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Research Article

RATIONALE OF PRF AS A RIGHTEOUS MODALITY IN ORAL POTENTIALLY MALIGNANT DISORDER FOR PERIODONTAL REGENERATIVE THERAPY

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ABSTRACT

Background: Although little information is available regarding the real prevalence of PMDs in the general population, a commonly accepted prevalence of 13.7% has been reported in Indian population. 40-69 years is the average age of patients with PMDs, which is 5 years before occurrence of oral cancer. Thus there are lot of such cases that requires a periodontal regenerative therapy and this study is carried out to assess the usefulness of regenerative procedures in OPMD cases.

Materials and Methods: Sixty patients were divided into four groups, i.e., 15 samples in Group 1 (Leukoplakia);15 samples in Group 2 (Oral Lichen Planus), 15 samples in Group 3 (Oral Submucous Fibrosis) and 15 samples in Group 4 (Control) with random gender distribution. PRF was prepared from blood samples of all patients and were subjected to cell block cytology method of histological analysis and slides were prepared to histologically assess the changes in (i) Weight of PRF (ii) fibrin network patterns in terms of density and (iii) entrapment of platelets and white blood cells (WBCs) within fibrin meshwork.

Results: In the present study quantitative and qualitative assessment of the PRF clot comprising its fibrin density and pattern of localization of WBCs and platelets in the OPMDs and normal counterpart inculcated to have a highly significant correlation(p value<0.05).

Conclusion: From the current study it can be concluded that Oral potentially malignant disorders can be considered as one of the influencing factors on quantity of PRF as well as on the fibrin network pattern. Thus treatment plan involving PRF in such patients should be modified.

INTRODUCTION

The clinical concept of malignant transformation in oral mucosa has been proposed for more than 100years.The World Health Organization (WHO) defined the term "potentially malignant disorders" as the risk of malignancy being present in a lesion or condition either during the time of initial diagnosis or at a future date. Even though little knowledge is available regarding the real occurrence of PMDs in the general population, a commonly accepted prevalence of13.7% has been reported in Indian population. Average age of patients with PMDs is 40-69 years, which is 5 years before occurrence of oral cancer. Unfortunately, in recent years 5% of PMDs has been observed in personsunder 30. Premalignant disorders are usually found on the buccal mucosa, followed by gingivae, tongue and floor of the mouth.[1]

The following disorders are regarded as being potentially malignant: 1) Leukoplakia/Erythroplakia, 2)Oralsubmucous fibrosis, 3) Palatal lesions in reverse smokers, and, although still somewhat questionable 4) Oral Lichen Planus, and 5) Discoid Lupus Erythematosus.[2]

The prevalence of leukoplakia for all ages is approximately 4.02%, with an increasing prevalence in adults. The male-

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female ratio varies in different parts of the world. The prevalence of oral lichen planus is in general accepted to be approximately 1.38%. This chronic disorder mainly affects middle-aged people.OSF predominantly occurs in Indians and Southeast Asians with a prevalence rate of 8.06%.This condition is more common in young adults, aged 20−40.[3]Thus there are lot of such cases that requires a periodontal regenerative therapy and this study is carried out to assess the usefulness of regenerative procedures in OPMD cases.

Platelet-rich fibrin (PRF) based regenerative procedure is one of such procedures which is globally used with high success rates that braces the periodontal regenerative therapy. PRF is an immune and platelet concentrate collecting on a single fibrin membrane and all the constituents of a blood sample are favourable for healing and regeneration.[4]

