“EVALUATION OF CLINICAL OUTCOME OF NEGATIVE PRESSURE WOUND THERAPY IN GUSTILO ANDERSON TYPE IIIA/IIIB OPEN FRACTURES OF EXTREMITIES”

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DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

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Under the Guidance of

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The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the Synopsis entitled **"Evaluation of Clinical Outcome of Negative Pressure Wound Therapy in Gustilo Anderson Type IIIA/ IIIB Open Fractures of Extremities"** being investigated by Dr.ARUN KUMAAR S.P & Dr. Arun H S in the Department of Orthopaedics at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study**.

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EVALUATION OF CLINICAL OUTCOME OF NEGATIVE PRESSURE WOUND THERAPY IN GUSTILO ANDERSON TYPE IIIA/IIIB OPEN FRACTURES OF EXTREMITIES

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ABBREVIATIONS

S. No	Abbreviation	Explanation
1	BP	Blood Pressure
2	DRIS	Disability Rating Index Score
3	MESS	Mangled Extremity Severity Scale
4	NPWT	Negative Pressure Wound Therapy
5	RCT	Randomized Control Trial
6	VAC	Vacuum Assisted Closure
7	SSG	Split Skin Grafting
8	WHO	World Health Organization
9	FDA	Food and Drug adminstration

ABSTRACT

Background: Open-fractures are a common and serious injury that primarily affects young males. Enhancement in fracture management was noticed in the last decade. However, an infection with its complications still a concern, especially in case of open fractures for primary closure of the injured area. Newer technique called Vacuum Assisted therapy has become a therapy of choice for many orthopaedic surgeons.

Aim and Objective: To determine whether Vacuum Assisted closure reduces the duration of wound healing along with declines in frequency of infections after fixation of Gustilo Anderson Type IIIA/ IIIB fractures of the extremities.

Methodology: An observational and analytical study was conducted among 34 samples presented with Gustilo Anderson Type IIIA/ IIIB fractures of the limbs presented to department of Orthopaedics, R. L. Jalappa hospital, Kolar from December 2019 to July 2021. Negative-pressure wound therapy for closure of wound after fixation of fractures. Patients were followed up for one month. The outcomes of the intervention are presented both in tables and diagrams.

Results: The mean age of the contemporary data samples was 37.06 ± 10.340 years. The prevalence of infection before Vacuum-assisted closure dressing was 80.6% and the prevalence of infection after Vacuum-assisted closure dressing was 19.4%. The difference in

proportion beforehand vs subsequently the intervention is statistically significant ($p < 0.001$) according to the McNemar Test. Hence Vacuum-assisted closure dressing benefits to decrease the rate of infection. The mean dimension of wound before Vacuum-assisted closure therapy was 66.05cm^2 and the mean dimension of wound after Vacuum-assisted closure therapy was 27.97cm^2. The difference in mean before and after the intervention is found to be statistically significant according to the Paired T Test ($p < 0.001$). Hence, Vacuum-assisted closure dressing helps to decrease the wound size and it was proved statistically.

Conclusion: Vacuum-assisted closure treatment is a viable and beneficial treatment option for complicated fractures with large soft-tissue abnormalities.

Keywords: Negative-pressure wound therapy, Vacuum-assisted closure, wound healing, open fractures.

INTRODUCTION

INTRODUCTION

Compound fractures are more common due to growing trend toward high-speed motor vehicle accidents.[1] Open or compound fractures are those fractures that have an open wound at or closer to the fracture site.[2] Complications encountered in orthopaedic practise due to open fractures can be minimized. They're frequently linked to osteomyelitis and an exalted risk of acquiring deep infections. In open-fractures, the infection percentage was revealed to be 16-66 percent in various investigations.[3–10]

Since the skin is contravened in open fractures, there is an augmented risk of developing infection. The following factors must be considered by the managing surgeon: the illness, the mechanism of damage and the fracture types. Contingent on the characteristics described above, fracture ought to be treated individually.[3]

The Gustilo-Anderson open fracture classification system is commonly used to assess the severity of open fractures. This approach assesses the severity of an injury grounded on wound size, contamination and tissue injury. The practice of external fixation, debridement and a vacuum-assisted closure is illustrated as a management method.[11]

Over the last decade, the practice of Negative-Pressure Wound Therapy (NPWT) treating complex and big wounds has grown in popularity. Modern NPWT systems, which include an open-pore foam sponge, an adhesive dressing, on top of a vacuum pump that produces negative pressure, was employed to indulgence tissue defects surrounding open fractures and chronic, contaminated wounds as an adjuvant to surgical debridement. Accompanying skin grafts and preventing wounds at peril of rupturing are two further applications.[12]

The usage of negative pressure in open wounds is known as vacuum-assisted closure (VAC) and it dramatically promotes wound healing at the macro besides micro levels.[13]

Vacuum-assisted wound closure can be utilised in its place to more traditional wound treatment techniques. Negative pressure allows the wound to heal more quickly or with fewer reconstructive alternatives. Argenta and Morykwas presented the vacuum-assisted closure as a non-pharmacologic/non-surgical method for modifying wound healing in 1997.[14,15] VAC has two advantages: it totally isolates the wound, reducing the possibility of secondary contamination from the environment and it minimises limb oedema. The exclusion of oedema enhances capillary blood flow, which boosts oxygen and nutrition delivery to the wound,[14] because of the capillary ingrowth into the wound, VAC also restricts bacterial multiplication. This gives oxygen as well as international immune cells, which fight bacterial growth.[16] VAC was used for reducing morbidity, expense and hospitalisation time while also improving patient comfort.[17–19]

Vacuum-assisted closure is a commercially existing device that is currently in extensive clinical usage as a dressing for a variety of wound types. Although VAC was first published in the works for open fracture injuries, there is no evidence of its effectiveness in the research done among Indian population.[20]

Quite a few systematic reviews were directed to confirm the effectiveness of NPWT, but they worked on descriptive analysis of retrospective studies and case series and the cases were assorted with other types of wounds such as burns, diabetic ulcers and pressure sores, which had different pathogenic mechanisms and prognoses than open fractures. Though some evidence for NPWT in the dealing open fractures was discovered, the conclusion became very perplexing when it came to specific details for a paradoxical result in prior evaluations. The present literature attempted to expound the effect of VAC-dressing in a big open-fracture in the current investigation.

AIMS & OBJECTIVES

OBJECTIVES

AIM AND OBJECTIVES

1) To describe the clinical outcome which pertain to
   a) Duration for the healing of soft tissues,
   b) Frequency of infections by means of VAC in closure of wound after fixation in management of Gustilo Anderson Type IIIA/ IIIB fractures of the extremities
2) To determine whether VAC reduces wound healing period besides diminution of the frequency of infections in closure of wound after fixation in management of Gustilo Anderson Type IIIA/ IIIB fractures of the extremities.

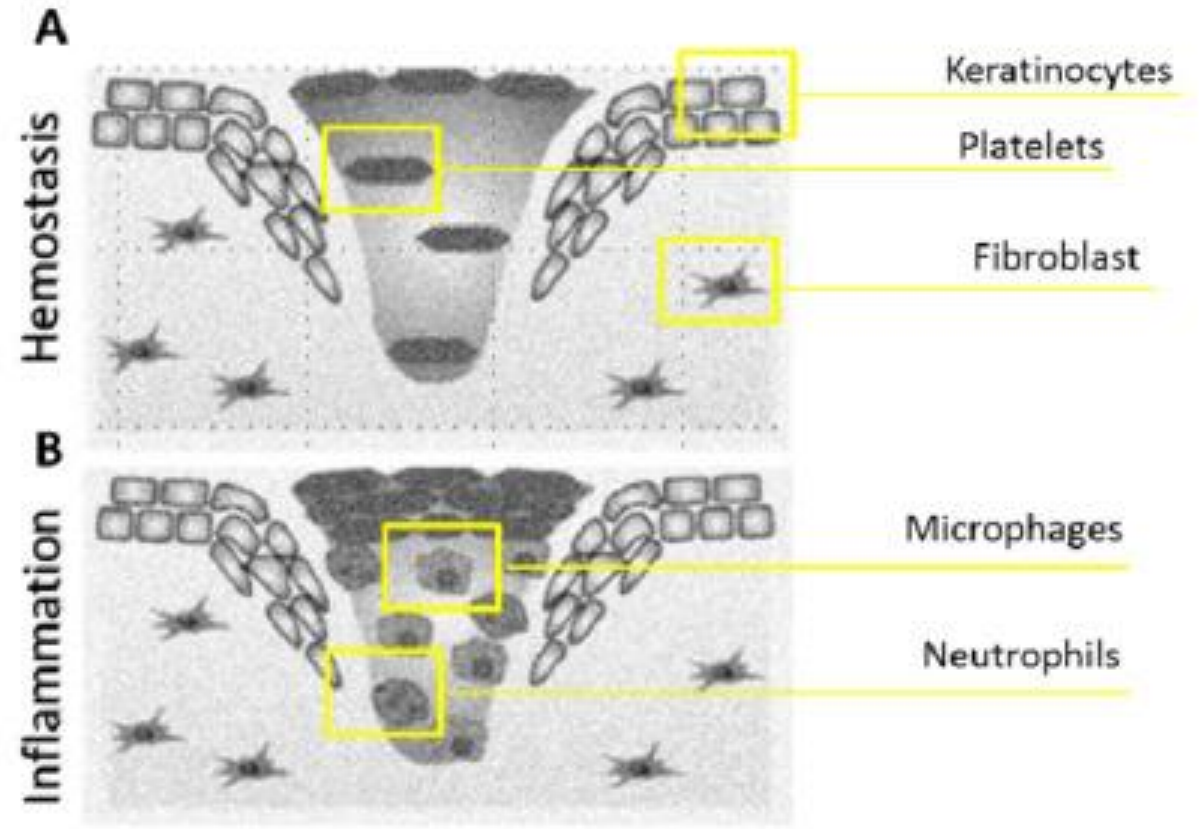
REVIEW OF LITERATURE

REVIEW OF LITERATURE

Wound healing

The term wound-healing is a byzantine besides energetic process that instigates as cell migration and progresses to closure. The process starts with debris removal, infection control, inflammation clearance, angiogenesis, granulation tissue deposition, contraction, connective tissue matrix remodelling and maturation. When this series of events fails, the wound becomes a lingering open wound with no structural or else practical integrity.[21,22]

Phases of Wound Healing



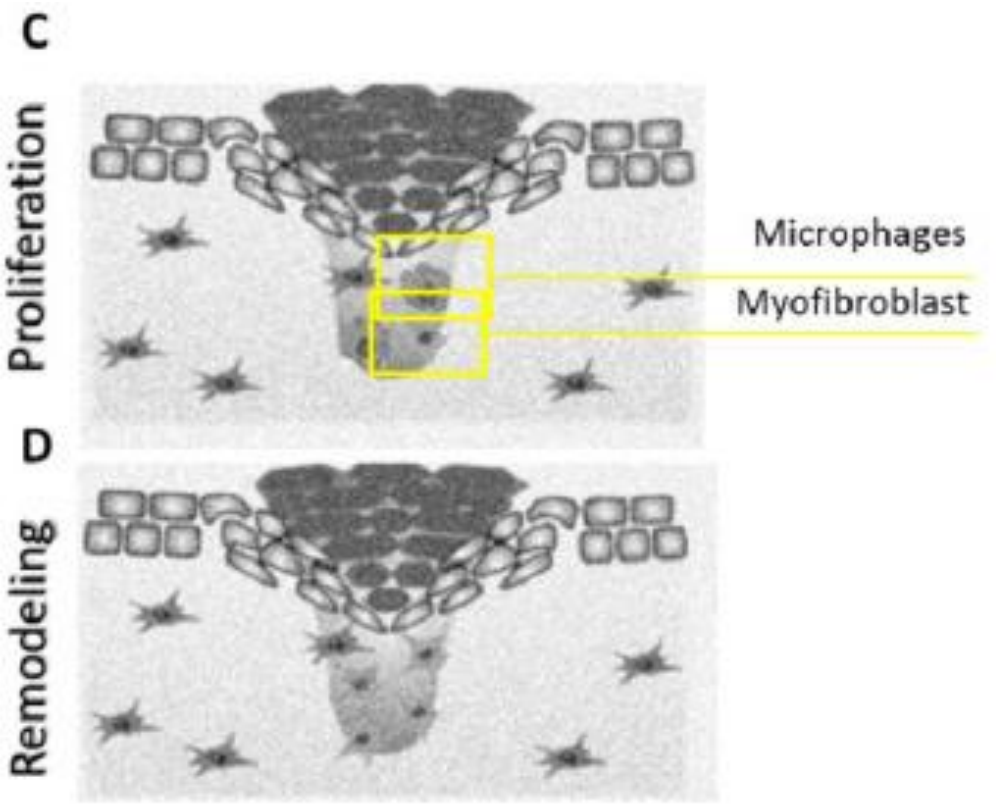


Figure 1: Diagram illustration of wound healing course tangled in each phase.[23]

Wound-healing is a big concern, especially in severe wounds and in the elderly with co morbidities. It causes discomfort, morbidity, lengthy treatment and the necessity for substantial reconstructive surgery, all of which place a significant social and economic incumbrance on the patient.

Problem Statement

As stated by the WHO, 5.8 millions of people die each year as a consequence of injuries (WHO 2014). These deaths make up a minor percentage of the total number of people harmed. Abrasions as well as small skin incisions or lacerations (tears) to wounds with severe tissue damage or loss, traumatic wounds (wounds induced by injury) can be coupled with injury to underlying tissues such as bone or viscera (internal organs). The mechanism of wound influences the degree of tissue damage: blunt trauma, penetrating injury, throng

injury, explosion injury, scalds, de-gloving wound and animal bites are all examples of traumatic wounds. The requirement for immediate assessment and have to management of simultaneous severe, life-threatening injuries frequently dictates initial treatment of traumatic wounds.[24]

Open Fractures along with Soft Tissue Defects

A fracture that is escorted by an open wound at or closer to the fracture site is known as an open (or compound) fracture.[24] Since the skin has been devastated and the opportunity for contamination is great, open fractures can cause severe morbidity and are inherently concerning. The proper and quick treatment of these injuries can support our patients and result in better outcomes.[10]

Although high-energy mechanisms produce the most striking damage patterns, patients frequently present with an open-fracture caused by a simple low-energy mechanism such as a fall. External-fixation and delayed-closure or fixation, as well as prompt irrigation, wound-debridement and primary closure, could all be options for each fracture. In this decision-making process, the condition of the soft-tissues nearby the fracture site is critical, since it usually determines the first care.

The deterrence of contamination, the attainment of bone union and the refurbishment of function are all well-known goals of open fracture therapy. As our knowledge base grows, we continue to study, refine and alter current treatment techniques for open fractures. Antibiotic use, the timing of initial surgical intervention, the type of wound closure, antibiotic delivery techniques, tetanus coverage, wound irrigation and adjuvant therapy to aid fracture union are all important factors to remember.[10]

Classification Systems

Despite the overall increase in outcomes following open fractures, the diverse upshots among distinct forms of varying severity inspired the enhancement of grading systems that categorise them according to the degree of the related soft tissue damage.[25] In the clinical setting, the goal of any fracture classification criteria is to assess the severity and guide in management that infers fracture anatomy and treatment parameters. There are 2 classification systems for open fractures that surgeons managing such injuries should be accustomed about this. The Gustilo categorization and the Mangled-Extremity-Severity-Scale are two of them (MESS).[9,26,27]

The Gustilo classification method was the usual method and is largely acknowledged as the basic open fracture classification system. The energy of the fracture, soft-tissue damage and contamination level are all taken into account by this approach. It has been tweaked since its inception to provide for a more accurate diagnosis of more serious injuries (i.e., Type III injuries).[26,28] The inter observer dependability of this system has been a source of concern in the literature.[29]

Initial efforts by Veliskakis[30] at scoring open fractures were distinguished by Gustilo and Anderson in 1976.[9] Gustilo et al. changed their categorization system into its existing form in 1984 after analysing their first classification of the most serious open injuries.[26] Finally, via their research on the deterrence of infection in open long bone fractures, they were able to arrive at a conclusion,[9,26]

Gustilo et al. defined the treatment guidelines of open fractures providing principles. This classification is excellent for training in the management of patients with orthopaedic trauma since it is a well-known and that has turn out to be the benchmark for diagnosing open fractures.[31]

Table 1: Gustilo open fracture classification system[9,26]

Gustilo type	Definition
I	Open fracture, clean wound, wound length <1 cm
II	Open fractures, wounds longer than 1 cm without severe soft-tissue injury, flaps and avulsions are all examples of open fractures.
III	An open or segmental compound fracture with substantial soft-tissue injury, laceration or loss. Such as farm injuries, fractures that requires vascular-repair and fractures that have been open for more than 8 hours previous to management are all included in this category.
IIIA	Despite severe soft-tissue laceration or destruction, Type III fractures have adequate periosteal covering of the fracture bone.
IIIB	Soft-tissue loss, periosteal stripping and bone destruction characterise type III fractures. It's usually linked to a lot of pollution. Will almost always necessitate a second soft-tissue covering operation (i.e. free or rotational flap)
IIIC	Regardless of the degree of soft-tissue injury, type III fractures are accompanied with an artery injury that requires treatment.

Description of the Gustilo-Anderson Classification

During the inspiring paper[9], although it was communal acquaintance that open fractures compelled immediate debridement and also irrigation, there was a great deal of ambiguity about how diverse forms of injury responded to the therapies available at the time. The unique study[9] comprised an primary retrospective estimation, followed by a prospective test.

The retrospective portion of the study looked at 673 open long-bone fractures in 602 individuals to see how primary vs secondary closure, primary internal fixation and antibiotics

were casted-off in open long-bone fractures. Primary closure short of primary internal fixation as well as prophylactic antibiotics for Type I plus Type II open fractures lowered the chance of infection by up to 84.4 percent, while acute internal fixation as well as primary closure following segmental fractures, wide lacerations, avulsion, or else hurtful amputation increased the risk of subsequent osteomyelitis by up to 84.4 percent.[9]

Gustilo and Anderson then monitored over 350 cases prospectively. They divided open injuries under three categories depending on the dimensions of wound, the presence of osseous injury and extent of contamination, because of the various grievance forms, augmented morbidity from accompanying injuries, widespread soft tissue damage or forfeiture around the fracture sites, poor vascularity, wound infection, or else fracture instability. Infection was found in ten percent to fifty percent of above mentioned Type III open fractures.[26] With such an extended severity, aetiology and prognosis, it became clear that a solitary cataloguing was inadequate; the occurrence of these grievances made the matter much more serious[32]. Then these high-energy open-fractures were again sub classified by Gustilo et al. into 3 types namely A, class B and C apropos the involvement of the soft-tissue injury, necessity for vascular-reconstruction and worsening prognosis, as follows[26]: Type IIIA = open-fractures with acceptable soft tissue treatment of a fractured bone despite widespread laceration of soft-tissue or flaps, or high-energy trauma irrespective of the dimensions of the wound; Type IIIB = open-fractures with widely widespread soft tissue damage loss along with stripping of periosteum and bone exposure. This typically, allied with massive contamination[26]; and Type IIIC = open fractures related with arterial injury necessitating restoration.[26]

Traditional Treatment of Open Fracture Injury

The wound concomitant with an open fracture is typically underestimated in its depiction of the underlying soft tissue injury. Traumatic event to the periosteum, muscle, fascia and subcutaneous tissue is often more severe than expected, particularly when the injury is caused by crushing force.

Over time, the open fracture wound changes and necrotic tissue will be visible. As the wound heals, the amount of exposure of the below structures may change. A wound that is visible during injury is frequently assumed to be amenable to delayed primary closure. However, following many debridement and internal fracture stabilisation, the consequences of changing limb shape and soft tissue swelling affect the wound, necessitating more advanced soft tissue coverage procedures.

An open fracture is notoriously difficult to treat. The early debridement and wound irrigation are the cornerstones of treatment. This procedure is repeated every 48 to 72 hours until the incision is free of contaminants and necrotic tissue.

