A CLINICAL STUDY ON EFFICACY OF NANOCRYSTALLINE SILVER DRESSING IN DIABETIC FOOT ULCERS

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ABSTRACT: INTRODUCTION: Diabetes is the highest risk factors associated with limb threatening ischemia. Foot ulceration and infection are the major causes of hospitalization in people with diabetes. Signs of infections in these lesions are likely to be masked as people with diabetes may not show typical inflammatory response to infection. Despite this inherent difficulty, diagnosis of infection is essentially clinical. In addition infection of diabetic foot often involves superficial and deep tissues, including bone. Dressings have a part to play in the management of wounds. They traditionally provide a moist wound environment, handle excessive exudate, aid in debridement, and promote disorganised, stalled healing. The control of infection remains a major challenge. Nano-technology makes it possible to expand the surface area of Silver particles markedly to Nano scale. They markedly expand the surface area of silver particles, increasing their contact with bacteria or fungi, and vastly improving its bactericidal and fungicidal effectiveness.

AIMS AND OBJECTIVES: 1. To study the efficacy of Nano crystalline silver in the management of diabetic foot ulcer. 2. To compare time required for healing of diabetic foot ulcer with Nano crystalline silver against conventional dressing. 3. Number of Nano crystalline silver dressing required in comparison with conventional dressing.

STUDY DESIGN: Prospective and observational study.

SAMPLE SIZE: 100 cases were studied prospectively.

INCLUSION CRITERIA: Patients aged more than 20years with diabetic foot ulcer, Ulcers of size less than 10×10 cm.

EXCLUSION CRITERIA: Clinical signs of infection, cellulites; X-ray showing Osteomyelitis. Doppler showing atherosclerosis and varicosities. Uncontrolled diabetes, significant medical diseases. Patients on steroids, immunosuppressive agents, radiation, or chemotherapy.

STUDY SETTING: S. V. R. R. G. G. Hospital, Tirupati.

STUDY PERIOD: From Jan 2013 till November 2014.

STUDY METHODS: Patient fulfilling the inclusion and exclusion criteria is selected. Consent is taken. Patients are randomly selected into two groups. Cases in the test group will be treated with topical Nano crystalline silver dressings with bedside surgical debridement will be done whenever indicated. The control group will be treated with conventional topical dressings and bedside debridement. The patients will be followed up for 7 weeks and ulcer status will be noted using visual score.

CONCLUSION: Nano silver dressings are safe, effective, with a slight beneficial edge to the conventional dressing with solutions like betadine in terms of promoting wound healing, and are more patient compliant in view of Less pain while changing the dressing, Less number of dressings required, Less duration of hospital stay. This indicate that Nano silver dressings may be used as an adjunct in management of diabetic foot ulcer and seems to be more efficient than conventional dressings in this regard.

KEYWORDS: Diabetic Foot, Debridement, Nano Crystal Technology, Nano silver Dressings.

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INTRODUCTION: Diabetes mellitus is a universal disease, which appears to spare no age group from child hood to old age. It is the disease which affects whole of the body involving many of the organs and systems, notably the eyes, the kidney, the blood vessels and peripheral nerves.[1] Diabetic foot is one of the major complications of diabetic patient; about 3-4% of individuals with diabetes mellitus currently have foot ulcers or deep infection. 15% develops foot ulcers during their life time. If untreated they end in lower extremity amputation. 85% of non-traumatic lower extremity amputations are due to diabetic foot.[2,3,4] The high prevalence of diabetes mellitus in India is 5% in urban and 1 to 1.5% in rural that is 60 million feet are at risk.[5] This puts a staggering load on any attempt to provide on organized diabetic foot care service.

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PATHOPHYSIOLOGY OF DIABETIC FOOT: Altered metabolism: Diabetes mellitus is a metabolic disorder and hence the defects observed in diabetic wound healing are thought to be the result of altered protein and lipid metabolism and thereby abnormal granulation tissue formation.[6]

Impaired No Synthesis: Nitric oxide increases fibroblast proliferation and thereby collagen production in wound healing. Also, L-arginine and nitric oxide are required for proper cross linking of collagen fibers, via proline, to minimize scarring and maximize the tensile strength of healed tissue.[7] The diabetic patients exhibit reduced ability to generate nitric oxide due to reduced production of nitric oxide synthase.[8,9]

There are 4 main causes for development of foot lesion in a diabetic:

1. **Neuropathy:**[10] The commonest Diabetic poly neuropathy is distal symmetrical neuropathy, affecting about 30% of all diabetic people.[11,12] Vascular and metabolic factors are both implicated in the pathogenesis of diabetic neuropathy. Proposed hypotheses are chronic hyperglycemia, Oxidative stress, Neurotrophic factors, Vascular factors.[13]
2. **Peripheral Vascular disease(PVD):**[14,15] Macro vascular disease like Atherosclerosis, Medial calcification, Diffuse intimal fibrosis and Micro vascular disease like Arteriosclerosis, Specific diabetic microangiopathy, diabetic Fibrosis.
3. **Charcot foot:**[16,17] Bone and joint damage in the tarso metatarsal joints and mid-tarsal joints leads to two classical deformities, The Rocker bottom deformity and The Medial convexity.
4. **Infection:** Infection of the plantar space accounts majority of diabetic foot infections. Majority start with infected ulcers on the plantar aspect of the foot and nail bed infections. In the initial stage there is usually an area of localised cellulites, then infection progresses to deep space infection, ascending cellulitis, lymphangitis and lymphadenopathy.

