"A ONE YEAR RANDOMISED CONTROLLED TRIAL TO COMPARE THE EFFECTIVENESS OF HONEY DRESSING VS. POVIDONE IODINE DRESSING FOR DIABETIC FOOT ULCER AT DR PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI"

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This is to certify that the dissertation entitled **"A ONE YEAR RANDOMISED CONTROLLED TRIAL TO COMPARE THE EFFECTIVENESS OF HONEY DRESSING VS. POVIDONE IODINE DRESSING FOR DIABETIC FOOT ULCER AT DR PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI"** is a bonafide research work done by **REG NO. BH0116009**

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LIST OF ABBREVIATIONS USED

A	-	Absent
Ant.	-	Anterior
Post.	-	Posterior
BF	-	Bacteroides fragilis
CVD	-	Cardiovascular disease
E. coli	-	Escherichia coli
F	-	Female
g - t	-	Granulation tissue
H.W	-	House wife
Lt foot	-	Left foot
M	-	Male
M.S	-	Minimal slough
N	-	Normal
N.A	-	Not applicable
No.	-	Number
NWD	-	Not well defined
O-H-A	-	Oral hypoglycaemic agent
P	-	Present
Rt foot	-	Right foot
S	-	Spontaneous
S. Aureus	-	Staph aureus
SA	-	Spinal anaesthesia
S	-	Slough tissue
T	-	Traumatic

TE	-	Tendon exposed
WD	-	Well defined
NS	-	Not significant
S	-	Significant

ABSTRACT

Background and objectives

The increasing prevalence of diabetes has resulted in concomitant illnesses. The critical effects of hyperglycemia include micro-vascular complications (nephropathy, neuropathy and retinopathy) and macro-vascular complications (coronary artery disease, stroke and peripheral arterial disease). Diabetes is a leading cause of non-traumatic lower extremity amputation, which is often preceded by a non-healing ulcer. Dressing plays a major role in healing of wounds in combination with debridment. This study compared the effectiveness of Honey dressing with povidone iodine dressing of grade 2 diabetic foot ulcers. Honey is one such product which is a collection of nectar from many plants, which is processed by honey bees. Honey is well known for its high nutritional and medicinal value. Honey has potent antibacterial activity which is useful in preventing and removing wound infections. It has been used as a wound care product since decades, and its use as a wound healing agent was reported for treating, chronic leg ulcers, burns wounds from many years.

Objective of this study is to find out the effect of honey dressing when compared with povidone iodine dressing for reduction of wound size in diabetic foot ulcer.

Materials and methods

This one year randomized controlled trail was done in Department of General Surgery, KLES Dr.Prabhakar Kore hospital, Belagavi from January 2017 to December 2017. A total of 64 Diabetic Patients taking insulin or oral hyperglycaemic and suffering from with Wagners grade 1 and 2 diabetic foot ulcers which are not healed were enrolled. These patients were divided into two groups of 32 each that is

Group A (Patients in this group underwent dressing with Unprocessed Honey) and Group B ((Patients in this group underwent dressing with povidone iodine).

Results

The mean wound size in honey dressing was 23.16 cm^2 at baseline, 23.16 cm^2 at 1st day follow up, 23.16 cm^2 at 3rd day follow up, 19.38 cm^2 at 5th day follow up, 16.13 cm^2 at 7th day follow up, 12.44 cm^2 at 10th day follow up and the end of 15th day, it was 10.69 cm^2. The difference in the wound size in Honey dressing group at 1st day, 3rd day, at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05). The difference in the wound size in honey dressing group at 15th day follow up period with baseline value were statistically significant (P value <0.05). The mean wound size in povidone dressing was 23.03 cm^2 at base line, 22.94 cm^2 at 1st day follow up, 22.94 cm^2 at 3rd day follow up, 20.28 cm^2 at 5th day follow up, 17.06 cm^2 at 7th day follow up, 16.13 cm^2 at 10th day follow up and the end of 15th day, it was 15.06 cm^2. The difference in the wound size in Povidone Iodine at 1st day, 3rd day, at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05) and the difference in the wound size in honey dressing at 15th day follow up period was statistically signicicant (P value <0.05).

Conclusion

This study shows more favourable results with honey dressing as compared with povidone iodine dressing for reduction of wound size in diabetic foot ulcers.

Key words

Unprocessed Honey dressing, Diabetic foot ulcers, Povidone iodine dressing

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INTRODUCTION

Diabetes mellitus (DM) is chronic and progressive endocrine disorder that results in hyperglycemia. Globally, diabetes is considered one of the major health problems and there is increasing prevalence. The prevalence of diabetes worldwide was 2.8% and is estimated there may be increase 4.5% by 2030. At present, 200 million people worldwide are suffering from diabetes and predicted to increase up to 333 million by the end of 2025.[1-2]. Data of epidemiological studies have indicated that the worldwide incidence of DM has been increasing by 3–6% with an approximate prevalence of one in 400 by 18 years of age [3].

Diabetes is a growing challenge in India with estimated 8.7% diabetic population in the age group of 20 and 70 years. The rising prevalence of diabetes and other no communicable diseases is driven by a combination of factors - rapid urbanization, sedentary lifestyles, unhealthy diets, tobacco use, and increasing life expectancy[4].The global prevalence of type 2DM (T2DM) has shown rapid growth over the past few decades. It is estimated that there will be 30 - 40 million diabetic patients in India, China by 2025 [5-6]. According to the statistics of the International Diabetes Federation (IDF), two will develop diabetics and two will die of it in every 10 seconds [7]. Therefore, diabetes has become a serious health problem that causes a socioeconomic burden in many countries. Diabetic patients suffer from lower extremity complications which including neuropathy, arterial disease, ulcerations which may cause of diabetic foot infections[8-9]

Diabetic foot ulcers are one the reasons for major cause of morbidity and disability in diabetic patients. They are often the common cause for amputations when

they are associated with ischaemia or neuropathy[10]. 6.9% patients are affected with diabetics during their life time.[11] Ulcerations are the most common cause of amputations.[12] There was the resistant bacterial strains noted which hampers the healing moreover there was drug side effects and organ toxicity [13-15]

Peripheral sensory neuropathy is one of the major reasons for ulcer formation. The decrease in sensation allows trauma to go unnoticed. Ulcers developing in such areas have increased pressure commonly to heel or toes. Sensory loss, motor deficits and muscle weakness may result due to injury or damage to the nerve.[16] The neuropathy causes decrease in sensation of pain and temperature in the foot. This combination of motor and sensory loss causes a change in the mechanics of the foot causing for pressures ulcers. This increases the risk for ulcer formation.[17-18] The other major factor in foot ulcer development is peripheral arterial disease. This is caused by plaque buildup in the arteries which eventually decreases blood flow to the small vessels in the periphery of the feet. There is decrease in blood flow, wounds are not able to heal due to the lack of oxygen, nutrients, and white blood cells all of which are carried in the blood.[19] With the combination of peripheral neuropathy, change in foot mechanics causing an increase in trauma, and lack of blood flow to the lower extremity, diabetes patients are vulnerable to ulcers of the foot with an inability to heal these wounds in a usual time period.[20-22]

As of now there is no cure for diabetes so the goal is to treat the disease early by first changing patient's lifestyles by encouraging incorporation of a healthy diet, decreased sugar intake, and increased physical activity. After lifestyle changes the next step is to use oral medications to decrease blood sugars like Metformin, Sulfonyureas in addition to using injectable insulin.[23]Also, comorbidities such as

hyperlipidemia and hypertension are treated to avoid chronic complications. Ulcers are a common complication of diabetes, and their regular treatment includes debridement, irrigation, and application of some type of dressing including hydro gels, foams, iodine, absorbant polymers or skin replacements. These dressings help to keep the wound moist for autolytic breakdown and healing as well as provide an antibacterial component[24]

Proper treatment is necessary if not, the amputation of the affected bone becomes unavoidable[25]. Wound healing is a complex process involving skin repair after injury [26]. it is also a long process in which devitalized and dead cellular structures and tissue layers are replaced. Many treatment approaches have been adopted that includes the use of topical and systemic antibiotics and ointments[27]. Many recent advances in antimicrobial therapy has been done though, diabetic foot ulcers remain a serious problem. Many numerous topical and systemic agents have been used either alone or in combination for the eradication of infections, but many have been eliminated because of resistance. These agents may lead to complications including drug side effects, and organ-specific toxicity[28-30]. Diabetic wound infections caused by drug-resistant organisms are becoming more common and they have resistance to many commonly used antibiotics, that leads to increasing costs and morbidity[31-32]. With an increasing frequency of antibiotic-resistant pathogens, modern medicine directs attention to natural products with increased antimicrobial property for clinical practice. Unprocessed Honey is one such product which is a collection of nectar from many plants, which is processed by honey bees. Honey is well known for its high nutritional and medicinal value[33]. Honey has potent antibacterial activity which is useful in preventing and removing wound infections [34]. It has been used as a wound care product since decades, and its use as a wound healing agent was reported

for treating venous leg ulcers, chronic leg ulcers, burns from may years [35-41], burns. Unprocessed Honey has several natural substances that may contribute for antimicrobial activity including an low pH osmotic effect, and the production of hydrogen peroxide (H2O2) [42-45]. Many investigations have revealed that Unprocessed honey fights antibiotic-resistant strains of bacteria and helps preventing bacterial growth in spite of wounds been heavily infected[46-47]. Furthermore, as Unprocessed honey is a natural product, it does not induce microbial resistance, even if the honey is not able to kill the microbes[48].

OBJECTIVE

The objective of this study is to find out the effect of honey dressing when compared with povidone iodine dressing for reduction of wound size in diabetic foot ulcer.

REVIEW OF LITERATURE

Diabetes mellitus, is a group of diseases characterized by high blood glucose levels that result from defects in the body's ability to produce and/or use insulin. It is a condition primarily defined by the level of hyperglycaemia giving rise to risk of microvascular damage (retinopathy, nephropathy and neuropathy). It is associated with reduced life expectancy, significant morbidity due to specific diabetes related microvascular complications, increased risk of macrovascular complications (ischaemic heart disease, stroke and peripheral vascular disease), and diminished quality of life. Several pathogenetic processes are involved in the development of diabetes. These include processes, which destroy the beta cells of the pancreas with consequent insulin deficiency, and others that result in resistance to insulin action. The abnormalities of carbohydrate, fat and protein metabolism are due to deficient action of insulin on target tissues resulting from insensitivity or lack of insulin. Diabetes mellitus may present with characteristic symptoms such as thirst, polyuria, blurring of vision, and weight loss. In its most severe forms, ketoacidosis or a non–ketotic hyperosmolar state may develop and lead to stupor, coma and, in absence of effective treatment, death. Often symptoms are not severe, or may be absent, and consequently hyperglycaemia sufficient to cause pathological and functional changes may be present for a long time before the diagnosis is made. [49]

History

For 2,000 years diabetes has been recognized as a devastating and deadly disease. In the first century A.D. a Greek physician, Aretaeus, described the destructive nature of the affliction, which he named "diabetes" from the Greek word for "siphon". Physicians in ancient times, like Aretaeus, recognized the symptoms of diabetes but were powerless to treat it effectively. In the 17thcentury a London physician, Dr. Thomas Willis, determined whether his patients had diabetes or not by sampling their urine. If it had a sweet taste he would 8 diagnose them with diabetes mellitus-"honeyed" diabetes. This method of monitoring blood sugars went largely unchanged until the 20th century. Before the discovery of the insulin little could be done for patients suffering from diabetes. Low calorie diets prolonged their lives but left them weak and near starvation. But in 1921, doctors in Canada treated patients dying of diabetes with insulin and managed to drop high blood sugars to normal levels. Since then, medical breakthroughs have continued to prolong and ease the life of people with diabetes. In the ’50s, it was discovered that there were two types of diabetes: "insulin sensitive" (type I) and "insulin insensitive" (type II). Two thousand years have passed since Aretaeus spoke of diabetes as "the mysterious sickness". It has been a long and arduous process of discovery, as generations of physicians and scientists have added their collective knowledge to finding a cure. It was from this wealth of knowledge that the discovery of insulin emerged in a small laboratory in Canada. Since then, medical innovations have continued to make life easier for people with diabetes. In the 21st century, diabetes researchers continue to pave the road toward a cure. Today, it is unclear what shape the road will take; perhaps another dramatic discovery like insulin waits around the corner, or possibly researchers will have to be content with the slow grind of progress (Satley, 2008) [50-52]

BURDEN OF DISEASE

The prevalence of diabetes worldwide was 2.8% and is estimated there may be increase 4.5% by 2030. At present, 200 million people worldwide are suffering from diabetes is predicted to increase up to 333 million by the end of 2025. Data of epidemiological studies have indicated that the worldwide incidence of DM has been increasing by 3–6% with an approximate prevalence of one in 400 by 18 years of age [3].The global prevalence of type 2DM (T2DM) has shown rapid growth over the past few decades. It is estimated that there will be 30 - 40 million diabetic patients in India, China by 2025. [52]

Types of Diabetes [53-54]

- **Type 1 Diabetes Mellitus**
- **Type 2 Diabetes Mellitus**
- **Gestational Diabetes**
- **Other types:**
    - **LADA (Latent autoimmune diabetes of adults)**
    - **MODY (maturity-onset diabetes of youth)**
    - **Secondary Diabetes Mellitus**

Type 1 diabetes

Was previously called insulin-dependent diabetes mellitus (IDDM) or juvenile-onset diabetes.

Type 1 diabetes develops when the body's immune system destroys pancreatic beta cells, the only cells in the body that make the hormone insulin that regulates blood glucose.

This form of diabetes usually strikes children and young adults, although disease onset can occur at any age.

Type 1 diabetes may account for 5% to 10% of all diagnosed cases of diabetes.

Risk factors for type 1 diabetes may include autoimmune, genetic, and environmental factors.

Type 2 diabetes

Was previously called non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes.

Type 2 diabetes may account for about 90% to 95% of all diagnosed cases of diabetes.

It usually begins as insulin resistance, a disorder in which the cells do not use insulin properly. As the need for insulin rises, the pancreas gradually loses its ability to produce insulin.

