NANO-CRYSTALLINE SILVER DRESSINGS IN COMPARISON TO CONVENTIONAL NORMAL SALINE DRESSINGS IN THE MANAGEMENT OF LOWER LIMB ULCERS. - INTERVENTIONAL COMPARATIVE STUDY

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Dissertation submitted to

BLDE (Deemed to be University) Vijayapur, Karnataka

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

GENERAL SURGERY

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KARNATAKA

2020

Nano-crystalline Silver dressings in comparison to conventional Normal saline dressings in the management of lower limb ulcers.

- Interventional Comparative study

MASTER OF SURGERY

In

GENERAL SURGERY

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

ANATOMY

Lower limb has complex mechanics and structure. The Ankle is one of the most vital joint and works like shock absorber and propulsor of the lower limb. The foot provides flexibility , resilience and can sustain enormous pressure.

The foot and Ankle contains 33 joints, 26 bones, muscles, ligaments ,tendons, nerves ,vessels and soft tissue.

Support, balance and mobility are provided by these components together . A malfunction or structural disturbances in any one part can result in the deranged function elsewhere.

Figure 1. BONES - MEDIAL VIEW

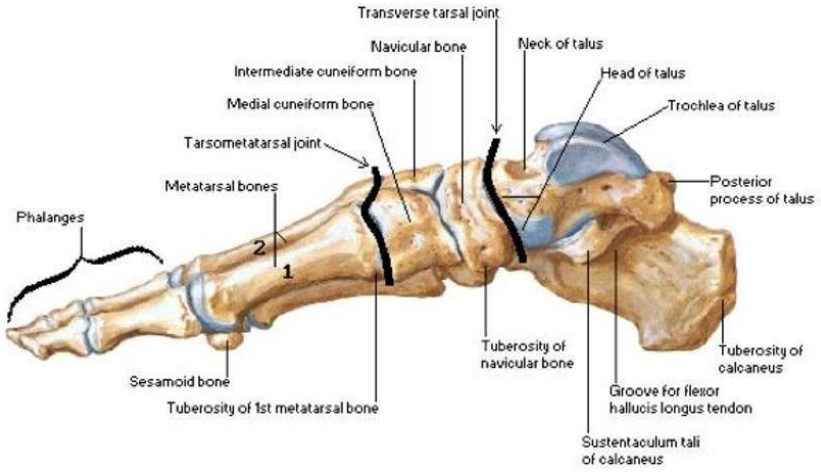
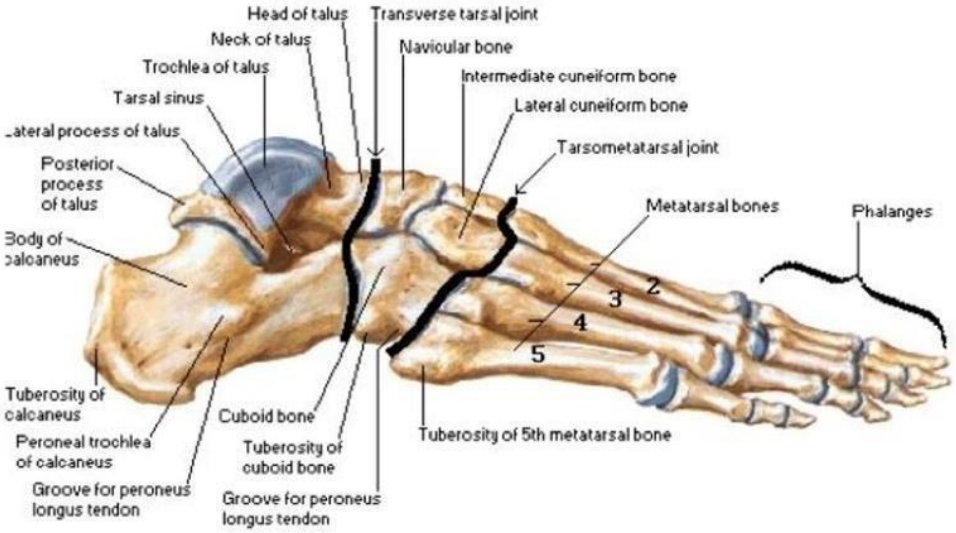


Figure 2. BONES - LATERAL VIEW



Skin

Skin over foot at the dorsal aspect is very elastic thin and pilosebaceous in nature with thick hair. It is less than 2-3 mm in thickness and fibrous septa reach deep till fascial tissues. The skin over the plantar surface is 5mm in thickness over weight bearing regions like heel , head of 1st metatarsal and laterally over the sole. It lacks sebaceous and hair follicles, although it has a lot of sweat glands. Although few elastic fibres are continuous with those of the dermis, the hypodermis is made up of loose, areolar connective tissue, the majority of which is collagenous. Blood vessels and nerve endings are abundantly present in the hypodermis.

Of comparison to the rest of the body, the subcutaneous tissue in the sole is more fibrous, rough, and stinging.

Deep fascia:

Inferior extensor retinaculum is connected with the thin membrane that covers the dorsum of the foot , it unites with plantar aponeurosis and in the front it en-sheathes tendons over the dorsum.

Plantar aponeurosis:

The entire sole is covered by it. It develops posteriorly from the medial and lateral tubercles of the calcaneous below the calcaneo-tendinous insertion. It inserts by separating into five slips, one at each of the five toes, and spreads out across the sole. The plantar aponeurosis is the name for the thick and powerful intermediate region.

Forefoot:

Five phalanges, often known as toes, and their matching metatarsals make up the forefoot. The phalanxes are composed of several smaller bones. The great toe (hallux) can move up and down because to its phalanges, joints, and sesamoid bones which are 2 in number here. The other four phalanges have two interphalangeal joints and three bones apiece. Five metatarso-phalangeal joints attach the phalanges to the metatarsals. Half of the body's weight is supported by the forefoot, which also evenly distributes pressure on the foot's

Midfoot:

Midfoot is formed by arches of foot, it acts like "**shock absorber"**. Midfoot bones are cuboid, 1st 2nd and 3rd cuneiform and navicular .Muscles and plantar fascia aid connecting it to forefoot and hind foot.

Hind foot:

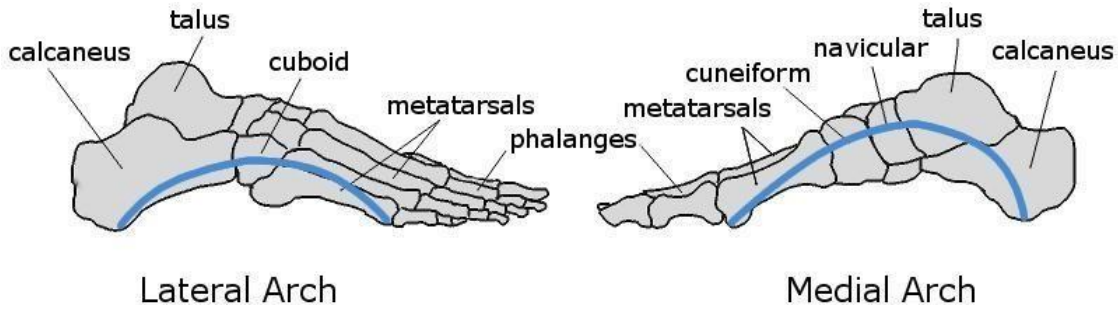
It has connects midfoot to the talus and has 3 joints. Superiorly talus links to Tibia and Fibula, hence allowing cranio caudal movement.. The largest foot bone is the calcaneus. It meets the talus to create the subtalar joint, which allows the ankle to spin. There is a layer of fat that cushions the bottom of the calcaneus..

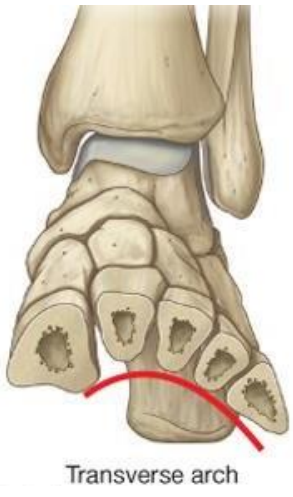
The Arches of Foot:

There are three arches of foot. . Transverse arch comprises of the cuneiform bones, cuboid and five metatarsals. Laterally longitudinal arch comprises of 4th , 5th

metatarsal bones ,calcaneus and cuboid bones. Medially, longitudinal arch comprises of calcaneum, navicular, talus, three cuneiforms, and the three metatarsal bones. The configurations of the bones and ligaments support the foot's arches. In addition to each other, tendons and muscles maintain the foot arch.

Figure 3. ARCHES





Muscle, Tendon and Ligaments of Lower Limb:

Twenty muscles in the foot give the foot its shape, hold the bones in place, and contract and extend to provide the limb movement. There are four layers of muscles on the bottom of the foot: Flexor digitorium brevis, Abductor hallucis, and Abductor digiti minimi are among the muscles in the first layer. The Lumbricals, Flexor hallucis longus, and Flexor digitorum accessory are the muscles in the second layer. Adductor hallucis, Flexor digiti minimi brevis, and Flexor hallucis brevis make up the third layer. Peroneal longus tendons, four dorsal interossei tibialis posterior, and three plantar interossei muscles make up the fourth layer.

Arterial supply of sole:

Medial plantar artery:

It emerges below the flexor retinaculum as a final branch of the posterior tibial artery. It supplies great toe at its medial side at the very end. While giving rise to numerous cutaneous, articular, and muscular branches along the way.

Lateral Plantar Artery:

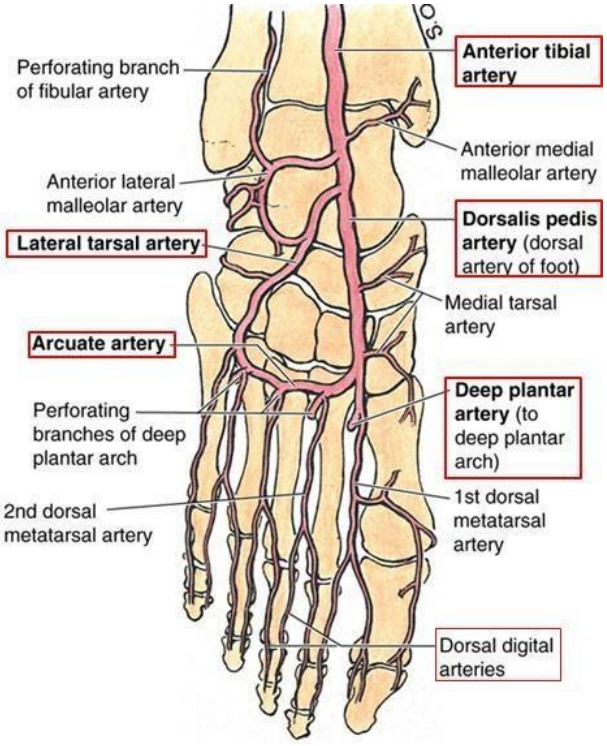
It is one of the posterior tibial artery's terminal branches.. It produces branches that are muscular, cutaneous, and articular as it travels through the body. The neighbouring surfaces of the lateral four phalanges and the same side of the little toe get digital arteries from the plantar arch.

Dorsalis Pedis Artery:

The first dorsal interosseous muscle's two heads are where the first plantar metatarsal

artery, which supplies the cleft between the great and second phalanges, emerges from the sole. Shortly after, it connects to the lateral plantar artery.

Figure 4 : ARTERIAL SUPPLY



(A) Dorsum of foot

Veinous drainage of sole:

The posterior venae comitantes are formed when the medial and lateral plantar veins combine behind the medial malleolus with the respective arteries.

Nerve supply of Sole:

Medial Plantar Nerve:

The medial plantar nerve is the final branch of the tibial nerve.

It provides the first lumbrical muscle, the flexor digitorium brevis, flexor hallucis brevis, and abductor hallucis with muscular branches.

The skin over medial 3 and 1/2 toes are bordered on both sides by plantar digital nerves.

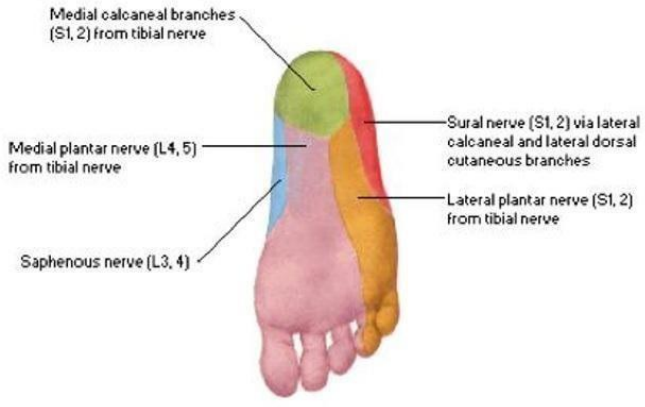
Plantar Nerve (Lateral):

A final branch of the tibial nerve is the lateral plantar nerve.

1. The quadratic plantae and abductor digiti minimi are connected to the main trunk by cutaneous branches, which then supply laterally over the sole.

2. Terminal superficial branch supply to the interosseous muscles of the fourth intermetatarsal space and the flexor digiti minimi.

3. All interossei, with the exception of those in the fourth intermetatarsal space, the abductor hallucis, and the second, third, and fourth lumbricals, are supplied by the deep terminal



branch.

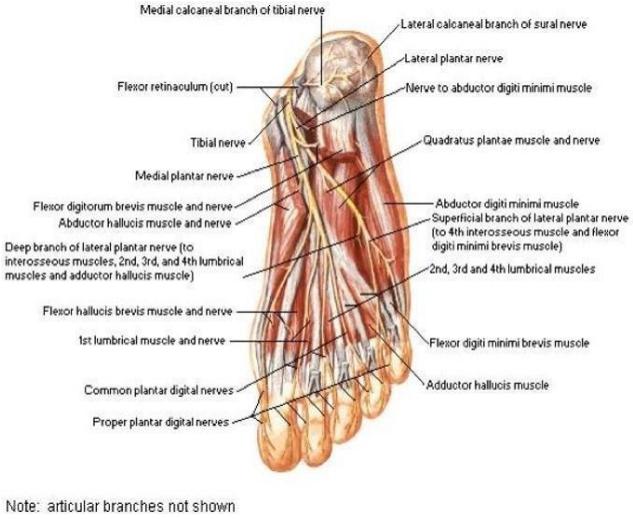


Figure 5 : NERVE SUPPLY

Dorsal venous arch:

The dorsal venous arch is located in the subcutaneous plane and empties medially into the great vein of saphenous (GSV). The GSV emerges from front of the leg, anterior to the medial malleolus. Small saphenous vein enters the leg at the back of medial malleolus.

Arterial supply of dorsum of foot

Dorsalis Pedis :

It is the continuation of anterior tibial artery at the ankle joint. It concludes by descending into the sole and moving between the two heads of the first dorsal interosseous muscle, where it connects with the lateral plantar artery to form the entire plantar arch. It produces the First dorsal metatarsal artery, Lateral tarsal artery, and

Arcuate artery.

Nerve Supply of Foot

Peroneal Nerve (Deep branch):

The medial branch of the deep peroneal nerve supplies the skin on the adjacent sides of the great and second toes. The lateral branch feeds the extensor digitorium brevis muscle.

Spaces of Foot:

Four median Plantar Spaces:

1. The flexor digitorium brevis and the plantar aponeurosis are separated by the first gap.

2. The second opening is situated between the flexor digitorium brevis, quadrates plantae, and the conjoined long flexor tendons.

3. The flexor digitorium longus and the oblique head of the abductor hallucis are separated from each other by the third gap.

4.The oblique head of the abductor hallucis muscle, the second and third metatarsal bones, and their interosseous muscles make up the fourth deepest space.

Infections may spread from one compartment to another . Since the sheaths of the entire flexor tendon extend from the toes and proximally to the distal head of the metatarsal bones, either a localised area or one of the four spaces may exist within these sheaths. The 3rd layer of sole is enclosed inferiorly by plantar fascia and superiorly by the metatarsal and small muscles and ligaments of the foot. It is

continuous distally into the through the lumbricals and web space along with the long flexor tendons.

Propulsion of foot:

Standing immobile:

The metatarsal heads in the front and the heel at the back both upset the body weight.

Walking:

The lateral margin of the sole and metatarsal heads gradually become heavier as the body travels forward. When the heel lifts, the etatarso-phalangeal joints of the fingers lengthen and the plantar aponeurosis contracts, lifting the longitudinal arches.. Hence forward movement is achieved.

1. By gastroenemius and soleus (and plantaris) muscle at the Ankle joint, where foot is used as a lever.
2. Flexors of the foor help flexing the toes to implement final force in forward direction.

The strong contraction of the flexor digitorium longus keeps the toes extended and prevents folding under pressure by contracting the lumbricals and interossei. Long

flexor tendons help to plantar-flex the ankle joint during this action.

ANATOMY OF LEG

The leg is the lower limb region between the knee and the foot. It comprises two bones: the tibia and the fibula. The role of these two bones is to provide stability and support to the rest of the body, and through articulations with the femur and foot/ankle and the muscles attached to these bones, provide mobility and the ability to ambulate in an upright position. The tibia articulates with the femur at the knee joint. The knee joint consists of three compartments.

- Medial tibiofemoral compartment
- Lateral tibiofemoral compartment
- Patellofemoral compartment

At the Ankle, the tibia and fibula create the articular surface for the talus. The ankle mortise is a specialized articulation providing support and optimizing motion and function through the Ankle joint. A typical Ankle joint optimizes and allows for physiologic mobility of the foot and its associated joints and articulations. The bones and fascia also divide the lower leg into four compartments

- Anterior compartment
- Lateral compartment
- Posterior compartment, superficial
- Posterior compartment, deep

Bones & Joints

1. Tibia

- Present medially in the leg, bears weight of the leg
- It connects proximally to the femur and fibula and distally to the talus and fibula.Landmarks of Tibia:
    - Condyles of Tibia:
        - Femoral condyles and horizontal proximal surfaces articulate.
        - They are eparated by the lateral and medial intercondylar tubercles
    - Tibial tuberosity:
        - Triangular, supero-anterior area where condyles connect
        - Site of attachment for the patellar tendon
    - Shaft: anterior, lateral, and posterior surfaces
    - Medial malleolus
        - Distal projection
        - Articulates with the talus as part of the Ankle

2. Fibula

- Thin, lateral bone of the leg
- Articulates with the tibia proximally and distally and with the talus distally
- Important landmarks:
    - Head:
        - Serves as the site of attachment for ligaments of the knee
        - Articulates with the tibia
    - Neck
        - Narrow
        - The common peroneal nerve wraps around it.
    - Shaft: medial, lateral, and posterior surfaces
    - Lateral malleolus

- Distal projection
- Articulates with the talus as part of the ankle

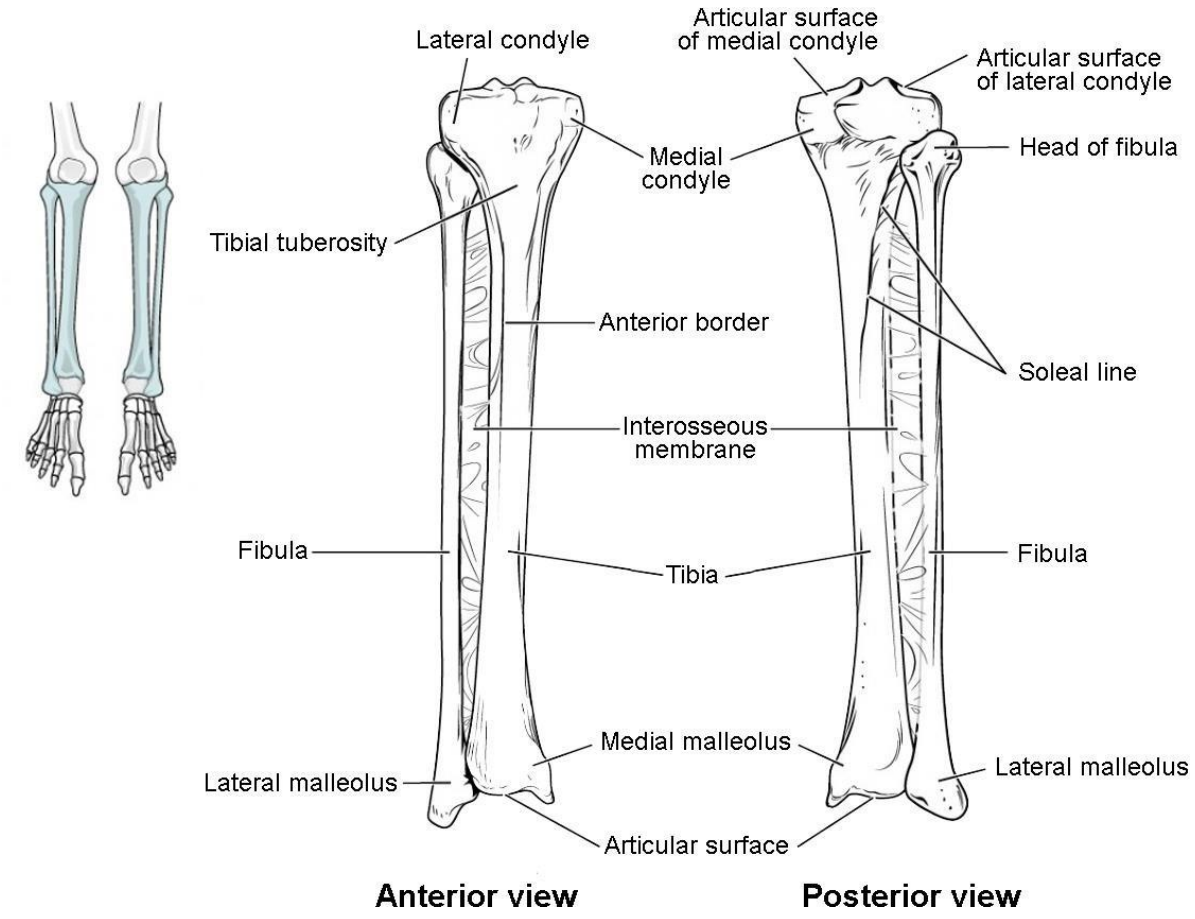


Figure 6

Joints of leg:

- **Tibiofibular joint (Proximal) :**
  - Arthrodial joint in-between the condyle of tibia laterally and the head of fibula.
  - Stabilized by a tough capsule and numerous ligaments
- **Tibiofibular (Distal) :**
  - It is formed by syndesmosis of the distal tibial and fibular end
  - It is stabilized by interosseous membrane and multiple ligaments .
- **Interosseous membrane**
  - Tissues fibres are directed laterally and downwards.