Though platelets, leukocytes and cytokines play an important part in the biology of this biomaterial, the fibrin matrix supporting them certainly constitutes the determining element responsible for the real therapeutic potential of PRF.[5]With an effort to achieve the goal of regeneration the use of various biomaterials such as platelet-richplasma (PRP), platelet-richfibrin (PRF) have been introduced, tried and tested. The greatest challenge in clinical research is the development of bioactive surgical additives, which helps to regulate inflammation and increase the rate of healing process.[6] Fibrin glue was originallydescribed in 1970 and is formed by polymerizing fibrinogen with thrombin and calcium.[7] However, the biochemical architecture of fibrin fibrillae obtained by this protocol is condensed tetramolecular orbilateral junctions. This type is least favourable to cytokine enmeshment and cellular migration.[8] For regeneration, it is known that a blood clot is the centre focus of initiating any soft tissue healing and bone regeneration polymers: This leads to arigid network, not very favorable to cytokine enmeshment and cellular migration,[9]leading to cytokines which are small soluble molecules, released too quickly to be closely built inside the fibrin matrix during polymerization.[10] Thus, PRF was first developed in France by Choukroun *et al*. in 2001. This second generation platelet concentrate eliminates the risk associated with the use of bovine thrombin. On the other hand, a slow physiological fibrin polymerization results in higher percentage of equilateral junctions, leading to a flexible network capable of cytokine entrapment and cellular migration.[11] The natural blood clot is seen to contain 95% red blood cells (RBCs), 5%platelets, <1% white blood cells (WBCs) and numerous amounts of fibrin strands.[12] The natural blood clot contains only a small percentage of platelets which, however, are seen to have a central role to play in the regeneration process. Hence, PRF was introduced and is based on a simple strategy of enhancing the healing capacity of a natural blood clot by supplementing the natural blood clot within creased platelet concentrations. Thus, the factors that affect fibrin formation and structure may be: [13]

1. Genetic factors,
2. Acquired factors like (abnormal concentration of thrombin and factor XIII inplasma, blood flow, platelet activation oxidative stress, hyperglycemia, hyperhomocysteinemia, medications, and cigarette smoking) and
3. Other parameters (such as microgravity, pH, temperature, reducing agents and concentration of chloride and calcium ions).

Aims and Objectives

PRF blood clot contains platelet concentration (>97%), high enough to accelerate the soft and hard tissue healing in periodontal regenerative procedures which may vary with patients with different potentially malignant disorders.[14] Thus, the present study aims to analyse and evaluate the quantitative and qualitative variations in PRF and fibrin network patterns of the PRF clot, isolated from individuals of four different groups comprising of OPMD and normal counterpart.

MATERIALS AND METHODS

The study protocol involved blood sample collection after patient selection, PRF preparation, weighing of PRF for quantitative assessment and preparation of slides forhistological analysis and evaluating qualitatively.

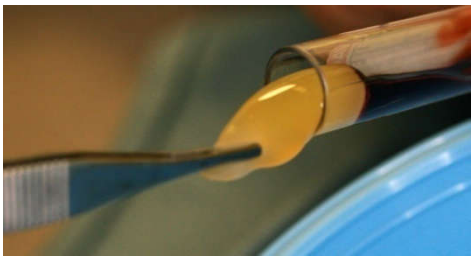
Patient selection and distribution

The study was performed at I.T.S Dental College, Hospital and Research Centre, Greater Noida. Patients participating in the study included those reporting to the Department of Oral and Maxillofacial Pathology for complete blood count. The inclusion criterion for this study was patients with oral potentially malignant disorder between the age group of 20 to 50 years. The exclusion criteria included patients with platelet and coagulation disorders, systemic diseases/conditions, medications affecting the blood, pregnant and lactating women. Sixty patients were divided into four groups, i.e., 15 samples in Group 1 (Leukoplakia), 15 samples in Group 2 (Oral Lichen Planus), 15 samples in Group 3 (Oral Submucous Fibrosis) and 15 samples in Group 4 (Control group with no sign of OPMD) with random genderdistribution.

Plateletrichfibrin preparation (Fig.1a, b, c, d)

a

b



c

d

Figure 1 Platelet-rich fibrin preparation (a) centrifugation machine; (b) platelet-rich fibrin clot; (c) & (d) platelet-rich fibrin clot with red blood cell's layer

