. The skin of the foot undergoes many changes during the aging process. It becomes thinner, even parchment like; loses its elasticity, usually atrophies; and loses hair. It loses its hydration or water content because there is generally less perspiration and lubrication. The skin loses its suppleness, becomes brittle and dry, and injures easily. This condition, accompanied by a diminished blood supply, can be quite serious.

The normal toenail includes the nail matrix, nail bed, nail plate, lunula, eponychium, cuticle, and nail folds. The nail plate is a sheet of keratin and is important in clinical medicine, as it reflects the health events of the previous months. The nail plate grows slowly forward until it breaks free of the nail bed at the free edge. The tissue below the free edge is termed the hyponychium. The matrix is the area under the proximal nail fold, cuticle, and lunula. The arterial supply to the nail bed stems from small arterial branches from the dorsal and plantar anastomoses. Nail growth is affected by age, trauma, chemicals, environment, and disease.[17]

WOUND HEALING

Normal wound healing follows a predictable pattern that can be divided into overlapping phases defined by characteristic cellular populations and biochemical activities: (a) hemostasis and inflammation, (b) proliferation, and (c) maturation and remodeling.

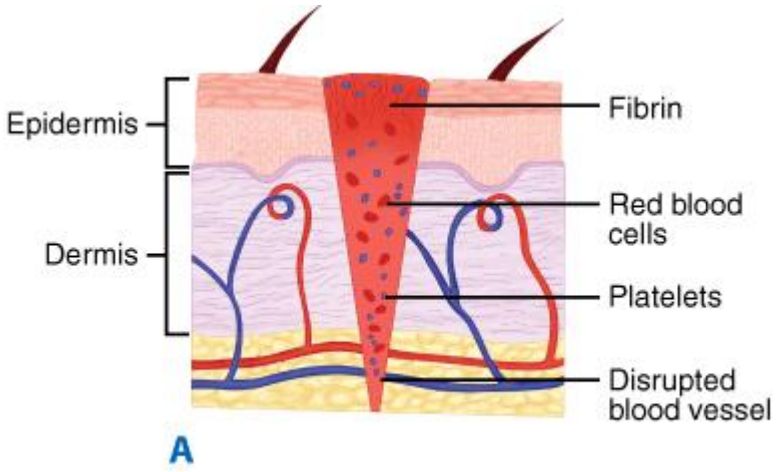
This sequence of events is fluid and overlapping, and in most circumstances spans the time from injury to resolution of acute wounds. All wounds need to progress through this series of cellular and biochemical events that characterizes the phases of healing to successfully re-establish tissue integrity.

Hemostasis and Inflammation

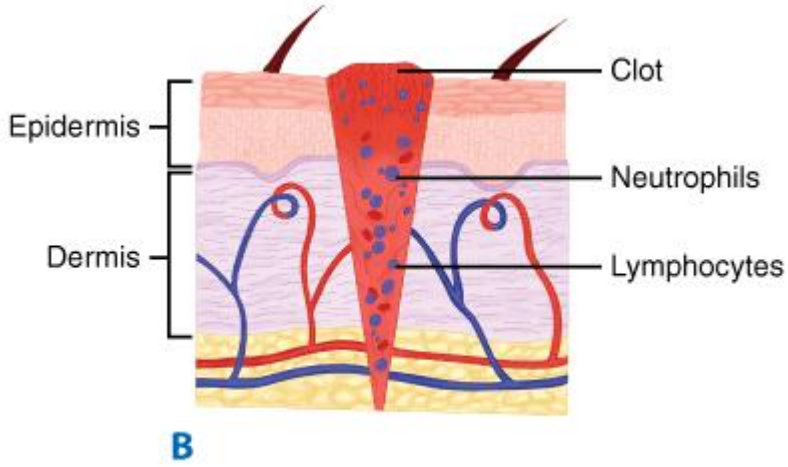
Hemostasis precedes and initiates inflammation, with the ensuing release of chemotactic factors from the wound site. Wounding by definition disrupts tissue integrity, leading to division of blood vessels and direct exposure of extracellular matrix to platelets. Exposure of subendothelial collagen to platelets results in platelet aggregation, degranulation, and activation of the coagulation cascade. Platelet α-granules release a number of wound-active substances, such as platelet-derived growth factor (PDGF), transforming growth factor beta (TGFβ), platelet-activating factor, fibronectin, and serotonin. In addition to achieving hemostasis, the fibrin clot serves as scaffolding for the migration into the wound of inflammatory cells such as polymorphonuclear leukocytes (PMNs, neutrophils) and monocytes.

The phases of wound healing viewed histological :

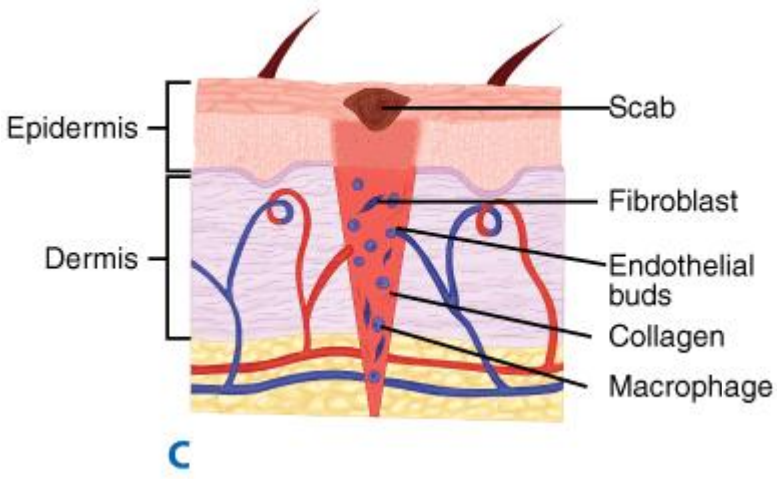
FIGURE 6



A. Hemostatic phase.



B. Inflammatory phase.



C. The proliferative phase

Cellular infiltration after injury follows a characteristic, predetermined sequence. PMNs are the first infiltrating cells to enter the wound site, peaking at 24 to 48 hours. Increased vascular permeability, local prostaglandin release, and the presence of chemotactic substances, such as complement factors, interleukin-1 (IL-1), tumor necrosis factor alpha (TNF-α), TGFβ, platelet factor 4, or bacterial products, all stimulate neutrophil migration.

The postulated primary role of neutrophils is phagocytosis of bacteria and tissue debris. PMNs are also a major source of cytokines early during inflammation, especially TNF-α,[18]which may have a significant influence on subsequent angiogenesis and collagen synthesis (see Fig. 6B). PMNs also release proteases such as collagenases, which participate in matrix and ground substance degradation in the early phase of wound healing. Other than their role in limiting infections, these cells do not appear to play a role in collagen deposition or acquisition of mechanical wound strength. On the contrary, neutrophil factors have been implicated in delaying the epithelial closure of wounds.[19]

The second population of inflammatory cells that invades the wound consists of macrophages, which are recognized as being essential to successful

healing.[20] Derived from circulating monocytes, macrophages achieve significant numbers in the wound by 48 to 96 hours postinjury and remain present until wound healing is complete.

Macrophages, like neutrophils, participate in wound debridement via phagocytosis and contribute to microbial stasis via oxygen radical and nitric oxide synthesis (see 6C). The macrophage's most pivotal function is activation and recruitment of other cells via mediators such as cytokines and growth factors, as well as directly by cell–cell interaction and intercellular adhesion molecules. By releasing such mediators as TGFβ, vascular endothelial growth factor (VEGF), insulin-like growth factor, epithelial growth factor, and lactate, macrophages regulate cell proliferation, matrix synthesis, and angiogenesis.[21,22]Macrophages also play a significant role in regulating angiogenesis and matrix deposition and remodeling. [23]

Hemostasis and Inflammation[24]

Following injury, a wound must stop bleeding in order to heal and for the injured host to survive. It is therefore not surprising that cellular and molecular elements involved in hemostasis also signal tissue repair. Immediately after injury, the coagulation products fibrin, fibrinopeptides, thrombin split products, and complement components attract inflammatory cells into the wound. Platelets activated by thrombin release insulinlike growth factor 1 (IGF-1), transforming growth factor α (TGF-α), transforming growth factor β (TGF-β), and platelet-derived growth factor (PDGF), which attract leukocytes, particularly macrophages, and fibroblasts into the wound. Damaged endothelial cells respond to a signal cascade involving the complement products C5a, tumor necrosis factor α (TNF-α), interleukin-1 (IL-1), and interleukin-8 (IL-8) and express receptors for integrin molecules on the cell membranes of leukocytes. Circulating leukocytes then adhere to the endothelium and migrate into

the wounded tissue. Interleukins and other inflammatory components, such as histamine, serotonin, and bradykinin, cause vessels first to constrict for hemostasis and later to dilate, becoming porous so that blood plasma and leukocytes can migrate into the injured area.

The very early wound inflammatory cells increase metabolic demand. Since the local microvasculature is damaged, a local energy sink results, and PaO_2 falls while CO_2accumulates. Lactate in particular plays a critical role, since its source is mainly aerobic, and its level is tightly regulated by tissue oxygen levels. Oxidative stress is an important signal for tissue repair. These conditions trigger reparative processes and stimulate their propagation.

Macrophages assume a dominant role in the synthesis of wound healing molecules as coagulation-mediated tissue repair signals fall. Importantly, macrophages, stimulated by fibrin, continue to release large quantities of lactate. This process continues even as oxygen levels begin to rise, thereby maintaining the "environment of injury." Lactate alone stimulates angiogenesis and collagen deposition through the sustained production of growth factors. Unless the wound becomes infected, the granulocyte population that dominated the first days diminishes. Macrophages now cover the injured surface. Fibroblasts begin to organize, mixed with buds of new blood vessels. It has been shown that circulating stem cells, such as bone marrow–derived mesenchymal stem cells, contribute fibroblasts to the healing wound, but the extent of this process is as yet unknown.

Fibroplasia and Matrix Synthesis

Fibroplasia

Throughout wound healing, fibroplasia (the replication of fibroblasts) is stimulated by multiple mechanisms, starting with PDGF, IGF-1, and TGF-β released

by platelets and later by the continual release of numerous peptide growth factors from macrophages and even fibroblasts within the wound. Growth factors and cytokines shown to stimulate fibroplasia and wound healing include fibroblast growth factor (FGF), IGF-1, vascular endothelial growth factor (VEGF), IL-1, IL-2, IL-8, PDGF, TGF-α, TGF-β, and TNF-α. Dividing fibroblasts localize near the wound edge, an active tissue repair environment with tissue oxygen tensions of approximately 40 mm Hg in normally healing wounds. In cell culture, this PaO_2 is optimum for fibroblast replication. Smooth muscle cells are also likely progenitors because fibroblasts seem to migrate from the adventitia and media of wound vessels. Lipocytes, pericytes, and other cell sources may exist for terminal differentiation into repair fibroblasts.

Matrix Synthesis

Fibroblasts secrete the collagen and proteoglycans of the connective tissue matrix that hold wound edges together and embed cells of the healing wound matrix. These extracellular molecules assume polymeric forms and become the physical basis of wound strength. Collagen synthesis is not a constitutive property of fibroblasts but must be signaled. The mechanisms that regulate the stimulation and synthesis of collagen are multifactorial and include both growth factors and metabolic inputs such as lactate. The collagen gene promoter has regulatory binding sites to stress corticoids, the TGF-β signaling pathway, and retinoids, which control collagen gene expression. Other growth factors regulate glycosaminoglycans, tissue inhibitors of metalloproteinase (TIMP), and fibronectin synthesis. The accumulation of lactate in the extracellular environment is shown to directly stimulate transcription of collagen genes as well as posttranslational processing of collagen peptides. It is clear that the

redox state and energy stores of repair cells occupying the wound regulate collagen synthesis.

The increase in collagen messenger RNA (mRNA) leads to an increased procollagen peptide. This, however, is not sufficient to increase collagen deposition because procollagen peptide cannot be transported from the cell to the extracellular space until, in a posttranslational step, a proportion of its proline amino acids are hydroxylated. In this reaction, catalyzed by prolyl hydroxylase, an oxygen atom derived from dissolved O_2 is inserted (as a hydroxyl group) into selected collagen prolines in the presence of the cofactors ascorbic acid, iron, and α-ketoglutarate. Thus, accumulation of lactate, or any other process that decreases the nicotinamide adenine dinucleotide (NAD^+) pool, leads to production of collagen mRNAs, increased collagen peptide synthesis, and (provided enough ascorbate and oxygen is present) increased posttranslational modification and secretion of collagen monomers into the extracellular space.

Another enzyme, lysyl hydroxylase, hydroxylates many of the procollagen lysines. A lysyl-to-lysyl covalent link then occurs between collagen molecules, maximizing mature collagen fiber strength. This process, too, requires adequate amounts of ascorbate and oxygen. These oxygenase reactions (and therefore collagen deposition) are rate limited by tissue oxygen level, PaO_2. The rates are half-maximal at about 20 mm Hg and maximal at about 200 mm Hg. Hydroxylation can be "forced" to supernormal rates by tissue hyperoxia. Collagen deposition, wound strength, and angiogenesis rates may be increased and accelerated as tissue PaO_2 is elevated.

Angiogenesis

Angiogenesis is required for wound healing. It is clinically evident about 4 days following injury but begins earlier when new capillaries sprout from preexisting

venules and grow toward the injury in response to chemoattractants released by platelets and macrophages. In primarily closed wounds, budding vessels soon meet and fuse with counterparts migrating from the other side of the wound, establishing blood flow across the wound. In wounds left open, newly forming capillaries connect with adjacent capillaries migrating in the same direction, and granulation tissue forms. Numerous growth factors and cytokines are observed to stimulate angiogenesis, but animal experiments indicate that the dominant angiogenic stimulants in wounds are derived first from platelets in response to coagulation and then from macrophages in response to hypoxia or high lactate, fibrin and its products.

Epithelialization

Epithelial cells respond to several of the same stimuli as fibroblasts and endothelial cells within the mesenchymal area of a wound. A variety of growth factors also regulate epithelial cell replication. TGF-α and keratinocyte growth factor (KGF), for instance, are potent epithelial cell mitogens. TGF-β tends to inhibit epithelial cells from differentiating and thus may potentiate and perpetuate mitogenesis, though it is itself not a mitogen for these cells. During wound healing, mitoses appear in the epithelium a few cells away from the wound edge. The new cells migrate over the cells at the edge and into the unhealed area and anchor to the first unepithelialized matrix position encountered. The PaO_2 on the underside of the cell at the anchor point is usually low. Low PaO_2 stimulates squamous epithelial cells to produce TGF-β, likely suppressing terminal differentiation and again supporting further mitosis. This process of epidermal-mesenchymal communication repeats itself until the wound is closed.

Squamous epithelialization and differentiation proceed maximally when surface wounds are kept moist. It is clear that even short periods of drying impairs the

process, and therefore wounds should not be allowed to desiccate. The exudates from acute, uninfected superficial wounds also contain growth factors and lactate and therefore recapitulate the growth environment found at the base of the wound.

Collagen Fiber Remodeling and Wound Contraction

Remodeling of the wound extracellular matrix is also a well-regulated process. First, fibroblasts replace the provisional fibrin matrix with collagen monomers. Extracellular enzymes, some of which are PaO_2-dependent, quickly polymerize these monomers, initially in a pattern that is more random than in uninjured tissue, predisposing early wound to mechanical failure. Progressively, the very early provisional matrix is replaced with a more mature one by forming larger, better organized, stronger, and more durable collagen fibers. The very early wound provisional matrix usually mechanically fails within the matrix itself (days 0–5). Next, mechanical failure occurs at the matrix-tissue interface or fusion point. The mechanism for connecting the wound matrix to the uninjured tissue border is poorly understood.[24]

FACTORS AFFECTING WOUND HEALING

Local Factors:

Type, size, and location of the wound: A surgical heals faster. Injuries in richly vascularized areas (e.g., the face) heal faster than those in poorly vascularized ones (e.g., the foot). In areas where the skin adheres to bony surfaces, as in injuries over the tibia, wound contraction and adequate apposition of the edges are difficult.