Type 2 diabetes is associated with older age, obesity, family history of diabetes, history of gestational diabetes, impaired glucose metabolism, physical inactivity, and race/ethnicity.

African Americans, Hispanic/Latino Americans, American Indians, and some Asian Americans and Native Hawaiians or Other Pacific Islanders are at particularly high risk for type 2 diabetes.

Type 2 diabetes is increasingly being diagnosed in children and adolescents.

Gestational diabetes

A form of glucose intolerance that is diagnosed in some women during pregnancy.

Gestational diabetes occurs more frequently among African Americans, Hispanic/Latino Americans, and American Indians. It is also more common among obese women and women with a family history of diabetes.

During pregnancy, gestational diabetes requires treatment to normalize maternal blood glucose levels to avoid complications in the infant.

After pregnancy, 5% to 10% of women with gestational diabetes are found to have type 2 diabetes.

Women who have had gestational diabetes have a 20% to 50% chance of developing diabetes in the next 5-10 years.

Secondary DM

Secondary causes of Diabetes mellitus include:

- Acromegaly,
- Cushing syndrome,
- Thyrotoxicosis,

- Pheochromocytoma
- Chronic pancreatitis,
- Cancer
- Drug induced hyperglycemia:

- Atypical Antipsychotics - Alter receptor binding characteristics, leading to increased insulin resistance.
- Beta-blockers - Inhibit insulin secretion.
- Calcium Channel Blockers - Inhibits secretion of insulin by interfering with cytosolic calcium release.
- Corticosteroids - Cause peripheral insulin resistance and gluconeogensis.
- Fluoroquinolones - Inhibits insulin secretion by blocking ATP sensitive potassium channels.
- Naicin - They cause increased insulin resistance due to increased free fatty acid mobilization.
- Phenothiazines - Inhibit insulin secretion.
- Protease Inhibitors - Inhibit the conversion of proinsulin to insulin.
- Thiazide Diuretics - Inhibit insulin secretion due to hypokalemia. They also cause increased insulin resistance due to increased free fatty acid mobilization.

PATHOPHYSIOLOGY [55-56]

An understanding of the pathophysiology of diabetes rests upon knowledge of the basics of carbohydrate metabolism and insulin action. Following the consumption of food, carbohydrates are broken down into glucose molecules in the gut. Glucose is absorbed into the bloodstream elevating blood glucose levels. This rise in glycemia stimulates the secretion of insulin from the beta cells of the pancreas. Insulin is

needed by most cells to allow glucose entry. Insulin binds to specific cellular receptors and facilitates entry of glucose into the cell, which uses the glucose for energy. The increased insulin secretion from the pancreas and the subsequent cellular utilization of glucose results in lowering of blood glucose levels. Lower glucose levels then result in decreased insulin secretion. If insulin production and secretion are altered by disease, blood glucose dynamics will also change. If insulin production is decreased, glucose entry into cells will be inhibited, resulting in hyperglycaemia. The same effect will be seen if insulin is secreted 9 from the pancreas but is not used properly by target cells. If insulin secretion is increased, blood glucose levels may become very low (hypoglycemia) as large amounts of glucose enter tissue cells and little remains in the bloodstream. Multiple hormones may affect glycemia. Insulin is the only hormone that lowers blood glucose levels. The counter-regulatory hormones such as glucagon, catecholamine's, growth hormone, thyroid hormone, and glucocorticoids all act to increase blood glucose levels, in addition to their other effects.

DIAGNOSIS [57-58]

Table 1.2.3 WHO diagnostic criteria of diabetes and intermediate hyperglycemia	
Diabetes mellitus	
Fasting plasma glucose	≥7.0mmol/l (126mg/dl)
or	
2–h plasma glucose*	≥11.1mmol/l (200mg/dl)
Impaired Glucose Tolerance (IGT)	
Fasting plasma glucose	< 7.0mmol/l (126mg/dl)
and	
2–h plasma glucose*	≥7.8 and <11.1mmol/l (140mg/dl to 200mg/dl)
Impaired Fasting Glucose (IFG)	
Fasting plasma glucose	6.1 to 6.9mmol/l (110mg/dl to 125mg/dl)
and (if measured)	
2–h plasma glucose*	<7.8mmol/l (140mg/dl)

Targets of diabetes care measures/tests	
Test/ measure	**Target value**
Blood pressure, mmHg	<130/80
Body mass index, Kg/m2	<25
HbA1c, %	<6.5
Total cholesterol, mg/dl	<200
LDL cholesterol, mg/dl	<100
Serum creatinine	<1.09

Management of DM [59-61]

- A - **Diet and Exercise**
- B - **Oral hypoglycaemic therapy**
- C - **Insulin Therapy**

A. Diet

Diet is a basic part of management in every case. Treatment cannot be effective unless adequate attention is given to ensuring appropriate nutrition.

Dietary treatment should aim at:

- Ensuring weight control
- Providing nutritional requirements
- Allowing good glycaemic control with blood glucose levels as close to normal as possible
- Correcting any associated blood lipid abnormalities
- **The following principles are recommended as dietary guidelines for people with diabetes:**
- Dietary fat should provide *25-35% of total intake of calories but saturated fat intake* should not exceed 10% of total energy. Cholesterol consumption should be restricted and limited to 300 mg or less daily.
- Protein intake can range between 10-15% total energy (0.8-1 g/kg of desirable body weight). Requirements increase for children and during pregnancy. Protein should be derived from both animal and vegetable sources.
- Carbohydrates provide *50-60% of total caloric content of the diet.* Carbohydrates should be complex and high in fibre.
- Excessive salt intake is to be avoided. It should be particularly restricted in people with hypertension and those with nephropathy.

Exercise

Physical activity promotes weight reduction and improves insulin sensitivity, thus lowering blood glucose levels.

Together with dietary treatment, a programme of regular physical activity and exercise should be considered for each person. Such a programme must be tailored to the individual's health status and fitness.

People should, however, be educated about the potential risk of hypoglycaemia and how to avoid it.

Oral Hypoglycaemic Medications

Drug Class	Drug Name	Mechanism of action
Biguanides	Metformin	Inhibit glucose production by liver
Sulfonylureas	Glimepride Glipizide Glyburide	Increase insulin secretion by pancreatic beta cells
Meglitinides	Repaglinide Nateglinide	Increase insulin secretion by pancreatic beta cells
Thiazolidinediones	Pioglitazone Rosiglitazone	Increase glucose uptake by skeletal muscle
Alpha-glucosidase inhibitors	Acarbose Miglitol	Inhibit carbohydrate absorption in small intestine

B. Insulin Therapy

Short-term use:

- Acute illness, surgery, stress and emergencies
- Pregnancy
- Breast-feeding
- Insulin may be used as initial therapy in type 2 diabetes
- in marked hyperglycaemia
- Severe metabolic decomposition (diabetic ketoacidosis, hyperosmolar nonketotic coma, lactic acidosis, severe hypertriglyceridaemia)

Long-term use:

- If targets have not been reached after optimal dose of combination therapy or BIDS, consider change to multi-dose insulin therapy. When initiating this, insulin secretagogues should be stopped and insulin sensitisers e.g. Metformin or TZDs, can be continued.

  The majority of patients will require more than one daily injection if good glycaemic control is to be achieved. However, a once-daily injection of an intermediate acting preparation may **be effectively used in some patients.**

  Twice-daily mixtures of short- and intermediate-acting insulin is a commonly used regimen.

In some cases, a mixture of short- and intermediate-acting insulin may be given in the morning. Further doses of short-acting insulin are given before lunch and the

evening meal and an evening dose of intermediate-acting insulin is given at bedtime. Other regimens based on the same principles may be used.

A regimen of multiple injections of short-acting insulin before the main meals, with an appropriate dose of an intermediate-acting insulin given at bedtime, may be used, particularly when strict glycaemic control is mandatory.

Self-Care

- Patients should be educated to practice self-care. This allows the patient to assume responsibility and control of his / her own diabetes management. Self-care should include:
    - Blood glucose monitoring
    - Body weight monitoring
    - Foot-care
    - Personal hygiene
    - Healthy lifestyle/diet or physical activity
    - Identify targets for control
    - Stopping smoking

COMPLICATIONS [62-64]

Complications	Prevalance (%)
Hypertension	61
Dyslipidemia	89
Macrovascular complication	28.5
Retinopathy	36
Nephropathy	26
Neuropathy	44.5
Foot ulcers	4

DIABETIC FOOT ULCER

ANATOMY OF FOOT [50-66]

The foot is an extremely complex anatomic structure made up of 26 bones and 33 joints that must work together with 19 muscles and 107 ligaments to execute highly precise movements. At the same time the foot must be strong to support more than 100,000 pounds of pressure for every mile walked. Even small changes in the foot can unexpectedly undermine its structural integrity and cause pain with every step.

The feet are flexible structures of bones, joints, muscles, and soft tissues that let us stand upright and perform activities like walking, running, and jumping. The feet are divided into three sections:

- The forefoot contains the five toes (phalanges) and the five longer bones (metatarsals).

- The midfoot is a pyramid-like collection of bones that form the arches of the feet. These include the three cuneiform bones, the cuboid bone, and the navicular bone.

- The hindfoot forms the heel and ankle. The talus bone supports the leg bones (tibia and fibula), forming the ankle. The calcaneus (heel bone) is the largest bone in the foot.

Muscles, tendons, and ligaments run along the surfaces of the feet, allowing the complex movements needed for motion and balance. The Achilles tendon connects the heel to the calf muscle and is essential for running, jumping, and standing on the toes.

Muscles

Dorsal aspect

Extensor Digitorum Brevis

Extensor Hallucis Brevis

Ventral aspect

First Layer-	**Abductor Hallucis**
	Flexor Digitorium Brevis
	Abductor Digiti minimi
Second Layer-	Quadratur Plantae
	4 Lumbricals
Third Layer-	Flexor Hallucis Brevis
	Adductor Hallucis
Fourth Layer-	Plantar Interossei
	Dorsal Interossei

Arterial Supply

- Dorsalis Pedis artery
- Anterior Tibial artery
- Posterior Tibial artery
- Peroneal artery

Nerve Supply

- Deep peroneal nerve.
- Superficial peroneal nerve.
- Tibial nerve - Medial plantar nerve; Lateral plantar nerve.

Arches of Foot

1. Medial longitudinal arch
2. Lateral longitudinal arch
3. Transverse arch

Functions

1. They functions as a shock absorbers in stepping and jumping.
2. They distribute body weight to the weight bearing areas of the sole mainly heel and the base of the toes.
3. They act as a springs mainly the medial longitudinal arch which helps in walking and running.

Movements

Ankle Joint	=	Dorsiflexion, Plantar flexion
Mid Tarsal Joint	=	Adduction, Abduction
Sub Talar Joint	=	Inversion, Eversion
Metatarsophalangeal Joint		Flexion, Extension

DIABETIC FOOT ULCERS [67-68]

Definition- Infection, ulceration or destruction of deep tissues associated with neurological abnormalities & various degrees of peripheral vascular diseases in the lower limb.

- 40% - 60% of all non traumatic lower limb amputation
- 85% of diabetic related foot amputation are preceded by foot ulcer
- 4 out of 5 ulcer in diabetics are precipitated by trauma
- 4% -10% is the prevalence of foot ulcer in diabetics

Risk Factors

- Diabetics for more than 10 years
- Peripheral Neuropathy
- Peripheral Vascular Disease
- Anatomical foot deformity
- Hyperglycaemia
- Trauma
- Smoking, Tobacco consumption
- Previous history of Amputation
- Previous history of foot ulcer

Common Sites

- Plantar surface of foot (metatarsal heads and midfoot)
- Toes (dorsal interphalangeal joints or distal tip).

Pathology

It can be due to 1) Neuropathy

2) Vasculopathy

3) Immune Dysfunction

Sensory Neuropathy

Loss of pain sensation

⬇

Unnoticed trauma (thermal, mechanical or chemical)

⬇

Progression of lesion

⬇

Callous formation

⬇

Tissue necrosis and damage

⬇

Development of cavities filled with serous fluid

⬇

Erupts into surface

⬇

Ulcer formation

Motor Neuropathy

Weakness and Wasting of foot muscles

⬇

Foot deformities

⬇

Abnormal gait

⬇

Ulceration

Autonomic Neuropathy

Decreased sweating

⬇

Dry and Brittle skin

⬇

Fissures/Cracks

⬇

Secondary Infection

⬇

Ulceration

VASCULOPATHY [69]

1) Macroangiopathy- Atherosclerosis of large arteries
2) Microangiopathy- Increased thickness of basement membrane and endothelial proliferation, there is capillary damage

Decrease blood supply

⬇

Poor antibiotic penetration

⬇

Poor wound healing

Characteristics

Characteristics	Neuropathic foot	Ischaemic foot
Skin temperature	Warm	Cold
Pain	Painless	Pinful
Color	Not altered	Dependent rubor
Ulcer	Tip of the toes	Margins of foot
Pulses	Bounding	Feeble or Absent
ABI	More than 0.9	Less than 0.9
Complications	Charcots joints	Critical Ischaemia

MANAGEMENT [55- 71]

Evaluation

- Etiological assessment: Neuropathic, ischemic, or neuro-ischemic.
- Characteristics of Ulcer: Size, depth, appearance, discharge and location.
- Physical examination- Signs of inflammation, Skin and Nails of foot
- Screening for Musculoskeletal Status
- Screening for neuropathy- Check for Pain, Vibration, Touch, Temperature Sensations
- Screening for Vascular Status- Pulsations, Color changes, Signs of ischaemia, prescence of edema
- Biochemical Investigations-CBC, RBS, HbA1c,
- Color Doppler for Vascular Pathology
- Discharge of Wound C&S
- Xray Foot for Osteomyelitis, Fractures
- MRI

TREATMENT

1) Metabolic Control
2) Microbiological Control
3) Mechanical Control
4) Vascular Managment
5) Education

Ulcer

⬇

Removal of Callus

⬇

Eradication of Infection (Proper Antibiotics)

⬇

Excess Keratin Should be removes from base of ulcer for formation of granulation tissue formation

⬇

Regular dressings

⬇

Foot Care

Danger signs which requires immediate attention

- Redness and swelling of foot
- Cellulitis,Discoloration,Crepitus
- A Pink Painless pulseless foot even without gangrene indictes critical limb ischaemia

Treatment Protocols

1. Bed Rest
2. Intravenous Antibiotics
    - It is necessary to provide wide spectrum of antibiotics cover
    - Thus Mixed therapy is necessary for overall coverage of organisms
        - Amoxicillin
        - Metronidazole (to cover Anaerobes)
        - Clinamycin / Genatmycin (to cover Gram Negative Organisms)
        - Antibiotics has to be decided once Culture report comes
3. An Intravenous Insulin pump may be needed to control blood glucose
4. Surgical Debridement
    - To drain out and abscess cavity
    - To remove all infected and necrotic tissue
    - To remove devitalized and infected bones resulting in osteomyelitis.
5. If necrosis has developed in digit Ray amputation is carried out.
6. Once proper granulation tissue forms Skin grafting is needed to accelerated wound healing
7. Joint and Wound should be immobilized for proper healing
8. Regular dressings should be carried Out in most effective manner and with Most effective Anti Microbial agents for proper and early healing of _Ulcer, One such agent is Raw Honey which we have used and conducted the study .