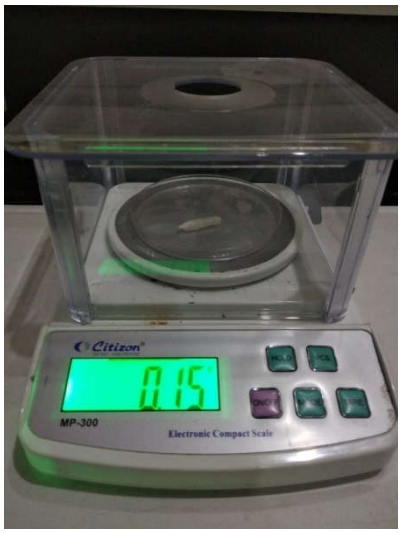
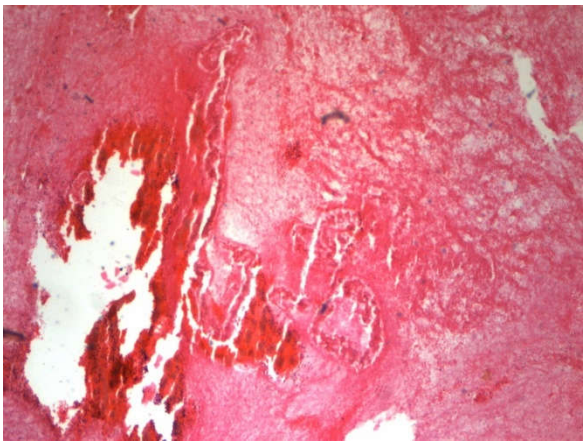
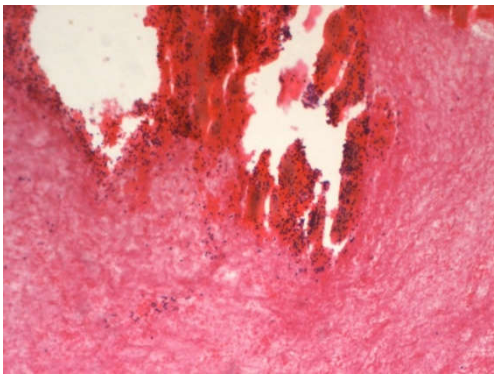


Figure 2 Weighing of PRF :RS232C Electric Balance

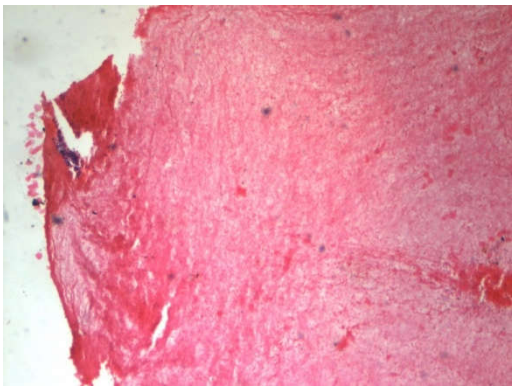


a



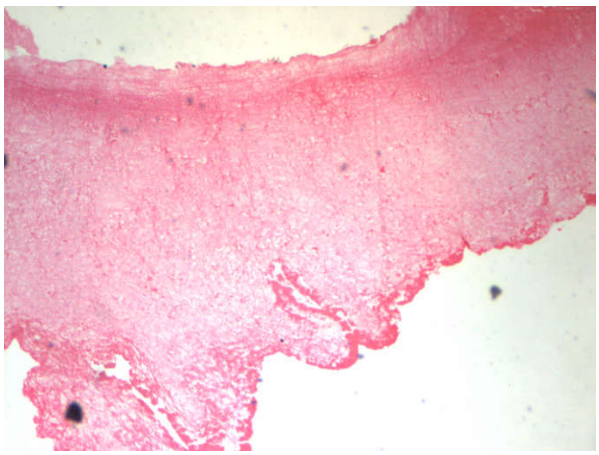
b

c

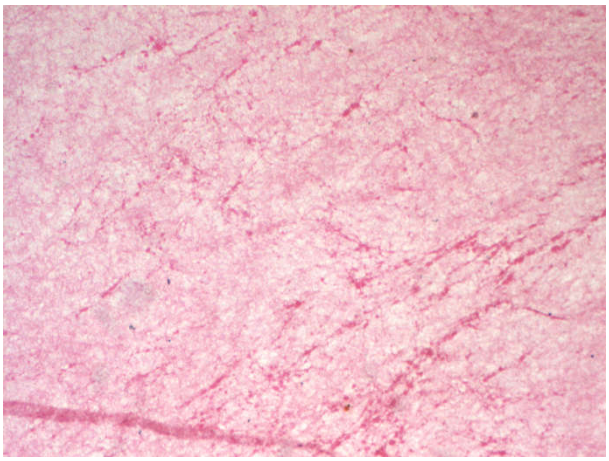


d

Figure 3 (a),(b),(c)&(d)Platelet-rich fibrin clot slide preparation for histological analysis of Normal patients. Blue areas-platelet and white blood cell aggregates, pink areas-dense fibrin network predominantly, pocketing like pattern