Traditionally, it was recommended to keep at least the traumatic zone of the lesion open to allow fluid and bacteria to drain from the wound bed. When there are no more symptoms of tissue necrosis in the incision, definitive soft tissue covering can be done. This can be accomplished through delayed initial wound closure, split-thickness skin grafts over a vascularized bed and rotational or microvascular tissue transfers for more severe lesions.[33]

This raises the dilemma of how to appropriately maintain an open fracture wound in the interim between debridement and definitive soft tissue coverage. The perfect open fracture wound management system would inhibit desiccation of vulnerable vital structures, promote vascular ingrowth, remove oedema from the wound bed, restrict bacterial proliferation,

reduce pain, avoid repetitive trauma to the wound bed and avoid secondary microbial contamination from the environment. Modest dressing replaces, skin replacements, as well as antibiotic-impregnated bead pouches are all common ways to treat an open fracture wound.

Traditional wet-to-dry dressing changes have been performed with a number of solutions such as dilute povidone-iodine, dilute bleach, antibiotics, or plain saline. The wound is dressed with a wet dressing and allowed to dry. When the dry dressing is removed, necrotic tissue is removed as well. Dry dressings can be used on wounds that have a lot of exudates. Dressing adjustments provide the benefit of universal supply availability.

Desiccation of exposed structures, skin maceration in wounds with substantial effluent, repetitive wound stress and procedure discomfort are drawbacks. The most serious downside is the high risk of subsequent wound contamination due to recurrent contact to the environment. This is almost certainly a major component in post-traumatic infections.

The usage of skin replacements for the temporary therapy of open fracture wounds is little documented. Xenografts, human allografts and a variety of synthetic membranes are all used as skin substitutes. Allograft and xenograft can be applied on a clean wound bed and adhere to living tissue. Both help to retain the wound saturated, restrict bacterial growth, reduce wound discomfort and protect the underlying components.

Synthetic adherent dressings with an inside layer of collagen that can bond to a clean wound bed and an exterior synthetic layer that is bacteria-resistant are available. Exudate is reduced and underlying structures are protected from desiccation, thanks to the tight adherence. Synthetic membranes, unlike human skin, can be stored at room temperature and have a longer shelf life. Unfortunately, these drugs are not commonly available and can be costly. Furthermore, when these compounds are removed, pieces of collagen may stay in the wound bed and cause persistent inflammation.

An antibiotic-impregnated bead pouch is an effective way to treat an open fracture wound. During the polymerization of polymethyl methacrylate, some antibiotics, particularly tobramycin, can be mixed in and produced into small spheres. The antibiotic will leak from the beads, resulting in bactericidal concentrations in the surrounding tissue.[34] The technique of a bead-pouch was developed by Henry et al., in which antibiotic-bead-chains were put in an open-fracture wound and covered with an occlusive dressing.[35] This approach reduce microbial contamination in the wound. The usage of a bead-pouch as an appendage to debridement and parenteral antibiotics was sighted to diminish infection rates in open fractures.[35–37]

The time-consuming nature of antibiotic bead manufacturing in the operating room is the main downside of this technology. Some have suggested using pre-made chains of antibiotic beads to speed up the process.[38] It's also challenging to get a good seal with the occlusive dressing, especially when dealing with large wounds or wounds that are close to external fixator pins. A wound with an insufficient occlusive dressing might leak antibiotic-rich fluid, cause skin maceration and expose the site to subsequent bacterial contamination.

Recent Literature in management of Open Fracture

In the past, open fracture closure was postponed after initial debridement and closure has been assisted in 7 days if the wound was unsoiled or left open to restore by secondary intention if there is larger soft tissue defect because of fear of gas gangrene.[39] Though, with recent developments in open fracture management comprising initial antibiotics, tetanus prophylaxis and thorough drainage, bothers regarding deep infection in addition to primary wound closure may be less appropriate.[39,40] Current evidence reinforces initial primary closure of open fractures in carefully chosen patients and fractures.[40,41]

In a prospective cohort of type IIIA or lower limb fractures who undertook primary wound closure subsequent surgical fixation, infection rate (4%) and non-union (12%) rates were considerably lesser than a corresponding delayed closure cohort.[40]

Comparably, in a retrospective cohort study conducted by Jenkinson et al, initial primary closure of type I-IIIA fractures was related with a lesser infection rate (4.1%) compared to late primary closure (17.8%).[41] A deep infection percent of 4.7% was also observed in a retrospective assessment of 297 type I-III fractures, which reinforced trying initial treatment for all open fractures.[42]

Prompt primary closure necessitates the orthopaedician to decide that the open fracture to be "adequately debrided," a judgement that involves experience.[39,40] Mostly Gustilo and Anderson type 1 and type II open wounds can be closed primarily subsequent systematic Drainage. Mostly Gustilo and Anderson type IIIA wounds can be closed following a repeated drainage, if clean and tension-free closure can be accomplished. Clear contraindications to initial primary closure comprise gross contamination with faeces, filth, stagnant water, agriculture related injuries, freshwater boating calamities, antibiotic commencement more than 12 hours post-injury, or doubtful soft tissue viability during preliminary I&D.[42] Open fractures with linked irreducible joints and bare articular cartilage necessitate judicious surgical management.[43]

Negative-pressure dressings

Negative-pressure dressings are a popular choice for treating open fracture wounds in the interim. A variety of systems are now available on the market. They all have a base unit pump that provides negative pressure, a canister that collects wound drainage and a length of tubing that connects this to the sealed wound. The NPWT device operates by applying and dispersing negative pressure equally across the wound bed, either with an open cell foam

dressing or a gauze dressing.[44] Foam and gauze have both been demonstrated to be similarly efficacious at wound contraction and blood flow stimulation at the wound edge.[45] Foam has been demonstrated to have a quick granulation time.[46] However, ingrowth, which has the ability to disrupt the epithelialization process and be unpleasant when the foam is changed, can counteract this.[47–50]

The negative pressure dressing is made out of a polyurethane ether foam sponge that is trimmed to fit the shape of the wound, as seen in Figure 1. A non-collapsible evacuation tube is inserted into the sponge. The effluent is collected in a canister linked to the evacuation tube.

The control box, which regulates the force given by the dressing, is connected to the canister. An occlusive drape is used to keep the dressing in place (Figure 1). For the first 48 hours, the normal setting is 125 mm sub-atmospheric pressure, followed by intermittent mode (5 minutes on, 2 minutes off) for the remainder of the therapy.

This cyclic phase has been shown in clinical investigations to speed wound healing. To prevent aggressive granulation tissue ingrowth into the sponge, the dressing is changed every 48 to 72 hours. This is in line with the debridement schedule.

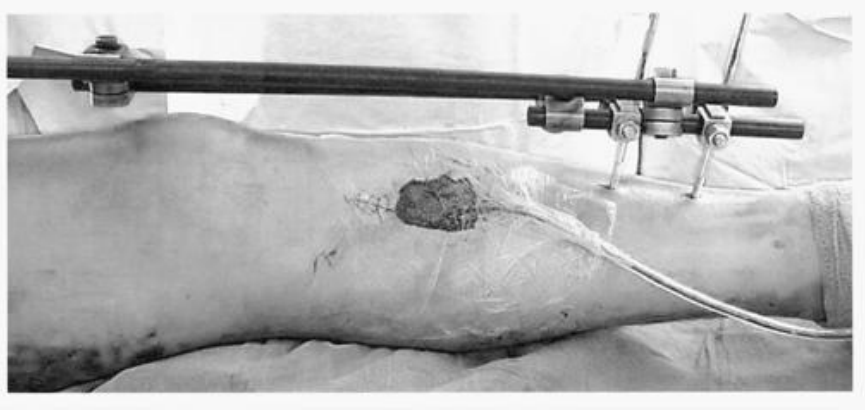


Figure 2: The negative pressure dressing[16]

The open fracture wound has been dressed with a negative-pressure dressing, as shown in Figure 2. The sponge has been shaped to meet the wound's contours. The sponge is linked to a source of constant sub-atmospheric pressure via an evacuation tube. The dressing was done with a sterile occlusive drape.

Negative pressure dressing is employed to treat long-lasting wounds caused by pressure, venous stasis, radiation, diabetes mellitus, as well as vasculitis. Dressing will be endured as the wound heals elsewise granulating bed can be addressed with a less invasive method like split-thickness skin grafting.[14] Negative pressure dressings were used to treat dehisced as well as contaminated operative injuries in the chest and abdomen. Therapy is repeated until a split-thickness skin graft can be applied to the bed.[14,51] This is a significant benefit for individuals who are frequently ineligible for standard wound treatments such free microvascular tissue transplants. Acute wounds such as major soft tissue avulsions and gunshot wounds have been effectively treated with negative pressure dressings.[14]

Negative pressure dressings have also been demonstrated to be effective limb-threatening injuries. Negative pressure dressings were casted-off on a cluster of patients failed free flap covering, wounds overlarge to fully cover with free flaps, or infected wounds expecting for free flap coverage. Despite the fact that some patients required extra surgical procedures, no amputations occurred and all patients had appropriate soft tissue coverage.[16]

Mechanism of Action of NPWT

- Macro-deformation of the wound occurs when the suction spread via the foam sponge brings the wound edges closer together, depending on the deformability of the surrounding tissues. This cuts down on the expanse of area that needs to be healed through primary closure or secondary granulation (See Figure 2).

- Microscopic distortion of the wound surface. NPWT causes 5-20% strain across the healing tissues, which surges cell division and proliferation, growth factor production and angiogenesis, according to computer models.[52]
- Removal of oedematous fluid and exudate from the extracellular space, as well as inflammatory mediators and cytokines, which have a long-term effect on the microcirculation's ability to maintain damaged tissue. This can result in further tissue necrosis, which is commonly seen after more debridement.
- A warm, wet environment that prevents the wound from drying up and indorses evolution of granulation tissue.[53] (See Figure 3).

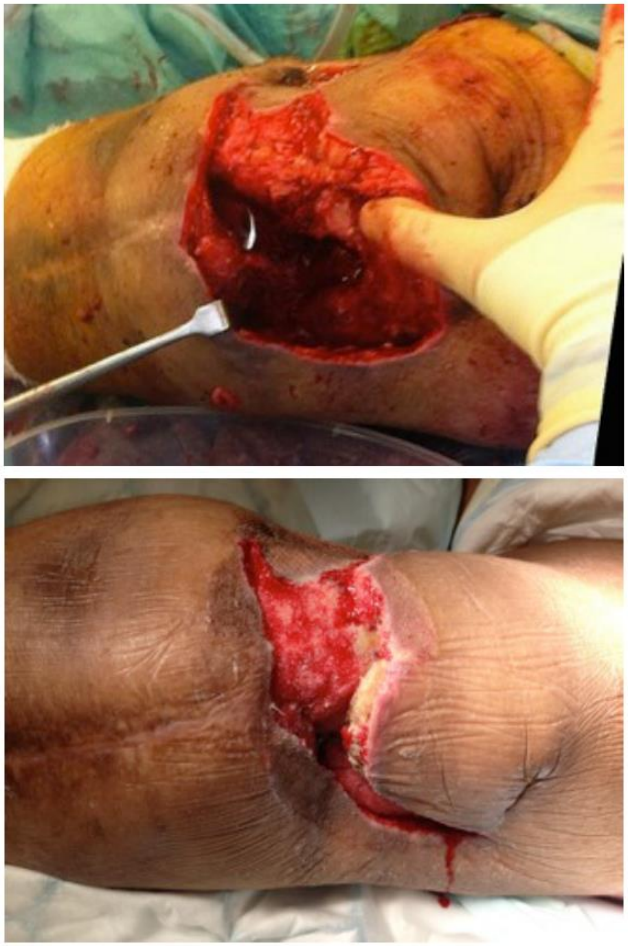


Figure 3: NPWT dressings can fetch wound edges nearer unruffled and endorse the production of granulation tissue in large wounds

Advantages of NPWT

Negative pressure dressings provide various advantages for acute open fracture wounds. First, the occlusive dressing totally isolates the wound from the environment, reducing the danger of secondary contamination. The presence of oedema in the wound bed raises tissue pressure, delaying capillary inflow and obstructing venous and lymphatic outflow. The negative pressure dressing reduces oedema, which improves capillary blood flow and promotes oxygen and nutrition delivery to the wound.[14,16] Compounds that are harmful to wound healing are also removed when oedema fluid is removed. The growth of keratinocytes, fibroblasts and vascular endothelial cells has been demonstrated to be suppressed by factors eliminated from chronic wound fluid.[14,54,55] This fluid also contains elevated quantities of proteases (e.g. collagenase, elastase) and their breakdown products.[14,56–58] Second, bacterial reproduction is restricted. Capillary ingrowth occurs due to the dressing. This increase in vascularity allows more oxygen and immune cells to enter the body, which inhibits bacterial growth. Despite the fact that the wound remains colonised, wounds managed with negative pressure dressings show a reduction in bacterial load after 3-4 days. Bacteriological colonisation has been measured at 102-103 organisms per gramme of tissue in quantitative cultures. Bacteriological tallies of a lower amount than 105 organisms per gramme of tissue are associated with successful wound healing,[14,59] finally, applying mechanical stress to a wound creates a favourable environment. When negative-pressure is given to a lesion, the rate of cell mitosis, the development of new blood vessels and the recruitment of nearby cells all increase.[14,60–62] It is not uncommon for the evolution of granulation tissue to be so aggressive in this favourable environment that a less invasive surgical procedure, such as a split-thickness skin graft, used for achieving soft tissue coverage rather than a more taxing procedure, such as a microvascular tissue transfer. The principal drawback of this method is that it necessitates the use of specialised equipment.

Applying the dressing has a steep learning curve, especially with big wounds, multiple wounds and wounds around external fixator pins.

Disadvantages of NPWT

Negative pressure dressing treatment can lead to complications. Some individuals experience agonising pain . This normally goes away with time. The majority of other issues stem from incorrect dressing application. With a bandage that reaches to the undamaged skin, erythema around wound borders can be detected. This erythema may be confused for cellulitis, but it is actually hyperaemia and it will go away soon once treatment is stopped. If the occlusive drape is applied excessively firmly in this area, skin necrosis surrounding the evacuation tube has been reported. Granulation tissue ingrowth into the sponge has been observed, most frequently duration of dressing lasting more than 72 hours.[14]

Following that, the Food and Drug Administration (FDA) published a guidelines for healthcare practitioners for how to use NPWT devices.[63] by using this online reference users are recommended to:

'Receive proper instruction on how to use the device, including its indications and contraindications, as well as how to recognise and treat potential consequences.' NPWT training for patients and their caregivers who will be using this device at home should strictly follow how to:

- Operate the device safely offer a copy of the manufacturer's printed instructions for patient use
- Respond to auditory and visual warnings
- Change dressings

- Recognize the signs and symptoms of infection, such as redness, warmth and discomfort
- Contact relevant healthcare providers, particularly in emergency scenarios
- Respond to emergency situations; for example, if bright red blood is observed in the tubing or canister, cease NPWT immediately, apply direct manual pressure to the dressing and refer.'

Vacuum-assisted closure therapy

The method negative pressure therapy was first utilised to speed up wound preparation at the bedside. Morykwas in addition Argents used animals in their research to see how topical negative pressure therapy affected local blood flow, granulation tissue formation, bacterial clearance and flap survival. They then employed a foam dressing to control wounds, which allowed them to alter the vacuum pressure and choose between continuous and intermittent modes.

Clinical indications

Vacuum assisted closure therapy has been casted off in a variety of medical and surgical disciplines, demonstrating its effectiveness in acute as well as chronic wounds as well as post-operative rehabilitation. The indication for its usage in orthopaedic trauma departments first centred on open fractures with soft tissue defects, but it's now commonly utilised in contaminated wounds and more recent research is accumulating on its capacity to help closed incisions with a high chance of wound breakdown. Its effectiveness on skin grafts is now well established.[12]

Contraindication

- Malignant wounds
- Untreated osteomyelitis
- Fistulae to organs or bodily cavities
- Necrotic tissue
- Exposed arteries/nerves/anastomotic site/organs
- Blood dyscrasias, those on anticoagulants
- Actively bleeding wounds
- Nearby invasive-sepsis
- Augmented pain
- Symptoms of infection such as fever, pus or foul-smelling drainage
- Allergic-reaction to the adhesive[64]

Complications

- Failure of the VAC-system
- Wound-infection
- Pain
- Bleeding
- Allergies to the adhesive-drape
- Skin excoriation
- Restricted mobility
- Tissue-adherence to the foam
- Lack of most patients' compliance
- Skin necrosis.[65]

Benefits of VAC therapy

- Reduced dressing changes
- Patient comfort
- Shorter hospital stays
- Lower bacterial-load
- Increased skin perfusion
- Reduced oedema
- The provision of a closed moist wound-healing environment

Mechanism of action

Vacuum assisted closure therapy has been proven in human and animal research to improve granulation tissue growth, blood flow, wound area reduction and inflammatory response modulation.[15,66] VAC produces wound contraction, wound environment stabilisation, decreased oedema due to wound exudate clearance and cell micro-deformation. Because of these benefits, VAC can speed wound healing by increasing blood flow, reducing bacterial load and bettering wound bed preparation for following coverage.[67,68]

Negative pressure compression promotes the development of hypoxia due to decreased perfusion beneath the foam, which stimulates angiogenesis and local vasodilation due to nitric oxide production.[69–71]

Following injury, oedema is caused by hypobaric interstitial pressure besides increased vascular permeability.[72] Higher tissue pressure induces vessel compression and augmented intravascular fluid velocity, that lowers intravascular hydrostatic pressure. These two principles cause result in a decrease in intravascular fluid outflow and oedema. Furthermore, increased blood velocity causes extracellular fluid to be drawn inside the vessel. Furthermore, negative-pressure wound therapy's compressive energies physically push

oedema outside the wounded tissues. VAC therapy immobilises the wound, which aids in healing.[73,74]

Vacuum assisted closure produces microdeformation/ microstrain of cells, which results in tissue expansion and also the release of growth factors.[52,75,76] However, according to a recent study, the pressure in the basic injury is strangely raised.[77] Normal tissue has a capillary perfusion pressure of 10–35 mmHg.[64]

Optimum negative pressure

The best way to use negative pressure is a source of debate. When compared to low vs high vacuum suction, studies on animal models showed that 125 mmHg vacuum resulted in more granulation tissue formation. Low-pressure suction (25 mmHg) causes less fluid to drain from the wound, less toxins to be removed and less cell deformation. They concluded the pace of granulation tissue production is slowed. The higher suction pressure of 500 mmHg induces greater mechanical deformation of tissues, resulting in localised perfusion loss and granulation tissue development reduction. As a result, a negative pressure of 125 mm Hg is deemed optimum.[78]

Various degrees of negative pressure (10–175 mmHg) had different effects in different wounds. Negative pressure (125 mm Hg) is required for acute traumatic wounds, while 50 mm Hg at intermittent cycles is recommended for chronic non-healing venous ulcers.[79,80]

Intermittent versus continuous VAC

Intermittent negative-pressure is advised because it increases blood flow during the "off" part of the vacuum. The pace of granulation tissue production is twice as fast with intermittent negative-pressure compared to continuous negative pressure, according to studies. (Intermittent: 103 percent vs. continuous: 63 percent).[15]

Air leaks in the dressing should be prevented because they allow air to flow continuously across the wound surface, causing tissue desiccation and the formation of eschar. This eschar closes the wound with trapped exudate, causing the wound to deteriorate.[81]

Randomised controlled trial used VAC for severe wounds, there is Grade "C" evidence, as a connecting remedy amongst numerous debridement is evaluated as Grade "B." Only for the administration of skin grafting techniques is a strong recommendation (Grade "A") proposed.[82]

Cost

Many researches on various wounds imply that VAC may be more cost-effective than traditional wound care treatments since it involves fewer dressing changes and fewer wound reconstruction alternatives. Wound recovery is faster and treatment and hospitalisation are shorter overall. Although VAC dressings are more expensive than standard dressings, the overall cost of therapy with VAC is lower in the long run.[83,84]

Figure 4: Nearby obtainable material to gather VAC dressing[64]

Figure 5: Pre-operative fractured wound of foot[64]