Vascular supply: Wounds with impaired blood supply heal slowly. For example, the healing of leg wounds in patients with varicose veins is prolonged. Ischemia due to pressure produces bedsores and then prevents their healing. Ischemia caused by arterial obstruction, often in the lower extremities of diabetics, also prevents healing.

Infection: Wounds provide a portal of entry for microorganisms. Infection delays or prevents healing, promotes the formation of excessive granulation tissue, and may result in large, deforming scars.

Movement:. Early motion, particularly before tensile strength has been established, subjects a wound to persistent traumas, thereby preventing or retarding healing.

Ionizing radiation: Prior irradiation interfere with blood supply and result in slow wound healing. Acutely, irradiation of a wound blocks cell proliferation, inhibits contraction, and retards the formation of granulation tissue.

Ultraviolet light: Exposure of wounds to ultraviolet light accelerates the rate of healing.

Systemic Factors:

Infections delay wound healing.

Metabolic status: Diabetes mellitus is associated with delayed wound healing because of increased wound infection in diabetics.

Nutrition: Malnutrition impedes wound healing. Methionineand Zinc is needed for proper healing. Vitamin C, required for collagen synthesis and secretion if deficient results in impared wound healing.

Hormones: Corticosteroids impair wound healing by inhibition of collagen synthesis, anti-inflammatory actions and depression of protein synthesis. Thyroid hormones, androgens, estrogens, and growth hormone also influence wound healing.

PATHOGENESIS OF DIABETIC FOOT

Knowledge of the pathophysiologic changes caused by diabetes Mellitus is essential for proper understanding and treatment of foot problems in diabetics. Sustained hyperglycemia, neuropathy, ischemia and infections are the principal pathogenic factors.

Neuropathy:

Diabetic patients develop sensory, motor and autonomic neuropathy. The neuropathic foot is characteristically healthy. It is well nourished, has hair, maintains good dorsalis pedis pulses and posterior tibial pulses, has a high arch. Callus formation is common on pressure points on the soles or toes, and there may be sweating. This indicates lumbar sympathetic activity. On the soles, thick calluses can act as foreign bodies and cause bruising of the subcutaneous tissues with extravasation of blood and serum from the capillaries. This leaves a culture medium pool for local bacteria to

grow and cause an abscess. The condition may be unsuspected because of anaesthesia conferred by the neuropathy and can expand without being detected until the patient develops generalized infection or the process is detected by it's foul odour. At times the overlying callus is so hard that the infection can more easily involve the underlying joint capsule and the metatarsal head than the callus itself. Thus osteomyelitis associated with calluses can be explained and easily understood. The same disease process may occur on the top of hammer toes or in relation to the deformed and collapsed bones and joints associated with Neuro-osteoarthropathy / Charcot's foot.

Vasculopathy:

Both the microvascular and microvascular disease is seen indiabetics. Atherosclerosis is accelerated in diabetics. Infrageniculate vessels (Posterior tibial, Anterior tibial, peroneal vessels) are severely affected. Occlusive disease with microcirculatory impairment of cellular exchange decreases perfusion.

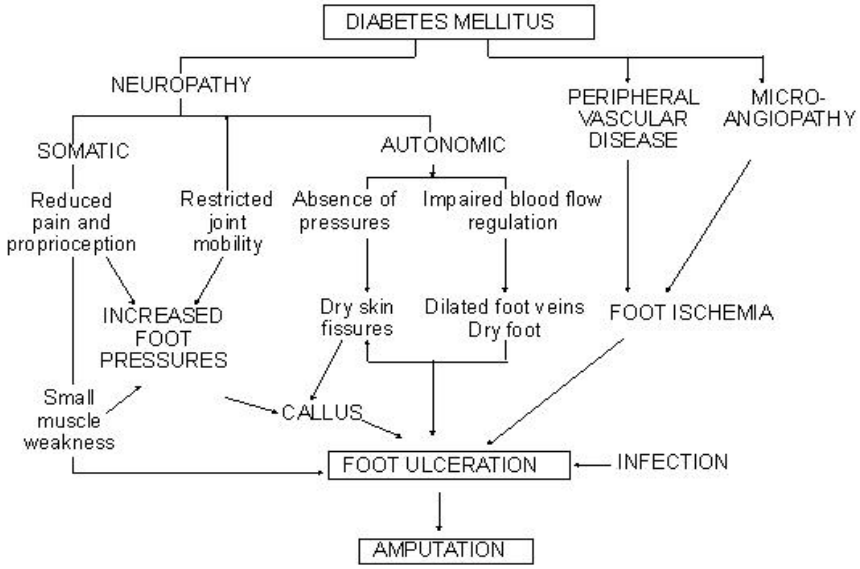


FIGURE 7 : PATHOGENESIS OF DIABETIC FOOT

Ischemic foot, is characteristically dry, atrophic, scaly, hairless, and undernourished. It is cool to touch. The nails are thick and overgrown with dry scales piled up under the nail itself. Such ischemic feet often have a small infection beside or behind a nail or in the depth of a fissure. The ischemic foot may show small flat, dry atrophic ulcers with full thickness skin necrosis in the centre with a rim of erythema surrounding the ulcer as a Crimson Corona from such ulcers or spots of gangrene, there may be a spreading area of cellulitis or a red streak of lymphangitis leading up to the foot or leg to tender lymphnodes in the groin. Wet gangrene of ischemia is seen when a higher vessel, one in the· thigh or calf becomes suddenly occluded. The tissues are still wet and the ensuring gangrene is manifested first by pallor, then rubour, and finally the

unmistakable blistered skin with underlying blue black tissue shining through. Only the ischemic foot has these characteristics, and often the destruction of tissues is so great that even mechanical restoration of blood flow in to the patent major vessels is not sufficient or not in time to revive the injured tissues or to restore them. Therefore higher amputations are necessary.

Diabetic Angiopathy is classified in to 2 types

1)Macro Angiopathy

a)Atherosclerosis-charecterised by formation of atheromatous plaque in the vessel wall predisposing to thrombus formation.

b) Monckeberg's Sclerosis / Medial calcific sclerosis

- Characterized by calcification of the media of mascular arteries

c) Intimal fibrosis - It is a part of normal aging process.

2)Microangiopathy:

Small vessel disease consists of changes in the arterioles and capillaries. Occlusion of these capillaries results in patchy areas of gangrene. The capillaries are a frequent site of diabetic microangiopathy. Homogenous periodic Acid Schiff positive thickening of the capillary wall is considered a hallmark of diabetic microangiopathy because of basement membrane thickening.

Banson and Lacy In their study involving 18 diabetic and 17 non diabetics, found capillary basement membrane thickening is 88% of the diabetic and 25% of non diabetic.

Infections

Infection affects diabetic control, and uncontrolled diabetes causes infection due to hyperglycemia at the wound site that forms a nidus for hyperglycemia, conversely, hyperglycemia may interfere with the healing of an abscess.

Mild infections are usually due to surface organisms like staphylococcus and streptococcus. Severe infections is polymicrobial that includes aerobic gram +ve, anaerobic gram negative rods (E-Coli, Klebsiella, Pseudomonas and Proteus) and anaerobes. Identification by cultures and appropriate antibiotics is necessary for controlling these infections. When the infectious process dissects deep in to the soft tissue and breaches the periosteum, osteomyelitis results. In acute stages of osteomyelitis, the process is suppurative one. There is necrosis of both cortical and cancellous bone with formation of sequestrum. Later on chronic osteomylitis develops.

GUIDELINES IN THE EXAMINATION OF THE DIABETIC LEG AND FOOT

History: It is important to start by checking the patients diabetic history. A long history of insulin dependent diabetes is associated with a higher incidence of neuropathy, retinopathy and nephropathy called the "Diabetic triopathy". Severe triopathy is thought to be secondary to basement membrane thickening and in patients who have this, wound healing appears to be delayed. This is particularly true of those patients with renal failure. Consideration of patients renal function is also important in deciding if they are candidates for arteriography for further evaluation of their arterial insufficiency.Screening and protection programme for patient at risk of ulceration, Patients with an ulcer should be supervised under multi disciplinary foot care services The core specilaist of foot care team consists of :

A. Highly trained specialist podiatrist

B. Highly trained specialist orthotists

C. Nursing care for diabetic foot

D. Diabetologist expertees[25]

Hypertension, tobacco consumption, and Hypercholesterolemia are also factors in the development of arterial insufficiency. Severe cardiac disease may be a contrainduction for aggressive management of diabetic foot problems.

Patient's presenting problem discussed In detail. If patient describes a rapidly enlarging lesion associated with drainage or swelling, perhaps with red lines extending up the leg and fever or shaking chills, then infection would seem very likely.

The classic distal polyneuropathy seen in diabetics may not only cause lesions on its own but may mask the symptoms of infection and arterial insufficiency.

Development of painless ulcer over a pressure point. Patients may complain of pain, burning sensation of the feet, that the feet feels cold or as encased in concrete or of the sensation of walking on glass. Sometimes patient describes knife like shooting pains running up the legs, usually both the legs are involved and this may be helpful in distinguishing neuropathic from ischemic pain.

The history of ischemia is frequently present in patients with diabetic foot lesion, although again these symptoms may be masked by the presence of neuropathy. A history of cardiac or carotid disease would certainly make the presence of peripheral vascular disease more likely. Claudication or pain in muscle groups on exercise is the usual presenting symptoms of peripheral vascular insufficiency.

Occasionally, particularly in smokers, inflow disease is responsible for the ischemia, and then buttock and thigh claudication may be the presenting symptom. This is leriche syndrome due to obstruction of the aortoiliac vessels, may be associated with impotence in men.

Physical Examination:

It is helpful to start the examination away from the area of interest. The carotids should be checked for bruits and the presence of pulses in the arm. Atrial fibrillation as a possible source of embolisation can also be noted. The abdomen should be carefully examined for an abdominal aortic aneurysm, which also may be a source of distal emboli.

Assessment of Diabetic foot :

Includes identification of

A) Neuropathy B) Vasculopathy C) Osteopathy D) Infection

The feet should be examined for

i) Dry skin, fissures, cracks

i i) Ulcers, sinuses, cavity

i i i) Calluses, hyperkeratosis

iv) Infection

v) Foot deformity vi) Gangrene.

A) Neurologic Examination to assess sensory and motor disturbances

i) Monofilament testing

ii) Vibration testing with tuning fork.

iii) Tendon reflexes

iv) Pain

B) Vascularity is assessed by examining the

i) Distal pulses

ii) Ankle brachial pressure index

iii) Transcutaneous oxygen saturation

iv) Duplex arterial study.

C) **Osteomyelitis is assessed by X - ray examination of the foot**

D) Infection is assessed whether it is superficial or deep.

It is common to have all three processes occurring at the same time.

Thus an infected neuropathic lesion in a patient with poor circulation is not unusual. Significantly neuropathy may hide signs and symptoms of ischemia, and the resultant failure to diagnose the underlying ischemia may result in mistreatments. Severe neuropathy may result in a pain free foot that is autosympathectomised and thus, warm, but at the same time, severely ischemic. A minor procedure on such a foot may result in gangrene.

GRADING :

1. **Wagner's Classification**

0 - Intact Skin

1 - Superficial ulcer of skin or subcutaneous tissue

2 - Ulcers extend into tendon, bone, or capsule

3 - Deep ulcer with osteomyelitis, or abscess

4 - Gangrene of toes or forefoot

5 - Midfoot or hindfoot gangrene.

2. **University of Texas Classification**

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

3. PEDIS CLASSIFICATION

Grade	Infection Severity	Clinical Manifestations
1	Uninfected	Wound without purulence or inflammation
2[b]	Mild	≥2: purulence or erythema, pain, tenderness, warmth, or induration; cellulitis ≤2 cm around ulcer; infection limited to skin/subcutaneous tissue; no other complications
3[c]	Moderate	Infection as above plus >1: cellulitis >2 cm, streaking, deep tissue abscess, gangrene and with some life-threatening; involvement of muscle, tendon, joint, or bone
4[d]	Severe	Infection plus systemic toxicity or metabolic instability; fever, chills, tachycardia, hypotension, confusion, vomiting, severe hyperglycemia, acidosis, or azotemia

PEDIS stands for perfusion,extent/size,depth/tissue loss,infection and sensation.

b-medication for treatment is oral.

c- medication for treatment can be oral or parenteral based on clinical situation.

d- medication for treatment is IV ,atleast initially.

RISK FACTORS:[26]

1.Peripheral neuropathy,

2.Peripheral arterial disease,

3.Poor sugar control,

4.Poor footwear,

5.Underlying infection and

6.Duration of diabetes are recognised risk factors for foot.

PREVENIVE MEASURES:[27]

The preventive measures and management of diabetic complications consists of the following:

- Life style modification
- Blood pressure control
- Lipid management
- Glycemic control
- Smoking cessation.

Nail and skin care

- Diabetic patients should examine their feet on a daily bases. The maceration especially between the toes is usually caused by fungal infection and should be observed carefully. It is recommended to use a mirror in order to better observe the plantar surface of the foot. In case, if the diabetic patient's vision is compromised due to retinopathy or the patient is unable to perform the daily examination of own feet, another individual who is fully trained should do the task for the patient.
- The feet should be washed and dry at least once a day. It is important to dry the inter-digital spaces between the toes very carefully.
- The temperature of the water used for rinsing the foot should be less than 37 centigrade. It is recommended to use the elbow or forearm in order to estimate the temperature of water. This method helps to prevent accidental burning of the extremities due to characteristic glove stocking neuropathy in diabetic patients.
- The diabetic patients, especially the ones with sensory neuropathy, should not use the heating pads over their bodies. It is also recommended to warn the patient not to place their feet close to the heaters during the winter.

- All the patients especially those with diabetic neuropathy or high risk diabetic foot should be instructed to use footwear both indoors and outdoors. It is recommended to wear special shoes with adequate size when the patient is walking indoor on the carpet. The use of the shoes without stockings in diabetic patients should be discouraged. In patients with neuropathy, it is also recommended to use the footwear's with enclosed frontal part in order to prevent the minor trauma to the fore foot.
- It is recommended to observe and physically examine inside the patients shoes on a daily basis. This recommendation is given to detect any external objects inside the shoes and to look for pressure effect on different surfaces of the patient's feet. The lateral engorgement of the shoes is an indication of pressure exerted by the first and fifth metatarsals and the swelling observed in the frontal part of the shoes is caused by the pressure of distal phalanges of the first digit.
- Diabetic patients due to autonomic neuropathy present with increased perspiration in the upper thorax and lower extremity, perspiration decreases. As a result, the dryness of the plantar surfaces of the feet and heels is common. The minor trauma combined with the dry skin creates cracks which facilitate the entrance of microorganisms into the skin and consequently foot infection is inevitable. It is recommended to apply lubricants containing urea or salicylates with the ability to penetrate the dry and hyperkeratosis skin. However, the use of lubricants in the inter-digital spaces is discouraged.
- In diabetic patients it is recommended to change and put on clean socks on a daily bases.
- The patients should be discouraged wearing tight sock and the seams should be toward the outside. In diabetic patients it is not advisable to ear stockings up to the knee.

- Especially in female diabetics, any kind of manipulation of the nails is not recommended. The nails should not be cut in a rounded fashion, the straight across cut is recommended.
- The patient should be instructed not to use any kind of chemical substances or commercial pads or plasters in order to treat the calluses of the feet.

DFU is a costly and debilitative disease with severe consequences in diabetic patients. It is important to carefully and completely train the preventive measures as well as foot care to all diabetic patients. The recommendation should be reinforced every time a physical examination is performed. The physician should be certain that the patient has understood the recommendations and is able to care for their feet effectively. It is extremely essential to remind diabetic patients to specifically care for their feet.