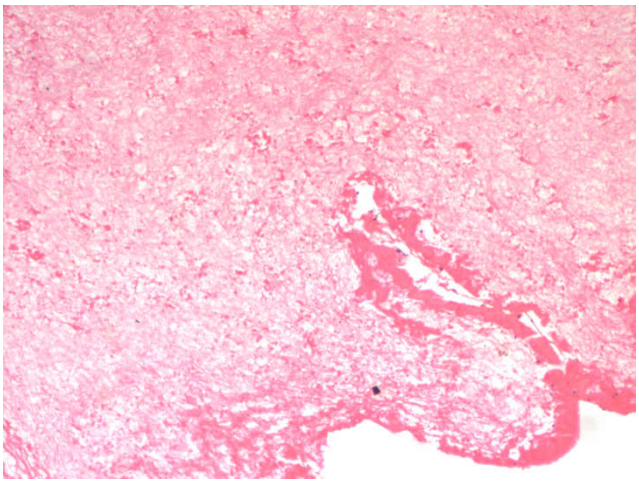


a

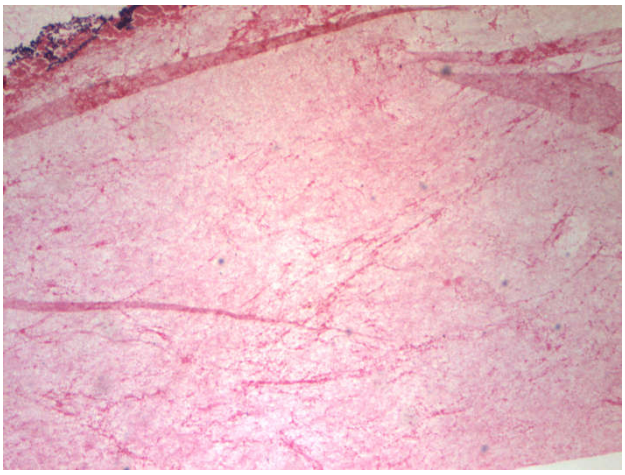


b

Figure 4 (a)& (b) Platelet-rich fibrin clot slide preparation for histological analysis of Leukoplakia patients. Red areas-red blood cell, blue areas-platelets and white blood cell aggregates, dark pink areas-dense fibrin network pattern, light pink areas-loose fibrin network pattern

