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Original Research Article

Autologous Platelet Rich Plasma DressingVersus Normal Saline Dressing in Diabetic Foot Management

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Conflict of interest: Nil

Abstract:

Purpose: This study aimed to compare platelet-rich plasma (PRP) versus conventional ordinary dressing in the management of diabetic foot wounds.

Background: Diabetic foot wound treatment poses a considerable burden on the medical system, with long waiting times for healing in the public hospital system. PRP enables efficient treatment of many patients with hemostatic, anti-inflammatory, and analgesic substances.

Patients and Methods: This prospective study was focused on 100 diabetic feet wounds. Patients were divided into two groups: and group A received PRP dressing (*N*=50, 50%). group B received conventional ordinary dressing (*N*=50, 50%) The mean follow-up period was 3 weeks.

Results: Mean weeks for complete healing is significantly lower in platelet rich plasma group than normal saline group. Significant difference of initial and final area of infection site is 542.8 inplatelet group, 277.4 in normal saline group. Percentage of area reduction (mean 39.25) is significantly higher in platelet group.

Conclusion: There have been considerable advancements in the use of PRP in therapeutic processes in recent years in tissue regeneration therapy. PRP is a powerful tool for the treatment of chronic wounds and very promising for diabetic foot wounds; PRP enables healing, and reduces infection rates and exudates.

Keywords: PDGF, PGI2, PMN, PRFM, PRF-L.

Introduction

Chronic wounds are characterized by a long inflammatory phase that hinders regenerative wound healing. Chronic wounds, especially in patients with diabetes mellitus (DM), are a major health challenge. The goal of wound care in chronic ulcers is to facilitate healing and prevent lower extremity amputations using standardized protocols of wound care. The standard treatment algorithm includes a complete patient and wound assessment, history, physical examination, and a variety of diagnostic tests that determine the need for infection control intervention, revascularization, excision and debridement, skin graft/flap, wound protection, and education.

Diabetic foot is defined as 'Infection, ulceration or destruction of tissues of the foot associated with neuropathy and/or peripheral artery disease in the lower extremity of a person with (a history of) diabetes mellitus. Non-healing diabetic foot ulcers are a common cause of amputation andrepresent significant costs to health care system and reduce patient quality of life. The goal of diabetic foot ulcer treatment is to obtain wound closure as expeditiously as possible. Accepted therapeutic objectives and standards of care for diabetic foot ulcers include wound debridement, pressure relief in the wound area, appropriate wound management (e.g., moist wound healing), infection management, ischemia management, medical management of comorbidities, and surgical management as needed. Apart from these conventional methods to facilitate wound healing various new methods are emerging such as cellular therapies which include platelet-rich plasma (PRP). This can have an adjunctive role in a standardized,quality treatment plan. [4]

Platelets release certain growth factors from alpha granules which are located in thrombocyte cell membrane which include platelet derived growth factor (PDGF), epidermal growth factor (EGF), platelet derived angiogenesis factor and platelet

factor. [5] These factors act locally on wound and hasten the healing process. Platelet extract has been used in many studies and has shown impressive results in healing of chronic non-healing diabetic foot ulcers. Since not all patients can afford commercially available recombinant platelet gel for dressing, platelet extract from the patient's own blood has beenused in trials on chronic wound.

The purpose of this study is to evaluate how autologous platelet-rich plasma (PRP) affects initial wound healing trajectories of chronic, non-healing diabetic foot wounds in a hospital care setting.

Hence, this study intends to demonstrate the therapeutic role of autologous platelet rich plasma in healing of chronic non-healing diabetic footulcers.

Methodology

Aim of the study

- To compare the efficiency of autologous platelet rich plasma inchronic non healing ulcers in comparison to conventional dressing.
- To assess the response to autologous platelet rich plasma in terms of complete wound closure in study and control groups.

Type of study: Prospective study.

Duration of study: 1 year (June 2021 to May 2022)

Sampling procedure: simple random sampling

Sample size: 100

Inclusion criteria

- Age group of 18 to 80 years with long standing non healing diabetic footulcers
- Patients who had given written consent to be a part of study group
- Ulcers greater than or equal to 4 weeks duration
- Ulcers less than or equal to 15 cm sq. in size
- Hb greater than 10 g/DL

Exclusion criteria

- Screening platelet count less than 1,00,000/micro litre
- Patients with known or suspected osteomylitis
- Patients with immunodeficiency
- Patients with serum creatinine above 1.5 mg/Dl
- Severe infection

Method

The present study was carried out at Government Theni Medical College, Theni, where 100 patients with diabetic foot ulcer more than 4 weeks participated in the present study. Using a pretested and predesigned proforma the study population was randomized into either study group or control group using a computerized randomization chart.

Name, age, sex, presenting complaints are recorded. Detailed physical examination including nutritional status, built, status of vascular system, neurological system are recorded. Investigations like hemoglobin, total leucocyte count, differential count, FBS, PPBS, urine sugar, blood urea, serum creatinine, lipid profile, wound culture and sensitivity, radiograph of the affected part are done. Medical treatment and wound debridement, regular wound dressings will be carried out. Healthy wound will be managed with splitskin grafting and secondary suturing.