- Connects medial fibular border with lateral tibial border
- Gives stability to the leg
- Has a passageway on the proximal end through which the anterior tibial vessels can flow to reach the anterior compartment of the leg.

Fascial Compartments of Leg

The anterior, posterior, and transverse intermuscular septa, along with the interosseous membrane, divide the leg into four fascial compartments as described below:

1. Anterior compartment:
    - It is divided from lateral compartment by anterior intermuscular septum .
    - It is divided from the deep posterior compartment by the interosseous membrane.
    - Contains 4 muscles:
        - Tibialis anterior
        - Extensor hallucis longus
        - Extensor digitorum longus tendon
        - Peroneus tertius tendon
2. Lateral compartment of Leg:

    The anterior intermuscular septum divides it from the anterior compartment.

    The posterior intermuscular septum divides it from the superficial posterior compartment. It contains 2 muscles Peroneal longus and peroneal brevis.

3. Posterior (superficial) compartment of Leg:
    - The lateral compartment and medial compartment are separated by the posterior intermuscular septum.
    - It contains 3 muscles:
        - Soleus muscle
        - Gastrocnemius muscle
        - Plantaris muscle

4. Deep posterior compartment of Leg:
    - The tibia and interosseous membrane separate the anterior from the posterior compartment.
    - The transverse intermuscular septum separates it from the superficial posterior compartment.
    - It contains 4 muscles:
        - Popliteus
        - Tibialis posterior
        - Flexor digitorum longus
        - Flexor hallucis longus

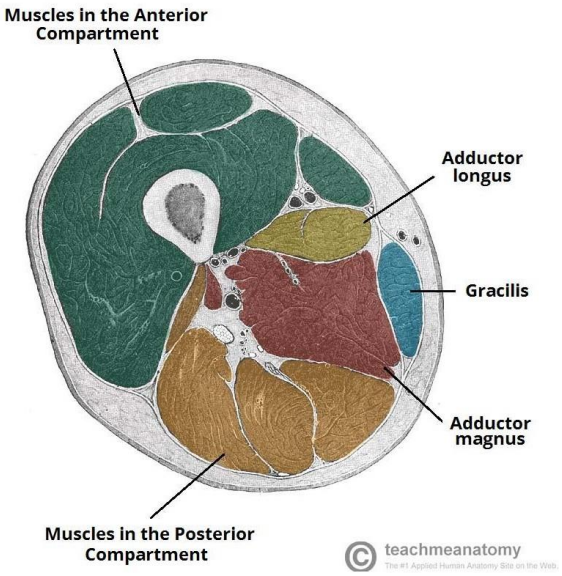


Figure 7

Anterior Compartment of Leg — Dorsiflexor Muscles

- The anterior compartment contains four muscles that control the dorsiflexion or extension of the foot and/or toes.:
    - Tibialis anterior: powerful dorsiflexor of the foot
    - Extensor digitorum longus: extends the 4 lateral toes and the foot
    - Extensor hallucis longus: extends the great toe (hallux) and the foot

Table: Anterior compartment — dorsiflexor muscles

Muscle	Origin	Insertion	Innervation	Function
Tibialis anterior	Lateral condyle, lateral surface of proximal tibia, and interosseous membrane (IO)	Medial cuneiform and base of 1st metatarsal	Deep fibular nerves (L4, L5, S1)	• Dorsiflexion of Ankle • Inversion of foot
Extensor digitorum Longus	Lateral condylar area of tibia, anterior fibular surface	Middle and distal phalanges of 2nd to 5th digits		• Extends digits 2–5 • Dorsiflexes ankle
Extensor hallucis longus	Anterior surface of midfibula and IO	Great toe distal phalanx		• Extends hallux • Dorsiflexes ankle
Fibularis tertius	Anterior surface of lower fibula	Fifth metatarsal base		• Dorsiflexes ankle • Supports Eversion

    - Peroneus tertius: everts and dorsiflexes the foot

- Common nerve supply:

  deep fibular nerve(ventral rami of L4–S2)

- Common blood supply: anterior tibial artery which is a branch of

  popliteal artery

Lateral Compartment of Leg — Evertor Muscles

- Additionally known as the leg's peroneal compartment
- The main muscles involved in foot eversion are those listed here; they cause flexion and extension at ankle joint..
    - peroneus longus and peroneus brevis muscle.
- To enter the foot deeply in respect to the fibular (or peroneal) retinaculum, both muscle tendons pass posterior to the lateral malleolus.
- Nerve supply: superficial branch of fibular nerve
- Blood supply: perforating branches arising from the anterior tibial artery

Table: Lateral compartment evertor muscles

Muscle	Origin	Insertion	Innervation	Function
Fibularis longus	Head and proximal ⅔ of lateral fibula	First metatarsal and 1st cuneiform	Fibular nerves (superficial branch)	
Fibularis brevis	Midportion of lateral fibula	Fifth metatarsal base		Everts foot ; weak plantar flexion

Posterior Compartment of the Leg — Flexor Muscles

- The transverse intermuscular septum separates the flexor muscles into two layers ie. superficial and deep, with posterior compartment's muscle being principally in charge of plantar flexion.

- The muscles of the superficial layer include the soleus and paired gastrocnemii of the triceps surae, as well as the plantaris.
- The following muscles are part of the deep layer: Popliteus, Tibialis posterior, Flexor digitorum longus, and Flexor hallucis longus.
- Muscular tendons pass behind the medial malleolus, with the exception of the popliteus
- Common nerve supply: tibial nerve
- Blood supply: fibular vessels (deep layer) and posterior branch of tibial artery

Posterior compartment of the leg (Superficial layer)

Blood Supply

- **Arteries of the leg**

  The popliteal artery provides blood to the lower leg. The artery splits at the lower margin of the popliteal fossa and branches out to the anterior compartment, anterior tibial artery, posterior compartment, and lateral compartment to form the tibioperoneal trunk.

- **Veins of the leg**

There are two venous drainage systems in the leg:
The superficial or saphenous veins are found in the subcutaneous tissue, while the deep venous branches combine to create the popliteal vein.

Deep veins of the leg:

The following veins are associated with the arteries of the same name and are located deep within the deep fascia of the leg:

o Anterior tibial veins

o Posterior tibial veins o Peroneal veins

• Converge to produce the popliteal vein near the inferior border of the popliteus muscle.

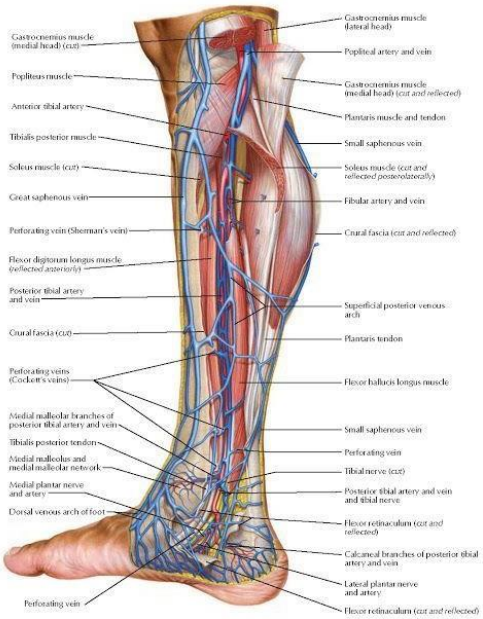


Figure 8

Venous drainage

Innervation of Leg

The lumbosacral plexus provides the lower limb's sensory and motor innervation. (L1–S4).

Sciatic nerve: The nerve that extends the farthest from the lumbosacral plexus separates into the tibial nerve and the common peroneal nerve after sprouting multiple branches.

Tibial nerve- It passes through the tarsal tunnel and runs inferior and posterior to the medial malleolus in the ankle, supplying motor function to the posterior compartment of the leg as well as multiple sensory branches to the entire leg (the sural, medial calcaneal, medial and lateral plantar nerves).

Sural nerve: It supplies sensory nerves to the epidermis and dermis of sole at lateral side and posterolateral part of leg ,mainly the distal 1/3rd.

Common peroneal nerve(common fibular nerve): The short head of the biceps femoris receives motor function from it before splitting into 2 branches:

Fibular nerve (Deep branch): It is a branch of common peroneal nerve, which provides sensory information to the first interdigital space of the foot and motor function to the anterior compartment.

Fibular nerve (Superficial branch): It is a branch of common peroneal nerve and provides sensory information to the dorsum of the foot as well as motor function to the lateral compartment of the leg.

Saphenous nerve - It is a femoral nerve branch that emerges from the femoral triangle and has purely sensory function for the skin on the medial half of the leg.

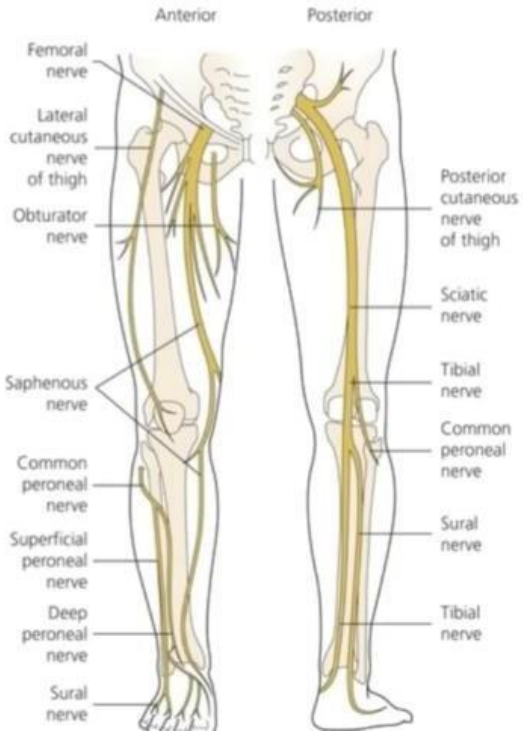


Figure 9: Nerves of Leg

WOUND

DEFINITION

Wound is a break or discontinuity in the integrity of skin or tissues. It can be a simple wound or a complex wound.

CLASSIFICATION OF WOUND[1]

1. Tidy wounds

They are wounds of surgical incisions and caused by sharp objects.

Usually primary suturing is done. Healing is by primary intention.

2. Untidy wounds

They are:

• Crushed

• Tear

• Avulsion

• Devitalized injury

• Vascular injury

• Multiple irregular wounds

• Burns, etc.

Fracture may be present.

OTHER CLASSIFICATION:

1. Clean incised wound-It is a wound caused by sharp objects like knife, glass or blades. Primary suturing is done and it heals by first intention leaving a thin, linear scar.

2. Lacerated wound-Wound edge is devitalized, crushed and wide. It is treated by wound excision and delayed primary suturing. Scar formed is wide and prone for hypertrophic scar formation.

3. Bruising, contusion.

4. Hematoma.

5. Closed blunt injury.

6. Puncture wounds and bites.

7. Abrasion-It is superficial, and is due to shearing of skin in which surface is rubbed off. It heals by epithelialization.

8. Traction and avulsion injury.

9. Crush injury-Is caused by war wounds, road traffic accidents, and tourniquet.

It leads on to-

• Compartment syndrome

• Muscle ischemia

• Gangrene, loss of tissue.

10. War wounds and gunshot injuries.

11. Injuries to bones and joints may be open or closed.

12. Injuries to nerves, either clean cut or crush.

13. Injuries to arteries and veins (major vessels).

14. Injury to internal organs may be penetrating or non-penetrating (blunt) injuries.

CLASSIFICATION OF SURGICAL WOUNDS[2]

1. Clean wound

• Herniorrhaphy

• Excisions

• Surgeries of the brain, joints, heart, transplant.

• Infective rate is less than 2%.

2. Clean contaminated wound

• Appendicectomy

• Bowel surgeries

• Gallbladder, biliary and pancreatic surgeries.

• Infective rate is up to 30%-high.

3. Contaminated wound

• Acute abdominal conditions

• Open fresh accidental wounds.

4. Dirty infected wound

• Abscess drainage

• Pyocele

• Empyema gallbladder

• Fecal peritonitis.

WOUND HEALING

STAGES

• Stage of inflammation.

• Stage of granulation tissue formation and organization. Here as the result of fibroblastic activity, svnthesis of collagen and around substance occurs.

• Stage of epithelialization-It occurs in 48 hours.

• Stage of wound contraction and connective tissue formation.

• Stage of scar formation and resorption.

• Stage of maturation.

SEQUENCE OF EVENTS OF WOUND HEALING

The fundamental purpose of the skin is to protect from outside environmental injury and as a first line of defence. Injury-related loss of skin continuity and integrity may result in chronic wounds that never heal, which may cause serious impairment. It takes a variety of tissues, cell types, and matrix elements to properly repair a wound, which is a complex process[11].

Following a wound injury, a series of events happen, including the inflammatory , proliferative , and remodelling phases occur in order to facilitate tissue restoration. [18] Despite being designed to protect the host from infection, neutrophils can secrete enzymes that cause tissue damage minutes after injury. In turn, this frequently delays the resolution of inflammation and results in wounds that do not heal. [49] Wound stagnation has been linked to a number of factors, including the presence of biofilm, necrotic tissue, poor circulation, and prolonged pressure. [50]

Non healing ulcers of chronic nature is one of the commonest conditions leading to IPD care in surgical wards. Common causes uncontrolled Type 2 Diabetes , Peripheral occlusive vascular disease (POVD), Trauma and Varicose ulcer. The lower limb is more frequently impacted. This illness affects almost 1% of adult population, and twelve percent of non-healing ulcer foot cases end in amputation. The likelihood of an ulcer forming in the opposite extremity increases once a patient has had an amputation. Even if the limb is saved, the patient will still experience physical, mental, and financial pain from the protracted use of antibiotics, hospitalisation, and recurrent wound debridement. Additionally, new kinds of bacteria that are resistant to antibiotics are developing on a daily basis, raising the expense of treatment. Nevertheless, recurrent debridement of ulcer, toileting with normal saline, and antibiotics are the mainstays of care for chronic non-healing ulcers in most hospitals.

Following a wound injury, a series of wound healing phases is initiated, leading to tissue repair. There are three clearly defined overlapping phases:

1) Phase of inflamation

2) Phase of proliferation

3) Phase of remodelling[18].

Massive neutrophil infiltration into the subdermal area of wound as part of the inflammatory phase is especially important on the first post-injury day. Neutrophils are hypothesised to protect the host from infection by fending off an invasive microbe and removing cellular waste starting minutes after injury. Activated neutrophils release a variety of bioactive chemicals during this process, including proteases and reactive oxygen intermediates, which can seriously harm tissue. The resolution of inflammation is frequently postponed in non-healing wounds, which best illustrates this [49].

Kinds of wound healing

Healing by 1st intention : It is healing of a clean incised wound, which leads to thin, linear scar.

Healing by 2nd intention: It is healing of an infected wound, which leads to wide and poor scar mark.

GRANULATION TISSUE

It consists of the growth of new capillaries, fibroblasts, RBCs, and WBCs mixed together with a thin fibrin layer on top.

TYPES:

- Healthy granulation tissueIt happens when an ulcer is mending. With a serous discharge, it has a sloping edge. When touched, it bleeds. When the granulation tissue is healthy, skin grafting takes nicely. Before performing a skin graft, streptococci growth in culture must be less than 105/gramme of tissue.

- Unhealthy granulation tissue: It is pale with purulent discharge. Its floor is covered with slough. Its edge is inflamed and edematous. It is a spreading ulcer.
- Unhealthy, pale, flat granulation tissue: It is seen in chronic nonhealing ulcer (callous ulcer).
- Exuberant granulation tissue (Proud flesh): It occurs in a sinus wherein granulation tissue protrudes out of the orifice of the sinus like a proliferating mass. It is commonly associated with a retained foreign body in the sinus cavity.
- Pyogenic granuloma: It is a type of exuberant granulation tissue. Here granulation tissue protrudes out from an infected wound or ulcer bed, presenting as well localized, red swelling, which bleeds on touch.

Different Discharges In An Ulcer :

Serous :	in healing ulcer.
Purulent : 1. Staphylococci : 2. Streptococci : 3. Pseudomonas :	in infected ulcer. yellowish and creamy. bloody and opalescent. greenish color.
Bloody:	malignant ulcer, healing ulcer from healthy granulation tissue
Sero-purulent	
Sero-sanquinous:	serous and blood.
Serous with sulfur granules:	actinomycosis
Yellowish:	tuberculous ulcer.

ULCER

ULCER[21]: Any breach in the continuity of the overlying epithelium is called as ulcer , epithelium being skin or mucous membrane. It occurs as a result of molecular death of epithelium or its removal following trauma.

PARTS OF AN ULCER

- **FLOOR –** The visible surface
- **EDGE –** Part of ulcer between the margin and floor.
- **MARGIN -**Junction/Boundary between the ulcer & normal epithelium
- **BASE** - It is part of the underlying tissue over which the ulcer rests.

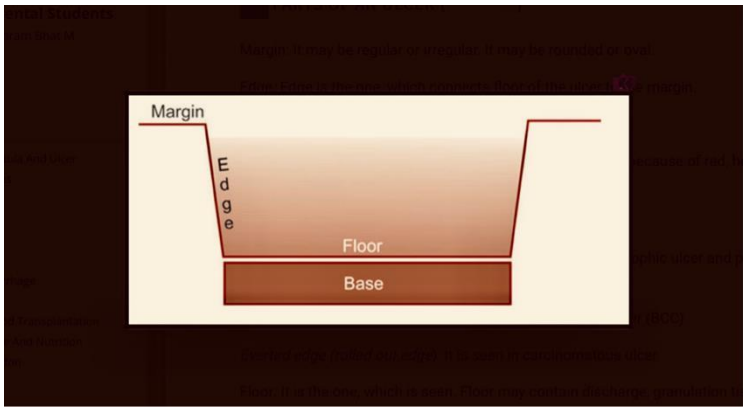


Figure 10

Different types of Edges:

It helps in diagnosing and also knowing condition of the ulcer.

. Spreading ulcer - Inflammed, edamatous

. Healing ulcer- 3 Zones

• Inner/ reddish – granulation tissue

• Middle/ blue – growing epithelium

• Outer/ white – developing scar

1. Undermined edge – The pathology destroys the underlying tissue faster than the overlying epithelium. Eg: TUBERCULAR
2. Punched out – At this point, the edge descends at a right angle to the skin's surface. Disease doesn't spread to surrounding skin. Eg: GUMMATOUS, TROPHIC
3. Sloping Edge- It is seen typically in - Healing Ulcers Traumatic Ulcers Venous Ulcers
4. Elevated & Pearly white with Beaded edge - Typical of Rodent Ulcer
5. Rolled out / Everted edge - Growing edge section piles up and spills over the surrounding skin. Eg. Squamous Cell Ca. / Ulcerated Adeno Ca.

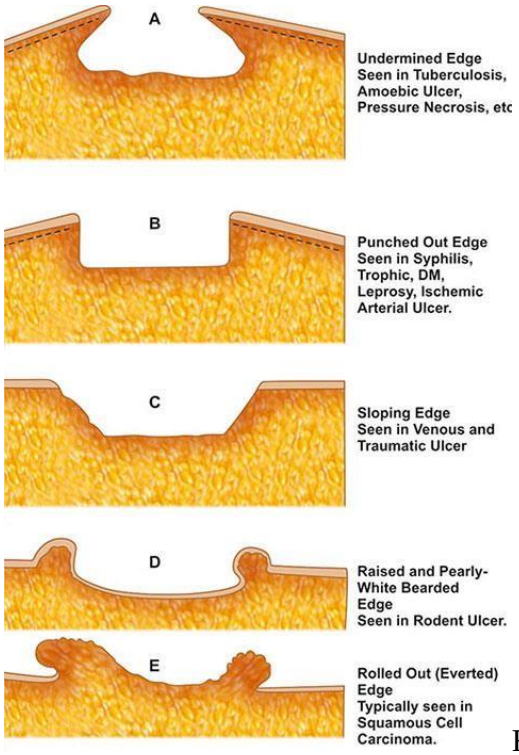


Figure 11

ULCER PATHOPHYSIOLOGY

It consistes of 3 phases :-

1. Extension Phase :- Floor – covered with exudates with purulent discharge and infuriated base
2. Transition phase :- Prepares for healing. Induration diminishes and Discharge becomes more serous. Granulation tissue appears in this phase.
3. Repair phase :- Granulation →Fibrous tissue →Scar. Epithelium extends from the healing edges to floor (1mm/day)

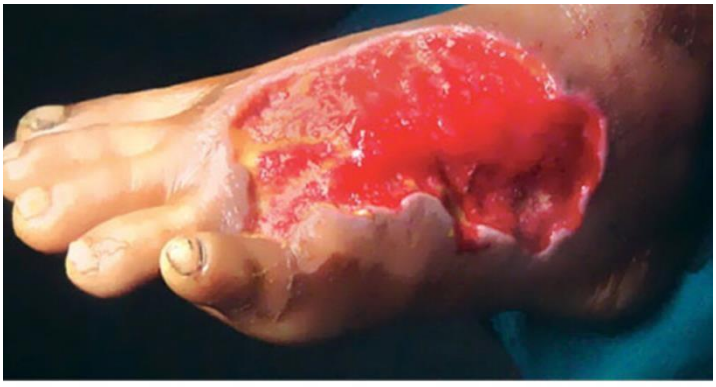
ULCER CLASSIFICATION

- Clinical classification and
- Pathological classification .

