CLINICAL STUDY OF STERILE COLLAGEN PARTICLES (BIOFIL) IN THE MANAGEMENT OF CHRONIC NON HEALING ULCER

BY

DR.RAGHUPATHI.R

Dissertation submitted to the

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the requirements for the degree of

M.S.GENERAL SURGERY – BRANCH I

DEPARTMENT OF GENERAL SURGERY

THANJAVUR MEDICAL COLLEGE AND HOSPITAL

THANJAVUR – 613004.

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

APRIL - 2017

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UNIT CHIEF, PROF.DR. M.ELANGOVAN M.S.,

Department of General Surgery,

Thanjavur Medical College,

Thanjavur-613004

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Dr.M.VANITHAMANI M.S.,Mch.,
The Dean,
Thanjavur Medical College,
Thanjavur – 613004.

Dr. M. ELANGOVAN M.S.,
The Professor and HOD,
Department of General surgery,
Thanjavur Medical College,
Thanjavur – 613004.

DECLARATION BY THE CANDIDATE

I solemnly declare that this Dissertation "**CLINICAL STUDY OF STERILE COLLAGEN PARTICLES (BIOFIL) IN THE MANAGEMENT OF CHRONIC NON HEALING ULCERS "** was done by me in the Department of General Surgery, Thanjavur Medical College, and Hospital , Thanjavur under the Guidance and Supervision of my PROF.DR.M.ELANGOVAN M.S. Department of General Surgery, Thanjavur**,** Medical College, Thanjavur between September 2014 and August 2016.

This Dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University , Chennai in partial fulfilment of University requirements for the award of M.S Degree (GENERAL SURGERY) BRANCH- 1

R.RAGHUPATHI,

Postgraduate Student,

Department of General Surgery,

Thanjavur Medical College.

Thanjavur - 613004.

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Dr.R.RAGHUPATHI

ETHICAL COMMITTE CLEARANCE CERTIFICATE

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

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CLINICAL STUDY OF STERILE COLLAGEN PARTICLES (BIOFIL) IN THE MANAGEMENT OF CHRONIC NON HEALING ULCERS
ABSTRACT:
INTRODUCTION:
Chronic non healing ulcer patients may lead into a significant problems and prolonged stress. Pain and discomfort is the main complaint of chronic non healing ulcer patients. Pain may be acute or continuous which may exacerbate with frequent changing of dressings. Wound contracture and scar formation is the end process of any wound healing ulcers. Collagen plays a very important role in the stage of wound healing process. Collagen particles are used in chronic non healing ulcer management to evaluate their efficacy when compared with conventional method dressings in a study which was conducted by our department.
OBJECTIVES:
To compare the efficacy of healing process in chronic non healing ulcer patients using collagen particles with those of conventional method dressings (betadine dressings).
MATERIALS & METHODS:
This study was a non - randomized, a prospective type of study evaluated for a duration of September, 2014 to August, 2016 in Thanjavur Medical College and Hospital, Thanjavur,
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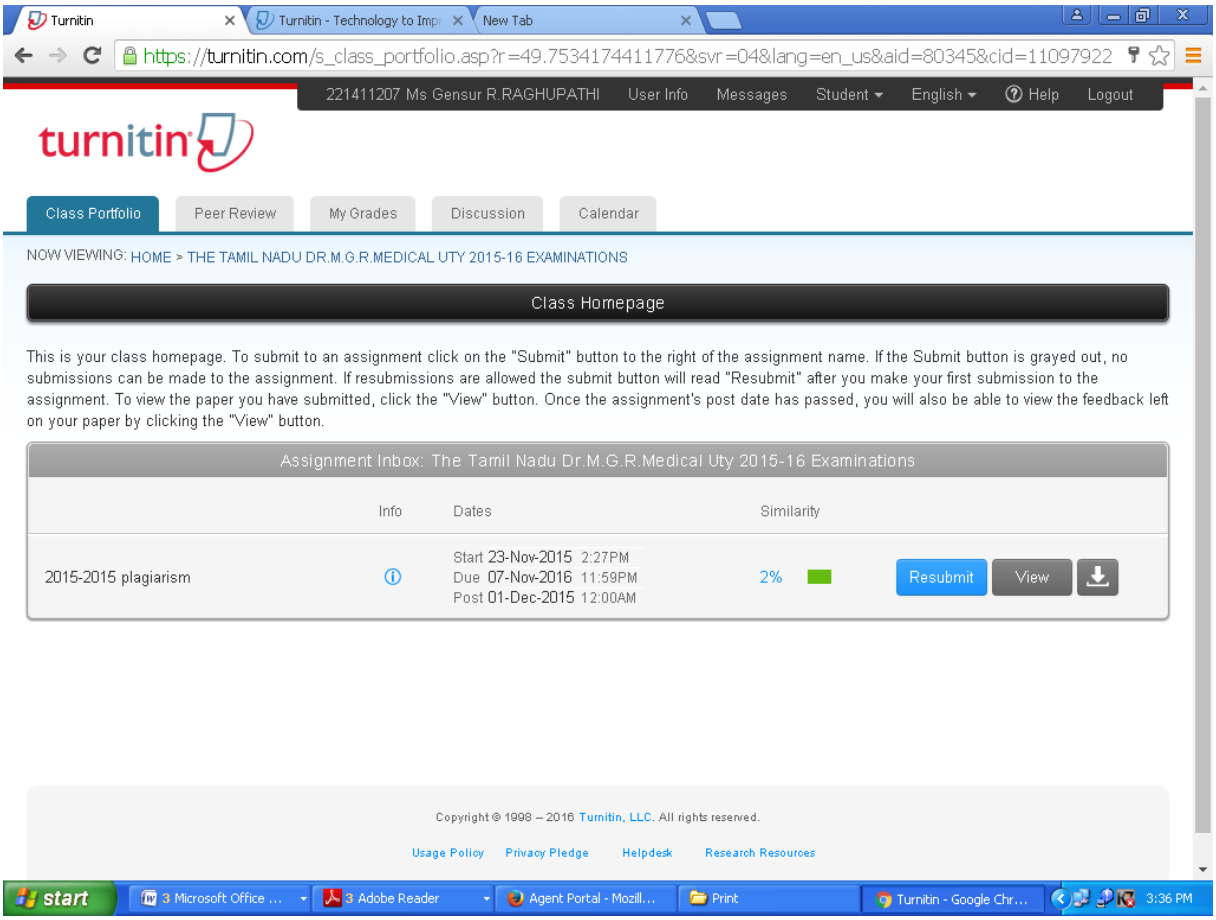
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LIST OF ABBREVIATIONS USED IN THE STUDY & MASTER CHART

DU	**DIABETIC ULCER**
AU	**ARTERIAL ULCER**
VU	**VENOUS ULCER**
TU	**TRAUMATIC ULCER**
PU	**PRESSURE ULCER**
CP	**COLLAGEN PARTICLES**
CM	**CONVENTITIONAL METHODS**
NH	**NON HEALING**
SSG	**SPLIT SKIN GRAFT**

ABSTRACT

INTRODUCTION:

Patients who are suffering from non healing ulcers may lead into a significant problems and prolonged stress. Pain and discomfort are the main complaint of chronic non healing ulcer patients. Pain may be mild or severe, pricking or burning which may exacerbate with frequent changing of dressings. Wound contracture and scar formation is the end result of any wound healing ulcers. Collagen plays a very important role in the stage of wound healing process. Collagen particles are used in chronic non healing ulcer management. To evaluate their efficacy when compared with conventional method dressings this study which was conducted by our department.

OBJECTIVES:

To compare the efficacy of healing process in chronic non healing ulcer patients using collagen particles with those of conventional method dressings (betadine dressings).

MATERIALS &METHODS:

This study was a non - randomized, a prospective type of study evaluated between September, 2014 to August, 2016 in Thanjavur Medical College and Hospital, Thanjavur, Tamilnadu, India. This is the comparative study between collagen particles dressing study group and conventional method dressing

control group. Around a total of 104 patients with chronic non healing ulcers in various region and various types were taken in this study. Study group consisting of 52 patients and control group have 52 patients.

STUDY RESULTS:

The study shows which was a significant increase in the wound healing rate percentage being 92.3% in the study group when compared to the control group percentage being 42.3%.

CONCLUSION OF THE STUDY:

Collagen particles dressings found to be effective in the management of chronic non healing ulcer patients compared to the conventional method of betadine dressing. Collagen particles plays an important role by forming an early granulation tissue and scar formation and reduces the duration of the hospital stay.

KEYWORDS:

Collagen particles, conventional methods, healing, non-healing, amputation

INTRODUCTION OF THE STUDY:

Wound healing process may include many inflammatory cell mediators and other important cells, and the cellular complex protein & extracellular matrix. Wound, ulcer, and tissue damage results from many causes which may vary from surgical wounds, burns wounds, traumatic wounds, diabetic wounds etc. Wound may result following a contusion, hematoma, laceration or an abrasion. Sometimes spontaneous after blister formation.The skin continuation should be adequately maintained because it plays an important role in attaining homeostasis.

Chronic non healing ulcer patients were a complex group and they need adequate treatment to understand the nutrition status, immune system of the patients, psychological problems, physiology and the metabolism of various important internal organs. Some ulcers were very difficult to handle like diabetic foot ulcers, chronic venous ulcers, traumatic ulcers, arterial ulcers and pressure sore ulcers. An ideal dressing method should be economical to patients, very easy to use, adequately available dressing to cover the wound, good relief from pain, protecting wound from serious infections, increases the healing process, provides moisture environment, it should be elastic to wounds, non-allergic and adequate adhesion to the wound and promote epithelisation and granulation tissue & scar formation.

Among numerous type of new dressing methods, biological wound dressing methods like collagen particles plays a crucial role in maintaining physiological interaction between wound bed and surface environment and bacteria which was impermeable to the wound surface. Collagen is the most abundant complex matrix protein in our human body which plays an important role in the end processing of wound healing. It is very essential to create functional integrity of the wound because of the collagen deposition, maturation, and remodelling in the wound healing process.

Collagen is known as an endogenous substance protein, which forms an important cellular complex in our human body connective tissue system and it is very important to our skin also. Collagen stimulates the wound healing process by promoting the formation of granulation tissue and scar. Scar tissue composed of collagen fibres and its components. Collagen forms molecular diversity in the body's protein scaffold.

Collagen particle dressing methods increase the efficacy of the wound healing process over conventional dressing methods like betadine dressings by easy stimulating the collagen formation with higher the efficacy of wound healing rates. The other conventional dressing methods like betadine dressings have smaller amount of collagen formation and scar tissue formation which results in increased in number of days of wound healing process. Collagen particles dressing methods have one more advantage when it was compared to

conventional method dressings in the form of non-immunologic, non-pyrogenic, because of natural form & it is very easy to apply, non-allergic. I also gives good relief of pain because speedy formation of granulation tissue.

Dressings containing sterile collagen particles were used in the study and compared with conventional (betadine) dressings. The aim of our study was to evaluate the efficacy of collagen particles, with an objective to compare the rate of healing process using collagen particles with those of conventional methods.

AIMS:

The aim of study is to evaluate the efficacy of the collagen particles with an objective to compare the rate of healing process using collagen particles with those of conventional dressing.

PRIMARY OBJECTIVES:

To increase the rate of wound healing process

Prevention of infection

To avoid Split Skin Graft whenever possible

SECONDARY OBJECTIVES:

Perception of pain decreased by the patients after applying the dressing methods

To improve the formation of granulation tissue and minimize the scar formation.

LITERATURE REVIEW

DEFINITION OF WOUNDS:

Wound is defined as break in the continuity lining epithelium of the skin, mucous membrane and other tissues, can be accompanied with destruction of structure & function of that skin or other near by tissues.

A Wound is otherwise known as dysfunction of any tissues like soft tissue, bone and other important organs.

A ulcer is considered as one of the types of wounds.

WOUNDS CLASSIFICATION:

I. CLASSIFICATION BY RANK &WAKEFIELD:

a. **TIDY WOUNDS:**

These wounds may resemble surgical site incisions and it may be caused by sharp instruments or objects.

Tidy wound is incised, clean, healthy wound without any cellular or tissue loss.Primary suturing may be done. Healing is achieved by mainly primary intention.

b. **UNTIDY WOUNDS:**

These may be due to:

Crush injury

Tear injury.

Avulsion injury.

Devitalised injury.

Vessel injury or vascular trauma

Irregular wounds may be multiple

Burn wounds

Bony fracture can be present in untidy wounds

Wound dehiscence, severe infections, decreased or delayed wound healing were common here.

II. CLASSIFICATION ACCORDING TO WOUND TYPES:

a. **Clean incised wounds:** These are defined as clean cut edge wounds with linear edge.

b. **Laceration:** These wounds are with loss of tissues or cellular component.

c. **Bruising & contusion like wounds**: These are the wounds where minor soft tissues or cellular injury along with discoloration & formation of haematoma without any break in skin continuity.

d) **Haematomaformation**:

Depending upon sites they can be subcutaneous / intramuscular / subfascial / intra-articular. Some of small haematoma can easily get absorbed. Large haematoma may get infected which forms an abscess. So it must be drained adequately under suitable anaesthesia either general / intra venous regional anaesthesia / local block anaesthesia. Some time haematoma may contain only red coloured plasma like fluid which may be aspirated by using wide bore needle if necessary.

e. **Blunt injury closed wounds**

f. **Puncture and stab wounds.**

g. **Abrasion like wounds**:

These are superficial & it may due to shearing force to the skin where the surface is stripped off from bed. It is healed by primary epithelialisation.

These are the epidermal injury which expose dermis &cutaneous nerves accordingly.

h. **Avulsion injury wounds.**

i. **Crush injury wounds:**

These are the wounds which may be caused by war, road traffic accidents, prolonged application of tourniquet etc. These may lead on to:

Compartment syndrome, muscle ischemia, signs of gangrene, tissue loss and loss of function.

j. **War & gunshot injury wounds.**

k. **Injuries to bones & joints which can be either open / closed**

l. **Injuries to nerves which may be clean incised wound / crush wound.**

m. **Injury to major vessels which can be either arteries or veins**

n. **Injuries to internal organs which can be deep penetrating or non-penetrating like blunt injury type wounds.**

o. **Penetrating wounds:**

They are commonly due to stab injuries in various sites and depth. Most common example is stab injury abdomen and chest. It can be tiny on the body surface but it damages to the deeper internal organs like liver, spleen, major vessels like inferior vena cava, mesenteric vessels or intestines and lungs which can be serious and fatal. Stab injury abdomen patients must be admitted and evaluated by ultrasound or CT (computerised Tomography) scan abdomen.

Closed observation of the patient is very essential while managing these patients often sometimes they need wound exploration under suitable anaesthesia in operation theatre.

III. CLASSIFICATION OF WOUNDS ACCORDING TO THICKNESS :

Superficial wounds:

These may involve only epidermis & papillary dermis.

Partial thickness wounds:

These are the wounds with damage to the dermis with layers behind deepest part of the dermis, hair follicle shafts & sweat glands.

Full thickness wounds:

These are the wounds with extensive loss of skin and subcutaneous tissues.

Deep wounds:

Theseare the wounds which extends deeper, may cross the deep fascia into muscle layers or deeper structures.

Complicated wounds:

Theseare the wounds which is commonly accompanied with vessel or nerve injury.

Penetrating wound:

These are the wounds which penetrates into natural cavities or internal organs.

IV. CLASSIFICATION ACCORDING TO INVOLVEMENT OF BENEATH STRUCTURES:

Simple wounds:

They wounds which involves only one organ or tissue.

Compound wounds:

These wounds which may involve mixed tissues.