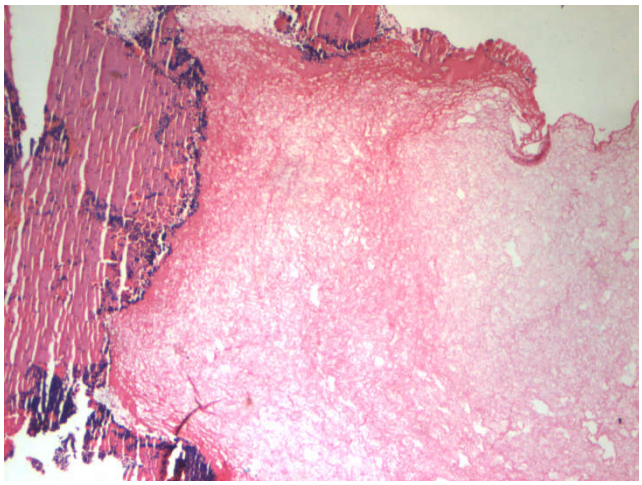


a

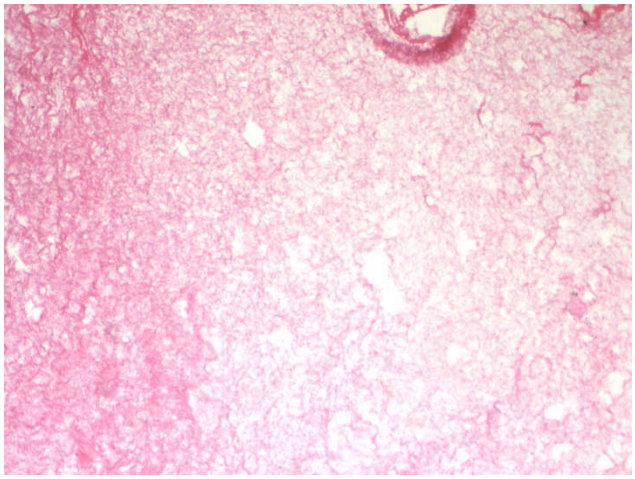


b

Figure 5 (a)& (b) Platelet-rich fibrin clot slide preparation for histological analysis of Oral Lichen Planus patients. Red areas-red blood cell, blue areas-platelets and white blood cell aggregates, dark pink areas-dense fibrin network pattern, light pink areas-loose fibrin network pattern



a



b

Figure 6 (a)& (b) Platelet-rich fibrin clot slide preparation for histological analysis of Oral Submucous Fibrosis patients. Red areas-red blood cell, blue areas-platelets and white blood cell aggregates, dark pink areas-dense fibrin network pattern, light pink areas-loose fibrin network pattern

Intravenous blood 5 ml (by venipuncture the antecubital vein) was collected with a 5 ml disposablesyringe and was immediately transferred to disposable vacuum test tube and centrifuged at 3000 rpm for15 min in the centrifugation machine (REMI). Because of differential densities, it resulted in the separation of three basic fractions: A base of RBCs at the bottom, a cellular plasma on the surface, and finally a PRF clot between the two. The PRF clot thus formed was obtained with the help of sterile tweezer and scissors by cutting it in sucha manner as to preserve a small RBC layer since the platelets and WBCs are concentrated in anintermediate layer located between RBC and the PRF clot.[10]

Quantitative assessment of Platelet-rich fibrin (Fig.2)

The isolated PRF clot was fixed for 24 hrs and then weighed using RS232C Electric Balance.

Qualitative assessment of Platelet-richfibrin clot via histological analysis

The isolated PRF clot was prepared into slides by cell block cytology method. Cell block cytology method is the latest method of analysing fine needle aspiration cytology (FNAC) fluids. However, for this study the same cell block cytology method was applied to study the PRF clot.

The procedural steps followed for obtaining the PRF clot slides by this method were as follows:

Step 1: Fixing - the isolated PRF clot, was transferred into a medium sized perforated stainless steel cassette inside which, a small chit containing the patient number was written with a lead pencil was placed, for the purpose of identification. The cassettes containing the PRF clot were transferred into a container containing 10% formalin where they were fixed for 24 h. The aim of fixing is to preserve the biological tissues in life like state, thereby preventing autolysis or putrefaction.

Step 2: Tissue processing - after 24hrs, from the 10% formalin containing container, the cassettes we retransferred into a perforated stainless steel cylindrical container which were subjected to various steps such as dehydration, clearing, and infiltration of wax into the PRF clot as they passed through various processing solutions such as 10% formalin, 60%, 70%, 80%, 90%, and 100% is opropanol alcohol, xylene (two changes) and paraffin wax in an orderly manner. The aim of tissue processing is to remove water from tissues and replace with a medium that solidifies to allow thin sections to be cut.

Step 3: Embedding - Leuchars blocks were used as molds for embedding tissue using paraffin wax. Embedding grants support to the tissue section for sectioning and production of a slide.

Step 4: Tissue sectioning - sectioning was done using Leica microtome and sections of 4 μm thickness were sliced.

Step 5: Dewaxing - sections were deparaffinized firstly by heating the slides for about 55°C and then immediately were dropped into xylene to eliminate wax. The purpose of dew axing is to allow the tissues to be stained.

Step 6: Tissue staining - sections were stained using hematoxylin and eosin stain. Staining is employed to give contrast to the tissue as well as highlighting particular features of interest.

Step 7: Slide numbering - based on the order of patients maintained in the registers of the department of periodontics and oral pathology.

Step 8: Histological slide analysis was carried out using compound microscope at 10x and 40x magnifications. (Fig.3a,b,c,d)

The stained sections of PRF clot, in all the four groups were assessed for (i) Dense fibrin network pattern (ii) Loose fibrin network pattern (iii) Entrapment pattern of platelets and WBCs within the dense and loose type of fibrin networks.