Prevention [70-72]

1. Screening
    - The foot must be examined routinely at onset of diabetics and annually thereafter.
    - Patient should be aware of foot care
    - Active lesions seen must be treated immediately after consulting doctor
    - Callus, Skin Cracks, Discoloration should be identified and treated.
    - Blood Sugar must be controlled.
2. Footwear
    - They are required for redistribution of weight bearing forces from vulnerable parts of foot.
    - Moulded soles made from substances or microcellular rubber are suitable for long term use.
    - Failure to wear special footwear is one of the reason of recurrence.
3. Follow up at Diabetic Foot Clinic.

Different types of dressings [73]

Types of Dressings		
Category	**Composition**	**Examples**
Nonadherent fabrics	Fine mesh gauze with non adherent and anti bacterial properties	Vaselien gauze, Xeroform, Telfa, Scarlet red
Gauze	Wide mesh gauze	Plain Gauze
Foams	Hydrophobic polyurethane sheets	Lyofoam,Allevyn,Vigifoam,Flexzan
Nonbiologic Films	Clear polyurethane membrane with acrylic adhesive on one side	Tegaderm, Mefilm,Carrafilm, Opsite, Biooclusive
Hydrocolloids	Matrix conataining gelatin, pectin, cellulose	Hydrocol, Hydroheal,Duoderm,Nuderm
Alginates	Cellulose like polysaccharide fibers	Algiderm,Algosternal,Seasorib, Sorbsan
Hydrogels	Polyuethylene oxide or cellulose	Vigilon, Nugel, Tegagel, Flexigel
Homograft	Genetically unique humans	Cadever skin
Xerograft	Interspecies eg- pig	Pigskin
Amnion	Human placenta	Placentrix
Antibacterial	Antibacterial properties	Mupirocin, Soframycin, Neomycin

COMPOSITION OF UNPROCESSED HONEY[74-93]

Carbohydrates80%-Fructose, Glucose, Sucrose, Diasaccharides, Maltose, Isomaltose

Proteins and Aminoacids

Vitamins-Vitamin B, C

Minerals- Calcium, Iron, ZincPotassium, Magnesium.Selenium, Mangenese

Antioxidants- Flavanoids,Ascorbic acid, Catalase

Acids-Acetic,Formic,Citric,Lactic,Gluconic,Malic Acids

PROPERTIES OF UNPROCESSED HONEY [74-93]

Antimicrobial Activity

Honey has both bactericidal and bacterostatic properties. This antimicrobial effects are due

- **Honey Acidity**

Honey has acidic PH 3.3 – 4.5, The acidity of honey is primarily caused by the presence of gluconic acid. It is formed through the action of a glucose oxidase enzyme produced by the bees. Honey acidity is considered to be one of the factors that contributes to its antimicrobial activity. In addition, because of its acidic nature, honey can prevent microbial biofilm formation and cross contamination. The acidity of honey creates an environment that facilitates the release of oxygen from the hemoglobin that is required for newly growing cells and the stimulation of white blood cells. It is possible to increase the oxygen release rate from hemoglobin by lowering the wound pH via honey application, thus increasing tissue granulation and improving the wound healing rate in diabetic patients.

- **Honey's Osmotic Effect**

Honey that contains <20%water is hyperosmolar. By being hyperosmolar, honey creates an unfavorable environment for the growth and survival of microorganisms [78]. High osmolarity substrates such as honey, glucose, and sugar pastes can inhibit microbial growth because water molecules are chemically tied to

the sugar molecules, thus creating a nonconducive environment for organism survival, leading to death. Sugar has been shown to accelerate wound healing in many patients with wounds, burns, and ulcers. The sugar content of honey is purportedly responsible for its antibacterial activity, which is contributed entirely by the osmotic effect. Hyperosmolar substances tend to draw fluid into the wound area to make a viscous solution, thus providing a protective layer against cross contamination Therefore, a highly osmolar solution, that is, honey, can be safely employed for diabetic wound treatments

- **Hydrogen Peroxide**

The hydrogen peroxide (H2O2) that is found in honey is steadily produced by oxidation from the glucose oxidase enzyme (which is secreted into nectar from the hypo pharyngeal gland of bees), and it is also a potent antibacterial agent. The H2O2 produced by honey is not cytotoxic because its H2O2 concentration is approximately 1000 times lower than that of the 3% solution commonly used as an antiseptic The low concentration of hydrogen peroxide may act as a "messenger" in healing promotion and may stimulate both fibroblasts and epithelial cells. H2O2 reportedly stimulates fibroblast proliferation.

- **Nitric Oxide**

Nitric oxide (NO) plays an important role in the immunological response, inflammatory response, cell movement, and killing mechanisms of bacteria and viruses and also supports different types of organ-related functions.NO is very active in the proliferative stages during wound healing in patients. Nitric oxide is able to reverse healing impairment in diabetic patients. NO end products are known to be

present in honey, and the concentration of these metabolites varies depending on the honey type. The increased production of NO products by honey in different body fluids improves wound healing and provides the antimicrobial and immune regulatory actions of NO. The NO present in honey and NO-derived end products could be other potent ingredients that could help patients recover from diabetic wounds.

Controlling Wound Odour

Honey has the potential ability to minimize offensive smelling wounds through its strong osmotic action, which draws exudates and lymph fluid from the wound out towards the surface to add the moisture needed for autolytic debridement. Honey can deodorize wound odor through two mechanisms. First, the presence of some anaerobic bacteria such as *Bacteroides* spp. *Peptostreptococcus* spp., and *Prevotella* spp. is documented to produce malodor. Second, wound odor is produced by the creation of amino acids through the decomposition of serum, tissue proteins, and dead cells by bacteria

Honey Minimizes Scar Formation

The free radicals formed by excessive or prolonged inflammation can stimulate fibroblasts to produce a hypertrophic scar made of collagen fibers. Hypertrophic scars can be difficult to counteract during wound healing, and they can be alleviated by honey. Honey stimulates epithelial cell growth at the skin level and produces soft, smooth, and regular scar surfaces in 80% of cases following complete healing. Topham (2002) reported three potential mechanisms behind scarless healing when honey is applied to wounds as follows: (1) the production of hyaluronic acid from glucose suppresses the formation of fiber-forming collagens; (2) attaching sugar

to collagen changes its structure and suppresses its activity and (3) glucose creates an environment in the wound area that directs wound healing proteoglycans to act without producing excessive amounts of collagens.

METHODOLOGY

This randomized controlled trial was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi over a period, from January 2017 to December 2017.

Study design

The study design was a randomized controlled trial.

Study period and duration

This study was carried out for the duration of one year from January 2017 to December 2017.

Place

This study was done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi a tertiary care teaching hospital attached to KLE University's Jawaharlal Nehru Medical College, Belagavi.

Source of Data

Diabetic Patients taking insulin or oral hyperglycaemic and suffering from diabetic foot ulcers which are not healed, over a period of > 3 weeks and for which debridement is required for healing Patients, were enrolled.

Sample size

The present study was comprised of 64 Patients taking insulin or oral hyperglycaemic and suffering from diabetic foot ulcers which are not healed, over a period of > 3 weeks and for which debridement is required for healing patients divided into two groups of 32 each.

- The patient will be randomly divided into 2 groups.
- First group (32 pt) with Unprocessed Honey dressing.
- Second group (32 pt) with Povidone iodine dressing.
- Informed consent will be obtained from all the patients.
- Wound dressing will be changed on alternate days for 6 weeks of follow up or till complete healing.
- If there is soakage or discharge dressing will be changed every day with water soluble Povidone Iodine and unprocessed honey
- Wound healing status to be monitored at 2 week interval
- Same antibiotic will be used for both the groups to compare the exact status of wound in both the groups, there should be no difference in wound status by using different antibiotics in both groups.

Assesment

- Ulcers are observed over a period of 15 days
- Ulcer area is measured on days 1, 3, 5, 7, 10,and 15.
- Ulcer area is measured using transparent graph paper
- Each box of graph paper is counted and area is given in mm^2 .

Selection criteria

Inclusion

1. Patient included in the study are those who are suffering from diabetic foot ulcers.
2. Ulcers which are not healed , over a period of more than 6 weeks and for which debridement is required for healing, only clinically clean wounds without signs of inflammation, purulent discharge
3. Patients with grade 1 and 2 according to Wagners classification.

WAGNER`S CLASSIFICATION

1. Grade 0: No ulcer in a high risk foot.
2. Grade 1: Superficial ulcer involving the full skin thickness but not underlying tissues.
3. Grade 2: Deep ulcer, penetrating down to ligaments and muscle, but no bone involvement or abscess formation.
4. Grade 3: Deep ulcer with cellulitis or abscess formation, often with osteomyelitis.
5. Grade 4: Localized gangrene.
6. Grade 5: Extensive gangrene involving the whole foot

Exclusion

1. Ischemic limb
2. Associated osteomyelitis
3. Cellulitis
4. Diabetic ketoacidosis
5. Exposed bone
6. Hb level less than 10gm%
7. Patient with known allergy to honey or povidone iodine.

Ethical clearance

The study was approved from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belagavi.

Informed Consent

The eligible patients who fulfilled the selection criteria were informed in detail about the nature of the study and a written informed consent was obtained.

Method of collection of data

The demographic data was obtained through an interview. Patients were asked for the past history, ulcer duration, diabetic history and treatment history. Further these patients were subjected to clinical examination. The wound observation was performed for ulcer characteristics such as site, size, shape, edge, margin, floor, base,

discharge, surrounding skin and slough / necrotic tissue. These findings were noted on a predesigned and pretested proforma.

Investigations

The patients underwent following investigations.

- Fasting blood sugar
- Complete blood count.
- HbA1c
- Renal Function test
- Urine R/M
- Wound discharge for C/S
- X-Ray foot – Antero-posterior and Lateral view (as and when required)
- Color Doppler (as and when required)

Randomization

The patients were divided into two groups of 32 each viz. Group A and group B based on closed envelope method as below

- First group (Group A) with Honey dressing.

  Second group (Group B) with Povidone Iodine dressing.

Treatment

All the patients underwent debridement of wound. Empirical antibiotics Ceftriaxone and Metronidazole were started and changed to sensitive antibiotics after culture and sensitivity report. The dressing was done as below.

Group A

Honey dressing was done.

Group B

Povidone Iodine dressing was done.

Dressings were done using same technique – cleaning and application of Honey/Povidone Iodine and putting a dressing.

- Prior to application, the lesion was cleaned of debris and digested material by gently rubbing with gauze pad by normal saline.
- Unprocessed Honey was applied on sterile gauze pad, which was then applied to the wound and properly secured.
- Povidone Iodine Soaked gauze was kept on the wound and dressing was secured.
- Wound dressing will be changed on alternate days for 6 weeks of follow up or till complete healing.
- If there is soakage or discharge dressing will be changed every day with Povidone Iodine and unprocessed honey
- Wound healing status to be monitored at different days within 2 weeks interval

- Same antibiotic will be used for both the groups to compare the exact status of wound in both the groups, there should be no difference in wound status by using different antibiotics in both groups.

Outcome variables

- Debridement of slough/nonviable tissue, reduction in ulcer size, granulation.
- Discharge, odour, induration noted for over all response to treatment
- Ulcer was assessed by the investigator at the beginning of the study. Ulcer mapping was made and size was recorded.
- Ulcers are observed over a period of 15 days
- Ulcer area is measured on days 1,3, 5, 7, 10,and 15.
- Ulcer area is measured using transparent graph paper
- Each box of graph paper is counted and area is given in mm^2.

Follow up

- The patients were evaluated at beginning of dressing, two weeks.

Statistical analysis

The data obtained was coded and entered in Microsoft Excel Spreadsheet. Study group (Honey dressing, Povidone iodine dressing) was considered as primary explanatory variable.

Wound size was considered as primary outcome parameter. Other Wound related parameters like discharge, appearance of granulation tissue and status of edges etc were considered as secondary outcome variables.

Age, medication duration (in years), wound size in cm2, hemoglobin (gm/dl), hba1c (%), blood urea (gm/dl), serum creatinine (gm/dl) were as considered as potential confounders.

All the quantitative variables were checked for normal distribution within each study group. Normally distributed quantitative variables were compared by mean and standard deviation using independent sample t-test.

Normality test using SPSS

A shapiro- wilk's test ($p>0.05$) [94-95] and a visual inspection of their histograms, normal Q-Q plots and box plots showed that the study group (Honey dressing, Povidone iodine dressing) and wound size different follow up time periods was non-normally distributed.[96-98]

Non-normally distributed quantitative variables were compared by median and Inter quartile range using Mann-Whitney U-test. The categorical variables were compared between two groups using cross tabulation and Chi square test/Fisher's exact test.

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.[99]

RESULTS

A total 64 people were include in the analysis.