Prevention is better than cure

Prevention of ulceration and recurrence once ulceration has occurred are the ultimate goals of any modern team approach to the diabetic foot.

Wagner's Grade 0: Foot are the patients who are potentially "at risk" to develop ulcer or infection due to varying degree of neuropathy and joint deformities. They need regular assessment annually for neuropathy and vascular status. Hence the role of proper footwear and hygiene cannot be overemphasized. The diabetic patient and his family must establish a routine for daily foot and shoe inspection and hygiene.[28] Every patient must be taught to shake his shoes at and inspect them before wearing. Proper hygiene must become a religion. Washing the feet everyday with mild soap and rinsing and drying thoroughly especially between the toes are advised.

The physician or health care provider must always set the example. Controlling blood glucose, weight, and blood pressure; eliminating smoking; encouraging daily exercises are important. Periodical neurological and vascular examinations are important. Early recognition and prompt reporting of a problem are encouraged.

Classification and Staging[29,30,31,,32]

After completing the basic assessment, it will now be possible to classify the diabetic foot. For practical purposes, the diabetic foot can be divided into two distinct entities:

1. The neuropathic foot.

2. The neuroischaemic foot.

Neuropathy is nearly always found in association with ischaemia, so the ischaemic foot is best called the neuroischaemic foot. In rare cases the foot may clinically be ischaemic without signs of neuropathy, but in practice, the diabetic ischaemic foot is treated in the same way as the neuroischaemic foot, and thus, we have continued with the two main divisions. It is essential to classify the diabetic foot by differentiating between the neuropathic and the neuroischaemic foot as their management will differ in many respects. Usually there will be no doubt as to which category the foot should be placed in. However, if the examiner has any doubt as to the correct classification, then the foot should be regarded as neuroischaemic, because if a neuroischaemic foot is wrongly classified as neuropathic, with resulting failure to do further tests to confirm ischaemia and adapt the care plan accordingly, this may lead to preventable catastrophe and loss of the foot.

Neuropathic foot:

The neuropathic foot is a warm, well-perfused foot with bounding pulses and distended dorsal veins due to arteriovenous shunting. Sweating is diminished so skin and any callus tend to be hard and dry and prone to fissuring Toes are flexed and the arch of the foot may be raised Ulceration commonly develops on the sole of the foot, associated with neglected callus and high plantar pressures.

Despite the good circulation, necrosis can develop secondary to severe infection. The neuropathic foot is also prone to bone and joint problems which we refer to as Charcot's osteoarthropathy.

Neuroischaemic foot:

The neuroischaemic foot is a cool, pulseless foot with poor perfusion and almost invariably also has neuropathy. The colour of the severely ischaemic foot can be a deceptively healthy pink or red caused by dilatation ofcapillaries in an attempt to improve perfusion. The neuroischaemic foot may be complicated by swelling, often secondary to cardiac failure or renal impairment. Ischaemic ulcers are commonly seen around the edges of the foot, including the apices of the toes and the back of the heel, and are associated with trauma or wearing unsuitable shoes. The neuroischaemic foot develops necrosis in the presence of infection or if tissue perfusion is critically diminished.

Even if neuropathy is present and plantar pressures are high, plantar ulceration is rare. This is probably because the foot does not develop heavy callus, which requires good blood flow.

After classification of the diabetic foot, it is necessary to make the appropriate staging in its natural history. The natural history of the diabetic foot can be divided into six stages:

Stage 1: Normal foot.

Stage 2: High-risk foot.

Stage 3: Ulcerated foot.

Stage 4: Infected foot.

Stage 5: Necrotic foot.

Stage 6: Unsalvageable foot.

This simple staging system covers the entire spectrum of diabetic foot disease but it emphasizes the development of the foot ulcer in stage 3 as a pivotal event demanding urgent and aggressive management. However, each stage demands specific treatment.

Stage 1: At this stage, the patient does not have the risk factors of neuropathy, ischaemia, deformity, callus and swelling rendering him vulnerable to foot ulcers. The normal foot is characterized by no symptoms, including no pain and examination is normal.

Stage 2: The patient has developed one or more of the risk factors for foot ulceration including neuropathy, ischaemia, deformity, callus and swelling. These risk factors need addressing to reduce susceptibility to ulceration. Patients without active foot ulceration but a history of ulceration should be regarded as very high risk. Within stage 2 there are specific conditions which are nonulcerative but require treatment.

These include:

Severe chronic ischaemia

Acute ischemia.

There are also specific complications of neuropathy:

Neuropathic fractures.

Charcot's osteoarthropathy

Painful neuropathy.

Stage 3: The foot has a skin breakdown. Although this is usually an ulcer, it is important not to underestimate some apparently minor injuries such as blisters, skin fissures or grazes, all of which have a propensity to become ulcers if they are not treated correctly and fail to heal quickly. Ulceration is usually on the plantar surface in the neuropathic foot and on the margin in the neuro-ischemic foot.

Stage 4: The foot has developed infection with the presence of purulent discharge or cellulitis which can complicate both the neuropathic foot and the neuro-ischemic foot.

Stage 5: Necrosis has supervened. In the neuropathic foot, infection is usually the cause. In the neuro-ischaemic foot, infection is still the most common reason for tissue destruction although ischaemia contributes. In some cases ischaemia alone can lead to necrosis of a previously intact foot, with slow onset of dry necrosis and necrotic toes which appear shriveled.

Stage 6: The foot cannot be saved and will need a major amputation. Reasons for major amputation:

Extensive necrosis which has destroyed the foot. Severe infection which puts the life at risk Agonizing ischaemic pain which cannot be relieved. The simple staging based on natural history system used to make an initial assessment of the diabetic foot at whatever stage in the natural history it might be. The stage sets the place in the natural history and also determines treatment. The aim is to keep all diabetic feet at as lower stage as possible.

Other classifications of the diabetic foot such as the Wagner system, the University of Texas system and the Nottingham S (AD) SAD system are essentially classifications of ulcers and do not cover the whole natural history of the diabetic foot.

THE DIABETIC FOOT: MEDICAL AND SURGICAL MANAGEMENT

A Baseline Approach in Managing the Acute Problem of the Diabetic Foot:

1. Appraise problem

a) Careful inspection with emphasis on webspaces and back of heels.

b) Record pulses, venous filling time, rubor

c) Record sensation.

2. Describe lesion

3. Describe Necrotic tissue, probe Sinuses with sterile probe to determine the extent of disease

4. Culture pus for aerobic and anaerobic organisms

5. Begin broad spectrum antibiotic until appropriate antibiotics can be given according to culture and sensitivity.

6. Medical Management of Diabetes - Blood sugar monitoring and antidiabetic measures to achieve good glycemic control, Doppler study of vessels.

7. X - ray both feet to exclude osteomyelitis.

8. No weight bearing [33]

a. Hospitalize with absolute bed rest when indicated

b. Crutches or walker when feasible.

9. Surgical Management of the Problem.

a. No soaks

b. Antibiotics

c. Medical Management of diabetes

d. Dressing change atleast once daily.

e. Surgical debridement, frequently if necessary.

f. Consideration for possible arterial reconstruction

g. Drainage or open amputation.

10. Rehabilitation

a. Podiatrist for patient education, preventive maintainance, orthotics, healing sandals and special shoes.

b. Nutritionist to advice on diet needs.

c. Surgeon to ensure proper wound healing and proper prosthetics

d. Physician to make final decision about diabetes management.

e. Psychiatrist to return to normal activity.

Multidisciplinary Management:

The aim in managing diabetic foot problems is always to keep the patient at as low a stage as possible. At each stage of the diabetic foot it is necessary to take control of the foot to prevent further progression.

Primary Treatment of Foot Ulcers: When the examination reveals a foot at stage 3, 4 or 5 there is a need for a great sense of urgency: treatment should begin without delay. No one person can take control of the diabetic foot. Successful management needs the expertise of a multidisciplinary team including the following: Podiatrist, Physician, Nurse Orthotist, Radiologist, Surgeon.

Strategies for saving the diabetic foot:

- Correction of vascular risk factors.
- Improve the circulation.
- Regular foot Inspections.
- Treatment of foot ulcers.
- Treatment among medical disciplines.
- Prescribing special shoes.
- Patient Education.

Improving Circulation: A regular walking program will improve waiking distance in 80% of the patients. Recently a drug Cilostozole is used. The ultimate treatment for claudication is vascular surgery is to by-pass narrowed vessels or endarterectomy. Transcutaneous angioplasty is also useful. The surgical approach to aorto-iliac occlusive disease is aorto-ilac endarterectomy, bypass grafting and extra-anatomic reconstructions.

In the patients with simple occlusion of the superficial femoral artery, autogenous saphenous vein by pass from the common femoral artery to the popliteal artery is done. For patients with significant disease in the above knee popliteal artery, a bypass to the distal popliteal artery is done.

Patient Education: The patient should be taught about good diabetic control, foot-care, dangers of smoking etc. By this the patient can prevent injury to insensitive foot and detect foot lesions earlier. Maintenance of records of investigations.

Local Treatment of Diabetic Foot

Uncontrolled diabetes affects infection and infection adversely affects diabetes.

1) Debridement and Drainage:

Drainage means opening all abscesses, probing carefully, and laying open all sinus tracts, debriding all necrotic tissue and providing unhindered dependent drainage of pus in the resting foot. The pus must drain down and out. Gas in the tissues can often be felt as crepitus or may be the first detected on x-ray film. This is a serious finding and must be treated immediately by open drainage of all infected spaces and prompt i.v. antibiotics.

Drainage of an infected area may involve amputation of a necrotic toe or toes or even an open amputation. Such amputations are drainage procedures primarily. The

avascular joints tolerate infection badly, and ultimately the infected joints in the toes and the feet have to be removed. When an infected area has been enclosed, it is important to plan and attempt to salvage tissue for a possible definitive wound closure.

Most foot infections do not require extensive incisions and debridement, yet the principles must always be remembered.

Types of debridements:

1. Surgical Debridement : The removal of nonviable, contaminated and infected tissue using surgical technique under anaesthesia.

Advantages :

• Fast and selective

• Used in wounds with large amount of necrotic or infected tissue.

• Performed by skilled medical personal.

• Used to convert chronic wound to acute wound environment

Disadvantage :

• Require sterilized operating room and coagulating equipment.

2. Conservative Sharp Wound Debridement :

Involves removal of only clearly visible devitalized tissue periwound callus. Requires surgical instruments such as Forceps, Scissors, and Blade/Scalpel. Can be performed at bedside setting. It is Faster and more selective than chemical/enzymatic debridement, and usually painless.

3. ENZYMATIC DEBRIDEMENT

Specific enzymes are applied to the wound to remove/digest necrotic tissue and dissolve the devitalized tissue. Most common types are: Collagenase (Santyl), and Pepain-Urea.

Enzymatic Advantages: it can be used in patients

• Receiving anticoagulants

• Who has contraindication for surgery.

• Outpatient dressing.

Disadvantages:

• Can be used with metals (flamazine, silver, zinc, others) or with topical antiseptics (Dakins's solution).

• Expensive.

4.Mechanical debridement

Use of moist saline gauze drying out, adhering to wound bed and removing devitalized tissue. Hydrotherapy.

Advantage: Good for larger wounds with a significant amount of devitalized tissue.

Disadvantages: Painful and Non-selective (removes both healthy & necrotic tissue)

Indication: Used when the local bacterial burden is more of a concern than stimulation of healing in a Non-healing wounds.

5. Autolytic: Accomplished with the use of dressings such as hydrocolloids and hydrogels to support the ability to debride. Hydrocolloid- acts to retain body's own moisture in wound.

Hydrogel-donate water into necrotic tissue to liquefy it.

Disadvantages:

- Cannot be used Occlusive dressing not indicated in infected wounds or over an eschar in pulse less extremity.
- Less aggressive and slower.
- Hydrocolloid fluid odour often mistaken for (infected) wound.

6. Chemical debridement:

Specific chemicals are used to remove necrotic tissue.

- Acetic acid (0.5%-5%) Low pH, effective against Pseudomonas species.
- Chlorhexidine: Active against gram-positive and gram-negative, organisms.
- Hydrogen peroxide: Desloughing agent with some bactericidal effect. May damage healthy granulation tissue and air emboli if packed in deep sinuses.
- Sodium hypochlorite solution-High pH causes irritation to skin. Dakins and Eusol are also used.
- Silver Nitrate Sticks—Remove mucosal or hypergranulation tissue. May be used for hemostasis.

2) Dressings

Dressings are used to serve the following purposes.

1. Contain wound drainage.

2. Debride a wound.

3. Protect an area from trauma.

4. Protect an area from contamination.

5. Promote proper wound healing.

The basic equipment necessary for bedside foot care is :

1. Sterile debridement set containing

a. Sharp scissors for debriding.

b. Blunt ended needle wound probe.

c. Smooth forceps.

2. Sterile toenail clippers.

3. Sterile gauze dressings.

4. Tube guage, paper tape, culture tubes.

5. Topical Agents

- Povidone iodine 2.5% -Bactericidal.

It is effective against a variety of pathogens including *Staphylococcus aureus*. The solution dries and tends to discolor skin. It may also cause local irritation to the periwound skin. - Dakin's solution (chlorazene 0.25%). antiseptic solution containing sodium hypochlorite and developed to treat infected wounds. Dakin's solution is prepared by passing chlorine into a solution of sodium hydroxide or sodium carbonate. The solution is unstable and cannot be stored more than a few days. The Carrel–Dakin treatment consists of the periodic flooding of an entire wound surface with the solution.

- Bactracin ointment – antibacterial.

- Vasceline guage.- is a fine mesh, absorbent gauze impregnated with white petrolatum. This product remains moist, nontoxic and nonirritating. It is non-adherent and sterile. - Normal saline.

Dakin's solution:

It is a chlorine releasing agent that is both bactericidal and active in loosening necrotic tissue to aid in local debridement. Dakin's also helps to control fetid odours from severely infected wounds.

Open wounds require packing using an unfilled guage moistened with atherapeutic solution. Changing packing two or three times a day is recommended for debridement of a necrotic wound. Allowing sufficient time between dressing changes gives the packing time to begin to dry and therefore provide gentle debridement as the packing is removed from the wound. Unfilled guaze is recommended for packing

wounds. Care must be taken not to pack the wound too tightly as it tightly obstructs drainage.

A properly applied dressing will not constrict the foot or leg and slip, possibly causing wound trauma or exposure. Spiraling or wrapping the roller guage in a figure of eight fashion is the best way to prevent a tourniquet effect and will decrease the risk of compromising the circulation of the foot.

Classification of Dressings

Wound dressings have evolved over the years on the principles of providing protection to wound raw surface, absorbing exudates, controlling infection and promoting granulation tissue formation and creating ideal environment for healing. Dressings can be classified into two major categories according to usage as follows:

1. **Short term application dressings:** These dressings require replacement at regular intervals.

2. **Long term applications or skin substitutes:** They can be further subdivided into:

Temporary - Applied on fresh 'partial thickness wounds' until complete healing is ensured.

Semi- Permanent - Applied on 'full thickness wounds' until autografting. Based on the type of material used for the preparation of dressing they may be classified as conventional, synthetic and biological dressings. Within each category, the dressings may be further classified into:

1. Primary Dressing - A dressing in physical contact with the wound bed.

2. Secondary Dressing - A dressing that covers the primary dressing.

3. Island Dressing - A dressing that is constructed with a central absorbent portion surrounded by an adhesive portion.

A. Conventional Dressings

These dressing materials are made up of fabric material such as gauze, but allow evaporation of moisture resulting in a dry desiccated wound bed and allow entry of exogenous bacteria into the wound. This led to the origin of compound dressings such as Tulle grass which is wide mesh gauze impregnated with medical grade paraffin. This results in a relatively non-adherent dressing. Further developments in 1980 involved incorporation of antibacterial agents such as carbolic acid and mercuric chloride, penicillin and polymyxin creams in combination with absorbent dressings.