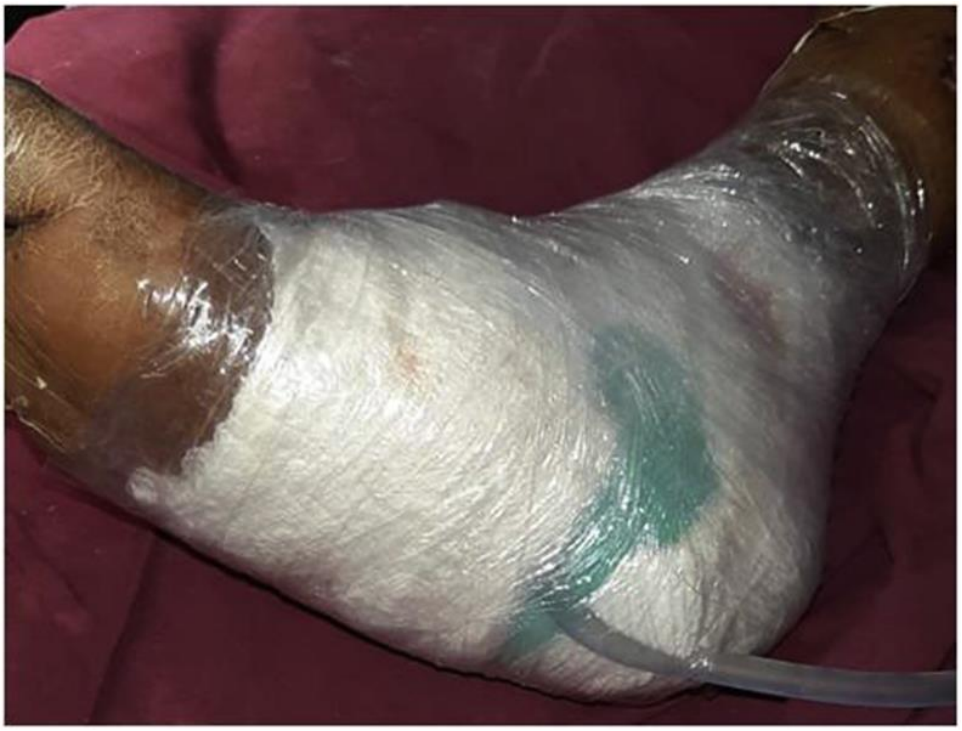


Figure 6: Indigenously prepared VAC in place[64]

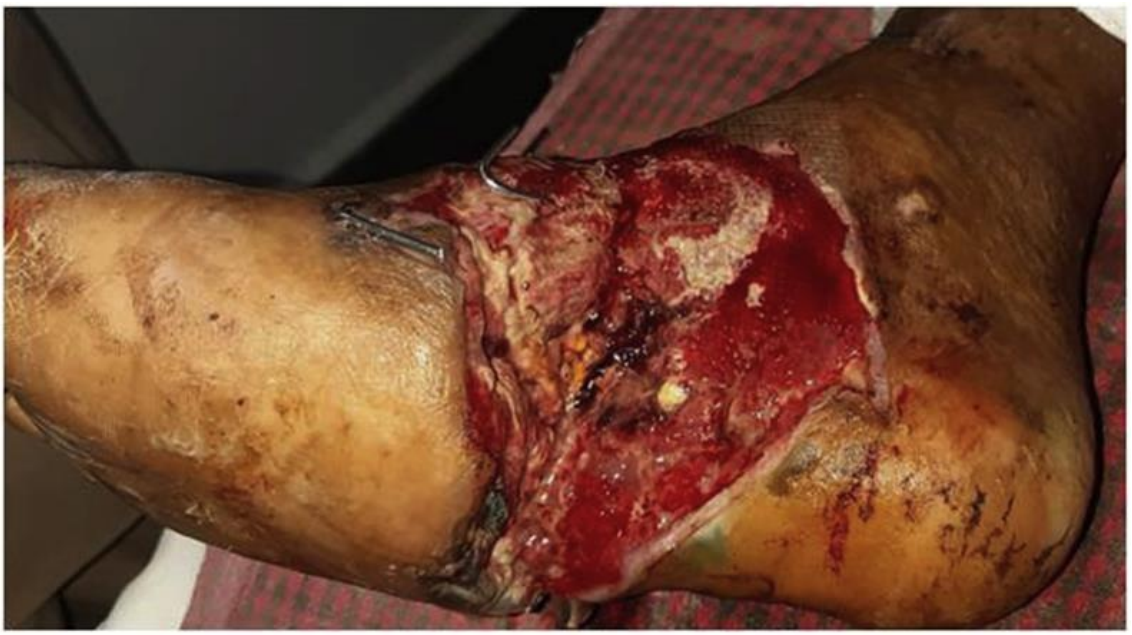


Figure 7: Post VAC lesion following two sessions[64]

Articles describing the role of VAC Dressing in Large open fractures

1. A Prospective Study with a Single Center Himanshu Suman et al. from Indore[3] investigated 30 patients with major open long bone fractures who got VAC therapy during the early stages of treatment. Following percutaneous pinning or external fixation, a VAC dressing was done. They discovered that the average time to the first debridement was 8.20 hours (range: 2– 23). The VAC treatment took ten days on average (range: 3–16). In nine cases, overdue wound closure was used. The mean reduction in wound dimensions between pre- and post-VAC administration was 43.06 percent (range, 20– 60 percent) in the remaining 21 patients. VAC application enhances blood circulation, hastens the creation of granulation tissue, decreases the amount of infection from the hospital environment, minimises oedema, eliminates the requirement for subsequent interventions and reduces wound dimensions, according to the study.
2. The WOLLF Randomised Controlled Trial[85](RCT) compared outcomes in 226 people who received negative pressure dressings with 234 who received standard dressings. All of them had a significant open fracture that had not been sewn up. The primary outcome was a 12-month self-reported Disability Rating Index score on a scale of 0 to 100, with higher scores representing more disability. There were no clinically significant changes in impairment, deep infections or healing between the groups who received negative pressure dressings and those who received normal dressings, according to the researchers. At 12 months, the negative pressure group had a mean Disability Rating Index score of 45.5, while the usual therapy group had a score of 42.4 (adjusted mean difference 3.9). Deep wound infections occurred in 7.1% people in the NPWT group and 8.1% people in the conventional care group after 30 days (difference 1%). By six weeks, 52 percent of the NPWT group and 51.7 percent of the

usual treatment group had healed their wounds (odds ratio 1.0). Throughout the study, both groups' quality of life was similar.

3. Stannard et al.[86] studied 62 severe open fractures, all getting an initial irrigation as well as debridement and returning to theatre every 48-72 hours till wound closure. 37 fractures were randomly assigned to NPWT intervals, while the remaining 25 were treated with normal fine mesh gauze. There were considerably fewer infections in the NPWT-group than in the control group (0 acute and 2 delayed versus 2 acute and 5 delayed, p=0.024). Twenty-one of the 58 patients in the study received a rotational, free flap or skin transplant, however the infection incidence in this group was not examined individually.
4. An additional study by Sinha et al.[22] randomised 30 open musculoskeletal injuries to NPWT dressings substituted every 3-4 days or standard dressings daily. On days 4 and 8, after the first debridement, measurements were taken every time the dressings were changed and tissue samples were taken for histological analysis. The NPWT-group had a significantly smaller wound (mean 13.24 mm against 3.02 mm, p=0.0001), a much smaller percentage of bacterial growth (60 percent no growth versus 20 percent) and substantially increased angiogenesis, granulation-tissue and fibrosis (Wilcoxon signed-rank test p 0.05). All of the patients recovered without infection, with the exception of one who required a free flap.
5. As an illustration of the ability of NPWT to inspire the development of granulation tissue over extended periods, Lee et al.[87] prospectively managed 16 patients with open wounds in the site of foot region and ankle region with exposed tendon or bone. On treatment, after the initial debridement, NPWT was employed and dressing change in every 3-4 days for 11-29 days and 15 of the 16 patients healed by

secondary intention (granulation tissue development), with just one instance requiring a free flap. There were no infections reported.

6. Blum et al.[20] retrospectively reviewed 229 open tibial fractures with 72% getting NPWT and 28% conventional dressings. They discovered that the NPWT group had a lesser percent of deep infection (8.4% versus 20.6%) (p=0.01). When the Gustilo classification, a univariate predictor of deep infection, was adjusted for the brutality of the injury, NPWT was found to lower the probability of deep infection by nearly 80%. Even when taking into account the substantially greater rate of free flaps in the NPWT group (28 percent versus 14 percent, p=0.03), this is an exceptionally high figure.
7. Over a comparable retrospective period in the unchanged trauma centre, Liu et al.[88] from the Department of Plastics and Reconstructive Surgery found that soft tissue coverage within 3 days of injury and instantly following fracture repair with exposed metal ware reduced pre-flap wound infection and improved surgical results in open lower limb injuries. NPWT offered good interim wound coverage and did not cause a delay in the rebuilding of the free flap.
8. The study conducted in Turkey[89] included a total of 21 patients (4 female, 17 male with a mean age of 27 years, range: 3–64 years) with Gustilo-Andersen Type 3 open fracture were involved. Follow-up lasted an average of 25.67 months (range: 9–52 months). Thirteen of the patients had Type IIIB fractures and eight had Type IIIC fractures. In an operating room setting, wound dimensions were measured following a substantial debridement and irrigation. After then, VAC was used. The average time between the trauma and the operation was 7.57 hours (range: 2–23 hours). In seven cases, the incision was treated with delayed primary suturing. The wound dimensions measured after the last VAC application were noticed as reduction

in mean of 40.02 percent in the remaining 14 patients (range, 20-60 percent). Seven patients received a skin graft, two received a free flap, four had a fasciocutaneous flap and one received a fasciocutaneous flap + graft. Due to graft failure, two patients had to have revision surgery. A deep wound infection occurred in five patients (23.8%). Two patients had osteomyelitis as a result of the infections (9.5 percent). They found that combining VAC with skin traction sutures reduces the wound size dimension of a graft or flap to be applied in Type IIIB and C open fractures.

9. A systematic-review and meta-analysis done by Liu et al[90] observed that the 8 RCTs with 421 patients and the 6 retrospective and 14 cohort studies with 488 patients, the NPWT led to a decreased infection rate, briefer injury coverage time, wound healing time and hospital stay length, as well as a lower amputation rate. However, there was no statistically significant difference in the requirement for flap surgery, the proportion of free flaps, the incidence of flap failure, or the chance of fracture non-union. Only one RCT found that infected individuals had a higher physical component score on short form 36. They came to the conclusion that NPWT can considerably lower the chance of infection in open fracture treatment while also speeding up the wound healing process. Some data, but not much, proposes that NPWT might support minimise the severity of limb injury and hence provide the limb a chance to prevent amputation. Although the use of NPWT in the flap area is likely safe, it should be done with caution. The advantage of NPWT over traditional wound dressings still needs to be proven in other areas.
10. In a prospective randomized clinical trial study[91], 90 individuals of open fractures who were enrolled in the study, were divided into two groups. Of which, group I received NPWT, while group II received standard wound care. After that, patients were observed for a month. The number of dressing changes during the month varied

depending on the dimension of the wound. P value of 0.05 revealed a significant difference in wound healing rates between group one (NPWT) and group II (traditional wound dressings). The frequency of infection did not change significantly between those two groups (P=0.6). NPWT speeds up the healing of extremities wounds. It is less expensive and can be utilised to treat wounds on the extremities instead of surgery.

11. Joethy et al[92] conducted a study in which they concluded that prevalence of infection decreased and failure of flap procedure also diminished in NPWT groups.

MATERIAL & METHODS

MATERIALS AND METHODS

STUDY DESIGN:

The observational-analytical study was conducted among patients with closure of wound by using VAC in treatment of Gustilo Anderson Type IIIA/ IIIB fractures of the limbs.

STUDY AREA:

Department of Orthopaedics, R. L. Jalappa hospital attached to Sri Devaraj Urs Academy of Higher Education and Research Tamaka, Kolar.

STUDY PERIOD AND DURATION:

From December 2019 to July 2021 for a duration of one year eight months

STUDY POPULATION:

All patients admitted to R. L. Jalappa hospital and diagnosed with Gustilo Anderson Type IIIA/ IIIB fractures of the limbs presented to department of Orthopaedics during the period between December 2019 and July 2021.

SAMPLE SIZE CALCULATION

Calculated based on the Infection Rate as observed in a study on "Negative Pressure Wound Therapy in Grade IIIB Tibia Fractures: Fewer Infections and Fewer Flap Procedures"?[93] where it was observed that infection rate was 5.4% with 8 % absolute error.

$$\text{Sample size} = \frac{z^2 1-{}^{a}/_{2} P(1-p)}{d^2}$$

p: Expected proportions of 5.4% infection rate

d: Absolute precision = 8%

1-α/2: desired confidence level = 1.96 (95% confidence level)

Z : confidence interval

The estimated sample size was 31 expecting a drop rate of 10% during the follow up. The final sample size as calculated as 31 + 3 = 34.

SAMPLING METHOD:

All patients admitted to RLJ hospital during the period between December 2019 and July 2021.

INCLUSION CRITERA:

Patients aged between 18 to 60 years with Gustilo and Anderson type IIIA/IIIB fractures of the limbs presenting to the hospital.

EXCLUSION CRITERIA:

- Patients with bone shortfall
- Patients on immunosuppressive drugs , steroids and anti coagulants therapy
- Patients noticed to have peripheral vascular disease.
- Anemia
- Patients with bleeding diathesis.

METHOD OF DATA COLLECTION

Open wound scoring system used[87]

Score (grade)	Status of wound
0	Closed wound
1	Skin or soft tissue defect
2	Bone, tendon, implant exposure (any 1)
3	Bone, tendon, implant exposure (any combination of 2 or more) Associated
4	Associated or Residual infection quencies

Gustilo - Anderson classification system was employed to evaluate fracture type among all participants. 34 Patients having Open Type IIIA/IIIB limb fractures of Gustilo Anderson Classification after satisfying the inclusion criteria and informed written consent was take into account for the study. A thorough clinical history, clinical examination and investigations like CBC, BT, CT, Blood Grouping and Rh typing, Culture sensitivity of the wound swabs and x-rays was examined.

Internal fixation or External fixation of the fracture was completed within 72 hours. or else subsequently thorough debridement of compound fractures and achieving a unsoiled wound with skin and soft tissue loss, sponge foam was hired on the wound. Adhesive drape was utilised to conceal the wound. Ultimately, the inner end of a suction tube was acquaint within the dead wound space and the outer end of it was linked to the device. Wound dressings were replaced every 48 hours and negative pressure nonstop for 10-14 days. Pressure was

sustained at 125 mm Hg continuously or intermittently 5 minutes on followed by two minutes off.

Patients were followed up for one month. Patients were advised to come to the hospital for scheduled check-ups after being discharged and all participants were followed up during the study. Wounds were inspected weekly and following, measurements was documented presence of granulation tissue, wound bed becomes healthy, reduction in wound drainage and reduction in dimensions of wound. Intervention therapy was completed when adequate granulation base is accomplished.

The size of wound was quantified by placing two pieces of transparent plastic sheets directly on the wound and marked the outline of the wound with a permanent ink marker on the outer sheet.

The inner plastic sheet was casted-off. The outer plastic sheet with wound outlined was placed on calibrated graph paper. The size of the wound was then quantified by the greatest diameters horizontal and vertical measurements were taken.

Follow up was done on once in a week till complete wound healing which may be either primary or by utilizing secondary interventions by tissue transfer. Antibiotics was given according to the culture and sensitivity report.

STUDY VARIABLE

- Age
- Gender
- Mean duration of hospital stay
- Site of fracture
- Type of fracture
- Mean area of wound before and after VAC application
- Type of fixation

- Wound complications
- Duration of VAC therapy
- Duration of follow up
- Time between initial trauma and initial debridement
- Pre-existing co-morbidities
- Wound infections before and after VAC application
- Number of dressings
- Duration of wound healing

ETHICAL CONSIDERATION

Ethics approval was attained from Ethics Committee. All ethics morals were followed in the study. The composed data was utilized only for the anticipated purpose of the study. The dignity and welfare of participants were shielded at all times from ethics point of view. The research data remained censored throughout the study and the researcher obtained the participants consent to use their real names in the research report.

STATISTICAL ANALYSIS:

- Data entered in MS excel spread sheet and analysed using IBM SPSS 23 software.
- Discrete data like gender, infection rate, type of fractures etc were presented by frequency and percentages.
- Quantitative measures like age, wound size, duration of hospital stay, reduction in flap procedures etc were presented by mean, standard deviation and confidence interval.
- Mc Nemar test was used for test of significance to compare difference in proportion.
- Paired t – test was used for linking the difference in wound size.
- Probability (p) value less than 0.05 will be considered as statistically significant.

RESULTS

RESULTS

1) Descriptive statistics:

Socio-demographic profile:

Comment: The mean age was 37.06 years with a standard deviation of 10.340 as shown in the below table and diagram.

Table 2: Age distribution of the study participants (n=34)

Age	
Mean	37.06
Median	36.50
Mode	36
Std. Deviation	10.340
Minimum	20
Maximum	59
Interquartile range	29.75 - 45.00

Figure 8: Age distribution of the study participants (n=34)

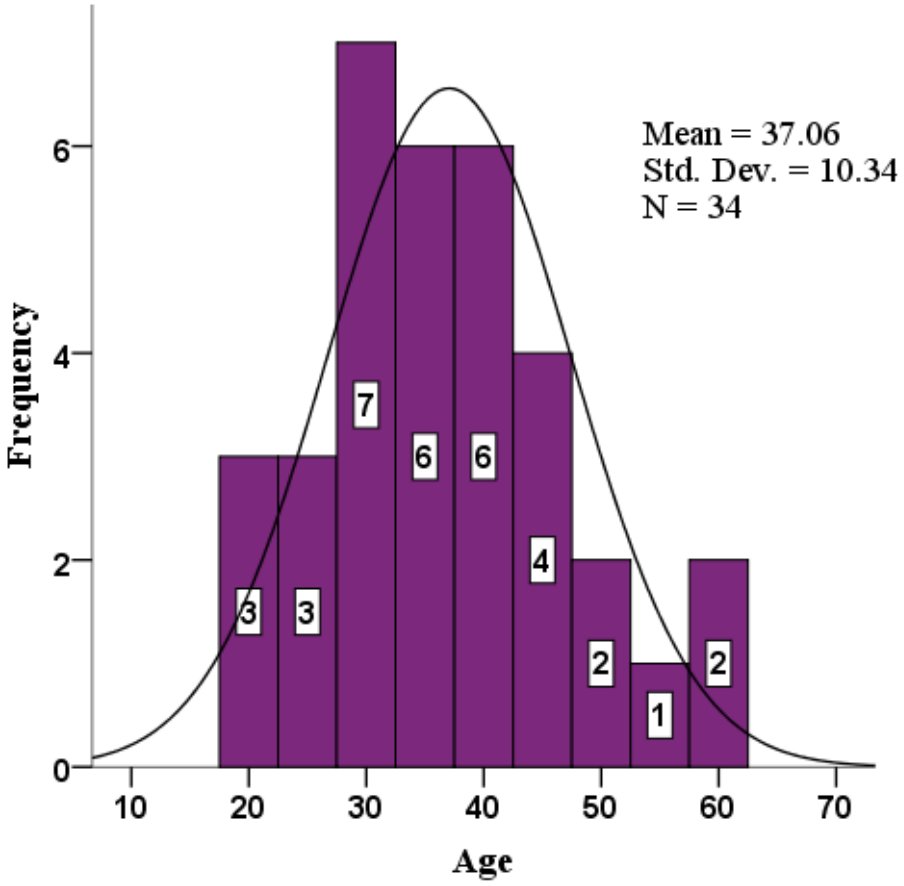


Table 3: Gender distribution of the study participants (n=34)

Gender	Frequency	Percent
Female	8	23.5
Male	26	76.5
Total	34	100.0

Comment: Nearly 76.5 percent were males and the remaining were females as shown in the table and diagram.