CLINICALLY, an ulcer can be of 3 types.—

(a) Spreading ulcer, when there is no evidence of granulation tissue, the skin around the ulcer is inflammatory, and the floor is covered in a large amount of foul-smelling slough. The edge is inflamed, swollen, and fagged. Lymph nodes that drain fluid are bloated, painful, inflamed, and may even become suppurated when abscesses form.

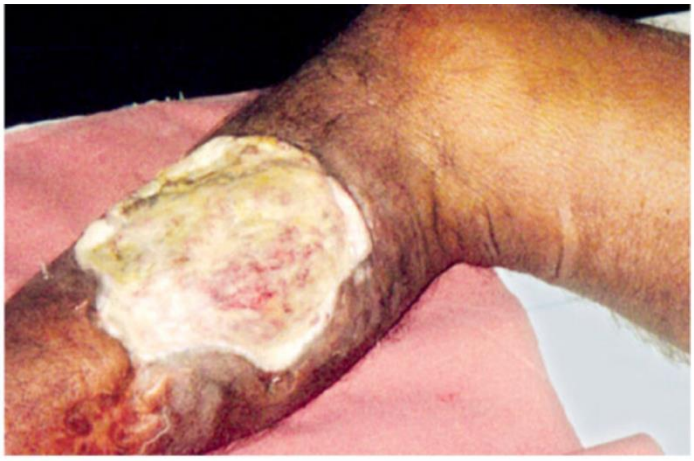
(b) A healing ulcer is one that is healing. Healthy granulation tissue that is pink or reddish in colour covers the floor. The margin has bluish hue with developing skin whereas the edge is crimson with granulation.. The discharge is serous and scanty.



Healing ulcer with healthy granulation tissue

Figure 12

(c) Chronic ulcer signifies that there is no indication that the ulcer will heal. The floor having pale granulation tissue. The classic wash-leather slough that is an example of this type occasionally appears in gummatous ulcers. Scant or no discharge is present. Along with the border and surrounding skin, the base has significant induration.



Callous ulcer in the leg. Note the slough on the surface of ulcer with no signs of healing

Figure 13

PATHOLOGICALLY, the ulcers can be classified into :

(A) Other non specific ulcers.— They are of following types:

(1) Traumatic ulcer — :

(i) mechanical- e.gulcer in the oral cavity due to sharp tooth, pressure ulcer due to splints etc. or

(ii) Ulcer due to electrical injuries

(iii) Chemical burns and caustic injury.

(2) Arterial — Thromboangitans obliterans, atherosclerosis, primary and secondary Raynaud's disease etc.

(3) Venous — e.g. varicose ulcer .

(4) Trophic ulcer/neurogenic ulcer — e.g. decubitus ulcer.

(5) Malnutrition associated e.g. tropical ulcers, It is seen commonly in tropical countries. Infestation by Bacteroides species following a small trauma.

(6) Ulcers can also be associated with certain co morbid conditions like gout, diabetes, anaemia, avitaminosis, erythrocyanosis frigida, rheumatoid arthritis etc. A diabetic ulcer can form from a small wound to a glucose-loaded tissue, but it can also happen from ischaemia brought on by diabetic atherosclerosis and diabetic neuropathy.

(7) There are few other kinds of ulcer e.g. Bazin's ulcer, Martorell's ulcer etc. Fat teenage girls are more likely to develop Bazin's ulcer, which starts as indolent sores and purplish nodules on the calves. People with hypertension are more likely to develop Martorell's or hypertensive ulcers. Here patches of necrosis are observed initially . It cannot be classified under arterial group as it is not generally associated with atherosclerosis.

(B) Specific ulcers — e.g. Meleney's ulcers, actinomycotic ulcer, tuberculous ulcer.

(C) Malignant ulcers — e.g. Epithelioma, Marjolin's ulcer, rodent ulcer and malignant melanoma

LOWER LIMB ULCERS[29]

Ulcers of the lower limb are one of the commenst issues encountered by surgeons. Few ulcers commonly seen are as follows:

Venous ulcer— Venous ulcers are mostly brought on by aberrant venous hypertension in the lower leg. Additionally used as synonyms for venous ulcers are the terms "varicose ulcer," or "post thrombotic ulcer," and "gravitational ulcer." When the calf pump and the primary deep veins are healthy, even the tiniest movements can empty the superficial veins and lower the superficial venous pressure. The ankle perforating veins serve as the

primary venous drainage pathway for the ankle skin when it is erect. The damaged valves of this vein will result in localised venous hypertension. This condition is made worse by a large deep vein blockage. Ankle venous hypertension is mostly caused by the post-canalization of the thrombosed deep veins, which results in the loss of the deep veins' valves. One of the grave complication of venous ulcer is Marjolin's ulcer.

The majority of venous ulcers develop after several years of venous illness, therefore patients are often between the ages of 40 and 60. Women experience it much more frequently than men. Before a venous ulcer forms, the skin is uncomfortable and painful, there is pigmentation, and there may be eczema. The ulcer initially hurts, but as it progresses and becomes chronic, it stops hurting.

Venous ulcers are situated at the gaiter's zone mostly along the medial aspect of ankle. Any size and shape of venous ulcer is possible. The edge is purple-blue in colour and slopes. The developing epithelium has a narrow, blue border. A light-colored granulation tissue makes up the floor. This ulcer seldom enters the deep fascia and is typically superficial and flat. Seropurulent discharge with bloody tinge.

The base adherent to the underlying structures. The surrounding area shows signs of chronic venous hypertension — pigmentation, induration and tenderness. There may be previous scars present. Varicose veins rarely seen in the proximal limb. In case of superadded infection, regional lymph nodes will be enlarged.

When the ulcer edge of long standing nature becomes raised and everted, a suspicion of marjolins ulcer be considered and biopsy should always be taken to confirm malignancy.

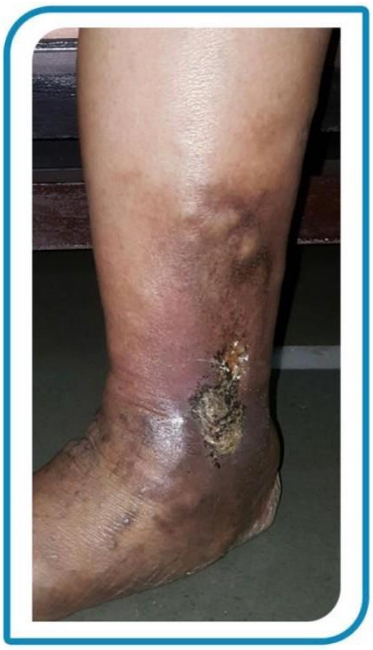


Figure 14:Venous ulcer

Arterial Ulcer— These are less commonly seen . Ulcers are due to ischemic changes in peripheral arterial disease. Elderly males with peripheral vessel disease commonly present with arterial ulcer. In contrast to venous ulcers, the ulcers frequently punch out, cause destruction of underlying tissue exposing the tendons. History of intermittent claudication with pregangrenous changes are diagnostic often.

Ulcer from congenital arteriovenous fistula- These are rare in occurence. This ulcer's special characteristics include its early onset and the presence of venous invasion in its vicinity.

Erythrocyanoid ulcer (Bazin's ulcer)— They are associated with "erythrocyanosis frigida", which is an exclusively seen in young women.The predisposing variables are an abnormal quantity of subcutaneous fat, thick ankles, and a weak vascular supply. The posterior tibial and peroneal arteries give rise to perforating arteries that feed the lower 1/3rd lower limb with bloodThese arteries may be excessively tiny or even nonexistent in erythrocyanoid instances, leading to low-grade ischaemia over the entire ankle region. The patient discovers an abnormally high sensitivity to temperature fluctuations in the

skin of the ankles. The ankle is bluish, chilly, and frequently sensitive when it's cold outside. In warmer conditions, persistent reactive hyperaemia manifests as a hot, oedematous, painful, and swollen ankle.

The patient is much troubled by chilblain. Palpation of the leg will reveal tiny, painful nodules that are superficial and degenerate into ulcers.. These ulcers are small and multiple.

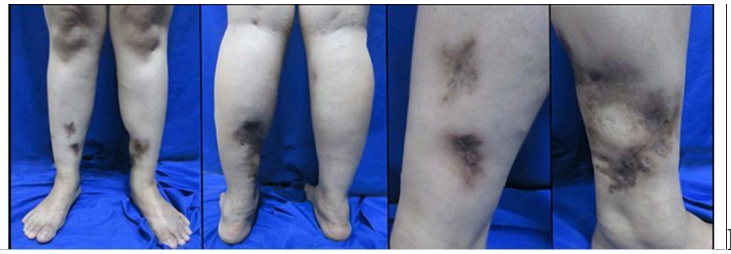
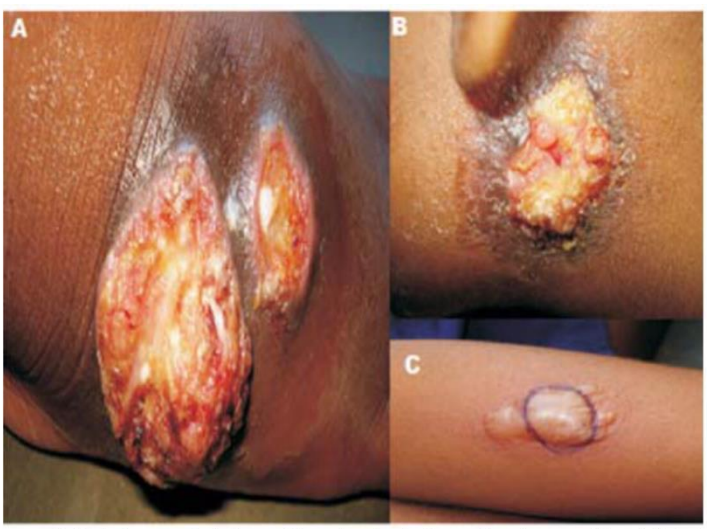


Figure 15: Bazin's ulcer

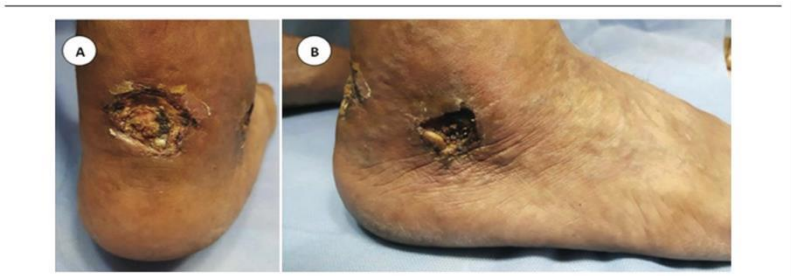
Gummatous ulcer - Ulcer of this type is seen in tertiary syphilis cases.These ulcers occur due to endarteritis, necrosis and fibrosis and common over the subcutaneous bones, in the scrotum in relation to the testis, upper part of the leg etc. There is no pain or tenderness . Due to the early closure of the lymphatics by the perivascular inflammatory reaction, lymph nodes are rarely implicated unless they are subsequently infected. The W.R. and Kahn tests are successful. Punched out edges with wash leathery slough in the floor is characteristic.



Multiple deep seated ulcers with undermined edge on the dorsum of the right foot (A), and left retro auricular area (B); Highly reactive mantoux test(C).

Figure 16

Martorell's ulcer (hypertensive ulcer)— Its often related to senile atherosclerosis. On the rear or outside side of the calf, a small patch of skin may abruptly become necrotic and peel off, with punched-out ulcer that descends to the deep underlying tissues.It may be bilateral. It is remarkable that all foot pulses on the periphery are often felt.



Initial presentation: two ulcers of two- and four-cm diameter overlying the tendo-Achilles and lateral malleolus, respectively. Both ulcers had necrotic bases with hemorrhagic crust and sharp edges. (a) Posterior view. (b) Lateral view.

Figure 17

Infective ulcers— Like staphylococcus aureus ulcer, which when occurs causes redish, scabby and more than one sores on the leg. This kind generally always results from ongoing reinfection brought on by dirty contaminated dressings and habits. Anaemia and poor nutrition are the predisposing factors. Directly above sheen is a "footballer's ulcer" brought on by staphylococcal infection and repetitive trauma. If left untreated, the ulcers persist and attach to the bone.

Meleney's ulcer— A previously present chronic ulver may convert to meleney's or may

occur de novo (in ulcerative colitis) . It is caused by the symbiotic relationship between staphylococcus aureus and micro-aerophilic non-haemolytic streptococci. Burrowing, or undermining, of the ulcer with significant amounts of granulation tissue on the floor, is the most significant clinical feature. A deep purple zone encircles the erythematous outer zone that surrounds it. These ulcers cause the patient to become toxemic, are extremely painful and tender, have a propensity to spread, and get worse if left untreated.

Ulcers complicating various diseases— These ulcers could appear out of nowhere or in venous ulcers that suddenly worsen and become more painful. Often associated to gross anaemia, polycythemia, Rheumatoid arthritis, osteitis deformans, ulcerative colitis etc

A nodule first develops into an ulcer, which can vary in size, become punched-out, shallow (remain in the subcutaneous tissue), painful, indurated-free, and slow to heal.

The convexity of the anteriorly bowed tibia is where most secondary osteitis deformans (Paget's disease) ulcers are located, with the margins tightly adhering to the bone that forms its foundation.

Tropical ulcer— Bacteroides Fusiformis following a trivial trauma or insectbites commonaly causes tropical ulcers. The lesion begins as a papule-pustule with an indurated and inflammatory zone surrounding it. At this stage of acute lymphadenitis, pain is a crucial symptom. These pustules rupture in two to three days, resulting in ulcers with elevated and undercut margins. Significant feature is copious serosanguineous discharge accompanied by significant discomfort. The ulcer grows stubborn and takes months or even years to heal. Pigmented scar is often found which seems like a parchment membrane. These ulcers may sometimes turn to squamous cell carcinoma.


Figure 18

Yaws— Treponema Pertenue is the causative agent here. Lower limb ulcers in persons mostly who walk barefooted. It is characteristically painless and heals with scarring.


Figure 19

INVESTIGATIONS :

1. Routine blood examination— e.g. total count and differential count of W.B.C., haemoglobin, R.B.C. count, E.S.R. should always be done in a patient with an ulcer.

Random blood sugar to exclude diabetes.

In ulcers associated with tuberculosis E.S.R. will be high. The patient may be anaemic.

2. Examination of the Urine — particularly sugar estimation, to exclude diabetes, is important.

3. Culture from ulcer discharge is particularly important in inflamed and spreading ulcers.

4. Chest X-ray — Finding any major lung focus is crucial in tuberculous ulcers. In the case of malignant ulcers, it's crucial to exclude out lung metastases.

5. Biopsy — in suspected malignancy,it is very important. The biopsy is often taken at the edge of ulcer along with the healthy surrounding area. After that, the biopsy sample is histologically evaluated to determine the tumor's kind, degree of invasiveness, and if it is differentiated or anaplastic.

6. X-ray is done to look for nearby bony involvement. ‘Sabre tibia' is seen in gummatous ulcer.

7. Contrast Radiography— CT angiogram and venous doppler

SURGICAL DRESSING[15]

Lister developed antiseptic dressings in 1867 by soaking gauze and lint in carbolic acid. Creating the appropriate environment for wound healing is the fundamental goal of wound dressings. To ensure that the wound heals as effectively as possible, the dressing should support the significant changes occurring during healing.

. Goals of Wound Dressings

To aid wound healing and maintain moisture

Comfortability

Pain reduction

Odour reduction

Non-allergenic , non-irritating in nature

Allow gaseous diffusion

Safety

Cost-effective

Convenient

. PRINCIPLES

1. Applying a bandage to an ulcer imitates the epithelium's barrier function and stops additional harm. Application of compression also promotes hemostasis and restricts edoema.

2. By regulating the amount of moisture and oxygen around the wound, dressing material occlusion promotes healing.

3. It also enables gas and water vapour to be transferred through wound surface to exterior.

4. Skin are impacted by occlusion, and it has been demonstrated that open wounds experience greater inflammation and necrosis than covered wounds.

5. Occlusion also promotes the production of dermal collagen, inhibits the migration of epithelial cells, and prevents tissue desiccation.

6. Occlusion is not preffered in infected and wounds with heavy discharge because it may encourage bacterial growth [(14).]

WOUND CLEANSING SOLUTIONS

According to current theories of wound healing methods, the wound dressing should ideally foster epidermal regeneration while acting as an infection barrier.

According to the theory, dressings gain their osmotic capacity by water evaporation, which renders them hypertonic and gives them a moist environment to maintain their physiologically appropriate and isotonic qualities.[51]

Perhaps the most important aspect of managing acute and chronic wounds is proper cleansing to produce a wound environment ideal for healing. Cleaning techniques can vary amongst different healthcare professionals, organisations, and facilities, and frequently are based on unique experiences and personal preferences (52). There are many different cleaning agents, and choosing one should be determined on how well it cleans while avoiding cytotoxicity.

Cleaning the wound lowers the chance of infection and enhances the environment for healing. It loosens and removes bacteria, exudate, purulent debris, and any remaining topical chemicals from earlier dressings. Most wounds need to be cleaned both before and after receiving a new dressing.

Goals:

• To determine the most correct mode of cleansing.

• Reduce chemical irritation.

• Reduce mechanical trauma

PROPERTIES OF AN IDEAL DRESSING

• Free from bacteria

• Allowing exchange of gas

• Absorbs exudate

• Not adherent to wound

• Free from toxins and fibre

• Not causing allergy

• Maintain hemostasis.

• Comfortable to the patient

• Pocket friendly to the patient.

Properties of wound care solutions

1. Normal saline[48]:

It being isotonic to blood and offering a moist environment for healing of wound, these dressings are utilised in wound healing. Open wounds can benefit from the use of 0.9% normal saline dressings,hence routinely employed with acknowledged success in clinical settings [46].

They are cost effective and easy in application. The normal saline dressing's hypertonicity creates an osmotic gradient that allows wound fluid to be absorbed, which increases its efficiency as a wound dressing.

However,it lacks surfactants,hence less effective in removing debris , bacteria from wound and its surroundings. Additionally, there are no preservatives to stop bacteria development in normal saline.

However, for more serious wounds, normal saline might not be strong enough:

- Because normal saline washes away physical substances contained within the wound, it doesn't effectively control bioburden.
- Normal saline isn't always strong enough for infected or necrotic wounds, as it doesn't function as an antibacterial agent. Nor does it include surfactants, which assist with physically removing bacteria and more stubborn debris.
- A container of saline may start attracting bacteria not long after it is opened. Patients and their caretakers are recommended to dispose of the solution within 24 hours after opening.

2.Povidone Iodine[47]:

Broad spectrum antibiotic effective against a number of infections, including Staphylococcus aureus.

Cytotoxic to normal tissues.

Skin dryness and discoloration is often noticed.

Because they skin irritation around the lesion, the AHCPR recommendations ban the use of antiseptic solutions like povidone iodine, hydrogen peroxide, or sodium hypochlorite.

At amounts safe for healing, they are ineffective at killing bacteria.

3.Hydrogen Peroxide:

When administered adequately, the effervescent washing aid in dislodging debris from wound surface.

Cytotoxic to healthy cells and granulating tissues.

If used at full potency, irrigation with ordinary saline is advised after usage.

Incapable of eliminating germs.

Avoid applying to wounds that have tracts of sinus.

4. Sodium Hypochlorite or Dakin's Solution:

•It ismharmful against healthy and granulation tissue.

• Has cidal effect against most micro organisms.

• Controls infection and clears necrotic tissue.

• Reduces odour.

• Not to be used for longer than a week.

5. **Alginates**[15] Promotes the debridement of slough; is hemostatic in nature due to the release of calcium ions. It can absorb 20 times its own weight and is both incredibly absorbent and biodegradable. Brown seaweed-based; useful for moist wounds or cavities.

6. Films[15]

• Bacteria and liquid resistant

•Maintains moisture around the wound.

•Allows water and air permeation.

•Doesn't act as absorbent.

•Used as both 1 degree and 2nd degree dressings

7. Foam dressing[15]

• Polyurethane and silicone formulations.

• Comes both as adhesive and non adhesive formulations.