Table 1: Descriptive analysis of group in study population (N=64)

Group	Frequency	Percentages
Honey dressing	32	50.00%
Povidone iodine dressing	32	50.00%

Among the study population, 32(50%) participants had honey dressing and remaining 32(50%) participants had povidone iodine dressing.(Table 1)

Table 2: Comparison of baseline quantitative parameters between two groups (N=64)

Parameter	Mean ±SD		P value
	Honey dressing (N=32)	Povidone iodine dressing (N=32)	
Age	62.84 ± 10.05	58.88 ± 12.78	0.172
Medication duration (in years)	6.13 ± 1.7	6.28 ± 1.51	0.699
Hemoglobin (gm/dl)	11.24 ± 1.14	10.74 ± 1.08	0.074
HBA1C (%)	7.75 ± 0.67	8 ± 0.72	0.156
Blood urea (gm/dl)	13 ± 3.40	11.66 ± 2.27	0.065
Serum creatinine (gm/dl)	0.956± 0.15	0.997±0.18	0.348

There was no statistically significant difference between two groups in other baseline parameters like age, medication duration, wound size in cm^2, hemoglobin (gm/dl), HBA1C (%) and serum creatinine (gm/dl) (P> 0.05).The mean blood urea honey dressing group was 13 ± 3.39gm/dl and the povidone iodine dressing group was 11.34 ± 2.22gm/dl, the differenceof blood urea (gm/dl) between two groups was statistically insignificant (Table 2& Figure 1, 2, 3, 4, 5, 6)

Figure 1: Error bar of comparison of age between two groups (N=64)

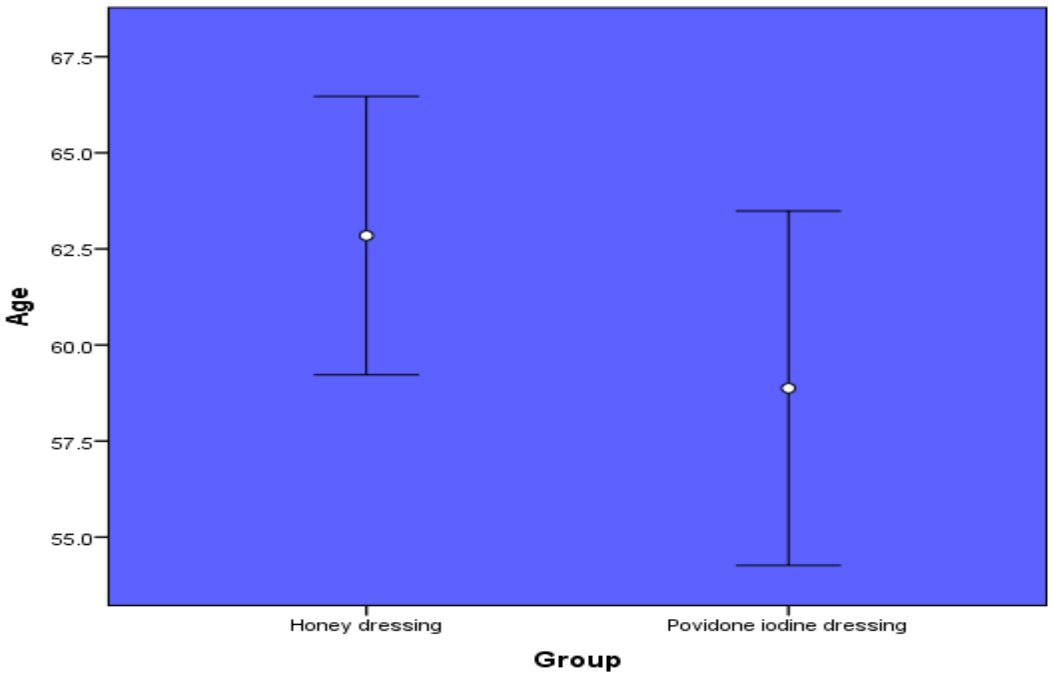


Figure 2: Error bar of comparison of medication duration (in years) between two groups (N=64)

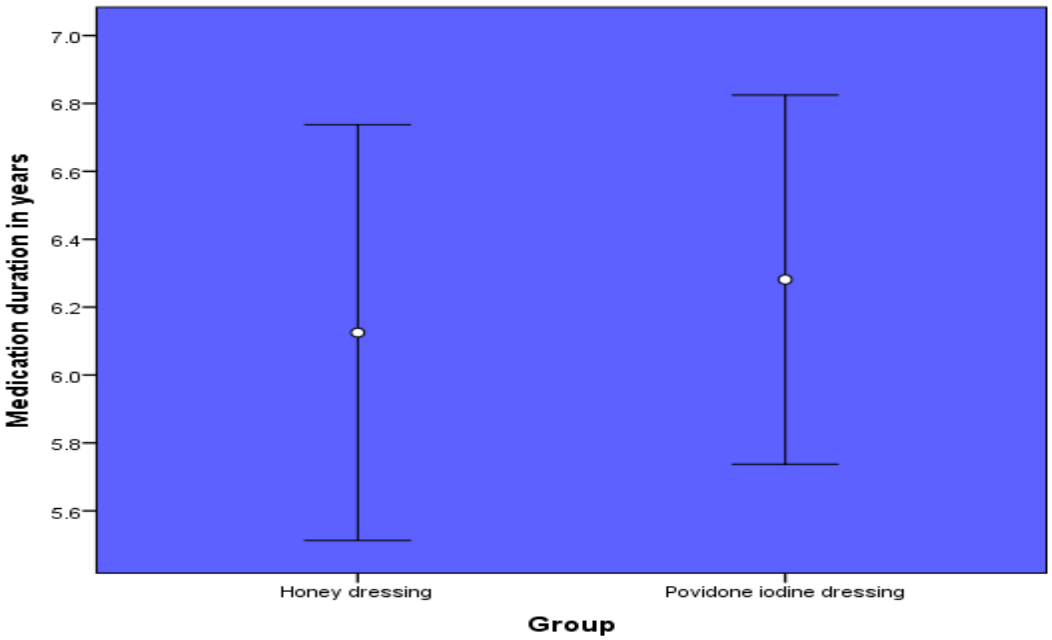


Figure 3:Graph of comparison of hemoglobin (gm/dl)between two groups (N=64)

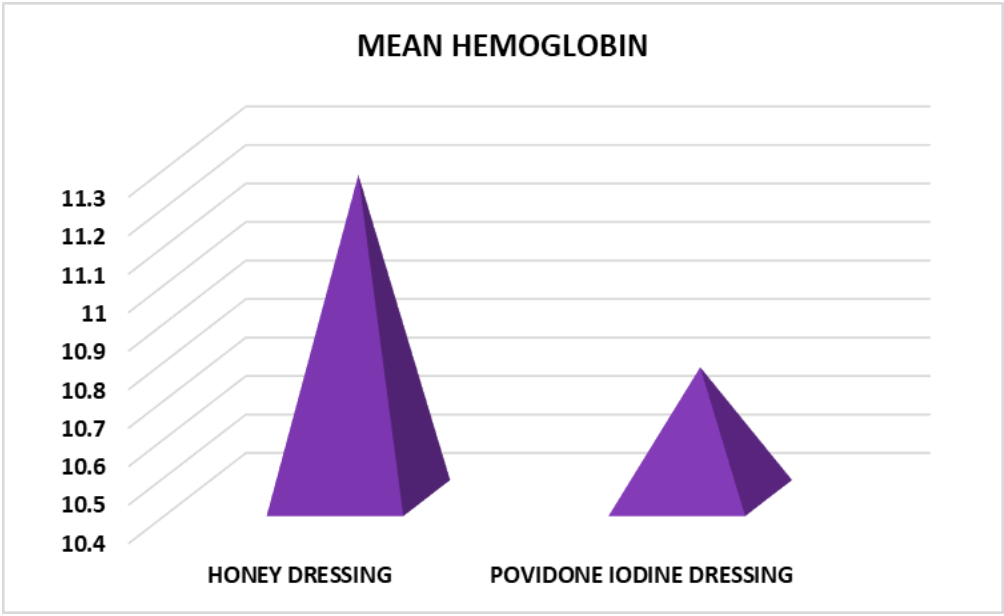


Figure 4: Graph of comparison of HBA1C (%) between two groups (N=64)

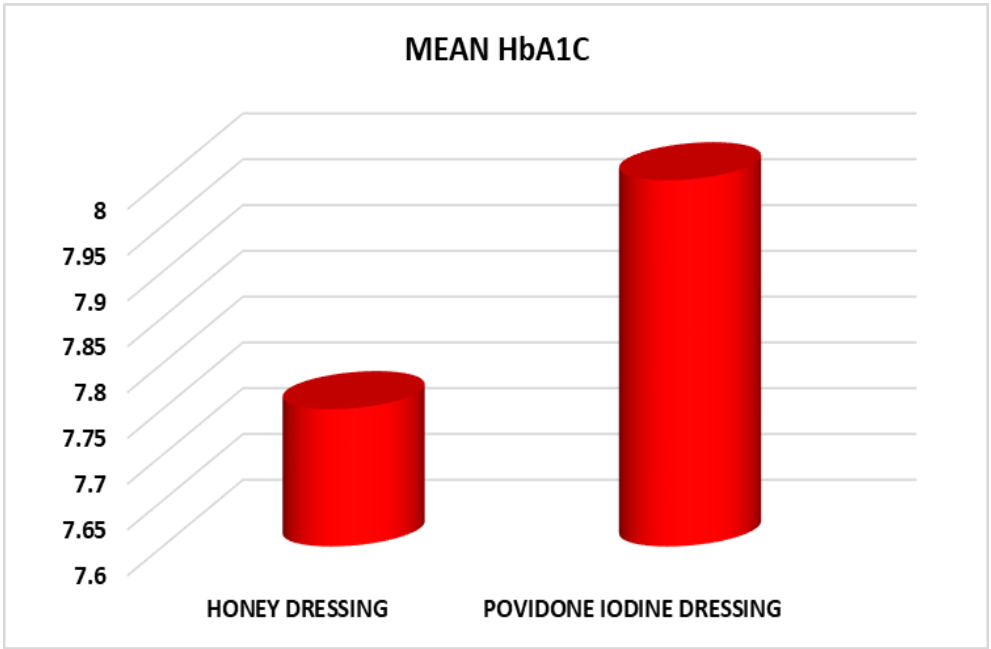


Figure 5: Graph of comparison of blood urea (gm/dl) between two groups (N=64)

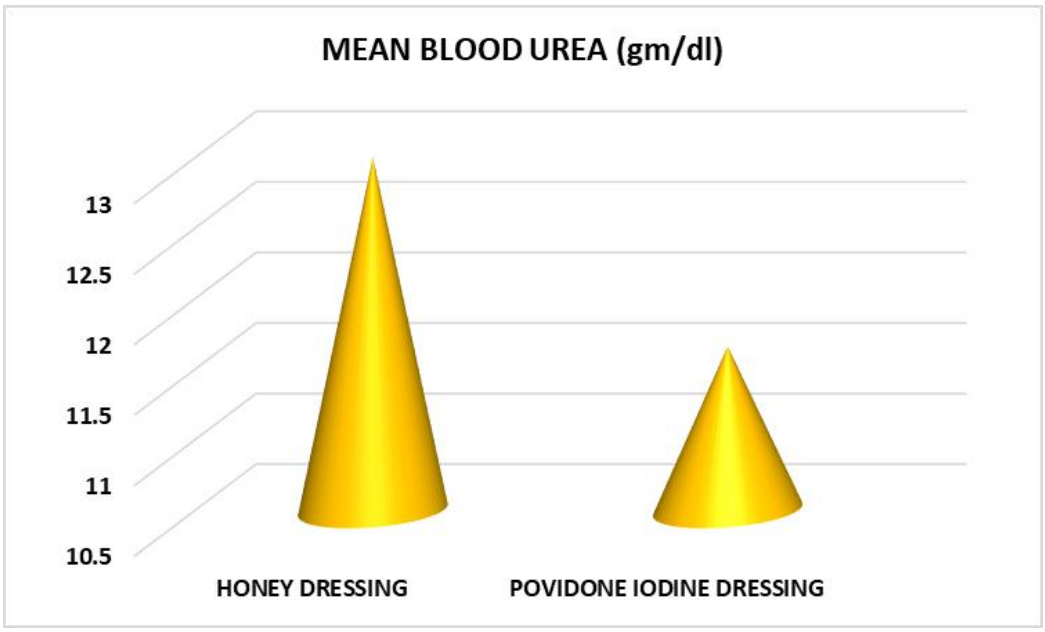


Figure 6: Chart of comparison of serum creatinine (gm/dl) between two groups (N=64)

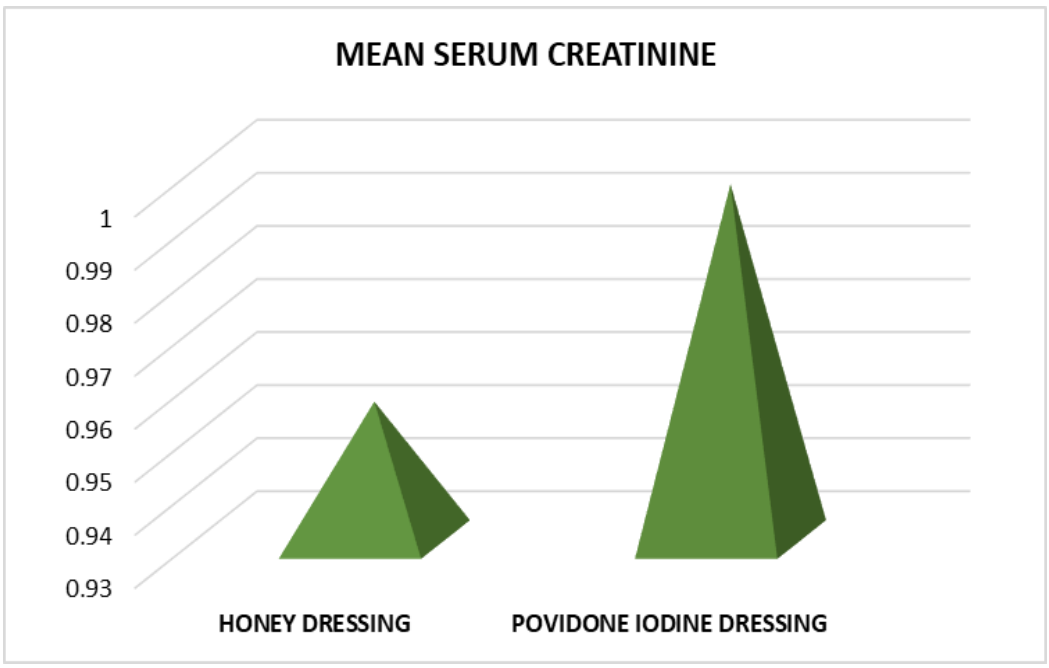


Table 3: Comparison of group with demographic parameter in the study population (N=64)

Demographic Parameters	Honey dressing(N=32)	Povidone iodine dressing(N=32)
Male	26 (81.3%)	24 (75%)
Female	6 (18.8%)	8 (25%)
Occupation		
Farmer	18 (56.3%)	15 (46.9%)
Worker	10 (31.3%)	14 (43.8%)
Bus driver	4 (12.5%)	3 (9.4%)

Among the honey dressing group,26 (81.3%) participants were male, remaining 6 (18.8%) participants were female. Among the povidone iodine dressing group,24 (75%)participants were male remaining 8 (25%)participants were female.Among the honey dressing group,18 (56.3%) participants were farmer, 10 (31.3%) participants were worker and 4 (12.5%) participants were bus driver. Among the povidone iodine dressing group,15 (46.9%) participants were farmer, 14 (43.8%) participants were worker and 3 (9.4%) participants were bus driver.