Figure 9: Gender distribution of the study participants (n=34)

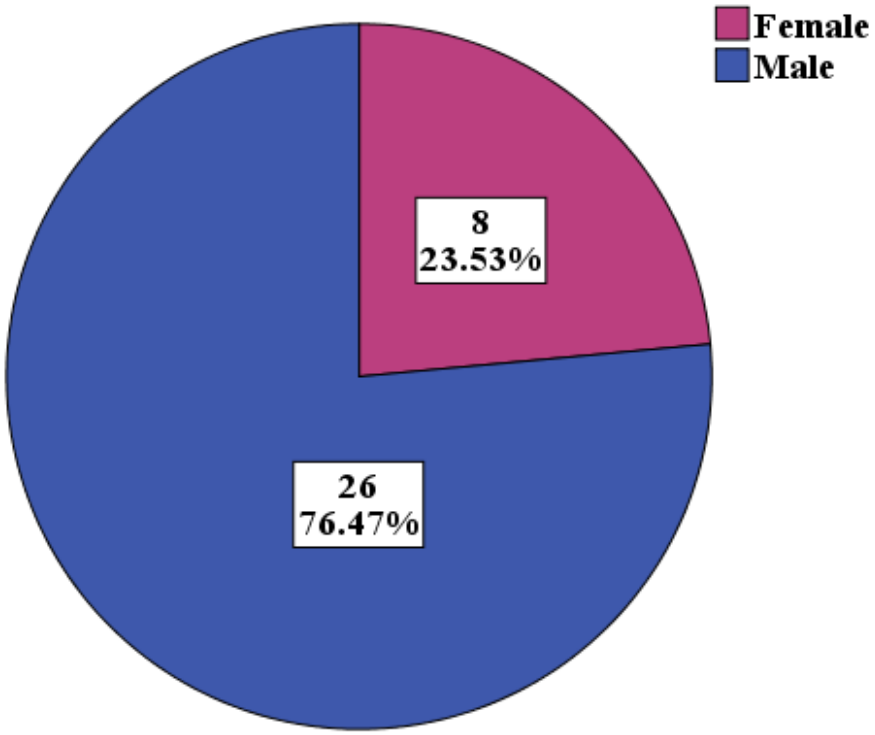


Table 4: Distribution of study participants according to the place of trauma (n=34)

Type of injury	Frequency	Percent
RTA	27	79.4
Work place injury	7	20.6
Total	34	100.0

Comment: About 79.4 percent of trauma was due to road traffic accidents and the remaining as workplace injury as shown in the table and diagram.

Figure 10: Distribution of study participants according to the place of trauma (n=34)

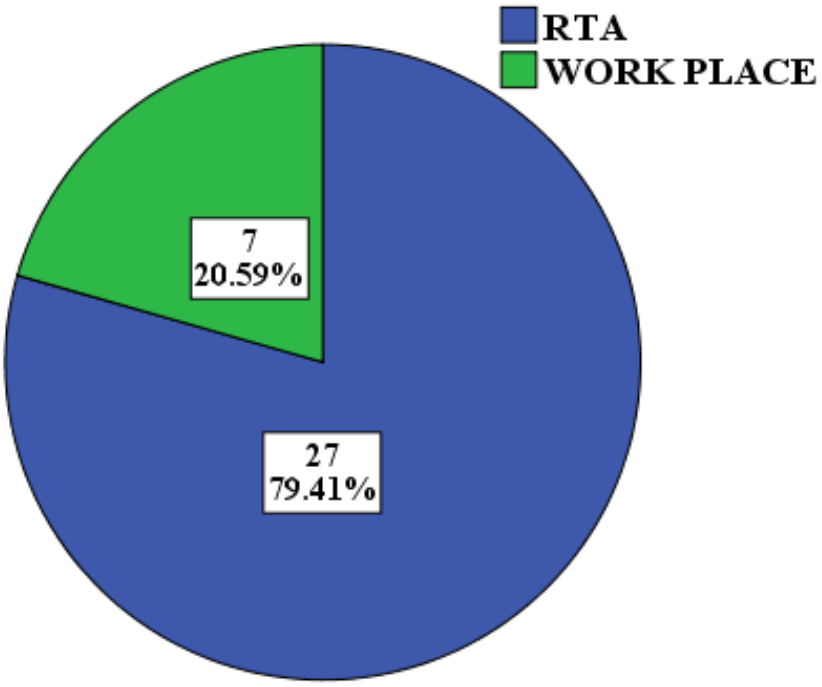


Table 5: Distribution of study participants according to the type of fracture (n=34)

Type of Fracture	Frequency	Percent
3A	12	35.29
3B	22	64.71
Total	34	100.0

Comment: About 64.71 percent of the individuals were classified as Gustilo Anderson type 3 B and about 35.29 percent were classified as 3A as shown in the table and diagram. In the study participants, there was no neurovascular defect found.

Figure 11: Distribution of study participants according to the type of fracture (n=34)

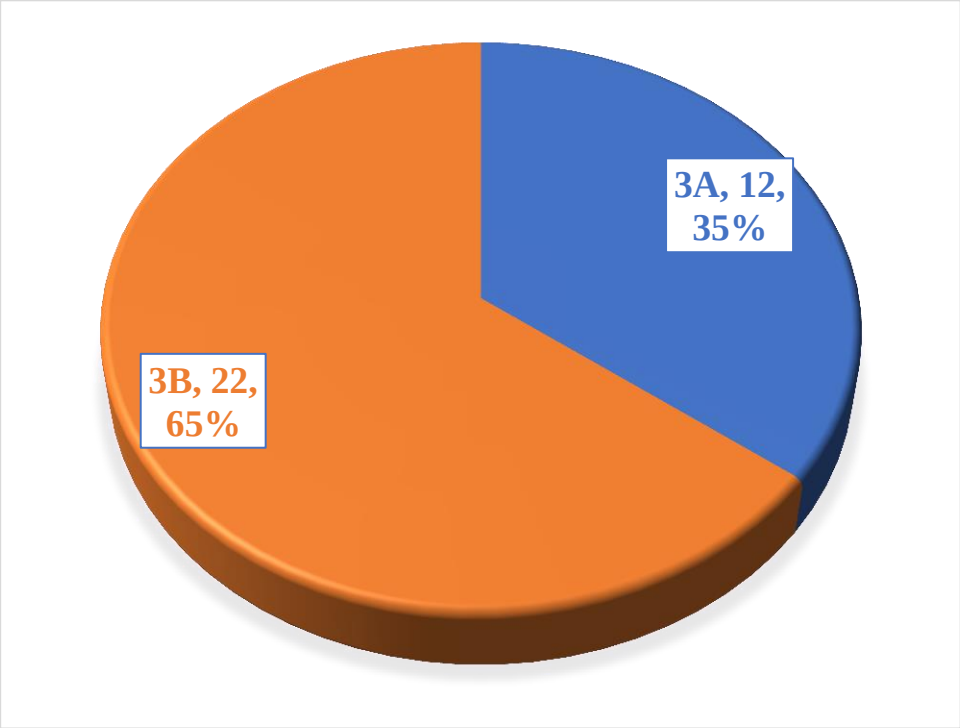


Table 6: Distribution of study participants according to open wound grading system (n=34)

Open wound grading	Frequency	Percent
Grade 2	18	52.9
Grade 3	12	35.3
Grade 4	4	11.8
Total	34	100.0

Comment: About 52.9 percent of the study participants were classified as grade 2 and 35.3 percent were classified as grade 3 by the open wound grading system as shown in the table and diagram.

Figure 12: Distribution of study participants according to open wound grading system (n=34)

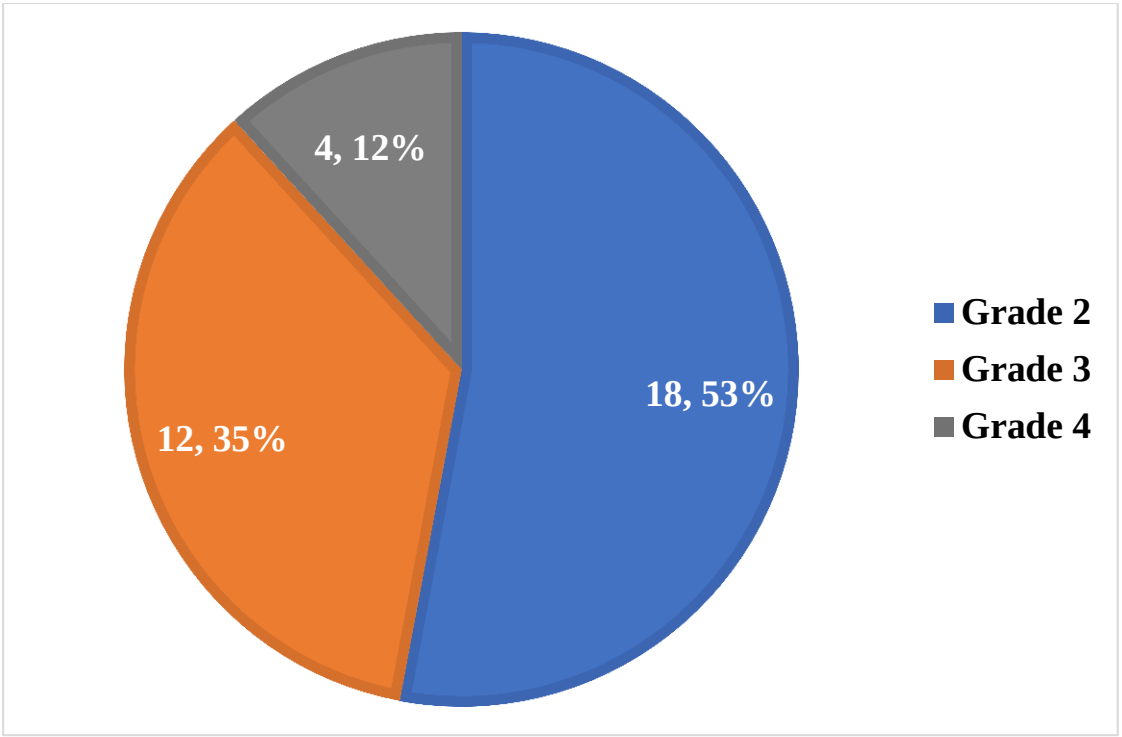


Table 7: Time between trauma and initial debridement

The time between trauma and initial debridement	
Mean	13.76
Median	8.00
Mode	8
Std. Deviation	11.492
Minimum	8
Maximum	48
Interquartile range	8.00 - 12.00

Comment: With respect to the time duration between the occurrence of trauma and initial debridement, the mean time difference was 13.76 minutes with a standard deviation of 11.492 minutes as shown in the table.

Table 8:Area of wound dimension before and after intervention

Area of wound dimension	Wound dimension before intervention	Wound dimension after intervention
Mean	66.059	27.97
Median	62.000	24.00
Mode	48.0	40
Std. Deviation	28.8926	15.822
Minimum	32.0	8
Maximum	160.0	60
Interquartile range	46.000 - 80.000	16.00 - 40.00

Comment: With regards to the difference in surface area of wound dimension before and after intervention the mean surface area was 66.059 cm square before intervention and 27.97 cm square after intervention as shown in the below table.

Figure 13: Area of wound dimension before and after intervention

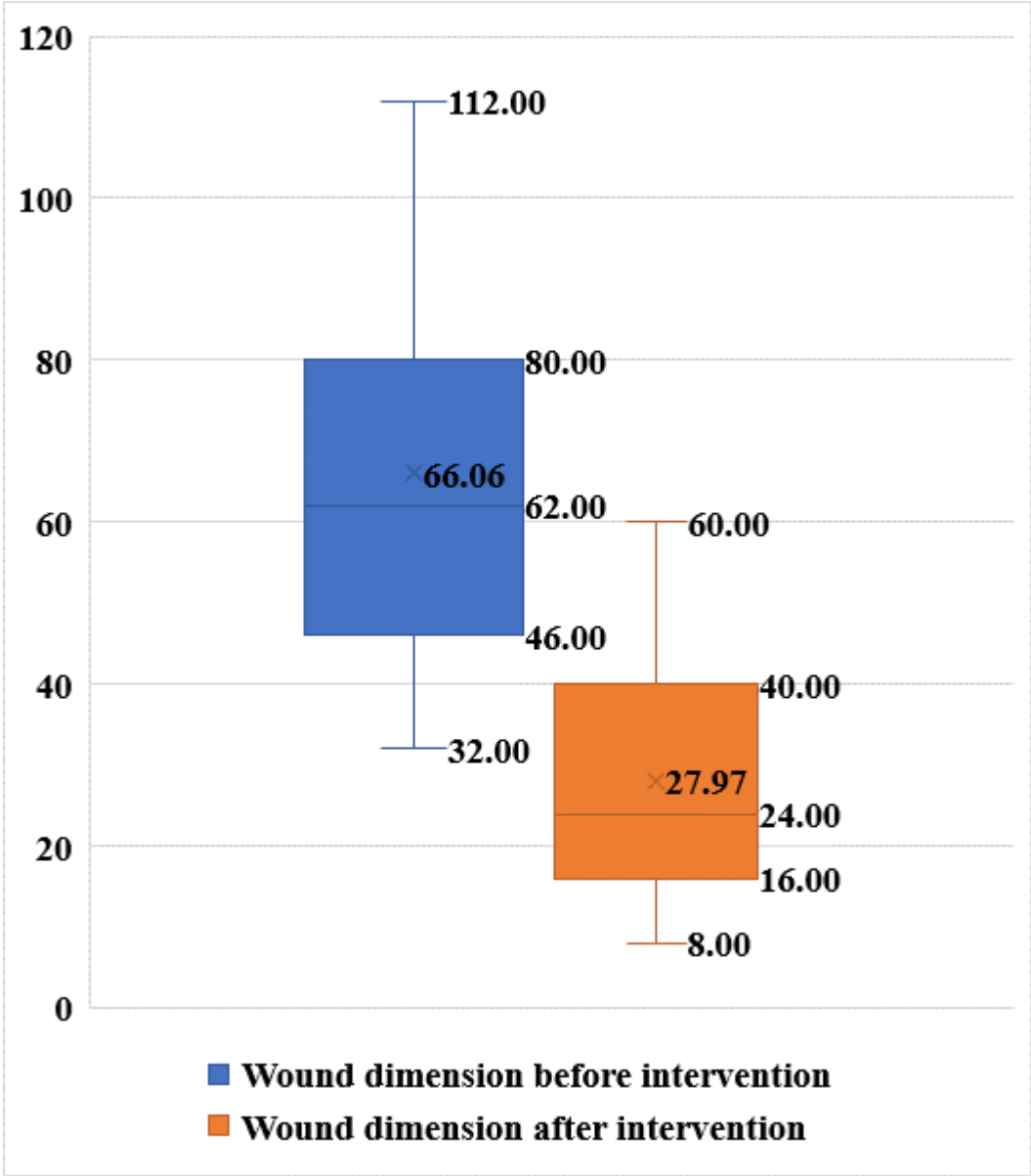


Table 9: Distribution of study participants according to the initial intervention after surgery (n=34)

Initial intervention	Frequency	Percent
External fixation	13	38.2
External-fixation with percutaneous pinning	1	2.9
Internal fixation	9	26.5
Percutaneous pinning	11	32.4
Total	34	100.0

Comment: About 38.2 percent of the study participants were intervened with external fixation and 32.4 percent of the individuals were intervened by percutaneous pinning after surgery as shown in the table and diagram.

Figure 14: Distribution of study participants according to the initial intervention after surgery (n=34)

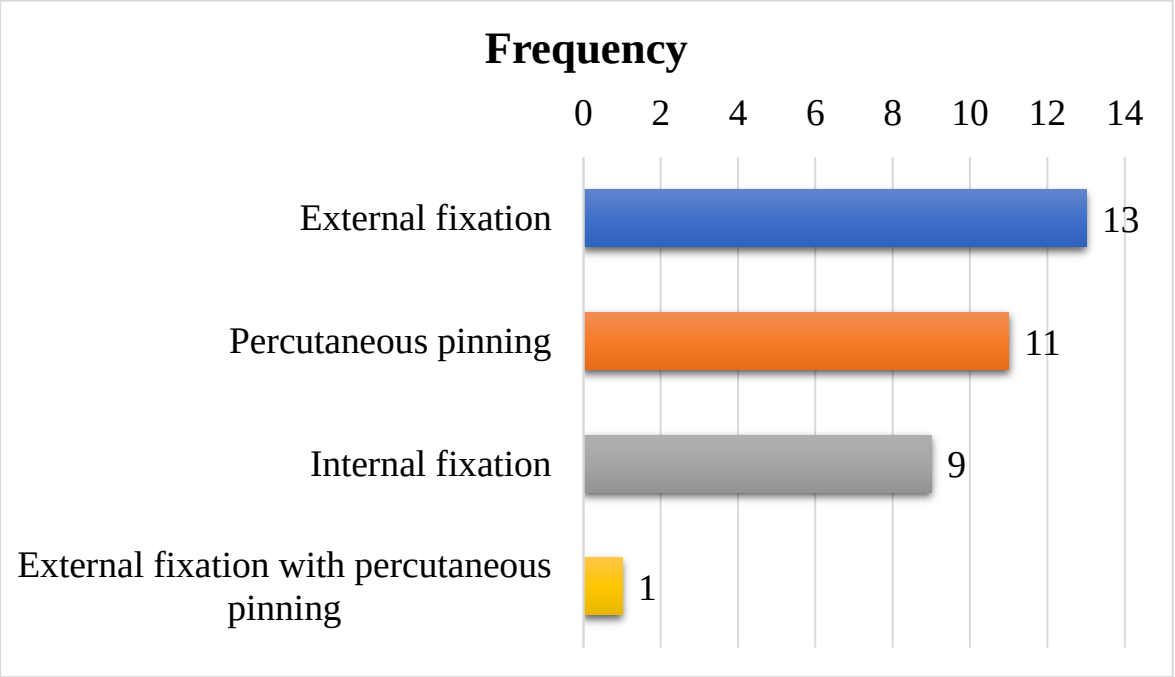


Table 10: Distribution of study participants according to the management by fixation (n=34)

Management by fixation	Frequency	Percent
External fixation	25	73.5
Internal fixation	9	26.5
Total	34	100.0

Comment: Almost 73.5 percent were managed by external fixation and the remaining were done by internal fixation as shown in the table and diagram.

Figure 15: Distribution of study participants according to the management by fixation (n=34)

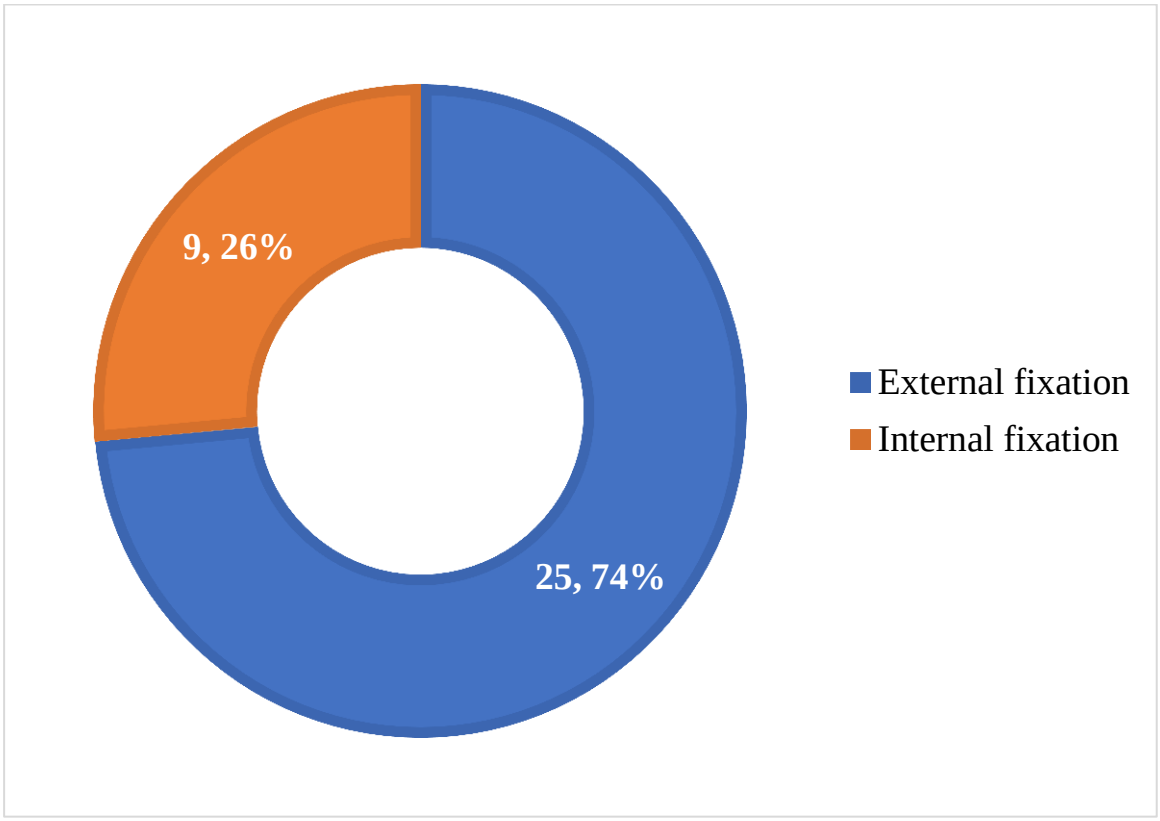


Table 11: Outcome following the intervention among the study participants (n=34)

Outcome following the intervention	Duration of hospital stay	Duration of VAC	Duration of follow-up
Mean	19.38	11.00	3.12
Median	20.00	12.00	3.00
Mode	12	6	3
Std. Deviation	5.914	4.573	.880
Minimum	10	6	2
Maximum	30	18	5
Interquartile range	14.00 - 24.00	6.00 - 12.50	2.00 - 4.00

Comment: With regards to the mean duration of hospital stay, mean duration of VAC and the follow-up the mean duration was 19.38, 11and 3.12 days respectively.