• Low to moderate exudate this changes depending on the MVTR (ability to transmit water vapour to exterior).

• Contact dermatitis is a known adverse effect.

8. Hydrocolloid dressings[15]

• One of the 1st "modern dressings"

• Encourages the production of healthy granulation tissue, debridement, and moist wound healing.

• Waterproof and obstructive

• Wounds with low to moderate exudate and restricted absorption.

• If applied to infected wounds, exercise caution.

• On removal, there can be a faint odour.

9. Hydrofibre[15]

Although it functions similarly to alginate, this substance is not one.

•Has the same building blocks as hydrocolloids.

•Transforms into soft gel after absorbing wound fluid.

• Extremely absorbent

• Encourages debridement

•Absorbs and holds onto exudate and germs.

10. Hydrogel dressings[15]

• Have a high percentage of water .

• To offer gel-forming characteristics, starch molecules (carboxy methyl cellulose) are combined.

• Encourages the debridement of slough and eschar.

Hydrogel sheets could lessen pain.

Use with caution on infected wounds; additional dressing is necessary.

11. Contact wound layers

•These dressings are non- adherent and done for superficial wounds with granulation tissue.

•Avoids injury to the wound bed.

•Viscose fabric that is ultra-knitted.

Atruaman is triglyceride-impregnated.

•Mepitel is a soft silicone contact layer for skin that is delicate.

12. Absorbent dressings[15]

Some dressings may bind germs to reduce the load of bacteria and infection in severely oozing wounds.

13. Antimicrobials

•Cadexamor iodine – used judiciously in thyroid patients having iodine sensitivity and renal compromise.

•Aqucel AG -1.2%

•Actisorb silver – with charcoal for odour

14. PHMB (Polyhexamethylene biguanide) dressings

•Surfacactant and hence wound cleanser

•The hydro gel form helps debridement

•Contains betadine which has bactericidal effect.

15. Honey dressing[42]

•Medical-grade Manuka honey impregnated non-adherent alginate

•Makes debridement easier

•Slows down bacterial growth

•Decreases odour.

HISTORY-Silver being used as antibacterial

Silver was 1st used in 1800s as an antibacterial agent but its use declined soon after introduction of antibiotic agents however its use re emerged extensively in the last 20 years.

Silver has been used to disinfect liquids and water storage since the beginning of time. Silver coins were employed for this by early Americans and ancient Greeks (7). Silver was used to cure leg ulcers, acne, venereal infections, and epilepsy before the 1800s. Silver pencils were used for the debridement of warts and ulcers. To hasten healing and lower post-operative infections, silver foil was wrapped around surgical incisions. (4;6;7).

In order to prevent postpartum eye infections, 1% silver nitrate solution was injected into conjuntiva sacs in the late 19th century. Moyer and Monafo introduced silver nitrate 0.5% solution in the late 1960s for the management of burns. (4;6;7).

However, silver nitrate dressings require a lot of work because they must be reapplied or re-moisturized every second hourly. The amount and rate of free silver discharged into the woundbed were found to be related to the effectiveness of silver as an antimicrobial. (19).

Fox introduced silversulfadiazine cream for the treatment of burn wounds in the late 1960s. By significantly lowering the prevalence of burn wound infections, this fundamentally changed how burn wounds are managed.

Silversulfadiazine cream has a brief duration of action, weak burn eschar penetration, and generates a pseudo-eschar.

ACTION OF SILVER ON WOUND

Silver is broad-spectrum antibiotic with antiseptic, antibacterial, and anti-inflammatory effects. (13;17; 4,6; 19;23; 7).

In soluble forms, such as Ag+ or Ag0 clusters, silver is physiologically active. In addition to other ionic silver compounds, silver nitrate and silver sulfadiazine also contain the ionic form of silver, known as Ag+. Ag0, or uncharged metallic silver, is present in nanocrystalline silver(6).

Free silver cations have a strong antibacterial impact that kills microorganisms right away by preventing cellular respiration and impairing bacterial cell membrane function. This happens when silver ions and tissue protein come together, changing the way bacterial cell membranes are built and ultimately causing cell death. Additionally, bacterial DNA and RNA are bound and denatured by silver cations, which hinders cell division. (33; 39; Yin et al 1999; 4; 19;31;32;6).

PROPERTIES AND ACTIONS OF NANOCRYSTALLINE SILVER

Nanotechnology is used to create clusters of incredibly tiny, highly reactive silver particles from nanocrystalline silver (28). Smaller silver particle size enhances bioactivity and silver solubility as more wound surface area comes into touch with the metal.

The growth of nanocrystals that are each 15 nanometers broad and contain between 30 and 50 atoms occurs when an electric current is delivered into a negative pressure chamber with an argon anode. The argon ions knock off the silver atoms as they move towards the substrate to be coated. A meta-stable, high-energy version of elemental silver is created as a result of this change in the crystal's lattice structure. (6).

When nanocrystalline silver is applied to a wound, it releases clusters of extremely reactive silver cations that can number up to 100 parts per million. These cations destroy cell

membranes, inactivate bacterial cell DNA, and bind insoluble complexes in microorganisms. (3;22; 12; 19; 6).

Compared to other forms of silver, such as 0.5% silver nitrate or silversulfadiazine, nano silver releases 30 times fewer silver cations. But the amount of silver released is greater, and it is released continuously. (6).

According to research, treatments containing sustained-release silver have a bactericidal effect that effectively manages odour and exudate, lowering the risk of colonisation and preventing infection. (3;22; 12; 19;28).

WOUND ENVIRONMENT

Keeping microbes under control in a wound environment aids in wound healing. Microorganisms, such as bacteria or fungus, are prevalent in chronic wounds and can quickly contaminate and infect an acute wound, substantially slowing wound healing.

The wound-healing process can be affected by high bacterial counts, multi-resistant organisms, and bacterial biofilms, especially in chronic wounds (34). By competing with host cells for nutrition and oxygen and producing waste products that are harmful to host cells, bacteria slow the healing of wounds.

Increased blood cytokines, increased matrix metalloproteinase, and decreased growth factors are all symptoms of bacterial wound infection and can have a negative impact on how quickly a wound heals. Local wound infection results in tissue death, an expansion of the wound, wound hypoxia, and vascular obstruction, all of which slow down the healing process. (41).

On wound surfaces, there exist complex bacterial colonies called "biofilms" that are embedded in a polysaccharide matrix and function as a single organism in their own habitat (34; 41). Up to 1000 times greater resistance to conventional antibiotics can be found in

bacterial biofilms. In critically colonised wounds, biofilms are common and can lead to wound infection. (34;41).

When bacterial cells create and secrete a wide range of enzymes and toxins onto the wound, this is known as a wound bioburden. Complex wounds have 104 colony forming units (cfu)/g or cm2, but an infected wound has 105 cfu/g or cm2 of bacteria (40). By removing non-viable tissue through debridement or by applying an antimicrobial dressing like a sustained released silver dressing, this bacterial burden can be decreased.

In an effort to provide a more favourable environment for wound healing by lowering the level of wound bioburden, sustained released silver dressings have become more widely utilised to treat both chronic and acute wounds in recent years. (44).

IN VITRO EVIDENCE OF NANO CRYSTALLINE SILVER

Wright et al. evaluated the bactericidal efficacies of 3 different topical silver applications—silver nitrate solution, silver sulfadiazine cream, and nanocrystalline silver—against a control dressing to treat 11 clinical isolates of antibiotic-resistant bacteria.

The organisms were seeded onto each dressing, incubated for 30 minutes, and then rinsed with a recovery solution. The organism survival rate was then determined by culturing the recovered organisms. Every trial dressing showed the capacity to lower the amount of germs that were still alive. The silver nitrate solution was the least effective, and the nanocrystalline dressing was the most effective.

The researchers came to the conclusion that silver was effective at killing the tested strains of bacteria that were resistant to antibiotics. Compared to the other trial dressings, nanocrystalline silver was found to be more efficient against a wider spectrum of bacteria and to kill the tested bacteria more quickly.(39)

When Yin et al. tested the antibacterial efficacy of nanocrystalline silver against five clinically significant bacteria, they also tested the efficacy of silver nitrate solution, silver sulfadiazine cream, and mafenide acetate. They discovered that nanocrystalline silver was more rapid in delivering silver cations and reduced bacteria more quickly than the other experimental dressings.

All forms of silver products had a similar killing mechanism, however nano-silver killed more quickly because bacteria absorbed silver more quickly in nano-silver samples. (45).

In 1999, Wright et al. investigated the topical treatments' in vitro fungicidal effectiveness. The survival rate of the inoculated fungi was assessed after incubation on mafenide acetate, silver nitrate, silversulfadiazine cream, and nanocrystalline silver dressings. It was discovered that all of the antimicrobial treatments were effective against fungus. The fastest death rate and broadest spectrum activity against fungus were obtained by the nanocrystalline dressing. (39).

In a different in vitro experiment, Thomas et al. compared 4 silver-containing dressings, including ActisorbTM, Silver220TM, AvanceTM, and Contreet-HTM, and found that nanocrystalline silver had more rapid antimicrobial activity against both Gram positive and Gram negative bacteria as well as a yeast.

The same researchers ran a second trial that year using a wider range of silver items. Once more, they showed that the antibacterial activity of silver compounds varied; some had little to no impact on the studied microbes. At each reading interval, nanocrystalline silver destroyed 99.9% of methicillin-resistant Staphylococcus aureus. (32).

Fraser tested the effectiveness of Nanocrystalline silver and silversulfadiazine cream against 8 typical burn wound infections in an in vitro study in 2003. They showed that Nanocrystalline silver was less effective than silversulfadiazine cream at killing all tested organisms. (9).

The following year, the same researcher carried out a second in vitro experiment to examine the cytotoxicity of silversulfadiazine cream and Nanocrystalline silver applied to the centres of culture plates seeded with keratinocytes, incubated for seven hours, and then read for keratinocyte survival rates on the culture medium plates. Nanosilver was found to be less harmful to keratinocytes than Silversulfadiazine cream. (10).

In a different in vitro investigation, Poon et colleagues investigated how silver on local application affects keratinocytes and fibroblasts. The two experimental dressings were silver nitrate solution and silver nanocrystals. They showed that silver was poisonous to germs as well as skin cells like keratinocytes, fibroblasts, and fibroblasts. They advised against using silver products in areas with exposed quickly reproducing keratinocytes, such as donor sites, superficial partial thickness wounds, and applications using undifferentiated cultured keratinocytes. (24).

In conclusion, the research shows that employing nanocrystalline silver to treat wounds is effective, according to in vitro evidence. When used as an antibacterial, nanocrystalline silver is effective against the majority of common bacterial strains, including multi-resistant strains and fungus spores. According to in vitro evidence, nanocrystalline silver has a wider antibacterial spectrum activity, is toxic to keratinocytes and fibroblasts, can be utilised as a protective covering over skin grafts, and has the best killing rates for a variety of microscopic organisms.

EVIDENCE OF ANIMAL STUDIES FROM NANOCRYSTALLINE SILVER

In a porcine model of contaminated wounds, Wright et al. in 2002 investigated early healing events and the effectiveness of nanocrystalline silver on the levels of matrix metalloproteinase, cell apoptosis, and healing. They discovered that nanocrystalline silver promoted rapid wound healing in the initial days following injury and that the proteolytic environment of wounds treated with nanocrystalline silver was altered by the reduction of matrix metalloproteinase.

Matrix metalloproteinase levels have been found to be excessively high and pro-inflammatory in chronic ulcers compared to acute wounds. This might be a factor in some wounds' inability to heal. In wounds treated with silver that was not nanocrystalline, cellular apoptosis occurred more frequently. According to this theory, nanocrystalline silver plays a part in changing the inflammatory processes in wounds and promoting the initial stages of wound healing. (43).

In an animal model of wound healing, the same scientists in 2003 contrasted nanocrystalline silver and a gauze dressing impregnated with polyhexamethylene biguanide. They discovered that compared to wounds dressed with non-nanocrystalline silver products, those treated with

nanocrystalline silver products advanced to full granulation more quickly and had lower bacterial bioburden levels. The scientists came to the conclusion that a dressing's antibacterial properties alone are insufficient to facilitate wound healing. The polyhexamethlyene biguanide-infused gauze dressing slowed the healing process and extended the inflammatory response. (44).

In full thickness burn wounds infected with pseudomonas in rats, Ulkur et al. in 2004 compared Nanocrystalline silver, chlorhexidine acetate, and silversulfadiazine cream as topical antibacterials. Due to the seldom need for dressing changes, they came to the conclusion that nanocrystalline silver might be ideal for dressing. (35).

The following year, the same authors evaluated the topical antibacterial effects of Nanocrystalline silver, chlorhexidine acetate 0.5%, and fusidic acid 2% in methicillin-resistant Staphylococci-contaminated full thickness rat burn wounds. The most effective treatment for burn wounds contaminated with methicillin-resistant Staphylococcus aureus was fusidic acid, although Nanocrystalline silver was favoured since it might reduce the number of times that dressings needed to be changed. (36).

Supp et al. investigated the cytoxicity and antibacterial efficacy of Nanocrystalline silver in 2005 in order to minimise microbial contamination in cultured skin substitutes transplanted onto anthymic mice. After grafting with cultured skin substitutes for 1, 2, 3, and 4 weeks, the cytoxocity of nanocrystalline silver was evaluated. They discovered that polluted wounds healed comparably to controls under Nanocrystalline silver therapy. As a result of these findings, it was suggested that it might be utilised as a protective dressing to reduce environmental contamination of cultured skin substitutes for wound-controlling organisms. (30).

In conclusion, although there are few research on nanocrystalline silver in animals, the literature that has been examined indicates that it may play a role in modifying inflammatory processes in wounds and promoting the initial stages of wound healing. Given that it reduces the number of times a dressing needs to be changed, there is evidence to support nanosilver's effectiveness as an antimicrobial dressing.

EVIDENCE FROM HUMAN STUDIES ON NANOCRYSTALLINE SILVER

In 1998, Tredget performed a matched-paired, randomised study on 30 patients to assess the effectiveness and safety of nanocrystalline silver for the treatment of burn wounds. The results showed that while the pain levels of the nanocrystalline silver-treated patients were initially lower, they became comparable to those of the silver nitrate group of patients after two hours. Additionally, they discovered that those who received treatment with Nanocrystalline silver saw fewer dressing changes and instances of wound infection. (33).

Voight presented case studies of six individuals who received Nanocrystalline silver treatment for venous ulcers. They claimed that these patients' wounds had healed, with one having a 5-month-old ulcer being treated with Nanocrystalline silver in 194 days and another having a 5-week-old ulcer being treated with Nanocrystalline silver in 27 days.

In a different case study, Voight showed the effects of nanocrystalline silver on four patients who had debicutus ulcers; one ulcer that had been present for 24 months was cured in 27 days, while another that had been present for two weeks was healed in 14 days. In every case where nanocrystalline silver was used, they showed a decrease in exudate fluid quantities. The usage of Nanocrystalline silver dressing was studied in a multi-centered (41 centres) survey by the same authors. They found that Integra, a dermal regeneration template for full thickness burns repair, was covered with Nanocrystalline silver in up to 52% of the centres surveyed (61% of which used the material). Additionally, they stated that 4.8% of the respondents utilised Nanocrystalline silver as their primary dressing. They came to the conclusion that nanocrystalline silver is affordable, improves wound healing, and can be used on all kinds of wounds. (38).

In a single centre, open-label, unblinded pilot trial conducted in 2002 by Kirshner et al., 11 extended-care facility outpatients or residents with chronic wounds of mixed aetiology were used to explore the effect of silver in wound healing. Every wound had a history of at least three months, and in the three weeks before the research, there had been no shrinkage of the wound size. All of the patients received Nanocrystalline silver treatment, and after the first week, the dressings were changed every other day. All used dressings were saved for fluid collection and analysis. The authors discovered a decrease in matrix metalloproteinase activity in the first two days of treatment in eight individuals who finished the research. This

demonstrated that if the nanocrystalline silver dressing is used consistently, the changed matrix metalloproteinase activity may persist. (16).

Demling and Desanti evaluated the effects of XeroformTM (3% Bismuth tribromophenate) and Nanocrystalline silver as dressings over mesh skin grafts in 2005. Twenty patients were treated with Nanocrystalline silver on one wound and XeroformTM with 0.01% neomycin and polymyxin on the other lesion. Each patient had two regions of mesh skin grafts. Every three days, wounds were examined, and wound swabs were taken. They discovered that compared to conventional XeroformTM dressings, Nanocrystalline silver significantly enhanced the pace of wound closure. (5).

In 2004, Dunn presented data from the 2003 European Burns Association convention on the positive results achieved by numerous European practitioners using Nanocrystalline silver on burn victims. Children with partial to full thickness burns received nanocrystalline silver dressings. Beyond its antibacterial properties. Wounds treated with nanocrystalline silver typically get better and heal on their own or in conjunction with surgical procedures. There have been reports of decreased pain levels, dressing changes more infrequently, wound exudate, and surgical procedures. (6).

Following dressing changes, 14 burn patients were studied by Varas et al in 2005. Patients who received either Nanocrystalline Silver or Silversulfadiazine Cream dressings at random for their two burn regions experienced reduced discomfort from the Nanocrystalline Silver-treated wounds. (37).

Fong et al. examined the use of Nanocrystalline silver in reducing the incidence of early burn wound infection and its economic effectiveness in two comparative patient care audits and a historically controlled matched paired comparison in 2005. Patient care audits showed that the silver sulfadiazine cream treated group (control group) had a 55% infection rate whereas the nanocrystalline silver treated group (treatment group) had a much reduced infection rate (5.2%).

Additionally, they noted lower pain levels in the Nanocrystalline Silver patients, and based on staff members' subjective observations of the ActicoatTM group of patients as well as the Nanocrystalline Silver and silversulfadiazine group of patient, it appears that the

Nanocrystalline silver patients felt better overall due to lower pain levels and fewer dressing changes. (8).

In a 2005 open pilot research, Sibbald found that an ionised silver dressing with sustained nanocrystalline silver release (Acticoat 7) can reduce bacterial burden and speed wound healing in venous ulcers that aren't healing as quickly as they should (26). The anti-inflammatory activity of prolonged released nanocrystalline silver in the treatment of chronic venous leg ulcers was reported by the same authors in the same study. Nanocrystalline silver dressings have an antibacterial and permissive but selective anti-inflammatory action in reducing venous ulcer size. (27).

No wound infections or positive blood cultures were discovered during the trial period, and Rustogi et al. concluded that Nanocrystalline silver is suitable for use as a dressing for neonates after evaluating the safety and efficacy of its use in treating primary burn injuries and other skin injuries in premature neonates in 2005. (25).

In conclusion, the literature review of nanocrystalline silver used on humans for wound management suggested that nanocrystaline silver is cost-effective, decreases the incidence of burn wounds, lowers pain thresholds during dressing changes, lowers dressing change frequency, lowers matrix metalloproteinase activity, lowers wound exudate and bioburden levels, and promotes wound healing in chronic wounds. In vivo studies have shown that nanocrystalline silver is safe for skin cells, including keratinocytes and fibroblasts.

AIMS AND OBJECTIVES:

TO STUDY THE EFFICACY OF NANO-CRYSTALLINE SILVER DRESSINGS IN COMPARISON TO CONVENTIONAL NORMAL SALINE DRESSINGS IN THE MANAGEMENT OF LOWER LIMB ULCERS

Objectives:

To compare parameters like reduction in ulcer surface area, appearance of granulation tissue, reduction in slough , reduction in discharge and culture sensitivity before and after treatment.

MATERIALS AND METHODS:

Patients attending outpatient department and/or who were admitted in B.L.D.E (DU)'s Shri. B. M.Patil Medical College, Hospital and Research Centre, Vijayapura in the Department of Surgery during period of **Jan 2021–Oct 2022 with lower limb ulcers** were included in the study. A comparative interventional study was conducted with total **70 patients** with **35 patients** in each group selected **alternatively**. After undergoing a detailed clinical examination, relevant investigations, the initial wound area, presence/absence of discharge, slough , granulation tissue and culture will be recorded after debridement by measuring length x width (provided ulcer should be less than 15cm x15 cm).

Both groups will be subjected to once-daily dressings. The patients will be followed up on a daily basis for a period of 2 weeks in both groups. Results will be calculated by using the Chi Square test.

► **RESEARCH HYPOTHESIS**: Nano crystalline silver local application is beneficial in faster and effective wound healing.

REVIEW OF LITERATURE

Silver ions as small as 100 ppm were studied by Deitch in 1987, Heggars and workers in 2002, Lansdown in 2002, and Dunn in 2004. It was discovered that silver ions inhibit electron transport, inactivate microbial DNA, damage cell membranes, and form insoluble complexes with microorganisms. They showed that silver nanoparticles significantly reduce exudate and odour, which lowers the chance of colonisation and prevents infection. [3]

In 1998, "Tredget" and his colleagues performed a matched-paired, randomised research to assess the effectiveness of silver nano-crystalline particles for treating burn wounds. They discovered that these patients experienced less pain and wound infection than those treated with silver nitrate solution. [33].

In contaminated wounds of the pig model, "Wright" and his colleagues in 2002 investigated the effects of early healing and the effectiveness of silver nanoparticles on matrix metalloproteinase, cellular apoptosis, and healing. He made full-thickness incisions on the backs of pigs, inoculated them with coagulative negative strains of Staphylococci, Fusobacterium species, and Pseudomonas, and then covered them in silver nano-crystalline particles. He discovered that it facilitated quick wound healing in the initial days following injury and that matrix metalloproteinase levels were decreased. [43].

