Original Research Article TO COMPARE THE OUTCOME OF USE OF PRP WITH THAT OF CONVENTIONAL METHODS ON NON-HEALING ULCERS

Dr. Akhilesh Shukla¹ (Asst. Prof.) & Dr. Mahak Bhandari² (Prof.)

Dept. of General Surgery, Sri Aurobindo Medical College & P.G. Institute, Indore (M.P.)^{1&2} Corresponding Author: Dr. Akhilesh Shukla

Abstract

Background & Methods: The aim of the study is to compare PRP with that of conventional methods on non-healing ulcers. All patients in study underwent a detailed history taking including general examination. Under aseptic precautions 20 ml of venous blood was drawn and added to a test tube containing acid citrate dextrose in a ratio of 9:1 (blood: Acid citrate dextrose), centrifuged at 5000 rpm for 15 min to separate the red blood cells from the platelets and plasma.

Results: The comparison of mean area affected at different duration between the two treatment methods. There has been no significant changes up to 4 weeks. The table shows a significant change after 6 weeks (p<0.05). However in further weeks, the comparison of mean area affected is not significant but shows that the number of patients recovered under PRP method is high.

Conclusion: We found more cases were above 45 years of age. To study the etiology and pathogenesis a much larger group with control is needed. The only significant finding was that among the study groups most patients had associated co-morbidities, that are considered to be causative of chronic ulcers. Time taken for healing was significantly less in PRP group as compared to Conventional method group. The functional outcome of PRP use is also better in comparison to conventional group.

Keywords: prp, conventional, non-healing & ulcers. **Study Design:** Comparative Study.

1. Introduction

Chronic nonhealing leg ulcer is defined as the "loss of skin and subcutaneous tissue on the leg or foot, which takes more than 6 weeks to heal[1]. Chronic ulceration of the lower leg, including the foot, is a frequent condition, causing pain, social discomfort, and generating considerable costs. The prevalence of leg ulcers is well documented to be vary between 0.18% and 1%.2 The major causes of lower extremity ulcers are diabetic, venous, arterial, and neuropathic[2].

Chronic wounds are characterized by a long inflammatory phase that hinders the regenerative wound healing. Chronic wounds, especially in patients with diabetes mellitus (DM), are a major health challenge[3]. The goal of wound care in chronic ulcers is to facilitate healing and prevent lower extremity amputations using Standardized protocols of wound care.

A wound is generally acknowledged as all manner of tissue damage resulting in the disruption of the original tissue architecture and homeostasis[4].

Cutaneous wound healing is an essential process consisting of the collaboration of many cell strains and their products. Tissue regeneration and repair processes start immediately after the onset of the lesion. Tissue repair is a simple linear process in which the growth factors cause cell proliferation, thus leading to an orchestra of dynamic changes that involve soluble mediators, blood cells, the production of the extracellular matrix, and the proliferation of parenchymal cells. The skin healing process[5-7], illustrates the principles of repair for the majority of tissues.

2. Material and Methods

Present study was conducted at SAIMS, Indore for 01 Year. Cases of non-healing ulcers who fulfilled the inclusion criteria and gave voluntary and informed consent to be part of a study were enrolled in this study group. Informed consent was taken from all patients included in the study.

All patients in study underwent a detailed history taking including general examination. Under aseptic precautions 20 ml of venous blood was drawn and added to a test tube containing acid citrate dextrose in a ratio of 9:1 (blood: Acid citrate dextrose), centrifuged at 5000 rpm for 15 min to separate the red blood cells from the platelets and plasma. Then the supernatant and the buffy coat composed of platelets and plasma was collected and centrifuged again at 2000 rpm for 5-10 min. The bottom layer about 1.5 ml was taken and 10% calcium chloride was added (0.3 ml for 1 ml of PRP). The mean platelet count was 3.8 Lakhs/cumm (SD 0.95) and the mean final concentration of platelets in PRP was 6.05 Lakhs/cumm Activated PRP was applied onto the wound after proper surgical debridement and were dressed with a non-absorbent dressing (paraffin gauze).

Inclusion criteria:-

- 1. Clinically diagnosed non healing ulcers (>3 weeks duration)
- 2. Patients who give written informed consent.

Exclusion criteria:-

- 1. Those who have biopsy proof of any malignancy
- 2. Patients not willing to give written consent



Fig 1: Ulcers due to vascular insufficiency

Fig 2: Chronic Non- healing ulcers



3. Result

Table No. 1:	Age distribution	of patients in	both groups.
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	No. of Patients >45years	No. of Patients<45years
PRP Group	27(80%)	8(20%)
Conventional Group	27(80%)	8(20%)

In Our study we found 80% in >45 years & 20 % <45 years, 35 patient in each group.

	Diabetes	Varicose Veins	Neurological Paralysis	None
PRP Group	07	06	08	14
Conventional Group	07	06	08	14

Table No. 2:-Comparison of associated co-morbidities in both groups

Table No. 3:- Comparison of mean value of Area of wound between the groups at different Durations

	Duration	GROUP	Mean	S.D	P value
1	Pre	PLATELET RICH PLASMA	21.5	12.4	1.000
		Conventional	21.5	12.4	1.000
2	1 Week	PLATELET RICH PLASMA	18.0	11.6	0.983
		Conventional	17.9	11.7	
3	2 week	PLATELET RICH PLASMA	13.49	9.72	0.738
		Conventional	14.21	9.48	
4	After 4 week	PLATELET RICH PLASMA	10.17	7.96	0.497
		Conventional	11.41	8.25	
5	After 6 week	PLATELET RICH PLASMA	5.40	6.53	0.020*
		Conventional	8.78	7.05	0.029**
6	After 8 week	PLATELET RICH PLASMA	3.90	5.54	0.218
		Conventional	5.34	5.90	0.316
7	After 3 Month	PLATELET RICH PLASMA	2.49	4.82	0.694
		Conventional	3.05	4.67	
8	After 6 Month	PLATELET RICH PLASMA	2.71	3.29	0.632
		Conventional	3.37	3.36	0.052

The comparison of mean area affected at different duration between the two treatment methods. There has been no significant changes up to 4 weeks. The table shows a significant change after 6weeks (p<0.05).However in further weeks, the comparison of mean area affected is not significant but shows that the number of patients recovered under PRP method is high.