Figure 16: Outcome following the intervention among the study participants (n=34)

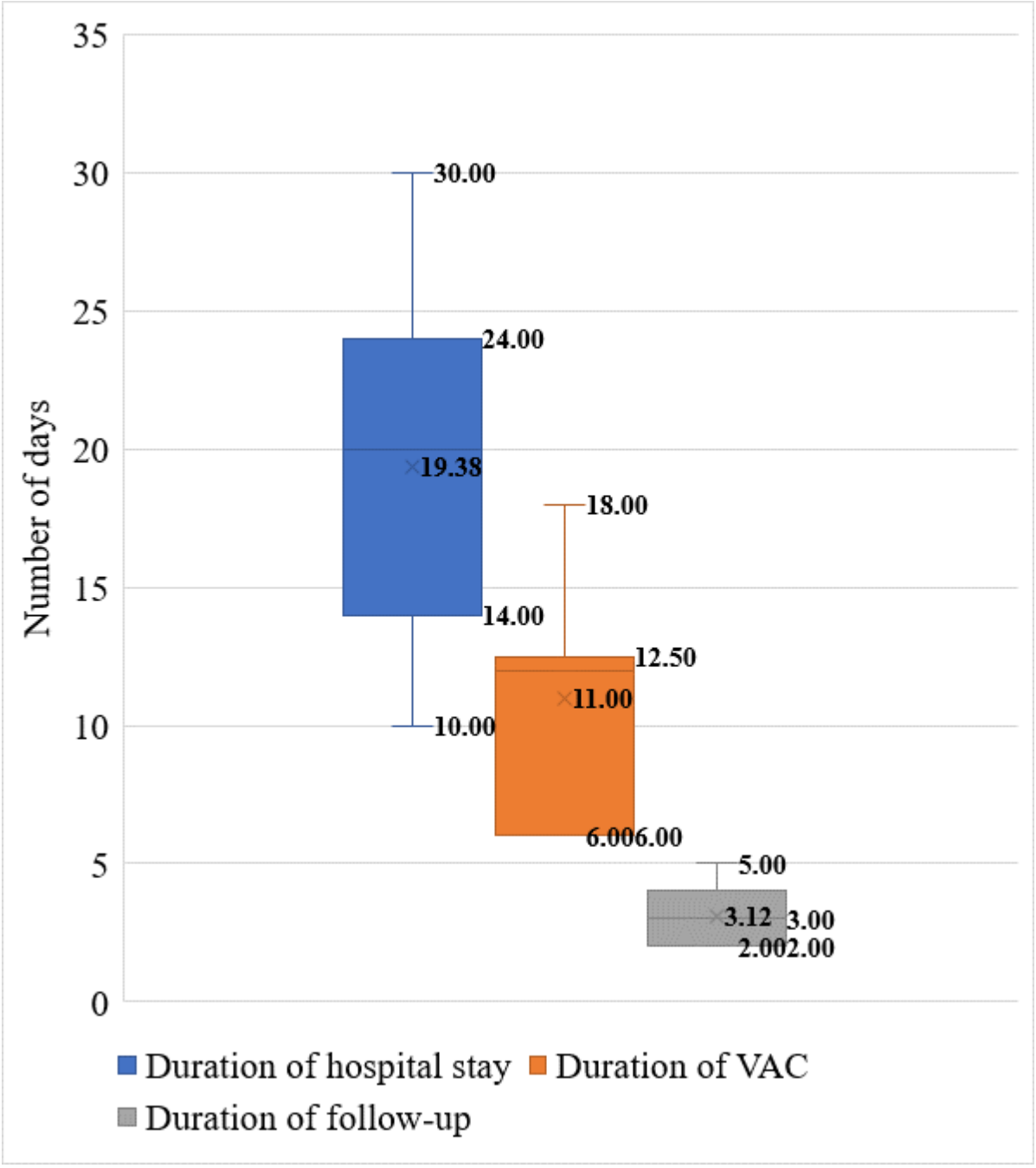


Table 12: Distribution of wound healing time among the study participants (n=34)

Wound healing time	
Mean	18.47
Median	18.00
Mode	12
Std. Deviation	5.534
Minimum	10
Maximum	28
Interquartile range	13.50 - 24.00

Comment: The mean duration of wound healing time was 18.47 days with a standard deviation of 5.534 days as shown in the table and diagram.

Figure 17: Distribution of wound healing time among the study participants (n=34)

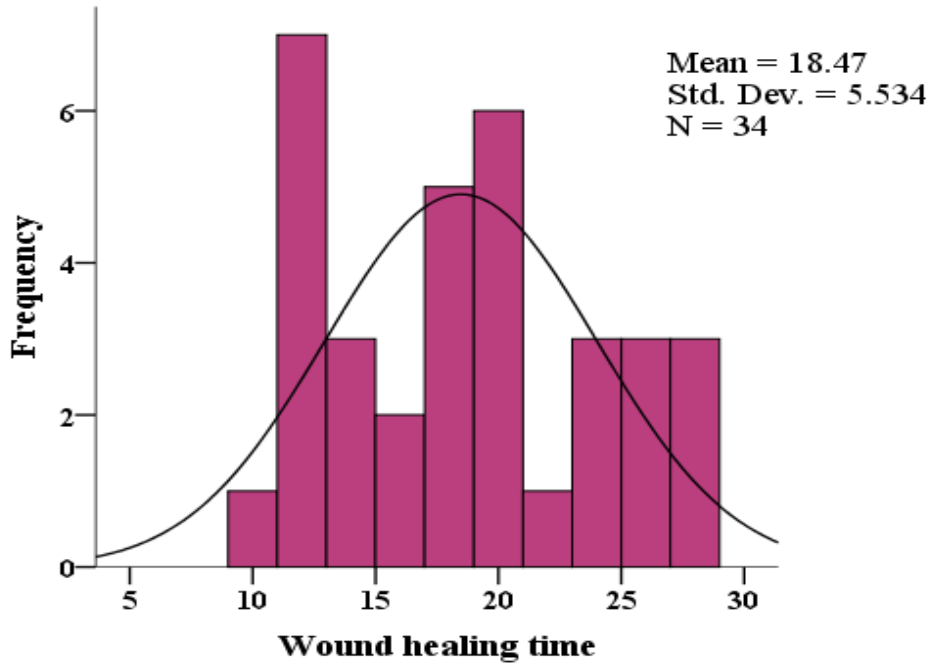


Table 13 :Distribution of study participants according to their comorbidities (n=34)

Comorbidities	Frequency	Percent
No co-morbidity	25	73.5
Anaemia	2	5.9
Diabetes	4	11.8
Hypertension	2	5.9
Thyroid disorder	1	2.9
Total	34	100.0

Comment: The most common co-morbidity was Diabetes Mellitus (11.8%) and nearly 73.5% of the participants didn't presented with any co-morbidities.

Figure 18: Distribution of study participants according to their comorbidities (n=34)

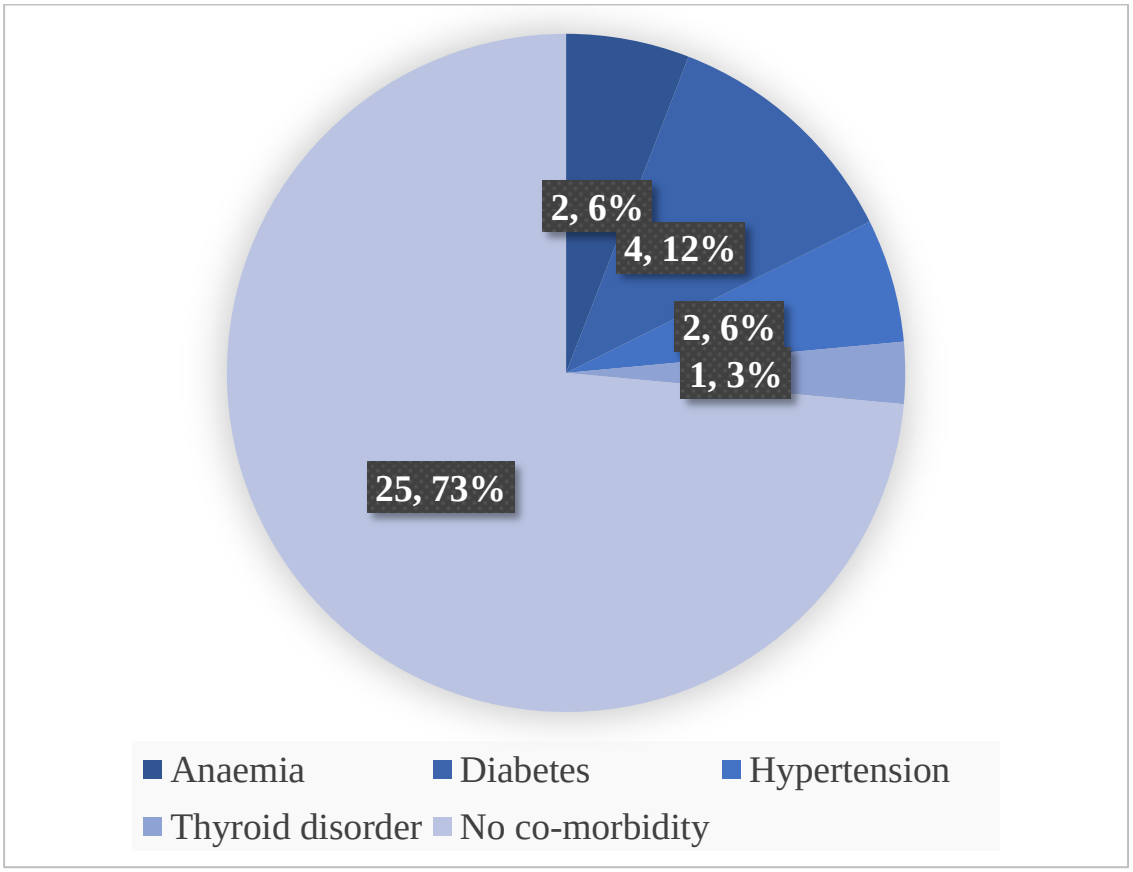


Table 14: Distribution of study participants according to the wound complication (n=34)

Wound complications	Frequency	Percent
Nil	13	38.2
Skin maceration	21	61.8
Total	34	100.0

Comment: Skin Maceration was seen among 21 subjects. This is the only complication of VAC dressing the current study.

Figure 19: Distribution of study participants according to the wound complication (n=34)

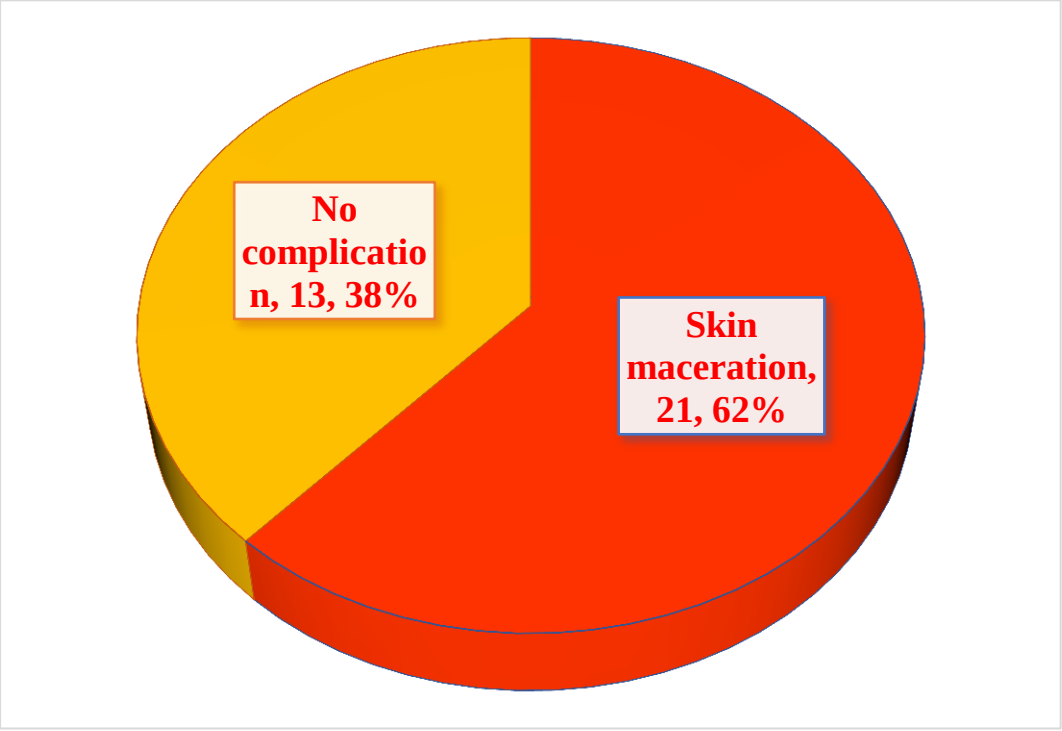


Table 15: Deep infection among the study participants (n=34)

Deep infection	Frequency	Percent
Yes	6	17.6
No	28	82.4
Total	34	100

Comment: In the study, the prevalence of deep-infection among the study participants was 17.6%.

Figure 20: Deep infection among the study participants (n=34)

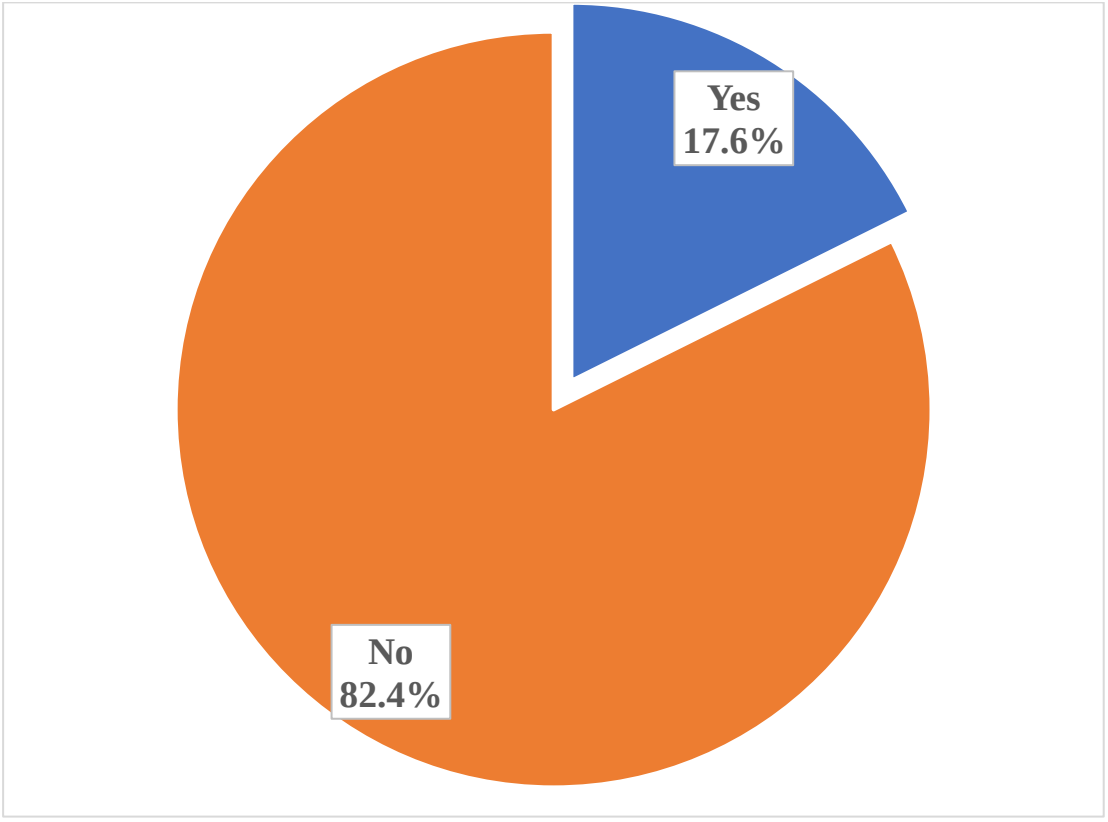


Table 16: Need for skin graft among the study participants (n=34)

Skin graft	Frequency	Percent
Yes	22	64.7
No	12	35.3
Total	34	100

Comment: Among the study participants nearly 64.7% need skin graft for wound healing in the study.

Figure 21: Need for skin graft among the study participants (n=34)

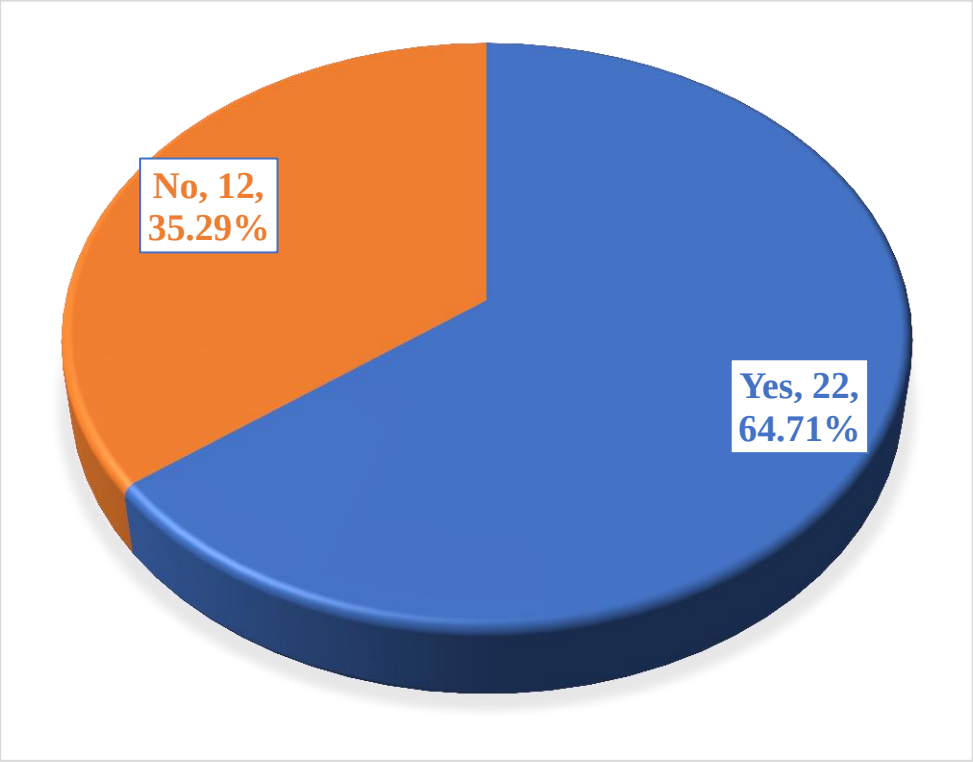


Table 17: Pre-VAC infection among the study participants (n=34)

Pre-VAC infections	Frequency	Percent
Yes	31	91.2
No	3	8.8
Total	34	100

Comment: The prevalence of Pre-VAC infection in the study was 91.2%.