According to studies done by "Smith and Nephew" in 2003, nano-crystalline silver has a greater surface area of contact with wounds due to its smaller particle size. Additionally, it disperses highly active silver particles, enhancing silver's bioactivity and solubility. [28].

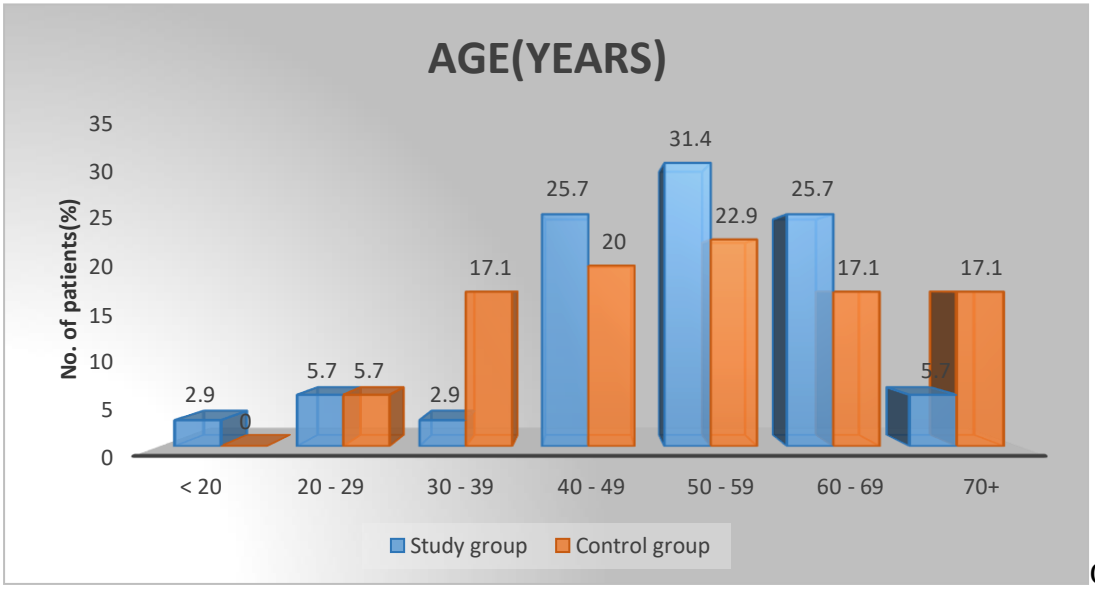
In a study on chronic ulcers, "Wright" and his colleagues discovered that matrix metalloproteinase levels are abnormally high and pro-inflammatory compared to acute wounds and chronic wounds, and that nano-crystalline silver plays a role in modifying the inflammatory events in wounds and promoting early wound healing. [43].

RESULTS

A total of 70 patients with lower limb ulcers were included in the study and alternatively divided in two groups ; Study group (Nanocrystalline group) and Control group (Normal saline group)

Table 1: Age distribution

Age(Years)	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
< 20	1	2.9	0	0		
20 - 29	2	5.7	2	5.7		
30 - 39	1	2.9	6	17.1		
40 - 49	9	25.7	7	20.0		
50 - 59	11	31.4	8	22.9		
60 - 69	9	25.7	6	17.1		
70+	2	5.7	6	17.1		
Total	35	100.0	35	100.0	7.895	0.2459



Graph 1

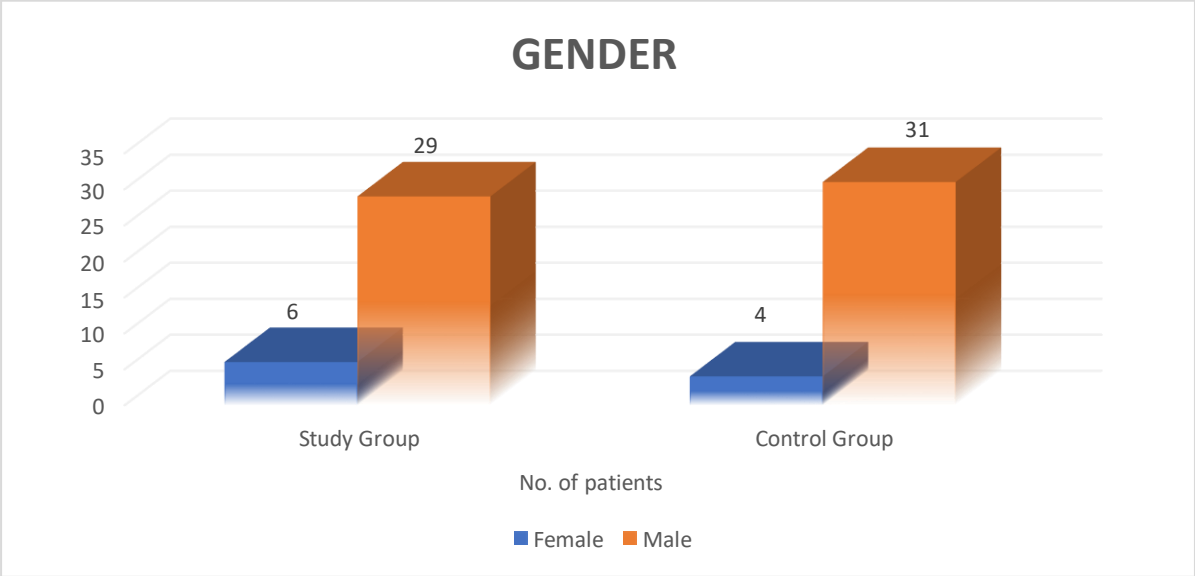
Table 1 (Mean Age)

Age(Years)	Study group		Control group		**Independent Samples t Test** T test/	P value
	Mean	Std. Deviation	Mean	Std. Deviation		
Age	52.20	13.970	53.03	15.171	0.238	P=0.813
Statitially insignificant						

Age distribution of patients was between 16-78 years of age with more percentage of the patients belonging to 50-59 years followed by 40-49, 60-69, 20-29 and 70+ years age groups in study group. In Control group more percentage of the patients belonging to 50-59 years followed by 40-49, 60-69 and 30-39 and 20-29 years age groups (Table 1, Figure 1).

Table 2 Gender(Male/Female)

Gender	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Female	6	17.1	4	11.4		
Male	29	82.9	31	88.6		
Total	35	100.0	35	100	0.4667	0.4945
						Statistically insignificant

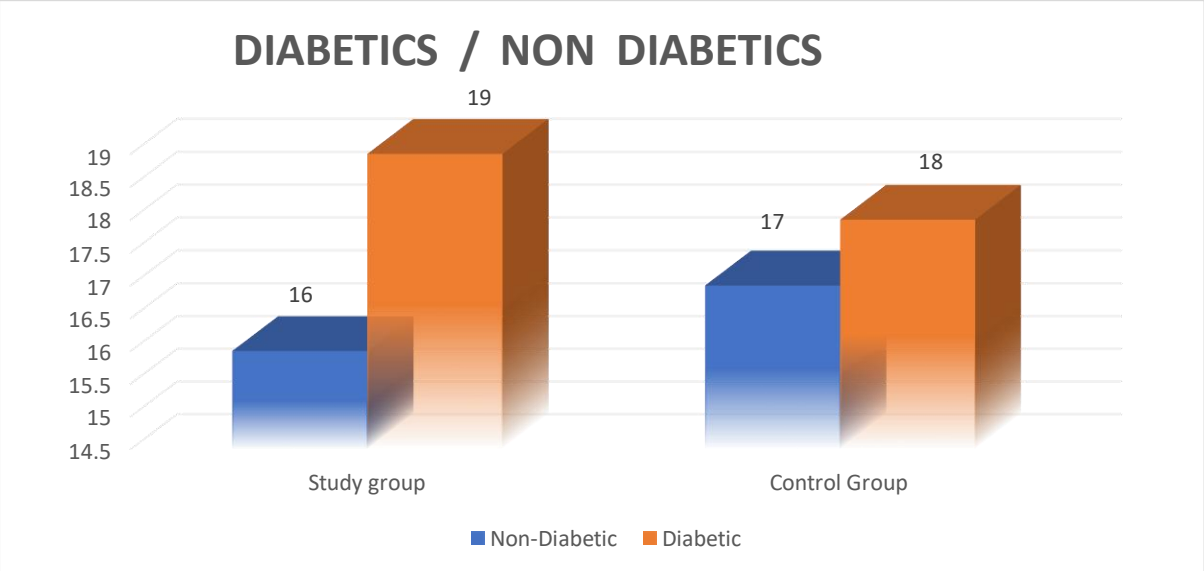


Graph 2

In the present study, the incidence of lower limb ulcer was higher in males than in females . Study group had 29(82.9%) males and 6(17.1%) females , Control Group had 31(88.6%) males and 4(11.4%). Th results were comparable in both groups and were statistically insignificant. (Table 2) (Figure 2).

Table 3: Diabetics Vs Non-Diabetics

	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Non-Diabetic	16	45.7	17	48.6		
Diabetic	19	54.3	18	51.4		
Total	35	100.0	35	100	0.0573	0.8108
						Statistically insignificant

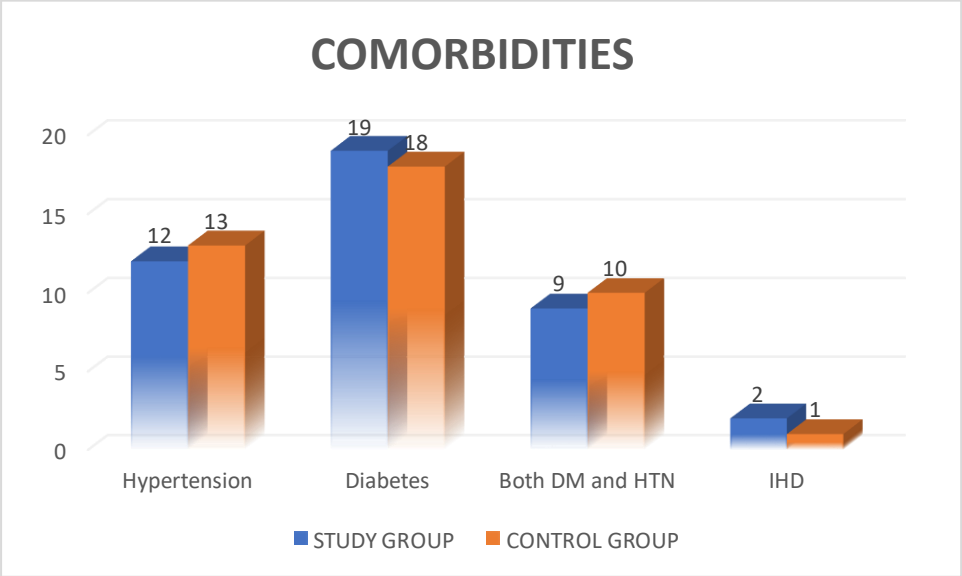


Graph 3

In the present study, the incidence of diabetics vs non-diabetics were comparable with p value being insignificant. In study group the incidence of diabetics were 16 (45.7%) and non-diabetics were 19 (54.3%). In control group the incidence of diabetics were 18 (51.4%) and non-diabetics were 17 (48.6%). (Table 3, Figure 3)

Table 4 (Comorbidities)

	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Hypertension	12	34.2	13	37.1		
Diabetic	19	54.3	18	51.4		
Both Diabetic and hypertensive	9	25.7	10	28.5		
Ischemic heart disease	2	5.7	1	2.8		
Total	35	100.0	35	100	0.0573	0.6108
						Statistically insignificant



Graph 4

In the present study comorbidities like diabetes , hypertension and ischemic heart disease were found with more incidence of diabetes. In study group the incidence of diabetes was found in 19 (54.3%) patients, incidence of hypertension was present in 13 (34.2%) patients , incidence of both diabetes and hypertension was found in 9 (25.7%) patients. In control group

the incidence of diabetes was found in 18 (81.4%) patients, incidence of hypertension was found in 13 (37.1%) patients , incidence of both diabetes and hypertension was found in 10 (28.5%) patients. (Table 4, Figure 4)

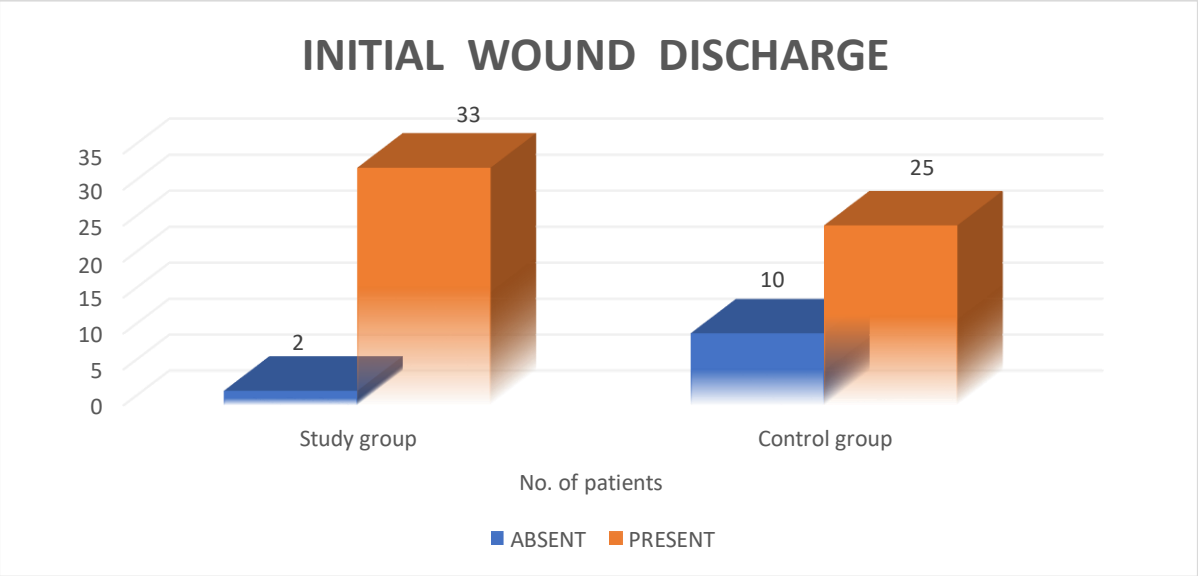
Table 5 (Average random blood glucose among diabetics)

	STUDY GROUP	CONTROL GROUP		P- value
Average Random blood sugar	226.21	190.3		
Average Hba1c levels	7.7	7.7		
Total	19	18	0.0573	0.8108
				Statistically insignificant

In the present study there were 19 diabetic patients in study group with average random blood glucose levels 226.2 mg/dl and average Hba1c levels 7.7 whereas there were 18 diabetic patients with average random blood glucose levels 190.3 mg/dl and Hba1c levels being 7.7. The results were statistically insignificant. (Table 5)

Table 6 (Initial wound discharge) :

Initial Wound discharge	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT	2	5.7	10	28.6		
PRESENT	33	94.3	25	71.4		
Total	35	100.0	35	100	6.437	**0.0112**
						Statistically significant

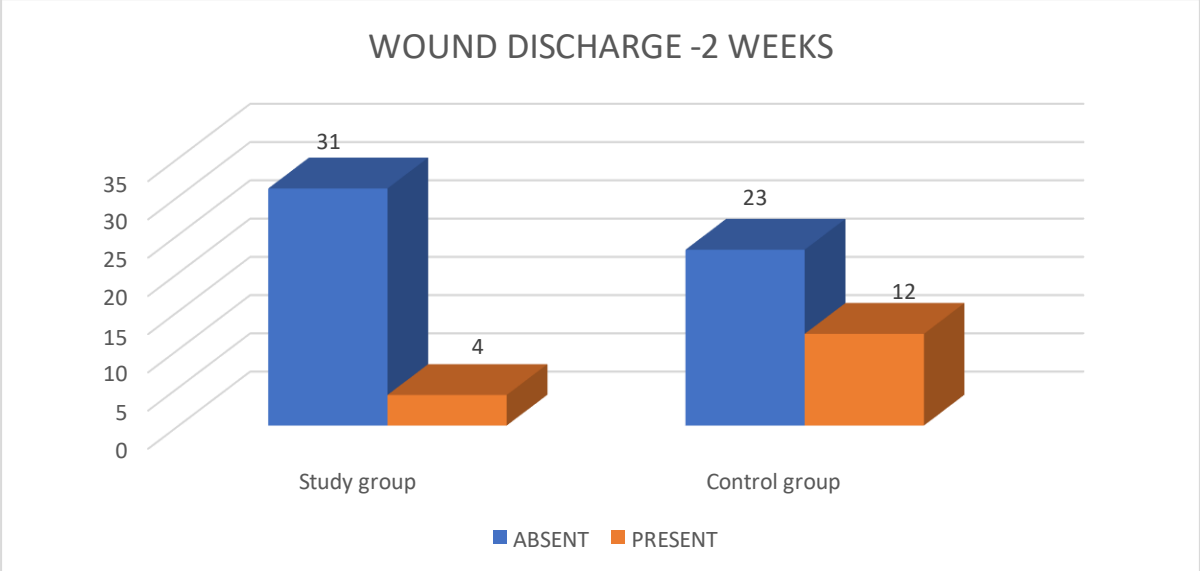


Graph 6

In the present study, incidence of wound discharge was present in 33 (94.3%) of patients and absent in 2 (5.7%) patients in study group. In control group initial wound discharge was present in 25 (71.4%) patients and absent in 10 (28.6%) of patients. The results were **statistically significant with p-value 0.0112**. (Table 6, Figure 6)

Table 7 (Wound discharge after 2 weeks):

Wound discharge- 2 weeks	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT	31	88.6	23	65.7		
PRESENT	4	11.4	12	34.3		
Total	35	100.0	35	100	5.185	**0.0228**
						Statistically significant

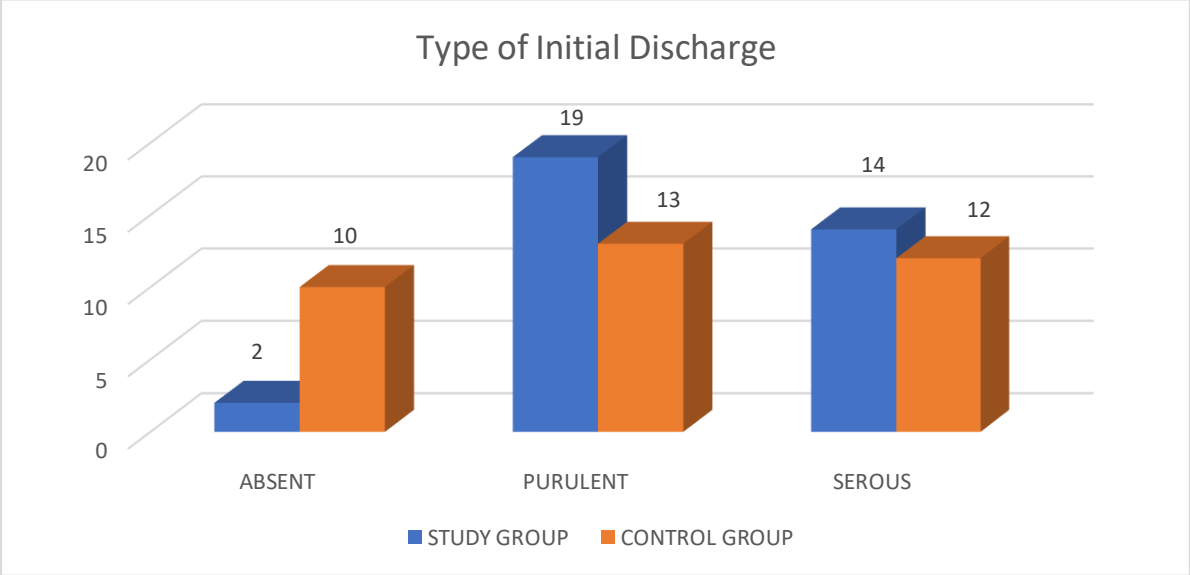


Graph 7

In the present study, incidence of wound discharge after 2 weeks of treatment was present in 4 (11.4%) of patients and absent in 31 (88.6%) patients in study group. In control group wound discharge after 2 weeks was present in 12 (34.3%) patients and absent in 23 (65.7%) of patients. The results were **statistically significant.(p value 0.0228)** (Table 7, Chart 7)

Table 8 (Type of Initial discharge)

TYPE OF DISCHARGE (P/A)—initial	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT	2	5.7	10	28.6		
PURULENT	19	54.3	13	37.1		
SEROUS	14	40.0	12	34.3		
Total	35	100.0	35	100.0	6.381	**0.0115**
						Statistically significant