(Table 3& figure 7,8)

Figure 7: Bar chart of comparison of group with gender in the study population (N=64)

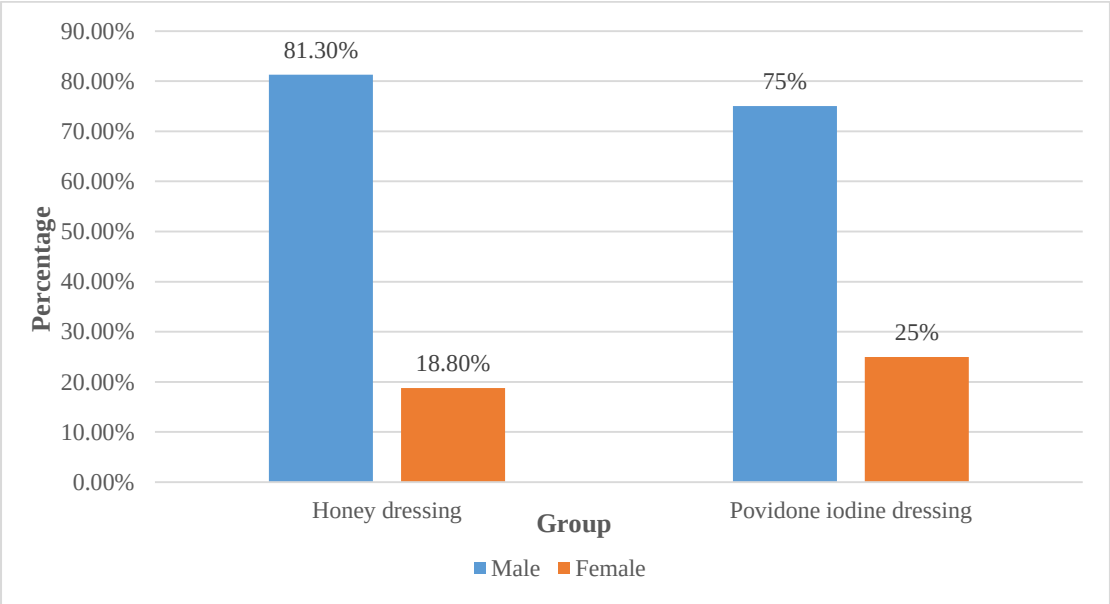


Figure 8: Bar chart of comparison of group with occupation in the study population (N=64)

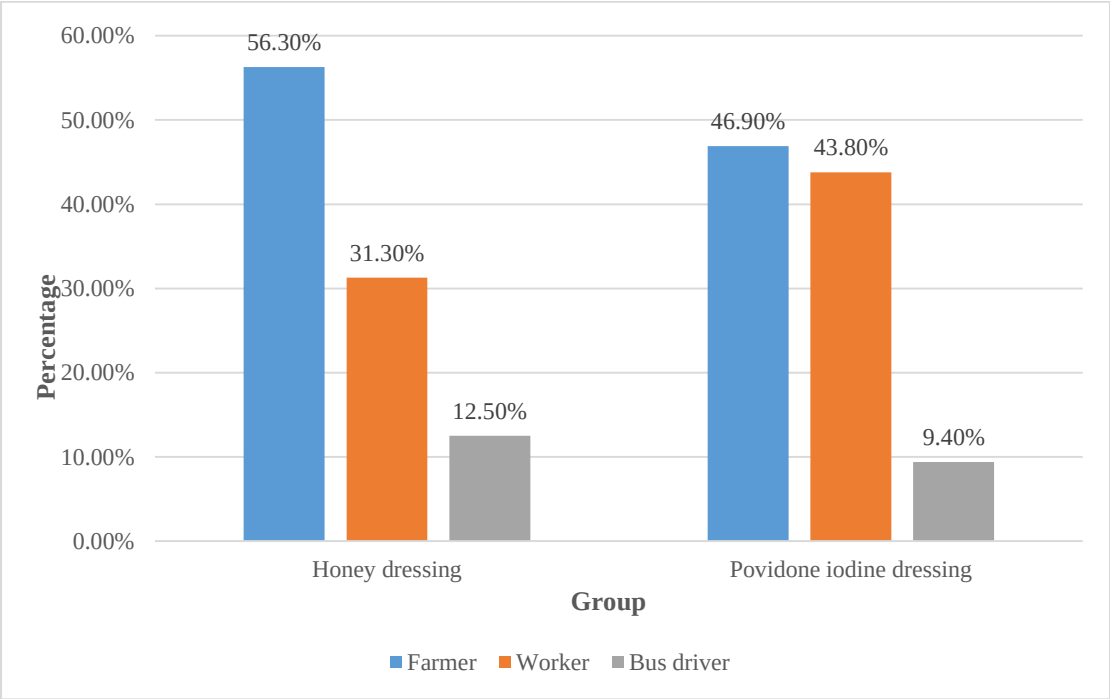


Table 4: Comparison of wound observation between two groups (N=64)

Wound Observation	Group		Chi square	P-value
	Honey dressing(N=32)	Povidone iodine dressing(N=32)		
Site				
			0.484	0.785
Left foot	20 (59.4%)	19 (53.1%)		
Right foot	12 (37.5%)	13 (40.6%)		
Shape				
Circular	19 (59.4%)	21 (65.6%)	0.282	0.869
Irregular	1 (3.1%)	1 (3.1%)		
Oval	12 (37.5%)	10 (31.3%)		
Edge				
Slopping	32 (100%)	32 (100%)	*	1.000
Margin				
Well defined	32 (100%)	32 (100%)	*	1.000
Floor				
Slough	32 (100%)	32 (100%)	*	1.000
Base				
Tendon	32 (100%)	32 (100%)	*	1.000
Discharge				
Serous	32 (100%)	32 (100%)	*	1.000
Surrounding skin				
Normal	32 (100%)	32 (100%)	*	1.000
Slough necrotic issue				
Slough tissue	32 (100%)	32 (100%)	*	1.000

**Chi square test not applicable.*

Among the honey dressing group,120 (59.4%) participants had left foot and 12 (37.5%) participants had right foot. Among the povidone iodine dressinggroup,19 (53.1%) participants had left foot and 13 (40.6%) participants had right foot. The difference in the proportion of sites between groups was statistically not significant (P value 0.785).The difference in the proportion of shap**es** between groups was statistically not significant (P value 0.869).(Table 4)

Table 5: Comparison of wound size in cm ^{2} within each group at different time follow up periods (N=64)

	WOUND SIZE					
DAYS	**HONEY DRESSING**		**POVIDONE IODINE DRESSING**		**p VALUE**	**INFERENCE**
	MEAN	**S.D.**	**MEAN**	**S.D.**		
BASE	23.16	10.94	23.03	11.57	0.9666	NS
DAY 1	23.16	10.94	22.94	11.52	0.9415	NS
DAY 3	23.16	10.94	22.94	11.52	0.9415	NS
DAY 5	19.38	8.14	20.28	10.51	0.7139	NS
DAY 7	16.13	7.37	17.06	10.22	0.6928	NS
DAY 10	12.44	6.16	16.13	9.88	0.0868	NS
DAY 15	10.69	5.13	15.06	8.97	0.0258	S

The mean wound size in honey dressing was 23.16 cm^2 at baseline, 23.16 cm^2 at 1st day follow up, 23.16 cm^2 at 3rd day follow up, 19.38 cm^2 at 5th day follow up, 16.13 cm^2 at 7th day follow up, 12.44cm^2 at 10th day follow up and the end of 15th day,it was 10.69 cm^2. The difference in the wound size in Honey dressing group at 1st day,3rd day,at 5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05). The difference in the wound size in honey dressing group at 15th day follow up period with baseline value were statistically significant (P value <0.05). The mean wound size in Povidone dressing was 23.03cm^2 at base line, 22.94cm^2 at 1st day follow up, 22.94cm^2 at 3rd day follow up, 20.28cm^2 at 5th day follow up, 17.06cm^2 at 7th day follow up, 16.13cm^2 at 10th day follow up and the end of 15th day, it was 15.06cm^2. The difference in the wound size in Povidone Iodine at1st day,3rd day,at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05)and the difference in the wound size

in honey dressing at 15th day follow up period was statistically significant (P value <0.05).(Table5& figure 9)

Figure 9: Trend line diagram of comparison of area of the ulcer (in cm^2) (N=64)

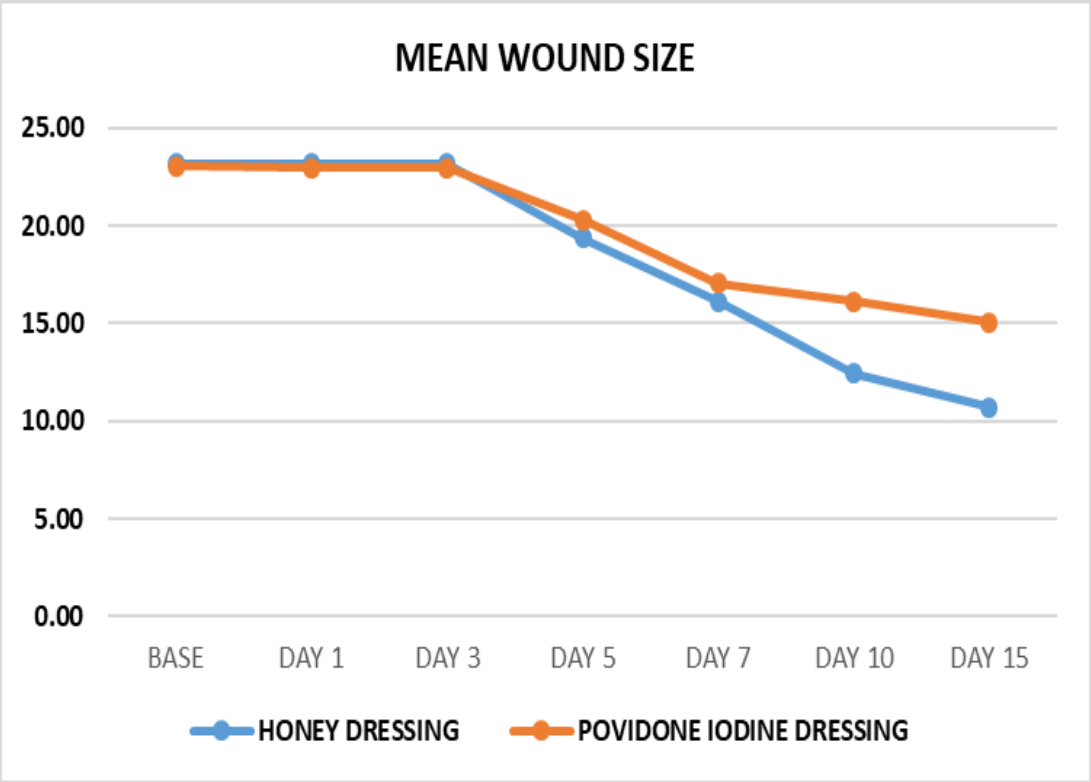
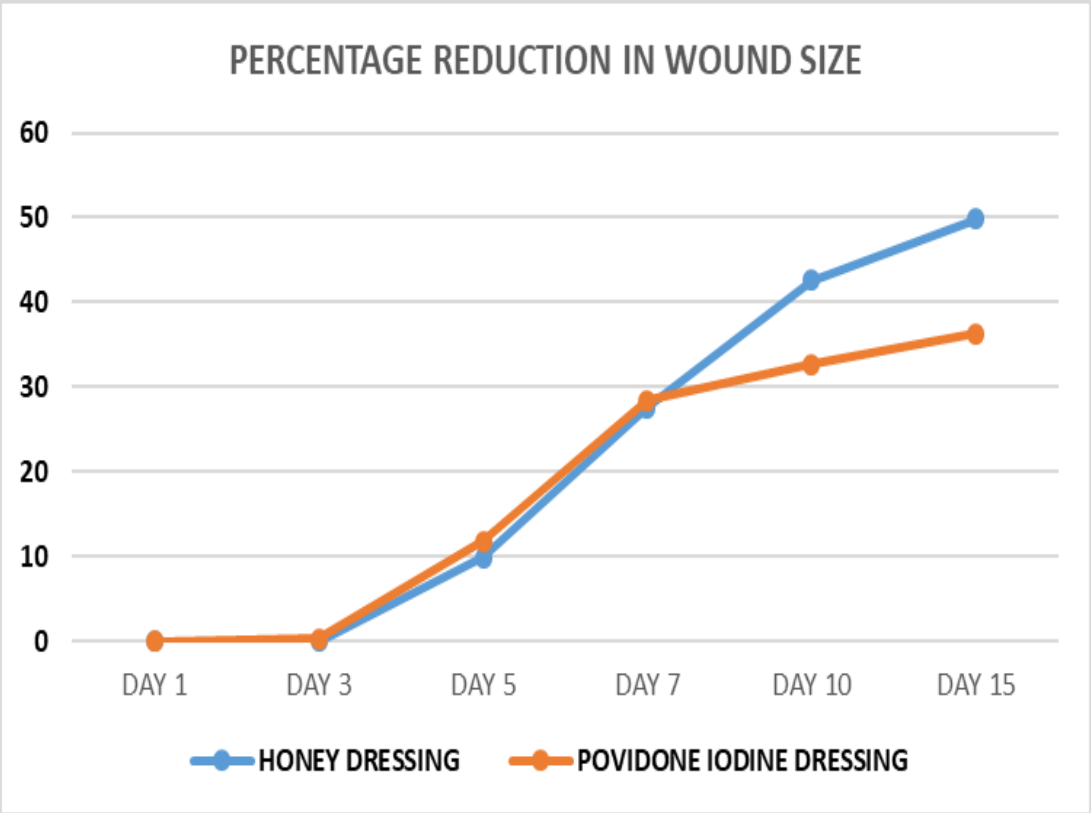