4. Discussion

A study conducted on a total of 52 patients were treated using a blood-bank platelet concentrate, and 48 were included in the control group (treatment with topical fibrinogen and thrombin). The use of those concentrates avoided the requirement of blood aspiration from the patient or posterior platelet separation[8]. Complete wound healing was achieved in 79% of patients in the treatment group in comparison with 46% in the control group (P<0.05). The times required for complete healing were 7±1.9 and 9.2±2.2 weeks in the blood-bank platelet concentrate-treated and control groups, respectively (P<0.05). Patient satisfaction with

treatment was also significantly higher in the interventional group (P<0.05). No adverse events related to the study treatment were identified[9-11].

Number of dressings required were far lesser in PRP group patients as compared to Conventional group decreasing the number of hospital visits and the cost of overall treatment was also markedly reduced in PRP conducted the largest study[12]. A total of 52 patients were treated using a blood-bank platelet concentrate, and 48 were included in the control group (treatment with topical fibrinogen and thrombin). The use of those concentrates avoided the requirement of blood aspiration from the patient or posterior platelet separation. Complete wound healing was achieved in 79% of patients in the treatment group in comparison with 46% in the control group (P<0.05) [13]. The time required for complete healing were 7±1.9 and 9.2±2.2 weeks in the blood-bank platelet concentrate-treated and control groups, respectively (P<0.05). Patient satisfaction with treatment was also significantly higher in the interventional group (P<0.05). No adverse events related to the study treatment were identified.

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5. Conclusion

We found more cases were above 45 years of age. To study the etiology and pathogenesis a much larger group with control is needed. The only significant finding was that among the study groups most patients had associated co-morbidities, that are considered to be causative of chronic ulcers. Time taken for healing was significantly less in PRP group as compared to Conventional method group. The functional outcome of PRP use is also better in comparison to conventional group.

6. References

- Cotran RS, Abbas AK, Fausto N, Robbins SL, Kumar V.Robbins & Cotran: Patologia -Bases Patológicas das Doenças. 7. ed. Rio de Janeiro: Elsevier; 2005. 1592 p.
- 2. Gonçalves RV, Souza NTA, Silva PH, Barbosa FS, Neves CA. Influência do laser de arseneto de gálio-alumínio em feridas cutâneas de ratos. Fisoter Mov. 2010;23:381–388
- 3. Rodriguez PG, Felix FN, Woodley DT, Shim EK, The role of oxygen in wound healing: a review of the literature. Dermatol Surg. 2008 Sep; 34(9):1159-69
- 4. Tandara AA, Mustoe TA, Oxygen in wound healing--more than a nutrient. World J Surg. 2004 Mar; 28(3):294-300.
- 5. Edwards R, Harding KG, Bacteria and wound healing. Curr Opin Infect Dis. 2004 Apr; 17(2):91-6.
- 6. Davis SC, Ricotti C, Cazzaniga A, Welsh E, Eaglstein WH, Mertz PM, Microscopic and physiologic evidence for biofilm-associated wound colonization in vivo. Wound Repair Regen. 2008 Jan-Feb; 16(1):23-9.

- Bjarnsholt T, Kirketerp-Møller K, Jensen PØ, Madsen KG, Phipps R, Krogfelt K, Høiby N, Givskov M, Why chronic wounds will not heal: a novel hypothesis. Wound Repair Regen. 2008 Jan-Feb; 16(1):2-10.
- 8. Hardman MJ, Ashcroft GS, Estrogen, not intrinsic aging, is the major regulator of delayed human wound healing in the elderly. Genome Biol. 2008; 9(5):R80.
- 9. Gilliver SC, Ashworth JJ, Ashcroft GS, The hormonal regulation of cutaneous wound healing. Clin Dermatol. 2007 Jan-Feb; 25(1):56-62.
- 10. Franz MG, Steed DL, Robson MC, Optimizing healing of the acute wound by minimizing complications. Curr Probl Surg. 2007 Nov; 44(11):691-763.
- 11. Arnold M, Barbul A, Nutrition and wound healing. Plast Reconstr Surg. 2006 Jun; 117(7 Suppl):42S-58S.
- Gallagher KA, Liu ZJ, Xiao M, Chen H, Goldstein LJ, Buerk DG, Nedeau A, Thom SR, Velazquez OCJ, Diabetic impairments in NO-mediated endothelial progenitor cell mobilization and homing are reversed by hyperoxia and SDF-1 alpha. Clin Invest. 2007 May; 117(5):1249-59
- 13. Falanga V. Wound healing and its impairment in the diabetic foot. Lancet 2005;366:1736-43.
- Brölmann FE, Ubbink DT, Nelson EA, Munte AK, van der Horst CM, Vermeulen H. Evidence-based decisions for local and systemic wound care. Br J Surg. 2012;99:1172– 1183.