V. CLASSIFICATION ACCORDING TO TIME CONSUMED:

Acute wound:

It may defined as duration up to 8 hours of traumatic injury.

Chronic wound:

They are defined as wounds after 8 hours of traumatic injury.

VI. **SURGICAL WOUNDS – CLASSIFICATION:**

a. **Clean wound occurs in:**

Surgery for inguinal hernias

Swelling excisions.

Surgeries done to brain, bone, joints, cardiac, renal transplant surgeries.

Infective rate is less than 1%.

b. **Clean contaminated wound occurs in:**

appendix surgeries

Intestinal surgeries.

Gallbladder, liver &pancreas surgeries.

Infection rate being 9%.

c. **CONTAMINATED WOUND OCCURS IN:**

Acute abdominal surgeries

Traumatic wounds.

Infection rate being 10 to 30%.

d. **DIRTY INFECTED WOUND OCCURS IN:**

Abscess drainage procedure

Pyocele surgeries

Empyema gallbladder surgery

Fecal peritonitis surgeries

Infective rate being 50 to 70%.

WOUND HEALING – TYPES:

HEALING BY PRIMARY INTENTION (FIRST INTENTION):

It may occur in a clean incised wound or surgical incision wound. Wound edges may get approximated with sutures and various materials. Here more epithelial regeneration occurs than fibrosis formation. Here wound healing may be rapid with adequate closure. Scar may be simple linear, smooth, and supple**.**

HEALING BY SECONDARY INTENTION (SECOND INTENTION):

It may occur in a wound with severe soft tissue or epithelial loss which may occur in major traumatic injury, burn wounds & infected wound. Here healing occurs slowly with fibrosis formation. It may further lead into a wide deep scar, often they become hypertrophied & contracted. It can end up in disability. Re-epithelialisation may occur from remaining skin dermal elements or wound edge margins etc.

HEALING BY TERTIARY INTENTION / DELAYED PRIMARY CLOSURE:

After adequate wound debridement & control of local sepsis, wound may be closed with sutures or it may covered using split skin graft. Primary contaminated wounds or mixed tissue wounds heal by tertiary intention / delayed primary closure.

WOUND HEALING – STAGES:

Stage of inflammation process.

Stage of granulation tissue formation &organisation.Because of increased fibroblastic activity.synthesis of collagen & its ground substance occurs here.

Stage of epithelialisation.

Stage of scar formation and resorption.

Stage of maturation.

WOUND HEALING – PHASES:

Phase of inflammation (Lag or Substrate or Exudative Phase):

It occurs soon after the wound healing process. Duration is about 4-6 days. Signs of inflammation are present which are rubor, calor, tumour, dolor and loss of function.

Macrophages secrete fibroblastic growth factor which stimulates the angiogenesis process.

Polymorphonuclear leukocytes (PMN leukocytes) can appear after 48 hours of wound inflammation which may secrete important inflammatory mediators & free radicals. These type of cells can remove the clots, foreign bodies and bacteria from site of wound.

Chemical factors participated in the wound healing process are:

Growth factors which may be platelet derived, epidermal or transforming.

Interleukins involved in wound healing process are:

Tumour necrosis factor and its components.

Prostaglandins and its products.

Collagenase and its components &elastase and its components.

Here haemostasis, coagulation and chemotaxis occurs. Coagulation begins in wound haematoma → formation of platelet fibrin thrombus → release of cytokines, PDGF (platelet derived growth factor), transforming growth factor β (TGF-β), platelet activating factor, fibrin, serotonin. Chemotaxis causes neutrophil migration first, and then activation of macrophages, lymphocytes leading into phagocytosis, wound debridement, matrix activation, angiogenesis. Chemotaxis factors are complement factors, interleukin-1, TNF-α (tumour necrosis factor-α) TGF-β and platelet factor. Activated macrophages produce free radicals and nitric oxide; release cytokine to activate lymphocytes which release interferon and interleukin (called as lymphokines). These actions are reduced in diabetes mellitus, Cushing's syndrome and immunosuppression increasing the rate of sepsis.

Phase of proliferation (Collagen/Fibroblastic Phase):

Collagen and glycosamines are produced by fibroblasts. It begins in 7 days and lasts for 6 weeks. Hydroxyproline and hydroxylysine are synthesised by specific enzymes using iron, alpha ketoglutarate and vitamin C. Tropocollagen is produced which aggregates to form collagen fibrils. 80–90% of their final strength (in postoperative wounds) is achieved in 30 days. Here proliferation of venular endothelial cell with fibroblast at wound margin and bed occurs by the action of cytokines and released growth factors. This angiogenesis and fibroplasia causes formation of granulation tissue which contains fibroblasts, neocapillaries, collagen, fibronectin and hyaluronic acid. Later neutrophils lead into apoptosis and die which are phagocytosed by macrophages; later macrophages go for apoptosis which are cleared by lymphocytes into draining lymph nodes.

Phase of remodelling (Maturation Phase):

It begins at 6 weeks and lasts for 2 years. There is maturation of collagen by cross-linking which is responsible for tensile strength of the scar. Collagen production is not present after 42 days of wound healing.Initially fibrin, fibronectin, proteoglycan deposition occurs; later collagen protein develops to form scar. Normal dermal skin contains 80% type I (20% type III) collagen; granulation tissue contains mainly type III collagen; scar contains both type I and III collagen equally. Basic essential components of collagen are proline and

lysine. Hydroxylation of lysine and later glycosylation of this hydroxylysine decides the collagen molecule type. Hydroxylation of both proline and lysine as essential step needs adequate concentration of vitamin C, iron and α-ketogluteric acid. Collagen deposition in the wound is assessed by quantity of hydroxyproline excreted in urine. There is a balanced activity of collagen production and degradation of collagen (collagenolysis). Collagen is broken down by collagenase and MMPs (matrix metallo proteins). Procollagen through procollagenase → collagen fibril → cross-linking → collagen fiber → deposition.Deposited collagen → through collagenase → degradation and collagenolysis. Scar strength is 3% in 1 week; 20% in 3 weeks; 80% in 12 weeks.

FACTORS WHICH INTERFERES THE WOUND HEALING:

LOCAL FACTORS MAY BE:

Local sepsis.

Presence of dead tissue & foreign body.

Poor perfusion.

Venous or lymphatic stasis.

Tissue tension.

Haematoma formation.

Large area defect or poor apposition of edges.

Recurrent traumatic injury.

Local irradiation.

Specific site of the wound, it may over the joints & back which has poor healing process.

Underlying many diseases like osteomyelitis & malignancy of the wounds.

It may due to mechanism & type of wound which may be incised wound /lacerated wound /crush wound / avulsion wound.

Tissue or cellular hypoxia which may reduce the local macrophage activity & fibroblast Activity.

GENERAL FACTORS MAY BE:

Age factors, obesity & smoking factors.

Under nutrition, deficiency of essential elements like zinc, copper, manganese.

Deficiency of vitamins (Vitamin C & Vitamin A).

Anaemia

Malignancy

Renal failure

Jaundice / hepatic damage

Diabetes mellitus & metabolic diseases

AIDS & immune compromised diseases

Steroids & cytotoxic chemotherapy drugs

Different neurological diseases

ULCER DEFINITION:

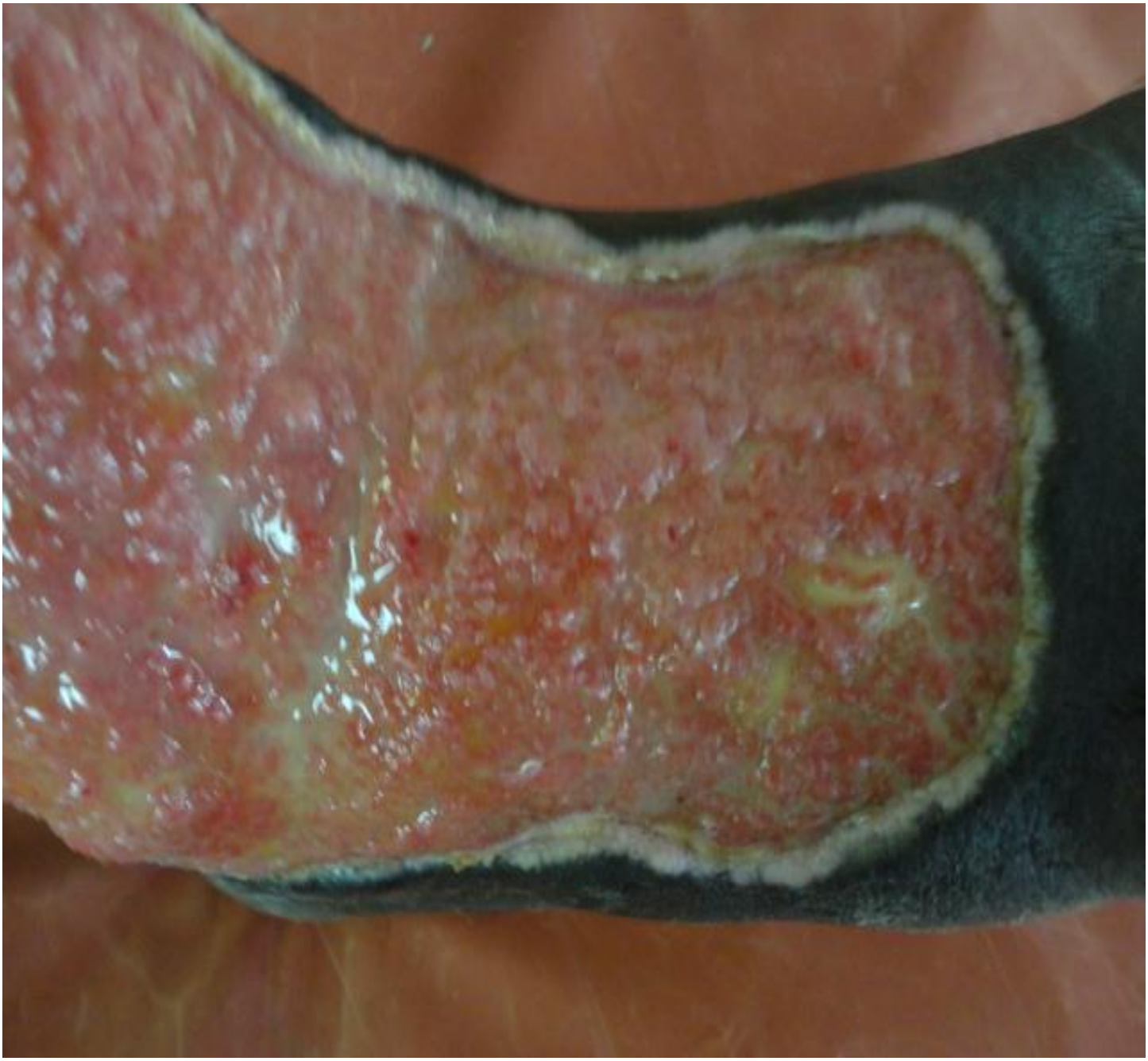
An ulcer is defined as tear or break in the continuity of the covering epithelium, either it may be skin or mucous membrane which may due to molecular cell death.

PARTS:

a. **Margin:** either regular / irregular and it can be rounded / oval.

b. **Edge:** It connects floor to the margin of ulcer.

ULCER



Edges may be:

Sloping:

These type of edges were seen in a healing ulcer. It has 3 parts. Inner part is red because of healthy granulation tissue, middle part is blue in colour due to epithelial proliferation, outer part is whitish in colour because it has scar or fibrous tissue.

Undermined edge:

These type of edges seen in a tuberculous ulcer patients which may advances in deeper plane in the subcutaneous tissue where epidermis proliferates inwards.

Punched out edge:

These type of edges were commonly seen in syphilitic ulcer patients & trophic ulcer patients. It may be due to endarteritis.

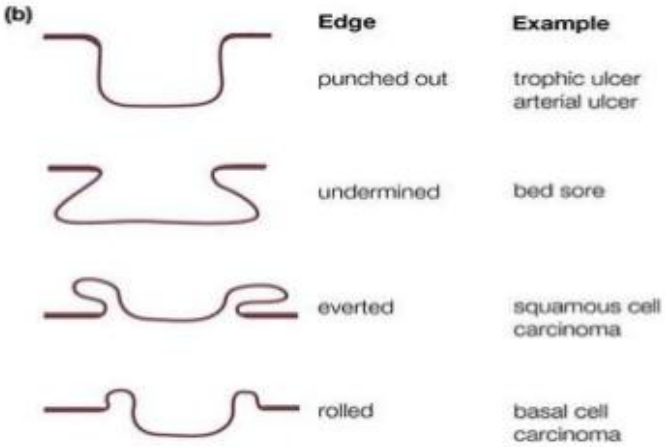
Raised &beaded edge:

These type of edges were seen in rodent ulcer (Basal Cell Carcinoma) which is due to proliferating active epithelial cells.

Everted/ rolled out :

These type of edges seen in a carcinomatous ulcer patients which may due to spilling of the proliferating malignant cells over the normal skin.

The classic appearances of various ulcers are presented



. Infective ulcers due to *Mycobacterium* species, and bed sores, tend to have an undermined edge while a trophic ulcer is punched out and typically round in surface shape. A raised firm ulcer edge may indicate malignancy

c. **Floor:** It is the one which what we were seen.it may contain discharge, granulation tissue or slough over it.

d. **Base**: It is the one which ulcer rests. It can be bone or soft tissue.

CLASSIFICATION I (OR) CLINICAL CLASSIFICATION:

a. **Spreading type ulcer:** Here edge may be inflamed & oedematous

b.**Healing ulcer:** Sloping edge with healthy red granulation tissue with copious amount of serous discharge.

c. **Callous ulcer:** Floor may contain unhealthy granulation tissue with indurated edge or base which lasts for many months to years. Ulcer may not show any type of tendency to heal. It may due to callous formation of the patient.

CLASSIFICATION II (OR) PATHOLOGICAL CLASSIFICATION:

a. **Specific ulcers are:**

Tuberculous ulcers

Syphilitic ulcer

Actinomycosis ulcers

Meleney's type of ulcer

b.**Malignant ulcers may be:**

Carcinomatous type of ulcers

Rodent ulcers

Melanotic type of ulcers.

c. **Nonspecific ulcers are:**

Traumatic injury ulcers: It is mainly due to mechanical, physical, chemical injuries.

Arterial type of ulcers: It can be due to Atherosclerosis or TAO (Thrombo Angitis Obliterans)

Venous type of ulcers: It can be gravitational type of ulcer or post-phlebitic type of ulcer

Trophic ulcer/Pressure sore/bed sores

Infective ulcers: Bacterial infection of an ulcer

Tropical ulcers:

It can occur in tropical countries which may be callous type of ulcer example being Vincent's ulcer.

Ulcers may be due to chilblains & frostbite / cryopathic ulcers.

Martorell's hypertensive ulcers.

Bazin’s ulcers.

Diabetic ulcers.

Ulcers may due to haematological conditions like leukaemia, polycythemia, jaundice, collagen based diseases and also lymphedema.

Cortisol ulcers may be due prolonged use of cortisol / steroid based creams to many dermatological conditions. These ulcers are being callous type of ulcers which may last for longer duration.