Classification of Severity of Foot Infection in Patients with Diabetes Mellitus: Lipsky,[18] has proposed a classification that utilizes depth of a wound, presence of ischemia, presence of infection, and systemic toxicity to designate severity of wound infection.

MICROBIOLOGY:[19] A simple practical classification of foot infection is:

Non-limb-threatening infections are those occurring in patients who have not previously received antimicrobial therapy. Staph aureus and Group B streptococci, are the predominant pathogens.[20,21]

Limb-threatening foot infections are generally polymicrobial. Cultures from these infections yield on average 4.1-5.8 bacterial species per culture. Both gram-positive cocci and gram-negative rods are commonly isolated from a single lesion, and in 40% of Infections both aerobic and anaerobic organisms are recovered.[22,23,24]

Antibiotic Therapy: Antimicrobial treatment in patients with diabetes is empirical and thereafter revised based on the results of cultures. Lipsky et al.[25] Demonstrated that oral therapy with clindamycin or cephalexin for 2 weeks in patients with previously untreated non-limb-threatening foot infection resulted in satisfactory clinical outcome in 96 and 86%, respectively.

Biomechanics of Ulcer Formation: Motor neuropathy results in 'claw' toes and prominent pressure points over the metatarsal heads. This when combined with sensory neuropathy, the stage is set for unperceived pressure injury to the foot. The autonomic neuropathy causes loss of sweat and oil gland activity results in dry skin that easily cracks fissures. In addition to these non-vascular effects neuropathy can directly impair effective perfusion due to arterio venus shunting of blood around the capillary bed. In addition neuropathy destroys "C" fibers which are responsible for nociceptive response which is the basis for wheal and flare response of skin to a noxious stimulus. To this compromised foot, the cumulative effect of glycation and diminished perfusion due to macro angiopathy results in ulceration. The insensitive foot can be injured by external forces in three main ways. Before going into them simple equation:

Pressure=Force / area must be remembered.

1. A constant pressure maintained for several hours may cause ischemic necrosis.
2. A high pressure for a short period of time will cause direct mechanical damage.
3. Repetitive moderate stresses probably represent the most common cause of foot ulceration among neuropathic patients.

Signs and Symptoms:

a) Neuropathy:[26] The signs and symptoms of neuropathy include - paresthesia, hyperasthesia, hypoesthesia, radicular pain, loss of vibratory and position sense, anhydrosis, and heavy callus formation over pressure points and trophic ulcers. Radiographic changes are demineralistion, osteolysis, Charcot's joint. Autonomic Neuropathy Syndromes Cardiovascular, Gastrointestinal, Genitourinary symptoms.

b) Vascular Insufficiency:[27] Intermittent claudication, cold feet, nocturnal pain, rest pain, absent pulses, blanching on elevation, dependent rubor, atrophy of subcutaneous fatty tissues, shiny skin, loss of hair on foot and toes, thickened nails, gangrene, blue toe syndrome, acute vascular occlusion.

Major Infections:

1) Cellulitis: These patients manifests with edema involving dorsum of the foot with shiny skin.

2) Abscess: The most important signs are swelling and redness which can be seen on the dorsum of the web spaces of the foot. The most characteristic sign is separation of the toes due to diffuse edema of the deep tissues of the foot.

3) Ulcer: Ulcer may present on the dorsal or plantar aspect of the foot. Plantar ulcers also called as trophic or penetrating ulcers which are typically painless. The earliest change is an area of hyperkeratosis often over a metatarsal head.

4) **Gangrene:** Areas of gangrene may occur on parts of the foot that are exposed to pressure.

INVESTIGATIONS:

a. Urine Sugar & Ketone Bodies.
b. Fasting & Post Prandial Blood Sugar.
c. Glucose Tolerance Test.
d. Metabolic Profile and Glycosylated Hemoglobin.
e. Culture and Sensitivity Test.
f. Radiology of the Foot: for Osteomyelitis changes.
g. Noninvasive vascular laboratory study.
   - Pulse-volume recording(PVR) or plethysmography:
   - Duplex Scan.
   - CT Scan and MRI.
   - Contrast Angiography.
   - Magnetic Resonance Angiography (MRA).
   - Ankle-Brachial index (ABI).

TREATMENT: Classification and Staging:[28,29,30] After completing the basic assessment, it will now be possible to classify the diabetic foot. For practical purposes, the diabetic foot can be divided into two distinct entities: The neuropathic foot & the neuroischaemic foot.