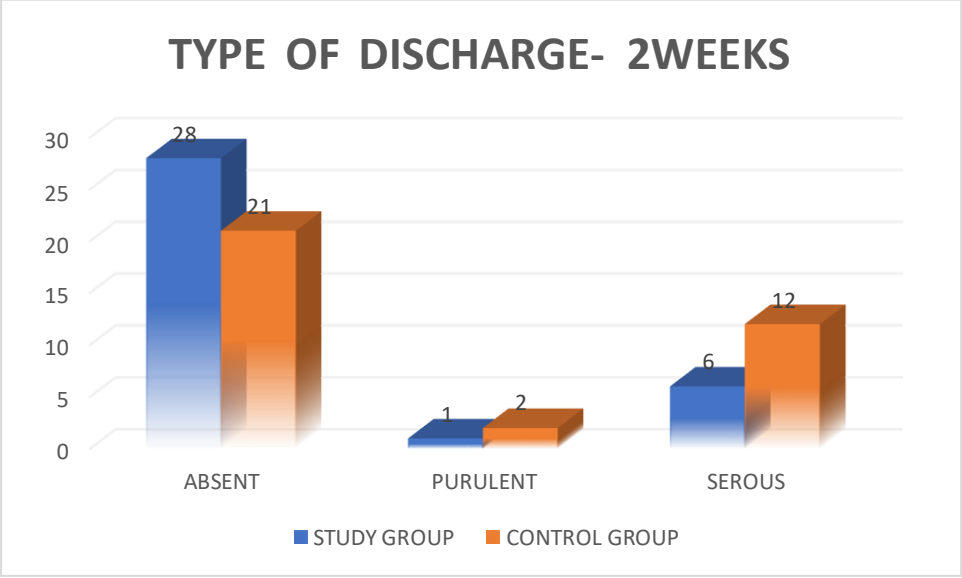
Graph 8

In the present study, the incidence of purulent discharge was higher . In study group 19 (54.3%) patients had purulent discharge, 14 (40%) patients had serous discharge where as

2(5.7%) patients had no discharge initially. In control group 13 (37.1%) patients had purulent discharge, 12 (34.3%) patients had serous discharge where as 10 (28.6%) patients had no discharge at the beginning of study. The results were **statistically significant**. (Table 8, Figure 8)

Table 9 (Type of discharge after 2 weeks):

TYPE OF DISCHARGE (P/A)-AFTER	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT	28	80.0	21	60.0		
PURULENT	1	2.9	2	5.7		
SEROUS	6	17.1	12	34.3		
Total	35	100.0	35	100.0	3.333	0.1889
						Statistically insignificant



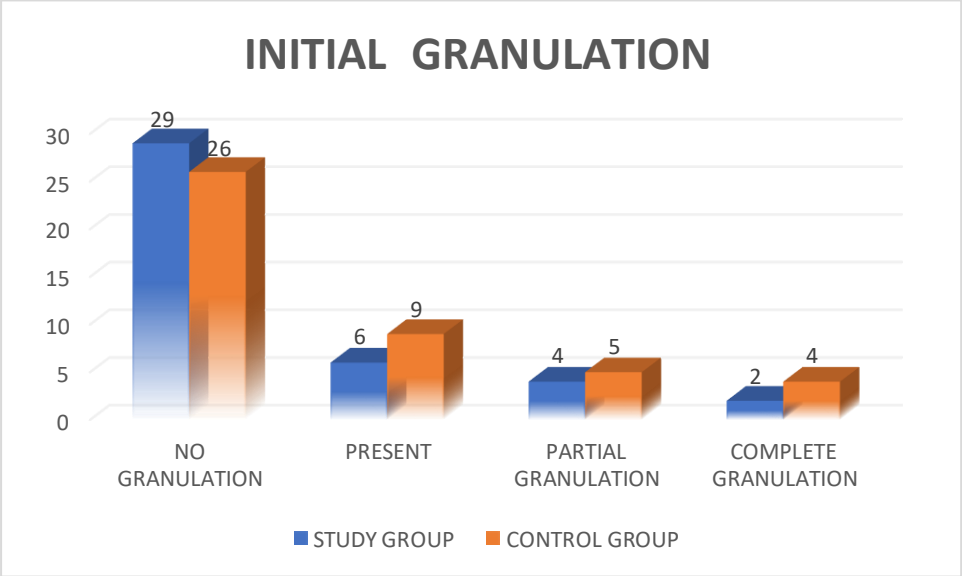
Graph 9

In the present study, after two weeks the incidence of no discharge was higher in both the groups . In study group 1 (2.9%) patient had purulent discharge, 6 (17.1%) patients had serous discharge where as 28(80%) patients had no discharge initially. In control group 2 (5.7%)

patients had purulent discharge, 12 (34.3%) patients had serous discharge where as 21 (60%) patients had no discharge at the beginning of study. The results were comparable in both study and control group hence, statistically insignificant. (Table 9. Graph 9)

Table 10 (Granulation tissue- initial)

GRANULATION TISSUE -INITIAL	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
NO GRANULATION	29	82.9	26	74.3		
PRESENT	6	17.1	9	25.7		
PARTIAL GRANULATION	4	11.4	5	14.2		
COMPLETE GRANULATION	2	5.7	4	11.4		
Total	35	100.0	35	100.0	0.7636	0.3822
						Statistically insignificant

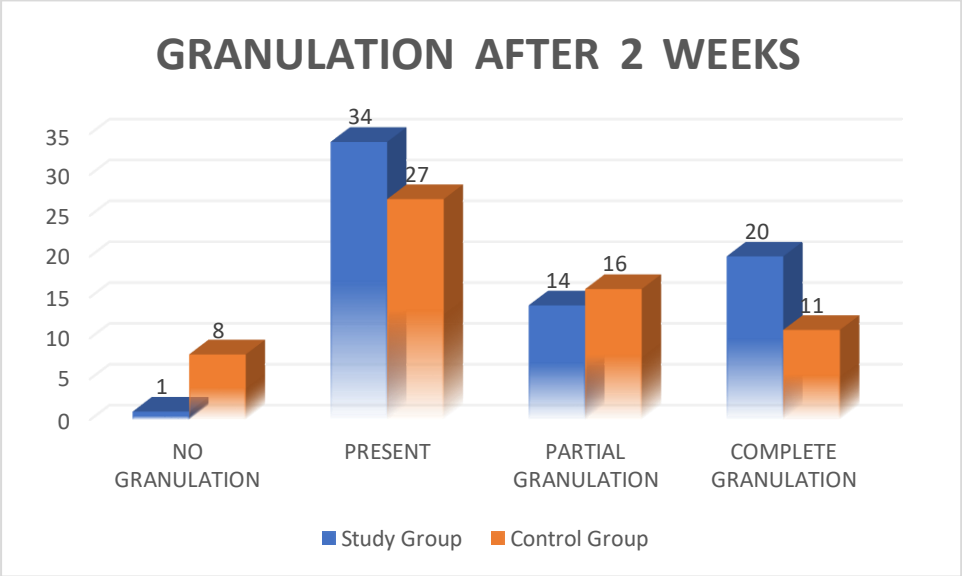


Graph 10

In the present study, granulation tissue was absent is most of the patients in both the groups. In study group, granulation tisuue was present initially in 6 (17.1%) of patients of which partial granulation was found in 4 (11.4%) patients , complete granulation was present in 2 (5.7%) patients , absent/no granulation tissue in 29 (82.9%) patients . In control group initial granulation tissue was present in 9 (25.7%) patients of which partial granulation tissue was present in 5 (14.2%)patients and complete granulation tissue was present in 4 (11.4%) patients and absent/ no granulation tissue in 26 (74.3%) of patients. The results were comparable and statistically insignificant. (Table 10, Figure 10)

Table 11 (Granulation tissue after 2 weeks)

GRANULATION TISSUE -2 WEEKS	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
NO GRANULATION	1	2.9	8	22.9		
PRESENT	34	97.1	27	77.1		
PARTIAL GRANULATION	14	40	16	45.7		
COMPLETE GRANULATION	20	57.1	11	31.4		
Total	35	100.0	35	100.0	6.248	**0.0124**

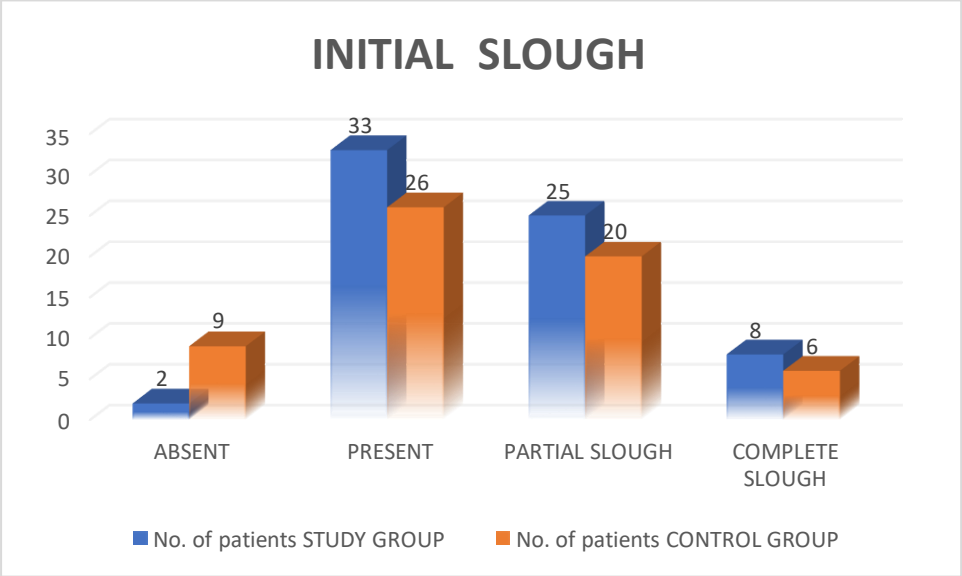


Graph 11

In the present study, after 2 weeks , incidence of granulation tissue was higher in study group. Granulation tisuue was present in 34 (97.1%) of patients of which 14 (40%)patients had partial granulation tissue and 20 (57.1%) patients had complete granulation tissue ; absent/no granulation in 1 (2.9%) patients in study group. In control group granulation tissue was present in 27 (77.1%) patients of which16 (45.7%) patients had partial granulation tissue and 11 (31.4%) patients had complete granulation tissue , absent/no granulation tissue in 8(22.9%) of patients. **The study group shows better results in appearance of granulation tissue and the results were statistically significant**. **(P value 0.0124) (Table 11, Graph 11)**

Table 12 (Slough – initial):

SLOUGH-INITIAL	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT	2	5.7	9	25.7		**0.0215**
PRESENT	33	94.3	26	74.3		
PARTIAL SLOUGH	25	71.4	20	57.1		
COMPLETE SLOUGH	8	22.8	6	17.1		
Total	35	100.0	35	100.0	5.285	**Statistically significant**

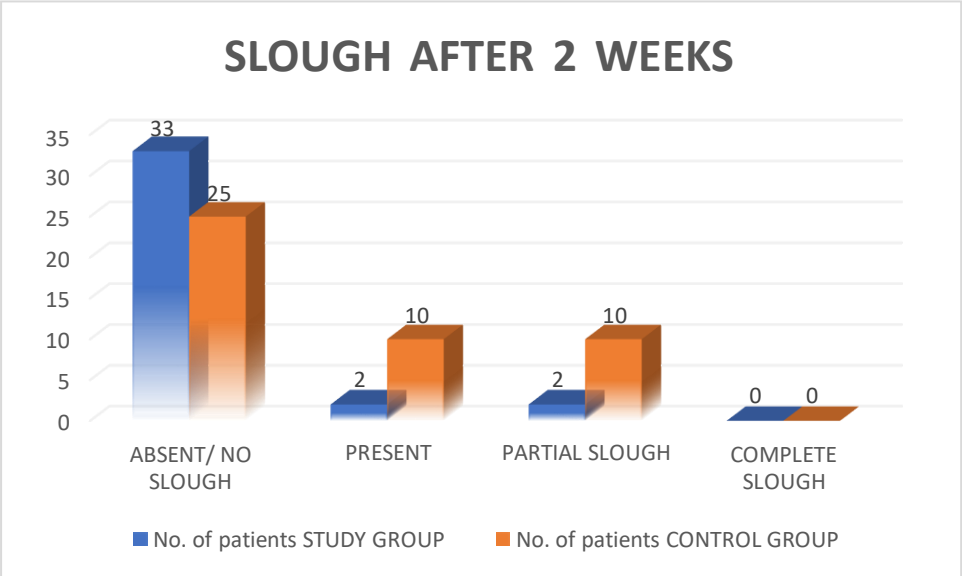


Graph 12

In the present study, the incidence of slough was higher in both the groups. In study group 33 (94.3%) patients slough was present of which 25 (71.4%) had partial slough and 8 (22.8%) patients had complete slough, 2 (5.7%) patients had no slough initially. In control group 26 (74.3%) patients slough was present of which 20(57.1%) had partial slough and 6 (17.1%) patients had complete slough, 9 (25.7%) patients slough was absent at the beginning of study. The results were **statistically significant with p value 0.0215**. (Table 12, Graph 12)

Table 13 (Slough after 2 weeks) :

SLOUGH-AFTER	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT/ NO SLOUGH	33	94.3	25	71.4		
PRESENT	2	5.7	10	28.6		
PARTIAL SLOUGH	2	5.7	10	28.6		
COMPLETE SLOUGH	0	0	0	0		
Total	35	100.0	35	100.0	6.437	**0.0112**
						Statistically significant

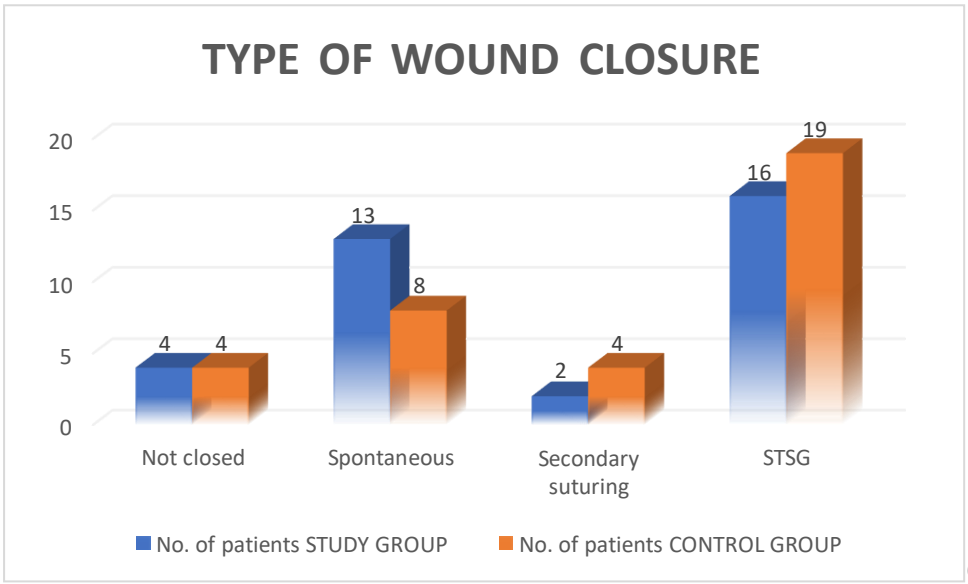


Graph 13

In the present study, the incidence of slough after 2 weeks was higher in control group. In study group 2 (5.7%) patients partial slough was present, 33 (94.3%) patients slough was absent. In control group 10 (28.6%) patients partial slough was present, 25 (71.4%) patients slough was absent after 2 weeks of study. Hence **study group shows significant reduction in slough as compared to control group and the results were statistically significant.(p value-0.0112) (Table 13, Graph 13)**

Table 14 (Type of wound closure) :

TYPE OF WOUND CLOSURE – Not closed	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Not healed /Not closed	4	11.4	4	11.4		
Healing by secondary intention /Spontaneous	13	37.1	8	22.9		
Secondary suturing	2	5.7	4	11.4		
STSG	16	45.7	19	54.3		
Total	35	100.0	35	100.0	2.114	0.5490
						Statistically insignificant

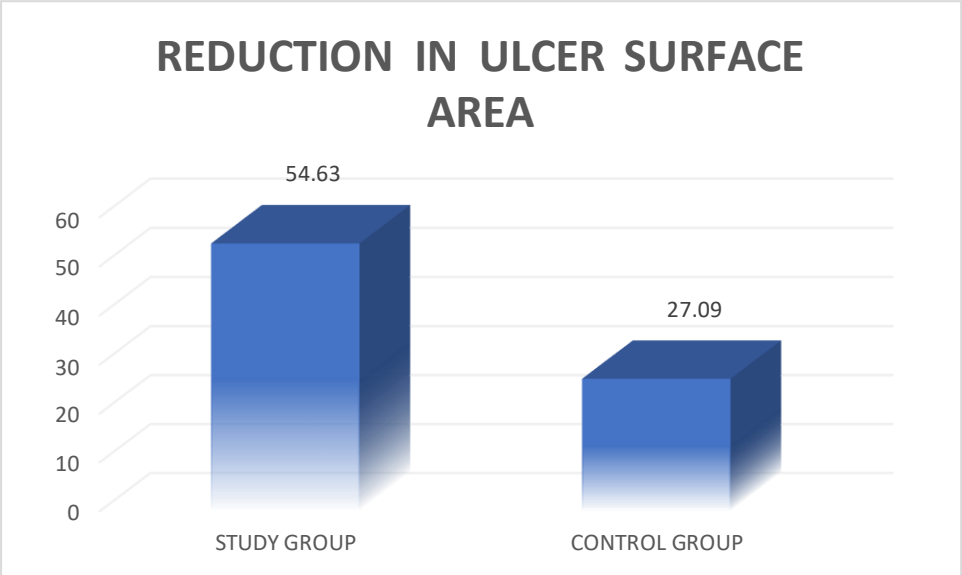


Graph 14

In the present study higher incidence of split thickness skin grafting (STSG) was done for wound closure in both study and control groups. In study group 16 (45.7 %) patients underwent STSG, 2 (5.7%) underwent secondary suturing , 13(37.1%) patients showed spontaneous closure , 4 (11.4%) wounds were not closed . In control group 19 (54.3 %) patients underwent STSG, 4 (11.4%) underwent secondary suturing , 8 (22.9%) patients showed spontaneous closure , 4 patients (11.4%) wounds were not closed / not . The results were comparable and statistically insignificant. (Table 14, Graph 14)

Table 15 : Comparison of Reduction in Ulcer surface area between study and control group

Reduction in Ulcer surface area	Study group		Control group		Mann whitney U test	P value
	Mean	Std. Deviation	Mean	Std. Deviation		
Reduction in Ulcer surface area	54.63	30.235	27.09	26.764	308.500	**P=0.0001***
***: Statistically significant**						

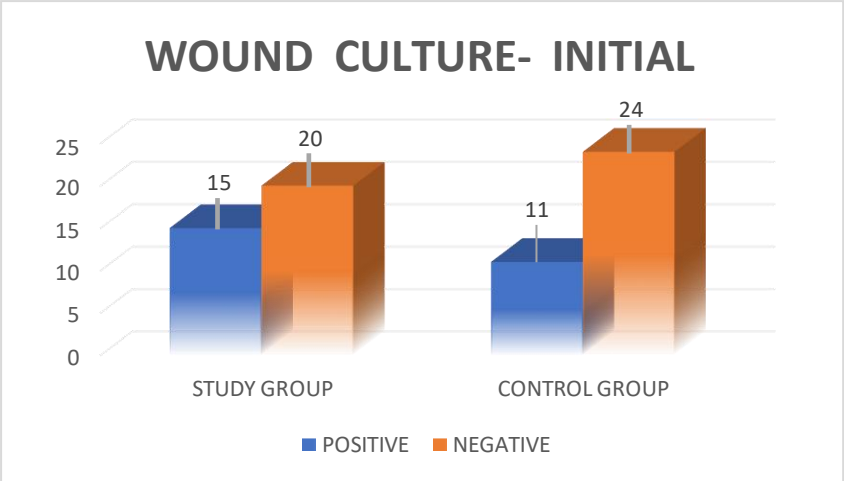


Graph 15

In the present study,incidence of mean reduction in ulcer surface area was seen more in study group than in control group. In study group mean reduction in ulcer surface area is 54.63% and in control group the mean reduction in ulcer surface area is 27.09% . **The results are statistically significant.(p-value 0.0001*) (Table 15, Graph 15)**

Table 16: Culture sensitivity (Initial)

CULTURE SENSITIVITY	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
POSITIVE	15	42.9	11	31.4		
NEGATIVE	20	57.1	24	68.6		
Total	35	100	35	100	0.9790	0.3224
						Statistically insignificant

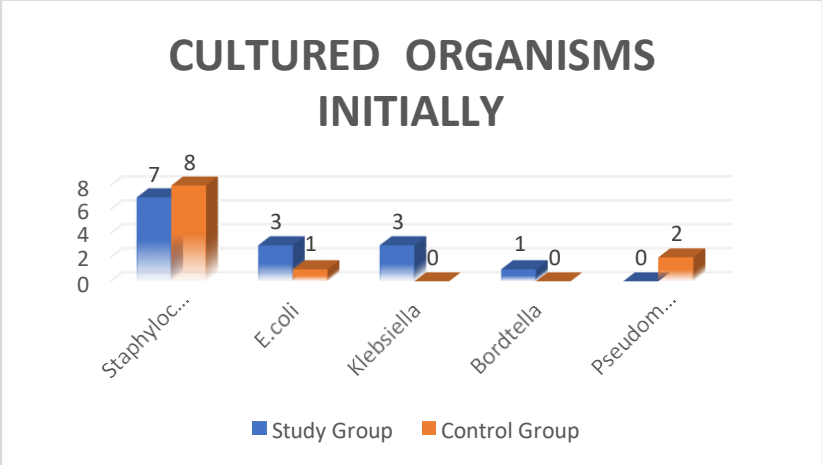


Graph 16

In the present study initially, higher incidence of negative culture was noted as compared to positive culture in both the groups. However,there were more positive cultures in study group. There were 15 (42.9%) patients who had culture positive and 20 (57.1%) patients had culture negative in study group. In control group, 11 (31.4%) patients had culture positive and 24 (68.6%) patients had culture negative. The results were statistically insignificant. (Table 16, Graph 16)