Out of 100, patients, 50 took treatment in the form of conventional normal saline dressings and 50 took treatment with autologous Platelet rich plasma dressing. Off-loading of pressure from the affected area were done in both the groups. Photographs of the ulcers before and after the dressings were taken, along with culture and sensitivity of the ulcers. After undergoing a detailed clinical examination, and relevant investigations, the initial wound area was recorded after sharp debridement by Measuring length x width. Both the groups were subjected to dressings. Platelet rich plasma dressing was done twice weekly. The patients were followed up a period of 3 weeks in both the groups. The outcome that is the area of the target ulcer was measured by using a metric tape. Results were calculated by using student 't' test.

- Platelet rich plasma (PRP) is an autologous product, with large number of platelets in a small volume of plasma. It is derived by centrifugation ofthe whole blood. PRP is effective in improving the natural way of woundhealing, soft tissue and bone reconstruction
- PRP enhances epithelial, epidermal and endothelial regeneration. It promotes angiogenesis, collagen synthesis, soft tissue healing and reducedermal scaring.
- PRP produces its effects via the release of growth factors from alpha granules to accelerate wound healing. This process begins within minutesand 90% of the GF are secreted within 1 hour. This process continuous for about 7 days. The rate of wound healing is directly proportional to theamount of platelets found in the site.
- PRP functions as a tissue sealant and drug delivery system and platelets initiates wound re pair by releasing locally acting growth factors via alpha granules degranulation.
- Platelet derived growth factor (PDGF-AA, BB and AB isomers)
- Transforming growth factor beta (TGF beta), platelet factor4(PF4)
- Interleukin-1(IL-1), platelet derived angiogenesis factor (PDAF)
- Vascular endothelial growth factor (PDEGF), epithelial cell growthfactor (ECGF)

- Insulin like growth factor (IGF), osteocalcil(Oc), osteonectin(On), fibrinogen(Ff), vitronectin(Vn), Fibronectin(Fn) and thrombospondin 1(TSP1)

Observation and Results

Table 1: Comparison of Age

Age In Years	PlateletRich Plasma Dressing	Normal Saline Dressing
< 40	30	25
> 40	20	25
Total	50	50
Mean	44.92	45.16
SD	13.06	11.88
T' Value	0.096	
P' Value	**0.924 Not Significant**	

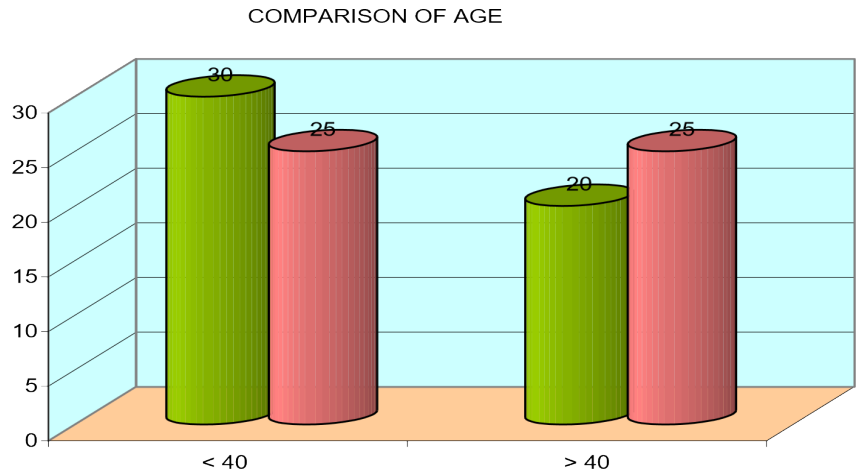

Figure 1: Platelet Rich Plasma Dressing

Normal Saline Dressing: Mean age of platelet group patients is 44.92 and normal saline group mean age is 45.16. No significant difference between both groups regarding age. P value 0.924 not significant.

Table 2: Comparison of Gender

Gender	PlateletRich Plasma Dressing	Normal Saline Dressing
Male	28	34
Female	22	16
Total	50	50
Chi sq' Value	1.061	
P' Value	**0.303 Not Significant**	

No significant difference between both groups regarding gender. P value is 0.303 Not significant

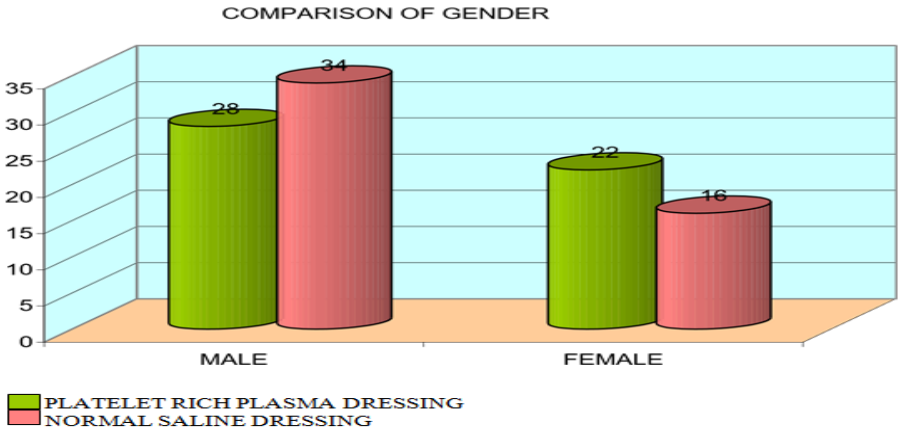

Figure 2:

Table 3: Comparison of Type of Ulcer

Type Of Ulcer	PlateletRich Plasma Dressing	Normal Saline Dressing
Spontaneous	9	12
Traumatic	41	38
TOTAL	50	50
chisq' value	0.241	
p' value	**0.623 Not significant**	

Traumatic ulcer is higher in both groups when compared with spontaneous ulcer. No significant difference between both groups regarding type of ulcer. P value is 0.623 not significant.

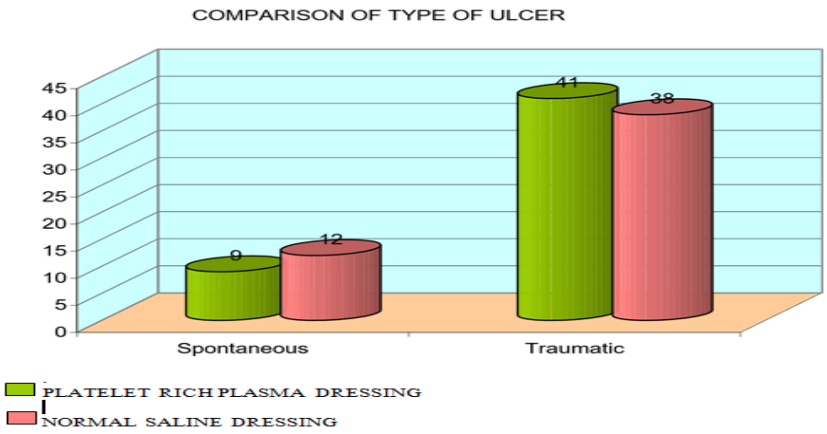

Figure 3:

Table 4: Comparison of Site

Site	PlateletRich Plasma Dressing	Normal Saline Dressing
Plantar aspect	17	14
Dorsal aspect	13	16
Lateral malleolus	8	5
Medial malleolus	12	15
TOTAL	50	50
chisq' value	1.63	
p' value	**0.653 Not significant**	

Plantar and dorsal aspect is the common site in both groups. Nosignificant difference between the site. P value is 0.653 not significant.

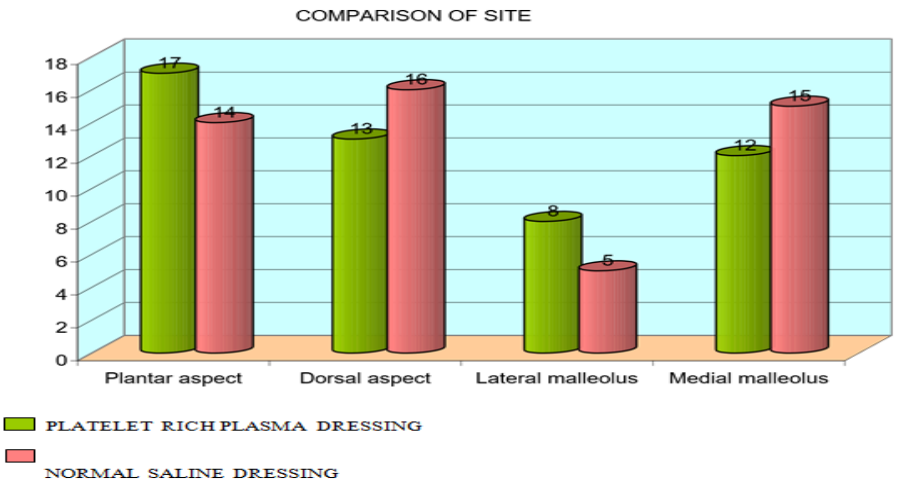

Figure 4:

Table 5: Comparison of Mean Initial Area

Initial area(IA)	Platelet Rich Plasma Dressing	NormalSaline Dressing
MEAN	1223.56	1278.46
SD	426.64	430.75
t' value	0.64	
p' value	**0.523 Not significant**	

No significant difference between both groups regarding mean initialarea of infection site. P value is 0.523 not significant.