Figure 22: Pre-VAC infection among the study participants (n=34)

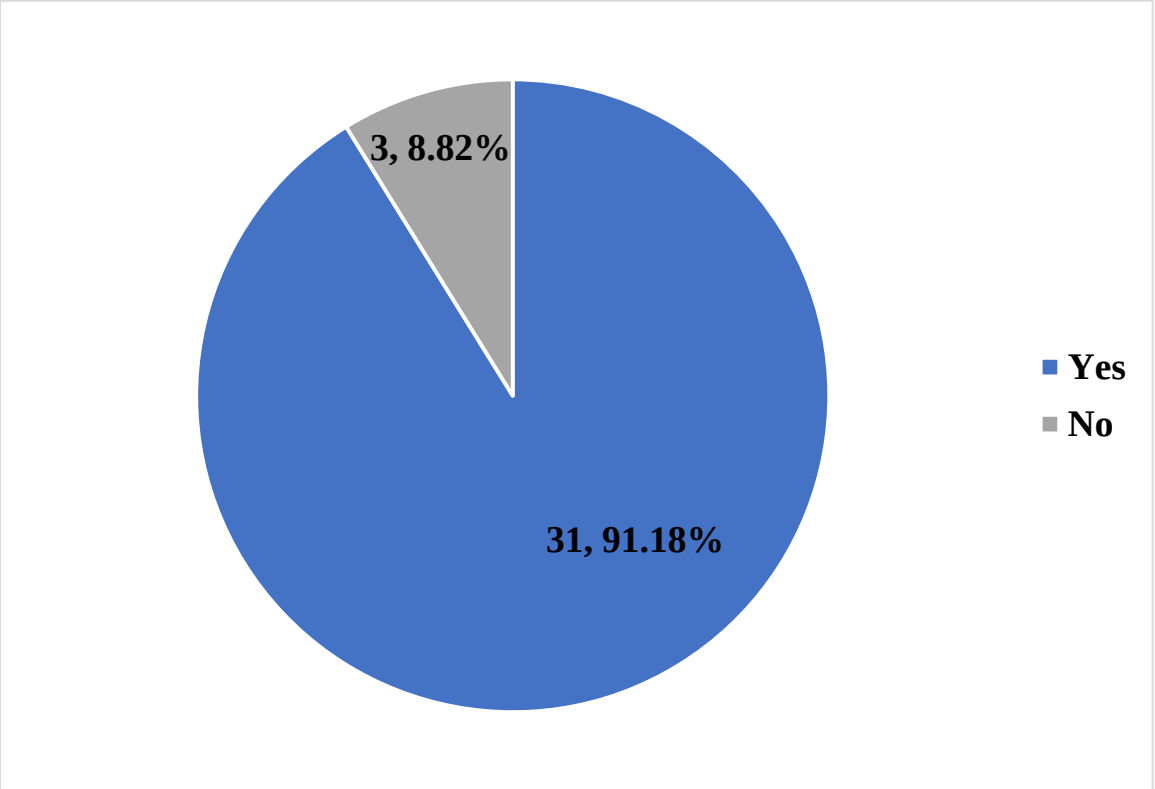


Table 18: Disease organism responsible for pre-VAC infections among the study participants (n=34)

Pre-VAC infections	Frequency	Percent
No Growth	3	8.8
Acinetobacter	3	8.8
E. coli	2	5.9
E. coli, Acinetobacter	1	2.9
Klebsiella	2	5.9
Klebsiella, E. coli	1	2.9
Klebsiella, Pseudomonas	2	5.9
Proteus	4	11.8
Pseudomonas	2	5.9
Staph Aureus	11	32.4
Staph Aureus, Pseudomonas	2	5.9
Staph Aureus. Acinetobacter	1	2.9
Total	34	100.0

Comment: When comes to pre-VAC infection status about 8.8 percent have no growth and 11.8 percent have proteus infection. The organisms responsible for pre-VAC infections are shown in the table.

Table 19: Post-VAC infection among the study participants (n=34)

Post-VAC infections	Frequency	Percent
Yes	6	17.6
No	28	82.4
Total	34	100

Comment: The prevalence of Post-VAC infection in the study was 17.6%.

Figure 23: Post-VAC infection among the study participants (n=34)

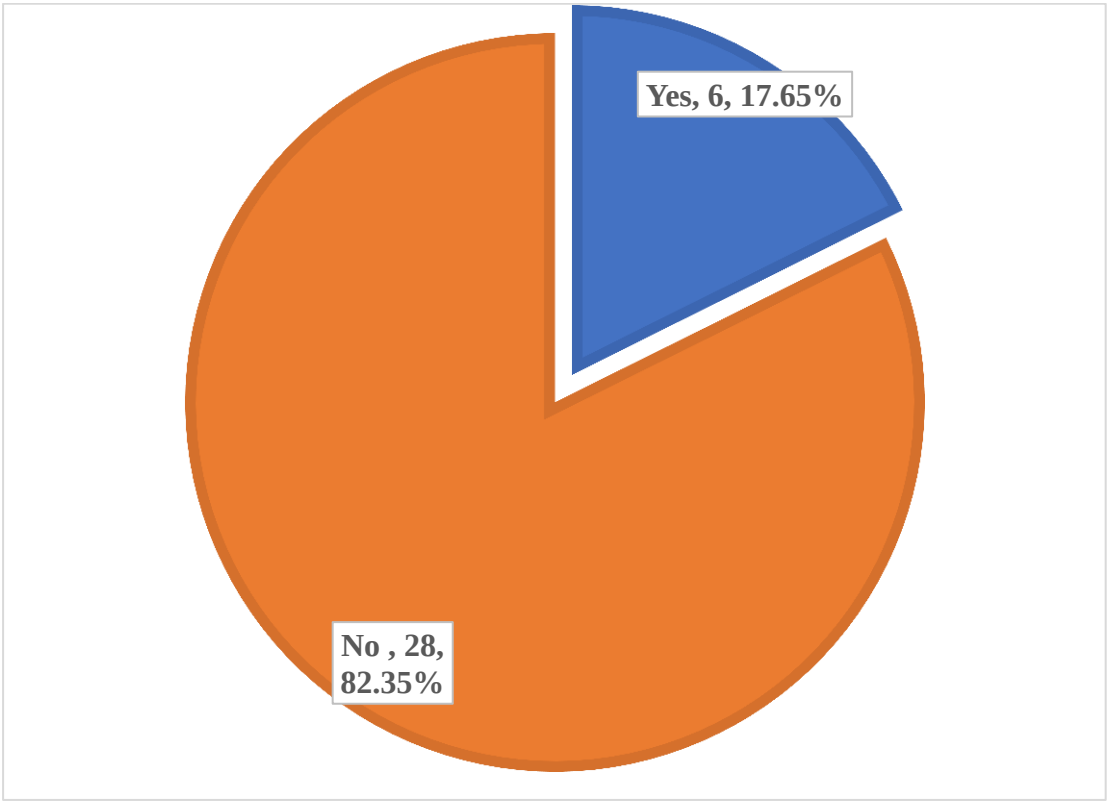


Table 20: Disease organism responsible for post-VAC infections among the study participants (n=34)

Post-VAC infection	Frequency	Percent
Pseudomonas	2	5.9
Staph Aureus	2	5.9
Acinetobacter	1	2.9
Proteus	1	2.9
Nil	28	82.4
Total	34	100

Comment: With regards to post-VAC infection status about 82.4 percent have no growth and 5.9 percent have Pseudomonas and Staph Aureus infection each. The organism responsible for post-VAC infections are shown in the table.

Figure 24: Disease organism responsible for post-VAC infections among the study participants (n=34)

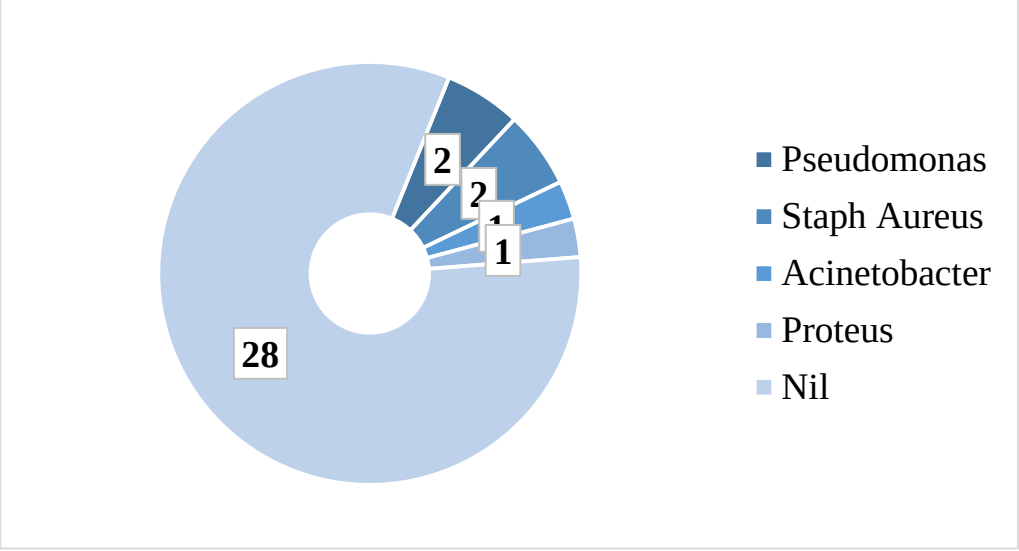


Table 21: Association of wound dimension before and after intervention by Paired T test. (n=34)

Wound dimension	Mean	Std. Deviation	Mean difference	P - Value
Wound dimension before intervention	66.059	28.8926	33	< 0.001
Wound dimension after intervention	27.97	15.822		

Comment: The mean dimension of wound before VAC therapy was 66.059 and the mean dimension of wound after VAC therapy was 27.97. The difference in mean before and after the intervention is found to be statistically significant according to the Paired T Test ($p < 0.001$). Hence, VAC dressing helps to decrease the wound size and it was proved statistically.

Table 22: Association between before and after infection of study participants after the intervention of VAC by McNemar Test (n=34)

Infections before and after VAC dressing		Post VAC infection (n (%))		P - Value
		No	Yes	
Pre-VAC infection	No	3 (100)	0(0)	< 0.001*
	Yes	25 (80.6)	6 (19.4)	

Comment: The prevalence of infection before VAC dressing was 80.6% and the prevalence of infection after VAC dressing was 19.4%. The difference in proportion before and after the intervention is found to be statistically significant ($p < 0.001$) according to the McNemar Test. Hence VAC dressing helps to decrease the rate of infection and it was proved statistically.

DISCUSSION

DISCUSSION

The current study's participants were 37.06 ± 10.340 years old on average. This finding can be compared to the mean age of 38 years in a study conducted by Joethy et al in Singapore in 2013[92] and the mean patient age of 39 ± 18 years in a prospective, randomised and interventional study conducted by Sinha k et al in India in 2013[22] (ranging from 18 to 76 years). In the current study, males made up around 76.5 percent of the participants, while females made up the rest.

Road traffic accidents caused 79.4 percent of the trauma in this study, with the rest coming from employment injuries. This finding is similar to a prospective, randomised and interventional study conducted in India by Sinha k et al in 2013[22], in which the most common cause was realized to be a road traffic accident with 22 (73.33 percent) patients, trailed by machinery injury with 5 (16.66 percent) patients and an accidental-fall from height with 3 (10 percent) patients. Traumatic injuries are frequently linked with severe skin loss, which exposes tendons, bone, or metal, as well as wound management challenges. In many aspects, these injuries resemble chronic ulcerative lesions of the foot associated with ischemia illnesses like diabetes mellitus. The rapid development of granulation tissue over the wound and blood vessels in and around the wound is necessary for wound healing. Furthermore, the collagenase and metalloproteinase ingredients in interstitial fluid from open wounds limit local blood flow and disrupt wound healing. In this regard, NPWT is extremely successful at removing interstitial fluid.

Gustilo Anderson type 3 B was assigned to 64.71 percent of the people, whereas type 3A was assigned to 35.29 percent. External fixation handled over 73.5 percent of the cases, while internal fixation handled the rest. According to the open wound grading method, 52.9 percent of the study participants were classified as grade 2 and 35.3 percent as grade 3.

In this study, roughly 64.7 percent of the participants require skin grafts for wound healing. The requirement for free flap surgery was shown to be reduced by 30% in a comparison study of traditional dressings and NPWT for lawnmower injuries of the lower leg[94], A significant reduction in the need for secondary soft tissue surgery is thought to be a significant benefit of NPWT[95]. Dedmond further stated that grade 3 wounds with an open tibial fracture healed without the requirement for a secondary soft tissue procedure such as a free flap.[96]

Deep infection was attained to be present in 17.6% of the subjects in the existing investigation. According to a retrospective cohort study conducted by Blum et al in 2012[97] when used for the dressing of traumatic wounds in open tibial fractures, NPWT reduces the chance of deep infection (8.4%). When multivariate analysis was used to compensate for Gustilo type, it was discovered that using NPWT reduced the probability of deep infection by about 80%. When utilised for the dressing of Gustilo type IIIA/B fractures, a study conducted by Gill et al in 2016[98] shown that VAC treatment reduced the chance of deep infection by 7%. As a result, VAC dressing is thought to minimise the chance of deep infection in Gustilo type fractures.

Diabetes Mellitus was the most commonly associated co-morbidity in our study (11.8 percent), while roughly 73.5 percent of the patients had no co-morbidities. This finding contrasts with a 2013 study by Joethy et al in Singapore, which found that the prevalence of pre-existing co-morbidities was (11–12%).[92]

The average duration of VAC and follow-up was 11 days and 3.12 days, respectively. This conclusion was similar to that of Lee et al. in Korea[87] who found that the average duration of VAC therapy was 18.4 days (range, 11– 29 days). Suman et al.,[3] in India reported in their study that the average period of VAC-application was ten days (range, 3–16). In the study,

the mean duration of VAC-dressing in patients with and without infection was 7.78 ± 0.42 days and 8.79 ± 1.19 days, respectively.

In this study, the mean wound healing time was 18.47 days, with a standard deviation of 5.534 days. The duration of wound healing in VAC was found to be shorter with Type III tibial fractures, according to Hou et al.[99]

The mean time delay between the occurrence of trauma and the beginning of debridement was 13.76 minutes, with a standard deviation of 11.492 minutes. In contrast, Suman et al., in India by 2021[3] reported in their study that the mean period between the trauma and the first-debridement was 8.20 hours (range, 2–23) in their study.

The six-hour window between operation debridement and operative debridement of open lower-limb fractures was once thought to be critical in reducing infection rates and this time period was dubbed the "golden period" for wound treatment. Regardless of the origins of the six-hour rule, debridement of open fractures within six hours of damage is a widely acknowledged standard of treatment, even though some writers have claimed that debridement during the golden period had no benefit.[92]

Articles supporting VAC therapy reducing the dimension of wound in open fractures after surgical intervention

Before VAC therapy, the average wound dimension was 66.059 and after VAC therapy, the average wound dimension was 27.97. According to the Paired T Test, the difference in mean before and after the interventional management is statistically significant (p 0.001). As a result, VAC dressing has been statistically proven to help reduce wound size. Vacuum aided closure (VAC) is a good way to speed up wound closure.

Sinha k et al., employed VAC for Musculoskeletal injuries in a prospective, randomised and interventional trial published in India in 2013[22] They discovered that following VAC, soft tissue deficiencies shrank by more than 5 mm to 25 mm (a decrease of 26.66 percent), but wound size shrank by less than 5 mm with normal wound care. They came to the conclusion that VAC care aided the quick production of nutritious granulation tissue, reducing healing time besides reducing secondary soft tissue defect covering procedures. Similar investigations by Joseph et al[21], Morykwas and Argenta[14]and Morykwas et al.[15] found that VAC was more successful than traditional wound dressings in lowering wound widths over time.

Articles supporting VAC therapy reducing the rate of infection in open fractures after surgical intervention

Infection was prevalent before VAC dressing at 80.6 percent, while it was prevalent after VAC dressing at 19.4 percent. According to the McNemar Test, the difference in proportion before and after the intervention is statistically significant (p 0.001). As a result, VAC dressing helps to reduce the rate of infection, which has been scientifically proven.

Sinha k et al employed VAC for open Musculoskeletal injuries in a prospective, randomised and interventional trial in India by 2013[22] and found that after 4 days, 20% of cases showed no bacterial development, while on the 8th day, 60% of cases showed no bacterial growth. Similar investigations by Morykwas and Argenta[14], Banwell et al[100]and Morykwas et al[15] have shown that VAC would eliminate bacteria from infected wounds.

CONCLUSION

CONCLUSION

The prevalence of infection before VAC dressing was 80.6% and the prevalence of infection after VAC dressing was 19.4%. Vacuum assisted closure confers a decent help for speedy closure of the wound. The mean dimension of wound before VAC therapy was 66.05cm^2 and the mean dimension of wound after VAC therapy was 27.97cm^2. Hence VAC dressing benefits to decrease the rate of infection.

There is a cumulative body of data encouraging VAC as an adjunctive mode at all phases of treatment for Grade IIIA/B open fractures. There is a relationship between decreased infection rates and quick wound healing with VAC treatment.

RECOMMENDATION

The patient recovers faster when the NPWT method is used as part of a multi-directional strategy. It is a viable and effective treatment for treating compound fractures with significant soft-tissue defects that can be used instead of microsurgical soft-tissue transfer. It also minimises the risk of infection and allows the limb to be saved. We further believe that, as compared to traditional wound dressing changes, the vacuum-assisted closure system extends dressing intervals, reduces cost and minimises patient suffering. As a result, a prospective randomised multi-center trial should be conducted to establish the efficacy of NPWT in the treatment of Gustilo-Anderson type IIIA/ IIIB open fracture soft tissue abnormalities.

For open wound treatment and closed surgical wounds, NPWT has vastly improved. Modifications to the device will be made in the future to make it easier for patients to use and to allow it to be used in a wider range of anatomic locations. Finally, more high-quality research is needed to better identify the outcomes related with NPWT, particularly in terms of specific therapeutic applications and cost. The utility of NPWT in the outpatient context should be the focus of future research.

LIMITATION

- This is an observational study; an experimental study design includes Randomized Controlled Trial would have provided better association
- This study has no control or comparison group with other type of dressing
- Fairly smaller sample size.
- Wound grading was simpler in this study.
- Severity of injury, smoking may be a confounding factor that was not studied. However, as the traumatic force contributes to the sternness of wounds.
- Application of VAC required long hospitalization of the cases.

SUMMARY

SUMMARY

- Compound fractures are pretty more common due to growing trend toward high-speed motor vehicle accidents. They're frequently linked to osteomyelitis and an exalted risk of acquiring deep infections.
- The use of external fixation, debridement and a vacuum-assisted closure is illustrated as a management method.
- Negative pressure dressings are a popular choice for treating open fracture wounds in the interim. Topical negative pressure regulates local blood flow, helps granulation tissue formation, bacterial clearance and flap survival.
- The aim was to determine whether VAC reduces wound healing period besides diminution of the frequency of infections in closure of wound after fixation in treatment of Gustilo Anderson Type IIIA/ IIIB fractures of the extremities.
- The observational study was conducted among 34 patients with Gustilo Anderson Type IIIA/ IIIB fractures of the limbs in R. L. Jalappa hospital attached to Sri Devaraj Urs Academy of Higher Education and Research Tamaka, Kolar during the period between December 2019 and July 2021.
- Vacuum Assisted Closure was casted off for closure of wound after fixation of fractures. Patients was followed up for one month.
- The existing study's participants were 37.06 ±10.340 years of age.
- Males made up around 76.5 percent of the participants, while females made up the rest.
- Road traffic accidents caused 79.4 percent of the trauma in this study, with the rest coming from employment injuries.