Table 17 : Initial Culture positive organisms

CULTURE SENSITIVITY	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Staphylococcus	7	20	8	22.8		
E.coli	3	8.5	1	2.8		
Klebsiella	3	8.5	0	0		
Bordtella	1	2.8	0	0		
Pseudomonas	0	0	2	5.7		
Total	35	100	35	100	0.9790	0.3224
						Statistically insignificant

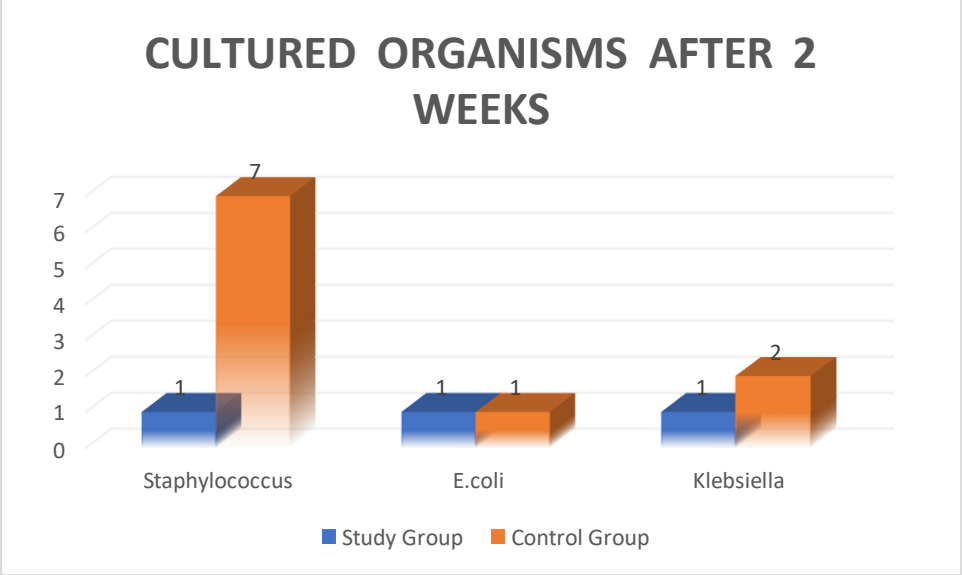


Graph 17

In the present study initial culture in study group came positive for 14 patients ie. Staphylococcus in 7 patients, E.coli in 3 patients, Klebsiella in 3 patients. Bordtella in 1 patient . In control group 11 patients initially showed culture positive ie. Staphylococcus in 8 patients, E.coli in 1 patient and Pseudomonas in 2 patients. The results were statistically insignificant (Table 17, Figure 17)

Table 18 : Culture positive organisms after 2 weeks

CULTURE SENSITIVITY	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Staph. aureus	1	20	7	22.8		
E.coli	1	8.5	1	2.8		
Klebsiella pneumonie	1	8.5	2	0		
Total	35	100	35	100	4.629	**0.0314**
						Statistically significant

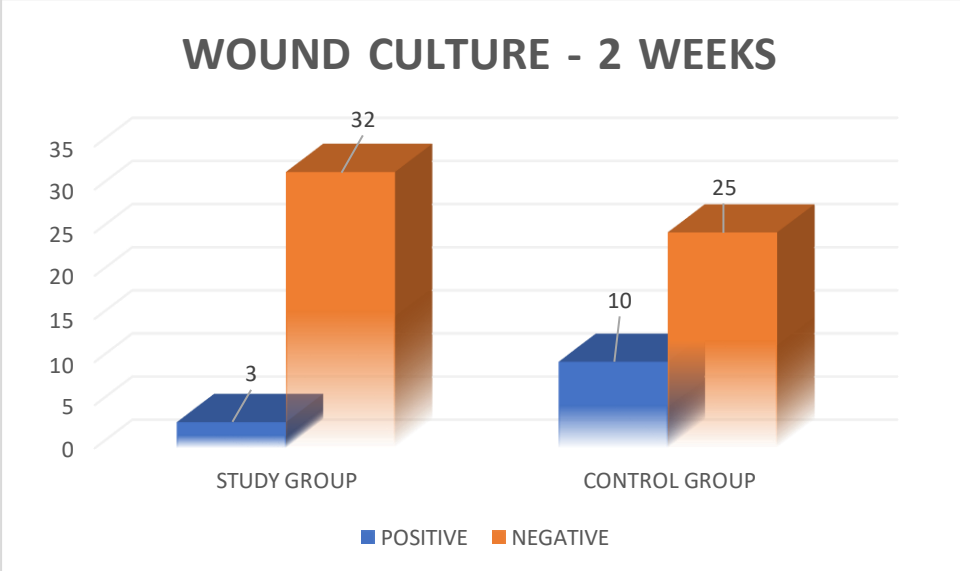


Graph 18

In the present study, after 2 weeks of treatment significant reduction in culture positivity was noted in study group as compared to control group. In study group, 3 patients were still positive culture from wound site ie. Staphylococcus in 1 patient, E.coli in 1 patient and Klebsiella in 1 patient. In control group, 10 patients still had positive culture ie. Staphylococcus in 7 patients, E.coli in 1 patient and Klebsiella in 2 patients. The results were **statistically significant with p value of 0.0314**. (Table 18, Graph 18)

Table 19: (Wound culture- after 2 weeks)

CULTURE SENSITIVITY	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
POSITIVE	3	8.5	10	28.6		
NEGATIVE	32	91.5	25	71.4		
Total	35	100	35	100	4.629	**0.0314**
						Statistically significant

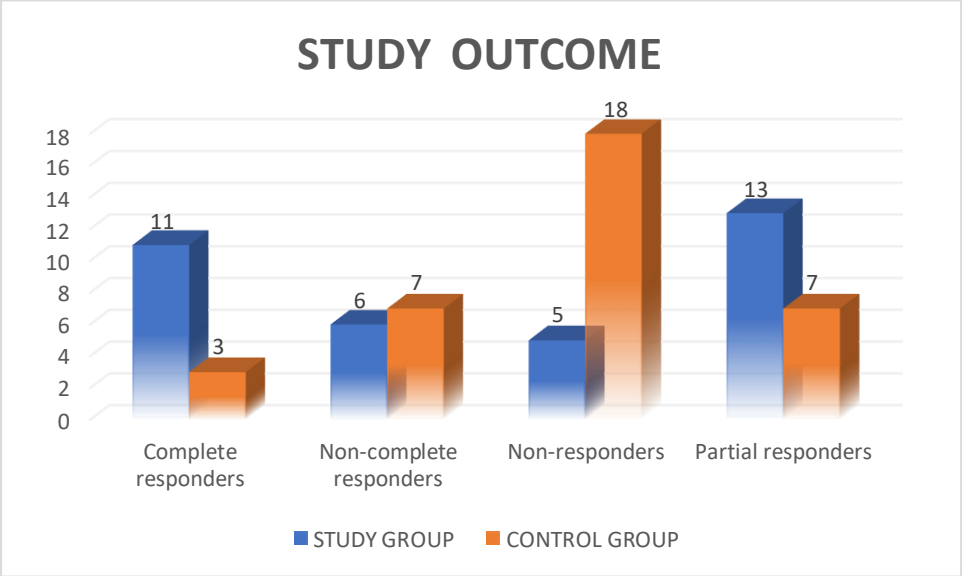


Graph 19

In the present study after 2 weeks, higher incidence of negative culture was noted as compared to positive culture in both the groups. However,there were more positive cultures in control group as compared to study group. There were 3 (8.5%) patients who had culture positive and 32 (91.5%) patients had culture negative in study group. In control group, 10 (28.6%) patients had culture positive and 25 (71.4%) patients had culture negative. Hence the study group is more efficiently inhibiting the growth of bacteria as compared to control group and **the results were statistically significant.(p-value 0.0314) (Table 19, Graph 19)**

Table 20: STUDY OUTCOME

Study outcome	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
Complete responders	11	31.4	3	8.6		
Non-complete responders	6	17.1	7	20.0		
Non-responders	5	14.3	18	51.4		
Partial responders	13	37.1	7	20.0		
Total patients	35	100	35	100	13.796	**0.0032**
						Statistically significant



Graph 20

Depending on the findings at the end of the study period the patients were categorized into the undermentioned categories [54].

1. Complete Responders: Complete healing of lower limb ulcers

2. Partial Responders: A 50% or greater reduction in the product of the two longest perpendicular diameters from baseline.

3. Non-complete responders: Less than 50% reduction in the product of the two longest perpendicular diameters from baseline.

4. Non-responders: No reduction or increase in ulcer area over base line.

In the present study, incidence of complete and partial responders were more in study group as compared to control group. However the incidence of non-responders and non-complete responders were more in control group as compared to study group. In study group, 11 (31.4%) patients were complete responders, 6 (17.1%) patients were non complete responders, 5 (14.3%) patients were non responders and 13 (37.1%) patients were partial responders. In control group, 3 (8.6%) patients were complete responders, 7 (20%) patients were non complete responders,18 (51.4%) patients were non responders and 7 (20%) patients were partial responders. **The results were statistically significant (p-value 0.0032) (Table 20, Graph 20)**

In the present study the incidence of lower limb ulcers was higher in the age group of 50-59 years

DISCUSSION

Lower limb ulcers are one of the most common problems encountered by general surgeons.The chronic pain and disability impose a big threat to patient's quality of life along with the financial burden due to prolonged treatment. More than 70% required surgical intervention and in more than 40% there is a toe or foot amputation [53].

Selection of an appropriate dressing with timely replacement can expedite the healing. Various modes of treatment and wound dressings are used but still treatment failure rate is very high and many patients still end up with limb amputations. Nanotechnology using silver ions offers greater antimicrobial property. The smaller silver particles produced are lesser toxic to human tissue cells due to increased surface area to volume ratios [54,55]

It is a comparative interventional study where a total of 70 patients with lower limb ulcers in between the age group of 16 – 80 were included in the study and randomly divided in two groups ; Study group (Nanocrystalline group) and Control group (Normal saline group). Parameters like Age, Sex, Diabetic/Non-Diabetic status, and wound characteristics like Wound surface area, presence/absence of discharge, type of discharge, presence/absence of slough, granulation tissue and reduction in mean ulcer surface area were compared at zero and after 2 weeks of treatment. At the end of study it was also noted whether the wound healed with secondary intention spontaneously, or with secondary suturing / split thickness skin grafting.

Depending on the findings at the end of the study period the patients were categorized into the undermentioned categories [54].

1. Complete Responders: Complete healing of lower limb ulcers

2. Partial Responders: A 50% or greater reduction in the product of the two longest perpendicular diameters from baseline.

3. Non-complete responders: Less than 50% reduction in the product of the two longest perpendicular diameters from baseline.

4. Non-responders: No reduction or increase in ulcer area over base line.

In the present study the incidence of lower limb ulcers were higher in 50-59 age group with mean age 52.2 in study group and 53.03 group in the control group. The age and sex distribution were comparable in both the groups and the results were statistically insignificant.

In the present study, the incidence of lower limb ulcer was higher in males than in females . Study group had 29(82.9%) males and 6(17.1%) females , Control Group had 31(88.6%) males and 4(11.4%). The results were comparable in both groups and were statistically insignificant. In a study conducted by Varun Gupta et. al the mean age of study group was 54.47 years, whereas in control group the mean age was 59.93 years. Age and sex distribution was comparable in both groups and statistically non significant.[56]. The results were comparable with the current study.

	Mean Age
Our Study	52.2±11.8 years
Varun Gupta et al	54.47±5.46 years

Table 21

The incidence of diabetics vs non-diabetics were comparable with p value being insignificant. In study group the incidence of diabetics were 16 (45.7%) and non-diabetics were 19 (54.3%). In control group the incidence of diabetics were 18 (51.4%) and non-diabetics were 17 (48.6%).The results were comparable in both study and control groups and the results were statistically insignificant.

Comorbidities like diabetes , hypertension and ischemic heart disease were found with more incidence of diabetes in both the groups. In study group the incidence of diabetes was found in 19 (54.3%) patients, incidence of hypertension was present in 13 (34.2%) patients , incidence of both diabetes and hypertension was found in 9 (25.7%) patients. In control group the incidence of diabetes was found in 18 (81.4%) patients, incidence of hypertension was found in 13 (37.1%) patients , incidence of both diabetes and hypertension was found in 10 (28.5%) patients.

The initial incidence of wound discharge was present in 33 (94.3%) patients and reduced to 4(11.4%) patients post treatment whereas in control group discharge was present initially in 25 (71.4%) patients and after 2 weeks of treatment discharge was still present in 12 (34.3%) patients. Hence the **study group shows significant reduction in wound discharge as compared to control group and the results were statistically significant**. (**p value 0.0228).**

In study group 19 (54.3%) patients had purulent discharge, 14 (40%) patients had serous discharge where as 2(5.7%) patients had no discharge initially. In control group 13 (37.1%) patients had purulent discharge, 12 (34.3%) patients had serous discharge where as 10 (28.6%) patients had no discharge at the beginning of study.After two weeks the incidence of no discharge was higher in both the groups . In study group 1 (2.9%) patient had purulent discharge, 6 (17.1%) patients had serous discharge where as 28(80%) patients had no discharge initially. In control group 2 (5.7%) patients had purulent discharge, 12 (34.3%) patients had serous discharge where as 21 (60%) patients had no discharge at the beginning of study. Hence the type of discharge post treatment (after 2 weeks) were comparable in both study and control group and were statistically insignificant. In a study conducted by Varun Gupta et. al, initially purulent discharge was present in both study and control group, but there was a gradual shift from purulent to serous discharge and the reduction in discharge was faster in study group ,results being statistically significant[56]. The results are comparable with current study.

	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT/ NO GRANULATION	1	2.9	8	22.9		
PRESENT	34	97.1	27	77.1		
PARTIAL GRANULATION	14	40	16	45.7		
COMPLETE	20	57.1	11	31.4		

GRANULATION						
Total	35	100.0	35	100.0	6.248	**0.0124**
						Statistically significant

As per Varun et. al

Granulation tissue	Initial		2 weeks	
	Study	Control	Study	Control
Absent	15	15	2	8
Present	-	-	13	7
Total	15	15	15	15
	30		30	

Table 22

In the current study, granulation tissue was absent is most of the patients in both the groups. In study group, granulation tisuue was present initially in 6 (17.1%) of patients and absent in 29 (82.9%) patients in study group. In control group initial granulation tissue was present in 9 (25.7%) patients and absent in 26 (74.3%) of patients.However after 2 weeks , incidence of granulation tissue was higher in study group. Granulation tisuue was present in 34 (97.1%) of patients and absent in 1 (2.9%) patients in study group. In control group granulation tissue was present in 27 (77.1%) patients and absent in 8(22.9%) of patients. The **study group shows better results in early appearance of granulation tissue and the results were statistically significant (p-value 0.0124).** Unlike the present study, in a study conducted by Varun Gupta et. al, the appearance of granulation tisuue in both nanocrystalline group and normal saline group were similar with results being statistically insignificant (p value 0.283) [56]

Our study:

SLOUGH-AFTER	Study group		Control group		Chi square test	P value
	No. of patients	Percentage	No. of patients	Percentage		
ABSENT/ NO SLOUGH	33	94.3	25	71.4		
PRESENT	2	5.7	10	28.6		
PARTIAL SLOUGH	2	5.7	10	28.6		
COMPLETE SLOUGH	0	0	0	0		
Total	35	100.0	35	100.0	6.437	**0.0112**
						Statistically significant

As per Varun et. al :

Slough Tissue	Initial		2 weeks	
	Study	Control	Study	Control
Absent	-		8	6
Present	15	15	7	9
Total	15	15	15	15
	30		30	

Table 23

The initial incidence of slough was higher in both the groups. In study group 33 (94.3%) patients slough was present, 2 (5.7%) patients had no slough initially. In control group 26 (74.3%) patients slough was present, 9 (25.7%) patients slough was absent at the beginning of study. The results were statistically significant.After 2 weeks, the incidence of slough was higher in control group. In study group 2 (5.7%) patients slough was present, 33 (94.3%)

patients slough was absent. In control group 10 (28.6%) patients slough was present, 25 (71.4%) patients slough was absent after 2 weeks of study. **Hence study group shows significant reduction in slough as compared to control group and the results were statistically significant (p value-0.0112).** In a study conducted by Varun Gupta et. al there was significant reduction in slough in nanocrystalline silver group as compared to normal saline group and the results were statistically significant (p value 0.045). The results were comparable to the current study.

In the current study higher incidence of split thickness skin grafting (STSG) was done for wound closure in both study and control groups. In study group 16 (45.7 %) patients underwent STSG, 2 (5.7%) underwent secondary suturing , 13(37.1%) patients showed spontaneous closure , 4 (11.4%) wounds were not closed . In control group 19 (54.3 %) patients underwent STSG, 4 (11.4%) underwent secondary suturing , 8 (22.9%) patients showed spontaneous closure , 4 patients (11.4%) wounds were not closed . The results were comparable and statistically insignificant. In a study conducted by Varun et al the incidence of spontaneous closure was highest followed by split thickness skin grafting followed by secondary suturing,however the results were statistically insignificant (p value 0.063)

In the present study, incidence of mean reduction in ulcer surface area was seen more in study group than in control group. In study group mean reduction in ulcer surface area is 54.63% and in control group the mean reduction in ulcer surface area is 27.09% . Hence the **study group shows significant reduction in ulcer surface area as compared to control group and the results are statistically significant (p-value 0.0001*).** In a similar study conducted by Varun Gupta et. al mean initial ulcer size in study group was 55.67 cm square and in control group was 54.93 cm square and at the end of 8 weeks the mean ulcer size in the study population was 9.23 cm square and 18.31 cm square in the control group. The results were statistically significant and comparable to our study.[56]

	Reduction in mean surface area in study group	Reduction in mean surface area in control group
Our Study	54.63 %	27.09 %
Varun Gupta et al[56]	83.42%	66.66%
Sharma et. al [57]	85.63%	68.63%

Table 24

Initially, higher incidence of negative culture was noted as compared to positive culture in both the groups. However,there were more positive cultures in study group. There were 15 (42.9%) patients who had culture positive and 20 (57.1%) patients had culture negative in study group. In control group, 11 (31.4%) patients had culture positive and 24 (68.6%) patients had culture negative.After 2 weeks, higher incidence of negative culture was noted as compared to positive culture in both the groups. However,there were more positive cultures in control group as compared to study group even after 2 weeks of treatment. There were 3 (8.5%) patients who had culture positive and 32 (91.5%) patients had culture negative in study group. In control group, 10 (28.6%) patients had culture positive and 25 (71.4%) patients had culture negative. Initial culture in study group came positive for 14 patients ie. Staphylococcus in 7 patients, E.coli in 3 patients, Klebsiella in 3 patients. Bordtella in 1 patient . In control group 11 patients initially showed culture positive ie. Staphylococcus in 8 patients, E.coli in 1 patient and Pseudomonas in 2 patients. After 2 weeks of treatment significant reduction in culture positivity was noted in study group as compared to control group. In study group, 3 patients were still positive culture from wound site ie. Staphylococcus in 1 patient, E.coli in 1 patient and Klebsiella in 1 patient. In control group, 10 patients still had positive culture ie. Staphylococcus in 7 patients, E.coli in 1 patient and Klebsiella in 2 patients. Hence **the study group is more efficiently inhibiting the growth of bacteria as compared to control group and the results were statistically significant (p-value 0.0314).** Wright J et al., Yin H et al., Voight D et al. conducted a similar study and attributed to the potent and rapid antibacterial activity of nanocrystalline silver. [58-60]

In the present study, incidence of complete and partial responders were more in study group as compared to control group. However the incidence of non-responders and non-complete responders were more in control group as compared to study group. In study group, 11 (31.4%) patients were complete responders, 6 (17.1%) patients were non complete responders, 5 (14.3%) patients were non responders and 13 (37.1%) patients were partial responders. In control group, 3 (8.6%) patients were complete responders, 7 (20%) patients were non complete responders,18 (51.4%) patients were non responders and 7 (20%) patients were partial responders.Hence **the study group responded more efficiently and effectively as compared to control group and the results were statistically significant (p-value 0.0032)**

Sharma R et al,in a similar study recorded a higher percentage of complete responders (84.6%) which could be attributed to the longer duration of treatment in their study (12 weeks), however they also supported the fact that nanocrystalline silver ions accelerate healing of wounds [57]

In a different case study, Voight showed the effects of nanocrystalline silver on four patients who had debicutus ulcers; one ulcer that had been present for 24 months was cured in 27 days, while another that had been present for two weeks was healed in 14 days. In every case where nanocrystalline silver was used, they showed a decrease in exudate fluid quantities. The usage of Nanocrystalline silver dressing was studied in a multi-centered (41 centres) survey by the same authors. They found that Integra, a dermal regeneration template for full thickness burns repair, was covered with Nanocrystalline silver in up to 52% of the centres surveyed (61% of which used the material). Additionally, they stated that 4.8% of the respondents utilised Nanocrystalline silver as their primary dressing. They came to the conclusion that nanocrystalline silver is affordable, improves wound healing, and can be used on all kinds of wounds. (38).