WAGNER’S GRADING OF ULCER / CLASSIFICATION OF ULCER:

Grade 0: Pre ulcerative lesion or healed ulcer

Grade 1: Superficial type of ulcer

Grade 2: Ulcer deeper to subcutaneous tissue which expose the soft tissues or bone

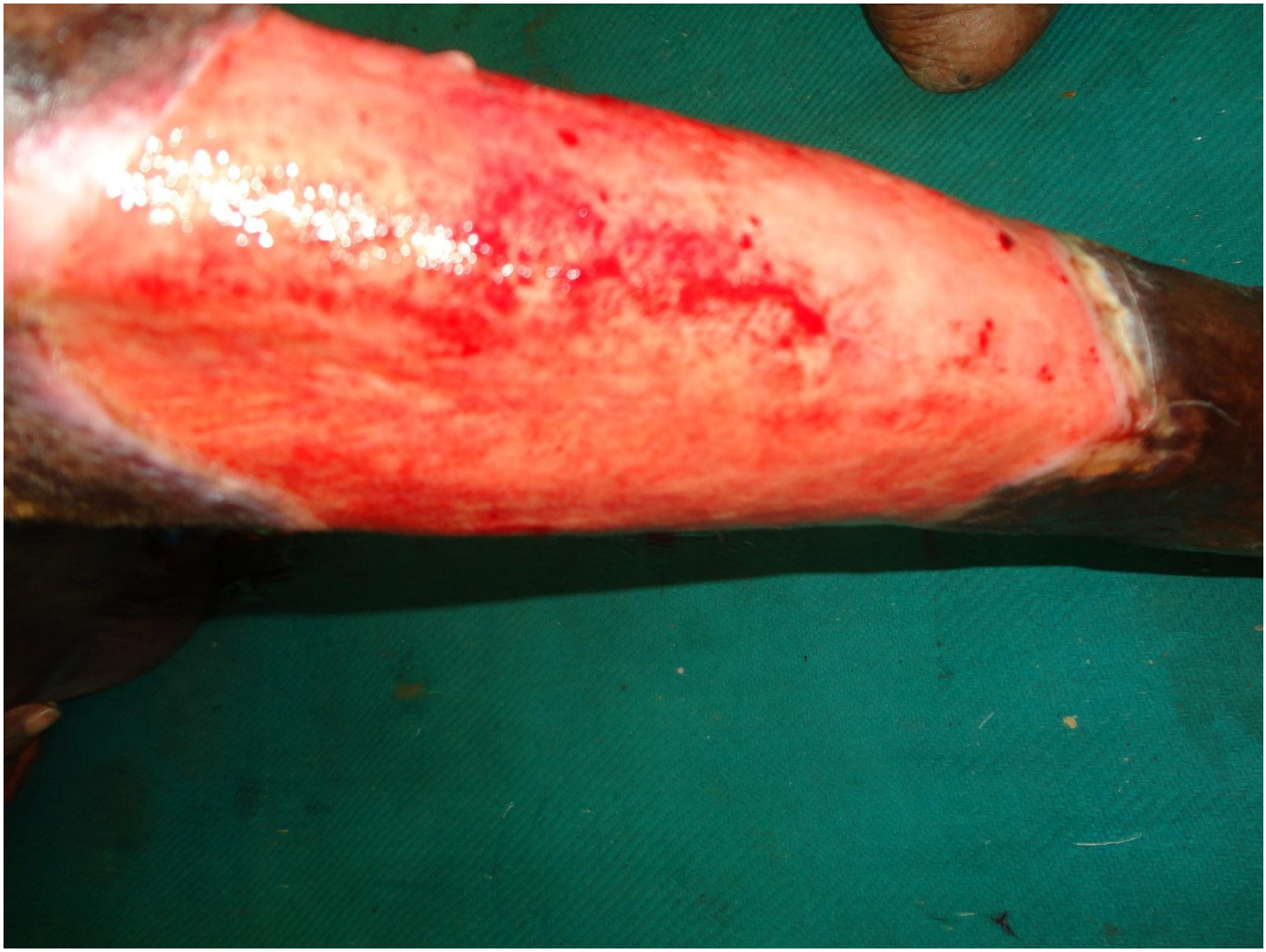
Grade 3: Abscess formation beneath or osteomyelitis of underlying bone

Grade 4: Gangrenous limb or foot

GRANULATION TISSUE FORMATION:

It is the process where proliferation of new capillary buds & fibroblasts formation which may be intermingled with red blood cells & white blood cells with fibrin cover formation over it.

RED HEALTHY GRANULATION TISSUES



TYPES OF GRANULATION TISSUES:

Healthy red granulation tissue:

It may occur in a healing type of ulcer which may be sloping edge. May be bleeding on touch with copious amount serous discharge. There are 5 Ps of the granulation tissues which are pinkish, punctate like haemorrhages, presence of pulse, pain free, pin point head granulation.In the presence of healthy red granulation tissue skin graft taken up well.

Unhealthy pale tissue:

It is associated with purulent discharge where floor is covered only with slough. Its edge was inflamed and oedematous. It may be a spreading type of the ulcer.

Unhealthy, pale, flat tissue:

It may be seen in the chronic non healing ulcer / callous ulcer patients.

Exuberant like tissue:

It can occur in a sinus or ulcer where granulation tissue comes out of the sinus or ulcer bed looks like a proliferating mass. It is mostly associated with a left foreign body in the sinus tract.

Pyogenic granuloma formation:

It may be one of the type of exuberant granulation tissue. Well localised infected granulation tissues which may bleed on touch is characteristic.

DIFFERENT DISCHARGES IN AN ULCER PATIENTS:

a. Serous discharge: It may be seen in healing type of ulcers

b. Purulent discharge: It is seen in infected type of ulcers.

Staphylococci infected ulcers: shows yellow & creamy like discharge

Streptococci infected ulcers: shows bloody & opalescent discharge

Pseudomonas infected ulcers: shows greenish colour discharge because of pseudocyanin

c. Bloody discharge: seen in malignant type of ulcers, red healthy granulation tissue with healing ulcers

d. Seropurulent discharge

e. Serosanguinous discharge: means both serous and bloody discharge

f. Serous with sulphur granule discharge: seen in Actinomycosis ulcrers

g. Yellowish discharge: It may be seen in tuberculous ulcer patients

h. Smell of the discharge.

INVESTIGATIONS FOR AN ULCER PATIENTS:

TC, DC, ESR, Study of the discharge: Culture and sensitivity, grams stain, AFB study, cytology for malignant cells

Edge wedge biopsy: It is very important because edge may contain multiplying cells. Normally we should take at least two biopsies from the ulcers.

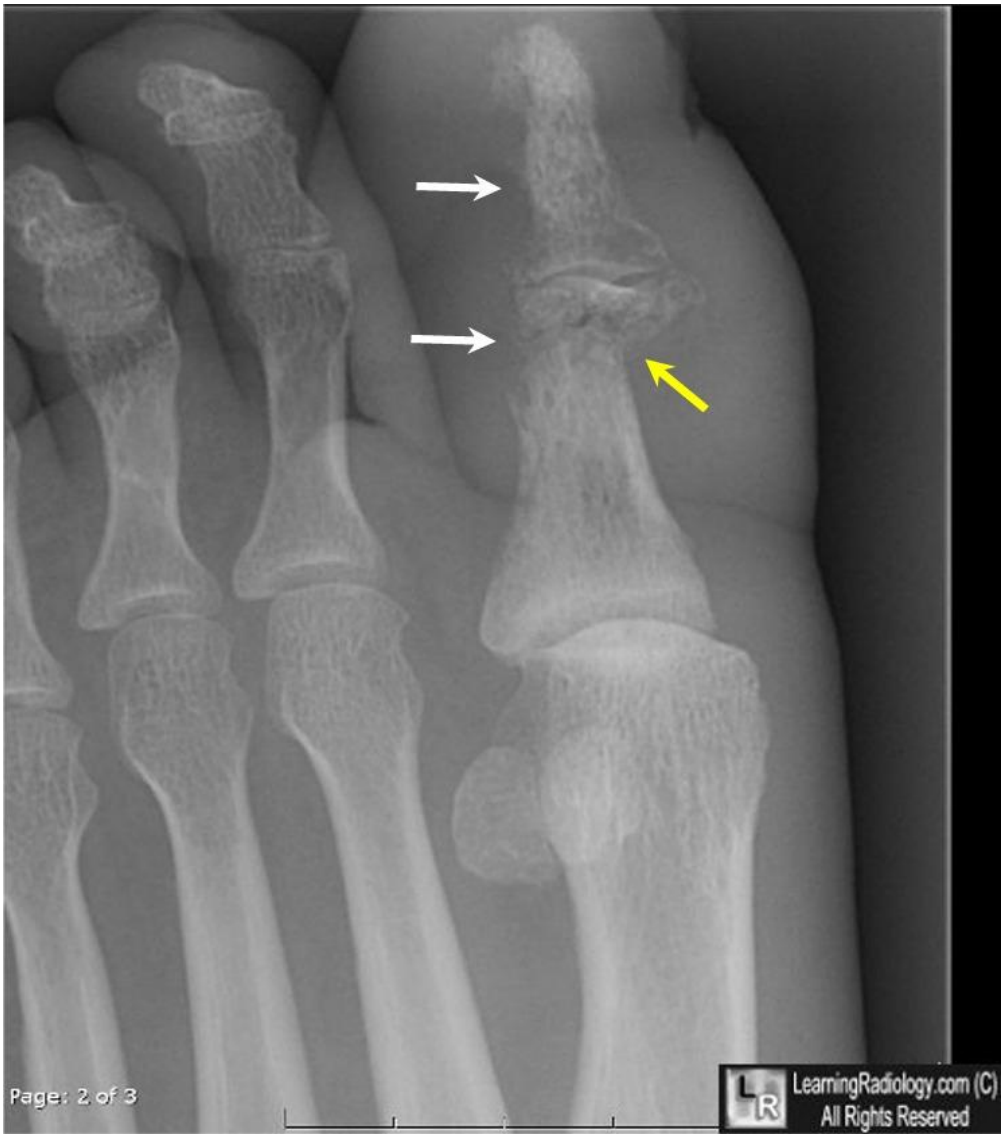
Biopsy from centre of the ulcer most probably inadequate because of the central necrosis.

X-ray of the affected part to look for any periostitis of bone / osteomyelitis of underlying bone.

FNAC (Fine Needle Aspiration Cytology) of the draining lymph nodes if it is palbable.

Chest X-ray PA view, Mantoux test may be required in suspected case of tuberculous ulcer patients.

X RAY FOOT SHOWS OSTEOMYELITIS:



ULCER ASSESSMENT:

Etiology of the wound, which includes diabetic / venous / arterial / infective ulcers.

Assess the clinical class of the ulcers

Wound assessment is very important because of its anatomical location, size & length of the wound, ulcer edge, its mobility & fixity to underlying structures, induration of base, surrounding ulcer area, local vascular supply. Wound perimeter can be used for wound assessment.

Presence of systemic features of patients, to know the regional lymph node status, function of the affected limb, joint active movements, peripheral pulses, position& vibration sensations could be evaluated. Infection severity can be evaluated by culture and sensitivity of wound discharge. Special investigations like edge wedge biopsy of ulcers, X-ray of affected part, blood sugar value of patient, ultrasound arterial / venous Doppler study, CT angiogram.

WOUND DEBRIDEMENT:

It is otherwise known as removal of dead tissue. Small superficial ulcers can be debrided in ward alone. Large ulcers debridement may need wound debridement in operation theatre under suitable anaesthesia. All infected dead, devitalised, necrotic slough tissues were removed. Before debridement, slough

can be separated adequately. Sometimes devitalised tissue itself separates on its own by achieving autolysis. For debridement, collagenase enzyme were adequately used. Hydrotherapy & other dressing methods were nonselective method of debridement of wounds.

DRESSINGS DONE BECAUSE OF:

To keep the ulcer bed moist

To keep the surrounding skin is dry.

To reduce the pain.

To soothen the tissue.

To protect wound from environment.

It is an otherwise called an absorbent for the discharge ulcers.

ULCER DRESSINGS MAY BE:

Cotton like dressing: It is very cheap but it is traumatic

Paraffin like dressing – non sticky

Foam like dressings which may increase the absorbent nature. These may reduce the maceration & then reduce the incidence of dressing like hydrophilic polyurethane foam dressing methods.

Hydrocolloid like dressings may help in removal of slough & autolysis of devitalised tissues.

Transparent film like dressings were advantage of being water proof which may permit oxygen & water vapour particles across & thus prevent the contamination of wounds.

Hydrogel like dressings may be mostly used for clean wounds.

CHRONIC NON HEALING WOUNDS – ETIOLOGY:

Recurrent local sepsis

Traumatic injuries

Absence of rest to the wounds

Poor perfusion

Tissue hypoxia

Wound oedema

Sensation loss

Malignant ulcers

Special causes like tuberculosis

Fibrosis of ulcers

Periostitis of bone or osteomyelitis of the underlying bone

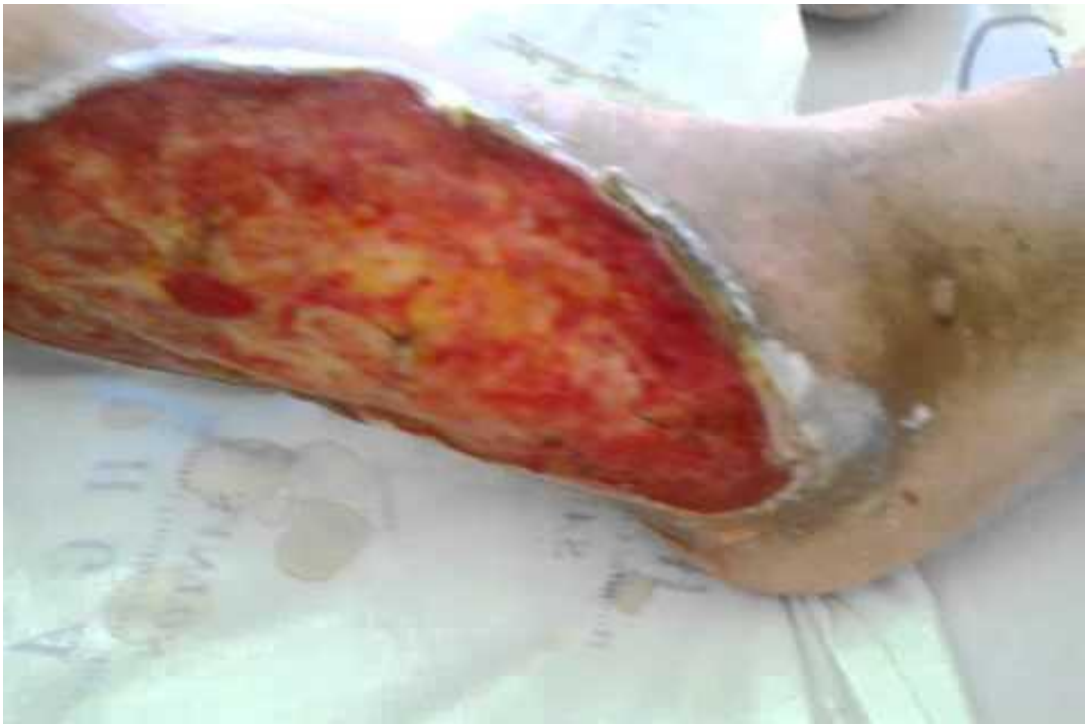
TRAUMATIC ULCERS:

Such ulcer occurs after trauma. It may be mechanical— dental ulcer along the margin of the tongue due to tooth injury; physical like by electrical burn; chemical like by alkali injury. Such ulcer is acute, superficial, painful and tender. Secondary infection or poor blood supply of the area make it chronic and deep. Footballer's ulcer is a traumatic ulcer occurring over the shin of males due to direct knocks on the shin. It is staphylococcal infection with a chronic and deep ulcer. Traumatic ulcers can occur anywhere in the body due to Trauma. Trauma causes infection, necrosis, fasciitis, crush injury, endarteritis of the skin leading into formation of large/deep non healing ulcer.