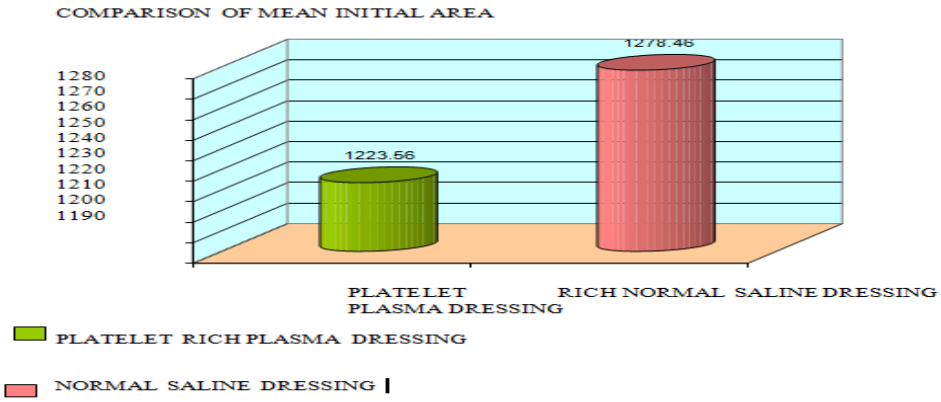

Figure 5:

Table 6: Comparison of Mean Final Area

Final area(FA)	Platelet Rich Plasma Dressing	NormalSaline Dressing
MEAN	680.72	1001.1
SD	162.99	357.48
t' value	5.766	
p' value	**< 0.001 Significant**	

Final area of infection site is 680.7 in platelet group, 1001.1 in normal saline group. This difference is significantly lower in platelet group. P value is < 0.001 significant.

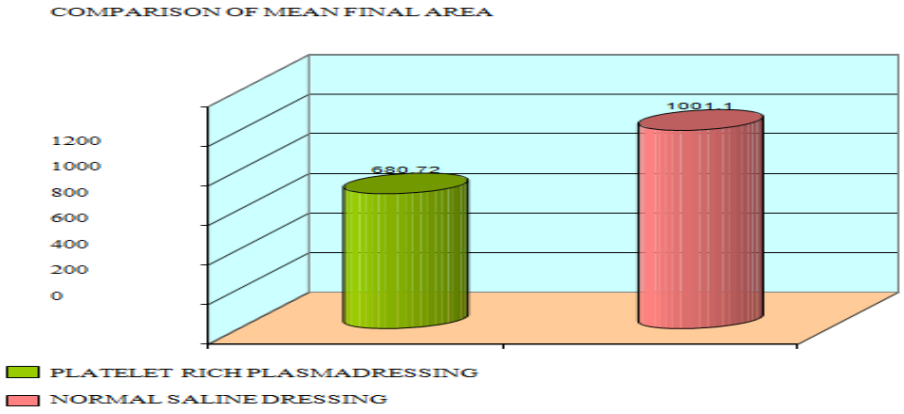

Figure 6:

Table 7: Comparison of Difference of Initial Area and FinalArea

IA-FA	Platelet Rich PlasmaDressing	Normal Saline Dressing
MEAN	542.84	277.36
SD	416.92	436.03
t' value	3.112	
p' value	**0.002 Significant**	

Significant difference of initial and final area of infection site is 542.8 in platelet group, 277.4 in normal saline group. P value is 0.002.

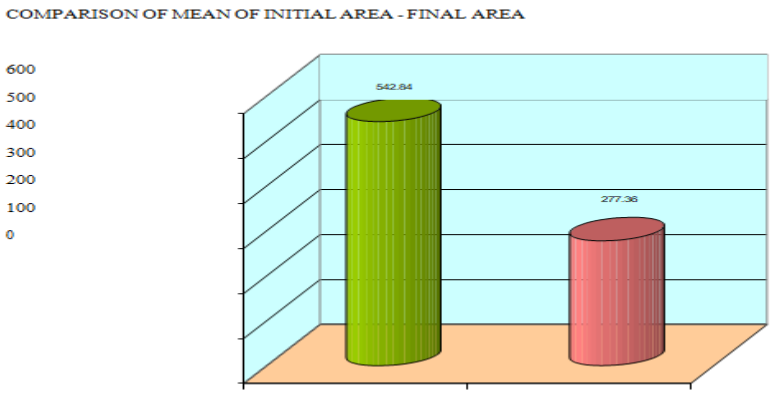

Figure 7:

Table 8: Comparison of Complete Healing

weeks for completehealing	Platelet Rich Plasma Dressing	NormalSaline Dressing
MEAN	3.172	4.56
SD	0.872	0.733
t' value	8.613	
p' value	**< 0.001 Significant**	

Mean weeks for complete healing is significantly lower in platelet richplasma group than normal saline group. 3.17 In platelet group and 4.56 in normal saline group. P value is < 0.001 significant.