Table 6:Comparison of percentage reduction of wound size between each group at different time follow up periods (N=64)

	PERCENTAGE REDUCTION WITH RESPECT TO BASE LINE					
	HONEY DRESSING		POVIDONE IODINE DRESSING		p VALUE	INFERENCE
DAYS	MEAN	S.D.	MEAN	S.D.		
DAY 5	9.87	24.44	11.79	18.34	0.7201	NS
DAY 7	27.54	17.44	28.39	17.05	0.8429	NS
DAY 10	42.58	18.91	32.73	15.74	0.0255	S
DAY 15	49.87	20.24	36.30	14.05	0.0025	VS

The mean percentage reduction of wound size in both groups of day 1, 3 were almost same and stastically not significant, while in honey dressing group was 9.87 at 5th day follow up, 27.54 at 7th day follow up, 42.58 at 10th day follow up and the end of 15th day,it was 49.87. The difference in the wound size in Honey dressing group at 1st day,3rd day,at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05). The difference in the wound size in honey dressing group at10th and 15th day follow up period with baseline value were statistically significant (P value <0.05). The mean percentage reduction in wound size in Povidone iodine dressing group was 24.44 at 5th day follow up, 17.44 at 7th day follow up, 18.91 at 10th day follow up and the end of 15th day, it was 20.24. The difference in the wound size in Povidone Iodine at 1st day,3rd day,at5th day, 7th day, follow up period with baseline value were statistically not significant (P value >0.05).and the difference in the wound size in honey dressing at 10th and 15th day follow up period was statistically signicicant (P value <0.05).(Table6& figure 10)

Figure 10: Trend line diagram of Percentage reduction of wound size (N=64)



DISCUSSION

The increasing prevalence of diabetes has resulted in concomitant illness. The critical effects of hyperglycemia include micro-vascular complications (nephropathy, neuropathy and retinopathy) and macro-vascular complications (coronary artery disease, stroke and peripheral arterial disease). Diabetes is a leading cause of non-traumatic lower extremity amputation, which is often preceded by a non-healing ulcer. The lifetime risk of foot ulceration in people with diabetes is 15%-20%. More than 15% of foot ulcers result in amputation of the foot or limb. Several other population-based studies indicate a 0.5%-3% annual collective incidence of diabetic foot ulcers. The prevalence of foot ulcers reported varies from 2% to 10%. Approximately 45%-60% of all diabetic foot ulcerations are purely neuropathic, whereas 45% have both neuropathic and ischemic components. It has been estimated that around 15%-27% patients with diabetes require lower limb amputations predominantly (50%) due to infection.[93]

Infection, ulceration or destruction of deep tissues associated with neurological abnormalities and various degrees of peripheral vascular diseases in the lower limb (World Health Organization definition, 1995).

Dressing plays a major role in healing of wounds in combination with debridment. This study compared the effectiveness of Honey dressing with povidone iodine dressing of grade 2 diabetic foot ulcers. The present randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi over a period, from January 2017 to December 2017. A total of 64 Diabetic Patients taking insulin or oral hyperglycaemic

and suffering from diabetic foot ulcers which are not healed with Wagners grade 1 and 2 were enrolled. These patients were divided into two groups of 32 each that is Group A (Patients in this group underwent dressing with Honey) and Group B ((Patients in this group underwent dressing with povidone iodine).

In the present study male outnumbered females. That is majority of the patients in group A (81.3%) and group B were males (75%). However the sex distribution pattern in group A and group B was not significant (p=0.545).

Among the honey dressing group,18 (56.3%) participants were farmer, 10 (31.3%) participants were worker and 4 (12.5%) participants were bus driver. Among the povidone iodine dressing group,15 (46.9%) participants were farmer, 14 (43.8%) participants were worker and 3 (9.4%) participants were bus driver. The difference in the proportion of occupations between groups was statistically not significant (P value 0.582)

Among the honey dressing group,1, 20 (59.4%) participants had left foot and 12 (37.5%) participants had right foot. Among the povidone iodine dressing group, 19 (53.1%) participants had left foot and 13 (40.6%) participants had right foot. The difference in the proportion of sites between groups was statistically not significant (P value 0.785).The difference in the proportion of shapes between groups was statistically not significant (P value 0.869).

The mean wound size in honey dressing was 23.16 cm^2 at baseline, 23.16 cm^2 at 1st day follow up, 23.16 cm^2 at 3rd day follow up, 19.38 cm^2 at 5th day follow up, 16.13 cm^2 at 7th day follow up, 12.44 cm^2 at 10th day follow up and the end of 15th day,it was 10.69 cm^2. The difference in the wound size in Honey dressing group at 1st

day, 3rd day, at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05). The difference in the wound size in honey dressing group at 15th day follow up period with baseline value were statistically significant (P value <0.05). The mean wound size in povidone dressing was 23.03cm^2 at base line, 22.94cm^2 at 1st day follow up, 22.94cm^2 at 3rd day follow up, 20.28 cm^2 at 5th day follow up, 17.06 cm^2at 7th day follow up, 16.13 cm^2 at 10th day follow up and the end of 15th day, it was 15.06 cm^2. The difference in the wound size in Povidone Iodine at 1st day, 3rd day, at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05) and the difference in the wound size in honey dressing at 15th day follow up period was statistically signicicant. (P value <0.05)

The mean percentage reduction of wound size in both groups of day 1, 3 were almost same and stastically not significant, while in honey dressing group was 9.87 at 5th day follow up, 27.54 at 7th day follow up, 42.58 at 10th day follow up and the end of 15th day, it was 49.87. The difference in the wound size in Honey dressing group at 1st day, 3rd day, at5th day, 7th day, 10th day follow up period with baseline value were statistically not significant (P value >0.05). The difference in the wound size in honey dressing group at10th and 15th day follow up period with baseline value were statistically significant (P value <0.05). The mean percentage reduction in wound size in Povidone iodine dressing group was 24.44 at 5th day follow up, 17.44 at 7th day follow up, 18.91 at 10th day follow up and the end of 15th day, it was 20.24. The difference in the wound size in Povidone Iodine at 1st day, 3rd day, at5th day, 7th day, follow up period with baseline value were statistically not significant (P value >0.05). and the difference in the wound size in honey dressing at 10th and 15th day follow up period was statistically signicicant (P value <0.05)

Overall the present study showed that, dressing with Honey influences granulation and thereby promotes early healing compared to dressing with povidone iodine. However we have less sample size compare these findings with other studies. We hypothesize that, dressing with Honey might have multiple beneficial effects on wound bed preparation and healing, through the removal of necrotic plug by the enzymatic action.

There was one study conducted at AIIMS, New Delhi by Sonia Gulati et al. which showed Honey dressing is more effective as compared to povidone iodine dressing in achieving complete healing, reducing wound surface area and pain, and increasing comfort in subjects with chronic wounds [100]

One study conducted by A Shukrimi at el. They conducted a prospective study to compare the effect of honey dressing for Wagner's grade-II diabetic foot ulcers with controlled dressing group (povidone iodine followed by normal saline). Surgical debridement and appropriate antibiotics were prescribed in all patients. There were 30 patients age between 31 to 65 years old (mean of 52.1 years). The mean healing time in the standard dressing group was 15.4 days (range 9 - 36 days) compared to 14.4 days (range 7-26 days) in the honey group ($p<0.005$) In conclusion, ulcer healing was not significantly different in both study groups. Honey dressing is a safe alternative dressing for Wagner grade-II diabetic foot ulcers.[101]

Several authors have reported that honey enhances wound healing rate, compared to other conventional or topical applications in a variety of clinical conditions, namely, burns, chronic wounds, infected surgical wounds, and pressure ulcers. In 1999 Kramer conducted a review of the clinical trials in which povidone iodine was used for cleansing, irrigating, and dressing wounds. He concluded that

povidone iodine did not effectively promote good wound healing and did not reduce bacteriological wound infection [102].

Overall this study shows more favorable results with honey dressing as compared to dressing with povidone iodine in reduction of wound size of diabetic foot ulcers. However, the limitations of the study were smaller sample size. Further multicentric studies with large sample size are required to confirm these observations.

CONCLUSION

This study shows more favourable results with honey dressing as compared with povidone iodine dressing for reduction of wound size in diabetic foot ulcers.

SUMMARY

Many numerous topical and systemic agents have been used either alone or in combination for the eradication of infections, but many have been eliminated because of resistance. Diabetic wound infections caused by drug-resistant organisms are becoming more common and they have resistance to many commonly used antibiotics, that leads to increasing costs and morbidity. With an increasing frequency of antibiotic-resistant pathogens, modern medicine directs attention to natural products with increased antimicrobial property for clinical practice. Honey is one such product which is a collection of nectar from many plants, which is processed by honey bees. Honey is well known for its high nutritional and medicinal value. Honey has potent antibacterial activity which is useful in preventing and removing wound infections. It has been used as a wound care product since decades, and its use as a wound healing agent was reported for treating, chronic leg ulcers, burns wounds from many years [35-41]

The present randomized controlled trial was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi over a period, from January 2017 to December 2017. A total of 64 Diabetic Patients taking insulin or oral hyperglycaemic agents and suffering from diabetic foot ulcers which are not healed with Wagners grade 1 and 2 were enrolled. These patients were divided into two groups of 32 each that is Group A (Patients in this group underwent dressing with Honey) and Group B (Patients in this group underwent dressing with povidone iodine).

The mean wound size in honey dressing was 23.16 cm^2 at baseline,that is 23.16 cm^2at 1^{st} day follow up, 23.16 cm^2 at 3^{rd} day follow up, 19.38 cm^2 at 5^{th} day follow up, 16.13 cm^2 at 7^{th} day follow up, 12.44 cm^2 at 10^{th} day follow up and the end of 15^{th} day,it was 10.69cm^2. The difference in the wound size in Honey dressing group at 1^{st} day, 3^{rd} day, at5^{th} day, 7^{th} day, 10^{th} day follow up period with baseline value were statistically not significant (P value >0.05). The difference in the wound size in honey dressing group at 15^{th} day follow up period with baseline value were statistically significant (P value <0.05). The mean wound size in povidone dressing was 23.03cm^2 at base line, 22.94 cm^2 at 1^{st} day follow up, 22.94cm^2 at 3^{rd} day follow up, 20.28 cm^2 at 5^{th} day follow up, 17.06cm^2 at 7^{th} day follow up, 16.13cm^2 at 10^{th} day follow up and the end of 15^{th} day, it was 15.06cm^2. The difference in the wound size in Povidone Iodine at 1^{st} day, 3^{rd} day, at5^{th} day, 7^{th} day, 10^{th} day follow up period with baseline value were statistically not significant (P value >0.05) and the difference in the wound size in honey dressing at 15^{th} day follow up period was statistically signicicant. (P value <0.05)

Overall this study shows more favourable results with honey dressing as compared to dressing with povidone iodine in reduction of wound size of diabetic foot ulcers. However, the limitations of the study were smaller sample size. Further multicentric studies with large sample size are required to confirm these observations.

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ANNEXURE I – CONSENT FORM

INFORMED CONSENT

TITLE OF THE STUDY: - "**A ONE YEAR RANDOMIZED CONTROL TRIAL TO COMPARE THE EFFECTIVENESS OF HONEY DRESSING VS. POVIDONE IODINE DRESSING FOR DIABETIC FOOT ULCER AT KLE DR PRABHAKAR KORE HOSPITAL, BELAGAVI"**

PRINCIPAL INVESTIGATOR: Dr. _____

INTRODUCTION AND PURPOSE:

The diabetic foot ulcers are common complication of diabetes mellitus. They are managed by conventional methods of dressings like normal saline, Povidone iodine and eusol etc. Topical Honey application could be used as an alternative to currently available methods of dressing. Studies have shown that it has better healing properties than conventional topical agents. This study is being undertaken to compare the efficacy of Honey dressing versus Povidone iodine dressing in diabetic foot ulcer.

PROCEDURE:

I request you to kindly participate in the study titled: **A ONE YEAR RANDOMIZED CONTROL TRIAL TO COMPARE THE EFFECTIVENESS OF HONEY DRESSING VS. POVIDONE IODINE DRESSING FOR DIABETIC FOOT ULCER AT KLE DR PRABHAKAR KORE HOSPITAL, BELAGAVI"**

If you agree to participate in the study please furnish the details pertaining to the study.

BENEFITS:

1. Honey has the potential to be an inexpensive, noninvasive and non-labour intensive means to promote wound healing of diabetic foot ulcers.
2. Honey has antibacterial action, honey also appears to stimulate lymphocytic and phagocytic activity and helps in healing diabetic foot ulcer

RISKS:

- No proven side effects

ALTERNATIVES:

If patient is not willing to take part in the study, his / her treatment or any other investigations the patient wants to undergo, in future, in KLE's Dr. Prabhakar Kore Hospital and MRC will not be affected by his / her decision.

VOLUNTARY PARTICIPATION/WITHDRAWAL:

Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part I can later change my mind and withdraw from the study. My decision will not change the present or future health care or other services that I receive. The study doctor or the sponsor may stop my participation in this study. I will tell of any important new findings that may change my willingness to continue to take part. If I choose not to take part in the study I will receive the standard treatment.

COSTS:

NIL

COMPENSATION:

In the event that I become injured as a result of taking part in this study, treatment will be offered to me, No reimbursement, compensation or free medical care is given.