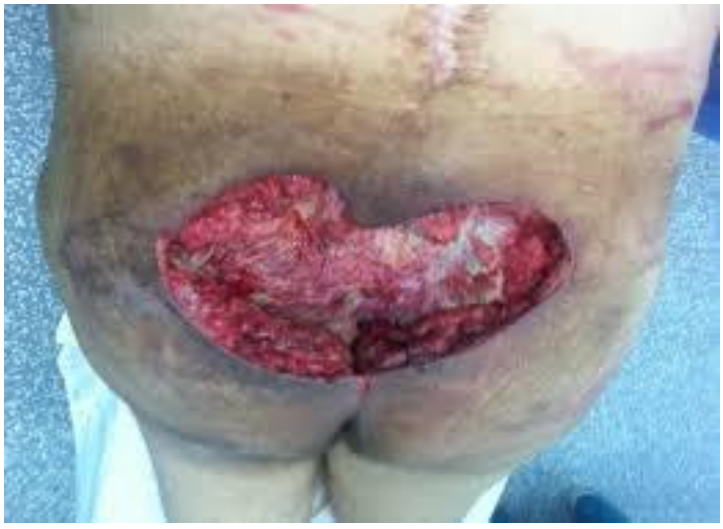
BED SORES (PRESSURE ULCERS):

Pressure sore is tissue necrosis and ulceration due to prolonged pressure. Blood flow to the skin stops once external pressure becomes more than 30 mmHg (more than capillary occlusive pressure) and this causes tissue hypoxia, necrosis and ulceration. It is more prominent between bony prominence and an external surface.

TRAUMATIC ULCER:



PRESSURE SORES:



Pressure sores may due to:

Poor nutrition.

Decreased blood supply.

Neurological diseases.

COMMON SITES :

It may over the ischial tuberosity bone

Over the sacrum.

Over the heel.

Over the heads of metatarsal bones.

Gluteal regions

In the shoulder.

Over the occiput.

FACTORS STIMULATES THE BED SORE ULCERS:

Anaesthetised patient do not have normal stimulus to relieve the pressure sores Necrosis of ulcer may be worsen by nutritional deficiencies

Over the bony prominences and in malnourished patients, inadequate padding is most cause skin soiling, maceration, infection, necrosis caused by urinary incontinence in the paraplegia like patients

CLINICAL FEATURES OF PRESSURE ULCER PATIENTS:

It may occur in 6% of all inpatient hospitalised patients.

Punched out ulcers which are painless

Ulcer may be immobile where base was only bone.

NEUROLOGICAL CAUSES OF PRESSURE SORES:

Diabetic neuropathy, peripheral neuritis, tabesdorsalis, spina bifida, leprosy, spinal injury, paraplegia patients, peripheral nerve injury patients, syringomyelia patients.

STAGING OF PRESSURE ULCER PATIENTS:

Non-blanching like erythema seen in early superficial ulcer patients.

Partial thickness skin loss ulcers seen in late superficial like ulcer patients.

Full thickness skin loss like ulcers which extending to subcutaneous tissue but not enough amount to fascia known as early deep ulcer patients.

Full thickness skin loss like ulcers have fascia & underlying structures like muscle or tendon or bone which was known as late deep ulcers.

FROSTBITE ULCERS:

It may due to because of exposure to cold below the freezing point / cold wind.

Arterial vasospasm, proteins denaturation & cell component destruction may occur here.

It may lead into gangrene formation of the affected part.

Frostbite ulcers are always almost **deep ulcers.**

MARTORELL'S ULCERS:

It is almost always seen in hypertensive patients which often patients with atherosclerosis. It is mostly occur in calf region which may be bilateral & ulcer becomes very painful."

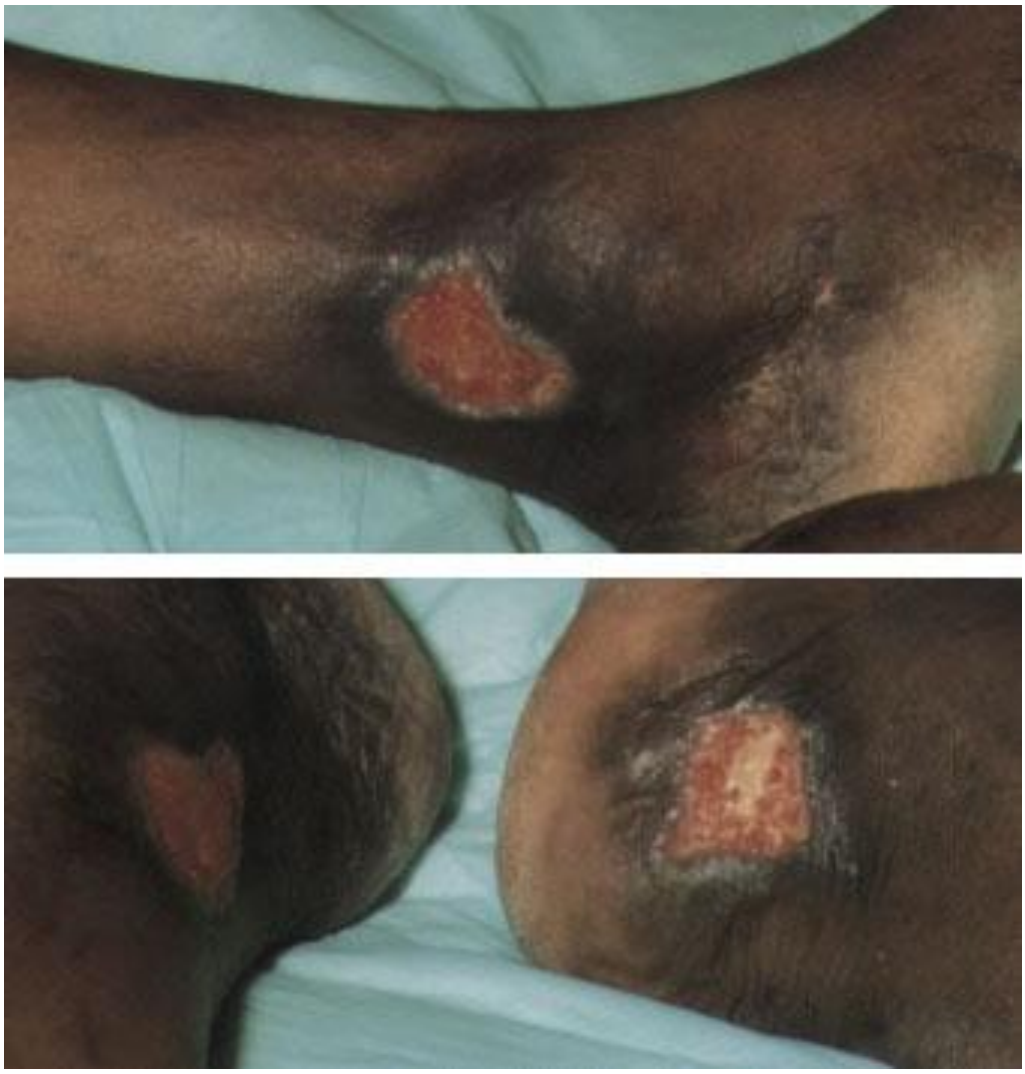
Necrosis of calf skin occurs with sloughing away and formation of deep, punched out ulcers extending into the deep fascia. There is sudden obliteration of the arterioles of the calf skin. All peripheral pulses are present. It takes months to heal.

ARTERIAL / ISCHEMIC ULCER:

These type of ulcers most commonly seen in toes, foot and legs which can occur even in upper limb digits also. It may because of poor vascular supply following blockage in the digital arteries or medium sized arteries.

Atherosclerosis & TAO (Thrombo angiitis obliterans) are most common etiology in the lower limbs. In upper limbs, cervical rib, Raynaud's phenomena & peripheral vasculitis are most common causes. Ulcer following trauma, which becomes non-healing & may lead to spreading with scanty amount granulation tissue formation. Ulcer may become very painful, tender & may even hyperaesthetic. Digits may become gangrenous. Intermittent claudication of limbs, ulcer rest pain are common entity. Signs of ischemia were obviously seen in the surrounding areas. Signs of ischemia are pale, dry mottled skin, brittle nails, patchy lesion in the ulcers & hair loss. Ulcer can be deep which invades the deep fascial plane which further expose the muscle tendons & muscles and underlying bony structures. Devitalised muscle tendons which may look white pale or greenish pus discharge on it.

ARTERIAL ULCERS



CARCINOMATOUS ULCER (OR) EPITHELIOMA (OR) SQUAMOUS CELL CARCINOMA (SCC):

These type of ulcer mostly begins from prickle cell layer of dermis. It begins as a single nodule or ulcer but soon after it forms an ulcerative type of lesion with edges being rolled out or everted. Floor may contain necrotic material, unhealthy pale white tumour granulation tissues & blood. The ulcer may bleed when touch & it is more vascular & friable. At the level of the base and edge, induration may felt. It may be seen as circular shape or irregular shape. Initial mobile ulcers which becomes non mobile when it infiltrates into the deeper structural level. The ulcer is typically foul smelling which may due to slough like necrotic material, local sepsis & polyamides from the proliferating tumour cells. Discrete, hard regional lymph nodes sometimes may be palpable which was initially mobile but soon after becomes fixed. Lymph nodes may fungate accordingly. Ulcer & lymph nodes were painless to start with. It becomes very painful & tender when it infiltrates into the deeper tissues or secondary infection. Systemic spread is very rare. It represents as a loco regional malignant ulcer or disease. Verrucous carcinoma which is commonly exophytic& locally advanced malignant well differentiated squamous cell carcinoma without lymph nodal spread.

MARJOLIN'S ULCERS:

It is slow growing locally malignant lesion which is defined as well differentiated squamous cell carcinoma occurs in an unstable scar for prolonged duration.It is mostly seen in chronic type of venous ulcer scar which may be seen in burns scar & scar of previous snake venom bite. Such lesion may be ulcerative or proliferative.

Edges can be everted or may not be everted. The ulcer may have no pain because the scar may not have nerve fibres. It may not spread into lymphatic system because the scar does not have lymphatic system. At the level of the base and edge, induration may felt. The ulcer is often having marked fibrosis also.

RODENT ULCER:

It is one of the form of basal cell carcinoma which is ulcerative & it is commonly seen in face.

These type of ulcers may have central dry scab with peripheral active & beaded edge. Here floor may be pigmented. It erodes into deeper structures like soft tissues, cartilages & bones. So it is otherwise called as rodent ulcer. Because of large tumour cells, lymphatic system may be blocked so that it may not spread into regional lymph nodes. Blood spread is always almost absent

here. It is locally advanced malignant ulcer. It is seen mostly in face, also may present rarely in tibia, external genitalia, mucocutaneous junction. Ulcer does not occur in the mucosa.

MELANOTIC ULCERS:

It is one of the form of melanoma which is ulcerative which may occur in the skin as a pre-existing mole. These ulcer may be pigmented with a halo around ulcer. It is very rapidly growing ulcer which may have satellite nodules & 'in-transit' like ulcer lesions. It is the most aggressive skin tumour which may arise from melanocyte cells. Pigmented ulcers which may spread rapidly to the regional lymph nodes. Haematogenous spread to lungs, brain, liver and bones are very common entity. It may be seen in the mucosa & genitalia and also seen in eye. It is one the systemic malignant disease.

DIABETIC ULCERS:

ETIOLOGY:

Hyperglycaemia in the tissues can stimulates infection

Diabetic vascular microangiopathy can affect the cutaneous & systemic microcirculation.

Increased level glycosylated haemoglobin (HbA1c) which may decrease the oxygen curve.

Increased level glycosylated tissue protein which may decrease the oxygen utilization. Diabetic neuropathy which involves all sensory system, motor system & autonomous components etc. Associated patients with atherosclerosis.

SITES:

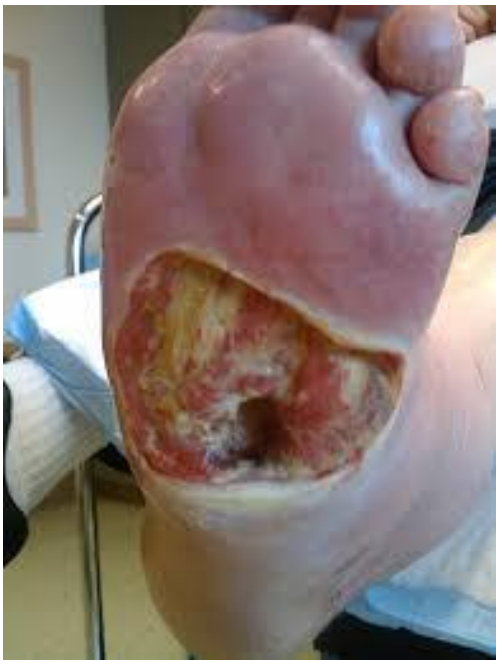
Plantar aspect of foot is the most common site of diabetic ulcer.

Lower limbs - legs

Upper limbs, back, scrotal area, perineum.

Signs of ischemia.

DIABETIC ULCERS:



INVESTIGATIONS FOR ULCER:

Both random blood sugar & fasting blood sugar.

Urinary ketone bodies estimation.

Pus discharge from ulcer for culture & sensitivity.

X ray of the affected part to rule out underlying osteomyelitis.

Arterial Doppler study of the limbs may be needed.

Glycosylated haemoglobin (HbA1c) estimation.

PROBLEMS WITH DIABETIC ULCERS:

Diabetic neuropathy in the foot region which may result into clawing of the toes & hammer toe can be due to intrinsic muscle weakness. Multiple deep abscesses & underlying osteomyelitis of inner bones are usual. Decreased leukocyte function, resistant to infection and spreading type of cellulitis. Arterial insufficiency of the affected area. Septicaemia, worst form is diabetic ketoacidosis. Associated other cardiac diseases like ischaemic heart disease, coronary heart disease

TREATMENT:

Diabetic control by insulin injection.

Antibiotics according to culture & sensitivity.

Nutritional supports & supplements.

Regular cleansing of wounds, adequate through wound debridement, daily dressings.

Once ulcer become granulates, it may be covered with skin graft or skin flap.

May end up in Toe/foot/leg amputation.

Microcellular rubber (MCR) shoes which prevent minor injuries.

MELENEY'S ULCER (OR) POSTOPERATIVE SYNERGISTIC GANGRENE:

These type of ulcer most commonly seen in the postoperative patients wounds either in abdomen or chest wall which may like empyema drainage or after surgery for peritonitis patients. Acute rapidly spreading type of ulcer which may invade deep into subcutaneous tissues.

AETIOLOGY:

It is most commonly seen in older age group & immune compromised patients & after surgery for such infected cases. It may be caused by microaerophillic streptococci & Staphylococcus aureus, anaerobes etc.

SITES:

It is most commonly seen in abdomen & thorax region. It may start from wound margin & may spread rapidly. Other areas of the skin may be affected. Severe form of infection may lead into endarteritis of the skin leading to further ulcer & destruction.

CLINICAL FEATURES:

Toxaemic features.

Spreading type of tender ulcer with pus discharge.

Abundant granulation tissue with both purple zones & red zones.

TUBERCULOUS ULCER:

Mycobacterium tuberculosis is the causative agent. It is usually presents as cold abscess later forming ulcer in the chest wall, neck, axilla and groin. It can also be primary tuberculosis of the skin (commonly in face). Ulcer can be single or multiple, oval or rounded which presents with undermined edge, mostly painful and tender with caseating material on the floor. Ulcer is usually superficial, matted, firm, non-tender regional lymph nodes may be present.