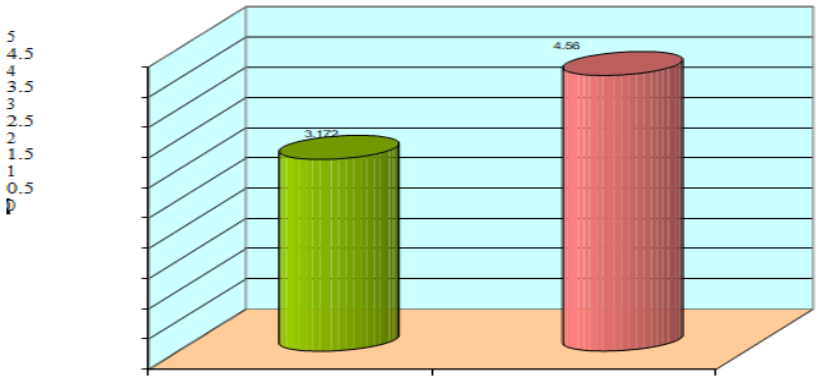

Figure 8:

Table 9: Comparison of Percentage Area Reduction

% area Reduction	Platelet Rich PlasmaDressing (%)	Normal Saline Dressing (%)
<15	1 (2.0)	26(52.0)
15-30	20 (40.0)	5(10.0)
>30	29 (58.0)	19 (38.0)
Total	50(100.0)	25(100.0)
MEAN	39.25	17.288
SD	21.003	26.575
t' value	4.585	
p' value	**< 0.001 Significant**	

Percentage of area reduction (mean 39.25) is significantly higher inplatelet group, 17.29 in normal saline group. P value is < 0.001 significant.

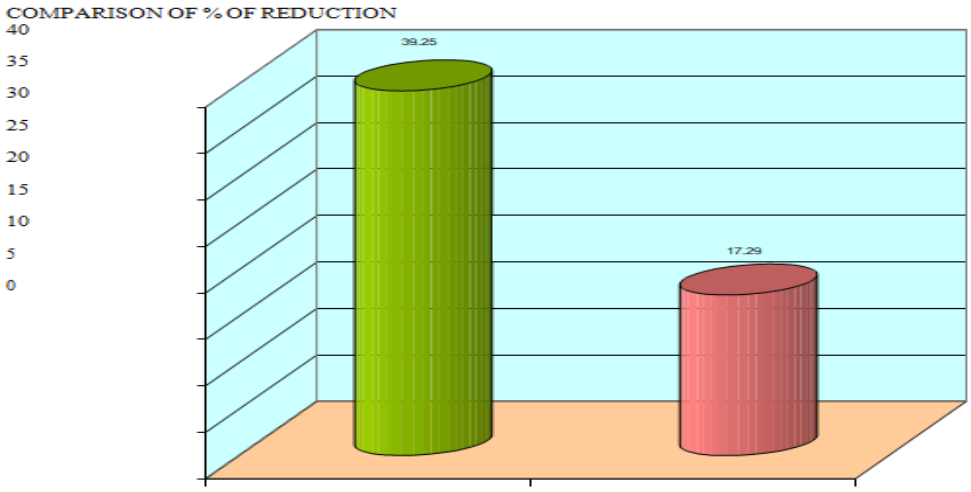

Figure 9:

Results and Summary

- Mean age of platelet group patients is 44.92 and normal saline group mean age is 45.16. No significant difference between both groups regarding age. P value 0.924 not significant.
- No significant difference between both groups regarding gender. P value is 0.303 Not significant.
- Traumatic ulcer is higher in both groups when compared with spontaneous ulcer. No significant difference between both groups regarding type of ulcer. P value is 0.623 Not significant.
- Plantar and dorsal aspect is the common site in both groups. No significant difference between the site. P value is 0.653 Not significant.
- No significant difference between both groups regarding mean initial area of infection site. P value is 0.523 Not significant.
- Final area of infection site is 680.7 in platelet group, 1001.1 in normal saline group. This difference is significantly lower in platelet group. **P value is < 0.001** significant.
- Significant difference of initial and final area of infection site is 542.8 in platelet group, 277.4 in normal saline group. **P value is 0.002**.
- Mean weeks for complete healing is significantly lower in platelet rich plasma group than normal saline group. 3.17 in platelet group and 4.56 in normal saline group. **P value is < 0.001** significant.
- Percentage of area reduction (mean 39.25) is significantly higher in platelet group, 17.29 in normal saline group. **P value is < 0.001** significant.

Discussion

Platelet Rich Plasma (PRP):

Platelet-rich plasma (PRP) is an autologous product, with large number of platelets in a small volume of plasma. It is derived by centrifugation of the whole blood. PRP is effective in improving the natural way of wound healing, soft tissue and bone reconstruction. PRP incorporates high concentrations of fibrin, PDGF, into the graft mixture. Through recent studies, it has been learnt that PRP has wide uses in clinical wound healing. When added to small bony defects, PRP increases the bone density. In case of larger defects, it is used in combination with grafting material. PRP can also be exogenously applied to soft tissues, as it promotes tissue sealing and wound healing. If we use PRP preoperatively, it decreases the hospital stay and the post-operative need of blood and blood products. PRP in recent times has also found its application in the field of cellular therapeutics and tissue engineering. Platelet-rich plasma (PRP) is a fibrin tissue adhesive. It is different from fibrin glue by the high platelet content. The platelets have a capacity to accentuate wound healing and osteogenesis. PRP accelerates the hemostatic cascade to stimuli, and also antagonizes the steroidal effect of delay in wound healing. PRP produces an antimicrobial effect, due to its high content of leukocytes. PRP can be used as an effective hemastatic agent. PRP enhances epithelial, epidermal and endothelial regeneration. It promotes angiogenesis, collagen synthesis, soft tissue healing and reduces dermal scarring.