1. **Neuropathic Foot:** The neuropathic foot is a warm, well-perfused foot with bounding pulses and distended dorsal veins due to arteriovenous shunting. Sweating is diminished, so skin and any callus tend to be hard and dry and prone to fissuring. Toes are flexed and the arch of the foot may be raised. Ulceration commonly develops on the sole of the foot, associated with neglected callus and high plantar pressures. Despite the good circulation, necrosis can develop secondary to severe infection. The neuropathic foot is also prone to bone and joint a problem which is referred as Charcot's osteoarthropathy.
2. **Neuroischaemic Foot:** The neuroischaemic foot is a cool, pulseless foot with poor perfusion and almost invariably also has neuropathy. The colour of the severely ischaemic foot can be a deceptively healthy pink or red caused by dilatation of capillaries in an attempt to improve perfusion. The neuroischaemic foot may be complicated by swelling, often secondary to cardiac failure or renal impairment. Ischaemic ulcers are commonly seen around the edges of the foot, including the apices of the toes and the back of the heel, and are associated with trauma or wearing unsuitable shoes. The neuroischaemic foot develops necrosis in the presence of infection or if tissue perfusion is critically diminished. Even if neuropathy is present and plantar pressures are high, plantar ulceration is rare. This is probably because the foot does not develop heavy callus, which requires good blood flow.

The natural history of the diabetic foot can be divided into six stages:

Stage 1: The patient does not have risk factors like neuropathy, ischaemia, deformity, callus and swelling rendering. The normal foot is characterized by no symptoms, including no pain and examination is normal.

Stage 2: The patient has developed one or more of the risk factors. Patients without active foot ulceration but a history of ulceration should be regarded as very high risk. Within stage 2 there are specific conditions which are nonulcerative but require treatment. These include: Severe chronic ischaemia & Acute ischemia.

Stage 3: The foot has a skin breakdown, minor injuries such as blisters, skin fissures or grazes, all of which have a propensity to become ulcer. Ulceration is usually on the plantar surface in the neuropathic foot and on the margin in the neuro-ischemic foot.

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

Stage 5: Necrosis has supervened. In the neuropathic foot, infection is usually the cause. In the neuro-ischaemic foot, infection is still the most common reason for tissue destruction although ischaemia contributes.

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

Other classifications of the diabetic foot such as the Wagner system, two more recently described classification systems, SAD system, (size, area, depth), sepsis, arteriopathy and denervation) and the PEDIS (perfusion, extent, depth, infection, sensation) systems.

Wagner-Meggits Classification of Diabetic Foot lesions:

Grade 0: At risk foot no obvious ulcer, but thick callus prominent metatarsal heads, claw toes or any bony abnormality.

Grade I: Superficial ulcer not clinically infected.

Grade II: Deeper ulcer often infected but no bone involvement.

Grade III: Deep ulcer, abscess formation bony involvement.

Grade IV: Localized Gangrene.

Grade V: Gangrene of the whole foot.

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

Primary Treatment of Foot Ulcers: When the examination reveals a foot at stage 3, 4 or 5, there is a need for a great sense of urgency. Treatment should begin without delay. Successful management needs the expertise of a multidisciplinary team including the following: Podiatrist, Physician, Nurse, Orthopaedic Surgeon, Radiologist, Surgeon.

Strategies for Saving the Diabetic Foot: Correction of vascular risk factors; Improve the circulation; Regular foot Inspections; Treatment of foot ulcers; Treatment among

medical disciplines; Prescribing special shoes; Patient Education.

Improving Circulation: A regular walking program will improve walking distance in 80% of the patients. Cilastozole is also used to improve circulation. The ultimate treatment for claudication is vascular surgery is to by-pass narrowed vessels or endarterectomy. Transcutaneous angioplasty is also useful. The surgical approach to aorto-iliac occlusive disease is aorto-ilac endarterectomy, bypass grafting and extra-anatomic reconstructions. In the patients with simple occlusion of the superficial femoral artery, autogenous saphenous vein bypass from the common femoral artery to the popliteal artery is done. For patients with significant disease in the above knee, a bypass to the distal popliteal artery is done.

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

SURGICAL MANAGEMENT:

Debridement:[31]