- According to the open wound grading method, 52.9 percent of the study participants were classified as grade 2 and 35.3 percent as grade 3.
- Before VAC therapy, the average wound dimension was 66.059 and after VAC therapy, the average wound dimension was 27.97. According to the Paired T Test, the change in mean before and after the intervention is statistically significant (p 0.001). As a result, VAC dressing has been statistically proven to help reduce wound size. VAC is a good way to speed up wound closure.
- Infection was prevalent before VAC dressing at 80.6 percent, while it was prevalent after VAC dressing at 19.4 percent. According to the McNemar Test, the difference in proportion before and after the intervention is statistically significant (p 0.001). As a result, VAC dressing helps to reduce the rate of infection, which has been scientifically proven.
- VAC, a gift that is practicable treatment method to treat compound-fractures with massive soft-tissue defects.

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ANNEXURE

ANNEXURE - I
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

PATIENT INFORMATION SHEET

STUDY TITLE: "EVALUATION OF CLINICAL OUTCOME OF NEGATIVE PRESSURE WOUND THERAPY IN GUSTILO ANDERSON TYPE IIIA/IIIB OPEN FRACTURES OF EXTREMITIES"

Study location: R L Jalappa Hospital and Research Centre attached to Sri DevarajUrs Medical College, Tamaka, Kolar.

Details- Patients diagnosed with open type IIIA/IIIB fractures admitted in orthopaedics ward from opd at R.L.J. HOSPITAL AND RESEARCH CENTRE, attached to SRI DEVARAJ URS MEDICAL COLLEGE,TAMAKA, KOLAR

Patients in this study will have to undergo routine blood investigations CBC, ESR,Blood Grouping and Rh typing, BT,CT and Culture Sensitivity.
Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board. For further information/ clarification please contact

Dr.ARUN KUMAAR S P (Post Graduate),

Department Of ORTHOPAEDICS,

SDUMC , Kolar

CONTACT NO : 8056673210

ANNEXURE - II

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Case no:

IP no:

TITLE: '' EVALUATION OF CLINICAL OUTCOME OF NEGATIVE PRESSURE WOUND THERAPY IN GUSTILO ANDERSON TYPE IIIA/IIIB OPEN FRACTURES OF EXTREMITIES''

I, _____ aged _____ ,after being explained in my own vernacular language about the purpose of the study and the risks and complications of the procedure, hereby give my valid written informed consent without any force or prejudice for vac therapy in type IIIA/IIIB fractures which is an Therapeutic Procedure to be performed on me. The nature and risks involved in the Therapeutic procedure have been explained to me to my satisfaction.

I have been explained in detail about the Dissertation study on "**EVALUATION OF CLINICAL OUTCOME OF NEGATIVE PRESSURE WOUND THERAPY IN GUSTILO ANDERSON TYPE IIIA/IIIB OPEN FRACTURES OF EXTREMITIES''**

being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research. I hereby give consent to provide my history, undergo physical examination, undergo the Therapeutic procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose the procedure, may be video graphed or photographed. All the data may be published or used for any academic

purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study.

A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

(Signature/Thumb impression & Name of patient)

(Signature/Thumb impression & Name of Pt. Attendant) Witness:----------

(Signature & Name of Research person /doctor)----------

ANNEXURE - III

ಶ್ರೀ ಿೀ ದೇವರಾಜ ಅರಸು ಉನ್ನ ತ ಶ್ಷ ಣ ಮತ್ತು ಸಂಶ್ೀಧನೆಯ ಅಕಾಡೆಮಿ, ಬಹು, ಕ

ೋಝಿಕೊಡಾರ – 563101

ತಿಳಿವಳಿಕೆಯ ಸಮ್ಮತಿ ನಮೂನೆ

ಗುಸ್ಟಿಲೊ ಆಂಡರ್ಸನ್ ವಿಧನ ೪೪೪೦ / ೪೪೪೪ ಮಾದರಿಯ ಅಸ್ಥಿಭಂಗಗಳಲ್ಲಿ ಶೂನ್ಯ ಸಹಯೋಗದೊಂದಿಗೆ ಉಪಸಂಹರಿಸುವ ಶಸ್ತ್ರ ಚಿಕಿತ್ಸೆಯವೈದ್ಯಕೀಯ ಫಲಿತಾಂಶದ ಬಗ್ಗೆ ಒಂದು ಅಧ್ಯಯನ.

ನಾನು, _____ ವಯಸ್ಸಿನ _____, ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲ್ಪಟ್ಟ ನಂತರ ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ತೊಂದರೆಗಳು ಮತ್ತು ತೊಡಕುಗಳ ಬಗ್ಗೆ ವಿವರಿಸಿದ ನಂತರ, ಮುಚ್ಚಿದ ಕಡಿತ ಮತ್ತು ಆಂತರಿಕ ಸ್ಥಿರೀಕರಣ / ಓಪನ್ಗೆ ಯಾವುದೇ ಬಲದ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯವಾದ ಲಿಖಿತ ವಿರೋಧಿ ಸಮ್ಮತಿಯನ್ನು ನೀಡಿ ನನ್ನ ಮೇಲೆ ನಡೆಸಬೇಕಾದ ರೋಗನಿದಾನ ಮತ್ತು / ಅಥವಾ ಚಿಕಿತ್ಸಕ ಪ್ರಕ್ರಿಯೆ / ವರ್ಗಾವಣೆ / ಕಾರ್ಯಾಚರಣೆ ಅಥವಾ ಯಾವುದೇ ಅರಿವಳಿಕೆ ಅಡಿಯಲ್ಲಿ _____ ನಂತಹ ಪ್ಲೇಟ್ ಮತ್ತು ತಿರುಪು / ಸಂಪ್ರದಾಯವಾದಿ ನಿರ್ವಹಣೆಯೊಂದಿಗೆ ಪ್ಲೇಟ್ ಮತ್ತು ಸ್ಕ್ರೂ / ಸಂಪ್ರದಾಯವಾದಿ ನಿರ್ವಹಣೆಗೆ ಒಳಪಡಿಸುವುದು ಯೋಗ್ಯವಾದವು. ಕಾರ್ಯವಿಧಾನದಲ್ಲಿ (ಶಸ್ತ್ರಚಿಕಿತ್ಸಾ ಮತ್ತು ಅನಾಸ್ಥೆಟಿಕಲ್) ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳು ನನ್ನ ತೃಪ್ತಿಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

"ಕಾರ್ಟಿಲೆಜ್ ದೋಷಗಳ ಕ್ಲಿನಿಕಲ್, ಎಂಆರ್ಐ ಮತ್ತು ಆರ್ತ್ರೋಸ್ಕೊಪಿಕ್ ಮೌಲ್ಯಮಾಪನಗಳ ಹೋಲಿಕೆ ಮತ್ತು ಮೊಣಕಾಲಿನ ಆಂತರಿಕ ವಿಘಟನೆ" ಕುರಿತು ಕ್ಲಿನಿಕಲ್ ರಿಸರ್ಚ್ ಕುರಿತು ನಾನು ವಿವರಿಸಿದ್ದೇನೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆ ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರ ಮಾಡಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವರಾಗಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ. ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಇಂಜೆಕ್ಷನ್ ಪ್ರಕ್ರಿಯೆಗೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಬೇಕು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ವೈದ್ಯರಿಗೆ / ಇನ್ವೆಸ್ಟಿಗೇಟ್ಗೆ ನೀಡುವಂತೆ ನಾನು ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ವಿಧಾನ, ಇತ್ಯಾದಿ ವೀಡಿಯೊವನ್ನು ಗ್ರಾಂಪ್ಡ್ ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಕೆಟ್ಟ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಇನ್ವೆಸ್ಟಿಗೇಟ್ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಂದುವುದಿಲ್ಲ.

ಈ ಮಾಹಿತಿಯುಕ್ತ ಸಮ್ಮತಿಯ ಫಾರ್ಮ್ ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಪ್ರತಿಸ್ಪರ್ಧಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

_____ _____ _____

(ರೋಗಿಯಪರಿಚಾರಕನಸಹಿ & ಹೆಸರು) (ರೋಗಿಯ / ಗಾರ್ಡಿಯನ್ನಸಹಿ / ಹೆಬ್ಬೆಟ್ಟಿನಗುರುತು&ಹೆಸರು)

(ರೋಗಿಯಸಂಬಂಧ)

(ಸಂಶೋಧಕನ / ವೈದ್ಯರಸಹಿ&ಹೆಸರು)

ANNEXURE -IV

CASE PROFORMA

Case no:

IP no:

TITLE:

"EVALUATION OF CLINICAL OUTCOME OF NEGATIVE PRESSURE WOUND THERAPY IN GUSTILO ANDERSON TYPE IIIA/IIIB OPEN FRACTURES OF EXTREMITIES"

1. BASIC DATA

Name Age/Sex

Address

Mobile No.

Date of Procedure

Date of Admission/OP

Date of Discharge

HISTORY :

MODE OF TRAUMA:

GENERAL PHYSICAL EXAMINATION:

VITALS:

Pulse- B.P-

RR- Temp-

SYSTEMIC EXAMINATION:

CVS-

RS-

PA-

CNS-

TYPE OF OPEN FRACTURE:

NEURO VASCULAR DEFICIT(IF ANY):

PRE EXISTING SYSTEMIC ILLNESS :

MALNUTRITION	
ANAEMIA	
TUBERCULOSIS	
DIABETES	
HYPERTENSION	
THYROID DISORDER	
OTHERS	

LOCAL EXAMINATION: PRE NPWT POST NPWT

SITE OF WOUND		
DIMENSION OF WOUND (cm)		
BONE EXPOSED OR NOT		
TENDONS EXPOSED OR NOT		

2. DIAGNOSIS:

3. INVESTIGATIONS

COMPLETE BLOOD COUNT	
BLEEDING TIME & CLOTTING TIME	
BLOOD GROUPING & Rh TYPING	
CULTURE SENSITIVITY WITH WOUND SWABS	

4. TREATMENT(Initial Surgery):

TIME INTERVAL BETWEEN TRAUMA & INITIAL DEBRIDEMENT	
WOUND DEBRIDMENT	
INTERNAL FIXATION WITH NPWT	
EXTERNAL FIXATION WITH NPWT	

5. OPEN WOUND GRADING SYSTEM (SCORE):

SCORE (GRADE)	STATUS OF WOUND
0	CLOSED WOUND
1	SKIN OR SOFT TISSUE DEFECT
2	BONE, TENDON, IMPLANT EXPOSURE (ANY 1)
3	BONE, TENDON, IMPLANT EXPOSURE (ANY COMBINATION OF 2 OR MORE) ASSOCIATED
4	ASSOCIATED OR RESIDUAL INFECTION QUENCIES

6. POST OP:

IV ANTIBIOTICS	

7. INFECTIONS:

DEEP INFECTION	
PRE VAC INFECTION	
POST VAC INFECTION	

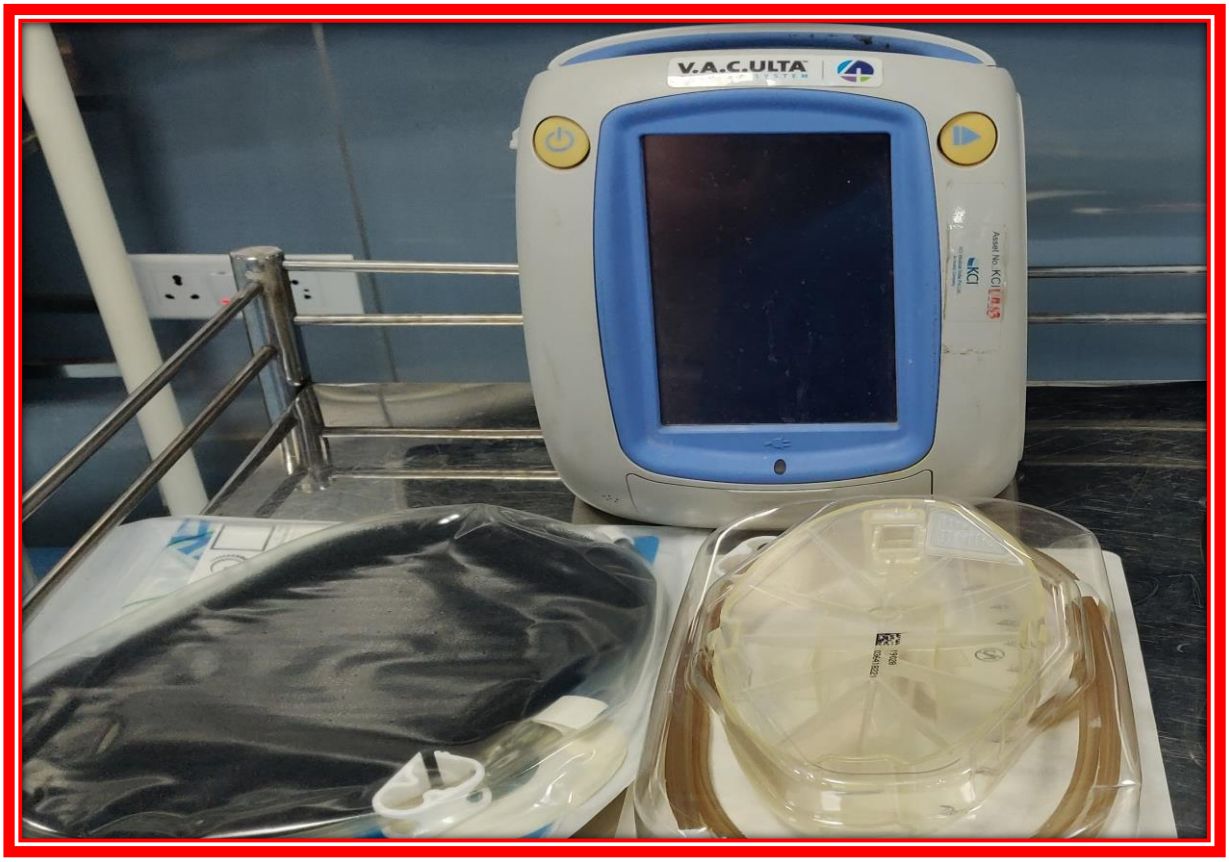
8. FOLLOW UP

DURATION OF HOSPITAL STAY (DAY'S)	
DURATION OF VAC DRESSING (DAY'S)	
WOUND HEALING TIME (DAY'S)	
NUMBER OF DRESSING DONE	
DEFINITIVE SKIN COVER PROCEDURES(SSG, FLAP COVER)	
WOUND COMPLICATIONS	
DURATION OF FOLLOW UP (WEEKS)	

ANNEXURE- V

DATA COLLECTION PHOTOS

VACUUM ASSISTED CLOSURE DEVICE

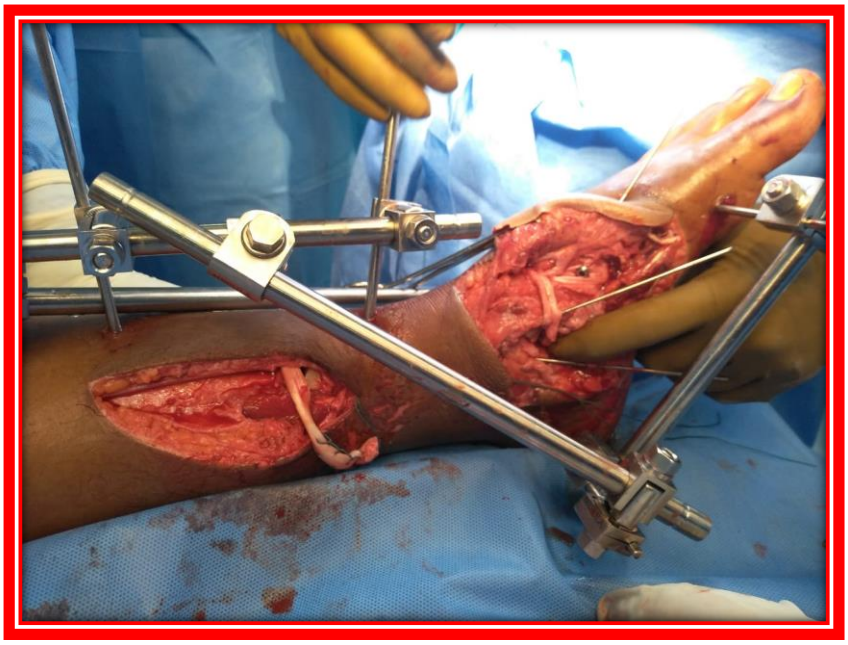


PATIENT-1

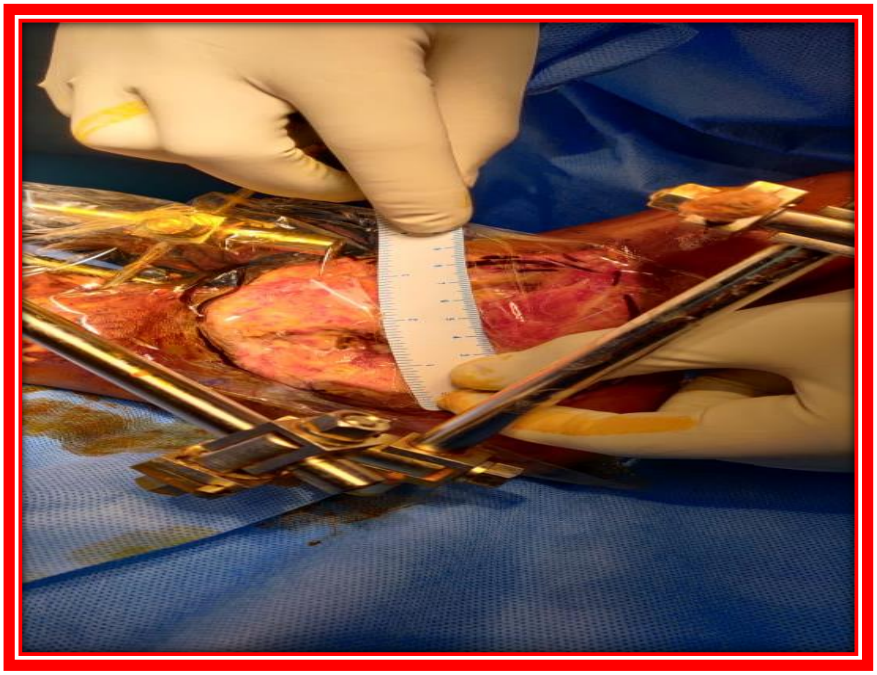
41year female case of open type 3b left distal third both bone fracture of left leg with open fracture of left medial cuneiform

1) Image was taken in EMD

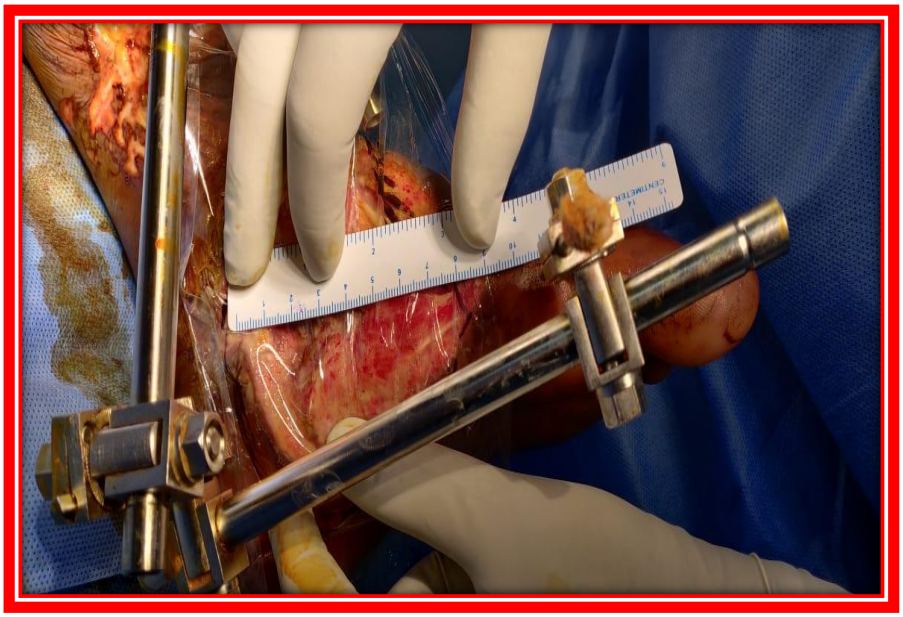
2) Intra-op images after exfix + percutaneous pin followed by VAC application