RESULTS

Quantitative analysis

The PRF clot obtained was first assessed for its quantity and was weighed. The results revealed that there was a significant decrease in the quantity of PRF obtained in cases of the patients with Oral Lichen Planus, Oral Submucous Fibrosis and Oral Leukoplakia. Significant correlation was found in case of weight of PRF when statistically assessed (p value <0.05).

Table 1 Descriptive statistics in relation to WEIGHT OF PRF

Weight of PRF	N	Mean	Std. Deviation	95% Confidence Interval for Mean	
				Lower Bound	Upper Bound
Normal	15	.2087	.03796	.1876	.2297
Leukoplakia.	15	.1120	.02597	.0976	.1264
Lichen planus	15	.1653	.12276	.0974	.2333
Oral submucous fibrosis	15	.1287	.01552	.1201	.1373
Total	60	.1537	.07442	.1344	.1729

Qualitative analysis

The histologic stained sections of PRF clot in all the 4 groups were analysed for dense and loose fibrin network pattern and the entrapment pattern of platelets and WBCs within the dense and loose type of fibrin networks. The sections in general, showed an outermost layer of RBCs, followed by the dense fibrin network layer with maximum number of platelet and WBCs entrapped and the inner layers as moving away from RBC layer were seen to be dominated by loose fibrin network pattern with reduced entrapment of platelets and WBCs.

Table 2 The inferential statistics of the parameters recorded using ANOVA for WEIGHT OF PRF

WEIGHT OF PRF	Sum of Squares	df	Sig.
Between Groups	.083	3	.001
Within Groups	.244	56	
Total	.327	59	

Table 3 Descriptive statistics in relation to FIBRIN DENSITY

Fibrin density	N	Mean	Std. Deviation	95% Confidence Interval for Mean	
				Lower Bound	Upper Bound
Normal	15	1.8667	.35187	1.6718	2.0615
Oral leukoplakia.	15	1.1333	.35187	.9385	1.3282
Oral lichen planus	15	1.1333	.35187	.9385	1.3282
Oral submucous fibrosis	15	1.0667	.25820	.9237	1.2097
Total	60	1.3000	.46212	1.1806	1.4194

Table 4 The inferential statistics of the parameters recorded using ANOVA for Fibrin Density

Fibrin Density	Sum of Squares	df	Mean Square	Sig.
Between Groups	6.467	3	2.156	.000
Within Groups	6.133	56	.110	
Total	12.600	59		

Table 5 Descriptive statistics in relation to WBCS/Platelets Distribution

WBCS/Platelets Distribution	N	Mean	Std. Deviation	95% Confidence Interval for Mean	
				Lower Bound	Upper Bound
Normal	15	1.1333	.35187	.9385	1.3282
Oral Leukoplakia.	15	1.8667	.35187	1.6718	2.0615
Oral Lichen Planus	15	1.8667	.35187	1.6718	2.0615
Oral Submucous Fibrosis	15	1.8667	.35187	1.6718	2.0615
Total	60	1.6833	.46910	1.5622	1.8045

Table 6 The inferential statistics of the parameters recorded using ANOVA for WBCS/Platelets Distribution

WBCS/Platelets Distribution	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	6.050	3	2.017	16.288	.000
Within Groups	6.933	56	.124		
Total	12.983	59			

Table 7 Kruskal-Wallis Test

Test Statistics[a,b]			
	WEIGHT OF PRF	Fibrin density	WBCS/Platelets Distribution
Chi-Square	33.408	30.280	27.493
Df	3	3	3
Asymp. Sig.	.000	.000	.000