1. **Surgical/Sharp Debridement:** Surgical/sharp debridement is usually painful; therefore, the patient should be premedicated with systemic analgesics and/or local anesthetics.
2. **Mechanical debridement:** Mechanical debridement uses force to remove necrotic tissue. This may include the use of irrigation fluids delivered by pulsed lavage, the agitation of water during whirlpool therapy, or the removal of tissue with wet-to-dry dressings.
3. **Autolytic Debridement:** Autolytic debridement can be accomplished by placing occlusive or semiocclusive moisture-retentive dressings (eg, hydrocolloids or transparent films) over the wound and allowing the natural wound fluids to soften eschar; proteolytic enzymes within the wound fluids digest and liquefy necrotic tissue.
4. **Enzymatic Debridement:** Enzymatic debridement uses topical enzymes to remove necrotic tissue by digesting and dissolving the devitalized tissue in the wound bed. Two enzymatic debriding agents are currently available; one preparation contains papain and urea in a cream base; the other is composed of Collagenase in a petrolatum base.
5. **Biologic Debridement:** Biologic debridement is the use of living organisms to remove necrotic or dead tissue from a wound. The most commonly used biologic debridement method involves the use of maggots that have been medically sterilized or disinfected.
6. **Ultrasonic Debridement**: It employs an electrical current, which piezoelectric crystals then convert to mechanical vibrations. The mechanical vibrations stimulate a probe, which in turn amplifies the vibrations. This mechanical energy converts into acoustic energy, which subsequently transfers to the tissue in the wound bed and peri-wound tissue.

Nano Silver Compounds:[32,33]

Silver is one of the most famous antimicrobial substances. Nano-technology makes it possible to expand the surface area of Silver particles markedly to nano scale. They markedly expand the surface area of silver particles, increasing their contact with bacteria or fungi, and vastly improving its bactericidal and fungicidal effectiveness.

Silver has antiseptic, antimicrobial, anti-inflammatory properties and is a broad spectrum antibiotic.

Mechanism of Action: Silver is biologically active when it is in soluble form i.e., as Ag+ or Ag0 clusters Ag0 is the uncharged form of metallic silver present in nano crystalline silver (Dunn 2004). Free silver cat ions have a potent antimicrobial effect which destroys microorganisms immediately by blocking the cellular respiration and disrupting the function of bacterial cell membranes. This occurs when silver cat ions bind to tissue proteins, causing structural changes in the bacterial cell membranes which in turn cause cell death. Silver cat ions also bind and denature the bacterial DNA and RNA, thus inhibiting cell replication.

Silver has a very broad spectrum of microbial coverage, including yeast, fungi, mold, and even antibiotic-resistant bacteria such as methicillin-resistant Staph aureus (MRSA) and vancomycin-resistant enterococci (VRE) when used at appropriate concentrations. Silver is a bactericidal material that kills on contact by inhibiting the respiratory chain at the cytochrome level, as well as, interfering with electron transport, denaturing nucleic acids, inhibiting DNA replication, and altering cell membrane permeability.

The challenge is to keep a high enough concentration of agent with a long enough residual activity to prevent developing resistance. The suggested concentration of silver in any preparation is greater than 30 to 40mg/L to be effective. Nanocrystalline silver technology is able to more consistently maintain adequate concentrations (at least 70mg/L) with good residual activity, keeping levels elevated over longer periods of time.

It Acts by Following Mechanisms:

1. Provides moisture to the wound bed, preventing dehydration of granulation tissue & also promotes autolytic debridement.
2. Will allow the vital gaseous exchange across its surface but wouldn't allow entry of microbes in the wound, thus forming antiseptic and particle barrier.
3. Will soothe pain, through its cooling effect.
4. Provides slow, extended release of active ionic silver for broad antimicrobial effectiveness and helps to prevent contamination from external bacteria.
5. Decreases wound surface contamination.
6. Promotes wound healing through inhibition of Matrix Metalloproteinases.

So far no major microbial resistance has been noted with silver and amorphous form gives better results than silver salts. It has extended antimicrobial spectrum involving Pseudomonas and MRSA as well. It is biocompatible and can be washed away without disruption of granulation tissue. Being transparent gel it will allow direct monitoring of the wound. Such an environment facilitates and enhances wound healing. It is non-cytotoxic, nonirritating and non-staining.

Indications: Pressure Ulcers, Venous Ulcers, Dermal lesions, Second degree burns, Donor Sites.

Precautions: When visible signs of infection are present in the wound, proper antibiotics should be used, debrided along with silver crystals.

Contraindications: There are no absolute contraindications but its utility is limited when used in heavily exudating wounds or wounds covered with thick eschar.

Other surgical options are Skin Grafting,[34,35] Skin and Muscle Flaps[36] and Amputation.[37,38]

AIMS AND OBJECTIVES:

1. To study the efficacy of Nano crystalline silver in the management of diabetic foot ulcer.
2. To compare time required for healing of diabetic foot ulcer with Nano crystalline silver against conventional dressing.
3. Number of Nano crystalline silver dressing required in comparison with conventional dressing.

MATERIALS AND METHODS: The study was a prospective, observational and comparative study. The patients who were admitted with diabetic foot ulcer in S.V.R.R.G.G. Hospital, Tirupati from November 2013 to November 2014 were included. The number of patients included in our study was 100 out of which 50 were in test group with conventional dressings and 50 were in another test group with Nano silver dressings.