TROPICAL ULCER:

It is common in monsoon hit humid areas with repeated outbreaks but can also occur in subtropics. Trauma or insect bite can lead onto infection exclusively in the lower part of the lower limb. It is an acute ulcer of the skin observed in tropical regions. It is increased in people with lower socioeconomic group, anaemia, and malnutrition and vitamin deficiency. It is generally caused by Fusobacterium fusiformis (Vincent's organisms) and Borrelia vincentii.

Pustule which can burst in three days, causing a spreading ulcer with an undermined edge which I painful, brownish floor and serosanguineous discharge can be present. Spreading stops within few weeks, but ulcer can persist for many months to years. Eventually a chronic, large non healing ulcer forms with pain, and serosanguineous discharge can occur, during healing it causes a slight pigmented, parchment scar. Squamous cell carcinoma can develop rarely in it.

VENOUS ULCER (GRAVITATIONAL ULCER):

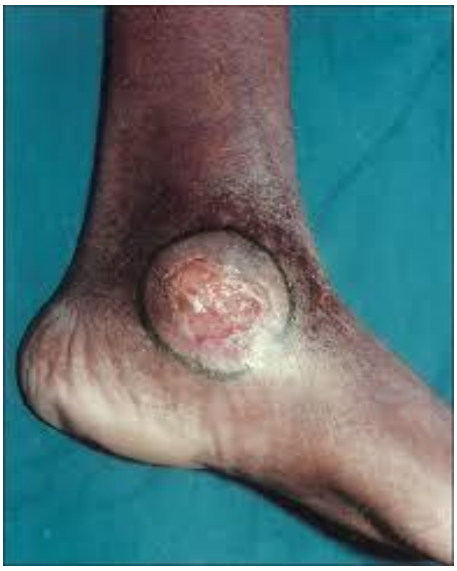
It is situated around the ankle (gaiter's zone) due to chronic ambulatory venous hypertension caused by varicose veins (long saphenous vein /short saphenous vein/ perforators) or post phlebitic limb. Post-phlebitic limb is

characterised by veins which is partially recanalised following deep venous thrombosis leads to increased venous pressure by ankle perforators.

Varicose veins are common in females. About 50% of venous ulcer is due to varicose veins; 50% due to post-phlebitic limb (previous DVT). Pain, discomfort, pigmentation, dermatitis, lipodermatosclerosis, ulceration, periostitis, ankle joint ankylosis, talipes equinovarus deformity and Marjolin's ulcer are the outcome of varicose veins and later of venous ulcer.

Ulcer is usually painful but may become chronic was painless. Ulcer is usually vertically oval; generally located on the medial aspect; sometimes on lateral side; usually on both sides of the ankle; but never above the middle third of leg. Floor is often pale or without granulation tissue. Induration and tenderness can be seen at ulcer base. Inguinal lymph nodes (vertical group) are frequently enlarged. Ulcer usually attains very large size which is mostly non-healing, and callous. Ulcer heals on rest and offloading treatment; but reforms again after sometime. Scarring can occur due to repeated healing and ulcer formation. This unstable scar of long duration may progress into squamous cell carcinoma it is called as Marjolin's ulcer.

VENOUS ULCERS:



SYPHILITIC ULCER:

Nowadays it becomes rare entity. It is caused by Treponema pallidum. It is sexually transmitted disease. Various clinical lesions are observed in many stages of syphilis. John Hunter inoculated himself with syphilis organism to study the clinical features and effects. After 24 years of inoculation, he died from rupture of syphilitic aortic aneurysm at the age of 65. Genital chancre is painless, button like, indurated, does not bleeds on touch; usually present in corona or frenum of penis, often on lips, breasts and anal region; appears four weeks after initial infection **first stage** of the disease (**primary syphilis**). Groin lymph nodes may get enlarged which can be painless, firm, discrete, along with genital chancre. No suppuration will occur. Extra genital chancres in lips and breasts show corresponding enlarged nodes which are inflamed, sometimes painful and also may usually be matted.

During **second stage** (termed as **secondary syphilis**) mucous involvement with white, thickened patches present commonly in the mouth. There it presets as raised, flat, hypertrophied, and warty epithelial patches at mucocutaneous junctions termed as condylomalata. Generalised, discrete, painless, hard lymph nodes are usually palpable, epi-trochlear and suboccipital nodes are pathognomonic. Ocular involvement with iritis can occur. Musculo skeletal manifestations include arthritis, other systems are also involved like hepatitis, meningitis, syphilitic ostetis with 'ivory' sequestrum, coppery red skin rash, moth-eaten alopecia can occur in second syphilis.

In tertiary/late stage syphilis gummatous ulcer develops. Deep, punched out, painless, lesion with wash leather slough present in the floor, with 'silvery tissue paper' like scar and usually present over the subcutaneous bones like tibia, sternum, skull, palate or other area. It can also present over the tongue, scrotum. It is a delayed type hypersensitivity reaction with endarteritis obliterans and vasculitis. Perforation of nasal septum/palate can be seen. Clutton's joint and Sabre tibia are usually seen. Lymph nodes are not affected in a case of tertiary syphilis. Neurosyphilis (tabes dorsalis), aneurysm of arch of aorta are other characters of tertiary syphilis.

Tabes dorsalis clinically manifesting as generalized paralysis of insane is known as **late tertiary or quaternary** syphilis. Long asymptomatic period

mostly from secondary to tertiary is called as **latent syphilis**. Secondary syphilitic stage shows humpty number of circulating

Treponema spirochaetes in blood where as in tertiary stage treponemes are scarce or absent.

COLLAGEN:

Collagen is the most available protein in the human body and is a major building block of ECM. It has three polypeptide chains that are enriched in hydroxyproline amino acids and are rolled together into a triple-helical structure. More than twenty different collagen types have been classified in humans; main types are type I, II and III and they contribute to 80% of the body's collagen. Type I and III are vital for wound healing.

COLLAGEN PARTICLES:

ROLE OF COLLAGEN IN WOUND HEALING:

In healing wound a series of events happens that includes platelet accumulation, cell contraction , inflammation, fibroblast proliferation, , angiogenesis and re-epithelialisation leading to scar formation and wound remodelling. Collagen is very important in wound healing due to its chemotactic role. It attracts cells such as fibroblasts and keratinocytes to wound site. This helps in chemical debridement, angiogenesis and re-epithelialisation. A chronic wound is usually stopped at one of these healing phases. This usually occurs during the inflammatory phase and is linked to increased levels of matrix metalloproteinases (MMPs) in the wound site. In normal wound healing, proteases are attracted to the wound site during the inflammatory phase and plays an important role in bringing down unhealthy ECM for the new tissue formation. However, when MMPs are present at elevated levels for a long period of time, this lead to destruction of healthy ECM, which results in delayed wound healing and an increase in wound size. When the excess MMPs is not countered by normal physiological processes, alternative methods are needed to reduce protease level in wound. This explains the role for dressings containing collagen in the management of chronic wounds.

BIOFIL:

COLLAGEN IN WOUND HEALING:

Stops Bleeding

Helps in wound debridement by attracting monocytes

Provides a Matrix for Tissue and Vascular Growth

It may attack fibroblast cells which may directly involves the cellular migration.

Collagen which binds with fibronectin which may stimulate the cell adhesion

Improves the growth, differentiation &migration of the Keratinocyte cells.

Integrity of the tissues can be increased by organized fibres. Collagen may stimulate the deposition of oriented & organized fibres

SALIENT FEATURES:

Possess good absorbing characteristics

It may derive from fish origin

It may be non-toxic, non-antigenic, non-immunologic & non pyrogenic

INDICATIONS:

Non-healing ulcers

Traumatic wound

Diabetic foot ulcers

Surgical wounds

Pressure ulcers

Tunneled and undermined wounds

Infected and non-infected wounds

COLLAGEN DRESSINGS:

Native intact collagen provides a substrate for new tissue growth or natural scaffold. Dressings with collagen are thought to provide the wound with an alternative collagen source that can be degraded by the high levels of MMPs, leaving the endogenous collagen to carry on normal wound healing. There are many number of collagen dressings available using a variety of carriers and combining agents such as gels, polymers ,pastes and oxidised regenerated cellulose. The collagen contained in these materials also varies in type, source. Some dressings contain native (type I) collagen in which the triple helix formation is present; others contain denatured or reconstituted collagen, which is gelatin. Collagen dressings contain collagen obtained from bovine and porcine sources. Though these collagens are purified, there remains a concern regarding the prion diseases. There also have been issues regarding the usage of porcine collagens into scar tissue, due to some cultural / religious issues. Human-derived collagens are linked with minimal immunological concerns, but they tend to be more expensive than animal-derived collagens. Collagen derived from fish has been proposed as a cost-effective source of collagen for use in dressings in recent days.

COLLAGEN DRESSINGS:

COLLAGEN DRESSINGS ARE AVAILABLE IN:

- Powders.
- Amorphous gels / pastes.
- Gel-impregnated dressings.
- With and without adhesive borders.
- Standard-size wound dressing pads.
- Ropes for undermining or cavity wound fill."

Collagen is often used along with other products to give additional effects such as the expansion of enzyme removal to elastase (ORC-oxidized regenerated cellulose) or gelling (alginate). Some products can promote native long-chain collagen as superior for scaffolding effect and activation of growth factors, while others promote denatured particles for their ability to provide more active binding sites that rapidly interact and remove MMPs and increased levels of amino acids for use in building new collagen biomers. Newer class advice the use of hydrolyzed collagen which are ready to use bioactive and are easily taken up by the matrix of the wound. Collagen dressings if used, may be tried for fast initiating the healing of wounds that are stopped in inflammatory phase, they may be helpful in reducing inflammation as well as pain. They can attract and activate fibroblasts, it may provide a scaffolding effect.

ACTIVATED, NATIVE, PROCESSED AND DENATURED COLLAGENS:

Native collagen materials present a more natural 3-D structure that may provide a more natural environment for fibroblasts and better targets for MMPs. They also bind better to elastin and may aid in conserving elastin levels. Processed or denatured collagen may provide a more readily available source of amino acids necessary for tis-sue reconstruction and a higher number of exposed active sites to divert MMPs from digesting newly formed tissue.

HOW FREQUENTLY SHOULD DRESSINGS BE CHANGED?

Wet wounds need dry dressing and dry wounds need wet dressing, initial dressing changes two to three times per week, depending on the exudate level. Remaining particles in the wound should be washed off. Excessive granulation can occur with collagen dressings. If present, collagen product can no longer be used and the over granulation tissue treated using a 1% hydrocortisone cream for two weeks.

APPLICATION METHODOLOGY:

Cleanse the ulcer bed with use of normal saline without causing trauma & can adjust the irrigation pressure to take out the infected slough & can apply topical medication if needs**.**

Sprinkle collagen particle sufficiently to cover the wound surface. In tunneled & undermined wounds, collagen particles may be converted into a paste like material or a soluble solution with use of normal saline to aware that all collagen particlesare entered into wound cavity**.**

Cover the wound with an absorbent dressing of choice**.**

The frequency of application of collagen particles after cleaning the ulcer with use of normal saline solution can be initially on a daily basis and subsequently reduced to once in 2-3 days according to the condition / Wound stage can be judged by whom the treating surgeon until the wound is healed spontaneously in the case of shallow wounds.

In the case of deep wounds, collagen particles helps to prepare the wound bed with healthy granulation for subsequent closure with graft / flap cover enabling permanent closure. Treat with topical / systemic medication if necessary.

In the study the variables considered were: decrease in wound size, number of debridements required, duration of healing. A comparison was made with that of betadine dressing. Follow up was done for a period of 6 months.

PRECAUTIONS & WARNINGS:

Wounds may appear larger during the first several days of treatment due to the reduction of edema.

Do not pack the wound tightly with collagen particles.

Always maintain moist wound environment.

Give enough irrigation to the wound with normal saline during decreasing changes to prevent damage to the wound bed, which will cause trauma and delay the healing process.

An increase in drainage may be seen in the first several days of treatment. This is a common wound healing response to the initial use of collagen based wound management products.

Collagen particles are made from Fish collagen and hence no allergic reaction has been reported

STORAGE:

Collagen particles can be stored in a dry place temperature between 5° to 25ºC. It is sterilized by gamma sterilized which may have shelf life of three years.

MATERIALS & METHODS:

This study was a non-randomized and prospective comparative study between use of collagen particles and betadine dressing group. It included 104 patients with 52 in each group, 52 patients were subjected to collagen particles dressing and 52 patients to betadine dressing. Comparison between these two groups was done with respect to parameters of wound area, number of debridements,

number of dressings done and mode of healing. Institutional Ethical committee clearance was taken before starting the study and also consent from all participants. The study was conducted from September 2014 to august 2016, in Thanjavur Medical College and Hospital, Thanjavur, Tamilnadu, India.

INCLUSION CRITERIA:

All non-healing ulcers were included like diabetic ulcers, venous ulcers, traumatic ulcers, pressure sores, amputation stump ulcers at least 6 weeks of duration

An area of at least $1cm^2$

EXCLUSION CRITERIA

1. Ulcers having exposed bone without granulation tissue.
2. Conditions which interferes the wound healing process are malignancy, connective tissues disorders, immune system disease.
3. Current medication with dialysis patients, patients on steroids, immunosuppressive drugs, radiation & chemotherapy.
4. Any known allergy to any of the dressing materials.
5. Patients who do not completed 12 weeks follow up and default.

LIMITATIONS OF THE STUDY:

The present study had the following limitations. First is that it was not a randomized study, so similarity among type of ulcers and size of ulcer was not maintained. Second, sample size was very less where we included only 52 patients in each group. Third is the cost factor, as collagen dressing is not very cost effective.

LIMITATIONS:

This study was conducted only for 12 weeks.

The wound was studied only in two dimensions.

Observer and patient were not blinded increasing the risk of bias.

Wound volume measurement rather than area would have been a more accurate approach of judging results.

The study, in spite of its shortcomings, does indicate that topical application collagen particles is more effective than conventional dressing therapy in healing a chronic non healing ulcers and that it has the potential to be a useful and safe adjunct to wound healing.

OBSERVATIONS AND RESULTS:

TABLE 1: AGE

Particulars	**No of respondents (n=104)**	**Percentage (100%)**
Below 30yrs	7	6.7
31 to 40yrs	13	12.5
41 to 50yrs	27	26.0
51 to 60yrs	21	20.2
61 to 70yrs	20	19.2
71yrs & above	16	15.4

Out of 104 patients in this study, 7 patients were below 30 years, 13 patients were age between 31 to 40 years, 27 patients were age between 41 to 50 years, 21 patients were age between 51 to 60 years, 20 patients were age between 61 to 70 years, 16 patients were age above 71 years.