In a single centre, open-label, unblinded pilot trial conducted in 2002 by Kirshner et al., 11 extended-care facility outpatients or residents with chronic wounds of mixed aetiology were used to explore the effect of silver in wound healing. Every wound had a history of at least three months, and in the three weeks before the research, there had been no shrinkage of the wound size. All of the patients received Nanocrystalline silver treatment, and after the first week, the dressings were changed every other day. All used dressings were saved for fluid collection and analysis. The authors discovered a decrease in matrix metalloproteinase

activity in the first two days of treatment in eight individuals who finished the research. This demonstrated that if the nanocrystalline silver dressing is used consistently, the changed matrix metalloproteinase activity may persist. (16).

CONCLUSION

Nanocrystalline silver dressings offer significant reduction in ulcer surface area, early disappearance of wound discharge and slough; and early appearance of granulation tissue. It also proves to be a potent antibacterial and offers significant reduction in culture positivity of wounds. **Hence nanocrystalline silver is beneficial and highly effective in comparison to normal saline dressings in the management of lower limb ulcers.**

IMAGES FROM STUDY GROUP (Before and After)

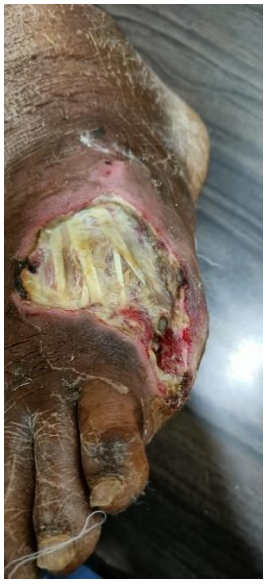



IMAGE 1

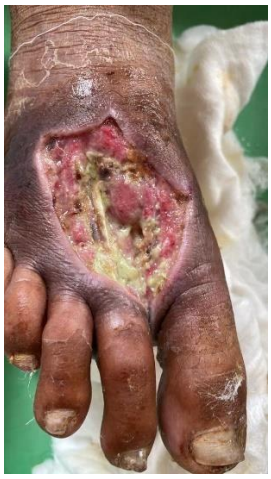
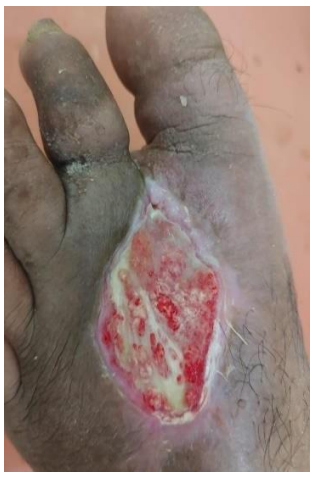



IMAGE 2

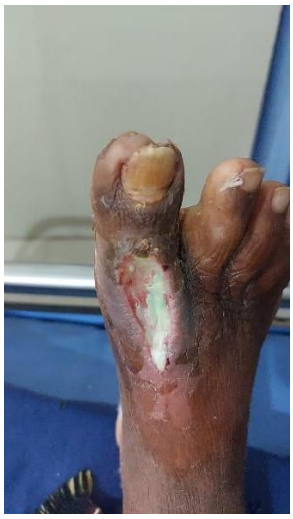



IMAGE 3

IMAGES FROM CONTROL GROUP (Before and After)

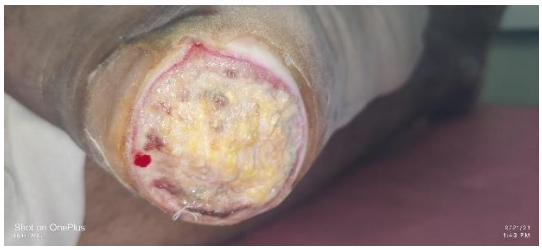
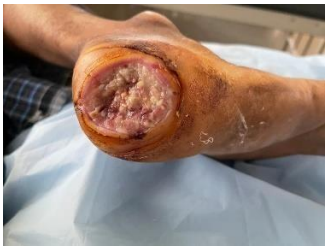



IMAGE 4

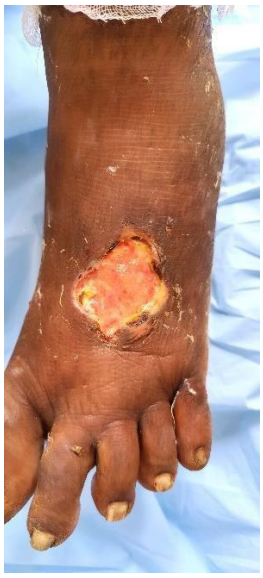



IMAGE 5

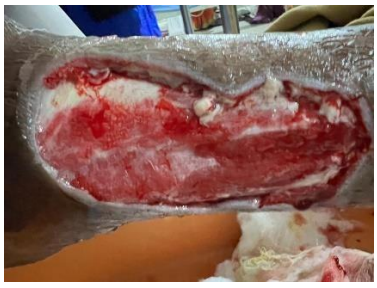
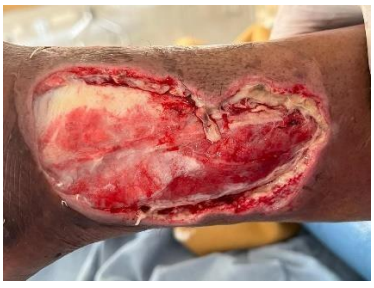



IMAGE 6

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ANNEXURE 1

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/100-09/2021 Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: To study the efficacy of Nano-crystalline silver dressings in comparison to conventional dressings in the management of

Name of PG student: Dr Arun Pandey, Department of Surgery

Name of Guide/Co-investigator: Dr A.V.Patil, Professor
Department of Surgery

DR .S.V.PATIL
CHAIRMAN, IEC

Institutional Ethical Committee
L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

ANNEXURE 2

INFORMED CONSENT FORM

TITLE OF THE PROJECT:

"NANO CRYSTALLINE SILVER DRESSINGS IN COMPARISON TO CONVENTIONAL NORMAL SALINE DRESSINGS IN THE MANAGEMENT OF LOWER LIMB ULCERS"

NAME OF THE INVESTIGATOR: DR. ARUN PANDEY
NAME OF THE GUIDE: DR. ARAVIND V PATIL

CONFIDENTIALITY OF RECORDS:

I understand that medical information produced by this study will become a part of this hospital record and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of only by a code number

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.
I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.
I have explained to_____the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study, provided such use is only for scientific purposes (s). I fully consent to participate in the above study.

Dr Arun Pandey
(Investigator)

Participant's Signature

ANNEXURE 3

PROFORMA

Date:

Name:
Age:
Sex:
IP NO:
Occupation:
Address:

Chief complaints:

HOPI:

Past history:

Family history:
Personal history:

General physical examination:

Vitals
Pulse:
BP:
RR:
Temperature

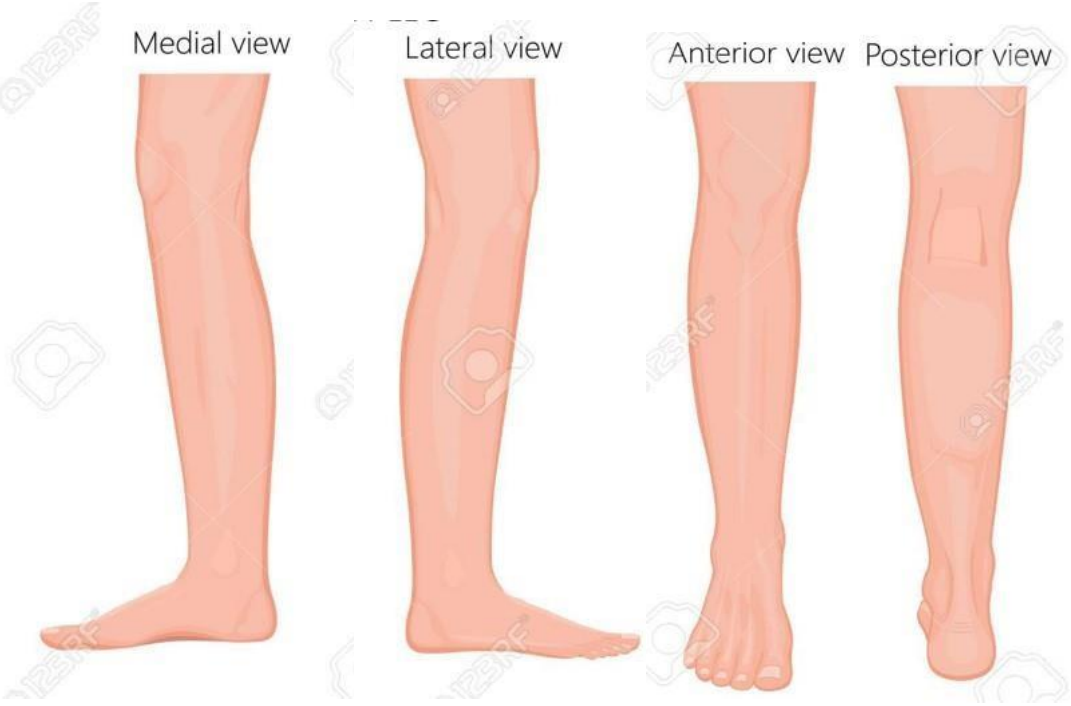
Systemic examination:
Per abdomen:

Cardiovascular system:

Respiratory system:

Central nervous system:

LOCAL EXAMINATION:



a) Presence of wound discharge

b) Type of wound discharge (purulent/serous)

c) Granulation tissue

d) Changes in the size of wound (largest transverse diameter and largest vertical diameter also including the depth)

e) Presence of slough (percentage of total surface area).

Investigations:

1. HEMOGRAM- HB , TC , PLATELET COUNT
2. BLOOD SUGAR
4. BT CT
5. HIV , HBsAg , HCV

When required: 1) Hba1c
2) Doppler USG
3) Culture sensitivity
4) ECG

Final diagnosis:

Treatment:

Cost of the procedure -

Duration of the procedure -

Outcomes/followup

Comments (if any)

ANNEXURE 4 (MASTER CHART)

	SEX	IP NO.	Hba1c	RBS (mg/	DIABETIC (Y/N)	DRESSING WITH	WOUND DISCHARGE (P/A)		TYPE OF DISCHARGE (P/A)		GRANULATION TISSUE (P/A)		SLOUGH TISSUE (P/A)		WOUND SIZE (before)	WOUND SIZE (after)		Reduction in Ulcer surface area		TYPE OF WOUND CLOSURE				STUDY OUTCOME				Culture initial	Culture after 2 weeks
3							initial	2 weeks	initial	2 weeks	initial	2 weeks	initial	2 weeks						Not closed	spontaneous	Secondary suturing	STSG	Complete responders	non complete responders	non responders	Partial responders		
4	M	128935		110	N	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	4*3 CMS	1*1CMS		92	92%		YES			YES				E.coli	Sterile
5	F	168201		104	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	4*3 CMS	3*3 CMS		25	25%		YES				YES			Staph epidermidi	Sterile
6	M	184926	7.4	233	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	5*4 CMS	4*4 CMS		20	20%				YES				YES	Klebriella pneum	Sterile
7	F	115308	7.2	252	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	15*10CMS	12*10CMS		20	20%				YES		YES			Sterile	Sterile
8	M	131407		97	N	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	2*2 CMS	0.5*0.5CMS		93.75	93.75%		YES			YES				Sterile	Sterile
9	M	186307	6.8	281	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	4*4	3*3		43.75	43.75%		YES						YES	Staph aureus	Sterile
10	M	157311	8.3	208	Y	NANOCRYSTALL	PRESENT	PRESENT	PURULENT	PURULENT	ABSENT	PRESENT	PRESENT	ABSENT	3*3	2*1		77.7	77.70%	YES						YES		Bardtella	E coli
11	M	141622		114	N	NANOCRYSTALL	PRESENT	PRESENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	8*4	6*2		62.5	62.50%			YES					YES	Staph aureus	Sterile
12	F	100571		98	N	NANOCRYSTALL	PRESENT	PRESENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	10*4	9*4		10	10%				YES		YES			Sterile	Sterile
13	M	114523	7.4	208	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	15*10	13*9		22	22%				YES		YES			E.coli	Sterile
14	M	200832	7.3	342	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT	10*8	8*6		40	40%	YES			YES				YES	Sterile	Sterile
15	M	216223		136	N	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	1*2	0.5*0.5		87.5	87.50%		YES			YES				Sterile	Sterile
16	M	194126		124	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	6*3	4*2		55.5	55.50%			YES					YES	Sterile	Sterile
17	M	167112		128	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	3*2	1*1		83.3	83.30%		YES			YES				Sterile	Sterile
18	M	132966	8.2	246	Y	NANOCRYSTALL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	3*3	1*2		77.7	77.70%		YES			YES				Sterile	Sterile
19	M	242333		78	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	15*10	15*10		0	0%				YES			YES		Sterile	Sterile
20	M	94166	7.8	210	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	5*5	4*4		36	36%				YES				YES	E.coli	Klebriella pneumonia
21	M	203294	7.6	216	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	10*4	8*2		60	60%				YES				YES	Staph. Aureus	Sterile
22	M	201943	6.9	243	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	6*4	6*4		0	0%				YES			YES		Sterile	Sterile
23	M	143733	8.1	270	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	SEROUS	PRESENT	PRESENT	PRESENT	ABSENT	3*2	1*1		83.3	83.30%		YES			YES				Sterile	Sterile
24	M	196402	8.2	219	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	2*2		0	100	100		YES			YES				Staph. aureus	Sterile
25	M	159056	7.8	188	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	4*4	3*1		81.25	81.25%		YES			YES				Sterile	Sterile
26	F	258358	8.4	184	Y	NANOCRYSTALL	PRESENT	PRESENT	SEROUS	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	15*10CMS	14*8CMS		25.3	25.30%	YES						YES		Staph aureus	Sterile
27	M	245726		110	N	NANOCRYSTALL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	14*8	12*6		35.7	35.70%				YES				YES	Sterile	Sterile
28	M	263081		95	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	10*6	9*5		25	25%				YES		YES			Sterile	Sterile
29	M	203294	7.6	222	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	15*3	10*2		55.5	55.50%				YES				YES	Klebriella pneum	Sterile
30	M	159055		116	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	6*4	2*3		75	75%		YES			YES				Sterile	Sterile
31	M	28935	8.2	248	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	3*4	0.5*0.5		97.9	97.90%		YES			YES				Staph aureus	Sterile
32	F	241622		88	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	10*8	8*8		20	20%	YES						YES		Sterile	Sterile
33	M	179792		80	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	6*6	2*2		88.8	88.80%				YES				YES	Sterile	Sterile
34	F	311309	7.6	210	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	3*3	2*2		55.5	55.50%				YES				YES	Sterile	Sterile
35	M	301652	7.8	310	Y	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	5*3	3*1		80	80%		YES						YES	Sterile	Sterile
36	M	203294	8.3	216	Y	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	5*3	3*3		40	40%				YES		YES			Sterile	Sterile
37	M	148177		142	N	NANOCRYSTALL	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	3*3	2*2		55.5	55.55%										
38	M	339960		130	N	NANOCRYSTALL	PRESENT	ABSENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	5*5	2*2		84	84%				YES				YES	Klebriella	Sterile
39																								YES					
40																													
41	M	102326	7.3	210	Y	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT	5*4	4*4		20	20%				YES				YES	Sterile	Klebriella pneumonia
42	M	115962		108	N	NORMAL SALINE	PRESENT	ABSENT	PURULENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	3*3	3*2		33.3	33.30%	YES						YES		Sterile	Sterile
43	M	256455	6.9	264	Y	NORMAL SALINE	PRESENT	PRESENT	SEROUS	SEROUS	PRESENT	PRESENT	PRESENT	ABSENT	15*10	15*9		10	10%				YES		YES			Sterile	Sterile
44	M	140431		90	N	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	3*3	3*2		33.3	33.30%		YES					YES		Sterile	Sterile
45	M	210677	8.4	257	Y	NORMAL SALINE	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	2*2	2*2		0	0%		YES				YES			Sterile	Sterile
46	M	24019	7.8	210	Y	NORMAL SALINE	PRESENT	ABSENT	SEROUS	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	2*2	0.5*0.5		93.75	93.75%		YES			YES				Sterile	Sterile
47	M	261224	8.2	180	Y	NORMAL SALINE	PRESENT	ABSENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	6*5	5*5		16.6	16.60%	YES							YES	Sterile	Sterile
48	M	187241	8	195	Y	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	1*1	1*1		0	0%		YES					YES		Sterile	Sterile
49	M	210829		114	N	NORMAL SALINE	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	6*5	5*4		33.3	33.30%				YES		YES			Sterile	Sterile
50	M	132496		106	N	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT	5*5	3*3		64	64%				YES				YES	Sterile	Sterile
51	F	21805		94	N	NORMAL SALINE	PRESENT	PRESENT	PURULENT	PURULENT	ABSENT	PRESENT	PRESENT	PRESENT	4*4	4*3		25	25%				YES			YES		Staph aureus	Sterile
52	M	232162		96	N	NORMAL SALINE	PRESENT	ABSENT	PURULENT	ABSENT	PRESENT	PRESENT	PRESENT	ABSENT	10*2	9*2		10	10%			YES				YES		Sterile	Sterile
53	M	190682	6.9	245	Y	NORMAL SALINE	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	4*2	3*1		62.5	62.50%				YES		YES			Staph aureus	Sterile
54	M	248113		119	N	NORMAL SALINE	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	2*1	2*1		0	0%		YES					YES		E.coli	Sterile
55	M	194089	7.2	176	Y	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	3*3	3*3		0	0%				YES			YES			
56	F	208613	7.4	211	Y	NORMAL SALINE	PRESENT	PRESENT	SEROUS	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	5*2	5*2		0	0%			YES					YES	Sterile	Sterile
57	M	203294	7.7	265	Y	NORMAL SALINE	PRESENT	PRESENT	PURULENT	PURULENT	ABSENT	ABSENT	PRESENT	ABSENT	6*5	5*5		16.6	16.60%				YES			YES		Sterile	E.Coli
58	M	266984		114	N	NORMAL SALINE	PRESENT	ABSENT	SEROUS	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	4*1	2*1		50	50%			YES					YES	Sterile	Staph aureus
59	M	239605	8.1	287	Y	NORMAL SALINE	PRESENT	PRESENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	7*5	7*5		0	0%				YES			YES		Sterile	Sterile
60	M	201942		140	N	NORMAL SALINE	PRESENT	PRESENT	SEROUS	SEROUS	PRESENT	PRESENT	ABSENT	ABSENT	15*10	14*10		6.66	6.66%				YES			YES		Sterile	Sterile
61	M	204680	8.6	180	Y	NORMAL SALINE	PRESENT	PRESENT	SEROUS	SEROUS	ABSENT	PRESENT	ABSENT	ABSENT	14*8	14*8		0	0%				YES			YES		Sterile	Sterile
62	M	203294	8	117	Y	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	4*2	2*1		50	50%		YES						YES	Sterile	Sterile
63	M	264479	7.5	180	Y	NORMAL SALINE	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	12*6	10*4		44.4	44.40%				YES				YES	Pseudomonas	Sterile
64	M	13916		98	N	NORMAL SALINE	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT	8*4	6*3		43.75	43.75%				YES		YES				
65	M	24019	8.1	104	Y	NORMAL SALINE	PRESENT	ABSENT	SEROUS	ABSENT	PRESENT	HEALED	ABSENT	ABSENT	1*1		0	100	100%		YES			YES				Sterile	Sterile
66	F	290537		132	N	NORMAL SALINE	PRESENT	PRESENT	PURULENT	SEROUS	ABSENT	PRESENT	PRESENT	ABSENT	15*8	13*7		24.16	24.16%				YES		YES				
67	M	285883	7.4	213	Y	NORMAL SALINE	PRESENT	ABSENT	PURULENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	10*8	10*6		25	25%				YES			YES			
68	M	289881		164	N	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	3*3	1*1		66.66	66.66%		YES			YES					
69	M	302889		132	N	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	4*1	4*1		0	0%			YES				YES		Pseudomonas	Sterile
70	M	265582		136	N	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	1*1	1*1		0	0%	YES						YES			
71	M	80953		140	N	NORMAL SALINE	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	4*4	4*3		25	25%				YES			YES		Sterile	Sterile
72	M	324422		142	N	NORMAL SALINE	PRESENT	PRESENT	SEROUS	SEROUS	PRESENT	PRESENT	PRESENT	ABSENT	12*6	10*5		30	30%				YES			YES		Sterile	Sterile
73	M	310630		138	N	NORMAL SALINE	PRESENT	PRESENT	PURULENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	6*6	5*5		30.55	30.55%				YES		YES			Sterile	Sterile
74	F	312447	7.5	244	Y	NORMAL SALINE	PRESENT	PRESENT	SEROUS	SEROUS	PRESENT	PRESENT	PRESENT	ABSENT	8*4	8*3		33	33%				YES			YES		Sterile	Sterile
75	M	316412	7.6	185	Y	NORMAL SALINE	PRESENT	PRESENT	PURULENT	SEROUS	ABSENT	ABSENT	PRESENT	PRESENT	2*2	2*2		0	0%	YES						YES		Sterile	Klebriella