Inclusion Criteria:

1. Patients aged more than 20years with diabetic foot ulcer.
2. Ulcers of size less than 10×10 cm.

Exclusion Criteria:

1. Clinical signs of infection, cellulites; X-ray showing Osteomyelitis.
2. Doppler showing gross atherosclerotic arterial changes and venous abnormalities like varicosities.
3. Malnutrition, uncontrolled diabetes.
4. Other clinically significant medical conditions that would impair wound healing like renal, hepatic, hematological, neurological, and immunological diseases.
5. Patients receiving corticosteroids, immunosuppressive agents, radiation, or chemotherapy within one month prior to entry into the study were also excluded.

Method of Collection of Data: A detailed history was taken including history of the mode of onset, duration, progress of ulcer and information about diabetes status was obtained in the form of duration of DM and medication. Ulcer examination was performed. All the patients underwent the following investigations: Hb, blood sugar levels, serum Creatinine, urine ketone bodies, X ray foot, Doppler (both arterial and venous).

Dressing Technique:

For Conventional Dressing: If ulcer contained slough, patients underwent debridement to have clean ulcer base. Following debridement, base was cleaned once with wet gauze piece soaked in normal saline. Wet gauze piece in betadine solution was kept over the ulcer which was covered with gauze pad and roller bandage.

For Nano silver solution/gel Dressings: After cleaning the ulcer with help of normal saline, silver nano gel applied and the wound was closed with gauze pad and roller bandage.

Dressings are changed according the need once or twice in the day if there is soakage of dressing. Patients are discharged from the hospital after significant reduction in ulcer size or in some cases after formation of granulation tissue and after applying partial thickness skin graft.

Percentage reduction in the ulcer size was measured at the time of discharge using Ulcer Planometry. Number of dressings required for each group, Number of days of hospital stay were recorded. Patients are reevaluated on the outpatient basis for 2 months.

OBSERVATIONS AND RESULTS: Most of the patients fell in the age group between 51-60 years. The Mean±SD for test group 1 is 52.73±9.880 and test group 2 is 55.50±12.113. So age distribution is statistically similar between the two groups with P=0.215.

DISCUSSION: The numbers of patients studied were 100 and randomly divided into two Test groups of 50 each. Both groups were matched.

Age Distribution: In our study the Mean±SD for test group 1 is 52.73±9.880 and test group 2 is 55.50±12.113. Thus the prevalence of leg ulceration progressively increases with increasing age.

Diabetic foot ulcers are more common in 5^{th}-6^{th} decade. The prevalence of diabetes increases with age.[39] Age distribution in our study is in accordance to the age distribution of the study conducted by Rao Harish et al in 2012.

Sex Distribution: In our study total numbers of males are 65(65%) and females are 35(35%). In the study conducted by Rao Harish et al, 2012[40] males (75%) had increased incidence of chronic leg ulcer as compared to female (25%).

The NHDS(National Hospital Discharge Survey) a well-known govt. source, documented higher hospital rates of diabetic foot and admissions in males.[41] Sex distribution in the present study is similar to that of Frank J moffat et al[42] i.e., Males have increased incidence of diabetic foot ulcer compared to that of females.

Onset of Diabetic Foot Ulcer: Almost all the time we came across the diabetic ulcer we could find trauma being the common cause for developing the ulcer. The reason is obvious. It is the neuropathy that triggers the ulcer formation. In our study also 69% of the study subjects acquired ulcers out of trauma and the rest developed spontaneously by means of rupture of bullae.

Site of Ulcer: In a study conducted by Edmond et al, in 1986,[43] it was proven that 93% of foot ulcers were on plantar and forefoot area. Most of the diabetic foot ulcers are invariably shoe related and due to gait abnormalities. They can be prevented by appropriately sized foot wear.

Diabetic foot ulcer is more common on plantar aspect of foot. In our study incidence of ulcer on plantar aspect is 69 % which proves the above thesis.

Duration of Hospital Stay: In the present study, duration of hospital stay is 20.54±7.044 days in test group1 and 36.19±7.24 days in test group 2.

In a study conducted by Charne N Miller at al,[44] the mean wound healing rates were similar for the silver and iodine groups with silver recording a marginally higher healing rate(average 52.10; SD 51.89) compared with iodine(average 51.69; SD 52.46). Although there was no significant difference in the time taken for wound healing when compared.

Percentage Reduction of Ulcer: In the present study percentage reduction of ulcer with Nano silver gel dressing is 94.81% and with conventional dressing is 84.69% which is significant. (P value<0.001).

In the study by Charne N Miller et al, a comparison of the number of wounds that healed within each treatment group was explored for the wound duration and wound size segmentations. In similar findings, there was no difference between the treatment groups in the number of wounds healed overall for young [w2(1) 50.07, p > 0.05] and old wounds [w2(1)50.17, p>0.05] as well as for small [w2(1)50.10, p > 0.05] and big wounds [w2(1)50.02, p > 0.05]. In our study, Percentage Reduction of Ulcer is more than that of the study conducted by Charne N Miller et al.

Under circumstances provided silver dressings were more effective in comparison with the routine conventional dressings in healing the diabetic ulcers.

Number of Dressings: In our study number of nano silver gel dressings required per patient is significantly less compared to conventional dressing group.