a. Kruskal Wallis Test
b. Grouping Variable: 1

Table 8 Post Hoc Tests

Multiple Comparisons

Dependent Variable: Bonferroni

Groups	Comparison groups	Weight of prf		Fibrin density		WBCS/Platelets Distribution	
		Mean Diff. (I-J)	Sig.	Mean Diff (I-J)	Sig.	Mean Diff (I-J)	Sig.
Normal	Leukoplakia.	.09667*	.001	.73333*	.000	-.73333*	.000
	Lichen planus	.04333	.465	.73333*	.000	-.73333*	.000
	Submucous fibrosis	.08000*	.010	.80000*	.000	-.73333*	.000
Oral leukoplakia.	Normal	-.09667*	.001	-.73333*	.000	.73333*	.000
	Lichen planus	-.05333	.186	.00000	1.000	.00000	1.000
	Submucous fibrosis	-.01667	1.000	.06667	1.000	.00000	1.000
Oral lichen planus	Normal	-.04333	.465	-.73333*	.000	.73333*	.000
	Leukoplakia.	.05333	.186	.00000	1.000	.00000	1.000
	Submucous fibrosis	.03667	.803	.06667	1.000	.00000	1.000
Oral submucous fibrosis	Normal	-.08000*	.010	-.80000*	.000	.73333*	.000
	Leukoplakia.	.01667	1.000	-.06667	1.000	.00000	1.000
	Lichen planus	-.03667	.803	-.06667	1.000	.00000	1.000

The results of the study revealed that the type of fibrin varied with the different OPMDs which showed either dense or loose type of fibrin network pattern. In few groups the slide showed both dense as well as loose type of fibrin network pattern. In the groups of normal patients there is domination of dense type of fibrin network pattern with focal platelets and WBCs (*Fig*3 a,b,c,d). Whereas, in the group of Oral lichen planus (*Fig*.4a,b), Oral leukoplakia (*Fig* 5a,b) and Oral submucous fibrosis (*Fig* 6a, b) there is domination of loose type of fibrin network pattern with scattered platelets and WBCs, Fibrin density and platelet/WBCs distribution-the result was significant (p value<0.05)

DISCUSSION

PRF first described by Choukroun *et al*., as a new second generation of platelet concentrate. PRF is used to promote wound healing, bone regeneration, graft stabilization, wound sealing, and hemostasis. Because the fibrin matrix is better organized, it is able to more efficiently direct stem cell migration and the healing program. Release of growth factors from PRF through *in vitro* studies and good results from *in vivo* studies led to optimize the clinical application of PRF.[5] The platelets and leukocyte cytokines play an important part in this biomaterial, but the fibrin matrix supporting them is very helpful in constituting the determining elements responsible for its real therapeutic potential. It was observed and shown that the cells are able to migrate from fibrin scaffold; while some authors demonstrated the PRF as a supportive matrix for bone morphogenetic protein as well.

It enhances the healing capacity of a natural blood clot by supplementing the natural blood clot with increased platelet concentrations.

Fibrin degradation products (FDP) are substances that remain in the bloodstream after the body dissolves a blood clot. The fibrinolytic (clot-busting) system manages and regulates clot dissolving. The digestion of fibrinogen with various concentrations of trypsin results in the formation of a variety of degradation products. The breakdown products produced by trypsin-digested fibrinogen were studied and all showed`antithrombin' activity.[16]

There have been studies done to assess the quantity of FDPs in potentially malignant disorders and these were found to be increased in OSMF, Leukoplakia and oral lichen planus.[17][18]

Extensive research has been carried out till date, to study the functions and properties of platelets and WBCs within the PRF clot and how they can affect the outcome of the treatment. However, this is one of its kind study which tries to shift the focus towards a better understanding of the fibrin network in terms of its quantity, arrangement, distribution and hence, the important role it can play in influencing the functions and activity of the platelets in the regenerative therapies of OPMD patients.

In the current research, we could observe the following: The variations in the platelet - WBC distribution within the PRF clot: The highest platelet/leukocyte density is found in the 1 mm of the yellow clot, just after the red clot. The platelet/leukocyte distribution becomes increasingly scarce as they move away from the red clot.
Loose type of fibrin pattern-mainly in the areas associated closely with the sites of platelet distribution and close to the RBC layer and the density of fibrin network reduced in terms of quantity and quality in patients having Oral Leukoplakia, Oral Lichen Planus and Oral Submucous Fibrosis.

Dense type of fibrin pattern-in relation with area away from the platelet distribution. This study thus shows denser fibrin network and a larger amount of WBC and platelet entrapment in control group.

Yet there were intra group variations noted, which could be because of nutritional status, the gender of the patients, the influence of which was insignificant in this study.