Recent innovation uses silicone polymer in place of paraffin. These dressings are less adherent to the wounds making dressing changes less traumatic. The concept of moist wound dressings gained importance during the mid 1980's. Based on a study by Atiyeh BS, EI-Musa KA, Dham R, full and partial thickness cutaneous wounds, when exposed to wet and moist environment showed improved healing. It has been found that moist environment prevents dessication of denuded dermis or deeper tissues and allows faster and unimpeded migration of keratinocytes over the wound surface and also facilitates the cytokines to exert their effects on wound contracture and re epithelization.[34]

Since the conventional dressings had limitations for application on full thickness wounds, research into the development of more advanced wound dressings for the treatment of wounds has resulted in the development of synthetic and biological dressings.

B. Synthetic Dressings :

These dressings can be classified into

1. Films - They are homogeneous dressings composed of a polymer sheet coated on one side with an adhesive. The most commonly used Polymers include polyurethane, polyethylene, Polycaprolactone, poly tetrafluoroethylene, dimethyl aminoethyl methacrylate. Film dressings are well suited for superficial wounds, but lack of absorbing capacity and impermeablity to water vapour and gases cause accumulation of wound fluid beneath the dressing and hence leakage of exudate and entry of exogenous bacteria to the wound surface. Therefore, they are not convenient for larger wounds.

2. **Foams and sprays** - Foam dressings are sheets of foamed solutions of polymers such as polyvinyl-alcohol and polyurethane which are superior to film dressings in that they provide thermal insulation and help to maintain a moist environment at the surface of the wound. Furthermore, they are gas permeable, non-adherent, light and comfortable. Examples are silastic foam and lyofoam, which have the advantage of being formable insitu to treat irregular cavity wounds. However, these dressings are difficult to use in certain anatomical areas. Spray dressings are more comfortable to the wounds surface and they are totally portable. Most sprays are copolymers e.g., Aeroplast is a copolymer of hydroxy vinyl chloride acetate modified maleic resin ester. Further studies have resulted in the development of dressings composed of spray and foam combinations e.g., gelatin based spray able foam.

3. Composite dressings - These are composed of laminates of two or more layers. The outer layer is designed for durability and elasticity and may serve as a rate controller for water evaporation, while the inner layer is designed for maximum adherence and elasticity. Composite dressings may be classified as follows:

A.Hydrocolloid dressings- These are composed of laminates of two or more formulations containing a cocktail of elastomeric adhesive and gelling agents.

Carboxy methyl cellulose is the most common absorptive ingredient acting as absorbent for wound fluid.

1. Granuflex - This material consists of an outer protective layer of polyurethane foam and an inner layer consisting hydrocolloid / polymer complex.

2. Epigard - This is a composite of an layer of reticulated polyurethane laminated to an outer sheet of microporous poly tetra fluoroethylene (PTFE) Adherence, availability, sterility, long shelf life and low cost are its major advantages.

3. Biobrane - This is a composite of an ultra thin porous membrane of polydimethyl siloxane bonded to an inner nylon mesh. Roberts et al and Stein successfully used ilese for both superficial and deep donor sites. Roberts[35] and Stenn[36] successfully used these for both superficial and deep donor sites.

b. Hydrogel sheets - These are sheets of 3-D networks of cross linked hydrophilic polymers. They interact with aqueous solutions. The most commonly used polymers are polyethylene oxide, polyacrylamide and polyvinylpyrrolidine. Owing to their unique cooling ability, they may be of great benefit to be used as a first aid measure for thermal burns. An example is Vigilon which is reinforced polyethylene oxide hydrogel sandwiched between 2 polyethylene films. They are however slippery to use and difficult to keep in place in a high sheer stress.

c. Hydrogel Amorphous - These are similar in composition to sheet hydrogels except that the polymer has not been cross linked to form a sheet. They contain small quantities of collagen, alginate or complex carbohydrates. They are unique in their ability to donate moisture to a dry wound eschar and facilitate autolytic debridement in wounds. But owing to the viscosity of the amorphous hydrogel, it may be difficult to retain it in the wound bed. However, hydrogel dressings exhibit more rapid rate of closure and reepithelialization as compared with the hydrocolloid wound dressing.

d. Gels - Several types of gel based dressings have been developed. For example Wichterlie and Lim[37] produced hydroxyethyl methacrylate (HEMA) based hydrogel which was biocompatible and no toxic. Subsequently Nathan[38] developed PHEMA (Hydran) polyhydroxyethyl methacrylate based hydrogel which is directly formed on the wound surface. Later, in 1977 a new type of wound covering 'Geliperm' was developed by Wokalek[39] which

is formed by polymerization of agarose and acrylamide. Further modification resulted in the development of a cross linked polyethylene oxide hydrogel.

e. Super Absorbents - This dressing has an island configuration consisting of an extra thin hydrocolloid as the adhesive portion with a central area of non woven absorbent covering the superabsorbent particles encased inside, e.g., Combiderm, Conva Tec., etc.

All the above dressings act only, as temporary dressings and do not help in massive burn injuries with very limited skin donor sites and are usually combined with alternative wound closure techniques.

c. Biological Dressings

These are derived from natural tissues usually consisting of various formulations and combinations of collagen, elastin and lipid. They are far superior to synthetic dressings in that they:

1. Restore a waiver vapour barrier and prevent dehydration of the wound
2. Decrease evaporational heat loss
3. Decrease protein and electrolyte losses in wound exudate
4. Prevent bacterial contamination of the wound and hence protect the wound and patient from sepsis
5. Permit less painful dressing changes

6. Permit painless movement over joints

7. Facilitate debridement of wounds

8. Create good granulation tissue bed for auto grafting of deep wounds

9. Can be used to test for successful subsequent autograft

10. Decrease healing time of partial thickness burns and donor sites and

11. Improve quality of healing, inhibit excessive fibroblasts and decrease contraction.

Biological dressings range from allograft, heterografts from pigs, dogs and other species, to embryonic membranes, embryofoetus and neonatal skins; films of reconstituted collagen from bovine and other sources, fibrin, cultured epidermal grafts, dermal matrix grafts and cultured dermal matrix composite grafts.

Routine Foot Dressings

1. Moisten guaze with appropriate solution and pack the wound gently.

2. Fashion a heel cup from cut, folded and taped abdominal pad.

3. Fluff two 4 inch guaze sponges over toes

4. Secure the primary dressing, including heel cup by using a spiral roller guaze by wrapping in a figure of eight fashion.

5. Apply paper tape to secure the roller guaze.

Growth Factors and Bioengineering

The rapidly aging population and patients with multiple concomitant pathologies present an increasing population of patients with non-healing and problem wounds. Many of these patients are not surgical candidates, or surgical procedures have failed to close their wounds. These wounds are particularly worrisome when an orthopaedic component is included, since bone and hardware must be covered as quickly as possible to prevent infection and even worse complications.

1. Growth Factors

Greater understanding of the healing process at the cellular level has resulted in the use of growth factors like becaplermin, recombinant platelet-derived growth factor, are produced through recombinant DNA technology. According to a study by Steed et al[40] debridement enhances the effectiveness of becapiermin in healing chronic neuropathic ulcers.

2. Human Skin Equivalents

Modern human skin replacement dates back to the 1960s, when advances in tissue culture technologies led to the cultivation of human epidermal cells. These were obtained via biopsy of the patient's tissue and subjected to trypsin to separate the dermis from the epidermis.

The keratinocytes were then grown in vitro to produce sheets of autologous epidermal tissue. These sheets were fragile, delicate to handle, and provided only 50 percent to 60 percent permanent take. New tissue required two to three weeks growth time, and lacked a dermal component, vital in skin grafting.

More dermis grafted means less wound contracture and scarring, more tensile strength and better cosmetic results. Refinements in the development of a matrix led to the development of Dermagraft, a living, metabolically active, immunologically inert dermal tissue.

Dermagraft contains normal dermal matrix proteins and cytokines, and is composed of cultural neonatal fibroblasts grown on a polyglycolic acid bioabsorbable mesh. As the tissue grows it produces extracellular proteins and closely resembles human skin.

In studies by Gentzkow et al[28] patients were enrolled with full-thickness diabetic ulcers that had adequate perfusion. Pooled data showed that 51 percent of those who received a weekly application of Dermagraft for 12 weeks achieved complete healing vs. 31.7 percent in the control group.

Apligraf, another living tissue equivalent, was approved by the Food and Drug Administration in 1998 for venous leg ulcers. Apligraf consists of bovine Collagen matrix containing fibroblasts and connected to a layer of stratified epithelium. The result is a sheet of tissue with both dermal and epidermal layers, metabolically and biochemically comparable to human skin. The dermoepidermal junction is flatter, however, and there are no melanocytes, Langerhans cells, lymphocytes or hair follicles present.

In a study by Falanga et al[40], 293 patients with non-healing venous ulcers received either compression therapy or Apligraf. At six months, 63 percent of the patients receiving Apligraf healed vs. 49 percent in the control group and did so more quickly than the control group - 61 vs. 181 days to closure.

3. Miscellaneous Topical Agent

Collagen: Collagen is critical in the proliferative phase of wound healing. Exogenous sources of collagen primarily purified bovine extracts, are available as gels, particles, and in an alginate dressing. Exogenous collagen provides additional protein for tissue repair. As a foreign agent it might also revert the chronic wound to an inflammatory phase, "jump-starting" the healing process.

Donaghue et al[41] evaluated the alginate dressing (Fibracol, Johnson and Johnson, Arlington, Texas) in the treatment of diabetic foot ulcers. Seventy-five

patients were randomly assigned to either a collagen-alginate dressing or gauze dressing group. At the end of the study, the mean reduction in wound size was 80.6 percent for the collagenlginate group and 61.1 percent for the gauze group. Complete healing was achieved in 48 percent of the collagen-alginate group and 36 percent in the gauze group.

Hyaluronic Acid: Hyaluronic acid is involved in the structure and organization of the extracellular matrix and is associated with increased mitotic activity. It is a high molecular weight polysaccharide synthesized in the plasma membrane of fibroblasts and other cells. The ability of injured fetal tissues, which are high in Hyaluronic acid, to heal without scarring has prompted extensive research.

Beta Glucan: It is a major cell-wall carbohydrate extracted from such grains as oats and barley. The biological activity of beta glucan results from its ability to bind macrophage beta-glucan receptors and promote macrophage stimulation.

Beta glucan products enhance the activities of not only macrophages but also neutrophils, natural killer cells, T cells and B cells. Beta glucan is thought to increase macrophage infiltration, speeding the onset of fibroplasia and fibro genesis, stimulation of increased tissue granulation, and enhanced reepithelialization. Beta glucan is available as either BCG matrix or Glucan II. Both are available in multifilament mesh dressings; BCG matrix is also impregnated with collagen.

.**Silver Arglaes:** Silver compounds are powerful antimicrobials, useful in promoting healing. Arglaes is an inorganic phosphate similar to other compounds such as silver nitrate, silver oxide and silver chloride. It consists of fused sodium and calcium phosphates with small amounts of silver in the presence of water, these materials release free silver ions.

4.Anabolic Steroids

Oxandrolone : Oxandrolone is an anabolic steroid with a high anabolic and low androgenic ratio, and has anticatabolic, protein-sparing properties. Exogenous anabolic agents clubbed with nutritional intervention can result in a threefold to fourfold higher rate of protein synthesis than with nutritional interventions alone. Demling and De Santi[42] studied eight patients with non-healing wounds and a 10 percent or greater loss of body weight. Nutrition was optimized over four weeks, without significant effect on weight gain or healing. Adding oxandrolone resulted in gains of approximately 4 pounds per week across 12 weeks. During this time, five wounds closed completely and three others were 75 percent closed.

5. Adjuvant Modalities

1.Topical Hyperbaric Oxygen Therapy:

The therapy is based on achieving an atmospheric pressure of 1.02 to 1.03 atm, which is thought to stimulate fibroblast, growth, collagen formation and neoangiogenesis. This provides a lethal environment for anaerobes, often a normal part of the diabetic foot's flora. Topical hyperbaric oxygen is administered using a sealed polyethylene bag over the affected area and administering 100 percent oxygen to a pressure between 20 and 30 mmHg. Treatments last 2 to 2 ½ hours. In a study by Landau[43], 50 patients with diabetic ulcers were treated with topical hyperbaric therapy, alone or with a low-energy laser. On average, 25 treatments were performed over three months. Forty-three of the 50 patients experienced resolution of their ulcers.

2.Radiant Heat Bandage:

Heat therapy has long been employed, especially for musculoskeletal conditions, but it has not been widely used as a wound healing modality. Heat increases local blood flow, subcutaneous oxygen tenson which improve healing mechanisms. In clinical studies by Santilli and Robinson [44] on patients with venous leg ulcers, those who used radiant heat bandage devices reported significant decreases in both wound size and pain across two weeks with no adverse effects.

3.Ultrasonic Stimulation

Therapeutic ultrasound equipment The equipment used to produce therapeutic levels of megahertz ultrasound typically consists of a microcomputer-controlled high frequency generator linked by a coaxial cable to an applicator or treatment head. The treatment head contains a disc of a piezoelectric material, such as lead zirconate titanate (PZT), which acts as a transducer changing one form of energy into another,

in this case into ultrasound. When an alternating voltage is applied across such a disc, it expands and contracts at the same frequency as the oscillation, transducing electrical energy into ultrasound. A similar system is made use of for kilohertz ultrasound but the frequency of vibration is much lower and the transducers have a different composition and mode of operation.There is evidence that both megahertz and kilo- hertz ultrasound can stimulate the healing ol chronic wounds.

4.LASER (LOW INTENSITY LASER THERAPY)

Laser is an acronym for 'light amplification by the stimulated emission of radiation'. Low Intesity Laser Therapy (LILT) involves use of medical lasers that operate at intensities too low to damage living tissues. Their action is photobiomodulation : they can stimulate inactivated tissue components.

Equipments:

It has 3 essential components:

- Lasing medium, which is capable of being energized sufficiently for lasting to occur.
- Resonating activity containing the lasing medium.
- Power source that transmits energy into the lasing medium.

5. Electrical stimulation [45]

Electric current has been shown to facilitate wound healing in animal models and improves blood flow to the foot in vascular studies in diabetes patients. Peters et al (2001) in diabetic foot, the treatment comprised a dose of 50v with 80 twin peak monophonic pulses per second delivered for 10 minutes followed by 10 minutes of 8 pulses per second of current. All patients also received traditional wound care of debridement, collagen wound gel, and pressure reduction at the site of ulceration.

6.VACUUM-ASSISTED CLOSURE THERAPY [46]

Also known as negative pressure therapy and sub- atmospheric therapy, vacuum-assisted closure (VAC) therapy is a non-invasive technique entailing exposure of a wound to pressure of less than one atmosphere (Morykwas & Argenta 1997. Clare et al 2002. Egington et al 2003). The effects of this include

1. Dilation of the arterioles improving the blood supply to the wound
2. Removal of excess fluid thus reducing ocdema
3. Reduction in bacterial colonization of a wound by drawing off many of the bacteria with this fluid
4. Improved granulation tissue formation, resulting in progressive wound closure.