Preparation of Activated PRP:

Activated PRP is prepared by two methods,

1. Manual double spin method
2. Automated method

Manual double spin method

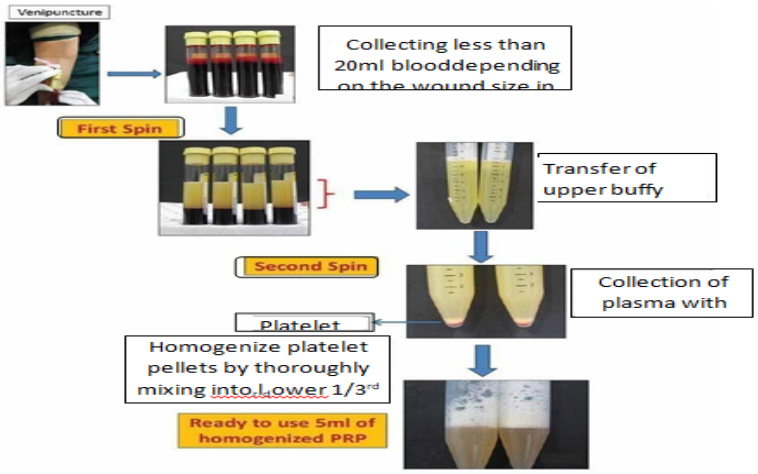

Figure 10:

Automated Devices

Various automated devices are available in the market for the production of activated PRP. Although time saving, these devices are expensive and also with unproven efficacy.

Classification of Platelet Concentrates

Based on the quantity of fibrin and leukocyte, PRP can be broadly classified under following categories,

1. PRP-p(platelet –rich plasma-pure)
2. PRP-L(platelet –rich plasma and leucocyte)
3. PRF-p (platelet –rich fibrin pure)
4. PRF-L(platelet rich fibrin and leucocyte)

Factors affecting wound healing:

Local factors:

- Type, size, and location of the wound: a surgical wound heals faster.
- Injuries in richly vascularized areas (e.g., the face) heal faster than those in poorly vascularised ones (e.g., the foot). In areas where the skin adheres to bony surfaces, as in injuries over the tibia, wound contraction and adequate apposition of the edges are difficult.
- Vascular supply: wounds with impaired blood supply heal slowly.
- For example, the healing of leg wounds in patients with varicose veins isprolonged. Ischemia due to pressure produces bedsores and then preventstheir healing. Ischemia caused by arterial obstruction, often in the lower extremities of diabetics, also prevents healing.
- Infection: wounds provide a portal of entry for microorganisms.
- Infection delays or prevents healing, promotes the formation of excessive granulation tissue and may result in large deforming scars
- Movement: early motion, particularly before tensile strength has been established, subjects a wound to persistent traumas, thereby preventing or retarding healing. Ionizing radiation: prior irradiation interferes with blood supply and result in slow wound healing. Acutely, irradiation of a wound blocks cell proliferation, inhibits contraction, and retards the formation of granulation tissue.
- Ultraviolet light: exposure of wounds to ultraviolet light accelerates the rate of healing.

Systemic Factors:

Regional vascularity: The vascularity of the area surrounding the wound is important. Impaired perfusion results in poor healing. Infections delay wound healing.

Metabolic status: Diabetes mellitus is associated with delayed wound healing because of increased wound infection in diabetics.

Nutrition: Malnutrition impedes wound healing. Methionine and Zinc is needed for proper healing. Vitamin C, required for collagen synthesis and secretion if deficient results in impaired wound healing. Hormones: Corticosteroids impairwound healing by inhibition of collagen synthesis, anti-inflammatory actionsand depression of protein synthesis. Thyroid hormones, androgens, estrogens, and growth hormone also influence wound healing.

Complications of Wound Healing:

Deficient Scar Formation: Inadequate formation of granulation tissue oran inability to form a suitable extracellular matrix leads to deficient scar formation and its complications. Wound Dehiscence And Incisional Hernias: Dehiscence or bursting of a wound results from an increase in pressure from within. Abdominal wound dehiscence carry a mortality of 30%. Ulceration: Wounds ulcerate because of an inadequate blood supply or insufficient vas-

cularization during healing, as in case of leg wounds associated with varicose veins or severe atherosclerosis. Persistent trauma in areas of impaired sensation results in trophic or neuropathic ulcers, example tabes dorsalis, leprosy and diabetic peripheral neuropathy. Excessive Scar Formation: An excessive deposition of extracellular matrix at the wound site results in a hypertrophic scar or a keloid. Histologically both of these types of scars exhibit abundant, broad and irregular collagen bundles, with more capillaries and fibroblasts than expected for a scar of the same age. It indicates a "maturation arrest" or block, in the healing process. Excessive Contraction: An exaggeration of the processes of contraction results in severe deformity of the wound and surrounding tissues, example palmar contracture (Dupuytren's contracture), plantar contracture (Lederhosen disease) and Peyronie's disease (contracture of penile cavernous tissue). Contractures are particularly conspicuous in the healing of burns. Contractures of the skin and underlying connective tissue may compromise joint movements. Contractures in the oesophagus or intestines lead to obstruction of passage of food.