These results show that the fibrin network density and their entrapment capacity of platelet and WBCs share a positive correlation. This clearly points out at the fact that, platelets and fibrin are interdependent in carrying out the process of wound healing and that their interaction is the key to successful wound healing and hence, regeneration. This interdependency can be explained on the basis of the functions and role of platelets and fibrin network in natural mechanism of wound healing that follows at the site of vascular injury.[7]

Platelets accumulate in significant numbers at the site of vascular injury, when sub endothelium be comes exposed to the bloodstream.[19] Fibrillar collagen is a major thrombogenic component of sub endothelium, and it triggers platelet

thrombus formation in flowing human blood.[20] The formation of a platelet thrombus is dependent on the platelet glycoprotein IIbIIIa, which binds to its bivalent ligand fibrinogen and thereby cross links the platelets.[21] In addition, activated platelets participate in coagulation by providing assembly sites for the factor VIIIa (F.VIIIa)/F.LXa and F. Va/F. Xa complexes.[22] This results in the generation of thrombin on the platelet surface and the formation of a fibrin network. Thus, this step throws light on a very important function of platelets, i.e. "pro-coagulant activity" which the collagen adherent platelets exhibit and hence mediate the fibrin network formation at the site of injury.[23]

Thus, correlating the interactions between platelets and fibrin network in the natural process of wound healing, with that of biochemical analysis of the PRF composition indicates that, the fibrin network density and distribution is directly proportional to the distribution of platelets in association with them.

Now that the fibrin formation is initiated by the platelets, this formed fibrin continues to form in networks at the site of injury and in turn entraps the platelets and secures them to stay at the site for a prolonged time and continue with the function of growth factor release an essential step in wound healing process.

Thus, fibrin network supports the platelets to efficiently carry out their functions by acting as a scaffold. Hence, it would not be wrong to say that, the binding of fibrin (ogen) to haemostasis proteins and platelets as well as to several different cells such as endothelial cells, smooth muscle cells, fibroblasts, leukocytes, and keratinocytes is indispensable during the process of wound repair.

Laurens *et al*. stated that the outcome of wound healing depends largely on the fibrin structure, such as the thickness of the fibres, the number of branch points, the porosity, and the permeability.[24] Second, wound healing also depends upon the platelet concentration and their functions. Hence, focusing on the fibrin network functions in wound healing and their interaction with platelets and WBCs become very important for us to know in detail, the factors which can influence the fibrin network structure. Thus, this is the first study that attempts to explore the influence of one such factor called oral premalignant disorders, on the fibrin network arrangement and the resultant variations and hence, their influence on the interactions between platelets and fibrin networks.

Dohan *et al*. showed the potential benefits of using PRF in periodontal regeneration is due to the cytokines which are present in platelet concentrates and the fine, flexible fibrin network which is more elastic in naturefavouring cytokine entrapment and cellular migration,[10] thus highlighting the importance of fibrin network in periodontal regeneration.

This article shows how potentially malignant disorders can influence the fibrin network patterns and hence, platelet - WBC entrapment which in turn could influence the cytokine entrapment. Therefore, it can be stated that potentially malignant disorders can be one of the influential factors in determining the PRF clot efficacy obtained from subjects of different OPMD groups and hence, can be an important predictor of how much success to expectin terms of regeneration when treating subjects with OPMDs.

To study the fibrin networks, the changes in their efficacy their ability to entrap the platelets and WBCs we performed the cell block cytology method using haematoxylin and eosin stain, proved to be satisfactory in identifying the variations in the fibrin network pattern and distribution and also the variations in platelet-WBCs concentrations entrapped within them. However, drawbacks noticed were, inability to correctly appreciate the changes in the individual fibrin strand morphology and thickness, difficulty in identifying and separating out the platelets and WBCs from each other represented by haematoxylin staining and exact counting of the platelets entrapped within the fibrin meshwork.

From this study, it can be stated that, the two major constituents of PRF, i.e., the platelets and fibrin network are interdependent thus, clearly showing that this interaction between them is the basis for the various properties of the PRF clot formed. The interdependency is that platelets actively participate in the initiation of coagulation cascade and then formed fibrin network pattern in turn entraps the platelets and WBCs and help them perform their functions for a sustained period of time.

CONCLUSION

This study concludes that OPMDs can affect one of the factors playing a significant role in altering fibrin network patterns and hence, its interaction with platelets thus, influencing the quality of the PRF clot applied for periodontal regeneration procedures. However, further studies focusing on finding out the influence of nutrition and gender in association with OPMDs on the fibrin network pattern and their platelet-WBC entrapment capacity could be beneficial.

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