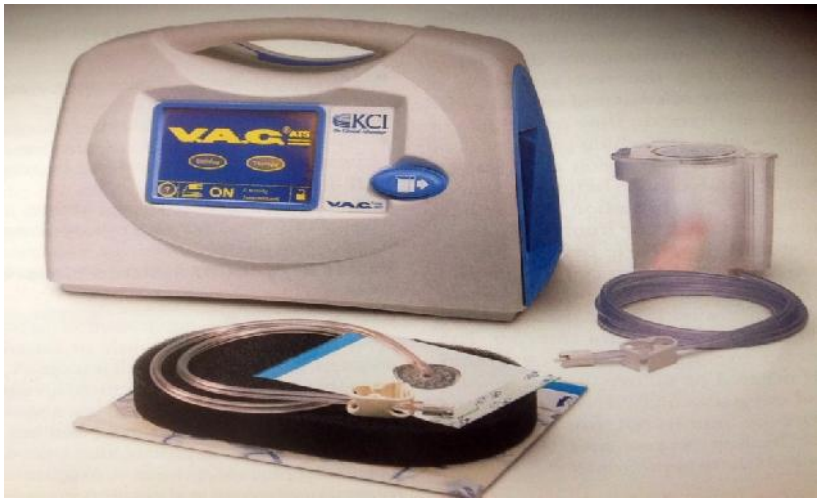


FIGURE 8 : VAC Device with equipments

What is vacuum-assisted closure therapy?

VAC therapy is the application of subatmospheric either continuously or intermittently, to an open wound such as a leg ulcer. A VAC device to do this has been manufactured for clinical use by Kinetie Concepts, Inc. It delivers negative pressure (vacuum) uniformly to the wound bed and to the tissue adjacent to it. Case studies have documented its effectiveness Morykwas & Argenta 1997) in the treatment of acute and chronic wounds.

Vacuum-assisted closure equipment

The VAC negative-pressure equipment consists of:

1. VAC negative-pressure unt
2. VAC PAC dressing pack. containing sterile foam dressing. suction tubing. occlusive transparent drapes.
3. Canister to collect the exudate and tubing for connection between the VAC unit and the foam dressing.

Application of vacuum-assisted closure to a wound

The foam dressing is cut to the shape of the wound and applied to the wound, which has been irrigated with normal saline.

The foam dressing and at least 3.5 cm of surrounding intact skin is covered with the occlusive transparent drapes to ensure an occlusive seal and convert the open wound into a con- trolled closed wound; the drape is also sealed to the tube leaving the dressing The free end of the tubing from the foam dressing is connected to the tubing on the unit.

The unit is deliver the type of negative set to pressure required, continuous or intermittent. and the device is switched on

.**Treatment parameters (supplied by Kinetic Concepts, Inc.)**

These vary according to the type of wound being treated. The following example is for venous stasis, arterial insufficiency and diabetic ulcers:

Cycle: continuous for 5 days

Duration: ideally 24 hours a day. The dressing should be removed if therapy has to be discontinued for more than 2 hours a day

Target pressure: 50-75 mmHg Dressing change: every 48 hours or every 12 hours if the wound is infected.

Bioeffects of vacuum-assisted closure therapy

The continuous subatmospheric pressure used at the commencement of treatment draws fluid from the wound bed and surrounding tissue. decreasing local interstitial pressure and allowing vessels previously compressed or collapsed to dilate, restoring blood flow.

The intermittent negative pressure applied later assists the proliferative phase of repair when granulation tissue forms.

Mechanism

The use of negative pressure therapy to remove tissue fluid from the wound bed and its oedematous surroundings is based on the supposition that the removal of this fluid will enhance the healing process.

Certainly reduction in oedema and therefore of pressure on the microcirculatory system is advantageous in that the reduced pressure allows the vessels to dilate, with the result that perfusion of the wound is improved.

Also the fluid from chronic wounds contains factors that, when applied to cells in vitro, suppress cell division and protein synthesis.

However later in the healing the fluid process contains beneficial growth factors secreted by macrophages and other cells,factors that can enhance the development of granulation tissue.

The analysis of fluid withdrawn from chronic and hea;ing of wounds has demonstrated this change (Moryakwas & Argenta 1997). It should therefore, only be used until this change occurs.

Another mechanism of action of the VAC is the mechanical stimulation of cells by tensile forces placed on the surrounding tissue when the applied vauum collapses the foam dressing.This results in deformation of the cells anchored in the tissues. Integrins act as transmembrane bridges between the applied extracellular forces and the cytoskeleton. Perturbation of the integrin bridges distorts the cell membrane and results in the release of second messengers that trigger changes in gene expression with subsequent increases in cell proliferation and protein synthesis.

Effect of vacuum-assisted closure therapy on acute wounds

The treatment parameters recommended by Kinetic Concepts are as follows:

Cycle: continuous for the first 48 hours to evacuate excess fluid from the wound; intermittent thereafter (standard: 5 min on. 2 min off) to promote granulation tissue formation

Duration: ideally 24 hours a day

Target pressure: 125 mmHg ressing change: every 48 h or every 12 hours if the wound is infected.

This produces a rapid reduction in oedema followed a by an acceleration of granulation tissue formation.

Effect of Vacuum-assisted closure on chronic

VAC has been used successfully to treat venous ulcers,arterial insufficiency and neuropathic

(diabetic) ulcers.

The recommended treatment parameters are:

Cycle: continuous treatment for the duration of the therapy

Duration: ideally 24 h a day

Target pressure: 50-75 mmHg

Dressing change: every 48 h or every 12 h if the wound is infected.

All the adjunctive therapies described can assist in wound healing if used in an appropriate manner. Most act by assisting in the resolution of inflammation so that the proliferative phase of healing begins earlier leading to speedier wound closure. Cell activity is jump-started by changes in membrane permeability, with the result that healing is accelerated. The clinician responsible for treating n injured patient is better equipped to select the most beneficial treatment for the patient if armed with an understanding of each therapy and of its mode of action.

The ultimate test of any therapy is the way in which the patient responds to it. Systemic changes in the patient should be documented and their wounds should be imaged non-invasively throughout healing by, for example, digital photography and high-resolution diagnostic ultrasound. Changes in wound structure and physiology should be quantified where possible so that valid comparisons can be made. The sharing of findings vi peer-reviewed journals and conference presentations will add to our understanding of these therapies and improve the lot of injured patients Patients heal their wounds themselves: a major role of the nurse is to help them do so.

Argenta and Morykwas16 determined that intermittent negative pressure at 125 mmHg promoted wound healing by improving blood flow, granulation tissue growth rates and nutrient flow while reducing bacterial levels.

Based on these findings, Kinetic Concepts (San Antonio, Texas) developed the VAC system. The VAC consists of a wound dressing (a charcoal - impregnated sponge - like material) connected by tubing to a wound canister, with a pump that creates negative pressure. A transparent drape or film over the dressing establishes the seal needed to create a vacuum. The pump can be adjusted for various levels of intermittent or continuous pressure. Exudate is collected in the cainster. The VAC also is said to reduce edema.

METHODOLOGY:

Source of data:

Eligible patients who are admitted to Surgical wards, K.L.E.'s Dr. Prabhakar Kore Hospital attached to J.N.M.C, Belgaum will be taken for this study after obtaining their consent.

Method of collection of the data:

- **Study design-**

  A RANDOMISED CONTROL TRIAL

- **Sample Size:**

  Applying formula

  $n= [2(Z\alpha + Z\beta)^2 (S_1^2 + S_2^2)] \div (X_1 - X_2)^2$

  a total of 60 cases will be taken up for the study. They

  will be divided into two groups of 30 each by computer

  generated random numbers.

By reviewing literature, it was found that a study conducted by Asghar Akbari ; mean foot ulcer area decreased from 46 to 43 mm^2 in control group. Effect size double that of control.

Control group (X_1) = 3

Experimental group (X_2) = 6

Therefore, $(X_1 - X_2)^2 = 9$

$Z\alpha = 1.96$; $Z\beta = 0.84$

$S_1 = S_2 = 3$ (Standard Deviation)

A total of 60 cases were taken up for the study.

The cases were allotted into either of the two groups by an 'Opaque Envelop Method'.

- **Study Period:**

  January 2013 – December 2013.

Inclusion Criteria

- Diabetic patients Type 1/Type 2 of age > 18 years
- Size:
- < 15 cm at the greatest dimensions.
- Wagner grade 2 class.
- Clean/Unclean wounds
- Patients with controlled diabetes with fasting blood glucose levels 100-150 mg/dl(at the time of admission and 24 hours later).
- Patients giving consent for the trial.

Exclusion Criteria:

- Immunocompromised patients.
- Osteomyelitis.
- Collagen vascular diseases.
- Malignancy.
- Patients who are not on regular follow up.
- Ischemia/Peripheral Vascular Disease

Procedure:

Based on the selection criteria patients were enrolled after obtaining written informed consent.

Further they were divided into two groups by computer generated random number as

- Group A – received Vaccum Assisted Closure (VAC) Therapy.
- Group B – received Conventional Topical Povidone Iodine Dressing (CTPID).

Debridement was done if necessary.

DRESSING:

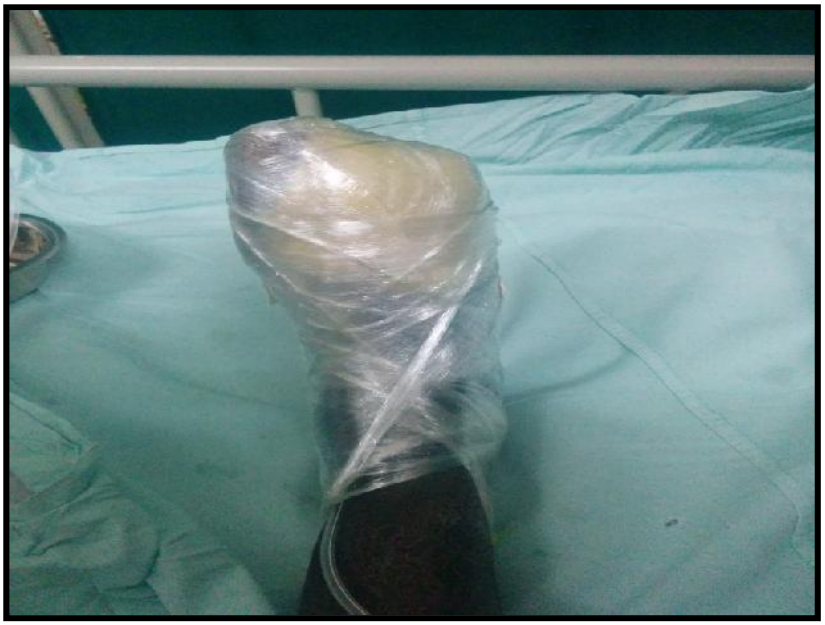
In group A , Vaccum Assisted Closure (VAC) Therapy is done.

In group B , conventional povidone iodine dressing (CTPID) is done.

VAC Dressing:

VAC DEVICE CONSISTS OF-

- VAC Negative Unit
- Foam
- Drain tube
- Transparent Adhesive tape



PHOTOGRAPH 1:MATERIALS USE FOR VACT

Method of application :

Prior to application of VAC therapy, radical debridement is performed on all wounds. The method of application is as follows:

- Cleanse the wound.
- Cut foam fit to gently so as to fit the the wound cavity.
- Put a drain in a curled manner.
- Trim plastic drapes to cover the foam and 3-5 cm around intact wound tissue.
- Connect drain to a negative pressure unit (wall suction).
- A standard negative pressure is maintained at 100-125 mmHg.
- Change of dressing performed at regular intervals of 48-72 hours.
- At each change, the wound is carefully assessed to see if slough had surfaced, so as to perform additional debridement before placing a new dressing.

Treatment Parameters:

1. Cycle – Continuous application (it is disconnected whenever patient wants to visit wash room and reconnected back)

2. Duration - Maximum 14 days.

3. Target pressure – 100-125 mmHg

Conventional Povidone Iodine Dressing :

- The wound is cleaned with povidone iodine.
- A gauze soaked in povidone iodine is placed on the wound and dressing is done.

PHOTOGRAPH 2: CONVENTIONAL TOPICAL POVIDONE IODINE

ASSESSMENT:

At each dressing change, the wound will be carefully assessed to determine if the wound is healthy, clean and granulating by noting various parameters as mentioned in Wound Observations (VI).

If infection or slough is surfaced in the wound, additional surgical debridement is performed, before a new dressing could be applied.

An area of an ulcer is assessed on Day 0 and Day 10. To calculate and compare area of an ulcer, a digital image of the ulcer is taken with the help Sony Ericsson Xperia Arc Camera,8 MP.

It is transferred to laptop and the same image is processed using Adobe Photoshop Elements.

The ulcer area and the standard square (1cm × 1 cm) of the graph are painted black.

The black areas are calculated in pixels, and the standard square and ulcer are compared to calculate the area of an ulcer.

Wound will be observed for granulation, discharge and culture-sensitivity at the end of each week and recorded.

RESULTS:

As mentioned 60 cases were taken up for the study.

AGE WISE DISTRIBUTION:

The age wise distribution of the patients in this study is as given below:

GRAPH 1:

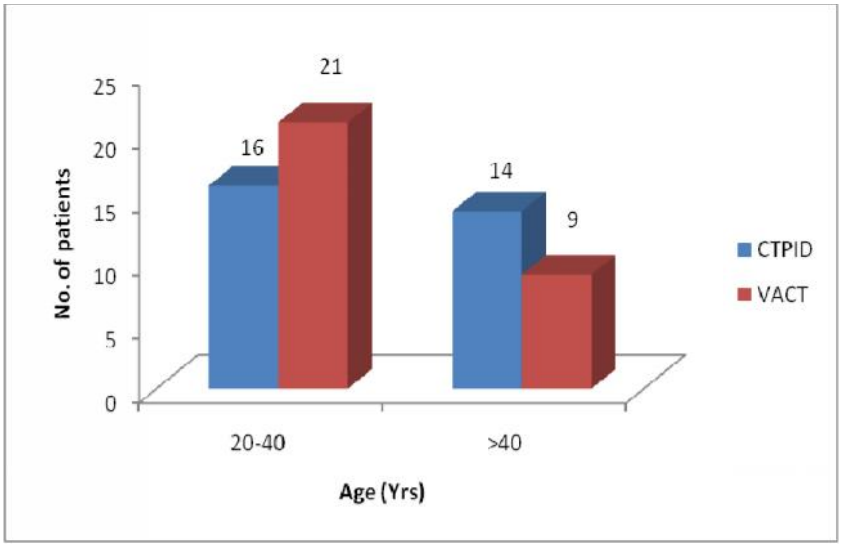


TABLE 1

		Group		
		CTPID	VACT	Total
Age	20-40	16 53.3%	21 70.0%	37 61.7%
	>40	14 46.7%	9 30.0%	23 38.3%
Total		30 100.0%	30 100.0%	60 100.0%

	Value	df	p-value
Pearson Chi-Square	1.763	1	.184

TABLE 1

The above table shows that, out of 30 patients in CTPID group 16 patients were in the age group of 20-40 years and 14 were more than 40 years.In VACT group ,out of 30 patients, 21 were in age group of 20-40 years and 9 were in the group of more than 40 years.The P value between both the groups was statistically not significant (p-value 0.184).