CONFIDENTIALITY:

All information collected about me during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

QUESTION:

For any enquiries and in case of research related injury/ illness, you may contact following person.

DR. _____
Professor of pathology
Head of Ethical Committee,
Dept of Pathology, J. N. Medical College,
K.L.E. University, Belagavi-590010.
Ph no-9480275601

Dr. _____
Post-Graduate,
Department of
General surgery,
J.N.Medical College,
Belagavi-590010.
09979259629

Dr. _____
Guide , Professor,
Department of General
Surgery
J.N.Medical College,
Belagavi-590010.
0831-2554080

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form.

Signature /Left thumb print of the participant or legally authorized representative.

Participant's Name :…….

Signature/ Left thumb impression. : …….

Name of the legally authorized representative:…….

Signature/ Left thumb impression. :…….

Witness's Name : …….

Signature/ Left thumb Impression of witness:…….

Investigators name and Signature : …….

Date and Place : …….

ANNEXURE II – PROFORMA

PROFORMA

PATIENT IDENFICIATION DATA

GROUP: CASE NO. :

I.P/ O.P.D NO.: D.O.A:

NAME:

AGE: D.O.S:

SEX: D.O.D:

OCCUPATION:

DIABETIC STATUS

Medication

Drug	Dose	Duration

ULCER DETAIL

1. Mode of onset

Traumatic	
Spontaneous	
Pressure	
Others	

2. Duration

WOUND OBSERVATION

1. Site
2. Size
3. Shape
4. Edge
5. Margin
6. Floor
7. Base
8. Discharge
9. Surrounding Skin
10. Slough /necrotic tissue

NEUROLOGICAL EXAMINATION

Sensory Loss -

Motor Weakness -

VASCULAR EXAMINATION

	Right			Left		
	Normal	Weak	Absent	Normal	Weak	Absent
Femoral artery						
Popliteal artery						
Ant. Tibial artery						
Post Tibial artery						
Dorsalis Pedis artery						

FOOT DEFORMITY

	Yes	No
Bunion		
Toe deformity		
Flat foot		
Foot drop		
Charcots foot		

DEBRIDEMENT

Date :

Anesthesia :

INVESTIGATIONS

CBC

FBS , PPBS,HbA_1C

Blood Urea

Sr. Creatinine

Urine: Routine and microscopy

X-ray Foot (as and when required)

Tissue culture/ sensitivity

Colour Doppler (as and when required)

1) <u>TREATMENT</u>

2) <u>LOCAL TREATMENT</u>

<u>Dressing</u>

Days - 1, 3,5,7,10,15

3) <u>OBSERVATION</u>

DAY	SIZE	DISCHARGE	GRANULATION	EDGE
1				
3				
5				
7				
10				
15				

ANNEXURE III - PHOTOGRAPHS

HONEY DRESSING **POVIDONE IODINE DRESSING**


DAY 1


DAY 3

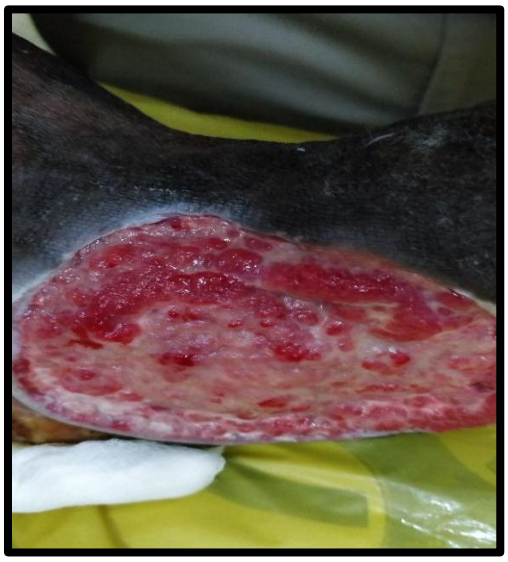
HONEY DRESSING **POVIDONE IODINE DRESSING**

DAY 5

DAY 7

HONEY DRESSING **POVIDONE IODINE DRESSING**

DAY 10



DAY 15

ANNEXURE IV – KEY TO MASTER CHART

Gender

1= Male

2= Female

Occupation

1= Farmer

2= Housewife

3= Bus driver

Medication

1= Insulin

Mode of onset

1= Traumatic

Deformities

1= Present

2= Absent

Pulsations

1= Present

2= Absent

Size is in **cm^2**

HONEY DRESSING

s.no	IP No	Age	Gender	Occupation	Medication drug	Medication dose	Medication duration (in years)	Mode of onset	WO site	WO size (Sqcm)	WO shape	WO edge	WO margin	WO floor	WO base	WO discharge	WO surroundingskin	WO sloughnecrotictissue	Sensoryloss	Motorweakness	Right femoral artery	Right poplitea lartery	Right ant tibial artery	Right post tibial artery	Right dorsalis pedis artery	LEFT femoral artery	LEFT popliteal artery	LEFT ant tibial artery	LEFT post tibial artery	LEFT dorsalis pedis artery	Bunion	Toe deformity	Flat foot	Foot drop	Charcots foot	Anesthesia	Haemoglobin(CBC)	FBS	PPBS	HBA1C	Blood urea	Serum creatinine	Urine	X-Ray foot	Tissue culture sensitivity
1	815434	58	1	1	1	Sliding Scale	6	1	L	56	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	10	0.7	Normal	1	
2	812911	72	2	2	1	Sliding Scale	11	1	L	30	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			7	12	1	Normal	1	
3	801334	60	1	2	1	Sliding Scale	10	1	R	30	Irregular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	14			8	18	1	Normal	1	
4	813183	48	1	1	1	Sliding Scale	7	1	L	12	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	17	1	Normal	1	
5	812259	70	1	1	1	Sliding Scale	9	1	L	56	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			7	18	0.7	Normal	1	
6	785718	30	1	1	1	Sliding Scale	8	1	R	10	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	17	0.7	Normal	1	
7	784955	57	1	2	1	Sliding Scale	8	1	L	12	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10.8			7	19	0.9	Normal	1	
8	784696	56	2	1	1	Sliding Scale	6	1	R	42	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	18	0.7	Normal	1	
9	808629	70	1	1	1	Sliding Scale	7	1	R	42	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	13			9	18	0.9	Normal	1	
10	754223	68	1	1	1	Sliding Scale	5	1	L	12	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			7	17	0.9	Normal	1	
11	811694	71	1	1	1	Sliding Scale	5	1	L	28	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	10	1	Normal	1	
12	819596	51	1	2	1	Sliding Scale	5	1	L	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			9	10	1	Normal	1	
13	818080	65	1	3	1	Sliding Scale	5	1	R	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	9			7	10	1	Normal	1	
14	803477	65	1	2	1	Sliding Scale	5	1	L	21	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			9	10	0.8	Normal	1	
15	809802	62	1	3	1	Sliding Scale	6	1	R	25	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			9	11	1	Normal	1	
16	810120	49	1	1	1	Sliding Scale	6	1	R	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			7	18	1	Normal	1	
17	761518	65	1	2	1	Sliding Scale	7	1	R	18	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	10	1	Normal	1	
18	800157	63	1	1	1	Sliding Scale	5	1	L	21	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	17	1.1	Normal	1	
19	799381	60	2	2	1	Sliding Scale	4	1	L	20	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	13	1	Normal	1	
20	798630	55	1	3	1	Sliding Scale	5	1	L	28	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	0.8	Normal	1	
21	781212	71	1	1	1	Sliding Scale	5	1	L	28	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			7	10	1	Normal	1	
22	826977	69	1	1	1	Sliding Scale	6	1	L	28	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	10	1	Normal	1	
23	792861	75	1	1	1	Sliding Scale	6	1	R	21	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	0.9	Normal	1	
24	824200	75	1	1	1	Sliding Scale	5	1	L	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	0.9	Normal	1	
25	821668	75	1	1	1	Sliding Scale	5	1	L	20	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	10	1	Normal	1	
26	795618	65	1	3	1	Sliding Scale	5	1	R	12	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	10	1.2	Normal	1	
27	790080	75	2	2	1	Sliding Scale	6	1	R	12	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	13			8	14	1	Normal	1	
28	816658	63	1	1	1	Sliding Scale	3	1	L	8	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	13			7	10	1.2	Normal	1	
29	808816	50	2	2	1	Sliding Scale	6	1	L	9	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			7	13	1.4	Normal	1	
30	790012	75	2	2	1	Sliding Scale	6	1	R	12	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			7	12	1	Normal	1	
31	802858	63	1	1	1	Sliding Scale	8	1	L	8	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			7	11	1	Normal	1	
32	825047	60	1	1	1	Sliding Scale	5	1	L	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			7	10	0.8	Normal	1	

POVIDONE IODINE DRESSING

S.no	IP No	Age	Gender	Occupation	Medication drug	Medication dose	Medication duration (in years)	Mode of onset	WO site	WO size (Sqcm)	WO shape	WO edge	WO margin	WO floor	WO base	WO discharge	WO surroundingskin	WO sloughnecrotictissue	Sensoryloss	Motorweakness	Right femoral artery	Right poplitea lartery	Right ant tibial artery	Right post tibial artery	Right dorsalis pedis artery	LEFT femoral artery	LEFT popliteal artery	LEFT ant tibial artery	LEFT post tibial artery	LEFT dorsalis pedis artery	Bunion	Toe deformity	Flat foot	Foot drop	Charcots foot	Anesthesia	Haemoglobin(CBC)	FBS	PPBS	HBA1C	Blood urea	Serum creatinine	Urine	X-Ray foot	Tissue culture sensitivity
1	780328	64	2	1	1	Sliding Scale	5	1	R	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	1.2	Normal	1	
2	784041	45	1	1	1	Sliding Scale	6	1	R	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			7	10	1.2	Normal	1	
3	780285	75	1	2	1	Sliding Scale	6	1	L	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	1.1	Normal	1	
4	780475	60	1	1	1	Sliding Scale	5	1	L	24	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	1.2	Normal	1	
5	782415	81	2	1	1	Sliding Scale	3	1	L	12	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			7	10	0.8	Normal	1	
6	711925	62	1	1	1	Sliding Scale	6	1	L	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			7	10	1.2	Normal	1	
7	820837	68	1	2	1	Sliding Scale	4	1	L	12	Irregular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	14	1.2	Normal	1	
8	820728	61	2	2	1	Sliding Scale	6	1	R	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			7	11	0.8	Normal	1	
9	816945	55	1	1	1	Sliding Scale	6	1	L	12	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	11	1.2	Normal	1	
10	807200	68	1	1	1	Sliding Scale	7	1	R	36	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			9	12	1.1	Normal	1	
11	819428	51	1	2	1	Sliding Scale	5	1	L	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	11	1.1	Normal	1	
12	816945	55	1	2	1	Sliding Scale	6	1	L	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	8			7	11	0.7	Normal	1	
13	818207	48	1	3	1	Sliding Scale	8	1	L	15	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	11	0.7	Normal	1	
14	810246	63	1	2	1	Sliding Scale	5	1	L	18	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	14	0.6	Normal	1	
15	808935	60	1	2	1	Sliding Scale	7	1	L	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			7	10	1	Normal	1	
16	810071	64	1	1	1	Sliding Scale	6	1	R	30	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10.8			8	10	1	Normal	1	
17	781632	54	2	2	1	Sliding Scale	7	1	R	30	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	10	1	Normal	1	
18	823119	60	2	2	1	Sliding Scale	8	1	R	36	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10.9			8	18	0.9	Normal	1	
19	807113	30	1	3	1	Sliding Scale	7	1	L	42	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	10	0.9	Normal	1	
20	809044	40	1	1	1	Sliding Scale	8	1	R	30	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			9	10	1	Normal	1	
21	803044	50	1	2	1	Sliding Scale	10	1	L	20	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	10	1	Normal	1	
22	823490	30	1	3	1	Sliding Scale	8	1	R	56	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	19	0.8	Normal	1	
23	818907	50	1	1	1	Sliding Scale	6	1	L	30	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			7	10	1	Normal	1	
24	817394	70	1	1	1	Sliding Scale	7	1	L	48	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	12			8	10	1	Normal	1	
25	823746	60	2	2	1	Sliding Scale	8	1	R	15	Oval	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			9	10	1.1	Normal	1	
26	817060	70	1	1	1	Sliding Scale	5	1	L	8	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			9	13	1.1	Normal	1	
27	812902	48	1	1	1	Sliding Scale	4	1	L	15	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			9	10	1.2	Normal	1	
28	797358	70	2	2	1	Sliding Scale	5	1	R	21	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			10	12	1	Normal	1	
29	809182	89	2	1	1	Sliding Scale	8	1	L	18	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	13			8	10	1.2	Normal	1	
30	825486	56	1	2	1	Sliding Scale	8	1	R	20	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	11			8	10	1.1	Normal	1	
31	822043	62	1	1	1	Sliding Scale	5	1	R	36	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	13			8	13	0.5	Normal	1	
32	814989	65	1	2	1	Sliding Scale	6	1	L	24	Circular	Slopping	Well Defined	Slough	Tendon	Serous	Normal	1	2	2	1	1	1	1	1	1	1	1	1	1	2	2	2	2	2	Spinal	10			8	10	1	Normal	1	