AGE DISTRIBUTION

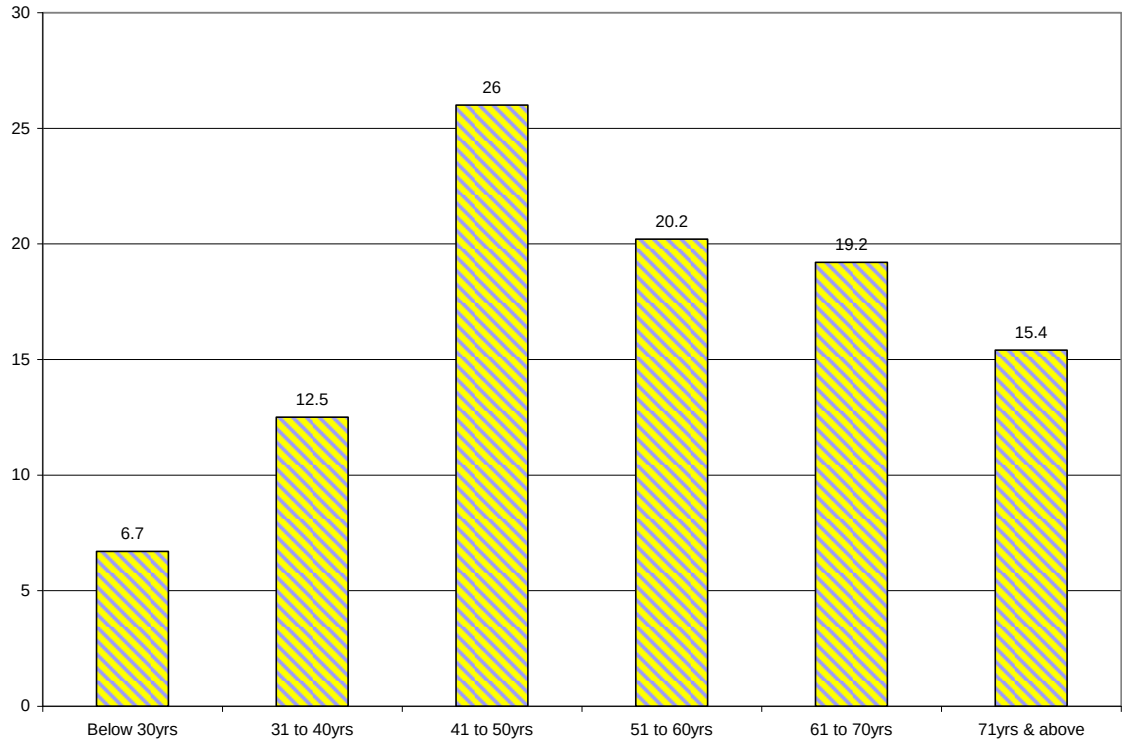


TABLE 2: SEX DISTRIBUTION

Particulars	No of respondents (n=104)	Percentage (100%)
Male	**87**	**83.7**
Female	**17**	**16.3**

out of 104 patients in this study , about 87 patients are male patients, remaining 17 patients are female patients.

SEX DISTRIBUTION:

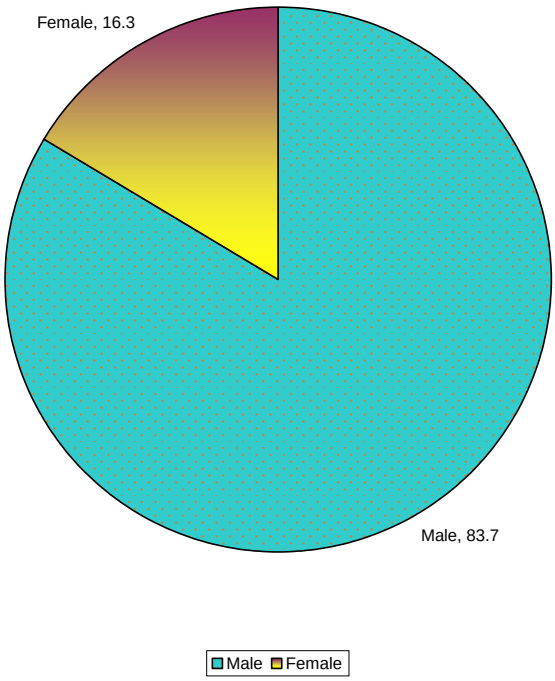


TABLE 3: ULCER

Particulars	No.of respondents (n=104)	Percentage (100%)
AU	14	13.5
DU	62	59.6
PU	8	7.7
TU	7	6.7
VU	13	12.5

Out of 104 patients in this study, 62 patients belonging to diabetic ulcer patients, percentage being 59.6%, 14 patients were arterial ulcer patients accounts for 13.5%, 13 patients were venous ulcer patients accounts for 12.5%, 8 patients were pressure ulcer patients accounts for 7.7%, 7 patients were traumatic ulcer patients accounts for 6.7%.

TABLE 4: ULCER: CHI-SQUARE TEST :

Ulcer	CP		CM		Total		Statistical inference
	n	%	n	%	n	%	
AU	4	7.7%	10	19.2%	14	13.5%	X^2=4.165 Df=4 0.384>0.05 Not Significant
DU	33	63.5%	29	55.8%	62	59.6%	
PU	3	5.8%	5	9.6%	8	7.7%	
TU	4	7.7%	3	5.8%	7	6.7%	
VU	8	15.4%	5	9.6%	13	12.5%	
Total	52	100.0%	52	100.0%	104	100.0%	

ULCER DISTRIBUTION:

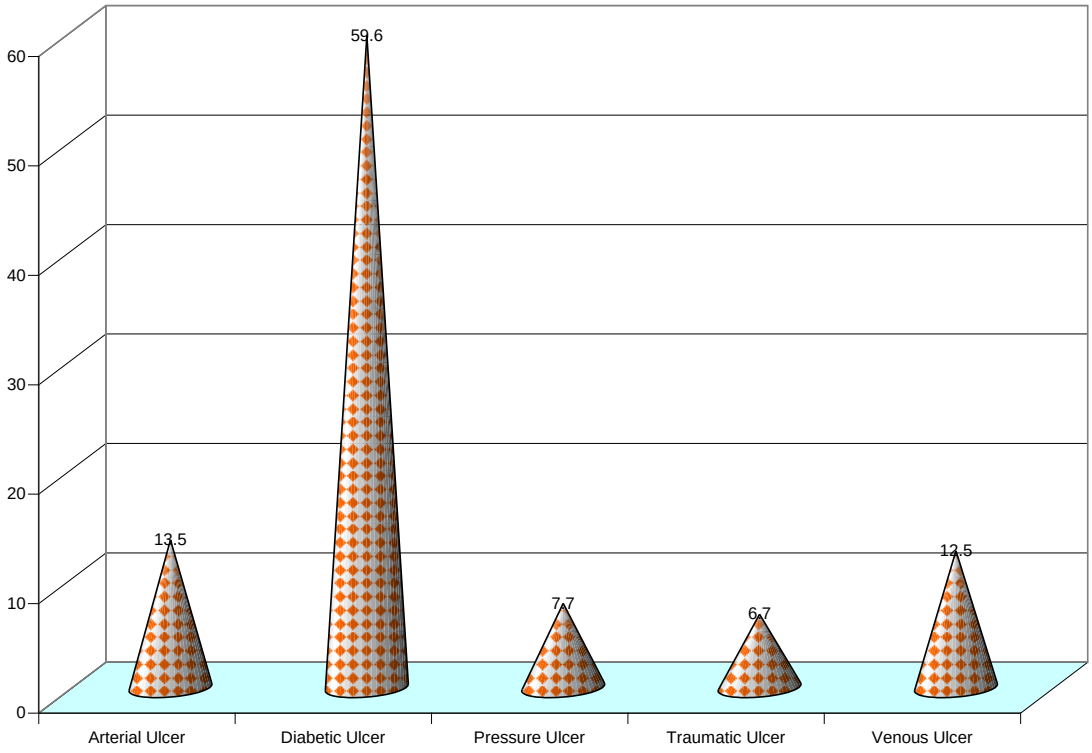


TABLE 5: HEALING

Particulars	No.of respondents (n=104)	Percentage (100%)
No	**34**	**32.7**
Yes	**70**	**67.3**

Out of 104 patients in this study, 70 patients were healed both in study group and control group percentage being 67.3%**.**

HEALING DISTRIBUTION:

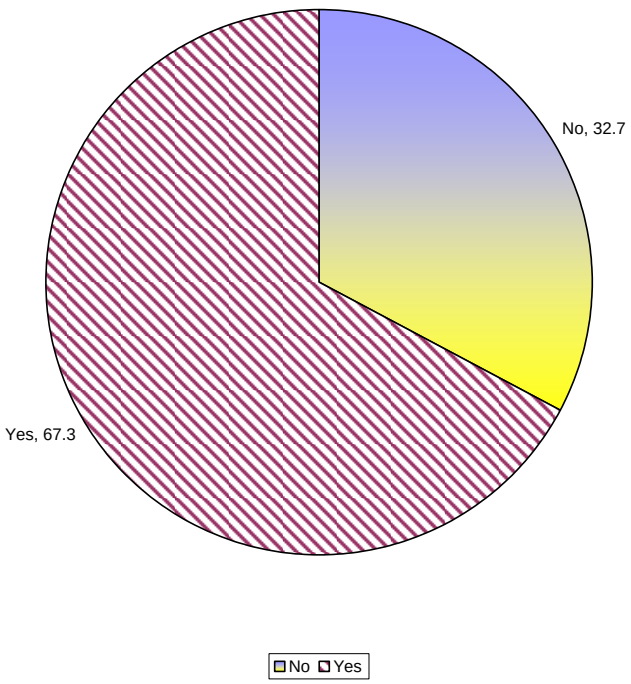
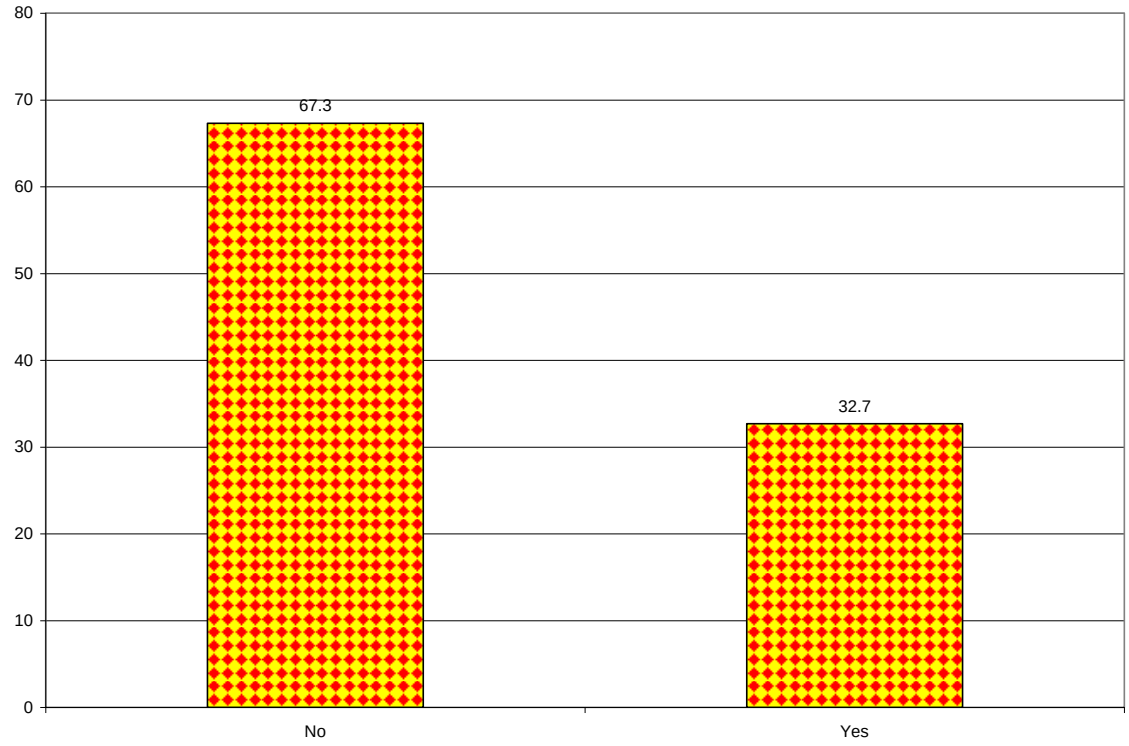


TABLE 6: NON HEALING

Particulars	No.of respondents (n=104)	Percentage (100%)
No	70	67.3
Yes	34	32.7

Out of 104 patients in this study, 34 patients were non healers in both study group and control group percentage being 32.7% for a duration of 12 weeks.

NON HEALING DISTRIBUTION:



AMPUTATION DISTRIBUTION:

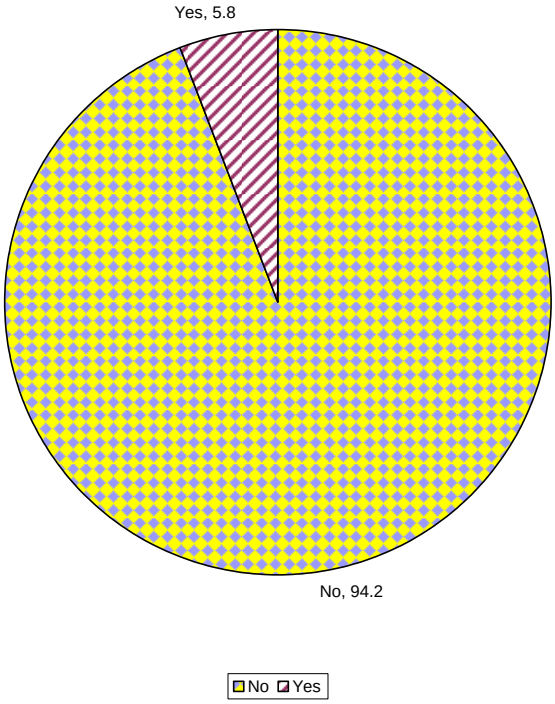


TABLE 8: CHI-SQUARE TEST:HEALING

Healing	CP		CM		Total		Statistical inference
	n	%	n	%	n	%	
No	4	7.7%	30	57.7%	34	32.7%	X^2=29.539 Df=1
Yes	48	92.3%	22	42.3%	70	67.3%	0.000<0.05
Total	52	100.0%	52	100.0%	104	100.0%	Significant

CHI- SQUARE TEST: HEALING DISTRIBUTION

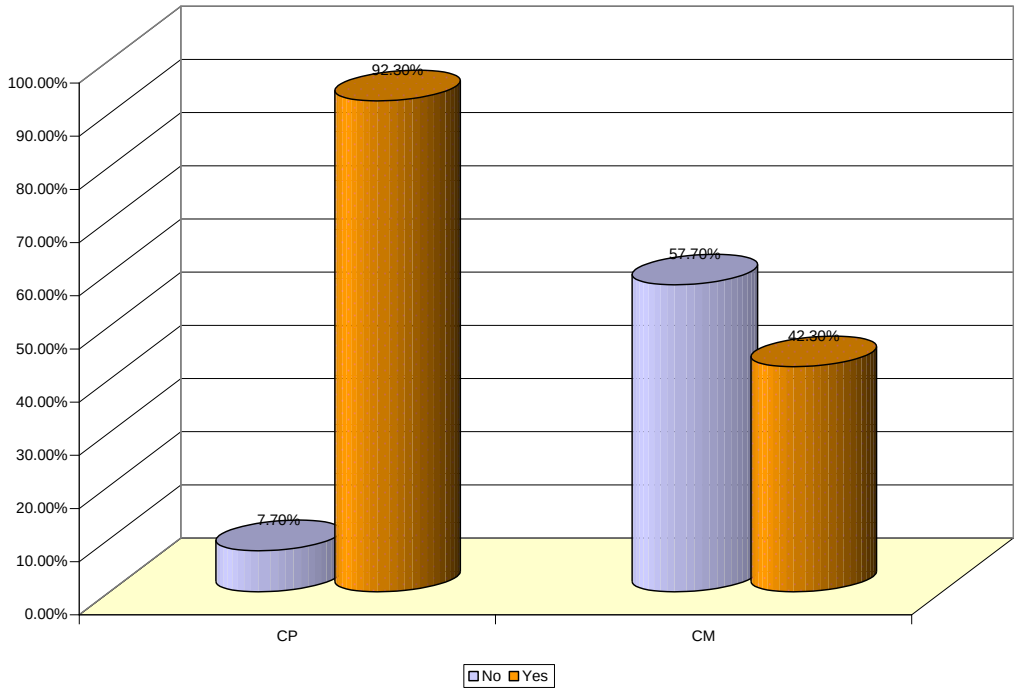


TABLE 9: CHI-SQUARE TEST:NON HEALING

Non Healing	CP		CM		Total		Statistical inference
	N	%	n	%	n	%	
No	48	92.3%	22	42.3%	70	67.3%	X^2=29.539 Df=1
Yes	4	7.7%	30	57.7%	34	32.7%	0.000<0.05
Total	52	100.0%	52	100.0%	104	100.0%	Significant