SEX WISE DISTRIBUTION:

GRAPH 2

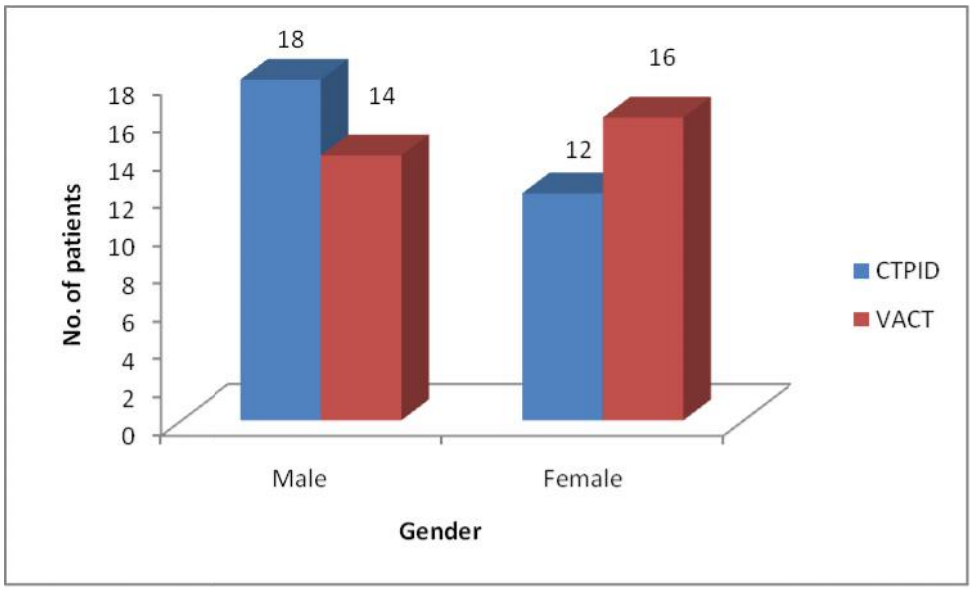


TABLE 2

		Group						
		CTPID	VACT	Total		Value	df	p-value
Sex	Male	18 60.0%	14 46.7%	32 53.3%	Pearson Chi-Square	1.071	1	.301
	Female	12 40.0%	16 53.3%	28 46.7%				
Total		30 100.0%	30 100.0%	60 100.0%				

TABLE 2

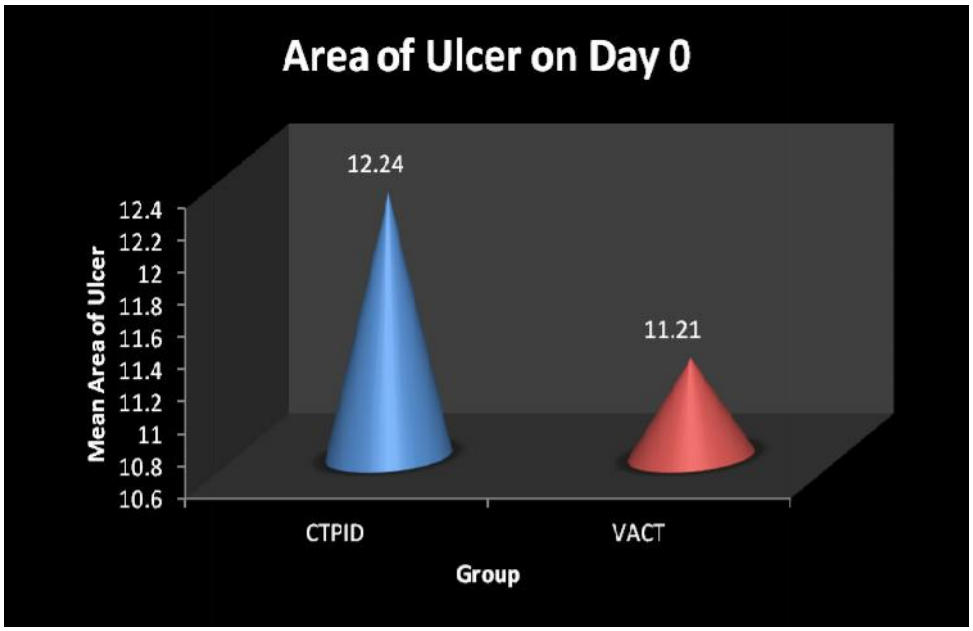
The above table and graph shows that, there were 18 male and 12 female patients in CTPID group and 14 males and 16 females in VACT group. The P value in both the groups was statistically not significant.

AREA OF AN ULCER

DAY 0

	Group	N	Mean	Std. Deviation	Std. Error Mean	t-value	p-value
Area of the ulcer 0	CTPID	30	12.24	4.666	.852	.815	.419
	VACT	30	11.21	5.115	.934		

TABLE 3



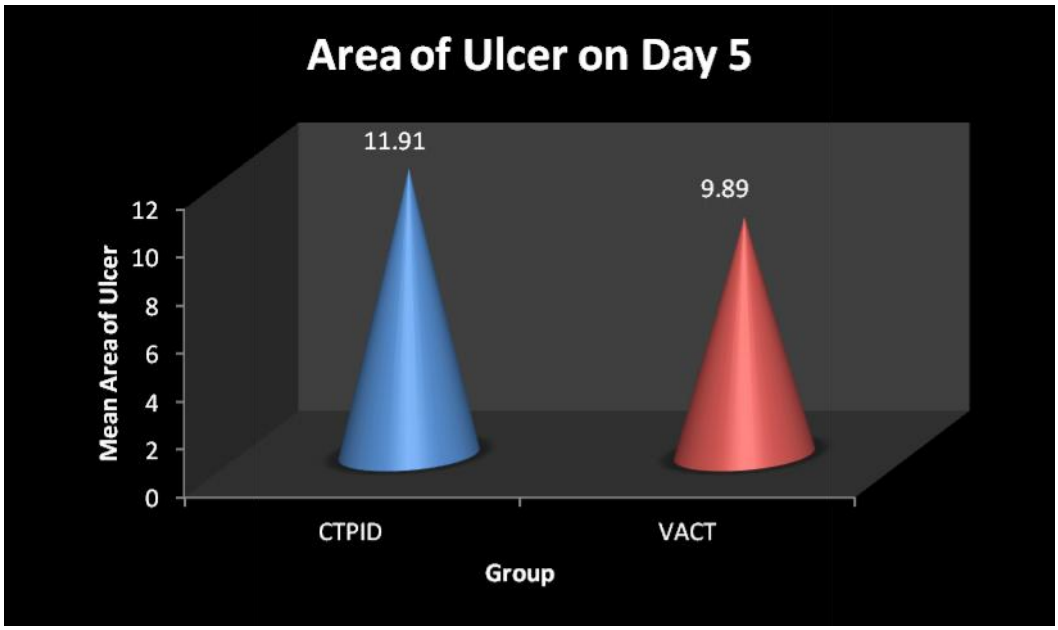
GRAPH 3

The above table and graph shows that the mean area of an ulcer in CTPID group on day 0 was 12.24 cm^2 as compared to 11.21 cm^2 in VACT group. The calculated p value was 0.419 which is more than 0.05 and hence was statistically not significant.

DAY 05

Group	N	Mean	Std. Deviation	Std. Error Mean	t-value	p-value
CTPID	30	11.91	4.726	.863	1.610	.113
VACT	30	9.89	4.958	.905		

TABLE 4



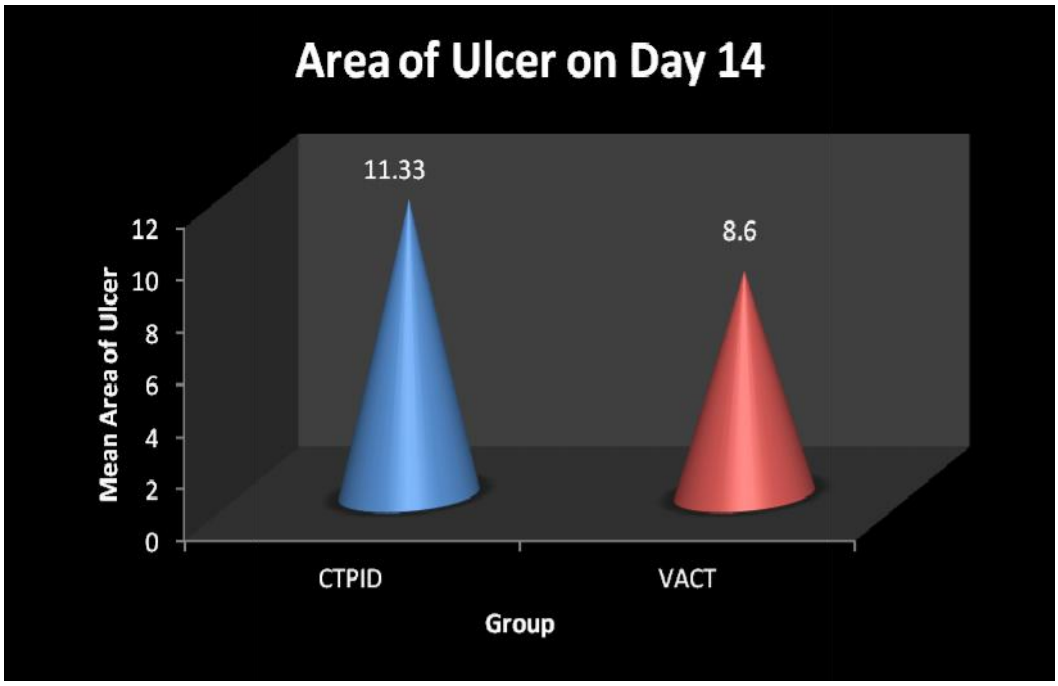
GRAPH 4

The above table and graph shows that the mean area of an ulcer in CTPID group on day 5 was 11.91 cm^2 as compared to 9.89 cm^2 in VACT group. The calculated p value was 0.113 which is more than 0.05 and hence was statistically not significant**.**

DAY 14

Group	N	Mean	Std. Deviation	Std. Error Mean	t-value	p-value
CTPID	30	11.33	4.593	.839	2.239	.029
VACT	30	8.60	4.861	.888		

TABLE 5



GRAPH 5

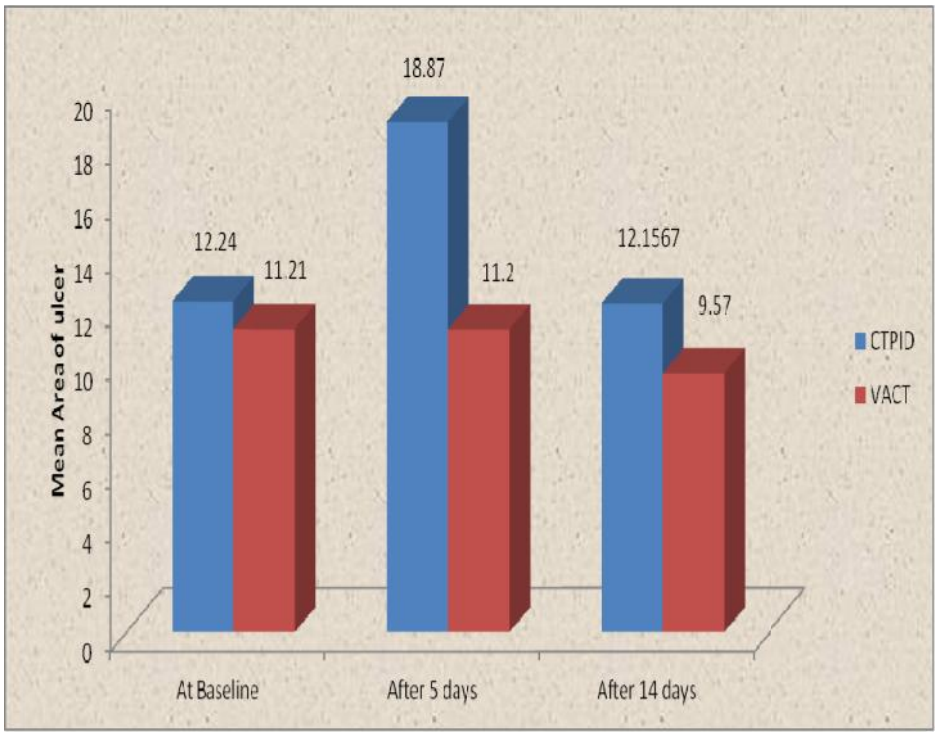
The above table shows that the mean area of an ulcer in CTPID group on day 14 was 11.33 cm^2 as compared to 8.6 cm^2 in VACT group. The calculated p value was 0.029 which is less than 0.05 and hence was statistically significant. Thus, there is significant reduction in wound area in VACT.

TABLE 6

Summary of area of ulcer on day 0, 5, 14

	Group	N	Mean	Std. Deviation	Std. Error Mean	t-value	p-value
Area of the ulcer 0	CTPID	30	12.24	4.666	.852	.815	.419
	VACT	30	11.21	5.115	.934		
Area of the ulcer 5 days	CTPID	30	11.91	4.726	.863	1.610	.113
	VACT	30	9.89	4.958	.905		
Area of the ulcer 14 days	CTPID	30	11.33	4.593	.839	2.239	.029
	VACT	30	8.60	4.861	.888		

Total 60 patients were included in our study of diabetic foot ulcers. P value less than 0.05 was considered to be significant.



GRAPH 6

The mean area of an ulcer on day 0 in CTPID group was 12.24cm^2 and in VACT group was 11.21 cm^2. On 5th day after application of respective treatment, the

mean area was 11.91 cm^2 inCTPID group and 9.89 cm^2 in VACT group. This value when statistically compared was not significant (P value of 0.113).

The mean surface area of an ulcer on day 14 was 11.30cm^2 in CTPID and 8.6cm^2 in VACT group. This decrease in surface area on day 14^{th} was statistically significant (P value of 0.029).

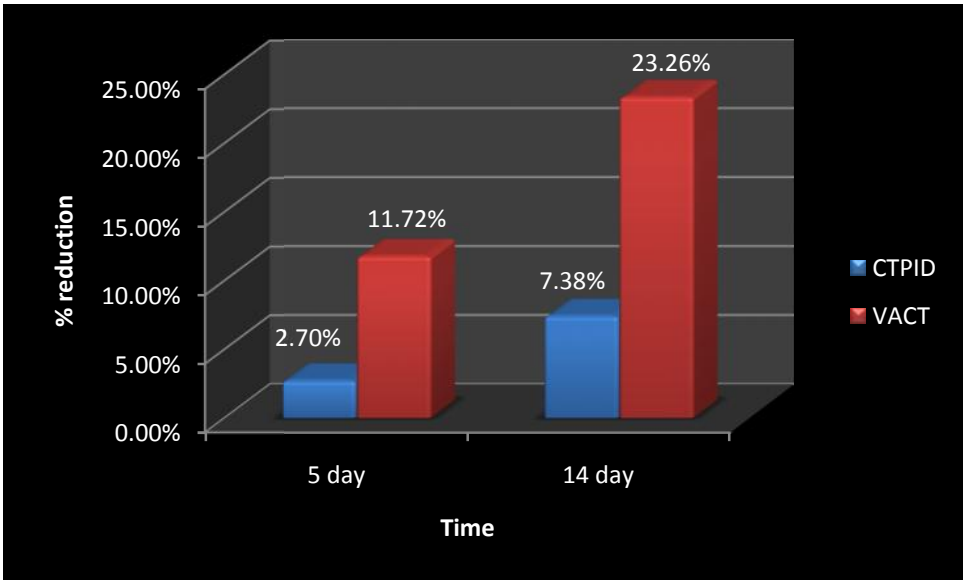
TABLE 7

PERCENTAGE REDUCTION IN AREA OF AN ULCER

Group	Percentage reduction on day 5	Percentage reduction day 14	p-value
CTPID	2.7%	7.38%	<0.001
VACT	11.72%	23.26%	<0.001
p-value	<0.001	<0.001	

Similarly the percent reduction in area of an ulcer on 5thday in CTPID group was 2.7% and VACT group was 11.72%.Total percent reduction in area of an ulcer on 14th day in CTPID group was 11.72% and 23.23% in VACT group.

P value < 0.001 which was statistically significant.