In the study by Charne N Miller et al, it was concluded by the results that the time taken and number of dressings taken for healing of ulcers in both cases with silver and betadine were similar.

In another RCT conducted by Munter KC, Beele H, the number of dressings for chronic wounds with silver dressings were on an average 18. The main objective of the study was to evaluate the time required for healing which was less in Nano silver dressing and also the number of dressings required are also less when compared with conventional dressings. Mean±SD of Test group1 and test group 2 are 10.92±3.052 and 17.54±6.008 and P<0.001, which is a significant result.

Thus our study is in accordance with the results of study by Charne N Miller et al.

SUMMARY: The numbers of patients studied were 100 and randomly divided into two Test groups of 50 each. Both groups were matched.

One group was dressed with Nano silver gel and the other group with conventional dressings. A comparative study was done between the both groups regarding the time of healing, number of dressings and percentage reduction of ulcer size.

1. In Age distribution, 37 patients are between 51-60yrs of age.
2. In Sex distribution, Males(65) are affected more than females(35).
3. In Mode of onset, 69% of ulcers are traumatic in onset.
4. Plantar aspect and forefoot are most common sites of ulcer in 69%.
5. Fasting Blood sugar levels between 101- 200 mg% are in 81% patients.
6. Post prandial Blood sugar levels between 201- 300 mg% are in 71%.
7. Patients receiving insulin were 55%, for good glycemic control.
8. Average duration of hospital stay is less in test group1 where patients stay for 2-4 weeks and in test group 2, a patient stay for 5-6 weeks and P value<0.001 and is highly significant.
9. Percentage reduction of ulcer is between 91-99%, in 45 patients of test group1. While only 10 patients in test group 2 had 91-99% reduction in ulcer size with P value<0.001 and is highly significant.
10. Number of dressings required per patient are10.92±3.052 in Test group 1 and is 17.54±6.008 in test group 2, P value<0.001 and is significant.

Thus using Nano silver dressings in the treatment of diabetic foot ulcers are found to be safe, effective, promoter of wound healing, promotes epithelization, accelerates healing, eliminates anaerobes and breaks microbial synergy more effectively than conventional dressing. Hence Nano silver gel prove to be more effective in the management of diabetic foot ulcers.

Although both antimicrobials were ultimately comparable over a 6-7 week period, these findings suggest that when wanting to achieve an immediate reduction in wound size and when attending clients with large and

chronic leg ulcers or a history of protracted healing, a silver antimicrobial can be considered for the first choice.

This study is done to know the efficacy of Nano crystalline silver and other conventional dressings. The performance of each of the two groups was comparable in terms of overall healing rate and the number of wounds healed. However, use of silver compounds was associated with a quicker healing rate during the first 2 weeks of treatment and in wounds that were larger, older, and had more exudate. This trial provides some insights as to circumstances in which one product may be preferred over the other.

CONCLUSION: Nano silver dressings are safe, effective, with a slight beneficial edge to the conventional dressing with solutions like betadine in terms of promoting wound healing, and are more patient compliant in view of.

1. Less pain while changing the dressing.
2. Less number of dressings required.
3. Less duration of hospital stay.

The above results indicate that Nano silver dressings may be used as an adjunct in management of diabetic foot ulcer and seems to be more efficient than conventional dressings in this regard.

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Classification	Superficial Ulcer or cellulitis	Deep soft tissue or bone involved	Tissue necrosis or gangrene	Systemic toxicity or Metabolic instability
Mild	+	-	-	-
Moderate	+	+ -(no gas/fasciitis)	+ -(minimal)	-
Severe	+	±	±	+

Age in Years	Test group 1		Test group 2	
	No.	%	No.	%
21-30	1	2	1	2
31-40	4	8	4	8
41-50	15	30	15	30
51-60	22	44	15	30
61-70	8	16	7	14
71-80	0	0	8	16
>80	0	0	0	0
Total	**50**	**100**	**50**	**100**
Mean±SD	52.73±9.880		55.50±12.113	

Table 1: Age distribution

Percentage reduction in the ulcer size was measured at the time of discharge using Ulcer Planometry. Number of dressings required for each group, Number of days of hospital stay were recorded. Patients are reevaluated on the outpatient basis for 2 months.

Sex Distribution	Test Group 1		Test Group 2	
	No.	%	No.	%
Male	31	62	34	68
Female	19	38	16	32
Total	**50**	**100**	**50**	**100**

Table 2: Sex Distribution

Total number of males in the study was 65(65%) and females were 35(35%). The male and female ratio of the test group no 1 is 62%: 38% and the test group 2 is 68%:32%. Hence Sex distribution is statistically similar between the two groups with P=0.737.

Sl. No.	Type of Onset	Test Group 1		Test Group 2	
		No.	%	No.	%
1	Traumatic	35	70	34	68
2	Spontaneous	15	30	16	32
	Total	**50**	**100**	**50**	**100**

Table 3: Onset of Diabetic foot Ulcer

Total no. of patients with ulcer due to traumatic in onset were 69(69%) and spontaneous in onset were 31(31%). Trauma is most common cause of diabetic foot ulcer. Trauma is trivial in nature in most of the cases. Distribution of foot ulcer was similar between two groups with P=0.213.