About The Dressing Material Used In This Study:Autologous Platelet Rich Plasma (PRP)

Platelet-rich plasma (PRP) is defined as plasma with a platelet level above peripheral blood concentration. Platelets are the storage pools of growth factors including platelet-derived growth factor, transforming growth factor-ß, platelet-derived epidermal growth factor, vascular endothelial growth factor, insulin-like growth factor-1, fibroblastic growth factor, and epidermal growth factor. When platelets come into contact with exposed endothelium within wounds or damaged tissues, these factors are released and work in harmony with tissue-repair mechanisms such as chemotaxis, cell proliferation, angiogenesis, extracellular matrix deposition, and remodeling to promote appropriate wound healing. Hence, the idea was proposed that increasing platelet concentration in an injured tissue would result in increased levels of multiple bioactive factors and, subsequently, improve the natural healing process.

PRP also known as platelet-enriched plasma, platelet-rich concentrate, autologous platelet gel, and platelet releasate. Platelet releasate have been used to treat wounds since 1985. PRP serves as a growth factor agonist4 and has both mitogenic and chemotactic properties.

Mechanism of Action of Platelet-Rich Plasma

PRP functions as a tissue sealant and drug delivery system, and platelets initiate wound repair by releasing locally acting growth factors via α - granules degranulation

A-granules of platelets contains.

- Platelet-derived growth factor (PDGF-AA, BB, and AB isomers)
- Transforming growth factor-β (TGF-β), platelet factor 4 (PF4)
- Interleukin-1 (IL-1), platelet-derived angiogenesis factor (PDAF)
- Vascular endothelial growth factor (VEGF),
- Epidermal growth factor (EGF)
- Platelet-derived endothelial growth factor (PDEGF), epithelial cell growth factor (ECGF)
- Insulin-like growth factor (IGF), osteocalcin (Oc), osteonectin (On), fibrinogen (Ff), vitronectin (Vn), fibronectin (Fn), and thrombospondin-1 (TSP-1).

These growth factors help in healing by attracting un-differentiated cells in the newly formed matrix and triggering cell division. PRP may suppress cytokine release and limit inflammation, interacting with macrophages to improve tissue healing and regeneration, promote new capillary growth and accelerate epithelialization in chronic wounds.

Preparation of the material

The participants' blood was drawn from the antecubital region. One mL sodium citrate solution was added in vaccum tube and then, 10 mL blood was drawn from the patient based on wound size. Samples were gently shaked to thoroughly mix the anticoagulant with the blood.

Patient in study group is treated with PRP. Platelet rich plasma is made manually by drawing 10 ml of blood by venipuncture.5ml of blood is put in a two test tube each, and adding anticoagulant citrate dextrose (ACD). Centrifuge for 10 minutes at 2000 rotation per minute. Three layers obtained as, top layer plasma, middle layer buffy coat, RBC at the bottom. Plasma and the buffy coat layer was separated by pippet, and put in test tube mixed with calcium chloride ($CaCl_2$). Second centrifugation done for 10 minutes at 2000 rotation per minute.It resulted in three layers, as top platelet poor plasma (PPP), platelet rich plasma and at bottom and RBC. The platelet poor plasma is discarded and platelet rich plasma is separated and taken in syringe, which is injected in wound site. This platelet rich plasma dressing is done biweekly for four weeks and assessed for wound contracture.

Studies have shown that these frequent but small blood draws do not have an effect on hemoglobin, hematocrit, or platelet count.

Studies Supporting the Use of Platelet-Rich Plasma

Driver et al. (2006) carried out the first prospective, randomized, controlled multicenter trial in the United States regarding the use of autologous PRP for the treatment of diabetic foot ulcers.

Methods Participants included 72 patients, suffer-

ing from an ulcer of at least four weeks duration. In this study, investigators compared the effectiveness of autologous PRP gel to that of normal saline gel for 12 weeks. The primary objective of this study was to evaluate the safety of PRP and the incidence of complete wound closure, defined as 100 percent re- epithelialization, when compared to the control treatment, and a secondary objective was rate of wound closure. Patients were randomized into two groups standard of care with PRP gel or control (saline gel) — and were evaluated biweekly for 12 weeks. Results After excluding 32 patients from the final per-protocol analysis because of failure to complete treatment and protocol violations, the authors found that 68.4 percent (13/19) of patients in the PRP group and 42.9 percent (9/21) in the control group had wounds that healed. Wounds in the PRP group healed after a mean of 42.9 days (SD 18.3) vs. 47.4 days (SD 22.0) in the control group. This study is the first published to use the autologel™ System. Mcaleer et al. (2006) used autologous PRP in healing of chronic lower extremity wound in a case study of a 57-year-old man with type 2 diabetes and a wound of six months duration. Reapplication of platelet gel was performed weekly.