CHI- SQUARE TEST: NON HEALING

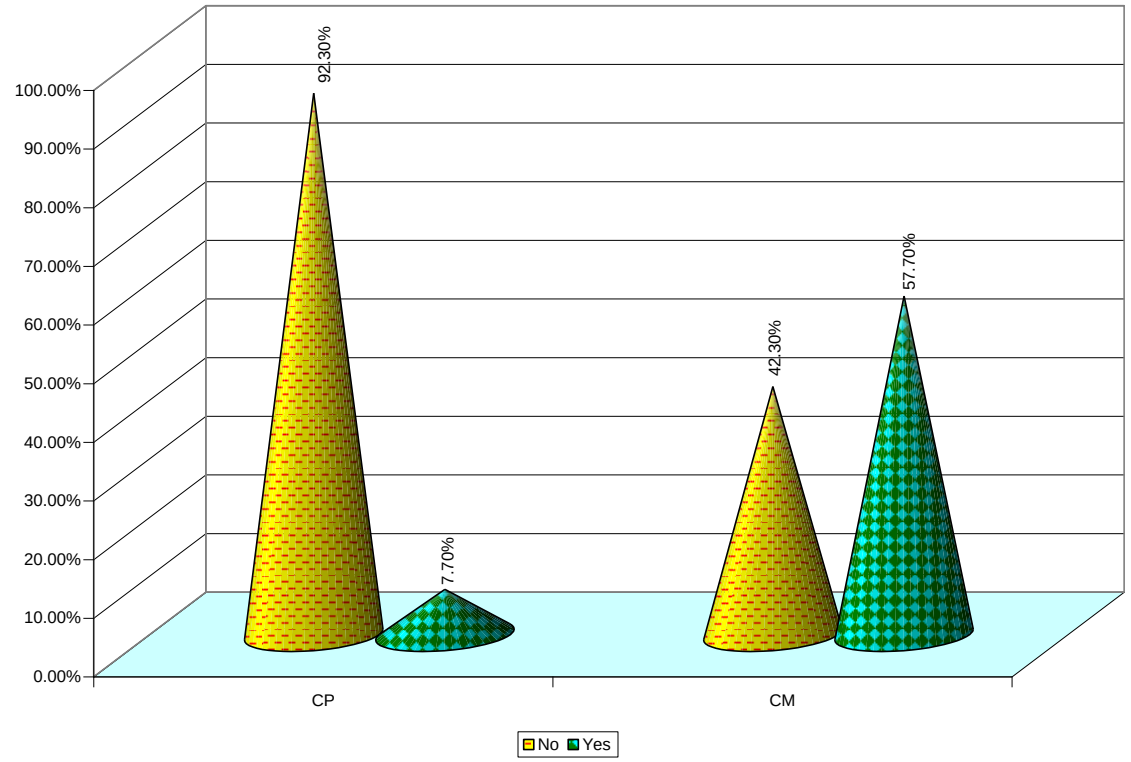
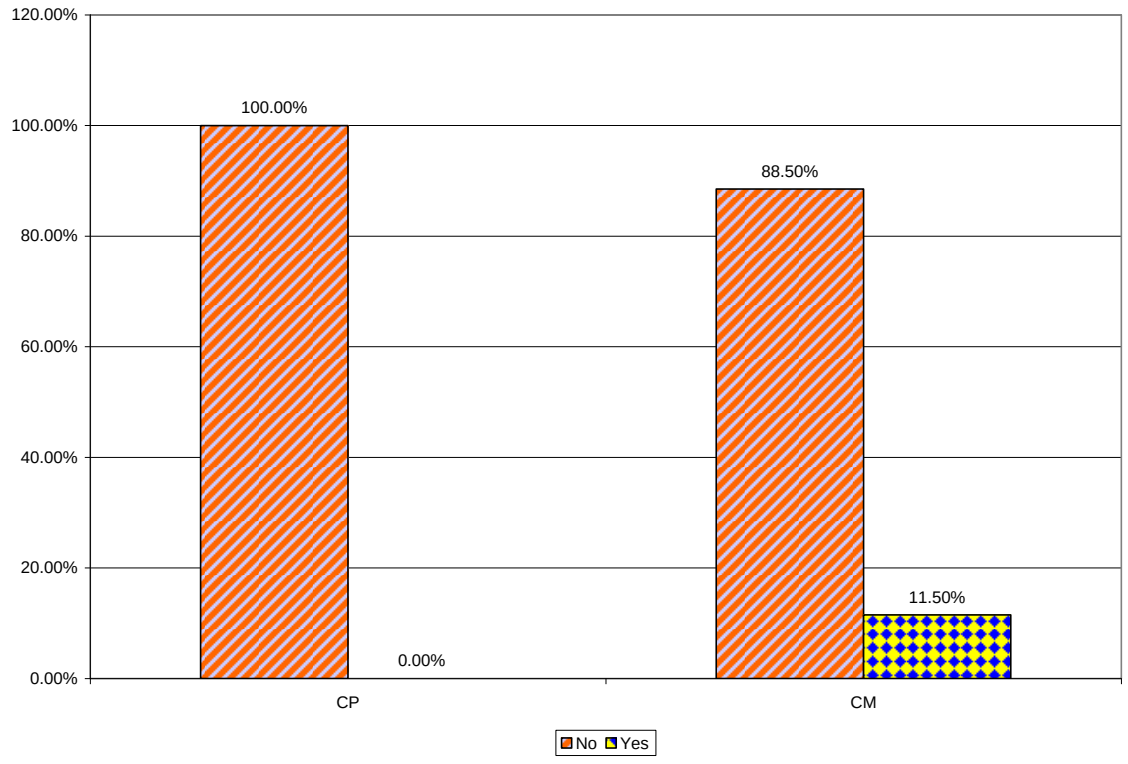


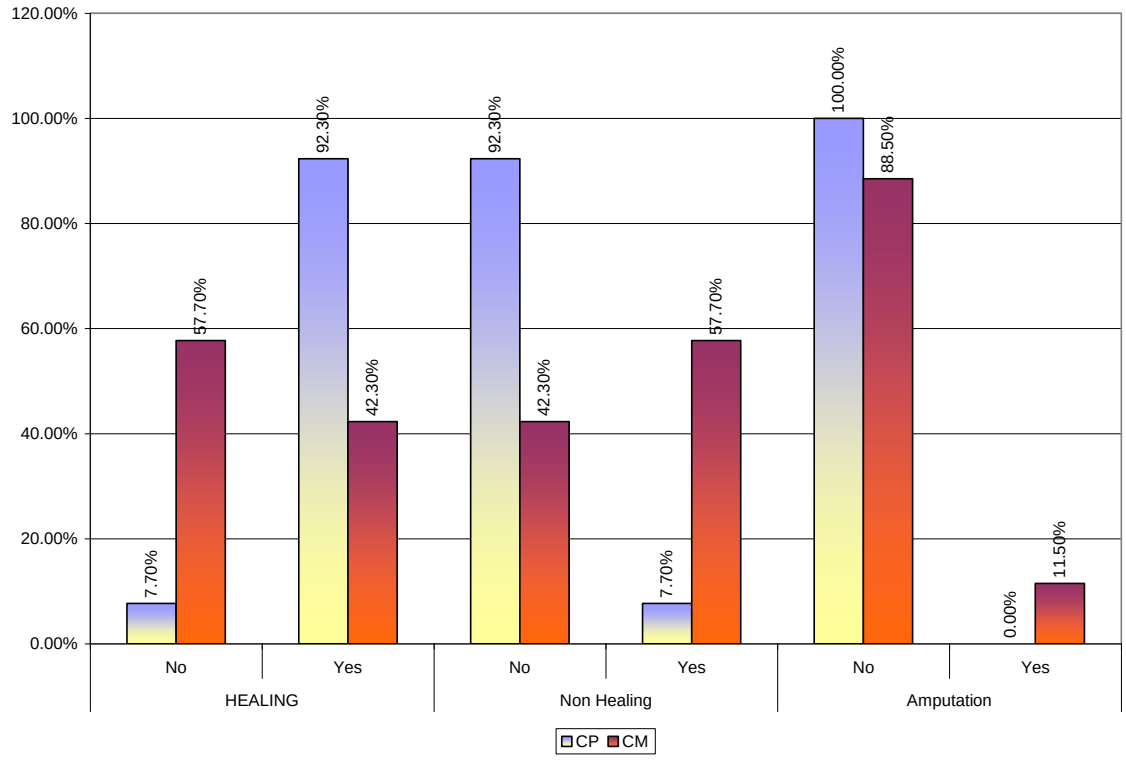
TABLE:10: CHI-SQUARE TEST: AMPUTATION

Amputation	CP		CM		Total		Statistical inference
	n	%	n	%	n	%	
No	52	100.0%	46	88.5%	98	94.2%	X^2=6.367 Df=1
Yes	0	.0%	6	11.5%	6	5.8%	0.012<0.05
Total	52	100.0%	52	100.0%	104	100.0%	Significant

CHI-SQUARE TEST: AMPUTATION



EFFICACY OF COLLAGEN PARTICLES (CP) COMPARE TO CONVENTIONAL METHOD (CM) DRESSING:



DISCUSSION:

Wound healing involves expression of various growth promoting factors which promotes cell differentiation, proliferation & migration, collagen deposition and formation of new connective tissue matrix. Collagens are Proline-rich proteins that are fibrous with long, stiff, triple stranded helical structure comprising of three α-chains. The major collagen molecules that give tensile strength to skin are heterotrimeric collagen type I, formed by 2 alpha 1(I) chains & one alpha 2(I) chain &homotrimeric collagen type III, formed by three α1 (III) chains. The use of the collagen particles in improving the wound healing process by stimulating fibroblast activity.Wound healing process may be complex process which involves a lot of chemical & biological reactions. Collagen plays an important role in extra cellular component for wound repair & tissue remodeling. The use of collagen particles as delivery system which may be comprehensive & diverse. Collagen particles may be converted into soluble solution & it can be molded into several forms of drug delivery systems. It is biocompatible & safe. The use of collagen particles in biomedical use may be rapidly growing & it can be used widely to bioengineering areas. Collagen particles are good biomaterial for use of biomedical implantable devices & it can be used as a matrix for tissue regeneration outside of our body. Collagen

based membranes are chemotactic for regenerative cells & it can stimulate the migration & fibroblasts attachments through its ability to create space.

Coming to advantages of collagen dressings over conventional dressings, frequency in change of dressing is reduced causing less discomfort for the patients, no case of allergic response to collagen particles has been reported in our study and application methodology is easier when compared to sugar dressings or vacuum dressings where a negative pressure system is required. Disadvantages are, this is not a cost effective treatment when used for bigger wounds, cannot be applied over infective wounds where debridements at initial setting are needed. The use of new dressing modalities with use of collagen particles can increase the potentials in wound healing but further studies are needed to prove the efficacy.

Present study is a prospective study regarding collagen particles dressing versus conventional dressing. It was conducted between September 2014 to August 2016 in which 104 patients who presented with chronic non healing ulcers of various etiologies were chosen by random sampling technique, and were grouped into two groups consisting of 52 patients each to show the efficacy of collagen particles dressing.

The present study made a comparison between the collagen particles dressing and conventional method dressing. The efficacy and wound healing capacity of both the methods were gauged using suitable statistical test. The study revealed some interesting results.

The study constituted a total of 104 participants. The Age of the patients ranged from 27 to 92 years. Mean age was 54.81 ± 16.134 years in cases and 54.27 ± 15.575 years in controls. The difference in mean age between cases and controls was not statistically significant ($p>0.05$).

Majority of the participants in the study were males in both the case and the control group. The difference in sex distribution of case and controls was not statistically significant ($p>0.05$). This could be because males are more prone to traumatic wounds and the prevalence of diabetes is also known to be higher in middle-aged males.

The study revealed that the participants presented with various wounds. However, post debridement wounds were the most common in both case and control group followed by post traumatic wound. Majority of the wounds were present in the lower limbs, followed by upper limb, chest, back and abdomen.

The wound duration in the cases and control groups were not found to be statistically significant ($p>0.05$). The mean duration of wound in cases was 16.90 ± 4.960 weeks and 15.56 ± 4.376 weeks in control group.

The percentage of wound healing was also compared between the cases and controls after 12 weeks. Though there was no statistically difference that was recorded between the groups based on the percentage of wound healing, the cases group recorded a higher percentage of wound healing compared to the control group. The case group in the study recorded 92.3% in wound healing compared to 42.3% in the control group.

The study highlighted some important distinctions between the use of collagen particles in wound healing and conventional methods. The study marked essential benefits in the healing procedure by use of collagen. However, further in-depth attempts are needed in order to make a robust case of newer form of wound dressing / healing

CONCLUSION:

Collagen particles are effective in hastening the healing process by formation of early granulation tissue and wound contraction. So that, the number of debridements and dressings required can be reduced; as supported by the present study. Daily dressings may be uncomfortable for the patient causing pain affecting their social well being. Collagen dressings can be changed once in 2-3 days depending on the wound burden subjecting the patients for less discomfort.

Collagen particles wound dressing methods have significant role in increasing the rate of the wound healing process compared to conventional method dressings like betadine dressings. It may decreases the chances of requirement of Split Skin Graft and also reduce the rate of amputation in limbs. Further, it also reduces the hospital stay and also cost of treatment of patients & use of antibiotics.

Probably its spongy network contributes to exudate absorption & blocking of possible extensions of the wound, preventing bacterial growth that wound delay the healing process. Since the sample size of study is not too large, further studies will be required to evaluate the efficacy of collagen particles usage.

When we compare the use of collagen granule dressings with the control group (betadine group) for the treatment of chronic non healing ulcers, the following conclusions were derived.

1. Collagen particles showed faster and better healing rates among the study group

2. Area reduction was statistically significant in the study group

3. There was no adverse effects or reactions seen when collagen particles were applied over the wound

APPENDIX

CASE PROFOMA:

CHRONIC NON-HEALING ULCERS

NAME: **AGE:** **SEX:**

HOSPITAL IP NO: **ADDRESS:** **OCCUPATION:**

SOCIO- ECONOMIC STATUS:

CLINICAL EXAMINATION OF AN ULCER:

Patient history

Mode of onset

Duration of ulcer

Pain – onset, duration, radiating, progression, aggravating & relieving factors

Pus discharge from the wound

Past history of associated disease & treatment history

LOCAL EXAMINATION:

INSPECTION:

Site, Size, Shape

Numbers

Margins, Edge, Floor

Discharge from wound

Surrounding area of ulcer

PALPATION:

Tenderness

Warmth

Palpation of edge for any induration

Palpation of base for any induration & fixity

Depth

Bleeding on touch

deeper structures palpation

Surrounding area of skin &tibial bone or calcaneum or any other underlying bones

Joint mobility

Regional lymph nodes examination is very essential

SYSTEMIC EXAMINATION:

Examination of peripheral arterial pulse & cardio vascular system

Examination of varicose veins in patient standing position

Examination of abdomen

Examination of the spine

BIBLIOGRAPHY

1. Pudner R, Wound management: The management of patients with a leg ulcer. Journal of Community Nursing,1998. 12(3): 26–33.