Sl. No	Site	Test group 1		Test Group 2	
		No.	%	No.	%
1	Plantar	34	68	35	70
2	Dorsum	16	32	15	30
	Total	50	100	50	100

Table 4: Site of Ulcer

Total no. of patients with ulcers on plantar aspect of foot is 69(69%) and on dorsum are 31(31%). Diabetic foot ulcers are more common in plantar aspect of foot due to increase of foot pressure.

FBS Levels	Test Group 1		Test Group 2	
	No.	%	No.	%
<100	0	0	0	0
101-200	42	84	39	78
201-300	8	16	11	22
301-400	0	0	0	0
Total	**50**	**100**	**50**	**100**

Table 5: Fasting Blood Sugar Levels

Mean±SD of Test group 1 is 171±31.837 and test group 2 is 179±25.265 and are statistically similar.

PPBS Levels	Test Group 1		Test Group 2	
	No.	%	No.	%
<100	0	0	0	0
101-200	18	36	9	18
201-300	32	64	39	78
301-400	0	0	2	4
Total	**50**	**100**	**50**	**100**

Table 6: Post prandial Blood Sugar Levels

Mean±SD of Test group 1 is 228.54±39.928 and test group 2 is 239.87±47.032 and are statistically similar.

Period of Stay(Weeks)	Nano silver Dressings		Conventional Dressings	
	No.	%	No.	%
1 to 2	12	24	0	0
2 to 3	17	34	0	0
3 to 4	21	44	0	0
4 to 5	0	0	13	26
>5 weeks	0	0	37	74
Total	**50**	**100**	**50**	**100**

Table 7: Average Duration of Hospital Stay

Mean±SD is 20.54±7.044 days for test group 1 and for test group 2 Mean±SD is 36.19±7.246 days and P<0.001 and is highly significant. In test group 1, 44% of patients stay for 3-4 weeks whereas in test group 2, 74% of patients stay for 5-6 weeks.

Percentage reduction of ulcer size	Test group 1		Test group 2	
	No.	%	No.	%
61-70	0	0	2	4
71-80	0	0	15	30
81-90	5	10	23	46
91-99	45	90	10	20
Total	**50**	**100**	**50**	**100**

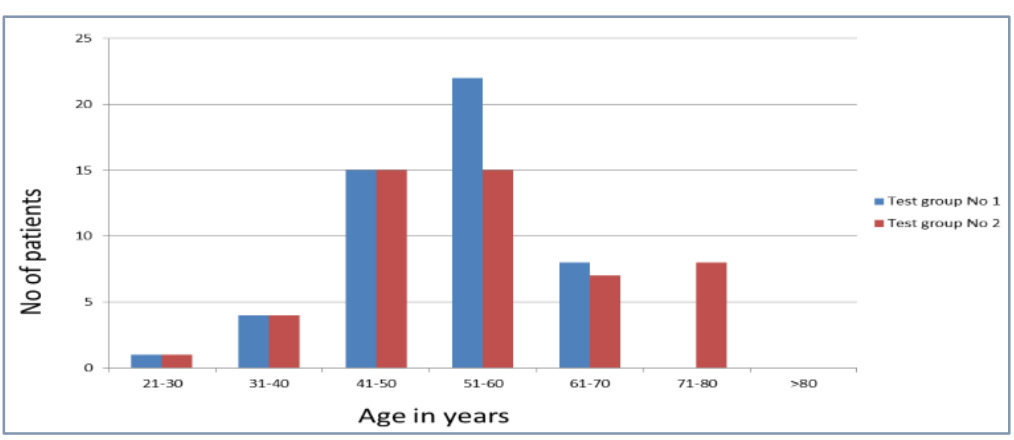
Table 8: Percentage reduction of ulcer size

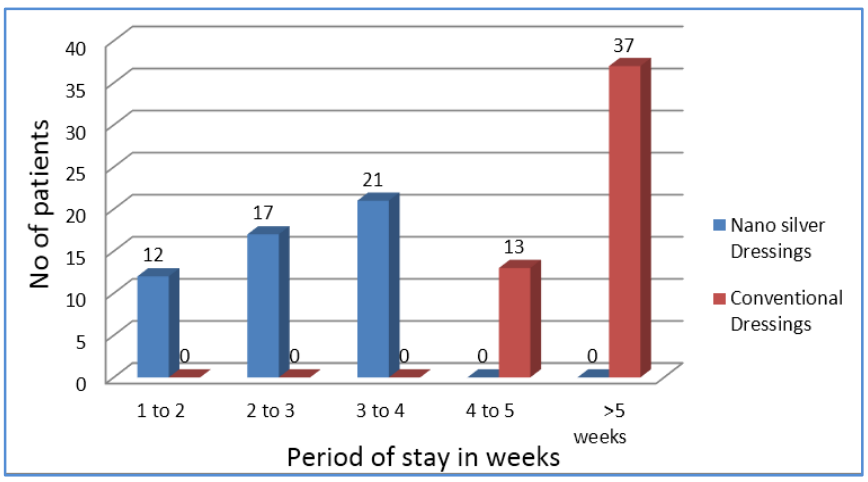
Mean±SD of test group1 and test group 2 are 94.81±3.971 and 84.69±8.449 and P< 0.001 and is highly significant. 91-99% reduction in size of ulcer is seen in 45 out of 50 patients in test group 1 whereas in test group 2 only 10 out of 50 shows 91-99% reduction in size.