Complete closure of the ulcer was achieved by the fourth week of treatment with PRP. PRP can be successful in healing wounds that have failed to heal by other treatment techniques Crovetti et al.(2004) a prospective study regarding the efficacy of platelet gel (PG) in healing cutaneous chronic wounds. Methods 24 patients enrolled in this study varied in origin, and etiologies included diabetes-related, vascular insufficiency, infectious disease, posttraumatic, neuropathic, and vasculitis-related. Protocol for this study consisted of once-weekly PG applications of either autologous or homologous origin. Results, nine patients had healed completely, two went on to receive cutaneous grafts, four had stopped treatment, and nine had responded partiallyand were still receiving treatment.

Although pain was reported as reduced with the application of PG. O'Connell et al. (2008) a pilot study involving the treatment of chronic lower-extremity ulcers with autologous platelet-rich fibrin matrix membrane (PRFM). Methods total patients 21 out of that12 patients with 17 venous lowerextremity ulcers and nine patients with 13 nonvenous lower-extremity ulcers, all who had failed to respond to at least four weeks of conventional treatment. Results complete healing was achieved in 66.7 percent of the patients with venous lower-extremity ulcers in 7.1 weeks (median six weeks) followingan average of two applications of PRFM per patient. Of the nonvenous lowerextremity ulcer group, 44 percent of patients treated with PRFM healed completely during the study period. It suggests that platelet-rich substances such as PRFM have the potential to heal chronic lower-extremity ulcers that have failed to heal by conventional methods. Rationale to use Autologous Platelet-Rich Plasma rather than Allogenic or Homologous Plasma Autologous preparation, PRP is safer to use than allogenic or homologous preparations and there is no chance of infections which can be transmitted through blood and blood products. Such as HIV, hepatitis, West Nile fever, and Creutzfeldt- Jakob disease. PRP requires no special considerations regarding antibody formation, effectively preventing the risk of graft vs. Host disease and leading to better acceptance by patients.

It is every surgeon's desire that after dressing the wound, it should heal without any complications. Successful wound dressing should keep the wound moist and be devoid of any adverse reactions such as infection, maceration and allergy. Diabetic foot ulcers are chronic wounds, stuck in inflammation phase and shows cessation of epidermal growth. The present study was conducted at Theni Medical college and Hospital, Theni to study the effect on chronic diabetic wound healing dynamics. In the present study it was seen that the incidence of diabetic foot ulcerswere more in males (62%) as compared to females (38%). The second national data source, NHDS documented higher hospital rates in males suffering from diabetic foot ulcer. According to age, 45% cases are above 40years of age. 55% cases are below 40years of age. The prevalence of diagnosed diabetics increases with age (the diabetic foot). In this study patients with vascular complications such as pulse less limb and the patients with osteomyelitis were excluded.

In this study, 79% of the ulcers were traumatic in origin, trauma being the triggering factor secondary to neuropathy. 21% were spontaneous in originsecondary to blister rupture or unnoticed trivial trauma. 31 patients had ulcer on the plantar surface of the forefoot and 29 patients had ulcer on the dorsum of foot. Study conducted by Edmonds et al in 1986, (Edmonds) showed more foot ulcers were on plantar and fore foot areas. Most of the diabetic foot ulcers are invariably shoe related and due to gait abnormalities. They can be prevented by appropriate sized footwear. However in our study the incidence of ulcers over the plantar aspect of the foot were not as high as postulated by Edmonds et al. Most of the patients (78%) were on insulin for control of sugar whereas only 22% were on Oral Hypoglycaemic Agents. In our study it was observed that participants receiving PRP dressing had better wound contraction of 38.04%. As compared to the group receiving only conventional dressing (normal saline dressing) in whom the mean wound contraction was 27.45%, these were found to be statistically significant on unpaired Student t test ($p<0.004$) suggesting that PRP dressing enhances wound healing in diabetic wounds. No patients underwent ma-

jor amputations in both the groups.

Feasibility of this study:

In the present study we have taken 100 patients suffering from Diabetes Mellitus with foot ulcers. Patients were taken up for study based on inclusion and exclusion criteria. Out of 50 patients, 50 (28 males, 22 females) were study cases and 50 (34 males and 16 females) were control.

Participants included in the study group were treated with the PRP dressing biweekly for three weeks. All 50 patients selected for PRP treatment complied for the three weeks period of the study. The initial area measurement was taken on first week and final area measurement on third week was taken on transparent sheet. All 50 patients selected as a control complied for the three weeks duration period of the study. The initial area measurement on first week final area measurement on three week was taken on transparent sheet. We have applied the following formula to calculate % reduction in area of wound after three weeks period in both cases and control groups. Rate of contraction of wound after four weeks of treatment = (Initial area – Final Area) X 100

Initial area

We have found 38.04 % rate of contraction of wounds in the control groups as compared to 27.45% contraction of wounds in study group. Therefore, study groups are having % more wound contraction as compared to control group. On applying unpaired student t-test $p<0.004$ which is significant.

Conclusion

Autologous platelet gel is more effective than the local antiseptic dressing interms of healing rate and prevention of infection in clean diabetic ulcers.

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