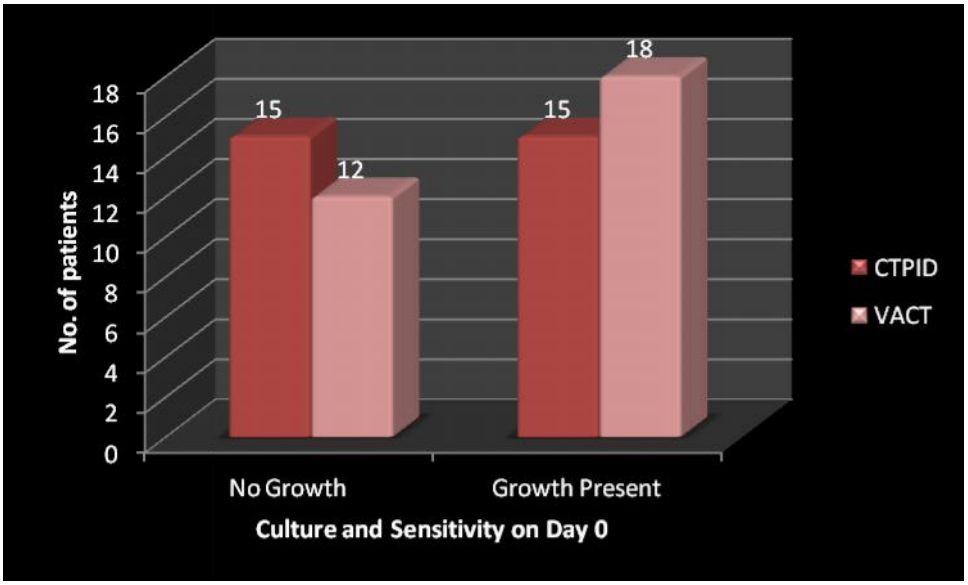
GRAPH 7

TABLE 8

INFECTION STATUS OF AN ULCER (CULTURE)

DAY 0

		Group		
		CTPID	VACT	Total
Culture on Day 0	No growth	15	12	27
		50.0%	40.0%	45.0%
	Growth present	15	18	33
		50.0%	60.0%	55.0%
Total		30	30	60
		100.0%	100.0%	100.0%
		Value	df	P-value
Pearson Chi-Square		0.606	1	.436

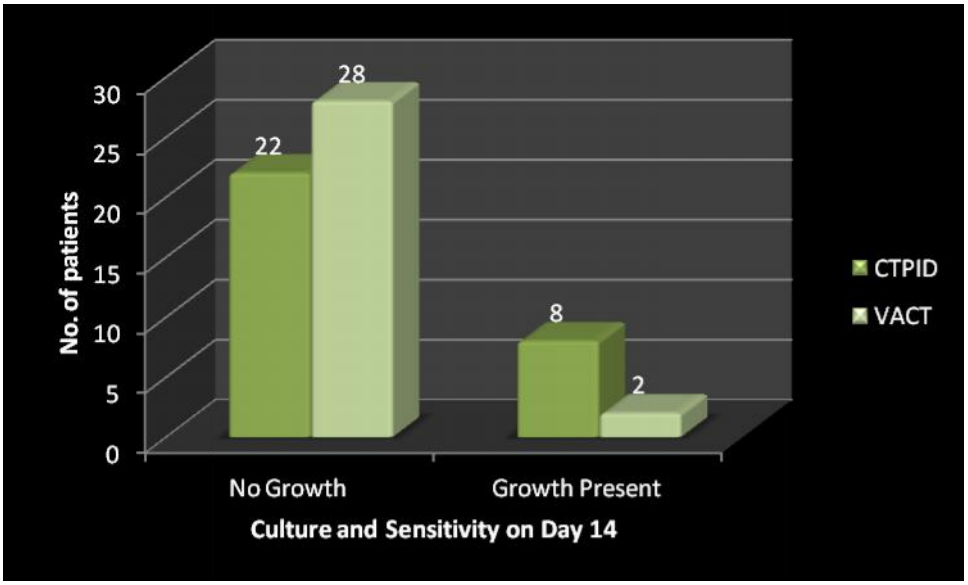


The above table and graph shows that out of 30 patients on day 0 in CTPID group,15 patients showed no growth of organisms whereas 15 patients showed positive growth. In VACT group, out of 30 patients 12 patients had no growth and 18 patients showed positive growth. The calculated p value was 0.436 which was statistically not significant**.**

TABLE 9
INFECTION STATUS OF ULCER

DAY 14

		Group		
		CTPID	VACT	Total
Culture on Day 14	No growth	22	28	50
		73.3%	93.3%	83.3%
	Growth present	8	2	10
		26.7%	6.7%	16.7%
Total		30	30	60
		100.0%	100.0%	100.0%
		Value	df	P-value
Pearson Chi-Square		4.320	1	.038



GRAPH 9

The above table and graph shows that out of 30 patients on day 14, in CTPID group, 22 patients showed no growth of organisms whereas 8 patients showed positive growth. In VACT group, out of 30 patients 28 patients had no growth and only 2 patients showed positive growth. The calculated p value was 0.038 which was statistically significant. Thus, there was a significant difference between both the groups on day 14 for culture of an ulcer.

DISCUSSION:

Total 60 patients were included in our study of diabetic foot ulcers. P value less than 0.05 was considered to be significant. The mean area of an ulcer on day 0 in CTPID group was 12.24cm^2 and in VACT group was 11.21 cm^2. On 5th day after application of respective treatment, the mean area was 11.91 cm^2 inCTPID group and 9.89 cm^2 in VACT group. This value when statistically compared was not significant (P value of 0.113). The mean surface area of an ulcer on day 14 was 11.30cm^2 in CTPID and 8.6cm^2 in VACT group. This decrease in surface area on day 14th was statistically significant (P value of 0.029).

Total percentage reduction in area on day 5 was 2.7% in CTPID group and 11.2 % in VACT group.On 14th day, decrease in total percentage of an area was 7.38% in CTPID group and 23.26% in VACT group (p value 0.001 was statistically significant).

When culture and sensitivity was compared, on day 0 , 15 patients out of 30 CTPID had growth of organisms whereas 18 patients out of 30 showed growth in VACT group. When statistically compared was not significant (P value of 0.436)

Repeat culture on day 14th ,22 patients showed growth in CTPID group whereas in VACT group only 2 patients had positive growth. When compared,there was statistically significant difference (p value – 0.038) between CTPID and VACT group on day 14th.Thus application of VAC helps to reduce infection status of an ulcer.

In a study conducted by Abdullah Etoz,mean age of the patients in the NPWT group was 66.2 years (range, 54-77) and 64.7 years (range, 56-74) in the control group (P = 0.506). Before treatment, the mean area was 109 cm^2 in the NPWT group and 94.8 cm^2 in control group (P = 0.729). There was no significant difference in

groups regarding initial wound surface area and patient age ($P > 0.05$)Following therapy, the mean wound surface area decreased 20.4 cm^2 (109 cm^2 to 88.6 cm^2) in the NPWT group, and decreased 9.5 cm^2 (94.8 cm^2 to 85.3 cm^2) in the control group ($P = 0.032$). The difference in the rate of surface area decrease between the groups was significant. The surface area of wounds in the NPWT group was reduced more effectively than control group wounds ($P < 0.05$).[47]

The following table shows

VARIABLES	Study by Abdullah Etoz		Present Study	
	VACCUM GROUP	CONTROL GROUP	VACCUM GROUP	CONTROL GROUP
1.SAMPLE SIZE	12	12	30	30
2.MEAN AGE	66.2 YRS	64.7YRS	35.7	36.4
3.AREA OF AN ULCER(MEAN) BEFORE	109cm^2	94.8cm^2	11.21cm^2	12.24cm^2
AFTER	80.6cm^2	85.3cm^2	8.60cm^2	11.33cm^2

In another study conducted by Isago T, Nozaki M. Kikuchi Y, Honda Tand Nakazawa H, where the efficacy of topical negative pressure moist dressings was assessed, in pressure ulcers, by comparing the mean rate of reduction of ulcer area and depth before and after application of the dressing. The mean reduction in ulcer depth and surface area were 61.2% and 55.1% respectively. This study clearly shows the

efficacy of topical negative pressure dressings in the management of pressure ulcers which are usually resistant to healing by conventional types of wound dressings. [58]

A study was conducted to evaluate the impact of negative pressure wound therapy after severe open fractures on deep infection. 59 patients with 63 severe high energy open fractures were enrolled in this study. 23 patients with 25 fractures randomised to the control group and underwent initial irrigation and debridement followed by standard fine mesh gauze dressing, with repeat irrigation and debridement every 48-72 hours until wound closure. 35 patients randomised to the NPWT group and had identical treatment except that NPWT was applied to the wounds between irrigation and debridement procedures until closure. The study concluded that control patients developed 2 acute infections (8%) and 5 delayed infections (20%), for a total of 7 deep infections (28%), whereas NPWT patients developed 0 acute infections, 2 delayed infections (5.4%) for a total of 2 deep infections (5.4%). There is a significant difference between the groups for total infections (P=0.024). The study concluded that NPWT represents a promising new therapy for severe open fractions after high energy trauma.[49]

The concept of moist wound dressings which came into vogue in 1960 whichrevolutionized wound care .This led to further research in this direction leading to influx of many products .People have tried various non conventional topical agents in wound healing such as aleovera, antacids , benzoyl per-oxide ,collagen ,gentian violet, impregnated guaze, insulin, mercurochrome oxygen therapy, sugar and vinegar. Each claiming a better wound healing rate than the others. As the concept of outcome based medicine evolved, the need for better wound dressing modality became more acute.

Now wound dressing systems were compared not only on the basis of the rate of granulation tissue formed or the rate of wound healing but also on the cost and duration of hospital stay of the patient which was considered as a measure of the morbidity of the patient.

In a study,conducted by Luca DallaPaolaand et al [50], it was seen that infection control was better and faster in the VACT group as compared to the CTPID group (10±18 days Vs 19±13 days). In our study 50% and 60% patients belonging to the CTPID and VACT group respectively showed presence of infection on day 0 of examination, when these same patients were compared on the day 14 it was found that 26.7% and only 6.7% of patients in the CTPID and VACT group respectively showed presence of infection. Thus in our study also it was found that infection control is better and faster in the VACT group as compared to the CTPID.

CONCLUSION

Chronic Diabetic Foot ulcers present a significant challenge to treating physicians. Treatment involves multiple modalities including debridement, assessment, and treatment of infection, revascularization if indicated, and sufficient off-loading of the foot . A key component of the healing process is debridement because it enables removal of devitalized and necrotic tissue. Debridement is critically important to the initiation of healing. VACT and other wound healing technologies work in conjunction with debridement as the foundation upon which the wound healing process can begin . As observed in this clinical trial, the use of VACT in concurrence with debridement of the affected foot increases the number of Diabetic foot ulcers healed and decreases the length of time required for ulcer healing compared with CTPID. In addition, the prescription of off-loading may have also contributed to positive results in both groups. Therefore, it appears that VACT in addition to established standards of care enhances successful healing and closure of Diabetic foot ulcers.

In our present study it was concluded that reduction in the mean surface area of the ulcer was significantly more in the VACT group as in comparison to the CTPID group. Overall control of infection was better than the as compared to conventional dressing group.

Thus we conclude that mean surface area of the ulcer is reduced and infection is controlled better in the VAC Therapy, making VAC a superior option in the management of patients with Diabetic foot Ulcers.

But further studies with larger population will be needed in the future before VAC Therapy can be added to the wide spectrum of treatment modalities available in the management of diabetic ulcers and ulcers of other aetiology.

SUMMARY

Vaccum Assisted Closure Therapy (VACT) has a significant effect in reduction of total area in diabetic foot ulcers as compared to conventional povidone iodine dressing.

VACT also shows a significant reduction in infection status of a diabetic foot ulcers.

The method of application of Vaccum Assisted Closure as used in this study is a simple, effective, inexpensive as compared to Vaccum devices which are more expesive with similar outcomes and it has beneficial effects when compared to conventional povidone iodine dressing.

In a country like India,where there is an increasing demand for treatment of diabetic foot ulcers ,a newer modality can be tried as compared to conventional techniques considering all the factors.