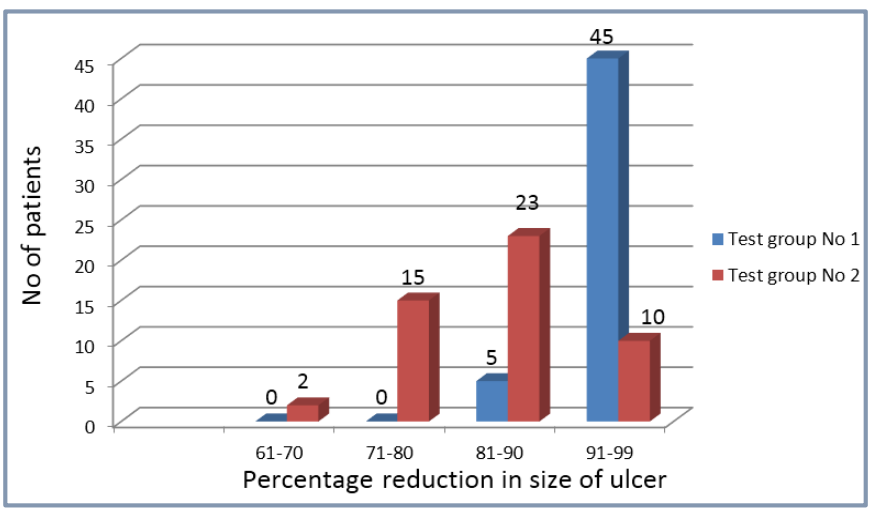
No. of Dressings	Test Group 1		Test Group 2	
	No.	%	No.	%
1-5	1	2	0	0
6-10	27	54	0	0
11-15	20	40	0	0
16-20	2	4	20	40
21-25	0	0	23	46
26-30	0	0	7	14
Total	**50**	**100**	**50**	**100**

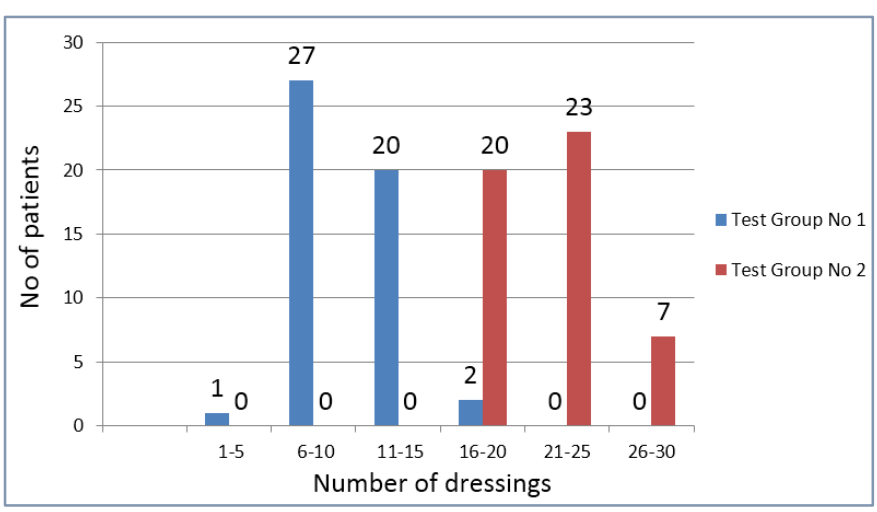
Table 9: No. of Dressings Required

Mean±SD of Test group1and test group 2 are 10.92±3.052 and 17.54±6.008 and P<0.001.




No of patients
40
35
30
25
20
15
10
5
0
12
0
17
0
21
0
0
13
0
37
1 to 2
2 to 3
3 to 4
4 to 5
>5 weeks
Period of stay in weeks
Nano silver Dressings
Conventional Dressings


No of patients
45
40
35
30
25
20
15
10
5
0
0
2
0
15
5
23
45
10
61-70
71-80
81-90
91-99
Percentage reduction in size of ulcer
Test group No 1
Test group No 2


No of patients
30
25
20
15
10
5
0
1
0
27
0
20
0
2
20
0
23
0
7
1-5
6-10
11-15
16-20
21-25
26-30
Number of dressings
Test Group No 1
Test Group No 2