Colour doppler	Treatment	Local treatment	L.T dressing	Day1 size	% REDUC	Day 1 discharge	Day1 granulation	Day1 edge	Day3 size	% REDUC	Day 3 discharge	Day3 granulation	Day3 edge	Day5 size	% REDUC	Day 5 discharge	Day5 granulation	Day5 edge	Day7 size	% REDUC	Day 7 discharge	Day7 granulation	Day7 edge	Day10 size	% REDUC	Day 10 discharge	Day10 granulation	Day10 edge	Day15 size	% REDUC	Day 15 discharge	Day15 granulation	Day15 edge
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	56	0	Serous	Slough	Slopping	56	0	Serous	Slough	Slopping	45	19.6429	Minimal	Present	Slopping	42	25.00	Nil	Present	Slopping	30	46.43	Nil	Healthy	Slopping	30	46.43	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	30	0	Serous	Slough	Slopping	30	0	Minimal	Slough	Slopping	20	33.33	Nil	Present	Slopping	20	33.33	Nil	Present	Slopping	12	60.00	Nil	Healthy	Slopping	12	60.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	30	0	Serous	Slough	Slopping	30	0	Minimal	Slough	Slopping	27	10.00	Nil	Present	Slopping	20	33.33	Nil	Present	Slopping	16	46.67	Nil	Healthy	Slopping	8	73.33	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	12	0.00	Minimal	Present	Slopping	6	50.00	Nil	Present	Slopping	6	50.00	Nil	Healthy	Slopping	4	66.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	56	0	Serous	Slough	Slopping	56	0	Minimal	Slough	Slopping	42	25.00	Minimal	Slough	Slopping	42	25.00	Nil	Present	Slopping	36	35.71	Nil	Healthy	Slopping	30	46.43	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	10	0	Serous	Slough	Slopping	10	0	Serous	Slough	Slopping	15	-50.00	Nil	Present	Slopping	10	0.00	Nil	Present	Slopping	8	20.00	Nil	Healthy	Slopping	8	20.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	12	0	Serous	Slough	Slopping	12	0	Serous	Present	Slopping	12	0.00	Nil	Present	Slopping	6	50.00	Nil	Present	Slopping	6	50.00	Nil	Healthy	Slopping	6	50.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	42	0	Serous	Slough	Slopping	42	0	Minimal	Present	Slopping	42	0.00	Nil	Present	Slopping	25	40.48	Nil	Healthy	Slopping	20	52.38	Nil	Healthy	Slopping	16	61.90	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	42	0	Serous	Slough	Slopping	42	0	Minimal	Slough	Slopping	33	21.43	Nil	Present	Slopping	30	28.57	Nil	Present	Slopping	20	52.38	Nil	Healthy	Slopping	20	52.38	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	17	-41.67	Minimal	Slough	Slopping	12	0.00	Nil	Present	Slopping	8	33.33	Nil	Healthy	Slopping	6	50.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	28	0	Serous	Slough	Slopping	28	0	Serous	Slough	Slopping	20	28.57	Minimal	Present	Slopping	18	35.71	Minimal	Present	Slopping	10	64.29	Nil	Healthy	Slopping	10	64.29	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	24	0	Serous	Slough	Slopping	24	0	n	Present	Slopping	18	25.00	Nil	Present	Slopping	15	37.50	Nil	Healthy	Slopping	15	37.50	Nil	Healthy	Slopping	8	66.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	24	0	Serous	Slough	Slopping	24	0	Minimal	Slough	Slopping	16	33.33	Minimal	Present	Slopping	15	37.50	Nil	Present	Slopping	8	66.67	Nil	Healthy	Slopping	8	66.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	21	0	Serous	Slough	Slopping	21	0	Minimal	Present	Slopping	18	14.29	Minimal	Present	Slopping	18	14.29	Nil	Healthy	Slopping	18	14.29	Nil	Healthy	Slopping	16	23.81	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	25	0	Serous	Slough	Slopping	25	0	Serous	Slough	Slopping	16	36.00	Nil	Present	Slopping	16	36.00	Nil	Present	Slopping	16	36.00	Nil	Healthy	Slopping	6	76.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	24	0	Serous	Slough	Slopping	24	0	Serous	Slough	Slopping	27	-12.50	Minimal	Present	Slopping	18	25.00	Minimal	Present	Slopping	15	37.50	Nil	Healthy	Slopping	15	37.50	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	18	0	Serous	Slough	Slopping	18	0	Serous	Slough	Slopping	20	-11.11	Nil	Present	Slopping	10	44.44	Nil	Present	Slopping	10	44.44	Nil	Healthy	Slopping	10	44.44	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	21	0	Serous	Slough	Slopping	21	0	Minimal	Slough	Slopping	18	14.29	Minimal	Present	Slopping	18	14.29	Nil	Present	Slopping	10	52.38	Nil	Healthy	Slopping	10	52.38	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	20	0	Serous	Slough	Slopping	20	0	Serous	Slough	Slopping	16	20.00	Minimal	Present	Slopping	15	25.00	Nil	Present	Slopping	8	60.00	Nil	Healthy	Slopping	8	60.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	28	0	Serous	Slough	Slopping	28	0	Serous	Slough	Slopping	18	35.71	Nil	Present	Slopping	18	35.71	Nil	Present	Slopping	18	35.71	Nil	Present	Slopping	10	64.29	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	28	0	Serous	Slough	Slopping	28	0	Serous	Slough	Slopping	20	28.57	Nil	Present	Slopping	15	46.43	Nil	Present	Slopping	8	71.43	Nil	Healthy	Slopping	8	71.43	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	28	0	Serous	Slough	Slopping	28	0	Serous	Slough	Slopping	18	35.71	Nil	Present	Slopping	18	35.71	Nil	Present	Slopping	10	64.29	Nil	Healthy	Slopping	10	64.29	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	21	0	Serous	Slough	Slopping	21	0	Serous	Present	Slopping	18	14.29	Nil	Present	Slopping	12	42.86	Nil	Present	Slopping	12	42.86	Nil	Healthy	Slopping	10	52.38	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	24	0	Serous	Slough	Slopping	24	0	Serous	Slough	Slopping	15	37.50	Nil	Present	Slopping	15	37.50	Nil	Present	Slopping	8	66.67	Nil	Healthy	Slopping	8	66.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	20	0	Serous	Slough	Slopping	20	0	Serous	Present	Slopping	12	40.00	Nil	Present	Slopping	12	40.00	Nil	Present	Slopping	9	55.00	Nil	Healthy	Slopping	9	55.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	12	0.00	Minimal	Present	Slopping	6	50.00	Nil	Present	Slopping	6	50.00	Nil	Healthy	Slopping	4	66.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	14	-16.67	Nil	Present	Slopping	12	0.00	Nil	Present	Slopping	12	0.00	Nil	Healthy	Slopping	12	0.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	8	0	Serous	Slough	Slopping	8	0	Minimal	Slough	Slopping	8	0.00	Nil	Present	Slopping	8	0.00	Nil	Healthy	Slopping	6	25.00	Nil	Healthy	Slopping	6	25.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	9	0	Serous	Slough	Slopping	9	0	Serous	Slough	Slopping	12	-33.33	Minimal	Present	Slopping	9	0.00	Nil	Present	Slopping	9	0.00	Nil	Healthy	Slopping	8	11.11	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	12	0	Serous	Slough	Slopping	12	0	n	Slough	Slopping	11	8.33	Nil	Present	Slopping	12	0.00	Nil	Present	Slopping	10	16.67	Nil	Healthy	Slopping	10	16.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	8	0	Serous	Slough	Slopping	8	0	Serous	Slough	Slopping	10	-25.00	Nil	Present	Slopping	8	0.00	Nil	Healthy	Slopping	6	25.00	Nil	Healthy	Slopping	6	25.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	1	24	0	Serous	Slough	Slopping	24	0	Serous	Slough	Slopping	18	25.00	Nil	Present	Slopping	15	37.50	Nil	Present	Slopping	12	50.00	Nil	Healthy	Slopping	10	58.33	Nil	Healthy	Slopping

Colour doppler	Treatment	Local treatment	L.T dressing	Day1 size	% REDUC	Day 1 discharge	Day1 granulation	Day1 edge	Day3 size	% REDUC	Day 3 discharge	Day3 granulation	Day3 edge	Day5 size	% REDUC	Day 5 discharge	Day5 granulation	Day5 edge	Day7 size	% REDUC	Day 7 discharge	Day7 granulation	Day7 edge	Day10 size	% REDUC	Day 10 discharge	Day10 granulation	Day10 edge	Day15 size	% REDUC	Day 15 discharge	Day15 granulation	Day15 edge
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	15	0.00	Serous	Slough	Slopping	8	46.67	Minimal	Slough	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	15	0.00	Minimal	Slough	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Nil	Present	Slopping	8	46.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	15	0.00	Serous	Slough	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	24	0	Serous	Slough	Slopping	24	0	Serous	Slough	Slopping	24	0.00	Serous	Slough	Slopping	15	37.50	Minimal	Present	Slopping	15	37.50	Minimal	Present	Slopping	15	37.50	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	12	0.00	Minimal	Slough	Slopping	9	25.00	Minimal	Present	Slopping	9	25.00	Nil	Present	Slopping	9	25.00	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	24	0	Serous	Slough	Slopping	24	0	Serous	Slough	Slopping	24	0.00	Minimal	Slough	Slopping	24	0.00	Minimal	Present	Slopping	20	16.67	Nil	Present	Slopping	20	16.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	12	0.00	Serous	Slough	Slopping	12	0.00	Minimal	Present	Slopping	10	16.67	Minimal	Present	Slopping	10	16.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	15	0.00	Serous	Slough	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Minimal	Present	Slopping	12	20.00	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	12	0	Serous	Slough	Slopping	12	0	Serous	Slough	Slopping	12	0.00	Serous	Slough	Slopping	6	50.00	Minimal	Present	Slopping	6	50.00	Nil	Present	Slopping	6	50.00	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	36	0	Serous	Slough	Slopping	36	0	Serous	Slough	Slopping	36	0.00	Serous	Slough	Slopping	30	16.67	Minimal	Present	Slopping	30	16.67	Minimal	Present	Slopping	25	30.56	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	15	0.00	Serous	Slough	Slopping	12	20.00	Minimal	Present	Slopping	12	20.00	Minimal	Present	Slopping	12	20.00	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Minimal	Slough	Slopping	15	0.00	Minimal	Slough	Slopping	8	46.67	Nil	Slough	Slopping	8	46.67	Nil	Present	Slopping	8	46.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	8	46.67	Serous	Slough	Slopping	8	46.67	Nil	Present	Slopping	6	60.00	Nil	Present	Slopping	6	60.00	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	18	0	Serous	Slough	Slopping	18	0	Serous	Slough	Slopping	18	0.00	Serous	Slough	Slopping	10	44.44	Nil	Present	Slopping	10	44.44	Nil	Present	Slopping	10	44.44	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	16	-6.67	Nil	Slough	Slopping	15	0.00	Nil	Present	Slopping	8	46.67	Nil	Present	Slopping	8	46.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	30	0	Serous	Slough	Slopping	30	0	Serous	Slough	Slopping	30	0.00	Serous	Slough	Slopping	25	16.67	Minimal	Present	Slopping	25	16.67	Nil	Present	Slopping	25	16.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	30	0	Serous	Slough	Slopping	30	0	Serous	Slough	Slopping	30	0.00	Serous	Slough	Slopping	30	0.00	Minimal	Present	Slopping	30	0.00	Nil	Present	Slopping	25	16.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	36	0	Serous	Slough	Slopping	36	0	Serous	Slough	Slopping	36	0.00	Minimal	Slough	Slopping	30	16.67	Minimal	Present	Slopping	30	16.67	Nil	Present	Slopping	25	30.56	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	42	0	Serous	Slough	Slopping	42	0	Serous	Slough	Slopping	42	0.00	Serous	Slough	Slopping	36	14.29	Minimal	Present	Slopping	36	14.29	Nil	Present	Slopping	30	28.57	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	30	0	Serous	Slough	Slopping	30	0	Serous	Slough	Slopping	30	0.00	Serous	Slough	Slopping	25	16.67	Minimal	Present	Slopping	25	16.67	Nil	Present	Slopping	25	16.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	20	0	Serous	Slough	Slopping	20	0	Serous	Slough	Slopping	20	0.00	Serous	Slough	Slopping	20	0.00	Minimal	Present	Slopping	18	10.00	Minimal	Present	Slopping	8	60.00	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	56	0	Serous	Slough	Slopping	56	0	Serous	Slough	Slopping	42	25.00	Minimal	Slough	Slopping	42	25.00	Minimal	Present	Slopping	36	35.71	Nil	Present	Slopping	36	35.71	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	30	0	Serous	Slough	Slopping	27	10	Serous	Slough	Slopping	30	0.00	Serous	Slough	Slopping	20	33.33	Minimal	Slough	Slopping	20	33.33	Minimal	Present	Slopping	20	33.33	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	48	0	Serous	Slough	Slopping	48	0	Serous	Slough	Slopping	35	27.08	Serous	Slough	Slopping	35	27.08	Minimal	Slough	Slopping	35	27.08	Minimal	Present	Slopping	35	27.08	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Minimal	Present	Slopping	8	46.67	Nil	Present	Slopping	8	46.67	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	8	0	Serous	Slough	Slopping	8	0	Serous	Slough	Slopping	6	25.00	Minimal	Present	Slopping	6	25.00	Nil	Present	Slopping	6	25.00	Nil	Present	Slopping	6	25.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	15	0	Serous	Slough	Slopping	15	0	Serous	Slough	Slopping	8	46.67	Nil	Present	Slopping	8	46.67	Nil	Healthy	Slopping	8	46.67	Nil	Healthy	Slopping	8	46.67	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	21	0	Serous	Slough	Slopping	21	0	Serous	Slough	Slopping	18	14.29	Minimal	Present	Slopping	18	14.29	Minimal	Present	Slopping	18	14.29	Nil	Present	Slopping	16	23.81	Nil	Present	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	18	0	Serous	Slough	Slopping	18	0	Serous	Slough	Slopping	10	44.44	Nil	Present	Slopping	10	44.44	Nil	Healthy	Slopping	8	55.56	Nil	Healthy	Slopping	8	55.56	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	20	0	Serous	Slough	Slopping	20	0	Serous	Slough	Slopping	12	40.00	Minimal	Present	Slopping	12	40.00	Nil	Present	Slopping	12	40.00	Nil	Healthy	Slopping	10	50.00	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	36	0	Serous	Slough	Slopping	36	0	Serous	Slough	Slopping	25	30.56	Minimal	Present	Slopping	25	30.56	Nil	Present	Slopping	20	44.44	Nil	Healthy	Slopping	20	44.44	Nil	Healthy	Slopping
Not Done	Inj Ceftriaxone 1gm BD	Dressing	2	24	0	Serous	Slough	Slopping	24	0	Serous	Slough	Slopping	15	37.50	Minimal	Present	Slopping	15	37.50	Nil	Present	Slopping	15	37.50	Nil	Healthy	Slopping	12	50.00	Nil	Healthy	Slopping