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Serial ID	IP NO.	Age	Sex	Address	Occupation	Education	Socio-econon	H/o Diabetes:	If answer to 8 is yes, current status:	On medication for diabetes:	H/o Hypertension	If Yes to Q12, current status	Level of hemoglobin	Applicant willing to give consent	Chief complai	Mode of onse	Duration of ul	Received any treatment elsewhere for same	If yes to Q.23, Surgical or medical treatment?	Outcome of treatment?	Method of dressing A or B?	WOUND OBSE							
																						Site	Surrounding Skin	Size(Before dressing Day 0)	Size (Day 5)	Size (Day of discharge(if the >95% red granulation is present) /day 14)	Shape (Before dressing	Shape(Day 5)	Shape (Day of discharge(if the >95% red granulation is	Edge (Before dressing
1	523451	2	1	1	4	5	2	1	1	1	2	3	3	1	6	1	3	1	2	1	1	Right Foot	1	1	1	1	3	3	3	1
2	543487	3	1	2	3	3	2	1	1	1	2	3	3	1	2	1	2	1	2	1	2	Right Foot	3	2	2	2	3	3	3	2
3	523321	2	1	1	4	4	2	1	1	1	1	1	1	1	1	1	1	2	3	4	2	Left Foot	1	1	1	1	3	3	3	3
4	542177	2	2	1	2	2	1	1	1	1	2	3	1	1	2	3	3	1	2	2	2	Right Foot	1	3	3	3	3	3	3	1
5	556789	3	1	1	3	2	1	1	1	1	2	3	2	1	1	1	2	1	2	1	1	Right Foot	2	2	2	2	3	3	3	2
6	537621	2	1	1	1	1	1	1	1	1	2	1	1	1	6	4	3	1	2	1	1	Left Foot	3	2	2	2	3	3	3	3
7	521347	3	1	1	3	3	2	1	1	1	2	3	1	1	3	3	2	2	3	4	2	Right Foot	1	1	1	1	2	2	2	1
8	534121	2	2	2	2	2	1	1	1	1	2	3	2	1	1	1	1	2	3	4	2	Right Foot	3	3	3	3	3	3	3	3
9	543278	2	1	1	4	4	2	1	1	1	2	3	1	1	4	2	2	1	2	1	2	Left Foot	1	1	1	1	2	2	2	1
10	534272	3	1	1	1	1	1	1	1	2	1	1	3	1	6	4	2	1	1	2	1	Left Foot	4	3	3	3	3	3	3	4
11	523472	3	1	1	3	3	2	1	1	1	2	3	2	1	4	4	3	2	3	4	2	Right Foot	1	1	1	1	1	1	1	2
12	527861	2	2	1	3	3	2	1	1	1	2	3	1	1	1	1	2	1	2	1	1	Left Foot	4	2	2	2	3	3	3	2
13	512361	2	2	1	2	2	1	1	1	1	2	3	2	1	6	3	3	1	2	2	2	Left Foot	2	3	3	3	3	3	3	5
14	511890	2	1	2	4	4	2	1	1	1	2	3	3	1	6	4	2	2	3	4	1	Right Foot	4	3	3	3	3	3	3	1
15	537789	3	1	1	2	1	1	1	1	1	1	1	3	1	1	1	2	1	2	2	1	Right Foot	1	2	2	2	1	1	1	2
16	523488	2	1	2	4	5	2	1	1	1	2	3	3	1	4	1	2	2	3	4	2	Left Foot	2	2	2	2	3	3	3	2
17	523678	3	1	2	3	3	2	1	1	1	2	3	3	1	1	1	2	2	3	4	1	Right Foot	1	1	1	1	1	1	1	1
18	522348	3	2	2	4	4	2	1	1	1	2	3	2	1	2	2	2	2	3	3	1	Left Foot	2	3	3	3	3	3	3	1
19	512446	2	1	2	4	2	1	1	1	1	2	3	3	1	6	4	3	2	3	4	2	Left Foot	2	2	2	2	3	3	3	4
20	513456	2	2	1	3	3	1	1	1	2	2	3	1	1	6	4	2	2	3	4	2	Left Foot	4	2	2	2	3	3	3	1
21	523772	3	1	2	3	2	1	1	1	1	2	3	2	1	2	3	2	2	3	4	1	Left Foot	3	2	2	2	3	3	3	4
22	547891	3	2	2		2	2	1	1	1	2	3	2	1	3	3	2	2	3	4	2	Right Foot	4	2	2	2	2	2	2	2
23	524332	3	2	2	2	1	2	1	1	1	2	3	2	1	2	3	2	1	2	2	1	Right Foot	1	2	2	2	3	3	3	3
24	534908	2	2	2	2	2	2	1	1	1	2	3	3	1	1	1	2	2	3	4	2	Left Foot	2	1	1	1	3	3	3	4
25	526225	2	2	2	3	3	2	1	1	1	2	3	1	1	4	2	3	2	3	4	1	Right Foot	1	2	2	2	3	3	3	2
26	562425	2	2	2	2	2	1	1	1	1	2	3	1	1	5	4	1	2	3	4	2	Left Foot	4	2	2	2	2	2	2	2
27	552345	2	1	1	2	2	1	1	1	1	2	3	3	1	1	1	3	1	2	1	1	Left Foot	1	2	2	2	2	2	2	4
28	515417	3	2	2	2	2	1	1	1	1	2	3	3	1	1	1	2	2	3	4	2	Right Foot	4	3	3	3	3	3	3	4
29	516521	2	1	2	3	3	2	1	1	1	2	3	3	1	5	4	2	2	3	4	1	Left Foot	1	2	2	2	3	3	3	3
30	526441	3	2	2	1	1	1	1	1	2	1	1	3	1	4	4	2	2	3	4	1	Right Foot	2	2	2	2	1	1	1	2
31	542367	2	1	1	3	3	2	1	1	1	2	3	2	1	1	1	2	1	2	2	2	Right Foot	4	1	1	1	1	1	1	3
32	513253	2	2	1	2	1	1	1	1	1	2	3	1	1	6	1	3	1	2	1	1	Left Foot	1	2	2	2	2	2	2	2
33	548642	3	1	1	3	3	2	1	1	1	2	3	2	1	4	4	3	2	3	4	2	Right Foot	1	1	1	1	1	1	1	2
34	533261	2	2	1	3	3	2	1	1	1	2	3	1	1	1	1	2	1	2	1	1	Left Foot	4	2	2	2	3	3	3	2
35	529520	2	2	1	2	2	1	1	1	1	2	3	2	1	6	3	3	1	2	2	2	Left Foot	2	3	3	3	3	3	3	5
36	540134	2	1	2	4	4	2	1	1	1	2	3	3	1	6	4	2	2	3	4	1	Right Foot	4	3	3	3	3	3	3	1
37	545676	2	2	1	2	2	1	1	1	1	2	3	2	1	6	3	3	1	2	2	2	Left Foot	2	3	3	3	3	3	3	5
38	523581	2	1	2	4	4	2	1	1	1	2	3	3	1	6	4	2	2	3	4	1	Right Foot	4	3	3	3	3	3	3	1
39	525991	3	1	1	2	1	1	1	1	1	1	1	3	1	1	1	2	1	2	2	1	Right Foot	1	2	2	2	1	1	1	2
40	535491	2	1	2	4	5	2	1	1	1	2	3	3	1	4	1	2	2	3	4	2	Left Foot	2	2	2	2	3	3	3	2
41	508875	3	1	2	3	3	2	1	1	1	2	3	3	1	1	1	2	2	3	4	1	Right Foot	1	1	1	1	1	1	1	1
42	533423	3	2	2	4	4	2	1	1	1	2	3	2	1	2	2	2	2	3	3	1	Left Foot	2	3	3	3	3	3	3	1
43	531226	2	1	2	4	2	1	1	1	1	2	3	3	1	6	4	3	2	3	4	2	Left Foot	2	2	2	2	3	3	3	4
44	546671	2	2	1	3	3	1	1	1	2	2	3	1	1	6	4	2	2	3	4	2	Left Foot	4	2	2	2	3	3	3	1
45	510438	2	1	2	4	2	1	1	1	1	2	3	3	1	6	4	3	2	3	4	2	Left Foot	2	2	2	2	3	3	3	4
46	539544	2	2	1	3	3	1	1	1	2	2	3	1	1	6	4	2	2	3	4	2	Left Foot	4	2	2	2	3	3	3	1
47	543122	3	1	2	3	2	1	1	1	1	2	3	2	1	2	3	2	2	3	4	1	Left Foot	3	2	2	2	3	3	3	4
48	542188	3	2	2		2	2	1	1	1	2	3	2	1	3	3	2	2	3	4	2	Right Foot	4	2	2	2	2	2	2	2
49	534732	3	2	2	2	1	2	1	1	1	2	3	2	1	2	3	2	1	2	2	1	Right Foot	1	2	2	2	3	3	3	3
50	523117	2	2	2	2	2	2	1	1	1	2	3	3	1	1	1	2	2	3	4	2	Left Foot	2	1	1	1	3	3	3	4
51	517636	2	2	2	3	3	2	1	1	1	2	3	1	1	4	2	3	2	3	4	1	Right Foot	1	2	2	2	3	3	3	2
52	529520	2	2	2	2	2	1	1	1	1	2	3	1	1	5	4	1	2	3	4	2	Left Foot	4	2	2	2	2	2	2	2
53	543217	2	1	1	2	2	1	1	1	1	2	3	3	1	1	1	3	1	2	1	1	Left Foot	1	2	2	2	2	2	2	4
54	534732	3	2	2	2	2	1	1	1	1	2	3	3	1	1	1	2	2	3	4	2	Right Foot	4	3	3	3	3	3	3	4
55	528716	2	1	2	3	3	2	1	1	1	2	3	3	1	5	4	2	2	3	4	1	Left Foot	1	2	2	2	3	3	3	3
56	528986	3	2	2	1	1	1	1	1	2	1	1	3	1	4	4	2	2	3	4	1	Right Foot	2	2	2	2	1	1	1	2
57	521188	2	1	1	3	3	2	1	1	1	2	3	2	1	1	1	2	1	2	2	2	Right Foot	4	1	1	1	1	1	1	3
58	533182	2	2	1	2	1	1	1	1	1	2	3	1	1	6	1	3	1	2	1	1	Left Foot	1	2	2	2	2	2	2	2
59	544571	3	1	1	3	3	2	1	1	1	2	3	2	1	4	4	3	2	3	4	2	Right Foot	1	1	1	1	1	1	1	2
60	532267	2	1	1	2	2	1	1	1	1	2	3	3	1	1	1	3	1	2	1	1	Left Foot	1	2	2	2	2	2	2	4

																												ANY FOOT DE			IF DEBRIDEMENT HAS BEEN DONE			INVESTIGATIO
Edge (Day 5)	Edge (Day of discharge(if the >95% red granulation is	Margin(Before dressing	Margin (Day 5)	Margin (Day of discharge(if the >95% red granulation is present) /day 14)	Floor (Before dressing	Floor (Day 5)	Floor (Day of discharge(if the >95% red granulation is	Base (Before dressing	Base (Day 5)	Base (Day of discharge(if the >95% red granulation is present) /day 14)	Discharge (Before dressing	Discharge (Day 5)	Discharge (Day of discharge(if the >95% red granulation is present) /day 14)	Slough /necrotic tissue (Before dressing Day 0)	Slough /necrotic tissue (Day 5)	Slough /necrotic tissue (Day of discharge(if the >95% red granulation is present) /day 14)	Area of the ulcer (Before dressing	Area of the ulcer (Day 5)	Area of the ulcer (Day of discharge(if the >95% red granulation is	Culture and sensitivity report (DAY 0	Culture and sensitivity report (DAY 14/DAY OF DISCHARGE)	Toe deformity	Charcot's foot:	Foot drop:	Date	Type of anaesthesia:	No of debridements:	
1	1	1	1	1	1	1	1	2	2	2	2	2	3	3	3	2	6	5	4.4	2	1	2	2	2	1/7/2013	SAB	1	
2	2	1	1	1	2	1	1	2	2	2	4	2	2	2	6	6	12	11.2	9.2	2	1	2	2	2	2/4/2013	SAB	1	
3	3	1	1	1	2	2	1	2	2	2	4	3	1	3	6	6	7	6.7	5.6	2	1	2	2	2	3/6/2013	SAB	1	
1	1	1	1	1	3	1	1	1	1	2	1	1	1	6	6	6	20.4	19.4	18.3	1	1	2	2	2			0	
2	2	1	1	1	3	2	2	3	3	2	4	4	2	4	3	6	13.6	13	11.8	2	1	2	2	2	1/08/2013,7/8/13	SAB	2	
3	3	1	1	1	2	2	2	3	3	3	2	2	2	3	3	3	11.6	11.2	9.7	2	2	1	2	2	15/6/13	SAB	1	
1	1	2	2	1	2	1	1	2	2	2	3	1	1	6	6	6	7.8	6.3	5.5	1	1	2	2	2			0	
2	2	2	1	1	2	2	1	2	2	1	4	2	1	3	2	1	18.6	17.4	15.7	2	1	2	2	1	7/3/2013	SAB	1	
1	1	2	1	1	1	1	1	1	1	1	1	1	1	6	6	6	5.8	5.1	4.3	1	1	2	2	2			0	
4	3	2	1	1	3	2	2	3	3	2	5	4	3	5	4	2	19.6	19.4	19	2	1	2	2	2	4/02/13,21/02/20	SAB	2	
2	1	1	1	1	2	2	1	2	2	1	3	3	1	3	3	6	6.3	4.5	4	1	1	2	2	2	15/2/13	SAB	1	
2	2	1	1	1	2	2	1	3	2	1	2	2	1	3	6	6	7.4	7.3	7.3	1	1	2	2	2			0	
4	**3**	**2**	**2**	**2**	**3**	**3**	**2**	**2**	**2**	**2**	**5**	**4**	**1**	**5**	**3**	**3**	**18.6**	**16.7**	**15.2**	2	1	**2**	**2**	**2**	**3/6/2013**	**SAB**	**1**	
1	1	1	1	1	1	1	1	2	2	2	4	2	4	3	3	3	22.6	22.3	20.2	1	1	1	2	2			0	
2	2	1	1	1	2	2	1	2	2	2	1	1	1	2	2	6	13.3	12.9	12.1	1	1	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	3	6	6	9.4	7.5	6.5	2	1	2	2	2	4/4/2013	SAB	1	
1	1	2	2	2	2	2	2	2	2	2	4	2	2	2	2	2	6.4	6.4	6.3	2	2	2	2	2			0	
1	1	2	2	3	3	2	1	2	2	2	4	2	2	2	1	2	16.4	16.3	16	2	2	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	2	6	6	9.8	7.7	7	2	1	2	2	2			0	
1	1	2	2	1	2	1	1	2	2	2	4	2	1	2	3	6	10.3	7.8	7	2	1	2	2	2			0	
3	2	2	1	1	2	2	2	3	3	2	4	4	3	5	4	3	11.6	10.2	8.9	1	1	2	2	2	8/6/2013	SAB	1	
2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2	13.6	12.3	11	1	1	2	2	2	7/4/13,10/4/13	SAB	2	
3	3	2	2	2	2	1	1	2	2	2	3	2	2	5	3	2	11.4	11	9.8	2	1	2	2	2	7/9/2013	SAB	1	
4	3	2	2	2	3	2	2	3	2	2	5	4	2	5	4	2	4.8	3.6	3	2	2	1	2	2	4/10/2013	SAB	1	
2	2	1	1	1	1	1	1	1	1	1	1	1	1	6	6	6	12.6	12.1	11.7	1	1	2	2	2			0	
2	2	2	2	2	2	2	1	2	2	2	1	1	1	6	6	6	10.2	8.9	7.7	1	1	2	2	2			0	
3	3	2	2	2	2	2	2	2	2	2	3	3	3	3	2	2	11.2	11	10.5	2	1	2	2	2			0	
3	3	2	2	2	3	2	2	2	2	2	3	3	2	3	2	6	19.8	17.8	16.5	2	1	2	2	2	8/8/2013	SAB	1	
2	2	2	1	1	2	2	1	3	2	2	3	3	1	6	6	6	10.5	10	9.6	1	1	2	2	2			0	
1	1	2	2	2	2	2	2	2	1	1	3	2	2	2	2	6	7.6	7.3	7	2	1	1	2	2			0	
3	3	1	1	1	1	1	1	2	2	2	1	1	1	2	2	6	5.4	4	3.5	1	1	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	2	2	6	9.9	9.3	8.6	1	1	2	2	2				
2	1	1	1	1	2	2	1	2	2	1	3	3	1	3	3	6	7	6.6	4.5	1	1	2	2	2	10/10/2013	SAB	1	
2	2	1	1	1	2	2	1	3	2	1	2	2	1	3	6	6	6.6	6.5	6	1	1	2	2	2			0	
4	3	2	2	2	3	3	2	2	2	2	5	4	1	5	3	3	19.4	17.8	16.4	2	1	2	2	2	11/5/2013	SAB	1	
1	1	1	1	1	1	1	1	2	2	2	4	2	4	3	3	3	22	21.7	21.1	1	1	1	2	2			0	
4	3	2	2	2	3	3	2	2	2	2	5	4	1	5	3	3	18.6	17	15.6	2	1	2	2	2	13/2/13	SAB	1	
1	1	1	1	1	1	1	1	2	2	2	4	2	4	3	3	3	22.6	22.6	22	1	1	1	2	2			0	
2	2	1	1	1	2	2	1	2	2	2	1	1	1	2	2	6	13.3	13.1	13	1	1	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	3	6	6	9.4	8.8	8	2	1	2	2	2	14/4/13	SAB	1	
1	1	2	2	2	2	2	2	2	2	2	4	2	2	2	2	2	6.4	6.4	6.4	2	2	2	2	2			0	
1	1	2	2	3	3	2	1	2	2	2	4	2	2	2	1	2	16.4	16.3	16.3	2	2	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	2	6	6	9.9	8.7	6.7	2	1	2	2	2			0	
1	1	2	2	1	2	1	1	2	2	2	4	2	1	2	3	6	11.2	9.9	7.8	2	1	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	2	6	6	9.8	8.8	6.7	2	1	2	2	2			0	
1	1	2	2	1	2	1	1	2	2	2	4	2	1	2	3	6	10.3	8.9	7.5	2	1	2	2	2			0	
3	2	2	1	1	2	2	2	3	3	2	4	4	3	5	4	3	11.6	11.6	11.3	1	1	2	2	2	2/10/2013	SAB	1	
2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2	13.6	10.2	8.7	1	1	2	2	2	3/3/13,9/3/13	SAB	2	
3	3	2	2	2	2	1	1	2	2	2	3	2	2	5	3	2	11.4	11.4	11	2	1	2	2	2	17/8/13	SAB	1	
4	3	2	2	2	3	2	2	3	2	2	5	4	2	5	4	2	4.8	4	3.1	2	2	1	2	2	14/3/13	SAB	1	
2	2	1	1	1	1	1	1	1	1	1	1	1	1	6	6	6	12.6	12.2	11.7	1	1	2	2	2			0	
2	2	2	2	2	2	2	1	2	2	2	1	1	1	6	6	6	10.2	8.9	5.5	1	1	2	2	2			0	
3	3	2	2	2	2	2	2	2	2	2	3	3	3	3	2	2	11.2	11	10.5	2	1	2	2	2			0	
3	3	2	2	2	3	2	2	2	2	2	3	3	2	3	2	6	19.8	19	18.3	2	1	2	2	2	11/9/2013	SAB	1	
2	2	2	1	1	2	2	1	3	2	2	3	3	1	6	6	6	10.5	10.4	9.7	1	1	2	2	2			0	
1	1	2	2	2	2	2	2	2	1	1	3	2	2	2	2	6	7.6	7	6.7	2	1	1	2	2			0	
3	3	1	1	1	1	1	1	2	2	2	1	1	1	2	2	6	5.4	5	4	1	1	2	2	2			0	
1	1	2	2	1	2	2	1	2	2	2	2	2	1	2	2	6	9.9	9.3	9	1	1	2	2	2				
2	1	1	1	1	2	2	1	2	2	1	3	3	1	3	3	6	7	6.3	5.2	1	1	2	2	2	11/11/2012	SAB	1	
3	3	2	2	2	2	2	2	2	2	2	3	3	3	3	2	2	13.3	13	12.4	2	1	2	2	2			0	