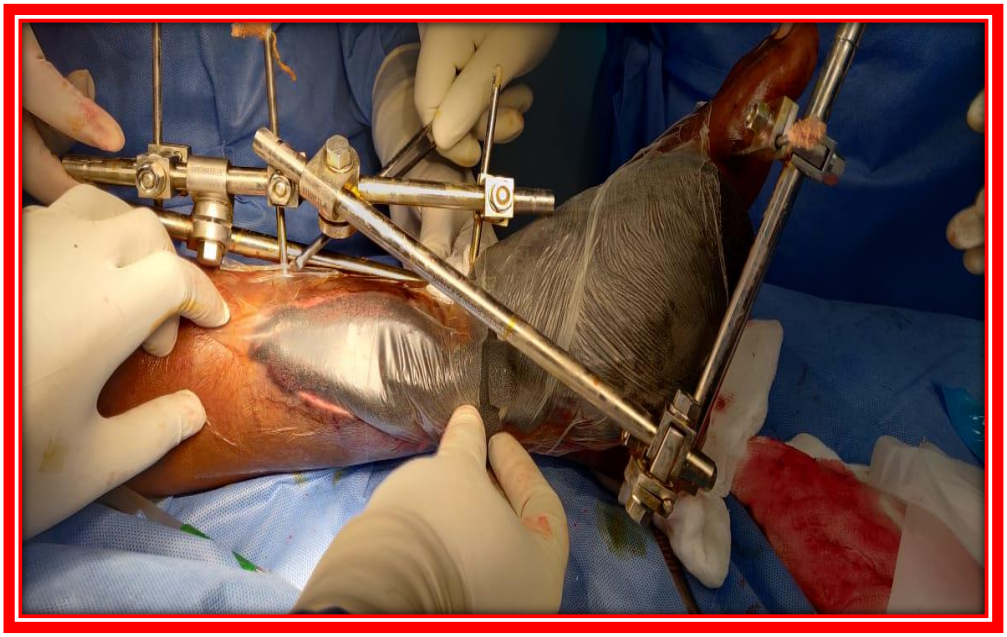
Wound debridement was done



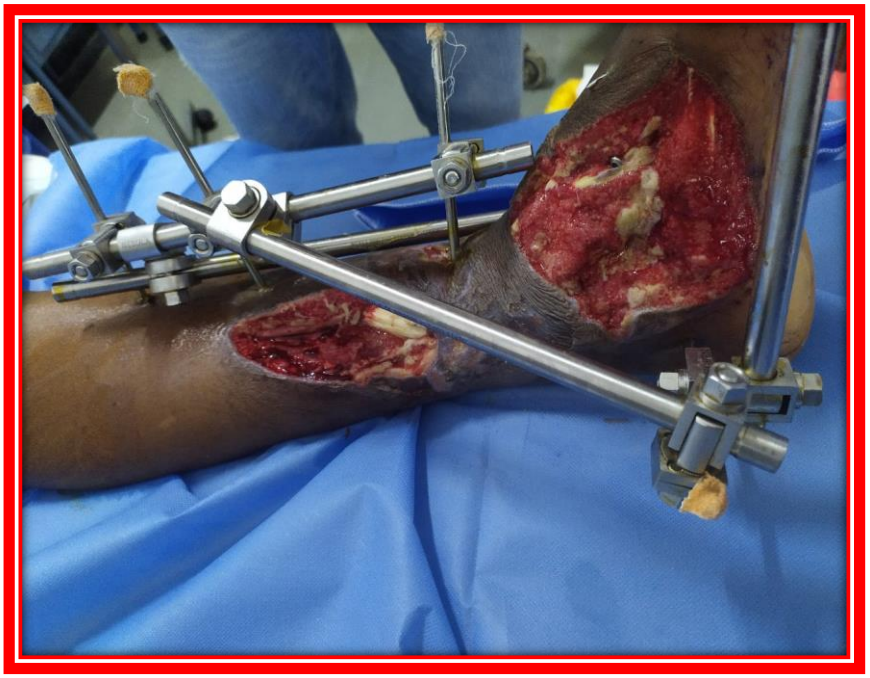
Area of wound was measured in vertical dimension



Area of wound was measured in horizontal dimension



VAC application was done



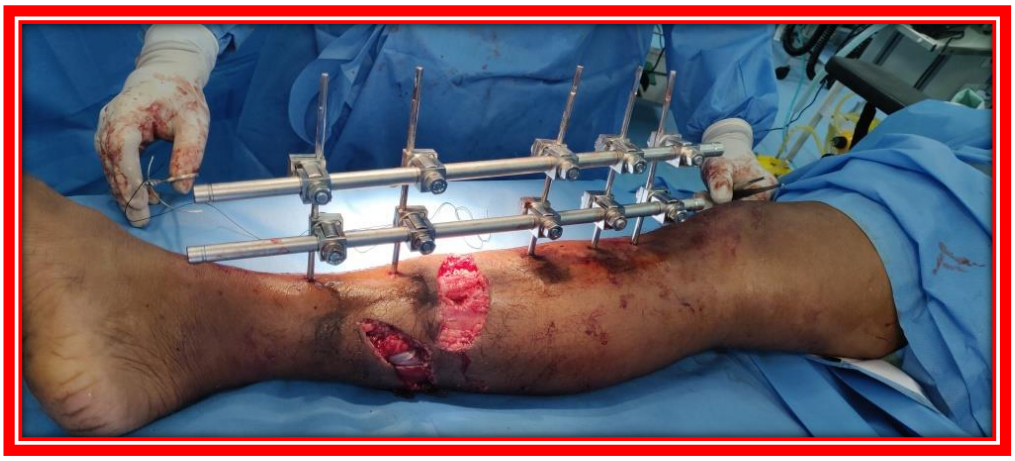
Post VAC image

Drain collected in canister

Post SSG

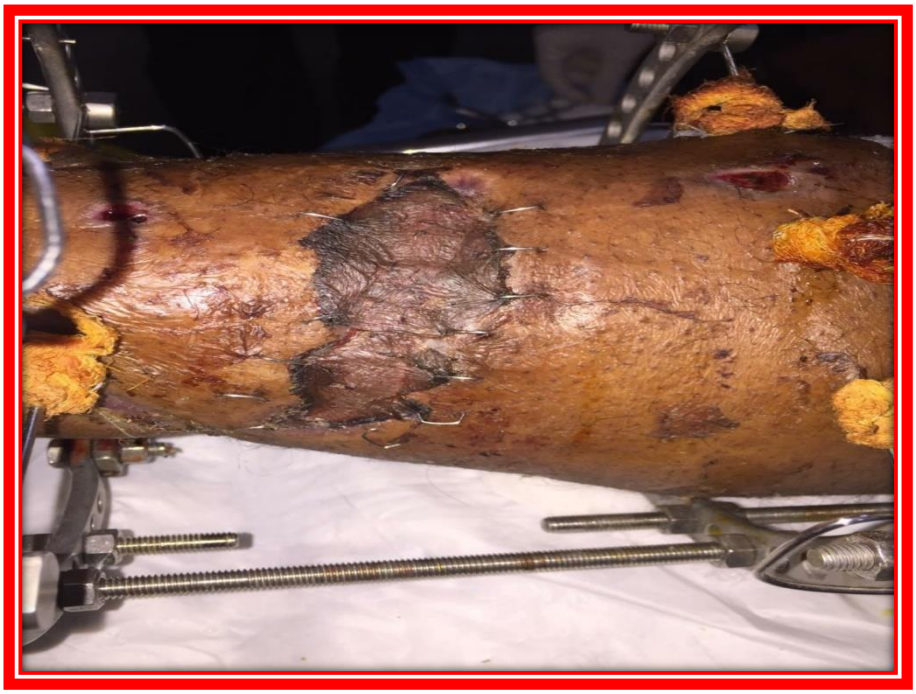
PATIENT 2:

46 year old male with open type 3b fracture of right leg at diaphyseal region without distal neuro vascular deficit

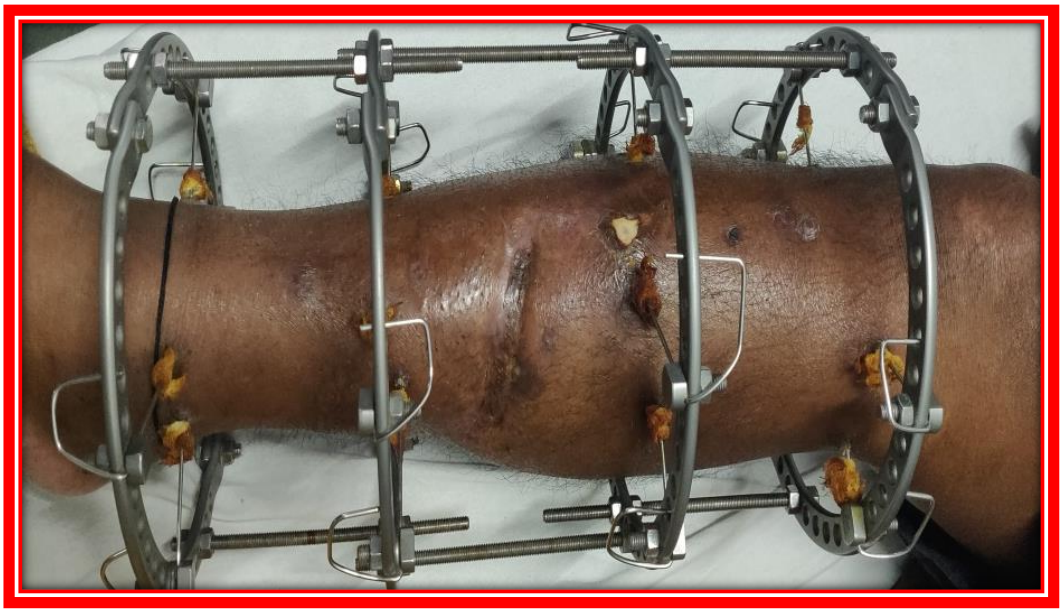


Intraoperative image after wound debridement + Exfix application

Bedside image after VAC application



VAC removal + SSG application



Post SSG image

ANNEXURE-VI

KEY TO MASTER CHART

M	-	Male
F	-	Female
UHID. No	-	Unique hospital identification
numberRTA	-	Road Traffic Accident
EF	-	External fixation
IF	-	Internal Fixation

MASTER CHART

UHID NO	AGE	SEX	TRAUMA	TYPE OF OPEN FRACTURE	NEURO VASCULAR DEFECT	FRACTURE	INITIAL SURGERY	TYPE OF FIXATION	OPEN WOUND GRADING SYSTEM	TIME INTERVAL BETWEEN TRAUMA AND INITIAL DEBRIDEMENT	DIMENSIONS OF INITIAL WOUND(cm) before NPWT	CO MORBIDITIES	DIMENSION OF WOUND (cm) post NPWT	WOUND COMPLICATIONS	DEEP INFECTION	PRE VAC INFECTION	POST VAC INFECTION	NUMBER OF DRESSINGS	DURATION OF HOSPITAL STAY(day's)	DURATION OF VAC(day's)	WOUND HEALING TIME(day's)	SKIN GRAFT	DURATION OF FOLLOW UP (weeks)
79610	42	M	RTA	3B	NO	LEFT FEMUR SHAFT	EF	EXTERNAL FIXATION	2	8 hours	16X10	NIL	8X4	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	20	12	17	YES	3
814254	46	M	RTA	3A	NO	RIGHT TIBIA DIAPHYSIS	EF	EXTERNAL FIXATION	2	8 hours	12X6	NIL	6X3	NIL	NIL	STAPH AUREUS	NIL	1	11	6	11	YES	2
814319	38	M	RTA	3B	NO	RIGHT CALCANEUM	PERCUTANEOUS PINNING	EXTERNAL FIXATION	3	12 hours	8X6	DIABETES	4X2	SKIN MACERATION	YES	E.COLI,ACINETOBACTER	ACINETOBACTER	2	20	14	19	NO	3
833804	41	F	RTA	3A	NO	LEFT DISTAL TIBIA + LEFT	EF + PERCUTANEOUS	EXTERNAL FIXATION	3	12 hours	14X8	ANAEMIA	10X6	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	19	12	18	YES	3
819464	36	M	RTA	3B	NO	3RD,4TH & 5TH	PERCUTANEOUS PINNING	EXTERNAL FIXATION	3	8 hours	10X8	NIL	7X5	NIL	NIL	KLEBSIELLA,E.COLI	NIL	1	12	6	12	NO	2
815257	47	M	WORK PLACE	3A	NO	RIGHT DISTAL FEMUR +	IF	INTERNAL FIXATION	2	24 hours	14X8	DIABETES	10X5	SKIN MACERATION	NIL	KLEBSIELLA,PSEUDOMONAS	NIL	3	26	18	25	YES	4
839984	32	F	RTA	3A	NO	RIGHT DISTAL TIBIA	IF	INTERNAL FIXATION	2	8 hours	12X8	NIL	8X5	NIL	NIL	E.COLI	NIL	1	12	6	12	NO	2
824920	36	M	RTA	3B	NO	RIGHT SEGMENTAL	EF	EXTERNAL FIXATION	2	8 hours	14X5	NIL	11X3	SKIN MACERATION	YES	STAPH AUREUS.ACIN	STAPH AUREUS	3	28	18	26	YES	4
877491	39	F	WORK PLACE	3A	NO	RIGHT MULTIPLE	PERCUTANEOUS PINNING	EXTERNAL FIXATION	4	48 hours	12X4	NIL	8X2	NIL	NIL	STAPH AUREUS	NIL	1	15	6	14	NO	3
848787	58	F	RTA	3B	NO	LEFT DISTAL FEMUR	EF	EXTERNAL FIXATION	2	8 hours	12X8	HYPERTENSION	10X6	NIL	NIL	NO GROWTH	NIL	1	12	6	12	YES	2
847998	24	M	WORK PLACE	3A	NO	LEFT SHAFT OF FEMUR	IF	INTERNAL FIXATION	2	12 hours	14X5	NIL	12X3	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	19	12	18	YES	3
848169	39	M	RTA	3B	NO	LEFT CALCANEUM	EF	EXTERNAL FIXATION	3	8 hours	10X5	NIL	7X3	NIL	NIL	PROTEUS	NIL	1	10	6	10	NO	2
867304	22	M	RTA	3B	NO	BOTH BONE SEGMENTAL	IF	INTERNAL FIXATION	2	12 hours	10X8	NIL	8X5	SKIN MACERATION	YES	STAPH AUREUS,PSEU	PSEUDOMONAS	3	26	18	24	YES	4
877503	45	M	RTA	3B	NO	RIGHT MEDIAL	PERCUTANEOUS PINNING	EXTERNAL FIXATION	3	12 hours	8X4	NIL	4X2	NIL	NIL	NO GROWTH	NIL	1	14	6	14	NO	3
840634	31	F	RTA	3B	NO	BOTH BONE FRACTURE AT	EF	EXTERNAL FIXATION	3	12 hours	12X6	ANAEMIA	10X4	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	20	12	18	YES	3
879030	37	M	WORK PLACE	3A	NO	LEFT DISTAL FEMUR	EF	EXTERNAL FIXATION	2	8 hours	14X6	NIL	11X3	SKIN MACERATION	NIL	E.COLI	NIL	2	22	12	20	YES	3
893337	29	M	WORK PLACE	3A	NO	DIAPHYSEAL FRACTURE OF	EF	EXTERNAL FIXATION	2	8 hours	18X6	NIL	15X4	SKIN MACERATION	YES	STAPH AUREUS,PSEU	STAPH AUREUS	3	28	18	26	YES	4
855760	36	M	RTA	3B	NO	DIAPHYSEAL FRACTURE OF	EF	EXTERNAL FIXATION	2	8 hours	12X4	NIL	9X2	SKIN MACERATION	NIL	PROTEUS	NIL	2	18	12	18	YES	3
864218	28	F	RTA	3B	NO	SEGMENTAL FRACTURE OF	IF	INTERNAL FIXATION	3	8 hours	10X6	THYROID DISORDER	7X3	NIL	NIL	ACINETOBACTER	NIL	1	15	6	15	NO	3
871387	34	M	RTA	3B	NO	MULTIPLE METATARSAL	PERCUTANEOUS PINNING	EXTERNAL FIXATION	4	48 hours	8X4	NIL	5X2	NIL	NIL	KLEBSIELLA	NIL	1	16	6	15	NO	3
878671	45	M	RTA	3A	NO	LEFT TALUS FRACTURE	PERCUTANEOUS PINNING	EXTERNAL FIXATION	4	24 hours	10X4	DIABETES	8X3	SKIN MACERATION	NIL	ACINETOBACTER	NIL	2	21	12	20	YES	3
877688	39	M	RTA	3B	NO	DIAPHYSEAL FRACTURE OF	EF	EXTERNAL FIXATION	2	8 hours	14X4	NIL	11X2	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	24	12	24	YES	4
899069	32	M	RTA	3B	NO	LEFT SHAFT OF FEMUR	IF	INTERNAL FIXATION	2	12 hours	10X4	NIL	8X2	SKIN MACERATION	NIL	STAPH AUREUS	NIL	3	30	18	28	YES	4
839523	37	F	RTA	3A	NO	RIGHT BIMALLEOLAR	EF	EXTERNAL FIXATION	3	8 hours	8X8	NIL	4X5	NIL	NIL	ACINETOBACTER	NIL	1	15	6	14	NO	2
899371	30	M	RTA	3B	NO	RIGHT CALCANEUM	PERCUTANEOUS PINNING	EXTERNAL FIXATION	3	8 hours	6X6	NIL	4X2	NIL	NIL	NO GROWTH	NIL	1	13	6	12	NO	2
897867	56	M	WORK PLACE	3A	NO	SEGMENTAL FRACTURE OF	EF	EXTERNAL FIXATION	2	8 hours	12X6	NIL	10X4	SKIN MACERATION	NIL	PSEUDOMONAS	NIL	2	21	12	20	YES	3
897299	22	M	RTA	3A	NO	DIAPHYSEAL FRACTURE OF	IF	INTERNAL FIXATION	2	12 hours	10X6	NIL	8X3	SKIN MACERATION	YES	PROTEUS	PROTEUS	2	23	12	22	YES	4
929767	20	M	RTA	3B	NO	RIGHT SEGMENTAL	IF	INTERNAL FIXATION	2	12 hours	8X6	NIL	6X3	SKIN MACERATION	NIL	KLEBSIELLA	NIL	3	30	18	28	YES	5
929465	51	M	RTA	3B	NO	MULTIPLE METATARSAL	PERCUTANEOUS PINNING	EXTERNAL FIXATION	3	8 hours	6X6	HYPERTENSION	4X4	SKIN MACERATION	YES	KLEBSIELLA,PSEUDOMONAS	PSEUDOMONAS	2	24	12	24	NO	4
912907	30	M	RTA	3B	NO	RIGHT 3RD &4TH	PERCUTANEOUS PINNING	EXTERNAL FIXATION	3	8 hours	8X4	NIL	5X2	NIL	NIL	STAPH AUREUS	NIL	1	12	6	12	YES	2
910963	26	M	RTA	3B	NO	LEFT BIMALLEOLAR	EF	EXTERNAL FIXATION	3	12 hours	10X8	NIL	8X6	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	20	12	20	YES	3
933676	24	M	RTA	3B	NO	LEFT CALCANEUM	PERCUTANEOUS PINNING	EXTERNAL FIXATION	2	12 hours	8X4	NIL	5X2	NIL	NIL	PROTEUS	NIL	1	14	6	12	NO	2
944371	49	M	WORK PLACE	3B	NO	BOTH BONE FRACTURE OF	IF	INTERNAL FIXATION	2	8 hours	12X6	NIL	10X4	SKIN MACERATION	NIL	STAPH AUREUS	NIL	2	21	12	20	YES	4
946251	59	F	RTA	3B	NO	RIGHT TALUS +	PERCUTANEOUS PINNING	EXTERNAL FIXATION	4	48 hours	8X6	DIABETES	6X4	SKIN MACERATION	NIL	PSEUDOMONAS	NIL	3	28	18	28	YES	5