2. Anon, The Saint Vincent Declaration on diabetes care and research in Europe. ActaDiabetol, 1989. 10(Suppl): 143–144.

3. Kerstein MD, Economics of quality ulcer care. DermatolNurs, 2003. 15(1): 59–61.

4. Jiwa F, Diabetes in the 1990s—an overview. Stat Bull MetropInsur Co, 1997. 78(1): 2–8.

5. Callam MJ, Ruckley CV, Harper DR, Dale JJ, Chronic ulceration of the leg: extent of the problem and provision of care. BMJ,1985. 290(6485): 1855–6.

6. Bauling PC, A review of the impact of dressings on quality of life. In: Suggett A, Cherry G, Mani R, Eaglstein W, eds. Evidence-based woundcare: proceedings of a conference sponsored by Smith and Nephew held in York, UK on 17th November 1997. Royal Society of Medicine Press, London, 1998.39–42.

7. Singer AJ, Clark RA. Cutaneous wound healing. N Engl J Med. 1999;341:738- 746.

8. Witte MB, Barbul A. General principles of wound healing. SurgClin North Am. 1997;77:509-528.

9. Loots MA, Lamme EN, Zeegelaar J, Mekkes JR, Bos JD, Middelkoop E. Differences in cellular infiltrate and extracellular matrix of chronic diabetic and venous ulcers versus acute wounds. J Invest Dermatol. 1998;111:850-857.

10. Jude EB, Boulton AJ, Ferguson MW, Appleton I. The role of nitric oxide synthase isoforms and arginase in the pathogenesis of diabetic foot ulcers: possible modulatory effects by transforming growth factor beta 1. Diabetologia. 1999;42:748- 757.

11. Baker SR and Stacey MC. (1994) Epidemiology of chronic leg ulcers in Australia Australian and New Zealand Journal of Surgery 64 258-261.

12. O'Brien JF, Grace PA, Perry IJ and Burke PE (2000) Prevalence and aetiology of leg ulcers in Ireland Irish Journal of Medical Science 169 (2) 110-113.

13. Pham HT. Wound care in diabetic foot ulceration. Wounds. 2000;12(suppl B): 82B-89B.

14. American Diabetes Association. Consensus Development Conference on Diabetic Foot Wound Care: 7-8 April 1999, Boston, Massachusetts. Diabetes Care. 1999;22:1354-1360.

15. Steed DL, Donohoe D, Webster MW, Lindsley L, for the Diabetic Ulcer Study Group. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. J Am Coll Surg. 1996;183:61-64.

16. Mian M, Beghe F, Mian E. Collagen as a pharmacological approach in wound healing. Int J Tissue React. 1992;14(Suppl):1–9.

17. Donaghue VM, Chrzan JS, Rosenblum BI, Giurini JM, Habershaw GM, Veves A. Evaluation of a collagen-alginate topical wound dressing in the management of diabetic foot ulcers.Adv Wound Care. 1998;11:114-119.

18. Bell E, Erlich HP, Buttle DJ, Nakatsuji T: Living tissue formed in-vitro and accepted as skin-equivalent tissue of full thickness. Science 1981; 211:1052-1054.

19. Alvarez OM, Biozes D: Cultured epidermal autografts for partial thickness and full thickness wounds. ClinDermatol 1984; 2:54-67.

20. Wieman TJ, Smiell JM, Su Y. Efficacy and safety of a topical gel formulation of recombinant human platelet-derived growth factor-BB (becaplermin) in patients with chronic neuropathic diabetic ulcers: a phase III randomized placebocontrolled double-blind study. Diabetes Care. 1998;21:822-827.

21. Pollak RA, Edington H, Jensen JL, Kroeker RO, Gentzkow GD, and the Dermograft Diabetic Ulcer Study Group. A human dermal replacement for the treatment of diabetic foot ulcers.Wounds. 1997;9:175-183.

22. Pham HT, Rosenblum BI, Lyons TE, et al. Evaluation of a human skin equivalent (Apligraf), for the treatment of diabetic foot ulcers in a prospective, randomized, clinical trial. Wounds. 1999;11:79-86.

23. Baker SR, Stacey MC, Jopp-McKay AG, Hoskin SE, Thompson PJ. (1991) Epidemiology of chronic venous ulcers British Journal of Surgery 78 864-867.

24. Callam MJ, Harper DR, Dale JJ, Ruckley CV. (1987) Chronic ulcer of the leg: clinical history British Medical Journal 294 1389-1391.

25. Cornwall JV Dore CJ, Lewis JD. (1986) Leg ulcer epidemiology and aetiology British Journal of Surgery 73 693-697.

MASTER CHART

SI NO	NAME	AGE	SEX	IP NO	DOA	ULCER	DUR WEEKS	CP	CM	HEALING	NH	AMPUTATION
1	MARISAMY	48	M	64423	10.9.14	DU	18	YES	NO	YES	NO	NO
2	KUMAR	36	M	66541	14.9.14	DU	12	YES	NO	YES	NO	NO
3	MURUGAN	77	M	69987	20.9.14	AU	16	NO	YES	NO	YES	NO
4	PERUMAL	65	M	71023	26.9.14	DU	19	YES	NO	YES	NO	NO
5	KATHAYI	56	F	72002	30.9.14	DU	13	NO	YES	YES	NO	NO
6	DEIVAM	59	M	72998	3.10.14	VU	18	YES	NO	YES	NO	NO
7	DHARMA	43	M	73567	15.10.14	DU	12	YES	NO	YES	NO	NO
8	SIVANESAN	78	M	76321	23.10.14	PU	16	NO	YES	NO	YES	NO
9	BALU	45	M	77001	28.10.14	DU	13	NO	YES	YES	NO	NO
10	SAROJA	56	F	80011	2.11.14	DU	12	YES	NO	YES	NO	NO
11	MOOKAN	85	M	82342	15.11.14	PU	15	YES	NO	NO	YES	NO
12	MUTHUVEDI	64	M	84567	22.11.14	DU	17	YES	NO	YES	NO	NO
13	SIVAM	34	M	86990	26.11.14	AU	12	NO	YES	YES	NO	NO
14	ARUMUGAM	32	M	87135	30.11.14	VV	18	YES	NO	YES	NO	NO
15	KARUPPAN	65	M	88971	2.12.14	DU	22	NO	YES	NO	YES	YES
16	CHINNATHA	75	F	91232	11.12.14	DU	14	NO	YES	YES	NO	NO
17	AYYAKANNU	92	M	93421	19.12.14	DU	15	YES	NO	YES	NO	NO
18	KANAGU	48	M	94899	25.12.14	AU	12	NO	YES	NO	YES	NO
19	BUTTAN	80	M	95675	31.12.14	DU	16	YES	NO	YES	NO	NO
20	RAJAMANI	65	F	3200	4.1.15	DU	18	YES	NO	YES	NO	NO

21	SURESH	42	M	4321	8.1.15	DU	19	YES	YES	YES	NO	NO
22	VETRI	37	M	5345	15.1.15	VU	14	NO	YES	YES	NO	NO
23	DAVASU	64	M	6787	22.1.15	DU	12	NO	YES	NO	YES	NO
24	PANDI	53	M	7109	26.1.15	DU	14	YES	NO	YES	NO	NO
25	SUBASH	28	M	7999	30.1.15	TU	12	YES	NO	YES	NO	NO
26	PETHAIYAN	62	M	8098	2.2.15	DU	17	NO	YES	YES	NO	NO
27	SAKUNTHALA	47	F	12009	10.2.14	DU	18	YES	NO	YES	NO	NO
28	PAULRAJ	43	M	13675	16.2.14	DU	15	NO	YES	NO	YES	NO
29	MUTHU	56	M	14321	22.2.14	DU	19	YES	NO	YES	NO	NO
30	RAMAN	51	M	15009	26.2.14	AU	12	YES	NO	YES	NO	NO
31	SOKKU	60	M	16876	3.3.15	DU	14	NO	YES	YES	NO	NO
32	PALANI	63	M	17876	9.3.15	VU	12	NO	YES	YES	NO	NO
33	RAMESH	29	M	18654	16.3.15	TU	12	YES	NO	YES	NO	NO
34	CHANDRA	66	F	22001	23.3.15	AU	12	NO	YES	YES	NO	NO
35	MANOHAR	49	M	23453	28.3.15	VU	12	YES	NO	YES	NO	NO
36	MUSTAFA	55	M	24343	3.4.15	DU	19	YES	NO	NO	YES	NO
37	MUTHUSAMY	63	M	25676	10.4.15	DU	22	YES	NO	YES	NO	NO
38	AHAMEDALI	43	M	27654	12.4.15	AU	12	NO	YES	NO	YES	NO
39	SUBRAMANI	49	M	28988	19.4.15	DU	12	YES	NO	YES	NO	NO
40	KANNAN	28	M	31234	25.4.15	TU	12	NO	YES	YES	NO	NO
41	RANI	36	F	32765	2.5.15	DU	22	NO	YES	YES	NO	NO

42	XAVIER	44	M	33980	6.5.15	DU	24	YES	NO	YES	NO	NO
43	MANI	50	M	35008	10.5.15	DU	21	YES	NO	YES	NO	NO
44	SHANKAR	65	M	37987	20.5.15	DU	28	NO	YES	NO	YES	YES
45	GUNASEELAN	66	M	38999	25.5.15	VU	12	NO	YES	NO	YES	NO
46	ARPUTHAM	83	F	40543	1.6.15	DU	24	NO	YES	YES	NO	NO
47	JAYARAMAN	56	M	42349	8.6.15	DU	26	YES	NO	YES	NO	NO
48	RENGASAMY	42	M	43657	14.6.15	AU	12	NO	YES	YES	NO	NO
49	MARUTHU	39	M	46754	20.6.15	VU	12	YES	NO	YES	NO	NO
50	KASINATHAN	67	M	47006	26.6.15	DU	22	NO	YES	NO	YES	NO
51	KALIYAN	70	M	48876	2.7.15	DU	24	YES	NO	YES	NO	NO
52	SUNDARI	48	F	49843	9.7.15	AU	12	NO	YES	NO	YES	NO
53	LOGANATHAN	58	M	50543	15.7.15	DU	12	NO	YES	YES	NO	NO
54	PRABHU	30	M	54321	23.7.15	VU	12	NO	YES	NO	YES	NO
55	NATARAJAN	44	M	55987	30.7.15	DU	16	NO	YES	YES	NO	NO
56	PARVATHI	60	F	56654	1.8.15	DU	15	YES	NO	YES	NO	NO
57	UTHIRAPATHI	79	M	57865	8.8.15	DU	18	NO	YES	NO	YES	YES
58	GANESAN	91	M	59008	14.8.15	PU	12	YES	NO	YES	NO	NO
59	RAMU	82	M	62345	20.8.15	DU	18	YES	NO	YES	NO	NO
60	SIVAKUMAR	47	M	63431	23.8.15	DU	16	NO	YES	YES	NO	NO
61	AYYAPAN	35	M	64321	4.9.15	AU	12	YES	NO	YES	NO	NO
62	SELVAM	44	M	65872	12.9.15	VU	12	YES	NO	YES	NO	NO

63	MATHI	27	M	66990	16.9.15	TU	12	NO	YES	NO	YES	NO
64	ANAND	35	M	68435	25.9.15	VU	12	NO	YES	NO	YES	NO
65	PANJALI	65	F	70098	28.9.15	DU	22	YES	NO	YES	NO	NO
66	MOOKAIYAN	75	M	71987	2.10.15	PU	12	NO	YES	NO	YES	NO
67	MURALI	49	M	73218	6.10.15	DU	26	YES	NO	YES	NO	NO
68	ARUL	48	M	76509	13.10.15	DU	17	NO	YES	YES	NO	NO
69	PUSHPAM	56	F	78896	25.10.15	PU	12	YES	NO	YES	NO	NO
70	RAJAPPA	55	M	79565	29.10.15	DU	22	NO	YES	NO	YES	NO
71	RAJARAM	48	M	81876	2.11.15	AU	12	NO	YES	NO	YES	NO
72	MAHESAN	67	M	83457	6.11.15	DU	28	YES	NO	YES	NO	NO
73	SEKAR	65	M	85432	14.11.15	DU	16	YES	NO	NO	YES	NO
74	NAGAMMAL	51	F	86541	21.11.15	VU	12	YES	NO	YES	NO	NO
75	GANAPATHI	49	M	88009	2.12.15	DU	28	NO	YES	NO	YES	YES
76	RAJ	32	M	89654	7.12.15	AU	12	YES	NO	YES	NO	NO
77	SAMIKANNU	77	M	90564	24.12.15	DU	18	NO	YES	YES	NO	NO
78	PONNAIYYA	84	M	94778	28.12.15	PU	12	NO	YES	NO	YES	NO
79	SAMUVEL	29	M	1008	1.1.16	TU	12	YES	NO	YES	NO	NO
80	KUMARAN	55	M	4009	9.1.16	DU	16	NO	YES	NO	YES	NO
81	THANDAPANI	66	M	5432	20.1.16	DU	19	YES	NO	YES	NO	NO
82	ELAVARASAN	59	M	7765	2.2.16	DU	16	NO	YES	NO	YES	NO
83	MANIRAJ	34	M	9876	15.2.16	TU	12	YES	NO	YES	NO	NO

84	DEIVANAI	42	F	13765	25.2.16	DU	12	NO	YES	NO	YES	NO
85	KATHAMUTHU	77	M	16987	7.3.16	AU	12	YES	NO	YES	NO	NO
86	SOLAI	61	M	19874	18.3.16	DU	22	NO	YES	NO	YES	YES
87	MOORTHY	59	M	22564	28.3.16	DU	20	NO	YES	YES	NO	NO
88	ELANGOVAN	46	M	26599	6.4.16	VU	12	YES	NO	YES	NO	NO
89	SATHISH	34	M	28981	16.4.16	AU	12	NO	YES	NO	YES	NO
90	MURUGESAN	38	M	30741	26.4.16	DU	22	YES	NO	YES	NO	NO
91	SENNAPAN	42	M	32573	5.5.16	DU	15	YES	NO	NO	YES	NO
92	BALAGURU	49	M	35666	16.5.16	DU	17	NO	YES	NO	YES	NO
93	PECHIMUTHU	53	F	38009	22.5.16	PU	12	NO	YES	YES	NO	NO
94	SEVALAI	57	M	41984	4.6.16	DU	19	YES	NO	YES	NO	NO
95	NAGARAJ	59	M	44434	10.6.16	VU	12	YES	NO	YES	NO	NO
96	RAJAN	61	M	46008	18.6.16	DU	20	NO	YES	NO	YES	NO
97	VEDIYAPPAN	80	M	48065	2.7.16	DU	24	YES	NO	YES	NO	NO
98	SELVAKUMAR	30	M	49985	14.7.16	TU	12	NO	YES	YES	NO	NO
99	SELVI	40	F	52032	22.7.16	DU	23	YES	NO	YES	NO	NO
100	BALAJI	43	M	53004	28.7.16	AU	12	NO	YES	NO	YES	NO
101	SARAVANAN	55	M	56995	3.8.16	DU	26	YES	NO	YES	NO	NO
102	PONNUSAMY	81	M	58862	15.8.16	PU	12	NO	YES	NO	YES	NO
103	KASIAYYA	66	M	61894	21.8.16	DU	25	YES	NO	YES	NO	NO
104	DANAM	46	F	63243	28.8.16	DU	18	NO	YES	NO	YES	YES